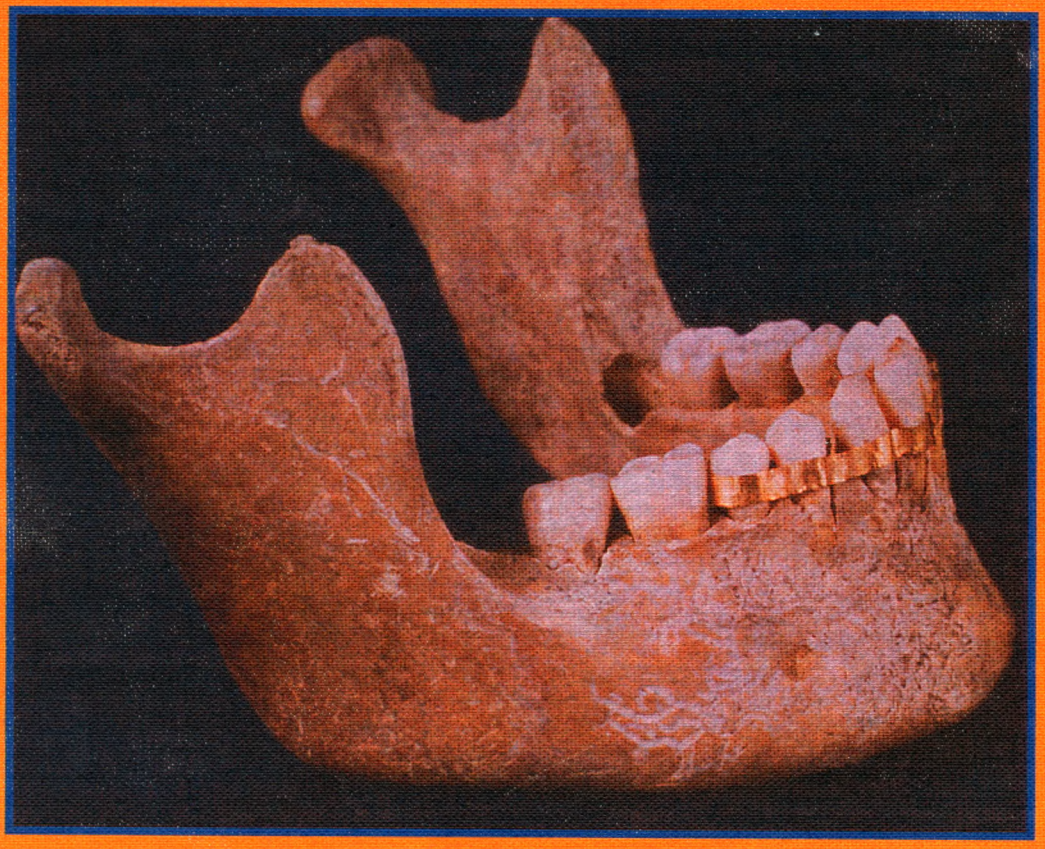
How Anthropology Informs the Orthodontic Diagnosis of Malocclusion's Causes

by Robert S. Corruccini



How Anthropology Informs the Orthodontic Diagnosis of Malocclusion's Causes

Robert S. Corruccini

Mellen Studies in Anthropology
Volume 1

The Edwin Mellen Press
Lewiston•Queenston•Lampeter

Library of Congress Cataloging-in-Publication Data

Corruccini, Robert S.
How anthropology informs the orthodontic diagnosis of malocclusion's causes / Robert S. Corruccini.
p. cm. -- (Mellen studies in anthropology ; v. 1)
Includes bibliographical references and index.
ISBN 0-7734-7980-5
1. Teeth--Abnormalities--Genetic aspects. 2. Jaws--Abnormalities--Genetic aspects. 3. Malocclusion--Genetic aspects.
4. Orthodontics, Corrective. I. Series.
RK523.C67 1999
617.5' 22--dc21 99-24675
CIP

This is volume 1 in the continuing series
Mellen Studies in Anthropology
Volume 1 ISBN 0-7734-7980-5
MSA Series ISBN 0-7734-7918-X

A CIP catalog record for this book is available from the British Library.

The Edwin Mellen Press
Box 450
Lewiston, New York
USA 14092-0450

The Edwin Mellen
Box 67
Queenston, Onta
CANADA L0S 1

The Edwin Mellen Press, Ltd.
Lampeter, Ceredigion, Wales
UNITED KINGDOM SA48 8LT

Printed in the United States of America

Table of Contents

List of Illustrations

implications of functional disorders have been recognized in the medical profession of orthopedics for a long time and, whatever mechanical and surgical procedures are employed, muscle exercises and training are constant parts of treatment. Noting the excellent results thus achieved in the treatment of skeletal deformities of children, there is every reason to follow the example set by the orthopedists.

The studies of Corruccini are a major focus of research challenging the orthodontists to participate in searching for possible environmental factors causing the prevalence of dentofacial deformities in industrial societies. The present book is therefore of high clinical importance which may establish an etiologic framework for improving the diagnosis and treatment of orofacial maldevelopment and, hence, develop our discipline toward a subspeciality of orthopedics, toward a real branch of medicine.

Rolf Fränkel

Acknowledgment

The research recounted in this book was directly supported by the National Science Foundation (grant BNS 8119875 as well as a NATO Fellowship) and indirectly or jointly supported by numerous other grants and fellowships. I am indebted to my many co-authors, colleagues and friends that have collaborated in so many ways on this research, as indicated in the Literature Cited; I shall not mention them individually as they have shared in authorship of the scholarly articles reviewed here.

Regarding the Etruscan studies, we thank the Director of the National Museum of Anthropology and Ethnology, Prof. P. Messeri, the Archeological Superintendent for Tuscany, Prof. F. Nicosia, and the Director of the National Archeological Museum of Florence, Dr. A. Maggiani, for having put the materials at our disposal for study.

Regarding Australian aboriginal studies, Graeme Pretty (South Australian Museum) facilitated the study of skeletal specimens. The Yuendumu longitudinal study was supported by the National Health and Medical Research Council, Canberra and grant DE 02034 from the National Institute of Dental Research. Burton Dawes volunteered invaluable advice and insight.

The Indian twin study was supported by the Smithsonian Institution (SFCP 301999) and the U.S. twin studies by NIDR awards to R.H.Y. Potter and L. Green.

The Office of Research Development and Administration of Southern Illinois University provided graduate research assistant support and other support for various of these projects.

So many individuals and colleagues have provided encouragement, advice, and hospitality over the years. Aside from my co-authors, I thank Denis Goose, J.C. Sharma, H.S. Gill, Eduardo Pardini, Brunetto Chiarelli, Chiara Bullo, Roscoe Stanyon, Ed Harris, Ed Hunt, C. Loring Brace, Robert Mucci, Don Ortner, Clark Larsen, Howard Bailit, Ingrid Wustmann, Alan Harn, Owen Lovejoy, Robert Mensforth, Erik Trinkaus, Richard Thorington, Bill Proffit, Bill

Hylander, Ken Wehrenberger and the Pacific Coast Orthodontic Study Consultation Group, Richard Smith, Ken Kennedy, John Lukacs, Luca Bondioli, Jack Kelso, Debbie Steagall, Steve Ward, Francine Berkowitz, Pritmohinder Gill, Linda Gerber, Mari Bouvier, Jerry Levelsmeier, the late Jim Niswander, Gorgias Gambacorta, Sally Stick, Sundeep Sharma, Cliff Sink, Alice Hausman, Keith and Lori Jacobi, Paula Atchison Arthur, Adeline Masquelier, John Phelps, and Dennis Laake for favors and ideas too numerous to list. Special thanks to Rolf Fränkel for his kind interest.

One does not go through so many years of productive academic change and development without a lot of love from the support group. I appreciate especially my parents, Mildred and Robert Joe Corruccini, the latter of whom transmitted my valued and remarkable (and oft remarked upon) Carabelli cusps. To Carol, Annie and Joe, victims of malocclusion all (as was I), I devote this book, but not without remembering the special qualities of my own boyhood orthodontist, Dr. Bell of Boulder.

RSC

1. Introduction.

Today, the orthodontic specialty of the dental profession copes with a very high rate of crowding, rotation, maleruption and other irregularities of teeth and jaws. Surveys (Lombardi and Bailit, 1972) indicate nearly half of youths in countries of the affluent Western world could use or have availed themselves of orthodontics. In the United States of America some 40-60 percent of youths have malocclusions (Kelley and Harvey, 1977), with orthodontic therapy rated desirable to mandatory, if not already underway. Decades ago the U.S. Health and Human Services Department released the official statistic that orthodontics has become the largest single item of normal expense American parents can expect to encounter in raising a child (prior, that is, to college). For these reasons, orthodontics textbooks (which of course are oriented toward the Western populations mentioned above, because that is where one finds orthodontists) agree that malocclusion is not a disease state in the usual sense, for it is closer to being a norm than an aberration.

When could this curious state of affairs have become typical for our species? When did the condition of malocclusion arise to plague humans, and could it have always been so widespread? Is it logical that humans (or for that matter, other animals) could always have had such frequent disorder of the correct tooth-to-tooth relation during biting? In fact, such prevalent malocclusion is not natural in nonindustrial human populations (Corruccini, 1984), and any detectable occlusal malalignment in nonhuman animals is quite rare (see e.g. Corruccini and Beecher, 1984). Therefore we are presented with a mystery concerning the inception, rampant increase, and cause of the disorders that have today spurred a

mandibular incisor was lost premortem in the figured girl, and the point of the prosthesis was to close the gap that would have been externally visible since it involved an anterior tooth.

To cope with our curiosity, a sample of 50 Etruscan skulls (all lacking the prosthetic treatments) were gathered in 1986 to constitute our study sample. All are housed in the National Museum of Anthropology and Ethnology within the Institute of Anthropology, University of Florence. The majority (32) came from the zone of Chiusi and Chianciano in Siena Province and form part of the Dini collection. The remainder are from proveniences in Tuscany or adjacent regions: Solaia and Colle Val D'Elsa (Siena); Cortona (Arezzo); Talamone and Marsiliana (Grosseto); Volterra (Pisa); Castiglion del Lago (Perugia); and Marzabotto (Bologna) and were consigned to the Museum from parts of diverse collections. They comprise a time range no more precisely definable than from the VII to I Centuries B.C., although at least 80 percent of the cases can be circumscribed by the VII and IV Centuries. All were cataloged as definite Etruscans (not, for example, "Etrusco-Romans"), and were collected in the 18th or very early 19th Centuries.

Tooth rotation/displacement count (the number of noticeably malaligned anterior and posterior teeth with badly malaligned teeth counted twice: Kelley and Harvey, 1977), M1 transverse crossbite, and horizontal buccal segment relation were scored. The latter two traits were expressed in tenths of the diameter of the upper molar's major occlusal cusp (see Kelley and Harvey, 1977; Corruccini, 1984). The maximum 95 percent statistical confidence interval values were compared with a worldwide sampling of nonmodernized versus industrialized populations (total n=2371: Corruccini, 1984; Corruccini and Choudhury, 1985).

At most, this Etruscan sample could be ascribed a rotation and displacement tooth count of 0.44; mean scores of at least 1.38 typify modern foragers whereas a much higher mean of no less than 4.20 obtains in the large sampling of modernized (that is, western or westernized) peoples. Similarly, for

crossbite and for buccal segment discrepancies, Etruscans showed one-half or less average extent compared to the more recent samplings. While the tooth rotation/displacement figure is somewhat underestimated in Etruscans because of few post-mortem retained incisors, the other two traits should be under no scoring bias.

Etruscans thus resembled hunter-gatherer (and other non-acculturated) people much more than the frequently maloccluded modern western people, confirming the very recent epidemiological proliferation of malocclusion. True, some of the low scores for Etruscans must result from inability to score the relations among missing teeth, especially incisors (although the alveoli could nevertheless be observed), and more reduction in the apparent level of occlusal variability would occur if maloccluded adults were more likely to lose teeth and hence be missing from the sample. Nevertheless, these nonrandom factors could not have been large enough influences to create distinctive results such as these. It is reasonable to infer that the Etruscan sample falls into the lower range of occlusal variations shown by human foragers and nonindustrialized agriculturalists, as opposed to modern and industrialized samples.

We earlier (Corruccini, 1984, 1991; Pacciani and Corruccini, 1986) reviewed the etiological factors possibly related to cross-cultural patterns of occlusion. These shall be treated anew in more detail later in this volume. Aside from suggested genetic mechanisms (which are now largely discredited: see later chapters), various dietary factors are major potential determinants. Among these, the interproximal attrition brought about by dietary grit may increase arch space in aboriginals. We favored an old anthropological idea that dietary consistency and toughness promote alveolar remodeling and proper permanent tooth eruption, bringing about ideal adult occlusion; when nonresistant processed foods become ubiquitous after industrialization, and the eruption and cuspal coordination of teeth lose the critical pathfinder influence of vigorous masticatory pressures, malocclusion shows a rapid rise.

Etruscan diet, even for the nobility, was not intensively refined. Meat

derived mostly from domestic animals, especially goats, sheep and pigs (De Grossi Mazzorin, 1985). The domesticated animals were not slaughtered young (almost never in the first year). While De Grossi Mazzorin documents a little wild game, Camporeale (1984) suggests more of a hunting and wild fruit component to the diet. Cereals, wheat and grains for a relatively coarse bread were the staple foods (Cristofani, 1985), and Neppi Modona (1959) and Bonacelli (1928) document the legume and other crop food sources.

A more intensive comparison with 69 19th-Century Tuscan skulls (Moggi-Cechi, Pacciani and Pinto Cisternas, personal communication) showed 13 percent of individuals with notable anterior rotation/displacement of teeth, twice as frequent as Etruscans (6.8 percent) but the score did not differ significantly and the Tuscan occlusal health (at the industrial revolution's initiation) was still relatively good.

World Patterns in Occlusion

These findings were a recent part of a sequence of cross-cultural investigations into frequencies and severities of occlusal anomalies, that has involved myself for 20 years but continues a line of thinking whose history extends into the 19th century. All these studies rest on the conviction that solving the mysterious sudden prevalence of malocclusion requires a critical perspective on etiology, that derives from looking at populations different from ourselves (i.e., from Americans or Western Europeans). Therefore the occlusal question is quintessential anthropology -- that is, trying to pinpoint exactly what constitutes the natural or ancestral human state of affairs through using cross-cultural and archeological data. As we shall see, our "primitive" condition is to be well-occluded, and this has significant implications for investigating malocclusion.

Malocclusion is a deviation from a rarely attained perfect arrangement among teeth, that does not necessarily cause a functional problem. Therefore, the developing trend has been to speak of "occlusal variation" to avoid the connotation of the word malocclusion. Two broad sets of theories have been

proposed to explain irregular occlusal variation. One is based on genetic arguments; the other emphasizes the role of environment. The difficulty of separating these factors is obvious, since controlled human experimentation is not possible. For many years orthodontic texts indicated belief that the genetic factor is most important, thereby rendering any preventive measures impossible (Katz et al., 1976). Lundstrom (1948) showed that overbite, Angle's buccal segment relation criterion, and spacing-crowding of teeth are heritable in decreasing order, and many articles have been written demonstrating special occlusal similarities in families. This demonstrable heritability has not caused orthodontic researchers to discard environmental causes totally, but they tend to emphasize determinants such as thumb-sucking, mouth breathing, abnormal muscle patterns, inadequate masticatory function, abnormal swallowing patterns, and premature deciduous tooth loss rather than decreased chewing stresses and exercise resulting from soft, processed foods.

For example, some environmental influences listed by Litton et al. (1970) and Garn (1961) are: mouthbreathing, premature deciduous tooth loss, hormonal intervention, endocrine disturbance, trauma, habits, posture, disturbance of synchronous growth in separately growing parts, prenatal insult, and independence of tooth formation. Other renditions of environmental etiologies are found in Shapiro (1969), Chung et al. (1971), Watnick (1972), Schouboe and Houge (1973), Jago (1974), Nakata, Yu and Nance (1974) and Chung and Niswander (1975). Oral habits are frequently blamed in general (Nanda, Khan, and Anand, 1972; Moore, McNeill and D' Anna, 1972; Infante, 1976). Nutrition is sometimes vaguely alluded to (e.g., Wiener and Purser, 1957). Other things encountered in the literature include sleep positions, head posture, systemic disease affecting overall physiological development, pre- and perinatal factors possibly associated with climate (Janerich and Carlos, 1968), and environmental effects on peri-oral muscular behavior (Jacobs, 1966; Ingervall and Helkimo, 1978).

Gene-Based Thinking

There are three basic theories to explain any putative genetic increase in prevalence of occlusal variation in modern urbanized populations:

Gene Theory 1. Genetic inbreeding allows normally rare traits to find expression in the homozygous state. Genetic studies on occlusion are not yet conclusive on this point (Niswander, 1975; Smith and Bailit, 1977). The fact that malocclusion appears rapidly (within one generation) in some aboriginal peoples, once they are contacted by Western cultures, also calls this explanation into question.

Gene Theory 2. Genetic admixture causes independent assortment and disharmonious mixing of various shapes and sizes of tooth and jaw attributes such that they no longer fit one another. Although one of the most ancient in orthodontic thinking, this idea is still encountered today (e.g., Lavelle, 1972). Sim Wallace (1904) dealt with it long ago:

> "If we assume that the maxilla is not developed on account of a 'hereditary tendency' for bone not to be developed by the stimuli which do develop bone, then we may believe this. However, the hypothesis need hardly be discussed, as the idea is preposterous. Then as regards the idea that a small jaw may be inherited from one parent and the large teeth from the other, it is evident that the size of the crowns of the teeth determines the amount of development of bone on the posterior borders of the body of the maxilla and provided there is adequate forward translation of the maxilla by the developmental stimuli mentioned, then the size of the maxilla will necessarily correspond in size to that of the teeth.... More in pity than with satisfaction, I observed in the 2nd edition of Messrs. Smale and Colyer's text-book, ... the old theory that large teeth may be inherited from one parent and small jaws from the other is still maintained."

One weakness of the mixing-of-parts theory is that mismatches leading

to dental spacing would be expected to be as frequent as dental crowding, whereas the latter is much more prevalent. Lombardi and Bailit (1972) explain that teeth and jaws are highly polygenic traits; their inheritance requires much linkage, which would lessen independent segregation undergone during meiosis. At any rate people do not exhibit mismatched arm and/or leg length if their parents are of disparate stature. "We are not confronted, for example, with individuals with impacted brains because they received genes for large brains and small skulls", satirizes Mucci (1982). The currency of Functional Matrix thinking, that "bone need have only the genetic capacity to form and to react, and that its growth is controlled ultimately by its local and general environment" (Johnston, 1976), also weakens the concept of such genetically independent parts. The functional matrix hypothesis does not seem to have experienced much application to the question of masticatory disuse and its craniofacial correlates.

According to Bixler (1974) genetically homogeneous population groups tend to have "normal" occlusion, whereas populations subject to racial admixture have experienced significantly greater incidence of jaw discrepancies and occlusal disharmonies. Bixler summarized the findings of a cephalometric study on triplets (conducted by Kraus et al., 1959) as indicating that while the morphology of an individual bone is under genetic control, the environment plays a major role in determining how various bony elements are combined to achieve a harmonious craniofacial skeleton. Chung et al. (1971) concluded to the contrary that human racial crosses present no additional risks for "malocclusion."

Many anthropologists have reasoned that in recent times orofacial reductions appear to be affecting jaws more than teeth, and consequently disharmony of size is occurring (Keith, 1924; Hooton, 1946; Goose, 1956, 1963). Moorrees and Reed (1954) measured the mandibular teeth and found a lack of association between tooth size and dental arch size to be responsible for the crowding and spacing that they observed. Hooton (1946) opined that though an evolutionary trend of reduction in facial and dental growth has long existed, the process has suddenly been accelerated under urban, cereal diet conditions.

Dickson (1970) offered a slight variant in reasoning, namely that explosive population increase allows malocclusion to proliferate. Goose (1956, 1963) attributed the decrease in palate breadth in modern British skulls (versus those from earlier historical periods in Britain) to the change to a softer diet which led to a lack of function as the causative factor. Since not everyone is affected, one could speculate that some individuals possess a genetic background less conducive to the proper development of their jaws in the absence of this factor. Studying tooth breadth and alignment in the permanent dentition, Lundstrom (1955) observed that large or small teeth were associated randomly with large or small jaws. In this regard, Foster et al. (1969) noted that in British children aged 21/2 to three years, correlation coefficients between total arch length and dentition size were high for both arches and both sexes.

Gene Theory 3. Natural selection pressures have been relaxed for humans because of technology. Hence, genetic mutations allowing irregular occlusion are no longer selected out and so they accumulated in the population over many successive generations (e.g., Wolpoff, 1969).

Regarding the decrease in dental arch size in modern communities, as compared to the medievals, Brash (1924) stated that the narrow, contracted arch was found in the Middle Ages, and it is only the incidence thereof that has increased which may be due to racial heterogeneity or "Survival of the Unfit" (see also Brash et al., 1956). Again, as with the inbreeding theory, a major drawback to this explanation is the fact that aboriginal populations develop a significant incidence of malocclusion within one generation after contact with Western technology and food products.

Harris and Smith (1980) think earlier works (Harris and Kowalski, 1976; Harris, Kowalski and Walker, 1975a,b) have wrongly attributed observed resemblances to genetics. Similarity due to shared environment is the basis for genetic models of cultural inheritance (Carlson and Van Gervin, 1977; Lundstrom and McWilliam, 1987). Garn, Cole and Bailey (1979) have shown that shared environments ("cohabitation effect") result in family resemblances for a number

of features, irrespective of whether the individuals living together are genetically related.

Good Occlusion is "Natural"

An important consideration in understanding this occlusal variation is the tendency among nontechnologic human societies for virtually all individuals to show nearly ideal occlusion. Lombardi and Bailit (1972) give variable incidences from the literature of severe "malocclusions" in human populations that range from 17-38 percent in Western groups but only 7-16 percent in non-Western groups. The lower latter figures pertain to incompletely urbanized "anthropological" populations. Malocclusion thus is a malady of "civilized" humans (Hunt, 1961). This observation gives rise to a theory widely favored among anthropologists: that malocclusion arises from the lack of chewing stress in the modern processed diet and the ensuing lack of stimulation and direction provided to the growing jaws and erupting teeth (Wood Jones, 1926; Keith, 1931). Fossil hominids lack any indication of severe malocclusion, at least in the sense of its definition among modern humans (Pereira and Evans, 1975; Begg, 1954). Some review articles on the etiology of occlusal variation (Moorrees et al., 1971; Smith and Bailit, 1977) do not mention lack of chewing stress as a possibly responsible agent, even though growth processes are strongly associated with development of malocclusion and growth is influenced by environmental requirements. However, many references have been made to dental occlusions that are less crowded or malaligned and with fewer discrepant relations, in (a) less developed, acculturated, or earlier components of many populations as compared with (b) their more modernized or industrialized counterparts (see reviews in Richards and Barmes, 1971; Davies, 1972; Corruccini and Whitley, 1981; Corruccini, 1984, 1991; and Corruccini and Macchiarelli, 1987).

Early studies by Price (1936) found perfect occlusion among Eskimos, while after contact with industrialized societies the incidence of malocclusion rose to 50 percent or more (Price, 1936; Williams, 1943; Wood, 1971). The diet had

shifted to softer foods, including refined sugars. Klatsky (1948) demonstrated that technologically primitive human groups show larger jaws and better occlusion than contemporary Americans. Price (1935) attributed the rapid increase in malocclusion to industrialization and based his conclusion on studies of several ethnic populations (1939). Similarly, a study of Taiwan aboriginals showed that they had nearly ideal occlusion. Lu (1977) suggested that the raw vegetables in their diet provided adequate jaw growth from muscular stimulation by vigorous mastication.

Campbell (1938) and Campbell and Lewis (1926) gathered small amounts of data from nomadic versus settled and rationed Australian Aboriginals in different regions, finding more frequent attrition, perfect occlusion, and edge-to-edge incisal relations in the former. Clinch (1951) executed a preliminary comparison along the lines formulated by Campbell, comparing malocclusion prevalence among an unacculturated sample of 108 Haasts Bluff Aboriginals and another "civilized" (living on a reservation) group of 54 individuals. She found normal Class I occlusions much more common in the nonurbanized sample, with sharply increased Class II, Class III and deep bite prevalence in the urban, even though the caries rate was identical. Bjork and Helm (1969) showed that Australian Aboriginals of Yuendumu had considerably less frequent occlusal discrepancies of many types when compared with other diverse populations, particularly modern European ones. Furthermore, Helm (1979) found more orthodontic problems in modern Danes than either medieval Danes or Australian Aboriginals and suggested this may be associated with a lack of vigorous masticatory function and dental attrition in modern societies.

Keith (1920, 1924) has shown that overbite did not appear with the food-producing revolution, and it did not come to categorize the European occlusion until after the Middle Ages. Brace (1977) suggests that the parallel between adoption of the table fork and the appearance of overbite is too striking to be coincidental. Kiliaridis (1986) lists other studies demonstrating lower malocclusion prevalence in medieval as opposed to modern-day peoples

(Lundstrom and Lysell, 1953; Mohlin et al., 1978; Helm and Prydso, 1979).

Relatively good teeth in ancient Greece were an attribute of presumed vigorous chewing mechanism; defective modern teeth occur with frequent definite overbites, more crowding, and more projecting chins (Angel, 1944). According to Angel, among ancient Greeks no association between definite overbites and decayed teeth is demonstrable.

Goose (1972) documented a trend toward narrowing arches in first-generation Chinese immigrants to England, a trend that is well advanced in European people. Goose et al. (1957) as well as Barnard (1956) showed rural British and Australian children had better occlusal health than nearby urban children. Modern-day New Zealand Maori suffer constricted palatal growth compared to earlier crania (McCann et al., 1966). Van Reenen (1966) found occlusal anomalies at a 27 percent rate in partially sedentized or agriculturalized South African Bushmen, but only at 7.5 percent in nomadic Bushmen. Finally, Niswander (1967) showed that in a nonacculturated group of South American Indians only 5 percent had malocclusion while 45 percent of a long-acculturated community of Indians living at the same post had malocclusion.

Although crowding, psychological stress, environmental noise, or prenatal disturbances might contribute to such rapid changes in occlusion, much information indicates that the key altered variable in these circumstances was diet and the associated stress of mastication. For instance, Ahlgren et al. (1973) and Moyers (1949) found lessened electromyographic activity recorded for masticatory muscles and variable muscle function in persons with malocclusion. Histology detects neuromuscular changes that affect skeletal change in growth, which in turn was affected by change in masticatory muscle demand (Kiliaridis et al., 1988). Watt and Williams (1951) and later investigators have shown that the maxillary dental arch becomes narrower in rats fed a water-softened diet as opposed to a hard form of food. This is superficially similar to the maxillary collapse shown by Goose (1972), Lavelle (1973) and many others to be increasing between parents and offspring and to afflict many American youths needing

orthodontic treatment (Kelley and Harvey, 1977).

Waugh (1937a, b), Klatsky and Fisher (1953), and Oppenheimer (1966) documented a direct relation between reduction in chewing power and demand made on supporting structures, underdeveloped muscles of mastication, and incidence of malocclusion. Weijs and Hillen (1986) found the very important result that variation in masticatory muscles affects various osteodental structures of the face, and does so variably, in humans. Results such as these led Lombardi and Bailit (1972) to suggest the need for more research directed toward the effect of masticatory stimulation on jaw development and congruency of occlusion. Bite forces are demonstrably important to temporomandibular joint growth (Moore, 1965; Hinton and Carlson, 1979). The mandibular condyle is known to grow in response to "strenuous oral function during growth" (Hinton and Carlson, 1979).

Also worth noting is the virtual inevitability of some relapse of orthodontically treated occlusions once treatment is finished, no matter what interventions are attempted or risk factors studied (Blake and Bibby, 1998), in the modern context of mild chewing exercise.

What Precontemporary and Contemporary Textbooks in Orthodontics Have to Say

A discussion and listing of the etiologies of malocclusion has always been a fairly obligatory (although frequently brief) aspect of the orthodontic textbooks upon which the practitioners of this specialty are raised.

It is interesting to survey the enumerated etiologies, as I have done at several major universities through the courtesy of their orthodontic departments' reading rooms. There is a remarkable degree of uniformity to the standard list of causes encountered (Moyers, 1988; Proffit, 1986; Nanda, 1983; McNamara, 1976, 1977; Thurow, 1977; Begg and Kesling, 1977; Salzmann, 1974; Dawson, 1974; Wheeler, 1974; Graber, 1972; Sassouni, 1971; Ramfjord and Ash, 1971; Lundstrom, 1960; Brash, 1956; Melsen, 1991; van der Linden and Boersma,

1987). This standard list comprises "functional environment" which is then specified (very narrowly) as nasorespiratory and postural in nature. Caries (causing tooth movement or arch collapse), loss of teeth, and supernumerary teeth are blamed. Habits (especially finger/thumb sucking and tongue thrust), muscle recruitment, muscle malfunctions, abnormal swallowing, and mouth-breathing and/or adenoids are invariably listed. The tongue may be too large or too small, there may be unstable occlusal stops, contraction of the arches, insufficient growth, temporomandibular dysfunction, lack of attrition, lack of normal occlusal adjustment throughout maturation, premature loss of deciduous premolars, or suboptimal lip/tongue/cheek pressures. Hormonal or endocrine variations, trauma, prenatal insult, disturbed growth, and uncoordinated tooth formation are variably listed. Malnutrition as opposed to hypervitaminosis, and bottle-feeding versus breast-feeding, are contradictorily given as causes.

Above all, genetic factors (as detailed above) are given a strong textbook emphasis. Indeed, among these sections a monotony of causes are blamed, and the causes of those causes (e.g., what is the cause of the abnormal muscle pattern etiology?) are transferred ambiguously to a generalized combination of heredity and environment.

What I find fascinating is the minimal revision the etiology list has undergone since the very early days of orthodontics. It has been passed on down almost verbatim from Angle (e.g., 1907). Passed on, that is, with one major exception. Angle listed and elaborated the "disuse" theory, that is, the idea that use stimulates growth: Prehistoric humans had good occlusions but with modern food preparation, there has been a marked general effect. Later texts published 1920-1980 (see the long list in Klatsky and Fisher, 1953:160-163) directly borrowed Angle's classic etiology list and even his sequence, but routinely excluded just the disuse etiology. Some, however, made mention of Wolff's Law. Brash et al. (1956) did briefly mention refined diet and muscular stimulation. Lundstrom (1960:174) took the trouble to contest the idea: "The fact that one of a pair of siblings may have well developed jaws and the other a marked crowding,

even though the diet is the same suggests that, if the physical properties of the diet are factors, they cannot be important ones. It is also difficult to account for the extreme variation in the size of the jaws today when a soft diet is the general rule. Whatever the answer to this question, it is hardly conceivable that the reduction in the intensity of mastication, in historical times, is a factor of real importance in the etiology of malocclusion." This outlook downplays the role of gene-environment interaction in etiology; the idea behind the disuse mechanism is that strenuous function guided development and masked "hidden mutations" and unimportant genetic variation that accumulated, unseen by natural selection, through human prehistory. This variation, including all the various directions malocclusion takes, then was released (so goes the argument) when the canalizing role of strenuous use was very recently removed.

In major recent textbooks the disuse etiology receives some mention. Proffit (1986) finds this mechanism "not obvious" and favors other mechanisms that would have a longer duration in influencing the oral region such as lip, tongue and cheek pressure. His insistence on a 6 hour daily minimum for an agent to have a skeletal effect (exceeding the daily duration of mastication) is equally not obvious to me.

In Moyers' (1988) latest edition, there is reference to the way in which muscular determination of bony growth is in "vogue" due to Moss' functional matrix thinking (for a rather philosophical update see Moss, 1997). Moyers thinks mastication is an unlikely influence unless forces are heavy and/or act over long periods of time. After some discussion Moyers (1988:152) concedes "The evidence seems to indicate that our highly refined, soft, pappy modern diets play a role in the etiology of some malocclusions." He goes on to give tongue thrust, finger sucking and mouth-breathing some 30 times as much discussion space as disuse, although the former agents are reliably linked only to relatively rare occlusal anomalies (namely, posterior crossbite and openbites). In fact substantial ambiguity and uncertainty remain concerning respiratory and nasal obstruction in relation to growth (Vig, 1998).

As shall be emphasized repeatedly below, the majority of modern malocclusions take the form of crowded and hence distorted arches, and the variations thus entailed conform to the expectations of the disuse theory, whereas only a small minority of specific syndromes are attributable to the functional factors that see so much attention in orthodontic literature.

The Oldness of the Idea Concerning Masticatory Exercise

Much of ongoing writing activity in the areas of malocclusion etiology is reinventing the wheel; the major ideas about functions and disturbances, the leading role of genetics, and the disuse theory and other theories, were already in circulation in the previous century.

The eternal nature of the rhetoric surrounding orthodontic opinions is well demonstrated by Sim Wallace's (1904) book, constructed from various articles extending into the previous century. Sim Wallace was a dentist among the early heterodox thinkers, who had an influence upon Campbell (1925), Klatsky and Fisher (1953) and other later investigators. It is appropriate here to give a sizable extract of Sim Wallace's (1904:pp. 2-28, 50-57, 62-71 et seq.) writing, and to think how appropriate and applicable much of it is today, nearly a century later. Firstly he complains about his contemporary clinicians' narrowmindedness, and states the problem::

> "Of course what follows will reach but a small section of the dental profession in the immediate future. The progressive members are not too numerous, and lack of knowledge of the biological questions involved -- especially the question of heredity -- makes it impossible always to expect either intelligent appreciation or criticism.
>
> When we consider that, in order to get an efficient masticatory organ it is necessary that all the teeth should articulate with the greatest nicety, we are brought face to face with one of two alternatives: either the correlated growth of the upper and lower jaws and teeth must be extremely exact and

definite, which it is not, or there must be some controlling influence common to both, which, as it were, guides the teeth to their exact position and arrangement...."

Sim Wallace then discusses the influence of chewing powerfully during growth:

"During the few years that the deciduous teeth are capable of full functional activity, what must be the effect of the tongue of eating the soft pap, liquid extracts and refined foods of the present day, which require little or no chewing. I think it will be evident from what has been said on the influence of functional activity on the growth of muscle, that the development of the muscular fibres of the tongue is not as great as if it had been used while chewing fibrous food-stuffs, which require at least ten times the amount of mastication. The consequence of this is seen in the fact that the deciduous teeth are sometimes not translated outwards, and the spaces which should develop between them do not form, and the alveolar arch is not as broad as it ought to be."

Sim Wallace goes on to implicate other effects:

"My own observations show that a want of full use of the teeth gives rise to high palate, and this is supported by the fact that among patients of the upper classes--who are brought up on soft, refined foods--a high palate is much more frequent than among the working classes....

But it is not only the outer surfaces of the bone that the heavy pressure and strain of efficient mastication stimulate to develop, but it affects the deposition of bone in the articular sutures. Thus, then, there is a deposition of bone along the median suture uniting the maxilla and palate bones and a consequent broadening of the nasal fossae, especially the lower part, and so the nasal fossae are enlarged. Then, too, when we consider the

effects of the external pterygoid muscles pulling the maxillary bones from the condyles of the mandible right and left alternately, are we not justified in expecting and finding that the various sutures running transversely across the base of the skull between the occipital and maxillary bones should be caused to ossify more fully, thus translating the maxillary bone forwards and increasing the size of the naso-pharynx in the region so frequently occupied by adenoids."

He ties in some evolutionary implications:

"I maintain that Nature does not scorn mechanical forces when it suits her purpose to make use of them.... In works on the irregularity of teeth heredity is put forward as one of the main general causes, but this cause is so frequently introduced when the causation is obscure, that one feels inclined to think that it is introduced in order to make a pretense of knowing, when in truth the authors neither know the cause nor perhaps what they mean by heredity.

With regard to the irregularities of the teeth, if we consider any of them hereditary, we are placed face to face with the stumbling-block that our ancestors a few generations back were free from them."

And he supplies logical support from applied orthodontics:

"Although teeth are regulated and retained with a retention plate for years, if they are not left in a state of equilibrium, they will gradually become displaced immediately the retention plate is removed. How the arch of the teeth could be expanded and expected to remain in the expanded position without the pressure of a correspondingly augmented tongue is one of the strangest expectations which any class of mechanical men have yet harboured.

The idea apparently entertained by some, that after a time the teeth

may become fixed in the jaw with cast-iron rigidity is contrary to the notorious fact at the very foundation of the treatment of irregularities, that the teeth may be moved by pressure."

Sim Wallace proceeds to quote, cynically, contemporary hereditarians (Smale and Colyer) on the cause of malocclusion: "The brain and the osseous structures of the face derive their main blood supply from the same source, viz., the common carotid arteries. The strain of modern education--indeed the whole environment of the individual--entails a greater call upon the brain than a primitive mode of living, and thus necessitates a larger supply of nutritive material to that structure. This increased supply is probably provided at the expense of the osseous structure, including the teeth, with the result that these structures degenerate."

He also takes another humorous look at pseudo-evolutionary explanations from his time in quoting a contemporary clinician (Tomes): "For if the type of face nowadays considered to be beautiful be investigated, it will be found that the oval, tapering face with a small mouth, &c., does not afford much room for ample dental arches. On the other hand, the type of face which we considered bestial has a powerful jaw development. Perhaps generations after generations seeking refinement in their wives may have unconsciously selected those whose type of face hardly allows the possibility of a regular arrangement of the full number of teeth." At this point Sim Wallace answers "what form of irregular arrangement is it that men prefer above all others to such an extent that they have allowed the women with well-developed jaws to perish barrenly?"

The range of ideas from the 1890's is not so different from some of what is still encountered in the 1990's -- as I shall subsequently try to demonstrate.

The Idea Reintroduced: Klatsky and Fisher

In 1953 the anthropological dentists Klatsky and Fisher undertook in a

book to remind the profession of this theoretical framework, extolling the memory of Sim Wallace in the process, and again making numerous points that are not at all anachronisms in the present time. They therefore (Klatsky and Fisher 1953: pp. x, 2, 22-25, 36-38, 171-178 et seq.) deserve a lengthy extract here. First they introduce the idea that malocclusion is a "disease of civilization":

> "Education in physical anthropology is of greatest importance to the dental student and teacher. The dental profession, in spite of the enormous accomplishments in the mechanical and biological fields has, so far, failed in its greatest task of preventing dental disease and disorder of the human masticatory apparatus. Our communities suffer more from caries, periodontal disease, underdeveloped jaws, and malocclusion than ever before in the history of mankind. The masticatory organs of ancient, prehistoric, and contemporary primitive races, who did not come in contact with civilization, were better developed, functioned more efficiently than ours, and were not susceptible to disease as those of civilized peoples. This knowledge is revealed to us by anthropologists who have investigated the dental organs of those peoples....
>
> Dental caries, malocclusion, and other pathological conditions and abnormalities, while they were found in some skulls of ancient and prehistoric man, their incidence has constantly been increasing with the advent of civilization and are most prevalent in the mouth of modern civilized man. They are undoubtedly a product of our modern civilization brought about mainly by lack of use and function due to change in the physical and textural consistency of the food we consume."

The "disuse" idea is, again, defined:

> "The jawbones of modern civilized man are markedly underdeveloped, there is a great increase in the incidence of malocclusion, and

an enormous increase in the dental caries and other diseases. These diseases and abnormalities are brought about by the loss of use and function of the masticatory organs due to change from hard, bulky, and fibrous food to a diet of soft and liquid consistency which does not require normal mastication. Our modern time-saving and labor-saving devices, products of our civilized mode of living, are mainly responsible for the present-day degenerative changes in our masticatory organs.

From the above, we can readily see what an important factor function is in determining the size and structure of living organs, and yet so little attention is given to this subject by most of our dental investigators. Lack of use of our masticatory organs is greatly responsible for the diminution in size of the jaws and the overcrowding of teeth which we have been observing of late."

Reasons underlying this mechanism are specified:

"Why do the jaws of modern man diminish in size and quality? To find a satisfactory answer, it will be necessary to re-examine the three basic factors which influence growth and development .... and see which one is most responsible for these retrogressive changes in our jaws.

Heredity is obviously not the causative factor, because heredity is more conducive to organic stability and balance than change and disharmony. By the genetic transmission of physical traits from parent to offspring, nature tends to preserve the general pattern, shape, and size of the body, and to develop its component organs in relative proportion....

Environment cannot possibly be considered as the cause, because environment favors modern man as against ancient or contemporary primitive man. Modern, civilized people enjoy better climatic conditions; they are better protected against the inclemencies of the weather; they are better housed, better clothed, and, above all, better nourished than the average

ancient or primitive people.

Having eliminated the other two factors which influence growth and development, we must come to the logical conclusion, that the third factor, namely, function, plays an important role here, and that lack of function is mainly responsible for the degenerative changes in the jaw-bones of civilized man which we have been observing of late. This was confirmed long ago by no less an authority than Sir Arthur Keith:

'The narrow bony opening to the nose, with its gill-like nasal spine, its raised and sharp sill, so often seen in modern English skulls, are conditions never present in Englishmen of the pre-Roman periods. Contracted palates, crowded and defective teeth, deformed jaws, sunken cheek bones, do not become common in English graves until we reach the eighteenth century. The appearance of these structural changes in Englishmen cannot be attributed to the introduction of new racial element from abroad. No doubt these facial changes are due in part to the soft nature of our food, and to the disuse of our muscles of mastication.' "

In comments that are still relevant today Klatsky and Fisher explain that the nutritional input to craniofacial matters is usually misconceived:

"Cognizant of the fact that the diet of the average primitive is of inferior nutritive value to that of the average European the anthropologist is prompted to eliminate nutrition as a possible factor in superiority of the dental organs of the former over those of the latter. This condition is attributed rather to the hard, bulky, resistant foods consumed by primitives, as against the soft, refined, non-stimulating diet of modern civilized man. Greater attention must be given to this very important factor, and more research along these lines should be encouraged.

It is regrettable that modern dental investigators are neglecting this phase of the subject. They seem to be lured more by the dramatic aspects of

nutrition than by the "prosaics" of food texture. In their desire to clothe their research in great scientific respectability, many students confine their investigations to the biochemical significance of food. The dental literature of today is replete with discussions on the relation of diet to dental and oral health. Most of these discussions, however, emphasize the metabolic and nutritional aspects of food."

Epidemiological Context

Continuing the historical slant, malocclusion used to frequently be referred to as a "disease of civilization", signifying that its is found (or at least reported) primarily in developed, urbanized populations. The phrase has several unfortunate connotations; recently Trowell and Burkitt (1981) classified as "Western diseases" many afflictions (such as congestive heart disease and hypertension) that are common in the industrialized world but considerably rarer in less developed societies living in conditions more characteristic of the ancestral human environment. The shift from predominantly infectious to noninfectious degenerative disease deaths typifies the "epidemiologic transition" through which European-derived populations have passed, and developing societies are currently passing. The transition is a major focus of research and health care planning and plays a critical role in the search for etiological factors.

Corruccini (1984, 1991) adapted the concept of the epidemiologic transition to various occlusal and orthodontic studies, alluding to the numerous instances of nomadic or unmodernized peoples displaying predominantly good anatomic occlusion, while settled and acculturated components of the same populations have the high western style prevalence of crowded and crooked dental arches. This might signal an epidemiological transition in occlusal health, although comparison of data has been hampered by inadequate controls and by lack of standardization of the term malocclusion.

Obviously, the malocclusion mystery is one specialized aspect of the age-old dichotomy between environment and heredity as primary agents of human

variation. The futility of that simple dichotomy will become evident in the ensuing chapters, as it has been in most avenues of human investigation. The quest for precise specification of the circumstances of malocclusion, as detailed below, thus might be seen as a worthwhile and novel effort considering the prevailing ambiguity of thinking, the enormity of the medical problem, and its expense.

