of age, and increases gradually to 44.5 per cent. in cases beginning after 45.

2. *The Duration of the Illness before Treatment.*—In cases coming into hospital who have been ill under one year the death-rate was 35 per cent.; between one and two years, 75 per cent. The rate falls after the fourth year, as after that length of time the disease may be regarded as chronic and symptoms are always less acute.

In addition to these points, early development of very acute symptoms, such as diarrhoea and tachycardia, adds to the gravity of the prognosis.

D.—COURSE.

Treatment consisted in rest in bed in all cases. Various drugs were used according to the most prominent symptoms in each case.

Up to 1908 most patients were treated by the electric battery, and more recently by *x* rays. There was considerable temporary improvement, but possibly no more than by other means. It is too early to speak of the permanency of the improvement under *x* rays.

Rodagen, thyroidectin, antithyroid serum, and anti-thyroidin have all been used, and while each has been successful in some cases, none has had a generally good enough result to encourage its universal use.

Of 93 cases 64 have been traced at periods varying from six months up to ten years since leaving the infirmary.

- 18.75 per cent. declare themselves quite well and able to work.
- 20.3 per cent. are definitely improved.
- 20.3 per cent. are much the same.
- 4.75 per cent. have developed myxoedema.
- 6.25 per cent. were transferred to surgical wards.
- 29.7 per cent. are dead (6.25 per cent. while under treatment in the infirmary).

Many patients who are entirely cured of all disturbing symptoms and who are otherwise absolutely well, retain some permanent enlargement of the thyroid.

I am indebted to the past and present honorary physicians of the Royal Victoria Infirmary for permission to make use of their cases and notes, and especially to Professor Murray, of Manchester (late of Newcastle), who has allowed me to use many particulars of cases treated by him.

THE CAUSE OF APPENDICITIS.

BY

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AMBULANCE.

THE question as to what brings about the pathological conditions in an appendix which give rise to appendicitis has been widely discussed from several standpoints.

I venture to suggest a theory, which I think is new, as in the literature I can find no allusion to it.

All cases of appendicitis are in the first place due to a mechanical cause, and this consists in a rotation of the organ about its meso-appendix.

I consider the acute inflammatory process, whether it go on to abscess formation or not, to be purely secondary to this rotation.

This is assumed for the following reasons:

- (a) The valve of Gerlach is by no means a reliable valve.
- (b) In almost all quiescent appendices, which have never given rise to any symptoms and which have been removed during pelvic operations, I have always been able to culture intestinal organisms from their cavities—that is, the normal contents of the appendix are not sterile.

What Happens when the Appendix Rotates?

Anatomically the appendix is quite free to rotate on its mesentery, and as to what actually takes place will depend upon the degree of rotation.

If the rotation be complete, then the condition is similar to that of an ovarian cyst completely twisted about its pedicle. The venous return is stopped, but the arterial supply is not wholly cut off. We therefore get initially a state of acute congestion. Infection supervenes on this

damaged organ, and gangrene with abscess are the usual termination.

This type of case is familiar in that its onset is sudden, the pain is acute, the patient vomits, which vomiting I take to be purely reflex; in fact, the initial symptoms are similar to those of a twisted ovarian cyst in the female.

In less acute cases the rotation is not complete, and the venous return is not wholly cut off. These patients, if put to bed and kept quiet, invariably get over their first attack.

The symptoms at the onset are not so acute; there may be vomiting, certainly some pain, there is tenderness over McBurney's point, but all this usually subsides.

These patients, if operated upon in the acute stage, have, always in my experience shown some degree of rotation of the appendix. The organ is stiff and congested, and there are subserous haemorrhages.

Similar cases operated upon in the quiescent periods have shown the familiar kink in the organ, and if this be carefully examined it will be found to be produced by a partial rotation of the organ about its mesentery.

The third type of case is that where there is a long meso-appendix and the organ from mechanical conditions I shall mention directly is rotating on its mesentery and recovering itself.

This type of appendix has, in my experience, invariably been the one that has given rise to gastric symptoms, which Sir Berkeley Moynihan has called "appendix dyspepsia." My experience in this type of appendix is interesting, and I hope others will have noticed this. In four cases I performed a laparotomy with the purpose of performing gastro-enterostomy. In all four cases the stomach was dilated, but no ulcer or disease was to be found, though the symptoms were those of gastric ulcer. In these cases a very movable caecum was present with a long appendix suspended by a long meso-appendix; there had been marked constipation in all four cases. I did nothing further than remove the appendix, and the result in all four cases was highly satisfactory, as all the gastric symptoms disappeared and the bowels moved regularly.

