On the eleventh day after admission the fits ceased, but the child was now very weak and continued to vomit occasionally. By careful feeding the general condition improved and the infant gained weight, reaching 5 lb. 10 oz., but on the twenty-first day after admission continuous fits occurred for two hours, resulting in a loss of 10 oz. in the following week. During the quiet period the stools had been normal, but after the second series of fits they became green and offensive again. Following this the fits and vomiting ceased completely, and after an interval of a week weight was gained rapidly. Repeated examination failed to reveal any pathological condition. It was noticed at this time that the overmoulding of the skull bones was not quite so marked as at first, and a very small anterior fontanelle could be felt. Also the child made no attempt to grip anything, and paid no attention to the feeding-bottle. Examination revealed that it was blind.

Commentary

No muscular paresis or cutaneous sensory loss had become evident throughout the course of the illness, and in view of the nature and frequency of the fits I came to the conclusion that they were directly due to the persistent overmoulding of the skull bones causing pressure on the brain when physiological increase of the cerebrospinal fluid occurred. The points on which I base my diagnosis are that the fits were of an unusual nature and that they appeared to be due to a depression of vital centres in the brain rather than to irritation or reaction caused by toxic products of any pathological condition.

Although the stools were green during the first series of fits and a gastro-intestinal condition might be inferred from this, there was no rise in the temperature and none of the usual signs of toxæmia. Also the stools were normal between the eleventh and the twenty-first day of admission, and only became green again after the short second series of fits, being an effect and not a cause of the fits. The pulse rate did not rise above normal. The fits were not of an epileptiform nature, and the family history was devoid of any stigmata.

The provisional diagnosis of slight cerebral hæmorrhage due to overmoulding and possible rupture of the tentorium cerebelli was untenable in view of the continuous fits combined with the non-appearance of paresis. Also the blindness points to general cerebral pressure rather than to local irritation caused by hæmorrhage.

It is reasonable to assume that after feeds or during handling of an infant there is a physiological increase of cerebrospinal fluid pressure, and since the fontanelles are obliterated, the tentorium cerebelli stretched taut, and the anterior and posterior fossae encroached on by the persistent overmoulding, any increase of pressure would exert its full effect on the brain, causing vomiting. In addition the medulla oblongata would be lower than normal, extending approximately to the foramen magnum, and the increase of pressure would tend to force it further down, causing pressure on the vital circulatory, respiratory, and heat-regulating centres in the medulla. Lumbar and cisternal puncture would thus be contraindicated. Exception might be taken to this view on the grounds that such dislocation of the medulla does not occur in hydrocephalus, but in that condition the fontanelles and sutures are open, and any rise in pressure would be taken up by the lax tissues until the skull bones adjusted themselves to the increased calibre of the brain.

With regard to the late appearance of the fits, it is probable that the rather precipitate birth and consequent sudden overmoulding of the skull bones caused a severe degree of shock, resulting in a lowered blood pressure and decreased secretion of cerebrospinal fluid, the fits

only appearing when the child began to recover and normal function was restored.

Rectal salines would seem to be contraindicated, but starvation did not allay the fits, and it became obvious that their occurrence after feeding had to be accepted or the child would die from lack of nourishment.

The blindness is probably due to optic neuritis following on the intermittent pressure. Light reflex and accommodation are normal, and there is no paralysis of ocular muscles.

There has been no recurrence of fits or vomiting; the child weighs 7 lb. 14 oz., and cries lustily on occasion. It has the full use of its limbs, with no apparent cutaneous sensory loss. The anterior fontanelle is now about half an inch wide; the posterior fontanelle is non-existent, and the moulding much less than on admission. The blindness is improving.

The x-ray plate shows the overlapping bones, the photographs being taken when the child was 3 months old.

CHRONIC DISSEMINATED TUBERCULOSIS ILLUSTRATED BY A CASE OF CORTICO- PLEURAL DISEASE

BY

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(WITH SPECIAL PLATE)

The primary complex resulting from the first infection with the tubercle bacillus and the established stage of phthisis are clearly defined and familiar conditions. So are some of the results of hæmic dissemination such as acute miliary generalization, meningitis, and tuberculous disease of the bones. Chronic hæmatogenous tuberculosis of the lungs is not so well known, but it deserves particular attention, for there are good reasons for thinking that it may be the precursor of other important conditions. Either it may for a time remain abortive, and after recrudescence and local invasion of the bronchi precede and produce ordinary (bronchogenic) phthisis, or more directly and within a shorter time it may develop into a chronic pulmonary condition superficially resembling ordinary phthisis but having its own characteristic features. Hence chronic hæmatogenous disease of the lung may present itself in a variety of ways, which Pagel (1933) has classified and described in detail. These generalized forms of tuberculosis, which include the so-called chronic miliary tuberculosis, show distinctive features in the lungs, which Sayé (1937) thus summarizes: "There is constant involvement of the lungs, in which the lesions assume both as regards situation and stage of development a symmetrical lobar distribution. . . . One finds fibrous miliary lesions and hyperplasia of the interstitial tissue, and marginal distribution, diffuse interstitial emphysema, caseous pleurisy, and a special type of cavity—'dry, not surrounded by dense lung tissue, round thin-walled (Lochkaverne).' These cases nearly always die of meningitis or acute miliary tuberculosis." The determining cause of death is therefore the last overwhelming discharge into the blood stream of bacilli which have most probably

been previously distributed in smaller doses and on rare occasions: thus in the series of post-mortem reports of twenty-one cases studied by Hoyle and Vaizey (1937) tuberculous meningitis was reported in nine. The case to be described belongs to this group, showing disseminated miliary lesions which in the lungs were distributed in the periphery and along the fissures—an example of the form first described by Pagel as cortico-pleural chronic miliary tuberculosis (op. cit., p. 282), and recently studied by Stefko (1936).

