

REVIEW OF THE LITERATURE



A Proposed Etiology of Cervicogenic Headache: The Neurophysiologic Basis and Anatomic Relationship Between the Dura Mater and the Rectus Posterior Capitis Minor Muscle

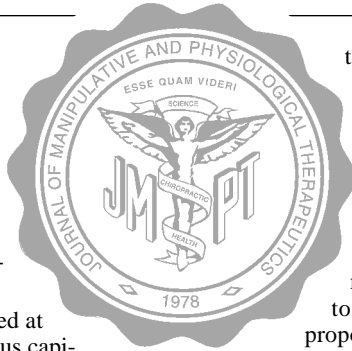
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ABSTRACT

Objective: To examine the neurophysiologic basis and anatomic relationship between the dura mater and the rectus capitis posterior minor muscle in the etiologic proposition of cervicogenic headache.

Data Sources: On-line searches in MEDLINE and the Index to Chiropractic Literature, manual citation searches, and peer inquiries.

Results: Connective tissue bridges were noted at the atlanto-occipital junction between the rectus capitis posterior minor muscle and the dorsal spinal dura. The perpendicular arrangement of these fibers appears to restrict dural movement toward the spinal cord. The ligamentum nuchae was found to be continuous with the posterior cervical spinal dura and the lateral portion of the occipital bone. Anatomic structures innervated by cervical nerves C1-C3 have



the potential to cause headache pain. Included are the joint complexes of the upper 3 cervical segments, the dura mater, and spinal cord.

Conclusion: A sizable body of clinical studies note the effect of manipulation on headache. These results support its effectiveness. The dura-muscular, dura-ligamentous connections in the upper cervical spine and occipital areas may provide anatomic and physiologic answers to the cause of the cervicogenic headache. This proposal would further explain manipulation's efficacy in the treatment of cervicogenic headache. Further studies in this area are warranted to better define the mechanisms of this anatomic relationship. (*J Manipulative Physiol Ther* 1999;22:534-9)

Key Indexing Terms: Headache; Chiropractic Manipulation; Cervical Vertebrae

INTRODUCTION

The pain and discomfort experienced from headaches are significant indicators that anatomic and physiologic changes have taken place. Headaches can result from cervical spine joint complex dysfunction,¹ vascular disease, metabolic dysfunction, brain tumors, and trauma. The severity of this disorder leads to more than 18 million annual office visits in the United States.² The Nuprin Report has documented that, "156 million full-time work days are lost each year because of headaches, at an estimated cost of 25 billion dollars in lost productivity."³ Headaches are also found to be the most common reason for the use of over-the-counter analgesic medication.⁴ The extensive impact of headaches on a patient's quality of life far exceeds that of other chronic conditions such as osteoarthritis, hypertension, and diabetes.⁵ Health care practitioners find headaches to be one of the most commonly addressed complaints. One study found that 27% of those reporting a headache have used a form of alter-

native management. Chiropractic was found to be the most common practitioner-based health care sought.⁶

The diverse nature of headaches and their symptoms have allowed for great disparity in the literature as to causation and management of resulting conditions. The failure to accurately determine the cause of chronic benign headaches (eg, cervicogenic, migraine, or tension type) has led to a variety of management techniques and treatment options that may or may not address the true nature of the patient's complaint. Studies conducted by the International Headache Society suggest that cervicogenic headaches account for approximately 15% to 20% of all recurrent benign headaches.⁷ The neurophysiologic basis for the cervicogenic headache is the convergence that takes place in the trigeminocervical nucleus between receptive fields of cervical nerves C1-C3 and the nociceptive afferents from the trigeminal nucleus. Structures innervated by the first 3 cervical nerves have shown a potential for causing a cervicogenic headache.⁸ The neurophysiologic basis and recent anatomic disclosure by Hack et al⁹ will be thoroughly examined in this review. This finding may provide anatomic evidence that implicates cervical spine joint complex dysfunction as a cause of cervicogenic headache. He reported a connective tissue bridge between the rectus capitis posterior minor muscle and the posterior spinal dura at the atlanto-occipital junction⁹ (Fig 1). This recent discovery, along with the suggested neurophysiologic mechanism, may provide significant evidence relating headache to the cervical spine joint complex dysfunction.

