A Paper
ONACUTE PERFORATING DUODENAL AND
GASTRIC ULCERS.*

BY

JOHN H. WATSON, M.B., B.S., F.R.C.S.,
SURGEON, VICTORIA HOSPITAL, BURNLEY.

THE earliest account of a perforating gastric ulcer was presented to the Royal Society by Christopher Rawlinson in 1729, and the earliest description of a perforated duodenal ulcer was written by Jacopo Penada of Padua in 1793. Dr. Samuel Gee in his Bradshaw Lecture for 1892 gave a translation from the French of a most graphic account written by Madame De La Fayette of the death agony of Henrietta, Duchess of Orleans, on June 26th, 1670, at St. Cloud. It was thought by the English Royal House that this unfortunate Princess had been poisoned at the instigation of her husband; but Littre in 1872 pointed out that the description of the Duchess's acute illness and the post-mortem findings were without doubt typical of a perforated gastric ulcer. In the *Bibliothèque Nationale* there is preserved a "Mémorial of a Surgeon of the King of England who was present at the opening of the body of Madame" which contains these interesting comments:

"On opening the abdomen fetid gas escaped; the whole of the abdomen was full of a sanious, putrid, yellow, watery fluid, mixed with oil; the outside of the stomach looked healthy and the inside was free from disease except for a little hole in the middle of the anterior wall, which had been accidentally produced by the surgeon with the point of his scissors."

Benjamin Travers (a former President of the Royal College of Surgeons) in 1816 described very accurately several cases; two of these, which I take the liberty of quoting, have very interesting post-mortem findings.

"Case 1.—Mr. —, aged 35, of a strumous habit but enjoying generally good health, was seized whilst dining in company, with an excruciating pain in the abdomen, which he described as unlike any he had ever felt. . . His abdomen was tense and hard, his respiration somewhat agitated, flatus rose in quantity from his stomach, but he had no disposition to vomit . . . and he died in a warm bath at 6 a.m., sixteen hours after the attack of pain. Post mortem a large quantity of fluid tinged with bile was found contained in the pelvis; and about a finger's breadth below the pylorus appeared a circular foramen, having a peritoneal margin of the diameter of a writing pen. It proved to be the centre of an irregular superficial ulcer of the mucous coat, including in its extent two-thirds of the ring of the pylorus."

"Case 2.—Mills, a hairdresser, had occasionally for the seven preceding years suffered sudden and very violent attacks of abdominal pain, from which he had always been speedily relieved by a wine-glass full of brandy, on the day of the fatal attack enduring severe pains. . . On reaching home this pain became intolerable and he died 36 hours from the commencement of the intense pain. Post mortem a circular aperture of the peritoneum, large enough to admit a crow's quill, was found at the junction of the duodenum and stomach."

Travers sums up the principal signs of these perforating ulcers in a paper published in the *Medical and Chirurgical Transactions* of 1817, in the following striking way:

"Sudden most acute and unremitting pain, radiating from the scrobiculous cordis or the navel to the circumference of the trunk, and even to the limbs: its intensity absorbs the whole mind of the patient, who, within an hour from the enjoyment of perfect health, expresses his serious and decided conviction that if the pain is not speedily alleviated he must die: coeval with the attack of pain there is a remarkable rigidity and hardness of the belly from a fixed and spastic contraction of the abdominal muscle."

Apart from Travers there was no contribution of any importance to the literature of perforating ulcers of the stomach in the early nineteenth century by any English surgeon, but several physicians, including Matthew Baillie, John Abercrombie, Sir George Baker, William Brinton, and George Budd, wrote descriptive articles in which they described not only ulcers of the stomach, but also ulcers of the duodenum. Abercrombie, in fact, suggested a

dietary which in many respects resembles the routine medical diet of to-day. Brinton, who made a remarkable number of post-mortem investigations, and analysed some 1,200, makes the statement that of the less frequent ulcers on the anterior wall of the stomach as many as 85 per cent. perforate, and most of them into the general peritoneal cavity.

Until the close of the nineteenth century it was generally considered that a perforation of any gastric ulcer into the general peritoneal cavity meant certain death. In the year 1888, however, Mikulicz attempted to close an acute perforation of the stomach by suture, but without success; a successful case was reported by Heusner in 1892. At the British Medical Association Meeting at Bristol in 1894 Pearce Gould opened the discussion on the operative treatment of perforating gastric ulcers, and recorded the fact that he knew of only seven cases in which an operation had ended in success. In this same year H. P. Deane of the London Hospital recorded a case in which he had successfully sutured a perforated duodenal ulcer; and another was reported by L. A. Dunn in 1896. Since this time there has been an ever-increasing number of cases recorded each year, and there is a marked improvement in the results of operative treatment. This is shown by the following table, which gives the figures of a dozen English surgeons.

