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NOTES ON A CASE OF FISSURA ABDOMINALIS :

OPERATION AT AGE OF 2½ HOURS.*

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THE comparative rarity of a case of this type coming to operation, and the apparent recovery from the immediate effects of operative interference, are the reasons why I thought fit to report the case.

The subject was a seven to eight months premature female child, born before the arrival of Dr. Ferguson on June 5th.

When I saw the child two hours later a large mass of bowel lay exposed outside the abdomen without peritoneal or other covering. On washing the bowel with warm saline solution it was seen that the prolapse consisted of all the large intestine and several inches of the small, with a patent Meckel's diverticulum. A part of the bowel was covered by the gelatinous matter of the umbilical cord, a well-marked line indicating where it joined the skin. Close to the right of the base of the umbilicus, and joined to it by the gelatinous matter, was the orifice of the patent diverticulum. This diverticulum was about 2 in. in length, its diameter that of a cedar pencil, and joined the small intestine about 3 in. from its termination.

I removed the diverticulum flush with the bowel, and excised the rest of the gelatinous matter, freeing the umbilical cord at the same time from the margin of the fissure. The fissure, which was between 1 in. and 2 in. in length, was slightly enlarged towards the pubes, and the intestines placed within the abdomen. The deep layers of the abdominal wall were united by continuous catgut suture, and the skin by several points of silkworm gut, the umbilical cord being fixed in the upper angle of the wound. The child was slightly sick after the operation, but rallied well, and four hours after was fed with 3j of milk and 3iij of water; this was repeated every six hours. The following day a rectal catheter was passed and a considerable amount of flatus got rid of. Olive oil was administered by the mouth, but had no effect, so a small simple enema was given, with satisfactory result. The bowels subsequently acted naturally. There was no difficulty with micturition.

The diet was increased on the third day, the child still seeming hungry and taking well. The stitches were removed on the fifth day, when the wound was soundly healed; no signs of distension were present. On the sixth day, however, the child became jaundiced and passed a clay-coloured motion, and some purpuric spots appeared on the lower extremities. It was somewhat listless, and did not enjoy its food as before. It died on the evening of the eighth day.

I confess to being disappointed in the result, but feel that, could the child have been returned to its mother and had the advantages of breast feeding and animal warmth, it would probably have survived, as it never exhibited an abdominal symptom and seemed to be progressing satisfactorily until the sixth day.

I have not been able to find much information in the literature, but would refer to a case recorded by Mr. Lockhart Mummery¹; it was not operated on and died. In Mr. Edmund Owen's work on the *Surgical Diseases of Children* an excellent photograph and remarks on treatment appear.

REFERENCE.

¹ *British Journal of Children's Diseases*, 1904.

ASCARIS LUMBRICOIDES AS CAUSE OF APPENDICITIS.

By ALDO CASTELLANI, M.D.,

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IN a very interesting paper published in the *BRITISH MEDICAL JOURNAL* of March 26th, 1906, Dr. F. E. Anley has suggested the hypothesis that *Ascaris lumbricoides* might be the cause of some cases of appendicitis—particularly in the tropics where that worm is so common. In support of his theory Dr. Anley quotes two cases in which the symptoms of the disease subsided when a round worm was passed in the stools. He is of opinion that the inflammation might be set up by the worm attempting to

enter the appendix or by the worm obtaining a partial entrance, if no more, into the organ.

Nearly two years ago I had the opportunity of making in Ceylon a *post-mortem* examination which seems to me to prove that Dr. Anley's supposition is in some cases an actual fact.

The body was that of a young Tamil girl of about 14 years of age. The clinical history sent with the body was unfortunately not very complete, but the following principal points could be gathered. The girl had been suffering from intestinal helminthiasis for a long time and had passed round worms in the stools on several occasions. She was given santonin. Shortly after the administration of the drug she had vomiting, pain and tenderness on the abdomen, especially on the right iliac fossa, with some rise of temperature. This condition lasted apparently unchanged for about two days, when the girl died suddenly.

The *post-mortem* examination was held a few hours after death. There was nothing to be noted concerning the thoracic organs. There was no effusion into the peritoneal cavity, and the peritoneum did not show any sign of inflammation.

