no obvious lesions at laparotomy, are relieved of symptoms by appendectomy. That intussusception occurs and reduces itself, even in the normal abdomen, was strikingly demonstrated in Alvarez's film of intestinal movements, exhibited in Ireland by the Kodak Company two years ago. Additional support is given to this opinion by Myles's unpublished observation of such an invagination of the appendix into the caecum occurring spontaneously and reducing itself with regular rhythm in a patient on whom he was operating for caecal hernia.

Liek⁴ and others, on account of the frequent absence of microscopical changes, state that many cases diagnosed as "chronic appendicitis" owe their symptoms to local spasm of neurotic origin, and treat these cases, often successfully, with large doses of atropine. The view expressed above is capable of explaining their results: laparotomy causes the invagination to become reduced, unless it is held in place by adhesions, and so no lesion is found at operation; the atropine relaxes the spasm of the appendix sphincter, thus preventing recurrence of the intussusception, and thereby relieving the patient's symptoms. It has frequently been shown experimentally that simple laparotomy as a rule abolishes all intestinal movements, and therefore it is not surprising that the actual occurrence of such an intussusception has not been oftener observed: if appendectomy were performed under local and splanchnic anaesthesia (which does not inhibit peristalsis) it might be possible to get additional evidence as to the truth of the above explanation.

SUMMARY

1. A case of appendiceo-caecal intussusception is described.

2. The clinical history, as found in the literature, is summarized.

3. Evidence is adduced suggesting that the condition is not as rare as would appear from the literature, and that it may be the real lesion in many cases of "chronic appendicitis."

REFERENCES

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 (These two papers contain a complete survey of the earlier case reports.)
¹ Clubbe: *Diagnosis and Treatment of Intussusception*.
⁴ Liek: *Irrwege der Chirurgie*, 1929.

INFARCTION OF THE HEART WITH PERICARDIAL EFFUSION IN A YOUNG PHTHISICAL SUBJECT

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The following report deals with the occurrence, in a young tuberculous subject, of the symptoms and signs of cardiac infarction, with the subsequent development of a pericardial effusion.

A Moslem male, aged 28, was admitted to hospital on August 8th, 1930, with chronic tuberculous disease of both lungs. Constitutional symptoms were severe; there was considerable wasting, and the sputum contained numerous tubercle bacilli. No signs of extrapulmonary tuberculosis were detected, nor was there any evidence of cardiac disease. The patient made good progress until October 6th, by which time the temperature had settled down to normal, and the weight had increased by a stone. Tubercle bacilli were, however, still present in the sputum. On the latter date, about 7 a.m., while resting quietly in bed, the patient suddenly experienced

pain of an extremely severe nature all over the cardiac area and under the lower part of the sternum, the position of maximal intensity being just inside the apex. The pain radiated widely—down the inner side of the left arm into the hand, down the right arm as far as the elbow, and down the left side of the body and left leg as far as the knee. It yielded only to large doses of morphine, nitrites having no effect. The patient was very restless, and complained of difficulty in breathing. The possibility of pneumothorax was considered, but there was no sign of this condition. The pulse became very weak and rapid, but was quite regular. The heart sounds were almost inaudible. The temperature, which was subnormal when the pain started, soon became elevated. The patient was very bad for three days, in spite of cardiac stimulants, and he became unconscious for about three hours on October 8th. After this date there was gradual improvement, and by the 12th the pain had gone, but the temperature continued to swing to over 101° F.

On October 14th, however, at 4 p.m., the pain returned in its original agonizing form, the position of maximal intensity being now in the epigastrium. It radiated to the back and down the inner side of the left arm. Severe collapse again accompanied the pain, and the patient was in a critical condition until October 23rd. By this time there was a slight icteric tinge in the conjunctivae, and the blood serum gave an indirect positive van den Bergh reaction. The liver was four fingers below the costal margin, and somewhat tender. The circulating leucocytes were found to number 18,000 per cubic millimetre, the differential count being: polymorphs 84 per cent., lymphocytes 13 per cent., monocytes 3 per cent. The blood pressure ranged about 90/70. On October 17th a pericardial rub was detected inside the apex, and remained audible until November 2nd.

