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Review

Can Mechanical Loading of the Spinal Dura Influence Cerebrospinal Fluid Movement?

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Abstract

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During a fresh human cadaver dissection around 2002, the spinal dura was opened along its length. Light hand pressure over the sacrum produced a visible wave that travelled cranially through fluid in the exposed canal. No pressure, displacement or flow was measured. Opening the dura also changed the conditions that govern fluid movement in life. The observation therefore raises a question; it cannot answer it. At the upper end of the canal, anatomical studies have traced connective tissue from the suboccipital muscles to the cervical dura. Fibres from the nuchal ligament also reach the dura at C1–C2. Their continuity offers a plausible route by which movement could alter the dural boundary, but the size of any effect during life remains unknown. This hypothesis-led mini-review examines that anatomy alongside measurements of cerebrospinal fluid (CSF) movement and proposes experiments with the dural sac intact, including simultaneous recording of respiration, pulse and pressure. In the anatomical study considered here, Zheng and colleagues examined 30 human head and neck specimens: 20 by dissection and ten as P45 plastinated sections. They identified a dense band within the nuchal ligament, which they called the “to be named ligament.” That study did not measure cerebrospinal fluid flow.

Keywords: Myodural bridge; rectus capitis posterior minor; rectus capitis posterior major; vertebrodural ligament; cerebrospinal fluid; craniosacral therapy; fascia.

Abbreviations: CSF, cerebrospinal fluid; OCI, obliquus capitis inferior; RCPma, rectus capitis posterior major; RCPmi, rectus capitis posterior minor; TBNL, to be named ligament; VDL, vertebrodural ligament.

1. INTRODUCTION

The proposed anatomical route begins where the rectus capitis posterior minor (RCPmi) reaches towards the cervical dura with other fibres arriving by neighbouring paths. Dissection, plastination and histology have documented these connections (Zheng et al., 2014; Hack et al., 1995; Scali et al., 2011; Scali et al., 2013; Pontell et al., 2013; Nash et al., 2005; Humphreys et al., 2003; Zheng et al., 2018). Here, mechanical loading refers separately to active suboccipital muscle contraction, passive head movement and externally applied sacral contact. These interventions differ in force direction, magnitude and duration and must not be treated as mechanically equivalent. This review asks what happens when load travels through the documented attachments: does the intact dural sac change shape, and can any resulting cerebrospinal fluid movement be measured? These questions matter to neurosurgeons, neuroradiologists, spine surgeons and

clinicians treating headache. They also matter to practitioners who attribute fluid movement to gentle contact. Anatomical continuity alone cannot establish the effect of that contact. The author's earlier dissection provides the personal starting point. With the spinal dura open, light pressure over the sacrum produced a visible cranially travelling wave in the exposed fluid. The observation was unrecorded and the open canal could not retain pressure as it does in life. It motivates a controlled experiment with the dura closed; it does not demonstrate normal CSF circulation.

1.1 The Suboccipital Muscles and Their Dural Connections

Rectus capitis posterior minor (RCPmi) runs from the posterior tubercle of C1 towards the medial inferior nuchal line. Rectus capitis posterior major (RCPma) begins at the spinous process of C2 and reaches farther laterally on that line. Hack and colleagues described a connection from RCPmi across the posterior atlanto-occipital interval to the dura (Hack et al., 1995). Scali and colleagues identified another route from RCPma at the C1–C2 interval and then examined that connection histologically (Scali et al., 2011; Scali et al., 2013). Obliquus capitis inferior also contributes fibres in this region (Pontell et al., 2013). Nash and colleagues used sheet plastination and confocal microscopy to trace RCPmi fascia, tendinous fibres and perivascular sheaths towards the dura (Nash et al., 2005). Humphreys and colleagues likewise identified connective tissue attachments in the upper cervical canal (Humphreys et al., 2003). In five fixed human specimens, Zheng and colleagues stained the attachments and found predominantly parallel type I collagen fibres. They traced contributions from RCPmi, RCPma and obliquus capitis inferior through the two posterior interspaces to the dura (Zheng et al., 2018). This architectural arrangement offers a pathway capable of transmitting tension, but does not measure force reaching the living dura. The term "site-specific fascia tuning pegs" has previously been used to describe local muscular and ligamentous influences on tissue tension (Sharkey, 2020). Here, a site-specific fascia tuning peg means a local muscular or ligamentous attachment whose measured loading alters tension or shape at a defined dural region. The prediction is a reproducible, load-dependent displacement of the adjacent dural boundary in the direction of the applied traction, followed, if fluid coupling occurs, by a temporally related pressure or CSF signal. Neither a deformation magnitude nor a clinically meaningful threshold can yet be specified from available evidence; pilot measurements must quantify displacement in millimetres and pressure changes in defined pressure units. Failure to detect a load-related displacement beyond calibrated measurement uncertainty and sham responses would weigh against the proposed mechanism under the tested conditions. The original tuning-pegs publication concerned upper-limb nerve entrapment (Sharkey, 2020); applying that model to the craniocervical junction remains preliminary rather than established.

