THE LETTSOMIAN LECTURES ON PERITONITIS.

Delivered before the Medical Society of London, January, 1894.

By FREDERICK TREVES, F.R.C.S.,

Surgeon to, and Lecturer on Surgery at the London Hospital; Examiner in Surgery, University of Cambridge.

LECTURE II.—THE CAUSES OF PERITONITIS.

OF the many classifications of peritonitis which have been attempted, those are the most satisfactory in which the etiology has been taken as a basis. Even when this method of division has been regarded, there is no disease which has been broken up into more varieties or burdened with more differentiation than has the affection under consideration. In the very elaborate monograph on peritonitis by Spillmann and Ganzinotti,¹ no fewer than twenty-six different forms of the disease are described, and even these are subjected to many and complex subdivisions. Every addition to our knowledge of inflammation of the great serous membrane tends to make the classification of its various forms more and more simple.

It has now become evident that peritonitis depends almost entirely, if not entirely, upon infective processes, and that these agencies are concerned, directly or indirectly, in every form of the disease. There are those who maintain that there is no form of peritonitis which is not due to infection, and although the data upon which such an assertion is based are not entirely perfect, there is yet much presumptive evidence in favour of its accuracy. Indeed, the domain of non-infective peritonitis is becoming rapidly more and more dubious.

In dealing with any classification in which an infective process is the main element, there are many general considerations to be borne in mind. On the one hand, there is the dose of the poison, and on the other hand there is the soil into which it is introduced. The dose must obviously be liable to considerable variations, not only in amount but also in virulence, while the conditions offered for growth by the inoculated body must differ to even a wider and more complex extent.

The resistance of the individual to the growth of septic organisms is influenced by a multitude of circumstances, the value of which is scarcely susceptible of being formulated. There are questions of age and of health, and of such unfavourable affections as Bright's disease, diabetes, and alcoholism. There are local conditions which lend themselves to the more ready cultivation of germs, such as extensive damage to the peritoneum, and extensive interference with its functions of absorption, or at least with that ill-understood power it possesses of dealing with noxious micro-organisms. There are the presence of ascites, and the coexistence of extravasations of blood, bile, or intestinal matter in the peritoneal cavity. There is, finally, the entirely undefined peculiarity of the individual which appears to alone render one man susceptible and another almost exempt.

In the great majority of instances peritonitis depends upon what Tavel and Lanz² call "continuity infection." By this is understood the direct spreading of the infection to the peritoneum from a neighbouring tissue other than the serous membrane. Under such heading would be included peritonitis following hernia or any visceral lesion or disease capable of producing the trouble, puerperal peritonitis, most forms of pelvic peritonitis, and certain varieties of tuberculous peritonitis. It would exclude the larger number of the cases of inflammation of the serous membrane which follow accidental or operation wounds.

With peritonitis due to infection by continuity must be included what is conveniently termed by the authors just named "chemical peritonitis." Here the peritoneum is invaded not so much by the micro-organisms themselves as by their chemical products. The serous membrane may absorb these products from adjacent tissues, for in a few examples of peritonitis no micro-organisms have been discovered in

the exudation after careful examination by competent observers.

CLASSIFICATION.

The following classification of peritonitis, according to its cause, appears to be justified by our present knowledge of the subject.

1. *Peritonitis due to Infection from the Intestine.*—Under this heading are included most cases of peritonitis associated with hernia, with intestinal obstruction, and with perforation; peritonitis due to any form of ulceration of the bowel; to enteritis; to cancerous growths of the gut and to troubles in the appendix. There will also be included peritonitis due to inflammatory changes in the biliary canals, and some forms of peritonitis following upon operation.

The micro-organism which is usually found associated with these different forms of peritonitis is the bacterium coli commune.

2. *Peritonitis due to Infection from Without.*—This division will include puerperal peritonitis, peritonitis consequent upon inflammatory troubles in the genital organs or in the parietes of the abdomen, and some forms of peritonitis following upon operation.

The micro-organisms usually associated with the varieties considered under this heading are pyogenic cocci, and notably the streptococcus pyogenes.

3. *Peritonitis due to the Pneumococcus.*

4. *Tuberculous Peritonitis.*

5. *Peritonitis of a Doubtful Nature.*—Under this purposely indefinite heading it will be convenient to discuss (a) peritonitis due to irritants; (b) forms of the trouble reputed to depend upon rheumatism, gonorrhœa, syphilis, Bright's disease and alcoholism; and (c) peritonitis met with in the newly born.

In order to form an approximate idea of the relative frequency of different forms of peritonitis, two tables are appended. The first is an analysis of 100 cases of peritonitis taken from the records of the London Hospital. The only test applied in the selection of these cases was evidence that the clinical details and the accounts of the necropsy (in the fatal examples) were as complete as possible. The latter accounts are from the pathologists' reports. The clinical details in the 100 cases are practically complete as regards temperature, account of vomiting, state of the bowels, pulse, respirations, etc., and in the majority of instances a daily statement of the patient's symptoms is given. For the making of this laborious collection of cases I am indebted to Mr. A. Hallidie, F.R.C.S.

TABLE I.

