Headaches and the cervical zygapophysial joints

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Introduction

According to the World Health Organization, patients with severe headaches or migraine suffer the same degree of disability as a patient with quadriplegia or dementia. Population-based studies confirm that the impact of severe headaches on the quality of life is substantially burdensome. Severe and persistent pain is often associated with profound personal suffering and the increased risk of suicide is not uncommon. In the Australian context, headaches generate some two million consultations (13%) annually at a cost of greater than $700 million per annum.

Definition

Cervicogenic or cervical headache is pain perceived in the head whilst the primary source resides in any structure of the cervical spine. Cervical headaches are due to a referral of pain from the neck and do not imply nerve compression. Facilitated by a system of neuronal convergence within the upper segments of the cervical cord, cervical or cervicogenic headaches typify a form of somatic referred pain that may originate from the zygapophysial joints, intervertebral discs, muscles, ligaments, or other components of the cervical spine. Hence, unlike migraine or other variants of primary headaches, cervicogenic headache does not represent a distinct neurological entity.

Synonyms

Cervical headache, cervico-encephalic syndrome, cervical spine syndrome, cervicocerebral syndrome, cervical migraine, occipital neuralgia, occipital myalgia-neuralgia syndrome, vertebragenic or spondylogenic headache, post-traumatic muscle contraction headache, residual occipital neuritis, great occipital trigeminus, painful intervertebral dysfunction, cervical occipital pain, neuralgic headache, migraine cervicale, post-traumatic headache, basilar migraine, cervical-occipital neuritis/neuralgia and reflex tension myalgia.

Characteristics

Epidemiology

There is very limited validated epidemiological data on either the incidence or the prevalence of cervicogenic headaches. Non-uniform diagnostic criteria confounded by idiosyncratic ambiguities have severely eroded reliability studies. It is likely that the reported prevalence of 16-60%, irrespective of IASP classification is reflective of the contextual environment within which the patients were reviewed. None of the studies implicated a specific component of the cervical spine as the primary source of nociception.

Scientifically, best and most rigorously studied are the cervical zygapophysial joints. A substantial body of anatomical and pathological data now complement the clinical evidence that implicates the cervical zygapophysial joints as the most common source of chronic post-traumatic headache. By way of comparative diagnostic blocks, specifically investigated the co-existence of headaches in patients presenting with post-traumatic cervical zygapophysial joint pain. In what must be the only published randomized double-blind control study, the C2/3 zygaphysial joint yielded a prevalence of 49%, the C5/6 and C4/5 contributed 13% and 4% respectively, 7% was attributed to the C3/4 and 2% to C6/7. Amongst patients in whom headache was a dominant feature, the C2/3 synovial joint accounted for 53%. These results were replicated in a recent independent study. Where the cervical disc was the putative source, more than 60% of respondents undergoing surgical fusion of the cervical spine reported that headache had been the dominant symptom.

Causes and differential diagnosis

Heterogeneous group

By far the commonest cause is trauma to the cervical spine either recent or remote. Headache may occur predromally in vertebral artery dissection, vascular compression of C2 cervical root, meningioma, cervical cord infarction, spontaneous dissection of the carotid artery, and Chiari type malformation.

Neuralgias

The common perception that occipital neuralgia is due to entrapment of the greater occipital nerve has been challenged. Anatomical studies have disputed the putative pathomechanism and hence surgical extrication of the nerve and its ganglion may not be justified. Unlike occipital neuralgia, however, C2 neuralgia is a distinctive form of headache caused by lesions affecting the C2 spinal nerve (for example, venous or arterial anomalies). A separate disorder, neck tongue syndrome is characteristically recognized by acute unilateral occipital pain associated with simultaneous ipsilateral numbness of the tongue. This
Cervical intervertebral discs

As a source of cervicogenic headache, the intervertebral discs have been implicated in a few observational studies. Following cervical fusion, a proportion of patients reported complete relief of their headaches whilst in others relief had been incomplete.68-70

There are no clinical features by which the disc could be implicated as a source of pain to the exclusion of other structures. As a diagnostic utility, provocation discography lacks specificity,58-61 and has a high false positive rate.62,63 Not only does discography reveal symptomatic discs at multiple levels, it is also uncommon to find discogenic pain limited to a single level.64-65

