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Components and Clinical Implications of the Myodural Bridge Complex

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ABSTRACT

Introduction: the myodural bridge complex, a fibromuscular connection between the suboccipital musculature and the spinal dura mater, was first identified in 1995. Since then, research into its structure, function, and associated pathologies has expanded significantly.

Review: cadaveric and histologic evaluations have defined the contributing structures and identified its presence in a variety of animals. While the anatomy is well-established, investigations into its function is ongoing. Anatomical studies have shown the myodural bridge is crucial for maintaining structural integrity of the spinal cord during movement and may also be involved in cerebrospinal fluid (CSF) dynamics. Although the exact role remains elusive, emerging evidence suggests that dysfunction within this complex may contribute to a variety of craniocervical disorders, particularly cervicogenic and tension-type headaches. Dysfunctional alterations, such as hypertrophy or atrophy, could lead to changes in dural tension, nociceptive activation, and subsequent chronic pain syndromes. Changes in CSF dynamics may be implicated in conditions characterized or influenced by alterations in intracranial pressure, such as Chiari Malformation and syringomyelia.

Conclusion: this perspective piece integrates anatomical, developmental, and clinical data to provide a holistic view of the myodural bridge. This approach is intended to highlight the clinical implications of myodural bridge dysfunction and recommend its inclusion in the differential diagnosis when evaluating patients with cervicocephalic pain. Moving forward, research should transition from anatomical evaluation towards developing and evaluating the efficacy of therapeutic interventions. By translating anatomical knowledge into practical treatment strategies, providers will be better equipped to address the needs of patients suffering from myodural bridge related disorders.

Keywords: Myodural Bridge; Cervicogenic headache; Cerebral spinal fluid dynamics; Craniosacral therapy; Chronic neck pain.

Introduction

In 1995, Hack *et al*. discovered a fibromuscular connection between the musculature of the suboccipital region and the cervical spinal dura mater, later termed the myodural bridge complex (MBC).[1] Since its discovery, this structure has been identified in various species, suggesting a potentially significant, or even essential, function given its anatomic conservation. This conclusion is further supported by mounting evidence suggesting that dysfunction within the MBC is associated with various pathological processes in humans, particularly cervicocephalic pain and conditions associated with alterations in the circulation of cerebral spinal fluid (CSF). Despite the growing body of data and several compelling theories, the exact function of the MBC remains uncertain. Specifics regarding function will be discussed, but it is sufficient to say current data suggests that the MBC has both passive and active roles, which contribute to support of the spinal cord as well as CSF dynamics.

While most research has focused exclusively on the anatomical or physiological aspects of the MBC, this paper offers a multidisciplinary exploration by integrating anatomical, physiological, developmental, and clinical data into a cohesive model. Because the structure is well-defined, and constructs are being developed to solidify our understanding of function, this new perspective is intended to shift the focus toward the clinical significance of MBC dysfunction for diagnostic and therapeutic considerations.

Literature Review

Structure

A landmark 1995 study by Hack et. al identified a previously undiscovered connection between the musculature of the suboccipital region and the dura mater of the spinal cord in human beings, which they termed "the myodural bridge"[1]. While this study is credited with the initial discovery, the concept of a muscular-dural connection dates back to the 1950s when French researchers Lazorthes, Poulhes, and Gaubert described a possible connection between the rectus capitis posterior minor (RCP-minor) and the spinal dura mater of the cervical region in their paper: "La duremere de la charniere cranio-rachidienne"[2]. Regardless of the individuals responsible for its discovery, this muscular-dural connection has become an area of increasing interest.

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This interest in the myodural bridge has inspired anatomical, histological, and radiographic examination of the area. Originally thought to only contain fibers of the rectus capitis posterior minor (RCP-minor), the myodural bridge is now believed to also be composed of fibers from the rectus capitis posterior major (RCP-major), obliquus capitis inferior (OCI), and a structure dubbed the "to be named ligament" (TBNL) – see Figures 1 & 2[1-17]. This expanded identification of components fundamentally altered our understanding of the myodural bridge, transforming it from a singular connection between the RCP-minor and the dura into a more complex entity.

