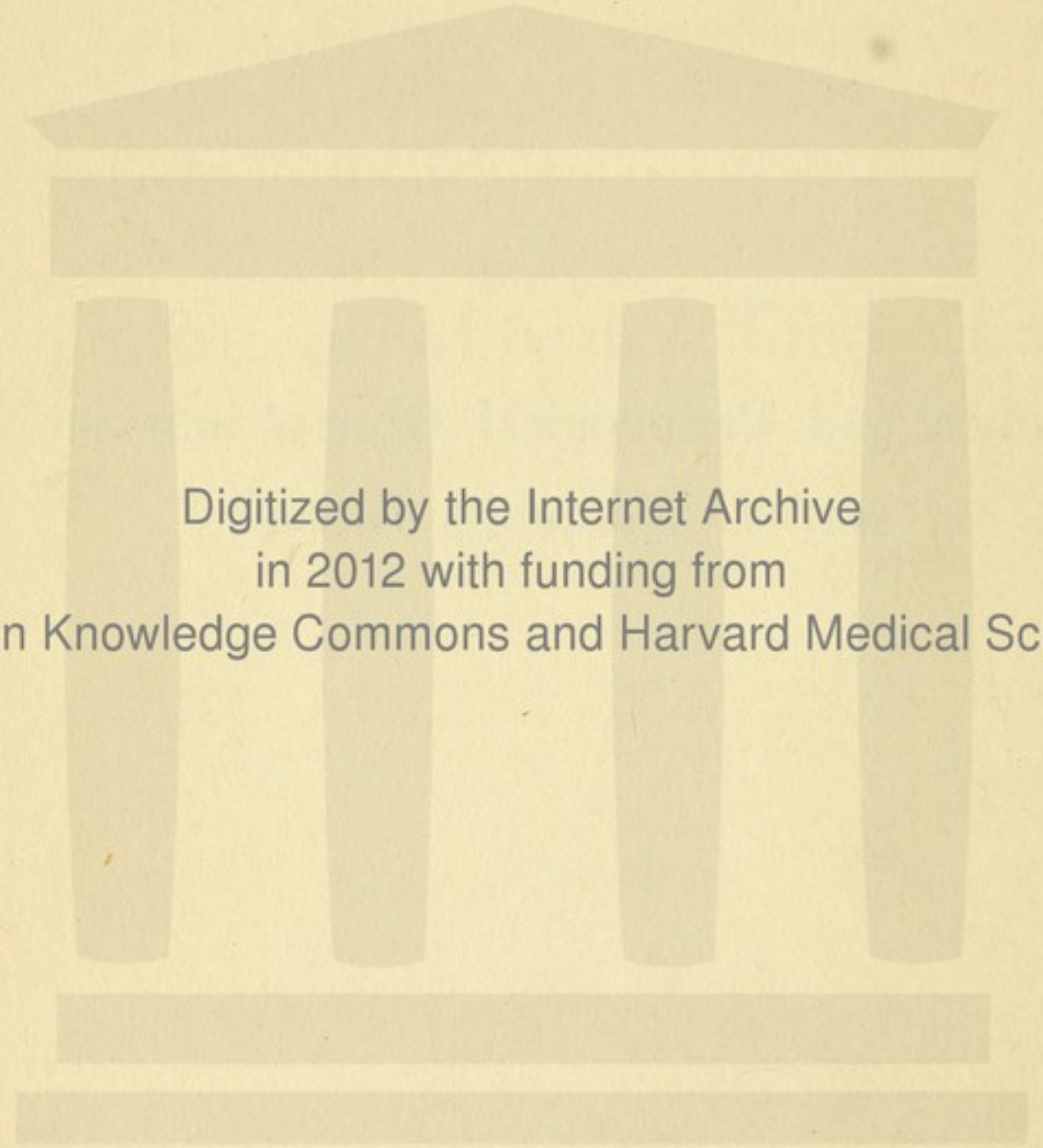
Clinical and pathological observations on acute abdominal diseases due to conditions of the alimentary tract and the uniformity of their origin : the Erasmus Wilson Lectures 1904 / by Edred M. Corner.

Clinical and Pathological Observations on Acute Abdominal Diseases



Clinical and Pathological Observations on Acute Abdominal Diseases

DUE TO CONDITIONS OF THE ALIMENTARY TRACT AND THE UNIFORMITY OF THEIR ORIGIN

The Erasmus Wilson Lectures 1904

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Contents.

I. PREFACE

As the material collected for the Erasmus Wilson Lectures delivered at the Royal College of Surgeons, March, 1904, was a great deal too large for the purpose and also for publication in the Journals, it was decided that it should be brought out in book form, without any separation of the various sections. The Lectureship being a pathological one, stress was laid chiefly on that side of the subject. Also a pathological title, "Acute Infective Gangrenous Processes (Necroses) of the Alimentary Tract," was selected. When it was decided that the whole of the material should be published together, it was felt that a change in the title must be made. This is the reason why the present one was adopted.

The main object of the lectures is to direct attention to the identity of the pathological changes concerned in the production of all acute perforative and gangrenous processes of the alimentary tract, such as appendicitis, perforations of gastric and duodenal and other ulcers, cholecystitis and acute diseases of the gall bladder, gangrene in strangulated herniae, diverticulitis or the inflammation of Meckel's diverticulum, and perhaps in acute pancreatitis and liver abscess. More than this, an endeavour will be made to point out a probable and uniform bacterial origin for all these in the action of pyogenic organisms, streptococci and staphylococci; thus bringing the pathology of acute surgical diseases of the alimentary tract into harmony, if not absolute identity, with the well-known abscess formation and suppuration in other parts of the body.

126, Harley Street, W.
April, 1904.

II. INTRODUCTION

THE name gangrene is one which has been used for many, many years. But very few know that it is derived from a Greek word, *χραίν*, to gnaw. From the original meaning, therefore, it is most applicable to such processes as ulceration, suppuration, phagedaena, etc., that is to say, the death of small particles or molecules. Unfortunately it has become usual to apply it to the "death of visible portions of tissue," and in consequence, is understood to imply the death of large parts such as limbs or parts of limbs. The common meaning of the word then is very different from its original significance. Gangrene frequently covers also the putrefactive processes which occur after the death of the tissues. The old names of sphacelation and sphacelus refer to the separation of the dead from the living tissues, i.e., the process of sloughing and the slough of dead tissue cast off.

The mere fact that a tissue is possessed of life presupposes that it must die. When the whole body dies, the process is called death, not gangrene. When a part of the body dies, it should be called local death. Adopting the customary use of Greek phraseology, these two catastrophes may be called general or total necrosis, and local or partial necrosis. This is the more exact nomenclature, though it will be a long time before the word gangrene is expunged from our text-books. Death or necrosis is the natural end of all living tissue. When it occurs as a general dissolution of the body, the death of the parts is bound up with that of the whole, and no attention is paid to them. But

the untimely death of a part does attract attention, no matter when and where it is. Our text-books take ample consideration of the death or gangrene of limbs. I shall endeavour to bring together examples of the untimely death or necrosis of different parts of the alimentary canal, and to show that the tissues there are subject to the same laws of life and death as are those elsewhere ; the differences in the results being due to the differences in the situations in which the necrosis occurs. Broadly speaking, there are two forms of death, one of which is followed by drying and mummification and the other by wetness and decomposition, which, when local, are erroneously termed dry and moist gangrenes (or gnawings). The dryness and moistness are important physical conditions. But a much more important distinction is between the aseptic, relatively so to judge from results, and the septic necrosis or death, the former being dry and the latter moist. I have termed the septic process "infective necrosis."

In his presidential address to the Clinical Society of London in 1896, Dr. Buzzard,[1] among other things, discusses whether "those who follow the profession of medicine have no right to speak of their work as Science." I should like to point out that through the practice of the art of surgery, the surgeon has opportunities of making many scientific observations on man. The subject to which I wish to direct your attention in these lectures is that of acute infective necrosis or gangrene of the various regions of the alimentary tract. The life or death of a part of the intestinal canal is one of the greatest importance, especially in abdominal surgery, and through the action of the swift process, already mentioned,

[1] *Clin. Soc. Trans.* 1896, xxix. p. liii.

are brought about some of the most terrible tragedies in the annals of life.

Every surgeon, in the practice of his profession as an operator, is in a position to make observations on his cases, in a way, similar to those which the physiologist and pathologist make on the animals upon which they experiment. Be it, however, clearly understood that the similarities rest on the opportunities and not on the methods. For the surgeon does his work to cure or alleviate the condition for which his services are required; whilst the others plan their experiments without regard to any benefit which might accrue to the subject thereof. The results of the latter, however valuable, are applied by inference, or indirectly, to man; those of the former have the great advantage of being directly applicable to the subject on which they were made. In many cases, the results are those of formal procedures, and may be of small value, but no one can fail to add to this, especially through the medium of abdominal work, in the way of meeting the unexpected, being the victim of circumstances, mishaps, misfortunes, and last—but by no means least—mistakes.

The evidence upon which these lectures are based has been derived in such ways from the study of man during the last five years. And I would like to acknowledge, yet again, my thanks to the staff of St. Thomas' Hospital for the opportunities of observation so freely accorded me. And rather with the object of emphasizing this line of research, which is the peculiar property of the surgeon, I have abandoned entirely the more popular method of experimentation, and performed no operations on animals. But, please understand clearly that I in no way underestimate the very great value of such methods of research.

III. THE CLINICAL IMPORTANCE OF ACUTE INFECTIVE NECROSIS IN ACUTE ABDOMINAL CASES

THE death or gangrene of part of the alimentary tract forms one of the most important fields which, by reason of the recent rapid advances, abdominal surgery has taken within its scope. The resulting catastrophes, such as appendicitis, perforations of ulcers, etc., have long been known, as well as the enormous mortality which accompanies them. With increase of our knowledge of the pathology of these cases and improvements in operative technique, the death-rate has of later years been very considerably lowered. Unfortunately the clinical signs, by means of which we must try to recognize the internal progress of the pathological process, are, by their very nature, relatively coarse, late and inexact means of gaining that urgently required information, the diagnosis, the possession of which is of vital importance to the patient. The manifestations of an acute abdominal disease are, unfortunately, often very obscure as to their origin. And much valuable time is lost before the decision is made as to whether the abdomen must be opened or not. The tremendous change in the mortality made by such delay is not even yet quite fully appreciated.

All acute abdominal crises begin similarly, with shock, collapse, vomiting, etc., which symptoms have been called by Sir Frederick Treves, peritonism. This may be termed the first stage. Later, in the second stage, the diffuseness of the manifestations ceases, and localizing signs and symptoms appear. But it must be remembered that though the outward appearances become more local, the inward infection may be spreading. At this period a diagnosis may be made. Later, as the infection becomes more and more diffuse, the

signs again become more and more general; and the diagnosis is again impossible. It is, in consequence, a difficult task to obtain figures which can give anything like a truthful idea of the practical importance and frequency of the disease which is the subject of these lectures.

To help to meet this disadvantage, during the years 1900, 1901 and 1902 a careful record was kept of every acute abdominal case which was admitted to St. Thomas' Hospital. In this way, I have obtained a series of 456 consecutive cases, quite unique in that the diagnosis was verified in every instance by operation or by post-mortem examination. Practically everyone came either under the personal observation of my colleague, Dr. A. E. Russell, or myself. It is our good fortune to be able to place before the medical profession a large and quite unique series of acute and consecutive abdominal cases spread over a period of three years. The distribution of these cases may be briefly reproduced :—

I. TABLE OF ACUTE ABDOMINAL CASES AT ST. THOMAS' HOSPITAL, 1900, 1901, 1902.

TABLE I.

Appendicitis	37 per cent.
Intestinal obstruction (other than intussusception)	24 „ „
Intussusceptions	15 „ „
Perforations	11 „ „
Gynaecological	6 „ „
Peritonitis of unknown origin	2 „ „
The rest	5 „ „

Cases of gangrene or necrosis of the alimentary tract vary considerably in their severity. That is to say, acute, subacute, and more or less chronic clinical varieties are recognizable. As it is to the first of these classes to which I wish mainly to direct attention, an attempt has been made to

estimate the relative practical importance of the pathological factor of acute necrosis in these acute abdominal cases. Briefly, it forms, as a primary process such as in appendicitis, etc., fifty per cent. of these cases, and is also secondarily associated with many of the other causes. The large percentage of fifty indicates the great frequency of this necrobiotic process as a primary change, and its occurrence in instances of acute abdominal disease as a secondary condition still further emphasizes the vast importance of the process. A number of cases are lost and the patients die in consequence of this secondary change ; and in abdominal work one must guard against its occurrence just as surely as we try to cure the primary lesion. It is desirable that the full practical import be recognized before the more scientific aspect of the question is touched upon. As a primary affection it may be seen in the perforation of gastric, duodenal and other ulcers, of carcinomatous ulcers, appendicitis and diverticulitis, etc. ; as a secondary process in strangulated herniae, volvulus, intussusception, strangulation by bands, perforation of the caecum in intestinal obstruction lower down in the large intestine, etc.

To proceed to its more strict definition, the process may be defined as one of death or necrosis of the tissues of the gut, dependent in the acute cases mainly on the action of septic organisms, and in subacute or chronic ones mainly upon the interference with the blood supply. In the former, the acute, the organisms act primarily on the tissues and through inflammation of the vessels and subsequent thrombosis secondarily cause interference with the blood stream ; in the latter, the subacute, the blood supply may be interrupted primarily and then the organisms act. Mr. Watson Cheyne, in his excellent article in Sir Frederick Treves' *System of Surgery*, divides the causes of gangrene or necrosis into three

classes—the direct, the indirect, and the specific. But, unfortunately, such a classification can only stand so long as inquiries into it are not prolonged or pertinent. For then is seen the impossibility of discriminating between the three divisions. I would like to suggest that, broadly speaking, two extremes exist :—

(1) In this class, the necrosis or death is due to the deprivation of the tissue of blood, i.e., aseptic death.

(2) In this class, the necrosis is due to the action of micro-organisms, i.e., septic death.

In between these two extremes there exists every variety of mean or intermixture of the factors. The above may, from the point of view of the tissues, be termed the local factors. But the general condition of the individual will take a more or less important secondary part in the process of local death. The flora of the intestines and the fluid of the peritoneal cavity are such that pure anaemic or dry gangrene are unknown there. The omnipresence of moisture and organisms render the gangrenes, in this situation, of the moist variety. In reality, the tissues of the alimentary tract are subject to the same laws of life and death as are those of the rest of the body, and it is my intention to apply to them the above general principles. The branch of inquiry with which this first lecture will deal is the relationship between the integrity of the gut and interference with its blood supply. Upon this subject surgery has a good deal to say.

The great clinical difference which is noticed between gangrene or necrosis due to cutting off of the blood supply, and that due to activity of bacteria, is the different time taken by these factors to produce the local death. The former is

slow in action, the latter very rapid ; and between these two extremes there are all varieties of the intermixtures of the factors, which are, naturally, by far the most common.

IV. THE EMBOLISM AND THROMBOSIS OF THE VISCERAL VESSELS OF THE ABDOMEN.

Embolism and thrombosis of the abdominal visceral vessels forms an obvious line of inquiry into the nature of the relationship which exists between the integrity of the intestinal wall and interference with the blood supply. At the first, it was my intention to serve up this subject completely with its literature. But, the number of the cases recorded of late has increased so wonderfully, that the time has now passed when this would be worth the print and paper, except in a monograph on the subject. Dr. Moyes (1) in 1880 reported on 64 cases ; Watson (2) in 1894 collected 27 more cases ; Deckart (3) in 1902, 66 cases ; Koebling (4) also in 1902, 64 cases ; but the most extensive is by Gallavardin (5), in 1901, whose zeal enabled him to unearth 126 cases ! Since these authors, a large number of further examples have been published and a few unpublished ones referred to, so that the actual number will be about 150.

With the exception of a few instances, such as those of Elliott (6), Gordon (7) and Anfrecht (8), every reported case has been fatal. Early surgical intervention has saved these. Almost all have been reported merely for their pathological interest. It is in the history of most diseases that they are first published as unique pathological curiosities. Slowly pathological interest leads to the awakening

and kindling of clinical acumen, and lo ! it is found to be quite a common disease. Appendicitis forms the most exemplary instance of this history.

