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Dynamic Physiologic Changes in Lumbar CSF Volume Quantitatively Measured By Three-Dimensional Fast Spin-Echo MRI

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Study Design. Lumbar MRI of normal adults. Image analysis to measure lumbar CSF volumes at rest and during physiologic maneuvers.

Objectives. 1) Validate an MRI technique to measure CSF volumes, 2) use this technique to measure the resting volume of lumbar CSF, 3) measure changes in CSF volume with physiologic maneuvers, and 4) demonstrate the anatomic basis for these volume changes.

Summary of Background Data. Studies using radiograph and radionuclide myelography in dogs and humans in the 1960s–1980s qualitatively showed decreases in spinal CSF volume with physiologic maneuvers. Theories were proposed to explain these changes, but they could not be confirmed because only the contrast-laden CSF was visualized using these techniques.

Methods. Four adult volunteers had lumbar MRI using a fat-saturated T2-weighted three-dimensional fast spin-echo sequence. Quantitative analysis of images was used to measure lumbar CSF volume; the technique was validated using a water phantom. Lumbar CSF volume was measured 1) at rest, 2) with hyperventilation, 3) with abdominal compression, and 4) with both hyperventilation and abdominal compression.

Results. Resting lumbar CSF volume ranged from 28 to 42 mL. Reversible changes in lumbar CSF volume resulting from physiologic maneuvers are visualized by MR myelography and measured. The volume change (10% reduction in volume with hyperventilation, 28% with compression, and 41% with combined hyperventilation and abdominal compression) is directly visualized to be caused by engorgement of the epidural venous plexus, compressing the thecal sac.

Conclusions. MRI provides a noninvasive means to measure spinal CSF volume and demonstrates the anatomic basis of physiologic volume changes. This has important implications for spinal anesthesia. [Key words: cerebrospinal fluid, flow dynamics, three-dimensional MRI, MRI volume measurement, myelography, spinal canal] **Spine 2001;26:1172–1178**

Considering the important role that spinal fluid volume plays in the daily activities of physicians engaged in clinical practice and neurologic research, it would be useful to measure this volume and its changes in an accurate and safe manner. However, as recently as 1992, the eminent neurologist Dr. Robert Fishman made the following observation[8]: "Despite the importance of the CSF volume in understanding intracranial compliance, the pressure-volume curve, and the changes in both that occur in disease, there is a paucity of such data because of the difficulty inherent in measuring CSF volume. The generalization that CSF volume is normally about 150 mL is based on old data obtained by CSF drainage in patients undergoing pneumoencephalography or at autopsy. In fact, there are no reliable data regarding this very point."

We describe a technique using magnetic resonance imaging (MRI) to quantitatively measure the volume of cerebrospinal fluid (CSF) in the spinal subarachnoid space from T11 through S2, and directly demonstrate dynamic changes in spinal CSF volume with the physiologic maneuvers of hyperventilation and abdominal compression, as well as the anatomic basis for these volume changes. This MRI technique is noninvasive, safe, free from ionizing radiation, and relatively simple.

Methods

Validation of Quantitation. Before human volunteers were scanned, a water phantom was constructed to simulate the spinal canal and to validate the accuracy of the volume measurement technique. A long ⅜-inch-diameter (0.95 cm) Teflon rod (simulating the spinal cord) was placed longitudinally inside a long piece of ⅝-inch inner diameter (1.6 cm) Tygon tubing (simulating the thecal sac), which was stoppered at one end. The length of this slightly curved tubing/rod coaxial assembly was approximately 61.5 cm. Water (simulating CSF) was carefully poured into the tubing; 74 mL was needed to fill the tubing around the central rod completely. The top of the assembly was then sealed with waterproof silicone cement.

