

intussusception, but I had to confess that it might have been occasioned by an enlarged and inflamed gland, or even by a faecal accretion. Rectal examination proved negative. I expressed my suspicions but did not meet with much credence, the more so as another member of the family, a boy, aged three years, had also that night passed a blood-stained motion. Now, the firmest adherent to the doctrine of the necessity for suspecting the existence of an intussusception in every unexplained abdominal derangement cannot conscientiously assert that this lesion occurs in an epidemic form in families. I was reluctantly compelled, therefore, tentatively to acquiesce in a diagnosis of dysentery. This I did the more readily in view of the fact that the treatment adopted—frequent doses of sodium sulphate by the mouth, together with high rectal injections of astringent solutions—was not directly inimical to either condition. Two days later, on Nov. 7th, I again saw the patient with Mr. Eddowes. No change had yet occurred in the frequency and character of the motions but the child was distinctly losing ground. There was no vomiting now or at any other stage of the illness. On abdominal examination I was unable to discover the mass which had been present at my first visit, but Mr. Eddowes informed me that he had distinctly detected it a few hours before my arrival. Now this alternating presence and absence of the tumour, accompanied by frequent bloody and mucous stools of an otherwise not decidedly unhealthy texture, is so characteristic of the condition that I with more firmness declared that in my opinion the case was one of subacute incomplete intussusception for which an immediate exploratory incision was indicated. Once more my dictum was rudely shaken by the discovery that the elder child, before mentioned, had also that morning voided a blood-stained motion. On the previous occasion his stool had been pale, loose, and distinctly blood-stained, leading one to fear that he might be at the beginning of an attack of dysentery; this time the motion was healthy and well-formed, containing only a streak of blood such as is not uncommonly produced in children by a rectal polypus. I performed a rectal examination with a view to discovering a growth of this character but the result was negative. I next suggested that the original patient, the baby, should be examined in a similar quest. A whiff of chloroform was consequently administered and a careful rectal examination undertaken, both digitally and by inspection through a speculum; the result was *nil*. Abdominal palpation also proved negative. On Nov. 9th Mr. Eddowes telephoned to me to the effect that the child was slowly sinking. On ascertaining that the elder boy was perfectly well and had passed no further blood in his stools I once more declared my views as to the existence of an incomplete intussusception. This time I decisively asserted my opinion, insisting that in immediate operative interference lay the only possible hope of saving the life of the patient.

On the following morning, the ninth day of the illness, Dr. G. A. E. Murray of Johannesburg was summoned to perform a laparotomy, which I witnessed. On opening the abdomen in the middle line coils of distended small intestine at once protruded. On tracing the gut downwards it was found to grow narrowed and its walls were seen to be discoloured, the congestion becoming more intense until, about the upper third of the ileum, a very small enteric involution was met with. The intussusception was readily reducible and the mesentery at this point was then anchored by a stitch or two to the peritoneum of the anterior abdominal wall. The whole operation occupied not more than some 20 minutes, the greatest haste being necessary in view of the extremely collapsed condition of the patient. Hot bottles, stimulants, and subcutaneous injections of normal saline solution were energetically employed, but without avail, for the child never rallied and death supervened two hours later.

*Remarks.*—This case appears to me to present many features of unusual interest which seem to render it worthy of the detailed description I have given of its course. In the first place, there is the fact of the comparative rarity of these cases of incomplete intussusception in which the occlusion of the bowel is incomplete and the passage of faeces continues unimpeded, whilst there is little or no vomiting. These cases may last for weeks or become chronic and exist for months or years, death in most instances being due to exhaustion. Secondly, there was the considerable amount of hæmorrhage from the bowel. Usually, in subacute or chronic intussusception the catarrhal changes at the neck of