Looking amongst the reported cases of embolism and thrombosis, it is noticed that some died within twenty-four hours of the onset of symptoms, some on the second day, others on the third, fourth, and so on. Yet one of Elliott's (9) died as late as the twelfth day, one of Kiliani's (10) on the fifteenth day, Munro's (11) on the twenty-second day. Some have presented clinical pictures of acute abdominal disease, some of subacute, and some of almost chronic. And yet, in all these varieties, the diagnosis of embolism has been rendered certain by the autopsy ! One is forced to wonder if spontaneous recovery is not possible in the face of such variations in the length of the illness.

In most cases of embolism the clot comes from the left auricle and takes its origin in mitral disease. And I remember well, when clerking for the late Dr. Miller Ord at St. Thomas' Hospital, a little boy with mitral disease who had a sharp attack of abdominal pain and was ill for a few days. The diagnosis made was that of appendicitis, but can it have been that a small embolus lodged in a branch of one of the mesenteric arteries ?

Before inquiring into what the pathologists may have to say to this suggestion, it is desirable that some account be given of the important points in the work already done. Blockage of the arteries is more common than that of the veins in the proportion of about five to one. Involvement of the superior mesenteric artery bears as high a proportion as about forty to one of the inferior mesenteric artery. This is extremely interesting, and the anatomical reasons given for this, like so many anatomical reasons for surgical events, lack perspective. The real explanation

seems to me to be, that the statements are made only from a study of fatal cases. For the appreciation of this line of argument, it is desirable that a brief description of the anatomical distribution of these mesenteric arteries should be given. The superior mesenteric artery springs from the front part of the aorta about half an inch below the origin of the coeliac axis and opposite the first lumbar vertebra. It then passes obliquely downwards and forwards in front of the left renal vein, the lower part of the head of the pancreas, and the third or transverse part of the duodenum ; opposite the latter it enters the mesentery of the small intestine, along the root of which it continues to descend giving off branches into the mesentery to the small intestine from its left and convex side, and retroperitoneally to the large intestine on its right or concave side. The branches to the small intestine divide and form four or five arterial arcades, the last of which is within half an inch of the bowel, and from it straight branches are distributed to the walls of the gut.

These straight branches within the gut wall communicate freely with the vessels of the segments above and below. Anatomically the small intestines have a wide and excellent blood supply, expressive of very many alternative routes. On the concave right side of the artery far fewer vessels are given off, thus giving to us an index or method of gauging the severity of the embolic lesion. The first artery to come off is the inferior pancreatico-duodenal, the next is a large branch, the middle colic, then the right colic and the ileocolic, and finally, the terminal branches to the caecum and appendix. The main artery as it proceeds diminishes fairly rapidly in size.

The embolus may be situated in the main trunk so as to block the inferior pancreatico-duodenal artery, or the

middle colic, or the right colic, or the ileocolic, or the terminal branches. And in each case a corresponding amount of small intestine will have its blood supply cut off on account of the involvement of the corresponding number of the vasa tenuis intestini. The superior mesenteric artery supplies with blood no less than about 25 feet of the length of intestine and is the sole artery to that region. The inferior mesenteric artery supplies the descending colon, iliac and pelvic colon by itself, but the rectum gets no fewer than three arteries, the superior rectal from the inferior mesenteric, the middle rectal from the anterior division of the internal iliac or its inferior vesical branch, the inferior rectal from the internal pudic branch of the same division of the internal iliac artery.[1] The duodenum gets its blood supply from two sources, namely, the hepatic branch of the coeliac axis and from the superior mesenteric artery. The stomach is supplied by the branches of the coeliac axis. The enormous importance of the integrity of the superior mesenteric as compared with that of the other visceral arteries is easily seen, as having manifold the largest and longest distribution it naturally happens, that if the main stem is occluded, there will be infinitely greater difficulty in the successful establishment of an anastomotic circulation, than there will be in the cases of the other visceral arteries which supply smaller lengths of bowel.

Anatomically, it is seen that Nature has been very prolific in supplying means for this end in the anastomotic arcades, etc. Physiologically, the case is not so clear. Litten (12) in 1875 showed that, in dogs, ligature of the stem of the superior mesenteric artery led to necrosis of the small intestine and

[1] The old name of "haemorrhoidal" for these arteries has been rejected in accordance with Professor Alexander Macalister's suggestion.

the death of the animal, whilst he also found that such was not the case when the coeliac or the inferior mesenteric artery was occluded. Consequently, he argued that, though anatomically not an end artery, the superior mesenteric was one functionally. Emil Fricker (13) of Berne, 1902, performed a number of ligations of arteries supplying the stomach and duodenum, entirely confirming Litten's results. Hence there seems reason to suspect that lesions of visceral arteries other than the superior mesenteric need not be fatal, and consequently, as our knowledge of the cases under discussion is based almost solely on post-mortem records, this artery will be found responsible in, to say the least, an undue proportion.

The next point which interests us is the evidence for the existence of an anastomotic circulation. Firstly, there is the length of time, which has elapsed after the lodgment of the embolus, at which gangrene has been found. Lothrop (14) records the absence of gangrene of the intestine after four and five days had elapsed since the onset of the illness, Osler (15) after seven days, Munro (16) after twenty-two days, and so on. Secondly, with an embolus in the main trunk of the superior mesenteric artery, practically the whole small gut and the large as far as the splenic flexure has been found gangrenous, yet with a precisely similarly situated lesion only a small segment of ileum has been necrotic after a history of several days' illness, Koster (17). Or, in thrombosis of the superor mesenteric vein, Bradford (18) found only eighteen inches of jejunum gangrenous on the fifteenth day. Or, in the case of occlusion of the main trunk of the inferior mesenteric artery only three to four inches of the descending colon were gangrenous on the twelfth day, Elliott (19). So that there is ample evidence to prove the existence of power to form an anastomotic circu-

lation, which will be successful if the area to be resupplied is not too great, as seems to be the case with blockage of the main trunk of the superior mesenteric artery, though not so when only a branch is involved. So my little boy with mitral disease, already mentioned, who had an attack of abdominal pain, may have had an embolus after all.

The ease or difficulty for the establishment of a collateral circulation is insufficient to account for all the variations found in these cases. Some light as to the possible cause is shown by extremely rapid instances which exhibit an acute infective necrosis or death of the bowel. For instance, Sievres (20) found incipient gangrene of the gut in a man of 56 years of age, who died within twenty-four hours of the occurrence of embolism of his superior mesenteric artery. Collier and Burgess (21) record a somewhat similar case. Dr. Frederick Taylor's (22) case died in thirty hours, and showed similar necrotic signs. And many other authors have found most extensive gangrene present by the second to the third day. These acute cases are in all probability due to the action of micro-organisms on the tissues of the bowel wall, especially that of pyogenic cocci, which have become active on account of the anaemic condition of the bowel, brought about by the interruption of the blood supply. Their absence, or better, comparative absence, may account for the subacute cases of longer clinical duration. As yet, however, no bacteriology worthy of the name has been done in these cases. Pilliett (23) has suggested that the thrombosis may have been due primarily to the action of organisms. Pake's (24) case, in which organisms were found in an antemortem clot, renders this suggestion probable. However, the majority of the cases are due to embolism, and not primary thrombosis.

The relationship of the sizes of the areas deprived of

their blood and the intensity or degree of the bacterial infection offers an explanation for all the varieties found in the clinical features already mentioned. But, that the bacterial agent is not always all important is shown by the fact that the part of the gut found gangrenous when the superior mesenteric artery is obstructed, is almost always that part of the ileum which is about the middle of the area to be resupplied with blood, and therefore the last which the anastomotic circulation will reach. And also, this part, the centre of the ileum, is not one of the great breeding places for organisms such as the caecum is, yet the former becomes gangrenous and the latter not. The formation of an anastomotic circulation is sometimes much impeded by the formation and the extension of thrombi, secondary to the embolism. When the occlusion has been slow and the "botanical" condition of the bowels good, the collatera circulation will be found to work admirably.

Having now illustrated the formation of an anastomotic circulation in obstruction to the visceral vessels, by means of the variations in the time elapsing before the occurrence of the gangrene, the extent of the gangrene, the situation of the gangrene, the immunity with lesions to visceral vessels other than to the widely distributed superior mesenteric artery, etc., some most remarkable cases are brought forward which illustrate the continuation of life after the total obliteration of the visceral arteries.

Virchow (25) has reported two cases in which he found, post mortem, that the main trunk of the superior mesenteric artery had been converted into an impervious fibrous cord. The intestines appeared normal. The clinical records of the previous illnesses are wanting. Similarly Tiedemann (26) has given an account of a kindred case. Professor Chiene (27) of Edinburgh reported fully a still more remark-

able case of a similar and more extensive condition. As the record is in a journal not often consulted by medical men, the account is briefly reproduced here. The specimen was found in the anatomy rooms in a female subject, who had died of " paralysis " at the age of 65. The aorta was extensively atheromatous. There was an aneurism on the abdominal aorta and the inferior mesenteric artery arose from the lower part of the sac. The coeliac axis arose in its usual position, but for its first half inch was converted into a fibrous cord. The branches beyond this point were fully injected with paint for the dissection, so that there had been established a perfect anastomotic circulation. The occlusion of the trunk of the axis must have robbed the stomach of its entire blood supply. Presumably, the process must have been very slow to enable an anastomotic circulation to be established without giving rise to any necrosis of the viscus. Both the superior and inferior mesenteric arteries were similarly converted into impervious fibrous tissue close to their origins, their branches being full of the injection of paint. This woman had had the most serious interference possible with the arterial blood supply of the whole of her alimentary tract, the process of occlusion going on presumably at the same time in each of the three vessels. Yet there were no changes in the intestines such as to call attention to them. Professor Chiene's account of the course of the anastomotic channels is as follows :—

" This case is probably the only recorded example of an obliteration of the three anterior visceral branches of the abdominal aorta at their origins, and of a consequent enlargement of the secondary anastomoses for the supply of the viscera ; the stomach, liver, spleen, pancreas and duodenum being supplied by the left lower intercostals, and from the left renal and suprarenal arteries, which inosculated

with the lumbar arteries of the same side; the remainder of the intestines receiving its supply from the internal iliacs through a large plexus surrounding the rectum, except the caecum and ascending colon, which received its blood supply from the last dorsal artery on the right side." This confirms the existence of a free communication between the visceral vessels insisted on by Sir William Turner (28) in 1863. Professor Chiene's case is much more extensive than either those of Virchow or Tiedemann, and therefore has been quoted to illustrate that a properly functional anastomotic blood supply can be constructed during the slow obliteration of the arteries which supply the alimentary tract from oesophagus to anus!

To sum up the chief points, which an examination of the records of examples of obstruction of the visceral vessels has shown, the following tables may be formed:—

EMBOLISM AND THROMBOSIS OF VISCERAL VESSELS

I. CLINICAL TABLE

1. Embolism is most common in men past middle life.
2. It is most common secondary to valvular disease of the heart, especially mitral disease.
3. The onset is generally sudden, with symptoms of peritonism.
4. In one class, the picture is one of intestinal obstruction, peritonitis due to appendicitis or the perforation of a gastric ulcer.
5. There is also another class in which blood is passed per rectum, and which signifies infraction of the bowel.
6. Subacute and chronic cases are seen as well as the most acute.
7. In some cases it is most probably spontaneously recoverable from, especially if only a small area of bowel is rendered bloodless.

II. PATHOLOGICAL TABLE

1. For a constant situation of the embolus in the superior mesenteric artery, as in the main trunk, all varieties of clinical cases have been reported, from the most acute and fulminating to

cases like Professor Chiene's, which recovered, apparently, without symptoms.

2. With a precisely similar lesion, some subjects die in 30 hours and some not for from 12 to 20 days.
3. Gangrene of the gut has been noticed to have occurred within 30 hours, and sometimes not after 22 days.
4. A collateral circulation can be formed in slowly developing cases of visceral vascular obstruction, e.g. Chiene's case. And even in more acute instances an attempt at a collateral circulation is made.
5. The superior mesenteric artery is almost invariably picked out for the site of the embolus in fatal cases. Possibly because it will always cause symptoms and almost always death, which need not occur with involvement of the inferior mesenteric and its branches, or those of the coeliac axis.
6. The explanation of the individual variations, clinical and pathological, seems to depend upon the pathogenicity of the bacteria present in the gut, as to the severity, rapidity, or other characters of the cases. On the bacteriology of this affection there is no work done.

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V. THROMBOSIS OF THE MESENTERIAL VESSELS IN STRANGULATED HERNIAE

The association of thrombosis of the mesenteric vessels with the strangulation of a hernia I have observed several times, and am also sure that it is not always a matter of great importance. Professor Kocher of Berne told me that he lays great stress on the presence or absence of pulsation in the arteries of the mesentery of a knuckle of strangulated gut, in judging whether or not that bowel can be returned to the abdomen. If the pulse is absent he prefers to resect the intestine rather than run any risk in returning it. Since then, I have made many examinations of such mesenteries, and have been frequently unable to find any pulsation, and yet no evil has resulted from the replacement of the bowel in the abdominal cavity. Presumably, therefore, occlusion of the vessels of a temporary nature is frequently present, and does not necessarily cause gangrene of the bowel. And again, resection of the gut is not necessarily needed when that arterial pulse is absent. Again, this thrombosis may be associated with perfectly recoverable bowel, as will be seen later. And there is still more to be learned.

In two or three instances, whilst doing a "herniotomy," the mesentery has been injured in the process. The bleeding vessels, once an artery of some size, and therefore of some importance, ligatured and the rent sutured. These cases did uniformly well. In one of them, in which the mesentery was noted to be loaded with fat, there was scarcely any bleeding. Thinking this over afterwards, it seemed that the vessels were thrombosed, so explaining the fact that no haemorrhage resulted from the tear. In all of these cases the rents were more or less perpendicular to the long axis of the bowel, the obliquity only involving a small length on a line parallel with the same axis. So that at the most, only half an inch of the bowel had its blood supply interfered with. The suggestion that, at least in the last case, the mesentery tore, and was not divided by the herniotomy knife, is strengthened by the following example, in which a previously thrombosed and fatty mesentery tore with the greatest ease.

A woman, C.M., aged 63, was admitted to St. Thomas' Hospital on July 27, 1902. She had known of the presence of a right-sided femoral hernia for a year, and it had now been strangulated for thirteen hours. The pain and the vomiting had been very severe. At the operation, the sac contained a little bloody fluid and a loop of small intestine, which, though a little dark in colour, was obviously returnable. Gimbernat's ligament was incised, and gentle traction applied to the bowel. But try as one would, the mesentery kept separating from the intestine, and in spite of great care and a second and freer "herniotomy," four and a half inches of bowel were denuded. There was no haemorrhage, the vessels being thrombosed. About a foot of the bowel was resected, and the ends united by a circular enterorraphy. A radical cure was then performed. Beyond the occurrence

of a haematoma in the situation from which the large fatty hernia sac had been removed, the convalescence was without incident.

This case definitely proves the existence of a thrombosis of the mesenteric vessels in a strangulation of medium severity and of thirteen hours' duration. But one of the most interesting points is that the bowel looked quite good, and if the mesentery had not been torn, would undoubtedly have been returned to the abdomen. If it had, what would have happened ? In the light of the above sections of this lecture, the patient might quite well have recovered, an anastomotic circulation becoming established ; or, on the other hand, the gut might have necrosed. The result would have depended on the pathogenicity of the organisms present, secondary extension of the thrombus, etc.

