EXPECTANT TREATMENT OF PERFORATED PEPTIC ULCER*

BY

A. DAVIS BEATTIE, F.R.C.S.

Surgeon, Leicester General Hospital

The surgical treatment of acute perforated peptic ulcer has through the past three decades gradually tended to become more conservative. Even the method now generally employed at most surgical centres in this country—simple closure of the perforation by suture or an omental plug without any attempt at careful peritoneal toilet or drainage—has often seemed an almost unnecessary procedure. Surgical intervention sometimes seems to do more harm than good. When operating on such patients every surgeon of experience must often have noticed that the perforation had already undergone spontaneous closure. In most instances this seems to be due to adhesions between the bowel and the under surface of the liver, though occasionally the hole is found to have been sealed successfully by omental adhesions alone. In these circumstances one has often had the feeling that to separate the adhesions and then to suture the perforation is defeating the ends which have already been achieved by the natural healing processes of the body.

The articles of Taylor (1946) and Visick (1946) therefore excited my interest. But it appeared to me that most previous attempts to secure a control series of such cases treated conservatively had been vitiated by limiting the selection to particular clinical varieties. If this method of treatment was to be of use in modern surgery, it seemed that its potentialities ought to be fully investigated by employing it on a sufficiently large unselected series of cases. Moreover, the suggestion in Taylor's article, that conservative measures should be reserved for patients whose stomachs are relatively empty at the time of perforation, did not seem logical. The degree of peritoneal soiling ought to make no difference to the success or failure of conservative measures, provided that any remaining stomach contents are aspirated before expectant treatment begins, as it is never possible to remove all the food debris from the peritoneum even at laparotomy. Nor should the length of time between onset and admission to hospital have any influence on the success of such treatment.

Most of the experimental work on the bacteriological content of the stomach during an acute exacerbation of peptic ulcer suggests that infection is invariably present, so that the accepted division of the resultant peritonitis into an early aseptic and later septic stage would appear to be incorrect. In any case, experience of penetrating war injuries of the abdomen complicated by gross peritoneal soiling and of the surgery of acute abdominal inflammatory conditions shows that the peritoneum is able to deal with an amazing degree of septic contamination provided that this does not continue to take place. It was undoubtedly the progressive irritation of the peritoneum by infected gastric contents which was mainly responsible, before the days of chemotherapy, for the high post-operative mortality in cases of perforation in which there had been a long time delay between onset and operation.

It seemed, therefore, that there would be little additional risk to the patient if expectant measures were employed on every occasion, provided that the stomach was kept empty and that laparotomy was immediately instituted if the clinical condition failed to respond satisfactorily to this treatment. Encouragement to follow this course was given by two further clinical observations. It has been no infrequent experience to note, during routine operative procedures for peptic ulcer, the presence of adhesions suggesting spontaneous sealing of an undiagnosed acute perforation. Further, simple conservative measures have proved surprisingly successful in a number of cases of acute perforation in which the patient's general condition or some concomitant disease contraindicated operative treatment.

Between October, 1947, and October, 1950, therefore, all cases of acute perforated peptic ulcer admitted to my wards were primarily treated conservatively, no attempt being made to select them from the point of view either of time interval between onset and admission or of the length of time after a meal when the perforation occurred. In order, however, to ensure that only true cases of perforation should be included, patients who failed to satisfy the following criteria have been discarded from this series: (1) Those who did not give a classical clinical picture of perforation; (2) those who failed to show the typical picture of gas under the diaphragm on x-ray examination after the institution of

