ptom. The relations of peristalsis are rather the following. Numerous clinical observations show that in acute diffuse peritonitis normal evacuation, that is, a normal degree of peristalsis, is found in individual cases. In puerperal peritonitis peristalsis may be even increased. To this one might fairly reply that the cause of diarrhoea was not the anatomical process of inflammation as such, but other causes acting indirectly. However, clinical observation and the results of experiments upon animals which Grünbaum undertook at my suggestion, teach us that in other forms of peritonitis also a distinct increase of peristalsis may be present at the beginning. Hence paralysis of the bowel can in no way be described as an invariable symptom of acute diffuse peritonitis.

Such clinical observations as Professor Nothnagel refers to may be found in Treves's well-known lectures on peritonitis.⁵ He says:

In 100 cases of peritonitis from the London Hospital the bowels were classed as "loose" in 28 instances. In some of these 28 cases there was actual diarrhoea, in other the bowels acted with frequency and without artificial aid; while in a third series of cases a free action was maintained by enemata or by aperients. In two cases of peritonitis in which diarrhoea was met with it was noteworthy that the peritonitis did not appear until after the diarrhoea had set in, a circumstance which suggests the action of the colon bacillus.

By this he seems to indicate that the diarrhoea might have led to the diffusion of the colon bacillus, and thus might have caused the peritonitis, but Grünbaum's experiments make it quite possible to suppose that the peritonitis caused the diarrhoea.

But there seems to be still another way in which experiment and clinical observation mutually throw light on one another. In the Bradshaw Lecture for 1892 upon the Signs of Acute Peritoneal Disease, Dr. Gee makes the very interesting suggestion that the pain of early acute peritonitis is really due in great measure to colic. Thus, after having pointed out how difficult it sometimes is to distinguish colic from peritonitis, he continues:

But it may be that in contrasting thus the pain of peritonitis with the pain of colic, we are seeking for a distinction where there is no real difference. Indeed, it is highly probable that the earlier physicians were often right, and that much of the spontaneous and paroxysmal pain of peritonitis is due to colic, that is to say, to vermicular contractions of the intestines much more powerful than usual. Surgeons tell us that when they open the abdomen in order to remove an ovary, the intestines are seen to be at first in very active movement; and that these movements may be painful is proved by what we sometimes observe in intestinal tympanites, when the peristaltic contractions of the bowels can be seen through the abdominal walls, each contraction being attended by an unnatural sensation or by actual pain.

From some remarks in other parts of this lecture, it would appear that Dr. Gee did not consider that peritonitis might cause an increase of ordinary peristalsis. Had he done so, however, it would only have increased the probability that his suggestion as to colic was correct.

We are now, I think, justified in believing that as the result of irritation round the intestine (produced most frequently by peritonitis), the following symptoms may be produced, that is, increased peristalsis amounting sometimes to severe spasm in the small and greater portion of the large intestine, and relaxation of the sphincter, frequent evacuations, and, in severe cases, intense straining and tenesmus, when the walls of the rectum are involved.

It is not, however, necessary to suppose that such symptoms will be prominent or even recognisable in every case of peritonitis or of perirectal irritation. If we take into account the following considerations this will not be difficult to understand. The peristalsis of the small intestine may be so irregular or so transient that it may not show itself as diarrhoea. Grünbaum found that the large intestine became paralysed before the small; hence the effect of the increased peristalsis might only be to fill the large intestine with an accumulation of faeces and gas. Again, in relation to the rectum it might be that certain forms of organism which often produce pelvic abscesses such as the gonococcus do not cause an irritation which is sufficiently intense to give rise to the symptoms in question. Or, again, it may be necessary that a certain area of the rectal wall should be affected in order that a perceptible result should be produced, and pre-existing adhesions may prevent this.

But, on the other hand, it seems not unreasonable to extend our view of the possibilities of reflex effects upon the intestine. If the afferent nerves of the intestine (whether of the mucous or peritoneal surfaces) can reflexly stimulate the intestinal muscular coat, why not afferent nerves belonging to the same splanchnic system but coming from other viscera in the abdomen, pelvis, or even thorax? This would explain the presence of colic from renal or biliary calculus, or from a severe onset of pneumonia, as in such cases as Mr. Symonds

has recently referred to.⁶ Already we know that the walls of the stomach are reflexly affected by such irritation, for vomiting is a common symptom in such cases.

