SUCCESSFUL PAIN RELIEF BY MOTOR CORTEX STIMULATION FOR EXTENSIVE THALAMIC PAIN—CASE REPORT

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Abstract: Thalamic pain is known as the most difficult pain syndrome to treat. This paper describes our case treated successfully by motor cortex stimulation for extensive thalamic pain. A 59-year-old man with left thalamic pain was readmitted two years after an episode of right thalamic hemorrhage. He complained of tearing pain in the left eye, burning pain in the left upper and lower extremities, and chest pain. After one week of test stimulation, permanent implantation using two electrode arrays to include the area of the lower extremity was done. Excellent pain relief has been sustained for 10 months.

Index Terms

thalamic pain, motor cortex, brain stimulation

INTRODUCTION

The concept of thalamic pain was established by Dejerine[1] in 1906. It is the central pain associated with cerebrovascular disease around the thalamus. Defferentiation pain (DP) secondary to central nervous system (CNS) lesions, including thalamic pain, is known as the most obstinate pain syndrome[2)3)]. Although chronic stimulation of the thalamic relay nucleus for thalamic pain has been reported to produce unsatisfactory results, Tsubokawa et al[3)] reported that excellent pain control was obtained by chronic motor cortex stimulation in patients with thalamic pain.

This paper reports a case of extensive thalamic pain in which motor cortex stimulation gave remarkable relief of the pain.

CASE REPORT

On May 1, 1991, a 59-year-old man was admitted to Osaka Neurological Institute with left hemiparesis (2/5) and hemisensory disturbance. Previous medical treatment had included daily oral ingestion of 20 mg of nifedipine and 30 mg of sodium depapril for hypertension. General physical conditions were normal. Computed tomography (CT) of the brain revealed a mild degree of right thalamic hemorrhage (Fig. 1). Cerebral angiography was conducted on admission, and demonstrated no abnormal findings (e. g. no aneurysm, no arteriovenous malformation, no occlusion of major vessels). The symptoms were improved considerably by

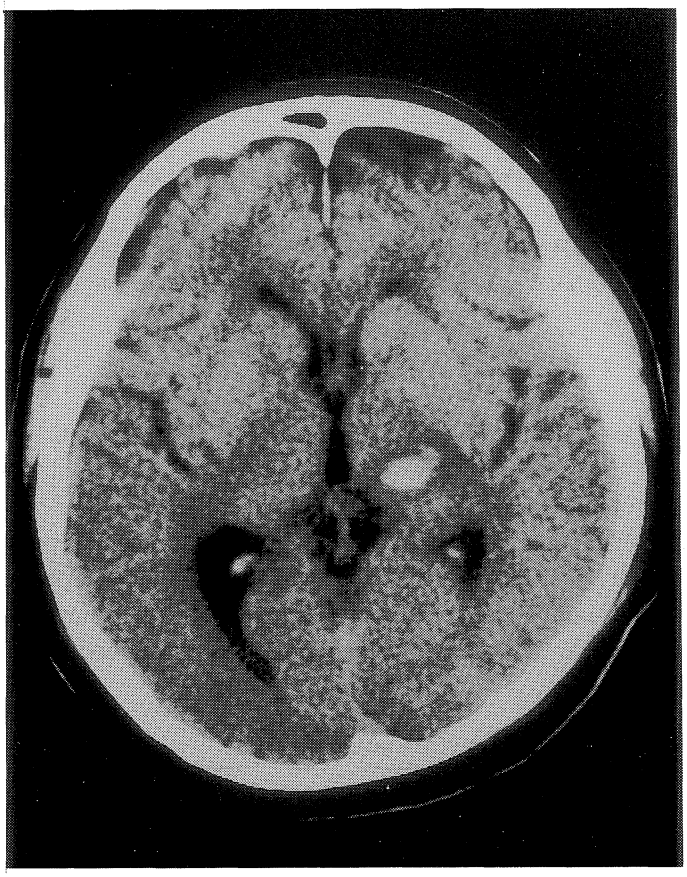
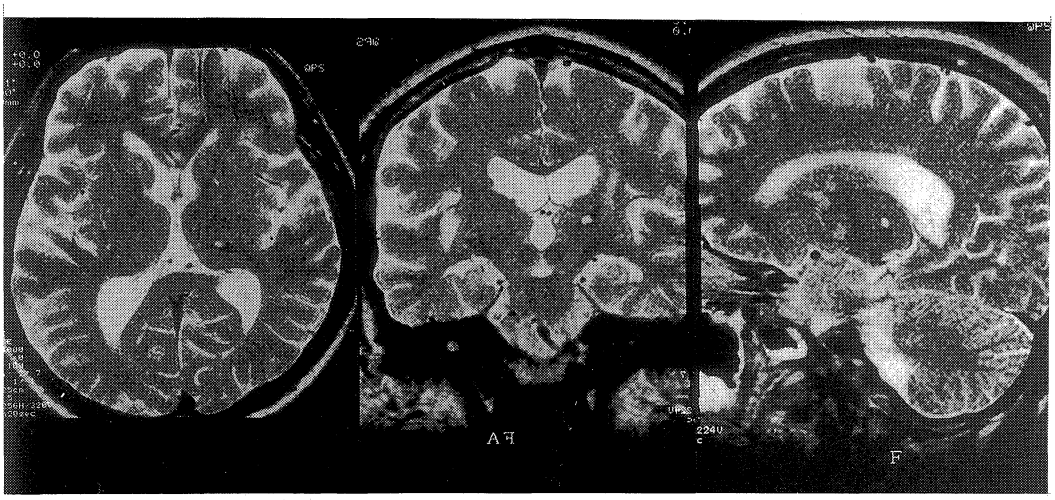


