a few cases. The tongue was nearly always coated at the dorsum and red at the margins. Tonsillitis and pharyngitis were fairly common. No parotitis was met with. Nausea and vomiting were usual at the commencement, but rarely persisted throughout the disease. Constipation was the rule, and was followed by diarrhoea at the terminal crisis. Dysenteric symptoms were present in a few cases throughout the malady. No changes were observed in liver or spleen that could be attributed to dengue.

Urinary System.—Albuminuria was common but transient. One case of acute nephritis was reported. Chronic kidney disease is likely to end by uraemia as the result of an attack of dengue. Diabetic patients when attacked sometimes developed a fatal coma, even during convalescence. Epididymitis, orchitis, cystitis, etc., were not met with.

Complications.—These were rare. Those met with were: pharyngitis, rhinitis, gastritis, enteritis, nephritis, and thrombosis.

Sequelae.—Itching of body, irritation of palms and soles, mental depression, vertigo, motor paresis, peripheral neuritis, staccato speech.

Children were just as liable to be attacked by dengue as adults. Males and females were affected equally. No deaths occurred among children.

Convalescence may be short or long—one day to three months. Patients lose weight, and usually suffer from mental depression, debility, loss of appetite, bradycardia, migraine, and neuritis. Relapses are rare.

Diagnosis.

In this season (December) in Egypt dengue has to be diagnosed from the following diseases:

(a) Malaria: by examination of blood films, size of spleen and liver, time incidence of febrile attacks, and rapid pulse.

(b) Measles: starts less suddenly; presence of Koplik's spots, coryza, absence of pains, rapid pulse, and deep red rash.

(c) Influenza: is usually accompanied by respiratory involvement, rapid pulse, and absence of rash.

(d) Scarlet fever: diagnosed by presence of throat symptoms, rash, rapid pulse, absence of pains, and blood examination.

(e) Small-pox: in the first three days of dengue it is very difficult to differentiate it from small-pox. Later on the course of the malady soon manifests its nature. History of vaccination may help.

(f) Articular rheumatism: diagnosed by history of malady, presence of acid sweats, and joint lesions.

The treatment is symptomatic and prophylaxis involves antimosquito measures.

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DUPLICATION OF THE SPINAL CORD.

BY

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AN excellent review of the known cases of partial doubling of the spinal cord was written in 1906 by A. Bruce, Stuart McDonald, and J. H. H. Pirie¹; for the reference to it I am indebted to Dr. Kinnier Wilson. Nevertheless, the following case perhaps deserves a note.

The patient, a well-developed man, aged 51, was admitted to hospital on March 29th, 1928, with a spastic parietic ataxic gait and signs suggesting combined degeneration of the spinal cord. He had a pale, sallow face; the liver was considerably enlarged, and the spleen could be felt, reaching just below the ribs. The gastric contents after a test breakfast contained no free hydrochloric acid. The erythrocyte count was 3,392,000 and the haemoglobin was 70 per cent. The white cells were 17,800 (polymorphonuclears 74 per cent., lymphocytes 17 per cent., monocytes 9 per cent.). The Wassermann reaction was negative. There was a little irregular pyrexia, and apparently a remnant of pneumonia in the middle part of the right lung, which possibly explained the presence of a polymorphonuclear leucocytosis. According to the history obtained the patient had noticed something wrong with his feet in August, 1927, and since December, 1927, he had been taking an "ox-liver juice" twice daily for anaemia. The result of the ox-liver treatment was said to have been good, till weakness in his legs developed ten days before admission.

In the hospital, in spite of liver diet, etc., the weakness in the legs increased, paradoxical incontinence of urine and cystitis, with bedsores, developed, and death occurred on April 26th.

The necropsy and microscopical examination confirmed the diagnosis of combined degeneration of the spinal cord; but during the macroscopical examination it was noticed that, close along the left side of the lower part of the spinal cord, there was another cord-like body of the same consistence as the cord, but smaller (about 1 cm. in diameter) and without nerve roots proceeding from it. Its upper end arose from the substance of the cord by a base somewhat over 1 cm. in diameter, the uppermost part of its insertion being $7\frac{1}{2}$ cm. above the terminal point of the conus terminalis. At its lower (free) end it tapered to a point, which was $1\frac{1}{2}$ cm. above the lower end of the conus terminalis.

Transverse microscopical sections of this macroscopically tumour-like appendage of the spinal cord showed it to be quite obviously a fairly well-developed duplicate cord with a central canal, etc. This was kindly confirmed by Dr. J. G. Greenfield, who also confirmed the fact that the duplicate cord showed well-marked signs of combined degeneration, just as did the main spinal cord (from which the cauda equina arose).

