

## ACUTE DISTENSION OF AFFERENT LOOP AFTER POLYA GASTRECTOMY

BY

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Acute distension of the afferent loop is a rare complication of Polya gastrectomy which has always been attributed to an organic obstruction at the stoma. In some cases the nature of the obstruction has been clearly demonstrated. Thus, acute angulation at the stoma may arise from displacement of the afferent loop either behind the efferent loop (Quinn and Gifford, 1950) or in front of it (West, 1953), or from volvulus of the anastomosis (Hoyte *et al.*, 1957).

In the few reported cases which have arisen in the early post-operative period the cause of the distension has not been so convincingly shown. Wangenstein (1941) described a case in which a patient died on the second day after operation, and in which no causal obstruction was found at necropsy, although he later suspected that there may have been kinking at the anastomosis from a slight forward rotation of the afferent loop. Henson (1955) reported a case which developed on the sixth day after a Polya-Hofmeister-Finsterer operation and wrote: "The obstruction appeared to be at the valve and due to occlusion of the jejunum from without by the closed portion of the cut end of the gastric remnant." Simon (1956) also described a case, developing on the eighth day, which he attributed to angulation of the afferent loop at the stoma. Two further cases which arose even earlier after operation are reported for two reasons: there was no definite organic obstruction at the stoma, and the clinical features differed from those of cases previously reported.

### Case 1

A frail man of 50 with inactive tuberculosis of the lumbar spine and right hip-joint underwent a short-loop Polya antecolic isoperistaltic (afferent loop to lesser curvature) partial gastrectomy, with full-length anastomosis, for chronic duodenal ulceration with pyloric stenosis. On the first day after operation he took 570 ml. orally and 608 ml. was aspirated from a Ryle tube left in the gastric remnant. On the second morning 300 ml. was taken and 100 ml. aspirated; bowel sounds were present, and the nasal tube was withdrawn; he passed a loose motion the same evening. His pulse rate, however, had by then gradually risen from 88 immediately after operation to 120. On the third morning he deteriorated abruptly, the radial pulse becoming imperceptible and blood pressure unrecordable. He was pale and rational, and complained only of extreme exhaustion. There was no pain or vomiting and the abdomen was neither rigid nor distended. The plasma non-protein nitrogen had risen from 38 mg./100 ml. pre-operatively to 165 mg. (normal range 20-40 mg./100 ml.); the plasma sodium had fallen from 136 to 125 mEq./l. (normal range  $138 \pm 5$ ). There was therefore evidence of considerable dehydration and sodium depletion. The plasma amylase was 320 Somogyi units/ml. (normal range 0-160) and the electrocardiogram was normal.

In the absence of any physical signs a provisional diagnosis was made of leakage from the duodenal closure. A nasal tube was passed and continuous suction applied. Plasma and oxytetracycline were given intravenously, and streptomycin and chloramphenicol intramuscularly. Two hours later slight abdominal distension was evident and x-

ray examination showed moderate distension of many loops of small bowel and also subphrenic gas, the latter being of doubtful significance in view of the recent laparotomy. After receiving 3,600 ml. of plasma in 5½ hours he improved, the pulse rate falling to 100 and blood pressure rising to 130/110 mm. Hg. Twelve hours after his initial collapse he deteriorated again, his blood pressure falling to 70/40 mm. Hg and pulse rate rising to 116. The abdominal distension had increased considerably.

Under general anaesthesia the abdomen was reopened. There was a small amount of clear free fluid in the abdomen and no evidence of leakage from the duodenal stump or the anastomosis. The gastric remnant appeared normal, but there was gross distension of a normally placed viable afferent loop; its mesentery was thick with oedema. No obstruction to this loop at the stoma was evident from without, but, presumably from its downward drag, there was tilting of the anastomosis, with consequent angulation, and perhaps obstruction, of the efferent loop. Unhappily, the Ryle tube was in the oesophagus. A stomach tube passed orally was guided by the surgeon into the afferent loop and 700 ml. of turbid fluid was aspirated. A Ryle tube was left in this loop and a jejunostomy was made in the efferent loop. He survived operation by about 15 minutes.

*Necropsy* (Dr. M. E. H. Halford).—There was no peritonitis and the gastric remnant was normal. The duodenal closure was intact, but the whole duodenum was dilated, as was the proximal loop of the anastomosis, which was viable and normal in position. The remainder of the small intestine was moderately distended. From within, the anastomosis showed an unexpected degree of oedema. Tuberculosis was found in the right kidney and both epididymides. The left kidney was enlarged and otherwise normal. The lungs showed scarring but no active tuberculosis. The brain was normal.