Since the above case, I have become cognizant of a paper by Thévenot and Patel, in the *Archives Provinciales de Chirurgie*, Paris, x, 1901, p. 340, on the tearing of the mesentery in strangulated herniae, of which, with some difficulty, I have succeeded in obtaining a copy. They report one case of their own, in which a successful resection of the bowel was performed in consequence of the tearing of the mesentery. The duration of the strangulation in a right femoral hernia before operation was twenty-four hours, and the bowel, small intestine, was apparently healthy. M. Dransart (*Thése de Lille*, 1896–1897, No. 36) has recorded two similar cases. M. Colle's unpublished case occurred in a man of sixty-six years of age, who had a right inguinal hernia, which had been strangulated only six hours. Thévenot and Patel associate the extreme friability with the fattiness of the mesentery, as was so in the case which I have recorded at length. M. Jaboulay (*Traité de Chirurgie, Le Dentu and Delbet*) regards it as due to congestion, oedema and in-

flammation. And I would further like to add, thrombosis of the vessels and the consequent weakening of the tissues.

As in my case, the gut in that of Thévenot and Patel looked quite healthy ; so did also that of M. Colle's. This is an extremely interesting point. And as far as one is able to judge, vascular thrombosis is far more frequent in strangulated herniae than is suspected, and the more commonly gives rise to no trouble whatsoever ; the most important proviso being the absence of septic organisms. These cases illustrate that the blood supply of small segments of the bowel can be completely cut off without necessarily producing sloughing of that tissue.

VI. WOUNDS AND SURGERY OF THE MESENTERY

The study of wounds, ruptures and surgical proceedings on the mesentery will assist in showing the relationship or degree of interdependence which exists between interference with the blood supply and the integrity of the tissues of the walls of the intestine. It has been held generally that the bowel which has been separated from its mesentery must die. Yet every surgeon must have seen cases in which the direct blood supply of a segment of bowel has been damaged, if not completely cut off, and yet little or nothing has resulted. In separating adhesions, as in pelvic surgery, it is by no means uncommon for part of the ileum to be denuded of its mesentery ; and I have seen no less than four inches so stripped, yet no " artificial anus " resulted, but merely a small, insignificant and temporary faecal fistula. Cantwell,

in the *Annals of Surgery*, 1899, ii. p. 597, says: "In my efforts to deliver the tumour from the abdomen (sarcoma of stomach) about four inches of small intestine was torn from its mesentery; this I attached as well as I could." The patient recovered without any untoward symptoms at all. For this reference I am indebted to Mr. H. A. T. Fairbank, Surgical Registrar of Charing Cross Hospital.

These instances show that bowel when stripped of its mesentery, and therefore temporarily deprived of its blood supply, can recover completely or partially. In appendix cases, with much matting from adhesions, I have seen parts of the ileum separated from its mesentery and yet neither necrosis nor faecal fistula have resulted.

Mr. Makins in his classical book on his *Surgical Experiences in South Africa*, says (p. 420): "It stands to reason, however, that injuries to the mesentery would be much more frequent proportionately to wounds of the gut than is the case in ruptures seen in civil practice, since the whole area of the mesentery is equally open to injury." It may be then surely concluded that in many cases the wounds in the mesentery do no harm to the bowel. Amyx's case, published in the *Medical Record of New York*, 1903, is an interesting example in which there were nineteen wounds of the gut and four lacerations of the mesentery. Part of the gut was excised and an anastomosis made, and the rest was sutured. The man recovered, showing that some wounds of the mesentery may do little or no harm to the bowel.

Mr. Bernard Pitts, in the St. Thomas' Hospital Reports for 1897, describes two cases of multiple rents of the mesentery, both being fatal before sufficient time had elapsed for gangrene of ordinary degree of severity to be found. He says (p. 103): "We know that each portion of the bowel

is dependent upon the mesentery belonging to it for its nutrition, so that in ruptured mesentery we have not only the danger of haemorrhage, but also the prospect of gangrene." A vertical or axial rent of the mesentery must be obviously of little importance as compared with a transverse one, i.e. one parallel to the direction of the bowel. The injury of a big vessel close to the root of the mesentery is more likely to be fatal than one close to the gut, on account of the larger area of bowel rendered bloodless in consequence. No matter where the vascular lesion is, anatomically a collateral circulation can be formed, and the all-important factor is the activity of the organisms within the bowels which may cause gangrene wherever or whatever the lesion may be. Vertical splits are made in mesenteries in exploring the pancreas, posterior wall of the stomach, von Hacker's gastrojejunostomy, etc., and with impunity.

In performing an anastomosis, especially with continuous sutures, numerous small masses of tissue must be deprived of their blood supply. Yet nothing of importance need happen, if—to put it popularly—the " bugs are favourable." Consider each little tuft of tissue as it is ligatured, it need not necrose or slough ! In performing an end to end anastomosis, circular enterorraphy, it is always emphasized that the weak spot in the suturing is at the mesenterial attachment to the bowel, to overcome which a rectangular stitch is sometimes recommended, which neatly and completely will cut off all the direct blood supply to the anastomosis. Yet no ill results need accrue. I believe that this teaching is untrue, and that all the stitches are subject to a like risk, which depends almost entirely on the action of microorganisms, that is to say, sepsis. An end to end anastomosis, in which the above mentioned mesenterial stitch is passed, is the example in which the blood supply

of the sutured portions is most effectually cut off. Yet these cases do very well.

Aldrich, in the *Annals of Surgery*, 1902, xxxv. p. 342, describes the case of a man, aged fifty-three, who fell on his buttocks and slowly got worse and worse until he died on the evening of the seventh day. Death was due to slow intraperitoneal haemorrhage from a ruptured mesenterial vessel, but there was no change in the bowel. Botesen, in an unconsultable journal of 1903, describes a case of recovery after ligature of a wounded mesenterial vein. Most surgeons will some time or other have ligatured such veins in the course of operations without any untoward results happening.

Numerous arteries of the stomach have been ligatured for haemorrhage from gastric ulcers without any result to the stomach or the haemorrhage. These cases prove that, on a small scale, the blood supply of the alimentary tract may be interfered with without necessarily causing the death of the part supplied.

Finally, two similar cases of injury to mesenterial vessels are recorded; in one gangrene of gut occurred, and in the other it did not.

Mr. Cosh, in the Medical and Surgical Reports of the Presbyterian Hospital, New York, records the case of a boy of ten, who fell from the window of a fifth storey to the ground, striking his belly on a bar. He was admitted to the hospital after the lapse of thirty hours, with temperature 100·5, pulse 124, respiration 42. On the third day laparotomy was performed and a large haematoma of the mesentery and a gangrenous coil of the jejunum found. Resection of the gut was followed by death. The gangrene presumably resulted from rupture of an artery within the mesentery and a collateral circulation could not be formed to supply the anaematised part before it occurred.

To show that necrosis of the bowel is not a necessary accompaniment of such a case, an example is recorded on which I operated on March 18, 1902. A boy of eleven was run over by a two-wheeled vehicle. There was a broad line of bruising running transversely across the back just above the level of the crista ilii, with a corresponding but less severe contusion on the front of the abdomen. There was no free fluid in the belly, no blood in the urine, etc. The temperature was 99 degrees, the pulse 100. Next day the temperature rose to 100·2 degrees, and the pulse to 112; the abdomen became tender, the right side dull and rigid. An incision was made through the right rectus muscle. The presenting coils of intestine showed one or two bruises. Beneath the peritoneum in the right iliac fossa was a large haematoma, which did not extend over the brim of the pelvis, but lifted up the caecum and the whole of the mesentery with the small intestines. The peritoneum, parietal and visceral, on the right side of the abdomen were infected and a search for a further lesion was made. In the concavity of the hepatic flexure of the colon, the bowel was partially torn through for one and a quarter inches in length; apparently the mucous membrane was incompletely torn through. The rent was sutured, and the abdomen washed out with saline solution. Nothing was done to the mesenterial haematoma, as there was perfect circulation of the blood in the small bowel so far as could be ascertained. There was a little fluid present, but no blood, in the peritoneal cavity. Healing resulted by first intention. Convalescence was retarded by an attack of bronchopneumonia. In this case, although the lesion was severe, there was no intestinal necrosis.

This section illustrates again the fact that a small portion of the bowel can have its blood supply cut off and not under-

go gangrene or necrosis. The last two cases possibly picture the results of a septic and a comparatively aseptic condition of the gut with a similar vascular lesion in each. In the first the bowel died, in the second it did not.

It is hoped that these sections have proved that the blood supply is not the most important factor in the production of gangrene and perforation of the gut, which it has been supposed to be. And it is further hoped that the following sections will prove the enormous rôle of bacteria in causing these local catastrophes.

VII. THE PHYSICAL PHYSIOLOGY OF THE ALIMENTARY TRACT AND APPENDICITIS.

Acute infective necrosis will be most familiar to you in appendicitis, so that the first problem which will be brought forward is, "Why is it that this site is so especially selected by the process?"

In order to understand this question clearly, a short discussion must be made into that much neglected region, the physics (not the chemistry) of digestion.

Food is first reduced to a more or less broken up and pulpy condition in the mouth, in which state it enters the stomach, where it is further fragmentated by the action of hydrochloric acid, pepsin, and churning muscular movements. The casein of our primitive natural food, milk, is precipitated and also fragmentated. The pylorus, the smallest portion of the alimentary canal, allows or should allow no large pieces to pass into the duodenum. The work of

the stomach may be regarded as chiefly, if not entirely, preparatory to chemical digestion and absorption, and to partake of the physical nature of fragmentating the food. The more perfect the work of the stomach, the more completely will the food be reduced to a molecular condition, and consequently, the more perfectly will it be adapted for the chemical action of the bile and pancreatic secretions. The twenty odd feet of the small intestine will be given over to these chemical changes and the absorption of the nutriment. The food has now reached the ileocaecal valve, and it is the duty of that sphincter only to allow water and indigestible débris to enter the caecum. Our ancestors had large caeca in which that material was dried by the absorption from it of water and possibly any nutritious matter left. Further, it is of obvious advantage to excrete solid and not liquid dejecta. To this end, amongst others, has been directed the secretion of mucus by the glands of the large intestine ; which structure has also been adapted for the end of passing onward solid or semi-solid faeces, the most obvious modification being the aggregation of the fibres of the longitudinal muscular coat into the three bands, the taeniae muscularis.

I should like to attract attention to the similarity in the physiology of the pylorus and the ileocaecal valve. The former is the portal between fragmentation on one hand and chemical digestion on the other ; the latter stands between chemical digestion and specialized absorption on the one hand, and mere straightforward absorption of water on the other. Professor Arthur Keith, on anatomical grounds, has pointed out in a very recent communication the homologies between the pylorus and the ileocaecal valve (*Proc. Anat. Soc.*, 1903).

But I wish further to direct your attention to the fluid

or semi-fluid material which is passed through the ileocaecal valve from the small intestine into the caecum. The latter is not adapted mechanically for passing on fluid by peristalsis ; it is too large and thin walled to contract efficiently, unless it is tightly distended like the bulb of a Higginson's syringe. Again, in man, gravity will work against it. As a result the faeces must remain in the caecum until the absorption of fluid and the deposition of mucus have rendered them of the proper consistency for transit along the large intestine. Hence, it is easy to see why the caecum is so often the most capacious part of the large bowel, and why at operations surgeons so frequently find it distended out of all proportion to the rest of the alimentary canal, the bowels being full of fluid.

Just as there is a natural " rest " for food in the stomach, so is there another for the products of digestion in the caecum, and in both, the contents are prepared for their course in the next region of the alimentary tract. To turn to the pathological significance of this pause or rest, it will be seen at once that, as a consequence, the caecum will be the breeding ground of bacteria, par excellence, of all parts of our intestine. The enumeration of the colonies from different parts of the alimentary tract by Cushing and Livingood, Lorrain Smith and others bear this out, and has proved that colonies of bacteria are more frequent by hundreds and thousands here than in any other part of the intestinal tract, even after a period of starvation. And it is only to be expected that this difference will show manifold increase when food is taken. It is now very easy to understand why Nature has given us so large a quantity of the protective lymphoid tissue in the ileocaecal region, to which the appendix is an intestinal " tonsil," an index of the fermentive processes which are going on within the caecum, a culture tube for

the bacteria therein contained. Hence the frequency of appendicitis.

Before proceeding to the pathology it must be repeated that the great clinical difference between gangrene due to interference with the blood supply and that due to the action of microorganisms, is the rate at which the necrosis progresses and the speed with which it occurs.

The existence of this faecal pause or rest in the caecum is exemplified not only by the frequency of inflammation and the invasion of the appendix by bacteria, but also by the frequency with which tuberculosis and actinomycosis of the alimentary tract affect this region. The occurrence of inflammation in the appendix will, almost of necessity, impair the efficiency of its muscular contractions or peristalsis to expel its contents, the organ, in consequence, becoming an inert culture tube for the organisms of the caecum. And hence, the extreme frequency of subacute or chronic appendicitis, a disease of infective origin.

Having pointed out the probable causes which predispose the appendix to frequent bacterial invasion, there is no need to enter at all into the various forms of appendicitis, except perhaps to point out that the action of microorganisms is in all probability at the root of all the attacks of inflammation of this organ. In cases in which the appendix has sloughed on account of an acute infective necrosis or gangrene, the part affected is almost invariably the terminal portion. The usual explanation given is that the tip has no mesentery, and therefore no direct blood supply. Though this is a factor, it must also be pointed out that the terminal part is that portion which is least efficiently emptied by peristalsis, and therefore that in which bacteria may grow undisturbed and also, presumably, the region most liable to be severely attacked by them. The vascular lesion or thrombosis must

be almost invariably secondary to the action of micro-organisms, bearing out what has been said in the previous lecture.

It does not often come within the surgeon's experience to have sufficient opportunities to estimate the approximate time required for the appendix to become perforated or gangrenous. Unfortunately, the cases are only recognized as requiring surgical aid *after* the gangrene has occurred, and not before. But I have had the opportunity to operate upon some acute cases within twenty-four to thirty-six hours after the onset of the illness, and have found the appendix perforated or gangrenous within that time, the shortness of which requires the virulent action of bacteria. And in two exemplary cases, the infection being from the lumen or interior, the whole mucous membrane was a necrotic slough within twenty-four hours of the onset, whilst the outer coats were as yet but little affected.

There are not many problems with regard to the pathological changes in the appendix which can be put before you in this course of lectures, as every severe attack is an acute infective necrosis. But the spreading of this necrosis is one which has never received sufficient attention as yet. The most instructive way, in which this subject can be introduced to you, is by means of that of intestinal suturing.

VIII. INTESTINAL SUTURING

It stands to reasonable explanation that surgeons should not have the opportunity of studying the spreading character of the disease, as their observations are largely confined to the time of operation and not afterwards, and their chief interest is naturally in the successful recovery of their case. As a consequence, the subject of the spread of the necrosis, which is always fatal, has never been adequately studied, and is now brought before you. In my paper in the St. Thomas' Hospital Reports, 1900, vol. xxix. p. 349, a case was quoted in which a boy of fourteen years of age was operated on for appendicitis with diffuse peritonitis, the gangrene or necrosis, which had started in the appendix, had extended on to the caput coli for about half an inch. This portion of the caecum was resected, apparently freely, and sutured. At the post mortem thirty hours later it was found that the infective necrosis had extended further up the caecum, and the stitches had cut through, being quite useless.

This was the first case that led me to believe that it was infection or sepsis which was the cause of the so-called leaking of an anastomosis. The stitching which unites two segments of the alimentary tract, of course, must be water-tight. But after this, those stitches must not be septic, whether infected from within the bowel or by the surgeon. The line of suturing which unites the mucous membrane will in almost every case become septic from and be discharged into the lumen of the bowel. But, it should last long enough to protect the second line of suturing, and so allow the outer coats of the gut to become firmly united before it gives way.

In doing an anastomosis do the surgeons see that the

edges of the mucous membrane are as nicely coapted as they do the skin edges ? Infection from the bowel must be almost universal in the stitches inserted in these coats. The function of the first line of sutures is to protect the second for a sufficient length of time to allow of firm union. Again, it does not appear to matter whether a continuous or interrupted suture is used, provided that the first line of suturing is water-tight. It is sepsis rather than imperfections in stitching which cause the failures of anastomosis. Continuous sutures have in their favour the fact that they are more quickly inserted and give more uniform and perfect coaption of surfaces. Whilst if either the continuous or the interrupted are septic, failure will result. The first row, uniting the mucous membranes, will in almost every case become infected and will be discharged sooner or later into the bowel.