TABLE I.—List of Cases

Case	Age	Sex	Time Interval between Onset and Admission (Hrs.)	Time to Settle (Hrs.)	Chest Complications	Type of Ulcer	Remarks
1	38	M	12	20	No	Duodenal	Laparotomy. Death 48 hours later
2	57	M	6	(48)	Yes	Anterior gastric	
3	35	M	6	36	"	Duodenal	
4	57	M	24	24	"	"	
5	43	M	18	30	No	High lesser curve gastric	
6	42	M	3	22	Yes	Duodenal	Death during expectant treatment. Laparotomy. Recovery
7	68	M	26	36	No	"	
8	37	M	7	24	"	"	
9	52	M	9	32	"	"	
10	61	M	5	(12)	"	Anterior duodenal	
11	36	M	3	(24)	"	Anterior gastric	
12	26	M	3	36	Yes	Duodenal	
13	46	M	5	18	"	"	
14	43	M	7	22	No	"	
15	32	M	2	28	Yes	"	
16	38	M	4	42	"	"	Inoperable at x-ray later
17	65	M	22	30	No	"	
18	48	M	3	16	Yes	"	
19	43	M	5	44	No	"	
20	33	M	4	24	"	"	
21	45	M	9	14	Yes	"	
22	21	M	3	20	No	"	
23	21	M	2	14	Yes	"	
24	68	M	5	32	No	"	
25	55	M	3	28	Yes	"	
26	37	M	9	31	No	"	Death during expectant treatment
27	37	M	8	17	"	"	
28	52	M	4	48	Yes	Carcinoma stomach	
29	30	M	5	18	No	Duodenal	
30	46	M	5	(48)	"	Anterior gastric	
31	29	M	3	14	"	Duodenal	Laparotomy. Death 72 hours later
32	24	M	4	34	"	"	
33	47	M	3	25	Yes	"	
34	36	F	18	34	No	"	
35	72	F	2	(24)	Yes	Anterior gastric	
36	28	M	21	18	No	Duodenal	
37	45	F	4	34	"	"	
38	47	M	7	19	"	Low lesser curve gastric	
39	44	M	6	22	"	"	
40	38	M	3	13	"	Duodenal	

*A paper read at the inaugural meeting of the West Midland Surgical Society at Leicester on January 13, 1951.

Figures in parentheses denote time when laparotomy was done or when death occurred.

treatment; and (3) those on whom subsequent barium-meal examination of the stomach and duodenum failed to show either the scar of a healed ulcer or continuing ulceration. It was felt that only in this way could indefinite clinical conditions such as "acute exacerbation of peptic ulcer" and "subacute leaking perforation," which do not represent accurate clinical entities, be satisfactorily excluded. In all, 40 cases (37 male, 3 female) have satisfied these provisions. Table I gives details of all cases. Table II shows the age distribution and also the time interval before beginning treatment.

TABLE II.—Age Distribution and Time Interval Between Onset and Admission

Age	No.	Time Interval	No.
20-29	6	Under 5 hours	17
30-39	12	5-9 "	16
40-49	12	10-14 "	1
50-59	5	15-19 "	2
60-69	4	20-24 "	3
70 and over ..	1	25-29 "	1

The youngest was aged 21 and the oldest 72.

Treatment

The treatment employed on all these patients was immediate aspiration of any remaining stomach contents on arrival at hospital, followed by continuous Wangenstein gastric suction by means of an intranasal Ryle's tube of modified design, with additional perforations over its lower 10 in. (25 cm.). Intramuscular injections of morphine, 1/6 gr. (11 mg), were given six-hourly and intravenous infusion was instituted. Continuous gastric suction is essential in order that the stomach and duodenum may be kept as empty as possible of secretion and regurgitated intestinal content until effective sealing of the perforation has had every chance to take place, and to counteract any gastric dilatation which might result from air-swallowing. Provided that one has staff who understand the method, it is also much easier from the nursing angle than the less effective procedure of intermittent aspiration. The amount of fluid given intravenously was 3 litres in every 24 hours—a continuous rotation of 5% glucose-saline, plasma, and normal saline. In addition, all cases were treated with injections of penicillin, 500,000 units being given in divided doses each 24 hours.

This treatment was continued until the acute symptoms settled. After a varying time the board-like rigidity of the abdomen disappeared and the muscles gradually relaxed. This took place first on the left side of the abdomen and later in the epigastric region and right iliac fossa. Some residual tenderness and guarding

TABLE III.—Time Taken to Settle Under Expectant Treatment (Excluding Cases Dying or Operated on)

	No.		No.
12-18 hours	9	31-36 hours	5
19-24 "	9	Over 36 "	3
25-30 "	9		35

The shortest time was 13 hours and the longest time 48 hours.

² always remained in the epigastrium and down the right flank to the right iliac fossa for several days, gradually passing off as the peritoneal inflammation resolved. The associated clinical evidences of recovery were a falling pulse rate and the advent of active bowel sounds on abdominal auscultation. Table III shows the varying periods of time required for the acute symptoms to settle. It will be noticed that, out of the 35 successful results of

expectant treatment, 27 cases settled within the first 30 hours and only 3 had failed to respond by the end of 36 hours.