2. A Methodology for Comparative Occlusal Studies.

According to Niswander (1975) "malocclusion" is complex and appears mostly to represent disharmonious or extreme combinations of a number of continuously varying characters. Few investigators any longer divide the whole range of occlusal variations into subjective dichotomies of malocclusion versus normal or good occlusion. The first concerted attempt to develop a standard method of recording occlusion was made by the Commission on Classification and Statistics for Oral Conditions at the Second International Conference on the Epidemiological Assessment of Dentofacial Anomalies, in 1969 (Baume et al., 1973). The classification should be measured along a continuum and the numbers should be meaningful in terms of intervals and rank order. I have adapted variables describing occlusal variation from Kelley and Harvey (1977:Appendix I) as in Corruccini (1984). These in turn are derived, explained and figured in Baume et al. (1973). Most of the variables go into computation of a synthetic measure of malocclusion, the Treatment Priority Index (TPI). All variables are judged with arches approximated in maximal intercuspation, sometimes referred to as centric occlusion.

Overjet is the anterior distance from the labial surface of the maxillary central incisor perpendicular to the labial surface of the corresponding mandibular central incisor. If one of the central incisors is obviously displaced relative to the arch, or badly rotated, then the other should be used for the measurement in order to best reflect the skeletal as opposed to dental occlusal component. Overbite is the fraction of the lower central incisor's labial crown height (from the cervix) that is vertically overlapped by the maxillary central incisor. In the rare case of

openbite, a negative number is registered as the amount open relative to lower incisor crown height. The openbite should also be recorded in mm for calculating the TPI.

Posterior crossbite is the amount of transverse displacement from ideal alignment of the upper first molar. This is expressed as the estimated fraction of the protocone (mesiolingual cusp) diameter that is displaced buccally (negative score) or lingually (positive) away from ideal relation, which is with the protocone apex in the mandibular antagonist's central occlusal fovea. Buccal segment relation (BSR) is the sagittal discrepancy of the maxillary first molar's paracone (again expressed as cusp diameter fraction) from overlap with the mandibular first molar's buccal groove; mesial displacement (Class II) is scored positive and Angle Class III negative. These values define crossbite and BSR "type", while the absolute value gives crossbite and BSR "extent".

If the situation is bilateral or if involving more teeth than the first molar, BSR and crossbite scores can be incremented by as much as 0.4. Values for crossbite and BSR are recorded only from one side if occlusal relations on the other side were disrupted by a lost tooth or other abnormality. This continuous-variable scoring method differs a bit from Kelley and Harvey and Baume et al.; in order to properly calculate the TPI, one must also note the number of teeth involved in crossbite relationship of at least cusp-to-cusp magnitude, and note whether one or both sides of the arch are in BSR discrepancy, which class of discrepancy, and whether involving cusp-to-cusp or full displacement from normal. The full condition, for example, in the case of mandibular distoclusion would have the maxillary first molar's mesiobuccal cusp overlying the space between the mandibular first molar and distal premolar.

Tooth displacement score is the sum of noticeably rotated or displaced (R/D) teeth, with badly malaligned (displaced more than 2 mm), rotated (more than 45 degrees) or impacted teeth counted twice. These variables are combined in the composite epidemiological TPI (see Kelley and Harvey, 1977). This index particularly emphasizes tooth displacement score, but the weight of that plus

incisor overjet and overbite anomalies is influenced by the BSR discrepancy and points for crossbite discrepancy are added afterward.

Many questions remain concerning occlusal indexes, of which a variety exist (Parker, 1998). This TPI is merely a comprehensive summary of conditions in my usage and treatment need is only vaguely indicated by the numbers. At any rate only 1% to 2% of modern occlusions are ideal so much subjectivity surrounds the meaning attached to index values (Parker, 1998).

A few standard whole-arch measurements are also of particular value. Maxillary arch length is the chord from the interincisal point to the left first molar's most distobuccal margin (the right side is used if the left arch is distorted or has a tooth missing). Maxillary arch breadth is the maximum transverse distance across the buccal side of first molar crowns. Absolute or relative maxillary arch breadth is one of the critical barometers of changing occlusal health.

In the present studies analytical emphasis will be placed on separate occlusal variables as well as the combined index designed to summarize over-all "need" for orthodontic treatment. The individual components making up the TPI are meaningful but the weighted total score may be misleading. My only purpose in using TPI was to derive an overall measure of occlusal variation that compounds the individual measures. The TPI is merely an epidemiological variable as used here and has no value for comparisons beyond our immediate purpose. Accordingly, we shall avoid defining malocclusion narrowly, referring instead to the statistical range of occlusal variation.

TPI criteria appear more replicable among different observers than orthodontists' evaluations of malocclusion severity. Thus, the necessity of treatment should not be considered directly related to TPI scores; the former must rest on value judgments and the accumulated experience of orthodontists. The usefulness of the TPI and related indices lies in epidemiologic studies, not clinical diagnosis. Contrary to the assumptions involved in classifying occlusal variation, no two malocclusions are ever identical. Therefore, separate analysis of the components of malocclusion may be more meaningful than analysis of a

summation of the components. It is acknowledged that our analysis takes into account only the relation of the teeth with one another in centric occlusion and does not attempt to distinguish in detail between dental and skeletal malocclusions; several of the observed traits are mixed indicators.

Data in our various studies have come from three sources: living populations (assessed by direct oral examination and denture wax-bite forms), dental stone casts, and skulls. Different data sources create some problems with comparability, but the same occlusal traits have been scored along the same continuum in all instances. There is some indication of correspondence between occlusal assessments based on casts and on oral examination, and among clinicians, nonclinical scorers and parents (Waltz et al., 1980; Ast et al., 1965; NCHS, 1967; Kelley and Harvey, 1977: Appendix IV).

Other variables could be added such as anterior crossbite, posterior openbite (scissors bite), and spacing (more than 2 mm between adjacent teeth that is not caused by a missing tooth). These features are partly redundant with those above, or sufficiently rare to be of limited statistical use in contrasting populations.

Only those specimens are scored that have no missing teeth (congenitally or otherwise), and at least all but one permanent tooth apart from M3 fully erupted, to avoid the various effects on the occlusion. Thus, the "adult" occlusion for these purposes is in arches having fully erupted central incisors through second molars in at least all but one quadrant. To avoid aging effects, occlusion is ideally scored for ages 12-25. For older population segments, up to two missing teeth have generally been allowed as a necessity in accumulating adequate sample sizes.

3. Studies on Occlusal Variation in Varied Human Populations.

In this chapter, I shall survey the variations in types and frequencies of occlusal anomalies according to the methods detailed previously. This shall be done in a roughly chronological sequence.

The Rural Kentucky Study

In 1979 while lecturing to a graduate class in Dental Anthropology, I mentioned the "disuse" theory for malocclusion that I had originally been alerted to by Don Ortner (Curator of Physical Anthropology at the Smithsonian Institution). I made a point of the United States population not being suited to testing of the chewing stress theory, since it is too uniform in dietary habits. Isolated rural populations which preserve (at least in part) a more traditional diet, I suggested, may offer the best opportunity to study variation in the development of occlusion. A first-year graduate student (who was eventually to receive his doctorate from Southern Illinois University), Darrell Whitley, was sitting in the front row and opined that he knew of, indeed grew up in, such a community. It is located in the Mammoth Cave region of central Kentucky.

Much of the traditional way of life of these people (all white) has been maintained (Whitley, 1979, 1985), but two major changes have been the movement of industry and mechanized farming into the area in the last 25 years. Traditionally, tobacco (the only cash crop), gardens, and orchards were grown by each family. Apples, pears, cherries, plums, peaches, potatoes, corn, green beans, peas, squash, peppers, cucumbers, and onions were grown for consumption, and fruits and nuts, grapes, and teas were gathered by individuals. In the diet of these

people, dried pork and fried, thick-crust cornbread (which were important winter staples) provided consistently stressful chewing. Hunting is still very common in the area.

Recently, markets and mechanized farming have made grain crops important as cash crops, and these are replacing the traditional farming as the people purchase more of their food. Younger residents are becoming industrial workers and often do not have time to participate in cultivation and group harvesting of food. Introduction of the tractor in the early 1950's began a transition in dietary habits that was completed with the appearance of large-scale mechanized farming in the last 25-30 years. Nevertheless, for some of the local people, life styles have changed very little since the 1920's. Social relations centered around working in groups to harvest the tobacco at precisely the right time, work groups usually consisting of several households from a single ascending-generation kin group.

Until recently the community never experienced any professional dental care, with the exception of some extractions. Among Appalachian peoples in general, there is low level of concern with dental health, and most treatment is undertaken in direct response to painful symptoms (Friedl, 1978). Tooth retention and dental health appear good, nevertheless, especially among females, who still retain most of their teeth into old age.

We decided this population was of particular interest for studies of occlusal variation in relation to diet, and I embarked upon the first (Corruccini and Whitley, 1981) of a 20-year program of studies described in this volume. In the Kentucky group, dietary variation and change covered a much wider range than would be possible in most other segments of the American population, and some control could be exerted over other environmental factors. For instance, most of the aboriginals cited in occlusal studies in the first chapter underwent not only a drastic change in diet but in many other habits and living arrangements, as most of them were on reservations or were immigrants to a new environment. Denser population and higher stress levels accompanied their dietary change. In the

Kentucky group, while diet changed, residence has not. Importantly, there has been no distortion of occlusal phenotypes through orthodontics.

Another feature of the target population is that it is highly inbred, especially by American standards. Almost all area residents are relatives. However, marriage distances have increased in the most recent generation, indicating a decrease in inbreeding.

Thus we started a project to describe occlusal variation in this rural community, to correlate occlusal variation with dietary and age variation and with a measure of inbreeding. Cooperation in terms of interviews and making oral examinations and bite impressions was satisfactory, but such was not the case for taking of alginate molds, so all occlusal observations were based on the former. The dietary criteria were developed, following some field study, to reflect stages of change in the interaction between subsistence, social organization, and economy in this community and also reflected Whitley's own experiences with the consistency of the traditional local foodstuffs. Dietary categories graded from modern (diet is entirely commercially purchased) to traditional: dried, fried, salt-cured pork is the basic meat, supplemented by wild game (again fried); cornbread is made from stone-ground corn meal; wild and garden foods complete the diet.

Gender was shown to be not associated with any of the occlusal variables (Corruccini and Whitley, 1981). Diet and age showed comparable patterns of correlation. Both were significantly associated with several of the major occlusal variables composing the TPI, and both showed the strongest single correlation with the TPI as a compounded measure of occlusal variation. Furthermore, all the correlations were negative, demonstrating that occlusal variation increases steadily with either decreasing age or decreasing amounts of traditional components to the diet.

Parental birthplace distance (PBD), conversely, was not demonstrably related to occlusal variation or jaw size. To support the theory that inbreeding is related to malocclusion in this population, significant negative correlations would be expected.

Having completed these screening analyses, we statistically compared occlusal variables across age and dietary variables to test the null hypothesis that the occlusions were random in relation to modernized and traditional sample components. As with ensuing analyses described in this volume, the results are presented below as line diagrams with indications of the magnitude of the statistic of difference, compared against the amount that would indicate 5 percent or less probability (one-tailed) of having sampled two such trait distributions from the same population.

Arch breadth was smaller and significantly more variable in the younger individuals while arch length did not differ. This result is not an artifact of adolescent growth, since only individuals were measured with complete eruption of the permanent dentition posterior to the point where the measurement was taken. The youthful individuals were more variable despite loss of fewer teeth (tooth loss would be expected to enhance variation). Bigonial breadth, measured from an area affected by the medial pterygoid and masseter muscle action, was considerably smaller in the younger sample.

Overjet, BSR, and tooth-displacement score were considered significantly different in the samples. The TPI showed sharply increased occlusal variation in the younger sample. The variance of the TPI was so effectively partitioned that variation between dietary samples was twenty times greater than variation within samples.

Those members of the Kentucky community raised on softer and more cariogenic foods, thus, were more variable from the ideal in their occlusal patterns. We did not attempt to diagnose malocclusion as such in individual cases (as always), but the correlation on a population level between occlusal variation and increased malocclusion should be obvious. Our results supplemented those from other studies showing better occlusion in the more isolated rural areas of various populations. The possible significance of dietary differences in this regard needs further study, but, whatever the ultimate cause, the study presented another among many documented examples of a sudden epidemiologic transition in

occlusal health correlated with the final developments of the industrial revolution.

The occlusal transition in the Kentucky community could not have been genetic in origin. These people constitute a single breeding population with no genetic influx; the greatest distance between birthplaces of parents or between birthplaces of a parent and a subject of this investigation was only 15.5 miles. The worsening occlusion has occurred concomitantly with a trend toward lessening inbreeding, and among individuals the correlation between PBD and the TPI was nearly zero. From this and the transition studies cited earlier, we concluded that the genetic etiology of malocclusion may be analogous to that accepted for coronary heart disease, likewise a "disease of civilization." That is, family resemblance in occlusion reveals not the presence of genes for good or bad occlusion but a genetic predisposition or susceptibility to be diverted from programmed oral growth pathway by environmental forces. Variation in this genetic predisposition would be masked by a uniformly tough, noncariogenic diet. Just as aboriginal peoples uniformly lack heart disease because of high physical activity, low psychological stress, and low cholesterol intake, so also they lack malocclusion because their diets are high in chewing stress and low in simple carbohydrates.

Caries activity, especially interproximal caries reducing space in the deciduous dentition, has been suggested as an important contributing factor to the occlusal epidemiologic transition. The literature does not support a strong caries-malocclusion relation (Hill et al., 1959; Ast et al., 1962; Murray, 1957; Lovius and Goose, 1969; Pelton and Elsasser, 1953; Hixon et al., 1962; Katz, 1977); the relation between malocclusion and periodontal disease is likewise variable (Morris, 1966). We could not control for the effect of caries in the permanent dentition of our sample study other than by measuring tooth loss. However, the soft diet subsample showed no reduction in maxillary arch length, such as would be expected with loss of space resulting from interproximal caries. The more noteworthy effect was on maxillary arch breadth, the decrease in which was shown to be solely a result of soft diet in test animals (Watt and Williams, 1951;

and many others).

Premature deciduous tooth loss has also been proposed as an explanation of increased modern human occlusal variation. Again, both positive and negative tests of this hypothesis have been reported (Lundstrom, 1955; Miyamoto et al., 1976; Baume, 1973; Paunio, 1973). We cannot reject premature deciduous tooth loss as a determinant of increased occlusal variation in our data, and it remains a strong possibility as a correlate of malocclusion.

The dietary changes tested in our study are associated with age changes, which inevitably affect the occlusion. Age difference cannot be used to explain the lesser occlusal variation in the older individuals. Age progression increases occlusal variation (Smith and Bailit, 1978; Lombardi and Bailit, 1972), as mesial drift increases crowding while any reduction in crowding engendered by tooth loss would be overcompensated by abnormal contacts resulting from drift and spacing.

Statistical differences between the different at-risk groups is reported below as it will be throughout this work. A test may be chi-square (for occurrence data), t-test (for measurement data) or analysis of variance (for multiple samples). It will be considered "highly significant" if the probability for the null hypothesis (i.e., sampled with random variation from the same statistical universe) is less than 5% divided by the number of tests based on the same sample (the Bonferroni principle), "marginally significant" if the probability is otherwise between 0.5% and 5% (roughly), and "not significant" otherwise.

We opined in closing that there is a real possibility of a relation between occlusion and dietary consistency, the outstanding implication of which is the concept of preventive steps related to food toughness. Previous discussions of prevention in orthodontics (Tully, 1973) have actually only suggested early intervention, such as with early extraction of certain permanent teeth. The functional implications of the chewing stress model indicate that the interaction between dietary consistency and occlusal development deserves more experimental research attention and support than it has received in the past. Also in need of further investigation is the involvement of premature loss of carious

Occlusal Contrasts in Traditional and Modernized Rural Kentuckians:

Statistical Significance:	Trait	Modernized	Traditional
Highly sig.	Excess Overjet	26%	3%
Not sig.	Deep Overbite	22%	19%
Marginally sig.	Crossbite	1.3 teeth	0.6 teeth
Highly sig.	BSR	1.6 teeth	0.4 teeth
Highly sig.	R/D	4.6	2.2
Highly sig.	TPI	6.9	2.3
Marginally sig.	Scissors Bite	26%	8%
Not sig.	Maxil. Breadth	39.9 mm	41.1 mm
Highly sig.	Bigonial Br.	102 mm	109 mm

deciduous teeth in the development of occlusal variation. The preventive possibilities associated with this causal process are already indicated by the trend toward fluoridation and improved caries resistance in the United States, which so far has not been accompanied by concomitant reduction in malocclusion.

Punjab Studies

As the rural Kentucky study was being completed I met with an Indian colleague, Samvit Kaul of Panjab University, with whom I had collaborated on some osteological studies. It was his idea that the Punjabi population of north-west India shows several particularly valuable characteristics for a larger-scale epidemiological study of occlusal variation.

Within a short geographic distance Punjabi communities vary from a completely urban and modern social level to immigrant laborers and farmers living outside the city in reduced circumstances. The city of Chandigarh was started

only 45-50 years ago as a capital for the new Indian state of Punjab; the urbanization process has been condensed in relatively recent times. The city's higher socio-economic stratum includes children born in an urban environment. The lowest stratum, on the other hand, is largely from rural communities and tends to retain its original dietary and residence habits. Neither group has had access to significant amounts of orthodontic care (less than 2 per cent of the upper middle class).

We determined therefore to solicit funding for a survey of occlusal variation in Chandigarh youths in relation to various environmental and social characteristics, especially diet.

The subjects were drawn from and examined at seven schools inside or within several km of Chandigarh. The schools were selected to provide a representative cross-sectional sample of roughly 1 percent of the schoolgoing population between the ages of 12 and 16. This age range ensured eruption of the full permanent dentition through the second molar, and minimal effects from tooth loss or caries. Youths missing more than one tooth or retaining more than one deciduous molar were excluded. More than 500 total subjects were included.

Subjects selected at each school were interviewed by Indian team members to ascertain age, place of birth, parents' birthplaces, education, occupation, and income. Oral examinations indicated tooth emergence and number, anomalies, periodontal disease (gingivitis scored from absent to advanced following WHO [1971] criteria), and number of carious (decayed or filled) teeth. Then the subjects were passed to a location where the occlusion (in terms of angle Class I, II or III) and dental crowding were noted, and a soft wax bite impression (Aluwax denture forms) was taken of the mouth in centric occlusion. From oral examination and wax impression, we consensually assessed the occlusal variables. As the interviews for social and epidemiological data were conducted in Hindi and recorded separately from the Western observers scoring occlusal variables, the latter were collected without knowledge of exact socioeconomic group (SEG) or other characteristics. The SEG in this society predicts the acquisition of urban,

western-influenced lifehabits almost perfectly.

Gender was not significantly associated with any occlusal variables except maxillary size measures.

Occlusal Contrasts in Urban and Rural Punjabi Youths

Significance	Trait	Urban	Rural
Not sig.	Overjet (%)	46	34
Marginally sig.	Overbite (%)	15	0
Highly sig.	Crossbite (%)	18	6
Highly sig.	BSR (%)	37	7
Highly sig.	R/D	4.0	2.8
Highly sig.	TPI	4.72	2.87
Highly sig.	Max Br. (mm)	40.3	42.0

Significant differences in occlusion were found according to SEG. Particularly, the lowest SEG, predominantly rural in origin, is set off from the higher, mainly urban SEGs in having less variation from ideal occlusal relations, and in having broader maxillary arches. Existing theories probably do not satisfactorily explain this repeated finding. There was no significant correlation of age with any occlusal variable (even the arch size measurements) within any of the SEG levels, so age differences were not a factor.

We discovered confirmatory findings by Tewari (1966), showing 45 percent "malocclusion" in the Chandigarh higher SEG contrasted with merely 25 percent for the lower SEG. These data were not interpreted in the rural-urban context. An "early mixed diet that is coarse and fibrous" at termination of breast-feeding was identified by her as a probable major determinant of the findings.

Chinese Immigrants to Liverpool

Studies of immigrants from underdeveloped areas to the Western societies have had major influence on thinking in most epidemiological subspecialities. One such population is the Chinese immigrant community residing in Liverpool. Studies on this group have been made in respect of inheritance of dental traits (Goose, 1972; Lee, 1977) and prevalence of dental disease (Goose, 1972; Lee, 1977). I therefore contacted Professors Goose and Lee about conducting the ensuing study, this time considering native versus immigrant contrasts in occlusion.

Over 100 families living in Liverpool and the surrounding districts had been visited. Some of the families were unsuitable for this survey owing to either the parents being edentulous or to the children having too few permanent teeth for adequate analysis. This reduced the number of families available for study to 100 exactly.

All parents were immigrants originating from the Canton area of China; their period in the U.K. ranged from several to over 30 years. Unlike their parents, all the children were born and bred in the U.K.

The families had been visited at their homes where upper and lower impressions were taken using an alginate impression material in disposable stock trays. The impressions were cast as soon as possible using high grade dental stone.

I scored occlusal variables for the entire collection taken in random family order, in consultation with Lee (Corruccini and Lee, 1984). While only those offspring casts were scored that had no missing teeth (congenitally or otherwise) and at least all but one permanent tooth apart from M3 fully erupted (to avoid the various effects on the occlusion), for parents up to two missing teeth were allowed. Tooth survival was good in the parental Chinese, however; some 78 offspring (average age 14) and 74 parents (average age 38) were thus finally sampled.

In terms of incisor overjet, there was a more anterior maxilla in the

offspring (a general trend of modernization). The difference is not significant statistically; the mean of about 3 mm is at the normative value for American white and black youths as stipulated by the criteria of the TPI (Kelley and Harvey, 1977). Overbite was similar among parents and offspring, both samples showing average incisor overlap of three-tenth of the height of the lower incisor.

Occlusal Contrasts between Chinese Immigrant Parents (Traditional) and their Liverpool-born, Modernized Offspring:

Significance	Trait	Modernized	Traditional
Not sig.	Overjet (mm)	3.24	3.08
Not sig.	Overbite (fraction)	0.31	0.33
Highly sig.	Crossbite (cusp)	0.27	0.16
Highly sig.	BSR (cusp)	0.49	0.25
Highly sig.	Distal R/D	1.6	0.6
Highly sig.	Mesial R/D	3.1	2.2
Highly sig.	Total R/D	4.7	2.8
Highly sig.	TPI	4.9	2.5
Highly sig.	Max Br. (mm)	57.2	58.4

Crossbite deviations of transverse occlusal relations were mostly with maxilla to the lingual side, as in most human populations. There was significantly more crossbite in offspring. BSR type was significantly more variable in the offspring, signifying greater average deviation around stipulated norms, and BSR extent was significantly larger. The abnormality was predominantly with mandibular molars too far anterior, as usual for non-European groups. Only European ancestry "whites" show predominant class II malocclusion with the

upper arch displaced.

Postcanine tooth rotation and displacement was nearly three times as prevalent in the offspring, rejecting the null hypothesis soundly. Displacement of anterior teeth likewise was significantly less in parents.

As before, our only purpose in using the TPI was to derive an overall measure of occlusal variation that compounds the individual measures. The TPI is merely an epidemiological variable here, and has no value for comparisons beyond our immediate purpose. Accordingly, defining malocclusion (in the strict sense) was avoided, referring instead to the statistical range of occlusal variation. The TPI was smaller and less variable in parents. The distributional difference in the TPI was marked. For instance, some 51 per cent of the parents but only 28 per cent of offspring fell below a value of 3.0. Although indexing of the scores is always quite arbitrary, a value of 3 or less was taken to correspond roughly with so-called ideal occlusion. At the other extreme, a TPI as large as 10 would surely qualify as malocclusion to any clinician (notwithstanding variations in assessment); 6 per cent of offspring exceeded this value in contrast with none among the parents ($p<0.02$).

Arch breadths were larger in children.

The older Chinese thus were significantly less variable from occlusal ideals in spite of their greater deleterious age changes (tooth loss, wear and migration of teeth increasing the TPI scores). There was more variance in age in the parents, in contrast to their lesser occlusal variance. There was no statistical correlation between age and occlusal variables in children. A likely etiological agent is the urban environment because genetic change can be ruled out. As discussed at length in the introductory chapter, previous studies documenting deterioration of occlusion have remarked upon the diet, particularly its physical consistency, and how that changes with Western contact. A change to a less coarse diet associated with the exertion of less force on the jaws in chewing, is likewise a key possible factor in the Chinese occlusal transition.

Narrowing of the maxillary arch is found in many industrialized groups

with rising malocclusion incidences (Davies, 1972; Corruccini, 1990). Other environmental correlates of malocclusion co-occur with urbanization. Premature deciduous tooth loss is a contributing factor, as may be periodontal disease. Chronic allergy, respiratory infection and mouth-breathing (Marks, 1965; Harvold, Vargervik and Chierici, 1973) undoubtedly are greater in the Liverpool environment of the children and could cause some effects similar to soft diet.

We concluded in summation that the change in oral function, diet and respiration resulting from urban migration is a likely cause of the deterioration in occlusion in younger Chinese.

Melanesian Populations

Another important study situation was noted, among Melanesian populations. A Harvard expedition in 1966 collected many dental casts of Solomon Island populations, including a spectrum from little affected to those highly affected by contact with Westerners. One of the largest groups sampled was the Nasioi, who have a wide distribution on Bougainville Island and were sampled primarily near coastal villages. The Nasioi at the time of casting were somewhat industrialized but sweet potatoes, taro, yams and bananas remained their staples (Smith and Bailit, 1979). Tinned food is growing in importance (Bailit et al., 1968). The Nasioi show much displacement of anterior teeth resulting from pipe-stem chewing (Smith and Bailit, 1977).

A closely related group is the Baegu. The Baegu inhabit a more inland, less contacted and affected area of Malaita.

The authors cited above who have studied these collections, mention the only previous dental-occlusal study among the Solomon Islanders, that done on New Britain inhabitants by Janzer (1927). The Gazelle-Peninsula of north eastern New Britain was visited in the 1890's by R. Parkinson (1907), who excavated a pre-colonial and mostly pre-contact cemetery of the inhabitants. Parkinson also documented many physical and ethnographic similarities among New Britain Islanders and others of the Solomons including specifically North

Bougainville. Taro and bananas were the staples of these people (Parkinson, 1907) but many tough items supplemented the diet: cabbage, spinach, breadfruit, and wild pig. Of Parkinson's recovered skulls 424 were sent to the Staatliches Museum fur Volkerkunde, Dresden, while another 182 found their way to the Museo Nazionale di Antropologia in Florence.

Janzer's (1927) description of the Dresden collection included observations on alveolar prognathism, arcade shape, and the form of edge-to-edge incisor bite, but it provides little data on actual malocclusion except mention of one Class II case and several apparent crossbites. The material culture exhibit in the Museo Nazionale di Antropologia shows the New Britain pipes had very short stems or mere holes for the mouth for smoking -- these were obviously not chewed upon habitually as was the case among modern Nasioi and Baegu. Some craniometric description of the Florence skulls is given by Lombardi, Pardini and Pardini (1976).

There is very little tooth loss or caries in all these collections. The transition from traditional to Western influenced diet among these closely related populations allows one to examination of effects of dietary transition on jaw growth and occlusion.

With the active assistance of Howard Bailit and especially Ed Harris, I used a "grab" sample from the collection of Solomon Island populations (119 Nasioi and 87 Baegu). Records for the Nasioi provided exact age, sex, date of casting, and family. Sex was estimated for the New Britain skulls. In collaboration with Elsa Pacciani, the paleobiologist with the superintendency for archeology of Tuscany, and with the help of Ingrid Wustmann of the Dresden museum, I similarly scored 144 crania of the New Britain collections.

Gender difference was absent in every occlusal variable, as has been found in essentially all previous studies. The arch dimensions were significantly larger in males in the mandible only. The sex distribution did not differ from random sampling, and the sexes were aggregated into single samples for each group. Age differences were assessed by correlation in the Nasioi sample for

which age at time of casting was recorded. There were no significant correlations, with the exception of mandibular and maxillary lengths. These dimensions tended to decrease by 0.26 mm per year, undoubtedly through interstitial attrition. Arch breadths were not similarly affected. In my other studies, lack of correlation between occlusal variables and age has been consistently found between ages 12-30 (a range which encompasses at least 80 percent of the samples in the present study).

In terms of basic statistical results, both upper and lower arch length dimensions showed a sequence of smaller values in less contacted peoples, which undoubtedly relates to greater grit and attrition in those with more traditional diets. Although the New Britain group had shorter arches, they were significantly broader in the maxillary arch. Maxillary broadness is one component of a strong and healthy masticatory apparatus, and maxillary narrowing inevitably accompanies worsening dental occlusion in many ethnic populations when their diets modernized. Below, results are abbreviated as a comparison of the more

Occlusal Contrasts between early New Britain Crania and Modern Nasioi:

Significance	Trait	Modernized	Precontact
Marginally sig.	Overjet (mm)	2.6	2.3
Highly sig.	Overbite	0.3	0.15
Highly sig.	Crossbite	0.18	0.11
Highly sig.	BSR	0.30	0.14
Highly sig.	Distal R/D	1.08	0.24
Highly sig.	Mesial R/D	2.61	0.94
Highly sig.	Total R/D	3.69	1.18
Highly sig.	TPI	3.25	1.75

modernized Nasioi to the precontact New Britain sample.

Incisor overjet was smaller in New Britain, coming slightly closer to an edge-to-edge incisor bite that characterizes aboriginal people. The difference with these samples could be ascribed to random error however.

Incisor overbite, on the other hand, was statistically significantly smaller in New Britain, and less variable. This again relates to the proper ancestral state of edge-to-edge occlusion. The fact that variance of the traits was also less in New Britain indicated that fewer individuals had phenotypes departing extremely from the norm.

Posterior tooth crossbite type differed, the Nasioi having a more buccal deviation of the lower molars when crossbite occurred. Thus the more Western-influenced group departed significantly more from orthodontic ideals. New Britain showed much less extent and variability of crossbite.

Buccal segment differences in type were within confidence limits for sampling error, but New Britain was much less variable in this trait. This showed up in BSR extent, which averaged half as much in New Britain as in the Nasioi or Baegu.

Distal tooth rotation and displacement score is an important variable for comparison, because it is not affected by the considerable anterior tooth distortions brought about in these people by pipe-clenching. There were hardly any crowded, impacted or malaligned distal teeth in the New Britainers, a clearly significant difference from Nasioi and Baegu. Baegu had a smaller score than Nasioi, again in keeping with the gradation of refined products in the diet, but this amount of difference could have occurred between 5 percent and 10 percent of the time in random samples from the same population. Anterior tooth displacement was also less in New Britain, but this has a direct explanation in the pattern of pipe-use described above.

The TPI (reflecting combined occlusal variables) was markedly smaller and less variable among early New Britain inhabitants. Some very broadly interpreted implications of the distribution of TPI scores can be attempted. For

instance, following the trend toward increasing Western influence over these people, some 79 percent of the New Britainers had TPI score below 3.0 (ordinarily signifying nearly classic ideal occlusion), compared to 61 percent for Baegu and 57 percent for Nasioi. Only one percent in New Britain had a score above 7.0 (signifying severe occlusal handicap), compared to 10.3 percent of the Baegu and 12.6 percent of the Nasioi.

Thus, the prehistoric Melanesians, who subsisted on less refined diets, were significantly less variable from occlusal ideals. Almost the only obvious possibilities as causal agents are dietary change and genetic difference.

Genetic admixture with colonists could only be an extremely slight factor in the contacted populations. There is no evidence over many villages across Bougainville Island of systematic differences in occlusion (Smith and Bailit, 1977) despite regular changes in geographical genetic relatedness.

The change in oral function resulting from dietary change, on the other hand, we interpreted as the likely cause of some of the deterioration in occlusion in more recent Melanesians (apart from the lateral anterior tooth distortions). An interesting individual (Corruccini and Macchiarelli, 1987) figured into our thinking on this point. With the strenuous oral regime of use, we felt that even occlusions that started off wrong for some reason tend toward congruency. "Occlusion should always be evaluated on the basis of functional potential rather than simply on the basis of the common morphologic classifications often used in orthodontics. For instance, a cross bite may represent optimal functional occlusal relationships in a person with a small maxilla and a large mandible. The absence of pathologic manifestations and the presence of unhindered functional movements are factors which are of much greater importance in the evaluation of occlusion than the standard of cuspal interdigitation used as a basis for the diagnosis of malocclusion. Morphologic and static classifications of malocclusion have a much greater esthetic than functional significance" (Ramfjord and Ash, 1971). Berry (1976) is even blunter: "now, with malocclusions increasing and the need for masticatory efficiency eliminated by civilization, it is difficult to regard

our teeth as anything but conventional decorations. The present pattern of much clinical dentistry emphasizes not only the conservation and preservation of the teeth but in such a way that cusps are often fully restored, and the tooth with gross attrition is regarded as being pathological. We are teaching students to maintain and rebuild occlusions to designs for which there is no physiological basis whatever."

Studies on Pima Indians

The Pima are a reservation Amerindian group from whom the late and revered Albert Dahlberg and associates collected about ten thousand dental casts between 1948-1970.

These casts have been studied for evidence of odontometric heritability (Potter et al., 1968, 1983) and differences with fluoride variation in well water. An initial study (Irie and Dahlberg, 1967) presented some occlusal data and concluded that permanent dental occlusion did not change between ages 12-16. Extending this comparison to older Pima offered the advantage of assembling occlusal change occurring with dietary change. The Pima traditionally relied on a heavy bean dietary staple grown in their own gardens. Tortillas were also a favorite, the indigenous corn long-cooked and stone-ground. More recently, approximately since 1950, the Pima do not grow their beans, as the all-important country store became the source of commercial beans and processed corn meal. In post-war years the children started receiving good government sponsored dental care, while this has not reached the adults.

The dietary transition, considered to be a factor in the dental occlusal epidemiological transition, was fairly subtle in this population, entailing a change from homegrown to commercially purchased versions of the same foods with the latter more refined and more cariogenic. The effect of this and other changes on the Pima occlusion were the subject of the study described below, conducted together with Rosario Potter and Al Dahlberg.

A stratified cross-sectional sample from the cast collection was used, in

order to represent the various collection times (Corruccini, Potter and Dahlberg, 1983). Records provided exact age, sex, date of casting, and family. If yearly serial casts of the same individual were available, the most recent one (when the individual was the oldest) was used. Only those casts were scored that had no missing teeth (congenitally or otherwise), and at least all but one permanent tooth fully erupted (excluding consideration of M3) such that the full permanent occlusion alone could be assessed. For older individuals (over age 40) it was necessary to allow up to two missing teeth to improve sample size. There were as many as six yearly casts over a nine-year period for younger Pima. Most of the casts of older Pima came from the earlier (1949-1957) series of collections, such that the effects of aging on dentition could be canceled in the comparison with younger Pima, many of whom were cast between 1963-1968. Average age was 14.7 for young and 27.7 for old Pima. We established lack of correlation of occlusion and age (between 11-30 years) in the younger sample, similar to Irie and Dahlberg (1967) and also true of the much larger samples of Kelley and Harvey (1977).

Taking 1950 as roughly the cut-off time for traditional Pima diet and considering that dietary effects on permanent occlusion probably act up to 12 years of age, when deciduous premolars (to dentists, these are deciduous molars) are replaced and M2's are erupted, we would expect individuals 30 or younger (by 1969, the last year of casting and the baseline date for this study) to show occlusal effects from dietary change and those over 30 (by 1969) to have developed occlusally during the traditional diet period. The former group was defined as the younger sample and the latter group as the older sample. Every case had two chronological data, the number of years ago born from 1969 (YAB) and actual age at casting. Thus the younger sample consisted of 247 Pima with YAB of 30 or less, and there were 67 older Pima with YAB of 31-50.

Every variable was tested for gender difference, and none was found with the exception of the metrical arch variables, to agree with Irie and Dahlberg (1967) for Pima, and Kelley and Harvey (1977) for large American samples. The

exception was BSR type, where the females showed significantly less deviation from the norm so that this variable was compared between young and old samples with the sexes separated.

Occlusal Contrasts in Traditional Parental versus Modernized Offspring Generations of Pima:

Significance	Trait	Modernized	Traditional
Not sig.	Overjet (mm)	3.3	3.0
Not sig.	Overbite	0.3	0.3
Not sig.	Crossbite	0.3	0.2
Highly sig.	BSR	0.4	0.2
Highly sig.	Distal R/D	1.0	0.6
Not sig.	Mesial R/D	3.2	3.0
Marginally sig.	Total R/D	4.2	3.5
Not sig.	TPI	4.7	3.9
Marginally sig.	Mand Br. (mm)	55.7	56.5

Basic statistical results, in terms of incisor overjet, were that there was a more anterior maxilla in the younger sample. This reflects a general trend of modernization (an edge-to-edge incisor bite actually being the norm in aboriginals). The difference was not significant statistically; the mean of about 3 mm is at the normative value for American white and black youths as stipulated by criteria of the TPI (Kelley and Harvey, 1977). However, older Pima were less variable. Overbite was quite similar among old and young, both samples showing average incisor overlap of three-tenths of the lower incisor's crown height; again this is within the orthodontic norm.

Crossbite deviations of transverse occlusal relations occurred mostly with maxilla to the lingual side, as in all human populations. There was slightly more crossbite in younger Pima, but the difference was not significant.

BSR type and extent both were significantly more variable in younger Pima, signifying greater average deviation around stipulated norms, and mean BSR extent was significantly larger in the younger Pima. The abnormality was predominantly with mandibular molars being too far anterior, which is usually the case for non-Western groups. Only Western European-derived whites tend to show predominant "Class II" malocclusion with the upper arch displaced anteriorly. When the sexes were separated, differences were still highly significant for males; only eight of 48 older Pima had cusp-to-cusp or worse BSR anomaly, compared to 96 out of 239 younger males. There was no difference in the much smaller female sample.

Postcanine tooth rotation and displacement was nearly twice as prevalent in the young sample with a highly significant difference between the groups. Displacement of anterior teeth was also less in the older Pima raised on traditional diet, but more variable (a few older Pima had very severely crowded incisors due to mesial drift). Although 16.4 percent of older Pima showed perfect anterior tooth alignment versus 10.6 percent of the younger, the difference was not significant.

The TPI was smaller, though not significantly so, and less variable in older Pima. Again age affected results as some older cases had tooth loss, attrition and mesial migration of teeth that increased TPI scores, while many others maintained remarkably good occlusion. Thus there was a distributional difference, 13 older (19.4 percent) as opposed to 19 younger (6.9 percent) individuals having perfect TPI scores, i.e., no appreciable deviation from ideal anatomical occlusion in any variable. The difference is significant at p=1 percent.

The TPI, again, while a valuable method of combining different occlusal variables, carries unfortunate connotations of clinical assessment. Another approach is to combine occlusal variables by canonical variates analysis to find

whether a statistical combination that most effectively discriminates older from younger samples is significant. This turned out to be the case.

Arch lengths were significantly greater in younger subjects, while the difference in arch breadths (larger in the older) was less. This is the maxillary shape trend found in all surveyed industrialized groups with rising malocclusion incidences, that is, relative palatal narrowing (and deepening), relating to the "maxillary collapse syndrome" frequent in American youths (Kelley and Harvey, 1977) and to general facial narrowing (Davies, 1972). The relative effect on length and breadth also related to aging effects: mesial tooth drift, accentuated by some tooth loss and interstitial attrition, reduced arch length with age. Correlation between maxillary arch length and YAB, with effects of chronological age removed by partial correlation, showed zero rather than significantly negative correlation. Among arch measurements, only maxillary breadth correlated significantly (negatively) with TPI.

A Peripheral Topic: Premature Deciduous Tooth Loss

The effect of eruption sequences and premature deciduous tooth loss (PDTL) has caused speculation in discussions of malocclusion etiology. Early shedding of a deciduous tooth, premature eruption of its successor, and an excessive time lag between those two events are interrelated factors in occlusion. Earlier epidemiological results have been quite inconclusive regarding the PDTL factor (Lundstrom, 1955; Miyamoto et al., 1976; Baume, 1973; Corruccini et al., 1983; Anderson et al., 1980). The longitudinal series of Pima casts allows a unique test of PDTL influence in an occlusally variable population. Teeth lost prematurely due to advanced caries could be identified and considered as a separate category from prematurely lost teeth that have no detectable carious involvement.

A PDTL score was devised to record noticeable degrees of deciduous tooth exfoliation occurring prematurely from the normal schedule. Due to maturational variation and secular trends (the young sample erupting second

molars a year earlier on average than the older sample), it is important to compare tooth succession by an absolute time schedule and by a relative scale that compares a given tooth to others which ordinarily should be shed earlier or later.

A deciduous tooth class lost prematurely by one year counted as one point, including maximally 1/2 point counted for a tooth erupting one year earlier than either a normally synchronous antagonist or an antimere. Since four categories are recognized and one must be held constant within the sequence against which to gauge the others, this PDTL score ranges from zero to three by 1/2-point increments.

The role played by PDTL in creating occlusal variation could be studied only in the younger Pima subsample where longitudinal casts from about ages 6-12 were available; there were about 190 such cases of serial records for individuals.

For occlusal variation in relation to PDTL in this younger subsample only, an analysis of variance showed no significant differences. The highest F-ratio for any measurement or occlusal variation was 1.85 for distal tooth displacement (.10>P>.05). PDTL scores in the upper half of the range had somewhat higher R/D counts. The only other variable with a relatively high partitioned variance was overbite (F=1.69), with the biggest increase in occlusal variation again occurring with PDTL scores of 2 or above. The smallest F ratios were associated with arch measurements.

Correlations between PDTL and occlusal variables were examined. All correlations were very nearly zero except those for overbite (r=0.15) and R/D (r=0.14), both borderline significant correlations indicating slight increase in vertical incisor overlap and in rotated teeth with more PDTL teeth. One major reason for considering PDTL important to the increase in occlusal variation with urbanization is the possible relation to caries which, if serious, could cause early tooth loss. For cases with macroscopic carious destruction in deciduous teeth (n=30), a notation was made and these were later removed from the sample (non-carious n=160). Among the non-carious cases, correlations were consistently

larger (though the difference is slight) between PDTL score and occlusal variables than in the carious cases, where PDTL correlations were all near zero. Whatever the relation between tooth succession and occlusion may be, it seems to be unconnected to caries.

In conclusion, the older Pima, raised less recently on less refined diets than the younger Pima, were significantly less variable from occlusal ideals in spite of their greater deleterious age changes. The most obvious suspects as casual agents for this difference are dietary change and genetic change.

Genetic admixture could have only been extremely slight in the intergenerational time. Orthodontists have often invoked the notion that "race mixture" causes malocclusion (refuted by Chung et al., 1971; Horowitz and Osborne, 1971), in that one may inherit jaw size from one parent which may be disharmonious with tooth size inherited from the other, or malfitting upper and lower jaw profiles from two different ethnic predecessors. Other than this notion, it could also be theorized that the white U.S. population has a higher frequency of deleterious alleles for occlusion through accumulating mutations, and that admixture with whites raised the Pima malocclusion prevalence. However, these mechanisms are both very unlikely, due to the short time involved and the lack of evidence of increased genetic flow. What little admixture that has been documented among Pima seems more toward Mexican mates.

It could be that biased sampling operated in the older group, since only individuals with nearly intact dentitions were scored and these could have had better occlusion than excluded cases when younger. Only an extended longitudinal design could answer this; we hope the facts that most cases age 40+ were excluded, and that the different sample average ages of 15 and 28 span a period of little occlusal change, mitigate this problem.

From our data, we concluded that the change in oral function resulting from dietary change is a likely factor in the deterioration of occlusion in younger Pima. Physical anthropologists and geneticists remain suited to test this theory in various rapidly urbanizing societies.

Bengali Youths

To extend the rural/urban model in a rapidly developing society, using a detailed interview protocol for these factors, the Bengali population in and around Calcutta, India was surveyed by Ahmed Chowdhury (a Ph.D. recipient from Southern Illinois University) in collaboration with myself.

A total of 150 male adolescents aged 13-17 yr were randomly drawn from urban and rural communities (75 each) for survey of occlusal variation relative to socio-environmental factors. Youths having permanent teeth only were included in the sample; those missing more than one tooth due to decay, or retaining any deciduous teeth were excluded from oral examination, in order to restrict analysis to the relatively stable adult occlusion. The population in both rural and urban areas of West Bengal are deprived of even minimal orthodontic care. The urban sample was selected from a model high school in the city of Calcutta where children are from relatively affluent families. Today Calcutta is one of the highly industrialized urban centers of India. Rural subjects were drawn from a school in a farming community of the district of Hoogly.

