

Plenary Session on Headache

Cervicogenic headache

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That disease or dysfunction of the neck may present as headache is an idea that is not widely accepted in North American traditional medicine. This review focuses on 'cervicogenic headaches'. Topics include the mechanisms of cervicogenic headache, cervical signs to suspect that a patient's headache originates in the neck, diagnostic manoeuvres that are nonspecific and unreliable, laboratory tests that may assist in establishing a diagnosis of cervicogenic headache and treatment of this condition.

Key Words: *Cervical spine, Cervicogenic, Headache*

Céphalée cervicogène

RÉSUMÉ : Qu'une maladie ou une dysfonction du cou puisse se présenter comme une céphalée est une idée qui ne fait pas encore l'unanimité en médecine traditionnelle nord-américaine. La présente revue se concentre sur « les céphalées cervicogènes ». Les sujets traités portent notamment sur les mécanismes de la céphalée cervicogène, les signes cervicaux permettant de soupçonner que la céphalée d'un patient a son origine dans le cou, les manoeuvres diagnostiques qui sont non spécifiques et douteuses, les épreuves de laboratoire qui peuvent aider à établir un diagnostic de céphalée cervicogène et le traitement de cette affection.

The idea that disease or dysfunction of the neck commonly presents as headache is an old one that has yet to gain wide acceptance in North American traditional medicine. The publications in the European literature by Sjaastad and colleagues (1), and their statements that 'cervicogenic headaches' are a major entity that is frequently misdiagnosed, prompt a re-evaluation of this concept.

MECHANISMS OF CERVICOGENIC HEADACHE

For neck disorders to cause headaches, there are three necessary conditions (2): there must be pain-sensitive structures in the neck; there must be identifiable pathways and mechanisms through which nociceptive impulses arising in the neck can be referred to the head; and there must be identifiable pathological or physiological processes that stimulate the pain-sensitive structures in the neck.

The first two conditions are easily satisfied. There are many pain-sensitive structures in the neck, including the uncovertebral joints of the cervical spine, the synovial joints of the occipito-atlanto and atlanto-axial junctions, the annulus fibrosus of the intervertebral discs, the spinal ligaments, the periosteum of the

vertebral bodies, the cervical muscles and their attachments to bone, the cervical nerve roots, and the vertebral and carotid arteries. There are many mechanisms through which nociceptive impulses arising from these structures may be referred to the head including the following.

- The C2 sensory root and its extensions, the occipital nerves, supply sensation to the back of the head, so that high cervical lesions may produce posterior headache.
- Although sometimes said not to exist, there is a C1 sensory root that supplies sensation to the orbitofrontovertex region (3).
- Tentorial nerves serve as anastomoses between the trigeminal nerve and C2 in the posterior fossa (4), and when stimulated by posterior fossa lesions may refer pain to V₁.
- The spinal tract and nucleus of the trigeminal nerve descend to the level of C2 to C4, where they are in continuity with the dorsal grey column of the upper cervical cord; stimuli applied to the upper cervical nerve roots can excite potentials in the trigeminal apparatus (5).

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There are some convincing examples, and many less well substantiated hypotheses, of diseases or dysfunction of the neck producing noxious stimuli that can be translated into headache.

Congenital anomalies of the craniovertebral junction

Congenital anomalies of the craniovertebral junction regularly produce headaches, which are typically occipital but can extend bifrontally. Such anomalies include basilar invagination, congenital atlanto-axial dislocation, separate odontoid process and occipitalization of the atlas. These bony anomalies may coexist with 'soft tissue' anomalies such as hydrocephalus and Arnold-Chiari malformation, which may themselves cause headaches. McRae (6,7) reported unilateral or bilateral occipital or suboccipital pain as the presenting complaint in 26% of patients with bony craniovertebral malformations. Typically, the pain was described as 'bursting', was triggered by flexing the neck or bending forward and was eased by lying down. Vomiting could occur. Mechanisms of head pain may include stretching of the upper cervical nerve roots, tonsillar descent with the production of pressure gradients between the intracranial and intraspinal compartments, and hydrocephalus.

Acquired lesions of the craniovertebral junction

Acquired lesions of the craniovertebral junction also are regularly associated with suboccipital or occipital pain that may spread bifrontally. Such lesions include meningiomas of the foramen magnum, neurofibromas, and ependymas of the upper cervical region, all of which may stretch the upper cervical nerve roots. Lesions of the skull and spine proper, such as multiple myeloma, metastatic cancer, osteomyelitis, Pott's disease and Paget's disease, produce periosteal pain, and Paget's disease may also lead to basilar invagination which can cause headache either by traction on the upper cervical nerve roots or via hydrocephalus.