The phantom was placed in the MRI unit and was scanned using the same technique described below for the human subjects, except that fat saturation was not used. The optimum uniformity of the scanner magnetic field is in approximately the central 48 cm of the bore, so that two overlapping longitudinal three-dimensional fast spin-echo (3D FSE) passes were needed to cover the entire 62-cm phantom, one with 48 cm field of view (FOV) and 512 × 512 matrix, and another with 26 cm FOV and 256 × 256 matrix, with the phantom being moved longitudinally between the two sequences so that each segment was roughly centered in the bore. A small 3-mm plastic bead containing water was taped to the outside of the phantom about one third of the way from the bottom and was included in the field of view of both scans, as a marker of absolute position for the volume calculation. Volume calculations were then performed as described below and yielded a calculated water volume of 71 mL, in good agreement with the actual volume of 74 mL (an error of 4.1%).

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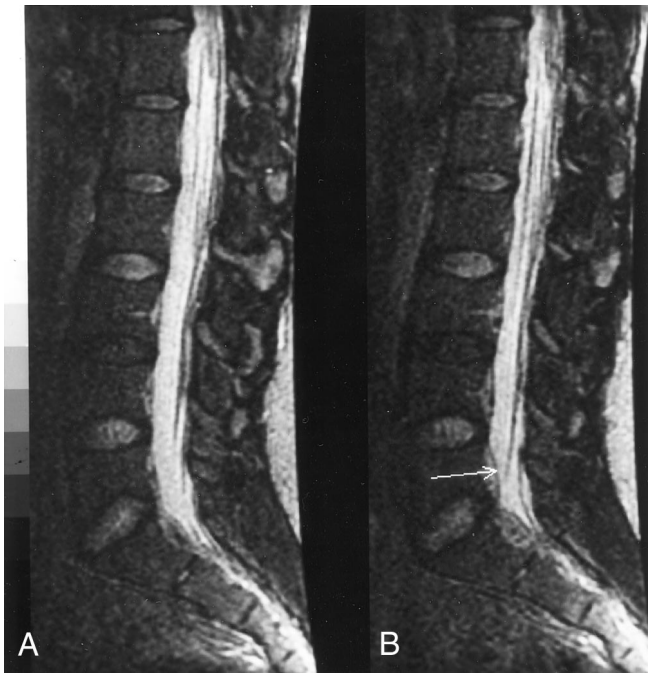

Figure 1. Representative 1-mm sagittal fat-saturated T2-weighted partition images of case 3. **A,** At rest. **B,** With abdominal compression and mild hyperventilation. Note the markedly engorged epidural venous plexus at all vertebral levels in **B**, indicated, for example, at L5 (arrow), deforming the thecal sac and causing decreased lumbar CSF volume.

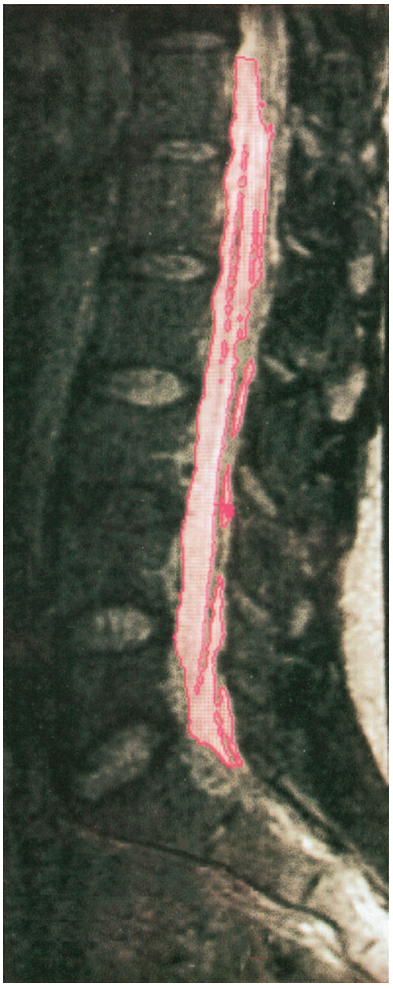

Figure 2. Segmentation of the slice illustrated in Figure 1b, systematically determined as described in the text. The dark red line delineates the border of the selected area, from T11–T12 down to the termination of the thecal sac at S2. The slice volume is computed by multiplying the selected area by the slice thickness (1 mm), and the total lumbar CSF volume is computed by summing the volumes of all the CSF-containing slices.