Peritonitis.	Number of Cases.	Sex.		Average Age.	Deaths.	Recoveries.	Time of Death.				Exudation in Fatal Cases.		Vomiting Absent or very Slight.	Bowels Loose.		
		Males.	Females.				In 36 hours.	In 48 hours.	3rd to 4th day.	5th to 7th day.	After 7th day.	Commencing.			Plastic.	Serous.
With hernia	10	3	7	52	10	0	0	0	6	2	2	3	3	0	2	2
With perforation of bowel ...	9	4	5	40	9	0	4	2	0	3	1	2	3	3	3	3
With gross disease of bowel ...	6	4	2	40	6	0	3	0	0	3	1	1	2	1	2	3
With disease of appendix ...	22	19	3	22	6	16	0	0	0	5	1	0	1	1	3	4
Starting from lesions about the pelvis	18	5	13	36	11	7	3	1	1	6	0	3	1	0	2	6
Following abdominal wounds By extension from the pleura	14	2	12	36	14	0	0	1	5	5	3	2	6	1	2	4
Tuberculous	8	2	6	20	5	3	0	0	0	1	4	0	2	3	1	0
Diffuse cancer of peritoneum Causes of peritonitis unknown	2	2	0	42	2	0	0	0	0	0	2	0	0	2	0	1
	100	43	57		70	30	10	4	14	23	19	11	21	12	15	23

The second table is from the monographs by Fraenkel and Tavel and Lanz.⁴

TABLE II.—Cases of Peritonitis in Man in which Micro-organisms were found in the Exudation.

	Fraenkel.	Tavel and Lanz.	
	Found Alone.	Found Alone.	In Association.
Bacterium coli commune	11	15	16
Streptococcus ...	7	3	15
Staphylococcus ...	1	2	6
Pneumococcus ...	1	0	2
	20	20	39

I.—PERITONITIS DUE TO INFECTION FROM THE INTESTINE.

As already stated, the micro-organism which appears to be usually answerable for the numerous forms of peritonitis met with under this heading is the bacterium coli commune. This most remarkable and common bacillus was first described by Escherich⁵ in 1885; its definite pathogenic properties remained imperfectly recognised until 1889, when they were expounded by Laruelle,⁶ by Roux and Rodet,⁷ and many others. Within the last few years the literature appropriated to this little organism has become so extensive as to be almost overwhelming.⁸

The morphology of the bacillus—with which we have no concern in this place—has been elaborately dealt with by Wurtz,⁹ Macaigne, and other writers. It has been shown to be identical with the bacillus pyogenes foetidus obtained by Passet in 1885, from the stinking pus of a perirectal abscess. It has been proved to be the same micro-organism as the bacillus lactis aërogenes, and the pyogenic urinary bacterium described by Clado and Albarran in 1891.¹⁰ In many particulars it closely resembles the bacillus of typhoid, but the distinguishing features of the two micro-organisms have been fully pointed out by Trambusti,¹¹ Péré,¹² and Wurtz.¹³

The bacterium coli commune exists normally in the human body, and is found in individuals in a state of perfect health. It has been demonstrated to be present along the whole length of the alimentary canal, from the mouth to the anus.¹⁴ Its natural habitat is the bowel. It is said to be especially numerous in the duodenum¹⁵ and in the colon, and it has been found in the bile passages. It is certainly the most abundant and the most constant of the bacteria found normally in the human body, and has been estimated to form 95 per cent. of the micro-organisms met with in the bowel of man. Sternberg¹⁶ gives a list of forty-eight micro-organisms which have been isolated from human fæces or the contents of the intestine. Of this number twenty-five are non-pathogenic, and of the pathogenic forms not a few represent the same bacillus under different names. There is no doubt that special names have been given to micro-organisms which are mere variations of one common form.

This matter is well dealt with in the excellent monograph by Tavel and Lanz,¹⁷ a work which forms by far the most important recent contribution to the subject of the etiology of peritonitis. Tavel and Lanz regard the term bacterium coli commune as a generic term applicable to an extraordinarily numerous family of bacteria. They themselves indicate some twenty varieties in this great family. These authors also have pointed out that the colon bacillus is sometimes mobile and possessed of cilia, and sometimes immobile. They do not, however, associate any pathogenic differences with these two forms, which they have admirably figured.

The bacterium coli commune has a striking feature in that it appears to vary in virulence. So far as experiments upon animals are of value it would seem to be quite harmless when taken from the normal intestine. A culture of the bacillus so obtained has no effect when injected into the peritoneum of animals.¹⁸ If, however, the bowel become the seat of certain diseased conditions (or it may almost be said of any diseased condition), then the bacillus becomes at once virulent. Virulence has been found to be developed in cases in which the bowel was obstructed, strangulated, or inflamed, in venous congestion of the gut, in œdema of the bowel wall,

in diarrhœa, in typhoid fever, and in cholera. In a case of diarrhœa produced by tartar emetic this colon bacillus was found to have become virulent.¹⁹ It also appears to develop qualities for evil in instances of advanced constipation.

