EXTRADURAL CORTICAL STIMULATION FOR NEUROGENIC PAIN AND PARKINSON'S DISEASE.
THE TURIN EXPERIENCE.

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In 1988, a japanese group introduced motor cortex stimulation for central pain (TSUBOKAWA ET AL. 1993). In 1990, Meyerson extended its use to trigeminal neuropathic pain (MEYERSON ET AL. 1993). Later, we found parietal cortex stimulation effective in two cases (CANAVERO and BONICALZI 1995; CANAVERO ET AL. 1998). World experience suggests that cortical stimulation is effective in about half of the total population of central pain patients over the long-term (more than 2 years)(KATAYAMA ET AL. 1998). Predictors of outcome have been sought and found. The japanese group found best results in those patients who were either barbiturate or ketamine responsive, had no or mild motor weakness in the painful area, or had muscle contraction induced on motor cortex stimulation (YAMAMOTO ET AL. 1997; KATAYAMA ET AL.1998).

Certainly, many failures are reported, and some initial successes turn into later failures (CIONI ET AL. 1996; FUJII ET AL. 1997; MAYERSON ET AL. 1993; NGUIEN ET AL. 1997; GARCIA-LARREA ET AL 1997). The barbiturate test may be falsely negative or positive (MIGITA ET AL. 1995) an may induce somnolence (KATAYAMA ET AL. 1998). In 1995, Migita and colleagues reported that transcranial magnetic stimulation (TCMS) may predict the result of cortical stimulation.

We report our series of patients submitted to cortical stimulation.

PATIENTS AND METHODS.
Since December 1993, we implanted 12 patients: 5 patients with central pain, one with both central and neuropathic pain, 4 with neuropathic pain, one whit neuropathic pain with "pure" central generator, and, in 1998, one women with advanced Parkinson's disease. All pain patients were assessed with propofol test (see in CANAVERO ET AL. 1995) and most with SPECT and/or PET (a few). Nine patients ( one of whom from the above-mentioned group) with central or neuropathic pain were submitted to transcranial magnetic stimulation with MAGSTIM Model 200 stimulator or its CALDWELL analog. A round coil was used for motor or SI stimulation at several energization parameters. 100 stimuli in two series with very short break to allow heat dissipation were delivered.

RESULTS.
5 out 11 pain patients had their pain reduced 30 to 100% at short term follow-up. All were propofol responsive, save for one patient in whom pain paroxysms were so short to hinder proper evaluation of the test. All non-responders were propofol-unresponsive, as of central pain. Neuropathic pain is propofol-unresponsive: one neuropathic pain patient was relived. All analgesic effects were lost over a few months, save for one that is continuing to obtain relief. Four patients out of 9 had their
pain lessened by TCMS. One of these who was implanted drew benefit from cortical stimulation. This is the only patient who received both. The one patient with Parkinson's disease drew generalized benefit from motor cortex stimulation on one side with disappearance of rigidity, dyskinesias and improvement of bradykinesia. Two of the successful cases were treated with SI stimulation. Of the whole series, two infection developed and regressed with antibiotics. The first patient was accidentally exposed to high voltage stimulation for too long: pain vanished for 36 hours (only to reappear later) and transient aphasia was observed.

**CASE REPORT 1.**
This woman suffering facial pain following trigeminal iuxtaponine rhizotomy (see in CANAVERO ET AL. 1995,1998) was assessed. She was 100% propofol responsive. TCMS was administered at several energizations (65% to 100%). MI and SI on both sides were stimulated. Only stimulation of left (sic!) SI consistently provided pain relief (about 30%) on a VAS scale on three occasion, with benefit being reported since the 180th impulse and lasting out several minutes. Paresthesias on the left cheek were reported. A quadripolar stimulator (Mod. 3587A,MEDTRONIC) was implanted extradurally on SI. During test stimulation in the OR, left hemifacial paresthesiae (i.e.ipsilateral to the stimulator!) were obtained with motor parameters (2Hz, 450 μsec, 10 V, rapid ramping). For less than two months, patient obtained 100% relief (1V, 25 Hz, 90 μsec, ON 1Hr/OFF 30 min, 3+/0-). Placebo stimulation was ineffective. Stimulation SPECT was normal (R/L:98-102 vs prestimulation 111-112). No epileptic foci were disclosed by EEG.

**CASE REPORT 2.**
This 66 year old women developed an ischemic stroke in 1994. Within a month she complained of continuous, cycling ripping pain in the left hemisoma, particularly the limbs. Neurologically, there was left hemiplegia, touch, pain and thermal anesthesia on the left, tactile and cold allodynia. MRI showed an extensive hemispheric porencephaly plus brainstem ischemic lesions. SPECT highlighted right panhemispheric hypoperfusion (bar the occipital lobe). Somatosensory evoked potentials were normal (!). Propofol test, as per patient 1, reduced the pain from VAS 10 to 7. TCMC, as per patient 1, at 80% energization (more was not tolerated) reduced leg pain from VAS9 to 6.

**CASE REPORT 3.**
This 61 year old hypertensive man developed subacute post-stroke pain in 1994, as his right hemiplegia slowly recovered over six months. He described his pain in the face and arm as burning and ripping and that in leg as aching and band-like. VAS scores ranged between 5 and 10. There was no allodinya. Carbamazepine at 600 mg was ineffective, and so was amitriptyline at about 50 mg (larger doses were precluded by prostatic problems). Intrathecal morphine (1 mg) administered at another institution made the patient comatose. Tramadol was also ineffective. Both propofol test and motor cortex TCMC as per patient 1 were ineffective.

**CASE REPORT 4.**
This women suffered central pain ever since removal of parietal oligodendrogloma. PET showed parietal plus thalamic hypometabolism on the right. Propofol test (and placebo) were ineffective (MAX VAS 10). TCMC stimulation was administered (100*2,2 sec latency, 1 min interval) at 100% energization. No effect was seen.

**DISCUSSION.**
Extradural cortical stimulation is an expensive technique with a very low complication rate (subdural or extradural hematomas).
As for central pain, propofol test is superior to the barbiturate test (see discussion in CANAVERO ET AL. 1995): it reduces brain central pain in 50% of the patients and cord central pain in 80%. Other unpublished Italian experiences confirm these data (Drs. FRANZINI and DARIO). TCMS may predict successful neurostimulation. It was particularly useful in our case 1, in which a wrong site of stimulation would have been otherwise chosen, with attendant failure. Parietal (somatosensory) cortex stimulation is effective as motor cortex stimulation for central pain, provided propofol test is positive. TCMS, but not propofol test, may help decide which neuropathic pain patients to stimulate. In order to produce excitation just underneath the desired point, the magnetic coil should be tilted anteriorly and stimulation delivered with the edge of the round coil to produce localized stimulation. The now available MULTISTIM, which can deliver trains of impulses without interval may be even better. Whereas both propofol and/or TCMS predict immediate successes, they cannot predict long-term success. Further study is required.

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