At present, there is a relative consensus that these are the primary structures that make up the myodural bridge, leading to the comprehensive concept known as the "myodural bridge complex." This evolution in terminology reflects a deeper understanding of the MBC's potential roles in neuromuscular control and its implications for cranio-cervical health. The identification of multiple components has prompted researchers to explore not just the anatomical structure, but also the complex functional interactions within the MBC and between various components of the MBC.

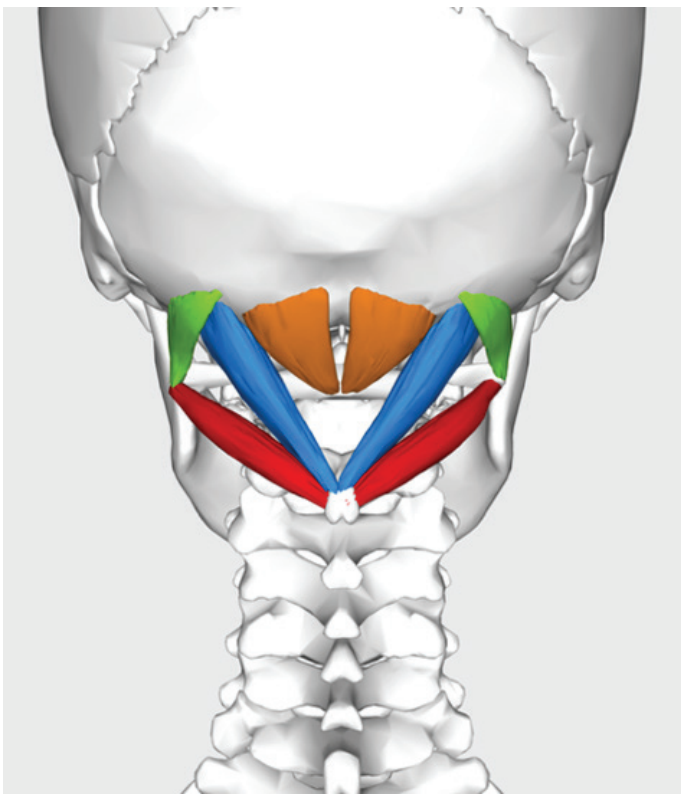

Figure 1. Suboccipital Musculature. Posterior view of the suboccipital muscles.
Legend: Orange = rectus capitis posterior minor; Blue = Rectus capitis posterior major; Red = obliquus capitis inferior; Green = Obliquus capitis superior.
Reproduced from Wikipedia, Suboccipital muscles (image: "Suboccipital_muscles09.png"), available under the Creative Commons Attribution-ShareAlike 4.0 International (CC BY-SA 4.0) license. Retrieved 10/24/2025.