In any case experiments show that the virulence when once developed may vary considerably in degree. Tavel and Lanz describe an interesting case of localised abscess due to mischief in the vermiform appendix. The pus was swarming with the colon bacillus. Quantities of the same bacillus were found in the peritoneum, but there was no peritonitis. Much gas is discovered in the peritoneal cavity. When the intestine is healthy the bacterium coli commune has little disposition to escape through the gut wall or to invade the tissues of the body after death. If, however, the intestine be damaged or diseased, then the bacterium shows an active inclination to penetrate through the bowel wall, and is usually found to have extensively invaded the tissues after death.²⁰ The disposition of the bacillus to pass through the intestinal wall under the circumstances just stated is well illustrated in examples of slight degrees of strangulation of the bowel produced by experiments in animals.²¹

The effect of injecting a culture of the virulent colon bacillus into the peritoneum of animals varies—other things being equal—according to the dose. In the slightest cases the animal, after an illness in which diarrhœa is a symptom, recovers. In another grade a localised purulent peritonitis is produced, which follows a chronic course. In a third degree death is rapidly brought about by a diffuse fibro-purulent peritonitis. In instances in which a large dose is employed, the animal dies of acute sepsis before any phenomena of peritonitis are produced. When peritonitis is produced, it is usually purulent. The presence of fluid in the peritoneum, and especially of blood, greatly aids the development of peritonitis in these experiments.²² The activity also of the bacillus is much increased, if it be injected with sterilised fluid from the intestine as a medium. If a sterile saline solution be used the effect is diminished.²³ The endothelial cells of the peritoneum do not appear to act as phagocytes, for in various experiments in which the colon bacillus was injected into the peritoneal cavity, it was never once found by Ziegler within these cells.

It has been clearly demonstrated that the bacterium coli commune can produce pus after inoculation. It has been found in man in a state of almost pure culture in the pus from an ichthio-rectal abscess. So far as the human subject is concerned, it has been shown that these various forms of peritonitis which are assumed to be of intestinal origin, depend mainly and in many instances solely upon the bacterium coli commune.

In peritonitis associated with hernia, the colon bacillus is the micro-organism most commonly found in the sac. This is the case in all stages of strangulation, and is independent of the character of the fluid, the bacilli being obtained in cases in which the fluid in the sac is copious and opaque, as well as in those in which it is clear and scanty. In fifteen examples of hernia, Bönnecken²⁴ found the bacterium coli in the sac in thirteen instances.

The bacillus so commonly found in the hernial sac by Clado,²⁵ in 1889, is now known to have been the colon bacillus. Tavel and Lanz examined bacteriologically the exudation in the sac, or in the adjacent peritoneum in twenty-one cases of strangulated hernia, in two cases of "hernial hydrocele," and in one case of suppuration of the sac. In eighteen instances they discovered no micro-organisms of any kind. In five examples—including three in which the gut was apparently not involved in the rupture—bacteria were found, the most common being the bacterium coli commune. The authors consider the cases unassociated with micro-organisms to be examples of "chemical peritonitis," and to be due to the products of bacteria rather than to the germs themselves; they do not, however, commit themselves to the opinion that the bacillus chiefly concerned is that now under consideration. Laruelle²⁶ has demonstrated the bacterium coli commune in gangrenous hernia, and in gangrene of the gut produced artificially in animals.

Audin²⁷ has shown, by a series of most carefully conducted experiments, that micro-organisms can pass through the gut wall into the peritoneal cavity, when the strangulation is of quite a slight grade, and is very far short of producing even

a limited gangrene. Bönnecken's²⁸ investigations give practically the same result. Cornil²⁹ found bacteria actually in the substance of the wall of a partly necrosed intestine. Tavel and Lanz are of opinion that while micro-organisms cannot pass through the wall of healthy bowel, they are very ready to escape if the gut be the seat of even a slight lesion. It is only fair to say that Orth's³⁰ experiments do not support these very widely held opinions.

Macaigne gives an account of thirty-five cases of peritonitis of intestinal origin, in which the colon bacillus was the only organism present, or at least the principal one. These thirty-five cases are distributed as follows:—

With troubles in the appendix	10 cases
With typhoid fever	9 "
With ulcerative enteritis	6 "
With perforation	3 "
With cancer of colon	3 "
With hernia	2 "
With thrombosis of mesenteric vessels	1 "
With ulceration of gall bladder	1 "

Total 35

The researches of Welch, Ziegler, Fraenkel, and Malvoz all bear in the same direction.

The colon bacillus has been shown by Cornil, Babes, and others to be almost exclusively the bacterium concerned in perforative peritonitis. Barbacci³¹ states also that it is practically the only bacillus to be produced in cultures from the exudation in perforative peritonitis in man. The same applies to perforative peritonitis produced experimentally in dogs. In this form of peritoneal inflammation, the other micro-organisms found in fæces develop in the serous cavity, but only for a time. The bacterium coli commune alone survives. Barbacci points out that the liberal development of this bacillus is greatly favoured by the presence of such a medium as is provided by the fluid contents of the intestine, a material which readily damages the peritoneal endothelium. He considers that death is due to absorption partly of the bacterial products, and partly of the noxious chemical matters contained in the intestinal fluid which escapes into the belly cavity.