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Table 1. IHS diagnostic criteria for cervicogenic headache

<p>A. Pain localized to neck and occipital region. May project to forehead, orbital region, temples, vertex, or ears.</p> <p>B. Pain is precipitated or aggravated by special neck movements or sustained neck posture.</p> <p>C. At least one of the following:</p> <ol style="list-style-type: none"> 1. Resistance to or limitation of passive neck movements. 2. Changes in neck muscle contour, texture, tone, or response to active and passive stretching and contraction. 3. Abnormal tenderness of neck muscles. <p>D. Radiological examination reveals at least one of the following:</p> <ol style="list-style-type: none"> 1. Movements abnormalities in flexion/extension. 2. Abnormal posture. 3. Fractures, congenital abnormalities, bone tumors, rheumatoid arthritis, or other distinct pathology (not spondylosis or osteochondrosis).

From Nilsson N, Christensen HW, Hartvigsen J. The effect of spinal manipulation in the treatment of cervicogenic headache. *J Manipulative Physiol Ther* 1997;20:326-30.⁴⁴

This overview and subsequent correlation of the literature will examine the recent anatomic finding and apply this knowledge to the proposed neurophysiologic mechanism involved in the cervicogenic headache.

This article consists of a qualitative and critical review of reports on the treatment of chronic benign headaches by spinal manipulation. Studies that reported only on those individuals designated as benign headache patients were used. Single-participant case studies were excluded from this review. Five independent studies were examined in the confirmation of the connective tissue bridges at the atlanto-occipital junction between the rectus capitis posterior minor muscle and the posterior spinal dura. One study was also included that revealed a connection between the ligamentum nuchae and the posterior spinal dura. The search strategy in these reports included on-line searches in medical databases (MEDLINE, Index to Chiropractic Literature), manual citation searches, and peer inquiries.

DISCUSSION

Cervicogenic headaches are described as, “referred pain perceived in any region of the head caused by a primary nociceptive source in the musculoskeletal tissues innervated by cervical nerves.”¹⁰ The actual source of pain originates not in the head but in the cervical spine joint complex. Structures innervated by cervical nerves C1-C3 have been shown to be capable of producing cervicogenic headache pain. Possible sources of pain include the C2-C3 intervertebral disk annular fibers, muscles, joints, ligaments, and related dura mater of the upper cervical spine.⁸ Structural or functional abnormalities can occur in any of these components and manifest during rest or active or passive ranges of motion.⁷

In 1990, diagnostic criteria were established by the International Headache Society for the accurate diagnosis of cervicogenic headache (Table 1). Four categories classifying the mechanism of cervicogenic headaches have been suggested by Vernon. *Extrasegmental* refers to the myofascial structures and ligaments of the cervical spine. *Interseg-*

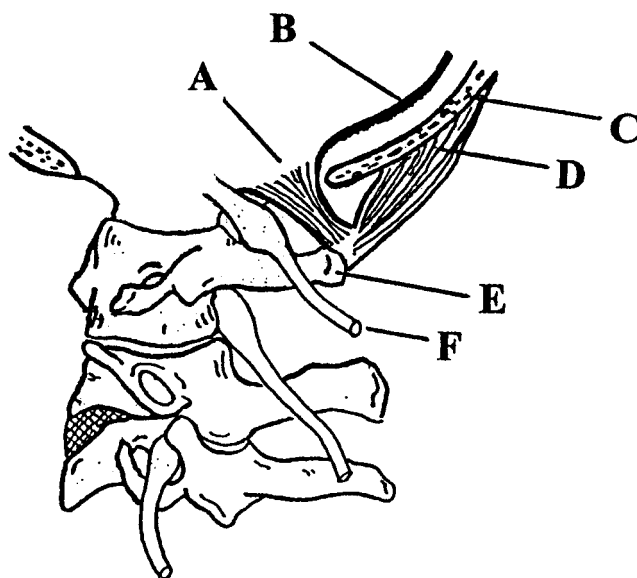


Fig 1. Lateral view of the upper cervical joint complex. (A) connective tissue. (B) Dura. (C) Occipital bone. (D) Rectus capitis posterior minor muscle. (E) First cervical vertebra. (F) First cervical nerve root.

mental refers to the joint complexes of C2-3, C3-4, the articulations, ligaments, and deep intersegmental muscles of the occiput-C2. *Infrasegmental* deals directly with the innervating nerves contained in and around the intervertebral foramina of the cervical spine. *Intrasegmental* mechanisms deal exclusively with the spinal cord and medullary dorsal horn, including the nucleus subcaudalis of the trigeminal nerve.¹¹ This classification system attempts to localize the anatomic and pathologic causative agents responsible for the cervical headache. Joint dysfunction affecting pain-sensitive structures in the neck and vascular malformations along the cervical C2-C3 nerve roots have been suggested causes in cervicogenic headache.¹²

