PERFORATION OF THE STOMACH AND DUODENUM.

There is abundant evidence for the view that these serious lesions may undergo a natural cure, but this must not be offered as an excuse for delay in their treatment, because they still rank as the most dangerous of the abdominal emergencies.

Simple ulcer of the stomach or duodenum is the recognized cause of the great majority of perforations, and if they can be satisfactorily closed before the leaking contents have widely infected the peritoneum, recovery is almost a certainty.

A history of previous "indigestion" is obtainable in the great majority; then comes a sudden overwhelming pain. In duodenal cases the intestinal contents, leaking into the subhepatic pouch, escape down the gutter on the outer side of the ascending colon and caecum into the pelvis, and may cause the chief pain, tenderness, and rigidity to be referred to the right iliac fossa. This accounts for the frequency with which such cases are sent with a diagnosis of appendicitis. Mistakes will be less frequent if a history typical of duodenal ulcer is available, and if the sudden overwhelming onset receives due consideration.

[Photographs of specimens of ruptured gastric and duodenal ulcers were shown.]

CASE III.—Ruptured Gastric Ulcer.

This youth, aged 17 years, was admitted January 8th, 1920. He brought with him the following note from his doctor:

Dear Sir,

I hope that you will admit F. C., aged 17 years. Diagnosis is—Ruptured gastric ulcer.

History.—The patient has been troubled with slight dyspepsia (he describes it as a sickly pain, though he never vomited, coming on about two hours after food) for the last three weeks. At 6.50 this evening, when at work, he was suddenly seized with severe abdominal pain. He attempted to walk home, but when he got half way was found by a lady standing leaning against a pole and groaning with pain. The lady helped him home, and got him there with difficulty. He has vomited twice since getting home.

I saw the patient at 8 p.m. He was lying on his back groaning with pain. He was very pale, cold, and collapsed. Pulse 112, and very faint; temperature 95.6°. His abdomen was as hard as a board on examination and very tender. Liver dullness present. Breathing was thoracic in character.

Yours truly,

The patient was driven to the infirmary, well packed with blankets and hot-water bottles, in an ambulance, for six miles.

On admission he was in good condition. Skin warm, pulse 75, of good volume. There was board-like rigidity of the whole abdomen.

Operation (five hours after perforation) showed a leaking ulcer on the gastric side of the pylorus. This was closed by pyloroplasty through the ulcer.

GALL STONES AND CHOLECYSTITIS.

It is not sufficiently recognized that if a gall stone suddenly blocks the neck of an infected gall bladder, conditions of great danger may quickly develop. A distended inflamed gall bladder in the course of twenty-four hours may become gangrenous, and the patient seriously ill. Unless quickly relieved by operation, rupture of the gall bladder allows of the discharge of its infected contents into the peritoneum, and peritonitis, likely to be fatal, results. Diagnosis is based upon the previous history of gall-stone attacks, but chiefly on the discovery of a hard, tender rounded lump in the position of the gall bladder under the right costal margin. [A coloured drawing of a gangrenous gall bladder with stone impacted in its neck was shown.]

OVARIAN CYST WITH TWISTED PEDICLE.

In women this is a common cause of an abdominal emergency. If the tumour has been previously recognized, or is evident on examination, and a sudden attack of abdominal pain has developed with tenderness and enlargement of the lump, it is probably due to torsion of the pedicle of an ovarian cyst. In the majority of instances a serious attack has been preceded by one or more minor seizures—which have left the patient apparently little the worse. In serious attacks the vascular supply of the tumour is interfered with, and gangrene of the tumour and peritonitis are the terminal results. [A drawing of an ovarian cyst with twisted pedicle was exhibited.]

ECTOPIC GESTATION.

Another serious abdominal emergency peculiar to women is caused by intra-abdominal haemorrhage, due to rupture of an ectopic gestation.

