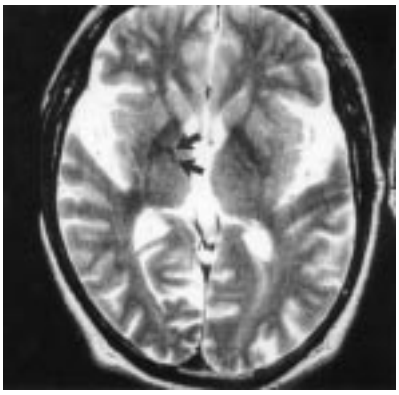

Postoperative MRI confirming the position of stereotaxic lesions in the right thalamus (black arrows).

days with no symptomatic effect or objective change in his AIMS score.

In April 1997 ventral thalamotomy was performed on the right side in two stages under local anaesthesia. A Bennett spheroid guide had previously been inserted under general anaesthesia using CT guidance and a Leksell frame. Details of the lesions are shown in the table. The first lesion was relatively anterior and it reduced the torticollis, neck pain, hypertonia, and the dyskinesia of the contralateral limbs and allowed him to smile and laugh. One week later a second lesion was placed posteriomedial to the first. This abolished the residual "cogwheeling" of the left upper limb and improved his dexterity. There were no surgical complications. Postoperative MRI (figure) 8 months after the procedure confirms the position of the two lesions in the right thalamus.

Twelve months later the patient remains well with minimal dystonic neck movements and no evidence of abnormal posturing of the left arm and off all medication. His AIMS score is now 8/40.

Although the efficacy of thalamotomy has long been recognised in secondary dystonia[6] we are not aware of any reports of its use in drug induced dystonia. The mechanism of drug induced dystonia is not yet known and extrapolating the surgical results for treatment of dystonia of other aetiologies may not be appropriate. The reported mortality from thalamotomy ranges from 0.4% to 6%.[2] Recent experience with pallidotomy indicates an incidence of severe clinical complications of between 2%-8%.[2] Because of the proximity of the optic tract to the globus pallidus persistent visual defects are a well known risk of pallidotomy, up to 14% in one series.[3] It is too early to be certain of long term efficacy but 12 months after operation the patient remains well and off all medication. We conclude that thalamotomy should also be considered in patients with medically refractory drug induced tardive dystonia and dyskinesia.

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Gabapentin in the treatment of painful diabetic neuropathy: a placebo controlled, double blind, crossover trial

Painful neuropathy is a common and disabling problem in patients with longstanding diabetes mellitus. Tricyclic antidepressant drugs and other chronic analgesics have been beneficial in some patients,[1] but no agent successfully relieves pain in most patients and adverse effects often preclude their use in high doses. Anecdotal reports suggest that gabapentin ameliorates pain associated with neuropathy and other neurological conditions with few side effects.[2 3] We conducted a randomised, double blind, placebo controlled trial to study the effect of low dose gabapentin in patients with painful diabetic neuropathy.

We recruited 40 patients with painful diabetic neuropathy who had (1) diabetes for at least 6 months on a stable dosage of insulin or oral hypoglycaemic agent, (2) distal symmetric sensorimotor neuropathy as shown by impaired pin prick, temperature, or vibration sensation in both feet and absent or reduced ankle reflexes, and (3) daily neuropathic pain in the acral extremities, of at least moderate severity, for over 3 months that interfered with daily activity or sleep. Excluded were those with diabetes and chronic renal insufficiency, painful diabetic plexopathy, or lumbosacral polyradiculopathy, peripheral vascular disease, another painful condition, or other cause for neuropathy. Patients were randomly assigned to gabapentin (300 mg capsules) or placebo for 6 weeks (phase I) followed by a 3 week washout period and then crossover (phase II). The dose of gabapentin or placebo was increased by one capsule every 3 days to a stable dosage of one capsule three times daily (900 mg/day) that was maintained throughout the remainder of the treatment period. The low dosage of gabapentin was chosen to minimise adverse effects that might compromise blinding. Treatment with stable dosages of nonsteroidal anti-inflammatory agents or narcotics were permitted during the trial but patients discontinued all other chronic analgesic medications 3 weeks before study entry.

At the beginning and end of each treatment period, patients rated their level of pain over the preceding 24 hours on a 10 cm visual analogue pain scale (VAS), ranging from 0 ("no pain") to 10 ("worst pain ever"). Present pain intensity (PPI, "rate how much pain you have at this moment," using a similar 0–10 scale) and the McGill pain questionnaire (MPQ) were recorded at the initial and final visits of each treatment period.[4] At the end of each treatment period patients provided a global assessment of pain relief: none, mild, moderate, or excellent, as compared with the level of pain preceding each treatment period. The global assessment of pain relief was dichotomised (none/mild *v* moderate/excellent) for purposes of analysis. The protocol was approved by the Institutional Review Board at St Elizabeth's Medical Center and all patients gave written informed consent.