*Cause of Death*.—Dehydration and sodium depletion may well have contributed to the shock, but rapid correction of the blood chemistry led only to transient improvement; hence distension alone was almost certainly the major factor. This mechanism has been demonstrated experimentally by Taylor *et al.* (1933), who showed, in the dog, that distension of the proximal intestine to 100 mm. Hg without obstruction led to prostration within a few hours and death, usually within 24 hours, in the absence of a significant fall in the plasma chloride level or interference with the blood supply of the gut. In contrast, denervation of the loop before distension was associated with no ill-effects for the first 48 hours, the animal becoming ill only with the development of peritonitis due to the experimental method.

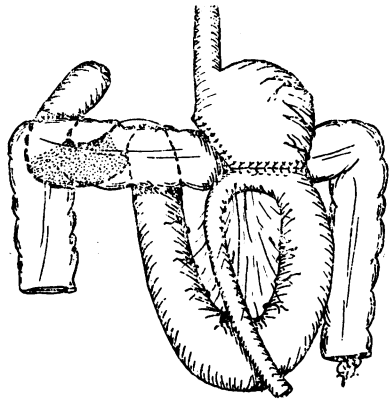
### Case 2

A robust man of 36 underwent partial gastrectomy of similar type to that in Case 1, but with a half-length stoma, also for chronic duodenal ulceration with pyloric stenosis. Preliminary investigations included normal chest x-ray examination and normal estimations of his haemoglobin, blood urea, serum proteins, and serum chlorides. He was well until the second post-operative day, when his pulse rate rose from 84 to 104, and the abdomen became slightly distended. His intake by mouth was 300 ml. of water, and 350 ml. was aspirated through his Ryle tube. On the third day he became anxious and restless; the distension increased, although occasional high-pitched bowel sounds were heard; his oral intake was 375 ml. and 510 ml. was aspirated. On the fifth day the Ryle tube was removed, as only 29 ml. had been aspirated in the previous 12 hours, against an intake of 660 ml. He passed a loose motion, but remained distended and very restless. On the seventh day a light diet was started and three loose motions were passed.

On the eighth day he deteriorated rapidly after complaining of diffuse abdominal pain for the first time. His pulse rate rose again from 86 to 100, and his systolic blood pressure fell to 60 mm. Hg. There was no vomiting, but a Ryle tube was passed and 126 ml. withdrawn. His serum

amylase was 400 Somogyi units/ml. A diagnosis of diffuse peritonitis was made, penicillin and streptomycin were given intramuscularly, and under general anaesthesia the lower part of the abdominal incision was opened. The operation was limited to the insertion of a finger into the peritoneal cavity, and about 600 ml. of bile-stained fluid was released which yielded *Proteus vulgaris* and *Escherichia coli* on culture. Deterioration continued and he died 18 hours later.

**Necropsy (Dr. J. Guy).**—The abdomen contained a large volume of bile-stained pus. Loops of distended small intestine were bound together with purulent fibrinous exudate. The whole duodenum and afferent loop were grossly dilated.



Post-mortem findings in Case 2. The duodenum and afferent loop were greatly distended. The stippling shows the zone of duodenal infarction.

internal examination. The gastric remnant was not distended and the stoma was widely patent. The pancreas was normal. The cranial cavity was not examined.

**Cause of Death.**—The duodenal infarction may be explained by the work of Dragstedt *et al.* (1929), who showed that the duodenum in the dog is more vulnerable to infarction from raised intraluminal pressure than any other part of the intestine. Eisberg (1925) showed that in the small intestine of the dog the vasa recta pierce the muscularis shortly after leaving the mesentery, while in the large intestine they pierce the muscularis nearer the anti-mesenteric border. Dragstedt *et al.* (1929) confirmed this work and showed that the vessels pierce the muscularis in the duodenum even earlier than in the jejunum. They accordingly suggest that the longer the vessels remain superficial in the wall of the bowel the better is it able to withstand distension. Similar considerations may apply in man.

#### Aetiology

The absence of a mechanical displacement of the afferent loop with consequent obstruction at the stoma has been emphasized in both cases. Both operations were completed without technical doubt or difficulty and with minimal "turn-in" of bowel wall. Oedema at the suture line may have caused some degree of obstruction in the first patient, who died on the third day, but in Case 2 there was no evidence of obstruction at the stoma, although the distension was sufficient to cause duodenal infarction. There may, however, have been marked initial oedema which provoked the distension and then subsided. But atony alone from denervation must be considered because some vagal fibres are always divided on section both of the stomach and of the duodenum. Indeed, to this Roux *et al.* (1950) attributed, with due anatomical evidence, chronic distension of the afferent loop and bilious vomiting.