		Cases.	Died.	Mortality Percentage.
Caird, F. M.	1896-1903	54	34	63.0
	1904-1913	193	71	36.7
Short, R.	1911	58	26	44.0
Sargent, P.	1904	49	21	41.9
Morley, J.	1912	112	48	42.9
Sherren, J.	1912	28	12	42.8
Struthers, J.	1920	90	20	22.2
Southam, A. H.	1922	40	3	9.0
Mills, P.	1925	40	11	27.5
Moyrihan, B.	1920-1925	237	55	25.0
Turner, G. G.	1903-1927	194	48	25.0
Williams and Walsh	1922-1929	158	47	29.7
Watson, J. H.	1922-1930	110	21	19.1
		1,363	417	31.6

Undoubtedly with earlier diagnosis and prompt surgical treatment this high mortality rate should be very considerably lowered in the future; in fact, Professor Young of Glasgow had a remarkable series of 17 cases of perforation of pyloro-duodenal ulcers between July, 1926, and July, 1927, without a death, and remarks that thirty years ago such a succession of good recoveries would have been regarded as impossible.

The series of 110 cases of perforating gastric and duodenal ulcers here recorded include 7 occurring in my private practice, and the remaining 103 in hospital practice (Victoria Hospital, Burnley, 85 cases; Reedyford Hospital, Nelson, 14 cases; Hartley Hospital, Colne, 4 cases). Of this number 20 have been operated on by my colleagues, Messrs. Mackenzie, McCreadie, and Davidson, at the Victoria Hospital.

In this list there are only eight women patients, and of these six were labelled gastric perforations and two duodenal. Of the 102 cases occurring in males, roughly one-sixth are recorded as being situated in the pyloric portion of the stomach, and the other five-sixths perforated either at the pyloro-duodenal junction or in the duodenum.

There was one case only of a posterior perforation, occurring in a man aged 63 years; another perforation was recorded as being about the middle of the lesser curvature; and there were two other cases in which perforation took place nearer the cardia.

As to the age incidence the conditions appear to be most common in the third and fourth decades, the youngest

* Read before the Manchester Surgical Society, March 18th, 1930.

subject being 16 years, the oldest 67: both these had distal ulcers close to the pyloro-duodenal junction. In my experience this acute abdominal crisis occurs most frequently in the winter months, especially about Christmas time. No case of perforating malignant ulcer has been included.

As a general rule there was a previous history of indigestion over a varying period of a few months to several years. There were, however, two instances in which the history of previous ulcer symptoms was doubtful. The time between the acute symptoms and the time of operation has unfortunately not been accurately recorded, but I have a series of cases from one hospital (Nelson) in which these data have been carefully noted, and it varies from two hours to sixteen hours; the average interval accordingly is seven hours in these cases.

Age.	Occupation.	Site.	History.	Operation.	Perforated.	Result.
			Years.		Hours.	
29	Clerk	D.U.	5	25/6/25	5	Good health.
31	Ex-soldier	D.U.	Nil.	15/10/26	6	Good health.
49	Weaver	G.D.U.	2	3/6/27	6	Good health.
53	Postman	D.U.	1	4/5/27	16	Occasional indigestion.
45	Shopman	D.U.	3	7/6/27	3	Good health.
45	Twister	D.U.	4	31/7/27	5	Occasional indigestion.
39	Weaver	D.U.	1½	5/3/28	6	Good health.
38	Overlooker	D.U.	10	1/9/28	7	Good health.
58	Weaver	D.U.	2	31/10/28	4	Good health.
36	Weaver	P.U.	6	6/1/29	2	Good health.
44	Weaver	G.D.U.	3	19/12/29	14	Died, 3 hours.
41	Manager	G.D.U.	3	21/12/29	4	Died, 24 hours.
31	Weaver	P.U.	2	8/1/30	6	Good health.

In my experience the most common causes of death in the fatal cases have been: (1) shock from perforation and operation; (2) peritonitis and paralytic ileus; and (3) broncho-pneumonia.

When considering the site of the perforation it is interesting to compare a series of diagrammatic representations given by various authorities during the past twenty-five years (for instance, those of Sargent,¹ Morley,² Deaver,³ Stewart,⁴ Einhorn⁵). It will be seen that most of the ulcers are indicated as being near the lesser curvature, and in the later diagrams most are located in the neighbourhood of the pylorus, with a definite majority on the duodenal side. Posterior perforations are relatively rare, and practically none seem to occur near the greater curvature.