It is well known that abdominal operations in which any extensive handling of the mesentery is necessitated give rise in the patient sometimes to acute dilatation of the stomach. If, then, there is anything present which is constantly tugging on the mesentery over any length of time, it is quite reasonable to suppose that the stomach will react in the same way, but in a more chronic fashion. The process, it is fairly agreed, is reflex, and I think, seeing that these cases were cured, we have in this the explanation of the so-called "appendix dyspepsia."

Surgeons will, I think, recognize how difficult it often is to trace an appendix and its whereabouts when it has gone on to a condition of abscess formation, and those who do not believe in amputation of the appendix at these times, but who are content to find the abscess and drain it, will not have, perhaps, the opportunity of bearing me out; but they will surely find, if they go further with the operation of removing it, that the difficulty lies in the fact that the appendix has completely rotated on its own mesentery, and has become bound down, and there often appears to be no meso-appendix.

This, however, is a fallacy, the meso-appendix being shortened by the twist.

What, then, causes this Rotation?

The chief factors which bring this rotation about are:

- (a) A movable loaded caecum.
- (b) Loss of tone in the abdominal muscles.

The most important is the second of these two factors, for on it in some measure depends the first.

A loaded movable caecum is free to move in three ways:

1. It can descend.
2. It can move laterally.
3. It can expand forwards.

These three conditions are just those required for a rotation of the appendix on its meso-appendix, and the only condition that can bring these about is constipation.

Loss of tone in the abdominal muscles is one of the commonest causes of constipation and later of some amount of enteroptosis.

I do not think I am far wrong in saying that if people took regular exercise and kept their abdominal muscles in good trim, if people in sedentary occupations would resort to physical exercises designed to keep up the tone of their abdominal muscles, and if there were less conveniences for those who can walk, there would be less appendicitis.

The interesting question arises as to whether a "strain" can cause an attack of appendicitis. I think it can. I have had a few cases where the condition has followed a sudden strain. If suddenly the abdominal muscles contract, especially on a loaded caecum, it is quite easy to see how this could bring about a sudden rotation of the appendix on its meso-appendix.

What I have said in this paper has struck me before the beginning of the present war, but from statistics I have observed since mobilization in August I am still further convinced.

I had a number of cases of acute appendicitis I operated upon early after mobilization, but out of two brigades there has not been an acute case for nearly three months. I think this speaks for itself.

TWO CASES OF DIAPHRAGMATIC STAB WOUND.

WITH OBSERVATIONS ON THE PRODUCTION OF DIAPHRAGMATIC HERNIA AND OPERATIVE TECHNIQUE.

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CASE I.

A CHINESE rickshaw puller, aged 30, was brought to the General Hospital, Seremban, by the police on April 7th 1912, with a stab wound of the right chest.

History obtained from the Police.—At 6.30 a.m., while purchasing a ticket at the railway station, his companion when standing behind him thrust a small hunting-knife with a 4-in. blade into the right side of his chest.

State on Admission.—He was admitted at 9.30 a.m., three hours after receiving the wound. His clothes, consisting of a cotton jacket and pants, were saturated with blood. He was exsanguine, very restless, and unable to answer any questions owing to his fainting and collapsed condition. Respiration was shallow and frequent (54 per minute), with grunting expiration. The pulse could not be counted at the wrist. There was a stab wound in the fourth intercostal space in the anterior axillary line, from which blood trickled in a constant stream and was being churned into a bloody froth by the air exchange through a patent thoracic fistula. The abdomen was tender over the right side, most definitely in the iliac region and loin.

Operation.

A vertical elliptical incision round the stab wound was made from the third to the seventh rib and the outlined ellipse of skin excised. The fifth and sixth ribs were resected for $4\frac{1}{2}$ in. The periosteal beds were each incised in the whole length thus exposed. Two Kocher clips were passed from these wounds to include the tissues of the fifth space. The structures were divided between the clips, and the flaps thus produced were folded outwards. When the blood and clot filling the chest were turned out, blood was seen to be entering the pleural cavity through a wound in the muscular portion of the diaphragm about 3 in. long, with a direction, from within, outwards and forwards. Respiration was very embarrassed, but at once improved when the lung was gripped in a clamp and part of the lower lobe held outside of the wound, thus fixing the mediastinal structures, as pointed out by Kelly. A finger, in the diaphragmatic incision, discovered a wound of the subjacent upper surface of the liver, just in front of the coronary ligament. A gauze plug, introduced through the diaphragmatic rent, failed to control the haemorrhage. The diaphragmatic wound was extended a little in both directions, and pressure by an assistant on the abdomen helped to fix the liver. A wound, about $1\frac{1}{2}$ in. deep and some $2\frac{1}{2}$ in. in length, bleeding freely, was well exposed. This was closed by one long mattress suture of catgut passed deep to the wound from right to left and back from left to right, tied parallel to the wound. A sharp-pointed, fully-curved needle, being all that was to hand, was used, and

