Stimulation of internal capsule for relief of chronic pain

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The severe spontaneous pain associated with lesions of the central nervous system has been successfully suppressed by electrical stimulation of the posterior limb of the internal capsule. The physiological basis for this result is not understood but the authors' hypothesis is that the pain suppression is due to stimulation of parietal corticofugal inhibitory fibers.

**KEY WORDS** • pain • electrical stimulation • paraplegia • internal capsule • parietal cortex • thalamus

Electrical stimulation of specific areas in the central nervous system has shown promise in the treatment of chronic pain syndromes of both peripheral and central origin. It has been demonstrated that electrical stimulation through electrodes placed over the dorsal columns of the spinal cord can effectively ameliorate many cases of pain arising in a variety of peripheral sites, and of varying etiology. Electrical stimulation of subcortical sites in the brain has also been demonstrated to be effective in the treatment of certain severe chronic pain states. Relief has been reported from stimulation in the septal region, caudate nucleus, and posterior diencephalon.

We have recently reported control of intractable pain associated with facial anesthesia dolorosa in four patients. The stimulating electrodes were placed in the nucleus posterior ventralis medialis (PVM).

Encouraged by this initial success with diencephalic stimulation in the treatment of facial anesthesia dolorosa when the thalamic somatosensory neuron was stimulated, we next explored the possibility that similar stimulation of the somatosensory neurons in the region of the posterior limb of the internal capsule might provide relief from the pain associated with lesions of the central nervous system.

**Method**

Patients with the syndrome of pain associated with cerebral pathology were initially selected. Subsequently the technique was extended to patients with pain related to spinal cord injury.

The operative technique for the insertion of the stimulating platinum electrodes has been previously described. The multicontact flexible electrodes* are implanted stereotaxically.

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*The electrodes and electronic hardware were manufactured by Medtronic Inc., Minneapolis, Minnesota.
Stimulation of internal capsule to relieve chronic pain

The effect of various parameters of stimulation via the temporarily externalized electrode leads upon the patient’s spontaneous pain is evaluated for several days. If no benefit is achieved, the electrode is removed. If effective pain relief results, however, the electrode is connected to a subcutaneous receiving unit for permanent transcutaneous stimulation.

Case Reports

Case 1

This patient has been reported in detail elsewhere, and is mentioned here only to complete our series of five cases. This 47-year-old man sustained a traumatic subdural hematoma and contusion of the right temporal parietal cortex, which resulted in a transient left hemiparesis and permanent hemianesthesia. Six months later he began to experience painful dysesthesias involving the left forearm, hand, and lower leg; these increased in severity over a period of 1½ years. The neurological examination demonstrated a right parietal lobe syndrome, and the pain syndrome was thought to be related to a destructive lesion of the parietal cortex.

The stimulating electrode was placed in the right internal capsule at the level of the posterior commissure in the coronal and horizontal planes and 25 mm lateral to the midline. The initial stimulation provided immediate relief, which lasted for 4 days. He has maintained excellent control of pain over the succeeding 12 months. His most effective relief is obtained by a time modulated stimulus (ramping current). This stimulus envelope progresses linearly for 30 seconds from 0.5 to 4.5 V and then repeats. The stimulus pulse is biphasic with a 0.4 msec duration. The frequency can be controlled by the patient. The optimal frequency has been between 100 and 150 Hz. The patient obtains effective pain relief following stimulation for periods varying from hours to several days.

Case 2

This 54-year-old woman suffered a cerebral embolus resulting in a spastic right hemiparesis and a right visual field defect. Four months later she developed a constant aching pain involving the left arm and leg. In April, 1971, a left stereotaxic thalamotomy was performed with relief of pain for 2 months. The thalamic lesion was enlarged and a cingulotomy was performed in May, 1972, but there was no benefit from these procedures. In March, 1973, a stimulating electrode was placed in the left internal capsule (coordinates 8 mm, posterior 4 mm above the mid AC-PC point, and 23 mm to the left of the midline). Initial stimulation of 100 Hz did not totally relieve the patient’s pain, but an estimated 50% to 75% suppression of pain is now being achieved using a ramping current at 30 to 60 Hz.

Case 3

This 60-year-old man suffered a stroke in 1967, resulting in a complete left hemiplegia which cleared in a few days. Two years later there was a gradual onset of pain involving the left arm and leg including the fingers and toes. This became progressively more severe and in the 2 years prior to admission the patient required meperidine hydrochloride every 4 to 6 hours for relief. The pain was described as deep and burning. Stimulating electrodes were stereotaxically implanted on August 30, 1973 (coordinates, posterior 14 mm, 1 mm inferior and 20 mm lateral to the mid AC-PC point). Pain relief following initial brief stimulation lasted 10 days and was maintained with satisfactory masking of the pain by intermittent stimulation for 1 month. After this initial period, although stimulation produced paresthesias in the painful area, it no longer suppressed the pain. A review of the target coordinates suggested that the electrode had been placed in the lateral posterior border of the nucleus posterior ventralis lateralis (PVL) of the thalamus rather than in the internal capsule. The patient was, therefore, rehospitalized and the electrode portion shifted 7 mm further laterally. Stimulation at this location has augmented the degree of relief from spontaneous pain, although it is not complete.

