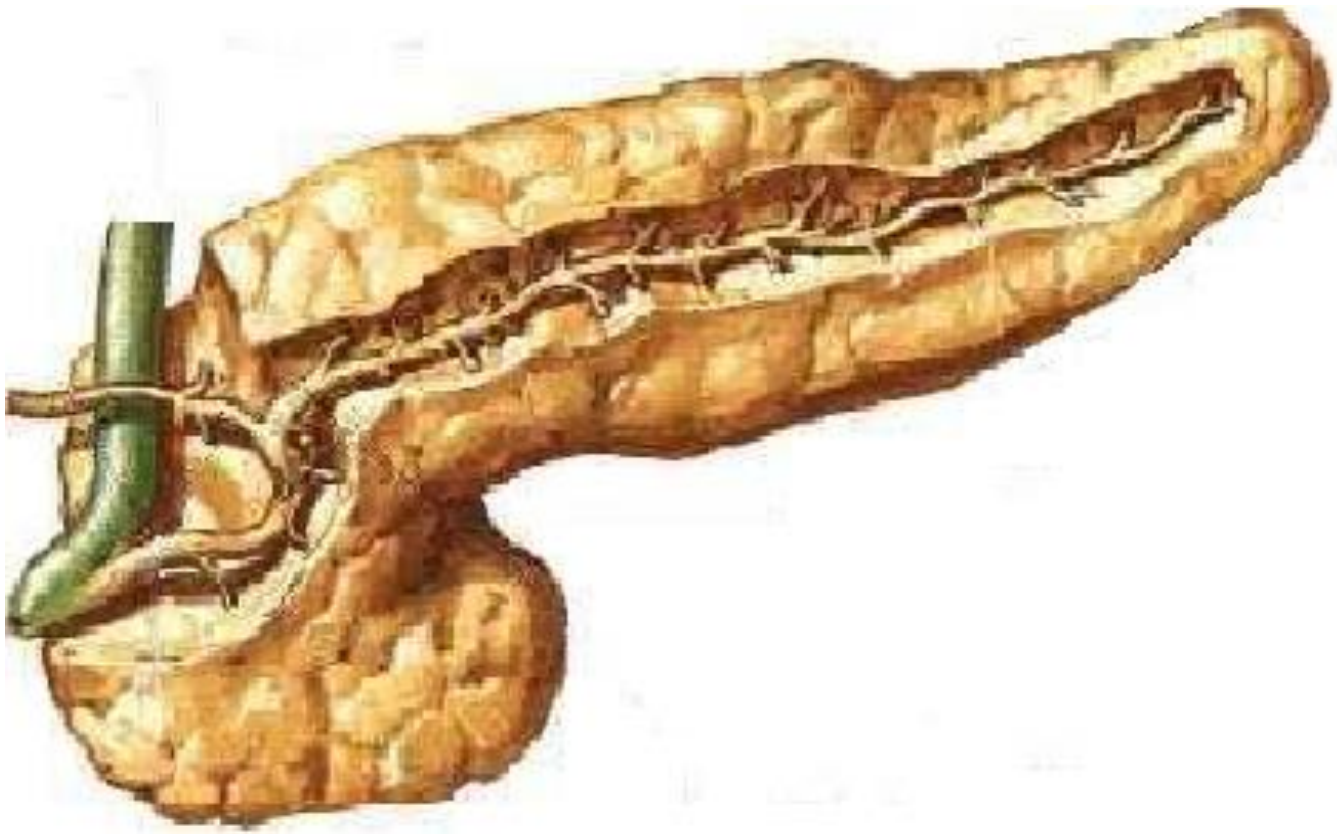
Orenburg State Medical University
Surgery Department

Acute Pancreatitis



Terminology

- **Acute pancreatitis** — acute aseptic inflammation of the pancreas with demarcation, with the pancreocytes' necrosis and ferment autoagression with gland's necrosis and secondary infection addition.
- Pancreonecrosis = destrucrive pancreatitis = necrotic pancreatitis

- Abbreviations:

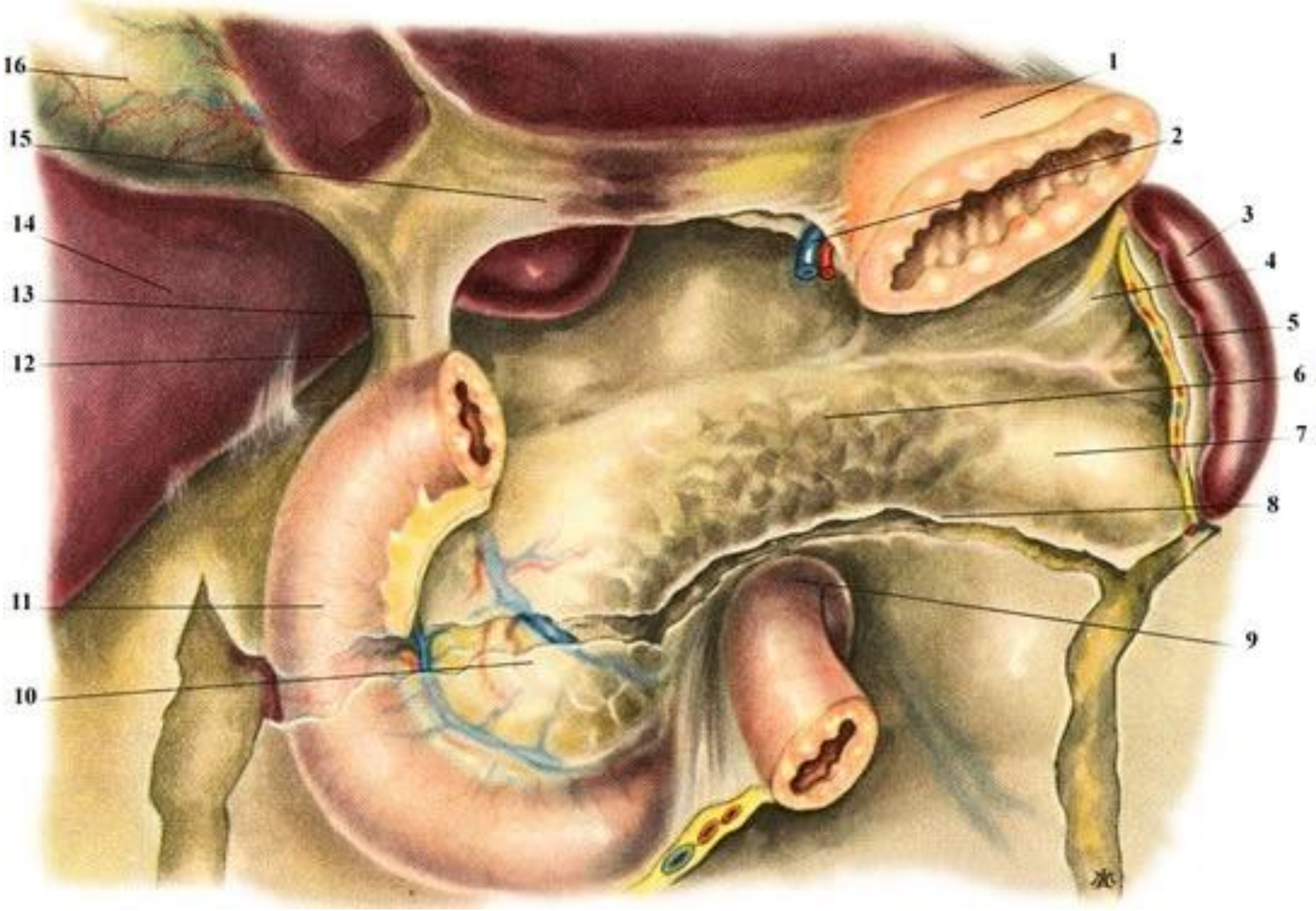
AP – acute pancreatitis

PG – pancreatic gland

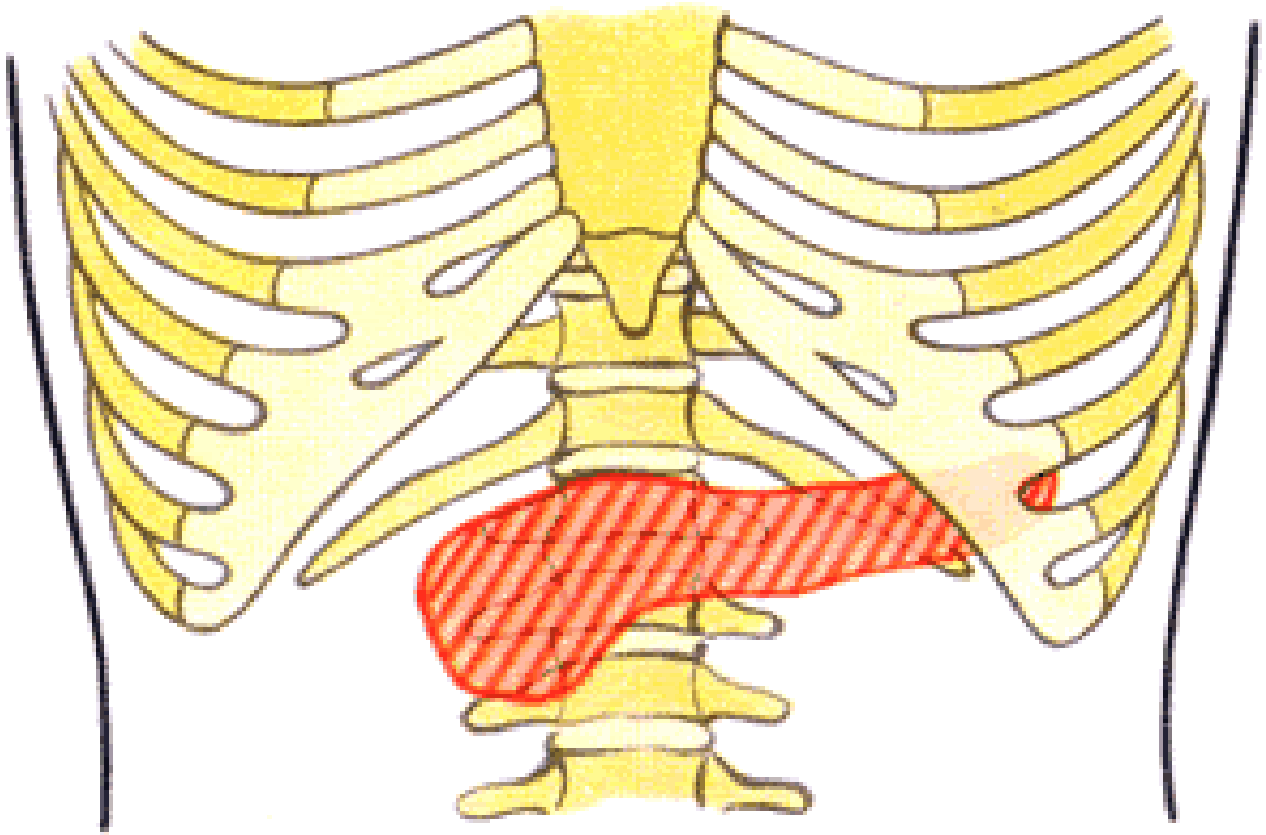
Some statistics

- From 3 to 6% of urgent abdominal pain cases.
- 3rd place after acute appendicitis and acute cholecystitis.
- Lethality from 3 to 9 %, within destructive forms from 40 to 70 %.
- the most difficult problem of the today's abdominal surgery situations (lack of some pathogenesis' issues, no strict consensus on treatment in every case).

Pancreatic anatomy

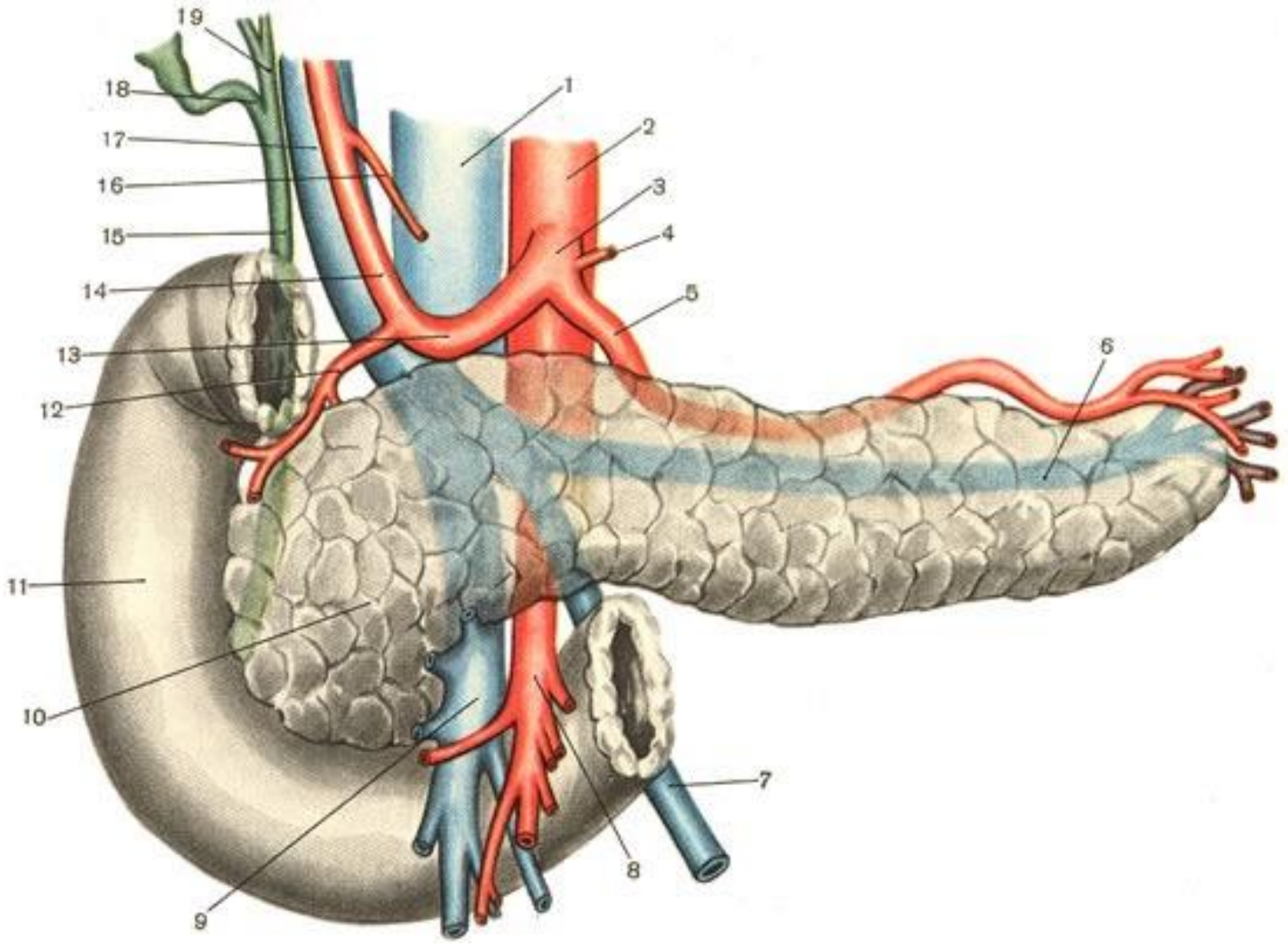


Skeletotopy of the pancreas
girdle pain

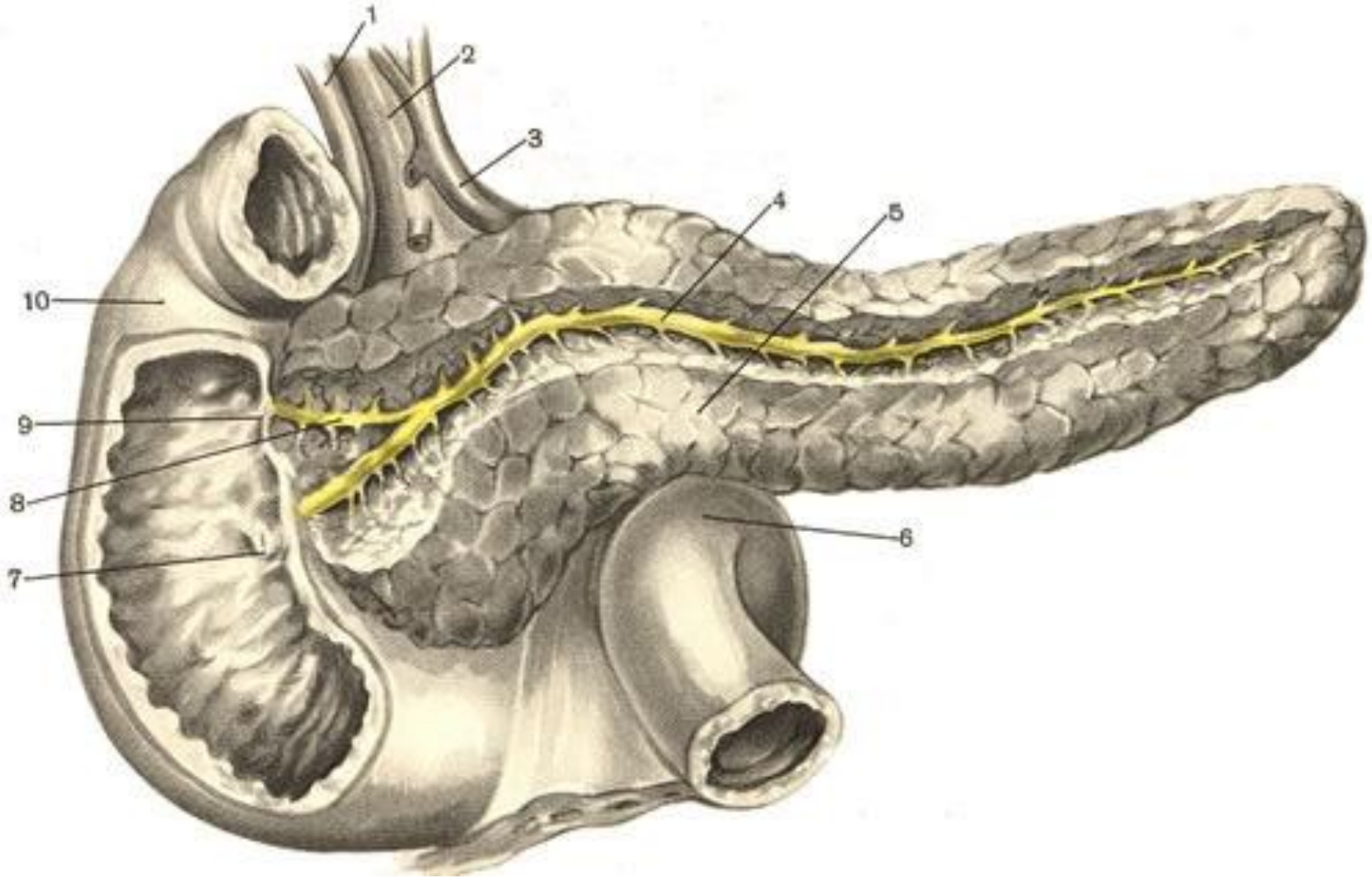


girdle pain

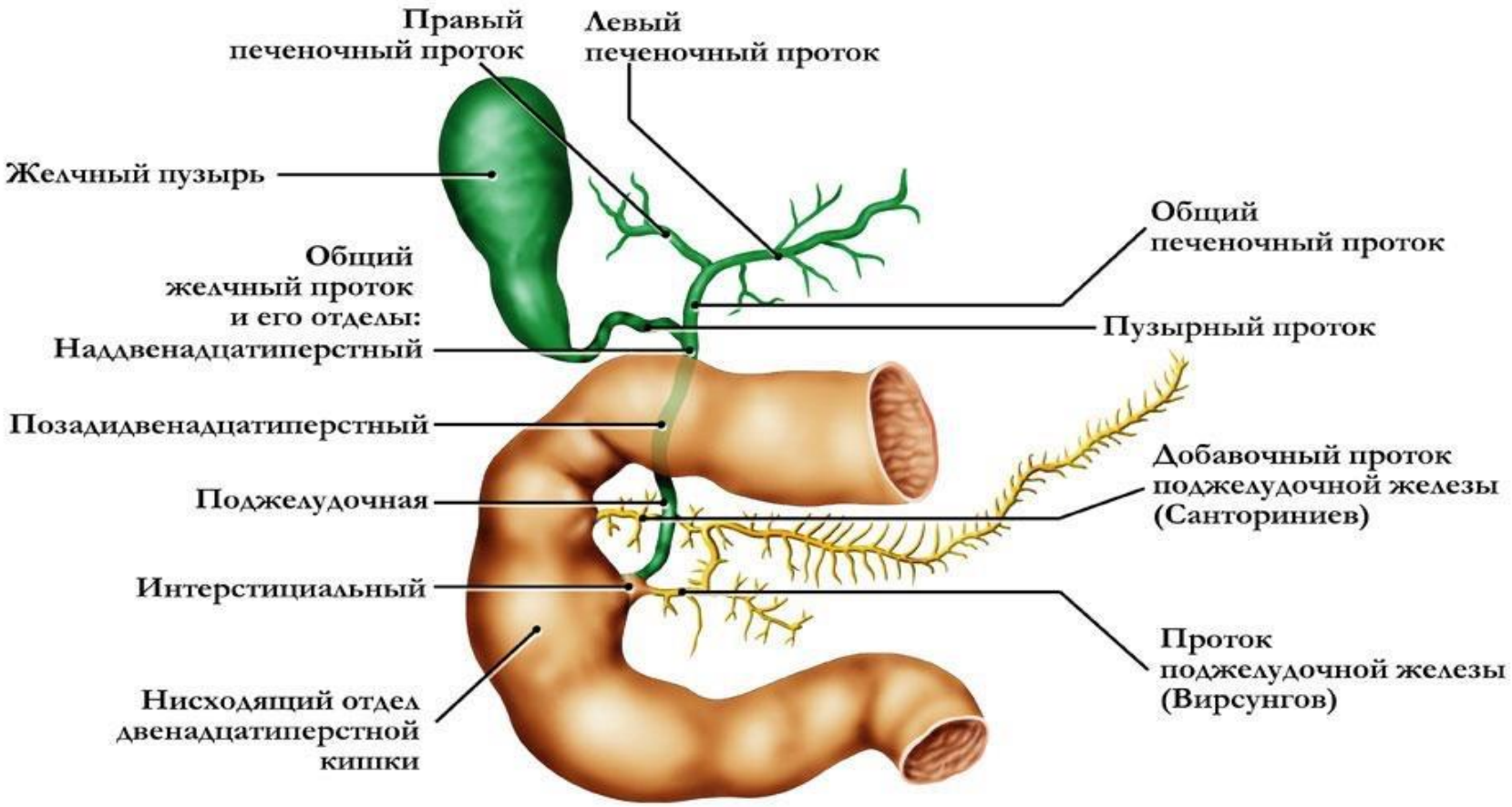
Topography with the duodenum, main bile duct, portal and inferior cave veines, abdominal aorta and it's branches



