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Relapse of benign partial epilepsy of children in adulthood: report of a case

Sir: Benign partial epilepsy of children with rolandic EEG foci, also called rolandic epilepsy, has a good long-term prognosis.¹ Epileptic seizures disappear spontaneously and never occur after 16 years of age.² We report a case of relapse in adulthood.

A thirty-year-old, right handed man has no family history of epilepsy. At the age of 10 yr he had his first seizure, characterised by jerks involving the left arm and the left face. Later fits were rare and mainly occurred during sleep until the age of 13 yr, when secondary generalisation occurred on two occasions. EEG performed when aged 11 yr showed rolandic discharges. Right carotid angiography was normal. He was seizure-free from 13 to 21 yr of age and without anticonvulsant therapy from 15 yr of age. At the age of 21, he began to have seizures with similar characteristics, often followed by secondary generalisation until aged 28 yr; thereafter he had only simple motor seizures affecting the left upper limb with a frequency of one to four per month. Right carotid angiography performed at 21 years of age, and an enhanced CT scan performed at 30 yr of age, were normal as was the neurological examination. An EEG performed at 30 yr of age showed a right rolandic focus (fig). The features of epilepsy of our patient in childhood are those observed in typical rolandic epilepsy: seizures began at school age, were mainly focal motor in type and rare in occurrence, were sleep related and disappeared at puberty. EEG showed rolandic discharges. Neurological and neuroradiological findings were normal. After a seizure-free period of 8 yr, he relapsed, with epilepsy similar to that of his childhood.

Recurrence in adulthood of isolated gen-

Fig. EEG showing right central spike focus (line)

eralised convulsive seizures in patients who have suffered from rolandic epilepsy in childhood has been reported³ as a later manifestation of convulsive predisposition. This does not seem to be the case in our patient who had an electroclinical picture similar to that observed in his childhood when the association of partial motor seizures and rolandic discharges is characteristically found.⁴ Recurrence of epileptic seizures after a long seizure-free period is thus possible in focal "functional" epilepsies. The good prognosis of rolandic epilepsy is not invalidated by our case report. When seizures relapse without apparent aetiological factors in a patient with previous benign partial epilepsy, further neuroradiological examination is not necessary in the absence of neurological deficit and with normal CT scan findings.

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Left temporal lobe abscess presenting with an acute amnesic syndrome 28 years after contralateral temporal lobe abscess

Sir: The amnesic syndrome can result from bilateral damage to the hippocampus and temporal lobes. We describe a syndrome similar to transient global amnesia as the presenting symptoms of a left temporal lobe abscess in a man whose contralateral temporal lobe had been damaged twenty-eight years earlier by an abscess and its surgical removal.

A 33-year-old left-handed man presented to the casualty department with

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left-sided earache and otorrhoea. These symptoms resolved in six days with antibiotic treatment but four days later a memory and speech disturbance developed. This was sudden in onset and lasted approximately two weeks. His wife reported intermittent slurring of his speech and he found he could not remember people's names, including those of his three children and of several other relatives. He also had difficulty remembering place names, for example when buying train tickets. Despite these features suggestive of nominal dysphasia, neither he nor his wife were aware of any hesitancy or other abnormality of spontaneous speech. Furthermore, he found he had forgotten how to climb a previously well-known system of ladders and scaffolding in his work as a brick-layer, although he retained insight into the problem. In addition, when shopping, he could not remember what he had intended to buy. He had no difficulty with fingers or money and there was no left-right disorientation. He was able to recognise the people whose names he had forgotten. His reading and writing were unaffected. Recovery of the abnormality of memory and of speech occurred gradually. He reported these symptoms a week after he had recovered, and at that time examination revealed no neurological abnormality. He was afebrile and otoscopy was normal. Routine biochemical and haematological tests were also normal. Coronal tomography of the skull revealed a deficiency of the tegmen tympani on the left. A CT brain scan showed an intracerebral abscess in the posterior temporal region on the left and evidence of the previous right posterior temporal surgery (fig). An EEG showed marked diffuse right-sided, mainly temporal, slow activity associated with sharp waves; there were no EEG abnormalities on the left side.

Twenty-eight years earlier, when the patient was 5 years old, a right-sided otogenic posterior temporal lobe abscess had been removed at craniotomy, having been aspirated on three occasions through a burr hole. The specimen measured 3.5 cm × 2.5 cm. He made a full recovery with no neurological deficit and had no seizures during the subsequent 14 years. An EEG prior to removal of the right-sided abscess revealed slow high voltage waves throughout the right hemisphere, maximal over the temporal lobe and an EEG twelve years later also showed a severe, but by then more focal, abnormality over the right temporal area.

Because of the bilateral temporal lesions

it was considered that surgical treatment might permanently impair his memory and management was therefore conservative, comprising amoxycillin, flucloxacillin, sodium fusidate and metronidazole in addition to phenytoin. Serial CT brain scans showed complete resolution of the left temporal abscess after 12 weeks' antibiotic therapy. Eight months later he remained well without recurrence of the abscess.

