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The Mechanism of Volume Regulation by Cervical Neuroforaminal Veins as was Previously Demonstrated for the Internal Vertebral Venous Plexus (IVVP) in the Cervical and Lumbar Region

Mecanismo de Regulación del Volumen por las Venas Neuroforaminales Cervicales como se Demostró Previamente para el Plexo Venoso Vertebral Interno (PVVI) en la Regiones Cervical y Lumbar

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SUMMARY: Head and cervical spine movements cause narrowing or widening of neuroforamina. In healthy individuals these movements do not cause symptoms of radiculopathy. This implies a compensating volume-regulating mechanism of the neuroforamina. Such a mechanism has been postulated in the years before CT and MRI for the neuroforaminal veins. Dural sac indentations with emptying and refilling of the internal vertebral venous plexus (IVVP) were postulated in the lumbar region using myelography. Emptying of the IVVP occurs in the lumbar spine when moving towards maximal extension and refilling while moving towards maximal flexion. Such indentations have not been shown in the cervical region. With MRI this mechanism has been demonstrated during axial rotation in the C1-C2 segment. It consists of emptying and refilling of the IVVP and thus prevents dural sac compression. During spinal surgery, the IVVP and connecting neuroforaminal veins may be damaged. Because the clinical implications of dysfunction of this protecting mechanism of the IVVP and its neuroforaminal venous connections are not clear, the consequences of such damage are unknown. Therefore, these venous structures should be examined by studying the cervical spine in supine position and, if possible, in different postures (flexion, extension and axial rotation) using MRI with contrast-enhancement and fat suppression. These images may be a basis for future advancement of clinical care.

KEY WORDS: Anatomy; Cervical neuroforaminal veins; Myelography.

INTRODUCTION

The term cervical radiculopathy refers to symptoms caused by nerve root dysfunction. This dysfunction can result from causes such as cervical disc herniation, tumor and presence of osteophytes leading to neuroforaminal stenosis (Woods & Hilibrand, 2015). Narrowing of the neuroforamen during movements of the cervical spine may also contribute to compression of the spinal root ganglia and nerves.

Mao *et al.* (2016) have described how cervical flexion leads to widening of the neuroforamen, resulting in an increase of the neuroforaminal cross sectional area (NCSA) as compared to the neutral position, while cervical extension causes NCSA reduction. This leads to a substantial NCSA difference between the extreme flexion and extension positions, as can be demonstrated in the C3-C7 segments, with respective NCSA differences of 27 %, 31 %, 18 % and 19 %, for these segments. Segments with greater ranges of motion show larger NCSA differences between the extreme flexion and extension positions (Mao *et al*., 2016). Muhle *et al.* (2001) have described a difference of the NCSA of about 50 % between the cervical segments in maximal extension and maximal flexion. Moreover, axial rotation in the cervical segments, which is coupled to lateroflexion, reduces the ipsilateral NCSA and increases the contralateral NCSA. During supine performance of Spurling's test in young healthy individuals the NCSA decreased to approximately 70 % of the control NCSA condition demonstrated with MRI (Takasaki *et al*., 2009).

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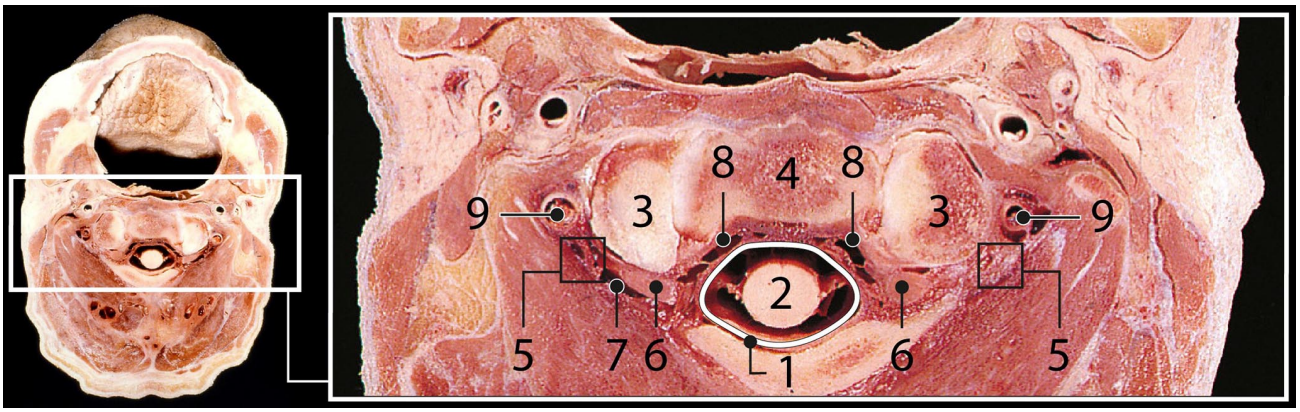

