Is schizophrenia a neurodevelopmental disorder?

SIR,—In response to our leading article (19 September, p 681) Drs John L Waddington and Hanafy A Youssef point out (17 October, p 997) that schizophrenic patients with tardive dyskinesia have an excess of "developmental (primitive/neonatal) reflexes" and suggest that schizophrenics with anomalies in brain development are particularly susceptible to tardive dyskinesia. Their idea is an interesting one which is supported by the evidence that some preschizophrenics may already show involuntary movements[1] and also by the finding of Bartels and others[2] that tardive dyskinesia is especially common in "non-genetic schizophrenia"; we have shown that a history of pregnancy and birth complications is obtained more frequently from schizophrenics without a family history of psychiatric disorder than from those with such a history.[3]

Drs S J Cooper and D J King (24 October, p 1068) take us to task for regarding the possible aetiological roles of viral infections and birth complications as separate. We did not make our views clear enough; certainly we accept that viral infections during pregnancy may induce growth or other abnormalities in the fetus leading to secondary perinatal complications. The importance of viral infection during pregnancy has recently been emphasised by Mednick *et al*, who studied the rates of schizophrenia in young adults exposed during fetal life to the influenza epidemic which swept Helsinki in 1957[4]; those exposed to the epidemic during their second trimester of fetal development had an increased risk of later schizophrenia.

Dr M T Abou-Saleh (14 November, p 1278) suggests that brain damage caused by obstetric complications explains the scan abnormalities and cognitive deficits commonly found in so called type II schizophrenia. We concur with this and suggest that the deficits of affect and cognition which are seen in this type of chronic schizophrenia are actually continuations of longstanding deficits present before the onset of psychosis and dating from the initial brain insult. This goes against the traditional notion of schizophrenia as a deteriorating disorder.[5] Dr Abou-Saleh's point about higher rates of perinatal injury in developing countries is not easily resolved. We would simply note that severe mental retardation is a syndrome which numbers obstetric injury among its causes; yet, like schizophrenia, it shows a surprisingly stable global incidence.

Dr Abou-Saleh also states that it is difficult to reconcile the neurodevelopmental hypothesis with the genetic basis of the disorder but then goes on to suggest that early developmental anomaly might involve the very brain structures which mediate the effects of genetic factors. Exactly! Mednick *et al* consider that "a part of the genetic vulnerability to schizophrenia consists of a heightened sensitivity of the brain to perinatal insult."[4] Considerable evidence implicates the hippocampus. The genetic predisposition to schizophrenia might well comprise the inheritance of a pattern of neuronal migration into the hippocampus which is especially vulnerable to hypoxic-ischaemic damage.[5]

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1 Manschreck TC. Motor abnormalities in schizophrenic patients. In: Nasrallah HA, Weinberger DR. *The neurology of schizophrenia*. Amsterdam: Elsevier, 1986.
2 Bartels M, Mann K, Friedrich W. Tardive dyskinesia: marked predominance of nongenetic schizophrenia. *Biol Psychiatry* 1985;20:94-119.
3 Lewis SW, Murray RM. Obstetric complications, neuro-developmental deviance and risk of schizophrenia. *J Psychiatr Res* (in press).
4 Mednick SA, Parnas J, Schulsinger F. The Copenhagen high-risk project, 1962-86. *Schizophr Bull* 1987;13:485-95.
5 Murray RM, Lewis SW, Owen MJ, Foerster A. The neurodevelopmental origins of dementia praecox. In: McGuffin P, Bebbington P, eds. *Schizophrenia: the major issues*. London: William Heinemann (in press).

The Royal College of Psychiatrists and South Africa

SIR,—Dr Alistair King (5 December, p 1488) asks how the Royal College of Psychiatrists "gives in so easily to a group of bigoted and narrow minded anti-South Africa extremists."

The damage which apartheid policies have caused to South Africa's people in terms of both health and health services in general and psychiatric services in particular has been well and repeatedly documented. The appalling contrasts in living conditions between whites and blacks are described, for instance, in the report of the Commonwealth Eminent Persons Group,[1] which contains such "bigoted extremists" as Malcolm Fraser, Lord Barber, and the Primate of the Anglican Church of Canada. Their visit was part of the Commonwealth's response to the problems raised by South Africa, which led to the Commonwealth's Nassau Accord. This made a number of recommendations, which included "a discouragement of all cultural and scientific events except where these contribute towards the ending of apartheid or have no possible role in promoting it." This was adopted as a policy by our college earlier this year.