My experience leads me to believe that perforating ulcers, particularly of the duodenum, have become more frequent in recent years, especially in men. The men affected are mostly of the artisan class employed in mills and workshops, who are careless about their food, their habits, and general mode of living.

The first cases I had to deal with twenty years ago were generally gastric ulcers which had perforated some days previously and were admitted to hospital with a subphrenic abscess: one woman, I remember, vomited a large amount of purulent material and died shortly after admission. At the post-mortem examination there was discovered a subphrenic abscess connected with an abscess at the base of the right lung.

The incidence of proximal perforations (gastric) is said to be more frequent in women than men, but I have not seen a case of perforated gastric or duodenal ulcer in a woman in the last two years. It is difficult to account for this increased number of perforating ulcers in men; I have no satisfactory explanation to offer, but I am quite sure that they are being discovered earlier and more often, for which full credit must be given to the general practitioner.

Peritonitis resulting from leaks of this character fortunately differs materially from peritonitis resulting from an acute perforating appendix. It is less virulent

for at least two reasons: (1) the contents of the stomach and duodenum are practically sterile shortly after a meal; (2) the patient is most likely a chronic dyspeptic and is probably taking only small quantities of light food. When, therefore, rupture occurs, as it does most frequently three or four hours after a meal, the extravasation is in many cases sterile. This is not an absolute rule. Dugeon⁶ found streptococci present in 12 out of 23 cases, but the organism was of low virulence. The *Staphylococcus albus* was present in 10 cases, a sterile exudate in 5, but he emphasizes the fact that when the colon bacillus is present the infection is always more severe. The time at which perforation occurs is therefore highly important as regards the toxicity of the exudate. Perforations occurring late during the process of gastric digestion are often more or less innocuous because of the small number and low virulence of the bacteria present. This is a very good reason for not draining the peritoneum, which is well able to take care of this type of infection if the primary leak is promptly controlled. When, however, the rupture occurs immediately after the digestion of a large meal with plenty of fluid, and where the amount of extravasation is great, the response of the peritoneum is by no means so satisfactory and the resulting infection may even be overwhelming.

GROSS PATHOLOGY.

There are certain striking gross pathological features of these juxtapyloric ulcers which call for remark. In the first instance they may be grouped into pre-pyloric and post-pyloric types.

Pre-pyloric Type.—There is usually considerable infiltration and thickening of the wall of the pylorus, thus encroaching on the lumen; adhesions may or may not be present.

Post-pyloric Type.—This type presents a varying amount of scar tissue, depending on the kind of ulcer, which may be: (a) Medium-sized ulcer with considerable scar tissue formation and more or less extensive adhesions deforming and constricting the lumen; the ulcer tends to spread on to the pylorus. (b) Small ulcer with slightly indurated base; there is a marked absence of adhesions. (c) Multiple ulcers of the so-called "kissing" type are rare.

The perforation may vary from pin-point to 6 mm. or 7 mm. in diameter, and is typically a punched-out hole in the crater of the ulcer. Generally speaking, most acute perforating duodenal ulcers are single, round, slightly indurated, 1 cm. to 2 cm. in diameter, nearly always on the anterior surface of the first one and a half inches of the duodenum. A great number occur in the duodeno-pyloric formix, as pointed out by Jefferson.⁷ The depth of this sulcus varies considerably, being shallow in relaxed hypotonic stomachs and well marked in gastric ulcer, where gastric hypertonus is the rule. These duodeno-pyloric (Jefferson) or pyloro-duodenal (Young) ulcers are for the most part duodenal, but because the pyloric veins are frequently obliterated in these cases many have been probably classified wrongly as pyloric ulcers.

When once an ulcer has eroded through the mucous coat and well into the muscular lining of a hollow viscus its progress depends primarily on two circumstances: either the serosa becomes glued to some adjacent structure or an acute abdominal crisis is precipitated by sudden rupture. The ulcers which are most likely to perforate into the general peritoneal cavity are obviously those on the anterior wall of the stomach and duodenum, where the possibility of the formation of adhesions is least probable. When destruction has proceeded to such an extent that the subperitoneal coat is reached, a sudden intense muscular spasm is likely to cause a rupture of the ulcer base.

From the above-mentioned pathology these perforating ulcers may, for practical purposes, be placed in two classes: (1) the smaller ulcers with little induration; (2) the larger calloused ulcers, causing more or less deformity and producing a varying amount of obstruction to the exit of food from the stomach.

