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Occipital Nerve Stimulation for the Treatment of Occipital Neuralgia—Eight Case Studies

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■ ABSTRACT

Objective. The aim of this study was to examine the hypothesis that subcutaneous occipital stimulation influences pain due to occipital neuralgia.

Materials and Methods. Between 2001 and 2004 eight patients with intractable occipital neuralgia were referred to our center. Their records were reviewed. Each patient was interviewed over the telephone. They were all offered a trial of stimulation using a percutaneous lead over 1 week. If they achieved 50% pain reduction a permanent lead was implanted. The impact of occipital stimulation was measured by pain score, analgesic requirements, and employment status.

Results. Seven proceeded to a permanent stimulator. There was a reduction in the visual analog score postimplantation in five of the seven patients. The total quantity of opiates taken after implantation showed a marked reduction. Of the seven who had a permanent implant two acquired full-time employment.

Conclusion. Occipital neuralgia is a useful and reversible treatment for intractable occipital neuralgia. ■

KEY WORDS: occipital, neuralgia, stimulation, headache, neuropathic pain

INTRODUCTION

According to the International Association for the Study of Pain (IASP), occipital neuralgia is described as "pain, usually deep and aching, in the distribution of the second cervical dorsal root"

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(1). An understanding of the complex anatomy of the cervical plexus should precede the diagnosis of occipital neuralgia.

The nerves of interest are the upper three cervical nerves. The anterior rami of the upper four cervical nerves unite by a series of loops to form the cervical plexus. This supplies the skin and muscles of the neck and innervates the diaphragm. The posterior primary ramus of C1 is entirely a motor nerve and supplies the superior oblique, inferior oblique, rectus capitus, and semispinalis capitis muscles (2).

The posterior primary ramus of C2 emerges between the posterior arch of the atlas and the

lamina of the axis, curves around the inferior border of the inferior oblique muscle, to which it sends a branch and then divides into a large medial and a small lateral branch. The medial branch is the greater occipital nerve. This pierces semispinalis capitis and then trapezius. It is joined by a filament from the medial branch of C3 and then ascends in company with the occipital artery to supply the skin of the occipital region as far as the vertex (2).

The lesser occipital nerve (C2) hooks around the spinal accessory nerve (XI) then ascends along the posterior border of the sternocleidomastoid. It pierces the deep fascia in the upper part of the medial aspect of the posterior triangle. It then splits up into the auricular, mastoid, and occipital branches. The occipital branch is sensory to the skin in the occipital area immediately above and behind the mastoid.

The possible sources of cervical spinal pain that might be referred to the head are dictated by the distribution of the upper three cervical spinal nerves. Through their various branches these nerves innervate the joints and ligaments of the median atlantoaxial joint, the atlanto-occipital joint, and lateral atlantoaxial joints, the C2-3 zygapophyseal joint, the suboccipital and upper posterior neck muscles, the upper prevertebral muscles, the spinal dura mater of the posterior cranial fossa, the vertebral artery, the C2-3 intervertebral disc, and the trapezius and sternocleidomastoid muscles. All of these structures can be sources of pain and should be considered in the differential diagnosis of cervicogenic headache (3).

In most cases, the cause of the neuralgia is not found. However, there are examples of occipital neuralgia caused by lesions to the nerves and occipital structures include exuberant callus formation, hypermobile posterior arch of atlas, compression of the greater occipital nerve by the semispinalis capitis and trapezius muscles, upper cervical cavernous hemangioma, diabetes, temporal arteritis, and multiple sclerosis (4-10).

Patients with occipital neuralgia usually present with an associated cervicogenic headache. This is defined by the IASP as "attacks of moderate or moderately severe unilateral head pain without change of side, ordinarily involving the whole hemicranium, usually starting in the neck or occipital area, and eventually involving the fore-
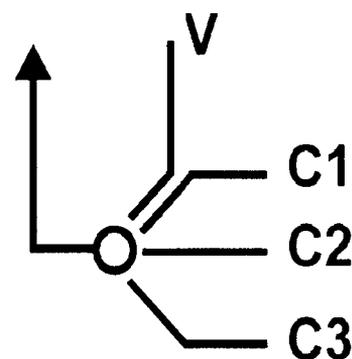

Figure 1. Depicts convergence of the cervical afferents on the trigeminal (cranial nerve V) nucleus.

head and temporal areas, where the maximal pain is frequently located. "The headache usually appears in episodes of varying duration in the early phase, but with time the headache frequently becomes more continuous with exacerbations and remissions. Symptoms and signs such as mechanical precipitation of attacks imply involvement of the neck" (1).