In the most dangerous variety of this lesion diagnosis has to be made chiefly upon the signs of internal haemorrhage. An early impregnated ovum has eroded through the Fallopian tube, usually close to the uterus, and such furious haemorrhage follows that life can only be saved by operation.

In the more common variety one or two periods have been missed and the patient suspects that she is pregnant. The first warning that all is not right arises from a sudden attack of pain, which may be repeated once or more before the final rupture (Case IV). This pain is probably due to colic of the tube in its endeavour to expel the impregnated ovum. At this stage vaginal and bimanual examination can discover the distended tube at one side of or behind a uterus which offers evidences of pregnancy, and examination of the breasts may support this suggestion. If left alone the patient is suddenly overwhelmed by pain, and the symptoms of internal haemorrhage more or less quickly develop. In many the loss of blood has been so great as to allow of an additional sign—shifting dullness in the flanks—to be discoverable before operation.

Some of the most wonderful resurrections due to surgery have been obtained by operation in these cases, and death following it has been rare, except when the patient was already moribund before the abdomen was opened.

CASE IV.—Ruptured Fallopian Pregnancy.

This married woman, aged 29, was admitted January 14th, 1920. Four days before she was suddenly seized with pain in the lower part of the belly, "worse than labour pain." She felt at once very ill, the pain "went up to her heart," and made her feel as if she would die. She vomited several times.

On admission she was very ill, pale and anaemic, with dirty tongue, and was complaining chiefly of rectal tenesmus. (This is a common symptom in ectopic gestation.) There was a tender lump in the right iliac region with some rigidity.

Previous History.—Two children, elder 10, younger 8 years. Never well after last labour. Menstruation irregular, seldom a week without haemorrhage till four months ago. Then haemorrhage ceased for five weeks. Two months ago she thought she must be pregnant because her breasts swelled and milk came from them. A month before admission she had an attack of pain which lasted for two or three hours and made her feel faint. The pain was accompanied by free haemorrhage and the discharge of a "skin." This pain had been repeated several times before the final attack (examination then would have discovered a tubal swelling). She had consulted three doctors, but was not examined till her final seizure, when the condition was correctly interpreted.

On opening the abdomen, it was found to be distended with blood and clot, and there was a pregnancy in the right Fallopian tube.

REFERENCE.

¹ See Triple Syndrome in Abdominal Emergencies, BRITISH MEDICAL JOURNAL, January 3rd, 1914.

REMARKS ON THE ASSOCIATION OF AORTIC ENDOCARDITIS AND AORTITIS.

BY

J. E. MACILWAINE, M.D., B.Sc., D.P.H.,

PHYSICIAN IN CHARGE OF OUT-PATIENTS, ROYAL VICTORIA HOSPITAL, BELFAST.

In bringing forward this subject my object is to allow the members of the Branch to see two morbid specimens which we have been recently fortunate enough to secure. I would like to acknowledge my indebtedness to Dr. Boyd Campbell who has been associated with me in studying these cases, and who has taken the notes and made all the blood examinations.

For a considerable period it has been my experience to see a number of genuine endocarditic lesions of the aortic valve where clinical evidence seemed to point to the fact that the myocardium was not the important consideration in prognosis and treatment. I have had the advantage of making an electro-cardiographic examination in practically all the cardiac patients I examined, and this method added in some cases a considerable weight of evidence for the conviction that myocardial change was not in itself at all sufficient to endorse the grave prognosis which evidently the general condition of the patient demanded.

After reading Sir Clifford Allbutt's wonderful contribution to cardiac literature in his work on arterial disease,¹ it

* Read before the Belfast Branch of the British Medical Association on February 12th, 1920.

was borne in upon me that in all cases of aortic endocarditis we must think primarily of what is the condition of the aorta itself.