A further feature of both cases was generalized ileus: in Case 1 it was moderate in degree and did not become evident until several hours after the onset of shock, which is believed to have arisen reflexly from distension of the afferent loop. Perhaps the ileus had a similar reflex origin, either by an entirely nervous route or through the medium

The third part of the duodenum, where its distension was maximal, showed infarction, which ceased abruptly in the middle of the fourth part (see Fig.). The retro-duodenal vessels were thrombosed. The duodenal closure and the anastomosis were intact, and no mechanical ob-

struction of the afferent or efferent loops was evident on external and

of an irritant peritoneal exudate. In Case 2 the ileus was severe from the second day, although, untreated with antibiotics, he appeared too well for any serious suspicion of peritonitis, nor was any source for an early peritonitis found at necropsy after his sudden deterioration and death with duodenal infarction on the ninth day. Perhaps it had a common origin with the afferent-loop distension from a more extensive division of the vagus, for paralytic ileus is an accepted complication of formal vagotomy.

Reference must also be made to the use of clamps. Burge (1956) attributes paresis of the jejunal loop, and consequent failure of the gastric stump to empty, to inclusion of its mesentery in a clamp. W. M. Capper (1957, personal communication), with similar suspicions, avoids altogether the use of clamps at gastrectomy. In Case 1 clamps were not used; in Case 2 a light Carwardine clamp was placed across the gastric remnant and a similar clamp may have been used on the jejunum, possibly including its mesentery. Atony can no more be attributed to a single cause in the afferent loop than in the gastric remnant, where it is common. An adverse factor in the afferent loop must be the slow accumulation of bile and pancreatic juice, although the volumes secreted immediately after partial gastrectomy are less than the normal total of 1,500 ml. a day (T. J. Butler, 1957, personal communication). Perhaps gradually increasing distension from this cause prevents spontaneous recovery from an atony which would otherwise have been transient.

#### Clinical Features

Henson's (1955) patient, a woman of 35, was well until the sixth post-operative day, when she developed severe abdominal colic without vomiting. Anxiety developed, there was a raised pulse rate, and a tender resonant swelling was present in the left upper quadrant. X-ray examination confirmed this to be a distended loop of small intestine. Simon's (1956) patient, a man of 57, developed abdominal pain, vomiting, and distension on the eighth day. A tender mass was felt in the right upper abdomen, but x-ray examination showed no abnormality. In both these cases the diagnosis was suggested by the symptoms and signs, and surgical treatment was effective. In the present two cases the clinical clues were minimal, for there was neither pain nor vomiting. In Case 1 the only symptom was exhaustion, and there were no abdominal signs until diffuse distension developed a few hours after the onset of severe shock. In Case 2 restlessness, abdominal distension, and slight diarrhoea preceded collapse on the eighth day.

The case for a reflex origin of these symptoms and signs from a distended afferent loop appears to be strengthened by the observations of Henson (1955), who examined the records of eight cases of duodenal leakage and found that, with the exception of one case of early rupture, all the patients showed slight but definite signs of "some preliminary disorder," which he thought was possibly related to obstruction of the afferent loop before actual leakage began. The absence of hyperpyrexia is also of interest. Wangenstein's patient died with hyperpyrexia two days after Polya gastrectomy for carcinoma, and at necropsy dilatation of the proximal loop and the duodenum was found. He accordingly obstructed the duodenum in the dog by severing the pylorus and occluded the jejunum a short distance beyond the duodeno-jejunal flexure. Death with hyperpyrexia was a uniform finding. In man, however, hyperpyrexia has not been described even in cases in which afferent-loop obstruction was clearly of the closed-loop type.

#### Diagnosis

Even in retrospect accurate diagnosis was difficult in the cases described, with their vague symptoms, limited signs, and negative investigations. It is not possible to define clinical distinctions between afferent-loop ileus and other complications such as peritonitis from duodenal leakage, acute pancreatitis, and acute intestinal obstruction of

mechanical origin. Indeed, attempts at a more accurate diagnosis, if they lead to delay, are misplaced, as urgent surgical relief is required in all these conditions except acute pancreatitis—and even this, after Polya gastrectomy, needs confirmation by laparotomy, because marked elevation of the serum amylase is not diagnostic. High levels may occur with duodenal leakage and with closed obstruction of the afferent loop.