Muscle pain

Despite the lack of anatomical, histological, biochemical, and electrophysiological evidence,55, 67 notions such as “muscle spasm” and “pain-spasm-pain cycle” have been widely publicized as potential causes of headaches.68-70 Contrary to published evidence,71-73 as a cause of headache, muscle spasm, either as an autonomous clinical entity or as a manifestation of myofascial syndrome, currently enjoys an increasing resurgence amongst practitioners who advocate bacterial toxin as a therapeutic agent.77-79

Several of the tender spots, ascribed to “trigger points” of the myofascial variety, overlie cervical zygapophysial joints.80 Hence, the alleged muscle tenderness or hyperalgesia is most likely a manifestation of central sensitization, with the zygapophysial joints being the continual source of primary nociception.81-86 Consequently, to avoid misdiagnosis and hence inappropriate treatment, the distinction between “trigger points” and painful zygapophysial joints is paramount. In the absence of any plausible biological mechanism, the treatment of “trigger point syndrome” is entirely empirical. Intraleisional administration of local anaesthetic may offer short-term palliation but there are no scientific studies that would attest to its long-term efficacy. A systematic review found that neither “dry” nor “wet needling” – irrespective of the injectate – was better than placebo.87

Zygapophyisal joints

Zygapophysial joints: the evidence

To be a source of headache, cervical spinal structures should
- have a nerve supply
- evoke headaches upon stimulation,
- relieve the headache when anaesthetized
- be ideally described in anatomical, biomechanical, radiological, and pathological terms,58 and,
- treatment should confer complete and prolonged relief of pain.89

Neuroanatomy

Peripheral. Archetypical of somatic referred pain (that is, somatic origin of pain as distinct from visceral origin of referred pain) headache may arise in any of the cervical structures and the pattern of referral is primarily dependent upon the peripheral distribution of the upper three cervical nerves; hence, any structure innervated by the first three cervical nerves can be a potential source. Known sources include the suboccipital and upper posterior neck muscles,90, 91 the intervertebral discs,92, 93 the synovial joints including the C2/3,90,91 the joints and ligaments of the median atlanto-occipital joint and the lateral atlanto-axial joints, 94 the spinal dura mater,95,96 the vertebral artery,24 the C2 nerve root/ganglion,97 the synovium and facet joint capsules,98-102 and structures within the posterior cranial fossa.103 Neurohistochemical techniques have confirmed the presence of small diameter nerve fibres (nociceptors) and mechanoreceptors in the synovium and the synovial joint capsules,98-102 thus providing the neuronal circuitry not only for proprioception but also for the transmission of pain impulses.

Central connections. Fundamental to the genesis of cervical headache is the trigemino-cervical nucleus, a column of grey matter formed by the pars caudalis of the spinal nucleus of the trigeminal nerve and the grey matter of at least the upper three cervical spinal cord segments (Fig. 1).8 The supratentorial pain producing structures are innervated by the first division of the trigeminal (ophthalmic) nerve, the fibres of which pass through the trigeminal nerve root and descend to the level of C1/2 spinal cord segment to synapse on second order neurons in the trigemino-cervical nucleus.104 Infratentorial and cervical structures are largely innervated by the C2 and upper three cervical nerves respectively.8,90,105 The latter in turn synapse over the same range of trigemino-cervical nucleus as the ophthalmic nerve. Prior to entering the substantia gelatinosa, the C2 afferents extend caudally to the third spinal segments and proximally to the pyramidal decussation, whilst the C3 afferents may extend superiorly as far as the corresponding fibres of C2.105 This arrangement allows nociceptive afferents from the trigeminal nerve and from the first three cervical spinal nerves to form multiple collateral channels which converge onto common second order neurons106 and it is this neural
circuitry of “convergence” that facilitates the perception of referred pain. Hence, stimulation of one group of afferents may be perceived as arising from a region innervated by another group. Referred pain therefore is a sensory illusion that results from such convergent circuitry and does not imply spinal nerve compression.\textsuperscript{107} Perceptually the brain is unable to distinguish whether the incoming information from the second order neuron was initiated by the vertebral afferent or other convergent fibres and hence attributes its origin to both.\textsuperscript{108} Spinal referred pain is nociceptive in nature, that is, the pain is initiated by stimulation of nerve endings of afferent fibres and this peripheral stimulation distinguishes it from neurogenic and radicular pain.\textsuperscript{108} Afferent fibres from the region of referred pain are not stimulated by the causative agent.\textsuperscript{108}

