rare this is in women in middle life), atrophy occurs, and the individual gradually declines.

Apart from the physiological investigations above mentioned, observations are being carried out, at my suggestion, by the above method in the following directions:

The relation of the calcium salts to infective processes, acute and chronic, especially with regard to the temperatures, to the opsonic power of the blood and to the fibrosis which results from chronic toxæmias. This investigation will undoubtedly prove the value of calcium salts in certain types of pneumonia and similar affections.

The influence of menstruation on the opsonic power of the blood from this standpoint.

An estimation of the lime salts in the blood in bone diseases, such as rickets and osteomalacia.

The relation of the calcium salts to the thyroid secretion and their bearing on thyroid disease. Owing to the frequent occurrence of this disease in women and the relation often seen to the genital functions important results should be obtained.

The significance of the calcium salts in neuroses and in such diseases as spasmodic asthma and urticaria.

I am myself making a series of observations on the disorders of menstruation, on the neuroses of women, and on the diseases of pregnancy, such as vomiting, chorea, eclampsia, and thrombosis, many of which conditions seem undoubtedly to have a definite relationship to a disordered calcium economy on the part of the patient.

Also, in conjunction with Professor J. E. S. Moore, I am carrying out a clinical and experimental investigation into the influence the calcium salts may have in the onset of cancer and the cancer period.

I have much hesitation in putting forward this formidable list, on which some work has been done already, lest I should appear to overstate the importance of the subject. I should not now have had the courage to do so had I not had, and did I not still hope to have, the generous advice, co-operation, and assistance of my colleagues and others interested in these researches. In this connexion I should like to express my obligations to Sir James Barr, Professor Benjamin Moore, Dr. Grimsdale, and Dr. Pantland Hick.

REFERENCES.

¹ Liverpool Medical Institution, May 3rd, 1906, vide *Liverpool Medical-Chirurgical Journal*, July, 1906. ² BRITISH MEDICAL JOURNAL, March 23rd, 1907, p. 717: On the Use of Calcium Salts in Pneumonia.

FURTHER OBSERVATIONS ON THE ETIOLOGY AND PATHOLOGY OF PEPTIC ULCER.*

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INTRODUCTION.

THE present communication on the etiology of peptic ulcer presents two phases, developed by the author's experimental researches on animals. By feeding animals, principally dogs, with a certain strain of bouillon cultures of *Bacillus coli communis* for several months, spontaneous or induced, genuine peptic ulcers of the stomach or duodenum were found in every animal experimented upon, the ulcers causing death, either by perforation or hæmorrhage. Certain changes in the blood serum and cellular changes found indicated a general process characterised by diminution of the normal protective bodies, resulting in autoctolysis. The blood changes and general condition seemed to be produced by the absorption of products formed in the gastro-intestinal tract. Histological examinations of the induced peptic ulcers showed no round-cell infiltration or other evidences of a healing process.[†]

In the first series death occurred from perforation or hæmorrhage; in the second series the feeding of bouillon

of *B. coli* was stopped as soon as there appeared indications of the formation of the ulcer. Then allowing sufficient time to elapse for healing to take place, the animal was killed. On *post-mortem* examination we found round-cell infiltration and cicatrization of the incomplete ulcers, in various stages of healing, thus further showing the close relation between the feeding experiments with bouillon cultures of *B. coli communis* and spontaneous or induced peptic ulcers in animals.

Controls were made by observation on 271 dogs, 189 of which were healthy and were killed at the dog-pound, and the other 82 dogs that had died from various diseases—injury, poison, etc. On *post-mortem* examination no peptic ulcers were found in the stomach or intestines of any of these 271 dogs, showing that the occurrence of peptic ulcers both in the healthy street dogs and diseased animals is at least exceedingly rare.

HISTORICAL.

