

**МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ**

**УЧРЕЖДЕНИЕ ОБРАЗОВАНИЯ  
«ГОМЕЛЬСКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ»**

**Кафедра хирургических болезней № 1**

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# **ХРОНИЧЕСКИЙ ПАНКРЕАТИТ И ЕГО ОСЛОЖНЕНИЯ**

**Учебно-методическое пособие  
для студентов 5 курса факультета по подготовке специалистов  
для зарубежных стран медицинских вузов**

## **CHRONIC PANCREATITIS AND IT'S COMPLICATIONS**

**The educational methodical work  
for 5-th year students of the Faculty of preparation of experts  
for foreign countries of medical higher educational institutions**

**Гомель  
ГомГМУ  
2014**

УДК 616.381-002.7-06(072) = 111

ББК 54.574.2,43(2Англ.)я73

А 54

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А 54 Хронический панкреатит и его осложнения: учеб.-метод. пособие для студентов 5 курса факультета по подготовке специалистов для зарубежных стран медицинских вузов = Chronic pancreatitis and it's complications: The educational methodical work for 5-th year students of the Faculty of preparation of experts for foreign countries of medical higher educational institutions / В. Анджум, А. А. Призенцов, А. Г. Скуратов. — Гомель: ГомГМУ, 2014. — 24 с.

ISBN 978-985-506-633-1

Учебно-методическое пособие содержит учебный материал по теме «Хронический панкреатит и его осложнения». Соответствует учебному плану и программе по хирургическим болезням для студентов высших медицинских учебных заведений Министерства здравоохранения Республики Беларусь.

Предназначено для студентов 5 курса факультета по подготовке специалистов для зарубежных стран медицинских вузов.

Утверждено и рекомендовано к изданию научно-методическим советом учреждения образования «Гомельский государственный медицинский университет» 30 декабря 2013 г., протокол № 10.

**УДК 616.381-002.7-06(072) = 111**

**ББК 54.574.2,43(2Англ.)я73**

**ISBN 978-985-506-633-1**

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«Гомельский государственный  
медицинский университет», 2014

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## SURGICAL ANATOMY OF PANCREAS

The word pancreas is derived from the Greek «pan» (all) and «creas» (piece of meat). Pancreas is a retroperitoneal organ and was considered as a cushion for the stomach and shield for major vessels (aorta, inferior cava vein and portal vein).

Pancreas is situated retro-peritoneally at the level of L1–L2 and location at anterior abdominal wall is about 5–10 cm above the umbilicus. The gland weighs 70–90 gm, length 15–20 cm, width 3–4 cm and thickness is 2–3cm. It is divided into head, neck, body and tail. Head is disc shaped and lies within the curve of duodenum. A part of it extends to the left behind the superior mesenteric vessels, called uncinata process. Neck is a narrow part, which connects head to body. Behind the neck, at its upper border, the superior mesenteric vein joins the splenic vein to form the portal vein. Body runs upward and to the left across the midline. Tail passes forward in lienorenal ligament and comes in contact with hilus of spleen.

Main pancreatic duct is formed by ventral bud duct (duct of head) in the head of pancreas, which fuses with dorsal duct (duct of body and tail) in the pancreatic head and then runs posteriorly and inferiorly to lie next to and eventually join with bile duct at ampulla of Vater in the posteromedial wall of 2<sup>nd</sup> part of duodenum.

### **Chronic pancreatitis:**

It is a continuing inflammatory and degenerative process of the glandular tissue of the pancreas, leading to fibrosis and sclerosis of the parenchyma of the organ with a decrease in its exocrine and endocrine function.

### **Epidemiology:**

Frequency of chronic pancreatitis (CP) in GIT diseases is from 5.1 to 9%. Male — female ratio is about 4:1 and the mean age of onset is about 40 years of age. Incidence — 7.6 cases per 100 thousand people per year, prevalence — 50–70 patients per 100 thousand of population.

It is common in people aged 40–60 years.

### **Aetiology:**

- High alcohol consumption accompanied by a protein- & fat-rich diet.
- Diseases of the bile ducts (choledocholithiasis, papillostenosis).
- Pancreatic duct obstruction, due to stricture or occlusion.
- Peptic ulcer disease.
- Hyperparathyroidism.
- Atherosclerosis of the pancreas.
- Prolonged use of some medications (steroids, sulfonilamides, etc.).
- Viral infection (incl. Coxsackie, hepatitis B, and C).
- Hyperlipidemia, inadequate protein diet.
- Cystic fibrosis.
- Idiopathic.
- Hereditary pancreatitis.
- Infantile malnutrition.

Despite the variety of the causes of chronic pancreatitis, most often disease develops and progresses more rapidly as a result of alcohol abuse, which has led to highlight «alcoholic» and «non-alcoholic» pancreatitis.

#### **Theories of chronic alcoholic pancreatitis:**

1. The primary effect of alcohol on the external secretion of the pancreas to produce the protein precipitates, which obstruct the pancreatic duct and secondary damage to the acinar cells.

2. A direct toxic effect of alcohol on acinar cells causes their fatty degeneration, resulting in the loss of the ability to synthesize enzymes and periacinar fibrosis.

3. Aggressive effect of alcohol on the duodenal mucosa causes edema (stenosis) of major duodenal papilla (MDP) and impaired outflow of pancreatic secretion.

4. Under the influence of alcohol detoxification function of the liver becomes impaired, which leads to the formation of free radicals of oxygen, which damage the pancreas.

### **CLASSIFICATION OF CHRONIC PANCREATITIS**

#### **ICD - 10**

- K85 Acute pancreatitis.
- K86 Other diseases of pancreas.
- K86.0 Chronic pancreatitis, alcoholic etiology.
- K86.1 Other chronic pancreatitis.
- K86.2 Pancreatic cysts.
- K86.3 Pseudocyst of the pancreas.
- K86.8 Other specified diseases of the pancreas.
- K86.9 Unspecified disease of the pancreas.
- K87\* Lesions of the gallbladder, biliary tract and pancreas, in diseases classified elsewhere.

**The Marseilles-Roman classification** (1988) has three main types of chronic pancreatitis.

**I.** Chronic calcify pancreatitis. The most common cause — alcohol, as well as hyperparathyroidism.

**II.** Chronic obstructive pancreatitis. Observed in severe narrowing of the main pancreatic duct or its major branches, or major duodenal papilla. Causes - gallstone disease, trauma, tumors, congenital anomalies.

**III.** Chronic parenchymal-fibrotic (inflammatory), pancreatitis.

#### **The etiological classification of chronic pancreatitis (TIGAR-O)**

- **T**oxic-Metabolic (alcohol, nicotine, hypercalcaemia, renal failure, drug-induced, toxic).
- **I**diopathic (early start, late start, tropical).
- **G**enetic (hereditary mutation of trypsinogen, deficiency of alpha-1-antitrypsin).
- **A**utoimmune (isolated, syndromic (associated with Sjogren's syndrome, nonspecific inflammatory diseases of colon, primary biliary cirrhosis).

