emphasising the financial savings it allows. Outpatient ECT was chosen in our case because given that the physical examination, routine blood tests, chest X-ray and electrocardiogram were normal, it was felt beneficial for the patient to be spending most of his day among those he knew, particularly as hospital admission had contributed to his present mental state.

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Are polioviruses a cause of schizophrenia?

SIR: The thought-provoking commentary by Eagles on a possible role of polioviruses in the development of schizophrenia (*Journal*, May 1992, **160**, 598–600) raises the possibility that two other closely related groups of picorna enteroviruses (Coxsackie and ECHO) may also be involved in the genesis of some instances of schizophrenia. Like polioviruses, the incidences of Coxsackie and ECHO virus infections peak during the summer, both are neurotropic (Sells *et al*, 1975), and can be passed from mother to foetus (Modlin *et al*, 1981). Poliovirus sensitivity genes may encode several subunits of the membrane-bound picornavirus-receptor complexes that are members of the immunoglobulin super family, some of which may be identical to intercellular adhesion molecules (see Selinka *et al*, 1991). The picornavirus receptor subunits might be combined in various ways to yield multimeric receptor complexes that are more or less specific for certain viruses, but with some overlap. For example, Siddique *et al* (1988, cited by Eagles) found that several rodent–human hybrid cell lines, infected with appropriate fragments of human chromosome 19, could subsequently be infected by both poliovirus 1 and ECHO virus 11. If later reinfection is required for the full development of schizophrenia, as Dr Eagles suggests, the reinfection might therefore also be by one of the Coxsackie or ECHO viruses, in previously sensitised and immunocompromised individuals. The idea of required reinfection receives support from the epidemiological findings of Hare & Walter (1978) on the seasonal variations in the admissions of schizophrenic and manic patients with peaks in July and August, corresponding roughly to the peaks in the incidence of all three enterovirus infections. The somewhat earlier peaks in psychiatric admissions may be due to pre-existing sensitisation of these patients, with most non-sensitised individuals presenting with other symptoms of viral infection 1 to 3 months later. The peaks of enterovirus infections are followed, approximately six months later, by peaks in excess births of individuals who will, 25 to 30 years later, develop schizophrenia or mania. These findings suggest that infected mothers may pass a psychotogenic enterovirus to their foetuses during the first or second trimester of pregnancy when the foetal brain is most sensitive to infective damage, as Dr Eagles points out. Such early viral infection may result in selective destruction of excitatory glutamatergic and cholinergic neurons in the foetal brain (Squires & Saederup, 1991) as well as causing the foetus' immune system to treat the invading viral antigen as non-foreign, leading to permanent sensitivity to the virus.

The hypothesis of *in utero* sensitisation and immunocompromise by an enterovirus predicts greatly lowered levels of antibodies to this virus in schizophrenic adults and can be easily tested by determining the relative abilities of blood samples, taken from schizophrenic and non-schizophrenic adults, to neutralize various enteroviruses *in vitro*.

Perhaps repeated reinfections of sensitised individuals, over a lifetime, may be a cause of the progressive deterioration seen in schizophrenia.

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