

Hearing loss is not a diagnosis; rather it is a symptom, and patients require careful assessment. The Royal National Institute for the Deaf proposes that hearing aid dispensers should perform this function. Failure to recognise and further investigate asymmetric sensorineural hearing loss or unilateral glue ear may lead to neglect of serious disease such as acoustic neuroma or postnasal carcinoma. Though the incidence of such diseases is low, even the small sample of 100 patients in this study included seven who warranted further investigation to exclude such conditions. Recognising glue ear and other middle ear abnormalities, which may be amenable to surgical correction, requires skill in otoscopy. Our results suggest that otoscopy performed by general practitioners may miss abnormalities in a considerable number of patients. As a result of inadequate assessment some patients may be denied the option of surgical treatment.

Comparison of the group referred specifically for the provision of a hearing aid with the group referred for assessment suggests that general practitioners already perform some screening and in many cases assess their patients' needs correctly. The group referred for a hearing aid contained a higher proportion of patients who required an aid (46/54) compared with those referred for assessment (23/46). Similarly a greater proportion of patients referred for assessment had hearing loss amenable to surgery. The kappa values for interobserver agreement (table II), however, were substantially different for each group, showing much poorer agreement in the group referred for hearing aids. When viewed alongside the age distribution of

patients in each group the findings suggest that any screening which is presently being done is based predominantly on the patient's age rather than clinical assessment.

We do not seek to criticise general practitioners in this regard; many will have had little or no formal training in otoscopy since undergraduate days. A recent survey showed that roughly one sixth of vocational training schemes included ear, nose, and throat attachments, and only a quarter of these included full time six month posts.<sup>6</sup> For the Royal National Institute for the Deaf to suggest that general practitioners wishing to provide hearing aid services based in their own practices might receive "supplementary training which could be organised during a single or two days"<sup>7</sup> is frankly naive. The suggestion that screening should be performed by the dispenser is unacceptable, and were general practitioners to delegate in this manner they would almost certainly carry ultimate legal responsibility.

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## Lesson of the Week

### Multiple forms of epileptic attack secondary to a small chronic subdural haematoma

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Comparatively rare manifestations of epilepsy may be the presenting features of a subdural haematoma

Since the introduction of computed tomography (CT) small or asymptomatic chronic subdural haematomas have been recognised with increasing frequency, though their detection may depend on the use of contrast enhancing agents.<sup>1</sup> Patients with chronic subdural haematoma commonly present with headache, disturbances of higher cerebral function such as personality change and confusion, or features of raised intracranial pressure. Hemiparesis, with dysphasia if the lesion is over the dominant hemisphere, is the most frequent focal neurological deficit. Epilepsy is traditionally thought of as a rare presenting feature.<sup>2</sup> We describe a patient in whom several different forms of epilepsy were the only clinical manifestations of a very small chronic subdural haematoma.

#### Case history

A 75 year old man was admitted after a witnessed tonic-clonic convulsion. Over the previous two weeks he had suffered three episodes of sudden, complete inability to speak, during which he remained fully conscious without any abnormal movements being noted. Each episode lasted about five minutes. He was seen by his general practitioner, who diagnosed transient ischaemic attacks but did not prescribe aspirin. The patient denied headache, and neither he nor his family could recall any recent head injury. He

was taking chlorpropamide for maturity onset diabetes but there was no previous history of epilepsy or cerebrovascular disease. He had never had an excessive alcohol intake. Full neurological examination on admission showed nothing abnormal, and in particular there was no disturbance of higher cerebral function. Blood glucose, electrolyte, and calcium concentrations remained normal throughout.

Over the next 24 hours attacks were witnessed during which the patient would become vacant and his head and eyes would deviate to the right. His right arm would then become elevated with external rotation of the shoulder followed by alternating flexion and extension at the elbow while the wrist remained flexed and the fingers extended and abducted. These attacks would last between five and 60 seconds and were followed by a variable period of confusion. They could occur several times an hour, often without complete recovery of consciousness in between. During periods of full consciousness, however, no neurological deficit could be detected. An attack could be terminated with intravenous diazepam, which would continue to act for about 30 minutes. He was given a loading dose of intravenous phenytoin followed by conventional oral doses.