this produced no fresh bleeding from its tracks when the stitch was tied. Four interrupted sutures through the capsule with fine catgut got tidy approximation of the previously everted lips. The incision in the diaphragm, now some $4\frac{1}{2}$ in. in length, was closed by a continuous blanket stitch of catgut extending a little beyond each end of the incision. A small incision of the margin of the lower lobe of the lung was sequestered by a catgut ligature. The chest flaps were united, and the skin closed without drainage. He had received saline (normal), 2 pints, into a vein of the left arm during the above manipulations, and the anaesthetic used was ether two parts, chloroform one part, on a mask.

The pulse when he left the table was 144, soft, and with a collapsing vessel. Six hours later the pulse was 128. He then had a deposition taken by a magistrate, involving half an hour's oral examination and the identification of his assailant. Continuous proctoclysis during the next twelve hours sustained an almost constant pulse-rate. He rapidly developed bronchitic sounds over the left lung, and breath sounds were never present on the right side. He died in respiratory distress thirty-two hours after the accident.

Necropsy.—Right lung collapsed; no haemorrhage in chest. Liver wound dry; some soiling of the right abdomen with tarry blood; about 2 oz. of blood in pelvis. Left lung, marked capillary bronchitis.

CASE II.

A Chinese coolie, aged 31, working on the Labu F.M.S. Rubber Estate, was sent to the Seremban General Hospital by the manager of the estate at 2 p.m. on October 23rd, 1912.

History obtained on Admission.—During a quarrel on the previous evening he was struck over the left chest with a sharp-pointed, triangular file. He had suffered considerable pain in the chest and abdomen during the night. This had been relieved by opium, administered to him by his friends. As he was worse in the morning, and had vomited several times, the manager was informed. With the case he sent a note informing me that "the man was vomiting, and wanted to pass water constantly."

State on Admission.—Eighteen hours after the injury he was drowsy from opium, with contracted pupils, but still complained of some general abdominal pain. He was a little cyanosed, and had frequent shallow respirations of 30, and a full, strong pulse of 60 per minute. There was a triangular-shaped wound on the left chest over the eighth interspace in the mid-axillary line. This small wound had been plugged with gauze, and the surrounding skin was blistered by some strong antiseptic dressing. The left chest was hyper-resonant, and breath sounds were absent. The abdomen was full, with all the muscles rigidly on guard, in spite of the liberal opiate. Tenderness was general, and was best demonstrated when steady pressure was suddenly relaxed. There was some bulging and tenderness of the pelvic pouch on rectal examination, and the pelvic peritoneum was tender. When the small gauze plug was removed from the wound there was no bleeding, and air could not be detected as entering the chest cavity. A director was passed into the wound in a forward and inward direction, with a downward inclination. This oblique penetration of the chest wall produced in the dorsal position complete closure of the thoracic fistula. When the valve was momentarily obliterated by the director, air was aspirated through the wound, and blood trickled out.

Operation.

As everything was ready in the theatre for another laparotomy, the operation was started within half an hour of his admission. Under chloroform, a 3 in. median sub-umbilical incision was made, and about half a pint of blood was evacuated. Distended small intestine crowded into the incision, but was not allowed to herniate. A tube was passed into the pelvic pouch, and the lips of the peritoneum temporarily closed by "Kocher" forceps applied above and below it. A penetrating wound of the diaphragm was diagnosed, and a wound of the liver or spleen suspected. An oblique subcostal incision, falling 2 in. below the left costal edge, extending from within an inch of the mid line to below the tip of the eleventh rib, was made. The anterior sheath of rectus was dissected up and down from the muscle, and, at its outer edge, the anterior portion of the internal oblique aponeurosis was divided at its line of fusion with the external oblique aponeurosis. The rectus was raised from the posterior sheath and retracted inwards. The internal oblique was split, and the abdomen opened through the transversalis and peritoneum for the whole length of the wound. Pads, below and to the right, controlled the distended gut. The spleen was delivered, but was found unwounded; it was remarkably "unpathological" for a resident in this country. The diaphragm could be well inspected through this