The study protocol was approved by the Johns Hopkins Joint Committee on Clinical Investigation. Four healthy adult volunteers (male age 32 years, females ages 26, 33, and 41 years) had lumbar spine MRI scans on a 1.5-T scanner (GE Signa 5.3, General Electric Medical Systems, Milwaukee, WI), using a fat-saturated 3D FSE sequence, acquiring 48 contiguous 1-mm T2-weighted partitions in the sagittal plane (TR 3500 msec, TE [effective] 100 msec, echo train length 32, FOV 28 cm, 256 × 256 matrix; phased-array spinal coil; imaging time 5 minutes; nominal voxel size 1.0 mm × 1.1 mm × 1.1 mm = 1.2 mm^3) (Figure 1).

The technique used is similar to that described by Krudy[14] and El Gammal et al[7] but has improved slice spatial resolution (1-mm sagittal partitions) because of the use of 3D FSE rather than the standard two-dimensional FSE, which is currently limited to 3- or 5-mm-thick slices. Nearly isotropic 1-mm^3 voxels are obtained, improving the accuracy of volume measurements. 3D MR myelography using a gradient-echo steady-state sequence was described by VanDyke et al,[21] but that technique employed 2-mm-thick slices, and more importantly had the problem of visualizing only the CSF space and not other structures. The current fat-saturated 3D FSE technique retains the contrast sensitivity to discriminate epidural structures, and in particular is able to explicitly visualize the epidural venous plexus and discs, while maintaining good delineation between intrathecal CSF and contiguous structures.

The lumbar region was imaged with the volunteers in the following conditions: 1) at rest, 2) with mild hyperventilation (coaching the volunteers to breathe deeply, with normal respiratory rate), 3) with firm abdominal compression by an elastic girdle compressing a piece of bath soap under the right costoxiphoid angle, and 4) with a combination of firm abdominal compression and hyperventilation. The order of the maneuvers was arbitrarily varied. The maneuver of mechanical abdominal compression was chosen to simulate vena caval obstruction by an abdominal mass. Resting images were repeated on two of the subjects weeks later, and the resting CSF volumes were calculated again. FOV extended from the distal termination of the thecal sac at S2, up to the T11–T12 interspace. In one subject the occlusion of the inferior vena cava (IVC) resulting from firm abdominal compression was directly imaged by a T1-weighted abdominal MRI sequence obtained during the imaging session, without and with abdominal compression.

Images were processed on the ISG Allegro system (ISG Technologies, Toronto, Canada). The fairly heavily T2-weighted fat-saturated sequence resulted in good delineation between bright intrathecal CSF and all other contiguous anatomic structures including the conus and cauda equina and epidural structures, making segmentation straightforward and reproducible (Figure 2). Upper and lower pixel intensity thresholds were visually determined once for each imaging sequence and were then applied by a computer "automatic seed" algorithm to determine the region of interest and its volume for