Its interpretation is, of course, a matter of debate, and we have specifically excluded the possibility of a complete academic boycott of South Africa. The original purpose of Professor Simpson's visit, and the reason for his invitation, was for him to give an account of his experience to a committee of our college which is particularly concerned with the abuse of psychiatry. This purpose was achieved.

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1 Commonwealth Eminent Persons Group. *Mission to South Africa*. (Commonwealth report, 1986.) London: Penguin Books, 1986.

Advanced training for ambulance crews

SIR,—In the article by Dr J M Rowley and colleagues (28 November, p 1387) the number of lives saved when ambulancemen used basic resuscitation techniques and defibrillation only was impressive.

In Chester we operate a flying squad service manned by a doctor which responds at the request of the first ambulance crew or general practitioner to attend collapsed patients. Our experience of out of hospital cardiac arrests is rather different. In the past 30 months 90 patients with cardiac arrest have been attended, 11 of whom have ultimately survived to leave hospital (12%). All the survivors were in ventricular fibrillation, but in only two cases were cardiac rhythms re-established using defibrillation alone. The remaining survivors required intubation and the administration of intravenous drugs as well as defibrillation before a pulse was re-established.

I would commend the use of defibrillators by emergency medical or paramedical staff who are first to attend collapsed patients. Our experience implies that back up by staff trained in the more advanced techniques of resuscitation would save even more lives. I would therefore support both supplying all ambulances with defibrillators and the development of a second tier service manned by staff with advanced training.

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SIR,—It seems that Dr J M Rowley and others (28 November, p 1387) have failed to imagine the aspects of this subject that lie outside the confines of their own discipline.

I wholeheartedly agree with their conclusion that the new training for ambulancemen is "an example of the excellent being the enemy of the good" if only the treatment of cardiac arrest is considered. In that situation, yes, the primary need is to defibrillate—and quickly.

Ambulances have this funny habit, however, of being called to patients who have been injured in car accidents and have suffered blood loss or have lost control of their airway or to patients who have taken too much insulin and need 50% dextrose fast—to name but two simple examples. In these life threatening but reversible conditions the timely intervention of an ambulanceman who can put up an infusion of plasma expander, support the airway, and if necessary intubate or give the required intravenous energy quickly may make the difference between the patient's next port of call being the hospital or the mortuary.

Of course, good cardiac care is an important skill for ambulancemen. So, too, is good trauma care and good life support care generally. Please, gentlemen, off with the blinkers.

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Toxic myocarditis in paracetamol poisoning

SIR,—We agree that there is evidence for toxic myocarditis in some cases of fulminant hepatic failure, including those due to paracetamol poisoning (31 October, p 1097), but many of the features described by Dr Riadh A Wakeel and others are consistent with a diagnosis of uncontrolled cerebral oedema and, ultimately, brain stem compression rather than features of cardiotoxicity per se.[1,2]

The sinus tachycardia followed by sudden arrhythmia, hypotension, altered level of consciousness, dilated pupils, absent brain stem reflexes, respiratory depression, and cardiac asystole are all consistent with such changes.[1,2] That absent pupillary responses and brain stem reflexes can be reversed in some cases by hyperventilation and by a mannitol induced diuresis would strongly suggest that uncontrolled cerebral oedema is at least a factor in their cause.[3] The absence of visible tonsillar compression at necropsy does not, in our experience, exclude brain stem coning as a mode of death, and the authors noted the presence of diffuse cerebral oedema. This is particularly important since early recognition and intensive control of cerebral oedema is a major factor in achieving a survival of 50-60% from fulminant hepatic failure even in those who reach grade III-IV encephalopathy.[3,4] We would have considered this 15 year old patient to have had a relatively good prognosis despite the undoubted severity of the poisoning in view of her youth, the maintained urine output, low level of serum creatinine, and maximum prothrombin time of only 65 seconds.

The pathological findings of Dr Wakeel and