Opinion

Occipital nerve block in the management of headache and cervical pain

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Cephalalgia


The origins of chronic headache and the role of the greater occipital nerve in headache syndromes are reviewed. The anatomical pathways and physiological basis of these headaches are discussed with a view to synthesizing some current concepts of headache generation. Studies of occipital nerve blockade for treatment of headaches of various types are assessed and a retrospective analysis of our own experience is presented. □ C1 ganglion, cervical, cardio-trigeminal relay, chronic, cluster, headache, local anaesthetic, occipital nerve block, occipital neuralgia, neurogenic inflammation

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The International Headache Society (IHS) (1) classification of headache is based on clinical phenomenology. Definitions are derived from descriptors on the possible assumption that common presentation defines a valid pathological entity. Migraine and tension-type headache for example have a number of common features. In this review we highlight common features of certain headache types from which no pathophysiological references are drawn but which can be interpreted to advantage in their management. A specific treatment modality, namely repetitive occipital blockade, will be assessed in the light of these features.

Clinical observations

If the symptoms of some, but not all patients are followed over time, an evolution of headache type may occur (2). Whether this implies a change in the central generators of the headache or a change in end organ receptivity is not certain, nor can we speculate on this here. In many patients some of the pain of headache is experienced in the occipital area. This is true both for migraine and so-called tension type headache. In some patients the headache evolves, starting with symptomatic and unequivocally migrainous attacks with throbbing hemieral pain, changing to a more occipital position with a less throbbing quality. Anthony (2) postulates that a gradual change occurs which leads to irritation of the neural structures of the upper cervical spine. Irritation of the structures of the upper cervical spine has long been recognized to cause pain referred to the anterior parts of the head. There is a body of evidence that physical irritation of both the greater occipital nerve and the C2 and C3 roots can cause hemieral pain to occur steadily (3, 4). Sjaastad was probably among the first to describe so-called cervicogenic headache (5). In his paper he discussed strictly unilateral pain starting in the neck and often accompanied by conjunctival injection, lacrimation and lid oedema. Migraine-like symptoms such as ipsilateral visual blurring, as well as dizziness, were also noted. Pfaffenrath (6) described 15 such cases. These patients had occipitally based pain which could be triggered by pressure on the paracervical structures and/or the greater occipital nerve. The symptoms are given in Table 1. The patients were treated with C2 blockade, which provided relief for varying amounts of time. In 5 of the 15 patients, pain was relieved from between two and six weeks.

As in many headache syndromes, the closer one looks at the clinical features the more difficult it is to confine them to one definition. Since none of Pfaffenrath's cases had any radiological abnormalities, it is difficult to say that they were indeed derived from cervical pathology. True, they began in the neck and the greater occipital nerve was tender, but, as Anthony pointed out, many typical migraine sufferers exhibit these features.

Cluster headache is a clearly defined entity characterized by episodic unilateral retro-orbital pain, rhinorrhea, ptosis and miosis. The cranial occipital junction may be tender on the affected side of the head. Some patients describe an evolution of the headache, starting in the back of the neck moving to behind the eyes, with a typical cluster attack.

The headache of the post-traumatic syndrome is a poorly defined entity. Usually the pain is occipital and precipitated by movement of the head or neck.
About half the patients with migraine also exhibited cervical tenderness in the post-traumatic group. Numbers, showed a trend towards there being more symptoms and signs in 50 successive post-traumatic and associated with nausea and vomiting; in other words, migraine (7). We recently compared ten patients with migraine, "cervicogenic" headache, cluster headache caused by arterial malformations (19) and tumours (20). Although different features of migraine, "cervicogenic" headache, cluster headache and post-traumatic headache attest to separate causes, possibly these headaches may constitute different modes of expression of which the sensory network is capable, each of which can be preferentially triggered by specific circumstances. Thus, the neck should be carefully examined in cases of headache where a cervical origin is suspected (21), but failure to find pathology does not mean that the cervical nerves are uninvolved.

The occipital nerve

Because blockade of the occipital nerve is a treatment modality under discussion, the anatomy will be reviewed briefly. The greater and lesser occipital nerves are the second pair of cranial nerves. They originate from the occipital part of the brainstem (C1-Th2) and course through the cranium to innervate the dura mater and the vertebrobasilar arterial tree. The greater occipital nerve (GON) is the larger of the two and innervates the occipital, nasopharyngeal, and middle cranial fossa regions. The lesser occipital nerve (LON) innervates the cervical and posterior parietal regions. Both nerves are accessible for blockade, usually at the upper cervical level (C2-C3).

The pathogenesis of head pain

Although the IHS classification of headache (1) defines and categorizes each headache sub-group clearly, but it may also be temporal, frontal, episodic, throbbing, and associated with nausea and vomiting; in other words, migraine (7). We recently compared ten patients with migraine, "cervicogenic" headache, cluster headache caused by arterial malformations (19) and tumours (20). Although different features of migraine, "cervicogenic" headache, cluster headache and post-traumatic headache attest to separate causes, possibly these headaches may constitute different modes of expression of which the sensory network is capable, each of which can be preferentially triggered by specific circumstances. Thus, the neck should be carefully examined in cases of headache where a cervical origin is suspected (21), but failure to find pathology does not mean that the cervical nerves are uninvolved.

