hypothalamus, are clearly anterior to the limits of the haematoma (fig). Moreover, the absence of weakness suggests that the internal capsule, located between the haematoma and the hypothalamus, is unaffected. Hypothermia might therefore be ascribed to a damage to the efferent pathways from the hypothalamus to the brainstem.

Bilateral lesions of the hypothalamus seem necessary to produce thermic dysregulation, whereas unilateral damage to the medulla oblongata appears to be sufficient.⁶ In our case, the initial downward gaze palsy indicates a bilateral involvement of the upper mesencephalon.⁷ This can be compared to Wernicke's encephalopathy where hypothermia may occur, and where the mesodiencephalic lesions, although more extended rostro-caudally, are bilateral.⁸

To our knowledge, no previous observation of hypothermia related to a mesodiencephalic lesion has previously been reported. Its rarity might be explained by the need for bilateral lesions, and the fact that hypothermia may go unnoticed.

G GAYMARD
H CAMBON
D DORMONT*
A RICHARD
C DEROUESNE

Clinique des Maladies du Système Nerveux,
Service de Neuroradiologie,*
Hôpital de la Salpêtrière, Paris, France

- 1 Reuler JB. Hypothermia: pathophysiology, clinical settings, and management. *Ann Intern Med* 1978;89:519-27.
- 2 Bauer HG. Endocrine and clinical manifestations of hypothalamic disease: a survey of 60 cases with autopsies. *J Clin Endocrinol* 1954;14:13-31.
- 3 Mann MW, Degos JD. L'hypothermie dans l'encéphalopathie de Wernicke. *Rev Neurol* 1987;143:684-6.
- 4 Boshes B. Syndrome of the diencephalon. In: Vinken PJ, Bruyn GW, eds. *Handbook of Clinical Neurology*. Amsterdam, 1969;16:432-63.
- 5 Hammel HT. Regulation of internal body temperature. *Ann Rev Physiol* 1968;30:641-70.
- 6 Lipton JM, Dwyer PE, Fossler DE. Effects of brainstem lesions on temperature regulation in hot and cold environments. *Am J Physiol* 1974;226:1356-65.
- 7 Pierrot-Deseilligny C, Chain F, Gray F, Serdaru M, Escourolle R, Lhermitte F. Parinaud's syndrome. Electro-oculographic and anatomical analysis of six vascular cases with deductions about vertical gaze organization in the premotor structures. *Brain* 1982;105:667-96.
- 8 Victor M, Adam RD, Collins GH. *The Wernicke's-Korsakoff syndrome*. Philadelphia: FA Davis, 1971.

Intranasal apomorphine: a new treatment in Parkinson's disease

Apomorphine, a directly acting dopamine agonist, has recently been used in the treatment of Parkinson's disease complicated by motor fluctuations. Benefit is seen rapidly and reliably following subcutaneous injection. We have sought alternative, more convenient routes of delivery for the drug. Effective mucosal absorption has been reported¹ and we now describe the use of apomorphine delivered intranasally.

Eight patients with Parkinson's disease were studied. Their mean age was 58.1 years (48-70), duration of disease 12.9 years (5-22) and length of treatment with levodopa 11.8

Figure The motor response and plasma concentration following single 6mg intranasal administration of apomorphine.

years (4-19). All had severe on-off fluctuations which had already been shown to respond to intermittent subcutaneous injections of apomorphine.

Patients were assessed in an off period following withdrawal of usual medication using a modified Webster rating scale,² timed walking over a 12 metre course and unilateral hand-tapping for 30s on digital counters mounted 20 centimetres apart. Apomorphine solution (10mg/ml) was administered, intranasally, using a metered-dose device and the above assessments were repeated at regular intervals until motor performance returned to baseline.

