

In our patient an acute akinetic state, which may have reflected an acute reduction of functional dopaminergic activity, was followed by a sustained improvement in the TS. This gives further support to the hypothesis that dopaminergic or other functionally related pathways are involved in the aetiology of TS.

#### References

- CAMPBELL, A. & BALDESSARINI, R. J. (1985) Prolonged pharmacologic activity of neuroleptics. *Archives of General Psychiatry*, **42**, 637.
- LADER, M. (1983) Benzodiazepine withdrawal states. In *Benzodiazepines Divided* (ed. M. R. Trimble), pp. 17–31. Chichester: Wiley.
- MEYER, B. C. & ROSE, D. (1986) Remarks on the etiology of Gilles de la Tourette's syndrome. *Journal of Nervous and Mental Disease*, **174**, 387–396.
- RASTOGNI, R. B., LAPIERRE, Y. D. & SINGHAL, R. L. (1978) Some neurochemical correlates of "rebound" phenomenon observed during withdrawal after long term exposure to 1,4-benzodiazepines. *Progress in Neuro-Psychopharmacology*, **2**, 43–45.
- SHAPIRO, A. K. & SHAPIRO, E. (1968) Treatment of Gilles de la Tourette's syndrome with haloperidol. *British Journal of Psychiatry*, **114**, 345–350.
- SNYDER, S., TAYLOR, J. M., COLE, T. J., *et al* (1970) The role of brain dopamine in behavioural regulation and the actions of psychotropic drugs. *American Journal of Psychiatry*, **127**, 117–125.
- VON ECONOMO, C. (1931) *Encephalitis Lethargica: its Sequelae and Treatment* (trans. K. O. Newman). Oxford: Oxford University Press.
- WOOD, P. L., ETIENNE, P., LAL, S., *et al* (1984) Benzodiazepines and GABAergic of nigrostriatal neurons: lack of tolerance. *Progress in Neuro-Psychopharmacology*, **8**, 779–783.

Simon Wright, MB, ChB, MRCPsych, *Registrar in Psychiatry*; \*Malcolm Peet, MB, ChB, MRCPsych, *Consultant Psychiatrist, Northern General Hospital, Herries Road, Sheffield S5 7AU*

\*Correspondence

---

*British Journal of Psychiatry* (1989), **154**, 259–261

## Treatment of Mood Disorder Associated with Binswanger's Disease

EILEEN M. JOYCE and RAYMOND LEVY

Binswanger's disease is a cerebrovascular disorder affecting deep white matter and is associated with dementia and affective disturbance. In the case reported, the mood disorder was successfully treated with a combination of lithium and amitriptyline, resulting in an improved quality of life despite continuing cognitive decline. This underlines the importance of treating the affective component of organic dementing conditions on its own merit.

Binswanger's disease is a variant of cerebrovascular disease in which the periventricular and deep white matter bear the brunt of the neuropathological process and in which there are atherosclerotic changes in local nutrient arteries (Olzewski, 1962). The clinical accompaniment has been reviewed by Caplan (1985) and consists of a progressive dementia with or without neurological signs, beginning in middle or old age, and often associated with psychiatric disturbances such as depression, euphoria, irritability and anxiety. The dementia takes the form of a generalised slowing of

intellectual processes, memory impairment and disorientation.

The advent of computerised tomography (CT) and magnetic resonance imaging (MRI) has allowed changes in the white matter to be identified *in vivo*, and the neologism leuko-araiosis has been coined to describe such a finding (Hachinski *et al*, 1987). Whether leuko-araiosis actually represents Binswanger's disease is under debate (Hachinski *et al*, 1987) because, while the latter used to be a relatively rare finding at post mortem, the former is becoming an increasingly common observation.

Nevertheless, an association between the cardiovascular indices of stroke and hypertension with leuko-araiosis has been described (Kinkel *et al*, 1985; Inzitari *et al*, 1987) and, indeed, one report has convincingly demonstrated an association between leuko-araiosis, as an isolated finding, and intellectual impairment, suggesting that this might be a marker for early dementia (Steingart *et al*, 1987).

