MATTERS ARISING

Hospital outpatient clinics, a neurology audit in South Catalonia

I read your letter about outpatient practice in Bristol, United Kingdom.¹ A similar audit in our hospital has some common aspects but differs greatly in others. The results are based on 245 new outpatient referrals to the consultant neurologist in Verge de la Cinta Hospital (Tortosa) between 1 January and 30 June 1991. This is a 202 bed district general hospital in the south of Catalonia, with a referral area population of 135 000 comprising the counties of Montsià, Baix Ebre and Terra Alta.

Fifty seven per cent (n = 139) of new outpatients were referred by the different departments of our own hospital, mostly internal medicine and traumatology, and 30% (73) by general practitioners. Four per cent (11) of those attending were categorised as "urgent" in referral letters, while 26% (64) were considered "preferent", an intermediate priority category, and 70% (170) "routine". Seventy per cent of total referrals were attending for a diagnosis, 27% for drug treatment or physical therapy and 3% for both reasons. The mean waiting time for the "urgent" group was two days, median 0.6, SD 2.7, range 0-8; mean waiting time for the "preferent" group was 14 days, median 11.6, SD 9.87, range 1-51; and mean waiting time for the "routine" group was 29.6 days, median 29.1, SD 11.40, range 7-61.

The preliminary diagnoses of new outpatients were similar in the three groups, without a significant relationship between priority category and presence or absence of a definite disease at consultation. The most common diagnoses, based on ICD classification, were migraine or headache (17% of total seen), disorders or peripheral nervous system (16%), mostly entrapment neuropathy and root lesion, epilepsy (13%), vague symptoms (12%), stroke (10%), Parkinson's disease (6%) and syncope (5%).

Forty four per cent (107) of new outpatients were discharged back to their referral source, while only 2% (5) were admitted to hospital after the consultation. Forty per cent (100) received outpatient specialised investigation, for example, 52 had a CT scan, 26 an electroencephalogram and 22 electromyography.

Only one patient of the "urgent" group was admitted to hospital after consultation, while the other four were of the "routine" group, one of them with myasthenia gravis.

Some of these aspects are similar to the audit mentioned previously, for example, the predominant diagnostic role of the neurological consultation, the main diagnoses, the small number of people admitted to hospital after consultation and some cases of inappropriate priority classification, but other results are very different. Our waiting time seems quite acceptable for the three priority categories, and this is probably the reason of the proportionately larger number of people in each a category and the small number considered "urgent". The short waiting time is explained by the number of sessions

devoted by the consultant to attending the outpatient clinic.

Another point is the high number of patients receiving specialised investigation. The main reason for this result is the highly restrictive criteria for hospital admission because of the problem of a shortage of beds. The great number of disorders of peripheral nervous system accounts for the EMG studies.

In conclusion, the results of this study are as follows: 1) A predominant diagnostic role of the neurological outpatient consultation; 2) A small proportion of patients with serious disease; 3) An acceptable waiting time, and 4) The considerable number of patients receiving specialised investigations.

M LÓPEZ ALEMANY
Neurology Unit, Department of Internal Medicine
Hospital Verge de la Cinta,
43500 Tortosa, Spain

1 Wood VA, Hewer RL, Campbell MJ, Colley JRT. Hospital outpatient clinics: an efficient specialist service? A neurology audit. *J Neurol Neurosurg Psychiatry* 1991;54:370-1.

The Brain in schizophrenia

The excellent editorial by Ron and Harvey¹ notes that "to have forgotten that schizophrenia is a brain disease will go down as one of the great aberrations of twentieth century medicine". However, I think it is open to question as to whether schizophrenia can be considered as a brain disease in the same way as established brain diseases such as vital or atrophic disorders of the CNS. There may be more general reservations with the validity of the concept of schizophrenia itself,² but I have four specific reservations with calling schizophrenia a brain disease:

- 1 Unlike most brain diseases, there is as yet no diagnostic pre- or post-mortem biological or other physical marker for schizophrenia.
- 2 Compared with most brain diseases, there is no predictable pattern of deficit in sensory or motor functions or in "primitive" reflexes.
- 3 Unlike most brain diseases, psychological or psychosocial variables play a significant part in the aetiology and stability of outcome of many patients with schizophrenia.
- 4 The relationship between neurobiological features of patients with schizophrenia and the pattern or severity of psychiatric disturbance is much more equivocal than in the case of analogous relationships in brain diseases.