Fig. 1 Anatomical cross-section of the dural sac (1) including the spinal cord (2) and surroundings at the level of the lateral atlanto-axial joints (3) and base of the dens axis (4). In the region of the left and right neuroforamina C1-C2 (5) are presented the spinal ganglia and nerves (6) surrounded by neuroforaminal veins (7). Large parts of the IVVP (8) are presented in the anterior half of the epidural space. The vertebral arteries (9) are also surrounded by a venous plexus. This cross-section crop is from the original slice (source could not be identified) of figure 7.25 in: Gosling J. A.; Harris P. F.; Humpherson J. R.; Whitmore I.; Willan P. L. T. Human Anatomy: Text and Color Atlas, 6th edition.

In healthy individuals, movements of the head and cervical spine do not lead to symptoms of radiculopathy, indicating the presence of a mechanism by which the narrowing and widening of the neuroforamina during motion is compensated for.

The presence of volume regulation by neuroforaminal veins. Before the availability of CT and MRI Penning (1998) postulated that posture-dependent narrowing and widening of the cervical neuroforamen is associated with compression of veins in the neuroforamen during extension and refilling of the veins during flexion. The neuroforaminal veins connect to the internal vertebral venous plexus (IVVP), as described by Breschet in 1829, by Batson in 1940 and recently by Nathoo *et al.* (2011). These veins are shown in Figure 1, an anatomical transverse cross-section at the level of the C2 vertebra. This venous network surrounds all major traversing structures (Clemens, 1961; Daniels *et al*., 1986; Flannigan *et al*., 1987). This is illustrated in Figure 2, a cross-section of a virtual neuroforamen. Approximately forty percent of the NCSA is filled with motor and sensory nerve structures, leaving 60 % for volume regulation.

Penning (1998) also suggested that semi-liquid fat in the neuroforamen has a function in this process, however no publications by Penning (1998) relating to the volume-regulating mechanism of veins in the cervical part of the spine are available.

We also hypothesize that blood content in the neuroforaminal veins, but also present in veins in the groove for the spinal nerve in the transverse process of the vertebra, acts as a volume-regulating mechanism compensating for the widening and narrowing of the neuroforamen during

