Fascia: The Tensional Network of the Human Body

The science and clinical applications in manual and movement therapy

Edited by

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Glossary

Heike Jäger

- Actin A globular protein found in all eukaryotic cells, polymerizes to microfilaments; one of the three major components of the <u>cytoskeleton</u> and thin filaments of the contractile apparatus. Actin has an array of functions including muscle contraction, cell signaling and morphology, vesicle and organelle movement, cell motility, phagocytosis, and cytokinesis.
- **Adhesions** Inflammatory bands of scar-like tissue that form between two surfaces inside the body.
- **Adhesive capsulitis** An inflammatory condition that restricts motion in the shoulder, commonly referred to as "frozen shoulder".
- Alpha smooth muscle actin One of six known smooth muscle actin isoforms. In addition to its presence in organ tissue, alpha smooth muscle actin has been identified in myofibroblasts, where it plays an important role in focal adhesion maturation and in cell motility.
- **Angiogenesis** A physiological process involving the growth of new blood vessels from pre-existing vessels, for example in the process of wound healing.
- **Aponeurosis** A thin, flat tendon-like expansion of fascia important in the attachment of muscles to bones.
- **Apoptosis** A morphologic pattern of cell death affecting single cells, marked by formation of cytoplasmic blebs, shrinkage of the cell, condensation of chromatin, and fragmentation of the cell into membrane-bound apoptotic bodies that are eliminated by phagocytosis. It is a mechanism for cell deletion in the regulation of cell populations.
- **Bradykinin** A nonapeptide produced by activation of the kinin system in a variety of inflammatory conditions. A potent vasodilator, it also increases vascular permeability, stimulates pain receptors, and causes contraction of a variety of extravascular smooth muscles.
- **Calcitonin gene-related peptide (CGRP)** A 37-amino acid polypeptide is formed from the alternative splicing of the calcitonin/CGRP gene and acts as a potent vasodilator and neurotransmitter. Widely distributed in

the central and peripheral nervous systems; also present in the adrenal medulla and gastrointestinal tract.

- **Cell signaling** The process by which a cell receives and acts on some external chemical or physical signal, including receiving the information at specific receptors in the plasma membrane, conveying the signal across the plasma membrane into the cell, and subsequently stimulating a specific cellular response.
- **c-fiber** Unmyelinated nerve fiber that conducts action potentials at a velocity of less than 2.5 m/s in humans.
- **Chondroblasts** Immature cartilage cells that produce the cartilaginous matrix.
- **Chromatin** The more readily stainable portion of the cell nucleus, forming a network of nuclear fibrils. Comprised of DNA attached to a protein structure base (primarily histones). Occurs in two states, euchromatin and heterochromatin, with different staining properties, and during cell division coils and folds to form the metaphase chromosomes.
- **Collagen** The most abundant protein in mammals; a major component of fascia, giving it strength and flexibility. At least 14 types exist, each composed of tropocollagen units that share a common triple-helical shape but varying somewhat in composition between types, with the types being localized to different tissues, stages, or functions.
- **Compartment syndrome** Involves the compression of nerves and blood vessels within a fascial compartment, leading to impaired blood flow and muscle and nerve damage. Most common in the lower leg or forearm.
- **Cytokines** A generic term for non-antibody proteins released by one cell population (e.g., primed T lymphocytes) on contact with specific antigens, which act as intercellular mediators, as in the generation of an immune response.
- **Cytoskeleton** The conspicuous internal reinforcement in the cytoplasm of a cell, consisting of tonofibrils, terminal web, or other microfilaments.

Deep fascia (or fascia profunda) The dense fibrous fascia that interpenetrates and surrounds the muscles as well as muscle groups.

Differentiated myofibroblast A myofibroblast that expresses alpha smooth muscle actin stress fiber bundles.

- **Dry needling** An invasive procedure in which an acupuncture needle is inserted at myofascial trigger points to inactivate it.
- **Dupuytren's contracture** A thickening and contracture of the palmar fascia.
- **Dynamometer** An instrument for measuring the force of muscular contraction.
- **Ehlers–Danlos syndrome** A group of inherited connective tissue disorders, caused by a defect in the synthesis of <u>collagen</u>. Occurs in at least 10 types, varying in severity from mild to life-threatening. Transmitted genetically as autosomal recessive, autosomal dominant, or X-linked recessive traits. The major manifestations include hyperextensible skin and joints, easy bruisability, friability of tissues with bleeding and poor wound healing, calcified subcutaneous spheroids, and pseudotumors.
- **Elastin** A scleroprotein, the essential constituent of yellow elastic connective tissue. Brittle when dry, but when moist is flexible and highly extensile.
- **Electron microscopy** An imaging technique using electrons to illuminate and create an image of a specimen. It has much higher magnification and resolving power than a light microscope, with magnifications up to about two million times compared to about 2000 times. Unlike a light microscope, which uses glass lenses to focus light, the electron microscope uses electrostatic and electromagnetic lenses to control the illumination and imaging of the specimen.
- **Endomysium** The fascial layer that sheaths single muscle fibers.
- **Endotenon** A thin fascial membrane within a tendon that invests each collagen fibril and each collagen fiber, and envelops the primary, secondary and tertiary fiber bundles.
- **Endothelium** The layer of epithelial cells that lines the cavities of the heart, the lumina of blood and lymph vessels, and the serous cavities of the body.
- **Epimysium** The fascial layer that envelops an entire muscle.
- **Epineurium** The outermost fascial layer of a peripheral nerve, surrounding the entire nerve and containing its supplying blood vessels and the lymphatic system.
- **Epitenon** A fine, loose connective tissue sheath covering a tendon over its entire length.
- **Extracellular matrix** Any material produced by cells and excreted to the extracellular space within the tissues. Takes the form of both ground substance and fibers and is composed chiefly of fibrous elements, proteins involved in cell adhesion, and glycosaminoglycans and other molecules. It serves as a scaffolding holding tissues

together and its form and composition help determine tissue characteristics.

- **Fascia** The soft tissue component of the connective tissue system. Interpenetrates and surrounds muscles, bones, organs, nerves, blood vessels and other structures. Fascia is an uninterrupted, three-dimensional web of tissue that extends from head to toe, from front to back, from interior to exterior. Fascia can refer to dense planar fascial sheets (such as the fascia lata) as well as joint capsules, organ capsules, muscular septa, ligaments, retinacula, aponeuroses, tendons, myofascia, neurofascia, and other fibrous collagenous tissues (see introduction Chapter, page xvi).
- **Fascial distortion model (FDM)** A diagnostic as well as treatment system for fascial distortions developed by Stephen Typaldos (see Chapter 7.15).
- **Fasciotomy** A surgical incision or transection of fascia, often performed to release pressure in compartment syndrome.
- **Fibroblasts** Flat elongated fascial cells with cytoplasmic processes at each end, and a flat, oval, vesicular nucleus. Fibroblasts form the fibrous tissues in the body, including tendons, aponeuroses, supporting and binding tissues of all sorts.
- **Fibronexus** An adhesion in a myofibroblast that links actin across the cell membrane to molecules in the extracellular matrix like fibronectin and collagen.
- **Fibrosis** The formation of fibrous tissue, as in repair or replacement of parenchymatous elements.
- **Gap junctions** Direct connections between the cytoplasms of two cells, allowing various molecules and ions to pass, e.g., most sugars, amino acids, nucleotides, vitamins, hormones, and cyclic AMP. A gap junction channel is composed of two connexons (or hemichannels), which connect across the intercellular space. In electrically excitable tissues, these gap junctions serve to transmit electrical impulses via ionic currents and are known as electronic synapses.
- **Glycosaminoglycans (or mucopolysaccharides)** High molecular weight linear heteropolysaccharides having disaccharide repeating units containing an *N*-acetylhexosamine and a hexose or hexuronic acid; either or both residues may be sulfated. This class of compounds includes the chondroitin sulfates, dermatan sulfates, heparan sulfate and heparin, keratan sulfates, and hyaluronic acid. All except heparin occur in proteoglycans that consist of glycosaminoglycans <u>covalently linked</u> to a <u>protein</u>.
- **Golgi receptors** Mechanosensory receptors found in dense proper fascia, in ligaments (Golgi end organs), in joint capsules, as well as around myotendinous junctions (Golgi tendon organs).
- **Hyaluronic acid** A glycosaminoglycan; part of the extracellular matrix of synovial fluid, vitreous humor, cartilage, blood vessels, skin, and the umbilical cord. Along with lubricin, it maintains viscosity of the extracellular matrix, allowing for necessary lubrication of certain tissues.

Glossary

Hypermobility (or laxity) A greater than normal range of motion in a joint, which may occur naturally in otherwise normal persons or may be a sign of joint instability.

Hypertonia Excessive tone of the skeletal muscles that increases their resistance to passive stretching.

Hypertrophy The enlargement or overgrowth of an organ or part due to an increase in size of its constituent cells.

Hypocapnia A deficiency of carbon dioxide in the blood, resulting from hyperventilation and eventually leading to alkalosis.

Hysteresis A property of systems that do not instantly react to the forces applied to them, but react slowly, or do not return completely to their original state.

Immunoassay A biochemical test that measures the concentration of a substance in solution, using an antigen/antibody reaction.

Immunofluorescence A technique that uses fluorescent dye-labeled antibodies to visualize subcellular distribution of biomolecules of interest.

Integrins Refers to any of a family of heterodimeric celladhesion receptors, consisting of two non-covalently linked polypeptide chains, designated α and β , that mediate cell-to-cell and cell-to-extracellular matrix interactions.

Interstitial fluid The extracellular fluid that bathes the cells of most tissues but which is not within the confines of the blood or lymph vessels and is not a transcellular fluid. Formed by filtration through the blood capillaries and drained away as lymph. It is the extracellular fluid volume minus the lymph volume, the plasma volume, and the transcellular fluid volume.

Laminin An adhesive glycoprotein component of the basement membrane. It binds to heparan sulfate, type IV collagen, and specific cell-surface receptors and is involved in the attachment of epithelial cells to underlying connective tissue.

Ligament A band of fascia that connects bones or supports viscera. Some are distinct fibrous structures; some are folds of fascia or of indurated peritoneum; others are relics of fetal vessels or organs.

Mechanoreceptors Sensory receptors that respond to mechanical pressure, deformation or proprioception.

Mechanotransduction The mechanism by which cells convert a mechanical stimulus into chemical activity.

Mepyramine An antihistaminic pharmacological substance that is frequently used as an in vitro contractile agent for tissues containing myofibroblasts.

Microdialysis A diagnostic technique that uses an ultrafine needle to biopsy the chemical components of the fluid in the extracellular space of tissues.

Morphogenesis The evolution and development of form, as in the development of the shape of a particular organ or part of the body.

Myofascial pain syndrome A chronic musculoskeletal pain disorder associated with local or referred pain, decreased range of motion, autonomic phenomena, local twitch response in the affected muscle, and muscle weakness without atrophy. **Myofibroblasts** Differentiated fibroblasts that combine the features of both fibroblasts and smooth muscle cells. Due to their expression of stress fiber bundles containing alpha smooth muscle actin and due to strengthened adhesion sites on their membrane, these cells possess a much higher contractile potential than normal fibroblasts.

Myosin The most abundant protein in muscle, occurring chiefly in the A band. Along with actin, it is responsible for the contraction and relaxation of muscle. Myosin uses ATP hydrolysis to generate force and to "walk" along the filament. It is the main constituent of the thick filaments of muscle fibers.

Neuropathy A functional disturbance or pathological change in the peripheral nervous system.

Neuroplasticity The changes that occur in the organization of the brain as a result of experience.

Nociceptors Receptors for pain that are activated by physical, mechanical, thermal, electrical, or chemical stimuli.

Osteoblasts Cells that arise from fibroblasts and are associated with the production of bone.

Oxytocin A nonapeptide secreted by the magnocellular neurons of the hypothalamus and stored in the neurohypophysis along with vasopressin. It promotes uterine contractions, milk ejection, contributes to the second stage of labor and is released during orgasm in both sexes. In the brain, oxytocin regulates circadian homeostasis, such as body temperature, activity level, and wakefulness. It is involved in social recognition, bonding, and trust formation.

Pacinian corpuscles Lamellar or lamellated large encapsulated nerve endings located in fascia that are sensitive to vibration, and acceleration of movement. They require dynamically changing stimuli and do not respond to static pressure.

Perimysium The fascial membrane which groups individual muscle fibers (between 10 to 100+) into bundles or fascicles.

Perineurium An intermediate layer of fascia in a peripheral nerve, surrounding each bundle (fasciculus) of nerve fibers.

Piezoelectric The ability of some materials to generate an electric potential in response to applied mechanical stress.

Plantar fasciitis An inflammatory condition of the plantar fascia.

Plantar fibromatosis The formation of fibrous, tumorlike nodules arising from the deep layer of the plantar fascia, manifested as single or multiple nodular swellings, sometimes accompanied by pain but usually unassociated with contractures.

Prestress Endogenous tension.

Procollagen The precursor molecule of collagen, synthesized in the fibroblast, osteoblast, etc., and cleaved to form collagen extracellularly.

Prolotherapy (or RIT, regenerative injection therapy) An injection therapy used to treat chronic ligament, joint, capsule, fascial and tendinous injuries to promote non-surgical soft tissue repair and to relieve pain. **Proprioception** Perception mediated by sensory nerve endings found in muscles and fascia, which give information concerning movement and position of the body.

- **Proteoglycans** Heavily glycosylated glycoproteins that are found in the extracellular matrix of fascia, composed mainly of polysaccharide chains, particularly glycosaminoglycans, as well as minor protein components that form large complexes, both to other proteoglycans, to hyaluronan and to fibrous matrix proteins (such as collagen).
- **Protomyofibroblasts** Develop from fibroblasts under mechanical tension. They form cytoplasmic actincontaining stress fibers that terminate in fibronexus adhesion complexes.
- **Reticular fibers** Fascial fibers composed of collagen type III that form the reticular framework of lymphoid and myeloid tissue and also occur in the interstitial tissue of glandular organs, the papillary layer of the skin, and elsewhere.
- **Retinaculum** A thickened band of fascia that retains an organ or tissue in place.
- **Ruffini endings** Types of lamellated corpuscle that are slowly-adapting receptors for sensations of continuous pressure.
- **Sarcoplasmic reticulum** A special form of a granular reticulum found in the sarcoplasm of striated muscle and comprising a system of smooth-surfaced tubules forming a plexus around each myofibril.
- **Sclerosis** An induration or hardening caused by inflammation, fascial thickening, or disease of the interstitial fluid.
- **Serotonin** A monoamine vasoconstrictor, synthesized in the intestinal chromaffin cells or in central or peripheral neurons and found in high concentrations in many body tissues, including the intestinal mucosa, pineal body, and central nervous system.
- **Substance P** An undecapeptide that functions as a neurotransmitter and as a neuromodulator and belongs to the tachykinin neuropeptide family.
- **Superficial fascia** Comprised mainly of loose areolar connective tissue and adipose. In addition to its subcutaneous presence, this type of fascia surrounds organs and glands, neurovascular bundles, and is found at many other locations.
- **Surface electromyography** A technique in which electrodes are placed on (not into) the skin overlying a muscle to detect the electrical activity of the muscle.
- **Tendon** A fibrous cord of fascia by which a muscle is attached to the skeleton.
- **Tendon sheath** A membranous sleeve which envelops the tendon and creates a lubricated low-friction environment for easy movement.
- **Tensegrity** A structural principle which uses isolated components in <u>compression</u> inside a net of continuous tension.
- **Transdifferentiation** A biological process that occurs when a non-stem cell transforms into a different type of

cell, or when an already differentiated stem cell creates cells outside its already established differentiation.

- **Transforming growth factor** Two classes that are structurally or genetically not related to one another. TGF- α binds the epidermal growth factor receptor and also stimulates growth of microvascular endothelial cells. TGF- β exists in several subtypes, all of which are found in hematopoietic tissue, stimulate wound healing, and in vitro are antagonists of lymphopoiesis and myelopoiesis.
- **Trigger point** Palpable as localized hardening in the muscle; pain evoked by pressure on the tender spot is recognized as being familiar by the patient, local twitch response is possible; limitation of stretch range of motion, and some weakness of that muscle.
- **Tropocollagen** The basic structural unit of collagen; a helical structure consisting of three polypeptide chains, each chain composed of about a thousand amino acids, coiled around each other to form a spiral and stabilized by inter- and intrachain covalent bonds.
- Tropoelastin The precursor of elastin.
- **Tropomyosin** Along with troponin, regulates the shortening of the muscle protein filaments actin and myosin. In the absence of nerve impulses to muscle fibers, tropomyosin blocks interaction between myosin crossbridges and actin filaments.
- **Ultrasound elastography** A non-invasive imaging method to measure stiffness or strain of soft tissue or to provide images of tissue morphology or other biomechanical information.
- Vimentin filaments Intermediate filaments of the cytoskeleton that are responsible for maintaining cell integrity. They act as cytoskeletal support structures, play a role in mitosis, and are clustered particularly around the nucleus, probably helping to control its location.
- **Vinculin** A protein found in muscle, fibroblasts, and epithelial cells that binds actin and appears to mediate attachment of actin filaments to integral proteins of the plasma membrane.
- **Viscoelastic** Describes materials that exhibit both viscous and elastic characteristics when undergoing plastic deformation. Viscous materials, like honey, resist shear flow and strain linearly with time when a stress is applied. Elastic materials strain instantaneously when stretched and just as quickly return to their original state once the stress is removed. Viscoelastic materials have elements of both of these properties and, as such, exhibit time dependent strain.
- **Wolff's law** The theory developed by 19th century anatomist/surgeon Julius Wolff stating that bone in a healthy person or animal will adapt to the loads it is placed under. If loading on a particular bone increases, the bone will remodel itself over time to become stronger to resist that sort of loading. The converse is also true, i.e., if the loading on a bone decreases, the bone will become weaker due to turnover as it is less metabolically costly to maintain and there is no stimulus for continued remodeling required to maintain bone mass.

Welcome to the world of fascia!

This book is the first comprehensive text in a new field in musculoskeletal therapy and research: the fascinating world of fascia. Fascia forms a continuous tensional network throughout the human body, covering and connecting every single organ, every muscle, and even every nerve or tiny muscle fiber. After several decades of severe neglect, this "Cinderella of orthopedic science" is developing its own identity within medical research. The number of research papers on fascia in peer-reviewed journals has shown a steady rise. The first International Fascia Research Congress, held at the Conference Center, Harvard Medical School in October 2007 was followed by a second in Amsterdam in 2009 and there will shortly be a third in Vancouver in 2012. Similar to the rapidly growing field of glia research in neurology, this underestimated contextual tissue, fascia, is being found to play an important role in health and pathology.

Hypotheses which accord myofascia a central role in the mechanisms of therapies have been advanced for some time in the fields of acupuncture, massage, structural integration, chiropractic and osteopathy. Practitioners in these disciplines, especially those which do not have the longevity of osteopathy or chiropractic, are generally unaware of the scientific basis for evaluating such hypotheses. Many practitioners are unaware of the sophistication of current laboratory research equipment and methods. Laboratory researchers, in turn, may be unaware of the clinical phenomena which suggest avenues of exploration. Thirty years ago the study of physical medicine and rehabilitation included muscle strengthening, anatomy, exercise physiology, and other aspects of therapeutic modalities. What was notably less present in the scientific and medical literature was how to understand and treat disorders of the fascia and connective tissues. Since then much additional information has been developed, particularly since 2005 (see Fig. 0.1).

The purpose of this book is to organize relevant information for scientists involved in the research of the body's connective tissue matrix (fascia) as well as for professionals involved in the therapeutic manipulation of this body wide structural fabric. While it grew out of materials presented at the First and the Second International Fascia Research Congresses in 2007 and 2009 (www.fasciacongress.org), it reflects the efforts of almost 100 scientists and clinicians.

Not only a packing organ

As every medical student knows and every doctor still remembers, fascia is introduced in anatomy dissection courses as the white packing stuff that one first needs to clean off, in order "to see something". Similarly, anatomy books have been competing with each other, in how clean and orderly they present the locomotor system, by cutting away the whitish or semitranslucent fascia as completely and skillfully as possible. Students appreciate these appealing graphic simplifications, with shiny red muscles, each attaching to specific skeletal points. However, these simplified maps do not fully describe how the real body feels and behaves, be it in medical surgery or during therapeutic palpation.

To give an example: in real bodies, muscles hardly ever transmit their full force directly via tendons into the skeleton, as is usually suggested by our textbook drawings. They rather distribute a large portion of their contractile or tensional forces onto fascial sheets. These sheets transmit these forces to synergistic as well as antagonistic muscles. Thereby they stiffen not only the respective joint, but may even affect regions several joints further away. The simple questions discussed in musculoskeletal textbooks "which muscles" are participating in a particular movement thus become almost obsolete. Muscles are not functional units. no matter how common this misconception may be. Rather, most muscular movements are generated by many individual motor units, which are distributed over some portions of one muscle, plus other portions of other muscles. The tensional forces of these motor units are then transmitted to a complex network of fascial sheets. bags, and strings that convert them into the final body movement.



Fig. 0.1 • Number of peer reviewed scientific papers on fascia • Papers on fascia indexed in Ovid Medline or Scopus have grown from 200 per year in the 1970s and 1980s to almost 1000 in 2010.

Similarly, it has been shown that fascial stiffness and elasticity play a significant role in many ballistic movements of the human body. First discovered by studies of the calf tissues of kangaroos, antelopes, and later by horses, modern ultrasound studies have revealed that fascial recoil plays in fact a similarly impressive role in many of our human movements. How far you can throw a stone, how high you can jump, how long you can run, depends not only on the contraction of your muscle fibers; it also depends to a large degree on how well the elastic recoil properties of your fascial network are supporting these movements.

If the architecture of our fascial network is indeed such an important factor in musculoskeletal behavior, why has this tissue been overlooked for such a long time? There are several answers to this question. The development of new imaging and research tools now allow us to study this tissue in vivo. Another reason is that this tissue resists the classical method of anatomical research: that of splitting something into separate parts that can be counted and named. You can reasonably estimate the number of bones or muscles; yet any attempt to count the number of fasciae in the body will be futile. The fascial body is one large networking organ, with many bags and hundreds of rope-like local densifications, and thousands of pockets within pockets, all interconnected by sturdy septa as well as by looser connective tissue layers.

What is fascia?

This varied nature of fascia is reflected in the many different definitions of which exact tissue types are included under the term "fascia". The International Anatomical Nomenclature Committee (1983) confirmed the usage of previous nomenclature committees and used the term "fascia superficialis" for the loose layer of subcutaneous tissue lying superficial to the denser layer of "fascia profunda." While most medical authors in English-speaking countries followed that terminology, it was not congruently adopted by authors in other countries. The nomenclature proposed by the Federative Committee on Anatomical Terminology (1998), therefore attempted to lead towards a more uniform international language (Wendell-Smith 1997). It suggested that authors should no longer use the term fascia for loose connective tissue layers, such as the former "superficial fascia", and to apply it only for denser connective tissue aggregations. However, this attempt failed significantly (Huijing & Langevin 2009). Most English textbook authorities continued to use the term "superficial fascia" to describe subcutaneous tissues (Standring 2008). In addition an increasing number of non-English authors - following the common Anglo-Saxon trend in international medicine – have started to adopt the same terminology as these American or British colleagues.

Similarly there has been confusion on the question which of the three hierarchical muscular tissue bags – epi-, peri- and endomysium – could be included as fascia. While most authors would agree to consider as fascial tissues, muscular septi and the perimysium (which is often quite dense, particularly in tonic muscles) there is less consensus on the endomysial envelopes around single muscle fibers, based on their much looser density and higher quantity of collagen types III and IV. However, almost all authors emphasize the important continuity of these intramuscular connective tissues, and this continuity was shown extending even within the muscle cell (Purslow 2009). So where does fascia stop?

Another area , still to be resolved, are the visceral connective tissues. For some authors the term fascia is restricted to muscular connective tissues. Visceral connective tissues – no matter whether they are of loose composition like the major omentum or more ligamentous like the mediastinum – are often excluded. In contrast, more clinically oriented books have placed a lot of emphasis on the visceral fasciae (Paoletti 2006, Schwind 2006).

As valuable as these proposed anatomical distinctions within soft connective tissues are, their very detail may lead to unwitting exclusion of important tissue continuities which are only perceived on the larger scale. For example, the clinical significance

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of the continuity of the fascia of the scalene muscles of the neck with the pericardium and mediastinum inside the thorax is often surprising in our discussions with orthopedic surgeons, although less so to osteopaths or general surgeons. Figure 0.2 shows another example of perceptual tissue exclusion, based on terminological distinction. Here one of the sturdiest portions of the iliotibial tract has been excluded from this important tissue band, since it did not fit the distinct nomenclature defined by the authors of this paper.

Based on this background a more encompassing definition of the term fascia was recently proposed as a basis for the first Fascia Research Congress (Findley & Schleip 2007) and was further developed (Huijing & Langevin 2009) for the following congresses. The term fascia here describes the 'soft tissue component of the connective tissue system that permeates the human body'. One could also describe these as fibrous collagenous tissues which are part of a bodywide tensional force transmission system. This view of an interconnected tensional network is partly inspired by the tensegrity concept, as



Fig. 0.2 • Example of a fascia dissection based on specific terminology • This dissection was used in an otherwise excellent treatise on the iliotibial tract (ITT). Following the proposal of the Federative Committee on Anatomical Terminology (1998) to distinguish between aponeuroses and fasciae, the authors chose to describe this tissue as an aponeurosis. Congruently with this decision, their dissection and illustration therefore excluded all tissue portions with a non aponeurotic character. Unfortunately this included one of the most dense and most important portions of the iliotibial tract: the connection to the lateral iliac crest, posterior of the anterior superior iliac spine. Notice the common thickening of the iliac crest at the former attachment of this ligamentous portion (located at a straight force transmission line from the knee over the greater trochanter), reflecting the very strong pull of this tissue portion on the pelvis. (TFL: tensor fascia lata.) Illustration taken with permission from Benjamin et al 2008)

described in Chapter 3.5. The complete fascial net then includes not only dense planar tissue sheets (like septa, joint capsules, aponeuroses, organ capsules, or retinacula), which may also be called "proper fascia", but it also encompasses local densifications of this network in the form of ligaments and tendons. Additionally it includes softer collagenous connective tissues like the superficial fascia or the innermost intramuscular layer of the endomysium. The cutis, a derivative of the ectoderm, as well as cartilage and bones are not included as parts of the fascial tensional network. However, the term fascia now includes the dura mater, the periosteum, perineurium, the fibrous capsular layer of vertebral discs, organ capsules as well as bronchial connective tissue and the mesentery of the abdomen (Fig. 0.3).

This more encompassing terminology offers many important advantages for the field. Rather than having to draw most often arbitrary demarcation lines between joint capsules and their intimately involved ligaments and tendons (as well as interconnected





Fig. 0.3 • Different connective tissues considered here as fascial tissues • Fascial tissues differ in terms of their density and directional alignment of collagen fibers. For example, superficial fascia is characterized by a loose density and a mostly multidirectional or irregular fiber alignment; whereas in the denser tendons or ligaments the fibers are mostly unidirectional. Note that the intramuscular fasciae septi, perimysium and endomysium - may express varying degrees of directionality and density. The same is true although to a much larger degree - for the visceral fasciae (including soft tissues like the omentum majus and tougher sheets like the pericardium). Depending on local loading history, proper fasciae can express a two-directional or multidirectional arrangement. Not shown here are retinaculae and joint capsules, whose local properties may vary between those of ligaments, aponeuroses and proper fasciae.

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aponeuroses, retinacula and intramuscular fasciae), fascial tissues are seen as one interconnected tensional network that adapts its fiber arrangement and density according to local tensional demands. This terminology fits nicely to the Latin root of the term "fascia" (bundle, strap, bandage, binding together). It is also synonymous with the nonprofessional's understanding of the term "connective tissue". "Connective tissue research" is too broad a term, as this includes bones, cartilage and even blood or lymph, all of which are derivatives of the embryologic mesenchyme. In addition, the contemporary field of 'connective tissue research' has shifted its primary focus to tiny molecular dynamics from the macroscopic considerations of several decades ago. The newly forming field of fascia research requires both macroscopic and microscopic investigations. This text has undertaken the task of serving both areas. Even if sometimes microscopic details of collagenous tissues are explored, an effort will be made to always relate these findings to the body as a whole.

While we see great advantages in our wider definition of fascial tissues, we acknowledge that more traditionally oriented authors will continue to restrict the term fascia to dense planar layers of "irregular" connective tissues, in distinction from more regular oriented tissues like aponeuroses or ligaments. In some areas such a distinction is indeed possible and may be clinically useful (e.g. at the fasciae and aponeuroses of the lumbar region). We therefore suggest including twelve additional specifying terms wherever possible, into the detailed description of a fascial tissue. These specifying terms were proposed by Huijing & Langevin (2009): dense connective tissue, areolar connective tissue, superficial fascia, deep fascia, intermuscular septa, interosseal membrane, periost, neurovascular tract, epimysium, intra- and extramuscular aponeurosis, endomysium. However, we also note that many important areas of the body are characterized by gradual transitions between such morphological categories, and a more geometrical description of local collagen architecture (in terms of dominant fiber directions, tissue thickness and density) may then be more useful to understanding specific tissue properties (see Fig. 0.2).

This textbook, as have the fascia congresses, has taken the difficult role of being oriented toward both the scientist and the clinician. Material presented spans anatomy and physiology of fascia in Part 1, through clinical conditions and therapies in Part 2, to recently developed research techniques in Part 3. We have pointed out the definitional struggles the researcher faces surrounding fascia: Which tissue? What fiber directions? What is connected to what? These research tools will allow the extension of this debate to more clinical areas as well, to help define which tissues are affected and what directions forces are applied in the clinical therapies. It is our hope that clinicians and scientists, both together and separately, will rise to these challenges to advance our basic understanding and our clinical treatment of fascia.

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General anatomy of the muscle fasciae

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Introduction

The soft connective tissues associated with muscle tissue can be referred to as muscle fasciae (MF). A recent description of MF structure and function in both skeletal muscle and cardiac muscle (Purslow 2008) lists 15 previous reviews in addition to a great deal of original source material, and the reader is referred to these substantial sources for detailed information. Here we shall summarize the main features of their structure, composition, and functional properties. There is a tendency in previous literature to call MF "tubes" or "sheaths" that surround each fiber or fasciculus. The concept of muscle fasciae as connective tissue structures provides a better understanding of the functional anatomy of these structures. MF form a three-dimensional matrix that is continuous throughout the entire organ, providing connections between fibers and fascicles, rather than separating them.

General structure and composition of muscle fasciae

The following description of the general structure of fasciae associated with striated muscles summarizes the consensus of a number of sources and is schematically shown in Fig. 1.1.1.

Each individual muscle is surrounded by the **epimysium**, a connective tissue layer that is continuous with the tendons attaching the muscle to the bones. The **perimysium** is a continuous network of connective tissue, which divides the muscle up into fascicles or muscle fiber bundles. The perimysial network merges into the tendons and into the epimysium at the surface of the muscle, and is mechanically connected to them. Within each fascicle or muscle fiber bundle, the **endomysium** is a continuous network of connective tissue that separates individual muscle fibers.

Generally speaking, these connective tissue layers are composed of collagen fibers (and occasionally also elastin fibers) in an amorphous matrix of hydrated proteoglycans (PGs), which mechanically links the collagen fiber networks in these structures. Listrat et al. (2000) and Passerieux et al. (2006) identified seven molecular types of collagen in muscle (types I, III, IV, V, VI, XII and XIV). Types I, III and V are fibrillar collagens (fiber-forming types). Types I and III are the most prevalent in mammalian striated muscle. Types XII and XIV are thought to act as molecular bridges connecting the fibrillar collagens to other components in the amorphous matrix. The basement membrane layer of the muscle fibers contains non-fibrous type IV collagen, together with proteoglycan components such as laminin and fibronectin and heparin sulfate-containing PGs, and forms the boundary between the phospholipid cell membrane and the collagen fiber networks of the "reticular layer" of the endomysium.

Collagen fibers are mechanically stabilized by the formation of covalent crosslinks. The formation of crosslinks is essential for the mechanical strength and stiffness of collagen fibers, as without them the collagen molecules slide past each other under load and the fibers have no strength. Throughout gestation and during postnatal maturation there are



Fig. 1.1.1 • Schematic depiction of the fasciae associated with a skeletal muscle showing epimysium defining the surface of the muscle, perimysium separating muscle fascicles, endomysium separating muscle fibers, and the contractile myofibrils within each muscle fiber. Courtesy of Dr Loong Tak Lim.

substantial changes in the types and amounts of covalent crosslinks that mechanically stabilize the collagen molecules in muscle fasciae. The subject of crosslink formation during maturation and aging of connective tissues is reviewed in excellent detail by Avery & Bailey (2008). Here, we shall just note that muscle fasciae are rich in covalent crosslinks, that these crosslinks are known to undergo maturation changes in both endomysial and perimysial connective tissue, and that compounds promoting glycation crosslinks can be incorporated into the body from dietary sources and from tobacco smoke. Thus, diet and lifestyle may conceivably affect the mechanical properties of some connective tissues, including muscle fasciae, via crosslinking of collagens.

The amounts and composition of muscle fasciae vary between different muscles in the body. A comparison of transverse sections through different muscles from the same species (Purslow 2005, Fig. 1.1.2) shows that the continuous perimysial network surrounds or separates fascicles of very different sizes and shapes in different muscles. This difference also results in different thicknesses of perimysial connective tissue. These variations, especially in the amount and spatial organization of the perimysium, have long been attributed to variations in mechanical roles of different anatomical muscles. If this is correct, then the muscle fasciae must play strong roles in the normal physiological functioning of each muscle. Some possible explanations of these roles are emerging but are far from complete.



Fig. 1.1.2 • Scanning electron micrograph of endomysial structures remaining after removal of the myofibrillar contents of muscle cells. The endomysium forms a continuous lattice connecting all the muscle fibers in the fascicle.

Functional anatomy of the endomysium

There are three distinct structures separating the surface of one muscle fiber (cell) from its adjacent neighboring fiber:

- 1. At the surface of the fiber is the plasma membrane (plasmalemma) of the muscle cell, which is approximately 9 nm thick.
- **2.** Outside the plasma membrane is the endomysial basement membrane. It is approximately 50–70 nm thick and is composed of two layers: the lamina lucida (or lamina rara) next to the plasma membrane, and an outer lamina densa.

Each muscle fiber has its own plasmalemma and basement membrane surrounding it. Filling the space between the basement membranes of two adjacent muscle fibers is the third layer:

3. The collagen fiber network (or reticular) layer, comprised of a network of collagen fibrils and fibers in a proteoglycan matrix. Schmalbruch (1974) reported reticular layer thicknesses of $0.2-1.0 \ \mu m$ in frog sartorius muscle.

As shown by classical transmission electron microscopy images of longitudinal muscle sections (Trotter & Purslow 1992, Fig. 1.1.2), the thickness of the endomysium varies with muscle length, becoming thicker at short muscle lengths and thinner as the muscle is extended. The fibrous reticular layer is a common structure shared between adjacent muscle cells and forms a continuous network that runs across the whole muscle fascicle. Muscle cells (with their individual plasma membranes and basement membranes) occupy the polygonal "holes" in the endomysial network.

The reticular region of the endomysium is often described as a random or quasi-random network of irregularly wavy fine collagen fibers. The network is not truly random. There is a preferred direction in the wide distribution of collagen fiber orientations, and this preferred orientation changes with muscle length (Purslow & Trotter 1994).

A large number of muscles in animals from many phyla contain muscle fibers which are not continuous along the entire length of fascicles, from tendon to tendon. Trotter (1993) lists 28 studies on a wide range of muscles from humans, mammals and birds showing series-fibered architecture. Muscle fibers in series-fibered muscles are relatively short compared to the length of the fascicle, particularly so in avian species where individual fibers can be as short as 0.4-2.6 cm. The endomysium is the only structure that links these muscle fibers together in the fascicle. Transmission of tension generated in intrafascicularly terminating fibers to the ends of the fascicles necessitates transmission of force through the endomysial network, as this is the only structure continuously linking the fibers. The endomysium is very compliant to tensile forces acting within the plane of the network and so can easily deform to follow the length and diameter changes of muscle fibers in contracting and relaxing muscles. However, the transmission of force between adjacent muscle fibers by shear through the thickness of the endomysium (translaminar shear) is a different property, and one that turns out to be an efficient force transduction pathway (Trotter & Purslow 1992; Purslow & Trotter 1994; Trotter et al. 1995). Any linkage that transmits force from intrafascicularly terminating muscle fibers to tendinous attachments must not deform too much in order to be efficient. Especially in isometric muscle contractions, any significant stretching in the length of the fascicle due to stretchy connections would result in a very poor transmission of contractile force. Purslow (2002) showed that the displacements along the long axis of

the muscle due to translaminar shearing of the endomysium are insignificant. The functional significance of this is that the endomysium provides a shear linkage of force from one muscle cell to its neighbors which is highly efficient while still being able to deform easily in the plane of the network so as to allow the muscle fibers to change length and diameter as they contract and relax.

The functional anatomy of the endomysium linking adjacent muscle fibers in continuous fibered muscles appears identical to that of endomysium in series fibered muscles. The obvious inference is that load sharing between adjacent muscle cells is a common function in both continuous-fibered and series-fibered striated muscles and even cardiac muscle (Purslow 2008). The endomysium, therefore, forms a continuous three-dimensional connecting matrix which tightly shear-links adjacent fibers together to coordinate force transmission in a fascicle and keep fibers in uniform register.

Functional anatomy of the perimysium

The amounts and spatial distribution of perimysium vary much more between muscles in the body than do those of endomysium. Using two pennate muscles from the cow and the rat, Passerieux et al. (2007, see Fig. 1.1.3) showed that the perimysium is a well-ordered structure that lies throughout the muscles. Thick amounts of perimysium enclosing large fascicles of myofibers form tubes in a honeycomb arrangement in the direction of myofibers, the walls of the tubes in continuity with tendons at their ends and in continuity with epimysium at the outer surface of the muscle. The walls of the tubes are made of two (or even more) flat layers of long wavy collagen fibers running in the same direction in each layer. The direction of collagen fibers from each layer crosses the direction of myofibers at $\pm 55^{\circ}$ and crosses the fibers from the adjacent layer at an angle of about 120° . In addition, the long collagen fibers can overlap between adjacent walls of tubes so that the assembly of tubes is a very coherent structure. Many of the collagen fibers from layers spread out of the tubes as wide flat cables dividing the content of the tubes into secondary fascicles of myofibers, then separate successively as thinner cables giving smaller fascicles (this is possible in the case of the muscle of the cow because the collagen fibers are up to 5 cm in length, with a



Fig. 1.1.3 • View of one PJP (junction of terminal branches of a perimysial cable on the top with the surface of one myofiber at the sarcomeric level). Note the presence of some endomysium at the bottom of the myofiber. Bovine muscle; scanning electron microscopy; bar = $100 \mu m$.

straight 3 cm portion into the walls of tubes and a curved 2 cm portion into the fascicles of myofibers. At the end of the process, small cables join the surface of the myofibers so that the small cables form a rather regular network of long collagen fibers (Passerieux et al. 2007, see Fig. 1.1.3) and rather regular numerous contacts with each myofiber.

The perimysial layers separating two fascicles are comprised of two or more crossed-plies of wavy collagen fibers in a proteoglycan matrix. The long axis of each set of collagen fibers lies at \pm 55° to the longitudinal axis of the fascicle when the muscle is at its relaxed (resting) length. This angle increases as muscle shortens and decreases if it is passively stretched out. The waviness of the collagen fiber bundles also changes with muscle length, being maximal at the resting length of relaxed muscle. Perimysium is easily deformed in tension and does not exhibit a high tensile stiffness until it has been stretched far enough that the collagen fibers have become aligned along the stretching direction and the waviness in the fibers pulled out straight (Lewis & Purslow 1989). Thus, the perimysium can show a high tensile stiffness and carry large loads in tension, but only at very large extensions well beyond the range of working lengths in living muscle.

The tensile properties of the perimysium are therefore similar in nature to the endomysium and it is tempting to suppose that the perimysium could also act to transmit the forces generated in fascicles to their adjacent neighbors by translaminar shear. Although it is undoubtedly the case that force transmission by such a mechanism can be invoked in extreme circumstances of muscle damage or surgical disconnection of the tendinous attachments to some fascicles, there are two considerations that weigh against this mechanism under normal working conditions in living muscle. First, detailed analysis (Purslow 2002) shows that simply because perimysium is so much thicker than endomysium, deformations caused by shear through its thickness would be of orders of magnitude greater than in the endomysium, and so perimysium would represent a rather sloppy and inefficient force transmission pathway at physiologically-relevant muscle lengths. Second, why should perimysial content and architecture vary so much more than the endomysium if it is fulfilling the same kind of functional role?

Schmalbruch (1985) cites an old model by Feneis (1935) supposing that perimysial structures provide "neutral" connections between muscle fascicles which allowing muscle the fascicles to slide past each other, as the geometry of a muscle changes upon contraction. Measurements of "borders" between fascicles in ultrasonic images of human muscles in clinical and sports studies and their rotation on contraction allow these shear strains to be estimated. Using the values in the literature from seven such clinical studies (Purslow 2002), it is possible to show that shear strains within actively contracting human muscles are substantial and vary considerably between quadriceps, vastus lateralis, gastrocnemius and tibialis muscles. The theory that division of muscle into fascicles facilitates shear deformations explains why fascicle shape and size vary so much from muscle to muscle. However, until detailed quantitative assessment of any relationship between perimysial architecture, fascicle size and the distributions of shear strains in working muscles has been carried out, this theory remains just an interesting possibility.

Perimysial–endomysial junction zones

The endomysium surrounding muscle fibers is connected to the perimysium by intermittent perimysial junctional plates (PJPs), described by Passerieux et al. (2006).

If, like the endomysial network, the perimysium principally acts to transmit muscle force, then the perimysial-endomysial junction must necessarily be mechanically strong and noncompliant. Alternatively, if the perimysium has only a limited role in myofascial force transmission under normal physiological conditions, but is more involved in relieving shear displacements between fascicles during muscle contractions, then the connections could be expected to be more tenuous.

PJPs are staggered at the surface of each myofiber and separated by a distance of approximately $300 \ \mu m$. They are made of a set of branches of collagen fibers at the end of cables (Fig. 1.1.3), which arise from the tubes separating fascicles. The branches cross the perimysial layer of myofibers and reach their surface on the top and between costameric structures, with attachments on the reticular work of perimysium and the basement membrane of myofibers.

Fracture procedures (as seen on Fig. 1.1.3) show that the perimysium remains present only in the regions where perimysial plexi are attached to myofibers, which leads to the conclusion that these points of attachment between perimysium and myofibers are rather strong and therefore allow transmission of contractile force in synergy with the endomysium. However, the long dimensions of these collagen fibers suggest that the perimysium is stressed after the endomysium and acts under large intramuscular displacements or eccentric contractions produced during downhill exercises.

Perimysium and intracellular subdomains

Among the main cytoplasmic components of myofibers, nuclei and mitochondria are of importance because they control, respectively, metabolism and energetic production of myofibers, and it can be considered that their position in myofibers is of particular interest. Regarding nuclei, it was thought that they are distributed at the same distance along



Fig. 1.1.4 • View of a PJP-associated intracellular subdomain. Perimysium as thin filaments at the borders of the myofiber is in the vicinity of a nucleus and a large subsarcolemmal accumulation of mitochondria. Rat muscle; transmission electron microscopy; bar = 1 μ m.

the myofibers, each of them at the control of a surrounding "myonuclear domain". However, Roy et al. (1999) found that nuclei have a clustered distribution along the myofibers and the number of nuclei in clusters varies with exercise. The case of mitochondria is somewhat different: they are distributed with a great regularity at the level of sarcomers except for large subsarcolemmal accumulations. Nuclei and subsarcolemmal accumulations of mitochondria are linked to the cytoskeleton in regions where myofibers are crossed by capillaries (Ralston et al. 2006) which are embedded into the perimysium, and Passerieux et al. (2006) found that they are statistically co-localized with PJPs (Fig. 1.1.4).

In addition, the lack of the collagen VI component of perimysium is associated with apoptosis of nuclei and mitochondria of myofibers (Irwin et al. 2003) so it can be expected that the terminal branches of perimysial cables play an important role in mechanotransduction when they are stressed under myofiber contraction.

Conclusions

Muscle fasciae are important to the functioning of muscle tissues. Load transmission between tightlylinked adjacent muscle fibers within fascicles allows for coordination of forces, protection of damaged areas of fibers against over-extension and, in series-fibered muscle at the very least, is a major pathway for the transmission of contractile force. Substantial evidence exists to show that perimysium and epimysium can also act as pathways for myofascial force transmission. However, definition of boundaries between muscle fascicles by the perimysium may also have a role in allowing the whole tissue to accommodate large shear displacements. As detailed elsewhere in this book, muscle fasciae are in a continuous dynamic balance between synthesis and remodeling so as to be continually adapted for their mechanical roles in working muscles.

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Somatic fascia

Frank H Willard

Global organization of fascia in the body

Overview of the organization of somatic fascia in the body

When we think about the somatic portion of the body, images of skeletal muscle, bones, and joints usually present to mind. However, none of these structures can suffer much direct contact without developing significant pathology. For protective reasons, all of the somatic structures are embedded in a matrix of soft connective tissue termed fascia the bandage or packing substance of the body. Muscles develop in a matrix of connective tissue such that the adult organ is surrounded by an epimysium, bone arises in a matrix of embryonic fascia termed mesenchyme, which in the adult form becomes the periosteum, and joint capsules consolidate out of a thickening in mesenchyme (Gardner 1963) that ultimately forms a fascial covering over the dense layers of the capsule. In each case, the fascial sheet embracing the somatic structure protects it from direct abrasion by surrounding structures while also providing a conduit through which neurovascular bundles can easily penetrate. By surrounding the components of the somatic system, fascia creates complex and continuous planes or sheets of connective tissue that unite all portions of the body and present continuous planes along which anatomists tend to dissect (Huber 1930).

The functions of fascia tend to dictate its structure. Fascia must be capable of significant distortion in multiple planes of direction and return rapidly to its native shape. This type of action is best met by constructing fascia out of irregular connective tissue where the fibrous component is interwoven; thus proper fascia is defined as connective tissue with an irregular distribution of fibrous elements as opposed to those tissues containing parallel arrays such as are seen in tendons, ligaments, aponeuroses and joint capsules (Clemente 1985; Standring 2008). The irregular weave of the fibrous component allows for movement and resistance in all directions but is master of none. Thus, tearing fascia apart can be difficult in all planes of dissection. Conversely, due to the highly regular arrangement of collagen fibers in a tendon, ligament, or aponeurosis, these structures can provide maximal resistance of stretch in one or a limited number of directions but can easily be shredded with finger tips when stressed in orthogonal planes.

The density of the fibrous component of fascia will vary tremendously with is location and function. Thus, fascia underlying the skin must be very movable and therefore has a lower density of collagenous fibers; this is often given the ambiguous term *superficial fascia* (Singer 1935; Clemente 1985; Standring 2008). Alternatively, the fascia that invests muscle, ligament, tendon, or joint capsule is providing a stronger support role and is often termed *investing fascia*, the density of its collagen fibers being considerably higher; however, they are still irregular in weave (Singer 1935; Clemente 1985; Standring 2008).

Finally, unlike the highly differentiated structures of the somatic system – muscle, tendon, ligament, and aponeurosis - fascial planes tend to lack precise borders. Muscles have fairly recognizable origins and attachments, and where they joint with tendons a precise line can be seen even in the microstructure. Attachments of muscle-tendon complexes to bone are definitive, forming an enthesis. However, the lack of precise borders seen in the fascial tissue facilitates the formation of long planes spanning multiple organ systems or compartments surrounding multiple muscles. When entering fascial compartments, neurovascular bundles - which themselves are surrounded by irregular, dense, connective tissue fascial wrappings course along or through fascial planes that would otherwise represent obstacles if composed of highly organized, regularly arranged fibrous elements such as seen in an aponeurosis, tendon, ligament, or joint capsule. Lymphatic flow, which would be quickly interrupted if forced through tissue with precise boundaries, can flow easily through lymphatic vessels distributed in the irregular tissue of the fascial plane. From this discussion it is evident that the function of fascia in the somatic body is closely associated with its structure.

Architecture of fascia – the four primary layers

General approach

Several attempts at describing the fasciae of the body have been published (Gallaudet 1931; Singer 1935; Benjamin 2009). This chapter focuses on irregular connective tissue or "proper fascia" and will describe a system of four primary layers that cover the axial portion of the body. Modification of this fundamental plan will allow accommodation of the limbs.

The four primary layers in the torso are arranged as a series of concentric tubes (Plate 1.2.1). Starting with the outermost layer of fascia, it is best termed the panniculus or panniculus adiposus: a term used by Singer (Singer 1935) in his treatise on fascia and strongly urged for general usage by Last (Last 1978) in his textbook of anatomy. Deep to the pannicular layer is the axial fascia of the torso. This layer gives rise to the investing fascia or epimysium of the axial muscles, peridentium and periligamentum of tendons and ligaments, and the periosteum of bone. The axial layer of fascia is continuous with the appendicular (investing) fascia in the extremity. As with the pannicular layer, the axial layer can be subdivided; however, again, in this chapter it will be treated as a primary layer. Internal to the axial fascia are two additional layers: the first surrounds the neural structures and can be termed **meningeal fascia** and the second surrounds all body cavities and is best termed **visceral (splanchnic) fascia**. In considering the limbs, the pannicular layer extends outward covering the entire surface of the limb. Under the pannicular layer, a fascial layer of similar composition to the axial fascia is present surrounding the muscles of the extremity, and can be termed appendicular fascia. It lies deep to the pannicular fascia and invests the appendicular muscles. Regional names often relate the fascia to a specific muscle, i.e., deltoid fascia, pectoral fascia, etc. Internal to the appendicular fascia is the intramuscular septum housing the neurovascular bundles; this septal layer is most likely to be derived from the axial fascia at the base of the limb.

Four primary layers of fascia

Pannicular fascia

The outermost layer is the pannicular fascia (Singer 1935) and is often termed superficial fascia (Clemente 1985; Standring 2008). This layer can be subdivided into several sublayers as outlined in Chapter 1.3. The pannicular layer is derived from the somatic mesenchyme and surrounds the entire body, with the exception of its orifices such as the orbits, nasal passages, and the oral and aboral openings. It is composed of irregular connective tissue with marked regional variation in collagen fiber density as well as variation in adipose cell density (Fig. 1.2.1). While the outermost portion of this layer is typically invaded by much adipose tissue, the inner portion is more membranous in nature and generally very adherent to the outer portion, except over the abdomen where the two can be easily separated by blunt dissection. The thickness of the pannicular layer is highly variable in the human population. In the region of the head and neck, humans have several thin muscles embedded in the pannicular fascia; these are the platysma and associated facial muscles innervated by the facial nerve. Pannicular fascia covers both the axial and appendicular body.

Axial fascia

The second layer is the axial or investing fascia (deep fascia, as described in Clemente (1985) and Standring (2008)). Axial fascia is fused to the panniculus peripherally and extends deep into the body, surrounding the hypaxial and epaxial muscles. This layer, like the pannicular layer, is derived from mesenchyme and forms the primitive matrix in which

Fig. 1.2.1 • The pannicular layer of fascia. This is an anterior view of the thorax and abdomen of a male and female cadaver. The body on the left (A) is that of a 54-year-old male and on the right (B) a 54-year-old female. Both specimens have had the dermis removed to reveal the pannicular layer of fascia. From the Willard/Carreiro Collection, with permission.



skeletal muscles, tendons, ligaments, aponeuroses, and joints develop. The mesenchymal matrix then contributes to the epimysium of skeletal muscle, the periosteum of bone, the peritendon of the tendons, as well as the investing layer surrounding the joint capsule. The peritendon subdivides into an epitenon, which grasps the regular collagenous fiber bundles of the tendon, and a paratenon, which surrounds the entire tendon; both layers form the peritendon and are constructed of irregular collagenous bundles (Jozsa & Kannus 1997). The arrangement of fascia around an aponeurosis is similar to that of a tendon or ligament; however, the terminology used has created some confusion. In the older literature. two terms exist: "aponeuroses of attachment" and "aponeuroses of investment" (Singer 1935). Aponeuroses of attachment referred to the well-organized bands of dense connective tissue that made up the true aponeuroses that attached the muscle to its target; while those "of investment" made up the irregular connective tissue composing the investing or axial fascia surrounding the true aponeuroses.

The axial fascia can be described as being composed of two, parallel, connective tissue tubes and course anterior and posterior to the vertebral column (Fig. 1.2.2a–c). Developmentally, these two tubes would be separated by the notochord, which is approximated by the vertebral column in the adult. The anterior tube surrounds the hypaxial muscles and attaches to the vertebral column at the transverse process. The hypaxial muscles include the longus and scalene muscles in the cervical region, the intercostal muscles in the thoracic region, and the oblique and rectus muscles in the abdominal region. The posterior tube of the axial fascia surrounds the epaxial muscles and is attached to the transverse processes. The spinous process of each vertebra divides the epaxial fascial tube into two "half-tubes" (Fig. 1.2.2c). The paraspinal muscles of the back are contained in the "half-tubes" of the epaxial fascia.

Complex fascial relationships exist where the extremities meet to the axial portion of the body. Axial fascia extends into the extremities as the intermuscular septum and the appendicular fascia investing individual muscles (Fig. 1.2.3). The fascial sheath that surrounds the neurovascular bundles such as the brachial plexus and lumbosacral plexus extends outward to form the intermuscular septum, in which branches of the neurovascular bundle will course as they progress distally in the extremity. In the upper extremity, the axial fascia surrounding the brachial plexus is regionally termed the axillary sheath; however, it represents an extension of the axial fascia - specifically, it extends from a portion of the axial fascia regionally termed the "prevertebral fascia" (see Fig. 1.2.4). The use of the term "axial fascia" as a primary descriptor for all these layers places an emphasis on their common developmental origin and their similarity in microstructure as well as their structural continuity in the adult.

The arrangement of fasciae on the body wall is made very complex by the attachment of the limbs.



Fig. 1.2.2 • (A) An axial plane spiral CT taken through a male thorax. The white outline surrounding the axial muscles – hypaxial and epaxial – marks the course of the axial fascia. (B) Shading representing the hypaxial and epaxial muscle compartments taken from the section in A. (C) A diagram illustrating the epaxial and hypaxial fascial column. From the Willard/Carreiro Collection, with permission.

The muscles of the upper extremity, such as the pectoralis, trapezius, serratus anterior, and latissimus dorsi muscles, form long wing-like expansions that wrap over the torso to attach to the spinous processes on the midline of the body or to structures that ultimately attach to the midline such as the thoracolumbar fascia. These extremity muscles are passing internal to the panniculus of fascia and external to the axial fascia as they embrace the axial body wall. Each muscle is surrounded by a layer of investing or appendicular fascia. The arrangement of these fascia sheets can be seen in the two dissections



Fig. 1.2.3 • The fascia surrounding the neurovascular bundle in the axial portion of the body extends outward to the extremity. From L. H. Mathers, et al, 1996, with permission.

illustrated in Figs 1.2.3 and 1.2.4. In Fig. 1.2.4(a) the epidermis and dermis have been removed to expose the underlying pannicular fascia. An elongated rectangular window (outline) was opened into the pannicular fascia to expose the investing fascia (appendicular fascia) of the pectoral muscles. In Fig. 1.2.4(b) the window was enlarged and divided into two portions; the medial portion still has appendicular fascia covering the muscle but in the lateral portion the investing appendicular fascia has been removed to expose the pectoralis muscle. With the inferolateral border of the pectoral muscle elevated (Fig. 1.2.4 inset), the investing (appendicular) fascia separating it from the serratus anterior can be visualized. To expose the axial investing fascia of the intercostal muscles, the pectoral major and serratus anterior muscles and their investing appendicular fascia must be removed (Fig. 1.2.5a). In this figure, the pectoralis major and serratus anterior muscles have been removed to expose the intercostal muscles and their thin layer of axial investing fascia (note that the pectoralis minor muscle is still attached). Finally, Fig. 1.2.5(b) exposes the intercostal muscles by carefully dissecting away all axial fascia.

Meningeal fascia

The third fascial layer is meningeal fascia, which surrounds the nervous system. This layer includes the dura as well as the underlying leptomeninges; it derives from the primitive meninx that surrounds the embryonic nervous system. Specifically, the spinal meninges are most likely derived from the somatic mesoderm, while the brainstem meninges arise from cephalic mesoderm and the telencephalic meninges from the neural crest (Catala 1998). Meningeal fascia terminates with the development of the epineurium that surrounds the peripheral nerve.

Visceral fascia

The fourth fascial layer is visceral fascia and is by far the most complex of the four main layers of fascia. Embryologically, this layer of fascia is derived from the splanchnic tissue and thus surrounds the body cavities – pleural, pericardial, and peritoneal. The visceral layer follows the visceral pleura and peritoneum and provides the conduit for neurovascular bundles entering the visceral organs as well as a drainage route out of the organ. On the midline of the body, the visceral fascia forms a thickened mediastinum that extends from the cranial base into the pelvic cavity. This layer of fascia will be discussed in a separate chapter.

Summary

The term fascia represents a form of connective tissue that is widely distributed throughout the body and composed of irregular, interwoven collagenous fiber bundles of varying density. Fascia plays multiple roles in the body; it invests most structural elements, being highly protective in nature, and can also provide a lubricating function. Its network of interconnections between skeletal elements appears to provide a mechanism for limited force transduction and its cellular composition strongly suggest both an immune function and a neurosensory role as well.

Although many regional names exist for specific fascia, this chapter has attempted to present only four primary layers of fascia for consideration. The outermost or **pannicular layer** (superficial fascia) is mainly composed of loose connective tissue and fat and surrounds the entire torso and extremities except over the exposed orifices. Next there is a complex arrangement of **axial fascia** (deep or investing fascia) composed of denser irregular connective tissue investing muscles, tendons, ligaments, and aponeuroses. Axial fascia also extends into the extremities (appendicular fascia), where it has similar composition and function to its axial counterpart. This network of fascia provides protection and lubrication



Fig. 1.2.4 • (A) Photograph of a male with the epidermis and dermis removed to expose the pannicular fascia on the chest wall. A vertical window has been cut in the pannicular fascia to expose the underlying appendicular fascia. (B) The same specimen with the appendicular fascia cleaned off the pectoralis major and serratus anterior muscles in the right side of the window. In the inset (lower middle) the lateral margin of the pectoralis muscle has been elevated to expose the appendicular fascia that separates the pectoralis major from that of the serratus anterior. Elevating the serratus would reveal a fusion of appendicular fascia on the inner aspect of the serratus and axial fascia on the underlying intercostal muscles and ribs. From the Willard/Carreiro Collection, with permission.



Fig. 1.2.5 • Anterior views of the thoracic wall of a 54-year-old female. In (A) the axial (investing) fascia of the thoracic wall is present and it is difficult to visualize the underlying structures. In (B) the axial fascia has been removed to reveal the underlying intercostal muscles and the ribs. From the Willard/Carreiro Collection, with permission.

Somatic fascia

for the elements of the musculoskeletal system and most likely also transmits some force transduction during muscle contraction. The axial fascia is arranged in two sleeves or tubes separated by the vertebral column, from which extensions into appendicular fascia occur. The final two layers are encased within the axial fascia: these are the meningeal and visceral fascia, both specializations designed to protect the nervous system and the visceral organs, respectively. Limiting the presentation to the primary layers rather than their subcomponents helps emphasize the continuity of fascial planes in the body.

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Fascia superficialis

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Introduction

Skin, comprising epidermis and dermis, covers the whole surface of the body and is its largest organ. Immediately subjacent is an enveloping layer of dense and areolar connective tissue and fat called the superficial fascia (synonyms – fascia superficialis, hypodermis, subcutaneous tissue, tela subcutanea) (Standring et al. 2008; Langevin & Huijing 2009). Being co-extensive with the skin, it too comprises a very considerable tissue mass that conveys blood vessels and nerves to and from the integument.

The subcutaneous tissue connects the skin to the underlying dense deep fascia which invests muscles and aponeuroses throughout the body. Skin, with the subjacent superficial fascia, provides a protective cushion for the musculoskeletal framework over which they slide. Sheets of collagen fibers coupled with elastin facilitate this mobility (Kawamata et al. 2003). The spaces between the collagen sheets facilitate sliding, whilst stretching results in realignment of collagen fibers within the sheet. Skin shape and position are restored by elastic recoil. The tortuosity of blood vessels and nerves through superficial fascia allows them to accommodate stretching.

Van der Wal (2009) considers fascia as a connective tissue continuum throughout the body. Thus subcutaneous connective tissue provides a unique general pathway through and between regions which blood vessels, nerves and lymphatics can traverse (Wood Jones 1946; Benjamin 2009).

Its widespread distribution, its mechanical role, and the ability of fibroblasts to communicate via their gap junctions, suggest fascia may form a bodywide mechanosensitive integrating signaling system analogous to that of the nervous system (Langevin 2006) and relevant to interpreting fibrositis.

Gross structure and distribution

Fiber bundles (retinaculae cutis) traverse the subcutaneous layer from dermis to deep fascia, strengthening their connection. The subcutaneous tissue can be differentiated into a bulky superficial fatty layer, *panniculus adiposus*, and a deeper, mostly vestigial layer, *panniculus carnosus* (McGrath et al. 2004). Within the fatty layer itself, a membranous component can also be recognized. Classically, this is seen in the lower abdominal wall as the fascia of Camper (superficial fatty layer) and of Scarpa (deeper membranous layer) (Standring et al. 2008). Recent studies (Abu-Hijleh et al. 2006) indicate that the membranous layer has a much wider distribution in the body than suggested by earlier studies (see below).

The fat cells are aggregated into clumps or lobules by fibrous septa. In the adult human the vast majority of the fat is of the white type, with only a very small amount of the energy-rich brown fat. About 20% of body weight in a healthy adult male is white fat and it comprises up to 25% in females. Subcutaneous fat accounts for about 50% of total fat storage in the body. The storage is dynamic, the fat itself within the cells being renewed every 2–3 weeks. The amount and distribution of the fat varies with age, sex, and site. In infants and young children its distribution is quite uniform through all regions (except for the suctorial pad in the cheek). It increases steadily in amounts throughout early childhood. In adults the number of fat cells remains constant, the actual number being established during childhood and adolescence and under genetic control. Nevertheless, it has been found that approximately 10% of fat cells are renewed annually in adults (Spalding et al. 2008). In old age there is a decrease in fat. Concerning the sexes, subcutaneous fat is thicker in females, as evidenced by their smoother body contours. Measurements of subcutaneous tissue in the anterolateral aspect of the thigh recorded a depth of 0.74 cm in males and 1.74 cm in females (Song et al. 2004). Using an objective assessment of limb subcutaneous tissue elasticity by an indentation technique, Zheng et al. (1999) noted that the tissue thickness increases by 26% when the underlying muscle contracts. With regard to site differences, fat is particularly thick in the buttocks, hips, waist, thighs, soles, palms, breast, and cheeks. It is thinnest or absent in evelids, lips, pinna







(excluding the lobule), external nose, penis, scrotum, and labia minora.

A distinct membranous layer in the superficial fascia in restricted areas of the body, including the lower anterior abdominal wall (Scarpa's fascia), and perineum (Colles' fascia) is well documented. A membranous layer is also present in relation to the saphenous veins (Caggiati 2000, 2001). More recently, a well-defined membranous sheet was found in the superficial fascia of many regions of the body (Abu-Hijleh et al. 2006; Fig. 1.3.1), its arrangement and thickness varying according to gender, region, and the surface studied. This sheet is more prominent on the posterior aspects of the trunk and extremities than on the anterior. In certain regions, such as the periphery of the female breast, arm, back, and thigh there may be more than one membranous layer separating the fat into two or more layers (Fig. 1.3.2).

> Fig. 1.3.1 • (A) Layered dissection of the male breast region (chest wall). The membranous layer (ml) forms a single continuous layer in this dissection and the corresponding ultrasonogram (B) where it is sharply demarcated from the surrounding hypoechogenic fatty tissue layer (fl). (C) Layered dissection of the anterior leg region. Two superficial veins are seen: a tributary vein (tv) lies superficial to the membranous layer, and the main (saphenous) vein (v) is enclosed in a compartment formed by the muscular fascia (mf) deeply and by the membranous layer (ml) superficially. These two fascial layers fuse peripherally, delineating a compartment resembling an "Equptian eye"-shape using ultrasonography (D). In (D), the main vein is anchored to the fibrous wall of its compartment by a connective tissue lamina (arrow). s, skin; m, muscle.

> Fig. 1.3.2 • (A) Ultrasonogram of the anterior thigh region. At least two membranous layers (ml1 & ml2) can be identified within the superficial fascia.
> (B) Histological appearance of an excised membranous layer (ml) between two fatty layers in the superficial fascia. fl, fatty tissue layer; s, skin; m, muscle.

The superficial fascia in the face differs from other regions. Macchi et al. (2010) have shown that the superficial fascia has a laminar connective tissue layer (the superficial musculoaponeurotic system, SMAS; Mitz & Peyronie 1976). It is interposed between superficial and deep fibroadipose layers. The superficial layer connects the dermis with the superficial aspect of the SMAS. The deep layer connects the deep aspect of SMAS to the parotid–masseteric fascia. Adipose lobules occur within both layers.

Components and their relation to function

- 1. Interstitial fluid: Subcutaneous tissue contains a rich plexus of lymphatics and lymphatic capillaries. There is a dynamic balance between production of tissue fluid from blood capillaries and its absorption into subcutaneous lymphatics. Drainage is assisted by contraction in underlying muscles. Normally there is a negative pressure of -2 or - 3 mmHg (Sven & Josipa 2007), which is influenced by external atmospheric pressure acting on the skin surface. Disturbances in the normal dynamics of tissue fluid formation may increase the pressure, fluid accumulating and resulting in edema, the distribution of which is influenced by posture. It may be shown by "pitting" of the skin in response to locally applied pressure. Conversely, fluid may be lost, as in severe dehydration.
- **2.** *Force absorption*: Subcutaneous mixtures of "fibroadipose tissue" function as important pressure absorbers withstanding compressive and shearing forces on the body surface (Benjamin 2009). The best examples are seen in the palm of the hand and sole of the foot, particularly the fat-pad of the heel where strong fibrous septa pass from the dermis deeply to the plantar aponeurosis, dividing the fat into clearly defined compact lobules.
- **3.** *Thermal insulation:* Subcutaneous fat insulates the body against heat loss. The greater the thickness of fat, the more effective is the insulation. This is particularly important in warm-blooded terrestrial animals.
- Energy source: Subcutaneous fat provides a very significant reserve of stored energy (Marks & Miller 2006). The source of energy is triglyceride, which contains oleic and palmitic acids present in

the fat droplets of the adipocytes. In severe starvation the body's subcutaneous fat is utilized, resulting in tissue shrinkage. Brown fat, which is more abundant in newborns, provides a rapid source of energy and heat.

5. Vascular arrangements: Superficial veins lying in the subcutaneous tissues may be quite large and easily visible, especially in limbs, where they are available for intravenous injection or transfusions. Studies on the saphenous veins in the lower extremities indicate that they lie interfascially rather than subfascial (Papadopoulos et al. 1981; Caggiati 2001). Each vein lies within a compartment delimited by two fasciae: the muscular fascia deeply and the membranous layer superficially. The two fuse peripherally, forming a deep compartment of the hypodermis resembling an "Egyptian eye" shape when viewed by ultrasonography (Fig. 1.3.1) (Caggiati 2000; Abu-Hijleh et al. 2006). Within their compartments the veins are anchored by a connective tissue lamina. Tributaries of the saphenous veins course superficial to the membranous layer in the subdermal fatty layer (Fig. 1.3.1) (Caggiati 2001; Abu-Hijleh et al. 2006), lacking fascial wrapping in contrast to the main veins. A similar arrangement exists along the cephalic vein and its tributaries in the upper extremity (Abu-Hijleh et al. 2006). There are also fascial canals in the fingers (Doyle 2003) containing digital vessels and nerves.

The fascial relationships of superficial veins, particularly the saphenous system, have important clinical implications for hemodynamics and the pathophysiology of varicosities. Muscular contraction stretches the membranous compartment in which the main vein lies, diminishing the vein's caliber and therefore blood flow. The interfascial course of the main vein could constrain its excessive dilatation, thus diminishing the risk of varicosities. In contrast, the absence of any fascial sheathing to the more superficial tributaries could explain why varicosities more commonly affect them, since they lie outside the saphenous compartment (Caggiati 2000; Abu-Hijleh et al. 2006).

Because it is a ready source of energy, subcutaneous fat has a very rich blood supply. It also plays a part in temperature regulation. Small arteries supply two plexuses (Young et al. 2006). The more superficial is the subpapillary plexus lying in the dermis close to the hypodermis. The second deeper plexus is the cutaneous plexus and it lies in the hypodermis. The two plexuses freely communicate. Their state of distension or constriction determines the skin temperature and skin color in light-skinned races. Marked pallor of the skin, which is seen in acute shock, results from vasoconstriction in the arterial plexuses in the hypodermis. The smaller veins accompany the arteries and are similarly arranged into two plexuses. This arrangement provides rich arteriovenous communications, allowing shunts to occur that control blood flow through the skin and are used in thermoregulation of body temperature.

- **6.** *Muscle*: In quadrupeds such as horses and cattle there are extensive sheets of subcutaneous muscle forming a distinct layer, the *panniculus carnosus*, in the deeper part of the hypodermis. This has a protective function, enabling the animal to dislodge skin irritants such as insects. In man this layer is only vestigial, with remnants of smooth muscle in the scrotum, penis, anus, nipples, and labia majora. In the face, subcutaneous striated muscles are more organized with a protective function, being arranged as sphincters and dilators around the orifices, particularly the eye and mouth. In the neck, the platysma muscle is usually well defined, extending across the mandible into the upper neck.
- 7. Fibers: Fibers control the biomechanical properties of subcutaneous tissue, particularly its tensile and elastic properties as reflected in the overlying skin. Three types of fibers are present in the hypodermis, principally collagen and elastin, but also reticulin, a type of collagen. Collagen provides tensile strength when the overlying skin is stretched. The principal form of collagen is type I but types III and V are also present. The fibers surround fat cells, grouping them into lobules. They also connect the dermis with underlying deep fascia. Elastic fibers are concerned with stretching and elastic recoil of the overlying dermis and epidermis. These fibers are in the form of a continuous network which contains mostly mature elastin (but also immature elaunin and oxytalan) fibers. In mechanical tests of skin and subcutaneous tissue, the response to uniaxial tension shows linear and viscoelastic characteristics (Iatridis et al. 2003).
- **8.** Cells: Excluding adipocytes which store fat and form the majority of cells, a very important group of cells in subcutaneous tissue are fibroblasts. Besides synthesizing the proteins that form collagen and elastin fibers and other matrix

proteins in subcutaneous tissue, they take part in degradation of collagen and other fibers. Fibroblasts are integral to mechanotransduction. They communicate with each other via gap junctions and respond to tissue stretch by shape changes which influence tension within the connective tissue, mediated via the cytoskeleton (Langevin 2006). These responses to mechanical load could result from changes in cell signaling and cell-matrix adhesion. Cell shape changes may influence tension within the connective tissue itself. Brief stretching decreases transforming growth factor-beta 1 fibrillogenesis, which may be pertinent to development of manual therapy techniques for reducing the risk of scarring/fibrosis after an injury (Langevin 2006; Benjamin 2009).

Macrophages, derived from blood monocytes, are significant components since they are phagocytic for cell debris and also process and present antigen to immuneresponsive lymphocytes that enter the hypodermis from blood capillaries. Other blood cells found in subcutaneous tissues are neutrophils. These also are protective and normally circulate through the interstitial fluid irrespective of participating in inflammatory responses.

Mast cells, which have some of the features of blood basophils in the appearance of their granules, are also present in subcutaneous tissue. They produce heparin, serotonin, and histamine, which act on small blood vessels affecting their permeability. They mediate immunoglobulin E (IgE)-dependent forms of inflammation. Finally, plasma cells form IgE in the hypodermis.

- **9.** *Extracellular matrix-ground substance*: The matrix contains glycoproteins (including fibrillin and fibronectin, which are necessary for the stretching and recoil properties of elastin, and fibronectin, which controls the deposition and orientation of collagen fibers), glycosaminoglycans and proteoglycans, also hyaluronic acid, chondroitin, and dermatan sulfates.
- **10.** Other components: The deeper parts of the coiled sweat glands extend down into the hypodermis. The roots of hair follicles are also located in the subcutaneous tissue.

Numerous nerve fibers traverse the subcutaneous tissue to reach the dermis and epidermis. Whilst most of the specialized and nonspecialized nerve endings terminate in the dermis, there is one specialized ending that is normally located in the hypodermis. This is the lamellated Pacinian corpuscle which mediates vibration and pressure sensations.

Aging changes in subcutaneous tissue

Age-changes in the hypodermis are reflected on the surface of the body in the appearance and properties of the skin. Wrinkles and creases appear with increasing age. A decrease in the number of fibroblasts is accompanied by a decrease in the number of collagen fibers, which also become disorganized. The fibers disrupt and lose shape. Elastic fibers also decrease and become misshapen, appearing thickened and frayed. Using an indentation technique and measuring rebound it was noted that in both males and females there was a progressive decrease in elastic recoil through succeeding decades, starting from the third decade (Kirk & Chieffi 1962). The relationship was nonlinear. The amount of fat gradually declines as the fat cells atrophy. Quantitative and qualitative characteristics of the fibroadipose connective system are changed and its viscoelastic properties become reduced. The skin and underlying superficial fascia relax and stretch, resulting in ptotic soft tissues, pseudo-fat deposit deformity, and cellulite (Lockwood 1991; Macchi et al. 2010). There is also atrophy of sweat and sebaceous glands, leading to drying of the overlying skin.

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Deep fascia of the shoulder and arm

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The deep fascia of the shoulder

The deep fasciae of the shoulder present characteristics that are similar to both the fasciae of the trunk and of the extremities. In particular, the fasciae of the pectoralis major, deltoid, trapezius, and latissimus dorsi muscles form a unique layer, enveloping all of these muscles and passing over the serratus anterior, where it forms a strong fascial lamina. This myofascial arrangement agrees with the description of the trunk as reported by Sato and Hashimoto (1984), who affirm that the pectoralis major, latissimus dorsi, and trapezius muscles form an additional myofascial layer with respect to the muscular planes in the rest of the trunk.

The fasciae of the pectoralis major, deltoid, trapezius, and latissimus dorsi muscles are relatively thin, collagen fiber layers. All of these fasciae adhere firmly to their respective muscles due to a series of intramuscular septa that extend from the internal surface of these fasciae, dividing the muscle itself into many bundles. A true epimysial fascia, or epimysium, is not discernible between this deep fascia layer and the underlying muscle. A number of muscular fibers originate from the inner side of these fasciae, as well as directly from the intramuscular septa.

The **pectoral fascia** originates from the clavicle, but only the deep layer of the pectoral fascia adheres to the clavicular periosteum, whereas its superficial layer continues upwards with the superficial lamina of the deep cervical fascia, which surrounds the sternocleidomastoid and the trapezius muscles. Medially, the deep layer of the pectoral fascia inserts into the sternal periosteum, while the superficial layer extends beyond the sternum to continue with the pectoral fascia on the other side. Distally, the pectoral fascia is reinforced by some fibrous expansions originating from the rectus abdominis sheath and by the fascia of the contralateral external oblique muscles. In particular, the pectoral fascia presents a mean thickness of 151 μ m, and it increases in a craniocaudal direction, to reach a mean thickness of 578 μ m in the mammary region. Over the xiphoid process, the pectoral fascia forms a clearly visible, interwoven pattern of fibers (Stecco et al. 2009).

The **deltoid fascia** appears to be of variable thickness from subject to subject, without any apparent correlation to the size of the underlying muscle mass. The fascia adheres strongly to the muscle and connects the different parts of the deltoid. According to Rispoli et al. (2009), it was consistently possible to distinguish the three portions of the deltoid muscle (anterior, lateral, and posterior), to a varying degree, with each portion continuing with the brachial fascia. The deltoid fascia continues with the fascia covering the trapezius muscle. In particular, the superficial fascial layers are in continuity, while the deep layers insert into the scapular spine and clavicle, in continuity with the periosteum.

On histologic examination, the deltoid and pectoral fasciae appear to be formed by undulated collagen fibers, arranged more or less transversely with respect to the underlying muscles. An elevated number of elastic fibers are evident with van Gieson stain (approximately 15% of all the fibers), forming an irregular mesh. The S100 stain highlights rare nerve terminations, arranged in a homogeneous manner throughout the entire fasciae.

After detaching the superficial muscular layer, the **clavipectoral fascia** is visible. There is an ample plane of cleavage between the pectoralis major muscle and this fascia due to the presence of loose connective

tissue, which allows the deep layer of the pectoral fascia to glide autonomously with respect to the clavipectoral fascia. The latter is a strong connective layer arising from the clavicle and extending distally to enclose the subclavius and pectoralis minor muscles. Laterally, the clavipectoral fascia continues with the axillary fascia and the fascia of the coracobrachialis muscle. The clavipectoral fascia can be divided into two parts: one covering the pectoralis minor muscle and one that forms a triangular shaped layer between the upper border of this muscle and the clavicle, called the coracoclavicular fascia. The anterior thoracic artery and nerve, and the cephalic vein pierce the coracoclavicular fascia. Its thicker, lateral border, which extends from the coracoid process of the scapula to the cartilage of the first rib, is known as the costocoracoid ligament. It separates the cavity of the axilla from the anterior chest wall.

Singer (1935), describes the subscapularis fascia as being the thinnest of the different fasciae surrounding the muscles of the scapula; however, it is a well-defined lamina. Laterally, it continues with the axillary and infraspinatus fasciae, and superiorly, with the supraspinatus fascia.

The **infraspinatus fascia** covers the infraspinatus and teres major muscles. The deltoid and latissimus dorsi muscles cover part of this infraspinatus fascia, while only the fascia that joins the latissimus dorsi, trapezius, and deltoid muscles covers the part lying in a superficial plane. In this portion, the two fasciae adhere to each other, forming a strong fascial plate.

The **supraspinatus fascia** covers the supraspinatus muscle and continues with the fascia of the levator scapulae muscle. It varies in thickness, and occasionally contains some adipose tissue. According to Bektas et al. (2003), the spinoglenoid ligament, usually implicated in the compression of the suprascapular nerve, could be evidenced only in 15.6% of cases, while a thickening of the distal third of these two fasciae is always present. It is probable that this thickening could cause dynamic compression of the suprascapular nerve.

The **axillary fascia** is formed from the union of the superficial fascia and the deep fascia. It continues laterally with the superficial fascia of the arm and the brachial fascia, medially with the pectoralis major fascia and the coracoclavicular fascia, and posteriorly with the fasciae of the latissimus dorsi and subscapularis muscles. The axillary fascia contains numerous lymph nodes and is pierced by numerous nerves and vessels. Hence, it quite similar to the cribriform fascia of the thigh and, likewise, it is filled with a plug of fibrous tissue and fat.

The deep fascia of the arm

The brachial fascia and the antebrachial fascia form the deep fasciae of the arm. The superficial fascia in the arm is clearly evident within the subcutaneous adipose tissue, and it is easily detached from the deep fascia.

The brachial fascia is a strong, semitransparent laminar sheet of connective tissue that covers the arm muscles. It presents a mean thickness of 863 μ m (SD \pm 77 μ m), being thinner in the anterior region as compared to the posterior region. Collagen fiber bundles, with different directions, are easily identifiable within this fascia. They exhibit a prevalently transverse course with respect to the long axis of the arm, although longitudinal and oblique collagen bundles are present. The brachial fascia is easily separable from the underlying muscles, while it attaches to the lateral and medial intermuscular septa and the epicondyles (Plate 1.4.1). Proximally it is continuous with the axillary fascia, and the fasciae of the pectoralis major, deltoid, and latissimus dorsi muscles.

The antebrachial fascia appears as a thick, whitish layer of connective tissue, sheathing the flexor and extensor muscles compartments and extending septa between them from its internal surface. The mean thickness of the antebrachial fascia is 0.75 mm, yet this increases (mean value 1.19 mm) in the wrist region, forming the flexor and extensor retinacula of the wrist. Fiber bundles running in various directions form the antebrachial fascia. At the wrist, these bundles thicken and are arranged, from proximal to distal, in multiple layers extending in mediolateral and lateromedial directions (Plate 1.4.2). Many muscular fibers insert onto the inner surface of the antebrachial fascia in the proximal portion of the forearm, whereas this same fascia is always easily separable from the underlying muscles in the distal portion of the limb, attaching only to the radial and ulnar styloid processes and the pisiform bone. The tendon of the palmaris longus muscle pierces the antebrachial fascia in the distal third of the forearm, running superficial to the fascia before continuing with the palmar aponeurosis. The flexor carpi radialis and ulnaris muscles lie beneath the antebrachial fascia but, distally, their epitenons fuse with this fascia, so that the latter appears to envelop them at the wrist. The antebrachial fascia also forms the roof of Guyon's canal, through which the ulnar artery and nerve pass. In the palm, the antebrachial fascia continues laterally and medially with the thenar and hypothenar fasciae, and with the thick, transversal fiber bundles tensed between the eminences. In the mid-palm region, this thickening is continuous with the deep layer of the palmar aponeurosis. Muscular fibers of the thenar and hypothenar muscles also insert onto the inner surface of the fascia.

On histological analysis, three layers of parallel collagen fiber bundles, separated from each other by a thin layer of loose connective tissue, form the deep fasciae of the arm. The alignment of these bundles is parallel in each single layer but differs from layer to layer. Many vessels are present, mostly in the loose connective tissue layers, and are intermingled with the fibrous bundles. The collagen fibers represent less than 20% of the total fiber volume. The elastic fibers form a thin, irregular mesh, and are more evident within the loose connective tissue dividing the collagen layers. The histology of the wrist retinacula presents some differences. In particular, the fiber bundles are more densely packed, there is less loose connective tissue, and the van Gieson stain did not evidence any elastic fibers. Nerves are present throughout the brachial and antebrachial fasciae, although differences can be found according to the area and individual subjects. Small unmyelinated nerves are observed in all specimens, whereas Ruffini, Pacini and Golgi-Mazzoni corpuscles are present only in some, and mainly at the level of the wrist retinacula (Stecco et al. 2007).

The palmar aponeurosis

In the hand, the palmar aponeurosis adheres tightly to the skin due to the presence of thick fibrous septa (retinacula cutis). Two principal fibrous layers characterize the palmar aponeurosis: a superficial one, with a longitudinal disposition of fibers tightly adherent to the skin, and a deeper one, sited distally over the heads of the metacarpal bones, with a transverse arrangement of fibers. The transverse layer adheres to the longitudinal layer, which is more superficial. The vertical septa detach from the deep aspect of the transverse layer. These septa form the flexor tendon compartments of the last four digits, dividing them from the neurovascular compartments, and anchoring the deep aspect of the palmar aponeurosis to the metacarpal bones. The deep transverse layer could be considered a local specialization of the deep fascia of the palm, the distal equivalent of the antebrachial fascia. On the contrary, the longitudinal component of the palmar aponeurosis derives from the fusion of the laminar layer and of the deep compartment of the subcutaneous tissue, of which it could be considered a specialization. As the fibers of the palmaris longus tendon are found to be in continuity only with the longitudinal fiber system, it is suggested that this muscle should be considered as a proper tensor of the superficial fascial system of the subcutaneous tissue. Whenever the palmaris longus muscle is absent, the palmar aponeurosis is nevertheless present, but its superficial macroscopic appearance demonstrates decisive disarrangement. This suggests an active role of the palmaris longus muscle, via mechanical tension, in determining the longitudinal disposition of the fibers of the superficial layer.

The myofascial expansions

The fasciae of the pectoralis major, latissimus dorsi, and deltoid muscles continue distally with the brachial fascia (Standring et al. 2005; Rispoli et al. 2009); however, there are diverging descriptions and often this continuity is considered as anatomical variations with no functional significance. Dissections in the shoulder region of unembalmed cadavers (Stecco et al. 2008) have evidenced the constant presence of specific myofascial expansions originating from pectoralis major, latissimus dorsi, and deltoid muscles, and all of which merge into the brachial fascia.

Marshall (2001), while reviewing the deep fascia of the upper limb, states, "all these fascial attachments provide an excellent illustration of how the thickness and strength of aponeuroses and fasciae precisely mirror the forces generated by muscular action". In effect, these myofascial expansions could reduce movement near the entheses, as well as the stress concentrated at these sites. Indeed, the precise orientation of these tendinous expansions, apparently correlated to the spatial planes, and the different actions performed by the muscles, could suggest another functional role. In particular, the clavicular part of the pectoralis major, activated during shoulder flexion, presents an expansion onto the anterior portion of the brachial fascia. Therefore, during any forward movement of the arm, the contraction of these clavicular fibers of pectoralis major will stretch the anterior portion of the brachial fascia. The extension of the arm is performed by the latissimus dorsi, stretching the posteromedial portion of the brachial

fascia, and by the posterior part of the deltoid, stretching the posterolateral portion of the brachial fascia. The resultant is a vectorial force that stretches the posterior portion of the brachial fascia. The lateral part of the deltoid, mainly involved in the abduction of the arm, extends a myotendinous expansion towards the lateral intermuscular septum and the overlying brachial fascia. Lastly, expansions that originate from the chief adductor muscles (i.e., the latissimus dorsi and the costal portion of the pectoralis major muscles), extend towards the medial intermuscular septum. This means that, during adduction, the synchronous contraction of latissimus dorsi and of the costal fibers of pectoralis major muscles produces tension in the medial portion of the brachial fascia (Fig. 1.4.1).

These myofascial expansions are not present only in the pectoral girdle. In particular, the biceps brachii muscle gives an expansion, called lacertus fibrosus tendon or bicipital aponeurosis, onto the anterior antebrachial fascia (Plate 1.4.3). Besides, the palmaris longus muscle sends some tendinous expansions to



Fig. 1.4.1 • Schematic representations of the connections between the pectoral girdle muscles and the brachial fascia, according to the spatial planes. The connections between the latissimus dorsi muscle and the posterior and medial portions of the brachial fascia; between the deltoid muscle and the lateral intermuscular septum and the lateral portion of the brachial fascia; and between the pectoralis major muscle and the anterior (with the clavicular part) and medial (with the costal part) portions of the brachial fascia are represented.

the flexor retinaculum and to the fascia overlying the thenar muscles and the flexor carpi radialis tendon often sends an expansion onto the transverse carpal ligament. Some muscular fibers of flexor pollicis brevis and palmaris brevis also insert directly into the palmar aponeurosis. Also in the posterior part of the upper limb, numerous muscular insertions into the fascia have been described: the medial head of the triceps extends distally a tendinous expansion into the antebrachial fascia, and the extensor carpi ulnaris into the fascia of the hypothenar muscles. Finally, the abductor digiti minimi sends a tendinous expansion into the extensor aponeurosis (Fig. 1.4.2). During forward movement of the entire upper limb, the contraction of the clavicular fibers of pectoralis major will stretch the anterior region of the brachial fascia. Simultaneous contraction of the biceps muscle stretches the anterior region of the antebrachial fascia, by virtue of its bicipital aponeurosis, while the palmaris longus pulls on the flexor retinaculum, palmar aponeurosis and thenar fascia. In this way, these myofascial connections form an anatomical continuity between the various muscular components involved in flexion of the upper limb. Since the muscular fascia is richly innervated (Stecco et al. 2007) and this innervation is mostly of a proprioceptive type (mechanoreceptors, free nerve endings, etc.), stretch of specific zones of the fascia could activate specific patterns of proprioceptors. While the bony insertions of the muscles actuate their mechanical actions, their fascial insertions could play a role in proprioception, contributing to the perception of movement.

The resiliency of the deep fasciae of the arm

Resiliency studies of the deep fasciae of the arm using dynamometers show that the fascia could transmit the forces generated by muscular contraction. The lacertus fibrosus and the expansion of triceps sustained an average traction of 5.63 and 6.72 kg, respectively. The expansions of the pectoralis major and latissimus dorsi muscles sustained approximately 4 kg. The tendinous expansions in the hand are weaker, bearing a traction of about 2.5 kg. The resilience appears to be related to the muscular mass and force.
Fig. 1.4.2 • Schematic representation of the connections between the deep fascia of the upper limb and the underlying muscles. (A) Anterior vision. The expansions of the clavicular and costal parts of the pectoralis major muscle, of the biceps muscle (called also lacertus fibrosus), of the palmaris longus and flexor carpi radialis muscles are highlighted. They stretch the anterior portions of the brachial, antebrachial and thenar fasciae and the palmar aponeurosis. (B) Posterior vision. The expansions of the latissimus dorsi muscle and of the posterior part of the deltoid muscle into the brachial fascia are highlighted. The expansion of the triceps muscle stretches the posterior portion of the antebrachial fascia, while the abductor digiti minimi and extensor carpi ulnaris muscles stretch the ulnar side of the dorsal fascia of the hand.



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1.5

Deep fascia of the lower limbs

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Introduction

In the literature, different fasciae such as the fascia lata, iliotibial tract, plantar fascia, crural and gluteal fasciae are described in the lower limbs but only a few words are dedicated to their macroscopic and histologic description. Recent studies highlight the unifying role of connective tissue in the limbs. In particular, these studies have demonstrated the serial continuity of the different fasciae, demonstrating how the gluteal fascia continues with the fascia lata, the crural fascia, and lastly, the plantar fascia. The deep fasciae also blend with the periosteum, tendons, and ligaments (Benjamin 2009). The many functions of the deep fascia of the lower limbs include its role as ectoskeleton for muscle attachments. and the creation of osteofascial compartments for muscles. Its role in venous return, the dissipation of tensional stress concentrated at the site of entheses. and the fact that it serves as a protective sheet for underlying structures has also been recognized.

As our understanding of the anatomy and physiology of these structures expands, the important role of the deep fasciae in the interaction among the different muscles of the limbs has become more apparent. The deep fasciae could be considered to be like a bridge, passing over the joints and the septa to connect different muscles, but recent studies (Langevin 2006a) also recognize a primary role in the perception and coordination of movements, due to the unique mechanical properties and dense innervation of these fasciae. Different researches also suggest that the deep fasciae present a basal tension. This basal tension could be due to the stretching of the underlying muscle by muscular or tendineous insertions (Stecco et al. 2009), or by the action of the myofibroblasts within the fascia (Schleip et al. 2006).

Gross anatomy

Three fundamental structures form the fasciae of the lower limbs: the superficial fascia, the deep fascia, and the epimysium.

The superficial fascia is a collagen layer that divides the hypodermis into three distinct layers: the superficial adipose tissue, a membranous intermediate layer, or true superficial fascia, and the deep adipose tissue. The thickness of the two adipose tissue layers varies in the different zones of the limbs, determining specific, regional relationships between the superficial fascia and the skin, and between the superficial and deep fasciae.

The deep fascia consists of a lamina of connective tissue that is, generally, easily separable from both the underlying muscles and the overlying superficial fascia. In fact, there is virtually an uninterrupted plane of gliding between the deep fascia and the muscles surrounded by their epimysium, with just a little layer of interposing, loose connective tissue to facilitate gliding. This loose connective tissue appears as a pliable, gel-like gelatinous substance. Histologic studies demonstrate that fibroblasts are widely dispersed within this tissue and that collagen and elastic fibers are disposed in an irregular mesh.

A few strong intermuscular septa originate from the inner surface of the deep fascia of the lower limbs and extend between the muscle bellies, dividing the thigh into different compartments, and providing an origin to some lower limb muscle fibers (Plate 1.5.1).

It is important to recognize that even though the deep fascia of the lower limbs in the thigh is called fascia lata, and in the leg crural fascia, it is actually the same structure. This fascia appears as a thick, whitish layer of connective tissue, similar to an aponeurosis, with an average thickness of 1 mm and, in general, the deep fascia of the lower limbs is thicker in the posterior regions of the limbs. Nevertheless, studies concerning the variations in thickness of the lower limb fasciae have demonstrated some regional differences. In particular, the deep fascia of the anterior thigh presents a mean thickness of $944 \pm 102 \,\mu\text{m}$. It is thinner in the proximal region (541 \pm 23 μ m) and thicker near the knee $(1419 \pm 105 \,\mu\text{m})$, while in the middle third of the thigh it presents a mean thickness of $874 \pm 62 \,\mu\text{m}$. In the lateral region, it is reinforced by the iliotibial tract. The iliotibial tract is not separable from the deep fascia by dissection. Therefore, anatomically, it can not be considered a separate entity, but a reinforcement of the lateral aspect of the fascia lata.

The crural fascia has an average thickness of 880 μ m, which progressively decreases from 1 mm in the popliteal region to 700 μ m in the distal third of the leg.

Around the knee and the ankle, the deep fascia is reinforced by additional fibrous bundles, commonly called retinacula; however, it should be emphasized that in all of the fasciae many fibrous bundles running in different directions are macroscopically visible.

The retinacula

The retinacula are typically regional specializations of the deep fascia; in particular, they are thickenings of the deep fasciae and, as such, are not separable. They appear as a strong fibrous bundle with a mean thickness of 1372 µm and a criss-cross arrangement of the collagen fibers. The retinacula have many bone insertions, and these entheses may be fibrocartilaginous. At other points, they can glide over the bones thanks to interposing loose connective tissue between the retinacula and the periosteum. From a functional point of view, the retinacula of the ankle have classically been considered as a pulley system, maintaining the tendons adherent to the underlying bones during movements of the tibiotarsal joint, and as important elements for ankle stability, connecting various bones. However, in 1984, Viladot et al. stated that they may play an important role in proprioception. For

example, the peroneal retinacula may be stretched by inversion of the ankle joint, activating reflex contraction of the peroneal muscles. Ankle retinacula can be easily evaluated by magnetic resonance imagery, as they appear as low signal intensity bands, sharply defined in the context of the subcutaneous tissue in T1-weighted sequences, with a mean thickness of 1.25 mm (SD \pm 0.198). Retinacula can also be subjected to traumatic ruptures, sometimes resulting in subluxation of the underlying tendons. Also the knee retinacula could be easy evaluated by magnetic resonance imaging, as they appear clearly as low signal intensity bands. In patients affected by patellar femoral malalignment or anterior knee pain, differences in thickness, innervations, and vascularisation are appreciable.

Fibrous expansions and muscular insertions

In a few specific regions, the muscles of the lower limb connect with the deep fascia via fibrous expansions or direct insertions of their muscular fibers. These expansions and insertions are well worth some in-depth discussion for their potential functional implications.

While the iliotibial tract could be considered the tendon of the tensor fascia lata and the gluteus maximus muscles, it is also a reinforcement of the fascia lata. It has extensive attachments to the lateral intermuscular septum in the thigh and many muscular fibers of the vastus lateralis muscle also originate from this septum. Therefore, during movement of the lower limb, the lateral intermuscular septum is stretched proximally by the gluteus maximus, and distally by the vastus lateralis muscles (Plate 1.5.2). Distally, the iliotibial tract is attached to Gerdy's tubercle at the upper end of the tibia, but it also has an expansion into the antero-lateral portion of the crural fascia. Fairclough et al. (2007) suggest that iliotibial band syndrome is not due to frictional forces created by moving forwards and backwards over the tibial condyle during flexion and extension of the knee, but to tensional changes within the iliotibial tract itself. Similarly, the sartorius, gracilis, and semitendinosus muscles form the pes anserinus in the medial portion of the knee, but they also extend some expansions into the medial aspect of the crural fascia (Fig. 1.5.1). Besides, the quadriceps muscle has some obliquely directed fascial expansions arising from the vastus medialis and lateralis muscles, that pass anterior to the patella fusing with the fascia lata,



Fig. 1.5.1 • Schematic representation of the lines of forces inside the popliteal fascia, created by the tractions of biceps femoris muscle (1) and semimembranosus (3) proximally, and by the tractions of the two heads of the gastrocnemius muscle (2) distally.

and contributing to knee retinacula formation, and the distal tendon of the semimembranosus muscle has two expansions in the popliteal region: one extends into the posterior wall of the knee joint capsule, forming the oblique popliteal ligament, and one extends into the fascia of the popliteus muscle. Distally, the proximal portions of the gastrocnemius muscle are inserted directly onto the fascia, so that these muscular fibers could be considered as tensors of the fascia (Plate 1.5.3), and anteriorly the tibialis anterior muscle and flexor hallucis longus insert onto the overlying fascia and the intermuscular septum. In this way, around the knee, it is quite difficult to separate the deep fascia from the underlying muscles and tendons.

At the level of the ankle and the foot the deep fascia also presents some muscular and tendinous insertions. In particular, numerous muscular fibers from the extensor digitorum brevis and abductor hallucis muscles originate, respectively, from the inner side of the inferior extensor and flexor retinacula. As mentioned previously, these retinacula are reinforcements of the deep fascia of the foot. Finally, the Achilles tendon not only attaches to the posterior aspect of the calcaneus, but also has fascial continuity both with the plantar aponeurosis over the back of the heel and with the fibrous septa of the heel fat pad. According to Moraes do Carmo et al. (2008), a progressive age related diminution of the number of fibers connecting the Achilles tendon to the plantar fascia could be evidenced by dissection: in neonatal subjects, a consistent thickness and continuity of fibers are evidenced as compared to middle-aged subjects, where only superficial, periosteal fibers continuing from the Achilles tendon on to fascia are found, while in elderly feet, no connections could be highlighted. All these myofascial expansions may stabilize tendons, reducing movement near the enthesis and, consequently, minimizing the stress that concentrates at bony insertion sites, but they also permit selective stretching of the fascia, and the development of specific lines of forces within the deep fascia that could determine collagen fiber disposition, as well as have a role in the activation of specific patterns of proprioceptors during movement. The implications of these above-mentioned studies in terms of the observed loss of equilibrium in the aged could be significant.

Microscopic anatomy

Classically, the deep fasciae of the limbs are classified as irregular, dense, connective tissue with the mere function of enveloping muscles. More recent studies provide important evidence of a definite microscopic organization of the deep fascia of the limbs. On microscopic evaluation, the deep fasciae of the lower limbs and the retinacula are formed by collagen fiber bundles presenting a slight, undulating arrangement. Surprisingly, the collagen fibers actually represent less than 20% of the total volume of the fascia. In the retinacula, the fibrous bundles are more densely packaged and there is less loose connective tissue. The fibrous bundles are regularly disposed in two or three distinct layers of parallel collagen fiber bundles. Each collagen layer presents a mean thickness of 277.6 \pm 86.1 μ m. Inside a single layer, all the collagen fiber bundles are aligned in parallel, while this orientation differs from layer to layer (Fig. 1.5.2). The angle between the orientation of the fibers in adjacent layers, evaluated in the x-y

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plane, appears to be about 78°. Normally, there is a complete independency between the different layers due to the presence of a thin layer of loose connective tissue (mean thickness $43 \pm 12 \,\mu\text{m}$) that permits sliding between layers. Nevertheless, in some regions isolated bundles of collagen fibers connect the layers. In all of the body, the role of the loose connective tissue is to cushion and separate different structures. It is known that this tissue is an important reservoir of water and salts for surrounding tissues and it could accumulate different degradation products. Eventual variations in the contents of water, ions, or other substances could potentially alter the biomechanical proprieties of the loose connective tissue and so interfere with the sliding mechanism of the different fascial layers.

The dominant cells inside the deep fasciae of the lower limbs are the fibroblasts, although the accumulation of actin stress fibers within these cells in response to mechanical loading has led some authors to consider many of these cells to be myofibroblasts (Schleip et al. 2007). However, where dense fascia is subject to significant levels of compression, e.g., in certain retinacula or in parts of the plantar fascia, the cells have a chondrocytic phenotype and the tissue can be regarded as fibrocartilage. According to Langevin et al. (2006b), the resident fibroblasts inside the fascia are integral to mechanotransduction. They communicate with each other via gap junctions and respond to tissue stretch by shape changes mediated via the cytoskeleton. In stretched tissue, the cells are sheet-like and have large cell bodies, whereas in resting tissue, they are smaller and dendritic in shape, and have numerous, attenuated cell processes. The cell shape changes may also influence tension with the connective tissue itself.

With van Gieson elastic fiber staining, the deep fasciae reveal some elastic fibers with a volume fraction comprising a range of 0.3–1.5%. They form an irregular mesh and are more evident inside the loose

Fig. 1.5.2 • Schematization of the deep fasciae of the limbs, showing the composition in three different layers. In each layer the collagen fibrous bundles are parallel to each other, while adjacent layers show different orientations of the collagen fibers. Each layer is separated from the adjacent one by a thin layer of loose connective tissue that permits the different layers slide one on the other.

connective tissue dividing the various collagen layers. On the contrary, no elastic fibers were found inside the retinacula. In some subjects, some welldelimited bundles of muscular fibers have been evidenced within the fascia lata itself.

The specific orientation of the collagen fibers, with the presence of an initial crimp at rest, the irregular distribution of elastic fibers and the variable presence of loose connective tissue implies a great complexity in the biomechanical behavior of the deep fasciae of the lower limbs. In particular, it demonstrates a strong anisotropy, best defined as the condition of not having properties or characteristics that are the same in all directions. Even in the absence of an elevated percentage of elastic fibers, the crimped conformation of the collagen fibers, and the angle that forms between the directions of fibers in adjacent layers, would allow the deep fasciae a certain mechanical adaptability. In fact, this is evident clinically in compartment syndromes, where the deep fasciae of the limbs resist high pressure without apparent damage, whereas under normal physiological conditions the same structures adapt to the volume variations of muscles during their contractions.

Furthermore, it is interesting to note that the deep fasciae of the lower limbs around the joints and along the tibial crest present specific adhesions to the joint capsules and/or bones. These points become areas where tensional stress concentrates, representing the meeting point between hard and soft tissues. In particular, where fascia connects with bones, entheses are designed to reduce this concentration of stress, and the subsequent anatomical adaptations are evident at the gross, histologic, and molecular levels.

Numerous vessels, with a mean caliber of $102.15 \,\mu$ m, follow rather tortuous paths through the different collagen layers of the deep fasciae of the lower limbs. Nerve fibers are also found in all the deep fasciae, with a volume fraction of about 1.2%. They are particularly numerous around vessels,

but are also distributed homogeneously throughout the fibrous components. The intrafascial nerves are connected to the collagen fibers, and often oriented perpendicularly to the collagen fibers, and so, in all probability, they could be stimulated by stretching of the collagen fibers. In some specimens Ruffini and Pacini corpuscles are also highlighted. Some small nerve fibers exhibit characteristics typical of autonomic nerves. More specifically, they are adrenergic and likely to be involved in controlling local blood flow. According to Sanchis-Alfonso and Rosello-Sastre (2000), changes in innervation can occur pathologically in fascia. These authors report the in-growth of nociceptive fibers, immunoreactive to

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The deep fascia of the limbs also presents specific relationships with the large vessels and nerves and contributes to forming their protective sheaths. Around these delicate structures, the fascia is disposed in multiple layers separated by loose connective tissue, creating a structure similar to a telescope. This permits a type of sliding system among the different layers, preserving the nerves and vessels, cushioning them from the traction to which the fascia is subjected. Whenever this protective mechanism is altered, a compressive syndrome, either of a nervous or vascular structure, could develop.

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The thoracolumbar fascia

An integrated functional view of the anatomy of the TLF and coupled structures

Andry Vleeming

Introduction

To understand and treat low back pain, or even better, locomotion in vertebrates, models based on descriptive anatomy are generally used. This branch of anatomy was developed to answer the question what structures does our body consist of, and to categorize them. Categories like spine, pelvis, and legs, are primarily based on bone anatomy.

Functional anatomy of the locomotor system, which is strongly linked to biomechanics, attempts to explain how bones, ligaments, and muscles operate as a system. Consequently, use of categories like spine and pelvis can be misleading. From a biomechanical and neurophysiological point of view they are fully integrated. "Back muscles", for instance, are categorized in descriptive anatomy as typically spinal. However, parts of these "back muscles" bride the sacroiliac (SI) joints. As an example, in man the multifidus muscle shows an extensive attachment to both the sacrum and the iliac crest (Vleeming & Stoeckart 2007).

With the use of descriptive anatomical models it is tempting to regard pain in the area of the SI joints as a separate syndrome, not as part of low back pain. However, these joints are fully and crucially integrated in the spine–pelvis–leg mechanism (Snijders et al. 1993a). To function properly this mechanism needs stability of the pelvis (external movement of the pelvis) and the SI joints and symphysis (internal movement of the pelvis), for effective use of the three levers connected to the pelvis: two legs and the spine. Effective load transfer across the SI joints requires specific action of a variety of muscles, leading to sufficient compression of the SI joint, preventing shear (Snijders et al. 1993a, b). In increasing compression the biceps femoris and gluteus maximus muscles play a role (Vleeming et al. 1989a,b, 1992b 1996; DonTigny 1990; Vleeming 1990; Van Wingerden et al. 1993). Both muscles are attached to the sacrotuberous (and partially the sacrospinous) ligament which functionally bridges the SI joint. Obviously, pain in the area of the SI joints is not necessarily a local problem; it can be symptomatic of a failed load transfer system (Snijders et al. 1993a, b).

The strong thoracolumbar fascia (TLF) (Tesh et al. 1987) can be used for load transfer from the trunk to the legs (Mooney et al. 2001). The posterior (PLF) and middle layer (MLF) of this fascia are of special interest because of the multiple connections with muscles. The main interest is whether muscle-induced tension of this fascia can assist in effectively transferring load between spine, pelvis, legs, and arms.

From an anatomical point of view the following was noticed:

In all preparations, the posterior layer of the thoracolumbar fascia covers the back muscles from the sacral region through the thoracic region as far as the fascia nuchae. At the level of L4–L5 and sacrum strong connections exist between the superficial and deep lamina. The transverse abdominal and internal oblique muscles are indirectly attached to the thoracolumbar fascia through a dense raphe formed by fusion of the middle layer (Adams & Dolan 2007) of the thoracolumbar fascia and both laminas of the posterior layer. This "lateral raphe" (Bogduk & Macintosh 1984; Bogduk & Twomey 1987; DeRosa & Porterfield 2007) is localized lateral to the erector spinae and cranial to the iliac crest.

Superficial lamina (Fig. 1.6.1)

The superficial lamina of the posterior layer of the thoracolumbar fascia is continuous with the latissimus dorsi, gluteus maximus, and partly the external oblique muscle of the abdomen and the trapezius muscle. Cranial to the iliac crest, the lateral border of the superficial lamina is marked by its junction with



Fig. 1.6.1 • The superficial lamina. (A) Fascia of the gluteus maximus. (B) Fascia of the gluteus medius. (C) Fascia of external oblique. (D) Fascia of latissimus dorsi. (E) Cross-hatched appearance of the superficial lamina due to different fiber directions of latissimus dorsi and gluteus maximus. 1. Posterior superior iliac spine. 2. Sacral crest. LR, part of lateral raphe. Arrows (at left) indicate, from cranial to caudal, the site and direction of the traction (50 N) given to trapezius, cranial and caudal part of the latissimus dorsi, gluteus medius and gluteus maximus, respectively. From Vieeming et al., 2007, with permission.

the latissimus dorsi muscle. Barker and Briggs (2007) reported that the superficial lamina also has an attachment of variable thickness to the lower border of the rhomboid major muscle (Barker & Briggs 2007).

The fibers of the superficial lamina are orientated from craniolateral to caudomedial. Only a few fibers of the superficial lamina are continuous with the aponeurosis of the external oblique and the trapezius. Most of the fibers of the superficial lamina derive from the aponeurosis of the latissimus dorsi and attach to the supraspinal ligaments and spinous processus cranial to L4. Caudal to L4–L5, the superficial lamina is generally loosely (or not at all) attached to midline structures such as supraspinal ligaments, spinous processes, and median sacral crest. In fact they cross to the contralateral side, where they attach to the sacrum, posterior superior iliac spines, and iliac crest. The level at which this phenomenon occurs varies: generally caudal to L4 but in some preparations already at L2–L3.

At sacral levels the superficial lamina is continuous with the fascia of the gluteus maximus. These fibers are orientated from craniomedial to caudolateral. Most of these fibers attach to the median sacral crest. However, at the level of L4–L5, and in some specimens even as caudal as S1–S2, fibers cross the midline, attaching to the contralateral posterior superior iliac spine and iliac crest. Some of these fibers fuse with the lateral raphe and with fibers derived from the fascia of the latissimus dorsi. Due to the different fiber directions of the latissimus dorsi and the gluteus maximus, the superficial lamina has a cross-hatched appearance at the level L4–L5, and in some preparations also at L5–S2.

Deep lamina (Fig. 1.6.2)

At lower lumbar and sacral levels the fibers of the deep lamina are oriented from craniomedial to caudolateral. At sacral levels these fibers are fused with those of the superficial lamina. Since fibers of the deep lamina are continuous with the sacrotuberous ligament here, an indirect link exists between this ligament and the superficial lamina. There is also a direct connection with some fibers of the deep lamina.

In the pelvic region the deep lamina is connected to the posterior superior iliac spines, iliac crests, and the long posterior sacroiliac ligament (O'Rahilly et al. 1990). This ligament originates from the sacrum and attaches to the posterior superior iliac spines.

In the lumbar region fibers of the deep lamina derive from the interspinous ligaments. They attach to



Fig. 1.6.2 • The deep lamina. (B) Fascia of the gluteus medius. (E) Connections between the deep lamina and the fascia of the erector spinae. (F) Fascia of the internal oblique. (G) Fascia of the serratus posterior inferior. (H) Sacrotuberous ligament. 1. The posterior superior iliac spine. 2. Sacral crest. LR, part of lateral raphe. Arrows (at right) indicate, from cranial to caudal, traction to serratus posterior inferior and internal oblique, respectively. From Vleeming et al., 2007, with permission.

the iliac crest and more cranially to the lateral raphe, to which the internal oblique is attached. In some specimens, fibers of the deep lamina cross to the contralateral side between L5–S1. In the depression between the median sacral crest and the posterior superior and inferior iliac spines, fibers of the deep lamina fuse with the fascia of the erector. More cranial, in the lumbar region, the deep lamina becomes thinner and freely mobile over the back muscles. In the lower thoracic region fibers of the serratus posterior inferior muscle and its fascia fuse with fibers of the deep lamina.

Kinematics

The study presented here confirms some previous studies and disagrees with others. The bilaminar structure of the posterior layer of the thoracolumbar fascia has been described by Fairbanks, and O'Brien (1980), Gracovetsky (1990), Macintosh and Bogduk (1986), Bogduk and Macintosh (1984), and Bogduk and Twomey (1987). They describe the orientation of the fibers of the superficial and deep lamina as, respectively, caudomedial and caudolateral. The present study confirms the orientation of the laminae and their attachments. It is noteworthy that according to most studies (Fairbanks & O'Brien 1980; Bogduk & Macintosh 1984; Bogduk & Twomy 1987) the latissimus dorsi is mentioned as the significant structure from which fibers of the superficial lamina originate. The gluteus maximus as origin for the formation of the superficial fascia is ignored. Bogduk and Macintosh (1984) state that fibers located caudally from L3 decussate to the contralateral site, although it was not possible to trace the precise origin of these fibers because of strong fusion to midline structures. The present study confirms the phenomenon of crossing fibers. The level of the crossing varies from L2–S2. In contrast to the study of Bogduk and Macintosh, no connections were found between the serratus posterior inferior and the superficial lamina: Its fascia is exclusively connected to the deep lamina.

Bogduk and Macintosh (1984) and Bogduk and Twomeny (1987) describe the deep lamina as a structure consisting of bands of collagen fibers extending from the lumbar spinal processes to the iliac crest and lateral raphe. However, we can not confirm the existence of bands of collagen fibers; we find a continuous layer. The authors pay no attention to the sacral part of the deep lamina. As a consequence, the connections with the sacrotuberous ligaments are omitted. Therefore the biomechanical model as proposed by Bogduk and Twomey (1987) is incomplete. The bracing effect of the thoracolumbar fascia on the lower lumbar spine and SI joints, essential for proper load transfer between spine and legs (Snijders et al. 1993a,b), can only be adequately described if the caudal part of the thoracolumbar fascia is included.

Overarching arguments about the anatomy of the TLF

Another approach in looking at the thoracolumbar fascia in an integrated anatomical way is the anatomical work of Willard (2007). Willard shows that the TLF, including the myofascial sheath over the multifidus, is continuous with the supraspinous ligament,



Fig. 1.6.3 • The attachment of the multifidus sheath to the supraspinous ligament. The thoracolumbar fascia (TLF) and multifidus sheath have been sectioned longitudinally to reveal the multifidus muscle (Mu). (A) The iliocostalis and longissimus muscles have been removed. From Willard, In Vleeming et al., 2007, with permission.

ligament flavum, and facet joint capsule, with the deeper ligaments as described here (Fig. 1.6.3).

Barker and Briggs (2007) summarize that the middle layer (MLF) of the TLF and the posterior layer (PLF) have a suitable morphology for generating transverse tension and are capable of transmitting tensile loads from attached muscles to all lumbar vertebrae. The MLF, however, provides a more direct route and is indicated to transmit the majority of tension of the transversus muscle (Fig. 1.6.4). Tension in both layers (PLF and MLF) can influence features of segmental control in the sagittal plane and would be predicted to have the greatest effects in the transverse plane.

A biomechanical model of the SI joints was proposed (Snijders et al. 1993a,b). It was stated that joints with predominantly flat surfaces are well suited to transfer large moments of force but are vulnerable to forces in the plane of the joint surfaces (Vleeming et al. 1990, 1992; Snijders et al. 1993a,b). Therefore flat joint surfaces go with restricted joint excursions. In a model (Vleeming 1990) of the SI joints based on anatomical and biomechanical findings, the principle of form and force closure was discussed. Form closure refers to a stable situation with closely fitting joint surfaces, where no extra forces are needed to maintain the state of the system, given



Fig. 1.6.4 • Middle layer of lumbar fascia (MLF). Note the MLF is continuous with the transversus abdominis (TrA) via inferolateral fibers and has thick attachments to the transverse processes. MLF, middle layer of lumbar fasciae; TrA, transversus abdominis. From Barker, In Vleeming et al., 2007, with permission.

the actual load situation. In this situation no lateral forces are needed to counterbalance the effects of the vertical load. With force closure a lateral force is needed.

The SI joint with its undulated form and symmetrical ridges and depressions combined with compression and the generated friction is an example of a joint (like any other joint) remaining stable through a combination of form and force closure (Vleeming et al. 1990). In case force closure is not sufficient, e.g., due to insufficient muscle action and hence insufficient ligament strain, form closure becomes important.

Pelvic instability (and peripartum pain) can be partially and temporarily relieved by application of a pelvic belt, a device fitting the model of self-bracing of the SI joints (Snijders et al. 1976; Vleeming et al. 1992). Force closure is increased by such a belt, located just cranially to the greater trochanter and caudally to the SI joints (Vleeming et al. 1992a,b; Snijders et al. 1993a). The belt can be used with small force, resembling the action of the laces of a shoe. As shown in this study, the coupled function of gluteus maximus and contralateral latissimus dorsi also creates a force perpendicular to the SI joints (Mooney et al. 2001). The same is especially true of the caudal fibers of the transversus and the internal oblique muscle.

The thoracolumbar fascia

Conclusion

In this chapter we have discussed the thoracolumbar fascia from a "fascial" point of view and its role in transferring forces between spine, pelvis, and legs, in relation to stabilization of the lower lumbar spine and SI joints.

The gluteus maximus and the latissimus dorsi merit special attention since they can conduct forces contralaterally, via the posterior layer (Mooney et al. 2001). We also discussed briefly the role of the MLF and especially the important effect of the transverses and internal oblique muscles on the MLF.

However, reality is more complex.

Directly anterior to the deep layer of the PLF lies the very strong erector spinae aponeurosis, overlying both the longissimus and the lumbar part of the iliocostal muscle and the deeper-lying multifidus muscles. This aponeurosis blends together with the multifidus to the dorsal side of the sacrum and partially to the ilium. Contraction of both the erector muscle and the multifidus will increase the tension in the deep lamina, directly by pull, and indirectly by dilating the complete posterior layer of the thoracolumbar fascia, but also affecting the tension of the middle layer. If the transverses and the internal oblique fire earlier than other muscles in healthy individuals, the "floor" (MLF) of the TLF envelope will be tensed, also with a small effect on the PLF. In that case, erector and multifidus contractions will become more efficient because the slack of the fascial envelope is diminished.

For that reason we have to realize that within the envelope of the TLF (posterior, middle, and anterior layer) this strong aponeurotic fascia/tendon is present.

Furthermore, we have to realize that any external movement of the pelvis through the hip joints influences the tension in the TLF, and also the relative flexion or extension position of the trunk with or without lateroflexion and rotation. All these factors will change the external and internal dynamics – force and pressure – of the fascial envelope (see also Fig. 1.6.5).

Even more distant effects have to be considered.

The fascia latae of the leg and especially the most pronounced part, the iliotibial tract, can be expanded and tensioned by the large vastus lateralis muscle. For example, when a soccer player kicks the ball while the standing leg is flexed, both the vastus lateralis and gluteus maximus will be active. Both of these muscles influence each other's function, because a big part of the gluteus maximus is directly connected to the iliotibial tract of the fascia latae, which can also be dilated ("pumped up") by the vastus.

We have discussed in this chapter that the gluteus maximus couples the leg to the hipbones and to the TLF. Therefore, the pathway of fascial transfer is not restricted to the envelope of the three layers of the TLF.

Fig. 1.6.5 • (A) Muscle contraction resulting in increased tension to the thoracolumbar fascia. EOA, external oblique: ES. erector: IOA, internal oblique; LD, latissimus dorsi; PM, psoas major; QL, quadratus lumborum; SP, spinous process; TA, transversus abdominus; VB, vertebral body; TP, transverse process. (B) Posterior view of the musculature that attaches to the TLF. Note the forces that are generated to the fascia from the latissimus dorsi muscle (1) from above, the gluteus maximus (2) from below, and the internal obligue muscle (3) and transversus abdominus muscle from the front (4). From de Rosa and Porterfield, In Vleeming et al., 2007, with permission.



Descriptive topographic anatomy likes to dissect and analyze structures topographically, while they are actually functionally related. One has to realize, especially from a rehabilitation perspective, that within the TLF envelope muscles are made of connective tissue of different kinds of tensile strength: "loose" connected tissue, tendons, aponeurosis, and fascia. So it is an illusion to consider the TLF a connective tissue envelope "filled" with muscle tissue! It is a functionally coupled connective tissue unit filled with contractile elements.

In the beginning of this chapter we discussed force closure of the spine and pelvis. Activation of the multifidus and the erector muscles and the subsequent tension through the erector spinae aponeurosis (all lying within the TLF envelope) develop a strong extension effect of the lower spine and a nutation effect on the sacrum (Fig 1.6.6). When moving from a lying to a standing or sitting position the sacrum nutates relative to the ilium and increases tension of the sacrospinous, sacrotuberous, and interosseous ligaments, thereby further increasing force closure of the sacroiliac joint.

Nutation is coupled to extension of the lumbar vertebra. Nutation of the SIJ means posterior rotation of the ilium relative to the sacrum. Extension of the lumbar vertebrae coupled with posterior rotation of the ilium increases the tension in the iliolumbar ligaments, coupling force closure of the SIJ to the lumbar spine.

Finally, also due to the coupling between the thoracolumbar fascia, muscles, and other large fascial systems, one has to be very cautious in categorizing certain muscles exclusively as belonging to the arm, spine or leg.

In transferring forces between spine, pelvis, and legs, the thoracolumbar fascia plays a crucial role, transferring forces cranially, caudally, and also diagonally.



Fig. 1.6.6 • Nutation and counternutation.
(A) Nutation winds up the sacrotuberous ligament.
(B) Counternutation winds up the long dorsal sacroiliac ligament. From: Vleeming et al Movement, stability & lumbopelvic pain, 2nd edition. Churchill Livingstone Elsevier, Edinburgh, 2007: page 118, Fig. 8.6.

With all this in mind, we hopefully can appreciate how to design a more suitable and realistic rehabilitation protocol for our lumbopelvic patients.

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The deeper fasciae of the neck and ventral torso

Rainer Breul

Introduction

In early embryogenesis the originally unitary body cavity is divided by the development of the transverse septum, the future diaphragm, and the pleuropericardial septum secondarily into the thoracic cavity and the peritoneal cavity. Both structures arise from the mesenchymal concentrations of connective tissue in the neck (see below).

The two pleural cavities and the pericardium – pericardial cavity – are formed within the thoracic cavity. These cavities are completely filled with their respective contents (lungs, heart, abdominal organs) and a small quantity of serous fluid.

Both pleural cavities are lined with the parietal pleura; this continues along the hilum of the lungs into the visceral pleura. At the top, the parietal pleura forms the two pleural domes, which rise up on both sides over the upper thoracic aperture and reach the posterior head of the first rib. The parietal pleura folds in on itself in a caudal direction deep into the costodiaphragmatic recess. In this area the pleural and peritoneal cavities are separated from each other by the thin muscular plate of the diaphragm. The domes of the diaphragm, which rise up well into the thoracic space, are joined to each other by the central tendon.

Medially, the two pleural cavities border the mediastinum, which is closed at the bottom by the diaphragm, but in a cranial direction is in direct connection with the connective tissue-filled crevices of the neck.

The anterior mediastinum contains the pericardium with the heart and its arterial and venous vessels; posterior from here lie the trachea with the origins of the bronchial tubes, the aorta, the esophagus with the branches of the vagus nerve, and the thoracic duct.

The cranial border of the abdominal cavity is the diaphragm, the lateral border is the abdominal wall, and the caudal border is formed by the pelvis and the pelvic floor. With the parietal peritoneum as internal lining, it encloses a serous cavity, which contains the digestive organs. Posteriorly, the parietal peritoneum borders a connective tissue space, the retroperitoneal space, which contains the retroperitoneal vessels and organs.

Neck fascia

A visceral cord lies in front of the cervical spine consisting of nerve and vascular bundles and cervical organs. A transverse section at the level of the first tracheal cartilage shows that the visceral cord is fairly central and the posterior border is directly on the cervical spine. It is surrounded by a connective tissue muscular coating composed of three fascia and pretracheal and prevertebral muscles and a dense covering (Fig. 1.7.1).

The visceral cord in the neck is therefore mobile against its covering and is lifted and lowered by the act of swallowing and can follow any movement in the cervical spine. Furthermore, the vascular nerve bundle formed by the carotid sheath is arranged with the internal jugular vein, the common carotid artery, and the vagus nerve so that it remains as protected as possible from the changes in position and shape of the cervical spine and the viscera of the neck. PART ONE Anatomy of the Fascial Body



Fig. 1.7.1 • Horizontal section through the neck at the level of the first tracheal cartilage. Note how the infrahyoid muscles (9,10 and 12) are enveloped by the fascia colli media. 1 Spatium suprasternale, 2 thyroid gland, 3 internal jugular vein, 4 arteria carotis communis, 5 vagus nerve, 6 anterior scalene muscle, 7 branches of brachial plexus, 8 fascia colli superficialis, 9 sternohyoid muscle, 10 sternothyroid muscle, 11 sternocleidomastoid muscle, 12 omohyoid muscle, 13 thyroid capsule, 14 fascia colli profunda, 15 prevertebral muscles, 16 vertebral artery and vein, 17 scalenus medius muscle, 18 trapezius muscle, 19 fascia nuchae.

The junction of the carotid sheath with the omohyoid muscle further ensures that the reflux venous blood flow runs from the brain independently of position.

Arrangement of the three neck fascia

Fascia colli superficialis (lamina superficialis fasciae cervicalis)

Lying directly below the skin and platysma, the superficial sheet of the cervical fascia stretches over a wide area. As continuation of the masseteric fascia it runs from the mandible and the floor of the mouth over the interposition of the hyoid bone in a caudal direction, after which it is attached to the clavicle and the manubrium sterni and continues into the pectoral fascia. It shifts significantly during respiration because of its attachment to the sternum. It forms a lateral sheath around the sternocleidomastoid muscle, which glides freely within this covering. At the back it covers the corpus adiposum of the lateral cervical triangle and continues to the trapezius muscle. Upwards in a posterior direction it traverses the mastoid process and extends from there as far as the superior nuchal line.

The superficial neck fascia is structured differently in different regions; in the area of the digastricus muscle, the anterior part of the trapezius, and the lower third of the sternocleidomastoideus it is delicate. In the cranial third, the superficial fascia is very strong and is almost immobile against the subcutis. In the lower part of the lateral cervical triangle, it is like a sieve because of the passage of the supraclavicular nerve and its accompanying vessels. Because of its many connections to neighboring structures it is under permanent tension in a living being (Fig. 1.7.2).

Fascia colli media (lamina pretrachealis fasciae cervicalis)

The middle neck fascia is composed of the fascia coverings of the infrahyoid muscles and forms a firm triangular skirt in front of the cervical viscera. Cranially, it grows fixed to the hyoid bone; caudally it runs with the coverings of the sternohyoidei and sternothyroidei through the upper thoracic aperture behind the sternum and inserts at the manubrium sterni. Laterally,



Fig. 1.7.2 • Sagittal view of the fasciae of the neck.

it is fixed to the posterior surface of the clavicle and is bordered posterolaterally by the fascia coat of the omohyoideus, which arises from the medial angle of the scapula and runs in an arc to the hyoid. The two omohyoid muscles span the middle neck fascia and are firmly held in place by their tendons. As the middle neck fascia is involved in the formation of the carotid sheath, the lumen of the internal jugular vein is held open by its traction. This principle also applies to the superficial veins of the neck and the deep veins, which come from the area of the neck and shoulder girdle.

Above the sternum is a space between the middle neck fascia and the superficial neck fascia filled with fatty tissue and veins, the suprasternal space. It extends laterally as far as the sternocleidomastoideus and cranially as far as the level of the ring of cartilage around the larynx. Both fasciae are welded together to the hyoid bone.

Fascia colli profunda (lamina pretrachealis fasciae cervicalis)

The deep neck fascia is fixed to the anterior longitudinal ligament of the cervical spine and provides a connective tissue cover for the prevertebral muscles (Mm. longi colli) and the lateral neck muscles (Mm. scaleni). These coverings are therefore considerably involved in the construction of the posterior wall of the inside space of the neck. Cranially, the deep neck fascia is fixed to the base of the skull. Laterally, it is connected to the fascia of the levator scapulae, the nuchal fascia and the superficial sheet of the neck fascia. From the scalenus medius it reaches the clavicle, and together with the scalenus anterior it reaches the outer surface of the thorax. Over this area it covers the nerve and vessel bundle to the upper extremities – the brachial plexus and the subclavian artery.

Caudally, the deep neck fascia travels into the endothoracic fascia.

Connective tissue tracts radiating from the deep neck fascia of the neck into the suprapleural membrane contribute to the fixation of the pleural dome (see below).

Fascia of the thorax

The superior thoracic aperture represents the connection to the neck and is bordered by the first pair of ribs, the sternum, and the first thoracic vertebra. The insertion into the thorax is diagonal and the superior sternal margin is at the level of the second thoracic vertebra in mid-respiration.

While the lungs fill the pleural cavities on both sides, the chest viscera lie in the middle layer of connective tissue, the mediastinum, which represents the continuation of the visceral space of the neck. This axial connective tissue layer extends from the base of the skull to the diaphragm, offers space for the organs, and leads the vessel and nerve bundles from the head into the thorax or from the thorax through the anterior and posterior scalene gap to the arm.

The intrathoracically positioned endothoracic fascia and parietal pleura form a functional unit as fascial derivates and are linked to the mediastinum with the diaphragm, pericardium, trachea, and other structures. As in many cases with connective tissue, they are considered together, as follows, and their vessels, nerves, and lymph supply are dealt with separately.

Endothoracic fascia

At the upper thoracic aperture the deep posterior and middle anterior neck fascia are in direct contact with the endothoracic fascia; this lines the wall of the pleural cavity – apart from the mediastinum. Note that the endothoracic fascia in the area of the chest wall, the pleural dome, and the diaphragm is composed of a layer of sometimes loose subserous connective tissue from the parietal pleura and passes into the connective tissue of the mediastinum at the sternum and the vertebral column. At the chest wall, the endothoracic fascia extends between the costal pleura and the ribs and the thoracic fascia (fascia of the intercostal muscles and the transverse thoracic muscle). The intercostal nerves and vessels run into the endothoracic fascia in the posterior part, and in the anterior part the thoracic vessels above the third rib. The section between the diaphragmatic pleura and the diaphragm is also described as the fascia phrenicopleuralis.

Pleural cavity

The serous pleural cavity borders the mediastinum on both sides; it contains the lungs, which are completely covered by the sac-like pleura visceralis (pulmonaris). At the lung stem (main bronchus, vessels and nerves), the parietal pleura crosses into the pleura visceralis. The respiratory movements of the chest wall and the diaphragm cause the volume of the pleural cavity to alternately increase and decrease in size. In the pleural cavity, the pleura visceralis moves against the pleura parietalis, from which it is separated by a capillary space filled with a few milliliters of serous fluid. As long as the pleural cavity is intact, the lungs exert strong traction on the parietal pleura by means of capillary strength over the surface during respiration; the parietal pleura, for its part, is adapted by its connective tissue texture to this type of demand.

Parietal pleura

As the costal pleura, the parietal pleura lines the chest wall, as the mediastinal pleura it lines the lateral surface of the mediastinum, and as the diaphragmatic pleura it lines the diaphragm. The parietal pleura is significantly more firmly fixed to its surroundings than the visceral pleura because of the demands made on it by mechanical traction. The costal pleura is firmly attached to the endothoracic fascia and the diaphragmatic pleura to the phrenicopleural fascia.

Bulges in the pleural cavity serve as reserve space – pleural recess – into which parts of the lungs can slide during deep inspiration.

The costodiaphragmatic recess is a deep channel between the costal pleura and the diaphragmatic pleura at the lower margin of the chest wall. During deep inspiration, this reserve space unfolds and the lungs enter up to two intercostal spaces deep into the recess.

The costomediastinal recess also unfolds during inspiration, when the heart sinks down and the ribs

lift and make available another, smaller, reserve space for breathing.

Pleural dome/cervical pleura

The parietal pleura protrudes like a dome out of the upper thoracic aperture, rising up to about 2–3 cm because of the diagonal position of the first rib. The free lateral area of the pleural dome is strengthened by the fibre-rich suprapleuralis membrane, which splits off from the endothoracic fascia. The suprapleuralis membrane is joined firmly to the first rib with tracts of strong connective tissue. At the level of C6/7 arise tracts of fibers from the deep neck fascia, which insert at the pleural dome as the pleurovertebral ligament. They prevent the pleural dome falling during inspiration. In contrast to the middle and lower lung segments, the tops of the lungs only unfold slightly, as they have no immediate recess as a reserve space.

The subclavian artery and vein arch over the pleural dome, while the phrenic nerve runs along its medial side. The cervicothoracic ganglion of the cord of the sympathicus margin lies posterior to the tip of the cervical pleura at the head of the first rib.

Fascial structures in the mediastinum

The mediastinum extends from the posterior side of the sternum and the bordering ribs to the anterior side of the thoracic spine and is delimited on both sides by the lateral mediastinal pleura (Fig. 1.7.3). The superior mediastinum extends from the upper thoracic aperture to about the lower margin of T4 and becomes the inferior mediastinum in a caudal direction. Three sections can be distinguished here from the front to the back:

- The anterior mediastinum, a very flat space filled with connective tissue between the sternum and the pericardium.
- Middle mediastinum containing the pericardium and the heart.
- Posterior mediastinum, the actual connecting space between the thorax and the neck, with the esophagus, thoracic aorta, azygos and hemiazygos veins, thoracic duct, and the sympathicus trunk.

The pericardium encloses a serous cavity, which completely surrounds the heart (Fig. 1.7.4). It consists of an outer fibrous pericardium and an inner serous pericardium. On the wall of the aorta at the





Fig. 1.7.4 • Connection of the cervical fasciae into the upper thoracic outlet. Note the position of the membrana bronchopericardiaca.

pulmonary trunk and the superior vena cava, the serous pericardium wraps around the visceral sheet of the heart muscle, the epicardium, which covers the heart muscle as a serous skin. Between the serous pericardium and the epicardium is a space filled with serous fluid.

The fibrous pericardium consists of dense, crisscrossing collagen fibers, which are interspersed by elastic trellis-like grids. This texture allows the physiological reshaping of the heart during its action and at the same time prevents overextension of the pericardium.

The fibrous pericardium is firmly fixed to the diaphragm at its lower surface and to the central tendon in the surroundings of the inferior vena cava. Medially, the pericardium is almost completely covered by the mediastinal pleura and is attached to it by connective tissue. The phrenic nerve runs through this connective tissue together with the pericardiophrenic vessels to the diaphragm.

From the posterior surface of the sternum at the level of the manubrium the sternopericardiacum superius ligament splits from the middle neck fascia to the upper margin of the pericardium, and at the level of the xiphoid process the sternopericardiacum inferius ligament runs to the attachment of the pericardium at the diaphragm.

Behind the pericardium lies a frontally situated connective tissue sheet, the bronchopericardiac membrane, which stretches out with its strong connective tissue fibres between the bifurcation of the trachea, the initial part of the main bronchi, and the upper diaphragmatic fascia.

Fascia of the abdominal wall

Structurally, there are three layers to the abdominal wall (Fig. 1.7.5):

- A superficial abdominal fascia lying on the area below the skin and the subcutis fatty tissue as part of the general fascia.
- A middle layer consisting of very closely attached structures of flat abdominal muscles and their aponeurotically formed muscle fascia. With the abdominal muscles it closes the bony aperture that is delimited by the lower thoracic aperture and the upper pelvic margin as well as the posterior part of the lumbar spine. This middle layer is covered by the inner abdominal fascia.
- A deep layer that contains the posterior retroperitoneal space and includes a powerful layer of connective tissue and is delimited by the parietal peritoneum as far as the peritoneal space.

Superficial layer

Caudally, the pectoral fascia continues as part of the superficial body fascia, superficial abdominal fascia, into the muscle tendon sheet of the M. obliquus externus abdominis, which for its part turns into the fascia lata of the thigh. It is firmly fixed in the area of the linea alba with the external aponeurosis, and in the inguinal area with the inguinal ligament.

In men, this fascia continues as the fascia spermatica externa at the lateral inguinal ring together with the aponeurosis of the M. obliquus externus abdominis and surrounds the spermatic cord. In women, delicate connective tissue fibers follow the teres uteri ligament, which runs through the inguinal canal and is affixed to the labia majora.

Middle layer with transverse fascia

This is made up of the flat lateral abdominal muscles (M. obliquus externus abdominis, M. obliquus externus abdominis, and M. transversus abdominis), the straight anterior muscle (M. rectus abdominis), and a posterior muscle (M. quadratus lumborum, see fascia transversalis). The flat muscles are separated from each other by their muscle fascia and the thin matted connective tissue layers between. On the anterior side, the flat muscles follow the aponeuroses of the flat sheets of tendon, which weave through in the midline and join with the opposite side. This is where the long ligament of the linea alba arises and extends from the symphysis to the xiphoid process. In the area of the navel, the other crossing collagen fibers are arranged in a ring to form the annulus umbilicus, which is strengthened at the rim by collagen fibers running in a circle.

The M. rectus abdominis is fixed in position by a very strong connective tissue sheath – the vagina



Fig. 1.7.5 • Composition of rectus sheath and alignment of fascia transversalis.

musculi recti abdominis – and guided during contraction into this sheath.

The anterior purely tendinous sheet of the rectus sheath is formed from the external aponeurosis and the anterior sheet of the internal aponeurosis and strengthened at the level of the arcuate ligament by the transverse aponeurosis, which is now anterior. The posterior sheet is formed as far as the arcuate ligament by the posterior sheet of the internal aponeurosis, the transverse aponeurosis, and the transverse fascia. Below the arcuate line, the posterior wall of the rectus sheath is still formed by the transverse fascia.

The transverse fascia is the internal abdominal fascia, which covers the anterolateral inner surface of the musculoaponeurotic wall of the abdominal space. It is joined with the subserous connective tissue of the parietal peritoneum and is loose and mobile in places and fixed and immobile in others.

In the area of the arcuate line as well, it continues until it attaches to the inner contour of the linea alba. In this area it does not follow the fascia of the M. transversus abdominis, which is involved in the construction of the anterior sheet of the rectus sheath. Cranially, the transverse fascia covers the abdominal surface of the diaphragm. In a posterior direction it continues as a thin layer on the fascia of the M. quadratus lumborum and M. psoas major. At the anterior caudal part, the transverse fascia is fixed to the inguinal ligament and turns into the fascia of the M. iliacus. Above the inguinal ligament is the inner inguinal ring where the transverse fascia inserts and surrounds the spermatic cord, the epididymis and the testicles as the fascia spermatica interna.

Deep layer

With the posterior section of the parietal peritoneum, this delimits a connective tissue retroperitoneal space (Fig. 1.7.6). Its internal relief is characterized by the lumbar spine, which protrudes into the median plane and the muscle bellies of the major psoas muscle on both sides. Laterally from the psoas this space extends to a deeper channel, the renal bed containing the kidneys and the adrenal glands.

In the area of the adipose capsule it can be up to 5 cm thick or more, depending on nutritional condition. Also in the medial retroperitoneal space lie the great axial vessel and nerve bundles, such as the abdominal aorta, inferior vena cava, the lumbar part of the truncus sympathicus, large autonomic plexus, and the initial part of the cisterna chyli; in the posterior and lateral sections the subcostal nerve, the nerves from the lumbar plexus, and segmental blood and lymph vessels run in an anterior and caudal direction.

In the anterolateral direction it narrows to a thin layer of connective tissue between the transverse fascia and the parietal peritoneum.

The parietal peritoneum and the retroperitoneal space pass into the lesser pelvis at the linea terminalis and end at the pelvic diaphragm, which forms the caudal conclusion for both spaces.



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Visceral fascia

Frank H Willard

Introduction

The organ systems of the body, whether visceral or somatic in nature, are composed of highly differentiated tissue and require an elaborate support system for their maintenance. This sustentacular system is a connective tissue network comprised of irregularly arranged collagen and elastin fibers with their supporting cells embedded in a matrix of glycoproteins, all of which is termed fascia. The density of fibrous elements is highly regionally variable as well as individually variable. The role of fascia as a packing or investing tissue, surrounding and protecting organ systems, seems well accepted in the textbook literature (Gardner et al. 1986; Drake et al. 2010).

Although fascia has been described in several academic treatises (Gallaudet 1931; Singer 1935), there still remains much confusion regarding distribution and naming surrounding the fascias of the body cavities (Skandalakis et al. 2006). A previous chapter in this text suggested that there are four primary fascial layers in the body: (1) pannicular (often termed superficial), (2) axial and appendicular (often termed deep or investing or muscular fascia), (3) meningeal fascia surrounding the central nervous system, and (4) viscera (or splanchnic) fascia surrounding the body cavities and packing around the internal organs (Chapter 1.2). This current chapter will examine the visceral fascias of the body and attempt to present a unifying concept concerning their organization and continuity.

Visceral fascia

Extending from cranial base to pelvic basin and lining the body cavities, visceral fascia is by far the most complex of the four main layers of fascia. Embryologically, this layer of fascia, termed mesenchyme, is derived from the splanchnic tissue and it is into this loose matrix that the body cavities – pleural, pericardial, and peritoneal – expand in size. As this expansion occurs, the visceral fascia becomes compressed outward against the somatic body wall as well as consolidated medially along the midline. In the adult, the composition of visceral fascia is typically described as loose, irregular, connective tissue containing a varying amount of adipocytes (Plate 1.8.1).

Functionally, visceral fascia provides the packing tissue for the midline structures of the body. The midline fascia forms a column that extends from its attachments to the cranial base, through the cervical region into the thorax, where it occupies the mediastinum. At the diaphragm, this column passes through the aortic and esophageal openings to enter the abdomen. Descending through the abdomen into the pelvic basin, the midline fascia forms a continuation of the mediastinum. In the pelvic basin, the visceral column of fascia surrounds the midline structures. This entire mediastinal region of the body contains the major vasculature, such as the aorta, the caval venous systems, and the thoracic duct, as well as the great abdominopelvic plexus of autonomic nerves. These structures and their branches become invested in fascia layers, which accompany the neurovascular bundles as they extend outward to reach individual organ systems (Anderson & Makins 1890).

Early studies had suggested four layers of visceral fascia are present in the walls of the body cavities: (1) muscular fascia surrounding the body wall muscles (at one time termed "parietal fascia"; see discussion in Thompson 1901; Derry 1907a,b); (2) the fascia forming neurovascular sheaths; (3) fascia surrounding individual organs; and (4) fascia underlying the pleural and peritoneal linings (reviewed in Hollinshead 1961). The muscular or "parietal" fascia is essentially the axial and appendicular fascia described in Chapter 1.2. In general, the remaining fascial layers can be considered to form an extensive visceral fascial matrix.

Sentinel descriptions of visceral fascia can be found in the Journal of Anatomy and Physiology by Anderson & Makins (1890). These original descriptions emphasized the continuity of visceral fascia from the nasopharyngeal and cervical region, through the thorax and abdomen, to levator ani in the pelvic region. However, most of the recent anatomical research concerning visceral fascia has been done from a surgical perspective designed to solve clinical problems involving access into a specific region or excision of tissue in cases of neoplastic growth (for example, see Garcia-Armengol et al. 2008). Although the finegrained analysis of individual fascial planes is necessary from a surgical perspective, the narrow focus of these studies tends to obscure the overall picture of this continuous fascial matrix in the body. In the remainder of the chapter, the general organization of visceral fascia in the cervical, thoracic, abdominal and pelvic areas will be presented. Although in any of these regions there are multiple and complex arrangements, for the sake of an overview, we will attempt to generalize as much as possible.

