II.—Hernia through the Oesophageal Orifice of the Diaphragm.

As may be seen from Fig. 1, there was only one specimen in the series of 34 which showed a hernia at the oesophageal orifice. This specimen is in the museum of Guy's Hospital, and shows the greater part of the stomach in the right pleural cavity. Mr. Lawford Knaggs has described several cases; Mr. W. A. E. Waller has recently placed a remarkable one on record in a young man of 19; Dr. Barclay-Smith found the condition associated with great dilatation of the sigmoid colon. Mr. Rigby and the writer have recorded a case in which a partial hernia of the stomach through the oesophageal orifice was associated with enteroptosis.

III.—Herniae through the Right Leaflet of the Diaphragm.

As shown in Fig. 1, there were only two cases of hernia through the right leaflet—one in a child of 4 months, where a process of the liver, about the size of a cricket ball, occupied a sac formed from the expanded central tendon; and another, also in a child, where a process of the liver lay free in the floor of the right pleural cavity. There can be little doubt that both of these were congenital in origin and were produced by an abnormal development of the liver within the septum transversum of the embryo. Professor Arthur Thomson has recorded a similar occurrence in the rabbit, the abnormal process of liver resembling an accessory heart.

IV.—Hernia into the Pericardium.

There were three cases of hernia into the pericardium through a perforation of the central tendon of the diaphragm. In the case of a man of 50 a process of omentum occupied the orifice; in a man of 47 a process of liver formed the extruded part. In neither of these cases was there a hernial sac, but in the third case, in a child at birth, the process of liver was enclosed in a sac formed by the central tendon. It is possible that the two cases in adults were the result of a former injury. Elder and Postlethwaite report a case where the lobe of a malformed liver occupied a diverticulum of the right central tendon, the diverticulum reaching the level of the second rib.

V.—Hernia through the Left Dome of the Diaphragm.

Herniae through the left pleuro-peritoneal passage are four times more common than through the right, and the same statement holds true as regards the various forms of hernia which occur at the domes and sterno-costal parts of the diaphragm. As may be seen from Fig. 1, herniae occur at two sites on the left dome—an anterior or sterno-costal site, and a posterior or central site. In the present series of 34 cases there were 5 at the sterno-costal site and 2 at the central. Of the sterno-costal form all were in adults varying in age from 19 years to 50, and in every case there was a history of a former injury by which the abdomen had been suddenly and forcibly compressed. There was no specimen of hernia at this site in a newly-born child. Cases of hernia in adults as the result of a former injury have been recorded by Mr. Lawford Knaggs, Professor A. M. Paterson, Mr. J. P. Roughton, and Mr. Don. In the two central cases the abdominal contents were enclosed by a sac formed from the extended diaphragm in one case (a child which lived forty-three hours), and in the other (a child at birth) the orifice in the diaphragm was free, and there was no sac. Both of these cases were clearly of congenital origin.

SUMMARY.

Herniae through the diaphragm fall into two groups, the congenital and the acquired, the former being to the latter in the present series of 34 cases as 26 : 8. The congenital herniae are chiefly those which occur at the unclosed pleuro-peritoneal passages (21 cases), the others being formed (5 cases) by developmental extrusions of the abdominal viscera, chiefly liver, through the septum transversum. In a certain proportion of congenital cases it appears possible to adopt surgical measures for the cure of the condition.

I should like to avail myself of this opportunity of expressing my indebtedness to the curators of the various hospital museums in London for the facilities given me of examining the valuable specimens under their care.

BIBLIOGRAPHY.