Fig. 1. Computed tomography (CT) reveals the right small thalamic hemorrhage.

glycerol. He was discharged with mild left hemiparesis (4/5) and hypesthesia after a 22-day -admission. He complained of spontaneous pain in the left side of the body since June, 1992. Although he had been treated with various agents including anticonvulsants, antidepressants, and anodynes, these agents exerted no beneficial effect at all. The pain became increasingly intractable, and he could not move because of the pain. He desired to commit suicide. We proposed trying chronic motor cortex stimulation, a new therapeutic approach. He agreed, and was readmitted on April 28, 1993. Neurological examination showed left hemiparesis (3/5), which was aggravated by pain, and analgesia in the left side of the body. Light tactile stimuli applied to the painful area yielded a dysesthesic response. Fine discrimination of light tactile stimuli was very poor. He complained of tearing pain in the left eye, burning pain in the left upper and lower extremities, and chest pain. The symptoms were so severe that he felt as if he were being crushed by a crusher. Magnetic resonance imaging (MRI) demonstrated a very small spotty low intensity area on T1 weighted images and a high intensity area on T2 weighted images in the left ventral posterolateral nucleus of the thalamus (Fig. 2). Serial single photon emission computed tomography (SPECT) demonstrated a remarkable hypoperfusion area in



A B C

Fig. 2. Axial (A), coronal (B) and sagittal (C) magnetic resonance imagings (MRI) demonstrate a very small spotty high intensity on T2 weighted images in the left ventral posterolateral nucleus of the thalamus.

