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DISEASES OF OPERATED STOMACH

a video lecture for 4th grade students

in speciality 31.05.01 General Medicine

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LECTURE PLAN:

Relevance of the problem and statistical data

Summary of surgical methods for ulcerative disease treatment

Terminology and classification of post-resection disorders

Mechanism of post-resection syndrome development

General clinical characteristic

Treatment of post-resection syndromes and problems of prevention

Literature

Operated stomach diseases (OSD) are inherently iatrogenic, being a consequence of a surgical intervention that drastically changes anatomic and physiological ratios and interrelations of the digestive system organs.

Depending on the nature of surgery performed, two types of postoperative disorders are distinguished:

post-resection

post-vagotomy

HISTORY OF THE DISEASE

Over the past two decades, the ratio between *post-resection and post-vagotomy disorders* has changed, which reflects the opinions of surgeons on the therapeutic tactics for complicated and non-complicated gastroduodenal ulcers.

Thus, in early 90s, the main method for surgical treatment of ulcerative disease was supposed to be selective proximal vagotomy that was performed for both complicated and non-complicated ulcers.

At the present time, patients with non-complicated ulcerative disease almost never undergo surgical treatment due to the developed possibilities in pharmaceutical therapy of gastroduodenal ulcers and sharp decrease in disease recurrence after H. pylori eradication.

Vagotomy as a self-standing method of choice has only retained its significance in a small group of complicated gastroduodenal ulcers.

In this connection, as well as due to the increased frequency of surgeries (resections) for cancer, the modern physician more often encounters post-resection disorders:

dumping syndrome

hypoglycaemic syndrome

afferent loop syndrome

peptic anastomotic ulcer

post-resection dystrophy

post-resection anaemia

The clinical variants of these disorders are closely related to the type of the surgical intervention performed. Therefore, it is necessary to know precisely what exact surgery was provided to the patient in each case.

MAIN TYPES OF GASTRIC SURGERY

Gastric resection.

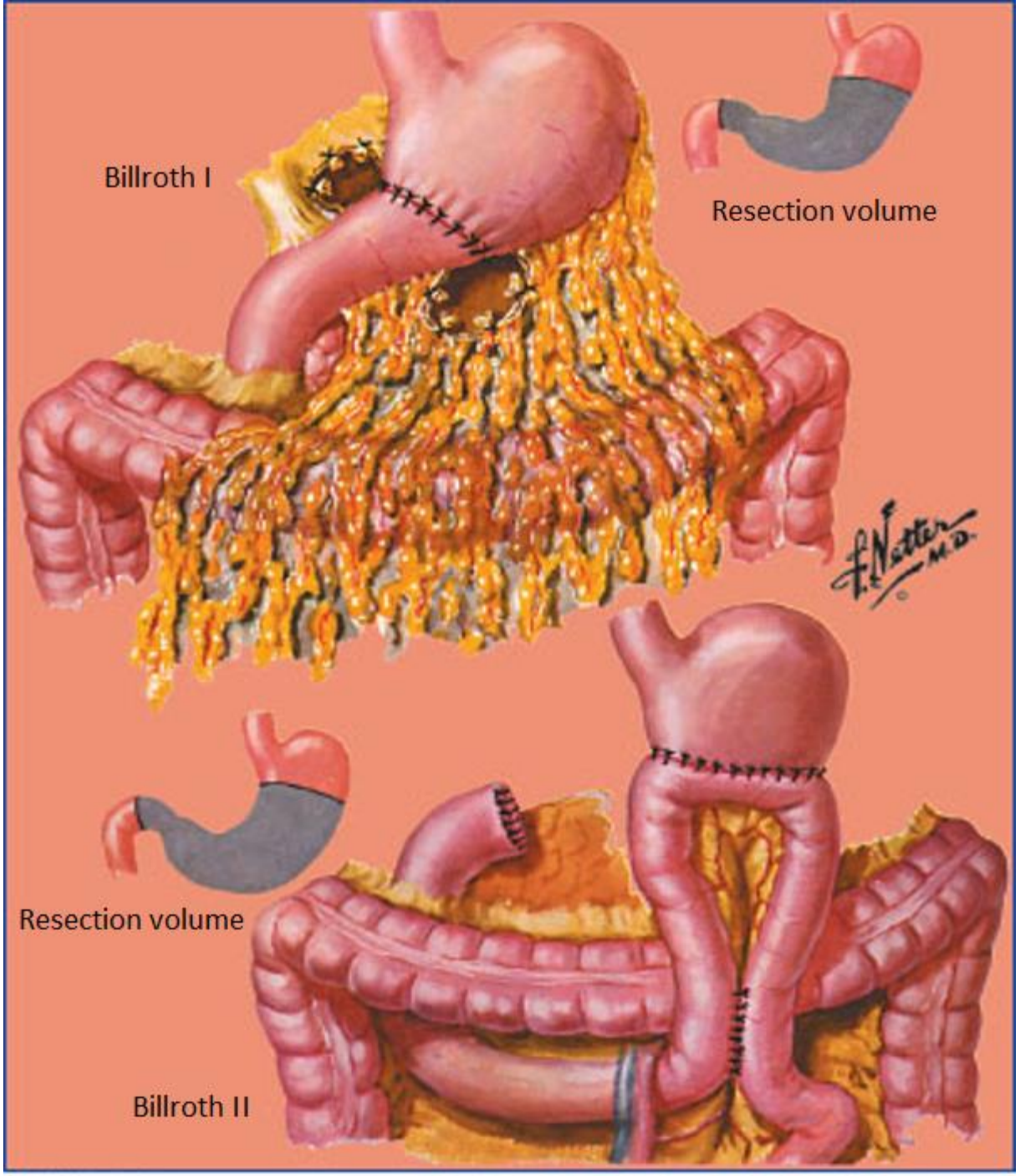
A total of three main types of gastric resection exist:

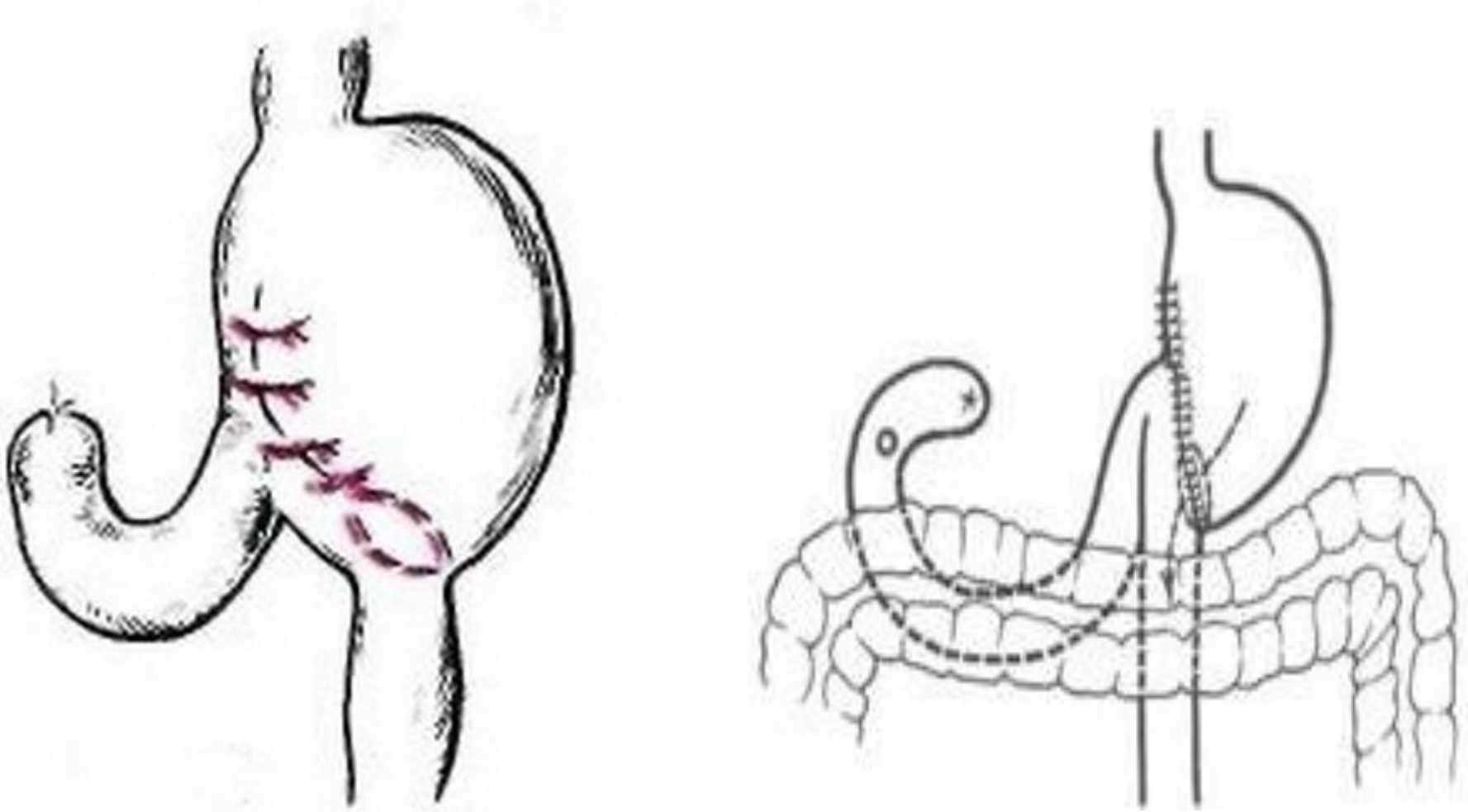
Billroth I surgery

Billroth II surgery

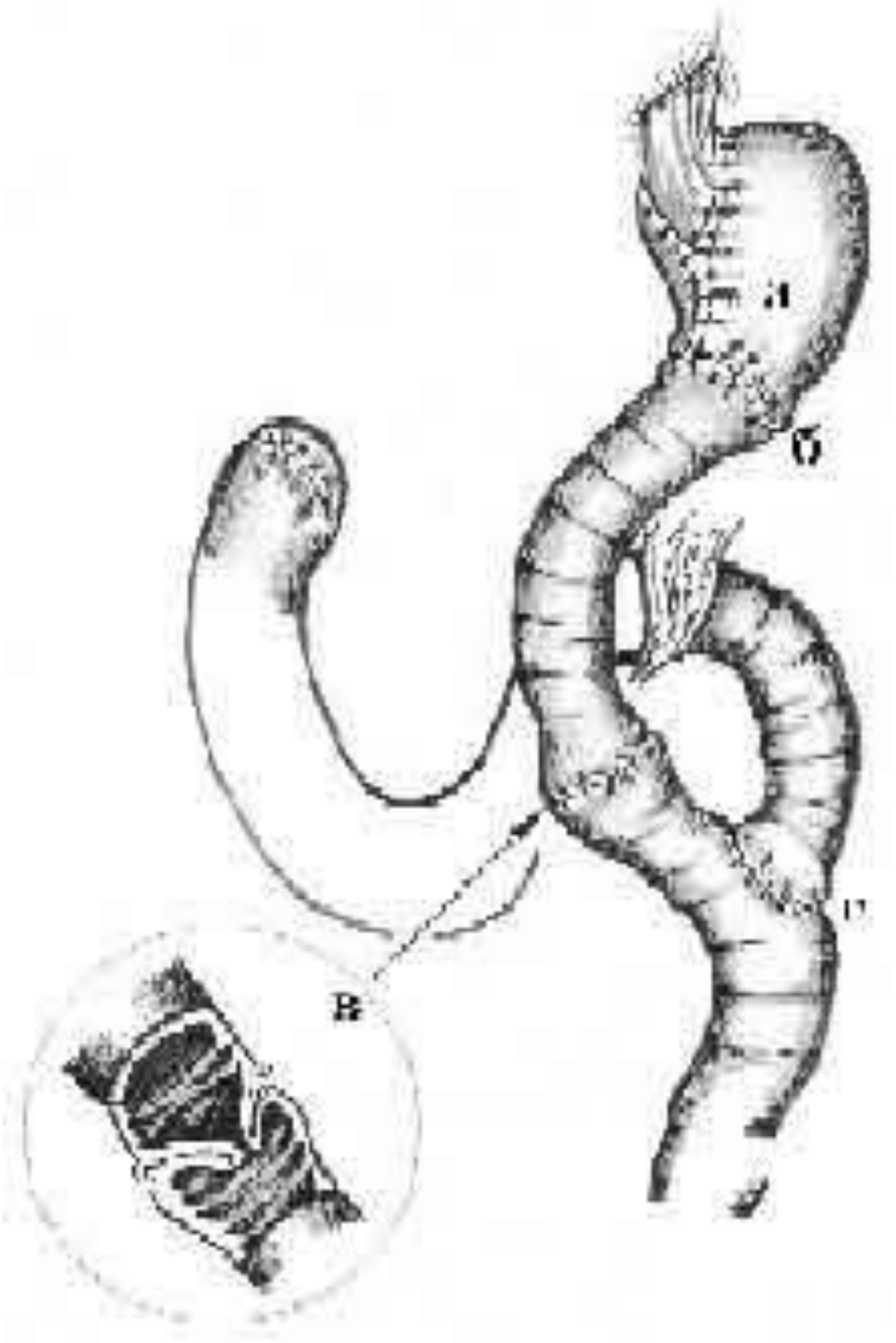
Roux surgery

All other suggested surgeries are modifications of the three.


Billroth I
Resection volume
F. Netter M.D.
Resection volume
Billroth II



Billroth II gastric resection,
modification by Hofmeister-Finsterer



Gastric resection with Roux anastomosis

AETIOLOGY AND PATHOGENESIS

The development of OSD is based on various impairments of anatomic and physiological activity of the digestive. There is a number of prerequisites for OSD development.

the nature of surgery (the volume and method of gastric resection, selectivity and completeness of vagotomy) and the disease the patient was operated for

concomitant gastrointestinal diseases which may decrease the compensatory capabilities of the organism and create the conditions for ISD development

CLASSIFICATION OF POST-RESECTION SYNDROMES

Group I: functional disorders.

Dumping syndrome

Hypoglycaemic syndrome

Afferent loop syndrome (functional)

Group II: organic disorders.

Peptic anastomotic ulcer

Cicatricial deformation of anastomosis

Gastrocolic fistula

Gastric stump cancer

The errors in choice of the ileum for anastomosis

Afferent loop syndrome (mechanical nature)

Severe ulcerative oesophagitis, jejunitis, etc.

DUMPING SYNDROME

Occupies the leading place among post-resection disorders. According to different authors, it emerges in 3.5-38% of patients after Billroth II gastric resection.

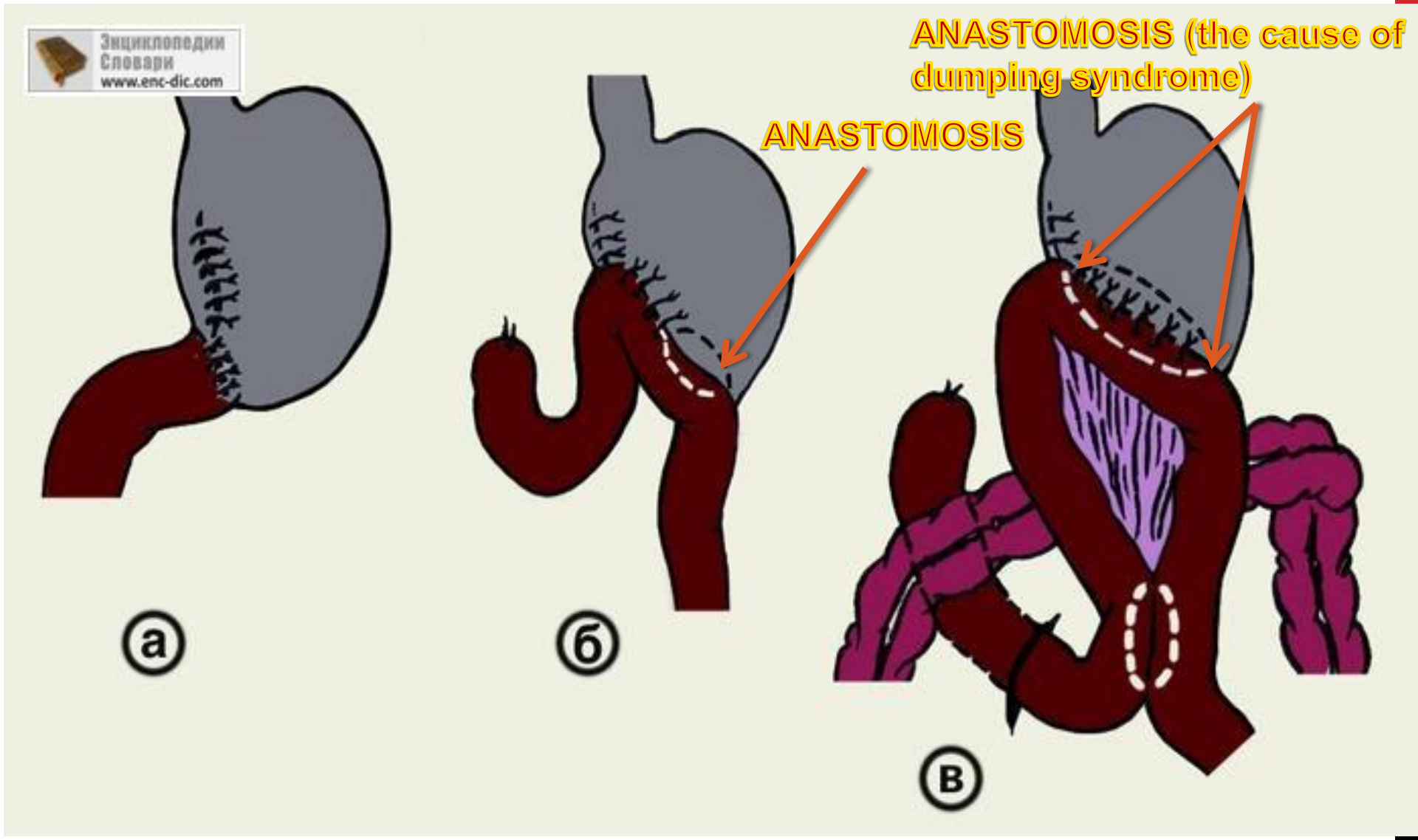
The pathogenesis of dumping syndrome is complex. In its development, the main significance is attached to accelerated evacuation of gastric contents and rapid passage of food masses along the small intestine.