The appendix was observed to be protruding, and presented engorged superficial vessels, and here and there small areas of fibrinous exudation. The organ was quite free, no adhesions whatever being present. To the touch it was hard, and felt as if it contained a cylindrical body. The intestine having been opened, this body was seen to be an ascaris firmly imbedded for about one half of its length in the appendix, the other half being free in the intestine. The worm was very firmly inserted into the appendix, and could not be pulled out, although some force was used. The worm was apparently dead, as the free portion did not show any movements. On squeezing the appendix towards the base a little purulent liquid oozed from between the walls of the appendix and the ascaris. This liquid was examined bacteriologically; the *B. coli* only was found. The small intestine contained numerous ascarides, some of which were still alive. No other helminths were present. The mucosa of the small and large intestine was slightly hyperaemic, but no ulcers of any kind were present; the mesenteric gland were not enlarged.

It might be said that the worm entered the appendix after the death of the patient; it seems to me that this cannot have been the case, as the worm was very firmly imbedded, and was dead when the examination was made.

A photograph of the appendix containing the worm was taken, and is here given. Later on sections of a portion of the appendix were examined microscopically. They showed a diffuse leucocytic infiltration of all the coats; the mucosa was swollen, and several minute haemorrhages were seen. The lumen was completely occupied by the worm, the external surface of which was surrounded by a zone of desquamated and exudated cells from the mucosa, which showed several erosions.

In conclusion, it appears to me that the result of this *post-mortem* examination clearly proves that ascarides

* Read at the annual meeting of the Ulster Branch of the British Medical Association, June 21st, 1906.

under certain circumstances may be a cause of appendicitis. In the case I have related a point which might be discussed is whether the santonin may not have indirectly caused or facilitated the entrance of the worm into the lumen of the appendix; it is well known that according to the experiments of Schroeder and Coppola, santonin has a convulsing action on the ascarides, rendering their movements for a time much more active. The onset of appendicular symptoms soon after the administration of the drug is somewhat suggestive.

MEMORANDA: MEDICAL, SURGICAL, OBSTETRICAL.

GUMMA OF THE BRAIN.

W. S., aged 50, a well-built fair-complexioned man, was seen on January 4th, 1903. He lay apparently unconscious. By vigorous shouting he could be roused to semiconsciousness and was able in response to give an unintelligible mumble but could not speak. Feeding had to be conducted by spoon or feeder. Faeces and urine were both passed under him without his apparently being aware of the fact or any rate without his taking any notice of such occurrence. The right arm was completely paralysed and flaccid. There was a marked drop of the right angle of his mouth, paresis with rigidity of his right leg. Both knee jerks were active and the plantar reflexes flexor in type. On the penis was a large papilloma thought to be syphilitic. His history was as follows: He was an old soldier now following the occupation of carter. On November 10th, 1902, he fell off his cart, it was thought in a fit, was found unconscious on the road and taken home. On recovering it was found that his right arm was weak; he was at that time able to get about, but not able to work. He has had several fits since. On November 27th it was noticed that his speech was getting thick, and since then he progressively got worse until he reached the condition observed on January 4th, 1903. The treatment adopted was potassium iodide 1 drachm, and liq. hydrarg. perchlor. 1 drachm three times a day. Also the inunction of half a drachm of ung. hydrarg. daily. His condition on January 31st, 1903, was as follows: Bright and lively in manner, intelligent, talking incessantly during the whole of the waking hours. He possessed perfect movement of face, arm and leg, but with some weakness of the right arm; eating largely of an ordinary diet, able to get up and be weighed, no sign of stomatitis. On February 8th he had gained 10½ lb. during the previous week, was walking about and going down to the garden. Before long he was at work again, and has continued at work ever since. About nine months later he had a fit, due apparently to the old scar, and he has had two since. The fits are completely controlled by small doses of bromide, and he has never since had any sign of recurrence. He is now (June, 1906) a fine-looking man, weighing about 14 stone, and doing a hard day's work. His optic neuritis cleared up, and has apparently not affected his sight.

Nottingham.

F. H. JACOB, M.D.

OCCIPITO-POSTERIOR PRESENTATION.

I READ Dr. Geddes's paper in the JOURNAL of March 10th with much interest, especially as his figures very nearly coincide with mine, and I was, until I read his paper, under the impression that occipito-posterior presentations were much more common in Australia than at home.