The peculiar type of the round peptic ulcer confined principally to the stomach and duodenum early led to the conclusion that the ulcers were the result of autodigestion of a portion of the mucous membrane rendered susceptible by some unknown cause. That the living healthy mucous membrane could not be digested was explained by Stahl as due to the protection offered by the "sensitive soul." Hunter's living principle displaced the idea of the "sensitive soul," and with the development of cellular pathology we acquired the idea of the vital resisting power of the tissues, which is yet used in pathological expressions. The vital resistance of the tissues is represented in more modern terms by Weinland, who holds that an antipepsin ferment residing in, and a part of, the gland cells protects them from destruction. It is easy to understand why so much experimental work has been done to produce a lessening of the resistance of the tissue by diminishing or destroying the vitality of local areas of the mucous membrane of the stomach. These experimental investigations, clinical observations, and *post-mortem* examinations may be grouped as follows:

(1) Attempts to produce ulcers by mechanical and physical injury, such as those by Ritter, Decker, Matthes, Schmidt, Korte, and others; (2) by chemical injury, such as employed by Roth, Matthes, Schmidt, and Ewald; (3) by inducing blood changes, thus lessening resistance, such as hæmoglobinaemia, by the methods employed by Silberman, Cohnheim, and Fütterer; (4) disturbances of the local circulation to conform to Virchow's idea of embolism and thrombi were worked out by Panum, Klebs, and Welti, Talma, Rindfleisch, and Müller; (5) injuries to nerves and nerve centres, such as accomplished by Schiff, Ebstein, Brown-Séquard, Vedora, Yzeren, Saitta, Ophüls, Roch, and Ewald; (6) local infection was studied by Cohnheim, Bottcher, and Nauwerck.

Ulcers were produced in many cases; but these artificial methods were either no other than would cause ulcer formation in any part of the body, or they were foreign to the conditions present in ulcer of the stomach in man.

EXPERIMENTS.

With some modifications I have carried on similar experiments in attempting to produce artificial ulcers, following out at first the methods of mechanical and chemical irritation, later taking up injections of toxins, then the question of metabolic disturbances, and, finally, the problem of the genuine induced peptic ulcer. Mechanical and chemical irritation failed to produce ulcers, even with such radical means as the feeding on mustard oil for fourteen months. The possibility of systemic disturbances, altered metabolism, impaired nutrition, being etiologic factors in the production of ulcer led to the experiment of confining animals in very close quarters for a long period of time. Of the 96 guinea-pigs and 36 rabbits used in this experiment, all the rabbits died, while 6 guinea-pigs survived nine months of confinement. Two genuine peptic ulcers were found in the 6 surviving guinea-pigs.

These induced ulcers, produced for the first time in the long series of experiments, suggested the possibility that systemic conditions were important factors; that alteration in the toxic state of the alimentary canal, with consequent changes in the blood, might play some rôle in the formation of the ulcer. To modify, then, the bacterial status of the alimentary tract, experiments were begun by

* Presented before the Section of Pathology and Bacteriology, British Medical Association, Toronto, August, 1906.

† The full report, with protocols, physiological examinations and micro-photographs, was presented at the International Medical Congress, Lisbon, 1906, and published in the *Journ. Amer. Med. Assoc.*, June 9th, 1906, vol. xlv, pp. 1753-1763.

introducing cultures of *Bacillus coli communis* into the circulation and into the stomach. The toxin of *Bacillus coli* being intracellular suggested the experiments of using killed as well as living cultures.

Intravenous inoculations of dogs and rabbits with cultures of *Bacillus coli* for a long period of time (one to six months) failed to produce ulcer of the stomach. But it was found that artificial ulcers made by removing portions of the mucous membrane of the stomach did not heal in 4 cases out of the 20 animals receiving the above inoculations. Genuine induced peptic ulcers were produced, however, in every experiment in which cultures of *Bacillus coli* were fed to dogs. Bouillon cultures of this organism were given daily in increasing amounts, from 50 c.cm. to 2,000 c.cm., for two to four months, in connexion with an ordinary meat diet, and in other cases with meat extractives in addition to the bouillon cultures.* During the course of the experiments observations were made of the blood changes such as the agglutinating, haemolytic, and coagulating power, the bacteria in the blood and stomach, the systemic disturbances, the evidences of infection, the symptoms of ulcer, as pain, haemorrhage, etc. It was found that the dog's serum agglutinated *Bacillus coli* in high dilution, that the coagulating time of the blood was slower, that haemolysis was present, that bacteria were at no time found in the blood, and that few, if any, symptoms of systemic disturbance appeared. In one case, where death resulted from haemorrhage from a pyloric ulcer, symptoms of internal haemorrhage developed a few days before death.