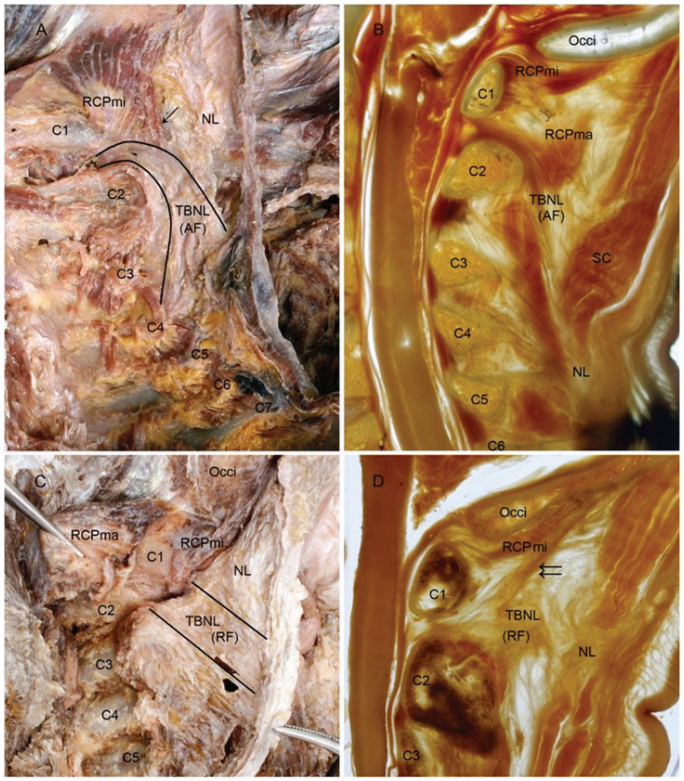

Figure 2. The "To Be Named Ligament" (TBNL) and vertebrodural ligament in the posterior cranio-cervical junction.
A, C: Lateroposterior aspect of the nuchal ligament.
B, D: Median sagittal section of the head–neck in a P45 plastination sheet.
Dissected specimens and P45 plastination sheets illustrate the arcuate and radiated fiber components of the TBNL. The arcuate fibers originate from the lower posterior border of the nuchal ligament (NL) below C3 and extend anterosuperiorly across the axis into the atlanto-axial interspace, attaching to the posterior cervical dura mater. The rectus capitis posterior minor (RCPmi) contributes muscular fibers to the TBNL. Radiated fibers arise opposite the C2–C3 spinous processes and also connect to the posterior dura.
Legend: TBNL = To Be Named Ligament; NL = nuchal ligament; AF = arcuate fibers; RF = radiated fibers; RCPma = rectus capitis posterior major; SC = splenius capitis; C1–C7 = first to seventh cervical vertebrae; Occi = occipital bone.
Reproduced from Zheng N *et al.*, PLoS ONE 2014; 9(8): e103451. doi:10.1371/journal.pone.0103451.

Function:

Since the original description of the MBC in humans, a similar structure has been identified in various species, including land mammals, sea mammals, avia, and reptilians[18-26]. The presence of this structure across different species led Zheng *et al.* to conclude that the MBC must serve a necessary function[6]. Several theories have been proposed:

A. Resistance to Dural Infolding:

The first theorized function of the MBC, proposed by Hack *et al.*, focused on maintaining integrity of the spinal cord during flexion and extension of the cervical spine[1, 27]. In their cadaveric studies, Hack and colleagues observed that the connective tissue bridge between the suboccipital muscles and the dura mater could provide mechanical support to resist dural infolding. This theory is supported by earlier research from Tachibana *et al.*, which used radiographic imaging

to demonstrate that extension of the head and neck could lead to inward folding of the spinal dura mater[28]. The resistance to dural infolding provided by the MBC would theoretically maintain patency of the spinal cord during cervical movements, preventing compression or alterations in intracranial pressure.

B. Proprioception:

Over time, understanding of the MBC's function expanded to include its potential role in proprioception. Histological analyses, by Scali and colleagues revealed the presence of proprioceptive neurons throughout the tissues involved in the MBC[29]. These findings, based on immunohistochemical staining techniques suggest that neurons within the MBC monitor changes in dural tension and communicate these changes to the associated musculature. This detection enables a biofeedback loop that leads to adaptive contraction or relaxation of the suboccipital muscles. This adaptation may play a role in resisting dural infolding and maintaining the subarachnoid space, ensuring stability of the spinal cord during movement[5,6,29].

C. CSF Dynamics:

Building on the theory of proprioceptive biofeedback, recent evidence suggests that the MBC may also play a role in CSF dynamics[4-6,18,30-33]. Studies by Dou *et al.* and Zheng et. al employed a combination of anatomical dissections, imaging techniques, and biomechanical simulations to explore this hypothesis[4-6,18]. They found that the proprioceptive fibers within the MBC, which help maintain appropriate dural tension, could also contribute to the circulation of CSF. Xu *et al.* speculated that the MBC may act as a pump, providing mechanical force to facilitate CSF flow[32]. This theory has also been explored by Sui *et al.* and Labuda *et al.*, who proposed that the MBC could alter subdural and subarachnoid volumes through its contractile activity, creating a negative pressure gradient to promote CSF circulation[30,34]. These studies used dynamic MRI and computational modeling to simulate the effects of MBC contraction on intracranial pressure and CSF flow, providing a plausible mechanism for this proposed function.