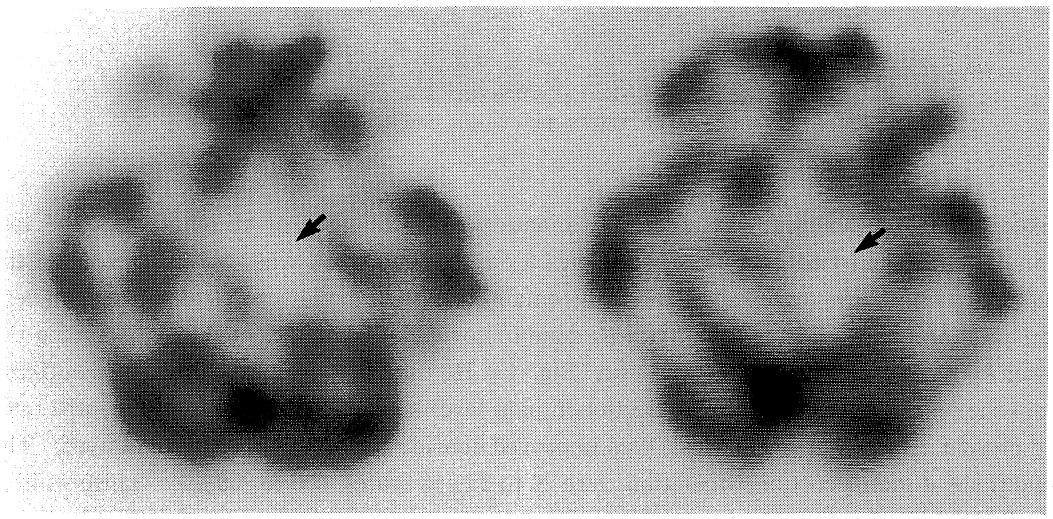


Fig. 3. Serial single photon emission computed tomography (SPECT) demonstrates remarkable hypoperfusion area in the right basal ganglia (arrow).

the right basal ganglia (Fig. 3). The pain was morphine-resistant and barbiturate-sensitive in each test. An electric array (Medtronic, Inc., Minneapolis, Minnesota), having four plate electrodes 0.5 cm in diameter arranged at 1 cm intervals, was implanted by small-scale craniotomy under local anesthesia. The location of the precentral gyrus was confirmed by monitoring somatosensory evoked potential (SEP). Stimuli were delivered by monophasic square wave pulse (rate : 20 pps, pulse width : 250 μsec, voltage : 5 V), and muscle contraction of the left upper extremity and pain relief were achieved during the operation. This effect

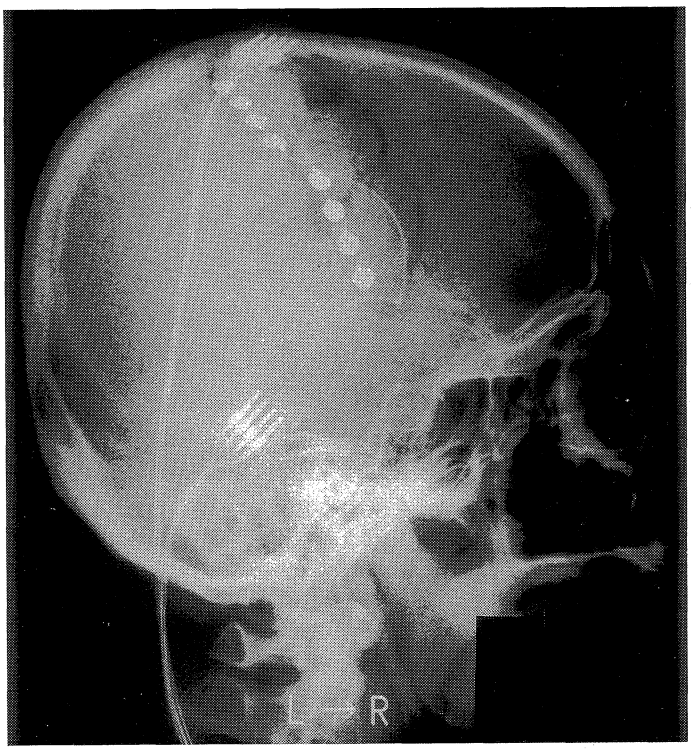


Fig. 4. Roentgenogram shows permanent implantation using two electric arrays.

lasted for 4 to 6 hours following the termination of 10 minutes of stimulation. The test stimulation was carried out five or six times a day for one week when he suffered from the thalamic pain. However, he complained of remaining pain in the lower extremity. Permanent implantation of two electrode arrays was done to cover the whole affected area including the lower extremity under general anesthesia (Fig. 4). He used this system only when he suffered from the pain, and an 80% to 90% reduction in pain level has been obtained for 10 months until the present. The right hemiparesis was improved considerably as the pain was allayed. The conditions of motor cortex stimulation were 20 to 25 pps (rate), 200 to 250 μsec (pulse width), 4 to 5 V (volts) for the upper extremity. The voltage was increased to 6 to 8 V for the lower extremity.