Post-mortem examination revealed ulcers in either the stomach or duodenum, the number varying, however, from a few in the duodenum to numerous typical peptic ulcers in the stomach. Histological examinations were made of the ulcers in various stages of their development, and of the liver and kidneys. The chief points of interest noted in this study were: (1) Catarrhal conditions of the mucosa; (2) local necrosis of the cells; (3) breaking down of the glandular structures; (4) disappearance of the chief cells; (5) marked hyperplasia of the parietal cells, which were large and prominent; (6) vacuolization of the parietal and chief cells; (7) complete degeneration of the cells; (8) no inflammatory reaction, such as round cell infiltration, either surrounding, or at the site of, the ulcer.†

The factors concerned in the production and persistence of ulcer of the stomach and duodenum appear from the experiments to indicate a dual condition: There seems to be some toxic condition produced which overcomes natural resistance, resulting in *cytolysis*, and possibly some chemical substance formed within the alimentary tract which, when absorbed, may neutralize the protective bodies in the blood and tissues, resulting in *autocytolysis*.

These facts, obtained through several years of experiments, warrant the conclusions that (1) ulcer of the stomach and duodenum can be produced in dogs by feeding *Bacilli coli communis* for a variable length of time; and that (2) we have now for the first time a firm basis by which to solve the problem of the finer or underlying etiology of ulcer.

As observed in our mention of the first series of the feeding experiments with bouillon cultures of *B. coli communis* (which were continued over a considerable period of time), there was in the ulcers that were formed no round-cell infiltration or connective-tissue formation or other evidences of a healing process. As the feeding experiments continued, the ulcers that formed, resulting in the death of the animal either from perforation or haemorrhage, indicated a progressive destructive process confined to the ulcerated areas in the stomach or duodenum.

In the second phase or series of the experiments it was necessary to determine the result of the stopping of feeding experiments with the bouillon cultures of *B. coli communis*. After one or two months had elapsed from the beginning of the experiments and the changes in the blood and other distinct evidences of systemic disturbances had been found, the feeding of the bouillon cultures of *B. coli* was stopped, and the animals kept under good general hygienic conditions on ordinary food for one month. The dogs were then killed. Ulcers were found in the stomachs of the dogs in various stages of the healing

process. Fig. No 1 is an example of a typical deep ulcer found in the stomach of a dog, which was allowed to undergo the process of round-cell infiltration and cicatrization, showing that the factor that prevents healing, as well as that which induces the formation of ulcer, had been removed.

During the course of the experiment of feeding dogs with cultures of the *B. coli communis*, examinations of the blood changes were made about every two weeks, for example, blood count, haemoglobin curve, haemolysis, agglutination, alkalinity, bacteriolysis and phagocytosis. The control blood examinations were also made as often as possible. It will be of interest to make a partial further report of these findings.

1. Blood Count and Haemoglobin Curve.

The percentage of haemoglobin did not change materially during the feeding experiments of most of the dogs. In the case of two, however, the percentage dropped to 70.

Similarly the number of red blood corpuscles remained about the same. The number of leucocytes, while found to be high (about 12,000 to 15,000) normally in dogs not experimented with, nevertheless increased in the experimental dogs, in several cases to about 25,000.

2. Haemolysis.

An increase in the haemolytic power of the experimental dog's serum was noted during the course of the experi-

Fig. 1.—Deep cicatrized peptic ulcer at pyloric end of the stomach (dog) found after stopping the feeding experiment.

ment, but great difficulty was experienced in testing the haemolysis, because dog's corpuscles are so readily broken down.

3. Agglutination.

In every case agglutinins were found to increase in amount during the experiment, so that at the end of about four weeks' feeding marked agglutination of *B. coli* took place in four hours with a serum dilution of 1 in 500.

It was not found possible to increase the agglutinins to so high a degree or so quickly by feeding dogs with cultures of *B. coli* as in the case of intravenous inoculations of the bacteria.

4. Alkalinity.

Titration of the serum of the experimental dogs with fortieth normal oxalic acid showed very little change in the reaction of the serum. However, while the alkalinity came within the limits of normal dog's serum—for example, 0.001 to 0.008—in most of the dogs it remained low—for example, nearer 0.001 than 0.008.

