avoid too frequent reimplantation. Eden's case represents considerable overdosage (200 mg every 3 months for 3 years) and I would not want readers without practical experience of this form of therapy to interpret the overdosage syndrome described as a significant risk in more routine practice.

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MOTONEURON DISEASE AND PAST POLIOMYELITIS

SIR,—Dr Martyn and colleagues (June 11, p 1319) suggest that motoneurone disease (MND) is a rare and delayed consequence of an infection with poliovirus. The role of poliovirus in the aetiology of MND remains equivocal1 because of the problems associated with prospective case-control studies and lack of laboratory proof of poliovirus infection in cases of MND.

Polioencephalitis is very common in India but the incidence of MND does not seem to be higher than elsewhere. Although exact population-based data are lacking, surveys of residual paralysis due to polioencephalitis reveal the annual incidence to be 15 per 100 000 in the general population.2 Poliomyelitis develops in about 70 000 children every year in India.3 The incidence must have been even higher in the past when community health services were lacking. Despite this high incidence of poliomyelitis, the occurrence of MND in the population is similar to the rest of the world (4 per 100 000, as quoted in an epidemiological study from Bangladesh).4 Hospital-based studies do not favour past poliovirus infection as a risk factor for later development of MND in this country. In a case-control study of antecedent events in MND at our centre, no case of past poliovirus infection was found in 43 cases. There is no indication of a rise in the incidence of MND in the past years. MND formed 0-58% of all neurological cases admitted to our centre, which is similar to the frequency of 0-11% in Chandigarh.5 These results are lower than the frequency of 3% in this country in 1969.6 The possibility of chronic polioencephalitis infection giving rise to MND was excluded by the failure to demonstrate in-situ hybridisation with a poliovirus probe on sections of spinal cord obtained from patients with this disorder.7

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DEVIKA NAG


LUPUS ANTICOAGULANT AND RECURRENT FETAL LOSS: SUCCESSFUL TREATMENT WITH GAMMAGLOBULIN

SIR.—Pregnant women with a lupus anticoagulant (LA) may be at higher risk for recurrent abortion and infants' deaths.1,2 These complications are associated with arterial or venous thrombosis.1,2 The LA may interfere with the production of procoagulants by the vessel wall and other tissues, including pregnant myometrium.3,4 Since prostacyclin may have a physiological role in pregnancy, inhibition of prostacyclin may have an adverse effect on fetal outcome. However, other mechanisms could also be involved.5

Treatment with high-dose prednisone (40-60 mg per day) in combination with aspirin (75 mg per day) may improve the outcome of pregnancies in women with an LA and a poor obstetric history.6,7 But such treatment for several months may cause serious maternal side-effects and the effect on the surviving fetuses is unknown.8 In addition, therapeutic benefit is not always obtained.9 Thus alternative treatments deserve evaluation. We describe here a patient with an LA and recurrent fetal loss who was successfully treated during pregnancy with human intravenous immunoglobulin (IV Ig).

This 28-year-old woman was referred to our hospital during week 16 of her thirteenth pregnancy. She had had nine early spontaneous abortions, two intrauterine deaths, and a perinatal death at 26 weeks. Mild hypertension and a false-negative VDRL test had been observed during a previous pregnancy. Other causes for the recurrent abortions were excluded. Physical examination and routine blood biochemistry and urine analysis were normal. Antinuclear factor, DNA antibodies, lupus erythematosus phenomenon, tests for rheumatoid factor, and cryoglobulinemia were all negative, and serum immunoglobulin and complement levels were normal. She had a false-negative test for rubella.