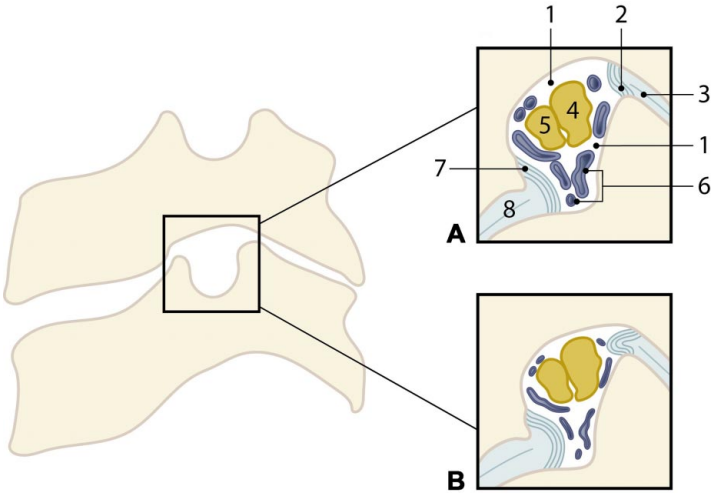

Fig. 2 Cross-sections through the cervical neuroforamen in maximal flexion (a) and extension (b): 40 % of the cross-section consists of the sensory root including spinal ganglion (4) and the motor root (5). The veins (6) are embedded in semi-liquid fat (1). The intervertebral joint (3) and its capsule (2) are positioned on the dorsal side and the uncovertebral joint (8) and its capsule (7) on the ventral side. In (b) the semi-liquid fat is, as suggested by Penning (1998), partly displaced out of the neuroforamen and the veins are emptied. In (a) fat and blood have returned (Adapted from figures 5.5.2 and 5.5.3 in the textbook of Penning, 1998).

movements of the cervical spine. Our hypothesis is based on data about a comparable volume-regulating mechanism of the internal vertebral venous plexus (IVVP) described previously in two papers co-authored by two of the authors (Penning & Wilmink, 1981; Reesink *et al*., 2001). We have not found more information about volume regulation by veins in and around the vertebral column in PubMed 1961 up to 2021.

Data about volume regulation by the IVVP. The most recent study, performed about 20 years ago, was carried out by Reesink *et al*. (2001), at the level of the atlanto-axial segment during axial rotation (Fig. 3).

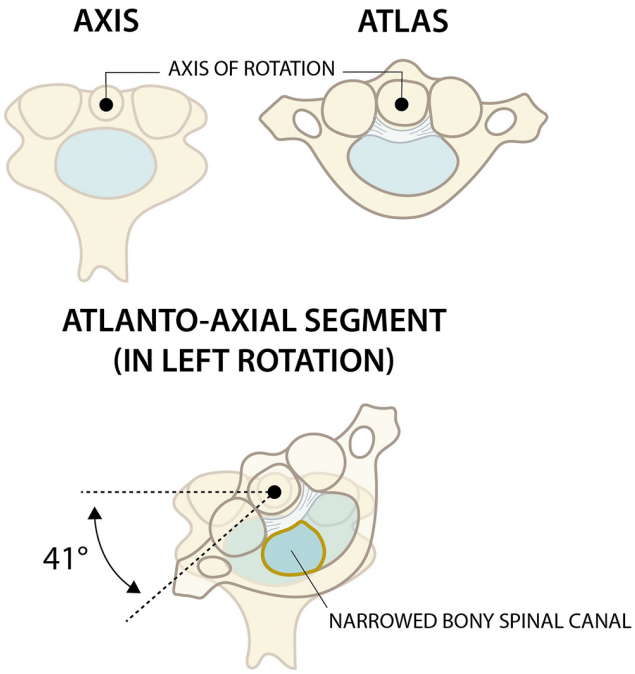

Fig. 3. Atlas and axis are superimposed. The cross-sectional area of the vertebral canal in maximal axial rotation is markedly reduced compared to that in the neutral position (Adapted from figure 4.10 in the textbook of Penning, 1998).

The second was carried out about forty years ago by Penning & Wilmink (1981) and focused on the position and shape of the lumbar dural sac in maximal extension compared to maximal flexion of the lumbar spine (Fig. 4).

In the study of Reesink *et al*. (2001), it is mentioned that upon excursion from the neutral position of the atlanto-axial segment to maximal axial rotation (Fig. 3) a 39 % reduction of the cross-sectional area of the spinal canal occurs, as was demonstrated by Tucker & Taylor (1998). In post-gadolinium contrast-enhanced fat suppressed T1-weighted MRI scans the blood distribution in the IVVP at the level of the atlanto-axial segment was investigated. In the neutral position of this segment a 50:50 ratio between

