only a month or two. No alternate psychiatric diagnosis could be applied to these patients, for whom schizoprenia and anorexia nervosa had been diagnosed previously. Perhaps, in the case of the first lady, continued medication masked more florid psychotic features. Both patients on occasions appeared crudely manipulative, attention-seeking, and unreliable.

It would be interesting to see more accounts of patients with possible homosexual erotomania and whether, like the two I have described, such patients fulfil at least as many of the diagnostic criteria as their heterosexual counterparts. Also, a pooling of various results might lead to the development of a successful treatment of this distressing condition.

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Serial Cognitive Profiles in Levodopa-induced Hypersexuality

A patient with recent-onset Parkinson's disease was tested for mood, physical disability, and cognition, before treatment and then during and after a period of levodopa-induced hypersexuality. The effects of different anti-Parkinsonian drugs on cognition and behaviour are described. The unique cognitive data from this case support the hypothesis that hypersexuality is a manifestation of enhanced libido and not frontal disinhibition.

Eight per cent of patients with Parkinson's disease report restoration of sexual activity after treatment with levodopa (Duvoisin & Yahr, 1972), which has often been attributed to improved physical performance. However, restored sexual activity can arise in the absence of motor improvement, and frankly hypersexual activity occurs in 0.9% of levodopa patients (Goodwin, 1971).

Parkinsonian hypersexuality usually arises amidst the agitation, confusion and sometimes euphoria of an acute brain syndrome. A small number of cases have been reported occurring in clear consciousness, of which the present case is an example, and they are of special neuropharmacological interest since, in some cases, hypersexuality may correspond with peak levels of levodopa (O'Brien *et al*, 1971; Quinn *et al*, 1983). There have also been reports of levodopa-induced hypersexuality being enhanced with deprenyl, and of increased sexuality due to bromocriptine, which may bring longstanding deviant sexual practices to light (Quinn *et al*, 1983).

The effect of dopaminergic drugs in producing hypersexual behaviour has also been shown in male mammals (Baraldi & Bertolini, 1974; Malmnäs, 1976; Paglietti *et al*, 1978), and is blocked by pimozide, suggesting that the dopamine system is involved in enhancing sexual drive. Inhibition of sexual behaviour is thought to be due to the action of serotonin (Tagliamonte *et al*, 1969), which is displaced from its intraneuronal storage sites by levodopa (Wüerthele & Moore, 1977). Thus it has been suggested that levodopa might promote sexual behaviour by affecting both chemical systems.

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The anatomical basis for these effects is unknown, and it is unclear whether disinhibition of frontal cortical control over sexual behaviour could play a part. In Parkinson's disease, certain neuropsychological tests of frontal functioning appear to be selectively impaired early in the course of the illness (Lees & Smith, 1983). This has been attributed to the progressive dopamine denervation of the frontal cortex in a number of studies, reviewed by Harvey (1986). Levodopa treatment would therefore be expected to improve frontal test performance. It has recently been suggested that, although this may occur in some cases, the doses required to treat the movement disorder may be excessive for systems subserving frontal functions, impairing them further due to an 'overdose' effect (Gotham *et al*, 1986). In the light of this observation, it would be worth knowing if levodopa-induced frontal functions deterioration is necessary for hypersexual behaviour to occur, as would be expected if disinhibition of sexual restraint is an important factor. The case presented affords the first known opportunity to test this hypothesis.