After the improvement in the patient's clinical condition showed that no further leakage was taking place from the perforation, inferring that adequate closure had occurred, he was placed on an early Meulengracht diet, which was rapidly increased to full gastric diet within a week. Ambulation was not allowed until all tenderness in the right iliac fossa had disappeared, which usually took about a week, but progress after this stage was always rapid and the average length of time in hospital was only 10-12 days. It was not considered safe to carry out the subsequent barium-meal examination for at least a further month, so that the patients were discharged home on continued medical regime for this period and attended the out-patient department later for their gastro-intestinal x-ray examination. I have personally witnessed an instance of the secondary perforation of a duodenal ulcer under the x-ray screen as the result of an improvidently early barium-meal examination.

Indications for Operative Intervention

This paper has deliberately been entitled "Expectant treatment of perforated peptic ulcer" because I believe that there is no place in surgery for purely conservative treatment, except possibly in very late cases whose poor general condition precludes any attempt at operation. All cases should invariably be carefully watched and kept constantly under review during the first 48 hours of expectant treatment, with a view to operative intervention should they fail to show obvious clinical improvement. The indications for laparotomy are failure of the abdominal rigidity and tenderness to pass off, and a pulse rate which either does not fall satisfactorily or continues to rise.

Accurate assessment of the clinical condition of these cases is not as difficult as might be imagined and does not seem to be unduly influenced by the morphine administered. Even the ward nursing staff were soon able to decide with considerable accuracy whether a patient was responding effectively to treatment. Actually, most cases which were going to settle satisfactorily showed obvious indications of doing so within the first 12 hours, even if their subsequent improvement was tardy or intermittent.

Results

Table IV gives an analysis of the results. Operative intervention was indicated only as a secondary procedure in three cases.

TABLE IV.—Analysis of Results

	Total No. of Cases	No. Recovered
Duodenal ulcer	32	31
Gastric ulcer:		
Lesser curve	3	3
Anterior	4	1
Gastric carcinoma	1	1
Total	40	36

Case 2.—A man aged 57 had a six-hour history of perforation before admission. He appeared to settle satisfactorily during the first 12 hours of expectant treatment but failed to maintain this improvement, so that laparotomy was carried out at the end of 48 hours. He was found to have a large anterior gastric perforation, which appeared

at first sight to have localized by adhesions between the stomach and the anterior abdominal wall. This localization, however, proved not to be complete, and the resultant local collection of fluid was leaking slowly into the general peritoneal cavity. On separation of the adhesions a perforation measuring nearly 3 in. (7.5 cm.) in diameter was found, and was closed only with considerable difficulty. Death occurred within the next 48 hours from cardio-renal failure, probably secondary to toxæmia from his peritonitis.

Case 11.—A man aged 36 had a three-hour history prior to admission. He failed to improve enough by the end of 24 hours to justify continued expectant treatment. Laparotomy was therefore carried out, and he was found to have a perforation of the anterior surface of the stomach, measuring about 1 in. (2.5 cm.) in diameter, which appeared to have completely sealed by adhesion to the anterior abdominal wall. But in view of the operative findings in the previous case and the fear that secondary leakage might result in this instance also, from later partial separation of the adhesions, it was felt wiser to break them down completely and suture the perforation. This was accordingly done, and subsequent recovery of the patient was uneventful.

Case 35.—A woman aged 72 had a two-hour history. She too failed to improve satisfactorily, and laparotomy was carried out after 24 hours of expectant treatment. She was found to have a large anterior gastric ulcer of acute type with no attempt whatsoever at localization. The perforation was sutured, but she failed to stand the strain of operation, and died 72 hours later from "peripheral failure," after her abdominal condition appeared to have settled satisfactorily.

It will be noted that all three of these patients needing secondary laparotomy were suffering from acute perforations of anterior gastric ulcers, only one of which seemed to have undergone satisfactory spontaneous closure.

Two further deaths occurred during expectant treatment.

Case 10.—A man aged 61 had a five-hour history. In addition to his abdominal catastrophe he was suffering from acute heart failure on admission and died from this cause within the next 12 hours. His general condition was such as to have contraindicated any attempt at operative surgery during the whole of this period, even had this trial of expectant measures not been in progress. Post-mortem examination showed a large acute perforation of the anterior wall of the first part of the duodenum, which appeared to have sealed satisfactorily by adhesion to the under surface of the liver. Had he lived, therefore, he might well have been a successful case of spontaneous closure.