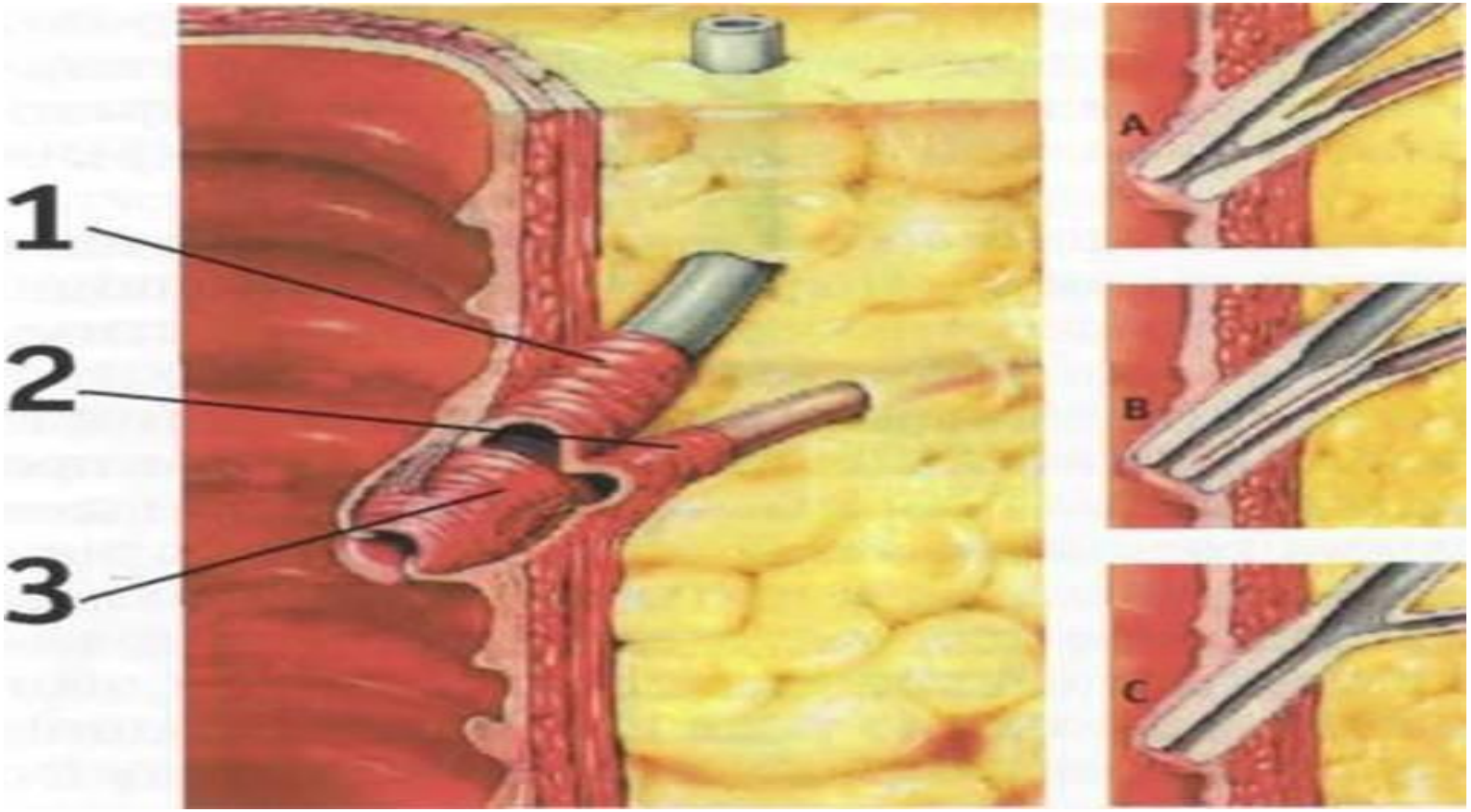
Excertory pancreatic ducts and their opening in the duodenum



Pancretobiliary system



Anatomy of big duodenal papilla



Pancreatic secretory function

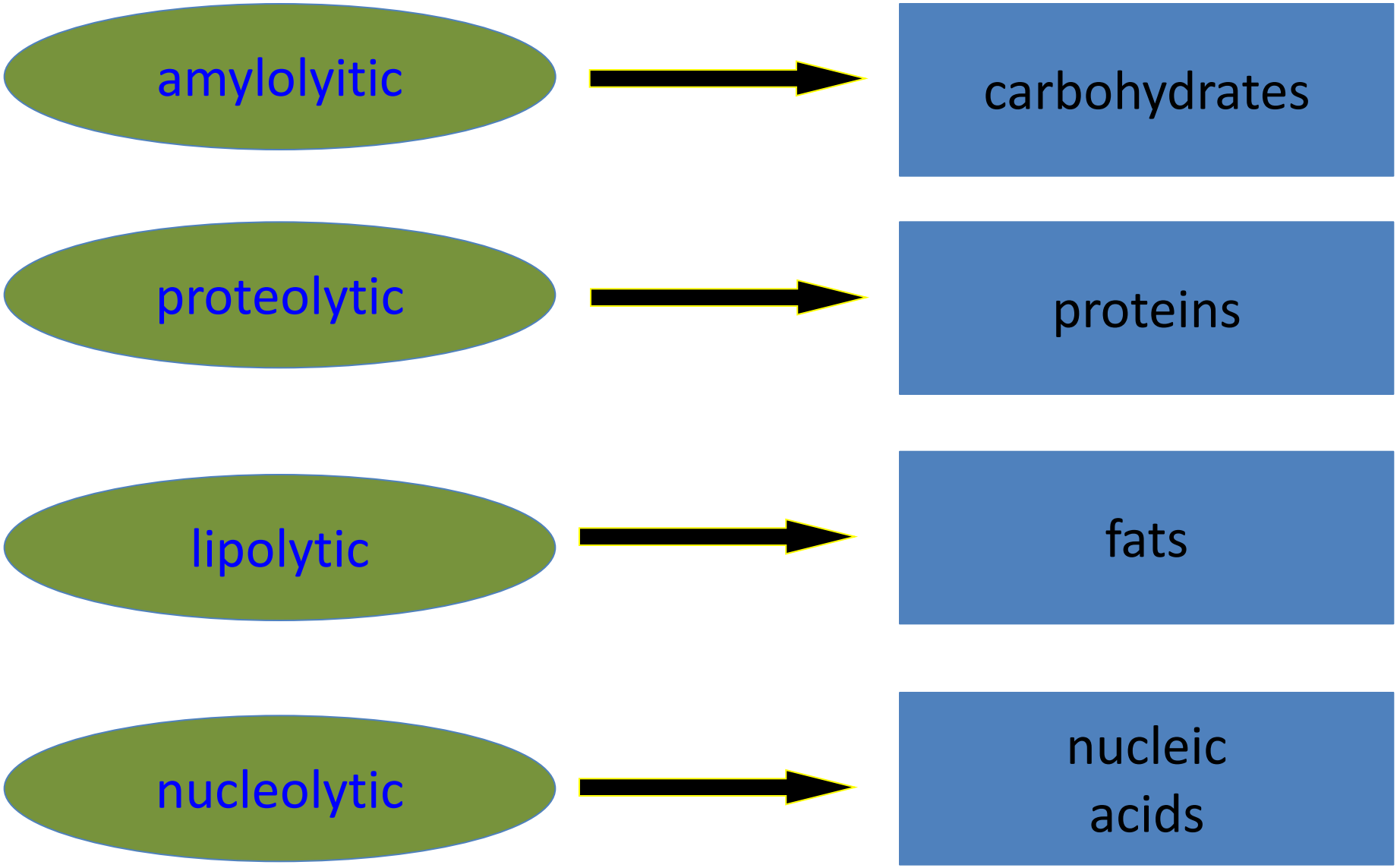
Exocrine secretion

Endocrine secretion

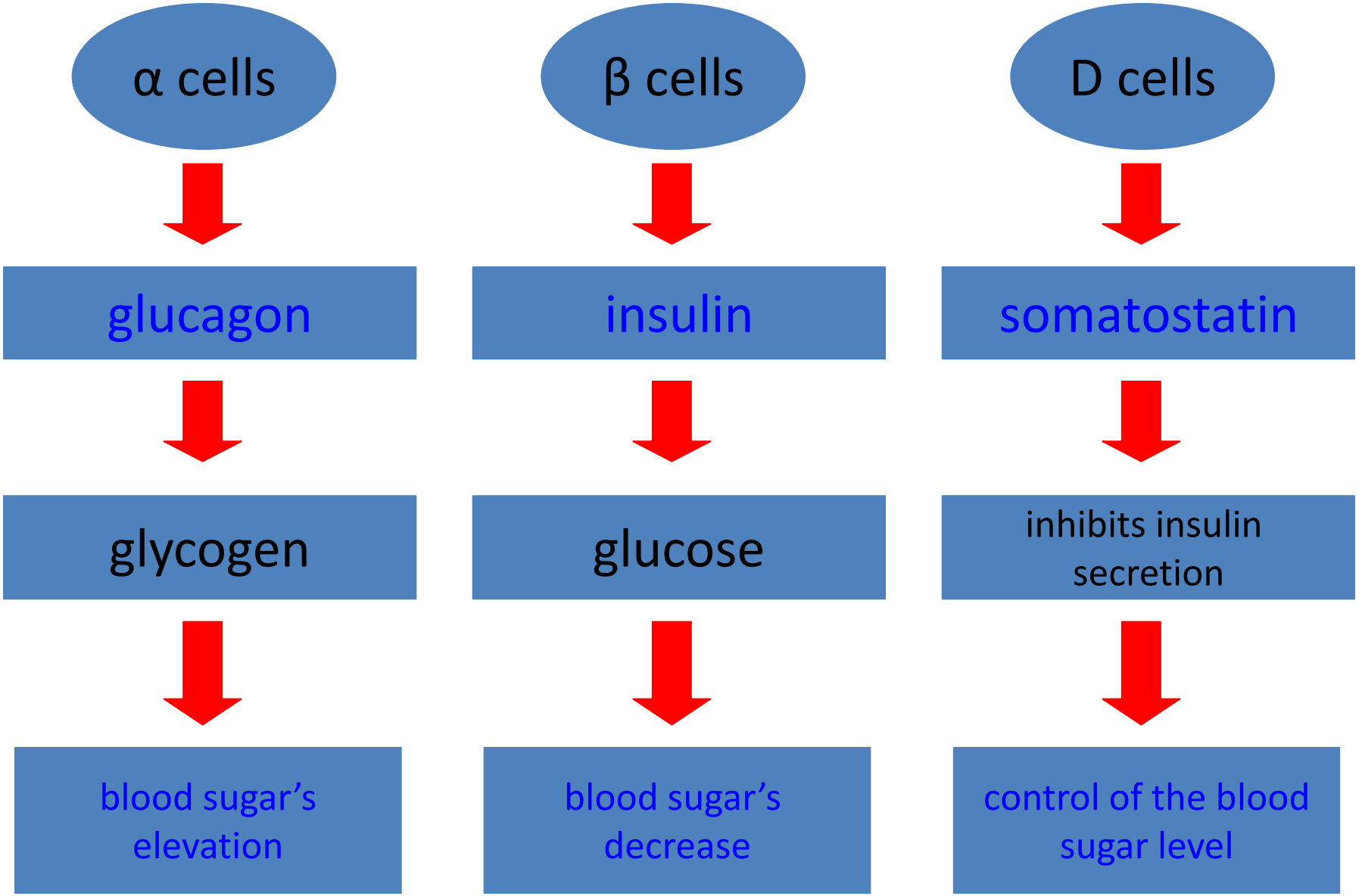
Exocrine function

- Enzyme excretion
- Water, hydrocarbonate, electrolytes for the acid stomach content (hydrokynetic function)

Pancreatic enzymes



Endocrine secretion



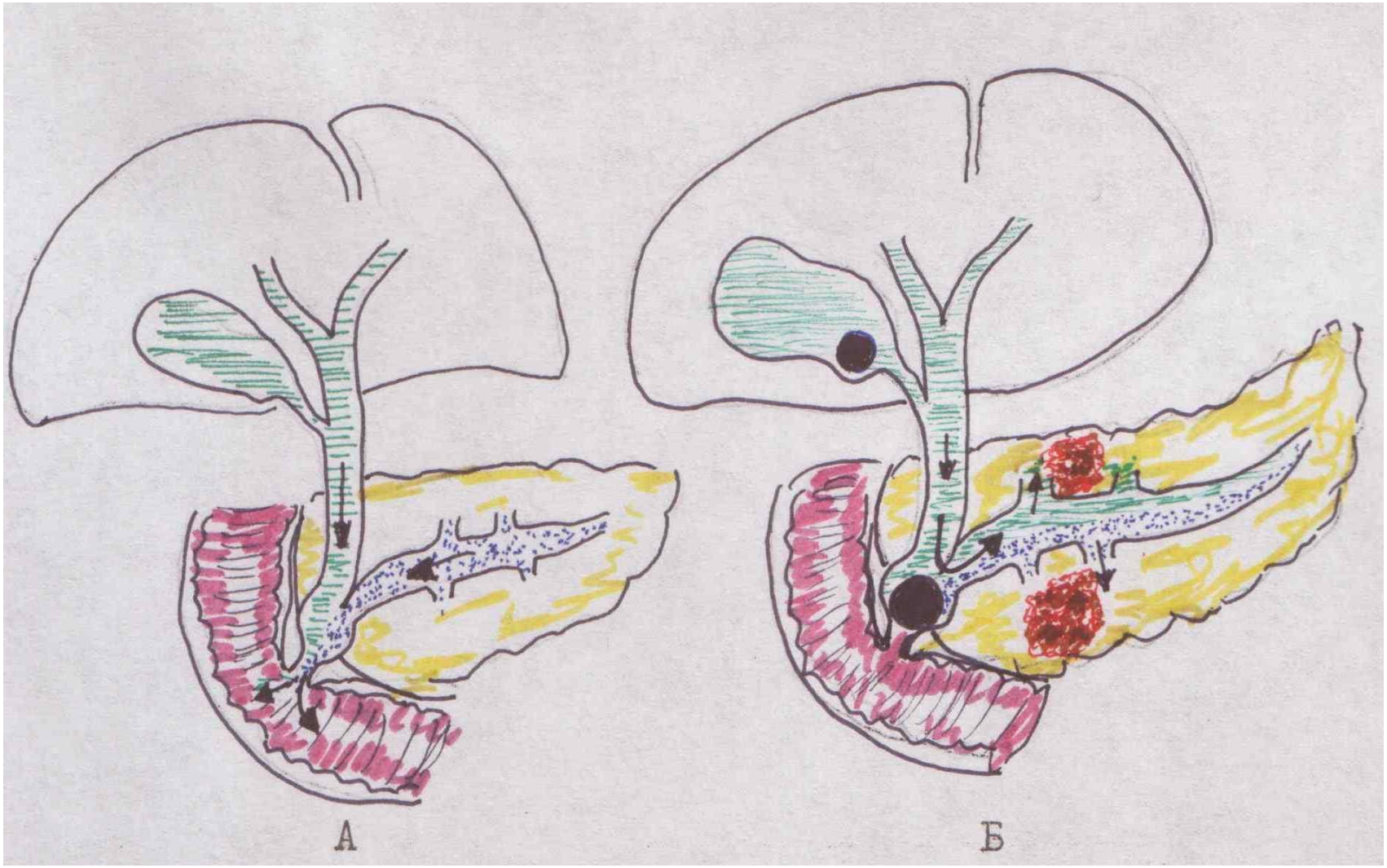
Etiology: duct system depressurization

1. Gallstones disease (30-40%), including terminal region of the bile duct pathology (choledocholythiasis, odditis, cholangitis, big duodenal papilla's stricture)
2. Alcoholism (30-75%). mechanism:
   a. Pancreatic excretory function's stimulation by alcohol.
   б. Pancreatic secret's evacuation alternation because of the Oddie's sphinctor's spasm due to the duodenum's irritation (morphine-like action).
3. Trauma of the pancreas: penetrating wounds, closed wounds, intraoperative wounds.
4. Gastrointestinal tract diseases (stomach and duodenum's ulcers with penetration, chronic duodenal passability alternation)
5. Blood flow alternation – chronic mesenteric ischemia (atherosclerosis alternation of the pancreatic vessels), centralisation of the blood flow

Etiology

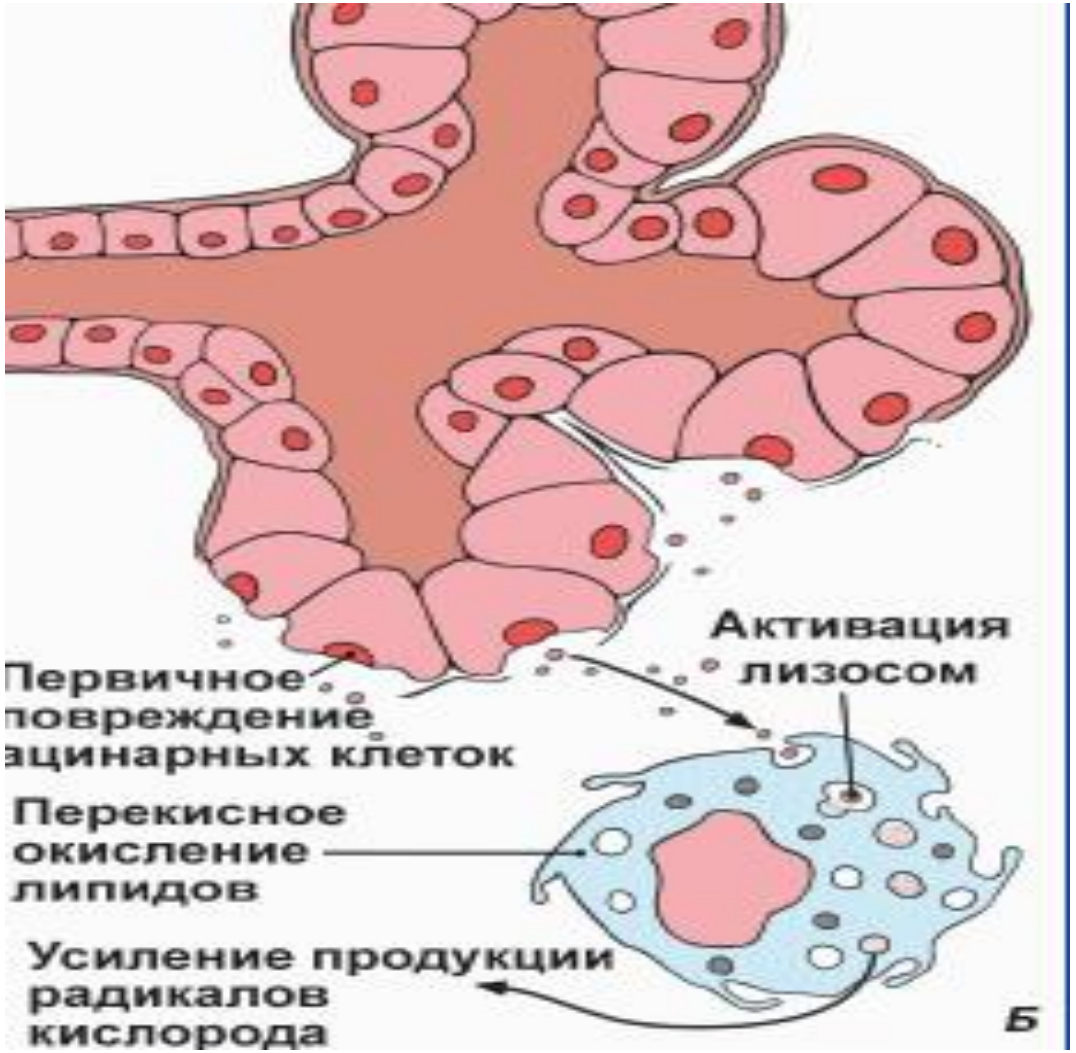
6. Endocrine issues.
   a. Hyperparathyroidism – hypercalcemia – pancreatic proteolitic enzymes activation.
   b. Hypothyroidism
   c. Lipid metabolism alterantions – hyperlipidemia, including hereditary (Friderichsen's disease)
   d. Protein metabolism alternation, insufficient protein input
7. Chronic infections (B and C hepatitis viruses, epidemic parotitis, CMV)
8. Pancreatotoxic medications (sulfanylamines, tetracyclins, non steroid anti-inflammatory treatmets, furosemid, estrogens, immunosupressants, steroids)
9. Rare anomalies (pancreatic development anomalies, hyperaminoacidurie, allergic and auto allergic reactions).

