LETTERS TO THE EDITOR

A neurosurgical Munchausen revisited

In 1983 Henderson *et al* described a young man who was repeatedly admitted to hospital feigning subarachnoid haemorrhage. In at least two of the admissions he developed associated left sided limb weakness and he had undergone numerous investigations including repeated CT head scan, angiography and CSF examination.

In 1986 a patient with the same initials, date of birth and occupation was admitted to Guy's Hospital with a history of severe headache and left hemiparesis. The similarities to the previously published case were recognised and it became apparent that this was indeed the same patient. He was referred for a psychiatric opinion and was admitted for inpatient therapy for a period of two weeks with management following behavioural lines combined with group psychotherapy. There was an apparent improvement and the patient was discharged.

In June 1988 a patient with the same initials but slightly different date of birth was admitted to the neurosurgical ward at the Western General Hospital in Edinburgh with the acute onset of severe headache and left hemiparesis. He was recognised as being the same patient who was admitted to Guy's two years previously with Munchausen's syndrome and enquiry to the general practitioner revealed that there was a dossier containing details of 200 hospital admissions in the previous three years in the United Kingdom, Ireland, Belgium and Holland. The clinical presentation was always the same and the patient had undergone repeated investigation including CT scan and lumbar puncture.

The opinion from the psychiatrists was that the patient had aimed to completely deceive medical practitioners again on this admission and that he had no genuine motivation to accept any help that might be offered to try and modify his behaviour. The patient was therefore confronted, with an immediate resolution in the weakness so that he was able to leave the hospital ward stopping off only to collect money from the Social Work Department to enable him to travel home.

Two months ago the same patient was referred for a neurological opinion at the Western General Hospital for investigation of recurrent headache and hemiparesis. Before admission could be arranged the patient moved to Glasgow and has subsequently been admitted to the Chesterfield and North Derbyshire Royal Hospital with acute headache and hemiparesis which was promptly recognised as being due to Munchausen's syndrome. During this admission there was evidence of numerous admissions to hospitals in Glasgow, Liverpool and Peterborough and when confronted the patient made a rapid recovery and left hospital.

The purpose of this letter is to bring to the attention of general physicians, neurologists and neurosurgeons throughout the United Kingdom of the existence of this patient with Munchausen's syndrome. The evidence suggests that he has repeatedly been admitted to hospital for investigation of acute headache and left hemiparesis and has undergone repeated radiological investigations and lumbar puncture. The frequency of these admissions is difficult to judge but it is of some concern that he has now attended the Western General Hospital on two occasions in the last three years, despite the fact that he knows he may be recognised.

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Henderson LM, Bell BA, Miller JD. A neurosurgical Munchausen tale. *J Neurol Neurosurg Psychiatry* 1983;**46**:437-9.

Rectal apomorphine: a new treatment modality in Parkinson's disease

In 1987 Stibe *et al*[1] reported the successful use of apomorphine to relieve "off period" symptoms in patients with response fluctuations during levodopa therapy for Parkinson's disease. Subsequent publications have confirmed their results.[2,3] Stibe *et al* injected the drug subcutaneously or via an infusion pump, but recently Kapoor *et al*[4] reported that intranasal application of apomorphine was as effective as subcutaneous injection, and suggested that it could become the route of choice.

We report on the results of rectal administration of apomorphine to three patients with severe response fluctuations to anti-Parkinson therapy. The drug was administered as an enema in a concentration of 10 mg/ml at the beginning of the "off period". All three patients had previously improved after subcutaneous or intranasal apomorphine. Informed consent was obtained before the start of therapy. Existing anti-Parkinson medication was continued unchanged.

The mean age of the patients was 65 years, the mean lapse of time since Parkinson's disease was diagnosed was 7·7 years, and the mean duration of levodopa therapy was 6·9 years. Motor disability was assessed using the Columbia Rating Scale and by quantitative measurement of walking time, hand tapping and foot tapping, and pinboard testing as described by Lees *et al*.[3] Pharmacokinetic data (Cmax and Tmax) were obtained from blood samples taken at short intervals after the enema was administered.

The first patient showed no motor response to 10 mg apomorphine, although substantial plasma levels were shown. (Tmax 9 minutes, Cmax 7 ng/ml). Previously 5 mg apomorphine subcutaneously had been sufficient to relieve "off period" akinesia. Increasing the rectal dose to 25 mg, however, had a beneficial motor effect equivalent to that obtained with a subcutaneous dose of 5 mg. Tmax was 15 minutes and Cmax 19 ng/ml.

The second patient, known to respond positively to 3 mg apomorphine subcutaneously, showed an equivalent motor response to 15 mg rectally. Tmax was 15 minutes and Cmax 14·6 ng/ml. The duration of response was 35 minutes with a latency of onset of 15 minutes.