THE ENTRANCE OF HYPEROSMOLAR FOOD INTO THE SMALL INTESTINE LEADS TO A NUMBER OF DISORDERS:

– increase in osmotic pressure in the intestine with diffusion of the liquid into its lumen and, as a result, decrease in the CBV

– rapid absorption of carbohydrates stimulating excess production of insulin, which first leads to hyperglycaemia and then hypoglycaemia

– irritation of the receptor mechanism of the small intestine leading to stimulation of biologically active substance production (acetylcholine, kinins, histamine, etc.), increase in the level of gastrointestinal hormones (secretin, cholecystokinin, motilin, vasoactive intestinal polypeptide, etc.)


Энциклопедии
Словари
www.enc-dic.com
ANASTOMOSIS (the cause of dumping syndrome)
ANASTOMOSIS
а
б
в

THE CLINICAL PICTURE OF DUMPING SYNDROME INCLUDES:

vasomotor component **(weakness, sweating, pronounced heartbeats, pallor or hyperemia of the face, sleepiness, increased ABP, vertigo, faintness)**

gastrointestinal component **(heaviness and discomfort in the epigastrium, abdominal murmur, diarrhoea, as well as vomiting. belching and other dyspeptic phenomena)**

These phenomena appear during food intake or 5-20 minutes later, especially after intake of sweet or dairy products. The duration of attacks is 10 minutes to several hours.

BY THE SEVERITY:

Mild dumping syndrome is characterised by intermittent, short-term weakness episodes appearing after intake sweet or dairy foods. The general condition of the patients is quite satisfactory, the work capacity does not decline.

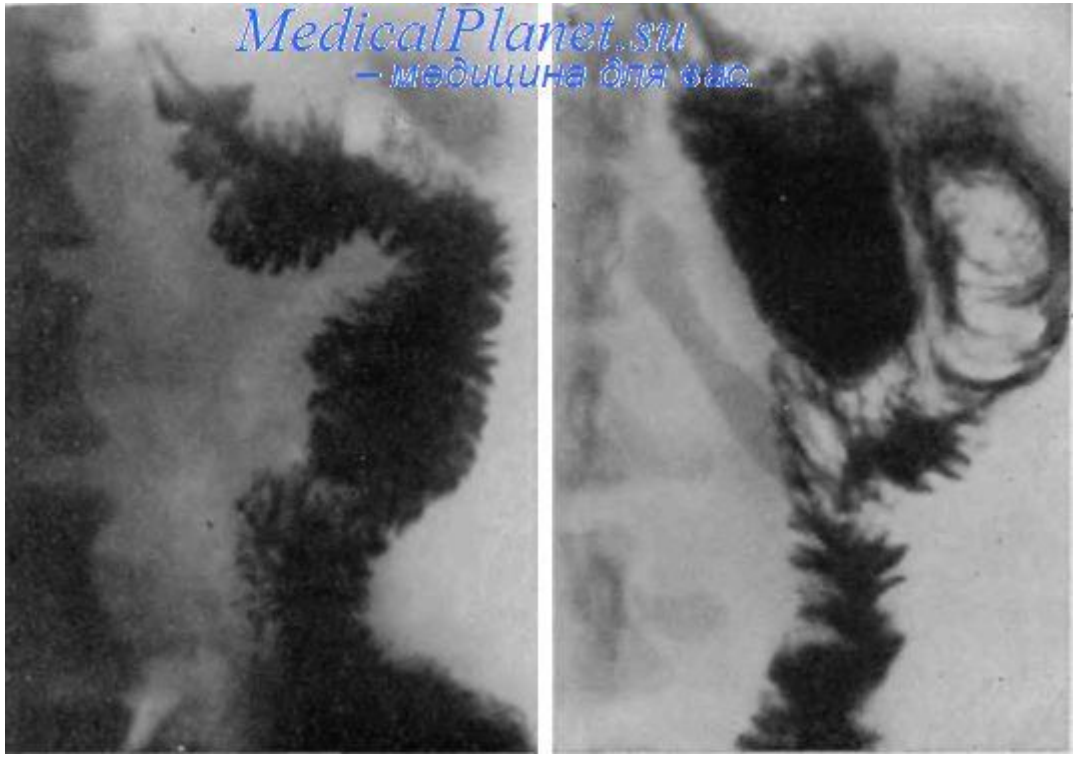
In moderate dumping syndrome, pronounced vasomotor and intestinal impairments appear after intake of any food, especially sweet and dairy products, due to which the patient is forced to assume the horizontal position. The work capacity is reduced.

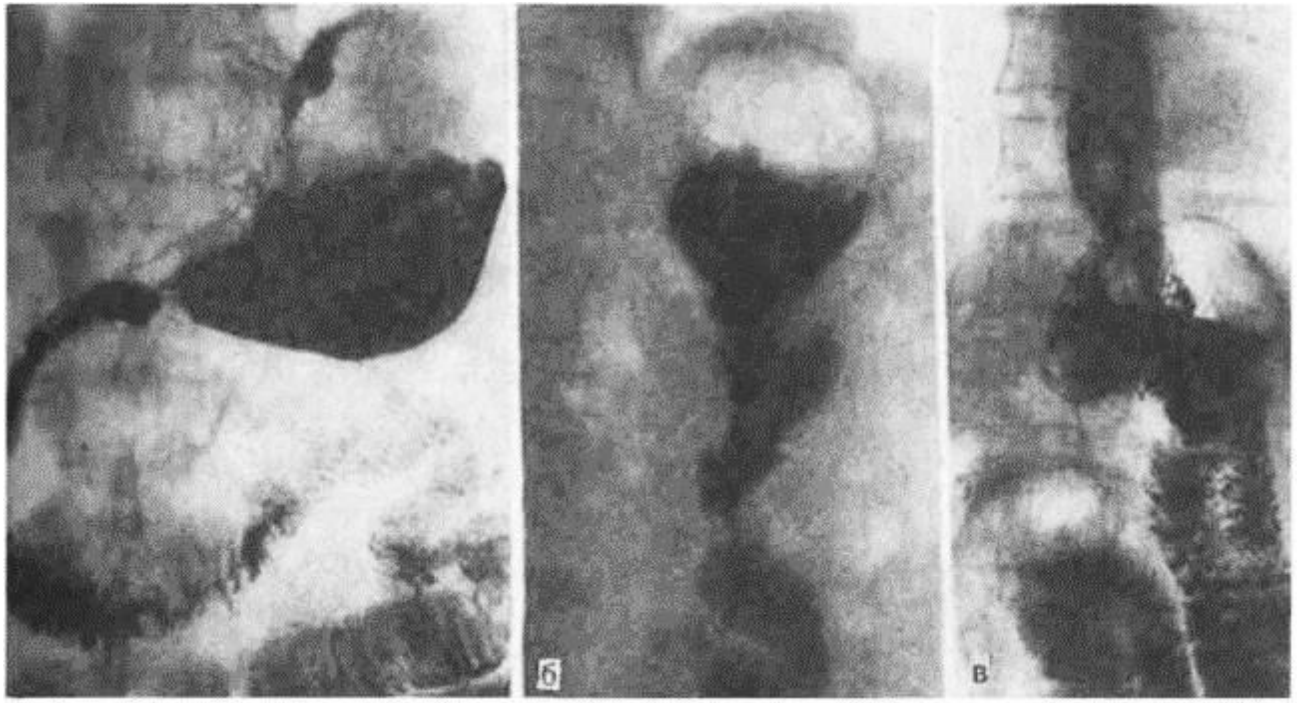
In severe dumping syndrome, almost any food intake is accompanied by a pronounced and long weakness episode, vertigo or faintness. The working capacity declines severely, the patient becomes disabled.

DIAGNOSIS OF DUMPING SYNDROME

In presence of characteristic symptoms, it is not complicated. Rapid evacuation of barium suspension (“dumping”) from the gastric stump and accelerated passage through the small intestine revealed by means of radiological examination.

A characteristic glycaemic curve after carbohydrate load confirm the diagnosis.





Radiological picture of an operated stomach
а) After Billroth I resection, б) after Billroth II, c) after gastrectomy

TREATMENT OF DUMPING SYNDROME.

I. **Conservative (mild and moderate syndrome)**

Diet therapy

Transfusion of blood plasma and protein substances.

Correction of water-electrolyte imbalance

Application of digestive enzymes (festal, creatine, etc.)

Application of sedatives (elenium, valerian, belloid, water therapy, physical exercise)

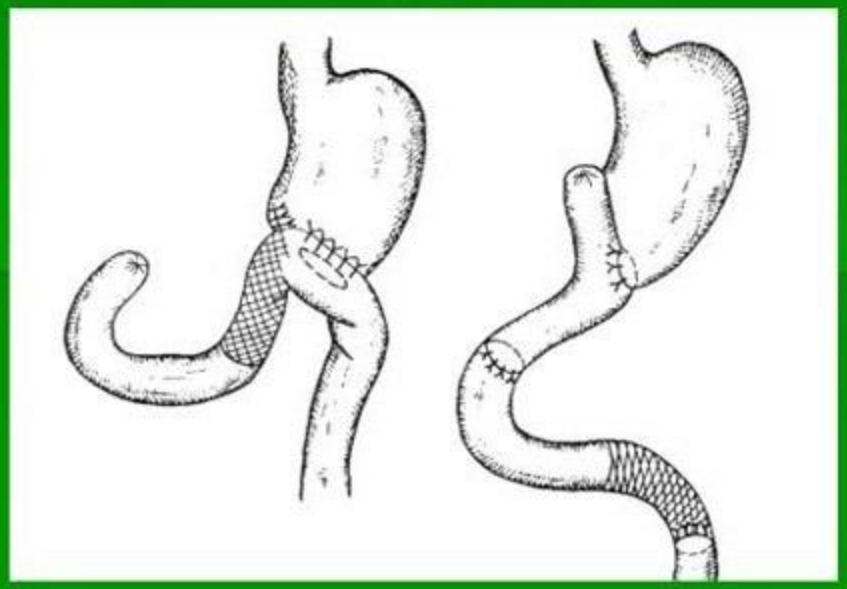
II. Operative:

Adequate resection

Restoration of duodenal passage.