- **Recurrent and Severe** (recurrent post-necrotic and severe acute pancreatitis, ischemic).

- **Obstructive** (biliary, duct obstruction, pathology of MDP and papillary diverticulum of duodenum).

**Pathogenic classification:**

- **Primary CP** — inflammatory process right from the start develops in the pancreas.

- **Secondary CP** – develops gradually on the background of gastrointestinal diseases (caused by disorders of adjacent organs).

Associated with this pathogenic forms of CP:

1. Autonomous (primary).
2. Biliary pancreatitis.
3. Papillo-pancreatitis.
4. Duodeno-pancreatitis.

**Variants of morphological changes in pancreas:**

- **Parenchymal CP** — main pathological changes are localized in the acini or interlobular connective tissue;

- **Ductal CP** — main pancreatic duct is expanded, deformed and determined as «Chain of Lakes» (with or without lithiasis of Wirsung duct).

- **Papilloduodeno Pancreatitis** –in the case of hyperplasia of MDP and duodenal wall around it, is characterized by a uniform extension of the main pancreatic duct along its entire length.

**Clinical forms of CP (M. I. Kuzin, 1985):**

1. Chronic recurrent pancreatitis (the most common form) — is characterized by periods of exacerbation with pain and periods of remission, during which the patient remains relatively well.

2. Chronic painful pancreatitis — constant pain.

3. Chronic latent (silent), pancreatitis — at the first place — impairment of the exocrine and endocrine function.

4. Pseudotumor inflammatory CP — with a primary lesion of the pancreatic head, clinical features are much like pancreatic head cancer with obstructive jaundice.

**According to the severity:**

1. Mild: exacerbation of 1–2 times a year, the function of pancreas is preserved.

2. Moderate: exacerbation of 3–4 times a year with a moderate dysfunction.

3. Severe (terminal, cachectic stage): frequent exacerbations, severe impairment of exocrine (Pancreatogenic persistent diarrhea, progressive weight loss, poly hypovitaminosis) and endocrine (secondary diabetes) functions.

## CLINICAL FEATURES OF CHRONIC PANCREATITIS

1. Abdominal pain. Epigastric region, often encircling character, becomes severe 1.5-2 hours after eating or drinking alcohol. Might be recurrent and permanent. In a later stage as atrophy and fibrosis of the pancreas develops pain decreases and may even disappear.

Pain syndrome is associated with:

- a) impairment of the outflow of pancreatic juice in the duodenum and hypertension in the pancreatic ducts and their subsequent extension;
  - b) inflammatory and cicatricial changes both in the pancreas and in parapancreatic tissue in which the nerve ends get constant pathological stimulation leads to the development of persistent pain;
  - c) compression of the common bile duct;
  - d) compression of the duodenum.
2. The progressive weight loss (associated with exocrine insufficiency, abstinence from eating because of the pain).
  3. Pancreatic dyspepsia. Loss of appetite, aversion to fatty foods, belching, nausea, vomiting, flatulence.
  4. Disorder of stool: first constipation, then diarrhea, or unformed stool.
  5. Steatorrhea and kreatoreya with pieces of undigested food - the secretion of lipase and trypsin is lower than 10 % of normal (in severe CP).
  6. Polyhypovitaminosis symptoms: dry, peeling skin, glossitis, stomatitis, etc.

## COMPLICATIONS OF CHRONIC PANCREATITIS

Occurs in approximately 30 % of patients. The most common are:

- Cysts;
- Fistulas;
- Obstructive jaundice;
- Wirsungolithiasis;
- Duodenal obstruction;
- Segmental portal hypertension.
- Pancreatic ascites, pleural effusion (a complication of fistulas).
- Intraductal bleeding
- Secondary diabetes.

### **Biliary hypertension (cholestasis)**

— In 10–30 %. Characterized by persistent or recurrent jaundice due to compression of the distal common bile duct by pancreatic head (similar to the pancreatic head cancer).

### **Duodenal obstruction**

From the compensated form (heaviness in epigastric region, belching, loss of appetite) to decompensated form (symptoms of high intestinal obstruction).

**Segmental portal hypertension** — due to fibrotic changes in distal pancreas, which can result in thrombosis or obliteration of the splenic vein. Develops splenomegaly and hypersplenism, varicosis of veins of omentum and gastric cardia (sometimes leads to bleeding), ascites.

**Pancreatic ascites and pleural effusion** — manifestation of internal pancreatic fistula (pancreatic-abdominal and pancreatic-pleural).

**Pancreatic bleeding** — is rare. A rupture of aneurysm in the pancreatic duct. Pain syndrome, clinical features of bleeding from the upper gastrointestinal tract.

**Diabetes** observed in more than 30 % of patients, develops about after 10–12 years from the start of CP. Not only insulin, but glucagon deficiency is also present. Neuropathy is often and angiopathy is rare.

## DIAGNOSIS OF CHRONIC PANCREATITIS

### On the base of:

- complaints;
- medical history;
- physical examination;
- laboratory reports;
- instrumental methods.

## LABORATORY DIAGNOSIS OF CHRONIC PANCREATITIS

- Uncomplicated CP does not cause marked changes in laboratory parameters.
- In exacerbation of *CP*: increased amylase and lipase in blood and urine, leukocytosis with left shift of formula.
  - In alimentary exhaustion, with internal pancreatic fistula: anemia, hypo- and dysproteinemia (increasing of b — and y — globulin, decreasing of albumin).
- The first signs of biliary hypertension on pre-jaundice stage: increased alkaline phosphatase and gamma-glutamyl: in progression — bilirubin increase.
- In duodenal obstruction as it progresses: signs of haemoconcentration, hypokalemia, metabolic alkalosis, increased urea and creatinine.
- In Splenic vein thrombosis: signs of hypersplenism (erythro-, leuko- and thrombocytopenia).

For the differential diagnosis of CP and pancreatic cancer: determining the level of tumor markers — carbohydrate antigen CA-19-9 and carcinoembryonic antigen (CEA).

### Investigation of external secretion of pancreas:

**A. Direct / Invasive study of pancreatic secretion** with duodenal intubation or by Wirsung duct intubation through the MDP, in external fistula — a study of the fistula discharge. Determine the amount of juice, the concentration of bicarbonate and the key enzymes (amylase, lipase, trypsin) on an empty stomach and after stimulation.

• **Lundh test:** This is a direct test of pancreatic function in which duodenal contents are collected for two hours following a meal containing carbohydrate, protein and fat. Low enzymic activity — amylase, trypsin or lipase — indicates pancreatic insufficiency. It is less informative than the secretin / CCK-PZ test because it does not differentiate between a decreased pancreatic response due to a reduced ability to secrete, or a reduced ability to transfer the stimulus, neurally or hormonally.



• **Secretin / CCK-PZ test:** The secretin / cholecystokin-pancreozymin test may be used to assess pancreatic exocrine function. It is an invasive test and considerable skill is required to collect duodenal juice uncontaminated by gastric juice.

