

both intracerebral and subdural in location.¹ The apoplectic 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.

JEFF W CHEN
HOI SANG U
Departments of Surgery and Neurosurgery
MARJORIE R GRAFE
Department of Pathology,
University of California,
San Diego Medical Center,
225 Dickinson Street,
San Diego,
California 92103, United States

Correspondence to: Dr Chen

- 1 Helle TL, Conley FK. Haemorrhage associated with meningioma: a case report and review of the literature. *J Neurol Neurosurg Psychiatry* 1980;43:725-9.
- 2 Walsh JW, Winston KR, Smith T. Meningioma with subdural hematoma. *Surg Neurol* 1977;8:293-5.
- 3 Lazaro RP, Messer HD, Brinker RA. Intracranial hemorrhage associated with meningioma. *Neurosurgery* 1981;8:96-101.
- 4 Everett BA, Kusske JA, Priban HV. Anticoagulants and intracerebral hemorrhage from an unsuspected meningioma. *Surg Neurol* 1979;11:233-5.

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.

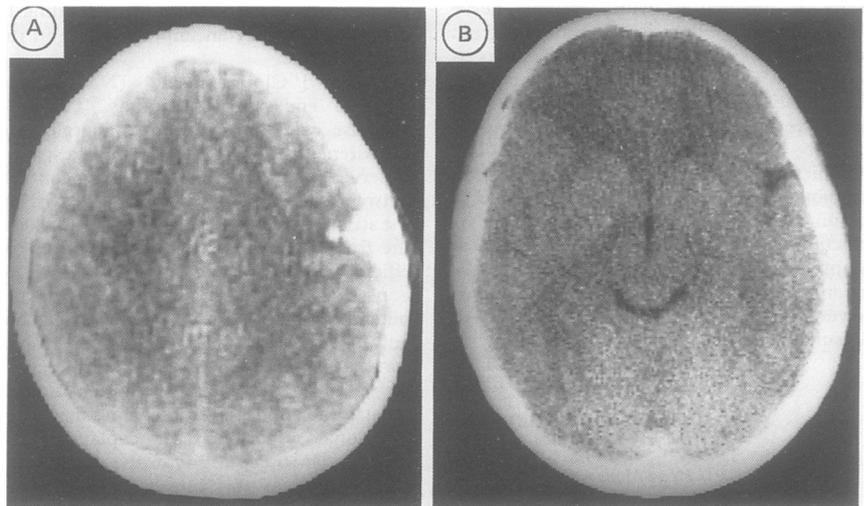


Figure a) Initial CT scan demonstrating small haemorrhage in the left fronto-parietal region; b) two years later, an area of focal cortical atrophy is seen at the site of the previous haematoma.

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 Liepmann¹ 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

relatively "pure" articulatory dyspraxia, the responsible lesion was smaller than in these other case reports. The traumatic haemorrhage destroyed a small area of the inferior aspect of the left precentral gyrus leading to scarring and shrinkage of the Rolandic operculum by the time the second scan was performed two years later. It is impossible to conclude whether damage to the cortex alone was responsible for the disorder or whether subcortical trauma led to additional cortical disconnection, particularly in view of the inner "dumb-bell" area of haemorrhage seen on the initial scan. Presumably, there is a relatively small collection of neurons responsible for the organisation of articulation in the dominant precentral gyrus close to, but distinct from, Broca's area which when damaged produces the curious syndrome of articulatory dyspraxia.

C E CLARKE
D S HOLDER
S CURRIE

Department of Neurology, St James's University
Hospital, Leeds LS9 7TF

J STONE
Department of Speech Therapy, Harrogate District
Hospital, Harrogate, UK.

Correspondence to: Dr Clarke, Department of
Neurology, St James's University Hospital, Beckett
Street, Leeds LS9 7TF, UK.

- Liepmann H. Das krankheitsbild der apraxie ("motorischen asymbolie") auf grund eines falles von einseitiger apraxie. *Monatschrift für Psychiatrie und Neurologie* 1900;8:15-40.
- Critchley M. Articulatory defects in aphasia. *J Laryngol Otol* 1952;66:1-17.
- Wertz RT, LaPointe LL, Rosenbek JC. *Apraxia of speech in adults. The disorder and its management*. London: Grune and Stratton, 1984.
- Lecours AR, Lhermitte F. The "pure form" of the phonetic disintegration syndrome (pure anarthria): anatomo-clinical report of a historical case. *Brain and Language* 1976;3: 88-113.
- Tonkonogy J, Goodglass H. Language function, foot of the third frontal gyrus, and Rolandic operculum. *Arch Neurol* 1981;38:486-90.
- Sugishita M, Konno K, Kabe S, Yunoki K, Togashi O, Kawamura M. Electropalatographic analysis of apraxia of speech in a left hander and in a right hander. *Brain* 1987; 110:1393-417.

BOOK REVIEWS

All titles reviewed here are available from the BMJ Bookshop, PO Box 295, London WC1H 9TE. Prices include postage in the United Kingdom and for members of the British Forces Overseas, but overseas customers should add £2 per item for postage and packing. Payment can be made by cheque in sterling drawn on a United Kingdom bank, or by credit card (Mastercard, Visa or American Express) stating card number, expiry date, and your full name.