2. FROM NUCHAL LIGAMENT TO POSTERIOR DURA

Zheng and colleagues dissected 20 specimens and examined another ten as P45 plastination sections (Zheng et al., 2014). The dense band within the nuchal ligament was visible in 29 suitable specimens; the horizontal section did not allow that band to be assessed in the same way. In 22 of the 29, its fibres arose below C3 and curved over C2. In seven, they began higher and ran more directly towards the C1–C2 interval. Some RCPmi fibres joined the band before it entered the interval. The authors named it the to be named ligament (TBNL; Figure 1). Its two courses show why the connection cannot be drawn as a single, identical cord in every person. The TBNL reached the posterior cervical dura through the C1–C2 interval. Zheng and colleagues described a wider vertebrodural ligament (VDL) at this site (Zheng et al., 2014). One contribution arose from the posterior arch of C1 (V1). The TBNL supplied another (V2), while a third came from the C2 lamina (V3). These fibres met at a visibly thickened area of dura. Significantly, a connection was found in all 30 specimens. Its components varied in size, and the authors classified five patterns among the 29 specimens in which the whole arrangement could be assessed. Figure 2 shows these contributions as they reach the dura (Zheng et al., 2014). Their observed positions identify potential load paths. The images cannot establish the force carried by each path.

3. DISCUSSION

3.1 Could This Anatomy Contribute to CSF Displacement?

At C1–C2, movement loads the attachments described above. The parallel collagen fibres identified by Zheng and colleagues provide an anatomical route for force transfer (Zheng et al., 2018). Zheng and colleagues proposed that the suboccipital muscles might contribute to CSF movement through these connections (Zheng et al., 2014; Zheng et al., 2018). The anatomical evidence alone cannot establish a pumping action. The 2014 dissections noted slight lateral drawing of the posterior dura during neck rotation; however, the displacement was not measured nor was there a record of CSF movement (Zheng et al., 2014). Mechanical strain offers a plausible route by which movement might affect the dura. In experimental ex vivo and in vivo connective-tissue preparations, applied stretch changes fibroblast shape and cytoskeletal organization (Langevin et al.,