A double-lumen radio-opaque tube is passed following an overnight fast. One opening enables the collection of gastric secretions; the other, of duodenal secretions. Three specimens of duodenal secretions are collected:

- basal secretion;
- following intravenous secretin;
- following intravenous CCK-PZ.

The volume, pH, amylase and bicarbonate contents of all samples are measured. In the normal person:

- secretin administration primarily stimulates bicarbonate secretion;
- cholecystokinin primarily stimulates pancreatic enzyme secretion.

In cases of exocrine pancreatic failure, bicarbonate secretion is lost early and enzyme secretion lost later. Abnormal results are obtained in chronic pancreatitis, enzymic activity and bicarbonate falling before there is any obvious reduction in the volume of juice. In pancreatic carcinoma, results may also be abnormal. A marked feature is a low volume of juice especially when the tumor is at the head of the pancreas producing obstruction. Secretin may cause a paradoxical increase in gastrin levels if the patient has a gastrinoma.

**B. Indirect / non-invasive methods:**

• **Coprological study** — low information test. Reveals the undigested fats and undigested muscle fibers.

• **NBT-PABA-test** (N-benzoyl-tryrosyl para-aminobenzoic acid) — more modern technique and most widely used test of pancreatic exocrine insufficiency in the UK with a specificity of 95 % and a sensitivity of 80–90 %.

• NBT-PABA is hydrolysed by pancreatic chymotrypsin to PABA which is then absorbed and excreted in the urine. To control for variation in PABA absorption a trace quantity of C14-labelled PABA is given with the bulk unlabelled NBT-PABA.

• Study the concentration of pancreatic **elastase-1** in the stool, which passes through the bowel and remains in an unchanged form.

• **The oral test with 13C-glycerides**, the degree of cleavage in the gastrointestinal tract which is judged by the level of carbon-13 in the exhaled air.

**Investigation of internal secretion of pancreas:** study of blood and urine glucose, glucose tolerance tests.

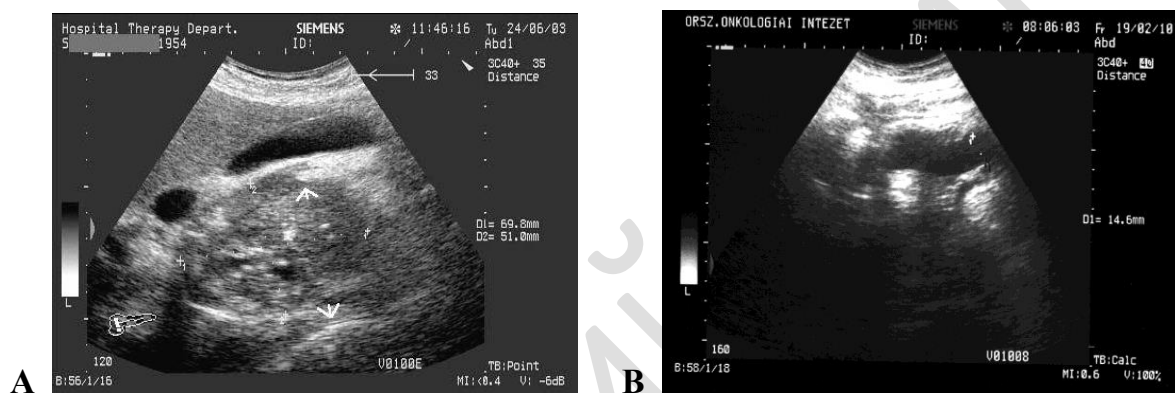
**Instrumental methods of diagnosis of CP:**

**Classical (trans-abdominal) ultrasound** — the first line of diagnosis (pancreas size and structure, diameter of pancreatic duct and stones in it), state of other organs (Figure 1).

**The main echographic signs of chronic pancreatitis are:**

• heterogeneity echostructure of pancreas mainly due to sites of increased echogenicity;

- uneven expansion of the main pancreatic duct;
- calcifications in the parenchyma and stones in the pancreatic duct;
- changes in the size of the pancreas (increased in an exacerbation due to edema or decrease as a result of atrophy and fibrosis in the later stages of the disease);
- uneven echogenicity of the pancreas;
- uneven, sometimes «chippy», contour of the pancreas;
- tenderness on probe pressure or palpation of the pancreas under ultrasound guidance;
- additional sonographic signs of chronic pancreatitis: compression of the major vessels, common bile duct, presence of retention cysts and pseudocysts;
- no changes in pancreas at early stages of chronic pancreatitis on usual transabdominal U.S.G.

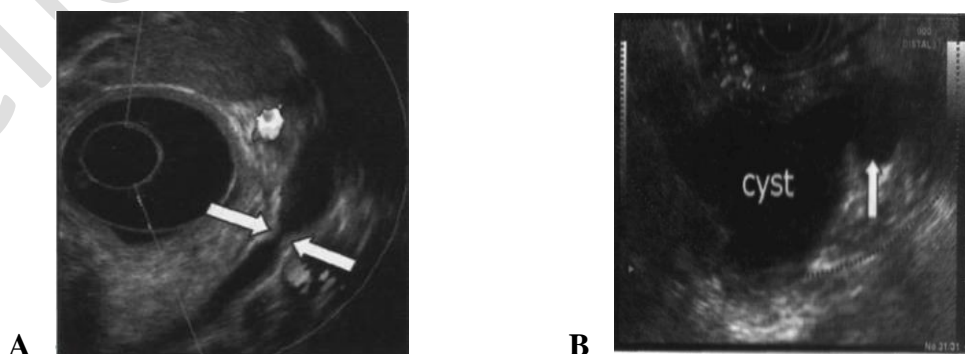


**Figure 1 — Typical ultrasound of pancreas:**

**A — Ultrasound image of the pancreatic head during exacerbation of chronic pancreatitis. Oblique scan of the right hypochondrium. Head dimensions are increased up to 5 cm (indicated by arrows). Heterogeneous echostructure of parenchyma due to hypo- and hyperechoic foci, few calcifications.**

**B — extension of Wirsung duct with atrophy of the pancreatic parenchyma.**

**Endoscopic ultrasound (endosonography, EUS) —** new highly informative noninvasive test for pancreas and bile duct diseases (not less informative than CT and ERCP). To perform this test requires special equipment (endoscope with ultrasound sensor). Sensitivity — 85 %, specificity — 67 % (figure 2).