Types of operations for surgical correction of dumping syndrome:

- Gastrojejunoduodenoplasty with inclusion of the duodenum into the food digestion process (via inserting a 10-15cm part of the small intestine between the gastric stump and the duodenum): Sakharov-Henley surgery (postion-wise flow of the contents from the gastric stump into the duodenum with improvement of absorption and digestion in the small intestine).



HYPOGLYCAEMIC SYNDROME.

DESCRIBED FOR THE FIRST TIME BY LAPP AND DIEBOLD IN 1931 (IN LITERATURE, THIS CONDITION WAS TITLED THE POSTPRANDIAL OR HYPOGLYCAEMIC SYNDROME)

THE DEVELOPMENT FREQUENCY OF THIS SYNDROME IS 10-50% OF THE CASES

CLINICAL PICTURE OF HYPOGLYCAEMIC SYNDROME

acute muscular weakness (2-3 hours after food intake)

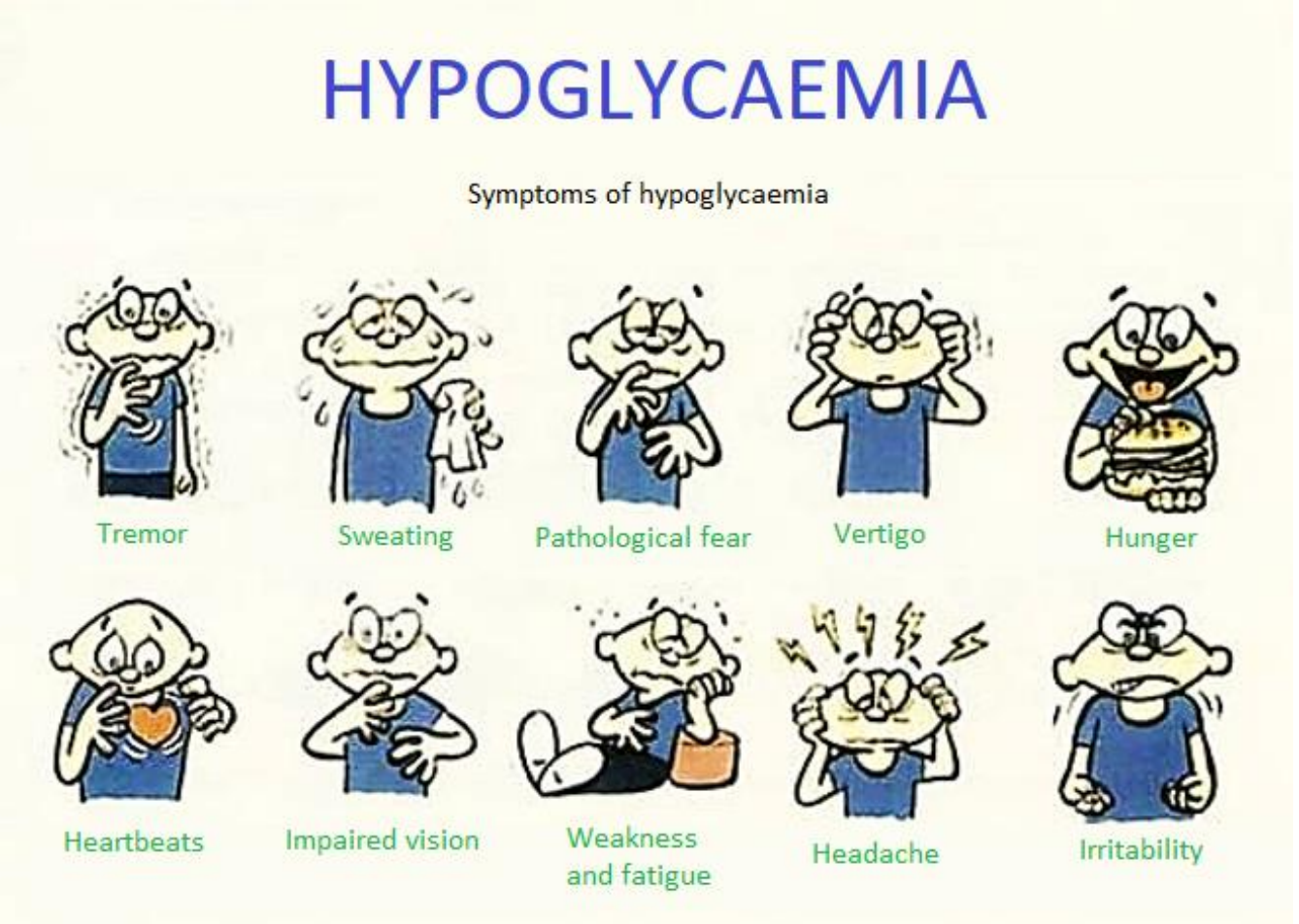
fatigue

headache

decreased ABP

sensation of hunger

loss of consciousness



PATHOGENESIS OF HYPOGLYCAEMIC SYNDROME.

1. **Intensified pancreatic insular function**
2. **Hyperinsulinemia as a response to hyperglycaemia**
3. **Action of the secretin enzyme influencing the insulin production**

CLINICAL PICTURE BY THE COURSE OF THE DISEASE:

1. **Mild (1-2 attacks per week, 1.5-2 hours). Work capacity is retained**
2. **Moderate (2-3 attacks per week. The patients are informed and eat more frequently if possible)**
3. **Severe (attacks several times per day every day, the patients assume horizontal position, carry sugar and bread with them)**

MECHANISM OF HS

It is considered that, as a result of accelerated emptying of the gastric stump, the small intestine receives a large amount of carbohydrates ready to be absorbed at one time. Blood sugar level rises rapidly and drastically, hyperglycaemia leads to a response from the humoral regulation system with excessive production of insulin. The decrease in insulin volume leads to the decrease of sugar concentration and development of hypoglycaemia.

DIAGNOSIS OF HS

Based on a characteristic clinical picture.

The syndrome manifests through excruciating hunger sensation, spastic pain in the epigastrium, weakness, increased sweating, sensation of hotness, pronounced heartbeats, vertigo, darkened vision, tremor of the whole body and sometimes loss of consciousness. The attack emerges 2-3 hours after food intake and lasts from several minutes to 1.5-2 hours.

The glycaemic curve after glucose load in most patients is distinguished by rapid and drastic increase and the same character of decrease of blood glucose below the initial level.

HS is often combined with dumping syndrome but may be observed on its own.

TREATMENT OF HYPOGLYCAEMIC SYNDROME.

Conservative:

1. Diet therapy
2. Transfusion of protein substances
3. Vitamin therapy
4. Adherence to the regimen of work and rest

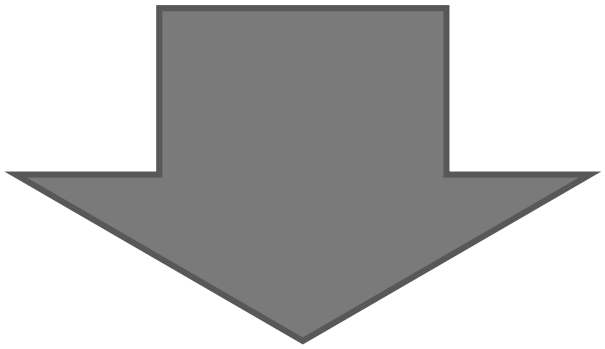
Surgeries:

1. Adequate resection (absolute and relative indications)
2. Plastic surgery (different inserts)
3. Restoration of duodenal passage

SYNDROME OF POST-RESECTION GASTRIC ASTHENIA

(Recently, it has been distinguished as a separate syndrome)

The cause is the impairment of digestive functions of the stomach, pancreas, liver and small intestine, which leads to:



development of atrophic changes in the gastric, duodenal and small intestine stump.

achlorhydria, decrease of the digestive function of the gastric juice, development of pathological flora (jejunitis, duodenitis, hepatitis, cholecystitis) and dysbacteriosis.

impairment of protein, lipid and carbohydrate absorption.

impairment of mineral exchange (Ca, phosphor – osteomalacia, osteoporosis: 1-3%)

The syndrome is aggravated by development of anaemia (iron deficiency and megaloblastic)

AFFERENT LOOP SYNDROME

Described by Braun in 1883 (duodenostasis, duodenobiliary dyskinesia, cecum syndrome, blind loop syndrome). The current name was proposed by Marshall in 1950.

Observed in 3.5-42% of the cases.

