skull films were few.⁵ The logical extrapolation is not, however, that all patients who might have had skull radiographs should undergo CT, but rather that CT is the examination of choice when the clinical condition reasonably raises concern about a treatable intracranial haemorrhage. Clearly, one would wish the threshold for suspicion to be such that there were few negative examinations, but that patients who needed scanning were not overlooked. The extra efforts required to organise emergency CT rather than skull films might effectively discourage poorly indicated requests.

IVAN MOSELEY

Lysholm Radiological Department, National Hospital for Neurology and Neurosurgery, Queen Square, London WC1N 3BG, UK

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A window on the role of dopamine in addiction disorders

The paper *Hedonistic homeostatic dysregulation in patients with Parkinson's disease on dopamine replacement therapies* by Giovannoni *et al* in this issue of the *Journal* (pp 423–8)¹ provides an important new perspective on the neurobiology of addiction.

These investigators have studied a small number of patients with Parkinson's disease who develop features of addiction to dopamine replacement therapy. The patients were mostly men with early onset Parkinson's disease responsive to dopaminergic therapy. They showed an increasing need for dopaminergic treatment in excess of that normally required to relieve parkinsonian signs and symptoms. The patients developed marked dyskinesias and felt "on" only during dyskinetic periods; they developed behaviours typical of drug dependent patients including drug seeking, drug hoarding, an unwillingness to reduce dopaminergic therapy, and accelerating increases in the amount of dopaminergic treatment used. The addiction produced impairment in social and occupational functioning with violent behaviour, interruption of marital relations and friendships, absence from work, and legal difficulties. Hypomanic or manic behaviour with psychosis was often evident during "on" periods; after withdrawal of dopaminergic replacement therapy a withdrawal state characterised by dysphoria, depression, irritability, and anxiety emerged. The pattern seemed to be exacerbated by the introduction of subcutaneous dopaminergic therapy. Other characteristics of the syndrome included obtaining a "kick" or "high" from use of dopaminergic therapies, hypersexuality, walkabouts (walking great distances while "on"), pathological gambling and shopping, and eating disorders and food craving. Treatment is marginally successful and involves the use of an atypical antipsychotic drug, limiting and monitoring of dopamine replacement therapy, and drug addiction rehabilitation if acceptable to the patient.

This unique and unusual syndrome provides direct evidence for a role for dopamine in addiction syndromes and supports a role for dopamine in similar addiction syndromes associated with cocaine and amphetamine. The brain region most likely involved in mediating these effects involves mesolimbic dopaminergic projections and the nucleus accumbens.

An important clue to the neurobiology of these disorders relates to the fact that hedonistic homeostatic dysregulation is rare in patients with Parkinson's disease. This suggests that a specific predisposition to the addictive effects of dopamine replacement therapy is present in affected patients. They may be genetically predisposed to addiction and efforts to characterise their unique genetic constitutions may provide an important insight into the neurobiological basis of addiction in general. These patients represent a natural experiment in which a pharmacological probe has disclosed a unique genetic predisposition that can be identified with results that may be generalisable to other addiction disorders. The syndrome represents yet another example of neurobiological insights and opportunities developed through careful clinical observations of patients with Parkinson's disease. This disorder continues to be a well spring of neuropsychiatric understanding providing insight into depression, psychosis, mania, anxiety, hallucinations, sleep and dream disorders, sexual deviations, and now addictive disorders.

JEFFREY L CUMMINGS

Departments of Neurology and Psychiatry and Biobehavioral Sciences, UCLA School of Medicine, Department of Neurology, 710 Westwood Plaza, Los Angeles, CA, USA
cummings@ucla.cdu

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