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Efficacy of sublingual apomorphine in Parkinson's disease

Apomorphine administered subcutaneously either by multiple injections or by continuous infusion is used to treat on–off fluctuations in Parkinsonian patients.[1 2] However, the complexity of the techniques of injection, especially with continuous infusion minipumps, and the frequency of local side effects have limited the widespread use of the therapy.[2]

Apomorphine taken orally reduces the on–off effects in Parkinsonian patients but the high doses (400–1600 mg/day), required to obtain a therapeutic response, leads to dose-dependent uraemia.[3]

Sublingual apomorphine is prescribed as an emetic in the treatment of alcoholism. The rich vascularisation of the sublingual area makes absorption very rapid. Also, the catabolism of apomorphine may be slowed down by a diminution of the hepatic first-pass metabolism.

We carried out a study to assess the efficacy of sublingual apomorphine in 8 patients with idiopathic Parkinson's disease. Approval for the trial was granted by the ethical committee of the Faculty of Medicine of Clermont-Ferrand. All patients were given 20mg of domperidone three times daily at least 72 hours before the first administration of apomorphine. Levodopa therapy and dopamine agonists were stopped at least three days before the beginning of the trial. The study was in two steps. On day one, apomorphine was injected subcutaneously in one 3mg dose. On day two, patients were given

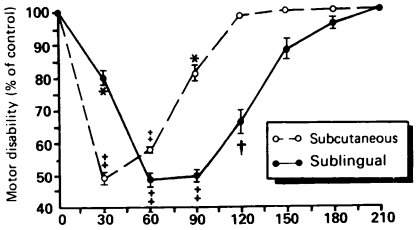

*Figure Time course effect of two different routes of apomorphine in eight Parkinsonian patients. The bars indicate SEM. *P<0·05, ‡P<0·01, †P<0·001 vs corresponding predrug administration. Statistical analysis was performed with the rank sum two sample test.*

18mg (six 3mg tablets) apomorphine sublingually. Assessment of motor function was made by the modified Columbia scale[4] before administration and every 30 minutes thereafter until therapeutic response ceased. The swiftness of the response, determined by the first signs of improvement in the motor score, and its duration were also measured.

Improvement occurred in all patients whether apomorphine was administered subcutaneously or sublingually (fig). Subcutaneous apomorphine had an effect within a mean time of 14 minutes (extreme values: 10–15 minutes) for a mean duration of 77 minutes (extreme values: 50–110 minutes); these results are in line with those of other reports.[1 5] The effect of sublingual apomorphine was slower (mean delay of onset: 30 minutes; extreme values: 20–35 minutes) but more sustained (mean duration: 120 minutes; extreme values: 85–200 minutes). The two routes of administration induced a comparable therapeutic response with a maximum mean improvement of 50% in the score on the Columbia scale.

The only drawback to sublingual tablets is their bitter taste. No clinical or biological side effects were observed. Sublingual apomorphine is of interest in the treatment of idiopathic Parkinson's disease because it is simple to administer, harmless when given once and has long-lasting effect. Other studies are required to evaluate more fully this route of administration in Parkinson's disease.

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CSF somatostatin-like immunoreactivity in dementia of Parkinson's disease

Decreased cortical concentrations of somatostatin-like immunoreactivity (SLI) have been one of the principal biochemical abnormalities found in the brains of patients with Alzheimer's disease.[1] Degeneration of somatostatinergic neurons has also been implicated in the pathophysiology of dementia in Parkinson's disease.[2] Whereas brain somatostatinergic deficiency seems to be reflected in the cerebrospinal fluid (CSF) of patients with Alzheimer-type dementia,[3] studies of somatostatin CSF-concentrations in Parkinson's disease have so far produced conflicting results.[3 4] This study was performed to re-assess whether CSF-SLI levels are altered in Parkinson's disease and whether there is a correlation between CSF-SLI concentrations and cognitive performance in patients with Parkinson's disease.

