minimising tissue damage; and bacteria, chemicals, and other damaging substances must not be introduced into the wound. It should be of the appropriate shape and design to permit rapid, accurate, and precise suturing. The needle is most easily manipulated if it is appropriate to the specific wound site and if the needle holder is the best one for that needle. Care in selecting the surgeon's needle will certainly contribute to better care of patients—and should also give greater comfort and satisfaction to the craftsman surgeon at his work.

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Subacute sclerosing panencephalitis

Subacute sclerosing panencephalitis (SSPE) is fortunately an uncommon disease. In the United States1 and in Britain2 it affects only one per million children each year, but the incidence seems higher in the Middle East.3 4 The mean age of onset is about 8 years, and there is an insidious process of personality change, deterioration in school performance, and difficulties with speech as common initial manifestations. Later myoclonic jerks are a prominent feature, and the disease then progresses remorselessly to spastic quadriaparesis and loss of intellect. The condition is usually fatal within one or two years, but a few children survive for much longer.

There is little doubt that SSPE is due to persistent infection of brain cells with measles virus. Inclusion bodies within cerebral neurones were described by Dawson in 1934,2 but it was not until 1965 that Bouteille et al6 drew attention to their similarity to measles virus on electron microscopy. Soon after this observation patients with SSPE were found to have high titres of measles antibody in the serum and the cerebrospinal fluid and measles antigen was found in brain tissue.7 In 1969 measles virus was cultured from the brain of a patient.8

Why these very few of the millions of patients with measles should develop a chronic infection of the central nervous system is unknown. Patients with SSPE have often had measles unusually early.129 In hamsters the effect of intracerebral injection of SSPE virus varied with age,10 chronic encephalitis being produced more readily in weanling animals. There may be racial and ethnic differences in susceptibility to SSPE; in the United States whites are more commonly affected than blacks, but in South Africa non-whites predominate.2 In Israel Arabs and Sephardic Jews seem more at risk than Ashkenazi Jews. In the United States the disease is more common in rural areas,11 an observation that led to a suggestion (unconfirmed by objective tests) that an added zoonotic infection might be implicated. The persistence of the virus in SSPE may be due, at least in part, to the presence of a high-molecular-weight factor which inhibits lymphocyte responses to it.12

Attempts at treatment have so far been largely unrewarding. Amantadine,13 5-bromo-2-deoxyuridine,14 and transfer factor15 have all been unsuccessful. Since 1974, however, there have been several case reports of improvement after treatment with isoprinosine, an antiviral agent; and Huttenlocher and Mattson have now reported a series of 15 patients treated with this drug.16 Five of the patients showed improvement lasting for two years or more, and there are six still alive over four years from the onset of symptoms (but two of these were long survivors before starting treatment). Critical analysis of Huttenlocher and Mattson's patients shows important differences from other series,17 18 which may have influenced the results.16 Their patients were older than usual (mean age at onset of symptoms 12-8 years compared with the usual 7 or 8 years) and the sexes were equally represented (males usually predominate by two or three to one). The 19 patients listed by Risk et al17 as having undergone spontaneous remission (six of their own patients and 13 from the literature) showed the same characteristics (mean age at onset 12-5 years, 9 boys, 10 girls). Some of Huttenlocher and Mattson's patients had very slowly progressive disease, which had lasted for up to six years before the start of treatment. Another recent report19 gives details of six treated patients, of whom four continued to deteriorate, one remained unchanged, and one showed minimal improvement.

Clearly isoprinosine is not a cure for SSPE. The drug seems to be of no value in the rapidly progressive disease, but it may increase the rate of remission in older children with slowly progressive disease. Larger controlled trials, probably requiring international co-operation, will be needed to establish the value of the drug. Possibly the publicity given to isoprinosine will stimulate the development and assessment of other drugs which may be more effective.

The other unresolved question about SSPE is whether or not live measles vaccine can cause the disease. Data from the United States SSPE registry20 suggest that the dramatic decline in measles between 1964 and 1968 is being reflected in a similar fall in incidence of SSPE since 1971 and that this fall is a consequence of immunisation. A small but constant number of cases of SSPE (about five each year in the United States) are being reported in patients with a history of immunisation with live attenuated virus but not of measles. With the decline of epidemic measles these cases are forming an increasing proportion of the total, rising from 12% in 1970 to 38.5% in 1974. Nevertheless, the risk of developing SSPE after measles seems to be about 10 times greater than the risk after vaccination. The best hope for the control of this vile disease seems to lie in high levels of vaccination and possibly in the development of new antiviral agents.

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