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PSEUDO-BEREAVEMENT IN THE MUNCHAUSEN SYNDROME

DEAR SIR,

In their paper on Feigned Bereavement (*Journal*, July 1978, **133**, 15-19) Dr J. Snowdon *et al* describe twelve cases of a relatively common variant of the Munchausen Syndrome. Fifty per cent of my series of 12 cases of the Munchausen Syndrome described a feigned bereavement. The characteristic pseudologia phantastica of the Munchausen is only likely to be either noticed or effective when it deals with emotionally gripping topics, and personal danger or loss is a most suitable theme, both in relation to its reliable effect on the audience's sympathy and in reducing the likelihood of especially searching questioning, such is our usual social embarrassment when faced by grief. So it is tactically useful.

Pseudo-bereavement was a notable feature of three of the four in my series whom I was able to investigate and treat over a significant period of time. In each case, their 'bereavement' was especially poignant: in one, his young wife had died of breast cancer while in bed with him, on their first wedding anniversary, after suckling their new-born child; in another, while on holiday abroad, he'd returned to his hired car only in time to see it struck by a drunken driver, when it burst into flames cremating his mother while he watched; the third had recently suffered the death from leukaemia of his young wife. In each of the four cases studied in detail, real bereavement or loss of a major relationship had been a significant factor in relation to the start of their hospital addiction. The true and lasting sense of loss was embellished so as to affect others comparably to the depth of the patient's own feelings. One persistently referred to his lies as 'exaggerations'. His lies often convince us because he has first convinced himself; although the facts are wrong, the emotional tone is usually correct.

The Munchausen behaviour is definitely not consciously determined or even fully consciously formulated. Those who have entered therapy confirm their frustration with the compulsion to behave as they do, which interferes with other, consciously-formed plans. They describe episodes of 'finding' themselves in hospital again, without full awareness of how they got there. External documentation confirmed that they had had a very disturbed

childhood, with separations and abandonment by parents. They showed several features of the personality disorder described in relation to the Borderline Syndrome, and were similarly sensitive in relation to the Borderline Syndrome, and were similarly sensitive in response to rejection and abandonment.

I see no reason why the presence of depression should in itself lead to the slightest doubt that these patients are cases of the Munchausen Syndrome. The Syndrome does not confer immunity to depression, and the circumstances of their childhood and adult life are such as to make the development of a depression not only possible but even likely. Depression is often a real concomitant of the Munchausen Syndrome, and treatment of the depression may be important in helping the patient control his maladaptive behaviour. Of course, psychiatric symptoms themselves may be simulated, as in one of my series, but this is less common than might be expected. (The two cases of Cheng and Hummel, *Journal*, July 1978, **133**, 20-1) certainly do *not* confirm their claim that 'many . . . present with psychiatric symptoms'.

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NECROPHILIA, MURDER AND HIGH INTELLIGENCE

DEAR SIR,

Dr Lancaster's lucid case report Necrophilia, Murder and High Intelligence (*Journal*, June 1978, **132**, 605-8) raises interesting points in both legal and clinical areas.

Psychiatrists with a forensic concern particularly might have welcomed a fuller account of the legal aspects of this case. Notwithstanding the comments on 'Defect of Reason', 'Disease of the Mind' and 'Nature and Quality' it would be illuminating to know why the defence chose to plead insanity under the McNaughton Rules rather than diminished responsibility under the Homicide Act, 1957. As Dr Lancaster says, 'once the defendant admitted to the stabbing there was little chance of his not being convicted of murder'. Quite so; Walker (*Crime and Insanity in England*, Vol. 1. Edinburgh 1968) states: 'it is surprising that Counsel should still think the defence of insanity worth attempting . . . If it is easier to show on the evidence that the client is suffering from diminished responsibility, why embark on the more difficult task of convincing a Judge and Jury that he did not know the nature and quality of his act, or did not know that it was wrong?'