Case report

A retired, separated, 64-year-old steelworker, living with his daughter, developed Parkinson's disease two years ago and was referred to the psychiatrist with panic attacks and depression showing a mixture of endogenous and neurotic features. He was admitted for psychiatric treatment two months after the onset of Parkinson's disease, and psychological tests were performed then before he ever received any anti-Parkinsonian medication. These consisted of four subtests from the Wechsler Adult Intelligence Scale (WAIS) (Wechsler, 1955): three to assess verbal intelligence – similarities, digit span recollection, and vocabulary – and one to test visuospatial perception – the picture completion test. Three tests were used to assess a range of frontal lobe function impairments: Nelson's Modified Wisconsin Card Sorting Test (MWCST) (Nelson, 1976), which obtains the proportion of perseverative errors during a cognitive set-shifting procedure using card categories, 50% being the cut-off point for frontal impairment; a Verbal Fluency Test, based on that used by Newcombe (1969); and the Modified Picture Arrangement Test, an untimed picture sequence recognition test derived from the WAIS picture arrangement test. Finally, the Beck Depression Inventory (Beck *et al*, 1961) was used to measure depression, and the King's College Rating (Quinn *et al*, 1982) to assess physical disability.

The results (Fig. 1) provide an estimated verbal intelligence of 100. The scaled subtest scores from the WAIS were above the means expected for his age, except digit span recollection, which was very slightly below the mean. The frontal functions tests showed perseveration above Nelson's

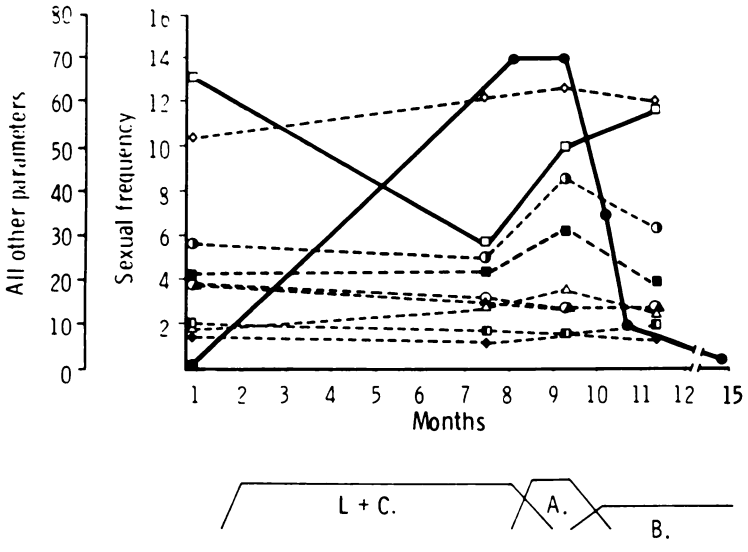


FIG. 1.

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cut-off point for frontal impairment, a verbal fluency in the lower normal range, and normal picture sequencing. Nomifensine was commenced with only slight improvement of his mood; when levodopa and carbidopa were added, his depression became less marked and there was some physical improvement.

He was discharged to the out-patient department, where before long a young female patient, whom he hardly knew, showed the staff a lurid better he had sent her in which he made her the object of his fantasised sexual activities, including cunnilingus. This was most unexpected since his mood was still sombre and at times tearful. His daughter reported that he had also started making nocturnal visits to a 12-year-old neighbourhood girl who was widely held to be of ill-repute. He would encourage the girl to visit him by giving her money. This was quite uncharacteristic of his usual behaviour, and he had not engaged in a relationship with the opposite sex since he and his wife had separated 13 years before. It was learnt that his libido had increased six weeks after the maximum dose of levodopa, 400 mg a day, was reached. There were sexual dreams and regular morning erections; voyeuristic interest in females, with accompanying erections and masturbation; and paederastic and rape fantasies. His sexual activity, measured by frequency of masturbation, had increased greatly. He conceded that his deviant propensities were of lifelong standing, though he had not acted on them before. At this time he was lucid and euthymic. His psychological tests were repeated (Fig. 1).

