Proceedings of the winter meeting of the British Neuropsychiatric Association, London Zoo (Regent's Park), 20 January 1995

The topic of the meeting was the neuropsychiatry of vascular disease.

Dr M Brown (London) gave the key note lecture on **vascular dementia: fact or fiction**. After a historical introduction he reviewed recent studies suggesting that the lifetime prevalence for vascular dementia may be comparable with that of Alzheimer's disease, that it may account for 20% of all cases of dementia, and that it may contribute to a further 20%. Several neuropathological mechanisms may be involved. Multi-infarct dementia usually results from multiple emboli leading to infarction of large volumes of brain. By contrast, Binswanger's disease is associated with diffuse atrophy of the subcortical white matter and the characteristic CT and MRI appearances known as leukoariosis. Subcortical ischaemia, the most likely mechanism, may be due to low perfusion caused by arrhythmias or hypotension. Lacunar state is another type of vascular dementia, which can coexist with Binswanger's disease. The clinical diagnosis of vascular dementia is difficult and the ischaemic score is an unreliable guide. Greater accuracy can be achieved by combining clinical history and imaging, but caution is needed in interpreting CT and MRI findings as leukoariosis is also present in Alzheimer's disease and in up to 20% of normal elderly subjects. The management of vascular dementia includes screening for vascular risk factors and investigation of sources of emboli. Treatment, aimed at controlling these risk factors, includes antiplatelet therapy, anticoagulation, and control of diabetes and hypertension. It is also important to avoid hypotension as it may precipitate Binswanger's disease.

The controversy surrounding **affective changes after stroke** was discussed by Dr S Reich (London). Early American studies have reported a relation between depression after stroke and the presence of lesions close to the left frontal pole. These studies have examined patients soon after stroke and have as a rule excluded those with severe aphasia. Studies with more representative samples and those including longer follow up have failed to replicate these findings, although an association between depression and anteriorly placed lesions may still be present. Areas for future study are the characterisation of symptoms of depression after stroke, the role of previous psychiatric illness, and the possibility that undetected pre-existing vascular disease may predispose to affective symptoms.

A talk on the **management of mood dis-**

order after stroke was given by Dr N Lincoln (Nottingham). She discussed the need to detect and treat depression after stroke. Her own studies have suggested that patients treated in specialised stroke units adjust better psychologically and have less mood disturbances a year later than those treated in conventional wards. She also highlighted the more successful psychological adjustment achieved by those who entered a leisure rehabilitation programme. Pilot studies of cognitive behaviour therapy given early after stroke are also promising.

Professor S Newman (London) discussed the **neuropsychiatric consequences of coronary artery bypass surgery**. Surgery for ischaemic heart disease is now commonplace with a risk for neurological complications of less than 5%. Professor Newman's own studies comparing preoperative and postoperative performance have shown that about 30% of patients have some degree of cognitive impairment when tested eight weeks after surgery and that the same proportion remained cognitively impaired 12 months later. Reduction of microembolic events during surgery by using filters in the arterial line resulted in a dramatic reduction of postoperative cognitive impairment. Subjective memory complaints were also present in about a third of the patients in the early postoperative period, but correlated poorly with the presence of cognitive impairment and were in most cases indicative of depression. The presence of type A personality and an internal locus of control were good predictors of depression.

Dr J Hodges (Cambridge) presented his work on **amnesic stroke syndromes**. Severe amnesic syndromes can result from bilateral thalamic strokes. After an initial period of impaired consciousness and abnormalities of voluntary upward gaze, an amnesic syndrome similar to the Wernicke-Korsakoff syndrome (profound anterograde and temporally graded retrograde amnesia) can be seen. Autobiographical memory is particularly affected and delusional paramnesia may be present. Bilateral thalamic damage disconnects frontal and medial temporal memory systems and results in a breakdown of the retrieval-verification processes critical for the recall of autobiographical memories mediated by the frontal lobes. A similar amnesic syndrome may result from forebrain damage as a consequence of bleeding anterior communicating aneurysms. Left posterior cerebral artery infarction results in verbal memory

impairment, right hemianopia, and alexia without agraphia, whereas contralateral infarction results in subtle non-verbal memory deficits and prosopagnosia.

The meeting ended with Dr R Wise (London) presenting his research on **language and the brain, as seen with PET**. It is now possible to study regional cerebral blood flow and oxygen extraction in patients during the acute stages of aphasic stroke. Early studies highlighted the effects of subcortical lesions on cortical structures and demonstrated perfusion deficits in the infarcted areas. Recent activation studies in single subjects have increased our understanding of the process of language recovery after stroke. Measurements of regional cerebral blood flow during a verbal generation task suggest that

the survival of the "edge of infarct tissue" is associated with recovery. Laterality shift (the activation of new areas in the contralateral hemisphere) was not evident using this paradigm, and it is uncertain whether it may play a part in language recovery.

The BNPA summer meeting will take place in Cambridge on 16–17 July 1995. The topic on 16 July will be **the neuropsychiatry of movement disorder**. On 17 July the British Association of Psychopharmacology will join the BNPA and the topic will be **the psychopharmacology of brain imaging**. Members will be invited to present papers and clinical cases.

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