In seven cases there was a prompt response to 6mg (0.6ml) intranasal apomorphine. No adverse effects were noted and the nasal spray caused no local irritation. In these seven patients the mean Webster scores improved

from 21.8 to 10.9; walking times from 17.3 to 9.0 seconds; and tapping counts from 29 to 50. The onset of the motor response occurred after a mean interval of 8.9 minutes (6-15) and persisted for a mean duration of 44 minutes (36-55). Similar times were reported by the patients for subcutaneous use and equivalent improvements in the measures of Parkinsonism were also seen with both methods of administration. The onset and duration of response appeared to correlate with blood levels of apomorphine obtained in one case (fig). The intranasal dose was between 1.5 and twice the subcutaneous one. The patient who did not respond to 6mg did so following a larger dose of 8mg. She had a long history of nasal congestion, perhaps hindering the mucosal absorption of apomorphine.

This preliminary study suggests that intranasal delivery may offer an effective alternative to subcutaneous injection of apomorphine. The benefits of the latter, including the speed and quality of motor response, appear to be retained in most cases with this simpler technique, prompting further evaluation of its long-term use.

R KAPOOR
N TURJANSKI
J FRANKEL
B KLEEDORFER
A LEES
G STERN

Department of Neurology,
University College and Middlesex Hospitals,
School of Medicine, London

M BOVINGDON
R WEBSTER
Department of Pharmacology,
University College, London, United Kingdom

Correspondence to: Dr Lees.

- 1 Walton RP, Lacey C. Absorption of drugs through the oral mucosa. *J Pharmacol Exper Ther* 1935;54:61-76.
- 2 Kempster PA, Frankel JP, Bovingdon M, Webster R, Lees AJ, Stern GM. Levodopa peripheral pharmacokinetics and duration of motor response in Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1989;52:718-23.

Somatostatin in cerebrospinal fluid after generalised convulsions or cerebral infarction in humans

A role for somatostatin in the generation of epileptic seizures is discussed as increased concentrations of this peptide in epileptic foci have been reported.¹ In the cerebrospinal fluid (CSF) of rats increased levels of somatostatin-like immunoreactivity (SIR) were found following ethylenetetrazol-induced convulsions.² The same group of investigators, however, was unable to demonstrate a change of CSF SIR levels in nine epileptic patients presenting with generalised convulsions.³

We have measured SIR by specific radio-immunoassay⁴ in the CSF of 16 patients with epilepsy (table). Of these, eight patients were

treated with phenytoin, two with phenytoin and phenobarbital, two with carbamazepine and primidone, one with bromazepam. Three patients received no anti-convulsant drugs. There were no significant differences or trends in SIR levels apparent when patients were grouped according to their drug treatment.

In six patients generalised convulsions preceded the lumbar puncture by periods of several hours to three days. Their levels were compared with those of epileptic patients without recent seizures, of control patients without proven neurological diseases and of patients with cardiovascular disease (CVD) and cerebral infarction. The mean level of the epileptic patients was not significantly different from that of the control patient group and that of the group of patients with

Table Clinical data and SIR levels

Group	n	Age mean (SD)	Sex (female/male)	SIR mean (SD) pmol/l
Control patients	27	35 (3)	10/17	113.9 (6.3)
Epileptic patients	16	46 (4)	6/10	135.6 (9.4)
Subgroup with recent convulsion	6	52 (8)	2/4	166.6 (12.3*)
Cerebrovascular disease	20	51 (4)	5/15	125.5 (11.3)
Subgroup with recent infarction	5	54 (6)	1/4	184.6 (21.6**)

*significantly different from epileptic patients ($p < 0.01$, Wilcoxon test)

**significantly different from cerebrovascular disease ($p < 0.01$)

Figure Linear regression of CSF SIR levels and intervals between pathological event and collection of CSF in patients with generalised convulsion (●); and in patients with cerebral infarction (○). Correlation coefficients were calculated on a Hewlett-Packard HP 9825A. Pearson's correlation coefficient was 0.985 for patients with generalised convulsion and 0.989 for patients with cerebral infarction. Horizontal line and shaded area represent mean (SD) level in control patients without proven neurological disease.