When I saw the long polypoid tumour-like appendage lying along the left side of the lower portion of the spinal cord, I at first thought that I had made a mistake in the diagnosis, and that the patient's paraplegia, etc., might have been due to a tumour; but microscopical examination, as already stated, completely cleared up the nature of the tumour-like structure, proving it to be a congenital abnormality, a doubling of the lower part of the spinal cord. One may wonder whether in this case lipiodol would have been partially arrested at the site of origin of the duplicate cord had it been used to ascertain the site of a possible tumour.

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THE ETIOLOGY OF MONGOLISM;

WITH A CASE OF MONGOL TWIN.

BY

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THE occurrence recently, in my hospital practice, of a case of twins, one of whom was a Mongolian imbecile, the other not, stimulated investigation into the etiology of that distressing and increasingly common type of mental deficiency. But the *ultimate* cause of Mongolism is, I suppose, as yet unknown, nor can I find much to elucidate it.

The peculiar history so often obtained—namely, that the child is either the first of a very young mother, or, as often happens, is born many years after the previous one, and most frequently of a mother nearing the menopause—warrants the assumption that this developmental error is in some way due to immaturity or exhaustion of her procreative powers.¹ At what stage in the development of the ovum or foetus this makes itself felt has been the subject of a good deal of argument.

The problem is this: Is Mongolism inherent in the unfertilized ovum, or is it acquired after fertilization? A third proposition might be that Mongolism is imparted to the ovum in fertilization, were it not that the history appears to point to the maternal factor so constantly. As a generality it may be assumed that parents are approximately the same age, the father being on the average a year or two the elder. This would make the odds even as between the two parents, were it not that there is no evidence, known to me, that the children of elderly men tend to Mongolism otherwise than when their wives are approaching the menopause. Nevertheless, this possible factor cannot be entirely brushed aside without consideration.

An argument in favour of the post-fertilization embryonic theory is that Mongols are so often the subjects of other congenital abnormalities. Of these the commonest are malformations of the heart, followed a long way behind by congenital cataract—defects which obviously cannot

occur till embryonic development has proceeded far enough to start the formation of the organs implicated; and the heart, for example, is perfectly formed, though in miniature, by the end of the seventh week after conception.² By analogy it is argued that similarly the Mongolian characteristics (which are in effect congenital malformations) take their origin in the embryonic³ period of the first six weeks of intrauterine life. This may be countered by the argument that an ovum already the subject of an inherent defect (of a nature which is wonderfully true to type) is more likely to go wrong in other directions than one which is not.

David M. Greig, in a recent long and erudite article,⁴ favours a theory that Mongolism is a defect of foetal growth, though he concludes that this must have started in the embryonic period mentioned already. He bases on his minute and fully recorded observations on three Mongolian skulls the deduction that Mongolism "seems rather to be a defect in growth (foetal) than a defect in development (embryonic)." He states that "the main defect in the skull is want of growth of the facial bones, chiefly the maxillae and of the alveolar processes anteriorly," with various consequences to the adjacent bones, into which I need not enter. As an interesting aside, however, it may be mentioned that although obliquity of the palpebral fissures and nearness of the eyes are characteristic of the Mongolian facies, there was no obliquity of the superciliary margins in these skulls, nor any undue approximation of the bony orbits. With regard to the reported arrest of development of the skull base he goes on to say: "Deficiency of the anterior wall of the acoustic meatus, imperfect closure of the foramen ovale and foramen spinosum, flatness of the occipital condyles, and in the youngest skull an indication of the component parts of the occipital bone, are evidences of delayed development, but are not features which have interfered with the growth or size of the skull-base. If any arrest of development has occurred it is in the brain, not in the bones . . ."; and later, "Before the age of 16 the growth of the Mongol's brain has ceased." He states as his final opinion that "even from the skull alone there seems evidence that development normally begun has mapped out all structures and features in the embryo, but has failed to lead them to perfection during foetal growth." Pointing out that the first appearance of ossification in the skull appears in the maxillae on the thirty-ninth day, followed by the premaxilla three days later, and the nasal bones on the fifty-seventh day, he says: "Obviously it is during this period that the departure from normal growth shows itself, and this failure in the bones is accompanied or followed by defective growth elsewhere, notably in the nervous system."

For myself, though open to conviction, I believe the maternal influence, which appears to be almost undeniable, and whatever it may be, is exercised upon the ovum during its maturation, and therefore before fertilization has followed and segmentation started.