In the same way it has been demonstrated that this micro-organism is the active agent in the majority of cases of peritonitis starting from the appendix. Adenot³² gives four cases of so-called appendicitis attended with suppuration, in the pus of which the colon bacillus was found in a pure culture. In another instance in the exudation upon the surface of a non-perforated appendix containing a concretion, this bacillus was the only micro-organism found. Tavel and Lanz, in the valuable work already quoted, deal with twenty-three cases of trouble in the appendix. An examination was made either of the pus within or without the appendix, or of the exudation into the peritoneal cavity immediately around the diseased process. The material for this bacteriological examination was obtained during the patient's life. In three instances no micro-organism was found. In nineteen examples the colon bacillus was found either alone or in association with other pathogenic germs. The streptococcus was found in several cases, the pneumococcus was met with in two examples, but not in pure culture. The staphylococcus pyogenes citreus was in one instance the only micro-organism discovered. Tavel and Lanz give five cases of internal strangulation of the bowel and one case of resection of the gut. In three of these the colon bacillus was found in the exudation of the peritonitis which resulted.

Chemical Peritonitis.—One of the five cases affords a good example of "chemical peritonitis." The patient, aged 40, had symptoms of intestinal obstruction. Laparotomy was performed on the seventh day. Strangulation by an omentum band was discovered; there was general peritonitis; the abdomen was drained. The fluid escaping from the drainage tube proved to be sterile on cultivation. The patient recovered.

Bignami,³³ Bastianelli,³⁴ Le Sage, and Macaigne³⁵ have shown that in suppurative or ulcerative troubles of the gall bladder or biliary passages the bacterium coli commune is often the only bacterium found. Roswell Park³⁶ has also furnished a case illustrative of this association. In three examples of peritonitis following suppurative inflammation of the gall bladder, Tavel and Lanz found the bacterium coli commune in two instances and pyogenic cocci in the third.

This bacillus has also been found in cases of peritonitis following operation, and on this subject Dr. Welch writes as follows:³⁷

Its presence several times in pure culture, in laparotomy wounds treated aseptically, although apparently not a source of serious trouble, was not a matter of indifference. It was generally accompanied with moderate fever, and a thin, brownish, slightly purulent discharge, of somewhat offensive but not putrefactive odour. The smooth and rapid healing of the wound was interfered with. In some of the cases there was evidence of intestinal disorder.

In the cases of peritonitis after laparotomy in which the colon bacillus is found, it is probable that such an injury had been inflicted upon the intestinal peritoneum at the operation as to permit the bacterium to escape through the bowel wall. It has been shown that it requires but little damage to the bowel or to its serous covering to allow of such escape.

It is well known that pneumonia is not an uncommon sequel of peritonitis, and it is possible that in certain of these cases the colon bacillus is the active agent. It has been found in the pleura when pleurisy was present (Laruelle), and has been obtained by puncture from the hepatised lung some days before death from peritonitis (Macaigne). Fisher and Levy give two cases of strangulated hernia with pulmonary complications, and in both instances the colon bacillus was found in the affected parts of the lung.³⁸

Finally, it has been assumed that some of the constitutional symptoms attending severe constipation may be due to the absorption of the products of this widely diffused bacillus.

The exudation associated with the bacterium coli commune when it attacks the peritoneum is often characteristic. It is well seen in cases of inflammation about the appendix. The fluid is at first clear and greenish looking. It then becomes greenish yellow, thin, semi-opaque, and foul smelling. If it pass on to suppuration the pus has no especial characters except that it is frequently offensive. In chronic cases there is sometimes produced a white, soft custard-like material which is often met with in operations upon the appendix during a quiescent period and which I do not think is changed pus nor a residuum left by pus. In some long-standing cases I have scraped away a full teaspoonful of this matter from about the cæcum.

In concluding the account of this variety of peritonitis it may be pointed out that the irritant which induces the peritonitis and which reaches it from the intestine may follow more than one path. (1) Bacteria may escape through a perforation in the bowel; (2) they may pass through the wall of a segment of intestine which has been in some degree damaged but not perforated; and (3) chemical products of bacteria may reach the peritoneum by either one of these routes.

II.—PERITONITIS DUE TO INFECTION FROM WITHOUT.

It has been already observed that this variety of peritonitis usually depends upon pyogenic cocci. Of these the streptococcus is the one most usually met with. It is often alone, or it may be found in association with micro-organisms, among which, however, it is the chief.

That the streptococcus is the usual agent in puerperal peritonitis has been shown by Widal, Bumm, Fraenkel, and Courtois-Suffit.³⁹ It has been met with in association with putrefactive bacteria in examples of perforative peritonitis by Prédohl and others. The staphylococcus is very rarely met with alone in cases of peritonitis, and is usually associated with the more virulent form of coccus. (See Table II.)

So far as experiments go it has been shown that injections of pyogenic cocci into the peritoneum act more vigorously if mixed with a fluid difficult of absorption, or if introduced with a corrosive fluid capable of damaging the peritoneal endothelium.

In twenty instances reported by Pawlowsky,⁴⁰ in which the staphylococcus was introduced into the abdomen of animals, death followed in eighteen cases from purulent or hæmorrhagic peritonitis. A drop, or even half a drop, of fluid from a case of puerperal peritonitis may kill an animal when injected into the abdomen, whereas in a control experiment a syringe of the exudation from an example of peritonitis after operation may not lead to a fatal result.

The pyogenic cocci, and most usually the streptococci, have been met with in cases of peritonitis after operation in man, and experiments upon animals show how the noxious

effect of those micro-organisms may be assisted by extravasations in the peritoneal cavity, by long exposure of the peritoneum, and by damage to its delicate surface. In the operation cases it is most probable that the streptococcus gains an entrance through the wound. In puerperal peritonitis it, no doubt, reaches the abdomen through the pelvic organs following the veins or lymphatics, or the Fallopian tube.