CVD (table). However, the subgroup of epileptic patients with convulsions, occurring within a period of several hours to three days preceding the lumbar puncture showed a significantly enhanced mean level of SIR. Linear regression of values against time interval between convulsion and lumbar puncture revealed that CSF levels were negatively correlated with intervals (fig). Both, slope of decline and regression coefficient were quite similar in a subgroup of CVD patients, studied within five days of a hemispheric infarction.

Our findings contrast with the negative results of Pitkänen *et al.*,³ who compared "ictal" levels (one to two hours after generalised convulsion) with "interictal" levels (one to four days after convulsion) in the same patients. Interictal levels of SIR in the lower normal range were reported earlier by Kohler *et al.* in lumbar fluid⁴ and by Wolf in ventricular fluid⁵ of epileptic patients. In convulsing rats cisternal CSF levels of SIR increased within five minutes and decreased after 30 minutes,² an increase in lumbar levels in humans can only be expected after the ventriculo-lumbar transport time of about two hours,⁶ and an alteration of release of SIR may show a much more protracted time course due to postictal depression or other metabolic factors. According to our observations, normal lumbar levels of SIR can be expected within three to five days after the acute event.

Although the number of our patients is small, we conclude preliminarily that 1) lumbar levels of SIR increase after generalised convulsions in epileptic patients; 2) this increase is non-specific with respect to seizure generating mechanisms and similar to increases induced by other conditions of acute tissue hypoxia, for example, cerebral infarctions; 3) the negative findings of Pitkänen *et al.*³ could be explained by sampling "ictal" and "interictal" levels at inadequate time intervals.

HINRICH CRAMER
KARL MARTIN HEITZELMANN

Neurologische Universitätsklinik der Universität
Freiburg,
Hansastraße 9, 7800 Freiburg, FRG

- 1 Kato N, Higuchi T, Friesen HG, Wada JA. Changes of immunoreactive somatostatin and beta-endorphin content in rat brain after amygdaloid kindling. *Life Sci* 1983;32: 2415-22.
- 2 Pitkänen A, Jolkkonen J, Honkanen K-L, Riekkinen P. The effect of pentylenetetrazol-induced convulsions on somatostatin-like immunoreactivity in rat cerebrospinal fluid. *Neuropeptides* 1987;9:19-24.
- 3 Pitkänen A, Jolkkonen J, Riekkinen P. Beta-endorphin, somatostatin, and prolactin levels in cerebrospinal fluid of epileptic patients after generalised convulsion. *J Neurol Neurosurg Psychiatry* 1987;50:1294-7.
- 4 Kohler J, Schroeter E, Cramer H. Somatostatin-like immunoreactivity in the cerebrospinal fluid of neurological patients. *Arch Psychiatr Nervenkr* 1982;231:503-8.
- 5 Wolf A. Immunoreaktives Somatostatin im Ventrikel-Liquor neurochirurgischer Patienten. (Thesis). Medical Faculty, University of Freiburg 1985, 106.
- 6 Ebert MH, Kartzinell R, Cowdry RW, Goodwin FK. Cerebrospinal fluid amine metabolites and the probenecid test. In: Wood JH, ed. *Neurobiology of Cerebrospinal Fluid*. Plenum Press, New York, London: Plenum Press, 1980, Vol 1:97-112.

Autoscopy in hemianopic field

Autoscopy is the visual perception of oneself or part of one's body into the external visual space. Although this is the conventional definition, autoscopic phenomena need not always be visual nor need it be perceived in front of the viewer. It has been described in normal subjects, in organic neurological, functional (migraine, epilepsy) and psychiatric disorders. Autoscopy in focal cerebral lesions is a rarity and autoscopic images appearing in the hemianopic field are still rarer. Our case had a right occipital infarct with autoscopy in the left hemianopic field.

