

# Cervicogenic Headache: A New Perspective

**Rob Sillevs\* and Valerie Weiss**

Department of Rehabilitation Sciences, Florida Gulf Coast University, Fort Myers, Florida, United States

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**\*Corresponding author:** Rob Sillevs, Department of Rehabilitation Sciences, Florida Gulf Coast University, 10501 FGCU Boulevard South, Marieb 428, Fort Myers, Florida 33965, Florida, United States

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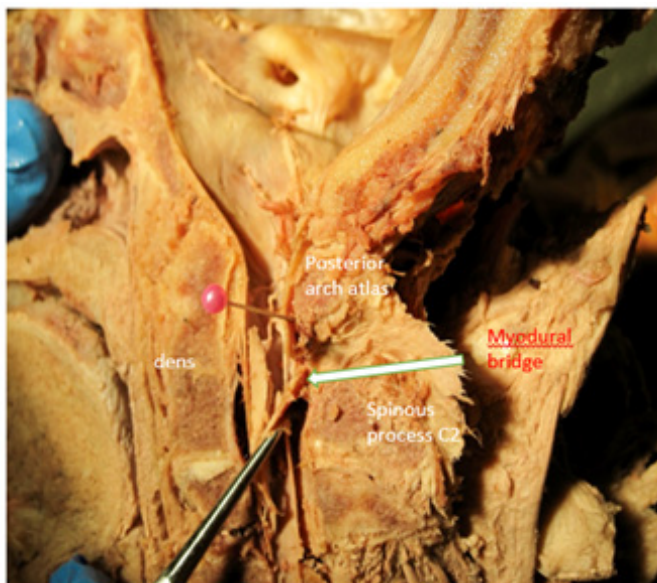
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## Mini Review

Headaches are common and result in a significant societal burden. Approximately 50% of the adult population in the United States is affected by headaches. Headaches result in a significant number of sick days and thus result in a loss of overall productivity. Currently, around 4% of the population experiences Cervicogenic Headaches (CGH) [1,2]. The prevalence of CGH is about 15% of all headaches [3]. Cervicogenic headaches are defined as “headache caused by a disorder of the cervical spine and its component bony, disc and/or soft tissue elements, usually but not invariably accompanied by neck pain” by the International Headache Society [4]. Individuals with CGH are typically managed by a variety of providers, such as general practitioners, neurologists, interventional pain specialists, chiropractors, and physical therapists. The exact underlying mechanism that results in CGH remains elusive. Despite the fact that there is no definite causation, individuals with CGH are likely to be treated with both pharmacological and non-pharmacological interventions. Considering the exact causative mechanism for CGH remains unclear the current management strategies and their long-term effects have to be called in question. The most current common rationale explaining the origination of CGH is based on referred pain from the upper cervical spine. Although any structure in the upper cervical spine could be considered causative, it appears that the Atlanto-Axial joint (AA) and the C2-3 zygapophyseal joint are the most frequent sources of CGH. Anatomical dissections demonstrate a direct relationship between the trigeminal nerve and spinal nerves C1 through C3 at the trigeminocervical nucleus: [5,6] the dorsal horns of the upper cervical levels synapse within the trigeminocervical nuclei. This pathway would allow nociceptive signals from the cervical spine to converge with the second-order trigeminal neuron. Furthermore, this neurogenic route could affect the ophthalmic branch of the trigeminal nerve and thus explain the subjective perception of orbital pain in subjects with CGH [5-7].

Another rationale for developing CGH might be directly related to the muscles that move the upper cervical region. Thus, muscular dysfunctions have been identified as a possible contributing factor for the development of CGH. Humans typically function in the upright position, and based on this, the upper cervical spine muscles will have to maintain the head position against the vertical gravitational compressive force. This need for muscle control commonly leads to increased muscle tone in the posterior upper cervical spine and the development of trigger points. It has been demonstrated that trigger points in the cervical musculature can refer back to the head and face [8,9]. More recently, collagenous tissue connections between the nuchal ligament and the dura mater have been demonstrated [10]. This connection implies a direct mechanical relationship between the cervical spine and the

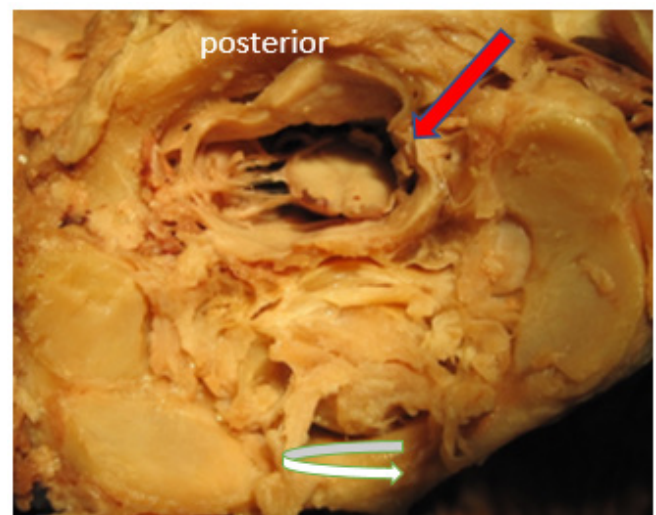
dura mater. Likewise, fascial connections exist between the Rectus capitis posterior minor, Rectus capitis posterior major, and the Obliquus capitis inferior with the surrounding dura [11,12]. These connections between muscle and dura have been identified as “myodural bridges,” spanning the arches of C1 and C2 and blending with the meningovertebral ligaments. The myodural bridges expand from there to cross the epidural space and connect into the posterior aspect of the dura (Figure 1) [11,12]. One could wonder about this anatomical significance of the connection between ligament, muscles, and the dura mater. This interrelatedness of structures might explain why clinicians have identified positive neural tension testing when treating individuals with cervicogenic headaches [13-15].