Biliary pancreatitis mechanism



Alcoholic pancreatitis mechanism

Traumatic pancreatitis mechanism



Pathogenesis

Hypertension or trauma → depressurization

of the duct system → pancreatic enzymes outflow

in the interstitium

↓

Lipolytic cascade (fat necrosis) →

acidosis

↓

Proteolytic enzyme's activation →

Proteolytic cascade (hemorragic necrosis) →

systemic vascular alternations

Classification

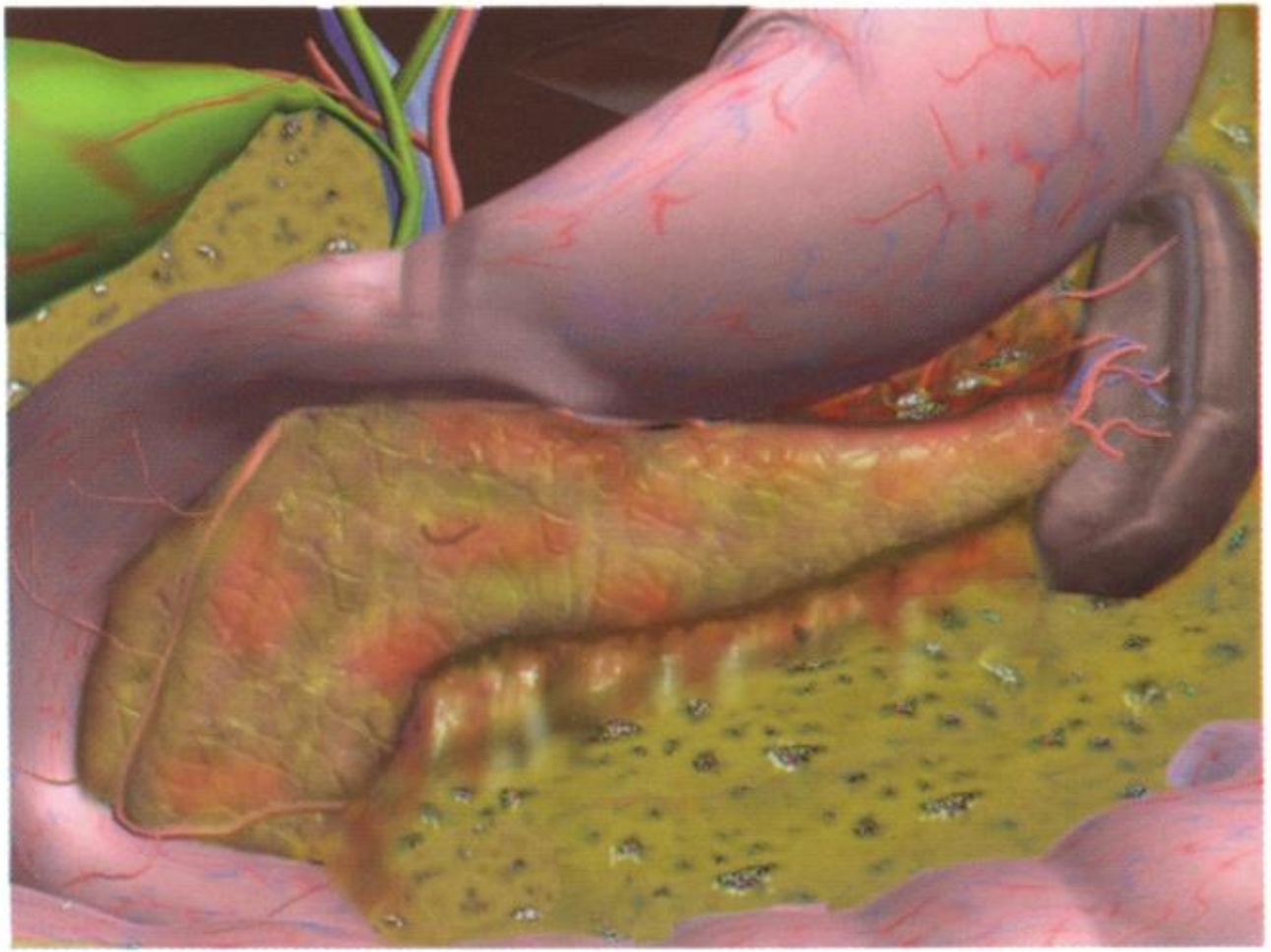
I. Oedematous (intersitial) panreatitis.
II. Steril pancreonecrosis.

- based on the necrosis characteristics:
  fatty, hemorragic, mixed;
- based on lesion abundance:
  little sources, big sources, subtotal;
- based on localisation:
  head, queue, generalized.

III. Infected pancreonecrosis.

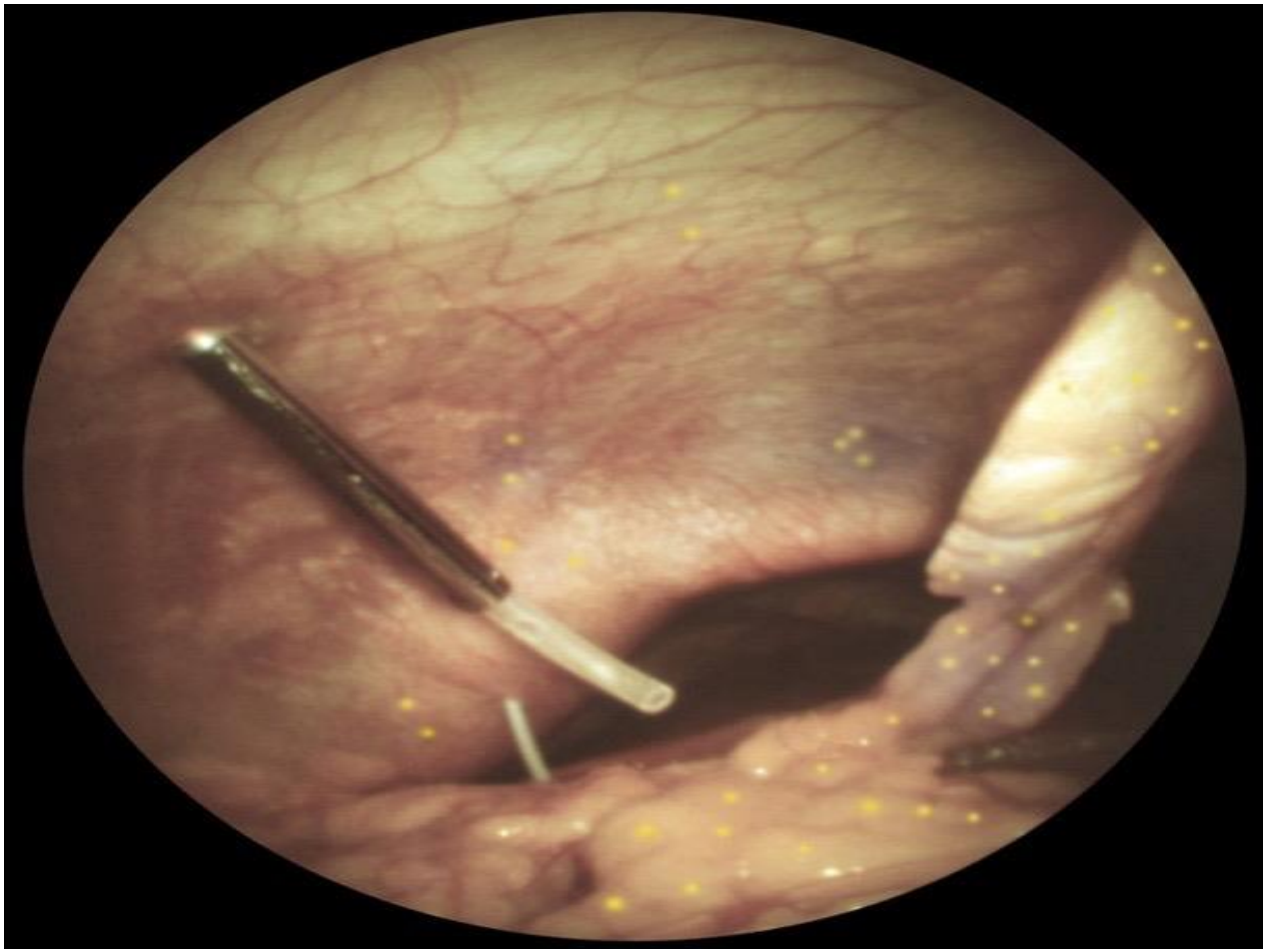
- Complications:
  I. Parapancreatic infiltrate.
  II. Pancreatic abscess.
  III. Peritonitis: fermentative (abacterial), bacterial.
  IV. Septic flegmona of the peritoneal fat: parapancreatic, paracolic, pelvic.
  V. Arrosive hemorrage.
  VI. Mechanical jaundice.
  VII. Pseudocyst: steril, infected.
  VIII. Inner and exterior digestive fistulas.

Pathologic anatomy (interstitial form)



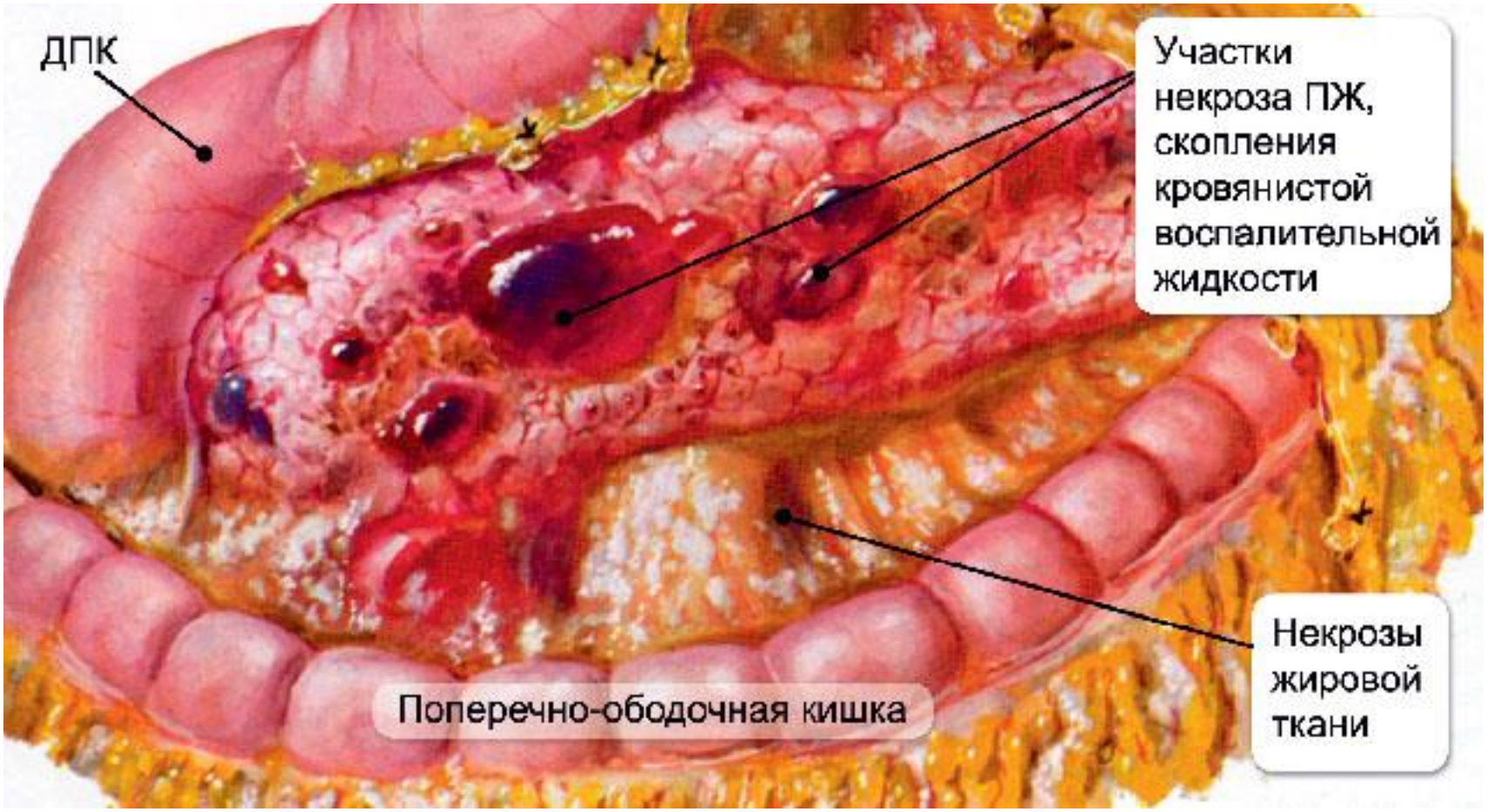
Интерстициальный острый панкреатит; отечная форма.

Pathologic anatomy



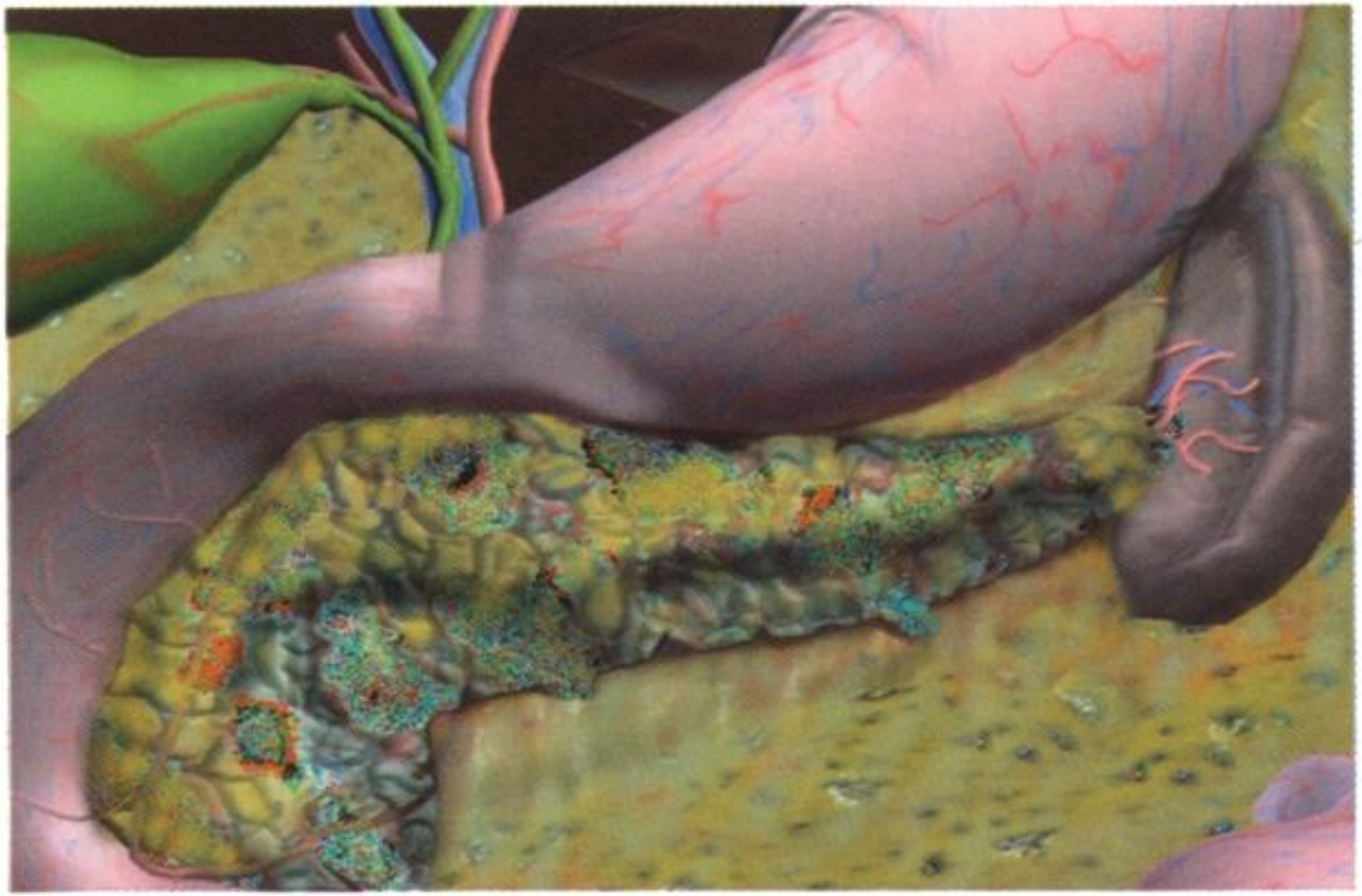
Fatty pancreonecrosis

Pathologic anatomy



Hemorragic pancreonecrosis

Pathologic anatomy (destructive form)



Некротический острый панкреатит; деструктивная форма

Periods of the Pancreatitis progression

1. **Hemodynamic alterations period** (pancreatogenic shock), first 3 days
2. **Period of multyorganic failure** (shock organs syndrome), 4-10 days
3. **Period of sequestration and suppurative complications**, 10 and more days:

3.1. aseptic destructive complications

3.2. septic destructive complications (abdominal pancreatogenic sepsis)

Multiorgan failure – result of the systemic alteration of the microcirculation

1. Liver failure syndrome (all liver's functions failure);
2. Renal failure syndrome (General renal failure);
3. Cardiovascular failure syndrome;
4. Respiratory failure syndrome;
5. Encephalic failure syndrome (encephalopathy, delirium);
6. **Enteral failure syndrome** (paralysis, bacterial colonisation, translocation). Intestins – sepsis mover

Clinic of the AP

Mondor's triade

- Pain
- Vomiting
- Meteorism

Oedemous pancreatitis

Mild form of pancreatitis (interstitious oedema of the pancreatic gland)

- 80 % of patients with AP
- Lethality less than 1 %

Pancreonecrosis

Destructive form of the AP

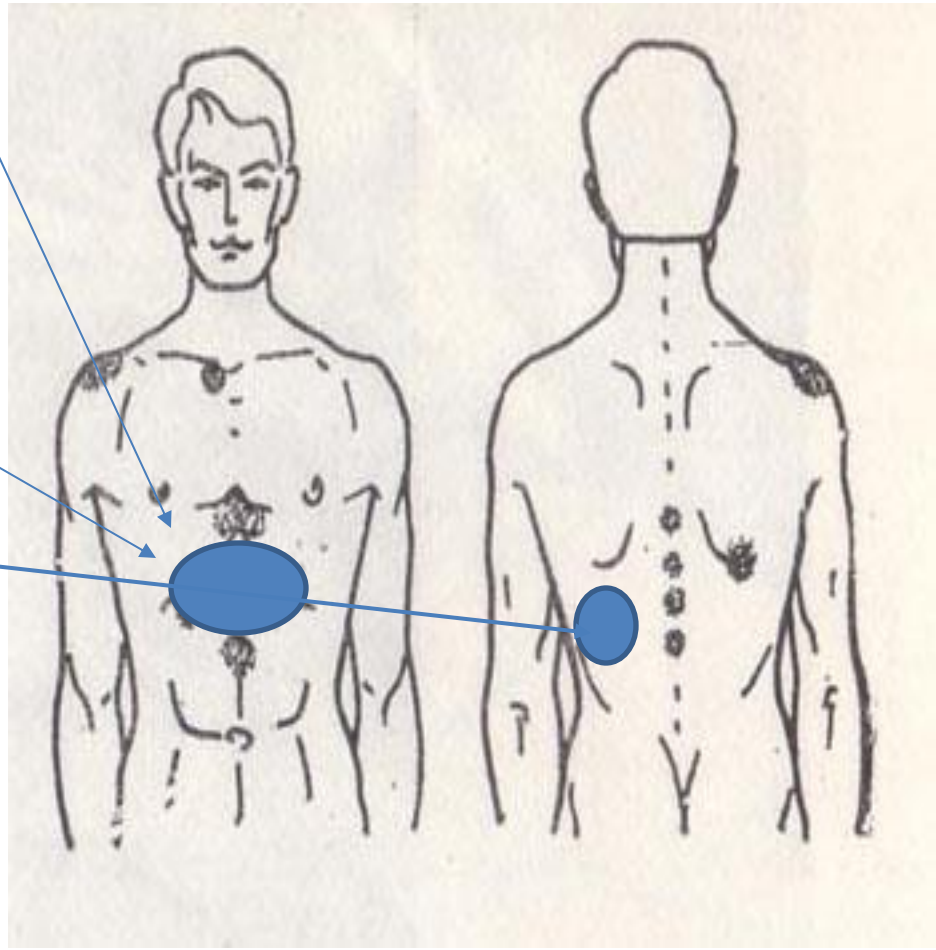
- 20 % of patients with AP
- Lethality up to 30 %