Cervical visceral fascia

In the cranial region, visceral fascia surrounds the pharynx and its attachment to the cranial base. Superiorly, visceral fascia includes the pharyngobasilar and pharyngobuccal fascia and, as such, fuses to the cranial base surrounding the attachments of the superior constrictor muscles (Last 1978). Cervical visceral fascia extends inferiorly into the neck, surrounding the nasopharynx, oropharynx, and remaining cervical viscera. Thus at the cranial base, cervical fascia has a

flared opening surrounding the nasal passageways and the mouth (Plate 1.8.2, Section 22 and 46).

In the neck, visceral fascia incorporates such regional fascias as pretracheal, retropharyngeal, and alar (carotid sheath) fascias as well as the fascia surrounding the thyroid cartilage and thyroid gland (Plate 1.8.2, Section 66 and 86). Thus, visceral fascia can be conceived of as a continuous vertical sleeve lying internal to the hyoid muscles, anterior to the longus muscles, and extending into the thorax.

Thoracic visceral fascia

Upon entering the thorax, the visceral fascia is forced to accommodate the two pleural cavities; this it does by flattening on to the thoracic wall, where it is termed endothoracic fascia (Plate 1.8.2, Section 112). Centrally, visceral fascia expands in bulk and forms the packing substance of the mediastinum (Plate 1.8.2, Section 112 and Plate 1.8.3, Section A). In the mediastinum, visceral fascia surrounds the great vessels of the heart and thickens to become the fibrous pericardium anteriorly, while posteriorly, the visceral fascia forms a loose matrix surrounding the aorta, esophagus, trachea and primary bronchi, and the thoracic duct. This matrix is very loose to allow distension of the esophagus upon swallowing. Normally, no significant condensations of fascia are present in this region, leastwise they would lead to dysphagia. Finally, visceral fascia surrounds the bronchi as they pass through the root of the lung; this fascia becomes continuous with the stroma of the airways and the septa of the lung.

Abdominal visceral fascia

Visceral fascia also accompanies the esophagus and aorta into the abdominal cavity. Here, the visceral layer spreads outward to surround the peritoneum, where it is termed endoabdominal fascia posteriorly and transversalis fascia anteriorly. The endoabdominal fascia thickens significantly along the posterior midline and forms a vertical column analogous to the mediastinum of the thoracic (Plate 1.8.3, Sections B & C). The inset in the lower left corner of Plate 1.8.3 shows the posterior body wall of an 84-year-old female after removing all of the peritoneal organs. The visceral fascia forms a curtain over the body wall that thickens noticeably at the midline, where it covers the major vascular and neural channels such as the abdominal aorta and the inferior vena cava. Extensions of the abdominal mediastinal fascia pass into the mesogastrium, mesentery, and mesocolon to reach the visceral organs of the abdomen. It is along this pathway that the blood supply, innervation, and lymphatic channels reach the peritoneal organs of the abdomen. This situation is remarkably similar to that seen with the mediastinum of the thorax, where visceral fascia invests the structures in the root of the lung and accompanies these structures as they pass deep into lung tissue.

On the posterior body wall a particularly thick mass of fascia surrounds the kidneys. This perirenal fascia has been termed Gerota's fascia. It expands off the midline following the renal vasculature to form a large mass, dense in adipocytes, surrounding the capsule of the kidneys. Posteriorly, the perirenal fascia blends with the axial (investing) fascia of the psoas muscle and the quadrates lumborum.

Pelvic visceral fascia

In the pelvic basin, the endoabdominal fascia is continuous with the endopelvic fascia, which then surrounds the inferior region of the peritoneum. The inferior border of the endopelvic fascia is the pelvic diaphragm, composed of the levator ani and coccygeus muscles. The pelvic diaphragm itself is lined with axial or investing fascia derived from the somatic body wall. Inferior to the pelvic diaphragm is the ischiorectal (ischioanal) fossa; it is packed with pannicular fascia (Plate 1.8.3, Section D and Plate 1.8.4). The anterior and inferior border of the endopelvic fascia fills the retropubic space surrounding the base of the urinary bladder.

At the level of the sacral promontory, the visceral (endoabdominal) fascia forms a median fold surrounding the hypogastric plexus (presacral nerve) and two slightly more lateral folds surrounding the common iliac vessels and associated lymphatic channels. Inferior to the sacral promontory, the median fold divides with the hypogastric plexus to sweep laterally, joining the vessels in the lateral fold. This allows the visceral (endopelvic) fascia to surround the midline organs – rectum, reproductive organs, and urinary bladder (Plate 1.8.3, Section D and Plate 1.8.4).

As in the thorax and abdomen, the endopelvic fascia thickens on the midline, where it again forms a mediastinum surrounding these organs. The endopelvic fascia serves as a conduit over which the major organ systems in the pelvic basin receive their blood supply and innervation as well as their lymphatic drainage. In the female, extensions of the pelvic mediastinal component of the visceral fascia form the core of the broad ligament and condensations of this visceral fascia form the transverse cervical ligament of the uterus. From the cervix of the uterus, posterolaterally directed bands of visceral fascia form the sacrouterine ligaments that reach back to the sacrum and underlie the prominent rectouterine folds. In both sexes, condensations of visceral fascia surround the rectum where it has been termed the mesorectum (Havenga et al. 2007; Garcia-Armengol et al. 2008).

Summary

Visceral fascia can be traced from the cranial base into the pelvic cavity. It forms the packing surrounding the body cavities where it is compressed against the somatic body wall. It also forms the packing around visceral organs, many of which it reaches by passing along the suspensory ligaments such as the mesenteries. This fascia also functions as a conduit for the neurovascular and lymphatic bundles as they radiate outward from the thoracic, abdominal, and pelvic mediastinum to reach the specific organs.

Visceral ligaments

A note here is necessary concerning the use of the word "ligament" when referring to structures found in the body cavity. Visceral ligaments are in no sense of the word similar to the ligaments seen in the somatic body parts. Ligaments in the somatic portion of the body are structures that join bone to bone and are composed of dense, regular, connective tissue, which is itself surrounded by a thin periligamentum of investing fascia, typically part of the axial or appendicular fascia. The word "ligament" used for structures found in the body cavities, such as pulmonary ligaments, Treitz's ligament, transverse cervical ligament, or broad ligament refers to a loose condensation of visceral fascia and in some cases surrounded by a thin serous membrane, critically these are all irregular connective tissue of varying density and thickness. Visceral ligaments generally are nowhere near as strong as somatic ligaments, nor are they as clearly defined on dissection. Unlike ligaments in somatic tissue, visceral ligaments typically function to carry blood supply and innervations to an organ system or to loosely anchor an organ in the body cavity. Visceral ligaments also need to be distinguished from fibrotic adhesions that typically develop secondary to irritation and inflammation.

Adhesions

Fibrotic adhesions derive from areas of chronic inflammation (Wynn 2008). Activated immune cells release cytokines that stimulate fibrocytes to generate additional collagen during the repair

process. The collagen laid down is irregular in arrangement and is thus similar to fascia in its construction. When excessive, these bands of increased collagen can form adhesions that can reach pathological proportions. Adhesions can occur in any region of the body, either visceral or somatic. In the abdomen and pelvis, adhesions can envelop the tubular bowel and be strong enough to obstruct movement within its lumen or interfere with reproductive organ functions. Similarly, adhesions forming in and around synovial sheaths in the carpal tunnel can interfere with movement of the digital flexor tendons.

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Membranous structures within the cranial bowl and intraspinal space

1.9

Torsten Liem Ralf Vogt

Embryonic growth dynamics of the dural membrane according to Blechschmidt

Understanding the dynamics of embryological development enables us to understand many structural, physiological, functional, and dysfunctional interrelationships that are important for diagnosis and therapy. In this chapter you will get an idea of the peculiar dynamic of meningeus growth; the developmental dynamics of the dura in interaction with the development of other tissue structures. With this information you can understand structural dysfunctions found during investigations. Furthermore, you can perceive, feel, understand, and treat those dysfunctions in relation to the time-factor, the dynamics of pre- and postmatural dependencies, and form-building processes.

Blechschmidt (1973, 1978) advises that connective tissue forms according to the environmental forces at hand (Figs 1.9.1-1.9.3). The wellvascularized nervous system creates different biodynamic fields that lead to tensile stress in the back of the developing embryonic spine and to compression in the front of it. In the course of that development, the base of the skull beneath the brain is flattened and compressed and is formed as cartilaginous pre-structures (Fig. 1.9.4). The cranial roof is formed out of skin under tensile growth-stress and the bony structures are created by dermal ossification of flattened and tensed membranous connective tissue. Because of that development, the outer dura mater is a strongly anchored to the inside of the cranial bones. With increasing excentric growth

of the cerebrum, the tensile resistance of the antibasal and laterodorsal cranial walls increases gradually, so that the brain regions bend away from each other. The brain fissures develop, in which falx and tentorium become denser. Falx cerebri and tentorium cerebella develop by compression of mesenchymal tissue between the brain hemispheres, respectively between the cerebrum and cerebellum during embryonic brain growth.

The embryonic heart follows the diaphragm in a descending movement, while the brain ascends in its growing process. The meningeal membranes envelop and support the brain and the spinal cord. They consist of three layers: the inner layer is the pia mater, then follows the arachnoidea, and the outer layer is formed of the dura mater on the inside of the skull and spine.

Intracranial membrane system

Pia mater (soft inner layer of the dural membrane system)

The pia mater, which contains vessels, is the innermost layer of the three meningeal membranes. It consists of a thin layer of connective tissue with a lot of elastic fibers and is very close to the gyri of the brain substance itself, but is not fused with it. Vessels enter the brain from it. Furthermore, it creates the choroid plexus, a network of bold vessels, which enter the brain ventricles and produce the cerebrospinal fluid (CSF).



Fig. 1.9.1 • Influence of the brain on the development of the cranium according to Blechschmidt. Schema of brain portions of an embryo of 28 mm. 1: Cerebrum, 2: diencephalon, 3: midbrain, 4: cerebellum, 5: medulla oblongata. Modified from Blechschmidt, 1978, with permission.



Fig. 1.9.2 • Influence of the brain on the formation of the cranium according to Blechschmdt. Expansion of the developing brain induces formation of a thick fascial membrane (dura) between the cerebellum and cerebrum, as well as of a smaller membrane between the frontal and temporal lobes of the cerebrum. Convergent arrows: developmental push of the brain is met by tensional resistance of dural membranes. 1: Cerebellum, 2: midbrain, 3: right cerebral hemisphere. Embryo size approx. 29 mm. Modified from Blechschmidt, 1978, with permission.

Arachnoidea (middle layer of the meningeal membranes)

The arachnoidea has a gauze-like spongy structure and can be differentiated into two layers. The outward layer beyond the dura mater is separated from it by a thin cleft, the **subdural space**, which contains



Fig. 1.9.3 • View of right and left dural membranes in the frontal area. Left and right dural membranes are shown. Arrows: developmental push of both cerebral hemispheres. 1: Falx cerebri as very tough fascial membrane. Embryo size approx. 29 mm. Modified from Blechschmidt, 1978, with permission.



Fig. 1.9.4 • Lateral view of dural membranes. The densation field at the basis of the dural membranes leads to later development of the cartilaginous cranial base (pointed area). Convergent arrows: dural membrane capable of resisting tension. Smaller arrows: developmental expansion of brain. Embryo size approx. 29 mm. Modified from Blechschmidt, 1978, with permission.

some veins and nerves. The inner layer consists of a trabecular framework.

Between the arachnoidea and pia mater there is the **subarachnoidal space**. Both membranes are connected by the trabecular framework in the subarachnoidal space. It is filled with CSF and forms the outer CSF spaces. It is narrow at the apex of the skull, but because the arachnoidea follows the pia mater, larger caverns are created in areas where the brain and bony cranial wall are further apart, at the base of the cranium. These widened, CSF-filled spaces are called **cisterns** (cerebromedullar cistern, interpeduncular cistern, chiasmatic cistern, cisterna ambiens). Outgrowths of the arachnoidea, the arachnoidal villi, project into these spaces in the venous drainage of the inner skull, particularly that of the sagittal sinus. Through these villi the CSF can flow out into the venous system. The arachnoidea continues as the perineurium (nerve sheet) of the nerves passing through.

Dura mater (hard outer layer of the meningeal membranes)

The dura mater consists of dense, uneven, very strong connective tissue with a lot of collagenous fibers. It is very tight and not permeable to the CSF. A special layer of flat fibroblastic cells without extracellular collagen and extracellular space can be found at the transition between the dura and arachnoidea (Haines et al. 1993). This dural borderline can be divided into **periostal dura** and **meningeal dura**. There is no epidural space like that in the spine. The dura mater continues as the **epineurium** of efferent nerves that leave the skull. The skin of the scalp and the dura are connected by emissary veins, which may be under enormous tension. In the region of the ethmoid bone cells, the tegmentum and sinus sigmoideus, the dura is very flat.

The inner layer (dura meningealis) is weaker in its structure than the outer dura layer or the arachnoidea (Haines et al. 1993). The dura of an adult can resist stronger forces than that of a newborn (Dragoi 1995). According to Arbuckle (1994), this fibrous structure enables the intracranial dura and the intraspinal dura to transmit different forces. These "pathways for transmission of forces" find their way by the fibrous structures of the dura, the socalled "stress-fibers". Arbuckle defines the following groups: horizontal, vertical, transversal, and circular. The direction of fibers in the dura mater cranialis can possibly be traced back to the results of mechanical forces during embryonic development, when collagenous fibers are brought into line by stress forces (Hamann et al. 1998). Between the dura periostale and dura meningeale there are some other important structures apart from the venous blood sinuses.

• Endolymphatic bag: a sort of baglike tube, part of the ductus endolymphaticus, which is located at the rear wall of the petrous bone between the two dura layers.

- Meningeal arteries: terminal branches of the carotid arteries.
- Sympathetic nerve fibers travel between the dural layers of intracranial vascular walls (coming from the superior cervical ganglion and the plexus caroticus), as do the sensitive fibers of the 5th and 10th cranial nerves and of the 1st and 2nd cervical nerves.
- Trigeminal cave (Meckel's cavity): a peculiar cavern of the dura for the ganglion of the 5th cranial nerve (also called trigeminal nerve ganglion, semilunar ganglion, or Gasserian ganglion), located at the front side of the apex of the petrous part of the temporal bone above the foramen lacerum.

Horizontal and vertical dural system

The membranes inside the cranium are connected anatomically as well as functionally and so affect each other. Due to their different positions and orientations they can be divided into four septa: the falx cerebri, tentorium cerebella, falx cerebelli, and diaphragma sellae. Collagenous fiber bundles of the falx cerebri and falx cerebella form arcs in the anterior, intermediate, and posterior regions that cross each other perpendicularly. In the course of growth, fiber organization modulates from a 45° angle to a 90° angle (Dragoi 1995).

According to Delaire (1978), the horizontal system (tentorium cerebella and diaphragma sellae) acts as a clamp or tightener for the cranial base, while the vertical system (falx cerebri and falx cerebella) tightens the cranial vault. Delaire argues that the tension in the horizontal and vertical dural system is maintained and regulated by the continuous tone of the neck muscles and the sternocleidomastoid muscle, but this is controversial. According to Ferré et al. (1990), movement of the neck muscles can be transmitted via the aponeurotic part of the scalp. However, only very weak and secondary movement can be sensed there, even though the aponeurotic structure of the scalp is clearly movable, unlike the highly immobile falx cerebri and falx cerebelli. According to Sutherland (1939), tension in every part of the membranous system can have an influence at all other parts of that system due to the structural bond. The dural membranes safeguard the integrity of the cranium, especially in early childhood, by way of their attachment to the cranial bones, in case of exposure to force. Additionally, it is thought that involuntary "jointed" movements of similar cranial bones are regulated via synchronicity with the rhythm of the primary respiratory mechanism. Every difference in tension at one side of the membrane alters the complete unit and leads to a new balance.

The falx cerebri divides each cranial hemisphere from the other. Its anterior downward border attaches at the gallic crest of the ethmoid bone; continues across the foramen caecum, the frontal crest and the borders of the sulcus for the superior sagittal sinus of the frontal bone; then follows the parietal crest of the parietal bones, the sulcus sagittalis of the occipital bone, until it gets to the internal occipital protuberance. There the falx is involved in the formation of the straight sinus and at this structure both layers of the falx cerebri detach from each other and continue into the tentorium cerebelli. At the parietal bones it forms the sagittal sinus, while its free margin contains the inferior sagittal sinus.

The tentorium cerebelli ("La Tente", Winslow 1732) divides the cerebrum and cerebellum from each other and stretches over the cerebellum like a tent. Above the tentorium there are, apart from the cerebral hemispheres, the subcortical nuclei and the thalamus. Like the falx cerebri and falx cerebella, the tentorium starts at the straight sinus and is attached there, too. The tentorium is attached posteriorly at the inner occipital protuberance and at both sides at the transversal groins of the internal occipital bone, where it forms the transverse sinus. Sideways, it leads along the sinus across the parietomastoideal suture and attaches for a short distance with its superior layer to the inferior back edge of the parietal bone. Meanwhile, the inferior layer can be found as an attachment to the mastoid process of the temporal bone. This is an important place, because its attachments continue from there along the mastoid process and the superior edge of the petrous part of the temporal bone, where it encloses the superior petrosal sinus. The inferior lateral layers are attached to the clinoid processes of the sphenoid bone. The free internal borders of the tentorium continue in an anterior direction, cross over the anterior inferior layers, and are attached to the anterior clinoid processes of the minor wings of the sphenoid bone. Here, where the internal branches of the tentorium cross the outer branches, the abducent nerve can be found. Obviously, the abducent nerve can be disturbed by tentorium tension. A large oval opening, the tentorial incisura, is occupied anteriorly by the midbrain and the interpeduncular cistern, and posteriorly by the rounded end (or splenium) of the corpus callosum.

Extracranial membranous system

Pia mater spinalis

This membrane contains vessels and nerves. From the pia mater, on both sides of the spinal cord, a connective tissue plate, the **denticulate ligament**, leads to the dura mater spinalis. It fixes the spinal cord and divides the two spinal nerve roots from each other. The pia mater follows the inner side of the spine and ends as a long, slender filament, the filum terminale, at the dorsal surface of the coccygeus bone.

Arachnoidea spinalis

The arachnoidea has "extremely poor vascular and nerve supply" (Doppmann et al. 1969). It accompanies the dura mater to the nerve roots and allows it to be bathed in cerebrospinal fluid. The membranes follow the nerves to the intervertebral foramina, where they envelop the spinal ganglia. The arachnoidea then continues as the **perineurium** of the spinal nerve.

Dura mater spinalis

The dura mater spinalis forms a tight tube of collagenous fiber, leading from the foramen magnum of the occipital bone, where it is fixed, to the canalis sacralis, transferring at S3 Level into the filum terminale, which attaches in a fanlike manner at the periosteum of the coccygeus bone. Its course follows the curvatures of the vertebral channel. At the border between the foramen magnum and the vertebral channel there are two layers of dura mater: the outer, periosteal layer, and the interior layer, which is the true dura mater. Between the two layers, there is epidural space, which enables a sliding motion between the dura and the vertebral channel. The epidural space is a fictitious cavity (Parkin & Harrison 1985; Newell 1999), a "true potential space" (Breig 1960). In the upper cervical spine the epidural fat tissue is less developed (Breig 1960). There is no complete consensus concerning the structure of human dura mater, especially regarding the direction of the collagenous fibers, which are responsible for biomechanical functions. The spinal dura mater is longitudinally orientated (Patin et al. 1993), and the gills, consisting of elastin and collagen, are directed longitudinally (Patin et al. 1993; Runza et al. 1999). Longitudinal tensile strength and stiffness is much more than the transversal stress. Longitudinal stress, occurring from longitudinal spinal movements, is for the most part carried by the longitudinal collagenous fibers and this leads further to upwardly or downwardly situated neighboring structures.

In the upper cervical area connective tissue follows a transverse course (von Lanz 1929). According to a study of the spinal dura mater in dogs, collagenous fibers are organized into longitudinal bundles, which are straight when stretched and curly when in a relaxed state (Tunituri 1977). Elastic fibers are orientated multidirectionally and are like a network. In the rear part of the spinal dura mater, elastin makes up 13.8% of the total, and 7.1% in the frontal part. In the thoracic region the proportion of elastin is higher than in all other regions (Nakagawa et al. 1994).

The spinal dura mater is largest at the level of the craniocervical junction and at the level of the lumbar spine (Lazorthes et al. 1953).

The spinal dura mater is only loosely attached to the spinal channel, except for its cranial and caudal fixations, so enabling movement of the dura against the canal (Parkin & Harrison 1985; Hogan & Toth 1999). It is supposed that the dura is able to transmit the fine movements of the CSF from the cranial bowl to the sacral bone. As a continuation of the falx cerebelli and the intracranial dura, the spinal dura mater inserts firmly into the occipital foramen. According to von Lanz (1929), the dura is fixed at the following structures in particular:

- At the basilar part of the occipital bone (leading through the membrana tectoria)
- At the ligamentum transversum atlantis
- At the ligamentum longitudinale posterius
- At the periosteum of the squama occipitalis at the arc of the atlas (C1) and axis (C2) joints
- At the atlanto-occipital und atlantoaxial joints.

Furthermore, the dura is attached firmly at the third cervical vertebra (Klein 1986; Upledger & Vredevoogd 1994); according to our own investigations, this is not always so (Liem 2000).

Ligamenta craniale durae matrae spinalis

The attachment of the spinal dura mater to the occipital bone, supported by fibers between the arc of the atlas (C1), the arc of the axis, and the posterior rim of the atlanto-occipital joint and the foramen magnum are called "ligamenta craniale durae matrae spinalis" according to von Lanz (1928). Rutten et al. (1997) found attachments to the ligamentum flava of C1 to C3 and the deep layer of the nuchal ligament. Rutten et al. (1997), supported by Hack et al. (1995), hypothesize that parts of these ligaments could function as tensioner of the upper cervical spine during motion and that the M. rectus capitis posterior minor controls the tension in that region. That results in failure of the antifoldingmechanism of the dura in the case of muscular injury of that muscle (Klein 1986).

M. rectus capitis posterior minor and M. obliquus inferior

According to Kahn et al. (1992), von Lüdinghausen (1967), and others (Hack et al. 1995; Alix & Bates 1999; McPartland & Brodeur 1999), the spinal dura mater is attached to parts of the M. rectus capitis posterior minor, the inferior oblique muscle, and also the nuchal ligament. This has an effect on the foldingprevention of the dura and, with regard to the nuchal ligament, on the rotatory movements of the skull (Mitchell et al. 1998).

Interspinal ligaments of the dura mater

The attachments from the dura mater spinalis to the interior walls of the spinal canal can be divided into anteroposterior orientated ligaments and lateral orientated connections. As always in medicine, the borders are floating.

In the same way, attachments to the flaval ligament and the posterior longitudinal ligament transmit forces in a sagittal plane. Sometimes they are more a protection for the soft tissues between spinal cord and spine than a real ligament. That is also true for the **Trousseau Fibreux de Soulié**. On the one hand they connect the dura mater with the posterior longitudinal ligament, on the other hand they connect the spinal dura mater with the periosteum. That leads on both sides to a coverage of the anterior epidural venous plexus (Trolard 1888).

A lateral attachment is the **ligamentum** sacrodurale anterius or Trolard's ligament, which is situated between the dura and vertebral bodies and arcs in the lower lumbar and sacral spine. Similarly, **Hofmann's ligaments** are situated between the spinal dura mater and the superficial layer of the posterior longitudinal ligament (Hofmann 1898; Fick 1904; Doppmann et al. 1969; Schellinger et al. 1990). The **ligamentum dorsolateralia duralis** or **Hofmann's lateral ligaments** are described by Spencer et al. (1983) and form a connection between the dural coverage of the spinal nerve and the vertebral periosteum. Spencer et al. called them "lateral Hofmann's ligaments". These are supposed to prevent a posterior evasion of the spinal nerve in case of a pain causing disc protrusion.

These attachments are also known as "meningovertebral ligaments". At the level of every intervertebral foramen are the opercula of Forestier. They are a junction between the dural coverage of the outgoing spinal nerve and the periosteum of the particular vertebra (Trolard 1888; Forestier 1922; Lazorthes 1996; Grimes et al. 2000). The opercula envelop the intervertebral foramen from inside and outside; that means they are situated at the inside and outside of the vertebral canal. The transformidal ligaments embrace the intervertebral foramen along the outside. Giradin (1996) described them as either the greater parts of the opercula of Forestier or as the incomplete opercula or "false ligaments". The ligamentum denticulatum leads from the pia mater to the dura mater and connects the spinal cord from both sides of the occipital bone to the level of L2 with the dura. This is like a supporting structure, which allows the spinal cord to be suspended in the cerebrospinal fluid. The attachment spikes of the ligament cross the subarachnoidal space laterally, penetrate the arachnoidea and are finally fixed at the dura mater in between the dural covers of the spinal nerves (Key 1870). The "rhomboid halter" (Lang & Emminger 1963; Key & Retzius 1981) is a diamond-shaped plate of connective tissue, which embraces lower parts of the afterbrain and the upper spinal cord at its anterior side. It blends with the dura mater together with the topmost spikes of the denticulate ligament. The ventral roots of the 2nd cervical nerve should be situated posterior of the "rhomboid halter" and the ventral roots of the 1st cervical nerve posterior or anterior to it. The downward directed rhombic tip is mostly situated in the region of the fissura mediana anterior at the C4 level (Lang 1981).

All these interspinal ligaments and membranous structures are supposed to support the movement

of the dural tube inside the spine and to prevent folding or other injury-causing mechanisms to the dura or the spinal cord.

Vascularization of the meningeal membranes

Intracranial vascularization

The dural system is vascularized arterially by, above all, the meningeal arteries – terminal branches of the inner and outer carotid arteries. They are situated between dura and bone.

Intraspinal vascularization

The posterior spinal arteries are found in pairs (branches of the posterior inferior cerebellum artery) – the anterior spinal artery (branch of the vertebral artery) and meningeal branches of the intercostal arteries.

Anterior and posterior internal vertebral venous plexuses form in the epidural space. The internal vertebral venous plexuses are embedded in fat that is semiliquid at body temperature.

The valveless plexus veins are of special importance for physiological function and for dysfunction. Via the intervertebral canals they communicate with the lumbar veins, the intercostal veins, the azygos and hemiazygos veins, and with venous plexuses in the nuchal region (marginal and occipital sinuses of the dura mater). These anastomoses enable multidirectional drainage of venous blood without congestion.

Meningeal nerve supply

Intracranial innervations

The upper part of the dural system is innervated mainly by branches of the trigeminus nerve, the lower part by cervical nerves 1 to 3, and branches of the vagus nerve. All meningeal nerves contain postganglionic sympathetic fibers, which have their origin directly or indirectly in the superior cervical ganglion via the internal carotid plexus or the maxillary plexus, accompanied by branches of the medial meningeal artery.

The parasympathetic supply is achieved by the greater petrosal nerve (from the parasympathetic part of the 7th cranial nerve) and branches of the vagus nerve and glossopharyngeal nerve.

Intraspinal innervation

Meningeal branches of the spinal nerves and the nerve plexus of the posterior longitudinal ligament, as well as perivascular plexuses of the root arteries, form the intraspinal innervation.

Tasks of the dural system

- Together with the CSF, the dural system supports and maintains the structure of the brain.
- It protects the skull shape, especially in early childhood.
- Protection in case of mechanical trauma.
- Shock-free absorption of mechanical forces delivered to the skull because of its special construction (together with the elasticity of the bones, the pillar-like construction of the skull, the nasal sinuses, and the attachment of the viscerocranium to the neurocranium (Drenkhahn & Zenker 1994)).

Reciprocal tensile membrane

The dura mater forms the ligamentous apparatus of the bony skull, meaning that both of the dural layers can be seen as a mechanical unit. According to Delaire (1978), the horizontal system (tentorium cerebelli, diaphragma sellae) acts as tensioner for the cranial base, while the vertical system (falx cerebri, falx cerebelli) acts as a tensioner for the cranial roof. Tension of the vertical and horizontal system is maintained and regulated mainly by the tone of the neck muscles and the sternocleidomastoid muscle.

Sutherland (1939) calls the dural membrane system a "reciprocal tension membrane system". That was to show the functional unity of that membrane. The reciprocal tension membrane of the brain and spinal cord is meant to be the structural connection of the single cranial bones with the task of guiding and limiting the range of motion of these bones. By using the structural connection of all membranes, tension in any part of the system is able to influence all other parts. The dural membranes regulate the involuntary articular motion of the cranial bones and the sacral bone due to their attachments at the skull and sacral bone. These membranes, moving reciprocally to each other, are on a permanent quest for optimum balance. Any tension at one end of the membrane changes the complete unit and causes a new balance. It has to be mentioned here too, that changes in breathing will also change tension in the intracranial membranes; for example, the tentorium cerebelli moves in synchronicity with the diaphragm.

Sutherland fulcrum

To maintain the balance of membrane motion and tension in all directions, the membranes have to operate based on a **fulcrum**, or a stable point. These stable points have to be formed to float, in order to move automatically to keep the motion of the cranial bones uniform in case of changes, e.g., of the outer tensile forces.

The center of this intracranial membrane system is a virtual point, which is situated at the uniting point of the tentorium cerebelli, falx cerebri, and falx cerebelli in the course of the straight sinus. This point is also known as **Sutherland's fulcrum** (Magoun 1976) or the **automatic shifting suspended fulcrum**. The dynamic forces affecting the membranes are brought into balance here. The whole intracranial and intraspinal dural tension membrane moves and organizes around this point. And together with that, it centers the involuntary articular motion of cranial bones and influences coming from outside the craniosacral system. "Sutherland's Fulcrum is a central attachment, at the same time mobile and adaptable" (Richard 1978).

Possible effects of abnormal dural tension

- Venous drainage dysfunction of the skull via the venous sinuses.
- Decreased drainage of the brain.
- Vascular supply disorders of brain tissue.
- Disorders in fluctuation of CSF.

- Headache, intracranial and retro-orbital pain via the sensible nerve supply of the dural membranes (CN V, X and 1st, 2nd and 3rd cervical nerves).
- Face pain and abnormal tone of the chewing muscles via CN V and the trigeminal ganglion, which is covered with dura and is vulnerable to dural stress.
- Dysfunction of any cranial nerve and nerve ganglia, e.g., at the cranial openings and the intracranial dural membranes, as well as at the dural coverage of cranial nerves.
- Limitation in movement and mobility of the cranial bones, the sacral bone, and the coccygeal bone.
- Dysfunction of spinal nerves (at the emission points in the dura mater).
- Transmission of dural tension via fascial attachments and the epineurium of the spinal nerve.
- Abnormal tension in one of the dural double layers will always have an effect on the other dural parts.
- Disturbance of the pituitary gland (diaphragma sellae).

Future tasks and open questions

There are still some remaining open questions concerning the real lines of forces within the fascial system of the cranial bowl and spine. Is there really transmission of movement? For what purpose? Where is the motor of these forces? Which effects do we get by using different procedures in the conservation and fixation of specimens concerning tissue characteristics (mainly concerning the transmission of tension/motion)? The surveys performed to date have mostly been with laboratory specimens.

Is it possible to transmit connective tissue tension from outside the dural system to the dura and vice versa? And – if yes – by what means and for which functional and clinical meaning?

These and many more questions have to be answered by future scientific studies. That entails the use of links between medical and engineering sciences like bionic science or the results of biomimetic robotic research and viscoelastic forces (Witte et al. 2004; Doschak & Zernicke 2005; Fernandez & Pandy 2006).

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Diaphragmatic structures

Serge Paoletti

Introduction

The diaphragm is a muscular fibrous structure between the chest and the abdomen. It separates two areas with different physiological functions: an upper area geared to the circulation and the exchange of gases and a lower area intended for metabolic assimilation and elimination. Its role is not just respiratory but, as we will see, it takes part in abdominal movement and the mobilization of fascial transmission, and through the intermediary of these it interacts with the cranial, cervicothoracic, and perineal regions.

Embryology

In week 4 we see the appearance in the embryo of three sections, which will divide the body cavity into the pleural, pericardial, and peritoneal cavities. The first to form is a horizontal layer: the transverse septum of our future diaphragm. It divides the body cavity into a primitive pericardial cavity, the future chest, and in the peritoneal cavity, the future abdomen (see Fig. 1.10.1).

The differential growth of the head, the neck, and the trunk will gradually displace the transverse septum from the neck region in a caudal direction until the definitive placement of the future diaphragm. Its anterior margin finally attaches to the anterior wall of the trunk at the level of the 7th thoracic vertebra, while posteriorly it adheres to the esophageal mesenchyme at the level of the 12th thoracic vertebra. The frontal pleuropericardial folds appear on the lateral wall of the primitive pericardial cavity and grow in a medial direction to unite with each other, as well as with the anterior facet of the mesoblast of the anterior intestine to form the definitive pericardial cavity and the pleural cavities. The muscle fibers participate in the formation of the lower part of the esophagus (Botros et al. 1983). Note that these two cavities initially communicate with the peritoneal cavity through the pleuroperitoneal canals passing behind the transverse septum/ ampullary crest.

At this stage there is fascial continuity between the chest and the abdomen. Thereafter, a pair of transverse pleuroperitoneal membranes develops in an anterior direction to join with the transverse septum/ampullary crest and form with this the definitive diaphragm which closes the pleuroperitoneal canals in the 7th week (Greer et al. 2000). The left pleuropericardial canal is wider than the right one and closes later. The mesenchyme associated with the anterior intestine contracts to form the right and left crura. This closure does not constitute an interruption between the thoracic and abdominal cavities; the diaphragm merely represents an intermediary point between the pleuropericardial and abdominal structures. This continuity is an important element in the transmission of forces and pressures between the regions above and below the diaphragm, particularly during movement of the diaphragm.

At the same time, the myoblasts become differentiated in the transverse septum/ampullary crest and will constitute the origin of the muscular part of the diaphragm. They are innervated by segments



Fig. 1.10.1 • Embryologic development of the diaphragm.

C3, C4, and C5 and on joining constitute the phrenic nerves which follow the migration of the transverse septum/ampullary crest, provide numerous collaterals (as we will see), and innervate the diaphragm, although the peripheral portion coming from the para-axial mesoblast is innervated by the spinal nerves T7 and T12. The definitive diaphragm is formed from four embryonic structures: the transverse septum/ampullary crest, which will provide the central tendon; the pleuroperitoneal membranes; the para-axial mesoblast of the trunk wall; and the esophageal mesenchyme.

During embryonic development, the nonclosure of the pleuroperitoneal canals is the cause of congenital diaphragmatic hernias, which have a frequency of 1/2000 to 1/4000 births. This nonclosure is more often partial than total and the left side is more often affected than the right, in a proportion of four to eight times, probably because the right cavity is wider, closes later, and contains the liver, the capsule of which is dependent on the diaphragm. Genetic factors also play a part in diaphragmatic hernias (Holder et al. 2007).

Organization

Three parts can be distinguished at the level of the diaphragm (see Fig. 1.10.2).

Central part

The phrenic center: This is a fibrous layer shaped like a trefoil, stemming from the transverse septum and comprising three leaflets: the anterior leaflet is the most extensive and very rich in lymphatic vessels. Then there is also one leaflet on the right and another one on the left.

It is formed from two types of fibers arranged in three groups (Menck et al. 1990): fundamental fibers directed sagittally in the anterior leaflet, oblique fibers in the lateral leaflets, as well as the connecting inferior and superior semicircular bands. As they cross, they circumscribe a nonstretchable fibrous opening for the passage of the inferior vena cava. This multidirectional, multilayered conformation reinforces the diaphragm's strength and distributes the forces over the whole surrounding area to reduce the exertion of the vertical components on the abdominal mass. The fibers of the central tendon are made up of three groups.

Peripheral part

This is muscular and radiates from the phrenic center over the whole surrounding area of the ribcage. One can distinguish two important fasciae: the anterior fascia and the lateral fascia. The sternal


Fig. 1.10.2 • Diaphragmatic interactions.

or anterior fascia insert at the xiphoid process and exchange fibers with the triangle of the sternum. The chondrocostal or lateral fascia inserts at the internal superior margin of the last six ribs, where it intercross with the transverse fibers. It then continue to the superior part with the endothoracic fascia and to the subdiaphragmatic part with the transversalis and transverse fascia.

Posterior part

This part is formed by the crura of the diaphragm and the arcuate ligaments.

The crura of the diaphragm

There are two of these: the right crus inserts into the body of L1 and L3 and the corresponding discs; whereas the left crus is shorter and inserts into the body of L1 and L2 and the corresponding disc. The growth of the right crus derives from the root of the mesentery at the level of the muscle of Rouget (circular fibers). Note that this growth sends a branch to the esophageal hiatus and certainly contributes to the constitution of the functional sphincter of the cardia. These crura extend along the common anterior vertebral ligament and form a continuity between the occiput and the sacrum. Crura emerge in an anterosuperior direction from the muscular fibers which intercross at the median line in front of D12 to form the aortic hiatus. There are two accessory crura, right and left, from each side of the main crus, which provide a passage for the splanchnic nerves.

The two arcuate ligaments

The first one consists of medial arcuate ligament or lumbocostal arch: This is a fibrous arcuate ligament running from the transverse apophysis of L1 to the body of L1. It crosses over the anterior facet of the psoas and constitutes a fascial continuity with its fascia.

The other one is the lateral arcuate ligament or lumbocostal arch: This extends from the transverse apophysis of L1 to the apex of the 11th and 12th ribs. It matches the aponeurosis of the transverse and forms a fascial continuation with the abdomen via the transversalis fascia, which itself continues along the pelvic fascia (Lierse 1990). It constitutes the costolumbar hiatus.

Relationships and role

The diaphragm has superior relationships to pericardium, the pleura, and to the lungs. Inferior relationships exist to the right hepatic flexure, the liver, to the large tuberosity of the stomach, spleen, and to the left hepatic flexure. Finally, posterior relationships exist to the third duodenum, to the pancreas, as well as to the greater and lesser splanchnic nerves.

While the principal role of the diaphragm consists of ensuring pulmonary ventilation, it also plays a significant role in the separation of the thoracic and abdominal regions, including the avoidance of the dissemination of infections between the two regions. It is also involved in the hemodynamics, playing the role of a pump encouraging return circulation (Willeput et al. 1984; Verschakelen et al. 1989).

It constitutes a very important fascial relay point ensuring the transmission between the supra- and subdiaphragmatic forces and also constitutes a dampening down factor of these forces, as we will see in the mechanics of the diaphragm.

Mechanics of diaphragmatic contraction

During inspiration, contraction of the diaphragm does not just mobilize the lungs but it also interacts with a number of other structures and functions (see Fig. 1.10.3). As the diaphragm contracts, it has a tendency to flatten and lower. The right part descends less than the left because of the presence of the liver, but contraction on the right is more powerful than on the left (Whitelaw 1987). However, during inspiration, modification of the shape of the diaphragm is very limited because of the rigidity of the central tendon (Boriek & Rodarte 1997). Descent of the central tendon of the diaphragm (phrenic center) is also held back by the pericardial fascial transmission and support for the viscera.

Contraction of the diaphragm is not uniform and the resulting tension is unevenly distributed. During inspiration the greatest tension is in the central part. However, because of the anatomical construction, particularly the central tendon which is formed of three layers of multidirectional fibers (Menck et al. 1990), the greatest part of this tension is transmitted peripherally following the direction of the muscular and fascial fibers, and only a small part is transmitted vertically (Boriek et al. 2005).

Interaction with the rest of the body

The diaphragm is mechanically linked to surjacent and subjacent zones and interacts with them via both an intracavity and exocavity route.

The intracavity route

The forces transmitted by the diaphragm will transfer cephalically and caudally, constituting a functional unity which will act as much from a mechanical as a physiological point of view.

Caudally

The diaphragm constitutes a relay for the endothoracic fascia and the pleura, which continue via the transversalis fascia and the peritoneum, then via the transversalis fascia and the presacral fascia via the peritoneum (Lierse 1990; Skandalakis et al. 2006).

The peritoneum constitutes the pelvic diaphragm formed conversely to the diaphragm; it constitutes a descending fascial continuation, as we have just seen, and is principally constituted by the levator muscle of the anus and the pelvic fascia (Schmeiser & Putz 2000).



Fig. 1.10.3 • Inferior view of the diaphragm.

The thoracic diaphragm and the pelvic diaphragm circumscribe the abdominopelvic cavity and form two inverse domes, which function in synergy.

During the descent of the diaphragm part of the pressure is transmitted, as we have seen, to the abdominal mass and then to the perineum (Boriek et al. 2005). During inspiration the perineum descends to lessen the effects of the pressure. You only have to watch a woman breathing in a procubitus/procumbent position to appreciate this synergy. This is more evident in a woman, whose perineum is weaker because of the urogenital groove and because it is influenced by the hormonal system (Fritsch et al. 2004).

Cephalically

At the superior part, the diaphragm is in relation to the scapular girdle through the intermediary of the endothoracic fasciae and the pleura (Sato & Hashimoto 1984).

The suspensor ligaments of the pleural dome connect it with the cervicodorsal region.

Centrally, via the pericardium and the pharyngeal and peripharyngeal aponeuroses, the diaphragm is linked to the hyoid bone and via this to the base of the skull.

The esophagus also constitutes an anatomical continuation; in fact, the endothoracic and transverse fascia attach to the inferior hiatus of the oesophagus (Apaydin et al. 2008).

This superior transmission is not linear and is fortunately reduced by tissular elasticity, the different insertions, especially of the endothoracic fascia at the level of the ribs, the pericardial ligaments, and at the superior part the hyoid bone, on which all the fascia, take over and which constitutes a significant point for dampening the ascending forces.

The peripheral route

Because of its numerous attachments, such as the ribs, sternum, and vertebrae, the contraction of the diaphragm transmits cephalically and caudally.

During inspiration the curvature lessens and we notice a mobilization of the whole spine from the cervical region to the sacrum.

This transmission is performed principally by the supraspinous ligament, which runs from the occiput to the sacrum and the anterior common vertebral ligament, which runs along the crura of the diaphragm. This ligament is a stabilizer of the spine and is permanently mobilized during respiration (Akaishi 1995).

Synergy of the contraction of the diaphragm

During inspiration the diaphragm descends via the abdominal mass and mobilizes the perineal diaphragm, which deadens the descending pressure. However, it also interacts with the cervicothoracic diaphragm. The notion of the thoracic diaphragm is not admitted by everyone (Ranney 1996), although it constitutes an anatomical reality, notably in the pleural dome, divided by the cervicothoracic fascia, itself dependent on the fasciae of the neck. All the descending and ascending structures form a relay point at the level of the scapular girdle. This region represents a very important absorption point for the ascending and descending forces.

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During inspiration the endothoracic fascia, the pa-

rietal pleura, and the ligament create a superior fixa-

tion point allowing the vertical diameter of the

thorax to increase and the lungs to fill. This relation-

ship with the cervicothoracic diaphragm also allows

continuous revitalization of a number of local struc-

tures. To sum up, the diaphragm is not just an inspi-

ratory muscle but is involved in the vitalization of the

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Fascia as an organ of communication

Robert Schleip

The previous section of this textbook demonstrated the topographical continuity as well as the specialized local adaptations of the global fascial network. Agreed, it is possible to dissect this continuous network into hundreds of different sheets and bundles. provided one is sufficiently talented in working with a surgical scalpel and given one has a clearly depicted guideline on where to place the cuts. However, when left without a dissection manual and looking at the tissue alone, it becomes apparent that all of those whitish collagenous membranes and envelopes seem to act together as one interconnected fibrous network. Nevertheless, for decades, ligaments, joint capsules, and other dense fascial tissues have been regarded as mostly inert tissues and have primarily been considered for their mechanical properties.

Already during the 1990s advances were being made in recognizing the proprioceptive nature of ligaments, which subsequently influenced the guidelines for knee and other joint injury surgeries (Johansson et al. 1991). Similarly, the plantar fascia has been shown to contribute to the sensorimotor regulation of postural control in standing (Erdemir & Piazza 2004).

This chapter will explore the potential of the fascial network as one of our richest sensory organs. Given the right stature, the overall mass and volume may be bigger than that of the fascial body. However, the surface area of the many million endomysial sacs and other membranous pockets endows this network with a total surface area that by far surpasses that of the skin or any other body tissue. Interestingly, compared with muscular tissue's innervation with muscle spindles, the fascial network is innervated by approximately 6 times as many sensory nerves than its red muscular counter part (Mitchell & Schmidt 1977). Additionally even the spindle receptors in the muscles are themselves found primarily only in areas with force transfer from muscle to connective tissues (van der Wal 2009). This includes many different types of sensory receptors, including the usually myelinated proprioceptive endings (Golgi, Paccini, and Ruffini endings), but also a myriad of tiny unmyelinated 'free' nerve endings, which are found almost everywhere in fascial tissues, but particularly in periosteum, in endomysial and perimysial layers, and in visceral connective tissues. If one includes these smaller fascial nerve endings in the calculation, then the amount of fascial receptors may possibly be equal or even superior to that of the retina, so far considered as the richest sensory human organ (Mitchell & Schmidt 1977). However, for the sensorial relationship with our own body - whether it consists of pure proprioception, nociception or the more visceral interoception - fascia provides definitely our most important perceptual organ (Schleip 2003).

Many years ago, the author was involved in a dispute between instructors of the Feldenkrais Method of somatic education (Buchanan & Ulrich 2001) and teachers of the Rolfing Method of Structural Integration (Jones 2004). Advocates of the second group had claimed that many postural restrictions are due to pure mechanical adhesions and restrictions within the fascial network, whereas the leading figures of the first group suggested that "it's all in the brain", i.e., that most restrictions are due to dysfunctions in sensorimotor regulation. In support of the sensorial hypothesis they cited the vividly published report of Trager (Trager et al. 1987; Juhan 1998), who had observed the disappearance of many muscular restrictions during general anesthesia in many of his patients. Subsequently, a small experiment was set up involving several representatives of those two schools, in which three patients undergoing orthopedic surgery gave their consent for having their range of motion tested (in passive arm elevation, as well as foot dorsiflexion) in the surgical theater immediately before and after commencement of general anesthesia. Given the limited scientific rigor of this preliminary investigation, the result nevertheless was convincing to all involved; most of the previously detected restrictions appeared to be significantly improved (if not absent) during the conditions of anesthesia. It seemed that what had been perceived as mechanical tissue fixation may at least be partially due to neuromuscular regulation.

The ongoing interdisciplinary dispute after this event led to a rethinking of traditional concepts of myofascial therapies, and several years later a first neurologically oriented model was published as a proposed explanatory model for the effects of myofascial manipulation (Cottingham 1985), later copied and expanded by many others in the field (Chaitow & DeLany 2000; Schleip 2003).

While fascial stretch therapies and manual fascial therapies often seem to have positive effects on palpatory tissue stiffness as well as on passive joint mobility, it is still unclear which exact physiological processes may be underlying these responses. Some of the potential mechanisms will be addressed in the clinical section of this book (Chapters 7.1-7.24); they may be due to dynamic changes in water content of the ground substance, to altered link proteins in the matrix, to an altered activity of fascial fibroblasts, as well as other factors. However, today an increasing number of practitioners are basing their concepts to some extent on the mechanosensory nature of the fascial net and its assumed ability to respond to skillful stimulation of its various sensory receptors.

The question then is: what do we really know about the sensory capacity of fascia? And what specific physiological responses can we expect to elicit in response to stimulation of various fascial receptors?

This second section of our textbook will explore some of these intriguing questions. The first chapter in this section will be of particular interest, as it gives a solid overview on what is currently known on the importance of fascial tissues for our sense of proprioception. While, in the past, much emphasis was placed on joint receptors (being located in joint capsules and associated ligaments), more recent investigations indicate that more superficially placed mechanoreceptors, particularly in the transitional area between the fascia profunda and the fascia superficialis, seem to be endowed with an exceptionally rich density of proprioceptive nerve endings (Stecco et al. 2008; Tesarz et al. 2011). While this may be relevant for the practice (and often profound beneficial effects) of skin taping in sports medicine – as well as for other therapeutic fields – further research is necessary to confirm whether the innervation of this superficial fascial layer does indeed play a leading role in proprioceptive regulation.

The sensory nature of fascia includes also its potential for nociception. The chapter on this perspective is written by leading experts in that field. all from Heidelberg University. They summarize their recent years of research about the nociceptive potential of the lumbar fascia. Their choice of the lumbar fascia as field of inquiry is, of course, not accidental. While some cases of low back pain are definitely caused by deformations of spinal discs, several large magnetic resonance imaging studies clearly revealed that for the majority of low back pain cases the origin may have to be searched for elsewhere in the body, as the discal alterations are often purely incidental (Jensen et al. 1994; Sheehan 2010). Based on this background, a new hypothetical explanation model for low back pain was proposed by Panjabi (2006) and subsequently elaborated on by others (Langevin & Sherman 2007; Schleip et al. 2007). According to these authors, microinjuries in lumbar connective tissues may lead to nociceptive signaling and further downstream effects associated with low back pain. The new findings from the Heidelberg group - reported in the next chapters - concerning the nociceptive potential of the lumbar fascia, therefore promise to have potentially huge implications for the diagnosis and treatment of low back pain. As this is a newly emerging field, their research will definitely trigger further research investigations into this important (and very costly) field within modern health care.

Two other chapters will complete this section. One will cover the newly rediscovered field of fascial interoception, which relates to mostly subconscious signaling from free nerve endings in the body's viscera – as well as other tissues – informing the brain about the physiological state of the body. While sensations from proprioceptive receptors are usually projected via the somatomotor cortex, signaling from interoceptive endings is processed via the insula region in the brain, and is often associated with an emotional or motivational component. This field also promises interesting implications for the understanding and treatment of disorders with a somatoemotional component, such as irritable bowel syndrome or essential hypertension.

Finally, this section includes inspiring perspectives on neural communication dynamics within the fascial network. We invite the reader to read these pages with an open-minded attitude, although some of the potential mechanisms presented there appear to be of a hypothetical nature. However, it would certainly not be the first time within the fascinating field of fascia research that a hypothesis previously considered daring might lead to new and substantial insights with clear clinical applications.

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Proprioception

Jaap C van der Wal

Proprioception, mechanoreception and the anatomy of fascia

It is likely that the connective tissue continuum of fasciae and fascial structures serves as a body-wide mechanosensitive signaling system with an integrating function analogous to that of the nervous system (Langevin 2006). Without doubt fasciae and fascial structures play a substantial role in the process of proprioception (Langevin 2006; Stecco et al., 2007b; Benjamin 2009). Fascial components like membranes and septa or deep and superficial fascia are an intricate and integrated part of the locomotor apparatus (Wood Jones 1944; Standring 2005). To play that functional, role in proprioception, the fascial structure should be equipped with adequate neuroanatomical substrate ('proprioceptors'). For the quality of the centripetal information, however, how the mechanical architecture of the connective tissue structure at stake relates to the skeletal and muscular tissue in a given area is an important feature (Benjamin 2009; Van der Wal 2009). Only if a given fascial structure has a mechanical architectural relationship with muscular or skeletal elements, is it able to provide the mechanoreceptive information needed for proprioception. This means that the aptitude of a fascial structure to provide centripetal mechanoreceptive information depends on its architecture and structural relationship with muscular and skeletal tissue and not simply on its topography (Van der Wal 2009).

In this chapter proprioception is defined in the neurophysiologic way – as the ability to sense the position and location, orientation, and movement of the body and its parts. In a more strict sense, proprioception could be defined as the process of conscious and subconscious sensing of joint position and/or motion (Skoglund 1973; Fix 2002). Here, the more explicit meaning of the psychological definition of 'proprioception' as sometimes applied, as the notion 'body image sense' or 'body awareness' is not meant. Proprioception, in this context, has to be discriminated from **exteroception**, relating us with the outer world, as well as from **interoception**, informing about visceral and metabolic processes (see elsewhere in this book).

The morphological substrate of proprioception – encapsulated or unencapsulated mechanosensitive sensory nerve endings (mechanoreceptors) and related afferent neurons (see Fig. 2.2.1) – is considered to provide the centripetal information needed for the control of locomotion or for the maintenance of posture (Barker 1974). On the level of the brain this information is integrated with information originating from other sources (e.g., more specific proprioceptive sense organs like the labyrinth or skin receptors) to the overall conscious and subconscious awareness of position and motion (kinesthesis and statesthesis).

In this context, mechanoreception is not synonymous with proprioception. Proprioception relates to mechanoreception as seeing relates to the retina. The mechanoreceptive information needed for the process of proprioception originates not only from fasciae and other connective tissue structures but also from mechanoreceptive or even tactile information from skin, muscles, joint surfaces, and joint structures. Mechanoreceptors are triggered by



Fig. 2.2.1 • Types of mechanoreceptors. (A) Free nerve ending (FNE); (B) Ruffini corpuscle (RC) or spraylike endings; (C) lamellated corpuscle (LC) or Paciniform ending; (D) Golgi tendon organ (GTO); (E) muscle spindle (MS).

mechanical deformation like squeezing, stretching or compression. In order to understand their contribution to the proprioceptive information it is not only important to know their topography (where and in which elements of the locomotor apparatus they are located) but also how they are spatially and mechanically related with the various (tissue) components of the system. Proprioception in the fascia is not only provided by the mechanoreceptors that are located within or are immediately attached to the fascial structures, but also the architecture of the fascia plays an instrumental role in the process of proprioception. It can do so by mediating forces that cause deformation of receptors (which in fact represents the main stimulus for mechanoreceptors) that are not directly attached to the fascia itself. The term "ectoskeleton" has been proposed by some authors (Benjamin 2009) to capture the idea that fascia could serve as a significant site of muscle attachment, constituting a kind of "soft tissue skeleton". Mechanoreceptors situated within muscles as anatomical units may orient as to their distribution and spatial organization to the fascial layers to which the muscle fascicles insert and between which muscular tissue is interposed in the process of force transmission. In such cases, the fascial architecture plays an instrumental role in the process of proprioception without the necessity for the connective tissue structures themselves being directly equipped with mechanoreceptive substrate (Van der Wal 2009).

So, to evaluate the significance of fascial structures as to the proprioceptive input from a certain body region it is not only important to know the anatomy of the given fascia ('where') but also its architecture, i.e., functional relationship ('how'). Many fascial structures play a direct or indirect role in force transmission. Most anatomy textbooks, however, describe the locomotor apparatus as a system built up from discrete elements involved in positioning, motion, and force transmission: i.e., muscles (with tendons and aponeuroses) and ligaments. In this outdated concept, muscles represent the main elements in the system, which in atlases are often presented as discrete anatomic structures with the surrounding and 'enveloping' connective tissue layers removed. When connective tissue is met as a layer, a membrane, a fascia covering a body structure or organ or region, is usually given a name derived from the anatomic substrate that the layer covers. Fasciae therefore are most often defined as a suborganization of the 'primary' anatomy of organs (e.g., muscles) This is all related to the 'dissectional mind' that still prevails in the anatomy atlases and textbooks and considers the locomotor apparatus as built up from anatomical elements. When Schleip (2003a, b) mentions the fascia as "the dense irregular connective tissue that surrounds and connects every muscle, even the tiniest myofibril, and every single organ of the body forming continuity throughout the body" or as the "organ of form" (Varela & Frenk 1987), he actually presents fascia as an important integrative element in human posture and organization of movement (locomotor apparatus). Therefore an analytical and 'dissectional' approach of the 'anatomy' of the fascia cannot do justice to the role of fascial tissue and structures in proprioception.

Connectivity and continuity

The primary connective tissue of the body is the embryonic mesoderm. The mesoderm represents the matrix and environment within which the organs and structures of the body have been differentiated and in fact are 'embedded'. Blechschmidt (2004) distinguished the mesoderm, as germinal layer, as an 'inner tissue' in opposition to the ectoderm and endoderm as 'limiting tissues and proposed not to call it a 'derm' but to call it 'inner tissue'. The primary "inner tissue" is the undifferentiated connective tissue **mesenchyme**, which in principle is organized in three components: cells, intercellular space (interstitial substances), and fibers. In the functional development and differentiation of the primary connective tissue, there are two patterns of 'connection'. The first pattern is the development of 'intercellular space', which represents a fissure functioning as a sliding and slipping space. This is seen in the formation of coelom (body cavities), joint cavities, and also in bursa-like gliding spaces between adjacent tendons or muscle bellies. In this pattern spatial separation is ensured, and in this way motion is enabled. The second pattern is the formation of a binding medium. That can be fibers (e.g., as in regular dense connective tissue structures like the desmal sutures in the skull, interosseous membranes, and ligaments) or interstitial substrate and matrix (for example, in cartilaginous joints). In osteopathic circles, the continuum and continuity of the 'connective tissue apparatus' in the human is emphasized. Such a view is in harmony with the view that the principal function of mesoderm as "inner tissue" is 'mediating' in the sense of 'connecting' (binding) and 'disconnecting' (shaping space and enabling movement).

This view of two patterns of connectivity is also applicable to the anatomy of fasciae. In general, fasciae in the musculoskeletal system exhibit two different mechanical and functional aspects:

- Fasciae of muscles adjacent to spaces that are filled with loose, areolar, connective tissue ('sliding tissue') and sometimes with adipose tissue. They enable the sliding and gliding of muscles (and tendons) against each other or against other structures. In such splits globular or oval mechanoreceptors triggered by compression (see below) could inform the brain about the displacement and movement of fascial tissue and related structures.
- Intermuscular and epimysial fasciae that serve as areas of insertion for muscle fibers that in this way can mechanically reach a skeletal element without necessarily being attached directly to the bone. They appear as intermuscular septa but also as so-called superficial fasciae (like fascia cruris and fascia antebrachii,) providing a broad insertion area for muscle fibers. If provided with more stretch susceptible receptors, such fascial layers could inform about stresses of the fascial tissue in relation to the transmission of forces.

This indicates that fasciae exhibit a variety of mechanical relationships with neighboring tissue and therefore may play quite different functional roles as to proprioception as well. The fasciae of the organs and of muscles often represent the 'gliding fascia' type; in this context, coelomic cavities actually function as 'joint spaces' enabling motion of the organs. Many epimysial muscle fasciae function in a similar way, providing mobility between a muscle and its neighborhood. However, fasciae like the fascia cruris (tibial fascia) or the retinaculum patellae function as epimuscular aponeuroses.

Architecture is different and more than anatomy

To understand the mechanical and functional circumstances of the fascial role in connecting and in conveying stresses and in proprioception, it is therefore more important to know the architecture of the connective and muscle tissue than the regular anatomical order or topography. This applies to every fascial layer in the human body. One must know both where they are situated (anatomy) and how they are connecting and connected (architecture). Depending on the architectural relationship of the fascial connective tissue with the muscular tissue, not only juxta-articular connective tissue (like interosseous ligaments) may provide proprioceptive information about joint movement or joint position, but epimysial, intermuscular, aponeurotic, fascial layers can also play a functional role in such processes (directly or indirectly).

In this context, two views on the organization of connective and muscular tissue may be described. On the one hand, there is the well-known view that muscular and connective tissue structures have to be considered as discrete anatomical elements. In this concept muscles function as the dynamic force-transmitting structures and they are organized in parallel to ligamentous structures as the more passive force-transmitting elements. The non-ligamentous fascial connective tissue is considered as auxiliary to the muscle units as tendons and aponeuroses. Areolar fascial connective tissue shapes, space in between the anatomical elements, providing the opportunity for sliding and mobility. Such architecture is exhibited clearly in the distal regions of the limbs, where separate muscle entities (bellies with tendons) function in parallel to underlying joint capsules with or without capsular reinforcing ligaments. Here, mechanoreceptive substrate in the fascia serves the (unconscious) perception of this sliding and movement.

On the other hand, a pattern can be described where connective tissue and muscular tissue are organized mainly in series with each other in a more 'transmuscular' organization. Huijing et al. (2003) point out that, often, muscles which from an anatomical perspective are considered as morphologically discrete elements cannot be considered as isolated units controlling forces and movements. Detailed studies of the lateral cubital region of man and rat showed such architecture quite clearly (Van der Wal 2009). Nearly all the deep and superficial regular dense connective tissue (RDCT) layers are organized in series with muscle fascicles (presented as muscle compartment walls). Collagenous fibers that run from bone to bone – thought to be stressed passively by displacement of the articulating bones - hardly occur. Instead, there occur broad aponeurotic layers of RDCT to which relatively short muscle fascicles insert, which, on the opposite side, are directly attached to skeletal elements. Such configurations of muscle fascicles attached to the periosteum of one articulating bone, and via a layer of RDCT indirectly attached to another articulating bone, could be considered 'dynamic ligaments'. Such 'dynaments' are not necessarily situated directly beside the joint cavity or in the deep part of the joint region (Van der Wal 2009).

The substrate of mechanoreception

Connective tissue and fasciae are richly innervated. (Stilwell 1957; Schleip 2003b; Stecco et al. 2007a; Benjamin 2009). Considerations such as 'architecture versus anatomy' mutatis mutandis may also apply for the spatial organization of mechanoreceptors, the morphologic substrate for proprioception. To study the role and function of mechanoreceptors in the process of proprioception, it may be important to know where they actually are located in such regions and how they are or are not connected with the relating tissue elements. In general, however, mechanoreceptors are often reported to occur either as muscle receptors or as joint receptors. Muscle receptors are mechanoreceptors present in the muscles, including their auxiliary structures such as tendons, aponeuroses, and fasciae. Muscle spindles and Golgi tendon organs (GTOs) are the best-known types of such receptors (Barker 1974). Joint receptors are considered to be situated in joint capsules and related structures, including reinforcing ligaments. These receptor types are usually ordered according to the (ultra)structure of the receptor itself, physiologic features, type of afferent nerve fiber, and other parameters (Freeman & Wyke 1967a, b).

Mechanoreceptors are in fact free nerve endings (FNEs), whether or not equipped with specialized end organs. The main stimulus for such receptors is deformation. Variation exists as to the microarchitecture of the ending. On the one hand, there exists the principle of lamellae around a relatively simple nerve ending. This represents the principle of the ball- or bean-shaped Vater Pacini or paciniform corpuscles, often called lamellated corpuscles (LC). On the other hand, there is the more spray-like organization of the nerve ending wrapping around and in between the deformable substrate such as connective tissue fibers. Those are the spindle-shaped Ruffini corpuscles (RC) or GTOs. These two types of microarchitecture roughly relate to the type of mechanical deformation that is at stake: compression for the lamellated bodies and traction and torsion for the spray-like type. Other varying parameters are threshold, adaptivity, and adjustability. In this general classification, the muscle spindle is a spindle-shaped spray-like ending organized around specialized muscle fibers equipped with the extra possibility of adjustable length (Strasmann et al. 1990).

As stated above, mechanoreceptors are in general reported to occur as either 'muscle receptors' or 'joint receptors'. In this concept, muscle receptors are mechanoreceptors present in the muscles and joint receptors are considered to be mechanoreceptors situated in joint capsules and related structures like ligaments. In this context, the concept often prevails that joint receptors play the leading role in the process of monitoring joint position or movement for the purpose of statesthesis and kinesthesis, while muscle receptors are relegated to motor functions that operate at a subconscious or reflex level (Barker 1974).

Mechanoreceptors associated with muscles, including the muscle auxiliary structures such as tendons, are usually classified as follows (see Fig. 2.2.1):

- FNEs (unencapsulated).
- Muscle spindles (sensory endings with encapsulated intrafusal muscle fibers).
- GTOs (type III endings, relatively large spraylike endings, i.e., 100–600 μm diameter with high threshold and very slow-adapting).

The mechanoreceptors typically associated with joints are (see Fig. 2.2.1):

- FNEs (unencapsulated).
- LC (type II ending with a two- to fivelayered capsule, less than 100 μm length, with low threshold and rapidly adapting).

Here, this term is preferred to the notion 'paciniform corpuscle'.

 RC (type I ending, relatively small spray-like ending, up to 100 μm with low threshold and slow-adapting).

The functional role of architecture of the connective and muscular tissue in mechanoreception

In an extensive study of the spatial organization of the morphological substrate of proprioception in the proximal lateral cubital region of the rat (Van der Wal 2009), an inventory has been made of mechanoreceptors that may occur in direct or indirect relationships with the connective tissue layers in the joint region. A spectrum of mechanosensitive substrate occurs at the transitional areas between the regular dense connective tissue layers (organized as epimysial or intermuscular layers and septa) and the muscle fascicles organized in series with them. This substrate exhibits features of the mechanosensitive nerve terminals that usually are considered to be characteristic for joint receptors and for muscle receptors. At the so-called superficial antebrachial fascia, as well as at the intermuscular fascial layers RC as well as LC were present between the fascial layer and inserting muscle fibers. Sometimes, even, one pole of a muscle spindle was attached to those layers.

Based upon the architecture of the connective tissue and upon the spatial distribution of the substrate of mechanoreception, it is assumed that the joint receptors here are also influenced by the activity of the muscle organized in series with the collagenous connective tissue near those receptors. This supports the idea that the stresses during joint positioning are conveyed mainly via those collagenous layers and also are involved in triggering the related mechanoreceptors. In the region studied there exists no basis in morphology for socalled joint receptors that are deformed exclusively by passive strain in collagenous connective tissue structures induced by displacement of the articulating bones. The substrate of proprioception that was found in and near the RDCT apparatus in the lateral cubital region has features of mechanoreceptors that are usually linked with 'joint receptor' substrate, as well as of mechanoreceptors usually present in muscles and related tendons. It is obvious that, in cases like this, the fascial layers together with the *in series* inserting muscular tissue function as a kind of 'dynamic ligaments' or 'dynaments'.

Very often myofascial areas are richly innervated and covered by nerve plexuses. In the previouslymentioned study on rat and humans (Van der Wal 2009) it was shown that over the proximal (epimysial) antebrachial fascia, as well as over the fascia 'covering' the supinator muscle (in fact a supinator aponeurosis), extensive plexuses were present. Plexiform arrangements of peripheral nerves sprouting over tendons and ligaments are a consistent feature in the innervation pattern in the periarticular aponeuroses of the knee and elbow joint (Wilson & Lee 1986). Stilwell (1957) states that such networks terminate in small 'paciniform corpuscles' and in 'freely ending axons' on the surface connective tissue of tendons, aponeuroses, and muscles, and in periosteal connective tissue, nearly always in the vicinity of other mechanoreceptors. The type of axons present in the nerve fascicles of the plexuses studied here (Van der Wal 2009), as well as the demonstrated (or putative) origin of those axons from the substrate of mechanoreception in the studied material, support the notion that such perior juxta-articular nerve plexuses are not exclusively involved in nociceptive processes, as stated by Freeman and Wyke (1967a, b). This also means that the substrate of proprioception does not necessarily have to be situated within the fascial fibers to play a functional role in proprioception. Regarded mechanically, the intermediate zones between fascial dense connective tissue and adjacent muscle fibers and/or adjacent loose areolar connective tissue might be of interest as a source of mechanoreceptive information. In areas where the fascial connective tissue is so dense that it allows little dislocation or deformation, as is the case in most ligamentous structures, it seems logical that the innervation is more involved in nociception or sympathetic vascular regulation. In the latter respect, it is worth mentioning that there exist ligaments that are mechanically important yet poorly innervated and ligaments with a key role in sensory perception that are richly innervated (Hagert et al. 2007; Benjamin 2009). It all relates to the degree to which deformation is allowed (since it is deformation that forms the major stimulus for mechanoreceptive triggering), as well to the microscopic level (the kind of mechanoreceptor) and to the macroscopic level the (architecture of the fascia and related tissue).

Dynaments: more than ligaments or muscles

The findings in the studies described earlier regarding spatial distribution and the organization of socalled 'muscle receptors' were even more relevant to the concept that is brought forward here. Those receptors appeared not to be organized according to principles of anatomy and topography, but to cope with the functional architecture of the connective tissue complex of the epi-, inter- and submuscular RDCT layers in relation to muscular architecture. In all the antebrachial extensor muscles studied, the distribution of muscle spindles per muscle area is uneven. If the spatial distribution of muscle spindles is considered per muscle, it is difficult to detect a common distribution pattern in all muscles (see Plate 2.2.1). The spatial distribution of those receptors, however, becomes understandable from the regional functional architecture of the connective tissue and fascia, i.e., the RDCT structures. The muscular zones that are dense in muscle spindles and GTOs are the stress- and forceconveying zones of the muscle, which are in series with the connective tissue complex proximally and in series with the peripheral tendons distally. This arrangement provides a common principle that may explain many kinds of distribution patterns. Of course, sometimes architectural units coincide with specific topographic entities, as in this study the supinator muscle with its aponeurosis nearly represents the anatomy of a 'dynament'.

As Huijing et al. (2003) pointed out, based upon mechanical arguments that the muscles are not isolated units controlling forces and movements as often thought, apparently also on the level of spinal sensorimotor control the muscles should no longer be considered the functional entity in the locomotor system (English & Letbetter 1982; English & Weeks 1984; Van der Wal 2009). Such considerations again match the task-dependent models of brain control well: motor units are not necessarily organized with respect to individual motor nuclei, but are organized according to behavioral tasks. The concept of the locomotor apparatus being built up by architectural units of muscular tissue in series with collagenous connective tissue is more consistent with such trans- or supramuscular models than is the concept in which muscles function as the entities that maintain joint integrity parallel to ligamentous structures.

Classification of mechanoreceptors in proprioception

In consequence of the identification of an in-series organization of muscular tissue and regular dense connective tissue structures in the locomotor apparatus (mainly tendons distally and muscle compartment walls proximally), three configuration types of mechanoreceptors were identified in the mentioned study (Van der Wal 2009). *Mutatis mutandis* this spectrum could also be considered to represent the substrate of proprioception of the fascia:

- Muscle spindles, GTO (RC), FNE, and LC are found in areas between muscular tissue and RDCT layers. This configuration coincides with the conventional muscle-tendon spectrum of sensory nerve endings (Barker 1974; Von Düring et al. 1984).
- LC and FNE are found in areas in which RDCT adjoins reticular connective tissue, gliding spaces. This configuration coincides mainly with the spectrum of sensory nerve endings usually indicated as articular receptors (Freeman & Wyke 1967a, b; Halata 1985).
- Only FNE are present in the transition to the skeletal attachment (periosteum). This configuration coincides with the endotenonial spectrum of sensory nerve endings with mainly (mechanoreceptive) FNE from group III and IV fibers (Von Düring et al. 1984).

Most plexuses in or near the regular dense connective tissue of fascial layers contain nerve fibers of type III and IV. Nerve fibers of group III (or A delta type) are afferent from mechanoreceptors; nerve fibers of group IV (or C-type) are afferents from FNE that are either nociceptive or mechanosensitive (strain).

In the above-mentioned configurations, RC are not indicated as a separate category but GTO and RC are considered to be the same receptor type, presenting gradual differences depending on the texture of the surrounding tissue. The quartet MS - GTO/ RC-LC-FNE represents the complete spectrum of mechanoreceptors in the locomotor apparatus. In this way, the three main types of so-called muscle receptors (MS, GTO, and LC) are combined with the three types of so-called capsular (or joint) receptors (RC, LC, and FNE). Depending on the local situation, this quartet therefore represents the spectrum of mechanoreceptors involved in the proprioceptive function of fasciae and fascial structures. The activity and role of a mechanoreceptor is defined not only by its functional properties, but also by its architectural environment. It is the architecture of the fascial connective tissue in relation to the muscular tissue components and skeletal elements that plays a major role in the coding of the proprioceptive information that is provided.

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Interoception

A new correlate for intricate connections between fascial receptors, emotion, and self recognition

Robert Schleip Heike Jäger

Introduction

While the sense of proprioception is fairly well known to therapists working with fascia, interoception and its inclusion in fascial therapies may be a "new concept" for many. The concept is not so new: in the nineteenth century it was called coenesthesia: the neurological model of a mostly unconscious sense of the normal functioning of the body and its organs. Early German physiologists called it Gemeingefuehl or "common sensations" and differentiated them from the five senses of Sherrington's early writings. Recently, however, the same concept has been intensely revived under the term interoception, and novel insights regarding the anatomical, physiological, and neurological details of this sensory system have led to an almost explosive increase of scientific attention and exploration.

Disorders such as anxiety, depression, or irritable bowel syndrome have subsequently been described as interoceptive disorders. Most notably, it has been proposed that the neural pathways associated with interoception may be considered as a potential correlate for consciousness (Craig 2009). The sensory receptors for interoception are free nerve endings, most of which are located in fascial tissues throughout the human body. It is helpful to understand that proprioception and interoception are organized differently in the human brain and that very different afferent pathways are involved in them.

What is interoception?

Previous concepts of interoception often focused on visceral sensations only. Current concepts describe interoception as a sense of the physiological condition of the body, which includes a much wider range of physiological sensations, including, for example, muscular effort, tickling, or vasomotor sensations (see Box 2.3.1). These sensations are triggered by stimulation of unmyelinated sensory nerve endings (free nerve endings) that project to the insular cortex rather than to the primary somatosensory cortex which is usually considered as the main target of proprioceptive sensations (Berlucchi & Aglioti 2010).

Feelings from these sensations not only have a sensory, but also an affective, motivational aspect and are always related to the homeostatic needs of the body. They are associated with behavioral motivations that are essential for the maintenance of physiological body integrity.

Sensual touch

A recent and surprising addition to the above list of interoceptive sensations is the sense of sensual or pleasant touch. This discovery was triggered by examinations of a unique patient lacking myelinated afferents in whom slow stroking of the skin with a soft brush triggered a faint and obscure sensation of pleasant touch (and general well-being), although

Box 2.3.1

Interoceptive sensations

- Warmth, coolness
- Muscular activity
- Pain, tickle, itch
- Hunger, thirst
- Air hunger
- Sexual arousal
- Wine tasting (in sommeliers)
- Heartbeat
- Vasomotor activity
- Distension of bladder
- · Distension of stomach, rectum or esophagus
- · Sensual touch.

Afferent pathways associated with these sensations follow the lamina I spinothalamocortical tract towards the insular cortex.

the patient was unable to recognize any stroking direction. Functional magnetic imaging showed that this vague sensation was accompanied by a clear activation of the insular cortex, while no activation was seen in the primary somatosensory cortex. (Olausson et al. 2010).

Based on the innervation of primate skin and on subsequent studies with other patients it was concluded that the affected sensory receptors are unmyelinated C-fiber afferents with a low mechanical threshold, and that these endings are themselves connected with neural interoceptive pathways. Those afferents have a slow conduction velocity (0.5-1.0 s delay from stimulus to arrival in the brain). Since these receptor types have never been found in the palm of the hand despite numerous microneurographic recordings, it is assumed that they are present in hairy skin only and are absent in glabrous skin. It is concluded that human skin contains particular touch receptors which form a system for social touch that may underlie emotional. hormonal (for example oxytocin), and affiliative responses to caress-like, skin-to-skin contact between individuals (Fig. 2.3.1). The profound importance of such a system for human health and well-being has long been indicated (Montague 1971), at least since the classical study of Harlow (1958) with baby rhesus monkeys that express affection for a surrogate mother in response to tactile comfort.



Fig. 2.3.1 • The discovery of interoceptive receptors in human skin. Besides proprioceptive nerve endings, human skin contains interoceptive C-fiber endings which trigger a general sense of well-being. The connections of these slowly conducting receptors do not follow the usual pathway of the pyramidal tract towards the proprioceptive areas in the brain. They rather project to the insular cortex, a key player in the regulation of interoception, This was recently discovered through experiments with patients lacking myelinated afferents. Whenever their skin was gently stroked, they responded with an increased sense of general well-being, although they were unable to detect the direction of stroking. Subsequent brain imaging studies revealed that the touch activated their insular cortex, while no activation was seen in proprioceptive brain areas. It is concluded that human skin contains special touch receptors, with a slow conduction velocity, which are part of a neurobiological system for social touch. ©iStockphoto.com/Neustockimages.

A new phylogenetic development

The afferent neurons related to interoception terminate in lamina I, the most superficial layer of the dorsal horn of the spinal cord. This lamina projects strongly to the sympathetic cell columns of the thoracolumbar spinal cord, where the sympathetic preganglionic cells of the autonomic nervous system originate. From there they project to the main homeostatic integration sites in the brainstem. The latter include brainstem regions – such as the parabrachial nucleus – which are densely interconnected with the amygdala and hypothalamus. In addition, they project to the insular cortex.

Interestingly, this particular "lamina I spinothalamocortical pathway" is a comparatively recent phylogenetic acquisition of primates. It evolved from the afferent portion of the evolutionary ancient system that maintains the homeostatic integrity of the body. In mammals, the activity of lamina I neurons is integrated in the parabrachial nucleus; it is only from there that they are further projected to the insular cortex via the ventromedial thalamic nucleus (Craig 2009). In primates, however, there are direct projections from lamina I to thalamic regions from which they are further conveyed to the insular cortex (Fig. 2.3.2). In other words, primates possess a more direct route between the afferent region for interoceptive sensations in the spinal cord (lamina I) and their insular cortex. No comparable difference in terms of neuronal architecture between primates and other mammals has been observed regarding their processing of proprioception.

The insular cortex itself is organized in a hierarchical manner: primary sensory inputs related to interoceptive sensations project to the posterior insula. They are then progressively elaborated and integrated across modalities in the middle and anterior insula (Devue et al. 2007). Finally, the highest



Fig. 2.3.2 • A novel short-cut route for interoception in primates. In mammals, the main pathway of interoception starts with free nerve endings, which project to the lamina I of the spinal cord. From here they project to the prebrachial nucleus in the brainstem, and it is only from there that they are further projected to the insular cortex via the thalamus. In primates, however, there are additionally direct projections from lamina I to the insula via the thalamus. Primates therefore possess – as a novel phylogenetic acquisition – a more direct route between the afferent region for interoceptive sensations in the spinal cord and the insular cortex (black arrow).

integrative level is expressed in the anterior insula which has intimate connections with the anterior cingulate cortex. Together they form an emotional network in which the limbic insular component is involved in sensory reception and conscious feelings, and the cingulate cortex serves as the motivational and motor component for the behavioral expression of the feelings.

When observing the emotional behavior of nonprimate animals, our tendency towards anthropomorphic inferences suggests that they experience bodily feelings in the same way as we do. However, their different interoceptive pathways indicate they don't, because the phylogenetically new pathway that conveys interoceptive sensations to the thalamocortical levels in primates is either rudimentary or absent in nonprimate animals (Craig 2003).

The anterior insula-cingulate network is also credited with the specific function of self recognition (Devue et al. 2007). Craig (2009) provided impressive evidence that the anterior insular cortex is a peculiarly human brain structure that is crucial for integrating all subjective feelings related to the body, and especially to its homeostatic conditions, into emotional experiences and conscious awareness of the environment and the self. He suggests that the human insular cortex and its peculiar spinothalamic afferent pathways set our species apart from other mammals by supporting consciousness of the body and the self. This view is congruent with the somatic marker hypothesis of Damasio (1994), which proposes that humans use nonconscious somatic sensations, such as "gut feelings", to guide their decision making, particularly when facing complex and conflicting choices. Similar to Craig's concept of the uniqueness of human interoception, this model sees the human insular cortex - together with its newly acquired direct spinothalamic afferent pathway – as key players for the integration of body perceptions and mental processes.

Interoception and somatoemotional disorders

The described (re)discovery of the importance of interoception in human self regulation – and of the unique neural architecture that regulates the processing of these internal body sensations in humans – triggered a multitude of studies examining the correlations between interoception and particular aspects of human health. Apparently, many complex disorders with a somatoemotional component are associated with clear differences in interoception. While this is currently a new and exciting field of research in psychobiological medicine, many of the studies so far published reveal an association of such pathologies with interoceptive processing. However, the precise system dynamics of these associations (including the differentiation between primary causative and secondary effects) still need to be elucidated for most of the interoceptive disorders. The following disorders are examples of such complex interactions.

Anxiety, as well as depression, has been shown to go along with significant alterations in interoceptive processing. They are connected with increased but noisy interoceptive input, the processing of which is amplified by self-referential belief states via an enhanced top-down modulation in response to the poorly predictable interoceptive states (Paulus & Stein 2010). Both of these somatoemotional disorders seem not to be disorders of the afferent interoceptive signaling, but can be understood as altered interoceptive states as a consequence of noisy amplified self-referential belief states concerning the interoceptive sensations.

Similarly, brain imaging studies of patients with irritable bowel syndrome revealed a disrupted modulation of insular cortex responses to visceral stimuli (such as in response to experimentally induced painful rectal distension as well as to the subsequent relaxation). It is suspected, that these dysfunctional regulations may provide the neural basis for altered visceral interoception by stress and negative emotions in these patients (Elsenbruch et al. 2010).

Drug addictions, as well as other addictions, have also been proposed to be interoceptive disorders. Apparently, the primary goal of these disorders is that the addicted individual aims to obtain the effects of the drug use ritual upon their internal body perception. The representations of the achievement of this goal in interoceptive terms by the insula contribute to how addicted individuals feel, remember, and decide about performing the related rituals. Similar interoceptionrelated insular dynamics have been suggested for other addictions and cravings, such as excessive sex, gambling, smoking, or eating (Naqvi & Bechara 2010).

In essential hypertension an increased interoceptive awareness has been observed, even in the early stages of this disorder, and its contribution to the prospective development of this common cardiovascular syndrome has been discussed (Koroboki et al. 2010). Finally, aging and post-traumatic stress disorders have been shown to be associated with a significant decline in interoceptive awareness. Mindfulnessbased therapies, focusing on subtle somatic sensations are therefore suggested as helpful therapeutic approaches (van der Kolk 2006).

Fascia as an interoceptive organ

In musculoskeletal tissues only a minority of the sensory nerve endings are myelinated mechanoreceptors concerned with proprioception, such as muscle spindles, Golgi receptors, Paccini corpuscles, or Ruffini endings. The vast majority - or 80% of afferent nerves - terminate in free nerve endings (Schleip 2003). Termed "interstitial muscle receptors", they are located in fascial tissues such as the endomysium or perimysium and are connected with either unmyelinated afferent neurons (then called type IV or C-fibers) or myelinated axons (type III or A δ fibers). Indeed, 90% of these free nerve endings belong to the first group, to the slowly conducting C-fiber neurons (Mitchell & Schmidt 1977). Functional magnetic imaging studies by Olausson et al. (2008) revealed that stimulation of these C-fiber neurons results in activation of the insular cortex (which indicates a clear interoceptive role of these receptors) and not of the primary somatosensory cortex which is usually activated by proprioceptive input.

A surprising conclusion from this is that the number of interoceptive receptors in muscular tissues by far outnumbers the amount of proprioceptive endings. In numerical terms, one could estimate that for every proprioceptive nerve ending in these tissues there are more than seven endings that could be classified as interoceptive receptors.

While some of these free nerve endings are thermoreceptors, chemoreceptors, or have multimodal functions, the majority of them do in fact function as mechanoreceptors, which means they are responsive to mechanical tension, pressure, or shear deformation. While some of these receptors are high threshold receptors, it has been shown that a significant portion (approximately 40%) can be classified as low threshold receptors, which are responsive to light touch, even to touch as light as "with a painter's brush" (Mitchell & Schmidt 1977). Most likely they are therefore also responsive to the tissue manipulation of myofascial therapists.

Interoception

Manual therapy and interoception

When treating muscular tissues myofascial therapists are usually concerned with direct biomechanical effects on non-neural tissues or with the stimulation of specific proprioceptive nerve endings, such as muscle spindles, Golgi receptors, etc. However, the above considerations suggest that it is advisable that manual therapists target the interoceptive receptors and their related upstream effects to a much larger degree than is usually taught or practiced.

Some of the interoceptive nerve endings in muscle tissues have been classified as ergoreceptors; they inform the insula about the work load of local muscle portions. Their mechanical stimulation has been shown to lead to changes in sympathetic output, which increases the local blood flow. Stimulation of other interoceptive nerve endings has been shown to result in an increased matrix hydration, via an augmentation of plasma extravasation, i.e., the extrusion of plasma from tiny blood vessels into the interstitial matrix (Schleip 2003).

It could therefore be extremely useful to pay attention to the autonomic responses at each moment – and to the limbic-emotional (or insular) response of the client, while monitoring the touch direction (plus its speed and magnitude) in such a manner that a profound change in local tissue hydration as well as other autonomic effects can be achieved. It would also be advisable to invite a perceptual refinement - and some verbal feedback of the client regarding their interoceptive perceptions. While proprioceptive sensations may be in the foreground during the actual stroke application, those finer interoceptive sensations are usually easier to perceive in periods of at least several seconds of rest between different manipulative strokes. Subjective sensations of warmth, lightness/heaviness, spaciousness, density/fluidity, nausea, streaming, pulsation, spontaneous affection, or a general sense of well-being may be such interoceptive sensations that can be triggered by myofascial tissue manipulation. From the therapist's perspective, subtle changes in the client - such as an increased local tissue hydration, changes in temperature, in skin color, in breathing, micromovements of the limbs, pupil dilation, and facial expression - can serve as valuable signals for physiological effects related to interoceptive processes.

Therapists who apply mechanical stimulation to visceral tissues, such as visceral osteopaths, should

also profit from a larger recognition of interoception and related physiological as well as psychoemotional effects. Recent discoveries concerning the richness of the enteric nervous system have taught us that our "belly brain" contains more than 100 million neurons (Gershon 1999). Most of these are located either in the connective tissue zone between the inner and outer layers of the muscularis externa (Auerbach's plexus) or in the dense connective tissue layer of the submucosa (Meissner's plexus). Many of these visceral nerve endings are directly concerned with interoception and are connected via the "lamina 1-spinothalamocortical pathway" with the cortical insula, as described above. Considering that several complex disorders such as irritable bowel syndrome are associated with a disrupted modulation of insular responses to visceral stimuli, it is conceivable that a slow and careful application of manual forces to visceral tissues - if accompanied by a sense of safety and mindfulness of the client - could be a useful, if not ideal, approach for enhancing a healthy interoceptive self regulation.

Myofascial as well as visceral therapists should also not be surprised when encountering psychoemotional responses which may include changes in internal body perception, in self awareness, or affiliative emotions. These may be triggered by their stimulation of interoceptive free nerve endings in the skin, in visceral connective tissues as well as in muscular tissues.

Movement therapies and interoception

In competitive sports, the attention is often focused on external goal achievements. Frequently, it is also focused on the task of overriding internal sensations of discomfort, tiredness, etc. In contrast, complementary medicine associated practices - like Yoga, Tai Chi, Chi Gong, Feldenkrais, Pilates, Body Mind Centering, or Continuum Movement - usually encourage a perceptual emphasis on finer sensations in one's own body. However, depending on the focus of the individual teacher or respective school, the internal perception is sometimes directed almost entirely towards proprioceptive refinement. For example, a student of such training approaches may learn to feel minute movements of individual vertebrae or to control their lumbar lordosis within a multitude of loading situations. Nevertheless, they may remain an

"interoceptive moron", who may, for example, be unable to differentiate whether their visceral sensations at a given moment are signs of an empty stomach, of stage fright-induced "butterflies", of empathy-driven "gut feelings" about another person's dilemma, or may simply be an acute gastritis.

In contrast, some teachers of these practices also include a skilled fine-tuning of the student's perception for interoceptive sensations. This may include emphasizing sensations such as a subtle tingling under the skin, sensation of a general or localized warming, a subjective sense of internal spaciousness, a feeling of aliveness, an inner silence, an emotional "homecoming", or a meditation-like change in general self awareness. For example, gravity-oriented changes in body positions – such as some upside-down postures in yoga practices – could easily trigger new and interesting (and hopefully unthreatening) sensations in visceral ligaments, which can foster interoceptive refinement. Given the recent research indications for a close correlation of many psychoemotional disorders such as irritable bowel syndrome, anxiety, or posttraumatic stress disorder - with a disrupted interoception, it is conceivable that some of these movement practices may have a strong therapeutic potential for these disorders. Typically, these therapeutic practices foster an attitude of inner mindfulness, of refining "internal listening skills", and they frequently alternate brief periods of active motor attention with subsequent periods of rest where the students pay attention to small interoceptive sensations within their body. Not surprisingly, some studies already indicate a positive health-enhancing effect of such "mindfulness based therapies" for a large number of common clinical conditions (Astin et al. 2003).

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Nociception: The thoracolumbar fascia as a sensory organ

2.4

Ulrich Hoheisel Toru Taguchi Siegfried Mense

Introduction

In the literature, the thoracolumbar fascia (TLF) is usually assumed to have a mechanical function connecting the latissimus dorsi muscle as well as the abdominal muscles to the spine and iliac crest. It continues craniad up to the skull and caudad to the fascia of the lower extremity. Actually, it connects the latissimus dorsi muscle to the gluteal muscles, thus functionally linking the arm with the leg. Other functions are: (1) forming a sheath around muscles thatreduces friction during movements, (2) facilitating the return of venous blood to the heart, (3) providing an ectoskeleton for the attachment of muscles, and (4) protecting blood vessels and muscles from mechanical damage (e.g., the lacertus fibrosus of the biceps brachii muscle or the aponeuroses of the palm or soles (Benjamin 2009).

Recent data indicate that fascia in general is not just a passive structure but is contractile. The basis of the contractility is myofibroblasts that appear to be present in many fascia and perform very slow "contractions" lasting many minutes when the tissue is stimulated chemically in vitro (Schleip et al. 2007). In the opinion of many clinicians this finding is trivial, because the high prevalence of the Dupuytrensyndrome shows that the palmar aponeurosis has just this capability, although it is not clear if the mechanism of the Dupuytren contracture is dependent on myofibroblasts. The fascia has also been assumed to be involved in acupuncture effects, in that planes of connective tissue have a close relation to acupuncture points and react very sensitively to the rotations of acupuncture needles (Langevin et al. 2007).

Finally, the fascia has been discussed as a possible source of pain in patients with non-specific low back pain (Yahia et al. 1992). This type of back pain does not originate in bony structures of the spine or the facet joints but in the soft tissues of the low back (muscles, ligaments, fasciae). Non-specific low back pain is one of the most common pain complaints in industrialized countries; therefore, the clarification of a possible contribution of fascia receptors to this pain would be of importance not only for our understanding but also for the management of this type of pain.

To fulfill the role of a pain source in non-specific low back pain, the fascia should have a dense innervation with sensory fibers. However, the TLF was largely ignored as a subject of scientific studies and, therefore, little information is available on the innervation of the TLF and hence on the possible sensory role of the fascia. Even recently, Panjabi (2006) published a comprehensive report of mechanisms of pain generation in non-specific low back pain, but did not mention the TLF as a potential source. Interestingly, the innervation of other structures, such as the small ligaments and the intervertebral discs of the spine, was studied as early as the sixties with the histological methods available at that time (methylene blue; Hirsch et al. 1963). Stecco and colleagues (2007) likewise found abundant nerve fibers in the fasciae of the upper limb, including retinacula and the lacertus fibrosus.

Another problem in assessing the role of the TLF in low back pain is that the few studies performed earlier on fascial innervation are partly contradictory, or questionable with regard to the conclusions drawn by the authors. For instance, Bednar and colleagues (1995) stated that the TLF of low back pain patients "is deficiently innervated". The reason for this conclusion is that the authors did not find any sensory receptors in the tissue specimens they studied. However, the results of a histological study on human specimens by Yahia and colleagues (1992) did show that the TLF is innervated and exhibits free and encapsulated nerve endings. The encapsulated nerve endings probably represented mechanoreceptors. For this article, the free nerve endings are more interesting, because many of them are nociceptive and subserve pain. For the tibial anterior fascia, an important role in the pain of delayed onset muscle soreness (DOMS) has been suggested in a study on human subjects (Gibson et al. 2009). In this investigation, hypertonic saline was injected as a pain-producing agent into the muscle and the overlying structures after induction of DOMS in that muscle. The main result was that the injection directly underneath the fascia caused more pain than into the muscle itself.

The aims of this chapter are twofold: (1) verify or falsify the assumption that the thoracolumbar fascia is innervated, and if so, what types of fibers are present in the TLF; (2) obtain electrophysiological data on the responses of sensory neurons in the spinal dorsal horn to stimulation of the TLF. The underlying question is where in the spinal cord the information from fascia receptors are processed and how neurons with input from the fascia behave in general.

Innervation of the thoracolumbar fascia

The thoracolumbar fascia can play a role as a sensory organ only if it exhibits a dense innervation. Regarding the possible function of the fascia as a source of pain in patients with low back pain, the afferent (sensory) fibers are expected to include nociceptive ones. In our group, we studied the innervation of the thoracolumbar fascia in SD rats in whole mount preparations and coronal sections, both taken from the lumbar level. Close to the spinous processes of the spine the fascia had three layers:

- A thin outer layer consisting of parallel collagen fibers oriented transversely (in the coronal plane).
- A thick middle layer composed of massive collagen fiber bundles running obliquely to the long axis of the body.

• A thin inner layer consisting of loose connective tissue covering the underlying multifidus muscle (Fig. 2.4.1A).

All fibers were visualized with immunohistochemical techniques. As a universal marker for all nerve fibers, antibodies to protein gene product 9.5 (PGP 9.5) were used (Lundberg et al. 1988). The coronal section in Fig. 2.4.1(A) shows PGP 9.5-immunoreactive (ir) neuronal structures mainly in the subcutaneous and outer layer as well as in the inner layer. The fibers included fibers of passage (black arrows) and nerve endings (open arrow). Nerve endings are characterized by a granular structure at a low magnification which is due to the axonal expansions (varicosities) close to the nerve terminal (see below). The fibers of passage did not necessarily belong to the innervation of the fascia; theoretically they may supply tissues other than the fascia.

Figure 2.4.1(D,a) is a quantitative evaluation of the PGP 9.5-ir fibers. In a 5 mm long part of the fascia in coronal sections, the length of all fibers and nerve endings in the various layers of the fascia was measured and the mean fiber length was calculated. The various parts of each bar graph show the mean fiber length in a given layer. The evaluation showed clearly that the great majority of all fibers were situated in the subcutaneous tissue and outer layer of the fascia. In the middle and inner layer only a small fraction of all fibers was found. In the bar graphs subcutaneous tissue and outer layer are pooled, because the outer layer of the fascia proper was often continuous with the subcutaneous tissue.

The peptidergic sensory nerve endings were identified with antibodies to the neuropeptides calcitonin gene-related peptide (CGRP) and substance P (SP; Danielson et al. 2006). CGRP is present in a high percentage of sensory fibers and serves as a general marker for these fibers (Danielson et al. 2006). The SP-containing fibers are a subpopulation of the CGRP fibers, as all SP-positive fibers also contain CGRP. Both fiber types are involved in pain processes in that they induce neurogenic inflammation, i.e., vasodilation and increase in the permeability of blood vessels caused by action potentials that release CGRP and SP from the free nerve endings of sensory fibers (Mense et al. 1996). These action potentials originate in the dorsal roots or peripheral nerve and invade the nerve endings antidromically (against the normal direction of propagation).

One aim of the present study was to perform a quantitative evaluation of CGRP-ir and SP-ir nerve

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Fig. 2.4.1 • Innervation density of the thoracolumbar fascia. (A) Coronal section of the thoracolumbar fascia close to the spinous process L5 (thickness 40 µm). Outer layer: collagen fibers oriented transversely; middle layer: collagen fiber bundles orientated diagonally to the long axis of the body; inner layer: loose connective tissue covering the multifidus muscle. Black arrows mark PGP 9.5-immunoreactive (ir) nerve fibers of passage, open arrows PGP 9.5-ir nerve endings. (B) Three CGRP-ir terminal axons (arrows) with chains of varicosities in a whole mount preparation of the inner layer (dorsal view). The cross striation of the underlying multifidus muscle is visible. (C) SP-ir terminal axon (arrows) located in the subcutaneous tissue close to the outer layer (coronal section, thickness 40 µm). (D) Quantitative evaluation of the mean fiber length in the fascia layers indicated in A. (a) PGP 9.5- ir nerve fibers (PGP 9.5 is a universal marker for all nerve fibers), (b) CGRP-ir nerve fibers, (c) SP-ir nerve fibers. CGRP and SP are markers of peptidergic sensory nerve fibers. White part of the bar: subcutaneous tissue plus outer layer of the fascia; black: middle layer; gray: inner layer. Note that the middle layer of the fascia is free from SP-ir nerve fibers (c).



fibers and sensory endings. Such an evaluation is important because the density of neuropeptide-ir fibers (such as SP and CGRP) had been shown to vary greatly from one tissue to the other (McMahon et al. 1984, 1989; Brismée et al. 2009). Figure 2.4.1 (D,b and c) shows a quantitative evaluation of CGRP-ir and SP-ir nerve fibers. Compared to all (PGP 9.5-ir) fibers, the CGRP fibers were much less numerous: approximately 16%. As in other tissues, the SP-ir fibers were just a small fraction of the CGRP fibers. Figure 2.4.1(B, C) shows examples of free nerve endings that were ir for these neuropeptides. A typical feature of these sensory nerve endings was the widenings of the axon (the so-called varicosities; Fig. 2.4.1B) that look like a string of beads. The varicosities are the sites where stimuli are thought to act.

In the context of this article the SP-ir nerve endings are particularly interesting. As stated earlier, SP-ir fibers are assumed to be nociceptive, therefore the ending shown in Fig. 2.4.1(C) probably represented a nociceptor. In this case, it was situated in the subcutaneous tissue overlying the fascia proper. An interesting finding was that in the middle layer of the fascia no SP-ir fibers or endings were found (Fig. 2.4.1Dc). Teleologically this makes sense, because the middle layer has to transmit the forces that accompany all body movements. If nociceptive endings existed between the collagen fiber bundles of the middle layer they would be excited or even damaged (Sanchis-Alfonso & Rosello-Sastre 2000) by trunk movements. The result would be movement-induced pain even in subjects with otherwise intact low back. The paucity of fibers in the middle layer of the fascia is in line with the statements of Hagert and colleagues (2007), who distinguished between ligaments that are mechanically important yet poorly innervated (e.g., the ligaments of the wrist), and ligaments whose main role appears to be sensory. The latter structures have more loose connective tissue, and the nerve fibers are predominantly located in this tissue.

Regarding the extent of sensory innervation of the TLF, the following coarse calculation can be made: The peptidergic CGRP- and SP-ir fibers are not the only sensory fibers, because there are also nonpeptidergic/lectin positive thin sensory fibers. Even if the same number of nonpeptidergic sensory fibers are added to the peptidergic ones - which is probably too high a number, because both groups overlap (Hwang & Valtschanoff, 2005) – all sensory fibers would make up approximately 1/3 of all fibers. This means that about two-thirds of the innervation of the fascia is efferent, probably consisting of sympathetic postganglionic fibers. Indeed, preliminary results from our laboratory indicated that with a specific marker for sympathetic fibers (tyrosine hydroxvlase, an enzyme necessary for the synthesis of (nor) epinephrine) a large fraction of the fibers in the TLF can be labeled. This finding may explain the great influence psychological stressors have on the pain of patients with non-specific low back pain (Brage et al. 2007).

Electrophysiology

So far, just a few electrophysiological studies have addressed primary afferent fibers and dorsal horn neurons (second-order neurons) that have receptive fields (RFs) in lumbar soft tissues. The first description of mechanosensitive afferent units in lumbar intervertebral discs and adjacent muscles was made by Yamashita and colleagues in rabbits (1993). They identified 13 mechanosensitive units. The RFs of 10 units were located in the psoas muscle, three in the intervertebral disc area. The mechanical thresholds of the afferent units from muscle were low to high. On the other hand, the thresholds of units with RFs in the intervertebral disc area were exclusively high (>160 g). Yamashita and colleague suggested that the units in the psoas muscle may contribute to both nociception and proprioception, while the units in the disc area may serve as nociceptors. because they had a high mechanical threshold.

In the literature, there is no information about afferent fibers from the TLF. In 1995, unmyelinated nociceptors (n = 57) in paraspinal tissues of rats were classified according to the anatomical distribution of their RFs as follows (Bove & Light 1995):

- **1.** Musculotendinous units (44%): RFs within muscle, tendon, or associated fascia.
- **2.** Neurovascular bundle units (21%): RFs associated with a neurovascular bundle.

- **3.** Subcutaneous units (2%): RFs on the undersurface of the skin.
- **4.** Multiple tissue type (33%): Combination of the three types mentioned above.

It is worth mentioning that seven of the musculotendinous group receptors responded to stimulation of the fascia, although no detailed information about the mechanical threshold and the responsiveness of the unit to chemical and thermal stimulations was given. It is also noteworthy that the centers of RFs were located in the tail or base of the tail in most cases when the recordings were made from the dorsal roots L6 and S1. This finding demonstrates that there is a caudal shift of the RFs in the deep tissues of the low back relative to the segmental level of the dorsal roots through which the afferent fibers enter the central nervous system.

Electrophysiological (microelectrode) recordings of dorsal horn neurons receiving input from lumbar tissues in cats were made for the first time by Gillette and colleagues (1993). They recorded the neurons extracellularly in the extreme lateral dorsal horn of the spinal segments L4-L5. The lateral part of the dorsal horn receives input from dorsal ramus fibers innervating the low back soft tissues like the multifidus muscle (Grant 1993; Taguchi et al. 2007). Gillette and colleagues found that most neurons (72%) received excitatory convergent input from skin and deep tissues (sd cells). Some neurons (23%) had RFs restricted to the skin (s cells), and very few neurons (5%) had RFs in deep somatic tissues (d cells). The deep-tissue RFs were located in facet joint capsules, periosteum, ligaments, intervertebral disc, spinal dura, low back/hip/proximal leg muscles, and tendons. Again, most RFs in the deep tissues of the cat low back were located at the vertebral level L6–L7 while the recordings were made in the spinal segments L4-L5. Apparently, the caudal shift of the RFs relative to the spinal segment where the information is processed is a general phenomenon. So far, however, no systematic study on the relationship between the segmental location of the RFs in the low back and that of dorsal horn neurons processing sensory information from that region has been carried out. Likewise, possible changes in this arrangement under chronic patho(physio) logical conditions have not been examined.

Recent work of our group has revealed the existence of nociceptive input from the TLF to dorsal horn neurons, suggesting that the TLF is a possible source of low back pain (Fig. 2.4.2). In these



Fig. 2.4.2 • Dorsal horn neurons processing input from thoracolumbar fascia. (A) Fascial receptive fields of dorsal horn neurons recorded in thoracic spinal segment T 13 (a) and lumbar spinal segments L1 (b) and L2 (c). Numbers indicate spinous processes of lumbar vertebrae. Note that the receptive fields were shifted caudad relative to the spinal segments in which the neurons were recorded. (B) Proportion of dorsal horn neurons with input from thoracolumbar fascia (TLF) in spinal segments T13–L5. The spinal segments T13–L2 received TLF input, while in the segments L3–L5 it was missing. (C) Original registration of action potentials from a single dorsal horn neuron during and after pinching the fascia for approximately 1.5 s (black bar underneath the registration).

experiments, systematic extracellular recordings from dorsal horn neurons were made in the spinal segments T13-L5 in rats. The experimental procedures are described in detail elsewhere (Taguchi et al. 2008). Briefly, the experiments were done in vivo under deep anesthesia. The TLF and spinal cord were exposed by cutting the low back skin along the midline and removing the soft tissues and vertebral lamina covering the lumbar and sacral spinal cord. A pool was formed from the skin folds and filled with silicon oil to cover the recording sites in the spinal cord and the stimulation sites in the soft tissues of the low back more caudally. Usually, there was a distance of about 3-5.5 cm between the recording and the stimulation sites. The surgical exposure of the spinal cord did not damage the RFs in the low back tissues caudally of vertebra L2 since the spinal segments T13-L5 were located more cranially at vertebral level T10-T13 (Taguchi et al. 2008). The fascial RFs were searched by pinching (noxious mechanical stimulation) of the TLF with a sharpened watchmaker's forceps. A sample recording of the discharges of a single neuron in the segment T13 is shown in Fig. 2.4.2(C). The neuron showed afterdischarges after the noxious stimulus, which is seen mainly in nociceptive neurons. Many dorsal horn neurons with fascial RFs also responded to stimulation with a small cotton ball soaked with 5% hypertonic saline and put on the surface of the fascia. The responses to this stimulus often occurred at a very short latency. This finding is in line with the neuroanatomical results showing that the SP-positive nerve fibers were mainly found in the subcutaneous tissue and outer layer of the fascia (Fig. 2.4.1D,c).

Neurons with RFs in the TLF were found in the spinal segments T13-L2, but not in L3-L5 (Fig. 2.4.2B). Six to 14% percent of the dorsal horn neurons in the spinal segments T13-L2 had input from the TLF. The approximate centers of RFs in the TLF were located at the vertebral levels L3-L4. L4-L5 and L5-L6 when the recordings were made in the spinal segments T13, L1 and L2, respectively. The location of the fascial RFs was consistently shifted three to four segments caudally relative to their recording site (Fig. 2.4.2A). This shift was also true for dorsal horn neurons with muscular or other deep-tissue RFs in the low back (Taguchi et al. 2008). Usually, all RFs in the TLF were located on the side ipsilateral to the recording site (Fig. 2.4.2A). Most of the neurons with RFs in the TLF had convergent input from the skin and other deep tissues or regions in the low back, abdominal wall, hip, and proximal/distal leg. This finding may explain the diffuse nature of nonspecific low back pain in patients.

In experiments employing recordings from dorsal horn neurons with input from the gastrocnemiussoleus muscle and low back muscles, both low- and high-threshold mechanosensitive cells were found (Hoheisel et al. 2000; Taguchi et al. 2008). So far, such a classification based on the mechanical threshold of dorsal horn neurons has not been undertaken for neurons with input from the TLF. However, our results indicate that most of the neurons with fascial input had a high mechanical threshold and, therefore, likely were nociceptive neurons.

In experimentally induced pathological situations, the excitability of dorsal horn neurons was increased. In animals in which a tonic-chronic (6 days, duration) myositis of the multifidus muscle had been induced, the proportion of neurons with fascial RFs in the thoracolumbar region rose significantly. In addition, in the spinal segment L3 that does not normally receive input from the TLF, more than 10% of the cells responded to input from the TLF in myositis animals. These electrophysiological recordings from dorsal horn neurons of rats revealed that the TLF is an important source of nociception originating from the low back and could contribute to the pain of patients with chronic low back pain.

Interestingly, after induction of the myositis the increase in the proportion of neurons responding to input from the TLF was higher than that of cells responding to input from the inflamed muscle itself. In line with this finding, in human subjects in whom DOMS had been induced, the fascia covering the overexercised muscle became more sensitive to painful stimulation than the muscle (Gibson et al. 2009). Collectively, these findings suggest that in patients with nonspecific low back pain fascia tissue may be a more important pain source than the low back muscles or other soft tissues.

In conclusion:

- **1.** Dorsal horn neurons receiving input from the TLF do exist.
- **2.** The input from the TLF is processed in spinal segments cranial to L2 in animals with intact soft tissues.
- **3.** The approximate center of the RF in the TLF was always shifted several segments caudad relative to the spinal segment recorded from.
- **4.** In animals with a tonic-chronic myositis, the size of the RFs in the TLF expanded, the proportion of neurons with RFs in the TLF increased, and the segment L3 acquired TLF input.

These neurophysiological findings, together with the neuroanatomical data, are relevant for a better understanding of the mechanisms of nonspecific low back pain. Under chronic pathological conditions, an enhanced nociceptive input from the TLF could contribute to low back pain in patients.

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Fascia as a body-wide communication system

James L Oschman

A single-celled paramecium swims gracefully, avoids predators, finds food, mates, and has sex, all without a single synapse. "Of nerve there is no trace. But the cell framework, the cytoskeleton might serve."

Sherrington, 1951

Introduction

This chapter begins with some evolutionary considerations regarding communication in the fascia and other components of the extracellular matrix and within the cells that maintain them. These considerations lay a foundation for exploring the nature of non-neural and nonhormonal communications in the mammalian organism, as well as how the fascia interacts with the brain and therefore with consciousness.

When we think of communication in the human body we usually first think of nerves and synapses. The purpose of the above quotation is to remind us of the existence of evolutionarily ancient communication systems that are present in single celled organisms that are entirely lacking in nerves or synapses. How does a single-cell creature, such as a paramecium, lead such a sophisticated life? How does it hunt living prey, respond to lights, sounds, and smells, and display complex sequences of movements without the benefit of a nervous system? Bray (2009) proposes that cells are built of molecular circuits that perform logical operations, as electronic devices do. He also suggests that the computational properties of cells provide the basis of all the distinctive properties of living systems, including the

ability to embody in their internal structure an image of the world around them. These concepts, which are supported by the information to follow, account for the adaptability, responsiveness, and intelligence of cells and organisms. These properties also extend into the connective tissue terrain surrounding all cells in the mammalian organism.

Prokaryotes – organisms lacking a cell nucleus or any other membrane-bound organelles, even those as simple as flagellated bacteria – are likewise capable of sensing and responding to various environmental stimuli and moving toward or away from them as necessary for their survival. In this historical and evolutionary context, the nervous system is seen as a relatively new "invention" that functions in cooperation with an older communication system that has had a much longer period of evolutionary refinement – the body-wide communication system that is the topic of this chapter.

Because of the relative ease with which the nervous system can be studied, and because of its obvious importance, the brain has been studied with a vast array of analytical tools, and we know enough about it to fill many books and journals. However, one does not have to dig very deep into this literature to find that there are many unanswered questions. For example, the recent discovery that the connective tissue cells in the brain also form a communication system has returned the whole of neuroscience to the drawing board. In mammals, connective tissue cells called glia (the Greek word γλια means "glue") constitute some 50% of the volume of the brain. Decades of research have required revision of the traditional view that glial cells function purely for mechanical and nutritional support. We now know that glial cells interact morphologically, biochemically, and physiologically with neurons throughout the brain, modulate neuronal activity, and influence behavior (Castellano López & Nieto-Sampedro 2001; Koob 2009). A new cutting edge branch of both neuroscience and fascial research has been born, based on the relationship between connective tissue cells and neuronal processes. Those who study the fascia as an all-pervasive system, as will be defined below, will recognize that one of the most vital relationships in the body has to be the relationship between the connective tissue and the nervous system.

Some biologists regard the modern mammalian cell as a microorganism (e.g., Puck 1972). Mammalian cells contain miniature "musculoskeletal systems" composed of microtubules (the "bones" of the cell), microfilaments (the "muscles" of the cell), and other molecules that can act as a sort of "connective tissue" within the cell. These cellular components enable cells to change shape and to migrate from place to place. In recent years it has been discovered that bacteria also contain a number of cytoskeletal structures that are homologs of the three major types of eukaryotic cytoskeletal proteins, actin, tubulin, and intermediate filament proteins (summarized by Shih & Rothfield 2006).

The cytoskeleton is often regarded as the "nervous system" of the cell. The extracellular coats of the "primitive" microorganisms evolved into the mammalian extracellular matrix. Specifically, the extracellular sugar polymer coatings of individual bacteria, viruses, and protozoa extended the "reach" of these ancient organisms into their environment and formed the oldest and most pervasive information and defense system in nature. The connective tissue is the modern expression of these ancient cell coats.

This chapter is an exploration of the concept that these ancient communication systems persist throughout the modern mammalian organism and that their existence helps explain a number of phenomena that are difficult to account for by neural mechanisms. The inquiry has been guided and inspired in large measure by conversations with a broad range of bodywork, energetic, and movement therapists who have daily and remarkable encounters with these systems and who have therefore developed a keen curiosity about their nature.

The fascia

Findley and Schleip (2009) have defined fascia broadly to include all of the soft fibrous connective tissues that permeate the human body. Their definition has the important feature of blurring the arbitrary demarcation lines between various components of the connective tissue so that we can view the fascia as "one interconnected tensional network that adapts its fiber arrangement and density according to local tensional demands." Pischinger (2007) describes the fascial system as the largest system in the body as it is the only system that touches all of the other systems. Finando and Finando (2011) summarize evidence that the ancient acupuncture meridian system shares many structural, functional, and clinical characteristics with the fascial system. Specifically, like the acupuncture meridian system, the fascia may be viewed as a single organ, a unified whole, the environment in which all body systems function. There is a virtually one-to-one correspondence between the therapeutic approaches to the fascia and to acupuncture. For example, Pischinger (2007) states that needle puncture produces a reaction in the entire intercellular-extracellular matrix. The diversity of conditions that respond to acupuncture treatment may be explained by a review of the recently understood properties of the fascia. The involvement of the fascia in dysfunction and disease is pervasive. It is believed that, to some extent, the fascia will necessarily be involved in every type of human pathology (Paoletti 2006; Pischinger 2007). The fascia is the one system that connects to every aspect of human physiology. Langevin (2006) and Langevin and Yandow (2002) suggest that the fascia is a metasystem, connecting and influencing all other systems, a concept with the potential to change our core understanding of human physiology.

These are valuable perspectives as they help address the increasing interest in whole-systems phenomena that distinguish holistic manual therapies from methods that focus on parts rather than wholes. Experience often shows that formerly intractable health issues are resolved by taking a broader view of a patient's problems. Stated differently, "There are no local problems" (Spencer 2007), and the corollary, "There are no local treatments."

Along with these holistic perspectives come questions such as:

How do we account for the unitary nature of a living organism: the way it responds as a whole to any stimulus – as if every part of it knew what every other part is doing?

and:

How is it that an organism behaves as a whole, and not just a collection of parts?

Packard (2006)

These issues are related to the theme of this book, since much of the success of modern manual therapies stems from a willingness on the part of practitioners to unwind a patient's entire traumatic history, including all of the resulting compensations, which is very different from treating a current complaint.

Moreover, the way the interconnected fiber systems of the fascia adapt to both local and global forces takes us to one of the key unsolved issues in medicine and biology. This issue is the mechanism by which an organism develops from an embryo into an adult, and the equally important mechanism by which the adult organism references the embryonic formative processes when needed to restore the original structure after injury or disease. While there may be an impression that the mechanisms involved in morphogenesis are well known, they are not. Biological patterns persist in the face of changes in physical activity and trauma, but previous widely-taught ideas of how this is accomplished have been discovered to be inaccurate:

- DNA is *not* the blueprint of the organism.
- Ontogeny (morphogenesis or the developmental history of an organism) does *not* recapitulate (repeat) phylogeny (the evolutionary history of a species).
- The growth of an organism is *not* brought about by a set of linear cause and effect events like the construction of an automobile on an assembly line.
- Differentiation is no longer viewed as a one-way street, i.e., that once a cell has become "committed" to become, say an intestinal cell, it cannot revert to the undifferentiated state.

To thoroughly explore wound healing, the human body's capacity to adapt to and recover from stress and trauma and other essential biological phenomena, we extend the definition of fascia to include the denser parts of the connective tissues, cartilage and bone, whose fiber systems are continuous with the fascial elements in the soft tissues. The fiber systems in the fascia are embedded in a polyelectrolyte ground substance, and what distinguishes bone from soft tissues is the ossification of the ground substance. The fiber systems within bone are continuous with those in the soft tissues, for example at the places where tendons and ligaments insert into bone.

Tracing the kinetic chain through the living matrix

Since our exploration will also go beyond gross anatomy to the level of tissues, cells, organelles, nuclei, DNA, and other molecules, we introduce an even more encompassing concept, the **living matrix**. The living matrix includes the connective tissue and fascial systems as defined above as well as the transmembrane proteins (integrins and adhesion complexes), cytoskeletons, nuclear matrices, and DNA. Figure 2.5.1 illustrates the living matrix concept.

We can trace the molecules of the kinetic chain through the living matrix. The kinetic chain is an interconnected tensional network within the living matrix. All movement, of the body as a whole or of its smallest parts, is created by tensions carried through the living matrix. In laying out the following sequence of connections it must be recognized that some parts of the network have been studied more thoroughly than others.

We begin with the tilting of the head of the myosin molecule, widely regarded as the origin of all muscular movements. This tilting causes movement of the myosin filaments with respect to the actin filaments. The actin molecules in turn exert tension on the filaments of the Z disk. The Z disk in turn connects to the muscle cell surface (sarcolemma) and to collagen molecules in the endomysium. In this way, tensions developed within the sarcomeres are conducted to the surrounding endomysium. These tensions, as well as those conducted at the musculotendinous junctions, are further conveyed by tendons to the bones. The functional anatomy is complex, however, as has been carefully reviewed by Huijing (2007).

As the title of this chapter suggests, it is of interest to explore the possibility that the body-wide fascial network, the kinetic chain, and other components of the living matrix may serve additional roles beyond conducting tensions. One such role emerges when we explore the mechanisms by which the body adapts to the demands imposed upon it.

Regulation of fascial architecture

Wolff's Law (1892) is frequently mentioned as a key mechanism in the adaptation of the structure of the body to the ways it is used, abused, and traumatized:

PART TWO





Fig. 2.5.1 • The living matrix concept.

The form of the bone being given, the bone elements (collagen) place or displace themselves in the direction of the functional pressure and increase or decrease their mass to reflect the amount of functional pressure.

Cited in Bassett (1968)

We now know that Wolff's Law applies to more than bone - it is relevant for virtually all of the connective tissues, including tendons, ligaments, and so on. We can ask precisely what is the mechanism of Wolff's Law: precisely what connects "functional pressure" (tensions and compressions) with anatomical structure? The question is important beyond musculoskeletal mechanics. It is a key question in morphogenesis, since cellular migrations during development and during wound repair exert "functional pressures" on surrounding tissues that are important in determination of the "final form" of the tissues. Chen and Ingber describe how mechanical forces transmitted through the system ultimately reach the cytoskeleton and nuclear matrix, where they can produce biochemical and transcriptional changes by mechanochemical transduction (Chen & Ingber 2007).

Several additional signaling mechanisms have been explored to explain the connections between "functional pressure" and tissue structure. Each of these signaling mechanisms involves a particular form of energy conducted through the living matrix and/or through the associated water and fluid phases of the connective tissue. We begin with the role of electrical fields, then move on to light and sound.

Electrical fields and the piezoelectric effect

The collagen fibers and fiber bundles of the myofascial system are to a high degree associated in parallel arrays that give them their great tensile strength and flexibility, while at the same time giving them a high degree of crystallinity. This is a property of soft tissues that is not always taken into account. The crystals in the living matrix have little resemblance to familiar mineral crystals such as quartz or diamond. The hardness of mineral crystals arises because the units (atoms and molecules) of which they are composed are roughly spherical and are packed tightly together in very strong polygonal arrays. In contrast, the organic crystals comprising the myofascial system are composed of long, thin, flexible filaments such as actin, myosin, collagen, and elastin. The result is flexible rather than rigid crystals. In fact, they are best described as liquid crystals.

Liquid crystallinity gives organisms their characteristic flexibility, exquisite sensitivity and responsiveness, and optimizes the rapid noiseless intercommunication that enables the organism to function as a coherent coordinated whole.

Ho (1997)

Highly ordered systems of this kind have special properties that have been studied by physicists for a long time. The biological significance of these crystals was emphasized by Szent-Györgyi (1941):

If a great number of atoms is arranged with regularity in close proximity, as for instance, in a crystal lattice, the...electrons... cease to belong to one or two atoms only, and belong to the whole system...A great number of molecules may join to form energy continua, along which energy, viz., excited electrons, may travel a certain distance.

This statement introduced the important discovery, now confirmed, that proteins are semiconductors, and laid the foundation for the new field of electronic biology or solid-state biochemistry. Very few scientists appreciated the significance of this development. The barrier seemed to be unwillingness among most biologists to investigate the biological significance of quantum physics. Fortunately this situation has changed dramatically, and Szent-Györgyi's seminal work in molecular electronics and semiconduction in collagen is finally being recognized (Hush 2006).

Another important property of liquid crystals is piezoelectricity. When put under compression or tension, these materials develop electric fields. Deformations of bones, teeth, tendons, blood vessel walls, muscles, and skin all give rise to weak electric fields, which is thought to be a result of the piezoelectric effect. The piezoelectric constant for a dry tendon, for example, is nearly the same as that for a quartz crystal (Braden et al. 1966).

There is some disagreement as to whether the electric fields produced by deformations of connective tissues are entirely due to the piezoelectric effect. Another mechanism that could contribute to the electrical properties is the streaming potential (Bassett 1968).

There has been interest in the biological significance of these electrical effects. It appears that every movement made by the body generates electric fields due to the compression or stretching of bones, tendons, muscles, etc. In addition, electric fields occur as a consequence of nerve conduction and the depolarization of the muscle cell membranes. It has been proposed that all of these electric fields spread through the surrounding tissues, providing signals that inform the cells of the nature of the movement, loads, or other activities occurring elsewhere in the body. Cells such as fibroblasts and osteoblasts are thus able to adjust their activities in maintaining and remodeling the tissues according to the loads they are carrying. This is thought to be the mechanism of Wolff's Law, and the process by which movement and exercise maintain the skeleton, while long periods of bed rest or space travel in zero gravity conditions lead to loss of bone mass. Bodywork, energetic, and movement therapists of all kinds are familiar with the fact that tendons become thickened and hardened in response to chronic stress. They are also familiar with therapeutic methods that lessen the tensions in the myofascial network, by relaxing chronically tightened muscles and softening dense regions, particularly at the places where the tendons insert into the bones.

Athletes, musicians, dancers, and other performers experience the progressive adaptations of structure, function, motion, and energy that occur when an activity is practiced again and again. An extreme example is the body-builder, who through the stimulus of constant exertion, brings about a dramatic alteration in body form. Not only do the muscles increase in size and strength, but the other components of the myofascial system increase as well. The delicate skill of the concert violinist is an example of the same phenomenon - the gradual perfection of form and motion as the body adapts to the way it is used. It is thought that the orderly and concerted changes in structure just described, coordinated by the communications between the various tissues and the cells, are, at least in part mediated by electric fields produced, by the piezoelectric effect, streaming potentials, and other activities related to motor control. A stimulus for the research was the discovery by Dr. Robert O. Becker and others that weak electrical currents can facilitate the healing of bone fractures.

While a success with a therapeutic approach does not necessarily prove the theoretical mechanism that the approach is based upon, there is no question that clinical application of weak electrical fields can stimulate osteogenesis to the extent that the method has become widely used for treating nonunion and delayed union of bone fractures, even in bones unhealed for as long as 40 years (Bassett 1995).

Precisely how do electrical fields generated during motor activity get from their sources to nearby fibroblast or osteoblast cells, and thence to the cell nucleus where protein synthesis is regulated? Bassett (1995) summarized a cascade of activities that crosses the cell membrane, moves through the cytoskeleton to the nucleus and DNA. Physiologists regularly view charge transfer as the movements of charge carriers such as sodium, potassium, and chloride ions, as in other physiological processes. It is likely that other charge carriers and other forms of energy are involved as well. Light and sound may also be involved.

Interest in light emission from the fascial liquid crystals is supported by the discoveries of Fröhlich (1988), who demonstrated from both theoretical and experimental perspective that when the energy levels in these liquid crystals reach a certain point, the molecules begin to vibrate coherently, leading to the emission of coherent light. These light emissions have now been documented.

Light

Light or biophotons constitute yet another form of energy that is generated within the body and that moves through the living matrix. The modern era of biophoton research, from 1974 onwards, began with the work of Fritz-Albert Popp and his colleagues in Germany. During the past 30 years, Popp and colleagues around the world have demonstrated conclusively that living systems absorb and emit coherent biophotons. There are now about 40 groups, in a dozen or so countries, researching the theory and practical applications of this research, using state of the art techniques.

From this research we now know that all organisms, including humans, emit a glow that is too faint to be detected with the eye, but that can be measured precisely with photomultipliers that amplify weak signals millions of times. The intensity of this biophotonic glow is some tens of thousands of photons per square centimeter per second. Bischof (2005) calculates that this glow corresponds to the light of a candle seen from a distance of 15 miles. Biophotons range in wavelength from 200 to 800 nm, i.e., from the ultraviolet through the visible spectrum to infrared light. These emissions should not be confused with chemical bioluminescence, which is much stronger and has entirely different properties and origins. In contrast to chemical bioluminescence, biophoton emission increases in intensity hundreds or thousands of times before death of cells, and then ceases upon cell death. Injury to cells stimulates

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the production of biophotons. The coherent biophotonic light is not steady, but changes with any change in the activity of the organism. Biophoton output changes during the cell cycle, and is influenced by any change in the physiological state of the organism. A recent discovery is that biophotons are emitted from acupuncture meridians when points are stimulated with different methods used by acupuncturists (Schlebusch et al. 2005).

Popp has summarized years of biophoton research with the concept of *Gestaltbildung*: cell coordination and communication. With biophoton emissions, Popp provided an answer to the question of morphogenesis as well as *Gestaltbildung*:

- Photonic communication enables every cell to know what every other cell is doing.
- Weak light emissions orchestrate the body.
- Emissions occur at the quantum level.

For those interested in the diverse roles of fascia and connective tissue in whole-body communication, an important point of this section is that the liquid crystalline domains within the connective tissues are strong emitters and sensors of biophotons.

Muscle sounds

It is well known that contracting muscles produce sounds that can easily be recorded with standard microphones (e.g., Oster & Jaffe, 1980; Stokes & Cooper 1992). The recording of the acoustic myogram provides a simple, noninvasive, portable measure of the skeletal muscle that can be used for monitoring muscle fatigue, controling prosthetic devices or diagnosing pediatric muscle disease. It is instructive for readers to listen to their own muscle sounds. This is done simply by filling a bathtub to a depth that will immerse the ears when lying face up in the tub, taking care not to immerse the nose. Clenching the teeth or moving other facial and even neck muscles will produce rumbling sounds that can be heard with the ears under water. Those with sensitive hearing may be able to hear sounds produced by voluntary contractions of other muscles in the body. The point is that muscle contractions produce sounds that can be conducted through the tissues. Whether or not these sounds have regulatory significance seems to be unknown.

The recognition of muscle sounds adds another dimension to our considerations of the regulatory significance of information transfer through the living matrix. The reason is that sounds and any other forms of mechanical vibration will cause crystalline connective tissues to produce oscillating electrical fields of the same frequency as the sounds because of the piezoelectric effect. Hence, after considering just two forms of energy, electricity and sound, we are seeing some of the ways the energies interact.

Conclusions

The information summarized in this chapter is intended to introduce the reader to some of the possibilities of non-neural transfer of energy and information in the human body and the role of the fascia in these phenomena. Much of the evidence for these phenomena is circumstantial, and science, unlike the law, does not reach definitive conclusions on the basis of circumstantial evidence. It is challenging to study the phenomena discussed here because traditional measurement techniques do not apply. In contrast to the nervous system, one cannot simply insert a microelectrode into the fascia and establish the nature of the informational processes taking place. The author suggests that it will soon be possible to explore information processing in fascial systems, and entirely new perspectives on fascia and manipulative therapies will follow.

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Force transmission and muscle mechanics

General principles

Peter A Huijing

Muscle force is generated within sarcomeres within myofibers (i.e., muscle fibers). The sarcomere forces (summed according to the rules for serial and parallel arrangement) need to be exerted outside myofibers to be able to cause movement of body parts.

In short, the rules for summation of sarcomere effects indicate that forces are added for sarcomeres arranged in parallel, and shortening and shortening velocity are added for sarcomeres arranged in series.

There are two fundamental ways to look at force transmission.

- 1. The direct (mechanics) way: considers a force transmitted from the sarcomeres of a muscle onto, for example, the tendon and from there to bone to cause movement of a body segment.
- 2. The inverse mechanics way: deals with questions: which structures are the sources of so-called reaction forces that allow the muscle to exert force? An active or stretched passive muscle will shorten unless opposed by a load (an opposing force) that will prevent shortening to a length at which no force can be generated (slack length). This opposing force is a reaction force that is equal to the force exerted by the muscle (action = reaction), but has an opposite direction. It may be exerted by the tendon or by other structures arranged in series with the sarcomeres.

It is clear that the two approaches should yield the same answers. Even though the concept of inverse mechanics seems more complex initially, we will use this approach because it is easier to avoid mistakes.

As there are two types of structures arranged in series with sarcomeres, tendons and fascia, two types of force transmission are distinguished.

Myotendinous force transmission

Each myofiber (muscle fiber) is equipped (at least at one end) with a myotendinous junction (Fig. 3.1.1A). The thin filaments of the last sarcomere of myofibrils within myofibers are attached sideways through the sarcolemma to collagen fibers of the aponeurosis (tendon plate) that invade the invaginations of the myofiber but remain outside of the cell. The supramolecular structures involved in such connections are shown in Fig. 3.1.2A-C. The myotendinous loads exerted on the last sarcomere are transmitted to the next sarcomeres in series within each myofibril. If this is the only type of force transmission, forces in all sarcomeres in series within a myofibril need to be equal. If this is the exclusive reaction force available, and if all sarcomeres have identical properties, such sarcomeres will shorten until their identical force is in equilibrium with the reaction force. In static final conditions, this will yield identical sarcomere lengths within the myofiber.

Myofascial force transmission

Via the intracellular cytoskeleton and trans-sarcolemmal molecules, connections between sarcomeres and surrounding collagenous fibers do not exist only at the ends of myofibers, but are present along their full periphery. Therefore, multiple reaction forces are exerted onto sarcomeres within a myofiber. If these sideways connections supply a notable part of the reaction forces (which can also best be considered as a load on the



Fig. 3.1.1 • The myotendinous junction. (A) Low power electron micrograph showing this junction of one myofiber. The intracellular parts (e.g., sarcomeres) are dark, and light areas are extracellular materials. A considerable area of contact between the two exists because of many invaginations of the myofiber. The aponeurosis is made up from many little tendons belonging to single myofibers. (B) High power electron micrograph showing one invagination containing collagen fibrils (C) that will form the myofiber's small tendon (further to the right). Within the intracellular part thick and thin filaments of the last sarcomere are indicated. Across the sarcolemma and basal lamina (dark line border of the invagination and myofiber) the thin filaments are connected to the collagen fibrils.





sarcomere additional to the myotendinous one) we have force transmission onto the endomysium. We refer to such transmission as myofascial force transmission. As a consequence, not all sarcomeres in series need to be at identical lengths and forces: Forces of subsequent sarcomeres need not be identical since myofascial and myotendinous reaction forces can prevent some of them from shortening more than others that are exposed exclusively to myotendinous loads (Fig. 3.1.1B). It should be noted that some authors consider intramuscular myofascial force transmission as limited to transmission solely between adjacent muscle fibers (e.g., Chapter 1.1). In contrast, we will argue that the muscular connective tissue stroma itself is a pathway of force transmission leading to force transmission beyond the fascicle and even further beyond the borders of muscle (Chapter 3.2).

Myofascial force transmission

An introduction

Peter A Huijing

Intramuscular substrates of myofascial force transmission

Endomysia constitute tubes for each myofiber. Note, however, that endomysial walls are shared between adjacent tubes (and myofibers). This causes a continuous honeycomb type of structure of muscular connective tissues until the fascicle borders are reached (see Chapter 1.1). The collections of fascicles are made up in a similar way, having the perimysium as borders delimiting fascicles, so that within a muscle a stroma of continuous tubes is present that is delimited by the epimysium, surrounding the whole muscle.

Myofascial force transmission limited to the intramuscular domain is called intramuscular myofascial force transmission. This term is used also in the case of force transmission of a single myofiber or fascicle operating within its endomysial or perimysial tunnels (Ch. 8.4). In those cases, and that of a fully dissected muscle, reaction forces have to be exerted via tendons as these are then the only effective connections to the outside world (Huijing et al. 1998).

Epimuscular myofascial force transmission and its substrate

If a *myofascial* load (reaction force from fascial structures) is exerted onto muscle (Huijing & Baan 2001), force is transmitted onto the intramuscular

stroma via the epimysium. Therefore, such transmission is called epimuscular myofascial force transmission.

Two pathways are available for such transmission:

- Directly between two adjacent muscles (occurring exclusively within a muscle group: synergistic muscles). We call this specific case intermuscular myofascial force transmission.
- Between a muscle and some extramuscular structures, such as the neurovascular tract (i.e., the collagen-reinforced structure in which blood vessels, lymphatics and nerves are embedded), intermuscular septa between muscle groups, interosseal membrane, periosteum, general (or deep) fascia, etc. We call this extramuscular myofascial force transmission to emphasize the role of extramuscular tissues. Forces exerted this way may play a role in stabilizing joints, or be exerted on bones and other extramuscular structures, but may also be exerted at other muscles.

All of the tissues discussed above (with the exception of the aponeuroses and tendons) are part of a continuous fascial system. By itself, the fact that they are connected will not warrant force transmission; if the connections are very compliant (i.e., not stiff), force will only be transmitted after very high length changes that will stiffen connections. However, experiments indicate that also after more moderate length changes sizable fractions of muscular force may be transmitted. This means that fascial structures that are not dense depositions of collagen fibers may transmit some muscular force, and therefore it has been argued that the term "loose connective tissue" for such structures is inadequate (Huijing & Langevin 2009) and the term "areolar" is preferred for such tissues.

Effects of epimuscular myofascial force transmission

Proximo-distal force differences

Due to additional myofascial loading, forces exerted at the origin and insertion of muscle are not equal (Huijing & Baan 2001). The net myofascial load (i.e., the vector sum of all such loads, involving size and direction) will keep sarcomeres at one end of myofibers within muscle longer than at other locations within the same myofibers, i.e., different active forces exerted locally (Fig. 3.2.1A). A force equal to the additional load is integrated into the force exerted at the opposite end of the muscle. Active force of several muscles may appear also at the insertion of another muscle, as long as myofascial connections to the extratendinous tissues are intact (Rijkelijkhuizen et al. 2005, 2009).



Distributions of sarcomere lengths within muscle and its myofibers

Myofascial loading will cause a distribution of lengths of sarcomeres arranged in series within myofibers (serial distribution). Most of such additional force is borne by active sarcomeres (since they are very stiff), but may also be borne by the connective tissue stroma of muscle and will be added to the active force exerted.

If the point of application of myofascial loads on myofibers and their size and direction were identical, sarcomere length distributions would be limited to serial ones. However, a simple test illustrates that the situation is more complex. If one suspends equal masses from the tendon of a (horizontal) muscle, the muscle is pulled down exposing the extramuscular neurovascular tract (Plate 3.2.1A), but this tract is pulled down more at the distal tendon than at the proximal tendon. This indicates that the tract is stiffer at the proximal side of the muscle. As a consequence, myofibers located proximally within muscle will, on average, be longer than more distal ones. Therefore, parallel distributions of sarcomere lengths will be present as well. The nature of both types of distributions will vary with the specific conditions of myofascial loading. It is hard

> Fig. 3.2.1 • Myofascial force transmission and some of its consequences. (A) Proximo-distal force differences. The net myofascial load on the muscle is integrated into either the proximal force (left panel) or the distal force (right panel) depending on the loading direction. (B) Comparison of distal forces of unconnected and myofascially connected muscles after distal lengthening. As the agonistic muscle is lengthened the force in the syneraistic muscle drops. (C) Connections and myofascial loading of antagonistic muscles across the intermuscular septum. In these conditions part of the antagonistic muscle force will be exerted at the distal tendon (right) of the agonistic muscle.

to measure such distributions, but finite element modeling (Ch. 8.5) allows the study of such principles even in quite complex conditions of loading.

Myofascial interaction between muscles

Dissection experiments (Maas et al. 2005) indicate that intermuscular transmission plays a role, but extramuscular myofascial force transmission is the more immechanism for muscular interaction. portant Intermuscular mechanical effects are present through two coupled events of extramuscular myofascial force transmission (muscle to extramuscular tissues to another muscle). Myofascial interaction was shown for synergistic muscles involving both intermuscular and extramuscular transmission (Huijing & Baan 2001; Huijing 2003). During experiments this is apparent as follows: After the agonistic muscle is lengthened at its distal tendon while its synergistic muscle is kept at constant length (Plate 3.2.1B), distal force of the isometric synergistic muscle is decreased (compared to the unconnected case) with increasing lengths of its adjacent muscle. The lengthened muscle creates, or enhances, a distally directed myofascial load on the isometric synergistic muscle. At its proximal tendon, this load is integrated into force exerted by the muscle (as in Plate 3.2.1A). In contrast, distally exerted force is decreased because of such loading conditions.

Myofascial force transmission between antagonistic muscles is (by definition) only possible via extramuscular mechanisms, as such muscles are separated by compartment walls. Stiff connections are made between compartments by neurovascular tracts, but also other extramuscular fascial structures are expected to play a role. The effects and explanation of myofascial interaction between two muscle groups located at opposite sides of an intermuscular septum or interosseal membrane (Plate 3.2.1B) are similar to those described for synergistic muscles (Huijing 2007; Huijing et al. 2007; Meijer et al. 2007, Rijkelijkhuizen et al. 2007). In a series of experiments in rats, we have shown that such mechanisms and effects are active between all muscles of the lower leg (for an overview of results, see Rijkelijkhuizen et al. 2009). So, even antagonistic muscles located at opposite sides of the leg interact (e.g., m. tibialis anterior and triceps surae muscles, see Ch. 5.8 for similar physiological results in mice), and cannot be considered as fully independent entities. It should be realized that this means that part of the force exerted by active sarcomeres within a muscle may be exerted at the tendon of its antagonistic muscle. In fact, the proximal sarcomeres of myofibers are in series not only with more distal sarcomeres of the same myofiber, but via myofascial loading also with distal sarcomeres (and their adjacent endomysia) in the lengthened antagonistic muscle(s). It is evident that the classical concept of antagonistic muscles (as having opposite mechanical effects) and the nomenclature used to describe them is due for a fundamental update... that, however, is beyond the scope of the present chapter.

Muscular relative position also affects muscular force exertion

Experiments (Huijing 2002; Maas et al. 2004), as well as finite element modeling (Maas et al. 2003a,b; Yucesoy et al. 2006), indicate that a muscle kept at constant length and moved through its natural fascial context exerts tendon forces in proximal or distal direction that will vary according to the myofascial loading conditions that are altered with changes in relative position (Fig. 3.2.2).

Relative movement between synergistic muscles occurs due to differences in moment arms at joints crossed, and for bi- or polyarticular muscles due to movement in a joint not crossed by its adjacent monoarticular synergistic muscle. Relative movement of antagonistic muscles is the order of the day, since movement of a joint will have opposite effects on



Fig. 3.2.2 • Schematic example of effects of relative position on myofascial connections and loading. As the muscle of constant length is moved its relative position is changed with respect to other structures (e.g., other muscles or extramuscular structures). The effects for myofascial connections and the direction of loading on the muscle are indicated with the consequences for muscle force exerted at proximal (left) and distal (right) tendons.

muscular length: e.g., joint flexion will lengthen its extensor muscles and shorten its flexor muscles.

Complexity of myofascial loading of muscle

Above, we have provided the simplest examples of myofascial loading to clarify its principles. In reality, within the integrated myofascial system loading of muscles will be very complex.

Multiple and opposite loads on a muscle are considered common (for example, a proximally directed load by the neurovascular tract and a distally directed load by compartmental fascia). The latter is particularly evident for shorter muscles, but also present at higher lengths. If one cuts a tendon (Chapter 5.4), its passive muscle retracts somewhat. If such tenotomized muscle is activated, it will shorten only a little more, indicating a distal myofascial load keeping the muscle at length.

By changing the length and position of muscles, the direction of net loading may change because of (1) rotating a fascial component with respect to muscle or (2) making the mechanical effects of another fascial structure dominant.

If simultaneously exerted proximal and distal myofascial loads on a muscle are equal, the proximo-distal force difference will be zero. Therefore, the presence of such a difference constitutes absolute proof of epimuscular myofascial force transmission, but its absence does not necessarily mean that such force transmission is missing!

Since myofascial loading of a muscle under consideration may originate from all muscles with the body segment, it is clear that a lot needs to be known before the conditions determining the target muscle's force are specified in detail.

In addition, myofascial force transmission between muscles in adjacent segments is likely, because of the stiffness of the neurovascular tract coursing through the tissues of the body segments. There are some indications that inter-segmental myofascial transmission occurs (Vleeming et al. 1995; Huijing et al. 2009), but this needs further confirmation, particularly for living and active muscles (Huijing 2009).

Experiments and modeling performed so far have had proving the feasibility of epimuscular myofascial force transmission and its basic effects and principles as their goal. Therefore, it is clear that we have only scratched at the surface of this intricate and complex system and a lot more scientific and clinical work is needed to reveal the principles of its complexity.

Additional factors to consider

Experimental work discussed above has many limitations that need to be considered in a more generalized view. It is good to realize that the control needed for solid experimental proof precludes getting close to in-vivo conditions. At the same time, if one experiments in vivo in human or animal studies, it is often impossible to attain sufficient control of the conditions.

Joint movement

Joint movement (not included in experiments described above) affects actual stiffness of fascial components; e.g., the length and, particularly, tension within neurovascular tracts are very dependent on joint positions (Huijing 2009). Therefore, a recent study (Maas & Sandercock 2008) extending experiments (to cats) and including actual joint movement constitutes a real contribution to the field. Evidence of epimuscular myofascial force transmission between synergistic muscles of the calf was reported only if soleus muscle was at a length deviating from that imposed by the specific ankle angle. Maas and Sandercock (2008) concluded that epimuscular myofascial force transmission does not occur in physiological conditions in vivo, but may play a role when conditions deviate from normal. There is no doubt of the validity of their finding. However, in its generality, their conclusion about myofascial force transmission occurring exclusively in non-physiological conditions is premature, as evident also from emerging magnetic resonance imaging work in humans (Yaman et al. 2009; Huijing et al. 2011) with similar experiments: changing the knee angle caused local changes of strain, not only in gastrocnemius muscle, but also in synergistic soleus muscle kept at constant length due to a fixed ankle angle. The same was found for the full remainder of antagonistic muscles of the lower leg that also do not cross the knee.

Previously, some other studies indicated evidence of in-vivo epimuscular myofascial force transmission (Yu et al. 2007). One could argue, as was done by Herbert and co-authors (2008), that if only a small percentage of muscle force is transmitted myofascially, we could afford to neglect the whole process. However, having epimuscular myofascial force transmission as a fundamental mechanism of intact tissues completely changes the view of functioning of those tissues, even if the size of the related phenomena may be small in specific conditions.

Levels of muscular activation

One important aspect of the work discussed is that even though different levels of activation have been studied by varying firing rates of muscles (Meijer et al. 2006; 2008), such changes were always imposed uniformly on all muscle studied. In vivo, different muscles or muscle groups are active at varying levels of activation. By stimulating different nerves or their branches (Maas & Huijing 2009) this may be mimicked. Those results do not affect the principles as described above in a major way.