About 48 hours after admission a further change was noted, in that he was continuously confused with a fluctuating level of consciousness. On occasions he

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would respond to simple commands but otherwise muttered incoherently. Signs of motor automatism such as rubbing his body and attacks of exaggerated blinking were noted. During this period the electroencephalogram contained background rhythms dominated by theta and delta activity with recurring epileptiform discharges in the left hemisphere (fig 1). As before, intravenous diazepam stopped the muttering and signs of automatism temporarily, though adding oral clonazepam did not prolong control.

As the earlier seizures had a focal motor component, routine computed tomography had been requested, but in view of the fluctuating level of consciousness an emergency non-contrast scan was performed. This did not show any obvious localised collection, shift of the midline, or ventricular distortion. At that stage the cerebrospinal fluid was normal. After review by the neuroradiologists, however, a contrast enhanced study disclosed a small subdural collection over the left frontotemporal region (fig 2).

Further attempts were made to control the seizures including adding carbamazepine but the pattern persisted for a further 48 hours. At that point, five days after admission, we thought that neurosurgical intervention was indicated despite the apparently small volume of the haematoma. At operation only a few millilitres of thick, changed blood was found and removed. Nevertheless, the patient did not suffer any

further seizures. He was discharged without any focal neurological deficit and with normal higher cerebral function. A follow up electroencephalogram showed a normal background of alpha and beta rhythms devoid of "epileptic" activity. The patient remained free of seizures while taking phenytoin for over six months.

## Discussion

Though computed tomography has made detecting chronic subdural haematomas much easier, we may have to rely on subtle abnormalities such as the effacement of sulci to detect very small collections. Contrast enhancement may help to delineate some of these smaller collections, particularly when they are isodense with respect to the brain.<sup>1</sup> This should always be performed in patients with refractory partial seizures, as a considerable proportion will have underlying structural disease.<sup>3</sup> Though small subdural collections may be seen as incidental in computed tomograms and cause no ill effects, it seems likely that in our patient the collection was responsible for the epileptic focus in view of the focal nature of the seizures and the lateralised abnormality in the electroencephalogram. Whether the resolution of the epilepsy was directly attributable to the removal of the haematoma is debatable, as possibly the combined anti-convulsant regimen may have suppressed the seizures at about the same time as the surgery. We, however, think that the change from status epilepticus that was responding poorly to multiple drug treatment to complete control of seizures with a single anti-convulsant supports the decision to operate in this case. Though the patient was receiving chlorpropamide, we had no evidence to suggest that hypoglycaemia was contributory.

Seizures are thought to be a rare presenting feature of chronic subdural haematoma,<sup>2</sup> though Luxon and Harrison, who reported epilepsy or loss of consciousness as an initial symptom in 6% of their cases, suggested that this traditional viewpoint should be questioned.<sup>4</sup> It is our impression that when they do occur there is usually a large haematoma and there is interictal evidence of a focal neurological deficit. One previous report described simple partial seizures as the sole manifestation of a chronic subdural haematoma, and the authors made the point that such episodes might easily be mistaken for transient ischaemic attacks.<sup>5</sup> Our case is of interest because there were four distinct manifestations of the seizure disorder, two of which might not be recognised immediately as being due to epilepsy.

