both intracerebral and subdural in location.1 The apleoectopic presentation of menin-
giomas has been noted in cases with and without haemorrhage.1 Ischaemia, haemor-
rhage, and surgery 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 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 representa-
tive tissue from all evacuated haematomas for pathological analysis. Although the like-
lihood 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 articular
dyspraxia

In articular dyspraxia, multiple errors in articulation are produced in the absence of
damage to the motor or sensory pathways directly controlling the articular muscul-
iture. 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” articular dyspraxia caused by focal cerebral trauma and sub-
sequent 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 brusing and some soft tissue swelling in
the left parietal region. He was fully con-
scious and alert with normal higher intellec-
tual 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, bul-
var function was preserved with normal
swallowing, cough, palatal, and tongue
movements. No focal signs were apparent in
the limbs and reflexes were normal and sym-
metrical with flexor plantar responses.

Detailed assessment of language function
revealed normal auditory and written com-
prehension 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 intona-
tion. 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 record-
ings 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 con-
cluded that he was suffering from articular
dyspraxia without dysphasia. This was con-
firmed using the Boston Diagnostic Aphasias
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 demon-
strated a small area of focal cortical atrophy
in the left fronto-parietal region at the site of
the previous haematoma (figure b). An electro-
cerephalogram 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!

Articular dyspraxia is a distinctive dis-
turbance 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 hemi-
sphere strokes, being confused with the
associated dysphasia. The term articular
 dyspraxia is generally attributed to Liep-
mann1 and was popularised by Critchley.2
However, numerous other terms have been
used to describe the disorder including
aphasia, pure anarthria, pure word dumb-
ness, and pure motor aphasia.3

The often close association of articular
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” articular dyspraxia
demonstrated lesions in the inferior motor
strip of the left hemisphere.1 These lesions
included damage to both cortical and subcor-
tical tissue. CT and MRI studies in a further
patient showed a similar though more exten-
sive lesion affecting large areas of precental
and postcentral white matter.4 The latter
authors also reported a left handed patient
with the disorder caused by a corticousubcor-
tical haemorrhage in the lower part of the
right precentral gyrus. Angiography dem-
strated an underlying arteriovenous malfor-
mation.

In the present right handed case, also with a

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.
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 lesion 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.

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1 Liepmann H. Das krankheitsbild der apraxie ("motorischen asymbolie") auf grund eines falles von einzelnen apraxie. Monatschrift fur Psychiatrie und Neurologie 1900;8:15-40.


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 that there remain several groups of workers using different methods; applied, different workers appear to obtain divergent results. Transcranial Doppler which shows velocity and by inference flow in the basal arteries is compared to tests both xenon 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 oligaemia 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) lists a "recent study reported at the symposium ... " but to be fair, she 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 upgraded.

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

JMS PEARCE


Very few subjects in neuropsychiatry have succeeded in eliciting 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 psychoses. 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 the limbic system and its 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 the clinical imagination of The Psychoses of Epilepsy.

BK TOONE


This monograph begins with a review of the literature on the clinical syndrome of Transient Global Amnesia 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 brief account of the condition is presented and the symptoms 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 author does not appear to have been disheartened by this discussion. 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-
Cerebral localisation in articulatory dyspraxia.

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