Effects on functioning of the sensory apparatus

As our view of in-series and parallel arrangements of structures is renewed, it is clear that our views on neural sensors need to be adapted as well: many more receptors outside of muscle (e.g., in periosteum, intermuscular septa, compartment) will receive information about muscular conditions. Also intramuscularly, conditions for receptors may be different than thought previously. Classically, muscle spindles are considered as arranged in parallel to myofibers, and Golgi tendon organs as arranged in series with them. However, if a part of the stroma that contains muscle spindles is in series with sarcomeres, this receptor will also operate in series. Preliminary results indicate that this may be the case (Arikan et al. 2009). In any case, in accordance with results on epimuscular myofascial force transmission, receptors in muscle kept at constant muscle-tendon complex length increase their firing rates, as other muscles within the same segment are lengthened.

The most important basic principles of epimuscular myofascial force transmission have been discussed. On the basis of that, the conclusion is warranted that if we do not take such force transmission into account we will never fully understand muscular function. Similarly, it is likely that at least acute effects of manual therapy (Chapter 8.5) will involve some of these mechanisms.

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Myofascial chains

A review of different models

Philipp Richter

Most of the following models are based on personal experiences of the authors, combined with neurological and physiological theories. Even if these models are sometimes very different, they all have one thing in common: they show the locomotor system and the myofascial tissues as being one unit that always functions as a whole. The model of Thomas Myers will not be covered in this review as it will be addressed in the next chapter.

To fulfill its function, a muscular group must have a stable basis. This basis is given by another muscular group, which is again supported by another muscular group, etc. This process leads to the formation of muscle chains.

Kurt Tittel: muscle slings

Dr. Kurt Tittel uses the term "muscle slings" to describe the co-operation of muscle groups to exert coordinated movements. In very instructive illustrations, he explains the muscular chains that are active in sports activities, while also referring to the fact that muscles change or adapt their activity during the different movements. Therefore the images in his book only refer to their momentary state. Dr. Tittel shows the following muscle slings by referring to examples of sports activities.

Extension slings

Extension of arms and legs, and extensions of the torso:

- M. triceps surae
- M. quadriceps, m. tensor fasciae lata and tractus iliotibialis

- M. gluteus maximus, m. adductor magnus
- M. latissimus dorsi
- Mm. erector spinae
- Mm. rhomboidei
- M. trapezius
- M. deltoideus
- Elbow, hand and finger extensors.

Flexion slings

Flexion of arms and legs and flexion of the torso:

- Dorsal extensors of foot and toes
- Hamstring muscles
- M. iliopsoas, m. sartorius, m. tensor fascia latae, m. gluteus minimus, m. adductor longus et brevis
- Abdominal muscles and intercostal muscles
- Mm. scaleni, mm. sternocleidomastoidei
- Elbow, hand and finger flexors.

Muscle slings in static motion patterns

- Rhomboideus-serratus sling: moves the shoulder blade up/backward or down/forward.
- Levator-trapezius sling: moves the shoulder blade up and down.
- Trapezius-pectoralis sling: inward or external rotation of the shoulder blade.
- Trapezius-serratus sling: forward and backward movement of the shoulder blade.

Muscle slings in sidebending and rotation of the trunk

Connecting the spinal column ventrally with the lower extremities:

- Mm. rhomboidei
- M. serratus anterior
- M. obliquus abdominis externus
- Mm. adductorii on the other side
- Caput brevis m. biceps femoris
- Fibula + mm. peronei.

Connecting the shoulder ventrally with the foot:

- M. pectoralis major
- M. obliquus abdominis externus
- M. obliquus abdominis internus on the opposite side
- M. tensor fascia lata
- M. tibialis anterior.

Dorsal chain, connecting the arm with the leg on the opposite side:

- M. latissimus dorsi
- Fascia thoracolumbalis
- M. gluteus maximus
- Tensor fascia lata
- M. tibialis anterior.

These diagonal muscle slings are of great importance for movement and a wide range of sports such as football, tennis, spear throwing, shot-put, etc.; see Fig. 3.3.1.

Herman Kabat: proprioceptive neuromuscular facilitation (PNF)

Together with Margaret Knott and Dorothy Voss, Dr. Herman Kabat developed a treatment method for muscular paralysis of patients suffering from poliomyelitis. The particularity of this method is the idea of integrating paretic muscles into a muscle chain. The patient is supposed to make special motion patterns that include weak or paralyzed muscles.

The following motion patterns were used by Dr. Kabat:

- Trunk:
 - Flexion + lateral flexion+ rotation
 - \circ Extension + lateral flexion + rotation.
- Neck:
 - Flexion left (right) Extension right (left)
 - Flexion sidebending left rotation left and vice versa



Fig. 3.3.1 • Muscle slings of Kurt Tittel. This model describes muscle chains that are active in sports activities, while also referring to the fact that muscles adapt their activity during different movements. Kurt Tittel 2003: Beschreibende und funktionelle Anatomie des Menschen. 14th Edition. Elsevier Urban & Fischer, Munich

- Extension sidebending right rotation right and vice versa.
- Shoulder blade and pelvis:
 - Anterior elevation or depression
 - Posterior elevation or depression.
- Upper extremity:
 - \circ Flexion abduction external rotation
 - \circ Extension adduction internal rotation
 - Flexion adduction external rotation
 - \circ Extension abduction internal rotation.
- Lower extremity:
 - Extension abduction internal rotation
 - \circ Flexion abduction internal rotation
 - \circ Flexion adduction external rotation
 - Extension adduction external rotation.

Method

The therapist brings the segment to be treated into a starting position, in order to stretch all muscles, agonists, and synergists involved in the motion pattern. The patient is asked to perform the motion pattern through verbal instructions and tactile stimuli. These movements are performed until the muscle chain that needs to be treated is in an optimally shortened position, while its antagonists are stretched.

The interesting points of Kabat's model are the integration of the weak muscles into stronger muscle chains, and the performance of motion patterns while respecting neurological principles. The focus is on the activation of muscle chains.

Godelieve Struyf-Denys

Miss Struyf-Denys is a Belgian physiotherapist with osteopathic training, who presents a muscle chain model which includes three aspects:

- The stretching of shortened or hypertonic muscles dominates the treatment.
- Psychic and emotional disorders are, along with traumas, a cause for muscular dysbalance and static disorders.
- Every human has an innate pattern.

She was led by the osteopathic holistic idea and the ideas of Mézières a French physiotherapist, along with the theories of Piret and Béziers. According to Mézières, the reasons for postural disorders are partly dysfunctions and incoordination of the muscular system, but mainly shortening and hypertonicity in the area of erector spinae muscles, linked to reflex hypotonia of the abdominal muscles. According to Piret and Bezière, all movements are spiral-shaped; they are conditioned by the form of the joint surface, and the diagonal alignment of the pluriarticular muscles. Furthermore, Piret and Bezière are convinced that posture patterns have a mental, emotional cause. Struyf-Denys also knows the treatment method of Kabat.

Struyf-Denys describes five muscle chains on each half of the body, that normally function in a coordinated fashion. As a result of traumatic disorders or emotional stress, one of these chains will dominate the others, and this leads to malposture and deformed motion patterns.

The five muscle chains of Struyf-Denys are subdivided into three fundamental or vertical chains and two complementary or horizontal chains. The fundamental chains include the trunk muscles and proceed through a so-called secondary section into the extremities. The complementary chains are composed of muscles of the extremities. The fundamental chains are tied to the complementary muscle chains by the muscles of the shoulder girdle or the pelvic girdle, respectively.

According to Struyf-Denys, these five muscle chains depend on five psychological constitutions. The three fundamental chains are each allocated to a section of the cranium. The shape of this cranium section indicates the dominance of this chain; it also proves the existence of a specific psychological disposition.

The vertical or fundamental chain

The anteromedian chain

Primary section

- Hyoid muscle
- Sternal section of m. sternocleidomastoideus
- M. scalenus anterior
- M. subclavius
- Medial mm. intercostali
- M. transversus thoracis
- Pars costalis u. pars sternalis of m. pectoralis major
- M. rectus abdominis
- Pelvic floor muscles.

Secondary section: Connects the torso with the extremities

- Upper extremity:
 - $\,\circ\,$ Anterior section of the m. deltoideus
 - \circ M. brachialis
 - \circ M. supinator
 - $\,\circ\,$ M. abductor pollicis.
- Lower extremity:
 - M. pyramidalis abdominis
 - Mm. adductorii
 - $\,\circ\,$ Median section of the m. gastrocnemius
 - $\circ\,$ M. adductor hallucis.

The posteromedian chain

Primary section

- Mm. erector trunci
- Long neck extensors.

Secondary section

- Upper extremity:
 - M. latissimus dorsi
 - \circ Pars ascendens of the m. trapezius
 - $\circ~$ M. infraspinatus
 - \circ M. teres minor
 - $\,\circ\,$ Dorsal section of the m. deltoideus
 - $\,\circ\,$ Caput longum of the m. triceps brachii
 - Finger flexors
 - \circ Pronators.
- Lower extremity:
 - M. semitendinosus
 - M. semimembranosus
 - M. soleus
 - \circ Toe flexors.

The posteroanterior-anteroposterior chain

Primary section

- Deep paravertebral muscles
- Intercostal muscles
- Mm. splenius capitis and colli
- Mm. scalenus
- M. iliopsoas.

Secondary section

- Upper extremity
 - $\circ\,$ M. pectoralis minor
 - M. coracobrachialis

- Caput brevis m. biceps brachii
- $\,\circ\,$ Finger extensors.
- Lower extremity
 - M. vastus medialis
 - M. rectus femoris
 - \circ Toe extensors.

The complementary muscle chains

The posterolateral chain

- Upper extremity:
 - Pars horizontalis and descendens of the m. trapezius
 - M. supraspinatus
 - Middle section of the m. deltoideus
 - Lateral section of the m. triceps brachii
 - M. anconeus
 - M. extensor carpi ulnaris
 - M. flexor carpi ulnaris
 - M. abductor digiti minimi.
- Lower extremity:
 - M. gluteus medius
 - M. biceps femoris
 - O M. vastus externus
 - Mm. peronei
 - M. gastrocnemius lateralis
 - M. plantaris
 - Lateral section of m. abductor hallucis longus.

The anterolateral chain

- Upper extremity:
 - Pars clavicularis of the m. sternocleidomastoideus
 - M. pectoralis minor
 - 0 M. deltoideus
 - M. teres major
 - 0 M. latissimus dorsi
 - M. subscapularis
 - Caput longus m. biceps brachii
 - Supinator
 - M. brachioradialis
 - M. extensor carpi radialis longus et brevis
 - M. palmaris longus
 - Thenar muscles
 - $\,\circ\,$ Mm. lumbricales and mm. interossei palmares
 - M. flexor carpi radialis.

- Lower extremity:
 - O M. gluteus medius
 - O M. tensor fascia lata
 - M. tibialis anterior
 - O M. tibialis posterior
 - Mm. interossei plantaris
 - Mm. lumbricales.

Leopold Busquet

Leopold Busquet is a French osteopath. He produced a series of six books on the topic of muscle chains. In the first four books, he describes the muscle chains of the trunk and of the extremities. In his fifth book, he describes how the cranium adapts to the muscle chains and how the muscle chains react to cranial patterns. The sixth book deals with the viscero parietal connections. The author describes how the musculoskeletal system adapts to visceral disorders. There are five chains:

1. Static posterior chain which is composed of inert tissue, thus no real muscle chain per se.

It consists of the dura mater cranialis and spinalis, the ligaments of the spine, as well as the fasciae of the gluteals and the M. piriformis, the tractus iliotibialis and the fibula, with the interosseal membrane. This chain does not contain any muscular elements; therefore it is a passive chain.

2. Flexion chain (straight anterior chain)

This is the muscle chain that bends the head, the neck, and the torso, flexes the arms and turns them inwards. The flexion chain of the lower extremity consists of the foot extensors and the toe, knee, and hip flexors.

3. Extension chain (straight posterior chain)

The extension chain is about the antagonists of the flexors. Head and neck are stretched, the spinal column extended, the upper extremities stretched and exert an external rotation. The lower extremity shows a plantar flexion of the ankle and an extension of the toes, the knee, and the hips.

4. Diagonal posterior chain

The diagonal chains are torsion chains. Here, the shoulder approaches the opposite iliac bone. Busquet names the chains after the involved iliac bone. These chains predominantly include muscles with diagonally attributed fasciae (e.g., obliquus abdominis). The muscles of the extremities also belong to these chains. These diagonal chains are called "opening chains", because they cause an external rotation of the extremities. In the activation of the right diagonal posterior chain, the right ilium approaches the left shoulder, the left shoulder moves backwards and down, while the right ilium moves backwards and up. The left arm and the right leg perform an extension – abduction.

5. Diagonal anterior chain

This muscle chain consists of the ventral muscles with diagonally oriented muscle fibers. The trunk makes a forward torsion, as if it would roll in. In the case of a dominant right anterior chain the right ilium is anterior internally and the left shoulder is anterior inferiorly, with a flexion-adduction of the left arm and the right leg.

The composition of the five chains

- 1. Static posterior chain
 - Falx cerebri, falx cerebella and dura mater spinalis
 - Ligamentum nuchae and the ligaments of the vertebral arch
 - Fasciae of the gluteal muscles and the hip rotators
 - Tractus iliotibialis
 - Fibula and interosseus membrane
 - Plantar aponeurosis.

2. Flexion chain

- Trunk:
 - M. splenius capitis, m. splenius colli
 - M. sternocleidomastoideus
 - Mm. scaleni
 - Intercostal musculature
 - M. pectoralis major, m. pectoralis minor
 - Pars descendens m. trapezius, m. rhomboidei, m. teres major
 - M. rectus abdominis and pelvic floor musculature.
- Upper extremity:
 - $\,\circ\,$ Anterior section of the m. deltoideus
 - Elbow, hand and finger flexors.
- Lower extremity:
 - Hip and knee flexors
 - Foot dorsal extensors
 - Toe flexors.
- 3. Extension chain
 - Trunk:
 - Deep paravertebral musculature
 - M. quadratus lumborum
 - M. serratus posterior superior and inferior
 - M. trapezius

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- M. pectoralis minor
- O M. serratus anterior
- M. splenius colli
- Mm. scaleni
- $\,\circ\,$ M. latissimus dorsi and m. teres major
- M. pectoralis major.
- Upper extremity
 - $\,\circ\,$ Dorsal section of the m. deltoideus
 - Elbow, hand and finger extensors.
- Lower extremity
 - Hip and knee extensors
 - Plantar flexors
 - Toe extensors.
- 4. Diagonal posterior chain (e.g., right)

The right ilium moves backwards and up, and the left shoulder moves backwards and down, while the left arm and the right leg make an extension with abduction and external rotation.

- Trunk:
 - Iliolumbal fibers of the right m. quadratus lumborum and the costotransversal fibers of the left m. quadratus lumborum
 - Left mm. intercostali interni and the right mm. intercostali externi
 - Left m. latissimus dorsi
 - Pars ascendens of the left m. trapezius
 - Left m. teres major and m. pectoralis major
 - Left m. splenius colli and capitis
 - Left mm. scaleni.
- Lower extremity:
 - Right gluteals
 - Right m. sartorius
 - Right TFL
 - M. vastus lateralis
 - M. tibialis anterior
 - M. extensor hallucis longus.
- Upper extremity
 - Extensors and abductors of the arm, forearm, hand and fingers.
- 5. Diagonal anterior chain (e.g., left)

The left iliac bone moves forward and inward and the right shoulder moves forward and down; the left arm and the right leg perform an adduction–internal rotation.

- Trunk:
 - Left m. obliquus internus and right m. obliquus externus

- Left mm. intercostali interni and right mm. intercostali externi
- Left mm. pectoralis major and minor
- Right m. serratus anterior
- Right mm. rhomboidei
- Right mm. scalene
- Left mm. splenius colli and capitis
- Pars ascendens of the right m. trapezius and pars descendens of the left m. trapezius.
- Lower extremity: mm. adductorii right, m. vastus medialis, m. semitendinosus, mm. peronei, m. abductor halluces.
- Upper extremity: flexors and adductors.

Conclusion

Busquet describes a static fascial chain and four dynamic muscle chains. He refers to visceral and cranial causes and correlations in cases of muscular dysbalance. Cranial dysfunctions can be the cause of dominant muscle chains and can have an impact on the musculoskeletal system, as well as on organic disorders. Busquet describes two scenarios for visceral disorders:

- An organ needs space (e.g., flatulence): muscles are activated in a way to give the organs enough space to function properly.
- An organ needs support or painful structures must be relieved: the muscles will influence the locomotor system in a way that tensions can be reduced or an organ can find support.

Paul Chauffour: "The mechanical link in osteopathy"

In his book *Le lien mécanique en ostéopathie* (the mechanical link in osteopathy), Paul Chauffour very clearly describes the fasciae of the human body. He pays great attention to the insertions of the fascial membranes because of their importance for the conduct of different body parts in movement. He describes the behavior of the locomotor system in the four main movement patterns of the human body.

- Flexion = curling in, closing.
- Extension = stretching, opening.
- Anterior torsion (to the right or to the left).
- Posterior torsion (to the right or to the left).

Chauffour's motion patterns have many resemblances to Busquet's. Interestingly, he includes the biomechanics of the cranium and describes the cranial adaptations to the myofascial traits in the four movement components. He shows anatomical and functional causes for dysfunctions in the following "weak points".

Flexion pattern

C1: dens axis prevents flexion.

C2: is the first cervical vertebra with normal facets, therefore C2 is more weak than C1. C7: less stabilized than the other thoracic vertebrae, therefore more fragile in flexion. T4: weak point of the spine; the lowest insertion of the long neck muscles is at T4–T5 and the different parts of the m. trapezius pull on T4 when contracting.

T6: below T6, the vertebrae are stabilized by the fascia thoracolumbalis, whereas T6 is not stabilized. T12: most cranial insertion of the m. iliopsoas (flexor), thus T12 is a breakpoint between the forces acting from above and below.

L1 and L2: the crura of the diaphragm exerts a pull on L1 and L2.

Extension pattern

The complete thoracic spine, especially T7 and T11, is compressed by the muscles trapezius and latissimus dorsi.

L2 is under the trait of the diaphragm and the m. iliopsoas, which both resist the extension, when shortened or tense.

Forward torsion

C6: C6 is less stabilized than C7 and therefore weaker.

C7: this is also true for C7 in comparison to T1. T4: the central tendon and the long neck muscles associate the upper thoracic spine to T4 with the cervical spine.

T6: the aponeurosis of the m. latissimus dorsi goes to T7.

T10: the 11th and 12th ribs are not connected firmly to the thorax, therefore the segment T10–T11 is weaker in torsion.

T11: T11–T12 is the center of the spinal torsion.

L2: L1 and L2 are stabilized by the crura of the diaphragm; L3 is not.

Posterior torsion

C1: is stressed, because the sidebending of C1 and C2 are opposed.

C6: C7 is more fixed than C6, therefore C6 is more fragile.

T6: the fascia of the m. latissimus dorsi stabilizes TH7, but it does not stabilize T6.

T10: T11 is more mobile than T10.

T12: the fascia of the m. trapezius goes up to T12, therefore T12 goes along with the thorax.

The Richter–Hebgen model

Muscles enable humans to function. They adapt the posture to the needs of the body and try to avoid tensions and compressions in order to allow the body to function in as painless a way as possible. A pathologically dominant muscle chain imposes itself on the locomotor system. Posture and motion patterns are influenced by dominant muscle chains. Dysbalances of muscular tension are the reason for faulty postures like scoliosis, kypholordosis, etc.

The inspection, palpation, and movement tests allow the therapist to find the dominant muscle chain. The normalization of the muscle balance diminishes tensions and improves blood and lymph flow, which in turn makes the healing process possible. This model is based on the following premises:

- **1.** Every half of the body has a flexion chain and an extension chain.
- 2. There are two movement and position patterns:
 - \circ flexion + abduction + external rotation
 - \circ extension + adduction + internal rotation
- **3.** The body is subdivided into several movement units. These are explained functionally and neurologically.
- **4.** Flexion and extension alternate from one movement unit to the next and form "muscle slings" or lemniscates. This is also true for the upper extremities.
- **5.** Kyphosis and lordosis are the consequences of an abnormal dominance of both flexion chains or extension chains (left and right). Scoliosis is the result of the dominance of a flexion or extension chain and the reflex inhibition of its antagonists (antagonist inhibition and crossed stretch reflex).

- **6.** The cause of faulty postures is muscular dysbalance as a result of neurological malfunctions. Static disorders, organic malfunctions, physical and emotional traumas lead to a medullary sensitization and the resulting muscular dysbalances.
- **7.** The myofascial structures always react in chains and therefore as a whole. The dominant pattern continues in the cranial and visceral area and it can cause organic or cranial dysfunctions.

The three posture patterns:

1. Dominance of the flexion chain

- Increase of the torsion of the spinal column: kypholordosis.
- Synchondrosis sphenobasilaris in extension with internal rotation of the peripheral cranial bones.
- Thorax in expiration.
- Lower extremities in extension, adduction and internal rotation.
- Flat feet.
- Shoulder blade in abduction.
- Protraction of the shoulders.
- Internal rotation and adduction of the arms.
- Relative extension of elbow, hand, or feet.

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- 2. Dominance of the extension chain
 - Stretching the spinal column.
 - Synchondrosis sphenobasilaris in flexion with external rotation of the peripheral cranial bones.
 - Thorax in inspiration.
 - Lower extremity in flexion, abduction, external rotation.
 - Hollow feet.
 - Shoulder blades in adduction.
 - Retraction of the shoulders.
 - External rotation and abduction of the arms.
 - Relative flexion of elbow, hand, and fingers.
- 3. Torsion pattern (the most frequent pattern)

The flexion chain dominates one half of the body, and the extension chain the other. This leads to torsion of the spinal column (scoliosis), torsion of the pelvis with one leg in flexion–external rotation–abduction and the other in extension–internal rotation–adduction. The shoulder girdle and the upper extremities are likewise forced in opposite directions. In the cranium, torsion or sidebending–rotation is found.

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Anatomy Trains and force transmission

Thomas Myers

Introduction – extracellular matrix as metamembrane

Despite humans' biblical and Aristotelian penchant for naming parts, anatomists, however many of the bottomless barrel of terms they know, must admit that the human being is grown organically from a single egg, not assembled like a car from parts. The familiar industrial images that pervade our thinking about the body – the heart is a pump, the lungs are bellows, the brain is a computer, etc. – subtly promote the idea of isolated action and separated systems. We know in our heart of hearts, however, and should remember in our everyday clinical thinking, that our body always does and always has worked together in an unbroken concert from conception on out.

Starting at about fourteen days in embryological development, as cells proliferate and specialize, they create an extracellular matrix (ECM) between them (Moore & Persaud 1999). This delicate web-like intercellular gel provides the immediate environment of most cells, mixing varying proportions of fiber, gluey proteoaminoglycans, and water with diverse and circulating metabolites, cytokines, and mineral salts (Williams 1995). It is this ECM that provides most of the "tissue" bulk in many connective tissues, as the cells alter the ECM to form bone, cartilage, ligaments, aponeuroses, and the rest (Snyder 1975). The ECM grows along with the cells themselves and together they form a single organism connected, joined, and held together by the ECM.

The ECM is intimately connected to cell membranes and through them to the cytoskeleton via hundreds or thousands of binding integrins on

the cell surface (Ingber 1998). Forces from outside the cell are transmitted via these adhesive connections to the inner workings of the cell (Ingber 2006a). Thus, we can now understand that each cell, as well as "tasting" its chemical milieu, is "feeling" and responding to its mechanical environment – leading to the relatively new field of "mechanobiology" (Ingber 2006b). Forces also move in the other direction – from the cell to the ECM – in the case of muscular or (myo) fibroblast contraction that gets conveyed through the membrane to the surrounding ECM (Tomasek et al. 2002).

Connective tissue cells are particularly adept at promulgating and maintaining this system of the ECM, which operates under the following design constraints.

To allow trillions of cells to stand up and walk around in an organismic fashion, the ECM must:

- Invest every tissue without exception muscle, nerve, epithelia, and of course all the connective tissues themselves, from blood to bone.
- Be permeable enough to allow all local cells to be in the flow of metabolism yet tough enough to protect those cells from endogenous and exogenous forces.
- Vary widely, both across the body from tough bone and resilient cartilage to the lymphatic network of the breast and the aqueous humor of the eye.
- Be able to remodel itself over time to meet altered biomechanical conditions in growth, performance, healing, and repair (or pathologically in disease or degeneration).
- Transmit forces from one tissue to another with maximum precision, and maximal adaptability to sudden changes in load, while sustaining minimal cellular tissue damage.

The ECM acts as the "metamembrane" for the organism, creating an organismic boundary, restraining and directing movement, protecting delicate tissues, and maintaining the recognizable shape most of us maintain from day to day (Juhan 1987; Varela & Frenk 1987).

Dividing the indivisible

Although the ECM is manifestly one single whole, it is convenient to divide it into three sections:

- The tissues of the dorsal cavity the numerous glia within the nervous system itself, the meninges around the brain and spinal cord, and the perineural extensions out into the rest of the body (Upledger & Vredevoogd 1983).
- The tissues of the ventral cavity the strings, sheets, and sacs that separate the organs and hold them to the body wall, including the mesentery, mediastinum, and peritoneum (Barral & Mercier 1988).
- The tissues of the locomotor system the bones, joints, capsules, ligaments, fasciae, aponeuroses, and all the tissues surrounding and investing the skeletal muscles – endomysium, perimysium, epimysium, and their tendinous extensions (Chaitow 1980).

This last section, which due to the necessity for transmission of strong forces accounts for a significant percentage of the total protein of the body, can again be divided functionally into:

- An "outer" myofascial layer consisting of 600 or so muscles imbedded in the fascia necessary to hold them together, organize their movement, and deliver their force to the bones and other tissues.
- An "inner" layer of joint capsules, ligaments, and periostea that surrounds the skeleton and organizes its growth, protects it from dismemberment, and limits movement, thus providing efficient force transmission from one joint to the next (Myers 2009).

All of the above divisions are imprecise, due to the integrated nature of the ECM; it is sometimes impossible to tell where one section stops and the other begins, and functionally they are all in league with each other. This last division between the outer and inner "bags" within the musculoskeletal system

is particularly porous, since these structures have been shown to work in series more often than in parallel (Van der Wal 2009).

Isolating a muscle

After this preamble to holism, the remainder of this chapter will focus on some patterns within this "outer bag" of myofasciae. The traditional view of anatomy that has broadened our knowledge considerably has been gained by a reductionistic parsing of the body, largely with a scalpel. The result is "the muscle" as the predominant label for making named units from the unified soft tissue of this layer. Once a muscle is dissected from its neurovascular fascia, from its overlying areolar layer, and from its neighbors right and left, and the ligaments below, the muscle is analyzed solely in terms of what would happen if the two end points north and south (the so-called proximal and distal attachments) were pulled together in a concentric, isometric, or (with an opposing outside force) eccentric contraction (Williams 1995; Biel 2005; Muscolino 2010).

This isolationist muscle analysis separates <u>one</u> function out of the many and raises it to the level of <u>the</u> function. Most analyses of posture and movement proceed from the idea that individual muscles move bones while individual ligaments stabilize them (Kendall & McCreary 1983). Despite the couple of centuries of kinesiology that has taken this model to its limits, one may question whether the nervous system "thinks" in terms of individual muscles, or whether the muscle, a convenient division for the dissector, is even a distinct physiological unit. Neuromotor units within muscles may be a more useful division (Van der Wal 2009), or larger patterns – our focus for the rest of this chapter – may also tell us something useful about human movement and stability functioning.

More recent thinking, much of which is described in the previous chapter, has focused on functional wholes and interconnected patterns within this outer layer, rather than looking for the muscle or particular fascial structure as the culprit for systemic failure such as injury (or more pointedly, lack of injury repair). The *Anatomy Trains Myofascial Meridians* is yet another of these maps, owing much to the work that has come before, from Raymond Dart through Tittel, Mezières, Hoepke, and others, yet at the same time this system has some unique features (Hoepke 1936; Dart 1950).

The Anatomy Trains

The Anatomy Trains, then, is an attempt to describe common pathways of functional force transmission through the outer layer of myofascia (Plate 3.4.1; Myers 2009). Though they have some common ground with the meridians of acupuncture, they are based entirely on occidental fascial anatomy. To create a myofascial meridian, one must:

- Follow the grain of the (myo)fascial fabric from structure to structure.
- Go more or less in a straight line (pulls cannot go around corners except via "pulleys").
- Not pass through intervening walls of fascia that would block the force transmission.

Parsing the body in this way reveals 12 sets of connections through this outer bag, which are more fully described elsewhere (Myers 2009; www. AnatomyTrains.com). Generally, there are distinct and coherent lines of dissectable myofascial connection along the front of the body, along the back, along the sides, around the trunk and under the arches, along the arms, connecting contralateral girdles, and through the core of the legs and trunk.

Here are the fascial and myofascial soft-tissue structures involved in each line. The attachments of the individual muscles within the lines are known as "stations" within the Anatomy Trains schema, to denote that even though the line is connected to the "inner bag" of periosteum and ligaments at these junctures, the force transmission carries on via the fascia beyond the muscle attachment. The degree, timing, and precise mechanism of such force transmission has yet to be measured and confirmed, but early evidence suggests that it is worth our consideration to think that muscles are connected to muscles via the fascia (Huijing 2009). This idea might usefully be added to the more narrow convention: muscles attach to bones. Of course, no muscle attaches to any bone anywhere in the body; it is always via the intervening connective tissue structures (Van der Wal 2009).

- Superficial Front Line: Toe extensors, anterior crural compartment, quadriceps, rectus abdominis and abdominal fasciae, sternalis and sternal fascia, sternocleidomastoid.
- Superficial Back Line: Short toe flexors and plantar aponeurosis, triceps surae, hamstrings, sacrotuberous ligament, sacrolumbar fascia, erector spinae, epicranial fascia.

- Lateral Line: Fibularis muscles, lateral crural compartment, iliotibial tract, hip abductors, lateral abdominal obliques, internal and external intercostals, sternocleidomastoid, and splenii.
- Spiral Line: Splenii, (contralateral) rhomboids, serratus anterior, external oblique, (contralateral) internal oblique, tensor fasciae latae, anterior iliotibial tract, tibialis anterior, fibularis longus, biceps femoris, sacrotuberous ligament, erector spinae.
- Superficial Back Arm Line: Trapezius, deltoid, lateral intermuscular septum, extensor group.
- Deep Back Arm Line: Rhomboids, levator scapulae, rotator cuff, triceps, fascia along ulna, ulnar collateral ligaments, hypothenar muscles.
- Superficial Front Arm Line: Pectoralis major, latissimus dorsi, medial intermuscular septum, flexor group, carpal tunnel.
- Deep Front Arm Line: Pectoralis minor, clavipectoral fascia, biceps, radial fascia, radial collateral ligaments, thenar muscles.
- Front Functional Line: Pectoralis major (lower edge), semilunar line, pyramidalis, anterior adductors (longus, brevis, and pectineus).
- Back Functional Line: Latissimus, lumbosacral fascia, gluteus maximus, vastus lateralis.
- Ipsilateral Functional Line: Latissimus (outer edge), external abdominal oblique, sartorius.
- Deep Front Line: Tibialis posterior, long toe flexors, deep posterior compartment, popliteus, posterior knee capsule, adductor group, pelvic floor, anterior longitudinal ligament, psoas, iliacus, quadratus lumborum, diaphragm, mediastinum, longus muscles, hyoid complex, floor of mouth, jaw muscles.

It is important to note that, at this point, Anatomy Trains is only a scheme, a map. It is supported by clinical observation, common sense, and some initial dissection work, but the degree of force transmission across these lines has yet to be quantified, let alone reproduced at a scientifically verifiable level. It is also important to note that Anatomy Trains is not a treatment method, but a way of seeing that has been shown to be supportive of a number of approaches in physiotherapy and rehabilitation, personal and performance training, and manual therapies of all types.

Each myofascial meridian can be seen to have a function beyond the function of each individual muscle within it, and some of these meridians lend themselves to a "meaning" within human experience. These subjective but compelling elements can be observed in the clinic, suggesting global treatment plans that support the restoration that the therapist applies to locally strained or damaged tissues.

Running through the system briefly in this way, we can see that the Superficial Front Line, which runs up the body from the top of the toes to the mastoid process, is often shortened in patterns of chronic fear (as in the startle response). Though not scientifically verifiable, this pattern has been observed so often as to make it a truism: strong or chronic emotions of fear often leave a discernible mark in the postural and movement patterning of the body that manifests as muscular (and then fascial) shortening of the structures of the Superficial Front Line (see Fig. 3.4.1).

If this pattern is observed in a client, then opening, lengthening, and lifting the tissues of the Superficial Front Line will tend to support the longevity and efficacy of whatever treatment mode is chosen. Expressed in the negative, failing to include such global considerations in local treatment will leave the tendency for the patient to return to the patterns that led to the segmental failure in the first place.



Fig. 3.4.1 • A subject just before and just after an unexpectedly loud noise. The "startle response" (a common cross-species as well as cross-cultural event) can be seen as a reflexive attempt to protect the vulnerable parts of the body through a strong contraction of the Superficial Front Line. Courtesy of Frank Jones.

The Superficial Back Line (see Plate 3.4.2) must shorten and strengthen to bring us from the primary fetal curve into adult standing, which involves a balance among primary and secondary curves. Disturbance in this developmental process can result in imbalances among the primary and secondary curves. These imbalances in turn tend to guide the body toward chronically held muscular compensation. Chronically held muscle leads over time to fascial contracture, but the hypothesis based on observation is that this force can be transmitted (or more accurately "distributed" – see the section below on Tensegrity) to other spots in the body.

These meridians of myofascial fibers in continuous connection are offered as common (but not exclusive) pathways of myofascial transmission from one segment to another. The result is a common and recognizable pattern of posture that is held neurologically, muscularly, and (ultimately) fascially. It is the aim of many fascially based therapies to free these patterns so that the muscles and habit patterns have a chance to be changed for the better. In the absence of freeing these fascial contractures, techniques designed to change muscle tone or neurological habit will be fighting an "uphill battle".

To apply this logic to the client illustrated (see Fig. 3.4.2), the neck pain suffered by this preteen is directly related to the position of her knees. In bringing the knees into hyperextension, thus turning a "secondary" curve into a primary, she forces her body to compensate in the other two secondary curves, which could have resulted in low back pain or (as in this case) neck pain.

(This pattern may have been established in reverse order – we do not know simply by looking at a photograph. The order of adoption of a pattern is a matter of interest, but not of necessity: it is enough that the knee position and neck position are "related" and must be dealt with as a whole, regardless of the order in which they are acquired.)

Whatever locally has broken down or strained in her neck, the best and most lasting work will supplement whatever local treatment was applied with "fascial re-education" (many methods could be applied) of her chronic plantarflexion at the ankle, and hyperextension at the knee and lumbar spine. The post-treatment picture on the right shows how such patterns can (and need to be) considered as a whole. Notice how the Superficial Back Line presents itself as a series of balanced curves in the post-treatment picture. This tends to reinforce the new pattern and promote longevity in results.



Fig. 3.4.2 • Though problems always manifest locally, this girl pre-treatment (left) clearly has a global problem with balance in the Superficial Back Line, which is more coherent and balanced post-treatment (right), which will support the durability of the local results. Courtesy of Robert Toporek.

Tensegrity

The idea of tensegrity is well-explored elsewhere in this volume. Whether a human being's locomotor structure can accurately be described as a formal tensegrity as it is defined mathematically is open to question, but the fact that humans are "tensiondependent structures" is not. A bunch of human

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bones cannot be stacked on each other to recreate the skeleton, and even with the ligaments in place, our structure is hardly self-sufficient or sturdy. Only when these outer layers of "myofascialature" have been strung around the skeleton does the creature stand, adjust, and stabilize in a functional way (see Plate 3.4.3).

It is worth considering that these myofascial meridians act as the global, geodesic tension complexes that simultaneously stabilize and allow adjustments within the skeletal frame. In other words, the body is not (unless injured or misused) the "strain focusing machine" described in biomechanical texts, but is rather a "strain distribution machine", and the myofascial meridians provide common (again, not exclusive) pathways for the body's tensegrity to:

- Add pre-stress for stiffening via muscular or myofibroblast contraction.
- Relax pre-stress for adjustability via the muscles, myofibroblasts, or treatment.
- Relieve strain in one area of the body by exporting some of it to other parts up or down the "line".

Conclusion

Anatomy Trains Myofascial Meridians is an "argument from design" rather than an established scientific fact. This author understands that further research will modify the specifics of the lines, or expand the scope, or replace it with a better map. No map matches the territory, and new imaging methods will produce new maps.

That said, increasing clinical anecdote supports what can be gained in terms of efficacy and duration of results by including these global considerations and "whole fascial net" connections in assessment and treatment.

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Biotensegrity

The mechanics of fascia

Stephen M Levin Danièle-Claude Martin

Introduction

Fascia is the fabric of the body; not the vestments covering the corpus, but the warp and weft of the material. The other tissues, muscle and bone, liver and lung, gut and urinary, brain and endocrine, are embroidered into the fascial fabric. Remove all other tissues from their fascial bed and the structure and form of the corpus remains, ghostlike, but clearly defined. The fascial system is a continuum, a structure that evolved hierarchically from the one cell embryo to the organism, and it is constantly adapting to new stresses to meet the structural demands of the organism (Guimberteau et al. 2007). Fascia without stiffeners would be as limp as a rag doll; remove the hydroxyapatite crystals from bone, and the form of bones remains, but soft, as if the starch has been removed from a stiff shirt. Wolff recognized that bone is stiffened in response to compression stress and what must happen is that the support structure of the body, the fascia with its enmeshed bony stiffeners, evolves in accordance to physical laws (Wolff & Wessinghage 1892).

Fascia is a tension network, with all the collagen inherently stressed, the so-called "pre-stress" of biologic tissues. Where does the compression arise? It is easy to see in an archer's bow. The bowstring pulls the limbs of the bow towards the center belly of the bow, compressing it, and bending the bow into its characteristic shape. Now imagine the "bow" being compressed toward its belly by multiple bowstrings that encircle the bow and are all pulled at once. If the forces were balanced, the bow would not bend, but merely compress. Tension elements at each end that compress toward the center can balance to create a pure compression force, and in a tensioned fascial network bone will be laid down, according to Wolff's law.

The origins of biotensegrity

For this to happen, there must be some evolutionary structural process that is governed by the rules of physics and influenced by the genome. In 1981, a structural model was proposed that incorporated the physical laws related to triangulated (and therefore inherently stable) structural forms, "closest packing", and foams, and the "tensegrity" structures as conceived by Kenneth Snelson (Snelson 2009) and Buckminster Fuller (Fuller & Applewhite 1975) into a biologic model that would appropriately model organisms from viruses to vertebrates, their systems and subsystems: biotensegrity (Levin 1981). Biotensegrity reverses the centuries-old concept that the skeleton is the frame upon which the soft tissue is draped, and replaces it with an integrated fascial fabric with "floating" compression elements (bones in vertebrates), enmeshed within the interstices of the tensioned elements.

For a structure to be stable with flexible joints, it must be triangulated, as only triangles are stable with flexible joints. Biologic structures, their elements joined by surface tension, and flexible soft tissues, must be triangulated structures for them to exist. If not triangulated, it would require stiff joints, or constant, unobtainable, muscle forces to keep from collapsing. Of the three fully triangulated structures,

Fascial Force Transmission

the tetrahedron, octahedron and icosahedron, the icosahedron is the most suitable for biologic modeling. It has the largest volume for surface area, is omnidirectional, has the closest packing capabilities, and endo- and exoskeletal configurations, where the compression elements are either in its outer shell, or incorporated into the innards of the structure (Fig. 3.5.1). The internally vectored icosahedron is a tensegrity structure, simply defined as "floating compression" elements enmeshed in a continuous tension network. The compression elements are isolated from one another and the load is carried through the network, and not a compression-loaded "column of blocks", governed by gravity-oriented levers, as is the norm in most familiar structures. The tensegrity icosahedron can be linked in an infinite array, hierarchically and as fractals (Mandelbrot 1982; see Fig. 3.5.2). It is a low-energy structure, using minimal materials to enclose space and give maximum strength. Because of triangulation, it has flexible joints but is stable and adaptable. Its mechanics are nonlinear, which is consistent with biologic materials and structures. Columns depend on gravity to hold them together; without gravity, columns and structures that depend on columns for support would fall apart. Tensegrities are self-contained structures and do not rely on gravity as a cohesive force. Table 3.5.1 compares biologic structure properties with the properties of standard, lever mechanics and tensegrity icosahedral mechanics.

It is obvious that lever systems, the standard for over three centuries, do not match the qualities needed for biologic modeling, and tensegrity icosahedral systems are a perfect match.



Fig. 3.5.1 • "Exoskeletal" icosahedron, with 20 triangulated faces, 12 vertices, 30 edges, and its "endoskeletal" icosahedron counterpart. In the endoskeletal icosahedron, the triangulated outer shell is under tension and the internalized compression struts are "floating" within the tension shell. The compression struts span to opposite vertices; they do not touch one another, and do not pass through the center of the icosahedron.



Fig. 3.5.2 • Hierarchical tensegrity icosahedrons. The pattern is repeated at every organizational level, from subcellular to organism.

Like coins crowded together on a tabletop, bubbles in foam, cells in a beehive, biologic cells must conform and adjust to the pressures surrounding them. The individual cell must keep from being crushed by external forces. From the standpoint of efficiency and conservation of energy, crowded objects on a two-dimensional plane will pack closest as hexagons. Three-dimensional cells will conform to what has been known about foams for over 100 years; they will pack closest with three edges meeting at 120° and four edges meeting at a corner. Icosahedrons will pack closest around a central, smaller, icosahedron, following these rules. Fuller has described the closest packing of icosahedrons as the closest relationship of energy efficient, symmetrical, stable structures in three dimensions (Fuller & Applewhite 1975). In the past, cells were thought of as bags of fluid and the incompressibility of fluid kept them from being crushed. In the early 1930s, an internal cell skeleton (the cytoskeleton) was suspected, but it took another two decades to demonstrate it using the electron microscope. Ingber proposed that the cytoskeleton is a tensegrity structure with a mechanical structural framework to support cell integrity Table 3.5.1 Comparison of properties of biologic systems, lever mechanics and tensegrity icosahedral mechanics

Biologic systems	Lever systems	Tensegrity icosahedron
Nonlinear	Linear	Nonlinear
Global	Local	Global
Structurally continuous	Discontinuous	Structurally continuous
Gravity independent	Gravity dependent	Gravity independent
Omnidirectional	Unidirectional	Omnidirectional
Low energy	High energy	Low energy
Flexible joints	Rigid joints	Flexible joints

and he models these tensegrities as icosahedrons (Ingber et al. 1981). Following Wolff's law, the cytoskeleton will align itself in such a way as to resist the crushing compressive load, and the rigid tubulin of the cytoskeleton becomes its "bones". Levin proposed that the same mechanism created a hierarchical evolution of the musculoskeletal system, a hierarchical tensegrity (Levin 1982, 1986, 1988, 1990). Kroto, the Nobel Prize winner for his work on C₆₀, the icosahedral form of carbon, demonstrates the self-organizing properties of icosahedrons into sphere-like structures and "icosaspirals", helical structures of stacking icosahedrons (Kroto 1988). Icosahedrons and icosaspirals are ubiquitous in biologic structures, as demonstrated at every scale level: from 10^{-12} m (e.g., the fullerene molecule C₆₀ and some amino acids) to 10^{-9} m (viruses, microtubules), to 10^{-6} m (red blood cells, pollen grains), to 10^{-4} m and 10^{-3} m (radiolarians), all the way up to organisms such as pufferfish at 10^{-2} m, and greater. This hierarchy of structure development results in a fascial continuum, from subcellular to total organism.

Myofascia as the tensioner in the biotensegrity model

Central to this concept is the understanding that the fascia imparts a continuous tension to the system. Fascia displays the nonlinearity characteristic of all biologic tissues. In nonlinear tissues, the stress/strain relationship never reaches zero (a characteristic of linear materials); and there is always tension inherent in the system. It gives the "continuous tension", an essential component of tensegrity, that helps set the tone of the organism. There are active contractile elements in fascia (Schleip et al. 2005) and the fascial network is intimately bound to muscle (Passerieux et al. 2007). Muscle also has intrinsic "tone" and is never completely lax, and the entire fascial network is continually tensed, by both intrinsic tension and active contractions that can be "tuned". The mechanics of tensegrity structures are quite different than the lever mechanics that have been applied to biologic structures since Borelli's treatise (Borelli 1680). Contrary to lever mechanics, hierarchical tensegrity structures have only tension and compression members. There is no shear or torque, nor are there bending moments. Orientation in space has no effect on how the structure functions. Forces are distributed throughout the system rather than locally concentrated as they are in lever systems. The system functions as a single unit. All this makes for a more energy efficient system. Movement is not bending of hinges, but expansion, repositioning and contraction of tensegrities. An instant repositioning of tensegrities allows for freely moving joints while the triangulation imparts stability of form and function. Biotensegrity is the unifying mechanical structural concept that bridges the islands of information that we now have about fascia and its role in body functions, and makes them a unified archipelago for understanding fascia's role in anatomy and physiology.

Fascial training

The concept of biotensegrity not only offers a theoretical foundation to body mechanics and dynamics, it is also appropriate for establishing a concrete base to develop a process that can be seen as an internal fascial training. We propose mental motor imagery involving visual representation and kinesthetic awareness suggested by the principle of biotensegrity to support movement.

The stability of a tensegrity structure is due to the equilibrium between outward pushing of the rigid elements that tense the tension network, and inward pulling of the tension continuum that compresses the rigid elements without letting them touch each other: tensegrity structures can be seen as restrained expansion. Expansion (or space) creates tension. An increase of tension in a tensegrity structure lets it resist and become stronger. The training consists in using mental processes to generate a tangible feeling of the bones as space-makers and of the space between them. As a result, we can develop the perception of a tensional internal support. Once having found this internal support, it becomes possible to "relax" within it. "Relaxation", far from being a simple "lettinggo", with its well-known effect of collapsing and weakening, is a redistribution of tension within the tensile fascial network with the qualities of space and strength, and a balance of tension. Space, tension, resistance, strength, internal support, and relaxation are concomitant, even equivalent, characteristics.

Poles of movement

A further step of the training is to include these qualities in movement. While moving a tensegrity structure, we can make several observations. To move it, we grasp it at its two ends (Fig. 3.5.3) and impart a rotational movement in them, one in relation to each other, or move one end, stabilizing the other, which creates a relative opposite movement of the stable end. Movement has an intrinsically polar quality and we call those areas where movement is initiated, "poles of movement". Movement curves the structure, but the elements within respond by a new spatial organization without bending. Tension remains throughout the structure, on its concave side as well as on its convex side, and none of the rigid elements compress one another (Fig. 3.5.3). By focusing on the rotation of each pole separately, and letting each thumb follow a spiral whose direction is chosen to maintain tension on the concave side of the curve, we get a homogeneous curve, with all the elements involved relative to each other in a global movement (Fig. 3.5.3). If, instead, we focus on moving the poles toward each other in the space external to the structure, as is usually done in the movement instructions, the result will be an externally shorter distance between the poles and a sharp angle in the structure (Fig. 3.5.3). In this case, only a few elements of the structure have moved internally, the movement is local, and the tension is easily lost on the concave side.



Fig. 3.5.3 • Taking a tensegrity structure at the poles of movement and rotating them following the spirals maintains tension throughout the structure, also on its concave side (thumbs side). The movement is evenly distributed, the curve homogeneous. Moving the poles while focusing on the distance between them results in an unevenly distributed movement with a sharp angle in the structure.

In the body, poles of movement can be the two bone ends building a joint, the tensegrity structure between being the interarticular space. Poles can also be chosen as any two remote bones, like two vertebrae, the intervening tensegrity structure the considered segment of the spine, or head and foot, the tensegrity structure between being the whole body.

We may move one pole of a chosen body part, following the spiral that helps to maintain the tension on the concave side, while maintaining the other pole stable. The cervical curve and its poles, the head (occiput) and the first thoracic vertebra, can be taken as an example. If, when flexing the head slightly, we maintain awareness of the occiput moving along a spiral line directed upward and posterior,



Fig. 3.5.4 • Possible poles of movement in the spine. The text refers to the two upper poles comprising the cervical curve. The upper pole (occiput) follows the bold spiral directed upward and posterior in flexion, and the dashed spiral upward and posterior in extension. The bold spirals show the direction of the spirals according to the global mobilization of the spinal curves out of their more or less rigid shape.

(Fig. 3.5.4, upper bold spiral), it will prevent the head from "falling" forward and downward and it gives support to the front of the neck.

Integrating the myofascialskeletal system

Although the movement is subtle, one can feel the underlying vertebrae being carried along through the activated tension network around the neck. Perhaps one can also feel this movement spread over the spine and the whole body, since the body parts with their poles are all interconnected, as shown in Fig. 3.5.4 for the spine. The movement is slight, slow, employing minimal muscular force, and one can relax in the internally supported structure. If one takes one vertebra after the other as a pole and moves them in turn in the described manner, a complete flexion of the curve is achieved. Each movement is slight, but every part moves. The movement is well distributed,

occurring at every vertebral level, and the throat is not compressed. We can also change the direction and execute an extension following the dashed spiral (Fig. 3.5.4). Flexion moves the cervical curve evenly out of the lordosis, and extension moves it into the lordosis, but evenly and with the internal support that controls the movements that might disrupt the curve. This way, a body part that was rigid can be gently brought to life. If we now consider one joint the moving structure, awareness of the internal support, especially on the side of flexion, will prevent a closing or compression in the hollow of the curve.

In addition to the direction of movement given by the spiral, we also include the resisting quality of the tensioned elements. By training the kinesthetic perception of the subtle resistance that accompanies the movement (which is an adaptation from a mental technique used in a Chinese martial art), we enhance all the qualities already mentioned. It is also interesting to consider the resistance the result of two opposite movements: the movement actually performed and the counter-movement that slows it down. It is mentally challenging to perceive both simultaneously, but it is this training of the nervous system that results in a profound improvement of fluidity, strength, and elasticity of movement.

Summary

By internalizing these qualities, we can play with all the directions in space, connecting the spirals continuously in alternatively small or large movements, in slow or fast rhythms, which more overtly addresses the omnidirectionality of the fascial network, its elasticity, and its ability to react to different impulses such as stretch or vibration.

A characteristic of this training is the use of minimal muscular strength. Studies have shown that, whether a movement is mentally or physically performed, the nervous system tends to react similarly (Malouin et al. 2003) and muscle strength is developed (Ranganathan et al. 2004). It means that mental imagery allows us the use of muscular work in a remarkably economical manner to achieve optimal movement efficiency and ease.

With time, movements become naturally supported by the internalized principles of biotensegrity: the perception of internal space as well as the feeling of the ubiquitous tension that governs the mechanics of the body can lead to a maximal recruitment of the structure under optimally balanced tension. Consequently, movements become freer and more efficient, be it in movement disciplines, in daily activities, or in a therapeutic setting. An additional consequence of this approach to body structure and movement is to create a useful relationship to gravity. Instead of being a force that compresses our organism and makes us small and bent, gravity becomes a force that initiates space and strength in our structure.

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The subcutaneous and epitendinous tissue behavior of the multimicrovacuolar sliding system

3.6

Jean Claude Guimberteau

Introduction

Traditional basic concepts, terms, and information to describe natural organ intermobility seem to be at variance with anatomical reality. The traditional notion of different fascias or the sliding, gliding, collagenous system historically referred to as paratenon, connective or areolar tissues focuses on the separateness of these structures. Electron scanning microscopy suggests that this system does not consist of different, superimposed layers. In reality, there is a single, tissular architecture with different specializations. To emphasize its functional implications, we call this tissue the multimicrovacuolar collagenous (dynamic) absorbing system (MVCAS) (Plate 3.6.1).

Mechanical observations

In finger flexion, the movement of the flexor tendon is barely discernible in the palm. It is the same under the skin areolar tissue, which is the connective link between muscle, tendon, fat, aponeurosis and subdermal areas. The MVCAS system situated between the tendon and its neighboring tissue seems to favor optimal sliding. Tendon excursion can be large and rapid without resistance and without provoking any movement in neighboring tissue, thus accounting for the absence of any dynamic repercussions of such movement on the skin surface.

Microanatomical observations in vivo

Our observations are a result of over 20 years performing and improving complex flexor tendon transfers. We video-recorded the MVCAS during 95 in-vivo human surgical dissections using light microscopy (magnification $\times 25$) either directly under the skin or close to tendons, muscles or nerve sheaths. Further, an in-vitro study was carried out on human and animal samples, such as the flexor carpi radialis in cattle in which the organization is very similar to that of the human flexor profundus. The live results show what cadaver results can not: The MVCAS can be seen as a continuous structure composed of billions of dynamic, microvacuolar, multidirectional filaments, intertwining and creating partitions that enclose vacuolar shapes, organized in dispersed, fractal, pseudo-geometrics (Plate 3.6.1C). We want to express that the living matter is built of microvacuolar architectures (Plate 3.6.2).

Microvacuolar observations

Microvacuoles have diameters ranging from a few to several dozen micrometers and they vary in length from a few microns to a few millimeters, thus giving an overall disorganized, chaotic appearance (Plate 3.6.2A). The vacuoles are organized on several levels in different directions (Plate 3.6.2B): The pattern is pseudogeometric, polygonal, and tends to be icosahedric (Plate 3.6.2D). The levels are hierarchically arranged, fractally shaped and may span several partial subunits. The entire framework contains a highly hydrated (70%) proteoglycan gel. The lipid content (4%) is high. The sides of the intertwined vacuoles are composed of collagen 75% and elastine 25%.

An appearance of dynamic roles

The MVCAS we studied appears to be organized differently depending on the function of the structure. The collagen framework and the intravacuolar spaces give form and stability. The gel permits easy change of shape during movement while volume remains constant. The greater the distance that the structure must travel, the smaller and denser are the vacuoles. The microvacuolar structure has a consistency of conformation, capable of taking on many shapes, adaptable to the physical constraints that it undergoes, and has a form of memory allowing it to return to its initial position. A major role of this framework is to make sure that the structures can move freely without anything else moving around them. The overall configuration is highly efficient, combining great mechanical strength and lightness with thermodynamic energy conservation, diminishing friction with easy deformability (see Plate 3.6.5).

The tendon is not nourished by synovial fluid, but by its own vascular system, like every organ. A tendon has optimal functional value only when it is surrounded by its original sliding sheath and its vascular heritage. Our observation of this nutritional role has altered our surgical procedures. The MVCAS system is important for the nutrition of the structures embedded in it and acts as a frame for blood and lymph vessels. The histological continuity between the paratenon, the common carpal sheath and the flexor tendons illustrates the vascularization of this functional ensemble and introduces a new concept: The sliding unit, composed of the tendon and its surrounding sheaths.

A combined, transmitted and absorbed stress

Like a shock absorber, the microvacuolar system's function is to maintain the peripheral structures close to, but not mechanically affected by the body action in progress. Conversely, it also offers resistance, first minimally then increasing as the load increases, with

the fibers becoming more aligned in the direction of the stress. The energy stored in the fibers under tension gradually becomes lower the greater the distance from the stress, so the forces resulting from the pseudolinear stiffening are absorbed, and the structures become stabilized. When mechanical deformation does occur, each fiber is prestressed and connected to its neighboring fiber by a molecular adhesive link. We refer to this notion as "combined transmitted and absorbed stress" (Plate 3.6.3). When tension is applied to the link, the adjacent element undergoes tension and decreases in size little by little until it deforms. However, like rubber, the collagen fibers cannot be stretched indefinitely and may suddenly rupture. We find that vacuoles near areas of movement are deformed more than vacuoles at a distance. While we are not able to directly measure forces on an individual vacuole, this finding is likely because local forces are diffused to larger numbers of vacuoles the farther removed from the area of motion, and thus are less on any individual vacuole. This hypothesis is supported by our observation that in areas of greater motion, the vacuoles are smaller and thus would be able to transmit forces to more vacuoles in a shorter distance.

Trauma and vulnerability

MVCAS responsiveness and resiliency vary depending on the level of pathology present (such as edema, trauma, inflammation, obesity and aging), all of which create identifiable and unique changes in microvacuolar shape (Plate 3.6.4).

Edema is accommodated by increased intravacuolar pressure and collagenic distension, without any organic tissue destruction, but with fibrillar distraction limiting further distention and movement. Upon reduction of edema, restitutio in integrum will be the rule.

Open trauma destroys the precise interactions of the MVCAS. Hemorrhage, liquid extravasations, edema and hyperemia will disturb the mechanical balance, and the sliding system will require more force against resistance. Movement will be difficult. Tissues will become adherent from direct trauma and from lack of motion, which will further perturb mobility.

Inflammation induces intravacuolar hyperpressure with fibrillar dilacerations, creating small megavacuola and completely perturbing movement. Therefore, tissue is destroyed, similar to trauma reaction, and the restitutio in integrum will never be obtained. Permanent functional sequelae will be the result. Obesity begins both with adipocytes replacing glycolycans in the vacuola and with distension of vacuola and fibers. At this stage slow, progressive weight loss will still result in a return to the original morphology. Movement within the tissue is reduced and gravitation becomes more important in determining tissue morphology. In the second stage, vacuola are in extreme dilatation and the distension of fibers changes to dilaceration causing transformation to megavacuola that will in turn be filled by further adipocytes, changing body form. Only surgery will recreate tissue tension at this stage, by resecting excess skin and fat.

Aging represents a slow and progressive change to the physical balance of forces inside human tissue, with the predominance of gravitational forces rather than local motion on the MVCAS internal pretension.

MVCAS and globality

MVCAS appears to occur everywhere in the body, and it allows structures to adapt either to internal constraints or to the external environment. Even the intermediary structures, such as the deep premuscular fascia, are incorporated in this network and are connected with it on their superior and inferior faces, thereby increasing the shock absorbing properties of the tissue and allowing the structures to move interdependently. Whether it is in the abdominal, thoracic, dorsal, antebrachial regions or in the scalp, this tissue network is omnipresent: There is no space or wall where it is not to be found (see Plate 3.6.6). Even structures subject to little movement, such as nerves and the periosteum, are surrounded by this fibrillar tissue network, but with differences in the network itself and in the size of the vacuoles.

Seen in these terms, the whole structure of the body may be considered as an immense collagen network, differing according to the roles it must perform and the stresses it must undergo. In fact, MVCAS and the human body would seem to be the same tissue.

Research direction

What this sliding tissue system expresses is the essential fact that the human body is a whole system, so it calls for a more global vision regarding the way the different organs and structures work together.

The MVCAS system is organized to facilitate adaptation. Since it may be found in all living structures and at many levels, should it be considered as the quintessential architectural form of life? Could it be the initial structure, a network of vacuoles gathered together, self-organized and transformed into cells over time, then transformed by phylogenesis and chromosomal heritage?

Future research in molecular biology and the chemistry of proteins must examine the behavior of these basic but neglected structures of the human body. Traditional morphologic analysis cannot account for the intricate sequences and combinations of the fibrillar structures generated by movement. To be correctly understood, these phenomena must certainly be analyzed in terms of physical rules based on non-linear mathematics. The tendency to fractal geometric forms is found in all levels of living matter, and may be a fundamental building block that has developed during the course of evolution. Above all, the phenomena should be examined in live tissue in three dimensions with close attention to the relationships that exist between structures and their internal and external influences.

Conclusions

Everything points to MVCAS being the building block of an inter-organic network, functioning at different levels and performing three major mechanical roles: (i) responding to any kind of mechanical stimulus in a highly adaptable and energy-saving manner; (ii) preserving the structures, providing information during action and springing back to its original shape; (iii) ensuring the interdependence and autonomy of the various functional units.

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The physiology of fascia

An introduction

Frans Van den Berg

Manual therapists, chiropractors, physiotherapists, and osteopaths all need a thorough understanding of anatomy and physiology for the examination and treatment of a patient. Without knowledge of the anatomy of the locomotor apparatus, examination and making a diagnosis are impossible. A plausible explanation for the patient's symptoms can only be found with anatomical knowledge. This is the sense in which Cyriax (1978) regarded the examination of a patient as "applied anatomy". Anatomy shows us which structure is affected, while physiology teaches us which pathophysiological processes have taken place in the patient's tissue, why symptoms occur and which therapeutic stimulus is necessary for healing and regeneration.

In the field of manual therapy we are interested predominantly in the physiology of the locomotor apparatus and the connective tissue. For effective treatment, the therapist must know the construction, function, and physiological forces which stimulate the connective tissue. This is the only way that suitable therapeutic stimuli can be applied after injury and/or degeneration.

Kaltenborn (1989) saw in manual therapy a treatment application for so-called somatic dysfunctions (= disorders of the locomotor apparatus). These are manifested by pain, problems with joint mobility (hypomobility or hypermobility), and changes to other tissue (skin, subcutis, fascia, ligaments, muscles, etc.). This means that changes in the connective tissue caused by injury are not just limited to the primarily affected structure.

Our whole body physiology changes as a result of pain. The activity of the neuroendocrine system and the function of the internal organs change in this way. Muscle tone, the activity of the autonomic nervous system, the wake–sleep rhythm, and not least our behavior and conduct also change as a result of pain. All these changes are independent of the location or type of structure causing the pain.

An example will help us to understand this: If the primary pain is caused by a small tear in the annulus fibrosus (L5–S1), changes to all the structures in this area quickly appear. A "connective tissue zone" develops because of the changed tension and mobility of skin and subcutis against the body fascia. A "periosteal zone" also develops, with slight swelling and increased sensitivity to pressure, a hypertonic "muscle zone", and hypomobility of the relevant joints. Changed sensitivity to pressure and slight swelling of the ligaments also arise. All these changes are found not just locally in the area of the pain (L5–S1) but also in the autonomic area of origin – in our example, the area of about T10–L2.

Let us now accept the inverse argument that the primary pain is caused by an irritation of the uterus, the ovaries, the bladder, the kidneys, the prostate, the large intestine, etc. In this case too, the same changes occur in the autonomic area of origin at about the level of T10 to L2 by means of a viscerosomatic reflex. For the therapist, this mutual exertion of influence on the tissue means that it is sometimes very difficult to impossible to establish in the end which structure caused the primary pain.

Correspondingly, we run the risk in manual therapy that the symptoms felt by the patient in the locomotor apparatus are attributed exclusively to the locomotor apparatus as the cause – and the examination and later treatment will be limited to the musculoskeletal system. We also have to see patients as human beings and not reduce them to their locomotor apparatus. As explained, their symptoms can also be organic or have other causes (Van den Berg 2008).

Connective tissue of the locomotor apparatus

In this chapter we will restrict ourselves primarily to the connective tissue of the locomotor apparatus. The types of connective tissue relevant to manual therapy are hyaline joint cartilage and the unformed, taut, fibrous connective tissue. The latter can be found in the joint capsule, the fascia, and in the intramuscular and intraneural connective tissue. Networks are built by the collagen fibers and these can move and unfold in different directions. These networks occur because the tissue is strained and distorted in different directions. This gives rise to the mobility, which is typical for these structures.

Additional connections (pathological cross links) can arise under pathophysiological circumstances between the intercrossing collagen fibers in the network. These reduce mobility in the network and lead to capsule shrinkage and muscle shortening (see also Chapter 4.3) (Akeson et al. 1973, 1977, 1987, 1992; Grodzinsky 1983; Videman 1987; Brennan 1989; Currier & Nelson 1992).

The unformed, taut, fibrous connective tissue is definitely different from formed connective tissue which is found in tendons, ligaments, retinaculae, aponeuroses, etc. As this tissue is always stressed in the same direction, the collagen fibers tend to run parallel to each other. The therapeutic options are limited here, predominantly to deep frictions (performed after injury), with the aim of promoting circulation and optimizing healing (Van den Berg 2010).

Collagen has a tensile strength of about 500 to 1000 kg/cm². This extremely high stability explains why collagen and connective tissue – whether a joint capsule or a ligament – cannot be significantly extended (Leadbetter et al. 1990; Currier & Nelson 1992; Aaron & Bolander 2005). The original joint mobility is the maximum that can be achieved by using joint mobilization and/or muscle stretching. In adults, it is virtually impossible to achieve greater mobility than was originally there. The reason lies once more in the physiology: Connective tissue can only become longer by the deposit of collagen molecules strung sequentially together. This usually happens under the influence of growth hormones; the optimum influence occurs in the first eight years of life. Think of gymnasts or ballet dancers who begin in childhood and train to achieve the required mobility. Appropriate mobility and extension stimuli are also applied far more often among top athletes than in manual therapy treatment or even in a patient's home exercise program.

After periods of longer immobilization it is usually very difficult to regain the former level of mobility. The majority of patients invest too little time in performing the necessary exercises and think that two visits a week to the manual therapist are sufficient (Van den Berg 2007).

Paoletti (2001) describe the whole connective tissue of the locomotor apparatus as fascia. It is certainly true that the fascia apparatus holds together all the structures in our body, from our head to the tips of our fingers and toes. You could say that the joint capsule is a specialized fascia and that the ligaments are functional adaptations or swellings of the fascia.

Construction and function

Connective tissue consists of cells and extracellular matrix. We differentiate between fibroblasts, chondroblasts, and osteoblasts. Sometimes we talk of fibrocytes, chondrocytes, and osteocytes. The difference lies in the synthesis activity: Blasts have a higher synthesis activity than cytes, which are characterized by more mitochondria and a larger endoplasmic reticulum (Leadbetter et al. 1990; Currier & Nelson 1992; Finerman & Noyes 1992; Aaron & Bolander 2005).

In the embryo, all connective tissue cells stem from mesenchymal cells. The type of connective tissue cell into which the mesenchymal cells will develop is predominantly determined by the mechanical stress to which the cells and their cell membrane are subjected. As the cell membrane does not possess any great mechanical stability, the cell forms an extracellular matrix to protect against mechanical stress. The construction and composition of the extracellular matrix again depends on the form of the mechanical demands (Van den Berg 2010).

Traction or tensile load versus pressure

If the force on the tissue is predominantly traction, the fibroblasts formed as a result produce predominantly type I collagen fibers and only a few elastic fibers and a small quantity of ground substance. For example, the matrix of a tendon or a ligament is constructed of up to 97% collagen fibers. Only about 1% to 2% of the dry weight are elastic fibers and about 0.5% to 1% are ground substance. Ground substance serves here to reduce the friction during movement between the collagen fibers and allow diffusion in the tissue by deposit of water (Van den Berg 2010).

On the other hand, if pressure is the dominant force, as in hyaline cartilage and nucleus pulposus, the chondroblasts formed here produce almost entirely ground substance. The nucleus pulposus therefore consists of about 98% to 99% ground substance and up to about 1% to 2% very thin type II collagen. The collagen here has the task of mechanically protecting and stabilizing the ground substance (see also Chapter 4.2) (Buckwalter et al. 1988; Eyre et al. 1989; Currier & Nelson 1992).

The physiological construction of the connective tissue determines the required treatment, depending on the injuries: If there is an injury to the joint capsule, gradually increased extension (frequent movement without pain) should be applied to the tissue or cells, so that the original construction and stability of the joint capsule can be achieved.

However, if there is injury or degeneration of the joint cartilage, treatment should consist of the application of physiological force by compression. Accordingly, the joint should be regularly treated with gradually increased application and relief of axial force.

Physiological stimuli

It is therefore questionable that manual therapists frequently treat patients with problems in the area of the joint cartilage with traction. It is often advised that strain (axial loading) should also be minimized. It is obvious that this cannot lead to repair of the cartilage structures – there are no physiological stimuli.

Even after injuries to the intervertebral disc – this is usually a lesion of the annulus fibrosus which lends itself to traction – physiological stress should be included in the treatment. This means that flexion and rotation movements must be made. (In reality, however, this important stimulus to regeneration is very often forbidden to patients.)

Similar considerations should be taken into account for injuries to the meniscus as well: Although it repeatedly says in the literature that the meniscus has a weight-bearing function, this is very doubtful if you look at its histologic construction. The meniscus consists largely of type I collagen and has only 1– 2% type II collagen and hardly any ground substance. It follows that the meniscus is primarily designed for traction. As a result, therapy should include traction exercises for the meniscus. This is achieved by gradually increasing rotation with the knee joint flexed (Van den Berg 2010).

Wound healing and manual therapy

While the type of stimulus for a tissue can be derived from the histology and physiology, the wound healing phase tells us at what intensity we have to apply the necessary physiological forces/loading/stimuli during therapy.

Wound healing is divided into three or four phases (Fig. 4.1.1). The first "inflammatory phase" usually lasts 5 days and is divided into a vascular phase (trauma to day 2) and a cellular phase (days 3 to 5). Immediately after the injury bleeding normally occurs in the tissue. This bleeding activates the cells in the



Fig. 4.1.1 • Time scale for wound healing.

Wound healing
vascular phase to release important substances that initiate processes such as coagulation and wound healing. In the subsequent cellular phase, mobile fibroblasts migrate from the surroundings into the injury area. These cells are then called myofibroblasts. They promote wound contraction and are responsible for stabilizing the wound (Kloth et al. 1990; Cohen et al. 1992; Currier & Nelson 1992; Finerman & Noves 1992; Clark 1996; Aaron & Bolander 2005). In both phases, therapy with mechanical tissue stress must be very restrained. It is important to avoid further bleeding. Exercise and weight bearing are only allowed in an area of movement which is totally pain free. However, a prerequisite for this is appropriate pain perception by the patient, which is not dulled by analgesia.

In the subsequent "proliferation phase" (day 5 to day 21–28), the matrix synthesis started in the cellular phase is much intensified. Wound closure is achieved by a filigree network of type III collagen. This collagen is relatively thin and does not provide the tissue with any great mechanical stability. For this collagen network to attain an almost identical construction to the original tissue, the tissue in this phase of wound healing must be confronted with its normal physiological stress (Kloth et al. 1990; Cohen et al. 1992; Currier & Nelson 1992; Finerman & Noyes 1992; Clark 1996; Aaron & Bolander 2005).

Note that the proliferation phase in poorly perfused tissue such as tendon, ligament, meniscus, or intervertebral disc can also last up to 6 weeks. However, since the therapist is basically guided by information on the pain from the patient, this does not have any consequences at all for the type of treatment. In this case, it is only necessary for the amount of weight bearing to be increased more slowly.

Once the wound has been closed with type III collagen after conclusion of the proliferation, it is followed by the "reconstruction phase" (day 21–28 to day 360). A type of interim "consolidation" phase is also mentioned in some of the literature (day 21–28 to day 60) (Kloth et al. 1990; Cohen et al. 1992; Currier & Nelson 1992; Finerman & Noyes 1992; Clark 1996; Aaron & Bolander 2005). As far as therapy is concerned, stress on the tissue should now be slowly increased to push ahead the reconstruction of unstable type III collagen into stable type I collagen and return the tissue to its original stability. The increase in weight bearing in the therapy is dependent on what the patient requires of weight

bearing on the tissue for his work or sport. This means that if the patient has to lift a weight of 200 kg in his daily life, therapeutic training should continue until the training weight corresponds to the everyday weight.

Conditions for wound healing

Drugs

The starting point of physiological wound healing is inflammation. Logically, drugs that inhibit or even eliminate inflammation are counterproductive for wound healing. This disastrous effect is most obviously seen in tissue such as tendons, ligaments, and insertions. As these structures bleed very little when injured, due to the small amount of vascularization, only a small amount of inflammation is generated. The poorer the inflammatory reaction, however, the worse the prognosis for wound healing. The negative influence of anti-inflammatory drugs on wound healing has already been evidenced by many studies (Ng 1992; Billingsley & Maloney 1997; Muscara et al. 2000; Elder et al. 2001; Yugoshi et al. 2002; Marsolais et al. 2003; Sikiric et al. 2003; Bergenstock et al. 2005; Kaftan & Hoseman 2005; Murnaghan et al. 2006; Tortland 2007).

From a therapeutic point of view the standard method here is deep friction. The increased release of inflammatory mediators induced by stimulation of the tissue improves perfusion and wound healing.

Even the use of analgesics can be a disruptive factor for wound healing. The great danger of analgesic drugs is that the patient is no longer informed of the current ability to withstand stress on the tissue. As the natural warning signal which is pain is missing, the patient or even the therapist continues to exceed the physiological stress limits and thereby causes new and permanent damage. The wound healing process stagnates in a repetitive inflammatory phase (Bisla & Tanelian 1992; Brower & Johnson 2003; Dormus et al. 2003; Northcliffe & Buggy 2003; Scherb et al. 2009).

Nutrition

Because connective tissue consists predominantly of proteins, the take-up of protein through nutrition is very important. Protein is available in the form of plant or animal products. It is problematic that animal protein is acid-forming and can lower the interstitial pH value. If the pH value falls below 6.5 fibroblasts find it difficult to carry out their normal synthesis function. As a result, the tissue degenerates and healing can no longer take place (Geiersperger 2009; Van den Berg 2010).

As the energy from the mitochondria is predominantly made available by burning up glucose, a sufficient quantity of sugar in our diet is also important. However, if sugar is predominantly added through short chain carbohydrates, the strain on the pancreas is increased. The result can be the development of type II diabetes, a disease of modern civilization. If the blood sugar level is permanently too high, regeneration and healing are also made more difficult. Refined sugar is also extremely acid-forming (see above).

Finally, the consumption of fat should concentrate mostly on unsaturated fatty acids. Important here are the essential unsaturated omega 3 and omega 6 fatty acids. Prostaglandin 2, which is important after an injury, is produced from omega 6 fatty acids. Omega 3 fatty acids enable the formation of prostaglandins 1 and 3, which control inflammation as antagonists of prostaglandin 2. (In the western diet there is a predominance of omega 6 fatty acids.) An excess of saturated fatty acids, on the other hand, can cause atherosclerosis and tissue perfusion problems.

Vitamins, minerals, and trace elements are also essential for connective tissue stability. These substances allow stabilizing connective bridges in the collagen (Geiersperger 2009; Van den Berg 2010). See also Chapter 7.23.

Perfusion

In order to make the right nutrients for their synthesis processes available to the cells (as part of the healing process) – particularly after injury – the tissue must be sufficiently perfused. This is demonstrated not least in the classic signs of inflammation such as heat, swelling, and redness. The most negative influences on tissue perfusion are smoking (Holm & Nschemson 1988; Battie et al. 1991; Silcox et al. 1995; Hadley & Reddy 1997, Iwahashi et al. 2002; Oda et al. 2004; Zakaria & Sina 2007), atherosclerosis (Kurunlahti et al. 1999; Dwivedi et al. 2003; Turgut et al. 2008; Kauppila 2009), and increased sympathetic reflex activity.

Stress

Psychological stress causes the release of an increased level of stress hormones such as cortisol. Cortisol inhibits collagen synthesis and slows or even prevents healing and regeneration. Sympathetic reflex activity is also increased by stress (see above).

Internal organs

For the macronutrients from the diet to be reconstructed into micronutrients valuable for the cells, the function capacity of the digestive tract must be known. For optimum digestion, food should be well chewed to increase the contact surface between the food and the digestive enzymes. The smaller the portion that lands in the stomach, the easier it is for further digestion to take place. If it is too large, the gastric entrance is also mechanically stressed. Drinking during the meal thins the gastric juices and transports incompletely digested particles into the small intestine. This reduces the uptake of nutrients in the small intestine. If nonsteroidal anti-inflammatory drugs are taken, the mucous membranes of the stomach and small intestine are weakened, restricting the uptake of nutrients (Van den Berg 2010).

The liver, the gall bladder, and the pancreas are among the organs responsible for the production of digestive enzymes and the conversion of glucose to fat or of fat to glucose. The liver also plays an extremely important role in detoxification and the neutralization of acids. Other detoxification organs are the large intestine, the kidneys, the skin, and the lungs. Water is required for all detoxification processes and must be added in sufficient quantity (Van den Berg 2010).

Immune system

An immune weakness can develop as a result of dietary deficiency, poor large intestine function, the frequent taking of antibiotics, or as a result of the surgical removal of important parts of the immune system such as the appendix, the tonsils, etc. This can lead to autoimmune reactions which can then lead to chronification of inflammation and poor regeneration and healing.

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Fascia is alive

How cells modulate the tonicity and architecture of fascial tissues

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Cellular populations in fascia

Cells constitute only a minor portion of the volumetric quantity of fascial tissues. Nevertheless, they play a major role in modulating their architecture and stiffness. Among various cell-types the fibroblasts and sublineages are the most prominent cell-line in fascia. These cells are like nomadic construction workers, as well as cleaners and repair handymen, for the extracellular matrix. Their life span is estimated to be several months. In the past, metabolically active "fibroblasts" were distinguished from the less active "fibrocytes". More contemporary texts, however, now tend to describe both forms as "fibroblasts". Besides secreting precursors of most of the components of the extracellular matrix (a major exception being its large water content), and in secreting precursors for enzymes like collagenase that help in breaking these tissues down again, they also play important roles in tissue injury repair.

There are usually only a few immune cells like macrophages, a few mast cells, and some sporadic lymphocytes present in fascia. The mast cells contain granules rich in histamine and heparin, which play a key role in the inflammatory process. When activated, mast cells rapidly release these granules into the ground substance, activating blood flow and immune defense.

An often undervalued cell population within fascial tissues are univacuolar adipocytes. They are particularly abundant in areolar connective tissues, yet also in areas where fascial tissues frequently are engaged in shear and sliding motions. In addition, they are present in areas like the heel pad which are exposed to frequent pressure in addition to tensional loading. Here, the fat cells are arranged much more tightly and in smaller units than elsewhere, forming a most effective cushion. Although many people tend to regard these cells as less precious elements of their body, they fulfill important functions. This includes their recently discovered endocrinal functions: adipocytes are not only important producers of estrogen, but also of several other peptides and cytokines. Through these they influence appetite regulation, insulin/glucose regulation, angiogenesis, vasoconstriction, blood coagulation, and can even express proinflammatory conditions in the body. They are also one of the producers of the important cytokine transforming growth factor (TGF)-β, which we will address later. The detrimental effects of severe obesity on many physiological functions of the body are largely caused through several of these peptides and cytokines. On the other side, the common cosmetic surgery of liposuction can be expected to perturb local and global physiology and should therefore be considered with a degree of caution similar to a partial removal of other endocrine organs in the body. The humoral factors are transmitted to and from the adipocytes via the bloodstream. Fat tissue is well vascularized, especially below the superficial fascial layer. Therefore, another portion of cells in fascia make vascular, lymphatic and neural tracts, however small those vessels may be.

Fascial tonicity

In an examination of human lumbar fascia, a group of biomechanical investigators around Yahia et al. (1993) discovered its ability for tissue contraction. Three years later, the German anatomy professor Staubesand, in an examination of the human fascia profunda of the lower leg, documented the presence of smooth muscle-like cells (Staubesand & Li 1996). As he also found a rich presence of sympathetic nerve fibers in their vicinity, he postulated a potential close connection between sympathetic activation and fascial tonus regulation. Indeed, many clinicians report a frequent association between long-term psychological stress and a perceived increase in palpatory myofascial stiffness. Such increase in tissue stiffness seems to be present also at rest, a condition for which most electromyography experts agree that most skeletal muscles are electrically silent (Basmajian & DeLuca 1985). It has therefore been suggested that human resting muscle tone may be significantly influenced by changes in fascial stiffness (Masi & Hannon 2008).

This was the background for the authors' research group to provide a more thorough examination of human fasciae for the presence of contractile cells. In a collection of biopsy tissues - taken from human lumbar fascia, iliotibial tract, interspinous ligament and plantar fascia - immunohistochemical staining for the presence of α -smooth muscle actin (ASMA) stress fiber bundles was performed. Such staining is commonly used to identify the presence of cells with smooth muscle-like contractile features. Subsequent microscopic analysis then revealed that some of the stained cells could be identified as smooth muscle cells, which were then involved with the formation of blood vessels. The remaining cells were myofibroblasts, a type of connective tissue cells whose presence in fascia had previously been reported only from wound healing or pathological tissue contractures. These highly contractile cells - generally considered as a special phenotype of fibroblasts - were found in all tissue samples, although with very large density variations.

Unexpectedly, it was also revealed that the intramuscular perimysium seemed to express a higher density of myofibroblasts than the endomysium, perimysium, or fascia profunda. Interestingly, meat scientists report that tonic muscles tend to contain a thicker perimysium, giving them the appearance of tough meat (in contrast to the tender meat quality of phasic muscles, which have a much thinner perimysium; Borg & Caulfield 1980). It has therefore been suggested that the augmented resting stiffness of some tonic muscles could be related to an enhanced myofibroblast density in their perimysium (Schleip et al. 2006b). In some tissue samples of the lumbar fascia a dramatically increased density of myofibroblasts was found. Density was then comparable to that reported in Dupuytren contracture or frozen shoulder. This could suggest that the lumbar fascia may sometimes express a pathological condition similar to those two common fascial tissue contractures (Fig. 4.2.1).

It has been proposed that a tendency for high myofascial stiffness might be a polygenic human trait, being associated with a predisposition for living in colder climates (Masi & Hannon 2008). This is supported by the high prevalence of ankylosing spondylitis and Dupuytren contracture in people with a Northern European ancestry line. On the other hand, the condition of general joint hypermobility is more frequently expressed in people from Africa and Southern Asia. It has therefore been suggested that general joint mobility (and tissue stiffness) could be influenced by myofibroblast density in muscular fasciae (Remvig et al. 2007). This would be congruent with the finding that hypermobile people tend to have a slower wound contracture and reduced scar formation, whereas people with "Vikings' disease" (i.e., Dupuytren contracture) tend to have faster wound contracture, are more prone to scarring, and are also more often affected by other myofibroblast-driven



Fig. 4.2.1 • Indications for fascial pathology of "frozen lumbar"? Section of the posterior layer of the lumbar fascia at the level of L2. Note the high density of myofibroblasts, which in this patient is comparable to their reported density in the shoulder capsule during a "frozen shoulder" pathology. This suggests that, at least in this patient, the low back region could be affected by fascial contracture and stiffening similar to that of a "frozen shoulder". Arrows indicate examples of stress fiber bundles containing α -smooth muscle actin (a differential marker for myofibroblasts), which are stained here in dark gray. Length of image 225 µm. Adapted from Schleip et al., 2006a, with permission.

Fascia is alive

fascial contractures, such as frozen shoulder or plantar fibromatosis (Hart & Hooper 2005).

From myofibroblast contraction to tissue contractures

It is assumed that most myofibroblasts develop out of regular fibroblasts. This transition is stimulated by an increase in mechanical strain, as well as by specific cytokines Figure 4.2.2. Myofibroblasts play an important role during wound healing, and are also involved in many pathological fascial contractures (such as Peyronie's disease, hypertrophic scar, plantar fibromatosis, Dupuytren contracture, or frozen shoulder). Due to their possession of dense ASMA stress fiber bundles, their contractile capacity is four times stronger than regular fibroblasts.

In order to further investigate the potential contractile function of fascial myofibroblasts, the authors' group performed mechanographic in vitro examinations for active fascial tissue contractions. Using rat lumbar fascia within an organ bath environment, several substances were shown to induce measurable tissue contractions. These include the cytokine TGF- β 1 as well as thromboxane, a lipid produced by blood platelets and which is associated with blood clotting. Interestingly, thromboxane has been associated with the arachidonic acid pathway characteristic of chronic silent inflammations. Tissue contractions in the organ bath could be elicited in a time frame of 5 to 30 minutes and the measured



Fig. 4.2.2 • Two states of myofibroblast differentiation • In vivo, fibroblasts might contain actin in their cortex but they neither show stress fibers nor do they form adhesion complexes with the extracellular matrix. Under mechanical stress, fibroblasts will differentiate into protomyofibroblasts, which form cytoplasmic actin-containing stress fibers that terminate in fibronexus adhesion complexes. Protomyofibroblasts also express and organize cellular fibronectin – including the ED-A splice variant – at the cell surface. Functionally, these cells can generate contractile force. TGF- β 1 increases the expression of ED-A fibronectin. Both factors, in the presence of mechanical stress, promote the modulation of proto-myofibroblasts into differentiated myofibroblasts that are characterized by the de novo expression of α -smooth muscle actin in more extensively developed stress fibers and by large fibronexus adhesion complexes (in vivo) or supermature focal adhesions (in vitro). Functionally, differentiated myofibroblasts generate greater contractile force than protomyofibroblasts, which is reflected by a higher organization of extracellular fibronectin into fibrils. From Tomasek et al., 2002, with permission.

forces were strong enough to predict a potential influence on mechanosensory regulation and motoneuronal reflex regulation. However, they were significantly lower (and slower) when compared with skeletal muscle contractions with a similar cross sectional diameter (Schleip et al. 2006a).

Application of botulinum toxin into the organ bath led to a measurable decrease in tissue contraction. While botulinum toxin injections are well known for their specific effect on the synapse of the neuromuscular junction, it's most poisonous component, C3-transferase, has been shown to also inhibit myofibroblastic force production (Parizi et al 2000). Possibly this finding could explain the often dramatic results of botulinum toxin applications in some (but not all) cases of spastic muscular pareses (Snow et al 1990, Dietz 2002). If so, then a palpatory or ultrasound assisted examination of fascial stiffness in an individual patient could help to predict, whether or not such treatment promises to be useful. While myofibroblasts are able to induce small tissue contractions only (up to 4.1 μ N/cell) during a time frame of several minutes, an incremental addition of such cellular contractions over several hours and days can lead to long-term tissue contractures, which include matrix remodeling (Fig. 4.2.3). It is therefore conceivable that appropriate changes in the biochemical or mechanostimulatory environment of the fascial myofibroblasts could induce profound changes in tissue stiffness.

Modulators of fascial contractility

Let us explore which factors could influence fascial tonicity. The reported finding of the stimulatory effect of thromboxane – together with general physiological considerations on the contractile



Fig. 4.2.3 • Active cellular contraction as a step towards chronic tissue contracture • Three phases of force generation of connective tissue cells in an in vitro collagen lattice environment are shown. Migrating fibroblasts exert tractional forces on a compliant substratum. With sufficient stiffness of the substratum fibroblasts transform into myofibroblasts, which express a much higher capacity for cellular contraction. Creation of new matrix components then assists in stabilizing the new collagen organization, which leads in small incremental steps via collagenmatrix remodeling to long-lasting tissue contracture. Addition of cytochalasin D an inhibitor of filamentous actin formation in the contraction phase shortly after the force plateau is reached results in a total loss of force (not shown). By contrast, addition of cytochalasin D much later in the putative contracture phase (arrow) results in residual tension remaining in the matrix. This residual tension is due to irreversible remodeling-shortening of the collagen network. From Tomasek et al., 2002, with permission.

kinetics of fibroblasts and myofibroblasts – suggests that a proinflammatory biochemical milieu may tend to foster an increase in fascia stiffness. In return, an anti-inflammatory (nutritional or other) biochemical condition may go along with a tendency towards a moderate or lower fascial rigidity. For manual therapists it is therefore of interest, that cell culture experiments by Meltzer & Standley (2007) showed that a treatment protocol simulating the mechanostimulatory pattern of repetitive strain injury motions induced an increased fibroblast expression of proinflammatory cytokines, whereas the application of the mechanostimulatory pattern of an osteopathic indirect treatment protocol induced a more antiinflammatory cytokine expression.

The surprising discovery of important signaling properties of the gaseous messenger molecule nitric oxide in the 1980s revolutionized the field of physiology; it was also subsequently honored with a Nobel Prize. This versatile messenger substance is produced by many cells in the body and has been shown to be a profound relaxant on vascular smooth muscle cells. While systematic examinations of the effect of nitric oxide on fascial contractility have not yet been reported, the molecular dynamics involved in its effects on vascular cells suggest that it may also exert a similar relaxing effect on myofibroblastic contractile activity. If so, this would suggest that nutritional support with arginine and associated amino acids, as well as meditation founded on mindfulnessbased stress reduction (Stefano & Esch 2005), could influence fascial tonicity. Further research is necessary to confirm these hypothetical considerations.

Interaction with the autonomic nervous system

In his classic treatise on fascial tonicity, Staubesand suggested a strong influence between the autonomic nervous system (ANS) and fascial tonus (Staubesand & Li 1996). In particular, he suggested that sympathetic activation may lead to an increased cellular contraction within fascial tissues. Schleip et al. (2006a) used their in-vitro mechanographic examinations of fascial tissues to investigate whether they could elicit any measurable tissue contractions with sympathetic neurotransmitters (epinephrine (adrenaline), norepinephrine (noradrenaline), acetylcholine; or, respectively, their chemical equivalents). Nevertheless, in, spite of the great motivation and patience of all the investigators involved, no such effect could be found. It appeared that Staubesand's theory, as beautiful as it seemed, did not match with the complex physiological dynamics in real bodies. It also seemed to be in some conflict with the finding that fibroblasts, as well as myofibroblasts, often migrate through the tissue to a considerable degree; which seems to make a synaptic signaling transmission from sympathetic nerve endings difficult, if not impossible (see Plate 4.2.1).

It therefore came as a considerable surprise when a team of researchers from the field of psychoneuroimmunology recently reported that they had finally found a missing link between sympathetic activation and altered T3 cell expression in the lymph nodes, and that this link is the cytokine TGF-B1, a well known myofibroblast stimulator (Bhowmick et al. 2009). Note that, previous to this finding, it had been known for a long time that sympathetic activation, such as in psychological stress or anxiety, tends to have profound effects on the T3 cell activation of the immune system. However, it was not known through which exact pathway or cytokine transmission the communication between the ANS and this response of the immune system is accomplished. Their new clarification of this pathway now suggests that sympathetic activation induces an increased TGF-B1 expression; and - since this cytokine is known as the most potent stimulator of myofibroblasts contraction - that this may also lead towards an increased fascial contractility.

So far, the very potent stimulatory effect of TGF- β 1 on myofibroblasts has been documented in cell culture environments only. However, preliminary examinations by the authors' team are confirming the assumption that the addition of this cytokine at small physiological concentrations tends to also elicit clear tissue contraction of whole bundles of rat lumbar fascia in an organ bath (Schleip et al. 2006a).

This allows us to revisit – or rehabilitate – Staubesand's basic model of a close connection between fascial tonus and the ANS. The fact that the pathway via TGF- β l expression seems not to be dependent on a local synaptic connection fits well with the slow contractile kinetics observed in fascial contractions (see Fig. 4.2.3). In addition to the effect of TGF- β l already discussed, it is possible that sympathetic activation may exert other changes in the biochemical milieu of the extracellular matrix which influence cellular activity in fascia. One such effect could be a sympathetically induced change in pH in the ground substance. In fact, an in-vitro study by Pipelzadeh & Naylor (1998) indicated that



Fig. 4.2.4 • Proposed interaction between the autonomic nervous system and fascial tonicity. Sympathetic activation tends to activate TGF- β 1 expression (as well as probably other cytokines) in the body, which has a stimulatory effect on myofibroblast contraction, thereby leading to an increase of fascial stiffness. In addition, shifts in the autonomic nervous system state can induce changes in pH, which affects myofibroblast contraction as well (see Chapter 4.4). On the other hand, skillful therapeutic stimulation of mechanoreceptors in fascia – particularly of Ruffini or free nerve endings – can induce changes in the autonomic nervous system.

myofibroblast contractility can be significantly increased by a lowering of the pH. However, this study involved a small sample size only and has not yet been replicated by other research teams. More detailed considerations on the effect of the microenvironment on fascia will be found in Chapter 4.4.

Figure 4.2.4 illustrates a possible two-way interaction between ANS activation and fascial tonicity. Besides the influence of the ANS on cellular contractility in fascia, this graph also emphasizes the potential influence of therapeutic fascial stimulation on ANS tuning. Stimulation of non-nociceptive mechanosensory free nerve endings (belonging to either unmyelinated C-fibers or to myelinated A δ fibers) can influence ANS tuning. In addition, stimulation of Ruffini corpuscles – which are reportedly particularly sensitive to slow shear application – tends to inhibit sympathetic activation (Schleip 2003).

Indications for rhythmic oscillations of fascial tissues?

It has been known for some times that connective tissue cells – when embedded in a cell culture medium with a collagen grid – tend to express

periodic oscillations. In particular, it has been shown that they express rhythmic calcium oscillations and that these oscillations are accompanied by contractions of these cells upon their immediate environment (Salbreux et al. 2007). A recent study by Follonier et al. (2010) specifically demonstrated that myofibroblasts tend to oscillate in such an environment in temporal synchronicity, given that they are in mechanical contact with each other (Fig. 4.2.5). This study could also demonstrate thatthe synchronization of the observed contractions is not mediated via the gap junctions but via their adherence junctions. (Note that gap junctions are specialized for chemical signaling between cells. Adherence junctions on the other side are thickenings in the cell membrane - a typical characteristic of myofibroblasts - through which these cells exchange mechanical signals via integrin fibers with the extracellular matrix.) The observed myofibroblastic oscillations had a period length of 99 s (with a standard deviation of ± 32 s).

It is an intriguing question whether the very slow rhythm observed in these cell cultures – with one cycle taking more than one and a half minutes – could be related to the so-called "long tide" oscillations which are taught in biodynamic osteopathy (Becker 2001; Sills 2004). According to Sutherland (1990), this pulse can be felt through the listening hands of a trained practitioner who is in a deep state of mindful relaxation. Also called "breath of life", it has a reported period length of 100 s.

The almost exact congruence of reported period lengths between this clinical concept and the recent myofibroblast oscillation studies is remarkable. Whether they are indeed related to each other remains to be examined. A more than coincidental congruence between these two reported rhythms would require several preconditions. One of them is that there is indeed a sufficient inter-rater reliability of the palpatory perception. Another is that myofibroblasts in the real body express the same synchronization of their contractile activity as they do in the very differently composed - and more crowded - cell culture environment. Given that the integrin fibers are able to transmit mechanical cellto-cell signaling over some distance, it cannot be excluded that this may be possible. However, further research is necessary to elucidate whether the "breath of life" perception is indeed related to an active fascial contractile rhythm, or whether it is more likely due to other processes, such as the ideomotor perceptions of the practitioner (Minasny 2009).



Fig. 4.2.5 • Myofibroblasts express rhythmic calcium oscillations. (A) Recording of fluorescence activity of five individual cells, which were previously stained with Flura-2. (B) Fourier analysis of the dominant wavelengths of oscillating connective tissue cells in a cell culture environment. Further analysis revealed a common peak around 99 ± 32 s for 87% of the cells, as well as a second maximum of 221 ± 21 s for 13% of the cells. While most cells in the first group were myofibroblasts, the latter population fulfilled the morphological criteria of ASMA-negative fibroblasts. From Follonier 2010 with permission.

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Extracellular matrix

Frans Van den Berg

As already described in Chapter 4.1, extracellular matrix consists basically of three components: connective tissue fibers (collagen and elastic fibers), the ground substance (consisting of glycosaminogly-cans (GAGs), and proteoglycans (PGs)), and non-collagenous link proteins.

The matrix is produced by various connective tissue cells. The composition of the matrix and the relation between the individual components is determined by the mechanical stress which affects the cells in each case; see Fig. 4.3.1 (Leonhardt 1987). A large quantity of water is associated with the matrix: one of its functions is to enable essential processes, such as the diffusion of nutrients and waste products.

Collagen fibers

Collagen fibers are white, so collagen-rich tissue is correspondingly white. Collagen fiber "turnover", the reconstruction phase, normally lasts about 300 to 500 days (Fleischmajer et al. 1990; Currier & Nelson 1992; Van den Berg 2010). In tissues with longer turnover (e.g., intervertebral discs) a yellow to yellowbrown discoloration can be found in older patients; this is true, for example, in the case of the intervertebral discs (Ishii et al. 1991). After water, collagen is the second largest component of connective tissue. It represents approximately 30% of our body protein.

Collagen types

Twenty-eight collagen types have so far been identified. For many types, little is known about their specific tasks in the matrix and some are not known at all. The most important collagen types are types I, II, III, and IV. They represent approximately 95% of all collagen. Type I collagen makes up about 80% of these 95% (Leadbetter et al. 1990; Meyer et al. 2007).

Structure of collagen

Collagen basically consists of three long protein chains (polypeptides), each possessing the conformation of a leftward rotated helix and described as "alpha helix". These three helical polypeptides are then twisted together into a right-handed helix, to form a triple helix. That is the true collagen molecule. If it is intracellular, it is called a 'tropocollagenic molecule'. It is formed in the endoplasmic reticulum, has a length of about 280 nm and a diameter of about 1.5 nm.

The collagen molecules bind together in the interstitium and form a collagenic microfibril, also called a subfibril. Several microfibrils wrap around each other in a spiral and then form a collagen fibril. The diameter of the fibrils is 10 to 300 nm. (Collagen fibrils are mainly found in hyaline cartilage and the nucleus pulposus.)

Collagen fibers occur, for example, when the fibrils spiral around each other in type I and III collagen and a few other types of collagen. In tendons and ligaments, the fibers twist around each other again and form fibrillar bundles. The coils in the collagen are always opposed from one stage to the next – a left-handed helix is followed by a right-handed helix and then a left-handed one again, etc. During traction, the fiber spirals interwind and gain in stability. This is how collagen achieves its enormously high tensile strength of 500–1000 kg/cm², which is higher than steel. The stability of the collagen is dependent on the



Fig. 4.3.1 • Causal histogenesis of connective tissues according to Pauwel's theory of developmental differentiation. From Leonhard, 1987, with permission.

physiological cross links. These exist between the individual protein chains within the collagen molecules and as a result of binding of the collagen molecules with each other. The occurrence of cross links is a result of the biochemical bridges of certain amino acids, for the formation of which vitamin C is one of the important ingredients (Grodzinsky 1983; Fleischmajer et al. 1990; Currier & Nelson 1992; Brils et al. 1999a, b; Aaron & Bolander 2005; Van den Berg 2010).

Structure of the collagen network

The three-dimensional organization of the collagen molecules, from which the fibrils and fibers are formed, is geared appropriately to its mechanical stress. Changes in tissue shape lead to electrical voltage changes. Molecules use this piezoelectrical activity to organize the architecture of the tissue.

On the one hand, if the stress is always carried out in the same way, the collagen fibers will always orientate themselves to the strength line and as a result run parallel. In this case, shaped, taut connective tissue is referred to. Formed connective tissue occurs in tendons, ligaments, aponeuroses, etc. If, however, the stress on the tissue always coming from different directions, it leads to an interlaced lattice effect. This is known as unformed taut connective tissue. This is found in the capsules, fascia, and in the intraneural and intramuscular connective tissue.

Ground substance is found between the crossing collagen fibers. The water bound to it provides for friction-free movement of the fibers against each other. Under pathological circumstances – such as with a loss of ground substance – the collagen fibers get closer to each other and form so-called pathological cross links. These pathological connections reduce the ability of the collagen network to unfold. When examining patients we detect restricted movement (Akeson et al. 1973, 1977, 1987, 1992; Brennan 1989; Van den Berg 2010).

In order to loosen the pathological cross links in the tissue, the therapist mobilizes activity with intermittent extension stimuli. This stimulates the fibroblasts to increase synthesis (+200%) of collagenase, an enzyme that breaks down the pathological cross links again (Carano & Siciliani 1996).

In relaxation, collagen fibrils and fibers are normally in a wave-shaped form. This prevents the tissue reacting to stress too quickly and too explosively. The faster the stress has an effect on a structure, the greater it is. The physical law according to which force is the product of mass per acceleration $(F = M \times V)$, expresses this association. The wave shape is caused on the one hand by elastic fibers and on the other possibly by, for example, the myofibroblasts available in the fascia.

Elastic fibers

Elastic fibers are predominantly found in loose connective tissue, in elastic cartilage (external ear and tip of the nose), in the skin, the vascular wall, and also in tendons and ligaments. Some ligaments, such as the Lig. flavum of the vertebral column, are almost exclusively constructed of elastic fibers. The substance elastin, which is found in great quantities in elastic fibers, is yellowish in color. This results in the yellow color of the relevant structures. The part of the vessels made up of elastic fibers amounts to about 50%. In other connective tissue forms, such as the skin and tendons, it is only about 2 to 5%. Elastic fibers are produced in the endoplasmic reticulum of the fibroblasts and the smooth muscle cells.

Structure

Elastin is a structure protein with the same amino acid content as collagen. We differentiate between alpha elastin, which consists of about 27 protein chains each with about 35 amino acids, and beta elastin which has only two protein chains each with about 27 amino acids. Only 10% of the protein chains form a helix.

Elastic fiber microstructure

Elastic fibers consist of an amorphic mass of elastin which is surrounded by elastic microfibrils. The microfibrils serve to orient the elastin in the formation of the elastic fibers. Elastic fibers are very branching and have many connections to each other. This creates the net-like structure. They can extend by 100 to 150%. As they extend, they store potential energy in order to be able to return to their original form after stress. Three stages have been distinguished in the synthesis of elastic fibers: Oxytalan fibers, which occur only as elastic microfibrils; elaunin fibers, i.e., elastic microfibrils with small quantities of elastin; and fully formed elastic fibers. The breaking strength of the elastic fibers is about 300 N/cm². (Grodzinsky 1983; Fleischmajer et al. 1990; Leadbetter et al. 1990; Currier & Nelson 1992; Aaron & Bolander 2005; Van den Berg 2010).

Ground substance

Ground substance consists of GAGs and PGs and aggregates. GAGs exist both in the intracellular and extracellular space. The PGs and PG aggregate bind cells, collagen, and elastic fibers and bind themselves to water.

Structure

PGs occur if GAGs are bound to a protein chain. The PG gains a characteristic stretched form as a result of the strong negative loading of the GAG, because of which the molecules repel each other and seek the greatest possible distance from each other. This can be compared with the shape of a toilet brush. For PG aggregate to occur, many PGs must be bound to a central hyaluronic acid chain. Binding or link proteins are required for this.

Microstructure of a PG

The central protein chain of a PG contains more than 2000 amino acids.

The outer 60% of this chain is used as the connection site for about 80 to 100 chondroitin sulfate chains. The remaining 10% is used for about 50 to 60 keratan sulfate chains. Occasionally, a few other shorter oligosaccharide chains can be bound. The remaining 30% remain free. They are used for the binding of the PG to a hyaluronic acid chain using a connecting protein. Covalent binding causes the binding of the binding protein.

The GAGs which occurs in the connective tissue are: hyaluronic acid, chondroitin 4 sulfate, chondroitin 6 sulfate, dermatan sulfate, keratan sulfate, heparan sulfate, and heparin.

Functions

PGs and GAGs have many different functions. First, they stabilize the connective tissue by binding to collagen and elastic fibers, cells and water. They predominantly absorb forces that affect the unformed tissue and protect the collagen network from excessive stress. The other important task of the ground substance is the absorption of compression force on cartilage, intervertebral discs in the nucleus area, etc. (Buckwalter et al. 1988; Eyre et al. 1989).

The heavy negative loading of the GAGs and PGs causes the capacity to bind to water and because of its viscoelasticity allows the tissue to return to its original form after stress. The binding of water also enables the collagen fibers to move without friction against each other. The stored water simultaneously serves as a transport route for nutrients and waste products as well (Grodzinsky 1983; Fleischmajer et al. 1990; Currier & Nelson 1992; Aaron & Bolander 2005; Van den Berg 2010).

Besides elasticity and stability, GAGs and PGs also have a barrier and protective function. They form a type of sieve for large molecular structures from the vessels that could penetrate the tissue. They also protect the tissue and the cells from penetrating bacteria. Bacteria can only move through the dense network and penetrate the tissue with difficulty. Only if they excrete the enzyme hyaluronidase, which breaks down the matrix, can they penetrate deeper into the tissue where they then must be deterred by the immune system (Van den Berg 2005a).

Finally, GAGs loosen the chromatin structure within the cell nucleus so that the DNA is better and more easily detectable. In immune system cells a GAG binds to proteases, that is to enzymes which break up the proteins and prevent the cells destroying themselves.

The synthesis of hyaluronic acid chains, protein chains and some oligosaccharides takes place in the endoplasmic reticulum of all connective tissue cells. Other oligosaccharides and GAGs are formed in the Golgi apparatus of the cell. The turnover of hyaluronic acid is about 2 to 4 days; for the other sulfated GAGs it is about 7 to 10 days. This means that the cells must always remain synthetically active, otherwise there is the risk of a reduction in the quantity of ground substance. The residual products of GAGs, which can be released outside the cells, have a feedback effect on the cells and this controls synthesis. Even the mechanical distortion of the cells itself represents a stimulus to synthesis.

Noncollagen proteins

Link proteins count as noncollagen proteins. In the various tissues you can find very many different link proteins such as: Fibronectin, laminin, chondronectin, osteonectin, osteopontin, osteocalcin, decorin, tenascin R and C, anchorin DII, thrombospondin, integrin, vinculin, talin, vibronectin, α -actin, etc.

Their primary task is the binding of the collagen fibers to the cell membrane. This is how mechanical strain application to the tissue is transferred to the cell membrane. As already noted, the mechanical shaping of the cell membrane controls cell synthesis – i.e., extracellular signals regulate the intracellular activity via the interlinking proteins. Other specific functions of individual link proteins are the capacity of fibronectin to guide mobile cells through the connective tissue. The link proteins vinculin, spectrin, and actomyosin control the activity of the cell nucleus, the mitochondria, and the Golgi apparatus from the inside of the cell membrane. During the aging process, the quantity of link proteins increases and correspondingly reduces the mobility of the connective tissue.

Link proteins bind PGs on hyaluronic acid chains to PG aggregates in the ground substance; see Fig. 4.3.2 (Grodzinsky 1983; Fleischmajer et al. 1990; Currier & Nelson 1992; Aaron & Bolander 2005; Van den Berg 2010).

Water

The human body consists of up to about 60 to 70% water. About 70% of this is found intracellularly and 30% is extracellular. Up to 67% of the extracellular water is present between the cells as interstitial fluid and up to 20% is a component of the blood in the vessels. The remaining quantity (13%) is transcellular in the cerebrospinal fluid, in the axoplasmatic liquid in the nervous system, in the eyes, joints, abdominal cavity, etc.

Function

Water serves as a means of transport and as a solvent; it reduces friction and acts as a heat buffer. It allows oxidation and reduction – 99% of chemical reactions in the body require water – and it lends the tissue volume and thus has mechanical functions. The water



Fig. 4.3.2 • Fibroblast and extracellular components. From Krstc, 1988, with permission.

biosystem allows the optimum conveyance of energy and information as a result of the ratio between free and pseudocrystalline water at a temperature of $37 \,^{\circ}$ C. Interestingly, at this temperature, the ratio of pseudocrystalline water is exactly equal to that of free water (Van den Berg 2005b). As we know, the human body consists of up to about two thirds of water. We must correspondingly assume that subtle changes in the constitution of this internal water may have large effects on our bodies and well-being.

From the point of view of anatomy and physiology, we know that water in the form of synovial fluid acts as a layer of lubricant between joint surfaces. This means there is no resistance during movement and friction is eliminated. There are also synovial covering cells on the outside of tendons, ligaments, nerves and fascia, which permanently produce fluid that resembles synovial fluid. These structures too can move against other anatomical structures without friction.

If perfusion is reduced – think of increased sympathetic reflex activity caused by pain – the fascia fluid produced is reduced. This is, in turn, demonstrated as disrupted mobility or increased resistance.

Summary

Matrix and cells are found in a continuous dialogue and are dependent on each other. The matrix protects the cells against mechanical forces that are too great.

The forces on the network of collagen and elastic fibers and ground substance are transferred to the cell membranes via link proteins.

The cell is informed by these signals and is stimulated to keep synthesizing new matrix components. This rebalances the physiological breakdown of the matrix and the tissue retains its stability and mobility. A reduction in the load stimulus leads as a result to reduced synthesis activity of the cells and thus to a loss of matrix components.

This produces a lower level of stability and limited mobility because of the formation of pathological cross links. An important task for the therapist is to apply gradually increasing levels of force without causing pain, in order to promote the healing and regeneration processes and in this way restore mobility and stability.

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The influence of pH and other metabolic factors on fascial properties

4.4

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pH regulation and influence on fascial tissue

Intra- and extracellular pH is one of the main determinants of all biochemical reactions in the body. The name relates to the Latin expression *potentia Hydrogenii* and is a measure for the strength of acidity, i.e., concentration, of protons (H^+) . The organs, the immune system, coagulation, and all other systems of the body need to work in a specific microenvironment with a pH-optimum. In blood, the normal range of pH is very tight and has to be between 7.36 and 7.44 for ideal conditions. Under pathophysiological conditions, with a blood pH lower than 7.0 and higher than 7.7, there is a high probability for the individual to die due to organ dysfunction.

The kidney and the lungs work together to help maintain a blood pH of 7.4 by affecting the components of the buffers in the blood. To a lesser extent, acids are egested by skin, liver, and bowels. In the body there are at least three buffer systems: first, the most important is the carbonic acid–bicarbonate buffer; second, the phosphate buffer, which plays a minor role; and third, the hemoglobin protein, which can reversibly bind either H^+ or O_2 . During exercise, hemoglobin helps to control the pH of the blood by binding some of the excess protons that are generated in the muscle.

How does the most important buffer system, the carbonic acid–bicarbonate buffer work, and what are the influences of lungs and kidney on this buffer system?

Acid-base buffers in the blood keep the pH constant when hydrogen ions or hydroxide ions are

added or removed. An acid-base buffer typically consists of a weak acid, and its conjugate base. When protons are added to the solution, some of the base components are converted to the weak acid; and when hydroxide ions are added, protons are dissociated from the weak acid molecule. As mentioned above, the most important buffer is the carbonic acid-bicarbonate buffer in the blood. The simultaneous equilibrium reactions of interest in the blood are as follows:

$$H^+ + HCO_3^- \rightleftharpoons H_2CO_3 \rightleftharpoons H_2O + CO_2$$

(H⁺: hydrogen ion, HCO_3^- : bicarbonate ion, H_2CO_3 : carbonic acid, H_2O : water, CO_2 : carbon dioxide.)

Furthermore, the Henderson–Hasselbach equation gives an idea how this buffer system works:

$$pH = pK - log([CO_2]/[HCO_3^-])$$

The pH of the buffer solution is dependent on the ratio of the amount of the partial pressure of CO_2 and the HCO_3^- in the blood. This ratio is relatively constant, because the concentrations of both buffer components are very large, compared with the amount of H⁺ added or removed in the body during normal circumstances. Under heavy exercise or pathophysiological situation, the added H⁺ protons may be too great for the buffer alone to control the blood pH. When this happens, another organ must help to maintain a constant pH in the blood.

Figure 4.4.1 gives an idea how the most important organs, the kidney and lungs influence the bicarbonate–acid buffer. Physical exercise, for example, leads to a significant increase of acidic metabolites such as lactate and CO_2 due to glycolysis and the cellular respiratory chain. An increase of PART FOUR



Fig. 4.4.1 • Influence of lung and kidney function on the carbonic acid– bicarbonate buffer. Metabolic acidosis is counteracted in the short run by hyperventilation and thereby increases CO₂ elimination. In the longer run the kidney increased H⁺ secretion and reabsorption of bicarbonate (HCO₃⁻). Metabolic alkalosis is counteracted by hypoventilation and thereby decreased CO₂ elimination. Respiratory acidosis is reversed by an increased H⁺ secretion and reabsorption of (HCO₃⁻) and vice versa in respiratory alkalosis.

the partial CO_2 pressure in the blood, measured in the brain stem and in peripheral chemoreceptors located in the aortic arch and carotid arteries, is the most dominant breathing stimulator for the breathing center located in the brain stem. Activation of the breathing center leads to deeper and faster breathing, thereby increasing ventilation. Thereby CO₂ can be eliminated, which helps to keep the blood pH constant. Furthermore, under pathophysiological conditions - like in septic patients with a metabolic acidosis (pH < 7.3, and low buffer base) – breathing is often enormously activated to counteract the metabolic acidosis. Instead, patients with a metabolic alkalosis (pH > 7.5, and high buffer base), for example due to excessive vomiting, may show a depression of respiration with an increase of partial CO₂ pressure in the blood to keep the pH in the physiological range. However, this compensation mechanism is limited, because hypoventilation is only tolerated in narrow ranges. The kidneys provide a mechanism of saving basic metabolites by regeneration of the bicarbonate (HCO_3^{-}) and secretion of H⁺. Moreover, the kidney is important in respiratory diseases, like chronic obstructive pulmonary disease, where the patients suffer from a chronic high partial CO₂ in the blood, which induces a so-called respiratory acidosis (high CO2, normal bicarbonate at the beginning). The kidney has the ability to counteract this acidosis by regeneration of more bicarbonate and an increase of acid secretion with the urine. In contrast to the lung, the counteraction of the kidney to keep the pH constant is slower.

Taken together, the carbonic acid-bicarbonate buffer is the most important buffer for maintaining

acid-base balance in the blood and is mainly influenced by the kidney and lungs. This means, on the other hand, that in patients with lung and/or kidney disease it is important to control pH levels.

Chronic hyperventilation - also called "breathing pattern disorder" - tends to lead to a state of reduced CO_2 in the blood, known as hypocapnia. Hypocapnia goes along with increased alkalosis and leads to vasoconstriction as well as to increased nerve and muscle excitability. Hypocapnic alkalosis has been observed in anxiety as well as in other negative affective states and traits (Chaitow et al. 2002). Reversing sustained or spontaneous hyperventilation with therapeutic capnometry has proven beneficial effects in the treatment of panic disorder as well as asthma (Meuret & Ritz 2010). Interestingly, patients with psychogenic hyperventilation frequently show elevated lactate levels. While high lactates are usually associated with acidosis, it has recently been shown that in patients with psychogenic hyperventilation this correlation is not valid, due to adaptation processes (Ter Avest et al. 2011).

In this connection, it is very interesting that psychiatric disorders like panic disorders (PD) with chronic or acute hyperventilation may have an influence on fascial function. For example, it has been shown that patients with PD have a significantly, higher incidence of joint hypermobility syndrome (Martin-Santos et al. 1998). In another study, patients with PD suffered significantly more often with prolapse of the mitral valve, also indicating more lax connective tissue (Tamam et al. 2000). However, there are other studies that could not detect any significant relationship between PD and joint hypermobility syndrome or mitral valve prolapse (Gulpek et al. 2004). Furthermore, the exact pathomechanism behind these findings is still unknown. Genetic studies, for example, have looked at elastin polymorphism and could not find any association with PD (Philibert et al. 2003). It is known that patients with PD often have electrolyte disturbances, like hypophosphatemia (indicator of chronic hyperventilation), elevated lactate levels, and increased CO_2 sensitivity (Sikter et al. 2007). Whether these electrolyte disturbances may be explanations for the higher incidence of lax connective tissue in patients with PD has to be proven in the future.

Acid-base status is strongly linked with the electrolyte balance in the cells. Protons (H^+) and potassium (K^+) are the cations to which resting cellular membranes are permeable. This is the reason why H⁺ and K⁺ supersede each other in order to hold an electrochemical equilibrium across the membranes. However, the effect of acid-base status on the serum potassium is very complicated and depends on the nature of the disorder. In general, extracellular K⁺ concentration rises in acidic conditions and falls in alkalosis. This is important because K⁺ is stabilizing the resting membrane potential and K⁺ deviations can lead to heart arrhythmia, for example, and muscle fatigue. However, exercise can lead to a significant increase of extracellular K⁺ in the muscle. Indeed, K⁺ levels rise significantly after extensive physical exertion. Emergency medics need to treat cardiac arrhythmias in almost every marathon event, where of course factors other than high K⁺ contribute to heart instability. The K⁺ accumulation in the muscular microenvironment (i.e., within the transverse tubular system) has also been linked to muscle fatigue, which is counteracted by simultaneous elevation of muscle temperature, lactic acidosis, and the presence of endogenous catecholamine (Pedersen et al. 2003).

Not only global pH maintenance can be disturbed, but also a specific region in the body can be affected by local accumulation of acids. This is the case in inflammations, and for local acidosis it is not relevant if the cause is traumatic, infectious, or autoimmune.

What is the impact of pH on fascial function?

Until now there have not been sufficient studies on the influence of pH on fascial function. However, Pipelzadeh and colleagues were able to demonstrate in the superficial fascia of the lower dorsum of rats, when superperfused with lactic acid containing Krebs-solution (pH 6.6), that the contractions of the myofibroblasts induced by adenosine or mepyramine were significantly increased (Pipelzadeh & Naylor 1998). In contrast, alkaline conditions had no influence on the agonist-induced contraction of the myofibroblasts. It should be noted that this study used a small sample size only and that their results have not yet been verified (or falsified) by additional examinations. However, the authors conclude that this pH-phenomenon could be an important factor in wound contraction and healing beside other factors like growth factors, etc.

Growth factors

Collagen is produced in tendon fibroblasts, which are arranged in parallel along the main direction of tension. It is well known that the tendon fibroblast is considered a key player in tendon maintenance, adaption to changes in homeostasis, and remodeling in the case of disturbances to tendon tissue. Furthermore, fibroblasts are the major mechanoresponsive cells in the tissue (Kjaer et al. 2009). Induction of collagen expression in response to increased loading has been demonstrated in many cell and tissue types, and has been suggested to depend on a mechanically induced expression of collagen-inducing growth factors. These growth factors are then thought to work in an auto/paracrine manner to induce extracellular matrix protein (ECM) production (Kjaer et al. 2009). Several growth factors which stimulate collagen synthesis are expressed in response to mechanical loading. The most important ones are transforming growth factor- β 1 (TGF- β 1), connective tissue growth factor (CTGF), and insulin-like growth factor-I (IGF-I). Importantly, in human ligaments the loading induced collagen I and III expressions appear to depend directly on TGF-B1 activity (Nakatani et al. 2002). In conclusion, the named growth factors seem to play a very important role in tendon adaption in response to mechanical loading, by the induction of an increased collagen expression in fibroblasts.

It is, furthermore, interesting that hindlimb suspension (immobilization of the back legs) for 14 days in rats shows a dramatic muscle mass decrease in the soleus muscle and has almost no effect on tendon mass or expression of collagen I and III, TGF- β 1, and CTGF either in tendon or muscle. This means that the general response of tendon to unloading does not appear to follow a pattern opposite to that of the loading response. This could indicate that tendon tissue is protected from rapid changes in tissue mass during unloading, while muscle, which is known to act as a protein store, is subjected to substantial and fast changes in tissue mass (Kjaer et al. 2009).

Sex hormones

Estrogen receptors have been localized in synoviocytes in the synovial lining, fibroblasts in the anterior cruciate ligament (ACL), and cells in the blood vessel wall of the ligament (Liu et al. 1997). A more than 40% reduction in collagen synthesis and a reduction in fibroblast proliferation was observed in vitro in tissue samples from the ACL when estradiol was administrated in physiological doses (Liu et al. 1997). The authors conclude that this may be one explanation for the greater risk of women compared with men for certain kinds of injury in, for example, cruciate ligament rupture and disease of collagen-rich tissue. Furthermore, women with chronic intake of oral contraceptive (OC) have a higher risk of lower back pain, bone fracture, persistent pelvic pain, and pelvic joint instability. An explanation for this phenomenon was given in an in-vivo study, which demonstrated a lower exercise-induced increase in tendon collagen synthesis in women with high synthetic estradiol serum levels (HE-OC) from the intake of OC, compared with the women with no OC intake (LE-NOC) (Hansen et al. 2008). Furthermore, serum and the interstitial peritendinous tissue concentration of IGF-I and IGF-binding proteins showed reduced bioavailability in HE-OC compared with LE-NOC. Taken together, this study indicates that estradiol either directly, or indirectly via reduction of IGF-I, inhibits exercise-induced collagen synthesis (Hansen et al. 2008).

Relaxin

Relaxin is a dimeric peptide hormone that is structurally related to the insulin family of peptides. It was discovered nearly 80 years ago and was found to be mainly produced in the ovary and placenta during pregnancy and so initially regarded as a hormone of pregnancy. Humans have three relaxin genes, H1, H2, and H3, where H2 relaxin is the major circulating and stored form of relaxin (Samuel 2005). The most consistent biological effect of relaxin is its ability to stimulate the breakdown of collagen. It acts on cells and tissue to inhibit fibrosis, the process of tissue scarring which is primarily the result of excessive collagen deposition (Samuel 2005). Fibrosis itself is a universal response to chronic injury and inflammation of several organs and manifests as an excess accumulation of connective tissue. This results in an irreversible loss of tissue function. like in hepatic cirrhosis, pulmonary fibrosis, or renal fibrosis. The primary receptor on which relaxin influences collagen turnover is the LGR7 receptor (Samuel 2005). Relaxin gene knock-out (RLX-/-) mice demonstrated an age-related progression of interstitial fibrosis in the lung, heart, and kidneys, leading to organ damage and dysfunction (Samuel et al. 2005a). Furthermore, the RLX-/- mice developed a progressive scleroderma. This was shown by an age-related progression of dermal fibrosis and thickening in male and female RLX - / - mice, associated with marked increases in types I and III collagen (Samuel et al. 2005b). These data provided the first evidence that relaxin is an essential mediator of collagen turnover and protects several organs against fibrosis (Samuel et al. 2005a, b). Furthermore, it is interesting that administration of human recombinant H2 relaxin could reverse the organ fibrosis in RLX-/- mice. With those data and other studies, relaxin has emerged as a potential antifibrotic agent for the future in different diseases. The future will show if relaxin could also be a potential drug for fascial contractures induced by fibrosis, for example in chronic inflammation.

Corticosteroids

Overload tendon injuries are frequent in recreational and elite sports. One common treatment option is the local application of corticosteroids to diminish local inflammation and to reduce pain. However, the direct effects and side effects of corticosteroid administration on the tissue are not fully understood. In rats, the injection of methylprednisolone in the tail tendon significantly reduced tensile fascicle yield strength compared with the NaCl-injected rats (control group) (Haraldsson et al. 2009). Another group speculates that corticosteroid injection affects in some way the components of ECM and the mode by which these components contribute to the tensile strength of the fibril (Fratzl et al. 1998). Furthermore, corticosteroids should reduce decorin gene expression and inhibit the proliferation and activity of tendon tenocytes, leading to suppression of collagen production (Chen et al. 2007). Additionally, tendon cell migration, which is fundamental for tendon healing, is delayed after corticosteroid injection (Tsai et al. 2003). These corticoid-associated disturbances of tendon cell metabolism may affect the structural integrity of the tendon and weaken its mechanical properties.

However, the analgesic effects of corticosteroids in athletes with chronic pain both in the upper and lower extremities have been reported in many studies. The exact mechanism behind this pain relief effect of corticosteroids in tendinopathy remains elusive. However, the comparison of pain relief for patients with tennis elbow either receiving corticosteroid injection or physiotherapy favors the longterm outcome of physiotherapy (Bisset et al. 2006).

Taken together, corticosteroids are still widely used and accepted drugs in tendinopathy. However, inconsiderate injection of corticosteroids in tendon injuries or chronic pain in fascial structure should be avoided.

Lactate

Until the early 1970s, lactate (HLa) was largely considered a dead-end waste product of glycolysis due to hypoxia, the primary cause of the O_2 debt following exercise, a major cause of muscle fatigue, and a key factor in acidosis-induced tissue damage (Gladden 2004). This paradigm has shifted in the last 40 years.

Lactic acid is more than 99% dissociated into La^- and H^+ at physiological pH. During exercise

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and muscle contractions muscle and blood La- and H⁺ can rise to very high levels. The evidence of many experimental studies indicated that elevated H⁺ could depress muscle function by various mechanisms, for example by inhibiting maximal shorting velocity and myofibrillar ATPase, inhibiting glycolytic rate, and reducing Ca²⁺ re-uptake by inhibiting the sarcoplasmatic ATPase (leading to subsequent reduction of Ca²⁺ release) (Gladden 2004). However, in recent years studies have demonstrated that increased H⁺ under physiological temperatures does not have such dramatic negative effects on muscle function (Bangsbo et al. 1996). Furthermore, it has been shown that lactic acidosis protected against detrimental effects of elevated external K⁺ on muscle excitability and force (Nielsen et al. 2001). In the 1990s, La⁻ itself was considered to play some role in muscle fatigue. However, more recent studies have demonstrated only a minimal effect of La⁻ on muscle fatigue. Furthermore, it is very interesting that La⁻ is considered to play an important role in wound healing by enhancing collagen deposition and angiogenesis (Trabold et al. 2003). La⁻ is considered to stimulate collagen synthesis by an increase of collagen promoter activity leading to an increase of procollagen messenger RNA production and collagen synthesis. In the case of La⁻-stimulated angiogenesis in wounds, the major pathway appears to be enhanced vascular endothelial growth factor (VEGF) production in macrophages (Trabold et al. 2003).

In summary, La⁻ is no longer only a "bad guy" and has important functions in wound healing, by first enhancing collagen production in fibroblasts and, second, improving angiogenesis by an increased secretion of VEGF from macrophages.

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Fluid dynamics in fascial tissues

Guido F Meert

It is easy to forget that the concentration quotients of salts (NaCl, KCl, CaCl₂) in interstitial fluid and in water of an ocean are nearly identical. Our cells are, in a manner of speaking, swimming gel-like structures in an ocean of interstitial fluids, and we are carrying that ocean around with us.

Connective tissue consists of cells (fibroblasts and leukocytes), interstitial water, fibers (collagen and elastin) and matrix molecules (glycoproteins and proteoglycans). Interstitial fluids create a transport space for nutrients, waste materials and messenger substances and actually facilitate homeostasis between the extracellular and the intracellular region. In addition, the lymphatic system filters his supply out of the ocean of interstitial fluids and drains it into the venous system.

Recent research into connective tissue has produced many interesting results. Schleip and colleagues investigated the contractility of fascial tissue, a very exciting aspect for manual myofascial therapy (Schleip et al. 2005). We also know now, that all the cells of the human body are connected with each other via the connective tissue and are building an ingenious tensegrity-like construction. By mechanotransduction mechanical signals are being transduced to the nucleus and other organelles of the cells and even open the way to genetic "adjustments" (Ingber 2006).

We should search for answers as to how the living connective tissue and cytoplasm differs from a simple non-living mixture of the same chemical constituents in solution!

Properties of interstitial water

The structure of water is not completely understood. Formed from tiny molecules, water is very versatile. Szent-Györgyi (1975) called water the "matrix of life" and it interacts with cells and molecules in complex, subtle and essential ways.

In the human body, and therefore also in connective tissue, we are dealing with interfacial and bulk water, and the interfacial water seems to interact with the protein function (Bellissent-Funel 2005). Water molecules seem to build "polyhedrons". According to Chaplin water molecules are linked by hydrogen bonds in an expanded and coordinated tetrahedral arrangement (Chaplin 2004). According to this hypothetical model water at the smallest scale could be thought of as an equilibrium between two water tetramers: a) a collapsed structure A, held closely by non-bonded interactions, forming a more dense structure; b) a non-collapsed structure B, with molecules held further away and linked by hydrogen bonds to form a less dense structure. The equilibrium is then easily affected by the presence of solutes and surfaces and also by changes in temperature and pressure.

This model also proposes, that although the natural structuring in water at ordinary temperatures tends towards the "collapsed" structure A, the low density structure B can grow to form larger non-crystalline clusters based on dodecahedral water cluster cores, similar to those found in the crystalline "clathrate hydrazes"; as for example, the extensive icosahedral $(H_2O)_{280}$

aggregate built up from tetrahedrally hydrogen-bonded water molecules surrounding a dodecahedron made up of 20 water molecules, the basic clathrate cage (Chaplin, 2006). It seems suitable to ask, if we underestimate the importance of water in cell biology. Further investigation into this dimension is indeed warranted. Rearrangements of water molecules are ultrafast (Fayer et al. 2009). Simplified, the icosahedrons are presented in two states; a low-density expanded water-icosahedron and a high-density collapsed watericosahedron. Water molecules are able to convert bbetween the expanded and the collapsed version, without breaking the hydrogen bonds (Chaplin 2004).

Water is also able to form large regions of so called "structured water" or "liquid crystalline water". In structured water, water molecules move together, like a shoal of fish, without losing mobility. Liquid crystalline water has special features; namely a greater molecular stability, a negative electrical charge, a greater viscosity, molecular stringing together and the ability to absorb certain spectra of light (Pollack 2002).

Water in bulk seems to behave differently from water in confined spaces, but more research is needed into this (Ye et al. 2004). Water seems to have a fourth phase, beside gas, liquid and solid and that occurs at interfaces (Pollack 2002). It is surprising that the presence of an interface is more important for the dynamics of the hydrogen bonds than the chemical nature of the interface (Fenn et al. 2009). In the human body, fascial sheets, fibers, cell membranes, molecules and so on are building bigger and smaller interfaces with a hydrophobic or hydrophilic character for the interstitial fluids. Research reveals that water inside nanotubes appears to build "water cylinders" which allow protons to jump ultrafast. Biochemical reactions take place in confined spaces with interfacial water, comparable to nanotubes, at the surface of proteins, membranes, etc.

There is a great affinity between the polymers and the water molecules of the cell, which condenses the gel into a compact structure and enables the cell to move or to open ion-channels without breaking down. The extracellular matrix (ECM) builds also a gelatinous fiber network and "binds" the containing water.

There are three "populations" of water molecules in contact with collagen fibers (Peto & Gillis 1990):

- Water, bound within the triple helix of the collagen molecules.
- Water molecules, bound on the surface of the triple helix or bound with matrixmolecules (proteoglycans, glycoproteins, glycosaminoglycans).

• "Free" water in the space between the fibrils and fibers.

I would like to emphasize that flowing of this interstitial water happens in all directions between the cell–matrix interface. I will refer to the interstitial flow later.

Morphologic quality of interstitial fluids

The molecules and fibers of the ECM determine the properties of the interstitial gel. Furthermore fibroblasts, matrix molecules, enzymes and enzymeinhibitors regulate the composition of the gelatinous ground substance of the connective tissue. This is important, while the composition of this interstitial matrix determines the transport for nutrients and waste materials between capillaries and parenchymal cells, as well as the mechanical properties of the connective tissue.

The German anatomist and embryologist Blechschmidt (2004) found that the movement of microscopic particles ocurs in an ordered manner and has a kinetic aspect, which he called "metabolic movements". The flow of water, nutrients and waste materials lead to a canalization in the inner embryologic tissue and helps to form blood vessels. By stowing and condensing catabolites build the ground substance of the inner embryologic tissue. In "dilatation fields", by condensation of catabolites, water tends to flow toward this tissue and pushes cells apart. On the other hand, when water is pushed out of the embryologic tissue, a "densation field" develops and the cells pack closely together. The flow of fluids develops creative morphologic forces to help in forming the embryologic tissues It seems that the flow of water plays even some role in the folding of proteins and there seems to be interaction between the shell of water surrounding the proteins and the shape and characteristics of those proteins; therefore water "tunes" the way proteins, the building blocks of life, are functioning (Ball 2008).

Proteins have to fluctuate in order to work and there are two types of fluctuation. The α -fluctuations proceed in the bulk of water, surrounding the protein and β -fluctuations take place in the shell of water (two layers) around the protein (Frauenfelder et al. 2009). Biochemistry has to deal with the interactions between the molecule and its environment. The environment for molecules (cytokines, neurotransmitter, hormones, growth-factors...) released by cells is made up of interstitial fluids and the ECM.

Interstitial fluids as a medium of communication between the cells

Both collagens and water molecules have electric conductive and polarization properties, as do the matrix molecules. Polarization waves are possible, and protons can "jump" along the collagen fibers much faster than electrical signals can be conducted by nerves (Jaroszyk & Marzec 1993). The network of water molecules in the matrix network establishes an extraordinary, fascinating communication system.

Water molecules build dipoles and therefore water flow also means the flow of energy and information. It is therefore not a total surprise that some investigators suggest that chains of water molecules along collagen fibrils are acupuncture meridians (Ho 2008).

I think that the clue to the communication of connective tissue has multiple components:

- Mechanically: the tensegrity-building of the collagenous network with the geometry of fibers, matrix molecules and water molecules.
- Electrically and electromagnetically: the electron transport, the water bridges and hydrogen bonds with the ion-charges of dissolved substance and the hydrophobic and hydrophilic characteristics of biomolecules.
- Chemically: the interaction of the amino acids, carbohydrates, and fatty acids with their hormonal, neuronal, immunological, reparative and growth properties, and functions. The interstitial flow is an important driving force to enable the biochemical machinery.
- Energetically: liquid crystalline water is able to transmit signals and let information flow.

The "breathing" of the tissues

Fibroblasts exert tensile forces on collagen fibers of the ECM via integrins and thereby squeeze the ground substance. By decreasing their tension upon the collagen fibres, the ECM is allowed to take up fluids and to swell up (Reed et al. 2010). The squeezing of the ECM by the fibroblast–collagen network is stimulated, for example, by platelet-derived growth factor (PDGF-BB) or β 1-integrins, and the swelling of the ECM by relaxation of the fibroblast-collagen network is generated by pro-inflammatory cytokines. The major pro-inflammatory components seem to be prostaglandin E1 (PGE1), interleukin (IL)-1, IL-6 and tumor necrosis factor- α (TNF- α) (Martin & Resch 2009). There seem to be parallels between substances that decrease the interstitial fluid pressure (IFP) and substances that trigger the squeezing of the ECM, and substances that increase the IFP cooperate with substances that release the swelling of the ECM (Reed et al. 2010).

The IFP falls during acute inflammation within minutes and acts as a driving force for the formation of edema. That occurs because of the osmotic activity of the glycosaminoglycans (hyaluronan), which leads to a swelling of the interstitial matrix. The swelling of the tissue is balanced by the collagen network. Some cytokines (IL-1, IL-6, TNF- α ...) are able to lower the interstitial fluid pressure, others (prostaglandin F2 α , vitamin C...) manage to raise it.

Dynamic mechanical stresses and pressure gradients raise small fluid flows through the ECM of all the living tissues (Fig. 4.5.1). Because of the high flow resistance of the ECM, the interstitial flow is in all directions and flows much more slowly than the blood flow in vessels (Rutkowski & Swartz 2006). Although scientific investigation on interstitial flow is desperately needed, it is difficult to measure the interstitial flow in living persons.

It can be helpful to compare connective tissue with a sponge. By stretching or compressing the tissue, water is being extruded out of the connective tissue and makes the tissue more pliable and supple. After a while, water is resorbed and the tissue finds a new equilibrium. Manual therapies use this principle and squeeze and refill the tissue by pump- and soft tissue techniques (Meert 2006). On the one hand, by pumping the connective tissue therapists try to wash out pro-inflammatory substances and waste products. On the other, they attempt to dissolve adherences of the collagen network to enable the supply with oxygen and nutrients.

Breathing and the craniosacral rhythm can produce the same effects, milking and nourishing the ground substance in form of a dynamic balance between inspiration (swelling – straightening oneself – exorotation of the extremities) and expiration (detumescing – slumping down – endorotation of the extremities) with rhythmic "changing" of the interstitial pressure (Meert 2012) (Fig. 4.5.2). There are several body-rhythms (rhythm of the heart, of respiration, of peristalsis, of production of cytokines and hormones, of mental concentration or relaxation. etc.), which interfere with each other and ultimately produce a slow rhythm in the human body, unique for that person and that moment. One of the most fascinating rhythms that







Fig. 4.5.2 • Interstitial flow from a macroscopic view. From Meert G F Veno-lymphatische kraniosakrale Osteopathie. 2012 – Elsevier, München, with permission.

promotes this body- or tissue-rhythm, is the active vasomotion of lymphatics and vessels (Meert 2012). The author prefers to call this interference-rhythm the "tissue rhythm", because it seems that the tissues really make "breathing-movements".

Compression, traction, torsion, and shear stresses and also fluid shear stress exert forces on cells, receptors, and proteins. If the flow of interstitial fluids is decelerated, a contraction of the lymphatics and an increase of the frequency and amplitude of the active lymph pump (lymphatic vasomotion) is being induced (Gashev et al. 2002). Individual lymphangions are able to accommodate themselves independently to local changes of the flow of interstitial fluids (Venugopal et al. 2007). Endothelial cells of the vessels seem to be able to "feel out" the flow or the absence of flow of the interstitial fluids and react to it by secreting chemokines and several cytokines (Ng et al. 2004). By this interstitial flow, the cells seem to reveal the state of their environment and interact with cell migrations, cell differentiation, matrix remodeling and secretion of proteins and cytokines. Because proteins are too large to simply diffuse, flow in the interstitial spaces is necessary for the transport of those proteins from the blood to the cells, and vice versa. Investigations demonstrated that new capillary organization of endothelial cells occurred primarily in the presence of both vascular endothelial growth factor and interstitial flow (Helm et al. 2005).

It is one of the most fascinating experiences to learn to palpate, stimulate and channel those individual and subtle waves through the tissues of a patient. After breaking up fascial adherences by myofascial techniques, it makes sense to irrigate and purify the connective tissue. Finally, the battle between infections and the body's defenses is mostly fought in the connective tissue, leaving behind remains and fragments. The dynamics of the interstitial fluids seem to be an important key for normal tissue function and homeostasis and we can look hopefully to future research to decipher those mechanisms and bring some "fresh flow" in therapy and tissue engineering.

It is not for nothing that Andrew Taylor Still (1992) instructed us: "Let the lymphatics always receive and discharge naturally. If so we have no substance detained long enough to produce fermentation, fever, sickness and death."

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Fascia-related disorders: An introduction

Thomas W Findley

Part 5 presents a selection of disorders which are or may be fascia-related. It is not intended to be a comprehensive list, but rather to show a spectrum of types of diseases which may be encountered in the course of providing manual therapies. Each one illustrates different underlying physiological changes. Some chapters present local conditions, while others address systemic processes affecting multiple parts of the body. They are intended to stimulate thought and discussion regarding the role of fascia in manual therapies, among clinical practitioners and basic and clinical scientists.

Dupuytren's disease (Chapter 5.2) illustrates the contractile power that can be generated by the fascia. In the early stages, vertical fibers from the palmar fascia to the skin create indentations or "pits" where they attach; later on, palmar nodules develop between the palmar fascia and the skin. Longitudinal cords develop, particularly generating deformities in the little and ring fingers. There are surgical, local and systemic pharmacologic treatments directed at the mechanical connections or the underlying processes.

Chapter 5.3, Scleroderma, addresses autoimmune conditions that result in inflammation, scarring, and thickening of skin, superficial and deep fascia, blood vessels and lymphatics, and internal organs. This results in a much broader spectrum of changes than seen in the previous chapter. Patients with these conditions may have localized patches or lines of hardened tissues, or more systemic involvement affecting hands and feet, head and neck, gastrointestinal tract, lungs and heart. Conventional therapy directed toward the vascular or immune system or fibroblast proliferation can be complemented by manual therapy for specific components to restore mobility and pliability of tissues. Chapter 5.4 addresses the normal anatomy and biomechanical function of the plantar fascia and the clinical signs, imaging and histopathology in plantar fasciitis. The author proposes that normal physiological loading is not sufficient to create an overuse injury in normal plantar fascia. Rather, there is an imbalance of loading with tissue threshold for remodeling, from an underlying fascial defect which reduces tissue ability to accommodate load or from a neuromuscular defect which increases loading on otherwise normal tissues. Both of these conditions are seen in diabetes, as described, in the following chapter.

The "diabetic foot" (Chapter 5.5) is seen in almost 15% of persons with diabetes. Diabetes is a disease of small blood vessels in the body, affecting not only the pancreas but also nerves (hence "diabetic neuropathy") and peripheral skin. The elevated blood glucose levels lead to glycosylation of structural proteins in connective tissue, with fascial tissues becoming thicker and stiffer. Structural changes in the plantar fascia, Achilles tendon, and joint mobility of the lower extremity result in a constellation of changes which are termed the diabetic foot.

Frozen shoulder (Chapter 5.6) is a common condition, which progresses through freezing, frozen and thawing stages, also often associated with diabetes. Despite the defined clinical progression stages, the causes of primary frozen shoulder remain largely unknown, while numerous conditions can trigger the secondary form. Physical and pharmacological treatment varies according to the stage of the disease.

Myofascial trigger points (Chapter 5.7) are identified by manual palpation in everyday clinical practice in a wide variety of patients. Pathophysiological changes point to localized hypoxia with connective tissue shortening and crosslink, commonly induced by direct trauma, acute or chronic strain. Taut bands develop in conjunction with trigger points, resulting in restricted motion or coordination, decreased blood flow or sensation, or compression of neural or vascular structures.

While most of this chapter deals with conditions causing tissue shortening, Chapter 5.8 addresses the issue of tissue hypermobility seen in inherited connective tissue disorders such as Marfan and Ehlers–Danlos syndromes. Patients have muscle weakness, pain, and fatigability, with reduction in vibratory sense. Extracellular tenascin-X acts as a bridge between collagen fibrils and enhances stiffness of connective tissues. Deficiency in this seems to be directly related to the muscle symptoms in humans, and studies of a mouse model with tenascin-X deficiency show reduced epimuscular myofascial force transmission.

Finally, cerebral palsy with spastic paresis (Chapter 5.9) provides an example of a neurological condition affecting the ability to control and contract muscles, which may become atrophied, hypertrophied or fibrotic. Surgical and medical interventions have been developed to reduce muscle tone. Reviewing the muscle function, particularly during surgery to balance contraction power between wrist flexors and wrist extensors, has led to an appreciation of the role of the fascial connections between adjacent muscles, and even between muscles on opposite sides of a joint. These connections may vary a great deal from person to person. Further elucidation of these connections and how they form may lead to treatment plans more effectively tailored to the individual patient. A similar analysis may be useful in other neurological conditions such as stroke, multiple sclerosis, and spinal cord injury, which lead to increased tone in the muscles and have anecdotal reports of changes with manual therapies.

The current state of the art and science leaves therapists primarily relying on their palpation skills (seeChapter 6.2), coupled with an understanding of the underlying physiological processes, to design and direct therapy. This section illustrates a number of the physiological changes associated with both decreased and increased motion of fascial tissues.

The noninvasive imaging techniques described in Chapters 8.2 and 8.3 of this book have the potential to provide much more specific guidance to direct therapy and monitor tissue changes. Particularly important will be the development of ways to quantify the visual image, similar to what is now done in tissue elastography of the breast to detect more dense cancerous tissues. As these techniques become more widely used, the role of fascia in other conditions will become better documented and therapies will become more specific.