the involution are comparatively slight and the stools are characterised only by small hæmorrhages. Thirdly, there was the similarity to a case of dysentery—superficial, it is true, yet sufficiently exact to obscure the diagnosis. In a paper on chronic intussusception published in the *Transvaal Medical Journal* of November, 1905, I drew attention to the frequency with which diagnostic difficulties of this character arise. Fourthly, there were the disappearance and reappearance at intervals of the abdominal tumour. According to Treves, a tumour is felt on abdominal examination in only 50 per cent. and rectally in only 32 per cent. of these cases, and in the paper already quoted I have drawn attention to the recognised fact that the tumour, once it has been felt, may disappear from time to time. For this reason it is essential that the examination of the abdomen should be undertaken both thoroughly and repeatedly. Fifthly, the fact that another child of the same family should also, and at about the same time, have passed blood-stained motions was perhaps the most perplexing factor of this difficult case. Had it not been for this fact I should have insisted upon operative interference on the occasion of my very first visit, when the patient was still of comparatively good health and the prospects were favourable. In the circumstances we had to await developments in the second child and in the meanwhile lost valuable time. Mr. Eddowes has since informed me that a few days after the death of the patient a native servant of the same household had developed undoubted dysentery, for which he was successfully treated at the Johannesburg Hospital. The question thus arises as to whether it is possible that both children had also suffered from dysentery, the baby developing her intussusception as a sequela to this disease. If so, one would expect the elder child to have been constitutionally affected, whilst apart from his blood-stained motions he appeared throughout to be in perfect health. The parents themselves attached little importance to his condition and the mother informed me that all her children from infancy had been known occasionally to pass blood in their stools whilst enjoying perfect health. Lastly, the fact of the situation of the intussusception in the course of the ileum is of interest, the enteric being one of the rare forms of intussusception.

Johannesburg.

## A CASE OF PNEUMOCOCCAL CEREBRO-SPINAL MENINGITIS SIMULATING "SPOTTED FEVER."

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THE following case appears to be of especial interest at the present time in view of the prevalence of epidemic cerebro-spinal meningitis in various parts of the United Kingdom. The patient, a man, aged 24 years, was an architect's assistant and had been working in the vicinity of Hampstead for some months. He was a well-built young fellow of considerable prowess as an athlete. His previous health had been good, but there was a history of a slight attack of influenza about a month ago, and he is said to have had a severe attack of "sunstroke" (fever, severe headache, and vomiting) about eight years ago, when he was in bed a week. The patient's mother's brother is said to have died from meningitis some years ago after an illness of 36 hours.

The patient's illness began on Feb. 12th, when he returned from work in the evening complaining of headache and pains in the limbs. He vomited three times during the night. On the next day he got up for a few hours; he was "dull and heavy" but did not sleep. The temperature was 100.2° F. He took a dose of cascara and had several loose motions. During the night he apparently had a fit. On the 14th he was seen for the first time by us. In the morning the temperature was 100°, the pulse was 108, the respirations were 46, the tongue was dry and coated with brown fur, and the throat and pharynx were dry and glazed; there was no enlargement or ulceration of the tonsils. He was in a semi-comatose condition and could only with difficulty be roused. The heart and lungs were normal except that there were some dry rhonchi at the

right base. There was a well-marked eruption, chiefly on the shoulders, chest, and back, but extending also to the abdomen, arms, and legs. It consisted of purpuric patches, the larger about a quarter of an inch in diameter, and small petechiæ resembling flea-bites. Kernig's sign and *tache cérébrale* were present but not to a marked degree, and there were no retraction of the head or muscular rigidity, and no strabismus or nystagmus. The pupils were equal and somewhat dilated and reacted sluggishly to light. His face was flushed and his appearance resembled that of a pneumonia patient, the movements of the *alæ nasi* being well marked. The urine was of specific gravity 1034, with a slight trace of blood and albumin. In the evening he had delirium of a muttering type and was unable to answer questions. During the night he had a slight fit. On Feb. 15th the patient was worse. He was unconscious and the breathing was more laboured. The temperature was 101.2°, the respirations were 50, and the pulse was 110. The right side of the face was now paralysed and the right arm and leg were flaccid and motionless. The left limbs were in constant movement, the left hand picking at the bedclothes, and the fingers presenting movement of athetosis. There was no cardiac murmur. A few rhonchi were heard at the base of the right lung, but there was no evidence of pneumonia. There were no ocular symptoms and no head retraction or rigidity of muscles. The patient became worse and died at 6.30 on the morning of Feb. 16th. A specimen of the cerebro-spinal fluid withdrawn by lumbar puncture was found to contain large numbers of the *diplococcus pneumoniae* of Fraenkel. Weichselbaum's *diplococcus intracellularis* was absent.

A necropsy was made on Feb. 17th. On opening the skull a considerable quantity of thick purulent fluid was found over the vertex in the pia-arachnoid space, most marked over the left hemisphere and affecting chiefly the occipital and frontal convolutions. At the base the nerves were found to be matted together. The dorsal surface of the cerebellum was thickly coated with the exudate which extended over the pons into the orbital region and into the fissures of Sylvius and Rolando. The mesial surface of the brain was also involved. Both lateral ventricles were dilated with excess of fluid. The basal nuclei were healthy. There was no sign of hæmorrhage. In the left lung a few small areas of consolidation were found in the upper part of the lower lobe, but there was no hepatisation. The bronchial glands at the root of the left lung were caseous. The right lung was normal. The heart muscle was flabby but the valves were normal. The liver and kidneys were congested but otherwise normal. The spleen was enlarged and diffuent.

