sarcoid meningitis with cranial nerve involvement.[6] Transient benefit from chlorambucil in a case of sarcoid meningoencephalitis has been reported.[7] In our case, high dose intravenous cyclophosphamide, at doses recently used in multiple sclerosis,[8] quickly and dramatically improved the clinical picture. Although spontaneous remission cannot be excluded, it seems reasonable to assume a causal relation between introduction of treatment and the clinical improvement. We suggest that cyclophosphamide should be considered in cases of severe neurosarcoidosis when steroids are unsuitable or ineffective.

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In vivo distribution of catecholamine reuptake sites in human brain gives clues to the physiopathology of MPTP-induced Parkinsonism

Exposure to N-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) induces Parkinsonian clinical symptoms in humans,[1] due to prominent lesions of the nigro-striatal dopaminergic pathway.[2] Javitch *et al*[3] demonstrated that the catecholaminergic reuptake systems are involved in the pathogenesis of the disorder by transporting a metabolite, N-methyl-4-phenylpyridine (MPP^+), into the axonal terminals. Even though noradrenergic and mesolimbic dopaminergic neurons concentrate MPP^+ as well as nigrostriatal neurons, cell degeneration in primates is most prominent in the nigrostriatal dopaminergic system, and the locus coeruleus is relatively spared.[2,4] Javitch *et al*[3] suggested that this specific pattern of neuronal degeneration may come from species differences in the regional density of catecholamine uptake or from differential sensitivity of the catecholaminergic systems to the action of MPP^+. D'Amato *et al*[5] proposed that the uptake of MPP^+ into substantia nigra dopaminergic cell bodies and its binding to neuromelanin play an important role in the drug toxicity.

Using [^{11}C]nomifensine (NMF) and positron emission tomography (PET) to visualise and quantify dopaminergic and noradrenergic reuptake complexes, we observed in six volunteers a striking contrast between the high concentration of NMF in the striatum and the lack of specific uptake in the frontal cortex.[6] The mean partition coefficient of NMF between the specific and non-specific compartments was 0·95 in the putamen and 0·87 in the caudate nucleus but only 0·28 in the thalamus and nil in the frontal cortex. We suggest that the in vivo differences of reuptake site density between human striatal, thalamic, and frontal areas may be of functional importance for explaining the preferential susceptibility of the dopaminergic neurons in the substantia nigra pars compacta to MPTP compared to the reduced vulnerability of those in the ventral tegmental area and the noradrenergic neurons in the locus coeruleus.

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Unsuspected meningioma presenting as a subdural haematoma

Symptomatic haemorrhages associated with meningiomas are rare and most are subarachnoid in locaton.[1] Subdural haemorrhages are seldom caused by a meningioma.[2]

We report a case of a subdural haematoma in a patient on long-term anticoagulant treatment for chronic atrial fibrillation. A meningioma was unsuspected preoperatively and intraoperatively. Only after pathological examination of the blood clot was the tumour discovered.

A 79 year old, right handed white man who had been receiving Coumadin anticoagulation therapy for chronic atrial fibrillation for two years was admitted to this medical centre. He complained of headaches over the left side of his head for the previous three days. He had difficulty with word finding but no focal weakness or blurring of vision. There was no history of a recent injury. The patient had suffered a brain stem cerebrovascular accident 20 years previously and had residual mild numbness of his right side and weakness in his left leg. Examination showed the patient to be awake, alert, and fully oriented. His pupils were equal, round, and reactive to light and there was no papilloedema. His cranial nerves were intact apart from a pre-existing decrease in visual acuity in the right eye. Muscle power was normal except for a mild weakness in the proximal part of his left leg. Sensory examination showed a slight decrease to pinprick sensation over the right side of his body.

