Greater Occipital Nerve Block: A Diagnostic Test?

Often a neurosurgeon or orthopedic surgeon requests a diagnostic nerve block to determine, prior to attempting any surgical procedure, whether a specific cervical nerve root is the generator of the patient's symptoms.[1]

This statement from a contemporary textbook reflects the commonly held view that in pain management nerve blocks are diagnostic.

There is wide variability of headache syndromes treated by greater occipital nerve (GON) blockade.[1] The putative mechanisms by which they might relate to the GON are unclear. It seems a priori improbable that such diverse conditions as migraine (with its complex cerebral and brainstem mechanisms), cluster headache, occipital neuralgia, cervicogenic headache, whiplash syndrome,[2] and various tension type headaches, should either share a common aetiological mechanism or be responsive to the same treatment of a peripheral nerve.

Anatomy

The GON is composed of the medial fibres from the dorsal ramus of the second cervical nerve. The ventral ramus of C2 also contributes to the lesser occipital nerve and innervates deeper structures (periosteum of the occiput, vertebrae, etc.). In rats, a population of neurones of the dorsal horn at C2 shows convergent input from both dura and cervical skin and muscle territories, suggesting a functional continuum between the trigeminal nucleus caudalis and upper cervical segments involved in cranial nociception. GON stimulation in rats facilitates dural stimulation, implying a central mechanism at the second order neurone.[3] C2/C3 blockade is claimed to produce benefit of comparable order to GON blockade and both are said to be effective in the diagnosis and treatment of cervicogenic headache.[4]

The literature fails to incriminate specific anatomical structures as the source of cervicogenic pains. Very similar diagnoses invoke structures such as nerve roots, individual peripheral nerves, bony structures, and the non-specific cervicogenic pain/headache. For example:

"Diagnostic anesthetic blockade for the evaluation of cervicogenic headache can be directed to several anatomic structures such as the greater occipital nerve (dorsal ramus C2), lesser occipital nerve, atlanto-occipital joint, atlantoaxial joint, C2 or C3 spinal nerve, third occipital nerve (dorsal ramus C3), zygapophyseal joint(s) or intervertebral discs based on the clinical characteristics of the pain and findings of the physical examination."[5]

The authors state fluoroscopic or interventional MRI-guided blockade may be necessary, to assure specific localisation of the pain source; yet they fail to present evidence that such measures do inculpate the actual source of pain. The opposing view (which I share) is that of Silverman:

"there are no diagnostic imaging techniques of the cervical spine and associated structures that can determine the exact source of pain."[6]

Are nerve blocks diagnostic?

Neural blocks may be useful as an empirical way of treating diverse head and neck pains, but such a response is also often used as the criterion for diagnosis.[7,8] But such diagnoses, though clinically useful, are inexact and the procedure may be valid (if proven by properly designed trials) only as an empirical mode of controlling pain.

Blondi rightly notes that *"Occipital nerve blockade, … often results in a nonspecific regional blockade rather than a specific nerve blockade and might result in a misidentification of the occipital nerve as the source of pain."* And he says: *"occipital neuralgia is believed to arise from trauma to or entrapment of the occipital nerve within the neck or scalp, but the pain may also arise from the C2 spinal root, C1–2, or C2–3 zygapophyseal joints or pathologic change within the posterior cranial fossa."* If its source is the nerve roots, how can it be rationally considered to be a neuralgia of the occipital nerve?

Despite many published studies, the diagnostic utility of employing greater occipital nerve (GON) blockade in a variety of headaches and neck strains is unproven. Many trials contain small numbers. The physician administering the injection in many trials is not blinded to the treatment. Follow-up assessment is commonly at about four weeks, too brief a period may have detected significant differences in outcome. The local anaesthetic or steroid used, and the doses vary and are commonly chosen empirically. Controls are often omitted or poorly matched. And, interpretation is confounded by subjective criteria of pain relief and marked variation of techniques. There are therefore, several unresolved issues concerning both rationale, claimed benefits, and techniques.

Ashkenazi and co-workers report, *"The rationale of GON blockade for the treatment of headache is based on the anatomical connections between trigeminal and upper cervical sensory fibres at the level of the trigeminal nucleus caudalis."[9,10]* But is this alone a diagnostic foundation or a mechanism sufficient to explain such diverse head pains?

The methodologies of some of the studies are limited by lack of a standardised treatment protocol or by a retrospective design. In migraine, for example, improvement has been reported after GON blockade, but also after prophylactic drugs, and injected botulinum A toxin.[11] There are claimed to be four major 'trigger points' along the course of several peripheral nerves that may cause migraine headaches, which are therefore also treated by injection.[12] Among the peripheral nerves the greater occipital nerve has become a favourite target of needle wielders.