Twenty two patients with idiopathic Parkinson's disease undergoing routine inpatient treatment gave informed consent to a diagnostic lumbar puncture. Eleven had a history of progressive deterioration of memory and other intellectual functions (Parkinson-Dementia group), while there was no evidence for dementia in the other 11 patients (non-demented group). Further clinical details are summarised in table 1.

Eleven inpatients [four females, seven males; mean (SD) age 48 (12) years] without clinical evidence of CNS disease for whom CSF samples were available, served as controls, after agreeing to have an identical neuropsychological test battery as the patients (see below). Only patients with normal routine CSF findings were included in this control group.

Neuropsychological tests included: verbal IQ (VIQ) and performance IQ (PIQ) both

Table 1 CSF-SLI in Parkinson's disease (PD)

	Patient data (n=22)							
					Levodopa			
Group	*Male: Female*	*Mean age (range) years*	*Mean duration of illness (range) years*	*H + Y stage*	*Mean dose (range) mg*	*Mean duration (range) years*	*Concomitant treatment drug*	*Number of patients*
PD with Dementia (N=11)	6:5	67 (48–76)	9 years (1–21)	3, 4 (2–4)	952 (300–2000)	6, 1 (1–12)	Anticholinergics Bromocriptine Amantadine	4 1 1
PD without Dementia (N=11)	6:5	61 (45–74)	7, 45 yrs (1–16)	2, 7 (1–4)	760 (400–2100)	5, 6 (1–16)	Anticholinergics Bromocriptine Amantadine	4 1 2

H + Y = Hoehn and Yahr.

Table 2 Neuropsychological test results and SLI in three study groups mean (SD) scores

Score		Full Scale IQ	Verbal IQ	Performance IQ	Block Design max=42	Digit Symbol max=6	Logical Memory (MQ)	Associate Learning (MQ)	Wordlist generation	SLI (fmol/ml)
PD without dementia (N=11)	$\bar{x}$	100·9	103·0	97·9	7·8	6·8	91·4	99·1	50·4	49·1
	SD	(9·7)	(9·7)	(10·7)	(2·8)	(3·8)	(10·8)	(11·4)	(14·1)	(14·4)
PD with dementia (N=11)	$\bar{x}$	82·5**	91·6	76·6**	3·0†	2·9†	81·0†	70·6	25·6‡	56·4 ns
	SD	(7·5)	(11·9)	(7·1)	(2·0)	(1·8)	(5·7)	(36·4)	(8·0)	(24·6)
Controls (N=11)	$\bar{x}$	101·4	101·1	99·1	9·8	10·0	90·4	94·8	56·0	50·2
	SD	(15·8)	(11·0)	(16·9)	(2·9)	(4·4)	(4·5)	(18·2)	(17·1)	(17·7)

MQ = Memory Quotient (Wechsler Memory Scale)
** = $p \leq 0{\cdot}001$ One-Way ANOVA
† $p \leq 0{\cdot}05$ Kruskal-Wallis
‡ $p \leq 0{\cdot}001$ Kruskal-Wallis
SLI = somatostatin-like immunoreactivity.

measured using the verbal and performance subtests of the HAWIE, a German equivalent of the Wechsler Adult Intelligence Scale. The sum of all scaled scores yielded the full scale IQ (FSIQ). A German version of the Wechsler Memory Scale (WMS) was used to test verbal learning, while semantic memory and associative verbal production were tested by time limited (60s) word list generation for categories (supermarket, animals) and letters.

Lumbar punctures were performed in the morning after an overnight fast before the patients' arose. Three aliquots of 3ml each were collected into glass tubes and the third was immediately frozen and stored at −70° until assessment of SLI by radioimmunoassay as described previously.[5] The antiserum used equally detected somatostatin-14, somatostatin-28 and prosomatostatin.

No significant differences were found in an analysis of variance between mean SLI-concentrations of the three groups ($F = 0{\cdot}435$, $P = 0{\cdot}65$, $df = 2$).