Hack et al observed “a connective tissue bridge between the rectus capitis posterior minor muscle and the dorsal spinal dura at the atlanto-occipital junction in all specimens.”⁹ This bridge of connective tissue fiber was primarily orientated perpendicular to the spinal dura. The fibrous arrangement was noted to resist dural movement toward the spinal cord.⁹ The spinal dura has a tendency to fold inward in the direction of the spinal cord.¹³⁻¹⁶ Hack et al suggest that their finding of a connective tissue bridge may assist in the aforementioned dural enfolding⁹ and the resulting tension accountable for the thickening of the observed posterior spinal dura.¹⁷ This mechanism could undergo compromise if trauma to the upper cervical spine resulting in rectus capitis posterior minor muscular atrophic changes¹⁸ were to occur. The dura is an extremely sensitive structure, and it has been proposed that adverse tension in the spinal dura can cause cervicogenic headache pain.¹⁹ Hack et al⁹ speculate that the dura-muscular connection transmits forces from the cervical spine joint complex to the pain-sensitive dura. The proposed mechanism of a cervicogenic headache produced from

Table 2. Distribution of the C1-C3 spinal nerves*

C1-C3 Ventral rami	C1-C3 Dorsal rami
Atlanto-occipital joint	C 2/3, 3/4 zygapophyseal joints
Lateral atlanto-occipital joint	Semispinalis capitis, cervicis
Longus capitis	Multifidus
Longus cervicis	Longissimus
Rectus capitis anterior	Splenius capitis
Rectus capitis lateralis	C1-C3 Sinuvertebral nerves
Trapezius	Median atlanto-axial joint
Sternocleidomastoid	Transverse ligaments
Dura mater of posterior fossa	Alar ligaments
Vertebral artery	Dura mater of spinal cord
	Dura mater of clivus
	C2/3 intervertebral disk

From Olesen J. Classification and diagnosis criteria for headache disorders, cranial neuralgias, and facial pain. Copenhagen: International Headache Society; 1990.

*Anatomic sources of cervicogenic headaches.

adverse tension in the spinal dura¹⁹ could further substantiate the role of spinal manipulation as a viable treatment. On the basis of these findings, any pathophysiologic condition affecting the biomechanics of the cervical spine disrupts the balance between cervical spine joint complex stability and mobility. These events create the potential for cervicogenic headache pain.

Recent studies confirm the existence of the dura-muscular connection and make further observations from the conclusions presented by Hack et al.⁹ Rutten et al²⁰ confirmed the dura-muscular connection noted by Hack et al⁹ and subsequently observed separate strands between the dura mater and the posterior wall of the vertebral canal.²⁰ Research has also demonstrated the existence of the dura-muscular bridge in the Visible Human Female cryo-sectioned data set that is located at the National Library of Medicine.²¹ The dura-muscular connection has correspondingly been identified on magnetic resonance imaging scans.²² Mitchell and colleagues²³ observed continuity between the posterior spinal dura and the ligamentum nuchae in the dissection of 10 cadavers. A midline section of the ligamentum nuchae was observed traveling anterior toward the posterior spinal dura connection. This study also confirmed the rectus capitis posterior minor muscular connection to the cervical posterior spinal dura.^{9, 23} Mitchell et al²³ suggested that the dura-ligamentum nuchae connection aids in the spinal dura's resistance to inward folding, particularly during cervical spine extension. Hack replied to Mitchell and associates' finding by agreeing with the suggestion that the dura-ligamentum nuchae connection may contribute to the prevention of dural enfolding during upper cervical spine movements.²⁴ The observations made by Hack et al⁹ and Mitchell et al²³ support Becker's earlier speculation that fascial continuity exists between the skeletal muscle and the dura mater.²⁵

Mitchell et al²³ note the importance of these new anatomic observations in the examination of cervical spine biomechanics and the correlation it permits spinal manipulation practitioners. The correlation is systematically drawn between cervical spine complex dysfunction producing tension on the spinal dura resulting in symptoms commonly

seen in the cervicogenic headache presentation.²⁶ This proposition may affirm the role that spinal manipulation has been correcting the cervical joint dysfunction in the cervicogenic headache patient. Multiple causes of trauma to the cervical spine result in a large percent of patients suffering from headaches and chronic neck pain.²⁷ Understanding the proposed functions of the dura-muscular, dura-ligamentum nuchae connections may assist the physician in interpreting cervical spine joint complex dysfunction as a contributing cause to cervicogenic headache pain.

