of the original symptoms were reported to be negative. Although positive serological findings develop relatively late in pinta it was felt that this diagnosis need no longer be entertained. The improvement in one case following nearsphenamine was presumably a non-specific effect.

The world-wide distribution of this disorder, its involvement of different racial types, its independence of climate, and its invariable association with the use of mepacrine seem to leave no doubt of that drug's causative role. It also raises the question whether less typical cases may have escaped detection. Out of a profusion of ordinary tropical dermatoses only one other patient seen at the same time as the above group warrants brief description:

Sapper E., when admitted, displayed infiltrated erythematous plaques on the dorsum of both feet, on the extensor surface of his arms, on the back of the hands, ears, face, front and back of the neck, and a few in the axillae. He showed also some patchy loss of scalp hair. He had been taking mepacrine for eight months. The lesions had appeared ten weeks after he started taking the drug, and had advanced slowly thereafter. He had no symptoms. Shortly after his admission, at which time his case was tentatively diagnosed as subacute lupus erythematosus, a peculiar gross patchy hyperkeratosis of his palms appeared, with similar but smaller lesions on the feet. It was identical with that displayed in the typical tropical lichen-planus cases. He had no constitutional symptoms. His lesions gradually improved over the next two months, and he was then lost sight of.

Review of the history of Case 3 reveals a considerable resemblance to disseminated lupus erythematosus. Suggestive of this disease were the onset following unusual exposure to sunshine at a picnic—he was an orderly-room clerk and mostly kept indoors—the period of diarrhoea associated with an exacerbation of his rash several weeks after he had stopped taking mepacrine, and the persistently raised sedimentation rate. Against this, and supporting its classification as tropical lichen planus, were the relation to mepacrine, the complete absence of lesions of the face, and the extensive and severe involvement of the palmar and plantar regions.

Biopsy of a fresh macular lesion of the trunk in this case showed a non-specific histology resembling acute solar dermatitis. A hypertrophied scaly papule from the wrist appeared like typical lichen planus in most of its details. It showed hyperkeratosis, increase of the granular layer, acanthosis, liquefaction degeneration of the basal layer, and a diffuse infiltration of the upper cutis by lymphocytes and plasma cells merging with the basal cell layer. In the deeper part of the corium this infiltration was associated with vessels and hair follicles, and there were also some oedema and proliferation of the endothelium of the smaller vessels.

Summary

In a group of troops serving in scattered tropical areas, both malarious and non-malarious, tropical lichen planus was noted only in those who had taken mepacrine for malaria suppression. The incidence was approximately 1 per 2,000 troops. The initial symptoms appeared when mepacrine had been used for between two and three months. Some of the residual lesions seemed to be permanent. Some resemblance to disseminated lupus erythematosus is noted.

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POLIOMYELITIS: A SECOND ATTACK

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It is generally believed that second attacks of poliomyelitis are very uncommon, and this seems to be borne out by the small number of such instances reported in the literature. Quigley (1934) collected 12 cases, while Bridge *et al.* (1946) reported 6 among approximately 1,100 cases of poliomyelitis in and around Buffalo during the summer of 1944. In all some 37 cases have been reported in various American publications.

We feel, therefore, that it would be of interest to record the following case of a second attack which came to our notice recently. This was one of the 21 cases admitted to hospital in the Swansea area.

Clinical Summary

First Attack.—A girl aged 3½ was admitted to Hill House Isolation Hospital on Aug. 28, 1938, at the height of a poliomyelitis epidemic. The child had been generally out of sorts and listless for a few days, later becoming feverish, with occasional twitching of face and body. There was no history of obvious contact with infection, but her father worked in close association with a man whose child had been admitted to the same hospital on Aug. 19 suffering from poliomyelitis. On admission the child was pyrexial—temperature 102° F. (38.9° C.)—and showed left-sided facial paralysis and weakness of the abdominal muscles. C.S.F. findings were: colourless slightly granular fluid; 39 cells per c.mm. (all lymphocytes); globulin, faint trace; protein, 30 mg. per 100 ml.; glucose, 74 mg. per 100 ml.; chlorides, 702 mg. per 100 ml.; Lange curve, 0012210000. A facial hook was fitted and the child kept in bed for five weeks. She was discharged a week later with the hook still *in situ*. Subsequently she was seen at regular intervals by an orthopaedic specialist, and at the end of six months the facial muscles showed complete recovery of power and the facial hook was discontinued.

Second Attack.—During the current epidemic of poliomyelitis the child, now aged 12, was admitted to Hill House Hospital on Oct. 9, 1947, with a definite attack of the disease. She had had a cold a week previously, followed later by aches and pains "like 'flu'." On the day of admission she became feverish, developed a stiff neck, and had some difficulty in moving her right leg. Again there was no definite history of contact with infection, but a month previously two young children living opposite the patient had been admitted to hospital with poliomyelitis, and the parents of the two families had had frequent contact. On admission the temperature was 100.4° F. (38° C.); there was stiffness of the neck and spine, and a sluggish knee-jerk on the right side. Next day there was paresis of the right gluteus medius and quadriceps with loss of the knee-jerk on the same side and weakness of abdominals. C.S.F. findings were: clear fluid; 89 cells per c.mm. (90% lymphocytes); globulin, trace; protein, 100 mg. per 100 ml.; glucose, 80 mg. per 100 ml.; chlorides, 731 mg. per 100 ml.; Lange curve, ½12110000.