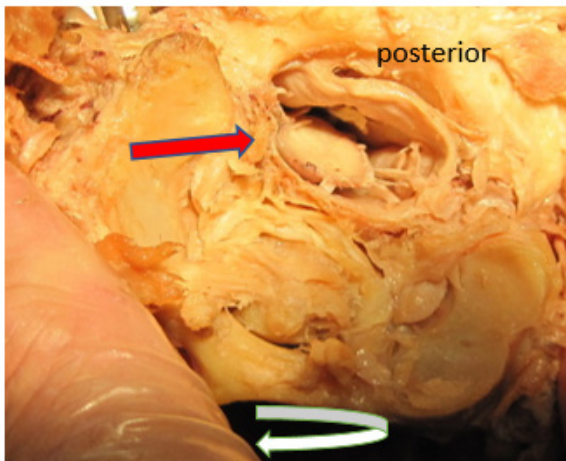
**Figure 1:** Midsagittal dissection demonstrating the myodural bridge between C1-2. White arrow indicates the dural connection.

Functional movement of the upper cervical spine is dependent on musculature, cervical joints, and stabilizing ligaments. The atlas plays a pivotal role in the rotational motion pattern of the upper cervical region. Hence, the position of the atlas relative to the axis appears to correlate with the presence of CGH as well [16,17]. Under normal circumstances, the atlas is well stabilized, relative to the dens of the axis, by the transverse ligament, not causing side effects. Of note, the transverse ligament has been identified as the strongest ligament in the region [18]. Mechanically, the transverse ligament allows the atlas to move in the transverse plane around the odontoid process. This rotational movement of the atlas is about 40-45 degrees relative to the axis, contributing to about 50% of the total rotation of neck [19]. The suboccipital muscles, especially contraction of the homolateral obliquus capitis superior and inferior muscles, will result in rotation of the atlas relative to the axis. It has been previously proposed and demonstrated that extended contraction of the homolateral obliquus capitis superior

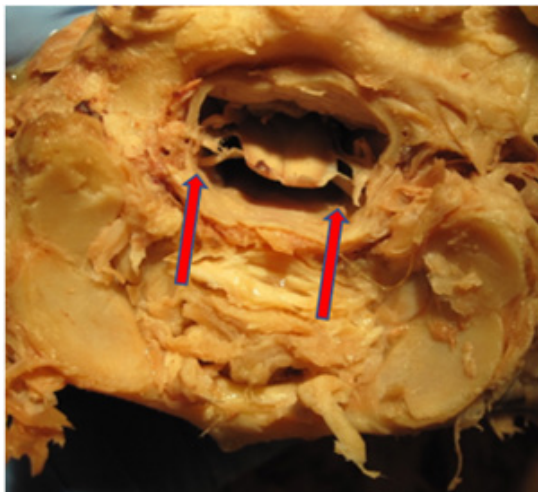
and inferior muscles can result in rotation of the atlas in a positional default position of the atlas [16,17]. (Figures 2 & 3) demonstrate such rotation of atlas performed manually on a dissected C0-C1 segment in the transverse plane. It can be noted that there is a significant shift of the spinal cord within the dural sac toward the side of rotation. When measuring this shift of the spinal cord to the rotational side compared to the neutral position (Figure 4), it was identical for both directions. Therefore, we postulate that this shift is significant and that as a result of ongoing unilateral muscular tone of the obliquus capitis inferior and superior muscles, there is tensioning of the dura. Kumar [20] identified that the cervical dura does not have a rich innervation of pain-sensitive nerve fibers as opposed to the cranial dura which does have pain-sensitive fibers. This is supported by the findings of Witten [21], who demonstrated that the cranial dura plays a contributing role in the development of headaches. Typically, subjects with CGH report headaches starting in the upper dorsal neck region (possibly due to prolonged muscle hypertonicity) and then spreading to the head. Jansen [22] demonstrated that after a cervical dorsal laminectomy freeing the dura from compression and or irritation, the subjects were relieved from CGH. Nosedá [23] suggest that the upper cervical muscle tone could result in dural tensioning with associated pain. They further demonstrated that the posterior dura overlying the cerebellum is innervated cervicovascular neurons, and this could lead to sensitization and pain of this region. In conclusion, there are various causes for cervicogenic headaches, including referred pain, muscular dysfunction, myodural bridges, and instability of the atlas. Moreover, there appears to be emerging evidence to consider the dura mater as a key component in the pathogenesis of cervicogenic headaches. Future research should further investigate the relationship between the dura mater and CGH as well as the position of the atlas related to dural tensioning.



**Figure 2:** Left rotation of atlas. The red arrow represents the movement of atlas to the left, relative to atlas and tension can be seen in the tissues on the right in the vertebral canal.



**Figure 3:** Right rotation of atlas. The red arrow represents the movement of atlas to the right relative to atlas and tension can be seen in the tissues on the left in the vertebra canal.



**Figure 4:** Neutral position of atlas and it can be seen that the spinal cord is symmetrical positioned in the dural sack. Red arrows indicate this position.

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