Although we have confined our attention to reflex excitement of the intestinal muscular coat from irritation, we must not forget the possibility of a reflex paralysis coming on at a later stage, and thus increasing the paralysis due to involvement of the muscular coat itself.

In conclusion we may briefly consider some of the advantages to be derived from the knowledge gained from this study of a few symptoms:

1. Such cases as the first two which I have just described will be clearly understood, and therefore promptly and efficiently treated when they present themselves.

2. The possibility that increased action of other parts of the intestine may be due to a cause outside and not inside the walls of the bowel may often help us to account for otherwise obscure symptoms in ways perhaps that we cannot as yet realise.

3. The probability that colic may in like manner be caused by irritation outside as well as inside the bowel may be most valuable in helping us to form a diagnosis in a difficult case. Hitherto most of us have only recognised that many of the symptoms of peritonitis and of colic (intestinal, renal, or biliary) are often very much alike. Now we may be prepared to believe that some of them are identical. This, instead of discouraging us, should have the opposite effect. We shall know what symptoms to accept as possibly common to several causes, and we shall not put too much stress upon them. This will make us seek more eagerly for points in differential diagnosis. Thus perhaps symptoms which otherwise might have been missed or passed over as unimportant will be found lying ready to our hand as invaluable guides when we understand how to look for and use them.

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TWO CASES OF OPERATION FOR PERFORATING GASTRIC ULCER.

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THE first case of perforating gastric ulcer I saw in 1896, in consultation with Dr. Bennett, who, having made the diagnosis, telegraphed me to bring instruments.

CASE I.

Previous History.—The patient was a girl of 19, well developed, and exhibiting no obvious signs of anaemia. She was a shop-girl, and had been accustomed to long hours and hurried meals. When 9 years old she had been treated for a tuberculous elbow, and later for a tuberculous dactylitis. For the last seven years she had enjoyed the best of health. Until a fortnight previously to her present illness, apart from a very occasional dyspepsia generally due to some dietetic indiscretion, she had exhibited no signs of ulcer. She admitted being treated for palpitation two years before, and that she had been ordered to take iron pills.

History of Attack.—A fortnight before I saw her she had returned from her summer holiday, and after an attack of sea sickness had complained of pain after every meal. From its severity, the ease with which it could be localised, and the tenderness on pressure, the doctor diagnosed a gastric ulcer, and gave full instructions as to diet. A few days later the urgent symptoms arose, and Dr. Bennett hurriedly attended. He found the girl in very great pain, extremely tender over the abdomen, with rapid pulse and drawn face.

Operation.—I was sent for, and in less than 3 hours after the onset of acute symptoms the operation was begun. A median incision was employed, and the stomach explored, when a small opening was found about large enough to admit a crow quill. Through this some food had travelled, consisting of partially digested milk. There were no adhesions, and barely any signs of even local peritonitis; a little lymph surrounded the orifice, which could be removed with the finger. The edges of the ulcer were not indurated. There had been some escape of gas into the abdomen. The ulcer was situated on the anterior surface of the stomach, a little below the lesser curvature and quite at the pyloric end. There was nothing, therefore, to prevent a rapid inversion of the ulcer, and having roughened the parts around, a row of Lembert sutures completed the occlusion. The abdomen was irrigated, and the wound was closed without drainage.

After-history.—The patient vomited twice during the night, and complained of great pain over the abdomen, and the temperature the following day was 101° and the pulse 115. Two days after the operation there was no pain, the tongue was moist, the pulse 66, the temperature normal, and the patient evinced but little desire for food or water. On the third day she was fed on small quantities of peptonised milk, which gave her no incon-

venience. In three weeks after the operation she was sitting up, and in seven weeks, having taken fourteen days' holiday, she resumed her work. Nearly three years have passed since the operation, and I have every reason to believe she is still well.

The operation lasted less than twenty minutes, and every circumstance favoured her recovery.