Clinic of the AP

- Sudden strong constant pain in the epigastric region with left hypochondrium irradiation, left loin region (1-3 days)
- uncessant vomiting, without relief
- Pale skin, lips cyanosis
- Tachycardia within 5-6 hours
- Possible hyperthermia (SIRS)
- Leucocytosis if satisfactory hemodynamics

Clinic of the AP

- **Kerte's symptom** (epigastric pain and rigidness)
- **Voskresensky symptom** (aortic pulsations lessen in the epigastrium)
- **Mayo-Robson symptom** (strong pain in the left vertebro-costal angle)
- **Peritoneal symptoms** (peritoneal irritation because of the hemorragic peritoneal infusion)



Symptoms of severe acute pancreatitis

- Begins with an unstable hemodynamics, up to collapse (arterial pressure<50 mm Hg)
- Strong pain symptoms. Pain localises in the epigastrium with the irradiation to the left hypochondrium, under left scapula
- Uncessant pain, without relief
- Signs of the dynamic intestinal blockage, intestinal paralysis, abdominal distention

Symptoms of the severe AP

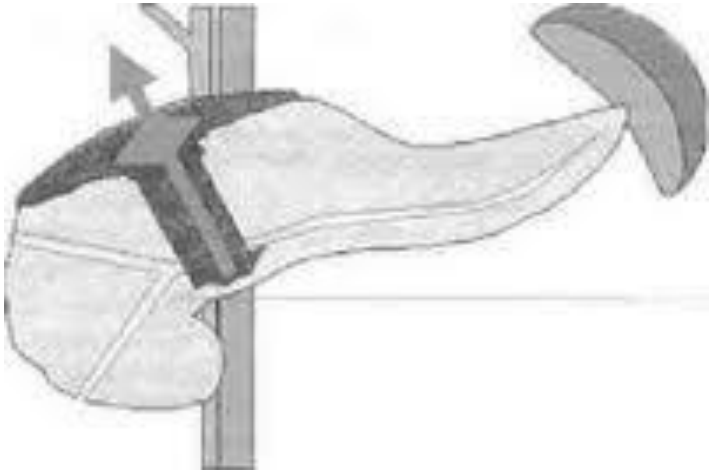
- Pain during superficial palpation of the abdomen, muscular contraction of the anterior abdominal wall , especially in the epigastric region
- Diminishing sound during percussion in the in the declivious regions (effusion)
- Leucocytosis with PNN, inflammation markers
- Blood and urine high levels amylase, lipase, tripsine (during first two days), high enzymes level in the peritoneal exsudate

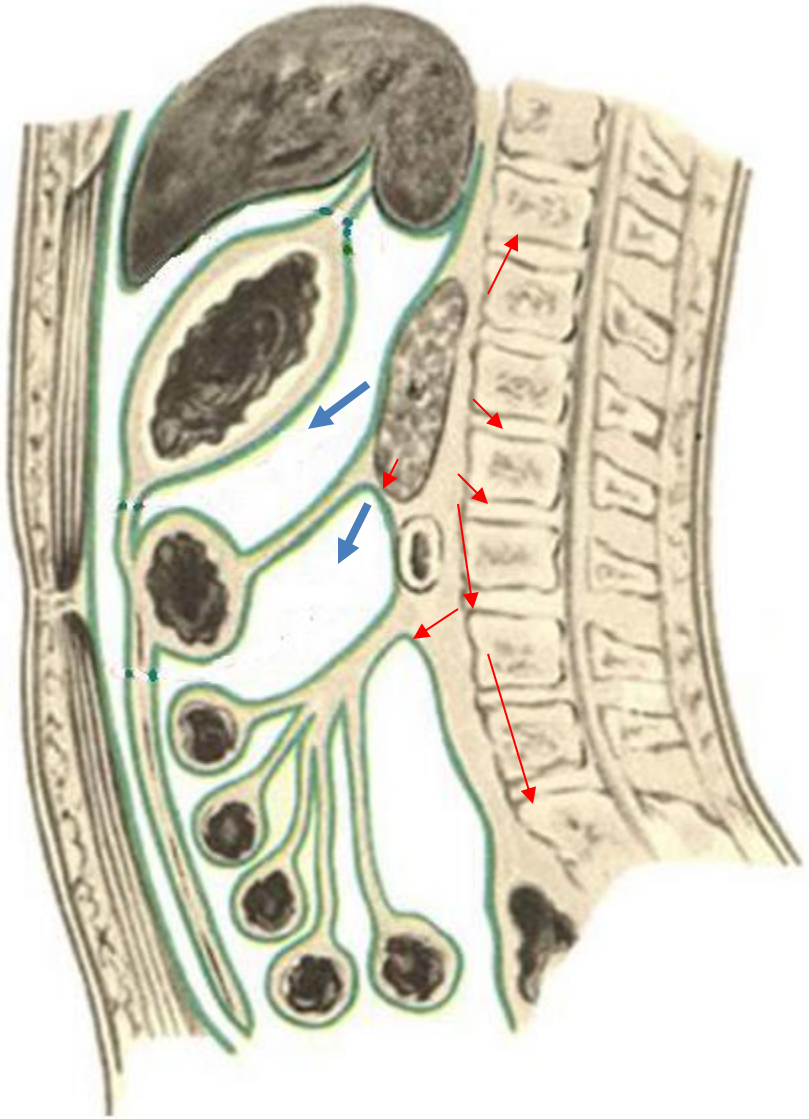
«Colour» symptoms

Skin pallor with cyanotic spots in severe cases:

- Mondor's symptom (violet spots on the face and body)
- Grey-Turner symptom (cyanotix spots on the lateral walls of the abdomen)
- Gruenwald's symptom (periombilical cyanotic spots)
- Holsted's symptom (cyanosis on the abdominal skin)
- Cullen's symptom (icteric colouring of the periombilical region)

Mechanism of the process' spreading



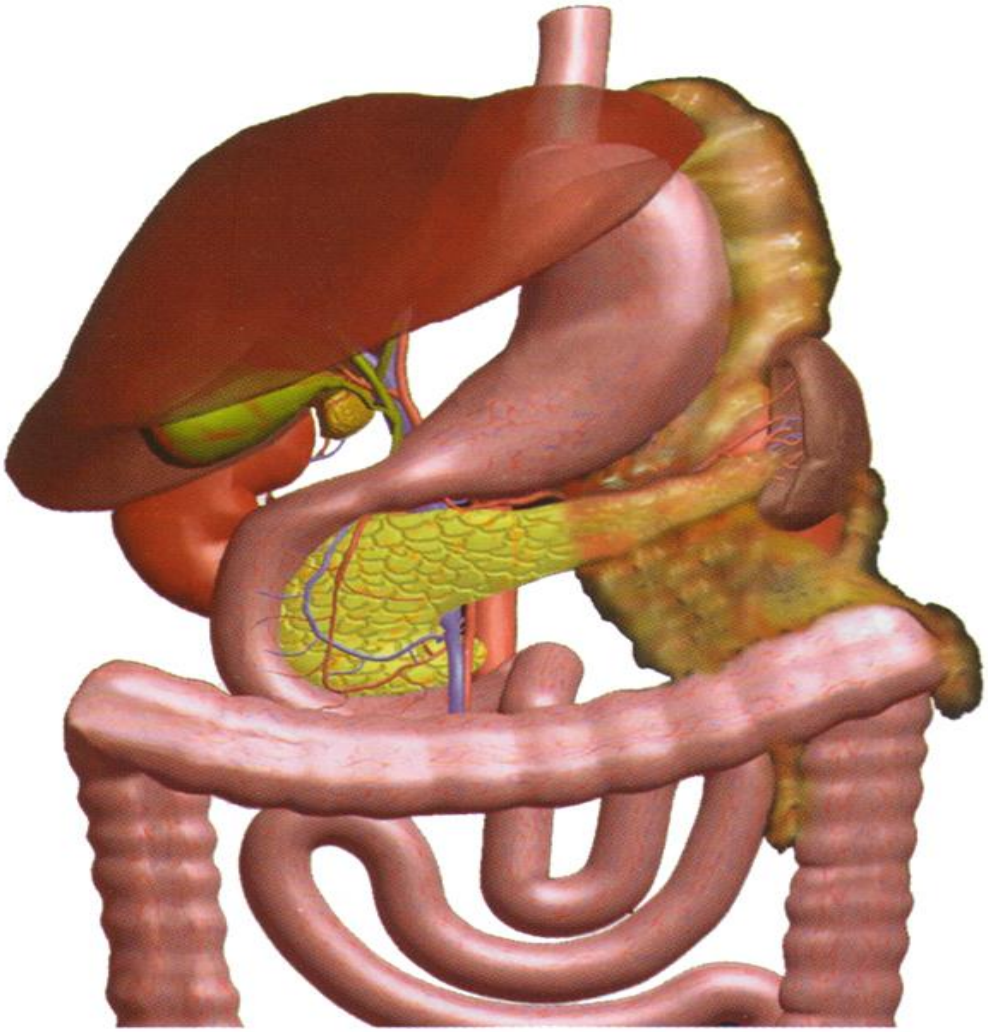


Exsudation type

1. Anterior (peritonitis);
2. Posterior (retroperitoneonecrosis);
3. Mixed type.

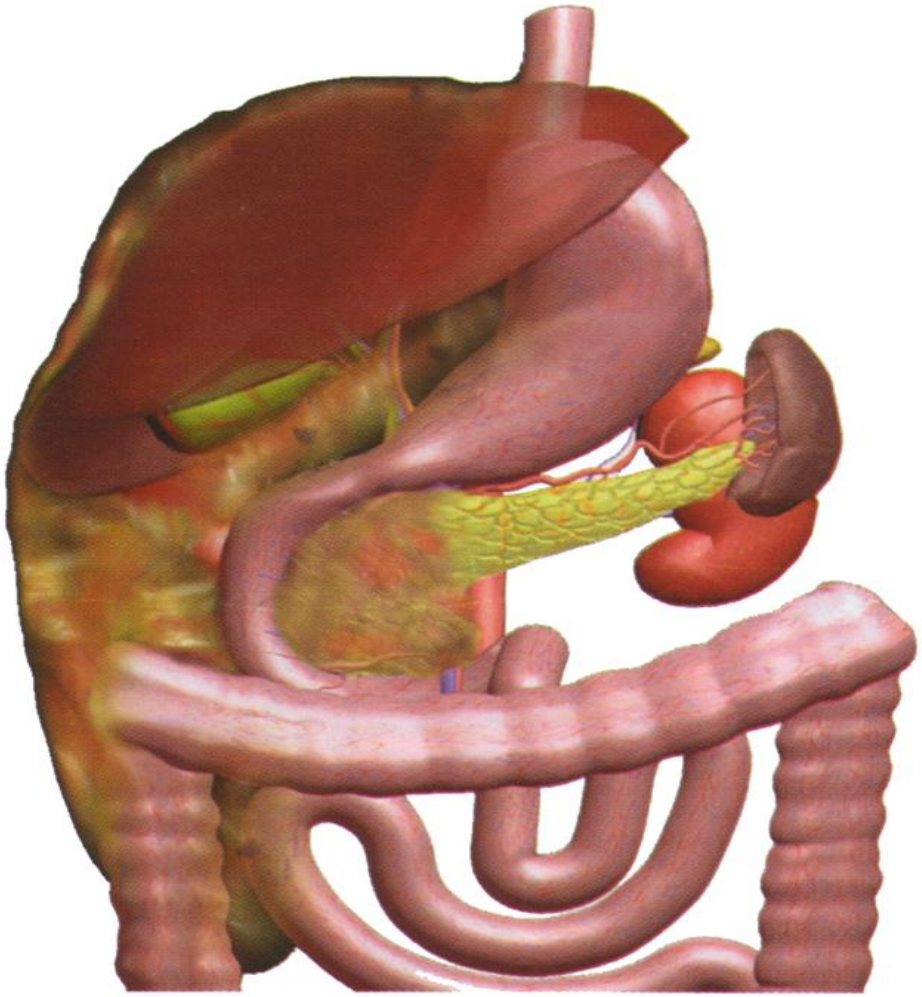
Mechanism of the process’ spreading

Left type



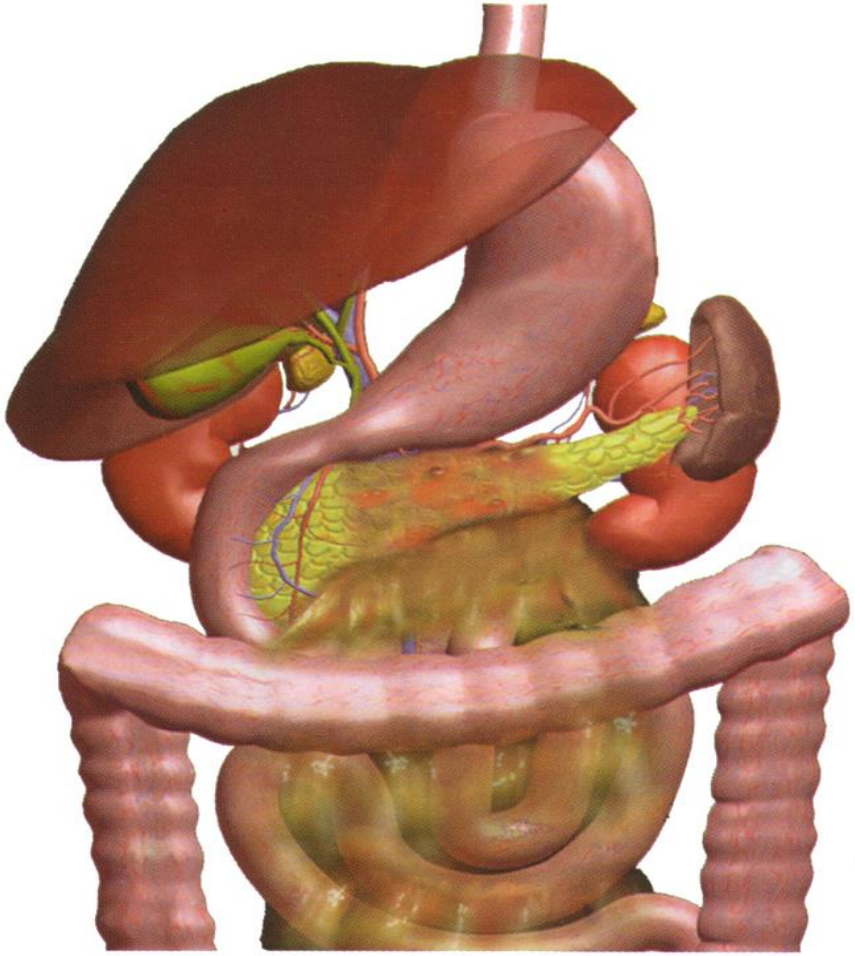
Зона распространения воспалительного процесса при некрозе тела и хвоста ПЖ.

Right type



Зона распространения воспалительного процесса при некрозе головки ПЖ.

Median type



Зона распространения воспалительного процесса при некрозе среднего отдела ПЖ.

Diagnosis of the AP

- 1. Clinical symptoms' evaluation.
- 2. Determination of the activity of the enzymes in the blood: amylase (lipase), in urine(amylase), peritoneal exsudate
- 3. Evaluation and control of the hemodynamic's determinants - arterial pressure, heart rate, shock index Альговера
- 4. Dynamics of the homeostatic determinants
- 5. Scales of severity (Ranson, Glasgow-Imrie)
- 6. Complexe instrumental diagnosis:

1) abdominal US

2) X-rays of the thorax and abdomen

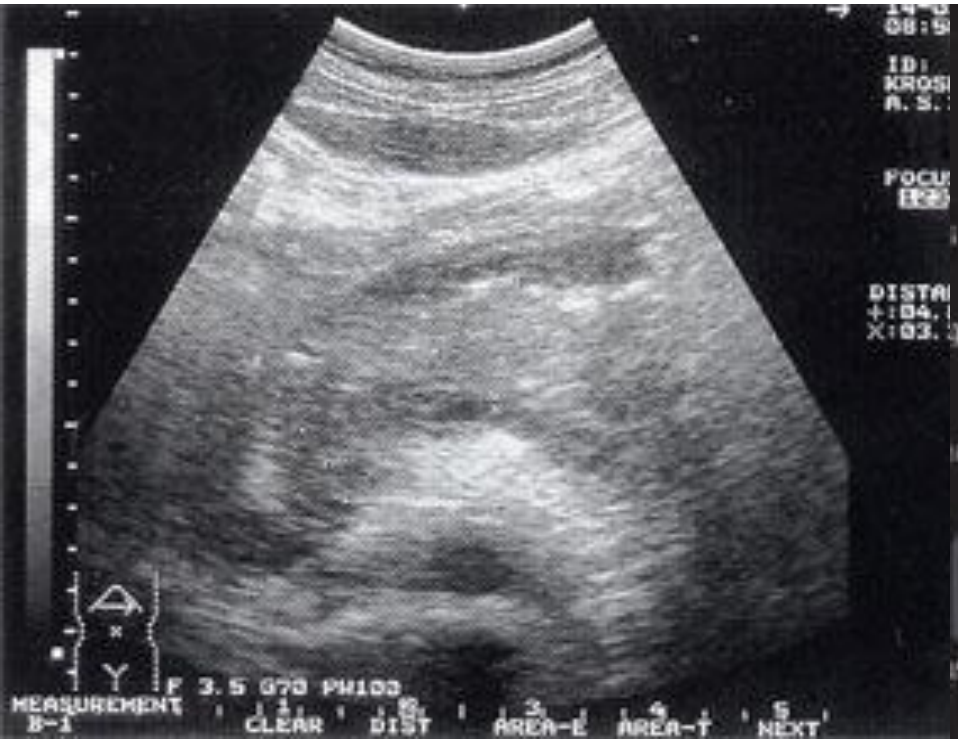
3) **CT**

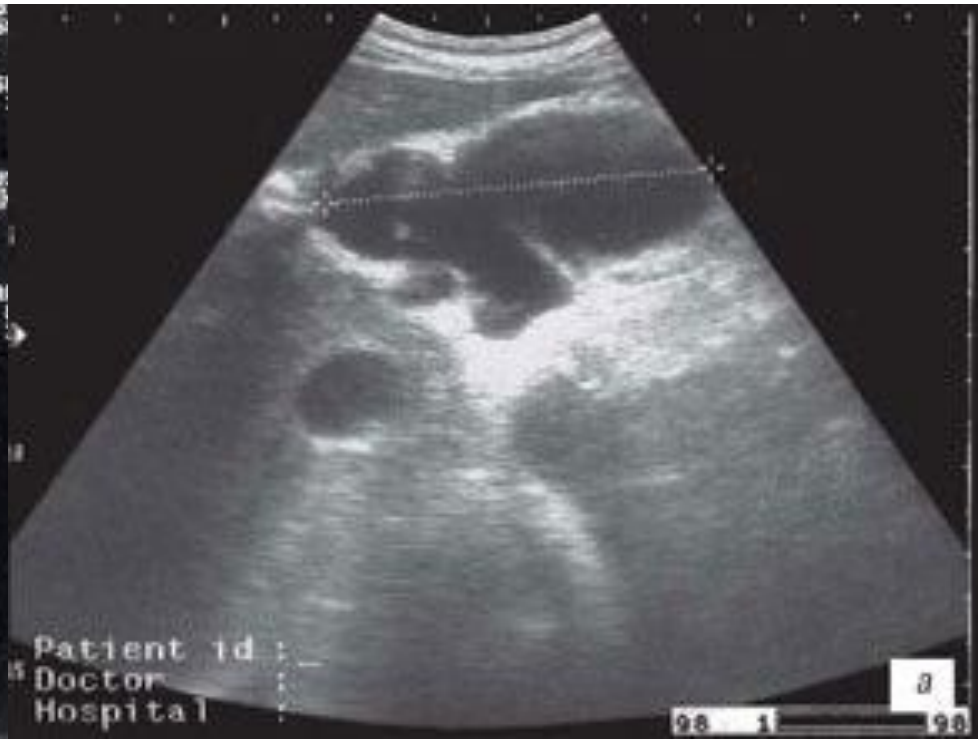
4) laparocentesis

5)laparoscopy

Ultra-sound diagnosis - method of choice

- Increased and fuzzy gland
- effusion in the , peritoneal space

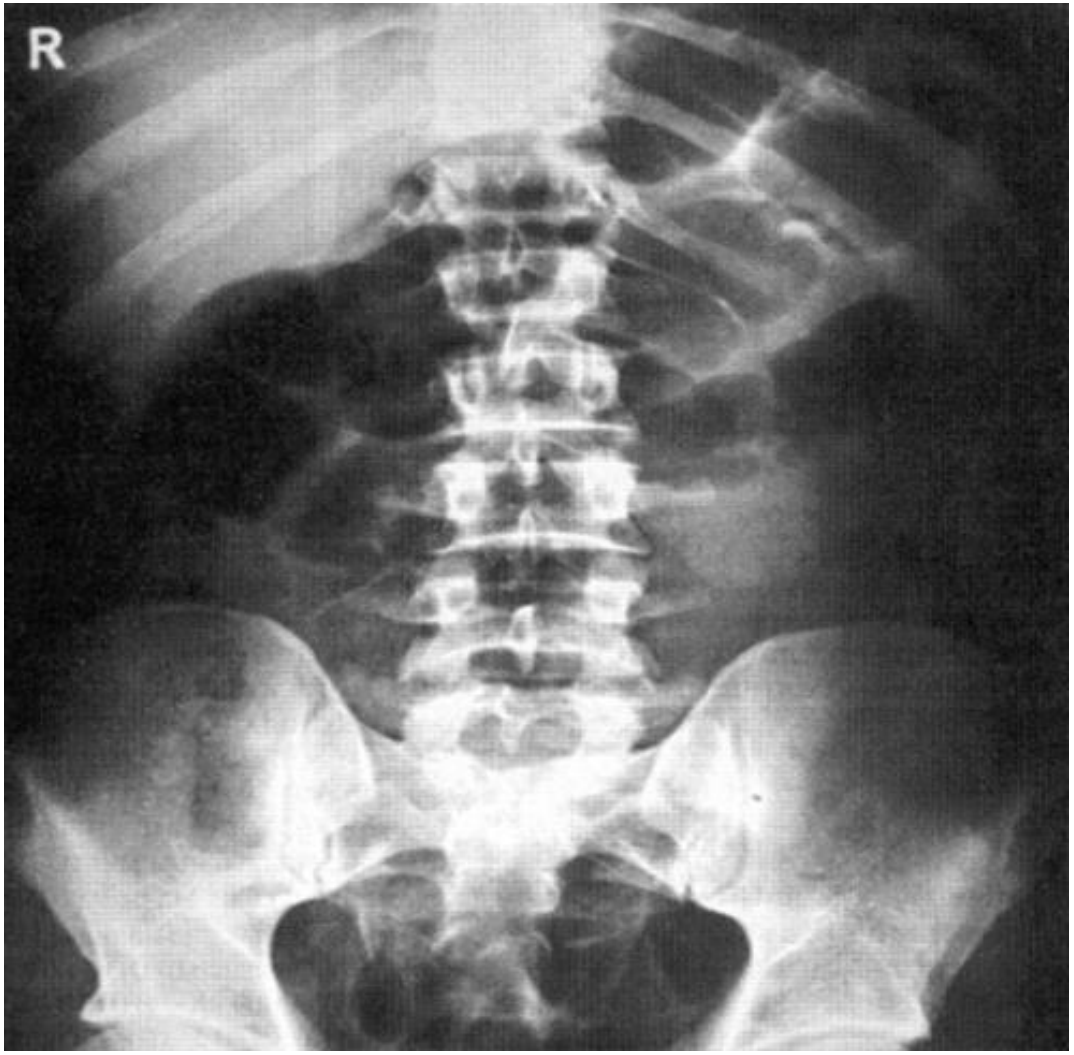




X-rays diagnosis

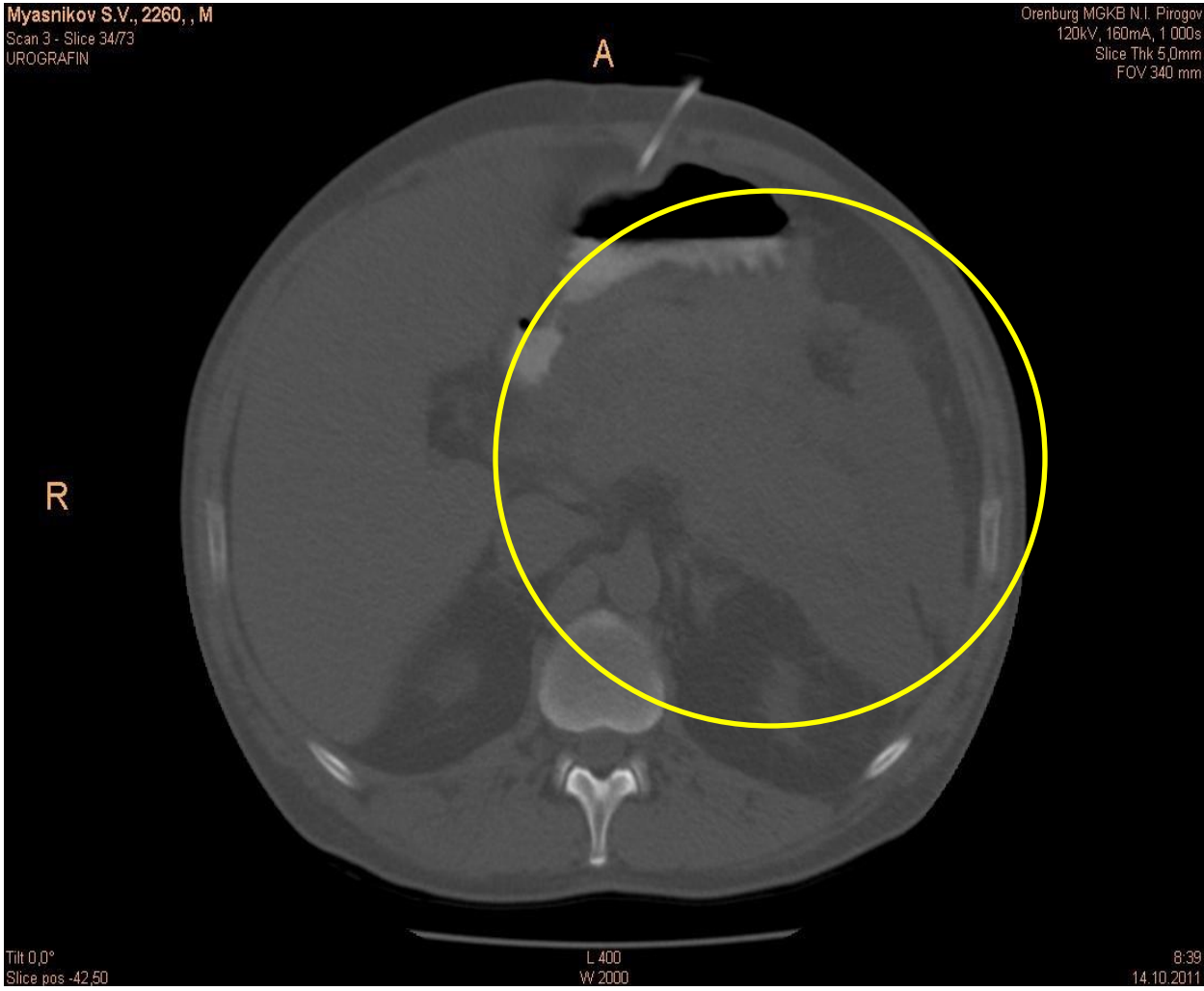
Intestins paralysis**(Gobier's symptom)**

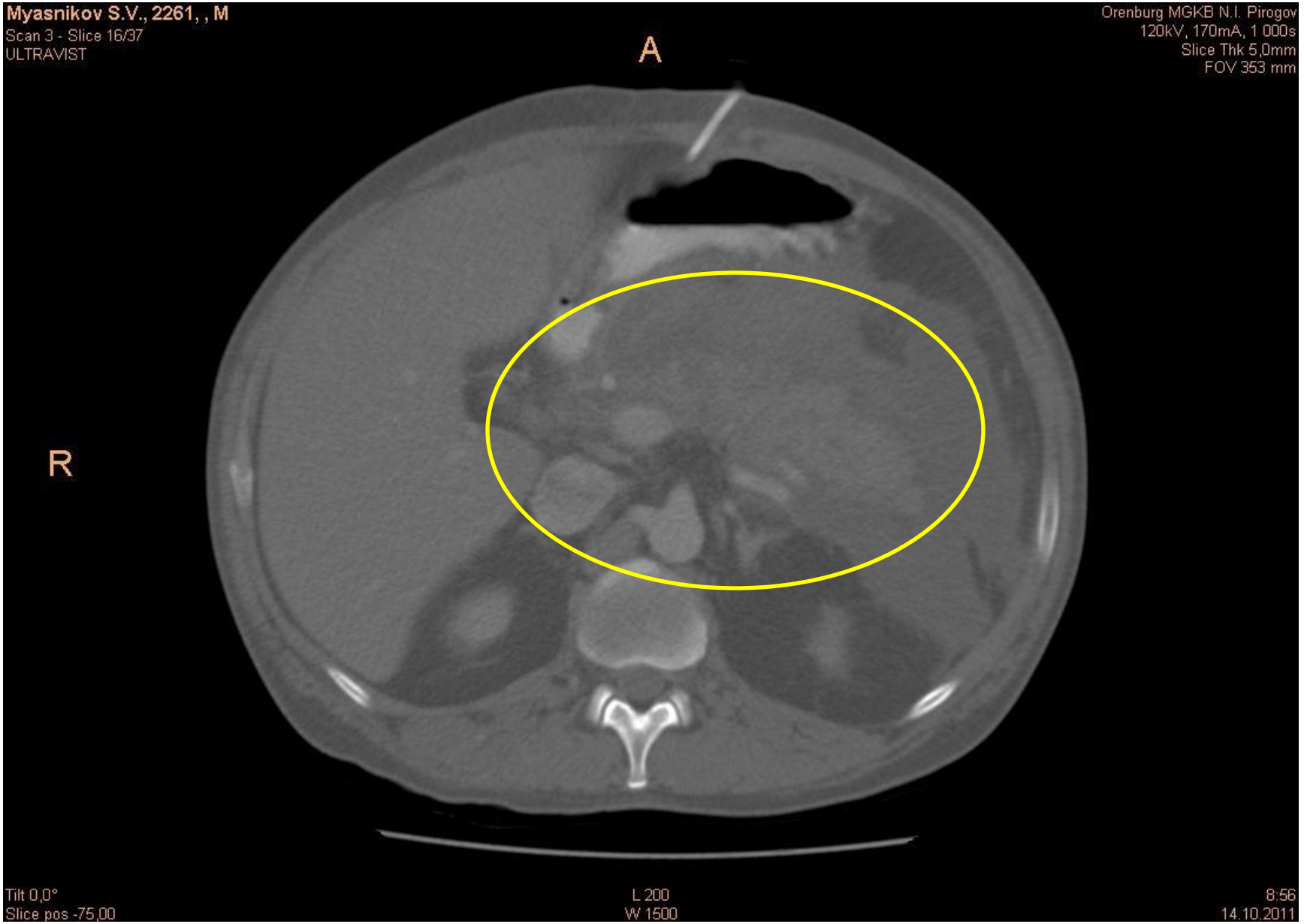
Differential diagnosis with the perforation of the empty organ



CT- method of choice of the abundance of lesion diagnosis

Volume of the peritoneal lesion, effusion liquid





CT with contrast injection - volume of the gland's lesion

CT severity scale (Balthazar)

- **A)** Normal pancreas (0 points);
- **B)** local or diffuse lesion of the pancreas with hypodense inclusions in its tissue with fuzzy contours, pancreactic's duct enlargement (**1 point**);
- **C)** Pancreatic tissues metamorphosis , analogic to the B-stage, with the inflammatory metamorphosis in the peripancreatic cellulose (**2 points**);
- **D)** Metamorphoses + rare liquid formations out of the pancreatic tissue (**3 points**);
- **E)** D metamorphoses + two or more formations out of the pancreas or abscess (**4 points**).

Laboratory diagnosis

Enzymes in the blood flow – Hyperfermentemia (first 2-3 days)

1. Blood amylase increases,diastase in the urine;
2. Blood lipase increases
3. Blood tripsine increases.

More specific and more expensive

in total necrosis there is no fermentemia

Laboratory diagnosis

- Bilirubin, transaminases

- Hyperbilirubinemia if biliary pancreatitis (mechanical jaundice);
- Parenchymatous hyperbilirubinemia (toxic liver lesion); cytolysis
- Increased transaminases (ASAT, ALAT)

Very important detox role of the liver

Calcium level determination (falls) – prognostic marker, necrosis marker.

Protein, urea, creatinine, etc. – evaluation of organ's failure

Evaluation of severity of AP

Very important diagnostic and prognostic stage

→ Prognosis

↓

Choice of the treatment strategy

Specific stratification scales: Ranson, Glasgow-Imrie, Apache II.

Scales of the multi organ's failure: SAPS, SAPS II, SOFA, MODS и тд.

To quantify the severity of the AP (in points) using clinical and lab analysis

Ranson's scale

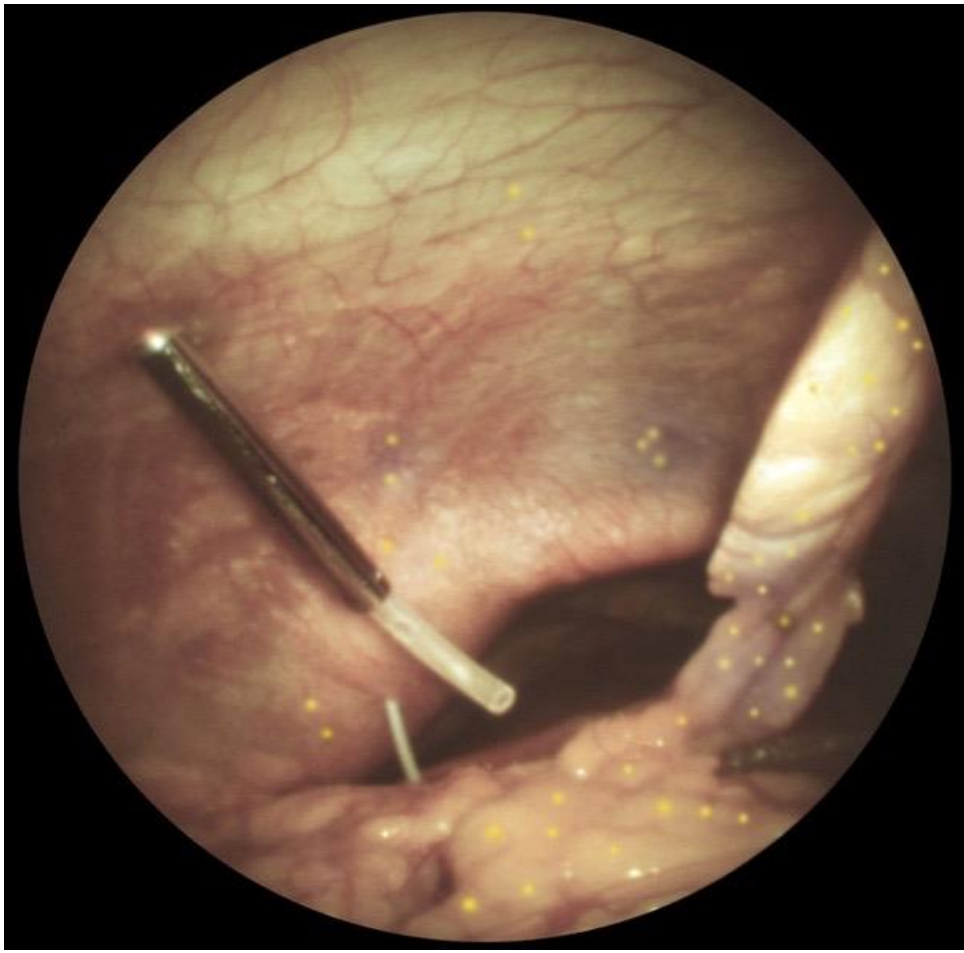
Ranson (alcoholic etiology or other)	Ranson (biliar etiology)
At admission	**At admission**
Age > 55 years	Age > 70 years
Leukocytes > 16 000/mm3	Leukocytes > 18 000/mm3
LDH > 350 U/l	LDH > 250 U/l
AST > 250 U/l	AST > 250 U/l
Glicemia > 200 mg/dl	Glicemia > 220 mg/dl
After 48 hours	**After 48 hours**
Reduction in hematocrit > 10%	Reduction in hematocrit > 10%
Increase in BUN > 5 mg/dl	Increase in BUN > 2 mg/dl
Calcium < 8 mg/dl	Calcium < 8 mg/dl
PO2 < 60 mmHg	PO2 < 60 mmHg
Base excess> 4 mEq/l	Base excess > 5 mEq/l
Fluid leakage > 6L	Fluid leakage > 4L

less than 3 signs – mild AP; 3 and more - severe AP

Laparoscopy

Diagnosis:

1. Steatonecrosis spots,
2. Hemorragic effusion,
3. Oedema, infiltration of the liver ligament, lig. hepatoduodenale, major omentum.



Video: laparoscopy

- Diagnostic stage

Differential diagnosis

- **Perforated ulcer of the stomach or duodenum !!!**
- Acute cholecystitis
- Intestinal blockage
- Intestinal ischemia (acute thrombosis and mesenteric emboly)
- Intestinal infection
- Myocardial infarction (abdominal form)
- inferior lobe pneumonia
- Aortal dissection

If AP diagnosis suspected - hospitalisation in the surgery department
If biliary AP – urgent endoscopic papillotomia

- Two main goals of the treatment

1. To stop the process:

1.1 Recuperation of the duct system function;

1.2 Blockage of the pancreatic secretion.

2. To prefer the aseptic process.

AP treatment

Hospital stage

Diagnostic procedures

Evaluation of the AP severity – **mild or severe AP**

Treatment protocole’s choice

Mild pancreatitis

Basic conservative treatment

In the surgery department

1. No alimentation (table 0)
2. Local hypothermia (ice on the abdomen)
3. Nasogastric sond (aspiration)
4. Infusion therapy and microcirculation lesions corrections in the pancreas:

- infusion therapy 30 ml for 1 kg of boy wight + forced diuresis during 24-48 hours
- rheological treatment (реополиглюкин, трентал)

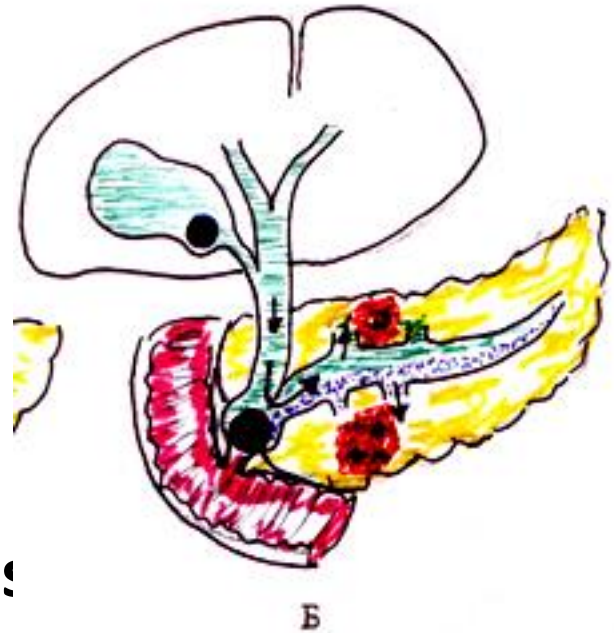
5. **Pancreatic secretion blockage:** М-холинолитики, блокаторы H_2 рецепторов, ИПП, 5-фторурацил
6. Spasmolytics

Mild pancreatitis

Basic conservative treatment

In the surgery department

1. 7. Desensibilising treatment (супрастин, димедрол, пипольфен)
2. 8. Bile excretion control.
3. (if choledocholythiasis, stricture) –
4. ESPT,
5. lithoextraction



7. **9. If no effect during 6 hours = diagnos**
   => hospitalisation in the reanimation department

Severe pancreatitis

Intensive conservative treatment

In the reanimation department

1. **Basic treatment**

2. **Treatment inhibiting the pancreatic secretion**

   - sandostatin

   during 3-5 days

3. **Antifermentive therapy**

   - aprotinin

During 3 days

Severe pancreatitis

Intensive conservative treatment

In the rea

4. **4. Painkillers and antiparalysis therapy**

- epidural anaesthesia
- General blood volume normalisation with normalisation of the central veinous pressure and hematocritis

In reanimation

5. **5. Transfusion:**

- Hemodilution (40-60 ml/kg a day) for hypovolemia extinction;
- crystalloid solutions (0,9 % NaCl, glucose + insuline, лактасол, salt solutions (45-80 ml/kg), Ringer's solution;
- colloid plasma remplacement (полиглюкин, реополиглюкин)
- correction КОС (5% р-р гидрокарбоната натрия из расчёта 2,5-3 мг/кг)

6. Detox therapy:

- Forced diuresis (фуросемид, маннитол, сорбитол); under diuresis control (не менее 4-5 мл/кг в час); contraindications: acute renal failure, chronic renal failure, acute cardiovascular failure, acute myocardial infarction.
- Extracorporal detox methods (hemosorbtion, hemofiltration, plasmapheresis, plasmosorbtion, thoracic canal's dreinage).
- Intestinal lavage through nasointestinal sond with salt electrolyte solution
- Peritoneal dyalysis during laparoscopy

- punction aspiration of the omental liquid with US control

7. Nutritive support (if no intestinal paralysis)

- glucose-salt solutions
- nutritive solutions (берламин, нутризон, нутрилан)

8. Prophylactic antibiotherapy

- меропенем, имипенем
- цефалоспорины III-IV поколений
- фторхинолоны II-III поколений + метронидазол

I. AP during first 24 hours. Strategy

- 1. Effective blockage of the pancreatic secretion (table 0, nasogastric sond, secretoblocators);
- 2. Massive infusion-detox therapy (не <50 мл/кг) – recuperation МЦР;
- 3. Antioxydant and antihypoxy therapy;
- 4. If biliary AP – occlusion's treatment (ЭПСТ, lithoextraction).