To speak broadly, aortic endocarditis can be divided into two etiological groups—namely, the syphilitic and the rheumatic. In a very exhaustive examination of men entering military service some observers came definitely to the conclusion that the commonest cause of the endocarditis in these recruits was the rheumatic virus.² On the other hand, experience of a considerable amount of work amongst the pensioners leads to the conclusion that the syphilitic virus is the principal etiological factor. This is a matter of the very greatest practical importance, and, fortunately, to-day we are in a position to consider it fully, and are able to arrive at a perfectly fair decision as to the etiology of most cases of aortic endocarditis.

I show specimens from two men who both exhibited the classical clinical appearance of aortic regurgitation. The first man died through the rupture of an acute aortic ulcer into the trachea, while the second died after a period of cardiac incompetence which lasted for a considerable time. These two cases have occurred in a series of twelve pensioners in hospital who suffered from aortic endocarditis with aortic reflux.

CASE I.

In the first man the cardiac condition was not in itself at all alarming, but the man's symptoms were persistent and severe. So much was this the case that the question of syphilitic aortitis was discussed, and a Wassermann reaction, which proved to be strongly positive, was obtained. This patient was in hospital for some months and underwent a course of mercurial inunction, with potassium iodide internally. He seemed improved, and had been able quietly to go about while in hospital; he was discharged as unfit for work. The symptoms persisted, and he was readmitted to hospital with hæmoptysis and dyspnoea, with severe constant pain behind the manubrium sterni and between the shoulders. He died suddenly, after a couple of days in hospital, coughing up a huge amount of blood. The necropsy showed the usual syphilitic aortitis with a considerable amount of inflammatory adhesion around the aorta, which was definitely dilated and closely bound down to the trachea. A large atheromatous ulcer had eaten its way through, with a considerable amount of cicatrization round it, into the trachea. Unfortunately, no x-ray plate of this man's chest was taken before he left hospital, but an ordinary careful clinical examination had not led us to suspect aneurysm, nor, indeed, was the *post-mortem* finding more than a dilatation of the aorta in its ascending and transverse portions. With the experience of this case we began, as a routine, to have a Wassermann reaction taken in all aortic cases, and so far, I think, there has only been one which gave a negative result.

CASE II.

The second case, in which we were enabled to obtain a *post-mortem* examination, was a pensioner, aged 40, who gave a definite history of specific infection twenty years previously. Clinically he presented symptoms very suggestive of acute aortic inflammation. There was well-marked aortic reflux with apparent mitral incompetence, and the general appearance of the patient was rather like septic endocarditis or general septic intoxication. The main symptoms complained of were increasing dyspnoea, pain upon effort behind the manubrium sterni; associated with this was an oedema of the lower extremities. The condition of the patient was evidently very grave, and we suspected, on account of the pain, and dyspnoea even at rest in bed, that there was some involvement of the aorta, as the Wassermann reaction was definitely positive. When we endeavoured to get an x-ray picture of the arch of the aorta it was discovered that the patient had a German machine-gun bullet in the thorax above the level of the arch of the aorta, between the oesophagus and the trachea. This bullet had nothing whatever to do with the man's general condition, as *post-mortem*, it was found not to be surrounded by any marked inflammatory reaction. Its presence there, however, when it became known to the patient, had a most disquieting effect. In fact, Dr. Robb, who was in charge of the patient, said "He never looked up after that bullet was discovered." Unfortunately, the patient's cardio-vascular condition was such that there was not much hope of improvement from the start. To this patient we gave small doses of novarsenobion, and at first there appeared to be definite evidence of improvement. However, the patient died before he had had a lengthy course—died rather suddenly of, apparently, complete failure of compensation. The necropsy in this case showed, in the first place, general evidence of old-standing syphilitic infection, and, in the heart, marked fatty degeneration; the aorta presented the picture of syphilitic aortitis, a type in which we have an acute inflammatory condition of the great vessel. The aortic valve shows chronic syphilitic endocarditis without any recent change. The inflammation, which is hæmorrhagic in parts, is most marked in the ascending portion of the vessel, and this is a usual occurrence. This patient was, unfortunately, too ill to be taken to the Royal Victoria Hospital for cardiographic examination.

