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Diseases of the operated stomach

Textbook for students of 6 courses

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This tutorial is devoted to endoscopic and interventional surgery. The authors present modern endoscopic, radiosurgical, radiosurgical methods of research and gave examples of diseases in which they are used.

The textbook " Diseases of the operated stomach" is developed on the discipline "Hospital surgery" in accordance with the requirements of the FSEI HPE, is intended for students of medical Universities and faculties, trained in the specialty 31.05.01 General Medicine.

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Minor postprandial complaints are commonly experienced by patients after gastric operations.

Sequelae of gastric surgery

Recurrence of the disease

Nutritional and functional sequelae
- Weight loss, anaemia: iron deficiency and B_{12} deficiency
- Milk intolerance
- Bone disease
- Dumping syndromes
- Reactive hypoglycaemia
- Bile vomiting
- Diarrhoea
- Small stomach syndrome

Mechanical complications
- Afferent/efferent loop obstruction
- Jejunogastric intussusceptions
- Gastro-oesophageal reflux

Others
- Cholelithiasis
- Bezoar formation
- Gastric carcinoma

These usually improve with time and dietary adjustments. In a cohort of patients, however, variously estimated at 5–20%, the symptoms are severe, persistent and cause considerable disability, often drastically reducing the quality of life, and malnutrition. The problem may affect the quality of life so adversely that the patient commits suicide. This regrettable outcome was encountered in two patients in a series of 321 patients with postgastric surgery problems referred to the author for remedial treatment/surgery over a period of 30 years. It seems likely from recent reports that similar problems are being encountered in patients after gastric bypass, pancreatic biliary bypass and duodenal switch operations for morbid obesity. The various postgastric surgery syndromes arise on a background of altered anatomy and physiology of the upper gastrointestinal tract, although the exact mechanisms responsible for some of the severe symptoms remain unclear.

In the author's personal series, no particular personality type is at increased risk, but disabling symptoms after gastric surgery are more commonly encountered in the following:

- female sex
- operations for peptic ulceration in the young (below 30 years)
- extensive gastrectomy with duodenal diversion (Pylya). Severe and persistent symptoms are rarely encountered after HSV if one excludes symptoms caused by recurrence of the ulcer. However, they occur with similar frequency to that reported after gastrectomy in patients who undergo truncal vagotomy with drainage or truncal vagotomy and antrectomy. The type of drainage procedure (pyloroplasty or gastrojejunostomy) does not affect the incidence of postprandial symptoms and other sequelae.

Nutritional consequences of gastric surgery

These consist of weight loss, anaemia and bone disease.

Weight Loss

Loss of weight or failure to gain weight is very common after gastric surgery and tends to be more marked after extensive gastrectomy, particularly of the Pylya type. Often there is early satiety due to loss of adaptive relaxation and disturbances of ghrelin release. In practice, significant weight loss is usually encountered in patients who obtain a bad functional result and who experience severe postcibal symptoms such that they are afraid to eat. The resulting diminished calorie and protein intake is the major factor, although malabsorption of fat and nitrogen and decreased small bowel transit time may be

operative, at least in some patients. Although mild steatorrhoea is common, severe fat malabsorption is rare unless there is a coexisting subclinical small bowel disease (e.g. gluten enteropathy) or gross bacterial overgrowth.

Anaemia

Iron-deficiency anaemia

Microcytic hypochromic anaemia is very common after vagotomy and drainage and gastric resections, especially in females. The incidence of this complication increases with time and approximates to 60% and 80% at 10–20 years in males and females respectively. The exact pathogenesis of the iron-deficiency anaemia is unclear but is probably multifactorial. The mechanisms thought to be important

include:

• shift to trivalent ferric iron at high pH followed by polymerization

• loss of a gastric juice factor which normally facilitates the absorption

of iron

• diminished splitting of iron–protein complexes by the reduced peptic

activity of the gastric juice

• enhanced binding of dietary iron to specific proteins (e.g. gastroferrin).

In view of the high incidence of iron-deficiency anaemia after gastric surgery, prophylactic treatment with oral iron (300mg q.d.s.) is nowadays recommended in all patients after gastrectomy and truncal vagotomy with drainage or antrectomy. This amount of daily iron supplementation allows sufficient absorption to restore serum iron levels to normal.

Macrocytic anaemia

This is the result of vitamin B12 deficiency. Malabsorption of this vitamin is invariable after total gastrectomy due to the loss of IF. However, megaloblastic anaemia takes several years to develop due to the large body stores of vitamin B12. These patients have an abnormal Schilling test and require 3B€ monthly injections of cyanocobalamin indefinitely. Subclinical deficiency of this vitamin is also encountered in some patients after partial gastrectomy and vagotomy with drainage or antrectomy, although frank megaloblastic anaemia is rare in these groups. The main factor responsible for the impaired absorption of dietary vitamin B12 in patients after partial gastrectomy and truncal vagotomy is the