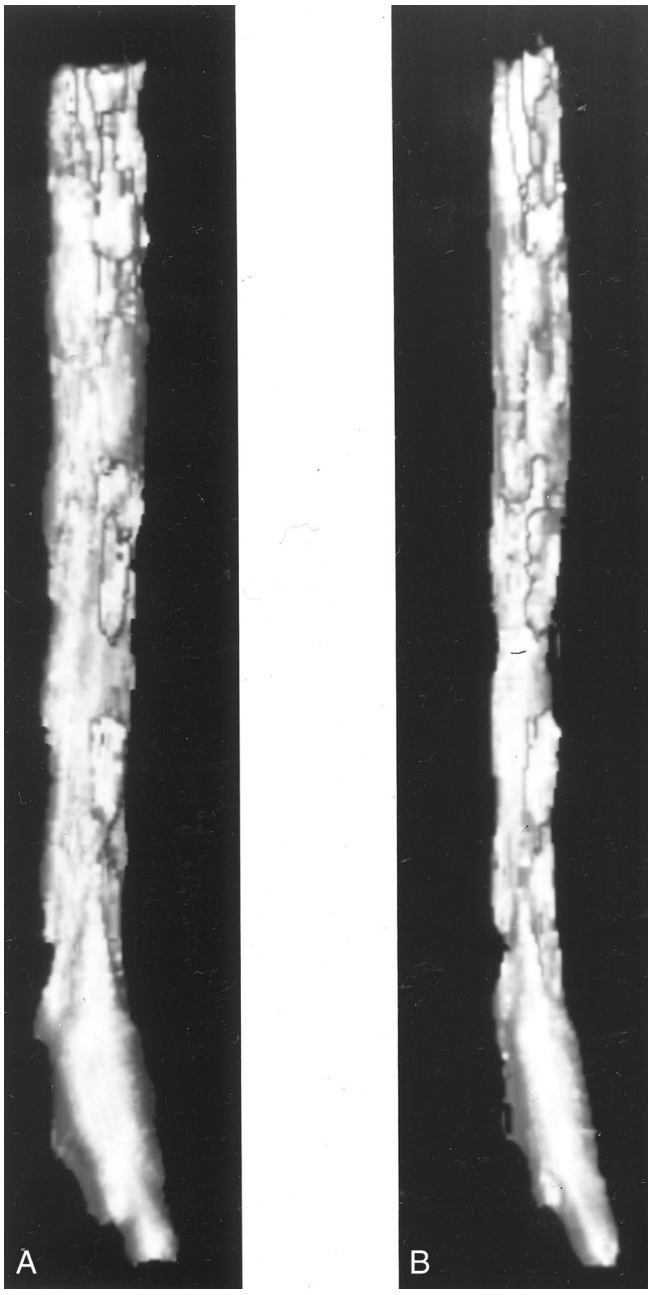

Figure 3. Three-dimensional surface rendering of the lumbar spinal CSF of case 1. **A,** At rest. **B,** With abdominal compression and mild hyperventilation. Note the significant narrowing and reduced volume of the subarachnoid space with abdominal compression and hyperventilation compared with the normal resting configuration.

each partition slice in that sequence. Within about 1 minute, 3D surface rendering of the lumbar intrathecal CSF space was performed (Figure 3), and a total volume was automatically calculated by summing the volumes of individual contiguous 1-mm sagittal partitions.

Some minor operator intervention was occasionally required to delineate small portions of the boundary between the intrathecal CSF and the epidural fat. All measurements were performed by a skilled technologist who has been working full-time for 10 years applying this segmentation program to neuroradiologic images, who was blinded to the physiologic