5. Bacteriolysis.

The bacteriolytic power of the serum was tested by comparing the number of bacteria in a bacterial emulsion

* Protocols of these experiments. See loc. cit.

† Macrophotographs and microphotographs of ulcers. See loc. cit.

of *B. coli* before and four and eighteen hours after adding dog's serum.

This bacteriolytic power became materially less during the course of the experiments, and was markedly less than that of serum of normal dogs.

6. Phagocytosis.

The extent of the phagocytosis was measured by counting the number of bacteria ingested by the leucocytes after one hour's incubation of a mixture of leucocytes, serum, and a bacterial emulsion.

The phagocytic index—for example, the average number of bacteria taken up by the leucocytes—was less in the case of the experimental dogs than in the case of dogs not experimented with.

The following are abstracts from the tables of the blood examinations:

Agglutination.

Method: 1. Bouillon suspension of twenty-four hours' growth of *B. coli communis* on slant agar.

2. Made dilutions—namely:

Control: 1 part bouillon × 4 parts emulsion.

Dilution 1 in 50: 1 part serum (diluted 1 in 10) × 4 parts emulsion.

„ 1 in 100: 0.5 part serum (diluted 1 in 10) × 4.5 parts emulsion.

„ 1 in 500: 1 part serum (diluted 1 in 100) × 4 parts emulsion.

„ 1 in 1,000: 0.5 part serum (diluted 1 in 100) × 4.5 parts emulsion.

3. Incubated twenty-four hours. Recorded degree of agglutination after four hours, eighteen hours, and twenty-four hours.

	Name and Date.	Dilution 1 in 50.		Dilution 1 in 100.		Dilution 1 in 500.	
		4 hrs.	18 hrs.	4 hrs.	18 hrs.		
Normal dogs	Woolly	0	0	0	0		
	Whitty:						
	April 2:	0	0	0	0		
	Trixie:	0	0	0	0		
	April 21:	0	0	0	0		
	Kate:	0	0	0	0		
May 8th, began feeding living bouillon cultures <i>B. coli communis</i> ...	April 2:	0	0	0	0		
	June 2:	xx	xx	x	x		
	June 12:	x	xxx	0	xx		
	June 21:	xx	xx	xx	xx	x	x
	June 21:	xx	xx	xx	xx		
	June 21:	xx	xx	xx	xx		
May 8th, ditto	Kate:	0	0	0	0		
	April 2:	0	0	0	0		
	May 22:	0	x	0	x		
	June 12:	x	xxx	x	xxx		
	June 21:	xx	xx	x	x		
	July 10:	xx	xx	x	xx	0	x
May 8th, ditto	Pete:	0	0	0	0		
	May 22:	0	0	0	0		
	June 1:	x	xxx	x	xx		
	June 21:	xx	xx	x	xx		
	July 10:	xx	xxx	x	xx	0	0
	July 10:	xx	xxx	x	xx	0	0
May 14th, began feeding killed cultures <i>B. coli communis</i> ...	Gyp:	xx	xx	x	x	0	0
	June 21:	xx	xx	x	x	0	0
	June 21:	xx	xx	x	x	0	0
	June 21:	xx	xx	x	x	0	0
	June 21:	xx	xx	x	x	0	0
	June 21:	xx	xx	x	x	0	0

Bacteriolysis.

Method: 1. Used sterile blood. Defibrinate centrifuge. Pipette off the serum.

2. Made a bouillon emulsion of twenty-four hours' growth of *B. coli communis* on slant agar. (Made slightly cloudy emulsion.)

3. Made known dilution of serum with bouillon. Added 1 loop of emulsion to 1 c.m. bouillon, then added 1 loop of this to each tube and to control.

Control: 4 parts bouillon × 1 loop emulsion (diluted).

(a) 2 parts serum × 2 parts bouillon × 1 loop emulsion (diluted).

(b) 1 part serum × 3 parts bouillon × 1 loop emulsion (diluted).