It is a common experience to see heart patients who have previously had a syphilitic infection, and who have usually come in consultation on account of pain—either true anginal attacks, or pain which occurs on effort behind the manubrium sterni, and which may be referred from there into the back, between the shoulders, or into one special shoulder, or sometimes up into the neck, sometimes into the arms, etc. It is extremely difficult in some of these cases to say whether true angina pectoris should be considered to be present, or, where the pain is not of the classical description of "breast pang," some other place in the classification must be sought for it. Some of these patients present definite myocardial change, as evidenced by clinical examination by the electro-cardiograph. Many, of course, present the ordinary clinical features of aortic reflux with occasional associated stenosis, but the one common ground is the symptomatology of retrosternal pain and dyspnoea upon effort. I have one patient before my mind in whom these attacks of pain were so frequent that in walking a distance of half a mile on the level he would have to stop as many as half a dozen times. In this patient no cardio-vascular change of any description could be detected on physical examination, nor was the mechanism of the beat in any way altered in the electro-cardiogram. The blood pressure was not excessively high—about 160. In this patient, on blood examination, the Wassermann reaction was positive. There was no question as to the genuine history of this retrosternal pain, and the strange fact was that this patient, once he got started on a Peace Day celebration march, was able to do the whole march of about five miles on an excessively hot day. I have no doubt his pain is due to aortitis on a syphilitic basis.

The diagnosis of aortitis must in the main depend upon the symptoms. It is possible, by means of x-ray examination, in most cases to show a general enlargement of the aorta. It is occasionally possible to hazard the opinion that in these patients there is more dullness behind the manubrium sterni than is normal. Well-marked pulsation in the episternal notch, tracheal tugging, and an occasional alteration in voice should be sought for. I would urge, however, as a matter of deduction, that if a patient with the signs of aortic endocarditis develops definite symptoms of pain, and dyspnoea, we may conclude that the aorta is often more to blame in the circulatory decompensation which results than the actual myocardium. It is well known to every clinical observer that the vast majority of patients who suffer from aortic regurgitation are free from pain for years. Therefore we might argue that it is only when the aortitis becomes marked that its pain becomes prominent.

The pathology of aortitis is of a chronic inflammatory type—that is to say, the aorta loses its resiliency. It requires but little consideration to appreciate fully the fact that, while the first *vis a tergo* in the circulation is the contraction of the heart muscle, the second *vis a tergo*—if one might so term it—is due to the resilience of the aorta, which takes up the shock of the force of impact the cardiac muscle gives to the blood, and returns this force in the form of the pulse wave transmitted over the arterial system. This mechanical necessity for the resiliency of the aorta has only to be considered in order that we may realize what a chronic inflammation of this thick elastic tube must mean. The source of this retrosternal pain would seem to be undeniably the aorta, and the cause the chronic inflammation that is present in it.

The prognosis in these cases of aortitis seems to me to be uniformly unfavourable. Certainly our experience, since we began regularly to do the Wassermann reaction, would point to the fact that the old clinical rule stands, that "Where symptoms begin to develop in an aortic lesion there is usually rapid decline of the circulatory efficiency." As already stated in the opening part of this paper, the myocardial condition does not seem to be a very great element in actual prognosis. In the first case cited, we were definitely certain, from the clinical side, that the myocardium was efficient, with a small aortic lesion, and this was borne out by *post-mortem* examination. The prognosis in the second case depended entirely upon the symptoms of the persistent severe pain and dyspnoea, with the fact that the general appearance of the patient was septic, and he had a positive Wassermann reaction.

With regard to treatment, in the first few cases we adopted mercurial inunction and large doses of potassium iodide, but for the past six months, in all the cardiac cases

with a positive Wassermann reaction we have endeavoured to improve the condition by intravenous injection of small doses of novarsenobillon. It would appear that there is some improvement possible, but unfortunately the Wassermann reaction persists in spite of this treatment. It is too early for us to state definitely whether we shall be inclined, as the result of this treatment, to alter the very unfavourable prognosis that we are inclined to form in aortitis.

