and the police. His wife was taken to hospital where, fortunately, she made a good recovery. He was charged with attempted murder and taken into custody. He denied any conflict with his wife; she was pregnant and they were both looking forward to the birth of their first child. He specifically denied any disagreement with her before they went to bed, while no sexual activity or attempted sexual activity occurred between them that evening. His wife, who left him after the assault and moved interstate, confirmed his story.

He came from a somewhat troubled background, in that his parents separated when he was a child, and for a time he was placed in a Boy's Home before reuniting with his father. He had a very close relationship with his father, both shared a common interest in black magic and seances. He was very distressed by the death of his father some years earlier and continued to maintain the contact with him by wearing some of his clothes on a regular basis. It was subsequently discovered that the article used in the apparent attempt to strangle his wife was the cord of the dressing gown previously owned by his father. Additionally the attempted strangling occurred very close to the anniversary of his father's death. Although the patient had a history of impulsive suicidal attempts, coupled with alcohol and drug abuse, these behaviours had not been evident for some time and there was no evidence to suggest that he had been so troubled, in any way, at the time of the attack on his wife. Again this history was confirmed by her. Our patient claimed that for some days prior to the attack his sleep pattern had been disturbed with night terrors/nightmares.

Physical examination and a range of laboratory investigations were normal. A skull x-ray, an EEG and CT scan of his brain were also normal. Neurological opinion was sought but no pathology was identified. Neuropsychological testing did not reveal any abnormality. An MMPI demonstrated a hypochondriacal profile.

At his trial, the defence raised the possibility of somnambulism which, at law, would fall within the category of non-insane automatism. He was found guilty by the jury and sentenced to 15 months hard labour, with a non-parole period of 11 months. This was suspended in favour of a probation order of three years with a condition to undertake any medical treatment as the supervising probation officer might direct.

Although it is clear that this man had an abnormal personality structure and some unusual interests, no convincing explanation or precipitant has ever been identified with respect to his assaultive behaviour. He had a history of disturbing dreams dating back to childhood. He had a vague partial recollection of one sleep-walking incident, some years earlier, when having gone to bed he woke up in the kitchen. No violence was exhibited on that occasion.

KENNETH P. O'BRIEN
ALAN N. E. FUGLER

Hillcrest Hospital & Northfield
Security Hospital, Adelaide
South Australia 5001

Tardive Dyskinesia and Parkinsonism

SIR: Tardive dyskinesia (TD) and drug induced Parkinsonism (PS) both occur in association with the use of neuroleptics. The co-existence of TD and PS has been documented (McCreadie *et al*, 1982) but interest has mainly centred on the unmasking or the amelioration of dyskinesia either by anti-cholinergic agents (Chouinard *et al*, 1979) or of GABA agonists (Gerlach, 1977). The subject has also been studied using multivariate statistics on populations who exhibit neuroleptic induced extra-pyramidal effects (Kidger *et al*, 1980). These studies report that three independent factors emerge which approximate to the clinical syndrome of Parkinsonism, oral dyskinesia and akathisia. No study appears to have specifically considered the relative severities of the TD and PS syndromes when they co-exist and no comparison has been made with a control non-dyskinetic Parkinsonian cohort.

The aim of our study was to test the null hypotheses that the severity of PS in a TD group is no different from that of a control group exposed to neuroleptics but without TD. We also examined the correlation between the severity of TD and PS in our index group. The groups were carefully matched for age, sex, and type of medication. The 16 patients in each group were rated on the Abnormal Involuntary Movements Scale (AIMS) (Guy, 1976) and the Webster rating scale (Webster, 1968). The criterion for diagnosis of TD was a clinically recognisable disorder and a score of two or more on any item on the AIMS. The patients were on long-stay wards and had a primary diagnosis of functional psychosis. All subjects were on neuroleptics at the time of assessment and 9/16 in each group had been on long-term anti-cholinergics.