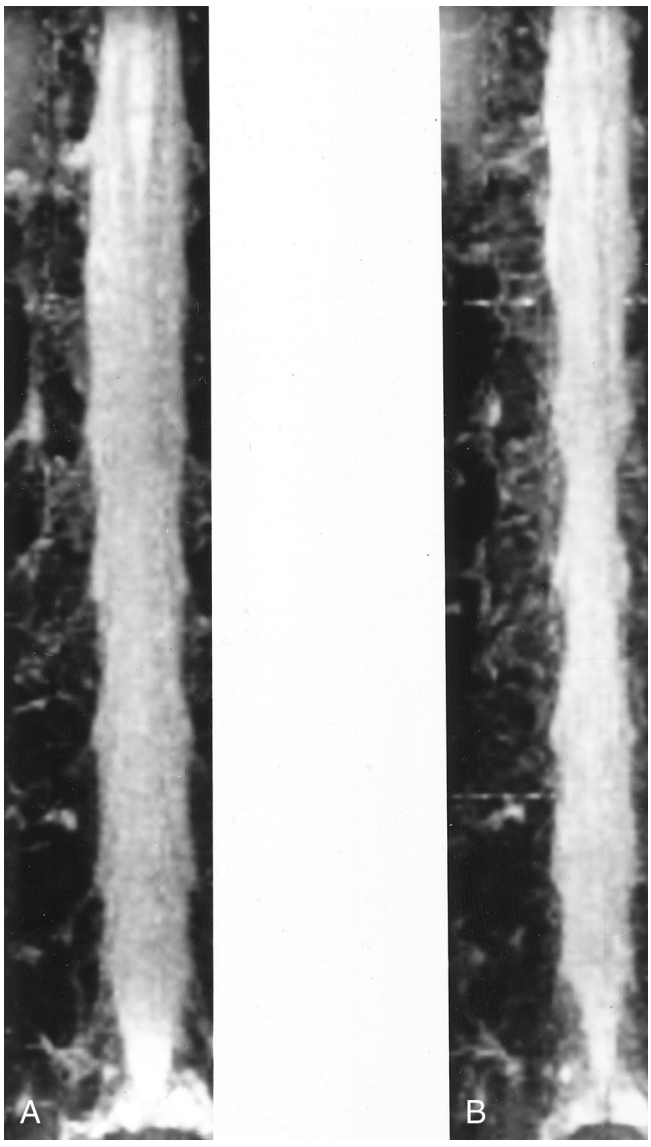

Figure 4. Coronal (AP) view MR myelogram of case 3. **A,** At rest. **B,** With abdominal compression and mild hyperventilation. The significant narrowing and reduced volume of the subarachnoid space with abdominal compression and hyperventilation compared with the normal resting configuration are again demonstrated.

conditions of the test subjects. Final segmentations were checked for accuracy by a board-certified neuroradiologist.

The images were also analyzed on the MRI console workstation, and maximum-intensity-projection composite images of the lumbar CSF were obtained, yielding MR myelograms (Figure 4).

■ Results

The volume of CSF in the thecal sac from T11–T12 to its distal termination ranged from 28 to 42 mL in the resting state for the four subjects. The resting volume of lumbar CSF in a given individual is reproducible, validated by repeat measurements several weeks apart; the calculated standard deviation is less than 1.1 mL.

As shown in Figures 3 and 4, there is a visible decrease in the lumbar CSF volume with combined abdominal

compression and mild hyperventilation compared with the normal resting state (averaging 41%). A similar but slightly smaller change (28%) is observed with abdominal compression alone. With mild hyperventilation there is only a mild decrease in CSF volume (10%). Results are tabulated in Table 1.

The paired *t* test (two-tailed) was used to derive confidence levels and *P* values associated with the differences in CSF volume resulting from the various maneuvers. As shown in Table 1, the differences are statistically significant (95% confidence levels shown for hyperventilation alone [$P < 0.022$] and abdominal compression [$P < 0.011$], and 99% confidence level [$P < 0.0002$] for hyperventilation combined with abdominal compression).

Figure 1 shows that the cause of the decreased lumbar CSF volume with abdominal compression is direct compression of the thecal sac by the engorged epidural venous plexus. The venous engorgement and compression of the thecal sac are reversible, as demonstrated by return to normal thecal resting configuration and volume when the subjects were re-imaged immediately after removal of abdominal compression.

Figure 5 directly confirms that the IVC is markedly compressed during the maneuver of abdominal compression.