The condition of the peritoneum at the time of operation depends in general upon the time that has elapsed since the actual perforation. I have found that the amount of fluid present within the peritoneal sac is very variable, even in the early cases; generally speaking, there is more

in cases of duodenal ulcer than in cases of gastric ulcer. This may be accounted for partly by the fact that the duodenal perforations are, on the average, larger than the gastric; further, the calloused pyloric ulcer is often associated with a contracted and deformed lumen, which blocks to some extent the escape of the contents. Much depends also on whether the unfortunate patient has been plied with drinks of various kinds, administered by anxious and well-meaning relatives, and on the intensity of the pylorospasm. As might be expected, many of my cases of the duodenal type perforated at a time when the stomach was, or should have been, empty—that is, when they were finishing the morning or afternoon work or going to bed at night—at a time when they would be beginning to feel hunger pains.

The response of the peritoneum to this leaking fluid I have found to be very variable. As a rule, in the early stages the gastric contents are more irritant than the duodenal; there seems to be more sero-pus and inflammatory lymph deposited about a leaking gastric ulcer. The duodenal contents fortunately are deflected to Morrison's pouch, and are then diverted to the pelvis along the outer side of the ascending colon, so there may at first be very little evidence of peritoneal irritation.

Apart from the extravasated fluid in these cases there is probably a considerable exudate produced by the peritoneum itself. On every occasion I have tested the accumulated fluid it has been neutral to litmus. There is a considerable amount of mucus in some instances, and bile, in large amount, in late cases of duodenal perforation. I have rarely seen particles of undigested food; the amount of free gas in the peritoneal cavity is also very variable. I have never had occasion or the opportunity to demonstrate a pneumo-peritoneum by radiography. As time goes on the peritoneal exudate becomes more purulent, and it is an established fact that, after six hours, the majority of cases are infected with staphylococci, streptococci, and *Bacillus coli communis* of virulent type; hence the need of a rapid and careful peritoneal toilet. In late cases with profound infection the peritoneal vessels will be found engorged with dark venous blood, and there is little or no bleeding from the wound—a bad omen.

SYMPTOMATOLOGY.

The description by Travers, already given, of the onset of the symptoms is brief, exact, and impressive; I have failed to find any other written account which gives so accurately the predominant signs. For the sake of analysis they may be tabulated.

1. Acute onset of agonizing pain, which entirely absorbs the patient's attention.
2. General deep tenderness of the abdominal wall.
3. Early and intense boardlike rigidity of the flat muscles of the abdominal wall.

In addition to these definite and most urgent symptoms there is usually associated:

4. A slow pulse of good volume and slightly raised or normal blood pressure in this early stage.
5. A subnormal or normal temperature.
6. Nausea and vomiting present in the majority of cases.
7. Shallow and rapid breathing (upper thoracic type).

Other less constant signs may be noted or elicited, such as superficial hyperalgesia, shoulder-tip pain, pallor and flushing of the face, sweating of the head and chest, pneumo-peritoneum, and loss of liver dullness. The patient is frequently very restless, moaning and groaning with pain; occasionally he holds himself very rigidly, lying on the back with the knees flexed. The presence of free fluid in the abdomen, abdominal distension, cold extremities, and evidences of profound shock and toxæmia are very late and ominous signs.

Of the predominant symptoms I have just tabulated the most urgent is undoubtedly the agonizing pain which is usually referred by the patient to the epigastric region. I have found difficulty in judging the exact site because the patient is so distraught by his suffering that he cannot sufficiently concentrate to give reliable answers, and usually dismisses the question abruptly by saying "It is all over," or "Give me something to ease the pain."

As to the actual cause of the intense pain, much has been written of a more or less discursive and indefinite character; this is probably due in part to the fact that

our knowledge of the visceral nerves and their function is as yet incompletely understood.

We know that visceral organs are not subjected to the same kind of stimuli as those which constantly play upon the surface receptors; further, hollow viscera are insensitive to these particular forms of stimulation (mechanical, thermal, electrical). In the usual course of events the reactions called forth in visceral organs do not affect consciousness; on the other hand, abnormal visceral stimuli give rise to sensations which are commonly referred to a somatic area removed from the point of stimulation.

