Cervicogenic headache: Current concepts of pathogenesis related to anatomical structure

T.A. Fredriksen, O. Sjaastad

ABSTRACT
A brief summary will be given of the putative etiological factors/topography that so far have been discussed in the context of cervicogenic headache (CEH), in approximately sequential order. Some of these factors have some basis in clinical research and practice; others are more speculative.

Introduction
Evidence for a cervical origin of headache would be: I) provocation by external pressure in the neck or by neck movements, II) transitory relief by the administration of local anaesthetics, and III) the subsequent, permanent alleviation of pain by surgery, directed towards structures in the neck.

CEH is a multifaceted syndrome in which probably different pathogenetic mechanisms result in the “same” syndrome - or at least apparently so. No single etiologic mechanism has been demonstrated to underlie CEH. The pathophysiology probably resides in different anatomical structures, and there may be more than one abnormality in a single case. Our current, very limited knowledge does not allow a complete understanding of the pathogenesis, let alone the pathophysiology, of CEH. It should be emphasised that the descriptions of head and neck pain in most of the works referred to here is rather superficial. There may be more similar similarities to CEH but, as stated time and again, similarity does not mean identity.

Various proposed pathogenetic models
The first structure brought into discussion in connection with headache stemming from the neck was the “vertebral nerve” by Barré (1); this line of approach was later followed by Lieou (2). Barré coined the term “syndrome sympathique cervicale postérieur”, stating that “irritation” of the cervical sympathetic vertebral nerve gave rise to the symptoms. The existence of such a nerve could not later be confirmed (3). There is, however, a rich supply of sympathetic fibres - a plexus - around the vertebral artery. Stimulation of this has been claimed to give rise to headache (4). Bogduk, however, for what it is worth in this context, found only a minor contraction of the artery itself upon stimulation in the monkey (3).

The vertebral artery was also brought into focus by Bärtschi-Rochaix (5), who in 1949 introduced the term “migraine cervicale”. He started his work in this field by studying headache after trauma, but ended up in 1968 stating that the headache was the result of degenerative changes in the spine (6). It was originally considered that a change in vertebral artery flow could give rise to headache. In later works, Grønbaek (7) carried out a liberation operation of the vertebral artery by uncoforamenectomy. This operation has also been advocated by other researchers, such as Kehr et al. (8). The headache described by these authors has many traits in common with CEH. One possible discriminatory feature in our view may be a more prominent dizziness (even in the form of true vertigo) in their cases, and also drop attacks. We have actually not observed such symptoms in our CEH patients, and this also goes for other recent, large series. The vertebral artery has in addition been of interest in Hauge’s work (9). He showed that the irritating contrast media of the period were able to produce headache similar to CEH, but sometimes with teichopsia (10).

