naming, and lecture. Tonal audiometry showed a sensory deafness on the left (60 to 80 dB loss) and a mixed deafness on the right (80 to 120 dB loss) with a prominent sensory component. Brain MRI disclosed a left inferior pontine infarction extending to the territory of the left anterior inferior cerebellar artery (AICA, figure). No lesion was found on the right side. Brainstem auditory evoked potentials (BAEPs) showed poorly synchronised responses with mildly delayed latencies of waves I (right 1.92 ms; left 1.96 ms); waves II to V were not recorded. Middle latency auditory evoked potentials were normal (right Na 19.4 ms, Pa 31 ms; left Na 20.1 ms, Pa 29.8 ms) demonstrating the sparing of higher auditory pathways. This pattern indicated a bilateral retrocochlear impairment. Echocardiography, ECG, and Holter-ECG were normal. Doppler sonography showed a stenosis of the left carotid artery of about 60%. Medical history, clinical picture, and the infarct location suggested that atherosclerosis of the basilar artery was the cause of stroke. The long term course showed a clear improvement and 10 months later, an important right hearing loss (50 to 70 dB) and a mild left hearing loss (10 to 30 dB) still persisted but allowed good intelligibility. Word repetition and environmental noise recognition were quite good. Nevertheless, some difficulties to localise sound persisted with systematic search to the left side.

According to Adams,⁵ deafness characterises the lateral form of inferior pontine infarct and is usually due to an infarction of the cochlear nucleus or auditory nerve. In the present case, the delayed latency of wave I supports the sparing of the distal part of the auditory nerves and the absence of waves II to V shows that deafness resulted from a bilateral cochlear nuclei or proximal auditory nerve impairment.⁶ The finding of a left inferior pontine infarct clearly supports this interpretation for the left deafness. On the right side, no lesion was found but the BAEP pattern strongly suggests a lesion within the same area. The right deafness is likely to be due to a lesion of the radicular portion of the auditory nerve, which is supplied by the auditory artery (branch of the anterior inferior cerebellar artery), or by a small pontine infarct even if it was not visualised on MRI.⁷

Spin echo T2 weighted axial MRI. Left sided infarction in the lateral inferior pons and in the middle cerebellar peduncle.

Sudden deafness due to a brainstem infarct could be underdiagnosed and we should look for this possibility using MRI and BAEP examination, especially in patients with risk factors for stroke and presenting with other neurological signs. This case also suggests that the deafness outcome may be favourable.

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Surgical treatment of quadrigeminal plate lipoma presenting with seizures and behavioural disorders

Intracranial lipomas are uncommon, rarely symptomatic, lesions. Those located in the quadrigeminal plate and ambient cistern account for 13% to 43% of cases and are less symptomatic. Of 37 quadrigeminal plate and ambient cistern lipomas reported in the literature, 15 were diagnosed during life, seven in children.¹ We had the opportunity to study an additional case of quadrigeminal plate lipoma in a four year old child, who presented with epilepsy and developed behavioural changes tending towards aggressiveness, which both subsided after surgery.

In 1986 the patient presented two episodes of tonic-clonic generalised epileptic seizures associated with fever; the EEG was normal. One year later, although he had benefited from therapy with phenobarbitone, he presented episodes of hypotonia, ataxia, motor incoordination, and confusion. A new EEG study disclosed bilateral paroxysmic abnormalities, such as sharp slow waves (figure A). The MRI disclosed a well defined lesion at the level of the left inferior colliculus, which showed a high signal in T1 and low in T2 sequence (figure B). Three months before admission, after the reduction of phenobarbitone dose, the child presented a new tonic-clonic generalised seizure. During the previous two years, his parents had noted

behavioural changes tending towards aggressiveness and hyperactivity. In 1988, the persistence of epileptic seizures, more than behavioural changes, led to the decision to remove the lesion surgically. In a semisitting position, a median suboccipital craniotomy was performed and a tumour, which extended from the left inferior colliculus to the contralateral brachium conjunctivum, was removed through a supracerebellar subtentorial approach. Histological examination confirmed the diagnosis of lipoma. The postoperative course was uneventful. The EEG disclosed only mild abnormalities. The child presented a few seizures after surgery, which were probably secondary to hypodose of the phenobarbitone. Since 1991 he has had no more seizures and since 1992 the EEG has been normal. The antiepileptic treatment was discontinued in 1994 and he remained seizure free without treatment. Concurrently, he became less aggressive and his social behaviour dramatically improved after surgery. At present he is attending school and performing well. Sequential CT and MR studies confirmed the total removal of the tumour.