The reason for globalized pain in occipital neuralgia is explained by convergence between cervical and trigeminal afferents in the spinal cord. Afferents of the trigeminal nerve descend through the spinal tract of the trigeminal nerve. Their collaterals terminate in the pars caudalis of the spinal nucleus of the trigeminal nerve and in the dorsal horns of their respective segment, and send ascending and descending collaterals to adjacent segments. Therefore, at any given cervical segment, second-order neurons that project to higher centers can receive a convergent input from afferents of the trigeminal nerve and the C1, C2, and C3 spinal nerves (Fig. 1) (3).

MATERIALS AND METHODS

Between 2001 and 2004 patients with intractable occipital neuralgia were referred to our Nepean Pain Management Center. These patients are referred to our tertiary referral center because they have failed conventional treatment.

Their diagnosis was confirmed by 1) pain-deep and aching in the distribution of the second cervical dorsal root; 2) scalp hyperesthesia; 3) chronic and recurrent episodes of pain; and 4) analgesia and reduction of headache on subcutaneous infiltration

of local anesthetic over the greater and lesser occipital nerves.

All patients who presented with occipital neuralgia were offered a trial of occipital stimulation. Before the trial takes place, each patient is reviewed by a psychologist. Patients are not offered a trial if the psychologist finds they have a major psychiatric illness such as a major depression or psychosis, an antisocial or borderline personality disorder, or if they are substance abusing.

A trial of occipital stimulation is performed with the patient awake and prone in an operating theater. The patient is given a small intravenous dose of midazolam to reduce anxiety but maintain cooperation with perioperative stimulation. The hairline is prepared with a wide shave over the anticipated length of the lead. One gram of cefazolin is given preoperatively. The site is prepared using povidone iodine, which is allowed to dry to air.

Image intensification is used to identify the posterior nuchal line at the level of C1 and the midline. Five milliliters of lignocaine are infiltrated subcutaneously in the midline. A Tuohy needle is tunneled subcutaneously from medial to lateral along the nuchal line. The Tuohy introducer is removed and replaced with a quadripolar percutaneous lead. This remains superficial to the greater and lesser occipital nerves. This is taped in place with steristrips and tested either in theater or recovery for the most satisfactory electrodes, rate, pulse width, and voltage. No changes are made to the patient's medications.

The patient is sent home and reviewed in the clinic in 1 week. The lead is then removed. Pain reduction and patient satisfaction are the trial end points. A successful trial includes > 50% reduction in pain and a willingness of the patient to proceed with a permanent implant.

The permanent implant is performed again with the patient prone but under general anesthesia. The lead inserted is a surgical lead (paddle-style electrode) because it can be sutured to the dense cervical fascia to prevent movement. Some patients required bilateral leads for bilateral neuralgia. The lead extension is tunneled subcutaneously to the generator located either in the subclavicular region or in the lower abdominal quadrant below the belt line (Fig. 2).

Intravenous third-generation cephalosporin antibiotics are administered 8 hourly for the first
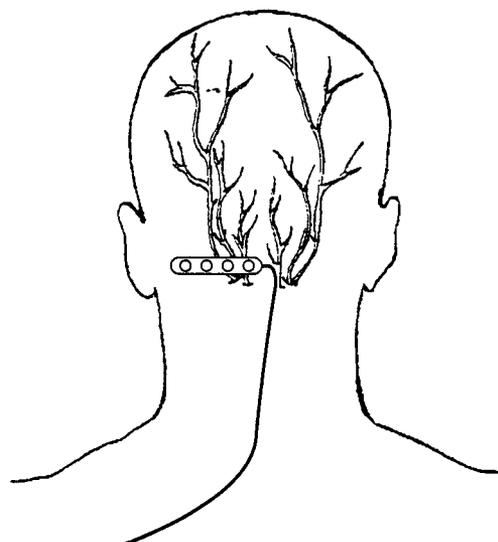

Figure 2. This sketch shows the location of the surgical lead superficial to the greater and lesser occipital nerves at the occipital protruberance.

48 hours and the patient is then discharged with a further 3 days of oral antibiotics.

The sutures are removed after 10 days. The patient is followed over the next 6 months in the clinic to manage medication changes and assess the efficacy of stimulation. Opiate withdrawal is not considered until the permanent implant is *in situ* for 2 weeks. All of the patients were telephoned after the permanent implant was placed.

The end points of permanent implantation include a visual analog score, averaged over a day, reduction in medications required, and changes in activities of daily living.

RESULTS

Eight patients were offered a trial of occipital nerve stimulation. One failed the trial and seven proceeded to a permanent stimulator. Those who had a permanent implant are discussed below.

The follow-up after permanent implant averaged 25 months with a range of 6–47 months. Three women and four men proceeded to a permanent stimulator. The average age at implantation was 46 years (range 30–65 years).