Two examples from my clinical practice illustrate the potential of musculoskeletal ultrasound. All images were taken with a new generation 12 mHz system (Terason T3000).

Case 5.1.1

A 47-vear-old woman was in a motor vehicle accident as a passenger in the right front seat. Five years after the accident she complained of continued lower abdominal pain and palpation showed that the area of maximum tenderness was located on a line across the abdomen 1 inch below the anterior superior iliac spine (the line of the lower edge of the seatbelt). Musculoskeletal ultrasound evaluation with a 12 Mhz probe showed a tear in the abdominal fascia correlating with the precise area felt by palpation. In this case the probe is oriented sagittally and the tear is evident near the right-hand side of the picture (see Fig. 5.1.1). Moving the probe laterally showed a straight line across the abdomen. In the images below, the image on the left is from the right side of the abdomen, the middle one from the middle and the one on the right from the left side. There is less fascia damage evident on the left side where the lapbelt is joined by the shoulder strap and hence forces are more diffused. The vertical line indicates the lower border of the lapbelt.

Case 5.1.2

A 48-year-old professional golf instructor with lateral epicondylitis (tennis elbow) had a 1-year history of pain, treated with cortisone injection, acupuncture, and physical therapy.

He had read about platelet-rich plasma injections and requested treatment (Mishra & Pavelko 2006; Peerbooms et al. 2010; Gosens et al. 2011). Ultrasound images taken during treatment document the location of injury and needle placement (see Fig. 5.1.2). The ultrasound shows fascial thickening at the lateral epicondyle, and there is a needle entering diagonally from the left side. In the right side, one can see the fluid injection, which consists of platelet-rich plasma containing growth hormone to speed fascial healing. After three injections spaced 2 weeks apart, with gradual resumption of activity, the patient became asymptomatic.



Fig. 5.1.1 • Musculoskeletal ultrasound evaluation with a 12 Mhz probe oriented sagittally. The image on the left is from the right side of the abdomen, the middle one from the middle and the one on the right from the left side. There is less fascia damage evident on the left side where the lapbelt is joined by the shoulder strap and hence forces are more diffused. The vertical line indicates the lower border of the lapbelt.

Fig, 5.1.2 • The ultrasound shows fascial thickening at the lateral epicondyle, and there is a needle entering diagonally from the left side. In the right image one can see the fluid injection as a white round area at the tip of the needle.



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Dupuytren's disease and other fibrocontractive disorders

Ian L Naylor

Introduction

Surprisingly, 188 years after Baron Dupuytren first described this condition of the hand, many fundamental details about the disease remain uncertain or unknown. What *is* known, however, is that it is a disease that affects the fascia in the hand and so it is usually classified as one of the fibro-contractive disorders. It is perhaps a very good example of the contractile power that the fascial elements within a structure can exert when they become "diseased".

This contractile disorder has received consideration in many textbooks. Highly recommended to the reader is the text written by Tubiana and colleagues in 2000. Despite being 10 years old, this textbook is still the most comprehensive approach to the disease and should be consulted for the detail that cannot be addressed in this short chapter. In contrast, this chapter will draw on and consider in some detail the more recent findings about this condition in an attempt to shed some light on the causes, consequences and possible treatments of this enigmatic disease.

Throughout history the use of the hand has been fundamental to man's development and many people have praised the hand for its "design" and abilities. One such person was the Englishman, Thomas Traherne (c.1637–1674,) who described the hands as:

The hands are a sort of feet, which serve us in our passage towards Heaven, curiously distinguished into joints and fingers, and fit to be applied to any thing which reason can imagine or desire.

Traherene: Meditations on the Six Days of Creation

In an individual afflicted by Dupuytren's disease, there is a usually a progressive decrease in their ability to carry out any tasks they could "imagine or desire" and they are brought to the point of severe frustration and annovance as the disease progressively and irreversibly develops, as ultimately the result can be a permanent deformity of the hand, with fingers firmly lodged again the palm, incapable of performing their everyday normal functions (Pratt & Byrne 2009). This, it is fully accepted, is the most extreme form of the disease, but when such a state is reached and sometimes even in those people who are suffering the less severe forms of the condition, then the tasks of trying to carry out "anything which reason can imagine or desire" become clearly very difficult and, in the worst case, impossible.

Who is afflicted by this disease?

Dupuytren's disease is primarily a disease of Northern Europeans, especially those who come from a Celtic or Scandinavian origin. In men, who are more likely to suffer this condition than women, it usually starts during the fourth or fifth decade of life, with a peak incidence at 50 years. In women, the peak incidence is 60–70 years. It does occur in other racial groups throughout the world but at a much lower incidence. The statistics do suggest, however, that there is not a uniformity of occurrence even within a specific Caucasian population. From all the evidence which has so far been collected there appears to be a hereditary component as the disease has a familial trait and autosomal dominance in some cases. Other suggested risk factors include Type 1 diabetes, alcoholism, smoking, antiepileptic drugs, occupations where physical forces are applied to the hand. Some of these factors are more controversial than others. This is just one of the many areas of uncertainty about the etiology of the disease which remain to be resolved.

A further complication which makes the disease even more complicated to understand is that not all individuals show the same rate of progression: usually, the later it starts the less aggressive it proves to be. The disease has an association with other fibrocontractive disorders such as Peyronie's and Ledderhose's disease (see below).

The basic problems of Dupuytren's disease

The beautiful delicacy, complexity and dexterity of the hand is brought about by the highly coordinated interaction of nerves, muscles, tendons, and bones, which just like everywhere else in the body are connected together with fascial elements. In the hand these elements are arranged in a very complex way and none more so than those that are found directly under the palmar skin. These elements are somewhat unusual since they form a thickened sheet of connective tissue, which is termed the palmar aponeurosis. This thickened layer of connective tissue is thought to provide physical protection for the flexor tendons which lie directly underneath it on their route to the fingers, so ensuring that despite stresses and loads placed upon the palm, the fingers can be manipulated. This ensures that these tendons are able to carry out their delicate and complex tasks free from constraints resulting from externally applied forces.

It is this connective tissue structure – the aponeurosis – which is considered to be central to the palmar problems of Dupuytren's disease. There is great speculation as to why this structure is affected by the disease and what factor(s) initiate the disease. There has also been speculation about what factors cause the condition to progress once it starts. It could be that these factors are the same or they could be different. Before speculating about these factors and what they could potentially do, it may be useful to give a brief outline of the condition, with special reference to the involvement of the fascial elements in the hand.

Basic anatomy of Dupuytren's disease

Many studies, from the perspectives of both basic anatomy (gross and microscopic) and clinical medicine, have established that the first palmar signs of Dupuytren's disease are the development of "skin pits", especially at the position of the distal palmar crease. These pits are caused by changes in the underlying vertical fibers of the aponeurosis attached to the overlying subcutaneous fascia of the skin. They are usually slow to develop and are due to progressive "stimulation" of the existing vertical connective tissue elements within the aponeurosis.

It should be stressed that the vertical fibers are fewer in number than the more extensive longitudinal and transverse arrangement of fibers within the structure, which provide skin anchorage when the palmar skin is subjected to shearing forces. So, the power of the vertical fibers to exert a force on the overlying skin is relatively low. However, once stimulated, these fibers do transmit a force and pits result from the contraction pulling against the more distensible skin rather than their anchorage points in the palmar aponeurosis.

This suggests that from the very beginning of the disease, the palmar fascia has changed from a highly complex, organized, passive protective structure to one which has developed the capacity to be contractile. Despite speculation, there is still no clear evidence as to why the vertical transmission of the contractile force that forms the pits should first occur. However, whatever the causes, the presence of such fibres as demonstrated in the micro dissection studies of McGrouther in 1982 is not in any doubt. McGrouther clearly demonstrated the vertical connections of the palmar aponeurosis to the skin at the site of the distal palmar crease.

Palmar nodules

The disease can then enter a very variable period. In some people, the disease does not progress from this stage or progresses very slowly. In others, there is proliferation of cells on the surface of the palmar aponeurosis and over time, as the number of cells increases, their mass is such that they can form a distinct, palpable and, eventually, externally visible, nodule full of cells (palmar nodules). It should be stressed that palmar nodules are superficial to the palmar aponeurosis and not an integral part of it. They exist between the palmar aponeurosis and the fascia of the overlying skin to which they can be adherent.

The proliferation of the cells in palmar nodules can be so pronounced that in times past when they were examined histologically they were sometimes diagnosed as malignant tumors (such as a sarcoma), resulting in amputation. Today, greater knowledge of the disease means such radical surgery is avoided. The proliferating cells are found in a meshwork of connective tissues of such a density that when they are bisected using a scalpel blade the term "gritty" has been used to give an indication of their great density, which is unlike that of the unaffected palmar fascia.

The condition gives rise to new connective tissue fibers and also new cells in a location where there should be no unnecessary connective tissue or additional cells. Biochemical studies have established that the newly formed collagen is a mixture of type III and type I, with collagen II being the predominant type associated with the nodules. The cells within the nodules have some of the functional characteristics of fibroblasts – synthesizing this new collagen – and ultrastructural similarities to some aspects of smooth muscle cells – giving rise to the contraction seen in the disease (Fig. 5.2.1).

The cells which develop within the palmar nodules have some rather unusual morphological characteristics when viewed using both optical and electron microscopy. They exhibit nuclear pleiomorphism using both techniques. With the greater resolution of the electron microscope, their cytoplasm contains myofilaments and there are also associated dense bodies. It is interesting to note that in 1972 Gabbiani and Majno used these ultrastructural appearances in such palmar nodules to develop further their



Fig. 5.2.1 • Dupuytren's contracture. From Forbes & Jackson, 2002, with permission.

ideas as to the existence and possible role(s) of myofibroblasts.

As the disease develops further and more nodular cells are formed, the greater contractile force generated by these myofibroblasts is transmitted to the rest of the aponeurosis along the longitudinal fibers. These consequently thicken and exert force on the proximal metacarpophalangeal joint (MP joint). Further disease progression results in palmar "cords" being formed, which may then spread their contractile effects to the MP joint and so cause deformity.

Palmar cords

The structures known as "cords" are histologically different from palmar nodules. They have fewer cells and relatively more collagen. These structures are said to cause more deformities of the fingers than palmar nodules (which only affect the MP joints). The cords are clearly abnormal collagenous structures which can be formed along the pre-existing fascia. One curious observation is that if the little finger is affected by the disease, resulting in a finger deformity which is seen as a flexion contracture of the proximal interphalangeal joint (PIP joints), this can actually occur without any palmar nodules being present. This again demonstrates how complex this disease can be, as it would be expected that palmar nodules were essential for the progression of the condition. This is simply not always the case.

Why are some fingers affected more than others?

The most commonly affected fingers are the little and ring finger. Why this should be so is unclear, as all flexor tendons to the fingers are potentially covered by the same aponeurosis. It would be of great interest to know whether there are structural variations in the palmar aponeurosis but as yet this has not been reported. This is not a criticism but a realization that there is a great difficulty in studying such a structure, as control samples for obvious reasons are almost impossible to obtain, except possibly in cases of trauma to the hand – and even then the damage caused may result in the normal architecture being deranged, resulting in false conclusions being reached. The problem is further compounded by there being no animal model for the disease. This is usually stated to be because no other species has a comparable structure and so no animal model is available to study this condition.

Are all myofibroblasts the same?

The problem of there being no animal model for the disease has caused a serious difficulty in attempting to test potentially new pharmacologic treatments in a rational way. Whilst it is true that myofibroblast can be "grown" in a variety of animal models, there is not one accepted method of doing this. Perhaps the most common technique is to use the croton oil induced granuloma pouch in the rat. In this technique, the croton oil is injected into an air blister made in the subcutaneous tissue space in the back of a rat. The croton oil clearly evokes a major inflammatory reaction and within 14–21 days causes a rapid and profound induction of myofibroblasts from the fibroblasts in the surrounding subcutaneous tissue space.

There is always the fundamental problem that the cells induced in the subcutaneous tissue space in rodents by this co-carcinogen, croton oil, may be different from cells induced by a fibrocontractive disease. These rapidly induced inflammatory stimulated cells may have potentially different receptors than those induced by the much slower development of a pathological process such as Dupuytren's disease. Whilst it is true the croton oil induced cells have all the ultrastructural features of myofibroblasts, whether or not they have the same receptors as the cells found in human conditions is a fundamental point, not as vet resolved. A further complication of extending basic research on myofibroblasts to Dupuytren's disease is that some of the more recent papers have used cells in culture derived from the lung. These are perhaps best termed "structural myofibroblasts"; they have all the structural characteristics, but whether such structural cells are comparable to disease-induced cells are in terms of their receptors remains for future research to evaluate.

This problem needs to be seriously addressed if rational treatments based on receptors are to be devised.

The lack of a suitable animal model has made progress in understanding both the initiation and progression of the disease a very slow task, further compounded by samples of the early form of the disease not being available for laboratory study, since operative procedures to remove tissue are not usually carried out until the disease is troublesome and requires some form of surgical correction; in other words, well after the disease has started. Using samples from later in the disease process presents other problems; for example, in the more developed cases regression of the cells may be present (a further complication because, although it may be useful to work out the types and properties of receptors present in cells undergoing regression, it is little help in establishing the initiation factors under study). As in many other diseases, working out how to prevent disease progression in the early stages would avoid later problems.

An alternative to animal models is to grow cells in tissue culture derived from nodules and cords of surgical specimens of Dupuytren's patients. This has been carried out on a number of occasions, but there are difficulties in this approach too. Many (although not all) assume that cells grown from explants represent the "transformed by disease" cells found in the nodules and cords. However, these may be the more motile cells, or in fact the structural cells from the aponeurosis in the tissues, not those involved in the disease. The cord cells differ from the nodule cells since they are clearly new structures and this criticism may be invalid. But in the cords there are fewer cells to migrate and develop. The final problem is that cells which are grown in tissue culture conditions do so in a somewhat unusual environment, albeit designed to mimic "normal" conditions of growth. The presence of antibiotics, fetal calf serum, and complex mixtures of ions may induce changes on cells which are already transformed. The problem of trying to simplify conditions may, in the long run, actually complicate understanding of which receptors are actually present. The final twist is that the mere presence of a receptor does not necessarily mean that it is involved in the process of Dupuytren's disease. The complexities of this enigmatic disease continue to frustrate attempts at understanding it fully.

What is the origin of the cells which cause the "pits" at the distal palmar crease?

When we consider such a question we enter the land of speculation rather than proven science. There are two possible sources for such cells. Either they develop from existing cells in the aponeurosis which for some reason
change their resting state to become capable of a phenotypic change, or they are derived from a source outside the aponeurosis and migrate to the superficial areas of this structure, there growing and bringing about a change in the function of the aponeurosis. In the latter case, the most obvious local source would be the connective tissue within the overlying palmar skin. It should be noted that the nodules never form on the ventral side of the aponeurosis.

That subcutaneous cells from the overlying skin could give rise to the source of the problem was suggested as long ago as 1963 (Hueston 1963). Hueston once told me it was compulsory for his surgical registrars to both read and understand his text prior to them being allowed to operate on Dupuytren's patients. Even in 1963 it was considered to be a complex disease! Hueston's theory has been reinforced over the years by available evidence, and one piece of evidence is particularly powerful in supporting his hypothesis. If the skin that overlies the palmar nodules is removed when the palmar nodules and/or cords are excised and a skin graft is applied from a nonpalmar source (using the technique known dermofasciectomy). then the condition's as recurrence is significantly less compared with those in whom the original skin is simply sutured together. This suggests that the subcutaneous fascia of the palmar skin in Dupuytren's patients may be unusual in its capacity to act as a reservoir of potentially "mobile and reactive fibroblasts" which move and are then transformed into cells which can bring about the contractile effect (the myofibroblast). But why some fingers are more prone to the effect of the overlving subcutaneous cells remains unknown.

What "instructs" the cells on the aponeurosis to contract?

When trying to develop logical strategies to treat this condition (either surgical or pharmacological), it is essential to address this question. In truth, we know very little about what instructs the cells to contract but recent evidence has suggested a number of possibilities.

Some of this evidence has been obtained from tissue culture experiments. Here, the recent ideas given by Hinz et al. (2007) should be read, concerning the placement of fibroblasts into conditions where they transform into myofibroblasts.

What "instructs" the cells in the aponeurosis to proliferate?

Agents that cause cell fibroblast/myofibroblast proliferation are much better understood than those agents which cause fibroblast/myofibroblast contraction. Mitogens have usually been studied in tissue culture using explants of cells growing from Dupuytren's nodules. Platelet-derived growth factor (PDGF) and basic fibroblast growth factor (bFGF) have been shown to be mitogenic for both normal fibroblasts and cells derived from Dupuytren's disease. Transforming growth factor (TGF)-beta receptors have been shown to present in cells derived from Dupuytren's disease. This growth factor exists in many forms: namely β 1, β 2 and β 3. The β 1 and β 2 forms have been shown to be mitogenic for myofibroblasts and a combination of the two is especially effective at high plating densities. Wipff et al. (2007) have also demonstrated the role of stress and TGF-B1 in converting fibroblasts into myofibroblasts, albeit in lung myofibroblasts.

Does a knowledge of the causative factors enable rational treatments to be suggested?

Surgical approach

The traditional treatment of Dupuytren's disease has been surgical, using fasciotomy, fasciectomy, and more recently dermofasciectomy. The first of these techniques severs the connections of the nodule from the skin. The second removes the nodules but leaves the original overlying skin and the third removes the nodules and replaces the overlying skin with a skin graft. No guarantees can be given for their success and the problem of recurrence in the first two techniques is a major problem.

The problem of recurrence necessitates surgical revision. These surgical revisions of the condition give rise to scar tissue and the growth of this can cause problems of displacement of nerves which, with the potential to damage them on further surgery, are not inconsequential. The large number of different techniques for surgical treatment of the disease suggests that this condition is far from easy to treat; in fact, some have suggested that the deformity that can follow surgery can in some cases be worse than the original disability. However, despite all these difficulties, for the majority of people affected by this disease it currently remains the only practical solution.

Pharmacological approach

As an alternative we may be able to use our increasing knowledge of the types of receptors possessed by myofibroblasts to design a specific and rational treatment, so avoiding the need for surgery. This will involve the use of "drugs" given either locally by injection into nodule or cord, or systemically to have an action(s) on:

- **a)** i) Stopping the initiation of the transformation of the cells from superficial fascia or whatever source to grow above the palmar aponeurosis. ii) Stopping the proliferation of cells once they have been initiated.
- **b)** Inhibiting the cellular powers of contractility so as to avoid the induction of more cells and to stop the movement of the MP or IP joint towards the palm.
- **c)** Inhibiting the cells, capacity to produce collagen so as to inhibit nodule and/or cord formation and so delay/inhibit nodules/cord formation.
- **d)** Selectively removing the collagen which has already been deposited.

Perhaps the most well-known use of a locally administered drug is the injection of corticosteroids, for example triamcinolone, directly into the palmar nodules over a period of 6 weeks. However, this is a technically difficult procedure and the results have always been somewhat controversial. The steroids may cause an inhibition of nodular cell division and have an effect on collagen synthesis, resulting in softening and flattening of the nodules, but may have an adverse effect on existing surrounding noninvolved fibroblasts, collagenous structures in the fascia, and in the skin above the palmar nodules and cords.

A safer, more selective alternative technique was first attempted in 1973 by Hueston. This involved selective removal of the collagen in the nodules and cords using a mixture of collagenolytic enzymes, and was described as "enzymic fasciectomy". Few reports have ever been published as to its success but a more recent technique utilizing the same basic concept (Hurst et al. 2009) used a much more powerful collagenase derived from *Clostridium histolyticum*. A very high success rate has been claimed for this technique with nodules and cords involving the MP joint in the palm.

An alternative approach was aimed at reducing the proliferation of myofibroblasts using the cytotoxic drug 5 fluorouracil (5FU), normally used for solid tumors, breast cancer, colorectal and skin cancers. Disappointingly, the results did not fulfill the expectations of the theoretical background (Bulstrode et al. 2004).

Another, approach was to use a drug that decreases the collagen release from the fibroblasts/ myofibroblasts by the use of the calcium channel blocker, verapamil. This has been formulated for topical use as a 15% gel and although it was first made available in 1998, comprehensive results are still awaited (Rayan et al. 1996).

The use of Tamoxifen (an antiestrogen/estrogen) has been suggested, but trials in Dupuytren's patients have not taken place.

Drugs recently suggested for the treatment of Dupuytren's disease are shown in Table 5.2.1. The list includes some of the more recent examples, including some very controversial ones which time alone will prove if they have any potential to modify the disease.

Two other fibrocontractive disorders will be briefly considered, as progress in understanding their

Table 5.2.1	Drugs that have recently been used for the	ł
treatment o	f Dupuytren's disease	

Drug	Usual clinical use		
Collagenase from <i>Clostridium</i>	Enzymatic destruction		
Verapamil 15% gel	Calcium channel blocker		
Imiquimod	Antiwart treatment		
N-acetyl ∟-cysteine	Mucolytic agent		
Sulfonate (MESA)	Used in some types of chemotherapy		
Vitamin E	Nutrition, antioxidant		
Neprinol – nattokinase	Proteolytic enzymes, serrapeptase		
Tamoxifen	Antiestrogen		
5FU	Cytotoxic		
Bromelin	Proteolytic enzyme		

etiology and treatment is even more controversial than for Dupuytren's disease.

Peyronie's disease

This fibrocontractive disease is less understood than is Dupuytren's disease. The site of the problem is well known to be in the Buck's fascia of the penis. The transformed fibroblast-like cells grow into thickened plaques in this fascial layer, causing irregularities to be seen when the corpus cavernosus fills with blood during the process of erection. There have been suggestions for both its surgical correction using excision of the plaque and pharmacological modifications using some of the same drugs which have been tried for Dupuytren's disease. Both surgical and pharmacological approaches are problematic. Myofibroblasts have been identified in the plaques but little is known about the receptors they possess. This is an area for much more investigation to be carried out.

Ledderhose's disease

This condition is very similar in many ways to Dupuytren's disease but occurs in the fascia of the feet, where it results in a thickened plantar aponeurosis, and in the arch of the foot. The nodules form similarly to palmar nodules but contractility is not as great a problem. The nodules can be removed surgically but with limited degrees of success. No pharmacological treatment is used.

Conclusion

So we finish almost where we started. Thomas Traherne (c. 1637–1674) suggested "The hands are a sort of feet", and little did he know all those years ago that, in terms of fibrocontractive disorders, hands and feet are remarkably similar in the diseases they can suffer: the hands *are* a sort of feet, and vice versa!

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"Frozen shoulder"

Axel Schultheis Frank Reichwein Wolfgang Nebelung

Introduction

Frozen shoulder is a benign, independent disease which normally follows the same course. Diagnosis is made from the clinical symptoms. It was described as "frozen shoulder" by Codemann back in 1934 and as "adhesive capsulitis" by Neviaser in 1945, and these synonyms are both still used. A reduction in active and passive mobility is the predominant clinical symptom, accompanied by severe pain, depending on the stage. A distinction is made between the primary and secondary forms (Lundberg 1969). The causes of primary frozen shoulder are still unknown. although it is highly associated with diabetes mellitus or metabolic disorders. The underlying cause of secondary frozen shoulder is frequently a prior disease, injury, or operation in the area of the shoulder. In terms of clinical symptoms, both the primary and secondary forms progress in stages which can be divided into three phases.

Stage I (the "freezing" phase) is characterized by a decrease in the range of motion (ROM), accompanied by sometimes intense pain which lessens in phase II (the "frozen" phase), when restriction of movement is the main symptom. There is a concentric reduction affecting all levels of freedom, with lateral rotation and limited abduction to the fore. In the final stage (the "thawing shoulder" phase), this restriction is released and shoulder mobility gradually increases again. The overall duration and the length of the individual stages cannot be predicted; with the duration of the disease being described as lasting from a few months to several years. The disease is considered to be self-limiting. The therapeutic process suitable for each stage begins in phase I with analgesic and anti-inflammatory measures, avoiding physiotherapy or manual therapy, although these form the main therapeutic component in phase II. Besides physiotherapy as an outpatient, shoulder mobilization under anesthetic or arthroscopic arthrolysis and intensive physiotherapy as an in-patient with a pain catheter may be considered in order to hasten the transition to phase III and regain free mobility as quickly as possible.

Determining the concept and classification

The term frozen shoulder covers diseases where the main symptom is active and passive restriction of movement. If the disease occurs without any evidence of any other cause, it is classed as primary or genuine frozen shoulder. This is generally understood as the cyclical picture of the disease of "capsular frozen shoulder" or "frozen shoulder" If a cause can be detected, however, which would in some way influence or provoke the limitation of movement of the shoulder joint, this is classed as a secondary form. This is where mechanical causes or e.g., glenohumeral arthritis can trigger immobilization, stiffness, or blocking of certain movements of the shoulder joint. Systemic causes can also favor the development of frozen shoulder, even if the association is not evident. The correct diagnosis is the most important step for primary frozen shoulder, where it is appropriate to rule out any other pathologies (Brue et al. 2007).

Epidemiology

The incidence of primary frozen shoulder is usually reported as 2–5% of the population Pal et al. 1986; (Hannafin & Chiaia 2000; Ricci et al. 2004), although many mild forms resolve without medical intervention. The incidence ranges from the ages of 40 to 70 years, and women are more commonly affected than men (Arslan & Celiker 2001). There is no prevalence of one side over the other (Bunker & Esler 1995). The chance of it occurring bilaterally is about 20–30% and a repeat occurrence of frozen shoulder once the patient has recovered from the disease is uncommon (Ogilvie-Harris & Myerthall 1997; Hannafin & Chiaia 2000).

Etiology and pathogenesis

Over the years, many theories and possible associations have been described, but the etiology of primary frozen shoulder remains unknown. Bulgen et al. (1976) observed an increased occurrence of HLA-B27, although this was not substantiated in later studies (Miller et al. 1996). Bunker et al. examined cytokine expression, growth factors, and metalloproteinases with frozen shoulder and found slightly increased values compared to the healthy population and the Dupuytren collective. A lack of metalloproteinase MMP-14 was discovered, which is necessary for the activation of the proteolytically effective enzyme gelatinase A (Bunker et al. 2000).

There is evidence from various working groups that the disease occurs more commonly with diabetes mellitus, where the risk of the disease is 10–19%, and with insulin-dependent diabetes mellitus even up to 36%. It is even possible that up to 42% of these patients can be affected on both sides. The course of frozen shoulder associated with diabetes is prolonged and incomplete regression with residual restriction of movement is reported (Bridgman 1972; Sattar & Luqman 1985; Fisher et al. 1986; Moren-Hybbinette et al. 1987). Lequesne et al. discovered a case of previously undiagnosed Type 1 diabetes among 60 patients with frozen shoulder (Lequesne et al. 1977). Other factors have been discussed, such as hyperthyroidism, autoimmune modulation, hormonal changes, and genetic predisposition (Hakim et al. 2003; Milgrom et al. 2008). Some authors suspect a correlation in connection with Dupuytren's contracture in the hands (Bunker et al. 2000;

Smith et al. 2001). Smith et al. found Dupuytren's contracture at the same time in 52% of (frozen shoulder) patients examined and reported in the literature comparison an eight times higher occurrence of Dupuytren's contracture. Ryu et al. found that in immunohistochemical tests in 11 operations for diabetic frozen shoulder there was an increase in neovascularization and an increased expressivity of vascular endothelial growth factor (VEGF) radiating from the synovia, and suspected a role in the pathogenesis (Ryu et al. 2006).

The pathogenetic substrate of the disease is initially a diffuse synovitis and capsulitis in stage I. which then proceeds in stage II to capsular fibrosis with an increase in fibroblasts and myofibroblasts. The typical development of a hypertrophic and contracted capsule thus leads to a restriction of movement (Neer et al. 1992; Hannafin et al. 1994; Bunker 1997). Ozaki et al. saw a central change in the coracohumeral ligament and the rotator interval. With frozen shoulder, both structures are definitely contracted, thickened, and adherent, which is how they oppose the mobility of the head of the humerus in the socket (Ozaki et al. 1989). Hand et al. found typical cells of the chronic inflammation of proliferative fibrosis in histological and immunohistochemical tests of biopsy material gained from arthroscopies from the rotator interval due to arthrolysis; the evidence of T-cells and B-cells suggests an immune modulated pathology and mast cells may play an important role in the cellular regulatory mechanism between inflammation and fibrosis (Hand et al. 2007).

In stage III, the capsular fibrosis is more or less completely dissolved again and the mobility of the head of the humerus improves once more. Arthroscopy of the shoulder brought new acroscopic impressions, so Neviaser & Neviaser (1987) described four stages:

Stage I: Slight synovitis

Stage II: Adhesive capsulitis and proliferative synovitis

Stage III: Reduction of synovitis, reduced size of the axillary recess

Stage IV: Severe reduction of the joint space.

The duration of the disease, which is in principle self-limiting, is reported as taking about 4–6 months for each stage; the course of the disease has also been described as up to 10 years without complete restitutio ad integrum (Warner 1997).

Compared with this, there are tangible causes for secondary frozen shoulder. Frozen shoulder is frequently seen following immobilization, injury, protective posture caused by pain, or postoperatively after surgical procedures and can also accompany omarthritis, calcific tendinitis, or rotator cuff lesions. An unusual cause of secondary frozen shoulder is dorsal shoulder dislocation. Frozen shoulder is also possible with systemic problems such as metabolic disorders or infections. With secondary frozen shoulder treatment is directed as required, depending on the causes.

Clinical symptoms

The typical characteristic of frozen shoulder is the active and passive restriction of glenohumeral mobility. Movement is limited in all directions in the most common form of primary or genuine capsular frozen shoulder. Clinical testing shows predominantly medial, abduction, and lateral rotation problems (Figs 5.3.1 and 5.3.2).

Systemic secondary forms show the same findings. Mechanical alterations often deviate from this guideline; depending on the causative mechanical disorder, there is then a different pattern of restricted movement. Secondary, noninflammatory causes are indicative of frozen shoulder if the clinically restricted range of movement is not pronounced to the same extent in all planes. A typical example is the so-called "bipolar" reduction in movement found in chronic rotator cuff lesions. After treatment of fractures or open procedures, combinations are often found with extensive subacromial growths, so the



Fig. 5.3.1 • Glenohumeral abduction disorder on the left with compensatory elevation of the shoulder due to frozen shoulder.



Fig. 5.3.2 • Reduction of lateral rotation with the arm adducted on the left due to frozen shoulder.

expression "subacromial adhesion syndrome" is often heard. When progress is idiopathic, the clinical course of the disease is almost standard in the three stages, although the duration of each phase is not predictable (Reeves 1975).

In phase I ("freezing"), the main symptom is the pain. Patients report a sudden event without an accident and rapid progression, with pain at rest and sometimes particularly severe pain at night. The inflammatory change to the joint capsule takes place. This is accompanied by a reduction in mobility (lateral rotation, abduction, medial rotation). The glenohumeral stiffness is initially scapulothoracically compensated, then is later demonstrated by abduction when lifting the shoulder. The time for this varies from a few weeks to several months (Murnaghan 1990; Bunker & Anthony 1995).

Patients remark that they can no longer perform small movements such as putting their hands behind their back or reaching for a wallet from their back pocket. The arm is held in a protective position, usually in adduction. The whole periarticular soft tissue coat is often painful, which can cause myogelosis and paravertebral problems, so the clinical picture is often confused.

The transition into phase II is blurred and is characterized by a regression in the pain, although the active and passive concentric stiffness remains and the shoulder is "frozen" The inflammatory process of the capsule is burnt out. Patients now report no pain at rest. Clinical examination shows the typical movement restrictions, often with a fixed stop at the end of the range of movement, although the examination may be almost pain free. In phase III ("thawing"), the stiffness is released and remobilization and release of restriction slowly take place. Here, too, the overall duration cannot be predicted. The self-healing tendency of secondary frozen shoulder is significantly less by comparison (Habermeyer & Agneskirchner 2002). If mechanical disorders are present, these do not disappear spontaneously and can only be eliminated by active therapy.

Imaging

For the basic diagnosis we recommend carrying out X-rays in three planes (outlet image, true ap, axilla). The primary frozen shoulder does not show any pathological plain X-ray findings. Glenohumeral arthrography is still hardly ever used for evidence of reduced capsule volume, although it is indispensable in everyday clinical life (Attmanspacher 2002). Nor does ultrasound provide any definite evidence. However, they can provide evidence of the involvement of swelling of the long biceps tendon and the surrounding fluid and a rupture of the rotator cuff as possible causes (Fett & Hedtmann 2002). Scintigraphy is not regularly used in making the diagnosis. There does appear to be a benefit in using it for the differential diagnosis to distinguish reflex dystrophy, as frozen shoulder can cause an isolated shoulder accumulation to form compared to multilocular accumulation along the whole arm. Magnetic resonance imaging (MRI) arthrography describes typical changes with thickening of the coracohumeral ligaments and the joint capsule and the interval due to frozen shoulder (Mengiardi et al. 2004), while an MRI of the shoulder without direct contrast medium application may show a signal change and thickening of the joint capsule and synovia. This places particular attention on the axillary recess. which is frequently thickened (Lefevre-Colau et al. 2005). The imaging can also reveal pathological findings which may explain the occurrence of secondary frozen shoulder and help with planning further treatment. An X-ray of the shoulder in three planes is usually sufficient for assessing the bony parts of the shoulder joint, and if anything is unclear another imaging test using MRI may show evidence of the causes of secondary frozen shoulder. Tumors or inflammatory processes can produce similar clinical pictures (Robinson et al. 2003).

Because of the long-term character and the permanent pain at the inflammatory stage, patients are often difficult to manage. A detailed discussion of the findings and an explanation of the actually benign nature of the disease often help. We recommend carrying out careful checks on the clinical findings and recording and documenting the range of movement; if progress is not typical, further imaging tests may possibly be sensible.

Because of the self-limiting course of the disease, primary frozen shoulder is the preserve of conservative treatment and the stage it is at will decide the therapeutic measures needed. If conservative treatment fails, progress is prolonged, or the end result is significant residual restriction of movement, mobilization under anesthetic, or better still arthroscopic arthrolysis, are alternatives. The individual patient situation should play a part in treatment planning. Compliance, profession, and functional demands are essential information when deciding on treatment.

Conservative treatment

At stage I the focus is on making certain of the diagnosis, gaining information on the clinical picture, adapting strain on the joint and trying to treat the pain. Acute inflammation with synovitis leads to pain, which occurs in all positions of the arm, including adduction and rest. Immediate exercises and physiotherapy are only indicated if progress is straightforward. In serious cases, physiotherapy exacerbates the symptoms and can delay the overall timing of the disease. Nonsteroidal anti-inflammatories only help in milder cases. The use of cortisone in addition to anti-inflammatory treatment is sensible and intraarticular administration is more effective than systemic application (Widiastuti-Samekto & Sianturi 2004). For intra-articular administration, we advise adding, for example, lidocaine (5-10 ml) and triamcinolone (20-40 mg), taking into account any contraindications, and the injection can be repeated for a second time after about 4 weeks. Alternatively, oral cortisone therapy can also be used to reduce the inflammation. According to Habermeyer, staged corticosteroid therapy can be carried out, commencing with 40 mg a day and reducing it by 10 mg every 5 days (Habermeyer & Agneskirchner 2002). The positive effect on the pain and mobility lasts for only about 6 weeks (Buchbinder et al. 2006), which nevertheless makes sense because of the general improvement in the clinical picture. In milder cases, or after the very inflammatory phase is over, cautious physiotherapy can be commenced; the extent of this must be adapted to the level of the inflammatory reaction. Manual therapy mobilization and local heat applications are recommended to improve joint play (Yang et al. 2007; Leung & Cheing 2008).

If the inflammation has "burnt out", physiotherapy is the main key to treatment. Patients can also be given exercises to carry out for themselves on a daily basis and carry out daily self mobilization (Kivimaki et al. 2007). Intensive physiotherapy should be carried out at least three times a week in combination with manual therapy mobilization. In difficult cases where stiffness fails to improve, physiotherapy can be carried out under inpatient conditions with an indwelling ISB catheter (interscalenus block), when mobilization of the contracted and swollen capsule can be carried out far more effectively, as the patient is completely free of pain due to regional anesthesia.

In our fast-living times, both doctor and patient and the health authorities are looking for quick results, so resistance to treatment and pressure of time cause the consideration of mobilization under anesthetic or arthroscopic arthrolysis.

Mobilization under anesthetic

Once the inflammatory signs have gone down and physiotherapy does not bring about any further improvement after 6 to 8 weeks, invasive methods may be considered, depending on the clinical symptoms. Mobilization under anesthetic is standard treatment (Farrell et al. 2005). However, the forced loosening of the joint capsule may lead to unintentional damage to the capsule and the labrum (Loew et al. 2005). Its relevance is unclear and the long-term results of manipulation under anesthetic are not always very good (Farrell et al. 2005). A blind, randomized, comparative study between mobilization under anesthetic followed by physiotherapy and physiotherapy alone showed only a slight benefit for the mobilized patient in regaining mobility. There were no differences in the overall scores and the pain (Kivimaki et al. 2007).

Besides the short-acting anesthetic, we recommend in every case the insertion of an ISB catheter, which leads to very efficient elimination of pain during subsequent treatment. The concomitant glenohumeral injection of a corticosteroid probably brings additional advantages. Theoretically, mobilization hides the risk of a subcapital fracture, although this is very rare if the procedure is carried out with caution, the patient is less than 60 years of age, and osteopenia is ruled out with a plain X-ray. Any forced application of strength should be avoided and attention should be paid to avoiding any very tight manipulation of the joint by using an appropriate grip technique close to the joint. There should be a definite audible and perceptible tearing of the capsule like the crunch of a snowball, as the contracted capsule gradually gives way. If there is no tearing sound, any improvement is unlikely. In the last few years, arthroscopic arthrolysis or mobilization using physiotherapy alone with an ISB catheter has practically displaced mobilization under anesthetic in our daily practice.

Arthroscopic arthrolysis

Surgical arthrolysis of the shoulder joint is, in principle, possible as an open procedure (Omari & Bunker 2001), but is really only carried out arthroscopically these days (Beaufils et al. 1999; Pearsall et al. 1999). Chen et al. carried out an arthroscopy with distension, debridement, release, and manipulation on patients with frozen shoulder where the inflammation had been treated conservatively; they achieved satisfactory results in a 23-month follow up in 173 of 186 patients (92%) and consider the method very effective (Chen et al. 2002). Musil et al. consider arthroscopic capsulotomy as the method of choice for nonresponders to conservative treatment (Musil et al. 2009). The open procedure requires significant access, which is unnecessary once the arthroscopic technique has been mastered.

The indication is basically the same as for mobilization under anesthetic. Although there are no relevant comparative studies, the arthroscopic intervention seems to be more efficient and seems to work more quickly than plain mobilization under anesthetic (Baums et al. 2007). It is probable that the sudden separation of the capsule leads to rapid freedom from pain after the pain of the operation has receded. Concomitant lesions such as subacromial impingement syndrome can also be treated. In the secondary forms, the causative factors can be treated at the same time.

With patients with tendinosis calcarea of the supraspinatus tendon and concomitant frozen shoulder. Chen both addressed the glenohumeral site arthroscopically and afterwards carried out several punctures of the calcium deposit and achieved satisfactory results (Chen et al. 2008). An additional ISB block makes follow-up treatment easier with arthroscopic arthrolysis as well. Arthroscopy of frozen shoulder is definitely made more difficult in terms of access because of the restricted, reduced size of the capsule. A cautious puncture technique is obligatory. After finding the biceps tendon, the arthroscope is cautiously inserted between the biceps tendon and the head of the humerus and the subscapularis tendon is visualized. The entry point of the anterior portal is found just above. First, the fibrosed tissue in the rotator interval (i.e., the space between the subscapularis and supraspinatus tendon) is vaporized with an electrical resector. The surgeon reveals the coracoid and the upper margin of the subscapularis tendon. In the next step, a periglenoidal capsulotomy is performed and the anterior capsule is divided almost next to the glenoid labrum. After changing the portal, the posterior capsulotomy is performed and both incisions meet up below; it is important to watch out for the axillary nerve at this point. After division of all the capsule structures, mobility is tested during the operation and minor follow-up mobilization may still be required. Subacromial decompression can be performed in the same session or if necessary, an arthroscopic resection of the acromioclavicular joint. If there is still intra-articular synovitis, we inject a corticosteroid into the joint at the end of the procedure, in which case the drainage should remain closed for 2 hours.

Reconstructive measures which require immobilization should not be carried out because of the aggressive follow-up treatment required following arthrolysis. The most important factors for the success of the procedure are the correct timing of the operation and appropriate follow-up treatment.

Treatment for secondary frozen shoulder

Secondary frozen shoulder is the consequence of other conditions, which are usually spent, so the acute inflammatory reaction is not present. Patients can, in practice, be categorized as stage II of primary frozen shoulder. Cortisone is therefore usually only rarely a suitable form of treatment and appropriate physiotherapy is the primary treatment. If mechanical factors are present which keep interfering with the mobility of the shoulder joint, these should be eliminated. This can be osteosynthesis material, but functional disorders can also lead to pain with certain movements, which in the end leads to lasting restriction of movement. Immobilization in a bandage (e.g., Gilchrist) also leads to stiffening of the shoulder joint. With increasing age in particular, the tendency to rapid stiffening of the shoulder joint increases. If the frozen shoulder is associated with metabolic events, we tend to be more likely to carry out arthroscopic arthrolysis. In principle, conservative measures are here often accompanied by poorer results.

Summary

Capsular frozen shoulder is a separate entity and the therapist should distinguish between the genuine and secondary forms.

In the acute stage, where pain is the leading symptom along with stiffness, cortisone can be applied either systemically or preferably glenohumerally, but physiotherapy exercises are not indicated. Later, after the pain has receded, physiotherapy represents the method of choice. If the frozen shoulder remains constant under long-term physiotherapy exercises, or only recedes slowly, the course of the disease can be significantly shortened in selected cases by physiotherapy with an indwelling interscalenus block, mobilization under anesthetic, or arthroscopic arthrolysis.

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Spastic paresis

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Introduction

Spastic paresis is a common term to denominate typical presentations of the motor disorders in a specific type of cerebral palsy. By definition, cerebral palsy (CP) describes a group of disorders of development of movement and posture, causing activity limitation, that are attributed to nonprogressive disturbances that occurred in the developing fetal or infant brain (Bax et al. 2005). In contrast to most other clinical disorders described in this book, CP is a neurological disorder that does not directly affect fascia or other connective tissues. From this perspective, this chapter deals with fascia-related phenomena in a clinical disorder that does not primarily affect fascia itself.

Spasticity is a neurological symptom characterized by a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks resulting from hyperexcitability of the stretch reflex (Smeulders & Kreulen 2007). Although the term spasticity is commonly added to the diagnosis of CP, it is seldom the vanguard of its clinical presentation. In the classical perspective, the muscles susceptible to spasticity are supposed to be insufficiently opposed by paretic antagonists and yield a preferred posture of the extremity towards endorotation, flexion, pronation and adduction. During active use of the extremity, interplay between agonistic and antagonistic muscle groups yields a new equilibrium resulting in a limited available range of motion, typical disturbed movement patterns and, eventually, awkward postures based on this equilibrium, where spasticity might be difficult to provoke as a clinical symptom. As such, these joint positions are not necessarily static contractures in the

classical sense. For a sizable part, they can acutely be altered in rest, after careful manipulation or under anesthesia. The role of fascia in this mechanism is underexposed in literature. How are the functional properties of intra-, inter- and extramuscular fascial connective tissues involved in this altered musculoskeletal balance?

Surgical treatment of the upper extremity in spastic paresis

Generally, interventions are aimed at decreasing postural effects of spasticity, improving function and cosmesis by various conservative therapy regimes, splinting regimes, pharmaceutical treatment and/or surgery. This chapter focuses on the surgical treatment of the upper extremity in cerebral palsy, because the role of fascia in spastic paresis of the upper extremity is best illustrated from observations during surgery. Surgical treatment is indicated only in carefully selected patients. It is paramount for the success of treatment that the desired goal of the patient is in agreement with a realistic and a surgically attainable goal. Patients with a desire for an improved manipulation of objects have to meet more strict criteria than patients with only a desire to relieve pain or an improved position for hygiene purposes. Goals of treatment can be (a) an improvement of active manual dexterity of the affected hand, (b) an improvement of bimanual dexterity by better positioning of the affected hand, (c) improved cosmetic appearance and (d) relief of pain and/or positioning of the hand for practical purposes such as personal hygiene. Before surgery, all patients are required to have (1) realistic expectations of what can be achieved, (2) an explicit motivation to reach that goal, and (3) access to a well-organized postoperative rehabilitation regime. If the goal of treatment includes an improvement of manual dexterity, the patient is also required (4) to have voluntary muscle control over the hand and forearm, (5) to show an active prehension of the affected extremity, and (6) to have sufficient insight into the principles of treatment to actively engage in the rehabilitation regime.

The next step is to compose the optimal combination of surgical procedures in one session that will meet the goal of treatment. A surgical plan is made combining three types of procedures: (i) decreasing unwanted function by means of tenotomy, aponeurectomy, tendon lengthening, or muscle release procedures; (ii) reinforcing the desired function on the paretic side by tendon transfer or tendon rerouting procedures; and (iii) stabilization of joint instability by arthrodesis (definite joint fusion), capsulodesis (tightening the joint capsule) or tenodesis (limiting joint function by tendon fixation) procedures. A long clinical experience with different surgical regimes is described in the literature, but evidence for a reliable algorithm is lacking. Surgical interventions are based on current assumptions about the nature of muscle function in spastic paresis and how they contribute to the awkward joint positions we aim to correct. The results of these interventions are not consistent in all patients and tend to be unpredictable. Therefore, patient selection as described above is very strict and international consensus exists to plan and perform surgery only at no risk of losing any existing hand function, and to set modest expectations of the clinical outcome.

A better understanding of how the interplay of the differently affected muscles in spastic paresis contributes to the disabling joint positions of the upper extremity might allow for a more reliable and predictable rationale in tailoring the surgical techniques to meet the desired functional result.

Spastic muscles

Successful treatment of patients with spastic joint positions by splinting, botulin toxin injection, surgical lengthening, or tenotomy of spastic muscles suggests that these muscles themselves are the prime cause of the limited range of motion of the joint involved during active and passive movement. Although clinically these observations are straightforward, it has been very difficult to find good scientific evidence regarding the specific cause of such joint position and even regarding the secret behind the success of the treatment.

Many of the efforts to provide evidence have been directed at alleged structural changes of spastic muscles as an adaptive response to pathological conditions. Hence, spastic muscles have been considered to be atrophied as a consequence of disuse (Gracies 2005), hypertrophic and fibrotic as an adaptive response to hypertonicity, increasing the amount of collagen within the muscles (Booth et al. 2001), while the longstanding shortened position of the joints has been proposed to cause structural shortening of both intramuscular connective tissues, muscle fibers, and joint capsules (Tardieu et al. 1982; Botte et al. 1988; Fry et al. 2007; Pontén et al. 2007). The role of fascia in spastic paresis has, as such, long been acknowledged. However, controversy exists, as most histological studies fail to prove that spastic muscles are fibrotic and structurally shortened. A few reports even showed biopsies of spastic muscle containing normal amounts of connective tissue (Romanini et al. 1989; Ito et al. 1996; Marbini et al. 2002). Partly in contrast, others reported an increased amount of connective tissue in some of the biopsies from several spastic muscles. However, even in those studies 50% of muscle biopsies were considered normal or only showed a limited abnormality, despite "the presence of static and dynamic contractures" involving the target muscles. Likewise, among muscle biopsies that did indicate fibrosis were those of patients who clinically did not show contractures (Castle et al. 1979; Rose et al. 1994). On the other hand, a sole report indicates a significant correlation found between clinically measured muscle tone and the amount of collagen in spastic muscle biopsies (Booth et al. 2001). Likewise, it has been more and more accepted that loss of serial sarcomeres within muscle fibers as a cause for structural shortening of muscle is not a common finding in spastic muscles, with muscle atrophy present in some, but not all (Fry et al. 2007; Pontén et al. 2007).

This review of the scientific work on spastic muscle function reveals a lack of a sound scientific consensus regarding the nature of the contribution of spastic muscles to the disabling joint positions of the extremities in spastic paresis.

Observations during surgery

Mechanical testing of spastic muscle tissue (Lieber et al. 2003) and direct measurement of passive length-force characteristics of partially dissected human spastic flexor carpi ulnaris muscle (FCU) (Smeulders et al. 2004), did not show unusual muscular characteristics that would explain the abnormal wrist position. Therefore, this implies that adaptation of muscular characteristics per se may not cause the joint limitation.

Interestingly, we noticed that tenotomy of FCU did not result in a major retraction of the muscle to slack length (even if the muscle was activated maximally). Instead, on tenotomy and after activation FCU shortened a little, but remained relatively close to its former insertion (Fig. 5.4.1) (Kreulen et al. 2003). Even more surprisingly, as the wrist was moved passively from flexion to extension during surgery, tenotomized FCU (not spanning the wrist any longer)



Fig. 5.4.1 • Diagram showing in the upper graph the mean shortening of the FCU muscle after tenotomy and after dissection, before and after tetanic contraction in millimeters (mm). The lower graph shows the mean passive excursion of the FCU during movement of the wrist from maximal flexion to maximal extension before tenotomy, after tenotomy, and after muscle dissection.

was lengthened almost to a similar degree as the intact FCU, showing that the FCU is connected to structures that span the wrist via epimuscular or epitendinous connective tissues (creating a distally directed myofascial load on FCU; for further explanation see Chapter 3.2). Even after tenotomy, such connections pull the FCU along on passive wrist extension. On wrist flexion, FCU retracted elastically so the process was repeatable during cyclic movement of the wrist. Apparently, the connective tissues involved were so stiff that they could transmit the full FCU force. and strong enough so that they did not break on such force exertion. Such FCU length changes after tenotomy diminished greatly, but did not fully disappear after partial dissection in distal-proximal direction (along 50-60% of the muscle belly, necessary for subsequent tendon rerouting). These results place the concept regarding the role of the fascia in spastic joint positions in a whole new perspective.

Previously, the role of connective tissues as a force-transmitting matrix has hardly been considered. Re-measuring length–force characteristics at the tenotomized distal tendon of FCU at different wrist joint angles allowed us to study the effects of fascial connections.

We also showed length-force characteristics measured at the distal tendon of the (partially dissected) FCU to vary dramatically between patients with comparable spasticity-related joint positions (Kreulen & Smeulders 2008), implying that the variables of the passive and active length-force characteristics of the semidissected muscles themselves cannot be the determining factor for developing an abnormal joint position. However, both active and passive length-force curves of the tenotomized FCU varied significantly when the muscles and connective tissues adjacent to the FCU were kept short (by manipulation of the wrist angle, not affecting FCU length), compared to when they were held at lengthened position (Smeulders et al. 2005). This proves that the relative length and position of adjacent structures codetermine the FCU characteristics and functional capabilities. Some patients showed differences in the length-force characteristics between flexion and extension of the wrist, particularly at low FCU lengths, and the passive force was highest in the flexed position of the wrist, while others showed differences at high FCU lengths, and FCU passive force was highest in the extended position of the wrist. Differences between patients were not directly related to the clinical presentation and severity of the spastic paresis (movement limitation), but these findings do prove that epimuscular fascial connections directly affect characteristics of spastic muscle and therefore its functional capabilities.

Epimuscular force transmission

Nonmyotendinous force transmission of muscle via adjacent tissues requires force to be transmitted through its fascial surrounding, called the epimysium. Such force may be transmitted directly to adjacent muscles, the fascial compartment consisting of connective tissues, and it may also involve tissues that have not been considered much as force transmitting entities (e.g., neurovascular tract). Typically, nerves and blood vessels are embedded in collagenreinforced structures for protection. The embedding structures are stiff enough to transmit force, they cross multiple anatomical compartments and enter muscle bellies and attach to myofibers at levels between their proximal and distal myotendinous junction. Therefore, they cannot be ignored when studying force transmission.

The consequences of epimuscular force transmission for a muscle function and, ultimately, in proposing an alternative hypothesis explaining the cause of spasticity-related joint position in cerebral palsy, can best be understood when the following phenomena are discussed.

1. Mechanical connections exist between muscles (and their fascial surroundings), and relative position changes between these muscles and fascial structures due to movement or contraction will affect the length, stiffness and direction of action (proximal or distal) of these connections. Such changes will affect quantity, as well as direction, of force that is exerted via epimuscular pathways. As a consequence, force exerted at the muscle's origin and insertion will be affected in different ways (Huijing & Baan 2001; Huijing 2007). For lower arm muscles, these effects of changes of relative position between synergistic muscles may be important mainly at the distal part of muscles, because this is where the biggest positional changes are expected. Due to sizable positional changes of antagonistic muscles during movement, dramatic effects of epimuscular force transmission may be expected if connections are present between antagonistic muscles. In fact, from animal experiments there is substantial functional evidence of such connections

(Huijing et al. 2007; Meijer et al. 2007; Rijkelijkhuizen et al. 2007), but also from some in-vivo human experiments involving magnetic resonance imaging (see Chapter 8.3).

- 2. There will be a distribution of sarcomere lengths even within the group of serially linked sarcomeres within the same myofiber (Yucesoy et al. 2006), because groups of sarcomeres will have unique connections to adjacent connective tissues that run outside the myofiber toward adjacent tissues. Also, the mean sarcomere lengths of myofibers will be distributed causing differences for different parts of the muscle (for details, see Chapter 3.2 and Yucesoy & Huijing 2007).
- **3.** Multiple myofascial loads may be present. Neurovascular tracts enter limb muscles proximally, thereby exerting a proximally directed epimuscular load on the muscle, while distal fascial connections that run over the joint (Kreulen et al. 2003) exert a distally directed epimuscular load. This example illustrates that more myofascial loads are expected to be exerted simultaneously on a muscle. If they work in different directions, counteracting effects of epimuscular loads are expected on the same muscle.

It should be clear that no direct proof of these phenomena exists as yet for human spastic muscles, as all the scientific evidence that is presented is circumstantial. However, direct proof of epimuscular force transmission is difficult to obtain in experiments on humans, as the structures involved are delicate and expected effects may be quite subtle at a local level. Any invasive method to actually measure forces may interfere with, or even destroy the force transmitting pathways involved. Nonetheless, the available circumstantial evidence may offer an adequate framework from which a theory can be proposed.

Towards an explanation of spasticity-related joint positions

The equilibrium resulting from the interplay of both spastic and unaffected muscles that yields the spasticity-related joint positions induces specific conditions of the involved limbs, because muscles that are affected by spastic stretch reflexes and hypertonicity are often kept in shortened position compared to unaffected muscles. This affects the myofascial loads on these muscles. The shortened position of one or more spastic muscles in relation to its surroundings may be more extreme than encountered in unaffected limbs. It has been shown that the distal myofascial load is high in shortened muscle (Huijing et al. 2007; Meijer et al. 2007; Rijkelijkhuizen et al. 2007), and that positional changes of the wrist did affect the force that was exerted at the distal tendon in spastic FCU, indicating a distally directed myofascial load (Huijing & Baan 2001). The fraction of force exerted by the muscle that is transmitted onto the distal tendon is affected, as with greater distal myofascial loads (yielding higher connective tissue stiffness) increasing percentages of the exerted force will be exerted via myofascial pathways. Therefore, forces of spastic muscles may not be merely exerted at the distal tendons, but may well be exerted mainly via epimuscular connections onto synergistic muscles that are shortened less, or onto extramuscular tissues. However, such distal myofascial force transmission alone would not explain the high moments that are required to cause the pathological and rigid joint positions of the spastic limbs, because a shortened muscle operates within a disadvantageous length range of its length-force curve, and would not be able to exert high forces.

Our hypothesis (Huijing 2007) is that the extreme conditions within a spastic limb cause proximally directed epimuscular myofascial loads originating from antagonistic muscles to be exerted on spastic or synergistic muscles, through pathways similar to those that have been shown to be effective in experimental work on healthy animals (Huijing et al. 2007; Meijer et al. 2007; Rijkelijkhuizen et al. 2007). The antagonistic extensor muscles are at high length (because of the angle of the flexed joint), as will be their adjacent connective tissues. Therefore, they will be subjected to a distally directed epimuscular myofascial load creating a path for force transmission via extramuscular fascial structures. Such a path may pass through the intermuscular septa (e.g., via neurovascular tracts) and transmit force onto the spastic muscle, and thus exert a proximally directed myofascial load on its myofibers and connective tissue stroma (Fig. 5.4.2). At the location of loading, this will keep sarcomeres long. This high force could, in principle, be exerted (a) at the distal tendon of the spastic muscle or (b) via the distal myofascial pathways (distal load), and therefore create a flexion moment at the joint. This would explain the patient's rigid joint flexed position. because the more active antagonistic muscles are, the higher flexion moment would be exerted. Obviously,



Fig. 5.4.2 • Schematic of the hypothesis of force transmission between spastic muscle and its antagonistic and synergistic muscles, explaining the movement limitation. The muscle fibers of each of these muscles are represented as rectangles and divided into a proximal, intermediate, and distal part. The size of these parts is representative of the length of the sarcomeres within them. The arrows indicate the forces exerted on the muscle fibers or their parts. The thick arrow at the left indicates the extramuscular myofascial transmission of force between the antagonistic muscle and the spastic agonist muscle, and the thick arrow at the right the epimuscular myofascial force transmission between the spastic and the synergistic muscles. Note the complex distributions of lengths of sarcomeres arranged in series within muscle fibers (no parallel distributions for sarcomere lengths are shown for the muscles). Also note that according to the hypothesis most of the force causing the movement limitation originates from proximal sarcomeres within the antagonistic muscle is transmitted by sarcomeres and/or their associated collagen fiber reinforced extracellular matrix of intermediate location within the spastic and synergistic muscles, and ultimately exerted at the distal tendons of the synergistic muscles. From Huijing PA et al., 2007, with permission.

such rigidity is enhanced by hyper-reflexivity and cocontraction of the spastic muscle. Due to the presence also of a distally directed load on the spastic muscle (as discussed above), the most distal sarcomeres with myofibers of the spastic muscle will be unloaded (and shorten) and the situation will be somewhat more complex: Part of the force will be transmitted either on synergistic muscles and exerted at their distal tendons (exerting flexion moments as well) or on extramuscular connective tissues passing the joint at the flexor side (therefore also capable of exerting flexor moments). Therefore, in these more complex conditions also, a flexed joint angle is inevitable.

The complexity of the condition is illustrated by considering the distribution of length of sarcomeres within myofibers of the spastic muscle: three groups can be distinguished.

- **1.** A most proximal group of sarcomeres are subjected to a proximal myotendinous load exclusively, with a corresponding length.
- **2.** A more distal group of sarcomeres that is loaded by a myotendinous load (transmitted by sarcomeres in series), as well as a myofascial load (originating from the antagonistic muscle). These loads are both proximally directed and will add and keep sarcomeres longer than in the most proximal section of the myofiber.
- **3.** Distally to the point of application of the distally directed myofascial load, sarcomeres will be exposed only to the comparatively small distally directed myotendinous load (which could even be zero if *myofascial* connections are very stiff) and, as a consequence, are shorter.

High sarcomere lengths in the middle third part of both spastic flexor and extensor muscles of the forearm have been measured using laser diffraction techniques and presented as representative for the whole myofiber (Pontén et al. 2007). We argue that the latter is incorrect. Although such proposed adaptation of the spastic FCU is unlikely (Smeulders et al. 2004), such high sarcomere lengths have previously been ascribed to muscle atrophy as an adaptive response to the short position (Lieber & Friden 2002), but this has never been proved. We propose that spastic FCU may not show any net effects of muscle atrophy or serial sarcomere number adaptation because of locally counteracting effects of adaptation (see also Chapter 8.4).

Conclusion

Again, we feel the need to emphasize that this model for the explanation of joint positions through epimuscular force transmission is a theory based on scientific experimental work and clinical observations that needs further scientific confirmation. Nevertheless, it fits the available data on spastic muscle and provides a new pathway of thinking about the nature of joint positions in spastic paresis, and possible therapeutic interventions

However, it is clear that the role of fascia in the musculoskeletal balance within the body cannot be ignored. The extent of its contribution to clinical pathology, the exact location of its mechanism, its adaptive features after therapeutic intervention, and its variability between patients are still unknown. Further scientific research unraveling mechanisms of myofascial force transmission in a functioning human extremity will affect therapeutic regimes in the future. The surgeon and therapist may be able to more objectively tailor their treatment plan to the specific needs of the patient.

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Diabetic foot

Sicco A Bus

Introduction

Diabetes mellitus is a chronic disease affecting over 200 million people worldwide, a number that will double in the next 20 years (www.diabetesatlas. org/content/diabetes). Diabetes may lead to several vascular and neurological complications, including ulceration, infection, or destruction of deep tissue in the foot. This "diabetic foot" affects approximately 15% of patients (Boulton et al. 2004). The foot ulcer is the key clinical problem in the diabetic foot that can cause infection and lead to lower-extremity amputation. Foot ulcer incidence is 7.2% per year in diabetic patients with peripheral neuropathy (Abbott et al. 1998). Peripheral neuropathy leads to a loss of protective sensation, which makes the patient unaware of foot trauma caused by the repetitive action of elevated mechanical foot pressures. These high foot pressures are secondary to structural abnormalities which include claw toe and Charcot deformity, prominent metatarsal heads, and changes in subcutaneous and periarticular connective tissue (i.e., tendon. fascia, ligaments, and joint capsule). Morphological changes in the plantar fascia and Achilles tendon, and limitations in joint mobility have been reported in diabetes. Changes in these foot structures share a common etiology related to longstanding hyperglycemia and they may influence foot mechanics during gait and lead to foot ulcers. The goal of this chapter is to provide insight into the changes that occur as a result of diabetes in the plantar fascia, Achilles tendon, and joint mobility of the lower extremity. The underlying mechanisms of these changes will be discussed, together with their

biomechanical and clinical implications, as well as available treatment options.

Methodology of testing

Different methods can be used to assess changes in foot structure, joint mobility, and biomechanical function in the diabetic foot.

Assessment of fascia, tendon, and ligament

Morphological changes in subcutaneous and periarticular tissues in the foot and lower leg are best assessed using in-vivo imaging techniques. With high resolution ultrasonography the geometric boundaries of superficial structures in the foot and lower leg can be assessed, from which tissue thickness can be measured (D'Ambrogi et al. 2003; Giacomozzi et al. 2005). More detailed qualitative and quantitative information of superficial and deep structures can be obtained using magnetic resonance imaging (MRI). MRI is superior to other imaging techniques in distinguishing soft tissue such as muscle, tendon, ligament, fascia, and fat, and can be used to measure tissue thickness and assess the presence of rupture. The plantar fascia can be tested functionally using Jack's test, in which the hallux is passively dorsiflexed while weight-bearing (Chuter & Payne 2001). A normal response is tightening of the fascia and a raise of the foot arch. Failure may indicate fascia dysfunction or rupture.

Assessment of joint mobility and stiffness

The so called "prayer sign" was originally used to classify patients with limited joint mobility (LJM). This is present when the patient fails to approximate the metacarpal-phalangeal joints while opposing the palmar surfaces of the hands in a praying position. Although this method is simple in use, it is not a direct measure of LJM in the foot. Typically, a goniometer is used to assess mobility of the foot and ankle joints in a nonweightbearing position. Goniometric measurements of joint mobility are quite reliable, with reported coefficients of variation of 8.5% for the subtalar joint and 7.4% to 11.0% for the first metatarsal-phalangeal (MTP) joint (Delbridge et al. 1988; Zimny et al. 2004). Mobility and stiffness of the first MTP joint can be assessed using a mechanical testing device (Birke et al. 1995). Joint stiffness can be calculated from vertical displacement of the first metatarsal head plotted against the applied force to the metatarsal head.

Pressure distribution measurement

Biomechanical function of the foot is most often assessed in diabetic patients by measuring the dynamic pressure distribution underneath the foot. Patients walk barefoot across a platform, which consists of a matrix of hundreds to thousands of sensors measuring vertical pressure. Most often the peak pressure and the time integral of this peak pressure are calculated for multiple anatomical regions in the foot, so that conclusions on local pressure effects can be drawn.

Nonenzymatic glycosylation

Structural changes in plantar fascia, Achilles tendon, and joint mobility in diabetic patients share a common etiology, namely nonenzymatic glycosylation of structural proteins in connective tissue secondary to the permanent hyperglycemic state in patients with diabetes mellitus (Bailey 1981; Brownlee et al. 1988). With nonenzymatic glycosylation, free glucose spontaneously attaches to structural proteins such as collagen and keratin (Schnider & Kohn 1980; Delbridge et al. 1985). Glycosylation of collagen causes an increase in intermolecular cross-linking and significant alterations in the structural stability of different collagen-rich subcutaneous and periarticular connective tissues, such as tendon, ligament, fascia, and joint capsule (Delbridge et al. 1988). Glycosylation of keratin causes hyperkeratosis of the skin (Delbridge et al. 1985). These tissues show reduced flexibility, increased tensile strength, and other morphological adaptations (Crisp & Heathcote, 1984), which are likely the cause for some of the structural abnormalities found in the diabetic foot.

Plantar fascia

The plantar fascia, or aponeurosis, is an important connective tissue structure which provides support, rigidity and stability in the foot under dynamic conditions (Hicks 1954; Sarrafian 1983; Sharkey et al. 1998). The aponeurosis consists of longitudinally oriented collagen and elastic fibers. It originates from the posteromedial calcaneal tuberosity and divides into five bands at the mid-metatarsal level, with each band inserting into the plantar plate and the skin. (Bojsen-Moller & Flagstad 1976; Theodorou et al. 2000, 2002). According to the windlass mechanism described by Hicks (1954), extension of the MTP joint during the propulsion phase of gait causes the aponeurosis to tighten and to draw the calcaneus and the metatarsal heads together. This results in a raised longitudinal arch and rearfoot supination, thereby making the foot a stable rigid lever in propulsion. On weight bearing in the first half of the stance phase, the arch flattens, which increases tension in the aponeurosis. This tension unwinds the "windlass", causing flexion at the MTP joint. Failure of the plantar aponeurosis most often occurs proximally. In nondiabetic subjects, aponeurosis rupture may reduce its stabilizing action and lead to collapse of the longitudinal arch or claw toe deformity, and may increase pressures in the forefoot (Hicks 1954; Sarrafian 1983; Sharkey et al. 1999). Fascia thickening may alter the height of the longitudinal arch (Arangio et al. 1998). The effects that diabetes has on the plantar fascia are largely unknown.

Rupture and fasciitis

The plantar fascia of diabetic patients with claw toe deformity showed discontinuity, indicating rupture, in one MRI study (Taylor et al. 1998). The authors suggest that the effects of nonenzymatic



Fig. 5.5.1 • The plantar aponeurosis shown as a low signal intensity structure on this sagittal plane magnetic resonance image of the foot of a neuropathic diabetic patient. The aponeurosis does not show discontinuity that would be indicative of fascia rupture.

glycosylation may render the aponeurosis less compliant and more prone to rupture. However, none of the patients with claw toe deformity in another MRI study showed discontinuity of the aponeurosis (Fig. 5.5.1) (Bus 2004). Also, signal intensity increases and substantial thickening of the aponeurosis at the calcaneal insertion compatible with plantar fasciitis have been found in neuropathic diabetic patients. However, these changes did not differentiate patients with toe deformity from those without (Bus 2004). Clearly, the data on fascia rupture and the role the aponeurosis plays in causing toe deformity in diabetic patients remain inconclusive.

Plantar fascia thickening

The plantar fascia may be thicker in diabetic patients than in healthy controls (D'Ambrogi et al. 2003; Bolton et al. 2005). High-resolution ultrasound images showed on average 2.0 mm thickness at the calcaneal insertion in healthy controls, 2.9 mm in diabetic patients, 3.0 mm in neuropathic diabetic patients, and 3.1 mm in diabetic patients with a history of foot ulceration (D'Ambrogi et al. 2003). With computed tomography, a thicker aponeurosis was found in diabetic patients than in healthy controls (mean 4.2 vs 3.6 mm) (Bolton et al. 2005). Increased fascia thickness in diabetes is likely also associated with nonenzymatic glycosylation of collagen; a reduction of 30% in collagen content in the plantar fascia has been found in diabetic patients (Andreassen et al. 1981). Apparently, with long-standing diabetes, geometric changes of the plantar fascia shown as thickening of this connective tissue structure may occur.

Biomechanical implications

Thickening of plantar fascia can influence biomechanical foot function in diabetes. Dynamic forefoot pressures were found to be significantly higher in diabetic patients with thicker plantar fascia than in diabetic and healthy control subjects with thinner plantar fascia (D'Ambrogi et al. 2003; Giacomozzi et al. 2005). Additionally, a significant correlation (r = 0.52) was found between fascia thickness and forefoot pressure in these patients. This association may be explained by the role plantar fascia plays in countering the flattening of the foot during mid-stance of gait. The vertical forces acting on the forefoot during arch flattening are countered by horizontal forces generated in passive structures such as the plantar fascia, which try to tie the forefoot and rearfoot together. With fascia thickening, resistance increases, meaning that larger vertical forces measured as higher pressures are required at the forefoot in order to flatten the foot during stance.

In patients with Charcot's neuroarthropathy, plantar fascia dysfunction or rupture may be indicated, based on a negative response to Jack's test (Chuter & Payne 2001). Additionally, rupture of the plantar fascia has been suggested as a potential factor in the increased forefoot-to-rearfoot pressure ratio that is found in neuropathic diabetic patients when compared to healthy controls (Caselli et al. 2002). A suggested forefoot drop as a result of fascia rupture (Sharkey et al. 1999), may cause increased loading in the forefoot and explain these results. More research is required to improve our understanding of the role that the plantar fascia plays in altering biomechanical function of the foot in diabetes.

Clinical implications and treatment

The clinical implications of structural changes in the plantar fascia in diabetic patients are not known. This may be the reason for the paucity of data on treatment options. Alterations in foot function associated with plantar fascia dysfunction may be indicative of its contributing role in foot ulceration in diabetic patients (Caselli et al. 2002; D'Ambrogi et al. 2003). However, direct assessments of this relationship have not been made to date.

Achilles tendon

The Achilles tendon is a fibrous structure which originates at the midcalf position and inserts into the middle part of the posterior surface of the calcaneus. It is the thickest and strongest tendon in the body. Contraction of the calf muscles pulls the Achilles tendon, resulting in plantar flexion at the talocrural joint. Injuries or abnormalities of the Achilles tendon include tendinosis, rupture, and shortening (equinus deformity). In diabetes, the Achilles tendon has been studied mainly for morphological adaptations (length and thickness) and for lengthening of the tendon as treatment option in foot ulcer patients.

Achilles tendon shortening/equinus deformity

Using electron microscopic evaluations, reductions in the collagen content of the Achilles tendon in diabetic patients have been found and are likely related to the effects of nonenzymatic glycosylation (Grant et al. 1997). These changes cause a loss of resilience and a functionally shorter tendon, which increases joint stiffness and reduces the amount of dorsiflexion. The prevalence of equinus deformity (dorsiflexion $<0^{\circ}$) in diabetic patients may be as high as 10.3% (Lavery et al. 2002), which shows that limitations in ankle joint mobility are prevalent and require serious attention in diagnosis and treatment.

Achilles tendon thickening

Just like the plantar fascia, the Achilles tendon may be thicker in diabetic patients. Tendon thickness at the calcaneal insertion was found to be 4.0 mm in healthy subjects, 4.6 mm in diabetic subjects, 4.9 mm in neuropathic diabetic patients, and 5.2 mm in patients with a history of ulceration (D'Ambrogi et al. 2003; Giacomozzi et al. 2005). Again, the likely cause of tendon thickening is nonenzymatic glycosylation. Alternatively, different walking strategies adopted by diabetic patients as a result of muscle weakness and/or neuropathy may lead to abnormal cumulative stresses in the tendon and cause the tendon to thicken (Maluf & Mueller 2003). Thickening of the Achilles tendon and plantar fascia is inversely correlated above a threshold of 3.0 mm fascia thickness ($r^2 = 61-79\%$) (Giacomozzi et al. 2005). As explanation, the dynamics of the foot during gait with a more flat footed initial contact caused by plantar fascia thickening may lead to less mechanical stress in the tendon and, therefore, a lower rate of tendon thickening (Giacomozzi et al. 2005). This contrasts with the above-mentioned "walking strategy hypothesis" for tendon thickening and points to the need for prospective analyses to understand these relationships.

Biomechanical and clinical implications

Achilles tendon shortening or thickening has both biomechanical and clinical implications. Patients with equinus deformity show higher dynamic forefoot pressures, likely because limited dorsiflexion causes an earlier heel rise during stance, which reduces the effective area to distribute pressures underneath the foot (Lavery et al. 2002; Orendurff et al. 2006). Patients with equinus have a nearly three times greater risk for high foot pressure (>850 kPa) (Lavery et al. 2002). Furthermore, dorsiflexion ROM is significantly inversely correlated (r = -0.39) with forefoot pressure (Orendurff et al. 2006). Univariate analysis shows patients with equinus deformity to be at greater risk of having a foot ulcer (odds ratio 2.3) (Lavery et al. 1998). In multivariate models, inclusion of other factors such as hallux rigidus, claw toe deformity, and elevated foot pressure removes the significance of equinus deformity.

Achilles tendon thickness has only been associated with foot pressure in multivariate models which also include plantar fascia thickness and first MTP joint mobility as factors (Giacomozzi et al. 2005). There is, overall, a low correlation (r = 0.41) between these structural parameters and foot pressure, but this increases substantially (r = 0.83) above a threshold of vertical forefoot loading (94% body weight), suggesting an important role for connective tissue changes in high foot pressures measured in diabetic patients. The clinical implications of tendon

Diabetic foot

thickening in the diabetic foot are not known. Overall, these data demonstrate the multicomponent pathogenesis of high foot pressures and ulceration in diabetes in which Achilles tendon changes may have a contributing role without being the most dominant explanatory factor.

Treatment: Achilles tendon lengthening

Lengthening of the Achilles tendon (ATL) has been used to overcome limited ankle dorsiflexion and to treat forefoot ulcers in equinus feet. In the immediate period after ATL treatment, forefoot pressures may reduce by as much as 27% (Armstrong et al. 1999; Maluf et al. 2004). Additionally, dorsiflexion ROM may increase with 15° and plantar flexor power during gait may decrease with 65% (Maluf et al. 2004). However, these biomechanical effects are not sustained over longer periods of time (8 months). The close association between forefoot pressures and plantar flexor muscle strength (r = 0.60) suggests that the return of pressure levels to baseline values over time may be due to a re-strengthening of the plantar flexor muscles after ATL (Maluf et al. 2004).

Lengthening of the Achilles tendon can also be effective in healing and secondary prevention of plantar diabetic foot ulcers. Between 93% and 100% of patients may heal successfully in about 40 days after ATL treatment, compared with 88% of patients healing in 58 days after total contact casting (Lin et al. 1996; Mueller et al. 2003). Furthermore, ulcer recurrence rates between 0% and 38% after 7-24 months have been found in patients treated with ATL compared with rates between 59% and 81% in patients treated with casting alone (Lin et al. 1996; Mueller et al. 2003). Despite these positive clinical results, several concerns have risen with using ATL treatment in diabetic patients (Mueller et al. 2004; Salsich et al. 2005). Heel pressures can increase substantially and as a result patients may develop plantar heel ulcers (Mueller et al. 2004). Self-perceived physical functioning may be reduced when compared with pre-ATL levels and when compared with conservative treatment (Mueller et al. 2004). This may require rehabilitative treatment aimed at improving gait performance and physical functioning in these patients. Thus, even though ATL seems a biomechanically and clinically effective treatment for foot ulcer patients, caution is required with this procedure since complications can arise and the effects may not persist over time.

Limited joint mobility

Adequate mobility in the joints of the foot and ankle is an important prerequisite for normal gait. Motion at the talocrural, subtalar, and MTP joints allows for adaptation to the surface and shockabsorption in initial stance, progression of the foot and tibia in mid-stance, foot rigidity for leverage in propulsion, effective push-off in terminal stance, and foot clearance in swing. Joint mobility limitations can disturb normal foot progression and lead to biomechanical changes which may be of clinical importance. LJM affects 30–50% of diabetic patients (Frost & Beischer 2001).

Limited joint mobility in the foot and ankle

The results from several studies on joint mobility in the talocrural, subtalar and MTP joints are summarized in Table 5.5.1. Joint ROM is significantly reduced in diabetic patients with a history of ulceration when compared with diabetic and nondiabetic control subjects (Delbridge et al. 1988; Mueller et al. 1989; Fernando et al. 1991; Birke et al. 1995; Viswanathan et al. 2003; Zimny et al. 2004). Furthermore, subtalar joint ROM is significantly associated with first MTP joint ROM (r = 0.53-0.59) and was found to be smaller in patients who have been diagnosed with LJM in the hands, based on the "prayer sign". LJM affects the joints of the upper extremity and lower extremity to a more or less similar extent (Delbridge et al. 1988; Fernando et al. 1991). In Caucasian patients, LJM seems to be more prevalent than in Black or Hispanic patients (Veves et al. 1995; Frykberg et al. 1998).

Joint stiffness tends to follow the same pattern as joint mobility: stiffer joints in cases with more severe foot disease (Birke et al. 1995). While nonenzymatic glycosylation is probably causative of LJM seen in diabetic patients, this does not fully explain the different findings with different states of foot disease. The role of peripheral neuropathy in joint mobility in diabetic patients is not clear (Viswanathan et al. 2003; Table 5.5.1 Joint range of motion (ROM) and stiffness data for the foot and ankle in different groups of diabetic patients and healthy controls

	Diabetes and previous ulcer	Diabetes and neuropathy	Diabetes control	Healthy control		
Subtalar joint ROM (°)						
Delbridge et al. (1988)	17.9		31.1	35.2		
Mueller et al. (1989)	26		31	35		
Fernando et al. (1991)		29, 18 ^a	29, 20 ^a	30		
Birke et al. (1995)	24.7 ^b , 25.7 ^c		32.3	31.7		
Viswanathan et al. (2003)	28.8	34.3	48.0	55.3		
Zimny et al. (2004)		17.9	28.4	31		
Talocrural joint dorsiflexion	(°)					
Birke et al. (1995)	2.2 ^b , 3.6 ^c		5.9	5.9		
First MTP joint ROM (°)						
Birke et al. (1995)	34.7 ^b , 31.6 ^c		46.8	47.2		
Viswanathan et al. (2003)	44.5	53.3	73.0	91.3		
Zimny et al. (2004)		35.3	62.0	59.4		
First MTP joint stiffness (kg	ı/cm)					
Birke et al. (1995)	12.1 ^b , 10.3 ^c		8.9	9.7		
^a Patients with diagnosed LJM based on the "prayer sign".						

^DPatients with previous ulcer at the first metatarsal head.

^cPatients with previous ulcer at other locations.

Zimny et al. 2004). Analysis of disease progression in conjunction with the development of LJM is necessary and will require prospective studies.

Biomechanical implications

Foot pressures can be substantially higher in diabetic patients with LJM than in patients without LJM. In one study, average peak pressures were 1425 kPa in neuropathic diabetic patients with LJM, 1250 kPa in diabetic patients with LJM, 1010 kPa in neuropathic patients without LJM, 565 kPa in diabetic controls, and 550 kPa in healthy controls (Fernando et al. 1991). Furthermore, joint ROM is strongly associated with measured dynamic forefoot

pressures (Plate 5.5.1) (Fernando et al. 1991; Birke et al. 1995; Zimny et al. 2004). Correlation coefficients were -0.67 to -0.70 for subtalar joint mobility (Fernando et al. 1991; Zimny et al. 2004), and -0.62 to -0.71 for first MTP joint ROM (Birke et al. 1995; Zimny et al. 2004). In the presence of LJM, the foot and ankle seem to lose their capacity to absorb shock and progress the foot effectively through stance, resulting in a reduced efficacy to maintain normal foot pressures. Finally, LJM measured at the first MTP and subtalar joints was found to be 80% sensitive and 90% specific to differentiate between high- and low-risk patients (Zimny et al. 2004). For these reasons, joint mobility assessment may accurately identify patients with high plantar foot pressures who are at risk for plantar ulceration, and may therefore be useful for foot screening purposes.

Clinical implications

As was discussed in a previous paragraph, diabetic patients with a history of foot ulceration have smaller joint ROM and stiffer joints (Delbridge et al. 1988; Birke et al. 1988). In these patients, subtalar joint ROM seems smaller in the affected foot (with ulcer) than in the nonaffected foot. The foot site showing the most significant LJM matched the site of prior foot ulceration in 79% of cases (Mueller et al. 1989). Additionally, patients with LJM show a much higher prevalence of prior foot ulcers (65%) than patients without LJM (5%) (Fernando et al. 1991). While these findings support the hypothesis of a link between LJM and foot ulceration in diabetic patients, in which elevated plantar foot pressure is probably the mediating factor (Fernando et al. 1991), causeand-effect relationships cannot yet be established.

Joint mobility has been assessed as part of a multicomponent analysis of foot ulceration in several prospective studies. These show that LJM in the foot and ankle may be a risk factor in foot ulceration (univariate model odds ratio 2.1 to 4.6) (Lavery et al. 1998; Boyko et al. 1999). However, in multivariate analysis only LJM at the MTP joint (hallux rigidus). albeit in combination with the presence of claw toe deformity, remained a significant factor (Lavery et al. 1998). Joint mobility was significantly reduced by 2° in the subtalar joint and by 14° in the first MTP joint in patients who developed an ulcer. However, odds ratios were small (0.97), and not significant in multivariate analysis (Pham et al. 2000). These data suggest that although LJM is a contributing factor in diabetic foot ulceration, other factors such as neuropathy and some foot deformities have more prominent roles. While joint mobility assessment may be used for foot screening purposes, simple measures of neuropathy and foot deformity may be more important to determine risk for foot ulceration.

Treatment: Joint mobilization

In clinical practice, generally only the consequences of LJM are treated; for example, by prescribing therapeutic footwear to patients with LJM. Joint mobilization through physical therapy does not seem to occur, even though it has been suggested that this may potentially benefit these patients (Mueller et al. 1989). A two sessions per week passive joint mobilization program in diabetic patients with LJM improved joint mobility in the short term (5 weeks), but this effect did not persist over time and deteriorated after termination of the program (Dijs et al. 2000). Alternatively, unsupervised active and passive joint ROM exercises in the home setting in patients with LJM reduced peak foot pressure (by 4%) and joint stiffness after one month (Goldsmith et al. 2002). These studies show that mobilization treatment may have only minimal and temporary effects. Therefore, patients may require permanent treatment in order to achieve lasting improvements. Larger well-designed prospective studies are required to test this hypothesis.

Conclusions

The diabetic foot is a complex and serious complication of diabetes, with many negative outcomes requiring medical treatment. This chapter emphasized changes occurring in the subcutaneous and peri-articular structures of the foot that may have implications for foot biomechanics and diabetic foot ulcer risk. Consistent findings are the presence of thicker plantar fascia, thicker and shorter Achilles tendon, and limitations in the mobility of the foot and ankle joints, all leading to increased plantar foot pressures in diabetic patients. Data on the presence of plantar fascia rupture in diabetes are inconclusive. Nonenzymatic glycosylation of connective tissue is regarded as the mechanism for these changes, even though a direct association has never been established. While data are lacking for plantar fascia, patients with the other structural abnormalities are at greater risk for foot ulceration, although the contribution of other patient-related and biomechanical factors in ulcer development may be more significant. There is little research on the management of these structural abnormalities. Only lengthening of the Achilles tendon in diabetic patients has been shown to be effective in healing and secondary prevention of foot ulcers, although complications may arise that caution the application of this procedure in the diabetic foot and may require adequate rehabilitative treatment to improve physical functioning. Studies on the effect of joint mobilization to treat LJM in the foot and ankle are small and inconclusive. No treatment options are known for changes in plantar fascia and thickening of the Achilles tendon. Clearly, more research is needed to improve our understanding of the treatment of plantar fascia, Achilles tendon, and joint mobility abnormalities in patients with a diabetic foot.

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Scleroderma and related conditions

Tanya M Ball

What is "scleroderma"?

Scleroderma is a generic term encompassing a spectrum of complex autoimmune conditions with similar, or overlapping clinical features. These can range from relatively localized disorders such as primary Raynaud's phenomenon, to significantly more debilitating systemic diseases, including systemic sclerosis (SSc) and systemic lupus erythematosus (SLE) (Denton & Black 2002; Assassi et al. 2007). This umbrella term encompasses SSc, SLE, mixed connective tissue disease (MCTD), Sjögren's disease (SjD), primary/secondary Raynaud's phenomenon (PRP/ SRP), and rheumatoid arthritis (RA). Scleroderma denotes a condition presenting as thickening, hardening, and scarring of the skin. Systemic refers to an autoimmune condition that affects the internal organs as well as the skin and superficial (subcutaneous and "deep investing") fascia layers. This chapter will focus on the systemic forms of the disease because of their wider significance for manual therapy (MT) intervention.

Conditions classified under the "scleroderma umbrella" generally share the following characteristics:

- **1.** All are chronic, inflammatory, autoimmune conditions.
- **2.** Etiology is multifactorial, with potential genetic, environmental, physical, and emotional health predisposing factors, with no known cause. Excessive coincidence or accumulation of such factors in relation to the individual's specific immune tolerance triggers a "mistaken identity" failure within this system. Healthy body cells are erroneously interpreted as foreign,

threatening entities to be destroyed by the creation of associated antibodies. Blood tests can identify disease-specific abnormal antibodies which predominantly target the skin and fasciae or connective tissues (CTs) (Denton & Black 2002).

- **3.** Prevalence is eight times greater in women than men, with onset between the mid 20s and mid 50s.
- **4.** While various interventions, including drugs, can help stabilize and manage these conditions, they have no known cure. Chronic inflammation of the affected tissues leads to debilitating consequences including severe pain, fatigue, further immune system overload, tissue and organ damage, loss of functional autonomy, and quality of life. Complications from systemic forms of scleroderma, usually associated with infection, critical cardiorespiratory or renal failure, can be fatal, though survival rates have risen significantly (Denton & Black 2002), with SLE survival rates improving from < 50% at 5 years in the 1950s, to 85% at 10 years nowadays (Merrell & Shulman 1955; Urowitz et al. 1997; Bernatsky et al. 2006; Abu-Shakram 2008).
- **5.** Pathological hardening, thickening, scarring of any tissue affects three essential systems of immediate and significant relevance to manual therapy (MT):

The vascular system

The immune system, including the lymphatic system; and

The fascial system (Denton et al. 1996, Denton & Black 2002; Assassi et al. 2007; Abu-Shakram 2008).

Clinical features of special relevance to MT

The most significant consideration from a MT perspective is the impact of scleroderma on the CTs or fasciae. A sound understanding of how these are affected is critical to appropriate clinical reasoning, and an effective treatment and management plan. Martin's (2009) case study of a diffuse systemic sclerosis (dSSc) patient summarizes the pathological processes involved, which are characterized by fibrotic changes associated with fibroblastic cell over-activity, leading to collagen overproduction within those tissues (Denton & Black 2002). Ensuing adverse tissue changes include thickening, shortening, hardening, and scarring, which in turn result in reduced range of motion (RoM), vascular, lymphatic, neural, joint, and visceral compression and constriction. Consequences include ischemic pain, with potential local tissue necrosis or infection, sensory, motor, and autonomic dysfunction, and further immune deregulation, thus completing the "downward spiral" (Adams et al. 2002; Denton & Black 2002). This intricately linked neurovascular-fascial pathology accounts for much of patients' pain, functional impairment, and psychological distress (Findley & Schleip 2007; Martin 2009).

It is important from a MT clinical perspective to appreciate the nature and process of damage to the varying fasciae of different structures and organs.

Features affecting the neurovascular and fascial systems

Vasculitis

Collagen overproduction within vascular walls (epi- and endothelium) leads to thickening, stiffening, narrowing of their internal diameter, and hence ischemia to the tissues and cells supplied. The very fine capillaries supplying oxygen at cellular level are the most susceptible. The most commonly involved tissues and organs include: the extremities – hands and feet, especially the fingers and/or toes, the gastrointestinal (GI) tract, lungs, heart, kidneys, as well as muscles and joints.

The myofascial system

Fibrotic myofascial change can vary in severity both in terms of area(s) affected and degree of ensuing restriction and dysfunction. It is near-universal in In the author's experience, fibrotic shortening and restriction particularly occur in the flexor muscle groups. Increased secondary spinal curves at the lumbar and mid–upper cervical levels are common, forcing the head anteriorly in relation to the body's center of gravity, with a posterior tilt ("chin poke"). Additional asymmetrical fascial changes occur both locally and over a distance, with postural and functional impact, compounded by pain, fatigue, and sometimes muscle weakness. The face, mouth, and jaw are commonly affected, which can lead to dental overcrowding, mouth opening restriction, and associated eating and other implications for daily life.

Joint inflammation (particularly in SLE patients) presents as acute swelling, pain, and stiffness. The wrists, hands, fingers, or feet and toes can develop permanent deformities, usually associated with secondary Raynaud's phenomenon and sclerodactyly.

Secondary Raynaud's phenomenon ("Raynaud's")

Most scleroderma sufferers experience abnormal, severe vasoconstriction to the small capillaries in their fingers and/or toes when exposed to cold, emotional stress, or immune system overload, for instance prolonged excessive working hours and inadequate sleep.

Typically, the affected hands and particularly the fingers become chronically swollen, which further compromises vascular and lymphatic circulation. Fibrotic changes, coupled with collagen overproduction as described above, thicken and stiffen the capillary walls, thereby reducing or even occluding their inner diameter.

Sclerodactyly

Aggressive fibrotic changes to the skin and subcutaneous fascia predominantly manifest in the hands, fingers, feet, and/or toes with swelling, tightening, hardening, and thinning of the skin, which takes on a waxy, shiny appearance. The tightness is frequently associated with shortening of underlying fasciae, resulting in claw-like finger deformities. Commonly, the wrist and metacarpophalangeal joints become locked in relative extension, while the proximal and distal interphalangeal joints (IPJ) become "locked" into mild to excessive flexion. The thumb saddle joint adducts, with IPJ hyperextension. When distal limb sections are affected, patients exhibit semi-flexed elbows or knees at rest due to excessive contracture.

Pulmonary sclerosis/pulmonary hypertension (PHT)

A thickening, hardening, and scarring of the alveolae and/or adjacent capillary epi-/endothelium can induce, through fibrosis, two related potentially life-threatening situations: (i) reduced oxygen uptake and carbon dioxide removal through the thicker, inelastic, fibrotic tissue layer, leading to excessive breathlessness; (ii) rise in pulmonary arterial pressure due to the same fibrotic narrowing or occlusion of the capillaries adjacent to the alveolae.

Pleuritis inflammation of the pleura, the outer fascial lining of the lungs, can also occur, causing chest pain aggravated with coughing.

Myo- and pericardiac damage

More rarely, interstitial pulmonary fibrosis as described above can progress to severe lung damage, triggering an immunoinflammatory response in the heart. This presents as pericardial swelling, left ventricular ("pressure pump") hypertrophy against reduced right ventricular ("volume pump") input in response to pulmonary arterial hypertension, arrhythmia, and diastolic dysfunction. Fibrosis of the heart muscle can be a further serious risk factor (Stamenkovic 2008, Allanore 2010).

GI tract

- 1. Upper GI tract: the lower esophageal sphincter (LES) opens upon swallowing, to enable food to be propelled into the stomach, and closes in between swallowing to prevent reflux of stomach contents back into the esophageal tube in a coordinated way, and is an essential part of overall peristalsis (Gaumnitz & Fayyad 2009). Atrophy and fibrosis result in acid and general stomach contents reflux when reclining, bending forward, or even sitting after food or drink consumption. Second, it results in difficulty in swallowing, notably solid food, and particularly when time has elapsed since fluid intake and/or with low salivation.
- **2.** Mid/lower GI tract: smooth muscle atrophy and fibrosis can result in distention of the intestinal diameter, leading to inefficient, sometimes

bidirectional peristalsis. Poor peristalsis can lead to cyclical episodes of bacterial overgrowth; symptoms can include alternating bouts of constipation and diarrhea, nausea, sometimes with vomiting, abdominal bloating, swelling, severe pain, tenderness, and extreme exhaustion.

3. Lower GI tract extremity/anal canal: fibrotic damage to the neurovascular–fascial structures of the inner and/or outer anal sphincter(s) affects the sensory-motor supply to the pelvic floor musculature, inhibiting the normal closure reflex. This can cause loss of full bowel control and intermittent leaking or incontinence.

GI tract involvement can have a particularly profound psychosocial impact on those affected and their families.

Other potential organ involvement

The same inflammatory/fibrotic process rarely affects the brain, liver, and/or kidneys.

Reduced white blood cells (leukopenia) or a tendency to bruise or an increased risk of bleeding, due to a low platelet count (thrombocytopenia) may also occur. Peripheral neurological disturbance can present as tingling, loss of sensation, numbness, or weakness.

Types of scleroderma, and where SSc fits in

Scleroderma is categorized according to the extent of skin and organ involvement, which with childhood onset can lead to growth deformities.

Localized scleroderma (LSc)

Morphea presents as: localized patches of thickened, sclerotic, usually discolored (darker or lighter) skin, most often oval or round in shape, which can appear anywhere on the body. These patches are normally painless but can be itchy.

Linear scleroderma presents as: lines of hardened, sclerotic skin that can appear on the head, arms, or legs, and can involve the subcutaneous (areolar) fascia as well as underlying myofascia and even bone.

Systemic sclerosis (SSc)

Limited cutaneous systemic sclerosis presents with: calcinosis; Raynaud's phenomenon; esophageal dysfunction; sclerodactyly; and telangiectasia (Denton & Black 2002), represented by the acronym CREST.

Diffuse cutaneous systemic sclerosis: has the highest incidence of fascial and organ pathologies, and higher morbidity and mortality rates (Denton & Black 2002; Stamenkovic 2006); with renal failure a common complication.

Mixed connective tissue disease (MCTD)

These patients have a mixture of local and diffuse tissue involvement.

Conventional medical management

Reduction in scleroderma-related mortality has led to a priority for morbidity management strategies (Merrell & Shulman 1955; Urowitz et al. 1997; Denton & Black 2002; Bernatsky et al. 2006; Abu-Shakram 2008). Disease stabilization and patient management remain a challenging order, often with limited to moderate success. Patient education in what treatments and self-management options are available is an essential component, as is the careful correlation between intervention selection and disease subset and stage (Denton & Black 2002).

Recent conventional medical prognostic developments include:

- Scleroderma "hallmark" antibody screening to identify patients at increased risk of complications.
- Preventative patient screening for pulmonary hypertension.

Conventional medical treatment and management aimed at stabilizing or even modifying the disease process broadly comprises three categories:

 Strategies targeting the vascular system, and notably Raynaud's symptoms. These include oral and intravenous (prostacyclin/iloprost) vasodilator and anticoagulant medication aimed at dilating capillary vessels and inhibiting clotting, with the view to maximizing blood flow through the finest vessels in the extremities and/or organs. Other conventional interventions for hypertension are also used with variable success to control pulmonary HT (Wigley et al. 1992; Denton & Black 2002).

- Immunosuppressive drugs, aimed at inhibiting autodestructive immune responses, have shown limited results for DCSSc patients, but remain largely ineffective for LCSSc sufferers.
- Antifibrotic therapy, in the form of antagonist drugs aimed at inhibiting fibroblast proliferation and extracellular matrix synthesis, is summarized by Denton & Black (2002).

In summary, while advances in medicine have reduced mortality and enhanced disease stabilization and patient (self-)management, these improvements remain limited, and elicit inconsistent responses from patient to patient, with frequent adverse medication side effects. This calls for the exploration of nonpharmaceutically based treatments to control, or even reverse symptoms, and notably tissue fibrosis.

Can MT help reduce or reverse scleroderma-related fibrotic changes?

Clinical evidence to date

Fascial release (FRT) and structural integration (SI)

Walton (2007) reports briefer Raynaud's attack episodes following a series of FRT treatments. Martin (2009) reports significant gains in chest expansion, temporomandibular (TMJ) mobility, and associated increase in mouth opening, marked wrist and finger joint RoM gains (of up to 100%), reduced digital ulcerations and overall Raynaud's symptoms, recovery of nail growth, and most importantly perhaps, reduced or eliminated pain in all affected areas. Benefits were said gradually to decrease with prolonged periods of nonintervention, but were readily restored with intermittent treatment resumption. Increased parasympathetic activity, regulation of associated autonomic functions, and other related beneficial changes following manual therapy interventions including FRT have been recently reviewed by Mover et al. (2004).

The author's first-hand clinical experience with some 20+ SSc patients in the UK and Europe shows

encouraging treatment effects of manual therapies, in conjunction with ongoing medical review and management, including via appropriate pharmaceutical drugs (Ball 2010), including enhancement of fascial "texture", glide, and remodeling.

- Decreasing: pain; use of pain medication; episodes of exhaustion; duration and frequency of GI tract malfunction; hand and finger swelling, stiffness, tenderness.
- Enhancing: functional mobility and RoM; hand and finger grip; mouth opening; functional autonomy; positive emotional state; and quality of life.

Rationale for potential MT efficacy

FRT techniques, the SI process, and the KMI[®] model

Through FRT manual manipulation of the connective tissues coupled with movement re-education, SI aims to restore length, symmetry, and balance to the body about its vertical skeletal axis in all planes (Myers 2009). In SI terminology, the aim is to restore optimal "tensegrity", integrity of equal tension in all planes between the fascial tissues "pulling", and hence applying compressive forces on, the skeletal structure. The Kinesis Myofascial Integration (KMI)[®] SI model developed by Thomas Myers (2009), which formed the basis of the author's therapeutic approach, rests on "reading and treating the cohesive myofascial continuities of the Anatomy Trains". The "Anatomy Trains" are a system of 12 key myofascial continuities, "myofascial meridians", that drape the body from either head or neck to foot or toe, from trunk to hand or finger, along a variety of relatively straight, spiral, curvy, or "basket-weave" alignments (Myers 1997, 2009). The process therefore involves accurate "body reading" to identify areas of shortened, congested, CT, compensated by overstretched, lengthened fascia elsewhere. "Interactive" manual manipulative techniques usually involving active client movement and participation are then methodically applied according to a structured treatment strategy based on associated clinical reasoning.

Potential mechanism of effect

SI/KMI®-attributed fascial changes among the general population, and specific clinical cases including scoliosis, have been well documented (Chaitow & DeLany 2003; LeBauer et al. 2008; Myers 2009). Similar interventions with scleroderma patients, possibly with greater repetition or frequency, using softening, releasing, spreading, and lengthening techniques, may generate tissue remodeling, over time leading to reversing fibrotic CT and skin changes (De Lany et al. 2002; Schleip 2003).

These immediate and longer-term changes would in turn:

- Enhance joint RoM and overall mobility.
- Relieve vascular, lymphatic, neural, joint, and visceral compression and constriction.

Leading to:

- Improved vascular and lymphatic circulation, and hence oxygen and nutrition supply to chronically ischemic, inflamed, or damaged tissues.
- Removal of congested interstitial fluid, metabolic waste, etc.
- Decreased pain and stiffness.
- Enhanced neural conduction sensory, motor, and autonomic.
- Enhanced visceral "position" and function.

In turn, decreased vascular, neural, and visceral compression would lead to reduced ischemic pain, raised pain threshold due to nociceptor nerve ending decompression, more effective respiratory and GI tract function.

The overall outcome from the above could reasonably be assumed to include:

- Reduced fatigue and anxiety.
- Enhanced tissue healing, functional mobility, autonomy, quality of life, emotional state, autonomic and immune function (Chaitow & DeLany 2000, 2003; Evans et al. 2000; Adams et al. 2002; Findley & Schleip 2007; Huijing et al. 2009; Martin 2009).

Scientific basis: potential therapeutic mechanisms

Fascial changes such as softening, increased pliability, flexibility, and mobility

These suggest a process of fascial tissue remodeling involving structural reorganization favoring greater flexibility, as proposed by Martin (2009). Pioneering research by Schleip (2003) and Wipff & Hinz (2009) on myofibroblast development, and which key factors determine whether or not they maintain their "contractile phenotype" on maturation, offers appealing, and in the author's view promising supporting evidence regarding the possible mechanisms involved: according to the study, mechanical stress such as FRT therapy is one of two principal factors, the other being the ability to utilize transforming growth factor-beta (TGF- β 1).

Restored independent sliding, gliding, and coordination between adjacent myofascial structures and associated enhanced areolar fascia "fluidity" (Fourie 2008)

This may likewise be attributed in part to the above process. Myers (2009) proposes the vivid analogy of "rolling" a grapefruit, thereby gently breaking up the various membrane layers separating the juice cells to facilitate the subsequent task of juicing, and the effect of FRT input on the areolar fasciae, intermuscular septae, and epimysia.

Decreased myofascial pain

This may reasonably be attributable to multiple factors. LeMoon (2008) proposes a convincing "fasciagenic" pain model where prolonged, unremitting fascial thickening and stiffening is deemed responsible for generating myofascial pain symptoms. Conversely, fascial relaxation interventions are proposed as a key strategy towards alleviating it.

Findings arising from the 2009 2nd International Fascia Congress

Building on earlier studies inter alia by Rijkelijkhuizen et al. (2005) and Schleip (Findley & Schleip 2007; Van der Wal 2009a, b) demonstrated how ligament tissue fulfills key functional roles in any and every joint position, because of the "in series" as opposed to "in parallel" organization of muscles, aponeuroses, ligaments, and periosteum, in an ever-continuous, uninterrupted body-wide pattern. The fresh cadaver dissections and in-vivo video clips testified to how the omnipresent interconnectivity of the fascial matrix ensured a trans-muscular tensile force transmission in series, involving muscle–aponeurosis–ligament tissue-periosteum, with an unbroken continuum both around a given joint, and from one joint to the next throughout the body. This offers compelling anatomical and physiological evidence as to how FRT-type therapeutic intervention at a given fascial site and layer might impact on deeper, manually unreachable tissue layers. These could be intrathoracic or abdominal myofascial and visceral structures and organs, with ensuing enhanced respiratory, visceral, and autonomic regulation.

Psychoneuroimmunology (PNI)

In light of the acknowledged correlation between scleroderma-type disease and sympathetic over-activity (Denton et al. 1996), it would seem reasonable to speculate that enhancing physical and psychological well-being in patients via SI intervention, including stimulating parasympathetic activity, might help regulate autoimmune response. Strategies could include placing greater focus on "end game" attention to the cervical and sacral areas, as well as consciously and sensitively addressing sympathetic responsive sites such as the thorax, shoulders, and abdomen.

Neuromuscular technique (NMT) and muscle energy technique (MET)

Evidence of NMT and MET efficacy in addressing chronic musculoskeletal pain and dysfunction is robustly supported by Chaitow (2003) and Chaitow & DeLany (2000, 2003): not only do these approaches take into account both local and distant potential sources of pain and dysfunction, but they seek to identify and address self-perpetuating factors such as myofascial trigger points (MTPs), areas of "locked short" versus "locked long" tissues reflecting loss of tensegrity, detrimental posture, and hence movement patterns. That NMT

aims specifically to alter the ground substance of connective tissue, to deactivate myofascial trigger points, and reduce ischemia, and to assess and beneficially modify postural alignment, musculoskeletal dysfunction, and neural entrapment

Chaitow & DeLany 2003.

offers plausible rationale for efficacy in reversing scleroderma-related fascial fibrotic change.

By the same token, the essence of the neurophysiological process underpinning MET techniques such as postisometric relaxation (PIR) and reciprocal inhibition (RI) is **inhibition**, in the sense of **enhanced relaxation** enabling shortened myofascia to be restored to a more 'normal' resting length. Its clinical efficacy in musculoskeletal therapy is well documented (Chaitow & DeLany 2003); Mitchell & Mitchell (1995, 1998, 1999) once again provide reasonable grounds for anticipating beneficial outcomes in the context of scleroderma.

Manual lymphatic drainage (MLD)

The efficacy of MLD in reducing acute and chronic edema and excessive interstitial fluid is likewise well documented (Chaitow & DeLany 2003; Willis 2004; Fourie 2009). Its acceptance in the UK as a useful modality to address lymphedema, including notably during pregnancy, following breast cancer

surgery/reconstructive surgery, is gaining momentum, with encouraging early evidence of efficacy for rheumatoid arthritis and scleroderma (www. bmlda.org.co.uk).

Conclusion

The current inadequacy of conventional medicine in alleviating, or reversing, scleroderma-related fascial change, anecdotal indications of MT efficacy in this respect, and the growing evidence-base supporting MT therapeutic mechanisms, jointly offer an exciting and wide-ranging opportunity for much-needed 'clinical' and 'laboratory' research in the field over the coming years.

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Trigger points as a fascia-related disorder

Roland U Gautschi

Trigger points (TrP)

Myofascial trigger points (mTrPs) are widespread and are frequently responsible for pain in the musculoskeletal system (Fishbain et al. 1986; Skootsky et al. 1989; Friction 1990; Gerwin 1995; Travell & Simons 1999; Jarrell 2004; Doggweiler-Wiygul 2004; Hwang et al. 2005; Anderson et al. 2006; Ardic et al. 2006; Borg-Stein & Wilkins 2006; Fernandez-de-las-Penas et al. 2006, 2007; Treaster et al. 2006; Ettlin et al. 2008; von Stülpnagel et al. 2009).

In the original sense of the word, a trigger point (TrP) is a point from which symptoms known to the patient, mostly in the form of referred pain, are caused (or triggered). Various types of TrPs have been distinguished (Travell & Simons 1999; Gautschi 2010):

• Active or latent TrPs. Active TrPs already demonstrate their characteristic pain pattern at rest or during physiological stress or movement. If an active TrP is provoked by using pressure or traction (stretching) or needling, this mechanical stimulation reproduces the pain (localized or referred) familiar to the patient. In contrast to this, latent TrPs are not spontaneously painful at rest or during physiological strain/exercise; latent TrPs are clinically silent. Not until it has been provoked by strenuous pressure can the pain mostly referred pain - be triggered, although the patient is not familiar with this from his/her everyday experiences. Latent TrPs can demonstrate all the clinical properties of an active TrP – with one exception: It is not possible to reproduce the symptoms emanating from the latent TrP.

- Depending on the manner and time of occurrence of a TrP, primary and secondary TrPs are differentiated into synergists and antagonists and satellite TrPs (arising in the referred pain zone of a primary TrP).
- If a TrP is in the muscle tissue, it is described as a myofascial trigger point. If a TrP lies in a tendon, a ligament, or in the periosteum, etc., it is known, respectively, as a tendinous, ligamentary, or periosteal TrP.

Pathophysiology

Myofascial trigger points (mTrPs) are nowadays a scientifically thoroughly researched phenomenon in the field of neuromusculoskeletal medicine.

There is pathophysiological evidence of localized hypoxia in the center of an mTrP (Brückle et al. 1990), a changed EMG potential, which can be interpreted as a sign of the malfunction of motor endplates (Travell & Simons 1999), and characteristic changes in the biochemistry. In the immediate surroundings of an mTrP, the concentration of substance P and CGRP, bradykinin, serotonin, norepinephrine (noradrenaline), tumor necrosis factor- α (TNF- α), as well as interleukin (IL)-1 β , IL-6, and IL-8 are markedly elevated, while the pH value is definitely reduced (Shah et al. 2005, 2008). The low pH value (5.4 instead of 6.6) and the two to four times increased concentration of pain and inflammatory mediators (compared to the reference tissue without active mTrPs) lead to a change in nociceptor activity in the sense of peripheral sensitization.
Rigor complexes are histomorphologically recorded in the nucleus zone of mTrPs (myosin and actin filaments remain in maximally close position) with reactive overextension of the bordering sarcomeres (Simons & Stolov 1976) and intramuscular connective tissue changes (Feigl-Reitinger et al. 1998).

The pathophysiological changes are like individual mosaic stones that fit together to form a picture. The factors which combine in the formation of mTrPs are summarized in the "energy crisis model" (Fig. 5.7.1) (Travell & Simons 1999; Mense et al. 2001).

Dysfunctional motor endplates (characterized by low-threshold distribution of acetylcholine; Fig. 5.7.1, Arrow A) or traumatic damage to the sarcoplasmic reticulum (by strain, traumatic overextension, or direct injury of a muscle with partial rupture of the sarcoplasmic reticulum; Fig. 5.7.1, Arrow B) cause a permanent contraction of locally restricted muscle fiber sections (contraction knot). Contraction knots compress the local blood vessels and the reduced perfusion (local ischemia) causes a local oxygen deficit (hypoxia). The permanent contraction in the contraction knot is associated with an increased energy requirement (ATP) and leads to a locally restricted energy crisis (ATP deficiency) with the local hypoxia.

Local ischemia, which leads to local hypoxia, prevents the synthesis of sufficient adenosine

triphosphate (ATP) in the muscle tissue. As a result of the ATP deficiency, the calcium ion pump fails (so that the contraction process in the muscle continues constantly – which also exhausts the available ATP) and the "softening effect" of the ATP, which is necessary for the solution process of the myosin head of the actin filaments, cannot function. Myosin and actin filaments therefore remain interconnected (rigor complex). Persistent rigor complexes in locally restricted areas of muscle fibers are the pathophysiological substrate of a myofascial trigger point. The muscle fiber sections bordering the shortened sarcomeres are overextended and lengthened as compensation. The affected muscle fibers are overall shortened and palpable as taut bands.

The local ischemia leads to ischemically caused hypoxic tissue alterations with local inflammatory processes. Inflammatory processes run through regularly differing phases and end in each case with the formation of a connective tissue scar. The connective tissue draws together and prevents the decontraction of the shortened sarcomeres. This is the first chronification stage where there are myofascial pain problems (Dejung 2009). Connective tissue shortening and changes (pathological crosslinks) gather both intramuscular collagenic tissue (endomysium, perimysium) and muscle fascia and intermuscular collagenic tissue over time (i.e., with chronic myofascial pain syndromes). Histomorphological examination shows that in muscle tissues with mTrPs, the endomysial



Fig. 5.7.1 • Model of energy crisis for the origin of mTrPs. From Travell & Simons, 1999, with permission.

Fig. 5.7.2 • In muscle tissue with mTrPs, the endomysial spaces are always narrower than in controls without mTrPs. Electron microscope image, enlarged 300 times. (A) Muscle tissue with mTrP: shrunken endomysium. (B) Control without mTrPs: endomysium normal. From Feigl-Reitinger et al., 1998, with permission.



spaces between the individual muscle fibers are always narrower than in controls without mTrPs (Feigl-Reitinger et al. 1998; Fig. 5.7.2).

Local ischemia acts as a nociceptive stimulus and leads to the release of sensitizing substances; myofascial pain is thus ischemic pain (Dejung 2009).

Clinical symptoms

Disorders directly induced by mTrPs reveal themselves in the form of:

- Pain (local and referred) with manifold qualities (dragging, stabbing, burning, or dull, definitely delimited or diffuse, superficial or "deep in the joint", etc.). The trigger point activity is sometimes expressed in the form of paresthesia, dysesthesia or hypoesthesia (tingling, burning, feeling "as if restricted by a tight cuff" or feeling "something is swollen", numbness, etc.).
- Motor function disturbance: reflex muscle weakness and muscle weakness caused by pain without atrophy and intramuscular and intermuscular coordination disorders are directly caused by mTrPs (Travell & Simons 1999; Lucas et al. 2004; Ivanichev 2007).
- Autonomic and trophic disorders: frequently deriving from the autonomic phenomena of mTrPs (Travell & Simons 1999). They can become apparent in any number of ways, both in the area of the trigger point itself and in the area of

the referred pain: Increase of skin temperature in the area of the mTrP, changes to the skin temperature and metabolism in the area of the referred pain, increased sweat secretion, nausea or dizziness, etc. They are interpreted as reflex responses of the sympathetic nervous system (Dejung 2009).

Taut bands that arise in connection with the trigger point pathology and connective tissue changes can for their part cause a series of problems. Such disorders indirectly induced by mTrPs include, for example:

- Disorder of intramuscular and intermuscular coordination: economy of movement is prevented by taut bands and connective tissue changes. As a result, this leads to poor posture and strain of the muscles and joints.
- Restricted movement: taut bands lead to shortening of the muscles, which in turn leads to reduced mobility and articular dysfunction (Lewit 2007). Fascia adhesions between neighboring muscles often cause drastically restricted mobility.
- Perfusion disorders: if the taut bands compress the blood vessels, this leads to perfusion disorders (formation of edema) and trophic/metabolic disorders.
- Neuromuscular entrapment: neural structures perforate the muscles at many sites. If the muscle fibers at these sites are tense as a result of mTrPs, they exercise pressure on the nerve structure. The nerve tissue is less well perfused

and this leads to symptoms in the supply area of the neural structure (dysesthesia, weakness, metabolic disorder/trophism).

- Irritation of deep sensitivity, proprioception and nociception: connective tissue dysfunction alters the flow of impulses which come from the receptors which lie in the connective tissue of the muscle.
- Peripheral chronification: connective tissue contractions overlie and fix the rigor complex, which means peripheral chronification of myofascial pain.

All the disorders induced by mTrPs – directly and indirectly – are described as myofascial syndrome (MFS).

Diagnosis

Manual palpation is the most commonly used method of identifying mTrPs in everyday clinical practice. Diagnosis by palpation is based on three main criteria (Travell & Simons 1999):

- Identification of the taut band belonging to the mTrP.
- Finding the most tender spot along the taut band.
- Reproduction of the pain pattern and other symptoms recognized by the patient on mechanical provocation of the mTrP (pressure, traction, needling).

Other characteristics occur with many, but not all, mTrPs and serve to confirm the diagnosis. These may include palpable knots at the site of the mTrP, referred pain, or a local twitch response to mechanical stimulation of the mTrP.

The reliability of the clinical diagnosis of mTrPs has been tested in various studies. It was revealed that the intertest reliability of the identification of mTrPs varies a great deal and indeed depends on the knowledge and experience of the therapist. The kappa values differ a great deal depending on the study and they range from poor reproducibility (k = 0.35) for nontrained and/or inexperienced therapists (Nice et al. 1992; Wolfe et al. 1992) to moderate (Njoo & van der Does 1994; Hsieh et al. 2000) and excellent reproducibility (k = 0.8) for specifically trained therapists and those experienced in palpation (Gerwin et al. 2007; Licht et al. 2007).

At the center of trigger point pathology lies a locally pronounced hypoxia (in particular an energy crisis; Fig. 5.7.1). Various causes can lead to oxygen deficiency and reduced energy (ATP deficiency) in the muscle tissue and as a result to persistent rigor complexes respectively contraction knots. The most common etiological factors can be summarized in the following categories:

- Direct trauma (e.g., immediate muscle injury as a direct result of violence in sport, accidents, etc.).
- Acute overextension of the muscles (e.g., caused by sport or accident).
- Acute strain (e.g., caused by sport or accident).
- Chronic strain of the muscles (e.g., caused by posture, repetitive movements at work or in training, long-term contraction in approximate position, eccentric muscle activity, stress-induced strain, etc.).
- Trigger point activity in other muscles (trigger point chains with secondary TrPs or satellite TrPs).

The above etiological factors frequently lead initially to the occurrence of latent TrPs, which are clinically silent. Latent TrPs can be activated by the further effect of mechanisms of origin but also by contributory factors such as cold, wet, draught, stress, etc. which do not demonstrate a damaging effect in healthy muscle tissue (so-called mechanisms of activation; Fig. 5.7.3). Active TrPs for their part can be retransformed by deactivation processes (such as rest, the body's own regeneration processes, therapy) to latent TrPs or healthy muscle tissue (Fig. 5.7.3).

Chronic myofascial pain is largely caused by a combination of various factors, when predisposing (e.g., poor level of fitness with reduced stamina), causative (e.g., acute strain) and perpetuating factors (e.g., incorrect posture at work, trigger point activity in synergists or antagonists, reduced endogenous deactivation mechanisms) frequently work together (details in Gautschi 2010).

Fascia and myofascial trigger points

Fascial tissue – in the widest sense – comprises all fibrous and collagen connective tissue formations (see Part one Anatomy). Fascial structures thus form





a network that surrounds and perforates the whole body and all the organs, and is organized in many different ways into pockets and chambers and connects everything with everything else. The different developments in the form of diversified septa, surrounding interlaced fibers, ligamental and capsular thickening, etc., can be understood as local adjustments of a coherent network to specific localized tension (Schleip 2009). Not only the outer muscle fascia (epimysium) but also the thin intramuscular connective tissue structures (such as the endomysium which surrounds each individual muscle fiber and the perimysium which surrounds the whole muscle fiber bundle) belong to this fascial network (Trotter 1992).

The muscle is structurally thus inseparably linked with the fascia organ, or to put it more specifically: It *is* a part of the fascia organ. In the myofascial system, the contractile elements of the muscles dynamize the fascial network and thus affect both optimum pretension of the tension elements of the tensegrity structure (see Chapter 3.5) and the movement of the whole system. The muscle cells, therefore, move around in the fascial network, so to speak like fish in a fishing net. Their movement exercises traction on the fascial structures which transfer into the periosteum, whereby the tensile force is transferred to the bones. Looked at from this angle, there is only one muscle, which "loafs around" in 600 or more fascial pockets (Myers 2001).

The fascia and the muscles are, therefore, in immediate interrelation. They mutually induce each other to share a common fate – in both good times and bad.

Dysfunctional fascia structures can in this manner provoke or maintain dysfunctions of the muscles (mTrPs) (see later), just as muscular pathology in the form of mTrPs always has a fascial component as well and can be the cause of fascia dysfunction (see later) (Fig. 5.7.4).

Fascia-induced muscle dysfunction

Pathological changes to the fascial structures can be caused by a number of factors: e.g., inflammatory processes, mechanical strain, metabolic dysfunction, injury, etc. (Fig. 5.7.4, Arrow 1; see also Part Four Physiology). Once fascia dysfunction has occurred, it can lead to muscular problems in particular mTrPs (Fig. 5.7.4, Arrow 2).



Fig. 5.7.4 • Interrelation between fascia dysfunction and myofascial trigger points.

- 1. Factors which lead to fascia dysfunction.
- 2. Fascia dysfunction as possible cause of the origin/maintenance of mTrPs.
- mTrPs as possible cause of the origin/maintenance of fascia dysfunction.
- 4. Factors which lead to the origin of mTrPs.

Fascia dysfunction in its role in the origin and activation of mTrPs

Dysfunctional fascia can contribute in different ways to the origin and maintenance of mTrPs.

Strain as a result of disorder of the fascia mechanics

Intramuscular and extramuscular connective tissue changes cause alterations to the pattern of movement: Pathological crosslinks reduce the elasticity of the muscle connective tissue (Fig. 5.7.5); intramuscular shortening, narrowing, and adhesion of fascial structures (endomysial and perimysial structures, see below) affect the intramuscular coordination and local metabolic supply; extramuscular fascia shortening and intermuscular fascia adhesions lead to restricted movement and disorders of intermuscular interaction. One-sided stress or strain does not remain locally restricted but spreads globally in the myofascial network – following the dynamics of tensegrity architecture (see chapter 3.5).

Fascia changes – whatever the reason they arise – regularly lead to changed posture and movement



Fig. 5.7.5 • Distortion of the connective muscle tissue collagen network. (A) Normal situation relaxed. (B) Normal situation stretched. (C) Situation with pathological crosslink relaxed. (D) Situation with pathological crosslink stretched. From van den Berg, 2003, with permission.

patterns. The resulting deviations from economic weight-bearing patterns create uneven strain on the musculoskeletal system so that myofascial (and also articular) structures are very probably in a chronically incorrect posture or chronically strained. Chronic overload is one of the most common etiological factors for the origin and activation of mTrPs.

Myofascial dysfunction frequently leads not only locally to the occurrence of mTrPs in individual muscles. As a result of persistent incorrect tension, tendinous TrPs often form in the muscle tendon transition zones and periosteal TrPs in the area of the insertion zone of the muscle. This is how multiple mTrPs form along structural or functionally kinetic chains (see Chapter 3) in synergistically active muscle groups.

Changes in sensory input

Fascia have an important task as a receptor organ (Chapter 2) and fascia dysfunctions always lead to a changed flow of impulses from the fascial mechanoreceptors as well. Fascia disorders therefore change sensory function (interoception, proprioception).

The control of motor function is an integrative sensorimotor provision and the sensory input which comes from the myofascial unit is important in the generation of motor function output. A changed sensory impulse flow therefore changes control of the muscles, which leads to muscular strain and can favor the origin and the activation of mTrPs.

There is evidence that a changed muscle activation pattern is a result of trigger point activity (Lucas et al. 2004). Whether and to what extent this could stand in connection with a changed sensory provision of fascial receptors has not yet been investigated.

It is just as obvious that autonomic regulation in the muscle is also modified by a change in sensory information. Changes in perfusion and metabolism are possible consequences.

Autonomic disorders

Local perfusion and metabolism of muscle tissue can be permanently disturbed by fascia dysfunction. Changes in sensory input (see above) can be just as much the cause as direct mechanical influences.

The superficial fascia of the muscle must ensure access for the nerve and vascular structures which supply the muscle (and enable optimum muscle function). Nerves, arterial and venous blood vessels all pass through the muscle fascia (perimysium) as



Fig. 5.7.6 • Perforating triad as it passes through the surface fascia of a muscle. Diagram of a hole in the fascia with "perforating triad". In each case a vein (perforating vein), an artery (perforating artery) and a nerve (perforating nerve) penetrate the surface of the fascia of a muscle. From Staubesand, 1994, with permission.

"perforators" (Staubesand 1994; Staubesand & Li 1996; see Fig. 5.7.6). If the resistance of the superficial fascia of a muscle is increased, the neural and vascular structures at these perforator sites can be compressed.

Such entrapment of the most distal nerve sections by the superficial fascia of a muscle restricts the optimum function of the nerve – and thereby the muscle. Motor function, sensory and autonomic nerve fibers can be affected; on the one hand, strength, coordination and mobility of the muscles are reduced; at the same time, the irritation of vasomotor fibers can change the perfusion of the muscle and with it reduce its capacity for regeneration.

As arterial and venous blood vessels usually perforate the superficial fascia of the muscle together with the nerve, the perfusion and regeneration potential of the muscle can also be directly affected in this way.

Such distal entrapment of autonomic nerve fibers or blood vessels can permanently reduce the regeneration capacity of the muscles. The strain limit of the muscles is thus reached earlier, which favors the occurrence of mTrPs. As long as these entrapments remain, they can prevent or weaken the positive effect of any treatment – both passive and active rehabilitation.

With the release of such mini-entrapments in the area of the perforators by using targeted therapy (see below), one of the possible factors for maintaining a neuromusculoskeletal problem can be eliminated: Motor function, sensory and autonomic nerve fibers are no longer irritated. This is the optimal prerequisite for the best possible innervation (both motor and sensory) as well as perfusion of the muscle, making it fully functional again.

Peripheral chronification

Intramuscular and extramuscular fascia changes play an eminently important (and until now underestimated) role as chronification factors in the periphery. On the one hand, they can work continually as mechanisms for the origin and activation of musculoskeletal problems (see above and Fig. 5.7.3). On the other hand, fascial disorders frequently prevent the spontaneous remission of mTrPs: The endogenous, autonomic deactivation mechanisms (Fig. 5.7.3) are prevented by fascia dysfunctions. Pathological crosslinks, connective tissue shortening, and adhesions mechanically affect the recovery of myofascial structures, while the irritation of the fascial receptor system changes the sensory input (and with it causes a faulty motor function output). Furthermore, as a result of minientrapments in the area of dysfunctional superficial fascia of the muscles, the local metabolism is restricted - and with it the regeneration potential of the muscle tissue.

It should be particularly emphasized that intramuscular connective tissue changes probably play a crucial role in the chronification of myofascial problems. Myofascial pain usually occurs in connection with ischemic damage stimuli in the muscle (see above). Pronounced ischemia in the area of the mTrP can cause localized tissue necrosis. This ischemically caused necrosis causes localized inflammatory processes, with reparations and collagen fibers deposited in the course of wound healing. Freshly formed connective tissue begins to contract under the influence of myofibroblasts (van Wingerden 1995). The connective tissue then goes through a shrinking phase, whereby inflammatory mediators strengthen the activity of the myofibroblasts and a low pH value (acid milieu) increases the contractility of the myofibroblasts (Pipelzadeh & Naylor 1998); the whole process ends with the formation of a scar. Dejung (2009) postulates that endomysial and perimysial connective tissue shortening occurring in such a manner in the TrP region covers the contracted sarcomeres and fixes their structure. The connective tissue draws together and prevents the decontraction of the sarcomeres of the rigor complex. This is the first stage of myofascial chronification; i.e., chronification which represents peripheral not central – chronification.

Trigger point-induced fascia dysfunction

On the one hand, fascia dysfunction can cause the occurrence and persistence of mTrPs (see above; Fig. 5.7.4, Arrow 2). On the other hand, mTrPs cause fascia dysfunction (Fig. 5.7.4, Arrow 3).

Mechanically-induced fascia dysfunction

mTrPs always occur in association with taut bands. Taut bands induce adjustment and often disorders of the fascia architecture and function.

- Taut bands put fascial structures under permanent tension (especially in the area of the transition from muscle to tendon and the muscle insertion sites) so that at these sites tendinous or periosteal TrPs often occur ("insertion tendinosis").
- Taut bands can reveal themselves in the form of muscle shortening. Restricted mobility and altered movement and posture patterns appear and can lead to adaptation processes and the decompensation of fascial structures.

Biomechanically-induced fascia dysfunction

There are marked localized changes in the biochemical milieu in the area of mTrPs. There is evidence of:

- Pronounced hypoxia (Brückle et al. 1990).
- A definite increase in inflammatory mediators such as bradykinin, substance P and CGRP (Shah et al. 2005, 2008).
- A significantly lower pH value; i.e., the tissue milieu is predominantly acidic (Shah et al. 2005, 2008).

These recorded changes in the biochemical milieu favor the origin and perpetuation of fascia dysfunction. This tendency to contraction of the fascia is strengthened in an acid tissue milieu and by messenger material associated with inflammation in the body (Pipelzadeh & Naylor 1998). The special significance of the inflammatory process has already been pointed out, being itself a reaction to ischemic tissue necrosis leading to fascia dysfunction in general and to peripheral chronification of myofascial problems in particular.

Therapeutic consequences

The causal therapy of (myo)fascial dysfunction is carried out based on the underlying (patho)physiology. In short, localized ischemia leads to localized hypoxia, which is at the center of the pathogenesis of myofascial pain and function disorders. The consequences of hypoxia are:

- ATP deficiency (energy crisis), which leads to the rigor complex (lack of the soft-tissue-making effect of ATP).
- Localized inflammatory processes, which cause connective tissue reactions (adhesions, shrinkage) (see above).

Myofascial trigger point pathology which has become chronic is characterized by two factors: Rigor complexes and connective tissue changes (adhesions, shortening), where the latter are largely responsible for the chronification of a myofascial problem (see above). Treatment which will be permanently effective must take both factors into account – rigor complexes and connective tissue changes.

Muscle release techniques which work exclusively on the reflexes or are only directed at the treatment of the rigor complex (dry needling, shock wave therapy), insufficiently affect the fascial aspect. Consistent and thorough treatment of the rigor complex and the changed fascial structures, using manual techniques targeted on the connective tissue, is necessary. It is a sole characteristic of the myofascial trigger point therapy IMTT[®] founded by Dejung (1988, 2009) that the rigor complex and the connective tissue changes are equally at the center of the therapeutic intervention (Swiss Approach). Four manual techniques are used to target treatment on both the rigor complex itself and the reactive changes to fascia structures (Table 5.7.1). Manual techniques are supplemented in the concept of myofascial trigger point therapy by stretching (technique V), functional strengthening of the muscles, and ergonomic measures (technique VI). Exercises at home to stretch/relax interrupt monotonous work postures and promote the regeneration capacity of the muscle fibers and the remodeling of fascial structures. Functional training supports the healing process thanks to physiological weightbearing and exercise and makes the myofascial unit more able to bear weight, while ergonomic interventions reduce incorrect loading. For permanent treatment success of chronic myofascial

Technique	Measures	Local tissue-specific therapeutic effects
I	Manual compression of the mTrP	Squeezing out the "inflammatory soup" and the local edema Reactive hyperemia following ischemia \rightarrow Increase in metabolism Relaxation reflex of the taut band belonging to the mTrP
II	Manual stretching of the TrP region	Squeezing out the "inflammatory soup" and the local edema Reactive hyperemia following ischemia \rightarrow Increase in metabolism Relaxation reflex of the taut band belonging to the mTrP Destruction of the local rigor complex Extension of the reactive connective tissue adhesions (pathological crosslinks) and shortening: \rightarrow Improvement in intramuscular supply and suppleness
III	Fascia stretching technique (Manual stretching of the superficial and intramuscular fascia)	$\begin{array}{l} \mbox{Loosening of the reactive connective tissue adhesions (pathological crosslinks)} \\ \mbox{and shortening:} & \rightarrow \mbox{Improvement in intramuscular mobility and supply} \\ & \rightarrow \mbox{Muscle more easily stretched} \\ \mbox{Stimulation of fascial mechanoreceptors} & \rightarrow \mbox{Relaxation reflex of the taut band} \\ \mbox{belonging to the trigger point} \\ & \rightarrow \mbox{Depression of sympathicus activity, depression of global tone} \end{array}$
IV	Fascia separation technique (Manual loosening of intermuscular fascia adhesions)	Loosening the adhesions between fascia of neighboring muscles \rightarrow Improvement in intermuscular mobility
V	Stretching, relaxation	Improvement of muscle stretchability
VI	Functional training, ergonomy	Supporting physiological strain and movement, the regeneration process and making the muscles more resilient Ergonomy reduces incorrect weight-bearing on the muscles

- דמטוב ס.ר.ד - דוועעבו טטוווג נוובומטע וסעוסס מטטוטמטווז. דובמנווובווג נבטוווועעבס מווע נוובוו וטטמו בוובטנס (ווטווו עמענסטווו בטנ	Table 5.7.1	Trigger point therapy	(Swiss approach):	Treatment technic	ues and their local	l effects (from Gautschi 2008
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pain, additional to local therapy of the myofascial structures, the perpetuating factors also have to be recognized and included in the treatment. Myofascial trigger point therapy IMTT[®] is a differentiated method being performed by specially trained physiotherapists and physicians. Further information is available at www. triggerpunkt-therapie.eu.

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Fascia-related disorders: Hypermobility

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Introduction

Fascia-related disorders are defined as disorders resulting from altered stiffness or compliance of the fascia. Stiffness is defined as the actual change in force for a given change in length (i.e., $\Delta F/\Delta l$). Compliance is the inverse of stiffness (i.e., $\Delta l/\Delta F$). Increased compliance of fascia yielding hypermobility occurs in various inherited connective tissue disorders (e.g., Ehlers–Danlos syndrome (EDS), Marfan syndrome, cutis laxa, and osteogenesis imperfecta). These multisystem disorders are characterized by varying involvement of vessels, skin, joints, bones, internal organs, eyes, muscle, and the peripheral and central nervous system.

This chapter will focus on changes in muscle function related to increased fascia compliance in Marfan syndrome and, particularly, EDS. After a short clinical description of these disorders, a description of neuromuscular involvement in both will be given, and then a discussion of results of studies regarding effects of increased fascial compliance on muscle characteristics in EDS.

Clinical features of EDS and Marfan syndrome

EDS is a clinically and genetically heterogeneous group of inherited connective tissue disorders characterized by joint hypermobility, skin hyperextensibility, and tissue fragility. In a minority of patients, EDS is caused by changes in extracellular matrix molecules collagen types I, III, V, lysyl hydroxylase, and tenascin-X ground remains unresolved (Beighton et al. 1998). The revised classification of EDS into six major types is based on clinical and biochemical features (Beighton et al. 1998). These types are: the classical, hypermobility, vascular, kyphoscoliotic, arthrochalasia, and dermatosparaxis types. The hypermobility type is most common in EDS, followed by the classical type. Together, they account for approximately 90% of all cases (Steinmann et al. 2002). The vascular type is far less common, but is associated with the occurrence of arterial dissections (i.e., tears within the wall of blood vessels, which allow blood to separate wall layers) and aneurysms (i.e., localized, blood-filled dilation: balloon-like bulge of a blood vessel caused by disease or weakening of the wall), which may cause severe neurological complications. The kyphoscoliotic, arthrochalasia, and dermatosparaxis types are rare. In 2001, a new autosomal recessive type of EDS caused by deficiency of TNX was identified (Schalkwijk et al. 2001). Subsequently, TNXB gene haploinsufficiency was found to be associated with the hypermobility type of EDS in a minority of patients (Zweers et al. 2003).

(TNX); however, in most patients the genetic back-

Marfan syndrome is another inherited connectivetissue disorder characterized by varying patterns of organ involvement, including the cardiovascular and pulmonary systems, eyes, skeleton, skin, and dura. At least 91% of all Marfan patients fitting clinical diagnostic criteria show mutation in the fibrillin-1 gene (*FBN1*) on chromosome 15, and 27% of the mutations are spontaneous (Loeys et al. 2004). Many symptoms do not present until puberty or later, and severe complications rarely develop before adulthood. With the availability of elective cardiac surgery and management by expert centers, mean life expectancy of Marfan patients increased from 32 years in 1972 to 41 years in 1993, and probably even further since then, approaching normal life expectancy (Silverman et al. 1995; Pyeritz 2009). Accompanying increasing life expectancy, there is a shift from life-threatening cardiovascular involvement to more chronic and less serious complaints such as ocular and orthopaedic disorders, and possibly, neuromuscular complications (Dean 2007; Hasan et al. 2007; Jones et al. 2007; Ramirez & Dietz 2007; von Kodolitsch & Robinson 2007; Voermans et al. 2009a).

Physical or manual therapy in EDS and Marfan syndrome focuses on reduction of musculoskeletal pain, maintenance of muscle force, increase of joint stability, reduction of scoliosis, and functional improvement (Braverman 1998). We will not discuss these therapeutic options in this chapter.

Neuromuscular involvement in EDS and Marfan syndrome

Overlooked for a long time, neuromuscular involvement in EDS and Marfan syndrome is expected, based on interactions between muscle fibers and extracellular matrix (ECM) molecules (Voermans et al. 2008). Muscle hypotonia, muscle rupture, fatigue, and musculoskeletal pain are included in the diagnostic criteria of EDS (Beighton et al. 1998), and we recently reported mild-to-moderate muscle weakness in EDS patients (Voermans et al. 2009b). Furthermore, muscle involvement in Marfan syndrome has lately received renewed attention, probably due to increasing insight into the role of ECM molecules in muscle pathophysiology (Behan et al. 2003; Cohn et al. 2007; Voermans et al. 2008). We will discuss this in more detail below.

Recently, mild-to-moderate neuromuscular involvement in a large proportion of EDS patients has been demonstrated (Voermans et al. 2009). Neuromuscular involvement was defined as consistent abnormal findings on questionnaires and/or physical examination, supported by abnormal results of appropriate ancillary investigations. Muscle weakness, myalgia, and rapid fatigability were reported by the majority of patients. Mild-to-moderate muscle weakness (estimated using manual muscle strength testing and with hand-held dynamometry) and mild reduction of vibration sense on physical examination were common (85% and 60%, respectively). Nerve conduction studies revealed axonal polyneuropathy in five patients (13%; predominantly occurring in the TNX-deficient type). Needle electromyography showed mainly myopathic features in nine patients (26%), and a mixed neurogenic-myopathic pattern in most (60%). Muscle ultrasound revealed increased echo intensity (48%) and atrophy (50%). Mild myopathic features (i.e., increased variation of fiber diameter, sporadic isolated atrophic fibers, and a mild increase of internal nuclei) were seen on muscle biopsy of five patients (28%). Creatine kinase was mildly elevated in four patients. Furthermore, patients with complete absence of TNX (i.e., TNXdeficient type EDS) are associated with more severe neuromuscular symptoms than those with reduced TNX serum levels and with reduced staining of TNX within muscle (i.e., hypermobility type EDS caused by TNXB haploinsufficiency). An inverse relation between residual TNX quantity in serum and muscle and degree of neuromuscular involvement is suggested by these results (Voermans et al. 2009b.) They also confirm the findings of previous case reports on neuromuscular symptoms in EDS (Bilkey et al. 1981; Banerjee et al. 1988; Bertin et al. 1989; Takaluoma et al. 2007; Voermans et al. 2007.)

In addition, a recent study on 10 Marfan syndrome patients showed neuromuscular involvement in the majority, albeit with variable severity (Voermans et al. 2009b). Four older patients reported muscle weakness, confirmed by physical examination. Furthermore, five patients had mild-to-moderate reductions of vibration sense, and all older patients reported mild functional impairments. Axonal polyneuropathy was found in four patients and electromyography myopathic and neurogenic changes in all patients, compatible with both myopathy and lumbosacral radiculopathy. Increased echo intensity and atrophy on muscle ultrasound was found in over half of the patients. Muscle biopsies (two patients) showed myopathic changes only in the older, female patient (Voermans et al. 2009a). Review of MR images showed lumbosacral dural ectasia in combination with lumbosacral spinal meningeal cysts in seven patients, with one patient showing an additional thoracolumbar kyphoscoliosis. Creatine kinase was not elevated. Previous cardiovascular complications (endocarditis, mitral-valve prolapse, and aortic root/ascending aorta dilatation) and the medication used (β -blocker) may have contributed to experienced fatigue reported by the Marfan patients. Furthermore, simvastatin, a lipid-lowering drug, can cause myalgia with signs of myopathy, and increases the risk of polyneuropathy (Gaist et al. 2002). However, results of previous studies suggest that cardiovascular disease and side effects cannot fully explain muscle weakness in Marfan patients (Percheron et al. 2007). Results from our study suggest that neuromuscular symptoms in Marfan syndrome are directly associated with moderate-to-mild myopathy and/or polyneuropathy, and the presence of dural ectasia with spinal meningeal cysts was related to lumbosacral radiculopathy. Myopathy in Marfan syndrome might be linked to abnormal fibrillin in muscle connective tissue (endo-, peri- and epimysium) (Behan et al. 2003: Voermans et al. 2009a), which results in excessive transforming growth factor β signaling, which contributes to abnormal muscle-cell development and reactions to injury or inflammation (Neptune et al. 2003; Cohn et al. 2007).

The results regarding inherited connective tissue disorders raise the question whether relatively mild myopathic changes in muscle biopsies in the minority of patients suffice to explain the mild-to-moderate neuromuscular involvement in the majority of EDS and Marfan patients. Non-neuromuscular features of EDS and Marfan syndrome may contribute to various neuromuscular symptoms: e.g., musculoskeletal pain, increased fatigability, and mild impairment may be caused by articular and skeletal problems in EDS (Voermans et al. 2009b). Cardiovascular symptoms and medication in Marfan patients will probably contribute to easy fatigability (Voermans et al. 2009a).

The finding of a close relationship between residual TNX levels and degree of neuromuscular involvement in EDS, and previous studies on the role of fibrillin and excessive transforming growth factor β signaling in Marfan syndrome, suggest another pathophysiological mechanism may take part. The abnormal ECM composition in these disorders may affect muscle physiology (i.e., as a result of increased compliance) or biochemically (i.e., altered signaling by ECM molecules).

Effects of TNX-deficiency on muscle characteristics in a mouse model of EDS

Abnormalities in muscle ECM composition may affect myofascial force transmission, which may contribute to muscle weakness in EDS (Voermans et al. 2007; 2009b) (for details on myofascial force transmission, see also a dedicated chapter within this book and Huijing (2007)). Effects of altered myofascial force transmission may help to understand why muscle force is mildly to moderately reduced in the majority of EDS patients, whereas conventional histological analysis of muscle shows only mild abnormalities in a minority of patients.

TNX is an ECM glycoprotein abundantly expressed in various tissues during embryonic development (e.g., tendons and perimysium of skeletal muscle (Matsumoto et al. 1994; Burch et al. 1995)). In adults, TNX is predominantly expressed in connective tissues of skeletal and cardiac muscle (Matsumoto et al. 1994) and is involved in collagen deposition and maturation (Schalkwijk et al. 2001; Egging et al. 2006). Several studies suggest that TNX acts as a bridge between collagen fibrils that are organized in bundles (Lethias et al. 1996), and may be important for enhanced stiffness of connective tissues (Lethias et al. 2006). The chemical structure of the TNX molecule (disulfidelinked trimer structure) is important for bridging. TNX interacts with types I, III and V fibrillar collagen molecules and with decorin, and binds to the fibrilassociated types XII and XIV collagens (Lethias et al. 2006). Finally, the FNIII domains of the TNX molecule may be important for elastic properties of the molecule itself (Lethias et al. 2006). The ultrastructure of the muscle ECM in TNX knockout mice is distorted. Preliminary results of collaboration with Dr. Delage in Bordeaux (as yet unpublished work), applying various electron microscopic analysis techniques, point at changes of collagen structures within myofascicles, and altered myofiber organelles are observed. Much more work on this is indicated.

TNX knockout mice have so far been used for detailed dermatological phenotyping and dermatological features of TNX-deficient type EDS (Mao et al. 2002; Bristow et al. 2005; Egging et al. 2007a, b). Additionally, articular and obstetrical aspects were investigated (Egging et al. 2006, 2008).

To investigate the effects of TNX-deficiency on muscle characteristics we used these mice to measure muscle force (Huijing et al. 2010).

During an isometric contraction the muscletendon complex length is set a constant value. However, actual length of active myofibers is dependent on the properties of the series elastic component (SEC). Note that, due to the effects of myofascial force transmission, SEC is not only determined by aponeuroses and tendons, but also by the *intra*muscular network of endo-, peri-, and epimysium in the case of a fully dissected muscle, as well as by the *inter*muscular connective tissues in the case of a muscle working within its full connective tissue context. In fact, sarcomeres within myofibers are linked to these types of SEC elements (tendinous and fascial connective tissues), which themselves are arranged mechanically in parallel to each other. Total compliance of SEC affects the rate of length change of the myofibers during the initial phase of isometric force exertion, and hence affects the rate of force build-up in time. The ratio of compliance of myofascial and myotendinous pathways will determine what percentage of the force exerted is transmitted in each pathway. In any case, the least compliant (i.e., stiffer) pathway will transmit most force. Vice versa, increased fascia compliance occurring in inherited connective tissue disorders is hypothesized to yield reduced myofascial interaction between muscles.