Once they have passed through the Gasserian/trigeminal ganglion, trigeminal afferents that convey pain and temperature do not synapse with second order neurons at the pontine level of the brain stem, but instead all three divisions turn inferiorly within the brain stem as the descending tract of the trigeminal nerve and synapse with the second order neurons principally within the upper cervical spinal cord.\textsuperscript{109,110}

Whilst fibres from all three divisions can be found as low as the second cervical segment,\textsuperscript{109} fibres from the ophthalmic division within the pars caudalis can be found as low as the fourth or fifth cervical segment.\textsuperscript{111} Furthermore, cervical afferent fibres from as low as C4 and probably C5 are found in the dorsal horn at the second cervical level.\textsuperscript{106,111}

The ophthalmic division extends most caudally and is most densely represented. Such a distribution facilitates greater convergence between the cervical and trigeminal afferents. Hence, somatic referred pain is more likely to be perceived as frontal headaches.

Although not a distinct anatomical entity, there is electrophysiological evidence that establishes the interaction between the upper cervical dorsal root and the trigeminal system. Neurons in the C1 and C2 segments correspond to a stimulation of afferents in both the upper cervical spinal nerves and the trigeminal nerve.\textsuperscript{112,113}

The spinal trigeminal tract also receives a small complement of fibres from the facial (7th), glossopharyngeal (9th) and the vagus (10th) nerves.\textsuperscript{109} Cumulatively, such an arrangement establishes the trigemino-cervical nucleus as the essential nociceptive nucleus of the head, throat, and upper neck.\textsuperscript{8}

Pain provocation

Patterns of referred pain described in clinical studies have been replicated in normal volunteers. By injecting hypertonic saline into the suboccipital muscles, Cyriax\textsuperscript{114} reported that the pattern of pain referred to the head is contingent upon the site of stimulation: the more cranial the site of injection the closer to the forehead the pain projected, whereas stimulation below the occiput confined projection of pain to the upper end of the cervical muscles. Other investigators reproduced patterns of referred pain in the orbital and frontal regions by either dry-needling the periosteum of the occipital condyle or by injecting hypertonic saline.\textsuperscript{115,116} Stimulation of the greater occipital nerve in experimental animals increased the metabolic activity in the ipsilateral caudal brain stem, upper cervical cord, and in the dorsal horn at the level of C1 and C2.\textsuperscript{117} The neuronal activation appeared contiguous with the pars caudalis and was in the same distribution when trigeminally innervated structures were stimulated.\textsuperscript{118}

Referred pain of a similar pattern can be evoked when certain cervical joints are stimulated. Normal volunteers who had their atlanto-occipital, lateral atlanto-axial,\textsuperscript{119} the C2/3, and their C3/4 zygaphophysial joint capsules\textsuperscript{120} distended with contrast medium perceived pain to originate in the occipital and suboccipital regions. All joint referral patterns were ipsilateral. The C2/3 pattern was distinguished from that of C3/4 by the former’s extension into the head. The onset of pain coincided with capsular distension. Stimulation of the lower zygaphophysial joints failed to refer pain to the head. Fukui et al.\textsuperscript{121} not only replicated the pain pattern first described by Dwyer et al.\textsuperscript{120} in a similar manner, but, in the same cohort of normal volunteers, reproduced the same patterns of pain by electrically stimulating the medial branches of their respective cervical dorsal rami.

Relief of pain

Whilst many of the earlier reports attested to the efficacy of local anaesthetics in relieving headaches ostensibly originating in the cervical spine,\textsuperscript{22} none identified or implicated a specific cervical structure as the primary locus of somatic referred pain. Irrespective of this, the zygaphophysial and atlanto-axial joints were often prime contenders.\textsuperscript{112,125} A semblance of target specificity was reported by Hunter et al.\textsuperscript{126} Patients who had local anaesthetic injected directly onto the second cervical root for suspected occipital neuralgia reported greater relief of pain than those in whom local anaesthetic was infiltrated around the peripheral distribution of the greater occipital nerve.