4. Plate 1 loop of control at once.

5. Incubated tubes eighteen hours. Plate 1 loop from each tube at the end of four hours and eighteen hours.

6. Counted colonies on plates.

Name and Date.	CONTROL.			Mixture.	NORMAL DOGS.	
	No. of Bacteria at once.	No. of Bacteria after 4 hours.	No. of Bacteria after 18 hours.		No. of Bacteria after 4 hours.	No. of Bacteria after 18 hours.
JIMKEY, June 25	560	3,796	Many	A	0	Many
" "	—	—	—	B	34	Many
TRIXIE, May 4..	—	28	1,248	A	5	160
" "	—	—	—	B	124	638
KATE, May 14..	—	28	1,248	A	2	1,341
" "	—	—	—	B	10	656
JIMKEY, July 10	15	122	Many	A	0	604
" "	—	—	—	B	2	Many
May 4 ...	—	28	1,248	A	5	160
" ...	—	—	—	B	124	638
May 22 ...	—	1,072	680	A	42	—
" ...	—	—	—	B	132	210
June 21 ...	361	19,800	Many	A	4,016	Many
May 4 ...	—	28	1,248	A	2	1,341
" ...	—	—	—	B	10	656
May 22 ...	—	1,072	680	A	6	—
" ...	—	—	—	B	70	120
June 12 ...	196	982	Many	A	410	Many
" ...	—	—	—	B	—	—
June 21 ...	361	19,800	Many	A	7,260	Many
" ...	—	—	—	B	Many	Many
July 10 ...	15	122	Many	A	2	57,600
" ...	—	—	—	B	8	Many
May 22 ...	—	1,072	680	A	32	—
" ...	—	—	—	B	41	374
June 12 ...	196	982	Many	A	105	Many
" ...	—	—	—	B	—	—
June 21 ...	361	19,800	Many	A	1,870	—
" ...	—	—	—	B	Many	—
July 10 ...	15	122	Many	A	6	16,256
" ...	—	—	—	B	9	Many
June 1 ...	—	968	Many	A	2,688	—
" ...	—	—	—	B	360	—
June 21 ...	361	19,800	Many	A	251	—
" ...	—	—	—	B	Many	—
June 1 ...	—	968	Many	A	379	Many
" ...	—	—	—	B	680	1,160
June 21 ...	361	19,800	Many	A	1,188	Many
" ...	—	—	—	B	24,961	Many
June 29 ...	560	3,796	Many	A	57	Many
" ...	—	—	—	B	1,220	Many

Alkalinity.

Method: 1. One part serum plus one part aqueous solution of rosolic acid.

2. Titrate with N/40 oxalic acid until the red colour had given place to a pure yellow.

3. Calculate alkalinity in terms of N/NaOH.

Alkalinity beef serum (normal) — 0.008
" dog " " — 0.008 to 0.001

	Living Cultures.			Killed Cultures.		Normal Dogs.
	May 8.	Sept.	May 18	May 16	May 14	
May 4 ...	0.0012	—	0.0014	—	—	Trixie, 0.0012
" 22 ...	0.001	0.0014	0.0014	0.002	—	Kate, 0.0012
June 1 ...	—	—	—	0.0047	0.0025	—
" 12 ...	0.0024	0.0016	0.0014	—	—	—
" 21 ...	0.0025	—	0.0025	0.0017	0.0025	—
" 29 ...	—	—	—	0.001	—	Jimkey, 0.0075
July 10 ...	—	0.0012	0.0012	—	—	" 0.0015

Phagocytosis.

Method: 1. One part leucocytes (removed from surface centrifuged blood), one part serum, one part bacterial emulsion.

2. Incubated one hour.

3. Made smears and stain (Wright's stain).

4. Count the number of bacteria in leucocytes.

NORMAL DOGS.

