

---

## CLINICAL REPORT

---

# Peripheral Neurostimulation in Supraorbital Neuralgia Refractory to Conventional Therapy

---

Juan M. Asensio-Samper, MD\*; Vicente L. Villanueva, MD<sup>†</sup>; Alfonso V. Pérez, PhD<sup>†</sup>; María D. López, MD\*; Vicente Monsalve, MD<sup>‡</sup>; Susana Moliner, MD\*; Jose De Andrés, MD, PhD, FIPP\*

*\*Anesthesiology and Multidiscipline Pain Unit, Valencia University General Hospital, Valencia; <sup>†</sup>Family and Community Medicine, Anesthesiology and Multidiscipline Pain Unit, Valencia University General Hospital, Valencia; <sup>‡</sup>Anesthesiology and Multidiscipline Pain Unit, Valencia University General Hospital, Valencia, Spain*

■ **Abstract:** Supraorbital neuralgia has been identified as an infrequent cause of headache that may prove very difficult to control pharmacologically. Peripheral nerve stimulation using electrodes to stimulate the nerve segmentally responsible for the zone of pain may constitute a management alternative in such cases. We present the case of a patient with headache because of posttraumatic supraorbital neuralgia, refractory to medical treatment, with good analgesic control after peripheral nerve stimulation.

Peripheral nerve stimulation may be considered a safe, reversible treatment for patients with headache secondary to supraorbital neuralgia who respond poorly to pharmacological treatment, thus avoiding irreversible alternatives such as surgery. ■

**Key Words:** Neurostimulation, supraorbital neuralgia, headache, neuropathic pain, peripheral nerve stimulation

Address correspondence and reprint requests to: Juan M. Asensio-Samper, MD, Unidad Multidisciplinar de Tratamiento del Dolor, Tres Cruces s/n, 46014—Valencia, Spain. Tel: +34 961972181; Fax: +34 961972182; E-mail: jmasensiosamper@hotmail.com.

Submitted: July 5, 2007; Revision accepted: October 1, 2007  
DOI: 10.1111/j.1533-2500.2007.00165.x

## INTRODUCTION

The supraorbital nerve is a pure sensory nerve and constitutes a terminal branch of the frontal nerve (which in turn is a branch of the ophthalmic division of the trigeminal nerve). Together with the supratrochlear nerve, it innervates the skin of the forehead, upper eyelid, the conjunctiva, and frontal sinus. After leaving the orbit through the supraorbital foramen, the nerve runs very superficially beneath the skin—thus explaining the ease with which it can be damaged as a result of trauma in this region.<sup>1</sup>

Supraorbital neuralgia is an infrequent neurological disorder included in the latest classification of the International Headache Society among the headaches belonging to “cranial neuralgia and other central causes of facial pain.”<sup>2</sup> The very low incidence and our imprecise knowledge of its clinical manifestations make this form of cranial pain difficult to diagnose in clinical practice.<sup>3</sup>

Despite the lack of knowledge of this type of neuralgia, the literature describes a series of characteristics (Table 1)<sup>4,5</sup> that aid diagnosis. Although the etiology and pathogenesis of supraorbital neuralgia are largely unknown, it has been well-established that open surgical release of the nerve was the sole definitive treatment

**Table 1.** Diagnostic Characterization of Supraorbital Neuralgia

- 
- A. Symptoms triad:<sup>4</sup>
- A.1. Severe and unilateral, stabbing cranial pain located in the frontal region (lateral zone of the forehead) and periocular area in the territory innervated by the supraorbital nerve, with the absence of trigger points.
  - A.2. Total but transient disappearance of the symptoms after selective supraorbital nerve block with local anesthetics.
  - A.3. Hypersensitivity over the nerve in the supraorbital notch (Tinel's sign).
- B. The disorder presents with signs and symptoms of sensory dysfunction (hypoesthesia, paresthesia, allodynia), and the typical "neuralgic findings" such as exteroceptive triggering mechanisms (photophobia)—these features generally being nonconstant, but more prevalent in the secondary presentations of the disorder (eg, posttraumatic).<sup>4</sup>
- C. The chronic forms present on a continuous or intermittent basis.<sup>5</sup>
- D. Female predominance of the idiopathic forms, with a mean age of 51.6 years<sup>6</sup>
- E. The idiomatic or primary forms are more frequent—trauma being one of the main mechanisms in the secondary presentations of the disorder.<sup>1</sup>
- 

option until only a few years ago. The use of different drugs, including antimigraine and neuromodulating agents, afforded minimal improvement, while supraorbital nerve block with local anesthetics provided complete but transient analgesia.<sup>6</sup>