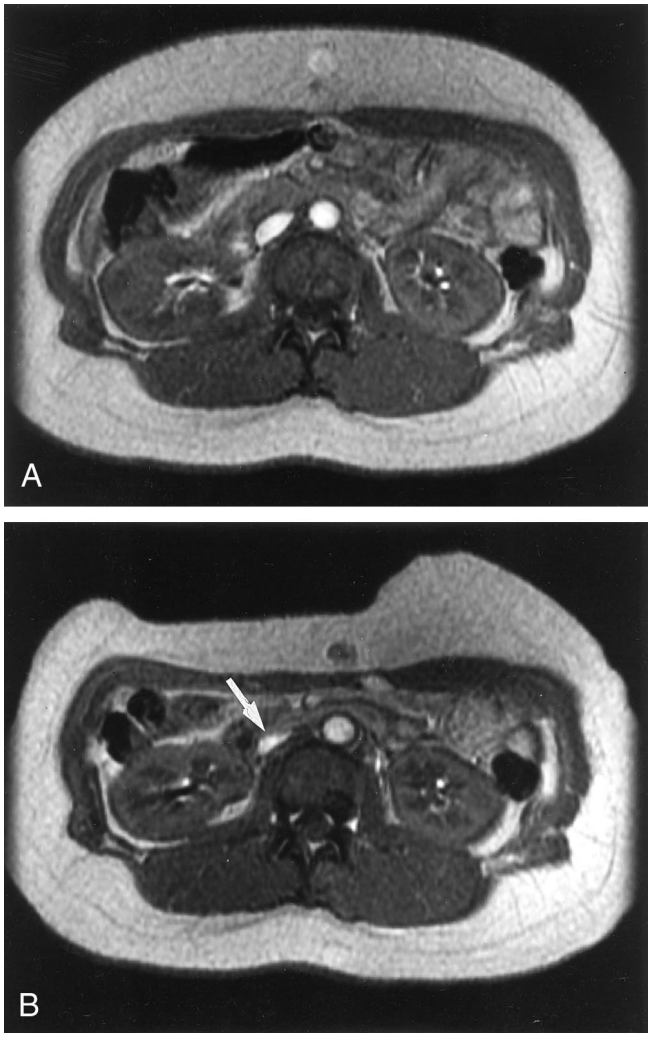

Figure 5. T1-weighted images of the abdomen at the level of the abdominal compression, just below the right costoxiphoid angle. **A,** At rest. **B,** With abdominal compression. The IVC (white arrow) is markedly compressed compared with **A**. Note the deformity–flattening of the anterior subcutaneous tissues and the abdominal musculature by the compression device.

■ Discussion

During the past 50 years, anesthesiologists performing spinal, epidural, or caudal blocks have become increasingly aware of the necessity to reduce by approximately one third the usual amount of local anesthetic drug in patients presenting with evidence of an enlarged abdomen and increased abdominal pressure, such as patients with intestinal obstruction, marked ascites, large abdominal or pelvic tumors, and term pregnancies.[3,4,6] Failure to invoke this dose-adjustment rule has led to excessive spread of local anesthetic solution in the subarachnoid or epidural space, resulting in a high sensory block and occasionally, a high motor block that led to respiratory insufficiency, severe hypotension, and, at times, sudden death.[11,17]

Two of the most important factors contributing to the exaggerated spread of spinal anesthesia have been hypothesized as follows[10,18]: 1) inferior vena caval compression resulting in retrograde engorgement of the lumbar, intervertebral, and epidural veins, causing mechanical compression and deformity of the dural walls, and 2) consequent reduction in the capacity of the subarachnoid space leading to a decrease in spinal fluid volume.[10,18]

Normally, the venous return from the abdomen, pelvis, and lower extremities is *via* the common iliac veins, the inferior vena cava, and its numerous upstream tributaries.[3] A large abdominal tumor mass or term pregnancy compressing the larger vessels leads to venous obstruction and an elevated venous pressure sufficient in magnitude to force open to a wider degree the collateral venous circulation involving the valveless ascending lumbar, azygos, paravertebral, and vertebral venous plexus.[3] The latter include the epidural veins of the lower portion of the thoracic, lumbar, and upper sacral areas of the epidural space.[2,9]

It has been hypothesized that the widespread venous engorgement of these epidural vessels causes them to enlarge to the point that they impinge on and distort the walls of the dural sac, changing its configuration to one that is more narrow and elongated, thus displacing CSF in a cephalad direction aided by the pulsations and "milking effect" of the effluent venous flow.[20] The result of the reduction in capacity of both the epidural and subarachnoid spaces simultaneously with narrowing of the dural sac accounts for the exaggerated spread of local anesthetic solution in these two spaces if the amount of active drug in the solution is not reduced.[1,4,10,16]

The effect of abdominal compression was verified in dogs by Hipona et al[13] and Hehre et al[12] employing epidural and spinal myelography. Inflating a balloon

Table 1. Physiologic Changes in Lumbar CSF Volume (T11–T12 to S2)