Eight of the urban youths had spent more than the first half of their life in rural (village) rather than urban environments: these urban migrants were added to the rural sample which then totaled 83 compared to 67 urban youths. Ethnic composition of the rural sample was 10 Muslims, 2 high-caste and 71 middle-low caste Hindus, while 8 Muslims, 7 high-caste and 52 middle-low caste Hindus made up the urban sample. The distributional difference among these socially (but probably not genetically) isolated components was not significant ($p>.20$).

Subjects were interviewed in Bengali through a structured schedule and information obtained on usual dietary intakes, extended habitual or infrequent thumb-sucking, place and duration of residence, economic (approximate monthly income), educational and occupational status of their family heads, and migrational status. A scale was derived for chewing stresses provided by diets (in terms of consistency or toughness), and a score was obtained from the summation of values of scaled food items in the diet. High scores were given for habitual raw

sugarcane chewing, green mango and guava, unprocessed country cakes and paratha (fried bread), and certain resistant vegetable preparations, while more processed staples such as chapatis and softer fruits resulted in lower scores.

The status of subjects in relation to occlusion/malocclusion was subjectively determined on the basis of oral examination of dental malalignment, crowding/rotation, irregular placement, impaction, and displacement/malpositioning. The number of these conditions was summed to quantify the syndromes (thus ranging from zero to 5).

Occlusal Contrasts: Traditional Rural versus Modernized Urban Bengali Youths

Significance	Trait	Urban	Rural
Highly sig.	Overjet (%)	70	40
Marginally sig.	Openbite (%)	9	1
Highly sig.	Total R/D	4.7	2.8
Highly sig.	Malocclusion (%)	70	48
Highly sig.	Syndrome Sum (count)	2.1	1.4

Sagittal incisor relationship was quantified from a score of 2 (extreme maxillary overjet) to -1 (mandibular overjet).

The occlusal syndrome sum was much higher in the urban sample, as was the tooth displacement score. Incisor overjet was significantly larger (significantly more anterior maxillary incisors) in urban subjects, but this in itself does not establish worse occlusion. The variation of scores is the parameter reflecting probability of abnormal low (mandibular prognathism) or high (anomalous overjet) scores, and did not differ significantly ($F=1.06$). The frequency of malocclusion, subjectively diagnosed from any of the individual

indicators, was significantly higher in the urban sample. The more objectively defined (and clinically serious) openbite trait, because of its overall infrequent expression, was only marginally significant according to the Fisher Exact Test.

There was considerable overlap of individual scores between the two samples. This did not indicate a basically similar pair of variable populations which yield a statistically significant result merely because of large sample sizes. The point is best illustrated using tooth displacement score, the most important occlusal variable. One can arbitrarily index any value at which "normal" variation leaves off and "malocclusion" is defined, and this point will naturally fall to the positive tail of the frequency distribution where there were many cases from the urban sample. For instance, a score of 10 or more is considered "severe occlusal handicap with orthodontic treatment considered mandatory" (Kelley and Harvey, 1977). At this extreme level probably no two clinicians would disagree about a diagnosis of "Malocclusion." Some 27 percent of urban youths exceeded that score compared to 1 percent of rural.

Most of the socioenvironmental variables correlated significantly with several occlusal variables, but they also correlated strongly with the rural/urban provenience. To extricate independent shared variance with occlusion, partial correlations between socioenvironmental and occlusal variables were calculated with rural/urban effect held constant. The following variables had no significant correlations: rural birth (implying possible genetic segregation by distance), years spent rurally prior to urban migration (average 1.2 yr urban and 14.4 yr rural), occupation of family head (predominantly higher and middle-rank professionals in urban, and semi-skilled workers, agriculturists and day laborers in rural subjects), education of family head (mostly graduate professional training in urban, and partial to no high school training in rural subjects), and thumbsucking habit. Thumbsucking would theoretically be expected to affect incisor relation and openbite traits. Age was significantly negatively correlated with syndrome sum; younger subjects tend to have higher scores even though there was no correlation with the five individual traits (i.e., occlusal status) making up that sum. This

result is difficult to explain since all subjects had erupted permanent teeth through second molars prior to being studied. It does not help to explain the rural/urban difference in occlusion since average age was not significantly different, and actually there were more subjects age 14 or less in the better-occluded rural sample.

The dietary consistency score was significantly correlated with occlusal status, syndrome sum and tooth displacement score. This etiological factor thus showed relatively consistent (although small) common variance with occlusion traits independently of provenience, suggesting the influence is common to both rural and urban sample subdivisions.

These results confirmed the generality of highly prevalent malocclusion and occlusal variation in urban, modernized, industrialized populations compared with predominantly good occlusion in rural, traditional populations (Corruccini, 1984, 1991). Among several identified factors, the consistency or toughness of dietary items, reflecting daily masticatory stresses that presumably affect oral-facial development, contributed the most additional shared variance with occlusal variation beyond that explained simply by the rural/urban distinction. Other urban environmental factors such as premature carious deciduous tooth exfoliation and oral breathing could also have been involved.

World Populations

In a review study (Corruccini, 1984), the comparison was expanded to several other combinations of human samples. One such contrast involved two prehistoric American Indian skeletal samples. One, from Dickson Mounds, Illinois, consisted of 66 skulls from the Woodland (horticultural) period to the Mississippian (agricultural) period. The other was from the Libben late Woodland swampside site in Northern Ohio, 73 dentitions of a group with a diet high in animal protein and unprecedented caries rates for an unindustrialized population (for unknown reasons). These particular samples were chosen both to expand the sample of truly aboriginal cultures and also to contrast occlusal effects of early

agriculture, on one hand, and of endemic caries on the other. The Libben series shows 42.8 percent carious teeth, about 5 percent of these being interproximal caries of the sort that could lead to tooth movements and rotations (personal observations of author and R.P. Mensforth). Otherwise, the diet of these Amerinds was of the unprocessed nature documented by Hinton (1981).

We (Potter et al., 1981) reported occlusal statistics for 328 white United States youths representing 164 twin pairs (mostly middle class). Dental casts had also been collected for some of the parents -- 107 individuals in all. The mean TPI computed for the youths was 5.01, equal to the 5.0 reported for Kelley and Harvey for 22,000 American youths of the same age and generation. Comparison of these with the parental generation in this instance does not involve so much environmental change, both generations being essentially completely industrialized. Therefore, it is possible to make a more controlled assessment of the type and amount of statistical effects accruing with chronologic age differences alone and associated tooth loss and migration, removal of edentulous specimens from samples, attrition, etc.

For the present study the great advantage of also collecting data on American blacks was recognized. For one thing, Kelley and Harvey (1977) noted that southern black female youths showed significantly better occlusion than the other sample components (regional, racial, or sexual) of American youth, all the other components not differing significantly among themselves. This recalls the rural-urban dichotomy characterizing previously discussed occlusal contrasts, if southern blacks are significantly more rural than northern blacks. Furthermore, it has been informally noted among anthropologists and forensic specialists that earlier blacks and those from southern regions of a more rural nature have a different distribution of occlusal characteristics. In fact, the better occlusions and broader arches in southern black adults have been used as an accurate criterion of forensic identification (C. O. Lovejoy, personal communication). Occlusal data largely comparable with mine were gleaned for modern, predominantly urban black youths while the earlier black sample, broadly analogous to the parental

white sample, was taken from two well-known medical school collections of cleaned dissecting room cadavers: Terry and Hamann-Todd collections. Some 129 skulls were scored; the average birthdate was about 22 years earlier than the white parents; the origin was 86.5 percent deep Southern United States in adequately documented morgue specimens, although all had migrated to the Cleveland and St. Louis areas before death. I would argue the little extra admixture with whites could have accumulated between older and younger black samples over these 45 years, perhaps a maximum of 5 percent over an existing 15 percent admixture.

Occlusal Contrasts in Various Populations

Significance	R/D Trait (score)
Highly sig.	Pima (4.7) vs. Prehistoric Amerinds (1.9)
Highly sig	U.S. Youths (4.6) vs. Primates (0.5)
Not sig.	U.S. Youths (4.6) vs. Parents (4.3)
Highly sig.	U.S. Black youths (4.5) vs. Parents (3.2)

For vertical incisor overbite, Blacks showed much lower means than whites, closer to the edge-to-edge condition. Both overjet and overbite illustrate the often noted tendency for Western orthodontic concepts of ideal or normal status to be highly influenced by the variation seen in Western populations, which is fairly different from that shown in other populations.

The results, comparing just the R/D variable for convenience's sake, illustrate the recency of the epidemiological transition in occlusion in these three population groups. Modern Pima have a great deal more malaligned teeth than even heavily carious prehistoric amerinds; American Blacks, with recent largescale transition from southern rural to northern urban concentrations, also have suffered

a recent deterioration in tooth alignment. American middle-class whites, on the other hand, having enjoyed a relatively constant affluence for several generations now, do not demonstrate the transition. That is why this group, the one that receives the lion's share of orthodontics, is so unrepresentative of the significant factors of change in human occlusion.

Sedentizing Australian Aboriginals

The Australian Aboriginal has long been of interest in dental anthropology (Campbell, 1925) and orthodontics (Begg, 1954). Begg based a classic theory for the etiology of malocclusion on his observations of dental variation in Aboriginal skulls. This stated essentially that adequate arch space in Aboriginals resulted from reduction (through interproximal attrition) in the amount of tooth material needing accommodation. The contemporary orthodontic emphasis on extracting teeth (usually premolars) in order to make room in the arches for the permanent dentition is sometimes said to owe heavily to this idea.

Theories for the high prevalence of malocclusion in westernized populations based on interpopulation variances and comparisons, would do well to include consideration of acculturated versus nomadic Australian Aboriginal populations.

Significantly, Campbell (1925:82) speculated:

> "In the preparation of his food, the (Australian) native undoubtedly adopted only the crudest of methods, and those which are very far removed from the so-called refinements of modern cooking. Whatever preparation his food did get, it was undoubtedly in such a condition when eaten as to require very vigorous masticatory effort. The whole structure and condition of the aboriginal's jaws and teeth point very clearly to this.... all dental diseases are practically limited to individuals of advanced years, and on the whole the aboriginal possesses a very efficient and perfect dentition. This is in marked contrast to the dental condition of present-day aboriginals

who have been brought up under modern civilized food habits. In the latter it is not unusual to see advanced caries and 'broken down' dental arches in natives by no means advanced in years.... I have on various occasions been informed by intelligent old natives that they considered the bad teeth of present-day blacks due to the food of white people.... The problem of the causes of dental disease and irregularities is not one calling for long and intensive research, but a simple lesson.... If the masticatory system be supplied with a diet which imposes upon the teeth and jaws the task of functioning in a thorough and physiological manner, then the tendency will be for the maintenance of normal and healthy conditions. Modern dietaries and methods of food refinements do not provide for this, thus the present-day oral and dental degeneracy. The problem in hand seems to consist in finding ways and means of educating the masses to appreciate the facts which are really quite obvious."

A collection of dental casts obtained from Australian Aboriginals living at Yuendumu, situated about 285 km northwest of Alice Springs in the Northern Territory of Australia (Campbell and Barrett, 1953; Barrett, Brown and Fanning, 1965), has enabled a more controlled investigation of occlusal changes following adoption of western life habits, including sedentism and refined foods.

The Yuendumu settlement was established in 1946 in the proximity of bore water supplies to provide a central area for provisioning Aboriginals who had left their tribal areas or nearby major cattle stations. The population is almost free of non-Aboriginal admixture and, apart from some Pintubi people, predominantly of the Walbiri tribe. This setting offers a stable genetic background against which to measure the effects of environmental change.

The older generation was arbitrarily defined as those born before 1937, thus having most of their dental development completed before Yuendumu's founding. They were mostly traditional nomadic hunters and gatherers: "many foods were eaten raw, particularly plant and plant products. Flesh foods received

minimum cooking either on an open fire or, in the case of larger game, in a crude earth oven of hot sand and ashes. Seeds were ground between stones to a flour which was mixed with water to form a thick paste before cooking over hot ashes. The Aboriginals used no eating utensils but relied almost entirely on teeth and hands" (Brown, 1985). On the other hand, the Aboriginals who grew up at the settlement were provisioned by the government, rations consisting mostly of flour and sugar and a midday hot stew. Considerable supplementary hunting still was conducted by the parental generation into the mid-1950's. Cash income soared in the mid-1960's, and expansion of the Yuendumu canteen occurred along the lines of supermarkets in modern cities. The Aboriginals who are now largely self-governing, have become increasingly dependent on Western food and food habits with corresponding increases in western-style problems of ill health and degenerative disease.

Caries experience of the Yuendumu people was extremely low by world standards, probably at least partly because of the high fluoride content (up to 1.8 ppm) of the drinking water at the settlement (Barrett, 1956). There is a tendency for relatively late premolar eruption (Barrett, 1957a, 1957b; Barrett et al., 1964; Brown, 1978; Brown et al., 1979), little premature deciduous tooth loss, and quick succession of permanent teeth (Brown, 1985). Dental stone casts of these people were collected between 1951 and 1971, with many individuals having rather extensive longitudinal series. Older individuals were only included if born no later than 1936, at least all but one of the permanent teeth (excluding M3) were present on at least one side, and if attrition had not advanced so far that normal occlusal relations could not be assessed. The group totaled 48 individuals. A matching sample of 48 younger (post-1936 birth) casts was obtained from the most recently collected material in order to maximize chronological difference between the two samples. Where longitudinal series were available, the earliest (youngest) cast was studied for an older individual and the latest (oldest) used for the younger sample members -- thus lessening the 27 yr difference between birth date to roughly 17 years between the actual difference in developmental age at

casting. In no case were less than full-arch occlusions studied for the younger sample, with permanent dentition from I1-M2 fully erupted, and minimum age at casting of 14 yr. It is necessary to have dentally adult specimens for the comparison because this population shows considerable crowding changes and poor predictivity of ultimate crowding from the late mixed dentition stage (Barrett, 1957a, b; Sampson and Richards, 1985). Both samples consisted of 25 males and 23 females.

To extend the comparison of occlusal variation, another sample of 48 dentitions, complete on at least one side, was studied among Aboriginal crania curated in the South Australian Museum. As far as known these were pre-contact individuals, recovered from a diverse continent-wide scattering of sites. No sex or age determination was undertaken other than to establish eruption of the permanent dentition. Intertribal and interregional physical variation is large in Australian Aboriginals (Brace, 1980; Smith, 1982), so the precontact cranial comparison incorporates much variation beyond the environmental contact construed for Yuendumu.

Finally, a sample of 35 individuals was taken from the Haasts Bluff cast collection (Heithersay, 1960). The Haasts Bluff settlement is situated about 150 km south of Yuendumu, and individuals from the two areas visit periodically. Haasts Bluff casts, collected in 1956 from newly-settled individuals mostly of Pintubi tribe, should represent an environmental milieu largely comparable to that of the older Yuendumu sample.

For every occlusal variable and arch dimension, sex difference was tested within the older and younger Yuendumu sample subdivisions, and correlation with birth year was calculated. Parametric descriptive statistics were determined for each sample following the testing for sex and age effects. The t-test for mean differences between pairs of samples was supplemented by Kolmogorov-Smirnov or chi-square tests because most variables were not normal in distribution (specifically, many were positively skewed); one-tailed significance at $p=0.05$ level was only indicated if both parametric and non-

parametric tests were significant. The F-test was employed to compare variances. The Yuendumu younger-versus-older contrast is of primary interest here; Haasts Bluff and precontact crania were compared against the older Yuendumu component since, given the pattern of variances and sample sizes, a significant result against older Yuendumu would also automatically achieve significance against the younger. There were several parent-offspring and half-sib pairs included in the cast samples, introducing small and perhaps counter-balancing amounts of Type I and Type II statistical error.

Arch length and arch breadth showed both sex difference and significant age-correlation. No other variable (notably the breadth/length ratio) showed either bias. These occlusal variables generally fail to demonstrate sex difference in humans (Corruccini, 1984) whether or not tooth size differs. Therefore sex difference was generally assumed absent for all between-sample comparisons with

Occlusal Contrasts between Precontact and Modern Settled Australian Aboriginals

Significance	Trait	Settled	Precontact
Highly sig.	Overjet (mm)	3.29	1.88
Highly sig.	Overbite	0.31	0.06
Not sig.	Crossbite	0.19	0.16
Marginally sig.	BSR	0.28	0.11
Highly sig.	Distal R/D	0.79	0.23
Highly sig.	Mesial R/D	2.19	1.23
Highly sig.	Total R/D	2.98	1.46
Highly sig.	TPI	3.35	1.96
Highly sig.	Max Br/L (ratio)	1.40	1.50

the exception of arch size.

Incisor horizontal overjet increased significantly from the older to younger Yuendumu samples. Overjet did not correlate significantly with year born within the older Yuendumu subjects, suggesting the difference was not merely age-related among the samples. Overjet did not differ in variability, signaling no increase in occlusal variation in younger Aboriginals that would overlap with excessively high or low values. The mean overjets were remarkably similar to Beyron's (1964) Yuendumu figures. Precontact crania showed a significant further reduction in overjet; overjet variation however remained fairly homogeneous.

Mean incisor vertical overbite, similarly, increased from older to younger Yuendumu generations but remained constant in variability. Thus it cannot be said that the younger individuals were in any sense worse occluded. Haasts Bluff was again very similar to Yuendumu contemporaries but the early cranial sample showed both mean and variance significantly reduced for overbite, much more closely approximating the classic edge-to-edge incisor relation of earlier hominids.

Posterior crossbite scores reflected a unique type of occlusion, with maxillary teeth positioned slightly buccally on average (i.e., negative scores) when casts were placed in closest approximation to maximum intercuspation or centric occlusion. This is the "alternate intercuspation" or "X-occlusion" previously described in Yuendumu Aboriginals (Barrett, 1953, 1969; Beyron, 1964; Brown et al. 1986) which, in its classic morphological form, yields a crossbite type score of about -0.2.

The older Yuendumu sample, while showing many negative crossbite scores, had a positive mean score which differed significantly from all other samples. Thus, while the greater attrition and mesial physiological drift occurring in older individuals undoubtedly resulted in some maxillary breadth reduction in all samples, with posterior teeth becoming progressively more anteriorly and lingually situated, only in the older Yuendumu sample did this lead to buccal

crossbite exceeding lingual displacement. Haasts Bluff and precontact crania also were significantly less variable in crossbite than Yuendumu, and Haasts Bluff showed significantly less mean divergence from the centric normal score of zero in crossbite extent.

BSR type was quite homogeneous among the three contemporary Aboriginal samples, but significantly less variable in precontact crania. For BSR extent, the null hypothesis was barely retainable among Yuendumu samples ($p<.06$).

Postcanine R/D count was significantly less in older Yuendumu contrasted with younger. Here we had the first instance of an occlusal difference that actually signified "better" occlusion in the traditional Aboriginals. The older Yuendumu R/D of anterior teeth was also somewhat less, contributing to a significantly lower total R/D score. Haasts Bluff and precontact crania were fairly concordant with older Yuendumu on this score.

The Treatment Priority Index (TPI) is a useful summary of occlusal deviations from the ideal, even if in a treatment-oriented framework that is largely irrelevant to non-Western populations. Older Yuendumu individuals averaged significantly lower TPI scores than younger. The absolute magnitude of their mean difference equating to about one point, however, was not great. Both samples showed a predominant condition of, at most, minor deviation from "virtually classic normal occlusion" (Kelley and Harvey, 1977), that is, arbitrarily, a TPI score of less than 4. Haasts Bluff and precontact crania had slightly lower average TPI values.

Older Yuendumu subjects had both reduced arch length and breadth compared to their younger counterparts. This may relate to smaller tooth size in the fluoride-affected younger individuals, but was undoubtedly a result mainly of interproximal attrition and mesial drift, and as always presented a problematic situation in comparing equivalent biological structures. Upon regressing maxillary breadth on year born in the older sample, and finding residuals from that regression within both samples, this age-controlled variable became relatively

larger (rather than absolutely smaller) in older subjects by 0.84 standard deviations but this was not significant ($t=1.41$; $p<.09$). While both maxillary length and breadth were correlated with age within the Yuendumu samples, the B/L ratio was not age-correlated and is significantly greater in older Aboriginals. This must be cautiously interpreted. Haasts Bluff and precontact crania on the other hand retained significantly broader maxillae, presumably in spite of attrition. Paradoxically, the slopes of regression lines of breadth on length (reduced major axis method, owing to the correlations varying from +0.46 to +0.69), did not differ significantly in the same sample comparisons that achieved significance with the ratio. The regression comparison was theoretically the sounder method for bivariate proportions. Precontact crania however significantly exceeded the slope for Yuendumu youths.

The larger overjet in more acculturated subjects corroborated a well-established general trend of modern populations (Brace, 1977). The mean overjet in the older, presumably more traditional Aboriginals exceeded most of the similarly pre-acculturation groups in Corruccini's (1984: Fig. 1) comparison. The deeper bites of younger subjects once more adhered to trends observed in other urbanized ethnic groups (Corruccini, 1984: Fig. 2). A more nearly edge-to-edge incisor relation in adults is in fact the norm for humanity through most of its long prehistory, not the overjet and overbite mean values adopted as esthetic norms by the orthodontic consensus (i.e., overbite of 1/3 and overjet of 3 mm; Kelley and Harvey, 1977) which the younger Yuendumu subjects closely resembled.

The prevailing condition of buccal crossbite is related to greater lateral shift (Brown, 1985) and alternate side chewing (Beyron, 1964) in Australian aboriginals. Whereas Brown (1985) cites the existence of alternate intercuspation in other ethnic groups, it has not been consistently seen in any of 20 diverse populations by Corruccini (1984) including New Britain Melanesians (Corruccini and Pacciani, 1983). Classic centric occlusion therefore does not exist in all Australian Aboriginals.

Yuendumu crossbite type variance was moderately low in comparison

to world populations (Corruccini, 1984: Fig. 3). The positive average values showed BSR discrepancy predominantly reflecting an anteriorly positioned maxilla, a feature shared with white populations, whereas previously all non-European groups have shown negative average BSR type. The Yuendumu variance was less than for any thoroughly urbanized sample in Corruccini (1984: Fig. 4). Mean R/D values in all four samples were quite low by inter-ethnic standards. The mean R/D for younger Yuendumu individuals was exactly intermediate between the samples of fairly unacculturated and fairly industrialized populations in Corruccini (1984: Fig. 5).

Yuendumu mean TPI's fell decisively within the group of other aboriginal or slightly acculturated world populations (Corruccini, 1984: Fig. 6), while larger scores, averaging roughly 5, typify modernized populations. The biological difference signified by the TPI may, in fact, be somewhat greater. Most of the Haasts Bluff and precontact cranial specimens had overjet of from zero to 2 mm, a very normal and healthy approximation to edge-to-edge incisor relation, but the TPI assigns about 1.3 points to the score sum for this feature (which is considered less than optimal by Western cosmetic standards). If one removes the penalty for such mild (and actually natural) close bite, mean TPI becomes 3.11 for younger and 1.98 for older Yuendumu, and only 1.40 for Haasts Bluff and 1.28 for precontact crania.

A classic expectation (Hunt, 1961) is to find narrower maxillary arches in modernized populations (leading to the typical maxillary collapse syndrome), due to insufficient growth or premature closure at the median palatal suture. Most modernizing populations showing increased occlusal variation, also develop narrowed and/or elongated maxillae and midfacial regions. The confirmation of this trend is problematic in the present study, owing to attrition and conflicting results from ratio and regression methods.

Overall, the change in occlusal variables between older and younger generations of Yuendumu Aboriginals was in the hypothesized direction and was statistically significant, but not dramatic. Furthermore, interproximal attrition

may have contributed to the arch shape difference. Indeed, the occlusal deterioration was barely noticeable in terms of subjective evaluation of the cast series. Percentage-wise, changes in dental occlusion in the Yuendumu Aboriginals appear to be less than the 30-year secular trends noted in stature and cephalometrics of this group (Brown, 1976). Haasts Bluff, which we had expected to be comparable to older Yuendumu in environment and occlusion, instead was yet less variable in crossbite and TPI scores.

The precontact crania, on the other hand, included with intent of exemplifying the "ancestral" human state of consistently normal occlusion, indeed showed broader and better aligned arches and generally lowered occlusal variation. Even so, there were several maloccluded individuals. Large interproximal and small occlusal surface carious lesions, low attrition gradient, and provenience suggest that some of the occlusally anomalous cranial specimens may have been in early contact with western traders along the north and south Australian coasts, and hence were not true precontact hunters/gatherers. The most marked case of maxillary collapse however was noted in a precontact skull cataloged as being from the Australian interior.

Comparisons within Yuendumu do not conform entirely to patterns of other modernization studies (reviewed in Corruccini, 1984, 1991; Corruccini and Chowdhury, 1985) which indicate rapid changes from predominantly good to predominantly imperfect occlusal conditions. A number of factors called for consideration in our (Corruccini et al., 1989) thinking:

(a) The environmental/masticatory transition between Yuendumu generations was undoubtedly not clear cut. In fact Barrett et al. (1965) point out that the Aboriginals at Yuendumu were at an early stage of transition from a nomadic hunting/gathering life style at the time the Yuendumu growth study commenced and for years afterward. Some of the older sample may in fact have spent years at cattle stations, finding intermittent work and western food, prior to settlement at Yuendumu. The younger generation, though born and raised at Yuendumu, was still exposed to indeterminate amounts of the coarser and tougher native foods

when these were still collected, off and on, by their parents until the 1960's. Even today, the canteen food is supplemented occasionally by food from hunting and gathering. Thus, the subtle occlusal difference may relate to proportionately subtle dietary changes.. Furthermore, cutlery is not in general use by either generation. In examining records and Tasman Brown's recall of individual subjects, none of the notably maloccluded older subjects was thought to have other than a traditional free-ranging pre-Yuendumu background, and only one of the five perfectly occluded younger subjects had come late to Yuendumu from the Western desert.

(b) The tendency for fluoride ingestion (during growth) to reduce crown size may also be relevant (Goose and Roberts, 1979). Tooth size in the Yuendumu population is relatively small compared with most other Australian Aboriginal groups (Brace, 1980; Townsend and Brown, 1979; Smith, 1982) and this may relate, at least in part, to the high levels of fluoride in their drinking water. Such an effect, if present, may have been more pronounced in the children raised on the settlement than the older individuals, thereby reducing the magnitude of occlusal discrepancies, particularly tooth crowding/maleruption between the two samples.

(c) The older Yuendumu subjects resemble their younger counterparts more than the precontact crania. Significant occlusal change toward western conditions may have already occurred in the Yuendumu Walbiri from these earlier ancestors, for whatever genetic or environmental causes. Thus an occlusal epidemiological transition, related to sustained contact with whites, may indeed have started in Australian aborigines but earlier in time than the settling of Yuendumu. Two possible selective agents, one evolutionary and one a sampling artifact, could be invoked.

(i) Natural selection against malocclusion could have been relaxed in recent Australian Aboriginal generations, allowing accumulation of deleterious genes. We feel this is highly unlikely, even though such crash adaptationist concepts are occasionally encountered in regard to modern human occlusion. The prior selection in relation to occlusion would have had to be very strong to have its

removal engender a detectable change in few generations, and this cannot be reconciled with accepted concepts of the highly polygenic, linked and incompletely penetrant nature of occlusal variation (e.g., Lundstrom, 1948; Smith and Bailit, 1977; Harris, 1975; Potter et al., 1981; Corruccini and Kaul, 1983).

While there are notions that tooth reduction has not kept pace with facial and jaw reduction in hominid evolution (see Brown, 1987), perhaps due to negative intraspecific allometry of tooth size (Gould, 1986), Brown (1987) argues that selection against excessively large teeth would have been stronger than selection for smaller faces. Both Pleistocene face and tooth reduction may be mechanistic, linked results of reduced body size (Macchiarelli and Bondioli, 1986; Brown, 1987). At any rate dental crowding in whites seems more related to smaller alveolar space, than to smaller jaws overall or to larger teeth (Howe et al. 1983).

(ii) It may be that the uniformly good occlusion of precontact cranial samples is related to better dental retention in occlusally healthy individuals. For example, the maloccluded individuals may have lost their teeth early due to periodontal complications (or, more likely, pulpal involvement and alveolar lesions: Clarke et al., 1986), and therefore may have not been included in sampling, or maloccluded individuals may have been generally unhealthy and died prior to dental maturity. Both could be at best minor factors.

(d) Perhaps interproximal attrition, which in Begg's theory provides more space for erupting teeth in the developing dentition, was not accelerated sufficiently in the older Yuendumu individuals. However, the attrition gradient clearly is greater in the older sample, whose significant arch shortening and mesial shift is not merely due to greater age. The increase in Yuendumu R/D over time is relatively greater in the posterior than in the earlier-erupting anterior teeth, which also conforms to the Begg model. On the other hand, this model has always been problematic for explaining other malocclusions: incisor crowding, openbite, and crossbite and BSR discrepancies (Lombardi, 1982; Corruccini and Lee, 1984; Corruccini, 1984; Dawes, 1986). A key test in our data would be the correlation

of R/D and maxillary length, expecting greater attrition to yield shorter yet less crowded arches. A non-significant negative correlation (-.20) results, contrary to Begg-based expectations; this becomes +.14 ($p>.35$) when controlling for the effect of year born on maxillary length.

Dawes (1986) demonstrates that Begg overstated the case for Australian Aboriginal attritional tooth loss even in his own study specimens. Crowded teeth still occurred in individuals whose teeth showed similar wear to the well-occluded cases in Begg's 1935 dissertation, the source of his celebrated 1954 paper's selectively culled data. Corruccini (1984) and Dawes (1986) have noted, furthermore, a paradox in Begg's model: mesial migration of permanent teeth with progressive attrition is supposed to provide the space for M2-3 to erupt normally, yet the same unstoppable process in the deciduous dentition would curtail the "leeway" space for the permanent canine and premolars, conceivably increasing the crowding and rotation of these teeth in preindustrial rather than modernized groups. However, this does not occur. There is need for longitudinal control in direct measurement of attrition and eruption space changes in relation to the adult occlusion. Such a test is feasible in the Yuendumu collections (see later chapter). For example, Sampson and Richards (1985) show permanent incisor crowding correlated with incisor size in these Aboriginals.

(e) The Australian Aboriginal may, whether for fortuitous, linkage-related, or selective reasons, be genetically buffered against growth disturbances in the orofacial complex compared with European-derived or other developed populations. If this is the case, the frequency of malocclusions and tooth malalignment would be less than otherwise expected. However, the dietary and environmental changes that occurred at Yuendumu during the years covered in this investigation were minimal compared with the rapid transitions to western habits that have been reported in other groups. Proffit (1975) and Proffit et al. (1975) show that Yuendumu Aboriginals in general evince smaller tongue pressure, a possible factor in occlusal equilibrium.

In summary, the data indicated a small but distinguishable change in

measurable occlusal characteristics from pre-contact skeletal specimens through older Yuendumu individuals to younger subjects, particularly in overbite, overjet, tooth rotations and displacements. We believe that the most likely explanation for the occlusal differences between the younger and older Yuendumu Aboriginals relates to altered diet and food habits resulting from a gradual change in life style from traditional hunting and gathering to a more settled existence with increasing adoption of western style food habits. Further studies are needed in the present Yuendumu population as this transition proceeds to determine if occlusal relationships are continuing to change. The present study reinforces the view that concepts of normal dental occlusion in modern societies need to take note of the changing nature of occlusal relationships in man, not only in individuals during growth but also in populations as a result of the complex interactions between genetic and environmental influences.

Synthesis

The TPI clarifies all these population differences and gives concrete expression to the epidemiologic transition in dental occlusion, in that all the modernizing or modernized samples show higher values and, indeed, usually exceed the entire confidence limits of less developed groups. One approximate definition of where "normal variation" begins to grade into "malocclusion" is a TPI value of 4 or more. In fact, this arbitrary boundary regularly separates industrialized from less industrialized groups. We can generalize results by classifying populations as "aboriginal" (New Britain, Dickson, and Libben), "moderately acculturated" (Nasioi, Baegu, rural Punjabis; older Pima, Chinese, Kentuckians, and blacks), and "completely industrialized" (younger Punjabis, and whites). About 70 percent of aboriginal subjects show a TPI of less than 4.0 (signifying relatively classic, ideal occlusion), and 60 percent of the moderately acculturated subjects, but only about 40 percent of industrialized peoples fall within this range (Corruccini, 1984). The figure for aboriginals seemingly contradicts the statement of Katz and associates (1976) that "perfection in

occlusion is the exception rather than the norm." This statement applies to Westernized populations only. At the other extreme, although indexing of the TPI is controversial, surely a TPI score exceeding 10 would qualify as malocclusion in virtually any orthodontist's view; no more than 1 percent of the aboriginal and 2 percent of the partly acculturated populations exceed that value, which is negligible, whereas 15 percent of industrialized persons exceed it, which is substantial.

I assert these results serve to modify two widespread generalizations: that imperfect occlusion is not necessarily abnormal, and that prevalence of malocclusion is genetically controlled so preventive therapy in the strict sense is not possible. Cross-cultural, worldwide, time-successive data dispel the notion that considerable occlusal variation is inevitable or normal. Rather, it is an aberrancy of modern urbanized populations. Furthermore, the transition from predominantly good to predominantly bad occlusion repeatedly occurs within one or two generations' time in these (and other) populations, weakening arguments that explain high malocclusion prevalence genetically. Cumulatively, over these study samples, there is no chance for consistent inbreeding, racial mixing, or genetic change to account for the transition. We see an analogous epidemiological transition in Western populations in heart disease, stroke, cancer, and diabetes, and these are now predominantly explained in terms of environment rather than genetics (Corruccini and Kaul, 1983). I would therefore suggest that the same implication when applied to dental occlusion is not too radical.

A second major set of opinions involves variable notions of gene mixing, racial crossing, and matching of discrepant parts. It has been repeatedly pointed out that modern genetic theory concerning polygenic traits is discordant with this thinking (e.g., Lombardi, 1982). A general class of "habits" has been associated with occlusal deviations; yet these help little in explaining the epidemiologic transition. For instance, Price (1935) noted "that in all these racial groups thumb- and finger-sucking were found to be universal habits and studiously encouraged by the mothers. Notwithstanding that, each primitive

group had practically complete immunity to irregularities of dental arches while on their native diets." Sim Wallace (1904) made the same point much earlier: "We may here refer to thumb-suckings which loom so largely in some books as causes of superior protrusion. It is possible that they might act as secondary factors in determining the kind of irregularity when crowding had already taken place, but that they produce such deformities as anterior protrusion is extremely improbable. Thumb and toe sucking is common among infants and exceedingly rare about nine years when the irregularities begin to appear. Moreover, regular teeth are perhaps quite as common among the children addicted to these habits as they are among those who are not. Some writers seem to have a craze for introducing most extraordinary factors to account for most ordinary irregularities. They seem to attach great importance to the fact that Mr. So and So has recorded a case due to such and such a cause."

Proffit (1975) shows that Australian aboriginals have smaller swallowing tongue pressures than Westerners, but swallowing and chewing pressures are not sufficiently prolonged to cause population differences; hence, Proffit suggests that resting tongue pressures may be involved.

Lombardi (1982), although supporting Begg's ideas, states: "An important area of investigation would be whether alveolar bone growth at an early age is affected by the functional stimulation of chewing forces," recalling Lombardi and Bailit's (1972) earlier statement that "further research is needed to determine whether there is a causal association between masticatory function and jaw development." This idea has a long history. Occlusal variation is predominantly a factor of discrepant jaw and tooth size. The functional (chewing stress/stimulation) model provides one explanatory mechanism for consistently insufficient jaw size to tooth size in urbanized modernized populations. Recent studies of laboratory animals raised on soft food show that a variety of occlusal variations may occur, theoretically attributable to normal-sized teeth erupting in undersized jaws. The alveolar processes of the mandible and maxilla, mandibular condyles, and median palatine suture are particularly affected, and both skeletal

and dental occlusion are affected. A multifactorial experimental study (in animals) of oral environmental effects on the developing occlusion could concurrently gauge effects of artificially softened, cariogenic, gritty, and hypernutritive food and of nasal obstruction. Occlusal variations developing among these groups and controls might specify the contributing factors suggested by the human epidemiological studies.

4. Correlative Studies.

As alluded to in the Introduction, a major phenomenon of human modernization and urbanization is the "epidemiological transition." This is a phenomenon of changes in frequencies of certain serious diseases. Omran (1971) classifies this shift from infectious to non-infectious disease worldwide, whereby pandemics of infection are gradually displaced by degenerative diseases as the chief cause of death. This change in disease pattern is closely associated with the demographic transition from high-fertility, high-mortality regimes to low rates of fertility and mortality. Omran (1971) divides the epidemiological transition into three phases, in the last of which heart disease, stroke and cancer replace communicable diseases as primary reasons for death. The Western world is presently well advanced in the stage of degenerative and industrial disease, with two-thirds of all deaths related to the above cardiovascular and malignant diseases (Gerber, 1978).

Essentially, the entire industrialized Western world has completed the epidemiological transition. This is where the vast majority of medically trained practitioners and researchers are located. Yet, epidemiological surveys in this population, designed to specify determinants of chronic diseases, will observe only a fraction of the existing world socio-environmental variability -- namely, that at the upper end of a continuum from traditional rural tendencies to urban industrialized conditions. Here I recognize a significant anthropological contribution in comparative epidemiology. Only by expanding one's outlook to include populations in plainer, more "ancestral" circumstances can a sampling range of environmental variation be included, which would pinpoint correlative

variables across this epidemiological transition and explain the development of chronic disease patterns. The Western medical profession, by and large, is not prepared or inclined to look at this type of variation, which necessitates a cross-cultural approach.

Trowell and Burkitt (1981) more recently have refined the notion of "diseases of civilization", calling them instead "western diseases" and providing a classification. The diseases on their list share a major emphasis seen also in other treatments of diseases of urbanization and modernization (Prior, 1971; Bodley, 1982; Clegg and Garlick, 1980). This is the concentration on serious and potentially fatal disorders. Aside from dental caries (actually an infectious disease) the only diseases potentially not fatal in Trowell and Burkitt's list are constipation and hemorrhoids! Omran's (1971) major triad of heart and hypertensive disease and cancer, together with diabetes, remain the prominent examples of western disease.

Cross-cultural surveys of coronary heart disease and cardiovascular conditions have done much to clarify etiological factors. I do not intend a comprehensive review of these, but some patterns are illuminating. Gerber (1980) and Gerber and Madhaven (1980) demonstrate that a variety of ethnic groups with low prevalence in traditional environments, acquire western levels of these conditions within one generation of immigration to fully developed countries. Genetic change of any kind can be ruled out as a factor in this change, as it is simply too rapid. This contrasts with older selection-based arguments for western acquisition of heart disease (e.g. Nye, 1966). Risk factors predicting the development of cardiovascular disease, such as blood pressure, have been consistently shown to rise with urbanization (e.g. Ward and Prior, 1980; McGarvey et al., 1980; Brown, 1981); again the transition is fairly immediate. Diet and fatness are important from childhood for determining the adult disease pattern (Trowell, 1975), thus the change is usually seen in first-generation offspring of migrants. Knowledge of the dietary factor plus those of sedentism and mental stress have allowed a meaningful program of prevention to evolve; the

idea of genetic causation of the new disease pattern has disappeared, although a genetic factor in relative susceptibility to the new environmental conditions is, of course, recognized.

Adult-onset diabetes mellitus is a western disease, and although there is variation in genetic predisposition to development of diabetes, all surveyed ethnic populations show increase with modernization and industrialization (e.g. O'Dea et al., 1980) with attendant income, obesity, carbohydrate diet, inactivity, and urbanization. Australian aboriginals furthermore reduced their metabolic glucose response upon resumption of the traditional lifeway (O'Dea et al., 1980), demonstrating that accumulation of genetic load is not the direct cause of diabetes susceptibility; rather activity reduction and simpler carbohydrates underlie the transition. As with heart disease, the change is too rapid to allow consideration of explanatory models of genetic adaptation or mutation; some evolutionary preadaptation to a hunter-gatherer life habit allowing susceptibleness to diabetes mellitus type II, may have been explosively expressed upon the relatively sudden human agricultural and industrial revolutions (Neel, 1963). At any rate this is clearly an environmental epidemiological transition; population differences in genetic predisposition or susceptibility predated the transition.

By contrast, diabetes earlier had provided the classic case for eugenicists of the supposed accumulation of deleterious mutations in the human population, mutations once removed by natural selection but now growing at an alarming rate because of technological buffering (see, for example, Dobzhansky, 1962). This idea gave rise to visions of the human race in the year 3000 A.D. (say) being entirely diabetic and in other ways unviable and degenerate. Historically we find similar notions (cf. Klatsky and Fisher, 1953) regarding reduction and degeneration of the teeth. This concern over "survival of the unfittest" is now untenable, for the mutation leading to diabetes could not possibly be accumulating rapidly enough to explain this virtually instantaneous epidemiological transition.

Epidemiological Transition in Minor Diseases

Reviewers of the epidemiological transition to western disease patterns concentrate on the fatal, and therefore most obvious conditions. There exists also a variety of generally non-serious (for westerners) chronic maladies that prevail in even larger percentages among the urban, industrialized world, which have been overlooked to date. Furthermore, evolutionary and anthropological logic suggests that these also must be rare in aboriginal populations (including the biological ancestors of western people), and that their sudden appearance in urbanizing societies indicates environmental causation.

What are the odds that hunter-gatherers could survive, burdened with the western 50+ percent prevalence of malocclusion (Kelley and Harvey, 1977), given their need to orally process coarse, raw and extremely tough dietary items daily such as roots and wild game? Does it stand to reason that savanna aboriginals would maintain reproductive efficiency if forced to locate game and plants with the western 60 percent prevalence of short-sightedness (e.g., Angle and Wissman, 1980) and other visual impairment? Would not nomadic band members risk extinction if respiratorily encumbered with western symptoms of frequent allergy and asthma? Certainly foraging bands have enough natural forces acting upon them, that such additional loads would imperil them with extinction. Do such maladies exist in aboriginal groups, that are relatively unbuffered from their environment by technology? Actually these minor chronic conditions must undergo an even more drastic epidemiological transition with modernization (considering their higher prevalence than serious diseases) if in fact they are rare among traditional unindustrialized peoples.

As we have earlier seen, the Punjabi population in and around Chandigarh, India, shows several special biosocial characteristics of value for a general study of the epidemiological transition, going beyond the malocclusion syndromes. Over a short geographic distance eastern Punjab communities cover the entire spectrum from quite urban, modern, sedentary professional levels to rural villages in which the agricultural way of life and reduced circumstances

typical of longstanding countryside isolation have been little changed. The planned capital city of Chandigarh was started as part of the rehabilitation program for the immense displaced population that migrated under duress from West Punjab (now Pakistan) after the partition of 1947. The city now has one of the highest per capita incomes in India. Migrants to the city inhabit characteristic areas and are easily discriminated from the long-term urban residents whose degree of westernization (or modernization) is almost completely predictable from income level. The difference in activity, environment and diet are far-reaching, yet there is thorough documentation of genetic homogeneity among the population components (Corruccini and Kaul, 1984; Kaul and Corruccini, 1984). Some of the literature cited earlier shows the serious western diseases rapidly on the rise in this urban population.

In this genetically-controlled study situation where fatal western diseases are rising, what is happening with the spectrum of bothersome (and in the U.S.A., quite expensive) minor chronic diseases? We resolved to take a purely cross-sectional survey approach, contrasting alike-aged rural and urban youths, to answer this question in regard to other conspicuous "diseases" besides malocclusion in the human laboratory constituted by the Punjab.

Chronic Allergy and Oral Breathing

"Hay fever", respiratory allergy, asthma and chronic oral breathing probably deserve inclusion on the list of western diseases. As such they may be suspected of being environmentally-induced, but probably this syndrome is complex. An immunological response involving Immunoglobulin E is implicated in white populations only (cf. Flander, 1981). A strong familial tendency has been demonstrated, again only in western groups (e.g., Cooke and Vanderveer, 1916; Gerrard et al., 1976). Nevertheless, the immigrant studies indicate one-generation change to western prevalence levels of allergy and asthma regardless of ethnicity (Glazer, 1969; Smith, 1973, 1976), and rural population components regularly have lower prevalence than urban (e.g., Godfrey, 1975; Merrett et al.,

1976).