The normal functioning of a hollow organ like the stomach involves the contraction of smooth muscle, but under physiological conditions this does not generally give rise to sensations. The actual adequate stimulus for the production of pain in the stomach which is the seat of an ulcer and which has suddenly perforated must in some way be related to the contraction of the unstriated musculature. This is what Mackenzie⁸ termed the production of pain by a "vital" stimulus, as opposed to the "mechanical" stimulus which is adequate to produce pain in other tissues. The predominating result is pain referred to certain areas of the outer body wall, but the impulse may spread widely according to the intensity of the stimulus and so progressively involve other visceral centres in the central nervous system, by this means producing vasomotor, cardiac, respiratory, and other reflex disturbances.

Hughson⁹ has expressed the opinion, from his experimental studies on dogs, that spasm of the pylorus from disease within the abdominal cavity is caused by irritation of the peritoneal surface. It is evident that with a perforation there is immediate soiling of the peritoneal surface by an irritant fluid, which would accordingly be sufficient to cause an intense reaction in the nature of a violent pylorospasm, reflex in character, and involving both vagal and sympathetic neurons. It has been shown by fluoroscopic examinations of patients with gastric ulcer that the pain in many instances coincides with the appearance of peristaltic contractions sweeping over the pyloric portion towards the contracted pylorus.

The operative findings in cases of perforating ulcers confirming these statements have, so far as I am aware, not been recorded, but I have on several occasions noted the sphincter pylori standing out as a thickened pallid band, and found on exploring the pylorus from the duodenal side that the pyloric canal was so contracted that it was difficult to find, and could only be forced by strong digital pressure. This narrowing of the lumen, I observed, was not due to a cicatricial constriction but to a contracted sphincter pylori, which gave way to steady pressure, and on dividing the sphincter later the hypertrophied muscle fibres were clearly demonstrated.

Mackenzie's early personal experiences as a surgeon in the first instance led him to believe that the violent contraction of the smooth muscle of a hollow viscus was the major factor in the production of visceral pain, and the weight of evidence to my mind appears to support his argument that under certain circumstances when the contractions of a hollow muscular canal like the pylorus become violent or spastic, or when the blood supply to the muscle is greatly diminished, the contraction of smooth muscle gives rise to pain; in fact, the most severe pains in the epigastric region are probably due to pylorospasm.

This pylorospasm depends upon an excessive activity of the visceral nerves of the stomach and duodenum; the stimuli they conduct create, according to Mackenzie, an irritable focus in the spinal cord (seven to twelve dorsal segments roughly). This condition of hyperexcitability profoundly affects the reception and conduction of various regional impulses, so much so that an ordinary tactile impulse in passing through this disturbed centre is so exaggerated that it produces a painful impression in the sensorium. Further, the afferent visceral stimuli may also bring about the phenomenon of referred pain in which the impulse is actually conducted to the surface receptors from the central nervous system along the somatic afferent nerve; and again, by a process of irradiation to other neighbouring visceral centres, various other reflex symptoms of an organic lesion are made manifest.

So far as my observations go superficial sensation does not as a rule appear to be increased in these acute cases; it is only on rare occasions I have been able to demonstrate any actual area of superficial hyperalgesia either of the abdominal wall or about the shoulder tip. But any attempt at deep palpation of the muscles of the anterior abdominal wall will cause severe pain. This tenderness is associated with a rigidly contracted muscle (viscero-motor reflex) and is but an exaggeration of the normal reflex which maintains the tone of these muscles. The reflexes involved in muscular guarding and rigidity are conveyed in part through reflex arcs, which may comprise both sympathetic and somatic neurons. Nevertheless a degree of pressure which when applied to other muscles of the body gives rise merely to a sensation, brings forth in these rigid muscles a sensation of severe pain. This can only mean some special influence is being exerted on these tender muscles, the effect of which is to increase the intensity of their reaction to stimulation—namely, a hyper-excitable centre in the segment of the spinal cord which is associated with the viscus in question.

The fact that the pulse is slow and of good volume in the early stages is an important feature to note, for despite the fact that even recent textbooks still affirm that the pulse is small and thready I have never felt a rapid small pulse in an early case. I have on several occasions had the blood pressure taken and found either a slightly raised or normal blood pressure (140 to 160 systolic)—never an early fall in blood pressure; so that the general statement that shock is a predominant feature in the early cases may and does lead to delay in making a diagnosis, for on several occasions I have been called out to these cases, and the doctor in charge invariably contended that as the pulse was so good he could not conceive that the pain was the result of an acute perforation. Later, of course, the pulse does become small and rapid, and there is a big drop in the blood pressure, but when this stage arrives the prospect of successful surgical treatment is getting beyond the realms of possibility.

