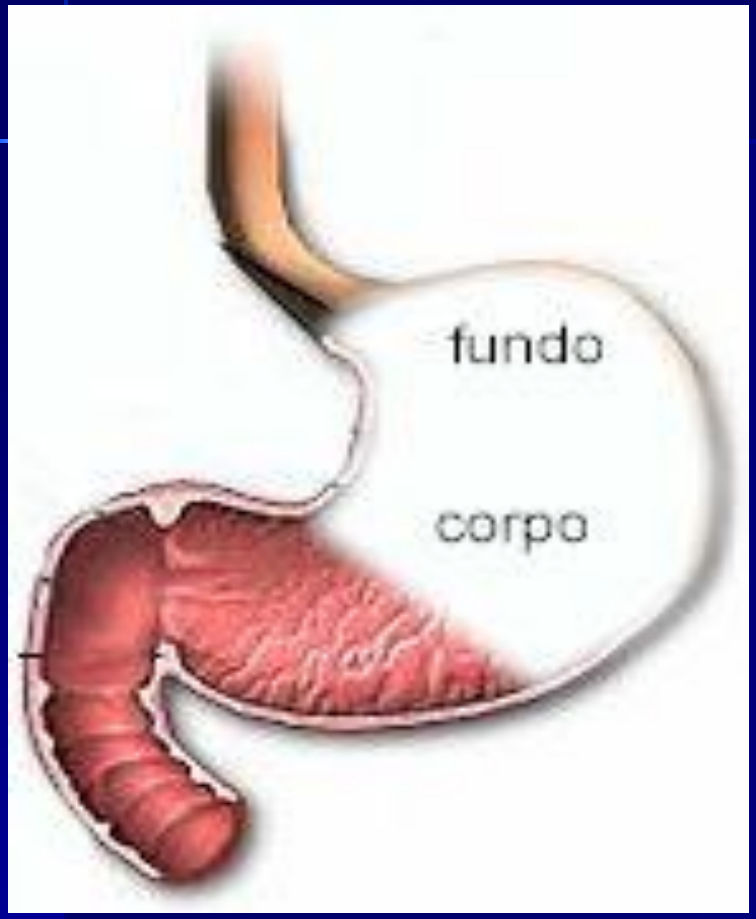
# Clinical syndromes in gastrointestinal tract diseases. Examination of patients with gastritis, gastric and duodenal ulcer.
# Gastric cancer.