We tested this hypothesis in a study focused on both *intra*muscular aspects (force–time characteristics of *maximally dissected* medial gastrocnemius muscle (GM)) and *epi*muscular aspects (length– force characteristics of triceps surae muscle (TS) and anterior crural muscles (TA + EHL and EDL)) *without major dissection* to detect changes in mechanical interaction between muscle groups of both TNX knockout and wild type mice. The experimental set-up is summarized in Fig. 5.8.1A,B.

The results show that altered properties of the SEC of muscle due to TNX deficiency affect characteristics of muscle function. More specifically, study of the *intra*muscular aspects points to changes in the SEC within the (maximally dissected) muscle-tendon complex. Results of the study of



Fig. 5.8.1 • Experimental set-ups and design. The following protocols were used to assess length–force, stimulation frequency, force–velocity, and fatigability characteristics. (A) Intramuscular aspects: maximally dissected medial gastrocnemius. The sciatic nerve was dissected free and its branches were cut, except for that innervating medial gastrocnemius (GM). The femur was clamped. GM was fully dissected, except for blood supply and innervating nerve. The distal GM tendon was connected to a force transducer. Length changes of GM were induced using a computer-controlled servomotor and contractions were induced by electrically stimulating the sciatic nerve to maximally activate all GM myofibers. (B) Intermuscular aspects: minimally dissected anterior crural and triceps surae muscles. Limited fasciotomy was performed distally to expose distal tendons of the tibialis anterior muscle (TA), extensor hallucis longus muscle (EHL) and extensor digitorum longus muscle (EDL), and to sever the retinaculae. The distal tendons of EDL were tied together and severed distally of the knot. Also, the distal tendons of TA and EHL were tied and severed from their insertions. This complex is referred to as TA+EHL complex. The distal tendons of TA+EHL and EDL, as well as the proximal EDL tendon, were connected to force transducers.

For measurement of the TS length–force characteristics, TA+EHL complex was held at a constant muscle–tendon complex length initially corresponding to the length at an active force of approximately 1/3 of optimum force. Similarly, for measurement of the TA+EHL length–force characteristics, TS was held at a constant muscle–tendon complex length initially corresponding to an active force of approximately 1/3 of optimum force. EDL was always kept at constant length and position.

*inter*muscular mechanical interaction show a reduction of myofascial interaction between muscles active within the full context of their surrounding connective tissues. An example is shown in Fig. 5.8.2A.



Fig. 5.8.2 • TNX-deficiency diminishes myofascial interaction between antagonistic muscles. (A) Active and passive length-force characteristics of the triceps surae muscle complex (TS) of healthy (WT) and TNX-deficient rats (TNX-KO). The muscles are operating within their regular connective tissue context. (B) Summed force exerted by the tibialis anterior and extensor hallucis longus muscles (TA+EHL) kept at constant length. In both healthy rats (WT) and TNX-deficient mice (TNX-KO), TA+EHL force is lowered after TS muscle-tendon complex is lengthened [Δ /(m + t)] progressively. However, this decrease in force is much lower (<25%) for diseased rats.

Active force is expressed as percent of initial force (at low triceps surae length).

Note that myofascial force transmission is also present for TNX-deficiency but to a much lower degree.

Affected intra- and epimuscular myofascial force transmission may drastically alter the required muscular coordination in physiological movements and interfere with mechanical interaction between antagonistic muscles (Matsumoto et al. 1994; Huijing 2007; Voermans et al. 2007; Huijing et al. 2008). We will explain these findings below.

Intramuscular changes: increased muscle compliance

At optimum length (λ_0) several properties of dissected GM are unchanged in TNX knockout mice compared to wild type mice: (1) Maximal isometric force and maximal power production; (2) Stimulation frequency–force relationship; and (3) Fatigability during a series of repeated isometric contractions. In contrast, at low lengths (($\lambda_0 - 4, \lambda_0$ -3.5, and $\lambda_0 - 3$), some GM properties were affected significantly in TNX knockout mice: (4) Active force exerted at lower lengths was lower; (5) The maximal rate of relaxation was lower; (6) The time delay between first stimulation pulse and the time of attainment of 2% of maximal active force was longer in TNX knockout mice.

This last finding is consistent with the hypothesis that relatively more slack must be taken up at lower lengths in TNX knockout mice. The other findings are related to the increased SEC compliance present in the disease, which causes more shortening to be imposed on the myofibers in TNX-deficient mice at the onset of contraction at low lengths. Vice versa, more lengthening is imposed on the myofiber at the onset of relaxation at low lengths.

Intermuscular changes: reduced epimuscular myofascial force transmission

The most important result is that TNX deficiency strongly affects myofascial force transmission within the lower mouse limb.

The EDL proximo-distal force difference which constitutes absolute proof of epimuscucular myofascial force transmission is lower in TNX-deficient than in healthy mice. The deficiency significantly affects net epimuscular myofascial force transmission, not only in the magnitude of the net myofascial load on EDL, but also in direction of loading. Whereas in wild type mice the direction changes rapidly with increasing TS length from proximal to distal to distal loading of EDL, this change of direction does not occur in TNX knockout mice.

Mechanical interaction between muscles was decreased in a major way: normalized distal active force of agonistic muscle (TA+EHL with triceps surae lengthening: (Fig. 5.8.2B) and vice versa: not shown) with increasing length of the antagonistic muscle.

It is concluded that TNX knockout muscles act more independently than healthy muscles. It seems inevitable that such altered function will require altered patterns of muscular coordination to allow effective movement.

Taken together, these findings indicate that the SEC of the muscle-tendon complex located within and between muscles is changed in TNX knockout mice. As such, these findings support the hypothesis that TNX deficiency reduces the stiffness of myofascial pathways and thus causes a pathological reduction of the force transmitted this way (Voermans et al. 2007). This study and previous animal experiments have shown that myofascial force transmission occurs between antagonistic muscles, which points to high interdependence of muscles and their role in higher levels of motor organization (Huijing 2007). Whether and to what extent altered myofascial force transmission affects muscular coordination and interferes with mechanical interaction between antagonist muscles in TNX-deficient EDS patients needs to be studied in detail. If neuromuscular control is not optimized for this situation, altered myofascial force transmission may explain symptoms of enhanced fatigability in patients suffering from EDS.

In conclusion, altered muscular function in TNX knockout mice is explained at least in part by changes of myofascial SEC of the muscle-tendon complex, which result in reduced intermuscular myofascial interaction between antagonistic muscles.

A full article on this work is by Huijing et al. (2010).

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5.9

Anatomy of the plantar fascia

Scott Wearing

The plantar fascia, or plantar aponeurosis, forms part of the deep fascia of the sole of the foot and provides a strong mechanical linkage between the calcaneus and the toes. Arising predominantly from the calcaneal tuberosity, the plantar fascia attaches distally, through several slips, to the plantar aspect of the forefoot as well as the medial and lateral intermuscular septa. Anatomically, the fascia can be divided into three bands: the medial, lateral, and central. While the medial and lateral bands are variable in nature, the central aponeurotic band represents the major component of the plantar fascia both structurally and functionally and is often cited as the plantar fascia proper (Sarrafian 1983).

The apex of the plantar fascia proper originates from the plantar aspect of the medial process of the calcaneal tuberosity, where its deep surface also serves as a partial origin for the flexor digitorum brevis. At the mid-metatarsal level, the plantar fascia separates into five longitudinally orientated bands, with each band subsequently dividing to form superficial and deep tracts just proximal to the metatarsal heads. While the two marginal superficial tracts course obliquely to the sides of the foot, the central superficial tracts insert into the skin, anterior to the metatarsal heads and contribute to the formation of the mooring and natatory ligament (Sarrafian 1983). The five strong deep tracts of the central band bifurcate to form medial and lateral sagittal septa, which course anteriorly and deeply to surround the medial and lateral aspect of the digital flexor, before inserting sequentially into the flexor tendon sheath, interosseous fascia, the fascia of the transverse head of the adductor hallucis, the deep transverse metatarsal ligament, and the base of the proximal phalanges via the plantar plate and collateral ligaments of the metatarsophalangeal joints. The most medial pair of septa also insert into the tibial and fibular sesamoids as well as the two heads of the flexor hallucis brevis muscle. By effectively spanning both the medial and lateral longitudinal arches of the foot, the plantar fascia maintains the height of the longitudinal arch in humans.

Biomechanical function of the plantar fascia

The plantar fascia supports the longitudinal arch of the foot during static stance. Metatarsal splaying and a deterioration in arch shape occur with sectioning of the plantar fascia during simulated static stance (Ker et al. 1987), suggesting that the plantar fascia forms part of a transverse and longitudinal tie-bar system within the foot.

During static stance, the medial longitudinal arch has been likened to a truss, with the plantar fascia acting as a tension element, or tie-bar, connecting two compressive elements (Hicks 1955). With weightbearing and internal tibial rotation, elongation of the arch is restricted, in part, by tension within the plantar structures (Sarrafian 1987). While all of the plantar ligaments appear important in restricting movement, Huang et al. (1993), demonstrated that the plantar fascia provided the largest contribution to arch maintenance, with plantar fasciotomy resulting in a 25% reduction in the stiffness of the arch. Interestingly, however, the arch retained 65% of its original stiffness following resection of the long and short plantar ligaments, the spring ligament and the plantar fascia, suggesting that other structures, such as bone geometry, may have the greatest effect on the stability of the medial longitudinal arch during static stance. Nonetheless, during static stance, the plantar fascia is thought to form part of a passive mechanism that is capable of modifying the stiffness of the medial longitudinal arch in relation to the applied load.

Under nonweightbearing conditions, dorsiflexion of the toes has been shown to increase tension within the plantar fascia, resulting in plantarflexion of the corresponding metatarsals and raising of the medial longitudinal arch; the so-called windlass mechanism (Hicks 1954). Under weightbearing conditions, however, such as during static stance, plantarflexion of the metatarsals is resisted by ground reaction force, and elevation of the arch is achieved by a complex movement of supination and external rotation of the foot and lower limb. Such a movement pattern is thought to increase the stability of the arch and activation of the windlass mechanism is clinically believed to be important during the propulsive period of gait.

When toe dorsiflexion is coupled with calf muscle activity, as occurs during terminal stance, internal loading of the plantar fascia may be effectively amplified. Carlson et al. (2000) noted that dorsiflexion of the first metatarsophalangeal joint beyond 30° induced fascial loads in excess of those of the Achilles tendon when terminal stance (45% of the gait cycle) was simulated by applying loads of up to 500 N through the Achilles tendon. While the findings are consistent with motion analysis studies in which approximately 20° of hallux dorsiflexion has been shown to occur before an increase in arch height (windlass mechanism) is evident, the model did not consider the arch-supporting effect of the intrinsic and extrinsic flexor muscles of the foot. The long digital flexors, and in particular the tibialis posterior, have been shown to exert an arch-supporting influence during quasistatic testing (Kitaoka et al. 1997), which is further amplified once the heel is elevated from the support surface (Sharkey et al. 1998). Moreover, the increase in arch height associated with the windlass effect during gait has been reported to coincide with peak intrinsic muscle activity, reduced activity in the gastroc-soleus complex, reduced vertical loading, ankle plantarflexion, peak horizontal propulsive force, and the onset of double limb support. Collectively, these factors would act to minimize the internal loading of the plantar fascia (Wearing et al. 2006). Thus, rather than producing an arch-raising (windlass) effect, the plantar fascia may be alternately viewed as a dynamic coordinator of movement, effectively synchronizing digital dorsiflexion with supination of the foot and external rotation of the leg.

Internal loading of the plantar fascia

The role of the plantar fascia during gait has largely been inferred from static loading of cadaver specimens and few studies have evaluated the internal loading of the plantar fascia during dynamic activities such as walking and running. Scott & Winter (1990) employed a two-dimensional, four-segment model of the lower extremity, in which plantar fascia was assumed to account for 50% of the bending moment at the midtarsal joint during running. Peak fascial loads in the order of 2.8 times bodyweight were predicted during the midstance and early propulsive periods of gait. Giddings et al. (2000) predicted comparable fascial loads (1.8 and 3.7 times bodyweight) during walking and running using a twodimensional, three-segment finite-element model of the foot in which the orientation and movement of osseous segments were obtained from cineradiography. While internal stress was noted to peak late in stance (60-70%), the predicted loads from both studies exceeded the ultimate tensile strength $(1189 \pm 244 \text{ N})$ reported for human bone-plantar-fascia-bone preparations during loadto-failure testing (Kitaoka et al. 1994).

Erdemir et al. (2004) estimated considerably lower peak fascial loads (538 \pm 193 N) during simulated walking, which occurred at 80% stance and were equivalent to 47% of the force applied to the Achilles tendon (1041 ± 213 N), and approximately 40% of the ultimate tensile strength of the plantar fascia (1372 \pm 347 N). The findings suggest that the plantar fascia has a strength-safety factor of approximately 2.5, comparable to the ACL (2.4), but considerably lower than bone (~ 6) and most animal tendons (~ 8), even high-stress tendons in which recovery of elastic strain energy is believed to be important ($\sim 2-4$) (Frost 2001). However, these models have failed to consider the influence of intrinsic foot muscles on the internal loading of the plantar fascia. The intrinsic muscles are anatomically well positioned to reduce fascial loading and, although normally quiescent during static bipedal stance, they have been reported to be active throughout the midstance and propulsive periods of gait.

The plantar fascia has been viewed by some as a purely tensile structure and the organization of collagen within the midsubstance supports such a hypothesis. However, the midsubstance of the plantar fascia does not attach directly to the calcaneus but, rather, a zone of uncalcified and calcified fibrocartilage is interposed between the midsubstance and bone proper. While such attachments are thought to provide a gradual transition from hard to soft tissue that assists with the dissipation of stress, fibrocartilage is typically found at sites within tendons and ligaments that are subjected to bending, shear or compressive forces, or their combination (Benjamin & Ralphs 1998). Given that the calcaneal attachment of the plantar fascia also serves as the origin of the flexor digitorum brevis muscle, the enthesis is likely exposed to bending, shear, and compression forces, which may predispose the attachment to injury (Wearing et al. 2006).

Plantar fasciitis

Plantar fasciitis is the most common disorder of the foot, affecting 10% to 20% of injured athletes (Rome 1997). The overall prevalence, however, is unknown, as plantar fasciitis predominantly occurs in sedentary, middle-aged individuals and may increase with senescence. Similarly, little is known regarding the clinical course of plantar fasciitis. Although 5 to 10% of sufferers progress to surgery, the majority ostensibly resolve within 6 to 18 months of the initiation of conservative therapy, leading some to suggest that plantar fasciitis is a self-limiting condition.

Clinical signs and symptoms in plantar fasciitis

Despite the complex nature of heel pain, the diagnosis of plantar fasciitis is typically based on clinical criteria alone. The most consistently reported patient symptoms include weightbearing pain involving the inferomedial aspect of the heel, which is exacerbated following periods of nonweightbearing and a history of post-static dyskinesia. In contrast to the acute pain that is localized to the midportion of the plantar fascia with partial fascial tears, the pain associated with plantar fasciitis is typically insidious in onset and reduces with ambulation.

Pain localized to the medial tubercle of the calcaneus at the attachment of the plantar fascia is considered pathognomonic. Pain extending along the course of the fascia has been less frequently reported. Palpable pain localized to other sites or structures of the rearfoot is generally considered to reflect other conditions. With the exception of sonographic and magnetic resonance imaging, there are few, if any, reliable clinical tests to aid in the diagnosis of plantar fasciitis. Increased pain with dorsiflexion of the hallux, once considered the sine gua non of the condition, has been shown to have low sensitivity compared to palpation. Reproduction of symptoms with dorsiflexion of the foot has also been widely advocated as a diagnostic indicator of plantar fasciitis. However, as with dorsiflexion of the hallux, the maneuver has been shown to simultaneously strain other closely related foot structures, raising questions as to its validity.

Imaging in plantar fasciitis

Radiographic

Radiographic examination may demonstrate the presence of an inferior calcaneal spur of unknown significance. Although bony spurs are more common in heel pain, spurs are not typically located within the fascia itself but are generally found either deep to its fibrocartilaginous attachment (Kumai & Benjamin 2002) or involving other structures, such as the origin of the flexor digitorum brevis, quadratus plantae, and long plantar ligaments. Given the proposed role of fibrocartilage in redistributing compressive, bending and shear forces, plantar heel spurs have been suggested to develop in response to bending at the fibrocartilage attachment, challenging the widely held assumption that they result from excessive traction within the fascia (Kumai & Benjamin 2002; Wearing et al. 2006).

Sonographic

The typical finding of plantar fasciitis on sagittal sonograms involves diffuse or localized hypoechoic areas within a thickened calcaneal attachment (Fig. 5.9.1). A sagittal thickness in excess of 4 mm at the fascial insertion, in addition to hypoechoic change, is a widely used clinical indicator of plantar fasciitis. The sensitivity and specificity of ultrasound in detecting plantar fasciitis ranges from 74–100% and 71–91%, respectively. Such studies, however, have adopted clinical features, rather than histologic evidence, as the diagnostic standard and are difficult to interpret in light of the relatively low specificity of clinical findings in



Fig. 5.9.1 • Sagittal sonogram of the plantar fascia in individuals with and without plantar fasciitis (A) In pain free individuals, the plantar fascia (PF) is characterized by a moderately echogenic, striated appearance and is approximately 3 mm thick at its calcaneal (C) attachment. From Tsai et al. 2000; Wearing et al. 2007 with permission. (B) Plantar fasciitis is characterized by diffuse or localized hypoechoic areas (*) within a thickened calcaneal attachment. From Gibbon & Long, 1999, with permission. A sagittal thickness in excess of 4 mm has been advocated for the diagnosis of plantar fasciitis. From Tsai et al., 2000, with permission. Note that the normally echogenic borders of the plantar fascia are poorly defined in plantar fasciitis. From Wall et al., 1993, with permission.

heel pain. While similar observations in tendon have been reported to coincide with histologically verified areas of degeneration, increased fascial thickness has also been reported in both Achilles tendinopathy and seronegative arthritis (Gibbon & Long 1999). Moreover, there is evidence that the thickness of the plantar fascial enthesis, at least in healthy individuals, is dependent on demographic characteristics such as bodyweight. In the absence of bodyweight-adjusted data, therefore, the sensitivity and specificity of ultrasound in the diagnosis of plantar fasciitis remains unknown.

Ultrasonography has also been used to assess clinical outcomes in plantar fasciitis. Reductions in plantar fascial thickness of 0.5 to 1 mm have been reported with resolution of heel pain. However, these reductions are barely more than the limits of agreement (± 0.6 mm) for repeated measurements of fascial thickness (Wearing et al. 2004). While sonographic measures of soft tissue thickness assume a constant speed of sound in tissue, the speed of sound is altered by the water and collagen content of soft tissues. Changes in collagen and fluid content are characteristic of plantar fasciitis and the proportion of these constituents is likely to change with symptom resolution. Measures of fascial thickness, therefore, are likely prone to small errors when used to monitor recovery from plantar fasciitis.

Magnetic resonance

The plantar fascia normally appears as a homogeneous flat band of low signal intensity on standard T1- and T2-weighted MR sequences. In contrast to ultrasonography, MR imaging has primarily focused on changes in signal intensity, rather than tissue dimensions, in plantar fasciitis. Increased signal within the fascia is considered characteristic of plantar fasciitis. Increased signal on standard MR imaging of tendon coincides with areas of degenerative change. However, degenerative change alone does not account for the hyperintensity noted on MR images. This may, in part, account for the relatively poor correlation between measures of MR signal intensity and clinical outcome in plantar fasciitis.

While MR imaging may depict the size of degenerative lesions more accurately than ultrasonography, the technique appears to offer minimal benefit in terms of sensitivity and specificity in detecting plantar fasciitis. The greater expense and restricted availability of MR imaging, therefore, leaves ultrasonography as the preferred first-line approach in screening for plantar fasciitis.

Histopathology of plantar fasciitis

The pathogenesis of plantar fasciitis is not well understood and is hampered by a lack of consistent nomenclature and reporting of pathological findings. The main histologic features reported for plantar fasciitis following surgical resection in chronic plantar heel pain are summarized in Table 5.9.1. Varying levels of collagen degeneration with fiber disorientation, elevated mucoid ground substance, and angiofibroblastic hyperplasia are the most common findings. However, active inflammatory infiltrate, as indicated by the presence of polymorphonuclear leukocytes, lymphocytes or macrophages, has been reported infrequently and primarily in association with partial fascial tears in athletic populations. Hence, while various combinations of degenerative change are commonly observed in plantar fasciitis, inflammatory-cell-mediated inflammation does not appear to be a predominant histologic finding, especially in older, sedentary individuals.

Degenerative change in the absence of inflammatory-cell-mediated inflammation has also been reported in chronic tendon disorders. Thus the mechanism underlying the development of plantar fasciitis may be more akin to that of tendinosis (tendon degeneration) rather than tendinitis (inflammation) and represent an advanced degenerative change.

Alternatively the absence of inflammatory infiltrate in chronic forms of plantar fasciitis may represent late sequelae of chronic inflammation. In tendon, degenerative change has been suggested to proceed through a series of acute and chronic inflammatory phases before overt degeneration develops (Hess et al. 1989) and inflammation may play an important role early in the disease process. However, degenerative lesions within the plantar fascia have also been reported, albeit to a lesser extent, in amputated limbs without a history of heel pain. Similarly, rupture of the plantar fascia (the end result of degenerative change), is not always preceded by symptoms of plantar fasciitis. While these observations suggest that degenerative change within the plantar fascia may proceed asymptomatically, they also raise the possibility that inflammation and degeneration do not represent a continuum of disease but reflect two independent processes.

Etiology

The mechanism leading to degenerative change of the plantar fascia is poorly understood. While animal studies show an association between mechanical overload and tendon degeneration (Soslowsky et al. 2000), repeated mechanical loading, in and of itself, is not sufficient to induce degenerative change. Rather, some other external or internal risk factor is required (Carpenter et al. 1998).

Internal risk factors including age, bodyweight (BMI), foot type, heel pad properties, and arch and ankle motion, when coupled with exposure to external risk factors such as unaccustomed exercise, prolonged standing or inappropriate footwear, are thought to predispose an individual to the development of plantar fasciitis when exposed to some unknown inciting event (Rome 1997). Intrinsic factors are widely held to increase strain at the fascial enthesis primarily by lowering the height of the medial longitudinal arch or by increasing direct compressive stress at the enthesis. Empirical evidence for the role of such risk factors in the development of heel pain, however, is controversial at best (Wearing et al. 2006). Studies in unilateral plantar fasciitis suggest that many parameters traditionally considered to be risk factors of plantar fasciitis are, instead, likely to be aggravating factors and have led to the development of a new model of plantar fasciitis (Wearing et al. 2004, 2007, 2009) (Fig. 5.9.2).

The model stems from observations that a thickened plantar fascial enthesis, while characteristic of plantar fasciitis, may also occur in the asymptomatic limb and in the absence of a history of pain

Study	Subjects	Method	Histological findings
Snider et al. (1983)	9 males and 1 amputated control limb	Light microscopy	4 variations noted: Collagen degeneration (100% of cases) Angiofibroblastic hyperplasia (56% of cases) Chondroid metaplasia (22% of cases) Matrix calcification (11% of cases)
Leach et al. (1986)	15 athletes	Not reported	Chronic granulomatous tissue Mucoid degeneration in some instances 2 cases of partial rupture Local inflammatory reaction
LeMelle et al. (1990)	2 subjects	Electron microscopy	Subject 1: Fibrocartilaginous degeneration Fibrovascular hyperplasia Subject 2: Irregular staining Fraying of collagen fibers No fibrosis or lymphocytic infiltration
Schepsis et al. (1991)	25 athletes	Not reported	Degeneration of collagen Collagen metaplasia Calcification
Tountas & Fornasier (1996)	21 sedentary subjects and 5 amputated control limbs	Not reported	Varying amounts of: Collagen degeneration Mucoid degeneration Fibrinoid degeneration Fibrovascular proliferation Partial rupture No active inflammation
Lemont et al. (2003)	50 heel spur samples	Not reported	Normal histological appearance (10 cases) Collagen fragmentation in association with mucoid degeneration (16 cases) Vascular ectasia of adjacent bone marrow (12 cases) Crystalline deposition (2 cases) No evidence of inflammation
Jardé et al. (1999)	38 heel spur samples (30 partial ruptures)	Not reported	Chronic granulomatous tissue (100% of cases) Angiofibroblastic hyperplasia (100% of cases) Chondroid or cartilage metaplasia (4 cases) Fibromatosis (4 cases)

Table 5.9.1 Reported histopathology in chronic plantar fasciitis

(Wearing et al. 2004). Thickening of the fascial enthesis associated with plantar fasciitis may, therefore, represent a bilateral or systemic process, such as that proposed in diabetes (Wearing et al. 2004) and suggests that either (1) the plantar fascia has an inherent mechanical deficiency and is incapable of tolerating normal levels of stress to which it is exposed, or (2) a mechanically sound plantar fascia is exposed to abnormal levels of stress bilaterally and responds by adaptive thickening. When exposed to an extrinsic factor, such as unaccustomed activity, prolonged standing, or inappropriate footwear, the upper strain threshold for modeling of the plantar fascia is exceeded, microdamage accumulates and degenerative change ensues. Although degenerative change may proceed asymptomatically, the source and



Fig. 5.9.2 • Proposed etiologic model in plantar fasciitis. Neuromuscular or intrinsic fascial deficits, when combined with extrinsic factors, induce plantar fascial strains that exceed the upper diametric strain threshold for modeling and result in thickening and degenerative change within the plantar fascia. Although mechanisms underlying the development of clinical symptoms are unknown, arch mechanics, midfoot loading and heel pad mechanics likely act as aggravating, rather than predisposing, factors. The potential roles of age and obesity in plantar fasciitis are currently unknown. Although manipulation of aggravating factors may provide an avenue for symptomatic relief in heel pain, it is unlikely to address the underlying cause of the condition.

mechanism underlying the development of painful symptoms in heel pain are unknown.

Traditionally, pain associated with plantar fasciitis was thought to arise from either inflammation or disruption of collagen fibers. However, inflammatory-cell infiltrate is rarely observed on histopathologic examination of specimens in chronic plantar fasciitis, and collagen disruption, as depicted with medical imaging, is poorly correlated with clinical symptoms. While there is some evidence that pain levels are positively correlated with neovascular ingrowth, as determined by power Doppler ultrasonography, positive color flow and hypoechogenicity are neither specific to nor consistent findings in plantar fasciitis and are often reported in fascia of asymptomatic limbs. Thus, neovascularization is not likely to be the primary cause of pain in plantar fasciitis. Although alternative biochemical hypotheses involving neurotransmitters, such as glutamate and substance P, have been implicated in tendon pain, the significance of these factors in plantar fasciitis remains unknown.

Intrinsic factors, such as the shape of the medial longitudinal arch, midfoot loading and the energy dissipating properties of the heel pad, likely act as aggravating, rather than inciting, factors in heel pain (Wearing et al. 2004, 2007, 2009). While the structure and movement of the medial longitudinal arch was found not to differ between individuals with and without plantar fasciitis during gait, lower arch structures were associated with greater levels of pain and fascial thickening (Wearing et al. 2004, 2007). Similarly, higher loading of the midfoot during walking was associated with greater self-reported levels of heel pain (Wearing et al. 2007), suggesting that tensile and compressional force may both evoke pain. Moreover, the energy dissipating properties of the heel pad were shown to be reduced in plantar fasciitis when compared to pain-free individuals (Wearing et al. 2009). The energy dissipation ratio is a measure of the energy lost by viscous friction within tissue and is believed to play an important role in damping high frequency vibrations. Reduced energy dissipation of the heel pad during walking may increase the vibrational loading experienced at the calcaneal attachment of the plantar fascia. Thus, treatments directed toward maintaining the shape of the arch. lowering midfoot loading and/or improving shock absorbency of the heel may help to reduce the severity of pain associated with plantar fasciitis. However, such treatments are unlikely to address the cause of the condition, which remains unknown. Similarly, the potential roles of age and obesity in the development of plantar fasciitis are vet to be elucidated.

Evidence for a neuromuscular deficit in plantar fasciitis

Both active (muscles) and passive (plantar fascia and ligaments) elements are important in the maintenance of the medial longitudinal arch (Fiolkowski et al. 2003). A neuromuscular deficit leading to altered muscular activity, particularly of the intrinsic foot muscles to which the fascia acts as a partial origin, may result in greater or more complex internal loading of the plantar fascia in individuals with plantar fasciitis. Reduced strength of the ankle and digital plantar flexors has been documented in plantar fasciitis, thus leaving passive structures, such as the plantar fascia to bear a relatively greater proportion of load. Although such findings do not preclude the potential role of reflex inhibition of musculature secondary to heel pain, Chundru et al. (2008) recently demonstrated that plantar fasciitis was significantly more common in individuals with atrophy of the abductor digiti minimi, suggesting muscular changes may precede clinical symptoms. Such a mechanism may also account for the fascial thickening noted in people with diabetic neuropathy, in which intrinsic foot muscle atrophy is common.

Evidence for an inherent fascial deficit

Direct evidence for an intrinsic mechanical deficit leading to a reduced capacity of the plantar fascia to tolerate normal internal loads is scant. However, degenerative thickening of the plantar fascia, while characteristic of plantar fasciitis, has also been reported in asymptomatic limbs and in the absence of a history of pain (Wearing et al. 2004), suggesting plantar fasciitis may represent a bilateral or systemic process. In tendon, such degenerative thickening is hypothesized to proceed asymptomatically and reduce the ultimate tensile strength of tendon (Soslowsky et al. 2000). Moreover, comparable degenerative change has been reported in similar "overuse" injuries of the intervertebral disc, the cruciate ligaments of the knee, and the Achilles and rotator cuff tendons (Järvinen et al. 1997). There is a genetic component, at least in part, to the degenerative changes noted in these structures. For instance, COL5A1 polymorphs, which are thought to be important in regulating fibrillogenesis and modulating fibril growth, have been associated with Achilles tendinopathy and cruciate ligament ruptures in humans, while COL9A2 polymorphs, involved in bridging collagens to noncollagenous proteins, are thought to play a role in degenerative disc disease. Whether these variants, and others, are directly involved in the development of such degenerative changes or in strong linkage disequilibrium with actual diseasecausing loci remains to be established. Similarly, potential gene-gene and gene-environment interactions are poorly understood. While there is evidence that

COL9A3 gene polymorphisms and persistent obesity may act synergistically to increase the risk of degenerative disc disease, potential genetic components that predispose an individual to the development of plantar fasciitis have not been investigated to date.

Summary

Traditionally, plantar fasciitis has been considered to be an overuse injury, in which lower limb biomechanics that promote excessive tensile strain within the plantar fascia are thought to predispose an individual to the development of chronic inflammation. However, scientific support for this premise is limited. Plantar fasciitis is characterized by a marked degeneration of collagen in which inflammatory cell infiltrate is not a predominant feature. Similarly, there is evidence that repeated physiologic loading is not sufficient to induce degenerative change in soft tissues and that factors previously considered to predispose to plantar fasciitis, such as the shape of the medial longitudinal arch, midfoot loading and the energy dissipating properties of the heel pad, are more likely aggravating factors, influencing levels of pain rather than initiating the condition. It is proposed that either an inherent fascial or neuromuscular deficit renders the plantar fascia incapable of tolerating normal levels of stress or exposes the fascia to abnormal levels of stress. When combined with an extrinsic factor, such as unaccustomed activity, the upper strain threshold for modeling of the fascia is exceeded and degenerative change ensues. Mechanisms underlying the development of pain in plantar fasciitis are unknown and further research is needed to determine the role of bending, shear and compression forces, along with potential neuromuscular and genetic components, in the disease process.

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Diagnostic procedures for fascial elasticity

An introduction

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This is the shortest section of this text, not because it is less important but because the field is the least developed. Given the paucity of evidence on palpation, the first chapter presents a method of assessment which could form the basis for a standardized and then testable palpatory technique for fascial disorders. Spinal palpatory findings differ between examiners, showing low to moderate reliability in the review by Seffinger and colleagues (2004); pain provocation tests were more reliable than soft tissue palpation, which is of course precisely what manual therapists consider to be most important. Inclusion of the clinical context rather than blind assessment of the patient improved reliability in upper extremity examinations (Hickey et al. 2007; Smith et al. 2010). Both Cibere et al. (2008) and Brunse et al. (2010) showed that test standardization improved those examination signs with poor rater agreement. Newer examination tools such as musculoskeletal provide such an opportunity, but Kim et al. (2007) found that they were able to identify structural problems but the results were unfortunately independent of the physical exam or the pain symptoms. Once a standardized assessment is developed, it can be improved into a psychometrically more sound test (Hunt et al. 2009). As Seffinger (Seffinger et al. 2004) concludes:

Given that spinal palpatory procedures are a cornerstone of diagnostic and therapeutic interventions across disciplines, for patients with low back and neck pain, professional societies and organizations need to enact continuing medical education programs and establish research procedures to address the reliability of spinal palpatory procedures. Chapter 6.2 addresses another vexing problem, hypermobility (HM). While there are multiple techniques for the management of people who have decreased range of motion, there are far fewer options for those with increased range. The British Medical *Journal* has begun a series of articles for physicians on conditions often missed, and the recent one by Ross & Grahame (2011) is highly recommended for your interactions with traditional medical practitioners. It lists a number of clues (besides joint mobility) that suggest the joint hypermobility syndrome, which in adults may include symptoms of pain, arthritis, autonomic dysfunction, gastrointestinal disorders. A somewhat different set of symptoms are found in children and adolescents, including late walking, ankle sprains, decreased coordination, and widespread pain.

There is not enough research on psychological aspects of fascial disorders to devote a whole chapter to this, but there are some intriguing findings regarding hypermobility. A strong association between hypermobility and anxiety and panic disorder was noted in a case control study by Martin-Santos and colleagues (1998), with 68% of persons with a diagnosis of anxiety showing hypermobility, compared to 10% of persons with medical or psychiatric diagnoses. That group has continued to find this association in the general population, with increased scores measuring trait anxiety in persons with hypermobility (Bulbena et al. 2004) in a general medical clinic, and increased scores measuring fear in a general population sample with hypermobility, compared to those without (Bulbena et al. 2006). Recently they have reported a 15-year follow-up of a general population recruited at ages 16 to 20 (Bulbena et al. 2011); in this population 20% have hypermobility, similar to the population rate in many other places. In the 15 years since entry into their study, an astounding 41% of the persons with hypermobility have developed panic disorder, compared to only 2% of the persons with normal mobility. There was also a significant increase of social phobia ($6\times$), simple phobia ($3\times$), and anxiolytic drug use ($4\times$) in persons with hypermobility.

These findings have been replicated by two other groups: Ercolani et al. (2008) found increased anxiety and somatic symptoms in a case control study, compared to normal persons and another group of persons with fibromyalgia. Garcia-Campayo et al. (2010) found a similar 20-fold increase in panic disorders in persons with hypermobility (61% compared to only 10% in the general population) and noted a direct correlation (r = 0.52) between the Beighton hypermobility score and the Panic and Agoraphobia Scale. For a recent review of the association between anxiety and hypermobility, see Garcia-Campayo et al. (2011).

The parallel between increased anxiety and panic disorder in HM and in persons with gastrointestinal (GI) disorders was noted by Zarate et al. (2010), who looked more closely at the correspondence between HM and GI disturbances. They found that 50% of new patients in a GI clinic had hypermobility, and hypothesize a change in the connective tissue and passive mechanical properties of the gut wall or concurrent changes in the autonomic nervous system as possible etiologies. The same group (Mohammed et al. 2010) looked at persons with rectal evacuation dysfunction, and found 32% reported hypermobility symptoms on a simple five question screening test, compared to 14% of controls. Vounotrypidis et al. (2009) looked at specific types of GI disorders and found joint hypermobility in 70% of clinic patients with Crohn's disease, 35% with ulcerative colitis, compared to 25% of controls.

In all of these studies, patients are given a hypermobility score based on specific numbers of joints involved, without specifying a specific pattern of increased mobility. In my clinical practice I have noticed patients who have distal hypermobility particularly in the arms, some with excessive but some with reduced straight leg raise. Voermans et al. (2009) nicely categorize the pattern of joint hypermobility found in persons with myopathies, and their findings are instructive as we continue to explore the issue of hypermobility in general. While they list findings by muscle disease, their joint findings can be categorized by the effect on joints in a fashion of more use to studies of fascia: I. Conditions with diffuse distal and proximal hypermobility. The elbow joint may start hypermobile but develop contractures – Marfans, Ehlers-Danlos classical type, tenascin-X and hypermobility type. In central core disease the ankle may develop contractures. IA. Diffuse distal and less so proximal mobility – multiminicore disease. II. Distal hypermobility only - Ehlers-Danlos vascular and kyphoscoliotic types. III. Distal hypermobility and proximal contractures – Ulrich congenital myopathy, Bethlem myopathy, congenital muscular dystrophy with joint hypermobility (CMDH). The ankle also develops contractures. IV. Mixed pattern – limb girdle muscular dystrophy shows hypermobility in wrist, PIP and MCP joints, but contractures in DIP, elbow and knee.

In many of these conditions the elbow develops contractures. This may reflect a tendency for trauma in this joint. In my residency training 30 years ago I was taught that physical therapy could aggressively stretch every joint in the body except the elbow, where aggressive stretching usually led to decreasing, not increased, range of motion. Or perhaps it relates to the muscle–ligament complex at the elbow so elegantly described by Van der Wal (2009): as the joint moves, the ligaments must change length to accommodate the changing distance between the bones, and this change is modulated by muscular connections into the ligament. So attempting to stretch the ligaments without addressing the muscular component is counterproductive.

The diagnosis of hypermobility is primarily done through well established clinical tests which have demonstrated good reproducibility (kappa scores of 0.75 to 0.85)(Remvig et al. 2007; Juul-Kristensen et al. 2007). Recently, clinical tests of skin elasticity have been developed, which show promise for quantifying hypermobility (Delalleau et al. 2008; Remvig et al. 2009; 2010; Farmer et al. 2010). Physiological changes have been found in motor control, posture, reflexes (Ferrell et al. 2007), force development in muscle (Mebes et al. 2008), proprioception (Fatoye et al. 2009), and these have been recently reviewed in the context of creating a physical rehabilitation program (Keer & Simmonds 2011). Electromyographic investigation of persons with suspected carpal tunnel syndrome found that hypermobility was more common, the more severe the carpal tunnel findings (r = 0.6) (Aktas et al. 2008). Eighty-five per cent of those with symptoms who had positive test findings also had hypermobility, while only 20% of those with symptoms but negative electrodiagnostic tests had hypermobility. These physiological findings will lead to more targeted research and treatments for persons with hypermobility.

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Fascial palpation

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Touch can be considered to be one of the special senses, along with sight, hearing and taste. Neuroanatomists commonly use the terms "somatic sensation" or "somatosensory" to describe this sensory or "afferent" function. Palpation implies touching with some form of therapeutic inquiry, if not necessarily overt diagnostic intent. Because touching another person is a deeply individual experience, the intent of the practitioner becomes a significant factor in the outcome (the gathering of information). Thus information can be gathered actively ("going and getting it"). At the same time, information can also be gathered by using a more passive approach, i.e., allowing the information to come into the practitioner's somatosensory system. In either case, a thorough and accurate visual picture of the underlying anatomy will always be a major asset to the therapist.

In recent years, manual therapeutics has recognized the fascial system as a prime target and has turned its attention there. In fact, several major schools of manual therapy, such as Structural Integration, or Rolfing[®], have built their treatment philosophy and rationale around the fascial system.

The fascial system has been described as an 'endless web,' that is, far more organized than was previously imagined (Schultz & Feitis 1996). Fascia surrounds the entire body just under the skin as the superficial fascia, and enwraps all the other tissues and organs of the body as the deep fascia. It is, as observed by Ida P. Rolf, PhD, the "organ of form" (Rolf 1977). But in addition to its anatomical and physical properties, the fascial system is increasingly recognized for its physiologic properties (Langevin et al. 2004, 2006).

Active versus passive assessment

Palpating a moving structure can produce a great amount of clinical information. This can be done actively where the client performs an action, or passively, where the practitioner moves the structure for the client. Motion quality, range, and end-feel can all be assessed using this method. In addition, motion against resistance can inform the practitioner as to the relative strength and/or sensitivity of a movement. Some of these approaches are to be found in the exercises later in the chapter.

6.2

When are you palpating?

Any time a therapist makes manual contact, a palpatory experience occurs, and palpation is routinely used to assess the condition of the client prior to any therapeutic intervention (whether or not a diagnosis is made). Palpation may be employed during a therapeutic intervention, and may be used to assess the results of such intervention(s).

Palpation tools

While the hand is probably the primary palpatory tool, manual therapists will occasionally use more than just the palms of the hands and fingers for this purpose, with knuckles, fists, forearms, elbows, and sometimes even involving tools (See Chapter 7.14) being employed.

The amount of pressure applied by the therapist will vary, depending on the depth of the structure that is being assessed. The direction of the applied pressure may vary, depending on whether the practitioner is performing a 'direct' or 'indirect' technique, i.e., pushing toward or away from a motion barrier, respectively. To an extent, such choices will depend on the relative acuteness or chronicity of the involved tissues. Additionally, the direction of palpation may be across a muscle, tendon, or ligament, depending on the information being gathered. Usually, the speed that a practitioner's contact moves through the tissues will be dependent on the rate of "release" that is noted, with slower movements indicated in more tender areas, or those areas that are palpably different; for example, more dense, in texture.

What is being palpated?

The different forms of manual therapy will have varying anatomical targets. However, regardless of the target, the ability to accurately visualize the structures beneath the hands is of utmost importance, whether the practitioner's hands are moving broadly or deeply. Such knowledge allows the practitioner to predict where the hands are headed and what they can expect to encounter. It will also contribute to the development of a treatment strategy, i.e., where to go next, or in some cases, where not to go next.

Two examples come to mind.

- First, the organization of the thoracolumbar fascia, where the superficial posterior layer has a significantly different directionality than the deeper layer. In turn, the middle layer has a significantly different thickness and organization than the first two. Obviously, this speaks to their different functional roles, and knowledge of the anatomy can influence the treatment plan.
- The second example involves the muscles of the posterior neck. This extraordinarily complex group has the all-important function of aligning the head such that the eyes are parallel to the horizon. At least a dozen muscles or muscle groups are present on each side of the midline. As the hands and fingers palpate from superficial to deep, different fiber directions will be encountered as well as different levels of muscle tone.

The need for a relaxed therapist

To maximize the information gathered from palpation, it is helpful to minimize the amount of noise in the system. As the therapist is bombarded with sensory information in their own body, coming not only from the skin, but also from the joints, muscles, and fascia, a conscious effort to reduce these signals is fundamental to palpation. In other words, the therapist should make an effort to improve the signal-tonoise ratio in his/her own system. This is achieved by ensuring that the body is well supported, from the ground up, that posture is correct, and that the upper limbs are as relaxed as possible. This is especially true of the muscles of the shoulder girdle, neck, temporomandibular joint, and face. In addition, during palpation, the breath should be slow, deep, and as unrestricted as possible.

Layers (see Figure 6.2.1)

Palpation by definition begins with skin-to-skin contact. Here, observations can be made regarding the skin: is it smooth, rough, oily, dry, sweaty, are



Fig. 6.2.1 • Typical fascial layering, seen here as in the thigh. Other areas contain more or fewer layers between skin and bone. From Chaitow, 2010, with permission.

its characteristics limited to a specific area, etc.? Palpation of the skin can be performed by simply moving the hands lightly along the surface without actually moving the skin.

The layer just deep to the skin is the superficial fascia. This layer is of varying depth around the body, and can be encountered by applying just enough pressure, such that the skin will move with the hands of the therapist. It is considered a loose, areolar type of connective tissue.

The superficial fascia is specialized in various areas to perform specific functions, such as cushioning the soles of the feet, and supporting the lower abdominal wall (Scarpa's fascia). Occasionally, the superficial fascia virtually disappears, leaving the deep fascia very close to the surface, e.g., the dimples at the posterior superior iliac spines.

Just deep to the superficial fascia is the deep fascia, which forms the "endless web" described by Schultz and Feitis (1996). It surrounds and supports muscles, nerves, blood vessels, and organs.

From a developmental point of view, the fascia is derived from the embryonic mesoderm and forms the template in which all other mesenchymal derivatives arise. For example, bones and muscles develop within sheaths of mesodermal tissue, the periosteum and epimysium, respectively. As a result, very rarely do muscles attach directly to bone. Usually there is some form of intervening connective tissue. In fact, each individual muscle cell is encased in the connective tissue endomysium, while groups of myofibers, the muscle fascicles, are wrapped in perimysium. Joint capsules, tendons, and ligaments are all specializations of mesodermal connective tissue and are considered fascial elements by many practitioners.

Accessing the deepest layers of fascia presents a challenge to the therapist. To address these deep layers, it is paradoxically best not only to go slower, but also to lighten the pressure applied. There seems to be an inverse relationship between pressure and depth.

Moving through the deep fascia is possible without causing discomfort to the client/patient. A phenomenon exists where the tissue seems to "melt" under the hands of the therapist. Moving slowly into the movement endpoint of the tissue usually will result in a release, and further movement potential.

This releasing phenomenon may relate to a property of the extracellular matrix known as thixotropy. However, at this time, the reported palpation experiences of manual therapists remain unexplained.

Communicating with the client

A problem with palpation is that although receptor and neuronal activity can be objectively measured, its interpretation is, by definition, subjective.

Such interpretation is expressed in language that can vary considerably. In any therapeutic relationship, effective communication is a key element. The therapist and client are in a partnership that will only function maximally if free lines of communication and trust exist. This entails making sure the client is in control of the situation, where he/she is able to understand the language of the therapist and that the client is aware of what's going to happen next.

It is important to remember that many of the mechanoreceptors in the fascia also double as pain receptors (nociceptors). As such, there will be an intensity threshold for every individual where pressure becomes painful. Since each person's experience of the phenomenon is different, it is important to monitor that experience periodically by asking the client about their experience, especially when addressing areas of potential tenderness (e.g., close to bones).

Palpating for information

It is axiomatic that practitioners who use their hands to manipulate soft or bony structures should be able, accurately and relatively swiftly, to feel, assess, and judge the state of a wide range of physiologic and pathologic conditions and parameters, relating not only to the tissues with which they are in touch but others associated with these, perhaps lying at greater depth.

The information a practitioner needs to gather will vary according to the therapeutic approach, possibly including:

- the range of motion
- the feel of joint play
- the relative weakness or tightness in muscles
- the amount of induration, edema or fibrosis in soft tissues
- the feel, densitiy and mobility of palpable fascial structures
- identification of regions in which reflex activity is operating
- possible differences in the quality of perceived tissue vitality (flaccid, toned, etc)
- variations in regions of the body
- and many other pieces of acquired information.

In other words, the individual practitioner needs to fit the acquired information into his or her own belief system, to use in accordance with whatever therapeutic methods are seen to be appropriate. The aim is therefore to help to identify what is under our hands when they are in contact with the patient, and, in the context of this book, what information we can gather regarding fascial structures and behavior in particular.

Palpation objectives

Philip Greenman (1989), in his book *Principles of Manual Medicine*, summarizes the five objectives of palpation. He suggests that the practitioner/ therapist should be able to:

- 1. detect abnormal tissue texture
- **2.** evaluate symmetry in the position of structures, both physically and visually
- **3.** detect and assess variations in range and quality of movement during the range, as well as the quality of the end of the range of any movement
- **4.** sense the position in space of yourself and the person being palpated
- **5.** detect and evaluate change in the palpated findings, whether these are improving or worsening as time passes.

These elements, described by Greenman, with the qualification that fascia is the focus in this chapter, are our major objectives in obtaining palpatory literacy in fascial palpation.

Palpate by "feeling", not thinking

Palpation allows us to interpret tissue function. Different histological make-up brings differing amounts of inherent pliability and elasticity; because of this, a muscle feels completely different from a ligament, a bone, and an organ, for example. Thus there is a 'normal' feel when they are healthy, that is different for each tissue. This has to be learned through repeated exploration of "normal" tissue and the practitioner builds a vocabulary of what "normal" feels like. Once someone is trained to use palpation efficiently, then finer and finer differences between tissues can be noted. This is vital, in order to be able to differentiate when something has changed from being "normal".

Kappler (1997) has explained as follows:

The art of palpation requires discipline, time, patience and practice. To be most effective and productive, palpatory findings must be correlated with a knowledge of functional anatomy, physiology and pathophysiology. It is much easier to identify frank pathological states, a tumor for example, than to describe signs, symptoms, and palpatory findings that lead to or identify pathological mechanisms. Palpation with fingers and hands provides

sensory information that the brain interprets as: temperature, texture, surface humidity, elasticity, turgor, tissue tension, thickness, shape, irritability, motion. To accomplish this task, it is necessary to teach the fingers to feel, think, see, and know. One feels through the palpating fingers on the patient; one sees the structures under the palpating fingers through a visual image based on knowledge of anatomy; one thinks what is normal and abnormal, and one knows with confidence acquired with practice that what is felt is real and accurate.

Kappler (1997)

These words define succinctly and with feeling the tool we use and the task we perform when we palpate.

Different parts of the human hand are more or less able to discriminate variations in tissue features, such as relative tension, texture, degree of moisture, temperature, and so on. This highlights the fact that overall palpatory sensitivity depends on a combination of different perceptive (and proprioceptive) qualities and abilities.

These include the ability to:

- register temperature variations and the subtle differences that exist in a spectrum of tissue states, ranging from very soft to extremely hard
- register the existence and size of extremely small entities, such as are found in fibrotic tissue or areas of myofascial trigger point activity
- sensitively distinguish between many textures and ranges in tone, from flaccid to spastic, and all the variables in between.

Physiology of touch

Palpatory perception also results, in large measure, from variations in the number and type (see summary in Box 6.2.1) of sensory neural receptors found in the skin and tissues of various anatomical regions, since these greatly influence the discriminatory capabilities of those regions.

• Light touch is generally accepted as being achieved via mechanoreceptors (such as Meissner's corpuscle and Merkel's disc, as well as hair-root

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Box 6.2.1

Receptors and perception (from Chaitow 2010)

Mechanoreceptors	
Light touch	Meissner's corpuscle
	Merkel's disc
	Hair-root plexus
Deep pressure	Pacinian corpuscle
Crude touch	Thought to be Krause's end-bulb
	Thought to be Ruffini's ending
Proprioception	C C
Muscle length,	Muscle spindle
tendon and limb	Golgi tendon organ
position	Joint/kinesthetic receptors
Nociceptors	
Pain	Free nerve endings
Thermoreceptors	
Warmth	Thought to be free nerve endings
Cold	Thought to be free nerve endings
Internal temperature	Hypothalamic thermostat

plexi) lying in the skin, muscles, joints, and organs. They respond to mechanical deformation resulting from pressure, stretch, or hair movement. It is in the skin that the greatest number of these receptors is found.

- Cruder touch perception is thought to relate to Krause's end-bulb, Ruffini's ending, and Pacinian corpuscles.
- Sensations of heat and cold are detected by thermoreceptors that are considered to be the free nerve endings in the skin.
- If cold is intense, detection is by nociceptors specialized pain detectors – that are also free nerve endings.

Filtering information

There is an apparent contradiction in that while finetouch receptor adaptation may reduce sensitivity, at times too much information is being received, and a degree of discrimination or filtering of information is required in order to make sense of it. Kappler (1997) summarizes this as follows:

A more significant component [of palpation skills] is to be able to focus on the mass of information being

perceived, paying close attention to those qualities associated with tissue texture abnormality, and bypassing many of the other palpatory clues not relevant at the time. This is a process of developing mental filters . . . The brain cannot process everything at once. By concentrating only on the portion you want, it becomes easy and fast to detect areas of significant tissue texture abnormality.

Kappler (1997)

Kappler et al. (1971) tested this concept and found that when they compared student examiners with experienced practitioner examiners, although the students recorded more palpation findings, the practitioners recorded more significant findings.

The experienced practitioners were filtering out the unimportant and focusing on what was meaningful, rather than being "overwhelmed with the mass of palpatory data".

An osteopathic palpation perspective

Gibbons and Tehan (2001) explain the basis of osteopathic ARTT palpation when assessing for somatic dysfunction (their particular focus is on spinal and joint dysfunction):

- A relates to asymmetry. DiGiovanna (1991) links the criterion of asymmetry to a positional focus, stating that the "position of the vertebra or other bone is asymmetrical". Greenman (1996) broadens the concept of asymmetry by including functional, in addition to structural, asymmetry.
- *R relates to range of motion*. Alteration in range of motion can apply to a single joint, several joints, or a region of the musculoskeletal system. The abnormality may be either restricted or increased mobility and includes assessment of quality of movement and "end feel".
- *T relates to tissue texture changes.* The identification of tissue texture change is important in the diagnosis of somatic dysfunction. Palpable changes may be noted in superficial, intermediate, and deep tissues. It is important for clinicians to distinguish normal from abnormal.
- *T relates to tissue tenderness.* Undue tissue tenderness may be evident. Pain provocation and reproduction of familiar symptoms are often used to localize somatic dysfunction.

Practical palpation

A series of fascial palpation exercises are described below. You are advised, after making contact with the tissues being investigated, to increase the pressure on the palpating fingers just sufficiently to make a contact with the tissues deep in the skin – meeting and matching the tissue tension, not invading it.

As the fingers lightly insinuate themselves through the tissues, various changes may be noted: mobility, tenderness, edema, deep muscle tension, fibrosis, and interosseous changes. All but fibrosis can be perceived in both acute and chronic lesions.

- Detection is a matter of being aware of the possible findings and practicing the techniques required to expose these possibilities.
- Amplification requires localized concentration on a specific task and the ability to block out extraneous information.
- Interpretation is the ability to relate the information received via detection and amplification.

Questions the palpating contacts should be asking involve the following descriptors. Is what I am feeling:

- superficial/deep
- compressible/rigid
- warm/cold
- moist or damp/dry
- painful/pain free
- local or circumscribed/diffuse or widespread
- relaxed/tense
- hypertonic/hypotonic
- normal/abnormal
- and where fascia is concerned, most importantly: do the tissues glide, or are they restricted?

Palpation exercises (Myers 2010)

A series of fascial palpation explorations (exercises) follow – based on the work of Tom Myers.

These describe palpation of the front, back, and sides of the lower leg, involving the lower aspects of what Myers has described (2008) as the Superficial Front Line, Superficial Back Line, Lateral Line, and the Deep Front Line.

For palpation exercises that explore these particular fascial pathways more comprehensively (space constraints allow only partial exposure in this chapter), readers should consult Myers' main text (Myers 2008), or Myers' chapter on fascial palpation in Chaitow (2010).

Palpation of the Superficial Front Line (Fig. 6.2.2)

Foot and lower leg:

- With the person supine, use one hand to hold the toes down into flexion while these are lifted up against your pressure.
- The other hand can explore the tendons of both the short and long toe extensors, which jump up through the skin as soon as the muscles are contracted.
- The short extensors angle off towards the lateral aspect of the foot; the long toe extensors pass under our first port of call, the extensor retinaculae.
- The most prominent tendon on the medial side is that of the tibialis anterior. In anatomy atlases, the extensor retinaculae are distinct structures, looking like gauze bandages over the tendons. In reality, those sharp distinctions are made by the dissector's scalpel, while to the therapist's palpating hand they are widely variable in both their thickness and their extent up the shin and down the foot.
- Keeping the muscles tight by having the individual continue to extend the toes against your resistance, pass your lightly touching fingers up those tendons



Fig. 6.2.2 • The lower aspects of the Superficial Front Line. From Chaitow, 2010, with permission.
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where they cross the ankle to feel the retinaculae between these tendons and the skin.

- Depending on individual characteristics, it may be almost impossible to feel these as distinct structures or you may be able to feel the upper and lower sections as distinctly as they are often portrayed in textbooks.
- In any case, note that the retinaculae are not really separate structures in themselves but thickenings of the crural fascia that surrounds the whole lower leg like a support stocking.
- Move up onto the front of the shin and move the skin over the flat surface of the tibia. How much does it move over the bone?
- This can vary from person to person. Can you feel the distinct layer of deep investing fascia (crural fascia) between the easily moving skin and the immoveable bone?
- This investing layer can be "opened" or "moved" on the bone and, in our experience, usually wants lifting cranially for best results.

Palpation of the Superficial Back Line (Fig. 6.6.3)

Foot and lower leg:

The Front Line is complemented by a line running up the back of the body.

• The first and familiar fascial feature within this line is the plantar aponeurosis, readily palpable when it is tightened by extending the toes.



Fig. 6.2.3 • Lower aspects of the Superficial Back Line. From Chaitow, 2010, with permission.

- As wide as all five toes at the ball of the foot, its edges are readily palpable (and sometimes tender), passing back along the sole and narrowing to a width of only 2 cm as it blends into the periosteum at the front of the heel.
- A branch of this fascia, the lateral band, can be felt between the outer lower edge of the calcaneus and the prominent base of the fifth metatarsal bone.
- This fascia is a major stabilizer of the lateral arch and is recommended for treatment in both pronated and supinated feet. The plantar fascia blends into a "bridle" of fascia around the heel that continues into the Achilles tendon.
- Follow this tendon up the calf to feel it thin as it spreads wide over the posterior surface of the soleus.
- At the popliteal fossa of the knee, there is a fascial connection between the heads of the gastrocnemius and the hamstring tendons linking around them.

Palpation of the Lateral Line (Fig 6.2.4)

Foot and lower leg:

Turning to the fascial continuity running up the side of the body, we can palpate the tendons of fibularis longus and brevis (peroneals) just inferior to the fibular malleolus. The fibularii run up the side of the lower leg to the lateral side of the fibula.

• If you have the individual point the toes (strongly plantarflexed), these muscles will be readily palpable between the bulge of the soleus and the



Fig. 6.2.4 • Lower aspects of the Lateral Line. From Chaitow, 2010, with permission.

tibialis anterior. On either side of these muscles are intermuscular septa that divide this lateral compartment from its neighbors.

These compartment walls are often shortened and tight in compartment syndrome and are well connected to the crural fascia that surrounds the lower leg. Relief of compartment syndrome symptoms will often result from deep release work on the crural fascia over the fibularii, as well as work into these intercompartmental septa.

- The anterior septum, between the tibialis anterior and the peroneals, can be found by palpating up from the fibular malleolus, tracking the small 'valley' between these muscles, ending just in front of the fibular head.
- The posterior septum can be tracked by starting in the space between the Achilles tendon and the back of the ankle, running in front of the soleus and ending just behind the fibular head.
- In the ideal, these 'valleys' should be easily accessed down to the fibula; in many cases, however, they are so bound that differential movement or any valley at all is difficult to feel. This is an indication for spreading work to open the valleys.

Palpation of the Deep Front Line (Fig. 6.2.5)

Lower leg:

One can find the distal end of the three tendons that comprise the lower end of this line on the medial side of the ankle.

- When the big toe is actively or passively extended, the flexor hallucis longus tendon can be palpated on the bottom of the foot running parallel to the inner edge of the plantar aponeurosis.
- At the ankle, this tendon can be felt by putting a thumb or fingertip in the medial space just in front of the Achilles tendon and behind the medial malleolus.
- Be careful of the tibial nerve, but you can feel, when the individual flexes and extends the big toe, the tendon moving on the back of the bone where it supports the talus in the medial arch.
- The strong tendon of the tibialis posterior can be felt by putting your fingertip just below the tip of the person's medial malleolus, and then having him/her point the toes and invert the foot. The tendon will push your finger substantially.



Fig. 6.2.5 • Lower aspects of the Deep Front Line. From Chaitow, 2010, with permission.

- The tendon continues under the foot but is too deep to be palpable there.
- The flexor digitorum longus muscle tendon lies about 1 cm posterior to the tibialis posterior but is small and sometimes difficult to find.

The three muscles are palpable only a few centimeters above the ankle before they disappear beneath the bulky soleus. The deep transverse septum, however, that runs behind them, separating these deep muscles of the Deep Front Line from the triceps surae of the Superficial Back Line, can be palpated – at least its outer edges.

- Place the individual supine with one knee flexed and the foot flat on the surface.
- Sitting by the foot, put your thumbs on the front of the shin, about midway up, and insinuate the fingers of your inside hand tightly behind the tibia.
- Do the same with the outer hand, going around the fibularii (peroneal) muscles to come through behind them to the posterior edge of the fibula.
- Once your hands are 'pinching' the deep transverse septum in this way, have the person lift and lower the heel and then the forefoot to feel the muscles moving past this septum.
- This approach is a better assessment tool than trying to read these muscles by palpating directly through the bulk of the soleus and gastrocnemius.

Though not strictly part of this line, the pes anserinus on the inside of the knee presents itself here for palpation; the three tendons of the sartorius, gracilis and semimembranosus can be palpated near their inferior

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end on the medial side of the femoral condyle just above the tibia.

Conclusion

The palpation exercises in this chapter are intended to offer sample experiences of the rich and varied potential that awaits palpatory exploration of the fascial network.

It is important to caution that palpation assessments are not considered highly reliable means of acquiring information. The variability of palpation methodology, and the skills employed, alongside the subjective nature of interpretation, combine to raise important questions about such assessment methods. And even if an assessment method is shown to be potentially reliable, the degree of accuracy via interpretation remains questionable. Until the development of quantitative methods such as ultrasound (see Chapter 8.2), and MRI and elastography (see Chapter 8.3), even for research purposes there were no alternative methods of fascial assessment. And these tools remain largely out of the reach of the clinician, leaving them with palpation as their sole tool. The clinician must constantly consider: What are you feeling? What is its status? What does it mean in diagnostic terms? How accurate are my findings?. Seffinger (2010) has offered a summary of these considerations as follows:

Precision is the measure of the variability in a [palpation] test, and is often used synonymously with reliability. A palpatory test is precise if it repeatedly measures the same thing with little variation. If a palpation test is precise and accurate, then it is both reliable and has validity.

Seffinger et al. (2004) identify the main element of reliability in palpation when they state that this is determined by comparing the reproducibility and concordance of diagnostic findings from the same examiner, and from different examiners palpating the same subject, or group of subjects. And they clearly find that the careful specification inherent in highquality research is necessary to achieve reliability; those studies which are less well designed find much less agreement among examiners.

It would therefore seem clinically prudent to use palpation methods as *a part* of a general evaluation, rather than relying completely on one means of assessment (i.e., palpation) alone, when formulating a treatment plan.

This, then, is the task of the clinician who wishes to be able to rely on palpation to complement other findings, signs and tests – to develop palpatory literacy.

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Hypermobility and the hypermobility syndrome

Assessment and management

Jane Simmonds

Introduction

The first clinical description of hypermobility has been attributed to Hippocrates in the 4th Century BCE, who described the Scythians, a central European tribe, as having such flabbiness and atony, that they lost battles due to their inability to draw their bows and arrows effectively because of their unstable shoulders and elbows (Beighton et al. 1999).

While hypermobility did not feature again in medical writings until the 19th century, Matthias Grunewald (1460–1528) observed hypermobility in "Saint Cyriaque" in the Heller Retable (Fig. 6.3.1), and later, Peter Paul Rubens observed hyperextension of the metacarpal joints, flat-footedness and hyper-lordosis in "The Three Graces" (1638–1640; Prado, Madrid). Furthermore, the musical successes of Paganini were attributed to his extreme hand mobility, in the 18th century (Larsson et al. 1993).

Joint hypermobility is a unifying feature of the heritable disorders of connective tissue (HDCT), (Beighton et al. 1999) a group of genetic disorders affecting connective tissue matrix proteins identified in the 19th century. These include the Ehlers–Danlos syndrome (EDS), Marfan syndrome (MFS) and osteogenesis imperfecta (OI). In recent years a more common, less serious connective tissue disorder with a mixed phenotype has been proposed, termed the joint hypermobility syndrome (JHS) and considered to be an atypical or forme fruste of HDCT, and sharing overlapping features with EDS, MFS and OI (Fig. 6.3.2). Some authorities consider JHS to be synonymous with EDS type III (Grahame 2003).

The HDCTs share many common features of chronic musculoskeletal pain, soft tissue and visceral injury, cardiovascular pathology and dysfunction, skin abnormalities, fatigue and neurogenic dysfunction (Bird 2007; Grahame 2009). Genetic and histologic investigations do not differentiate the conditions and therefore the diagnosis is made on clinical grounds, such as echocardiography to delineate cardiovascular involvement, and slit-lamp examination to establish ocular involvement (Hakim & Grahame 2003a). Although the cardinal features of EDS, NFS and IO include hyperextensible skin, marfanoid body shape, and brittle bones, respectively, these features are not pathognomic to each condition. Hakim & Grahame (2003a) stress that hypermobility of joints is also common to all, with subtle differences and similarities between the HDCTs.

Pathogenesis

JHS is a genetically inherited disorder, presenting with an autosomal dominant pattern, thought to affect the encoding of the connective tissue protein collagen (Grahame 2003). Individuals with JHS display an abnormal ratio of type III to type I collagen (Child 1986). Type I collagen has a high tensile strength and is the most common collagen in the body, abundant in tendon, joint capsule, skin, demineralized bone, and nerve receptors. Type II collagen is found in cartilage, and designed to withstand compressive stress, whereas type III collagen is much more extensible and disorganized, occurring in organs such as the gut, skin, and blood vessels (Beighton et al.



Fig. 6.3.1 • Matthias Grunewald (1460–1528) observed hypermobility in "Saint Cyriaque". From Matthias Grünewald in Wikipedia. Available from http://en.wikipedia.org/wiki/ Matthias_Gr%C3%BCnewald (accessed 16 August 2011).

1999), which may explain the inherent laxity or 'reduced tissue stiffness' (Russek 1999). Mutations in genes encoding collagen type V have also recently been implicated (Malfait et al. 2005), with type V





collagen under normal control interacting with type I collagen during fibrillogenesis and having a role in regulation of fibril diameter. A mutation in tenascin-X, a noncollagenous molecule, has also been suggested as contributing to joint hypermobility. An alteration in this process may potentially lead to thinner, fine, and more disorganized collagen fibers. Skin fibroblast biopsy analysis has allowed researchers to further investigate the microscopic structural discrepancies that may define HCTDs. Malfait and co-workers (2005) hypothesize that interference with the processing of the N-propeptide of either α -chain (α 1 or α 2) of type I collagen is responsible for EDS-like symptoms of skin laxity, joint subluxation, and dislocation.

Testing for hypermobility and hypermobility syndrome

The Beighton nine point scale scoring system (see Box 6.3.1, Beighton et al. 1973) is an adaptation of the method first described by Carter & Wilkinson (1964) and validated for adult populations by Bird et al. (1979). The Contempassis tool is a more sensitive tool for measuring generalized joint hypermobility, but the Beighton scale is usually favored by clinicians because it is easy and quick to apply (Bird 2007).

Both the Carter–Wilkinson and Beighton scales were developed for epidemiologic purposes and although widely used as a clinical assessment test for hypermobility are heavily biased towards the

Box 6.3.1

Nine Point Beighton Scale (Beighton et al. 1973) Nine Point Beighton hypermobility score

Individuals are scored on	Right	Left
their ability to		
Passively dorsiflex the 5th	1	1
metacarpophalangeal joint to		
≥ 90 °		
Oppose the thumb to the volar	1	1
aspect of the ipsilateral forearm		
Hyperextend the elbow to $\geq 10^{\circ}$	1	1
Hyperextend the knee to $\geq 10^{\circ}$	1	1
Place hands flat on the floor		1
without bending the knees		
	Total	9
New sector for a strengt for a sector state for a sec		

One point is gained for each side for each maneuver 1-4, with a total possible score of 9 points.

upper limb and pauciarticular hypermobility. Furthermore, a hypermobile joint falling outside of the five sampled joints may be missed (Hakim & Grahame 2003b). Adults are considered to be hypermobile if they have four or more hypermobile joints, while five or more joints is the suggested cut-off point in children (Rikken-Bultmann et al. 1997).

The revised Brighton Criteria (see Box 6.3.2) were developed to clarify the relationship between hypermobility and musculoskeletal disorders (Grahame et al. 2000). The criteria were developed for research purposes but may also be used as a clinical tool to diagnose JHS. It is important to appreciate that a single symptomatic hypermobile joint is sufficient to satisfy the diagnosis of JHS. The 1998 Brighton Criteria take into account localized and/or pauciarticular hypermobility and this phenomenon is incorporated as a minor criterion for the diagnosis, along

Box 6.3.2

The Revised Brighton Criteria (Grahame et al. 2000) The Revised Brighton Criteria

Major criteria

- 1. A Beighton score of 4/9 or greater (current or historical)
- 2. Arthralgia for longer than 3 months in 4 or more joints

Minor criteria

- 1. A Beighton score of 1,2 or 3/9 (0-3 if aged 50 or more)
- 2. Arthralgia in 1–3 joints or back pain or spondylosis, spondylolysis/spondylolisthesis
- 3. Dislocation in more than one joint, or in one joint on more than one occasion
- 4. Three or more soft tissue lesions (epicondylitis, tenosynovitis, bursitis)
- Marfanoid habitus (tall, slim, arm span >height, upper segment:lower segment ratio less than 0.89, arachnodactyly)
- 6. Skin striae, hyperextensibility, thin skin or abnormal scarring
- Eye signs: drooping eyelids or myopia or antimongoloid slant

8. Varicose veins or hernia or uterine/rectal prolapse Hypermobility syndrome is diagnosed in the presence of two major criteria or one minor major and two minor criteria or four minor criteria. Two minor criteria will suffice where there is an unequivocally affected first degree relative. Hypermobility syndrome is excluded in the presence of Marfan, with the exception of the hypermobility type (formerly EDS III).

Box 6.3.3

Five Part Questionnaire for identifying hypermobility (adapted from Hakim & Grahame 2003a, 2003b)

Five part questionnaire for identifying hypermobility

- 1. Can you now or could you ever place your hands flat on the floor without bending your knees?
- 2. Can you now or could you ever bend your thumb to touch your forearm?
- 3. As a child you amuse your friends by contorting your body into strange shapes OR could you do the splits?
- 4. As a child or teenager did your shoulder ever dislocate on more than one occasion?
- 5. Do you consider yourself double jointed?

Answers in the affirmative to two or more questions suggest hypermobility with sensitivity 80–85% and specificity 80–90%.

with a number of other connective tissue signs and symptoms.

Hakim & Grahame (2003a) have recently developed a five part questionnaire (Box 6.3.3) for identifying hypermobility. This easy-to-use tool has demonstrated very good specificity and sensitivity, of 80% and 90%, respectively (Hakim & Grahame 2003b). The strength of this tool is that potentially it can be used to screen individuals with diffuse musculoskeletal symptoms in whom no clear cut degenerative or inflammatory disease has been identified (Hakim & Grahame 2003b). It also has excellent potential as a diagnostic tool in population epidemiologic studies, where physical examination is not possible or is impractical (Hakim & Grahame 2003a,b). Professor Bird stresses that while all these scoring systems are useful for screening purposes, they are not a substitute for a careful clinical examination of all of an individual's joints and tissues (Bird 2007).

Marfan's syndrome

The reported prevalence and incidence of hypermobility and JHS varies in the literature. It is difficult to compare studies because of variation in the screening and diagnostic criteria. Gender, ethnicity, and age are important factors, with hypermobility, being more prevalent in females and those of African or Asian descent (Bird 2007). While the populations with the most flexible joints appear to have fewer problems (Bird 2007), further population studies are required to establish this observation. Joint flexibility and hypermobility decrease with age (Grahame 2009). The prevalence of hypermobility in children is between 10 and 25%, with a higher incidence in females than males (Larsson et al. 1987). The prevalence of hypermobility in adults also varies, from as little as 5% in the USA (Jessee et al. 1980) to between 25% and 38% in Iraq and 43% being recorded in the Noruba tribe in Nigeria.

Recent studies in clinical populations showed the JHS phenotype was present in 58% of females and 29% of males among nonCaucasians in a west London rheumatology clinic. Similarly, in multicultural Oman, 55% of female patients between 18 and 50 years attending the rehabilitation outpatient department exhibited the JHS phenotype (Clark & Simmonds 2011).

A key question unanswered by research is whether there are two discrete populations, those who are asymptomatic and those who are predisposed to developing symptoms?

Clinical presentation of hypermobility syndrome

Hypermobility does not necessarily result in problems and may sometimes be considered an asset, but can be the cause of a variety of debilitating symptoms (Grahame 2003). The reason why certain individuals develop symptoms and others do not is not yet clear. The degree of joint and skin laxity and bruising has some association with injury risk and is likely to be related to a number of biopsychosocial influences (Murray 2006). Testing skin laxity is an important aspect of the clinical assessment (Fig. 6.3.3).

Symptoms frequently commence in early childhood, with the potential to continue into adult life. The predominant presenting complaint is pain, which is often widespread and longstanding, ranging from 15 days to 45 years. As early as birth, specific problems have been associated with hypermobility, including dislocation of the hip. Kirk et al. (1967) reported three quarters of hypermobile adolescents developing symptoms by the age of 15. Murray & Woo (2001) and Adib et al. (2005) recognize JHS as one of the most frequent causes of musculoskeletal symptoms in children and adolescents, particularly girls, aged between 13 and 19 years. Tofts et al. (2009) affirm this, stating:



Fig. 6.3.3 • Testing skin laxity. One suggested method is to draw the skin up from the 3rd metacarpal. The transparency of the skin can also be noted.

this condition can have significant negative impact on an affected child's ability to function and participate in society and is much more disabling than generally recognised.

Musculoskeletal signs and symptoms

Virtually all parts of the musculoskeletal system may be affected in JHS, with particular problems occurring at different ages from a combination of developmental changes or differences in growth patterns and the degree of physical activity. Individuals frequently look well and move well, which does not match complaints (Russek 1999; Simmonds & Keer 2007). This may lead to the patient being misunderstood and made to feel like a hypochondriac or labeled as having psychological problems (Child 1986).

In children, lower limb arthralgia is the commonest presentation (Murray 2006). These pains are usually biomechanical in origin and provoked by prolonged weightbearing and may be associated with periarticular joint swelling. Benign nocturnal pains, so-called "growing pains", are common in childhood and have been linked to hypermobility. It has been suggested that these pains are a result of minor injury following unusual or excessive physical exercise and sport (Murray & Woo 2001). Persistent pain can lead to avoidance behavior which can precipitate a downward spiral of deconditioning, leading to reduced functional capacity and confidence.

Laxity of ligaments predisposing to joint instability, subluxation, and dislocation is commonly reported (Hakim & Grahame 2003a). Congenital hip problems have been linked with hypermobility, most frequently recurrent subluxations ("clicky hips"), and less often hip dysplasia (Adib et al. 2005; Murray 2006) Such clicking of joints is also commonly reported in many other joints as children get older, both spontaneously and as habitual cracking of joints of the hand, temporomandibular joint, and spine. The origin of this is thought to be the formation and collapse of "air bubbles" in the joint, with sudden "excessive" distension of the joint spaces.

Children and adults commonly have recurrent and persistent pain over the anterior knee during or after prolonged sitting, sporting activities such as bicycleriding, and climbing stairs. They show varying combinations of hypermobile patellae, qualitatively poor quadriceps muscle bulk or function, genu recurvatum, and genu valgus, with "mis-tracking" of the patella a common phenomenon. Secondary chondromalacia patellae and recurrent subluxation are thought to cause some of the symptoms, as well as recurrent sprains and strains from "hanging on the ligaments" in a genu recurvatum posture.

Pes planus is almost uniform in hypermobile individuals, with collapse of the medial longitudinal arch of the foot over time, and spreading of the forefoot and ankle eversion even though patients may appear to have high arches in the resting position. Symptomatic patients benefit from podiatry input. Virtually all young children have a flat-footed appearance, but in normal persons the mature arches develop as ambulation matures.

Back pain is another common presentation in both children and adults which should be thoroughly investigated, particularly in children and adolescents (Murray 2006). Hypermobility syndrome is one of the most common differential diagnoses in this population (Grahame 1999). Spinal pain frequently presents in adolescents and may be related to growth spurts and associated biomechanical changes. The pain is often a result of muscle spasm and can be associated with scoliosis which may extend throughout the spine. It may also be related to excessive unaccustomed physical activity (Simmonds & Keer 2007). Conversely, obesity, excessive sedentariness, and poor fitness may also be contributory factors. These disorders may be the forerunner of chronic adult low back pain and cervical pain disorders. Where pain is severe or disabling, then other diagnoses need to be considered (i.e., spondylolysis, spondylolisthesis) and these are also more likely in hypermobile subjects. In young adults with hypermobility, disc prolapse as well as early degenerative osteoarthritis also need to be considered (Murray 2006).

Thumb and finger laxity will often be the first visual clue of generalized hypermobility. Impaired hand function is frequently observed in school children where prolonged handwriting is necessary and in adults who work extensively on laptop and personal computers.

Osteoarthritis

Joint hypermobility is a risk factor for premature osteoarthritis in the hands, knees and spine (Bird et al. 1978). Postulated mechanisms include joint instability and overuse, errors in collagen genes, or linkage between the genes for osteoarthritis and collagen abnormalities. Jónsson et al. (1996) observed associations with osteoarthritis of the hands, in particular the thumb.

Osteopenia and osteoporosis

Reduce bone density is found in EDS (Mishra et al. 1996), and in patients with JHS. Gulbahar et al. (2005) found hypermobility increased the risk for low bone mass in women by 1.8 times (95% confidence interval 1.01–3.38). A pediatric study found a lower bone mineral density in hypermobile patients, especially those with musculoskeletal pain. This suggests preventative measures including diet and appropriate load-bearing exercise should be instituted in the young when bone accrual is vital.

Neurophysiological disorders

Hypermobility has been associated with congenital hypotonia or "floppy infant" syndrome, late walking, and clumsiness reported by parents (Adib et al. 2005), and delayed motor development, which may be persistent (Kirby & Sugden 2007). There is reduced proprioception in adults (Mallik et al. 1994; Hall et al. 1995) and altered neuromuscular control in adolescents. Individuals with JHS are less accurate at reproducing proximal interphalangeal joint angles (Mallik et al. 1994). Position sense at the knee is decreased, particularly the ability to locate end-range extension (Hall et al. 1995). Laxity and fragility of connective tissue, coupled with a decreased proprioceptive acuity and altered neuromuscular reflexes, are possible causes of the predisposition of individuals with JHS to damage and injury (Ferrell et al. 2007).

There is a decreased efficacy of injected or topical local anesthetics as measured by sensory pain

thresholds. JHS patients were three times more likely to report poor efficacy of local anesthetic compared with non-hypermobile controls (Hakim & Grahame 2003a). The suggested mechanisms are mechanical and due to rapid clearance of anesthetic through lax soft tissues, or neurophysiological, due to some abnormality of nociception (Hakim & Grahame 2003a).

Cardiopulmonary disorders

Despite early findings, mitral valve prolapse (MVP) is no more common in JHS than in the female population at large, and aortic root dilatation (in contradistinction to MFS and EDS type IV) does not occur (Mishra et al. 1996). Varicose veins are seen in 9.5% of individuals with a Beighton score of 7–9 (Al-Rawi 1985).

Non-musculoskeletal symptoms such as fainting and palpitations in adults with JHS may be due to autonomic disturbance (Gazit et al. 2003) such as orthostatic hypotension, postural orthostatic tachycardia syndrome, or hyper-responsiveness in the BJHS to both α and β adrenoreceptor stimulation. Peripheral neuropathy or reduced vascular tone with lower extremity pooling may be pathophysiological mechanisms for these findings. Autonomic dysfunction with orthostatic tachycardia and hypotension occurs in children with chronic fatigue and EDS (Rowe et al. 1999).

There is an association of EDS and JHS with asthma, wheeze and cough, independent of age, gender, and smoking habit. The basis for this link may be genetic or mechanical secondary to a connective tissue defect. Increased risk of pneumothorax has also been reported (Bird 2007).

Chronic widespread pain and fatigue

Fibromyalgia is a condition of widespread chronic muscular pain and fatigue of unknown etiology, with multiple tender points at specific sites across the chest, neck, upper and lower back, elbows, thighs, and knees. Features of fibromyalgia are often encountered in JHS patients but evidence is conflicting regarding whether there is a true association between the two conditions (Murray 2006).

Spinal growth in the adolescent years, and recurrent minor injuries and muscle fatigue may well contribute to the onset of fibromyalgia in hypermobile adolescents (Murray 2006). The association between fibromyalgia and hypermobility was first reported by Gedalia and confirmed by Sendur et al. (2007) but not found by others.

Chronic fatigue syndrome (CFS), a close relation of chronic pain syndromes in adolescents, has also been linked to hypermobility. In 2005, van de Putte et al. demonstrated more connective tissue anomalies in CFS with lower systolic BP, higher skin extensibility, and higher arterial stiffness, but not overall joint mobility. Barron and co-workers (2002) reported a Beighton score average of 4 in CFS patients compared to 1 in healthy controls. Rowe and colleagues in 1999 reported 12 patients with CFS who also met criteria for EDS (six classical, six hypermobile types), all of whom had postural tachycardia and orthostatic hypotension. There are possibly subgroups of patients with CFS or fibromyalgia who are hypermobile, but whether hypermobility is a contributing factor to these conditions is still to be determined (Barron et al. 2002).

Principles of management

Although limited in number, intervention studies show improvement with exercise (Kerr et al. 2000). Ferrell et al. (2004) report improvements in proprioception and pain with closed chain exercises for individuals with JHS aged between 16 and 49 years. Other authors have reported improvements with a graduated exercise program combined with education, behavioral and lifestyle advice (Russek 1999; Simmonds & Keer 2007).

Because of the ubiquitous nature of collagen, JHS will present with a variety of different signs and symptoms. Therefore current best practice management of JHS is essentially an individualized problem-solving approach (Simmonds & Keer 2007). A multidisciplinary approach is recommended, including occupational therapists, podiatrists, physiotherapists, osteopaths, sports therapists (and sometimes psychologists), depending on the individual's needs. Physical education teachers, sports coaches, music and dance and classroom teachers all need to be need to be involved in the wider management plan, especially where children are concerned.

The key principles of treatment and managing JHS include:

- Treating the treatable, for example acute soft tissue lesions and injuries.
- Relieving pain where possible through the use of soft tissue work, gentle mobilizations, electrotherapy and support of joints and tissues.

- Education and behavior modification to enable individuals to manage the condition with minimal reliance on medical input or medication.
- Improving the endurance and strength capacity of the postural support and joint stabilizing muscles.
- Improving balance and coordination.
- Improving stamina and general fitness.
- Re-educating the gait to avoid or correct abnormalities in biomechanics.
- Facilitating a return to normal activities and functioning and promoting an active lifestyle

These aims work together to ensure that the individual has improved functional capacity, improved special awareness, joint stability and control, enabling independence and the minimum of external support.

Joint stability and muscle strength and improving dynamic muscle control to supplement the ligamentous insufficiency should minimize trauma to joints. Children respond well to a muscle-strengthening program, and while they may not improve their muscle bulk, they do improve strength and neuromuscular coordination, thus making the muscles more effective (Maillard, personal communication 2008). If the individual is experiencing significant pain, static exercises in the hypermobile range may precede dynamic work and then resisted work (Kerr et al. 2000) progressing from nonweightbearing to weightbearing work. Hydrotherapy is useful if weightbearing and land-based exercises prove difficult. Splinting of hypermobile joints is rarely recommended, as this is likely to exacerbate the ineffective use of the muscles, causing them to become weaker still and more prone to injury. Activity-related finger and hand splints can be very helpful for specific activities, for example practicing a musical instrument. Devices such as pen grips can be a good adjunct to a hand-muscle-strengthening program, as they decrease the grip force required, reducing the pain and fatigue experienced in fingers and wrists during school work. Back supports and knee braces may be required during acute exacerbation of pain and dysfunction. Tape can also be beneficial to help support a vulnerable joint and also to help facilitate proprioception and postural control (Simmonds & Keer 2007).

Gait re-education and functional rehabilitation

A combination of hypermobile joints, reduced proprioception, altered motor control, weak muscles and reduced stamina can profoundly affect gait. The causes of the abnormalities need to be identified and worked on separately before the gait will improve. The use of video recording and a mirror can give the individual helpful visual feedback on specific deficits.

Individuals with JHS often adapt to their hypermobility by altering their body mechanics, leading to increased pain, pain in other locations, and fatigue. It is therefore important to work on specific functional activities and to develop energy-saving, biomechanically correct, safe, and pain-free ways of moving which can be included in their rehabilitation program: i.e., step-ups and step-downs on a stair, repeated sitting to standing from a chair (Simmonds & Keer 2007).

Individuals with JHS often present with very pronated, flat feet, contributing to lower limb symptoms and altered gait pattern. This often responds very well to the use of shoe orthotics – heel cups or arch supports – that will support the subtalar joint and the medial arch. Rather than encouraging weaker foot muscles, correcting the biomechanics of the foot has such a positive effect upon the whole gait pattern that it is a preferential course of treatment (van de Putte et al. 2005). This also reduces the abnormal forces and pain throughout the other joints further up the kinetic chain.

Balance and proprioception

Techniques related to the common proprioception and balance deficits should be incorporated into the rehabilitation program. Proske & Gandevia (2009) emphasize skin in proprioception and kinesthetic sense. We recommend enhancing sensory input via the skin, by the use of "hands on" movement facilitation, wearing of tight-fitting clothing and neoprene gloves, and tape during specific exercise or functional rehabilitation sessions. "Rhythmical Stabilizations" is a useful method for facilitating postural stability. This can be further facilitated by practicing standing on one leg, without shoes and socks on, and by using a balance (wobble) board on uneven surfaces. Weightbearing exercises in four point kneeling are useful for combined upper and lower limb proprioception. Balance and proprioceptive exercises can also include the use of foam rolls and the Swiss ball (see Fig. 6.3.4a, b).

Pain relief

It is important for patients and family to understand that the pain is due to the hypermobility and associated musculoskeletal insufficiencies and not to any



Fig. 6.3.4 • Proprioception, stability and balance can be enhanced with (A) Swiss ball and (B) Half roll exercises.

other pathology such as an inflammatory arthritis condition. It is then easier to understand why a rehabilitation program is the treatment of choice. Individuals often need to be reassured that the pain will ease, but only when the muscles are strong and fit and are protecting the joints more fully. It is often found that the pain is the last thing to improve and only does so slowly, and this should be emphasized at the start of the program. It is important for the individuals to realize that the pain of JHS does not signify damage or primary inflammatory arthritis, but indicates sprain- and strain-type injury due to poor control of the joints, and is usually benign and self-limiting if managed well. Other methods of pain relief, such as hot packs or cold packs on specific joints, may be of use. Transcutaneous electrical nerve stimulation (TENS) machines must be seen only as a supportive treatment and not as a solution, and their use will not replace the rehabilitation program (Murray 2006). Hypermobile individuals may have both hypo- and hypermobile segments. Manual therapy, including mobilizations of stiff hypomobile joints can be very helpful; equally, soft tissue massage, trigger point work, and myofascial release can also alleviate pain associated with muscle spasm. Relaxation and visualization techniques can help the individual to manage pain and can often be very useful at night-time.

Pacing

Pacing is an extremely important part of the rehabilitation of individuals with JHS (Simmonds & Keer 2007). Activities such as gardening, cleaning, shopping, and playing sport on a weekend may result in an increase in pain on a Monday and inability to attend school or work; however, by the next weekend they may have recovered enough to be able to do many activities again, and the cycle continues. This causes a constant "peaking and troughing" of symptoms and causes a major disruption to life. The idea of pacing is to stop this rise and fall of symptoms by leveling out activities and gradually building them up again. Specific tasks are, therefore, set for each day whether there is a lot of pain or just a little. These tasks are determined by the level of disability and fitness at the initial phase of the program. This is then gradually increased weekly until individuals are able to achieve functional activity goals.

Psychology

"Where pain and loss of independent function are profound, it is important to include a clinical psychologist or psychotherapist in the management team to identify psychosocial and emotional stressors, as well as patterns of belief and cognitive functioning which are maladaptive (Murray 2006). Psychologists or therapists with psychological training can help patients to develop pain management skills and help the family to cope and to understand the impact on the whole family. Many families find this support invaluable and are able to change many unhelpful coping strategies into helpful ones, and are able to leave behind the "chronic pain cycle" that may have developed.

General fitness

Individuals with JHS often become more sedentary due to their pain and weakness, and therefore frequently become deconditioned and lacking in general fitness (Simmonds & Keer 2007). It is therefore important to incorporate some aerobic fitness work into the rehabilitation program. Care needs to be taken early on in the program, when they still have suboptimal muscle strength, to ensure that the fitness aspect of the program is of low impact to the joints so that the joint symptoms are not increased and the individual and family do not lose faith in the program. Children and family are commonly resistant to therapy interventions as they have often experienced increasing pain from previous attempts. This is usually due to progressing the program too fast and/or poor compliance with regular home exercise needed to activate and build muscle strength. Liaising with school physical education teachers is an important aspect of the management of children (Simmonds & Keer 2007). Swimming and deep water running are desirable methods of exercise as there is generally less stress on the joints and they provide an opportunity for aerobic exercise. It is preferable that a normal swimming pool is used for ongoing management, as hydrotherapy pools are too hot for distance swimming. Tai Chi and Pilates are also recommended as they facilitate balance and control. Bicycling is also very good for aerobic work and again does not over-stress the other joints. Nordic pole walking can also be effective and it is hypothesized that the closed chain nature of the activity assists proprioceptive feedback and also facilitates the engagement of the trunk sling muscles, thus increasing core and trunk stability. However, as soon as the strength improves, normal aerobic and sporting activities can be included, provided that they form part of a pacing program.

Conclusions

Joint hypermobility Syndrome is a relatively common phenomenon, which may be an asset or may predispose to a range of clinical problems. Because of the ubiquitous nature of collagen, JHS may present in a variety of clinical presentations. Positive recognition, and avoidance of unnecessary investigations, and drug therapy are among the most important interventions. Most children are well managed with simple advice and reassurance, while adults frequently require a more structured rehabilitation program. Modification of activities and behavior may be required to redress the balance between healthy physical activities and high-impact physical pursuits. If untreated or undiagnosed, JHS may result in the development of a chronic pain cycle and a high level of disability. This will then require an intensive rehabilitation program to manage the symptoms effectively and improve functional capacity. It is vital that individuals with JHS and their families are clear in their understanding of the condition. It is also important to stress that a self-management-led program with support from health, sport, and exercise professionals is the most appropriate long-term treatment approach. The future will no doubt yield more appropriate assessment tools and perhaps genetic analyses for identifying individuals at risk, thus allowing earlier implementation of preventive strategies.

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Fascia: The Tensional Network of the Human Body

The science and clinical applications in manual and movement therapy

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Glossary

Heike Jäger

- Actin A globular protein found in all eukaryotic cells, polymerizes to microfilaments; one of the three major components of the <u>cytoskeleton</u> and thin filaments of the contractile apparatus. Actin has an array of functions including muscle contraction, cell signaling and morphology, vesicle and organelle movement, cell motility, phagocytosis, and cytokinesis.
- **Adhesions** Inflammatory bands of scar-like tissue that form between two surfaces inside the body.
- **Adhesive capsulitis** An inflammatory condition that restricts motion in the shoulder, commonly referred to as "frozen shoulder".
- Alpha smooth muscle actin One of six known smooth muscle actin isoforms. In addition to its presence in organ tissue, alpha smooth muscle actin has been identified in myofibroblasts, where it plays an important role in focal adhesion maturation and in cell motility.
- **Angiogenesis** A physiological process involving the growth of new blood vessels from pre-existing vessels, for example in the process of wound healing.
- **Aponeurosis** A thin, flat tendon-like expansion of fascia important in the attachment of muscles to bones.
- **Apoptosis** A morphologic pattern of cell death affecting single cells, marked by formation of cytoplasmic blebs, shrinkage of the cell, condensation of chromatin, and fragmentation of the cell into membrane-bound apoptotic bodies that are eliminated by phagocytosis. It is a mechanism for cell deletion in the regulation of cell populations.
- **Bradykinin** A nonapeptide produced by activation of the kinin system in a variety of inflammatory conditions. A potent vasodilator, it also increases vascular permeability, stimulates pain receptors, and causes contraction of a variety of extravascular smooth muscles.
- **Calcitonin gene-related peptide (CGRP)** A 37-amino acid polypeptide is formed from the alternative splicing of the calcitonin/CGRP gene and acts as a potent vasodilator and neurotransmitter. Widely distributed in

the central and peripheral nervous systems; also present in the adrenal medulla and gastrointestinal tract.

- **Cell signaling** The process by which a cell receives and acts on some external chemical or physical signal, including receiving the information at specific receptors in the plasma membrane, conveying the signal across the plasma membrane into the cell, and subsequently stimulating a specific cellular response.
- **c-fiber** Unmyelinated nerve fiber that conducts action potentials at a velocity of less than 2.5 m/s in humans.
- **Chondroblasts** Immature cartilage cells that produce the cartilaginous matrix.
- **Chromatin** The more readily stainable portion of the cell nucleus, forming a network of nuclear fibrils. Comprised of DNA attached to a protein structure base (primarily histones). Occurs in two states, euchromatin and heterochromatin, with different staining properties, and during cell division coils and folds to form the metaphase chromosomes.
- **Collagen** The most abundant protein in mammals; a major component of fascia, giving it strength and flexibility. At least 14 types exist, each composed of tropocollagen units that share a common triple-helical shape but varying somewhat in composition between types, with the types being localized to different tissues, stages, or functions.
- **Compartment syndrome** Involves the compression of nerves and blood vessels within a fascial compartment, leading to impaired blood flow and muscle and nerve damage. Most common in the lower leg or forearm.
- **Cytokines** A generic term for non-antibody proteins released by one cell population (e.g., primed T lymphocytes) on contact with specific antigens, which act as intercellular mediators, as in the generation of an immune response.
- **Cytoskeleton** The conspicuous internal reinforcement in the cytoplasm of a cell, consisting of tonofibrils, terminal web, or other microfilaments.

Deep fascia (or fascia profunda) The dense fibrous fascia that interpenetrates and surrounds the muscles as well as muscle groups.

Differentiated myofibroblast A myofibroblast that expresses alpha smooth muscle actin stress fiber bundles.

- **Dry needling** An invasive procedure in which an acupuncture needle is inserted at myofascial trigger points to inactivate it.
- **Dupuytren's contracture** A thickening and contracture of the palmar fascia.
- **Dynamometer** An instrument for measuring the force of muscular contraction.
- **Ehlers–Danlos syndrome** A group of inherited connective tissue disorders, caused by a defect in the synthesis of <u>collagen</u>. Occurs in at least 10 types, varying in severity from mild to life-threatening. Transmitted genetically as autosomal recessive, autosomal dominant, or X-linked recessive traits. The major manifestations include hyperextensible skin and joints, easy bruisability, friability of tissues with bleeding and poor wound healing, calcified subcutaneous spheroids, and pseudotumors.
- **Elastin** A scleroprotein, the essential constituent of yellow elastic connective tissue. Brittle when dry, but when moist is flexible and highly extensile.
- **Electron microscopy** An imaging technique using electrons to illuminate and create an image of a specimen. It has much higher magnification and resolving power than a light microscope, with magnifications up to about two million times compared to about 2000 times. Unlike a light microscope, which uses glass lenses to focus light, the electron microscope uses electrostatic and electromagnetic lenses to control the illumination and imaging of the specimen.
- **Endomysium** The fascial layer that sheaths single muscle fibers.
- **Endotenon** A thin fascial membrane within a tendon that invests each collagen fibril and each collagen fiber, and envelops the primary, secondary and tertiary fiber bundles.
- **Endothelium** The layer of epithelial cells that lines the cavities of the heart, the lumina of blood and lymph vessels, and the serous cavities of the body.
- **Epimysium** The fascial layer that envelops an entire muscle.
- **Epineurium** The outermost fascial layer of a peripheral nerve, surrounding the entire nerve and containing its supplying blood vessels and the lymphatic system.
- **Epitenon** A fine, loose connective tissue sheath covering a tendon over its entire length.
- **Extracellular matrix** Any material produced by cells and excreted to the extracellular space within the tissues. Takes the form of both ground substance and fibers and is composed chiefly of fibrous elements, proteins involved in cell adhesion, and glycosaminoglycans and other molecules. It serves as a scaffolding holding tissues

together and its form and composition help determine tissue characteristics.

- **Fascia** The soft tissue component of the connective tissue system. Interpenetrates and surrounds muscles, bones, organs, nerves, blood vessels and other structures. Fascia is an uninterrupted, three-dimensional web of tissue that extends from head to toe, from front to back, from interior to exterior. Fascia can refer to dense planar fascial sheets (such as the fascia lata) as well as joint capsules, organ capsules, muscular septa, ligaments, retinacula, aponeuroses, tendons, myofascia, neurofascia, and other fibrous collagenous tissues (see introduction Chapter, page xvi).
- **Fascial distortion model (FDM)** A diagnostic as well as treatment system for fascial distortions developed by Stephen Typaldos (see Chapter 7.15).
- **Fasciotomy** A surgical incision or transection of fascia, often performed to release pressure in compartment syndrome.
- **Fibroblasts** Flat elongated fascial cells with cytoplasmic processes at each end, and a flat, oval, vesicular nucleus. Fibroblasts form the fibrous tissues in the body, including tendons, aponeuroses, supporting and binding tissues of all sorts.
- **Fibronexus** An adhesion in a myofibroblast that links actin across the cell membrane to molecules in the extracellular matrix like fibronectin and collagen.
- **Fibrosis** The formation of fibrous tissue, as in repair or replacement of parenchymatous elements.
- **Gap junctions** Direct connections between the cytoplasms of two cells, allowing various molecules and ions to pass, e.g., most sugars, amino acids, nucleotides, vitamins, hormones, and cyclic AMP. A gap junction channel is composed of two connexons (or hemichannels), which connect across the intercellular space. In electrically excitable tissues, these gap junctions serve to transmit electrical impulses via ionic currents and are known as electronic synapses.
- **Glycosaminoglycans (or mucopolysaccharides)** High molecular weight linear heteropolysaccharides having disaccharide repeating units containing an *N*-acetylhexosamine and a hexose or hexuronic acid; either or both residues may be sulfated. This class of compounds includes the chondroitin sulfates, dermatan sulfates, heparan sulfate and heparin, keratan sulfates, and hyaluronic acid. All except heparin occur in proteoglycans that consist of glycosaminoglycans <u>covalently linked</u> to a <u>protein</u>.
- **Golgi receptors** Mechanosensory receptors found in dense proper fascia, in ligaments (Golgi end organs), in joint capsules, as well as around myotendinous junctions (Golgi tendon organs).
- **Hyaluronic acid** A glycosaminoglycan; part of the extracellular matrix of synovial fluid, vitreous humor, cartilage, blood vessels, skin, and the umbilical cord. Along with lubricin, it maintains viscosity of the extracellular matrix, allowing for necessary lubrication of certain tissues.

Glossary

Hypermobility (or laxity) A greater than normal range of motion in a joint, which may occur naturally in otherwise normal persons or may be a sign of joint instability.

Hypertonia Excessive tone of the skeletal muscles that increases their resistance to passive stretching.

Hypertrophy The enlargement or overgrowth of an organ or part due to an increase in size of its constituent cells.

Hypocapnia A deficiency of carbon dioxide in the blood, resulting from hyperventilation and eventually leading to alkalosis.

Hysteresis A property of systems that do not instantly react to the forces applied to them, but react slowly, or do not return completely to their original state.

Immunoassay A biochemical test that measures the concentration of a substance in solution, using an antigen/antibody reaction.

Immunofluorescence A technique that uses fluorescent dye-labeled antibodies to visualize subcellular distribution of biomolecules of interest.

Integrins Refers to any of a family of heterodimeric celladhesion receptors, consisting of two non-covalently linked polypeptide chains, designated α and β , that mediate cell-to-cell and cell-to-extracellular matrix interactions.

Interstitial fluid The extracellular fluid that bathes the cells of most tissues but which is not within the confines of the blood or lymph vessels and is not a transcellular fluid. Formed by filtration through the blood capillaries and drained away as lymph. It is the extracellular fluid volume minus the lymph volume, the plasma volume, and the transcellular fluid volume.

Laminin An adhesive glycoprotein component of the basement membrane. It binds to heparan sulfate, type IV collagen, and specific cell-surface receptors and is involved in the attachment of epithelial cells to underlying connective tissue.

Ligament A band of fascia that connects bones or supports viscera. Some are distinct fibrous structures; some are folds of fascia or of indurated peritoneum; others are relics of fetal vessels or organs.

Mechanoreceptors Sensory receptors that respond to mechanical pressure, deformation or proprioception.

Mechanotransduction The mechanism by which cells convert a mechanical stimulus into chemical activity.

Mepyramine An antihistaminic pharmacological substance that is frequently used as an in vitro contractile agent for tissues containing myofibroblasts.

Microdialysis A diagnostic technique that uses an ultrafine needle to biopsy the chemical components of the fluid in the extracellular space of tissues.

Morphogenesis The evolution and development of form, as in the development of the shape of a particular organ or part of the body.

Myofascial pain syndrome A chronic musculoskeletal pain disorder associated with local or referred pain, decreased range of motion, autonomic phenomena, local twitch response in the affected muscle, and muscle weakness without atrophy. **Myofibroblasts** Differentiated fibroblasts that combine the features of both fibroblasts and smooth muscle cells. Due to their expression of stress fiber bundles containing alpha smooth muscle actin and due to strengthened adhesion sites on their membrane, these cells possess a much higher contractile potential than normal fibroblasts.

Myosin The most abundant protein in muscle, occurring chiefly in the A band. Along with actin, it is responsible for the contraction and relaxation of muscle. Myosin uses ATP hydrolysis to generate force and to "walk" along the filament. It is the main constituent of the thick filaments of muscle fibers.

Neuropathy A functional disturbance or pathological change in the peripheral nervous system.

Neuroplasticity The changes that occur in the organization of the brain as a result of experience.

Nociceptors Receptors for pain that are activated by physical, mechanical, thermal, electrical, or chemical stimuli.

Osteoblasts Cells that arise from fibroblasts and are associated with the production of bone.

Oxytocin A nonapeptide secreted by the magnocellular neurons of the hypothalamus and stored in the neurohypophysis along with vasopressin. It promotes uterine contractions, milk ejection, contributes to the second stage of labor and is released during orgasm in both sexes. In the brain, oxytocin regulates circadian homeostasis, such as body temperature, activity level, and wakefulness. It is involved in social recognition, bonding, and trust formation.

Pacinian corpuscles Lamellar or lamellated large encapsulated nerve endings located in fascia that are sensitive to vibration, and acceleration of movement. They require dynamically changing stimuli and do not respond to static pressure.

Perimysium The fascial membrane which groups individual muscle fibers (between 10 to 100+) into bundles or fascicles.

Perineurium An intermediate layer of fascia in a peripheral nerve, surrounding each bundle (fasciculus) of nerve fibers.

Piezoelectric The ability of some materials to generate an electric potential in response to applied mechanical stress.

Plantar fasciitis An inflammatory condition of the plantar fascia.

Plantar fibromatosis The formation of fibrous, tumorlike nodules arising from the deep layer of the plantar fascia, manifested as single or multiple nodular swellings, sometimes accompanied by pain but usually unassociated with contractures.

Prestress Endogenous tension.

Procollagen The precursor molecule of collagen, synthesized in the fibroblast, osteoblast, etc., and cleaved to form collagen extracellularly.

Prolotherapy (or RIT, regenerative injection therapy) An injection therapy used to treat chronic ligament, joint, capsule, fascial and tendinous injuries to promote non-surgical soft tissue repair and to relieve pain. **Proprioception** Perception mediated by sensory nerve endings found in muscles and fascia, which give information concerning movement and position of the body.

- **Proteoglycans** Heavily glycosylated glycoproteins that are found in the extracellular matrix of fascia, composed mainly of polysaccharide chains, particularly glycosaminoglycans, as well as minor protein components that form large complexes, both to other proteoglycans, to hyaluronan and to fibrous matrix proteins (such as collagen).
- **Protomyofibroblasts** Develop from fibroblasts under mechanical tension. They form cytoplasmic actincontaining stress fibers that terminate in fibronexus adhesion complexes.
- **Reticular fibers** Fascial fibers composed of collagen type III that form the reticular framework of lymphoid and myeloid tissue and also occur in the interstitial tissue of glandular organs, the papillary layer of the skin, and elsewhere.
- **Retinaculum** A thickened band of fascia that retains an organ or tissue in place.
- **Ruffini endings** Types of lamellated corpuscle that are slowly-adapting receptors for sensations of continuous pressure.
- **Sarcoplasmic reticulum** A special form of a granular reticulum found in the sarcoplasm of striated muscle and comprising a system of smooth-surfaced tubules forming a plexus around each myofibril.
- **Sclerosis** An induration or hardening caused by inflammation, fascial thickening, or disease of the interstitial fluid.
- **Serotonin** A monoamine vasoconstrictor, synthesized in the intestinal chromaffin cells or in central or peripheral neurons and found in high concentrations in many body tissues, including the intestinal mucosa, pineal body, and central nervous system.
- **Substance P** An undecapeptide that functions as a neurotransmitter and as a neuromodulator and belongs to the tachykinin neuropeptide family.
- **Superficial fascia** Comprised mainly of loose areolar connective tissue and adipose. In addition to its subcutaneous presence, this type of fascia surrounds organs and glands, neurovascular bundles, and is found at many other locations.
- **Surface electromyography** A technique in which electrodes are placed on (not into) the skin overlying a muscle to detect the electrical activity of the muscle.
- **Tendon** A fibrous cord of fascia by which a muscle is attached to the skeleton.
- **Tendon sheath** A membranous sleeve which envelops the tendon and creates a lubricated low-friction environment for easy movement.
- **Tensegrity** A structural principle which uses isolated components in <u>compression</u> inside a net of continuous tension.
- **Transdifferentiation** A biological process that occurs when a non-stem cell transforms into a different type of

cell, or when an already differentiated stem cell creates cells outside its already established differentiation.

- **Transforming growth factor** Two classes that are structurally or genetically not related to one another. TGF- α binds the epidermal growth factor receptor and also stimulates growth of microvascular endothelial cells. TGF- β exists in several subtypes, all of which are found in hematopoietic tissue, stimulate wound healing, and in vitro are antagonists of lymphopoiesis and myelopoiesis.
- **Trigger point** Palpable as localized hardening in the muscle; pain evoked by pressure on the tender spot is recognized as being familiar by the patient, local twitch response is possible; limitation of stretch range of motion, and some weakness of that muscle.
- **Tropocollagen** The basic structural unit of collagen; a helical structure consisting of three polypeptide chains, each chain composed of about a thousand amino acids, coiled around each other to form a spiral and stabilized by inter- and intrachain covalent bonds.
- Tropoelastin The precursor of elastin.
- **Tropomyosin** Along with troponin, regulates the shortening of the muscle protein filaments actin and myosin. In the absence of nerve impulses to muscle fibers, tropomyosin blocks interaction between myosin crossbridges and actin filaments.
- **Ultrasound elastography** A non-invasive imaging method to measure stiffness or strain of soft tissue or to provide images of tissue morphology or other biomechanical information.
- Vimentin filaments Intermediate filaments of the cytoskeleton that are responsible for maintaining cell integrity. They act as cytoskeletal support structures, play a role in mitosis, and are clustered particularly around the nucleus, probably helping to control its location.
- **Vinculin** A protein found in muscle, fibroblasts, and epithelial cells that binds actin and appears to mediate attachment of actin filaments to integral proteins of the plasma membrane.
- **Viscoelastic** Describes materials that exhibit both viscous and elastic characteristics when undergoing plastic deformation. Viscous materials, like honey, resist shear flow and strain linearly with time when a stress is applied. Elastic materials strain instantaneously when stretched and just as quickly return to their original state once the stress is removed. Viscoelastic materials have elements of both of these properties and, as such, exhibit time dependent strain.
- **Wolff's law** The theory developed by 19th century anatomist/surgeon Julius Wolff stating that bone in a healthy person or animal will adapt to the loads it is placed under. If loading on a particular bone increases, the bone will remodel itself over time to become stronger to resist that sort of loading. The converse is also true, i.e., if the loading on a bone decreases, the bone will become weaker due to turnover as it is less metabolically costly to maintain and there is no stimulus for continued remodeling required to maintain bone mass.

Welcome to the world of fascia!

This book is the first comprehensive text in a new field in musculoskeletal therapy and research: the fascinating world of fascia. Fascia forms a continuous tensional network throughout the human body, covering and connecting every single organ, every muscle, and even every nerve or tiny muscle fiber. After several decades of severe neglect, this "Cinderella of orthopedic science" is developing its own identity within medical research. The number of research papers on fascia in peer-reviewed journals has shown a steady rise. The first International Fascia Research Congress, held at the Conference Center, Harvard Medical School in October 2007 was followed by a second in Amsterdam in 2009 and there will shortly be a third in Vancouver in 2012. Similar to the rapidly growing field of glia research in neurology, this underestimated contextual tissue, fascia, is being found to play an important role in health and pathology.

Hypotheses which accord myofascia a central role in the mechanisms of therapies have been advanced for some time in the fields of acupuncture, massage, structural integration, chiropractic and osteopathy. Practitioners in these disciplines, especially those which do not have the longevity of osteopathy or chiropractic, are generally unaware of the scientific basis for evaluating such hypotheses. Many practitioners are unaware of the sophistication of current laboratory research equipment and methods. Laboratory researchers, in turn, may be unaware of the clinical phenomena which suggest avenues of exploration. Thirty years ago the study of physical medicine and rehabilitation included muscle strengthening, anatomy, exercise physiology, and other aspects of therapeutic modalities. What was notably less present in the scientific and medical literature was how to understand and treat disorders of the fascia and connective tissues. Since then much additional information has been developed, particularly since 2005 (see Fig. 0.1).

The purpose of this book is to organize relevant information for scientists involved in the research of the body's connective tissue matrix (fascia) as well as for professionals involved in the therapeutic manipulation of this body wide structural fabric. While it grew out of materials presented at the First and the Second International Fascia Research Congresses in 2007 and 2009 (www.fasciacongress.org), it reflects the efforts of almost 100 scientists and clinicians.

Not only a packing organ

As every medical student knows and every doctor still remembers, fascia is introduced in anatomy dissection courses as the white packing stuff that one first needs to clean off, in order "to see something". Similarly, anatomy books have been competing with each other, in how clean and orderly they present the locomotor system, by cutting away the whitish or semitranslucent fascia as completely and skillfully as possible. Students appreciate these appealing graphic simplifications, with shiny red muscles, each attaching to specific skeletal points. However, these simplified maps do not fully describe how the real body feels and behaves, be it in medical surgery or during therapeutic palpation.

To give an example: in real bodies, muscles hardly ever transmit their full force directly via tendons into the skeleton, as is usually suggested by our textbook drawings. They rather distribute a large portion of their contractile or tensional forces onto fascial sheets. These sheets transmit these forces to synergistic as well as antagonistic muscles. Thereby they stiffen not only the respective joint, but may even affect regions several joints further away. The simple questions discussed in musculoskeletal textbooks "which muscles" are participating in a particular movement thus become almost obsolete. Muscles are not functional units. no matter how common this misconception may be. Rather, most muscular movements are generated by many individual motor units, which are distributed over some portions of one muscle, plus other portions of other muscles. The tensional forces of these motor units are then transmitted to a complex network of fascial sheets. bags, and strings that convert them into the final body movement.



Fig. 0.1 • Number of peer reviewed scientific papers on fascia • Papers on fascia indexed in Ovid Medline or Scopus have grown from 200 per year in the 1970s and 1980s to almost 1000 in 2010.

Similarly, it has been shown that fascial stiffness and elasticity play a significant role in many ballistic movements of the human body. First discovered by studies of the calf tissues of kangaroos, antelopes, and later by horses, modern ultrasound studies have revealed that fascial recoil plays in fact a similarly impressive role in many of our human movements. How far you can throw a stone, how high you can jump, how long you can run, depends not only on the contraction of your muscle fibers; it also depends to a large degree on how well the elastic recoil properties of your fascial network are supporting these movements.

If the architecture of our fascial network is indeed such an important factor in musculoskeletal behavior, why has this tissue been overlooked for such a long time? There are several answers to this question. The development of new imaging and research tools now allow us to study this tissue in vivo. Another reason is that this tissue resists the classical method of anatomical research: that of splitting something into separate parts that can be counted and named. You can reasonably estimate the number of bones or muscles; yet any attempt to count the number of fasciae in the body will be futile. The fascial body is one large networking organ, with many bags and hundreds of rope-like local densifications, and thousands of pockets within pockets, all interconnected by sturdy septa as well as by looser connective tissue layers.

What is fascia?

This varied nature of fascia is reflected in the many different definitions of which exact tissue types are included under the term "fascia". The International Anatomical Nomenclature Committee (1983) confirmed the usage of previous nomenclature committees and used the term "fascia superficialis" for the loose layer of subcutaneous tissue lying superficial to the denser layer of "fascia profunda." While most medical authors in English-speaking countries followed that terminology, it was not congruently adopted by authors in other countries. The nomenclature proposed by the Federative Committee on Anatomical Terminology (1998), therefore attempted to lead towards a more uniform international language (Wendell-Smith 1997). It suggested that authors should no longer use the term fascia for loose connective tissue layers, such as the former "superficial fascia", and to apply it only for denser connective tissue aggregations. However, this attempt failed significantly (Huijing & Langevin 2009). Most English textbook authorities continued to use the term "superficial fascia" to describe subcutaneous tissues (Standring 2008). In addition an increasing number of non-English authors - following the common Anglo-Saxon trend in international medicine – have started to adopt the same terminology as these American or British colleagues.

Similarly there has been confusion on the question which of the three hierarchical muscular tissue bags – epi-, peri- and endomysium – could be included as fascia. While most authors would agree to consider as fascial tissues, muscular septi and the perimysium (which is often quite dense, particularly in tonic muscles) there is less consensus on the endomysial envelopes around single muscle fibers, based on their much looser density and higher quantity of collagen types III and IV. However, almost all authors emphasize the important continuity of these intramuscular connective tissues, and this continuity was shown extending even within the muscle cell (Purslow 2009). So where does fascia stop?

Another area , still to be resolved, are the visceral connective tissues. For some authors the term fascia is restricted to muscular connective tissues. Visceral connective tissues – no matter whether they are of loose composition like the major omentum or more ligamentous like the mediastinum – are often excluded. In contrast, more clinically oriented books have placed a lot of emphasis on the visceral fasciae (Paoletti 2006, Schwind 2006).

As valuable as these proposed anatomical distinctions within soft connective tissues are, their very detail may lead to unwitting exclusion of important tissue continuities which are only perceived on the larger scale. For example, the clinical significance

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of the continuity of the fascia of the scalene muscles of the neck with the pericardium and mediastinum inside the thorax is often surprising in our discussions with orthopedic surgeons, although less so to osteopaths or general surgeons. Figure 0.2 shows another example of perceptual tissue exclusion, based on terminological distinction. Here one of the sturdiest portions of the iliotibial tract has been excluded from this important tissue band, since it did not fit the distinct nomenclature defined by the authors of this paper.

Based on this background a more encompassing definition of the term fascia was recently proposed as a basis for the first Fascia Research Congress (Findley & Schleip 2007) and was further developed (Huijing & Langevin 2009) for the following congresses. The term fascia here describes the 'soft tissue component of the connective tissue system that permeates the human body'. One could also describe these as fibrous collagenous tissues which are part of a bodywide tensional force transmission system. This view of an interconnected tensional network is partly inspired by the tensegrity concept, as



Fig. 0.2 • Example of a fascia dissection based on specific terminology • This dissection was used in an otherwise excellent treatise on the iliotibial tract (ITT). Following the proposal of the Federative Committee on Anatomical Terminology (1998) to distinguish between aponeuroses and fasciae, the authors chose to describe this tissue as an aponeurosis. Congruently with this decision, their dissection and illustration therefore excluded all tissue portions with a non aponeurotic character. Unfortunately this included one of the most dense and most important portions of the iliotibial tract: the connection to the lateral iliac crest, posterior of the anterior superior iliac spine. Notice the common thickening of the iliac crest at the former attachment of this ligamentous portion (located at a straight force transmission line from the knee over the greater trochanter), reflecting the very strong pull of this tissue portion on the pelvis. (TFL: tensor fascia lata.) Illustration taken with permission from Benjamin et al 2008)

described in Chapter 3.5. The complete fascial net then includes not only dense planar tissue sheets (like septa, joint capsules, aponeuroses, organ capsules, or retinacula), which may also be called "proper fascia", but it also encompasses local densifications of this network in the form of ligaments and tendons. Additionally it includes softer collagenous connective tissues like the superficial fascia or the innermost intramuscular layer of the endomysium. The cutis, a derivative of the ectoderm, as well as cartilage and bones are not included as parts of the fascial tensional network. However, the term fascia now includes the dura mater, the periosteum, perineurium, the fibrous capsular layer of vertebral discs, organ capsules as well as bronchial connective tissue and the mesentery of the abdomen (Fig. 0.3).

This more encompassing terminology offers many important advantages for the field. Rather than having to draw most often arbitrary demarcation lines between joint capsules and their intimately involved ligaments and tendons (as well as interconnected





Fig. 0.3 • Different connective tissues considered here as fascial tissues • Fascial tissues differ in terms of their density and directional alignment of collagen fibers. For example, superficial fascia is characterized by a loose density and a mostly multidirectional or irregular fiber alignment; whereas in the denser tendons or ligaments the fibers are mostly unidirectional. Note that the intramuscular fasciae septi, perimysium and endomysium - may express varying degrees of directionality and density. The same is true although to a much larger degree - for the visceral fasciae (including soft tissues like the omentum majus and tougher sheets like the pericardium). Depending on local loading history, proper fasciae can express a two-directional or multidirectional arrangement. Not shown here are retinaculae and joint capsules, whose local properties may vary between those of ligaments, aponeuroses and proper fasciae.

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aponeuroses, retinacula and intramuscular fasciae), fascial tissues are seen as one interconnected tensional network that adapts its fiber arrangement and density according to local tensional demands. This terminology fits nicely to the Latin root of the term "fascia" (bundle, strap, bandage, binding together). It is also synonymous with the nonprofessional's understanding of the term "connective tissue". "Connective tissue research" is too broad a term, as this includes bones, cartilage and even blood or lymph, all of which are derivatives of the embryologic mesenchyme. In addition, the contemporary field of 'connective tissue research' has shifted its primary focus to tiny molecular dynamics from the macroscopic considerations of several decades ago. The newly forming field of fascia research requires both macroscopic and microscopic investigations. This text has undertaken the task of serving both areas. Even if sometimes microscopic details of collagenous tissues are explored, an effort will be made to always relate these findings to the body as a whole.

While we see great advantages in our wider definition of fascial tissues, we acknowledge that more traditionally oriented authors will continue to restrict the term fascia to dense planar layers of "irregular" connective tissues, in distinction from more regular oriented tissues like aponeuroses or ligaments. In some areas such a distinction is indeed possible and may be clinically useful (e.g. at the fasciae and aponeuroses of the lumbar region). We therefore suggest including twelve additional specifying terms wherever possible, into the detailed description of a fascial tissue. These specifying terms were proposed by Huijing & Langevin (2009): dense connective tissue, areolar connective tissue, superficial fascia, deep fascia, intermuscular septa, interosseal membrane, periost, neurovascular tract, epimysium, intra- and extramuscular aponeurosis, endomysium. However, we also note that many important areas of the body are characterized by gradual transitions between such morphological categories, and a more geometrical description of local collagen architecture (in terms of dominant fiber directions, tissue thickness and density) may then be more useful to understanding specific tissue properties (see Fig. 0.2).

This textbook, as have the fascia congresses, has taken the difficult role of being oriented toward both the scientist and the clinician. Material presented spans anatomy and physiology of fascia in Part 1, through clinical conditions and therapies in Part 2, to recently developed research techniques in Part 3. We have pointed out the definitional struggles the researcher faces surrounding fascia: Which tissue? What fiber directions? What is connected to what? These research tools will allow the extension of this debate to more clinical areas as well, to help define which tissues are affected and what directions forces are applied in the clinical therapies. It is our hope that clinicians and scientists, both together and separately, will rise to these challenges to advance our basic understanding and our clinical treatment of fascia.

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Inclusion criteria and overview

Leon Chaitow

Criteria for topic inclusion in this chapter

As research into, and knowledge of, connective tissue and fascia have grown over the past decade, so has interest grown amongst practitioners and therapists in respect to their multiple roles in the body.

The renewed and expanded focus on this topic has resulted in two World Congresses on Fascia Research *First* International *Fascia Research Congress*, Harvard Medical School, Boston, October 2007 *Second* International *Fascia Research Congress*, Free University, Amsterdam, October 2009. As demand has grown for an evidence-base to support the safe use of manual and exercise-based therapeutic modalities (Sackett 2000), it has become increasingly important to attempt to establish which of the many techniques, modalities, systems and methods currently in use by manual therapists, practitioners and physicians, actually do influence fascial behavior.

Additionally, the mechanisms involved when fascial structures are treated, manually or by other means (needling, mechanical force, exercise, etc.), have attracted growing research interest.

The selection of topics in this chapter therefore reflects a wide spectrum of modalities, ranging from approaches where fascia is clearly and directly affected by the therapeutic methods under discussion, to those where effects on fascia are more speculative and general.

In some examples there is compelling evidence of a fascial connection, as in descriptions by Stecco and Stecco, of Fascial Manipulation[®], in Chapter 7.7.

In other chapters, the connection between the method under review and fascia is more hypothetical;

for example, in descriptions (Chapter 7.22) of the possible influences on fascial structures of Pilates methods. This in no way implies that Pilates does not influence fascial structures, for clearly that would be unlikely. However, the precise ways in which the exercises, positions, repetitions and activities associated with Pilates (or yoga for that matter) involve fascial structures remain largely under-researched and speculative.

Because lack of proof of efficacy is not the same as proof of lack of efficacy, the methods and modalities discussed in this section have been included either because the proposed mechanisms, associated with the particular method under review, are *known* to have fascial effects, or because they are *proposed* by the users of the method to have such effects, and that this proposition has not, as of now, been shown to be unfounded.

Where evidence does exist of connective tissue/ fascial influences in relation to particular therapeutic methods, research evidence is presented and discussed, together with further reading suggestions.

Where no research evidence exists to support proposed mechanisms and/or purported fascial connections, the discussions of these are expressed as hypotheses, as though they *may* be accurate, but that this has not, at this time, been verified.

Old methods updated and new ones emerging

Some methods and modalities that have been in existence for many years, such as connective tissue massage (or CTM, now commonly termed connective tissue manipulation), have seen major growth

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of interest and application. A greater understanding of the potential for reflex effects that may be obtained via active and sometimes marked stimulation of fascial structures, as well as the benefits to be noted in terms of functionality and mobility when dense areas of fascia are modified by these means, offers credence to some of the early theories associated with CTM. Prendergast and Rummer (Chapter 7.6) offer a comprehensive overview of modern CTM.

Much the same is true of Myofascial Release (MFR) techniques, which have evolved and in some cases been renamed (as has CTM, mentioned above) as a result of current research and investigation via dissection, as well as because of evidence emerging as a result of use of modern investigative tools such as real-time ultrasound and elastography. *Myofascial Induction* is the name that Andrzej Pilat, the author of Chapter 7.4, has suggested. In that chapter he has elegantly described the methods, as well as the known and hypothetical mechanisms associated with MFR, which appear to achieve its beneficial therapeutic effects via changes in the colloidal state of loose connective tissue, allowing it to modify in response to light sustained forces.

Other approaches have evolved directly from a greater understanding that has emerged from animal studies, as well as human dissection and clinical observation. These include the evolution of Fascial Manipulation[®] (FM[®]) in which the results of complex assessment and analysis of movement patterns lead to estimations as to the most likely fascial structures that require targeting for treatment. A plethora of clinical studies have helped to refine and confirm the underlying premise of FM[®]. In Chapter 7.7, Stecco and Stecco explain both the background and methodology associated with Fascial Manipulation[®].

Scars

Similarly, treatment of the connective tissue changes associated with scar tissue has attracted new attention, with a variety of modern assessment tools being used to measure such features as electromyographic (EMG) changes associated with scars, while realtime ultrasound (and EMG) evidence of changes following appropriate manual treatment methods has validated these approaches. Two chapters are devoted to this aspect of fascial involvement in pathophysiology and the treatment of the associated changes and symptoms. Valouchova and Lewit (Chapter 7.8) focus their attention on the painful repercussions from scars, sometimes at a distance, that may have been produced many years previously. For example, abdominal postsurgical scars are shown to be capable of contributing to the development of back pain. Their extremely gentle deactivation methods are comparable to, but different from, those described by Fourie (Chapter 7.17) in his focus on scar tissue commonly resulting from mastectomy.

Needling

Dommerholt (Chapter 7.2, dry needling), Wander and Weinshenck (Chapter 7.12, neural therapy), as well as Irnich and Fleckenstein (Chapter 7.9, acupuncture), have all considered the rationale, and the methodology, of using different forms of needling to achieve therapeutic benefits.

Dry needling focuses much of its attention on myofascial trigger points, while the focus of acupuncture is more reflexive, building on its long historical tradition, as well as on recent and evolving concepts of fascial communication pathways.

Neural therapy, unlike dry needling or acupuncture, incorporates introduction of substances (such as procaine) in order to modify focal areas of irritation commonly located in fascial structures.

In Chapter 7.11, Cusi describes prolotherapy, in which attempts are made by means of the injection of irritants into connective tissue, to encourage proliferation in areas of, for example, ligamentous instability.

Broad influences on connective tissues

The important area of nutritional considerations and fascia is comprehensively described, particularly in relation to inflammatory processes, by Hankinson and Hankinson (Chapter 7.23), while the wider effects of heat on connective tissue are considered by Klingler (Chapter 7.18), as are the more subtle therapeutic influences of microcurrent as described by McMakin (Chapter 7.16).

Tool assisted fascial approaches

Externally applied mechanical force is associated with several fascia-directed therapeutic modalities.

In Chapter 7.10, the potential benefits, cautions, and conceptual mechanisms associated with

traditional (East-Asian) methods, known as Gua sha, involving superficial unidirectional scraping techniques, focused on superficial and loose connective tissues, are described by Nielsen. A number of hypothetical fascia-related explanations are offered for the well-established clinical benefits.

A modern evolution of the Gua sha approach is the Graston technique, and this is fully described in Chapter 7.14.

Comeaux (Chapter 7.13) describes a further mechanical approach, in which rhythmic vibratory or percussive forces are applied, with – it is theorized – a variety of beneficial connective tissue effects.

Neural mobilization

Coppieters (Chapter 7.19) notes that nerves have a large component of connective tissue as part of their make-up, as well as being potentially influenced by the mechanical interfaces of all tissues through, and past, which they travel, and which they supply, including fascia/connective tissue. In Chapter 7.19, these features are discussed as possible factors in the evolution of neuropathic pain, as well as therapeutic options being outlined (neurodynamics).

Whole-body exercise/ movement systems

Yoga and Pilates both involve whole-body movement patterns and exercises.

The connective tissue/fascial links with yoga are discussed by Myers (Chapter 7.21). Along with other possible fascial links, many of the standard yoga postures (asanas) are shown to involve extensive myofascial kinetic chains.

In Pilates methodology, similar chains of myofascial influence can be seen to be operating, potentially influenced by the exercises associated with this systematized approach (Blom, Chapter 7.22). In Chapter 7.24, Mueller and Schleip describe the evolving understanding of the effects of movement/ exercise on fascia.

Whole-body manual systems

Rolfing (Postural Integration) has a long tradition of focus on fascia, and this is outlined by Caspari and Massa in Chapter 7.3. Rolfing (Postural Integration)

can be considered to have been largely instrumental in the new-found clinical and research interest in fascia's role in the general economy of the body, and its potential role in pathophysiology.

Similarly, osteopathic medicine has a tradition of interest in, and treatment of, fascia going back to its inception in the late nineteenth century. In Chapter 7.5, King offers detail of both historical and current osteopathic thinking in relation to fascia. Ongoing research into modeled osteopathic methods such as myofascial release, and strain–counterstrain has shown rapid restoration of distressed fibroblast cohesion in research by Standley and Meltzer (2008), elaborated on in Chapter 7.5.

Also emerging from osteopathic medicine is a therapeutic approach known as the *Typaldos Fascial Distortion Model*, which is described by Harrer in Chapter 7.15.

Stretching

In Chapter 7.20, Myers and Frederick investigate the effects of a variety of stretching approaches on fascia. Stretching is incorporated into a wide range of manual therapy and rehabilitation/prevention approaches, including many covered in other chapters, such as yoga, Pilates, myofascial release, osteopathic treatment, Rolfing/structural integration, as well as various forms of massage. This chapter (7.20) is arguably the one that offers the greatest degree of cross-over evidence, thus helping to suggest explanations, at least in part, for the effects of a various manual and exercise modalities and systems where stretching is an integral component.

Conclusion

The topics in this section are not definitive of all the therapeutic methods used in manual therapy, but they are representative. Dozens of others could have been included, with massage being a primary contender for inclusion. Because massage incorporates many of the elements discussed individually in different chapters in this section, and because of the paucity of research into specific massage–fascia effects, it was felt that a separate chapter would be redundant. What should become clear is that any modality that incorporates application of pressure, shear forces, rhythmic movement and stretching is working with and on fascial structures, whether the therapist is aware of this or not.

Intelligent use of manual clinical methods involving fascial structures, as evidenced in many examples

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in this section, is clearly more desirable than random, virtually accidental influence.

The drive towards a better understanding of what *affects* fascia, and what *effects* fascial treatment can have, is therefore work in progress.

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Trigger point therapy

Jan Dommerholt

Introduction

Trigger point therapy has been practiced for several centuries, but it did not really enter into the realm of clinicians and researchers until Travell identified typical referred pain patterns associated with trigger points, developed a structured approach for their management, and conceptualized myofascial pain as a separate diagnosis. A trigger point is defined as 'a hyperirritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band' (Simons et al. 1999). By definition, trigger points are located within a taut band of contractured muscle fibers and palpating for trigger points starts with identifying this taut band by palpating perpendicular to the fiber direction. Usually, trigger points develop as a result of local muscle overuse and are frequently associated with other dysfunctions, such as pain diagnoses with peripheral and central sensitization, joint dysfunction, dental or otolaryngic diagnoses, visceral and pelvic diseases and dysfunctions, tension-type headaches and migraines, hypothyroidism, systemic lupus erythematosus, infectious diseases, parasitic diseases, systemic side effects of medications, and metabolic or nutritional deficiencies or insufficiencies. Trigger points have been reported in all age groups, except infants (Dommerholt et al. 2006a).

Principles of trigger point therapy

Any therapeutic intervention should be evidenceinformed and based on scientific evidence, clinicians' judgments, expertise, and clinical decision making. The current thinking about trigger points is best captured by the evidence-informed integrated trigger point hypothesis (Stecco et al. 2009). Although the model is not a perfect theoretical concept, the hypothesis along with several recent modifications is the most comprehensive framework currently available to explain the role of muscle tissue in acute and more persistent pain conditions, and to guide therapeutic management (Gerwin et al. 2004, McPartland, 2004, Dommerholt et al. 2006a, McPartland and Simons, 2006). Trigger points are associated with dysfunctional motor endplates with an excess release of acetylcholine attributed to a variety of possible reasons, including an insufficiency of acetylcholinesterase, an increased sensitivity of the nicotinic acetylcholine receptors, an acidic pH, hypoxia, a lack of adenosine triphosphate, certain genetic mutations, certain drugs and particular chemicals, such as calcitonin gene-related peptide, di-isopropylfluorophosphate, or organophosphate pesticides (Gröbli & Dommerholt 1997; Müller & Stratz 2004). Numerous studies have supported the hypothesis in rabbit, human and equine models (for example Dommerholt et al. 2006a).

Physicians, physical therapists, chiropractors, bodyworkers, and other clinicians should work to decrease trigger point pain and reverse the observed hypoxia and low pH by releasing trigger points and normalizing tissue extensibility. Therapeutic interventions may include manual and modality-based approaches and techniques. Different compression techniques appear to have similar efficacy (Fernández-De-Las-Peñas et al. 2005; Gemmell et al. 2008). Trigger points can also be treated with invasive techniques, such as trigger point dry needling or injections with anesthetics, botulinum toxin, or serotonin antagonists (Dommerholt et al. 2006b).

Therapeutic interventions should target not only the dysfunctions, but also the contributing factors. For example, smokers should be encouraged to quit sensitizing nicotinic acetylcholine receptors. Patients should avoid exposure to sensitizing chemicals. Mechanical problems need to be corrected, including forward head postures, poorly designed ergonomic workstations, or significant leg length discrepancies, excessive pronation of the foot and ankle, scoliotic postures, among others. Pharmacological management is important to suppress and alter central sensitization.

Trigger point therapy

Noninvasive trigger point therapy

Manual approaches

Two recent review articles explored the efficacy of manual trigger point therapies, which may include ischemic compression, trigger point compression combined with active contractions of the involved muscle, myofascial release techniques, postisometric relaxation, connective tissue and fascial stretches, massage therapy, spray and stretch (Fig. 7.2.1), muscle energy techniques, neuromuscular therapy, other soft tissue mobilization techniques, such as skin rolling (see Figs 7.2.2, 7.2.3) and strain-counter strain, among others (Fernández-De-Las-Peñas et al. 2005; Rickards 2006). It is difficult to evaluate which manual approaches are the most effective, since in reality most clinicians use a multimodal approach (Chaitow & Delany 2003). Nevertheless, several studies demonstrated an increase in pain pressure threshold, a decrease in pain ratings on visual analog scales, or an



Fig. 7.2.1 • Ice and Stretch Technique. Reproduced with permission of Myopain Seminars 2010.



Fig. 7.2.2 • Soft Tissue Mobilization. Reproduced with permission of Myopain Seminars 2010.

improvement in objective tests, such as the Oswestry Disability Index. Massage therapy was more effective than spray and stretch in one study, but another study showed that spray and stretch increased the pain pressure threshold and was even more effective when combined with deep pressure massage. Spray and stretch was a hallmark of the approach used by Travell, who learned about the technique from Dr. Hans Kraus. The spray and stretch technique involves a vapo-coolant such as ethylchloride or fluorimethane contacting the skin over the muscle and into the referral zone of a trigger point. The spray is passed along the skin in sweeps several times while the muscle is stretched. This is repeated several times, with increased stretch on the muscle. Apparently, the skin cooling acts as a distraction for the stretch, which seems to facilitate inactivation of trigger points. Unfortunately, fluorimethane has a detrimental effect on the ozone layer and is rarely used anymore. A new 'spray and stretch' product was introduced a few years ago, but this product contains



Fig. 7.2.3 • Skin Rolling Mobilization. Reproduced with permission of Myopain Seminars 2010.

hydrofluorocarbons, which are powerful greenhouse gases classified as volatile organic compounds with a carbon dioxide equivalent of 1300, which means that the product has a 1300 times greater greenhouse effect than carbon dioxide (Dommerholt et al. 2006b; Dommerholt and McEvoy, in press). During the 1980s, Swiss physician Dejung developed a comprehensive treatment strategy which combined several muscle and fascia techniques, but the effectiveness of this combination of techniques has not been shown in a double-blind randomized controlled study (Gröbli & Dommerholt 1997). Generally speaking, there is some evidence of the short-term effectiveness of manual therapies, but there are few, if any, studies that demonstrate medium or longterm effectiveness beyond placebo (Rickards 2006).

Modality-based approaches

Modalities used in the treatment of myofascial trigger points include laser, ultrasound (US), and electrotherapy, among others. Modalities are rarely used in isolation, and are almost always a small part of the overall management approach. There is moderate evidence for the short-term effects of laser (light amplification by stimulated emission of radiation), which consists of a narrow monochromatic beam of photons of identical frequency used for a variety of ailments. There are several types of laser, such as gallium arsenide (Ga-As), helium-neon (He-Ne), and infrared diode laser (IR). The medium and long-term outcomes of laser need to be determined as well as the most appropriate and effective laser treatment parameters.

Ultrasound is a commonly used modality in chiropractic and physical therapy even though its efficacy is generally considered to be poor. As a trigger point therapy, ultrasound improved pain intensity, pressure threshold, and cervical range of motion in one study, but failed to have any effect in other studies. Researchers in Turkey recommended using a highpower pain threshold static ultrasound technique with continuous US, ramped gradually to the patient's maximum pain level, held for 4 to 5 seconds and reduced by 50% for 15 seconds, which resolved active trigger points more rapidly than conventional applications of ultrasound (Majlesi & Unalan 2004). Standard ultrasound applications do have a temporary antinociceptive effect on trigger points, but not sufficient to justify including conventional ultrasound in clinical practice (Dommerholt and McEvoy, in press).

Transcutaneous electrical nerve stimulation, or TENS, is the most researched electro-modality in the treatment of trigger points and there is good evidence of its short-term effectiveness. Interestingly, TENS did decrease overall myofascial pain, but did not reduce the actual trigger point sensitivity in several studies (Rickards 2006). There is no scientific evidence of medium or long-term effects of TENS. In a series of low-quality retrospective studies and case reports of frequency-specific electrotherapy in patients with myofascial pain and fibromyalgia, McMakin suggested that this form of electrotherapy is more effective than other electrotherapies, but there is no scientific evidence of the claims, even though in one of the articles she does report having observed a decrease in the concentrations of cytokines following the electrotherapy (McMakin et al. 2005).

Invasive trigger point therapy

Many scientific studies offer support for trigger point injections (Dommerholt & Gerwin 2010). Travell recommended injecting procaine hydrochloride to

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treat patients with myofascial trigger points. As procaine is not always available, the current best evidence suggests that a 0.25% lidocaine solution is the most effective anesthetic, even though many researchers use much stronger solutions of 1-2% lidocaine. Clinicians are injecting many other anesthetics, including mepivacaine, bupivacaine, levobupivacaine, and ropivacaine, with varying, but generally good, results. Some physicians inject trigger points with vitamin B_{12} in spite of a lack of scientific evidence. Patients with widespread pain may have vitamin B₁₂ deficiencies or insufficiencies and may require systemic vitamin B₁₂ supplementation, but there is no evidence that injecting myofascial TrPs with vitamin B_{12} is beneficial. Others suggest using nonsteroidal antiinflammatories or steroids, but again, there is no evidence of efficacy either, and intramuscular injections of steroids may actually lead to tissue breakdown and degeneration (Dommerholt & Gerwin 2010).

There are theoretical considerations which offer some support for injections of bee venom. Bee venom can activate catecholaminergic neurons, alpha-2 adrenergic and serotonergic pathways of the descending inhibitory system, and sympathetic preganglionic neurons, which may trigger an increased release of spinal acetylcholine. Active ingredients of bee venom include melitin, which has been shown to suppress lipopolysaccharide-induced nitric oxide and the transcription of cyclooxygenase-2 genes and pro-inflammatory cytokines, including interleukin-1b and tumor necrosis factor alpha in microglia. Bee venom also contains several other peptides, including apamin, adolapin, mast cell degranulating peptide, enzymes, and biologically active amines such as histamine and epinephrine, which may, at some level, contribute to the mechanism of pain relief following injections of bee venom, possibly by inhibiting c-Fos expression in the spinal cord (Dommerholt & Gerwin 2010). German researchers established that injections with the serotonin antagonist tropisetron were superior to injections with lidocaine (Müller & Stratz 2004). Other injectates used to treat patients with myofascial pain include diclofenac and botulinum toxin, for which there is growing, but at times conflicting, evidence.

Although the effect of trigger point injections was attributed to the mechanical effect of the needle as early as 1944, it took over three decades before needling with solid-filament needles was investigated. Recent studies have demonstrated that so-called dry needling (without an injectate) or intramuscular manual therapy is equally as effective as



Fig.7.2.4 • Intramuscular Manual Therapy (aka Dry Needling). Reproduced with permission of Myopain Seminars 2010.

injections with anesthetics (Dommerholt et al. 2006b; Dommerholt & Gerwin 2010). Dry needling (see Fig 7.2.4) is practiced mostly by physical therapists, but is also in the scope of practice of physicians, chiropractors in some jurisdictions, such as the US State of Maryland, acupuncturists, dentists, and even veterinarians. There are many studies of dry needling, supporting its efficacy, including a Cochrane Review (Furlan et al. 2005; Dommerholt et al. 2006b).

Fascia and trigger points

Traditionally, trigger point therapy has focused primarily on lengthening and resolving localized contracture knots in taut bands of muscle tissue, improving the local circulation, and addressing perpetuating factors. In spite of the term 'myofascial', fascial connections with muscles and the continuity of fascial structures are not commonly considered (Stecco et al. 2009). The two-volume Trigger Point Manual, which has become the standard reference work for trigger point therapy, does not include much information about the role of fascia in myofascial pain (Travell & Simons 1992; Simons et al. 1999).

Based on recent studies demonstrating that fascia is integrally linked to muscles, it would seem reasonable to explore whether specific treatments of fascia would have an impact on myofascial trigger points. For example, knowing that all muscles are enveloped by their epimysium, muscle fiber bundles by perimysium, and the deeper fibers by endomysium, can it be extrapolated the local myofascial stretches in the direct vicinity of trigger points would lengthen both fascial and muscle tissues? Perimysium is capable
of increasing muscle stiffness, which is a common finding in myofascial pain, and seems to adapt more to changes in mechanical tension than other intramuscular connective tissues, although there are direct connections with the epimysium and muscle fibers (Passerieux et al. 2007). It is conceivable that invasive trigger point procedures may change the viscoelastic properties or behavior of fascia. If that is confirmed, fascial manipulation techniques should probably play a greater role in trigger point therapy, as has already been suggested by Stecco and others (Gröbli & Dommerholt 1997; Stecco 2004).

