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Chronic Thalamic and Internal Capsule Stimulation for the Control of Central Pain

Yoshio Hosobuchi, M.D., John E. Adams, M.D., and Burt Rutkin, M.S.

Department of Neurological Surgery, University of California School of Medicine, San Francisco

Thalamic electrical stimulation by means of chemically implanted electrodes and an externally placed transmitter was effected in patients with central pain. Good response was obtained in four patients with anesthesia dolorosa, four with thalamic syndrome, and one with paraplegic pain. Two patients with medullary syndrome experienced no relief. The mechanism of thalamic stimulation in the relief of pain is discussed and further clinical trials are anticipated.

THE literature[3,4,6] documents well that electrical stimulation of certain foci in the brain can mask the aversive properties of pain. White and Sweet[8] reported Mark and Erwin's observations that stimulation of the contralateral thalamic sensory nucleus (PVM) at 50 Hz (using biphasic square waves) produced a tingling sensation that "displaced," or masked, the spontaneous pain of postherpetic neuralgia. In the last four years, we have observed similar pain relief from acute stimulation of the sensory nucleus during thalamotomy in four patients with anesthesia dolorosa. All four patients experienced immediate pain relief which was constant when the current was on and persisted for only two or three minutes after the current was off. All patients experienced the paresthesia that characteristically occurs with stimulation of the sensory nucleus.

Based on Erwin and Mark's results and our own observations of anesthesia dolorosa, we treated five patients with facial anesthesia dolorosa after retrogasserian rhizotomy by stimulation of the PVM through stereotactically placed, chronically implanted electrodes.[7] In four patients, facial pain was masked by electrically induced paresthesia; the fifth continued to experience pain in spite of the induced paresthesia.

All electrodes were initially externalized through separate scalp stab wounds for self-stimulation. When the provisional period of stimulation established the effectiveness of this method for controlling their facial pain, the patients were readmitted to the hospital for permanent implantation of platinum bipolar electrodes which were connected to subcutaneously implanted units receiving pulses across intact skin from an externally placed transmitter.

The positive results in four of five cases of facial anesthesia dolorosa encouraged us to extend the application of this nondestructive measure for treating pain problems arising from lesions in the central nervous system, problems which have proved extremely difficult to manage in the past.

Eight patients with dysesthesia characteristic of central sensory pathway lesions underwent acute ipsilateral sensory thalamic stimulation; four patients had thalamic syndrome, two from medullary ischemic lesions, one from cervical cordotomy dysesthesia and one from paraplegic pain. All patients experienced pain through the induced paresthesia without relief. Acute stimulation of the posterior portion of the ipsilateral internal capsule produced remarkable pain relief in four patients with thalamic syndrome and one paraplegic patient with pain below the sensory level. Three patients with thalamic syndrome and one patient with paraplegic pain now have chronically implanted internal capsular

Reprint requests: Department of Neurological Surgery, University of California Hospital, San Francisco, CA 94122

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electrodes that can be activated from an external system and that provide satisfactory relief from dysesthesia.

A similar method of treatment has been used for a patient with pain secondary to parietal cortical injury. He too has obtained satisfactory relief through intermittent self-stimulation of the ipsilateral internal capsule. Two patients with medullary syndrome did not receive pain relief through this method.

Comment

The mechanism is unknown by which stimulation of the thalamic sensory nuclei suppresses pain in facial anesthesia dolorosa.

If the clinically observed syndrome of facial anesthesia dolorosa is the physiologic correlate of deafferentation hyperactivity as observed in the feline brainstem following retrogasserian rhizotomy,[2] projected centrally this spontaneous neuronal hyperactivity may be inhibited or altered by stimulation of the main sensory nucleus.

No theory postulated to date can account for the myriad factors involved in thalamic pain syndrome;[5] therefore, the efficacy of the internal capsular stimulation for this condition still must be considered an enigma. However, if thalamic pain results from the liberation of thalamic activity, which is normally inhibited by the cerebral cortex,[5] the stimulation of the internal capsule may activate such a descending inhibitory pathway, thus displacing the spontaneous syndromic dysesthesia by indused paresthesia.

Obviously, further studies are required to determine the operative pathway of thalamic and internal capsular stimulation in controlling central pain. Our favorable results with the patients described certainly warrant further clinical trials of nondestructive approaches to the management of difficult pain problems.

Note

As of December, 1974, the authors reaffirm the validity of their conclusions.

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