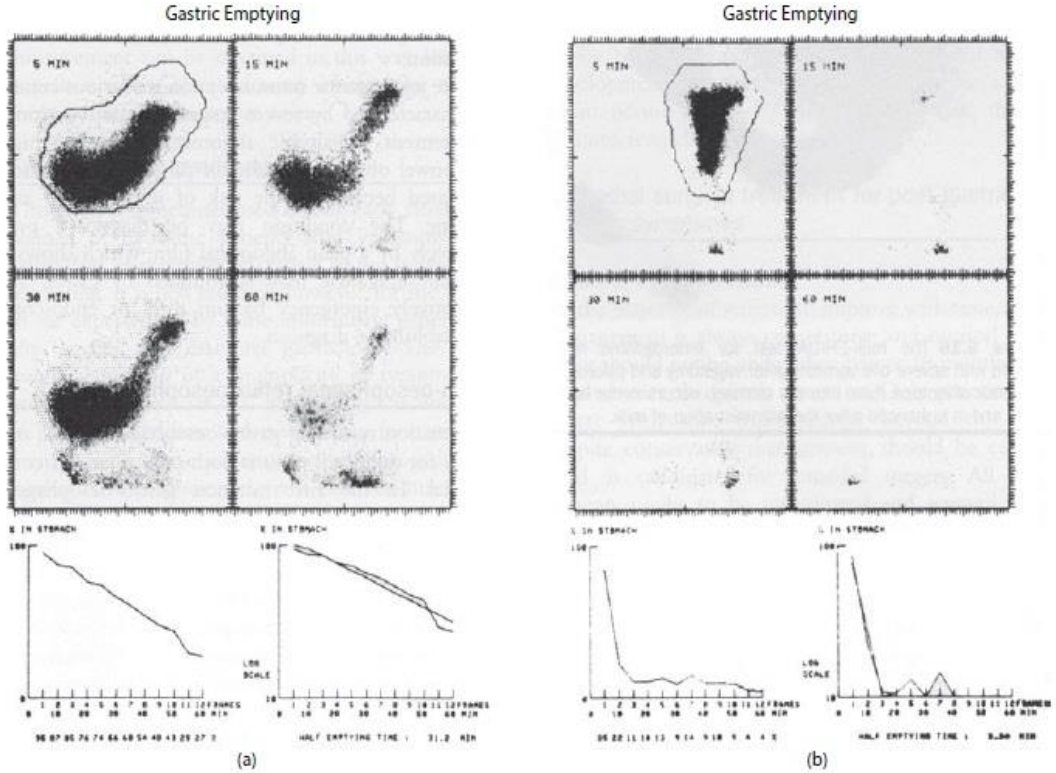
lack of acid environment which normally facilitates the release of vitamin B12 bound to ingested food. The reduced secretion of IF reported in some patients is considered to be less important in this group of patients in whom the Schilling test is normal. Treatment is with oral crystalline vitamin B12, which is administered between meals. Malabsorption of vitamin B12 may also be the consequence of bacterial overgrowth and steatorrhoea. Folate deficiency is rare and is only encountered in patients after extensive or total gastrectomy. It results from an inadequate dietary intake,

Bone disease

This complication develops several years after gastric resection with duodenal exclusion (Pylya) as the duodenum is the major site of calcium absorption. The majority of patients are females who classically develop osteomalacia 10–20 years after the gastrectomy. In fact, in the vast majority of cases features of both osteomalacia (demineralization of bone) and osteoporosis (loss of bone substance) are present. The biochemical features (raised alkaline phosphatase and serum calcium) usually predate the clinical symptoms by several years. The clinical features of postgastrectomy bone disease include generalized bone pains, weakness due to an associated myopathy and the development of stress fractures most commonly in the vertebral column. Treatment should be prophylactic with oral calcium and vitamin supplements. The established condition may be treated with bisphosphonates.

The dumping syndrome

Considerable confusion has been generated by the inclusion of patients with reactive hypoglycaemia in the group and referred to as



Gastric emptying of an isotope-labelled meal: (a) normal single exponential emptying; (b) rapid initial gastric emptying in a patient with severe dumping symptoms.

'late dumping' to differentiate this condition from patients with vasomotor symptoms which occur soon after eating and in this erroneous classification are designated as 'early dumpers'. There is now general agreement that patients with symptoms due to reactive hypoglycaemia which occur 2–3 hours after a meal should not be included in the dumping syndrome. Although the term 'dumping' was introduced by Mix in 1922, the first description of the symptoms of vasomotor dumping syndrome had been reported previously by Hertz in 1913. The syndrome, which is one of the commonest sequelae of gastric surgery, consists of postprandial vasomotor (systemic) and gastrointestinal symptoms. The dumping syndrome is associated with rapid gastric emptying, although some have postulated that the enterogastric reflux of bile is responsible for some of the symptoms. The fact is that both enterogastric reflux of bile and vasomotor dumping may occur in the same patient, and other than sharing the same aetiology (gastric surgery) there is no evidence that one syndrome causes the other. The vasomotor symptoms and signs (palpitations, vasodilatation, hypotension and fainting/having to lie down, etc.) occur within minutes of eating and are principally caused by hypovolaemia, which is accompanied by diminished cardiac output and reduced peripheral resistance. The attacks are typically precipitated by high-carbohydrate meals. The hypovolaemia is secondary to a massive outpouring of fluid from the vascular compartment into the small bowel lumen as a consequence of the hyperosmolar nature of the intestinal contents resulting from the precipitous gastric emptying. Several vasoactive peptides have been held responsible as mediators of the vascular and gastrointestinal manifestations of the dumping syndrome. These include kinins, substanceB€P, enteroglucagon, gastric inhibitory polypeptide and neurotensin. The gastrointestinal symptoms, which include diarrhoea, occur later during the course of a dumping attack and may be absent. Patients with mild to moderate dumping symptoms are managed satisfactorily with dietary manipulations. These patients are advised to eat small, dry meals (no fluids before or during meals) rich in protein and fat but low in carbohydrates. Additives that slow gastric emptying, such as methoxy-pectin or bran, are beneficial. However, remedial gastric surgery is required for patients with severe and persistent dumping (see below).