The convergence of trigeminal and cervical C1-C3 afferents allows the possibility of a cervicogenic headache to result from any pathologic condition affecting structures innervated by the above-mentioned spinal nerves.⁸ Therefore any joint complex dysfunction affecting the C1-C3 spinal nerves has the potential to result in cervicogenic headache pain. The list of structures supplied by spinal nerves C1-C3 is summarized in Table 2.⁸ Several of the anatomic structures listed in Table 2 have experimentally been shown to cause a headache if a noxious stimulus is present. These structures are as follows: the dura mater of the posterior cranial fossa, postvertebral muscles,²⁸ and the zygapophysial joint at C2-C3.²⁹ It has been shown experimentally that only those structures innervated by spinal nerves C1-C3 have the potential capacity for pain referral to the head.²⁸ The role of these structures involved in headache manifestation is implicated by abnormal palpatory and motion palpation findings and the relief experienced by anesthesia of the corresponding joint.⁸

Musculoskeletal pathologic conditions that compromise the cervical spine and spinal nerves C1-C3 include hypomobility or fixation, tender muscle points, and reduced cervical range of motion.³⁰ Muscular hypertonicity, with or without hypomobility, is found to be common in headache patients.³¹ These findings emphasize the importance of evaluating cervical spine movement patterns when treating a cervicogenic headache. Vernon et al³⁰ examined various sources of cervicogenic headaches, including the prevalence of fixations, tender points, and amount of lordosis in the cervical spine in patients with a headache complaint. Approximately 90% of the subjects were found to have hypomobile segments and multiple tender points in the cervical spine. Seventy-seven percent of all participants showed a substantial alteration of cervical lordosis. The findings consisted of reduced, total reduction, or actual reversal of the normal cervical spine curve.³⁰ This study suggested the presence of cervical spine joint complex dysfunction in headache patients, yet failed to use a controlled population. Nagasawa et al³² compared cervical spine radiographic findings of patients with headaches to a control group of nonheadache sufferers. It was found that the cervical spinal curvature index was less in headache patients than in the controls, with many patients having straight cervical curves. The presence of a straight cervical spine may result from a joint complex dysfunction causing excessive contraction of the cervical musculature.³³ This musculoskeletal pathology could be a predisposing factor in headache pain.

Table 3. Studies of the effect of manipulation on chronic benign headaches

Authors	Design	n	HA	Results
Nilsson ⁴⁴	RCT	53	CV	Manipulation had significant positive effect.
Lewit ⁴⁵	CS	41	T	Manipulation was most effective.
Lewit ⁴⁵	CS	93	T	Manipulation was most effective.
Droz and Crot ⁴⁶	CS	332	O	Manipulation was 80% successful.
Turk and Ratkolb ⁴⁷	COH	100	T	Manipulation was 75% successful.
Whittingham ⁴⁸	COH	26	CV	Manipulation had a 77% duration decrease and a 62% frequency decrease
Parker ⁴⁹	RCT	85	M	Manipulation was 28% successful and 47% successful 2 yr later.
Jensen ⁵⁰	RCT	19	PT	Manipulation made a significant difference vs ice.
Jirout ⁵¹	CS	200	T	Manipulation was 80% successful.
Mootz ⁵²	COH	11	T	Manipulation had a significant reduction of frequency and duration.
Stodolny ⁵³	COH	31	M	Manipulation was 75% successful.
Vernon ⁵⁴	CS	33	T	Manipulation was 85% successful, pre/post.
Wight ⁵⁵	CS	57	M	Manipulation was 75% successful.

COH, Cohort; CS, case series; RCT, random controlled trial; HA, headache; CV, cervicogenic; T, tension-type; M, migraine; PT, posttraumatic.

The neurophysiologic and anatomic basis for cervicogenic headaches relies on the convergence within the trigeminocervical nucleus⁸ and perhaps its relationship to the dura-muscular connection.⁹ Injury to the joint complex of the upper cervical spine can refer pain to the occipital region that is innervated by the dorsal rami of C1-C3. Upper cervical spine injury has also been shown to refer pain to those areas of the head innervated by the trigeminal nerve. This occurs as a result of the central processes of sensory cervical nerves C1-C3 that enter the spinal cord at the upper cervical region and converge with neurons of the trigeminal nerve in the trigeminal nucleus caudalis.³⁴ Second-order nociceptive neurons in the trigeminocervical nucleus that receive both cervical and trigeminal input have no means to differentiate their activation from cervical or trigeminal afferents. This allows the potential for neurons that are accustomed to trigeminal input to receive noxious stimuli from a source within the receptive fields of the first 3 cervical nerves.⁸ Central sensitization occurs after tissue injury when nociceptors demonstrate spontaneous activity, lowered thresholds, and increased responsiveness to noxious stimuli, leading to hyperexcitability and an alteration of neuronal processing in the central nervous system.³⁵ Nociceptors possess high thresholds of activation, which means that, under normal conditions, only stimuli that are potentially tissue damaging can depolarize a nociceptor.^{36, 37} Central sensitization is suggested to result from spinal plasticity,^{35, 38, 39} which is an adaptation of the nervous system in response to changes that occur in the internal or external environment.⁴⁰ A sensitized cervical joint complex nociceptor within the trigeminocervical nucleus receptive field could produce the pain associated with a cervicogenic headache.⁴¹ The activation of nociceptive afferents can also produce significant alterations in the receptive field properties of dorsal horn neurons, such as the expansion of receptive field size.⁴² If this occurs, the nociceptive information is transmitted to the thalamus, yet the original source of stimulus is equivocal because the brain, relying on previous input familiarities, cannot discern the origin of the stimulus and may perceive the activation from anywhere within the receptive field.⁸ This mechanism permits the possibility of dysfunction in the