Fluoroscopy now permits the precise deposition of minute amounts of local anaesthetic either directly into the joints or selectively onto the nerves that innervate them. A number of clinical studies in which headache has been relieved by selectively anaesthetizing the intervertebral joints complement observations in normal volunteers. Identified sources of joint pain include the atlanto-occipital joints,\textsuperscript{119,127} the lateral atlanto-axial joints,\textsuperscript{127,131} and the C2/3 zygaphophysial joint.\textsuperscript{123,135} Similar outcomes were reported in patients presenting with upper cervical rheumatoid arthritis\textsuperscript{136} and osteoarthritis of the atlanto-odontoid joints.\textsuperscript{137,138} A controlled study identified C2/3 zygaphophysial joint to be the most common source of headache after whiplash injury.\textsuperscript{140} Collectively, of all the possible causes of cervicogenic headache the cervical zygaphophysial joints have been most extensively studied and remain the commonest source of cervical pain referred to the head.

Biomechanical aberrations

Evidentiary confirmation that components of the cervical spine undergo considerable kinematical and biomechanical aberrations following simulated whiplash injury have been secured from studies in cadavers\textsuperscript{141,142} and in normal human
volunteers. Rapid sequence cineradiography confirmed that nearly all structures of the cervical spine are subject to a complex array of injurious perturbations long before the protective function of the cervical musculature is called into action. Within a few milliseconds of simulating a rear-end collision, physical forces impacting upon the thoracic spine are cranially deflected. Such a deflection initially causes the lower cervical segments to extend, which in turn cause the upper segments to flex, thus creating an "s-shaped curve".

With continuing application of whiplash loading, the inertia of the head catches up with the cervical spine and "whips" the entire head-neck complex into extension. Compressing the lower zygapophysial/facet joints as it slides anteriorly. The additive effects of simultaneous distraction (avulsion), compression, translation (sliding) and torsion caused the joint capsules and their respective meniscoids; neither these nor the small fractures were identified on imaging (Plate 2).

Pathology

Even with the advent of MRI, certain injuries to the soft tissue elements of the cervical synovial joints may not be overtly evident. Tears of the joint capsules, loss of articular cartilage with eburnation, and proliferation of subchondral bone have been confirmed at operations in symptomatic individuals. The aggregate data from experimental studies, cadavers, radiological and postmortem findings constitute a substantial body of evidence which confirms that the cervical zygapophysial joints and surrounding soft tissues are damaged in whiplash injury. Autopsies on victims of motor vehicle accidents who had died from unrelated causes revealed injuries to the joint capsules and their respective meniscoids; neither these nor the small fractures were identified on imaging (Plate 2). Complementary

Plate 2. Facet fracture not seen on x-rays. Courtesy Prof. J. Taylor, ref. 171.

Rapid sequence cineradiography is invaluable in demonstrating pre-and paravertebral haemorrhage, oedema including cord oedema, occult end-plate fractures, tears of anterior and posterior longitudinal ligaments, and tears of the anterior anulus. The fibres of the anterior longitudinal ligament are firmly fixed to the intervertebral disc and the periosteum of the vertebral body; hence sudden distraction may cause simultaneous avulsive tears to the fibres of the anterior anulus, the anterior longitudinal ligament and vertebral end-plates fractures with intra discal haemorrhage.

Surgical evidence

Surgery for the treatment of cervicogenic headaches has not been subjected to randomized studies. Logistically and ethically, devising such studies would be severely hampered by epidemiological and other constraints. Irrespective of this, a not-too-generous interpretation of the "N=1" study design does allow meaningful information to be harnessed from the available data. As such, the published observational studies are neither distracting nor discouraging. A surgicanoatomico-pathological relationship between the C2/3 zygapophysial joint, the third occipital nerve and occipital headaches was first reported by Trevor-Jones. Patients who had their third occipital nerve surgically decompressed reported complete relief of their occipital headaches. At operation, the third occipital nerve was seen to be entrapped by an osteophytic mass. Similar outcomes were reported by Poletti some 19 years later.