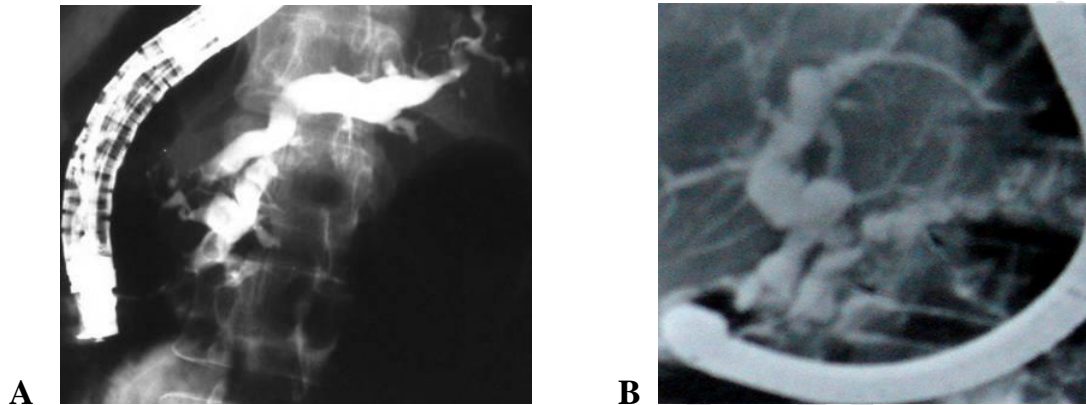


**Figure 2 — Endosonography of pancreas:**

**A — constriction of Wirsung duct; B — cyst of pancreatic head**

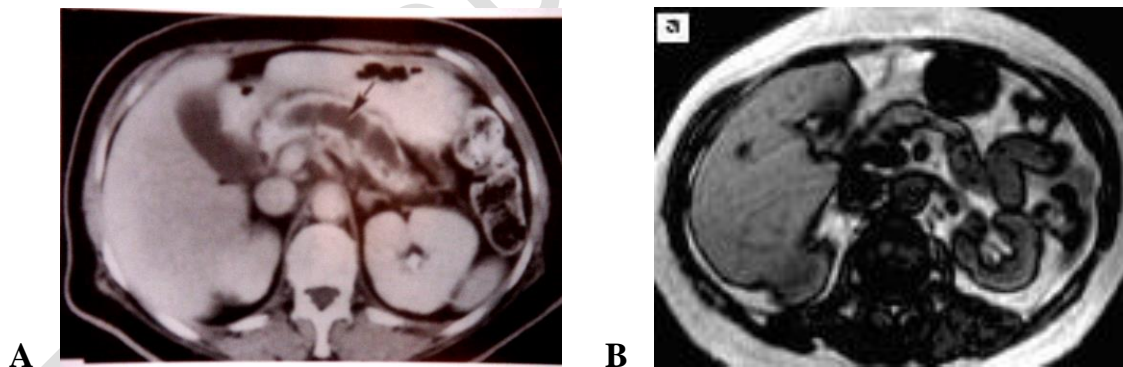
**Plain radiographs of the abdomen** — is rarely used, it is possible to identify calculi and calcification of the pancreas.

**Endoscopic retrograde cholangiopancreatography (ERCP)** — assessment of the Wirsung duct and its branches, bile ducts. It's an invasive method, there can be complications (acute pancreatitis, perforation of the duodenum) Sensitivity — 66–89 %, specificity — 89–100 % (figure 3).



**Figure 3 — ERCP: A — even expansion of Wirsung duct; B — uneven expansion of Wirsung duct-type «Chain of Lakes» with dilatation of the bile ducts**

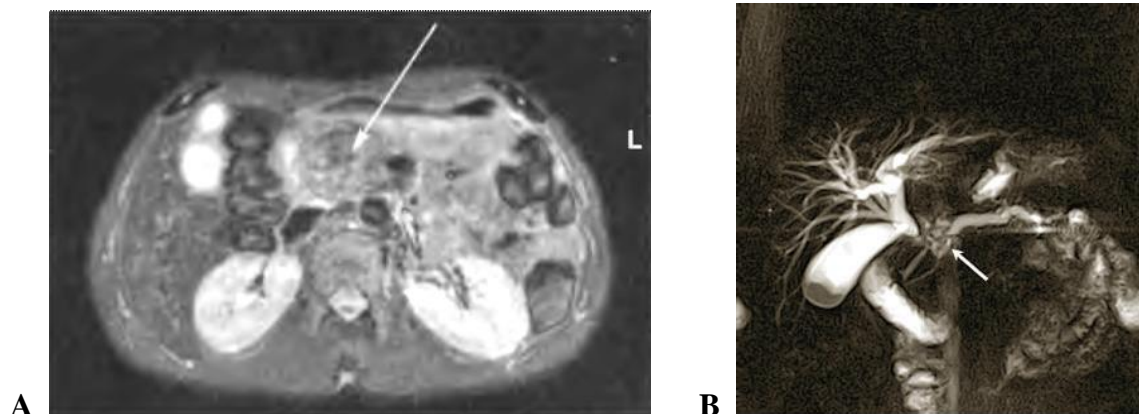
**Computed tomography (CT):** (with and without contrast intensification), figure 4.



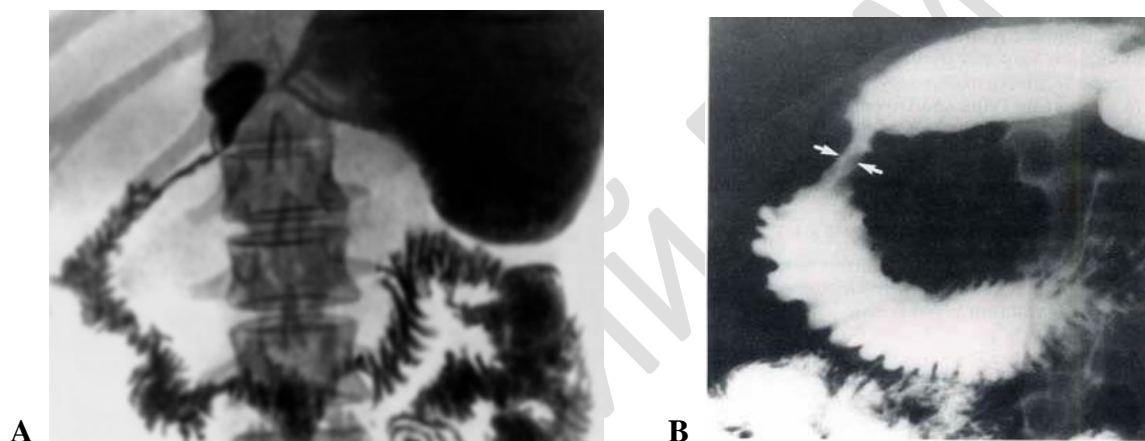
**Figure 4 — CT of the abdomen: A — expansion of the pancreatic duct with atrophy of the tissue around it; B — atrophy of the pancreas**

**Magnetic resonance imaging (MRI) and magnetic resonance cholangiopancreatography (MRCP)** — non-invasive and highly informative method, but the cost of the study is high (figure 5). The sensitivity and specificity is almost 90–95 %.

**Contrast X-rays of the stomach and relaxational duodenography.** Revealed indirect signs of CP compression and displacement of the stomach and duodenum, increasing retrogastral space of more than 5 cm, the acceleration or deceleration of the passage of barium suspension (figure 6).