In recent decades, neurostimulation techniques have become a decisive option for the treatment of conditions characterized by chronic neuropathic pain refractory to other treatments. Neurostimulation has grown in popularity and has largely displaced neurosurgical procedures<sup>7</sup> as it is minimally invasive and reversible—allowing the patient to test the system before deciding upon definitive implantation. Likewise, advances in hardware have extended the service-life of the equipment used, thereby reducing an inconvenience previously inherent to systems of this kind: the limited duration of the generator.<sup>8</sup>

Three neurostimulation techniques have been widely developed in clinical practice: posterior spinal cord stimulation, peripheral nerve stimulation, and deep brain stimulation. Of these three options, peripheral nerve stimulation is the instrument to be considered on an early basis in patients with severe peripheral neuralgia involving a single nerve, and refractory to other treatment modalities. This technique involves the placement of a subcutaneous-subdermal neurostimulating electrode in the territory innervated by the target nerve.<sup>8-11</sup>

We present a case of peripheral neurostimulating electrode implantation at the right supraorbital level to control headache with severe neuropathic pain secondary to sectioning of the ipsilateral supraorbital nerve following frontal bone fracture.

### CASE REPORT

A 34-year-old man was diagnosed with severe headache secondary to supraorbital neuralgia after a work-related accident in March 2002 (fall from a height of 9 m),

resulting in frontal fracture of the right zygomatic arch-malar region, and displaced fractures of both wrists. Surgical reduction of the facial fracture was carried out with osteosynthesis and placement of a titanium miniplate. Surgical reduction with osteosynthesis of the left wrist was also carried with external fixation of the right wrist fracture.

Following surgery, the patient developed right hemi-cranial headache extending from the orbital zone to the suboccipital region. The pain was of a daily nature and neuropathic character (described by the patient as an itching and deep burning sensation—with a visual analog scale [VAS] score of 10), and was associated to photophobia triggering intense, stabbing, and dull pain attacks.

Initial pharmacological treatment during the first 2 months provided no significant response (carbamazepine 800 mg/day, topiramate 200 mg/day, gabapentin 1200 mg/day, clonazepam 4 mg/day, and amitriptyline 25 mg/day). The patient wore polarized sunglasses continuously because of photophobia and rated the pain with a VAS score (0 to 10) as 9 to 10.

Subsequently, invasive techniques were trialed: anesthetic blocks were performed with ropivacaine (0.2%) 5 mg and posterior phenolization of the supraorbital nerve—with very transient improvement. The patient described 10 hours of pain relief (VAS score: 3 to 4) with local anesthetic injection and approximately 1 day after phenol local injection (VAS score: 4 to 5) performed 1 week after the ropivacaine block.

In July 2002, following psychological evaluation,<sup>12</sup> the patient was included in a peripheral nerve stimulation program. The first stage of the procedure (test period) involved implantation, under monitored sedation and local anesthesia, of a neurostimulation electrode (Quad PISCES<sup>®</sup>, Medtronic Neurological Inc., Minneapolis, MN, U.S.A.). After tunneling to the supraclavicular region, the electrode was connected to an



**Figure 1.** Generator location at abdominal level (projected over the right iliac ramus) and nerve stimulation cable trajectory to the right clavicular zone. Anteroposterior radiological projection.



**Figure 2.** Location of the electrode in the right orbit, in lateral radiological projection with visualization of osteosynthesis material in relation to facial fracture.

external generator (Figure 1). The patient was placed in lateral decubitus position on the operating table, inserting a curved needle from the temporal region to the supraorbital zone through which (under fluoroscopic guidance) the electrode was advanced and its tip was positioned over the sphenoidal sinus, marking the curve of the orbit (Figures 2 and 3). Verbal feedback from the patient ensured that stimulation optimally covered the painful zone—thus indicating adequate stimulation of the supratrochlear and supraorbital nerves. The position of the electrode was considered adequate, not only by fluoroscopic verification of its position, but also by intraoperative stimulation testing in the conscious patient. Likewise, low amplitude and a pulse width at the lower limits are requirements for correct insertion of the electrode.<sup>13</sup>

After placing the electrode in the target zone, an incision was made at clavicular level, and a trocar was used to tunnel across the anterior surface of the chest and abdomen to prepare for insertion of the electrode and connection at its lower end to the generator. Posteriorly, the operation was repeated on the lateral surface of the neck, retroauricular level, and along the



**Figure 3.** Location of the electrode tip over the right sphenoidal sinus, marking the curve of the orbit in anteroposterior radiological projection.

temporal zone, following a line between the upper margin of the ear and the external angle of the eye—followed by internalization in the supraorbital zone. A total of five incisions were required.

