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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

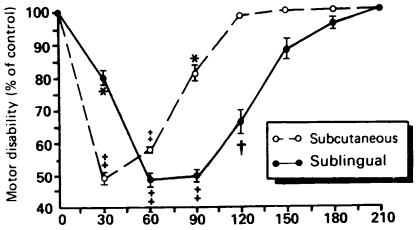

*Figure Time course effect of two different routes of apomorphine in eight Parkinsonian patients. The bars indicate SEM. *P<0·05, ‡P<0·01, †P<0·001 vs corresponding predrug administration. Statistical analysis was performed with the rank sum two sample test.*

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.