There were 31 men and nine women, with an average age of 62 years (SD 10.9 years, range 43–82 years). All but one had adult onset diabetes mellitus, with a mean duration of 14 years (SD 9.9 years, range 6 months-40 years). Ten had neuropathic pain limited to the feet, 19 had pain in the feet and legs, and 11 had pain in the feet, legs, and hands. The mean duration of neuropathic pain was 4 years (SD 3.5 years, range 4 months-15 years). Twenty five had previously used narcotics or other chronic analgesics to manage their pain.

Nineteen patients were randomised to the active drug and 21 to placebo during the first treatment period. The mean reduction in the MPQ score was 8.9 points with gabapentin compared with 2.2 points with placebo (p=0.03, two sample *t* test). There were no differences in the mean change of the VAS or PPI scores between gabapentin and placebo (table). Fourteen patients reported moderate or excellent pain relief with gabapentin only, six with placebo only, and three with both; 17 reported none or mild relief after both treatments (p=0.11, McNemar's test). There were no serious adverse events. Adverse effects were significantly more common with gabapentin (12 patients) compared with placebo (four patients, p<0.001, McNemar's test). The most common side effects of gabapentin were drowsiness (six patients), fatigue (four), and imbalance (three). All adverse effects resolved promptly after discontinuation of the drug.

Anecdotal reports suggest that gabapentin has beneficial effects in patients with various painful neurological conditions, including HIV neuropathy,[2] postherpetic neuralgia,[2] and reflex sympathetic dystrophy.[3] The mechanism of action of gabapentin in ameliorating pain is unknown, but animal studies suggest that its pain modulating properties may be linked to the release of the neuro-

Comparison of mean change in pain scales between gabapentin and placebo

Pain scale	*Gabapentin*	*Placebo*	*Difference*	*p Value*
MPQ	8.9 (2.3)	2.2 (2.2)	6.7 (3.2)	0.03
VAS	1.8 (0.5)	1.4 (0.3)	0.4 (0.6)	0.42
PPI	1.2 (0.4)	0.3 (0.5)	0.9 (0.7)	0.2
No of patients reporting moderate or excellent pain relief*	17	9		0.11

*Global assessment of pain relief.
MPQ=McGill pain questionnaire; PPI=present pain intensity; VAS=visual analogue scale; numbers in parentheses are SD.

transmitter GABA in spinal cord pathways that modify pain perception.[5]

There was statistical improvement in only one of four end points, the MPQ score, with gabapentin compared with placebo. The MPQ is a valid, consistent, and reliable measure of subjective pain experience, and usually correlates with other measures of pain intensity, including the VAS and PPI scales.[4] We designed the study to have an 80% power to detect a 20% reduction in pain scores, reflecting a modest but clinically important improvement. The mean change of the VAS and PPI scales and the patient's global assessment of pain relief were not significantly different from placebo. We used a crossover design because of its statistical efficiency, but the MPQ and VAS scores did not return to baseline after crossover in patients who received gabapentin in phase I (the washout period was inadequate); therefore, we may have underestimated improvement with gabapentin in the VAS scale that may have been detected using a parallel group design. Furthermore, a limitation of our study was that quantitative measures (for example, nerve conduction studies, quantitative sensory thresholds) were not used to further characterise the type of neuropathy. Because of the heterogeneous nature of neuropathic pain in our study patients, we may not have identified a subset of patients who improved with gabapentin. Alternatively, the dosage of gabapentin may have been too low to induce analgesia in patients with painful diabetic neuropathy, although similar regimens have been reported to be effective in patients with other painful conditions.[2 3]

The results of this study suggest that gabapentin is probably ineffective or only minimally effective for the treatment of painful diabetic neuropathy at a dosage of 900 mg/day.

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Single motor unit activity pattern in patients with Schwartz-Jampel syndrome

Two sisters, 9 and 11 years old, with typical clinical symptoms of Schwartz-Jampel syndrome were investigated. Conventional electromyographic investigation with concentrical needle electrodes in the biceps brachii and tibialis anterior showed continuous muscle activity (myotonic burst, high frequency discharges of single motor units with "bizarre" rhythmic activity). The single motor unit action potential (MUAP) was studied in detail by monopolar surface selective electrode with a small leading off area. The pattern suggests that the muscle membrane alone is the not the only reason for abnormality.