REFERENCES.

- ¹ Allbutt, *Diseases of the Arteries, including Angina Pectoris*.
² A Collective Investigation of Ten Thousand Recruits with Doubtful Heart Conditions. *BRITISH MEDICAL JOURNAL*, May 18th, 1918, p. 556; September 7th, 1918, p. 248; April 26th, 1919, p. 510; May 3rd, 1919, p. 544.

TREATMENT OF CEREBRO-SPINAL FEVER BY MONOTYPICAL SERUM.

BY

W. T. MUNRO, M.D., CH.B., D.P.H.,
 DISTRICT LABORATORY, COLCHESTER.

I VENTURE to bring to notice a digest of twelve consecutive cases of cerebro-spinal fever which have come to my notice, and to point out in particular that no case has died where it was possible to treat by monotypical serum.

The work of Dr. M. H. Gordon, C.M.G., who was in charge of the Central Cerebro-spinal Fever Laboratory, has demonstrated that 98 per cent. of cases of cerebro-spinal meningitis serologically fall into one of four types.

Those of us who were engaged in the work at out stations were supplied with agglutinating serums to the four types. It was a relatively easy matter to determine the type.

A growth on tryptic agar was obtained from the cerebro-spinal fluid, emulsified, heated to 65° C. to destroy the autolysin, and tested against the four types of agglutinating serums in dilutions of 1 in 50, 1 in 100, and 1 in 200; 0.5 c.cm. serum was mixed with 0.5 c.cm. emulsion of cocci, and the final dilutions then were 1 in 100, 1 in 200, and 1 in 400. Clear and absolute agglutination could usually be readily seen at the end of eighteen hours, and thus the case was typed. For treatment pooled serum containing 50 per cent. of antibodies to Type 1, and 50 per cent. of antibodies to Type 2 was issued, and in addition monotypical serum to be used after the type was determined.

It might be argued that pooled serum ought to contain antibodies to all the types, but typing of the cases as they came showed something like 40 per cent. to be Type 1, 42 per cent. to be Type 2, 16 per cent. to be Type 3, and 2 per cent. to be Type 4.

It will be at once seen that to add antibodies of Type 3 and Type 4 to such a pooled serum would reduce its value by 50 per cent. to 82 per cent. of the total cases. These serums were prepared under the direction of Dr. Gordon, and were tested both as regards their antimicrobial potency and their anti-endotoxic values.

Of the 12 cases which came to my notice, 8 recovered, and 4 died. The deaths all occurred in soldiers; two of them were dead before they were seen by me, and the other two comatose.

On July 11th, 1918, I was called to a military hospital to see a man who had been newly admitted. I saw him at once, but he had just died. The striking feature was a deep plum-coloured rash over the thighs, legs, arms, neck, cheeks, and even to the tip of the nose. The deep mottled colour and confluent nature of this rash marked it out for special notice. Lumbar puncture after death yielded a turbid fluid full of polymorphs and Gram-negative diplococci, and most of the diplococci were extracellular. The growth which was obtained proved to be Type 2.

The second case seen after death was from the same unit. A medical officer who came to see me later on the same day said that a man had been admitted to the same military hospital at 4 p.m. the day before, and had died at 4 o'clock that morning. I saw the body in the mortuary at 11.30 a.m. There was here, too, a deep plum-coloured rash, and puncture seven and a half hours after death yielded a growth which proved to be meningococcus Type 2. The fluid was turbid and full of polymorphs, and contained many Gram-negative diplococci, mainly extracellular.

I was not able to get any good histories of the illnesses of these two unfortunate cases. They were no doubt missed as cerebro-spinal fever cases owing to the number of men being dealt with in the July epidemic of influenza.