The catheter was affixed by an anchoring system to the fascia of the temporal muscle close to the orbit, thus avoiding possible retrograde displacement. For the rest of connections, we used loop fixations to avoid displacement with movements of the patient. Pre- and postoperative antibiotic coverage was provided with ciprofloxacin 500 mg/12 hours for 7 days.

After a 14-day test period, and having confirmed patient satisfaction and acceptance of the system with improvement of the symptoms (VAS score: 2), the generator was implanted by preparing a subcutaneous pouch in the abdominal region above the navel using a single midline incision to a depth of approximately 2.5 cm from the skin surface.

Following definitive implantation, the patient reported improved symptom control and an adapted life style. The medication was likewise gradually reduced until total discontinuation. Battery exhaustion occurred in July 2006. A Synergy generator (Medtronic®) was implanted with maintenance of the previous electrode.

## DISCUSSION

Supraorbital neuralgia is one of the possible conditions underlying frontal headache. A number of pathogenic mechanisms may be involved—the most prevalent secondary presentation being direct trauma to the supraorbital nerve because of its superficial trajectory.<sup>1,2</sup> A number of conservative as well as invasive (surgical) treatments have been advocated, though pharmacological management is the most common approach.<sup>3</sup> The use of opioids and neuromodulators—including antiseizure drugs and antidepressants—either alone or in combination, affords pain relief in approximately one-half of all patients with headache because of supraorbital neuralgia—particularly in the idiopathic presentations of the disorder. However, many cases, especially those with neuralgia secondary to trauma, seem less responsive to oral drug treatment over time—leading to progressive dose escalation, with undesirable effects in some cases, and difficulty maintaining the clinical benefits.<sup>14</sup>

Supraorbital nerve block with local anesthetics affords immediate relief in practically all cases, though the effect is transient. Nevertheless, nerve block is generally considered the standard approach for confirming the diagnosis of peripheral neuralgia; the disappearance of pain with block is considered a prerequisite for considering implantation of a peripheral neurostimulator.<sup>15</sup>

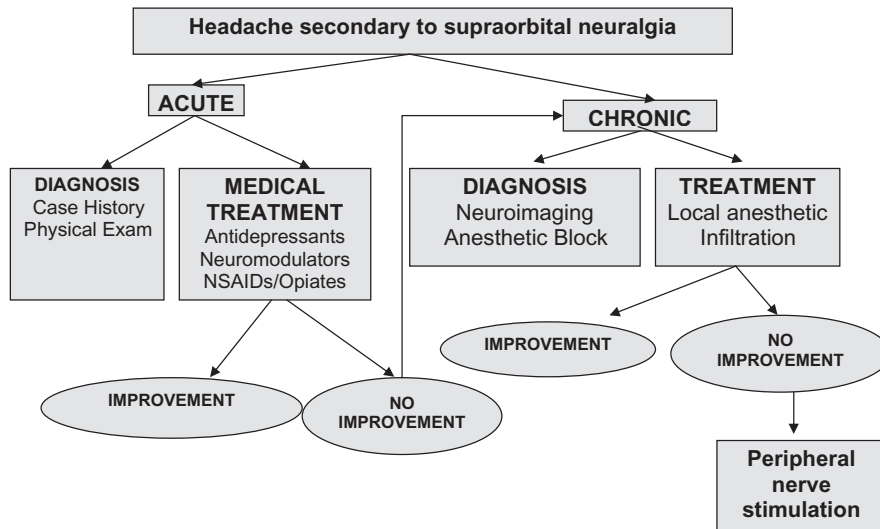
Invasive neuroablative procedures can be effective in the treatment of some pain syndromes refractory to medical management. However, their use is presently controversial, as the destruction of nerve tissue is irreversible. Moreover, the patient may develop painful neurinomas or causalgia that can worsen control of the neuropathic pain.<sup>3</sup> In contrast to neurodestructive procedures, neuromodulation with nerve stimulation is fully reversible. If the patient decides not to continue use of this treatment modality, the system can be removed usually without untoward sequelae.<sup>16,17</sup>