- Goal: to achieve the failure phenomenon

II. Sterile necrosis. Treatment strategy

- +

1. Pancreatotropic antibiotherapy (карбапенемы, фторхинолоны, цефалоспорины III-IV), пробиотиков.
2. Immunotrope therapy.
3. Detoxification
4. Intestinal lavage
5. Enteral sond alimentation

Intestin – infection promotor

- Goal: to prefer the aseptic necrosis

II. Sterile necrosis. Surgical strategy minimally-invasive surgery

- Pancreatogenic peritonitis – laparoscopy.
- Parapancreatic liquid content – punction under US control.

- Earl open operation –100% infection chance, lethality - 50%.

Open operation indications

- 1. Absence of the minimally invasive surgery possibility

- 2. Absence of the therapy's effectiveness and progressive patient's condition deterioration (total necrosis or not adequate therapy) – desperation surgery

III. Infected necrosis = abdominal sepsis

- +
- Active surgical strategy:
- 1. minimally invasive methods (punction drainage under US control);
- 2. open surgery

- GOAL: Ubi pus ibi incisio

Surgical indications in pancreonecrosis cases

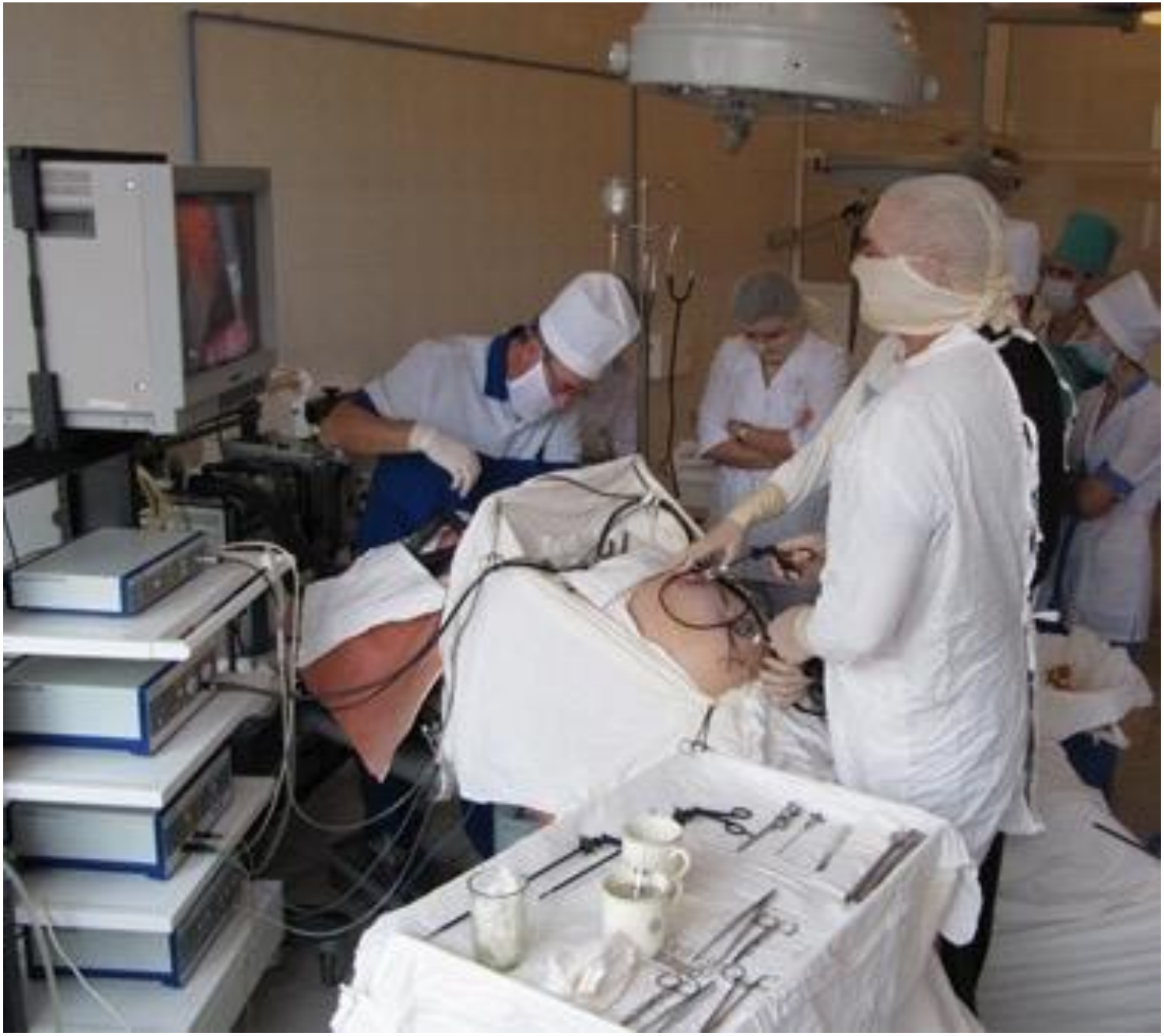
1. Pancreatogenic abacterial fermentative peritonitis – laparoscopy
2. Acute liquid parapancreatic content – US-controlled punction (drainage)
3. Constant or progressive multiorgan failure, despite adequate complexe intensive conservative therapy– laparotomy
4. Infected pancreonecrosis:

4.1. Pancreatogenic abscess – US-controlled drainage

4.2. Septic flegmona of the parapancreatic oe paracolic cellulose, suppurative peritonitis – laparotomy, lumbotomy

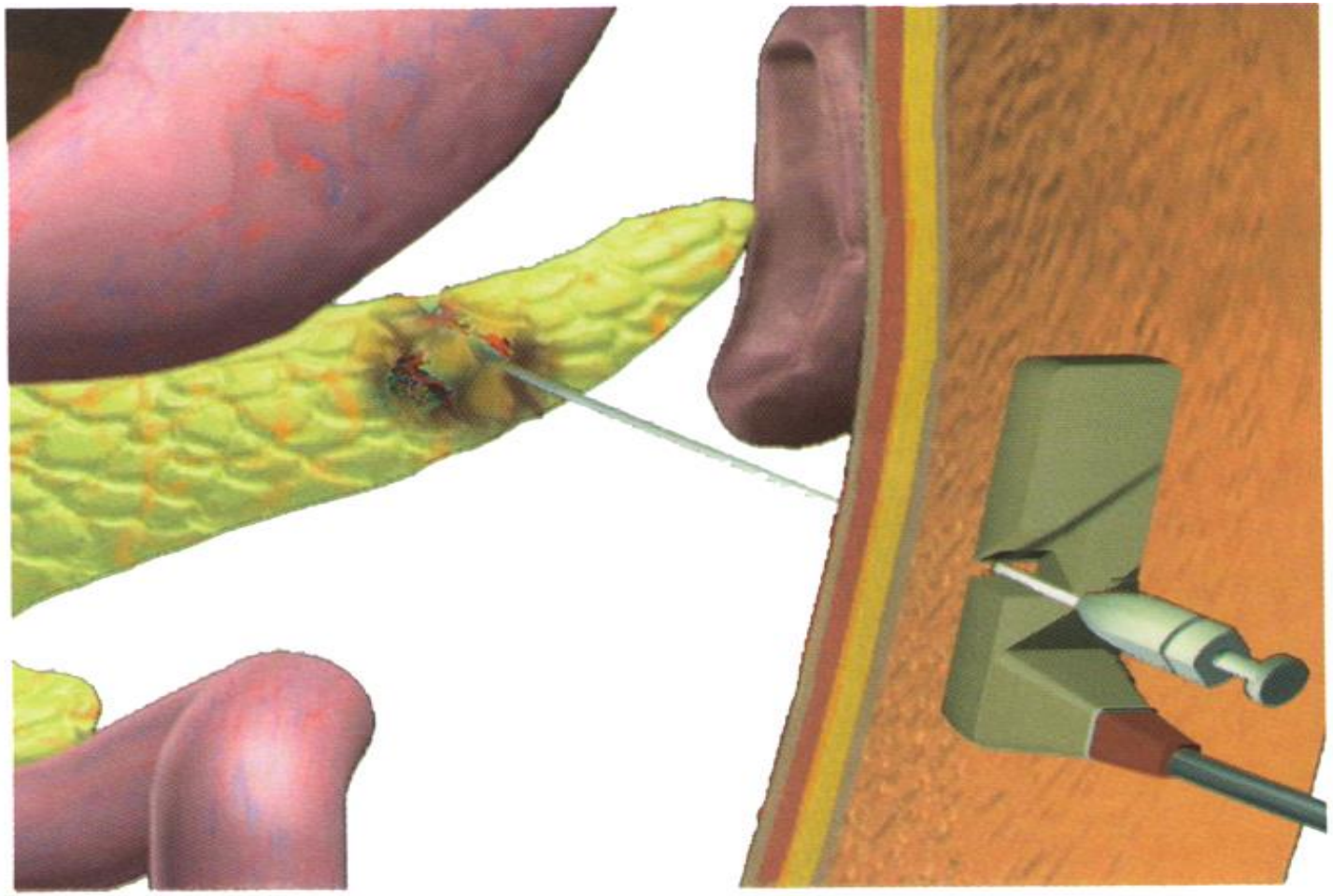
The goal of the surgical treatment – surgical detoxication

1. Hemorragic exsudate's removal from the abdominal cavity in order to prevent the enzymatic toxicity, abdominal cavity's drainage.
2. Omentum's drainage with pancreas' decompression by parietal peritoneum's section around the gland.
3. Rational drainage of the retroperitoneal space for the purulent source's sanation from toxic products of the inflammation and necrolysis

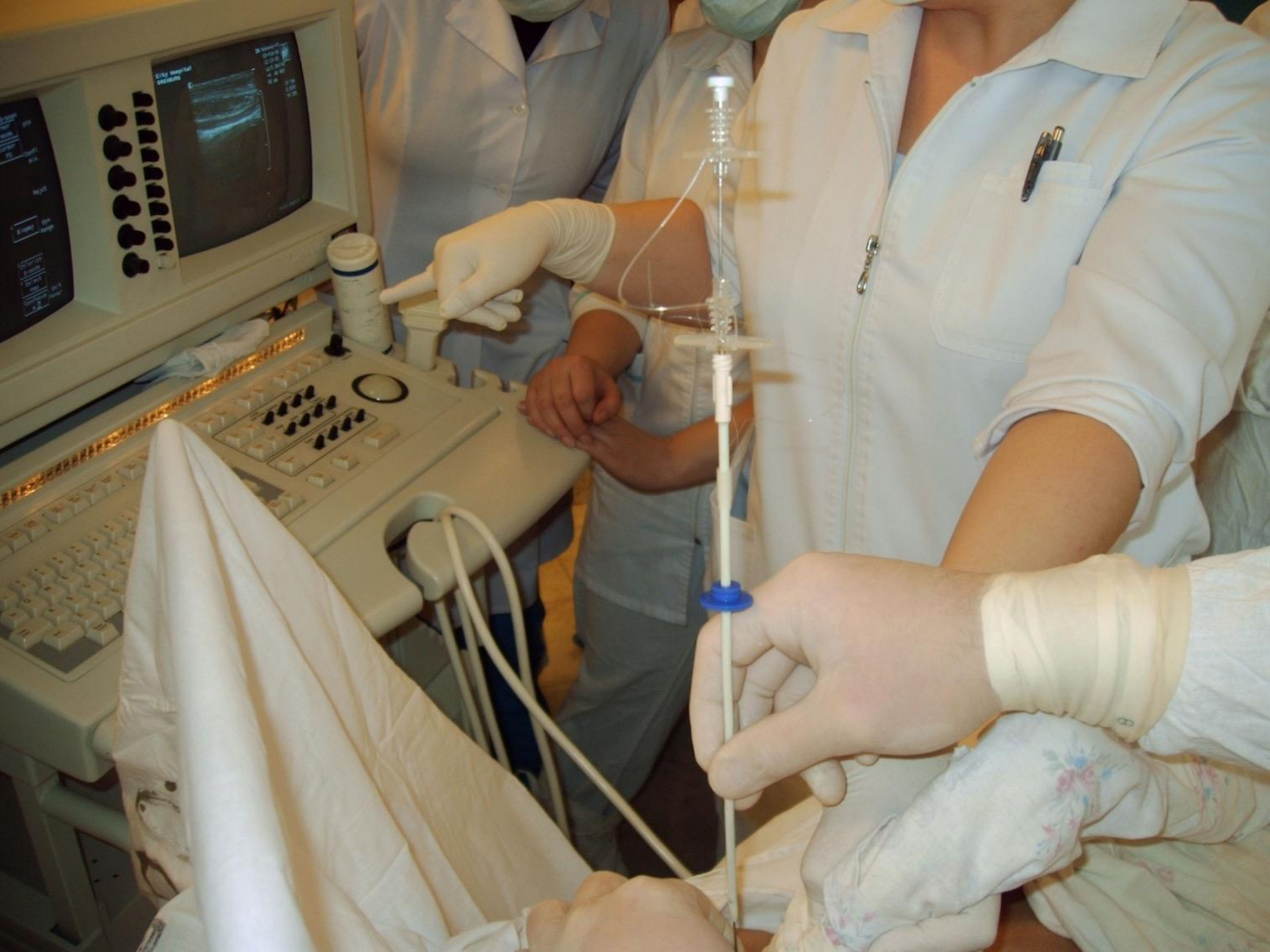


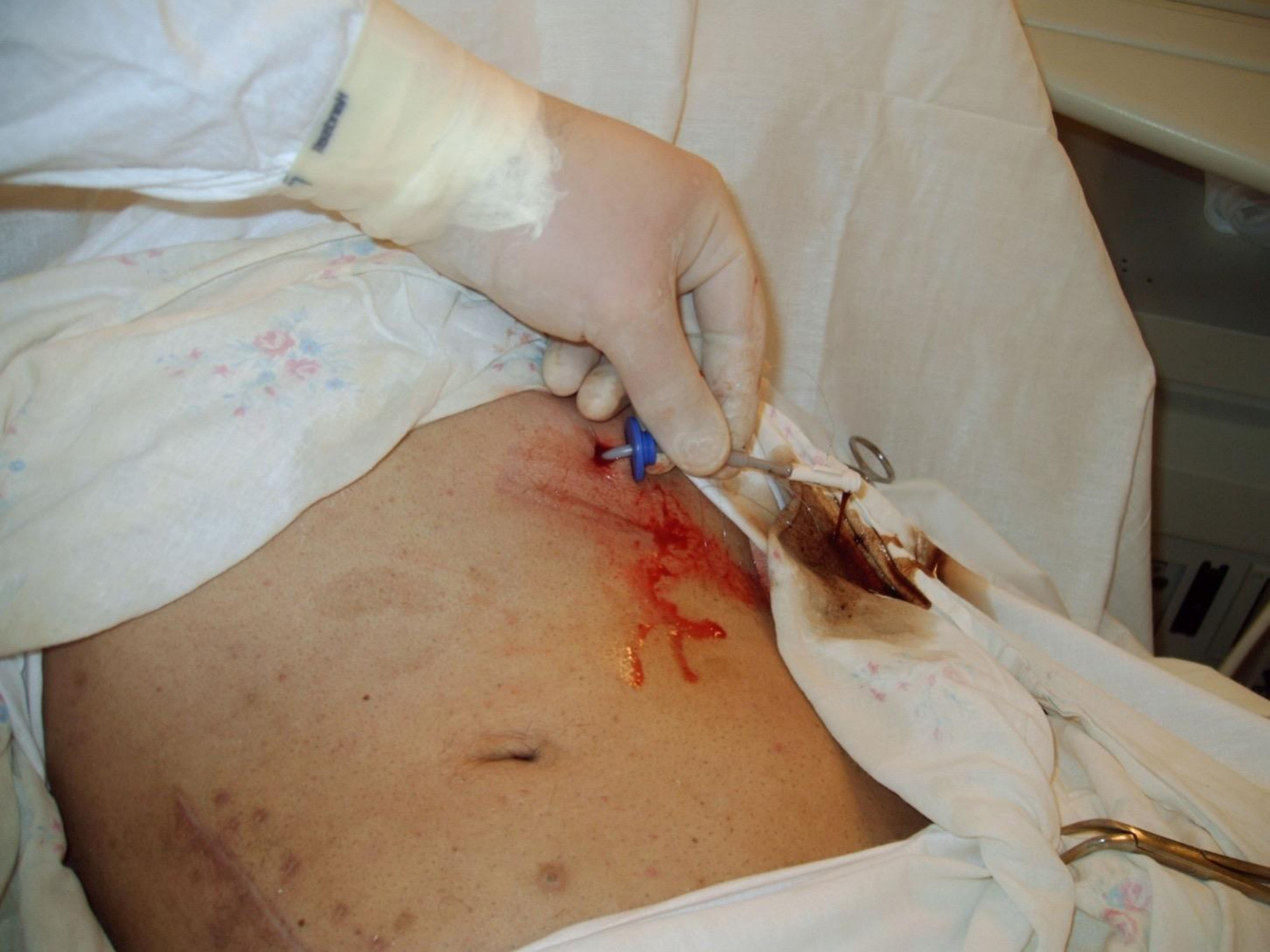
Laparoscopy, video

US controlled punction



Диагностическая пункция постнекротической кисты ПЖ.

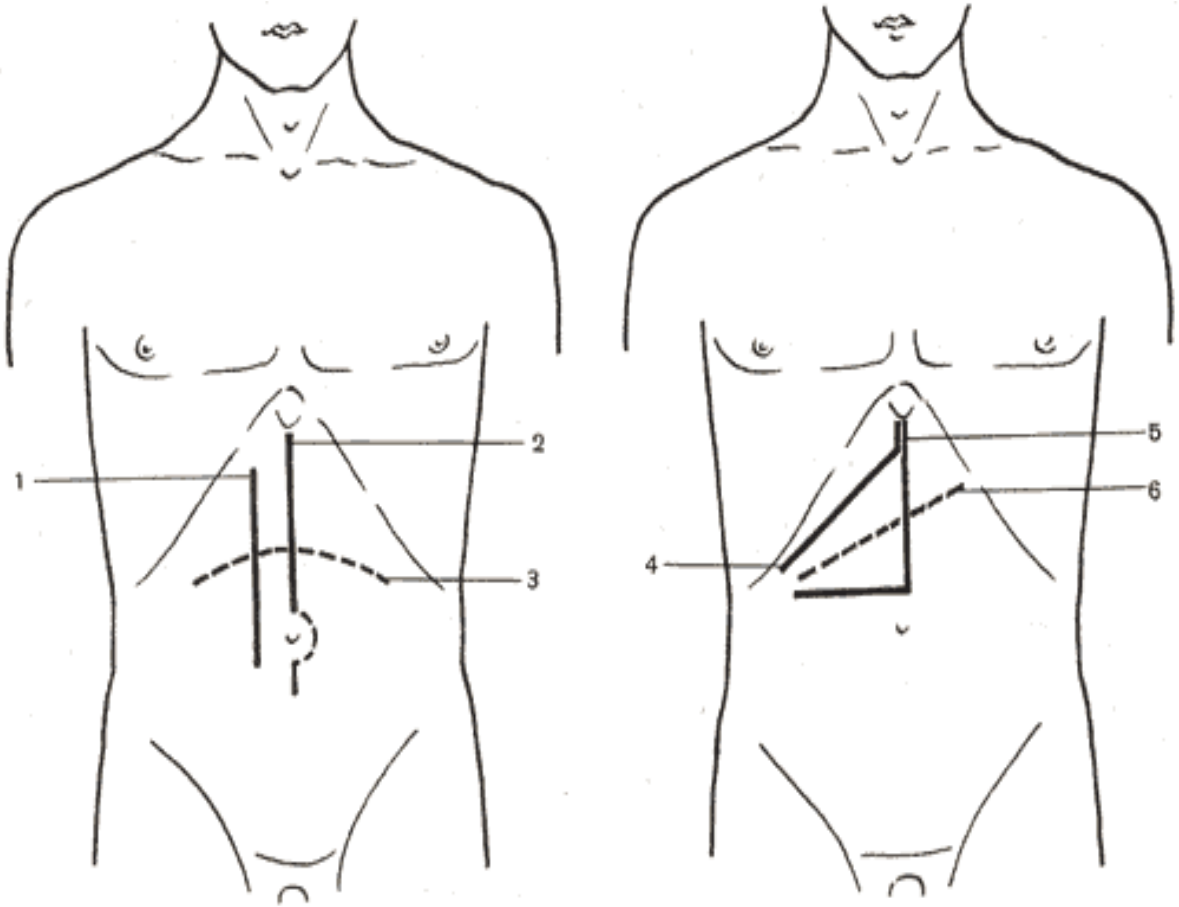




Surgicals accesses to the pancreas

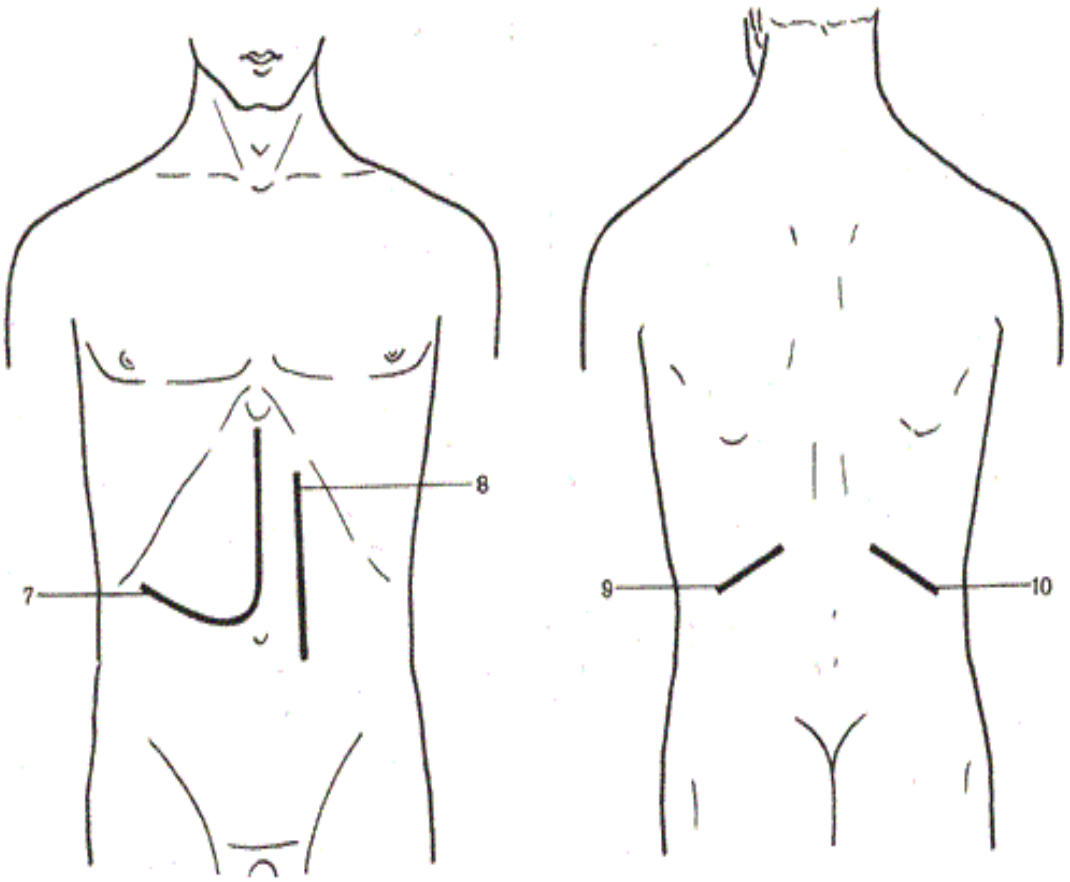
- Anterior abdominal wall
- Lumbar accesses