Reactive hypoglycaemia

This complication is relatively uncommon and has a reported incidence of 1–6% after gastric surgery. Reactive hypoglycaemia often exists with other symptoms, including

vasomotor dumping and diarrhoea. The symptoms, which occur 2–3 hours after a meal, are due to hypoglycaemia and include sweating, tremor, difficulty in concentration and, rarely, fainting. The diagnosis is best confirmed by an extended oral glucose tolerance test, which demonstrates an initial hyperglycaemia. This triggers an exaggerated insulin release with elevated plasma insulin and enteroglucagon that precede the hypoglycaemia. Reactive hypoglycaemia usually responds to dietary measures, including small low-carbohydrate, high-protein meals and very rarely requires remedial surgical treatment. Bile vomiting Vomiting of bile or bile-stained fluid before or after meals may be a manifestation of the following disorders:

- recurrent ulceration
- enterogastric reflux
- intermittent obstruction of the afferent or efferent loop of a gastrojejunostomy
- cardio-oesophageal incompetence.

Enterogastric reflux/reflux gastritis

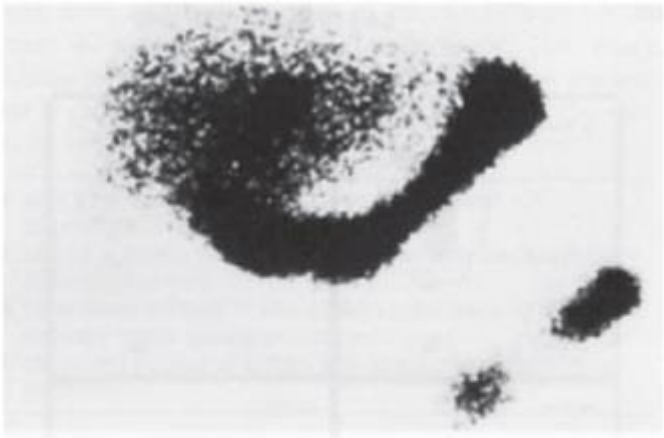
Reflux of upper intestinal secretions (bile/pancreatic juice/ succus entericus) into the stomach causes a reflux erosive gastritis and bile vomiting. The symptoms include epigastric pain, nausea and vomiting in the early postprandial period. The pain is usually of a burning nature, is aggravated by food and is not relieved by antacids. The attack usually culminates in the vomiting of bile-stained fluid 1–2 hours after a meal. Less commonly, the vomiting occurs in the early morning andB€ is preceded by nocturnal burning pain. The erosive gastritis leads to chronic blood loss with the development of an iron-deficiency anaemia and, occasionally, to overt acute gastric haemorrhage. The diagnosis is established by upper gastrointestinal endoscopy, which shows a diffuse gastritis with oedematous friable mucosa and superficial erosions, in addition to pooling of bile-stained fluid. Quantification of the enterogastric reflux is obtained by the modified EHIDA test. In this investigation, EHIDA is injected intravenously and is followed by external scintiscanning of the upper abdomen with a gamma camera. When the gallbladder is imaged by the isotope, contraction and emptying of the organ is achieved either with a milk meal (milk–EHIDA test) or by intravenous cholecystokinin (EHIDA–CCK test). The amount of enterogastric reflux is calculated as a percentage of the total abdominal radioactivity The symptoms of reflux gastritis and bile vomiting may be improved by the administration of bile salt-binding agents (cholestyramine, aluminium hydroxide, charcoal). However, conservative management along these lines often fails, when

remedial surgical intervention becomes necessary. Prolonged enterogastric reflux can result in atrophic gastritis and intestinal metaplasia. It has been incriminated as a factor in the development of carcinoma of the stomach after gastric surgery, although evidence for this hypothesis remains lacking.

Extrinsic loop obstruction

This rare complication occurs after truncal vagotomy and gastrojejunostomy and usually affects the afferent loop. The predisposing factors to the development of afferent loop obstruction include antecolic anastomosis of long loops (exceeding 20cm). The causes of extrinsic loop obstruction are:

- internal herniation
- kinking of the anastomosis
- adhesions
- volvulus
- stenosis
- jejunogastric intussusception
- development of carcinoma of the gastric remnant.



The milk–EHIDA test for enterogastric reflux. The patient experienced severe bile vomiting after vagotomy and pyloroplasty. Reflux of isotope (bile) into the stomach occurs in the fasting state but is considerably enhanced after the administration of milk (as a source of fatty meal).

Obstruction of afferent or efferent loops is usually chronic and intermittent but may be acute. The symptoms of chronic afferent loop obstruction include fullness, cramp-like pain and nausea within 1 hour of eating. The attack culminates in vomiting of copious amounts of bilestained fluid which relieves the symptoms. The presentation of acute afferent loop obstruction is with severe colicky abdominal pain, nausea and vomiting, which is characteristically free of bile. Abdominal tenderness is present. The condition may be complicated by the development of acute pancreatitis, jaundice and necrosis with

perforation. Acute jejunogastric intussusception is a serious condition characterized by severe epigastric pain, vomiting, haematemesis, a palpable abdominal mass with high small bowel obstruction. Urgent surgical intervention is required because of the risk of strangulation and gangrene. The condition may be diagnosed preoperatively by a plain abdominal film which shows a softtissue epigastric mass surrounded by the gastric air bubble. Alternatively, emergency barium meal or endoscopy will establish the diagnosis.