In 1901, I successfully sutured a perforated duodenal ulcer in a man who died of intestinal obstruction six to seven months later. At the post mortem, I made a most careful examination and found only three or four stitches present ; whilst at the operation the ulcer had been sutured with three rows of four to six sutures in each (silk). Almost all of these had been shed !

As yet I have never had the opportunity of examining post-mortem cases of my own, in which an anastomosis had been made successfully. I am happy to say that the subjects are still alive. If one looks at the figures in the communication of Mr. C. A. Ballance and Mr. Walter Edmunds to the Pathological Society, 1896, several illustrations are given in which the first layer of sutures is being discharged into the interior of the bowel. Stitches in the alimentary tract are exactly the same as those elsewhere, say in a hernia, in that, if clean or aseptic they hold well, but if septic, as they are much more likely to be, failure will be the result.

IX. APPENDICITIS IN HERNIA SACS

Whilst examining with Mr. Spurrier the conditions under which the appendix may exist in a hernia sac, two or three cases were found in which that organ had become gangrenous and the necrotic process spread to and involved the caecum.[1]

X. APPENDICITIS AND FAECAL OR INTESTINAL FISTULAE

The necrotic or gangrenous process may spread to other structures than the caecum, as was discovered when an attempt was made to remove the appendix at the same time at which the abscess was opened. In sixteen cases in which it was removed in this way, opportunity was obtained for observation on the results of its infective necrosis on the contiguous viscera. The observations shed a very important light upon the origin and occurrence of faecal fistulae, and as they are the results of the extension of the acute infective necrotic process, notes of some of the cases are briefly reproduced. To begin with, four observations on cases of appendix abscess will be made.

Case 1. Appendicitis, local abscess, faecal fistula which closed spontaneously.

E.R., female, aged 18, was operated on September 1, 1901,

[1] St. Thomas' Hospital Reports, 1902.

for an appendix abscess of some age having withstood considerable medical treatment. When the abscess was opened the distal half of the appendix presented in the pus, having sloughed off. The proximal half was adherent to the ileum, the wall of which, at the point where the necrosis of the appendix had taken place, was also sloughing and rotten, so that any attempt to remove this proximal part of the appendix would have resulted in the complete tearing across of the ileum. The wound was packed with gauze and partially closed. A faecal fistula resulted, which healed spontaneously in ten days.

Case 2. Appendicitis, local abscess, faecal fistula and spontaneous closure.

A.R., female, aged 48, was operated upon on July 17, 1902, for appendix abscess. A similar necrotic portion of the ileum was found which led to the formation of a faecal fistula when the slough separated. Spontaneous closure occurred in a "few days."

Case 3. Appendicitis, abscess, necrosis of wall of caecum, faecal fistula and spontaneous closure.

A.D., female, aged 17. Operation July 14, 1902. The gangrenous appendix was found adherent to the wall of the caecum, which was also necrotic. This led to the formation of a faecal fistula which closed spontaneously.

Case 4. Appendicitis, abscess, necrosis of ileum, faecal fistula and spontaneous closure.

A.S., female, aged 16. Operation June 26, 1901. The perforated appendix was apposed to a piece of sloughy ileum. It is very noteworthy that the necrotic or gangrenous process was much more extensive in the ileum than it was in the fons et origo mali, the appendix. The fistula after "some time" closed spontaneously.

These four selected cases demonstrate clearly that the

necrotic process in the appendix does spread to the neighbouring coils of bowel, and if the above are of any indicative value, three times as frequently in the small bowel as in the caecum. These cases also suggest that the faecal odour, so often noted in the discharge from an appendix abscess about the second or third day, is due to the formation of a small faecal fistula probably in connexion with the ileum; the presence of such a fistula has been entirely overlooked until improvements of technique enabled us to explore abscesses without infecting the rest of the peritoneum.

A fifth case suggests a similar origin, i.e. small bowel, but as nothing actually was seen, it cannot be proved. The case is, however, well worthy of record for another reason.

Case 5. Appendicitis, abscess, a second abscess on the left side of the abdomen with resulting temporary faecal fistula.

This case was seen with Dr. Sayres, of Woodford. E.M., female, aged 16. Operation for appendix abscess, November, 1903. A fortnight later a second abscess was opened in the left iliac fossa, and a concretion removed from the nearly healed track of the first. Four days after the second operation and on the left side she developed a faecal fistula which healed spontaneously in a few days' time. This faecal fistula, in all probability, arose from the necrosis or gangrene of part of the wall of the small bowel in the second abscess.

Besides these cases I have had the good fortune to operate upon three subjects of persistent faecal fistula, which resulted from appendicitis. Brief notes are given of these.

Case 6. Appendicitis, abscess, persistent faecal fistula, second operation, closure of opening in the small bowel, appendicectomy. Recovery.

W.J.J., male, aet. 7. First operation for abscess, November 21, 1901. A necrotic coil of small bowel was adherent to the abdominal wall. Persistent faecal fistula,

probably fairly high up in the *small intestine*, as it caused much irritation and excoriation of the surrounding skin with loss of weight. March 2, 1902, the fistula was successfully excised and the bowel closed by an enteroplasty. Recovery.

Case 7. Appendicitis, abscess, persistent faecal fistula, second operation, closure of the small bowel and removal of the appendix. Recovery.

B.P., male, aet. 8. First operation, March 3, 1901, for an abscess. Persistent faecal fistula. Second operation, March 27, 1902. Fistula found to be connected with the *ileum* about eight inches from the caecum. Recovery uneventful.

Case 8. Tubercular appendicitis, abscess, persistent faecal fistula, second operation, closure of caecum, appendicectomy. Recovery.

W.P., male, aet. 26. Admitted to St. Thomas' Hospital for a faecal fistula. An abscess had been incised three months previously. Operation, March 27, 1902. The fistula communicated with the *caecum*, and had arisen from a tubercular appendicitis, which had involved the proximal part of the appendix and sloughed a hole into the caecum. Recovery, but death from acute pneumonic phthisis within a year.

Seven cases of faecal fistula have been actually examined, five were connected with the small bowel, two with the caecum. And a further one in all probability arose from sloughing of part of the wall of the small bowel. Not one arose from imperfect closure of an appendix stump, as is popularly supposed to be their origin, although sixteen appendices were successfully removed at the same time that the abscess was opened.

I would like, yet again, to insist on the great frequency of temporary faecal fistulae in connexion with the small

bowel in cases of suppuration about the appendix, and that these are due to an extension of the necrotic process, which began in the appendix, to contiguous coils of bowel.

Reference must be made to a case reported by Dr. O'Connor, of Buenos Ayres, in the *Lancet* of August 16, 1902. The case was one of recurrent appendix abscess, which led to the perforation and necrosis of five inches of an adjacent coil of ileum. Dr. O'Connor successfully resected and united the bowel.

Necrosis of structures other than the walls of the alimentary canal are beyond the scope of these lectures. But it serves as an excellent example, to illustrate the action of acute infective necrosis in the ulceration of an appendix abscess into the iliac arteries or veins. The harmony between the well-known process of abscess formation and suppuration on one hand and that of perforations of the appendix on the other is illustrated as well.

XI. THE FREQUENCY OF APPENDIX ABSCESSES AND THEIR RELATION TO FAECAL OR INTESTINAL FISTULAE.

Before leaving the subject of the formation and frequency of faecal fistula consequent upon appendicitis, reference must be made to a condition which arises from it as a corollary. The fistulae originate in local infective necrosis or sloughing of the wall of a contiguous coil of bowel, usually the small intestine. In the examples quoted, and others not specifically mentioned, the fistulous communi-

cation would have formed whether the abscess had been opened or not. Had the abscess not been opened the fistula would have been an internal and not an external one. In other words, the abscess would have discharged into the bowel. As I believe must be, every case of appendicitis is intimately dependent, in most instances primarily and in the others secondarily, on the action of septic micro-organisms. This being so, abscess formation must be common. Yet only eighteen per cent. of appendicitis cases had localized abscesses in a series at St. Thomas' Hospital.

The natural conclusion must be that these abscesses are often discharged into the bowel and are not noticed; whether this occurs viâ the appendix or by means of the formation of an internal faecal fistula cannot be said. Judging an appendix case by the ordinary clinical experience of events when sepsis is present, it may be affirmed that in every case of appendicitis in which the temperature remains up more than four days, pus is present. In most instances the abscess discharges internally and disappears. To show that this disappearance is not always complete and the evacuation adequate, an illustrative case may be quoted.

In the examples of faecal fistula mentioned above, it was shown that in most cases, six out of eight, the communication was with the small bowel. This I believe to be so in the following instance, because after the discharge of the abscess two spontaneous stools were passed, the first normal in colour and consistence; the second copious, loose, slimy and offensive.

Case of appendicitis with local abscess which was evacuated into the (small) bowel, recurrence of the general

and local signs of abscess, operation, faecal fistula with spontaneous closure.

A girl, S.C., aged thirteen, seen with Dr. Roger-Smith, of Hampstead, had had at least one previous attack of appendicitis, and now was suffering from an acute one consequent on measles. On the fourth day the temperature fell to normal and the general symptoms subsided. By the eighth day the temperature had risen again to 102·6°, and an ill-defined mass was apparent in the abdomen, the girl again becoming ill. We met next morning to incise the abscess, but—

(1) The temperature had come down to between 99° and 100°.

(2) The patient was much better in herself.

(3) The abdomen was less tender and painful.

(4) The mass in the abdomen, which had hitherto been ill-defined, was now easily outlined and handled.

(5) There had been a sudden cessation of pain followed by the passage of—

(6) Two spontaneous stools with tenesmus, the bowels hitherto having been confined.

(7) The first stool was more or less normal, the second copious, loose, slimy and offensive.

No operation was performed. In a few days, however, it became imperative to open the abscess, a temporary faecal fistula resulting, as was expected. The convalescence was without incident.

Had the signs of the discharge of the abscess into the bowel been less definite, the case would have been regarded as one of non-suppurative appendicitis. In 1901, I lost a case of Dr. Cullingworth's whilst removing an appendix

during an interval of the attacks. Unfortunately, I overlooked a perforation of the caecum which had resulted from an apparently non-suppurative attack of appendicitis. Since then, during a similar operation, I have deliberately sewn up a perforation and have seen Mr. Clutton do so once or twice. The presence or persistence of such internal fistulae is of distinct importance in doing even a simple removal of the appendix in a quiet interval.

XII. INTESTINAL ABSCESSES IN CONNEXION WITH APPENDIX ABSCESSES

It usually happens that when an appendix abscess discharges into a hollow viscus, it clears up completely. But one must also remember that the opening may be insufficient to completely evacuate all the pus and the cure be imperfect. Again, the same opening, which allows the passage of pus into the intestines, will also allow intestinal contents to enter the abscess cavity. The recrudescence of the signs and symptoms of the abscess after its internal discharge are due to the formation of an abscess from the intestinal contents rather than inflammation of the appendix. This I believe to have been so in the case of the girl S.C., previously quoted.

It may be said that the first abscess was an appendix abscess, and the second an intestinal abscess. The character of the pus may indicate such an origin. That of the intestinal abscess would seem to be thinner, browner, and not so offensive; that of the appendix abscess thicker, yellower, and more offensive. Considering this, it seems tempting to

suggest that many appendix abscesses, by the time they are opened, contain intestinal contents, i.e. are intestinal abscesses. For all surgeons must have consciously or unconsciously noted differences in the pus evacuated therefrom, such as the olfactorily obvious varying degrees of intensity of the smell. All know that small intestine gases need not be, and usually are not, offensive, though they may easily become so when in an appendix abscess. In this way an explanation of the clinical phenomena so often noticed may be offered.

XIII. THE REMOVAL OF THE APPENDIX AFTER THE OCCURRENCE OF AN ABSCESS

A FEW years ago, Mr. Battle brought forward some cases supporting his contention that the appendix should be removed after abscess formation. That this should be done is not conceded by all surgeons. Yet these same surgeons unhesitatingly advise the removal of the appendix after a severe attack of apparently non-suppurative appendicitis. Judging by the ordinary clinical sequence of events which occur in a septic case, if the temperature remains up on the fourth day of the attack of a disease like appendicitis, which is due to septic micro-organisms, suppuration will be present. Therefore these surgeons have removed numbers of appendices in cases in which suppuration has occurred; their practice being unconsciously at variance with their teaching. But, of course, they have meant that the appendix should not be removed in cases of apparent suppuration with the formation of an abscess so obvious as to have

led to its incision. The question then becomes purely one of the degree of suppuration which is necessary to destroy the appendix. This degree we do not know how to recognize. And it seems just as illogical not to advise appendicectomy after obvious suppuration as not to urge it after a severe, though apparently non-suppurative attack. Those who have operated and removed the appendix after or during abscess formation know quite well that the pathological process, acute infective necrosis, does not often destroy it, that organ being just as ready for further attacks as is the case when it is removed during a quiet interval after an apparently non-suppurative outburst. Where then is the logic of the position that obvious suppuration removes all vice from an appendix ? Whilst if that suppuration is not obvious, the same organ is full of vicious potentialities and must be removed !

A person who has had an appendix abscess is for practical purposes just as liable to further attacks as one who has had a severe attack of appendicitis without apparent suppuration ; some have further attacks, some do not. But we cannot discriminate which of two will be affected and which will not, until the further attack occurs. Therefore, this position should be put before people, and advice in favour of the safer course, appendicectomy, given ; just as is done after a severe apparently non-suppurative attack.

XIV. ACUTE INFECTIVE NECROSIS IN STRANGULATED HERNIAE

In the St. Thomas' Hospital Reports for 1900, published in 1902, I wrote an elaborate paper on the occurrence of gangrene in strangulated herniae, in which it was endeavoured to show the relationship which exists between the results of interference with the blood supply of the ensnared bowel and those of the action of microorganisms. The great importance of the latter was emphasized by the recording of a case in which gangrene or necrosis of the bowel occurred in a strangulated femoral hernia, within twenty-four hours of the onset. This class of gangrene, or better, acute infective necrosis, as it is due to the action of microorganisms, was likened to an attack of gangrenous appendicitis or the perforation of a gastric ulcer. The study of the pathology of these hernia cases led to the application of the same principle of infective necrosis to the acute diseases of other parts of the alimentary tract, and so to the formation of the subject matter of these lectures. I do not intend to reproduce the material given in that paper on account of the large amount that there is already to be dealt with in this course. But, since that paper left my hands, further facts which bear on the subject have come into my possession. These I shall put before you, and so now merely enlarge on the work already published.

The practical importance of the subject of acute infective necrosis of the bowel is very great, both in its influence on the treatment and the diagnosis. As this subject is one which is of great interest, a great deal of attention has

been paid to it. It must be confessed that there is no doubt that some patients have been submitted to the more severe operation of resection of the bowel and anastomosis, both at home and abroad, who never required that operation at all! And again, in a prolonged study of the literature, the same point has struck me. And, though I am urging the early and wide resection of the bowel and anastomosis in cases of acute infective necrosis, I must confess that the statistics and statements in the literature on the subject are, even to a far larger extent than may be thought, quite unreliable. The gut, which one surgeon regards as gangrenous, is deemed recoverable by another, and who is to judge ?

TABLE IV.

GANGRENE IN STRANGULATED HERNIAE.

Variety.	Percentage in which Gangrene was found.
Inguinal	6·1
Femoral	19·5
Umbilical and Ventral	25·4
All cases operated on	14·2

Gangrene within 24 hours of the strangulation in 3% of gangrenous cases at St. Thomas' Hospital; from four London hospitals and two German kliniks, 10%.

In order to aid a better selection of cases, the signs of gangrene are reproduced from my paper, by which, I believe, this acute infective necrosis is recognizable, and are contrasted shortly with those ordinarily given for its recognition in strangulated herniae. In this way, I hope to define the position which has been taken up, so as to avoid the slur, to put it mildly, of excessive surgical zeal.