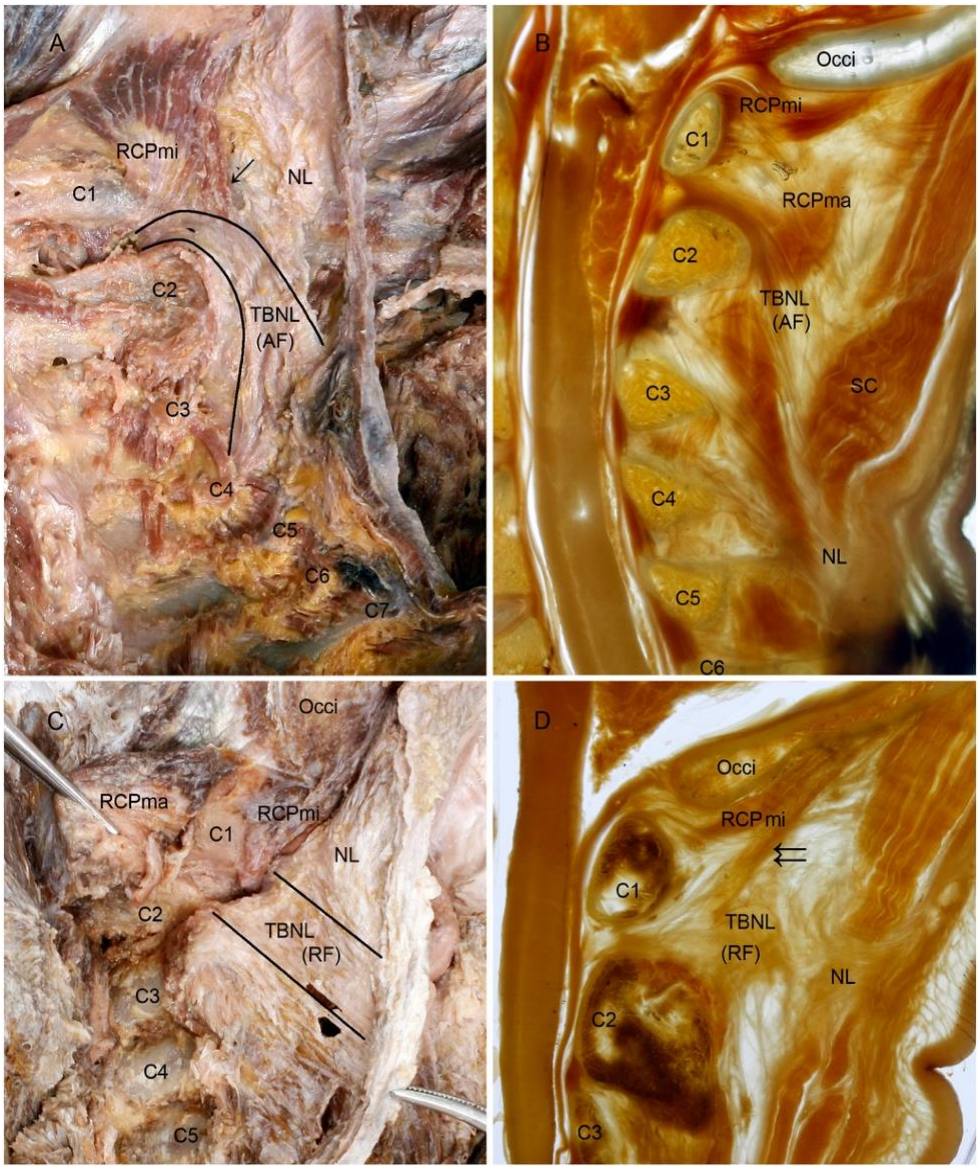


Figure 1. Dissection and sagittal P45 sections from Zheng et al. (2014). RCPmi, rectus capitis posterior minor; RCPma, rectus capitis posterior major; NL, nuchal ligament; TBNL, to be named ligament; AF, arcuate fibres; RF, radiating fibres; C1–C7, cervical vertebrae; Occi, occipital bone; SC, splenius capitis. Arrows indicate RCPmi contributions to the TBNL. Courtesy of Professor Hong-Jin Sui. Reproduced from Zheng et al. (2014) under the Creative Commons Attribution 4.0 International Licence.

2005); these findings do not establish the magnitude of strain or a cellular response in the human spinal dura. Whether movement produces comparable strain in the myodural bridge or dura, and how much deformation results, remains to be measured. Evidence elsewhere in the canal shows that the dural boundary is responsive to load. Dai and colleagues observed changes in the lumbosacral dural sac with lumbar flexion and extension (Dai et al., 1991). Martins and colleagues recorded changes in the spinal dura and CSF associated with