Of 70 confinements I attended in England of which I have a record, I had but 3 occipito-posterior presentations. In 276 cases attended in Western Australia there were 44 occipito-posterior—a percentage of 16.

Following Dr. Geddes's method of classification, I find that in 55 primiparae with vertex presentation 12 were occipito-posterior, or 21.8 per cent. (Dr. Geddes 20.8 per cent.). In 132 multiparae with vertex presentation, 22 were occipito-posterior, or 16 per cent. (Dr. Geddes 10.5 per cent.). (Prior to 1904 I did not record whether cases were primiparae or multiparae.)

I agree with Dr. Geddes as to the importance and difficulty of diagnosis. He makes no mention of abdominal palpation, which I regard as one of the most helpful means of discovering this condition. These cases

are always tedious, especially when, as often happens, the "waters break" early. How can we save our patients this long and trying time of ineffectual pains? The method of pushing the child into an occipito-anterior position, with the hands placed on the mother's abdomen as recommended in Herman's *Difficult Labour* I have not found successful. But when the cervix is well dilated, with the left hand grasping the child's head, and the right hand assisting through the abdominal wall, the head can often easily be turned into an occipito-anterior position. I have on several occasions successfully delivered in this way when traction with forceps was ineffectual.

Another matter upon which Dr. Geddes only just touches is the increased risk to the child. Of my 44 cases 5 were stillborn, 1 due to prolapsed funis, the others, I think, due to cerebral haemorrhage. Two children were born alive with signs of cerebral haemorrhage, and have been partially paralysed since; both have been trephined, but neither of them with entire success.

If forceps are used, I believe the tips of the blades sometimes may catch the child under the angle of the jaw, and cause death by compression of the carotids. The only way to avoid this disaster is, I think, to rotate the head as I have described above.

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OTECTOMY IN OTORRHOEA.

THE work of the past year has confirmed my opinion as to the advisability of removing diseased tissues in the middle ear. Otorrhoea does not generally receive the consideration it deserves. It is the cause of far more trouble than is generally supposed. In the county hospital I had recently, at one time, 3 cases of meningitis, 1 of intracranial suppuration, and 4 of extensive disease of the temporal bone, all the result of neglected otorrhoea or inefficient treatment. As these complications are in almost every case preventable by an operation that I have proved to be practically free from danger, I think it is my duty to press this matter upon the attention of medical men. It would be difficult to exaggerate the number of people whose health is being deteriorated and whose very life is threatened by the existence of this disease. They are to be found in every street, though many of them live in happy ignorance. Nor does every medical man always remember that the most easy path for disease to reach the brain is that of the aural cavities.

Coventry.

F. FAULDER WHITE.

NEURITIS IN PHTHISIS.

IN connexion with the case published in the BRITISH MEDICAL JOURNAL, June 23rd, 1906, p. 1464, the following will be of interest:

About two years ago I was called to see a man aged 30, who had complained of progressive debility, wasting, and pain in his legs for a few months. I found that he was extremely emaciated, and could hardly stand on his legs on account of the very great weakness. He did not complain of cough, and there was never any haemoptysis, or any other symptom of phthisis apart from the wasting. The legs looked exceedingly wasted, and were practically reduced to "skin and bone." They were painful and hyperaesthetic. There was no marked foot-drop. On account of his greatly debilitated condition I could only make a very cursory examination of his chest, and found somewhat suspicious, but by no means convincing, signs of pulmonary tuberculosis. He had been under hospital treatment for several months, but was not informed of the nature of his complaint. The urine was copious, sp. gr. 1006, and contained no albumen, no sugar, and no casts. The neuritis was therefore evidently not diabetic. The simultaneous onset of atrophy in all the muscles of his body excluded a diagnosis of progressive muscular atrophy to explain his wasting and loss of knee-jerks. I therefore came to the conclusion that he was probably suffering from pulmonary tuberculosis with peripheral neuritis as a complication.

I sent him to the London Hospital, and the physician under whose care he was admitted, was good enough to write to me that he could not, owing to the patient's condition, make a thorough investigation of the case, but that on the whole he agreed with me as regards the evidence