Many questions remain. For example, are there any relationships between trigger points, perimysium, and myofibroblasts? Perimysium is characterized by a high density of myofibroblasts (Schleip et al. 2006b), which suggests that fascia may play a significant role in muscle contractibility and possibly in the formation of myofascial trigger points (Schleip et al. 2005; Schleip et al. 2006b). If direct connections are present, would that suggest that myofascial trigger points are more prevalent in tonic muscles, since they contain more perimysium than phasic muscles (Schleip et al. 2006b)? The role of fascia in the formation and maintenance of taut bands is also not clear. Taut bands feel like ropy or stringy bands of contractured fibers and have been visualized with elastography using phase-contrast analysis of vibration-induced cyclic shear waves, but the resolution of the currently available elastography techniques is insufficient to analyze the contributions of fascia (Chen et al. 2008; Sikdar et al. 2009).

Could the instantaneous reduction of local and referred pain following dry needling or trigger point injections be related to stimulation of fibroblasts? Langevin and colleagues have shown that the effects of acupuncture needling can at least partially be explained by stimulation of fibroblasts (Langevin et al. 2001; Langevin et al. 2006). Myofibroblasts are commonly found in lumbar fascia and appear to be related to physical activity (Schleip et al. 2008). Does stimulation of trigger points involve myofibroblasts and result in similar mechanical signaling and a reduction in nociception?

Fascia and muscles are not linked solely from a biomechanical perspective. Taguchi and colleagues demonstrated that lumbar dorsal horn neurons receive input not only from muscles but also from the thoracolumbar fascia (Taguchi et al. 2008). Several researchers have confirmed the presence of free and encapsulated nerve endings in fascia, including Ruffini and Pacini corpuscles (Yahia et al. 1993: Stecco et al. 2008). What is the possible relationship between trigger points, trigger point therapy and these free and encapsulated nerve endings? Although the effects of manual therapy interventions are difficult to measure (Bialosky et al. 2009), there is some evidence that Pacinian receptors may be involved in high-velocity manipulation (Schleip 2003). Do trigger point dry needling or manual trigger point techniques specifically stimulate Pacinian receptors, and could this offer insights as to why trigger point dry needling frequently results in an immediate reduction of pain? Along these lines of inquiry, what is the role of fascial structures, especially the perimysium, in creating local twitch responses? Local twitch responses are involuntary spinal cord reflexes which are considered to be important in invasive trigger point therapies (Dommerholt et al. 2006b).

Summary and Conclusions

It is obvious that trigger point research needs to explore the role of fascia in the etiology, pathophysiology, and management of trigger points. While several noninvasive and invasive treatment options are currently available, with reasonable to good efficacy, there are still many unanswered questions. With increasing technological advances, it may soon be possible to actually visualize trigger points and their immediate environment.

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Rolfing structural integration

Monica Caspari Heidi Massa

Premises of the work

Rolfing Structural Integration¹, developed by Ida P. Rolf, PhD, organizes the human being in gravity. It enhances structural and functional integrity, as revealed by proper alignment and coordination. Two foundational premises distinguish Rolfing from other somatic practices: first, that physical balance, fluidity, ease and grace, and indeed personal wellbeing, all require appropriate adaptation to the field of gravity; and second, that the fundamental organ of structure is fascia.

Rolfers approach misalignment and chronic musculoskeletal complaints from the perspective that symptoms manifest a more generalized dysfunction; and that if posture and movement quality improve, complaints are likely to resolve spontaneously.² Rolfers therefore address the whole person, rather than the person's presenting complaints.

Key characteristics of fascia for Rolfing Structural Integration

Rolfing works because fascia is:

- physically and functionally continuous
- malleable due to its viscoelasticity
- able to register information and transmit it throughout the body, and
- responsive to gravity.

Fascia forms a continuous web throughout the whole body, surrounding all muscles, bones, nerves and organs. Because the fascia associated with any body structure typically lacks an anatomical name, we use the name of the structure itself to refer to its associated fascia. Through the fascia, the entire body is affected somewhat by any local change. This fascial continuity allows Rolfers to facilitate change in tissues distant from the contact point – even those that cannot be touched directly. To influence the body's many layers and planes of fascia, the distinctly vectored touch of Rolfing is highly educated and versatile.

The fascial web constantly changes shape, chemical composition, and physical properties to adapt to mechanical and other stresses. Recognizing fascia's capacity to self-correct over time, Rolfers facilitate limited positional and functional changes to which the body can adapt, allowing adequate time between interventions for the adaptations to occur. After each adaptation, new changes become possible.

The fascial web, dense with various mechanoreceptors, is a body-wide mechanosensory organ

¹ Dr. Rolf's work is now known by her original and preferred term, *Structural Integration. Rolfing* Structural Integration refers to the work of graduates and members of the Rolf Institute of Structural Integration, Dr. Rolf's original school. Though the many schools now teaching structural integration have much in common, this chapter speaks to Rolfing Structural Integration as it is taught by the Rolf Institute and practiced by its certificants. The Little Boy logo (Fig. 7.3.1) and the terms *Rolfing* and *Rolf Movement* are service marks of the Rolf Institute, registered in the US and other countries. The term *Rolfer* is a service mark of the Rolf Institute.

 $^{^2}$ For a collection of literature on how Rolfing affects conditions such as anxiety, cerebral palsy, and chronic pain and fatigue, see chapter bibliography.

(Schleip 2003) telling us where we are in space and what our bodies are doing. As information is collected and carried through the fascial web (Langevin 2006), the mechanoreceptors communicate with a self-regulating aspect of the neuromotor system.

Finally, fascia responds to gravity – the force to which we are all continuously subject. As a ship's sail needs wind to function, fascia needs gravity. In a sense, gravity is a fixed vector against which fascia organizes bodily structure and function.

Facilitating integrated structure and function

Much of the Rolfer's work is to balance opposing lengths and tensions within the fascial net. Structurally, Rolfers look for a *palintonic* quality of posture (relative segmental arrangement). The Greek palintonos refers to a dialog between opposites within an orthogonal order, which order is manifest in the relationships among structures, spatial dimensions, volumes and planes. An imaginary plumb line through the center of the body expresses occupation and use of space in the sagittal, frontal and horizontal planes (see Fig. 7.3.1). Posture both potentiates and limits movement options. Functionally, Rolfers assess movement for ease, fluidity, and contralateral movement in the limbs, shoulder and pelvic girdles, and spine. In general, as palintonic right angles are established in the structure, the diagonals of contralaterality emerge in function.

Before we can move, we perceive and orient to directions in space. The Rolfer's work reaches beyond fascial patterns to patterns of sensory perception (touch, vision, hearing, and proprioception) and neuromotor coordination (balance between tonic and phasic muscles, and between local and global body stabilizers), as dysfunction in either realm limits structural and functional order.

Finally, because Rolfers understand that both structure and function are in some sense relational attitudes based on all aspects of experience, they consider not only the client's perceptions of the social and physical environment, but also awareness of and attribution of meaning to them.

This chapter uses the traditional 10-session series to illustrate how Rolfers take advantage of fascia's key characteristics. The discussion of each session is descriptive only, and far from comprehensive as to elements or processes.



Fig. 7.3.1 • The Rolf Institute's logo illustrates progress to orthogonal order in three dimensions. The Little Boy Logo is a service mark of the Rolf Institute, registered in the US and other countries. Reprinted by permission of the Rolf Institute.

The traditional Rolfing Structural Integration series

Dr. Rolf's 10-session protocol is both a teaching tool and a basic strategy to deliver the work, though in practice the number and content of sessions are influenced by the client's needs and the practitioner's expertise.

The protocol builds on key characteristics of fascia as an organ of structure and communication, its logic producing an orderly and well-supported arrangement of body segments in 3-dimensional space along the imaginary plumb line. Because fascia wraps body parts in layers of varying depths, the protocol starts from the outer layers, first working inward – and then back outward. Since fascia is malleable according to changes in functional demands and over time, the protocol induces changes in the right order – i.e., in an order such that the fascia can adapt and integrate them. Because the density of mechanoreceptors and other characteristics of healthy fascia make it an information highway (especially for information related to the gravity response), the protocol addresses early on the fascia of key orienting areas such as the feet and the occiput. Finally, because fascia responds to gravity, the protocol starts from the ground and works up – and then back down – building from an adequate foundation a balanced structure free of torsions caused by cantilevers.

The logic of the protocol is functional, too; e.g., we begin by freeing the breath, progress through finding the ground, and conclude by integrating the person in his or her surroundings. The sequence facilitates progressive levels of movement integration, manifesting as enhanced contralaterality.

Session 1: Open the superficial fascia

We begin by opening the superficial fascia, giving particular attention to its attachments at bony margins (e.g., the iliac crest and scapular spine) and to regions where it limits the position of major bony segments (e.g., superficial rib fascia and femoral head fascia). This is essentially preparatory, as restriction in this outermost layer limits changes in layers below.

Structurally, we differentiate thorax from shoulder girdle, thorax from pelvis, and pelvis from legs. This differentiation is a precondition for a balanced and palintonic arrangement among these major segments. For example, as femoral rotation approaches neutral, the pelvis has greater independence from the legs and can find better balance over the feet. This, in turn, allows the pelvis to provide greater support for the thorax.

Functionally, Session 1 frees the breath. The ontogenic logic is evident: a newborn's first act is a deep breath. Improving the pelvis as a base of support enables adaptable sagittal plane movement of the upper body's center of gravity (at the approximate level of T4), which Rolfers call G-prime (G').

Session 2: Establish a base of support

The upright body needs sound and adaptable feet. Loaded with mechanoreceptors, the feet gather information for the entire body to maintain balance. As the fascial system as a whole responds to gravity, better feet allow greater ease throughout the body. Session 2 differentiates and makes adaptable the myofascia and bones of the feet and lower legs, and begins to release fascial restrictions of posterior structures such as hamstrings and spinal erectors.

Structurally, Session 2 balances the feet from front to back, and from the lateral arch to the medial arch (through the transverse arch); restores resilience to the interosseus membrane of the lower leg; and organizes the medial, lateral and posterior compartments of the lower leg. This brings greater order to G (the lower body's center of gravity, located at approximately L4) by giving the lower body a better place to rest its weight.

Functionally, it decouples the foot's intrinsic muscles from the extrinsic muscles crossing the ankle, allowing the toes movement independent of the ankles and improving the propulsion phase of the gait. Stimulation of the intrinsic muscles improves contact with the ground and introduces movement in the frontal plane by restoring the interplay between the cuboid and navicular bones. This enhances the ability of the feet (in conjunction with the eyes, inner ear, and temporomandibular joint) (Bricot 2001) to maintain dynamic equilibrium.

Work on the superficial spinal erectors begins the task of optimizing the transition point for contralateral spinal movement, ideally located between T8 and T10. A higher transition produces a long or exaggerated lordosis, which dissipates the impulse coming from the legs at the level of the abdomen. This manifests as excessive motion in the pelvic girdle and legs relative to that of the shoulder girdle and arms. Conversely, a transition below T8/T10 produces relatively flat lumbars and a long or exaggerated kyphosis. This configuration cannot efficiently transform the impulse from the legs into contralateral movement at the axial level, and the shoulder girdle and arms will compensate with excessive motion relative to that of the pelvic girdle and legs.

Session 3: Balance fascial span along the lateral line

Session 3 builds on the space, adaptability and support already achieved to address the relative positions of G' and G along the vertical lateral line of the body. If either G' or G is displaced forward or backward of the lateral line – i.e., if the lateral line between G' and G deviates from the vertical – the thoracic and abdominal volumes are distorted



Fig. 7.3.2 • These illustrations are based on photos which show a client before (A) and after (B) ten sessions. Note the normalization of thoracic and abdominal volumes.

(see, e.g., Fig. 7.3.2) Posture is in one sense an accumulation of relational attitudes, and habitual G'/G positions relative to the lateral line are their physical manifestations. There is no universal correlation among relative segmental positions and particular emotional states, but we know how our own bodies react to situations with small forward or backward displacements of either the chest or the pelvis.

This session addresses (a) the fascia of the arms and shoulder girdle, which can restrict the position and movements of G', and (b) the more superficial structures that influence the tilt of the pelvis over the femoral heads and permit forward flexion of the torso over the hip joints. Finally, quadratus lumborum and the abdominal flexors – the bridges between the thorax and the pelvis – are balanced to allow differentiation of and movement between these body segments.

Structurally, Session 3 evokes a palintonic arrangement of major body segments along a lateral line from the joints of Chopart to the glenohumeral joints. Easing restrictions between the thorax and the pelvis permits these segments to assume sagittal plane positions approximating horizontal.

Functionally, it frees the hip hinge to allow prevertebral length in activities that involve reaching. The balance of front-to-back spans from pelvis to ribs – and the horizontals it reveals – precedes diagonal (i.e., contralateral) limb movement, and encourages contralateral motion within the spine itself.

Session 4: Balance the spans of the inner and outer legs

Session 4 is the first of three sessions on the leg-topelvis relationship, and also the bridge from the lower limbs to the normally inaccessible prevertebral space. Though the territory of contact is the midline of the legs, fascial continuities among the adductors, pelvic floor tissues, and sacrolumbar prevertebral space, the adductor work affects structures we cannot touch directly.

Imbalances between the femoral adductors and abductors cause medial or lateral femoral rotation, either of which restricts hip flexion and affects the fascial organization and function of the pelvic floor, sacroiliac joints, and psoas. Session 4 frees the medial line by differentiating the adductors from the adjacent quadriceps (anterior) and medial hamstrings (posterior). Thanks to fascial continuity, as the adductors become better organized, the pelvic floor also becomes better organized and the fascial connection from the inner legs to the front of the spine becomes evident.

Structurally, this advances the work of Session 2 to equalize span and create dynamic balance between the inner and outer lines of the legs and feet. It carries the work from the feet to the front of the lumbar spine.

Functionally, Session 4 connects the feet to the spine through the fascia of the medial line, which indirectly affects the spinal transition point for contralaterality (see Session 2 discussion). Typically, both the Rolfer and client observe the legs seeming to reach into the abdomen, yielding a longer stride as the body organizes toward its midline and upward. Clients report a sense of greater volume and awareness to the prevertebral abdominal space.

Session 5: Connect the legs to the front of the spine

When span and tonus are balanced across large joints like the hip, gravity works through the joints. Session 5 focuses on structures crossing the hip hinge

Rolfing structural integration

anteriorly – the quadriceps and iliopsoas. The territory includes the fascia of the abdominal wall, which enhances continuity of the legs with the lumbar spine via the iliopsoas. As much of this territory cannot be touched directly, we affect it through fascial continuity.

Structurally, the goal is enough space and length on the anterior thigh and in the prevertebral region for full leg extension as well as accommodation of the organs that occupy the pelvic/thoracic visceral prevertebral column (Schwind 2006). When the fascia has sufficient length and the deep flexors move freely within the abdominal cavity, the bony pelvis will rebalance over the feet, reducing dysfunctional pelvic tilt and shift patterns (Fig. 7.3.3).

Functionally, only when the leg fully extends can the feet (especially the toes) truly propel the body forward and extend the spine. The psoas (running from the lesser trochanter to the bodies of the lumbar vertebrae) connects the legs to the spine without interference from the pelvic structures and can stabilize the head of the femur to allow contralateral leg motion and the emergence of the functional core. Functional differentiation of the core transversus abdominus from the superficial rectus abdominus is essential. In walking, gravity appears to take the leg from the front of the lumbar spine before the leg swinging smoothly forward.

Session 6: Establish posterior length, continuity and order

Session 6 addresses the back of the body, which is unified by a continuous fascial span from the soles of the feet all the way through the galea aponeurotica.

Fig. 7.3.3 • Correct alignment (A), and common postural malalignments (B through H) arising from various pelvic tilt and shift patterns caused by fascial restrictions.

This continuity allows local work to influence the entire posterior surface. Here Rolfers address deeper muscle chains, e.g., biceps femoris/transversus abdominus/multifidus; and diagonal spans united by the lumbar fascia, e.g., latissimus dorsi/gluteus maximus. At this point, Rolfers address spinal and sacroiliac torsions and counter-torsions.

Structurally, Rolfers address pelvic and spinal rotations by rebalancing spans of fascia that influence bone position. Differentiation of myofascial layers encourages discriminated function, and differentiation of the posterior leg structures enhances leg extension.

Functionally, Session 6 improves from the back the legs-to-spine connection crucial for the emergence of the functional core through the sensing and communicating tendencies of fascia: Weight or pressure triggers the mechanosensors embedded in the plantar fascia, activating the deep biceps femoris/transversus abdominus/multifidus chain, which stabilizes the lumbar spine as a fixed point for the iliopsoas. Improved contralateral movement within the spine augments contralateral coordination between the pelvic and shoulder girdles. Finally, all vertebrae should be capable of extension, despite the tendency toward flexion throughout the entire human life cycle.

Session 7: Organize the upper pole

Posture potentiates movement; but preceding any movement is perception of and orientation to the ground through gravity, three-dimensional space, and finally objects and others. Several key components



of the perceiving and orienting system – the suboccipital muscles, vestibular system, vision, hearing, and the temporomandibular joint – are in the head and neck. To paraphrase Dr. Rolf, when we do neck work, our fingers are as close as they'll ever get to the body's control structures.

As neck alignment influences occiput position, Rolfers treat the neck and head together. Hypertonic sternocleidomastoids betray absence of core support for the neck, interfere with the deeper scalenes' fine motor activity, and actually reduce visual and auditory fields. We enhance cervical mobility, especially in extension, because an erect neck requires a balanced relationship among trapezius, SCM, splenius capitus, and longus capitus. Finally, the normal position of the head on the neck requires balanced tonus of the masseter and the supra- and infrahyoid muscles.

Structurally, Session 7 improves conditions for action of the suboccipitals: though they have many times more stretch receptors than any other muscles, their receptors do not activate an internal stretch reflex; instead, they inform the tonic function of the entire body. Optimizing suboccipital function thus touches the whole through the fascial system.

Functionally, because balanced scalene activity is key to free head and neck orientation, Rolfers evoke the function of the prevertebral longus colli, which supports the neck and forms a core-stabilizing gestalt with transversus abdominus, multifidus and iliopsoas. Working together, the scalenes and longus colli decompress the cervicals and allow deep breathing; working alone, scalenes compress the cervicals into a hyperlordotic position and encourage shallow breathing. Because Session 7 is intimately involved with the senses, the work is more efficient when combined with education in perception and coordination.

Sessions 8 through 10: Integrate the girdles within the person, and the person within the environment

Dr. Rolf observed, "Anybody can take a body apart...but only a few can put one together." (Rolf 1977) Putting together is what happens in Sessions 8 through 10. While differentiating myofascial structures in Sessions 1 through 7, the intent is largely *within* the body, which responds through better alignment in gravity; support is essentially static, coming mainly from the ground. By contrast, the final sessions allow the body to relate better to gravity not only intrinsically, but also within the environment and relative to others. The intent is beyond the body, with dynamic support coming from the hands' relating into space.

The more differentiated body parts are, the more easily joints work. Yet, for the parts to act like an orchestra, we need integration as well. Sessions 8 and 9 each seek to improve the least integrated body area – often the pelvic girdle and legs in Session 8, followed by the shoulder girdle and arms in Session 9. One session accesses the *down* direction, mass and gravity; and the other the *up* direction, expansion and space.

The idea is to establish palintonic right angles in both the fascial and spatial planes. As the Rolfer positions the fascia, the client moves to facilitate orderly transmission of motion through the joint and associated tissues. This capitalizes on fascia's physiologic responses to changes in magnitude and direction of mechanical load. Because fascia is a body-wide mechanosensitive signaling network (Langevin 2006), Rolfers can facilitate contralateral movement at progressive levels of limbs, girdles, and spine.

While Sessions 8 and 9 address the pelvic and shoulder girdles to integrate the upper and lower bodies and improve how they relate as parts of a whole, Session 10's task is to facilitate integrated relationship of the person to objects and others in the environment. Its territory is the whole body. The typical pre-10 client has restrictions at the neck and ankles, which hearken to the initial functional issues of orientation to space and ground. This time, the Rolfer's work with the superficial fascia enhances the client's ability to be fully present to the outside while at the same time accessing support for the inside from the ground through gravity.

Through widely distributed interstitial mechanoreceptors, Rolfers adjust the superficial fascia to create clear horizontals at joints. This enhances contralateral movement. For Dr. Rolf, "[T]he job in the 10th hour is a relating job. It's a relating of planes of space, but it is also a relating of planes of fascia. Now you can't get those planes until you get a vertical. You can't get a vertical except as you approximate these planes" (Rolf).

As the Rolfer works, the client learns to interact more functionally with the outside. This requires release of fixations not only in the fascia, but in the patterns of perception, coordination, and meaning (psychology) that are often misperceived in terms of biomechanics. Clients who do not change these patterns tend, over time, to re-establish their original tissue fixations.

Integration is more about conclusion than perfection. Putting together relates imperfect segments to constitute the most functional whole currently possible. It brings closure to the process and sets the client free to integrate changes not only in fascia, but also in perception, coordination, and way of being in the world.

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Myofascial induction approaches

Andrzej Pilat

Introduction

Myofascial Induction Therapy (MIT) is a hands-on, full-body approach, focusing on restoration of altered fascial tissue function. During the application, the clinician stretches or compresses the specific region in order to transmit a low-intensity mechanical input. These actions modify fascial restrictions in order to adjust the tension distribution in the fascial network. It is hypothesized that this procedure can restore the ability to move more efficiently and to achieve better functionality with lower energy expenditure (Useros et al. 2008).

Fascial restriction is described as any impediment to optimal gliding, at both macroscopic and microscopic fascial organizational levels, between endofascial fibers and interfascial planes. Such restriction can cause anomalous tension and movement disorders (Fourie 2008).

One possible reason for a restriction of the fascial tissues may be excessive stimulation of collagen production inducing fibrosis, resulting in loss of its smoothness and/or isotropy, and creation of entrapment areas. This suggests that such entrapment areas may alter physiologic body movements in relation to amplitude, velocity, resistance, and coordination (Fourie 2008).

In the presence of long-term restrictions, fascial tissue becomes overloaded and suffers dysfunctional consequences. These changes first affect the loose connective tissues, followed by reorganization of regular or irregular dense connective tissue, such as tendons, ligaments, or capsules, creating excessive density and reorientation of fibers. Fascial restrictions of short durations affect the tissues locally, whereas restrictions of long duration induce a more global dysfunction pattern (Langevin 2006).

Fascial tissue is related to the interchange of body fluids and to mechanoreceptor coordination. A decrease of fascial mobility can alter the blood circulation and cause ischemia, deteriorating muscle fiber quality. And since many mechanoreceptors are embedded within fascia, altered proprioceptive afferents can change the ability of optimal muscles tonic contraction (Vaticon 2009). As a result, alterations in stabilizing functions, as well as in the coordination of joint movements, may occur, with consequent difficulty of joint compression at its optimal point of action, possibly leading to joint overload, inflammation and/or pain in myofascial structures (Lee 2001).

Some theories suggest that the three-dimensional fascial network can be involved in pain transmission, with peripheral pain possibly having an origin in the connective tissue (Liptan 2009; Han 2009). Taguchi et al. (2009) suggest that the thoracolumbar fascia is an important source of nociceptive input in chronic LBP patients.

Various concepts exist related to the manual treatment of fascial system restrictions, with different names used to describe similar treatment approaches (Chaitow & Delany 2002; Myers 2003). The conceptual bases of most of these are similar. Further clinical research is needed in order to unify and validate these clinical procedures (Remvig 2007).

Neurophysiologic mechanisms for releasing the restrictions of the fascial system

The application of MIT produces a mechanical stimulus in the connective tissue. The effect may occur at micro- or macroscopic levels of fascial organization, and may include a group of cells, tissues, organs, or the whole body.

Anatomical analysis in unembalmed specimens confirms continuity of the fascial system through the entire body (Fig. 7.4.1). Benjamin (2009), Stecco et al. (2008), Pilat (2009), Pilat (2010), Myers (2003), van der Wal (2009), Mass and Sandercock (2010), all demonstrate movement continuity on macroscopic levels, focused not only on the fascia-to-bone connections, but also on the direct fascia-to-fascia transmission, in both articular and intermuscular connections (Fig. 7.4.2). Ingber, (Wang et al. 2009) and Langevin (2010), among others, have demonstrated dynamic continuity at the microscopic, intracellular and intercellular levels.

In 1997, Ingber proposed intercommunication systems based on tensegrity principles (Ingber 1997; Pilat & Testa 2009). This suggests a system of shared tensions in the distribution of mechanical forces, at multiple body levels, possibly explaining the global reaction of the fascial system in response to mechanical stimuli (Khalsa et al. 2000). Different studies (for example, Wang et al. 2009) have shown that cell dynamic and active responses of the cytoskeleton, responding to mechanical forces from the extracellular matrix, induce tissue remodeling at both cellular



Fig. 7.4.1 • Transverse section of forearm. Note fascial continuity from the skin up to the bone. A Skin; B 'Honey comb' fascia with the fat nodules; C Superficial fascia; D Deep fascia; E Intermuscular septa; F Bone.



Fig. 7.4.2 • Longitudinal fascial continuity. A Brachial fascia; B Antebrachial fascia; C Fascia-to-fascia connection between brachial and antebrachial deep fascia at the posterior aspect of upper limb; D Triceps brachii insertion (tendon-to-bone connection); E Superficial-to-deep fascia continuity.

and subcellular levels. Taking into account that the structure of the body follows the principles of hierarchical assembly, the above process is not limited to cells, but also involves tissues and organs (Huang & Ingber 2000). Ingber, (Wang et al. 2009) showed continuity of mechanical stimuli from the cytoskeleton to intranuclear level, and Langevin (2010) demonstrated mechanical impulse continuity from the skin to the nucleus membrane in mouse fibroblasts.

In conclusion, any action performed locally can potentially involve function throughout the body. The correction of myofascial dysfunction can take place in only one, or in different, system segments.

It is hypothesized that mechanical stimuli can create at least three types of reactions:

 Piezoelectricity: This is a phenomenon exhibited by certain crystals that, when subject to mechanical tensions, acquire a polarization in their atomic structure, generating a difference of electrical potential and loads at their surface (Pilat 2003). The basic properties of the organism (i.e., elasticity, flexibility, elongation, resistance) depend to a great extent upon the ability to maintain a continuous information flow. Oschman (2003) affirms that information is transmitted electrically through the connective tissue matrix. Since collagen may be interpreted as a semiconductor (Cope 1975), it may be capable of forming an integrated electronic network enabling the interconnection of all fascial system components (O'Connell 2003). Further investigation is needed to evaluate how MIT may influence this property of the body network.

- Dynamics of the Myofibroblasts Response: Muscles comprise contractile tissues that enable the body to move. Fascia should be considered as an intramuscular connective tissue that forms a functional unit with muscle fibers. The fascial system is highly innervated by mechanoreceptors (Stecco et al. 2008). Consequently, mechanical input (manual pressure or traction) received by mechanoreceptors may create a broad range of responses in the fascial system that facilitate movement. Studies focusing on skin healing processes and pathologies such as Dupuytren's contracture, plantar fasciitis, frozen shoulder, that relate to actin microfilament contraction, strongly support this reasoning (Gabbiani 2007). Chaudhry et al. (2008), using a 3-D mathematical model for deformation of human fascia, suggest that mechanical forces applied during manual techniques can create mechanical changes in loose connective tissue (i.e., superficial nasal fascia). Schleip (2005) suggests that changes in resting tone of skeletal muscle fibers can transmit their tension force to the fascial tissue.
- Viscoelasticity: The viscoelastic properties of fascia have been observed in numerous studies: thoracolumbar fascia (Yahia et al. 1993), fascia lata (Wright & Rennels 1964), subcutaneous fascia of rats (Latridies et al. 2003), the fascia lata, plantar fascia and nasal fascia (Chaudhry et al. 2008). Concepts for practical treatment applications have been defined by Rolf (1994), Threlkeld (1992), Barnes (1997), Cantu and Gordin (2001), Pilat (2003), and Pilat (2009).

Method description

As previously mentioned, MIT is an ensemble of techniques that allow, through the elimination of dysfunctions, restoration of optimal function and balance. Treatment of fascial system dysfunction focuses on the correction inside and within the system, at both microscopic and macroscopic levels. It is important to note that the approach seeks local corrections, but also focuses on the recovery of global dynamic body balance, and the relief of pain.

The applications of MIT suggested below are based on the clinical experience of the author (Pilat 2003), and are based on theoretical frameworks discussed above. MIT may be combined with other manual therapy strategies, or an exclusive treatment procedure.

General observations for clinical applications

- The evaluation of fascial dysfunction should be included into clinical reasoning processes. An exhaustive case intake is required, with annotations concerning the duration of symptoms and a visual analog scale (VAS) evaluation.
- We suggest automatic exploration of general posture disorders and local dysfunction testing.
- Biomechanically, the myofascial system responds to compression and traction forces (Chaudhry et al. 2008). These two mechanical strategies can be used when applying MIT.
- The direction of the releasing movement is towards restriction barriers. Arbitrary directions of tissue engagement should be avoided. Restrictions may occur in various directions and planes. They may also occur in different directions in the same plane, or in the same direction in various planes, or in different planes in various directions. See also Chapter 7.5 for discussion of indirect myofascial release methods.
- There is no need for active muscle contraction by the patient, who may be asked to maintain a state of active passivity.

Clinical procedure principles

- The therapist applies a slow, three-dimensional compression or traction causing the tissue to become tense. This is referred to as the first restriction barrier (Fig. 7.4.3).
- The applied pressure is constant during the first 60–90 seconds. This is the time required for releasing the first restriction barrier according to the viscoelastic response (Chaudhry et al. 2008).
- During the first phase of the technique, the therapist barely causes the tissue to move.
- Upon overcoming the first restriction barrier, the therapist accompanies the movement in the direction of the facilitation, pausing at each new barrier.
- In each application, the therapist may overcome three to six consecutive barriers. The time required is usually 3 to 5 minutes. Depending on the severity of the lesion, the process may take up to 30 minutes.



Fig. 7.4.3 • Cross hands technique on the thoracic region. The aim is remodeling of connective tissue in order to facilitate gliding movement at intrinsic and extrinsic structure levels. The therapist is standing at the level of the prone patient's back, and places crossed hands on the patient's back, pressing with a slight force towards the table and craniocaudally, following the principles of induction movement.

• The tension applied to the tissue must be constant, so the pressure (force) applied may need to be modified after overcoming the first barrier. Pressure should be reduced if there is an increase in pain and/or excessive movement or activity.

Scientific evidence related to the results in Myofascial Approach

Recent studies have focused on microscopic changes in connective tissue, as well as on clinical reactions, both in pathological and healthy subjects.

• Leonard et al. (2009), studying wound healing processes in 20 patients with diabetic foot ulcers,

concluded that connective tissue manipulation improved peripheral circulation and also enhanced wound healing.

- Significant differences between pre and post measurements of pressure pain thresholds, with decreasing sensitivity of myofascial trigger points, were reported in adductor longus strains (Robba & Pajaczkowski 2009), upper trapezius muscle (Fryer & Hodgson 2005), and cervical muscles (Hou et al. 2002).
- Marshall et al. (2009) concluded that myofascial release reduced the severity and intensity of muscle pain in people with chronic fatigue syndrome.
- Hicks et al. (2009) reported that human fibroblasts secrete the soluble mediators of myoblast differentiation, and that myofascial release can regulate muscle development.
- An objective form for evaluating the effect of MIT applications in muscular lesions with dynamic sonoelastography was reported by Martínez (2010) (Fig. 7.4.4).
- Heart rate variability and blood pressure recovery improved following myofascial release, after physically stressful situations, compared with sham electrotherapy treatment (Arroyo et al. 2008a).
- Arroyo et al. (2008b) reported that application of an active recovery protocol using whole-body myofascial treatment reduces EMG amplitude and vigor when applied as a passive recovery technique, after a high-intensity exercise protocol.
- Application of a single session of a manual therapy (including Myofascial Induction) program produces an immediate increase of heart rate variability and a decrease in tension, anger status, and perceived pain in patients



Fig. 7.4.4 • Sonoelastography images of a 3 weeks' muscle injury evolution (A, B). Image (C) taken after 30 minutes of MIT application. Note the significant change in local elasticity determined by the color scale. Courtesy of Martínez R, 2010.

with chronic tension-type headache (Toro et al. 2009).

- Arroyo et al. (2009), in a randomized singleblind placebo controlled study, reported that Myofascial Induction may encourage recovery from a transient immunosuppression state induced by exercise, in healthy active women.
- Henley et al. (2008) demonstrated quantitatively that cervical myofascial release shifts sympathovagal balance from the sympathetic to the parasympathetic nervous system.

In a study involving 41 healthy male volunteers randomly assigned to experimental or control groups, significantly decreased anxiety levels were observed in healthy young adults after the application of Myofascial Induction treatment. Significantly lower systolic blood pressure values versus baseline levels were also observed (Fernández et al. 2008).

In conclusion, MIT has been shown to encourage:

- more efficient circulation of antibodies in the ground substance
- an increase in the blood supply (release of histamine) in the region of restriction
- improved orientation in fibroblast mechanics
- greater blood supply to the nervous system
- an increase in metabolite flow to and from tissues, facilitating the recovery process.

Summary

Myofascial Induction Therapy (MIT) is a nonstandard treatment approach that, under different names, is currently used by a considerable number of therapists worldwide. MIT meets the criteria (Harris 1996) that allow its use as a manual treatment modality. The most relevant evidence relates to:

- anatomical fascial continuity
- intra- and interfascial force transmission
- scientific validation of treatment parameters
- definition of possible side effects
- identification of contraindications such as aneurysms, systemic diseases, inflammatory soft tissue process in the acute phase, acute circulatory deficiency, advanced diabetes, and anticoagulant therapy.

At present, clinical evidence is limited and unified research criteria should aim to identify:

- more objective evaluation processes
- classification of strategies (local versus global approach)
- unification of parameters of force, timing, intensity and frequency of application
- identification and analysis of responses in different body systems
- identification and classification of nonresponders
- analysis of long-term results.

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Osteopathic manipulative therapies and fascia

Hollis H King

Introduction

In virtually every osteopathic manipulative procedure, consideration of fascial elements is explicitly acknowledged and in some instances is the primary focus of the manipulation. The founder of osteopathic medicine, Andrew Taylor Still, is noted for sayings attributing to fascia central and extraordinary properties related to manual treatment and properties of human nature and disease. 'I write at length of the universality of the fascia to impress the reader with the idea that this connecting substance must be free at all parts to receive and discharge all fluids, and eject all impurities ... A knowledge of the universal extent of the fascia is imperative, and is one of the greatest aids to the person who seeks the causes of disease' (Still 1902, 61).

There is ample evidence that the manipulation techniques predominantly used by Still would currently be considered as articulatory and myofascial release maneuvers (Van Buskirk 2006).

Brous (1997, 23–24) has stated: 'If all other organs and tissues were removed from the body, with the fascia kept intact, one would still have the replica of the human body.'

The principle of the continuity of fascia throughout the body is a mainstay of osteopathic manipulative treatment (OMT), and has been adopted by virtually all professions who deliver healthcare service by application of manually guided contact with patients and clients.

To illustrate the central place of fascia in OMT, examples from the major associated modalities are described. This is followed by brief descriptions of formulations as to the nature of human fascia, as it impacts medical and therapeutic considerations of human anatomy and physiology. Finally, clinical and basic science research related to fascia, from the osteopathic perspective, are discussed.

Fascia in the perspective of OMT

High-velocity low-amplitude (HVLA) thrust or impulse techniques

High-velocity low-amplitude (HVLA) procedure is commonly applied in manual therapy/manual medicine as well as OMT. From the perspective of OMT, HVLA is defined as 'osteopathic technique employing a rapid, therapeutic force of brief duration that travels a short distance within the anatomic range of motion of a joint, and that engages the restrictive barrier in one or more planes of motion to elicit release of restriction' (Educational Council on Osteopathic Principles (ECOP) 2009). In that the 'restrictive barrier' almost always involves dysfunctional ligaments, instruction in how to perform HVLA is accompanied by a detailed consideration of the fascia, in and around any joint, for which there is an HVLA treatment prospect.

Kappler and Jones (2003, 855) state, 'As the barrier is engaged, increasing amounts of force are necessary and the distance decreases. The term barrier may be misleading if it is interpreted as a wall or rigid obstacle to be overcome with a push. As the joint reaches the barrier, restraints in the form of tight muscles and fascia serve to inhibit further motion. We are pulling against restraints rather than pushing against some anatomic structure.'

Muscle energy technique

Muscle energy technique is defined as 'a form of osteopathic manipulative diagnosis and treatment in which the patient's muscles are actively used on request, from a precisely controlled position, in a specific direction, and against a distinctly executed physician counterforce' (Educational Council on Osteopathic Principles (ECOP) 2009). Since tendons attach to virtually all muscles, fascia is involved in almost all muscle energy manipulative techniques.

Ehrenfeuchter and Sandhouse (2003, 882) state, 'He (Fred Mitchell Sr.¹) wrote about the direct method treatments of soft tissues (with attention to fasciae) and the treatment using Neidner's fascial release² prior to articular correction. Muscle energy technique, he wrote, with its many ramifications, is a most useful tool in preparation of soft tissues. Ligamentous stretching may also be of use before articular correction is attempted' (Mitchell 1958).

Strain-counterstrain technique

"Also called Counterstrain, the OMT procedure developed in 1955 by Jones (1964) is defined as 'An osteopathic system of diagnosis and indirect treatment in which the patient's somatic dysfunction, diagnosed by (an) associated myofascial tenderpoint(s), is treated by using a passive position, resulting in spontaneous tissue release and at least 70 percent decrease in tenderness' (Educational Council on Osteopathic Principles (ECOP) 2009). Counterstrain technique involves shortening myofascial structures to reduce the nociceptive experience from firm palpation of a tenderpoint.

Glover and Rennie (2003, 1003) state, 'The location of a specific tender point is constant from one patient to another. This suggests a strong anatomic basis for their location. Different myofascial structures, including tendons, ligaments, and muscle bellies

² William Neidner, DO worked with children with muscular dystrophy. He developed an effective myofascial OMT which is described in detail on page 324 of the 2003 second edition of *The Foundations for Osteopathic Medicine*.

have all been found to contain tender points ... another interesting anatomic correlation is the close proximity of tender points in areas where motor points are found. A motor point is the site where the motor nerve pierces the investing fascia and enters the muscle it innervates.' Without description of the involvement of fascia, counterstrain technique could not be adequately explained

Balanced ligamentous tension and ligamentous articular strain techniques

Developed and first presented by Sutherland in the early 1940s, balanced ligamentous tension (BLT) and ligamentous articular strain (LAS) techniques have fascial elements at the core of musculoskeletal diagnosis and treatment (Lippincott 1949). The basis of balanced ligamentous tension, 'according to Sutherland's model, all joints in the body are balanced ligamentous articular mechanisms. The ligaments provide proprioceptive information that guides the muscle response for positioning the joint, and the ligaments themselves guide the motion of the articular components' (Educational Council on Osteopathic Principles (ECOP) 2009).

Just as a manual medicine/manual therapy practitioners follow fascial planes in directions of ease of motion, the application of BLT focuses on the ligaments and related fascia holding joints in position, placing these structures into a balanced tension position, so that inherent bodily forces and/or respiratory facilitation complete the articular correction. Carreiro (2003, 917) states, 'The physician must skillfully position the joint so that all forces within the articular mechanism converge on one specific point. This point then becomes the fulcrum around which the shift or change will occur ... The more skilled the operator, the more specific the convergence and the less force needed to correct the dysfunction. Very skilled physicians will merely ask the patient to exhale, or will flex the patient's head to articulate the joint."

The Lippincott (1949) article describes Sutherland's techniques that would be termed myofascial release because they were directed toward structures such as diaphragms (respiratory, thoracic inlet and pelvic) that were not specifically articular. The BLT techniques have been elaborated and expanded, based on the work of Rollin Becker, DO and others

¹ Fred Mitchell Sr. is credited with the development of what is now known as Muscle Energy Techniques.

who carried on the teaching of Sutherland's techniques, and the term LAS has come into use because of a teaching manual published with that name (Speece et al. 2009).

Myofascial release techniques

As noted above, attention to fascia has been central to osteopathic medicine since the 1890s. Myofascial release (MFR) is 'a system of diagnosis and treatment first described by Andrew Taylor Still and his early students, which engages continual palpatory feedback to achieve release of myofascial tissues. Direct MFR – a myofascial tissue restrictive barrier is engaged for the myofascial tissues and the tissue is loaded with constant force until the tissue release occurs. Indirect MFR – the dysfunctional tissues are guided along the path of least resistance until free movement is achieved' (Educational Council on Osteopathic Principles (ECOP) 2009).

As this book illustrates, there are a number different schools of thought and teaching on the subject of myofascial technique. Out of the osteopathic tradition, besides BLT and LAS, there are two other sets of techniques directly concerned with fascia, the fascia-ligamentous release - indirect approach (Chila 2003) - and the integrated neuromusculoskeletal release and myofascial release (Ward 2003). A careful reading of Chila (2003) and Ward (2003) reveals that the underlying principles are very similar, but specific hand placements and areas of body contacted are somewhat different. Taken together, the Chila and Ward approaches comprise a comprehensive system of the application of myofascial treatment techniques. In fact, teaching in US osteopathic medical schools draws upon and combines the techniques of BLT and LAS along with integrated neuromusculoskeletal release (INR) and myofascial release (MFR). However, in the present context it is helpful to describe techniques associated with different terminologies because that is how they are identified in texts and teaching as well as in documentation for medical procedure description and coding for reimbursement of healthcare services in the USA.

Osteopathy in the cranial field

Also known as cranial osteopathy, cranial manipulation techniques involve great attention to intracranial dura (Magoun 1966). 'Osteopathy in the Cranial Field (OCF) -A system of diagnosis and treatment by an osteopathic practitioner using the primary respiratory mechanism and balanced membrane tension ... Refers to the system of diagnosis and treatment first described by William G. Sutherland, DO' (Educational Council on Osteopathic Principles (ECOP) 2009). 'It has been stated that Sutherland did for the head that which Still did for the rest of the body, which was to delineate an anatomically based understanding of range and vector of motion and physiologic dynamics of cranial bones and intra-cranial structures' (King 2011a). Structures such as the falx cerebri and diaphragma sellae are contiguous with spinal dura mater, presenting a basis for fascial manipulative techniques that can affect brain centers (King 2007).

While OCF originated in osteopathy, in the context of this book it is important to acknowledge that cranial manipulation has other proponents and perspectives, e.g., craniosacral therapy (Upledger & Vredevoogd 1983) and sacro-occipital technique (SOT) (DeJarnette 1967; Hesse 1991). All cranial manipulation traditions embrace the fascial continuity perspective and its importance in the application of therapy and treatment procedures.

Osteopathic manipulative treatment – summary

The forgoing discussion amply illustrates the central position that fascia holds in the formulation and application of OMT. A similar discussion could describe the attention given to fascia in other OMT techniques such as facilitated positional release (Schiowitz et al. 2003), progressive inhibition of neuromuscular structures technique (Dowling 2003), functional technique (Johnston 2003) and articulatory technique (Patriquin & Jones 2003).

Osteopathic contributions to the understanding of fascia

Common compensatory pattern

The concept of the common compensatory pattern (CCP) is based entirely on the nature of fascial patterns of preferred motion (also termed 'ease of motion') (Zink & Lawson 1979; Pope 2003). Among the many structural findings in the CCP were a left iliac crest more cephalad than right, superior and slightly lateral left anterior superior iliac spines, the pubic symphysis being more cephalad on the left, the left leg seeming longer, and the right leg more externally rotated. Among the descriptions of dynamic palpatory findings, the following is an example with the patient in a standing position:

'The palms of the physician's hands are placed on the anterosuperior iliac spines so that the fingers follow the crests. The physician's right hand moves with ease superiorly and laterally over the tissues, while the left hand moves with ease inferiorly and medially, resistance is encountered when the hands are moved in the opposite direction' (Zink & Lawson 1979).

'The CCP can be seen as a bias of the fascias of the body along its length, occurring from the ground up. Such that, with respect to the feet, the pelvic girdle is found to be rotated to the right, the lower thoracic outlet to be left, the upper thoracic outlet to the right, and the craniocervical junction to the left' (Pope 2003).

There are four transition zones, the occipitoatlantal (OA), cervical thoracic (CT), thoracolumbar (TL), and lumbo-sacral (LS), and the finding in healthy people is that the direction of fascial ease of motion alternates between these areas (Fig. 7.5.1).

The reason it is called the CCP is that, according to Zink and Lawson (1979), it occurs in about 80% of individuals and the reverse occurs in 20% and has been named the uncommon compensatory pattern (UCCP). The utility of the CCP concept was that Zink believed that the transition zones between the alternating fascial planes, ease of motion directions, were weak anatomically and should always be treated no matter what else was found to be dysfunctional. If these alternating fascial ease of motion planes were not found to alternate, for example, if all planes seemed to rotate in the same direction, this was called an 'uncompensated' pattern and would eventually lead to more serious health problems if not treated.

Treatment of the transitional zones could be done by muscle energy procedure to the OA junction, cranial manipulation procedure to the cranial diaphragm, but more generally followed conventional myofascial manipulation procedures in which the operator's hands contacted the area of concern. The tissues are followed in their preference for side-bending, rotation and any other vector of motion such as forward or backward bending and then held in that position as the patient breathes in and out. The operator follows the tissue preferences until the tissues 'release' in a 'letting-go' or 'softening' palpatory manifestation and approach the midline again.

While speculative, the theory of the origin of the CCP is worthy of note as it may be related to the birth process, in which the fetal head and body go through a series of positions as the birth canal is traversed, leaving a lifelong fascial motion preference. Evidence for this view is that the majority of fetal presentations are with the left occiput presenting first, and this affects the fascial motion preference as well as the position of the labyrinthine structures



Fig. 7.5.1 • Compensated and Uncompensated Patterns. Adapted from and reprinted with permission from Kuchera W, Kuchera M 1994 Osteopathic principles in practice. Columbus, OH; Greyden Press.

in the temporal bones which then maintain postural balance in a compensated manner (Pope 2003). Zink himself felt the CCP was due to injury caused by the many falls a child has during its early development from infant to toddler (Zink 1977). If research can confirm either hypothesis, one implication would be for more awareness to treat infants and toddlers.

As with many concepts, there is often a thread from Greek philosophy or art. An intriguing observation (Quinn 2000) is the possible historical observation of the CCP in art as seen in Greek art, such as the statue *Cidian Aphrodite* (c. 340–330 BCE) and described by the Italian word *contrapposto*, which refers to the natural pose of a figure where 'the parts of the body are placed asymmetrically in opposition to each other around a central axis' (Janson & Janson 1997) (Fig. 7.5.2).



Fig. 7.5.2 • In this rendering of the statue, *Cidian Aphrodite*, from 340–330 BCE, contrapossto is demonstrated by the S curve of the axial skeleton in relation to the tilt of the hips and shoulders in opposite directions. Adapted from and reprinted with permission from Quinn, American Academy of Osteopathy Journal 2000, 10, 23.

Bioelectric fascial activation and release

Though developed by O'Connell (1998), based on A.T. Still's writing, portions are acknowledged to come from the work of Fulford (1996), and reference to the piezoelectric properties of fascia from Becker and Selden (1985). Typical manual contacts made in the application of myofascial release technique are described and taught with emphasis on the bio-responsive electrical potentials of the fascia. O'Connell discusses and applies the concepts of tissue memory and subtle palpatory technique to treat such phenomena. While not yet in the mainstream of OMT, these techniques are consistent with Still's holistic, body-mind-spirit philosophy. Other osteopathic physicians who also teach and write on these topics are Upledger (1990) who describes tissue memory in the context of what he calls an 'energy cyst,' and Jealous (2001) whose biodynamic cranial osteopathy formulations parallel O'Connell's holographic description of subtle palpatory treatment technique.

Research

From the osteopathic perspective, there have been systematic reviews and meta-analysis demonstrating the benefit of OMT for chronic low back pain (Licciardone et al. 2005). Further discussions have presented data supportive of benefit for OMT in a wide variety of conditions (King 2011a, 2011b), all involving OMT techniques discussed above as having consideration of fascia as a central component of the mechanism of action.

More to the central thrust of this book is the work of Paul Standley (Dodd et al. 2006; Eagan et al. 2007; Standley & Meltzer 2008), which has demonstrated on in-vitro preparations of human fibroblast cells the effects of continuous vibration and strain intended to simulate indirect OMT and myofascial release techniques. Plate 7.5.1 shows the human fibroblast cell morphology grown on an elastomere base in four conditions. The non-strained cells were not subjected to any vibration or strain during the experiment. In the RMS (repetitive motion strain) condition, the cells received 8 hours of repetitive motion with a 10% deflection of the elastomere base. In the CS (counterstrain) condition, which can also be described in this context as a form of myofascial release, the cells received a 6% deflection of the

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elastomere base for 60 seconds and were then sampled. In the RMS + CS condition, cells received the RMS protocol, 8 hours of vibration, followed 3 hours later by the 60 second CS protocol. The cellular morphology differences give a visual image to the possible impact of manual therapy. The data on the secretion of proinflammatory interleukins (Table 7.5.1) suggest the possibility of both the effect of musculoskeletal injury and the effect of indirect myofascial release (OMT) on cellular secretions.

Implications for the direction of manually guided forces on human tissues is suggested in the Meltzer & Standley (2007) results (Table 7.5.2) which show different secretion patterns for equibiaxial strain (one way stretching) versus heterobiaxial strain (stretching in all directions at once). The authors state, 'These divergent observations in HETERO vs. EQUI strained fibroblasts may underlie the relative efficacies of manual medicine treatments carried out in different tissue strain directions' (Meltzer & Standley 2007).

Table 7.5.1 Enzyme-linked immunosorbent assay results for interleukins, by strain profile, normalized by relative proliferation indices. Reprinted with permission of the Journal of the American Osteopathic Association (Dodd et al. 2006).

		Strain Profile, Mean \pm SE*				
Interleukin	BCS	RMS	24RMS	24I0MT	24RMS $+ 10$ MT	
Proinflammatory						
IL-1α	3.41 ± 0.47	$2.44 \pm 1.69 \dagger$	4.48 ± 0.54	4.05 ± 0.21	2.54 ± 0.74	
IL-3	8.63 ± 0.44	6.46 ± 1.12	5.80 ± 0.67	3.89 ± 0.54 ‡	3.42 ± 0.85	
IL-6	80.32 ± 7.42	$70.65 \pm 1.16 \dagger$	92.47 ± 4.36	89.07 ± 7.94	42.73 ± 1.28 †	
IL-7	1.63 ± 0.78	3.94 ± 2.23	4.86 ± 3.58	4.63 ± 2.40	2.15 ± 1.60	

*Mean of the three samples, with standard error, for each interleukin, calculated by dividing enzyme-linked immunosorbent assay result by proliferation index result. Enzyme-linked immunosorbent assay was conducted for IL-1 β , IL-2, IL-1ra, IL-4, and IL-16, but these interleukins were not detected. *p < 0.05 vs. 24RMS.

[‡]p<0.05 vs. BCS.

Abbreviations: BCS, baseline cell secretion; IOMT, indirect osteopathic manipulative techniques; RMS, repetitive motion strain; 24IOMT, 24 hours post-IOMT; 24RMS, 24 hours post-RMS.

Table 7.5.2 Comparisons of cytokine secretions between equibiaxially and heterobiaxially strained fibroblasts as a percentage of change from their respective nonstrained control cells (mean \pm SEM). Reprinted with permission of the Journal of Manipulative and Physiological Therapeutics (Eagan et al. 2007).

strained

 $^{a}p \leq 0.05$ vs. 0 (no change).

 ${}^{b}p = 0.05$ vs. heterobiaxial percentage of change.

NA, Not applicable; IL, interleukin; MDC, macrophage-derived chemokine; NO, nitric oxide; PARC = pulmonary and activation-regulated chemokine.

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Connective tissue manipulation

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Connective tissue is responsible for providing and maintaining form in the body, surrounding muscles, membranes, and fibers and all systems including the nervous and musculoskeletal systems. Therefore, connective tissue dysfunction can potentially have adverse effects on skeletal muscle, the peripheral and central nervous systems, joint mechanics, and visceral structures. Connective tissue restrictions significantly contribute to myofascial pain syndromes and successful treatment outcomes are dependent upon including a connective tissue evaluation, and, if dysfunctional tissue is found, utilizing Connective Tissue Manipulation (CTM) or Connective Tissue Massage in the treatment plan. This section describes connective tissue physiology, the local mechanical and proposed reflexive effects of CTM, as well as how CTM is performed, involving loose connective tissue. Evidence for the clinical benefit of CTM is provided.

Loose connective tissue contributes to superficial and deep fascia, the intermuscular septa. It surrounds blood vessels and nerves, and provides a framework for most organs. It is the layer of tissue between the skin and the muscle, and has also been referred to as connective tissue proper (Ebner 1975). More recently, this tissue has been termed areolar connective tissue (Langevin et al. 2009). In addition to binding structures and holding them in their anatomical spaces, areolar connective tissue stores fat and helps the body conserve heat. The matrix of areolar connective tissue aids in tissue repair by depositing collagenous fibers and forming scar tissue. Finally, nutrients travel from the blood vessels through this tissue to individual cells as well as carrying metabolites away from the cells towards the blood and lymph vessels. The structure of connective tissue is physiologically dependent upon vascularity and blood supply, and is influenced by suprarenal hormones. The fibroblasts and the mast cells in connective tissue have an inverse relationship with suprarenal cortical hormones. That is, a decrease in secretion of these hormones will result in an increase in fibroblastic and mast cell activity, resulting in a water-retaining effect on the tissues (Ebner 1975; Holey 1995).

When connective tissue becomes dysfunctional, the problems that arise are in proportion to the support the tissues provide when they are healthy. Several different terms have been used to describe connective tissue restrictions and/or dysfunction. According to Maigne (1995) cellulagia is defined as: 'neurotrophic manifestations that include subcutaneous tenderness and thickening.' It can be detected by using the 'pinch-roll' test, in which a fold of skin is rolled between the fingers causing pain, with the clinician noting thickening. Ebner (1975) used the term *trophic edema* to describe thickened hypersensitive loose connective tissue. The terms panniculosis and *fibrositis* have also been used to describe dysfunctional connective tissue (Travell & Simons 1993; Kotarinos 2008) involving inflammatory hyperplasia of the white fibrous tissue. This chapter uses the term subcutaneous panniculosis to describe thickened connective tissue that is sensitive upon pinch rolling (i.e. dysfunctional connective tissue).

In addition to presenting as thickened or dense upon skin rolling, areas of subcutaneous panniculosis may show vasomotor, pilomotor, and sudomotor reactions, increased subcutaneous fluid, and atrophy or hypertrophy of the underlying muscles (Chaitow 1988). Underlying muscle atrophy is the resultant effect of the thickened tissue interfering with proper functioning of sodium-potassium pumping mechanisms in muscles (Ebner 1975). Panniculosis can cause local nociceptive pain via the peripheral nervous system, and it is hypothesized to cause referred pain in distant locations through the central (Bischof & Elmiger nervous system 1963: Fitzgerald & Kotarinos 2003). Several mechanisms have been identified to explain how dysfunction develops in the subcutaneous tissue. These are as the result of visceral referred pain, in tissue superficial to myofascial trigger points, in the cutaneous distribution of inflamed peripheral nerves, and superficial to areas of joint dysfunction.

Viscerosomatic reflex

The viscerosomatic reflex is a reflex in which somatic manifestations occur in response to visceral disturbances. More specifically, the visceral–cutaneous reflex is a phenomenon where disturbances or disease in visceral organs refer pain along the distribution of somatic nerves which share the same spinal segment as the sensory sympathetic fibers to the organ affected (Head 1893; Janig 1996). The visceral–cutaneous reflexes have been studied by many and are commonly referred to as Head's zones, Chapman's reflexes, or Mackenzie's zones (Fig. 7.6.1).

The reflex is initiated by afferent impulses from visceral receptors, then impulses travel to the dorsal



Fig. 7.6.1 • Reflex abdominal areas (after Mackenzie).

horn of the spinal cord, synapse with interconnecting neurons, and connect with sympathetic and peripheral motor efferents, resulting in sensory changes in the blood vessels and skin (and also muscle and viscera) (Bischof & Elmiger 1963; Arendt-Nielsen et al. 2008). If the pathological visceral afferent stimulation becomes chronic, neurogenic plasma extravasation will occur in the skin, thereby causing vasoconstriction in the periphery, hyperesthesia, and thixotropic changes. In more recent literature, three plausible neural mechanisms have been identified in animal models to explain visceral-cutaneous reflexes. Takahashi (1996) noted that plasma extravasation occurred as a result of antidromic stimulation of C-fibers in spinal nerves. It is noted that with unilateral stimulation changes in the skin can occur unilaterally or bilaterally. Studies suggest that the actual mechanism may be a combination of the three described processes, as noted by Wesselmann and Lai (1997). One potential mechanism is described because dichotomizing sensory neurons have a branch to both the uterus and to the skin. Uterine inflammation could cause excitation of the visceral branch of the afferent neuron, leading to antidromic activation of the somatic branch, causing neurogenic plasma extravasation. Wesselmann also hypothesized that visceral afferent neurons may excite cutaneous afferent neurons. The result of this spinal mechanism is antidromic activation of cutaneous afferent fibers, again resulting in plasma extravasation. It was suggested that the sympathetic postganglionic nerve terminals must be intact, demonstrated by a decrease in plasma extravasation when the anterior spinal root was not intact (Wesselmann & Lai 1997). Similarly, basic science studies (Beal 1985) have shown that somatic disturbances cause visceral changes, otherwise known as the somatovisceral reflex (Sato 1995).

Superficial to muscles with myofascial trigger points

Travell and Simons (1993) reported a strong association between active myofascial trigger points and subcutaneous connective tissue restrictions. Dermographia/fibrositis commonly occurs most often over muscles of the back of the neck, shoulders and torso, and less frequently over limb muscles. In panniculosis, the subcutaneous tissue exhibits increased viscosity suggestive of thixotropy. Travell and Simons proposed that the connective tissue restrictions may be related to sympathetic nervous system activity involving mechanisms operating in the underlying myofascial trigger points. Treating the panniculosis can relieve myofascial trigger point activity and/or make the underlying myofascial trigger point more responsive to treatment. Travell and Simons identified the need for designed studies to evaluate the relationship between myofascial trigger point activity and overlying panniculosis.

Dermatomes of inflamed neural structures

The 'pinch-roll' test (of the subcutaneous tissue in the territory of a peripheral nerve) is commonly accepted as a clinical indicator of inflamed neural tissue. Referred pain is accompanied by hyperalgesia of the skin and subcutaneous tissues in the involved dermatomes. This hyperalgesia or hypersensitivity can be revealed by gently grasping a fold of skin between the thumbs and forefingers, lifting it away from the trunk and rolling the subcutaneous surface against itself in a pinch and roll fashion. The entire dermatome may be affected, or only partial tissue changes may be noted (Maigne 1996; Beco 2004).

Superficial to areas of joint dysfunction

Robert Maigne coined the term 'cellulagia' when reporting that intervertebral joint dysfunction causes neurotrophic reflexes. Cellulagia can occur in the skin innervated by the corresponding nerve roots and in tissue superficial to areas of vertebral dysfunction (Maigne 1996).

Physiology of connective tissue manipulation (CTM)

Connective tissue manipulation results in both local mechanical effects and hypothesized reflexive effects. The mechanical effects of the manual therapy technique include vasodilation, improved tissue mobility, decreased nocigenic chemicals, autonomic reactions (favorable and unfavorable), decreased hyperalgesia and improved tissue integrity (Holey 1995). Reflexively, it has been proposed that CTM stimulates a response in the sympathetic terminal reticulum in the skin. The impulse is then carried through the autonomic nervous system (ANS), through the sympathetic trunk and spinal cord, down the efferent autonomic root cells, to the segmental sympathetic ganglion or neighbor, and the diseased organ (Ebner 1975).

Understanding the physiology of connective tissue can help in understanding why unresolved connective tissue restrictions can cause visceral disturbance, muscle dysfunction, adverse neural tension, further connective tissue restrictions, and perseverance of myofascial pain syndromes.

Connective tissue manipulation

Connective tissue manipulation (CTM) (Bindegewebsmassage), originally developed by Elizabeth Dicke (1953) and a group of physical therapists and physicians in Germany, is a manual technique performed in restricted subcutaneous tissue to restore normal tissue mobility and circulation. It was initially developed as an adjunct therapy for existing organic disease such as cardiac or respiratory disease. More recently it has also been used to treat various myofascial impairments. Patients who develop restricted connective tissues, regardless of the etiology, report hypersensitivity to touch, intolerance to tight-fitting clothing, pain during tissue compression (e.g., pain in the posterior thighs when sitting), pain upon stretch (e.g., posterior thigh pain during a hamstring stretch), cutaneous pain without provocation, itching, and poor tissue integrity. The severity of the connective tissue restrictions correlates with the severity of symptoms being experienced. For example, mild tissue restrictions will cause slight tissue irritation when compressed for long periods, whereas moderate restrictions may cause hypersensitivity to touch. Severely restricted tissue can cause pain without touch, stretch or compression, and/or skin fissures. In the authors' clinical experience, such symptoms are often misunderstood, and go mis- or untreated.

Clinical research has exhibited evidence that body surface, organs, and connective tissue, within a segmental nerve supply, are connected by reflexive pathways (Holey 1995) (Fig. 7.6.2).

Originally, CTM techniques were developed to address connective tissue associated with a specific segmental nerve supply to specific visceral structures.

Fascia-Oriented Therapies



Fig. 7.6.2 • Connective tissue reflex zones (after Ebner).

The German therapists who developed CTM reported benefits for patients suffering from cardiac and respiratory diseases, peripheral circulatory deficits, neurological pathologies, gynecological and obstetric problems, and disorders of the digestive and urinary tracts. CTM is currently primarily used to address spinal and peripheral joint dysfunction, osteoarthritis and rheumatoid disease, nerve root pain, sciatica, and neuralgia (Gifford & Gifford 1994).

Evaluation

Dicke (1953), and more recently Ebner (1975), described strokes applied in specific directions and patterns, depending on the pathology. The authors' use of CTM is based on Dicke's technique in theory and practice, with some modifications. In this text, CTM is described as employed by the authors.

Connective tissue can only be assessed manually. There are no other current diagnostic tests that can accurately assess this tissue for dysfunction. Evaluation begins with inspection of the skin for visible signs of tissue dysfunction, to direct the treatment. Dysfunctional tissue may be discolored, drawn in, or flattened (Head 1893), with noticeable underlying muscle hypertrophy or atrophy, and/or skin integrity may be poor. Before a manual evaluation of the connective tissue, a thorough musculoskeletal examination is completed. The location of impaired tissue guides the clinician to the areas to begin the CTM assessment.

The tissue surrounding musculoskeletal dysfunction should be evaluated thoroughly. The starting location is typically distal to the primary impairment, moving from distal to proximal, until the entire area superficial to the impairments and the surrounding tissues has been assessed. To begin, the patient lies supine or prone, and, using minimal lubricant cream, the tissue is assessed by rolling it between the tips of the thumbs and fingers. The tips of the thumbs slide underneath the connective tissue layer, parallel to the underlying muscle, while the fingers grasp the subcutaneous tissue, pulling it towards the thumbs. The clinician should not use fingerpads as this will prevent a sufficient grasp of the tissue. Using the fingertips to mobilize the tissue is essential for correct performance of CTM. Short fingernails are therefore essential. The force of the tissue-grasp is firm and fairly superficial, with minimal pressure onto the skin. The direction of force is parallel to the tissue, not perpendicular. Less restricted tissue will mobilize more easily than more restricted tissue. The initial strokes allow the practitioner to palpate the tissue for contour, temperature, sensitivity, elasticity, turgor, and bulk. Restricted connective tissue will be colder, hypersensitive, less elastic, thickened ('dense'), and bulkier.

Dicke's CTM technique (1953) had the patient seated, with no lubricant used during assessment or treatment. The strokes used involved the tissue being gripped between the thumb and fingers, with variations involving the number of repetitions and the patterns used in different areas.

Treatment

Dicke's CTM protocol includes very specific strokes and patterns of movement through the body, depending on the pathology or impairment. Treatment started at the sacrum and continued in a cephalad direction to the lumbar region, then to the thoracic and cervical region. Depending on the pathology, the treatment pattern continued to the shoulder, pectoral region, deltoid region, forearm and hand, femoral region, posterior aspect of thigh, knee and leg, anterior aspect of trunk, subcostal region, pelvic margin, rectus abdominus, anterior chest, anterior pelvis, face, and lastly the posterior aspect of the head.

The CTM technique, as described in this text, does not follow such a strict protocol. The authors suggest that treatment should be identical to assessment in technique, with the tissue being mobilized until an improvement in mobility, decrease in sensitivity, and an increase in warmth are detected. The same tissue is mobilized in every direction to assure that improved mobility is achieved. This CTM approach is not dependent on a particular number of strokes, or a particular direction or pattern of strokes, but on positive changes in the tissues being treated. Treatment may be long or short term, depending on the severity of the tissue restrictions. Typically, a series of treatments is required, as the effects are cumulative. The treatment session may take from 30 minutes to 2 hours. The expected response, during treatment, from a patient with tissue dysfunction, is a report of a cutting or scratching sensation, or a feeling of dull pressure. In significantly restricted tissues, CTM is initially painful. The severity of tension in the tissue correlates to the severity of the pain response. As tissue mobility improves, painful sensations reduce. Individuals with healthy tissue seldom report uncomfortable sensations during CTM application. Because of the effect of CTM on the ANS, patients may report dizziness, nausea, increased sweating. A minority of patients may faint (Ebner 1975; Frazer 1978; Beal 1985). Frequently, patients will report a virtually immediate relief in visceral or myofascial pain and/or dysfunction (Ebner 1975; Gifford & Gifford 1994; Holey 1995).

The expected skin response, described by Lewis in 1927 as the 'triple response,' includes, sequentially, the appearance of a red line, then a red flush in the tissue if the stroke is repeated in the same area, then a slight swelling of the tissue (wheal). The first reaction always occurs if there is tension in the tissue and the technique is performed correctly, and the last two occur depending on the strength of the stimulus and the number of stroke repetitions. The skin response lessens as tension in the tissue decreases. Bruising is a common reaction following the first two to four treatments and is not a contraindication to treatment. Typically, the tissue remains sensitive for 2–3 days following treatment. After a series of treatments, post-treatment reactions diminish.

Special considerations include: overweight patients will have more tension, older patients will feel 'looser,' and certain anatomic sites will have more or less tension. For example, connective tissue overlying the thoracic spine and iliotibial band is typically tense. Patient education as to what to expect during and after treatment is advised.

The goals of CTM include improved circulation and tissue integrity, decreased ischemia, reduced nocigenic chemicals in the restricted tissue, decreased or eliminated visceral pain or dysfunction, and decreased adverse neural tension on peripheral nerve branches.

If these restrictions are not resolved, underlying muscle hypertonus can occur or increase, poor skin integrity can persist or worsen, adverse visceral reactions will continue or worsen, and the pathways of peripheral nerves can become restricted or compressed.

Contraindications

There are few contraindications to CTM. The only reported ones include manipulating tissue over a malignancy, acute inflammation or closed abscesses, and women in the third trimester of pregnancy (Goats & Keir 1991).

Evidence of clinical benefit

Basic science research has demonstrated the interaction between muscle, skin, viscera and central and peripheral nervous systems, supporting the importance of addressing connective tissue as part of any treatment program. Recent clinical research shows the physiological and clinical benefits of CTM. One study demonstrating the benefit of CTM evaluated the efficacy of a rehabilitation program based on the combination of CTM and McMennell joint

Case 7.6.1

The evidence of benefit of CTM can be demonstrated in a case study.

Lise was a 27-year-old female with a 10+ year history of vaginismus. Her first symptoms were inability to insert a tampon as a teenager. She was unable to engage in intercourse because of severe vaginal 'tightness' and pain. Her current symptoms included vaginal burning which increased with sitting and exercise, pain with sexual arousal, and severe clitoral hypersensitivity. A visual inspection of her vulvar tissues revealed very darkened labia (Fig. 7.6.3A), left greater than right, and a white clitoral hood. Upon examination, she had severe connective tissue restrictions along the bony pelvis and moderate restrictions in her thighs and gluteals. A Q-tip test to the vestibule revealed severe hypersensitivity at all points. Additionally, she presented with a hypertonic pelvic floor and adverse neural tension on the dorsal clitoral nerve branches bilaterally. She received physical therapy treatment which included primarily CTM, myofascial release to the pelvic floor musculature, and neural mobilization. After 3 months of therapy the clinician noted a decrease in pelvic floor muscle hypertonus, an increase in connective tissue mobility, particularly in the thighs and gluteals, and a decrease in adverse neural tension. The patient noted a considerable decrease in unprovoked vaginal pain, some decrease in pain with sitting, and a slight decrease in vestibule and clitoral hypersensitivity. After 6 months of therapy her pelvic floor was beginning to normalize, but remained unstable, the connective tissue around the bony pelvis was improving as was the color of the vulvar tissues and the clitoris (Fig. 7.6.3B), the vestibule had less hypersensitivity with Q-tip testing, and the clitoral nerve mobility was normalizing. Subjectively, she noted her sitting and walking tolerance continued to improve, vaginal pain was minimal, and vulvar and clitoral hypersensitivity was decreasing. The next 3 months of treatment focused on normalizing the connective tissue around the bony pelvis, particularly around the clitoris and vulvar tissues. At 9 months of treatment the color of her vulvar tissues and clitoris had improved further, Q-tip testing revealed minimal vestibule sensitivity, and her pelvic floor was within normal limits. At this time she had mild hypersensitivity of her vulva and clitoris, mild sitting discomfort, and mild to moderate discomfort with arousal and walking. After 11 months of physical therapy her vulvar tissues had improved dramatically versus her initial evaluation (Fig. 7.6.3C) and Q-tip testing revealed minimal to zero sensitivity in the vestibule.





Fig. 7.6.3 • A Before physical therapy treatment. B After 3 months of physical therapy treatment. C After 6 months of physical therapy treatment she reported she was able to insert a medium dilator without pain, had minimal to zero vaginal pain, minimal to zero vulvar and clitoral hypersensitivity with provocation, mild itching with arousal, and mild discomfort with long-term sitting and moderate walking.

manipulation, specifically for the hands of patients suffering from systemic sclerosis (Maddali Bongi et al. 2009). Of the 40 patients enrolled, 20 (interventional group) were treated for a 9-week period with a combination of CTM, McMennell joint manipulation and a home exercise program, while 20 (control group) were assigned to a home exercise program. The interventional group improved in multiple functional and quality of life tests at the end of the treatment (p < 0.0001) versus the control group. It was concluded that the combined treatment may lead to an improvement of hand function and quality of life.

In 2008, Kotarinos (2008), in association with the Urological Pelvic Pain Collaborative Research Network and the National Institutes of Health, examined the feasibility of conducting a randomized clinical trial to compare two methods of manual therapy, external and internal myofascial physical therapy (MPT), compared to traditional external global therapeutic massage (GTM) in patients with urologic chronic pelvic pain syndromes. CTM was the primary external myofascial technique in the MPT group. Both treatment protocols were standardized. Results showed that the MPT group had a positive response rate of 57%, significantly higher than the 21% response in the GTM treatment group (p < 0.03). This suggests that MPT (CTM) represents a clinically meaningful treatment option for patients with myofascial pelvic pain and dysfunction.

Brattberg (1999) investigated the effect of CTM in the treatment of patients with fibromyalgia. Forty-eight individuals diagnosed with fibromyalgia were randomized, with 23 in the treatment group and 25 in the reference group. After 15 treatments of CTM the treatment group reported a pain relieving effect of 37%, reduced depression and the use of analgesics, and positive effects in their quality of life.

Kaada and Torsteinbo (1989) examined the concentration of plasma beta-endorphins in 12 volunteers before, and 5, 30, and 90 minutes after a 30-minute session of CTM. They found a moderate mean increase of 16% in beta-endorphin levels from 20.0 to 23.2 pg/0.1 mL (p < 0.025), lasting for approximately 1 hour, with a maximum in the test 5 minutes after termination of treatment. They concluded that the release of beta-endorphins is linked with the pain relief and feeling of warmth and well-being associated with CTM.

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Fascial manipulation

Carla Stecco Antonio Stecco

Introduction

Fascial Manipulation[®] is a manual therapy for the treatment of musculoskeletal pain developed by Luigi Stecco, an Italian physiotherapist. This method, which has evolved over the last 30 years through anatomical studies and clinical practice, is based on a three-dimensional biomechanical model for the human fascial system (Stecco 1988, 1990, 1996, 2004; Stecco & Stecco 2009). The key premise of this model is that fascia is not just a uniform membrane, but it presents a specific organization and relationship with the underlying muscles. In particular, the fascia is seen as a:

- coordinating element for motor units (grouped together in myofascial units)
- uniting element between unidirectional myofascial units (myofascial sequences)
- connecting element between body joints via myofascial expansions and retinacula (myofascial spirals).

This biomechanical model is supported by in-depth studies of fascial anatomy and physiology. Numerous dissections of unembalmed human cadavers have evidenced:

- muscular fiber insertions directly onto deep fascia (Stecco et al. 2007a)
- fiber distribution according to precise motor directions (Stecco et al. 2008a, 2009a)
- myotendinous expansions that link adjacent segments (Stecco et al. 2009b).

Extensive histological analysis of deep muscular fascia has also provided evidence for hypotheses

concerning fascia's role in proprioception and tensional force distribution within the fascial system (Stilwell 1957; Yahia et al. 1992; Stecco et al. 2006, 2007a). Current anatomical research is oriented towards studies of:

- the superficial fascia, for its influence on lymphatic and venous return mechanisms
- the internal fasciae, for its influence on visceral dysfunctions (Stecco in press).

Treatment modalities specifically addressing these fascial layers have been developed.

The Fascial Manipulation[®] method for musculoskeletal dysfunctions is characterized by an analytical procedure that results in personalized treatment for each subject. A combination of codified movement and palpatory tests permits therapists to determine which *fascial points*¹ are involved in any given dysfunction. Each of these *fascial points* has a precise anatomical location within the fascial system, based on a functional interpretation of movement, as provided by the biomechanical model. A fundamental aspect of this method lies in differentiating between the area where the patient actually perceives pain, and the fascial points that require treatment.

The biomechanical model

The Fascial Manipulation[®] method considers the myofascial system as a three-dimensional continuum and aims to act upon the deep muscular fascia, including epimysium and retinacula, in the treatment

¹ These points/areas are described more fully, later in the chapter.

of musculoskeletal pain. This continuum is not aspecific, but well organized and easily analysable with an innovative biomechanical model that interprets the fascial system from a functional viewpoint. The base element or functional unit of this biomechanical model is the myofascial unit.

The myofascial unit

Each myofascial unit (MFU) comprises monoarticular and biarticular muscular fibers, the fascial structures, bones, nerve terminations and the specific portion of a joint involved in moving a body segment in a specific direction. In other words, each MFU is a functional unit composed of three elements that work in unison:

- the force-exerting element the unidirectional muscle fibers
- the coordinating element the fascia
- the perceptive element the nerve structures, the joint capsule and ligaments.

A significant characteristic of each MFU is the presence of both monoarticular and biarticular muscular fibers. The monoarticular fibers in each MFU are generally deeper fibers, specialized in moving a joint on one plane, and these fibers could be involved in the interplay between agonists and antagonists. In almost every MFU, a number of monoarticular fibers insert onto the intermuscular septum that separates two antagonist MFUs on the same plane. Whenever the agonist MFU is activated, traction exerted on the intermuscular septum could cause tension in the antagonist MFU, contributing to simultaneous adaptation, according to the inclination of the fibers and the segment involved. Recent studies of agonist and antagonist interaction by Huijing (2009) support this hypothesis.

The biarticular fibers in each MFU could intervene in synchronizing the activity of two in-series MFUs, modifying the position of the proximal segment in relation to movements of the distal segment, or vice versa, when necessary. At the same time, the monoarticular fibers of the respective MFUs could provide added stability for joints as they move.

Different studies evidence the role of monoarticular and biarticular muscle fibers in multiple joint movements (Savelberg & Meijer 2003; Kurtzer et al. 2006).

Within each MFU, some muscle fibers also insert directly onto the overlying fascia. These insertions could contribute to the maintenance of a basal tension of the fascia, and guarantee that fascia is stretched in a specific direction each time these muscle fibers contract (Stecco et al. 2008a, 2009b).





Within each MFU, two specific points can be identified (Fig. 7.7.1):

- A *center of perception* (*CP*): a precise area of the joint where traction exerted by the MFU on the joint capsule, tendons and ligaments is thought to converge. In a dysfunctional MFU this traction is not aligned along the correct physiological axis, causing joint movement to be incongruent or out of alignment. Over time, this could determine joint conflict, with friction and subsequent inflammation of periarticular soft tissues resulting in sensations of pain or joint instability.
- A *center of coordination* (CC): a small area on the deep muscular fascia where force exerted by the muscular fibers of a MFU converges (the 'point,' referred to earlier). The resultant myofascial forces could be transmitted to the surface of the deep fascia via its continuity with the endomysium, perimysium, and epimysium. The CC within each MFU is thought to have the role of coordinating the motor units that are comprised within that MFU.

Evidence exists regarding reduced coordination of motor units in the presence of joint pain (Mellor &

Hodges 2005), although the mechanism is unknown. The Fascial Manipulation model suggests a new neurophysiological basis for the coordinating role of the CC. During any movement, motor units are activated, causing muscle fibers to contract according to the degree and direction of required joint movement. Muscle spindle capsules, embedded between muscle fibers, are continuous with surrounding endomysium; therefore, when gamma fiber stimulation causes intrafusal spindle fibers to contract, a minimal stretch is propagated throughout the entire fascial continuum, including the fascia at the CC. If the fascia at the CC is elastic, then it will adapt to this stretch, permitting muscle spindles to contract normally, correct activation of alpha motor fibers, and subsequent muscular contraction to proceed smoothly. If fascia at the CC is not elastic, the muscle spindle contraction could interfere with motor unit activation. Incongruent motor unit activation would then result in uncoordinated movement, perceived at the CP either as joint instability or as pain (Pedrelli et al. 2009a).

For each body segment, six MFUs have been identified and each one is specific for one direction. This is also true for those joints that have limited movement on some planes (e.g., frontal plane for the knee or elbow) because these joints always have muscular and fascial components that act as stabilizers on those planes.

Each MFU is located on one of the three spatial planes. The MFUs associated with the movement of antemotion² (AN) are located in the anterior region of the limbs and trunk while the MFUs associated with retromotion³ (RE) are located in the posterior region. The MFUs associated with lateromotion (LA), movement away from the midline, are all located in the lateral region of the limbs and trunk, and those MFUs associated with mediomotion (ME), movement towards the midline, are located in the medial region. The MFUs associated with extrarotation (ER) are located in the retrolateral region of the limbs and trunk, and those associated with extrarotation (ER) are located in the retrolateral region of the limbs and trunk, and those associated with extrarotation (ER) are located in the retrolateral region of the limbs and trunk, and those associated with

intrarotation (IR) in the anterolateral region (Fig. 7.7.2). Each MFU is named according to the segment it moves and the direction in which it moves that segment. There are 14 body segments and 6 directions, making a total of 84 CC and 84 CP (Fig. 7.7.3).

The sequences

Myofascial sequences are formed by a succession of unidirectional MFUs positioned in a specific direction. This organization permits single MFUs to synchronize their activity, particularly during forceful



Fig. 7.7.2 • Sections of the trunk (CL, neck; TH, thorax; LU, lumbi; PV, pelvis). Six MFUs are recognized for each section. The different MFUs show a specific spatial disposition; in particular, the MFUs associated with the movement of antemotion (AN) [flexion] are located in the anterior region of the trunk, the MFUs associated with retromotion (RE) [extension] are located in the posterior region, the MFUs associated with lateromotion (LA) [lateral side-flexion] are all located in the lateral region of the trunk, the MFUs associated with mediomotion (ME) [medial side-flexion] are located in the medial region, the MFUs associated with extraotation (ER) are located in the retrolateral region of the trunk and those associated with intrarotation (IR) in the anterolateral region. In this figure the sequence of retromotion is highlighted.

² The authors have chosen the term ANTEMOTION because it has a precise directional significance. Even the CNS organizes movement according to spatial directions and not according to closure (flexion) or aperture (extension) of joints. The MFUs of ANTEMOTION (AN) are all implicated in the forward movement of a body segment on the sagittal plane and these MFUs are always situated in the anterior region of the body. At the knee, for example, the ANTEMOTION MFU is involved in coordinating the forward movement of the lower limb on the sagittal plane, commonly termed knee extension.

³ For the same reason as mentioned in Note 1, the authors have chosen the term RETROMOTION to define all backward movements of a segment on the sagittal plane.



Fig. 7.7.3 • Segments of the body • For each segment, six MFUs are recognizable. The segments are: DI, digiti/ fingers; CA, carpus/wrist; CU, cubitus/elbow; HU, humerus/shoulder; SC, scapula; CP, caput/head; CL, collum/neck; TH, thorax; LU, lumbi; PV, pelvis; CX, coxa/ hip; GE, genu/knee; TA, talus/ankle; PE, pes/foot. From Stecco 2004, with permission.

movements, and to monitor upright posture in the three spatial planes.

From an anatomical viewpoint, unidirectional MFUs connect to each other via:

- the muscular fascia, which unites them within the same fascial compartment
- the biarticular muscle fibers that extend between two MFUs in series
- the myotendinous expansions onto the overlying fascia that extend between segments.

The orientation of these myotendinous expansions (Stecco et al. 2007b, 2008a) guarantees that fascia is stretched simultaneously in more than one point, such that even minimal movement in a specific direction is perceived.

One example of a myofascial sequence is the ANTEMOTION sequence of the upper limb. It is formed by the following MFUs:

 AN–SC, forward movement of the scapula, motor units from pectoralis major (biarticular fibres) and pectoralis minor (monoarticular fibers) and their connecting fascia

- **2.** AN–HU, forward movement of humerus, motor units from the clavicular head of pectoralis major, long head of biceps (biarticular fibers), anterior deltoid, and coracobrachialis (monoarticular fibers) and their connecting fascia
- **3.** AN–CU, forward movement of elbow, motor units from biceps brachii (biarticular fibers) and brachialis (monoarticular fibers) and their connecting fascia
- **4.** AN–CA, forward movement of wrist, motor units from flexor carpi radialis (biarticular fibers) and flexor pollicis longus (monoarticular fibers) and their connecting fascia
- **5.** AN–DI, forward movement of fingers, motor units from flexor pollicis longus (biarticular fibers), flexor and abductor pollicis brevis (monoarticular fibers) and their connecting fascia.

Note that biarticular muscle fibers and deep fascia (brachial and antebrachial), onto which pectoralis major, biceps brachii and flexor carpi radialis all extend robust myotendinous expansions, unite these MFUs together in one myofascial sequence.

All myofascial sequences terminate in the extremities: fingers, toes, and head (Fig. 7.7.4). Tensional compensation beyond these extremities is not possible. Fascial fibrosis along a sequence could culminate in myofascial retraction of the digits and, over time, possibly lead to bony deformation (e.g., hammer toe). According to which digits are involved, the corresponding myofascial sequence is identifiable.

This overall concept of myofascial sequences can assist in the interpretation of the spread of tensional compensations throughout the fascial system. Different authors agree that compensations do not extend haphazardly (Rolf 1977; Zink & Lawson 1979; Myers 2001). Some of the more common types of compensations include:

- ascending/descending
- homolateral/contralateral.

If the human body were formed by a mere single articulation, then compensation between agonist and antagonist would be sufficient to maintain equilibrium. However, tensional equilibrium involves numerous segments and each articulation regulates its alignment in relation to proximal and distal


Fig. 7.7.4 • Confluence of the sequences in the hands, head and feet.

segments. In clinical practice, dysfunctions distributed over one spatial plane and, at times, over more than one plane, are much more frequent than pure, segmental dysfunctions. This method stresses the importance of re-establishing equilibrium through the correct interpretation of compensations, working appropriately to distend an entire sequence as well as restoring balance between agonist and antagonist MFUs (Day et al. 2009).

The spirals

Each joint commonly moves through intermediate degrees, shifting from one plane to the next. This requires a gradual decrease in the activity of one MFU, simultaneous increase of activity in an adjacent MFU, and activation of the appropriate rotatory MFU (in intra- or extrarotation). Furthermore, limb segments often move simultaneously in opposite directions, rather than simultaneously in a single direction. Other points on the deep muscular fascia, called centers of fusion (CF) participate with CC in the coordination of these more complex movements. Anatomically, CF are located over the retinacula, which are specialized reinforcements of the muscular fascia in periarticular regions (Stecco et al. 2008b, in press a). Retinacula actually continue from one joint to the next, via the oblique collagen fibers within the deep fascia itself, and these oblique fibers create macroscopically visible, extended spiral formations. During complex movements, e.g., walking or running, these spiralform collagen fibers wind and unwind, tensioning the retinacula, and thereby activating, deactivating, and synchronizing the CF. At a segmental level, CF are considered to be responsible for monitoring intermediate movements between two directions, whereas when activated in a myofascial spiral, they could monitor movements of adjacent segments in opposite directions.

Treatment

In the Fascial Manipulation[®] method, it is fundamental to go beyond the idea of treating the site of pain (CP), and to trace back to its fascial origin in the corresponding CC and/or CF requiring treatment.

Altering the fascia within an MFU could cause:

- inaccurate muscle recruitment
- nonphysiological joint movement
- activation of joint nociceptors
- joint pain.

Thus, it is logical to address the cause of the problem rather than the effect, and a clear understanding of MFU anatomy will assist in this research.

The Fascial Manipulation[®] method has a systematic assessment process for evaluating MFU function. After recording an accurate history, the next step involves specific movement tests to highlight nonfunctional MFUs. Since each MFU performs a single movement, at a single joint and in a specific direction, isotonic or isometric movement tests can reveal which plane of movement is more limited and/or painful. Therefore, to test the single MFUs, six specific movement tests have been chosen for each segment.

The next step is comparative palpation of potentially altered CC, as indicated by the movement tests. In this phase, it is important for therapists to know the precise localization of the different CC. The therapist compares sensations perceived by the patient during palpation (e.g., needle-like pain, referred pain) and the quality of the fascial tissue (e.g., fibrotic, lack of elasticity, etc.). Under normal conditions CC and CF are not painful, and do not produce referred pain when palpated, because if fascia is elastic it adapts to compression, and embedded receptors are not irritated. Pain on palpation indicates an altered state of the deep muscular fascia, implying that this fascia is unable to accommodate to stretch from underlying muscle fibers, and that embedded receptors, such as free nerve endings, have a lowered pain threshold due to overstimulation.

Accurate compilation of the appropriate evaluation grid will then highlight the degree of involvement of the various MFU, facilitating selection of CC/CF to be treated (Fig. 7.7.5). Single segment problems are relatively rare. Dysfunctions often involve adjacent segments (agonist/antagonist compensation), or compensation along fascial compartments (myofascial sequence), or an alternating pattern in different joints (myofascial spiral).

The biomechanical model is useful for interpreting the passage of compensation from one MFU to another, or the evolution from an initial segmental disturbance to a more generalized dysfunction. Evaluation may necessarily extend:

- from one MFU to a distal or proximal CC along the same myofascial sequence
- to CC in the antagonist sequence
- to associated CF.

This investigation process can also include so-called 'silent' CC and CF. These points are not indicated by the specific movement tests, and are deduced from the context of the dysfunctional movement or posture. In this way, each individual treatment consists of an individual selection of fascial points.

The treatment technique consists of a deep friction applied to a precise, limited area (altered CC/CF) (Fig. 7.7.6). The pressure required varies according to the area treated, ranging from 35 to 75 N, with no apparent correlation to body mass index or age (Pedrelli et al. 2009b). The aim of treatment is to provoke a localized increase in temperature (see Chapter 7.18).

By limiting the area of treatment, the effects of manual pressure are considered to be deeper and more intense. The direction of the manipulation is also important. It varies from region to region, according to depth of fascia and fiber direction, and indications are given for positioning both patient and therapist to obtain the most effective treatment for each fascial point. Heat produced by localized friction could modify the extracellular matrix (Chen & Ingber 1999). Enhanced fluidity of extracellular matrix would alleviate tension on receptors embedded within the fascial layers. This may account for the sudden 'release' sensation perceived by therapists after an average of 3 minutes of friction, whereas subjects often report a simultaneous reduction in localized pain and a sensation of reduced

			Assesssme	ent chart for	r fascial ma	nipulation	
Name			Address			Date of birth	
Occupation			Sport			Diagnosis	
Sipa					PaMo		
PaConc					PaMo		
PaPrev					Examinations, X-rays		
Parasthesia CP			DI PE		Surgery/Factures/Visceral		
Posture	Posture						
HYPOTHESIS: Segments					Plane		
MoVe	Segment Sag		ttal plane	Frontal plane		Horizontal plane	CF
PaVe	Segment						
Treatm	ent				Results after 1 week		
1							

3

Fig. 7.7.5 • Assessment chart for the Fascial Manipulation. SiPa, site of the pain; PaMo, pain movement; PaConc, concomitant pain; PaPrev, previous pain; CP, symptoms regarding the head; DI, symptoms regarding the hand; PE, symptoms regarding the feet; MoVe, movement assessment; PaVe, palpation assessment; CF, centre of fusion.

3

Fascial manipulation



Fig. 7.7.6 • Treatment position of the center of coordination of RE–LU (over the fascia of the erector spinae muscles, at the level of the first lumbar vertebra) according to Fascial Manipulation technique.

pressure over the treated point. Reported changes in referred pain experienced during treatment of an altered CC could be due to the normalizing of tension along a myofascial sequence.

This type of treatment produces a localized inflammatory response. This reparative process could allow for deposition of new collagen fibers; however, only a correct tensional balance will guarantee that this occurs according to the physiological direction of movement (Gray 1995). Hence, to avoid relapses, treatment should always be aimed at recreating balance within the entire fascial system.

As Fascial Manipulation[®] is applied at a distance from the actual site of joint pain (CP), it has very few contraindications and this method can be applied safely even during the acute phase of a dysfunction.

Relative contraindications include fever, suspected fracture, or seriously debilitated general health. The most significant drawback is, perhaps, operator inexperience and/or inadequate comprehension of the method. Without a good understanding of all the implications and correlations, restoring a correct equilibrium to the fascial system is rather difficult.

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Managing dysfunctional scar tissue

Petra Valouchová Karel Lewit

History

Treatment of scars by local anesthesia was first introduced by lay healers, the brothers Huneke, as early as 1947 (Huneke F 1947; Huneke W 1953). They found that they were able to treat painful conditions such as shoulder pain by injecting a scar which was situated at a distance from the painful structure and without any obvious relationship to it. This was followed by what was known in Germany as Neuraltherapie, using local anesthesia for any site of irritation ('focus') that might possibly produce a reaction at a distance, for example, in a painful structure. The most prominent author of this trend was Gross (1972). Many of those who used local anesthesia could not fail to notice that using any substance (Kibler 1958) obtained similar effects; for example, Frost et al. (1980), using physiological saline, finally turned to acupuncture. In the end, the scar was almost forgotten. This may explain the lack of pertinent literature (Lewit & Olsanska 2004).

Traditional rheumatology deals mainly with inflammatory conditions, in the past called painful conditions of the soft tissues, 'soft tissue rheumatism,' using terms such as 'tendo-vaginitis,' 'fibromyalgia' or "myositis,' conditions for which no inflammatory origin could be proved (Reveille 1997).

The authors suggest that there is currently a lack of sufficient understanding of the physiology and pathophysiology of soft tissue, in particular in its relationship to the motor system. For the soft tissues to move in harmony with the motor system, they have to stretch and shift smoothly with every movement of the body – and this is also true of the visceral organs (Ward 1993). Whenever this intricate mobility is impaired, symptoms are bound to occur. However, the authors suggest that these very complex movements are very little understood, and accepted norms are sadly lacking.

The 'active scar' a model of soft tissue lesions

- Definition: A normal scar (recent or chronic) behaves like any other normal soft tissue; its layers stretch and shift in harmony with the rest of the tissues surrounding the motor system. A scar is considered to be 'active' if at least one of its layers does not move in harmony with the rest, i.e., if resistance to passive movement in at least one direction can be palpated. This also applies to visceral organs (Ward 1993).
- Diagnosis: For better or worse, palpation is essential in assessment of scars. Starting with the most superficial layers, examination of the skin is most convenient: in a hyperalgesic zone there is increased sweating and the stroking finger immediately feels increased resistance or drag, without causing the patient any discomfort (Lewit 1999). To diagnose one tissue layer after another, the barrier phenomenon is essential (Lewit & Olsanska 2004).

A useful definition of the barrier is that it represents the point where the first slight resistance to passive motion is perceived. This depends, of course, on the skill of the examiner. The normal barrier is met only gradually; it easily springs, whereas a pathological barrier is abrupt and barely springs (Fig. 7.8.1).



Fig. 7.8.1 • The barrier phenomenon. A–A, the anatomical barrier; Ph–Ph, the physiological barrier; Path, the pathological barrier; N_0 , the normal neutral point; N_1 , the shifted neutral point if a pathological barrier is present.

The most superficial layers of the skin can be stretched, and after reaching the barrier, springing can be easily felt (Fig. 7.8.2). Such springing is hardly felt by the patient, but if there is a pathological barrier, he or she may feel a slight pain, like a pin prick.

To examine subcutaneous tissue, a skin fold is formed, which is usually thicker in a hyperalgesic zone. This fold can normally be easily stretched (Fig. 7.8.3) but offers increased resistance to stretch in active scars. For treatment, it is suggested that the fold should only be stretched, not squeezed.



Fig. 7.8.2 • Skin stretch.



Fig. 7.8.3 • Stretching of a soft tissue fold.

To examine resistance at depth, for example, in relation to visceral organs, or shifting fascia, only pressure in the direction in which resistance is felt is used (Fig. 7.8.4). Here it can be useful to increase pressure where increased resistance is felt, so as to provoke pain for diagnostic reasons.

In an active scar, not all layers may be active. What has made the diagnosis more difficult is that in recent years surgeons have increasingly used laparoscopy and lasers, so that no scarring occurs at the surface. This is also true for pathological deliveries (without a Cesarean), and deep wounds. For cosmetic reasons, the surgeon may also cut the skin at a distance from the deeper intervention. The authors have found that pathological resistance is frequently identified not only in the abdominal cavity, but also below the symphysis, with symptom referral into the pelvis. It is important to note that active scars in the abdominal cavity and pelvis restrict back flexion, which the patient feels as low back pain. Experience suggests that this can be relieved by treatment of scars on the abdomen and/or below the symphysis (Kobesova et al. 2007).

- *Treatment*: Technically treatment is very similar to diagnosis (see Figs 7.8.1 and 7.8.4).
 - \circ The first step is to engage the barrier.
 - The second is: to wait (!). After a short latency period (of a few seconds), release occurs spontaneously, without increasing pressure, carefully avoiding painful reactions, especially in deep structures. Any forceful pressure only interferes with release, which is essential for successful treatment. Pressure has to be maintained until full release, i.e., normalization of the barrier occurs, which should be felt by the therapist. If this is achieved, subsequent palpation should be pain free.



Fig. 7.8.4 • Applied deep pressure.

Obtaining release in the course of treatment is essential for reasons of differential diagnosis, in particular when treating visceral organs. If no release can be felt, resistance is not caused by dysfunction, but by a pathological condition such as appendicitis, or a gynecological disease. This constitutes a 'red flag', which must be respected.

As stated previously, the entire diagnosis and therapeutic intervention relies on palpation, and can be achieved only by skilled hands. This, unfortunately, is considered by some to be 'subjective,' and therefore 'unscientific.'

For this reason, 13 patients with active abdominal scars (10 women and 3 men, mean age 45) and 13 healthy controls (10 women and 3 men, mean age 27) were examined by surface electromyography (SEMG). Eleven patients were post-appendectomy and two post-Cesarean section, with the active scar in one case on the right and in the other on the left. All patients complained of chronic low back pain with no signs of nerve root involvement. Symptoms of back pain usually increased after surgery.

SEMG of the straight abdominal muscles and the erectors spinae was carried out on both sides, with

the patients lifting the head and shoulders supine and prone. The surface electrodes were placed on both sides of the hypogastric section of the rectus abdominis, and on the erector spinae, at the thoracolumbar junction. The patients with active scars were examined before and immediately after scar treatment.

SEMG of the straight abdominal muscles showed increased activity on the side of the active scar in six, and on the opposite side in seven, cases, on lifting of the head with the shoulders supine. This asymmetry decreased significantly (p = 0.045) after scar treatment (Figs 7.8.5 and 7.8.6). When comparing side differences in activity of rectus abdominis between groups, the group of patients showed significantly higher asymmetry (p = 0.029) than the group of healthy controls (Valouchova & Lewit 2009).

• *Importance, incidence:* In the course of 13 months, 58 cases with active scar were seen by one of the authors, out of 476 patients examined during this period, i.e., 16.8%. In each case the relevance of the scar was tested. It is suggested that when an active scar is identified it is important for treatment of the scar to be followed by re-examination. It is common for most or at



Fig. 7.8.5 • The graph of mean SEMG activity of the low part of rectus abdominis before scar treatment by soft tissue techniques. Vertical axis SEMG amplitude in μ V,horizontal axis time interval of repeated movement in %, column graph Diff. asymmetry in %, mean value calculated from 5 repetitions in μ V, peak–peak value from 5 repetitions in μ V, rectus I. sin left rectus abdominis, rectus I. dx. right rectus abdominis.

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Fig. 7.8.6 • The graph of mean SEMG activity of rectus abdominis after scar treatment by soft tissue techniques. Vertical axis SEMG amplitude in μ V, horizontal axis time interval of repeated movement in %, column graph mean value calculated from 5 repetitions in μ V, peak–peak value from 5 repetitions in μ V, Diff. asymmetry in %, rectus I. sin. left rectus abdominis, rectus I. dx. right rectus abdominis.

least part of the symptoms to be found to have normalized or improved. As the conditions of most patients are multifactorial, therapeutic results are usually determined also by other factors, and the most impressive effects are achieved in those patients in whom the active scar is the primary cause of symptom production. Such patients are usually considered as having no apparent changes in the motor system, and to be suffering due to some undetermined cause. On the other hand, if an active scar remains undiagnosed and untreated, it can be a most frustrating cause of therapeutic failure.

 Pathophysiology: This is to a large extent unknown. We do not know why among innumerable entirely irrelevant scars, one becomes active. It can be a scar dating from early childhood, or might be the result of an operation or abnormal delivery decades earlier. In the authors' experience, if painful symptoms begin shortly after an operation, and never quite clear, an active scar should be considered. If, on the other hand, an old scar is active, it does not mean that it has necessarily been constantly active. Experience has shown that if, after treatment, a scar ceases being active for some time period, it can recur, usually under stressful conditions such as in the course of infectious disease or in psychologically stressful situations. The lack of scientific data is due to a complete lack of research in this field. What is certain is that soft tissue treatment of scar tissues, as described above, is usually highly effective and clinically very rewarding.

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Acupuncture as a fascia-oriented therapy

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Introduction

Historical background

Acupuncture has increasingly been used in Western medicine over the last three decades. It originated with traditional Chinese medicine (TCM) in the early Han period and has been described systematically for the first time in the medical compilation "Huangdi Neijing" (*Yellow Emperor's Inner Classic*), whose texts date from the Han period (200 BCE to 200 CE) (Zhu 2001). Acupuncture means in its Chinese translation *zhen jiu* "needling burning". However, before the development of steel needles, acupuncture consisted of skin irritation using sharp objects (e.g., stones), local warming at defined body sites, and minimal surgical interventions like blood letting.

Nowadays, acupuncture is defined as needling at anatomically defined sites of the body (acupuncture points) or sensitive spots (*ah shi* points) for therapeutic purposes including so-called moxibustion, i.e., heating or warming of the skin at acupuncture points with the help of burning mugwort (*Artemisia vulgaris*) (Fig. 7.9.1).

Acupuncture includes different techniques of needle stimulation, e.g., repetitive thrusting, twisting, rotating, or electrical stimulation to achieve different treatment effects according to the theoretical background.

There are different acupuncture related techniques such as laser acupuncture, injection in acupuncture points, and acupressure. A huge body of further manual or tool-assisted treatment approaches are based on the concept of acupuncture points and meridians. The theoretical background of acupuncture is based on Chinese, Confucian-legalistic, social and political philosophy of the first century CE. Medical acupuncture is based on the subjective aspects of disease, in contrast to the diagnostic and therapeutic understanding in western medicine, which is based on objective measurable pathologies. Acupuncture consists of systematic analogy expressed in the early concepts of yin and yang, qi and the internal organs, and results of detailed observations of nature and life.

Yin and yang

Originally the light and shadow side of a hill, yin and yang are the two opposites of a dual principle as a pattern of organization for the whole cosmos but also for the physiology and anatomy of organisms.

Qi

Qi expresses an energetic concept of vitality circulating in every body, in the beginning more likely as a living matter. It might be weak, blocked, accumulated, or misdistributed – all this aiming to describe different subjective symptoms.

Acupuncture points

Acupuncture points are the specific sites through which the qi of the meridians and zang fu organs (see below) is transported to the body surface. The Chinese characters for an acupuncture point mean, respectively, "transportation" and "hole".



Fig. 7.9.1 • Choice of acupuncture instruments: different kinds of needles and of mugwort. From Imich, 2008, with permission.

Meridians

All 360 classical acupuncture points lie strung together on the body surface according to a yin-yang pattern. They are arranged in three systems (front, back, and lateral aspects of the body). Qi is supposed to circulate within these meridians (Fig. 7.9.2).

Internal organs (zang fu)

The concept of organs is based on the principle of the Five Phases – correlating organ dysfunction to other physiological and psychoemotional conditions. This traditional concept transcends to a large extent anatomic and physiologic points of view. Organs and meridians are internally and externally connected.

TCM holds that there is normally a state of relative equilibrium between the human body and the external environment on the one hand, and among the internal organs within the body on the other hand; i.e., the equilibrium between protective and pathogenic influences. Pathogenesis may be caused by external (e.g., annual recurrence of hay fever, improper diet) or internal (e.g., emotions, overstrain) factors. The occurrence of any disease is, therefore, on the basis of the philosophic background, due to



Fig. 7.9.2 • Meridian system: front, back and lateral aspects of the body. From Irnich, 2008, with permission. a relative imbalance of yin and yang. This imbalance may result in different symptoms expressed, for example, as a stagnation of the flow of qi in channels on the body surface or internal organs. Regulation of yin and yang is therefore a fundamental principle in the clinical treatment. To restore health, acupuncturists insert and manipulate needles or heat the skin using moxibustion at prescribed acupuncture points to promote the flow of qi and blood so they can recirculate through the meridians or in the relevant organs.

Patients and the therapist himself may feel a so-called "deqi phenomenon" (needle sensation), which in the framework of TCM is achieved by needling the acupuncture point. This phenomenon can be felt as propagated sensation along the meridians and is described as sore, aching, numb, warm, or radiating. Some acupuncturists consider the eliciting of a deqi response to be a precondition for an effective treatment.

All these concepts described in the "Huangdi Neijing" are still the basis of traditional Chinese acupuncture, but underwent different interpretations and receptions in past centuries, resulting in many different schools of acupuncture today. Even if the traditional Chinese acupuncture system is not comprehensible to many western people, it is itself logical and thoughtful.

Today, needle acupuncture comprehends a broad range of approaches including traditional Chinese acupuncture, with different understanding and interpretation in its respective schools; treatment includes microsystem acupuncture (e.g., ear acupuncture [mostly developed in Europe], Yamamoto New Scalp Acupuncture), dry needling of myofascial trigger points, or acupuncture forms further developed in other countries (e.g., Korea, Germany, Japan, Russia, Taiwan, United States).

Physiologic background

Acupuncture effects are mediated through different neurophysiologic mechanisms: activation of mechano- and nociceptors, descending inhibitory pathways (comprising diffuse noxious inhibitory controls), or spinal and supraspinal modulation form some of the explanations to describe local and distant needling effects. Basic research showed the release of different neurotransmitters (e.g., norepinephrine (noradrenaline), serotonin), hormones (e.g., estrogen, cortisol), and peptides (e.g., endorphin) to be related to acupuncture treatment. Nevertheless, there is no single course of action that explains the complex neurophysiologic and anatomic responses to acupuncture treatment.

Acupuncture points have been supposed to be spots characterized by a high density of neural receptors. In addition, acupuncture points have been found to be situated next to vascular, nerve, and ligamentous sheets, despite there being more than 10 000 sheets in the superficial fascia of the human body, most of them not correlating with an acupuncture point. Studies of electrical properties of acupuncture points have shown that the electrical skin resistance at these points can be increased or decreased when compared to the surrounding skin area. None of those findings was able to define acupuncture points anatomically.

A remarkable observation to explain acupuncture points and meridians comes from myofascial referred pain that was observed to spread along the supposed meridian courses. Dorsher & Fleckenstein (2008a, 2008b) compared the anatomic correspondence of the "common" myofascial trigger point locations described in the Myofascial Trigger Point Manual to the locations of classical acupuncture points (Fig. 7.9.3). Anatomic correspondence of a common myofascial trigger point and a classical acupuncture point means those points are proximate and are demonstrated by acupuncture and anatomy references to enter the same muscle region. There is at least a 93.3% correspondence, if the distance between points on the skin is at most 3 cm; anyhow, points had to enter the same muscle region. At a maximum skin distance of 1 cm, 37% of points can still be found to correspond. There are marked clinical correspondences of both the pain indications (up to 97%) and somatovisceral indications (> 93%) of anatomically corresponding common myofascial trigger point-classical acupuncture point pairs (classical acupoints that are proximate to and enter the muscle region of their correlated common myofascial trigger points). The spread of degi along the meridians seems to be the same phenomenon as the physiologically analogous concept of referred pain arising from myofascial trigger points in the myofascial pain tradition. This provides a clinical line of evidence that myofascial trigger points and acupuncture points likely might describe the same physiologic phenomena.

These correlations make the explanation of the connecting meridians between acupuncture points more feasible. Speculation in this regard has

PART SEVEN Fascia-Oriented Therapies



Fig. 7.9.3 • Referred pain patterns of myofascial trigger points of the back and their correlation to the bladder meridian and its respective acupuncture points. From Irnich, 2008, with permission.

continued since acupuncture's earliest days as to whether acupuncture meridians are conceptual constructs or have an anatomic basis. Connective tissue might mediate acupuncture effects: Langevin (2002a) showed that rotation after needling activates fibroblast by mechanosensory transduction. These local effects can be tracked in distant connective tissue, too. Additionally, some researchers have described a degree of overlap of meridians and the peripheral nervous system in the extremities, whereas others have postulated that the meridians may exist in the myofascial layer of the body, reflecting perceived sensations by stimulating fascial structures. An interesting observation might be that anatomically derived myofascial meridians have distributions similar to those of acupuncture meridians described by TCM.

However, it remains clear that the target tissue of acupuncture points varies, comprising not only myofascial trigger points but also nerves, bones, ligaments, vessels, and the autonomic nervous system.

Techniques

Acupuncture has been increasingly used in western medicine in the last three decades. In Germany, about 30 000 medical doctors apply acupuncture at least occasionally. This chapter will point out an integrative and pragmatic principle of acupuncture treatment, especially for diseases of the locomotor system. It has been systematically used and evaluated in large trials by medical acupuncturists of the Medical School of the Ludwig-Maximilians University of Munich and other members of the German Medical Association for Acupuncture, the oldest and largest medical acupuncture society of the western world (Irnich et al. 2001, 2002).

The applicable treatment spots range from painful points, meridians, and classical points, to extra-points microsystems and the traditional concept of organ diseases, zangfu. Treatment techniques, amongst others, might be needling, cupping, and massage techniques. Chinese medicine also comprises herbal medicine and Qi Gong.

We present the basic principle of treatment for diseases of the locomotor system, comprising five steps:

Start with distant points and/or microsystem points

Classical acupuncture points and microsystem points can have distant effects. Distant points are chosen to achieve immediate pain relief and improve range of motion. Their choice is based on the location of the acupuncture points in the meridian system. If the course of the meridian meets the affected body region its distant points may be used to release the symptoms. Microsystems meet the definition of distant treatment area even if the microsystem is located next to the affected area. Microsystem points are chosen according to their propagated projection



Fig. 7.9.4 • Analogies of different microsystems. Analogous points to treat neck pain. Preferable treatment area is the most sensitive microsystem. From Irnich, 2008, with permission.

zones (Fig. 7.9.4). Distant points and microsystems are known to soften tenderness and might especially be indicated in the first contact with the patient or highly acute conditions of the disease, as they are far away from the tender spots (e.g., SI 3 is known to release pain syndromes of head, neck, and shoulder; Table 7.9.1).

Table 7.9.1 Most effective distant points						
Frontal headache	ST 44, ST 36, LI 4					
Lateral headache	LIV 3, GB 41, TW 3 or 5					
Occipital headache	BL 60 or 62, SI 3					
Neck pain	BL 60 or 62, SI 3, Ex-UE 8					
Shoulder	ST 38					
Elbow	Microsystem ear					
Low back pain	BL 40, NP 67, Ex-UE 7, YNSA D-Zone					
Knee	Microsystem ear, extraoral incisors (mouth)					

Look for tender regional/ segmental points

Needling of painful spots (ah shi points) is an ancient concept of TCM. It only requires the localization of painful spots where acupuncture needles are inserted, taking into account anatomical and physiological knowledge of the underlying structures. The choice of segmental points has been described by traditional Chinese acupuncture (i.e., shu points).

Treat myofascial trigger points

Trigger point acupuncture might not only be applied for myofascial trigger points – other structures (myofascial, cutaneous, ligamentous, osseous) are also known to represent trigger points which can be treated similarly, with the difference that local twitch responses may not always occur, due to their nature. For further description, please see below.

Supplement with local meridian points or ah shi points

In case treatment of distant and regional points or myofascial trigger points does not produce results, local acupuncture or tender points could be added to the selected treatment points. Treatment techniques range from achieving deqi sensations penetrating acupuncture points to superficial manipulation aiming to evoke reflectory twitching of superficial muscular layers.

Treat internal organs in chronic diseases

Functional diseases, especially, require comprehensive approaches to understand the patient's situation, comprising biological, mental, social, or spiritual aspects of well-being. According to the traditional concepts (zang fu), illness is understood as an imbalance of physical and mental vitality (qi). The zang fu approach integrates the symptoms, needs, and worries of the patient, and surrounding factors, and aims to develop a longlasting, individualized treatment concept using different kinds of traditional Chinese philosophies and techniques. Acupuncture points, especially, will be chosen according to patterns following rules such as yin and yang in order to balance the patient.

Dry needling

In the following section we present a special needling technique applied in the treatment of myofascial disorders - myofascial trigger point acupuncture (named dry needling). Dry needling is an invasive procedure in which an acupuncture needle is inserted into the skin and muscle. As the name implies, it is directed at myofascial trigger points. Dry needling does not require fundamental knowledge of TCM and acupuncture, but technical skills are necessary. In addition, clinical experience implies that dry needling integrated in a classical acupuncture treatment is beneficial for the patient's outcome; therefore both techniques may be combined. This chapter gives practical instruction for fascia treatment. To understand the whole system of TCM comprising acupuncture, meridians, point selection, etc., we suggest the respective textbooks.

Once the skin is prepared and the myofascial trigger point is identified, the overlying skin is grasped or fixed between the thumb and index finger or between the index and middle finger. There are three different approaches to perform dry needling.

Direct dry needling

The needle is inserted approximately 1 to 1.5 cm away from the myofascial trigger point to facilitate the advancement of the needle into the myofascial trigger point. The grasping fingers isolate the taut band and prevent it from rolling out of the trajectory of the needle. The aim is to target the myofascial trigger point and to elicit a local twitch response, a visible or palpable quick twitch of the taut band under the fingertip, while examining the skin above the muscle fibers for this characteristic short and rapid movement (Fig. 7.9.5). The focus for the therapist is the tip of the needle and the texture of the surrounding tissue (fascia, muscle, connective tissue). The trigger point might be felt rubber-like at the tip of the needle. The local twitch response has been shown to predict the effectiveness of the myofascial trigger point needling. A quick removal of the needle outside the muscle is recommended to avoid the needle damaging the muscle fibers or the surrounding tissue, as shearing forces and tension could occur during the contraction of the muscle whilst twitching. The needle may be withdrawn to the level of the superficial fascia without exiting, and it should be redirected to the myofascial trigger point to repeat the process. The process of



Fig. 7.9.5 • Scheme demonstrating the exact targeting of the myofascial trigger point in order to elicit a local twitch response. From Irnich, 2008, with permission.

entering the myofascial trigger point and eliciting local twitch responses should proceed, attempting to extinguish the twitch response of the myofascial trigger points and to contact as many sensitive loci as possible. The grasping finger remains in position till the end of the treatment; besides marking the myofascial trigger point spot, it represents a diagnostic instrument to feel (a) the vegetative response of the patient such as (b) smaller twitch responses of superficial muscle layers.

Dry needling of the muscular fascia

Local twitch responses may be irritating the outer fascia of the corresponding muscle. The needle only touches the fascia, without penetrating and exploring the myofascial trigger point. This technique is especially used when needling larger superficial muscle layers (e.g., M. trapezius, M. levator scapulae). The needle is inserted approximately 1 to 1.5 cm away from the myofascial trigger point to facilitate the advancement of the needle in the direction of the muscle fascia and is manipulated at high frequency to elicit a local twitch response (Fig. 7.9.6).

Superficial dry needling

This special technique only requires insertion of the needle into the connective tissue above the myofascial trigger point. The needle will be removed after



approximately 30 seconds; meanwhile, detonization of the myofascial trigger point is proven by palpation. Treatment can be repeated with longer needle-in intervals. The aim of this procedure is not to release a local twitch response but indirect (e.g., neuronal) resolution of the myofascial trigger point (Fig. 7.9.7).

The local twitch response has been shown to predict the effectiveness of myofascial trigger point needling. Patients' responses regarding local twitch responses range from immediate relief to no effect if irrelevant myofascial trigger points are treated, or additional trigger points sustain pain and restricted motion. Twitch responses after needling normally appear as short and dull-pulling feelings comparable to a muscular fasciculation. This sensation might continue being apparent after treatment in the form of aching muscles or a post-treatment soreness, both therapeutically reasonable reactions (e.g., due to abnormal posture prior to the treatment). An integral part of myofascial trigger point therapy is therefore postprocedural stretching. After dry needling, the muscle group that was treated should undergo a stretch.

The choice of an appropriate acupuncture needle, such as for the depth of the muscular region aimed for, depends on the skills of the practitioner. In contrast to injection needles, acupuncture needles possess an atraumatic sharpening: instead of cutting they slide into the tissue, thus reducing the risk of surrounding hematoma. The appropriate choice of needle equates with the principle "as thin as possible and as long as necessary". Guiding tubes have become very helpful in the handling of thin needles. Patients sensitive to pain might more likely tolerate those needles; as the pressure of the tubes on the patient's skin irritates mechanosensitive afferent fibers, their noxious input whilst needling might be reduced, thus reducing patients' sensations, stress, and defense.



Fig. 7.9.7 \bullet Superficial dry needling. From Irnich, 2008, with permission.

The same effect might be released by triggering the skin with palpation of the fingers.

Evidence

There is a huge body of scientific literature showing the physiological and clinical effects of acupuncture. Regarding anesthesia, supportive acupuncture treatment is performed in postoperative pain, anxiolyses, and postoperative nausea and vomiting, based on promising results of rigorous randomized trials. However, there are many unsolved questions (e.g., specifity of theconcepts, indications, optimum dose).

Although clinical trials give evidence of the analgetic effects of acupuncture, the results of several meta-analyses could not provide a conclusive statement regarding the effectiveness of acupuncture in myofascial disorders: (a) Two meta-analyses suggest that acupuncture could be a valuable nonpharmacological tool in the short-term treatment of patients with neck pain or with frequent tensiontype headaches, commonly related to myofascial trigger points in head and neck muscles. Fu et al. (2009) recommend further studies that also address the long-term efficacy of acupuncture for neck pain. Irnich et al. (2002) could demonstrate that dry needling and acupuncture at distant points could cause immediate effects improving patients' range of motion but only distant points could achieve

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immediate pain relief in chronic neck pain. Another large scale trial from the same authors showed the superiority of acupuncture versus massage in relieving the restriction of motion (Irnich et al. 2001). A subgroup analysis showed that it was especially patients with neck pain associated to myofascial disorders that benefited from acupuncture treatment.

(b) A Cochrane review analysing acupuncture in shoulder pain reports short-term benefits with respect to pain and function; hence, due to the small number of clinical and methodologically diverse trials, there is little evidence to support or refute the use of acupuncture for shoulder pain. There is a need for further well-designed clinical trials.

(c) Other meta-analyses focus on the treatment of low back pain. Clinicians sometimes suggest that acupuncture and dry needling may be useful adjuncts to other therapies for chronic low back pain. Because most of the studies are of lower methodological quality, there certainly is a need for further higher quality trials in this area. It has not yet been proven that acupuncture could be superior to other active therapies.

When compared to myofascial trigger point injection, which has been suggested to be a promising therapy, it remains in doubt whether dry (i.e., acupuncture) or wet (i.e., injection) needling is therapeutically superior.

Summary

The term acupuncture comprises a broad range of different treatment approaches. Their basis can be found in the old textbooks of TCM. Acupuncture, and especially dry needling, are promising treatment options in the therapy of myofascial trigger points and related disorders when compared to other tool-assisted approaches but also in comparison to manual therapies. A good relationship between patient and acupuncturist based on empathy and understanding, as well as anatomical and therapeutic skills, are requisites for successful treatment.

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Gua sha

Arya Nielsen

Introduction

Gua sha is an essential modality of traditional East Asian medicine (TEAM), defined as instrumentassisted unidirectional press-stroking of the body surface that intentionally creates transitory therapeutic petechiae representing extravasation of blood in the subcutis. Gua sha has been used for centuries in Asia, and Asian immigrant communities, as a form of self or familial care in the home (Hautman 1987; Craig 2002), and by acupuncturists and practitioners of TEAM worldwide in clinical practice (Nielsen 1995; Zhang & Hao 2000).¹ With the advance of TEAM outside of Asia, Gua sha has been used over broad geographic areas, in multiple cultural settings, and by millions of people.

This chapter explains Gua sha's relevant terms, indications and contraindications, physiological relationship to connective tissue, and recommendations for safe practice.

Gua sha terms

Gua sha consists of repeated unidirectional pressstroking with a smooth-edged tool at a lubricated area of the body until sha petechiae appear 7.10

(Fig. 7.10.1). Gua sha can be applied with a simple metal cap with a round lipped edge. Traditional tools include a soup spoon, coin, honed horn, bone, jade, or stone (Fig. 7.10.2).

Unidirectional 'press-stroking' accurately describes the operation of Gua sha, though a literal translation of 'gua' from Chinese is to 'scrape' or 'scratch' (So 1987). While scraping implies abrasion to the surface, the skin remains intact; there is no abrasion or bruising, merely petechiae and ecchymosis beneath intact skin.

The term 'sha' has several meanings. Sha describes surface 'blood stasis' in a symptomatic or presymptomatic state. Sha also describes the petechiae raised from Gua sha. The literal translation of 'sha' from Chinese is 'sand, sharkskin, or red, raised, millet-size rash.' The fresh petechiae raised from Gua sha immediately begin to fade and blend to ecchymosis. See Figures 7.10.3 and 7.10.4.

Sha is also translated as cholera, wherein sha blemishes resemble cholera's end-stage rash. Gua sha in the East, like frictioning in early Western medicine (Jackson 1806), was used in the treatment of cholera and cholera-like disorders (So 1987), mimicking the crisis stage of the illness to produce a cure (Nielsen 1996).

Common translations of Gua sha in Western medical literature include 'coining,' 'scraping,' 'spooning,' 'cao gio' (Vietnamese) and 'kerik' (Indonesian) (Nielsen 2009).

¹ Recent adaptations of Gua sha have appeared in the form of Graston Technique and ASTYM.



Fig. 7.10.1 • A smooth-edged instrument is moved over the surface of the skin as it is pressed into the flesh. (A) pressstroking right to left; (B) press-stroking left to right) producing petechiae and ecchymosis. These simple metal caps have a smooth rounded lip. Nielsen 2009.



Fig. 7.10.2 • Gua sha tools: soup spoon, honed pieces of water buffalo horn and simple evenly round-lipped metal caps. The warming, cooling and/or neutral lubricants from Badger Balm (www. badgerbalm.com) are shown at the top. Nielsen A.



Fig. 7.10.3 • Fresh sha petechiae and ecchymosis on the back of a patient treated for chill and aversion to cold with knee pain and swelling. Nielsen 1995.



Fig. 7.10.4 • Picture taken the day after Gua sha treatment showing the fading of ecchymosis. Nielsen A.

Gua sha

How to Gua Sha

Box 7.10.1

How to Gua sha

- The area to be treated is first palpated to confirm the presence of sha stasis. A lubricant is applied, often oil based.
- Gua sha treatment begins at the most medial part of the ROI (region of interest), press-stroking into the flesh with a smooth round-edged instrument hard enough to 'contact' the fascial layer (see Fig. 7.10.1), but not so hard as to cause pain or discomfort.
- A stroke line is typically 4-6 inches long.
- Press-stroking is unidirectional at a stroke line, not back and forth.
- Closely timed press-stroking continues uninterrupted until sha petechiae are raised on a stroke line, typically 8–12 strokes.
- Stroking stops when all the sha is expressed as petechiae at the stroke line, before producing ecchymoses, which will occur naturally as petechiae begin to fade.
- Gua sha is continued at the next stroke line directly adjacent to the one before. This goes on until ROI is covered in a homogeneous fashion, avoiding the appearance of stripes that represent incomplete technique.

Indications

Gua sha is indicated for pain, problems with movement or range of motion, and/or disturbed organ or system function, including acute infectious or chronic illness (Nielsen 1995; Zhang & Hao 2000). In TEAM, pain represents stasis, reflected in the Chinese aphorism: 'No free-flow: pain; free flow: no pain' (Nielsen 1995). Sha is a form of blood stasis in the surface tissue where pain is fixed, persistent or recurring.

Pressing palpation confirms sha stasis when blanching appears that is slow to fade (So 1987), indicating normal surface perfusion is hindered. (See Plates 7.10.1a, b and c.)

The patient may notice an achy sensation during palpation that may ramify to other areas of the body. Sha may also be accompanied by fatigue. In TEAM, this stasis in the surface tissue is thought to express from, and to, internal organ or system function (Epler 1980; Lu & Needham 1980).

The benefits of Gua sha are commonly felt immediately and are sustained, to some degree, over time, where repeating treatment may be indicated to reach maximum benefit. The recovery of the tissue is expressed not only by immediate improvement in pain status, but by changes in pulse, tongue, digestion, urine, stool, sleep, libido, flexibility, mood as well as other presenting symptoms (Kaptchuk 2000).

A 2005 search of the Chinese medical literature database from 1984 to 2004 identified 120 studies using Gua sha for painful musculoskeletal conditions, as well as acute infectious illness, respiratory conditions, and autoimmune and inflammatory disorders (Nielsen 2009). A 2011 literature search found over 600 articles and studies on Gua sha in the Chinese medicine database in areas of internal medicine, surgery, gynecology, pediatrics, pain management and general medicine (Nielsen 2012).

There are randomized controlled trials in the Western literature for the effective use of Gua sha for neck pain (Braun et al. 2011) and breast engorgement (Chiu et al. 2010) as well as case reports of Gua sha in treatment of migraine headache (Schwickert et al. 2007), post herpetic neuralgia (Nielsen 2005). Chan et al. (2011) report a case where one Gua sha treatment reduced elevated liver enzymes and chronic inflammation in a patient with chronic active hepatitis B.

Contraindications

Gua sha is contraindicated where the dermis or flesh is injured or compromised as in sunburn, abrasion, rash or contusion (Nielsen 1995). In cases of injury, Gua sha may be applied, but not at the area of trauma.

Gua sha is:

- not contraindicated for patients who are weak or menstruating
- not contraindicated for pregnant women if over limited areas and can be indicated where other medicine is unsafe as in sinusitis, colds and cough, for headache, neck, shoulder, back and hip pain, and for sciatica (author's experience)
- not contraindicated in patients with a stable INR (international normalized ratio) who take anticoagulation medication because the capillary bed is not damaged with Gua sha (Nielsen et al. 2007).²

² The author (AN) has used Gua sha on hundreds of patients taking anticoagulation medication. See *Gua sha Step-by-Step: A Teaching Video* (www.guasha.com).

Biomechanism/physiology

Observation

What a provider observes when applying Gua sha is a gradual expression of small red petechiae (that can sometimes be brown, blue, very deep red or nearly black). The patient often feels exhilarated, invigorated, even excited. Acute pain is immediately affected, sometimes completely resolved. Nausea and vomiting cease (So 1987), wheezing and shortness of breath lessen or completely resolve; other acute symptoms are mitigated or resolve completely (Nielsen 1995). It would appear that it is the closely timed repeated press-stroking, the unidirectionality and the intentional extravasation of petechiae and their resolution over time that have physiological significance.

Research

Specific research contributes to a partial and increasing knowledge of Gua sha's therapeutic physiology, in that:

- Gua sha increases surface microperfusion 400% at a treated area but not outside the area treated (Nielsen et al. 2007)
- Gua sha immediately reduces pain local to and distal to a treated area (Nielsen et al. 2007)
- Gua sha upregulates antioxidant, cytoprotective heme-oxygenase-1 (HO-1) gene expression in multiple internal organs immediately after treatment and over a period of days (Kwong et al. 2009). Increase in HO-1 from Gua sha is thought to be responsible for the anti inflammatory hepatoprotection seen in the treatment of chronic active hepatitis B (Chan et al. 2011).

Gua sha and connective tissue

Painful conditions or illness may be accompanied by altered or inflamed connective tissue and are observed to respond to manual therapies, including Gua sha, although the biomechanism is not completely understood (Corey et al. 2009). Transduction of force and stretch are thought to cause connective tissue innervations and restoration (Iatridis et al. 2003; Standley & Meltzer 2008; Corey et al. 2009) but only to specific kinds of connective tissue, discussed below. Chaudhry et al. (2008) show that palpable sensations of physical release reported by manual therapists cannot be due to 'deformations produced' in the firm or dense fascial tissue.

There are at least three characteristics that distinguish Gua sha from other manual therapies that involve pressure or fascial stretch. Gua sha is characterized by (1) closely repeated unidirectional stroking that intentionally presses into the fascia; (2) application predominantly along a muscle and specifically not oscillating or across muscle tissue; and (3) intentionally creating transitory petechiae and ecchymosis. In fact, production of petechiae and ecchymosis necessitates closely timed repeated press-stroking that is unidirectional.

Connective tissue may respond to directionality as it does to tensile loading with collagen strands in parallel arrangement along the direction of the loads imposed (Langevin & Huijing 2009), as in dense 'regular' connective tissue. Interestingly, unidirectional acupuncture needle rotation produces more torque in the connective tissue and necessitates greater withdrawal force than bidirectional needle rotation, that is also connective tissue responsive, but dose-dependent (Langevin et al. 2007). What effect repeated unidirectional mechanics has on dense or non-dense connective tissue, or how the effect is transferred throughout the system, is modeled based on recent connective tissue research, discussed below.

Models

The most current model of therapeutic effect focuses on unspecialized, 'loose', non-dense connective tissue that forms an anatomical network throughout the body that is intimately associated with all other tissues, including organ systems (Langevin 2006). Electrical, cellular and tissue remodeling signals in the connective tissue are responsive to mechanical forces. These generate dynamic evolving patterns that interact with one another as a 'body-wide mechanosensitive signaling network' that influences. and is influenced by, function that is normal, pathological (Langevin et al. 2001; Langevin 2006) and, by inference, responsive to counteractive intervention. Iatridis et al. (2003) suggest that 'loose connective tissues may function to transmit mechanical signals to and from the abundant fibroblasts, immune, vascular, and neural cells present within tissues.'

Just such a conductive physiology is the functional paradigm of the organ and channel system of TEAM

Gua sha



Technique for Modern Practice. Edinburgh: Churchill Livingstone; 1995.

that evolved over a 2000-year history to understand illness and inform treatment, including acupuncture and Gua sha (So 1987; O'Connor & Bensky 1981). The system is based on the concept of 'Qi' that is defined as form and function (Kaptchuk 2000) and therefore inaccurately referenced as mere 'energy'. Ancient texts describe '3 qi' as 'steaming' at the 'cou li' (Epler 1980), which is translated as 'lining', and also as 'pores' because of the lining's ability to regulate: to open and close (Epler 1980; Unschuld 1986) (Fig. 7.10.5). The 'wei' (protective) qi emanates at the yellow greasy membrane corresponding to the fatty layer of the 'li'; the 'ying' (nourishing) gi courses through and nourishes the entire body and is activated when an acupuncture needle is inserted and rotated (Epler 1980).

TEAM was the first to propose this 'lining' as an actual organ, the San Jiao or Triple Burner (Huang 1972; Unschuld 1986); the only organ that lacked a recognized Western analogous structure, until the relatively recent focus on connective tissue. The San Jiao governs the upper, middle, and lower body or 'burners,' linking the 'exterior with the interior' the outer flesh to the organs via the channels that reside in the 'li' (Epler 1980; Unschuld 1986).

Qi moves vertically through the 'Jing' or main channels, and horizontally through the 'Lo' channels that connect the main channels to each other and to the interior tissue and organs through the 'cou li' lining (connective tissue). Langevin and Yandow (2002) have shown that most common acupuncture points exist at cleavage concentrations of connective tissue within and along meridian/fascial layers, suggesting that activation at these sites would, in fact, augment a connective tissue response. Insertion of a needle off site from an acupuncture point might activate a response but perhaps lesser than a known point. This is borne out by studies comparing acupuncture to control points, where control points demonstrate some therapeutic effect (Haake et al. 2007).³

One theory of mechanotransduction in skeletal muscles involves nitric oxide release initiating smooth muscle relaxation and vasodilatation (Hocking et al. 2008). Nitric oxide (NO) is an important mediator in both health and disease. It is an endogenous mediator of vasodilatation, also having effects on platelet function, inflammation, and pain perception (Mackenzie et al. 2008). In preclinical studies, NO was shown to help maintain gastric mucosal integrity, to inhibit leukocyte adherence to the endothelium, and to repair NSAID-induced damage, thus having a protective effect on the GI tract (Lanas 2008). NO-based intervention might produce substantial pain relief by increasing circulation, decreasing nerve irritation, and decreasing inflammation (Hancock & Riegger-Krugh 2008). Release of NO in the process of increased perfusion and vasodilation may account for the immediate pain relief experienced with Gua sha.

Endothelin-1 (ET-1) and endothelial constitutive nitric oxide synthase (ecNOS) mRNA expression have been shown to be time and mechanical force dependent (Ziegler et al. 1998a). Specifically, the effect of unidirectional force or stress differs from oscillating or alternating force or stress in vascular endothelium (Ziegler et al. 1998b). Gua sha is always applied with unidirectional stroking.

Moreover, circulation is predominantly unidirectional throughout the system, while capillary beds



³ The author's personal theory as to how acupuncture persisted for 2,000 years: the provider did not have to be that good in terms of precise point location. Some effect was seen. If adept in point location, a better effect was elicited.

have at least some bidirectional interaction with the surrounding tissue. Since unidirectional needle mechanics produce unique fibril activity in the connective tissue, it might be that for certain conditions the direction and kind of mechanical intervention is specific, that unidirectional press stroking force would invigorate blood flow and fascial mechanics differently than oscillating press force. In fact, Standley and Meltzer (2008) show that anti-inflammatory cytokine secretion is activated by directionality of myofascial release: pressure and shear that create uni-axial fibroblast strain can account for improved range of motion (ROM), decreased edema, and reduced analgesic requirements as well as for 'long-term benefits, despite short-term treatment.'

Safety

The most significant and consistent complication reported in the Western medical literature for Gua sha is the misattribution of the transitory therapeutic petechiae and ecchymosis as a burn, bruise, or dermatitis caused by abuse, battery or torture (Nielsen 2009). Physicians may still be taught to discourage use of Gua sha as 'harmful' and 'unacceptable in our culture' (Botash 2009). While not only untrue, such bias may discourage immigrant families from integrating conventional medical care with their traditional practices. For this reason, it is strongly recommended that a handout be given to patients explaining the technique, describing the normal presence of therapeutic petechiae and ecchymosis, and providing the clinician's contact information (www. guasha.com).

Close examination of reports of serious complications reveals a lack of convincing evidence, and a number of such reports are clearly invalid (Nielsen 2009):

- A report of microhematuria in an infant following Gua sha did not rule out infectious illness as causative (Longmire & Broom 1987)
- A brain bleed in a female patient was attributed to Gua sha treatment, despite symptoms and condition preceding Gua sha (Ponder & Lehman 1994)
- Burns caused by fire cupping were misattributed to Gua sha (Amshel & Caruso 2000)
- Camphor toxicity from liniments has been reported and disputed (Schneir & Clark 2002).

It is also the case that some actual complications may not be widely recognized or may not have been reported yet in the literature. Gua sha instruments are commonly used on different patients. In certain cases, after repeated press-stroking, the lubricant on the instrument can take on a pinkish tone, suggesting that extravasated blood cells may cross the skin surface, with potential risk of exposure to bloodborne pathogens.

Preventing risk of exposure to bloodborne pathogens can be accomplished by:

- Gloving both hands prior to and during Gua sha procedure
- Using disposable press-stroking devices, i.e., a single-use rolled-edged metal cap
- Decontaminating and autoclaving devices intended for multiple use
- Decanting lubricant into disposable treatmentsized containers to prevent cross-contamination, or using lotion from a pump dispenser as lubricant
- Following safe sequencing of palpation, gloving, needling, use of lubricant, application of procedure and clean-up.

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Prolotherapy

Manuel F Cusi

Introduction

Prolotherapy is an injection therapy used to treat chronic ligament, joint, capsule, fascial, and tendinous injuries. The goal of this treatment is to stimulate proliferation of collagen at the fibro-osseous junctions to promote nonsurgical soft tissue repair and to relieve pain (see Fig 7.11.1) (Klein & Eck 1997). Originally defined by Hackett as "the rehabilitation of an incompetent structure (ligament or tendon) by the generation of new cellular tissue", it has received a variety of names (Dagenais et al. 2005; Alderman 2007).

It now includes all regenerative methods by injection including dextrose-based, inflammation-based, plateletrich plasma, (adult) stem cell-based, and essentially any other injection method in which either growth factors are stimulated or disrepair factors are blocked.

Growth factors are powerful polypeptides that induce wide-ranging effects including cell migration, proliferation, and protein synthesis. These proteins may be produced by the affected cells or in other cells. These growth factors must avoid the binding proteins which could cause their inactivation, find their way to the area needing growth, and hook onto an appropriate receptor protein (Reeves 2000).

Prolotherapy has been used extensively in the USA since the 1930s (over 450 000 people have undergone prolotherapy treatment) and in other countries around the world. Yet it has not become a mainstream therapy (Mooney 2003). The abundance of case series studies and anecdotal evidence has not been supported by a large body of randomized controlled trials (Yelland et al. 2003; Dagenais et al. 2007).

History

Hippocrates (trans by Francis Adams 1946) (460-370 BCE) was the first to describe the intentional provocation of scar tissue formation by searing the shoulder capsules in the unstable shoulders of javelin throwers in Sparta. Two millennia later, in 1837, Robert Valpeau of Paris described the use of scar formation for the repair of hernias. One hundred years later, Yeomans (1939) extensively reviewed the genealogy of herniology and a variety of vein sclerosis techniques. Gedney (1937) applied these injection techniques to joints, the first being the sacroiliac joint. He maintained the term sclerotherapy, which remained in use until the 1950s. In that same year, Schultz (1937) described, in the Journal of the American Medical Association, a treatment for subluxation of the temporomandibular joint.

In the mid 1950s, George Hackett published a number of articles based on his more than 20 years' experience, culminating in his book *Ligament and Tendon Relaxation Treated by Prolotherapy* (Hackett 1956), where he claimed an 82% cure rate in a population of 1600 people with back pain (Hackett & Huang 1961). In 1983, Liu confirmed experimentally increases in ligament junction strength and diameter of collagen fibrils.

In 1995, prolotherapy was renamed by some as RIT (regenerative injection therapy), or "the injection of growth factor production stimulants to promote regeneration of normal cells and tissue" (Linetsky & Manchikanti 2005; Reeves et al. 2008).



Fig. 7.11.1 • CT scan of prolotherapy in the dorsal interosseous ligament of the sacroiliac joint. Radio-opaque contrast can be added to demonstrate correct placement of the hypertonic dextrose solution and local anesthetic. Courtesy of H. Van der Wall, and L. Wong.

Wound healing, repair and regeneration

An idea of tissue healing and repair is necessary to understand better the effects of prolotherapy.

Wound healing and repair of injured tissues follows three stages (inflammation, matrix deposition, and remodeling) in healthy individuals (Hildebrand et al. 2005). Wound healing generally leads to repair, and in many cases allows return to at least partial function of the injured tissue, but not to tissue regeneration. The repair process leads to a loss of function as a result of scar tissue formation. This is an important factor when dealing with connective tissue that functions in a mechanically active environment. The repair of connective tissue by scar formation - ultimately healing by second intention may restore connective tissue to its pre-injury length, but will not provide adequate (pre-injury) tensile strength in ligaments and tendons (Reeves 2000; Linetsky & Manchikanti 2005).

The inflammatory phase

Following an acute overt injury there is pain and bleeding. The latter is repaired with the formation of a fibrin clot, which prevents further bleeding and provides a provisional matrix for migrating cells. Blood clotting also releases pro-inflammatory substances, including components of the clotting cascade, cytokines and growth factors released from other cells such as platelets. This contributes to the subsequent migration, localization, and proliferation of other cells (mesenchymal cells, fibroblast-like cells, etc.), which sets the stage for the second phase of the repair process.

The matrix deposition phase

Once the fibrin clot is consolidated and the influx of a subset of cells and deposition of these new cells is established, the deposition of matrix molecules can begin. The purpose of matrix formation is to bridge the damaged area with residual ligamentous tissue. Two points need to be considered:

- **1.** The tissue deposited attempts to bridge the injured area, regardless of the tissue structure it attempts to gap or repair.
- **2.** The changes in the matrix deposited early in the process lead to the organization of deposited matrix that is different from normal tissue. In mechanically active tissues this will result in a severe compromise of the tensile strength of the new tissue formed: scar tissue is not as strong as the original ligamentous tissue.

In addition to matrix deposition in the early stage of healing, there is an increase in cellularity and in vascularity (Bray et al. 1996) due to the release of angiogenic factors in the early postinjury stages. Increased vascularity generates the influx of new micro-vessels. Those connective tissues with endogenous microvasculature will heal well, whereas those that are poorly vascularized (e.g., menisci) do not heal well. In the early stages of scar tissue formation there are almost no neural elements, and therefore minimal regulation of fibroblast-like cells or microvasculature.

The remodeling phase

This is a much slower process, which involves not only alterations in the remodeling of the existing matrix but also gene expression, cellularity, vascularity, and innervation (Hildebrand et al. 2005). The material deposited early is reorganized to suit the mechanical demands of the injured tissue. In the case of ligament, the organization of fibrils becomes oriented towards the axis of the ligament, whereas in other tissues such as skin, a more

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basket-weave arrangement takes place, which provides strength in multiple directions. This process may take months or even years, and the composition of the repaired tissue changes with time, as does the gene expression phenotype.

The advent and use of erythrocyte growth factor (erythropoietin) to stimulate red cell proliferation in patients with chronic anemia and even in preparation for acute blood loss in surgery has led to the study of growth factors and their effect in both musculoskeletal medicine and other areas outside medicine, such as endurance sports.

Improved understanding of the role of growth factors in tissue regeneration and healing is compatible with the traditional inflammatory reaction theory and takes it to a new dimension. Prolotherapy now includes the injection of external growth factors – blood, platelet rich plasma (PRP), adult stem cells, or the injection of growth factor stimulators (traditional prolotherapy solutions).

The application of growth factors to stimulate cell proliferation and extracellular matrix synthesis in tendinopathy has been described by Wang and colleagues (2006) and can represent a new look at the mechanism of action of prolotherapy solutions. The transplantation of mesenchymal stem cells into injured tendons has been shown to promote tendon healing in laboratory animal models (Smith & Webbon 2005). The injection of growth factors should produce structural changes in the tissues injected, and these changes result in improved mechanical quality and function. These changes have not been conclusively proven to date, but the possible role of growth factors represents an exciting development pathway. Further systematical study of the topic is required before definite statements can be made.

Mechanism of action and substances injected

Two types of substance are used in prolotherapy. The first is injection of growth factor-containing substances. Examples of this include injection of blood, and injection of mass-produced recombinant growth factors, PRP and mesenchymal stem cells. The second method is stimulation of growth factor production, in which the injected solution initiates production of growth factors (dextrose, inflammatory agents that initiate an inflammatory cascade to produce growth factors), and plasmid DNA (Reeves 2000).

In the classical understanding of the inflammatory reaction theory, there are four types of solutions, grouped according to the suspected mechanism of action (Banks 1991):

- 1. Osmotic (e.g., hypertonic dextrose) solutions are thought to provoke cell dehydration, with subsequent cell lysis, release of cellular fragments, which in turn attract granulocytes and macrophages. In addition dextrose could cause glycosylation of cellular proteins.
- **2.** Irritants (e.g., phenol) have a phenolic hydroxyl group that is believed to alkylate surface proteins; these either become antigenic or are damaged, and in turn attract granulocytes and macrophages.
- **3.** Chemotactics (e.g., sodium morrhuate) are chemically related to inflammatory mediators such as leukotrienes and prostaglandins, and possibly undergo conversion to these substances to mediate the inflammatory response.
- **4.** Particulate irritants (e.g., pumice flour) are believed to attract macrophages, leading to phagocytosis.

