The administration of calciferol in the form of high-potency ostelin tablets resulted in dramatic improvement of the skin lesions. The main improvement occurred during a three-weeks period of severe toxic effects unrelated to the level of serum calcium.

The relationship of sarcoidosis to tuberculosis is discussed. It may be that they are the same disease. The response of the skin lesions of sarcoidosis to treatment with calciferol resembles that of lupus vulgaris, this being another point in favour of a close relationship.

In view of the risks of "flaring up" a sarcoid lesion elsewhere and of causing sudden extension and ulceration of the skin lesions, calciferol should not be used in the treatment of sarcoidosis unless the skin lesions are extensive and disfiguring.

A delayed positive tuberculin reaction is described in sarcoidosis.

I am greatly indebted to Professor Charles Cameron for his advice and criticism and for the photographs taken in 1947. I am also indebted to the Department of Radiotherapy, Edinburgh Royal Infirmary, for the photograph taken in 1943. I wish to thank Dr. W. D. D. Small for permission to publish the case.

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ARSENICAL ENCEPHALOPATHY DURING TREATMENT OF TROPICAL EOSINOPHILIA

BY

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Prebble (1946) made an extensive survey of the literature on arsenical encephalopathy during treatment of syphilis and reported 187 cases of encephalopathy among Indian troops treated for syphilis as in-patients. He concluded that Indians are particularly susceptible to the condition.

He referred to a report by Glaser and Immermans on two non-fatal cases of encephalopathy which had received arsenic for conditions other than syphilis. Lees (1937) also observed this condition among cases of disseminated sclerosis and Hodgkin's disease after the administration of an arsphenamine product. Viswanathan (1947) reported the post-mortem appearances in a patient with tropical eosinophilia who developed encephalopathy after two 0.3-g. injections of neoarsphenamine. He, however, did not state whether syphilis was definitely excluded before treating the case as tropical eosinophilia. A further case of tropical eosinophilia in which encephalopathy developed after two 0.3-g. injections of neoarsphenamine and death occurred is described below.

Case Report

A Tamil mess cook aged 26 was admitted to 48 Indian General Hospital on Oct. 8, 1946, with a complaint of cough and breathlessness on exertion. The chest showed rhonchi all over it. A skiagram revealed changes associated with chronic bronchitis but no evidence of tuberculous infection. The total leucocytic count was 12,400 per c.mm., with 61% eosinophils—i.e., absolute eosinophilia of 7,564 per c.mm. The Wassermann reaction and Kahn test were negative. Tropical eosinophilia was diagnosed and 0.3 g. of neoarsphenamine was given on Oct. 24 and on Oct. 27. He complained of fever and shivering

after the second injection, and became dull and apathetic on Oct. 30. The temperature rose to 103.8° F. (39.9° C.); a blood film showed no malaria parasites. He suddenly became restless and began to have convulsions on Oct. 31; later he became delirious and finally coma supervened. His respirations were stertorous, eyes fixed, pupils dilated, and the reaction to light was sluggish. There was no neck rigidity. Abdominal reflexes were absent, deep reflexes were exaggerated, and plantar response was flexor. Blood pressure was 150/85, blood urea 60 mg. per 100 ml. Blood films showed no malaria parasites and urine contained no sugar or acetone. Coma deepened, and he died within eight hours of the onset of acute symptoms. Necropsy was performed ten hours after death. All organs, except the central nervous system, lungs, and liver, were within normal limits.

Macroscopic Appearances.—The pia-arachnoid all over the brain was congested and meningeal vessels were full. Cerebral grey matter was hyperaemic, and small blood vessels in the white matter of both hemispheres were congested. The choroid plexus was very engorged. No haemorrhages were detected on the surface of the brain or on section. The pleural cavity contained no free fluid or adhesions. The lining membrane of the right and left bronchi was congested. Bronchioles were slightly dilated; their mucous membrane was congested, and they contained mucopurulent secretion. The lungs showed no evidence of bronchopneumonia on palpation or on section. The liver was normal in size, and the cut surface revealed no change.

Microscopical Appearances.—Brain:—Examination revealed lymphocytic infiltration of the perivascular spaces and of the substance of the brain tissue in the vicinity of the capillaries. The capillary endothelium was swollen. Liver cells showed fatty change and foci of necrosis infiltrated by lymphocytes. I have no record of the histopathology of the lung. Cerebrospinal fluid taken during necropsy showed the following findings: total cell count, 280 cells per c.mm.; type of cells, 95% mononuclears, consisting of lymphocytes, plasma cells, and endothelial cells; stained deposit, no organisms; protein, 180 mg. per 100 ml.; Nonne-Apelt reaction, positive 1; Wassermann reaction, one volume of a 1 in 5 dilution showed partial inhibition of lysis; with one volume of a 1 in 2.5 dilution, one volume of undiluted fluid, and two volumes of undiluted fluid lysis of cells was inhibited. The Wassermann test was carried out by Wyler's modification.

Comment

This case was diagnosed clinically as arsenical encephalopathy. Post-mortem findings did not show haemorrhages or demyelination—important characteristic features of arsenical encephalopathy according to earlier writers. Prebble (1946), however, reported that these features were observed in only one out of 40 patients whose brains were closely studied by Krainer at the Central Military Pathological Laboratory, Poona. Microscopical appearances of the brain tissue in my case were similar to those described by him in some of his cases. The disease process in the liver was not so advanced that it could have caused death, but it was very probable that the damaged liver cells delayed the metabolism of arsenic as suggested by Friedman and Shinefeld (1941).