CAUSES:

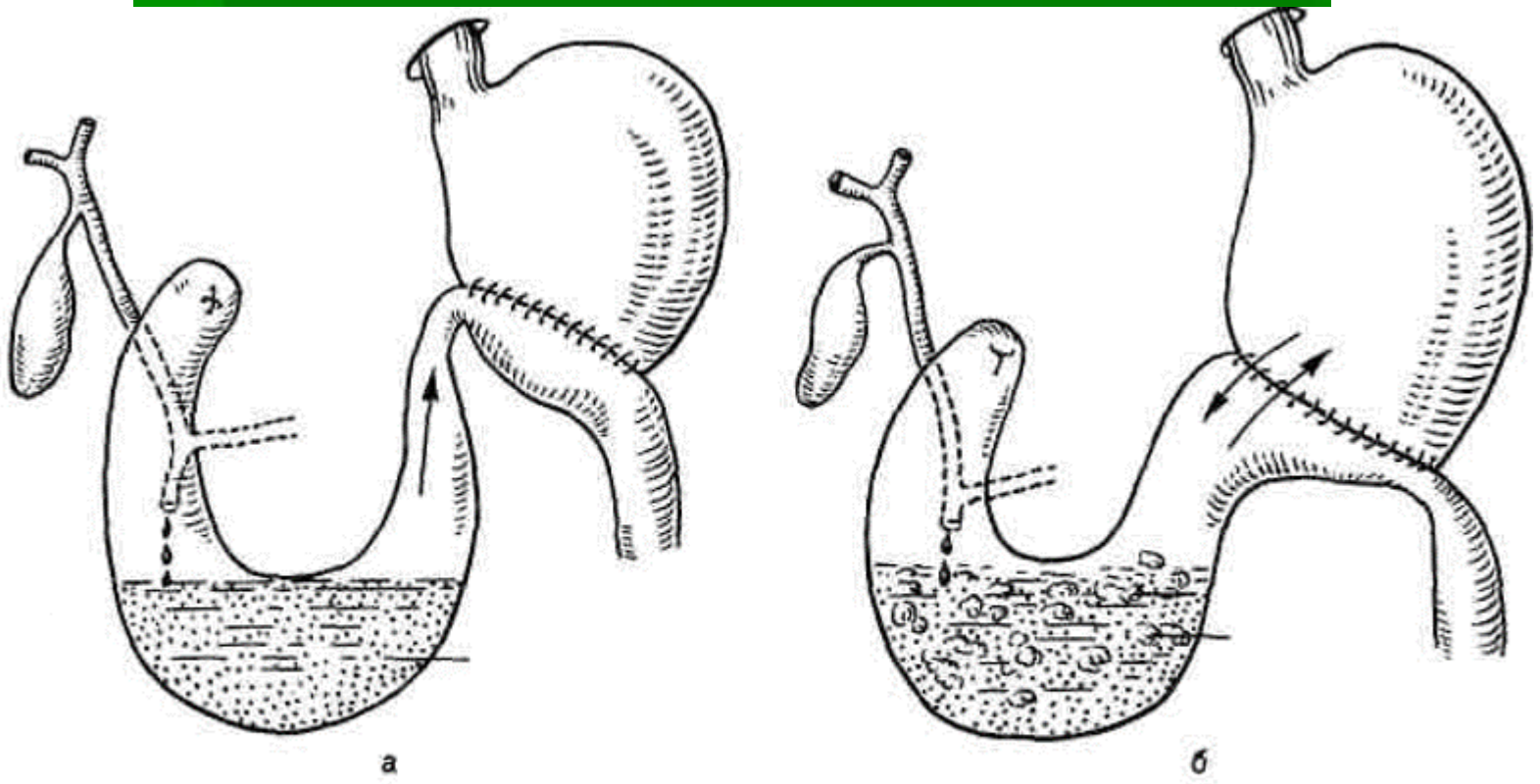
Acute obstruction **to 2% (adhesions, volvulus, deformation, anastomotic stenosis, kinking, complicated outflow of duodenal juice). The clinical picture: pain, vomiting, poisoning, increased body temperature, increased heart rate, leucocytosis.**

Chronic obstruction **shows the frequency of 21.5%**

1. **Adhesions and invagination (more often, after B-II surgery)**
2. **Biliary dyskinesis.**
3. **Damage to n. vagus.**

AFFERENT LOOP SYNDROME

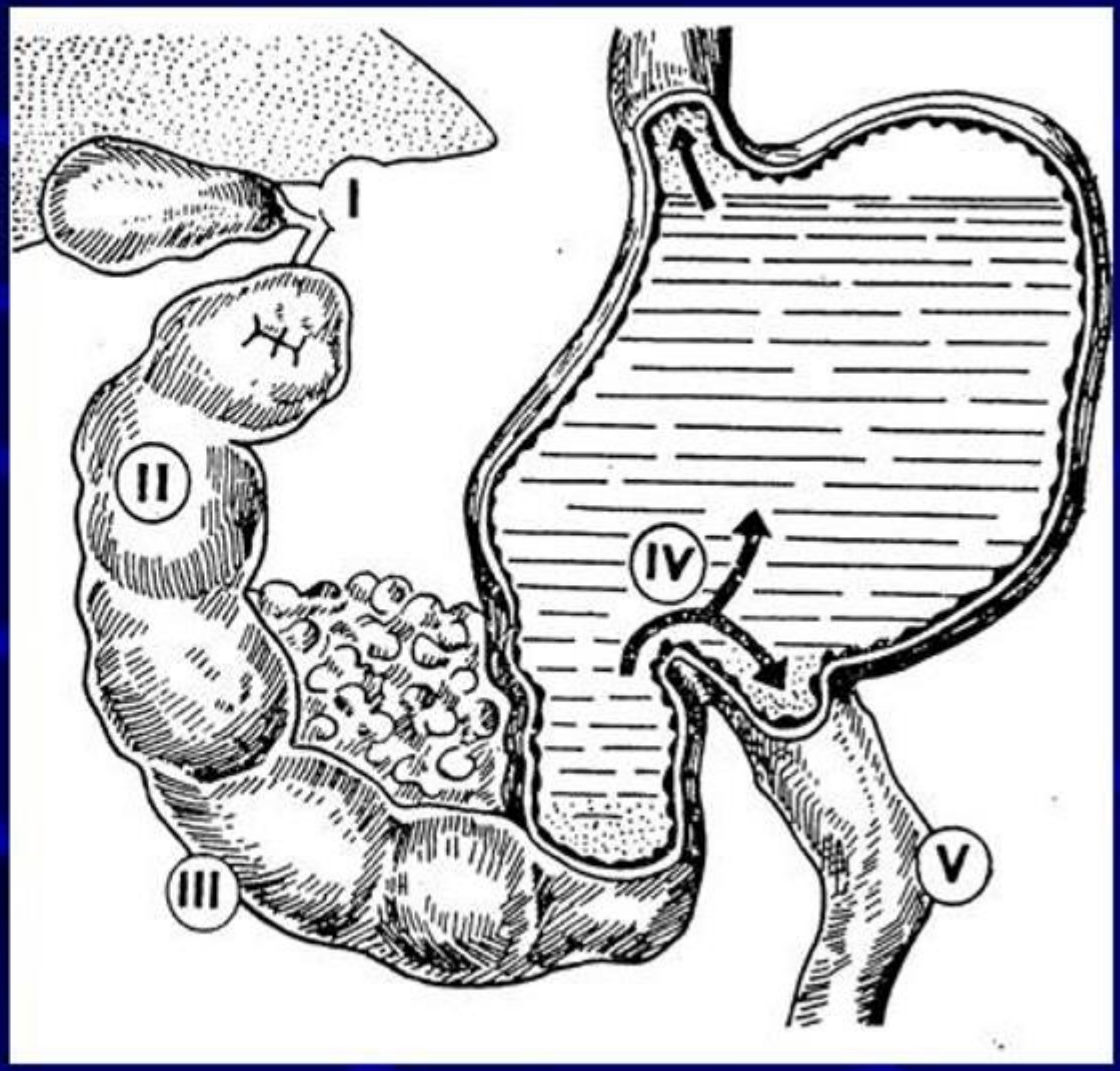
- May develop after Billroth II gastric resection accompaniet impaired evacuation of the gastric contents from the afferent loop, observed in 0.8-14% of the cases



Afferent loop syndrome
а) Afferent loop stenosis; б) efferent loop stenosis

Pathogenesis of afferent loop syndrome

congestion of bile in
e hepatic ducts,
rrhotic changes;
- development of
cterial microflora in
e blind loop;
- dyskinesis of the
ferent loop,
ngestion of intestinal
ntents;
- inflow of the
ntents from the
ferent loop
- hypermotor
skinesis of the
ferent loop.



CLINICAL FORMS OF AFFERENT LOOP SYNDROME.

A total of four forms of the syndrome are distinguished:

1. Atonic form (2-3 hours after food, there is heaviness, vertigo, weakness, vomiting with bile without food admixture, its oscillation-like movement). 10-15 minutes after food intake, bitter taste in the mouth appears with growing retrosternal pain and profuse vomiting with bile, up to 1 litre.

2. Hypertensive form (pain, vomiting with food and bile)

3. Food impaction (emerges 2-3 months after surgery. Characterised by vomiting with the food and then clean bile)

4. Partial obstruction of the afferent with impairment of evacuation of bile and pancreas

SEVERITY OF AFFERENT LOOP SYNDROME.