Case History

A male clerk, aged 45, was admitted to the sanatorium on July 27, 1937, obviously very ill, with typical signs of meningitis, and died two days later. In addition to the signs of meningeal disease a pleural rub was heard on the left side of the chest, where the percussion note was impaired. No tubercle bacilli were found in the sputum.

History.—The patient's sister said that he had been in good health until March, 1937, when he had "sciatica," but was well again in April. Early in June he complained of headache, shivering, and severe vomiting, with night sweats, and slowly became worse till admission. A radiograph taken on June 30 showed shadows typical of miliary tuberculosis and obliteration of the left costo-diaphragmatic angle.

Necropsy.—There was moderate wasting. Examination of the brain revealed an excess of cerebrospinal fluid, with congestion of vessels and miliary tubercles in the region of the middle cerebral artery. The left pleura was reddened with vascular injection, and between the parietal and visceral layers were universal fine adhesions easily separable by the hand. Section of the lungs revealed miliary tubercles, but with a special distribution. These tubercles were confined to the periphery of the lungs to a depth of about an inch, and lining the pulmonary fissures in a similar manner. Equally striking were the numerous tiny interstitial emphysematous bullae; they were of varying size, but the largest appeared as a small cavity about half an inch in diameter with smooth glistening walls. The kidneys contained a few scattered miliary tubercles, but the spleen, though softened and enlarged, showed none to naked-eye inspection. A stony calcified mass an inch in diameter was found in the mesentery.

Histological Examination.—The nodules were found to consist of small caseous centres with extensive peripheral proliferation of epithelioid cells, lymphocytes, and fibrocytes. In addition there were areas showing exclusively proliferative reaction in the form of small epithelioid cell nodules surrounded by "collapse induration"—that is, collapsed alveoli with fibrotic interstitial tissue. Histological examination therefore showed that this was certainly *not* an acute process, but one with a definite tendency towards healing or a chronic course at every individual focus (see Plate).

Commentary

Although the clinical history available and the radiograph seemed to indicate that acute generalized miliary tuberculosis would be demonstrated at necropsy, the actual findings suggest a chronic course of the pulmonary lesions, both from the restriction of the miliary foci to the cortico-pleural area and from their macroscopic and microscopical appearance.

One may ask if the pleura exercises a localizing influence on blood-borne tuberculous lesions, and the frequent involvement of the pleura, in the form of idiopathic pleurisy, supports this suggestion. Another explanation, recently upheld by Stefko, is that the pleural lymphatics may be the path of spread, but a closer connexion of the pulmonary nodules and the pleural lesions than was found in the present case ought then to result. Again, experimental work by one of us has produced a chronic haematogenous lesion of the cortico-pleural area

in rabbits by intravenous injection of virulent bacilli after previous immunization of the animals by infection with living avirulent or killed bacilli; the final distribution of the lesions in these animals was the result of absorption of the more central lesions (Pagel, 1936).

It may also be noted that a radiograph which shows a fine mottling in all fields may be a shadow picture of purely cortical lesions, and in such case a tomographic film might give better diagnostic help: evidence of former pleurisy would stress the need for this extra investigation, since its presence may be an indication of the chronic nature of the miliary disease.

Summary

A case of cortico-pleural chronic miliary tuberculosis is described, with the post-mortem examination. The relation of this condition to other forms of pulmonary tuberculosis is discussed.

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FRACTURE OF THE NECK OF THE FEMUR ANOTHER METHOD OF INSERTING THE SMITH-PETERSEN PIN

BY

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(WITH SPECIAL PLATE)

The insertion of a Smith-Petersen pin has now become the accepted method of treating fractures of the neck of the femur; but the best means of inserting it is still on trial. Every surgeon who has to deal with these fractures has evolved his own technique, and the respective advantages of the "open" and "blind" methods have yet to be established. Advocates of both methods claim superiority for the one or the other practice, and until experience has proved either of them to be of outstanding merit for the majority of cases it will continue to be of interest to hear of any method which has been consistently successful in any one surgeon's hands. This being so, I venture to describe a method I have evolved which, although "open," may be described as a compromise between the "open" and the "blind" operations, in that it does not expose the fractured ends, which are still protected by the strong anterior ligament of the hip-joint.

The Technique

For my purpose I have designed a director—made for me by the Medical Supply Association—which when placed against the outer surface of the femur at the correct level gives the angle for the insertion of the pin in relation to the long axis of the bone. The lateral plane is gauged by setting the director-edge parallel to a straight instrument laid along the flat front surface of the neck. This will be made clear by reference to the Special Plate.

F. A. H. SIMMONDS AND W. PAGEL: CHRONIC DISSEMINATED TUBERCULOSIS

FIG. 1.—Miliary tubercles in cortical areas and along interlobar fissure.

FIG. 2.—Miliary tubercles in cortical zone: small emphysematous bullae: pleural thickening and adhesions.

G. C. GORDON: PERSISTENT OVERMOULDING OF THE SKULL BONES CAUSING FITS IN AN INFANT

Radiographs showing the overlapping bones at age of 3 months.

J. M. TURNER: UNUSUAL CASE OF CHONDRIFICATION OF PATELLAR LIGAMENT

FIG. 1.—Photograph of the cartilaginous mass excised.

FIG. 2.—Lateral view of left knee-joint. Note (a) cartilaginous mass in the patellar ligament; (b) upward displacement of the patella.