During life the patient gave no history of syphilis or of lapsed treatment. He had been attached to the hospital as a mess cook for three years and had not sought admission to a special treatment centre for venereal diseases during that time; nor did he show any signs or symptoms of meningeal, cardiovascular, or parenchymatous syphilis during life. Microscopy of the brain tissue also did not reveal any evidence of syphilis. Lymphocytic infiltration of the perivascular spaces has been regarded as a general form of central nervous system reaction and is not indicative of any one pathological condition. The blood Wassermann reaction and Kahn test were negative four days before he was put on neoarsphenamine. Cerebrospinal fluid collected during necropsy, however, gave a positive

Wassermann reaction. Technical error was excluded by repeating the test on the same fluid, and cross-checking was afforded by three other tests on the fluid—namely, protein, total number of cells, and globulin. All three were increased. Absence of evidence of syphilis during life and on necropsy raised a question whether the positive Wassermann reaction was a biological false positive result. This is very probable, but could not be definitely decided, as spinal fluid was not examined during life.

Reynolds (personal communication) states that of the many conditions in which the biological false positive reactions have been reported in the spinal fluids encephalomalacia is possibly the only one which suggests that post-mortem changes can cause a false positive reaction in cerebrospinal fluid. Stokes *et al.* (1944) mentioned cadaver blood as one of the conditions giving false positive serological reactions. Thomson (1947) quoted Bertolozzi, who found positive reactions in sera of a large percentage of 50 corpses whose blood he examined, but Kolmer (1928) stated that blood collected before bacterial contamination takes place would not give false positive reactions. I could not find any reference in the literature to the false positive reaction in cerebrospinal fluid collected after death. Four other cerebrospinal fluids that were collected after death and examined by me two to three days later showed a trace of blood and were found to be anticomplementary. It is hoped, however, that those who have facilities will report on the incidence of false positive reactions in cerebrospinal fluid collected after death.

Summary

A case of arsenical encephalopathy in an individual in whom no evidence of syphilis could be established during life or on necropsy has been described.

Absence of evidence of syphilis suggested that the positive Wassermann reaction of the cerebrospinal fluid collected ten hours after death was a biological false positive.

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The Minister of Food has appointed a working party to make recommendations about precautions which are considered practicable and desirable for securing the observance of sanitary and cleanly conditions in the catering trade. Sir William Savage, consultant to the County Medical Officer of Health for Somerset, has accepted the Minister's invitation to act as chairman. The other members are: Mr. H. E. Burdett (Fullers, Ltd.); Mr. P. N. R. Butcher (Ministry of Health); Mr. H. Crane (National Union of General and Municipal Workers); Mr. W. C. Crozier (catering manager, Scottish C.W.S.); Mr. N. R. C. Dockeray (Ministry of Food); Mr. P. T. Grove (Permanent Vice-president of the Health and Pleasure Resorts); Mr. E. Hartley Smith (hotel proprietor); Mr. J. Hollingshead (Director of Catering Division of British Tourist and Holidays Board); Mr. R. Hood (Richard Davis, Ltd.); Mr. G. M. B. Hutcheson (Director of Hotels Division of British Tourist and Holidays Board); Brigadier S. O. Jones (Director of Home Holidays Division of British Tourist and Holidays Board); Dr. W. A. Lethem (Ministry of Food); Mr. E. Vivian Rogers (Wm. Hancock and Co., Ltd.); Mr. A. W. Ritchie (Chief Sanitary Inspector of the City of Edinburgh); Dr. A. J. Shinnie (Medical Officer of Health for the City of Westminster); Mr. L. W. Slark (John Gardiner (London), Ltd.); Dr. E. L. Sturdee (Ministry of Health); Dr. I. N. Sutherland (Department of Health for Scotland); Dr. R. Sutherland (Medical Adviser and Secretary to the Central Council for Health Education). The working party held its first meeting on Nov. 26.

AORTIC COARCTATION WITH PATENT DUCTUS ARTERIOSUS

BY

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Coarctation of the aorta and patent ductus arteriosus are now recognized as defects which are amenable to surgical treatment, but the opportunity seldom arises of treating them simultaneously. The following case is reported because of its rarity, the development of characteristic signs at an early age, and the electrocardiographic findings.

Case Report

The patient had been treated for pink disease at the age of 2 years 4 months. The presence of a systolic murmur was noted and the diagnosis of congenital heart disease made, but the precise defect was not determined. When seen on Nov. 27, 1947, at the age of 7, she presented typical signs of aortic coarctation. She had always been a quiet child and disinclined to take much exercise, but cyanosis had not been observed. Her nutrition was poor and she was undersized for her age.

A systolic murmur of a rumbling character was audible at the apex and a soft systolic murmur was heard along the left border of the heart and to a less degree over the praecordium. A more pronounced systolic murmur could be heard over the whole of the posterior thorax but was maximal over the scapulae. The blood pressure in both arms was 140/70. The femoral arteries were imperceptible and blood-pressure readings could not be obtained. In the stooping posture arterial pulsation was visible and palpable medial to the vertebral border of the left scapula. The electrocardiogram showed right axis deviation (Fig. 1). X-ray films of the chest revealed

FIG. 1.—Electrocardiogram taken on Nov. 27, 1947.

notching of the ribs and a double shadow in the region of the aortic knuckle. The possibility of a patent ductus was anticipated because of the right axis deviation (Gilchrist, 1947), although there was no systolo-diastolic murmur.

Operation, Nov. 28, 1947

Cyclopropane and oxygen were given through an endotracheal tube and closely fitting face-piece connected with a rebreathing system incorporating the Waters absorber. With the patient in the right dorso-lateral position a curved incision was made below the left breast from the edge of the sternum opposite the third costal cartilage to the posterior axillary line. A flap of skin and pectoral muscles was reflected up and the chest opened through the third interspace, with section of the