**Figure 5 — MRI in chronic pseudotumor pancreatitis:**  
**A — the arrow indicates the enlarged pancreatic head; B — pseudotumor pancreatitis with compression of the common bile duct and biliary hypertension**



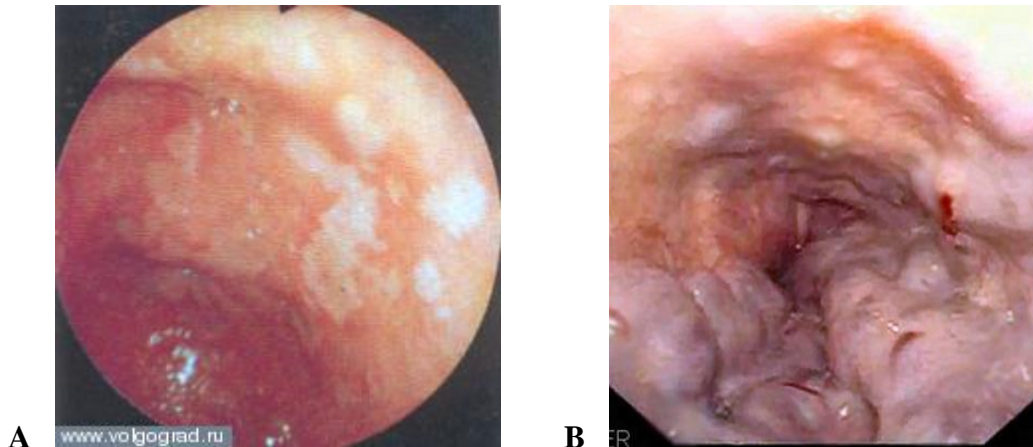
**Figure 6 — Contrast radiography of the stomach and duodenum:**  
**A — compression of the antrum and duodenum, spreads the duodenal loop;**  
**B — local compression of duodenum.**

**Angiographic study** (Celiaco- and mesentericography) — is now rarely used, mainly for the detection of vascular causes of CP (atherosclerosis of the pancreas).

**Radioisotope study** (with radioactive technetium, selenium) — for differential diagnosis of cancer pathology. Identify lesions with accumulation of specific material.

**Needle biopsy:** local involvement of the pancreas under ultrasound, CT scan or endoscopic ultrasound. Apply for morphological verification of diagnosis, mainly for suspected malignancy. However, the use of percutaneous fine-needle biopsy under ultrasound guidance has a high rate of false-negative results, and often distinguish these diseases is possible only after intraoperative biopsy and even pancreatoduodenal resection.

**Fibroesophagogastroduodenoscopy (Endoscopy):** In chronic pancreatitis can identify compression on the posterior wall of the stomach, deformation of the stomach and duodenum, erosion, signs of lymphostasis in parapancreatic region in the form of a whitish areas — lymphangioectasy, inflammatory changes of MDP, varicose veins of the esophagus and stomach in portal hypertension (complication of CP (figure 7)).



**Figure 7 — Endoscopy:**

**A — whitish areas - lymphangiectasy; B — varicose veins of the esophagus**

### **DIFFERENTIAL DIAGNOSIS OF CHRONIC PANCREATITIS**

The most frequent diseases of the intra and extra peritoneal organs, which need to carry out differential diagnosis of Chronic Pancreatitis, are given below:

- Acute cholecystitis.
- Cholangitis.
- Peptic ulcer disease.
- Acute appendicitis.
- Typhlitis.
- Myocardic infarction.
- Pneumonia.
- Osteochondrosis.
- Pyelonephritis.
- Acute intestinal obstruction.
- Diverticulitis.
- Ischemia of intestine.

### **TREATMENT OF CHRONIC PANCREATITIS**

Each exacerbation of CP should be considered as acute pancreatitis with the appropriate treatment tactics.

#### **Conservative therapy of CP:**

In non-exacerbation period: management of pain, correction of endocrine insufficiency. Diet, avoiding alcohol, enzymes, non-narcotic analgesics, antispasmodics.

#### **Indications for surgical treatment of CP:**

- Persistent pain with no effect of conservative therapy.
- Secondary CP due to cholelithiasis, peptic ulcer disease and duodenostasis.
- Calcification of the pancreas and wirsungolithiasis with impaired patency and expansion of Wirsung duct.

- Obstructive jaundice with tubular stenosis of the terminal part of the common bile duct.

- Duodenal obstruction
- Segmental portal hypertension.
- Chronic cysts.
- Pancreatic fistula, more than 3 months.
- Pancreatic ascites, pleural effusion.
- Failure to pre-operative exclusion of the malignancy.

**Surgical treatment of CP** can't eliminate the degenerative changes in the pancreas, and is directed to the treatment of complications and elimination of pain.

### **SURGICAL TREATMENT OPTIONS OF CHRONIC PANCREATITIS:**

#### **Operation on the biliary tract (in cholepancreatitis):**

- cholecystectomy, choledocholithotomy;
- overlay of biliodigestive anastomoses;
- papillosphincterotomy and papillosphincterowirsungotomy.

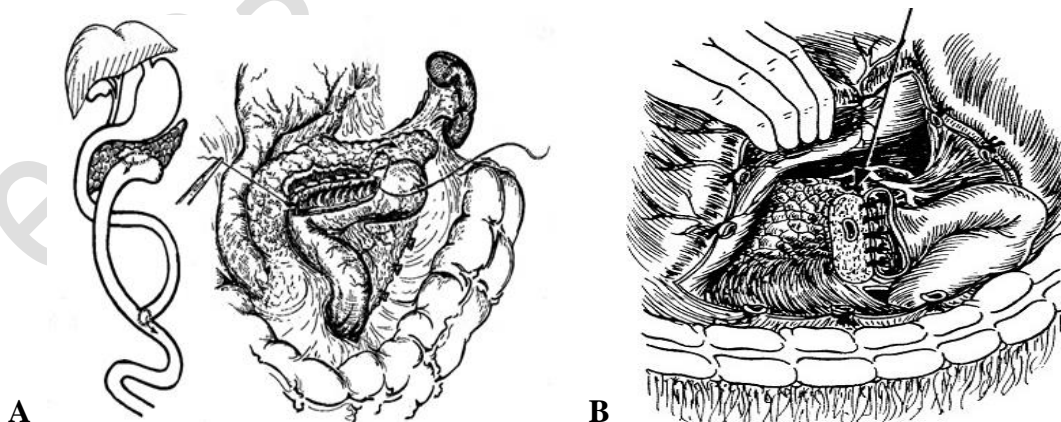
**Gastric surgery** — in secondary pancreatitis on the background peptic ulcer disease:

- resection of the stomach;
- selective proximal vagotomy (SPV).

**In portal hypertension with hypersplenism** — splenectomy (usually with the ligation of varicose veins of the gastric cardia and esophagus).

### **OPERATIONS ON PANCREAS**

In expanding ductal system of pancreas — perform longitudinal or caudal pancreatojejunostomy (operations Pyustau, Duval), figure 8.



**Figure 8 — A: longitudinal pancreatojejunostomy. After wide excision of the main pancreatic duct, impose anastomosis on isolated Roux loop of jejunum. B: caudal pancreatojejunostomy**

**Frey's Operation** is most popular in the United States. Technically, it is close to the longitudinal pancreatojejunostomy, but added resection of the pancreatic head tissue. Also resection of the pancreatic head (on Beger) with pancreatojejunostomy (figure 9).