Towards the end of October the cardiac dullness began to increase rapidly, and by November 5th there were signs of extensive pericardial effusion. On November 15th 6 ounces of fairly clear straw-coloured fluid were removed and examined. Red blood cells were present in fair numbers, and the lymphocyte was the predominating white cell. Guinea-pig inoculation was negative. The patient remained in hospital till November 16th, when he left at his own request. His condition was then fair. He had been almost free from pain during the last fortnight, and the temperature had begun to come down.

Apart from the fact that there were two distinct and typical attacks of severe pain, with collapse, there are some features of the case that deserve special mention. The age was much less than that at which infarction of the heart is usually seen. The great majority of cases occur between the ages of 50 and 70, although attacks are fairly common after 40. In a series of 145 cases analysed by Levine and Brown¹ only three were under 40, and Parkinson and Bedford² report 7 under 40 in a total of 183. These include two patients under 30, in both of whom coronary embolism was the cause of the infarction.

An acute pericarditis, as evidenced by a friction rub in the vicinity of the nipple and near the sternum, is not an uncommon feature of myocardial infarction. The development of pericardial effusion, however, seems to be rare. Levine and Brown found it in only one patient in their series. This was a man, aged 78, who had had cough and increasing dyspnoea for years. Parkinson and Bedford found it once among 100 cases seen clinically. X-ray evidence of effusion was present in their case ten days after infarction. In our case a friction rub was first heard three days after the onset of the second attack of pain, and enlargement of the cardiac area was detected about two weeks later.

We have not found any previous mention of pulmonary tuberculosis being associated with infarction of the heart. The latter occurs most frequently in patients with evidence of atheroma, hyperpiesis, or arterio-sclerosis, and in many cases there is a history of angina pectoris. Syphilis and rheumatic fever and gout are occasional causative factors. Levine and Brown point out that it is frequently associated with diabetes. In the case here

reported there were no clinical manifestations of any of these conditions. In spite of the fact that guinea-pig inoculation was negative, it is probable that the fluid was of tuberculous origin. Its characters and cytology suggested this, as did the prolonged irregular fever that accompanied its presence, although the latter may have been due to some extent to a reawakening of the pulmonary disease.

Electrocardiograms taken at frequent intervals after the first attack showed a normal P-wave and QRS complex in all leads. The so-called "coronary T-wave" was not present, but the portions of the curves normally occupied by the iso-electric R-T segment and the T-wave were made up of small undulations, each occupying about 0.1 second or less. This feature was most marked in Lead III.

Our thanks are due to Dr. D. L. Shrivastava, who took the electrocardiograms, and to Drs. K. K. Jaswal and Shujat Ali, house-physicians, for detailed notes of the case.

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- ¹ Levine and Brown: *Medicine*, 1929, viii, 245.
- ² Parkinson and Bedford: *Lancet*, 1928, i, 4.

Memoranda

MEDICAL, SURGICAL, OBSTETRICAL

NON-FILARIAL ELEPHANTIASIS

I was interested to read the account by Mr. D. J. Harries in the *British Medical Journal* (December 27th, 1930, p. 1079) of a case of non-filarial elephantiasis treated by a modified Kondoléon operation. Since these cases are rare I venture to record a case of my own treated in the same manner.

A female, aged 9, was admitted to the Hackney Hospital under my care in May, 1929, complaining of swelling of the left foot. The dorsum of the foot pitted on pressure, and in a few weeks the oedema had gradually spread upwards until the whole of the left limb was involved. The swelling was definitely painless; the skin was unaltered in colour, and apart from the inconvenience of fitting on a shoe the child had no difficulty in walking. A curious feature was that the oedema terminated abruptly at the level of Poupart's ligament in front and the posterior superior spine behind. The Wassermann reaction was negative, and the general health of the child was good.