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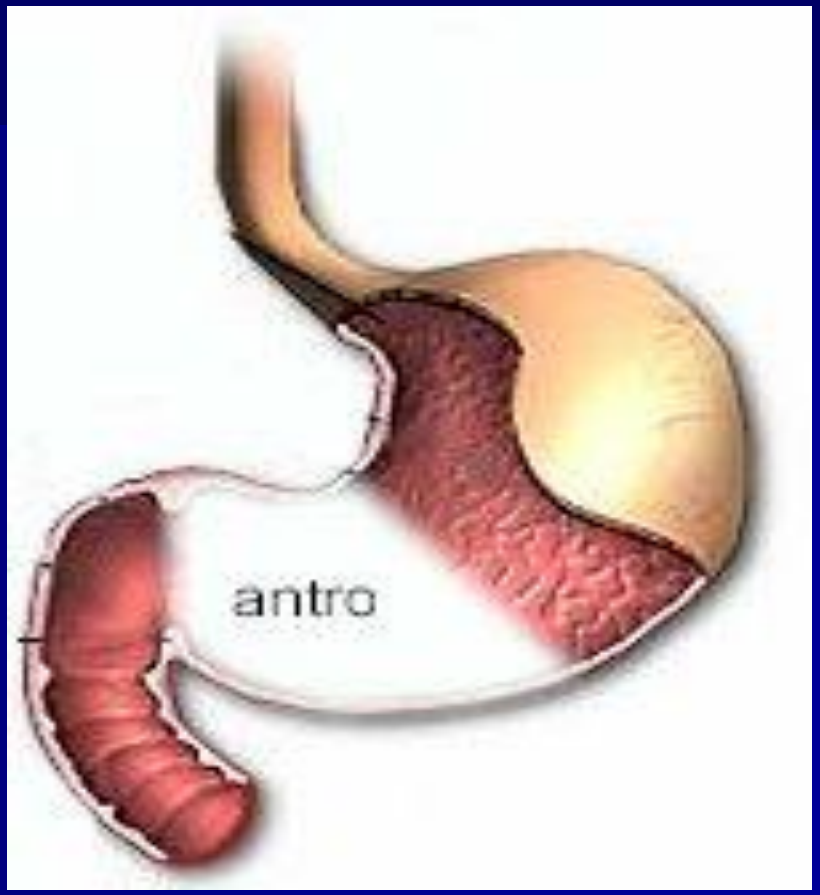
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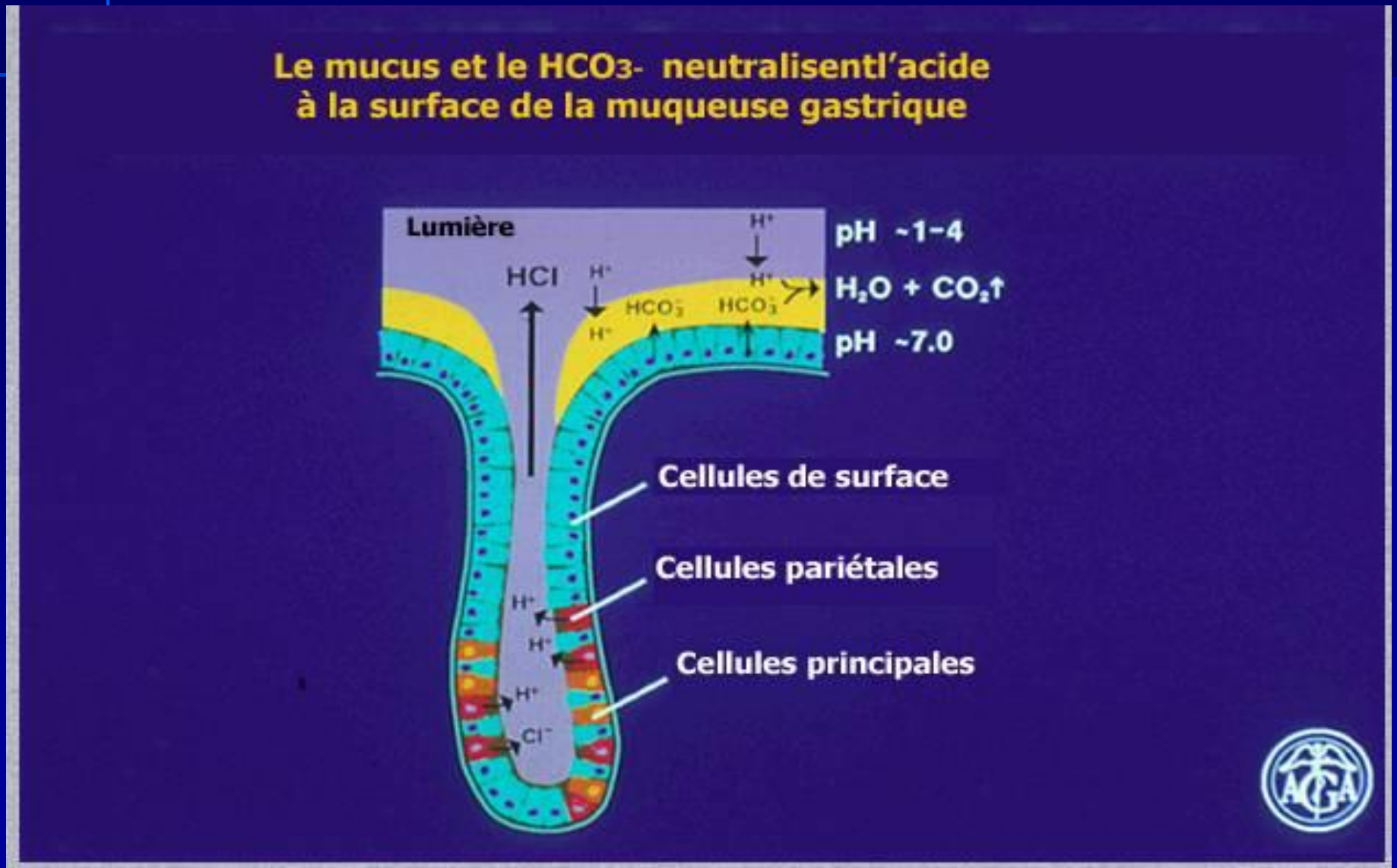
# Physiology of gastric secretion