Case 30.—A man aged 46 had a five-hour history. He was acutely shocked on admission to hospital despite his short history, and failed to recover sufficiently during the subsequent 48 hours' expectant treatment for laparotomy to be attempted. His blood pressure never rose above 70/30, and he finally died from what appeared to be primary peritoneal shock. Post-mortem examination revealed a very large anterior gastric ulcer with no attempt at localization.

Altogether, therefore, the mortality in this series of 40 cases was 10%, and the failure rate under expectant treatment was 12.5%. It is interesting that all except one of the cases of perforated duodenal ulcer recovered under expectant treatment, and that this solitary exception might also have been a successful cure had he not died from intercurrent disease. Another fact which stands out clearly is that the site of perforation in gastric ulcer has a definite bearing on the results obtained by conservative measures. It will be noticed that all three patients with lesser-curvature ulcer recovered, but that only one of the four cases of anterior perforation lived, and this only after secondary operative closure.

The single case of recovery from gastric carcinoma was probably an unusually fortunate result. Subsequent

barium-meal examination a month after his discharge showed a large carcinoma which must have been inoperable at the time of perforation. This patient died within a month of readmission to hospital, and post-mortem examination showed a primary ulcerative type of gastric carcinoma on the anterior wall of the stomach. This had apparently perforated and sealed successfully, although the degree of malignant infiltration of the anterior abdominal wall made reconstruction of the previous acute pathology difficult.

Conclusions

It appears justifiable from this series of cases to draw the conclusion that the expectant method of treatment in acute perforated peptic ulcer does not give results as good as those obtained by operative surgery. Before the advent of antibiotics and other forms of chemotherapy most large series of operative results gave a mortality varying between 15 and 18%, but the figures which are available since the last war show that the inclusive mortality of surgical methods of treatment is now less than 8%. In comparison with the latter figure, the mortality of 10% in this series is not satisfactory. Were it possible to be sure of the actual site of perforation, and to treat only the duodenal and lesser-curvature ulcers by expectant measures, the method might be justifiable. But unfortunately this is not generally feasible, and the mortality rate in the expectant treatment of anterior gastric ulcers in this series has been extremely high.

As the result of my experiences, therefore, I have entirely given up the expectant method of treatment. What is now done is to carry out laparotomy in every case, as soon after admission as possible. If the perforation is found to have sealed adequately, either by adhesion to the liver or by means of an omental plug, the abdomen is closed again with no further surgical intervention and conservative treatment is then instituted. Actual operative closure of the perforation is normally reserved for cases which do not appear to be sealing spontaneously, though it is carried out routinely, in every instance of anterior gastric ulcer.

Finally, it is interesting to note the high incidence of chest complications during expectant treatment. Of the 36 patients who lived, no fewer than 13 developed either localized pneumonitis or basal collapse. In 10 cases this was right-sided, in two it was on the left, and in one case it was bilateral. This is approximately the same incidence of pulmonary lesions as that following operative treatment of perforated peptic ulcer, and it appears to support the anaesthetists' contention that this complication is not primarily the result of their ministrations. It was indeed fortunate that, despite this high incidence of minor chest complications, all of them settled satisfactorily under routine chest treatment and chemotherapy, and that there was no case of subphrenic abscess or other secondary complication of such nature in this series.

Summary

The results are presented of treating expectantly 40 unselected cases of acute perforated peptic ulcer.

The cases were treated by immediate aspiration of the stomach contents, continuous gastric suction, repeated injections of morphine, and intravenous infusion. The patients were kept under constant review during the first 48 hours of expectant treatment, and laparotomy was performed if there was no obvious clinical improvement.

In this series the mortality rate was 10%, and the failure rate with expectant treatment was 12.5%. Thirty-one cases out of 32 with perforated duodenal ulcers recovered under expectant treatment, but only one out of four cases with anterior perforations of the stomach survived. There was a high incidence of chest complications during treatment.

The expectant method of treatment has now been abandoned in favour of early laparotomy in every case.

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SIGNIFICANCE OF RAISED SERUM- ENZYME LEVELS IN PERFORATED PEPTIC ULCER

BY

H. WAPSHAW, M.D., F.R.C.S.Ed., F.R.F.P.S.

Assistant Surgeon, Western Infirmary, Glasgow

Aside from renal considerations, a rise in the amylase content of the blood or urine is generally regarded as an indication of some form of damage to the exocrine system of the pancreas. The gross and the microscopical pathological changes may conform to one of the recognized types of acute pancreatitis or may be so slight as to be imperceptible: instances of the latter have been recorded in which presumably the duct of Wirsung has been affected by the presence of gallstones or of some other agency such as a T-tube left in the common duct for drainage (Millbourn, 1936; Lewison, 1941). In others, a chronic duodenal ulcer burrowing into the pancreas is the disturbing factor (Probstein, Wheeler, and Gray, 1939).