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are sensory nerves which enter into the second and, to some extent, the third cervical segments. The nerves enter the spinal cord via the tract of Lissauer to terminate in the substantia gelatinosa of the upper cervical cord, where they synapse. The infratentorial intracranial structures are innervated by the upper three cervical nerves. Sensory cutaneous distribution of the occipital nerve is over the back of the head anteriorly to the borders of the innervation of 5A. The C2 component is a more medial band extending from the superior nuchal line to this boundary. C1, when present, innervates an overlapping area more posteriorly. The greater occipital nerve passes over the superior nuchal line, mid-way between the mastoid process and the occipital protuberance, just lateral to the insertion of the nuchal ligaments. The lesser occipital nerve passes lateral to the greater occipital nerve, over the nuchal ridge.

Tenderness may be elicited in these areas in patients suffering from the headache syndromes described. The greater occipital nerve runs transversely and then turns, at right angles to run rostrally. Because of this the nerve is at risk of transection by parasagittal incisions in the upper neck. It then emerges through the aperture above the aponeurotic sling, between the trapezius and the sternomastoid. This renders untenable any speculation that it may be compressed by spasm in the trapezius. Similarly, the nerve is not vulnerable to bony compression between the posterior arch of the atlas and the lamina of the axis. How the occipital nerve becomes sensitive in the diverse headache condition described is still a matter of speculation.

Occipital nerve blockade has been used for the treatment of diverse headaches for a long time. Osier (in 1912) advocated occipital neururectomy for resistant cases of what he termed cervical occipital neuralgia. The techniques varied from author to author, but usually the procedure involved infiltration of the branches of the greater and lesser occipital nerve with a local anaesthetic, and a long-acting steroid. The technique described by Saadah and Taylor (22) is representative. The patient is placed prone, with the forehead flat on the table. The hair above and below the superior nuchal line is separated with 1 inch paper tape. The scalp is cleaned with iodine; 10 cc of 1% of 1% lidocaine, with 2 cc of betamethasone mixture (Celestone Soluspan 6 mg/cc is injected into the tender points). A 23 gauge needle is used. It is advanced to the occipital bone, slightly withdrawn, and then the injection is performed. The patient has to lie flat for half an hour afterwards to avoid the dizzy feeling which occurs when the occipital muscles are anaesthetized.

Rationale for occipital nerve blockade

Anthony (23) took a pragmatic approach and assessed the effects of occipital nerve blocks in a variety of headache syndromes. He injected Depo-Medrol in the occipital nerve of cluster patients and obtained a significant and, in some cases, long-lasting relief. He categorized four groups of patients with idiopathic headache: (1) Migraine, (2) migraine with greater occipital nerve irritation, (3) occipital neuralgia, (4) tension headache. He excluded all patients with a history of trauma. The diagnostic criteria of migraine with greater occipital nerve irritation and occipital neuralgia are provided in Table 2. The point of exit of the greater occipital nerve was located with a nerve stimulator and disturbances of sensation in its distribution were noted. Patients had: (1) no sensory change, (2) hypoalgesia, (3) hyperalgesia, or (4) dysesthesia. Anthony noted that headache frequency was increased in the patients with occipital nerve irritation and occipital neuralgia compared to the migraine group, and the age was greater (mean of 11.4 years older). The pain thresholds in the occipital nerve irritation and occipital neuralgia groups were significantly lower on the headache side. He concluded that in 30% of patients with idiopathic headache there is evidence that greater occipital nerve irritation exists and postulated that this may be a confounding variable in trials of anti-migrainous drugs. He also suggested that such patients may fail standard anti-migrainous therapy but respond to steroid occipital blocks, physiotherapy or total neurotomy. He ends by coining the diagnosis of “headache due to irritation of the greater occipital nerve”. We would prefer to call this, “headache—migrainous or tension or post-traumatic—accompanied

### Table 2. Diagnostic criteria.

**A** Migraine with GON irritation (MON)
1. History of established migraine.
2. Recent increase in frequency/severity of headaches with occipital radiation/origin of pain.
3. Headache always or almost always on the same side of the head.
4. Tenderness/reduced pain threshold of GON on the affected side.
5. Absence of sensory changes in the area of distribution of the GON on that side.

**B** Occipital neuralgia (ON)
1. Unilateral occipital headache, continuous or paroxysmal (neuralgic), always on the same side.
2. Circumscribed tenderness over the GON as it crosses the superior nuchal line.
3. Hypo or hyperalgesia or dysesthesiae in the area of distribution of the GON.
4. Relief of acute attacks by infiltration of the GON with local anaesthetic.