The chief points of interest in this case appear to be (1) the presence of a well-marked purpuric rash in cerebro-spinal meningitis of pneumococcal origin; and (2) the absence of such characteristic symptoms of cerebro-spinal meningitis as head retraction, muscular rigidity, strabismus, and nystagmus. The case also emphasises the importance of a bacteriological examination of the cerebro-spinal fluid in cases of this kind.

Hampstead, N.W.

## Clinical Notes:

### MEDICAL, SURGICAL, OBSTETRICAL, AND THERAPEUTICAL.

#### NOTES ON TWO CASES: RUPTURE OF THE AORTA AND RUPTURE OF THE LEFT VENTRICLE.

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THE first case was that of a female patient, aged 79 years. She was slightly demented and for several years had led a quiet, regular life. She had been confined to bed for three weeks prior to her death, suffering from bronchitis with pyrexia, but had been convalescent for four days when she died suddenly on the fifth morning. At 10.5 A.M. on that day, while lying in bed, she all at once became very pale and died immediately.

The post-mortem examination revealed the following conditions. Both lungs were markedly emphysematous; the pericardial sac was found to be distended with about four fluid

ounces of serum and a firm blood clot, weighing nine ounces, which encased the heart. The heart was large and the aorta somewhat dilated. On opening the aorta a large rent was seen in the lower and posterior walls of the arch, measuring three inches in length and extending round three-quarters of the circumference of the vessel. The blood had evidently broken through the inner and middle coats of the aorta, dissected its way down in the tissues between the aorta and pulmonary artery, and finally at the origin of the latter vessel from the heart had burst into the pericardial sac. The walls of the aorta were thinned and showed some small atheromatous patches, though there were no marked signs of degenerative changes. The valves of the heart were healthy and competent.

The second case was that of a male, aged 57 years, who was also slightly demented and had not done any physical labour for two years. For the past nine months he had been in failing health; his arteries were very atheromatous and his general appearance was that of premature senility. He complained of a general feeling of weakness but had no pains of an anginal nature. The night before his death he went to bed apparently in his ordinary health. The next morning, while still in bed, he suddenly became very pale, gave two inspirations of a gasping character, and died.

At the post-mortem examination the left lung was found to be collapsed completely, evidently an old-standing condition, and occupied only a very small space in the upper part of that side of the chest, to the wall of which it was firmly adherent. The right lung was slightly emphysematous. The pericardial sac contained about six fluid ounces of serum and five and a half ounces of soft blood clot. A rupture, measuring half an inch in length was present in the anterior wall of the left ventricle, close to the intraventricular septum and midway between the apex and base of the ventricle. The edges of the rupture were ragged and the hole through the ventricular wall was cone-shaped, the base of the cone being the external opening. The heart muscle was flabby, but there was no naked-eye appearance of fatty degeneration. The aorta was atheromatous and numerous calcified plates were seen in its walls. The descending branch of the left coronary artery was almost completely occluded at its origin by atheromatous degeneration of its walls, and at this point the vessel was of a stone-like hardness. This branch was traced down to the site of rupture and evidently the myocardium, deprived of its blood-supply by the atheromatous obstruction, had undergone the necrosis spoken of by Ziegler as *myomalacia cordis*. The wall of the ventricle thus weakened by the necrosis had given way. The fact that the retracted left lung gave no support to the heart on that side may also have been a factor in the resulting rupture. Both these patients had been inmates of the Royal Aberdeen Asylum for some years.

I am indebted to Dr. J. Reid for his permission to publish these cases, and I have to thank Dr. Duncan of the pathological department at the University of Aberdeen for his kind assistance in the preparing of the specimens.

Aberdeen.

#### NOTE ON A CASE OF STREPTOCOCCUS WOUND OF THE FINGER.

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THIS case is reported as the unusually short period of incubation is of interest.

The patient, who was a married man, 44 years of age, was seen in the accident ward of the Massachusetts General Hospital on Oct. 28th, 1905. The history was as follows. About 8.30 A.M. on that day he was sparring with a fellow workman and in delivering one of his blows he struck the back of the index finger of his right hand against his opponent's tooth, making an incised wound. He was seen at the accident ward before 12 noon that day. Examination showed a wound on the index finger just posterior to the first knuckle; this was seared over. There was no other wound found nor were there evidences of any septic places around the hand. On opening the wound pus was seen. A cover-glass preparation was made and a culture was taken. The wound was douched, very slightly enlarged, touched with carbolic acid and alcohol, and drained. The finger did well and the man