In the rare examples of peritonitis associated with erysipelas of distant parts (such as the face) the streptococcus must be assumed to reach the serous membrane through the blood.¹¹

III.—PERITONITIS DUE TO THE PNEUMOCOCCUS.

This variety of peritonitis has not yet been so soundly demonstrated as to possess an indisputable existence. In the first place, the connection of the pneumococcus with pneumonia is a subject about which there is much conflicting evidence. In the second place, peritonitis is a very rare sequel to pneumonia, although it is true that pneumonia is not an uncommon complication of peritonitis.

The pneumococcus has been found in the peritoneum in a large number of the subjects of fatal pneumonia, and yet the serous membrane has remained sound. In 151 necropsies in which the pneumococcus was demonstrated Netter⁴² found only two cases of peritonitis. However, cases of peritonitis have been reported by Weichselbaum,⁴³ Courtois-Suffit,⁴⁴ Sevestre,⁴⁵ Nélaton,⁴⁶ and Galliard,⁴⁷ in which the pneumococcus appears without doubt to have been the cause of the inflammation. The peritonitis in some of these cases was primary, and was the sole disease; in the other instances it was secondary to ordinary pneumonia. The pneumococcus would seem to be at one time noxious to the peritoneum, and at other times to be harmless. It is difficult to localise it in the peritoneum of animals by experiment. Cultures obtained from the exudation in certain instances of peritonitis in which the pneumococcus was the only micro-organism found and injected into animals caused death from general sepsis. Morisse,⁴⁸ whose monograph on this subject is by far the most complete, has collected eight examples of peritonitis in which, and in the associated lesions, the pneumococcus was the only micro-organism found. In seven of these there was either pneumonia or pleurisy. The exudation was purulent in five, and sero-fibrinous in three. Six of the patients died, and two recovered. In both of these laparotomy had been performed.

IV.—TUBERCULOUS PERITONITIS.

This variety of peritonitis is dealt with in Lecture IV.

V.—PERITONITIS OF A DOUBTFUL NATURE.

(a) *Peritonitis due to Irritants.*—On the subject of this form of peritonitis there is much conflict in opinion. If its existence be established, then the existence of a non-infective variety of peritonitis has to be at once acknowledged. Bumm⁴⁹ maintains that a non-infective peritonitis may be produced by mechanical, chemical, or thermal irritants, and states that it is usually plastic and localised. Pawlowsky injected sterilised croton oil into the abdomens of twelve animals. Of this number, eight died from hæmorrhagic peritonitis, and neither pus nor micro-organisms were found in the exudation, nor were there any signs of sepsis.

As a comment upon these experiments, it may be noted that pyogenic cocci (for example, the staphylococcus aureus) will act more readily, and in smaller doses if introduced into the peritoneum together with some corrosive fluid capable of injuring the serous endothelium, and further that such injury to the peritoneal surface—in cases in which no cocci are injected—encourages the escape of the colon bacillus from the intestine.

Fraenkel injected tincture of iodine and liquor ferri perchloridi into the peritoneum of certain animals. Peritonitis followed. If the exudation was examined at an early stage, or in the slightest cases, it was found to be clear and free from micro-organisms. If, however, the case were advanced, the exudation was purulent and swarming with bacteria which had passed through the bowel wall. A like explanation would appear to be adapted to an experiment by Thompson,⁵⁰ who found that a piece of wadding dipped in 1 in 1,000 solution of corrosive sublimate and placed within

the abdomen caused in some animals a fatal peritonitis. Jalaguier⁵¹ showed that tampons of sterilised gauze introduced into the peritoneal cavity of dogs caused no symptoms and no reaction, and when the animals were killed at the end of some months the gauze was found rolled up in a ball and encapsuled by adhesions. It was made evident, moreover, that it needed a foreign body of a certain size to even excite adhesions.

Fæces, if introduced into the peritoneal cavity, cause peritonitis, not by chemical action, nor as an irritant, but by reason of the micro-organisms or their products contained in the extravasation.⁵² If the material be sterilised no effect follows. Fresh filtered fluid from the bowel caused, when injected into the abdomen, a fatal peritonitis in the exudation of which the bacterium coli commune was found. The same fluid when filtered through many layers of gauze produced abdominal symptoms, but all the animals lived. When the fluid was sterilised it had no effect.

The same results have attended the injection of human bile into an animal's peritoneum. When the bile was untreated peritonitis usually followed, but when it was sterilised the injection was attended by no evil results. According to Naunyn⁵³, normal bile is sterile. According to Létienne⁵⁴, it may contain bacteria. It is said by others to be sterile so long as it contains free biliary acid. In inflammatory conditions of the bile passages the bacterium coli commune is commonly found. Lähr⁵⁵ has shown that in man a considerable effusion of bile may take place into the peritoneal cavity without causing peritonitis.

Blood is not of itself capable of producing peritonitis, although it affords a very favourable condition for the development of any bacteria which may gain access to the extravasation. This fact may be considered to be proved by the experiments of Penzoldt,⁵⁶ Cornil and Ranvier,⁵⁷ and others, and by the operation of peritoneal transfusion of blood performed on man by Ponfick,⁵⁸ Golgi and Raggi.⁵⁹ Orth⁶⁰ believes that it is possible to set up peritonitis by injecting a large quantity of blood into the cavity. He considers that it induces inflammation not so much through the action of bacteria as through the development of some fibrin ferment. Mr. Herbert Page⁶¹ has published three very interesting cases of peritonitis from hæmorrhage, but it is evident from the reports that the peritonitis was of a very slight degree and of very secondary importance.