With regard to nausea and vomiting, in all cases of intense pylorospasm and pain in the epigastrium there is reflex inhibition of the cardia from sympathetic influences, and at the same time there is the intense contraction of the abdominal muscles; but this is not spasmodic, so that actual vomiting is very slight or may be absent, and at the most consists chiefly of attempts to get rid of very viscid salivary secretion.

Respiratory difficulties are commonly observed to accompany acute abdominal disorders. The rapid and shallow respiratory movement is due to the fixation of the lower chest, principally by reflex spasm of the lower intercostals (fifth to twelfth) and the peripheral portions of the diaphragm (innervated by lower intercostal nerves). The dyspnoea takes the form of difficulty with the inspiratory phase of the respiratory cycle, particularly on an attempt at deep inspiration.

In addition to these predominant signs we meet with the other more variable ones, such as the pallor and flushing of the face, sweating of the face and upper chest, easily provoked pilomotor reflexes, and dilatation of the pupil. These secondary symptoms can also be explained on the theory of disturbed reflexes due to spreading from a hyperexcitable centre in the spinal cord, and by diffusion affecting secondary centres which give rise to the unusual or abnormal vasomotor, secretory, pilomotor, and pupillary reactions.

TREATMENT.

The only treatment for an acute perforating ulcer is operative, and should follow promptly on the diagnosis; with an early diagnosis in an otherwise good surgical risk the chance of cure is infinitely better to-day than the statistics previously quoted suggest. Even after allowing for bad risks, the mortality should not exceed 5 per cent., granted an early diagnosis and that an immediate operation is performed.

One of the first surgeons in this country to give serious consideration to active surgical intervention in these cases was Dobson of Bristol, who in 1883 published a paper in the *Bristol Medico-Chirurgical Journal* on a case of perforated gastric ulcer, and suggested as treatment early laparotomy, cleansing

of the peritoneum, and suture of the perforated ulcer; if this last were not possible, the formation of a gastric fistula.

Eleven years later, 1894, Pearce Gould, discussing the operative treatment of perforating gastric ulcers of the stomach and intestine, advocated the following measures: (1) simple washing out of the peritoneal sac and drainage; (2) suture of the ulcer, with or without excision of the edges, and cleansing of the peritoneum; (3) formation of a gastric fistula or drainage of the stomach, with cleansing of the peritoneum. His great contention was that:

"The key to the position lies in the perfect cleansing of the fouled peritoneum, for nearly every case of failure of the operation has been due to the imperfect cleansing of the serous sac and continuance of the peritonitis."

The method of cleansing advised was irrigation, which took the form of flushing from a douche can or with the use of a Higginson syringe, the solutions preferred for this purpose being either plain boiled water or normal saline, but weak antiseptic solutions (boric, carbolic, and creolin) were utilized by Morris and other believers in the antiseptic system. Drainage of the pelvis was instituted in every case, and, as a rule, a drainage tube was led down to the site of the lesion.

In 1897 Braun of Leipzig published the first case of successful suture in which a piece of omentum was grafted over the perforation, and an anterior gastro-enterostomy completed the operation. Shortly afterwards Moynihan, Patterson, and other British surgeons began to combine the posterior operation with simple suture, and for a time this was the method adopted by the majority of surgeons.

Mothersole¹⁰ published in 1906 two cases of resection and suture of perforated gastric ulcer.

Mills¹¹ reported, in a series of 40 cases published in 1925, better results with the combined operation than with simple suture, but he admits that he performed the simpler operation only in the most serious cases.

Bager's¹² figures, collected from fifty hospitals in Sweden, are very interesting. He has collected 1,767 cases operated upon by about a hundred surgeons during the period 1911 to 1925. Of these, 684 had the operation of simple suture, the mortality being 36.4 per cent.; in 616 cases the suture was supplemented by gastro-enterostomy, and the mortality was 23.4 per cent.; 84 cases were treated by resection, with a mortality of 25 per cent.; in 111 cases in which simple tamponade or drainage alone was instituted the mortality rose to 68.5 per cent. He says that the poorer results of simple suture, compared with the combined operation of suture and gastro-enterostomy, were due undoubtedly to the fact that patients treated by suture alone were the worst operative risks; further, he remarks that cases treated by suture would not have been improved by gastro-enterostomy, but in the cases treated by gastro-enterostomy the mortality would have been somewhat lower had the suture method been adopted. For similar reasons he is convinced that the cases treated by gastric resection were good operative risks, and that the mortality would certainly have been reduced had less radical measures been adopted.