cervical spine joint complex to be interpreted by the brain as noxious input anywhere within the trigeminocervical peripheral receptive field. This neurophysiologic pathway explains the referred cranial pain that is experienced in the cervicogenic headache. Therefore dysfunction of the cervical spine joint complex can lead to pain interpreted as originating from the anterior (CN V, C2 ventral ramus) or suboccipital (C1, C2, C3 dorsal rami) aspects of the head.⁴³

Understanding the suggested neurophysiologic mechanism for the cervicogenic headache allows for a potential correlation to be drawn with the dura-muscular connection observed by Hack et al.⁹ Joint complex dysfunction in the upper cervical spine affecting the dura-muscular integrity may activate nociceptors in the trigeminocervical nucleus receptive field promoting cervicogenic headache pain. Nociceptors in the dura mater⁴¹ could serve as the primary origin of pain in the presence of cervical joint dysfunction.

This proposed cause of the cervicogenic headache allows for the possibility that other chronic benign headaches (eg, migraine, tension type) may possess a predisposing cervical pain source. Cervical joint complex dysfunction present in patients diagnosed with noncervicogenic headache could have the capacity to refer cranial pain that is experienced in migraine and tension-type headache patients.¹¹ The presence of cervical spine joint dysfunction in cervicogenic, migraine, and tension-type headaches may explain the effectiveness of spinal manipulation on chronic benign headaches. The literature to date has examined the effectiveness of spinal manipulation in the treatment of these benign sources of headache. Studies on manipulation in the treatment of cervicogenic, migraine, and tension-type headaches are summarized in Table 3.⁴⁴⁻⁵⁵ The results of these studies suggest the efficacy of spinal manipulation on benign headaches when cervical pathology is present. A conclusion may be drawn that supports the effectiveness of manipulation for benign headaches where functional pathosis exists. No previous etiologic mechanism accounts for the cranial pain perceived in cervicogenic, migraine, or tension-type headache sufferers. However, the anatomic observation of the dura-muscular connection and our neurophysiologic model provides a sound etiologic basis.

CONCLUSION

The literature suggests a neurophysiologic mechanism for headaches in the presence of cervical spine joint complex dysfunction. The dura-muscular, dura-ligamentum nuchae connections in the upper cervical spine may have the potential to produce cranial pain in the presence of functional pathosis. This proposed relationship might aid practitioners in understanding the mechanisms involved in the patient experiencing cervicogenic headache pain. An understanding of this proposed rationale should enable treating physicians to initiate a conservative protocol of spinal manipulation for patients who have chronic benign headaches. This course of treatment has been shown to be an effective, noninvasive management for headaches with cervicogenic, migraine, and tension-type manifestations. However, it is necessary for continued research in this area so that all health care providers consider an initial conservative approach to headache management. Greater emphasis needs to be placed on the identification of cervical spine joint dysfunction in patients initially seen with headache pain. It is also important to classify cervicogenic, tension-type, and migraine headache patients to document more accurately the efficacy of manipulation in each category. The isolation of single headache categories in future studies will help not only to better validate the study but also to aid future practitioners in establishing defined conservative protocols.

It is generally accepted by those in the chiropractic profession that upper cervical spine joint complex dysfunction is a major contributor to headache pathogenesis. It has also been documented that spinal manipulation aids in reducing headache severity, duration, and frequency. It should be the goal of practitioners to reconsider the narrow definition of cervicogenic headache and acknowledge the greater prevalence that cervical spine joint complex dysfunction might play in the cause of all chronic benign headaches. Future research should incorporate means to better define the possible relationship that exists between the dura-muscular, dura-ligamentum nuchae connections and referred headache pain. These efforts will help to expand the circumference of knowledge to those outside the chiropractic community with the ultimate aim of initial conservative treatment protocols for all headache patients.

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