A sixty year old male was admitted with a history of bifrontal throbbing headache which started suddenly two weeks before. About one week after the onset, the headache became worse and almost simultaneously the patient started seeing his own image in front of him on the left side. Five days later, at the time of admission he was able to give a detailed account of his experience. The image first appeared at a distance of about 30cm in front of him, more towards the left, and persisted for three to four minutes. He could identify the face and upper part of the body including the colour of the shirt and the expression on the face. Thereafter, it appeared for a few minutes several times a day. There was no warning, and no general pattern to the frequency, time of occurrence, or movements performed by "the double". On one occasion, he saw his "double" while he was brushing his teeth. At another time he was sitting on his bed and saw the image turning around and walking away. The image always appeared to the left side and in front of him, and disappeared when the patient closed his eyes. At first the patient's emotional reaction was one of anxiety and amazement but later he became indifferent to the presence of "his companion". The patient did not drink alcohol nor use psychotropic drugs. There was no history of vascular headache or psychiatric disorder.

The general physical examination was unremarkable. Apart from the autoscopy and irritability, he was functioning normally and

was fully aware of the "unrealistic nature of his companion". There was bilateral papilloedema and left homonymous hemianopia but no other neurological abnormalities.

Routine blood and urine examinations were normal apart from a high blood sugar which was controlled with plain insulin. Blood urea, serum creatinine and serum proteins were normal. VDRL was non-reactive and LE cells were negative. ECG, EEG, VEP (full field stimulation) and radiographs of the head and chest were normal. CT scan showed a mixed density irregular lesion with contrast enhancement and surrounding oedema in the right occipital cortex consistent with an infarct.

Autoscopy has been defined as a "complex psychosensorial hallucinatory perception of one's own body image projected into the external visual space".¹ Autoscopy is seen in organic and psychiatric disorders. The body image is our concept of the shape, size and mass of our body and its parts.^{1,2} The concept of body schema is not an instinct. As a child grows, there is creation of a tridimensional model of the body as distinct from the surrounding space. External and internal stimuli contribute to the creation of this model which is dynamic and fluctuates according to circumstances. In addition to the organic parts, certain other attributes are also represented in this model, such as, ego or possessiveness. The conventional body image is not confined to the physical body alone but includes the clothes and objects of daily use.³

The body schema concept is a global function of the brain and requires processing at different parts for its expression. Various names and classifications have been suggested for body schema disturbances. The following classification is based on the tridimensional model. Changes in body schema are classified into those affecting its shape, size, mass and position in space.¹ According to this classification autoscopy is a disturbance of body schema affecting the shape.¹ Autoscopic experience involving senses other than vision have been described⁴ in which autoscopy may have tactile and auditory characteristics. The image involves the whole body or part of it, but it always includes the face.⁴ It may be transparent, opaque or coloured and may show expressions.^{2,4}

Autoscopy may be associated with infections and intoxications, especially chronic alcoholism and typhoid fever; psychoses; epilepsy; migraine, and diffuse and focal cerebral lesions.

There is no satisfactory explanation for this phenomenon. But it is likely that autoscopy is due to abnormalities in a high level system which is responsible for the representation of the body in its environment.⁵ A disturbance of this system may result in misinterpretation of the body in space. Although autoscopy does not have any localising value, it may be an early manifestation of a focal cerebral disease.

RANI BHASKARAN
ANAND KUMAR
PC KESAVAN KUTTY NAYAR
Department of Neurology,
Medical College Hospital,
Trivandrum 695011, Kerala, India

Correspondence to: Dr Bhaskaran.

- 1 Lukianowicz N. Body Image disturbances in psychiatric disorders. *Br J Psychiatry* 1967; 113:31-47.
- 2 Lukianowicz N. Autoscopic phenomena. *Arch Neurol Psychiatry* 1958;80:199-220.