We were initially interested in allergy as a correlate of deformed arches and facial narrowing in our occlusal study, because the clinical literature frequently mentions this connection (e.g., Marks, 1965). Long ago Sim Wallace (1904) noted the connection and suggested that dietary consistency was the hidden culprit: "Adenoids and mouth breathing are generally and correctly supposed to cause narrowing of the palate, and I believe with Dr. Campbell that one of the predisposing causes of adenoids is insufficient mastication .... Insufficient mastication, according to Dr. Campbell, predisposes to adenoids by the stagnation of lymph, and the less vigorous circulation of blood in the naso-pharynx."

Recall that previously we found quite higher levels of various measures of occlusal variation in the urban Punjabi youths, compared with a rural sample. The search for etiological correlates of this difference focused on diet, premature deciduous tooth loss, and on unexplored differences in allergy/asthma. We found that we could reject differential caries rates, genetic frequencies, age variation, inbreeding and gene mixing, periodontal disease, and interproximal attrition as explanations.

Accordingly, in the second year of the study we returned to urban and rural schools, systematically cross-sectionally sampling youths in the same 12-16 year age range (Corruccini and Kaul, 1983). The design was to identify subjects definable as chronic oral breathers, assess and quantify their key occlusal variables using earlier criteria, and compare them statistically with the general population. . Louisa Flander diagnosed probable chronic allergy and mouthbreathing in the field.

Habitual oral breathing and its relation to dentoskeletal occlusion has spawned a large and varied literature which is covered in recent reviews (Vig et al., 1981; O'Ryan et al., 1982; Bresolin et al., 1983; Smith, 1982; Vig, 1979; Sain, 1982). A general inconclusiveness, or one might say at leasta lack of consistency characterizes the literature (Vig, 1998; Yamada et al., 1997). There is variable mention of specific dento-occlusal features resulting from oral breathing (e.g.

anterior openbite and posterior crossbite are mentioned as resultant conditions in some, but not all studies).

Mouthbreathing is a thorny term, still undergoing refinement. Ambiguities such as mutually noninclusive morphological concepts of nasal obstruction, oral respiration, adenoid facies, long-face syndrome, etc. compound the problem. Vig (1981) attempted objective determination of respiratory mode with an apparatus for measuring nasal airflow, and concluded that the issue is more complicated than generally thought.

A new perspective is cast on the mouthbreathing-malocclusion connection by considering changing epidemiological conditions surrounding these diseases during cultural evolution. Human societies undergoing rapid modernization and acculturation show increases in both allergy/asthma (Corruccini and Kaul, 1983; Marks, 1965) and in malocclusion (Corruccini and Kaul, 1983; Corruccini, 1984), over the previously very low or nonexistent levels characterizing hunter-gatherer and prehistoric groups. It must therefore be asked whether increased urban tendencies toward oral breathing underlie some of the well-documented epidemiologic transition from good preindustrialized to frequently imperfect modern dental occlusion. We acknowledged limitations imposed by the fact that oral breathing is but one small part of the respiratory allergy spectrum, and that field examination primarily reflects dental occlusion even though there may also be skeletal components of traits such as incisor overjet.

Field diagnosis of respiratory allergy indicative of chronic oral breathing was based on positive complaint or history of breathing difficulty through the nose (at night, in certain seasons) from interviews, and on examination for clinical signs based primarily on Marks' criteria (Marks, 1965, 1973, 1977). These signs were boggy turbinates, nasal crease, wheeze, gaping habitus and lip incompetence, torus palatinus, deviated nasal septum, buccal ridge, allergic pseudopannus, and nasal obstruction. These traits occur in statistically significant frequency in children undergoing desensitization treatment for asthma and hay fever (Flander,

1982). One positive complaint plus at least one positive clinical sign in combination were frequently required for diagnosis as a chronic oral breather.

Chronic allergy was diagnosed (Corruccini, Flander and Kaul, 1985) for 21 percent of the urban youths and only 9 percent of the rural (chisquare= 8.45, $p<.005$), a statistically significant difference. However, males were significantly more susceptible than females (relative risk=2.23; $p<.01$). The sex-specific odds ratios were therefore compared for urban contrasted with rural samples, being 1.97 (significant) for males and 2.36 (not quite significant) for females. These results did not derive from mistaken diagnoses of colds, as infection was much higher in the rural youths.

Both dental malocclusion and respiratory allergy showed sharp and rapid rises in the urban environment. To what extent are the two phenomena interrelated? The question was approached by comparing frequency and amount of occlusal discrepancy within environment-specific samples of normals and mouth breathers (Corruccini et al., 1985).

Overjet showed the strong rural-urban difference previously mentioned, while diagnosed mouthbreathers did not differ from normals in either rural or urban environment. There was no consistent pattern in how normals and mouth breathers differed. Overbite showed less variation overall, and less difference between rural and urban samples. The mouthbreathing factor again was not significant.

Buccal segment relation anomaly, likewise, showed large divergence of urban from rural prevalence in the normal breathers, while the smaller mouth-breather samples did not differ significantly. Crossbite discrepancy also showed marked rural-urban differences. In this variable there was also a consistent increase in variation in mouthbreathers (all observed discrepancies were with the maxillary teeth displaced lingually). This was statistically significant at $p<0.01$ in the rural sample, but nonsignificant in urban youths.

The tooth displacement score and the TPI followed the pattern of no direct relation to mouthbreathing.

Thus, traditional rural and recently urbanized Punjabi populations differed sharply in prevalence and severity of occlusal variations, and they also differed to a roughly comparable extent in prevalence of respiratory allergy and asthma indicative of probable mouth breathing. Nevertheless, within the rural and urban samples there was little tendency for chronic oral breathers to show more frequent or greater discrepancies from occlusal norms.

Crossbite alone indicated a possible relation, as also found by Bresolin et al. (1983). Even this conclusion should be treated with some caution, as the difference was statistically significant only in the rural sample where mouth breathing was infrequent (the combined sample of 462 subjects showed $p<.01$ for the higher posterior crossbite prevalence in mouthbreathers).

This finding may be specific to this population, but should also be considered together with results from western and other nonwestern populations in seeking causes for the rapid rise in malocclusion in urbanizing and industrializing peoples (Corruccini and Kaul, 1983) There are many environmental and genetic determinants of facial form as we have seen by now.

Dietary refinement and, to some extent, respiratory allergy are conditions of westernization (Corruccini and Kaul, 1983); prevalence of malocclusion, maxillary and facial "collapse syndrome" (Kelley and Harvey, 1977), long face syndromes, and oral breathing all soar in rapidly modernizing, urbanizing societies. The "long-face syndrome" of vertical craniofacial dysplasia is basically of unknown genetic etiology among American patients (Fields et al., 1984).

The developing, non-Western societies are the settings in which to test etiological hypotheses then, not the relatively environmentally homogeneous West, especially when significant genetic change can be controlled. A later paper extended the previous studies of intra-oral characteristics and factors to external facial dimensions. We (Corruccini, Whitley et al., 1985) attempted to contrast the dietary consistency and oral respiration factors (in genetically constant and sex-controlled study situations) in developing populations, to seek generalizations

concerning the limited aspect of variation in facial length and breadth. The populations were Northwest Indian Punjabi youths (Corruccini et al., 1982; Kaul and Corruccini, 1984) and the circum-Appalachian west-central Kentuckians studied earlier by Corruccini and Whitley (1981).

We took three measurements: one lower jaw (bigonial) breadth, one of midfacial breadth (bizygomatic in Punjabis and bimaxillary in Kentuckians), and total facial height (nasion-gnathion).

The Punjabi sample consisted of the later (year 2 of study) 315 youths age 12-16 studied in schools inside the modern city of Chandigarh, or from more traditional rural villages up to 30 km from Chandigarh.

Three maturational variables (age, sitting height and stature) were recorded; these were regressed against the facial measurements to control general growth variation.

The Kentucky sample consisted of 78 young adults age 17-22 sampled in a college attended by nearby urban residents as well as a contingent from a fairly traditional rural area described in detail earlier (Corruccini and Whitley, 1981). Some eleven of the rural individuals came from farming families that raised most of their own food and some of whose staples required fierce masticatory forces, while the rest were raised on typical processed commercial food. We expected no relevant genetic differences among rural and urban components owing to the general American panmixis and the local sharp decline in inbreeding and increased mean marriage distances (from 2.2 to 8.4 km) in this generation.

Oral breathers were diagnosed by interview only in this Kentucky group. This design is adequate for field study of mouth-breathing as it is conventionally conceptualized, but we acknowledge the ambiguous relation recently emphasized (Vig, 1979; Vig et al., 1981) between amounts of nasal blockage, oral respiration and gape. Dietary consistency during growth was assessed by asking whether participants' families had regularly engaged in gardening and home food processing including deep-fried cornbread and cured pork.

We found relatively large bigonial contrasted with small bizygomatic and height measures in the rural Punjabi samples. The only clearly significant rural/urban difference was in bimaxillary breadth for the Kentucky males.

Six male and 7 female Kentucky subjects were assessed as mouth-breathers, of which most were rural: neither male/female nor urban/rural risk ratios approached significance. All facial measurement comparisons between mouth-breathers and normals (within sex and urban/rural categories) were non-significant. There was consistently about twice as much sex dimorphism in facial measurements in rural as in urban samples.

Morphometric separation of mouth-breathing from normal subjects was not noteworthy, and again can be ascribed to random variations. The dietary consistency factor, conversely, yielded a much more accurate discrimination of samples, identifying correctly 74 percent of individuals. The discrimination according to dietary consistency was determined by large bigonial and bimaxillary breadths contrasted with small height.

We (Corruccini, Whitley et al., 1985) suggested two provisional conclusions, subject to further cross-population testing. First, allergic mouth-breathing status, as here defined, does not result in longer or narrower faces, or indeed in any confidently detected morphometric difference.

Second, we do find difference in the non-industrialized, rural population components contrasted with urban counterparts, for which dietary refinement provides one obvious explanation. Nevertheless, the pattern of facial differences is dissimilar in Punjabis and Americans, suggesting forceful chewing does not necessarily result in the same changes in different populations. Face height rather than the breadths shows the most response to increased rural masticatory demand in Punjabis. A theoretically expected (Hunt, 1961) reduced height and increased breadth does occur in Kentucky young adults raised on unprocessed staples.

These are complex rather than uni-directional differences, which do not correlate with nutritional differences (i.e., the larger-faced rural Punjabis clearly have poorer nutrition). Therefore we do not believe that uncontrolled

maturational factors likely play a role in the results.

The environmental factors of possible significance for allergy include cow's milk in place of breast feeding, dust, certain insects, pollution and food additives (see Flander, 1981), all more prevalent in the urban environment, and immunological response to parasite infestation, more prevalent rurally.

Bite Force Studies

A major oral physiologic aspect of human occlusal variation is the force of biting. Human bite force has stimulated many studies. Anterior molars exert forces of about 15-17 kg during normal adult chewing, and about 28-39 kg as a maximum (e.g. Maddock, 1963; Carlsson, 1974; Best, Roberts and Ram, 1982; Proffit and Fields, 1983).

Early 20th century interest concerned the connection between dietary coarseness (hence force of habitual chewing) and malocclusion. A classic study, using a gnathodynamometer, found bite forces two to three times greater in non-industrialized Eskimos than in their counterparts who had been affected by European-type culture (Waugh, 1937a, b).

Bite force is relevant to the functional aspects of craniofacial dysplasia (Proffit and Fields, 1983; Fields at al., 1982). Disorders of dental occlusion tend to reduce bite force (Carlsson, 1974).

Following the first year's fieldwork on the Punjab project, Kaul and I saw investigation of bite force variation as the most profitable avenue for new etiological research to take. Our purpose was to investigate the possible relationship between bite force and occlusal variation in the youths of North India.

With the added assistance of Avery Henderson, who was advised directly by Bill Hylander, we returned to India with a device to measure bite forces (Corruccini, Henderson and Kaul, 1985). The bite-force measurement apparatus included a bite-force transducer, a portable strain indicator and a digital autoranging multimeter. Two bite-force transducers were constructed from a

proven design (Hylander, 1977). One end of a 130 mm long steel rod (diameter 1.5 mm) was inserted into a 10-by-10-by-5 mm plastic block and a 40-by-10-by-5 mm Plexiglass handle was fitted to the other end. Two 120-ohm-foil strain gages were bonded to the block, one on the left and one on the right side. Two gages were used for the construction of the bite block because subjects often bite near the edge rather than in the middle of the block. If biting occurs along the edge, the block is bent instead of simply compressed; this results in tension on one side of the block and added compression on the other. To eliminate the effects of bending, the two gages were connected in series. Therefore the added compression sensed by the gage on one side of the block was canceled by the tensile strain on the opposite side. The two gages were aligned perpendicular to the steel rod and connected in series with 32-AWG polyvinylchloride-insulated lead wires. The solder joints and gage tabs were insulated with a plastic coating and finally protected by a layer of 2-3 mm of fast-setting epoxy resin.

To measure the bite force, the gages were connected to form one arm of a Wheatstone bridge in a portable strain indicator and an LCD Digital Autoranging Multimeter, 10-megohm-input, was connected to the portable strain indicator. When force was applied to the plastic block, the plastic was deformed and strained and the electrical resistance in the two attached gages was altered. The change in resistance resulted in voltage changes across the Wheatstone bridge which were measured by the multimeter and displayed digitally.

Two observers of the team conducted the bite-force measurement procedure. One, Kaul, explained the procedure of biting to the participants and placed the transducer into the subject's mouth. The second observer read the electrical digital displays on the multimeter and recorded the readings.

For the biting procedure, the block portion of the transducer was inserted into the subject's mouth on the right side, the left side being used when there were diseased, sensitive, or missing teeth on the right. The steel rod was positioned parallel to the occlusal plane; the bite block was positioned between the maxillary and mandibular teeth and centered on the mandibular first molar.

Under these conditions, the bite force, approximately perpendicular to the occlusal plane, was then in line with the strain gages. Two bite-force measurements were taken, one instructed to be for a normal food-chewing bite (such as, e.g., on a chapati) and a second for maximum bite. The first bite was to give the subject familiarity with the transducer block. When the subject was comfortable, he or she was instructed to bite on the block as hard as possible for a static contraction period of 3 seconds. Maximum digital readouts were recorded for both bites. All readings were taken between 9:00 am and 12:30 pm. We found a high repeatability (r=+0.81) for the maximum bite force readings taken on different days.

The sample of 70 normal rural males yielded an average bite force (maximum) of about 37 kg, contrasted with 24 kg for their 51 urban counterparts. This very marked difference was not repeated in normal females (about 26 vs. 23 kg). However, rural (over 9 kg) and urban (less than 7 kg) females registered stronger differences in "normal chew" force.

Thus our raw data suggested considerable sex difference, and a rural-urban differentiation was obvious, while values fluctuated according to mouth-breathing diagnosis. Analysis of variance showed that maximum bite-force variation between rural and urban samples was 20 times as great as variance within those samples. The probability of this occurring by chance is less than 0.001. Partition of variance was not sharp between male and female bite forces for the normal chew values. There was more bite-force variation within than between mouth-breathers and normals. The oral breathing syndrome, one of several possible correlates of narrow, long faces and of malocclusion, seems to bear no relation to chewing stresses or reflected masticatory-musculature strength.

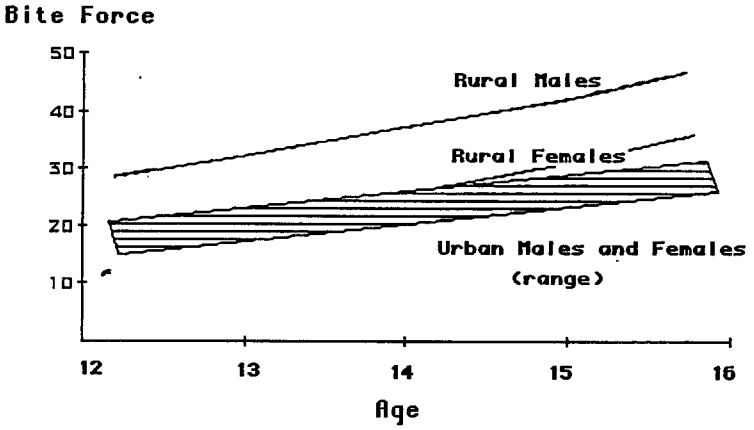
In view of gender difference, maturational variation and especially secular variation, it was desirable to control for the growth factors. The better-nourished urban subjects were taller and, on average, entered puberty one year earlier; they also erupted permanent teeth about a year earlier. Among age, statural and facial measures taken, bite forces had the highest correlation with

standing height (stature). Accordingly, stature was used as the independent variable in bivariate regression against normal chew and maximum bite-force mean-values were transformed to residual values away from this common regression line.

As there was again no difference whatsoever according to respiratory diagnosis, the samples were differentiated solely by sex and living environment. This regression-adjustment actually increased the amount of differences in growth-independent bite-force between rural and urban samples, because in the urban subjects stature was greater and the adolescent growth spurt began earlier. In terms of maximum bite force, even rural females exceeded urban males by 14 standard errors of the mean difference ($p<0.001$). The minimum rural-urban difference in normal bite was six standard errors ($p<0.001$).

A difference was present in sex dimorphism of bite force: rurally the sex difference was highly significant (nearly as much as the mean rural-urban difference) whereas the urban subjects showed less, non-significant sex dimorphism. That this pattern results from greater rural growth differentiation upon attaining adolescence, is supported by examination of inter-trait correlations. Morphological integration of bite force with age and with facial, dental arch and stature measures was significantly higher in the rural sample. Maximum bite force increased about 5.15 kg/yr over this age range in rural subjects, but only about 3.0 kg/yr in urban subjects. The tendency toward growth coordination with bite forces was great for age (see below), standing and sitting height, and maxillary arch breadth (but not length), and significant for face measurements (but generally only in the rural youths).

The facial measurements all correlated significantly and about equally with maximum bite force in the rural sample. There was no tendency for a greater influence on face width than on face height, and no tendency toward negative correlation between bite capacity and face height as might be expected in response to the long-face syndrome (Proffit, Fields and Nixon, 1982; Fields et al., 1982) either among or within samples.



The observed bite force values are broadly similar to those found by some other workers (Maddock, 1963; Best et al., 1982; Carlsson, 1974; Fields et al., 1982), but less than those of Proffit et al. (1982). The amount of comparability between different workers using different technology is unclear (Carlsson,1974) especially with regard to the factor of vertical gape (Dechow and Carlson, 1982). Results are strictly comparable only within a single study.

Ordinary chewing and maximum bite forces thus were significantly higher among more traditional rural Punjabi youths than among genetically

equivalent, urban youths. The lesser urban bite forces do not result from defects or disease of the supporting tissues. Periodontal disease was significantly more prevalent in the rural youths (Kaul and Corruccini, 1984); they have much less professional dental care. Caries was rare in the entire sample, with a mean 0.21 carious teeth per subject. Furthermore, caries were more often treated in urban youths, suggesting that they would have less pain and hence tendency to avoid hard biting. We believe the greater habitual chewing force in rural subjects is related to the greater maximum bite force capability, and probably to the better dental occlusion. Diminution of chewing function, as discussed at length earlier, could adversely affect oral-facial growth.

The lack of urban sex difference in bite force contrasted with the rural population. So did the pattern of correlations between bite force, stature, craniometric and age variables, implying a different pattern of early adolescent growth in the masticatory apparatus in the different environments. If chewing of resistant foods stimulates greater and more coordinated facial growth, this is reflected in the higher rural correlations; urban bite force tended to increase but little with age, suggesting that masticatory muscles are not sharing the general growth process. This might be explained by a soft diet and consequent lack of any need to increase mastication in response to increased metabolic needs. This also explains absence of urban sex difference in bite force at adolescence, before which there is little growth-rate difference between boys and girls. Body-size sex dimorphism, especially in stature, was conversely greater in the urban youths than in the rural. This relates to nutritional, not physical factors; the stronger bites occur rurally nevertheless.

Among Western children, Carlsson (1974) found bite force almost as strong as in adults; Proffit et al. (1982) on the other hand found that adults bite harder. The pattern in Western urbanized and industrialized society probably corresponds to the urban Indian situation where adolescent youths show no sex differentiation and not much age increase in bite force. The rural situation is quite different with occlusal development continuing throughout the period of rapid

growth.

A final anecdotal point relates to our functional explanation of Punjabi bite-force patterns. We sampled only apparently healthy and unimpaired subjects at our schools but, one day, a rural boy, a severe polio victim, was included in order that he would not feel left out. We expected a feeble bite but while his peers held him up he registered a maximum bite force exceeding by 42 per cent any other of the hundreds of other subjects, including many older and stronger rural boys. This was verified with a repeat reading several days later. This boy had always used his teeth as grasping tools in place of his useless hands.

Our findings promote the critical correlation between chewing demand, craniofacial and muscular growth, and occlusion. Longitudinal studies in environmentally heterogeneous populations such as this are needed to explore the relation further.

Visual Defects and Refractive Error

Myopia or short-sightedness is a disease that mirrors much of the situation just described for dental occlusion. Consideration of this disorder lends perspective to many of the points made regarding malocclusion. Genes are considered the determining factor in textbook treatments, and relatively little thought is given to causes. Some pioneer cross-cultural epidemiology by Young (1954, 1969) showed better eyesight in rural versus urban samples and in unschooled Eskimo parents versus their educated children. Young (1961) also proceeded to the logical primate experiment to test the effects of nearwork and close focusing on malformation of the eye (just as have Corruccini and Beecher [1982] for soft diet in regard to occlusal anomalies). Morgan and Munro (1973) show that Amerinds and Eskimos "have eyesight well suited to their nomadic and hunting society", which is merely logical given the human biological tendency toward adaptation. They show that refractive errors increase within one generation upon acquisition of reading habits. They list a series of common medical opinions about cause of refractive error which is highly reminiscent of

clinical outlooks on malocclusion: racial hybridization, increasing fecundity of myopes, differential mortality, and sexual selection, none of which explain the rapid rise in incidence. They suggest that the idea of myopia being preventable is radical to the ophthalmologic community. Bear (1982a) adds data from Newfoundland showing refractive errors rare in hunter-gatherers, while nearwork (particularly reading) seems to incur an increase that is far too rapid to be attributed to genetic mechanisms (Bear, 1982b, c; Bear and Richler, 1981, 1983; Bear et al., 1981).

The early attempts to demonstrate epidemiological transition in myopia due to functional factors were followed by strong reaction from the hereditarians and a general reversion in the literature to the view that defective vision is hereditary (e.g., Keller, 1973; Sorsby, 1970: Sorsby et al., 1957). Here again the views encountered are reminiscent of those of craniofacial clinicians; environmental explanations are discounted as old-fashioned and unsophisticated; the genetic factor is all-important. The finding that more educated, highly academic populations have more myopia (Dunphy et al., 1968; Peckham et al., 1977: Robinson, 1977) leads to what conclusion? That those who spend more time reading have more genes for myopia! "India is a land of eye disease" (Mann, 1966), and rapidly modernizing Indian localities are ideal for study of the visual epidemiological transition. A graduate student of ours, Christopher Hendel, developed a protocol to survey visual acuity during the year before our second visit to North India, with the assistance of Dr. Jerry Levelsmeier. We visited rural and urban Punjab schools to sample visual acuity in 12-to-16 year olds using the international "Tumble-E" eye chart (Hendel, Corruccini and Kaul, 1983). Compliance of the subjects was 100 percent. Vision that was measurably worse than 20/20 (including slight to severe refractive error) showed an urban/rural risk ratio of 2.19 which was highly statistically significant. In this variable there was significant sex difference however, female/male relative risk being 1.54 (the girls in rural schools, incidentally, engaged in much more nearwork such as embroidery). The sex difference was significant only in the rural sample

(relative risk=2.83). Sex-specific relative urban versus rural risk was 3.70 for boys and 1.80 for girls (both significant). Risk in both was more dramatic for severe visual impairment (20/50 or worse; that is, subjects could read a bottom line at 20 feet that normal subjects should have been able to read at 50 feet). Sex difference was not significant at this level ($p>.80$); prevalence was 16.3 times greater in urban youths!

Since infectious disease of the eyes is undoubtedly greater in the rural sample, the difference in refractive error may have been diminished which would increase Type II statistical error. These differences justify description as an epidemiological transition, confirming the earlier studies on other ethnic groups. Interviews and other data strongly suggest myopia is a disease of reading and of near-work in these youths. The possibility of preventive therapy using this finding is obvious; a few references exist for behavioral and distant-focusing therapy for vision (e.g., Levy, 1982).

The Epidemiological Transition in Genetic Variance and Heritability

Many old ideas for the etiology of occlusal variations seem archaic and quaint to practicing orthodontists today. The evidence for epidemiological transitions, in the uncommon instances that the clinical dental literature takes notice of it, is often ascribed to genetic mechanisms of deterioration and racial gene mixing. Some of this reasoning is reminiscent of Lamarckian inheritance of acquired characteristics, such as references to third ("wisdom") molars gradually decreasing in size and disappearing in the course of three generations, and mandibles growing noticeably shorter generation by generation. The opinion might be ventured that these beliefs could only persist in a medical speciality concerned with a non-fatal disease.

Personal experience in communicating these ideas to audiences of dental clinicians occasionally indicates persistence of the attitude outlined above. A "simple" explanation is considered improbable; interest in corrective approaches greatly outweighs interest in prevention, and it is considered unsophisticated to

ponder environmental versus genetic variables since it is so well known that the two interact. This tends to protect the genetic causation inclination.

We have seen how the serious western diseases have environmental causes and prevention but the minor western diseases are still considered genetic. Indeed, significant heritability estimates do exist for these minor maladies, estimates that have all been measured on western populations. As a generalization, to the medical specialties the heritability evidence validates belief in genetic causes of the high prevalence; thus there must have been a great increase in western populations in the genes responsible for the disease. However, Feldman and Lewontin (1975) state "Changes in frequencies of such diseases as diabetes mellitus, celiac disease, and schizophrenia, whose mode of inheritance is not known, are probably intermediate between the dominant deleterious diseases, and therefore will be largely determined by mutation rates" which would be extremely slow. The same can be said about speed of evolutionary increase in such diseases.

How can the heritability coefficients be reconciled with evidence of the environment as predominant cause of the epidemiological transition? Static heritability coefficients are computed in diverse ways, but always pertain to the concept of comparing genetic variance to environmental variance, usually in a ratio. Clearly, either an increase in genetic variance or restriction of environmental variance can increase such coefficients. Therefore high heritability does not necessarily imply that genetic change alone can bring about phenotypic change. This is particularly exemplified by the I.Q. controversy: "the analysis of variance is done (and heritability is calculated) with respect to a particular array of genotypes and environments in a specific population at a specific time. This array is usually a biased sample of the full array of genotypes and environments" (Feldman and Lewontin, 1975). Again, to quote an argument used against I.Q. genetic determinists: "Consider the case of skin color. If we estimate the heritability of skin color among white New Yorkers, including people of Italian, English, Puerto Rican, and Polish ancestry, we find a high heritability. Suppose

we now compare a group of New Yorkers who are left to winter in the city with a group of their well-to-do in-laws who spent the winter in Miami Beach. There would be a considerable skin color difference between groups, but no genetic causation" (Feldman and Lewontin, 1975). Therefore Feldman and Lewontin raise the concept of between-group heritability. Clearly, genetic variance must be measured across the epidemiological transition to properly answer whether the transition is genetically caused. This cannot be done in western society today. Our transition is complete, and in many basic aspects the environment is uniform.

Evidence is accumulating that heritability of physical disorders also undergoes a transition in modern industrialized environments. Many anthropometric and growth variables appear substantially heritable based on studies of western urbanized subjects, but genetic variance drops considerably when measured in rural, underdeveloped, or disadvantaged populations (Russell, 1976; Mueller, 1977). Russell and Mueller cite a combination of greater environmental variability (such as nutritional fluctuation, less technological buffering) with reduced parallelisms of environment among relatives, in these studies. This finding has been repeated in underprivileged as contrasted with urbanized children in the Chandigarh environs (Tanwer, 1977). Sibling correlations for instance, long a key criterion of heritability, are much lower with poorer nutrition in Mexico and Taiwan (Mueller and Pollitt, 1982). McGarvey et al. (1980) demonstrate that Samoan sib blood pressure correlations increase with modernization. There is increased inter- and decreased intra-familial environmental variation. Similarly Ward and Prior (1980) report hypertension heritability "apparently" increases in Tokelauan migrants to an industrial environment.

Bear (1982a) reports that common within-family patterns in nearwork inflate sib-sib genetic estimates for visual acuity. Eskimos show no parent-offspring correlations (Young, 1969) for vision (the sib generation was the first to enter school and cross the refractive epidemiological transition). Thus heredity can be fallacious unless measured both by parent-offspring and sib-sib methods

(both of which share the same genetic similarity: 50 percent); this revised approach as well as new twin analysis methods have led to drastic reduction of heritability estimates for dental occlusal variations (Corruccini and Potter, 1980; Harris and Smith, 1980; Potter et al., 1981).

An explanatory model lies in the mode of life to which we are truly bodily adapted -- not the urban, industrialized lifehabits the west has known for only 10 generations. Any environmental stimulus to proper physical development that is inevitably present would not be a factor in genetic adaptation -- such environmental buffering or canalizing factors could be more or less relied upon. Thus vigorous dietary stimulation to facial architecture and the alveolus was always present, for example. To the extent that this ensured proper integration of jaw growth and tooth eruption, genetic programming of some of the developmental pathway was redundant, and the human genome accordingly may not have been under selective pressure to incorporate the redundant information. Perhaps genetic variation accumulates but is not phenotypically expressed due to the masking or screening effect of a longstanding environmental force (such as tough diet in the case of malocclusion, or dynamic distant focusing in the case of the eye muscles, or strenuous lifehabits and low fat intake with respect to artery disease). Upon the sudden removal of such forces, the underlying trivial genetic variation in susceptibility can be expressed, giving false notion of genetic cause.

Anthropologists are ideally suited to test the model in developing societies, and the minor western diseases are useful for study since (a) they are so prevalent, (b) the confounding role of competing morality (i.e., degenerative mortality rising merely because of lessening infectious risks) is absent, and (c) research design and data collection are less difficult than with serious diseases which, in less developed areas, may involve sources such as recounted causes of death or hospitalization incidence (which are unreliable in less developed areas).

Figure 1. The upper jaw and maxillary teeth of a contemporary Yuendumu Australian aboriginal, demonstrating fairly normal alignment and occlusion although there is slight crowding of the lateral incisors and posterior premolars.

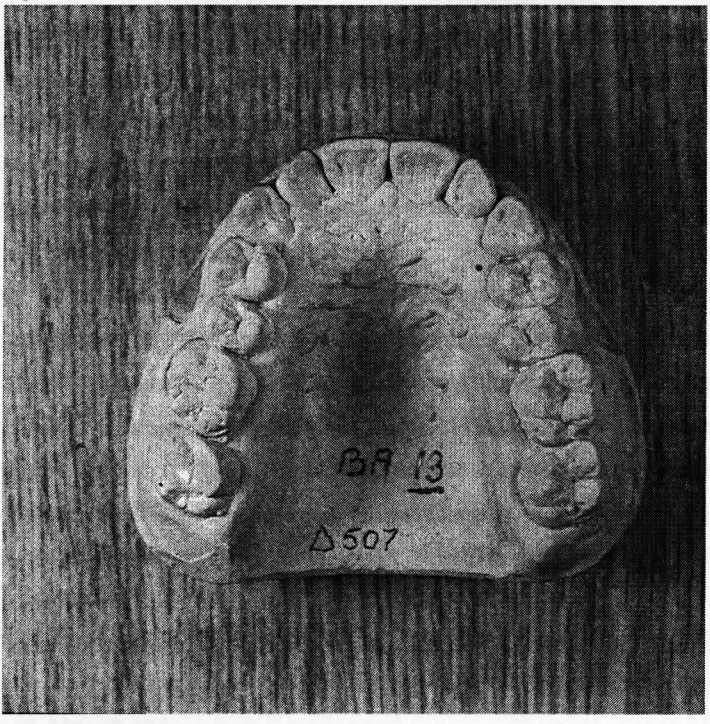


Figure 2. A Yuendumu subject raised at the settlement exhibiting extreme rotation and displacement of incisor teeth, conditions virtually absent from the free-ranging parental generation.

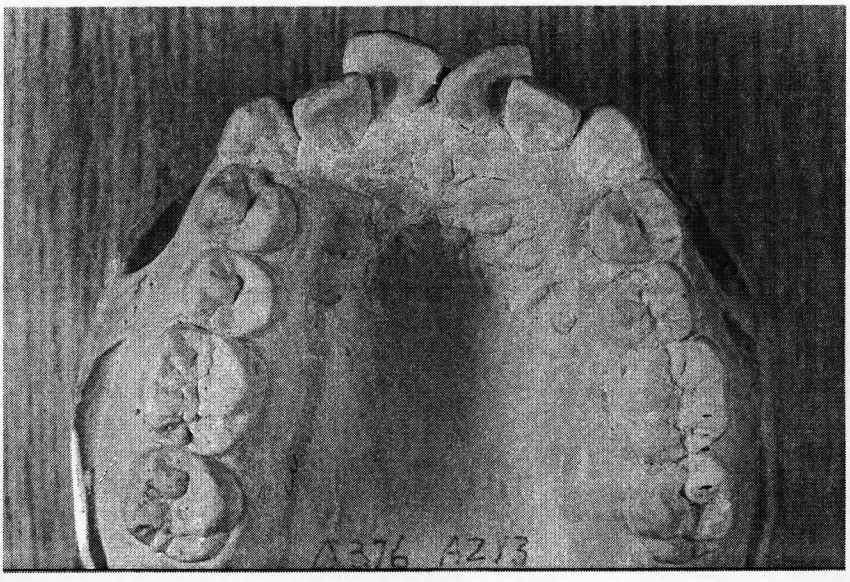


Figure 5. Left side: This female documents the next to largest leeway space (top) in the Yuendumu sample, and indeed a rare situation of spacing results in the permanent dentition (bottom). Furthermore, and in keeping with Begg's model, horizontal incisor overjet decreased substantially between the two phases. This occurred through differential mandibular arch translation more than change in incisor proclination. Paradoxically, there was (top) malalignment and space loss in the deciduous molar dentition. Right side: This male shows the largest arches and permanent teeth (early adult stage, bottom), as well as slightly below-average leeway space (top). There is only slight lateral incisor crowding, and some later correction of that with arrival of the permanent complement of teeth. The large unworn teeth should have represented a case of the worst of Begg's fears regarding crowding. On the other hand there was a large Class II discrepancy.

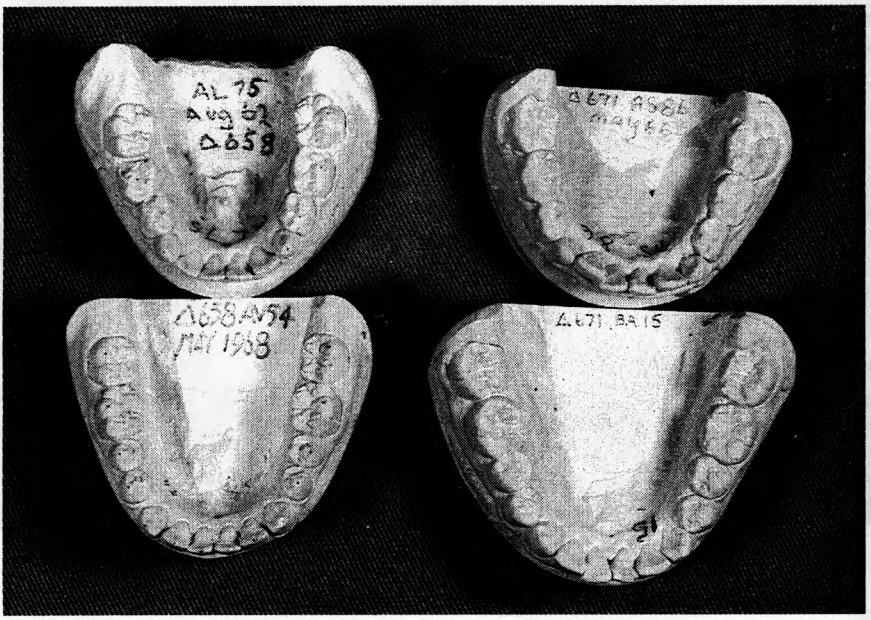


Figure 6. This case shows small deciduous post-incisor teeth (but left canine displacement), and small leeway (but no permanent tooth crowding). Thus the eruption of relatively oversized teeth has unexpectedly resulted in lessened crowding. There did develop, however, a deep bite which is most rare in Australian Aboriginals.

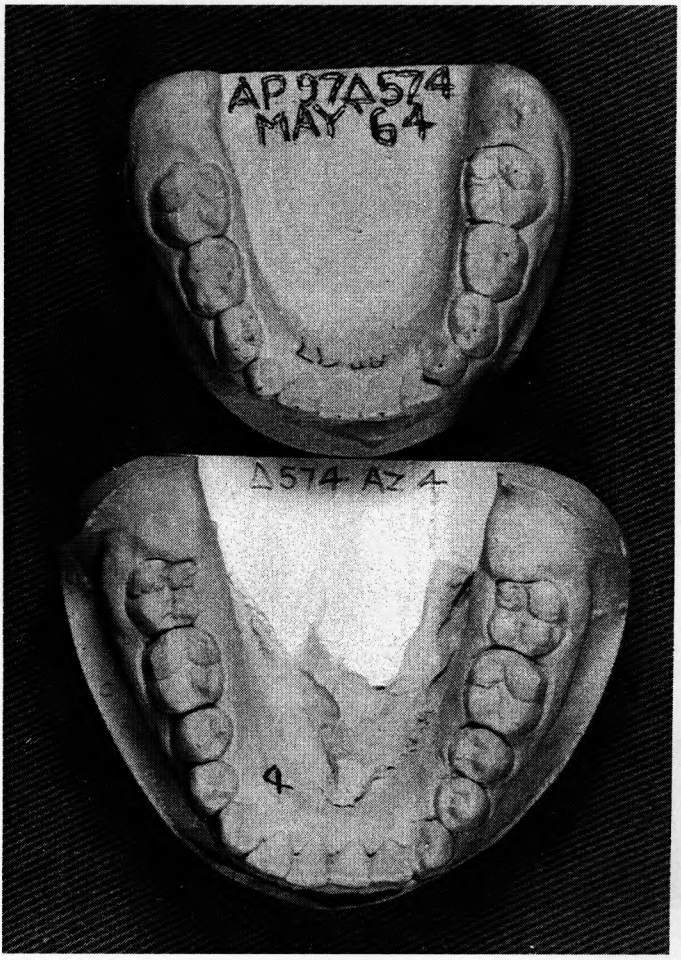
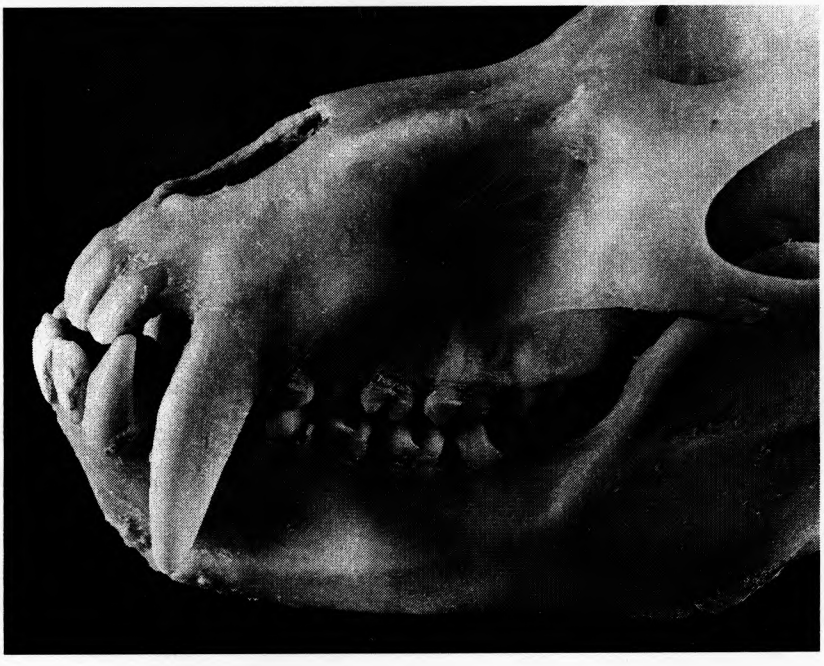


Figure 7. Maxillary arch and palate of an adult squirrel monkey that had been raised on artificially softened food. It displays features common in its soft diet sample of individuals: misaligned and somewhat crowded central incisors, and (slightly) lingually displaced premolars. While the latter feature is quite subtly expressed here (it is clearer in many other specimens of its sample) the contrast is consistent with naturally developing wild-caught squirrel monkeys which show facets indicating symmetrical attrition, correct cusp-to-fossa relationship, and the buccal anteroposterior crests on postcanine teeth all aligning in perfectly continuous manner, occlusally connecting the row of main buccal cusps and crests.

Figure 8. In centric relation and with maximum intercuspation, this baboon raised on soft diet has a full bilateral mandibular mesioclusion ("Class III") and mandibular incisor overjet, extraordinarily rare anomalies in wild monkeys.



5. Effect of Interproximal Attrition: the Begg Concept.

When for argument's sake we entirely exclude genetic changes while considering possible etiologies for extremely sudden rises in malocclusion, then basically two major ideas remain. One is the "disuse" mechanism repeatedly alluded to in this book, while the other concerns interproximal attrition as a tooth-size reducing factor.

In his seminal 1954 article, P.R. Begg reasoned "the relatively low incidence of malocclusion in Stone Age Man is largely due to the reduction by more than half an inch in the total length of each of his dental arches by tooth attrition, so that this lessened amount of tooth substance can be more easily accommodated by his jaws." Begg (e.g., 1965) was convinced that the only anatomically correct occlusion resulted from attrition. "It is a prerequisite that the inherited sizes of all of deciduous and permanent teeth, before they become worn, be greater than can be held by the tooth-bearing parts of the jaws in regular alignment on the centres of the alveolar ridges .... Unless there were excess of tooth substance relative to jaw size, Stone Age man would, early in life, have insufficient tooth substance to occupy fully the tooth-bearing parts of his jaws because tooth attrition is so extensive" (Begg, 1954). Thus Begg seemingly discounted the existence of plasticity in the alveolar region to respond to varying tooth load.

The underlying philosophy of Begg's orthodontic principles, particularly the need to extract permanent teeth to create arch space (as opposed to the earlier, Angle-inspired ideal of retaining all teeth), is supposedly founded on these observations of Australian Aborigine dentitions: "it is necessary to

mention briefly the importance of tooth extractions in orthodontic procedures. Teeth have been extracted extensively in the cases reported in this article -- not because the thin arch wire technique requires more tooth extractions than other techniques, but because extractions should be extensive for purely biologic reasons. This has been explained in detail in a previous article" (Begg, 1956).

While Begg made explicit a theory for malocclusion etiology based on attrition, he was certainly not the first to incorporate the notion into thinking about craniofacial biology. In fact Sicher (1953) foreshadowed Begg's influential writings, concluding "only few peoples and few individuals of most countries show a normal type of attrition. The majority of the population of the civilized world suffers from a lack or an abnormal pattern of wear of the teeth. The often claimed deterioration of the human dentition by the degenerative influence of civilization or 'self-domestication' seems to have its true reason in the breakdown of the delicately correlated and balanced changes in the functioning tooth, changes in which attrition plays a dominant role."

As did Campbell earlier (1925, 1946) and Fishman (1976) and Brown (1985) later, Begg (1954) argued that retention of unworn occlusal-cusp anatomy was not a characteristic of unmodernized populations. Since the cusps wear flat relatively early in life, Campbell and his successors suggest a function of cusps is to guide erupting teeth into proper position and occlusion, rather than being important to mastication. In a similar vein, Mills (1988) argues that mastication is facilitated by having dentin and enamel exposed on the tooth to provide tissues of differing hardness, which only occurs after attrition's action: "If only enamel were present on a worn tooth, the polished surface would be as useless as a glazed grindstone"; thus, "it is difficult to see why the shape of the unworn teeth, with its intricate pattern of cusps and fossae, ridges and grooves, has evolved."