I would therefore at present feel comfortable in calling schizophrenia a brain *dysfunction*, but I do not think there is yet sufficient evidence to call it a brain *disease*. It is possible that the term disease, if commonly applied to schizophrenia, may in the perception of some clinicians limit the range of viable therapeutic options.

NARINDER KAPUR
Wessex Neurological Centre, Southampton, UK

1 Ron MA, Harvey I. The brain in schizophrenia. *J Neurol Neurosurg Psychiatry* 1990;53:725-6.

2 Boyle M. *Schizophrenia: a scientific delusion*. London: Routledge, 1990.

Harvey and Ron reply:

Dr Kapur's reservations about describing schizophrenia as a brain disease are doubtless shared by others and seem to depend as much on how one defines "disease" as on

our current knowledge of schizophrenia. Indeed, it would be interesting to survey whether physicians, including neurologists, would include all the conditions they diagnose and treat as cerebral diseases within the limits laid out by Dr Kapur. Nonetheless, whether the term "dysfunction" or "disease" is preferred should not interfere with the logical process of defining more clearly what might be abnormal about the brain in schizophrenia. In our opinion there seems little danger of psychological and social therapies falling into neglect simply because we understand more about any underlying organic deficits; that would be treating the dysfunction rather than the patient.

IAN HARVEY
MARIA A RON
Institute of Neurology,
Queen Square,
London WC1N 3BG, UK

The Neuropsychological sequelae of attempted hanging

Medalia *et al*¹ have made a valuable contribution to our understanding of the neuropsychological consequences of attempted hanging. However, their use of the terms "hypoxia" and "ischaemia" may inadvertently add to the semantic confusion already present in the literature.

Specifically, according to the authors, "In circumstances other than combined cardiac and pulmonary arrest a relatively pure hypotensive or hypoxic state may occur; perhaps the best examples are cardiac arrest while intubated and ventilated during general anaesthesia (pure ischaemia) and carbon monoxide poisoning without circulatory collapse (pure hypoxia)." We find difficulty with this model. Firstly, while hypoxia may occur without ischaemia in chronic obstructive pulmonary disease or other cases of low levels of oxygen saturation, ischaemia from cardiac arrest, for example, cannot occur without rapid parallel compromise of oxygen delivery to the affected tissue. The presence or absence of ventilatory support is essentially irrelevant if there is no blood flow. That is, there is a state of "ischaemia with hypoxia." A term that is less clumsy is stagnant hypoxia,² which emphasises decreased oxygen availability due to decreased or arrested circulation. A model of the time course of stagnant hypoxia has been proposed which we have found useful to our understanding of "watershed" lesions.³ For example, while total circulatory arrest produces what has been called ischaemic hypoxia, the periods of lower but not zero blood pressure and blood flow surrounding total arrest (called oligemic hypoxia) demonstrate the particular vulnerability of watershed areas. It is during the oligemic phases that ventilation, or the lack of, may make a difference in lesion severity since without ventilation the hypoxia produced by circulatory slow down is that much greater. Incidentally, the term "oligemic hypoxia" also alerts us to reperfusion phenomena.

Secondly, it is useful to make a distinction between hypoxia produced by a lower availability of oxygen and that produced by the reduction of circulating haemoglobin as in carbon monoxide poisoning, if only because encephalopathic symptoms vary between the two aetiologies, at least in time of onset. The term for the case of haemoglobin com-