Continuous muscle activity is a prominent symptom in patients with Schwartz-Jampel syndrome. Some authors maintain that this may originate in the nerve or end plate. Lehmann-Horn *et al*[1] showed two muscle membrane abnormalities by voltage clamp and patch clamp techniques and concluded that spontaneous activity in the Schwartz-Jampel syndrome originated in the muscle membrane itself. Arimura *et al*[2] found a normal end plate function and assumed that the motor unit pattern influenced interdischarge interval changes. It is difficult to make a precise analysis of the MUAPs with concentric needle electrodes because of other interfering spontaneous activities. Thus a monopolar surface selective electrode with a small leading off area[3] was employed to obtain a more precise assessment of a single MUAP pattern.

The patients were two sisters, 9 and 11 years old, from consanguineous parents. They displayed short stature, bone deformities (kyphoscoliosis, pigeon breast, short neck, pes equinovarus), facial dysmorphism, muscle stiffness, and missing tendon reflexes in the lower limbs. Concentric needle EMG was performed when the patients were 7 and 9 years old and disclosed abnormality. The needle insertion, mechanical stimulation, and mild muscular contraction induced spontaneous activity. Myotonic discharges (fig 1 A and B) were found in all examined muscles (abductor digiti minimi, quadriceps femoris, tibialis anterior, biceps brachii).There were also spontaneous high frequency biphase potentials. Some of the high frequency discharges appeared as doublets or complex repetitive discharges. Routine nerve conduction studies (motor conduction velocity, distal latency, compound muscle action potentials, and sensory action potentials in upper and lower limbs) were normal.[4] Electromyographic investigations of single MUAPs were performed in biceps brachii and tibialis anterior muscles. Involuntary motor unit activity was recorded by monopolar surface selective electrode with a small leading off area[3] for 30 minutes. A Mistro 5+electromyograph and a Teac type recorder were employed to register the action potentials. Distance between the negative peaks of MUAP was measured with a resolution of 0.1 ms. After applying these electrodes we found single MUAP trains between myotonic discharges. They showed without provoking a burst of activity, as usually happens during needle electromyography.

Motor unit firing began with doublet discharges (fig 2 trace 1). After a few seconds MUAP alternated between doublets and triplets (fig 2 trace 2). and then the motor unit fired with stable triplets (trace 3). Similarly, triple discharges turned into quadruplets, and then multiplets (traces 4 to 11) and the number of firing impulses increased at the end of motor unit discharge. All multiplet impulses were similar in shape.

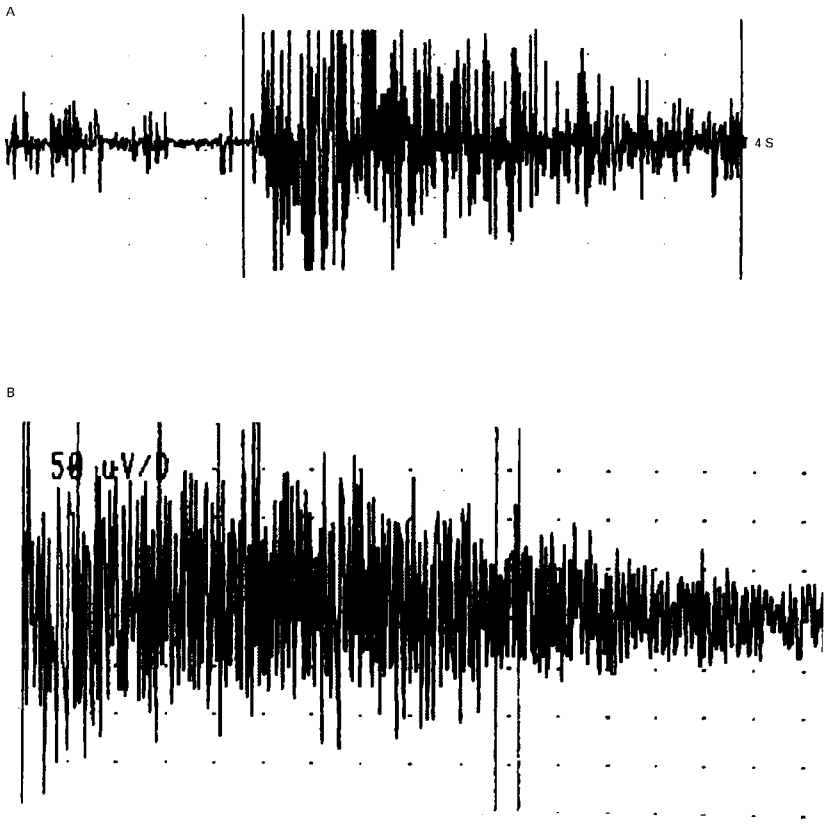

Figure 1 Myotonic discharge recorded by monopolar surface electrode with a small leading off area (A) and needle electrode (B) from biceps brachii muscle.