Even earlier, Sim Wallace (1904) foreshadowed some of these views: ".... There are other subsidiary effects of a diet which requires considerable mastication. The teeth do not rise unduly in their sockets, and the alveolus does not tend to become disproportionately deep. The cusps of the teeth get worn

down and the vertical diameter of the mouth is diminished. Increased rubbing of the teeth on their approximal surfaces diminishes the so-called mesio-distal diameter of the teeth, thus reducing the whole length of the arch by at lease one or two millimetres .... With regard to the diminishing of the vertical depth of the mouth, I have already advocated general decuspidation, more especially in relation to the prevention of caries. The operation must, however, not be carried too far, for during the eruption and arrangement of the teeth the cusps are of great importance in regulating the arrangement, and the amount of decuspidation should not much exceed what might normally have taken place."

Begg's specific figure of 10.54 mm loss of mandibular tooth material (in the mesiodistal axis, due to attrition prior to age 18) has been widely repeated in the anthropological and orthodontic literature (see e.g., Dickson, 1970; Wolpoff, 1971; Brace, 1977; Murphy, 1964; Fishman, 1976; Beyron, 1964). These reproductions occasionally appear together with an emphatic denial of the importance of other possible mechanisms decreasing the arch-versus-tooth size relationship, since the huge relative arch change implied by the above figure dwarfs other imaginable factors. Berry (1976) and Lombardi (1982) have recently re-endorsed some of Begg's thinking. The universal acceptance of Begg's notions is not clear from a survey of orthodontic textbooks. Moyers (1988) mentions a few of the specific components of Begg's mechanism but without specifically citing the theoretical writings; he also points out that some of the mesial drift phenomena Begg attributed to attrition also occur systematically in patients lacking attrition. Proffit (1986) points out that Begg didn't explain how late crowding (circa age 17-18) comes about; modern Australian Aboriginals (no longer experiencing marked attrition) still fail to develop this while U.S. whites with prior extractions still do. Kesling (1988) supports Begg's ideas. Ramfjord and Ash (1971) mention the idea but cite Beyron (1954) rather than Begg. Salzmann (1974), Sassouni (1971), Nanda (1983), Thurow (1977), Dawson (1974), Wheeler (1974), and Graber (1972) cite Begg, if at all, for his correction technique only. Lundstrom (1960) opines that the role of abrasion in malocclusion etiology "has

not been established."

There are theoretical and practical problems with the Begg model. The theoretical difficulties revolve around the integration of deciduous versus permanent tooth attrition with the sequence of deciduous tooth replacement. If rapid wear of the permanent premolars is to make room for the canine and distal molars to erupt, equally rapid wear on their deciduous predecessors would have closed down the "leeway space" between permanent incisors and first molar between the ages of c. 6-12 and threatened canine-premolar impaction. On the other hand, lesser abrasion on the deciduous canine and molars cannot be invoked as a space-creating mechanism for the eruption of the permanent incisors and as a means of enhancing Class I relationship for the first molars. Begg and Kesling (1977a) assert that extensive deciduous tooth wear allows M1 to erupt mesially into a Class I rather than cusp-to-cusp relationship. Lack of this wear only logically leads to Class II disorder, and cannot explain the minor Class III displacement that is relatively frequent in modern Australian Aboriginals and actually more frequent than Class II in most non-Western populations (Corruccini, 1984). These contradictions have been noted before (Lombardi, 1982; Corruccini, 1984; Corruccini and Lee, 1984; Dawes, 1986), and Begg was somewhat vague concerning whether deciduous or permanent teeth show the most relative attritional loss in his scheme.

Practical attempts to repeat Begg's research, in Australian Aborigines and in prehistoric Amerindians (Murphy, 1964; Beyron, 1964; Fishman, 1976; Dawes, 1986; Kaul and Corruccini, 1990), have consistently produced estimates of permanent mandibular tooth reduction (mesiodistal, prior to third molar eruption) of only 20 percent to 40 percent of Begg's celebrated figure of 10.54 mm. Fishman (1976) was surprised to find more dental crowding and incisor horizontal overjet with increasingly severe attrition. "Begg's contention that attritional occlusion is more normal and that lack of attrition in civilized society is the primary etiologic factor in malocclusion, is worthy of consideration although this conclusion is not clearly evident in this study. Many excellent occlusions

existed in skulls that demonstrated both slight and advanced attrition" (Fishman, 1976).

Dawes (1986) has considered this situation in the most detail. He finds many uncrowded Aborigine occlusions that lack wear as well as crowded dentitions showing much wear, concluding that 3.0 mm rather than the rarely questioned standard of 10.54 mm should be considered a maximum estimate for immature tooth reduction, and that this amount could not palpably improve premolar or incisor crowding (although, as did Fishman, he thought it might lessen third molar impaction). Dawes goes on to provide stunning documentary evidence of gradual, unconscious exaggeration of this attrition in Begg's database over the more than 25 years that Begg considered the issue.

Testing Begg's Theory

In assessing the particulars of Begg's theory, the problem becomes defining exactly how tooth reduction interacts with the eruption sequence to bring about improved occlusion, and formulating explicit metrical hypotheses for the emergence of morphological patterns of dental occlusal variability in relation to tooth and space loss or excess. In view of the broad acceptance of his quantitative assessments and variable acceptance of the ideas on etiology, detailed reexamination of Begg's theory is currently timely. The previously described collection of dental casts obtained from Australian Aborigines living at Yuendumu settlement in the Northern Territory of Australia (Campbell and Barrett, 1953; Barrett, Brown and Fanning, 1965), enabled a controlled investigation of occlusal changes through the critical developmental period (Corruccini, 1990), in the same general human variety upon which Begg based his thinking.

The Aborigines' dental eruption schedule is well documented (Barrett, 1957a, 1957b; Barrett et al., 1964; Brown, 1978; Brown et al., 1979). This population shows considerable crowding changes plus poor predictivity of later crowding from the late mixed dentition stage (Barrett, 1957; Sampson and Richards, 1985). Again, caries experience of the Yuendumu people was low

(Barrett, 1956; Barrett and Williamson, 1972). Thus this population is not exactly comparable to the pre-contemporary skeletal accumulation used by Begg, since it shows, among other features, somewhat smaller tooth size (Barrett et al., 1963; Townsend and Brown, 1979).

The absolutely critical feature of the younger Yuendumu subjects is their minimal occlusal and interproximal attrition displayed prior to middle adulthood. Thus these subjects possess the genetic tendency for tooth/arch size of their population, yet show almost none of the attritional progression considered vital by Begg. This uniquely allows testing of a number of hypotheses implied if not explicit in Begg's model (not all of which are consistent with one another), specifically:

(a) Larger permanent teeth overall will promote occlusal disorder, especially crowding and displacement of the arches;

(b) Larger deciduous canine and (pre-) molar dentition will intensify anterior (especially permanent incisor) crowding;

(c) Larger permanent canines and premolars within the leeway space will promote post-incisor crowding;

(d) A smaller leeway space arrangement (difference in deciduous tooth size and that of permanent successors) will promote more crowding, and more malocclusion in general, and inhibit natural improvement of anterior alignment. This rather static view, incorporating tooth-size relationships only, may be unrealistic in not recognizing malocclusion as a multifactorial process also involving interaction among alveolar development, arch growth, and trajectory of eruption along with natural mesial migration. However, it does directly test Begg's hypothesis;

(e) Larger anterior horizontal incisor overjet, both of the early mixed dentition and in the adult occlusion, will promote permanent tooth crowding (especially mandibular). Since earlier attainment of the "correct" edge-to-edge incisor relation, according to Begg, would create more arch space, a corollary expectation is for a larger difference between early mixed stage and early adult stage overjet

(physiological repositioning) to promote good occlusion.

Coincidentally, this research situation allows testing for two other malocclusion etiologies of longstanding interest. Premature deciduous tooth loss (PDTL), as reviewed by Lundstrom (1948) and Corruccini, Potter and Dahlberg (1983), has been mentioned often as promulgating disturbed occlusions. The idea is supported in Moyers' (1988) text but not in Proffit's (1986). The longitudinal castings of Aboriginals allow a measure of relatively early exfoliation and its relation to the adult occlusion. Also with a deep history is the idea that increased variation in occlusofacial structures, and decreased covariation or correlation (growth integration) among those structures accompanies increased prevalence of malocclusion (see Phipps et al., 1988, and Corruccini and Beecher, 1984 for reviews). This relates to the considerable literature on the relation between deciduous and permanent tooth size in general (e.g., Brown et al., 1980a, 1980b; Moorrees and Chadha, 1962; Moorrees and Reed, 1954, 1964). Thus:

(f) Higher PDTL scores will associate with more crowding and displacement of permanent teeth or with heightened malocclusion in general;

(g) The more maloccluded Aboriginal individuals will show larger sample variance and decreased variable-pair correlation among tooth size, arch size and deciduous to permanent analogs, than will the well-occluded subsample. It is not immediately obvious that this result is logically expected from the Begg mechanism although sometimes this is implied.

Note that the longitudinal framework of these hypotheses, made possible by the invaluable Yuendumu collection housed in the School of Dentistry of the University of Adelaide, contrasts with the uniformly cross-sectional approach (based on only 9 worn specimens) used by Begg. "Dental occlusion constantly changes throughout life in response to changing functional requirements. Observations limited to cross-sectional material provide an incomplete, and sometimes misleading, concept of dental occlusion and masticatory function" (Brown et al., 1989).

A sample of 50 Yuendumu Aboriginals was selected from individuals

that entered the study relatively late and thus were relatively habituated to the settlement environment during their growth/development. These were 25 males and 25 females. Advanced attrition was lacking in all these. For each, the cast representing the earliest mixed dentition period (permanent incisors and first molars fully erupted; deciduous canines and molars still in place; age 6-7), and that of the earliest adult dentition (all permanent teeth fully erupted exclusive of M3; age 13-15) were selected.

To address the above hypotheses the following measurements were taken (on the left side unless there was arch distortion or a missing tooth):

(1) Deciduous tooth chord, the mesiodistal length along the curve of the arch of the mandibular deciduous canine and (pre-) molars;

(2) Permanent tooth chord, the same length of the succeeding mandibular permanent canine and premolars;

(3) Leeway space, the difference of (2) from (1). This variable thus conforms with some but not all impressions of "leeway space" in that it is purely a sense of tooth size differential, not corresponding arch size;

(4) First permanent mandibular molar mesiodistal length;

(5) Total post-incisor permanent tooth size, the sum of (4) and (2);

(6) Deciduous chord crowding, the amount of mesiodistal tooth length needing to be added to (1) to make the chord from mesial canine to distal second molar equivalent to the sum of individual tooth lengths (a negative corresponding value was entered for spacing);

(7) Permanent chord crowding, the same as (6) for the permanent teeth erupting into the leeway space;

(8) Mandibular arch width, between the buccal extremities of the first permanent molar taken on the adult cast;

(9) Maxillary arch width, across upper first molars in the adult stage;

(10) Transverse molar relation, the difference between (9) and (8).

(11) Early incisor horizontal overjet, the anterior distance from the labial surface of the permanent maxillary central incisor perpendicular to the labial surface of

the corresponding mandibular central incisor, taken on the early mixed dentition stage cast;

(12) Adult stage incisor horizontal overjet;

(13) Overjet adjustment, the difference between (11) and (12) reflecting relative anterior repositioning of the mandibular incisors toward the edge-to-edge condition;

(14) Postcanine permanent mandibular tooth crowding, based on the rotation/displacement sum detailed for Treatment Priority Index calculations (see Kelley and Harvey, 1977), scored as 0 (none), 1 (some notable rotation/displacement) and 2 (2 or more teeth malaligned or one tooth badly malaligned, i.e. displaced more than 2 mm or rotated more than 45 degrees), with some fractions introduced subjectively in intermediate cases;

(15) Anterior (incisor and canine) permanent tooth crowding, scored from 0-2 as in measurement (14);

(16) Total crowding, the sum of (15) and (14);

(17) PDTL 1, a measure of anomalous exfoliation of the mandibular first deciduous molar relative to the appearance of the permanent canine. In this population these events are ordinarily roughly contemporaneous, thus the score was 0 for contemporaneity, 1 for a somewhat earlier molar loss and 2 for molar loss one year or more in advance of canine eruption. Cumulatively for both sides, the score can range from 0 to 4. Yearly casts through the age range of 5-15 were examined for PDTL variables;

(18) PDTL 2, scored the same way but comparing deciduous second molar loss with permanent second molar eruption;

(19) Total PDTL, the sum of (17) and (18).

All casts were judged with arches in maximally intercuspated centric occlusion. "Malocclusion" of a Class II or III variant of buccal segment relation, or of posterior crossbite was diagnosed if a cusp-to-cusp or worse discrepancy occurred (compare to Baum et al., 1973; Kelley and Harvey, 1977), or if openbite, or deep bite of more than 7/10 of the vertical height of the lower incisor

crown, or if a maximal crowding score was observed for either anterior or posterior teeth. In this way a subsample of 15 notably "maloccluded" subjects (8 with Class II or III, posterior crossbite, or incisor overbite or overjet discrepancy; 7 with high rotation/displacement scores) was distinguished from the 35 remaining subjects.

The hypotheses above were considered one-tailed and were tested with product-moment correlations between the pairs of relevant variables over the total sample, or by the t-test between the mal-occluded and well-occluded subsamples.

Hypothesis (a) could be tested by correlating tooth size and crowding, specifically variables (3), (4) and especially (5) with (7), (14), (15) and especially (16). With n=50 and a one-tailed hypothesis, a positive correlation of merely 0.24 is required to reject the null hypothesis at 5 percent probability, and this would represent only 6 percent shared variance between two variables. Therefore, expectations were high to find correspondence with Begg's theory if there is any biological reality to it. Even with such test conditions, none of these correlations approached significance. Similarly, mean permanent tooth size was very similar among occlusally different Aboriginal subsamples.

Deciduous chord (1) and permanent anterior tooth crowding (15) had a non-significant negative correlation; this held also for crowding within the teeth making up the deciduous chord (6); again, (1) was not larger in the maloccluded group (hypothesis b).

Permanent tooth chord (2) failed to approach a significant correlation with posterior crowding (14) or any other crowding measure, even of the teeth constituting the chord (7). Hypothesis (c) was not supported.

Hypothesis (d) concerning favorable leeway space is perhaps most crucial to Begg's model. Leeway space (3) did not attain a significant correlation with crowding measures (7,14,15,16) and was not significantly smaller (although it is somewhat smaller) in maloccluded individuals. The crucial relation was the expected negative one between leeway space and total crowding, for which r=-0.095.

Hypothesis (e) relates overjet variables to occlusion. Early mixed stage overjet (11) did consistently correlate positively with all the five crowding variables (6,7,14,15,16) and did so significantly with anterior and total crowding scores. The highest of these correlations represented 9 percent shared variance. Adult overjet (12) behaved similarly and correlations attained significance with (7), (15) and (16); the highest of these was a substantial $r=+0.477$ with anterior tooth crowding score. Most importantly, however, overjet change (13) did not show theoretically expected significant negative correlations with crowding. The strongest relation was $r=-0.122$ with anterior crowding. Furthermore the mean anterior adjustment of overjet, while smaller (actually, a larger negative, hence anterior maxillary adjustment) in maloccluded subjects, did not differ significantly. Both overjet measures were larger in the maloccluded, in accord with Begg-based expectations; the adult overjet difference made the closest approach to statistical significance ($p>0.09$).

Parenthetically, I noted that overjet adjustment correlated significantly with leeway space which might bear a logical relation to Begg's thinking. Unworn dentitions, in which large leeway simulates the beneficial spacing effects of permanent tooth attrition, do close down the overjet somewhat during this transitional phase.

PDTL scores (hypothesis f) showed no significant positive correlations with crowding; the summary measures (16 and 19) gave $r=+0.102$. On the other hand, mean total PDTL score was significantly elevated in the generally maloccluded sample ($p<0.025$) which showed other discrepancies in addition to simple crowding.

There were consistent variance/covariance differences in the 11 metrical (toot or /arch size) variables (1-5 and 8-13) among maloccluded and normal samples. All traits were more variable in the maloccluded subjects. Four of the variance ratios individually rejected the null hypothesis of equal variance: total permanent tooth size, permanent tooth chord, early and adult overjets. Bartlett's multivariate test rejected equality of the variance/covariance matrices at $p<0.0001$.

Pairwise correlations among these variables were almost all higher in the sample of occlusal normals; in particular, normals were higher correlated for deciduous vs. permanent tooth chords, mixed vs. adult overjet, maxillary vs. mandibular arch breadths, and other such logically related pairs of traits.

Although it does not relate to any of Begg's specifics, there was significant relative narrowing of the maxillary arch (10) in maloccluded individuals. This corroborates a general trend in many different populations (Staley, 1985; Corruccini and Whitley, 1981; Corruccini and Lee, 1984; Corruccini et al., 1983; Corruccini, Townsend and Brown, 1989).

Gender differences were significant for all tooth and arch size variables (except for deciduous chord), and for adult overjet, but not for any of the derived variables or scores with the lone exception of leeway space (larger in females). Therefore gender heterogeneity within the sample would not seem to be related to the patterns of variation discovered above, although it was interesting that sex-dimorphic permanent teeth succeeding non-dimorphic predecessors seemed to cause the reverse dimorphism of leeway space; this is characteristic of most populations in the mandible but not in the maxilla (Brown, Margetts and Townsend, 1980a). This sex contrast was not related to any difference in crowding or occlusal variables. Corruccini et al. (1990) found no sex differences in occlusion in Yuendumu subjects.

This study longitudinally compared tooth and arch variables in Australian Aborigines lacking interproximal attrition, to test aspects of Begg's theory. This approach was founded on the relation between tooth size, succession, and crowding being more pivotal and attainable than relations with attrition -- for who can say, in individual worn cases, what would have happened without the attrition?

Only adult-dentition incisor overjet in the absence of attrition bore any relation to crowding status. There seems to be something to the idea that failure of attrition and mesial drift to bring mandibular incisors toward the anterior plane of maxillary incisors, relates to increased crowding tendency especially of anterior

teeth. Of course, causality is not directly demonstrated by this correlation. It may be, for instance, that an independently determined mandibular crowding tendency shortens the lower arch, thereby increasing the maxillary overjet. Brace (1977) attributes the disappearance of edge-to-edge incisor relations in modern populations to use of cutlery rather than absence of attrition.

Other major features of the model posed by Begg could not be verified. Absolutely or relatively longer teeth did not relate to crowding in general, or in the relevant local areas or developmental stages. Unfavorable leeway space, though uncorrected by permanent canine-premolar attrition, did not relate clearly to crowding or other malocclusion syndromes. Moorrees and Reed (1954) did not agree with the opinion that crowding is related to absolute size of the teeth, as inferred by Lundstrom (1923, 1952). A similar view has been expressed by Hunt (1960). Others have reported unclear relations between leeway space and crowding (Moyers and Wainright, 1977; Hunter, 1977; Sanin and Savara, 1972; Arya et al., 1973).

On the other hand, mandibular incisor mesiodistal size (not tested in the present study) does seem to positively correlate with crowding of those teeth (Begg, 1935, Begg and Kesling, 1977) as verified metrically by Sampson and Richards (1985). However, Sharma and Kaul (1977) observed that in the Punjabi dentition, contrary to Moorrees (1957) and Begg (1954), wear did not contribute much to the development of an edge-to-edge occlusion. Brace and Mahler (1971) are of the view that wear is not essential to produce an edge-to-edge bite since this form of occlusion can be observed in individuals where wear is near absent. They suggest that the simple habit of holding food while it is being incised apparently prevents over-eruption of both the maxillary and mandibular incisors which create the overbite. Symphyseal growth also relates to overbite syndromes (Beckmann et al., 1998).

As alternative etiologies, PDTL and/or unspecified mechanisms narrowing the maxilla, increasing craniofacial variation and decreasing morphological integration (correlation) would be more convincing not only in this

study, but in general throughout my work. None of these causes assist clearly in explaining the most prevalent single expression of malocclusion, which is simple crowding leading to tooth rotation/displacement. If Begg's theory fails in explaining this as an outcome of teeth too large for the available alveolar arch space, then mechanisms must be energetically sought to explain arches too small to accommodate the teeth in modern populations (see Howe, McNamara and O'Connor, 1983).

The metrical and statistical points made in this analysis are evident from qualitative evaluation of individual cases. The largest and smallest individual leeway spaces in the sample fail to correspond to malocclusion patterns. I doubt whether clinical analysts would differ from my impression that tooth and leeway size lack consistent relation to crowding status. How the leeway is utilized is clearly critical, and this involves varying amounts of alveolar growth between the ages of early mixed and early permanent dentitions in the figured cases, a factor scarcely discussed by Begg.

Perhaps the effect of leeway space variation should not be ruled out at the inter-population level even if it is vague at this individual within-population level. Yuendumu Aborigines average 1-2 mm larger leeway space than generally worse occluded whites (Brown et al., 1980a,b). An issue is whether there is straightforward proportionality between the Aborigines' larger tooth size and the larger leeway, tentatively discussed by Margetts and Brown (1978).

The Formulation of Begg's Theory

Begg (1954) delivered a clear clinical policy statement: "as Stone Age Man's dental arches were continually reduced throughout life by toothwear, orthodontists have a well-founded precedent for reducing dental arches by tooth extraction and for discarding the practice of arch expansion whilst retaining the full complement of teeth."

Begg's concern with this issue may have influenced his data, rather than vice versa. This is a common enough influence upon us all in science (Gould,

1981) and indeed, the present instance recalls the unconsciously selective data handling treated in Gould's celebrated expose of 19th-Century craniometricians. As revealed by Dawes (1986):

> "Concerning the work of P.R. Begg, two age-related interproximal attrition assessments of the permanent Aboriginal dentition were carried out; the first being contained in his 1935 doctorate thesis Part IV (Tables 18 & 19) and the second published in 1954 (Tables 20 & 21). His calculations in 1935 were based on 154 unworn teeth and 98 worn teeth, the worn set being from nine mandibles at the development stage just prior to eruption of third permanent molars, and total arch loss amounted to 7.36 mm. His calculations in 1954 were based on 154 unworn teeth as earlier used and 126 worn teeth. Although Begg did not advance any explanation it is evident that some arches of less perimeter length were selected for the 1954 calculation, which earlier had not been included. Total arch loss amounted to 10.54 mm. To add to the uncertainty, he stated that the 1954 results were 'determined from measurements taken in 1930' (1965 P. 22 Chapter 2)."

The worn sample had been incremented from only 9 partial dentitions to 9 full dentitions (which with 2 teeth per side and 7 permanent teeth, resulted in the seemingly large sample of 126 teeth). Average summed worn tooth size (per side) decreased from 58.38 mm in 1935 to 56.78 mm in 1954. While only some of the tooth classes had the sample filled out in 1954 (preventing clear establishment of what happened) the 43 percent increase in attritional loss postulated in the later figure must have involved both adding and substituting specimens.

Dawes (1986:241) continues:

> "Further, the material used by Begg was from Museum sources that had earlier (1925) been used by Campbell for his measurement tables (Table 14 Section B). Had Begg used Campbell's 1925 mean measurements for unworn

teeth, totaling 645 observations whereas Begg had only 154, the 1935 result would have been reduced from 7.36 mm to 4.04 mm and the 1954 result reduced from 10.54 mm to 7.24 mm. Unfortunately Begg's 1954 result of 10.54 mm appears to have remained unquestioned and has been utilized as the standard for interstitial wear in this age group in both anthropological and dental literature. "

Begg's mean unworn tooth size sum (one side) was 62.06 mm in both 1935 and 1954. This mean is only 60.4 mm in the total sample used by Campbell (1925), and less yet (57.5 mm) in the relatively small Yuendumu teeth (Barrett et al., 1963). Thus in all likelihood Begg happened upon cases of both unusually large unworn teeth and unusually small worn teeth.

Dawes (1986) pinpoints a few additional curiosities in the evolution of Begg's thinking between 1935 and 1954. For instance, Begg (1965) derided "textbook normal" occlusal relations because they are nonattritional and "actually constitute a gross malocclusion" (Begg and Kesling, 1977b). However, Begg had the faster mandibular attrition rate moving lower teeth forward such that the deciduous second molars should be in a proper Class I relationship when permanent first molars erupt. The latter then, supposedly, had more room and also tended to assume Class I as soon as occluded. Yet Begg in 1935 stated Aborigine crania bearing deciduous teeth had flush terminal plane relationships, and first molars were nevertheless erupting directly into Class I relationship. Furthermore, contemporary Aborigines such as those at Yuendumu today no longer develop the marked attrition, yet still erupt first molars directly into Class I relationship! It has been widely accepted from Angle's time onward that forward eruption of permanent first molars due to carious or prematurely lost deciduous molars must be strenuously avoided, to avoid space loss and premolar maleruption -- yet Begg's attrition mechanism would cause such loss.

Relatively anterior eruption of mandibular first permanent molars in whites is known to correlate with good and stable Class I relations, but this

happens infrequently compared to Aborigines. In whites with frequent cusp-to-cusp early M1 relation, some of the mandibular leeway space is absorbed if the M1 moves mesially into Class I position subsequent to eruption, limiting the time available to the permanent canine/premolar segment to correct its occlusion (B. Dawes, pers. comm.).

Begg and Kesling (1977a) also were very explicit in calling for wear of permanent incisors and premolars to make room for the canine's eruption and to avoid further anterior crowding. Enough wear to have such an influence is impossible, especially as on average the canine in Aborigines erupts at about the same time as premolars (Barrett, 1957a, 1957b; Barrett et al., 1964; Brown, 1978; Brown et al., 1979). In attempting to document extremely rapid interstitial tooth reduction so soon after eruption, Begg (Dawes, 1986:256) mistakenly figured an unworn first molar as portraying a large mesial wear facet; the in fact incompletely erupted tooth was merely showing the ubiquitous Aborigine mesial curvature. In the final irony Dawes (1986:172) shows how Begg in 1935, using largely the same data as in 1954, concluded that "in most cases showing 'crowding' of the teeth in the Australian Aboriginal skulls, attrition, is just as well marked as it is in cases having teeth in regular alignment and normal occlusion".

There is no biologic or evolutionary justification for deciding about tooth extraction in these historical and metrical findings, and certainly there may be other (non-etiological) considerations that justify extractions. We emphasize that we are making a biological, not clinical argument. Insufficient alveolar or arch growth may be a valid motive for extraction. Howe et al. (1983) suggest alternative clinical strategies such as functional appliances or palatal expansion may make more sense in view of alternative etiological viewpoints to the belief in excessive tooth size. Radnzic (1988) and Forsberg (1988) also review variable or slight correlations between tooth crowding and tooth size in numerous studies. Radnzic (1988) suggests it would be simplistic to employ such results in treatment recommendations.

To simulate natural processes, one might wonder why tooth shortening

through grinding occlusal surfaces, and enamel stripping the mesial and distal facets, would not be more appropriate and less injurious than extractions. This possibility in a preventive context is referred to sporadically (e.g., Miethke and Behm-Menthel, 1988; Peck and Peck, 1975). Ramfjord and Ash (1971) suggest this is often recommended but potentially harmful if the interproximal papilla is infringed upon. Dickson (1970) states: "Many orthodontists accept that a proportion of the present population cannot accommodate in their mouths a full complement of teeth and that in these cases it is necessary to imitate the effects of this wear by removing a premolar on each side of both dental arches. So far there seems to have been no general attempt to imitate the other part of the situation by grinding off the cusps, although I did hear of an eminent dental surgeon incorporating pumice powder in his home-made bread for this purpose!"

Begg and Kesling (1977b) go into more detail:

> "Some day, as a partial solution to this problem, orthodontists may augment their mechanotherapy with some form of equilibration and continual reduction of tooth substance. This could be accomplished by mechanical stripping and grinding at regular intervals carefully related to each patient's rates of eruption and mesial migration. Patients could be instructed to use chewing gum containing varying amounts of tough roughage and carborundum dust to wear away cusps and proximal surfaces. If civilized man's teeth were subject to such controlled attrition, orthodontists would not have the problem of orthodontic extraction spaces sometimes remaining slightly open after treatment."

I question whether interproximal spacing alone would have any equilibrational effect without the added action of strong mastication. Aboriginals have not only gritty diets but tough gritty diets, and the chewing forces contribute to the mesial migration. The minute up-and-down movements between teeth that engender interproximal attrition undoubtedly are less with a soft diet and

unforceful chewing.

Contrary to Begg's incorporation of attritional features in his picture of normal occlusion, severe interproximal tooth loss cannot eliminate all developing occlusal problems, and probably causes some (Fishman, 1976). It may however alleviate third molar impaction. Nevertheless, Aborigines tend to make more room than whites for M3 through mesial tooth migration even in the absence of attrition (Bjork et al., 1984). Begg's 1954 assertions lacked "the restraint and degree of accuracy evident" in the progenitor 1935 work (Dawes, 1986). Nevertheless, this reexamination in terms of perhaps more modern concepts of scientific method is not intended to reflect adversely on Begg's important contribution to practice and thought in his time, nor on his vital longterm influence over orthodontic theory and technique.

The Amount of Tooth Substance Lost

Begg's promotion of the direct association between interproximal attrition and the provision of space in the arches has led occasionally to the general belief that the Australian Aborigine shows the most extensive wear among studied native groups (e.g., Wolpoff, 1971). Begg's result has been transmitted and utilized by many (e.g., Wolpoff, 1971; Brace, 1977). Further attempts by Beyron (1964), Fishman (1976), Murphy (1964) and Dawes (1986) have failed to attain consensus yet agree that the magnitude of Begg's assertion is questionable. We (Kaul and Corruccini, 1990) went further by utilizing the collection of Yuendumu dental casts to take a different approach to estimating attrition loss in the developing dentition in this native Australian population.

From the longitudinal cast series 25 pairs of mandibular casts (18 males, 7 females) were drawn for study (Kaul and Corruccini, 1990). For each pair these comprised a younger stage (earliest adult dentition, just after M2 eruption) and later stage (latest casting of permanent teeth to maximize years elapsed from the younger stage). Total age range was 11-23 years at the first and 18-36 years at the second castings. A second sample of 36 single casts (20 males, 16 females) of

older individuals born no later than 1946 but retaining almost all teeth, was then selected to maximize chronological age difference between younger and older samples. Age ranged from 25-50 for these.

Total mesiodistal diameters of permanent incisors through canine (anterior mesiodistal tooth chord) and premolar through second molar (posterior mesiodistal tooth chord) were recorded on the left side of each mandibular dentition using a dial caliper with sharp points. The two chords encompass the major point of curvature of the arch and approximate the total mesiodistal arch length. In the event any teeth were missing, or damaged due to caries, measurements were taken on the other side for both stages of an individual. Measurement error amounted to 1.07 percent for a sample of eight repeats.

The older individuals would have been already living at the settlement, rather than pursuing a traditional foraging life with high potential for interproximal attrition, for about 5 years on average prior to casting. In order to give all benefit of the doubt to Begg's hypothesis and maximize attrition estimates, we subtracted 5 years from each such individual's age such that their observed tooth size (and assumed attrition) would have been attained that much earlier when combined for analysis with the later of the two observations on individuals from the longitudinal sample. Furthermore, females demonstrated greater attrition than males by a surprising 25-50 percent when contrasting younger with older castings of individual subjects. To eliminate sex difference all variables were converted to female using the male-female mean difference to adjust male observations, again a decision to maximize attrition estimates.

Attrition estimates could then be approached in two ways. First, by adding the anterior and posterior tooth chords for a measure of total mesiodistal tooth material, annual attrition loss could be estimated in the longitudinal sample by regressing early-minus-late total tooth material on years elapsed between observations. Since permanent teeth reside in the mouth a maximum of 13 years between ages 6 and 18, the annual rate of tooth reduction multiplied by 13 yielded a maximum estimate of loss of permanent tooth material. Of course this is a

considerable overestimate because many teeth are in occlusion for much less than this time, but estimates were deliberately maximized because (a) young Yuendumu individuals had reduced attrition rates due to part-time reliance upon settlement food, and (b) the younger generation may have been reduced in tooth size compared to the free-ranging older generation, due to fluoridated water supplies at Yuendumu.

Total tooth loss in the longitudinal series of matched observations was about 0.9 mm, on the order of 0.1 mm per year on one side of the mandible.

Calculating linear regression equations, a rate of 0.118 mm/year of attrition tooth loss was obtained between matched pairs. Roughly the same slope resulted from regressing age and tooth chord in younger pair members pooled with the unmatched older individuals. Adjusting the latter for no attrition over their last 5 years and then pooling with the older members of matched pairs resulted in a substantially higher rate.

Crudely, total dental arch reduction could be estimated by multiplying the slope by 26 (2 sides times 13 tooth-years between ages 6 and 18). The maximized total mandibular estimates of loss due to attrition thus came out to about 3-4 mm when calculated for both sides, which is less than half of Begg's central estimate.

A more rational central estimate from our data, restoring males to the sample and substituting actual occlusal years per tooth class, would only be about 2 mm total loss (95 percent confidence limits: c. 1.3-3.4 mm). This would be somewhat underestimated due to reduced tooth size (Townsend and Brown, 1979) and less abrasive food recently at Yuendumu. Probably a general figure of c. 3 mm is reasonable from these data, consistent with the midpoint of Dawes' (1986) ultimate estimate of 2.4 mm and Murphy's (1964) of 3.6 mm (maxillary).

From his study of Australian aboriginal material, Begg (1954) observed that by the time the third molar starts to approach occlusal relationship, attrition has reduced the length of the dental arch by practically a millimeter per tooth, amounting in total to about the length of an unworn molar. Begg (1956) used this

to justify widespread extractions as a pre-orthodontic routine in the Western population experiencing no attrition, in order to make room in the arches for what he saw as the genetically programmed amount of tooth material. To Brace (1977), Begg's estimate of loss due to attrition is accurate for virtually all prehistoric and most non-industrialized modern populations.

Our analysis in Yuendumu Aborigine materials indicated an estimate of about 3 mm, but certainly no more than 4 mm, as the total mandibular loss in tooth length in the immature dentition due to attrition and mesial movement just prior to third molar eruption. That this estimate is more justifiably looked upon as being 3-4 mm rather than Begg's estimate of 10.54 mm is also indicated by Murphy (1964), Beyron (1964), Fishman (1976) and Dawes (1986). Thus, uncritical acceptance of the large Begg estimate, and its influence on thinking about malocclusion etiology and treatment, is no longer appropriate.

Our study was geared toward determining the maximum possible estimate of attrition in these materials (which are not necessarily reflective of Australian Aborigines in general nor of prehistoric humans), in order to examine Begg's celebrated study. While the method was less than ideal, we feel it goes a step further than the prior norm of cross-sectional designs based on variably collected prehistoric crania. Thus, while neither this nor any collection to our knowledge can provide the perfect study, our longitudinal aspect and larger sample sizes can help put into new perspective an old idea that needs rethinking.

6. Experiments Using Non-Human Animals.

Clearly, for all craniofacial growth and development studies animal experimentation is indispensable because such experimentation on humans is not feasible. Harvold (1968) has emphasized that theories and assumptions concerning the relationship between orofacial form and function be given credence or accepted as "general rules" only after adequate evidence is established through well designed animal studies. He pointed out, through animal experiments, that narrow and broad dental arches are respectively associated with micro- and macroglossia, and with a missing tongue the arches may completely collapse. Vargervick (1979) has also arrived at somewhat similar conclusions with experiments on the rhesus monkey, studying muscle influence on dental arch width. Narrowing of the maxillary arch occurred in response to lowering of the tongue which was required for oral respiration.

Over time many other experimental attempts to induce developmental change in the mammalian masticatory apparatus have been made. This is the obvious approach to testing mechanisms of malocclusion causation without the many difficulties of controlling variables in human epidemiological studies. "Mouth breathing" was implicated experimentally (Harvold et al., 1973) in the development of open bite and of abnormally narrow maxillary dental arch, attributed to altered muscle tension as the mandible is held slightly depressed during respiration. Similar differences were also found in soft-diet rats (Watt and Williams, 1951), probably due to a reduction in muscle use rather than increased, abnormally directed muscle tension. Yamada et al. (1997) verify that the rare

condition of openbite is the main outcome of nasorespiratory obstruction in monkeys, rather than there being a variety of resulting malocclusion syndromes including crowding.

Several workers have applied stress to the masticatory apparatus or have altered occlusal relationships using appliances fastened to the teeth of rats and rhesus monkeys (Hiniker and Ramsfjord, 1966; Charlier et al., 1969; McNamara, 1973). These appliances are designed to force protrusion of the mandible, resulting in some posterior bone deposition, bone resorption of the anterior edges of the glenoid fossa, lengthened condyle neck, and an increase in the angle between the ramus and the occlusal plane.

Using mature animals and a splint on the anterior teeth to force mandibular protrusion, Hiniker and Ramsfjord (1966) also found dentitional changes as teeth were forced to move to maintain occlusal relationships. This movement resulted in short-term periodontal traumas which abated when the teeth adapted and once again occluded adequately.

These studies demonstrate that forced realignments of occlusal relationships and mandibular movements lead to responses by the most flexible elements of the masticatory apparatus. In young animals, the growing skeletal system adjusts; in older animals, the dentition migrates to accommodate. While these experiments are important in assessing the response of the masticatory system to unusual mechanical stress, they do not create conditions likely to be encountered by the human masticatory system.

Experiments concerning the contribution of nutrition to normal occlusal development have taken the form of calorie deprivation of pigs. Tonge and McCance (1965, 1973), and McCance et al. (1968) used weanling animals, maintaining them for one year on a severe calorie-deficient diet, before attempting to "rehabilitate" some with proper foods. The authors found that their regime resulted in delayed dental development and eruption and "greater delay in the development and growth of the jaws." The consequences were tooth crowding and abnormal tooth-tooth contacts. Rehabilitation was only partially successful.

While calorie deprivation is undoubtedly a factor to consider in severely undernourished people, there is no evidence to correlate this deficiency with occlusal problems found in so many urbanized peoples of the western world.

While so many workers have commented over the decades on the probable influence of the hardness or softness of the diet on the developing masticatory system, various studies have examined this idea experimentally. Watt and Williams, (1951), Barber et al. (1963), Moore (1965), Beecher and Corruccini (1981) and Henrikson et al. (1977), used weanling rats divided into populations eating either pelleted rat chow as hard diet or crushed water-softened chow as a soft diet. In all studies, the animals were maintained for approximately four months. When compared to the hard-diet rats, the soft-diet animals: (1) were slightly smaller in body mass; (2) exhibited no molar wear; (3) had mandibles that were smaller, with condyles smaller and radiographically less dense; (4) had less width of the maxillary arch; (5) had smaller masseter and temporalis muscles; and (6) had skulls consistently smaller in mass and in linear dimensions, although with no significant differences in shape. Riesenfeld (1970) found rat facial reduction to approximate that resulting from removal of masticatory muscles when the diet was entirely liquefied. Much recent confirmation concerning masticatory hypofunction in rats is recently provided by Ulgen et al. (1997).

Dickson (1970) derided Watt and Williams for inferring dramatic arch change with softened diet. On account of Begg's (1954) writings, Dickson felt the 1 percent maxillary arch width reduction seen by Watt and Williams was inconsequential compared to the 8 percent arch perimeter reduction supposedly demonstrated by Begg to result from attrition. Therefore the attribution of great significance to dietary consistency in the development of modern levels of malocclusion was, to Dickson, "fallacious."

All mandibular structures related to chewing were affected by diet hardness. Molar wear was less with the soft diet. Attachment areas for the jaw-closing muscles, where muscle force is transmitted to the mandible, were smaller. The condyles, which resist bite (especially incisal) reaction force, were smaller.

Even buccolingual thickness of the mandible, which is essential to resist transverse bending, was less in the soft-diet animals.

More recently, Beecher and Corruccini (1981a) examined a small population of rhesus macaques which had spent a short period during adolescence on contrasting hard/soft diets. They found significant narrowing of the maxillary arch with no lessening in length in the soft-diet monkeys. This correlated with the histological findings by Bouvier and Hylander (1981) that significantly fewer secondary Haversian systems were present in the mandibular corpus of soft-diet monkeys than in the hard-diet monkeys of the same population.

While a number of measurements have been taken from animals in dietary consistency experiments, there has been no attempt to integrate these in such a way that interactions between different parts of the growing masticatory systems could be measured. Further, with only two groups, hard and soft diet, it could not be determined whether (1) the masticatory system responded in direct proportion to differences in dietary consistency, or (2) whether no differences should be expected until a threshold in dietary consistency difference was reached, resulting in a quantum change in the morphology of the masticatory system.

The Experimental Study on Rats

It was to fill these gaps in the experiment that our (Corruccini and Beecher, 1981b) study was carried out.

The experimental animals were Sprague-Dawley rats acquired at 21 days of age, evenly divided between males and females. Ninety animals were divided into three groups: Group I was fed pelleted rat chow (Purina Formulab); Group II was fed a gruel-like porridge consisting of ground chow moistened with water; Group III was fed the soft diet six days, with dry pellets provided every seventh day only.

We found the lab chow to be crumbly, easily breaking into small granules. This indicates that the "hard" diet was only minimally harder than the

gruel, engendering only subtle differences in bite force. We have no way of knowing the consistency of rat diets used in earlier experiments of this type.

The three populations were maintained on their respective diets for four months, the same length of time used in previous experiments of this type. After sacrifice, the heads were randomly numbered so that the measurer would not be aware of their group membership. Measurements were taken of body mass., fresh mass of the entire masseter, maxillary arch length (incisor to distal edge of last molar), maxillary arch breadth across buccal points of MI, mandibular length (incisor to MI), and anteroposterior length of condylar articular surface.

Maxillary breadth was affected most.

Metric Contrasts between Rats Raised on Hard and Softened Diets:

Significance	Trait	Soft-Diet	Hard-Diet
Not sig.	Maxillary L	24 mm	24 mm
Marginally sig.	Maxillary B	9.4 mm	9.6 mm
Not sig.	Mandibular L	8.9 mm	8.9 mm
Not sig.	Condyle L	3.3 mm	3.4 mm
Not sig.	Masseter Wt	1.42 gm	1.56 gm

The basic descriptive statistics showed the hard-diet population to be larger in all dimensions and disproportionately so in some. In particular, the maxillary breadth was markedly increased in the hard-diet reared animals; this measurement alone showed a differentiation that was too large (relative to within-sample variation) to be reasonably ascribed to chance. The population reared on intermittently hard diet was generally intermediate in the size of structures, although more similar to the soft group.

A more obvious feature of differences was manifested in the correlation structure of measurements within samples. The pairwise correlations were higher in the animals that had more masticatory resistance, and the differences were often significant. The strongest difference was in the growth relation of the masseter muscle, which was more integrated with growth in the condyle and upper arch breadth in hard-diet than in soft-diet animals.

There was increased multivariate variability in soft-diet animals, all the more striking in view of the fact that they were less variable than the hard-food animals in every univariate trait. In all respects, the intermittent-hard-diet group was intermediate, but more similar to the hard-diet sample.

Many have documented the better occlusion that existed in human populations prior to the onset of the industrial revolution. Only in the 19th and 20th centuries (it appears) has food become so processed that for practical purposes most chewing stress has been removed and little bite force is now called into play in the jaws of the growing child. Though the chewing explanation is most predictive of observed craniofacial changes in the rats, it remains unclear whether many orthodontists see this as a prominent explanation of the rising rate of occlusal variation in humans.

The growth independence of mandible and maxilla in humans has been frequently noted (Moorrees and Reed, 1954; Lavelle, 1972). In particular, the mandible may depend on muscular function to grow to average antero-posterior length, as shown by the tendency of some individuals with anodontia to have average-size mandibles (Thoma, 1938). Maxillary medio-lateral growth may be under closer genetic programming, since cases of anodontia show restricted growth while cases with muscular paralysis may develop normally. Garn (1961) lists breakdown of synchronous growth in lower and upper jaws as a possible cause of malocclusion, but does not give the source of the growth-correlation breakdown. Potter et al. (1976) demonstrate that upper and lower dental arches are under very different sorts of genetic control, with the maxillary teeth being more "conservative" and controlled by a fewer number of genes.

Lower correlation between upper and lower teeth in maloccluded individuals (as compared with normal) has been noted, and highlights the importance of high correlation between tooth and arch size to the development of good occlusion. Thus, considerable evidence ties upper-lower correlation diminution with development of malocclusion in humans. The prevailing explanation for such lowering of growth correspondence has been the effect of racial admixture between populations. Thus, the inheritance of different-sized parts that must occlude is attributed to the independent segregation of disharmonious genes from disparate ancestors, an old and unmeritorious idea dealt with earlier in this book..