Hunter & Mayfield (11) brought the upper cervical roots into focus in a series of 11 patients who had unilateral headache with traits possibly consistent with those of CEH. These patients were treated with avulsion of either the greater occipital nerve, a section of the sensory root of C2, or the intraspinal section of the
sensory roots of C2 and C3. They reported excellent results in those patients in the group who seemed to have a traumatic background for their headache. More recently, much emphasis has been placed on the 3 upper cervical roots as mediators in headaches originating in the neck. Kerr demonstrated the convergence of trigeminal and dorsal root afference in the upper cervical cord (12). This convergence of fibres suggested a basis for hemicranial pain spreading from the cervical to the trigeminal area (the “Kerr principle”). According to Kerr, this further suggests a reflex pathway that could account for the head turning in response to trigeminal stimuli. Kerr (13) further showed in a study in humans that stimulation of the fine filaments composing the dorsal root of C1 consistently produced pain in the head. C1 usually consists of 3 rootlets. Stimulation of the uppermost rootlet produced pain in the orbit. Mid-rootlet stimulation produced pain in the frontal area, while stimulation of the lowest one resulted in pain in the vertex area. Jansen et al. (14) later showed that irritation of the C2 and C3 roots might be responsible for unilateral headache with traits of CEH. They demonstrated the existence of “venous lakes” around the nerves in these patients and considered that these lakes could exert mechanical pressure upon the nerve structures, secondarily leading to pain. An initial therapeutic approach was, therefore, to remove these venous lakes. However, the beneficial results were of short duration, for which reason Jansen later conducted ganglionection. Many researchers have concentrated on the upper cervical roots and their peripheral extensions, namely the ocipital nerves in CEH. There is some evidence that external pressure upon the major and minor occipital nerves (entrapment mechanisms) may contribute to the pain condition in CEH and that liberation of the nerve(s) can alleviate the pain for a period of time in some patients (15), and possibly permanently in others (U. Rossi, personal communication). It should in this context be emphasised that CEH is a syndrome and not a disease. Most attention has been given to the greater occipital nerve (GON), where it passes through the tendon insertion of the trapezius muscle (16). The third occipital nerve is primarily the dorsal ramus of the C3 root, and it passes directly over the zygoapophyseal joint at the C2-C3 level. Trevor Jones (17) in 1964 showed that this nerve could be encroached upon by spondylotic spurs at the joint. The headache which he described might possibly fit the definition of CEH. Bogduk & Marsland later did blocking experiments and concluded that in 7 out of 10 consecutive patients the pain stemmed from the C2-C3 zygoapophyseal joint per se, being mediated by the third occipital nerve (18). There are, however, relatively massive reservations to be made to sweeping conclusions such as these. Cloward (19), describing his findings in 114 cervical discographies, demonstrated that anterior and posterior midline stimulation - or stimulation close to the midline - could produce more diffuse shoulder/arm pain, similar to the upper extremity pain that has been observed in CEH (20). Part of the CEH symptomatology could possibly be explained on this basis. There also seems to be evidence that this pain syndrome in some cases can be produced at levels lower than C3. The multilevel innervation of the disc and the joint could theoretically be responsible for the spreading of pain from lower levels, and secondarily - to the trigeminal area via the “Kerr principle” (see above). Signals of low cervical origin could in this way cause an illusion that the pain stems from a level as far up as C3. Possibly, there is also a more direct route, in that the tractus spinalis n. trigemini might be extended further down than to the usual C2 level. There are observations that spinal surgery in the form of Cloward or Robinson Smith operations can alleviate CEH with a pathology residing as far down as C7 (J. Jansen; H. Blume, personal communications; and personal experience). There is also evidence suggesting that the head pain could be produced at the disc level itself; it can, furthermore, result from encroachment upon the dorsal root by the disc. The first researchers who pointed to the disc as a possible head pain-generating factor were Raney & Raney (21). Their headache was, however, far less sharply drawn than the one outlined by Hunter & Mayfield (11). The facet joint has been the focus of considerable interest as a source of CEH pain in recent years, the pain putatively being mediated by the dorsal root. Many researchers have advocated radiofrequency (RF)-procedures directed to the facet joints and preferably at several levels simultaneously, since a “summation effect” has been considered to play a role (e.g. M. Sluijter, personal communication, 1998). Muscle tendinous insertions have also been a focus of interest for many researchers. The Blume operation (22) is an RF-procedure directed towards the planum nuchale. The theoretical background for this procedure has been to interrupt the firing from C fibres in the tendon areas. Some excellent results have been obtained in a long-term follow-up study (23).

Conclusion
Recent neurophysiological studies indicate that both CEH and cluster headache have a higher temperature threshold at various cephalic points than controls. The interpretation of this finding is critical; it may even point to central sensory derangement. A possible abnormality in thermoperception in the C2-C3 dermatomes has also been demonstrated on the affected side in CEH patients (24). In summary, one must conclude that none of the proposed pathogenetic mechanisms in CEH can be entirely ruled out. CEH is a syndrome, and in correspondence with this view, the underlying pathology will differ and as a result so will the therapeutic approach. A combination of pathological features may even be present in the single patient. All the proposed abnormalities are related to the neck, which remains the common denominator.

We are still faced with the following structures of possible pathogenetic/etiological importance in CEH:

1. The vertebral artery and its sympathetic nerve plexus.
2. The dorsal roots from C1 to C7.
3. The intervertebral discs down to C7.
4. The zygoapophyseal joints from C2-C3 down to C6-C7.
5. The peripheral nerves in the head/neck.
area, especially the major and minor occipitalis nerves, the third occipital nerve, and the major auricular nerve.

References