Computed tomography (CT) showed a large left sided subdural haematoma with isodense and hyperdense components. Coagulation studies showed a pro-time of 13·8 seconds (control 11·5 s) and a prothrombin time of 42·8 seconds (control 26·4 s). He was given fresh frozen plasma to correct these coagulation abnormalities. He then underwent a left frontal temporal parietal craniotomy with evacuation of the subdural haematoma. The haematoma was large, extending from the temporal floor to the falx cerebri and from the parietal boss to the frontal pole. It appeared to be a typical subdural haematoma. There were no apparent soft tissue components, nor was the haematoma attached to the leptomeninges. After complete removal of the clot, the entire exposed subdural space was examined. There was no mass on the undersurface of the dura nor on the cortical surface of the brain, and a source of the haemorrhage could not be identified. Postoperatively, the patient was lethargic and had severe expressive dysphasia. He obeyed commands well with equal strength in all four limbs. Further CT did not show reaccumulation of the subdural haematoma or evidence of a cerebrovascular accident. The patient's neurological condition gradually improved, and two weeks after operation he was discharged home. MRI scans, with and without gadolinium, were performed at six months and one year later but did not indicate any residual mass lesion.

All surgical specimens are routinely submitted for pathological analysis at this centre. Gross pathological examination of formalin-fixed clot specimens showed clotted blood with several pale foci but no distinct tumour nodules. Microscopic examination showed relatively fresh clotted blood interspersed with fragments of tumour. Some areas of the tissue clearly showed the pattern of a meningioma, with whorls of meningothelial cells and psammoma bodies. Occasional mitotic figures were identified, and there was extensive necrosis. Some of the necrotic tissue was basophilic and contained psammoma bodies; this was likely to be necrotic tumour. In other areas, the cells were spindle shaped, and the tissue contained numerous small, vascular channels. Some of the vascular areas were clearly in the tumour while others were considered to represent granulation tissue.

Benign tumours such as meningiomas are rarely associated with massive intracranial bleeds. Helle and Conley found only 43 cases of meningioma associated with haemorhage. Of these, only four haemorrhages were strictly in the subdural space, while five were

both intracerebral and subdural in location.[1] The apoplectiform presentation of meningiomas has been noted in cases with and without haemorrhage.[3] Ischaemia, haemorrhage, and oedema have been some of the immediate underlying causes. In this case, the history of headache and difficulty with word finding was consistent with the presence of a meningioma. The rapid clinical course, however, suggests that the intracranial haemorrhage was mainly responsible for the presenting symptoms.

The mechanisms responsible for bleeding into a benign tumour are unknown. Highly vascular meningiomas may possess abnormal tangles of vessels; as the tumour grows, stretching of the vessels leads to weakening of the vascular walls.[1] Alternatively, the cerebral oedema and venous obstruction commonly found with meningiomas may cause tumour infarction followed by haemorrhage.[1 3] The anticoagulation of our patient would have increased the chance of bleeding into a tumour. It is notable, however, that there is only one other reported case of a subdural haematoma with a meningioma in the presence of anticoagulation therapy.[4] It is a routine policy of the neurological surgery service at this university to submit representative tissue from all evacuated haematomas for pathological analysis. Although the likelihood of finding anything other than blood clot in such a specimen is low, cases such as the subject of this report justify the routine because the results can affect the patient's follow up and management.

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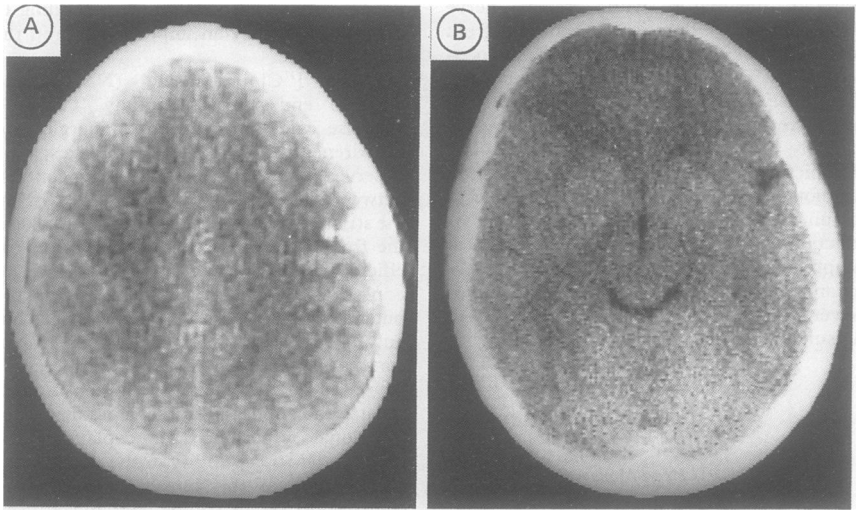

Figure a) Initial CT scan demonstrating small haemorrhage in the left fronto-parietal region; b) two years later, an area of focal cortical atrophy is seen at the site of the previous haematoma.