#### Treatment

Urgent evacuation of the distended afferent loop is essential. The passage of a nasal tube might possibly achieve it, but, failing this, operation is indicated after shock has been treated with appropriate vigour. In Case 1 there was much improvement after about six hours, but operation was unfortunately deferred until further deterioration had occurred. A stomach tube was then passed by the anaesthetist and guided by the surgeon into the afferent loop, which was emptied. A nasal tube was subsequently left in the afferent limb and an enterostomy was made in the efferent limb. An alternative would have been an entero-anastomosis between the two loops, a procedure still recommended as a routine prophylactic measure by Simon (1956). Although such a stoma must divert the alkaline pancreatic juice and bile to greater or less degree, he doubts on theoretical and practical grounds whether this predisposes to stomal ulceration. While this view may not be generally acceptable, routine nasal aspiration of the afferent loop after Polya gastrectomy can scarcely be criticized.

C. F. W. Illingworth (1957, personal communication) leaves a 7-mm. nasal tube in the afferent limb in two types of case: where the closure of the duodenal stump seems insecure and where there is reason to fear temporary obstruction at the anastomosis; for example, after a difficult gastrectomy for stomal ulcer. The wisdom of this practice is evident, but duodenal leakage by no means always follows a difficult closure, nor was afferent-loop distension in the two cases described associated with any technical doubts or difficulty. There is then a case for routine aspiration of the afferent loop until emptying is proved, although it may not prevent these complications absolutely. Wangenstein (1941) described a forked catheter, one limb of which was left in each limb of the anastomosis. He used it routinely and considered aspiration of the afferent loop the more important. The need for the surgeon to number afferent-loop distension among the early complications of Polya gastrectomy is indisputable. It may be necessary to reopen the abdomen urgently on slender evidence, but, once the decision is made, treatment is simple and success would seem almost certain.

#### Summary

Two fatal cases of acute distension of the afferent loop after Polya gastrectomy are reported.

The course in Case 1 suggests that severe shock can develop from the distension alone and shows that there may be no early physical signs whatsoever on examination of the abdomen.

The essential clinical feature in Case 2 was persistent abdominal distension in a patient who remained in good general condition until the onset, on the eighth post-operative day, of diffuse peritonitis from gangrene from the third part of the duodenum.

In both cases the absence is stressed of an evident mechanical factor to account for the distension: atony is suspected, and may have been due to an unusually extensive incidental division of vagal fibres.

Acute afferent-loop distension may be indistinguishable clinically from the other early abdominal complications of Polya gastrectomy. The treatment is rapid resuscitation, when necessary, and urgent surgical relief of the distension.

Acute afferent-loop distension is commonly suspected to be a cause of leakage from the duodenal stump, and the possibility of avoiding both these complications by routine post-operative aspiration of the afferent loop is discussed.

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## FAECAL OCCULT BLOOD TESTS WITHOUT DIETARY RESTRICTIONS

BY

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The most widely used tests for occult blood in faeces involve the use of benzidine. This substance is now difficult to obtain because its carcinogenic properties make its manufacture dangerous, and it seems likely that it will soon be completely unobtainable. It will therefore be necessary to use methods for detecting faecal occult blood which do not involve the use of benzidine. The orthotolidine method of Kohn and O'Kelly (1955) appears to be a suitable alternative, but when this method is used the patients must be put on a meat-free diet if false-positive results are to be avoided. This is not always convenient; special diets have to be ordered (and adhered to) and there is a delay of four days before the result is known. As Kohn and O'Kelly point out, it is quite easy to alter the sensitivity of the test, and we have tried to find a level of sensitivity such that negative results are obtained from patients who are on a normal diet and who are not bleeding into the gastro-intestinal tract.

#### Methods

The ages of the 49 hospital in-patients selected (27 males and 22 females) ranged from 16 to 85 years. They were on a normal hospital diet (excluding liver) and had no signs or symptoms of gastro-intestinal or other disease which might cause bleeding into the alimentary tract. Three specimens of faeces were collected from each patient, on consecutive days where possible. Each specimen was tested for occult blood by the test-tube method of Kohn and O'Kelly (1955), using five different levels of sensitivity.

*Details of Method.*—A piece of faeces about the size of a pea was mixed with 5 ml. of water, and the resulting suspension was boiled and cooled. A stock solution was prepared containing 4% orthotolidine "AnalaR" in absolute industrial alcohol. Five dilutions of this stock solution with 50% v/v acetic acid were made—namely, 33%, 20%, 10%, 5%, and 2%. These solutions were stored in the refrigerator and renewed monthly. Four drops of each dilution were treated with one drop of 20-volume hydrogen peroxide, and left to stand for one minute. If no blue or green colour developed, a drop of the faecal suspension was added.