- Ballantyne, J. W.: *Manual of Antenatal Pathology*, 1904, vol. ii, p. 477.
 Berry, R. J.: *BRITISH MEDICAL JOURNAL*, 1906, ii, December 22nd.
 Cautley, E.: *Lancet*, 1905, ii, p. 1472.
 Barclay-Smith, E.: *Journ. Anat. and Physiol.*, 1898, xxxii, p. 341.
 Don, A.: *Edin. Med. Journ.*, Nov., 1908.
 Elder and Postlethwaite: *Lancet*, 1908, i, p. 1073.
 Gladstone, R. J.: *Archives of Middlesex Hospital*, 1908, vol. xii.
 Keith, Arthur: The Nature of the Mammalian Diaphragm and Pleural Cavities, *Journ. Anat. and Physiol.*, 1905, xxxix, p. 244.
 Keith and Rigby: *Lancet*, 1903, i, p. 639.
 Knaggs, Lawford: *Lancet*, 1904, ii, p. 358.
 Paillard, G.: *Les variétés anatomiques de la hernie diaphragmatique congénitale*, 64 pp.; Paris, 1903.
 Paterson, A. M.: *BRITISH MEDICAL JOURNAL*, 1888, ii, p. 1207. *Proc. Anat. Soc. Great Britain and Ireland*, 1895, p. xxi; 1900, p. lii.
 Robinson, A.: *Proc. Anat. Soc. Great Britain and Ireland*, 1900, May, p. xxix (in *Journ. Anat. and Physiol.*, vol. xxxiv).
 Roughton, J. P.: *BRITISH MEDICAL JOURNAL*, 1906, ii, December 22nd.
 Thomson, Arthur: *Proc. Anat. Soc. Great Britain and Ireland*, 1901, May, p. xvi.
 Waller, W. A. E.: *Lancet*, 1910, ii, p. 1135.
 Williams, H. W.: *Lancet*, 1908, i, p. 931.
 Williams, Penrose: *Lancet*, June 8th, 1907.

TYPHOID PERFORATIONS AND PERFORATIONS OF THE GALL BLADDER.

By GEORGE E. ARMSTRONG, M.D.,

PROFESSOR OF SURGERY AND CLINICAL SURGERY, MCGILL UNIVERSITY;
 SURGEON TO THE MONTREAL GENERAL HOSPITAL; CONSULTING
 SURGEON TO THE WESTERN HOSPITAL AND TO THE
 PROTESTANT HOSPITAL FOR THE INSANE, VERDUN.

PERFORATION of the intestine in typhoid fever demands our most careful study because of its frequency and danger. Physicians and surgeons are together bound to do their utmost to lower the high mortality-rate that obtains at present in this the most lethal of all the complications of typhoid.

Osler says that 1 out of every 3 deaths occurring during typhoid fever is due to perforation, and this estimate of its frequency is confirmed by the studies of the late J. Allan Scott. If the same percentage of deaths occurs in Canada as are estimated by Brown and Taylor to obtain in the United States, we have 1,875 deaths each year in our Dominion from typhoid perforation.

The locus is generally in the terminal two feet of the ileum, but, of course, it may occur at any part of the ileum, jejunum, large intestine, or appendix. The pathogenesis of typhoid perforation is well known. The coagulation-necrosis of the lymphoid tissue opposite the mesenteric attachment, where the blood supply is poor, has been clearly described by many writers.

The problem before our profession is to lower the mortality rate; therefore, the first question is, What are the causes of the high death-rate in typhoid perforation?

1. The condition of the patient at the time of its occurrence. Towards the removal of this cause we can do little, although it adds greatly to the difficulties of recognition.

2. The difficulties in diagnosis. This is almost the whole question at the present time. Once perforation has occurred, we may agree pretty well on the remedy to be applied and on most of the details of carrying that remedy into effect; but how to make the diagnosis—that is the question above all others in typhoid perforation.

To begin with, let us clear the ground of one or two erroneous ideas. We must not expect perforation of the intestine in typhoid to be announced by definite and distinct symptoms. So long as we look for the same abrupt onset that takes place when a stomach or appendix ruptures in a patient who is in full health at the time, and for symptoms that are unmistakable, so long we may go on as we have been doing, and make our diagnosis when it is too late to be of benefit to the patient.

My second point is that we must clearly appreciate the difference between perforation and peritonitis. The time has passed when we could say that perforation could only be recognized by the resulting peritonitis. A third-year student can diagnose peritonitis; the master should be able to diagnose perforation.

It is generally a good rule to doubt a diagnosis when the symptoms lack in harmony, but in typhoid perforation the absence of harmony is a diagnostic point in favour of perforation. The change for the worse in the patient is often out of all proportion to the abdominal symptoms. The first symptom is pain, and, as a rule, it comes on suddenly. Abdominal pain is the danger signal, and should always receive immediate attention. In rare cases the pain comes

on gradually; this is the exception, however. It is commonly referred to the lower abdomen, often to the right side, but may be referred to the umbilical or epigastric regions. Tenderness is usually present, and at first is localized. The initial pain and localized tenderness are the most important indications of perforation, and should be considered and differentiated from the more widely diffused pain and more generalized tenderness that appear later, and indicate the presence of peritonitis.