Table 2 summarises the mean SLI values and the most relevant neuropsychological test results. Highly significant differences existed between the Parkinson-dementia group and the non-demented and control groups in several cognitive measures and the overall test pattern in the demented group was compatible with subcortical dementia. There were no significant differences between the non-demented group of Parkinson's disease patients and controls in any of the cognitive functions tested in this study (table 2).

Spearman rank correlation coefficients for possible relationships between CSF-SLI levels and scores of any of the neuropsychological test parameters did not reach significant values in any of the three groups.

This study thus failed to confirm previous findings of lowered CSF-SLI in Parkinson's disease[4] and agrees with the findings of Beal *et al*[3] in a smaller sample of Parkinsonian patients. Based on the finding of lowered cortical somatostatin concentrations in demented but not in non-demented Parkinsonian patients[2] it has been suggested that discrepant results concerning CSF-SLI in Parkinson's disease might be due to differences in the prevalence of dementia among the patient populations studied. This is not supported by the results of our study where there was no difference in mean CSF-SLI values between patients with and without dementia as assessed by formal neuropsychological testing. This does not exclude a role for cortical somatostatin depletion in the dementia of Parkinson's disease but suggests that it is less significant than in Alzheimer's disease. Cortical somatostatin depletion is apparently not reliably reflected in the CSF and CSF-SLI does not appear a useful biochemical marker for dementia in these patients.

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Multifocal astrocytoma presenting as action myoclonus

Action myoclonus is an uncommon presentation of intracranial tumour.[1] We have recently seen a multifocal astrocytoma present in this way.

A 46 year old right handed housewife presented with a three month history of abnormal movements of her left arm. Soon after waking one morning she was aware of jerking movements of the left arm when she reached out to touch her husband. Jerking persisted for a few seconds only and she thought no more of it. One week later she had several similar attacks, all following voluntary movements of the left arm. Over the next two months similar attacks recurred at least five times daily but often up to 100 times per day. Attacks would last between 10 and 15 s. They usually occurred during and interfered with eating, dressing and bathing. Her attacks consisted of coarse and irregular jerky movements involving the left shoulder and to a lesser degree the elbow and wrist. Attacks were consistently triggered by voluntary movement but not by the intention to move, nor by touch, pressure or startle. She had noticed a refractory period of about five minutes after an attack during which time she could use the left arm quite normally. She did not have attacks during sleep.

Examination revealed spasticity and exaggerated tendon reflexes in the left arm. All routine blood tests, examination of the cerebrospinal fluid (CSF), unenhanced cranial computed tomography (CT), somatosensory evoked potentials, and an electroencephalogram (EEG) performed during jerking were normal. Carbamazepine failed to control her abnormal movements and was stopped after 10 days because of a generalised rash. Subsequently the attacks became less frequent and stopped completely about a month later without any further treatment.

One month after her jerking had stopped, she developed mild pyramidal weakness of the left arm and slight clumsiness of her right hand. Magnetic resonance imaging (MRI) of the brain showed multiple lesions of different sizes involving both grey and white matter (figure). The largest lesions were in the left striatum, left frontal white matter, and right frontal and parietal cortex. None of the lesions produced mass effect. Subsequent cranial CT now showed ill-defined non-enhancing low density changes in the same places. All evoked potentials, EEG, re-examination of CSF, autoantibody screen, echocardiogram and tests for *Borrelia burgdorferi* and human immunodeficiency virus were normal or negative.

It was thought that she was probably suffering from some sort of inflammatory encephalopathy. She returned to her home abroad with arrangements for repeat cerebral MRI. After remaining well and asymptomatic for the following six weeks she developed generalised seizures which were treated with phenytoin. After the occurrence of a number of fits in rapid succession she did not recover consciousness.

She was readmitted to the National Hospital, six months from the onset of her symptoms. She had marked neck stiffness, swelling of both optic discs, her eyes were divergent and her pupils were semi-dilated and unreactive. She could flex her limbs to painful stimuli and her left arm and right leg were spastic. Both plantar responses were extensor and there were Cheyne-Stokes res-