The small size of the perforation, the absence of induration, the condition of surrounding peritoneum, the site of the ulcer, the small quantity and character of the escaped contents, and the fact that only three hours had elapsed since the rupture, all rendered the prognosis good and the operation easy. From a study of the girl's history, and the appearance of the ulcer, the possibility of a tuberculous origin is worthy of note.

The second case was sent to me at the Royal Southern Hospital on November 12th, 1898, by Dr. Humphreys, who had diagnosed a perforation:

CASE II.

History of Attack.—The patient was a woman, aged 21. Thirteen hours previous to admission she was seized with sudden violent abdominal pain and vomiting. I was in the hospital when she was admitted at 3 o'clock. She looked very distressed, complained of great thirst, and suffered intense abdominal pain. The pulse was 120, very weak, but not running in character. Her extremities were cold, and the abdominal muscles very rigid. A rub could be heard accompanying respiratory movements over left lower ribs in front.

Operation.—We decided to operate at once, and I wish to acknowledge the valued help of my colleague, Dr. Alexander. On opening the abdominal cavity the surfaces of the colon and stomach were seen to be coated with lymph, and to have become adherent to the lower surface of the liver. Adhesions had also formed between the stomach and the abdominal wall. These adhesions were carefully separated, revealing behind a cavity from which oozed brown fluid containing partially digested food. The opening from which this came was found high up beneath the liver on the anterior surface nearer the cardiac than the pyloric end. There was considerable difficulty experienced in bringing the ulcer sufficiently to the surface to enable us to stitch. The perforation would about admit a small finger. It was indurated and surrounded by lymph, which was firm and adherent, and was carefully removed by a Volkmann spoon. We then proceeded to introduce sutures, but found they immediately tore through. A silk suture was therefore passed as near the interior of the stomach, as we felt to be safe, and made to surround the gastric opening. This was tightened in purse-string fashion and leakage prevented. The wall of the stomach was further brought over it and fixed by a row of Lembert sutures, while this was further fortified by sewing omentum over the region of the ulcer. The operation area was carefully irrigated with saline solution, and a gauze drain inserted. The patient seemed to bear the operation without material increase of shock, and saline injections *per rectum* were given for the dual purpose of restoring vitality and allaying thirst.

After-history.—On the next and succeeding days she was starved with the exception of a drachm of water every hour and enemata of brandy, peptonised meat and milk. On admission her temperature was 100.8, and for the first four days it did not rise. Towards the end of the fifth day it fluctuated, its highest point being 101.2. From this time to the fourteenth day it often reached 102, but the patient in all other respects, save a rapid pulse, seemed fairly well. On the eighth day the drain, which had become adherent, was removed. My house-surgeon, Dr. Owen, to whom I am indebted, noted on the fourteenth day: "Patient, after the operation, progressed favourably up to last night, although throughout the fourteen days the breathing was a little rapid. She was, however, able to take bread, and a fair amount of beef-tea, milk, etc., and had not vomited since operation, and the temperature for the last week had reached 102° in the evening." She had no rigors.

Second Operation.—At 3 A.M., fourteen days after operation, she suddenly developed a ticklish cough, and soon expectorated a quantity of foul-smelling pus. Her breathing became rapid, and at 6 A.M. she looked cyanosed. I was away from home, but Dr. Alexander was fortunately at hand, and at 10 A.M. the patient was anaesthetised and the old sinus explored. There was no collection of pus found. An exploring needle was put into the thorax but only clear fluid withdrawn. The needle was next passed into the left hypochondrium and pus withdrawn: this was followed by a free incision giving exit to a quantity of fetid pus. The cavity was well washed out. The patient was throughout the proceedings very collapsed, and various restoratives were prescribed, including strychnine, digitalin, saline solution in axilla, brandy enemata, and sinapism to præcordium. At noon she had sufficiently rallied to inspire hope. In the evening she died.