Cerebral localisation in articulatory dyspraxia

In articulatory dyspraxia, multiple errors in articulation are produced in the absence of damage to the motor or sensory pathways directly controlling the articulatory musculature. It is distinct from, but frequently found in association with, motor dysphasia and oro-facial dyspraxia. This circumstantial evidence, together with information from imaging and necropsy studies, suggests that the cerebral substrate for the condition is damage to the inferior part of the dominant precentral gyrus. We describe a patient with relatively "pure" articulatory dyspraxia caused by focal cerebral trauma and subsequent intracerebral haemorrhage in a small area of the left precentral cortex.

An 18 year old right handed male presented the day after being hit on the left temple by a golf ball. Immediately after the injury he suffered difficulty with speech, in that he was able to think of words but experienced difficulty in pronouncing them. He also noted some brief paraesthesiae in the right thumb. There was no complaint of limb or facial weakness. He was previously well and did not smoke. There was no family history of premature vascular disease.

General examination was normal apart from bruising and some soft tissue swelling in the left parietal region. He was fully conscious and alert with normal higher intellectual function other than the abnormality of oral communication. There was a mild right upper motor neuron facial weakness but no other cranial nerve deficit. In particular, bulbar function was preserved with normal swallowing, cough, palatal, and tongue movements. No focal signs were apparent in the limbs and reflexes were normal and symmetrical with flexor plantar responses.

Detailed assessment of language function revealed normal auditory and written comprehension and no semantic or syntactic errors in his speech. There was no evidence of damage to descending pathways controlling articulation and thus no dysarthria. However, he displayed considerable difficulties with the control of articulation. His speech was laboured and syllabic with disturbed intonation. Multi-syllabic words were particularly difficult for him to say and the pronunciation of some vowels was inconsistent, with a tendency for both front and back vowels to centralise. He claimed that he could hear the correct sounds of words in his head but could not produce them. (Copies of sound recordings of the patient are available from JS on receipt of a blank cassette.) Reading and writing were unaffected and there was no evidence of oro-facial dyspraxia. It was concluded that he was suffering from articulatory dyspraxia without dysphasia. This was confirmed using the Boston Diagnostic Aphasia Examination.

A skull radiograph was normal but a CT brain scan two days after the injury revealed soft tissue swelling over the left parietal bone and a small focus of superficial haemorrhagic contusion low in the left fronto-parietal region (figure a). A repeat scan 21 days after injury was completely normal. A further scan was performed two years later. This demonstrated a small area of focal cortical atrophy in the left fronto-parietal region at the site of the previous haematoma (figure b). An electroencephalogram at this time was normal.

The patient received regular speech therapy over the following three months at the end of which his speech had improved considerably so that his friends and relatives considered it normal. However, he was still aware that he had to exercise more conscious control over the production of speech. When seen two years after the insult, his speech seemed normal but he reported that he still made several errors in articulation each day. He continued to play golf at the same club with a handicap of five!

Articulatory dyspraxia is a distinctive disturbance of articulation in the absence of direct damage to motor or sensory pathways relevant to articulation and is therefore a true dyspraxic syndrome. It is probably underdiagnosed in patients with dominant hemisphere strokes, being confused with the associated dysphasia. The term articulatory dyspraxia is generally attributed to Liepmann[1] and was popularised by Critchley.[2] However, numerous other terms have been used to describe the disorder including aphemia, pure anarthria, pure word dumbness, and pure motor aphasia.[2 3]

The often close association of articulatory dyspraxia with orofacial dyspraxia and expressive dysphasia suggests that the areas of brain responsible for the three conditions lie close together in the inferior aspect of the dominant precentral gyrus. Post-mortem studies in two right handed patients with comparatively "pure" articulatory dyspraxia demonstrated lesions in the inferior motor strip of the left hemisphere.[4 5] These lesions included damage to both cortical and subcortical tissue. CT and MRI studies in a further patient showed a similar though more extensive lesion affecting large areas of precentral and postcentral white matter.[6] The latter authors also reported a left handed patient with the disorder caused by a corticosubcortical haemorrhage in the lower part of the right precentral gyrus. Angiography demonstrated an underlying arteriovenous malformation.

In the present right handed case, also with a