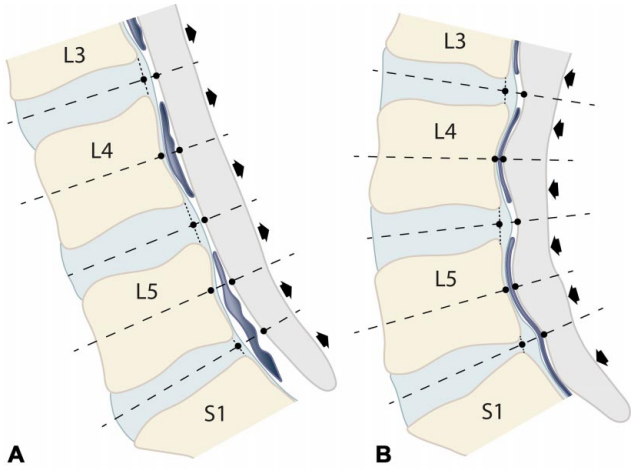

Fig. 4. Biomechanics of lumbosacral dural sac. A study of flexion-extension myelography. Spine. 1981; 6(4):398-408.) Drawings of lateral myelograms, in flexion (a) and extension (b). In (a) the anterior surface of the dural sac is straight and anterior epidural veins are full. In (b) the dural sac is displaced anteriorly by thickening flaval ligaments and indented by bulging discs at intervertebral levels combined with emptied anterior epidural veins at the mid-vertebral levels and the level of disc L5-S1. Note: The contour of S1 is positioned equally in a and b, thus showing the segmental ranges of motion of flexion-extension in the lumbar spine between L3 and S1 (Adapted from figure 7, of Penning & Wilmink, 1981).

left and right epidural area, containing the IVVP, was observed. This changed to 20:80 (ipsilateral:contralateral) cranial to the lateral atlanto-axial joints and a reverse shift occurs caudal to these joints. At the level of the lateral atlanto-axial joints the IVVP was completely collapsed in both maximally rotated positions of the atlanto-axial segment. Apparently, compensatory blood volume changes within the IVVP prevent compression of the dural sac and the spinal cord during atlanto-axial rotation (Reesink *et al*., 2001).

Indentations in the dural sac were described by Penning & Wilmink (1981) in the lumbar spine in maximal extension that were not present in maximal flexion. They also found an anterior displacement of the dural sac in maximal extension of the lumbar spine. A marked reduction in dural sac diameter at the intervertebral level was observed anteriorly by bulging of the posterior disc surface and posteriorly by shortening and thickening of the flaval ligaments (Fig. 4). Penning & Wilmink (1981) postulated that the natural response to local narrowing of the dural sac, which is a fluid-filled, elastic tube, would be to bulge in areas of less resistance. These are the epidural spaces behind the vertebral bodies that contain the anterior epidural venous plexus, which is especially extensive in the midline behind the vertebral bodies (Fig. 4). These veins are easily compressed, draining to valveless intravertebral and

paravertebral collaterals and thus probably functioning as "pressure stabilizers" allowing changes in epidural volume not leading to excessive intradural pressure changes in reaction to bulging discs and thickening of the flaval ligaments (Penning & Wilmink, 1981).

To the best of our knowledge, indentations of the dural sac during cervical spine movements in the lower cervical spine in healthy individuals have not been reported or studied yet.

Remarks on the data of volume regulation by the IVVP. The volume-regulating function of the IVVP and connecting veins related to motion of the bony spine has been studied only partially. The article by Reesink *et al*. (2001), provides information about volume regulation by veins in the cranial part of the bony vertebral canal, but only for axial rotation in the atlanto-axial segment. Veins around the vertebral artery and spinal nerves and ganglia are clearly visible as has been demonstrated about 15 years ago by one of the authors (JTW) on MRI scans of a single patient with a tumor in the cervical spine (Fig. 5).

However, no studies were found about volume regulation by veins in the neuroforamen and groove for the spinal nerve in the transverse process during flexion/extension or lateroflexion coupled to axial rotation caudal to C2, although substantial NCSA changes are described during movements of the cervical spine.