It will be noted that the following factors were common to both attacks: (1) A fairly typical history in the presence of an epidemic; (2) muscle paralysis and weakness; (3) changes in the C.S.F.—namely, increased cell count and raised protein content—which are consistent with a diagnosis of poliomyelitis. These, we feel, serve to demonstrate the true nature of the illness in both instances.

Discussion

The occurrence of a clinically recognizable second attack of poliomyelitis, in the ratio of one such case to a total of

only 21 cases, naturally raises a suspicion that immunity acquired after an attack of the disease may not be as complete and lasting as one is generally led to believe. This is the more significant in the light of modern teaching that latent and mild non-paralytic infections are exceedingly numerous during epidemic periods, and that the development of paralysis is an uncommon and unfortunate complication in a minority of cases. Mild atypical cases are unfortunately often missed; at best diagnosis can be confirmed only by expensive and impracticable animal-inoculation tests. In such circumstances it is very likely that atypical second attacks frequently go undetected. In an attempt to ascertain the degree and effectiveness of immunity following an attack of poliomyelitis, Bridge *et al.* (1946) investigated during an epidemic the incidence of illnesses regarded as mild poliomyelitic infections among 168 persons who had had the disease previously. There was definite evidence that 77 of the 168 had been further exposed, and 17% of those exposed revealed symptoms, such as fever, vomiting, muscle spasm, and transient weakness, suggestive of a mild form of the disease.

Whereas little is known of the antigenic structure of the viruses, there is definite evidence that different strains of the poliomyelitis virus exist. Rhodes (1947) describes the main members of the poliomyelitis group of viruses to be the monkey-pathogenic or simian strains, the Lansing, S.K., and M.M. strains, and Theiler's virus. It is possible that these strains are immunologically different and that each strain affords protection only against itself, despite the fact that the clinical syndrome may be the same in each case. Trask *et al.* (1937a and b) showed that strains isolated from various outbreaks in 1930 and 1931 were immunologically different, while Burnet and Macnamara (1929, 1931) found that monkeys which survived infection with one strain succumbed later to a different strain. Similarly, in 1937 Flexner showed that it was possible for monkeys to suffer two or even three attacks of poliomyelitis—an observation which had also been noted as early as 1913 by Kling and Levaditi.

Poliomyelitis occurs principally in infancy and childhood. Adults are not commonly attacked, presumably because of acquired immunity following latent or sub-clinical attacks. Nevertheless during the last war the incidence of poliomyelitis was much higher in troops stationed in the Middle East and India than in those in this country, one of the possible explanations being that the former were exposed to a strain against which they had no previous immunity (McAlpine, 1945). It is obvious, therefore, that we should be cautious about accepting the statement that one attack of poliomyelitis confers lifelong immunity, for if the immunological link in our chain of evidence is faulty then surely our measures for the prevention and control of the disease will be equally faulty.

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THE TETRALOGY OF FALLOT AND ITS SURGICAL TREATMENT

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The congenital heart lesion known by the popular name of "blue baby" was first introduced by the Englishman, Peacock, as the "blue maladie." Later on, in 1888, Fallot discussed the syndrome and provided the description which is characteristic of the condition as we know it to-day.

It was of interest to inquire into the incidence of the disease. White has stated that congenital heart disease constitutes between 2 and 3% of all cardiac lesions. The tetralogy of Fallot constitutes about 5% of the cases of congenital heart disease. If, however, a case of congenital heart disease has cyanosis, the probabilities are about 75% that the diagnosis is the tetralogy of Fallot.

The characteristic features of the syndrome described by Fallot are: (1) Stenosis of the pulmonary artery, (2) patent interventricular septum, (3) dextra position of the aorta, (4) hypertrophy of the right ventricle. Obviously there will be many variations from this particular set-up. In some the stenosis is greater than in others and the condition is apt to be associated with other congenital lesions.

Considering the possible causes of these defects occurring during the early development of the heart, Maude Abbott (1936) and Blalock and Taussig (1945) have suggested infectious disease of the mother during the first three months of pregnancy. German measles seems to be the commonest offender in this respect, but other conditions affecting the mother's health during this stage of development, when the cardiac chambers and such are being formed, may play an important part.

Signs and Symptoms

The clinical findings in the disease are as follows. (1) Cyanosis of the infant. It is usually present at birth, is continuous, and never disappears. (2) There is a strong systolic murmur that is heard best in the pulmonary area and may be heard fairly widely over the precordium and sometimes in the interscapular region. (3) Clubbing and marked cyanosis of the fingers and toes. (4) The most characteristic sign, apart from the cyanosis, is the limited tolerance of exercise. The patient has marked dyspnoea on the slightest exertion. The cyanosis increases and the child is able to walk only a short distance, when he becomes exhausted and squats down in a characteristic fashion.

Further investigation adds much to substantiate the diagnosis. To compensate for the lack of aeration of the blood there is a polycythaemia, which may be as high as 10,000,000 red cells per c.mm. or more. The haemoglobin is increased in proportion and may rise from normal up to 185 to 190%, and the volume of packed cells (haematocrit) up to 75%. The oxygen saturation of arterial blood is diminished, and in the worst cases it may be below 30%. In patients with a level such as this the exercise tolerance is greatly reduced. When the oxygen saturation is between 60 and 70% the exercise tolerance is improved, and above this level the clinical sign of cyanosis is not present.

The electrocardiogram shows right-axis deviation. X-ray examination of the heart shows enlargement. The characteristic enlargement is a projection of the left ventricle to the left with double apices, and an enlargement