We therefore concluded that our results suggest future directions for fruitful research based on the following observations. The amount of maxillary arch narrowing and collapse can be predicted by the amount of time the animals were chewing on hard food. Mediolateral maxillary growth seems dependent upon the muscular stimulation provided by rough elements in the diet. In this animal population the dissociation in size of occlusal features during growth, previously noted in certain groups of maloccluded humans and attributed to racial admixture, had apparently resulted from lower chewing stress required by a soft dietary consistency.

Ciochon, Nisbett and Corruccini (1997) followed up this approach in a carefully controlled growth experiment using an animal much more relevant to human occlusal analogies, the Yucatan Minipig. The Yucatan Minipig displays human-style blunt-cusped postcanine teeth, fused mandibular symphysis, relatively thick enamel, etc.

The following variables were collected: beginning body mass, final body mass, fresh superficial masseter weight, deep masseter weight, fresh temporalis weight, mandibular arch length (Interdentale to distobuccal M1), maxillary arch breadth at DP2 (i.e., DM2), bicondylar breadth, bizygomatic breadth, craniobasal length (Basion-Nasion), ramus height, bigonial breadth, occipital height, temporalis crest width, DP2-3 chord, mandibular thickness, incisor breadth chord,

mandibular overjet at M1, and ramus thickness.

In soft diet minipigs disproportionately smaller masseter and temporalis muscle mass, narrower jaws and face, and more crowding and maleruption of teeth occurred. A multivariate analysis showed the overall pattern of differentiation to be highly significant.

Squirrel Monkey Experiments

Replication of these experiments with a laboratory animal more closely related to man in terms of both biology and masticatory system was seen as a vital future need. The rat masticatory apparatus is quite specialized when compared to most mammals, especially humans: 1) Rats chew bilaterally with an up-and-forward power stroke (Hiiemae and Ardran, 1968), whereas most other mammals, including monkeys and humans, chew unilaterally with an upward, medial, and slightly forward power stroke (Ryder, 1878; Ahlgren, 1966; Kay and Hiiemae, 1974). 2) the mandibular symphysis of the rat is unfused and highly mobile during mastication, unlike the fused symphysis of humans and other anthropoid primates (Weijs and Dantuma, 1975). 3) The rat dentition is continually growing, with large chisel-like incisors, a large postincisor diastema, and molars featuring a series of buccolingually oriented crests. Humans and many other anthropoids have spatulate incisors and rather generalized postcanine dentition featuring cusps, crests, and basins which function as complex shearing and grinding surfaces (Kay, 1975). The unfused mandibular symphysis in the rat alters force distributions, and continual incisor function in rodents maintains a base level of function (whether in the presence or absence of forceful mastication) that has no parallel in man.

Among primate the squirrel monkey (*Saimiri sciureus*) makes a fine human analog for masticatory studies. Squirrel monkeys do have larger canines than do humans, and a small postcanine diastema, but chewing motions are basically similar (Kay and Hiiemae, 1974) and the postcanine dentition resembles humans about as closely as that of any other monkey (Swindler, 1976). Beecher

and I determined to investigate the consequences in occlusal and craniofacial development of a soft diet in growing squirrel monkeys. The opportunity came from observing the Primate Newsletter, and discovering a chance to "tissue-share" with other investigators having monkeys that were raised on experimental diets. However, we also included data and observations from four monkeys continually monitored from weaning to maturity.

Most of the 43 squirrel monkeys used in this study were involved in an atherosclerosis study. These animals could be divided into two groups -- those wild caught and those captive born at Litton. Until capture the diet of wild *Saimiri* included insect and fruit species with chitin, cellulose, and rinds providing masticatory resistance (Kay, 1975; Fleagle et al., 1981). After capture and until the start of the experimental diet (January 2, 1978), the diet was monkey chow biscuits, a food requiring masticatory force to break off a bite-size piece. From dental attrition, it was estimated that the captured animals were between 6 and 9 years old in January 1978. The animals were divided into two groups: The control group ate pelletized food which was water-softened because it was too hard when dry; the experimental animals had a similar diet, but softened also by the addition of high cholesterol additives. Thus, all animals ate soft food during the experiment. A previous study demonstrated that a high-fat, high-cholesterol diet does not affect bone growth and development (Bouvier and Hylander, 1981).

Offspring of these wild-caught squirrel monkeys were weaned onto their mother's diet, which was soft whether control or experimental. The animals could then be grouped into those whose dentitions developed on very soft foods (captive-born). Dental impressions, using Alginate and Denture Elasticon in custom trays, were taken from all animals in 1981. The impressions were then sent to Beecher who cast them in stone and kept all records of identity, and provided casts to Corruccini for measurement and scoring.

A second sample of four *Saimiri* was included, obtained at weaning (c. 6 months old) from Bowman Gray Medical School and maintained at Wright State

University. Two animals were fed Purina Monkey Chow biscuits, the other two ate water-softened biscuits. Dental impressions and full-head radiographs were taken monthly for the first 6 months, and bimonthly thereafter until dental maturity at 2 years of age.

The cases were scored and sorted double-blind as to occlusal variations from anatomically defined norms (Mills, 1955; Kelley and Harvey, 1977).

I additionally examined a sample of 70 wild-shot specimens (35 male, 35 female) from Panama and Colombia housed in the Smithsonian Institution's Division of Mammals. These were all normal, and not excessively aged. One striking feature of the occlusion of wild squirrel monkeys is the consistent presence of natural overjet and a small amount of overbite, superficially similar to the normal condition in the modern American population. This is very rare among nonhuman primates, almost all of which in the natural state display edge-to-edge incisor occlusion with no overjet and no overbite. The median value for central incisor overjet (amount upper incisor projects from the lower) was 0.6 mm, and 90% of the specimens fell within the range of 0.3 to 1.0 mm. A median overbite figure was obtained of 0.2; that is, the upper central incisor's incisal edge overlapped the lower central incisor by two-tenths of the crown height of the lower incisor.

The average tooth displacement score was 0.67 (i.e., the average number of noticeably displaced or rotated teeth plus two times the number of badly displaced teeth, came out to less than one tooth per animal). The average tooth displacement score in contemporary American youths, by contrast, is 5.1.

Among the categories of occlusal variables, the following anomalies were noted. One animal had buccal crossbite of a cusp-to-cusp extent on one side. There was one case of mandibular overjet of 1.2 mm. There were no observable buccal segment disharmonies nor any openbite or extensive overbite. No caries were seen. Results of this study set the standards for ideal occlusion used in analyzing the lab squirrel monkeys.

Characteristic arch patterns were occasionally analogous to common

human malocclusions -- mesially narrow and disproportionately long maxillary arches leading to incisor overjet, impacted malerupted premolars and second molars, malaligned premolar rows, crowed and rotated incisors. The tooth displacement count of about one was significantly greater in soft diet animals, but still not remotely approaching the urbanized human average of 5.1 (Draker, 1960; Kelley and Harvey, 1977).

Orofacial Contrasts between Squirrel Monkeys Raised on Hard and on Soft Diets:

Significance	Trait	Soft-Diet	Hard-Diet
Highly sig.	Total R/D	1.11	0.34
Highly sig.	Malocclusion (%)	78	14
Highly sig.	Maxillary Br (mm)	15.5	17.1
Highly sig.	Max Br/L	1.005	1.056
Highly sig.	Mandibular Br (mm)	13.6	14.4
Highly sig.	Palate Ht (mm)	3.44	3.99

Consistent metrical differences existed between the two experimental samples. Mediolateral arch breadths (maxillary across the third premolars and first molars, mandibular between second molars) were significantly larger in hard diet animals. Maxillary and mandibular arch lengths (from medial interincisal point to behind M$_2$), on the other hand were relatively unaffected by differences in dietary consistency. This finding is consistent with the similar experiments on rats.

Correlations were significantly lower in the soft diet animals, averaging +0.48 compared with +0.68 in hard diet monkeys (an overall difference significant at P=0.0001). The most significant differences were in maxillary breadths

regressed against palate heights, correlations that are positive in hard and negative in soft diet groups. Principal components analysis of metrical attributes confirmed the significant pattern of differences by showing a higher overall covariance in hard diet animals (eigenvalue 1 was larger compared to soft diet) and less dispersion away from the common growth vector (eigenvalue 2 was smaller in hard diet animals).

In the early months of the experiment, the maxillary arch breadths fluctuated wildly in the soft diet animals when compared with those on hard diet. Subsequently the maxillary arch growth in soft diet animals became more regular, but this dimension continued to distinguish the soft from the hard diet pair more surely than any other trait from weaning to maturity. Radiographs of cranial suture development, especially the lambdoid and sagittal sutures, showed distinct differences in calcification. The radiolucent area at the sutures was much broader in the soft than in the hard diet animals.

An anecdote may illustrate a final distinction arising from the soft diet. After the animals had been on the experimental diets for two months, we attempted to supplement the diets of the four monkeys with orange slices. We found, however, that the soft diet monkeys would not chew or suck the juice out of the slices, but rather the juice had to be squeezed out onto their food. The pair eating standard monkey chow biscuits readily ate the orange slices. This distinction suggests that some sensitivity to chewing pressure developed in the soft diet animals.

The tooth displacements, arch size/shape changes, poor craniofacial growth correlations, and retarded sutural closures comprised a suite of features we attributed to the large reduction in bite force throughout the growth period of soft diet animals. A variety of macroscopic occlusal anomalies also occurred, especially premolar maleruption and incisor displacement. It is apparent that the developing craniofacial region, especially the dentition, needs a certain amount of mechanical stress to attain its proper configuration. When rats were raised on soft diet with pelleted (hard) food offered once weekly, the resulting craniofacial

morphology was more similar to that of rats always fed only water-softened food (Beecher and Corruccini, 1981). We suspected the same findings would result from such a study conducted with primates as resulted from the rats, namely that there is a minimum amount of chewing stress necessary for proper craniofacial development. Stresses greater than the minimum will not significantly alter development, but stresses below it will result in the array of characteristics found in our soft diet squirrel monkeys.

Interproximal attrition and loss of arch space were negligible in our animals; periodontal disease, carious destruction, and tooth loss were present in both samples and actually greater in the group raised on hard diets. Therefore, these two frequently mentioned factors do not explain the observed differences. We conclude that chewing stress is an epidemiological factor with possible preventive implications for occlusal disorders.

Previous primate experimentation has also resulted in some occlusions resembling cases found in humans. Forced mouth-breathing by blocking nasal passages in macaques has resulted in the development of open bite and a narrower maxillary arch (Harvold et al., 1973). The limited interference with the masticatory apparatus suggests that the results might also be applicable to humans, yet we would argue that this model does not fit the circumstances of many cases of malocclusion, and cannot account for the rapid deterioration of occlusion in societies adapting to soft food.

The poor coordination of craniofacial growth (as measured by correlation) is known in maloccluded humans (Moorrees and Reed, 1954; Garn, 1961; Lavelle, 1972). At one time (Horowitz and Osborne, 1971) orthodontists ascribed this to a racial admixture causing mismatches -- e.g., large upper jaw from one parent and small lower jaw from the other. This is the theory that has now been discredited (Chung et al., 1971; Kaul and Corruccini, 1984).

Other hypotheses for developmental malocclusions invoke factors or utilize invasive experimental procedures not generally applicable as explanations for the pervasiveness of malocclusions in the West, or for the rapid increase in

malocclusions when people switch to a processed-food diet. Maxillary arch narrowing and increasing palatal height, the most significant and predictable results in the soft diet experimental animals, are like the "maxillary collapse syndrome" which is the most common occlusal problem in American youths (Kelley and Harvey, 1977). The early fluctuation in maxillary arch breadth among the two Wright State soft diet monkeys demonstrated that the buccolingual orientation of the teeth is very susceptible to disruption. The simultaneously occurring high palate in soft diet animals suggested that maxillary collapse is probably the result of growth differences in the mediopalatine suture as well as the failure of the maxillary alveolar process to align the teeth properly with their mandibular counterparts. We suggest that the bones of the palatal arch are, outside of the alveoli, most affected in their growth by a reduction in chewing stress.

This parallel in maxillary changes between humans and a relevant experimental animal strongly indicates that there is a close relationship between dietary consistency and developmental occlusion in people. Further strengthening the link between these experimental results and an etiology of human malocclusion is the predictive power of this paradigm in accounting for the transition from predominantly good to frequently bad occlusion within one generation when a human society acquires soft, processed "modern" foods (see earlier Chapters). The present study was the first to invoke a natural, noninvasive mechanism in primates that altered occlusal relations and could be relevant to a large proportion of humans. The other primate experimentation involved radical methods such as cutting muscles, blocking nasal passages, or inserting oral implants, disruptions not generalizable to the average person.

Noting that human samples with maloccluded dentitions frequently exhibit increased variation in individual occlusofacial structures and decreased morphological integration or correlation among sets thereof (Moorrees and Reed, 1954; Garn, 1961; Harris, 1971; Lavelle, 1972; Zingeser, 1973; Moore, 1981; Anderson and Popovich, 1983), we became interested in further investigations.

Possibly Bjork (1950) gave the earliest mention of lesser coordination of craniofacial growth in maloccluded individuals. He attributed this to racial admixture, an idea that is still common (e.g., Lavelle, 1972).

Zingeser (1973) demonstrated significantly greater variation from paired occlusal loci to nasion in retrognathic and dentally crowded individuals, compared to occlusal normals. He also showed upper anterior dentofacial height much more variable in humans than two nonhuman primate species. Moore (1981) calculated pairwise correlations among seven maxillary, mandibular, and ethmoidal dimensions and angles. A sample of humans averaged a coefficient of determination (shared variance) of 11.6% compared to 25.3% for samples of three ape species. He stated "a loosening of the co-ordination of growth between the different regions and subregions of the human facial skeleton as compared to that of the great apes .... is probably of considerable clinical significance, in that it may underlie the very high prevalence of disorders of facial growth in modern man," but this characteristic's "morphogenetic basis and evolutionary significance are not clear." More recently, Anderson and Popovich (1983) demonstrate that certain cranial dimensions are less intercorrelated or differently correlated in Class II maloccluded individuals.

In our samples of rats, Beecher and Corruccini (1981) showed average coefficients of determination over six measurements of 63.3% for those raised on hard diet and 41.1% for those raised on the artificially softened, easily masticated pellets. Over seven occlusofacial measurements on squirrel monkeys, Beecher and Corruccini (1983) showed average coefficient of determination of 45.5% for hard diet and 30.6% for soft-diet samples. All these differences were quite statistically significant. Significant also are the implications for humans, since in the developed world, where high malocclusion prevalence is found, diet is much softer than in traditional societies.

Dental crowding is rare in primates, being mentioned only in regards to abnormalities of supernumerary teeth (Schultz, 1972). Only about 2% of wild platyrrhine monkeys have impacted teeth (third molars only) and only about 1%

show irregular tooth placements (Smith et al., 1977), and even those low frequencies were possibly due to trauma rather than abnormal growth. Dickson (1970), citing Colyer's (1931, 1936) earlier surveys, showed that substantial frequencies of "malocclusion" in nonhuman primates could only be documented by counting any deviation from perfection, no matter how slight. The same diagnostic criteria yielded very nearly 100% prevalence of malocclusion in humans, merely demonstrating the great rarity of utterly ideal perfection in tooth alignment. Dickson and Colyer demonstrate that animals have deviations of much lesser severity than humans in these cases.

The Study on Baboons

The dietary consistency mechanism is therefore worthy of further experimental examination in close biological relatives of the human species. We (Corruccini and Beecher, 1984) reported contrasts in occlusofacial variability and correlation between baboon groups raised on diets of varying consistency. This extended our earlier work with a new species of longer maturational period and closer taxonomic relationship to man, and new experimental conditions -- namely, late introduction of soft food.

Dentally adult male baboon (*Papio cynocephalus*) heads were received and macerated. Some 24 animals had been raised over the last 108 weeks of their dental developmental period (generally beginning about 1 year prior to eruption of the last molars and canines) on a very soft, atherogenic diet consisting of cholesterol, lard, butter, egg yolks, and powdered chow. These will be referred to as the soft-diet or experimental baboons. The other 16 animals, selected to be age-matched, were wild caught within 4 years of sacrifice (as were most experimentals) but had always eaten naturally tough food or pellet chow; these were the hard-diet controls.

We took four linear measurements on each skull designed to directly indicate occlusal relations, and 16 more measurements yielded 12 pairwise combinations reflecting important oral-facial proportions or bilateral asymmetries.

The null hypothesis was that soft-diet monkeys will not show more variable or more poorly developed structures.

Contrasts between Baboons Raised on Hard and on Soft Diets:

Significance	Trait	Soft-Diet	Hard-Diet
Highly sig.	Overjet (mm)	0.24	1.37
Highly sig.	Malocclusion (%)	25	0
Marginally sig.	Incisor Displ. (mm)	0.27	0.13
Highly sig.	Dent.Bas.Asym.(mm)	1.07	0.51
Highly sig.	Palate L Asym.(mm)	1.40	0.64
Highly sig.	Sphenoid Asym.(mm)	0.99	0.72
Highly sig.	Ramus Asym.(mm)	1.45	0.57
Highly sig.	Up.Facial Asym.(mm)	0.20	0.04
Marginally sig.	Na-Ba/Ba-Ho ratio	0.19	0.13
Highly sig.	Ramus Br. ratio	0.76	0.81

Important in its reflection on conditions analogous to human malocclusion was the much greater variability in soft-diet experimentals, reflecting a wider range of morphologies and consequent greater probability of exceeding limits of a defined clinical norm. For instance, 10 of the 24 experimentals showed mandibular overjet, but all controls had maxillary overjet. Upper central incisor displacement (unevenness of right and left labial surfaces in the a-p plane) was absolutely greater in experimentals. Dento-basal asymmetry (maximum perpendicular difference from parallel alignment of occipital condylar and first premolar apical planes) was also significantly greater in experimentals. Palates were significantly higher in experimentals; however, experimentals were larger in

general (probably owing to the high kilocalorie experimental diet).

Palate length asymmetry (from alveolare to distobuccal M1) was much more variable in experimentals, signifying more departure from symmetry in both directions. Sphenoid length (hormion to distal maxillary tuberosity), mandibular ramus breadth, and upper facial height (orbitale to zygomaxillare) asymmetries all varied significantly more in experimentals. The left-right mean also differed for sphenoid, mandibular condylar, and ramal measurements signifying systematic side differences (perhaps reflecting directional functional asymmetry).

Anterior palate breadth (across second premolars) relative to palate length (subtracted) did not differ in mean but was significantly more variable in experimentals. Posterior relative palate breadth (at M3) did not differ. Upper facial breadth ratio (bizygomatic over external biorbital breadth) was larger in controls, possibly reflecting masticatory function of the temporalis, but the significance of this was marginal. The basal ratio, craniobasal to basioccipital lengths, was larger and more variable in experimentals. Mandibular ramus breadth (reflecting gonial development) relative to palate length was smaller and more variable in experimentals. Upper facial height relative to palate posterior breadth varied significantly more in experimentals.

The pairwise relation and variability of measurements was alternatively, and perhaps more satisfactorily, expressed by product-moment correlations. Some 90.5% of the values were higher in controls, obviously statistically significant. Moreover, 31 correlations attained a statistically significant difference level (two-tailed), and every one was higher in controls.

Arches of experimental individuals were examined in centric occlusion for gross occlusal anomalies after examining the controls plus 16 additional wild-caught baboons. There were six cases of abnormal occluded experimentals that prominently exceeded the range of variations seen in either the 32 controls (Fisher exact P=0.004) or in an additional sample of 300 wild primate dentitions of various species I examined. I observed a range of occlusal discrepancies: posterior crossbite, class III distoclusion, incisor openbite, extreme maxillary overjet,

impacted third molar. displaced incisors, and crowding of postcanine teeth (especially with the anterior premolar buccally displaced and posterior premolar lingually displaced).

The hypothesis of association of highly correlated occlusofacial dimensions with proper occlusal relations, in the presence of high-demand mastication, was thus experimentally verified. Moore (1981) states, "of the greatest clinical significance, is the generally looser co-ordination of human facial growth, as compared with the great apes. There can be little doubt that this is one of the factors underlying the very high prevalence of facial dysharmony in man." The occlusofacial morphological integration is sharply lowered in primates raised on soft foods that provide little masticatory stimulus. Insofar as that diet mimics the processed foods of the Western industrialized world, a basis is provided for further investigation of the role of resistant dietary items in coordinating occlusofacial growth and reducing occlusal variation and disharmony.

From all these results, I and coworkers have stressed that we need a study to test the relative extent to which various environmental factors (acting simultaneously) are significant in craniofacial development and dental occlusion. The experimental animal could be the squirrel monkey, an excellent model for the human masticatory apparatus, or the more economically feasible mini-pig, and dietary consistency could be tested in a multi-way context with nasorespiratory obstruction, etc.

7. Genetics and Twins.

The genetic content of craniofacial traits is fundamental to clinical thinking and to anthropological, interpopulation comparisons. Unlike most pathological medico-dental problems, malocclusions usually result from variations in normal development for which there is little specifiable cause (Proffit, 1986). This is one reason why "occlusal variation" seems more appropriate than the term "malocclusion", emphasizing the continuum of occlusal relationships with more severe cases being located at the extremes of the distribution (Smith and Bailit, 1977). It is generally accepted that these occlusal variations, excepting certain specific syndromes, result from a combination of genetic and environmental influences during development. The murkiness about the relative importance of these factors remains. The genetic basis of occlusal variation has been discussed for roughly the past hundred years, with studies adopting varied methodologies (for reviews see Weinberger, 1926; Brash, McKeag and Scott, 1956; Krogman, 1967, 1974; Jago, 1974; Isaacson et al., 1975; Smith and Bailit, 1977). The purposes have been to study mode of inheritance, admixture and inbreeding effects, linkage, population variations and heritability. Early traditional twin studies (Lundstrom, 1948) and intra-familial comparisons (Stein et al., 1956) indicated that occlusal traits were under considerable genetic control.

Genetic determinism is an ancient influence over thinking about etiologies of craniofacial irregularities. Therefore, the heredity-environment dispute has been important in orthodontic thinking for more than a century. To take a look again at Sim Wallace's (1904) writing: "The question arose, however, that if acquired characters were not transmissible, then what became of the current

assumption that the jaws were growing smaller on account of successive generations of disuse?... Such a condition often appears to be inherited, as parents, as a rule, bring up their children in much the same style as they were brought up themselves.... though heredity cannot be called the cause of the irregularity, yet it may determine the type which may be brought about provided mal-environment necessitates some irregularity."

Similarly, Klatsky and Fisher (1953) became sharp-tongued when discussing the clinical overimportance ascribed to genes:

> "The concept that most or all dental diseases and deformities are due to the natural process of evolution is a great impediment to the dental science. It tends to encourage the fatalistic view of: 'What's the use? You cannot fight against the immutable laws of Nature!' Such ideas serve as a deterrent to greater efforts toward a broader program of dental prevention. Hence present day preventive measures are confined mainly to old stereotyped operative and prophylactic procedures, and no attempt is ever made to restore use and proper function to the masticatory apparatus by changing our civilized methods of preparing food or other effective means .... But the most important question for us to consider is not so much the utility of this theory but its soundness. Is it true that our dental organs are doomed to utter destruction? Are we faced by the possibility that humanity of the future will consist of a toothless species? Does the science of evolution warrant such gloomy conclusions that nothing radical can be achieved in combating dental disease and disorder, putting a halt to the degenerating trend of our dental organs?"

Klatsky and Fisher pungently quote Gregory (1929):

> "What of the human face a million years from now: a short period compared with its entire history. If the present tendencies continue unchanged the white

people of those days will for the most part have lost all four of their wisdom teeth so that their total number of teeth will be twenty-eight. This will tend to make their jaws somewhat smaller. If they no longer eat meat and vegetables but take prepared extracts as food, their jaw muscles and jaws may be further weakened."

Thus one frequently encounters the Lamarckian scenario of humans in the year 3000, globe-headed, with vestigial jaws and a tiny hole of a mouth suitable only to the insertion of high-potency pills.

Since the genetic basis for occlusal variations and malocclusion has naturally been such a major consideration for orthodontists and craniofacial biologists, this is a most fundamental point. The different studies directed toward heritability of occlusion have varied widely in methodology. Lundstrom's (1948) classic study used the difference between quantified occlusal variables in monozygotic (MZ) and dizygotic (DZ) twins to measure genetic and nongenetic variance ratios. Overjet was highly heritable with a 3/1 ratio of genetic to nongenetic variance (75 percent heritability). Less heritable were arch-width differences at PI and MI, buccal segment relation (sagittal overjet), and overbite. Generally, heredity and environment appeared about equally important, but heredity has long been called the major etiologic factor in the severe malocclusions. Earlier twin studies by Detlefson (1928), Bachrach and Young (1927, 1928), and Macklin and Moore (1935) also showed that MZ similarity exceeded that shown by DZ twins in arch size and shape and occlusion. More recent contributions and reviews are Boraas et al. (1988) and Lauweryns et al. (1993).

Earlier and later studies than Lundstrom's have used analysis of similarity in other kinds of relatives to draw similar conclusions. Iwagaki (1938), Rubbrecht (1939), Moore and Hughes (1939), Stein et al. (1956), Litton et al. (1970), and Chung and Niswander (1975) compared prevalence among siblings or among offspring and their parents. All found that the chance of malocclusion

occurring in relatives of the maloccluded was higher than at random in the general population. As proponents of the twin method have pointed out, this proves little since family members also share greater environmental similarities than the general population.

Another possible weakness in occlusal heritability studies, shared with recent epidemiologic studies of occlusion, is the inclusion of individuals currently undergoing or who have undergone orthodontic treatment. Obviously, this will change the phenotype of the trait from what it was genetically (or environmentally) programmed to be. Aside from such a distortion of the data, the use of orthodontically treated twins will bias genetic analysis. To give just two theoretical possibilities, (1) the presence of dually maloccluded twins may affect the probability of parents seeking treatment and (2) families will often seek the services of the same practitioner, whose characteristic, preferred, or idiosyncratic methods may produce similar results which are not necessarily the result of similar genes.

A polygenic mode of inheritance is generally accepted for the range of occlusal variations (Nelson, 1969; Litton et al., 1970; Harris, Kowalski and Walker, 1975a, b). The classic work of Lundstrom (1948, 1955b, 1960) on twins bolstered the impression of the stronger genetic than environmental component for occlusal variations. The commonality of these studies has led the orthodontic specialty in general to conclude the genes are fundamental in causing malocclusion. This impression is strengthened by the frequent observation among practicing orthodontists of special occlusal similarities running in families they see. However, advances in methodology have provided new insight into the role of genetics versus environment in determining occlusal traits.

Twin Studies

More recent reports on twins (e.g. Corruccini and Potter, 1980; Potter et al., 1981; Corruccini, Townsend, Brown and Richards, 1990) and first-degree relatives (Harris and Smith, 1980) have emphasized the role of environmental

factors. Anthropological studies, demonstrating rapid increases in occlusal variation in non-industrialized populations following the adoption of western-style food habits, also add support to the notion that non-genetic factors, possibly related to masticatory function, are important contributors to variation within the arches (e.g. Corruccini, 1984, 1991). Historically, as Proffit (1986b) notes, prevailing views about the causes of malocclusions have affected the type of treatment offered to patients.

An understanding of the relative levels of genetic and environmental determination of occlusal traits impacts clinical orthodontic prevention and treatment planning, evolutionary theory (Calcagno and Gibson, 1988) and the directions for future genetic research. Advances in quantitative genetics methodology will some day redirect the research of occlusion of traits towards identifying the mechanism of gene action as well as random environmental effects. It is therefore timely to reexamine the results of earlier twin studies and to place the relative contributions of genetic and environmental factors to occlusal variation in proper perspective.

It is frequently speculated that genetic factors, such as racial out-crossing, inbreeding, and accumulation of mutations, explain the epidemiological transition in malocclusion that was discussed at length earlier. This outlook is unquestionably conditioned by the belief in genetic determination of malocclusion. Determination of the actual level of genetic determination of occlusal variation would result in a significant effect on treatment, if prevention (in the ordinary clinical sense) is not possible. Only genetic counseling and early intervention with appliances are possible alternatives to the usual approach of extraction and straightening, and the former are not always successful. Therefore, it is important to apply the most current developments in twin analytical methods to the question of heritability of occlusal variation.

In view of these points, I have undertaken several new analyses of heritability of occlusal characteristics with the inspiration of a succession of collaborators. These differ from the previous studies in (a) applying recent

proposed methods for statistical analysis of twins and (b) carefully selecting the twin sample to avoid biases resulting from manipulation of the phenotype (extracted teeth, straightening).

While the twin method is a powerful genetic analytical tool, it has usually been applied in Western white populations. Comparative twin analysis in non-Western populations offers obvious advantages. First, the universality of established Western heritability patterns can be examined. Second, genetic interpretations of quantified population differences become potentially falsifiable. For example, tooth sizes are frequently contrasted among human populations and assumed to reflect different allele frequencies, yet the evidence of genetic determination is almost entirely confined to a single gene pool of Euro-American Whites. The I.Q. controversy exemplifies the fallacy of applying heritability evidence from one population to the explanation of differences with another population. Third, recent refinements in twin analysis allow examination of hidden environmental influences in the classic genetic variance estimates. Applying these to twin samples from varying circumstances tests the actual environmental determination of "heritability" levels.

My ongoing involvement with twin analyses of occlusal variations owes directly to Dr. Rosario Potter, who, when I was visiting her lab to examine Pima collections in 1979, invited my use of twin casts under her curation. Ultimately our sample of U.S. white twins (Potter et al., 1981) consisted of 87 MZ and 77 DZ pairs, totaling 166 males and 162 females, modally 14 years of age at casting. These came from the Indiana University and State University of New York at Buffalo collections (more than half from the latter, under Dr. Larry Green's supervision).

Years later, as an offshoot of the epidemiological work described earlier in the Punjab, I and Dr. Krishan Sharma initiated the casting and occlusal study of twins identified by the Panjab University study. The Indian twins (Sharma and Corruccini, 1986) comprised 23 MZ and 35 DZ pairs, totaling 55 males and 61 females, of average age of 17.5 years.

On Indian twins no such previous report is available in the literature (indeed, very few non Western populations have been studied for any kind of comparative heritability). Our first paper therefore presented a detailed account of the new occlusal data from the northwest Indian (Punjab) twin sample as a unique expansion of the literature in this subject area. Years later I was able to add comparable data from the South Australian Twin Panel thanks to a sabbatical leave and the gracious cooperation of Grant Townsend and Tasman Brown of the University of Adelaide.

The present chapter discusses estimates of genetic variance for occlusal traits in Australian, South Asian, and U.S. twins, including tests of several basic assumptions underlying the twin model. A better understanding of the relative effects of genetic and environmental influences on different occlusal features, within and between different populations, should ultimately influence improved theory, treatment planning and preventive rationales in orthodontics. As part of an ongoing study of dentofacial structures in South Australian white twins, I scored my occlusal traits between the monozygous (MZ) and dizygous (DZ) twin pairs. There were two samplings from the South Australian twin panel: "restricted" and "unrestricted". Only individuals with complete permanent dentitions on at least one side (apart from third molars) were included in the restricted sample. This comprised 48 MZ and 34 DZ pairs. The twins, 84 males and 80 females, were aged between 13 and 26 years with an average age of 16.2 years. The unrestricted sample comprised these, plus twin pairs where one or both members showed evidence or a history of orthodontic treatment, or more than one missing tooth. This sample totaled 76 MZ and 60 DZ pairs.

It is not entirely clear how many of the previously reported twin analyses of occlusion have eliminated these potential biases. However, eliminating such affected pairs constitutes a different potential bias, in that twin pairs with at least one individual missing teeth or having had treatment, are a non-random exclusion. Much of the intrapair variance in the unrestricted sample results from DZ eruption and maturation differences -- complicating the

straightforward interpretation of purely occlusal variances. In Western societies with widespread orthodontic practices these biases are unavoidable. However, there was virtually no orthodontic treatment available to the Indian population, while the availability of longitudinal cast series in the U.S. sample allowed most occlusions to be scored pre-treatment (allowing an accurate phenotypic assessment).

Zygosities of all twins were determined by comparison of numerous genetic markers in the blood.

Occlusal traits were measured and scored directly from randomly-arranged dental models (co-twins scored separately), with opposing teeth in intercuspal position or centric occlusion.

The approach we have adopted for the genetic analysis is detailed in previous studies of Australian, U.S. and Indian twins (Townsend et al., 1988; Corruccini and Potter, 1980; Sharma and Corruccini, 1986). Several hidden assumptions implicit in the traditional twin model have been documented by Christian et al., (1974) and Kang et al., (1977). First, twin zygosities should not be associated with the mean of the trait under consideration (Christian and Norton, 1977; Christian, 1979). A modified t-test based on nested twin data has been recommended in which among-pair mean squares are used as the error term and the degrees of freedom are approximated (Christian et al., 1974; Christian, 1979). Significant differences in mean values between MZ and DZ twins would reflect inherent biological differences associated with the twinning process (Christian and Norton, 1977).

Second, the standard twin model of Haseman and Elston (1970) partitions total variance into within-DZ (WDZ) and within-MZ (WMZ) mean squares. Christian et al. (1974) consider that total variance within zygosities must be equal for the model to hold. If there is evidence of heterogeneity of total variance, then environmental factors are postulated to be unequal for MZ and DZ twins. The arithmetic mean of among- and within-mean squares (AMS and WMS) is then recommended to provide an unbiased estimate of twin genetic

variance (Christian et al., 1974). These environmental factors may result from competitive or convergent influences that differ for the two twin types (Kempthorne and Osborne, 1961). To test heterogeneity of total variances, one-way analysis of variance is performed, first treating twin pairs as groups of two to provide among-pair and within-pair mean squares for MZ and DZ categories, then an F' test (Christian et al., 1974) compares TMZ (AMZ+WMZ) and TDZ (ADZ+WDZ). The larger value is used as the numerator of a two-tailed F' test, and the 0.2 probability level is used to control for type 2 error as the test is relatively insensitive to common variance in the zygosities.

Manfredi et al. (1997) provide an example of recent twin analysis which does not consider total variance disparities between the zygosities but only provides the usual evidence for reduced intra-pair variance in monozygotes. Similarly reduced total variance in MZ twins would reduce or eliminate such evidence for significant heritability. Even so Manfredi and coworkers document many high cultural heritabilities and much fluctuation in estimated heritabilities for their orthodontic cephalometric traits.

As regards the third hidden assumption, genetic variance estimates will also be biased by inequality of environmental covariances of MZ and DZ twins (Christian, 1979). If environmental covariance is relatively greater for MZ than for DZ twins (CMZ>CDZ), heritability will be exaggerated. To test this, Christian et al., (1975) propose an F test contrasting the within-pair and among-pair mean squares of DZ twins (F = ADZ/WDZ). If this ratio fails to appreciably exceed a value of 1, then the evidence for genetic variance originates solely in the MZ twins, and it is unlikely that any substantial proportion of the total variance is genetic (Christian et al., 1975; Christian, 1979).

The classic genetic variance ratio (GVR) is calculated as F = WDZ/WMZ and tested for significance, provided the data pass the above tests. If the F' test yields a significant result, a modified among-component ratio, F_{ac}= (WDZ+AMZ)/(WMZ+ADZ), is used to provide an unbiased estimate of GVR. The rationale for this test is that AMZ/ADZ is likely to be just as valid and

sensitive as WDZ/WMZ when total variance is heterogeneous (Christian et al., 1974).

We presented basic statistics such as means, variances and intraclass correlation coefficients, r, for MZ and DZ twins. Three different estimates of heritability were calculated to quantify the proportion of total variance attributable to genetic influences. The within-pair variance heritability estimate h^2_{var} was computed as $h^2_{var}=4(WDZ-WMZ)/(TDZ+TMZ)$ following Kang et al. (1978). The Holzinger heritability coefficient $h^2_{holz}= (r_{MZ} - r_{DZ})/(1-r_{DZ})$ was also calculated as it has often been presented in previous twin analyses. In addition, the so-called path analysis model heritability estimate $h^2_{path} = 2(r_{MZ}-r_{DZ})$ used recently by Lundstrom (1984), was also determined. Each of these estimates can theoretically range from zero to 1 (or 0-100 percent) reflecting the proportion of observed phenotypic variation due to genetic factors. However, negative values or values greater than unity are mathematically possible raising interpretative problems. Estimates derived from twin studies are referred to as broad heritabilities since the genetic influences may include additive, dominance and epistatic effects. Lundstrom (1984) and Lundstrom and McWilliam (1987) have also calculated an estimate of cultural inheritance, according to the formula $c^2 = 2r_{DZ}-r_{MZ}$, to indicate the importance of familial similarity due to common familial environment rather than genetic effects.

When the DZ total variance exceeds that of MZ twins, the heritability estimate h^2 will be artificially high and genetic determination of the trait will seem greater than it actually is. In other words, if among-MZ variance is smaller at the same time that within-MZ variance is smaller, doubt is created about the genetic meaning of the latter.

American Twins

Some twenty of the forty traits on U.S. twins (Corruccini and Potter,

1980) had significant F ratios, and in nineteen of these the DZ's were more variable. This would be expected if environmental covariance were greater among MZ's (and there are many reasons to expect such a convergence). Thus, for these nineteen traits, the F ratio (measuring genetic variance and significance of heritability) was lowered when the proper comparison of variance (combined estimate) is used. This situation is not unknown, where MZ and DZ group means do not differ significantly but the total variances exhibit heterogeneity, as documented for tooth size and for many dermatoglyphic and biochemical traits in humans. Such pervasive levels of zygosity bias are seemingly unique to these occlusal traits, however.

Genetic variance and heritability estimates were reliable for the various arch size (length and breadth) measurements, as the underlying assumptions were all satisfied.

Nine out of 15 occlusion variables in total were found to have probabilities less than the chosen F' significance level of 0.20, which led us (Corruccini and Potter, 1980) to reject the within-pair estimate of genetic variance for these traits as unbiased. For the malalignment (R/D) traits, it was interesting that while anterior teeth malalignment showed greater environmental influence, posterior teeth malalignment showed greater genetic variance. For MZ-DZ correlation matrices and principal component loadings, we could not find any evidence for common genetic factors acting on the occlusion traits studied.

Our findings appeared to separate the sources of twin variance for occlusion traits. In contrast to those traits exhibiting greater environmental effects, univariate and multivariate measures of the overbite and spacing traits had significant estimates of genetic variance and less evidence for unequal environmental variance and covariance between zygosities.

Although the genetic source of twin variance seemed to be present in posterior teeth malalignment, a predominant environment source showed up in results for anterior teeth malalignment. Pending further evidence we advised that anterior and posterior teeth malalignment be treated as separate entities in any

further search for genetic mechanisms and for nonrandom environmental components of variation.

Our results were not dissimilar to those of Lundstrom's twin data (1948, 1955) and those of Chung and Niswander's sibling data (1975) in that the degree of genetic determination was low for buccal segment relationship. With respect to the overjet, crossbite and malalignment traits, our findings of a greater environmental than genetic source of variation were at variance with Lundstrom, who reported the highest heritability for overjet, and with Chung and Niswander, who reported genetic influence for all these traits. From our evidence it appeared that nonrandom environmental effects, familial in origin and associated with twin pair variance and possibly with sibling covariance, were not completely removed from the analysis in these earlier genetic studies. Heritability estimates averaged 0.33 before correction for environmental bias. After correction, the average estimate was reduced to 0.15. Thus greater environmental than genetic influences were demonstrated for most of the traits studied: overjet, crossbite, buccal segment relationship, malalignment, and TPI scores. For these traits, further explorations of the range of underlying nonrandom environmental causes of variance (prenatal and postnatal, familial and nonfamilial) may be more fruitful, we suggested, than any search for a specific genetic mechanism of transmission.

Indian Twins

Years later we (Sharma and Corruccini, 1986; Corruccini, Sharma and Potter, 1986) were able to expand on these results with the Indian sample. The F'-test revealed that MZ and DZ twins differed much more in variance than in means. Seven out of sixteen variables were found to have probabilities less than the chosen significance level, which resulted in rejecting the within-pair estimate of genetic variance as biased. In six instances, the total MZ variance was larger than the corresponding DZ variance. Only in spacing, DZ mean squares were significantly higher. These results, quite startlingly contradictory of the U.S. twin variances, were attributed to greater environmental variance in MZ twins.

The ADZ/WDZ ratio approached significance only for posterior displacement, crossbite extent and anterior displacement traits. An entirely different picture emerged for arch and palate measurements where the null hypothesis was soundly rejected for all traits. The twin model is unlikely to detect significant genetic variance without detecting a correlation within DZ twin pairs, so for most of the occlusal traits it was suspected that there existed a greater environmental covariance for MZ twins than DZ twins. These results confounded any inference drawn from the traditional twin studies for these traits.

In the Indian study the association between the type of twinning and mean value was significant only for one trait, but MZ twins tended to manifest larger means than their DZ counterparts. For example, TPI gave an indication, though not reaching a statistically significant level, that MZ twins have a higher malocclusion index than DZ twin pairs. MZ group means for bodily dimensions including head and face are generally less than those for DZ twins (Sharma, 1982), by contrast, accentuating the unusual twin variance patterns followed by occlusal variants.

If there is insufficient length and breadth of basal bone to support dentoalveolar structures, then crowding of the teeth will result. This may result in unfavorable tooth/tissue ratio. The finding of a higher malocclusion index in MZ twins attained greater significance in light of the known higher incidence of congenital malformations among MZ than DZ twins (Bulmer, 1970).

As contrasted to means, we found widespread variance heterogeneity among zygosities. This was more common in occlusal traits (e.g. overbite, rotation/displacement of teeth from ideal alignment, spacing and TPI scores) than in arch and palate dimensions. Another characteristic feature of the data was the MZ twin pairs having a higher total variance than DZ twin pairs. Following the model of Christian, Kang and Norton (1974), we reasoned that this is due to unequal environmental variance rather than greater MZ genetic variation or, in the words of Kempthorne and Osborne (1961), it is due to "competitive forces" that are different for the zygosities. Were a more extensive data base available,

including other classes of relatives, more complex analytical methods (especially path analysis) would have been appropriate for further specification of environmental and genetic influences.

Significant occlusal genetic estimates are invalidated by evidence of stronger environmental covariance among MZ twins by the ADZ/WDZ test. Only anterior tooth R/D (i.e. crowding of incisors and canines) maintained a robust expression of genetic variance; interestingly, this variable was the major influence on malocclusion indices and on cosmetic and qualitative judgments of orthodontic treatment need. Arch and palate dimensions, by contrast, showed significant genetic variance estimates.

In postnatal growth, soft tissues grow at a pace equal to that of the supporting skeleton if muscular balance is achieved by the lips, cheeks and tongue and not disturbed by abnormal or habitual behavior or pressure patterns (perhaps shared in families (Ballard, 1963).

According to conventional methods, both the American and Indian twin sets indicated consistently high, significant GVR for dental occlusal and arch traits, implying genetic determination of occlusion. Both samples yielded average WDZ/WMZ variance ratios of nearly 2.0 (although the Indian figures were significantly more variable for occlusion), suggesting about 50 percent simple heritability of the traits, and this is consonant with conventional notions encountered in the major orthodontic textbooks and also with Lundstrom's (1948) classic study.

We found much variance heterogeneity among zygosities in both samples, however, which necessitated revised among-component estimates. These tended to somewhat raise Indian GVR because Indian MZ twins were more variable, whereas a significant drop in average genetic variance was recorded for U.S. twins, in whom larger total DZ variances are pervasive. Such a level of zygosity heterogeneity is not ordinary, since much less has been shown by other types of data including dermatoglyphics, serum cholesterol, tooth size (Christian, 1978; Reed et al., 1975; Potter et al., 1979), and, again, a large anthropometric

set from these same Indian twins (Sharma, 1982: and also see Sharma et al., 1984; Byard et al., 1984).

Furthermore, four of the six significant Indian occlusal genetic estimates were invalidated by evidence of relatively stronger environmental covariance among MZ twins by the second (ADZ/WDZ) test. Thus, both twin sets evidenced large environmental biases on the zygosities, but the pattern was very different, while the Indian twins indicated consistently higher MZ environmental parallelism. Paradoxically, the environment in which "heritability" is measured (and the variability of that environment) had a crucial effect.