With the use of data from papers cited in PubMed we establish the following. The IVVP contains no mechanical valves and its walls are thin; no distinction can be made between the intima, media and adventitia. Almost throughout the whole IVVP the media lacks smooth muscle cells and is solely composed of collagen fibers (Clemens, 1961). The neuroforamen is surrounded by microvasculature, communicating with the IVVP. Visualization is possible by using contrast-enhanced MRI or post-contrast CT which allows detailed images of the neuroforaminal veins (Daniels *et al*., 1986; Flannigan *et al*., 1987; Tubbs *et al*., 2018). Daniels *et al*. (1986) showed the existence of large veins in this region, also in anatomical slices; however, they considered that their prominence was due to post mortem changes. It should be investigated with

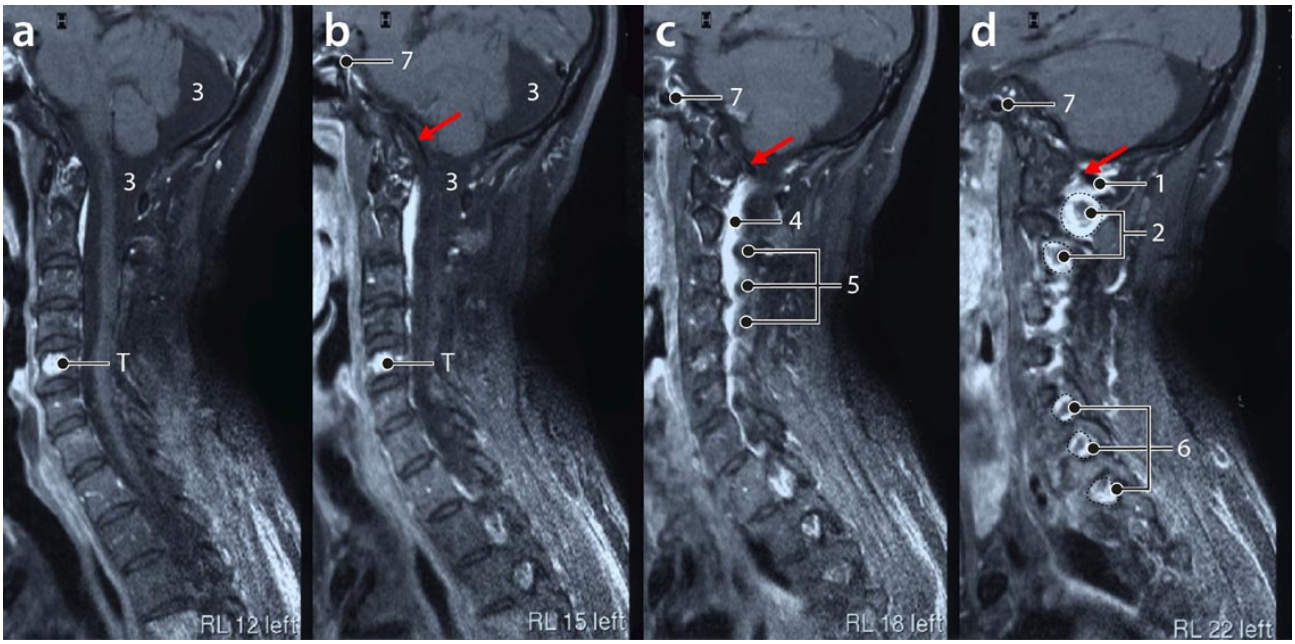

Fig. 5. Sagittal post-gadolinium contrast enhanced fat-suppressed MRI adjacent slices (thickness 3 mm) of a patient with a tumor (T) in the vertebral column. This series present the mid-sagittal slice (5A) up to the slice through the left neuroforamina (5D). In slice 5D a cross-section of the vertebral artery in the vertebral sulcus of the atlas is imaged (1), as also indicated by a red arrow in 5D and at the ventral side of the pons in 5C and 5B. The artery in 5D (hypointense) is surrounded by a venous plexus (hyperintense). This venous plexus shows contrast enhancement, because blood flow in this plexus is almost absent and the gadolinium in this plexus is still present in this post-injection phase as well as in the tumor (T in 5A & 5B). Also, the hypointense spinal nerves in the neuroforamina are surrounded by hyperintense enhancing veins (2 & 6). These neuroforaminal veins are circled by dashed lines. In slice 5B and 5A the cerebrospinal fluid for instance dorsal to the cerebellum and dorsal to the spinal cord is hypointense (3). Especially slice 5C presents the large longitudinal veins of the IVVP (4) with the spinal ganglia (5) as hypointense indentations dorsal to the enhancing IVVP. In 5D the hypointense spinal nerves C7, C8 and Th1 are visible (6) surrounded by hyperintense neuroforaminal veins. More dorsally, the chain of zygapophysial joints is present. The internal carotid artery is also visible in 5B, 5C and 5D (7).