In unrelated studies where occipital headaches were attributed to osteoarthritus of the lateral atlanto-axial joints, a significant proportion of patients secured complete relief of their headaches for as long as seven years following surgical fusion of these joints. In all cases, the suspected anatomical locus of pain was radiologically evident. Presurgically, total relief of the index pain was achieved with intra-articular deposition of local anaesthetic and surgical fusion conferred long-term relief. Clinically, diagnostically, and therapeutically, these outcomes fully satisfy the revised criteria and caveats for the diagnosis of cervicogenic headaches, recently enunciated by the International Headache Society.

Radiological evidence

Plain radiographs are generally insensitive for detecting subtle injuries including osseous lesions. Several retrospective and contemporary clinical studies have verified that fractures of the articular pillars might have been overlooked and hence under-reported. Both conventional and high-resolution computerized tomography is more reliable in detecting occult fractures than plain x-rays.

Improved sensitivity with corresponding reduction in the false-negative rates could be achieved by adopting specific projections, for example, "pillar views". Similarly, reconstructing the injury mechanism with its biomechanical implications may predict the nature of lesions; for example, compressive fractures to the atlanto-axial complex with possible subluxation following acute lateral flexion of the cervical spine.

With the advent of magnetic resonance imaging (MRI), injuries previously deemed elusive and "non-demonstrable" are becoming more evident. Magnetic resonance imaging is invaluable in demonstrating pre-and paravertebral haemorrhage, oedema including cord oedema, occult end-plate fractures, tears of anterior and posterior longitudinal ligaments, and tears of the anterior anulus. The fibres of the anterior longitudinal ligament are firmly fixed to the intervertebral disc and the periosteum of the vertebral body; hence sudden distraction may cause simultaneous avulsive tears to the fibres of the anterior anulus, the anterior longitudinal ligament and vertebral end-plates fractures with intra discal haemorrhage.

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studies confirmed that no tissue structure was immune from injury. Equally, independent observers reaffirmed that in some instances, fractures of the articular pillars, tears of the anterior annulus, bruising of the vascular synovial folds (menisci), haemorrhosis, various grades of vascular injuries, haemorrhage within the C2 spinal nerve and/or the dorsal root ganglion\(^\text{168, 173}\) could neither be confirmed nor discounted either clinically or radiographically.

Current understanding on pain pathophysiology would irrevocably implicate such injuries as potent sources of chronic nociception, capable of generating somatic and neuropathic pain.\(^\text{174, 175}\) There are no valid clinical signs, electrophysiological or radiological abnormalities by which such diagnoses can be refuted. Excessive reliance on x-rays to isolate a pain focus is erroneous and fallacious, and the concept of the so-called ‘nondemonstrable injury’ is best abandoned. Where organicity is elusive or inconspicuous, it behoves every practitioner to exercise considerable restraint rather than to impugn the patient’s veracity and invoke a psychiatric diagnosis of a dubious nature.\(^\text{176, 177}\)

**Diagnosis**

In 2004, the International Headache Society (IHS) revised its diagnostic criteria for cervicogenic headache.\(^\text{180}\) It required, inter alia, that the suspected cervical lesion be identified either by a biological marker or by imaging, and that complete relief of headache be secured by anaesthetizing – either the structure itself or the nerves which innervate it. Prior to its publication, the two major competing philosophies relied entirely on clinical features.\(^\text{178-180}\) Neither has been validated,\(^\text{181-186}\) nor are the features described unique to headaches of cervical origin. Similar features including allodynia\(^\text{187}\) have been described in migraine, tension headache, cluster headaches,\(^\text{181-188}\) and drug-induced headache.\(^\text{184, 188}\) In one study, 44% of patients attending a pain facility had more than one headache diagnosis whilst more than 70% of the cervicogenic group fulfilled diagnostic criteria for migraine.\(^\text{185}\) In sharing the same physiological mechanisms, it is not surprising that primary headaches and headache of cervical origin have similar clinical presentations.\(^\text{186}\)

To date, only controlled diagnostic blocks have been validated by which cervical sources of headaches can be isolated. Local anaesthetic may be injected into the putative pain source, or onto the nerves that innervate it. The primary objective is to locate the source by relieving the pain.\(^\text{40}\) Bogduk and Marsland\(^\text{132}\) were the first to devise and describe the concept of comparative diagnostic nerve blocks. When properly executed, two important variables emerge, namely, the relief of pain and the duration of such relief. Under normal circumstances, injecting a short-acting agent (for example, lidocaine) would relieve pain for a short duration, whilst a long-acting agent (for example, bupivacaine) would relieve pain for a longer duration, a paradigm ratified in subsequent studies.\(^\text{37, 188}\) Under fluoroscopic guidance, minute amounts of local anaesthetic can be injected into any of the upper cervical joints or onto the nerves that innervate them (Plate 3).\(^\text{37, 188}\) To minimize false-positive responses or other placebo effects, the use of controls is inescapable.