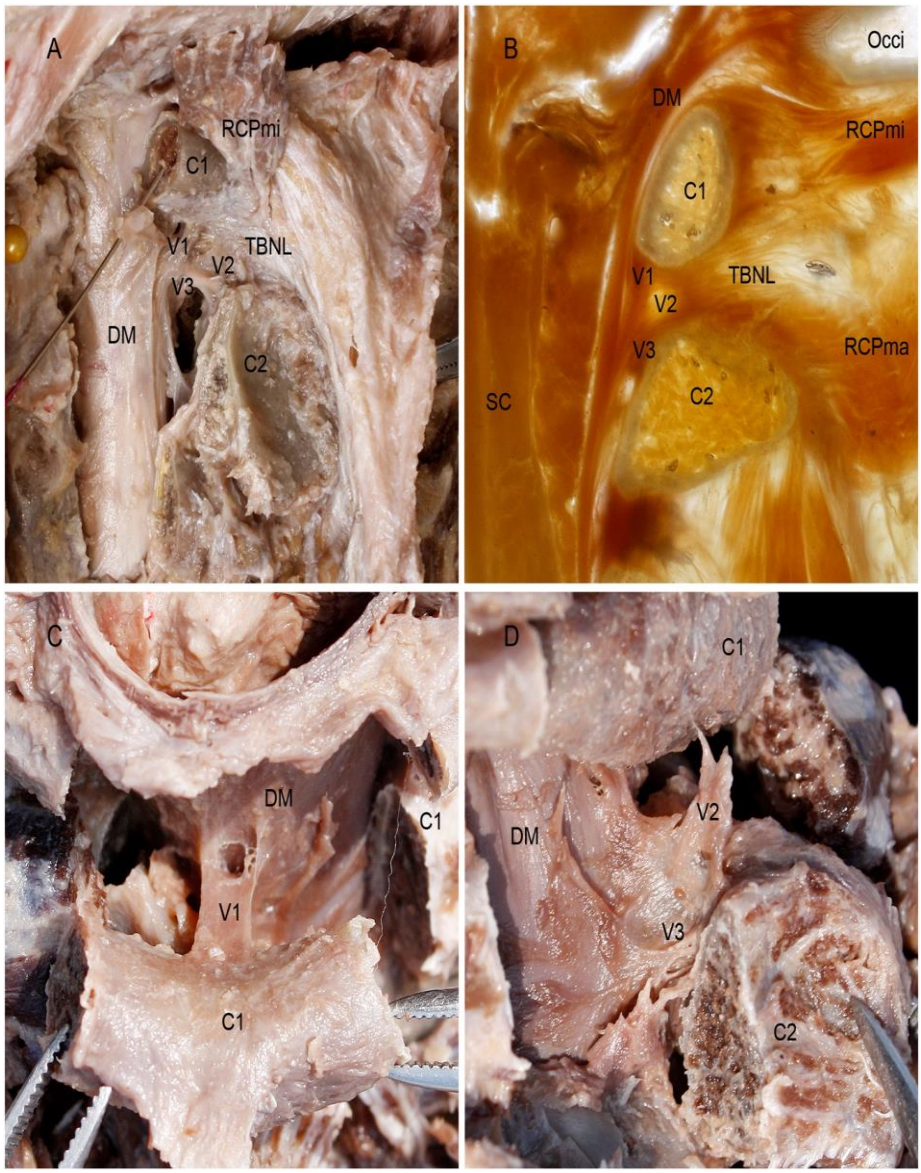


Figure 2. Vertebrodural connections in dissection and P45 section from Zheng et al. (2014). V1 arises from C1, V2 continues from the TBNL, and V3 arises from C2; DM, dura mater; RCPmi, rectus capitis posterior minor; RCPma, rectus capitis posterior major; TBNL, to be named ligament; Occi, occipital bone; SC, spinal cord. Panels A, C and D show dissection; B shows a sagittal P45 section. Courtesy of Professor Hong-Jin Sui. Reproduced from Zheng et al. (2014) under the Creative Commons Attribution 4.0 International Licence.

pressure outside the dural sac (Martins et al., 1972). In living volunteers, real-time MRI has separated respiratory and cardiac contributions to CSF motion (Yildiz et al., 2017). Lloyd and colleagues related respiratory CSF movement to pressure changes in the thoracic and lumbar spinal canal (Lloyd et al., 2020). These studies support the need to record breathing, pulse and pressure while testing a new source of movement. Imaging studies conducted by Xu and colleagues provide notable local observations. Following a brief period of head rotation, cine phase-contrast MRI demonstrated altered CSF flow near the C1 vertebra in 40 volunteers (Xu et al., 2016). A

subsequent study reported changes after head nodding and measured lumbar CSF pressure in a separate cohort (Xu et al., 2021). It should be noted that the protocols used in these studies did not isolate a single muscle or demonstrate dural deformation at its attachment site. Head position can affect cerebral venous pressure and respiratory function. Movement also changes the mechanical loading of the suboccipital region, where connective tissue links the muscles to the cervical dura (Zheng et al., 2014; Fargen et al., 2025; Zafar et al., 2018). The observed flow oscillates bidirectionally and should not be interpreted as evidence of sustained cranial circulation. The proposed tuning-peg action is supported by anatomical and physiological findings; however, the mechanical connection between these factors has not yet been quantified.