Dysfunction:

Interactions between the proprioceptive fibers of the MBC and the suboccipital muscles can lead to changes in dural tension. Proper function of this pathway supports the stability of the cervical spinal cord during movement, maintenance of the sub-dural and sub-arachnoid spaces, and mechanical forces that may contribute to CSF circulation. Although these functions are still speculative, there is mounting evidence to suggest that failure of these proposed mechanisms and dysfunction of the MBC is associated with various pathologic processes. The correlation between dysfunction and pathology involves both the passive and active roles of the MBC in maintaining the stability and function of the cervical spine and dura mater, as outlined below:

A. Cervicocephalic Pain:

The MBC consists of connective tissue fibers that link the suboccipital muscles, particularly the RCP-minor and RCP-major, directly to the cervical dura mater. Because of this reciprocal relationship, tension, strain, or dysfunction in these muscles can be transmitted directly to the dura mater. The dura mater is richly innervated with nociceptive fibers. When abnormal tension or mechanical stress is transmitted from the suboccipital muscles to the dura via the MBC, these nociceptive fibers can be activated, potentially leading to neck pain or pain that is perceived as a headache[17,27,35-38]. Since the MBC is muscular in nature, environmental triggers can cause hypertrophy or atrophy of the involved muscles. In the context of tension-type headaches, chronic muscle tension or hypertrophy in the suboccipital muscles could further exacerbate this mechanism, leading to persistent dural tension. Specifically, atrophy of the RCP-minor has been associated with chronic tension-type headaches[27,36,37].

B. Neuroinflammation, Central Sensitization, and Chronic Pain:

Activation of nociceptive fibers triggers a local and central inflammatory response intended to protect endangered tissue. If there is prolonged activation of nociceptive fibers, a chronic inflammatory state develops, affecting both the peripheral and central nervous system (CNS). Peripheral receptors can become hypersensitive to stimuli and hyperexcitable, leading to a lower threshold for ongoing inflammation. In the CNS, chronic neuroinflammation leads to vascular changes that alter permeability of the blood-brain barrier, allowing increased passage of immune cells that contribute to neuroinflammation[39].

This cycle of inflammation begetting inflammation, which is perceived by the individual as pain, is a maladaptive phenomenon referred to as central sensitization. Central sensitization is the process by which nociceptive pathways become hypersensitized due to prolonged activation of nociceptive pathways[40]. Eventually, this leads to structural changes in the CNS. This is referred to as maladaptive neuroplasticity – a process by which the brain's neural pathways are reinforced and perpetuate the experience of pain.

Instead of adapting in a way that reduces or alleviates pain, the nervous system undergoes changes that make pain signals more persistent and more easily triggered, even in the absence of the initial cause of pain. This can result in heightened pain sensitivity, chronic pain conditions, and increased difficulty in managing or reversing the pain state[39]. This is relevant to our discussion because of the presence of nociceptors

within the dura mater. Prolonged activation of dural nociceptors may be a source of neuroinflammation, maladaptive neuroplastic changes, and chronic cervicocephalic pain conditions such as cervicogenic headaches, tension-type headaches, and chronic neck pain.

Increased Intracranial Pressure (ICP):

The relationship between the MBC and CSF dynamics is an area of increasing interest. Initially, the MBC's influence on the flow of CSF was attributed to its ability to resist dural infolding during movement of the cervical spine[1,27]. Ongoing investigations have led to the development of several biomechanical models suggesting that the MBC may have a more direct influence on CSF dynamics. Changes in the function of the MBC due to muscular dysfunction or structural changes could lead to changes in dural tension, affecting ICP regulation. In addition to Idiopathic Intracranial Hypertension (IIH), alterations in the MBC are suspected to contribute to the pathophysiology of Chiari malformations[5,6].