There are few studies of therapeutic strategies in Binswanger's disease, and no evidence to suggest that successful treatment of the cardiovascular complications either arrests or improves the neuropsychiatric features. Biemond (1970) reported that the cerebrovascular dilator hydergine improved cognitive function in one patient. There is only one case report, to our knowledge, of a beneficial effect of psychotropic medication on the mood disorder associated with Binswanger's disease (Summergrad, 1985). In this case, a monoamine oxidase inhibitor, tranylcypromine, but not a tricyclic antidepressant, produced a marked improvement in mood and day-to-day activities. However, this patient had a 14-year history of a relapsing severe depressive illness and on admission was found to have hypopituitarism. Thus the depressive illness may have been coincidental but not integral to Binswanger's disease. We report a case in which the first episode of an affective disturbance arose in the context of a diagnosis of Binswanger's disease, and which was successfully treated with antidepressant drugs.

### Case report

The patient was a 70-year-old man with a nine-month history of social withdrawal, lethargy, intermittent disorientation and memory problems. On admission to hospital he had psychomotor retardation, poor appetite and weight loss; this picture was punctuated on at least two occasions by 48-hour episodes of hyperactivity and excitability. Cognitively he was disorientated in time and displayed impairment of recent memory. Physical examination revealed dyspnoea, fast atrial fibrillation and hypertension. Extensive investigations confirmed cardiovascular disease but did not reveal any treatable organic cause for his mental state. A CT scan demonstrated ventricular enlargement and patchy changes in the deep white matter. An MRI scan was performed (Fig. 1), which showed periventricular white-matter lesions, with no evidence of cortical infarction.

These findings suggested a diagnosis of Binswanger's disease in association with cardiovascular disease and hypertension. The physical complications were treated successfully with a combination of digoxin and diuretics. In view of the observed rapid cycling of mood, lithium carbonate (800 mg/day) was initiated. Because of cardiovascular risk, antidepressants were initially withheld; however, as there was no improvement of mood after two



FIG. 1. Magnetic resonance image (spin-echo) demonstrating areas of high signal intensity within the white matter (focal) and around the ventricles (confluent).

months, mianserin was added, up to a dose of 120 mg/day. After three months on this combination of drugs his mood improved, he became orientated and his memory appeared normal. He then stopped attending hospital and failed to take his medication. This resulted in rapid deterioration and he became quite clearly depressed. A combination of lithium carbonate at the previous dose and amitriptyline up to a dose of 150 mg/day was initiated, with subsequent and significant improvement, which has continued for over a year.

Although the impression was that cognitive function had improved along with his mood, this was not borne out by formal neuropsychological tests. On admission there was no evidence of general intellectual decline, but he performed poorly on specific tests of memory (Wechsler logical memory and associate learning (Wechsler & Stone, 1945), Kendrick object learning (Kendrick, 1985)). After clinical improvement, he continued to perform badly on the memory tests, and in addition his performance IQ estimated by the Wechsler Adult Intelligence Scale (Wechsler, 1955) had deteriorated, perhaps indicating the onset of a more global dementing process.

### Discussion

Several neurological disorders exist in which the pathological process mainly affects subcortical structures, and in which there is a neuropsychiatric component characterised by psychomotor slowing,

memory impairment, difficulty with problem solving and mood disorder. Examples are Parkinson's disease, Huntington's chorea, and progressive supranuclear palsy – the so-called subcortical dementias (Albert *et al*, 1974; McHugh & Folstein, 1975; Albert, 1978). Furthermore, diseases predominantly affecting cerebral white matter can also develop this clinical picture, for example multiple sclerosis and Binswanger's disease (Ron, 1985; Caplan, 1985). Although there have been no systematic studies of the management of the psychiatric aspects of these diseases, the affective disorder is often considered to parallel the cognitive decline and to reflect the underlying neuropathological changes. In this report we have described such a case in which the patient's quality of life was improved, despite continuing cognitive decline, by successful treatment of the mood disturbance. This underlines the importance of treating the psychiatric component of organic dementing conditions on its own merit.