For some years past I have personally given up the combined operation, being convinced in my own mind that the simplest surgical procedures which will correct the defective anatomical findings will give the best results. I venture to submit that in the majority of these cases the quickest, simplest, safest, and most effective technique is to excise the ulcer-bearing area, divide the sphincter pylori, and unite the cut edges of this open wound at right angles to the long axis of the gastro-duodenal tract. By this method the ulcerated area is removed; any secondary ulcer can be seen and treated; a better union is obtainable by the suture of healthy tissues; constriction of the lumen is avoided; and the prospect of future obstruction of the outlet to the stomach greatly lessened, if not entirely obviated.

The operation is performed under local anaesthesia combined with the administration of ether oxygen. Occasionally, in late cases, the operation has been performed with local anaesthesia alone, following Panchet's technique. The abdomen is opened with a paramedian or median incision; the liver is at once lifted up, and the stomach grasped by an assistant and pulled over to the left. By using a moist gauze abdominal compress a firmer hold is obtained, and by this means further leakage can be controlled. If there is a considerable amount of fluid in the abdomen and pelvis a suprapubic puncture can be done at this stage, and a wide, split rubber tube inserted to the bottom of the pouch of Douglas, thus instituting early drainage. Hot gauze packs rung out of saline are then placed in Morrison's pouch and below and internal to the site of the lesion, being tucked downwards towards the pelvis. A good view of the perforation must be obtained if an excision of the ulcer-bearing area is to be undertaken. The excision is made in the

form of a square with the diagonal lengthwise; if the sphincter pylori is not included the incision is continued through its centre. In those cases in which the sphincter is considerably thickened, a wedge is removed on either side of the incision. The sutures are then placed after the manner of a Horsley's pyloroplasty, an inner layer of interrupted through-and-through sutures of No. 1 catgut and a superimposed layer of Lambert sutures of linen or silk. The packs are now removed, and further pockets of exudate mopped up gently with moist gauze. After this toilet of the peritoneum the wound is carefully closed in layers without any local drainage, but the pelvic drainage tube is kept in for twenty-four to forty-eight hours. No drainage at all is required in early cases with little extravasation, and no form of irrigation is attempted.

Immediately after the operation the patient is given a pint of glucose and sodium carbonate solution (5 per cent.) per rectum. If there are indications of shock, as indicated by a weak and rapid pulse and low blood pressure, a subcutaneous or intravenous saline is administered; the patient is returned to bed, maintained in the recumbent position; radiant heat is applied to the abdomen, and the patient is kept under the influence of morphine. The patient is not raised to the Fowler position until the pulse shows a considerable improvement, but on no account is any use made of pituitary extracts.

REFERENCES.

- ¹ Sargent: *St. Thomas's Hospital Reports*, 1904.
- ² Morley: *Practitioner*, 1912.
- ³ Deaver: *Surgery of the Upper Abdomen*, p. 84.
- ⁴ Stewart: *Annals of Surgery*, 1922, p. 351.
- ⁵ Einhorn: *Surg., Gynecol. and Obstet.*, 1930, vol. i, No. 2, p. 418.
- ⁶ Dugeon: *Nelson Medicine*, pp. 5 and 583.
- ⁷ Jefferson: *Annals of Surgery*, 1916, p. 328.
- ⁸ Mackenzie: *The Basis of Vital Activity*, 1926.
- ⁹ Hughson: *Archives of Surgery*, 1924.
- ¹⁰ Mothersole: *Lancet*, 1905, vol. ii, p. 223.
- ¹¹ Mills: *Ibid.*, January 3rd, 1925.
- ¹² Bager: *Acta Chirurg. Scand.*, 1929, lxiv, Supp. xi.

TWO APPARENTLY UNRELATED OUTBREAKS OF BACILLARY DYSENTERY.

BY

A. T. W. POWELL, M.C., M.B., B.S., D.P.H.,

LATE MEDICAL OFFICER OF HEALTH, SCUNTHORPE AND FRODINGHAM
URBAN DISTRICT COUNCIL.

A FAIRLY extensive literature has of late accumulated with regard to bacillary dysentery in Great Britain, suggesting that the disease is endemic and that its unusual prevalence during the past few years is probably only the result of more careful investigation.