Surgical accesses



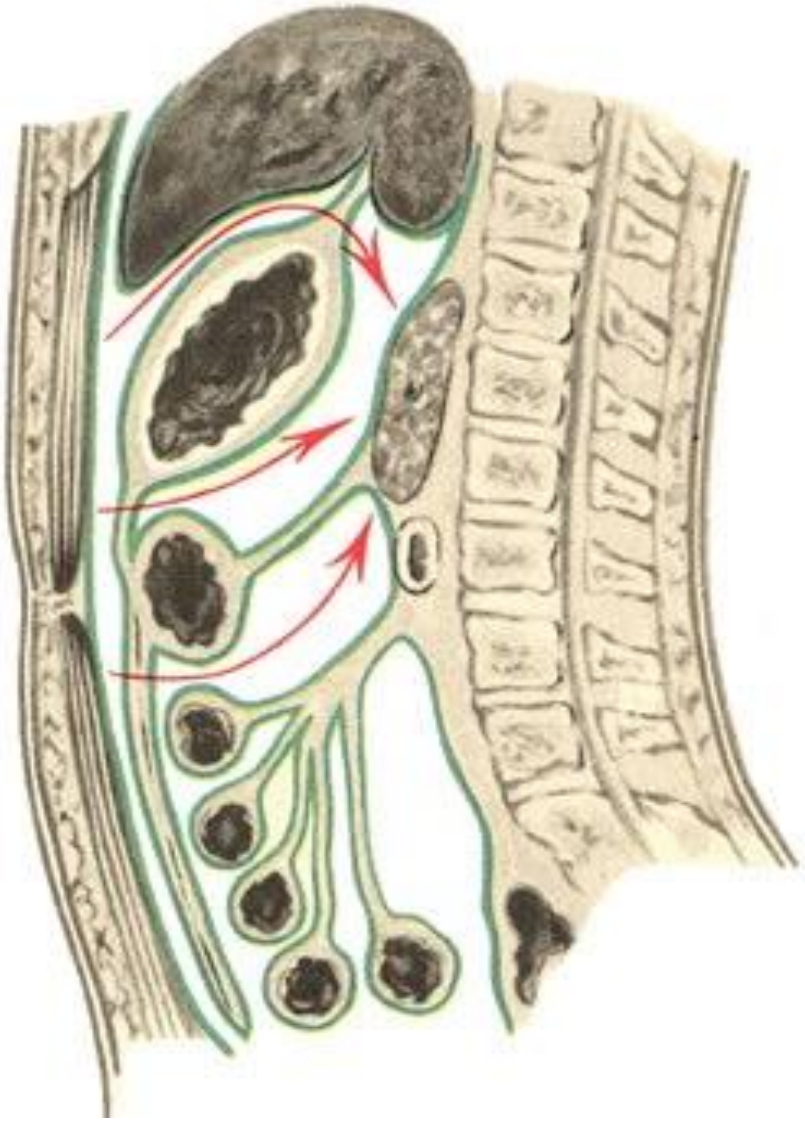
1 — right transrectal section
2 — superior median section
3 — horizontal section (Shprengels’)
4 — diagonal section (Fedorov’s)
5 — angle section (Tcherni)
6 — косопоперечный разрез (Аирд)

Хирургические доступы к поджелудочной железе

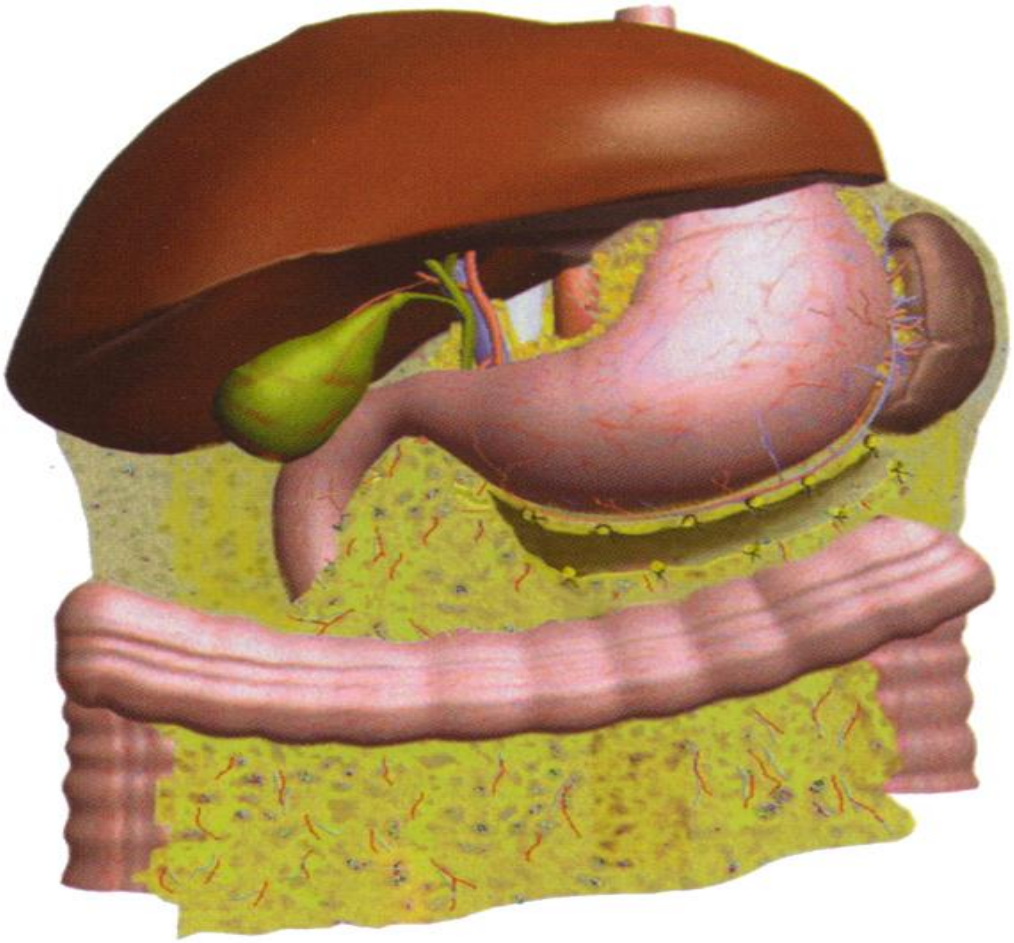


7 — angle section (Ryo-Branco)
8 — left transrectal section
9 — lumbar section fot the body's and queue's access
10 — lumbar section for the gland's head surgery

Schematic view

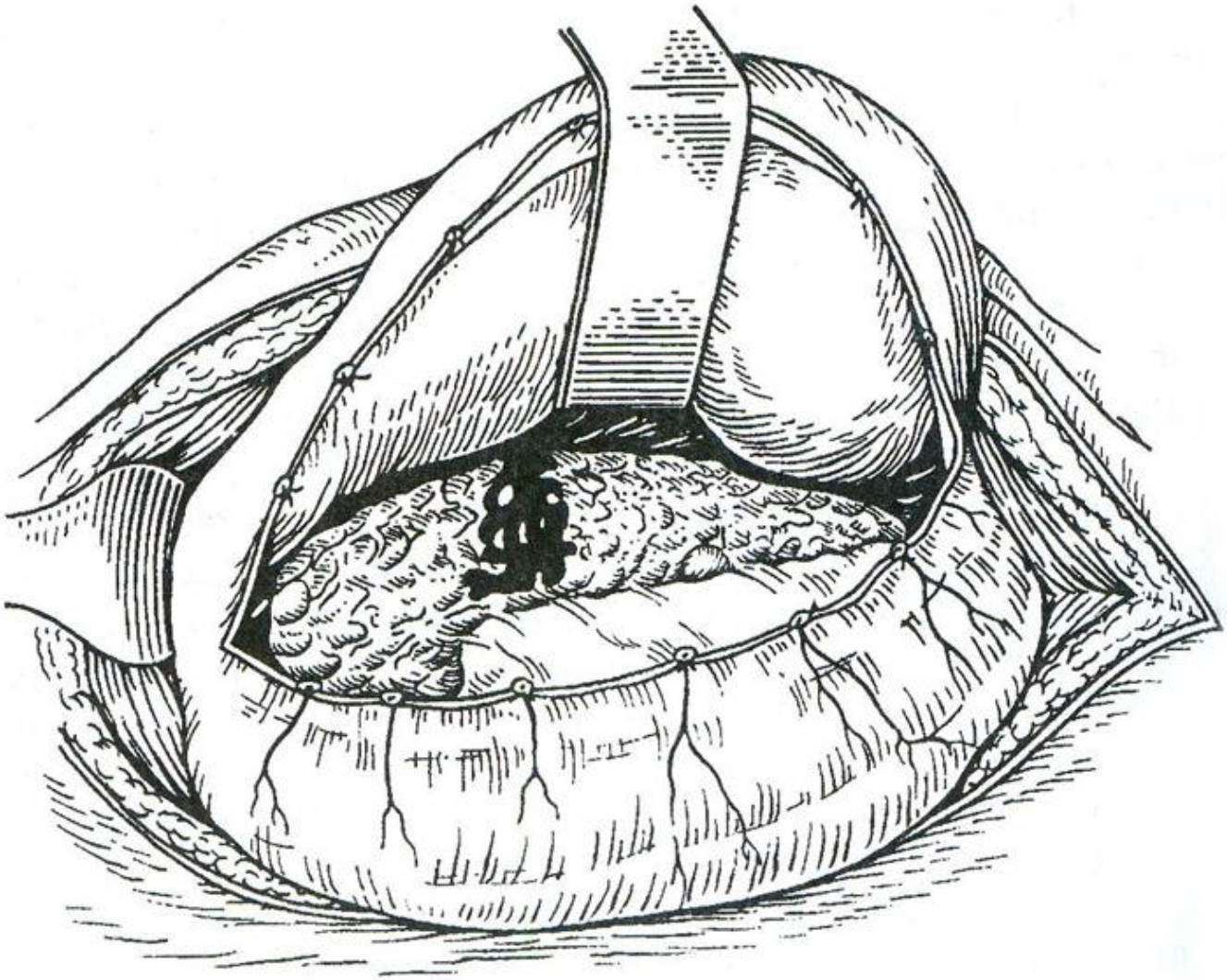


Omental revision

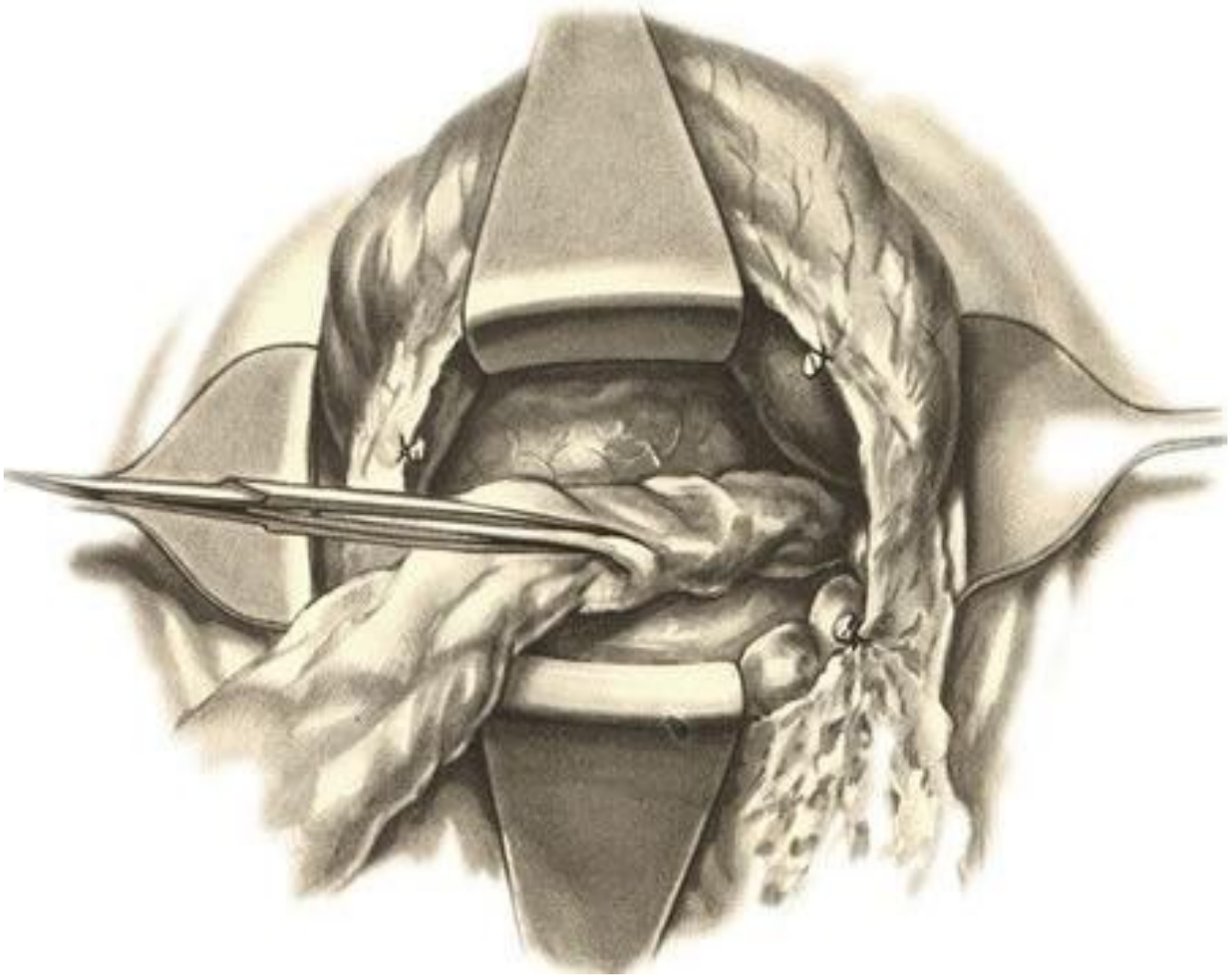


Широкое рассечение желудочно-ободочной связки для ревизии ПЖ.

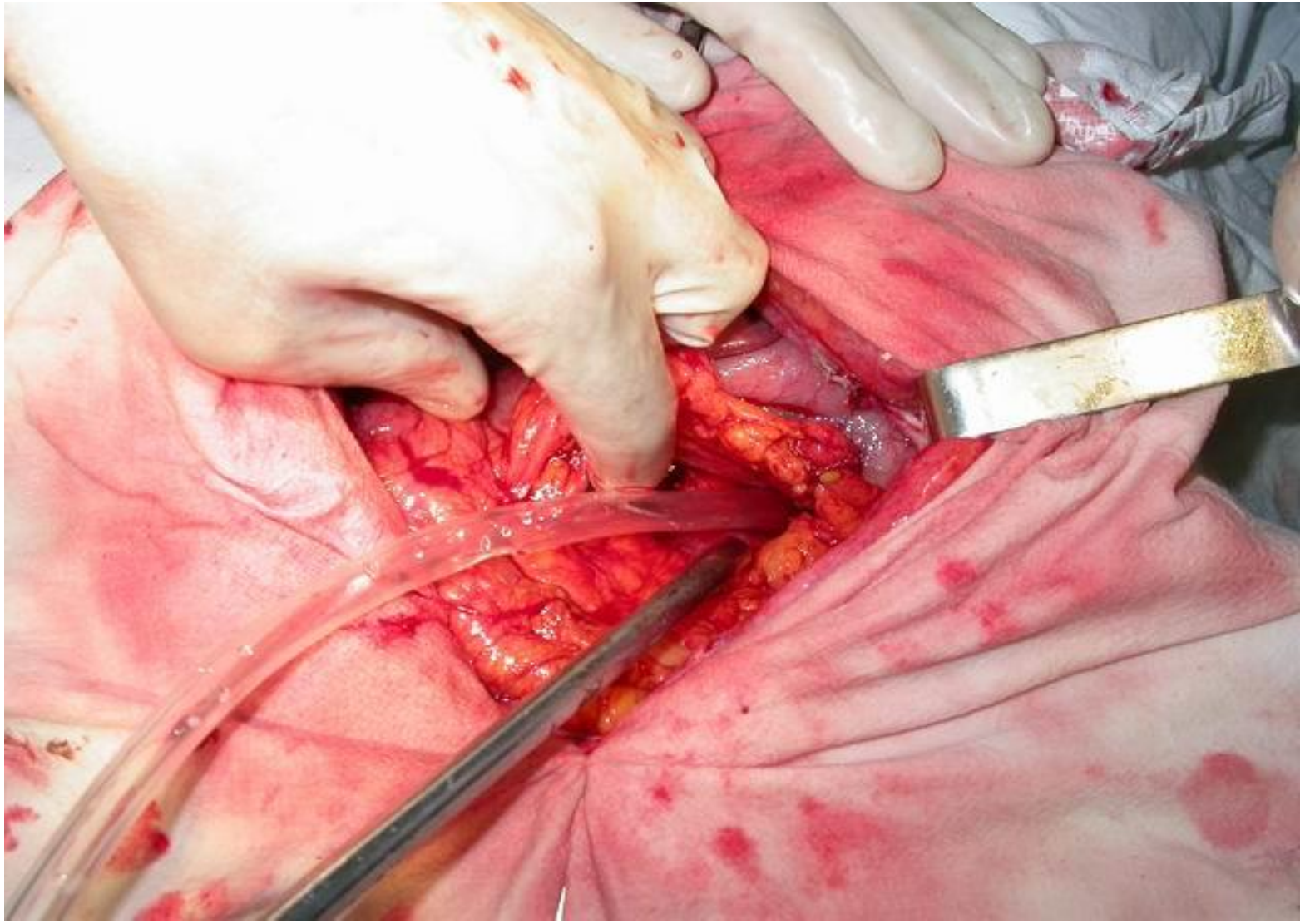
Omental revision



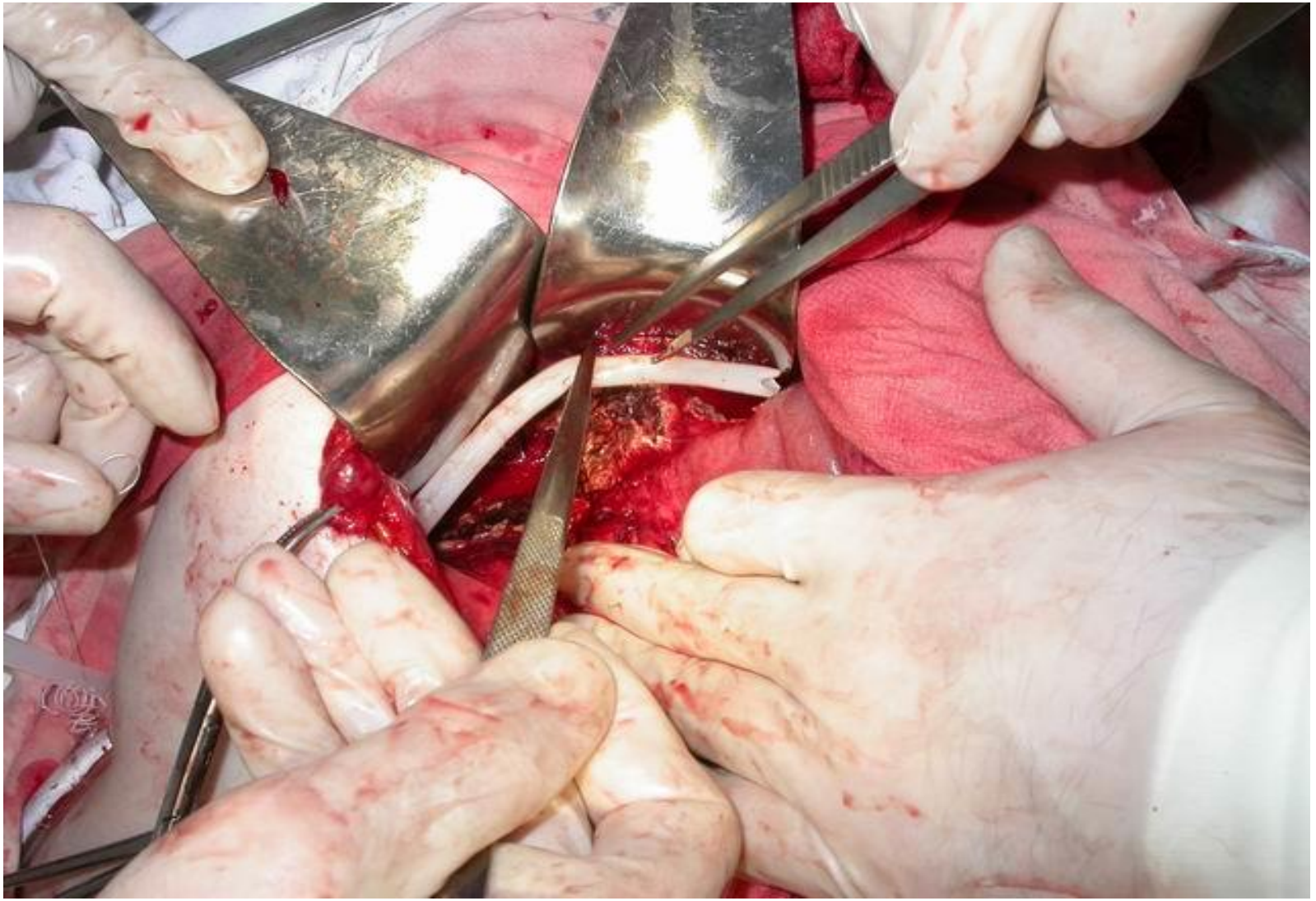
Operation during AP
Tampons into the section of the gland's capsule