1. ***Mild*** **(vomiting 1-2 times per month, belching).**

   Work capacity is retained

2. ***Moderate*** **(vomiting with bile, pain, attacks 2-3 times per week)**

3. ***Severe*** **(profuse vomiting with bile, pain every day, exhaustion and dehydration). Work capacity is lost**

TREATMENT OF AFFERENT LOOP SYNDROME

Conservative treatment **is of low efficacy.**

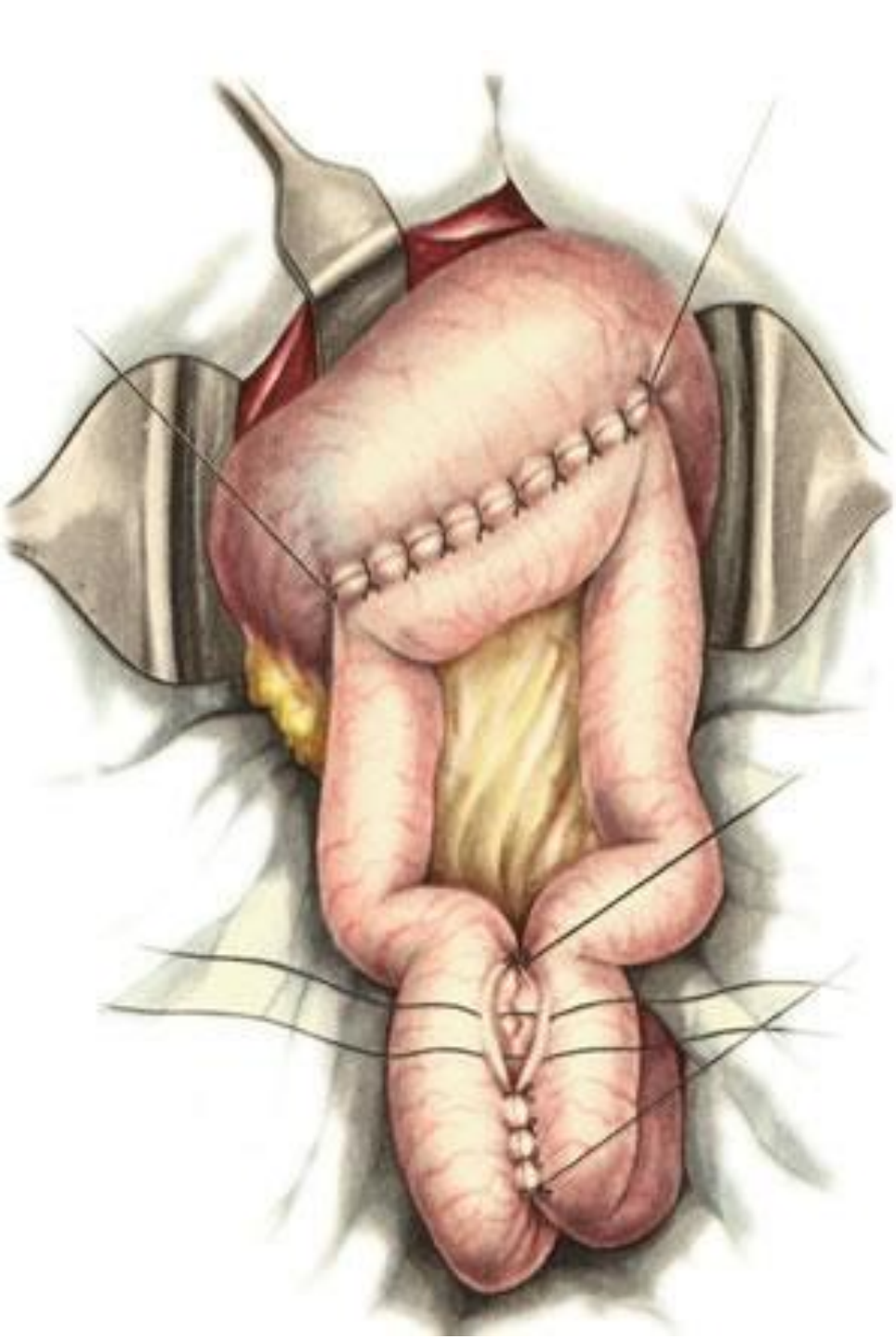
Surgical treatment:

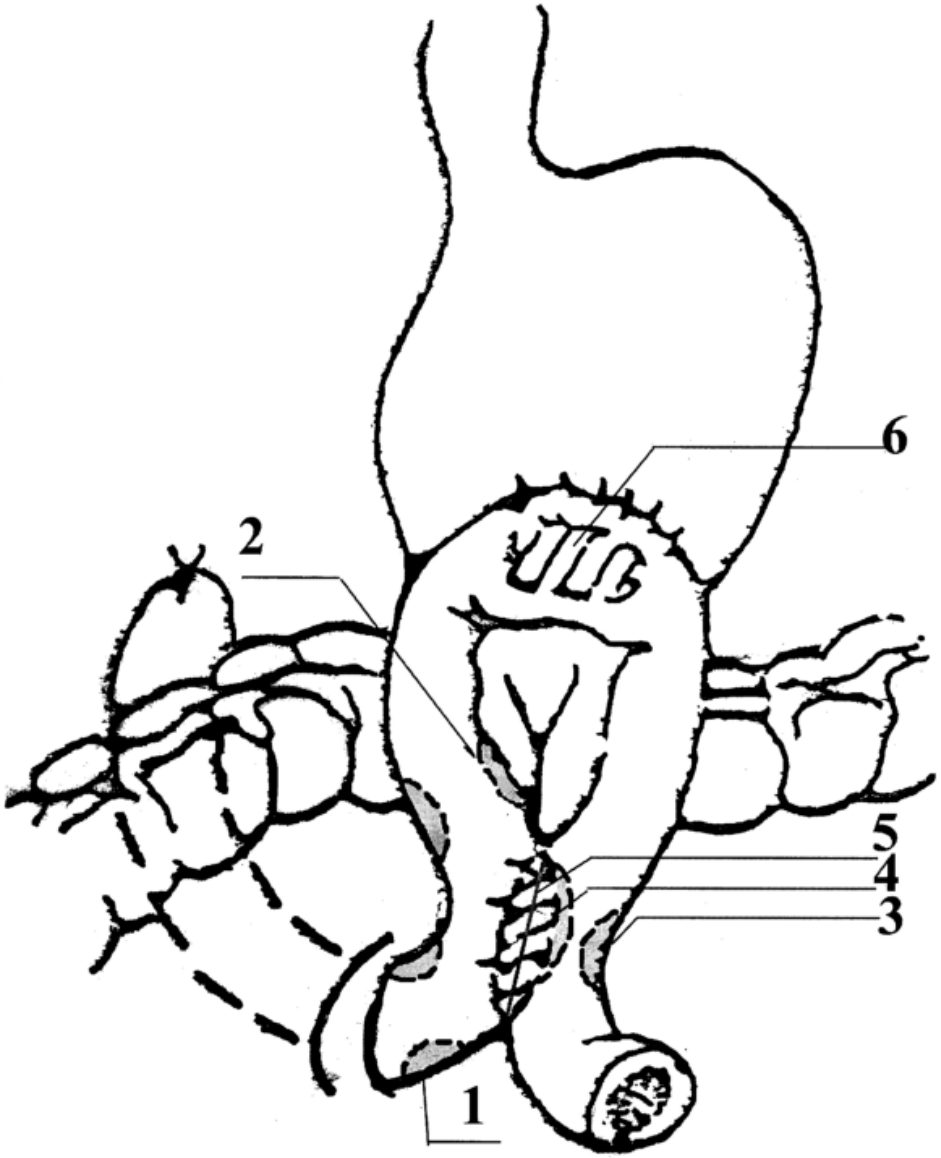
a) elimination of adhesions and kinkings

b) reconstructive surgeries

c) drain surgeries

Prevention of the syndrome is the correct performance of the initial surgery.





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GROUP II OF THE SYNDROMES ORGANIC IMPAIRMENTS

1. *Peptic anastomotic ulcer.*

We may find the record of this syndrome in works by Berg in 1897. The first clinical description was made by Haberer in 1929. The frequency of its development is 5-15%.

***Causes*:**

1. **Sparing gastric resection**
2. **Retaining the antrum with its mucosa**
3. **Vagal hypertonia in absence of vagal section**
4. **Endocrine diseases (10%):**

Zollinger-Ellison syndrome

Wermer's syndrome, "parathyroid adenoma"

Schimke syndrome (thick lips, large jaw, multiple neurinomas)

PEPTIC ANASTOMOTIC ULCER. CLINICAL PICTURE, DIAGNOSIS, TREATMENT.