Structures said to be involved in the pathogenesis of occipital headache include the aponeurotic attachments of the trapezius and semispinalis capitis muscles to the occipital bone, and entrapment of the GON within these aponeuroses, causing symptoms of 'occipital neuralgia'.[13] Whereas cervicogenic headache is a useful clin-

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In respect of diagnosis, the current evidence appraised suggests that the use of nerve blocks as the defining or pathogenetic criterion is both unsound and unreliable

Table 1: Variation of anatomical sites for greater occipital nerve.		
Study	**Vertical location of GON (cm)**	**Lateral location of GON (cm)**
1. Mosser et al.	3cm below EOP	1.5cm from midline
2. Loukas et al.	2cm below EOP	2cm from EOP
3. Natsis et al.	The site where the semispinalis capitis is pierced by the GON	
4. Bovim et al.	no anatomical landmark given for injection	
5. Becser et al.	Along intermastoid line	0.5 to 2.8cm from midline
6. Tubbs et al.	2cm above intermastoid line	4cm lateral to EOP
EOP = external occipital protuberance.		

ical description,[14] it is not a diagnosis that accurately inculpates the pathogenic structures involved, nor the mechanism of pain. The International Headache Society (IHS) proposed diagnostic criteria[15] for cervicogenic headache (11.2.1) are:

1. Pain referred from a source in the neck and perceived in one or more regions of the head and/or face.
2. Clinical, laboratory and/or imaging evidence of a disorder or lesion within the cervical spine or soft tissues of the neck, known to be, or generally accepted as, a valid cause of headache.
3. Evidence that the pain can be attributed to the neck disorder or lesion, based on either clinical signs that implicate a source of pain in the neck or abolition of headache following diagnostic nerve block.
4. Pain resolving within three months after successful treatment of causative disorder or lesion.

Interestingly, cervical spondylosis is NOT accepted as a valid cause. Taken in turn, these criteria don't confront these fundamental issues.

1. There is no hard evidence, only imprecise inference that the pain can be attributed to the neck disorder or lesion.
2. Disorders within the cervical spine or soft tissues of the neck, known to be, or generally accepted as, a valid cause of headache is an all-embracing and unproven generalisation that does not indicate the primary pathology.
3. Since there are no conclusive clinical signs that prove a source of pain in the neck, this is spurious; and, abolition of headache following nerve block is only suggestive, not diagnostic. Clinical signs or pain relief after nerve blocks do not constitute attributable proof of causation.
4. Pain resolving within three months after successful treatment of causative disorder or lesion, illogically and falsely assumes that response to treatment is proof of causation. Few trials extend to three months or beyond.

A further difficulty is that the techniques employed and for which success is claimed differ from one series to another.

Anatomical variability

An important but neglected factor is the anatomical variability (Table 1) of the greater occipital nerve.[16]

1. Mosser et al. dissected 20 cadaver heads to trace the normal course of the greater occipital nerve from the semispinalis muscle penetration to the superior nuchal line. Standardised measurements were performed on 14 specimens to determine the location of the emergence of the nerve using the midline and occipital protuberance as landmarks. The location of emergence was determined to be at a point centered approximately 3cm below the occipital protuberance and 1.5cm lateral to the midline.[12]
2. Loukas et al.[13] examined the course and distribution of GON and its relation to the aponeuroses of the trapezius and semispinalis capitis in 100 formalin-fixed adult cadavers. The greater occipital nerve was located at a mean distance of 3.8cm (range 1.5-7.5cm) lateral to a vertical line through the external occipital protuberance and the spinous processes of the cervical vertebrae. It was also located approximately 41% of the distance along the intermastoid line (medial to a mastoid process) and 22% of the distance between the external occipital protuberance and the mastoid process. The location of GON for anaesthesia or any other neurosurgical procedure has been established as one thumb's breadth lateral to the external occipital protuberance (2cm laterally) and approximately at the base of the thumb nail (2cm inferior)[13] This was the first study proposing landmarks in relation to anthropometric measurements.
3. Natsis and colleagues reported the course and the diameter of the GON in 40 cadavers.[17] In three cases, the GON split into two branches before piercing the trapezius muscle aponeurosis (TMA) and reunited after having passed the TMA, and it pierced the obliquus capitis inferior muscle in another three cases. The GON and the lesser occipital nerve reunited at the level of the occiput in 80% of the specimens. The nerve became wider towards the periphery. This may be relevant to entrapment of the nerve. In three cases, the GON split into two branches before piercing the TMA. Anaesthetic blockade of the GON for diagnosis and therapy was best aimed at the site where the semispinalis capitis is pierced by the GON.
4. An autopsy study by Bovim et al. on 20 cases without known headaches showed a marked variation in the relation between the GON and nuchal muscles.[16] The trapezius muscle was penetrated by the GON in 45% of cases, the semispinalis muscle of the head was penetrated in 90% of cases, and the inferior oblique muscle of head in 7.5% of cases. Macroscopic findings of possible compression were made in 11 cases (27.5%), "*indicating that nerve compression per se may be of minor importance since it seems to exist in the absence of headache.*"
5. Becser, Bovim and Sjaastad reported topography shown by dissection and careful measurements of 10 embalmed cadavers.[18] A great variability in nerve topography was seen interindividually and intraindividually. The greater occipital nerve ascended between 5mm and 28mm from the midline along the intermastoid line. The minor occipital nerve was found between 32mm and 90mm from the midline along the same landmark. In most cases, both the GON and the minor occipital nerve pierced the aponeurosis after branching. Thirteen GONs and eight minor occipital nerves also were embedded in this tissue. Twelve of the 20 GONs formed a rich network around the occipital artery. Importantly they commented: "*anatomic structures with an imminent risk of causing entrapment were not observed. … results suggest that optimal locations for blockade techniques should be reconsidered.*"
6. Tubbs and colleagues noted the surprising lack of surgical landmarks in the literature for avoiding the cutaneous nerves in this region. The GON was found to lie at a mean distance of 4cm lateral to the EOP. On all but three sides, a small medial branch was found that ran medially from the GON to the 3rd occipital nerve approximately 1cm superior to a horizontal line drawn through the EOP. The GON was found to pierce the semispinalis capitis muscle on average 2cm above the intermastoid line, and to divide into medial and lateral branches 0.5cm superior to the EOP.[19] This fits with the observation that 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…[20]