DISCUSSION

Health[4] was the first to perform stimulation for pain relief in 1960. Stimulation of the medial septal area in a woman with carcinoma of the cervix with pelvic pain resulted in pain relief accompanied by euphoria and a feeling of benign intoxication. Reynolds[5] reported that stimulation of small areas of the central gray, which was identical with the periaquedactal area, produced marked behavioral analgesia in rats in 1969. The "gate control theory" proposed by Melzack and Wall[6] provided a theoretical basis for brain stimulation therapy. At present, two anatomical systems are utilized for the control of pain by depth electrodes[7]. One is the

periventricular or periaqueductal gray matter. When this system is utilized, pain relief is thought to be derived from the release of endorphine into cerebrospilal fluid and stimulation of raphe-spinal neurons. The other is the thalamic relay nuclei or the posterior limb of the internal capsule. When this is utilized, the cortex is stimulated, and thereby the input of pain is inhibited. The former approach is effective against morphine-sensitive pain and the latter against morphine-resistant pain.

Deep brain stimulation such as thalamic relay nucleus stimulation achieves satisfactory pain control with DP secondary to peripheral nervous system lesions, but is rarely effective with DP secondary to CNS lesions[3)8)]. Tsubokawa et al[3)] reported that in 8 of 11 patients with thalamic pain (73 %), stimulation system was internalized since excellent pain control was achieved during a 1-week test period of precentral gyrus stimulation. The effect of precentral stimulation was sustained in 5 patients (45 %) during more than 2 years of follow-up. Their series included two patients with extensive thalamic pain, who complained of pain in the lower extremity as well as the upper extremity and underwent implantation of two electrode arrays using a relatively high intensity for the lower extremity.

Concerning the mechanisms of pain relief by precentral stimulation, it is postulated that this stimulatdon activates selectively non-nociceptive fourth-order sensory neurons, either ortho dromically or antidromically, which in turn inhibits hyperactive nociceptive neurons within the sensory cortex.

The mechanism of the effects of motor cortex stimulation has not been yet confirmed, but actually this method provides good pain control in some patients with thalamic pain, for which no other adequate therapy is available at present.

REFERENCES

1) **Dejerine, J.** and **Roussy, G.** : La syndrome thalamique. Rev. Neurol. **12**: 521-532, 1906.

2) **Tasker, P. R.** : Deafferentation. *in* Textbook of pain(Wall, P. D., Melzack, R., eds.). Churchill Livingstone, Edinburgh, p119-132, 1984.

3) **Tsubokawa, T., Katayama, Y., Yamamoto, T., Hirayama, T.** and **Koyama, S.** : Chronic motor cortex stimulation in patients with thalamic pain. J. Neurosurg. **78**: 393-401, 1993.

4) **Helath, R. G.** and **Mickle, W. A.** : Evaluation of seven years' of experience with depth electrode studies in human patients. *in* Electrical studies on the unanesthetized brain(Ramey, E. R., O'Doherty, D. S., eds.). PB Hober, New York, p214-247, 1960.

5) **Reynolds, D. V.** : Surgery in the rat during electrical analgesia induced by local brain stimulation. Science **164**: 444-445, 1969.

6) **Melzack, R.** and **Wall, P. D.** : Pain mechanism; A new theory. Science **150**: 971-979, 1965.

7) **Tsubokawa, T.** : The effect of brain stimulation on intractable pain caused by malignant tumor. J. Pain Clinic **2**(4): 473-481, 1981.

8) **Yamamoto, T., Katayama, Y., Tsubokawa, T., Hirayama, T.** and **Koyama, S.** : Motor cortex stimulation for the treatment of thalamic pain patients. Pain Res. **5**: 63-67, 1990.