Rheumatoid arthritis and ankylosing spondylitis of the cervical spine (8,9) can cause atlanto-axial subluxation with stretching of the upper cervical ligaments and nerve roots, producing occipital headache that can radiate anteriorly. These conditions also may result in inflammation of the upper apophyseal joints; the pain from this tends to remain occipital rather than being projected frontally.

There is a close, credible relationship between each aforementioned condition and headaches. These lesions are easily demonstrable, and their treatment usually ablates the headaches. In the following conditions, however, the relationship with headaches is more tenuous, either because no lesion or dysfunction of the cervical region can be convincingly demonstrated or because the lesion demonstrated (eg, cervical spondylosis) is so common that it is considered usual even in people without headache.

Cervical disc disease and cervical spondylosis

Cervical disc disease and cervical spondylosis are regarded by some to be common causes of headache (10), and by others to be 'innocent bystanders' in people who have headaches from other causes such as migraine or tension (11). Most of the population over the age of 40 have radiological changes of

cervical spondylosis; few have symptoms (12). When disc disease is symptomatic, in terms of producing brachialgia or myelopathy, headache may be an associated symptom in 25% of such patients (13). On the other hand, that study did not control for the fact that at least 25% of the general population have headache. Physiologically, it would be difficult for a condition affecting predominantly the lower cervical spine (C5 to C7) to involve the upper cervical roots (C2 to C3) to refer neck pain to the head.

Trauma, including whiplash injuries of the neck, is believed by many to be among the most common causes of chronic headache (14). This is impossible to disprove. It is a rare patient who cannot summon a history of at least a minor injury at some time, especially when interrogated by someone determined to establish a traumatic basis for the symptoms. Clearly, many people with an extension-flexion injury of the neck experience transient nuchal, occipital and sometimes frontal pain, lasting hours to days; likely, this is produced by stretching and spraining of cervical ligaments and muscles. What causes the more prolonged and intractable headaches that a minority of whiplash patients suffer is a controversial issue. The polar positions are that, on the one hand, neurosis, greed and lawyers are responsible, and, on the other, that as yet undemonstrable physical processes ranging in location from the brainstem to the neck muscles produce organic headaches. The recent review by Barnsley et al (15) gives a balanced view.

Posterior cervical sympathetic syndrome of Barré and the cervical migraine syndrome of Bartschi-Rochaix

The posterior cervical sympathetic syndrome of Barré (16) and the cervical migraine syndrome of Bartschi-Rochaix (17) are closely related concepts that have not gained wide acceptance outside Europe. In the first, symptoms of headache, neck discomfort, dizziness, visual blurring, impaired hearing and psychological disturbances are believed to be caused by osteophytes irritating the sympathetic nerve plexus that invests the vertebral arteries. In the second, direct involvement of the vertebral artery by osteophytes is postulated, with the same symptoms resulting. In both, trauma is believed to be a precipitant. Acceptance of the Barré syndrome is made difficult by the failure to demonstrate that stimulation of the sympathetic system indeed causes such symptoms. While it is true that vertebral artery occlusion by atherothrombosis or its involvement in dissection may produce neck and head pain and other symptoms (18), no vertebral artery pathology has ever been detected in the Bartschi-Rochaix syndrome.

Cervicogenic headache

'Cervicogenic headache' is a term coined and popularized by Sjaastad et al (1) and Fredriksen et al (19) to denote a clinical headache profile that suggests that the headache emanates from dysfunction in the neck. This profile includes infrequent, non-clustering, long-lasting attacks of unilateral headache of moderate severity, with associated features suggesting neck involvement (such as a prominent posterior component provocation of attacks by neck movements, and ipsilateral neck, shoulder or arm pain), and other features such as nausea, vomiting, phonophobia, irritability, dizziness, visual blurring on the same side as

the headache, lacrimation and conjunctival injection. Fredriksen and co-workers (19) reported the presence of trigger points in the neck which, when stimulated, occasionally or consistently reproduce the patients' symptoms; they also noted relief in many cases from C2 blockade with local anesthetic.

Many patients with this cervicogenic headache profile give a history of head or neck trauma, often occurring in temporal proximity to the onset of the headaches. However, in one series (20), the cervical spine x-rays of 15 patients with cervicogenic headaches were compared with those of 18 age- and sex-matched, headache-free controls; no differences were found.