Necropsy.—A *post-mortem* examination was made by Dr. Owen and Dr. Humphreys. They report that the sinus which was left at the top of the original laparotomy wound did not lead to any collection of pus. The upper surface of the liver was adherent to the lower surface of the diaphragm by very firm adhesions. The repair of the stomach performed ten days before was seen to be healed and the occlusion complete. The abscess cavity which was opened the day of patient's death was seen to lead towards the posterior part of the under surface of the diaphragm. On separating the left lung from the diaphragm to which it was firmly fixed, a small hole was discovered entering lung tissue and perforating diaphragm and discharging foul smelling pus. A probe passed through this perforation entered the abscess cavity explored the previous day by my colleague. On slitting up the walls of this cavity it was seen to be bounded by the under surface of the diaphragm above, and externally by the colon stomach and thickened neighbouring tissues below; its posterior part was the opening communicating with the left lung. The spleen was contained in the centre of this abscess cavity. On opening the chest some clear serum and lymph were found in the left pleural cavity and several pleuritic adhesions were apparent. On distending the stomach no fluid escaped. The wound in the viscus had quite healed, but four of the silk sutures were *in situ*, the others had disappeared. On the

mucous surface a small depression showed where the ulcer had existed. There was no ulcer on the posterior surface.

A greater contrast between two cases could hardly be found. The first was as simple as the second was complex. The fact that operation was not performed for over fourteen hours after perforation was itself sufficient to decide the issue. The adhesions and the involvement of the cardiac end, fixed as it is, rendered suturing a matter of considerable difficulty.

It is interesting to note that over 95 per cent. of cases unoperated upon die. Life is rarely prolonged over twenty-four hours. Ulcers are more frequent on the posterior than the anterior surface of the stomach, but perforations are much more frequent on the anterior aspect. This is probably due to the greater facility with which buttressing adhesions form behind. In 31 cases operated upon, collected by Barling, 22 were on the anterior surface, 5 on the posterior; subsequent cases confirm this proportion.

What was the cause of this perisplenic and subphrenic abscess not connected with the wound area? Had we found an ulcer on the posterior aspect it would have explained much; but it is noteworthy that cases are recorded where retrogastric and perisplenic abscess have caused death where perforation has occurred on the anterior surface alone. This may be accounted for in one of two ways: either by a direct trickling of septic material through the foramen of Winslow into the lesser peritoneal cavity or by lymphatic absorption. In an ulcer on the anterior surface the gastric contents have but to travel round the right free border of the lesser omentum, and both the greater and lesser peritoneal cavities are soiled; and if the foramen of Winslow be closed by adhesions the invasion of an abscess from the lesser cavity will be obstructed.

This raises a final question suggested recently by Willoughby Furner, of Brighton: Would it not be wise in all cases both to examine the posterior surface of the stomach and to irrigate the lesser peritoneal cavity, which can easily be effected through the layers of the great omentum or of the transverse meso-colon? The mere irrigation of the greater cavity may be the very means of infecting by septic particles the lesser one, and a survey of reported cases convinces me that where practicable this should be done. By such a precautionary measure the fatality attending operations for gastric perforations would, I venture to predict, be considerably lessened.

EXOSTOSIS OF THE VERTEBRAL BORDER OF THE SCAPULA: REMOVAL

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Exostoses of the scapula requiring removal are certainly unusual, but in the following case the size of the tumour and the deformity produced rendered such a proceeding desirable.

L. F., a little girl nearly 9 years old, was sent up to me by Dr. Wallace of Earl's Court and admitted into St. Thomas's Hospital for operation on June 23rd, 1898.

The history given was that she had had the swelling nearly all her life, but recently it had not seemed to grow. There was no complaint of pain or of any inconvenience. The parents thought that it might have been due to lifting the child by the arms.

On examination, the right scapula appeared "winged," its inferior angle being raised off the chest wall and displaced upwards and outwards. This proved to be due to a hard nodular tumour springing from the vertebral border of the scapula just above the inferior angle, and having a base of attachment about two inches across. In spite of this mechanical displacement, the muscular movements of the bone were normal. The tumour had caused retardation in the development of the bone so that the vertical distance from the spine to the inferior angle was one inch less on the right side.

On June 28th the tumour was removed. An incision was made over the tumour in the axis of the vertebral border, and this was converted into a T shape by a horizontal cut on its inner side. The thinned lower border of the trapezius was