Name and Date.	Polymorphonuclear Neutrophils.		Small Lymphocytes.		Mononuclear Leucocytes.		Eosinophiles.		Average in P.M.L.
	Number of Leucocytes Counted.	Number of Bacteria.	Number of Leucocytes Counted.	Number of Bacteria.	Number of Leucocytes Counted.	Number of Bacteria.	Number of Leucocytes Counted.	Number of Bacteria.	
JAMKEY—									
June 29 ..	25	124	7	35	0	0	12	2	5.0
July 10 ..	14	203	10	0	2	0	0	0	14.0
* TRIXIE—									
May 22 ..	9	22	3	0	1	0	6	3	2.4
June 12 ..	15	50	0	0	1	0	2	2	3.1
" " ..	10	0	1	0	2	0	3	0	
" " ..	18	135	7	0	1	0	4	30	7.5
† KATE—									
May 22 ..	9	32	1	0	0	0	0	0	3.5
June 21 ..	9	50	4	0	1	0	2	0	5.5
July 10 ..	15	120	10	11	2	4	0	0	8.0
‡ PETE—									
May 22 ..	8	19	4	0	6	0	0	0	2.4
June 21 ..	9	69	19	0	2	2	6	7	7.6
July 10 ..	10	80	3	0	5	0	6	18	8.0
§ GYP—									
June 1 ..	8	55	13	39	1	0	2	5	6.8
" 21 ..	16	36	4	6	3	0	2	0	2.2
¶ NIG—									
May 22 ..	10	0	2	10	3	24	8	57	
June 1 ..	19	165	23	21	5	0	3	0	
" 21 ..	8	16	5	0	3	0	0	0	
" 29 ..	6	62	15	0	5	0	0	0	

* May 8th, began feeding living cultures *B. coli*.

† May 8th, began feeding living cultures *B. coli*.

‡ September, began feeding living cultures *B. coli*.

§ May 14th, began feeding killed cultures *B. coli*.

¶ May 16th, began feeding killed cultures *B. coli*.

HISTOLOGICAL CHANGES.

The first change in the healing ulcer that appears microscopically is the presence of a round-cell infiltration

Fig. 2.—Ulcer partly developed when feeding experiment was stopped (dog). Beginning round-cell infiltration.

which begins at the upper lateral walls of the ulcer, near the surface of the healthy mucosa. These cells infiltrate the entire mucosa and reach downwards towards the base of the ulcer, which is shown in Fig. 2.

Another stage in the healing process shows the formation of bands of connective tissue which appear at the upper part of the wall of the ulcer, and dip downward

and inward (as shown in Fig. 3). The contraction of these bands draws up the base of the ulcer, and subsequently bands of connective tissue form the connecting strand with the opposite side of the ulcer wall. The high power shows the character of these strands (Fig. 4). Gradually the area of the ulcer is filled in by the walls of the ulcer drawing closer together.

Fig. 3.—Connecting bands of tissue "filling in," and by contraction drawing the walls of the lesion together.

Finally the area of connective tissue is reduced to a small strip forming a very narrow cicatrix, which is well shown in Fig. 5.

SUMMARY.

The conditions which normally should be active in stimulating round-cell formation and healing process are suspended in the first series of experiments; while in the second series—after a pause in the feeding experiments—the round-cell infiltration and healing by connective tissue replacement are re-established. These experiments lead

Fig. 4.—Same as Fig. 3, higher power (note elongation of nuclei).

us to the conclusion that the problems involved in the two series are fundamental and far-reaching, and enter into the question of the etiology not only of peptic ulcer but of many diseases.

The conditions produced in the first series, that permit ulcers to form in stomach or duodenum without a definite attempt at healing, seem to correspond to the changes

found in the blood; these in turn are produced by some bodies formed in the gastro intestinal tract.

The altered growth of the flora of the stomach and intestine in certain nutrient media, as produced in the

Fig. 5.—Completely healed ulcer (stomach of dog).

feeding experiments, indicates the important factors in the formation of ulcer of the stomach or duodenum, and the relation to other pathological conditions frequently associated with the lesions.

A CASE OF REPEATED ABDOMINAL SECTION FOR PERFORATION OF GASTRIC ULCERS.

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THE paper by Mr. Archibald Cuff, of Sheffield, in the BRITISH MEDICAL JOURNAL of February 2nd, much interested me on account of its close similarity to the following case of my own:

History.

A. H., aged 33, suffered from pain after food about the age of 18, and the first definite fact beyond this which I can ascertain is that he was admitted into the General Hospital, Nottingham, with an attack of haemorrhage from the stomach about fifteen years ago. He remained in the hospital one month. He suffered, more or less, with pain after food ever since, until on December 31st, 1905, at about 5 a.m., he was seized with intense abdominal pain. His supper overnight had consisted only of a little warm milk. I saw him with Dr. Fulton at about 1 p.m. the same day, and he was removed to the hospital.

First Operation.