Rigidity and muscle spasm are now to be looked for. Greater resistance of one rectus muscle is significant. The difficulty is that typhoid patients often complain of the abdominal pain, and are generally more or less tender in the right iliac region. A differential point is that the pain and tenderness of perforation are associated with an altered condition of the patient. The nurse or the friends generally notice that something has occurred. The expression is different. There may be a clammy perspiration. If the pain, tenderness, and rigidity are accompanied by nausea or vomiting, as is the case in a considerable percentage of cases, their importance as a diagnostic sign is greatly enhanced.

One should not conclude that there is no tenderness present without a rectal examination. In some cases the perforated loop lies low down in the pelvis. The patient may then not complain much of pressure upon the abdomen, but forward pressure of the finger in the rectum will at once cause him to cry out. It is in these cases that complaint may be made of frequent and painful micturition.

Later on, when the infection of the peritoneum has become diffused, and peritonitis has developed, careful inspection may note lessened abdominal movement during respiration, particularly of the lower abdomen and in men. The absence of liver dullness is only of value when the abdomen is quite flat, and especially if its presence has been previously noted. Alteration in the pulse, temperature, and respiration may be confirmatory, but it is often remarkable how little they are disturbed. The blood pressure is sometimes increased. I have not found the presence or absence of leucocytosis a symptom upon which much reliance can be placed. It may be present without perforation and absent when perforation is present.

None of these symptoms are new. They are mentioned in all textbooks. The point is to be on the alert, and not to allow them to pass unnoticed.

There were treated in the Montreal General Hospital from 1897 to May, 1910, 2,051 cases of typhoid fever. During this period there occurred 93 cases of perforation. In this series, then, perforation occurred in 4.53 per cent. of the cases. In addition, I operated upon three cases of typhoid perforation in another hospital and upon one case in a private house; of these four, three recovered. We have, then, 97 perforations, of which 78 were operated upon, and 24, or 30.76 per cent., recovered.

If we now examine the statistics for the various years, we find that the percentage of recoveries has steadily increased. During the year 1909 there were 16 perforations; 9 were operated upon, and 9, or 56.25 per cent., recovered. Our figures for the year 1909 and four months of 1910, or up to May last, are the best of all. During this period there were 22 perforations, 19 of which were operated upon, with 9, or 47.36 per cent., recoveries. In this series of 22 perforations there were two very promising cases lost, apparently, by delay in obtaining consent after the perforation was diagnosed.

Annual deaths in Canada from typhoid fever ...	1,875
Montreal General Hospital, 1897 to May, 1910:	
Cases of typhoid ...	2,051
Cases of typhoid with perforation ...	93 (4.5%)
Cases of perforation in Montreal General Hospital	93
Author's cases outside hospital ...	4
Total cases of perforation ...	97
Cases submitted to operation ...	78
Cases not operated on ...	19
Recoveries after operation ...	24
Percentage recovery (24 out of 78) ...	30.7

<i>Recent Results.</i>	
Perforations, January 1st, 1909, to May, 1910 ...	22
Submitted to operation ...	19
Recoveries ...	9
Percentage recovery (9 out of 19) ...	47.3

<i>Type of Fever in which Perforation Occurred.</i>	
Severe ...	50
Moderately severe ...	20
Mild ...	1
Ambulatory ...	6
Type not described ...	16
	93

Associated with severe haemorrhage ...	26
Multiple perforation—6 cases ... (per cent.)	7.7

Secondary operation for secondary perforation in 4 cases: Recovery ...	1
--	---

The increased percentage of recoveries is, no doubt, in part due to the character of the epidemic, but chiefly to earlier diagnosis and earlier closure of the opening. The earlier diagnosis has been due largely to the development of the idea among the resident hospital staff that it is a serious reflection on their skill and professional attainments to fail to recognize a typhoid perforation, no matter how slow and chronic may be its appearance. The final step towards lessening the mortality-rate in typhoid perforation is the recognition by every one of us that it is a serious and humiliating oversight to fail to recognize a perforation when it occurs.

The difficulties are greatly increased by the dull and apathetic state so common in typhoid, but these difficulties should only stimulate us to greater watchfulness.

I think it may be said that the diagnosis is, in many cases, to a certain degree uncertain. For this reason, in all cases in which we think a perforation is present, but where there is a reasonable doubt, an exploratory incision is to be recommended. I have twice opened the abdomen without finding any perforation. So far as I could observe, no harm resulted. This exploratory incision, undertaken after due and careful consideration, may sometimes save a life.

While it is quite true that the earlier the perforation is closed the more limited is the infection and the better the prognosis, yet some of our cases have been operated upon and have recovered when the condition, because of delay and advanced peritonitis, has seemed almost hopeless. Our experience in these cases would indicate that we are not justified in withholding the only chance there is unless they are really *in extremis*.