Arch size, by contrast, showed uncomplicated genetic variance patterns. Maxillary arch breadth appeared least genetic in both groups. This is a possibly meaningful detail, considering Hunt's (1961) assertion that median palatal suture growth and maxillary breadth are particularly affected during human growth by variable chewing forces associated with dietary consistency.

In our data, the highest heritability estimates produced by calculation of the customary genetic variance ratio were consistently invalidated by violating that ratio's hidden basic assumptions. In particular, upper tooth R/D, upper over lower arch length discrepancy, overjet, cross-bite, BSR, and the second and third principal components (involving BSR, overjet and R/D) exhibited sharply lowered genetic variance when recalculated using the F_{ac} criterion. These lowered heritabilities were not an automatic result of the method, for the F ratios could be raised if total MZ variance exceeded total DZ variance. MZ variance virtually always is less than that for dizygotes, however, and this implies environmental influence over the phenotypic convergence seen in monozygotes.

Arch size variation had a significant genetic component in these American twins. Tooth displacement and cross-bite were the most significantly heritable criteria of occlusion. Some of the cross-bite heritability figures were invalidated by monozygotic environmental covariance, however, while some of the tooth-displacement scores were questionable because of the MZ-DZ mean differences. Only cross-bite extent (absolute value of right plus left sides) and

tooth displacement scores for posterior upper and anterior lower teeth yield genetic variances that were both significant and trustworthy. Significant heritability of overjet, BSR, overbite, and total R/D could not be documented. The final average of heritability estimates for the five major components of occlusion used in calculating the Treatment Priority Index was 25 percent.

In summary, these results demonstrate a considerably increased environmental component of variance in occlusion, as contrasted with earlier estimates in the literature. The greater influence of the environment is also directly confirmed by the dizygote variance ratios, which often fail to demonstrate greater among- than within-pair segregation of variance.

Our h^2 figures computed according to the classic model are actually very similar to Lundstrom's earlier results despite sample size inequities. Lundstrom demonstrated maximum heritability of about 75 percent and average heritability of about 50 percent for occlusal features, corresponding to our initial figures. However, when averaging traditional h^2 with that modified according to the among-component formula (when the latter was indicated), heritabilities dropped sharply.

Thus, environmental determination of occlusal variation is roughly twice as important as was earlier thought, and we suggested that orthodontic researchers may wish to consider environmental correlates of malocclusion more vigorously in the future.

Australian Twins and an Inclusive Comparison

Under a strictly polygenic mode of inheritance with additive genetic effects we would expect maximum correlation coefficient values of 1.00 for MZ twin pairs and 0.50 for DZ twin pairs. The values for incisor relation traits (overjet and overbite) in MZ twins were all considerably less than the theoretical maximum when we (Corruccini, Townsend, Brown and Richards, 1990) compared the Australian, American and Indian twin samples. This emphasized possible

measurement error sources and/or that the occlusal traits are modified by cultural or environmental factors. Furthermore, there was disconcerting disparity both among the different heritability estimate methods and among different populations for the same method. There was generally more MZ within-pair difference, and more DZ among-pair difference in mean squares between populations for overjet. Distinctly larger MZ total variance obtained except in the U.S. sample for overjet. Similar observations held for the overbite trait, where within-pair variances in both zygosities were more stable than among-pair. Especially in terms of depressed DZ variance, the Indian sample showed divergent values for overbite. This is all the more intriguing when one considers the probable different and stronger pattern of environmental stresses in Indian society.

Crossbite type showed all variances quite restricted in the Indian twins, and U.S. variances were relatively inflated. For crossbite extent, only MZ twins were similar in the three Western white samples whereas only the DZ pairs were significantly correlated in the Indians. DZ total variance was very consistently in excess of MZ; U.S. twins had relatively high variances, and Indian twins relatively low.

BSR traits showed consistently lower mean scores for MZ twins, though never to a statistically significant univariate extent. BSR type variances were consistent except DZ among-pair variances were large in the U.S. sample and DZ within-pair variances small in the restricted Australian sample. For BSR extent there was some tendency for larger MZ total variance, and MZ variances (both among- and within-pair) were more consistent than DZ variances. In BSR the unrestricted-sample Australian results again, interestingly, resembled the Indian patterns as compared to the Australian restricted sampling.

R/D scores showed interesting contrasts. MZ total variance was consistently greater for posterior R/D but the opposite obtained for anterior R/D with the exception of Indian twins. The test for CMZ>CDZ was consistently healthy for once in rejecting the null hypothesis. The among-component genetic

variance ratio was considerably more repeatable than the within-pair ratio for posterior R/D, while the opposite was strongly the case in anterior teeth. U.S. twins were much more variable, and the genetic variances unusually unstable for posterior R/D. For anterior R/D within-zygosity variances were stable and among-zygosity variances much more diverse. Thus the genetic variances were relatively invariant. In contrast to the usual situation where heritabilities based on intrapair correlations are higher, the h^2 estimate based on variance formula was more significant for total R/D. The Indian twins evinced a very different overall pattern here compared to all Western samples. The U.S. twins were much more variable with the exception only of the Indian MZ among-pair variance.

Many of these points extended also to the results of the highly correlated TPI score, which epitomized the overall heterogeneity of results between populations. There were wild fluctuations in the measure of cultural inheritance, from the most extreme environmental convergence to the strongest case of divergence among MZ twins. The U.S. and Indian twins showed more MZ variance, and the U.S. only had elevated DZ variance. For this important summary variable, none of the samples excluding orthodontically treated twins yielded an appropriate measure of genetic variance that attained statistical significance.

Although it is generally agreed that a polygenic mode of inheritance is most likely to explain occlusal variation (Smith and Bailit, 1977), our findings indicated that complicated environmental effects contribute more to the variability observed than suggested by early twin studies (and the general tone of the literature). Significant genetic variance was noted for certain traits in certain populations, with limited consistency or repeatability over populations. Heritability estimates were generally low to moderate in magnitude, within-pair estimates for these traits centering around 20 percent.

The MZ intrapair correlation levels held steady between samples of Australian twins whereas the DZ correlations consistently dropped in the

unrestricted samples, creating many changes in results. It could be argued that the changes are both biologically valid and invalid in different frameworks. That is, the greater natural DZ variability is real, especially in terms of within-pair developmental disparity, but extra within-pair DZ variability due to only one member undergoing orthodontics is an artificial, man-made factor. The higher MZ scores for occlusal discrepancy in Indian twins, not seen in the twins from more affluent European-derived populations, suggests the possibility of less adequate maternal prenatal environment and concomitant intrapair competitive divergence in the Indians. Cross-cultural twin comparisons thus should assume greater theoretical importance to general deliberations of heritability of morphological traits in the future, especially with regard to evolutionary questions.

Considering the problems of sampling and differences in methodology, our conventional variance and correlation estimates are similar enough to those of Chung and Niswander (1975) and Lundstrom (1984). Furthermore, the trend noted by Harris and Smith (1980) in a non-western population for genetic components of variance to be greatest for overjet, less for overbite and least for molar relationships is variably confirmed in these present studies.

Another interesting feature is the apparent lack of genetic determination for crossbite, a relationship in the coronal plane. This is consistent with morphometric studies that indicate greater transmissibility for body length than breadth measures (e.g. Byard et al., 1985; Devor et al., 1986). Crossbite was the variable in our study that most consistently failed to fulfill the assumptions of equality of mean values and variances and of no zygosity environmental covariance.

The marked differences between heritability estimates for some of the occlusal traits (e.g. overjet), depending on their method of calculation, highlight the need for caution in interpreting these values. This corroborates Potter and Nance's (1976) assertion that genetic variance ratios are generally more informative than heritability estimates in studies of human populations. Many of both the unusually high and unusually low within-pair genetic variance estimates

are negated by the F' test, lessening the apparent range of estimates for a given trait.

Researchers are more hesitant today when extrapolating from twin studies to the general population. One must ask whether twins differ from singletons for the features under consideration. Boklage (1984) has provocatively stated on the basis of comparisons of dental crown size data that "MZ twins, DZ twins, and singletons are not the same kind of people when the question is about the way their heads are built."

In our cross-population investigation there was little evidence of differences in distributional properties of occlusal data between MZ and DZ twins. Mean values of occlusal traits in MZ twins did not consistently exceed those in DZ twins throughout these samples.

Many of the occlusal traits we have studied are inter-related and it is possible that multivariate approaches might disclose differences in distributional relationships between zygosities not evident in univariate analyses. In this regard, we performed discriminant function analyses. In contrast to the findings of Boklage (1984), this did not disclose statistically significant differences, although larger independent sample sizes are desirable for these types of approaches. Wilks' lambda statistic, based on the determinant of within-zygosity compared to between-zygosity covariance matrices, yields $p=0.078$, but this result is spuriously sensitive due to Type I error resulting from artificially doubling the samples through twin inclusion. Some 60 percent of the MZ individuals can be separated from 65 percent of the DZ by the linear discriminant function, the mean separation amounting to 3.32 standard errors of the mean difference. This would ordinarily indicate statistical significance by the t-test but the test is post-hoc. Box's M test detects significant heterogeneity of the covariance matrices, to be expected from the univariate results. The MZ matrix's determinant is 9.8 percent larger than the DZ, $p<0.0001$.

The question of whether the correlations noted between twins for occlusal traits reflect common genetic effects or common environmental influences is

critical. Traditionally, the twin model assumes that environmental effects are similar for both MZ and DZ twins, and that greater similarity within MZ pairs compared with DZ pairs reflects genetic influences. When the "cultural heritability" approach is adopted in our study, we find quite high values of c^2 for some traits, for example posterior tooth R/D and TPI, pointing to important cultural factors. The c^2 exceeded h^2 in 12 of 40 tests. However, the possibility that sampling effects may have inflated DZ correlations needs to be acknowledged. When we excluded any twin pairs where one or both members had a history of orthodontic treatment or permanent tooth loss, we effectively truncated the complete distribution of occlusal variation. Inclusion of a greater proportion of twin pairs with similar "good" occlusions could have tended to inflate correlations, particularly in the DZ category where greater within-pair discrepancies might otherwise be expected. On the other hand, this unavoidable sampling bias would have inflated the ADZ/WDZ ratio, hiding environmental covariance from the CMZ>CDZ test.

The twin model provides a valuable means of partitioning variability into causal components, but a number of assumptions should be checked first before extrapolating results to the general population. As sample sizes increase my Australian colleagues hope to undertake broader genetic analyses and path analysis, for example fitting models that include parameters for individual environmental variation, additive genetic and dominance variation. These approaches, together with more detailed longitudinal assessment of twin pairs where one or both members have received orthodontic treatment, should provide further insights into the nature and relative importance of the various determinants of occlusal variation.

How Much do Genes Tell Us?

Hereditarian thinking seems to be shifting in the orthodontic literature. King (1983) says:

"Human population studies and animal experimentation have provided considerable support for dietary consistency as an important etiologic factor in malocclusion. But this explanation has not met with widespread acceptance by the dental profession. Dentists, whose focus is usually limited to patients and their immediate families, are apt to emphasize genetic determinants in the etiology of malocclusion. This attitude is reminiscent of the profession's thinking about dental caries, a disease once thought largely to affect genetically predisposed individuals, but now universally recognized as very much dependent on environmental circumstance. It is likely that dentists, initially impressed by familial patterns of caries incidence, were misled into ascribing undue importance to genetic factors. This misconception persists as dental folklore among some patients who believe their caries susceptibility to be the consequence of soft teeth on one side of the family. It is possible that dentistry's myopic preoccupation with familial patterns of malocclusion has led to a similar misconception."

Another dramatic revelation has come from Harris (1990; see also 1991):

"Several craniometric and cephalometric studies of twins and siblings have documented a moderate genetic component for facial size and shape. The genetic influence has, however, been extrapolated to also account for tooth relationships (occlusion, malocclusion), generally in the absence of much data .... This paper described a longitudinal family study based on serial assessments of orthodontically untreated siblings from 4 yrs (full deciduous dentition) to 20 yrs of age (full permanent dentition) .... Results define a clear-cut dichotomy: Craniometric variables (k=25) uniformly have significant additive components; correlations [sib-sib] increase from age 4 to 20; and correlations average 0.43 at adulthood. Tooth-based variables of position and relationship (k=14) erratically achieve significance, and estimates decrease with age to the extent that no variable at age 20 has a correlation significantly

different from zero. In contrast to craniometric variables, with high heritabilities, almost all the occlusal variability is nongenetic .... It was concluded that most malocclusions (in the strict sense of dental malrelationships) are essentially acquired, not inherited."

Harris gives incredible average h^2 estimates (by age 20) of 90 percent for bone-based cephalometric variables and only 10 percent for tooth-based occlusal variables. That tooth position is almost all environmental is verified by Cassidy's (1996) related book. King et al. (1993) elaborate in an important direction from this sibling sample by showing that sibs treated for malocclusion (as opposed to the untreated ones above) have an opposite tendency - very low cephalometric and high occlusal heritabilities. They show that environmental covariance acting on similar genotypes is the most likely cause of this result.

Example: Treatment Priority Index Genetic Variances

Parameter	Australian:		U.S.A.	India
	Restricted	Unrestricted		
t'	-1.14	-0.87	-1.56	1.40
F'	1.07	1.10	1.30*	0.40*
CMZ>CDZ	4.13*	2.27*	2.29*	0.97
F	1.45	1.51*	(1.28)	(1.33)
F_{ac}	(1.06)	(1.34)	0.89	(3.00*)
h^2 (var)	.12	.20*	<.00	(>1.00)
h^2 (holz)	.26	.39*	(<.00)	(.70*)
h^2 (path)	.20	.48*	-.-	(>1.00)
c^2	.51	.15	.73(est.)	-.73
r_{MZ}	.71*	.63*	-.-	.69*
r_{DZ}	.61*	.39*	-.-	-.02
MZ AMS	10.87	12.53	19.44	20.37
MZ WMS	1.83	2.85	8.73	3.69
DZ AMS	10.95	9.75	25.58	4.73
DZ WMS	2.65	4.30	11.18	4.90
Genetic Variance	0.82	1.44	(2.44)	(1.21)

LEGEND:

t': nested AOV for MZ-DZ mean difference

F': total DZ/MZ variance

CMZ>CDZ: F-ratio ADZ/WDZ

F: within-pair genetic variance ratio

F_{ac}: among-component genetic variance ratio

h^2(var): within-pair or among-component variance heritability estimate

h^2(holz): Holzinger intrapair-correlation heritability

h^2(path): $2(r_{MZ}-r_{DZ})$

c^2: cultural heritability; $2r_{DZ}-r_{MZ}$

r_{MZ}: intrapair MZ correlation

r_{DZ}: intrapair DZ correlation

ams: among-pair mean squares; wms: within-pair mean squares

genetic variance: WDZ-WMZ mean squares

* signifies statistically significant test, rejecting a null hypothesis. Numbers in parentheses signify an untrustworthy result due to violation of an assumption.

8. Conclusion.

Anthropology has long had data and ideas to offer the craniofacial clinical specialties, of which the latter have taken notice to a variable degree. As we have seen, the "disuse" theory for occlusal deterioration, the idea that we no longer use our masticatory apparatus vigorously and so it does not grow properly, is quite old. Disuse was always included in the original etiological lists in Angle's and other early orthodontic textbooks. It vanished from such lists in later orthodontic texts. Nevertheless we still await a determination as to the mechanism's importance, both with regard to causation and to intervention and/or prevention of malocclusion. What is intriguing is the long historical interest in the disuse etiology, its regular periodic resurrection and subsequent resubmersion, and the eternal quality of the rhetoric surrounding orthodontic opinions.

Human occlusion has changed dramatically in very recent evolutionary times. Cross-cultural surveys therefore are of key importance to solving this mystery, for the Western world has finished crossing the developmental transition through which this change occurred. Examination of such etiologies as disuse, nutrition, genetics (heritability, admixture, and inbreeding), oral breathing, attrition, caries, deciduous tooth exfoliation, muscle recruitment, temporomandibular function, habits (nursing, sleeping and finger-sucking), bruxism (and even thegosis), tongue dynamics, and cutlery use must of necessity be conducted in "anthropological" populations in order to maximize the explanatory power of our findings.

Malocclusion syndromes follow certain non-random patterns of occurrence that support the idea of an underlying chewing stress principal factor

as opposed to the myriad other etiologies. As Sim Wallace (1904) long ago pointed out,

> "There is a forward force which is the resultant of the outward and slightly forward pressure of the tongue, and the inward and forward pressure of the buccinator muscle. (These are not imaginary forces, as the mucous membrane of the cheek at the side of and behind the hindmost molars is frequently marked and indented by its pressure on these teeth, and the marks caused by the pressure of the lower teeth on the tongue may also frequently be seen.) .... the mandible in the anterior part is but little changed in general size after the closure of the two lateral halves of the symphysis about the age of six months, while on the other hand the suture uniting the maxillary and palate bones remains open during the whole of the time that the development of these bones goes on. It consequently happens that the maxillary bones, from lack of developmental stimuli, retain the developing teeth more inwards than if the maxillary bones had been developed outwards. Moreover, the crowns of the maxillary teeth are normally outside the lower ones, and the act of masticating or biting tends to force the lower incisors inward, while this force cannot possibly take effect in a similar direction in the case of the upper incisors .... The result of this is that the crowns of the incisors of the upper jaw tend to radiate to a certain extent. On the other hand, the lower incisors and canines in a somewhat deficiently developed jaw tend to have the crowns falling inwards towards the tongue, the initial point from which these teeth develop not depending for its breadth to such an extent on the stimulus brought about by efficient mastication."

Sim Wallace's idea was that the buccinator muscle's relentless restriction, when unopposed by forceful tongue action on the other side, causes arch constriction and crowding. The idea has been energetically reintroduced by the late Sid Frederick (1985).

The functional deficiency process outlined above explains maxillae that are too narrow (which are vastly more common than maxillae too broad), and upper incisors that are too procumbent while lower incisors are too lingual and prone to deep bite (vastly more frequent that the opposite conditions, mandibular overjet and openbite, respectively). In addition, the ongoing mandibular growth occurs posteriorly with a forward trajectory from the condyles, and is thus most susceptible to inadequacy in the antero-posterior rather than lateral direction. Perhaps this explains why modern soft diet is associated with relatively deficient forward mandibular growth, leading to the orthognathism which in the white populations at least far exceeds mandibular prognathism. Further evidence lies in the greater frequency of mandibular than maxillary impacted third molars (Mucci, 1982; Klatsky and Fisher, 1953), a syndrome that is part of the overall malocclusion tendency treated in this book and that similarly undergoes a rapid epidemiological transition in modern times (Mucci, 1982). Maxillae, on the other hand, are more affected by lateral growth from the mediopalatine suture and therefore functional insufficiency causes maxillary narrowing -- thus posterior maxillary lingual crossbite is seen much more than its buccal counterpart.

Finally, remember that dental crowding and alveolar space insufficiency is easily the most common single form of malocclusion and contributor to orthodontic need. The prevailing etiologies of clinical interest (as identified by the relative amount of space dedicated to them in orthodontic textbooks) are of little help in explaining this crowding, as opposed to explaining openbite or crossbite or other comparatively rare (but admittedly serious) syndromes. Proffit (1986b) asserts that malalignment is genetically influenced but severe crowding has an added environmental input. Conversely contact relations are genetic in their most severe discrepancies. If this were so, one would expect soft diet experimental animals to exhibit more change in contact discrepancy between arches than malalignment within arches. The opposite is actually what happens.

What would happen if indeed "disuse" became widely accepted clinically as the underlying cause of most malocclusions? Are there realistic

prospects for changes in dietary consistency that would effectively prevent malocclusion in coming generations? I think not, and cannot seriously propose a program in that direction. Klatsky and Fisher (1953) also considered this problem in their concluding section:

> "The greatest problem is the restoration of proper function to these organs by means of hard, fibrous foods, such foods which stimulate the masticatory apparatus to good function, and those which cause a greater flow of alkaline saliva which serves as therapeutic lubricant of the teeth and oral cavity. The adoption of this preventive method by our public health institutions would be a great service to humanity in general and our children in particular. A wide educational campaign for this purpose will be necessary."

Klatsky and Fisher give some of the potential difficulties with using the function-stimulates-form argument in a preventative program:

> "Eating soft and liquid food has become a custom and habit. We have created a special code of so-called table manners. Father and mother feel it their parental duty to instill refined and graceful eating manners in their children. Food must be chewed gently and imperceptibly. Wide opening of the mouth, forceful chewing, lip smacking are bad style; hence, they are tabooed. Table manners, which are the direct outgrowth of our rushing civilization, were unknown to primitive people."

Klatsky and Fisher continue: "Brekhus cannot see eye to eye with us how this loss of function can possibly be restored to the mouth of modern man. He is particularly vehement in his arguments against a change from a soft to a hard diet." They quote Brekhus (1941):

> "... numerous members of the medical profession would rise up to warn us

against the effects that such a revolution in diet -- beneficial, perhaps, to our teeth -- would be likely to produce in our digestive system .... How long would it take for the combined efforts of the medical and dental professions to win the public way from its devotion to modern concentrated foods, even if those professions were sure such a change were desirable? How long would it take to overcome the protests of the manufacturers and advertisers of such foods? .... Despite the proved efficacy of vaccination, it has taken a century and a half to sell it to the public, and even now there are thousands of persons who fight for their right to remain unprotected from smallpox, though the means of prevention is simple, inexpensive, and of proved worth. How, then, could we expect to have such success in educating people to adopt a regimen that would not only disrupt their whole manner of life but that also could not at present be shown to be indubitably effective?"

I would have to agree, especially as regards the poor prospects for enticing our 2-5 year old children to masticate resistant foodstuffs -- especially when they know there are tenderer delicacies around somewhere. I have had no success with my own children in promoting the consumption of the few really tough items commercially available today. They know the macaroni and cheese is hidden somewhere and that it is just a matter of holding out.

As Moyers and Wainright (1977) observe, theoretical models of craniofacial growth are currently trendy. Moyers and Wainright's model emphasizes the inherent position of the mandibular first molar at eruption; growth then adapts to the established occlusal relation. "If this concept is correct it places much emphasis upon the role of built-in feed-back responses in the guidance of occlusal development. It also suggests strongly the rationale of and target sites for early orthodontic therapy, as contrasted with the usual late treatment where teeth are moved to camouflage a disharmonious face. In the new orthodontics our emphasis may change to altering the conditions which determine the pattern of occlusal development rather than altering the occlusion directly.

We will change the epigenetic environment of young children rather than banding the teeth of 12 year olds." Why can't chewing function play a central role in such manipulation of epigenetic conditions? Why is this possibility not stated? And why is the masticatory communication between maxillary and mandibular antagonists not accorded its rightful critical function in determining that early first molar occlusion? Petrovic and Stutzmann (1977) similarly develop a model of a maxillary-mandibular servosystem involving intercommunication between the antagonists, that adjusts mandibular growth through the intermediary of muscle function. They suggest altering the servosystem with appliances to alter the pattern of muscle activity. I fail to understand how they miss the potential input of strong vertical bite forces to such a system, creating stronger "input" to the occlusal guidance system established by the cusps.

If there is eventually to be the possibility of steps involving masticatory exercise to prevent or mitigate craniofacial disorder, these more likely will employ appliances and artificial alveolar stimulation rather than dietary alteration. This will likely be particularly critical during a certain age span (which needs to be pinpointed, by research that varies and staggers the timing of changes in dietary consistency).

My point in this book is not, at this juncture, to recommend any specific sort of therapy (although a spring-loaded chewing resistance device for alveolar exercise comes to mind), but rather to promote different directions of research. The door could be opened a bit wider by the clinical specialists to this brand of functional thinking, and much greater research and funding emphasis should go to Disuse and its ramifications rather than toward invasive experiments that have only corrective, not preventive, significance.

9. Bibliography

Ahlgren J (1966) Mechanics of mastication. A quantitative cinematographic and electromyographic study of masticatory movements in children, with special reference to the occlusion of teeth. Acta Odont Scand 24.

Ahlgren J, Ingervall B, Thilander B (1973) Muscle activity in normal and postnormal occlusion. Am J Orthodont 64:445-456.

Anderson DL, Popovich F (1983) Lower cranial height versus craniofacial dimensions in Angle Class II malocclusion. Angle Orthodont 53:253-260.

Anderson DL, Popovich F, Saunders M (1980) Occlusion related to eruption sequences. J Dent Res 59:540.

Angel JL (1944) Greek teeth: ancient and modern. Hum Biol 16:283-297.

Angle EH (1907) Treatment of Malocclusion of the Teeth. 7th Ed. Philadelphia: SS White.

Angle J, Wissman DA (1980) The epidemiology of myopia. Am J Epidem 11:220-228.

Arya B, Savara BS, Thomas DR (1973) Prediction of first molar occlusion. Am J Orthodont 63:610-621.

Ast DB, Allaway N, Draker HL (1962) The prevalence of malocclusion, related to dental caries and lost first permanent molars, in a fluoridated city and a fluoride-deficient city. Am J Orthodont 48:106-113.

Ast DB, Carlos JP, Cons NC (1965) The prevalence and characteristics of malocclusion among senior high school students in upstate New York. Am J Orthodont 51:437-445.

Bachrach H, Young M (1927) A comparison of the degree of resemblance in dental characters shown in pairs of twins of identical and fraternal types. Brit Dent J 21:1293-1304.

Bachrach H, Young M (1928) A comparison of the degree of resemblance in dental characters shown in pairs of twins of identical and fraternal types. Dent Cosmos 70:465-466.

Bailit HL (1975) Dental variation among populations. Dent Clin N Am 19:125-139.

Bailit HL, Ogan E, Leigh R (1968) Oral health of the Nasioi of Bougainville. Austral Dent J 13:353-359.

Ballard CF (1963) Variations of posture and behaviour of the lips and tongue which determine the position of the labial segments. Trans Eur Orthodont Soc

39:67.
Barber CG, Green LJ, Cox CJ (1963) Effects of the physical consistency of diet on the condylar growth of the rat mandible. J Dent Res 42:848-851.
Barnard PD (1956) Dental survey of state school children in New South Wales. National Health and Medical Research Council (Canberra) Special Report Series, 8.
Barrett MJ (1953) X-Occlusion. Dent Mag Oral Topics 70:279.
Barrett MJ (1956) Dental observations on Australian aborigines: water supplies and endemic dental fluorosis. Austral Dent J 1:87-92.
Barrett MJ (1957a) Serial dental casts of Australian Aboriginal children. Austral Dent J 2:74.
Barrett MJ (1957b) Dental observations on Australian Aborigines: tooth eruption sequence. Austral Dent J 2:217-227.
Barrett MJ (1969) Functioning occlusion. Ann Austral Coll Dent Surg 2:61-67.
Barrett MJ, Brown T, Cellier KM (1964) Tooth eruption sequences in a tribe of Central Australian Aborigines. Am J Phys Anthrop 22:79-89.
Barrett MJ, Brown T, Fanning EA (1965) A long-term study of the dental and craniofacial characteristics of a tribe of Central Australian aborigines.Austral Dent J 10:63-68.
Barrett MJ, Williamson JJ (1972) Oral health of Australian aborigines: survey methods and prevalence of dental caries. Austral Dent J 17:37-50.
Baume LJ (1973) The pattern of dental disease in French Polynesia. Int Dent J 23:579-584.
Baume L, and many others (1973) A method for measuring occlusal traits. Int Dent J 23:530-537.
Bear JC (1982a) The epidemiology and genetics of refractive error. Am J Phys Anthrop 57:168-169.
Bear JC (1982b) Why can't we explain refractive error? Canad J Optom 44:181-188.
Bear JC, Richler A (1981) Ocular refraction and inbreeding: a population study in Newfoundland. J Biosoc Sci 13:391-399.
Bear JC, Richler A (1983) Cylindrical refractive error: a population study in Western Newfoundland. Am J Optom Physiol Opt 60:39-45.
Bear JC, Richler A, Burke G (1981) Nearwork and familial resemblances in ocular refraction: a population study in Newfoundland. Clin Gen 19:462-472.
Beckmann SH, Kuitert RB,Prahl-Andersen B, Segner D, The RPS, Tuinzing DB (1998) Alveolar and skeletal dimensions associated with overbite. Am J Orthodont Dentofac Orthoped 113:443-452.
Beecher R, Corruccini R (1981a) Effects of dietary consistency on craniofacial and occlusal development in the rat. Angle Orthodont 51:61-69.
Beecher R, Corruccini R (1981b) Effects of dietary consistency on maxillary

arch breadth in macaques. J Dent Res 60:68.
Beecher RM, Corruccini RS, Freeman M (1983) Craniofacial correlates of dietary consistency in a nonhuman primate. J Craniofac Gen Dev Biol 3:193-202.
Begg PR (1935) Studies on certain aspects of the aetiology of malocclusion of the teeth and concomitant jaw anomalies. Doctorate Thesis, University of Adelaide.
Begg PR (1954) Stone age man's dentition. Am J Orthodont 40:298-312, 373-383, 462-475, 517-531.
Begg PR (1956) Differential force in orthodontic treatment. Am J Orthodont 42:481-510.
Begg PR (1965) Begg Orthodontic Theory and Practice. Philadelphia: Saunders.
Begg PR, Kesling PC (1971) The differential force method of orthodontic treatment. Am J Orthodont 71:1-39.
Begg PR, Kesling PC (1977) Begg Orthodontic Theory and Technique. 3rd Ed. Philadelphia: Saunders.
Berry DC (1976) Excessive attrition. In Poole DFG, Stack MV (eds) Eruption and Occlusion of Teeth. London: Butterworths. pp. 146-155.
Best E, Roberts D, Ram C (1982) Actual bite-force values using a photo-elastic check-bite wafer. J Dent Res 61:300.
Beyron HL (1954) Characteristics of functionally optimal occlusion and principles of occlusal rehabilitation. J Am Dent Assoc 48:648.
Beyron HL (1964) Occlusal relations and mastication in Australian aborigines. Acta Odontol Scand 22:597-678.
Bhalla V (1966) Blood group distribution pertaining to ABO, MNSs and Rh-Hr systems in the Indian subcontinent. Anthropologie 4:67-86.
Bixler D (1974) Genetic aspects of dental anomalies in children. In McDonald RE (ed.) Dentistry for the Child and Adolescent. St. Louis: Mosby.
Bjork A (1947) The Face in Profile. Svensk Tand 40 (Supp 5b).
Bjork A (1950) Some biological aspects of prognathism and occlusion of the teeth. Acta Odont Scand 9(1).
Bjork A, Brown T, Skieller V (1984) Comparison of craniofacial growth in an Australian Aboriginal and Danes, illustrated by longitudinal cephalometric analyses. Eur J Orthodont 6:1-14.
Bjork A, Helm S (1969) Need for orthodontic treatment as reflected in the prevalence of malocclusion in various ethnic groups. Sartryck Ur (Acta Socio-Med Scand) Suppl. 1:209-214.
Blake M, Bibby K (1998) Retention and stability: a review of the literature. Am J Orthodont Dentofac Orthoped 114:299-306.
Bodley JH (1982) Victims of Progress. 2nd Ed. Menlo Park: Benjamin Cummings.
Boklage CE (1984) Differences in protocols of craniofacial development related to twinship and zygosity. J Craniofac Gen Dev Biol 4:151-169.

Bonacelli B (1928) La natura e gli Etruschi. Studi Etruschi II:427-569.

Boraas JC, Messer LB, Till MJ (1988) A genetic contribution to dental caries, occlusion, and morphology as revealed by twins reared apart. J Dent Res 67:1150-1155.

Bouvier M, Hylander WL (1981) Effect of bone strain on cortical bone structure in macaques (Macaca mulatta). J Morphol 167:1-12.

Bowles GT (1977) The People of Asia. New York:Scribner's.

Brace CL (1977) Occlusion to the anthropological eye. In McNamara J (ed.) The Biology of Occlusal Development, Monograph 7. Ann Arbor: Craniofacial Growth Series, Center for Human Growth and Development, pp. 179-209.

Brace CL (1980) Australian tooth-size clines and the death of a stereotype. Curr Anthrop 21:141-164.

Brace CL, Mahler PE (1971) Post-Pleistocene changes in the human dentition. Am J Phys Anthrop 34:191-204.

Brash JC (1924) The genesis and growth of deformed jaws and palates. In The Growth of the Jaws, Normal and Deformed, in Health and Disease, London: Dental Board U.K.

Brash JC, McKeag HTA, Scott JH (1956) The Aetiology of Irregularity and Malocclusion of the Teeth. 2nd Ed. London: Dental Board U.K.

Brekhus, PJ (1941) Your Teeth. Minneapolis: University of Minnesota.

Bresolin D, Shapiro PA, Shapiro GG, Chapko MK, Dassel S (1983) Mouth breathing in allergic children: its relationship to dentofacial development. Am J Orthodont 83:334-340.

Brown DE (1981) The effects of social phenomena on physiological stress levels of Filipino-Americans. Am J Phys Anthrop 55:205-206.

Brown P (1987) Pleistocene homogeneity and Holocene size reduction: the Australian human skeletal evidence. Archaeol Oceania 22:41-67.

Brown T (1976) Head size increases in Australian Aboriginals: an example of skeletal plasticity. In Kirk RL, Thorne AG (eds), The Origin of the Australians. Australian Institute of Aboriginal Studies, Canberra, pp. 195-209.

Brown T (1978) Tooth emergence in Australian Aboriginals. Ann Hum Biol 5:41-54.

Brown T (1985) Occlusal development and function. In Simoes WA (ed.) : Functional Orthopaedics of the Jaws. Vistas in Neuro-occlusal Rehabilitation. Sao Paulo: Livraria Editora Santos. pp. 1-67.

Brown T, Abbott A, Burgess VB (1983) Age changes in dental arch dimensions of Australian Aboriginals. Am J Phys Anthrop 62:291-303.

Brown T, Abbott AH, Burgess VB (1986) Longitudinal study of dental arch relationships in Australian Aboriginals with reference to alternate intercuspation. Am J Phys Anthrop 72:49-58.

Brown T, Jenner JD, Barrett MJ, Lees GH (1979) Exfoliation of deciduous teeth

and gingival emergence of permanent teeth in Australian Aborigines. Occ Pap Hum Biol, (Australian Institute of Aboriginal Studies, Canberra) 1:47-70.

Brown T, Margetts B, Townsend GC (1980a) Comparison of mesiodistal crown diameters of the deciduous and permanent teeth in Australian Aboriginals. Austral Dent J 25:28-33.

Brown T, Margetts B, Townsend GC (1980b) Correlations between crown diameters of the deciduous and permanent teeth of Australian Aboriginals. Austral Dent J 25:219-223.

Brown T, Townsend GC, Richards LC, Burgess VB (1990) Concepts of occlusion: the Australian evidence. Am J Phys Anthrop 82.

Bulmer MG (1970) The Biology of Twinning in Man. Oxford:Clarendon.

Byard PJ, Poosha DVR, Satyanarayana M, Rao DC (1985) Family resemblance for components of craniofacial size and shape. J Craniofac Gen Dev Biol 5:229-238.

Byard PJ, Sharma K, Russell JM, Rao DC (1984) A family study of anthropometric traits in a Punjabi community: II. An investigation of familial transmission. Am J Phys Anthrop 64:97-104.

Calcagno JM, Gibson KR (1988) Human dental reduction: natural selection or the probable mutation effect. Am J Phys Anthrop 77:505-517.

Campbell TD (1925) Dentition and Palate of the Australian Aboriginal. Adelaide: Hassell.

Campbell TD (1938) Observations on the teeth of Australian aborigines, River Diamentia. Austral. J Dent 42:121-125.

Campbell TD (1946) Tooth cusps. Austral J Dent 50:305-307.

Campbell TD, Barrett MJ (1953) Dental observations on Australian Aborigines -- a changing environment and food pattern. Austral J Dent 57:1-6.

Campbell TD, Lewis AJ (1926) The aborigines of South Australia: dental observations recorded at Ooldea. Austral J Dent 30:371-376.

Camporeale G (1984) La Caccia in Etruria. Rome: G. Bretschneider.

Capasso L (1985) L'Origine delle Malattie. Chieti: Marino Solfanelli.

Capasso L (1986) Etruria: le meraviglie dei dentisti. In Capasso L, La Medicina nell'Antichita. Archeo Dossier, 13.

Capasso L (n.d.) Dental pathology and alimentary habits reconstruction of Etruscan population. Studi Etruschi 53(3).

Carlson DS, Van Gerven DP (1977) Masticatory function and post-Pleistocene evolution in Nubia. Am J Phys Anthrop 46:495-506.

Carlsson GE (1974) Bite force and chewing efficiency. Front Oral Physiol 1:265-292.

Casotti L (1957) Vetulonia Etrusca e stomotologia. Riv Ital Stomatol 12:96-112.

Cassidy KM (1996) Heritability Estimates of Arch Form: A Sibling Analysis. Memphis: University of Tennessee.

Charlier J-P, Petrovic A, Herrmann-Stutzmann J (1969) Effects of mandibular hyperpropulsion on the prechondroblastic zone of young rat condyle. Am J Orthodont 55:71-74.

Christian JC (1979) Testing twin means and estimating genetic variance. Basic methodology for the analysis of quantitative twin data. Acta Genet Med Gemellol 28:35-40.

Christian JC, Feinleib M, Norton JA (1975) Statistical analysis of genetic variance in twins. Am J Hum Genet 27:807.

Christian JC, Kang KW, Norton JA (1974) Choice of an estimate of genetic variance from twin data. Am J Hum Genet 26:154-161.

Christian JC, Norton JA (1977) A proposed test of the difference between the means of monozygotic and dizygotic twins. Acta Gen Med Gemellol 26:49-53.

Chung CS, Niswander JD (1975) Genetic and epidemiologic studies of oral characteristics in Hawaii's schoolchildren: V. Sibling correlations in occlusion traits. J Dent Res 54:324-329.

Chung CS, Niswander JD, Runck DW, Bilben SE, Kau, MCW (1971) Genetic and epidemiologic studies of oral characteristics in Hawaii's schoolchildren. II. Malocclusion. Am J Hum Genet 23:471-495.

Ciochon RL, Nisbett RA, Corruccini RS (1997) Dietary consistency and craniofacial development related to masticatory function in minipigs. J Craniofac Genet Dev Biol 17:96-102.

Clarke NG, Carey SE, Sirkandi W, Hirsch RS, Leppard P (1986) Periodontal disease in ancient populations. Am J Phys Anthrop 71:173-183.

Clegg EJ, Garlick JP (eds.) (1980) Disease and Urbanisation. London:Taylor and Francis.

Clinch L (1951) The occlusion of the Australian aborigines. Trans Eur Orthodont Soc 80-93.

Colyer JF (1931) Abnormal Conditions of the Teeth of Animals and their Relationship to Similar Conditions in Man. London: U.K. Dental Board.

Colyer JF (1936) Variations and Diseases of the Teeth of Animals. London: Bale, Sons, Danielsson.

Cooke RA, VanderVeer A (1916) Human sensitization. J Immunol 1:201.

Coon CS (1950) The mountains of giants: a racial and cultural study of the north Albanian mountain Ghegs. Pap Peabody Mus Archaeol Ethnol 23:1-105.

Corruccini RS (1984) An epidemiologic transition in dental occlusion in world populations. Am J Orthodont 86:419-426.

Corruccini RS (1990) Australian aboriginal tooth succession, interproximal attrition, and Begg's theory. Am J Orthodont 97:349-357.

Corruccini RS (1991) Anthropological aspects of orofacial and occlusal variations and anomalies. In Advances in Dental Anthropology (eds. M.A. Kelley and C.S. Larson). New York: Wiley-Liss, pp. 295-323.

Corruccini RS, Beecher RM (1982) Occlusal variation related to soft diet in a nonhuman primate. Science 218:74-76.

Corruccini RS, Beecher RM (1984) Occlusofacial morphological integration lowered in baboons raised on soft diet. J Craniofac Gen Dev Biol 4:135-142.

Corruccini RS, Chowdhury AFH (1985) Dental occlusal variation among rural and urban Bengali youths. Hum Biol 58:61-66.

Corruccini RS, Flander LB, Kaul SS (1985) Mouth breathing, occlusion, and modernization in a North Indian population. Angle Orthodont 55:190-196.

Corruccini RS, Henderson AM, Kaul SS (1985) Bite-force variation related to occlusal variation in rural and urban Punjabis (North India). Arch Oral Biol 30:65-69.

Corruccini RS, Kaul SS (1983) The epidemiological transition and anthropology of minor chronic non-infectious diseases. Med Anthrop 7:36-50.

Corruccini RS Kaul SS, Chopra SRK, Karosas J, Larsen MD, Morrow C (1983) Epidemiological survey of occlusion in North India. Brit J Orthodont 10:44-47.

Corruccini RS, Lee GTR (1984) Occlusal variation in Chinese immigrants to the United Kingdom and their offspring. Arch Oral Biol 29:779-782.

Corruccini RS, Macchiarelli R (1987) Variabilita occlusale ed anomalie nella modellistica antropologica. In Gambacorta G (ed) L'Ortodonzia nei suoi sviluppi storici. Milan: Ars Medica Antiqua, pp. 93-103.

Corruccini RS, Pacciani E (1983) Occlusal variation in melanesians from Bougainville, Malaita, and New Britain. Homo 33:15-22.

Corruccini RS, Pacciani E (1989) "Orthodontistry" and dental occlusion in Etruscans. Angle Orthodont 59:61-64.

Corruccini RS, Potter RHY (1980) Genetic analysis of occlusal variation in twins. Am J Orthodont 78:140-154.

Corruccini RS, Potter RHY, Dahlberg AA (1983) Changing occlusal variation in Pima Amerinds. Am J Phys Anthrop 62:317-324.

Corruccini RS, Potter RHY, Green LJ (1981) Variance of occlusion traits in twins. J Craniofac Gen Dev Biol 1:217-227.

Corruccini RS, Sharma K (1985) Within- and between-zygosity variance in oral traits among US and Punjabi twins. Hum Hered 35:314-318.

Corruccini RS, Sharma K, Potter RHY (1986) Comparative genetic variance and heritability of dental occlusal variables in U.S. and northwest Indian twins. Am J Phys Anthrop 70:293-299.

Corruccini RS, Townsend GC, Brown T (1990) Occlusal variation in Australian aboriginals. Am J Phys Anthrop 82:257-265.

Corruccini RS, Whitley LD (1981) Occlusal variation in a rural Kentucky community. Am J Orthodont 79:250-262.

Corruccini RS, Whitley LD, Kaul SS, Flander LB, Morrow CA (1985) Facial height and breadth relative to dietary consistency and oral breathing in two

populations (North India and U.S.). Hum Biol 57:151-161.
Craven AH (1958) A radiographic cephalometric study of the central Australian aborigine. Angle Orthod 28:12-35.
Cristofani M (1985) Le attivita produttive. In Catalogo della mostra "Civilta degli Etruschi". Regione Toscana: ELECTA, Chap. 6.
Davies DM (1972) The influence of teeth, diet, and habits on the human face. London: W Heinemann.
Dawes BE (1986) Dental arch crowding in prehistoric man, and in indigenous racial groups of North America and Australia. M.D.S. Thesis, University of Sydney.
Dawson PE (1974) Evaluation, Diagnosis, and Treatment of Occlusal Problems. St Louis: Mosby.
Dechow PD, Carlson DS (1982) Development of masticatory muscle force in macaques (abstract). J Dent Res 61:211.
De Grossi Mazzorin J (1985) Reperti faunistici dall'acropoli di Populonia: testimonianze di allevamiento e caccia nel III secolo a.C. Rass Archeol 5:131-171.
Detlefson JA (1928) Intrinsic or hereditary factors versus extrinsic or environmental factors in the profile of the facial skeleton. J Dent Res 8:419-420.
Devor EJ, McGue M, Crawford MH, Lin PM (1986) Transmissible and non-transmissible components of anthropometric variation in the Alexanderwohl Mennonites: 1. Description and familial correlations. Am J Phys Anthrop 69:71-82.
Dickson GC (1970) The natural history of malocclusion. Dent Pract 20:216-232.
Dobzhansky T (1962) Mankind Evolving. New Haven: Yale.
Draker HL (1960) Handicapping labiolingual deviations: a proposed index for public health purposes. Am J Orthodont 46:295.
Dunn CG (1894) L'Arte Dentaria fra gli Etruschi. Florence: G Barbera.
Dunphy EB, Stoll MR, King SH (1968) Myopia among American male graduate students. Am J Ophthal 65:518-521.
Feldman MW, Lewontin RC (1975) The heritability hang-up. Science 190:1163-1168.
Fields HW, Proffit WR, Nixon WL, Phillips C, Stanek E (1984) Facial pattern differences in long-faced children and adults. Am J Orthodont 85:217-223.
Fishman LS (1976) Dental and skeletal relationships to attritional occlusion. Angle Orthodont 46:51-63.
Flander LB (1981) Childhood Respiratory Allergy in Hawaii. Dissertation, University of Colorado, Boulder.
Flander LB (1982) Influence of chronic respiratory allergy on orofacial growth. Am J Phys Anthrop 57:188.