A "severe and generalised defect in memory involving continuous anterograde amnesia for the events of daily life together with some retrograde amnesia for the period preceding the critical brain lesion"¹ may follow bilateral damage to the hippocampus and anterior temporal lobes^{2,3} and similar abnormalities occur in patients with bilateral posterior cerebral artery territory infarction,⁴ in Korsakoff's syndrome⁵ and after head injury.^{6,7} In the syndrome of transient global amnesia associated with ischaemia in the posterior cerebral circulation amnesia occurs suddenly without evident precipitating cause.^{8,9} Retrograde amnesia may extend for hours or even weeks at the onset but this gradually shrinks in a patchy fashion until at the end of the episode, after a few hours or days,

there is a retrograde amnesia only for the duration of the episode itself. Apart from the anterograde amnesia there is no other abnormality during the attack.⁹ Transient amnesia of similar clinical type can occur in migraine and in temporal lobe epilepsy; these must be differentiated from psychogenic amnesia.¹⁰ All these causes could be excluded in our patient.

The amnesia in our patient resembled that found in transient global amnesia except that it was of longer duration and there were features, for example difficulty in remembering proper nouns, suggestive of nominal aphasia. Our patient was left-handed, despite the right-sided brain lesion sustained in infancy, but we have no evidence as to which hemisphere was dominant for speech. We suggest that the development of the left temporal abscess provided the bilateral temporal dysfunction necessary to induce the amnesic syndrome, the right temporal lobe having been damaged by the previous abscess and by the surgical removal of that abscess. The left temporal abscess was successfully treated without surgical excision, but the amnesic syndrome had already resolved before treatment had been started.

Fig. CT scan with contrast showing a left temporal lobe intracerebral abscess and previous surgical intervention in the right temporal lobe.

We are grateful to Professor ES Watkins for allowing us to report this case.

Matters arising

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Exteroceptive reflexes abnormalities in stiff-man syndrome

Sir: We read with interest the electrophysiological studies of a case of stiff-man syndrome by Meinck *et al.*¹ The authors analysed the properties of exteroceptive reflexes circuitry by means of stimulation of a limb mixed-nerve trunk. They showed that exteroceptive reflexes are pathologically enhanced in stiff-man syndrome, in so far as they lack habituation, show lowered threshold and simultaneous co-contraction of antagonistic muscles. In a previously reported case of stiff-man syndrome we observed comparable abnormalities of exteroceptive reflexes.² By means of stimulation of both mixed and purely sensory nerve trunks we demonstrated that exteroceptive reflexes lacked habituation phenomena and that they induced co-contraction of antagonistic muscles. Furthermore, exteroceptive reflexes were elicited only by their specific stimuli and only at the specific site of stimulation, without any enlargement of their receptive field. In contrast, we observed that monosynaptic reflex activity behaved substantially normally. Thus Meinck *et al.*'s data confirm our previous findings in showing that isolated abnormally enhanced exteroceptive reflexes activity underlines the major electrophysiological abnormalities in stiff-man syndrome. As regards the involvement of the central nervous system in stiff-man syndrome, we recall that the prevalence of generalised epilepsy in this syndrome is about 10%, that is, much higher than among the general population.² Furthermore abnormal muscular activity in stiff-man syndrome completely disappears during sleep, a behaviour typical of centrally originating motor disorders.³ These findings lend indirect support to the hypothesis that the loss of inhibitory control of exteroceptive activities reflects a more diffuse central derangement. The absence of any pathological correlates in the few cases necropsied, further shows that stiff-man syndrome is a functional rather than a structural disturbance.^{2 4-6}

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Meinck replies:

The fact that exteroceptive stimuli induce irradiating spasms in the stiff-man syndrome has been described earlier than was suggested by Martinelli and Montagna (our example¹⁻³). The contribution of Martinelli *et al.* to this particular problem consists of three sentences, namely the statement that "polysynaptic flexion and extension reflexes... were normal" whereas "non-nociceptive reflexes... behaved abnormally, lacking habituation phenomena and exhibiting co-contraction..." (p. 459), and the suggestion of "abnormal functions, even if not specific..., of the polysynaptic system" (p. 461).

Besides having difficulties in understanding the exact meaning of their somewhat sibylline statement, I think that we did not confirm Martinelli *et al.*'s (or others') previous results, but substantiated their observations by means of a systematic analysis.

In my opinion, the author's calculation of prevalence rates for generalised epilepsy in the stiff-man syndrome is seriously affected by at least two factors: firstly treatment of the stiff-man syndrome is usually performed with longterm administration of benzodiazepines at high dosages resulting in a high risk for epileptic seizure during withdrawal⁴. Secondly, the term "stiff-man syndrome" most probably comprises a wide variety of disorders which have in common muscular stiffness and spasms, but are poorly understood with regard to their individual aetiology and pathogenesis. The author's hypothesis of