The bowel in these cases is dark or black in colour, the

stricture is so tight that in the production of the strangulation both arteries and veins may be almost or even completely and simultaneously occluded. Two things result from this : Firstly, the sac almost invariably contains little or no fluid, and secondly, the gut is not oedematous or doughy, as is taught. On the contrary, it is thin, inelastic, and does not recover its shape when deformed, as it easily is. Its surface may glisten as the empty condition of the vessels interferes with the inflammatory dulling or glazing of the peritoneum covering it. It contains blood if the initial vascular strangulation has been incomplete ; no gas is present within its lumen having been absorbed by the extravasated blood. There may be signs of local peritonitis on the sac rather than on the bowel, as evidenced by dulling, glazing, etc., or a perforation of the bowel may be present. This necrosed bowel differs very markedly from that ordinarily described, on account of the different method of its production. The necrosis is the result of the combination of tight strangulation and the subsequent action of microorganisms. The above described signs are evidences of the former factor almost entirely.

Examples are given in the paper in the St. Thomas' Hospital Reports, 1900, pp. 347–349, which illustrate the following clinical types—

(1) A local infective peritonitis in the hernia sac without gangrene of the gut, due to the diapedesis of organisms through the wall.

(2) A general infective peritonitis after the reposition of the bowel within the abdomen and without gangrene of the strangulated loop, also due to the diapedesis of organisms.

(3) The occurrence of general peritonitis and gangrene of the strangulated loop after the return of apparently healthy bowel to the abdomen.

(4) The occurrence of gangrene of the bowel in strangulation of intestine, both internal and external to the abdominal cavity, within twenty-four hours of the onset of symptoms.

(5) The spreading character of this pathological process.

(6) The greater tendency of the necrotic process to spread upwards, i.e. in the distended bowel above, rather than downwards in the collapsed bowel below, the strangulating agent.

To the first of these illustrations I have little noteworthy to add, and so to save time and space, will pass on to the third point at once.

The case in which gangrene occurred after the apparently healthy piece of bowel, at the operation, had been returned to the abdomen, is so emphatic and remarkable that it is worthy of reproduction—

Case of Acute Infective Necrosis in a Strangulated Femoral Hernia.

A woman, forty-two years of age, was operated on for a femoral hernia of the right side, which had been strangulated twelve hours. The knuckle of small bowel looked in perfect condition, in every way, to be returned to the abdomen. It was of normal consistence, shiny peritoneal coat, and so forth. In fact, according to the views which were then held, there was not a moment's doubt as to its being replaced in the abdomen. On the fourth day after operation she died. Dr. Stacey Colman, who performed the autopsy, showed me the coil, which had been ensnared, completely gangrenous !

This instance was the first of the kind which I had met with, and the occurrence of gangrene of the bowel after

twelve hours' strangulation offered much food for meditation and reflection.

In the first place, I believe the necrosis was due to the action of microorganisms, but a secondary phlebitis and consequent thrombosis of the mesenteric vessels, which may also have been due to the microorganisms, may have played a part. The vessels were certainly not completely occluded at the time of the operation, as the bowel quite distinctly improved in colour after the edge of Gimbernat's ligament had been divided and the constriction relieved. So far as is known, thrombosis of the vessels of the mesentery after the reposition of the loop of intestine in the abdomen is far from common, but there are always a certain number of cases in which the gut has been returned and subsequently has become gangrenous. Little attention has ever been given to these cases, as the post mortem examinations are usually conducted by physicians, and surgeons are apt not to lay stress on these fatalities.

In the paper in the St. Thomas' Hospital Reports, to which I have often referred, a table was constructed showing how often this occurred (p. 361), and is reproduced here—

TABLE V.

TABLE SHOWING THE DISTRIBUTION AND FREQUENCY IN WHICH THE BOWEL BECAME GANGRENOUS AFTER HAVING BEEN REDUCED AT THE OPERATION FOR THE RELIEF OF A STRANGULATED HERNIA.

Variety of Hernia.	Number of Cases.	Number in which the gut became gangrenous after its return to the abdomen.	Per-centage.
Inguinal	212	7	3·3
Femoral	181	16	8·8
Umbilical	44	3	6·8
All varieties	437	26	5·9

It is not possible to hazard a suggestion as to whether these are due to mistaken diagnoses made at the operation or to a subsequent phlebitis and thrombosis of the vessels in the mesentery involved by the strangulation.

As I wish to direct special attention in these lectures to the instances of acute infective necrosis, I would like to mention a few more cases than the two of my own which have already been recorded (St. Thomas' Hospital Reports, 1900), in which necrosis occurred in the bowel within twenty-four hours of the onset of strangulation.

(1) A woman, aged sixty-four, had a femoral hernia which had been strangulated for less than twenty-four hours. Reduction was effected by taxis, the gut necrosed, and the woman died.

(2) A woman, aged fifty-two, had an umbilical hernia which had been strangulated for eleven hours. Reduction by taxis. Death resulted from peritonitis due to necrosis of the gut involved.

(3) A woman, aged fifty-nine, had a femoral hernia which had been strangulated for twenty-four hours, reduction by taxis, subsequent perforation, necrosis of the gut, and death.

(4) A man, aged fifty-nine, was operated on for a strangulated inguinal hernia which had presented symptoms for fifteen hours ; the gut was reduced and a " radical cure " performed. Death resulted from subsequent necrosis of the bowel and early peritonitis.

(5) A woman, aged seventy, femoral hernia, strangulated under twenty-four hours, resection of bowel and anastomosis ; death.

(6) A woman, aged sixty-four, umbilical hernia, strangulated twenty-four hours, artificial anus ; death.

(7) A woman, aged forty-four, ventral hernia, strangu-

lated twenty-four hours, resection and anastomosis; death.

The above cases have been taken from the Surgical Reports of the Middlesex and University College Hospitals, in order to show that gangrene of the bowel can occur within twenty-four hours' time at other hospitals than St. Thomas'. They are fatal cases, as most are, otherwise they would have found their way into other literature than Hospital Reports. It is also of interest to note that three out of the seven were instances in which the strangulated hernia had been reduced by taxis; one, in which the condition of the bowel was not recognized at the operation and three in which it was.

TABLE VI.

Table of Times at which Gangrene was found in a Strangulated Hernia at St. Thomas' Hospital.

Days of Strangulation.	Number of Cases.
under 1 day	2
1–2 days	11
2–3 ,,	13
3–4 ,,	8
4–5 ,,	11
5–6 ,,	11
6–7 ,,	2
over 7 ,,	9

Gangrene was found in 6·1 per cent. of inguinal herniae, 19·5 per cent. in femoral, and 25·4 per cent. of umbilical herniae. It occurred in 14·2 per cent. of all cases operated on. Acute infective necrosis of the bowel is, therefore, not a common disease if defined as the gangrene occurring within twenty-four hours of the onset. In the above it forms only three per cent. of gangrenous herniae, a percentage which is, in reality, far too small to represent any but the

most acute action of the micro-organisms. It would not form one per cent. of all strangulated herniae.

In the following table an attempt has been made to form a list which would show the frequency of the occurrence of gangrene within twenty-four hours of the onset of the strangulation of herniae at St. Thomas', the Middlesex, the University College, and St. Bartholomew's Hospitals, and Bruns' and Maydl's Kliniks abroad.

TABLE VII.

GANGRENOUS HERNIAE, SHOWING THE FREQUENCY OF ACUTE INFECTIVE NECROSIS.

	Number of Gangrenous Cases within 24 hours.	Number of Gangrenous Hernia.	Per-centage.
St. Thomas' Hospital . . .	2	67	3
Middlesex Hospital . . .	3	12	25
University College Hospital . .	6	33	18
St. Bartholomew's Hospital . .	2	28	7
Bruns' Klinik (by Hofmeister) .	7	27	25
Maydl's Klinik (by Michel) . .	3	54	5
TOTAL . . .	23	221	10

It is interesting to note that the school, S. Thomas' Hospital, from which the material was obtained for these lectures, and which same material directed attention to this condition of acute infective necrosis, shows the lowest percentage of its occurrence! And the figures from Hospital Reports are those least biassed by the personal equation of the recorder.

If we now turn to the literature which has been largely though not completely overhauled, it was found that the reported case in which gangrene was found earliest was that of Nové-Josseraud. It occurred in the left inguinal hernia of a young man, with a congenital sac associated with an

imperfectly descended testis. Twelve centimetres of the small bowel were resected, immediate anastomosis was followed by recovery. The duration of the strangulation was only seven hours, and the symptoms were most acute. Michel reports a fatal case in a woman of sixty-eight, who had a strangulated left "inguinal" hernia, of eight hours duration. That is to say, a fatal case of only one more hour of strangulation in an old woman, than in Nové-Josseraud's successful one in a young man.

Besides the case reported in my paper already quoted, in the St. Thomas' Hospital Reports, 1900, p. 358–360, I have operated on another, also successfully, which was associated with an imperfectly descended testis, as was that of M. Nové-Josseraud.

Case of Strangulated Inguinal Hernia, Acute Infective Necrosis of the Small Bowel, Resection and Anastomosis; Recovery.

A man, G.E., aged twenty-nine, was admitted to St. Thomas' Hospital on June 26, 1902, with most severe pain in *a left strangulated inguinal hernia of twelve to sixteen hours duration*. There was an imperfectly developed testis on the same side. He had known of the hernia for fifteen years and had worn a truss, which, as it had not been down for a "long while," was discarded. It was the first time the rupture had reappeared when he was brought to the Hospital. At the operation the sac contained a very little fluid. About six inches of small intestine had been suddenly expelled from the abdomen, the amount expelled causing immediate and tight strangulation of the loop. The intestine was full of blood, showing that the occlusion

of the vessels had not been complete at once. The blood within the bowel had absorbed all the intestinal gases. Owing to the distension of the coil of gut it had twisted on itself completely occluding everything. The bowel itself was thin, sticky, and inelastic; not oedematous, doughy or offensive. The vessels in its mesentery were thrombosed. About a foot was removed, the resection being most free above the obstruction. An end to end anastomosis was made with two rows of continuous silk stitches. The testis was underneath the gut and had a half twist on its spermatic cord. The gland was excised. A radical cure was performed with fine salmon gut. On the second day an enema yielded a good result of flatus, and on the fourth day another enema gave a large faecal result. Feeding by mouth began on the fourth day. Healing per primam.[1]

When once this necrotic, necrobiotic, or necrogenous process gets a good start, it may extend, especially up the bowel in the part above the constriction. I have already mentioned and illustrated this tendency to extend, in the St. Thomas' Reports, 1900. It is not easy to find records of cases in which gangrene has occurred after resection, as in Hospital Reports they escape notice, and surgeons do not publish them themselves. Nevertheless, Mr. Hewston has recorded one in a woman of sixty-five years of age, for whom he resected the small gut in a right femoral hernia, which had been strangulated four days. The resection had been to all appearance free. But, post mortem, the bowel was gangrenous for an inch above the line of suturing! There is no record of a case in which the gangrene spread downwards to any extent sufficient to cause trouble in treat-

[1] This case was read to the South Hants Branch of the British Medical Association, at Winchester, 1902, under the Presidency of Dr. David Browne.

ment.[1] It is important that the surgeon should bear in mind this pathological point. Professor Kocher, of Berne, some years ago, pointed out that it is not the amount of gut resected which kills the patient. It might almost be added that it is usually the insufficient resection of the gut which causes the fatal result; an anastomosis having been performed on tissues which were teeming with organisms. Mr. Barker, in *The Lancet*, 1903, i. 1576, strongly advocates a wide resection. Mr. Makins, in the Clinical Society's Transactions for 1903, has advocated rather extensive involution of doubtful bowel wall in these cases. And later, this has been emphasized by Dr. Crampe of the Königsberg Klinik, in the *Beiträge zur Klinischen Chirurgie*, Band xxxviii., 2 Heft, 1903. This is a most suitable and successful measure in proper cases as both have shown. But its applicability diminishes as the action of micro-organisms over the results of interference with the blood supply increases. Therefore it will be inapplicable in cases in which gangrene has occurred within a few hours of or shortly after the onset of strangulation; whilst it will be most suitable in patchy gangrene about the site of the constriction of the gut and not in oedematous parts of the loop. It is a measure far more applicable to doubtful pieces of bowel, as Dr. Crampe has urged, rather than those in which gangrene has already declared itself.

XV. MECKEL'S DIVERTICULUM

In the foregoing sections of this lecture, it has been pointed out that the appendix forms the terminal portion of our great natural intestinal "backwater," the caecum. Any

[1] Except Case II, page 67.

diverticulum of the intestine forms a cul de sac and a somewhat similar backwater. The most constant diverticulum of the human intestine is that described by Meckel, which represents the proximal part of the duct which connects the intestine and the yolk sac of the foetus. This little process arises from the ileum about thirty inches from the ileocaecal valve, and is present in two per cent. of subjects. It differs from the appendix in several most important features which naturally cause the differences in the pathologies of the two tubes. For instance, Meckel's diverticulum is not situated at a place utilized by the products of digestion as a "rest," as the appendix is at the caecum ; its lumen is almost invariably as large as that of the intestine from which it springs, and it is, as a result, well flushed out by the flow of the intestinal fluids caused by the peristalsis of the ileum, as well as by its own contractions. There is, therefore, no stasis of the faecal contents within the diverticulum, nor the opportunity for organisms to luxuriate within it to anything like the extent that there is in the appendix. Therefore it is not to be expected that the former will suffer from as many or perhaps as acute inflammations as does the latter.

These analogies and homologies interested me so thoroughly that an attempt was made to ascertain all the cases in which this blind process of bowel was diseased. The results have repaid the labour, and as they have never been collected previously, they are briefly reproduced. In all, I have extracted from hospital reports and literature over 300 cases, which are arranged in a table as follows—

TABLE VIII.

Diseases of Meckel's Diverticulum.

Cystic formation, retention cysts	7 cases.
Tuberculous disease	3 „
Typhoid ulceration	4 „
Chronic inflammation (diverticulitis)	3 „
Acute inflammation (diverticulitis)	3 „
Ditto with necrosis or gangrene	11 „
„ „ perforation	16 „
„ „ local abscess	4 „
„ „ torsion of the diverticulum	5 „
Fistula at umbilicus	20 „
Prolapse at umbilicus	15 „
Intussusception	23 „
Foreign bodies in the diverticulum	11 „
Diverticulum ruptured by a kick	2 „
Intestinal obstruction caused by the diverticulum (other than intussusception)	166 „
Ditto and volvulus (?)	3 „
Ditto and perforation	6 „
Volvulus (?)	1 „
Papillomata at umbilicus	12 „
Carcinoma	1 „
	316

The above list, although it gives all the records that could be found, has the disadvantage of minimizing the frequency of the more common affections, and magnifying that of those of rarer occurrence. By means of reducing the above catalogue to shorter proportions, and giving percentages instead of absolute figures, a better idea of the relative importance of the various factors can be gained.

TABLE IX.

Table of the Diseases of Meckel's Diverticulum.

Intestinal obstruction other than by intussusception	56 per cent.
Inflammation or diverticulitis (American)	13 „ „
Fistula, prolapse at the umbilicus	11 „ „
Intussusceptions	7 „ „
The rest	13 „ „

In the case of this diverticulum, the proportions of cases described as due to inflammation, or diverticulitis (American), bears to those in which the process caused intestinal obstruction, other than by invagination, the proportion of one to four. Whilst it is the reverse in the case of the appendix, inflammation being exceedingly common whilst it is rare for that organ to form a band and cause obstruction. This great difference is due to the previously mentioned factors, which have led to the comparative infrequency of inflammation in the diverticulum, and its situation amongst the coils of small intestine which it can in consequence easily ensnare and obstruct. But the difference in the relative frequency or infrequency of inflammatory processes in the appendix and the diverticulum is more apparent than real, as the following problem will show, and at the same time teach us to recognize a new, and when the diverticulum is present, a very common disease.

For practical purposes, the intestinal obstruction caused by Meckel's diverticulum, may be regarded as due to the ensnaring of a loop of bowel beneath the process which is adherent by its tip. The regions in which the adhesion has been described are given below—

TABLE X.

TABLE OF THE ADHESIONS OF MECKEL'S DIVERTICULUM.