Gastro-oesophageal reflux and oesophagitis

The situation regarding gastro-oesophageal reflux and surgery for duodenal ulcer is both confusing and controversial. In the first instance gastro-oesophageal reflux often accompanies duodenal ulcer and oesophagitis may, therefore, be present preoperatively. Transient dysphagia may occur after any type of vagotomy and this has been attributed to oedema of the lower abdominal oesophagus. It is now established that vagotomy itself does not affect the oesophageal highpressure zone, but damage to the oesophageal attachments, particularly the phreno-oesophageal membrane during the mobilization of the oesophagus, may cause cardiooesophageal incompetence. If this becomes subsequently associated with the development of enterogastric reflux of bile and pancreatic juice (neutral or alkaline reflux) a severe form of erosive oesophagitis develops. It is now documented that the eradication of *H. pylori* in patients with duodenal ulcer aggravates any underlying cardio-oesophageal incompetence and has been suggested as one of the factors accounting for the rising incidence of junctional cancers during the past 20 years.

Diarrhoea

The reported incidence of this complication varies widely, largely due to varying definitions. Three patterns of diarrhoea are encountered after gastric surgery:

- frequent loose motions
- intermittent episodes of short-lived diarrhea
- severe intractable explosive diarrhoea. Severe explosive diarrhoea is a serious, but rare disability, being encountered in 2% of patients after truncal vagotomy with drainage. It is often accompanied by dumping symptoms and is precipitated by food. Severe intractable diarrhoea is characterized by extreme urgency and often causes incontinence during an acute attack. Also the fluid motion is extremely foul smelling and this is often complained of by the partner and close family members. Although often associated with rapid gastric emptying, the exact mechanism is unknown. Malabsorption of bile salts and/ or fatty acids

consequent on the intestinal vagal denervation has been implicated but never confirmed. The small bowel transit is markedly exaggerated in all patients. A full malabsorption survey is necessary in all patients with severe diarrhoea as in a few patients this disability is secondary to a previously undiagnosed gastrointestinal disease (e.g. adult coeliac) or bacterial overgrowth consequent on a blind loop. Medical management is with a low animal fat diet, intestinal sedatives (codeine phosphate, loperamide) and bile salt-binding agents such as cholestyramine. Although temporary improvement can be obtained in this way, long-term benefit is rarely obtained with conservative measures and the patient either accepts and lives with the disability or asks for remedial surgical treatment. In the past 10 years, cases of severe explosive diarrhoea have been reported after laparoscopic fundoplication especially of the anterior (Watson) type. The incidence after this operation is about 2%. The presumed aetiology is damage to the vagal nerve trunks. These patients should be treated conservatively for 2 years as a considerable number resolve by this time. Remedial surgery may be considered if the condition persists beyond 2 years.

Small stomach syndrome

This term is sometimes used for the early satiety complained of by many patients after vagotomy, which causes loss of the receptive relaxation of the stomach during eating. It is however, best reserved for those unfortunate patients, usually females, who cannot eat after gastrectomy and whose quality of life is miserable and whose nutrition can only be maintained by enteral or parenteral feeding. In essence, these patients, because of pain and myriad other symptoms, are unable to eat and in essence starve themselves. Thus the condition inevitably leads to protein calorie malnutrition and cachexia similar to that encountered in advanced malignancy. In the author's experience, small stomach syndrome is refractory to conservative management, although in some patients nutrition can be maintained by elemental diets administered via a Clinifeed tube and an IVAC pump or via a feeding jejunostomy in those who cannot tolerate nasoenteral feeding. Although many can be trained to use this in their homes and maintain a reasonable nutritional state in this way, their quality of life is extremely poor and it is no wonder that some commit suicide. The alternative to feeding jejunostomy is home hyperalimentation with intravenous nutrition. Aside from cost, this is best avoided in these patients because of the inordinately high complication rates associated with the indwelling intravenous line. If the patient is fit and middle aged, surgical intervention designed to reconstruct a gastric reservoir and restore duodenal continuity provides the best chance for improvement and ability to maintain nutrition with an oral diet although supplementation is often needed.

Other complications

These include the formation of gallstones and bezoars and the development of gastric carcinoma. Vagotomy causes dilatation of the gallbladder. However, although there are a number of reports indicating an increased risk of gallstone formation after both vagotomy and partial gastrectomy, there is no firm evidence that gastric surgery predisposes to cholelithiasis. The factors implicated in the formation of bezoars after gastric surgery include hypoacidity, impaired proteolytic activity, inadequate mastication and loss of the antral pump. The majority of bezoars, which develop after gastric surgery, consist of undigested vegetable/fruit fibre debris (notably orange pith). Bezoars can cause chronic symptoms such as nausea, vomiting, abdominal discomfort, halitosis and early satiety. They can also lead to serious complications, e.g. small bowel obstruction, severe gastritis and ulceration, bleeding, perforation and malnutrition. Treatment is initially conservative by enzyme (cellulose) digestion or endoscopic fragmentation/removal. Surgical intervention is undertaken if medical/endoscopic therapy fails or because of development of a complication. There is good evidence that previous gastric surgery (partial gastrectomy, gastrojejunostomy) predisposes to the development of gastric carcinoma in the stomach remnant. Vagotomy does not appear to be implicated in this complication. Although reflux gastritis with the development of intestinal metaplasia, particularly of the type III variety, and bacterial overgrowth with the formation of nitrosamines in the hypochlorhydric gastric remnant have been implicated, the exact mechanism for the development of invasive carcinoma remains unknown. Also it seems likely that many of these patients had *H. pylori* gastritis that was not recognized at the time. This is now thought to have contributed to the development of gastric carcinoma. There is a long latent period of 15–20 years but the risk, though definite, is small.