We report a case of quadrigeminal plate lipoma in a child who presented initially with seizure disturbances. EEG disclosed diffuse symmetric abnormalities which were probably the expression of a centro-encephalic epilepsy induced by the mesencephalic lesion. Maiuri *et al*, in a review of over 200 cases, found that epileptic seizures were the most common feature in symptomatic cases.¹ These are usually ascribed to interhemispheric disconnections in cases of callosal lipomas, or to the tenacious adhesions of the tumour to the brain cortex. Lipomas, although they do not compress or displace the adjacent neural tissue, very often encase nerves and vessels involving the surrounding structures in regressive change within the tumour tissue. Alternatively, epileptic seizures are regarded as epiphenomena of coexisting congenital malformations such as callosal agenesis, microgyria, or hemispheric atrophy. These explanations, however, are unsatisfactory for small lesions located in the quadrigeminal plate, especially when neither hydrocephalus nor congenital abnormalities are associated.

Furthermore, a subcortical or brainstem induced epilepsy has been shown in animal models and found in patients with lesions in those areas.^{2,3} Browning *et al* found that brainstem epilepsy is independent of forebrain seizures and can be elicited even in the complete absence of connections between these two areas.⁴ Other authors have described convulsive fits elicited in cats and rats after stimulation of special points in the brainstem. EEG patterns were specific and significantly different from those of cortical epilepsy. So called tonic cerebellar fits have also been described as resulting from brainstem stimulation, and in laboratory studies controlled brainstem lesions facilitated or inhibited seizure activity. Other studies confirmed the possibility of inducing convulsive seizures by the stimulation of the brainstem, localising a low threshold convulsive area in the mesencephalic reticular formation. EEGs with generalised changes, predominantly synchronous theta waves, have been described in patients with tumours in the region of the aqueduct and with pineal region tumours, and pronounced EEG changes in more than 50% of patients

(A) High voltage spike and waves; bilateral generalised discharges. (B) The lesion appears homogeneously hyperintense on sagittal MR T1 weighted image (TR 350, TE 50).

with midbrain tumours have also been documented. The role of the pineal gland in modulating the EEG is also well documented in both experimental and clinical studies. In our patient, the complete restoration of normal EEG patterns and the subsidence of seizures after surgery suggest a close relation between symptoms and tumour.

Behavioural disturbances tending towards aggressiveness, similar to those we describe, have also been described in patients with lesions in the midbrain-forebrain area. The direct invasion of the upper midbrain may lead to severe behavioural disturbances through involvement of the midbrain-limbic system of Nauta.⁵ This system includes several structures located at the upper dorsal midbrain which, we suggest, may be encased by tumours of the pineal region. Weber reviewed clinical studies which disclosed changes in mental outlook and behavioural disorders in patients with tumours in the midbrain and pineal region; to explain these disturbances he suggested the direct involvement of the mesencephalic reticular system, the periventricular grey matter, and the midbrain-limbic system.³ Piatt and Campbell, in a more recent report, reviewing 37 pineal region meningiomas, identified 13 patients (37%) with dementia, emotional disturbances, or personality changes.⁶ Sano *et al* described 51 patients with violent, aggressive behaviour who significantly benefited from a selective posteromedial hypothalamotomy, with a pronounced calming effect in 95% of the cases.⁷

In conclusion, epilepsy and behavioural abnormalities in patients with tumours involving the quadrigeminal plate area, although rarely reported, are probably more specific than was previously thought. We suggest that surgery may be curative when

limited to symptomatic lipomas in which a relation between tumour location and symptoms has been recognised.

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Lambert-Eaton myasthenic syndrome and non-pulmonary small cell carcinoma

It is generally accepted that Lambert-Eaton myasthenic syndrome (LEMS) is associated with cancer in 50% to 60% of cases, the overwhelming majority of these cancers being small cell carcinomas of the lung. O'Neill *et al*¹ stated that in cases in which the cancer is not small cell carcinoma of the lung the association may be fortuitous. However, small cell carcinomas can sometimes arise in other organ systems. They are considered, like small cell carcinomas of the lung, to be of neuroendocrine origin and presumably have the same antigenic potential. Posner² states that occasional cases of LEMS can be seen in association with non-pulmonary small cell carcinoma, particularly of the prostate and cervix, although reference to these occurrences in the literature is scant. We present a case of LEMS clearly causally related to extrapulmonary small cell carcinoma.

A 70 year old housewife presented in April 1996 with a three month history of leg weakness and dryness of the mouth. She was otherwise well. Examination disclosed quite pronounced weakness of hip flexion and mild weakness of knee extension and arm abduction. Deep tendon reflexes were all intact and did not become brisker after exercise. Her creatine phosphokinase concentration was normal. Electrical studies disclosed a compound muscle action potential of 3.5 mV in abductor digiti minimi on supramaximal stimulation of the right ulnar nerve at the wrist, increasing in amplitude by 40% after 20 seconds of isometric exercise. A voltage gated calcium channel antibody