A consideration of the whole matter leads to the conclusion that the production of peritonitis by non-infective irritants has not yet been satisfactorily proved.

(b) *Peritonitis Secondary to Rheumatism, Gonorrhœa, etc.*

Spillmann and Ganzinotty⁶² have collected fifteen recorded cases of rheumatic peritonitis, with nine deaths. In some of these cases the peritonitis appeared alone, in others in association with rheumatic troubles in the joints, or in other serous membranes. The evidence, however, upon which the pathological assumption depends is by no means satisfactory, and will not stand a critical examination.

Peritonitis has been met with in association with gonorrhœa, but inasmuch as the gonococcus will not survive in the peritoneal cavity, it is to be assumed that pyogenic cocci have found a nidus in the discharge from the genitals, and have spread by extension to the peritoneum. Interesting articles on this subject have been published by Horowitz,⁶³ Menge,⁶⁴ and Wertheim.⁶⁵

The peritonitis reputed to be due to Bright's disease, to alcoholism, and to syphilis, has not been shown to have a specific etiological existence. It would seem rather to occur under conditions which are very favourable to bacterial growth, or to be due to extension from parts which are already inflamed. The subject is fully dealt with in the monograph by Spillmann and Ganzinotty, to which reference has already been made.

(c) *Peritonitis of the Newborn.*

The peritonitis met with in the newly born appears to be due to one of two conditions. It may depend upon puerperal infection, and be brought about by direct extension from the cord, which is found to be inflamed, or gangrenous, or the seat of an actually erysipelatous condition, or it may be due primarily to the giving way of the bowel in cases of imperforate anus, or to that rupture of the normal sigmoid flexure

which has been assumed by Zillner⁶⁶ and Generisch⁶⁷ to be due to direct pressure on the bowel during parturition.

NOTE.—For some fuller account of non-infective and of chronic non-tuberculous peritonitis reference may be made to special articles by Schroeder (*Zeitsch. f. Geburt. und Gyn.*, 1889, p. 493) and Strümpell (*Gen. Path.*, Leipzig, 1885).

REFERENCES.

