Few would disagree that tests of clinical competence are important and that they could be made much more objective. They should be supplemented by multiple-choice questions and careful assesment of clinical ability during the student's career. Even such apparent imponderables as medical students' attitudes can, it seems, be analysed and tested.[5]

Measles and Panencephalitis

Virologists have been increasingly turning their attention to subacute and chronic diseases of the central nervous system, many of which have hitherto had no satisfactory aetiological explanation. The impetus was increased by the findings that the chronic diseases visna, scrapie, and kuru appear to be caused by virus-like agents and by the concept of slow viruses and slow infections.[1]

Accumulating evidence that subacute sclerosing panencephalitis (S.S.P.) may be due to measles virus was recently reviewed and discussed at a conference at Bethesda in the U.S.A.[2] S.S.P. usually affects children of school age some years after they have had measles. There may be an insidious onset of intellectual deterioration or psychological disturbances, so that after a time the child becomes ineducable, or there may be a sudden onset with convulsions and a variety of neurological manifestations. Within weeks or months in either case the patient becomes bed-ridden, blind, and disorientated, the arms and legs are flexed and have plastic rigidity, myoclonic spasms occur 4 to 12 times a minute, the eyes wander purposelessly, and only painful stimuli elicit a feeble cry. Eventually coma leads to death unless there has been a previous fatal intercurrent infection. Remissions are common, however, and may last for weeks or years.

J. R. Dawson[3 4] first noted the presence of intranuclear inclusions in S.S.P. in 1934 ; later H. Pette and G. Döring[5] produced an encephalitis in rabbits, mice, and monkeys by inoculation of nervous tissue from patients, but no infective agent was identified. Numerous attempts to repeat these experiments have failed. F. E. Sherman and his colleagues[6] first demonstrated fluorescent staining of the cytoplasmic inclusions by labelled herpes simplex antibody. Other virus aetiologies have been suggested: infectious hepatitis,[7] vaccinia,[8 9] Coxsackie,[10] and the poliomyelitis vaccine virus.[11 12] Virus particles (60–80 mμ diameter)[13] have been described in astrocytes from the brains of patients with S.S.P. by several authors[14–16] but also in other diseases.[17]

At the conference R. M. Herndon and L. J. Rubinstein[13] reported that they had found particles indistinguishable from large myxoviruses budding from the cytoplasmic inclusions. E. H. Lennett and his colleagues[18] reported six cases of S.S.P. in which measles antigen could be demonstrated by fluorescent antibody in the nervous tissue in positions similar to the inclusion bodies ; all the cases had high levels of measles antibody—three of them showed increases over several months during the illness. Five of the patients had unusual amounts of measles antibody in the cerebrospinal fluid. B. R. Adels and his co-workers[19] reported on 55 cases: the levels of measles antibody in the S.S.P. were higher than those found in natural measles even if complicated by encephalitis. Repeated attempts to isolate virus from the nervous system in S.S.P. have so far failed.[18–20] S. A. Schneck[21] reported one case of S.S.P. with an onset three weeks after live measles vaccination and reviewed other central nervous system complications of live measles vaccination. In contrast J. Adams[22] described the pathological findings in the various forms of measles encephalitis which have the common features of cytoplasmic and intranuclear inclusions and small multinucleated giant cells.

There are four pieces of evidence that S.S.P. may be caused by measles virus: the presence of particles morphologically resembling it in the central nervous system ; the presence of measles antigen, demonstrated by fluorescent antibody ; exceptionally high levels of measles antibody, especially in the cerebrospinal fluid ; and suggestive evidence that measles virus invades the central nervous system in a proportion of cases even without clinical encephalitis.[23] Each of these pieces of evidence can be challenged individually,[2] but together they make a good case worthy of more intensive study. Isolation of the virus from the nervous tissue of patients with S.S.P. would be helpful and is probably the only way to produce incontrovertible evidence that an infection with measles exists ; the major difficulty may be the presence of a high antibody concentration, but this can be overcome if the infected cells can be cultured. The virus may, however, be present in a defective form with an altered mechanism of multiplication.[24] But the presence of virus does not in itself prove an aetiological relationship—it might be that the altered condition of the central nervous system in S.S.P. enables measles virus to persist. In the same sense it might be argued that the high level of antibody in these patients indicates merely exceptional immunological responsiveness, and that this might be related to the pathogenesis of the disease. However, there seems to be a reasonable probability that S.S.P. is caused by measles virus. Further support is given by the finding that measles encephalitis can assume a chronic form in which there is severe brain damage, including marked gliosis and diffuse demyelination.[22]

