hypothalamus, 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. In our case, the initial downward gage palsy indicates a bilateral involvement of the upper mesencephalon. This can be compared to Wernicke's encephalopathy where hypothermia may occur, and where the mesodiencephalic lesions, although more extended rostro-caudally, are bilateral.

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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**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) pmo/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control patients</td>
<td>27</td>
<td>35 (3)</td>
<td>10/17</td>
<td>1133-9 (6-3)</td>
</tr>
<tr>
<td>Epileptic patients</td>
<td>16</td>
<td>46 (6)</td>
<td>6/10</td>
<td>135-6 (9-4)</td>
</tr>
<tr>
<td>Subgroup with recent convulsion</td>
<td>7</td>
<td>52 (8)</td>
<td>2/4</td>
<td>116-6 (12-3)*</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>20</td>
<td>51 (4)</td>
<td>5/15</td>
<td>125-5 (11-3)</td>
</tr>
<tr>
<td>Subgroup with recent infarction</td>
<td>5</td>
<td>54 (6)</td>
<td>1/4</td>
<td>194-6 (21-4)**</td>
</tr>
</tbody>
</table>

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

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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 reported 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 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, timed walking over a 12 metre course and unilateral hand-tapping for 30s on digital counters mounted 20 centimetre 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 score improved from 21-8 to 10-9; walking times from 17-3 to 9-0 seconds; and tapping counts from 29 to 58.

We have measured SIR with specific radioimmunoassay in the CSF of 16 patients with epilepsy (table). Of these, eight patients were treated with phenytoin, two with phenytoin and primidone, one with bromazepam, two with benzodiazepines. Five 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 cerebrovascular disease.
for patients with proven neurological infarction (C). Correlation coefficients were calculated on a Hewlett-Packard HP 9825A. Pearson’s correlation coefficient was 0.985 for patients with generalised convolution and 0.909 for patients with cerebral infarction. Horizontal line and shaded area represent mean (SD) level in control patients without proven neurological disease.

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 Atkinson et al., who found no correlation between “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 fluid5 and by Wolf in ventricular fluid of epileptic patients. In convulsing rats cisternal CSF levels of SIR increased within five minutes and decreased after 30 minutes, 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 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 infarction; 3) the negative findings of Pittakén et al. could be explained by sampling “ictal” and “interictal” levels at inadequate time intervals.

HINRICH CRAMER
KARL MARTIN HEITZELMANN

Letters to the Editor

Autoptosis in hemianopic field

Autoptosis is the visual perception of oneself or part of one’s body into the external visual space. Although this is the conventional definition, autoptotic phenomena need not always be visual and can be perceived in front of the viewer. It has been described in normal subjects, in organic neurological, functional (migraine, epilepsy) and psychiatric disorders. Autoptosis in focal cerebral lesions is a “phantom limb” phenomenon, and autoptic images appearing in the hemianopic field are still rarer. Our case had a right occipital infarct with autoptosis 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 20 minutes before it was 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 to his 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 physiological examination was unremarkable. Apart from the autoptosis 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 cardiac examination of the heart 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.

Autoptosis has been defined as a “complex psychosensorial hallucinatory perception of one’s own body image projected into the external visual space”1. Autoptosis is seen in organic and psychiatric autopsies.2 The body image is our concept of the shape, size and mass of our body and its parts.3 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 external space. External and internal stimuli contribute to the creation of this model which is dynamic and fluctuates according to circumstances. In addition to the body 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 disorders. 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.4 According to this classification autoptosis is a disturbance of body schema affecting the shape.5 Autoptotic experience involving senses other than vision have been described6 in which autoptosis is a tridimensional auditory perception. The image involves the whole body or part of it, but it always includes the face.6 It may be transparent, opaque or coloured and may show exigencies.7

Autoptosis 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 autoptosis is due to abnormalities in a high level system which is responsible for the representation of the body in its environmental context. The presence of this system may result in misinterpretation of the body in space. Although autoptosis does not have any localising value, it may be an early manifestation of a focal cerebral lesion.

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