It may be said, I think, that if perforation occurs into the free peritoneal cavity the patient cannot recover unless the opening is closed. The only perforations that do not prove fatal unless closed by the surgeon are those occurring in the large bowel and between the layers of the mesentery. It has been demonstrated at the operating table that a small perforation in the small intestine may be temporarily closed by omentum, but that such cases would have recovered if they had been left alone is open to very serious doubt.

I have opened one abscess in the loin—post-typhoid—that seemed clearly to have been secondary to typhoid perforation of the colon.

If now we analyse our cases we find that perforation is more common in the severe forms of typhoid. Fifty of the perforations have occurred in cases described as severe, twenty in moderately severe cases, and one in a mild case. Six perforations occurred in ambulatory cases, and in one of these it seemed highly probable that perforation had occurred sixty hours before admission to the hospital. Strange to say, this man recovered.

Of the 97 cases in which perforation occurred, only 78 were submitted to operation. Nineteen cases were not operated on for various reasons, the chief being failure to obtain consent from the patient, parents or guardians, the desperate condition of the patient, and non-recognition of the lesion.

In several instances the patient's chances have been lessened by the delay in getting consent. One conspicuous example was a man, a patient in the Typhoid Emergency Hospital. The perforation was promptly recognized, and the patient sent to the Montreal General Hospital. He then refused operation, and insisted upon being removed to his home in a distant part of the city. At his home better counsels prevailed, and he was brought back to the hospital. Two long rides in the ambulance and a delay of twelve hours changed an early promising case into an all but hopeless one. These delays from failure to obtain consent are of too frequent occurrence, one reason probably being that we do not promise enough

and one of my objects in this paper is to show that our extreme pessimism is wrong, unscientific, and not sustained by our later experience. Surely, if 50 per cent. of these desperate and otherwise hopeless cases can be saved, our pessimism should give way to a good, vigorous optimism.

Twenty-six of our cases of perforation were associated with haemorrhage. Among these are not included instances of small blood-stained stools but only those cases in which the quantity of blood lost amounted to several ounces. As a class they are unfavourable. Three of them, however, recovered.

In six cases, or 7.7 per cent., more than one perforation was found at the time of operation. The perforations were generally not far removed from each other.

In four cases a second operation was undertaken for second perforation. One of these recovered. In two, recovery would have followed the second operation had not a third perforation occurred—in one case seven days after the second operation and in the other twenty-one days after the second operation. In one case two operations were performed. At the first operation one perforation was closed. At the second operation two perforations were closed. The child finally died, and at the autopsy the lower two feet of the ileum were necrotic, twelve perforations being counted.

Haemorrhage and perforation occurring subsequent to the operation for closure must be placed among the causes contributing to the high death-rate. Can they be prevented?

This raises one of the questions of technique. Is it enough to simply close the perforation found? What other procedure may be adopted?

It has been my practice to close the opening in the simplest way, that is, by one or two sutures through all coats, and a row of Lambert sutures placed transversely at right angles to the long axis of the bowel. I then infold in a similar manner with a row of Lambert sutures all the ulcers found to have a thin, suspicious, necrotic-looking base. Two alternatives suggest themselves, and have been carried out in a few of our cases. In two the suspicious portion of gut was resected with end-to-end anastomosis. They both died. In two the suspicious-looking gut was delivered, and cared for outside. The gut was wrapped in gauze, and allowed to rest on the abdominal wall. These two, after doing well for a time, eventually succumbed, one of them from pneumonia.

Resection should probably be reserved for special cases in which the danger of further perforations over a considerable section of intestines seems imminent, and the patient's general condition warrants undertaking an operation that requires a little more time and a little more shock. I have not seen a report of any series of cases treated by resection, but I am under the impression, from conversations held with surgeons, that resections in typhoid perforations have not been followed by any improvement in the mortality-rate. I believe, however, that it should be carried out under favourable circumstances and in selected cases.

The variety of anaesthesia to be employed is a question of more than passing interest.

We will all, I think, agree that all preparations, including the wash-up, should be done before any general anaesthesia is used.

Local anaesthesia possesses many advantages, and should be used whenever possible. In the case of foreigners who cannot understand our language, and in children, it is difficult, but it undoubtedly should be the method of choice. I must say, however, that most of my cases have been operated upon under general anaesthesia, and I cannot say that I have seen much harm from it. On the contrary, these cases are almost all surprisingly well during the ensuing twenty-four or forty-eight hours. If the patients are vomiting, this is a very forcible reason for using local anaesthesia, as we thus lessen the danger of an aspiration pneumonia.