Injections of inflammatory proliferant solutions in connective tissues have demonstrated ligament thickening, hypertrophy of the bone-tendon unit and the strengthening of tendon and ligament in animal studies (Hackett 1956; Liu et al. 1983; Ongley et al. 1988). The injection of hyper- or hypo-osmolar dextrose induces cells to proliferate and produce a number of growth factors. RIT was coined to reflect currently prevailing anatomic and pathophysiological trends in nomenclature. It stimulates chemomodulation of collagen by repetitive induction of inflammatory and proliferative stages leading to tissue regeneration and repair, thus increasing tensile strength, elasticity, mass and load-bearing capacity of collagenous connective tissues. This makes RIT a viable treatment for painful chronic enthesopathies, tendinosis, ligament degeneration, and laxity (Linetsky & Manchikanti 2005).

In retrospect, we can say that the original concept of prolotherapy solutions triggering the inflammatory cascade was overly simplistic. The mechanism of action is now considered to be multifaceted, and includes any or all of the following components (Klein et al. 1989; Reeves 2000; Yelland et al. 2004; Linetsky & Manchikanti 2005):

1. Cellular and extracellular matrix damage induced by mechanical needle injury stimulates the inflammatory cascade, which in turn governs the release of growth factors.

- **2.** Compression of cells by a relatively large volume of external fluid, as well as cell expansion or constriction due to osmotic properties of the solution injected that stimulates the release of intracellular growth factors.
- **3.** Chemomodulation of collagen through inflammatory, proliferative, regenerative/ reparative responses induced by the chemical properties of the solutions injected and mediated by cytokines and multiple growth factors.
- **4.** Chemoneuromodulation of peripheral nociceptors provides stabilization of antidromic, orthodromic, sympathetic and axon reflex transmissions.
- **5.** Modulation of local hemodynamics with changes in intraosseous pressure leads to reduction of pain. Empirical observations suggest that a dextrose/lidocaine combination has a much more prolonged action than lidocaine alone.
- **6.** Temporary repetitive stabilization of painful hypermobile joints, induced by inflammatory response to the solutions injected, that provides a better environment for regeneration and repair of the affected ligaments and tendons.
- **7.** Additional possible mechanisms of action include the disruption of adhesions that have been created by the original inflammatory attempts to heal the injury by the large volume of solutions injected. The relatively large volume of chemically nonirritating solution assumes the role of a space occupying lesion in a relatively tight and slowly equilibrating extracellular compartment of the connective tissue.

Indications, contraindications, complications, and risks

The general indication for prolotherapy is chronic musculoskeletal pain: chronic sprains and strains, myofascial syndromes, and arthritis. Whiplash injuries, medial and lateral epicondylitis of the elbow, knee, ankle, shoulder and other joint pain, tendinosis, and musculoskeletal pain related to osteoarthritis all fall within the three general indications. It is based on the premise that pain results from ligaments or entheses, and that these ligaments or entheses can be strengthened by the injection of irritant proliferant solutions into them. More recently, injection of hypertonic dextrose has also been used to restore Contraindications include potential local infection, allergies to the local anesthetic used or to some of the substances injected (allergy to shellfish is a contraindication to sodium morrhuate), injection into prosthetic joints, patients on anticoagulants who have a high INR (international normalized ratio).

Complications and risks can be needle related or substance related.

- Needle related:
 - Joint sepsis (Gray & Gottlieb 1983; Pal & Morris 1999). Infection rate post prolotherapy is not greater than post injection of corticosteroids, and is generally accepted at between 1 in 10 000 and 1 in 50 000.
 - Spinal headache (for injections near the spinal canal).
 - Peripheral nerve injury.
 - Pneumothorax (injections around the thoracic wall).
- Substance related:
 - Stiffness or soreness post injection. It typically can last 1–3 days.
 - Allergies (especially to shellfish in sodium morrhuate injections).
 - Chemical arachnoiditis (especially if using phenol in spinal or paraspinal injections).

The adverse effects of prolotherapy injections have been studied by Dagenais et al. (2006). Side effects related to prolotherapy for back and neck pain, such as temporary postinjection pain, stiffness, and bruising were found to be common and benign. Adverse events related to prolotherapy for back and neck pain are similar in nature to other widely used spinal injection procedures, and in general prolotherapy can be considered relatively safe when the more common solutions are used. Further study on this matter is required to replicate Dagenais' findings.

Techniques

There is a wide variety of injection protocols described in the literature. Usually, tender spots are identified with the palpating finger and the skin marked. The number of sites selected for injection, the composition of the proliferant (dextrose-based or dextrose in combination with other substances, often phenol and glycerin), and the volume of

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injectate varies, but is generally 0.5–1.00 ml per site injected. Injections are repeated regularly at varying intervals until the desired effect is achieved, with a maximum of weekly injections for up to 6–12 weeks. Injection may or may not be associated with manipulation. A bleb of local anesthetic is used sometimes, but not always (Reeves et al. 2000).

Outcomes and clinical evidence

In a systematic review of prolotherapy for chronic musculoskeletal pain, Rabago et al. (2005) stated in their conclusion that "there are limited high-quality data supporting the use of prolotherapy in the treatment of musculoskeletal pain or sport-related soft tissue injuries". Positive results compared with controls have been reported in randomized and nonrandomized controlled trials. Further investigation with high-quality randomized controlled trials with noninjection control arms in studies specific to sport-related and musculoskeletal conditions is necessary to determine the efficacy of prolotherapy. Literature interpretation is hampered by the variety of solutions and the variety of methods used. Nevertheless, there are several areas of literature in which the unique advantages of prolotherapy are being demonstrated (accessibility and low cost compared to alternative therapies such as surgery), to the point where close observation for follow-up studies is warranted.

A selection of some quality studies published so far follows, regardless of outcome, for specific conditions. There is a much wider body of published research, but its quality is variable and levels of evidence poor.

Lateral epicondylosis. Rabago et al. (2009) have identified strong pilot-evidence supporting the use of dextrose, polidocanol, whole blood, and PRP injections in the treatment of lateral epicondylosis. Prolotherapy in this summary includes under its umbrella all regenerative methods by injection including dextrose-based, inflammation-based, platelet/WBC based (platelet-rich plasma), adult stem cell-based, and essentially any other injection method in which either growth factors are stimulated or disrepair factors are blocked (as was mentioned earlier).

Achilles tendinosis. Sweeting & Yelland (2009) have found that prolotherapy alone is more effective than eccentric loading exercises – at present the "gold standard" – for chronic Achilles tendinosis. The

combination of both treatments is again superior to either of them individually. A nonrandomized study of 32 consecutive patients treated with intratendinous 25% dextrose injections (four injections on average) identified improvement of pain for activities of daily living (ADL) of 84%, and for sporting activity (71%). While not a randomized trial, a 94% (30 out of 32) follow-up rate at 12 months (4.5–28) suggests effective treatment (Maxwell et al. 2007).

Groin pain. A study of 24 elite level athletes (22 rugby, 2 soccer) with chronic groin pain, failure of all standard therapies, and failure to play at high level was reported by Topol et al. (2005). Twenty-two out of 24 returned to full play in sustained fashion. This study was then in essence repeated with 48 additional nonelite athletes, with identical results (Topol & Reeves 2008).

Plantar fasciitis (fasciosis). A small case series of 20 consecutive patients reports good to excellent results in 16 patients, which compares favorably with extracorporeal shock wave therapy (Ryan et al. 2009). However, further larger studies are necessary before confirming its effectiveness.

Low back pain. Most published clinical studies of prolotherapy are for low back pain. They include nonspecific low back pain, sciatica, and sacroiliac disorder. The five randomized control trials published (Mathews et al. 1987; Ongley et al. 1987; Klein et al. 1993; Dechow et al. 1999; Yelland et al. 2004; Dagenais et al. 2007), are of unequal quality and significance, and may be subject to different interpretations. In a recent study, Yelland and colleagues (2004) found that both injections of normal saline and dextrose solution resulted in a significant improvement, but that there was no statistically significant difference between normal saline (placebo) injections and prolotherapy. This highlights the difficulty of finding an appropriate placebo, because dry needling a ligament can cause an inflammatory reaction. It is therefore not a real placebo but a different intervention. Several consecutive case series since then have confirmed the positive clinical effects of prolotherapy (Hooper & Ding 2004; Wilkinson 2005; Cusi et al. 2008). These results need to be confirmed with well-designed randomized clinical trials that compare prolotherapy to a real placebo injection.

Patellar tendinosis. Two pilot studies (Alfredson & Ohberg 2005; Volpi et al. 2007) suggest that either polidocanol or PRP are effective to reduce pain and improve function. Again, this initial work needs to be completed with larger randomized trials.

Future challenges

While there is a considerable body of literature, the standard of the published research is of unequal quality. Techniques vary widely, as do substance injected, volumes, and frequency. Many studies can only be considered initial, and while promising, they need to be confirmed with soundly designed and conducted randomized trials.

One difficulty for the advocates of prolotherapy is the almost general lack of randomized controlled trials, with conditions (technique, protocol, inclusion and exclusion criteria) that can be replicated. The difficulty of how to arrange real placebo injections is a definite challenge. Comparing injection groups to noninjection groups is hardly randomization, but as mentioned above, a dry needle or an isotonic solution can produce a mechanical injury and generate an inflammatory response.

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The clinical expertise already gathered in the practice of prolotherapy needs the back-up of evidencebased research and experts' consensus to gain acceptance in mainstream medical practice, in keeping with the principles of evidence-based medicine (Sackett & Rosenberg 1995), for the benefit of all.

Summary and conclusion

Prolotherapy for treatment of musculoskeletal pain has been used for over 50 years. Present evidence with inclusion of systematic reviews, randomized and nonrandomized evidence indicates effectiveness of RIT in painful enthesopathies. The role of prolotherapy in "mainstream" medicine will improve with further quality research, including standard protocols, properly conducted randomized trials, patient selection, and defined outcome measures.

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Neural therapy

Rainer Wander Stefan Weinschenk

Introduction

Neural therapy is considered to be a regulatory and system-resetting therapy in which local anesthetics (LA) are injected in defined regions of the body. Homeostasis is thought to be re-established by extinguishing peripheral irritation and stimulating regulatory processes (Perschke 1989; Gross 1986, 1988; Heine 2006).

LA are commonly applied in surgery for local, regional, and nerve block anesthesia. However, they are also applied in segment therapy, trigger point therapy, and ganglion anesthesia.

Procaine is the preferred LA, due to its short duration of action and its positive effect on tissue perfusion; the latter also probably due to its metabolites (para-aminobenzoic acid and diethylaminoethanol). It is also assumed to influence cytokine metabolism (IL6, TNF-alpha, CRP) and activate the endocannabinoid system (Travell & Simons 1983; Heine 2006). The anti-inflammatory effect of LA has recently been discovered (Hollmann & Durieux 2000). The antiinflammatory effect is independent from the sodium channel action of LA. It lasts much longer than the anesthesia induced by LA. This is perhaps one of the most important explanations of the therapeutic properties of LAs. This mechanism also explains their relaxing effect on muscular trigger points (Heine 2006). In addition, LA reduces neurogenically induced inflammation by influencing neurotransmitters (Tracey 2009; Oke & Tracey 2009). LA also seem to have remarkable effects on the immune system (Cassuto et al. 2006; Rosas-Ballina & Tracey 2009).

The application of neural therapy is recommended only after the relevant knowledge of anatomy, physiology, and pharmacology has been acquired, and after a thorough training in the application of the therapy (for standards, see Weinschenk 2010 and Fischer 2007).

7.12

Neuroanatomy

All LA inhibit autonomic nerve conduction and therefore have a sympathetic inhibitory effect. These properties are connected with their influence on protein structures in the matrix (intercellular substance) (Pischinger & Heine 2007; Papathanasiou 2010).

Neural therapy is further based on segmental reflex mechanisms: all impulses coming from the periphery (peripheral nervous system) converge in the dorsal horn of the spinal cord (the central nervous system). Impulses originating from the cutis and subcutis, from joint structures, tense muscles and affected organs, from scars or injuries may become pathological. In the dorsal horn, they are normally eliminated by descending inhibitory systems and the patient remains symptom free. If these impulses become too strong, or if the inhibitory systems are impaired, there is a relay in three directions: via the anterolateral tract for the cortical perception of pain, via the motor anterior horn for the muscles, and via the lateral sympathetic horn and the sympathetic tract, which is connected to the peripheral nervous system. The anatomical interconnection of these pathways causes a positive feedback of the impulses through several segments (Jänig 1987).

These horizontal segmental projections may also be projected vertically beyond the segments by the muscular, fascial, sympathetic, and parasympathetic systems as well as the so-called functional chains, e.g. affected tooth \rightarrow disorder of the cervical joints \rightarrow functional scoliosis \rightarrow disorder of the sacroiliac joint \rightarrow lumbago (Wancura 2010, Wander 1992).

Procedure

Local therapy

In order to treat local inflammation, LA can be administered at the site of tenderness or inflammation. Typical indications are: skin inflammation, wasp bites, prolonged wound healing, muscle injury, tissue damage due to chemicals, and keloidal scars.

Segment therapy

The focus here is the neurologically defined segment of the spinal and cranial nerves. A segment consists of all structures innervated by one spinal nerve: cutis, subcutis, joints (also spinal joints), joint capsules, muscles, fascia, bones, and viscera. Every part of the segment reacts simultaneously on external stimuli of any other part, probably due to the convergence of all segmental impulses on the spinal level (Sessle et al. 1986). A therapeutic stimulus applied in one segment can influence another part of the segment. The easiest way to apply a stimulus is by intracutaneous injection in the appropriate dermatome, thereby producing a small papule or wheal.

In addition, the therapist may infiltrate scars, trigger points, fascia, the periosteum, or joint capsules (in particular vertebral joint capsules), and related blood vessels in the respective segment.

Example 1 Patient with shoulder pain (segment C4 and C5): two intracutaneous routes of papules (wheals): the first route goes from the C7 spinous process to the acromion and to the base of the deltoid muscle (segments $C4 \rightarrow C5 \rightarrow Th2$) and the second goes from the anterior axillary fold on the acromion to the posterior axillary fold (segments $Th2 \rightarrow C5 \rightarrow C4$). In this way, adjacent segments with ancillary function to the shoulder joint (ACG, SCG, clavicle, first and second rib) may also be included (Fig. 7.12.1).

Example 2 Patient with gastrointestinal symptoms in the upper abdomen: Injections of intracutaneous papules/wheals and subcutaneous injections in the area of the costal arch (so-called Vogler points, Fig. 7.12.2) and the fascia of the abdominal muscles.



Fig. 7.12.1 • Injection routes in a patient with shoulder pain. Internal organs can also be influenced using the knowledge of the cutivisceral and visceromotoric reflex arcs.



Fig. 7.12.2 • Injection point (Vogler point) in the area of the costal arch in a patient with gastrointestinal symptoms. From Weinschenk, 2010, with permission.

The xyphoid process (a rudiment of the seventh rib) is of special significance in this region and belongs to the T7 segment. The duodenum and the pancreas belong to the same segment. Because of the segmental organization of skin, muscle, fascia, and periosteum there is a connection between these structures and the stomach, the pancreas and the small intestine, through which these organs can in turn be influenced. Intracutaneous and deeper prefascial injection can also be applied in the anterior midline at acupuncture point CV12 (see Fig. 7.12.2). This point is connected with the celiac ganglion, which controls the sympathetic abdominal organs (Heine 2006; Wancura 2010).

Acupuncture points are considered to be passage points in the fascia (Heine 2006) through which nerves, blood and lymphatic vessels come to the surface. It is suggested that these structures can be influenced by injecting procaine at one of these points as well. Therefore neural therapists also choose acupuncture points for treatment (Heine 2006).

Extended segment therapy, ganglia therapy

If the segmental injections are not sufficient to improve the patient's symptoms, the clinician may block the afferent stimulus at the level of the spinal nerves, the plexus, the vessels, the sympathetic or parasympathetic ganglia (so-called extended segment therapy). By eliminating the afferent and efferent impulses, as well as simultaneously activating the descending endogenous inhibitory systems, the auto-organization of the peripheral nervous system can recover (see also Oke and Tracey 2009).

In the patient with shoulder pain (Example 1), this could be achieved by an injection into the stellate ganglion or the axillary plexus. In the patient with gastric problems (Example 2), extended segment therapy would consist of injection into the sympathetic celiac ganglion. In addition, an injection into the associated vertebral facet joints in the vicinity of the sympathetic trunk can be applied (indirect injection to the sympathetic trunk) (Kupke 2010), which affects 80% of the sympathetic afferent and efferent nerve fibers of the corresponding segment.

The *cranial parasympathetic* ganglia (ciliary, pterygopalatine, and otic) can be easily and safely accessed by injection. Their fibers accompany the trigeminal branches and their associated ganglia. Trigeminal irritation due to pathological processes in the sinuses, the teeth, local inflammation (e.g., granulomas), jaw disorders (e.g., TMJ syndrome), the tonsils and the ears can be treated by these injections.

The *sacral parasympathetic* nuclei are located in the lateral horn of the sacral cord (S2 to S4) and accompany the pudendal nerve. Thus gynecological diseases or sacral irritations can be positively influenced by neural therapy of the sacral parasympathetic ganglia, e.g., the inferior hypogastric plexus (Frankenhäuser plexus, Wander 2003).

Therapy via the interference field (Störfeld therapy)

An interference field is defined as an oligo- or asymptomatic region of the body which causes irritation in another remote location.

If a morphological substrate of the interference field can be found, it is also called *focus* (Mastalier & Weinschenk 2010). Interference fields, for example, can be scars or inflammation. Many mechanisms have been postulated to explain these remote effects. A neuroanatomic explanation is the segmental irritation, which is processed into the posterior horn. By projection to neighboring segments, it can cause secondary disorders. A cross-connection from trigeminal nuclei to their adjacent vagal nerve nuclei might also play an important role in projection disorders (Sessle et al. 1986).

The site of interference fields itself is asymptomatic, but its altered stimulus triggers autonomic dysregulation (e.g., increased muscle tension), of which the patient may be aware. Their major effect, however, is the pathologic irritation of a remote area.

More than 70% of the interference fields are located in the ENT (ear, nose, and throat) area and the teeth (Mastalier & Weinschenk 2010). Their stimuli cause characteristic changes of the neck, the so-called neck reflex points (NRP) or Adler-Langer pressure points (Fig. 7.12.3). NRP are easily detectable by their tenderness and sometimes swelling, and are strictly associated with interference fields in the corresponding segment of the frontal head (Weinschenk & Langer 2010). Projections of stimuli of the cervical spinal segments are not transferred to the corresponding segmental dermatomes, but almost exclusively to the myotomes of the same segment (Neuhuber 2007). They are no longer palpable following successful neural therapy of the corresponding interference field (Weinschenk et al. 2011). Epidemiology and the importance of the NRP in the detection of interference fields are subject to current research (Kolm et al. 2010).

Example 3 NRP 4 (= tonsils) is palpable as a painful swelling. After the injection into the tonsils, the NRP 4 together with its muscular tension (Mm. recti capitis, Mm. obliqui capitis, Mm. intervertebrales) is no longer palpable.

Fascia-Oriented Therapies



Fig. 7.12.3 • NRP (Adler-Langer pressure points). Painful areas in the neck muscles in patients with interference fields in the ENT and dental area. From Weinschenk, 2010, with permission.

Therapy via functional chains

The existence of NRP demonstrates the close relationship between structures of the facial regions and the segments of the cervical spine. The resulting changes of muscles and ligaments of the neck can be further transferred to lower parts of the spine. Asymmetric tension of the neck region may be conducted to the thoracic and lumbar regions of the spine, resulting in their counter-motion. Thus, a functional scoliosis might occur.

This close relation between cervical spine, thoracic and lumbar spine, and the sacroiliac joints is well known in many medical specialties, e.g., osteopathy (craniosacral therapy) (Fig. 7.12.4). Blockades of certain vertebral joints are part of this functional scoliosis and can easily be diagnosed by a trained manual therapist. Major diagnostic signs of the functional scoliosis will be found at C1, C4, T4, T10, and the L5/S1/sacroiliac joint.

Vertebral blockades can cause sympathetic stimulation of the respective vertebral segments and might lead to disorders of the associated internal organs (Wander 2010).

Systemic therapy

The vascular supply to the tissues is not segmentally organized. Blood vessels have their own 'vascular zones,' which can maintain skin and muscle pain beyond the corresponding spinal segmental organization (Gross 1986, 1988). All vessels (arterial, venous, and lymphatic) are accompanied by sympathetic fibers. In response to lesions, the vessels contract and cause disorders in both directions of flow, with corresponding pain symptoms. Injection of LA into the affected or other vessels will bring pathologic contraction back to a normal state.

Common routes of intravasal application of neural therapy are intravenous, intra-arterial, and infusion therapy. A well-known example is LA infusion in the treatment of tinnitus (Shea & Emmett 1981).

Indications, contraindications, complications

Indications: More than 100 years of clinical experience (Spiess 1902) indicates that neural therapy can be successfully applied in all functional and regulatory disorders, with and without pain, including: headache, vertigo, tinnitus, spinal disorders, musculoskeletal pain, neuralgia, chronic inflammation, thoracic and abdominal diseases, circulatory disorders, and other diseases (for an overview, see Weinschenk 2010; Hollmann and Herroeder 2010).

Contraindications for neural therapy are: advanced structural damage of organ systems (e.g., cirrhosis), genetic diseases, deficiency disorders, coagulation disorders, mental disorders. Structural changes and defects prevent neural therapy's beneficial effects (as in acupuncture and osteopathy).
Fig. 7.12.4 • The pelvis as an interference field: gynecological diseases or sacral irritations can correlate with ENT disorders and vice versa. ISG = sacroiliac joint. From Weinschenk, 2010, with permission.



Complications very rarely occur in neural therapy. The rule is: care reduces risk! The following complications can occur:

- allergic reactions (rare)
- injuries (e.g., bleeding, hematoma)
- infection
- patient's over-reaction following injection, e.g., collapse due to fear, stress, vagovasal orthostasis
- reaction due to additional medication or a secondary disorder (beta-blockers, tranquillizers; anticholinergic syndrome)
- central toxic or cardiac symptoms due to overdose (30–50 mg/day = 30–50 mL 1% solution).

Summary

Local anesthetics have various pharmacologic properties which make them potential drugs in the therapy of a variety of functional and pain disorders. Therapy follows the principle of 'as little harm as possible.' Beginning with simple injections, many beneficial results can be achieved. In the authors' clinical experience, the application to wheals in dermatomes, the infiltration of trigger points, and the injection adjacent to joint capsules are sufficient in many cases to induce a significant clinical improvement. The second stage is the injection into ganglia. Further, the clinician should look for the existence of interference fields and treat them.

Neural therapy extinguishes peripheral irritational stimuli, thus reducing stresses in various tissues, and facilitates the successful application of other treatment techniques (e.g., osteopathy). Acupuncture can then activate the descending inhibitory systems and achieve similar effects. After that, manual therapy can specifically be applied to transferred dysfunction caused at other segmental levels.

The combination of these various procedures has potentiating effects. Neural therapy should be taken into consideration, particularly in patients who do not respond to other therapies.

Research

Although LA have been used therapeutically since they were discovered in the late 1880s almost all the scientific literature that has been published is on their anesthesiologic application in surgery.

Most recently, following the important results on the anti-inflammatory effects of LA, more and more reports on their therapeutic effects have been published (for review, see Cassuto et al. 2006 and Hollmann and Herroeder 2010). Studies on the clinical effects of LA often cannot be found under the term 'neural therapy.' They have been published widely in different areas of medicine.

However, in recent years neural therapy has been integrated as a discipline in various universities worldwide, especially in South America, Switzerland (University of Bern) and Germany (University of Heidelberg). This provides a platform for future research in this field. There are various research projects in progress at the University of Bern (Fischer et al. 2011): for example, on the effect of neural therapy in patients resistant to conventional therapies, on the neuronal regulation of blood vessels, the importance of the sympathetic system in the development of chronic pain and inflammation, and a prospective study on pain and neural therapy. The outcomes will be published in 2012.

At the University of Heidelberg, major projects are addressing the molecular mechanisms of the anti-inflammatory effect of LA, NRP and their clinical importance in diagnosis and therapy, the correlation of neural therapeutic effects to vegetative parameters, and the quantification of side effects and complications of neural therapy. All these studies provide a promising platform for future research in this field.

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Dynamic fascial release – manual and tool assisted vibrational therapies

Zachary Comeaux

Introduction

In this chapter, fascia will be considered to represent the dissectible anatomic component, and also the continuum of mesodermally derived connective tissue. Combined with the neural system, this becomes a functional neuromyofascial syncytium in which the connective tissue component serves the role of form integrity, force distribution, and reactivity. The continuously connected universal distribution of connective tissue from the intracellular microtubules to the epidermis has been described elsewhere (Chen & Ingber 2007). Structurally, it reflects a fractal hierarchy in which each level is distinctively functional. The recent identification of alpha smooth muscle actin in fascia (Schleip et al. 2006) reinforces the concept of reactivity.

Historically, vibration had been used in light therapy, music and tone therapies, homeopathy, and radionics, as well as conventional radiation therapy (Abrams 1922; Kruser 1937; Vithoulkas 1980). This chapter focuses on therapies using vibration in the range of 1–100 Hz. It will provide a conceptual and historic framework to this topic, then describe in some detail one manual and one machine-assisted form of vibratory release with which the author is most intimately familiar. The author works and teaches in the context of osteopathic medicine and the manual approaches associated with that discipline.

History of manual and mechanical work involving fascia

The literature of early American manual medicine includes these words from Andrew Still, founder of osteopathy:

The fascia proves itself to be the probable matrix of life and death. When harmonious in normal action, health is good; when perverted, disease results.

Still 1902

.13

Wernham, a student of J.M. Littlejohn (who introduced osteopathic education to England), attests that rhythm was part of osteopathy since its inception (Wernham 2003). This expresses itself in his popularized General Osteopathic Treatment (GOT) and in derived Harmonic Technique (Lederman 1997; Hartman 2001). Both serve as general treatments. In the United States, T.J. Ruddy used patientactivated rhythmic motions to induce localized relaxation. This became the basis for Mitchell's Muscle Energy Technique and Vibratory Isolytic Technique (Mitchell 1998). These conceptualizations targeted specific local somatic dysfunctions.

The machine age, the late nineteenth century, produced debate regarding the use of manually and mechanically applied vibration, and its relevance to physiologic wellness. A perspective from that time can be obtained by comparing the work of A.

Hypothetical mechanism	Rationale and reference
Cumulative creep through successive cyclic loading of collagen fibers	Mechanical characteristics of collagen and the dynamic reciprocal functional and metabolic role in repetitive motion with muscle (Solomonow 2009)
Resetting alpha-gamma coordination in muscle activation changing tension patterns distributed by fascia	An extension of the muscle energy model (Mitchell 1998)
Phase coherence of quantum state of fascia as a tensegrity matrix	Application of the tensegrity structural model to the fascial organization of biologic systems. The fibrin matrix distributes force underlying structure and function (Chen & Ingber 2007). Fibrous network as a communication grid for coordinating encoded information within the fascial network; involves quantum vibrational energetic phase coherence (harmonics) for health (Ho 2008)
Entrainment of endogenous physiologic oscillators	Population coding model of neurobiologic function underlying recurrent activity (including depolariazation/repolarization cycle of neurons), rhythm of coherent depolarization of cells depicts a functional state. Phasic state changes entrain changes in rhythmic function of a population, resulting in functional change (Windhorst 1996; Zedka 1997; Farmer 1998)
Application of tonic vibratory reflex	Another route to altering tone through muscle spindle reflexes (Comeaux 2008)
Metaphysical	Descriptions using the term 'energy' as the term 'ether' was used in the twentieth century await empiric correlation (Comeaux 2002)

Table 7.13.1 Contemporary hypothetical explanations of effectiveness of vibration

Snow M.D. (Snow 1912) and articles in the *Journal* of Osteopathy from the same period contesting this approach (Bower 1904; Sullivan 1904) see Fig. 7.13.1. Robert Fulford reintroduced mechanical vibration into the context of osteopathic bodywork in the 1950s, using a percussion vibrator treatment (Comeaux 2002). Facilitated Oscillatory Release emerged from this as a manual application of oscillation with specific localized intent (Comeaux 2008). Manual vibration is also used in Trager work and the Vibromuscular Harmonization Technique of Roddick and Frere's Methode Rhythmique de Harmonization.

In the area of sports fitness training, whole-body mechanical vibration has emerged as a popular means of improving muscle tone and increasing lean body mass, using a variety of vibrating platforms. Manufacturers generally cite the effectiveness through generalized muscle contraction involving tonic vibratory reflex. Obviously, there are a variety of issues in selecting this therapy (Cardinale & Wakeling 2005), including variation in effect with different training schedules, inconsistent demonstration of strength/power with use of vibration, as well as lack of clarity as to optimal amplitude to engage natural dampening in the musculature. Additionally, tonic vibratory reflex involves an observed physiologic set of responses that are not easily characterized; in other words, various researchers have described different aspects of the phenomenon with varied results, reflecting that the processes are not yet completely understood. Tonic vibratory reflex is discussed later in this chapter in the context of the population coding model of neuromuscular coordination.

A contemporary list of proposed physiological mechanisms for the effectiveness of vibration is summarized in Table 7.13.1.

Hebb's hypothesis, harmonic function and oscillation

Fascia either directly or indirectly participates in the balance of tensions coordinated by the neural system. Population coding is a concept which complements the system of coordinative reflexes traditionally viewed as a primary means of neural coordination.



Fig. 7.13.1 • Early pneumatic percussion device, circa 1912.

Population coding is a concept derived from Hebb's attempts in neurophysiology to reconcile the spatial limitation with the extensive functions of the brain (Spatz 1996). He proposed an encoding process, since the skull could not contain enough space for task-dedicated tissue. In essence, it emphasizes that neuronal coordination involves patterns of rhythmic activity, not just dedicated cells and pathways. Individual neurons could participate synchronously in several operations. Functionality would result from phasic relationships and patterns of depolarization in addition to sheer connectivity. Despite limitations to the theory, the theme of rhythmic depolarization is a defensible model of neural coordination applicable on a peripheral as well as central level. Neural coordination is rhythmic and the controlling feature is phase synchrony across and between cell populations.

Both reflex and voluntary movements have been shown to demonstrate periodic rather than constant depolarization. Gross voluntary motion, muscle tone, and posture (including the cerebellar component) are composites of cyclic depolarization rather than a linear process (Windhorst 1996; Zedka & Prochazka 1997; Farmer 1998). This is similar to the experience of appreciating a constant object on a video screen which actually represents a signal refreshed at a rate of 24 cycles per second. Muscle tone, with its adaptive tendon and epimesial/perimesial (connective tissue) tension, is a function of rhythmic activity. This tension from postural movement has a reciprocal relationship with fascial tension. The applicable point to bodywork is that neuromuscular activity, both afferent and efferent, is rhythmic. Physical tone of structural tissue, including that occurring after trauma or strain, is determined by states of phasic depolarization.

Rhythmic reflexes – Tonic Vibratory Reflex (TVR) and related effects

TVR background

Tonic Vibratory Reflex (TVR) is a complex phenomenon that was originally described by Hagbarth. It involves the contraction of muscle in response to vibration in the range 0–100 Hz (Hagbarth & Eklund 1966). Martin and Park note a frequency-dependent excitation-contraction coupling leading to muscle fatigue (Martin & Park 1997). Many others show altered performance, notably undershoot or underextension of blinded voluntary movement, a kinesthetic illusion (Cody et al. 1990). Changes in muscle spindle activity betray involvement of discoordination of gamma-alpha motor neuron coordination controlling muscle tone (Burke et al. 1976; Vallbo & Al-Falahe 1990). In composite, these effects describe TVR as a discoordination of proprioception. But proprioception is a coordination of vestibular, ocular, cerebellar, cortical and alpha-gamma reflex effects. As a result, tonic vibratory reflex involves a complex of interactions. Curiously, locally applied vibrations cause reflexively coordinated movements of other body parts (Rossi et al. 1985; Zedka & Prochazka 1997; Han & Lennerstrand 1999). Additionally, spino-cerebellar disease or degeneration diminishes this effect (Abbruzzese et al. 1982).

TVR application

Nevertheless, the application of rhythmic afferent input can have intriguing effects. A most dramatic application of the principles described under TVR occurs in the work of microneurographic pioneer, Giseler Schalow (Schalow & Blank 1996). Beginning with work in open spinal surgical fields in partially cord-transected patients in hopes of reestablishing bladder control, Schalow developed a means of identifying pools of homologous nerve types. He demonstrated that there was a distinctive difference in patterns of phasic synchrony in the firing of homologous muscles of the lower extremity between his patients and normal controls.

In application. Schalow was able to show patient recovery of spontaneous movement and limited gait by suspending them in harness over a springboard. Initially, they were raised and lowered passively to simulate bouncing. Progressively, their limbs would reflexively respond. During this protocol, it was observed that the natural phasic character of rhythmic depolarization of neuromuscular firing gradually returned to postural muscles and the patients began spontaneous gait-associated movements. This activity involved progressive challenge of the patient to initiate synergistic contraction of the limb muscles. However, since it also involved externally applied rhythmic pressure to the limbs, possibly it entrained, by rhythmic afferent input, the natural protogenic pattern involved in gait.

Extrapolation to other clinical applications

This rhythmic application of afferent input shows transfer value, particularly in the specific application of the PVT and manual oscillation, and possibly to the use of vibratory platforms.

Although these neuroreflexive relationships pertain to the special connective tissue identified as striated muscle, the recent identification of adaptive actin fibers in fascial tissue makes this line of thought worth pursuing (Schleip et al. 2006). Empiric use of the procedures and devices described next underscores this relevance.

The percussion vibrator

Although it operates in the same frequency range as TVR, Robert Fulford conceptualized the application of oscillatory force in a different way. Considering matter, including the body, as fundamentally an expression of vibrating energy, he saw somatic dysfunction, the result of trauma and strain underlying complaints of pain, as a dysrhythmia. He referred to the residual tension in fascia as an 'energy sink,' or drain, by which the natural vibratory capacity of tissue was restrained from healthy resonance. The kinetic energy of injury was retained in the tissues. Modeling his thought from that of Walter Russell and Randolph Stone (Stone 1986), he considered the fascia to be a medium of transmutation of thought (another form of vibration) to action, in a biophysiologic as well as figurative sense. Treatment was then aimed at revitalizing, reenergizing tissue through positive thoughts, amplified by the percussion vibrator. He did so in both routine general protocols and specific individualized applications (Comeaux 2002).

In the general technique, vibration is applied over bony prominences (to maximally disseminate vibratory force through the fascial matrix) in a pattern from feet to shoulders. See Figs. 7.13.2 and 7.13.3.



Fig. 7.13.2 • Percussion vibrator, spinal application. Applicator may be advanced over each segment both to diagnose and treat.



Fig. 7.13.3 • Percussion vibrator, application in a limb. May be applied over bony prominence or directly over tissue you intend to release.

The organization was derived according to a conception of the distribution of the body's energy field, as described by Stone, as well as from experience working with residual birth trauma, even in adult patients. Specific, focal treatment could be applied anywhere that a decrease in vital resonance was detected. This lack of resonance relied on refined palpation to complement the more conventional parameters for defining dysfunction.

Fulford used a Foredom model percussor. More recently, the author has used a device introduced in the chiropractic community, namely the Vibracussor, considered to be an improvement, despite inaccuracies perceived in the technical descriptions in the accompanying manual. See Figs. 7.13.4 and 7.13.5.

Facilitated Oscillatory Release (FOR)

Attempting to bridge the American style of specific osteopathic diagnosis, vibratory motion, and physiology, the author discovered for himself the advantage



Fig. 7.13.4 • Contemporary version of Foredom percussor used by Dr. Fulford.



Fig. 7.13.5 • Vibracussor, a contemporary alternative preferred by the author.

of oscillation to enhance release. Originally, FOR was not conceptualized as being a free-standing method but rather as an enhancement whenever working against a myofascial barrier to free motion (Comeaux 2008). However, oscillation can be used as a minor component or a major component in engaging a patient's pattern of connective tissue tension. Again, it is envisioned that fascia, and other connective tissue including association with muscle tone, function as a continuum.

A reorientation to the basic concepts of wave physics is helpful in this context (Table 7.13.2).

In a spinal treatment, the patient may be placed prone. The practitioner stands facing the side of the patient and initiates a rhythmic standing wave by using the patient's pelvis, rocked from side to side, in a pattern allowing tissue to rebound naturally after each half cycle. The practitioner's other hand would be used successively to monitor the response of each vertebral segment to the induced motion. Once acquiring a feel for the anticipated response, it is possible to infer dampening, or dysfunction, at segments which show lesser response than expected.

One option during treatment involves exaggeration of the rhythm, with intent to apply constructive interference with extra energy in phase with the original rhythmic pattern. It is also possible to add

Wave	the rhythmic conveyance of energy through a medium		
Incident wave	a wave arriving at the interface of two media		
Reflected wave	some of the energy may be reflected back into the original medium		
Transmitted wave	some of the energy may be conveyed through the second medium		
Dampening	the attenuation of the energy of a wave due to friction or other interference with wave transmission		
Constructive interference	two waves have displacement in the same direction and their energy is additive		
Destructive interference	two waves have displacement in opposed directions and their energy is competitively diminished		
Standing wave	the recurrent incident wave and the reflected wave are additive and in a harmonious steady state		

Table 7.13.2 Basic wave mechanics – definitions

energy in the form of an impulse, out of phase with the standing wave, to induce change. The amount of force can be varied from gentle suggestion to the other extreme of confronting the myofascial or articular barrier to free rotational movement of the segment in question to encourage release directly.

As indicated above, FOR is not intended to be a system of protocols but as permission to integrate oscillatory or manual vibration as a corrective force anywhere that a restriction is sensed in the body's fascial or connective tissue network.

In an osteopathic context, the author favors muscle energy (postisometric relaxation) technique and direct myofascial technique, anatomically extrapolated according to need; but oscillation can be integrated into the spectrum of techniques, from thrust through cranial and other subtle techniques. Let me explain.

In the former, direct methods, where the barrier to free motion is approached with the intent of increasing range of motion, gross oscillation may increase efficiency and expedite release. The force may be modified using whatever leverage seems practical. Respiratory cooperation in the form of a relaxed expiration complements the oscillation. The two combined allow for more finessing of the approach to the restrictive barrier and therefore more control with less force in accomplishing the release. In indirect methods, in which force is applied away from the area of restricted motion, oscillation in the form of a mere flutter of the supported parts may advance and expedite the release sought. Similar tactics may be applied to sutural or membranous release in the cranium, and in other subtle techniques, if these are part of the reader's practice privilege.

In either case, the oscillation induces a sense of relaxation in the patient and reduces excessive tone that may have been retained in the fascial network. The decoupling of afferent and efferent input associated with TVR, described above, may also play a role here in relieving the inappropriate muscle hypertonia associated with some symptomatic complaints.

Regardless of the underlying physiologic impact, rhythmic motion seems natural as a way of dealing with a rhythmically functioning system. Although for convenience patients are assessed on an examination table, this is not their natural state, yet palpating while running, walking or dancing would be difficult. The rhythmic oscillation of the spine as in the spinal treatment described above seems more authentic in engaging the human rhythmically functioning neuromyofascial network (Comeaux 2008).

Other mechanical devices

Vibrating platforms

A number of vibrating platforms have been used for treatment or general fitness. Stated purposes include enhancing muscle tone and mass and therefore increasing strength, citing the involvement of tonic vibratory reflex, in place of resistance training (Cardinale & Lim 2003; Delecluse et al. 2003). As noted above, TVR is an intervention with complex results. The excitation-contraction uncoupling that occurs increases the rate of fatigue as well as the derecruitment of fibers, thereby requiring the remaining fibers to work harder. If effort is maintained in the form of standing, subjects are obliged to use their postural, antigravity muscles for support. The engaged fibers are therefore receiving resistance training by another method. They are selectively used to ensure body posture in their compromised state. In essence, this is resistance training, only under conditions in which the gross muscle is operating in a state of disorganization.

Deep oscillation

A further technology, marketed as Hivamat 200 and easily available, claims to create fascial change by applying an intermittent electrostatic charge to the collagen matrix, which creates cyclic movement in the deep tissues, leading to mechanical pumping and the redistribution of fluids. This is marketed as an adjunct to surgical or other wound healing, sports medicine and respiratory diseases (Seffinger 2009). Reference data are limited but outcome studies are available (Jahr et al. 2008; Aliyev 2009).

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PART SEVEN

Fascia-Oriented Therapies

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Graston technique®

A contemporary instrument assisted mobilization method for the evaluation and treatment of soft tissue lesions

Warren I Hammer

Introduction

This chapter is intended for manual therapists and clinicians who use their hands directly on the body to achieve therapeutic results. While manual therapists have grown to believe nothing can enhance their manual palpation skills – and, in fact, it is true there are some qualities that cannot be replaced by human touch, there is considerable evidence that the use of instrument-assisted soft tissue mobilization (IASTM) is making a major difference to clinicians and their patients.

IASTM, researched in the early 1990s, was introduced formally in 1994. The original patented technology, Graston Technique[®] (GT-IASTM), incorporates the use of stainless steel instruments to effectively treat soft tissue dysfunctions. While it may seem an unusual protocol for a manual therapist to use stainless steel instruments to treat soft tissue dysfunction, incorporating Graston Technique® instrument-assisted soft tissue mobilization (GT-IASTM) is growing as clinicians and patients become more familiar with its benefits. While no instrument could ever replace all the qualities of human touch, Graston Technique[®] (GT) is growing as clinicians and patients become more familiar with its benefits. The protocol is designed to provide an additional modality to the effectiveness of many of our handson methods. Actually, adequate manual dexterity is necessary for this method to be applied successfully.

The specially patented contoured beveled edges in the six stainless steel instruments comprising the GT instrument set greatly enhance palpatory skills. The technique is a comprehensive approach that, based on examination findings, integrates IASTM with a rehabilitation program, including targeted stretching and strengthening exercises. Lewit (1993) states that soft tissue techniques in general are applied to treat dysfunction and unless the dysfunction is understood the best of techniques may be used in the wrong place and at the wrong moment. In GT a functional examination is always stressed beforehand to determine the right place to use the instruments.

7

The use of GT is changing the paradigm of soft tissue methods only being used to restore dysfunction. Recent research is pointing to the additional role of affecting tissue pathology. The effect of mechanical load on degenerated soft tissue (Hammer 2008a) via its effect on fibroblasts and the extracellular matrix has added a new dimension to the treatment of dysfunction.

GT is especially valuable in treatments that rely on palpation of deep fascial fibrotic changes. It is especially effective in determining the direction of fascial barriers. GT's ability to feel has been compared to the use of a stethoscope for the detection of heart sounds. Graston Technique[®] incorporates at a minimum seven different strokes and has a variety of instrument angulations and pressures, depending on the contour of the body and the type of changes desired. The larger instruments can be used over broader areas while the smaller instruments can be used for localized treatment. At present there are thousands of clinicians and about 120 professional and amateur sports teams around the world using GT (Arnolt, personal communication 2009).

A major obstacle in the life of clinicians who use their hands is the development of repetitive trauma injury to their hands and upper extremities over time, adding to the difficulty of practice (Snodgrass et al. 2003). Most clinicians state that the use of these instruments reduces manual stress in their hands and upper extremities.

Rationale

It is established that "movement and mechanical forces maintain healthy cartilage, bone, muscle and tendons by regulating tissue remodeling. Remodeling is required to remove damaged cells and matrix (catabolic) and to replace damaged tissue (anabolic)" (Ramage et al. 2009). This scenario occurs with ordinary exercise. It appears that methods such as GT could, in effect, be performing a form of localized exercise to a lesion. In the tendon, for example, the fibroblast is considered a key player in tendon maintenance, adaptation to changes in homeostasis, and remodeling in cases of minor or more severe disturbances to tendon tissue (Kjaer et al. 2009). Studies have shown that, with mechanical load, increased fibroblastic proliferation (Davidson et al. 1997; Gehlsen et al. 1999) occurs that is responsible for reproducing the extracellular matrix (ECM), especially collagen 1, elastin, cytokines, and growth factors, among other important proteins. It is hypothesized that for degenerated connective tissue (i.e., tendinosis) GT re-initiates the inflammatory process by introducing a controlled amount of microtrauma to the affected area. A healing cascade is created by enhancing the proliferative invasion of blood, nutrients, and fibroblasts to the region, resulting in collagen deposition and eventual maturation. According to Kraushaar & Nirschl (1999), since tendons have an intrinsic capacity to heal, healing can occur if a fibroblast-driven process integrates old and new collagen in order to contribute to the final stability of the matrix. Khan et al. (2000) state that "The focus of any conservative management program should be to encourage collagen synthesis, maturation, and strength." But mechanical loading can be used to mechanically mobilize scar tissue, increasing its pliability and loosening it from surrounding healthy tissue without creating inflammation. Standley (2007) demonstrated that manual light myofascial treatment was anti-inflammatory and Yang et al. (2005) hypothesized that repetitive, small magnitude stretching was anti-inflammatory, while large magnitude stretching was proinflammatory.

tion and exercise are related to homeostasis, it is now clinically uncontested that positive healing results are occurring with manual loading. Miller et al. (2005) showed that by using acute exercise such as 1 hour of knee extension against resistance to fatigue, the collagen synthesis rate remained elevated for at least 2-3 days. Both a single loading bout as well as long-term habitual loading produced a markedly elevated collagen synthesis response (Langberg et al. 1999). A recent study on the effect of manual loading on soft tissue was conducted by Loghmani & Warden (2009). Bilateral transections of the medial collateral ligaments of rodents were performed. After 1 week, one side was treated with GT for 1 minute three times per week for 3 weeks. The opposite side served as an internal control. The GT-treated ligaments were assessed at 4 weeks and found to be 43.1% stronger, 39.7% stiffer, and could absorb 57.1% more energy before failure. Histological studies showed the treated ligaments had improved collagen fiber bundle formation and orientation within the scar region compared with the nontreated ligaments. Plate 7.14.1(A) shows the nontreated control. Plate 7.14.1(B) shows the GT-treated ligaments showing increased cellularity and more regularly oriented, elongated fibroblasts. At the end of 12 weeks there were minimal differences, although the GTtreated ligaments were 15.4% stiffer. Although the overall long-term effect of healing was similar, the study showed that the earlier return of ligament tissue-level biomechanical properties may allow quicker return to function with less susceptibility to reinjury.

Clearly, more studies are required to determine

the many effects of manual loading, but just as mo-

Applications

GT is effective almost everywhere on the body where there are superficial and deep fascia and retinaculum. Listed below are some of the common areas that have responded to IASTM. GT protocol consists initially of tissue warm-up (exercise, moist heat, ultrasound), treatment at least four days apart on the same area, 30 seconds to 1 minute for localized areas and 3–5 minutes for local regions. Immediately after treatment, stretching is performed and eventually, targeted strengthening. Cryotherapy is recommended if necessary. It is important to treat and evaluate areas from both a local and global kinetic perspective. GT is contraindicated for patients with open wounds, nonunion fractures, thrombophlebitis, hematoma, or

Graston technique

any other condition contraindicated for soft tissue manipulation, including patient intolerance or noncompliance (Hammer 2007).

Plantar fasciopathy, Achilles tendinopathy, lower extremity disorders

GT protocol is always based on evaluation and treatment of the kinetic chain and might include for the above areas treatment from the plantar fascia to the proximal Achilles tendon (Fig. 7.14.1), triceps surae, hamstrings, anterior and lateral thigh fascial areas. The chain is evaluated by passive stretching, resistive testing, gait analysis, palpation, and scanning with the instrument for restrictive fascial barriers. GT is effective for the treatment of ligamentous ankle sprains, including associated edema and the associated retinacula (Melham et al. 1998).

Knee

GT-IASTM treatment has been successful for anterior knee pain, postsurgical knee arthrofibrosis (Henry et al. 1999; Els 2006), quadriceps strain and tendinosis, tendinopathies (Wilson et al. 2000), collateral ligament sprain, popliteal strain, medial plica syndrome, compartment syndrome (i.e., tibialis anterior), iliotibial band friction syndrome, infrapatellar contracture syndrome, Osgood–Schlatter disease, patellofemoral retinaculum, pes anserinus bursitis, and nerve entrapments. While GT will be effective on the above local areas (Fig. 7.14.2), clinicians should always evaluate and treat proximal and distal fascial areas such



Fig. 7.14.1 • GT treatment for Achilles tendinosis.





as the fascia latae and its contents and the fascia below the knee. (See Local and global approach.)

Hip/pelvis

This treatment can be used for strain and myofascial restrictions of tissues surrounded by the fascia latae, tendinopathies, trochanteric and ischiogluteal bursitis (Hammer 1993), iliopsoas insertional tendinosis. Again, besides local treatment, a global approach to the lumbar, pelvic, and lower extremity fascia may be necessary.

Spine

All restricted spinal myofascial paraspinal areas from the pelvis, sacrum, lumbar, thoracolumbar fascia to the posterior scapula and shoulders (Fig. 7.14.3),



Fig. 7.14.3 • GT on posterior shoulder fascia with resistance



Fig. 7.14.4 • GT performed on cervical area.

to the cervical spine (Fig. 7.14.4), occiput and skull, plus the myofascia of the anterior chest, trunk, and abdominal areas can be treated. Also included are the interspinal, the long ligament, iliolumbar, and sacrotuberous ligaments. As in all the above areas, the clinician stresses the spine in all ranges of motion to elicit pain and stiffness and acknowledges the anatomical connections from the thoracolumbar fascia to the upper and lower extremity (Vleeming et al. 1989). Lumbar subacute compartment syndrome has been successfully treated by GT (Hammer & Pfefer 2005). GT is also effective for probable lumbar tendinosis (Hammer 2008b).

Shoulder

In this area, especially, a local (treating tendon insertions and capsules) and a global approach (treating muscle bellies and shoulder girdle myofascia) are appropriate. Lesions such as acromioclavicular sprain, adhesive capsulitis, rotator cuff and biceps tendinopathy, chronic bursitis (Hammer 1993), brachial plexus entrapments, and scapular dyskinesis due to myofascial alterations respond to GT.

Elbow, wrist, and hand

Strain of the myofascia of upper extremities, epicondylopathies (Wilson & Sevier 2000), tendinopathies (Haller et al. 2000), entrapment sites including among others radial tunnel, cubital tunnel, carpal tunnel (Fig. 7.14.5) (Baker & Wilson 1999; Wilson & Sevier 2003) and Guyon's tunnel syndrome.



Fig. 7.14.5 • GT treatment of carpal tunnel area.

Use of GT with movement and load

Movement is an important addition to the mechanical forces that translate into chemical signals essential for tissue homeostasis within the extracellular matrix and at the cellular level. Mechanical stimuli at the cellular level influence cell morphology, cytoskeletal organization, cell survival, cell differentiation and gene expression (Sarasa-Renedo & Chiquet 2005). Movement, along with mechanical load, influences fascial mechanoreceptors such as the Golgi, Pacini, Ruffini, and especially the most abundant type III & IV, resulting in changes in muscle tonus, proprioceptive feedback, inhibition of sympathetic activity, and changes in vasodilation and plasma extravasation (Schleip 2003). There is a strong possibility that freeing up fascia where spindle cells are located might be a significant factor in restoring normal muscle coordination (Stecco, L., Stecco, C., 2009).

There is some evidence (Hyde 2nd Fascial Conference poster, November 2009), and anecdotal evidence that using GT on a painful site with movement and proprioceptive stimulation will be more effective than static treatment. For example, active motion of the shoulder from internal to external rotation at 90° abduction might result in provoking pain on the anterior or posterior shoulder. GT would be used over the painful fascial site during movement until the patient states that the pain is greatly relieved or eliminated and the clinician feels a reduction in fibrotic adhesions. All active directions in any plane that provokes pain can be treated until the patient can move in a variety of directions without pain. At that point a load can be added to the involved area by way of a Thera-Band or weight to determine if additional pain will be provoked with the previously painful motions. GT treatment can then continue over the loaded painful areas again, until relief is attained. GT treatment over several visits will demonstrate decreased fibrosis, decreased tissue tension, and painless function. Many types of proprioceptive stimulation methods like vibration plates, stability boards, and loading devices can be used. Knees can be treated during squats and eventually squatting on a balance board. The lumbar spine can be treated on a balance ball (Fig. 7.14.6), and elbows can be treated during eccentric contraction.

Local and global approach

Friction massage has been successfully used over the years as a localized treatment to muscle bellies, musculotendinous areas and insertions (Cyriax 1984). It is also recognized that the proximal and distal fascial connections may be inhibiting the function of the joint area (Stecco & Stecco 2009). Stecco's work in Fascial Manipulation[®] has explored the myofascial kinetic chain and has established myofascial units, myofascial sequences, diagonals, and spirals where instrumentation may also be used on connective tissue acupoints (www.fascialmanipulation.com, www.fascialmanipulationworkshops.com).



Fig. 7.14.6 • GT treatment of thoracolumbar fascia using a balance ball.

Treating tendon insertions must take into account that the insertion site involves not only the enthesis itself, but neighboring tissues as well (Benjamin & McGonagle 2009). Currently it is recommended (see www.FAKTR-PM.com or www.grastontechnique. com) that GT treatment should be used on areas found locally; i.e., possible pain on the anterior glenohumeral area on resisted supraspinatus testing, but also treatment, should be directed to areas of pain found, for example, on global diagonal D1 and D2 patterns (Voss et al. 1985). The clinician should use his/her imagination and have the patient move in any direction that might provoke myofascial pain.

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The fascial distortion model

Georg Harrer

Introduction: connective tissue as mechanosensory system

In spite of the fact that connective tissue is in general envisioned as a scaffold with minimal metabolism and blood supply, it serves as a transportation network for all adjacent tissues. All structural changes in the connective tissue, whether caused by trauma or disease, lead to an alteration of the metabolism of all other tissues involved. This close relationship enables the connective tissue to be a very sensitive indicator for virtually any dysfunction in all areas of the body. Proprioception and nociception – the senses for position, touch and pain – are located in the connective tissue.

Wherever disease, tumor, or trauma cause damage in the body, pain occurs as soon as fascia is involved. This phenomenon, although well known in the intestines and the brain, is often not applied to the musculoskeletal system. The signaling function is not exclusively responsible for pain and touch, and muscle energy (with or without movement) is observed and measured by the surrounding connective tissue. Due to the close connection between fascia and nerves – every nerve consists mainly of connective tissue – information is precisely conducted. Overall, the fascial net is a complex and precise mechanosensory system. Not only are the positions of the limbs observed, but also all traumatic or metabolic dysfunctions.

The patient as expert – the Typaldos model

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Mainstream medicine takes little advantage of this most complex information network. Physicians increasingly ignore it and instead use technological investigation devices. The patient gets the impression that his perception and description of pain or discomfort disturbs, rather than supports, the diagnostic process. Accordingly, magnetic resonance imaging (MRI) scans are used to locate the disorder even though key studies suggest that MRI scans are, in spite of their spectacular resolution, questionable tools to locate the source of pain (Jensen 1994; Chou 2009).

In most contemporary medical healing approaches, it is the expert practitioner who makes a diagnosis due to his extensive training and skills. The patient in general is unable to be involved in these deliberations, due to the lack of a common language and the impression that their knowledge is inferior.

This frustrating experience was the origin of Stephen Typaldos' fascial distortion model (FDM) (Typaldos 2002). Typaldos, an osteopath and emergency physician, found it a frustrating experience to learn how little medical and osteopathic treatments could help patients suffering from so-called soft tissue injury. Conditions such as sprained ankles, lower back pain, and neck pain seemed to be almost unresponsive to the treatments he could offer within an emergency department. Improving his manipulation skills or increasing the diagnostic effort (MRI, ultrasound, nerve conduction, etc.) did not improve the outcome.

Typaldos instead started to ask the patients what kind of treatment they themselves thought might bring relief, and, to the patients' surprise, he applied the treatments they proposed; for example, pressing on a particular spot in a patient's back, which she was unable to reach herself, or pulling on the arm or on the skin of a patient as they suggested. In many cases these procedures had better results than any other therapy these patients had undergone previously in their career. This again was a big surprise to Dr. Typaldos. This method of diagnosis and treatment planning was unknown in both mainstream medicine and in the kind of osteopathy he had learned. On the other hand, the symptoms and reported improvement could not be explained by conventional diagnoses.

Typaldos' impression was that the patients had subconscious understanding of the nature of their condition and of possible solutions. Studying the patients' stories and gestures, he found recognizable entities for which he saw fascia as the common denominator. Typaldos postulated three-dimensional alterations of fascia that could apparently be repaired by the maneuvers and techniques he had successfully performed under the guidance of his patients. Typaldos discovered six "fascial distortions", as he called these new diagnoses (Table 7.15.1).

Table 7.15.1	Fascial	distortion	types	as	described	by
Typaldos						

Fascial distortion type	Description
Trigger band (TB)	Longitudinal crack within a band
Herniated triggerpoint (HTP)	Gap opening within a sealing fascia, with resultant protrusion
Continuum distortion (CD)	Shift of minerals within transition zone between bone and ligament
Folding distortion (FD)	Overstretching of folds in "accordion"-like structure
Cylinder distortion (CyD)	Entanglement of cylindrical coils in skin
Tectonic fixation (TF)	Loss of gliding ability in a joint

The fascial distortions

The six distortions are listed in the order they were discovered by Typaldos.

Trigger band (TB)

One type of connective tissue is banded fascia. This means almost all fibers are aligned in the same direction in order to resist forces along this direction. Due to their anatomy, these fascial bands are prone to injury caused by shearing forces. In the case of a shearing force applied to the banded fascia, the long fibers lose their coherence and a longitudinal crack occurs in the band. The bands then become shorter and the edges along the crack become twisted. The best analogy to envision the TB concept is probably the "zip-locTM" bag.

Herniated triggerpoint (HTP)

All compartments of the human body are separated by fascia. The main purpose of this kind of fascia is to seal the cavities and prevent enclosed organs from protruding out of the cavity. In general (apart from the thorax) the pressure gradient makes tissue and fluids prone to protrude towards the surface as soon as a gap in the sealing fascia opens (a hernia). Some of these hernias (e.g., inguinal hernia) are well explored and there are successful (mainly surgical) treatments available. Others are less well known and the complaints are not understood as a hernia. Only the concerted occurrence of an opening of the canal, a pressure peak, and the protrusion of tissue out of the compartment can be identified as pathologies. Each component occurring on its own is physiological.

Once a hernia is formed it is maintained by the pressure gradient. Spontaneous healing is not expected but reduction leads to repair. A closure of the aperture (which is physiological) is not strictly necessary but might help avoid a relapse.

In the FDM, hernias within the musculoskeletal system play a major role in explaining patients' complaints, and entirely new treatment options utilizing the concept of reduction, which would be deemed impossible in other models. Other medical models that concern these complaints are interpreted very differently.

Continuum distortion (CD) (see Fig 7.15.1)

To understand CD, it is necessary to look into the nature of bone and ligament in a fundamental way. In the traditional model the point where bone and ligament come together is called "insertion". A junction of two obviously different structures is postulated. This is contradictory to the enormous stability of this junction. In the continuum theory, as part of the FDM, bone and ligament are envisioned as one structure. Ligament is accordingly considered to be demineralized bone and a bone calcified ligament. Single bones are not considered to be components of "invivo anatomy" and only come into existence after collagen biodegradation, in mechanical injury, or in medical dissection studies. The same principle applies for ligaments.

That the majority of fibrous pathways are within bone suggests an important role for collagen fibers in bone stability. Fascial fibers can be considered as the highly tensile component of this compound, analogous to the steel in ferroconcrete. A purely mineralbased material can never be resistant to bending forces: it is only resistant to compression forces. The FDM postulates that the tissue in the transition zone has the ability to shift between two states (i.e., between bone and ligament). Depending on the demands, it can become ligamentous or osseous by shifting calcium matrix out of or into the bone. Since the calcium concentration is high in the bone and low in the ligament, the soluble mineral is prone to spread into the ligamentous zone driven by the force of entropy. Physical activity forces the calcium back into the bone by osteoblast activity in the bone and mechanical stress on calcium that has shifted into the

Fig.7.15.1 • Continuum distortion. From Liem & Dobler 2009.

ligament. The opposite is a common finding in immobilized patients with a lack of physical activity in intensive care units. Osteoporosis in the bones and exostosis in the ligament are the manifestations of this.

According to the FDM, this phenomenon occurs several times a day on a small scale when physical activity leads to a small shift of the transition zone towards its ligamentous configuration, and inactivity leads to a small shift towards osseous configuration. This shift is only possible if synchronized in the entire "insertion". As soon as one part of the transition zone shifts in the osseous configuration while the rest shifts into the ligamentous configuration continuum distortion occurs, leading to immediate loss of functional ability due to disturbed proprioception. CD is envisioned as a pathological step in the transition zone between bone and ligament. To envision bones and ligaments as one structure (the continuum) with calcified and decalcified zones allows an entirely new perspective on injury and complaints located in or adjacent to "insertions".

Folding distortion (FD) (see Fig. 7.15.2)

In the body there are numerous flexible junctions. These flexible junctions, generally called joints, are surprisingly durable, even with repetitive movement. A knee joint, for instance, can be bent and straightened without damage or resistance against the movement. The motion appears to be well guided by multiple structures. In FDM we consider the sum of all these protective structures to be like bellows. In technical engineering the demand for minimal



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wear and tear without disturbing flexibility is often met by the utilization of bellows. Examples for the use of bellows vary from the accordion instrument to the body's respiratory tubes. The bellows or accordion model allows entirely new perspectives on joint complaints.

In medial positions the folds always unfold and refold in the correct pattern. The bellows analogy inevitably leads to the following specific pathology. As soon as the accordion is overstretched the folds disappear. When now compressed towards the neutral position there is no guiding pattern for the folds to refold and so they wrinkle. We call this an unfolding distortion (uFD) because the extreme unfolding initially caused the pathology. In this crumpled state the bellows can no longer function properly and the joint is poorly guided. This accordion analogy suggests the opportunity to restore the bellows and its folds by controlled traction. Once the wrinkles are torn out of the bellows, the folds can refold properly again.

If the forces are in the opposite direction we are facing a refolding distortion. Extreme traction with consecutive compression leads to uFD and can be restored by controlled traction. Extreme compression with consecutive traction leads to refolding distortion (rFD) and can be restored by controlled compression.

Cylinder distortion (CyD) (see Fig. 7.15.3)

The most superficial tissue of the body is the skin. It is tensile in all directions. Different to other manifestations of connective tissue, the specific arrangement of the skin's collagen fibers allows far more elasticity. It is a system of spiral cylinders around the entire body with neither beginning nor end. The cylindrical coils go in virtually all directions.

This arrangement leads to equal resistance in all directions. Although the coils are interwoven, the

Fig. 7.15.2 • Folding distortion. (A–C) Physiological folding, joint stabilization against traction and compression, smoothing of folds at excessive traction. (D–F) Subsequent compression after excessive traction creates knitted folds which remain. (G, H) A "plopping" traction treatment recreates the natural folds. From Liem & Dobler 2009.



Fig. 7.15.3 • Cylinder distortion. From Liem & Dobler et al. 2009.

fibers have to move separately to allow equal distribution of tension to all coils in any movement. If these coils become entangled in each other the ability to move separately decreases, leading to disturbed proprioception in the entire region. In FDM this condition is called CyD. The entanglement can be caused by adhesions attached to the fibers of the coils, or by lateral stress to the skin. The geometry of entanglement is very complex and therefore prognosis and duration of the condition vary significantly. Unlike the other fascial distortions, it is difficult to predict the progress and duration of CyD.

Tectonic fixation (TF)

All slide bearings in the body consist of fascia. Some of these slide bearings possess anatomical features such as cartilage or synovial fluid and are therefore termed joints; other slide bearings lack these anatomical features but have similar functional ability. All slide bearings of the body show a similar construction. They consist of two corresponding sliding faces and a layer of lubricant in between. In so-called joints, these sliding faces are cartilage and the lubricant is synovial fluid. In other slide bearings, such as the scapula–thoracic articulation, the lubricant is interstitial fluid. These work the same way and perform a tectonic movement, a horizontal gliding.

TF is the loss of this ability to glide. In the joint, the synovial fluid has an additional function as nutrition for the non-vascularized cartilage. The production of synovial fluid is triggered by movement. It needs only a short immobilization to omit the production of synovial fluid. This causes stiffness of the joint. As soon as the joint is moved again the production of synovial fluid commences again. This restores the ability to glide and the nutrition of the cartilage.

Apart from joints there are numerous slide bearings surrounding tendons or between scapula and the thorax. In these slide bearings the interstitial fluid is the lubricant and the same rules apply. A lack of fluid leads to stiffness. Movement leads to more fluid. TF is always secondary either to other fascial distortions or immobilization due to external reasons. Once the reason for the immobilization is eliminated, children need only a few days to get back to their normal mobility, but in elderly people it may take much longer. The correction of the TF is only possible once the causative fascial distortion (one or more of the other five) is/are eliminated.

The diagnosis of fascial distortions

General considerations

As stated above, the essential hypothesis for any diagnostics in FDM is the supremacy of the patient's refined proprioception and nociception compared to any diagnostic tool from the exterior, whether based on palpation or technology. Since each of the six distortions is located in another type of fascia, they are felt differently by the patient. Also, the specific location is felt very precisely, provided that the distortion is local. Usually the patient only lacks the vocabulary to communicate these perceptions to the practitioner, and the skills or self-confidence to correct the distortion on his own. Verbal language is a poor tool to communicate pain and discomfort. Depending on the language there are only a few words for pain and discomfort; far too few to explain these complex impressions. Another obstacle to communication is the fact that patients and physicians think that medical knowledge and terminology improve these communication skills, but in fact the opposite seems to apply. The more the patients are knowledgeable of medical terminology and envision medical models for their condition, the more they are estranged from their own perception.

Diagnostics in the FDM rest on the following three pillars:

- 1. Body language.
- **2.** History, clinical symptoms, mobility tests.
- **3.** Palpation (only to locate the distortion).

Trigger band (TB)

- The patient runs the tips of his fingers along a distinct line. In general only the most painful section of the TB is shown with the fingers, not the entire pathway.
- Described as "burning", "pulling" pain. Worse in the morning. Restricted motion in one or more than one plane.
- The entire pathway is painful on pressure.

Herniated triggerpoint (HTP)

- The patient presses the fingers or the thumb firmly on to one spot, generally located on the torso.
- Described as "dull", "constant" ache in a specific area. Restricted motion in all adjacent joints.
- Severe pain on pressure can be well distinguished by the surrounding tissue, palpable tumor-like ball in the tissue.

Continuum distortion (CD)

- The patient points decisively to a singular point on a bone, in general close to a joint, using the very tip of the index or middle finger.
- Patient can pinpoint pain on a bone. Painfully restricted motion in a singular plane, often only in one direction.
- Very small spot highly painful on pressure.

Folding distortion (FD)

- The patient holds a joint with the hand.
- Described as "pain in the joint" or "in the center of the joint", impression of instability, remains

unchanged for a long period of time. No significant restrictions in motion.

Palpation is insignificant.

Cylinder distortion (CyD)

- The body language is a wiping motion with the palm of the hand along a limb or the torso. Also repetitive squeezing of a nonjointed area is presented.
- The patients complain about severe, deep pain, which cannot be explained by the objective findings. The pain is difficult to reproduce. The maximum pain is experienced at night-time. Different to TB, the CyD responds poorly to antiinflammatory drugs, morphine, and other medications. The patient also complains of bizarre symptoms such as the impression of swelling, tourniquet, tingling, numbness, and other symptoms, which are in general related to the realm of neurology but do not correspond to nerve or spinal root or other neurological explanations. Reports very painfully restricted motion but hard to reproduce; range of motion might appear as normal at the very time of testing.
- Palpation insignificant, no tenderness on pressure.

Tectonic fixation (TF)

- The patient moves the stiff joint forcefully.
- TF does not hurt! If there is pain involved it is the product of one or more of the other five fascial distortions. Significant reproducibly restricted motion in all planes. No passive motion possible.
- Palpation insignificant.

This proposed patient-guided diagnostic system inevitably leads to a redefinition of the patient-practitioner relationship. It is no longer the qualified, experienced, and highly educated practitioner who makes a diagnosis in a process that cannot be reproduced by the patient. Rather, we see a role reversal, with the patient in a better position due to the natural advantage of innervation, which enables the patient to present the diagnosis. The practitioner is only the interpreter and has not only to accept the patient's signals but also to take actions the patient subconsciously suggests, but for whatever reason is unable to perform sufficiently on their own. Oral language, maybe the most striking character that differentiates us from other animals, is strongly influenced by the intellect and so all oral statements can be distorted by multiple factors such as other physician's opinions, X-ray reports, internet research, and so on. The degree of medical knowledge of the patient is inversely proportional to the value of information that is achieved in history taking. Therefore in FDM the observation is focused on the body language, since this language is independent from the intellect. Furthermore, it is international and all nationalities can be assessed the same way.

General treatment of fascial distortions

FDM leads to a very specific understanding of the particular pathology on the level of fascia. Accordingly, the therapy has to be as specific. Basically, there are many approaches possible. Due to the fact that Stephen Typaldos had a professional background in osteopathy, the current treatment concentrates on manual approaches to correct fascial distortions. The techniques are highly specific and so only suitable for specific distortions. Since in manual maneuvers a directed vector of force must always have a specific impact on the particular fascia, something like a "general fascia technique" does not exist in FDM. Accordingly, the force has to be specific for every single fascial distortion. In principle, all fascial distortions are considered to be reversible. As soon as the type of distortion is diagnosed, specific manual techniques are utilized in order to correct the distortion. After every single step the complaints and restrictions are re-evaluated to guide the treatment.

Final statements

In order to apply FDM successfully, various skills such as the power of observation, and finesse in the manual techniques are required. Whether the treatment was successful can only be judged by the patient, since it is their perception of pain, restriction, instability, or weakness that leads to the decision to seek professional help. According to FDM, it can only be considered a success when all these complaints are sustainably eliminated. Clinical experience, as well as a first clinical trial, shows that this result can be achieved in a surprisingly high percentage (Stein et al. 2009).

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Frequency-specific microcurrent

Carolyn McMakin

Frequency-specific microcurrent is a means of treating myofascial pain and remodeling fascial adhesions and scar tissue. Frequency specific-microcurrent uses specific frequencies delivered as square wave pulses from a battery-operated two-channel device.

History of frequency-specific microcurrent (FSM) therapy

Microcurrent electrical neuromuscular stimulation (MENS) was developed in the 1970s as a batteryoperated physical therapy modality delivering current in the microampere range. An ampere (amp) is a measure of the strength of electric current and measures the rate of flow of charge in a conducting medium. One microamp (μA) equals 1/1000th of a milliamp (mA). By comparison, interferential, TENS, and high-volt pulsed galvanic stimulators deliver currents in the milliamp range causing muscle contraction, pulsing, and tingling sensations. TENS applies an electrical force that stimulates pain, suppressing A-beta afferent fibers which compete against A-delta and C fibers that transmit pain signals. Most TENS units deliver current around the 60 milliamp range (Kirsch & Lerner 1998). Although microcurrent devices are approved in the category of TENS for regulatory convenience, in practical use they are in no way similar and cannot be compared to TENS in their effect.

Microcurrent is subsensory and cannot be felt by the patient (Mercola & Kirsch 1995). Traditionally, microcurrent therapy has been used to increase the rate of healing in injured athletes, to treat and manage muscle pain and dysfunction and increase the rate of fracture repair (Rowley et al. 1974; Bertolucci & Grey 1995; Kirsch 1996; Kirsch 1997; Lambert et al. 2002).

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Current in the range of 10 up to 500 microamps was observed to increase ATP production, amino acid transport, protein synthesis, and waste product removal in rat skin, whereas ATP production leveled off between 500 and 1000 microamps and decreased when the current was above 1000 microamps (Cheng et al. 1982). TENS devices provide up to 60 times higher current levels than those seen to decrease ATP production, which may explain why TENS units have not been found to be effective in treatment of myofascial pain. Typical microcurrent applications use only low and simple one-channel frequencies such as 0.3 Hz, 3 Hz, 10 Hz, 30 Hz, and 300 Hz (Manley 1994).

The current protocols for the use of frequencyspecific microcurrent (FSM) in treatment of myofascial pain, trigger points and fascia, as described in this chapter, have been developed over the past 15 years. These were originally based on the clinical recommendations of a previous generation of practitioners using these methods. Via a process of trial and error, it was determined, through clinical use on volunteers, that the use of frequency combinations that did not produce improvement also did no apparent harm. The descriptions of the frequencies, based on the recommendations mentioned, were initially taken at face value, and used speculatively for various chronic and acute conditions, to determine if they would produce a change in symptoms and clinical improvement (McMakin 1998; McMakin 2004; McMakin et al. 2005).

FSM and inflammation

A current of 40 Hz was initially recommended as being useful to 'reduce inflammation.' Use of this frequency in a clinical setting suggested that it did only that and was not useful to change any other condition, and it was found that no other frequency would reduce inflammation. Use of 40 Hz on channel A and 10 Hz on channel B was found to reduce pain in fibromyalgia patients from an average 7.4/10 to 1.4/10 VAS and to reduce all of the inflammatory cytokines as measured by microimmunochromatography at logarithmic rates by factors of 10 to 20 times in 90 minutes. Medical cytokine researchers report that cytokines are difficult to modify, and change slowly when they can be made to do so. The control patient who had myofascial pain, but not fibromyalgia, was treated with a protocol for myofascial trigger points that did not include 40 Hz or 10 Hz and had no change in cytokines, although the myofascial trigger points and pain resolved (McMakin et al. 2005).

FSM and scar tissue

One set of frequencies has been found to be so effective at removing or remodeling scar tissue that they were shown to be effective in treating mature burn scarring. Eight patients with long-term burn scarring were measured for range of motion on a Monday by occupational therapists in the burn unit at Mercy St. John's Hospital in Springfield, Missouri. They were treated 1 hour per day for 3 days and range of motion was measured on Friday and weekly for 4 weeks following the treatment. Seven patients completed treatment and the eighth dropped out for reasons not involving the treatment. All patients had a statistically significant improvement in range of motion that persisted for the 4-week follow-up period. Clinical use in athletes and pain patients alike confirms this effect in softening and apparently eliminating scar tissue (Huckfeldt et al. 2003). However, the frequencies for fibrosis, scarring and hardening change only range of motion and tissue texture, and have no effect on inflammation or swelling.

Equipment

Frequency-specific microcurrent methodology has been taught in three-day seminars to medical, chiropractic, osteopathic and naturopathic physicians and physical therapists since 1997 as frequency-specific microcurrent in the United States, Australia and Ireland. The technique requires use of any microcurrent device that can provide an accurate frequency $(\pm 0.5 \text{ Hz})$ on each of two channels using a ramped square wave and alternating pulsed direct current.

Clinical outcomes in the treatment of myofascial pain

Two hundred and fifty new patients were treated in 1996 and the results in 137 cases of 'simple' chronic myofascial pain in various body regions due to prior trauma or chronic overuse were examined. Symptom duration ranged from 8 months to 22 years. The majority of patients had been treated by one or more prior therapies including prescription drugs, physical therapy, surgery, chiropractic, acupuncture, trigger point therapy and massage. Of those 137 patients, 128 completed treatment. Pain was reduced in 126 of those 128 from an average 5-8/10 to 0-2/10. Two patients had pain reduced from the 5-8/10 range to the 3-4/10 range. Treatment duration varied between 6 and 60 visits depending on the severity. complexity and chronicity of the case. Patients were told to return if the pain reoccurred or motion became limited. Only six patients returned for occasional follow-up treatments. The results seem to be long lasting and possibly permanent. No follow-up questionnaires were sent, so the exact long-term results were not documented in this initial group.

Further refinements in treatment techniques and frequencies resulted in improved patient response and reduced the number of treatments required. Data were retrieved from the charts of 100 new patients seen between January and June of 1997. There were 50 patients with head, neck or face pain resulting from chronic myofascial complaints. There were five patients with acute cervical injuries and 21 with chronic low back complaints. The rest were shoulder, other extremity, or thoracic pain. Most of the patients were referred to the clinic by a medical physician, chiropractor, naturopathic physician or another patient. Chronic pain was defined as pain lasting longer than 90 days after the precipitating trauma.

The outcomes were described as simple averages. The average chronicity was 4.7 years in head, neck and face pain, and after 11.2 treatments over a 7.9-week treatment period the average pain levels decreased from 6.8/10 to 1.5/10. There was no control group but the patients in some sense served as their own control since 88% (44/50) had failed with some other therapy. Seventy-five percent of patients (33/44) had failed with medical care, 54% (24/44) had failed with chiropractic, 38% (17/44) had failed with physical therapy, 11% (5/44) with naturopathic care, and 6% (3/44) with acupuncture. Many patients had used two or more of these therapies, with minimal to no permanent relief (Fig. 7.16.1).

The outcomes were better in the low back pain than in the neck pain group even though the low back pain was more chronic at 8.4 years versus 4.7 years in the neck pain group. Patients with myofascial trigger points in the low back muscles were treated an average of 5.9 times in 6 weeks and the average pain was reduced from 6.8/10 to 1.6/10.

In general, patients were treated twice a week with FSM, manipulation and massage. As they improved, their treatments were reduced to once a week, then once every 2 weeks. Half the neck pain patients took 10 or more treatments to obtain maximum improvement. It became evident that recovery from chronic neck pain required 11 treatments in 8 weeks compared to the low back patients' 6 treatments in 6 weeks, because their myofascial pain was complicated by or perpetuated by nerve irritation, disc and facet joint injuries and ligamentous laxity from trauma or degeneration. The low back patients all had simple myofascial pain due to trigger points, with no complicating factors. This observation illustrated the specificity of response. Trigger points perpetuated by nerves, discs, facets and ligaments do not respond as well to the treatments for trigger points directed solely at the muscle. When the treatment protocols for nerves, discs, facets and ligaments were added and used with a more accurate assessment and diagnosis, patient recovery became very efficient and consistent.

How FSM treatment differs from other fascia therapies

Frequency-specific microcurrent treatment offers several advantages for the manual therapist or treating practitioner. The treatment is pain free and comfortable as long as the manual technique is not too deep or forceful. The treatment can address the fascia in an entire region, such as the neck and shoulder, treating muscle couples simultaneously agonist and antagonist, thereby balancing function and structure more efficiently and reducing pain more effectively (Fig. 7.16.2).

The treatment does not require deep or forceful manual pressure. The most significant shift in manual technique with FSM is the need to reduce treatment pressure. The proper frequency combination produces an almost immediate change of state in the fascia. The fascia changes from stiff, firm, hardened and tender into a soft and almost jelly-like consistency.



Fig. 7.16.1 • Treating the anterior cervical fascia. The current and frequencies can be delivered by graphite gloves worn on the practitioner's hands. The softening and elongation of the fascia in the anterior cervical muscles, especially the scalenes and longus coli, at the same time as the posterior muscles relax and soften, increases cervical range of motion, reduces pain and produces rapid improvement in chronic neck pain.



Fig. 7.16.2 • Treating the cervical spine and shoulder fascia. This contact placement allows all of the cervical nerves, the spinal cord, all of the brachial plexus and all of the muscles of the cervical spine and shoulder to be treated simultaneously. The hands move every two to three seconds, using the finger pads with relaxed fingers to assess and treat the fascia as the frequencies are changed.

The FSM training advises manual therapists to follow the softening with gentle pressure and to change frequencies to address the taut areas that stand out amidst the tissues wth jelly-like consistency.

The treatment protocols for myofascial tissue suggest that the practitioner use frequencies thought to treat 'inflammation in the nerve and the spinal cord' first. This usually produces some softening of the fascia in approximately 80% of patients treated, making it consistent with the current understanding of neurological involvement in myofascial pain. This is followed by protocols to remove 'fibrosis and hardening' from the 'fascia, muscle belly and connective tissue' to address the fascia directly. Treatment for inflammation in the disc, facet joint or ligaments follows if treating the fascia directly does not produce the desired improvement.

However, taut fascia that is a result of visceral irritation, involving the digestive system, kidneys or ovaries, responds most effectively and often responds only to frequencies thought to address the visceral tissue, not the fascia. Trigger points or taut fascia in the lower abdomen that do not respond to the 'treat the nerve, treat the muscle, treat the joint' paradigm disappear in 10 minutes when the frequencies to 'remove inflammation' from 'the ovary' are used. This specificity represents the greatest challenge to practitioners using FSM to treat the fascia. The initial diagnosis must be correct or the practitioner must be willing to modify the preliminary assessment based on feedback and response during treatment.

The model to explain the frequency-specific effect

The model to explain how a frequency-specific effect could operate comes from the realm of quantum and biophysics. Physics has two branches that study in detail the structure, properties and function of matter.

Classical physics provides accurate descriptions of the properties of the body as a large collection of particles but only quantum physics can provide a model for our internal submicroscopic structure and function. The body appears to be a solid object that has all of the properties described by Newtonian physics. It has mass, momentum, inertia, and obeys the law of gravity. But it is, at the same time, an electromagnetic system with all of the properties described by quantum physics. The human body is as much energy as it is matter. This is not an esoteric or spiritual appreciation of the human condition; it is simply basic physics. Using frequencies and current to successfully modify the structure and function of biological tissue brings the practitioner to a practical appreciation of this quantum reality.

Current flow alone in some nonfrequency-specific microcurrent therapies using simple single-channel 0.3 Hz current creates some positive effect in treating the fascia but the most dramatic effects occur in response to specific frequencies.

In a blinded placebo controlled trial in mice, one frequency combination, 40 Hz on channel A (reduce inflammation), and 116 Hz on channel B (the immune system) reduced arachidonic acid induced lipoxygenase (LOX) mediating swelling in a mouse's ear, as measured with calipers, by 62% in 4 minutes. Three unrelated frequency combinations tested in the same model had no effect on inflammation or swelling. According to the researcher who performed the tests, no prescription or nonprescription drug has ever reduced inflammation in this animal model by more than 45% (Reilly et al. 2004).

Scar tissue responds only to specific frequency combinations that cause the tissue to elongate and soften dramatically, allowing increases, even doubling, of range of motion, within 10 to 20 minutes. If the scar tissue is very dense or chronic, the process requires more time and repeated treatment but is usually successful. The frequency to reduce inflammation does nothing for scar tissue; the frequency for removing scar tissue does nothing to reduce inflammation. Regardless of the condition being treated, when the frequency is correct the patient and the practitioner can often feel a sensation of warmth underneath the skin contact as the tissue begins to soften. Explaining these effects is accomplished by exploring the principles of biologic resonance.

Frequencies, measured in hertz (Hz), refer to the number of pulses moving through a conducting medium in 1 second. One hertz is a single waveform passing a fixed point in 1 second. Microcurrent devices usually output square wave pulses containing a large number of high-frequency harmonics instead of using sine waves because the clinical effects were found to be better with square waves. A square wave frequency of 40 Hz is technically a *pulse train* of 40 Hz - 40 square waves that pass a point in space every second. The high-frequency harmonics in the square wave do not change the frequency. Middle C played on the piano will sound different than middle C played on a flute because of the difference in harmonics but the note played is still middle C (Kirsch & Lerner 1998).

The author suggests that the frequencies create tissue changes by the principle of resonance. Resonance is the tendency of a system to oscillate at larger amplitudes in response to some frequencies and not others. Every mechanical system and every chemical bond has a resonant frequency. At the resonant frequency, even small driving forces can produce very large amplitude vibrations. These large amplitude vibrations can cause the system to oscillate so violently that it comes apart. Mechanical resonance destroyed the Tacoma Narrows Bridge when the resonant frequency of the bridge was matched by the frequency of oscillations in the bridge caused by the wind during a rain storm (Billah & Scanlan 1991; Oschman 2000). The resulting violent pendulum effect tore the bridge apart and created a most memorable visual example of the power of resonance. Resonant phenomena occur with every type of vibration or wave and every type of bond and structure.

If every chemical bond and every physical structure has a binding energy that holds it together and has a resonant frequency that will cause it to oscillate, then it is possible to imagine that a resonant frequency exists for every bond that will cause oscillations sufficiently violent to break the bonds that hold the structure together, including the cross-links that hold fascia in a shortened, taut or hardened configuration.

As the bonds began to vibrate, the fluids in the surrounding area would become warm from the friction of the vibration, much as your hands become warm when you rub them together on a cold night. This warming response to vibration could explain why the tissue being treated feels warm when the frequencies are correct.

Conceptual model

Scar tissue can be thought of as a physical structure made of collagen that is wound up tight and coiled in on itself like a rubber band that has been twisted to operate the propeller of a toy airplane. The coiled scar tissue is held together in this configuration by cross-linked bonds that keep it shortened and tight. Think of the collagen coil cross-links as the structure of the Tacoma Narrows Bridge. When the frequencies are used that seem to dissolve scar tissue, the scarring begins to soften almost immediately and over the next few minutes the tissue elongates and continues to soften until it feels almost normal and the range of motion has increased. Once the bonds that hold the coils tight break, the collagen unwinds, and as it elongates the cross-link binding sites are separated and cannot reconnect. In general, as shown in the burn unit project, once scar tissue dissolves it doesn't return. This is a model that has yet to be confirmed but it matches the clinical outcomes and observations over 12 years of use by hundreds of practitioners in thousands of cases. Only further research will confirm or modify the model.

All that is required for resonant phenomena to operate in a biological system are bonds that resonate and a conducting medium to convey specific frequency patterns. All electromagnetic bonds oscillate and the bonds in biological tissue are no exception. The conducting medium is formed when water molecules lining the fascia and the lymphatic and circulatory system vibrate and share electrons in such a way as to create a matrix that acts as a semiconductor (Szent-Gyorgyi 1988; Oschman 2000).

Coherent frequency patterns delivered in conjunction with current flow that increases cellular energy production could reasonably be expected to create a resonant effect. 'Living matter is highly organized and exceedingly sensitive to the information conveyed by coherent signals' (Oschman 2000).

Protein receptors in the cell membrane mediate all cellular functions. When the cell nucleus is removed, cells can still perform their functions normally for up to 30 days through the actions of proteins embedded in the membrane operating in a coordinated self-directed fashion in response to environmental signals such as neurotransmitters, hormones, nutrients, toxins and oxidative stress, emotions, thoughts and electromagnetic signals (Lipton 2008).

Drugs and nutrients act like a key in a lock to alter the configuration of cell membrane proteins and thereby change cell functions. A coherent frequency pattern could alter cell membrane protein configuration and cellular function like the key beeper that opens a car door lock from 20 feet away. This 'key-beeper' model might explain the effects of frequencies on the viscera, discs, facet joints, ligaments, ovaries, kidneys and colon that help fascia to soften and become less painful.

Regardless of the mechanism by which specific frequencies and microamperage microcurrent have their effects, the results achieved in clinical practice warrant further study. The inquisitive, thoughtful and open-minded clinician is encouraged to investigate this technique.

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Surgery and scarring

Willem J Fourie

Introduction

Nature provides us with a means of survival by restoring tissue integrity via granulation scar tissue in response to damage. While nonsurgical insults such as infection, chemotherapy, radiation and cancer may damage tissue and initiate the healing cascade, a common trigger to tissue healing and scarring is still injury and surgery. Maintenance of our well-being depends on the body's ability to guide the insult through an appropriate sequence of repair without complications.

For open wounds (including surgery) and severe internal tears (ruptured tendon or ligament) wound closure and tissue strength are critical and a certain amount of scarring is necessary and inevitable. Scars differentiate and attempt to become quasitissue specific in response to internal and external influences. Filling defects in loose, flexible tissue, scar tissue will change to duplicate the same tissue characteristics as far as possible in the final stages of healing. Impaired mobility of soft tissues can contribute to chronic pain and tissue stiffness as well as abnormal movement patterns within the musculoskeletal system (Bouffard et al. 2008).

Although all wounds pass through the same mechanism of repair towards full recovery, the final cosmetic and functional result may differ markedly. The ideal is for a scar first to close the wound and establish tissue stability and secondly to blend cosmetically with surrounding tissue, allowing pre-injury function.

The extent of the problem

Successful healing does not automatically correlate with return to full function. If, for example, a repaired tendon develops normal tensile strength but does not glide, it is a functional failure. Postsurgical adhesions result from injured tissues (following incision, cauterization, suturing or other means of trauma) fusing together to create abnormal connections between two normally separate surfaces of the body (Ergul & Korukluoglu 2008). Outcomes differ depending on the injured tissue, type of injury, genetic factors, and the presence of systemic disease.

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Alterations in the normal healing response could be associated with severe after-effects, either as a failure to heal (wound failure), or excessive repair including hypertrophic scarring, keloids, contracture, and adhesions (Occleston et al. 2008). For most patients, adhesions have little effect, while others may develop considerable clinical consequences, for example:

- After laparotomy, almost 95% of patients are shown to have adhesions at later surgery (Ellis et al. 1999). Intestinal obstruction, chronic abdominal and pelvic pain, female infertility and difficult reoperation with bigger risk are also reported (Ergul & Korukluoglu 2008; Salim et al. 2008).
- Even minimally invasive surgical procedures (e.g., arthroscopy) are reported as contributing to increased risk in developing knee osteoarthritis (Ogilvie-Harris & Choi 2000). This can be associated with surgical difficulties and

postoperative complications in primary total knee arthroplasty (Piedade et al. 2009).

- Previous abdominal surgery has been shown to be a factor in low backache, myofascial pain syndromes (Lewit & Olsanska 2004), and in compromised vascular anatomy of the abdominal wall (Rozen et al. in press).
- Adhesions, tissue fibrosis and loss of tissue glide between structures can be identified as the source of pain and restriction of movement and function in up to 72% of patients after surgery for breast cancer (Lee et al. 2009).

Understand fascial relationships

Fascia plays an important role in the body's musculoskeletal dynamics (Stecco & Stecco 2009). This includes tension transfer across the epimysium (the fibrous envelope surrounding a muscle), and between muscles (Huijing 2007). It further contributes to the development of muscle force (Aspden 1990) and functions as a responsive, dynamic and complex mechanosensitive system for coordinated movement (Schleip et al. 2006). Fascia controls the *quality* of movement while keeping the bony levers and spacers within a specific functional configuration.

A full understanding of fascial arrangements and their behavior under load is needed to understand how fascial restrictions can contribute to pain and dysfunction.

Anatomy of tissue layers

The body is arranged in several layers, from superficial to deep (Stecco & Stecco 2009):

- The skin is formed by the epidermis and dermis.
- The superficial fascia consists of two or more adipose, loose connective tissue layers separated by a membranous layer(s) of collagen and elastic fibres.
- The deep fascia envelops the large muscles of the trunk and forms fascial sleeves in the limbs.
 - In the trunk, the deep fascia is subdivided into three laminae. Each lamina is, in turn, bilaminated to accommodate superficial, intermediate or deep muscles in the trunk and neck. Thin layers of loose adipose tissue separate the various fascial laminae, allowing gliding between layers.

- Deep fascia in the limbs mostly glides over the muscles.
- The epimysial fascia lies beneath the deep fascia of the limbs. This interface consists of three distinct layers: the deep fascia, the fibrous envelope of the muscle (epimysium) and a loose areolar tissue layer between the deep fascia and epimysium (McCombe et al. 2001). The deep fascia of the trunk is often fused with and becomes the epimysial fascia for the muscles.

The superficial fascia allows muscles to slide beneath the skin as they contract, while the deep fascia synchronises motor activity in order to produce smooth, resistance-free economical movements (Stecco & Stecco 2009).

Surgery

The body's response to injury, either surgically or traumatically induced, is immediate. It signals repair to begin. Three dominant phases can be described: the inflammatory phase preparing the area for healing, the fibroplastic phase rebuilding the structure, and the remodeling phase providing the final form. One process is stimulated to begin, and its completion, in turn, signals another cellular response until the wound is bridged by scar. Different tissues heal at different rates. One wound can show various areas in different stages of healing. All surgery carries a risk of adhesions between tissues resulting in dysfunction in the form of restricted tissue glide, muscle imbalances, weakness, or loss of flexibility (Fig. 7.17.1). Disturbed patterns may even become evident some distance from the scar.

Inflammation is crucial to the healing response. It continues throughout all healing phases, stimulating and coordinating the functions of wound repair. Inflammatory mediators regulate all aspects of tissue healing and remodeling, whether planned (as in development) or unplanned (as in tissue repair after injury). One mediator, the cytokine transforming growth factor beta 1 (TGF- β 1), is unique in its wide-spread actions; from enhancing the deposition of extracellular matrix to acting as a potent regulator of repair by coordinating or suppressing the actions of other growth factors, cytokines and mediators (Henry & Garner 2003).

A prolonged inflammatory phase after surgery may result in proliferative scarring and increased fibrosis within the damaged area. Fibrosis represents **Fig. 7.17.1** • A A surgical scar in the deltoid muscle after an open reduction with internal fixation of a fracture of the humerus head. B Attempted external rotation of the arm. Note the adherence of the scar to deeper tissue layers.



a pathologic excess of normal tissue repair. Excessive or sustained production of TGF- β l is a key molecular mediator of tissue fibrosis. It consistently and powerfully acts on cells to encourage the deposition of extracellular matrix.

The connective tissue response to the internal (inflammatory mediators and growth factors) and external (motion and directional strain) stresses applied will determine how the scar matures. Thus the scar can become either dense and unyielding (Fig. 7.17.2) or pliable and mobile. Remodeling is not restricted to the injured area only. Neighboring, noninjured tissue also changes its collagen production rate in response to inflammation.

Every normal movement is accompanied by stretching, gliding and/or shifting within the



Fig. 7.17.2 • An extended field of view ultrasound image of the same shoulder showing the extent and depth of soft tissue scarring and adhesions. Note the destruction of superficial fascia continuity between the arrowheads.

surrounding soft tissue. Compromised soft tissue mobility may therefore impair motor function. Distortion of surrounding myofascial relationships can alter synergistic and antagonistic muscle balances and proprioception (Stecco & Stecco 2009). Adaptive patterns to complete or execute a movement and straining to overcome tissue resistance to produce movement are energy expensive and run the risk of producing further tissue damage (Manheim 2001). The ultimate outcome could be global dysfunction. Damaged tissues and structures need repair before the system can function efficiently again.

Treatment

Rehabilitation concerns the continuum of repair to full function while guiding the wound to return as close to pre-surgery architecture as possible.

The treatment and management of surgical scarring falls into two main categories:

- early management to guide the healing tissue to return as close to pre-surgery architecture as possible
- late treatment to address dysfunction that might have developed due to scarring and adhesions.

Vigorous therapy used too early can stimulate inflammation and edema, prolong the inflammatory phase, or disrupt the wound. Forceful mobilization aimed at breaking established scar tissue may create a new inflammatory response, ultimately causing further scar formation. A secondarily inflamed wound results in additional collagen deposition, compounding existing morbidity.

Therapeutic intention

With the cytokine TGF- β responsible for the secretion of collagen by fibroblasts, controlling and limiting inflammation is important in early wound care. Overstimulation of TGF- β may be implicated in the overproduction of new collagen, resulting in hypertrophic scarring and tissue fibrosis. Apart from drug and gene approaches to wound healing, the role of manual therapy also needs acknowledgment.

Scars are treated for mainly two reasons:

- to aid in controlling the inflammation and swelling in the early stages of wound healing
- to restore tissue function and gliding.

In a laboratory model Bouffard et al. (2008) used treatment sessions of stretching surgically induced scar tissue for 10 minutes twice daily for 7 days by applying a stretch that lengthened the damaged tissue area by 20% to 30%. They concluded that brief, moderate amplitude stretching (at 20-30% strain) of connective tissue decreases both TGF- β and collagen synthesis, thus reducing cross-binding adhesions between tissue layers. In-vitro studies by Yang et al. (2005) showed that repetitive stretching of tendons at small amplitude (< 4%) seemed to be anti-inflammatory and to restore tendon homeostasis, while large-amplitude stretching seemed to be pro-inflammatory in nature. Benjamin et al. (2008) remark that if these findings also prove to be applicable in vivo, it can then be assumed that regulated movement at moderate intensity might be beneficial for reducing inflammation. With less fibrosis and better tissue glide, less effort will be needed to produce normal movement and overcome tissue resistance. Reduced internal strain decreases treatment pain with better compliance to prescribed exercises. By being gentle early, rehabilitation can progress faster at later stages.

Before we start

The barrier phenomenon

Similar to joints, soft tissue has a specified range of available movement that can be divided into a physiologic and an anatomic range of movement:

- Physiologic range is necessary for smooth, unrestricted movement of underlying structures during normal movement (active range of movement).
- Anatomic range refers to where tissue can be stretched beyond the physiologic range and before

coming to a stop without discomfort or pain (passive range of movement).

• The distance between physiologic and anatomic limits constitutes a 'safety zone' protecting the body from damage should external forces be applied.

As in joints, there is a range within which minimal resistance to stretch or shift is encountered. When resistance is met, the anatomic barrier is reached. Under normal conditions, the barrier has a soft, elastic end-feel and can be moved easily, accompanied by a sensation that no unnecessary tension is present in the target tissue.

In a *pathological* barrier, the anatomic (passive) tissue range is reached prematurely. This barrier characteristically has a tense, restrictive feel, with an abrupt, hard or leathery end-feel. Normal physiologic movement may still be present with no apparent movement restriction, but there will be reduced protection when the tissue is strained.

Depth and grading of touch

An advantage of manual techniques is that the hand is a sensitive instrument which establishes a feedback relationship with the manipulated tissue. When treating wounds and scarring, the therapist should have a clear understanding of how deep and firmly to work. A grading scale of 1 to 10 could be used (Fourie & Robb 2009):

- Grade 1 to 3: Very light, mild and non-irritating. It feels like moving the eyelid on the eyeball without irritating the eye. No discomfort.
- Grade 4 to 6: Moderate to firm. This is where most massage techniques are performed. There may be mild discomfort, but no damage to tissue.
- Grade 7 and 8: Firm, deep and uncomfortable pressure with discomfort, but tolerable. There is a potential for tissue bruising. Trigger point work would be performed at this level.
- Grade 9 and 10: Deep, very uncomfortable or painful with a good potential for tissue damage. It is often described as 'surgery without anaesthesia.' An example of this grade would be deep transverse friction.

Evaluation

Scar evaluation aims to determine the quality, extent and depth of the 'premature or pathologic' tissue barrier.

- Quality refers to the perceived end-feel a normal soft, elastic or an abnormal solid, abrupt end-feel.
- The extent of the barrier refers to *where* in the available range resistance is encountered, and how large an area is involved.
- The depth of the tissue barrier may be subjective but an attempt should be made to distinguish between *which tissue layers* restrictions are felt: superficial between dermis and deep fascia, deep restrictions between muscles, organs or between a tendon and its sheath.

Three layers of fascial glide need assessment:

- Skin and superficial fascia manually glide the skin ON the deep fascia. Move hand and skin as a unit to the end of available tissue glide using a pressure grading of 2 to 3.
- Deep fascia and myofascial interfaces move one deep structure ON another. Change hand or finger position accordingly and glide tissue at a firm pressure grading of 4 to 6.
- Deep muscle and soft tissue on bone interfaces modify hand and/or finger position to test for specific directional restrictions with fingertip or thumb pressure at a pressure grade of 6 to 8. This may be experienced as discomfort by the patient and should be done with care.

This is an assessment of tissue *movement*, not of painful areas within the soft tissue. Palpation is for tissue mobility, flexibility and freedom of tissue glide. The position and direction of tight, hypomobile or inflexible tissue should be documented.

Four directions of scar movement need assessment:

- longitudinal along the length of the scar
- transverse across the long axis of the scar
- rotation clockwise and anticlockwise
- lifting the scar vertically away from deeper layers.

Treatment

Both the source of a restriction and the extent of the resultant dysfunction guide treatment. Primary treatment is directed at the restricted tissue glide (local source of the problem) before rehabilitating the abnormal condition that the patient presents with (the dysfunction). There must be clarity about the depth, site and extent of restricted tissue gliding.

Principles

- Treatment is directed at the mechanical restriction identified by evaluation.
- The goal is to move the tissue barrier towards a normal end-feel and amplitude.
- Approach treatment in a layered fashion, clearing one layer or compartment of restrictions before moving to a deeper or adjacent layer.
- Techniques are performed at or just before the palpable tissue barrier, at varying angles to the restriction.
- Use gentle touch grading during the early stages. For mature, chronically adhered scars more forceful treatment may be necessary.

How to treat

The use of gentle treatment in the early stages of healing safeguards against causing wound breakdown and increasing inflammation. For long-standing scars and adhesions, higher touch grades can be used. Care must, however, be taken to avoid triggering a *new* inflammatory response.

Approaches to engage and move the tissue barrier include (Lewit & Olsanska 2004):

- Engage the barrier directly and wait with a sustained pressure until the tissue releases and the barrier shifts after a short delay.
- Use a sustained stretch of the scarred tissue. Stretch could be uni- or multidirectional.
- Apply slow, rhythmic mobilizations towards and into the tissue barrier. Movement direction could be perpendicular to, at an angle to, or away from the tissue barrier.

Basic techniques

Gross stretch

This is the most superficial and least painful scar technique. By using finger or full-hand contact, take up all the tissue slack and apply a gentle stretch along the length of the scar. Hold, wait for release, and stretch again. Change hand position and repeat the stretch perpendicular to the original stretch. Repeat the stretch sequence diagonal to the previous position. Continue to stretch across the scar in a radiating pattern until no further stretch is possible (Fig. 7.17.3).

Gentle circles

With this technique, the fingers move the skin ON the deep fascia. Tissue movement is of an engaged shearing nature. Rest the fingers on the part to be treated (next to the scar). The heel of the hand may also rest on the body. Starting at 6 o'clock, push the skin around in a circle with the middle three fingers as if following the arms of a clock. Slowly move the skin towards the scar to engage and shear the tissue barrier while keeping the circle round and the pressure and pace even (Fig. 7.17.4A–D).



Fig. 7.17.3 • A Gross stretch with full hand contact. Longitudinal, transverse
Change hand position, repeat the circle and release. Treat the full length of the scar and repeat several times in a session, if needed. Alternatively, start the circle at 12 o' clock and pull down and stretch the skin and scar.

Firm upside down 'J' stroke

This technique is very similar to the previous one in starting position and depth. The stroke is directed towards the scar from about 1 inch (2.5 cm) away. Movement is slow and deliberate into the tissue barrier. When the barrier is engaged, the fingers shear away towards the left or right and the tissue is allowed to return to its nonstretched position. Repeat gently until the tissue barrier has moved, or discomfort subsides (Fig. 7.17.4C).

Vertical lifts

Vertical lifts are used to treat any scar that can be gripped between thumb and fingers. Grip an area of the scar and gently but firmly apply a vertical stretch.

Fig. 7.17.5 • A and B Sliding towards and 'under' the scar.

Hold, wait for release, and increase the stretch (Figs 7.17.5, 7.17.6). When no further stretch is available, change the angle of stretch while maintaining the vertical lift. Repeat the lift sequence from different angles until no further stretch is available.

Skin rolling

For adhesions between skin and superficial tissue and older established scars, lift the skin between thumb and fingers and gently roll the skin over the area or scar (Fig. 7.17.7). It may be necessary to repeat the rolling technique a few times over the same area in order to release any long-term adhesions.

Regardless of the modality used, an impairmentbased approach is recommended for treatment of restrictive scars and adhesions. The selection of technique, direction and depth is based on the level of dysfunction revealed during the assessment. This approach gives the therapist the flexibility to adapt treatment to the person, rather than treating the 'diagnosis.' Further, based on the treatment response,

B

Fig. 7.17.6 • A and B Vertical lifts.





Fig. 7.17.7 • A, B, C Skin rolling.

treatment can be modified in line with the patient's improvement or lack of progress (Fourie & Robb 2009).

When understanding the basic layered arrangement of tissue, the wound healing process and how to grade touch, any manual therapy or massage technique can be modified to achieve the aims of restoring tissue mobility, glide and flexibility.

Treatment is discontinued when the release has been completed in all directions and layers. This may not happen in a single treatment and might even take several months, especially in long-standing chronic scars. Care should be taken not to create wound breakdown or an inflammatory response to tissue mobilization.

Conclusion

In many cases, the problem may be irreversible, with scars becoming so fixed and strong that only surgery will release the adhesion. In established, fixed scars, where no tissue gliding is possible by manual means, treatment is aimed at creating more soft tissue space and flexibility in the surrounding tissue. In many cases, adhesive scarring might affect quality of life adversely; however, open, positive discussion with adequate explanation and intervention may vastly diminish the patient's anxiety, suffering and disability.

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7.18

Temperature effects on fascia

Werner Klingler

The use of heat is a common tool in the treatment of muscular disorders such as stiffness or myalgia. Early reports of heat-induced relaxation in connective tissue date back more than half a century (Rigby et al. 1959). Clinical data as well as in-vitro experiments demonstrate that heat in the therapeutic range leads to a temperature-dependent myofascial relaxation (Lehmann et al. 1970; Warren et al. 1971; Muraoka et al. 2005). In this chapter, the influence of temperature on the myofascial system under physiological and pathological conditions is addressed. Perspectives of therapeutic use and the effect on resting muscle tone are given.

Under physiological conditions the skeletal muscle and the fascial components interact closely. The sophisticated motor system enables humans to lift heavy weights as well as being able to perform the most fast and graceful movements, such as playing the piano. Skeletal muscle is composed of myofibers. which are formed by confluent and organized muscle cells. Contraction is initiated by calcium (Ca^{2+}) release from internal stores (sarcoplasmic reticulum), which activates the contractile proteins. Myosin is an enzyme, which acts as an adenosin triphosphate phosphatase (ATPase) and generates force by cross-bridge cycling against the actin filaments. Muscle relaxation is mediated by Ca²⁺ re-absorption into the sarcoplasmic reticulum by an ATP-consuming pump (sarcoendoplasmatic reticulum Ca²⁺ reuptake ATPase, or SERCA). The energy is replenished by glycolysis and the respiratory chain. All these enzymatic processes are temperature dependent and obey the logarithmic Arrhenius law (Fig 7.18.1).

Isolated fascia has a lower metabolic activity and contains fewer cells and mitochondria than muscle bundles. The viscoelasticity is determined by the specific composition and properties of the filaments, the chemical bonds, and other factors such as hydration. Several studies show that the viscoelastic properties of fascia are temperature dependent, although assessment is not easy, due to technical pitfalls (Lam et al. 1990). Temperature increase in fascia of up 40°C leads to reduced stiffness and more rapid elongation of the tissue, which in part can be attributed to a higher extensibility of collagen (Lehmann et al. 1970; Warren et al. 1971; Ciccone et al. 2006; Bass et al. 2007; Huang et al. 2009; personal data). In other words, there is a heat-induced fascial relaxation (Fig 7.18.2). Vice versa, passive cooling leads to an increase in stiffness (Muraoka et al. 2005). Spinal ligaments extend with temperature, as well. In sheep, this thermal expansion has been quantified to roughly 0.5 mm per lumbar segment (Hasberry & Pearcy 1986).

At first sight, skeletal muscle and adjacent fasciae seem to have conflicting features regarding temperature dependence. However, from a physiological point of view, the combination makes sense. Temperature distribution throughout the body is nonuniform.

Working muscle produces heat which facilitates metabolism in terms of a positive feedback loop. Most notably, Ca^{2+} turnover is actuated, which enhances muscle excitability and contraction. An increase of several degrees Celsius, e.g., in limbs from roughly 33°C to 39°C, leads to significantly higher viscoelasticity of fascia. In this case, muscle is less limited by fascial resistance and range of motion is increased. Hence, in most situations, this resembles a gain of function during exercise. At lower



■ Relaxation time ■ Time to peak ■ Time to peak ■ Time to peak ■ Time to peak ■ Time to peak ■ Time to peak ■ Time to peak ■ to peak

Fig. 7.18.1 • Contraction and relaxation parameters of skeletal muscle. A Freshly dissected muscle strips from human *m. gastrocnemius* were placed in a physiological organ bath. Force registrations were performed under electrical stimulation of the tissue (0.1 Hz; 1 ms; 25 V). Twitching is clearly temperature dependent. B The contraction and relaxation parameters of skeletal muscle (human *m. gastrocnemius*, mean \pm SEM, n = 10) obey the logarithmic kinetics of biological reactions.



Fig. 7.18.2 • Stretch response of fascia at different temperatures. A This original force registration shows a response of 4% stretch in *fascia thoracolumbalis*. In a cold environment (20°C), the fascia has a higher peak force and slowed relaxation compared to 40°C. B Cartoon of temperature effects on fasciae.

temperatures, i.e., at rest, the viscoelastic properties are adapted to serve stabilization and load-bearing function.

Painful contractures and reduced range of motion are frequently associated with rigid collagenous tissue within and surrounding skeletal muscle, as well as other connective tissue involved in force transmission. The fascial function, such as in joint capsules, tendons, or epi- and endomysium may be disrupted by trauma and/or inflammation. Although mediated differently, fascial dysfunction can also be caused by central nervous lesions, e.g., stroke. Undamped firing of the lower motoneuron leads to a perpetual overstimulation of dependent motor units and secondarily to fascial injuries.

In both cases, the myofascial imbalance results in tissue remodeling. Histologically, these areas show an involution of myofibers, an increase of connective tissue, an altered composition of elastin/collagen and invasion of myofibroblasts. These cells are typically found in scar tissue and exhibit contractile properties. The forces are strong enough to adapt wounds. There are several syndromes which describe fascial contractures, e.g., frozen shoulder, Dupuytren's disease, Peyronie's disease, and many others (Schleip et al. 2005).

In contrast to myofibers (skeletal muscle), the contractile properties of myofibroblasts are slow and smooth muscle-like. The activation of myosin light-chain kinase (MLCK), via a Ca^{2+} –calmodulin complex, is rudimentary and subordinate in myofibroblasts. The main biochemical pathway involves Rho-kinase, which is Ca^{2+} independent and inhibits myosin phosphatase. This, in turn, leads to a sustained and energy-saving contraction. The temperature dependence of myofibroblasts has not yet been evaluated systematically.

Under pathological conditions the portion of connective tissue increases. The viscoelastic properties determine range of motion, myofascial (im) balance and painful contractions. Under these circumstances it is unlikely that an individual could exercise the affected limb in a manner in which muscular activity could lead to significant heat generation. The lack of the physiological benefits of temperature-induced relaxation might support the beginning of a vicious circle. Unused skeletal muscle disappears and is replaced by connective tissue, which further limits movements. Increased susceptibility of fascial injuries, in the cold, is attributed to more rigid tissue response (Bass et al. 2007).

Applying heat in the therapeutic range, which means up to 40°C, may be one means by which to prevent the negative feedback loop. It has been shown that external application of heat increases the range of motion after development of a knee joint contracture (Usuba et al. 2006). In addition, the internal thermal effects generated by ultrasound may lead to an increased range of motion (Draper & Ricard 1995). Technically, there are many other ways of warming up myofascial tissue, e.g., hot bathtub, short wave diathermy or the transdermal application of pharmaceuticals, which increase regional blood flow.

The contraindications to heat application include acute inflammatory diseases, skin lesions and peripheral neuropathy because of the risk of burns. Heat effects are not confined to the biomechanical properties. There is also an influence on the central nervous system and peripheral nociceptors. However, the interaction between thermal and nociceptive pathways, and the relationship to pain perception, is a complex topic, which remains controversial (Green 2004).

In summary, heat in the therapeutic range leads to relaxation of many fascial contractures associated with myofascial dysfunction. In patients with low back pain, ruptures of the *Fascia thoracolumbalis* with prolapse of fatty tissue and muscle have been observed (Dittrich 1963; Faille 1978). Thickened fascia in patients with low back pain may be the correlate of fascial scarring (Langevin et al. 2009). Again, external heat application has been shown to be beneficial in low back pain in a Cochrane review (French et al. 2006). Warming up the 'frozen lumbars' may help the individual to stay active and reduce disability.

The lesson we learn from the differential temperature effects on fasciae and myofibers also helps to explain passive muscle tone (Simons & Mense 1998). Warmth leads to enhanced skeletal muscle excitability, faster contraction and relaxation parameters, as well as increased force generation. On the other hand, higher temperature leads to heat relaxation and reduced myofascial stiffness in vitro. Given that there is no voluntary innervation, this effect can also be observed in vivo. Hence, the regulation of fascial stiffness plays a major role in EMG-silent resting muscle tone.

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Neurodynamics: Movement for neuropathic pain states

Michel W Coppieters Robert J Nee

Introduction

The nervous system is a remarkable organ system. In many ways, it is well protected and strong. On average, 50% of a peripheral nerve consists of connective tissue (ranging from 22% to as high as 80%) (Sunderland & Bradley 1949). In addition, there are many other anatomical design features that enable the nervous system to handle the significant mechanical demands that are placed upon it during activities. Complete nerve lesions, such as nerve root avulsions, are rare and require a high-impact or high-velocity trauma.

This macroscopic robustness is, however, somewhat deceptive. Relatively small pressures may trigger a cascade of immune-inflammatory responses, resulting in peripheral neuropathic pain. Certain parts of the nervous system are less protected and more vulnerable to injury. The nerve root, for example, lacks some of the connective tissue layers of the peripheral nerve (epineurium and perineurium, see below), which reduces its protection against compression and inflammation associated with disc or joint pathology. Neuropathies such as carpal tunnel syndrome (CTS), cubital tunnel syndrome, and tarsal tunnel syndrome indicate that the nervous system is also more vulnerable in confined spaces where an increase in pressure will result in nerve compression.

Considering the relatively low prevalence of acute traumatic nerve injury, it is somewhat surprising that the classification system for nerve disorders most commonly used to date was developed more than 60 years ago during World War II. Seddon's classification describes three basic types of nerve injuries according to severity: neuropraxia, axonotmesis and neurotmesis (Seddon 1943). It is important to emphasize that this grading scheme and Sunderland's modification of the scheme (Sunderland 1951) are both based on gross pathology and loss of nerve function (impulse conduction). Negative symptomatology, such as loss of sensation (numbness) and loss of muscle strength (weakness) can be easily explained by the varying degrees of demyelination and axonal loss as described for the different categories. However, important positive symptomatology, such as neuropathic pain, is not properly accounted for in these classifications.

Sunderland acknowledged that many peripheral neuropathies may exist where altered nerve conduction may not be severe enough to be classified as a neuropraxia (Sunderland 1978). Due to a lack of understanding at the time, he could only refer to these disorders as *'irritative lesions'* or *'perverted nerves.'* Thanks to advances in neuroscience over the last decades, a much better knowledge of the pathophysiology of these nerve injuries is now available. Before discussing movement as a treatment modality for neuropathic pain states, we need to briefly review the structure, function and pathophysiology of the peripheral nervous system.

Structure, function and pathophysiology of the peripheral nervous system

Neurons are the core components of the nervous system and typically consist of a cell body, dendrites and an axon. Approximately every 1 to 2 mm, myelinated fibers possess unmyelinated gaps (nodes of Ranvier) where many ion channels are embedded in the membrane. These channels enable electrically charged atoms to transfer across the axolemma and give neurons the property of excitability. The action potential can jump from node to node, resulting in a faster, saltatory conduction. Myelin is produced and wrapped around the axons by Schwann cells. These cells also contribute to the immune response by the release of immune compounds, such as pro-inflammatory cytokines, which may contribute to pain and inflammation. In unmyelinated fibers, Schwann cells wrap around a single axon or group of axons, forming Remak bundles.

A peripheral nerve consists of multiple neurons, blood vessels and various connective tissue sheaths. The endoneurial tubule is the connective tissue sheath that surrounds an individual myelinated fiber or group of unmyelinated fibers. Several tubules are surrounded by perineurium to form fascicles. Fascicles are embedded in and surrounded by epineurium. The collagen fibers in the connective tissue sheaths interlace in all directions to form a strong and irregular network. A thin mesoneurium surrounds the peripheral nerve, which helps the nerve to slide relative to neighboring tissues.

The connective tissue sheaths are innervated by the nervi nervorum (Sauer et al. 1999). The nerve trunk is also sympathetically innervated via the perivascular plexuses of the blood vessels which enter the nerve (Bove & Light 1997). The small nervi nervorum are predominantly unmyelinated, and at least some function as nociceptors. As a result, the connective tissues may contribute to a pain experience, theoretically irrespective of pathological changes in conductive nerve fibers.

A very well-developed system of extraneural and intraneural blood vessels supplies the nerve. The blood vessels pierce the different connective tissue sheaths to provide oxygen and essential nutrients to the cells. The endothelium cells which line the interior surface of the endoneurial capillary bed, together with the perineurium, form the bloodnerve barrier (Yayama et al. 2010). Similar to the blood-brain barrier, it is designed to keep unwanted substances out of the perineurial space. Compression and intraneural ischemia (Yayama et al. 2010) and intraneural activated immune cells (Spies et al. 1995) may cause focal breakdown of the blood-nerve barrier. Sunderland (1976) described three pathological stages following persistent pressure in or around the nerve: hypoxia, edema and fibrosis. Pressure may

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lead to venous stasis. Ischemia and hypoxia may cause pain and other symptoms, such as paresthesia. If hypoxia continues, the blood–nerve barrier will break down, resulting in accumulation of proteins and edema. Because no lymphatic vessels cross the blood–nerve barrier, it may take a long time for edema within the perineurium and endoneurium to be reabsorbed (Rempel et al. 1999). Lymphocytes, fibroblasts, and macrophages will intrude as a reaction to previously shielded antigens contained within the perineurial space. This will initiate inflammation and eventually fibrosis or scar formation in the subperineurial space. Thickening of the epineurium and perineurial connective tissue sheath is also observed (Mackinnon 2002).

As a comprehensive overview of nerve pathophysiology is beyond the scope of this chapter, the reader is referred to selected reviews for further information (Rempel et al. 1999; Mackinnon 2002).

The double crush theory

The double crush theory states that axons which are compressed in one region become susceptible to damage at another site (Upton & McComas 1973). The basis for this hypothesis was the observation of a high prevalence of cervical radiculopathy in patients with CTS (5-18%) (Morgan & Wilbourn 1998; Moghtaderi & Izadi 2008), compared to less than 1% in the general population (Radhakrishnan et al. 1994). Although first formulated almost 40 years ago, little is known about possible underlying mechanisms to support the theory, and the existence of the phenomenon is often questioned. In a recent Delphi study (Schmid & Coppieters 2010), we asked a panel of international experts in peripheral nerve pathology to indicate their level of agreement with the statement that a nerve disorder is a predisposition for the development of a second nerve disorder. Two-thirds of the experts either agreed or strongly agreed with the statement, whereas one-third disagreed. When asked to list mechanisms to explain dual or multiple nerve injuries, the experts listed 22 possible processes. Previously suggested mechanisms such as impaired axonal transport or altered nerve biomechanics were included. In addition, many new mechanisms not previously linked to dual nerve disorders were suggested, such as ion channel alterations and immune-inflammation of the peripheral nerve, dorsal root ganglion, spinal cord, and higher pain centers.

Many clinicians have embraced the double crush theory as it provides a rationale for considering the health of the entire nervous system and its surrounding structures when examining and treating patients with neuropathic pain. It has been argued that failure to diagnose and treat these multiple levels of injury will result in failure to relieve patients' symptoms (Mackinnon 2002). However, due to a lack of research, clinical guidelines refrain from making treatment recommendations for patients with dual nerve disorders, such as cervical radiculopathy and CTS (American Academy of Orthopaedic Surgeons, 2008), and much basic research is needed before possible mechanisms for dual nerve disorders can be substantiated.

Movement for neuropathic pain states

There is relatively little research that has investigated the effects of movement on neuropathic pain, especially studies involving humans. A recent animal study revealed that an extended exercise program reduced signs of neuropathic pain in rodents who had sustained partial sciatic nerve injury (Kuphal et al. 2007). Following exercise, there was a reduction in cold allodynia and thermal hyperalgesia. In this section, the focus will be on one type of therapeutic movement, namely neurodynamic exercises or nerve gliding exercises.

Neurodynamic exercises: 'sliders' slide and 'tensioners' tension

Historically, the first mobilization techniques for the nervous system resembled neurodynamic tests, or 'tension tests' as they were initially called. Several biomechanical studies have revealed that the nervous system slides considerably relative to its surrounding structures and that strain in the nervous system increases substantially during neurodynamic tests or individual components of the test (Byl et al. 2002; Wright et al. 2005; Coppieters et al. 2006; Gilbert et al. 2007; Coppieters & Butler 2008). Because of the increase in strain, these initial mobilization techniques are now often referred to as 'tensioning techniques for the median nerve are elbow extension combined with wrist extension (Fig. 7.19.1B), elbow

extension with cervical contralateral side bending (Fig. 7.19.2B), or the combination of wrist and finger extension (Fig. 7.19.3). Obviously, exercises can include various combinations and can be much more functional than the techniques mentioned here, which have been subjected to biomechanical analysis (see below).

Even when movement-based management strategies are considered appropriate, the increase in nerve strain associated with tensioning techniques is often not deemed suitable (Coppieters & Butler 2008). For this reason, techniques consisting of a combination of movements in which elongation of the nerve bed at one joint is simultaneously counterbalanced by a reduction in the length of the nerve bed at an adjacent joint have been promoted (Butler 2000; Coppieters et al. 2004). These techniques were labeled 'sliding techniques,' because the clinical assumption is that, compared to tensioning techniques, sliding techniques result not only in a larger longitudinal excursion of the nerve relative to surrounding structures, but are also not associated with significant increases in strain. Sliding techniques are considered less aggressive and may be more appropriate for more acute injuries, in postoperative management and even for conditions characterized by inflammation around the nerve where large excursions may help disperse some of the inflammatory mediators (Coppieters & Butler 2008). In addition, neurodynamic exercise may contribute to the prevention of adhesions following surgery, may reduce endoneurial fluid pressure and edema, preventing progression to a fibrotic stage, and may limit hypoxia by improving intraneural microcirculation (Coppieters & Butler 2008).

Although sliding techniques were suggested more than a decade ago (Butler 2000), it wasn't until recently that a series of studies was conducted to test the clinical assumptions regarding the different biomechanical effects of different types of neurodynamic exercises. Longitudinal excursion and strain in the median nerve was measured during sliding and tensioning techniques and during single joint movements in cadaveric experiments (Coppieters & Alshami 2007) and in an in-vivo study using dynamic ultrasound imaging (Coppieters et al. 2009). Figure 7.19.1 and Figure 7.19.2 clearly demonstrate that different types of nerve gliding exercises have very different mechanical effects on the nervous system. The studies confirmed the clinical assumptions that with sliding techniques large longitudinal excursions of the nerve can be obtained without





Fig. 7.19.2 • Example of a sliding and tensioning technique for the median nerve involving the elbow and neck. Longitudinal excursion (in millimeters) of the median nerve in the upper arm (as indicated by the bar charts) was largest for the sliding technique (p < 0.0001), compared to the tensioning technique or single joint movements of elbow or neck (*not shown*). In the sliding technique, the distal movement of the median nerve associated with elbow extension is facilitated by the relaxation of the brachial plexus as a result of cervical ipsilateral side bending. In the tensioning technique, the distal movement of the median nerve is impeded by an increased tension in the nervous system resulting from cervical contralateral side bending. The gray shaded areas show the starting position; the arrows indicate the movement to reach the end position (*unshaded*). From Coppieters et al, J Orthop Sports Phys Ther. 2009;39(3):164–171, with permission.

significant increases in nerve strain. Longitudinal excursion and nerve strain associated with a particular joint movement were strongly influenced by the position or simultaneous movement of an adjacent joint. This biomechanical insight is valuable when designing progressive exercise programs for patients with neuropathic pain.

The fact that strain and nerve movement are transmitted along long sections of the nervous system and well beyond the proximity of the moving joint also demonstrates that it is virtually impossible to immobilize the nervous system by restricting movement of one or more joints. Wearing a splint is commonly advocated for certain entrapment neuropathies, such as CTS (American Academy of Orthopedic Surgeons, 2008; de Krom et al. 2008). Partial immobilization may indeed result in temporary relief in certain situations. Depending on the clinician's treatment philosophy, though, this partial immobilization can also be considered as controlled mobilization and as a first step in a progressive exercise program if symptoms are severe. Although large clinical trials are underway, currently there is still a lack of research to support either approach for CTS or other conditions. Studies have, however, demonstrated that even after surgical procedures such as ulnar nerve transposition, immediate mobilization results in more favorable results compared to delayed mobilization (Weirich et al. 1998).

Besides the biomechanical analysis of neurodynamic exercises, there is a growing interest in the hypoalgesic effects of these techniques. Thermal quantitative sensory testing procedures were used to evaluate the effect of a neurodynamic tensioning technique in healthy participants (Beneciuk et al. 2009). Compared to a sham intervention, an

Fig. 7.19.1 • Strain and longitudinal excursion of the median nerve at the wrist during combined elbow and wrist movements (A-B) and single joint movements of the elbow or wrist (C–F). For each of the six conditions, the corresponding diagrams in the middle column consist of three waveforms: the top waveform represents the change in nerve strain, the middle and bottom waveforms show the angle at the elbow and wrist as recorded from two electrogoniometers. For the elbow, 180° corresponds with full extension; for the wrist, 60° represents extension. The sliding technique was associated with the largest nerve excursion (mean: 12.4 mm; p < 0.001). Also note that the high peaks in nerve strain in the tensioning technique (when both the elbow and wrist are in extension) do not occur in the sliding technique. In the sliding technique, the increase in strain associated with elbow extension is counterbalanced by the simultaneous reduction in nerve strain at the wrist by moving the wrist from extension to neutral. The amplitude through which joints were moved was identical for the different conditions. From Coppieters & Butler, Man Ther. 2008; 13(3): 213–221, with permission.



(B) Tensioning technique

Fig. 7.19.3 • Example of a sliding and tensioning technique for the median nerve for the hand and wrist. In the sliding technique, the increase in nerve strain associated with finger extension (*top left image*) or wrist extension (*top right image*) is compensated by wrist flexion (*top left image*) or finger flexion (top right image), respectively. In the tensioning technique, the hand and wrist are moved from a position of minimal nerve strain (wrist and finger flexion, *bottom left image*) to a position of maximal nerve strain (wrist and finger extension; *bottom right image*). The sliding technique is suggested as a nonaggressive mobilization technique in the nonsurgical or postoperative management of patients with CTS. Modified with permission from Coppieters et al, J Orthop Sports Phys Ther. 2009;39(3):164–173.

immediate, but not sustained, reduction in temporal summation was observed. This is believed to be primarily mediated by C-fiber input (Price et al. 2002). No group differences were seen for A-delta mediated pain perception (Beneciuk et al. 2009). An immediate and sustained mechanical hypoalgesic effect, measured by the available range of motion during a neurodynamic test, was also observed for participants who received the neurodynamic exercises, but not for the control group. The same research team also conducted a study in patients with CTS. Compared to the sham intervention, a reduction of temporal summation was observed following neurodynamic mobilizations. Although the number of studies is limited and only one type of neurodynamic exercises has been evaluated (tensioning techniques), the authors conclude that neurodynamic exercises may demonstrate favorable neurophysiological effects (Bialosky et al. 2009).

Neighboring structures

The health of structures that surround the nervous system, such as fascia, muscles, joints, tendons and bone, is of crucial importance in patients with peripheral neuropathies. Abnormalities in these surrounding structures may impact negatively on the health of the nervous system and may obstruct or delay recovery. Assessment and possibly treatment of surrounding structures has long been an integral part of movement-based treatment strategies for patients with peripheral neuropathies (Hall & Elvey 1999). Mobilization of the cervical spine at the level of a relevant segmental motion restriction has been shown to be beneficial in patients with neuropathic cervicobrachial pain (Coppieters et al. 2003). Compared to a control intervention, cervical contralateral lateral glides resulted in a reduction in pain intensity and symptom distribution, and improved range of movement.

Evidence of clinical effectiveness

More important than the investigation of the biomechanical and neurophysiological effects is the evaluation of the long-term clinical effectiveness of neurodynamic exercises in patients with neuropathic pain. Unfortunately, there are no adequately powered randomized clinical trials with long-term follow up available yet. Two systematic reviews (Ellis & Hing 2008; Medina McKeon & Yancosek 2008) summarized the findings of a series of smaller studies. Comparisons across studies revealed a possible trend toward improved outcomes for neurodynamic exercises (Medina McKeon & Yancosek 2008). Most studies focused on CTS and it was concluded that trends toward pain and symptom reduction, improved sensation, and improved function and strength, combined with the low monetary and temporal cost, make neurodynamic exercises a reasonable treatment option (Medina McKeon & Yancosek 2008). More research is required to increase the level of evidence.

The bigger picture

This short chapter has focused on the peripheral nervous system. Although phenomena such as the double crush theory remind us that many processes are still not fully understood, we have gained a reasonable insight into nerve pathology in recent decades. We have also gained a good understanding of the mechanical demands that are placed on the nervous system, and how different types of neurodynamic exercises have very different biomechanical effects on the nervous system. Although important, an understanding of anatomy, biomechanics and pathology is only part of the bigger picture. We argue that nerve gliding exercises can be useful, but that these exercises - as is the case with other management strategies - have to be meaningful to the patient and integrated in a much wider approach, including pharmacologic treatment options. By 'meaningful' we mean that the patient must understand the factors that contribute to increased sensitivity of the nervous system. This is likely to require a basic knowledge of neuroscience and pain mechanisms, an understanding of the impact of threats, fear and stress, and an awareness of maladaptive behaviors. Painless compressed nerves remind us that it is not just local tissue pathology that determines an individual's pain state.

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Stretching and fascia

Thomas Myers Christopher Frederick

Introduction

Active or passive soft-tissue stretch is routinely applied in:

- manual therapies (massage, myofascial release, active release, muscle energy)
- rehabilitative physiotherapy protocols (prepostsurgical, post-traumatic)
- performance enhancement (athletic, dance, active isolated stretching)
- self-help methods (yoga, exercise warm-up/ cool-down routines)
- integrative pattern resolution (osteopathy, structural integration).

Much remains unresolved, however, as to the actual mechanics of stretching and what might be the lasting effects of protocols involving variables such as:

- intensity
- amplitude
- duration
- speed,
- direction

• repetition (i.e., pulsed, ballistic or cyclic stretching).

Additional variables can pertain.

In other words: how much, how fast, and how long for each stretch, as well as how often, in which order, and with what intent?

Variations among these factors produce many types of stretching, generally categorized (with overlaps) as: ballistic, dynamic, active, passive, static, isometric, isotonic, and broad categories of neuromuscular facilitation (e.g., PNF, muscle energy). Most studies have concentrated on the effects of stretch on muscular tissue and neuromotor response. Here, we review general concepts, focusing on evidence for the effects of stretching on connective tissue or fascia (using the term broadly) and the extracellular matrix (ECM).

7.2

Definition

Therapeutic stretching takes an area of tissue to the end of its accustomed range of movement, applying either self- or therapist-assisted additional lengthening.

Since tension and compression always coexist at 90 degrees to each other, when any given tissue is tensioned in one direction, cells and matrix are also compressed at right angles to the tension (Fuller 1975). Additionally, a linear stretch will be converted in the complexities of fibrous connections to bending, shear, or torsion in surrounding or 'downline' tissues (Franklyn-Miller et al. 2009).

Additional lengthening extends beyond the accustomed range to meet the definition of 'stretching.' Movements within the subject's usual range of motion (ROM) merit the term 'movement therapy' or conditioning, but not 'stretching.'

To be therapeutic, stretching should stay within the physiological range; overstretching can produce injury (Alter 2004). Indeed, many soft-tissue injuries are seen as the sequelae of local tissues being stretched excessively and too rapidly.

Stretching can be usefully applied to any soft tissue, from the skin, through superficial fasciae, fascia profundis, myofasciae, septa, aponeuroses, tendons, or ligaments, but not cartilage or bone. Thus, fascial stretching, intended or inadvertent, occurs in many of the approaches described in this book, and some fascial stretch will occur in the application of most manual or movement therapies.

Mixed evidence

Despite ubiquitous therapeutic and performancebased stretching, research is still divergent regarding its efficacy (Bovend'Eerdt et al. 2008). Most studies limit themselves to one or two types of stretch techniques, using restricted parameters of intensity and duration (Law et al. 2009). Few have studied the movement-based dynamic stretching that currently prevails in performance training and manual therapy. No consistent guidelines exist for practitioners or movement educators as to optimal measures of intensity, duration, or frequency (da Costa & Vieira 2008).

Contradictory advice therefore informs the how, when, and why of practical myofascial stretching. Evidence-based parameters remain the basis for further research, although some studies cited below point toward guidelines for positive connective tissue stretch responses.

Another factor in the lack of consistency in stretching research is that the organized study of the biomechanical properties of the various topologies and histologies of connective tissues is still in its infancy. Little is definitively known about lengthening and remodeling responses in vivo in anatomically intact tissues, as opposed to single structures isolated for testing in vitro (Standley 2009; Solomonow 2009).

Additionally, the broad emphasis on single-unit muscle stretching needs to be reconsidered in terms of new anatomical models linking muscles, fascia, and ligaments in dynamic series, rather than distinct parallel units that can be treated in isolation (Vleeming 2007; van der Wal 2009; Stecco & Stecco 2009). The evidence is quite clear: the use of the word 'isolated' in conjunction with the word 'stretching' is difficult to justify when a straight leg lift test produces 240% of the strain in the iliotibial tract that it does in the hamstrings (Fig. 7.20.1) (Franklyn-Miller et al. 2009).

Whether prescribed or self-sought, the choice of stretching methods for any given condition, and how to integrate this with other care, will be easier with more evidence concerning local and global tissue effects. lliotibial tract 240% Ipsilateral lumbar fascia 145% Lateral crural compartment 103% Achillies tendon 100% Contralateral lumbar fascia 45% Plantar fascia 26%







Fig. 7.20.1 • Presumptions about stretching, for instance that the force from a stretch is transmitted from insertion to origin, are challenged by research findings: strain transmission from a straight-leg lift test shows up in many other tissues beyond the hamstrings. Part B, C and D from Myers T 2009 Anatomy Trains, 2nd ed. Edinburgh: Churchill Livingstone.

Stretching: the evidence for tissue change

Previous chapters covered the background on connective tissue properties such as creep, stiffness, strain, hysteresis, elasticity, viscosity, plasticity, and thixotropy, as well as force transmission that follows. Fascial stretching is here briefly considered in terms of four potential and inter-related benefits.

- mechanical lengthening (and resulting segmental realignment)
- tissue hydration

- proprioceptive stimulation
- direct stimulation of connective tissue cells, notably fibroblasts.

Mechanical lengthening

All mammals display inflammatory responses to a wide range of trauma or infection from viruses or bacteria, and part of that involuntary response can be neuromyofascial tissue contraction (Grinnel 2009). Humans also respond to stresses at various levels via skeletal and smooth muscle contraction. Longterm problems arise not so much with reflexive or attitudinal hypertonic responses, but rather due to lack of a post-response return to normal states of tone and tissue relaxation, even after the threat, trauma or stress has passed. The subsequent cascade of soft-tissue and skeletal compensations automatically ensues to adapt function to what can become a chronic state of myofascial contraction, increased mvofibroblast activity, and fascial tissue contracture (Schleip et al. 2005; Langevin et al. 2009).

Many clinicians and researchers propose that chronic 'below the voluntary' muscle contraction eventually results in fascial 'thickening' (Langevin et al. 2009) or 'densification' (Stecco & Stecco 2009) or binding among layers that should slide on each other (Fourie 2009) in various parts of the ECM. These patterns result in chronic eccentric as well as concentric tissue loading, which taken together form bodywide 'soft tissue holding patterns' (Myers 2009).

In many cases, practitioners approach a body burdened with neuromyofascial imbalance via a program that seeks to differentiate, decompress, release, and lengthen areas of hypomobility, to give more space and movement. This approach is felt to have the immediate effect of decreasing the stress of tissues under excessive pulling forces or tension and dampening the effects of hypermobility in joints that compensate for hypomobility elsewhere.

The question remains, however, how far various connective tissues can be stretched, how long various topological and histological areas of connective tissue will retain such length when induced, and which cytokines or other chemical factors could be responsible for retention of such benefits. These complex questions are still being framed, let alone answered.

In general, less deformation occurs in connective tissue that is loaded more quickly than the same tissue loaded at a slower rate, suggesting that a slower stretch will be more effective in tissue lengthening than one that is rapidly applied.

The areolar tissue between the skin and the 'unitard' of the fascia profundis shows significant viscoelastic properties, allowing for immediate changes in architecture to accommodate changing forces (Myers 2009). This tissue demonstrates many interesting effects in direct cellular signaling, and is easily accessible for, and accommodative of, lengthening in response to an applied uniaxial stretch (Iatrides et al. 2003; Wang et al. 2009).

Within and around the muscle, the fasciae can be histologically divided into the endomysium around myofibrils, the perimysium around fiber bundles, and the epimysium around the muscle itself. Distribution of each of these varies widely between muscles (Purslow 2002). The dual needs for force transmission through the 'myofascial unit' while accommodating the vascular supply to the muscle cells dictate that both shear and longitudinal strains through the muscle will not be uniform through different phases of contraction and loading. This suggests that most of the 'release' felt in myofasciae during manual therapy is due to muscular relaxation rather than actual lengthening of the fascial elements. To the degree that some mechanical lengthening is available in the myofasciae, research suggests the perimysium may be the most easily adaptable of these three layers (Purslow 2002).

Tendons themselves show little propensity for changing resting length during the application of manual therapy; indeed, they would be poor choices for creating postural and joint stability if they were subject to such deformation. Ligaments vary in their composition, depending on how elastic they need to be, but ligaments with less elastin have been shown to respond elastically to short-term displacement, and with creep to long-term loading, but there is no evidence to show that ligaments will accept a permanent length change with the forces applied in short-term manual therapy (Solomonow 2009).

In dense connective tissue structures such as the iliotibial tract and plantar fascia, it is now evident that the clinician's feeling of 'fascial lengthening' is not coming from actual elongation in the fascial sheet itself, as the forces necessary to lengthen these dense fasciae are far beyond what can be generated therapeutically (Chaudry et al. 2011). A more likely mechanism is that via neurological feedback, muscles in series with the fascia treated are relaxing to produce the feeling of release (Schleip 2003).

A 'map' of the architecture of the ECM requires knowing where each structure 'should' be tied down to surrounding structures, and where it should slide relative to others (Fourie 2009). In addition to mechanical lengthening of tissues, dysfunction (and apparent 'shortness') is surmised to come from adjacent fasciae losing serous lubrication between layers, and the establishment of cross-linkages that will not allow movement between those layers. Thus, stretching can be applied not only to 'length' problems, but also to 'stuck layer' problems. By fixing one layer and requiring stretching movement of the adjacent layer, shear stress is created that allows the restoration of increased relative movement between the adjacent planes of fascia (Schwind 2006).

Anatomical research concludes that muscles attach and function with connective tissue, primarily in series rather than the traditionally assumed parallel arrangement (van der Wal 2009) (Fig. 7.20.2). Since each muscle slip attaches to fascial expansions that then attach to periosteum–ligaments–joint capsules, which ultimately attach to bone, a stretch designed to target a supposedly 'isolated' muscle can be directed laterally, obliquely, or longitudinally to other nearby structures (Franklyn-Miller et al. 2009).

Evidence for sustainable viscoelastic change using therapeutic forces in most dense fascia is unlikely. There may be some sustainable change in areolar or other 'loose' soft tissues, but other evidence is required to sustain the idea of 'fascial lengthening' using manual or stretch therapy.

Tissue hydration

Stretching is thought to increase circulatory flow to dehydrated tissues as well as reduce edema, by squeezing excess fluid from the intercellular space into lymphatic vessels. The value of tissue hydration is best appreciated when considering the dependent interactions of water and protein. As well as being the essential medium of cell metabolism, surface hydration is essential to proteins' structural stability and flexibility (Chen et al. 2008a).

Water around proteins can be divided into three categories, each with different functions: (1) bulk water surrounding the protein molecule, (2) bound water inside the protein, and (3) hydration directly interacting with the protein at the surface. Bulk water moves freely, assisting in protein diffusion. Hydration water forms aqueous networks around the protein surface to keep protein in solution. Individually bound water has multiple contacts that stabilize the protein structure from within (Chen et al. 2008b).



Fig. 7.20.2 • Ligaments, thought to run parallel with overlying muscles (A), can more accurately be viewed as acting in series with nearby muscles (B) (van der Wal 2009). From van der Wal J 2009 The Architecture of the Connective Tissue in the Musculoskeletal System in Fascia Research II: Basic Science and Implications for Conventional and Complementary Health Care, Munich: Elsevier GmbH.

Stretching and fascia

Nuclear magnetic resonance (NMR) imaging has demonstrated that water is extruded from tendons when loaded during stretching (Helmer et al. 2006). Some fraction of tendon hydration water then becomes NMR-visible, upon loading with stretch. This might occur as a result of water unbinding from macromolecules in response to load. This mobilizing and extruding/resorbing process might serve a role in lubricating the tendon during loading, or to increase the stiffness of the tendon in response to loading.

Ligament creep behavior seems related to the initial state of hydration, decreasing with decreased hydration and increasing with higher hydration (Thornton et al. 2001). This knowledge has influenced donor preparation and rehabilitation protocols of ACL and other graft reconstructions (Reinhardt et al. 2010).

In viscoelasticity, the 'elasticity' component generally refers to the collagen and elastin chains in fibrils, while the 'visco' component generally refers to the dynamic interaction of water with the hydrophilic proteins. The viscoelastic response in intramuscular connective tissue originates from gliding between collagen fibrils during stretch (Purslow 2002).

Research of fascia under tension and stretch has described simple modeling of the system as coupled time-dependent molecular gliding within fibrils and between fibrils within a fiber that produces an overall viscoelastic response (Puxkandl et al. 2002).