The New Genetics of Mental Illness. Edited by P MCGUFFIN AND R MURRAY. (Pp 304; Price £35.00.) 1991. Oxford, Butterworth-Heinemann. ISBN 0-7506-0029-2.

This is an important and timely book. The contributors are distinguished in their fields and it ranges over a wide spectrum of

disorders. The early chapters attempt to explain the complexities of modern genetics but without some background knowledge they would be difficult to digest. Once over this initial barrier, the enthusiasm begins to break through and large amounts of data are presented very clearly. Chapter four by Professor J H Edwards encapsulates the underlying message of the book. Modern genetic methods are extremely powerful when dealing with Mendelian inheritance. They rapidly lose focus and power when this is not the case. Unfortunately, this is not so with the majority of psychiatric disorders so that vast and complex studies are needed to make even small advances. Uncritical hopes of single point mutations cannot be justified.

Psychiatry needs some proven aetiological substrate. Every new advance is pursued with vigour and hope. Every new hormone assay, every new immunological test, every new imaging technique is applied to cohorts of psychiatric patients. Now we have the new genetics and it would be wonderful if it provided us with some markers to underpin our diagnoses. This book brings us down to earth and explains how unlikely that is. Abnormalities of mind remain tough nuts to crack.

This is a recommended text, especially for dewy eyed trainees hoping to net a Nobel prize with a bit of genetic research.

CM TONKS

Migraine and Other Headaches: The Vascular Mechanisms. Vol 1. *Frontiers in Headache Research Series*. Edited by JES OLESEN. (Pp 358; Price \$112.50.) New York, Raven Press. 1991. ISBN 0 88167 795 7.

Research in migraine and new drugs to combat it, progress apace. Olesen has gathered a large number of authorities to produce a highly technical book devoted mainly to cerebral blood flow studies. It is clear there remain serious difficulties in the methods applied; different workers appear to obtain divergent results. Transcranial Doppler which shows velocity and by inference flow in the basal arteries now complements both ¹³³Xenon tests of rCBF, and Tc-HMPAO tracer applied to SPECT studies of static tissue flow. The results are confusing. The editor's early work is confirmed: regional oligoemia in the occipital lobe(s), its failure to conform to arterial territories, its slow spread which usually outlasts the aura, and its confinement to classic migraine with normal results in common migraine. Cluster headache shows normal cerebral flow but dilatation of basal arteries. There is much more of interest, but interpretation is clouded by uncertainties, some of clinical definition, some technical.

Although the publishers and editor make no mention of a conference, the book reads very much like one. Each section ends with a summary by one of the experts, and one (Nyberg-Hansen) lets slip "a recent study reported at the symposium..." but to be fair, he does not say which one. If this is a symposium in print, why is this not plainly stated? If not, then the editing, writing and format should be upbraided.

Olesen's book is a valuable source of contemporaneous data for migraine researchers.

JMS PEARCE

The Psychoses of Epilepsy. By MICHAEL R TRIMBLE. (Pp 210; Price \$77.50.) New York, Raven Press, 1991. ISBN 0 88167 739 6

Very few subjects in neuropsychiatry have succeeded in exerting such a sustained hold on the clinical imagination as *The Psychoses of Epilepsy*; while among psychiatrists in particular this group of disorders has taken on a new significance in the search for an organic model for psychosis. Publication of this book is therefore timely.

The first half of the book examines the existing classifications for the epilepsies and for the psychoses and provides a summary description of limbic system structure and function. Aetiology, phenomenology and treatment of the inter-ictal, post-ictal and post-operative psychoses are dealt with in the second half. The clinical sections in particular are densely referenced and the book is a valuable resource for those wishing to pursue studies in this area. Methodologically, many of the studies fall rather short of the mark which may explain why so many of the controversies—forced normalisation, laterality of focus and so on—continue to rage unabated. The author's concluding summaries at the end of each section, lucid and balanced, are therefore most welcome. The book is not without its blemishes. The burning of the midnight candle is evident in a liberal sprinkling of factual errors. The reviewer was grateful to find mention of several of his papers but dismayed to encounter sizeable numerical mis-quotations in two of them. This aside, the book can be confidently recommended to those with an interest in understanding the organic contribution to abnormal experience and behaviour.

BK TOONE

Transient Amnesia: Clinical and Neuropsychological Aspects. Major Problems in Neurology Series 24. By JOHN R HODGES. (Pp 161; Price: £25.00.) London, W B Saunders & Co. 1991. ISBN-0-7020-1553-9.

This monograph begins with a review of the literature on the clinical syndrome of Transient Global Amnesia (TGA) and a discussion of the aetiological theories for this disorder. It is immediately apparent that many of the previously published series have been heterogeneous, containing not only patients with the distinctive disorder described by Fisher and Adams but also patients with additional, and atypical, clinical features suggesting a different aetiology. A description of the author's personal series of 114 patients with TGA begins by defining strict diagnostic criteria. The clinical features and epidemiology of the syndrome are reviewed including several descriptions of the author's personal observations of patients during attacks. The convincing epidemiological evidence against a thrombo-embolic cause for typical TGA is presented and the relationship between migraine and TGA discussed. The author concludes that TGA fulfilling his diagnostic criteria is a benign disorder with a good prognosis and a low risk of recurrence except in a small subgroup of patients who subsequently develop epilepsy.

In contrast, TGA with atypical features has a poorer prognosis and is thought frequently to be a manifestation of cerebrovascular dis-