These considerable differences in proposed landmarks for the GON are reflected in the diversity of proposed and tried treatment sites. Cervical epidural steroid injections are used in patients with spondylosis,[21] but are largely ineffective.[22] Greater and lesser occipital nerve blockade may provide temporary, but substantial, pain relief in some cases of similar efficacy to blockade of the C2 and C3 nerves.[23] Blockade, neurolysis, nerve resection, rhizotomy, and decompressive techniques have all been employed, each with claims of success. Bogduk's team reported[24] the efficacy of percutaneous radiofrequency medial branch neurotomy in the treatment of chronic neck pain in 71% of patients, but the precise nature and anatomical basis of the symptoms are not thereby clarified. What is the basis in the unrelieved substantial 29%?

Lieppman (1980) described[25] 164 patients

with comparable symptoms in whom he diagnosed 'occipital neuralgia' and found a high cure rate after occipital subcutaneous injections of lidocaine; however, he also found a 50% cure rate in patients given occipital subcutaneous injections of saline. A significant contributory placebo effect is probable, as in all pain syndromes.

Discussion

Thus, there are problems in accepting GON and related neural sites subjected to blockades as a rational as opposed to empirical method:

1. Anatomical structures threatening neural entrapment are seldom observed.[16]
2. Nerve compression per se may be of minor importance since it can exist in the absence of headache.[16]
3. There are many and considerable anatomical variations in the nerve so that consistent surgical landmarks in this region are surprisingly lacking,[12,13,16-18] and the injected substance may not have affected the GON.
4. Clinical techniques of localising the nerve are variable and imprecise.[18,19] Tenderness and evoked pain on palpation are notoriously unreliable.

An apparent response to neural blockade is often used as one of several criteria for the diagnosis of cervicogenic and other headaches.[26,27] but such a diagnosis, though of arguable clinical value, is itself inexact and justifies the procedure only as an empirical mode of controlling pain. As Pollman and colleagues commented, "*cervicogenic headache should therefore be understood as a homogeneous but also unspecific pattern of reaction.*"[28] The placebo effect[29] of injections is often underestimated or neglected. To the scientist, a major problem is that placebos influence patient outcomes after any treatment, including surgery. The well-known extent of placebo effects in acute head pains was illustrated in Harden and coworkers' trial which concluded:

This profound reduction observed after administration of a placebo prevented accurate evaluation of the effects of [ketorolac]. The placebo response must be considered in the design of future trials using intramuscular medications in the acute intervention of headache crises. In addition, the use of a standard analgesic is necessary to demonstrate both assay sensitivity and magnitude of response to placebo.[30]

Placebo effects and spontaneous remissions can cause apparently good results that are falsely attributed to the efficacy of any treatment claims.[31] As one example, Peres et al. treated 14 cluster headache patients with greater occipital nerve block. Four patients (28.5%) had a good response, five (35.7%) a moderate, and five (35.7%) no response. The authors concluded GON blockade is a therapeutic option for the transitional treatment of cluster headache,[32] yet their results are entirely consistent with a placebo effect. It is suggested that the placebo response may contribute significantly to the apparent successes of nerve blocks, but does not necessarily account for the relief of pain in all cases. Placebo effect does not imply psychogenesis, but is a genuine, validated phenomenon, which may be organically founded in regionally specific changes in brain function; for example, dorsal-cortical increases and limbic-paralimbic decreases in glucose metabolism demonstrated in a trial of antidepressant vs placebo.[33]

Conclusion

The lack of specificity of greater occipital or other peripheral nerve, and cervical root blockade in the treatment of diverse headaches, (including migraine, cluster headache, cervicogenic and occipital pains), though achieving variable empirical therapeutic success, is a continuing cause of diagnostic confusion and wooly thinking. Although various structures in the neck probably contribute to several patterns of headaches and neck pains, they have not to-date been adequately defined. More rigorous studies are needed to identify the several anatomical structure and the physiological mechanisms that underlie these ill-defined symptoms. In respect of treatment, these considerations agree with the conclusions of Bogduk[34] that "*The available evidence from the small number of case series and retrospective studies published in the peer-reviewed literature is insufficient to conclude that either local injection therapy or surgery is an effective treatment for occipital neuralgia or cervicogenic headache.*"

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