- ¹ *Dict. Encyclo. des Sc. Méd.*, Paris, 1887. ² *Über die Ätiologie der Peritonitis*, Basel, 1893. ³ *Wien. klin. Woch.*, Nos. 13 to 15, 1891. ⁴ *Loc. cit.* ⁵ *Fortschritte d. Med.*, 1885. ⁶ "Étude bactér. sur les Périton. par perforation," *La Cellule*, 1889, vol. 5. ⁷ *Lyon Médical*, 1889. ⁸ One of the most valuable monographs is *Le Bact. Coli Commune, son Rôle dans la Pathologie*, by Dr. Macaigne, Paris, 1892. Another good general account is by Malvoz (*Arch. de Méd. Expér.*, 1891, p. 503), and another by Welch (*Trans. Congress of American Phys. and Surg.*, vol. 2. ⁹ *Arch. de Méd. Expér. et d'Anatom. Pathol.*, January 1st, 1893. ¹⁰ *Compt. Rend. Soc. de Biol.*, 1891, p. 830. ¹¹ *Centralbl. f. allgem. Path.*, May 1st, 1892. ¹² *Ann. de l'Institut Pasteur*, July 25th, 1892. ¹³ *Arch. de Méd. Expér. et d'Anat. Path.*, January 1st, 1893. ¹⁴ Vignal, *Arch. de Phys. Norm. et Path.*, 1886-87. ¹⁵ See, however, on this point Gessner. "Über die Bakt. im Duod." *Arch. f. Hygiene*, Bd. ix., p. 128. ¹⁶ *Manual of Bacteriology*, New York, 1892. Booker isolated twenty-one different kinds of bacteria from the contents of the small bowel in cases of diarrhoea. ¹⁷ *Über die Ätiologie der Peritonitis*, Basel, 1893. ¹⁸ Le Sage and Macaigne, *Arch. de Méd. Expér.*, 1892, vol. iv., p. 350. ¹⁹ Fraenkel, *loc. cit.*; Macaigne, *loc. cit.* ²⁰ Paul Ziegler, *Über die intest. Form. der Peritonitis*, Munich, 1893. ²¹ Arndt, *Centralbl. f. Bakt.*, 1893, xiii., p. 173. ²² Ziegler, *loc. cit.* ²³ Laruelle, *La Cellule*, 1889, vol. 5. ²⁴ *Virch. Arch.*, vol. 120, p. 7. ²⁵ *Congrès de Chirurgie*, 1889. ²⁶ *La Cellule*, 1889. ²⁷ *Centralbl. f. Bakt.*, 1893, xiii., p. 173. ²⁸ *Loc. cit.* ²⁹ *Arch. de Méd. Expér.*, t. i., p. 353. ³⁰ *Centralbl. f. Chirurg.*, 1889, p. 849. ³¹ *Centralbl. f. Path. Path. Anat.*, October 12th, 1893. ³² *Compt. Rend. de la Soc. de Biologie*, 1891, iii., p. 740. See also cases of Roswell Park, *Annals of Surgery*, September, 1893, p. 310. ³³ *Boll. della R. Acad. med. di Roma*, 1891, f. iv. and v. ³⁴ *Ibid.*, f. vii. ³⁵ *Arch. de Méd. Expér.*, 1892, vol. 4, p. 350. ³⁶ *Annals of Surgery*, September, 1893, p. 311. ³⁷ *Trans. Congress of American Phys. and Surgeons*, vol. 2. ³⁸ *Deutsche. Zeit. f. Chir.*, 1891. ³⁹ *Gaz. des Hôpitaux*, November, 1890. *Münch. med. Woch.*, 1889, No. 42. ⁴⁰ *Centralbl. f. klin. Chir.*, 1888. ⁴¹ See case by Cheurlin, *Thèse de Paris*, 1879. ⁴² *Soc. Méd. des Hôp.*, May 22nd, 1890. ⁴³ *Centralbl. f. Bakt.*, January, 1889. ⁴⁴ *Soc. Méd. des Hôp.*, April, 1890. ⁴⁵ *Ibid.*, May, 1890. ⁴⁶ *Bull. Méd.*, October, 1890. ⁴⁷ *Soc. Méd. des Hôp.*, November, 1890. ⁴⁸ "Péritonite à Pneumococcus," Paris, 1892. See also an excellent article by Boulay, "Des Affections à Pneumococcus indépendantes de la Pneum.," Paris, 1891. ⁴⁹ *Munch. med. Woch.*, No. 42, 1889. ⁵⁰ *Centralbl. f. Gyn.*, January 31st, 1891. ⁵¹ *Gaz. Hebdom.*, 1892, p. 161. See also a case by Michaux, *Bull. et Mém. Soc. de Chir.*, April, 1893. ⁵² Ziegler, Pawlowsky, Reichel, and others. ⁵³ *Deuts. med. Woch.*, 1891, No. 5. ⁵⁴ *Arch. de Méd. Expér.*, t. iii., p. 761. ⁵⁵ *Schmidt's Jahrb.*, 1890, t. iii., p. 235. ⁵⁶ *Deuts. Arch. f. klin. Med.*, 1876. ⁵⁷ *Soc. de Biol.*, 1871. ⁵⁸ *Berl. klin. Woch.*, 1879. ⁵⁹ *Gaz. des Hôp.*, 1880. ⁶⁰ *Centralbl. f. Chir.*, 1889, p. 849. ⁶¹ *Trans. Clin. Soc. Lond.*, vol. xxv., 1892, p. 172. ⁶² *Dict. Encyclop. des Sc. Méd.*, Article, Peritonitis, Paris, 1887. ⁶³ *Wien. med. Woch.*, 1892, No. 2. ⁶⁴ *Zeits. f. Geburt. und Gynäkol.*, 1891, xxi., p. 19. ⁶⁵ *Centralbl. f. Gynäkol.*, 1892, No. 20. ⁶⁶ *Virch. Arch.*, Bd. 96, p. 307. ⁶⁷ *Ibid.*, Bd. 126, p. 485.

ON SOME CASES OF ACUTE INTUSSUSCEPTION IN CHILDREN.

By ARTHUR E. J. BARKER, F.R.C.S.,

Professor of the Principles and Practice of Surgery and Professor of Clinical Surgery at University College, London; and Surgeon to University College Hospital.

I AM desirous of placing on record four cases of acute intussusception in children which, taken with three others I have already published, constitute a complete list of all the cases of this condition which it has fallen to my lot to treat in patients of tender age. I may add that in adults I have besides twice bodily excised intussusceptions of the sigmoid flexure of the colon with complete success. But the total number of young patients coming under my personal care has been seven.

The first was a female infant, aged 4 months, whom I treated by inversion and injection of air and water twenty-eight hours after the onset of the condition, and with apparent reduction. The tumour however had returned nine hours later, and fourteen hours after the first injection was again treated as before with the same apparently good result. Some two hours later however the tumour was again found, and was again to all appearance reduced as before. Soon after this the child became exhausted and died; when it was found that the bowel had never been completely reduced, and was gangrenous at the point strangulated. There was no rupture of the gut, and reduction of all but the ileo-cæcal portion had been brought about each time the injection had been tried.

The second case has already been fully recorded,¹ and I will only say of it that the patient was a boy of 4 years old, whom I saw eleven hours after the onset, when I at once tried injection with water, and apparently with success as far as disappearance of the tumour was concerned. But five hours later the condition was as bad as before, and I at once performed median laparotomy, reduced the intussusception with some difficulty but with perfect success, the child recovering without a bad symptom.

The third case was seen in private, and is as follows:

February 14th, 1889. Sent for to see baby V., aged 16 months, at midnight,

and found a very healthy child, apparently not suffering very much. An intussusception had been diagnosed thirty-six hours before. This had come on immediately after an elder child had fallen in a sitting position once or twice upon the patient's abdomen while it lay in its perambulator with the strap and buckle crossing the latter. Dr. Gee had seen the child twenty-four hours after the onset of symptoms, and had employed inflation apparently with the best immediate results as regards disappearance of the tumour. The baby had, however, been evidently ill since, and the temperature had reached 103°, and the pulse was 140 to 160. Under chloroform I could detect no distinct tumour, but there was an increased sense of resistance under the right border of the liver. There had been no vomiting, and as both the medical men in attendance thought the child was somewhat better by the time I saw it, we agreed to await further symptoms. The next morning (February 15th) I was sent for early, as the child was manifestly worse. It had vomited twice since midnight and the abdomen was more distended. There was, however, no straining and no passage of blood or mucus. Pulse 140.