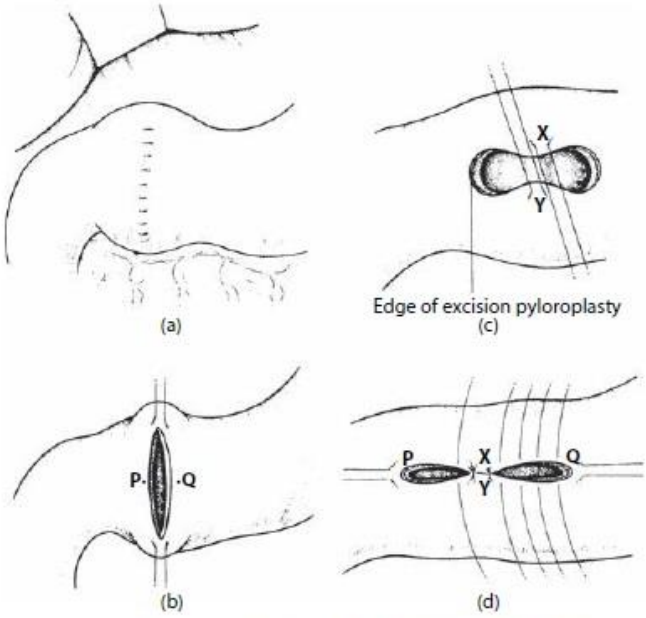
Remedial surgical treatment for postgastric surgery syndromes

General considerations As the majority of symptoms improve with time, initial management should always be conservative and surgical treatment should not be considered before 18 months to 2 years of presentation and with no improvement on conservative management. Only patients with severe symptoms which persist beyond this time despite conservative management should be considered as candidates for remedial surgery, and, ideally, the patient should request the intervention. All these patients need to be investigated and assessed (nutritionally and fitness for surgery). In addition, a detailed history of the symptoms during ‘bad days’ is essential to establish the *dominant symptom/disability* – what troubles the patient most and would improve his or her quality of life if treatment were to be successful. Remedial surgery should be directed to amelioration of this dominant symptom with the proviso, which must be conveyed to the

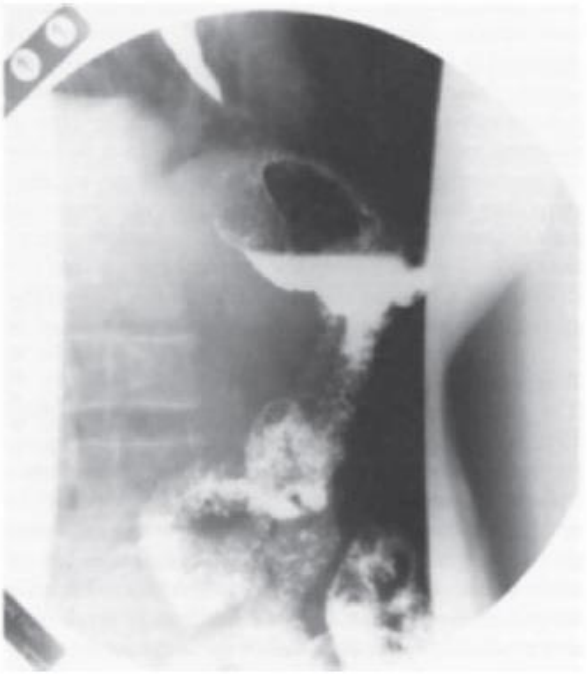
patient, that while the intended surgery may impart considerable benefit, a totally symptomfree outcome is rarely, if ever, obtained. Despite the classical descriptions of separate syndromes, most patients cannot be pigeonholed and, instead, have a mixture of symptoms, but a careful history will always determine the dominant symptom/ disability and remedial surgery should be directed exclusively to correction of this symptom if the patient is to benefit from remedial surgery.