Klingler et al. (2004) examined the water-binding capabilities of ground substance after stretching porcine fascial tissue. The water content was initially reduced, but after 30 minutes rest the water content surpassed the original and continued to increase up to 3 hours after the stretch, producing an increase in elastic stiffness of the tissue. The authors concluded that fascia seems to adapt hydrodynamically in response to mechanical stimuli, possibly due to a sponge-like mechanical squeezing and refilling effects in the bioarchitecture of hydrophilic glycosaminoglycans and proteoglycans.

To summarize, the colloidal nature of connective tissue means that hydrodynamics is a crucial element in the results of tissue stretching, both in reducing edema and in increasing the water supply to underserved proteins, so increasing the extensibility of the tissue.

Proprioceptive stimulation

Deep fascia holds 'a variety of both free and encapsulated nerve endings, especially Ruffini and Pacini corpuscles, also present, suggesting a proprioceptive capacity of the deep fascia' (Stecco et al. 2006). All the ECM displays a range of proprioceptors which respond to various stretch, pressure, vibration, and shear forces (Schleip 2003).

There are 10 times as many receptor endings in the ECM as within the muscle itself, with endings located in muscle tissue, possibly better described as 'listening' to the fascia within the muscle (van der Wal 2009).

These receptors mediate muscle response via spinal cord and higher center mechanisms.

Treatment methods involving stretching and compression are thought to involve responses from these receptors; for example, in Proprioceptive Neuromuscular Facilitation (PNF) (Moore & Hutton 1980) and Muscle Energy Technique (Chaitow 2006).

Such reflex mechanisms suggest that the intensity often employed during stretching may be excessive (Gowitzke et al. 1988).

Functional re-education of links in appropriately stretched myofascial chains has been shown to counteract habitual dysfunctional patterns (Richardson et al. 2004).

On a global scale, synchronizing breathing with stretching movements has been shown to produce better outcomes for pain reduction, probably through increasing parasympathetic responses (Vagedes et al. 2009).

Direct cellular effects

The effect of stretching on connective tissue cells themselves produces functional changes that in turn affect remodeling changes on the matrix itself. These changes appear to be direct mechanobiologic effects (Langevin et al. 2009).

It has been generally supposed that increased tension on the ECM stimulates fibroblasts to create more collagen, increasing the thickness of the matrix (Oschman 2000). And indeed, cells and their fibrous products do orient and show changes in function and gene expression in response to tensional loads. This is, however, a self-limiting process. Once the matrix is sufficiently dense, the cells no longer 'feel' an applied stretch, and thus reduce the production of new collagen to maintenance level (Bouffard et al. 2008).

Cyclical mechanical stretching of fascia demonstrates morphological changes in gene expression and protein synthesis that affect both the intracellular and extracellular matrices (Chen et al. 2008). It is not clear that therapeutic stretching lasts long enough to initiate these effects, but sufficient repetition of a stretch may produce such an effect (Standley 2009).

When cells are put under linear stretch, over lengthy durations (days and weeks), in response to wounds, postural set, and repeated activities in sport or work, they tend to reproduce. Cells that are compressed from every direction tend to commit suicide (to avoid tumor formation) (Ingber 2003). The duration of therapeutic stretches is too short to produce such effects.

In myofibroblasts, found primarily in large sheets of fascia and aponeuroses, the response to a mechanical stretch is to increase the amount and alignment of the contractile actin molecules within the cell and hook them through the cell membrane integrins to the matrix, exerting a palpable force to pre-stiffen the sheet of matrix in which the cell resides (Gabbiani 2003).

In the most clinically applicable study, Standley found that 90 seconds of simulated 'manual therapy' (pressure and shear on the cell) strongly reduced the effect of 8 hours of 'repetitive strain' through the matrix in which the cell lived (Standley 2009).

More research will refine knowledge of how fascia responds to mechanical forces. This newly discovered 'communicating system,' which rivals the neural system and vascular system in complexity and importance, is 'listening' to the cues provided by inadvertent and deliberate stretching to remodel itself accordingly.

Conclusion

Fascial therapies cannot avoid stretch, and stretching cannot avoid affecting various types of fascial tissues. Connective tissues respond differently to the various forms of stretch, depending on their density and composition, via a combination of mechanical lengthening, tissue anhydration and rehydration, proprioceptive feedback, and cellular responses modulated by both mechanical signaling and cytokine feedback.

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Fascia in yoga therapeutics

Thomas Myers

Yoga is experienced in that mind which has ceased to identify itself with its vacillating waves of perception.

> Yoga Sutras of Patanjali, c. ce 150 1.2 (as translated in Stiles 2002)

Yoga as a fascial therapy

Yoga (yoke, union, the balancing of opposites) is a form of somatopsychic self-training whose origins in the mists of prehistory are meshed with the related field of martial arts (Feuerstein 1998). Yoga was and is a primal exploration into 'Spatial Medicine.' How can function be altered by changing a person's shape (Myers 1998)? Yoga's first practical text, quoted above, was written nearly two thousand years ago.

In its entirety, yoga is a highly complex system for self-realization, whose maps and descriptions of the upper realms of mental, emotional, and spiritual states are full of allegorically rich imagery it shares with Hinduism and the healing system of Aryuveda (Lad 1984). The subtleties and range of yoga's 'eight-fold path,' chakras, and meditative states, however beneficial, are beyond our scope here, where we confine ourselves to the 'limb' of physical training known as *hatha yoga*.

Goals

When hatha yoga is applied as therapy, its goals include increased:

- strength
- balance

- stamina
- flexibility
- and relaxation.

Techniques

In practical terms, hatha yoga is taught in classes or applied more specifically in one-on-one sessions of yoga therapy. Most of the following principles and methods apply to both forms; some elements specific to the one-on-one form are detailed below. The primary tools of yoga therapy include:

7.2

- Pranayama (breathing practices designed to quiet the mind, induce the relaxation response, and improve autonomic physiology)
- Asana (physical postures and movements designed to engage/stretch shortened or bound tissues, strengthen weak muscles, and integrate movement)
- Dhyana (mindfulness).

Both of the first two methods require an element of mindfulness or attention, considered essential to the practice. Mindless repetition of the postures is thought to have less benefit, while 'any movement done mindfully and with attention to the breath is technically yoga' (Davis 2009).

In the service of this mindfulness as well as necessary anatomical precision, yoga classes and therapeutics make use of the following adjunct techniques:

- verbal cues or guided imagery concerning specific areas to move, relax, or give attention
- manual adjustment of the subject by the therapist
- pre- and post-therapy body scans to promote proprioceptive awareness of changes
- between-session exercise or ADL instruction.

Fascia-Oriented Therapies

Even within this limited realm of hatha, the modern expressions of yoga asana practices are bewildering in their variety. Here, we take an agnostic view, with no intention to promote one form of yoga over another. Neither can we do justice to the many 'brands' and variations which are practiced with different intents and intensities in a wide swath of settings from hospitals to gyms, from spas to athletic programs, from village halls to ashrams.

One yogic distinction we will allow to enter our discussion because it bears on yoga's effects on fascia:

- Ashtanga is a vigorous form that requires a higher strength quotient and raises the heart rate and internal body temperature of the practitioner (Swenson 1999).
- Vinyasa ('Flow yoga') uses a slower form of movement transitioning between postures (Kraftsow 1999).
- 'Classic' is a more physically static (but still mentally dynamic), positionally precise holding of postures (sometimes supported by physical 'props'). The Iyengar method of yoga is an influential proponent of this end of the spectrum, as is Bikram yoga (Iyengar 1966; Barnett 2003).
- 'Restorative' is a variation of static yoga designed to induce deep relaxation in various fully supported poses (Lasater 1995).

This list is neither exhaustive nor mutually exclusive: some classes or therapists will combine these approaches within one practice or even one class, and there are many other forms of yoga available. This author has been hoisted aloft in Acro-yoga, stretched in dyads in Partner yoga, twisted his fingers into *Mudra* yoga, and sung the devotional mantras of *Bhakti* yoga, and that only begins to tap the variety of yogic practices.

Yoga and fascia

There are studies indicating beneficial effects of yoga as therapy for certain physiological conditions (Nagendra 1986; Jain et al. 1993; Pilkington et al. 2005).

The effect of controlled breathing practices (pranayama) on fascial tissues and physiology is very hard to measure in isolation from other factors, but research points to where common sense would take us: that increased breath will better oxygenate tissues, and breathing movements will strengthen and coordinate the trunk from the neck to the pelvic floor (Farhi 1996; Iyengar 1996; Sherman et al. 2005; Kirkwood et al. 2005; Androjna et al. 2008). The effects of the practice of asana on the fascial tissues share much with the previous chapter on stretching, so these findings need not be repeated here (see Chapter 20). Subjective feelings and anecdotal reports of calm well-being, balance, and spring-in-the-step abound after yoga or yoga therapy, and these feelings are presumed to result from increased hydration of underserved tissues, expanded range of motion, the ability of previously bound tissues to slide on one another, and heightened proprioception and neural integration as areas return to awareness from the 'sensori-motor amnesia' cycle (Hanna 1988).

More obscure physiologic and spiritual benefits (to the autonomic system, glands, organs, or psychology) are ascribed to particular postures or practices, but these assertions are beyond our ability to prove or even provide useful comment. Provocative research in mechanobiology points to interesting possible global physiological effects of yoga asana (Arora et al. 1999; Langevin et al. 2001; Ingber 2003; Iatridis et al. 2003; Atance et al. 2004). Clearly, different cells have differing optimal mechanical environments, and are sensing and responding to integrin-mediated signaling that arrives via the extracellular fascial matrix.

Yoga asana and myofascial meridians

Many of the asana postures and stretches common to yoga therapy are designed to engage or challenge not just a single muscle, muscle group, or connective tissue structure, but rather to engage an entire kinetic chain or 'myofascial meridian' (Myers 2009). There are upwards of one thousand different asanas and variations in the yogic canon, but the major ones, seen in this way, can be grouped into 'families' designed to bring light and emphasis to different sections or issues within each meridian line (Kraftsow 1999).

In this section, we group common postures and therapeutic moves with the lines they engage. While this process may be considered simplistic, given the complexities within each pose, it does provide some sense of the scope and range of yoga therapeutics.

Forward bends/Superficial Back Line

The Superficial Back Line (SBL, Fig. 7.21.1) is a continuous strap of myofascial tissues spanning from the underside of the toes around the back of the body and across the top of the head to the brow ridge. The relative tension in the parts and whole of the SBL modulates the primary and secondary curves



Fig. 7.21.1 • The family of asanas that stretch the Superficial Back Line (A) includes forward bands such as seated or standing flexion, such as Uttanasana; (B&E) Downward Dog; (C) or Child's Pose; (D). Note that in the Boat (E), reciprocal inhibition is employed between the SBL and the Superficial Front Line (see figure 7.21.2), which must be engaged along with the Deep Front Line (see figure 7.21.4) for this posture. From Myers T 2009 Anatomy Trains, 2nd ed. Edinburgh: Churchill Livingstone.

of the spine, legs, and feet. The SBL is thus a key component of our ability to maintain an easy upright balance by having the major body-weight segments in vertical alignment.

Contracted, the muscles of the SBL create hyperextension through most of the body (though the knee is flexed). Stretched, the muscles and the fascia resist trunk and hip flexion. This creates a family of stretches designed to increase the body's ability to flex, often while maintaining extension at the knee.

Back bends/Superficial Front Line

The Superficial Front Line (SFL, Fig. 7.21.2) is a set of myofascial linkages up the front surface of the body from the top of the toes along the front of the thigh, and from the pubic bone to the mastoid process in the trunk. The SFL is involved in protecting our 'soft underbelly' and other vulnerable bits, and is thus commonly restricted in patterns



Fig. 7.21.2 • Because stress tends to shorten the Superficial Front Line (A) many postures are designed to open this line in sections or as a whole, from a simple Cobra or the standing back stretch that begins 'Namaskarya' (B) or the Bridge (C) through increasingly challenging back bends (D). Supta Virasana (E) looks deceptively simple, but is a challenge to short hip flexors. From Myers T 2009 Anatomy Trains, 2nd ed. Edinburgh: Churchill Livingstone.

of fear or protection, which also tend to dampen the excursion of the front ribs in breathing.

Contracted, the SFL produces flexion in the trunk and hips, but extension in the knee. In stretch, the SFL resists extension and hyperextension, creating a family of stretches designed to open this aspect of whole-body movement.

Lateral Line/Side bends

The Lateral Line (LTL) runs up the side of the body from the outer arch to the ear, running up the trunk in a criss-crossed switchback pattern like the lacing in shoes. The LTL provides lateral stability in the activities of daily life and sport, as well as modulating small twists through the trunk.

Contracted, the LTL creates ipsilateral sidebending and hip abduction. Stretched, it resists contralateral side bending. A smaller family of poses engages or stretches the LTL.

Spiral Line/twists

The Spiral Line (SPL, Fig. 7.21.3) runs around the body, from the side of the head around to the contralateral shoulder and ribs, and back across the linea alba in front via the abdominal obliques to attach to the ipsilateral hip at the ASIS. From the hip, the SPL continues in a loop that passes down the front of the thigh and leg to pass under the tarsum of the foot and pass up the side and back of the leg and thigh to connect up the erector muscles to the back of the head.

Contracted, the SPL contributes to flexion, extension, or side-bending depending on which part is tensing, but overall the SPL creates or maintains twisting or spiraling action in the horizontal plane. Stretched, it resists contralateral rotation. This creates a family of poses built around trunk and hip rotation.

Arm Lines/shoulder and arm stretches

The Arm Lines are continuous fascially and functionally connected strips of myofascia spanning from the axial skeleton to the fingertips along the four 'sides'



Fig. 7.21.3 • Twisting poses engage and stretch the two Spiral Lines (A) reciprocally (B, C). The Pigeon (D) stretches the lower Spiral Line. From Myers T 2009 Anatomy Trains, 2nd ed. Edinburgh: Churchill Livingstone.

of the arm. Due to their arrangement around the bones of the arm, and because the arms are 'designed' to favor mobility over stability, various positions are required to stretch these lines and different elements within the lines. A thorough accounting of yoga therapeutics for all the shoulder muscles is beyond our limited scope here, so a few more general stretches and strengthening poses for the arm are included.

Functional Lines

The Functional Lines connect one humerus with the contralateral femur; in other words, they connect contralateral girdles across the front and the back of the body. The Front and Back Functional Lines (FFL, BFL) are somewhat helical, like the Spiral Line, and are thus involved in all trunk twists as well as many sports actions.

Trunk and hip flexion poses would thus stretch the BFL, and extension poses would stretch the FFL. Trunk twists to the left will stretch the left FFL and right BFL and vice versa. Included here are two poses that particularly challenge the Functional Lines.

Deep Front Line

Interposed between the SFL and SBL fore and aft, between the two Lateral Lines right and left, and further wrapped with the Spiral and Functional Lines, the Deep Front Line (DFL, Fig. 7.21.4) comprises the body's 'core'. The DFL is a continuous track of myofascial tissues that spans from the inner arch up the inseam of the leg, connecting through the groin and ischium to the inner abdominal structures, and up from the posterior adductors through the pelvic floor to the front of the spine, the anterior longitudinal ligament, transversus abdominis, diaphragm, mediastinum and neck to the jaw and bottom of the skull.

Excepting the functions of adduction and breathing, most of the functions of these core muscles are recapitulated in the outer lines. Parts of the DFL help with trunk or hip flexion, other parts with hip extension. The backbends shown in the Superficial Front Line section above would thus also serve for the DFL, especially as the student becomes more adept and stretched out in the SFL. The strong dorsiflexion required in the Downward Dog, shown in the Superficial Back line segment above, will require lengthening of the lower part of the DFL if one is to



Fig. 7.21.4 • Deep Front Line (A) Core engagement is a goal of both yoga and Pilates, but it is difficult in that 'core' muscles are involved in all postures, but not correctly used by many beginning students. Balancing poses (B) and inversions (C) require recruitment of core muscles, as does the Boat pose (D). From Myers T 2009 Anatomy Trains, 2nd ed. Edinburgh: Churchill Livingstone.

perform the pose with the heels on the floor or mat. Thus, many of the poses either engage or stretch (or both) elements of the DFL.

Additionally, the act of balancing requires a coordination of all the lines, but particularly the activation and steadiness in these DFL structures. Therefore, we include some common balancing poses in this section, even though they will require activation of both the DFL and other surrounding lines.

Yoga therapeutics training standards

Caveat emptor: In Western Europe and North America – indeed, worldwide (there are more yoga teachers in California than in all of India (Davis 2009) – the profession of yoga teaching and yoga therapeutics is in its early development, so training standards and professional competency assurance are still in nascent stages, with widely varying skill levels. A 200-hour training is basic to most certified yoga

Fascia in yoga therapeutics

teachers, with moves afoot to extend the standard of training to 500 hours (or more), which is the level of training for most yoga therapists (Seitz 2010).

When seeking or referring to a yoga therapist or teacher, one can check whether the therapist/ teacher belongs to the International Association of Yoga Therapists (IAYT) or Yoga Alliance (YA) or holds some other form of certification (www.iayt. org, www.yogaalliance.org). Some yoga therapists are also trained physiotherapists, occupational therapists, and registered nurses, or have previous training in athletic coaching, Pilates, or personal training.

Although yoga injuries are not uncommon, this author's experience and preliminary research indicate that these are more often caused by zealous students overstretching themselves into injury rather than by heavy handed or misguided therapists.

Referral

Yoga therapeutics can lay claim to nearly two millennia of 'on-the-job training,' though modern clinical trials and replicated evidence-based research are just beginning in this field (see Fig. 7.21.5). Yoga therapy also lays claim to many psychological and organic physiological benefits that may be supported by anecdotal evidence, but are as yet thinly supported by formal studies (Morse et al. 1984; Ornish 2007).

Yoga applied therapeutically as a fascial release technique has much overlap with various techniques in applied manual therapy from physiotherapy and the so-called 'alternative' therapies. Yoga benefits from its combination of stretching, strengthening and balancing, differentiating it from a purely local strengthening approach, as is often undertaken in personal training or physiotherapy, or passive tissue-release techniques such as those often employed in manual therapy.

Yoga as therapy does clearly address coordinated and connected kinetic and fascial chains running throughout the body, as shown by these 'family portraits' of related poses. In the hands of a gifted therapist or teacher, these practices can lead to an open, strong, and balanced body. Some therapists specialize in yoga for older bodies, pre- or post-natal, for children, the differently-abled, or other specialized subject groups. In terms of referral, Restorative yoga postures can be undertaken by the more physically challenged, but most yoga classes and private therapy sessions require a motivated subject who is physically able to assume the postures.







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Pilates and fascia: The art of 'working in'

Marie-José Blom

Introduction

Pilates is regarded by its proponents as a comprehensive method of exercise and total body conditioning, created and pioneered by Joseph H. Pilates (1880–1967). The integrity of the method strongly rests on six basic principles: concentration, control, centering, precision, flowing movement, and breathing (Pilates 1945, 1998).

Pilates's methodology and approach to fitness and health took shape from 1914 to 1918 while he was detained on the Isle of Man as a prisoner of war. While living in the camp, he taught other residents the series of exercises that he had developed for personal use over the preceding decades, both in Germany and England (Redfield 2009). He created makeshift equipment from bed springs and frames to support and supplement his movement repertoire. Pilates immigrated to the United States in 1926 (Pilates 1927).

The blend of Eastern and Western philosophies

The design of the Pilates exercise sequences was strongly based on Yoga and the principals of Zen (Pilates 1945, 1998). Zen, as an established sect of Buddhism, means meditation, 'waking up' to the moment. This mindfulness and awareness is integrated in the Pilates movement with the objective of uniting the body and mind. Pilates's goal was a well-balanced body, with equal importance placed on both internal and external development. He established this

and connections, to affect the motility and circulation of the internal organs within the thorax, as well as in the abdomen and pelvic floor (Calais-Germaine 2005). The visceral organs contained in these cavi-

directly involved the spine.

2005). The visceral organs contained in these cavities are divided by the respiratory diaphragm, which also unites them. The thorax is connected via the pleurae of the lungs and via the pericardium to the diaphragm of pulmonary fascia. The abdominal cavity is connected to the diaphragm by the peritoneum and connects the two cavities. Pilates's movements, choreographed with the breath, are designed to stimulate movement originating from within, with the effect of breathing as described in Yoga principals (Pranayama) to control the mind (Ivengar 1966).

balance by creating movement sequences that focus on rhythmic breath, stimulating both circulation and lung capacity. The movements that followed

The function of integrated breathing aims to

cause elastic movements of both the thoracic and

abdominal cavities, and therefore, via the fascial

7.22

Fusion and integration of various disciplines

Pilates's early connections and collaborations involved the New York dance community, and its leading figures, including Ted Shawn, Ruth St. Denis and George Balanchine. His 'contrology' (explained below) teachings became heavily influenced by dance movement and the vocabulary of both classical and modern dance. This, coupled with his athletic



Fig. 7.22.1 • Joseph Pilates as educator. © 1961 I.C. Rapoport, www.rapo.com.

background as a boxer and gymnast, is reflected in all Pilates exercises (Eismen & Friedman 2004).

For many years this integrated approach existed as a well-preserved 'secret formula' of conditioning, rehabilitation and cross-training. By word of mouth, groups of dancers, actors and athletes would frequent Pilates's studio in pursuit of movement ease in a better-performing body. Originally created as one method or system, different models and approaches have been adapted over time within its teachings. These adaptations serve skillspecific training, as well as therapy and rehabilitation. Examples of these different models include Pilates for the Elderly, Pre- and Post-Natal Pilates, and various sport-skill specific adaptations. Contemporary Pilates is known for its infusion with modifications, therapeutic adaptations, and integration of other approaches such as feldenkrais, yoga, or dance.

Fascia, bound by lifestyle, can Pilates make it move?

The Pilates Method focuses on postural symmetry (alignment), breath control, center/core strength, spine-pelvis, and shoulder stability, well-balanced muscle tone and joint mobility through its complete range of motion (Pilates 1945, 1998). Extensive research in fascia reveals dense and irregular connective tissue continuity through the entire body that envelops and connects every muscle myofibril and internal organ (Schleip 2003; Huijing & Langevin 2009). It functions as a flexible 'soft tissue skeleton,' offering support, form and tensile tone through the entire body. The second nature of this mechanical fascial matrix involves neuro-communication. The ability to adapt, change and influence other systems in the body relates to the presence in fascia of a variety of mechanoreceptors and the presence of smooth muscle cells (myofibroblasts). The integrated system communicates tension and compression, thereby affecting cellular function (Ingber 1998). In that Pilates movement emphasizes control through oppositional length tension, it challenges and stimulates the tensegrity geometry (Fuller 1961) of the entire body, promoting biomechanical health and strength (Myers 2009).

Fascia, through the presence of myofibroblasts, forms an indirect link to the autonomic nervous system, further affecting the quality and tone of the muscular system, which may be influenced by breathing disorders (pH changes), emotional stress, or diet (Myers 2009, Oschman 2003). In contemporary life, movement and range of motion that support a healthy body may be compromised by stressful environments. For example, a 'desk-bound' lifestyle and lack of activity may lead to a fascial-bound, stagnating structure (Beach 2010). Rather than isolating muscle groups, the focus in Pilates rests on whole-body integration as a possible means to restoring fascial length, healthy texture, and optimal tissue hydration and resilience.

Myers (2009) (see also Chapter 7.21) has described how yoga movements can be seen to specifically affect and apply to fascial tracts ('meridians'). Similar parallels can be made with Pilates; for example, in exercises such as the 'Short Spine Massage' or 'Short Spine Stretch,' a traditional Pilates exercise performed through optimal length tension, omnidirectional and full-range articulated control, affecting the different fascial 'lines.'

Pilates achieves this effect by positioning and placement of the body. Unlike yoga, Pilates dynamically takes the body into specific repetitious movement patterns, ideally performed through full range, uniting breath control and precision. The movements are performed in a manner that reinforces full oppositional length tension, emphasizing eccentric control, which affects tensile stimulation through the entire network. Within the Pilates paradigm, this effect is thought to transfer into other areas of life with measurable results of coordination, strength, mobility, posture, grace, and self-confidence (Pilates 1945, 1998; Eismen & Friedman 2004).

Pilates principles and fascia

Concentration

Pilates emphasized the importance of 'being present' and being in the moment during the exercises, while paying attention to every detail of each movement

(Pilates 1945, 1998). Attention to detail is considered to promote an openness to explore and experience, that is, to stimulate the learning process (Doidge 2007). Through focused practice, this process is viewed as contributing to the change and 'remodeling' of the myofascial structures, influenced by the neural impulses delivered to it. As such, using the fascial systems is posited to be a learning and information medium. In this perspective, the fascial and myofascial system, densely populated with feed-back and feedforward mechanoreceptors, expands the opportunities for learning, change and remodeling. Schleip (2003) indicated that the fascial field not only reacts to verbal instruction, but also responds effectively to tactile corrective or directive instruction. Reinforcement by imagery, or 'idio-kinesis,' has also been observed to produce similar physical changes in the motor system (Franklin 1996; Doidge 2007).

This contemporary motor-learning model is intended to provide the environmental conditions



Fig. 7.22.2 • A Short Spine Massage, preparation and posturing – this addresses and loads the deep fascial front line (DFL). B Short Spine Massage, phase 2 – this is an extension of the full superficial backline (SBL). C Short Spine Massage, phase 3 – this phase eccentrically loads the lateral line (LL) and both functional lines (BFL and FFL). D Short Spine Massage, phase 4 – this is the resting and control phase. ©1998 Marie-Jose Blom, www.pilatesinspiration.com





Fig. 7.22.3 • Optimizing proprioception through positional correction with touch or props. ©2007 Robert Reiff, www.magiclight.com

for training of the feed-forward, or core, system and to enhance timing and motor skill coordination, which in turn stimulates neuro-plasticity (Doidge 2007). Over time, this process is thought to lead to new motor patterns that override established compensatory movements that may cause biomechanical strain. Concentration and embodiment of the movement in Pilates is considered to enhance coordination of the neuromuscular system with the myofascial system (Oschman 2003; Myers 2009). The emphasis on concentration on the entire body, through each movement, as opposed to compartmentalization, parallels the contemporary science-based anatomy models of a 'myofascial' or 'neural-myofascial, skeletal' continuum, organized and stimulated by directed movement patterns (van der Wal 2009; Myers 2009).

Movement patterns that are sequenced to follow the organization of the densely innervated fascial system are considered both to render deep structural support and to provide position-sense of the body and its movement in space. Pilates proponents posit that this result forms an integrated memory bank and communication system for retraining healthy movement. That is, optimal movement leads to a healthy structure, i.e., 'form follows function' (Wolff 1986). For example, in the Pilates footwork on the universal reformer, the correct placement of the feet will transfer the movement along the entire kinetic chain via fascial pathways.

When optimal foot positioning is not achieved in these movements, proprioceptive communication and retraining may be compromised (Myers 2009). Positional correction with touch or props (towel) has been observed to optimize proprioception and improve concentration through embodiment.

Control or contrology

Contrology is a term coined by Joseph Pilates to indicate a form of mastery and precision in the smaller, deeper aspect of every movement. This idea introduces the working of 'inner movement' or micro-movement. Attention to such detail is believed to stimulate activity on a deeper level where true sensing and connectivity are often lost (Richardson et al. 2004). In this approach, 'working in' rather than working out forms the foundation of deep postural support, true strength, and graceful movement.

Precision

Within the Pilates model, striving for the best possible performance renders every movement significant. As each movement is performed with precision, close attention is paid to the physical form, which is organized by a mental blueprint of the movement. First-hand observations by the author, and others, indicate that precision might be reinforced by movement imagery. It has been proposed that the union of mental and physical effort stimulates and recovers new neural pathways, a result of neuroplasticity (Doidge 2007).

Flowing movement

The heightened awareness of movement through the skill of precision is believed to lead to a movement flow that improves the efficiency of everyday movement. Promoting movement flow, coordination and grace is viewed as resulting in healthier tissues and a more vital body, well organized from within (Ingber 1998).

Centering

Centering in Pilates work has become known as Core Control, or movement control (Pilates 1945, 1998; Eismen & Friedman 2004; Richardson et al. 2004). Centering involves focus on the development of deep postural support, more specifically the deep musculofascial system. Fascial tissue structurally and mechanically differs from muscular tissue in that it is stiffer with less give, and therefore reacts through the pull of the attached muscles, contributing to stability of the lumbar spine (Richardson et al. 2004). The Pilates concept of centering may contribute to the pre-stress theory of tensegrity, where the pull of tension is distributed through all structures surrounding the spine and beyond (Ingber 1998; Myers 2009). This effect is believed to create a more resilient stability model, forming a flexible corset of support which contributes more to dynamic stability.

A well-designed corset of support

The material of a good support system will often reflect a combination of resilience, a multidirectional weave, and reinforced strength through multiple layers. The lumbar fascia does fit this design profile. The fascial part of the corset consists of three layers: the posterior lumbar fascia (PLF), the medial lumbar fascia (MLF), and the anterior lumbar fascia (ALF) (Vleeming 2007). The PLF extends from the midline to the thoracic and lumbar spinous processes and their ligaments, and attaches to the posterior ilium, crossing the midline to the opposite ilium. The MLF firmly invests the lumbar transverse processes, its inter-transverse ligaments, as well as the inferior iliac crest and the ilio-lumbar ligament. The transversus abdominis (TA) is continuous with this fascia, and with the abdominal fascia in the front. There, the PLF and MLF form an envelope that houses the multifidus. The ALF arises from the anterior transverse processes and forms a small envelope with the MLF; this envelope houses the quadratus lumborum (QL) before merging with the TA. The QL is concentrically active with the diaphragm upon inhalation and thereby contributes to stability through tensing its own fascial envelope.

This multilayered deep corset functions as a stability amplifier, with feed-forward action that can be learned or re-learned with centered 'focus.' Relearning to connect the corset layers is part of the neural communication skill of the deeper muscles, which is considered to lead to Contrology, Movement Control and Core Control, three definitions supporting the same meaning. Within the Pilates paradigm, success in optimizing the full function of the core corset lies in the connection and functional relation of the respiratory system (the diaphragm), the multifidus, and TA, in close synergy with the pelvic floor. Pilates integrates a specific breathing pattern into the movement repertoire and practice to reinforce this synergy. Breathing and the concept of Centering form the underpinnings of Core Control.

The breath in Pilates

Breathing pattern disorders, such as hyperventilation, may lead to respiratory alkalosis, and subsequent smooth muscle constriction, which, because smooth muscle cells are embedded throughout the fascial network, might lead to increased fascial tone (Chaitow 2002). To enhance thoroughly balanced breathing, as part of every Pilates exercise, Pilates taught that deliberate exhalation ensures a full inhalation. The movement combined with specific integration of the breath is viewed as promoting circulation. This concept has been reinforced by accounts of respiratory physiology (Chaitow 2002). It has been proposed that specific and directed integration of breathing with movement can encourage enhanced circulation and elimination of metabolic by-products, while promoting connective tissue hydration.

Instruction on better breathing is a key ingredient to fascial health and health in general. Such instruction is considered to be an important therapeutic tool in Pilates. In support of this position, extensive dissections, specifically directed to the fascial systems, have revealed the significance of the respiratory system via the fascia of the deep front fascial line (Myers 2009). Movement of the breath is visible in the spine and sacrum as a breathing wave, which might also reveal movement stagnation or abnormal movement patterns (Chaitow 2002).

Well connected

Starting with the diaphragm, the fascial connections functionally relate the lungs via the parietal pleura, as well as the heart via the pericardium, with the central tendon of the diaphragm. Further connections of the parietal pleura fascia also associate with the inner aspect of the scalene muscles (Myers 2009). Like the iliopsoas, the scalene tends to respond by tightness and hypertonicity due to postural and/or emotional stress (the fight-or-flight response). Through their reflex contractility, the scalenes might inhibit the elasticity of the lung pleura, compromising the coordination between the diaphragm, TA, and the pelvic floor, thereby affecting lumbo-pelvic stability through inefficient inter-abdominal pressure (Richardson et al. 2004).

The crux of the diaphragm below merges with the fascia of the iliopsoas, thereby connecting the diaphragm to the lower quarter, and influencing the movement of the hips (walking), while also blending with the anterior longitudinal ligament of the spine. The descending psoas then connects its inferomedial fascia with the pelvic floor fascia, linking with the conjoint tendon, and internal obliques (Gibbons et al. 2002). This fascial connection links the diaphragm mechanically and functionally to the TA and the pelvic floor.

The attention to detail and supported alignment, facilitated by Pilates exercises, is viewed as creating the space and length tension of these fascial structures that elongates and decompresses through movement of the breath.

From foot to core

The posterior pelvic floor fascial connections form a link, via the continuing intramuscular septum of adductor magnus and posterior tibialis, into the foot. Here, the fascia aids in load distribution, transmission of forces, and elastic rebound, influencing the postural behavior of the entire body. These fascial connections are materialized and emphasized through specific attention to detail in foot placement during Pilates footwork.

Alignment support from within

Unlearning poor habits is often more challenging than learning new habits. This problem is particularly true for posture and movement, which require an internal physical sensing or feel for the movement, as well as a feel for positioning. The Pilates perspective proposes that with more refinement a person may also learn to sense the mechanics of the movement. This ability of sensory awareness, or proprioception, is facilitated throughout the body by the highly proprioceptive fascia, among others (Schleip 2003). What is considered important is that feeling is introduced first (i.e., embodiment). Direct observations indicate that such feeling is greatly facilitated by assisting the body in a supported neutral alignment, which appears to be an essential condition for proprioceptive learning. For example, using a pillow brings the upper body slightly forward, so that the upper and mid back are 'opened' to facilitate better breathing, and even the smallest space in the lumbar area and the space between the lumbar spine and the floor (when in supine position) are filled. The author's long-term observations confirm that this passive support can release an otherwise habitual holding pattern.

'As within, so without': movement perceived from the inside reflects what happens on the outside

Within the Pilates perspective, movement and posture on the outside often reflect a person's internal state. This correspondence relates to the tensional balance of length tension, made continuous via the fascial system, an omnidirectional fascial system from deep to superficial. Motivating oppositional length tension made visible via the fascial system can provide continuous and dynamic stability in all directions, with optimal mobility and flexibility, very much like a tensegrity system (Fuller 1961). The application of the theory of tensegrity to human anatomy was introduced by Levine (2002), who coined the word bio-tensegrity, relating the tensegrity concepts to structural integrity in nature, as well as in human movement. Many Pilates movements, with or without equipment support, are performed with the concept of creating space 'within' or imaged as moving space on the inside.

Observations indicate that this inner space can be rearranged within the same movement pattern by changing the breathing pattern, and cuing oppositional length tension during the movement. To introduce diagonal 'connectivity' a light two-point diagonal touch during the movement appears to self-correct and improve control.

This Pilates approach to movement is envisioned as a whole body event with integrated wellness, addressing:

- Circulation via activity of the respiratory system (breath) and muscular system (movement); the stimulation of the cardio pulmonary system via movement and breath
- Flexibility of the arterial walls and suppleness of the connective tissues
- Visceral stimulation all organs are interconnected and invested in fascia, which through movement produces a continuous 'visceral massage'



Fig. 7.22.4 • This image suggests the internal blueprint of movement, space and support. ©1992 Lois Greenfield, www. loisgreenfield.com

- Lymphatic stimulations via pressure alternation between the abdominal and thoracic cavities stimulated by the movement of the diaphragm (Chaitow 2002)
- Tissue hydration fluidity through movement can lead to movement fluidity by imbibing interstitial fluids and therefore facilitating fluid exchange
- Force transmission the central role of the myofascial system in sharing and transferring load throughout the muscular and other systems (Mass & Sandercock 2008).

Specialized equipment: reformer or transformer

A focal point in the Pilates technique is the designed and technically evolved Pilates equipment, especially the Universal Reformer. The Universal Reformer is a long frame that accommodates a platform that runs in a track. The padded platform is connected by springs to the frame and offers variable resistance. The choice of spring resistance is considered to be particularly appropriate, as muscles behave similarly to springs. Muscles control and resist deformation resulting from internal and external joint loading, returning to their resting position following lengthening (Richardson et al. 2004). A coiled spring takes time to lengthen and return to its initial position after loading, not unlike the spring-like quality of the muscle. The springequipped reformer thus functions as an external coach, guiding internal graduated control. A pulley system is designed to allow for variation, fine-tuning, and integration of proprioceptive challenges using the upper or lower extremities. The equipment is designed to allow the body to be supported in the most productive postural position while introducing movement.

Depending on the movement and the individual, the equipment is intended to function as a tool that can provide more challenge or support for optimal movement learning. The use of variable spring resistance allows for uniform controlled loading, in alignment, through full range of motion of the joint. Direct observations indicate that this resistance offers a similar effect to myofascial tissue release by taking advantage of low load and long duration stress to the fascial tissue via deceleration.

Reformer versus machine

The Pilates reformer allows the body to move up through the intended functional sequence, addressing the 3-D fascial web. As such, Pilates can be used as an exercise modality in the therapeutic environment to mobilize, restore, and stabilize and can be used to remodel fascia to its optimal tension-length. On a practical level, the potentiality of the reformer rests in the skills of the teacher, who conveys the process of an exercise (the 'how') rather than the event of an exercise (the 'what'). In the author's experience, teaching the 'how' renders the machine a 'Reformer' (transforming the body), while teaching only the 'what' relegates it to remaining a machine creating a robot.

Through continued scientific developments, fresh understandings of functional anatomy removed from compartmentalization, in favor of a whole body approach to movement and wellness, have finally emerged in Pilates's therapeutic environments (van der Wal 2009).
Summary

Certain cautions and contraindications exist to traditional Pilates. Special care and caution should be taken in cases of:

- Osteoporosis Avoid movements of flexion and flexion/rotation combinations.
- Spinal stenosis Avoid hyperextension and external rotation combination.
- Spondylosis Avoid hyperextension and external rotation combination.
- Idiopathic scoliosis This requires a specialized approach of curve management, with attention to

decompression, balance and optimal length tension. The modifications needed differ with each individual case and require a well-trained clinical eye.

Any of these potential contraindications may be minimized by positional and postural support, with or without the use of the specialized Pilates equipment, and the changes brought forth by the movement restoring myofascial continuity. These options are best achieved in a therapeutic and clinical environment. Further research regarding the benefits and effects of Pilates in a true clinical environment is warranted.

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Nutrition model to reduce inflammation in musculoskeletal and joint diseases

7.23

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Musculoskeletal diseases are more prevalent than all chronic disease types, a major cause of pain and reduced quality of life. The Bone and Joint Decade (2002–2011), an international collaborative, is working to improve the quality of life for people with musculoskeletal conditions (American Academy of Orthopaedic Surgeons 2008). Arthritis is associated with varying degrees of joint inflammation and accompanied by destruction of connective tissue. Evidence suggests that the inflammatory and destructive components of cartilage are distinct disease processes, and that joint destruction may continue even when inflammation is suppressed (van den Berg 1998). Conventional treatment for inflammation includes long-term use of narcotic analgesics, nonsteroidal anti-inflammatory drugs (NSAIDs), and opioid drugs which have wellestablished adverse effects and safety risks. Nontoxic therapies and mechanism-based approaches are needed for the management of inflammatory diseases since cyclooxygenase-2 (COX-2) inhibitors were withdrawn from the market due to adverse cardiovascular effects (Yoon & Baek 2005). Nutrition offers a nontoxic long-term approach to chronic disease management, potential for reducing pain and inflammation, and supports optimal musculoskeletal function. Calorie control is critical in the treatment of musculoskeletal diseases, as excess weight increases stress on joints and exacerbates pain.

Inflammatory response

The inflammatory response includes activation of white blood cells, release of immune system chemicals, release of inflammatory mediators and prostaglandins.

Acute inflammation is the initial response of the body to harmful stimuli mediated by interleukins. Interleukin 1 (IL-1) and interleukin 6 (IL-6) activate neutrophils that recruit macrophages to injured tissue. Neutrophils also release cytotoxic and cytolytic molecules that cause destruction through lysis of muscle cells, fascia and surrounding tissue. IL-6 contributes to painful and persistent joint damage and chronic inflammation in rheumatoid arthritis. Chronic inflammation involves overproduction of IL-1. IL-6 and tumor necrosis factor (TNF), leading to tissue damage; C-reactive protein (CRP) releases IL-1, IL-6 and TNF, which are responsible for cartilage degeneration in arthritic joints. A persistently elevated CRP level is found in patients with rheumatoid arthritis. who are at risk for continuing joint deterioration (Otterness 1994).

Fatty acids: anti-inflammatory properties

Eicosanoids exert control over inflammation and include prostaglandins, prostacyclins, thromboxanes, and leukotrienes derived from the essential fatty acids (EFAs). EFAs must be supplied through dietary intake and cannot be synthesized by the body. Polyunsaturated fatty acids (PUFAs) include short chain alpha-linolenic acid (ALA) and long chain molecules, eicosapentaenoic acid, (EPA) docosapentaeonoic acid (DPA) and docosahexaenoic acid (DHA). Short chain omega-3 fatty acids oppose inflammation through decreased production of inflammatory prostaglandins, leukotrienes, and arachidonic acid. Food sources providing short chain omega-3 fatty acids include flaxseed, walnuts, canola and rapeseed oils. Long chain omega-3 fatty acids oppose inflammation by competing with arachidonic acid for conversion to pro-inflammatory cytokines IL-1 and TNF and additionally compete with COX and LOX enzymes that are up-regulated in the inflammatory process (James et al. 2000; Ringborn et al. 2001). PUFAs, especially total omega-3 fatty acids, are independently associated with lower levels of pro-inflammatory markers (IL-6, IL-1ra, TNF-a, CRP) and higher levels of anti-inflammatory markers (soluble IL-6r. IL-10, TGF-β) independent of confounders, supporting the opinion that omega-3 fatty acids are beneficial in treating diseases characterized by active inflammation (Ferrucci et al. 2006). Long chain omega-3 fatty acids are important constituents of an anti-inflammatory diet and include oily fish from cold northern waters, such as salmon and mackerel, sardines, herring, black cod (sablefish or butterfish), fish oil, algae, and DHA-rich eggs.

The pro-inflammatory eicosanoids prostaglandin E2, thromboxanes and leukotriene B4 derive from the omega-6 fatty acid arachidonic acid, which is maintained at elevated cellular concentrations by the high omega-6 and low omega-3 PUFA content of the modern Western diet (James et al. 2000). Arachidonic acid is a polyunsaturated omega-6 fatty acid found in phospholipids and a precursor for eicosanoid production. Arachidonic acid is obtained from meat, poultry, fish and eggs, or synthesized from linoleic acid. Linoleic acid is an omega-6 fatty acid found in vegetable oils such as corn, soy, safflower, sunflower, cottonseed, sesame and grape seed, some nuts and seeds. Excess consumption of saturated fatty acids such as palmitic and stearic increases inflammatory signaling through activation of macrophages, neutrophils and bone marrow-derived dendritic cells, leading to inflammation, impaired insulin signaling, and insulin resistance in white adipose tissue and muscle (Kennedy et al. 2009).

The ratio of omega-6 fatty acids to omega-3 fatty acids should be low (i.e., 3:1 or 5:1); however, American intake ratios range from 10:1 to 17:1 for omega-6 fatty acids to omega-3 fatty acids (Kris-Etherton et al. 2000).

Trans-fatty acids are unnatural fat species formed after hydrogenation from naturally occurring *cis*fatty acids found in margarines and shortenings. Dietary intake of trans elaidic fatty acid supports inflammation through hormonal imbalances that promote defective cell membranes and cancer. Research from the Nurses' Health Study demonstrated that TFA intake was positively associated with IL-6 and CRP in women with a higher body mass index (Mozaffarian et al. 2004).

Monounsaturated fatty acids, of which oleic acid is the most common, are found in canola, olive and peanut oils, some nuts, seeds, and avocadoes. Reduced concentrations of inflammatory markers are reported for individuals who adhere to the traditional Mediterranean diet that is abundant in olive oil (Chrysohoou et al. 2004). Oleocanthal, a compound found in olive oil, prevents the production of pro-inflammatory COX-1 and COX-2 enzymes similarly to the mechanism of action for NSAIDs, decreasing inflammation and pain sensitivity. The highest oleocanthal levels are found in stronger-flavored oils from Tuscany and regions using the same olive varietal. The consumption of 50 mL (3.5 Tbsp) of olive oil is equivalent to 200 mg ibuprofen. Weight management is required if olive oil is used as a nutritional intervention, to enhance anti-inflammatory properties, as 50 mL olive oil (\approx 400 kcal) yields a high caloric density (Beauchamp et al. 2005).

Fatty acid dietary supplements: anti-inflammatory properties

Ingestion of fish oil supplements effects reproducible alterations in eicosanoid metabolism that ameliorate inflammation, decrease production of IL- β in patients with rheumatoid arthritis, and alter the fatty acid constituents of cell membranes (Kremer 2000; James et al. 2000). Concentrated omega-3 fatty acids found in fish oil supplements offer benefits associated with fish consumption without exposure to harmful environmental toxins such as mercury, polychlorinated biphenyls (PCB) and organochlorine (OC) that can accumulate in fish. Analysis of five commercial over-the-counter brands of fish oil supplements available in the United States demonstrates that none of the brands contains detectable amounts of PCBs, OCs, nor significant amounts of mercury (Melanson et al. 2005). Additional product information required from companies who manufacture fish oil supplements includes those who employ sustainable fishing practices, and molecular distillation to minimize mercury and other toxins, content labeling to identify omega-3, potential for contamination, and product storage (Cannon 2009).

Schizochytrium microalgae is a DHA-rich fish oil alternative containing small amounts of EPA and almost no arachidonic acid. DHA-rich oils are located along coastal areas as part of the shellfish food chain, unrelated to toxic algae, demonstrate no allergic reactions in humans, and are free of contaminants such as PCB and mercury (Cannon 2009). Neptune Krill Oil (NKO) is a phospholipid carrier of omega-3 fatty acids EPA, DHA, as well as antioxidants, astaxanthin and a flavonoid, that may offer an alternative regimen for management of chronic inflammatory conditions. NKO is extracted from Antarctic krill (*Ephausia superba*), a zooplankton at the bottom of the food chain. Ingestion of NKO at a daily dose of 300 mg significantly inhibits inflammation and reduces arthritic symptoms within 7 to 14 days (Deutsch 2007).

Evidence exists that avocado/soybean unsaponifiables (ASU) contain sterols that are anti-inflammatory and provide protection against cartilage degeneration. The biologically active compounds found in avocado and soybean oils are classified as unsaponifiable lipids and include phytosterols beta-sitosterol, campesterol and stigmasterol (Lippiello et al. 2008). ASU may help patients reduce their consumption of NSAIDs, as shown by long-term symptomatic relief observed in patients with osteoarthritis of the hip (Soeken 2004). Several clinical trials have confirmed that gamma-linolenic acid (GLA), an omega-6 fatty acid found in borage seed oil, evening primrose oil and blackcurrant oil, reduces inflammation, tender joint scores, morning stiffness and requirement for NSAIDs (Kapoor & Huang 2006).

Patients should consult with their healthcare provider because fish oil supplements taken at certain dosages can cause inhibition of platelet aggregation and require monitoring for patients on anticoagulant drugs or aspirins, or impending surgery (Sanders & Sanders-Gendreau 2007; Cannon 2009).

Culinary spices and herbs: anti-inflammatory properties

The health benefits associated with herbs and spices require identification of specific bioactive substances that oppose inflammation. Unlike their pharmaceutical counterparts, many plants modulate inflammation via a multitude of pathways rather than inhibition of a single enzyme along the inflammatory cascade. Antiinflammatory actions are reported for spices and herbs, such as ginger, turmeric, saffron, curcumin, bromelain, German chamomile, licorice and capsaicin. Ginger and turmeric have been used to treat arthritis since ancient times (Craig 1999; Wargovich et al. 2001; Low Dog 2006; Tapsell et al. 2006; Aggarwal et al. 2009). Patients should consult with healthcare providers regarding herb ingestion, as large doses can be toxic, promote adverse medical consequences, or interfere with medications. See Table 7.23.1.

Fruits and vegetables: anti-inflammatory properties

Dietary intake abundant in plant-based foods decreases the risk of chronic diseases, due to phytochemicals (e.g., carotenoids and flavonoids) which promote antioxidant and anti-inflammatory properties. Several mechanisms have been proposed to explain the in-vivo anti-inflammatory action of flavonoids. One mechanism involves inhibition of eicosanoid-generating enzymes such as phospholipase A₂, COX and LOX, which reduces concentrations of prostaglandins and leukotrienes. Some flavonoids, especially flavone derivatives, express their anti-inflammatory activity at least in part by modulation of pro-inflammatory gene expression for COX-2, inducible nitric oxide synthase, and several pivotal cytokines (Havsteen 2002; Kim et al. 2004). Flavonoids are responsible for the deep color of fruits and vegetables, which is concentrated in skins and peels. Pomegranate extract (POMx) inhibits inflammation from activated human mast cells involved with connective tissue destruction and proteolytic activity associated with cartilage destruction, providing potential benefit for treating inflammatory diseases in which mast cells play an active role (Zafar et al. 2009). Crude extract of blueberries (Vaccinium corymalosum) is rich in phenolic acids, flavonoids, and anthocyanins displaying antinociceptive and anti-inflammatory activity which may be helpful in the treatment of inflammatory disorders (Torri et al. 2007).

Dietary antioxidants, such as the carotenoids beta-cryptoxanthin and zeaxanthin, in addition to vitamin C, might protect against the development of inflammatory polyarthritis. The action of carotenoids may vary, whereby the influence of some markers of inflammatory activity may be greater than those of other carotenoids. A modest increase

Culinary herbs/spices and botanical name	Anti-inflammatory components	Anti-inflammatory properties
Red pepper: chili, cayenne pepper, pimiento, cherry pepper (<i>Capsicum frutescens</i>)	Capsaicin	Potent inhibitor of substance P, neuropeptide associated with inflammatory processes and pain transmission
Ginger (Zingiber officinale)	Gingerol Paradol Zingerone	Inhibits COX 1, COX 2, 5-LOX, TNF, interleukin-1 β ; suppresses prostaglandin and leukotriene biosynthesis
Turmeric (Curcuma longa)	Curcumin	Inhibits TNF and COX -2
Rosemary (Rosmarinus officinalis)	Carnosol Rosmarinic acid	Decreases inflammatory cytokines, chemokines
Clove (Syzygium aromaticum)	Carvacrol Thymol Eugenol Cinnamaldehye	Inhibits COX 1,COX-2, 5-LOX, TNF and interleukin-1 β
Nutmeg (<i>Myristica fragrans</i>)	Myristicin Eugenol	Inhibits TNF- α and prostaglandin production
Cinnamon (Cinnamomum zeylanicum)	Eugenol Humulene Cinnamaldehyde	Inhibits COX- 1, COX- 2 5-LOX, TNF and interleukin-1 β

Table 7.23.1 Culinary herbs and spices: anti-inflammatory properties

in β -cryptoxanthin (carotenoid) intake equivalent to one glass of freshly squeezed orange juice per day is associated with a reduced risk of developing inflammatory disorders such as rheumatoid arthritis (Pattison et al. 2005). Bromelain, an aqueous extract

obtained from both the stem and fruit of the pineapple plant, contains a number of proteolytic enzymes associated with anti-inflammatory and analgesic properties demonstrated in clinical osteoarthritis trials (Brien et al. 2004). See Table 7.23.2.

Table 7.23.2 Fruits and vegetables: anti-inflammatory properties			
Food/botanical name	Phytochemicals	Anti-inflammatory properties	
Garlic (Allium sativum)	Ajoene and allicin	Inhibits TNF and inflammatory interleukins	
Onions(<i>Allium fistulosum</i>), apples, broccoli, berries, parsley, grapes	Flavones Quercetin	Inhibits COX and 5-LOX pathways; reduces release of arachidonic acid	
Citrus fruit and peel	Flavanones	Inhibits eicosanoid biosynthesis	
Red tomatoes, red grapefruit, watermelon and other red fruit and vegetables	Carotenoids: Lycopene	Limits inflammatory damages	
Broccoli, Brussels sprouts, cabbage and cauliflower	Indoles Isothiocyanates	Enhances down-regulation of inducible nitric oxide synthase, COX-2 and TNF- α expression	
Berries, cherries, red grapes, pomegranate, eggplant	Anthocyanins	Inhibits eicosanoid biosynthesis	

Beverages: anti-inflammatory properties

Tea and red wine contain catechin, a polyphenolic antioxidant plant metabolite, that quenches free radicals, protects against oxidative cell damage and demonstrates other health benefits in vitro and in vivo. Green tea (Camellia sinensis) polyphenols such as epicatechin, epigallocatechin, epicatechin-3-gallate, and epigallocatechin-3-gallate are potent antioxidants. Epigallocatechin-3-gallate is the most abundant of the polyphenolic compounds, accounts for 30–40% of the dry weight of green tea and possesses the greatest antioxidant activity (Sutherland et al. 2006). Some green tea catechins are chondroprotective, and consumption of green tea may be prophylactic for arthritis and benefit the arthritis patient by reducing inflammation and slowing cartilage breakdown (Adcocks et al. 2002). Resveratrol, a polyphenolic found in skins of red fruits, inhibits COX-transcription and offers a treatment modality for inflammatory diseases (Szewczuk et al. 2004). Cocoa-based products contain catechins and flavanols that modify the production of pro-inflammatory cytokines, synthesis of eicosanoids, activation of platelets, and nitric oxide-mediated mechanisms (Selmi et al. 2008).

Anti-inflammatory diet

An anti-inflammatory diet provides a natural and nonpharmalogical approach for decreasing inflammation associated with musculoskeletal diseases, with no known adverse effects (Sanders & Sanders-Gendreau 2007). The holistic and natural approach afforded by wholefood becomes less natural and less safe when specific nutrients are isolated, packaged and sold as a single anti-inflammatory product. Antiinflammatory dietary guidelines primarily emphasize plant-based foods, cold water fish, reduction in saturated fatty acids and trans-fatty acids, increase in omega-3 polyunsaturated fatty acids, optimal omega-3:omega-6 fatty acid ratios, anti-inflammatory culinary herbs and spices, and catechin or polyphenolic abundant beverages. Specific recommendations for omega-3:omega-6 fatty acid ratios can be problematic as an identical ratio can be achieved with different amounts of each fatty acid class (The American Dietetic Association 2007).

Nutritional education and counseling enhances competency related to food purchasing, storage, preparation and enrichment, modifications for various food cultures, dietary restrictions, and allergic reactions (e.g., fish). Dietary fats and oils must be properly stored to prevent lipid peroxidation (rancidity) or chemical decomposition, which can promote inflammation, premature aging, and degenerative changes in cells and tissues. Heat and light accelerate oxidation of fats and oils; however, the rate of rancidification can be decreased by refrigeration, or storage in a cool, dark place with little exposure to oxvgen or free radicals. Although the omega-3 fatty acid content of farm-raised and wild salmon is nearly equivalent, the higher overall fat content of farm raised salmon increases exposure to a higher level of PCB contamination. To reduce PCB exposure, trimming the fat on fish, and dry cooking methods, enable the PCB content in fat to dissipate (Klein 2005). Enrichment of soybean oil with stearidonic acid (SDA), a land-based omega-3 fatty acid, is a sustainable approach that increases the omega-3 index by raising erythrocyte EPA concentrations (Lemke et al. 2010).

A fat-restricted diet may provide insufficient omega-3 fatty acids for vegetarians who do not consume longer chain DHA and EPA omega-3 fatty acids from animal sources such as fish. Vegetarians require adequate shorter chain omega-3 alphalinolenic acid (ALA), which can be elongated into longer chain fatty acids such as DHA and EPA. ALA sources include walnuts, flax seed, hemp, chia seeds, dark greens, and tofu. Dietary recommendations for vegetarians include 3–5 grams of ALA, in a 2000 calorie diet, and omega-3 fatty acid supplementation from DHA/EPA sources; DHA-rich eggs provide 60–150 mg DHA per egg and microalgae supplements (Panebiano 2007; Cannon 2009).

The Anti-Inflammatory Nutrition Model (Fig. 7.23.1) demonstrates the potential to replicate anti-inflammatory properties in clinical practice. General recommendations for increasing specific food groups such as fruits and vegetables may not assure a clinical response, due to molecular and pharmacological variances of anti-inflammatory components. Fruit and plant extracts are a complex mixture of various constituents; however, it is not clear whether a single compound or mixture of compounds is responsible for the anti-inflammatory response (Schafer et al. 2006). Ellagic acid and quercetin, which are both found in pomegranate, exert a more pronounced effect against cancer than either compound alone (Seeram et al. 2005).



Fig. 7.23.1 • Anti-inflammatory nutrition model.

A 'preventative' catechin diet is difficult to quantify due to discrepancies between experimental and clinical studies, human factors such as self-reports of catechin intake, and variations in food product descriptions. The amount of green tea consumption required to achieve an anti-inflammatory response is difficult to quantify due to inconsistent catechin values in various brands, and differences in reported product purity (Sutherland et al. 2006). The actual catechin content from commercially available products ranges from 9% to 48% of label claims (Manning & Roberts 2003).

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Fascial fitness

Suggestions for a fascia-oriented training approach in sports and movement therapies

Divo G Müller Robert Schleip

Introduction

Whenever a football player is not able to take the field because of a recurrent calf spasm, a tennis star gives up early on a match due to knee problems, or a sprinter limps across the finish line with a torn Achilles tendon, the problem is most often neither in the musculature nor the skeleton. Instead, it is the structure of the connective tissue - ligaments, tendons, joint capsules, etc. - which has been loaded beyond its capacity (Renström & Johnson 1985; Hyman & Rodeo 2000; Mackey et al. 2008; Counsel & Breidahl 2010). A focused training of the fascial network could be of great importance for athletes, dancers and other movement advocates. If one's fascial body is well trained, that is to say optimally elastic and resilient, then it can be relied on to perform effectively and at the same time to offer a high degree of injury prevention (Kjaer et al. 2009). Until now, most of the emphasis in sports has been focused on the classic triad of muscular strength, cardiovascular conditioning, and neuromuscular coordination (Jenkins 2005). Some alternative physical training activities - such as Pilates, yoga, Continuum Movement, and martial art - are already taking the connective tissue network into account. Here the importance of the fasciae is often specifically discussed, though modern insights in the field of fascia research have often not been specifically included. We suggest that in order to build up an injury-resistant and elastic fascial body network it is essential to translate current insights in the field of fascia research into a practical training program. Our intention is to encourage physical therapists, sports trainers and movement enthusiasts to incorporate the principles presented in this chapter, and to apply them to their specific context.

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Fascial remodeling

A recognized characteristic of connective tissue is its impressive adaptability: when regularly put under increasing yet physiological strain, it changes its architectural properties to meet the demand. For example, through our everyday biped locomotion the fascia on the lateral side of the thigh develops a palpable firmness. If we were instead to spend that same amount of time with our legs straddling a horse, then the opposite would happen, i.e., after a few months the fascia on the inner side of the legs would become more developed and strong (El-Labban et al. 1993).

7.24

The varied capacities of fibrous collagenous connective tissues make it possible for these materials to continuously adapt to the regularly occurring strain, particularly in relation to changes in length, strength and ability to shear. Not only the density of bone changes, for example, as happens with astronauts who spend time in zero gravity wherein the bones become more porous (Ingber 2008); fascial tissues also react to their dominant loading patterns. With the help of the fibroblasts, they react to everyday strain as well as to specific training, steadily remodeling the arrangement of their collagenous fiber network (Kjaer et al. 2009). For example, with each passing year half the collagen fibrils are replaced in a healthy body (Neuberger & Slack 1953). The intention of fascial fitness is to influence this replacement via specific training activities which will, after 6 to 24 months, result in a 'silk-like bodysuit' which is not only strong but also allows for a smoothly gliding joint mobility over wide angular ranges.

PART SEVEN



Fig. 7.24.1 • Increased elastic storage capacity. Regular oscillatory exercise, such as daily rapid running, induces a higher storage capacity in the tendinous tissues of rats, compared with their nonrunning peers. This is expressed in a more spring-like recoil movement, as shown on the left. The area between the respective loading versus unloading curves represents the amount of 'hysteresis': the smaller hysteresis of the trained animals (*grey*) reveals their more 'elastic' tissue storage capacity, whereas the larger hysteresis of their peers signifies their more 'viscoelastic' tissue properties, also called inertia. Illustration modified after Reeves 2006. Reeves ND, Narici MV, Maganaris CN (2006) Myotendinous plasticity to ageing and resistance exercise in humans. Exp Physiol 91(3): 483–498.

Interestingly, the fascial tissues of young people show stronger undulations within their collagen fibers, reminiscent of elastic springs, whereas in older people the collagen fibers appear as rather flattened (Staubesand et al. 1997). Research has confirmed the previously optimistic assumption that proper exercise loading - if applied regularly - can induce a more youthful collagen architecture, which shows a more wavy fiber arrangement (Wood et al. 1988; Jarniven et al. 2002) and which also expresses a significant increased elastic storage capacity (Fig. 7.24.1) (Reeves et al. 2006). However, it seems to matter which kind of exercise movements are applied: a controlled exercise study using slow-velocity and low-load contractions only demonstrated an increase in muscular strength and volume; however, it failed to yield any change in the elastic storage capacity of the collagenous structures (Kubo et al. 2003).

The catapult mechanism: elastic recoil of fascial tissues

Kangaroos can jump much farther than can be explained by the force of the contraction of their leg muscles. Under closer scrutiny, scientists discovered that a spring-like action is behind the unique ability: the so-called 'catapult mechanism' (Kram & Dawson 1998). Here, the tendons and the fascia of the legs are tensioned like elastic bands. The release of this stored energy is what makes the amazing jumps possible. The discovery soon thereafter that the same mechanism is also utilized by gazelles was hardly surprising. These animals are also capable of impressive leaping as well as running, though their musculature is not especially powerful. On the contrary, gazelles are generally considered to be rather delicate, making the springy ease of their incredible jumps all the more interesting.

The possibility of high-resolution ultrasound examination made it possible to discover similar orchestration of loading between muscle and fascia in human movement. Surprisingly, it has been found that the fasciae of humans have a similar kinetic storage capacity to that of kangaroos and gazelles (Sawicki et al. 2009). This is not only made use of when we jump or run but also with simple walking, as a significant part of the energy of the movement comes from the same springiness described above. This new discovery has led to an active revision of long-accepted principles in the field of movement science.

In the past, it was assumed that in a muscular joint movement, the skeletal muscles involved shorten and this energy passes through passive tendons, which results in the movement of the joint. This classic form of energy transfer is still true – according to these recent measurements – for steady movements such as bicycling. Here, the muscle fibers actively change in length, while the tendons and aponeuroses scarcely grow longer. The fascial elements remain quite passive. This is in contrast to oscillatory movements with an elastic spring quality, in which the length of the muscle fibers changes little. Here, the muscle fibers contract in an almost isometric fashion (they stiffen temporarily without any significant change of their length) while the fascial elements function in an elastic way with a movement similar to that of a yo-yo (Fig. 7.24.2). It is this lengthening and shortening of the fascial elements that 'produces' the actual movement (Fukunaga et al. 2002; Kawakami et al. 2002).

It is of interest that the elastic movement quality in young people is associated with a typical twodirectional lattice arrangement of their fasciae, similar to a woman's stocking (Staubesand et al. 1997). In contrast, as we age and usually lose the springiness in our gait, the fascial architecture takes on a more haphazard and multidirectional arrangement. Animal experiments have also shown that lack of movement quickly fosters the development of additional cross-links in fascial tissues. The fibers lose their elasticity and do not glide against one another as they once did; instead, they become stuck together and form tissue adhesions, and in the worst cases they actually become matted together (Fig. 7.24.3) (Jarvinen et al. 2002). The goal of the fascial fitness training is to stimulate fascial fibroblasts to lay down a more youthful and 'gazelle-like' fiber architecture. This is done through movements that load the fascial tissues over multiple extension ranges while utilizing their elastic springiness.

Figure 7.24.4 illustrates different fascial elements affected by various loading regimens. Classic weight training loads the muscle in its normal range of motion, thereby strengthening the fascial tissues, which are arranged in series with the active muscle fibers. In addition, the transverse fibers across the muscular envelope are stimulated as well. However, little effect can be expected on extramuscular fasciae as well as on those intramuscular fascial fibers that are arranged in parallel to the active muscle fibers (Huijing 1999).

Classic Hatha yoga stretches, on the other hand, will show little effect on those fascial tissues which are arranged in series with the muscle fibers, since the relaxed myofibers are much softer than their serially arranged tendinous extensions and will therefore 'swallow' most of the elongation (Jami 1992). However, such stretching provides good stimulation



Fig. 7.24.2 • Length changes of fascial elements and muscle fibres in an oscillatory movement with elastic recoil properties (A) and in conventional muscle training (B). The elastic tendinous (or fascial) elements are shown as springs, the myofibers as straight lines above. Note that during a conventional movement (B) the fascial elements do not change their length significantly while the muscle fibers clearly change their length. During movements like hopping or jumping, however, the muscle fibers contract almost isometrically while the fascial elements lengthen and shorten like an elastic yoyo spring. In Kawakami Y, Muraoka T, Ito S, Kanehisa H, Fukunaga T 2002 In vivo muscle fibre behaviour during countermovement exercise in humans reveals a significant role for tendon elasticity. From Kawakami et al. 2002 J Physiol 540 (2): 635-646



Fig. 7.24.3 • Collagen architecture responds to loading. Fasciae of young people – shown on the left – express more often a clear two-directional (lattice) orientation of their collagen fiber network. In addition, the individual collagen fibers show a stronger crimp formation. In contrast, fasciae from older persons show a more irregular alignment with less crimp formation. As evidenced by animal studies, application of proper exercise can induce an altered architecture with increased crimp-formation. Lack of exercise, on the other hand, has been shown to induce formation of a multidirectional fiber network and a decreased crimp formation.

for fascial tissues which are hardly reached by classic muscle training, such as the extramuscular fasciae and the intramuscular fasciae oriented in parallel to the myofibers. Finally, a dynamic muscular loading pattern in which the muscle is both activated and extended promises a more comprehensive stimulation of fascial tissues. This can be achieved by muscular activation (e.g. against resistance) in a lengthened position while requiring small or medium amounts of muscle force only. Soft elastic bounces in the end ranges of available motion can also be utilized for that purpose. The following guidelines are developed to make such training more efficient.

Training principles

Preparatory countermovement

Here, we make use of the catapult effect described above. Before we perform the actual movement, we start with a slight pretensioning in the opposite direction. This is comparable with using a bow to shoot an arrow; just as the bow has to have sufficient tension in order for the arrow to reach its goal, the fascia becomes actively pretensioned in the opposite direction. In a sample exercise called 'the flying sword', the pretensioning is achieved as the body's axis is slightly tilted backward for a brief moment, while at the same time there is an upward lengthening (Fig. 7.24.5). This increases the elastic tension in the fascial bodysuit and as a result allows the upper body and the arms to spring forward and down like a catapult as the weight is shifted in this direction.

The opposite is true for straightening up - we activate the catapult capacity of the fascia through an active pretensioning of the fascia of the back. When standing up from a forward bending position, the muscles on the front of the body are first briefly activated. This momentarily pulls the body even further forward and down and at the same time the fascia on the posterior fascia is loaded with greater tension. The energy which is stored in the fascia is dynamically released via a passive recoil effect as the upper body 'swings' back to the original position. To be sure that the individual is not relying on muscle work, but rather on dynamic recoil action of the fascia, requires a focus on timing - much the same as when playing with a vo-vo. It is necessary to determine the ideal swing, which is apparent when the action is fluid and pleasurable.

The Ninja principle

This principle is inspired by the legendary Japanese warriors who reputedly moved as silently as cats and left no trace. When performing bouncy movements such as hopping, running and dancing, special attention needs to be paid to executing the movement as smoothly and softly as possible. A change in direction is preceded by a gradual deceleration of the movement before the turn and a gradual acceleration afterwards, each movement flowing from the last; any extraneous or jerky movements should therefore be avoided (Fig. 7.24.6).

Normal stairs become training equipment when they are used appropriately, employing gentle stepping. The production of 'as little noise as possible' provides the most useful feedback – the more the fascial spring effect is utilized, the quieter and gentler the process will be. It may be useful to reflect on the way a cat moves as it prepares to jump. The feline first sends a condensed impulse down through its paws in order to accelerate softly and quietly, landing with precision.



Fig. 7.24.4 • Loading of different fascial components. A Relaxed position: the myofibers are relaxed and the muscle is at normal length. None of the fascial elements is being stretched. B Usual muscle work: myofibers contracted and muscle at normal length range. Fascial tissues, which are either arranged in series with the myofibers or transverse to them, are loaded. C Classic stretching: myofibers relaxed and muscle elongated. Fascial tissues oriented parallel to the myofibers are loaded as well, as are extramuscular connections. However, fascial tissues oriented in series with the myofibers are not sufficiently loaded, since most of the elongation in that serially arranged force chain is taken up by the relaxed myofibers. D Actively loaded stretch: muscle active and loaded at long end range. Most of the fascial components are being stretched and stimulated in that loading pattern. Note that various mixtures and combinations between the four different fascial components exist. This simplified abstraction therefore serves as a basic orientation only.

Dynamic stretching

Rather than a motionless waiting in a static stretch position, a more flowing stretch is suggested. In fascial fitness there is a differentiation between two kinds of dynamic stretching: fast and slow. The fast variation may be familiar to many people as it was part of physical training in the past. For several decades, this bouncing stretch was considered to be generally harmful to the tissue, but the method's merits have been confirmed in recent research. Although stretching immediately before competition can be counterproductive, it seems that long-term and regular use of such dynamic stretching can positively influence the architecture of the connective tissue in that it becomes more elastic when correctly performed (Decoster et al. 2005). Muscles and tissue should first be warmed up, and jerking or abrupt movements should be avoided. The motion should have a sinusoidal deceleration and acceleration shape in each direction turn; this goes along with the

perception of a smooth and 'elegant' quality of movement. Dynamic, fast stretching has even more effect on the fascia when combined with a preparatory countermovement, as was previously described (Fukashiro et al. 2006). For example, when stretching the hip flexors, a brief backward movement should be introduced before dynamically lengthening and stretching forwards.

The long myofascial chains are the preferred focus when doing slow dynamic stretches. Instead of stretching isolated muscle groups, the aim is finding body movements that engage the longest possible myofascial chains (Myers 1997). This is not done by passively waiting, as in a lengthening classic Hatha yoga pose, or in a conventional isolated muscle stretch. Multidirectional movements, with slight changes in angle are utilized; this might include sideways or diagonal movement variations as well as spiraling rotations. With this method, large areas of the fascial network are simultaneously involved (Fig. 7.24.7).



Fig. 7.24.5 • Training example: the flying sword. A Tension the bow: the preparatory countermovement (pre-stretch) initiates the elastic-dynamic spring in an anterior and inferior direction. Free weights can also be used. **B** To return to an upright position, the 'catapulting back fascia' is loaded as the upper body is briefly bounced dynamically downwards followed by an elastic swing back up. The attention of the person doing the exercise should be on the optimal timing and calibration of the movement in order to create the smoothest movement possible.



Fig. 7.24.6 • Training example: elastic wall bounces. Imitating the elastic bounces of a gazelle, soft-bouncing movements off a wall are explored in standing. Proper pretension in the whole body will avoid any collapsing into a 'banana posture.' Making the least sound and avoiding any abrupt movement qualities are imperative. Only with the mastery of these qualities can a progression into further load increases – e.g., bouncing off a table or window sill instead of a wall – eventually be explored by stronger individuals. For example, this person should not yet be permitted to progress to higher loads, as his neck and shoulder region already show slight compression on the left picture.



Fig. 7.24.7 • Training example: the big cat stretch. A This is a slow stretching movement of the long posterior chain, from the fingertips to the sitbones, from the coccyx to the top of the head and to the heels. The movement goes in opposing directions at the same time – think of a cat stretching its long body. By changing the angle slightly, different aspects of the fascial web are addressed with slow and steady movements. B In the next step one rotates and lengthens the pelvis or chest towards one side (here shown with the pelvis starting to rotate to the right). The intensity of the feeling of stretch on that entire side of the body is then gently reversed. Note the feeling of increased length afterwards.

Proprioceptive refinement

The importance of proprioception for movement control is made clear by the case of Ian Waterman, a man repeatedly mentioned in scientific literature. This impressive man contracted a viral infection at the age of 19 which resulted in a so-called 'sensory neuropathy.' In this rare pathology, the sensory peripheral nerves which provide the somatomotor cortex with information about the movements of the body are destroyed, while the motor nerves remain completely intact. This meant that Mr. Waterman could move, but he could not 'feel' his movements. After some time, this giant of a man became virtually lifeless. Only with an iron will and years of practice did he finally succeed in making up for these normal physical sensations, a capacity that is commonly taken for granted. He did so with conscious control that primarily relies on visual feedback. He is currently the only person known with this affliction who is able to stand unaided, as well as being able to walk (Cole 1995).

The way Waterman moves is similar to the way patients with chronic back pain move. When in a public place, if the lights unexpectedly go out, he clumsily falls to the ground (see BBC documentary: The man who lost his body, http://bbc-horizon-1998-the-man-who-lost-his-7812922.cooga.net). Springy, swinging movements are possible for him only with obvious and jerky changes in direction.

If doing a 'classic' stretching program with static or active stretches, he would appear normal. As for the dynamic stretching that is part of our fascial training, he is clearly not capable, as he lacks the proprioception needed for fine coordination. It is interesting to note that the classic 'joint receptors' located in joint capsules and associated ligaments have been shown to be of lesser importance for normal proprioception, since they are usually stimulated at extreme joint ranges only, and not during physiological motions (Lu et al. 2005; Ianuzzi et al. 2011). On the contrary, proprioceptive nerve endings located in the more superficial layers are more optimally situated, as here even small angular joint movements lead to relatively distinct shearing motions. Recent findings indicate that the superficial fascial layers of the body are, in fact, more densely populated with mechanoreceptive nerve endings than tissue situated more internally (Stecco et al. 2008; Tesarz et al., in Press).

For this reason, we encourage a perceptual refinement of shear, gliding, and tensioning motions in superficial fascial membranes. In doing this, it is important to limit the filtering function of the reticular formation, as it can markedly restrict the transfer of sensations from movements which are repetitive and predictable. To prevent such a sensory dampening, the idea of varied and creative experiencing becomes important. In addition to the slow and fast dynamic stretches noted above, as well as utilizing elastic recoil properties, we recommend (based on experience) inclusion of 'fascial refinement' training in which various qualities of movement are experimented with, e.g., extreme slow-motion and very quick micro-movements which may not even be visible to an observer, and large macro-movements involving the whole body. To this end, it is common to place the body into unfamiliar positions while working with the awareness of gravity, or possibly through exploring the weight of a training partner.

The micro-movements are inspired by Continuum Movement (Conrad 2007). Such movement is active and specific and can have effects which are not possible with larger movements. In doing these coordinated fascial movements, it appears possible to specifically address adhesions, for example, between muscle septa deep in the body. In addition, such tiny and specific movements can be used to illuminate and bring awareness to perceptually neglected areas of the body (Fig. 7.24.8). Thomas Hanna uses the label 'sensory-motor amnesia' when referring to such places in the body (Hanna 1998).

Hydration and renewal

The video recordings of fascia by Dr. Guimbertau (see Chapter 3.6) have helped our understanding of the plasticity and changing elasticity of the water-filled fascia. This awareness has proven to be especially effective when incorporated into the slow dynamic stretching and fascial refinement work. An essential basic principle of these exercises is the understanding that the fascial tissue is predominantly made up of free-moving and boundwater molecules. During the strain of stretching, the water is pushed out of the more stressed zones, similar to squeezing a sponge (Schleip & Klingler 2007). With the release that follows, this area is again filled with new fluid which comes from surrounding tissue as well as the lymphatic and vascular network. The sponge-like connective tissue can lack adequate hydration at neglected places. The goal of exercise is to refresh such places in the body with improved hydration through specific stretching to encourage fluid movement.

Proper timing of the duration of individual loading and release phases is very important. As part of modern running training, it is often recommended to frequently interrupt the running with short walking intervals (Galloway 2002). There is good reason for this: under strain, the fluid is pressed out of the fascial tissues and these begin to function less optimally as their elastic and springy resilience slowly decreases. The short walking pauses then serve to rehydrate the tissue, as it is given a chance to take up nourishing fluid. For an average beginning runner, for example, the authors recommend walking pauses of 1 to 3 minutes every 10 minutes. More advanced runners with more developed body awareness can



Fig. 7.24.8 • Training example: octopus tentacle. With the image of an octopus tentacle in mind, a multitude of extensional movements through the whole leg are explored in slow motion. Through creative changes in muscular activation patterns, the tensional fascial proprioception is activated. This goes along with a deep myofascial stimulation that aims to reach not only the fascial envelopes but also into the septa between muscles. While avoiding any jerky movement, the action of these tentacle-like micro-movements leads to a feeling of flowing strength in the leg.

This cyclic training, with periods of more intense effort interspersed with purposeful breaks, is recommended in all facets of fascia training. The person training then learns to pay attention to the dynamic properties of their fascial 'bodysuit' while exercising, and to adjust the exercises based on this new body awareness. This also carries over to an increased 'fascial embodiment' in everyday life. Preliminary anecdotal reports also indicate a preventative effect of a fascia-oriented training in relation to connective tissue overuse injuries.

The use of special foam rollers can be a useful tool for inducing a localized sponge-like temporary tissue dehydration with resultant renewed hydration. However, the firmness of the roller and application of the body weight needs to be individually monitored. If properly applied and including very slow and finely tuned directional changes only, the tissue forces and potential benefits could be similar to those of manual myofascial release treatments (Chaudhry et al. 2008). In addition, the localized tissue stimulation might serve to stimulate and fine-tune possibly inhibited or desensitized fascial proprioceptors in more hidden tissue locations (Fig. 7.24.9).

Sustainability: the power of a thousand tiny steps

An additional and important aspect is the concept of the slow and long-term renewal of the fascial network. In contrast to muscular strength training in which big gains occur early on and then a plateau is quickly reached wherein only very small gains are possible, fascia changes more slowly and the results are more lasting. It is possible to work without a great deal of strain - so that consistent and regular training pays off. When training the fascia, improvements in the first few weeks may be small and less obvious on the outside. However, improvements have a lasting cumulative effect which, after years, can be expected to result in marked improvements in the strength and elasticity of the global fascial net (Fig. 7.24.10) (Kjaer et al. 2009). As the fascial proprioception becomes refined, improved coordination is probable.

It is suggested that training should be consistent, and that only a few minutes of appropriate exercises, performed once or twice per week, is sufficient for collagen remodeling. The related renewal process will take between 6 months and 2 years and will yield a lithe, flexible and resilient collagenous



Fig. 7.24.9 • Training example: fascial release. The use of particular foam rollers may allow the application of localized tissue stimulations with similar forces and possibly similar benefits as in a manual myofascial release session. However, the stiffness of the roller and application of the body weight needs to be adjusted and monitored for each person. To foster a sponge-like tissue dehydration with subsequent renewed local hydration, only slow-motion, subtle changes in the applied forces and vectors are recommended



exercise. The upper curve shows collagen synthesis in tendons is increasing after exercise. However, the stimulated fibroblasts also increase their rate of collagen degradation. Interestingly, during the first 1-2 days following exercise, collagen degradation outweighs the collagen synthesis, whereas afterwards this situation is reversed. To increase tendon strength, the proposed fascial fitness training therefore suggests an appropriate tissue stimulation 1 to 2 times per week only. While the increased tendon strength is not achieved by an increase in tendon diameter, recent examinations by Kjaer et al. (2009) indicated that it is probably the result of altered cross-link formations between collagen fibers. (From Magnusson et al. 2010, with permission.) Magnusson SP, Langberg H, Kjaer M (2010) The pathogenesis of tendinopathy: balancing the response to loading. Nat Rev Rheumatol 6(5): 262-268

matrix. For those who do yoga or martial arts, such a focus on a long-term goal is nothing new. For the person who is new to physical training, such knowledge of fascial properties can go a long way in convincing them to train their connective tissues.

Of course, fascial fitness training should not replace muscular strength work, cardiovascular training and coordination exercises; instead, it should be thought of as an important addition to a comprehensive training program.

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Fascia: Clinical and fundamental scientific research

Considering the scientific process

Peter A Huijing

Many of the chapters of this book deal with fascia in relation to practical aspects of the manual therapies.

The chapters within this section (and a few that, for practical reasons, ended up in other sections of the book, see Chapters 1.1, 3.1–3.2, and 5.8) are in some ways quite different: The knowledge described in the chapters of this section is mostly a result of fundamental scientific research, which in most cases did not have the manual therapies or other application in mind, and initially, may not even have had the fascia as the target. An example in case is the work on force transmission that was aimed at studying muscle function per se. However, unexpected and thought-stimulating results have indicated a new direction in which all kinds of fascial structures of the body are likely to play a major role.

Fundamental scientific work is necessary for future developments in science, but also in therapy in general, and likely also in the manual therapies. A problem with fundamental scientific work is that it is impossible to predict which work and which specific results will be highly relevant for practical application in the clinical fields. To politicians, science managers and clinicians, without an open mind, as well as a clear understanding of the actual process of scientific advancement, the scientific process described is highly unattractive, because money needs to be spent on projects for which the full outcome is unclear. The only thing that can be tested for is scientific quality, and even that is not always an objective process. Only in retrospect (sometimes many decades or in exceptional cases centuries later) is clarity provided as a few relatively small scientific steps ahead are combined into something very new. Then it also becomes clear that a lot of work was performed that did not yield any results for practical application even though some of it may have advanced general understanding of fundamental principles.

However, Comroe & Dripps (1974) analyzed and reported crucial sources of the top ten clinical advances in cardiovascular and pulmonary medicine and surgery over a period of 30 years (within the last century). Their conclusion was that 41% of over 500 key articles that allowed or contributed to these advances in a major way were written by scientists who had no direct interest in disease, and that 62% were the result of basic research. These results have later been the subject of sometimes heated scientific debate (e.g., Smith 1987) that does not always seem fully free of external motives (competition for limited available resources). Regardless of the proper scientific questions that may be asked concerning this, it is clear that both clinical and fundamental research activity is essential for the advancement of knowledge that may allow changing clinical practice.

The above may create the impression that the role of scientists is limited to just creating new knowledge. Even though that is their primary task, scientists have a moral obligation to be interested in the application of their work as well, if the knowledge gathered has reached a level where application is likely. The authors of more fundamentally oriented chapters of this book seem to be fully aware of this.

After contact between clinicians and scientists has been established, difficulties of language will inevitably arise (it should be clear that we do not mean different mother tongues by this, even though that can contribute to the confusion) and need to be overcome through agreement on a common set of ideas and nomenclature, before fundamental-clinical collaboration can work to advance knowledge and understanding.

Since both types of workers do that from a quite different perspective, the exchange of ideas will certainly not be unidirectional, but create potential chances for new insights on both sides. The recent interactions between clinicians and scientists in the socalled Fascia Research Congresses are good examples of willingness to create such common conditions, and also one of the major drives for the present book.

One factor in science may be quite puzzling to the non-scientist, and that is controversy. Actually, controversy on content and ideas is a major driving force of science and therefore an essential part of it. The intellectual clash of minds is crucial to filter out confounding information and select generally accepted methods and concepts. For that reason we have chosen not to remove all controversy from the content of the chapters. For example, among the scientific authors of this book, ideas about the importance of the continuity of the connective tissues in, for example, a limb may differ quite a bit: Some are convinced that the physiological effects of such continuity are limited to the borders of a fascicle within a muscle, and others think that they have sufficient evidence that intermuscular mechanical interaction is a prominent feature. If you read with an eye for detail you may even notice that the co-authors of one chapter may differ.

This means that the scientific (but probably also the clinical) material of this book should not be looked at as a static feature, but as knowledge that will be developing continually. In that sense we are certainly not presenting "the truth" as an unchanging character. By incorporating such aspects, this book will not be like most textbooks.

The dynamics of this process also require of both scientists and clinicians that they try to keep up with developments in each others' fields. That is certainly not an easy task, but it remains an essential one. It requires also from both sides that "unwanted" results (deviating from the preconceptions of each profession) should be fully considered in detail and accepted if sufficient evidence is presented.

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Imaging

Ultrasound

Helene Langevin Yasuo Kawakami

Introduction

Ultrasonography is now widely used to examine human as well as animal tissues. For this purpose, brightness-mode (B-mode) ultrasonography is often used. This mode creates a two-dimensional image of a tissue section by visualizing the portions where acoustic impedance (tissue density \times acoustic velocity) changes (Noce 1990). There are distinct differences in acoustic impedance at interfaces between adipose and muscle tissues and bones, thereby enabling delineation of their peripheries by ultrasonography. Ultrasound is also emerging as a useful tool to image and measure the structure and organization of the connective tissue network in normal and pathological conditions. During ultrasound imaging of biological materials, echoes generated by relatively homogeneous material (e.g., fat-containing areolar connective tissue) produce diffusely scattered signals, while echoes generated by interfaces of organized tissues (e.g., dense connective tissue layers) produce more correlated "specular" signals (Insana et al. 1985; Garra 1993; Kremkau 1998; Lizzi et al. 2006). Dense and areolar connective tissue planes, respectively, appear as echogenic and echolucent bands in two-dimensional ultrasound images (Langevin et al. 2007). Ultrasonography, therefore, is a useful tool to both visualize and quantify the structural characteristics of the dense and areolar connective tissue components of fasciae. Ultrasonography was first applied to humans by Howry & Bliss (1952), and used for the purpose of viewing the cross-section of skeletal muscles (Howry 1965). B-mode ultrasonography was then used to measure

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cross-sectional areas of skeletal muscles by Ikai & Fukunaga (1968). Since then, B-mode ultrasonography has been used to obtain sectional images of skeletal muscles in vivo. Spatial, as well as time resolution, of ultrasonography is rapidly improving.

When an ultrasonic probe of appropriate frequency (normally between 3 and 10 MHz, depending on the depth of the muscle) is placed on the skin in a longitudinal direction of the underlying muscle, one can observe hypoechoic striae within the muscle between the horizontal echoes (Fig. 8.2.1a). The former are from echogenic structures between perimysia such as fibroadipose septa (Fornage 1989), and the latter from the epimysia and aponeuroses. Kawakami et al. (1993) and Narici et al. (1996) confirmed that the striated patterns represent orientations of fascicles. Fascicle length and angle (with respect to aponeuroses) can be determined by measuring the length of a representative echo within the muscle and its angulation relative to the underlying echo (Fig. 8.2.1a). Among the factors affecting the force that a muscle can produce, the length and contractile velocity of its fibers are particularly important. This is because the force exerted by a muscle fiber is determined by its length and contraction velocity. For muscles like the triceps surae, in which muscle fibers run from the proximal to distal end of each fascicle (Kawakami et al. 2000a), measurement of the fascicle length can provide information of how muscle fibers develop forces. In this regard there is a particular advantage of ultrasonography, in that it enables measurement of fascicle behavior during contraction in real time. The disadvantage of ultrasonography may be a relatively limited scan



Fig. 8.2.1 • B-mode ultrasound images of human muscles. (A) Transverse (left panel, lateral-medial) and longitudinal (right panel, distal-proximal) B-mode ultrasound images of the human gastrocnemius medialis muscle. From top to bottom, skin to deep layer. In the right panel, a white line is drawn along a representative echo parallel to fascicle orientation. (B) Three-dimensional reconstruction of ultrasound images of the human gastrocnemius medialis muscle (Kawakami et al. 2000a). A part of the soleus muscle is also visualized. (C) B-mode ultrasound images of the triceps brachii muscle (long head) of a normal individual (top panel) and a highly trained bodybuilder (bottom panel). From right to left, distal to proximal. (D) B-mode ultrasound images of the gastrocnemius medialis muscle at rest (top) and during maximal isometric contraction (bottom). From right to left, distal to proximal.

area and two-dimensional, planar information of fascicles and tendinous structures which in reality are three-dimensional (Scott et al. 1993). These drawbacks can be partly solved by a three-dimensional ultrasound system in which multiple ultrasound images are reconstructed three-dimensionally (Fig. 8.2.1b) (Kawakami et al. 2000b).

From ultrasonic observation of individuals with widely varying hypertrophic status of skeletal muscles, Kawakami et al. (2006) found relationships between muscle size and fascicle angles for some limb muscles. Fig. 8.2.1c illustrates longitudinal images of the triceps brachii muscle that show an outstanding hypertrophic response (Kawakami et al. 1993; 2006). In a highly hypertrophied triceps, fascicles are packed curvilinearly with large angles from the deep aponeurosis. The angulation of fascicles relative to aponeuroses is typically seen in pennate muscles, and determines the component of muscle fiber forces in the direction of the line of action of muscle (Huijing et al. 1989). Large variability of fascicle angles suggests different degrees of fiber-tendon force transmission between individuals, and there is some evidence in support of this (Ikegawa et al. 2008).

Figure 8.2.1(d) shows longitudinal ultrasonic images of the gastrocnemius medialis muscle at rest and during maximal isometric contraction (Kawakami & Fukunaga 2006). Although this is a fixed-end contraction where the whole muscle-tendon unit length is kept constant, one can clearly observe changes in fascicle orientations by contraction: fascicles shorten with increasing angles. Contraction thus induces deformation of fascial organization of the muscle. Shortening of fascicles occurs at the expense of elongations of the tendinous structures (Griffiths 1991; Kawakami et al. 1998). During dynamic human movements, this muscletendon interaction plays an extremely important role. Kawakami et al. (2002) used ultrasonography to track length changes of the gastrocnemius fascicles during ankle hopping preceded by a counter-movement, and showed that fascicles contract isometrically when the muscle-tendon unit is being lengthened. In this phase, tendinous structures are lengthened and store elastic energy which is released during the shortening phase that follows, to add to positive mechanical work. A similar mechanism has been found during human walking (Fukunaga et al. 2001).

Previous studies have shown that the mechanical properties of tendinous structures, especially those of the aponeuroses, depend on the contractile status (passive or active, static or dynamic, long or short lengths) of fascicles (Zuurbier & Huijing 1992; Zuurbier et al. 1994; Lieber et al. 2000; Kato et al. 2005; Sugisaki et al. 2005). Hence, the mechanical behavior of the whole fascial continuum could change during contractions involving dynamic fascicle length changes under varying contraction intensities. If so, the interface between the aponeurosis, tendon, and fascicles could be stressed locally. In the human gastrocnemius muscle, this position corresponds to the distal end of the muscle belly, where muscle strain frequently occurs (Fig. 8.2.2). Fascial structures inside and outside the muscle, and their

Imaging



Fig. 8.2.2 • (A) Legs of an individual who experienced muscle strain at the distal end (triangle) of muscle belly of the gastrocnemius medialis. (B) Three-dimensional reconstruction of ultrasound images of the calf. Triangle represents the distal end of gastrocnemius belly. (C) B-mode ultrasound images of the distal end of gastrocnemius belly immediately after strain injury (top) and after healing (bottom).

behavior during contraction, are therefore physiologically and clinically relevant, and ultrasonography is a powerful tool to examine these in vivo.

Connective tissue outside of muscle also forms a complex, interconnected network that is increasingly recognized to play an important role in musculoskeletal function. In humans, superficial and deep fasciae are composed of layers of densely woven connective tissue alternating with layers of "loose", areolar connective tissue containing varying amounts of fat (Benjamin 2009; Huijing & Langevin 2009). An important function of the compliant, areolar layers is to allow the dense connective tissue layers to glide past one another (Stecco et al. 2006). Pathological conditions such as injury, inflammation, scarring and fibrosis can cause changes in the structure of connective tissue. For

example, human subjects with chronic low back pain were found to have increased thickness of perimuscular connective tissue in the lumbar region, compared with subjects without low back pain (Langevin et al. 2009). Therefore, non-invasive measures of connective tissue structure and function are important to develop a better understanding of connective tissue physiology as well as evaluate connective tissue pathology and the effects of treatments.

Imaging of extramuscular fascial structures and additional analysis

Combined ultrasound and histology examinations of the same tissue in human subjects undergoing surgery showed an excellent concordance between longitudinal echogenic sheets in 3D renditions of ultrasound images and collagenous sheets seen in 3D reconstructions of corresponding histological preparations (Plate 8.2.1). This was confirmed using a method commonly used in geostatistics (Goovaerts 1994) to evaluate structural continuity in the structure of subsurface materials (soil lithology) using ground penetrating radar or soil boring data (Castrignano et al. 2000; Petrone et al. 2004). In this method, spatial correlations are calculated within a range of distances from each data point in a chosen direction to generate semivariogram plots (customarily termed "variograms"). Parameters such as range, sill and nugget are used to evaluate the structure of spatial data sets (e.g., smoothness, roughness) in a given direction or plane (Robertson 1987; Goovaerts 1998). For example, laminar structures with high spatial continuity yield highly correlated data in the direction of the laminations. Variogram analyses of ultrasound and histology images showed that rank correlations between serial ultrasound and corresponding histology images were highly correlated both parallel (r = 0.79, p < 0.001) and perpendicular (r = 0.63, p < 0.001)p < 0.001) to the surface of the skin, indicating concordance in spatial structure between the two data sets.

In addition to evaluating connective tissue structure, continuous ultrasound recording during dynamic tissue perturbation can be used to evaluate the dynamic mechanical properties of the tissue. Acupuncture needles provide convenient mechanical probes to achieve specific mechanical stimulation of connective tissue. Acupuncture needle rotation causes winding of collagen around the needle, creating a tight mechanical coupling between needle and tissue (Langevin et al. 2001a,b, 2002). This is demonstrated in ex-vivo experiments using high frequency (50 MHz) ultrasound scanning acoustic microscopy of rat abdominal wall tissue explants with and without acupuncture needle rotation, followed by tissue fixation and histological examination (Langevin et al. 2002). This technique revealed prominent spiral patterns centered on the needle in ultrasound and histology images with needle rotation (Plate 8.2.2a). Fourier analysis performed on radial ultrasound scan lines centered on the needle showed significantly increased periodic order with rotation compared with no rotation.

Acupuncture needles also can be used to quantify the dynamic mechanical behavior of human connective tissue in vivo using techniques derived from ultrasound elastography. Ultrasound elasticity imaging is rapidly becoming a valuable noninvasive method for analyzing spatial patterns of tissue stiffness in vivo (e.g., in detection of localized stiffer tissue areas associated with breast and prostate malignancies). In the original elastography method, successive ultrasound images are acquired while the ultrasound probe compresses the tissue (Ophir et al. 1991; Konofagou & Ophir 1998). In the acupuncture needling method, the ultrasound probe remains stationary and acupuncture needle motion is used to produce the tissue displacement and deformation (Plate 8.2.2b)

(Langevin et al. 2004). As in classical elastography, the radio-frequency (RF) ultrasound data are processed off-line to generate displacement and strain images reflecting tissue behavior during needle movement (Ophir et al. 1999). Tissue displacement between successive ultrasound frames is calculated using cross-correlation techniques generating a series of displacement images (Plate. 8.2.2c). Using this method, it can be demonstrated that the mechanical coupling produced by needle rotation influences the behavior of the tissue during subsequent upward and downward needle movement. Increasing the amount of rotation has a significant linear effect on tissue displacement during downward and upward needle motion, as well as on rebound tissue displacement after downward needle movement (Langevin et al. 2004).

In conclusion, ultrasound allows both imaging and quantification of connective tissue and muscle structure as well as dynamic responses to local mechanical perturbations. Changes in connective tissue organization and biomechanical behavior may be important components of the pathophysiology of many conditions including chronic musculoskeletal pain (Langevin & Sherman 2007). Ultrasound-derived techniques such as those described above provide noninvasive tools that can be used as the outcome measure in translational studies investigating pathogenic and treatment mechanisms as well as clinical responses to treatments.

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Advanced MRI techniques for in-vivo biomechanical tissue movement analysis

8.3

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Introduction

If a moving body can be observed directly (whole body or its extremities), surface markers can be used and commercially available movement analysis systems (e.g., for gait analysis) exist for biomechanical analysis. Our focus in this chapter is on much more detailed quantitative analysis of moving tissues inside the body using magnetic resonance imaging (MRI).

MRI has sufficient soft tissue contrast and ability to view into different compartments, so it is ideal for and utilized heavily in routine anatomical soft tissue imaging. In recent years, with combined advances in fast imaging hardware, innovative movement imaging protocols, and advanced computational image processing tools, dynamic musculoskeletal imaging has moved more and more into the mainstream of clinical imaging practice. Although most of its current use remains qualitative, just showing 2D or 3D image sets in series as movies, practical techniques for detailed quantitative biomechanical analysis of moving tissues are available today for inquisitive clinicians and for researchers who are trying to unravel delicate relationships between soft tissue components during locomotion in vivo.

In the past, dynamic motion imaging utilized clinically inapplicable methods, such as 3D X-ray stereophotogrammetry that required the insertion of metal balls into the bones (Lundberg 1989). Computerized tomography (CT), with recent advances in its hardware and speed, can be utilized for the same purpose, if the focus is predominantly on the bones (Crisco et al. 1999). CT with advanced post-processing tools eliminates the need for implantable markers; however, X-ray doses become critical when multiple 3D datasets are needed. In comparison, ultrasound imaging produces relatively low resolution images for soft tissue movement analysis with modest contrast only at some of the tissue boundaries, but its use is much cheaper.

MRI, without ionizing radiation effects, is ideal for repeated and prolonged experiments, routinely required in musculoskeletal research. The basic principle is that molecules (e.g., hydrogen nuclei) acting like dipoles (their top-like movement is referred to as spin) are aligned by a static magnetic field and, by dynamic high-frequency radio signals, specific molecules are given special magnetic properties which can be imaged. Since these magnetic properties are temporary, real tissue properties can be used to study dynamic change of the tissues. MRI has an additional advantage that images can be acquired at positions and orientations defined by the user, as long as the subjects are not claustrophobic and the planned movement can be performed within the scanner's confined spaces. Ongoing efforts to provide nonclaustrophobic machines (such as open magnets) will help dynamic musculoskeletal imaging only in a limited way, since most of such systems suffer from signal-to-noise limitations due to their low static magnetic fields (yielding lower temporal and/or spatial resolution). Newer short- and wide-bore magnets with 1.5 T or higher magnetic fields provide some marginal solution, since movements are still restricted. Standing during MR imaging, most exercise routines, and weight bearing is still not common practice except with few specialized systems (Gilbert et al. 2006) or creative solutions. Most of the time, the equipment costs (both at startup and maintenance) and expertise required for its advanced utilization are the real limitations of dynamic MR imaging.

This chapter is divided into three additional sections: (1) introductory information about MR image formation, also giving a brief overview of classical musculoskeletal movement imaging and the tools utilized to analyze these dynamically acquired image sets. (2) A case study, involving aspects of therapeutic-like loading. (3) The introduction of some more advanced movement imaging techniques, allowing in-vivo measurements of local tissue displacement, velocity and even strain.

Dynamic MRI and in-vivo movement analysis

Routine MR imaging is usually a slow process; image information is gathered step by step by collecting a set of signals (echoes) at different excitation scenarios. At each step, unique phase and frequency information is embedded on the individual signals, based on their physical locations, which later helps their actual coordinate identification. The combined signals are filled one echo at a time into the "k-space" and the image is obtained via Fourier transformation (Stark & Bradley 1999). Since different echoes are obtained at different times, movement of the tissues could produce significant artifacts within images. Therefore, in routine MR imaging, movement is not welcomed; for example, cardiac MRI should deal with heart and respiratory motion during the image acquisition: a first approach is acquiring consecutive images as rapidly as possible ("real-time" MRI) and the second approach is segmented k-space imaging (Haacke et al. 1999).

Real-time MRI has limited spatial resolution (due to the limited number of k-space lines that can be acquired at a given time), and relatively poorer temporal resolution (typically 50–300 ms depending on spatial resolution). Improvements are achieved using echo-planar techniques (i.e., collecting several kspace lines per excitation (Epstein et al. 1999), by using longer and more exotic (e.g., spiral) k-space readout trajectories (Meyer et al. 1992) and by employing parallel imaging techniques requiring multiple receiver coils (Pruessmann et al. 1999), each with varying signal–noise-ratio (SNR) penalties.

Segmented k-space imaging is used extensively in cardiac imaging, permitting the acquisition of a set of

several heartbeats in a single ECG-gated, breath-hold scan. The main idea is to repeat the task and synchronize image acquisition and task repetition. This is achieved by partitioning the k-space data matrix into several "segments". The data of each k-space segment is acquired at a single repetition and successive segments are acquired in following repetitions (heartbeats). The image is effectively an average of all repetitions but could provide a series of temporal snapshots of a repetitive motion. The temporal resolution can be improved by reducing the segment size, at the price of an increased number of repetitions and total imaging time. Segmented k-space imaging techniques employ interpolation techniques to adjust for slight variations in the cyclic heart motion (Feinstein et al. 1997). Please note that heart movements can usually be considered as reliably repeatable, but this is not the case for some dynamic musculoskeletal movements. For further reading on fast imaging techniques, the reader is referred to one of several review articles, focusing mostly on cardiac implementations (Reeder & Faranesh 2000). Historically, heart movement analysis, especially of the left ventricle, has been the main model focus. Generic deformable surface models (Pentland & Horowitz 1991), models exploiting curvature information (Duncan et al. 1991), and 4D models with temporal constraints (Shi et al. 1994) have been described previously for cardiac analysis.

images at multiple cardiac phases over the course of

In musculoskeletal imaging, detailed kinematical models are built using MRI-derived 3D structures. For example, the peritalar joint complex is analyzed in vivo using 3D data sets acquired during foot movement at eight positions, ranging from extreme pronation to extreme supination (Stindel et al. 2001). Similarly, shoulder kinematics is examined using sequential increments of arm endo–exorotation and comparison to 3D models of glenoid and humerus (Rhoad et al. 1998). Other examples are movement analysis of the spine (McGregor 2001), patella (Sheehan 1999), wrist (Keir 2001), or building models for general locomotion (Arnold 2000).

Another set of applications of dynamic MRI involves detailed evaluation of internal movements occurring physiologically or in pathological conditions. For these applications, subjects are asked to perform certain maneuvers (e.g., straining) while inside the magnet, and newly acquired images are compared with control images (e.g., in assessment of pelvic floor defects; Rentsc 2001). Although the aim is to confirm initial diagnoses and evaluate the anatomy of defects of deeper structures, detailed quantitative analysis using the advanced techniques (previous section) could also be performed in almost all cases, when needed.

The standard method of kinematics is to follow objects using the temporal sequence of 2D and 3D data sets. All of these registration techniques utilize 4D datasets (3D surfaces or volumes plus time) that are essentially similar among all imaging techniques. However, due to its intrinsic and "tunable" high tissue contrast and progressively improving spatial and temporal resolution, all registration-based movement modeling approaches are easier to implement for MRI. From these images, the boundaries and edges of target tissues are first identified by tissue segmentation techniques and subsequently tracked using standard object registration methods. As an alternative, volumes could be tracked solely using image intensity information; our study employs one of these techniques.

Using MRI to quantify deformations caused by mock manual therapy

Three-D high-resolution magnetic resonance image sets were analyzed to quantify tissue deformations caused by therapeutic-like loading (e.g., Graston Technique[®]; Hammer 2008) of the human lower leg in vivo.

Methods

Five healthy subjects (male, 27 ± 3 years old, height 175 ± 7 cm and weight 73 ± 8 kg) who participated were positioned prone in a 3 T MRI scanner. The target leg (left) of the subjects was brought to a reference position: the ankle angle was fixed at 90° using an MRI compatible ankle-foot orthosis. In the initial (i.e., *undeformed*) state 3D high-resolution MR image sets were acquired. Subsequently, a rigid cylindrical indenter (diameter = 2.5 cm) was pressed against the posterior lower leg to create comparable surface indentation (8.33 ± 2.07 mm), causing application of forces normal to the mid-gastrocnemius region (Fig. 8.3.1a), predominantly loading its lateral head. A second image set was acquired in the deformed state.

Demons algorithm (Thirion 1998) was used to determine corresponding parts of the images and their displacements. After calculating the strains for each part (size = $0.8 \times 0.8 \times 0.8$ mm) principal strains representing peak local lengthening and shortening (predominantly first and third principal strains, respectively) were determined. It should be noted that the principal strain data are data that have been recalculated (actually the data matrix is rotated), so that shear strains are no longer present. A slice group consisting of 30 consecutive axial slices was selected (Fig. 8.3.1b) for each subject, such that the volume considered included the cylindrical indenter in the middle. Within each slice, five anatomical regions representing muscles/compartments were distinguished by outlining their boundaries: m. gastrocnemius, m. soleus, deep flexors, peroneal and anterior crural compartment (Fig. 8.3.1c). For each anatomical region separately, the principal strains for all subjects were pooled and the mean \pm standard errors (SE) were calculated. The interquartile range (IQR) values for the box & whisker plots are considered as measures of strain heterogeneity within anatomical regions.

Results

Both the first and third principal strains within the m. gastrocnemius targeted directly by therapeutic loading were substantial: the first and third principal strains equaled $44 \pm 8\%$ and $-17 \pm 2\%$ (mean \pm SE), respectively. This indicates that manual therapy may cause very high length changes within soft tissues immediately within the treatment vicinity. Note, however, that the principal strains within deeper m. soleus were also substantial: the first and third principal strains equaled $32 \pm 9\%$ and $-17 \pm 2\%$ (mean \pm SE), respectively. A remarkable finding is that the effects of mock-therapeutic loading remained sizable even for tissues more distant to the location of intervention: (1) within tissues of the deep flexors, local lengthening (mean \pm SE) was $16 \pm 1\%$ and local shortening $-15 \pm 5\%$, (2) within the tissues of the peroneal compartment these values were $17 \pm 6\%$ and $-11 \pm 3\%$, respectively, and (3) within the tissues of the anterior crural compartments $21 \pm 10\%$ and $-10 \pm 4\%$, respectively. Note that local lengthening effects of such loading are profound in directly targeted m. gastrocnemius and drop at least by half at more distant locations, but remain sizable nevertheless. In contrast, the reduction in the local shortening





deformed contours of the call area. Note that deformation of other areas can also be seen. (B) The proximo-distal location of 30 consecutive axial slices studied. (C) The undeformed tissue marked for muscles and compartments: (i) m. gastrocnemius (GM+GL), (ii) m. soleus (SOL), (iii) deep flexor compartment (DF), (iv) peroneal compartment (PER) and (v) anterior crural compartment (ANT). (D) Box & whisker plot of the first principal strains for each anatomical region. Reference data (ref) calculated by imposing rigid body motion quantifies the error of the strain calculation method. Small reference strain values (0.041 \pm 0.006) indicate the method's success. Note that high interquartile range (IQR) values calculated for passive m. gastrocnemius and m. soleus indicate strain heterogeneity in those muscles. The maximum value of first principal strains in each anatomical region is denoted by the top edge of the upper whisker (dashed line above the box). The horizontal line in a box represents the median value.

effect remains rather limited at larger distances throughout the lower leg cross-section.

The present results also show heterogeneity of the first and third principal strains (see Fig. 8.3.1d for a representation of variation of the first principal strains within each anatomical region studied). The IQR values of the principal strains were high not only for the target muscle (0.68 and 0.21 for the first and

third principal strains, respectively) but also for the synergistic m. soleus (0.44 and 0.15 for the first and third principal strains, respectively). This shows that therapeutic loading causes highly heterogeneous length changes for the tissues near the indenter. However, such effects are less heterogeneous for more distant tissues: IQR values for the first and third principal strains, respectively, equaled: (1) 0.17 and 0.14 for deep flexor, (2) 0.19 and 0.17 for peroneal, and (3) 0.18 and 0.08 for anterior crural compartments.

This MRI analysis shows that the method developed allows detailed quantification of deformations caused by manual therapies within both muscular and nonmuscular tissues. This may help to improve the, so far fairly limited, understanding of acute effects of such treatment and could lead to more detailed consideration of its mechanisms. It is concluded that therapeutic loading causes sizable length changes not only within tissues in the immediate vicinity of the location of loading but throughout the cross section. This also makes deformation at other levels within the entire lower limb likely and this may need special attention: (1) the therapist may have a higher control over the outcome by realizing where actions may also cause mechanical effects. (2) Such widespread effects also suggest the importance of epimuscular myofascial force transmission (see Chapters 5.4 and 5.5 for a discussion) in the mechanical effects of manual therapies. which needs to be assessed in new studies.

Advanced motion imaging tools of MRI

MRI is an imaging modality by which, with some controlled changes in molecular magnetic spin properties (via precise timing of sequential steps of appropriate application of radiofrequency excitations and gradients), unique tissue movement imaging can be achieved in vivo. These techniques are: (a) tagged magnetic resonance imaging (TMRI); (b) phase contrast MRI (PCMRI); (c) pulse field gradient based MRI methods (HARP and DENSE); (d) strain encoding imaging (SENC). This is an active research area in basic MRI research, and we will introduce each technique briefly below.

Conceptually, the simplest imaging technique for analysis of tissue movement is TMRI, where temporary magnetic fiducial markers, or *tags*, are created within tissues, and when imaged after a certain time, the shape changes of these tags reflect the underlying tissue motion. The parallel plane stripe pattern and the combination of two orthogonal plane tags forming a grid are the most common tags (Axel & Dougherty 1989) (Fig. 8.3.2).

PCMRI is another approach to tissue movement analysis based on the sensitivity of the phase of the MR signal to motion. It was used mainly for blood flow measurements, but stronger gradients can be used to obtain tissue velocity per voxel (and subsequently local strain-rate and strain) measurements of muscles in segmented acquisitions, such as for the myocardium. The basic principle is to acquire two datasets with two different velocity-encoding gradients, but otherwise identical acquisition parameters, and to subtract the two phase images. The resulting difference image will be proportional



Fig. 8.3.2 • Advanced motion imaging using TMRI and PCMRI. Normal cardiac cine MR image examples before and after the heart contraction (A, B). Same images when tags are laid just before contraction showing internal muscular patterns (C, D). Cardiac PCMRI cross-sectional images at mid contraction, where intensity on phase images is proportional to the tissue velocity in the horizontal (E), vertical (F), through-plane (G) directions and the corresponding anatomic image (H). PCMRI images are courtesy of Richard Thompson, NHLBI, NIH.

to the flow (or tissue movement) if the fluid (or underlying tissue) can be assumed to have a constant velocity during the acquisition window. The velocity field for a given image or space at different temporal snapshots can then be integrated to yield tissue displacement (Zhu & Pelc 1999) (Fig. 8.3.2).

In DENSE and HARP, a uniform pattern of phase modulation is encoded into the tissue at a chosen time, and the deformation of that pattern is detected at a later time and utilized to estimate the motion (Aletras et al. 1999, Osman et al. 2000). In SENC, a pattern similar to tagging is encoded into the tissue, and the strain in through-plane direction is directly measured by acquiring two images having different z phase encodes (Osman et al. 2001) (Plate 8.3.1).

Currently, dynamic MR imaging of the musculoskeletal system is usually performed in dedicated

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or open MR systems. With advancing imaging technologies, low-field MRI systems now claim specificity and sensitivity that are comparable to high-field systems in identifying musculoskeletal pathologies. These low-field systems also allow a wide range of dynamic studies; these are routinely analyzed only visually or using surface-based motion-tracking techniques.

MRI can do much more than provide detailed anatomic images, as was shown in our study. Advanced MRI techniques have the ability to yield even more direct quantitative tissue movement data. These are unique to MRI and are in sharp contrast with movement analysis, which is derived from surface or volume matching techniques. We believe in the future there will be more and more dynamic musculoskeletal studies and significantly more utilization of the advanced techniques that are unique to MRI.

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Roles of fascia in molecular biology of adaptation of muscle size

8.4

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Introduction

Skeletal muscle is able to adapt its properties in response to changes in functional demands because of its ability to activate and deactivate molecular systems for protein synthesis and degradation. Adequate functioning of these systems is particularly important in cases of muscle injury, neurological disorders, or chronic diseases associated with loss of muscle mass. With disuse, atrophy is a common adaptation limiting the capability to generate force. Optimizing intervention effects aimed at redressing this requires knowledge of the underlying mechanisms.

Mechanical loading of muscle tissue is a critical stimulus for adaptation of myofiber size. Knowledge of how mechanical loading of the muscle-tendon complex affects the mechanical and molecular environments of myofibers is essential for optimizing the therapeutic treatment of patients with complaints in the muscle-fascia unit. This chapter considers how mechanical loading of this complex activates molecular processes and how fasciae play a role in muscle regeneration and adaptation of muscle size.

Mechanical loading-induced muscle adaptation in vivo

The active force a muscle is able to exert at different lengths depends on the number of sarcomeres arranged in series (within myofibers) and in parallel (within the muscle). The more sarcomeres in series (i.e., longer optimum fiber length), the higher is the length range of active force exertion. However, this has no effect on the maximum force a myofiber can exert (at its optimum length). Therefore, optimal muscle force is determined by both the number of the myofibers and the number and size of myofibrils arranged in parallel within myofibers. The muscle cross-sectional area (A_f) perpendicular to the fiber direction at a standardized mean sarcomere length (e.g., optimum length) provides an estimate of the maximum force a muscle is able to exert.

Both prime parameters, $A_{\rm f}$ and serial sarcomere number, are highly adaptable in response to changes in mechanical loading of muscle. In myology, for adaptation of $A_{\rm f}$ the terms atrophy and hypertrophy are used specifically, whereas adaptation of the serial sarcomere number refers to changes along the myofiber.

How does mechanical loading stimulate adaptation of muscle size? Several in-vivo experiments indicate that mechanical loading-induced adaptation of muscle size is determined by both type and intensity of active contractile activity, as well as the strain applied onto the muscle.

Training

Training studies have shown that high intensity training, particularly consisting of eccentric contractions, most strongly stimulates muscle hypertrophy (Farthing & Chilibeck 2003). In contrast, disuse of muscle as occurs in low gravity conditions or limb suspension causes progressive and severe atrophy (Huijing & Jaspers 2005). Effects of these types of muscle overload and disuse on muscle remain unreported.

Muscle strain

Muscles also adapt their size in response to the length at which they are maintained. Experiments in which rodent muscles were immobilized at high lengths. for periods varying from several days to 4 weeks, have shown a 20% hypertrophy and a 15% increase in the number of sarcomeres in series (Williams & Goldspink 1978). This was arranged in such a way that the optimum length of the adapted muscle was attained at the immobilized position. Opposite effects have been reported for muscles which were immobilized in a maximally shortened position: yielding 30–40% atrophy as well as a similar reduction in serial sarcomere numbers (particularly low degree pennate muscle) (Williams & Goldspink 1978; Heslinga & Huijing 1993). Also for this condition, optimal force was reduced substantially and optimum length was attained at the immobilized position (Williams & Goldspink 1978; Heslinga et al. 1995).

From these results, a simple rule has been derived stating that for any muscle adaptation of A_f and serial sarcomere number is regulated in such a way that muscle optimum length is attained at the joint angle at which a muscle is most frequently active (Herring et al. 1984). Although the simple rule seems to be valid for several types of muscles and species, controversies and exceptions do exist. It has been suggested that, for some muscles, the length ranges of operation in daily activities differ from those predicted by the simple rule that muscles are operating around their optimum length (Burkholder & Lieber 2001).

Other evidence suggesting that high actual myofiber strain per se does not stimulate hypertrophy and increase of serial sarcomere number is derived from ex-vivo cultures of mature myofibers (see below). This indicates the need for more detailed understanding of mechanisms via which mechanical loading affects the rate of protein synthesis and degradation.

Molecular mechanisms of adaptation of muscle size

The quantity of proteins constituting force generating or passive elements within a myofiber is the net result of ongoing processes of simultaneous protein synthesis and degradation, both being modulated in response to mechanical loading. Protein synthesis involves three types of processes occurring in subsequent order.

Machinery for protein synthesis

Transcription of the deoxyribonucleic acid (DNA) of the genome

The genetic code itself resides within DNA strings located within the nuclei of the myofiber, but DNA is never applied directly in the synthesis of proteins. First, DNA is "transcribed" (copied) into messenger ribonucleic acid (mRNA) that carries "the recipe" from the DNA. This process is referred to as "transcription". The rate of protein synthesis depends on the quantity of DNA available, as well as on the rate of transcription. Most cells have only one nucleus, which means that the quantity of DNA cannot be regulated. Myofibers contain a large pool of nuclei. In adaptation, this pool may be enlarged or reduced by proliferation satellite cells (i.e., muscle stem cells resident between the sarcolemma and the basal lamina) which donate additional nuclei to the myofiber or remove nuclei (i.e., DNA degradation), respectively.

The rate of transcription is determined by the absence or presence of transcription factors that modify DNA molecules such that transcription of genes is facilitated or inhibited.

Translation of mRNA

Within the cytoplasm, mRNA binds ribosomes; this complex is used to "translate" mRNA information into a specific sequence of amino acids that constitutes the corresponding protein. The rate of protein synthesis depends also on the rate of translation. This rate is determined by the number of ribosomes per mRNA molecule, as well as by the rate of translation per unit mRNA.

Completion of the protein synthesis

The chain of amino acids undergoes post-translational modifications, which ultimately results in the mature protein.

The critical factor determining the overall rate of protein synthesis is the one that is limiting the whole chain of processes.
Machinery for protein degradation

Protein degradation is regulated by activity of proteolytic enzymes and by expression of cofactors leading to altered expression of these enzymes and their activation. The most important myofiber proteolytic system is the proteasome. Proteins to be degraded are bound to multiple ubiquitin molecules, which marks them for degradation by the proteasome complex (Jackman & Kandarian 2004).

Mechanochemical signaling and mechanotransduction for protein synthesis and degradation in muscle

Given balanced protein synthesis and degradation, hypertrophy and addition of serial sarcomeres requires either raising protein synthesis rates, or inhibiting degradation rates, or both. This means that mechanical loading triggers intracellular signaling pathways affecting the processes mentioned. To accomplish this, the mechanical load must be sensed and transmitted to the machineries for protein synthesis and degradation.

Myofibers are equipped with sensors via which protein synthesis and degradation are directly and/or indirectly affected (Fig. 8.4.1). Several types of mechanosensor are known: (1) stretch activated calcium channels within the sarcolemma that open as myofibers are stretched, allowing the influx of calcium into the cytoplasm. Other transmembrane-receptors such as (2) the integrin and (3) dystroglycan complexes connect the intracellular cytoskeleton to the extracellular matrix (ECM) that is reinforced by collagen structures within the basal lamina and endomysium. Mechanical loading of such receptors and channels activates enzymes associated with sarcolemma that subsequently elicit cascades of chemical reactions. This mechanochemical signal transduction stimulates muscle gene expression and/or rates of translation (Huijing & Jaspers 2005). Signaling via these receptors and channels is associated also with increased expression of growth factors that are secreted into the extracellular matrix. These factors act on myofibers in which they are produced (autocrine signaling) or on neighbouring myofibers and other cells (paracrine signaling). Among many growth factors expressed in muscle, the following play important roles in adaptation of muscle size: (1) insulin-like growth factor 1

(IGF-1), (2) mechano-growth factor (MGF), (3) basic fibroblast growth factors (bFGF), (4) hepatocyte growth factor (HGF), and (5) myostatin. On overloading muscle in vivo, mRNA expression of these growth factors is increased (Huijing & Jaspers 2005), with the notable exception of myostatin (reduced expression; Heinemeier et al. 2007). Most of these growth factors also play a role in activating satellite cells. Satellite cells are normally quiescent, but exposure to MGF, FGF or HGF initiates proliferation by cell division (Huijing & Jaspers 2005). In contrast, myostatin inhibits satellite cell proliferation (Huijing & Jaspers 2005). Activation of satellite cells is important for two reasons: (1) for repair of overload-induced myofiber damage, activation of satellite cells will provide new nuclei to replace degraded ones within the damaged myofiber parts; and (2) the number of nuclei within a myofiber may be the factor limiting the maximal capacity for mRNA transcription (Huijing & Jaspers 2005). However, some hypertrophy and addition of sarcomeres due to enhanced transcription and/or translation is possible without adding new nuclei (Petrella et al. 2008).

Myostatin and IGF-1 have multiple functions in regulating muscle protein synthesis and degradation. In addition to its role in satellite cells activation, IGF-1 stimulates transcription of muscle mRNA and its translation into protein (Glass 2005; Jaspers et al. 2008a). In addition, IGF-1 reduces expression of the ubiquitin ligases (Glass 2005), inhibiting protein degradation rate. Whereas IGF-1 has strong anabolic effects (protein synthesis), myostatin is its antagonist because of its opposite effects on synthesis and degradation (McFarlane et al. 2006).

An alternative way of mechanical loading of muscle yielding an increase in muscle size is by direct transmission of mechanical loading of transmembrane complexes and transmission via the intracellular cytoskeleton onto myonuclei. Such mechanotransduction will have two effects: (1) release of mRNA and ribosomes attaching to the cytoskeleton and their translocation to the sites where protein synthesis is required (Chicurel et al. 1998) and (2) nuclear deformation or conformational changes of chromatin within myonuclei that may directly affect transcriptional activity (Bloom et al. 1996). Mechanotransduction to the nucleus works almost instantly. However, its relative contribution to protein synthesis and degradation rates compared to mechanochemical pathways is unknown and warrants further investigation,



Fig. 8.4.1 • Schematic representation of the mechanochemical signaling pathways involved in the regulation of myofiber size. Two adjacent myofibers are shown, which are surrounded by their sarcolemma (SL) and a basal lamina (BL). The muscle fibers are anchored to the collagen fiber reinforced extracellular matrix by the integrin and dystroglycan/sarcoglycan complexes. Mechanical loading is sensed at these complexes at the myotendinous junctions of the myofibers as well as along the length of the myofiber. Loading of these complexes elicits cascades of signaling molecules (i.e., free radicals and enzymes). Another mechanosensor is the stretch activated calcium (Ca^{2+}) channel, which allows calcium influx into sarcoplasm. In addition to its role in the contraction, calcium also regulates the activity of particular transcription factors. Transcription factors may alter deoxyribonucleic acid in the myonucleus (MN) and affect the synthesis of muscular proteins directly or indirectly via expression of growth factors, which are released in the interstitial space (i.e., extracellular matrix). Several growth factors have been identified to be expressed within muscle, which likely play a major role in muscle adaptation. Among these are the insulin-like growth factor 1 (IGF-1), mechanogrowth factor (MGF), fibroblast growth factor (FGF), hepatocyte growth factor (HGF) and myostatin. Once released, these growth factors can bind to receptors in the membrane of the producing myofiber and stimulate signaling pathways that modulate the rate of transcription of DNA as well as the rate at which the ribosomal RNA (constituting the body of the ribosome) translates the mRNA into protein. The growth factors may bind to receptors in the membrane of the fiber that releases these factors as well as of its host satellite cells (SC), neighboring myofibers and/ or other cells such as fibroblasts and adipocytes.

The roles of fascia in the regulation of myofiber size

The continuous fascial networks within and around muscles constitute the structures mediating myofascial force transmission (Huijing 2003). In addition to the networks' function in force transmission, they are likely to play a role also in adaptation of muscle size in a mechanical as well as biochemical fashion.

Fascia accommodate many different cell types (e.g. fibroblasts, myofibroblasts, adipocytes, endothelial cells, macrophages and neuronal branches), likely playing roles in muscle adaptation. Viscoelastic characteristics of the environment of stem cells are a determining factor for differentiation into different types of cells and their mechanical characteristics (Engler et al. 2006). Therefore, it is expected that the mechanical properties of fascia in and around muscles affect properties of adult myofibers and other cell types by modulating expression of growth factors and cytokines and their paracrine effects on myofibers.

Apart from control of chemical factors by fasciae, substantial effects on muscle adaptation are expected due to epimuscular myofascial force transmission. Epimuscular connections allow force transmission between a muscle and extramuscular connective tissues, as well as between adjacent muscles (Huijing 2003, and see also Chapter 3.2). Experiments (e.g., Huijing & Baan 2003) and mathematical modeling (e.g., Yucesoy et al. 2002) have shown that epimuscular force transmission affects local deformations within myofibers and active and passive forces generated locally.

A finite-element model of muscle was developed specifically to learn about the local effects of epimuscular force transmission within myofibers (i.e., sarcomere strains and stresses). Modeling of extramuscular connections (e.g., the neurovascular tract), in active as well as passive muscle maintained at extended length $(\sim 12\%$ over optimum muscle length), indicates large sarcomere length distributions (1-30% of optimum sarcomere length) occurring within myofibers (i.e., serial distribution) and between them, at different locations within the muscle (i.e., parallel distribution, Plate. 8.4.1). This means that a given global strain applied to myofibers is strongly amplified or attenuated locally. Accordingly, it is likely that stimuli for mechano(-chemical) signaling and related atrophy/ hypertrophy or adaptation of serial sarcomere number are also local, instead of global, events. Therefore, fascia and epimuscular force transmission need to be considered as potentially important factors for regulating muscle adaptation.

Ex-vivo culture of mature, single myofibers

Testing of hypotheses regarding myofascial connection-induced adaptation of muscle size requires removing intramuscular and epimuscular connections. Ex-vivo culture of single mature myofibers with intact basal lamina and endomysium (Jaspers et al. 2004) allows study of the independent effects of mechanical connections (Plate 8.4.2A). Two weeks' culture of mature myofibers at both high and at low length caused dissimilar effects than those reported after in-vivo immobilization of muscles (see above). This involved myofiber cross-sectional area and serial sarcomere number, as well as expression of IGF-1. In contrast to those in-vivo results, these parameters remained unchanged during culture (Jaspers et al. 2004, 2008a). One may argue that ex-vivo cultured myofibers may have lost their capability for protein synthesis and adaptation. However, another set of experiments revealed that such preparations show hypertrophy in culture medium supplemented with insulin or IGF-1 (Jaspers et al. 2008a, b). This indicates that the cultured single myofibers still had the ability to adapt. Why does a single myofiber cultured at a high global fiber strain not respond to such mechanical loading? Is it removal of intra- and epimuscular myofascial force transmission? Assessing serial sarcomere length distributions, every millimeter along the myofiber length kept at a mean $\sim 12\%$ over passive sarcomere slack length showed that serial strain distributions are still present with the single myofiber, but were limited between 9 and 15% over passive sarcomere slack length. This indicates that strain distributions found by finite-element modeling for muscle with epimuscular connections do not occur within a single myofiber. Therefore, it has been hypothesized that epimuscular connections are crucial for mechano-(chemical) signal transduction in skeletal muscle (Huijing & Jaspers 2005), underlining the importance of fascia in the regulation of adaptation of muscle size. Obviously, this hypothesis is in need of further scientific confirmation.

Summary

Mechanical loading has been shown to be a stimulus for adaptation of muscle size. Since mechanical coupling between the myofiber and inter- and extramuscular connective tissue is present, it is hypothesized that fasciae play a role in the regulation of adaptation of muscle size. Variations in local stiffness of the epimuscular fascia are likely to cause local stresses onto the myofiber, different from the global stress that is accompanied by high strains locally. Such local mechanical effects elicit local biochemical signaling within myofibers affecting rates of muscle protein synthesis and degradation. These effects may be direct (affecting nuclei) or indirect via enhanced expression of growth factors and cytokines being released into the extracellular matrix and having autocrine and/or paracrine effects there.

In sum, mechanical interaction between epimuscular fascia and myofibers seems to be crucial for the regulation of adaptation of muscle size. This new view on the interaction and communication between muscle and fascia needs to be investigated further in order to make effective use of such mechanisms in training and therapy.

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Mathematical modeling

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Introduction

Quantification is central to scientific understanding of phenomena, and performing experiments is clearly the fundamental approach in doing that. By ensuring that only one factor is varied at a time and all others remain unchanged or stable, one can quantify the effects of the manipulated factor on the matter experimented.

In-vitro experiments

In experiments in vitro, aimed at determining the mechanical properties of tissue, specimens prepared as much as possible to standard dimensions are used. Although, such tests successfully characterize tissues as a material, the mechanics of the actual donor structure will remain disregarded. In-vitro testing of the whole structure experimentally might, at the least, be expensive, and sometimes even impossible.

In-situ experiments

On the other hand, in experiments in situ, aiming at determining physiological properties of tissue, care is taken to keep the target tissue as intact as possible. Even though valuable information characterizing tissue function can be gained, it is often of interest also to determine the mechanisms producing such function in health so that, for example, insight about etiology of diseases can be obtained. Doing that experimentally requires simultaneous measurements of additional parameters and has sizable limitations:

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each new measurement means further interference with the target tissue and the higher level of its organizational context, detracting from the main aim of keeping the structure intact. In addition, the resolution of the data collected may be marginal.

Mathematical models

If built according to a specific goal, mathematical models can immensely enhance insights gained experimentally and actually guide subsequent experimentation: tissue structures and the effects of diverse parameters can be studied inexpensively. Moreover, hypotheses generated with experiments can be tested and new hypotheses to be tested experimentally can be generated. In some cases, even conditions impossible to create experimentally or parameter values not measurable experimentally can be addressed (see below) and major principles concerning mechanisms producing tissue function can be isolated

Despite the existence of numerous reports of experimental studies on the mechanical behavior of various fasciae (Iatridis et al. 2003; Zeng et al. 2003), specific mathematical models proposed for fascia are rare, conceivably due to complex geometry and material properties. Taking a finite deformation approach, analytical models of human fasciae were developed recently in order to quantify deformations occurring in manual therapy (Chaudhry et al. 2008). In such models, the tissue is considered to be continuously distributed in space (i.e., as a continuum). This disregards the very fine structure and benefits from a systematic treatment of forces and deformations that continuum mechanics offers. Using analytical models, nonlinear material properties and large deformations can be handled; and solutions of the equilibrium equations are possible, but typically within a well defined material domain only. Therefore, complex geometries and mechanical interactions between adjacent structures cannot be addressed. Because fascial structures are architecturally highly complex and are continuous with other tissues (e.g., muscle, bone) this should be considered as a sizable limitation.

In contrast, with at least similar capabilities, the finite element method has the great advantage that geometrically highly complex structures can be modeled. This involves a general discretization procedure of continuum problems posed by mathematical statements. In this context, "discrete" represents constructing an adequate model using a finite number of well defined components (elements), the behavior of which is well understood, whereas "continuous" represents indefinite subdivisions implying differential equations and boundary conditions that characterize the mechanical equilibrium mathematically. The general procedure includes: (1) determination of the geometry of the system, (2) subdivision of the volume into a finite number of elements, (3) solution of the equilibrium equations for each element, and (4) assembly of the element solutions to obtain the solution for the complete system.

Modeling fascia and muscle tissue using the finite element method

The value of the finite element model may be substantial, provided that it is developed with a specific and well defined purpose. Such well established modeling goals determine: (1) the extent to which the actual problem can be simplified; (2) the modeling assumptions to be made; and (3) the relevant model output parameters and their interpretation. Recently, this approach has been applied successfully to modeling plantar fascia with a specific emphasis on surgical treatment of plantar fasciitis. Gefen (2002) developed a model for analysis of structural characteristics of the human foot during standing in order to investigate the biomechanical effects of surgical release of the plantar fascia. His results showed that a total fascia release can cause an extensive arc deformation compromising the foot's load-bearing ability. With similar intentions, Cheung et al. (2006) constructed a finite element

model of the ankle–foot complex aiming at quantifying the effects of different plantar fascia stiffness on foot geometry, with zero stiffness representing a fascia release. With decreasing fascia stiffness, their results also indicate a decreased foot arc height, as well as midfoot pronation. These studies indicate that the extent of plantar fascia release must be planned carefully and that models may provide a basis for such planning.

Finite element models developed to study the biomechanics of other fascial structures include, for example, anterior vaginal wall (Chen et al. 2008) and inguinal transversalis fascia (Fortuny et al. 2009). Also, muscle mechanics has been studied using finite element modeling (Gielen 1998; van der Linden 1998; Blemker et al. 2005). Despite the diversity of their modeling goals, the authors regarded muscle implicitly as a tissue that can be active and thereby change its properties. However, they did not model the muscle as operating within the context of a fascial integrity: (1) elements were used in which both active and passive properties of muscle tissue are lumped together, hence, the role of intramuscular connective tissues and their interaction with the contractile apparatus were not accounted for explicitly; (2) muscle was considered as an isolated entity, hence the continuity of intramuscular fascia (e.g., epimysium, perimysium, endomysium) and epimuscular fascia such as collagen reinforced neurovascular tracts or compartmental boundaries were not accounted for.

In contrast, the linked fiber-matrix mesh (LFMM) model developed by the authors during previous appointments at the University of Twente (Yucesoy et al. 2002) was designed specifically to study muscular mechanics within the context of fascial integrity. The mechanical roles played by such muscle-related fascia have been referred to as intra- and epimuscular myofascial force transmission (Huijing 1999; Yucesoy et al. 2005). Several publications focusing on the major effects of such force transmission (Maas et al. 2001; Yucesoy et al. 2003a; Meijer et al. 2007; Smeulders & Kreulen 2007; Yucesoy & Huijing 2007) typically report differences in muscle forces exerted by muscle at its origin and insertion, as well as the length range of muscle active force exertion being dependent on the actual mechanical conditions imposed (e.g., muscular relative positions).

The following concepts characterizing important phenomena also determined the modeling approach of the LFMM model:

1. Multi-molecular connections between the myofiber (i.e., muscle fiber) and the extracellular matrix (ECM) found along the full periphery

(Berthier & Blaineau 1997) of myofibers are capable of transmitting force (Street 1983; Huijing et al. 1998; Yucesoy et al. 2002). Therefore, the force balance determining the length of a sarcomere is much more complex than just the interaction between two sarcomeres arranged in series within the same myofiber; it also includes intramuscular myofascial loads: forces exerted by (i) the ECM and (ii) sarcomeres located in neighboring myofibers.

2. In vivo, muscle is not an isolated entity: direct intermuscular connections, i.e., collagenous linkages between epimysia of adjacent muscles, as well as indirect connection between muscles (i.e., via extramuscular tissues) form an integral fascial system capable of transmitting force (Huijing 2009; Yucesoy et al. 2010). Therefore, extramuscular myofascial loads acting on the muscle also take part in the balance of forces and hence in determining the length of a sarcomere.

Description of the model for isolated muscle: modeling to account for intramuscular myofascial loads

Skeletal muscle is considered explicitly as two separate domains: (1) the intracellular domain and (2) the ECM domain. The trans-sarcolemmal attachments are considered as elastic links between the two domains.

Two new elements were introduced into a finite element program: (1) the ECM element represents

the collagen reinforced ECM, including the basal lamina and connective tissue components such as endomysium, perimysium and epimysium; (2) the muscle fiber element representing myofibers.

Each muscle element combining these two elements represents a segment of a bundle of myofibers (fascicle), as well as its connective tissues and the links between muscle and connective tissue elements (Fig. 8.5.1a).

The geometry of the LFMM model (Fig. 8.5.1b) was defined as the contour of a longitudinal slice at the middle of the belly of an isolated rat EDL muscle, but can be made to fit any muscle architecture. The ECM domain is represented by a mesh of ECM elements (matrix mesh). In the same space, a separate mesh of muscle fiber elements is built to represent the intracellular domain (fiber mesh). The two meshes are rigidly connected to single layers of elements representing proximal and distal aponeuroses or tendon plates; a node representing myotendinous connection sites is the common node of ECM, myofiber and aponeurosis elements. In contrast, at the intermediate nodes, fiber and matrix meshes are linked elastically to represent the trans-sarcolemmal attachments of the (intracellular) cytoskeleton and ECM. A standard two-node uniaxial spring element models these links: their linear, high stiffness characteristics represent nonpathological connections between the myofibers and the ECM. Note that at the initial muscle length and in passive condition, these links have a length equaling zero.

Fig. 8.5.1 • Finite element modeling of skeletal muscle: LFMM model. (A) 2-D schematic representation of an arrangement of muscle elements. The intracellular domain composed of the active contractile elements (A) and intracellular passive cytoskeleton (T), is linked to the ECM domain (M) elastically. (B) The model of muscle with extramuscular connections consists of muscle elements (three in series and six in parallel) and aponeurosis elements. The 3-D local coordinate system used for the analysis is shown. The nodes of the matrix mesh marked by a white "+" sign have extramuscular connections to mechanical ground and the nodes marked also by a black square have stiffer connections.



Being 3-D structures, both ECM and muscle fiber elements have eight nodes. At the nodes, stress (force per unit area) and strain (normalized measure of deformation) are calculated on the basis of mechanical equilibrium. Between nodes, linear interpolation is performed to obtain such values. A 3-D local coordinate system representing the myofiber direction, cross-myofiber direction (perpendicular to the myofiber direction), as well as its thickness is used. The ECM element incorporates a strain energy density function that accounts for the nonlinear and direction dependent material properties, as well as the constancy of muscle tissue volume during length changes and contraction. For the muscle fiber element, the total stress acting exclusively in the local fiber direction is the sum of the active stress of active sarcomeres and the stress due to intracellular passive tension.

A standard eight-node element models aponeuroses.

Muscle operating within the context of fascial integrity: modeling effects of intra- and epimuscular myofascial loads, simultaneously

For this purpose, the LFMM model was extended to take into account the epimuscular connections:

- 1. *Extramuscular connections*. For the model, a set of fixed points represents bone, which is assumed to be rigid. For each muscle fascicle, nodes of the matrix mesh located at one-third of the fascicle length from its proximal end were linked to the modeled bone (Fig. 8.5.1b). This represents all extramuscular connections. The higher stiffness of the tissues constituting the extramuscular neurovascular tract (i.e., the collagen reinforcement of blood vessels and nerves) close to the muscle is taken into account by making the extramuscular links to the muscle matrix mesh stiffer for the three most proximal fascicles than for more distal ones (Fig. 8.5.1b).
- **2.** *Direct intermuscular connections*. The corresponding nodes of the matrix meshes of two muscle models were linked elastically.

Note that: (1) all epimuscular connections were modeled using standard uniaxial spring elements having linear length–force characteristics. (2) Initially (at a specified muscle length, and before changing any of the tendon positions), these links were set to have zero lengths.

Contribution of LFMM model to muscle mechanics

This model allows (1) quantifying muscle forces exerted at the proximal and distal tendons, for which agreement with experimental data is achieved by using suitable stiffness values selected for epimuscular links (Yucesov et al. 2003a, b); and even more importantly, (2) studying factors that cannot experimentally be observed: myofiber strain and stress distributions in the fiber direction. Note that such fiber strains quantify sarcomere length distributions both serial (within the same myofibers) and parallel (in different parts of the muscle), whereas fiber stresses indicate the local capability of force exertion of the myofibers. Assessing such model parameters in association with muscle length and force characteristics provides explanations for the mechanisms causing proximodistal force differences (Yucesoy et al. 2006) and those causing the dependency of muscle length-force characteristics on mechanical conditions. For example, increased sarcomere length heterogeneity (both serial and parallel) due to epimuscular myofascial loads causes a shift in muscle optimum length to a higher length (Willems & Huijing 1994). Therefore, in agreement with the modeling goals, the LFMM model has provided an important contribution to our understanding of the mechanisms of the key effects of epimuscular myofascial force transmission. Offering new insights for muscle mechanics, this has important implications regarding muscle function in health and disease, as well as treatment of pathologies.

A similar approach is expected to be relevant also in quantifying and explaining the acute mechanical effects of manual therapies and providing improved understanding of the mechanisms involved: therapeutic loading typically used in such treatments is expected to interact with the system of epimuscular myofascial loads.

Modeling of deformations caused by manual therapies

Approach

The LFMM model with extramuscular connections was used to initiate study of some basic principles of the acute mechanical effects of therapeutic loading of muscular tissue representative of manual therapies (e.g., Graston Technique®; Hammer 2008). For the active muscle set at a specified length and





position, an arbitrarily selected node, located approximately at the distal third of proximal aponeurosis (Fig. 8.5.2a, at arrow) was displaced downward (by approximately 1 mm). The muscle was studied at an intermediate length and at a position distal to its nonmuscular neighboring structures. The strain distributions in the local fiber and cross-fiber directions calculated for active muscle after such loading (a condition referred to as with manual therapy) were compared to those obtained with no added vertical displacement of the proximal aponeurosis (a condition referred to as with no manual therapy).

Deformations caused by such loading

Peak local shortening equaling 25% (indicated by MN in Fig. 8.5.2b and c), occurring at the proximal ends of myofibers located within the proximal part of the muscle, was not affected by the "manual therapy". In contrast, for all the remainder of the muscle, the effects of therapeutic loading were substantial in the local fiber direction: (1) peak local lengthening with manual therapy (equaling 30%) was about 10 times as high as calculated for this muscle part without it; (2) a pronounced heterogeneity of positive strains (indicating sarcomere lengthening) was found, such that towards the distal ends of myofibers stretching increased dramatically.

In fact, strain patterns for the fiber direction for this part of the muscle are completely changed.

In contrast, for the cross-fiber direction, the effects of therapeutic loading remained more local (in the middle of the belly; see Fig. 8.5.2d and e). However, such local deformations were enhanced substantially: (1) for the muscle with no manual therapy, no length change was found compared to its initial condition (i.e., strain equaled 0%). In contrast, therapeutic loading caused local shortening of up to 7% within numerous fascicles located proximally within the muscle; (2) peak local stretching occurred in the middle of intermediate fascicles (equaling 10% without therapeutic loading) and was increased by 50% with manual therapy.

This preliminary analysis shows above all that finite element modeling can be a powerful tool in quantifying the acute mechanical effects. This may help to improve the, so far fairly limited, understanding of the principal mechanisms of such treatment within a muscle. The results presented show that therapeutic loading does cause much higher strains locally – i.e., either amplified mechanical signals that could facilitate weakening of overly stiff connections by overstretching, or tissue remodeling on a longer time scale.

Additional modeling showed that the effects of the same amplitude of vertical displacement applied at a higher muscle length are much less pronounced, particularly for the fiber direction, but also for the cross-fiber direction. This suggests that the effects of manual therapies are not only muscle length dependent but they are also a function of muscle relative position. Hence it is concluded that an important determinant of the acute mechanical effects of therapeutic loading is related to epimuscular myofascial force transmission. Note that for assessing the effects of manual therapies in vivo with high resolution, magnetic resonance imaging can be a potentially powerful tool. The reader is referred to Chapter 8.3 for a discussion of this approach and the results of some preliminary analysis.

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