Zheng *et al.*[5,6]:

Building on the theory of resistance to dural infolding, it was proposed that the MBC provides a stabilizing force to maintain dural tension. Atrophy of the suboccipital muscles may reduce the stabilizing effect of the MBC, reducing the capacity to resist dural infolding. On the other hand, hypertrophy may increase the mechanical forces exerted on the dura, leading to altered CSF flow and increased ICP.

Li *et al.*[31]:

Suggested that changes in the tension of suboccipital muscles as well as muscle contraction could influence the movement of CSF through a pumping mechanism. Abnormal muscle tension due to hypertrophy could lead to increased tension within the MBC with subsequent changes in CSF dynamics, potentially leading to increased ICP. They found that hypertrophy of suboccipital muscles leads to increased ICP, while severing these muscles leads to decreased ICP. This mechanism may play a role in the development of IIH.

Dou *et al.*[18,19]:

Offered an alternative to stabilization or pumping forces by suggesting that the MBC alters the subdural and subarachnoid spaces through muscle contraction. Through alterations in tension, negative pressure zones are created that facilitate the movement of CSF. Dysfunction of this mechanism may play a role in conditions such IIH or other ICP-related disorders.

Zhang *et al.*[25]:

Evaluated the anatomical relationship between the suboccipital cavernous sinus and the MBC, suggesting that tension and movement of the MBC may influence venous sinuses in the suboccipital region. These fluctuations have the capacity to affect intracranial venous pressure and, by extension, ICP.

D. Syringomyelia:

Syringomyelia is a condition where a fluid-filled cyst (syrinx) forms within the spinal cord. It has been suggested that changes in spinal cord intramedullary pressure may contribute to the formation and expansion of syringomyelia. MBC dysfunction leads to changes in CSF circulation and pressure dynamics. As a result, changes in intramedullary pressure may contribute to the development and progression of syringomyelia[28,34,42].

E. Post-Surgical Headache:

Researchers have identified the formation of dural adhesions originating from the MBC that form following surgeries affecting the suboccipital region. Hack and Hallgren evaluated 30 case studies of individuals who experienced post-surgical headaches after surgery involving the suboccipital region and discovered the following[27]:

1. These headaches were often severe and debilitating in nature. Patients were refractory to standard treatments and elected to undergo a myodural release procedure.
2. During the subsequent surgery, all patients were found to have dural adhesions originating from the MBC that were not present during the original surgery. Following surgical removal, a dural substitute was placed between the spinal dura mater and the suboccipital musculature to prevent the reformation of adhesions.
3. All patients experienced significant relief of pain and symptoms following the procedure.
4. Because the dura mater possesses nociceptive fibers, these adhesions were assumed to be pain generators that stimulated nociceptors when tension was produced by the MBC.

F. Connective Tissue Disorders:

In addition to acquired dysfunctions, it is important to note that the MBC relies on the strength and integrity of fibromuscular connections. In individuals with connective tissue disorders (such as Ehlers-Danlos Syndrome), there is an inherent weakness in the fibromuscular connections, causing laxity within the MBC. In both human and equine studies, this laxity leads to abnormal motion of the spinal cord. Both structural laxity and abnormal spinal cord motion have been identified as pathogenic factors associated with chronic neck pain and cervicogenic headaches[43,44].

Treatment:

Current approaches to neck pain, headaches, and migraines include, but are not limited to physical therapy, exercise, oral and topical analgesics, postural

correction, injections, and surgery. While some of these options may address MBC dysfunctions, there is a dearth of information regarding targeted therapeutic interventions. Current therapeutics directly targeting the MBC are limited, but they include:

A. Manual Therapy:

John E. Upledger, DO, was one of the first clinicians to suggest the presence of a connection between the muscular and neurological systems - which he called the cranial-sacral system[45]. He theorized that manipulation of the cranial/cervical regions could affect the central nervous system and benefit individuals suffering from neck pain, headaches (chronic and tension-type), and migraines. This treatment modality - referred to as craniosacral therapy (CST) - has been found to be an effective, non-invasive treatment option for patients with chronic neck pain[46,47].