The only significant change in his cognitive functions while on the levodopa preparation had been a marked decrease in perseveration to within normal limits. In a systematised attempt to reduce his hypersexuality, during which the patient was unaware of any expectation of a drug-related change in his behaviour, the preparation was gradually replaced over a month with the dopamine agonist amantadine, at 200 mg a day. This strategy was justified by the generally accepted approach of treating early Parkinson's disease with the mildest effective agent. The weaker dopaminergic effect of amantadine did not affect his reported sexual activity or the WAIS subtests, though mood and physical state deteriorated and perseveration became more marked. Subsequent replacement of the amantadine with an anticholinergic agent, benzhexol, at 4 mg a day, was associated with a dramatic decline in sexual activity. His mood and physical state improved, although perseveration became even more marked. The WAIS subtests remained essentially unchanged throughout (Fig. 1).

It was not considered to be in the patient's best interests to attempt to precipitate a further episode of hypersexuality by reintroducing levodopa, and the reproducibility of levodopa's effect on libido has been long established. It has been possible to maintain him on anticholinergic agents and he has not experienced any further hypersexual episodes.

Discussion

This patient shows typical dopamine-induced hypersexuality, which was temporally unrelated to general cognitive decline, reduced physical disability, or improved mood. As has been shown to occur in Parkinson's disease (Lees & Smith, 1983), he had a selective frontal function impairment, which was shown on the MWCST before treatment. This impairment appears to have improved on the relatively low doses of anti-Parkinsonian agents used, to an extent proportional to their dopaminergic effects.

Although cerebral blood flow studies suggest that dopaminergic drugs may improve frontal metabolism (Guell *et al*, 1982; Bès *et al*, 1983), the direct effects of drugs on psychological tests of frontal functions are unknown. Still, it is well established that levodopa can produce improvement of other aspects of cognition, which occurs within about a month, then declines after some years of treatment (Rinne, 1983). This time-scale indicates that, in the present case study, the brief duration of treatment excludes an effect from declining pharmacological responsiveness of cognition. By withdrawing levodopa, a return to residual cognitive capacity is thought to occur within about 16 hours (Gotham *et al*, 1986). Thus cognitive effects from earlier levodopa treatment would not have added to those of the other drugs at the times their effects on cognition were tested. The tests therefore coincided with pharmacological influences of a specific kind.

The test results showed a selective deficit above the cut-off point for frontal impairment before and after the use of dopaminergic agents, which was repeatedly absent during their use, implying that they were benefiting frontal functions. Furthermore, it was only during treatment with these agents that hypersexuality was present. This demonstrates that frontal impairment, elicited with the MWCST, is not prerequisite for hypersexuality to occur. There remains the possibility of a more complex or variable relationship, and other tests of frontal cortex impairment, including *in vivo* imaging techniques, may be more appropriate to explore this area further.

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Secondary Affective Disorder in Survivor of Cardiac Arrest

A Case Report

A case of hypomania following cerebral anoxia due to cardiac arrest is reported. The concept of secondary affective disorder is reviewed and the need for early recognition of such cases is emphasised.

Secondary affective disorder is a recognised clinical entity but remains a neglected area in psychiatric literature, as pointed out by Lipowski (1980). Krauthammer & Klerman (1978) gave the concept of secondary mania and described its association with a variety of organic causes such as drug use, metabolic disorders, infections, intracranial neoplasms and epilepsy. Other organic conditions added to this list are cerebrovascular disorders (Jampala & Abrams, 1983), and head injury (Symonds, 1937; Tennent, 1937; Dalen, 1965; Whitlock, 1982; Khanna & Srinath, 1985; Bracken, 1987; Clark & Davison, 1987).

A wide range of disturbance of personality, judgement and behaviour have been reported in long-term survivors of cardiac arrest who are left with anoxic brain injury. Common personality changes after anoxic brain damage include increased irritability, verbal aggressiveness, violence, impulsiveness and moodiness – a constellation often described as 'affective incontinence' (Lishman, 1980). Mood change usually presents as depression in the setting of mild cognitive impairment (Reich *et al*, 1983), and it is suggested that depressive symptoms are unlikely to be reactive in nature as they usually persist long after apparent recovery from cerebral assault. Katz (1982)

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