One of them had been ill from July 2nd to 5th with influenza, and one returning from leave had made a long railway journey on July 7th. These two men were

stationed at a brigade training school. There were 56 other men there, and 6 of them were found to have meningococci in the posterior nasopharynx. With the two fatal cases this gives a carrier rate of 14 per cent. (8 out of 58). All the carriers proved to be Type 2.

The third death occurred in a man sent a thirty mile motor journey to a military hospital as "mental." He was seen just before death, when there was a deep mottled rash over arms and legs. Lumbar puncture at noon yielded a very turbid fluid full of polymorphs and Gram-negative diplococci. Pooled serum was given at the puncture, but he never rallied, and died a few hours later.

The fourth case was similar. He died three hours after being first seen. Both of these cases yielded Type 2 growths.

The feature common to them all was a deep mottled rash, and indeed I had begun to look on these rashes as being Type 2 cases, and I risked the type in the last case and gave Type 2 serum on the first and only puncture, and not a pooled serum. The second feature was the very great number of extracellular diplococci in the fluid.

In Case III no contacts were examined by me, and in Case IV, among 24 contacts 4 were positive, so that with the case there were 5 carriers among 25 men, giving the carrier rate of 20 per cent. Necropsies were not obtained.

Reviewing those cases, all were Type 2. Two were dead before any specific treatment was asked for, the second two were moribund when first seen, and died within four hours of lumbar puncture—that is, they were dead before the type could be determined. The age of three of the cases was 18 years, and one (Case III) was 37 years of age.

All the cases showed the usual signs and symptoms of the disease. There were eight recoveries. One was a woman aged 49, and one a boy aged 11. The others were soldiers, four of whom were 18 or 19 years of age, one was 24 years, and the last, a discharged soldier, 34 years of age. In all the cerebro-spinal fluid was turbid, with polymorphs and Gram-negative diplococci, mainly intracellular, but in one patient, whose condition was very critical for some days, the fluid showed many extracellular diplococci.

Seven of the cases showed slight rashes, mainly petechial, and only at points of pressure. Five of the cases gave growths which proved to be Type 1, two proved to be Type 2, one of these showed many extracellular organisms, and one case (the boy) was Type 3.

Causation.

Of the eight patients that recovered, three had suffered from influenza and were recovering when they took ill. Three had had long railway journeys before becoming ill. Of these, one left Aldershot on March 6th, 1919, and became ill on March 8th, 1919; a second, who left Portsmouth on February 12th, 1919, became ill February 15th, 1919; a third left France May 11th, 1919, and became ill three days after reaching home. This is to be compared with Case II among the deaths, who left his home, London district, July 7th, 1918, and became ill on July 10th, 1918.

No depressing factors such as T.A.B. inoculation or vaccination could be traced as having any connexion with the onset of the illness. Four only of the cases had less than six months' service and two of these were the two deaths first recorded. There was no overcrowding.

Examination of Contacts.

In two of the cases no contacts were examined by me. In the case of the woman who recovered, all the contacts examined proved negative with one exception. The patient was Type 1, and the contact was Type 2, but it was subsequently ascertained that this carrier had nursed a patient of Type 2 who died at Ipswich.

This patient could have hardly infected others, as she occupied a room to herself in a good-sized house, but it is noteworthy that a pure growth of Type 1 coccus was obtained from her nasopharynx. Of the five remaining cases, one slept in a six-roomed empty house with thirty other men. There were six carriers besides himself, all Type 1, a carrier rate of 23 per cent. A second, who occupied a billet with two other men, showed one carrier, that is, two out of three men were carriers. No others were found in the company. In the case of the third, the patient who took ill after coming from Aldershot, two out of the sixteen men examined were carriers, but they were Type 1 and the case was Type 2. This man could scarcely have infected the unit in two days; and the two carriers were accidental discoveries. The fourth case lived in barracks with thirty other men; four carriers were found, three of whom corresponded in type with the patient's coccus, therefore there were four serologically similar types