Dominant dumping symptoms

The easiest patients to manage surgically are those who experience severe dumping after truncal vagotomy with drainage. In these patients, the remedial surgery consists of either take down of the gastrojejunostomy or pyloric reconstruction depending on the type of drainage used. Surprisingly, symptomatic gastric retention is rarely encountered, although postprandial fullness is common and the patient should be advised to avoid heavy bulky meals, lying down after meals and to eat the evening meal at least 3B€hours before retiring to bed. In practice, prokinetic drugs do not help these patients. For patients with dominant dumping symptoms after partial gastrectomy or vagotomy with antrectomy, the more technically demanding isoperistaltic jejunal interposition (10–15cm) between the gastric remnant and the duodenum acts as an effective brake to slow the gastric emptying. Bile vomiting as the dominant symptom The commonest cause of this is enterogastric reflux. Excellent results are obtained in patients with vagotomy and gastrojejunostomy by take down of the latter. In contrast, pyloric reconstruction for bilious vomiting in these patients gives unsatisfactory results and should not be attempted. These patients are best



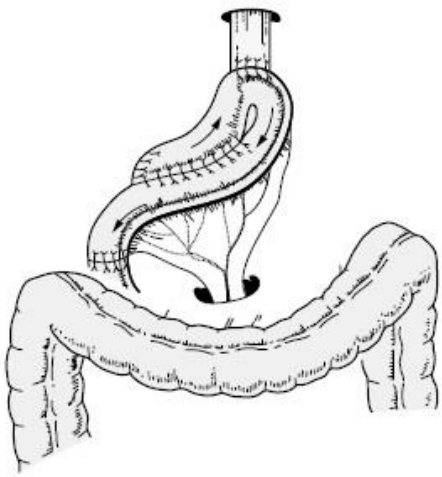
Pyloric reconstruction for dumping after Heineke–Mikulicz pyloropasty: (a) complete exposure of the pyloroplasty by freeing of adhesions; (b) insertion of stay sutures beyond both ends of the pyloroplasty scar which is carefully incised along its length; (c) precise restoration of the normal alignment of the pyloric muscular ring; (d) the realigned antropyloric segment is closed in a single layer with fine (3/0) carefully placed interrupted sutures.



Contrast radiology of isoperistaltic jejunal interposition between the gastric remnant and the duodenum.

served by an antrectomy and an isoperistaltic jejunal interposition (10–15cm) between the proximal stomach and the duodenum. Severe bile vomiting due to enterogastric reflux after partial Billroth I gastrectomy may be treated by Roux-en-Y diversion. Although very effective in abolishing the bile vomiting, this procedure may lead to bacterial overgrowth, but this is not usually a major problem. An alternative approach is reconstruction with an isoperistaltic jejunal loop between the gastric remnant and the duodenum. Dominant symptoms of the small stomach syndrome This is the most difficult symptom complex to treat. These patients are usually grossly malnourished and require a period of parenteral nutrition before the remedial surgical treatment. The best procedure consists of completion gastrectomy and the creation of a jejunal reservoir with an isoperistaltic conduit between the oesophagus and the duodenum. In these patients, the gastric stump is invariably inflamed and immotile and is therefore excised prior to commencement of the reconstruction. An improved outcome following this major reconstruction is reflected by the ability of the patient to sustain weight by a semisolid

low-roughage diet and is encountered in 50–60% of patients. The problem has been the prediction of patients who are likely to benefit from such extensive remedial surgery. Obviously, psychological factors are important in this respect and the patient requires to be well motivated to overcome the disability. Too much stress cannot, however, be based on psychological assessment as the psychological state of the patient may be the result of the disability. Preoperative weight gain on enteral feeding is reported to be a good indicator of a positive clinical outcome after remedial surgery for the small stomach syndrome. In patients who do not improve after completion of gastrectomy and creation of a gastric reservoir, or those who are unfit for major surgery, nutrition can be maintained by surgically constructed feeding jejunostomy. This is physiologically better, safer and more cost effective than permanent home hyperalimentation with parenteral nutrition.



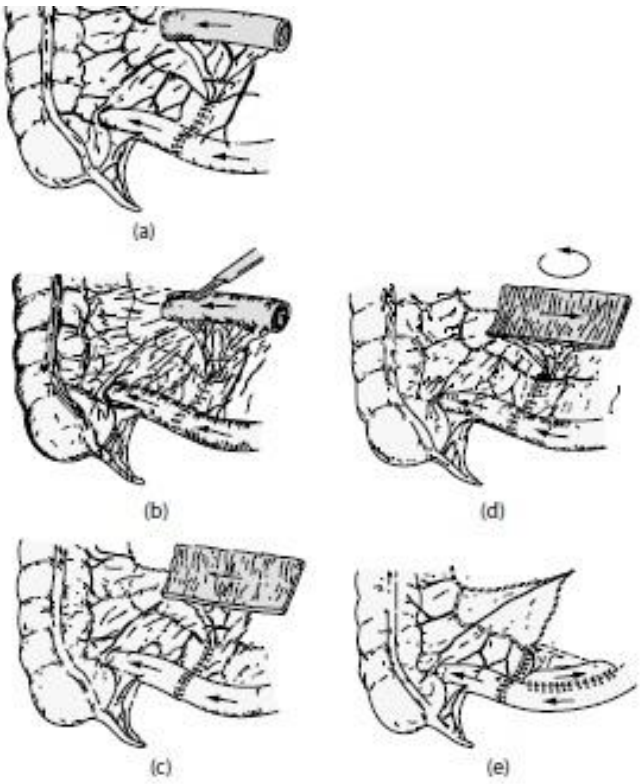
Jejunal pouch reconstruction with an isoperistaltic conduit for the small stomach syndrome. The useless gastric remnant is first removed before this reconstruction.

Barium contrast series in a patient with completion gastrectomy and reconstruction with a jejunal reservoir with an isoperistaltic conduit. The operation was performed for severe symptoms (small stomach syndrome) with good symptomatic result and weight gain on a semifluid oral diet.