This type of consequence of persistent latent infections in

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the central nervous system was predicted by H. E. Webb and C. E. G. Smith[25] and the possible mechanisms explained. There is no reason to suppose that such lesions cannot be caused by any virus which can gain access to the central nervous system (and which need not be capable of causing acute encephalitis). Indeed, there is recent evidence that repeated infections with the same virus may cause subacute and chronic diseases of the central nervous system in experimental animals in which it does not cause acute disease.[26]

Perhaps the most important question is whether live measles vaccine can cause this disease. If it does it is likely to be a rare complication which will become apparent some considerable time after vaccination. It is therefore essential that in future trials of measles vaccination careful and long-term surveillance should be maintained, and the same consideration should apply to any new live vaccine. This risk must not be exaggerated, particularly in areas of the world where measles is a severe and lethal illness,[27] but when the risk can be assessed it must be balanced against the risks of measles itself.[28] Inactivated measles vaccines have already been abandoned because they can aggravate disease due to subsequent natural infection.[29-31]

Benign Sixth-nerve Palsy in Children

An external rectus palsy in a child raises the suspicion of serious neurological disease—raised intracranial pressure, an infiltrating glioma of the pons, and tuberculous meningitis being possibilities. D. L. Knox, D. B. Clark, and F. F. Schuster,[1] however, have drawn attention to the benign sixth-nerve lesions that can occur in children after minor febrile episodes or upper respiratory infections. They report 10 patients aged between 1 and 15 with this condition seen in the course of 13 years at Johns Hopkins Hospital. There was a history of febrile or respiratory illness from 7 to 21 days before the onset of the external rectus palsy in eight of the ten patients. Two of the children had had recent otitis media, and one had scarred ear drums, but in none of them was there pain in the ear or eye when the sixth-nerve palsy appeared. In none of these ten cases were other abnormal neurological signs found ; in six in whom lumbar puncture was done the cerebrospinal fluid was normal, and in five there was a relative lymphocytosis in the peripheral blood. The prognosis was good. Improvement started within three to six weeks and complete recovery had occurred within ten weeks in all except one child, who recovered completely in nine months.

The aetiology of this condition remains uncertain. The authors suggest that it may be comparable to Gradenigo's syndrome,[2] in which otitis media is complicated by an ipsilateral sixth-nerve lesion. C. P. Symonds[3 4] suggested that the cause of the sixth-nerve lesion in this condition was thrombosis in the inferior petrosal sinus. The sixth nerve and the sinus pass from the posterior to the middle fossa through a tightly fitting dural sheath, Dorello's canal, and compression of the nerve could readily occur if the sinus became thrombosed. Function in the nerve would return with organization and canalization of the clot. The alternative explanation for the benign palsies suggested from Johns Hopkins is that the nerve lesion is due to a viral neuritis.

The practical problem remains: how far should investigations be carried out in a child with an isolated external rectus palsy of sudden origin ? If there is a history of a preceding febrile illness and if there are no other abnormal neurological signs, normal *x*-rays of the skull and sinuses, no abnormality in the cerebrospinal fluid, and no response to pharmacological tests for myasthenia gravis, it is reasonable to delay other investigations and keep the child under observation for three to six weeks, when improvement should be starting if he is suffering from this type of " benign sixth-nerve palsy."

Heatstroke

Since John Davy[1] measured the penetration of sunlight through the cranium we have learnt a great deal about heatstroke. Though many uncertainties remain,[2 3] there is now a broad area of agreement about its causation, prevention, and treatment.[4] Heatstroke results from an imbalance between heat gain and heat loss, with a rise in body temperature and subsequent collapse.[5] This imbalance may be due to inadequate mechanisms for heat loss, excessive heat production, and high environmental temperatures—alone or in combination. The mechanism of the collapse, with loss of consciousness, delirium, and convulsions, is uncertain.

Hyperpyrexia causes cellular damage, the severity of which is related to its duration. Death may occur in the acute phase of heatstroke, without recovery of consciousness, or it may occur later, after the body temperature has been restored to normal and consciousness has been temporarily regained. The later deaths are the result of profound cellular damage and haemorrhage into the brain and elsewhere.

This sequence of events makes the principles of the prevention and treatment of heatstroke clear. The occurrence of heatstroke, like that of frostbite,[6] implies that preventive measures have been allowed to break down. Thus excessively hot or humid environments should be avoided, or adequate protection provided,[7] and energy expenditure should be limited. Heat loss from the body should be aided both by the choice of suitable clothing and by prior training of the thermoregulatory mechanism by acclimatization.[8] The subject who is to work in a hot environment should be fit for his job, free from disease, well hydrated, and without an alcoholic hangover.

If by misfortune heatstroke does occur the first aim of treatment is to restore the body temperature to normal as quickly as possible by cooling. Nevertheless, it should be remembered that vasoconstriction or shivering may prevent over-enthusiastic efforts. A most effective and easily available

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