The chief argument in favour of this theory is the occasional occurrence of twins one of which is Mongol and the other not. This proves beyond a doubt that no maternal influence is present after embryonic development has begun; or that if there be any such influence it is selective in a manner highly improbable, though I recognize this argument is a two-edged sword. It has no bearing on the question of responsibility as between the parents, for obviously if one ovum can be inherently Mongol and the other not so, the same should hold good of the male elements. Known cases of twins both of which were Mongols are so rare—only some three in number—and their original records so inaccessible that although they were of the same sex complete proof that they resulted from uniovular pregnancies is wanting, but the assumption that it was so is warranted. There are also three cases on record where the twins were of the same sex, one being Mongol; but there is equally no proof that these were uniovular pregnancies. But on no occasion has it yet been recorded that twins of different sex were both Mongols, which is in accordance with the mathematical probabilities under this theory. Though the theory does not prohibit the possibility of binovular twins being Mongols it must be remembered that, apart from the six twin cases above

mentioned, and admitting the fact that the Mongol is often the last pregnancy, more than one Mongol in a family is almost, if not quite, unknown. But we are still as far as ever from solving the problem of the malign influence dooming the ovum to Mongolism, and the corollary of prevention.

In January, 1923, Halbertsma of Haarlem⁵ collected fifteen cases of twin pregnancies of which one twin was Mongol and the other normal. Occasional additions to this list have since been made, and to these I am able to add another instance.

On December 23rd, 1926, C. F., aged 4 months, was brought to the Royal Liverpool Children's Hospital because "he was not coming on." He was one of twins, the other being a girl, who, at my request, was brought on a subsequent occasion for comparison, and proved to be a normal and healthy child, though small for her age. The boy had the typical Mongolian cranial attributes and facial appearance, with the other characteristics of the Mongolian imbecile, including the loud bruit and the cyanosis of a congenital heart lesion. This child further complied with the frequent Mongolian history by dying of measles, contracted when he was 14 months old. The sister recovered.

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A CASE OF PRIMARY ACTINOMYCOSIS OF THE LUNG.

BY

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OWING to the fact that patients admitted to tuberculosis sanatoriums have mostly been diagnosed before admission one is apt to be biased in favour of tuberculous infection and to take for granted that the diagnosis is correct, instead of approaching the case with an open mind. During the course of the illness of a certain patient one or two complications ensued which raised some doubt as to the diagnosis of pulmonary tuberculosis being correct; hence I have thought it worth while to publish an account of the case.

Previous History.—Up to the age of 18 the patient had lived in South Africa, where, after leaving school, he was an apprentice in a lithography works. He came to England for a few months' holiday, and then went to Australia with a friend to search for gold. After a few months of unsuccessful prospecting they both worked on a ranch, sheep farming, for about eighteen months or two years. The patient then joined the army soon after the outbreak of war. He was sent to England and thence to France, where he was "gassed" in March, 1918; his war service amounted in all to three and three-quarter years when he was demobilized. He then joined a Government tug boat and remained at this work for four years. He returned to England, and after three months' holiday worked as a bricklayer for five months, later returning to his original work as a lithographer. His health gradually deteriorated, but he remained at work until June, 1927, when he complained of acute pain in the left side, anorexia, and loss of weight. He was first seen by a tuberculosis officer in October of that year, who, after having carefully watched the temperature (99.2°–102°) for a few days diagnosed the case as anaemia and pleurisy with effusion, and recommended sanatorium treatment. The sputum had been examined a month previously, with a negative result. There was no significant family history and no previous illness of importance.

Condition on Admission.—The patient was admitted to the Hertfordshire County Sanatorium on October 24th, 1927. He gave his age as 35, was very anaemic, and looked exceedingly ill. He had lost about 2½ st. in weight, and had not had a good night's rest for a fortnight. His temperature was 100° F., pulse 118, and respirations 24. He complained of pain extending from the right shoulder down the outer side of the arm as far as the elbow, but since no definite lesion could be found to cause the pain, it was attributed to neuritis. On examination of the chest the left side was found to be dull on percussion, the dullness being most marked at the left base posteriorly. There was also slight impairment of the note at the right apex. There were moist crepitations throughout the left side, and in addition diminished breath sounds at the base behind with amphoric breathing—signs compatible with pleurisy with a small effusion. His liver was palpable and slightly tender; the abdomen otherwise revealed no abnormalities. The urine contained no albumin or sugar; the specific gravity was 1020 and the reaction acid.

Subsequent History.—The pain down his right arm was treated with methyl salicylate and a neuralgia mixture was given. Nephenthe 20m was ordered, and the next day he appeared to