Cortical Stimulation for Central Pain

To The Editor: Motor cortex stimulation has been reported to relieve thalamic pain (Tsubokawa T, Katayama Y, Yamamoto T, et al: Chronic motor cortex stimulation in patients with thalamic pain. J Neurosurg 78:393–401, March, 1993). This has not been confirmed by some authors. We believe that central pain is sustained by an out-of-balance thalamoparietal oscillatory generator, whose anomalous activity is seen as parietal (and also frontal) cortex hypoactivity on single-photon emission computerized tomography (SPECT). In these cases, parietal cortex stimulation could rebalance this loop.

We have experience with a 54-year-old man who developed sensorimotor deficits due to cervical syringomyelia in 1992, which were much improved by the placement of a syringoperitoneal shunt in February 1993. In August 1992, he developed central pain to his left arm and hemi-torso. Amitriptyline and carbamazepine were ineffective in treating the pain. A propofol test was partially positive. 1992, which were much improved by the placement of a syringoperitoneal shunt in February 1993. In August 1992, he developed central pain to his left arm and hemi-torso. Amitriptyline and carbamazepine were ineffective in treating the pain. A propofol test was partially positive for 20 minutes. Single-photon emission computerized tomography showed right parietal cortex hypoperfusion, and localization was confirmed by magnetic resonance (MR) imaging (Fig. 1). Written informed consent was obtained to parietal cortex stimulation. The rolandic fissure was identified on the skin according to standard topography rules and marked with a pen. A rubber tube filled with a paramagnetic agent (Intralipid 20%) was applied on the skin mark and adjustments were made under MR imaging. Under neuroleptoanalgesia and local anesthesia, a 6-cm-long × 2-cm-wide craniectomy was fashioned on the expected projection of the Penfield homunculus arm–torso area overlying right SI. A quadripolar electrostimulator (model 3587A; Medtronic, Inc., Minneapolis, MN) was fastened onto the dura (Fig. 2); no motor effects could be elicited, but paresthesias were reported. The extension was then connected to the stimulator, tunnelled, and exteriorized. After 3 weeks, the electrocatheter was connected to an implanted pulse generator (ITREL II; Medtronic, Inc.) under general anesthesia. Analgesia was approximately 30% (up to 50% in the morning), with the following parameters: 5 to 7 V; 50 Hz; 300 μsec; 0 to 3 setting; dosing 30 minutes ON, 60 minutes OFF; cyclical; OFF by night. Analgesia was marked by renormalization of the SPECT anomaly during stimulation. The patient resumed an almost normal lifestyle and could even drive again after almost 2 years. A 1-month course of phenobarbital was administered. Serial electroencephalography showed no epileptic foci. Unfortunately, the pain spread to the remaining hemitorso and analgesia on the opposite side vanished. Single-photon emission computerized tomography during stimulation again showed the anomaly.

Parietal cortex stimulation, contrary to the conclusions of Tsubokawa, et al., can be analgesic (placebo stimulation was ineffective); effects may be temporary, as occurs for motor cortex stimulation and all other stimulation techniques. Best results are seen in barbiturate-responsive patients (see Tsubokawa, et al.) or in the case of propofol responsiveness. (A woman with central post-stroke whom we submitted to motor cortex stimulation derived no benefit and was propofol unresponsive.) A major conclusion of our SPECT study is that brain stimulation does not act through a placebo effect, as suggested. Cortical extradural stimulation is an exciting new modality of pain control that must be explored further.

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References