Five patients had unilateral and two had bilateral occipital neuralgia. Comorbidities included diabetes, hypertension, C5/6 disc degeneration, and a C6 fracture repaired with a C6-7 fusion. All

Table 1. Interventional Treatments Used to Manage Occipital Neuralgia Before and After Permanent Stimulation

Patient	Treatments before implantation	Treatments after implantation
1	Occip. N. LA + steroid	nil
2	Occip. N. LA	Frontal and occipital sc. Botox
3	Occip. N. LA + steroid	nil
	C2-3 dural sleeve LA + steroid × 2	nil
	C2-5 medial branch radiofrequency × 2	nil
4	C2-5 medial branch LA + steroid	nil
	C2-5 medial branch radiofrequency	nil
5	C2-5 medial branch LA + steroid	nil
6	Occip. N. cryotherapy	nil
7	Occip. N. LA + steroid	Frontal and occipital sc Botox
	Neurolysis greater and lesser occipital nerves × 2	

Key: Occip. N., occipital nerve; LA, local anesthetic; IM, intramuscular; medial branch, medial branch of the dorsal ramus; sc Botox, subcutaneous *Botulinum* toxin.

Table 2. Medications Before and After the Insertion of a Permanent Stimulator

Patient	Medications before implantation	Medications after implantation
1	Sodium valproate, Amitriptyline HCl	nil
	Gabapentin	
	Panadeine forte (codeine phosphate and paracetamol) 2/day	
2	Venlafaxine HCl	Panadeine forte 2/day
	Panadeine forte 8/day	
3	Morphine sulfate 100 mg Bd	Oxycodone HCl 5 mg/day
	Diclofenac sodium	
4	Morphine sulfate 50 mg Bd	Panadeine forte 1/day
5	Oxycodone HCL 120 mg/day	Morphine sulfate 25 mg Bd
	Neurontin 1200 mg/day	
	Fluoxetine HCl	
6	Codeine as required	nil
7	Oxycodone HCl 20 mg/day	Oxycodone HCl 25 mg Bd
	Pethidine HCl injections 5× per month	Pethidine HCl injections 4× per month

seven patients presented with unilateral or bilateral headaches (Tables 1 and 2; Fig. 3).

Fewer interventional treatments were required after implantation of an occipital stimulator. The total quantity of opiates taken after implantation showed a marked reduction. There was a reduction in the visual analog score (VAS) postimplantation in five of the seven patients. Of the seven who had a permanent implant, two acquired full-time employment. One of them is playing basketball. Two patients had persistent anxiety and depression. A full-time mother and a disability pensioner had persistent anxiety and depression.

Localized sepsis was a problem around the generator in one patient and the paddle lead in another. These patients required explantation and reimplantation when the sepsis settled.

The eighth patient who had a trial of occipital stimulation failed to achieve sufficient analgesia. He did not proceed to permanent implantation. On presentation he was receiving pethidine injections three times per week, gabapentin, and carbamazepine. His previous interventions included radio-frequency ablation of medial branches C2-5 and an open resection of the greater and lesser occipital nerves.

DISCUSSION

Treatment options for occipital neuralgia include medications and interventions following diagnostic local anesthetic blockade of the greater and lesser occipital nerves. This technique is well-described in a recent paper by Ward (11).

The local anesthetic blockade with or without steroid is sufficient to provide temporary analgesia and a diagnosis. However, inevitably the pain returns. Many patients are subsequently referred
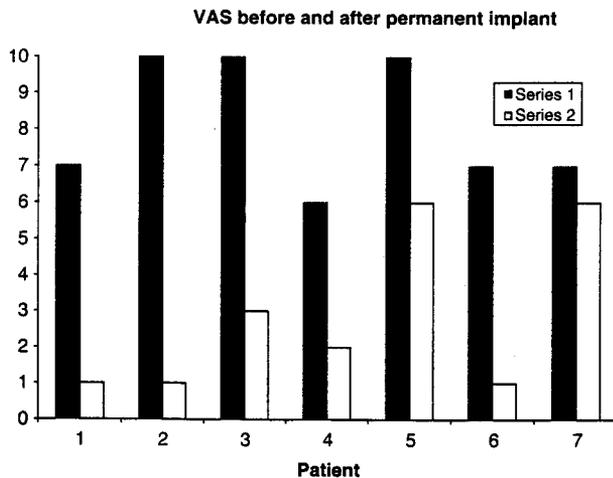
VAS before and after permanent implant

Figure 3. The visual analog score (VAS) averaged over a day was compared before and after implantation.

to a neurosurgical service. Surgical treatments have employed neurolytic or decompressive techniques. Neurolysis may be performed at the nerve roots of C1-3 (partial posterior rhizotomy), or at the ganglion (12-15).