Source: (23); GON = greater occipital nerve.
The investigators proceeded to do a prospective trial but included post-traumatic headache. All patients bilateral occipital neurectomy provided six months followed by injection of betamethasone, which had headache with a significant cervical component. The by irritation of the greater occipital nerve", removing but not such good effect in the post-traumatic and relief in patients with vascular and tension headaches received the full armamentarium of anti-migrainous of relief supplemented by further steroid injections. Hdocaine in the greater occipital nerve. This was patient responded dramatically to local injection of lidocaine in the greater occipital nerve. This was followed by injection of betamethasone, which had to be repeated every three to four weeks. Eventual, bilateral occipital neurectomy provided six months of relief supplemented by further steroid injections. The investigators proceeded to do a prospective trial in which patients were classified as by Anthony (23) but included post-traumatic headache. All patients received the full armamentarium of anti-migrainous drugs during follow-up. The authors reported a good relief in patients with vascular and tension headaches but not such good effect in the post-traumatic and post-infective group. Although there were no controls, none of these patients had responded satisfactorily to standard anti-migraine therapy. They concluded that the finding of a tender occipital zone was a sign that the greater occipital nerve was involved, and that interruption of this pathway could be a potential benefit in blocking the evolving pain cycle.

Blume et al. (24) describe radiofrequency electrocoagulation in the occipital nerve territory in 450 consecutive cases of headache with cervical involvement. They report good or excellent results in 85% of cases in a follow-up of two to ten years. They were equally successful in headache with, or without, trauma.

In our clinic we were impressed by the seemingly better response to occipital blockade in patients with a history of head trauma. As a preliminary to a prospective study, we carried out a retrospective study of 97 patients with migraine, and 87 with post-traumatic headache with clinical features that categorized the headache as migraine (1). All had been unresponsive to persistent medical therapy. Treatment was by a single or a series of injections of Depo-Medrol and Xylocaine into the area of the greater occipital nerve. It was difficult to quantify the headaches retrospectively. However, 63 of 87 post-traumatic headache patients, and 52 of 97 migraine patients, reported being "significantly better" on a patient rating scale up to six months after occipital nerve blockade. The post-traumatic group had a significantly higher response rate. (Chi-square $p < 0.01$.) In this study the presence of neck pain, occipital location of headache was not correlated with the degree of relief.

The greater occipital nerve is composed mainly of $C_2$-originating fibres but there is some involvement of $C_3$. The area supplied by it is also supplied by $C_2$, which is often found as a very thin nerve root, sometimes mistaken for an inconsequential strand of tissue (25). It is, however, the $C_2$ which is the crux of the cervicotorcular pain system. The greater occipital nerve may have very small branches and it may be impossible to block all of them. In patients who fail to respond to occipital nerve blockade, it may be necessary to block the more proximal $C_2$ nerve roots.

The apparent better response of post-traumatic patients to the nerve blocks has impressed us, and it appears that this difference may be statistically significant, at least in a retrospective trial. We are currently carrying out a prospective trial of 200 patients, both migrainous and post-traumatic, using occipital nerve blocks. It is worth reiterating that the patients who demonstrated improvement had previously not responded to anything in the way of medication which had been prescribed to them by experienced physicians.

Interestingly, and this did not come out in the study, many patients reported cessation of the headache while the block was in effect, but for reasons not as yet ascertained the relief was not maintained. This is a potentially interesting area of exploration. A number of possible directions come to mind. For example, first, should we be encouraging the creation of long-acting anaesthetics, and will this improve the percentage of people who get ongoing relief? Second, would this be an indication for surgery to maintain long-term effect of the blocking? Third, in patients who do not have prolonged relief, is their cervical pathology providing continuous irritation of the nerves which were blocked, and thus restarting the pain cycle? Fourth, it is unclear which specific nerves were blocked. It may be technically difficult to block the greater occipital nerve without also blocking the third occipital nerve and some of the fibres of the semispinalis capitis. It would be useful to know whether one or several nerves need to be blocked. Finally, we feel that a prospective study should be done on a multicentre basis using a common protocol. This is essential for evaluating this approach.

Conclusions
There are two convergent concepts which form the basis of our opinion. The first is the evolution of a variety of headache syndromes into a symptom pattern which includes occipital pain. The second is the treatment of diverse headache syndromes by a
technique which ostensibly blocks some of the inputs into the cervical trigeminal relay. The hypothesis is that the plasticity in the sensory pathways allows leakage of the pain-generating activity and more and more ganglia involving ever greater neural networks, until a point is reached where the cervicotrigeminal relay is involved. At this stage, interruption of the inputs into this relay can disrupt the expanding network and the whole process is blocked at least for a time. Pain activity generated in the cervical area can likewise spread into the trigeminal system and produce the sundry phenomena of the putatively trigeminal nerve syndrome, migraine. Again interruption of inputs into the system may disrupt the complete network.

Occipital nerve blockade is reasonably straightforward and is performed by many physicians, anesthesiologists, psychiatrists, neurologists and family doctors. Generally there is very little in the way of adverse effects. However, care must be taken in selecting the patient population. There is a temptation in some cases to perform this procedure rather haphazardly without proper assessment and follow-up. Appropriate clinical trials of this technique must be performed (25), for which definitive criteria must be developed. If this is not done the procedure may fall into disrepute and the opportunity lost for an alternative treatment modality in difficult and intractable headache patients.

References

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