The third patient, previously experiencing optimal benefit from 4 mg apomorphine intranasally, demonstrated a positive motor response to 10 mg rectally (fig). When 20 mg was given rectally, motor improvement was longer lasting, (60 minutes against 18 minutes), but was accompanied by slight though not disabling dyskinesias.

In our three patients, apomorphine appeared to be as effective in the relief of "off period" signs and symptoms when given rectally, as it was by the subcutaneous or intranasal routes. The effective dose, however, was two and a half to five times greater. Recently Hughes *et al*[5] reported their experience with high dose (200 mg) apomorphine suppositories. In our experience an enema has a much shorter latency of onset than a suppository, which suggests that it would be more useful for patients suffering frequent "off period" symptoms. The clinical effect was, however, also of much shorter duration

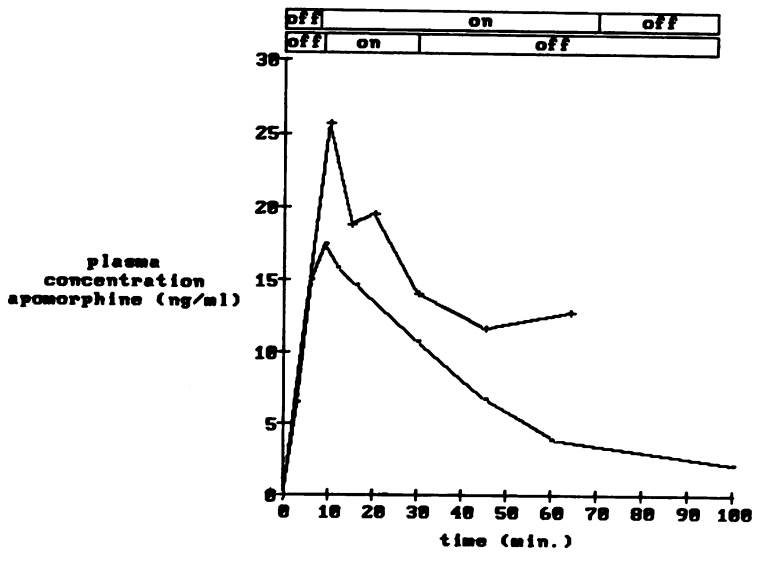

Figure Plasma apomorphine concentrations in the third patient after rectal application of 10 mg (.) and 20 mg (+) apomorphine. The motor response is shown in the upper bar for the 20 mg dose and in the lower bar for the 10 mg dose.

(35 minutes *v* 195 minutes respectively), a finding which might be explained by the differences in absorption of apomorphine from the two preparations.

These preliminary results suggest that apomorphine is effective when administered rectally as an enema and may be the chosen method for rapid relief of "off period" symptoms when other routes may be unavailable due to adverse effects or the patients' difficulty in applying subcutaneous or intranasal apomorphine. We advise regular proctoscopy when treatment is frequent or prolonged.

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1 Stibe CMH, Lees AJ, Stern GM. Subcutaneous infusion of Apomorphine and Lisuride in the treatment of Parkinsonian on-off fluctuations. *Lancet* 1987;**i**:871.
2 Jansen ENH, van Laar T. Oude wijn in nieuwe zakken; therapie met subcutane apomorfine bij de ziekte van Parkinson. *Ned Tijdschrift v Geneeskunde* 1990;**134**:889–91.
3 Frankel JP, Lees AJ, *et al.* Subcutaneous apomorphine in the treatment of Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1990;**53**:96–101.
4 Kapoor R, Turjanski N, *et al.* Intranasal apomorphine: a new treatment in Parkinson's disease. *J Neurol Neurosurg Psychiatry*, 1990;**53**:101–15.
5 Hughes AJ, Bishop S, *et al.* Rectal Apomorphine in Parkinsons disease. *Lancet* 1991;**i**:110.

HAM/TSP attributable to blood transfusion

A multi-centric case-control study was carried out to clarify possible environmental factors related to the onset of HTLV-I-associated myelopathy[1]/tropical spastic paraparesis[2] (HAM/TSP) in northern Kyushu, Japan, which comprises one of the most prevalent areas of HTLV-I in the world.[3]

The frequencies of blood transfusion before the onset of HAM/TSP were 33% (6/18) among male patients and 18% (12/67) among female patients, which were significantly higher than 8% in males and 9% in females in the general population. The age-adjusted summary odds ratios with 95% confidence intervals were 7·0 (2·9–17·0) for males and 2·4 (1·3–4·5) for females. The percentages of population attributable risk[4] of HAM/TSP attributable to transfusion were estimated to be approximately 29% (5–52) for males and 11% (1–21) for females.

The fraction of HAM/TSP attributable to transfusion after the introduction of blood screening for HTLV-I, in effect since 1986 in Japan, was definitely smaller than that before the programme. Our observations seemed compatible with a marked decline in newly diagnosed HAM/TSP patients after its introduction,[5] which may be part of the benefit of blood screening. Neither smoking nor drinking was related to the risk of HAM/TSP.

