saecoid meningitis with cranial nerve involvement. Transient benefit from chlorambucil in a case of sarcoid meningoencephalitis has been reported. In our case, high dose intravenous cyclophosphamide, at doses neurogenic necrotic in multiple sclerosis, 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 pathophysiology of MPTP-induced Parkinsonism

Exposure to N-methyl-4-phenyl-1,2,3,6-tetrahydro-pyridine (MPTP) induces Parkinsonian clinical symptoms in humans. Two prominent lesions of the nigro-striatal dopaminergic pathway.1 Javitch et al 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 loss in caudate dopamine content is associated with the loss of dopamine reuptake sites.2 Javitch et al suggested that this specific pattern of neuronal degeneration may come from species differences in the regional density of catecholaminergic uptake or from differential sensitivity of the dopaminergic systems to the action of MPP+.3 D’Amato and al demonstrated that the uptake of MPP+ into substantia nigra dopaminergic cell bodies and its binding to neuropeptides play an important role in the drug toxicity. Using [14C]nomifensine (NMFP) and positron emission tomography (PET) to visualize and quantify nigrostriatal and noradrenergic reuptake complexes, we observed in six volunteers a striking contrast between the high concentration of NMFP in the striatum and the lack of specific uptake in the frontal cortex. The mean partition coefficient of MPP+ between the specific and non-specific compartments was 0.95 in the putamen and 0.87 in the caudate nucleus but only 0.26 in the frontal and 0.12 in the striatal 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 haematoa

Symptomatic haemorrhages associated with meningiomas are rare and most are subarachnoid in locantion.1 Subdural haemorrhages are seldom caused by a meningioma.2 We report a case of a subdural haemorrhage in a patient receiving a long-term anticonvulsant treatment for chronic atrial fibrillation. The 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 anticoagula-

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both intracerebral and subdural in location. The apoplectic presentation of meningiomas has been noted in cases with and without haemorrhage. Ischaemia, haemorrhage, and angiomata 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. Alternatively, the cerebral oedema and venous obstruction commonly found with meningiomas may cause tumour infarction followed by haemorrhage. 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.

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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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 paraesthesia 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 deficits. 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 frontal-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 under-diagnosed in patients with dominant hemisphere strokes, being confused with the associated dysphasia. The term articulatory dyspraxia is generally attributed to Liepmann and was popularised by Critchley. However, numerous other terms have been used to describe the disorder including aphasia, pure anarthria, pure word dumbness, and pure motor aphasia.

The often close association of articulatory dyspraxia with oro-facial 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. 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. 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
Unsuspected meningioma presenting as a subdural haematoma.

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