***The clinical picture* resembles primary ulcerative disease.**

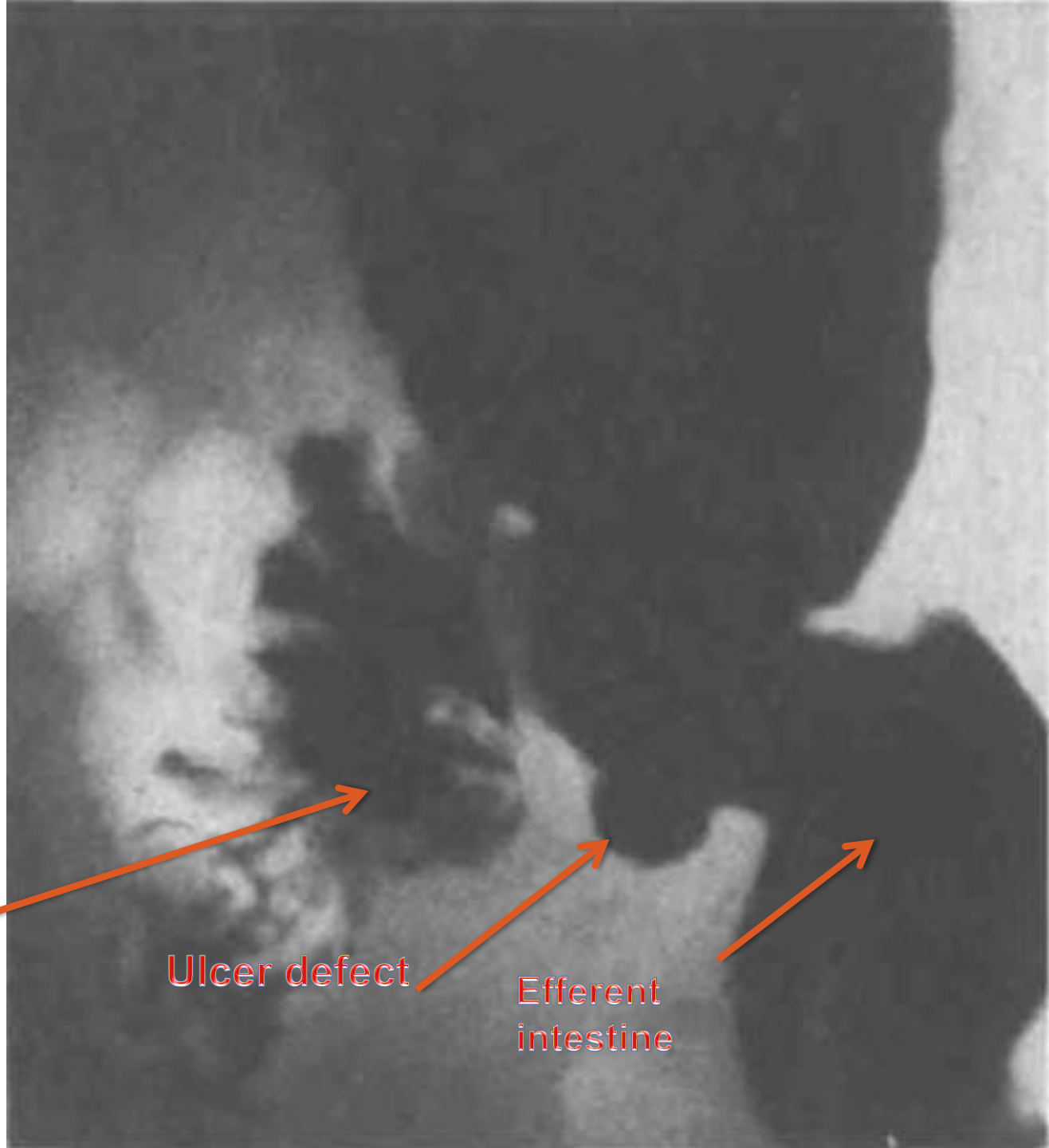
***The diagnosis* consists in gastric juice analysis, radiological examination, endoscopy, Inghimans sample – blood gastrin level measurement.**

Treatment

- conservative treatment is of low efficacy

- surgery is the main treatment method with high case fatality rate

Anastomotic ulcer after Billroth II gastric resection



POST-RESECTION DYSTROPHY (PD)

Most often develops after Billroth II gastric resection. Pronounced metabolic disorders that may also be classified as PD are observed in 3-10% of the cases.

In their pathogenesis, the leading role is considered to be digestion and absorption disorder due to insufficient pancreatic secretion and impairment of the small intestine.

DIAGNOSIS OF PD

The diagnosis is mainly based on clinical data. The patients complain about murmur and inflation of the bowel and diarrhoea. Absorption disorder symptoms are characteristic: body weight loss, hypovitaminosis signs (cutaneous changes, gingival bleeding, brittleness of nails, loss of hair), convulsions in the gastrocnemius muscles and bone pain due to mineral metabolism disorder. The clinical picture may be complemented by symptoms of liver and pancreas impairment as well as mental disorders manifested by the hypochondriac, hysteric and depressive syndromes.

PD patients are revealed to have hypoproteinaemia due to the reduced albumin levels, impairment of the carbohydrate and mineral metabolism.

POST-RESECTION ANAEMIA (PA)

Revealed in 10-15% patients after gastric resection. PA emerges in two variants:

– hypochromic iron-deficiency anaemia

– hyperchromic B12-deficiency anaemia

CAUSES:

iron-deficiency anaemia: haemorrhage from peptic anastomotic ulcers and erosions of the mucosa in reflux gastritis, which are often time remain unrevealed. The development of this variant of anaemia is facilitated by impairment of ionisation and resorption of iron due to accelerated passage through the small intestine and atrophic enteritis.

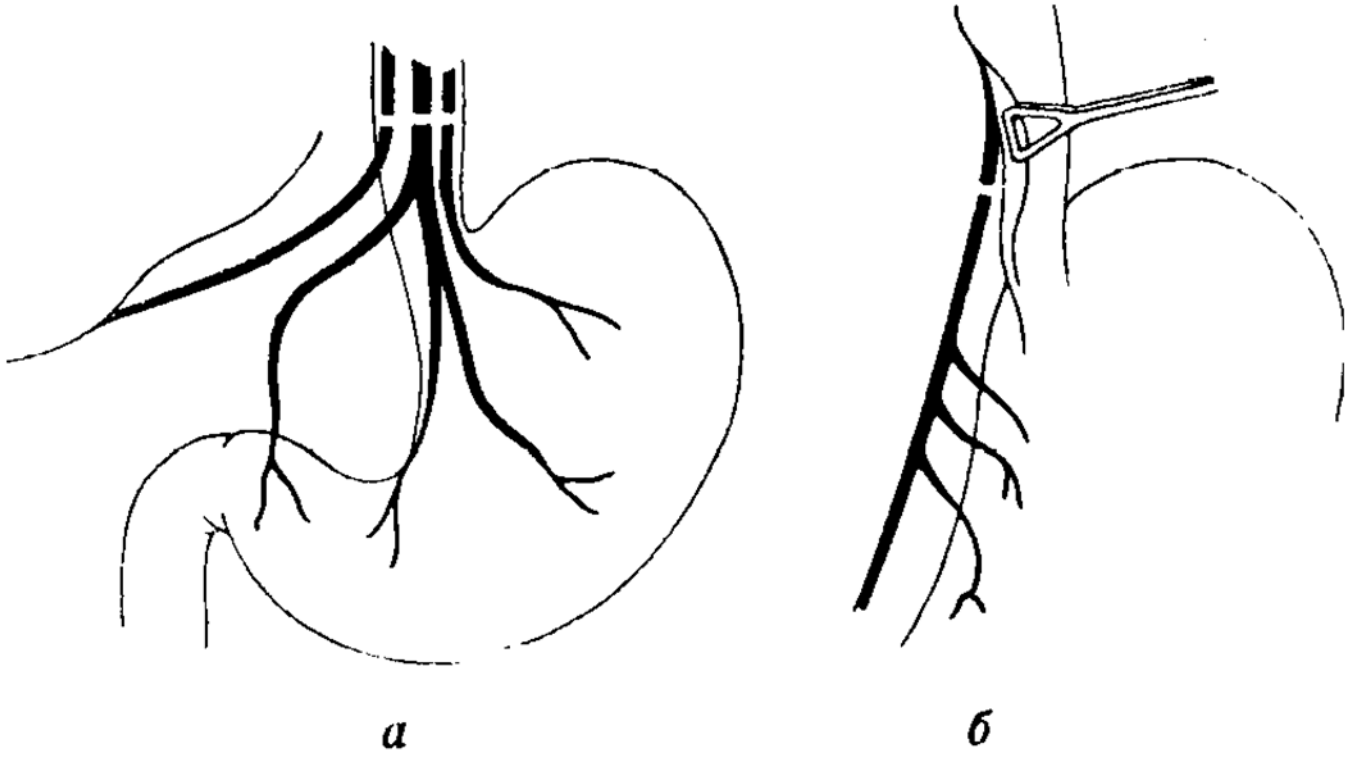
Hyperchromic anaemia

After resection of the stomach, the function of internal function production declines, which severely decreases utilization of vitamin B12 and folic acid. It is also contributed to by the change in the intestinal microflora composition. The deficit of these vitamins leads to megaloblastic haemorrhage and development of hyperchromic anaemia.

DIFFERENTIAL DIAGNOSIS OF PA

Based on assessment of peripheral blood and spinal cord. In peripheral blood, it is possible to observe hypochromia or erythrocytes and microcytosis in iron-deficiency anaemia as well as hyperchromia and macrocytosis in B12-deficiency anaemia.

POST-VAGOTOMY SYNDROMES



Dissection of the anterior (left) of n. vagus *(а)* and posterior (right) n. vagus *(б)*

DYSPHAGIA

A complication characteristic to the early postoperative period (appears every two weeks and disappears on its own after 1-2 months). However, it may develop within a later time period.

The frequency of dysphagic disorders after vagotomy amounts to 3-17% of the operated patients.

CAUSES:

Trauma and oedema of the oesophageal wall.

Denervation of distal part of the oesophagus leads to temporary impairment of the cardia function.

Development of dysphagia in later terms after vagotomy is associated with reflux oesophagitis and cicatricial changes in the surgery area.

DIAGNOSIS

In mild dysphagia, radiological and endoscopic methods usually do not make it possible to reveal any pathological changes in the oesophagus.

In patients with more pronounced and persistent dysphagic disorders, radiological examination reveals dilation and sharp narrowing of the distal part of oesophagus and endoscopic examination reveals reflux oesophagitis.

GASTRIC STASIS

May develop after all types of vagotomy. Even after selective proximal vagotomy, 1.5-10% of the patients show delayed emptying of the stomach.