Consideration of the evidence introduces the clinical aspects. Dr Lancaster establishes evidence of

confusion by eliciting a patchy memory, depersonalization, time disorder, disorder of visual perception, return to previous haunts and disturbance of sleep, and evidence of changed personality by what might be termed extreme 'out-of-character' acts. He suggests that these phenomena were caused by alcohol (in the earlier two necrophiliac episodes) and a combination of alcohol and clonidine (on the night of the stabbing). The contribution of clonidine may arguably be discounted. Even if all six tablets of 0.025 mg were ingested the dose is not of much magnitude. Hypertensive patients on clonidine may take up to ten times that amount per day and three times as much in a single dose. There is no known evidence that in dosage of 0.15 mg there is any significant interaction with alcohol.

However, on each of the three critical dates there is evidence of heavy alcoholic indulgence. Perhaps the Jury inclined to the view that the defendant's aberrations of behaviour were simply the result of drunkenness. That is understandable but there is an alternative formulation.

The clinical findings listed are all consistent with a diagnosis of temporal-lobe epilepsy, including the defendant's remembered feelings of fear. Ingestion of alcohol features high on the list of known precipitants and indeed stress is widely thought to play a part. The defendant's landlady appears to have been the last witness to have seen him and that a few hours before the crime. He is said to have 'had a glazed look, stared silently and did not accept a cup of tea offered'. She may well have been describing the state of altered consciousness occurring in an individual who is experiencing an epileptic fugue, rather than someone who is simply drunk. Was this approach considered and was the EEG investigated? A defence based on epileptic automatism and a plea of diminished responsibility might have enjoyed a more favourable outcome.

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DEAR SIR,

In my experience a plea of diminished responsibility has always involved the defendant knowing that he was killing someone but there were reasons which substantially impaired his mental responsibility for his act. The defendant had no real idea that he was killing someone even though he knew that he was stabbing at something in the bed; hence a plea of insanity was more appropriate. Epilepsy and particularly temporal lobe epilepsy was carefully considered and excluded from diagnosis by each of the

four psychiatrists who examined the defendant. The EEG was normal during routine examination but was not tested with the defendant under the influence of severe alcoholic intoxication. I would have very much liked the opportunity to have carried out this test.

I would not accept the behaviour as being simply due to alcoholic drunkenness but would accept that the defendant was one of those unusual persons in whom alcohol not only induces 'simple drunkenness' but in addition a most complex change of personality: 'Jekyll and Hyde' effect.

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SPECIFICITY OF SLEEP DEPRIVATION IN DEPRESSION

DEAR SIR,

There is substantial evidence that the majority of depressed patients deprived of sleep for up to 36 hours on one or more occasions show some degree of clinical improvement during the course of this regimen. In some instances, a single sleep deprivation treatment is said to be followed by sustained remission of depressive symptoms (e.g. Pflug, 1976).

Reviewing the then available studies, Roy and Bhanji (1976) cautioned that, as in the case of insulin coma therapy, the essential elements of the treatment could prove non-specific. Given that improvement sometimes occurs when depressives are treated solely by a placebo (e.g. Medical Research Council, Clinical Psychiatry Committee, 1965), this possibility cannot be ruled out. However, in experimental studies of a single case—a 73-year-old depressed lady—we have found that improvement in clinical state, as judged by 'blind' ratings on a 100 mm bipolar visual analogue scale (mania-depression), is contingent on the passage of an apparently critical period of wakefulness. This relationship has held under four conditions, and has done so on replication of each one (see Figure). (The four conditions were: the patient awoke as usual at about 6 a.m. and remained awake for the next 36 hours (○); 21 hours of wakefulness following nearly 3 hours of sleep taken between 2 and 5 a.m. (△); 21 hours of wakefulness, beginning at approximately 1 a.m., following an awakening 3 hours after sleep onset (□); and 21 hours of wakefulness following nearly 3 hours of sleep taken between 2 p.m. and 5 p.m., i.e. acute reversal of the sleep-wakefulness cycle (●). In all instances, the duration of sleep was ascertained by EEG recording, and the duration of wakefulness timed from the point at which the recording had been terminated). As the Figure shows, under these conditions, a