To the umbilicus	23 cases.
,, ,, anterior abdominal wall	7 ,,
,, ,, posterior ,, ,,	4 ,,
,, ,, mesentery	41 ,,
,, ,, omentum	5 ,,
,, ,, small bowel	7 ,,
,, ,, bladder	1 case.
,, ,, femoral canal	1 ,,
,, ,, pelvis	2 cases.
TOTAL	91 ,,

The diverticulum is recorded as being without adhesions in thirty-nine cases, but doubtless, this condition is not mentioned as often as the presence of the adhaesions is, because the latter are so frequently the source of trouble, thus attracting attention. The question may well be asked, " What are the causes of these adhaesions ? "

In the first place, they may be divided into two kinds, those due to errors of the development and those due to inflammation. In the former, the process may open at the umbilicus, be connected with it by a cord containing merely the remains of the vitelline vessels, or again, as my friend Dr. S. G. Scott, of Leeds, has told me, the connexion with the anterior abdominal wall may contain the remains of the same structures. The latter class of case may be due to an error of development, inflammation, or both. But for my purpose they will be considered as being of developmental origin, in order that the error, if any, be made on the safe side for my purpose, i.e. of finding too many developmental fixations. The second class of cases arise from inflammatory causes, such as the adhaesion to the mesentery, omentum, small bowel, etc. The inflammation or diverticulitis is due to the causes inherent to an intestinal " backwater," as exemplified in an acute degree in the diseases of the appendix, and in a subacute degree those of Meckel's diverticulum.

Placing these two classes side by side, it is seen that—

Developmental adhaesions formed	30	cases,	or	33	per	cent.
Pathological	„	„	61	„	67	„	„

The figures emphasize the frequency of what may be called a chronic or subacute diverticulitis, a disease hitherto unrecognized, yet far-reaching in its consequences.

Many are the points of interest connected with the pathology of this diverticulum, but there is not time nor indeed

is this the proper place to enter upon such a dissertation. To return to the subject of these lectures, acute infective necrosis, thirty cases of perforation and sloughing of the diverticulum have been recorded. As is the case with the appendix, the perforations usually occur at the tip, i.e. at the part from which the organisms are least completely evacuated. Though in the case of the diverticulum that part often has a direct blood supply. These cases are always diagnosed clinically as appendicitis. No primary vascular lesions have been described. The perforations found in Meckel's diverticulum which have been recorded, exhibit one very marked difference from those found in the appendix, namely their multiplicity. Mr. Makins described a case, in the *Path. Soc. Trans.*, xliv. p. 90, with numerous small perforating ulcers of the diverticulum. In the appendix it is very rare that more than one has been seen, in the stomach and duodenum two have been described, very rarely more. It is not easy to explain this peculiarity on the part of Meckel's process.

In the appendix the infection is gross on account of the host of organisms present and the inability of the tube to contract efficiently and expel them. In the case of the diverticulum there are probably not the number of organisms present, and again, this tube can evacuate itself. It would seem that in the appendix the organisms are retained in the lumen of the tube, and in the diverticulum they are imprisoned in the glands, otherwise they might be washed away. Hence, in the former, the gangrene or necrosis is gross, and in the latter it occurs in small, numerous, punctate patches. A similar explanation may account for the pin-hole perforations occasionally seen in the stomach, duodenum, etc.

Five cases of torsion of Meckel's diverticulum have been

recorded, the most extensive gangrene having occurred within twenty-four hours of the onset of symptoms, whilst the cases have been clinically of the most fulminating type. In the most severe instances, there may have been both interference with the blood supply and the action of virulent microorganisms. Mr. Carwardine has recorded a most unique and interesting case, which is reproduced shortly from the *British Medical Journal*, 1897, ii., 1637.

1. Case of torsion of Meckel's diverticulum, leading to the formation of a retention cyst, in a foetus.

Mr. Carwardine, of Bristol, operated upon a male baby, two days old, for intestinal obstruction. He found a large meconium containing cyst of Meckel's diverticulum, separated from the ileum by three complete twists, the bowel being also occluded by the traction of the turns. The torsion must have begun in foetal life, and it was unaccompanied by any signs of inflammation or necrosis in the cyst.

This case is absolutely unique as it stands ; but it possesses additional interest from the point of view of acute infective necrosis or gangrene. Before birth the meconium is sterile, and consequently Mr. Carwardine's case becomes an exceeding rare example of an aseptic torsion of the bowel and a vivid contrast with the fulminating examples just mentioned. Hence the absence of gangrene and signs of inflammation.

To form a further contrast with this very rare aseptic and in a local sense pathologically mild condition, examples are given of two of my own cases in which there was acute infective necrosis of both the diverticulum and the loop of ensnared bowel in examples of intestinal obstruction. The possibility of the occurrence of this double gangrene in the most common form of clinical manifestation caused by this process, 56 per cent. of all cases, emphasizes the practical importance of the lesions to be illustrated.

XVI. INTESTINAL OBSTRUCTION BY MECKEL'S DIVERTICULUM AND THE APPENDIX

CASE I. INTESTINAL OBSTRUCTION BY AN ADHERENT MECKEL'S DIVERTICULUM, ACUTE INFECTIVE NECROSIS OR GANGRENE OF THE DIVERTICULUM AND OF THE ENSNARED INTESTINE. DEATH.

W.E.C., male, aged 32, was admitted on July 28, 1901, to St. Thomas' Hospital, with the history of three days acute intestinal obstruction. The abdomen was opened and the obstruction found to be caused by a diverticulum of the ileum about four inches long and adherent by its tip to the mesentery of the small bowel. Underneath this band a coil of small gut was strangulated and also twisted on itself, so completing the obstruction. The diverticulum was sloughing and was removed, the bowel tapped and sutured, lavage, etc. A day later the lower segment of the wound was reopened and the ensnared coil found to have become gangrenous. It was withdrawn from the abdomen and Paul's tubes inserted. Death followed in a few hours.

CASE II. INTESTINAL OBSTRUCTION BY MECKEL'S DIVERTICULUM, INFECTIVE NECROSIS OF THE DIVERTICULUM, POSTOPERATIVE MESENTERIAL THROMBOSIS AND NECROSIS OF THE STRANGULATED LOOP, PERITONITIS. DEATH.

R.W., a boy, aged 10, was sent to St. Thomas' Hospital on February 3, 1904, with signs of peritonitis and intestinal obstruction of four days duration. At the operation the cause of the obstruction was found to be a coil of small bowel had become ensnared beneath an adherent diverticulum, also twisting on itself. The adhesion was to the

mesentery of the small bowel. The peritonitis was found to have arisen in part from a perforation at the site of constriction. The diverticulum was constricted over the ensnared loop and its tip was converted into a nearly free gangrenous cyst. The process was removed and the perforation sutured, etc. The boy died forty-eight hours later of peritonitis.

At the post-mortem examination it was found that the vessels in the mesentery of the ensnared loop had become thrombosed; they were not so at the operation, as this point was especially looked for in view of the postoperative necrosis in the previous case. The thrombosis had spread to the vessels in the mesentery of the ileum below the diverticulum. As a consequence the necrosis in the loop, which had been strangulated, had extended downward nearly as far as the caecum. About four feet of bowel were gangrenous at the post mortem.

This case illustrates the postoperative thrombosis of the mesenterial vessels which was most probably of bacterial origin, the consequent necrosis of the coil which had been ensnared and the extension of the necrosis downward in the gut with the thrombosis.

The gangrenous cyst, half-an-inch in diameter, at the tip of the diverticulum would very shortly have become separated, and in so doing, would, in all probability, have set free the adherent constricting band, so relieving the mechanical obstruction. The case might then have been regarded as a primary necrotic inflammation of the diverticulum. It is almost tempting to suggest that some of the reported cases may have been of such an origin. The idea does not seem to have suggested itself to those who have written on this subject.

When cases of intestinal obstruction due to an adherent

appendix are met with, it will be noticed that the distal part of that organ is inflamed, perforated, or surrounded by an abscess, just in the same way as the above illustrations have shown the corresponding part of Meckel's diverticulum to be. Mr. G. Grey Turner, of Newcastle, in his *Surgical Aspects of Appendicitis*, quotes a case in illustration of this.

He operated on a man for intestinal obstruction and found the appendix adherent by its tip to the back of the mesentery, and "there can be little doubt that there was mechanical obstruction produced by the adherent appendix acting like a band across the ileum." "It was adherent by its tip . . . and there was a small abscess in this situation." Hermes, quoted by Mr. Turner, describes a similar case in which the adherent tip of the appendix contained an abscess. The material and organisms which are dammed up in the tip of a blind process will lead to sloughing or acute infective necrosis of that part, as the above examples have shown to be the case with Meckel's diverticulum and the appendix. Non-virulent organisms sometimes die out, as is seen in some cystic appendices.

These examples complete the analogies between the appendix and its diseases to Meckel's diverticulum and its diseases.

XVII. CHOLECYSTITIS AND PERFORATIONS OF THE GALL BLADDER

The analogy between the gall bladder and the appendix is a very striking one. Both are diverticula of the alimentary tract being natural backwaters of it. Both are liable to acute and chronic infections from it. As a result of the

latter, in both, concretions and calculi are formed. True consequences, in the shape of drying of the secretions, are gall-bladder and appendix constipation; compare the scybala of the large intestine. Leaving other points of analogy, both are liable to acute infective disease which leads to necrosis (gangrene) or perforation. From an examination of specimens there is no difference discernible between these conditions in the appendix and in the gall bladder. Just as there are none from those of the stomach, duodenum, etc. The gall bladder forms one of a series of the diverticula of the gut which are prone to infective diseases. Which pathological processes, as in the case of the appendix, Meckel's diverticulum, etc., are brought about by the immigration of organisms from the alimentary tract and their growth and luxuriation in one of these natural arbours.

CASE I. CHOLELITHIASIS, EMPYEMA OF THE GALL BLADDER, PERFORATION, INTRAPERITONEAL ABSCESS, SUBDIAPHRAGMATIC ABSCESS, PUS IN THE LESSER SAC OF PERITONEUM.

Female, aet. 39, with a previous history of attacks of jaundice and gall stone colic. Two days before admission she was seized with pain in the upper part of the abdomen and sickness. Bowels confined. Patient was very weak and in bad condition. Six days after admission a swelling was noticed to the right of the umbilicus, which was incised the next day. A large quantity of pus and bile, plus one gall stone, was evacuated. Death occurred in two days time from exhaustion.

Post mortem, it was found that an intraperitoneal abscess had been opened, there was a left subdiaphragmatic abscess and the lesser sac of peritoneum was full of pus. The gall

bladder itself was lined with pus and perforated. The perforation was identical in appearance to those found in the stomach, intestines, etc. There were no more gall stones.

(For full report of the case, see St. Thomas' Hospital Reports, 1900, p. 68.)

CASE II. PERFORATION OF THE GALL BLADDER, GENERAL PERITONITIS.

Male, aet. 49, was admitted with a history of five days abdominal pains and vomiting, bowels confined at first, but latterly loose. Death in a few hours.

Post mortem, an intraperitoneal abscess was found all round the gall bladder, from whence the peritoneum of the upper part of the abdomen had been infected. The typical perforation was at the tip of the gall bladder, which contained eight small stones.

(For the rest of the report, see the St. Thomas' Hospital Reports, 1900, p. 70.)

The diseases of the three diverticula of the alimentary canal are similar in all respects. There remain two other structures intimately connected with the intestine, namely the liver and the pancreas. As diseases of the organs cannot be considered to come under the title of the lectures only the briefest references will be made to them.

XVIII. ACUTE PANCREATITIS AND ACUTE LIVER ABSCESS

It has been urged from a consideration of the clinical and pathological characters, that the acute perforating or gangrenous process is uniform in its origination throughout the alimentary tract. And also it has been urged that it is identical with the cause of perforations and gangrene in the diverticula of the gut, namely, the appendix, Meckel's diverticulum and the gall bladder. And one may go still a step further. Why should not the same organisms infect the pancreas and the liver? These glands are merely exaggerated diverticula of the intestine. It will therefore be expected that acute pancreatitis, with or without haemorrhage, will be caused by similar organisms. The same reasoning applies to acute liver abscesses. And both these diseases become comparable to appendicitis, being probably identical in the actual causation and the pathological lesion of acute infective necrosis. So that we may look for similar bacteriological results in all acute abdominal disease.

XIX. GASTRIC, DUODENAL AND OTHER PERFORATING ULCERS OF THE ALIMENTARY TRACT

Taking the perforations or gangrene of the appendix as the type of acute infective necrosis, it will be seen from clinical, pathological and bacteriological observations that there is no reason to place ulcers of the stomach, the duodenum and other parts of the alimentary canal upon a different basis.

Dr. Box, in the *British Medical Journal*, February 8, 1902, pointed out that such perforations fall into two categories. In one, the perforation, which is primary or acute, is surrounded by an oedematous inflammatory zone, and in the other the perforation is secondary, subacute or chronic, and without such a zone. This is quite true in a sense, but the oedema is due to the perforation or to the process that produces the perforation; and naturally it shows up more easily in healthy than in previously chronically inflamed or fibrous tissues. It must always be present in degree, because the slough, the separation of which has caused the perforation, is freed from the surrounding tissues by an inflammatory process, and therefore accompanied by swelling of the tissues. That the amount of the surrounding oedema is very various is seen in the abstracts of the following cases—

1. W.S., female, aet. 17. Perforation of gastric ulcer occurred six hours before operation. The ulcer was easily sutured, owing to the slight change in the surrounding tissues of the anterior wall of the stomach. Lavage. Recovery.

2. J.C., male, aet. 61. Perforation of gastric ulcer two hours or so before operation, the stomach being full at the time. The perforation was easily sutured for the same reasons as in the above case. Lavage. Twenty-fifth day, left subphrenic abscess opened. Recovery.

The ulcer was high up on the cardiac end of the posterior surface of the stomach.

3. W.T., male, aet. 33. Duration of perforation of gastric ulcer twenty-four hours. Suture easy. Lavage. Recovery. Ulcer on anterior wall.

4. J.C., male, aet. 34. Perforation of duodenal ulcer for twenty-four hours, on the anterior wall of first part. Suture

easy. Lavage. Recovery. Death six months later from intestinal obstruction.

The oedema surrounding the perforation has at least a twofold origin; from the perforation and from the peritonitis which ensues.

The above successful cases are instructive in a double sense, firstly, in that they recovered, and secondly, in that they reflect more credit on the diagnostician than on the surgeon, and to contrast with them, the following examples will show what valuable knowledge is to be gained from unsuccessful ones.

5. M.J., female, aet. 18. Gastric ulcer perforated for thirteen hours. At the operation it was found that the tissues surrounding the perforation were very rotten and difficult to suture. Lavage. Death after twenty-four hours. Post mortem, the stitching had completely given way owing to the spread of the gangrene or necrosis, which had caused the perforation.

6. ——, male, aet. 45. Twenty-four hours' perforation of a duodenal ulcer. Difficult suture. Lavage. Death. Post mortem, the stitching had given way through the spread of the gangrene to the tissues in which they were inserted.

In both the above, the stitches were carried wide of the ulcer.

I have selected cases which had perforated within twenty-four hours before operation, so as to exclude, as far as possible, the softening and oedema of the consequent peritonitis. The above exemplify that when suture of the perforation was easy, recovery resulted; and conversely, where it was difficult, death resulted from further necrosis of the tissues. The last phrase, "further necrosis of the tissues," gives the key to the explanation of the condition of the walls surround-

ing the ulcer. If the septic, perforating or necrotic process is strictly local, there need be very little surrounding inflammation and oedema. If, on the other hand, the same process is not localized focally to the perforation, i.e. as if focussed on it, the surrounding tissues will be inflamed and oedematous, and the gangrene or necrosis is apt to spread, leading to loosening of the stitches and a fatal result.

I would, therefore, like to follow Dr. Box's lead one step further and recognize two main divisions and two subdivisions of perforated gastric and other ulcers.