Third occipital headache

Bogduk and Marsland (21) postulated a 'third occipital headache' that could be a variant of cervicogenic headache. In a series of patients with occipital and suboccipital headache with frontal radiation and with associated features suggesting neck involvement (history of neck injury, precipitation of pain by neck movement or neck tenderness), the third occipital nerve was blocked – headache relief was seen in about two-thirds of the patients. In the other one-third, headache was not relieved by injection of C3 bilaterally or of C2 and C4. On subsequent occasions, those patients who obtained temporary relief from the C3 blocks failed to get relief from blocks of other cervical nerves. Bogduk and Marsland believe that these headaches may be caused by disease, perhaps traumatic, of the C2 to C3 zygapophyseal joints. In none of these patients, however, was there any radiological evidence of C2 to C3 arthropathy. The possibility that third occipital nerve blocks relieve headache by interrupting normal impulses to the nucleus caudalis trigeminalis, thus reducing the firing frequency of that nucleus (which has been implicated in the genesis of migraine and tension-type headaches), appears not to have been given much weight.

The more enthusiastic proponents of cervicogenic headache maintain that it is extremely prevalent, and that many cases are misdiagnosed as 'common migraine'. Given the close similarity (or identity) of the two clinical profiles, are many cases of common migraine misdiagnosed by enthusiasts as cervicogenic headache? How frequent is cervicogenic headache? Nobody really knows, and until there is a consensus about diagnostic criteria we are not likely to find out.

LOOKING FOR CERVICOGENIC HEADACHE

When should we suspect that a patient's headache originates in the neck? These seven cervical signs, elicited from history and examination, may be useful: occipital/suboccipital headache, especially if consistently unilateral; headache precipitated, worsened or relieved, consistently, by neck movements; abnormal postures of the head or neck; suboccipital/occipital tenderness, especially if consistently unilateral; consistent limitation of neck movements; abnormal craniocervical mobility; and occipital sensory, medullary, upper cord or upper cervical root signs.

Certain diagnostic manoeuvres are nonspecific and unreliable:

- identification of 'nodules' or 'trigger points' is subjective; some examiners find them all the time, and others not at all;

- the 'pinch and roll' manoeuvre, while visually impressive, requires validation in a controlled setting by blinded examiners;
- assessment of abnormal mobility, especially excessive segmental mobility, is a very subjective exercise;
- responses to heat, cold, massage, 'stretch and spray', manipulation, immobilization and traction are all subjective and should not be regarded as diagnostic criteria;
- improvement or disappearance of symptoms following injection(s) of cervical nerve(s) with local anesthetic and/or steroid is not diagnostic. Where 'convergence' is playing a role in the propagation of pain, interruption of normal impulses to the site of convergence may reduce activation of that site to the point where symptoms subside. Recall that Anthony (22) demonstrated that injection of the occipital nerves will help cluster headache, a condition in which cervicogenic mechanisms are not thought to be present.

Certain laboratory tests, carefully done and expertly interpreted, may assist in establishing a diagnosis of cervicogenic headache:

- plain x-rays of skull and cervical spine to show craniovertebral junction, with open mouth view of odontoid, and with oblique and flexion-extension views of cervical spine;
- computed tomographic scanning of base of skull and craniovertebral junction;
- magnetic resonance imaging of posterior fossa, cervical spinal cord and investing structures;
- occasionally, contrast myelography, especially in combination with computed tomography;
- occasionally, electrophysiological studies including electromyography, nerve conductions and evoked potentials from upper cord and brainstem.

TREATMENT OF CERVICOGENIC HEADACHE

Cervicogenic headache – which, in its broadest sense, includes the relatively few patients in whom a lesion has been conclusively demonstrated – is managed by treating the lesions in whatever specific surgical or medical modality is available.

For the very much larger group of patients with cervicogenic headache in whom a cervicogenic basis may be suspected but cannot be demonstrated, treatment is perforce 'empiric', ie, whatever works. While none of these modalities is specific or validated, some appear to work in some people some of the time. These include local heat and/or cold, massage and gentle manipulation within a physiological range (including muscle stretching). Prolonged immobilization likely should be avoided because it may culminate in neck muscle atony with the production of subsequent musculogenic pain. Strategic nerve blocks are acceptable for providing temporary benefit but they should not be given frequently (especially if steroids are being used), and both

the patient and the physician should be aware that this modality is only a nonspecific, symptomatic measure. Destructive procedures, such as C2 ganglionectomy or section of cervical nerves, should not be considered standard therapy until their merits have been established by large scale, rigorously controlled trials.

The value of nonsteroidal anti-inflammatory drugs and tricyclic antidepressants in cervicogenic headache has not been assessed in controlled clinical trials, but until these studies are done they may be carefully employed. As in any chronic head pain,

analgesics, imprudently prescribed or carelessly consumed, pose a risk of rebound headaches.

Finally, for many of these cervicogenic headache patients the prognosis is fairly good provided that the physician refrains from treatments such as excessive analgesics, vigorous manipulation, prolonged immobilization, overly frequent injections of anesthetics or corticosteroids, and surgical sections, all of which may worsen the situation. In cervicogenic headache, as Voltaire said, "The wise physician engages the patient while Nature effects a cure".

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