There is experimental evidence to show that the amylase, after its escape from damaged acini or ducts, finds its way into the peripheral blood mainly via the portal system: lymphatic absorption plays a secondary part (Popper and Necheles, 1940; Howard, Smith, and Peters, 1949). It would now appear that a free perforation of a gastric or duodenal ulcer has some claim to be regarded as one of the conditions likely to cause the blood or urine amylase levels to rise. Mikkelsen (1934) cited instances from his own experience in which this complication was associated with hyperdiastasia, but gave no details of the incidence or significance of this finding.

The first large-scale study of the problem was carried out by Hughes (1942). He investigated 40 patients with perforation of a gastric or duodenal ulcer and found four with a urinary diastatic index of the order of 300. I studied the concurrent amylase and lipase changes in the blood in a series of 30 perforated cases, in none of which was the pancreas involved by ulcer (Wapshaw, 1949). Slight deviations from the normal standards were noted in four and substantially raised values in two cases. High though the latter were, they fell short of the values obtained in two cases of acute pancreatitis which were comparable in respect of the duration of illness. Musgrove (1950) has recently added three more to the list, apparently the first to be reported from the Mayo Clinic. One was a perforated reticulum-cell sarcoma; the other two were ordinary perforated peptic ulcers.

The aetiological significance of the increases in enzyme content of the blood or urine is still uncertain. Hughes suggested that they might be due to (1) an overlooked patch of acute pancreatitis; (2) a second peptic ulcer lying posteriorly and adherent to the pancreas; or (3) transperitoneal absorption of the pancreatic ferments contained in the gastric or intestinal contents which had passed into the abdominal cavity. Neither of the first two probabilities was confirmed in the cases which were examined post mortem.

A group of workers at the Mayo Clinic—Pemberton, Grindlay, and Bollman (1950)—stimulated by Musgrove's findings, carried out what seems to be the first experimental attempt to trace the source of the surplus amylase. Not only did they duplicate in dogs the conditions in humans, but they were also able to define the perforations both in time and in location. A decided rise in the serum amylase was observed in animals which suffered perforation within one to two hours after a full meal—that is to say, when the pancreas was secreting to full capacity. If the perforation was delayed for three to four hours the amylase levels in the blood were unaffected. In samples of the peritoneal fluid withdrawn by abdominal paracentesis the amylase concentration of the early group was about six times that of the later. The mortality from peritonitis was very high, particularly among animals belonging to the former group, but, according to the control experiments, peritonitis cannot be held responsible for the hyper-enzymaemia. Pemberton *et al.* concluded that the determining factor was the amylase concentration of the free fluid in the abdomen.

In the course of previous clinical study I independently accumulated data bearing on points studied by the Mayo Clinic team, such as the times of the previous meal and the enzyme activity of the fluid inside the abdominal cavity encountered at operation. It seems of value to place these findings on record. The results are further amplified by the inclusion of repeat serum-enzyme observations which were made in most of the patients 24 hours after the initial readings.

Findings in 32 Perforation Cases

Pathological Data

It must be emphasized again that in none of the cases had the ulcer process implicated the pancreas. The site-incidence of the perforations was as follows: pyloro-duodenal, 29; gastric, 2; anastomotic, 1. Simple closure was carried out on 28 patients, with one death. Two patients were admitted to hospital with widespread peritonitis and died soon after (Cases 1 and 27). There were also two late cases which had developed sub-hepatic abscesses when first seen; both made a good recovery following open drainage (Cases 31 and 32).

In Case 27, at necropsy, a second ulcer was found lying posteriorly and loosely adherent to the pancreas. A few spots of fat necrosis were also observed in the fatty tissue of the lesser and greater omenta of this case. Yet the parenchyma of the gland appeared normal on section, and certainly the enzyme findings in the blood gave no reason for one to think otherwise.

Enzyme Findings in the Blood

Amylase and lipase estimations were carried out with the same sample of blood. In the former, the Somogyi iodometric test was employed. The normal values range between 70 and 200 units per ml. of serum. For