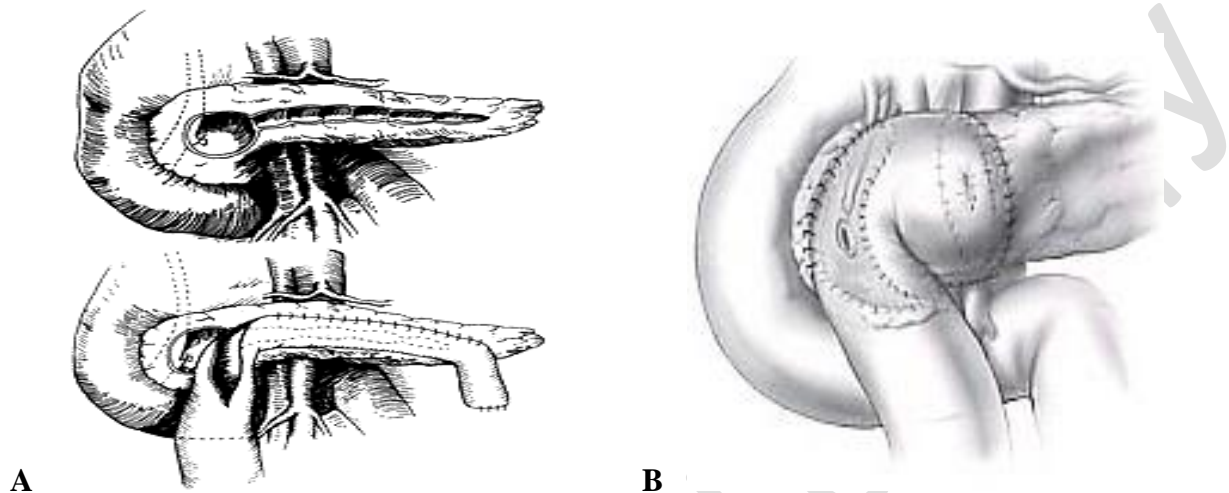


Figure 9: A — Frey's operation; B — Beger's Operation

- **Resection of the pancreas.**
- **Pancreatoduodenectomy** (Whipple's operation) is performed for suspected cancer or Pseudotumor of pancreas (figure 10).

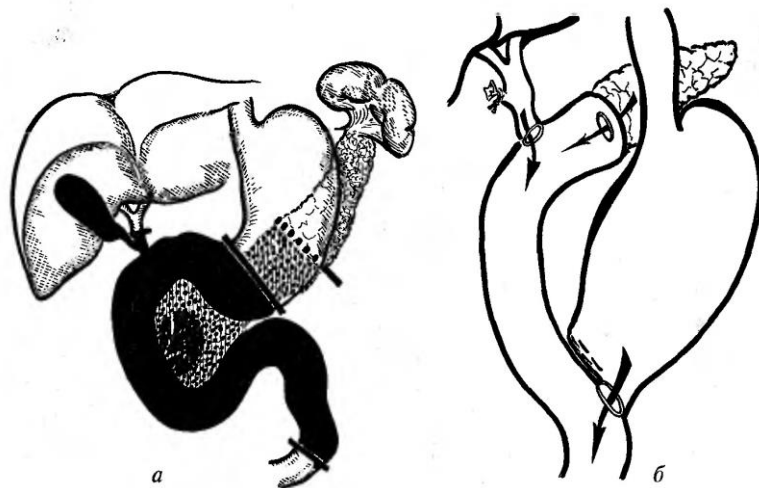
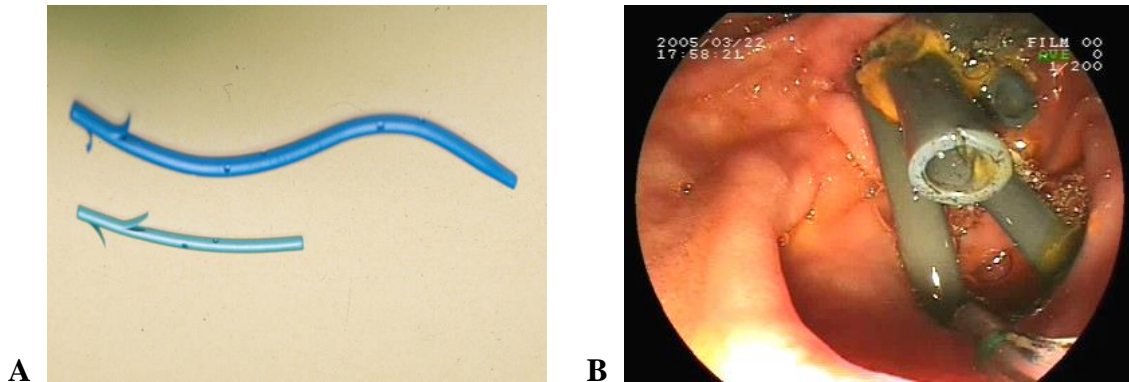


Figure 10 — Pancreatoduodenectomy:  
a — resection (shown in black); б — diagram of completed operation

- **External drainage** of main duct;
- **Disabling the exocrine function of the pancreas** (ligation of the Wirsung duct);
- **Endoscopic intervention:** EPST with the installation of a plastic stent through the MDP into the Wirsung duct (figure 11).



**Figure 11 — A — plastic stents; B — endoscopic appearance of double biliary-pancreatic drainage**

## **OPERATIONS ON AUTONOMIC NERVOUS SYSTEM**

For the treatment of persistent pain in non-extended ductal system are directed to interrupt abnormal afferent pain impulses:

- Right-sided subphrenic vagotomy.
- Left-sided splanchnicectomy with resection of the left semilunar node (by Mallet-Guy), can be done endoscopically (thoracoscopic) or «chemical splanchnicectomy» — by introducing alcohol or phenol.
- postganglionic neurotomy by Yoshioka.
- Marginal neurotomy by Napalkov-Trunin.

The efficiency of these operations is low. Currently have lost their significance.

## **PANCREATIC CYSTS**

— It is a fluid accumulation with surrounded capsule, located in the gland itself or/and para-pancreatic.

### **Aetiology:**

- 50 % — developed after severe forms of destructive pancreatitis (pancreo-necrosis);
- 20 % — Trauma of pancreas;
- 25 % — occurs in patients with CP;
- 5 % — «tumor cyst» (cystadenoma, cystadenocarcinoma).

## **CLASSIFICATION OF CYSTS**

### **According to origin of the cyst:**

#### **• Congenital:**

Cysts resulting from malformation of the pancreas and its ductal system (dermoid, teratomatous cysts, cystic fibrosis);



- **Acquired:**

1) Retention cysts that develop in the ducts strictures, resistant occlusion of their lumen with calculi and scars;

2) Degenerative arising from necrosis of tissues of pancreas in destructive pancreatitis, pancreatic trauma;

3) Proliferative — some forms of abdominal tumors, which include benign cystadenoma and malignant cystadenocarcinoma;

4) Parasitic.

