Letters to the Editor

hypothesis, are clearly anterior to the limits of the haematomata (fig). Moreover, the absence of weakness suggests that the internal capsule, located between the haematomata 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.1 In our case, the initial downward gait palsey indicates a bilateral involvement of the upper mesencephalon.1 This can be compared to Wernicke’s encephalopathy where hypothermia may occur, and where the mesodiencephalic lesions, although more extended rostro-caudally, are bilateral.6

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.

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

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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 (58-70), duration of disease 12-9 years (5-22) and length of treatment with levodopa 11-8 years (4-19). All had severe off-on 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,6 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 5. 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 as observed 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.

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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 focus have been reported.1 In the cerebrospinal fluid (CSF) of rats increased levels of somatostatin-like immunoreactivity (SIR) were found following ethylentetrazol-induced convulsions.4 The same group of investigators, however, was unable to demonstrate a change of CSF SIR levels in nine epileptic patients presenting with generalised convulsions.4

We have measured SIR by specific radio-immunoassay5 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. The 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

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Table  Clinical data and SIR levels

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Age mean (SD)</th>
<th>Sex (female/male)</th>
<th>SIR mean (SD) pmol/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control patients</td>
<td>27</td>
<td>35 (3)</td>
<td>10/17</td>
<td>1139 (63)</td>
</tr>
<tr>
<td>Epileptic patients</td>
<td>16</td>
<td>46 (2)</td>
<td>6/10</td>
<td>1356 (9-4)</td>
</tr>
<tr>
<td>Subgroup with recent convolution 6</td>
<td>2</td>
<td>52 (8)</td>
<td>2/4</td>
<td>1666 (12-3)</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>20</td>
<td>51 (4)</td>
<td>5/15</td>
<td>125 (11-3)</td>
</tr>
<tr>
<td>Subgroup with recent infarction 5</td>
<td></td>
<td>54 (6)</td>
<td>1/4</td>
<td>1946 (21-4)**</td>
</tr>
</tbody>
</table>

*significantly different from epileptic patients (p < 0.01, Wilcoxon test)
**significantly different from cerebrovascular disease (p < 0.01)
CVF (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 convolution and lumbar puncture revealed that CSF levels were negatively correlated with intervals (fig). Both, slope of decline and regression coefficient where quite similar in a subgroup of CVF patients, studied within five days of a hemispheric infarction.

Our findings contrast with the negative results of Lukianowicz et al., who considered “ictal” levels (one to two hours after generalised convolution) with “interictal” levels (one to four days after convolution) in the same patients. Interictal levels of SIR in the lower normal range were reported earlier by Kohler et al in lumbar fluid1 and by Wolf in ventricular fluid2 of epileptic patients. In convulsing rats central CSF levels of SIR increased within five minutes and decreased after 30 minutes,3 an increase in lumbar levels in humans can only be expected after the ventriculo-lumbar transport time of about two hours,4 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 Lukianowicz et al5 could be explained by sampling “ictal” and “interictal” levels at inadequate time intervals.

HINRICH CRAMER
KARL MARTIN HEITZELMANN

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 rare, 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 about 30 cm 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 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 on the left side 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. EEG, ECG, VEP (full field stimulation) and a cranial computer tomograph 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 psycosensorial hallucinatory perception of one’s own body image projected into the external visual space”. Autoscopy is seen in organic and psychiatric conditions.7 The body image is our concept of the shape, size and mass of our body and its parts.8 The concept of body schema is not an instinct. As a child grows, there is creation of a tridimensional model of the body and its 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.9

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 this body schema disturbance. 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.10 According to this classification, autoscopy as a disturbance of body schema affecting the shape, size and mass of the body is seen. The image involves the whole body or part of it, but it always includes the face.10 It may be transparent, opaque or coloured and may show expressions.11

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 and maintenance 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.

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