1. The chronic, in which the surrounding oedema and inflammation may be very slight.

2. The acute, of which there are two classes.[1]

a. The acute ulceration or necrosis, which may be likened to the action of a staphylococcus in a hair follicle at the back of the neck ; and owing to the thinness of the gastric or intestinal wall, the whole thickness is involved by the slough or core, a perforation resulting. As the area of infection is distinctly local and focal, there need be but little oedema of the contiguous tissues.

b. The perforation may occur in but a part of a wider area of septic infection, as the many sinuses of a carbuncle, and typified by the action of a streptococcus. In this case the surrounding tissues will be oedematous and inflamed. The perforation is local, but the infection is not focal to it but wider spread.

Clinically and pathologically there is no difference, *per se*, in the perforation of the stomach, duodenum, typhoid ulcer, appendix or Meckel's diverticulum, etc., and they may all be regarded as local examples of the general pathological process of acute infective necrosis.

In a monograph on acute abdominal cases, to be published

[1] See page 87.

by Mr. Battle [1] and myself, acute infective necrosis of the appendix was found to be the most frequent by far; the stomach came second, the duodenum third, and the rectum fourth. The last is associated in almost every case with carcinoma. There is nothing new to be said about the process.

XX. PERFORATIONS OF THE CAECUM

THERE is another perforation of the alimentary tract due to an acute necrosis which is of interest. With the obstruction due to a carcinoma of the sigmoid, death not infrequently ensues from a perforation of the caecum. It is not obvious why it is thus picked out. In a previous section of these lectures it was pointed out that the caecum was the situation where the products of digestion "rested or paused" until they had acquired the necessary consistence for their passage along the large intestine. As a consequence it became the great nursery and place for the luxuriation and growth of organisms, and in any condition where the flora is increased in number and virulency as it is in intestinal obstruction, the caecum is very liable to be selected for the site of a perforation. Kreutzer, in Langenbeck's *Archiv.*, 1903, lxx. Heft 2, records a case which exhibits the fulminating way in which organisms can attack the caecal region. The case was one of volvulus of the sigmoid and gangrene of the caecum occurred in a few hours time. During the work of preparing a paper on "Volvulus of the Caecum," Mr. P. W. G. Sargent and I have found many cases of early gangrene of that part of the alimentary tract.

[1] *The Surgery of the Diseases of the Appendix* (Constable).

XXI. INTUSSUSCEPTION

The following case may be quoted as an example of the occurrence of acute infective necrosis in an intussusception—

Enteric-ileocaecal intussusception ; acute infective necrosis of the intussusceptum.—The case is of sufficient clinical interest to deserve record, being one in which gangrene of the intussusceptum occurred within twenty-four hours of the onset, due to an acute infective necrosis of the bowel.

A male baby, aet. ten months, was admitted to the hospital under the care of Dr. Box, who diagnosed intussusception and had the operation proceeded with at once. An incision was made through the right rectus muscle. The intussusception was found in the middle of the transverse colon. The first portion was reduced quite easily by pressure, but the last half could not be stirred. As the child was very ill its only chance lay in reduction, resection being quite out of the question. Force was therefore used and though the intussusception was reduced the caecum burst in one place. This rent was quickly sewn up with continuous silk sutures. Some through and through stitches were used to close the abdominal wall. The intussusception was of the enteric-ileocaecal variety. The last eight to ten inches of the ileum were in a very parlous condition. The child died shortly afterwards, and at the autopsy it was found that the last ten inches of the ileum were gangrenous, the highest part being the worst. All this damage had been done well within twenty-four hours.

As the intussusception started ten inches above the ileo-caecal valve it is most probable that the intussusceptum pushed the valve in front of it. (St. Thomas' Hospital Reports, 1901, xxx., and *Annals of Surgery*, November, 1904.)

XXII. INTESTINAL OBSTRUCTION BY BANDS

As examples of acute infective necrosis of the bowel in cases of internal strangulation, two examples may be mentioned.

1. Intestinal obstruction by a band connected with a left inguinal hernia.

E. F., male, aet. 30, was admitted to St. Thomas' Hospital June 27, 1901, suffering from intestinal obstruction of thirty hours duration. The man was very ill indeed and was infused intravenously with three pints of saline at once. An incision was made and a black coil of small bowel presented. It was estimated that six feet of small bowel were tightly strangulated by a band, and had twisted on themselves, forming a volvulus. The coil itself was distended and full of blood. The patient's condition was too bad to admit of further procedure. Death resulted in sixteen hours. At the post-mortem examination Dr. Box found eight and a half feet of small bowel from the caecum upwards were gangrenous. The strangulating band originated in a left inguinal hernia sac.

2. Intestinal obstruction by bands.

A girl, aet. 23, was admitted to St. Thomas' Hospital suffering from intestinal obstruction of twenty-four hours' duration. At the operation three and a half feet of gangrenous small intestine were released from under a band and resected, Paul's tubes being inserted. The patient died in thirty-six hours, and at the post-mortem examination it was found that the gangrene had extended beyond the part resected, although the excision had been done to all appearances very freely, and so given rise to general peritonitis.

These two cases illustrate both the rapidity and the spreading character of the necrotic process.

XXIII. THE CLINICAL INTERPRETATION OF THE BACTERIOLOGY OF ACUTE INFECTIVE NECROSIS

In the preceding sections it has been attempted to prove to you that lesions of a similar pathological nature, acute infective necrosis or gangrene, occur in the stomach, the duodenum, the small intestine, the caecum, the appendix, the large bowel, Meckel's diverticulum, the gall bladder, strangulated herniae and other forms of intestinal obstruction. To the naked eye the character of the lesion in perforated gastric, duodenal and other ulcers, appendicitis, etc., is identical in each case. Hence the attempt has been made to urge singleness in the infective origin of the lesions. The discussion of the various organisms found in the peritonitic fluid, and elsewhere, in examples of such conditions, has been reserved for the third lecture, when, from the bacteriology, a further attempt will be made to show the universal activity of microorganisms and the probable supreme and universal importance of the rôle of septic cocci in the production of infective necrosis.

It is also hoped that much has been done to remove from the walls of the alimentary canal their traditional character for delicacy, and their proneness to become gangrenous or necrotic on the least provocation. The tissues of the alimentary tract are, in fact, liable to exactly the same laws of life and death as the others which constitute the body. But, owing to the natural septicity of the contents of the intestinal canal, moist gangrene, i.e. septic death or necrosis, is infinitely more common than a

dry gangrene, i.e. aseptic necrosis, if the latter exists there.

Before passing on, it may be pointed out that the great difference in the bacteriology of acute abdominal disease from that of other regions consists in the universal presence of a lusty, hardy and rapidly growing bacillus, the bacillus coli communis.

It has been shown, in the first lecture, that the integrity of the wall of the bowel was in a similar position to that of any other tissue in the body, and that its blood supply may be considerably interfered with without causing its death or necrosis. Then the influence of infective organisms was illustrated and shown to be the superlative cause of cases more rapid and acute than those in which there was only interference with blood supply.

It is now my endeavour to bring some light to bear on the various organisms which enter into the fray. In 1891 Fränckel drew attention to the bacteriology of peritonitis. He showed that the exudate derived from the injection of an irritant into the peritoneal cavity was at first sterile, yielding later abundant cultures of organisms. He obtained pure cultures of the bacillus coli communis in nine out of thirty-one cases of peritonitis ; in seven he obtained pure cultures of a streptococcus, and in one a pure culture of a staphylococcus. In 1893, Tavel and Lanz published the bacteriology of fifty-nine cases of peritonitis. Their results showed the presence of the bacillus coli communis alone in over fifty per cent., being about equally as frequent in pure as in mixed culture. Streptococci came next in order of frequency, forming about thirty-five per cent. of the cases, in which they were five times as often in mixed as in pure culture.

Numerous other authors have published work since then,

such as that by Hodenpyl, Welch, Barbacci, Malvosz, and so forth. Almost all of these authors found the bacillus coli communis to be by far the most frequent organism present, and naturally have assumed it to be the cause of the peritonitis.

Dr. Hawkins, in his work on *Appendicitis*, took up the same attitude. But up to now the bacteriologists have not been guided in the interpretation of their results by the surgeon or clinician. Again, with the result of improvements in their science, more accurate observations have been made. With these later methods, Deaver publishes figures in his book on *Appendicitis*, from which the following percentages may be derived; and which illustrate the differences in the bacteriology consequent upon the different clinical forms, namely, acute and chronic inflammation :—

I. CHRONIC APPENDICITIS.

Bacillus coli communis	90%	
ditto, plus staphylococci	6%	7%
ditto, plus streptococci	very rare	
Staphyloccoci	1%	
Streptococci	very rare	
Other organisms	3%	

II. "ACUTE APPENDICITIS."

Bacillus coli communis	70%	
ditto plus staphylococci	15%	26%
ditto plus streptococci	7%	
Staphylococci	4%	
Streptococci	rare	
Other organisms	4%	

The figures show very distinctly that pyogenic cocci are markedly more common in the acute than in the chronic forms both in pure and mixed culture, but, as might be expected, particularly so in the latter. A further distinction

can be drawn. In the figures given by Deaver, the pyogenic cocci are more frequently present with perforating than non-perforating ulcers of the appendix. Similarly, they are found more often in cases of diffuse peritonitis than with local abscess. When regarded from the clinical standpoint, the figures seem to indicate that the more acute a case is, and therefore probably the earlier an operation is demanded, the more frequently are pyogenic cocci found. Barbacci, in 1892, proved experimentally that almost any mixture of organisms injected into the peritoneum and "sufficient time" allowed to elapse, only cultures of the bacillus coli communis could be obtained therefrom. This bacillus grows in common media with such rapidity and luxuriance that slower growing organisms are swamped out and die or remain unnoticed.

III. ACUTE ULCERATIVE APPENDICITIS WITHOUT PERFORATION.

Bacillus coli communis	83%	
ditto plus staphylococci	6%	14·5%
ditto plus streptococci	6%	
Staphylococci	2·5%	
Streptococci	rare	
Other organisms	2·5%	

In conducting a bacteriological examination, with Dr. H. D. Singer,[1] of a case of acute emphysematous gangrene, the original cultures contained a streptococcus and a bacillus, related to the bacillus coli communis, termed by San Felice the bacillus oedematis aerobius. The coccus was recognized in a twenty-four hours culture and coverslip preparations, but after that date it was lost owing to the rapid growth of the bacillus. If this is the condition in the laboratory it should be also the condition inside the

[1] *Path. Trans.* 1901, lii. pp. 42–60.

animals. Further experimentation with gangrene and their organisms convinced me that the pyogenic cocci very materially assist the bacillus to obtain the start or impetus necessary in order for it to begin its career of ill, and then the cocci may be lost entirely, being swamped out by the rapid growth of the bacilli. Mixed cultures were injected into guinea-pigs, but only the bacillus was recovered, the streptococcus being lost, confirming in the animal's body the results obtained in the laboratory.

Applying these two laboratory principles to the conduct of organisms in the body, it is to be expected that the earlier a culture is taken the higher will be the percentage of streptococci to be found in it. Secondly, in the case of acute local appendicitis there will be a higher percentage present than that found in chronic cases. And yet again, in a rapidly spreading infection with diffusion of peritonitis, a yet higher percentage should be seen. Deaver's figures bear out all these considerations. And though one cannot regard it in consequence as proved, there is ample circumstantial evidence upon which to base the suggestion, that the pyogenic cocci are the great causes of acute infective necrosis, and that later they are liable to be killed out by the growth of the bacillus coli communis and be entirely absent from the cultures.

IV. ACUTE ULCERATIVE APPENDICITIS WITH PERFORATION.

Bacillus coli communis	70%	
ditto plus staphylococci	18%	
ditto plus streptococci	3%	30%
Staphylococci	9%	
Streptococci	very rare	

The previous reasoning answers very well for the case of streptococci, for though that is one of the most widely distributed of organisms, it is very easily killed and lost

in mixed cultures, and may literally vanish before a member of the colon group. Staphylococci are a more various group, which contains a number of very hardy members, tenacious of life. They are not nearly so easily killed and lost, and may even "stay out" the lives of the other organisms present in a mixed culture. One would therefore expect them to be found in peritoneal cultures more frequently throughout than streptococci are; and such is in reality the case. The figures bear out in every detail the statements which have just been made with regard to the various frequencies of the presence of staphylococci in chronic appendicitis, perforative and non-perforative appendicitis, appendicitis with localized abscess or diffuse peritonitis. Moreover, in four per cent. of acute cases and one per cent. of chronic, a pure culture of staphylococci was obtained; apparently a very rare condition indeed with regard to streptococci. It is not easy to understand how the colon bacillus escaped culture.

V. GANGRENOUS APPENDICITIS.

Bacillus coli communis	69%	
ditto plus staphylococci	12·5%	25%
ditto plus streptococci	12·5%	
Other organisms	6%	

The position which I would desire to put before you is this, that the action of organisms in the laboratory is carried out in the same way in the body, and that surgery can come to the aid of bacteriology and interpret the clinical results and the actions. It is now disclosed that the more acute the case is, the greater the abundance of the cocci of suppuration. The later the culture is taken, the fewer these become if they are found at all. The obvious inference is that the first step in the pathological catastrophe of perforation is started by these pyogenic cocci;

and owing to the subsequent "swarming" of our natural guest, the bacillus coli communis, their traces are hidden, and they themselves very often die.

In the paper with Dr. Singer, previously mentioned, it was found that the septic organisms in cases of mixed infection of traumatic gangrene are responsible for the bronzing of the skin which precedes the spread of the necrosis, seeming to act as forerunners, injuring the tissues and rendering them an easier prey to the specific bacillus.

The action is the same in the walls of the stomach or appendix as in the arm or leg, with the great exception that the thinness of the walls of the former results in their perforation and in the infection of so vast an absorptive surface as that of the peritoneum. A complete parallel exists between the known septic diseases of the skin and those, largely unknown, of the mucous membranes of the alimentary tract.

An infection from the interior of the bowel, like that on the surface of the skin, by its very nature must be "mixed," consisting of many organisms, and must contain nearly always the vastly predominant organism of the bowel, the bacillus coli communis.

VI. ACUTE LOCAL APPENDIX ABSCESS.

Bacillus coli communis	35%	
ditto plus streptococci	25%	45%
ditto plus staphylococci	5%	
ditto plus both the above cocci	5%	
Streptococcus and other organisms	5%	
Staphylococci	5%	
Other organisms	20%	

Last table constructed from Achard and A. Broca's figures, *Bull. et Mém. Soc. Méd. d'Hôp.*, Paris, 1897, 3s., xiv. 442–443.

There is, however, a further lesson to be learned, which

is of such importance that I may be excused for mentioning it here. The presence of the bacillus coli communis is so widespread in our alimentary canal, that no matter what foreign organisms arrive there, it multiplies so rapidly that it may overcome and completely exterminate them. Applying the same reasoning, which has been used for the processes which go on in the laboratory and the pathological processes in the peritoneal cavity outside the intestine, to the normal processes which take place inside the intestine, we are forced to realize that the bacillus coli communis will tend to destroy all invaders therein. When within the lumen of our intestines it is harmless to the tissues, it must be continually saving us from innumerable ills by destroying harmful bacteria. Its action may be likened to that of the leucocyte in the tissues. And the bacillus coli communis becomes recognized as one of our best friends, the knight-errant of our intestines, the phagocyte of our bowels! But as with all friends, evil communications corrupt good manners, on occasions, intercourse with streptococci and staphylococci lead to its wildly rushing in the breach which these organisms have prepared, and then the leaders of the insurrection are ruthlessly trampled on and killed out by the rabble of lusty bacilli which follow in their wake. History is repeated in the actions of the bacilli of our alimentary tract, and revolutionary leaders are killed by the very rabble they raise.

One point further, as a consequence of the above it is doubtful if the cultures made from a case represent all the organisms which may have been concerned in the disaster. Caution must always be exercised in making deductions from the results so obtained.

Mr. Watson Cheyne in an admirable article upon inflammation says, "The effects of the organisms on the tissues vary

with the kind that is introduced ; for example, the staphylococci tend to form circumscribed abscesses, while the streptococci are especially concerned in the production of diffuse spreading suppuration." This is, of course, a perfectly general statement. Both kinds of pyogenic organism acting on a comparatively thin walled organ like the stomach will be very apt to cause a perforation. But the action of the former will be to produce a local perforation, whilst that of the latter will be to produce perhaps a perforation which is merely a local sign in a more widely spread area of infection. In the former case the areas of infection and perforation coincide, and, in the latter, they do not.