	Subject 1 (32-year-old man)	Subject 2 (41-year-old woman)	Subject 3 (26-year-old woman)	Subject 4 (33-year-old woman)	Mean	Mean Difference* (mL)
Resting lumbar CSF volume (mL)	42.0	34.8	39.9	28.3	36.2	
Hyperventilation [mL (% change)]	40.2 (−4)	30.0 (−14)	37.1 (−7)	22.9 (−19)	32.5 (−10)	3.7 ± 2.7†
Abdominal compression [mL (% change)]	31.1 (−26)	23.2 (−33)	34.9 (−13)	15.3 (−46)	26.1 (−28)	10.1 ± 5.6‡
Abdominal compression and hyperventilation [mL (% change)]	25.4 (−40)	21.3 (−39)	25.4 (−36)	13.3 (−53)	21.3 (−41)	14.9 ± 3.8§

* Analysis using paired *t* test.
† $P < 0.022$ (95% confidence interval).
‡ $P < 0.011$ (95% confidence interval).
§ $P < 0.0002$ (99% confidence interval).

catheter in the inferior vena cava just below the renal veins produced prompt engorgement of epidural veins and caused narrowing and lengthening of the column of contrast dye when placed in either the epidural or subarachnoid space. In addition, the contrast column regularly ascended at least three or more interspaces in a cephalad direction in the epidural space and, to a lesser extent, in the subarachnoid space.

The effects of hyperventilation on the volume change and displacement of spinal CSF may be explained by Burrow's modification of the Monro-Kellie doctrine.[8] The original version of Monro's hypothesis published in 1783 postulated that the volume of blood within the brain was fixed (because of the cranium being a rigid box filled with a "nearly incompressible" brain) and did not change in health or disease. Burrows in 1846 modified this concept[8] by adding that the blood volume of the brain could change, but only reciprocally with the volume of brain and/or CSF within the cranial cavity. Hyperventilation is known to cause cerebral arterial vasoconstriction, reducing the intracranial blood volume. Hence, there must be a reciprocal and compensatory increase in intracranial CSF volume, drawing CSF cephalad from a reservoir in the spinal subarachnoid region.

Over the past 25 years, two scientific articles of particular interest have been published regarding the effects of hyperventilation and/or abdominal compression on the dynamics of spinal CSF and the spinal dura mater in humans. Martins et al[15] in 1972, after observing 12 patients following routine diagnostic myelography, studied contrast material in the lumbar spinal subarachnoid space while cerebral blood volume was reduced by hyperventilation for 2 minutes, as well as during other physiologic maneuvers. During hyperventilation the lumbar dural sac partially collapsed as indicated by the Pantopaque column narrowing and elongating. When hyperventilation was discontinued, the sac gradually assumed its original appearance. It was concluded that the spinal dural sac was a dynamic structure, readily changing its capacity in response to prevailing pressure gradients across its walls, acting as a reservoir for CSF. However, the mechanism for the change in dural configuration was not elucidated, although it was hypothesized that the epidural venous plexus was involved.

In 1986, Taneda et al[19] studied 12 patients following subarachnoid injection of the radioisotope In-111-DTPA into the lumbar area. Hyperventilation successfully lowered intracranial pressure in eight of 10 patients. Radioactivity increased in the cranial and thoracic areas and decreased in the lumbar area in the same eight cases. As explained above, the increase in intracranial CSF volume in response to hyperventilation in this study was a compensatory maneuver to fill this space after the decrease in cerebral blood volume resulting from hyperventilation. Thus, it was concluded that the spinal subarachnoid space plays the role of a CSF reservoir that changes its capacity in supplying CSF for intracranial fluid volume compensatory adjustments.

With abdominal compression, radioactivity always decreased in the lumbar area and increased in the thoracic. In the cranial area, although it usually increased, no apparent increase was observed at times. When the increase was very small, or there was no increase, there was a proportionately larger increase in thoracic radioactivity.

The qualitative findings described above have been quantitatively corroborated in the present study. Although the number of subjects in our study is small, the volume changes with physiologic maneuvers are consistent in all subjects and are highly statistically significant. The volume of CSF in the lumbar spinal subarachnoid space has been measured by our technique and ranged from 28 to 42 mL in four normal subjects. It decreased by 10% with hyperventilation and 28% with abdominal compression. These effects are roughly additive, with 41% lumbar CSF volume decrement with combined compression and hyperventilation.

Furthermore, the cause of the reversible change in morphology of the thecal sac with abdominal compression is directly visualized to be mechanical compression by the markedly engorged epidural venous plexus.