Severe explosive diarrhoea

Although the use of reversed jejunal segments has been advocated, the outcome of these operations is poor due to the development of episodes of postprandial colic, intestinal

obstruction, distension and bacterial overgrowth. The best results are obtained by the distal onlay ileal graft procedure, which is designed to create a passive non-propulsive segment of the small intestine some 30–60cm from the ileocaecal junction Some 50% of patients obtain significant benefit following this operation if they avoid fatty/dairy products. Some patients complain of postprandial colic after this operation with some abdominal distension. Revision (reduction of the length of the distal onlay graft) may be necessary if these symptoms persist. Remedial surgery fails in half the patients and, in these, a temporary loop ileostomy can be offered. If the patient finds this acceptable after a trial period of 3 months, the ileostomy is made permanent, otherwise it is reversed.



Reversed ileal onlay graft for severe explosive diarrhoea: (a) a 10–12 cm segment of ileum is isolated on an intact vascular pedicle some 30 cm proximal to the caecum; (b) continuity of the small bowel is restored by an enteroenteric anastomosis; (c) the isolated segment is split longitudinally along its antimesenteric border; (d) reversal of the flap; (e) suture of the reversed graft as an onlay to the adjacent ileum following an appropriate enterotomy, thereby creating a passive non-propulsive segment. (From Cuschieri, *Br J Surg* 1986;73:981, with permission).

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All the illustrated materials are taken from «Essential Surgical Practice. Higher Surgical Training in General Surgery. Fifth Edition. Edited by Alfred Cuschieri and George B Hanna»

Tests

1. Diseases of the operated stomach include all but:
   a) Adductor loop Syndrome
   b) Hypoglycemic syndrome
   c) Anastomosis
   d) Failure of gastroduodenal anastomosis
   e) Dumping syndrome
2. Diseases of the operated stomach of organic Genesis include all, except:
   a) adductor loop syndrome
   b) dumping syndrome
   c) Barrett's esophagus
   d) stomach stump cancer
3. For dumping syndrome, everything is true except:

a) it is a complex of vascular and neurovegetative and intestinal disorders
b) occurs 3-4 hours after eating
c) occurs as a rule when taking dairy and carbohydrate foods
d) it is caused by violation of humoral regulation of digestion
e) due to violation of osmotic pressure in the intestine

4. For the clinic dumping syndrome is characterized by all but:
   a) weakness, dizziness after eating;
   b) feeling of heat, facial flushing;
   c) vomiting.
   d) diarrhea.
   e) weight loss
5. For the 2nd degree of dumping syndrome is characterized by everything except:
   a) attack lasts 30-40 minutes
   b) chair instability
   c) quickening of the pulse by 10-15 beats
   d) body weight deficit up to 10 kg
   e) duration 1.5-2 hours
6. The principles of diet therapy for adductor loop syndrome are all, except:
   a) Frequent fractional meals in small portions (5-7 times a day); take food should be slow
   b) Limit sweets (sugar, honey, jam), very hot and very cold dishes, liquid sweet milk porridge, etc.
   c) Wash down food with as much liquid as possible to dilute it

d) Inadmissibility of receiving liquid together with other dishes
e) Lie down for 15-20 minutes after eating, especially after lunch

7. Complete gastrectomy is applicable in:
   a. Early dumping syndrome
   b. Late dumping syndrome
   c. relapse of peptic ulcer caused by the syndrome Zollinger-Ellison
   d. In Alkaline reflux gastritis
   e. Postvagotomy diarrhea
8. Irrigoscopy is of crucial importance in the diagnosis of-stick:
   a. Postvagotomy gastrostasis
   b. Gastro-thin-colonic fistula
   c. Recurrent peptic ulcer of gastro-duodenal anastomosis
   d. Dumping syndrome
   e. All listed above
9. The frequency of recurrence of peptic ulcer depends primarilyqueue from:
   a. patient's Age
   b. Duration of illness
   c. Localization and size of the ulcer
   d. Complications of peptic ulcer disease present at the time primary operation
   e. type of primary operation
10. For early dumping syndrome, which occurs shortly after eating, characterized by:
    a) drowsiness and weakness
    b) nausea, bloating, diarrhea, cramping pains, rumbling
    c) tachycardia
    d) increased blood pressure
    e) all of the above

Situational task 1

Man, Thirty nine years please refer to surgeon with complaints about the constant, growing, stupid, bursting pain in the right hypochondrium and epigastrium, with irradiation in the back by type «hoop's», growing after reception's fatty, dairy foods. Also bothers vomiting 1 times in 1-2 days, after what pain significantly reduced. In the vomit notes stagnant food masses and up to 500 ml biles. Constantly worried about the bitter

taste in mouth, weight loss on 7 kg., weakness, malaise. From anamnesis it is remarkable that 4 years ago on the altitude of ulcer bleeding operated – done resection stomach's. First the described complaints appeared 3 years ago. Was treated independently-took But- Silos with a temporary positive effect. However, over time complaints progressed, there was a fear of eating, which forced to seek honey. help. When inspecting a state of moderate severity. Skin and mucous membranes - pale, subicteric, skin turgor reduced. Peripheral lymph nodes are not changed. BMI-19 Breathing vesicular, no wheezing. BPD-16 in min. Pulse – 95 in min. AD-120 and 70 mmHg. Abdomen is soft, moderately painful in epigastrium and right hypochondrium, where palpated cylindrical shape education 4*6 cm. tugoelasticheskoy consistency, smooth, with surrounding tissues are not connected, when succussion over the area of which the noise of splashing is heard; there is no tension in the abdominal muscles. S-m Shchetkin-Blumberg is not defined. C-m bashing is negative bilaterally. Chair, diuresis-without features. In the clinical analysis of blood – er Twelve - 2.16*10 the macrocytosis, the CPU is 1.6, leukocytes – Nine 10*10, hypersegmented neutrophils. In the biochemical analysis of blood: bilirubin-total-45 mmol / l, direct-27 mmol/ l, indirect -18 mmol/l, blood amylase-56 mg. urine Diastase (according to Wohlgemuth) - 224 (norm-16-64).