Fleagle JG, Mittermeier RA, Skopec AL (1981) Differential habitat use by Cebus apella and Saimiri sciurea in Central Surinam. Primates 22:361-367.

Forsberg C-M (1988) Tooth size, spacing, and crowding in relation to eruption or impaction of third molars. Am J Orthodont Dentofac Orthoped 94:57-62.

Foster TD, Hamilton MC, Lavelle CLB (1969) Dentition and dental arch dimensions in British children at the age of two and one-half to three years. Arch Oral Biol 14:1031-1040.

Frederick, S (1985) The buccinator muscle and alveolar processes. Manuscript of lecture at Am Assoc Orthodont meeting, Las Vegas.

Friedl, J (1978) Health care services and the Appalachian migrant. Washington: Nat Ctr Health Serv Res.

Garn SM (1961) Research and malocclusion. Am J Orthodont 47:661-673.

Garn SM, Cole PE, Bailey SM (1979) Living together as a factor in family-line resemblances. Hum Biol 51:565-587.

Gerber LM (1978) Cardiovascular Disease Mortality among Filipino Hawaiians. Dissertation, University of Colorado, Boulder.

Gerber LM (1980) The influence of environmental factors on mortality from coronary heart disease among Filipinos in Hawaii. Hum Biol 52:269-278.

Gerber LM, Madhavan S (1980) Epidemiology of coronary heart disease in migrant Chinese populations. Med Anthrop 4:307-320.

Gerrard JW et al. (1976) The familial incidence of allergic disease. Ann Allerg 36:10-15.

Gill PS (1980) A study of genetic markers and dermal ridge configurations in the lowland Rajputs of Punjab. M.Sc. Thesis, Panjab University, Chandigarh.

Gill PS, Bhalla V, Sunderland E, Woolley V, Chopra SRK (1982) Digital and palmar dermatoglyphics in the lowland Rajputs of Punjab. Bionature 2:69-76.

Gill PS, Sunderland E, Woolley V, Chopra SRK (1983) ABO blood groups, ABH secretion, color-blindness and PTC taste thresholds in the lowland Rajputs of the Punjab. Dyn 8:15-25.

Glazer I (1969) Etiologic factors of bronchial asthma in Israel. Int Arch Allerg Appl Immunol 36:172-179.

Godfrey RC (1975) Asthma and IgE levels in rural and urban communities of the Gambia. Clin Allerg 5:201-207.

Goose DH (1956) Variability of the form of maxillary permanent incisors. J Dent Res 35:902.

Goose DH (1963) Dental measurement: as assessment of its value in anthropological studies. In Brothwell DR (ed) Dental Anthropology, New York: Macmillan, pp. 125-148.

Goose DH (1972) Maxillary dental arch width in Chinese living in Liverpool. Arch Oral Biol 17:231-233.

Goose DH, Lee GTR (1976) Prevalence of dental caries of Chinese in Liverpool.

Ann Hum Biol 3:187-188.
Goose DH, Roberts EE (1979) Possible influence of fluoridation on tooth crown size. J Dent Res 58:1562-1563.
Goose DH, Thomson DG, Winter FC (1957) Malocclusion in school children of the West Midlands. Brit Dent J 102:174-178.
Gould SJ (1981) The Mismeasure of Man. New York: WW Norton.
Gould SJ (1986) Of Kiwi eggs and the Liberty Bell. Nat Hist 95:20-29.
Graber TM (1972) Orthodontics. Principles and Practice. 3rd Ed. Philadelphia: Saunders.
Grainger RM (1967) Orthodontic treatment priority index. Vital and Health Statistics, Ser. 2, No. 25, USPHS, Washington.
Gregory WK (1929) Our Face from Fish to Man. New York-London: Putnam's.
Harris EF (1990) Heritability of craniometric and occlusal variables: a longitudinal sib analysis (abstract). Am J Phys Anthrop 81:236.
Harris EF, Johnson MG (1991) Heritability of craniometric and occlusal variables: a longitudinal sib analysis. Am J Orthodont Dentofac Orthoped 99:258-268.
Harris EF, Smith RJ (1980) A study of occlusion and arch widths in families. Am J Orthodont 78:155-163.
Harris JE (1971) Problems in the statistical inspection of cranio-facial variables during growth and development. In Moyers RE, Krogman WM (eds), Cranio-Facial Growth in Man. Oxford: Pergamon. pp. 229-237.
Harris JE (1975) Genetic factors in the growth of the head. Dent Clin N Am 19:151-160.
Harris JE, Kowalski CJ (1976) All in the family: use of familial information in orthodontic diagnosis, case assessment and treatment planning. Am J Orthodont 69:493-510.
Harris JE, Kowalski CJ, Walker SJ (1975a) Intrafamilial dentofacial associations for Class II, Division 1 probands. Am J Orthodont 67:563-570.
Harris JE, Kowalski CJ, Walker SJ (1975b) Dentofacial differences between normal sibs of class II and class III patients. Angle Orthodont 45:103-107.
Harvold EP (1968) The role of function in the etiology and treatment of malocclusion. Am J Orthodont 54:883-898.
Harvold E, Vargervik K, Chierici G (1973) Primate experiments on oral sensation and dental malocclusions. Am J Orthodont 63:494-508.
Haseman JK, Elston RC (1970) The estimation of genetic variance from twin data. Behav Gen 1:111-119.
Heithersay GS (1960) Attritional values for Australian aborigines -- Haasts Bluff. Austral Dent J 5:84-88.
Helm S (1979) Etiology and treatment need of malocclusion. J Canad Dent Assoc 12:673-676.

Helm S, Prydso U (1979) Prevalence of malocclusion in medieval and modern Danes contrasted. Scand J Dent Res 87:91-97.

Hendel CJ, Corruccini RS, Kaul SS (1983) Descriptive epidemiology of refractive error in a modernizing population of North India. J Hum Evol 12:487-490.

Henrikson P-A, Sagne S, Thilander H (1977) Bone, teeth, and muscle function. Calcif Tiss Res Supp 22:466-467.

Hiiemae K, Ardran G (1968) A cineradiographic study of feeding in Rattus norvegicus. J Zool 154:139-154.

Hill IN, Blayney JR, Wolf W (1959) The Evanston dental caries study. XIX. Prevalence of malocclusion of children in a fluoridated and control area. J Dent Res 38:782-794.

Hiniker JJ, Ramsfjord SP (1966) Anterior displacement of the mandible in adult rhesus monkeys. J Prosthet Dent 16:503-512.

Hinton, RJ (1981) Temporomandibular joint size adaptations in prehistoric Tennessee Indians. Tenn Anthrop 6:89-111.

Hinton RJ, Carlson DS (1979) Temporal change in human temporomandibular joint size and shape. Am J Phys Anthrop 50:325-334.

Hixon EH, Maschka PJ, Fleming PT (1962) Occlusal status, caries, and mastication. J Dent Res 41:514-524.

Hooton EA (1946) The evolution and devolution of the human face. Am J Orthodont 32:657.

Horowitz SL, Hixon EH (eds) (1966) The Nature of Orthodontic Diagnosis. St Louis: Mosby.

Horowitz S, Osborne R (1971) The genetic aspects of cranio-facial growth. In Moyers RE, Krogman WM (eds), Cranio-Facial Growth in Man. Oxford: Pergamon, pp. 183-192.

Howe RP, McNamara JA, O'Connor KA (1983) An examination of dental crowding and its relationship to tooth size and arch dimension. Am J Orthodont 83:363-373.

Hunt EE (1959) The continuing evolution of modern man. Cold Spr Harb Symp Quant Biol 24:245-254.

Hunt EE (1961) Malocclusion and civilisation. Am J Orthodont 47:406-422.

Hunt EE (1971) Pathfinder paper: genetics. In Moyers RR, Krogman WM (eds), Cranio-Facial Growth in Man. Oxford: Pergamon. pp. 343-350.

Hunter WS (1977) The dynamics of mandibular arch perimeter change from mixed to permanent dentitions. In McNamara J (ed), The Biology of Occlusal Development. Monograph 7, Craniofacial Growth Series, Ann Arbor: Center for Human Growth and Development, pp. 169-178.

Hylander WL (1977) In vivo bone strain in the mandible of Galago crassicaudatus. Am J Phys Anthrop 46:309-326.

Hylander WL (1979) The functional significance of primate mandibular form. J

Morphol 159:253-295.
Infante PF (1976) An epidemiologic study of finger habits in preschool children, as related to malocclusion, socioeconomic status, race, sex, and size of community. J Dent Child 43:33-38.
Ingervall B, Helkimo E (1978) Masticatory muscle force and facial morphology in man. Arch Oral Biol 23:203-206.
Irie M, Dahlberg AA (1967) Occlusal relationships of American Indian (Pima) dentition. IADR 45th meeting, Washington.
Isaacson RJ, Christiansen RL, Evans CA, Riedel RA (1975) Research on variation in dental occlusion. Am J Orthodont 68:241-255.
Iwagaki H (1938) Hereditary influence of malocclusion. Am J Orthodont 24:328-336.
Jacobs RM (1966) Cephalometrics and electrodynamic study of the occlusal complex in twins. Am J Orthodont 52:652-668.
Jago JD (1974) The epidemiology of dental occlusion: a critical appraisal. J Pub Health Dent 34:80-93.
Janerich DT, Carlos JP (1968) Birth characteristics and malocclusion. Pediatrics 42:270-275.
Janzer O (1927) Die Zahne der Neu-Pommern. Vierteljahrsschr Zahnhlk 43:289-319.
Johnston L (1976) The functional matrix hypothesis: reflections in a jaundiced eye. In McNamara JA (ed), Factors Affecting the Growth of the Midface. Monograph 6, Center for Human Growth and Development, Ann Arbor. pp. 131-168.
Kang KW, Corey LA, Evans MM, Christian JC, Norton JA (1977) Dominance and environmental variances, their effects on heritabilities estimated from twin data. Hum Hered 27:9-21.
Kang KW, Christian JC, Norton JA (1978) Heritability estimates from twin studies. 1. Formulae of heritability estimates. Acta Genet Med Gemellol 27:39-44.
Katz RV (1977) Relationships of occlusal states with dental caries and periodontal disease. J Dent Res 56:A43.
Katz S, McDonald JL, Stookey GK (1976) Preventive Dentistry in Action (2nd ed). Upper Montclair: D.C.P.
Kaul SS (1975) Estimation of age from the emergence of permanent teeth. Ind Ped 13:233-235.
Kaul SS, Corruccini RS (1990) Dental arch length reduction through interproximal attrition in modern Australian Aborigines. J Hum Ecol spec. issue 2:195-199.
Kaul SS, Corruccini RS (1984) The occlusal epidemiological transition in populations of North India. In Lukacs J (ed) The People of South Asia. New York: Plenum, pp. 201-216.

Kaul SS, Saini S, Saxena B (1975) Emergence of permanent teeth in schoolchildren in Chandigarh, India. Arch Oral Biol 20:587-593.

Kay RF (1975) The functional adaptations of primate molar teeth. Am J Phys Anthrop 43:195-216.

Kay RF, Hiiemae K (1974) Jaw movement and tooth use in recent and fossil primates. Am J Phys Anthrop 40:227-256.

Keil JE, Britt RP, Weinrich MC, Hollis Y, Keil BW (1980) Hypertension in Punjabi females: comparison between migrants to London and natives in India. Hum Biol 52:423-433.

Keith A (1920) Comment: The teeth and jaws of savage man. Trans Brit Soc Stud Orthodont 1916-20:85-86.

Keith A (1924) Concerning certain structural changes which are taking place in our jaws and teeth. In The Growth of the Jaws, Normal and Abnormal, in Health and Disease, London: Dental Board U.K., p. 14.

Keith A (1931) New Discoveries Relating to the Antiquity of Man. London: Williams and Norgate.

Keller JT (1975) A comparison of the refractive status of myopic children and their parents. Am J Optom 50:206-211.

Kelley JS, Harvey CR (1977) An assessment of the occlusion of youths 12-17 years. USPHS Vital and Health Statistics, Ser. 11, No. 162.

Kempthorne O, Osborne RH (1961) The interpretation of twin data. Am J Hum Genet 13:320-339.

Kesling PC (1988) Tip-edge Guide. T.P. Laboratories.

Kiliaridis S (1986) Masticatory Muscle Function and Craniofacial Morphology. Swed Dent J Suppl 36.

Kiliaridis S, Engstrom C, Thilander B (1988) Histochemical analysis of masticatory muscle in the growing rat after prolonged alteration in the consistency of the diet. Arch Oral Biol 33:187-193.

King DL (1983) Etiology of malocclusion. Birth Def 19:83-94.

King L, Harris EF, Tolley EA (1993) Heritability of cephalometric and occlusal variables as assessed from siblings with overt malocclusions. Am J Orthodont Dentofac Orthoped 104:121-131.

Klatsky M (1948) Studies in the dietaries of contemporary primitive peoples. J Am Dent Assoc 36:385.

Klatsky M, Fisher RL (1953) The Human Masticatory Apparatus: An Introduction to Dental Anthropology. Brooklyn: Dental Items of Interest.

Kraus BS, Wise WJ, Frei RA (1959) Heredity and the craniofacial complex. Am J Orthodont 45:172-217.

Krogman WM (1967) The role of genetic factors in the human face, jaws and teeth: a review. Eugen Rev 59:165-192.

Krogman WM (1974) Craniofacial growth and development: an appraisal. Yearb

Phys Anthrop 18:31-64.
Lauweryns I, Carels C,Vlietinck R (1993) The use of twins in dentofacial genetic research. Am J Orthodont Dentofac Orthoped 103:33-38.
Lavelle CLB (1968) Anglo-Saxon and modern British teeth. J Dent Res 47:811-815.
Lavelle CLB (1972) Maxillary and mandibular tooth size in different racial groups and in different occlusal categories. Am J Orthodont 61:29-37.
Lavelle CLB (1973) Variation in the secular changes in the teeth and dental arch. Angle Orthodont 43:412-421.
Lee GTR (1977) Ethnic variations in teeth morphology. Proc Brit Paedodont Soc 7:23-27.
Levy M (1982) Teach yourself to see better. Sci Dig Aug:34-35.
Litton SF, Ackerman LN, Isaacson RJ, Shapiro EL (1970) A genetic study of Class III malocclusion. Am J Orthodont 58:565-577.
Lombardi AV (1982) The adaptive value of dental crowding: a consideration of the biologic basis of malocclusion. Am J Orthodont 81:38-42.
Lombardi AV, Bailit HL (1972) Malocclusion in the Kwaio, a Melanesian group on Malaita, Solomon Islands. Am J Phys Anthrop 36:283-294.
Lombardi Pardini EC, Pardini E (1976) Craniometria della Nuova Britannia. Arch Antrop Etnol (Florence) 106:323-371.
Lovius BBJ, Goose DH (1969) The effects of water fluoridation on occlusion in groups of children in Anglesey. Eur Orthodont Soc Trans 45:69-77.
Lu K (1977) Dental condition of two tribes of Taiwan aborigines -- Ami and Atayal. J Dent Res 56:117-126.
Lundstrom A (1923) Malocclusion of the teeth regarded as a problem in connection with the apical base. Sven Tandlak Tidskr 16:147-296.
Lundstrom A (1948) Tooth size and occlusion in twins. Basel: Karger.
Lundstrom A (1952) On the correlation between tooth size and the irregularities of the teeth (crowding, spacing). Arch Orthodont 1:29-33.
Lundstrom A (1955a) The significance of early loss of deciduous teeth in the etiology of malocclusion. Am J Orthodont 41:819-829.
Lundstrom A (1955b) Variation of tooth size in the etiology of malocclusion. Am J Orthodont 41:872-876.
Lundstrom A (1955c) The significance of genetic and nongenetic factors in the profile of the facial skeleton. Am J Orthodont 41:910-916.
Lundstrom A (1960) Aetiology and prevention of malocclusion. In Lundstrom A (ed) Introduction to Orthodontics. New York: McGraw-Hill. pp. 159-187.
Lundstrom A (1984) Nature versus nurture in dento-facial variation. Eur J Orthodont 6:77-91.
Lundstrom A, Lysell L (1953) An anthropological examination of a group of medieval Danish skulls, with particular regard to the jaws and occlusal

conditions. Acta Odontol Scand 11:111-128.
Lundstrom A, McWilliam JS (1987) A comparison of vertical and horizontal cephalometric variables with regard to heritability. Eur J Orthodont 9:104-108.
McCance R, Owens P, Tonge C (1968) Severe undernutrition in growing and adult animals. 18. The effects of rehabilitation on the teeth and jaws of pigs. Brit J Nutr 22:267-268.
McCann M, Torney D, McCann W, Grant T (1966) Occlusal attrition, transpalatal dimensions and the early New Zealand Maori. Am J Phys Anthrop 25:87-89.
Macchiarelli R, Bondioli L (1986) Post-Pleistocene reductions in human dental structure; a reappraisal in terms of increasing population density. Hum Evol 1:405-418.
McGarvey ST, Schendel DE, Baker PT (1980) Modernization effects on familial aggregation of Samoan blood pressure: a preliminary report. Med Anthrop 4:321-338.
McNamara JA (1973) Neuromuscular and skeletal adaptations to altered function in the orofacial region. Am J Orthodont 64(6):578-606.
McNamara JA (ed) (1976) Factors Affecting the Growth of the Midface. Craniofacial Growth Monograph 6, Center for Human Growth and Development, Ann Arbor.
McNamara JA (ed) (1977) The Biology of Occlusal Development. Craniofacial Growth Monograph 7, Center for Human Growth and Development, Ann Arbor.
Macklin MT, Moore SA (1935) An example of a similar type of malocclusion in identical twins. J Hered 26:445-450.
Maddock IH (1963) How hard do we bite? Queen's Med Mag 55:46-47.
Majumder PP, Roy J (1982) Distribution of ABO blood groups on the Indian subcontinent: a cluster-analytic approach. Curr Anthrop 23:539-566.
Manfredi C, Martina R, Grossi GB, Giuliani M (1997) Heritability of 39 orthodontic cephalometric parameters on MZ, DZ twins and MN-paired singletons. Am J Orthodont Dentofac Orthoped 111:44-51.
Mann I (1966) Culture, Race, Climate and Eye Disease: An Introduction to the Study of Geographical Ophthalmology. Springfield: Thomas.
Margetts B, Brown T (1978) Crown diameters of the deciduous teeth in Australian Aboriginals. Am J Phys Anthrop 48:493-502.
Marks MB (1965) Allergy in relation to orofacial dental deformities in children: a review. J Allerg 36:293-302.
Marks MB (1973) Unusual signs of respiratory tract allergy. Ann Allerg 31:611-617.
Marks MB (1977) Recognizing the allergic person. Am Fam Physician 16:72-79.
Masali L (1985) La cura dei denti presso i popoli mesopotamici. In Vogel G,

Gambacorta G (eds), Storia della Odontoiatria. Milan: Ars Medica Antiqua, pp. 47-50.

Masali L, Peluso A (1985) L'odontoiatria nell'antico Egitto. In Vogel G, Gambacorta G (eds), Storia della Odontoiatria. Milan: Ars Medica Antiqua, pp. 51-66.

Melsen B (ed.) (1991) Current Controversies in Orthodontics. Chicago: Quintessence.

Menconi A, Fornaciari G (1985) L'odontoiatria etrusca. In Vogel G, Gambacorta G (eds) Storia della Odontoiatria., Milan: Ars Medica Antiqua, pp. 88-97.

Menezes DM, Foster TD, Lavelle CLB (1974) Genetic influences on dentition and dental arch dimensions. Am J Phys Anthrop 40:213-220.

Merrett TG, Merrett J, Cookson JB (1976) Allergy and parasites: the measurement of total and specific IgE levels in urban and rural communities in Rhodesia. Clin Allerg 6:131-134.

Miethke R-R, Behm-Menthel A (1988) Correlations between lower incisor crowding and lower incisor position and lateral craniofacial morphology. Am J Orthodont Dentofac Orthop 94:231-239.

Mills JRE (1955) Ideal dental occlusion in the primates. Dent Proc 6:47-61.

Mills JRE (1963) Occlusion and malocclusion of the teeth of primates. In Brothwell DR (ed), Dental Anthropology. New York: Macmillan, pp. 29-51.

Mills JRE (1988) Molar morphology and tooth wear. In Russell DE, Santoro J-P, Sigogneau-Russell D (eds) Teeth revisited: proceedings of the VIIth international symposium on dental morphology. Paris: Museum national d'histoire naturelle, pp. 343-350.

Miyamoto W, Chung CS, Yee PK (1976) Effect of premature loss of deciduous canines and molars on malocclusion of the permanent dentition. J Dent Res 55:584-590.

Moggi Cechi J, Pacciani E, Pinto Cisternas J (n.d.) Caratteristiche morfologiche dentarie in alcune popolazioni Toscane dal periodo etrusco ad oggi. Manuscript.

Mohlin B, Sagne S, Thilander B (1978) The frequency of malocclusion and the craniofacial morphology in a medieval population in Southern Sweden. Ossa 5:57-84.

Moore GJ, McNeill RW, D'Anna JA (1972) The effects of digit sucking on facial growth. J Am Dent Assoc 84:592-599.

Moore GR, Hughes BO (1942) Familial factors in dentofacial disturbance. Am J Orthodont 28:603-639.

Moore WJ (1965) Masticatory function and skull growth. J Zool 146:123-131.

Moore WJ (1981) Facial growth in primates with special reference to the hominoidea. Symp Zool Soc Lond 46:37-62.

Moorrees CFA, Burstone CJ, Christiansen RL, Hixon EH, Weinstein S (1971)

Research related to malocclusion. Am J Orthodont 59:1-18.
Moorrees CFA, Chadha JM (1962) Crown diameters of corresponding tooth groups in the deciduous and permanent dentition. J Dent Res 41:466-470.
Moorrees CFA, Reed RB (1954) Biometrics of crowding and spacing of the teeth in the mandible. Am J Phys Anthrop 12:77-88.
Moorrees CFA, Reed R (1964) Correlations among crown diameters of human teeth. Arch Oral Biol 9:685-697.
Morgan RW, Munro M (1973) Refractive problems in northern natives. Canad J Ophthal 8:226-228.
Morris ML (1966) Orthodontic-periodontic relationships. In Horowitz SL, Hixon EH (eds), The Nature of Orthodontic Diagnosis. St Louis: Mosby. pp. 192-216.
Moss ML (1997) The functional matrix hypothesis revisited. 4. The epigenetic antithesis and the resolving synthesis. Am J Orthodont Dentofac Orthoped 112:410-417.
Moyers RE (1949) Temporomandibular muscle contraction patterns in Angle Class II, Division 1 malocclusions: an electromyographic analysis. Am J Orthodont 35:837-857.
Moyers RE (1988) Handbook of Orthodontics. 4th Ed. Chicago: Year Book.
Moyers RE, Wainright RL (1977) Skeletal contributions to occlusal development. In McNamara J (ed) The biology of occlusal development. Monograph 7, Craniofacial Growth Series, Ann Arbor: Center for Human Growth and Development, pp. 89-111.
Mucci RJ (1982) The Role of Attrition in the Etiology of Third Molar Impactions: Confirming the Begg Hypothesis. Thesis, University of Illinois at Chicago Circle.
Mueller WH (1977) Parent-child correlations of stature and weight among school aged children: a review of 24 studies. Hum Biol 48:379-397.
Mueller WH, Pollitt, E (1982) The Bacon Chow study: effects of nutrition supplementation on sibling-sibling anthropometric correlations. Hum Biol 54:455-468.
Murphy TR (1964) Reduction of the dental arch by approximal attrition. A quantitative assessment. Brit Dent J 116:483-488.
Murray J (1957) Prevalence of malocclusion in fifteen-year-old children from fluoride and non-fluoride communities. Brit Soc Stud Orthodont Trans 55:55-60.
Nakata M, Yu PL, Nance WE (1974) Multivariate analysis of craniofacial measurements in twin and family data. Am J Phys Anthrop 41:423-430.
Nanda RS, Khan I, Anand R (1972) Effect of oral habits on the occlusion in preschool children. J Dent Child 39:449-452.
Nanda SK (1983) The Developmental Basis of Occlusion and Malocclusion.

Chicago: Quintessence.
National Center for Health Statistics (NCHS) (1967) Orthodontic treatment priority index (by Grainger RM). Vital and Health Statistics, Ser. 2, No. 25, USPHS, Washington, D.C.
Neel JV (1963) Diabetes mellitus: a "thrifty" genotype rendered detrimental by progress? Am J Genet 14:353-362.
Nelson GC (1997) Occlusal variation in modern India (abstract). Am J Phys Anthropol suppl. 24:177.
Nelson MM (1969) A brief study of the genetics of malocclusion. Trans Eur Orthodont Soc 45:55-68.
Neppi Modona A (1959) Scientists' contributions to etruscology. In Wolstenholme GEW, O'Connor E (eds), Medical Biology and Etruscan Origins (CIBA Foundation Symposium). London: Churchill, pp. 64-74.
Niswander JD (1967) Further studies on the Xavante Indians. VII. The oral status of the Xavantes of Simoes Lopes. Am J Hum Genet 19:543-553.
Niswander JD (1975) Genetics of common dental disorders. Dent Clin N Am 19:197-206.
Nye ER (1966) Natural selection and degenerative cardiovascular disease. Eugen Quart 14:127-131.
O'Dea K, Spargo RM, Akerman K (1980) Some studies on the relationship between urban living and diabetes in a group of Australian aborigines. Med Anthrop 4:1-20.
Omran AR (1971) The epidemiologic transition. Milbank Mem Quart 49:509-538.
Oppenheimer AM (1966) Tool use and crowded teeth in Australopithecinae. Curr Anthrop 5:419-421.
O'Ryan FS, Gallagher DM, LaBanc JP, Epker BN (1982) The relation between naso-respiratory function and dentofacial morphology: a review. Am J Orthodont 82:403-410.
Pacciani E, Corruccini RS (1986) Studio epidemiologico comparativo sull'occlusione dentale. Antropol Contemp 9:57-64.
Parker WS (1998) The HLD (CalMod) index and the index question. Am J Orthodont Dentofac Orthoped 114:134-141.
Parkinson R (1907) Dreissig Jahre in der Sudsee. Stuttgart.
Paunio K (1973) The role of malocclusion and crowding in the development of periodontal disease. Int Dent J 23:470-475.
Peck S, Peck H (1975) Orthodontic aspects of dental anthropology. Angle Orthodont 45:95-102.
Peckham CS, Gardiner PA, Goldstein H (1977) Acquired myopia in 11-year-old children. Brit Med J 1(6060):542-545.
Pelton WJ, Elsasser WA (1953) Studies of dentofacial morphology. III. The role

of dental caries in the etiology of malocclusion. J Am Dent Assoc 46:648-657.
Pereira CB, Evans H (1975) Occlusion and attrition of the primitive Yanomami Indians of Brazil. Dent Clin N Am 19:485-498.
Petrovic A, Stutzmann J (1977) Further investigations into the functioning of the "comparator" of the servosystem in the control of the condylar cartilage growth rate and of the lengthening of the jaw. In McNamara J (ed) The Biology of Occlusal Development, Monograph 7. Ann Arbor: Craniofacial Growth Series, Center for Human Growth and Development, pp. 255-291.
Phipps GS, German RZ, Smith RJ (1988) Comparative craniofacial variation in Navajo Indians and North American caucasians. Am J Phys Anthrop 76:145-154.
Potter RHY, Corruccini RS, Green LJ (1981) Variance of occlusion traits in twins. J Craniofac Genet Dev Biol 1:217-227.
Potter RH, Nance WE (1976) A twin study of dental dimension. 1. Discordance, asymmetry and mirror imagery. Am J Phys Anthrop 44:391-395.
Potter RHY, Rice JP, Dahlberg AA, Dahlberg T (1983) Dental size traits within families: path analysis for first molar and lateral incisor. Am J Phys Anthrop 61:283-289.
Potter RHY, Yu PL, Christian JC (1979) Association of twin zygosity with the mean and variance of tooth size. Acta Genet Med Gemellol 28:211-223.
Potter RHY, Yu PL, Dahlberg AA, Merritt AD, Conneally PM (1968) Genetic studies of tooth size factors in Pima Indian families. Am J Hum Gen 20:89-100.
Prabhakara Rao BSRC (1985) Historical background of evolution and growth of dentistry. In Rami Reddy V (ed) Dental Anthropology: Application and Methods. New Delhi: Inter-India, pp. 369-371.
Price WA (1935) Studies of relationships between nutritional deficiencies and (a) facial and dental arch deformities and (b) loss of immunity to dental caries among South Sea Islanders and Florida Indians. Dent Cosmos 77:1033-1045.
Price WA (1936) Eskimo and Indian field studies in Alaska and Canada. J Am Dent Assoc 23:417-437.
Price WA (1939) Nutrition and Physical Degeneration. New York: Paul B Hoeber.
Prior IAM (1971) The price of civilization. Nutr Today 6:2-11.
Proffit WR (1975) Muscle pressures and tooth position: North American whites and Australian aborigines. Angle Orthodont 45:1-11.
Proffit WR (1986a) Contemporary Orthodontics. St Louis: Mosby.
Proffit WR (1986b) On the aetiology of malocclusion. Brit J Orthodont 13:1-11.
Proffit WR, Fields HW (1983) Occlusal forces in normal and long-face children. J Dent Res 62:571-574.
Proffit WR, Fields HW, Nixon WL (1982) Evaluation of vertical occlusal forces and the normal and long face adults (abstract). J Dent Res 61:342.

Proffit WR, McGlone RE, Barrett MJ (1975) Lip and tongue pressure related to dental arch and oral cavity size in Australian Aborigines. J Dent Res 54:1161-1172.

Radnzic D (1988) Dental crowding and its relationship to mesiodistal crown diameters and arch dimensions. Am J Orthodont Dentofac Orthop 94:50-56.

Ramfjord SP, Ash MM (1971) Occlusion. 2nd Ed. Philadelphia: Saunders.

Reed T, Sprague FR, Kang KW, Nance WE, Christian JE (1975) Genetic analysis of dermatoglyphic patterns in twins. Hum Hered 25:263-275.

Richards ND, Barmes DE (1971) Social factors in dental epidemiology. In Richards ND, Cohen LK (eds) Social Sciences and Dentistry: A Critical Bibliography. The Hague: A. Sitjhoff, pp. 308-346.

Riesenfeld A (1970) On some "racial" features in rats, dogs and men. Homo 21:163-175.

Robinson D (1977) Letter to the editor. Brit Med J 1(6060):976.

Rubbrecht O (1939) A study of the heredity of the anomalies of the jaws. Am J Orthodont 25:751-779.

Russell M (1977) Parent-child and sibling-sibling correlations of height and weight in a rural Guatemalan population of preschool children. Hum Biol 48:501-515.

Ryder JA (1878) On the mechanical genesis of tooth forms. Proc Acad Nat Sci Phila 30 (Ser. 3):45-80.

Sain DR (1982) A cephalometric characterization of orthodontic patients who mouthbreathe (abstract). Am J Orthodont 82:353.

Salzmann JA (1974) Orthodontics in Daily Practice. Philadelphia: Lippincott.

Salzmann JA (1979) The validity of malocclusion measurement. Am J Orthodont 74:464-465.

Sampson WJ, Richards LC (1985) Prediction of mandibular incisor and canine crowding changes in the mixed dentition. Am J Orthodont 88:47-63.

Sanin C, Savara BS (1972) The development of an excellent occlusion. Am J Orthodont 61:345-352.

Sassouni V (1971) Orthodontics in Dental Practice. St Louis: Mosby.

Saxena B (1972) Eruption of permanent teeth, state of dental health and body size of school-going girls in Chandigarh. Thesis, Panjab University, Chandigarh.

Schoubou K, Hauge M (1973) The application of twin methods in orthodontic research. Scand J Dent Res 81:563-566.

Schultz AH (1972) Developmental abnormalities. Pathol Sim Primat 1:158-189.

Seth S (1968) A study of the A_1A_2BO blood groups system and ABO(H) secretion in six endogamous groups of Punjab. Am J Phys Anthrop 29:387-396.

Seth S, Bansal IJS, Seth PK (1971) Studies on haptoglobin and transferrin types in Khatris and Aroras of Punjab, India. Humangenetik 14:44-69.

Shapiro BL (1969) A twin study of palatal dimensions partitioning genetic and

environmental contributions to variability. Angle Orthodont 39:139-151.
Sharma JC, Kaul V (1977) Dental morphology and odontometry in Punjabis. J Ind Anthrop Soc 12:213-226.
Sharma K (1982) Genetic Contribution to Body Morphology. A Study Based on Human Twins and Pedigree Data. Ph.D. Thesis, Panjab University, Chandigarh.
Sharma K, Byard PJ, Russell JM, Rao DC (1984) A family study of anthropometric traits in a Punjabi community: I. Introduction and familial correlations. Am J Phys Anthrop 63:389-395.
Sharma K, Corruccini R (1986) Genetic basis of dental occlusal variations in northwest Indian twins. Europ J Orthodont 8:91-97.
Sicher H (1953) The biology of attrition. Oral Surg 6:406-412.
Sim Wallace J (1904) Essay on the Irregularities of the Teeth, with Special Reference to a Theory of Causation and the Principles of Prevention and Treatment. London: Dental Manufacturing Co.
Singh S (1966) Sikligars of Punjab. New Delhi: Sturling.
Singh S, Sareen KN, Goedde HW (1974a) Investigation of some biochemical genetic markers in four endogamous groups from Punjab (N.W. India). I. Protein and enzyme polymorphisms in serum. Humangenetik 21:361-346.
Singh S, Sareen KN, Goedde HW (1974b) Investigation of some biochemical genetic markers in four endogamous groups in Punjab (N.W. India). II. Red cell enzyme polymorphisms. Humangenetik 22:133-138.
Smith JD, Genoways HH, Jones JK (1977) Cranial and dental anomalies in three species of platyrrhine monkeys from Nicaragua. Folia Primat 28:1-41.
Smith P (1982) Dental reduction. Selection or drift? In Kurten B (ed), Teeth: Form, Function and Evolution. New York: Columbia University Press, pp. 366-379.
Smith RJ (1982) Development of occlusion and malocclusion. Ped Clin N Am 29:475-501.
Smith RJ, Bailit HL (1977) Problems and methods in research on the genetics of dental occlusion. Angle Orthodont 47:65-77.
Smith RJ, Bailit HL (1978) Variation in dental occlusion and arches among Melanesians of Bougainville Island, Papua New Guinea. I. Methods, age changes, sex differences and population comparisons. Am J Phys Anthrop 47:195-208.
Smith RJ, Bailit HL (1979) Prevalence and etiology of asymmetries in occlusion. Angle Orthodont 49:199-204.
Sorsby A (1970) Ophthalmic Genetics. 2nd Ed. New York: Appleton-Century-Crofts.
Sorsby A, Benjamin B, Davey JB, Sheridan M, Tanner JM (1957) Emmetropia and its aberrations. Med Res Counc Spec Rpt 293, London:HMSO.
Staley RN, Stuntz WR, Peterson LC (1985) A comparison of arch widths in

adults with normal occlusion and adults with Class II, Division 1 malocclusion. Am J Orthodont 88:163-169.

Stein KF, Kelley TJ, Wood E (1956) Influence of heredity in the etiology of malocclusion. Am J Orthod 42:125-141.

Strnad J (1978) The cause and effect of mouth-breathing as related to malocclusion. Rhinology 16:191-196.

Sunderland E, Sawhney KS, Cartwright RA, Jolly JG (1976) Studies of haptoglobin and transferrin types in four castes of the Panjab, Northern India. Hum Hered 26:16-24.

Sushma S (1972) Order and eruption times of permanent teeth, state of dental health, height and weight of school-going boys in Chandigarh. Thesis, Chandigarh: Panjab University.

Swindler DR (1976) Dentition of Living Primates. New York: Academic Press.

Tanwer S (1977) Nutrition and Physical Characters of Underprivileged Children in Chandigarh with special reference to Genetic-Environmental Interaction. Thesis, Chandigarh: Panjab University.

Tewari A (1966) Relationship of the incidence of malocclusion with the socio-economic status of people. J Ind Dent Assoc 38:192-207.

Thoma KH (1938) Principal factors controlling development of mandible and maxilla. Am J Orthodont 24:171-179.

Thurow RC (1977) Atlas of Orthodontic Principles. 2nd Ed. St Louis: Mosby.

Tonge C, McCance R (1965) Severe undernutrition in growing and adult animals. 15. The mouth, jaws and teeth of pigs. Brit J Nutr 19:361-372.

Tonge C, McCance R (1973) Normal development of the jaws and teeth in pigs, and the delay and malocclusion produced be calorie deficiencies. J Anat 115:1-22.

Townsend GC, Brown T (1979) Tooth size characteristics of Australian Aborigines. Occ Pap Hum Biol (Austral Inst Aboriginal Studs, Canberra) 1:17-38.

Townsend GC, Brown T (1979) Tooth size characteristics of Australian Aborigines. Occ. Pap. Hum. Biol. (Austral. Inst. Aboriginal Stud., Canberra) 1:17-38.

Townsend GC, Corruccini RS, Richards LC, Brown T (1988) Genetic and environmental determinants of dental occlusal variation in South Australian twins. Austral Orthodont J 10:231-235.

Trowell HC (1975) Pathological growth and maturation in infants and children associated with modern methods of feeding. J Trop Ped 21:192-198.

Trowell HC, Burkitt DP (eds) (1981) Western Diseases: Their Emergence and Prevention. Cambridge: Harvard.

Tully WJ (1973) Prevention of malocclusion and dentofacial anomalies. Int Dent J 23:481-488.

Ulgen M, Baran S, Kaya H, Karadede I (1997) The influence of the masticatory hypofunction on the craniofacial growth and development in rats. Am J Orthodont Dentofac Orthoped 111:189-198.

Van Der Linden FPGM, Boersma H (1987) Diagnosis and Treatment Planning in Dentofacial Orthopedics. London: Quintessence.

Van Reenen JF (1966) Dental features of a low-caries primitive population. J Dent Res 45:703-713.

Vargervik K (1979) Morphologic evidence of muscle influence on dental arch width. Am J Orthodont 76:21-28.

Vig KWL (1998) Nasal obstruction and facial growth: The strength of evidence fpr clinical assumptions. Am J Orthodont Dentofac Orthoped 113:603-611.

Vig PS (1979) Respiratory mode and morphological types: some thoughts and preliminary conclusions. In McNamara J (ed), Nasorespiratory Function and Craniofacial Growth. Craniofacial Growth Series 9:233-250.

Vig PS, Sarver DM, Hall DJ, Warren DW (1981) Quantitative evaluation of nasal airflow in relation to facial morphology. Am J Orthodont 79:263-272.

Waltz KJ, Albino JE, Lewis RA, Slakter MJ (1980) Measurement considerations in use of the treatment priority index. J Dent Res 59:441.

Ward R, Prior I (1980) Genetic and sociocultural factors in the response of blood pressure to migration of the Tokelauan population. Med Anthrop 4:339-366.

Watnick SS (1972) The inheritance of craniofacial morphology. Angle Orthodont 42:339-351.

Watt DG, Williams HM (1951) The effects of the physical consistency of food on the growth and development of the mandible and maxilla of the rat. Am J Orthodont 37:895-928.

Waugh LM (1937a) Dental observations among the Eskimos. J Dent Res 16:355-356.

Waugh LM (1937b) Influence of the diet on the jaws and the face of the American Eskimo. J Am Dent Assoc 24:1640-1647.

Weijs WA, Hillen B (1986) Correlations between the cross-sectional area of the jaw muscles and craniofacial size and shape. Am J Phys Anthrop 70:423-431.

Weijs W, Dantuma R (1975) Electromyography and mechanics of mastication in the albino rat. J Morph 146:1-34.

Weinberger BW (1926) Orthodontics: An Historical Review of its Origin and Evolution. Vol. I. St Louis: Mosby.

Wheeler RC (1974) Dental Anatomy, Physiology and Occlusion. Philadelphia: Saunders.

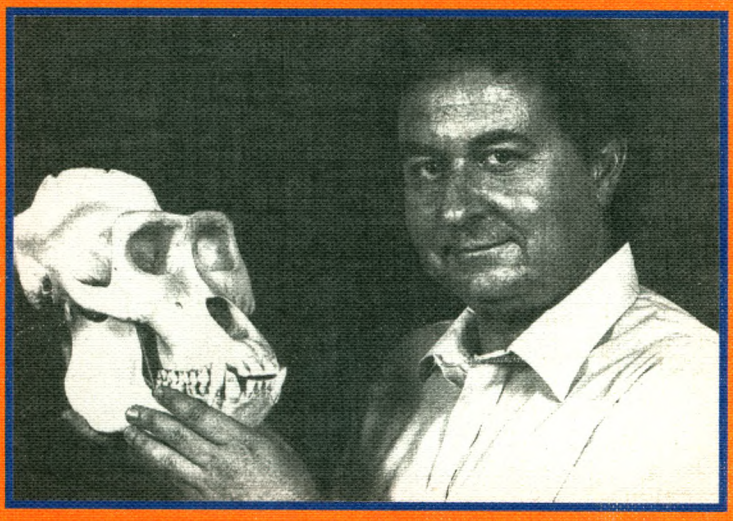
Whitley LD (1979) Labor and religion in a rural Baptist community. Manuscript, Southern Illinois University.

Whitley LD (1985) Beyond Decision Making: Rational and Nonrational information processes in a Baptist Farming Community. Dissertation,

Carbondale: Southern Illinois University.
WHO (World Health Organization) (1971) Oral Health Surveys -- Basic Methods. Geneva.
Wiener G, Purser AF (1957) The influence of four levels of feeding on the position and eruption of incisor teeth in sheep. J Agric Sci 49:51-55.
Williams C (1943) Investigations concerning the dentitions of the Eskimos of Canada's Eastern Arctic. J Periodont 14:34.
Wolpoff MH (1969) The effect of mutations under conditions of reduced selection. Soc Biol 16:11-23.
Wolpoff MH (1971) Interstitial wear. Am J Phys Anthrop 34:205-228.
Wood BF (1971) Malocclusion in the modern Alaskan Eskimo. Am J Orthodont 60:344-354.
Wood Jones F (1926) Arboreal Man. New York: Hafner.
Woolley V, Gill PS, Sunderland E (1983) Blood groups, haptoglobins and red cell isoenzymes of the Jat Sikhs of Ludhiana District, Panjab, India. Hum Hered 33:44-51.
Yamada T, Tanne K, Miyamoto K, Yamauchi K (1997) Influences of nasal respiratory obstruction on craniofacial growth in young Macaca fuscata monkeys. Am J Orthodont Dentofac Orthoped 111:38-43.
Young FA (1961) The effect of restricted visual space on the primate eye. Am J Ophthal 52:799-806.
Young FA, Beattie RJ, Newby FJ, Swindal MT (1954) The Pullman study: a visual survey of Pullman school children. Am J Optom 31:111-112.
Young FA, Leary GA, Baldwin WR, West DC, Box RA, Harris E, Johnson C (1969) The transmission of refractive errors within Eskimo families. Am J Optom 49:676-685.
Zingeser MR (1973) Occlusofacial morphological integration. Symp IV Int Cong Primat 3:241-257.

Name Index

Name Index



Robert S. Corruccini received his B. A. in Anthropology (summa cum laude) in 1971 from the University of Colorado, Boulder. His Ph. D. in Physical Anthropology came in 1975 from the University of California, Berkeley. He was in several roles at the U. S. National Museum of Natural History between 1970-1979 including Smithsonian Institution Research Fellow, 1976-1979. Becoming full Professor of Anthropology at Southern Illinois University, Carbondale in 1986, he was the SIU Outstanding Scholar for 1994. Dr. Corruccini has published seven books and several hundred journal articles over the last 20 years intensively treating the varying interpretations of the increasingly crooked state of human teeth.