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1 Osame M, Usuku K, Izumo S, *et al.* HTLV-I associated myelopathy, a new clinical entity. *Lancet* 1986;**i**:1031–2.
2 Gessain A, Barin F, Vernant JC, *et al.* Antibodies to human T-lymphotropic virus type-I in patients with tropical spastic paraparesis. *Lancet* 1985;**ii**:407–10.
3 Shibasaki H, Tokudome S, Kuroda Y, Yanagawa T, Yoshihara M. Prevalence of HTLV-I-associated myelopathy among HTLV-I carriers in Saga, Japan. *Neuroepidemiol* 1989;**8**:124–7.
4 Whittemore AS. Estimating attributable risk from case-control studies. *Am J Epidemiol* 1983;**117**:76–85.
5 Osame M, Izumo S, Igata A, *et al.* Blood transfusion and HTLV-I associated myelopathy. *Lancet* 1986;**ii**:104–5.

Suppression of motor neuron firing by transcranial magnetic stimulation in a patient with multiple sclerosis

Transcranial magnetic stimulation produces a short latency excitatory response in tonically active single motor units in small hand muscles.[1 3] This is shown by a peristimulus time histogram, which cross correlates motor unit firing times with the time of the stimulus. The excitatory response in the first dorsal interosseous muscle (FDI) occurs at 20–30 ms and is termed the primary peak (PP); in some motor units a secondary peak at 50–90 ms is also seen.[3] PP is characteristically followed by a compensatory period of zero firing probability, which reflects the advancement of discharges that would have occurred shortly after PP had no stimulus been applied. Complete suppression of firing in response to transcranial magnetic stimulation in the absence of an excitatory peak has not been reported. The response of 21 single motor units to transcranial magnetic stimulation has been recorded in nine patients with multiple sclerosis (MS), to study the neural mechanism of symptoms caused by upper motor neuron lesions.[3] Findings have suggested that spatial and temporal summation at the spinal motoneuron is impaired by a reduction in the velocity and synchronisation of central transmission. For healthy subjects, however, complete suppression of tonic activity in single motor units by transcranial magnetic stimulation was not observed.

The patient, a 48 year old female, initially presented in 1986 with paraparesis, weakness affecting the left arm, and diplopia. The diagnosis of MS was supported by the finding of oligoclonal bands in the CSF, enhancing white matter lesions on CT and by delayed VERs. She was subsequently free of symptoms for four years. She then re-presented with pyramidal weakness of the right arm [reducing the power of the right FDI to 4/5, MRC scale], increased tone in the left leg, extensor plantar responses and impaired joint position sense in the toes. During this clinical episode the response to transcranial magnetic stimulation of three voluntarily activated low threshold motor units from the weakened right FDI was recorded.

The patient was right handed and gave her informed consent to the experiments which were performed with the approval of the local ethics committee. The inducing current flowed in an anticlockwise direction through a circular (Novametrix 200) positioned tangentially at the vertex. Single MU potentials were recorded using fine concentric needle electrodes (Dantec type 13L58). The patient was asked to maintain repetitive motor unit firing and was aided by auditory and visual feedback of the motor unit discharge. Signals were amplified with a band pass of 32 Hz to 16 KHz (Medelec type MS6) and epochs of −250 to +250 ms relative to the stimulus were digitised at 10 kHz (Cambridge Electronic Design 1401) for subsequent analysis. Up to 120 stimuli were given for each motor unit, delivered at random with respect to the

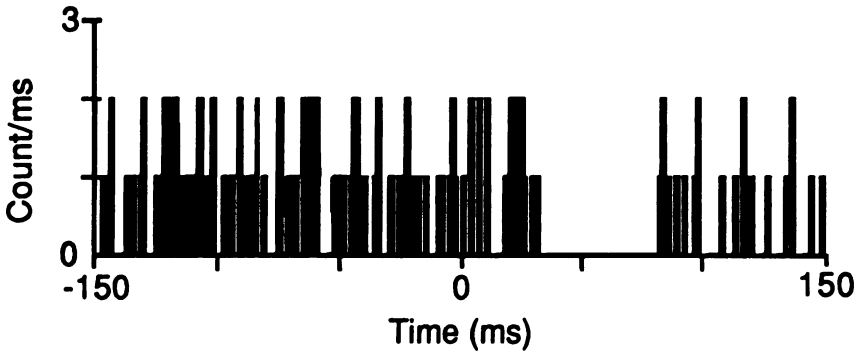

Figure Peristimulus time histogram constructed from the discharge of a single tonically active motor unit from FDI, recorded with a concentric needle electrode. Magnetic stimuli were delivered at time = 0, in 120 trials. There is a distinct period of zero firing probability with an onset latency of 34 ms and a duration of 47 ms. No excitatory peak is seen.