B. Surgery:

Surgical intervention - in the form of a myodural release procedure - has been found to be an effective treatment modality for refractory post-surgical headaches[27].

Discussion

A Call for Development and Evaluation of Targeted Therapy

The correlation between MBC dysfunction and cervicocephalic pain syndromes such as chronic headaches, chronic neck pain, post-surgical headaches, migraines, and ICP-related disorders leads to the first purpose of this paper: (1) to increase awareness around MBC dysfunction and encourage the inclusion of MBC dysfunction in the differential diagnosis when evaluating associated conditions; especially in cases refractory to standard treatment. Because of the complex interaction between suboccipital muscles, cervical dura mater, and dural nociceptors, MBC dysfunction has been associated with multiple cervicocephalic conditions. Through central sensitization and maladaptive neuroplasticity, it is possible for these conditions to develop into chronic pain syndromes. Thus, treating MBC dysfunction has the potential to address multiple pathologic processes, and early intervention has the potential to interrupt the process of central sensitization. All of this makes the MBC an ideal target for therapeutic intervention.

This brings us to the second purpose of this paper: (2) to recommend that MBC research should transition from anatomic evaluation towards the development and evaluation of therapeutic interventions. At this juncture, it can be argued that there is enough structural, functional, and pathologic understanding of the MBC to shift focus towards actively investigating therapeutic interventions. By concentrating on translating anatomical knowledge into practical treatment strategies, providers will be equipped to address the needs of patients currently suffering from MBC-related disorders.

In 2015, despite significant advances in surgical and nonsurgical management, neck pain was the 4th leading cause of disability in the world[48]. With the prevalence of neck pain that is refractory to current treatment modalities, it may be worthwhile to approach this – and related pathologies – from a new perspective. There is a notable gap in the literature regarding comprehensive, controlled studies that evaluate the effectiveness of treatments specifically targeting MBC dysfunction. Much of the current evidence is anecdotal or extrapolated from treatment of related conditions such as myofascial pain, highlighting the need for further research in this area.

Conclusion

Anatomical evaluation has confirmed that the MBC is a structure universally. Histological evaluation has demonstrated that this structure consists of connective tissue, muscle fibers, proprioceptive neurons, and nociceptive nerve fibers. Investigation into the function has led to the development of several theories regarding the role of the MBC. Researchers have been unable to determine the exact function of the MBC, but several biomechanical models have been developed to support the proposed theories. Despite the lack of clarity regarding function, the pathology associated with dysfunction of the MBC has been rather informative. This perspective piece attempts to provide a comprehensive understanding of the clinical implications of MBC dysfunction by combining anatomical, developmental, and clinical data.

While extant research has prioritized singular understandings of structure, function, or dysfunction, this article attempts to integrate all relevant data into one cohesive picture with the goal of emphasizing potential clinical implications. The purpose of this paper consists of two main objectives: (1) to increase awareness around MBC dysfunction and encourage inclusion of MBC dysfunction in the differential diagnosis when evaluating associated conditions, especially in cases refractory to standard treatment and (2) to recommend that MBC research should transition from anatomical evaluation towards the development and evaluation of therapeutic interventions.

Given the clinical implications of MBC dysfunction, it is the author's opinion that MBC dysfunction should be included in the differential diagnosis when evaluating patients with cervicocephalic pain and other associated pathologies. Additionally, it is recommended that MBC research transition from anatomic evaluation towards the development and evaluation of therapeutic interventions. While the

acquisition of knowledge is laudable, the application of that knowledge is of equal importance. Patients with conditions affected by MBC dysfunction will benefit from therapies targeting the fibromuscular and neurological components. It is also worth noting that pursuing a greater understanding of therapeutic interventions may further elucidate the mechanisms influencing function and dysfunction.

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