For several weeks the patient was kept in bed, with the foot supported on pillows. Despite treatment by massage, electricity, and drugs, the limb continued to increase in size, but never did the oedema spread above Poupart's ligament. In March, 1930, it seemed justifiable to perform a modified Kondoléon operation. An incision was made on the outer side of the leg from the anterior superior spine to the external malleolus, and a strip of skin, $1\frac{1}{2}$ inches wide, was removed together with a wider strip of oedematous subcutaneous tissue and deep fascia, the wound being closed with interrupted sutures. A similar operation was performed on the inner side of the leg, the incision extending from the saphenous opening to the internal malleolus. At intervals the muscle sheaths of the extensors were incised longitudinally. The subcutaneous tissue was markedly oedematous, and the deep fascia was not easily defined.

There was no alteration in the size of the limb for a week. A decrease was then evident in the region of the knee, and the whole of the area was definitely softer. For several weeks the child was kept in bed, and later the leg was rested on a chair, massage and electricity being administered regularly. Very little improvement, however, can be recorded as regards the dimensions of the limb, and the measurements taken in January of this year do not differ materially from those taken immediately before the operation. The general condition of the child at present is excellent. She walks without a limp, and the leg is growing normally. She is still, however, faced with the distressing prospect of an oedematous limb with almost the whole of her life in front of her.

It is interesting to compare this result with the more successful one obtained by Mr. Harries. He claims quite rightly that his result shows the value of a Kondoléon operation in the treatment of non-filarial elephantiasis. In the case I have described the effect was less striking. In fact, these divergent experiences bear out the accepted opinion that the results of the Kondoléon operation are not invariably completely satisfactory. Some writers have recorded cases which have responded to the operation with remarkable suddenness. In *Surgical Pathology* Boyd describes a case of a girl, aged 17, whose arm returned to its normal size four weeks after the operation. Six months later the left leg and foot became swollen, yet within four days of the operations on these limbs the swelling had subsided. Many other writers, however, have met with little or no success. One is forced to the conclusion that the operation is not to be entered upon optimistically, but since occasionally successful results have been obtained, such as in the case of Mr. Harries, there is no doubt that this treatment is worth a trial as a last resource. I agree with him that the operation is easy to perform, and there is little shock. Both cases support the statement that this disease is more common in females.

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MECKEL'S DIVERTICULUM CAUSING ACUTE INTESTINAL OBSTRUCTION

In view of the following unusual termination of an attack of diarrhoea and vomiting in a boy aged 10 years, I think the facts may be of some interest.

The boy was first seen on account of diarrhoea and vomiting, which had followed a surfeit of fruit tart. The temperature was normal, pulse rate 80, and any gross abdominal lesion was excluded by examination. As a result of a diet restricted to albumin water and the administration of bismuth carbonate and tincture of belladonna, the vomiting ceased on the next day and the diarrhoea on the third day. On the fourth day the patient began to vomit again, and despite treatment vomiting became more frequent. The pulse rate remained about 80, there was no rise of temperature, and nothing abnormal could be found on abdominal examination.

On the fifth day the vomiting still persisted, and the patient complained of pain in the abdomen. He described the pain as "coming and going." The pulse rate was 100, and the temperature was normal. The boy had an anxious expression. Beyond a suggestion of tumidity, nothing could be made out on abdominal examination. The administration of two enemata at intervals of ten minutes resulted in failure to pass faeces or flatus, and he was removed to the Merthyr General Hospital as a case of acute intestinal obstruction.

The abdomen was opened by a right paramedian incision below the umbilicus, and an intussusception of the ileo-colic variety found. There was only slight difficulty in reducing this; the apex of the intussusception proved to be a Meckel's diverticulum about one inch in length with a flattened tip, the tip having an umbilicated appearance. This was removed, the gut sutured, and the abdomen closed.

The patient made an uninterrupted recovery.

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VOLVULUS OF A LARGE APPENDIX EPIPLOICA

The following case, which presented rather misleading symptoms and signs, seems sufficiently obscure to be worthy of record.

A housewife, aged 50, was admitted to hospital on December 16th, 1930, complaining of abdominal pain and "anaemia." She had had a similar attack of pain five years before. On inquiry it appeared that for the previous three weeks she had suffered from acute intermittent epigastric pain, increasing in severity and worse after meals. She had vomited several times immediately after a meal; no haematemesis. Bowels usually regular, but diarrhoea present; no melaena; no blood per rectum.