Laparotomy, February 15th, at 2.15 P.M., fifty hours after onset. Spray, etc. The tumour was easily found bound down in the right flank at the spot where resistance had been felt before. It was 4 to 5 inches long and consisted of the last 8 inches of the ileum, prolapsed and strangulated through the ileo-cæcal valve. With some trouble it was brought out of the wound, but was then easily reduced by gathering in the intussusciptions from below with the fingers, and thus squeezing on the intussusception upwards. As it was slipping back through the ileo-cæcal valve and the intussusciptions was unfolding, it was seen that there was adhesive peritonitis glueing the surfaces together over an area equal to a six-penny piece, and here the peritoneum peeled off to that extent. At this spot there was much swelling and discoloration, but the bowel looked as if it would recover. There were two or three bubbles of gas under the peritoneum at the mesenteric border of the bowel at the point of greatest constriction, and here there was some thrombosis of the small veins of the mesentery with white lines on either side of them. There was some clear fluid in the abdomen, which was sponged out before closing.

The abdomen was closed within half an hour of the commencement of the operation, but had been only open a quarter of an hour. There was no shock, and the pulse at the end had fallen to 120. The patient was fed by the rectum, only a little brandy and opium to be given by the mouth. At 9 P.M. he looked fairly well. Pulse 160.

The next day, February 16th, the child seemed better in every way. The dressing was wet with urine, and was changed. At 1.30 P.M. there was a sudden cry of pain, and the child drew up his right leg and became restless. He then gradually failed, and died at 3.10 P.M.

Necropsy forty hours after death. No general peritonitis. The intussuscepted part of the ileum was still dark, but sound except at the point of greatest nipping. Here a slough was to be seen at the mesenteric aspect of the bowel, and in the centre of this a perforation, with escape of faeces, causing local peritonitis. The slough was seen on both sides of the mesentery, and appeared to correspond to an area supplied by one vessel. It was wedge-shaped, with the apex towards the mesentery, and was whitish-grey. It appeared to correspond to the thrombosed veins seen during the operation at the most constricted point. The spot mentioned above as having been stripped of its peritoneal covering was quite sound and clean. The specimen was removed *in toto*.

The fourth case was that of a male child of 5 months, whom I saw two days after the onset of the intussusception, and immediately used injection, apparently with success. But five hours and a-half later matters were as bad as before, and I performed laparotomy and reduced the strangulation. The child made an excellent recovery. In this case I reduced the intussusception with my finger in the abdomen before I could draw the part involved into view.

The fifth case has also been recorded fully.² The patient was a boy, aged 4 months, whom I did not see until the fourth day after onset of the intussusception. He was then much collapsed, and, as injection had been tried already in the country, I proceeded at once to laparotomy. On opening the belly the intussusciptions was already sloughing, and had two large perforations in it. Enterectomy was at once done, but the child died ten hours later of exhaustion.

In the sixth case, a boy aged 12, laparotomy was also done for the enteric variety of intussusception forty-eight hours after the onset. Here the jejunal strangulation could not possibly be reduced, and enterectomy was at once done. The boy died twelve hours later of collapse.³

The seventh case is the last of my own series, excluding adults. R. D., aged 7 months, a healthy child until her present illness, was admitted into University College Hospital on February 7th, 1893. She had hitherto been fed by the breast, and her motions had been natural. On February 6th, at 9 A.M., the bowels were opened for the last time before admission. At 1 P.M. the same day the child was sick, and had been so frequently until admission. During the night blood and mucus were passed by the bowel, and several times since. On admission in the evening she was pale, restless, and fretful, but showed no collapse. The abdomen was not distended, and was fairly soft, but on palpation there was rigidity of the muscles. A rounded swelling could, however, be felt in the right flank, reaching from under the rectus muscle to the back of the abdominal cavity on the same side; it was movable, but not freely. Nothing abnormal could be felt from the rectum, but the finger came away stained with blood, mucus, and faeces. At 9.45 I made an examination under chloroform, and found a curved reniform tumour reaching from just external to the right linea semilunaris on a level with the umbilicus to just beyond the middle line, midway between the latter and the base of the xyphoid cartilage. The whole mass was now freely movable; it was blunt below and tailed off above. By manipulation the mass was reduced by about one-half, and what remained lay in the right iliac region, and was easily grasped between finger and thumb. Manipulation gave rise to much gurgling and an evacuation of blood-stained mucus. Ten ounces of warm water were now run into the bowel with 3 ft. pressure. This appeared to fill the colon right up to the iliac fossa, but, on its being allowed to run off, the tumour was found unaltered.

Operation.—I then performed laparotomy in the usual way by a 2 inch

¹ *Vite Lancet*, vol. ii, 1888, pp. 200 and 102.

² *Vite Lancet*, January 9th, 1892.

³ *Vite Lancet*, January 9th, 1892.