TYPES OF GASTRIC STASIS

Mechanical: **may be conditioned by the obstruction of the gastric outlet in the area of pyloroplasty or gastroenteroanastomosis.**

Functional: **may appear due to impairment of the rhythm of the peristaltic wave of stomach, which leads to motions that are not coordinated time- and direction-wise, as well as mechanical overstretching of its wall.**

CLINICAL PICTURE

Motor and evacuation disorders of the stomach are manifested by

sensation of fullness in the epigastrium,

nausea

intermittent pain

In severe gastric stasis

almost permanent pain and sensation of heaviness in upper abdomen

profuse vomiting by congestive gastric contents

Vomiting alleviates the patient's condition, which drives the patient to provoke it.

DIAGNOSIS OF GASTRIC STASIS

Radiological examination reveals delay of the contrast mass in the stomach, torpid and superficial peristalsis as well as enlargement of the stomach

RECURRENCE OF ULCERATIVE DISEASE AFTER VAGOTOMY

Including after SPV, observed in 10-30% of the cases.

The cause of ulcer after vagotomy is usually insufficient denervation of the stomach and remaining high production of HCl. It promotes the recurrence and impairment of stomach evacuation.

DIAGNOSIS OF ULCER RECURRENCE

After vagotomy, it presents a non-trivial task while it is asymptomatic in 30-50% of the cases.

Therefore, it is necessary to examine the ulcerative disease patients after vagotomy two times per year, interchanging endoscopic and radiological techniques.

DIARRHOEA

Occupies a notable place among vagotomy complications. Most often, diarrhoea is observed in stem vagotomy with drain surgeries and is associated with abnormally rapid dumping of the liquid from the stomach into the small intestine, the change in composition of the small intestine flora, disorder of secretion and absorption of bile acids, which leads to intestinal hypermotility.

CAUSES OF DIARRHOEA

Associated with achlorhydria

impairment of exocrine function of the pancreas

atrophic changes in the mucosa of the small intestine.

CLINICAL PICTURE

Diarrhoea is manifested by:

liquid stool 3-5 times per fay. Sometimes diarrhoea is provoked by dairy products and carbohydrates, more often diarrhoea appears unexpectedly, accompanied with large gas production and manifested abdominal murmur.

The urgency of such episodes causes significant inconvenience to the patient. Abnormal defecation pattern is observed for several days, such cycles may repeat 1-2 times per month.

DUMPING SYNDROME AFTER SPV

Observed in 2-9%, drain surgeries increase its frequency to 10-34%. However, severe forms of the syndrome are observed very rarely.

PATHOGENESIS OF POST-VAGOTOMY DUMPING SYNDROME

In essence, it does not differ from the post-resection pathogenesis:

rapid emptying of the stomach

overfilling of the small intestine with food

hyperosmolarity due to rapid breakdown of polysaccharides

profuse production of biologically active substances in the bowel that accelerate peristalsis and affect the cardiovascular system.

DIAGNOSIS

Diagnosis of post-vagotomy dumping syndrome does not present difficulties and is mainly based in analysis of the patient's complaints.

PREVENTION OF DISEASES OF OPERATED STOMACH

An important place in prevention of OSD is occupied by:

strict adherence to the indications to surgical treatment,

optimal choice of surgery technique

technically correct rehabilitation procedures.

STAGE I

In the preoperative preparation period, the ulcerative disease patients are given antiulcer therapy (diet no.1, antacids, antisecretory pharmaceuticals, eradication of H. pylori infection), in gastric resection, they undergo general conditioning and symptomatic therapy. All this makes it possible to perfoirm surgery in more favourable conditions.

STAGE II

In the early postoperative period, **the patient is given bowel rest and active aspiration of gastric contents. As early as at this stage, it is possible to introduce enteral feeding through the probe. Starting from days 2-4, in absence of congestion signs in the stomach, the patient must adhere to a special diet. The feeding regimen is planned according to the principle of gradual increase of the load on the gastrointestinal tract and inclusion of a sufficient amount of protein. By the end of week 1, if the surgery did not provide for a significant decrease of acid production in the stomach, antacids are prescribed as well as, if necessary, H2-receptor blockers or proton pump inhibitors.**

STAGE III

Complex therapy aimed at compensation of the functions of different organism systems impaired by surgery, begins 2 weeks after the surgery and lasts 2-4 months. An important component of the complex treatment in this period is the diet, the aim of which is to facilitate the decrease of the inflammation process in the gastrointestinal tract, activate reparation as well as to prevent development of dumping reactions, hypoglycaemia, afferent loop syndrome, etc.

This is a physiologically valid diet with high content of protein (140g), normal contents of lipids (110-115g) and carbohydrates (380g), with restriction of mechanical and chemical irritants of the mucosa and the receptor apparatus of the gastrointestinal tract. High-melting fat, easily digestible carbohydrates and fresh milk are excluded. The patients must adhere to the split meal regimen. According to the indications, pharmaceutical therapy is continued: antacids, agents for peristalsis normalisation (Motilium, Imodium in diarrhoea, duspalatin).

STAGE IV

Later, even if the patient does not demonstrate signs of the disease of operated stomach, it is recommended to adhere to preventive measures in nutrition for 2-5 years: split meals (4-5 times per day), restriction of products and dishes containing easily absorbed carbohydrates and fresh milk. The ration is to be sufficiently diverse with consideration for the individual intolerance of products. As a rule, patients with favourable outcomes of the surgery do not require any pharmaceutical aid.

TREATMENT OF DISEASES OF OPERATED STOMACH

Treatment of OSD may be conservative and surgical. Diet therapy occupies the leading place in conservative treatment of OSD. The food must be diverse, rich in calories and vitamins, normal contents of lipids and complex carbohydrates with sharp restriction of simple carbohydrates. It is also advised to account for individual intolerance of products and dishes.

The patients usually show good tolerate of boiled meat, non-fast sausage, chops from lean meat, fish dishes, soups cooked with gravy meet or fish broth, fermented milk products, vegetable salads and Russian vinaigrette with vegetable oil. The most poorly tolerated food products are sugar, milk, sweet tea, coffee, stewed fruit drinks, honey, sweet liquid milk porridge, sweet dough bakery, especially hot. Food intake is to be split between meals, no less than 6 per day.

Such a diet and feeding regimen are usually acceptable in all types of OSD.

In dumping syndrome, it is recommended to start eating from solid dishes. it is advisable to lie in bed after feeding or to assume a half-lying position in a chair for 30 minutes. Pharmaceutical therapy in dumping syndrome is to be aimed at main links of its pathogenesis.

In general, the efficacy of pharmaceutical therapy in dumping syndrome patients is quite low, due to which sensible dietary guidelines prove more useful.

The specific feature of hypoglycaemic syndrome treatment is the necessity to arrest the hypoglycaemic attacks. The patient is to carry a piece of sugar with them or dried bread in order to arrest first signs of hypoglycaemia. Severe attacks of hypoglycaemia are not characteristic to this syndrome, thus intravenous injection of 40% glucose solution is rarely required.

In order to decrease the symptoms of afferent loop evacuation disorder, the patients should be recommended to lie down on their right side after food intake. Along with dietary measures, afferent loop syndrome manifestations may be reduced through repeated gastric lavage and drainage of the afferent loop using an endoscope.

For liquidation of the inflammatory component and for sanitation of the blind loop from the developed microbial flora antibacterial therapy is indicated.

In peptic anastomotic ulcer or recurrent ulcer after vagotomy, antiulcer therapy is prescribed, the principles of which do not differ from those for recurrence of the ulcerative disease.

In gastric stasis developed in the early period after vagotomy, the treatment is to begin from suction of the gastric content through the nasogastric probe. Functional gastric stasis patients are further recommended to restrict the volume of liquid intake, while solid food stimulates peristalsis in such cases. Improvement of gastric contents evacuation is achieved using prokinetics (Motilium). In absence of therapy efficacy, anastomotic bougienage is indicated.

Good therapeutic effect in post-vagotomy diarrhoea is provided by 1-2 capsules of Imodium (loperamide) per os 1-2 times per day. The preparation slows intestinal propulsion and increases the tone of sphincters. To improve food digestion, pancreatic enzymes are used, preparations that do not contain bile acids are tolerated better (Creon, Mezym forte).

In absence of prospects in conservative treatment of OSD, the decision regarding surgical intervention is made. The indications to the surgery are severe dumping syndrome and organic afferent loop syndrome.

PREVENTIVE MEDICAL OBSERVATION

Patients with post-resection and post-vagotomy disorders are subject to preventive medical observation. It includes systematic control over the health condition, periodic examination of the patient and performance of therapeutic and preventive procedures.

Most of the OSD develop in the early period after surgery and disappear within the first year.

These include:

mild and, partially, moderate dumping syndrome

dysphagia

functional gastrostasis

mild forms of post-vagotomy diarrhoea

Patients with such disorders, as a rule, do not require specific treatment and their work capacity does not decline significantly. Within the first 6-12 months after the surgery, such patients are to undergo observation. It is recommended for them to restrict products leading to digestive disorder and dumping syndrome (sweet drinks, fresh milk, etc.), eat slowly with proper mastication of food.

Patients with post-resection and post-vagotomy disorders of mainly functional nature are indicated to undergo sanatorium-resort treatment. The patients are referred to local sanatoriums no earlier than one month after surgery and to resorts with drinking mineral water and therapeutic muds no earlier than two months after the surgery. Sanatorium-resort treatment is contraindicated in pronounced in severe nutrition decline, anaemia, severe organic diseases of operated stomach.

The patients may be withdrawn from preventive medical observation in absence of clinical manifestations of the disease for three months and in positive general condition and state of health.

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Thank you for attention!