Case 4

This 79-year-old woman suffered the sudden onset of right hemiparesis and slurred speech in 1969. She recovered completely in 3 days. Two weeks later the symptoms suddenly recurred, now associated with “numbness” of the right arm, and was
followed 3 weeks later by spontaneous severe burning pain involving the right hand, arm and shoulder. This pain was unremitting and not relieved by trials of anticonvulsant, psychopharmacological, and analgesic drugs in increasing dosage. In October, 1970, stimulating electrodes were implanted in both the thalamic sensory relay nucleus (VPL) (8 mm posterior, 1 mm above and 14 mm to the left of the mid AC-PC point), and the posterior limb of the internal capsule (12 mm posterior, 1 mm above, and 24 mm lateral to the mid AC-PC point). Stimulation of the thalamic electrode resulted in unpleasant paresthesias and augmentation of the pain in the right arm. Stimulation of the internal capsule electrode was effective in producing paresthesias which not only were pleasant but successfully suppressed the spontaneous pain. Stimulation of the externalized lead of the internal capsule electrode for 1 month at home successfully suppressed the pain.

The patient was readmitted and at a second operation (5 weeks after the initial electrode implantation), the electrode was connected to the subcutaneous receiver. She continued to obtain successful relief of pain until September, 1973, when she suffered a third "stroke" which resulted in left hemiparesis and severe dysarthria. She is now bedridden, severely incapacitated and the internal capsule stimulation is no longer being used.

Comment on Cases 1-4

These four patients, each presenting with a clinical syndrome of pain and hyperalgesia following lesions involving central sensory pathways, achieved significant pain relief by electrical stimulation of the posterior limb of the internal capsule.

Two additional patients suffering from longstanding pain associated with a presumed lateral medullary syndrome were tested with stimulating electrodes placed in both the primary sensory thalamic nuclei and the posterior limb of the internal capsule without any effect on the spontaneous pain. Consequently, after a trial of stimulation for 10 days, the electrodes were removed.

The lesions associated with the clinical syndrome in the four patients treated successfully were thought to involve second or higher order relay neurons of the somatosensory projection system in the diencephalon and parietal cortex. It seemed logical, therefore, to apply the same methodology to central pain associated with lesions of the spinal cord. The following case was treated in this manner.

Case 5

This 36-year-old man suffered a gunshot wound of the upper thoracic spine in 1968. Emergency laminectomy disclosed a severed cord. Pain did not become a problem until 1970 when gradually two types of pain appeared spontaneously and became progressively worse. The first was sharp, lancinating, intermittent pain referred to both feet. The second was a constantly burning pain that at times was deep and aching and was felt in both the medial and lateral aspect of each leg below the knee. By the time of his admission in May, 1973, he was severely depressed and demanding large amounts of Oxycodone.

Stimulating electrodes were implanted in each internal capsule (coordinates 1 mm posterior to the posterior commissures, and 25 mm lateral to the midline). Stimulation via the externalized electrode leads completely suppressed both types of pain with the relief often persisting for 1 to 2 hours after the stimulus. He used the externalized electrodes for a period of 1 month at home and was then rehospitalized for the internalization procedure; during this procedure the position of the electrode in the left internal capsule was inadvertently changed. Subsequent activation of the left electrode produced no paresthesias and no pain suppression. However, stimulation of the right internal capsule electrode was still effective. This operative mishap provided a fortuitous control in judging the successful suppression of pain in the left leg by stimulation of the right internal capsule electrode.

In November, 1973, the patient returned to the hospital and the left internal capsule electrode was replaced in its original position. Since that time he has experienced complete control of pain by stimulating each electrode 3 to 4 hours per day. He now requires no analgesic medication.

Discussion

The severe, spontaneous pain resulting from central nervous system injury and commonly referred to as "thalamic pain" has been effectively suppressed by electrical
Stimulation of the posterior limb of the internal capsule. Stimulation in the same structure was also effective in reducing the pain associated with spinal cord transection.

We believe this relief of pain is a physiological phenomenon rather than suggestion. The stimulus threshold was critically related to relief, and pain control had not been achieved in any of the patients by previous treatments which might have elicited a placebo effect.

Although the evaluation of treatment results in patients with chronic pain is fraught with difficulties, we believe that the present cases demonstrate convincingly the value of chronic internal capsule stimulating electrodes in cases of pain of central origin (Table 1). The critical location of the electrode tip in Cases 4 and 5 supports the theory that the effect is specific to the structure stimulated rather than due simply to distraction, masking or suggestion (Table 2).

Although the mechanism of pain relief in these cases is not clear, some observations suggest a possible explanation for this effect of electrical stimulation of the internal capsule. A common factor in all of these patients was a spontaneous syndrome resulting from neuronal loss in central somatosensory pathways. There is evidence that the spontaneous pain syndromes resulting from such central lesions may be due to disruption of normal inhibitory connections between certain somatosensory relay neurons. The successful suppression of pain in these patients may be due to activation of inhibitory fibers which arise from the parietal cortex. Fibers arising in the cortex have been demonstrated to have an inhibitory effect at thalamic and spinal cord levels. Fibers ascending in the posterior limb of the internal capsule could also have a direct or recurrent inhibitory effect on cortical neurons.

Regardless of the mechanism of pain suppression, this technique is a simple, safe and reversible means of providing symptomatic relief for those often desperate patients whose pain is usually refractory to narcotic medication and destructive lesions.

### TABLE 2

**Placement of the electrode tip**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sagittal (mm)</th>
<th>Horizontal (mm)</th>
<th>Coronal (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>13 posterior</td>
<td>0</td>
<td>25 lateral</td>
</tr>
<tr>
<td>2</td>
<td>8 posterior</td>
<td>4 above</td>
<td>23 lateral</td>
</tr>
<tr>
<td>3</td>
<td>14 posterior</td>
<td>1 below</td>
<td>27 lateral</td>
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</tr>
<tr>
<td>5</td>
<td>14 posterior</td>
<td>0</td>
<td>25 lateral</td>
</tr>
</tbody>
</table>

* Reference point is the midpoint on a line drawn between the anterior and posterior commissures; coordinates were calculated from the Schaltenbrand and Bailey Atlas.
References


10. Richardson DE, Akil H: Personal communication 1, 1973


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