Neither the history, physical examination nor the imaging studies can distinguish between the various sources of cervical pain. Comparative diagnostic block is the only valid modality that can precisely localize the cervical source of pain,\(^\text{190}\) including the atlanto-occipital joints,\(^\text{115, 123}\) the lateral atlanto-axial joints,\(^\text{115, 127-130}\) the C2 spinal nerve in C2 neuralgia,\(^\text{53, 191}\) and the C2/3 zygapophysial joint.\(^\text{132, 133}\)

There are limitations. Not every structure is accessible to diagnostic blocks. False positive responses generated by patients’ expectations or physician bias can be avoided by having the patient assessed by an independent observer who is blinded to the nature of the injectate. Some patients have a paradoxically long response to short-acting anaesthetics; for example, with lignocaine pain relief may last for days.\(^\text{192}\)

**Treatment**

**Physical therapies**

Although routinely prescribed and generously administered, physical modalities such as ultrasound, laser, or interferential do not exert any lasting therapeutic effect. The biological mechanism by which these modalities putatively operate awaits scientific validation, but what does emerge consistently is that these modalities are no better than placebo.\(^\text{193, 194}\)

**Manual therapy**

For manual therapy, the evidence is inconclusive, ambiguous, and inconsistent. Studies are replete with methodological shortcomings.\(^\text{195}\) One study reported that in the short-term manipulation was more effective than massage in reducing the frequency and severity of headaches,\(^\text{196}\) and when combined with a non-steroidal anti-inflammatory, the modest gains did not extend beyond two weeks.\(^\text{197}\)

More recently, a controlled trial of six weeks’ duration randomly assigned participants to receive manipulative therapy, specific exercises or both.\(^\text{198}\) A week after completion, 76% reported at least 50% reduction in headache frequency.
and 35% reported a complete relief of headache. Although at 12 months 72% reported at least a 50% reduction in headache frequency, 24% sought additional or alternative treatment during the study period and 25% failed to achieve any benefit. The physiological mechanism by which improvement was secured was not explained. Claims that spinal manipulation is an effective treatment for cervicogenic headaches is undermined by considerable methodological limitations and it remains unclear to what extent the reported positive effects can be explained by manipulation or other non-specific factors.

Injection therapy

Diagnostic

Comparative diagnostic blocks may assist in isolating the source of pain and hence in establishing an anatomical diagnosis. In certain instances, blocks may determine the nature of treatment, predict its outcome and test the validity of clinical inferences.

Therapeutic

Local anaesthetics. That local anaesthetics can confer substantial and long-lasting relief has been widely reported, but formal studies validating its adoption as a primary therapeutic agent are wanting. Irrespective of this, the relief of headache by peripheral nerve blocks not only questions the legitimacy of neuronal convergence but also imputes a newer and different but undefined neuromodulatory effect.

Steroids. For occipital tenderness, steroids have been injected pericranially, ostensibly for its anti-inflammatory effect. However, the evidence for an inflammatory reaction has not been demonstrated. In-vivo studies have confirmed that tender points lack the typical inflammatory mediators and metabolites and are not sites of ongoing inflammation. Intra-articular steroids on the other hand are target specific and seem to exert a modest but appreciable beneficial effect. A small-uncontrolled study showed that patients who suffered chronic daily headaches, and in whom diagnostic blocks were positive, steroids resulted in total abolition of their headaches in 11% for as long as 19 months. In a further 50%, frequency of headaches was reduced to three per month, and these responded to oral analgesics.

From a biological and epidemiological perspective, a number of confounders negate the use of epidural steroids either diagnostically or therapeutically.