#### Acknowledgements

Dr Joyce is currently a Wellcome Trust Lecturer in Mental Health. We are grateful to Dr Maria Ron for making possible the performance of an MRI scan at the National Hospital for Nervous Diseases. The manuscript was typed by Mrs Maja Fisher.

#### References

- ALBERT, M. L. (1978) Subcortical dementia. In *Alzheimer's Disease: Senile Dementia and Related Disorders* (eds R. Katzman, R. D. Terry & K. L. Bick), 173–180. New York: Raven Press.
- , FELDMAN, R. G. & WILLIS, A. L. (1974) The 'subcortical dementia' of progressive supranuclear palsy. *Journal of Neurology, Neurosurgery and Psychiatry*, **37**, 121–130.
- BIEMOND, A. (1970) On Binswanger's subcortical arteriosclerotic encephalopathy and the possibility of its clinical recognition. *Psychiatria Neurologia Neurochirurgia*, **73**, 413–417.
- CAPLAN, L. R. (1985) Binswanger's disease. *Handbook of Clinical Neurology*, **46**, 317–321.
- HACHINSKI, V. C., POTTER, P. & MERSKEY, H. (1987) Leuko-araiosis. *Archives of Neurology*, **44**, 21–23.
- INZITARI, D., DIAZ, F., FOX, A., *et al* (1987) Vascular risk factors and leuko-araiosis. *Archives of Neurology*, **44**, 42–47.
- KENDRICK, D. C. (1985) *Kendrick Cognitive Tests for the Elderly*. Windsor: NFER-Nelson.
- KINKEL, W. R., JACOBS, L., POLACHINI, I., *et al* (1985) Subcortical arteriosclerotic encephalopathy (Binswanger's disease): computed tomographic, nuclear magnetic resonance and clinical correlations. *Archives of Neurology*, **42**, 951–959.
- McHUGH, P. R. & FOLSTEIN, M. F. (1975) Psychiatric syndrome in Huntington's chorea: a clinical and phenomenologic study. In *Psychiatric Aspects of Neurologic Disease* (eds D. F. Benson & D. Blumer), 267–286. New York, Grune and Stratton.
- OLZEWSKI, J. (1962) Subcortical arteriosclerotic encephalopathy. *World Neurology*, **3**, 359–375.
- RON, M. A. (1985) Multiple sclerosis: psychiatric and psychometric abnormalities. *Journal of Psychosomatic Research*, **30**, 3–11.
- STEINGART, A., HACHINSKI, V. C., LAU, C., *et al* (1987) Cognitive and neurologic findings in subjects with diffuse white matter lucencies on computed tomographic scan (leuko-araiosis). *Archives of Neurology*, **44**, 32–35.
- SUMMERGRAD, P. (1985) Depression in Binswanger's encephalopathy responsive to tranylcypromine: case report. *Journal of Clinical Psychiatry*, **46**, 69–70.
- WECHSLER, D. (1955) *Wechsler Adult Intelligence Scale Manual*. New York: The Psychological Corporation.
- & STONE, C. P. (1945) *Wechsler Memory Scale Manual*. New York: The Psychological Corporation.
- \*Eileen M. Joyce, MA, PhD, MRCP, MRCPsych, *Wellcome Trust Lecturer in Mental Health, Institute of Psychiatry, De Crespigny Park, London SE5 8AF*; Raymond Levy, PhD, DPM, FRCP, FRCPsych, *Professor of Old Age Psychiatry, Section of Old Age Psychiatry, Institute of Psychiatry, De Crespigny Park, London SE5 8AF*

\*Correspondence

---

*British Journal of Psychiatry* (1989), **154**, 261–262

## Wernicke's Encephalopathy in an 18-Year-Old Woman

STUART TURNER, LYNN DANIELS and STEVEN GREER

An unusually young woman (18 years old) who misused alcohol and developed Wernicke's encephalopathy is described.

Wernicke's encephalopathy is an infrequent but well-recognised complication of alcohol misuse in men

over the age of 20. We describe the clinical syndrome in a young woman who misused alcohol.