1. Your preliminary diagnosis. Justify it. Explain the pathogenesis.
2. What additional, special methods researches shown the patient?
3. Explain the reason for the laboratory changes.
4. Describe the patient's radiograph attached to the problem.
5. Assess the severity of the disease.
6. Your final diagnosis. Make a differential diagnosis your diagnosis with pancreatitis, cholecystitis, disease the head of the pancreas (S-m Courvoisier?).
7. What treatment is indicated for this patient. Describe the main methods surgical treatment.

Situational task 2

A man, 28 years turned to the surgeon with complaints of dizziness, fainting condition 10-15 minutes after eating, forcing the patient to occupy horizontal position, unpleasant sensation in the epigastric region, diarrhea 2-3 times a day especially after dairy food, weight loss of 12 kg for 3 months. From anamnesis it is noteworthy that 6 months ago he was operated on for scar-ulcer stenosis of the output part of the stomach-gastric resection is performed. First the described complaints appeared 1 month after the operation. For medical he didn't ask for help. However, over time the complaints progressed, appeared expressed weakness, dizziness, impossibility to perform daily work, which forced to apply for honey. help. When inspecting a state of moderate severity. Skin and mucous membranes - pale, skin turgor reduced. The patient is asthenic, emaciated. Peripheral lymph nodes are not altered. Breathing vesicular, no wheezing. BPD-16 in min. Pulse - 95 in min. AD-120 and 70 mmHg.my Stomach is soft, painless. S-m Shchetkina-Blumberg is not defined. C-m bashing is negative bilaterally. Chair by type of steatorrhea. In the clinical analysis of blood – er Twelve - 3.16*10 , microcytosis, poikilocytosis, CP-0.6, leukocytes – Nine 3*10 . In the biochemical analysis of blood: bilirubin – total-25 mmol / l, direct-10 mmol/ l, indirect-15 mmol/l, total protein – 45 g/l.

1. Your preliminary diagnosis. Justify it. Explain the pathogenesis.
2. What additional, special methods researches shown the patient?
3. Explain the reason for the laboratory changes.
4. Assess the severity of the disease.
5. What treatment is indicated for this patient. Describe the main methods surgical treatment.

Situational task 3

After 3 months after resection of 2/3 of the stomach by Billroth II in the modification of the Hofmeister-Finsterer for stomach ulcers, the patient complained of weakness occurring 15 minutes after eating, accompanied by a feeling of heat in the upper half of the trunk, sharp sweating. After a while he begins to feel dizzy, tinnitus, palpitations, trembling limbs, then joins the feeling of fatigue, drowsiness, loose stools, polyuria. No abdominal pain. Attacks are more pronounced when eating foods rich in carbohydrates. After surgery, the patient does not add weight. The body weight deficit is 8 kg.

Questions to the task of surgery

1. Your diagnosis? Classification of this pathology?

2. Pathogenesis of the disease?

3. What examination does this patient need?

4. What is the therapeutic tactics in the development of this pathology?

5. Indications for surgical treatment and the main types of operations?

6. How to prevent the development of this disease?

Check yourself

Tests

1	2	3	4	5	6	7	8	9	10
c	c	b	e	c	a	b	c	c	d

Situational task 1

1. The diagnosis of Dumping syndrome.

2. There are early and late (hypoglycemic) dumping syndrome.

According to the severity of the course, there are 3 degrees of dumping syndrome (mild, moderate, severe)

3. In pathogenesis, the main importance is the loss of the pylorus mechanism, rapid emptying of the stomach. When the duodenum is turned off from digestion, the intake of a large amount of concentrated and undigested food rich in carbohydrates into the jejunum leads to the expansion of the jejunum, the release of a large amount of intestinal hormones.

4. Diagnosis is based on the evaluation of clinical data, the results of provocative tests, the results of x-ray and endoscopic examination.

5. Conservative treatment is used for dumping syndrome of mild to moderate severity.

6. Surgical treatment is indicated for severe dumping syndrome, no effect from treatment, progressive weight loss. Options transactions: the sewing antiperistaltic segment of jejunum between the stomach and the small intestine, redoutensale (resection by Billroth-2), the reconstruction of the gastro-jejunal anastomosis in Roux.

7. To carry out anti-dumping operations.

Situational task 2

1. Dumping syndrome.

2. There are early and late (hypoglycemic) dumping syndrome.

According to the severity of the course, there are 3 degrees of dumping syndrome

3. In pathogenesis, the main importance is the loss of the pylorus mechanism, rapid emptying of the stomach. When the duodenum is turned off from digestion, the intake of a large amount of concentrated and undigested food rich in carbohydrates into the jejunum leads to the expansion of the jejunum, the release of a large amount of intestinal hormones.

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Situational task 3

1. Dumping syndrome.

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