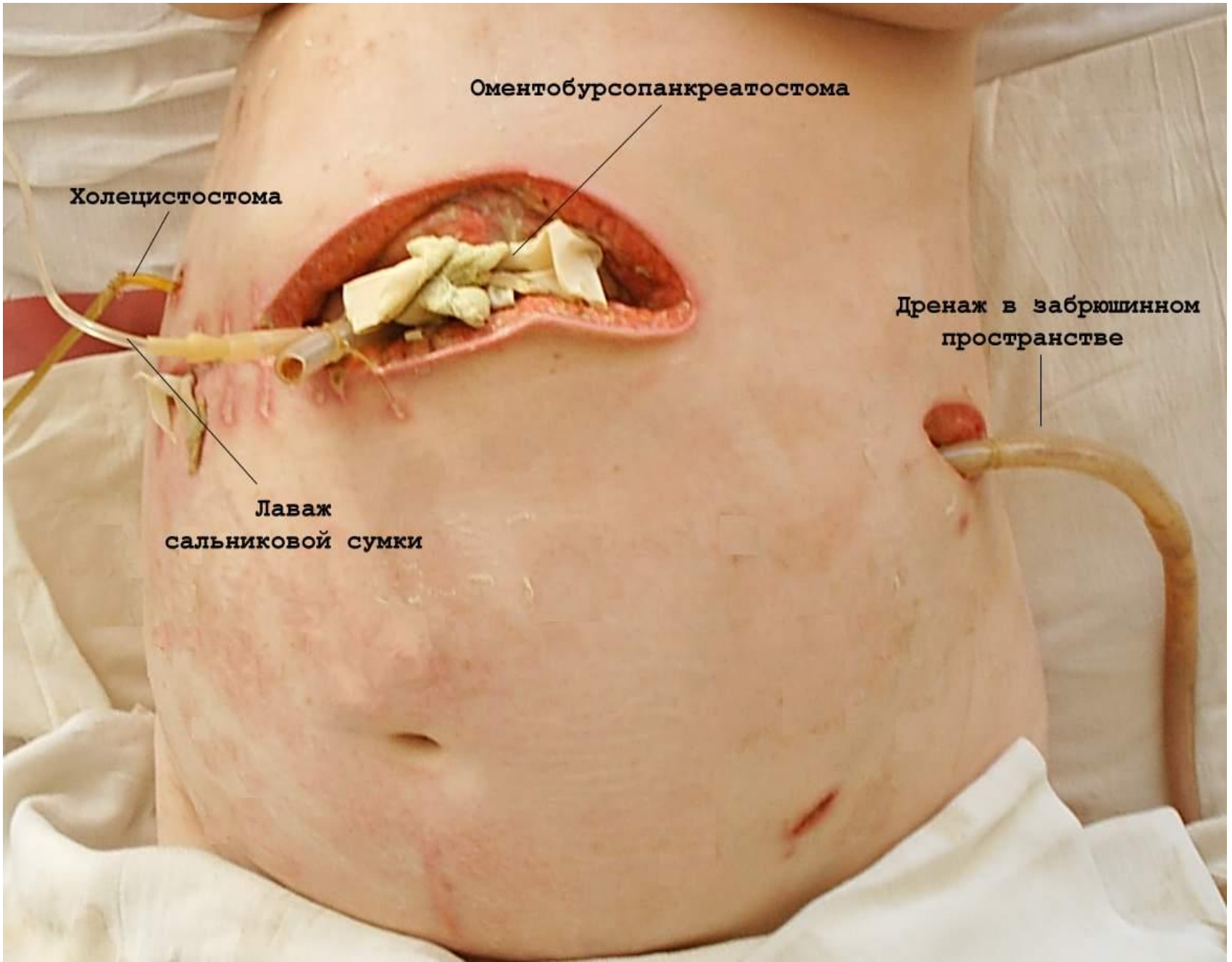


Operation during pancreonecrosis
Penetrating drainage into the omentum



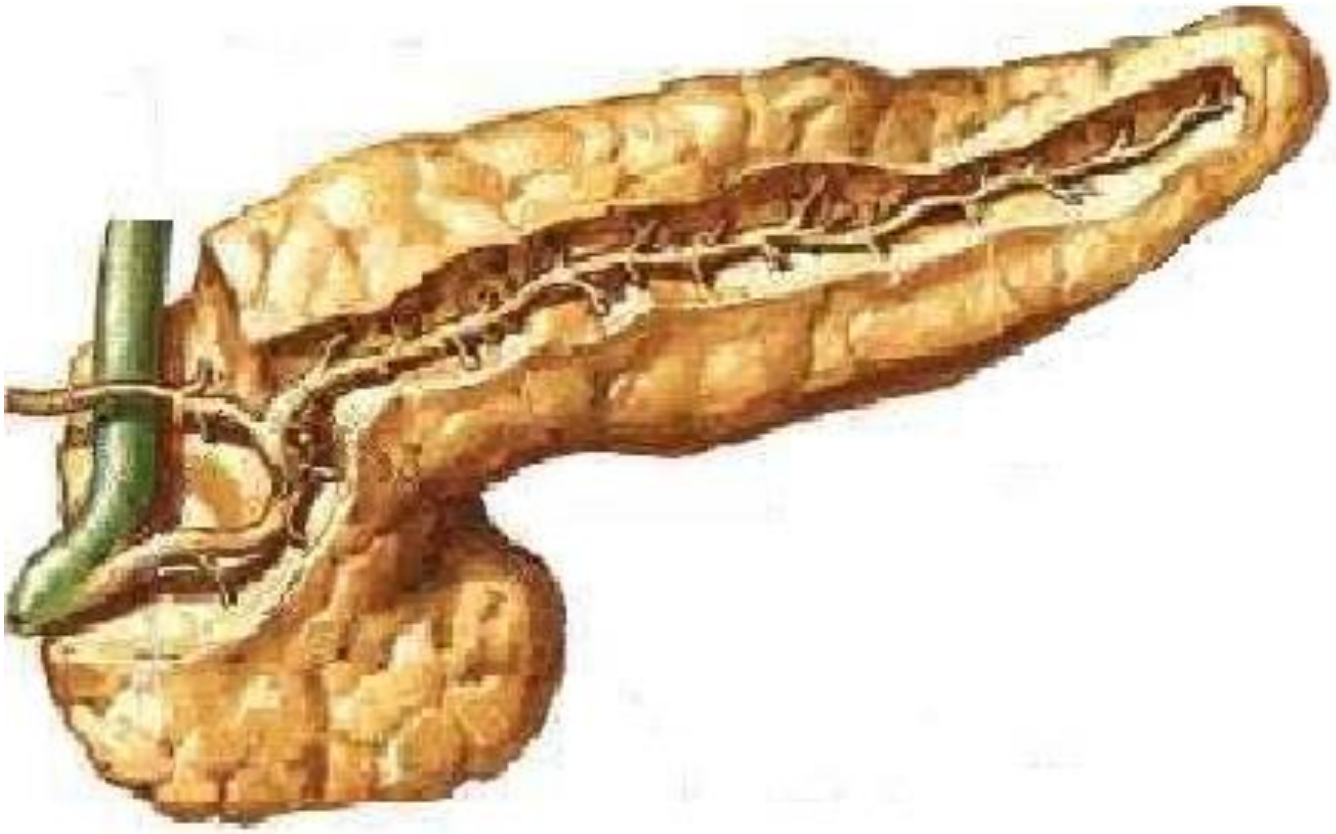
Establishing of the penetrating drainage




Оментобурсопанкреатостома
Холецистостома
Дренаж в забрюшинном пространстве
Лаваж сальниковой сумки

Orenburg State Medical University
Surgery Department

AP complications



Classification

Complications of the AP:

I. Parapancreatic infiltrat.

II. Pancreatic abscess.

III. Peritonitis: fermentative (abacterial), bacterial.

IV. Septic flegmona of the retroperitoneal cellulose: parapancreatic, paracolic, pelvic.

V. Erosive hemorrage.

VI. Mechanical jaundice.

VII. Psudocyst: sterile, infected.

VIII. Internal and external digestive fistulas.

Complications of the AP

Complications of the pancreatogenic toxemia

I. Pancreatogenic shock

II. Shock organ syndrome:

1. Encephalopathy
2. Acute cardiovascular failure
3. Acute respiratory failure
4. Acute liver failure
5. Acute renal failure
6. Enteral failure

Complications

Pancreatogenic destruction's complications

I. Aseptic complications:

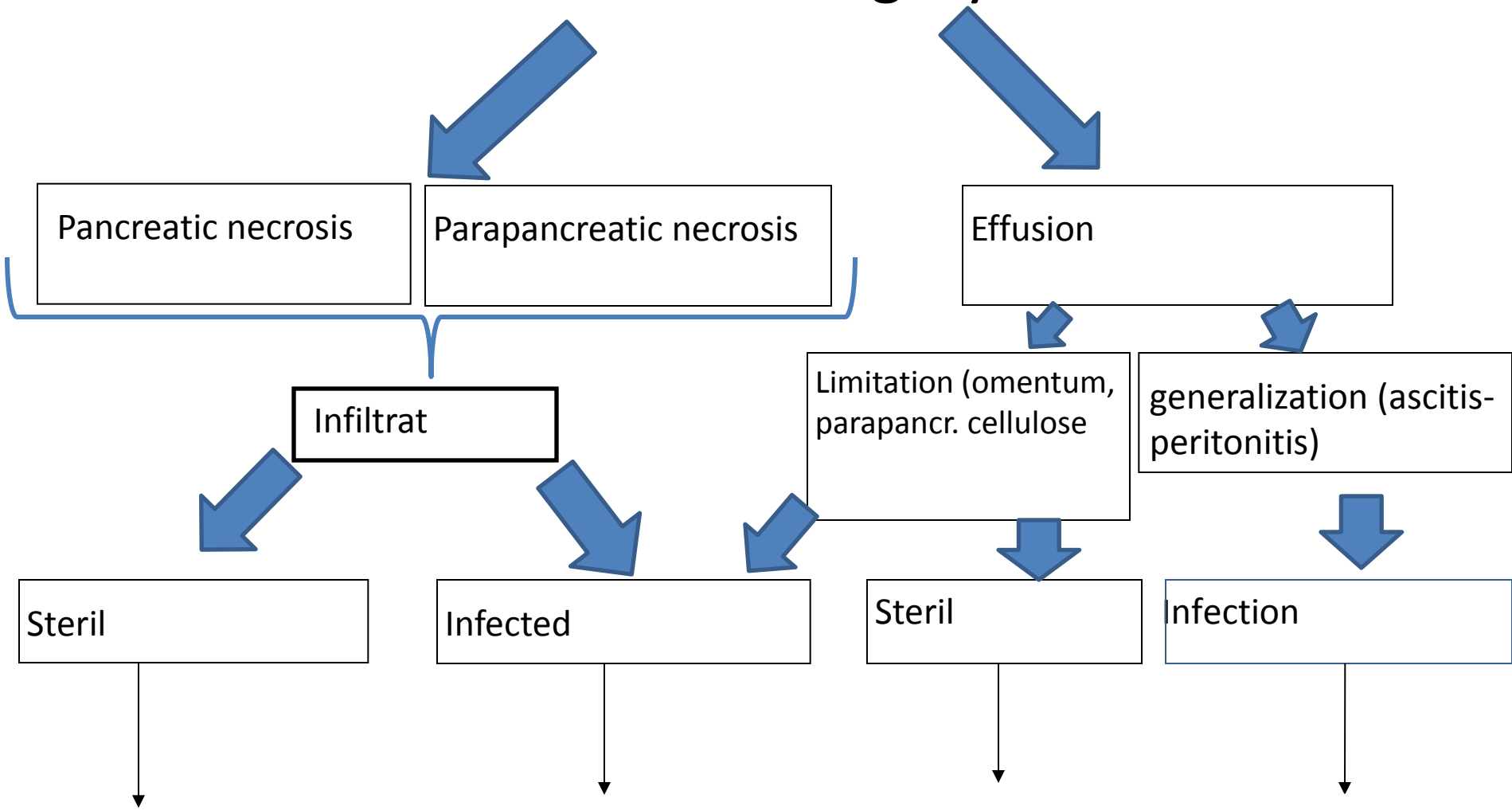
- *Free* pancreatogenic effusion in the peritoneal cavity (fermental *ascitis*-peritonitis);
- Limited effusion (parapancreatic liquid formation);
- Parapancreatic infiltrat;
- Sequesters;
- Pancreatic pseudocyst;
- Diabetis mellitus.

Complications

II. Septic (purulent complications)

- Bacterial (suppurative peritonitis);
- Abscess (pancreatic, omental, retroperitoneal);
- Retroperitoneal flegmona;
- Pancreatic fistula (external, internal)
- Erosive hemorrage
- Other complications

Pancreonecrosis algorythme



Infection of the pancreonecrosis

1. Endogene way:

- duodenopancreatic reflux (unlikely);
- Bacterial translocation of the intestins (intestins – sepsis promotor). ⟹ Earlier the gastro-intestinal tract begins to be functionnal, less the risk of the infection: intestinal resistance to the colonisation, abusive intestinal colonisation syndrome

2. Exogene way:

Surgical intervention (any). Risk increases with the operation's size.

Pancreatogenic effusion in the peritoneal cavity (ferment *ascitis*-peritonitis)

- Peritoneal symptoms with growing symptoms of the peritoneal irritation.
- Liquid in the peritoneal cavity during US.
- Strategy – laparoscopic drainage of the abdominal cavity

Limited effusion

1. Acute parapancreatic effusion (before 2 months, no capsule);
2. Pseudocyst (more than 2 months, capsule formation).

- US diagnosis
- Firstly, conservative strategy (complexe AP treatment).
- Disappears spontaneously (30-50%).
- If no resorption, growth, infection signs – intervention needed (US-controlled punction laparotomy).

Parapancreatic infiltrat

- Inflammatory tumor, conglomerate of losely fixed to one another tissues around the pancreas
- with participation of parietal peritoneum, big omentum, stomach, transverse colon, lig. gastrocolicum, retroperitoneal cellulose.
- Infiltrat – limitation, protection reaction

1. Formation (loose) of infiltrat – 3-4 days;
2. Formed (dense) infiltrat – after 5 days.

Parapancreatic infiltrate

Symptoms:

- Less abdominal pain;
- Better general patient's condition;
- Dense, not painful, fixed formation in the epigastrium,
- Infiltrate's size can vary a lot, can occupy all the upper abdominal region;
- There can be positive local symptoms and peritoneal symptoms - negative;
- Constant SIRS symptoms with the decreasing tendency

Parapancreatic infiltrate

- Diagnosis: US, CT.
- Treatment – AP treatment.
- Results:

1. **Resolution:** pain descreases, infiltrate dissapears, normalisation of the body temperature – Diminishing of the AP symptoms.
2. **Infection** → operation (minimally invasive → open interventions)

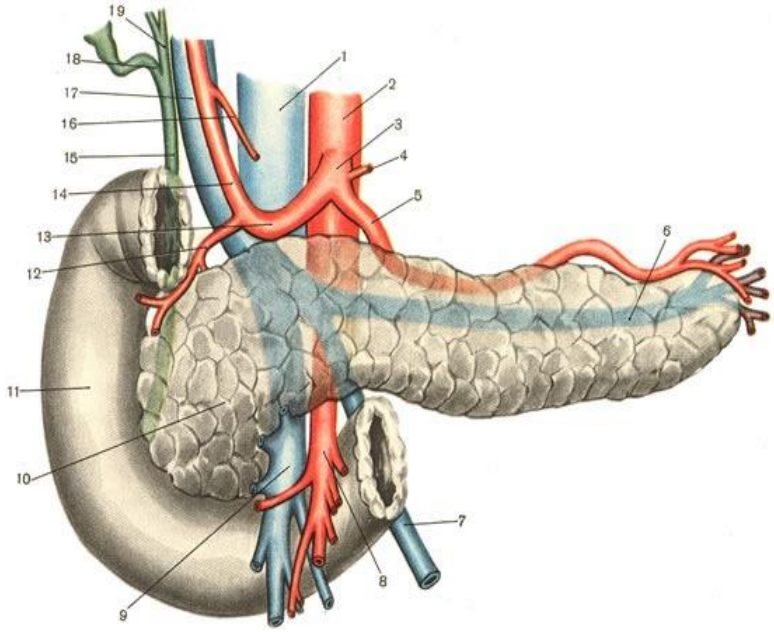
Limited infected effusion

1. Pancreatic abscess.
2. Infected psudocyst.
3. Septic flegmona of the retroperitoneal cellulose: parapancreatic, paracolic, pelvic.

   symptoms, diagnosis, treatment (in the acute appendicitis lesson).

   Retroperitoneal flegmona - dissection and drainage out of the peritoneum (lumbotomy).

Erosive hemorrage



Into the suppurative cavity conditions:

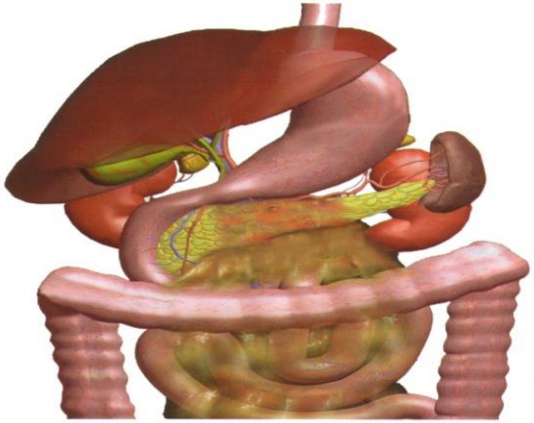
- around the pancreas there is
- a lot of main vessels;
- massive destructive process;
- Surgical intervention earlier.

Symptoms: general (hypotony, tachycardia, etc) and local (wound hemorrhage) blood loss symptoms.

Strategy: urgent intervention – tamponnade or closing, vessel's ligature.

Digestive fistulas

- Pancreaic fistulas;
- Intestinal fistulas (stomach, duodenum, small intestin, colon – colon transversum)



Зона распространения воспалительного процесса при некрозе среднего отдела ПЖ.

Teason – erosion of the empty organ's wall during suppurative necrotising process

Symptoms: presence of the empty organ's content in the wound . Degradation of the wound process (from duodenum - active pancreatic ferments, colon – agressive flora). Loss of the empty organ's content – progressive weight loss.