In a follow-up study, Zheng and colleagues identified a myodural bridge complex that connects the cervical dura to the rectus capitis posterior minor, rectus capitis posterior major, and obliquus capitis inferior muscles (Zheng et al., 2020). Ma and colleagues subsequently evaluated these anatomical connections in anaesthetised dogs (Ma et al., 2021). Passive head rotation, lateral flexion, and flexion–extension each resulted in increased CSF pressure. Electrical stimulation of the obliquus capitis inferior elevated mean CSF pressure from 12.41 to 13.45 mmHg. Severing its deep connective attachment abolished this pressure increase. Stimulation of the rectus capitis dorsal minor and both heads of the rectus capitis dorsal major, as designated in the canine study, did not produce statistically significant changes in CSF pressure (Ma et al., 2021). These findings support a contribution of the tested myodural attachment to transient CSF pressure modulation in anaesthetised dogs. The canine muscle nomenclature in that study (rectus capitis dorsal minor and major) is retained here and should not be assumed to establish equivalent force transmission in the corresponding human suboccipital muscles. Species-specific anatomy, anaesthesia and experimental stimulation limit direct extrapolation to human movement. A transient pressure rise is not evidence of sustained CSF circulation. The magnitude of dural deformation during typical human movement remains to be measured.

3.2 A Short Experimental Path

A fresh-frozen human specimen offers a practical first test, provided it is fully thawed and kept hydrated. It allows the dural sac to remain closed while preserving more of the tissue compliance needed to observe a small change in shape. Fixation can alter that behaviour. In human biceps tendon, formalin preservation greatly increased stiffness compared with fresh and fresh-frozen preparations; freezing also changed the measured stiffness (Hohmann et al., 2019). Comparisons of cadaveric muscles and tendons likewise show that preservation methods affect mechanical behaviour (Beger et al., 2020). Those results are from other tissues and cannot supply a correction factor for spinal dura. They explain why an embalmed specimen would be a poor sole model for this experiment, and why any cadaver result must still be checked in life. The first trial would leave the dura closed. Before data collection, the primary outcome would be specified as the time-locked sequence of applied load, local dural displacement and any subsequent CSF-related pressure or flow signal exceeding baseline drift and instrument noise. Sensor calibration, synchronization and measurement uncertainty would be documented; image measurements would be assessed by an observer blinded to load and sham condition. Pilot variability would inform a justified sample-size calculation for a subsequent confirmatory study. The specimen would be supported to prevent whole-body rocking, with temperature, hydration, prior freezing and thawing recorded. An instrumented contact over the sacrum would deliver a small, repeatable force. Imaging would track the dural wall while pressure sensors at lumbar and cervical levels record the timing and size of any transmitted signal. Sham contact and measurements of support movement would help identify motion artefact. After baseline recordings, controlled head rotation and extension could be applied separately. The force and angle of each movement would be measured. Only then would the dura be opened to examine whether the original visible wave can be reproduced under those altered conditions. The next step would study living participants. During controlled head movement, cine phase-contrast MRI could record CSF velocity while an anatomical imaging sequence tracks the cervical dural boundary. Muscle activity, head angle, breathing and pulse would be recorded together. A separate protocol could examine a light sacral contact with a matched comparison contact. A priori, a candidate loading-related signal would require a reproducible change from each participant's baseline, temporally following the applied load, exceeding calibrated measurement uncertainty and the distribution observed during matched sham trials after accounting for simultaneously recorded respiration and cardiac phase. Effect sizes and confidence intervals would be reported rather than relying on a visually apparent wave alone. The key observation is the timing: whether a measured load precedes a local dural change, and whether a CSF signal follows beyond normal respiratory and cardiac variation. Even a positive result would establish an association before it established the specific path of force. Clinical effects would require a different study.

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4. CONCLUSION

The muscles beneath the occiput have several documented routes to the cervical dura. The nuchal ligament adds another at C1–C2. Histology shows collagen fibres suited to bearing tension, and head movement has been associated with measurable changes in CSF motion. The missing observation is the one between them: what the intact dura does when a particular attachment is loaded. The author's

earlier sacral observation raises the same question from the other end of the canal. Measuring dural shape, pressure and CSF movement together can test whether these local attachments act as fascia tuning pegs and whether gentle contact produces a detectable effect.

DECLARATIONS

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CONFLICTS OF INTEREST

The author declares no conflict of interest.

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DATA AVAILABILITY STATEMENT

No new data were generated for this review.

AUTHOR CONTRIBUTION

John Sharkey conceived, wrote and approved the manuscript.

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