The first manifestation of neurological dysfunction was that of loss of speech with preservation of consciousness and no abnormal motor activity. There has been debate about the pathogenetic mechanisms underlying this clinical picture and those of other "transient neurological deficits," a term referred to transient ischaemic attack in this setting. Ross thought that a focal seizure discharge would be a reasonable explanation for such attacks,<sup>6</sup> but Melamed *et al* suggested that other mechanisms were more likely, including intermittent compression of surrounding vessels, spreading cortical depression of the type associated with migraine after mechanical stimulation of the cortex, and even coexistent thromboembolic transient ischaemic attack.<sup>7</sup> Moster *et al* noted that aphasia was the most frequent presenting symptom of these transient neurological deficits, though suggested that repeated small haemorrhages into the haematoma might be responsible.<sup>8</sup>

The subsequent clinical course in our patient suggests that the episodes of loss of speech were the first manifestation of epilepsy. It is possible that the chronic subdural haematoma caused a small area of

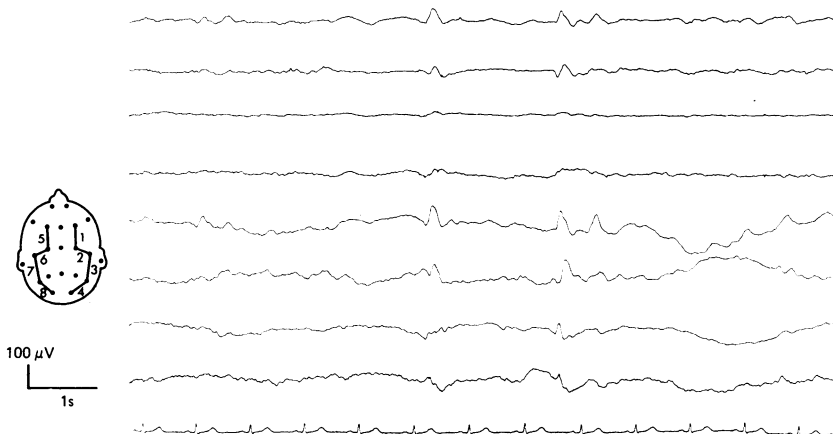


FIG 1—Eight channels of electroencephalogram and one of electrocardiogram recorded during period of complex partial status epilepticus showing epileptiform discharges predominantly in left hemisphere and abnormally slow background rhythms

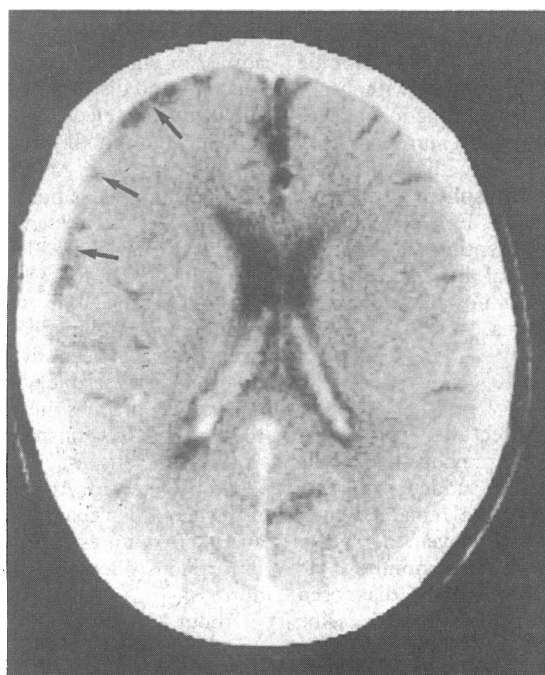


FIG 2—Contrast enhanced computed tomogram showing thin rim of subdural haematoma (arrowed) with effacement of sulci but no compression of ventricular system

cortical thrombophlebitis, but we would not have expected such transient, repetitive, or stereotyped attacks to arise from the purely ischaemic effects of such a lesion. It seems unlikely that there was any appreciable displacement of adjacent vessels because of the very small volume of the haematoma, and the tempo of the attacks would be more rapid than we should expect from spreading cortical depression. We cannot exclude thromboembolic transient ischaemic attacks as a cause but note that the patient did not have any attacks postoperatively. That the general practitioner diagnosed the episodes of loss of speech as transient ischaemic attacks is noteworthy at a time when antihaemostatic drugs are being prescribed routinely for any episode which is thought to be due to transient cerebral ischaemia.<sup>9</sup> This emphasises that the negative symptom of loss of speech might be due to epilepsy and that there may be an underlying lesion such as a chronic subdural haematoma which, at least theoretically, might be made worse by some drugs.