The mean age of the index group was 64.75 years (range 50–82) and of the control group 63.82 years (range 50–87). The Webster scores were compared between the groups and were not statistically different ($t = 0.926$, d.f. 30, NS). There was no correlation between the total AIMS and Webster scores ($r = -0.08$). We conclude that TD patients over 50 years have PS to a similar degree as an age- and sex-matched group without TD. In addition, in the TD group, the severity of PS did not predict the severity of TD. The co-existence of TD and PS and our findings of an independence of their relative severities calls into question the currently held view that PS results from a blockade and TD a hypersensitivity of dopamine receptors (Marsden *et al*, 1980). In our view the two conditions are either mediated through different dopamine systems or through independent but related neurotransmitter systems which have not yet been fully elucidated.

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FEMI OYEBODE
HAMISH MCCLELLAND
The Royal Victoria Infirmary
Queen Victoria Road
Newcastle upon Tyne NE1 4LP

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Mania Following Bereavement

SIR: I read with interest Rosenman and Taylor's case report of mania following bereavement (*Journal*, April 1986, **148**, 468–70). The authors state that such reports showing this association are uncommon. I report two further cases.

Case reports: (1) A lady who had no previous psychiatric history, first presented aged 49 years, two months after the sudden death of her husband. He had taken his own life whilst she was at work. At first, she grieved appropriately but six weeks after her bereavement she became restless, irritable and garrulous. She returned to normal mood within one month following treatment with neuroleptic medication and ECT. Four years later her mother died of carcinoma. She grieved initially but soon became cheerful. By the time of presentation, one month after her bereavement, she was restless, overtalkative, sexually disinhibited, giggly and expressed paranoid ideas with regard to her neighbours and sons. She said that nursing her mother for eight years had imposed a great strain on her and that her behaviour was a reaction to the lifting of this strain. She became euthymic within two months on treatment with haloperidol but 18 months later presented with a further manic episode. This occurred three weeks after a celebration in her husband's family to which she had not been invited and she had been initially very upset. She has been well in the nine years since the last affective episode.

(2) A 58 year old lady with a previous history of bipolar affective disorder presented the day after the funeral of her husband who had died suddenly of a myocardial infarct one week previously. Within 24 hours of his death she became restless, overtalkative and insomniac. On admission she talked incessantly and maintained that she felt "hilarious" in spite of occasional tearfulness. She believed she had special powers of healing people and that the television was telling her what to do. She could hear her husband talking to her. Her mood gradually stabilised on treatment with haloperidol, but one month after the bereavement she became depressed. She was then successfully treated with an antidepressant and discharged. One year later she presented with depression requiring treatment with ECT. Her mood stabilised but after a visit to her husband's grave three months later, she became manic with mixed affect. This resolved and she has been well for the last six months.

Rosenman and Taylor discuss the mechanism of manic response to bereavement. They cite the Post *et al* (1981) finding that a previous history of affective disorder predisposes to a rapid onset of mania. These two cases support this: (1) with no previous history of affective disorder did not develop mania until six weeks after her husband's death and (2) with a well established bipolar affective disorder developed mania within 24 hours of her bereavement. That repeated episodes of illness establish a facilitated pathway by which rapid changes of mood could occur, may be further supported by the recurrence of mania following case 2's visit to her husband's grave 15 months after his death, and following case 1's perceived rejection by her in-laws.

LISETTA LOVETT
University of Wales College of Medicine
Whitchurch Hospital,
Cardiff CF4 7XB

Reference

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SIR: The case report by Rosenman and Taylor (*Journal*, April 1986, **148**, 468–470) of mania following bereavement was of considerable interest. I report another two cases.

Case reports: (1) A 46 year old divorced engineer was admitted as an emergency in a hypomanic state on the evening of his mother's funeral. Instead of returning home from the funeral he had gone to his place of work where his behaviour had caused concern, the work's medical officer had arranged admission. On admission he was dressed in a