Decompressive techniques are aimed at reducing tension on the greater and lesser occipital nerves due to anatomical anomalies, for example, where they pass through muscles and fascia in the posterior cervical spine (16). Radio-frequency treatment of the occipital nerves provides good analgesia for some patients but others can develop a delayed deterioration in their condition similar to surgical ablation techniques (17). Govind et al. described thermo-lesioning of the third occipital nerve. In their follow-up, 97% experienced numbness but 55% suffered from dysesthesia (27).

The outcomes of surgical techniques vary from excellent to adverse. This includes a subset of patients who experience anesthesia dolorosa. For example, in a series by Stechison and Mullin, 3/5 had pain relief after ganglionotomy and 2/5 after decompression of the C2 dorsal root ganglion and nerve (15). In another small series by Horowitz (nine patients), 44% had relief of their pain following dorsal nerve root sectioning (14). Dubuisson, however, relived occipital neuralgia in 10/14 cases treated by partial posterior rhizotomy at C1-3 (13). Larger case series report up to 17% of patients that have no relief of their pain following a variety of surgical procedures with variable etiologies (18,19). A nondestructive neuromodulatory tech-

nique has been described. This is using subcutaneous occipital nerve stimulation (19-22).

Spinal cord stimulation is widely used for treatment of neuropathic pain especially in the lower limbs. However, occipital stimulation for the treatment of occipital neuralgia is uncommon. The few published papers describe patient outcomes in retrospective reports (19-21). Weiner describes 13 patients who underwent subcutaneous wire placement over the greater and lesser occipital nerves (20). One was explanted because the occipital neuralgia symptoms had resolved. Of the remaining 12 patients, eight had 75% or greater pain relief and one third 50% or greater pain relief. The follow-up was 1 to 6 years.

Oh inserted paddle electrodes over the greater and lesser occipital nerves for treatment of occipital neuralgia and transformed migraine (22). Of 18 patients who completed a 6-month follow-up, 14 reported excellent pain relief and two had good relief. There are reports of successful subcutaneous stimulation for treatment of other peripheral nerves (23). A prospective randomized trial of treatment vs. no treatment is yet to be performed.

Our results are similar to previously published reports. Reductions in opiate analgesia were marked. Improvements in activity levels were reflected in an improved ability to return to work. The VAS, which can be falsely recalled, is a less secure end point. Even so, our VAS demonstrates a clear reduction in pain. These trends concur with previous reports.

Lead migration and sepsis are potential problems with occipital stimulators. A paddle lead sutured to the occipital fascia can prevent lead migration. It is also a reversible procedure if the site is not adequately covered. There also should be adequate "play" in the extension.

Two patients had sepsis in their devices. The lead and the generator were removed and the sepsis was treated with antibiotics and debridement. We subsequently reviewed our preparation technique. A "neurosurgical" approach using fresh iodine paint and a formal shave of the occipital region and generator location is used. The prepared area also is covered with an adhesive, iodine-impregnated drape. There were no further episodes of sepsis after the changes to our preparation.

Pethidine dependency is noted in two patients in our review. One (patient 8), who failed the

trial, entered a detoxification program and is no longer requiring pethidine. The other patient (patient 7) had a good reduction of pain during the trial. However, after implantation the patient's VAS reduced from 7 to 6/10. She continued to require intramuscular pethidine and suffered a depressive disorder.

The use of opiate analgesia is common in chronic pain conditions. Opiate withdrawal headaches have been associated with shorter acting drugs such as pethidine. Also, pethidine is known as a drug of dependence and is no longer recommended for treatment of chronic pain conditions in the Australian state of New South Wales (24). Hence, patients with occipital neuralgia who are taking pethidine are now urged to undertake a detoxification program before they are offered permanent occipital stimulation. For our patients, pethidine dependency is a negative prognostic indicator. This is not noted to be a problem in other series.

Central, spinal cord stimulation has a number of postulated mechanisms. These include the gate-control theory, blocking of spinothalamic tracts, activation of supraspinal nuclei, and alterations to neurotransmitter release (25).

Why does subcutaneous stimulation of the occipital nerves cause a reduction in pain? The precise mechanism is unknown. The gate control-theory described by Wall in 1967 is still thought to be a reasonable explanation (26). More long-term reviews of this novel and effective technique to treat occipital neuralgia are required.

CONCLUSION

Occipital stimulation for treatment of intractable occipital neuralgia is emerging as an attractive treatment option. The pitfalls such as lead migration, sepsis, and opiate dependency should be considered. It is a technique that is easy to trial and is reversible should problems arise. Lead implantation is also straight-forward for those physicians who have interventional practices.

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