The next two manifestations of epilepsy in our patient were a generalised convulsion and then complex partial seizures with a prominent focal motor component. These second forms of attacks were very similar to those described by Rasmussen, which he correlated with lesions in the medial aspect of the intermediate frontal lobe and supplementary motor strip.<sup>10</sup> The clinical and electroencephalographic findings during the days before surgery would fulfil the criteria for non-convulsive complex partial status epilepticus.

Ballenger *et al*, in a review of published work, noted that two distinct forms of complex partial status epilepticus could be recognised,<sup>11</sup> and both were seen in our patient. In one group the patients have recurrent partial complex seizures. They are usually unresponsive, and adersive or tonic motor phenomena with stereotyped automatism are often noted. In the interictal phase there is improvement in responsiveness but incomplete return to normality. In the other group the patients have continuous complex partial activity in their electroencephalograms. Clinically the patients show quite pronounced fluctuations in response to verbal stimuli and their responsiveness varies from a mild confusional state in which simple commands can be obeyed to complete unresponsiveness. Stereotyped non-purposeful automatism is rare. Though our patient did not have continuous electroencephalographic monitoring, a standard recording

later in his illness showed continuing seizure activity at a time when his clinical state was very similar to that described above. Williamson *et al*, using implanted depth electrodes, reported that complex partial status epilepticus tended to occur with foci outside the temporal lobe and that the frontal lobe was frequently affected.<sup>12</sup> Their patients had symptoms very similar to those in our case.

It is important to recognise that these comparatively rare forms of epilepsy may be the presenting feature of a chronic subdural haematoma. Loss of speech may be treated erroneously as a thromboembolic transient ischaemic attack by using potentially detrimental drugs, and the confusional state caused by the complex partial status epilepticus may be attributed to co-existent but less treatable disorders in elderly patients. In our case the previous clear cut epileptic episodes had led us to suspect that the symptoms were due to epilepsy, but patients may present with such an episode as their first manifestation of epilepsy.<sup>11</sup> Though a fluctuating level of consciousness with a chronic subdural haematoma is most likely to be due to raised intracranial pressure, if the collection is small or the fluctuation persists after drainage of the haematoma the possibility of complex partial status epilepticus should be considered.

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## MATERIA PARAMEDICA

### A ray of sunshine for old ladies: a tip for the geriatricians?

"They die like flies," said the GP. This was in the 1930s, and she was referring to infantile gastroenteritis in general practice. The condition was highly infective and could kill several children in a large family. An outbreak in hospital could be equally frightening, causing deaths in children admitted for other conditions. Intravenous drip infusions and antibacterial drugs had not been invented.

Ten years later I was a "general" physician at St Giles's Hospital in north Camberwell. In that capacity I was deemed to be a specialist in all branches of medicine, which then comprised paediatrics, inter alia, and the giving of anaesthetics. Treatment had scarcely advanced in the meantime, but the importance of isolation was better understood. There were, however, no facilities for isolating these tiny patients. How was I to protect the other children in the ward?

The plan I adopted was to isolate the child with gastroenteritis by removing all the other children. There was room for one cot in each female ward, and a child was moved to the centre aisle between the rows of beds in

the old "Nightingale" wards. I had a hunch that children were less susceptible to adults' germs than to those that more specifically affected children. At all events, no child who was thus transferred picked up an infection.

Most remarkable and unexpected was the effect of a child's presence on the patients. Many of these ladies were elderly, and some were senile, taking no apparent interest in their surroundings. One by one they came to the cot side and took their turn at baby talk. The whole ward became a livelier, happier place, with conversation drifting to and fro. I have no doubt that each child also benefited from the affectionate attention received. Graham Hunter, a former geriatrician, describes the electrifying effect on elderly patients of visits from members of his staff who brought their newborn babies.<sup>1</sup> Perhaps there is a lesson here.

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