There is one point to be remembered when operating for typhoid perforation under local anaesthesia, and that is, that when the abdomen is opened there may not at first seem to be any indication that a perforation has occurred. No gas or fluids may escape, and, so far as can be seen through a small incision, the peritoneum and intestine may present a nearly normal appearance. This occurred to me

on one occasion. The symptoms in this case had been unusually well defined, and that there was a perforation seemed almost beyond question. On pulling up the lower end of the ileum the patient complained so loudly that I gave a little ether. Then, when the ileum was finally delivered, a small perforation was found nicely closed by adherent omentum. If this had been overlooked, doubtless a leakage and peritonitis would have followed soon afterwards.

The subject is one of great interest. The death-rate must necessarily remain high, but watchfulness, exploratory incisions in doubtful cases, and prompt closure should save a goodly number of typhoid perforations.

I take this opportunity to report a case of typhoid perforation of the gall bladder.

The patient was a little girl 10 years of age, referred to me by Dr. Molson. The typhoid had been of moderate severity. At the end of the third week, after three days of normal temperature, she complained of pain in the right hypochondrium. The following day an ovoid swelling appeared in the situation of the gall bladder. It felt as large as a turkey's egg, was exquisitely tender, and moved up and down during respiration. It was accompanied by a rise of temperature and acceleration of pulse. Under local applications the tumour became less tender and perhaps a little smaller. There was no jaundice. On the fourth day of the tumour another accession of pain decided me to operate. I opened directly into an abscess cavity, and in the abscess lay the gall bladder perforated in three places. The walls were everywhere necrotic. There were no stones. I did a cholecystectomy, and the little girl made a smooth and uneventful recovery.

The following is the report of Dr. Wolbach, pathologist in chief to the hospital:

The specimen is slightly enlarged, thin-walled gall bladder 10 by 4.5 cm. in its greatest diameters. The outer surface is much congested and presents numerous yellowish raised areas, in a few of which there are perforations extending through the wall. The mucosa is dark red, and contains, in addition to the perforations, many ulcers, 0.1 to 0.5 cm. in diameter. The large ulcers have yellow necrotic bases with undetermined edges. The thickened floor and the surrounding puriform infiltration of these ulcers produce the swelling noted on the external surface of the gall bladder.

Incision through the serous coat into the wall of the gall bladder in the neighbourhood of the perforations and ulcerations shows extensive oedema and puriform infiltration into the surrounding tissues.

Kaiserling specimen preserved.

Microscopic Examination.

The epithelium of the mucous membrane is nearly wholly absent; a few islands of epithelium represent deeper parts of folds in the mucosa. The line of the basement membrane is everywhere intact, except over the ulcers; so that the general loss of epithelium is probably post-operative. Throughout the sections examined there are evidences of oedema and many small areas of haemorrhage.

In thickened parts of the wall there is marked infiltration with lymphoid and plasma cells, and large mononuclear cells, many of which are finely vacuolated (compound granular cells).

Young connective tissue cells are present throughout the sections where there is oedema. The large mononuclear cells (endothelial cells) are occasionally grouped in large numbers about vessels, in lymphatics, and in areas of haemorrhage.

The above changes are most marked in the outer half of the gall-bladder wall—that is, subserous tissues and the elevated areas noted in the gross specimen are due to necrotic areas in this region, surrounded by granulation tissue. Near one large necrotic area there is a large vein with an early organized and canalized thrombus.

In the areas of surrounding necrotic tissue there are many polymorphonuclear leucocytes, but even here the number is not greatly in excess of the large mononuclear cells.

A few short bacilli are present in necrotic areas (methylene blue stain). No other bacteria can be found.

The blood vessels everywhere are engorged, but there is no evidence of recent thrombus formation.

SUMMARY.

Necrosis of gall-bladder wall following marked infiltration of the outer half with lymphoid, plasma, and endothelial cells, in which respect it differs from an ordinary suppurative process. The extensive oedema is possibly due to thrombosis of vessels, though only a single organized thrombus could be found. The character of the cellular reaction supports the theory of a direct infection of the gall-bladder wall by the typhoid bacillus as against pyogenic infection, or infection secondary to a cholecystitis.

The only demonstrable bacteria in the lesions are bacilli consistent in morphology with the typhoid bacillus.

The route of infection could be either through the blood stream or through lymphatics from infected lymph nodes which may be incorporated in the wall of the gall bladder.