new MRI or CT techniques whether this statement is correct. In the textbook by Penning (1998), it is suggested that the veins in the cervical neuroforamen facilitate volume changes during flexion/extension (Fig. 2). Because both motor and sensory nerve root as well as the spinal ganglion cannot change in volume during motion in the cervical segment (40 % of the NCSA), the remaining 60 % of the NCSA should be able to accommodate for all area changes caused by flexion, extension, axial rotation and lateroflexion.

The mechanism postulated by Penning & Wilmink (1981) on the occurrence of lumbar indentations of the dural sac in maximal extension has not been confirmed for the lower cervical spine in various positions, because similar studies have not yet been carried out in this latter region. Thus, clinical consequences of damaged veins in this region, but also in the lumbar region, are still unknown or have not been recognized as such.

With respect to the assumption of Penning & Wilmink (1981)that the dural sac is a fluid-filled elastic tube the following must be considered. The pressure in the IVVP and veins in the neuroforamen is very low, nearing zero mm Hg. Flow is even bidirectional in these venous structures (Nathoo *et al*., 2011). The normal pressure of the cerebrospinal fluid lies between 8-20 mm Hg (Adigun & Al-Dhahir, 2020). If the dura mater were to be an elastic easily deformable membrane, the dural sac should compress the IVVP and neuroforaminal veins in all postures of the cervical and lumbar spine. This does not occur because the outermost layer of the dura mater consists mainly of collagen fibers, which run in three directions, longitudinal, horizontal and transverse, both single and in groups (Dittmann *et al*., 1998). Thus, the spinal dural sac is rather robust, deformable indeed, but minimally elastic. Epidural veins which are filled with blood when the lumbar spine is in maximal flexion prevent acute high pressures within the dural sac by bulging of the intervertebral discs and thickening of flaval ligaments when the lumbar spine is moving to maximal extension. Unfolding and stretching of the anterior side of the dural sac when the lumbar spine is moving from maximal extension to maximal flexion is allowed by refilling these veins. This mechanism is possible because inelasticity of the dural sac prevents collapse of the epidural veins by intradural pressure when the lumbar spine is in maximal flexion.

Only in cases in which arterial pressure is present in the epidural space, as has been suggested by Beatty & Winston (1984) in cases of spontaneous cervical epidural hematoma or in case of a space occupying lesion such as a herniated disc (Abbed & Coumans, 2007), will the dural sac show major indentations, which may lead to symptoms of radiculopathy or myelopathy (McCartney *et al*., 2018).

Ramsey (1959) studied the distribution of epidural fat in different species, including humans. Generally, in humans more epidural fat is found in the lumbar region of the spine than in the cervical region. It can be assumed that more volume regulation by venous blood is needed in the epidural space cranial to Th1, because in this part of the spine the segmental flexion/extension ranges of motion, and thus the NCSA changes, are larger than in the thoracic and lumbar spine. Therefore, a small semi-liquid fat transportation mechanism in the cervical spine, as suggested for the lumbar spine by Penning (1998), is unlikely. Displacement of this fat can only be facilitated by volume changes in venous structures, otherwise the fat cannot migrate elsewhere.

CONCLUSION

This point of view manuscript describes the mechanism of volume regulation in the neuroforamina, as has been suggested so far for the lumbar neuroforaminal veins and was proven for the internal vertebral venous plexus (IVVP) within the cranial part of the cervical spine and in the lumbar spine.