The last twenty-four years have shown one or more outbreaks of bacillary dysentery in at least eleven areas; even so, the following recital is probably incomplete: 1906, Birmingham¹; 1911, Paddington²; 1915, Blackwell rural district³ (1,000 to 1,700 cases of diarrhoea mainly dysenteric in character associated with pollution of water supply); 1921, Ogmire and Garw urban district⁴ (1,100 cases—probably a water-borne infection); 1922, Lynton urban district⁵ (18 cases due to milk-borne infection); 1923 to 1926, Aberdeen⁶ (about eight outbreaks, totalling 50 cases); 1925, Ayr⁷ (five groups of 12 cases); 1926, St. Andrews (150 cases)⁸; 1928, Smethwick⁹; 1928, Newcastle¹⁰ (141 cases). Further cases are referred to by Nabarro,¹² Clayton,¹³ and Warren.¹⁴

Textbook descriptions of bacillary dysentery postulate epidemicity where sanitation is defective or where water and milk supplies are infected. Endemicity is regarded as the normal in Eastern cities, while the occasional outbreaks in this country are relegated to asylums. The following account of the Flexner and Sonne outbreaks in Scunthorpe will help to refute the foregoing ideas, and will support the statements of other observers (Fraser, etc.,⁶ Wiseman,⁷ Fyfe,⁸ Charles and Warren,¹¹ Nabarro,¹² and Warren¹⁴), regarding the marked variation in virulence and the unrecognized prevalence of bacillary dysentery.

The Aberdeen, Newcastle, St. Andrews, and Ayr outbreaks in particular emphasized the variable virulence both of Sonne and of Flexner infections. Sonne infections appear to have been regarded as the less serious, which

agrees with Thjötta's observation that the resultant diarrhoea is often not serious enough to warrant the attention of a physician. On the other hand, some of the Aberdeen Sonne infections could definitely be subdivided into severe and mild groups—the former again being subdivided into two further groups. The more serious Flexner type of infection at Newcastle proved especially virulent among children, while adults appeared immune. The experience of the Scunthorpe series agrees in detail with these observations. Another curious analogy with the Newcastle^{10 14} cases is that in both areas the outbreaks were at first regarded as food poisoning, several members of a family being taken ill, with fatalities resulting.

Flexner Outbreak.

On February 4th, 1930, a message was received from the doctor in attendance that the four children, aged 3 to 10 years, of a working-class family residing at Ashby, Scunthorpe, were suffering from what was believed to be food poisoning.

The house was at once visited, and one child (Be. G.), aged 3 years, was found to be dying. There was marked dyspnoea, and Kernig's sign was present. No pulse could be felt; no head retraction was observed; and the temperature was 100° F. The history was that the child had first been taken ill four days previously, complaining of abdominal pain, headache, and diarrhoea. Death occurred on February 4th. Ba. G., aged 8 years, was said to have been delirious on the evening of February 3rd, and within twenty-four hours had a temperature of 102° F. with abdominal pain and diarrhoea. A. G., aged 10, had headache on the morning of February 4th, but wanted to go to school. By the evening the pulse was 140 and the temperature was 103° F., with abdominal pain and diarrhoea. S. G. had a similar onset, the symptoms being complicated by whooping-cough of about seven days' duration. Both the father—an ironworker—and mother were quite well.

On revisiting the patients with the family doctor in the evening the three surviving children were found to be extremely ill with almost constant diarrhoea, the motions being greenish black in colour, resembling spinach, and emitting a peculiar smell which, later, was realized to be almost pathognomonic. There was no obvious blood or mucus, and apparently no vomiting. No splenic enlargement could be discovered, and no rose spots were seen. Specimens of faeces were obtained from each surviving child. A tentative diagnosis of food poisoning was made, but an hour's close questioning failed to suggest any incriminated foodstuff.

The routine procedure suggested by the Ministry of Health was followed, and four food samples were sent to the Ministry's laboratory for examination. In view of the earlier results obtained from the examination of faeces the examination of foodstuffs was not considered necessary. A post-mortem examination was carried out on the fatal case (Be. G.) on February 5th by the patient's doctor, and evidence of an enteritis was found. The following specimens were sent to the Ministry's laboratory: (a) small intestine, (b) kidney, (c) spleen, (d) section of liver, (e) stomach. Unfortunately no part of the large intestine was sent. On February 5th the condition of the patients was critical, especially that of the youngest child, S. G. Specimens of blood were obtained from the parents and the two children A. G. and Ba. G. and dispatched to the Ministry's laboratory.

The house involved was one of nine scattered old houses forming an isolated courtyard, with a common water supply from a shallow well. Immediate instructions were given to boil all water before use. Four pail closets were in use at the onset of the outbreak. The property generally speaking was old, but in fair habitable condition. Only one house was overcrowded by two families of four adults and nine children. New accommodation was found for the subtenants.

Detailed inquiries were made at all the remaining eight houses, but no history of illness was obtained except from the overcrowded cottage where the five children of the