Depending on the causes and mechanisms of the formation of cysts, the structural features of the walls and symptoms, pancreatic cysts are divided into true and false cysts.

To the **true** cysts include congenital cysts of the pancreas, acquired retention cysts, cystadenoma and cystadenocarcinoma. Distinguishing feature of the true cyst is the presence of an epithelial lining on its inside surface. True cysts are less than 10 % of all pancreatic cysts. In contrast, the false from the true cysts are usually of small size and often are random findings during ultrasound or during surgery undertaken for other diseases.

**False** (pseudocysts) — have only a fibrous capsule:

- postnecrotic;
- posttraumatic;
- idiopathic.

**True** cysts are always located intrapancreatically (organ cysts).

**Pseudocysts** often extrapancreatic — in the lesser omentum or retroperitoneal space.

**By onset** (Postnecrotic cysts):

- acute (up to 2–3 months of existence cysts);
- subacute (3–6 months);
- chronic (more than 6 months).

**By size:**

- small — only visualized on ultrasound;
- medium — palpable through the abdominal wall (up to 6 cm);
- large — more than 6 cm, deformed the anterior abdominal wall.

**Complications of cysts — 25 % of patients:**

- suppuration;
- perforation and peritonitis;
- Fistulas, external and internal;
- bleeding into the cyst, in the abdominal cavity, in the lumen of the gastrointestinal tract;
- mechanical jaundice;
- disorder of patency of the upper gastrointestinal tract;
- regional portal block;
- malignization.

### Clinical features of pancreas cysts:

- pain — dull epigastric pain, left upper quadrant;
- palpable tumor formation;
- dyspepsia, weakness, weight loss;
- symptoms associated with pressure of cysts on other organs;
- symptoms of other complications of cysts.

### DIAGNOSIS OF PANCREATIC CYSTS

- **USG** — the first stage of diagnosis (figure 12).



**Figure 12 — A: USG — cyst of pancreas;  
B — Endo USG (pseudocyst of body of pancreas)**

### Differential diagnosis of cysts in the pancreas. U.S.G. signs:

#### **Congenital cysts:**

- Anechogenic.
- Smooth edges.
- Often combined with cystic kidneys and liver.
- There are no signs of pancreatitis.

#### **Necrosis, pseudocysts in acute pancreatitis:**

- Irregular edges.
- Internal echostructure usually has a combined character (necrosis, hemorrhage, infection).
- inflamed echogenic walls (pseudocyst).
- aspirate: dreggy, greenish, bloody, purulent.

#### **Cysts in chronic pancreatitis:**

- Smooth edges.
- anechogenic.
- Size: 2–4 cm (up to 17 cm).
- aspirate: a sterile, contains few cells, rich in enzymes.

**Parasitic cyst:** single cyst or rosette shaped formation, echogenic wall.

**Cystic tumor** (by Kloppel):

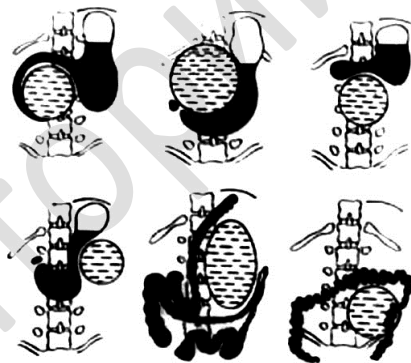
- Adenocarcinoma of the duct with signs of cysts: a solid or cystic tumor.
- Intraductal papillary mucinous tumor: cystic formation around the duct in the head of the pancreas.
- Mucinous cystic tumor (pistadenoma): combined solid-cystic formation in the body of the pancreas. Occurs mainly in middle-aged women.
- Serous cystic tumor (micro-cystic cystadenoma) solid-microcystic formation with pre-wall extension of the duct. A benign tumor that occurs predominantly in older women.
- A solid pseudopapillary tumor.
- Cystic neoplasms of unspecified nature.

**Pancreatitis of the head or tail of the pancreas:**

*Mild pancreatitis:* Hypoechoic tumor enlargement (painful when compressed). Reversible after the acute symptoms subsided.

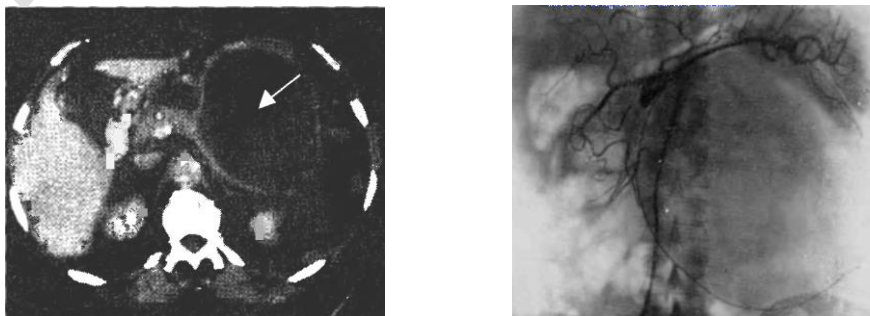
*Severe pancreatitis:* edema in combination with the accumulation of fluid inside the pancreas with necrosis or hemorrhage; accumulation of fluid around the pancreas (along the contour of pancreas or in spleen region).

• **Rtg-diagnosis** — contrast radiography of the stomach and duodenum. Identified: displacement of the stomach anteriorly, upwards and to the left, compression of the antrum and the narrowing of the descending part of the duodenum with deployment of the loop and other features of the mass formation (figure 13).



**Figure 13 — Radiographic signs of pancreatic cysts**

**Computed tomography and MRI** commonly used for differential diagnosis of abdominal mass lesions. Selective angiography is used in rare cases (figure 14).



**Figure 14 — A — CT of the abdomen (large cyst in the tail of the pancreas); B — selective angiography (an avascular mass in the pancreatic head)**

## TREATMENT OF PANCREATIC CYSTS:

- **Acute cysts** treated conservatively on the principles of treatment of acute pancreatitis (30 % of cases undergo spontaneous resolution within 2–4 months).

- **Postnecrotic cyst** operates not earlier than six months from the date of formation.

- **Small cysts** may regress spontaneously.

### Indications for surgery:

- The large size of cysts (more than 6 cm).

- The progressive growth of cysts.

- Complications of cysts.

- Suspected tumor character.

### Methods of operations in cysts:

- **Needle aspiration method** — in small cysts (under ultrasound guidance or CT).

- **External drainage of cysts** — (when suppuration). Leads to the formation of pancreatic fistula.

- **Marsupialization** — At suppuration of larger size cysts. D-caping the cyst with its walls sutured to the abdominal wall (figure 15).