In the cervical as well in the lumbar region, volume regulation in the vertebral canal of the IVVP has been demonstrated previously. During flexion/extension movements of the cervical spine, widening and narrowing in the neuroforamina, leading to an increase/decrease in NCSA's is described. However, whether volume regulation by veins prevents neural structures from being compressed during these movements has only been suggested in the papers of Reesink *et al*. (2001) and Penning & Wilmink (1981). With a search strategy in PubMed (see appendix 1) it is shown that no other articles about the mechanism of volume regulation in and around the spine have been published. To understand the dynamics of this mechanism in the neuroforamen 16 reference articles published in PubMed about the dynamics of the neuroforamen and anatomy/physiology of the epidural and neuroforaminal veins are used. These venous structures are illustrated in a classic anatomy slice (Fig. 1) and contrast enhanced MRI images (Fig. 5). Whether obstructions in the IVVP or neuroforaminal veins (thrombosis, embolus, fibrosis, cement) may lead to clinical problems should be further investigated. Moreover, what happens with (surgically) damaged venous structures in the epidural space and neuroforamina remains unclear. How much of the IVVP and/or neuroforaminal veins are necessary to become dysfunctional before clinical problems may occur on the

short or long term? We suggest examining the IVVP and neuroforaminal veins in the cervical spine in the neutral position in the supine individual (as demonstrated in Fig. 5) and, if possible, in various positions of the head and cervical spine. Firstly, to understand impact of the volume-regulating mechanism by venous structures in healthy individuals and secondly in patients with radiculopathy or myelopathy to determine possible obstructions in these venous structures which may play a role in their symptoms. This should be done preferably with contrast-enhanced fat-suppressed MRI in sagittal planes. A late-phase contrast enhanced MRI, such as is used in tumor diagnosis, might be well suited for visualization of these venous structures, but with an angulation perpendicular to the neuroforamen. These images may be a basis for future advancement of clinical care in patients with suspected compression of a spinal nerve and/or ganglion.

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Previous to writing this paper, Mr. Glenn HC Dams, Master Student Medicine and Clinical Research has carried out a scoping review (epidemiological graduation thesis) of functions of the internal vertebral venous plexus (IVVP) mentioned in literature.

VAN MAMEREN, H.; BOSELIE, T. F. M; WILMINK, J. T.; WILLEMS, P. C.; SANTBRINK, H. V. & DE BIE, R. A. Mecanismo de regulación del volumen por las venas neuroforaminales cervicales como se demostró previamente para el plexo venoso vertebral interno (PVVI) en la regiones cervical y lumbar. *Int. J. Morphol., 42(3)*:685-691, 2024.

RESUMEN: Los movimientos de la cabeza y la columna cervical provocan un estrechamiento o ensanchamiento de las neuroforaminas. En individuos sanos estos movimientos no causan síntomas de radiculopatía. Esto implica un mecanismo compensador de regulación del volumen de las neuroforaminas. Este mecanismo se ha postulado en los años anteriores a la TC y la RM para las venas neuroforaminales. Mediante mielografía se postularon hendiduras del saco dural con vaciado y llenado del plexo venoso vertebral interno (PVVI) en la región lumbar. El vaciado del PVVI se produce en la columna lumbar cuando se mueve hacia la máxima extensión y se rellena mientras se mueve hacia la máxima flexión. En la región cervical no se han observado tales depresiones. Con resonancia magnética se ha demostrado este mecanismo durante la rotación axial en el segmento C1-C2. Consiste en vaciar y rellenar la PVVI y así evitar la compresión del saco dural. Durante la cirugía de columna, la PVVI y las venas neuroforaminales que las conectan pueden dañarse. Debido a que las implicaciones clínicas de la disfunción de este mecanismo protector de la PVVI y sus conexiones venosas neuroforaminales no están claras, se desconocen las consecuencias de dicho daño. Por tanto, estas estructuras venosas deben examinarse estudiando la columna cervical en decúbito supino y, si es posible, en diferentes posturas (flexión, extensión y rotación axial) mediante resonancia magnética con contraste y supresión grasa. Estas imágenes pueden ser una base para futuros avances de la atención clínica.

PALABRAS CLAVE: Anatomía; Venas neuroforaminales cervicales; Mielografía.

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