Botulinum toxin. Its mechanism of action is dissonant with the known pathophysiology of cervicogenic headaches. For the treatment of cervicogenic headache there are no supportive data.

Prolotherapy. There are no data specifically for the management of cervicogenic headaches.

Radiofrequency neurotomy

As a therapeutic agent, thermal radiofrequency neurotomy (RFN) creates a mechanical barrier to the transmission of pain impulses emanating from a known source of pain. It achieves the desired effect by coagulating the nerve/s which innervate that structure. Thermal RFN is not cautery. It involves placing the exposed tip of a Teflon-covered electrode adjacent and parallel to the targeted nerve. High frequency low energy electricity conducted through the electrode generates heat in the surrounding tissues, which in turn coagulates the nerve. Radiofrequency does not offer a permanent cure. After a while, the nerve regenerates and pain may return. Relief may be reinstated by repeating the procedure. It is thus far the only established valid means of conferring complete relief of pain for a durable period.

One form of cervicogenic headache where RFN has been most successful is in the treatment of third occipital headache. Third occipital headache or the headache of Bogduk is so named because the headache perceived is transmitted by the superficial branch of the C3 dorsal ramus, the third occipital nerve. This nerve crosses the lateral and posterior aspect of the C2/3 zygapophysial joint and furnishes articular branches to the underlying joint. It is the sole source of innervations of this joint. Anaesthetizing the third occipital nerve with minute aliquots of local anaesthetic can be used as a test for pain emanating from the C2/3 zygapophysial joint. Unlike the dorsal rami at lower levels, the third occipital nerve is a large single nerve and it is the only dorsal ramus that crosses the joint line. The C2/3 zygapophysial joint has been shown to be the commonest source of post-traumatic cervicogenic headache.

There are no clinical features by which pain from the C2/3 joint can be diagnosed and diagnostic blocks are the only means by which a diagnosis could be secured with certainty.

For the treatment of third occipital headache, RFN offers the best-published outcomes (Plate 4). When meticulously executed, complete relief pain can be achieved in at least 88% of patients, in whom the medium duration of complete relief was 297 days, with some patients reporting continuing relief at the time of review. Once the nerve regenerates and

Plate 4 – electrode placement for RFN
should pain return, the procedure could be repeated and relief reinstated. In some patients in whom the procedure was repeated, the sustained relief of headache was achievable for more than two years. Unlike the primary headaches, third occipital headache is one form of cervicogenic headache that can be diagnosed with certainty and for which valid treatment is available. The cardinal indication for the procedure is complete relief of pain following controlled diagnostic blocks, which makes RFN a logical procedure.

**Conclusion**

Amongst the reasons headache treatment fails, incorrect diagnosis, physician bias and the failure to recognize secondary disorders predominate. Cervicogenic headache is not a neurological disorder. It is one form of somatic referred pain (sensory illusion) and not unlike arm pain associated with cardiac ischaemia. It has a distinct pathomechanism. This convergence of cranial and upper cervical afferents into a common synaptic region provides a logical explanation for the phenomenon of referred pain in the head and neck. Primary headaches (for example, migraine) on the other hand are driven from the brain, and the headache perceived reflects the interaction between nerves and blood vessels. Such neurovascular headaches have a unique pathophysiology and their clinical manifestations including contra-lateral limb allodynia point to a higher neuronal involvement.

Given this fundamental difference, the insistence that cervicogenic headache be validated by objective physical signs, electrophysiological and/or radiological abnormalities is spurious and no longer tenable. Morphological changes seen on imaging studies correlate poorly with symptoms and this inconsistency has been replicated.

Compared with primary headaches, cervicogenic headaches and in particular the third occipital headache is best understood anatomically and physiologically. A common disorder, contemporary evidence implicates the cervical zygaphysical joints as the most common source of post-traumatic headaches. Similar data for intervertebral disc, muscles, and other structures that constitute the cervical spine are not available. Cumulatively, data derived from experiments, clinical observations, radiological and post-mortem studies are irrefutable. Clinical features are neither valid nor reliable and as a diagnostic criterion, only controlled diagnostic blocks have survived scientific scrutiny. To ignore the evidence is to deny patients legitimate and valid treatment. It is likely that such iatrogenic behaviour may invoke certain legal imperatives.

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Headaches and the cervical zygapophysial joints


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