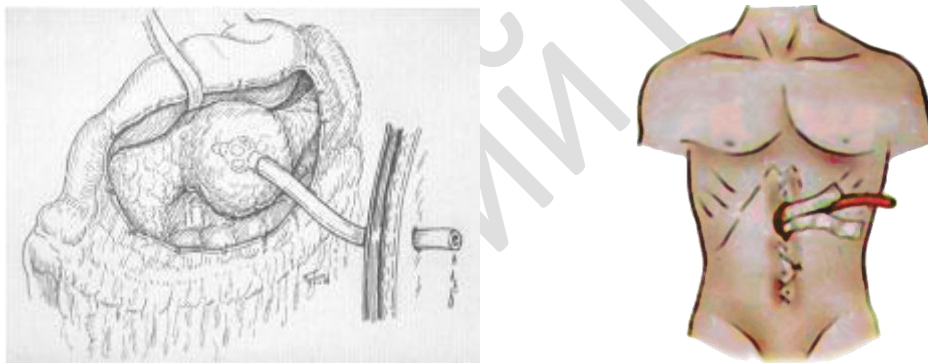


Figure 15 — A — external drainage of cysts of the pancreas;  
B — schematic representation of marsupialization of the pancreas

### Internal drainage of cysts (in chronic uncomplicated cysts), figure 16.

- Cystogastrostomy, including an endoscopic.

- Cystoduodenostomy.

- Cystojejunostomy.

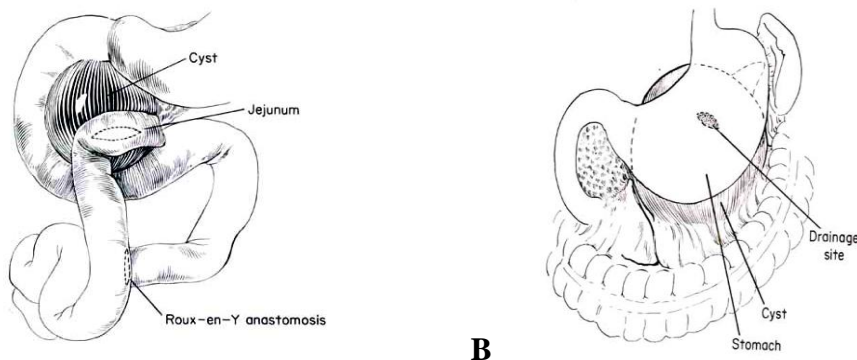


Figure 16 — A — cystojejunostomy with Roux-en-Y loop; B — cystogastrostomy

**Partial removal of the cyst:**

- Dissection and suturing of the cyst.
- Partial resection with suturing of the remaining wall of the cyst.
- Partial resection with external drainage.

**Complete removal of the cyst:**

- Cystectomy.
- Enucleation of the pseudocyst.
- Pancreatic resection together with the cyst.

## FISTULAS OF PANCREAS

— Abnormal communication between the duct system and/or post-necrotic cavity of the pancreas with other organs, cavities or the outer surface of the body.

**In 75–80 % — come from the body and tail.**

**Causes:**

- 30–55 % — are formed as a result of acute destructive pancreatitis.
- 12–40 % — trauma of pancreas with structural damage of the duct system.

**Other causes:**

- Spontaneous emptying of pancreatic cysts in the nearby organs and cavities;
- Difficulty of outflow of pancreatic secretions in the duodenum;
- In external drainage of omental bursa, bed of pancreas or cysts.

## CLASSIFICATION OF FISTULAS OF PANCREAS

**By etiology:**

- Postnecrotic/
- Post-traumatic (including surgical trauma)/
- After external drainage of pancreas cysts.

**By clinical features:**

- Permanent.
- Recurrent.

**Liaison with pancreatic ductal system:**

- Fistulas, associated with the ductal system:
  - patency of the duct is impaired;
  - patency of the duct is not impaired;
- Fistulas, not associated with the ductal system — parapancreatic (with or without pyogenic cavity).

**By Localization of the fistulous:**

- External fistula.
- Internal: the stomach, intestine, and other cavities.

**By the number of fistula channel:**

- Single-channel.
- Multi-channel.

**By infection:**

- Uninfected.
- Infected.

## CLINICAL FEATURES

**Period of formation of the fistula.** Clinical signs are associated with the release of pancreatic secretion in parapancreatic space, sequestration and autolysis of tissues. Complaints: epigastric pain, dyspepsia, elevated body temperature, the appearance of the mass in the pancreatic area.

**Period of functioning of the fistula.** In patients with external fistulas: presence of holes in the skin with pancreatic secretion (colourless, high-amylase and trypsin), maceration of the skin; if infected — an admixture of pus, blood, etc.

**Period of complications.** With prolonged loss of pancreatic secretion, oppression of exocrine function, depletion of patients, external bleeding and Whirsungorrhage.

## DIAGNOSIS OF FISTULAS

- Patient history.
- Biochemical tests of secretions of the fistula.
- Fistulography (figure17).
- ERCP.
- Ultrasound.
- CT, MRI.



**Figure 17 — Fistulography (fistula associated with pancreatic ductal system)**

## TREATMENT OF FISTULAS OF PANCREAS

### **Conservative:**

- oppression of exocrine pancreatic function,
- inactivation of the enzymes of pancreatic juice,
- sanitation of the fistulous,
- elimination of damage to the skin around the fistulous,
- correction of existing metabolic disorders.

**Indications for surgical treatment:**

- failure of conservative treatment for 3 months,
- violation of the outflow of secretions in the duodenum,
- fistula passing through the poorly drained cavity,
- progression of the inflammatory and destructive processes in the pancreas.

**TYPES OF SURGERY:**

- **Excision of the fistula with resection of pancreas** — almost a radical surgery.
- **Excision of the fistula with resection of pancreas + overlaying of pancreatojejunostomosis** — in impairment of Wirsung duct patency.
  - Formation of an **anastomosis** between the fistula and the hollow organ (stomach, intestine) — rarely used.
  - **Excision of fistula with ligation at its base** — with certainty in the patency of Wirsung duct.
  - **Sealing of the fistula and the duct of the pancreas** fast polymerizes materials after preliminary sanitation of the fistulous.

**PROGNOSIS**

Chronic pancreatitis with its numerous complications — disabling disease. Therefore, early diagnosis and best choice of an appropriate method of treatment can provide social and occupational rehabilitation of patients.

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Учебное издание

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**ХРОНИЧЕСКИЙ ПАНКРЕАТИТ  
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**Учебно-методическое пособие  
для студентов 5 курса факультета по подготовке специалистов  
для зарубежных стран медицинских вузов**

Редактор *Т.М. Кожемякина*  
Компьютерная верстка *С. Н. Козлович*

Подписано в печать 01.04.2014.  
Формат 60×84<sup>1/16</sup>. Бумага офсетная 70 г/м<sup>2</sup>. Гарнитура «Таймс».  
Усл. печ. л. 1,4. Уч.-изд. л. 1,53. Тираж 80 экз. Заказ № 124.

Издатель и полиграфическое исполнение:  
учреждение образования «Гомельский государственный медицинский университет».  
Свидетельство о государственной регистрации издателя,  
изготовителя, распространителя печатных изданий № 1/46 от 03.10.2013.  
Ул. Ланге, 5, 246000, Гомель.