Dr. Cayley, in the *Path. Soc. Trans.*, 1902, p. 282, records a case of streptococcus infection of the stomach, phlegmonous gastritis. The change was especially noted in the pyloric end. Both mucous and serous membranes were intact, although the patient died of general peritonitis, due to the diapedesis of organisms. Dr. Foulerton describes a case of pneumococcic gastritis, on p. 286 of the same volume. In this, the mucous membrane was necrosed, contrasting with its more or less healthy condition in Dr. Cayley's case. Again there was no perforation. Cases of epidemic streptococcus enteritis have been described, notably by Booker,[1] which have not been at all necessarily fatal. Streptococci can give rise on the skin to erysipelas, which is a capillary lymphangitis ; they can also cause cellulitis, a lymphangitis of the cellular tissue. The former is transient and can disappear entirely, and I cannot help thinking that many acute gastrites, appendicites and enterites are of a similar erysipaloid nature. The subject of the identity or relationship of the streptococcus erysipelatis with ordinary streptococci of suppuration will not be entered upon.

[1] *Centralblatt* and *Bakt. u. Parasit.*, 1889. Bd. v., S. 316.

The tale of the staphylococci is different, and an organism which typically causes a circumscribed abscess will in the gastric or intestinal walls cause a local perforation. These cocci are hardier than the streptococci are, and will, I believe, be found to be the chief cause in the production of the acute infective necrosis of the alimentary tract. Of direct proof I can offer none, as my own unaided bacteriological efforts were such as could only be managed in moments snatched from the extremely busy life of the Resident Assistant Surgeon of St. Thomas' Hospital; and though I have made a number of examinations of peritonitis, the exudation in strangulation both inside the abdomen and in hernia sacs, they are such that, though they may help to form ideas, I do not consider them worthy of being brought forward. It may be mentioned that my results guided me to the belief that pyogenic cocci will be found to be the great originators of perforations and gangrenes of the alimentary tract, and also to the uniformity of the pathological process, acute infective necrosis, in appendicitis, perforated gastric, duodenal and other ulcers, strangulated herniae, etc.

XXIV. THE BACTERIOLOGY OF STRANGULATED HERNIAE

Bundschuh, in the *Beiträge zur Klinische Chirurgie*, xxxi., Heft 2, found bacilli present in the fluid in the sacs in sixty per cent. of cases. Apparently this was the bacillus coli communis. The observations which I was able to complete were fifteen in number. Cultures, only containing bacilli, were only obtained in about thirty-three per cent of cases. But the result seemed to depend mainly upon two factors, namely, the severity and the duration of the strangulation.

The presence of the bacillus seems to make no difference in the prognosis or the primary union of the wound. Professor Welch years ago demonstrated the presence of the bacillus coli communis in surgically clean laparotomy wounds. It is, therefore, possible to go further than the usually enunciated dictum, that "all work goes to show that this organism is not pathogenic when it is present in the normal intestine, but when the intestine is injured, or the organism enters an unusual or abnormal situation of the body, it can set up various lesions." [1] To this may now be added, that it has been found in the peritoneal cavity in cases of intestinal obstruction and in the sacs of strangulated herniae without producing any obvious result from its presence. It may not have been doing harm in a number of places where it has been found. In a case of acute infective necrosis in a strangulated hernia, reported for the first time in these lectures, "cocci" were found in the coverslip preparations as well as bacilli, but in the cultures only bacilli were found. It is therefore not clear that, unaided, the ordinary colon bacillus of our intestine does us more harm than good. In fact, I believe, that it does us infinitely more good than evil turns, and certainly is a much libelled organism. But the ordinary colon bacillus has many relations, some of which can be extremely badly behaved. It is by no means easy to make sure of the "ordinary" bacillus coli communis. That is to say, to distinguish harmless, though they need not remain so, from harmful members of the colon group.

The dependence, for the initial start of the action of the colon bacillus on the bowel wall, upon "injury" alone is not so decided as has hitherto been taught. The proof of its existing without apparent harm in the tissues, as in intestinal obstruction and strangulated herniae, has been

[1] Muir and Ritchie's *Bacteriology*.

adduced. Again, by the action of the bacillus typhosus in enteric fever the mucous membrane of the bowel is seriously injured, and though in this disease the colonies of bacillus coli communis seem, or are said actually to be, numerically increased, yet perforations and gangrene of the bowels are not so common. Yet again, in tuberculous enteritis, perforation and gangrene are not nearly so common as would be expected, if the bacillus coli communis merely needs the tissues to be "injured" in order to begin its work of havoc upon them!

"It was formerly taught that there were no micrococci in the intestines. Recently they have been demonstrated." So says a recent bacteriological text-book. And there is every reason why they should not be found in ordinary cultures, and perhaps even on coverslip preparations, having been killed out by the growth of the colon bacillus. Yet, I believe that we must look to pyogenic micrococci for the starting point of cases of acute perforation and gangrene, i.e. acute infective necrosis, in whatever part of the alimentary tract it may be situated.

XXV. THE RELATION TO PHAGAEDENA, CANCRUM ORIS, ETC.

With the wild ardour of youth I once set out to attempt to elucidate the bacteriology of gangrenes, phagaedena, cancrum oris, etc. The sobering effects of this attempt lie in two chief directions. Firstly, in all cases a mixture of organisms was obtained and in the process of isolation and separation of these bacteria one either lost a lot of the organisms, or they, in varying degrees, their power of producing disease. And secondly, I doubt if it is often possible to attribute these diseases to the result of the action of any

specific organism or even organisms. These infections are of their very nature and situation "mixed" infections, and the omnipresent and ever represented organisms in them are the pyogenic cocci. That is to say, they are pre-eminently septic infections.

Bacteriology, as it now exists, offers at the best but an incomplete account; as has been said in a recent bacteriological text book, "it should be borne in mind that many of the bacteria which can be obtained in coverslip preparations cannot be cultivated." And it may be added that many, even the most important, may be lost even before the coverslip preparation can be made.

Oberwarth [1] records a case of primary gangrene of the tonsil, which proved fatal. Streptococci alone were found in the culture. In fact, streptococci and staphylococci are universally acknowledged to play a leading part in gangrenous dermatitis, stomatitis, tonsilitis, etc., i.e., where the colon bacillus scarcely exists; but owing to the vigorous growth of that hardy bacterium their importance has not as yet been recognized in gastric and intestinal necroses, and direct proof is still wanting. It is of considerable interest and supports my suggestion, that, in situations such as the tonsils and skin whence cultures need not be crowded out with the bacillus coli communis, if it is present at all, pyogenic cocci alone or predominantly are found. This may be regarded as another item of evidence indirectly in favour of the prime importance of pyogenic cocci in the production of acute infective necrosis of the stomach, duodenum, appendix, etc. The differences in the bacteriology of the results of their action, within and without the body, being due to the fact that in the alimentary canal their presence is masked by the colon bacilli. Thus it is suggested that

[1] *Deutsche Med. Woch.* Ap. 23 and 30, 1903.

the internal lesions mentioned above are septic processes such as are well known on the skin.

XXVI. ACUTE INFECTIVE NECROSIS OF THE ALIMENTARY CANAL IN VETERINARY WORK

It is my desire to bring home to my hearers the value of a careful examination of the records of veterinary surgery and pathology. A most fascinating study is afforded in this way, and one which will do much to aid and extend the experimental researches. Acute infective necrosis occurs in animals in the shape of gangrene of tracts and perforations of the alimentary canal. Unfortunately, as yet, veterinary pathology is not in so advanced a state, or perhaps better, the knowledge of it is not so widely spread as is that of human pathology, and the records and descriptions are consequently sometimes difficult to understand. Speaking generally, the diseases of animals are not as yet well worked out. But the future will doubtless furnish far greater stores than the somewhat meagre accounts now available.

THROMBOSIS OF A MESENTERIC VEIN.

I found the record of one case of mesenteric thrombosis in animals described by Connochie, in the *Veterinary Journal* of 1898, xlvi., p. 324. The title of the paper is "Vomition in the Horse due to Mesenteric Embolism," and is very misleading. The subject was a valuable hackney filly, rising three years old, which was in good condition. For two days she was noticed to be eating sparely; on the third day she was found lying down and rolling over and over; and on the fourth day she died. At the post-mortem examination an ante-mortem clot was found in the anterior mesenteric vein with resulting gangrene of the "intes-

tines." The case in reality seemed more acute than many of those of venous thrombosis occurring in man.

SUPPURATIVE TYPHLITIS.

A case of typhlitis such as must correspond with our appendicitis was also found, and is reproduced in this section. A horse was suddenly seized with violent colic and died in "some" days. At the autopsy "a quantity of reddish serum was found in the abdominal cavity, and flakes of lymph upon the intestines. On removing the caecum there was found at the place of attachment an induration from one to two centimetres thick, and in this an abscess the size of 'a nut' which contained grey, thick pus. Along the whole extent of the crook of the caecum the wall of the gut was thickened. The mucous membrane was very much inflamed, deep red in colour; the epithelium came away in flakes leaving a blackish corium."

The importance of this case in corresponding to appendicitis in man has been remarked on in a section of the second of these Lectures.

PERFORATIONS OF THE STOMACH.

Perforated gastric ulcer in animals is a subject covered with much confusion. Acute abdominal disease in animals seems largely to consist of three kinds, namely, colic, ruptured stomach and twisted guts. From the descriptions it is not easy always to decide upon the case as to whether the condition was inflammatory or the result of true bursting of the stomach which does occur in animals. Three genuine cases were found.

Journal of Comparative Medicine, 1897, xviii. p. 716. A horse died from "colic," but was found to have an irregular zigzag ulceration along the greater curvature of the stomach. There was also another ulcer near the pylorus.

In the first ulcer was a perforation about one inch long which had caused peritonitis and death.

John Freeman, F.R.C.V.S., *The Veterinarian*, lxxiv., 1901, p. 108. A Scotch deerhound had been ailing for some time with wasting. At first the diagnosis was that of worms, later pernicious anaemia. Post mortem, a perforated gastric ulcer the size of a half-crown was found. The other viscera were healthy.

Mosser, *Bull. de la Soc. Centrale de Méd. Vétérinaire*, February 23, 1903. At the post-mortem examination of a cow, 10–12 ulcers were found along the greater curvature of the stomach, varying in size from that of a pea to that of a franc piece. One of these had perforated causing peritonitis and death.

Ruptures of the stomach have been reported in far greater numbers than have true perforations, and occur always along the cardiac end of the greater curvature, being sometimes as much as 18 inches long. Mr. Malcolm, F.R.C.V.S., says that it is not an unfrequent occurrence, and that it is due to the violent contractions of a relatively small and weak stomach which cannot relieve itself by vomiting. In a certain number of these ruptured stomachs there is evidence of previous pathological change. For instance, in the case of Littlewood's, *Veterinarian*, 1894, xxxiv. p. 291, the walls were thickened and softened by parasites.

PERFORATION OF THE SMALL BOWEL.

Professor Penberthy, in the *Journal of Comparative Pathology*, 1896, p. 25, says that in enteritis "mortification may occur in 8–10 hours." Such rapidity demands the assumption of the action of microorganisms.

Perforations of the small bowel seem more common than they are in any other situation. In the *Journal of Com-*

parative Medicine, H. H. Dell records a case of three perforations in the ileum, associated with the presence of taenia saginata. The subject was a Gordon Setter bitch. He quotes Cadeac, *Rev. Vétérinaire*, 1888, as reporting a case of perforation in the duodenum of a terrier, due to a "taenia." Lahoque, *Receuil Vétérinaire*, 1888, describes three perforations in the small bowel of a dog, due to saenia serrata.

In the *Journal of Comparative Medicine*, 1899, xx. p. 379, it is reported that a seven-year-old bay mare died, and at the post-mortem, a perforation of the ileum, half an inch in diameter, was found, and general peritonitis. The ulcer was solitary, and there was no obvious cause.

PERFORATION OF THE LARGE BOWEL.

Captain Martin, in the *Veterinary Journal*, 1903, N.S., vii. pt. 41, p. 273, gives a most interesting account of a unique case. A chestnut mare was found to have an intense inflammation of the caecum, or typhlitis with perforation, and through that perforation a number of coils of small intestine had passed into the caecum and become strangulated!

Davis, *Veterinary Journal*, 1897, xliv. p. 170. The "floating colon" was found to be greatly inflamed over a patch about eight inches long and perforated. A calculus of 2⅝ lbs. weight was free in the abdominal cavity.

Professor Dewar, *Veterinarian*. A gelding, 8 years old, was found, post mortem, to have a "ruptured" colon 10 inches in length. The walls were 3–4 times as thick as normal.

Neyland and Leblanc, *Veterinarian*, 1896, lxix. p. 48. Post mortem on a mare who had had "colic." One metre from the anus the gut was ruptured, the "tear" being 10 inches in length and a large intestinal calculus was in the perforation!

XXVII. PERITONITIS IN ANIMALS

THE natural thing to look into to find support for the previously recounted cases was to refer to the records of instances of peritonitis and abdominal abscess. Unfortunately, the origin of these diseases is not always known or looked for. For instance, in the *Vet. Journal*, 1900, N.S. 1, p. 295, a report of a case is given from the Berlin *Thierarzt Wochenschrift* of a cow which had a large abscess in the peritoneum which discharged per rectum. There was "quite a litre of greyish yellow stinking filthy pus!" The sinus closed, and the animal recovered. The origin of the abscess is not known, but had such an accident occurred in a young human being, there would be little or no doubt that the appendix would be at the bottom of the trouble.

The presence of peritonitis, unfortunately, renders the animal unfit for food, and in consequence has not the importance in the veterinary world that it has with us. Brett, *Veterinarian*, 1889, lxii. p. 619, says that peritonitis is not common in cattle. And he gives accounts of three cases which were diagnosed as faecal impaction. Post mortem, two were called idiopathic peritonitis, and the third was regarded as due to "cold following calving." Littlewood, in the *Veterinarian*, 1890, lxiii. pp. 204–207, describes an epidemic of peritonitis which occurred among the horses of the Egyptian police reserve, in which about fourteen died of peritonitis for which no cause was demonstrated. Poisoning was excluded. All cases were acute, and the only definite thing noticed was that the inflammation was mostly in the pelvis and the "double colon." Wohmuth, *Veterinarian*, 1901, lxxiv. pp. 29–31, records three cases, one being due to a nail in the rumen;

another to an abscess in the wall of the rumen; and the third consequent on parturition. The other records merely illustrate its occurrence after operation or parturition.

Looking through these cases, veterinary surgery seems in the same state as that in which the surgery of mankind was some twenty years ago. The idiopathic peritonitis of the latter has turned out to be mainly due to the disease of the appendix, which our predecessors had overlooked. It remains with the future to see if those of the animal will find a similar explanation. In discussing the pathology and physiology of the caecal region, it was shown that there was every reason to expect inflammation in this place, and also the presence of lymphoid tissue. Beyond the influence of diet and position, there seemed to be no particular reason why this region should be very often affected in the biped and escape in the quadruped. It is to be expected that the disease, when closer attention is paid to it, will be far more frequent in the latter than it now appears to be. Veterinary surgeons have not, as yet, recognized anything of the kind. It has also been shown that the collection of lymphoid tissue in the caput coli of animals is the analogue of the appendix of man. And, therefore, it should also have some analogous diseases. It must have a somewhat similar *rôle* with regard to the action and growth of microorganisms. And it will, therefore, be liable to like inflammatory changes. These have not been fully recognized, but I have brought forward a case of abscess of the wall of the caecum, examples of localized intraperitoneal abscess near it and in the pelvis, peritonitis limited to and especially marked in the same region. And it is suggested that these have an origin analogous, to what they have in man, i.e. the appendix, in the lymphoid tissue of the caecum.

XXVIII. CONCLUDING REMARK

Professor Clifford Allbutt once wrote to me: "I warmly welcome any attempt to get away from anthropocentric medicine, to escape from the ptolemaic phase into the cosmic."

Let this sentence close the remarks that I have made upon the subject.

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