The abdominal compression had to be applied quite firmly to demonstrate measurable changes in lumbar CSF volume. When the subjects reported only mild or moderate sensation of abdominal compression, no significant change in volume of the epidural venous plexus or thecal sac was measured. This likely corresponded to incomplete or mild occlusion of the IVC, resulting in

insufficient back pressure to effectively engorge the venous plexus.

The reversible occlusion of the IVC resulting from firm abdominal compression was directly visualized by a T1-weighted abdominal MRI sequence obtained during the imaging session, without and with abdominal compression (Figure 5).

This directly confirms the theory[10] that obstruction of the IVC results in elevated venous pressure sufficient to engorge the collateral epidural venous plexus and cause marked enlargement of these vessels, which compress the walls of the dural sac, changing it to a narrower configuration. This decrease in lumbar CSF volume by about 28% explains the empirically observed necessity to reduce the volume of spinal anesthetic by about 30% in patients with abdominal masses: there is less CSF in the lower thoracolumbar region to dilute the intraspinal anesthetic that is administered in the lumbar region.

Positioning patients supine for spinal surgery, with abdominal bolster, may simulate the described technique of abdominal compression, which has been shown in this study to cause epidural venous plexus engorgement and resultant compression of the thecal sac. One may speculate whether this could exacerbate spinal stenosis during the surgery, but it would be essential to understand the pressures involved before concluding that there was resultant neural compression.

The additive effect of abdominal compression and hyperventilation represents a combination of two independent effects: the decrease in lumbar thecal volume because of venous plexus engorgement from caval obstruction, coupled with the effect of hyperventilation, which in decreasing cerebral blood volume requires reciprocal replacement of the deficit by intracranial CSF drawn from the spinal CSF reservoir below.

In the past, anesthesiologists and physiologists could only indirectly infer changes in spinal CSF volume with physiologic maneuvers by observing the effects of spinal anesthetics, or only qualitatively observe CSF volume changes with myelography. Although the few articles cited above[12,13,15] have used myelography to document changes in spinal CSF volume with physiologic maneuvers, the rather dramatic changes in spinal CSF volume and deformity of the thecal sac by marked physiologic engorgement of the epidural venous plexus have not been widely appreciated by physicians.

Myelography is invasive and nowadays generally unacceptable for research studies on human subjects; in addition, myelography and postmyelogram CT, as well as radionuclide techniques, expose the patients to ionizing radiation and produce estimated volume inaccuracies of 20–30%.[5] Furthermore, plain myelography cannot yield accurate quantitative volume measurements (although such measurements could conceivably be performed using postmyelography CT, albeit with added radiation exposure to the subjects).

Moreover, the anatomic basis of these volume changes (epidural venous plexus engorgement) cannot be directly visualized by other imaging techniques, except perhaps by CT with intrathecal and intravenous contrast. As demonstrated here, however, using T2-weighted 3D FSE MRI, changes in the spinal CSF volume may be noninvasively and accurately measured, and the engorgement and enlargement of the epidural venous plexus can be directly observed, all without administration of intravenous or intrathecal contrast agents. MRI (using a thick-slab inversion-recovery sequence, rather than T2-weighted thin-section 3D FSE) has previously been used to measure brain CSF volumes.[5]

This MRI technique (fat-saturated T2-weighted 3D FSE) may be of use to physiologists in understanding the dynamic physiology of the spinal CSF space in health and disease and may be of importance to anesthesiologists in understanding more clearly the physiology of spinal anesthesia, specifically the dilution or concentrating effect of these changing CSF volumes on intraspinal anesthetic agents.

■ Key Points

- MRI provides a noninvasive means to measure spinal CSF volume and demonstrates the anatomic basis of physiologic volume changes.
- Adult resting lumbar CSF volume ranges from 28 to 42 mL.
- Reversible lumbar CSF volume change (10% reduction in volume with hyperventilation, 28% with compression, and 41% with combined hyperventilation and abdominal compression) is caused by engorgement of the epidural venous plexus, compressing the thecal sac.

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