Peripheral neurostimulation is based on the same principles as posterior spinal cord stimulation. One of the difficulties of this technique, however, is correct positioning of the electrode to ensure adequate stimulation of the target zone, as scarring corresponding to some previous intervention or secondary to trauma may impede placement of the electrode. Furthermore, it must be taken into account that in order for nerve stimulation to be effectively transmitted, the nerve must be anatomically intact. Taken together, these aspects require neurostimulation electrode placement under local anesthesia, to allow dialogue and feedback between the physician and patient to ensure that nerve stimulation covers as much of the painful zone as possible. To ensure that neurostimulation will afford adequate long-term pain control, a 14-day test period is required before definitive internalization of the system.<sup>16–18</sup> The definitive generator implantation in this patient was performed in the abdominal region, above the navel. In this particular case, the abdominal location permitted better tissue coverage than could be obtained in the lumbar paraspinous region.

In view of the above considerations, we propose an initial diagnostic-therapeutic algorithm (Figure 4).

## CONCLUSIONS

Despite the technical difficulties involved, peripheral neurostimulation may be considered for early application in patients with severe peripheral neuralgia refractory to other therapies. In the case presented, significant improvements were obtained in analgesia, and in the occupational, personal, and social life of the patient. Although clinical experience with this therapy is limited, chronic peripheral neurostimulation is a promising option for patients with headache secondary to supraorbital neuralgia refractory to other treatments.



**Figure 4.** Proposed diagnostic-therapeutic algorithm in headache secondary to supraorbital neuralgia.

## REFERENCES

1. Penas-Prado M, Martinez-Salio A, Porta-Etessam J, et al. Neuralgia supraorbitaria postraumática: una entidad benigna. *Rev Neurol.* 2007;44:89–91.
2. Headache Classification Subcommittee of the International Headache Society. The international classification of headache disorders, 2nd ed. *Cephalalgia.* 2004;24(suppl 1):1–160.
3. Sjaastad O, Stolt-Nielsen A, Pareja JA. Supraorbital Neuralgia. On the clinical manifestations and a possible therapeutic approach. *Headache.* 1999;39:204–212.
4. Pareja JA, Caminero AB. Supraorbital neuralgia. *Curr Pain Headache Rep.* 2006;10:302–305.
5. Sjaastad O, Petersen HC, Bakketeig LS. Supraorbital neuralgia. Vaga study of headache epidemiology. *Cephalalgia.* 2005;25:296–304.
6. Caminero AB, Pareja JA. Supraorbital neuralgia: a clinical study. *Cephalalgia.* 2001;21:216–223.
7. Meyerson BA. Neurosurgical approaches to pain treatment. *Acta Anaesthesiol Scand.* 2001;45:1108–1113.
8. Stojanovic MP. Stimulation methods for neuropathic pain control. *Curr Pain Headache Rep.* 2001;5:130–137.
9. Blond S, Touzet G, Reyns N, et al. Neurostimulation in the treatment of chronic pain. *Neurochirurgie.* 2000;46:466–482.
10. Alo KM, Holsheimer J. Next trends in neuromodulation for the management of neuropathic pain. *Neurosurgery.* 2002;50:690–703.
11. Krames E. Implantable devices for pain control: spinal cord stimulation and intrathecal therapies. *Best Pract Res Clin Anaesthesiol.* 2002;27:2584–2591.
12. Monsalve V, De Andrés JA, Valia JC. Application of a psychological decision algorithm for the selection of patients susceptible to implantation of neuromodulation systems for the treatment of chronic pain. A proposal. *Neuromodulation.* 2000;3:191–200.
13. Bonezzi C. Location and type of electrodes and parameters of electrical current determining the clinical results of spinal cord stimulation. *European J Pain.* 1999;3:399–401.
14. Martelletti P, Van Suijlekom H. Cervicogenic headache: practical approaches to therapy. *CNS Drugs.* 2004;18:793–805.
15. Bovim G, Sand T. Cervicogenic headache, migraine without aura and tension-type headache. Diagnostic blockade of greater occipital and supraorbital nerves. *Pain.* 1992;51:43–48.
16. Slavin K, Nersesyan H, Wess C. Peripheral neurostimulation for treatment of intractable occipital neuralgia. *Neurosurgery.* 2006;58:112–119.
17. Popeney CA, Alo KM. Peripheral neurostimulation for the treatment of chronic, disabling transformed migraine. *Headache.* 2003;43:369–375.
18. Weiner RL. Peripheral nerve stimulation. In: Burchiel KJ, ed. *Surgical Management of Pain.* New York: Thieme Medical Publishers, Inc.; 2002:498–504.