At the operation (about 3 p.m.) I found a large ulcer in the cardiac end of the stomach, nearer the greater curvature, with a perforation in it about the size of a threepenny-piece. This was closed by a double row of continuous silk sutures, which invaginated the ulcer completely. The peritoneum was wiped out, and his recovery was uneventful. He left the hospital well, except for some oedema of his lower extremities.

Second Operation.

On October 2nd, 1906, at about 6 p.m., I was again asked by Dr. Fulton to see this man, and agreed with him that he had a perforated ulcer of the stomach. He was again admitted to the General Hospital, and I reopened the abdomen about 9 p.m. An entirely different ulcer was found this time, at the pylorus, with a small perforation at its base. This was closed in the same way as the former, and the peritoneum wiped out dry and the abdomen closed. At both this and the former operation the abdomen was completely closed.

At this second operation I carefully inspected the stomach, especially that part which was the site of the previous ulcer. No trace of it remained, and not an adhesion was left to indicate the catastrophe which had taken place in 1905. This removal of all traces of former pathological changes inside the peritoneum is remarkable, and

has been commented on frequently. In cases of suppurative appendicitis, when at the operation the appendix is not discovered, it is my habit to advise the subsequent removal of the organ. I therefore have on many occasions opened the abdomen of patients who have been the subjects of intense inflammation in this neighbourhood, and the frequency with which one discovers an apparently perfectly undamaged peritoneum is most remarkable.

As regards the patient about whom I am now writing, I am informed that he is perfectly well except for some slight oedema of his left leg. He is able to eat any article of food without pain, so I expect he will not take my advice and have a posterior gastro-jejunostomy performed on him.

Success in dealing with perforated gastric ulcers depends almost entirely upon the period which elapses between the catastrophe occurring and the operation, and to Dr. Fulton is due the recovery of this patient, as immediately he saw him he took steps to ensure instant operation. The interval between the perforation of the first ulcer and the operation was about eighteen hours, in the second case eight hours. In both cases only about two or three hours elapsed between his first being seen and the operation itself.

A CASE OF CELLULITIS

FOLLOWING INFECTION BY AN ORGANISM SET FREE BY THE WOUNDING OF AN OLD BLIND DIVERTICULUM OF THE LARGE INTESTINE.

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THE discrimination of cases of cellulitis into varieties corresponding with the causative organism is of much importance both with regard to prognosis and treatment, and of late years much progress has been made in this direction.

The case related below appears to warrant the assumption that it is an example of a rare infection possessing distinct and characteristic features. It was at first assumed to be an ordinary infection by the *Bacillus coli communis*, until the entire absence of gas formation as a part of the process was noted. This clinical fact was later fully substantiated by the results of cultural experiments made with the organism, which showed the bacillus to be both clinically and in cultural characters distinct from *Bacillus coli communis*.

The original development of the organism in a portion of the large intestine excluded from the rest of the alimentary canal is of interest as possibly explaining its presence and peculiarities, since it appears to be of the same nature as certain organisms cultivated from faeces by Houston.

History.

A. B., a seamstress, aged 35, had undergone inguinal colotomy fifteen years ago, in order to exclude a sinus into the rectum which had resulted from a former operation for the removal of diseased Fallopian tubes, which plastic operations had failed to cure. The artificial anus established at that time worked well, it was of small and convenient size, the bowels acted once daily, there was never any prolapse, the faeces were readily controlled by apparatus, and the patient followed her occupation without any disadvantage.

Eight months ago, however, a sudden attack of intestinal obstruction occurred, the patient was admitted into the hospital, and it being assumed that contraction of the artificial anus was the cause of the trouble, the latter was dilated, and after this the bowels again acted freely.

Two days ago the patient took a dose of castor oil, which was followed by a free evacuation, but last night she was seized with sudden pain in the lower abdomen, and since then she has vomited frequently and neither faeces nor flatus have been passed.

Condition on Admission.

The patient complained of acute pain and was vomiting frequently. The tongue was clean, pulse 120, temperature normal.

On inspection the abdomen was flat and not distended nor tender, the colotomy opening admitted the forefinger, the colon appeared empty, and this corresponded with the negative result of an enema given in the morning. The scars corresponding to the old fistula and the operations for its attempted closure were sound; beneath the centre one some hard masses, about the size of a walnut, seemed to indicate the presence of old faecal lumps.