- At the level of fundic glands there are two types of main cells:

1. parietal oxyntic cells secrete hydrochloric acid and intrinsic factor
2. peptic cells (zymogene) secrete pepsinogen.

   in acid medium pepsinogen is transformed to an active substance - pepsin.

# Main and parietal cells



# In the antrum there are endocrine cells

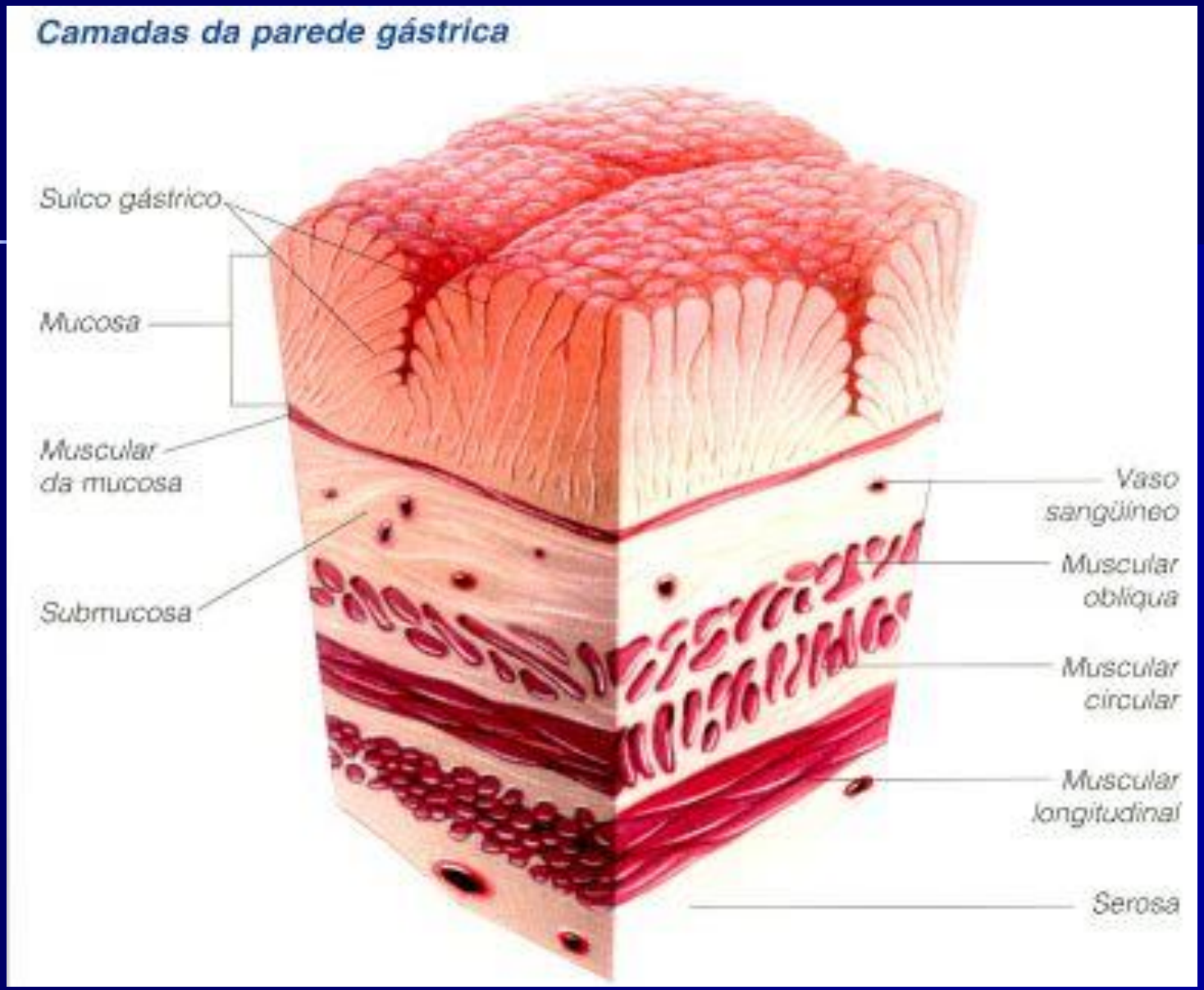
- C cell secrete and synthesize gastrine
- D cell secrete somatostatin
- EC cells secrete serotonin
- ECL cells, P,D and X cells were identified, but their functional role is not clear yet .
- Epithelial superficial cells produce mucus, bicarbonate.

# Gastritis

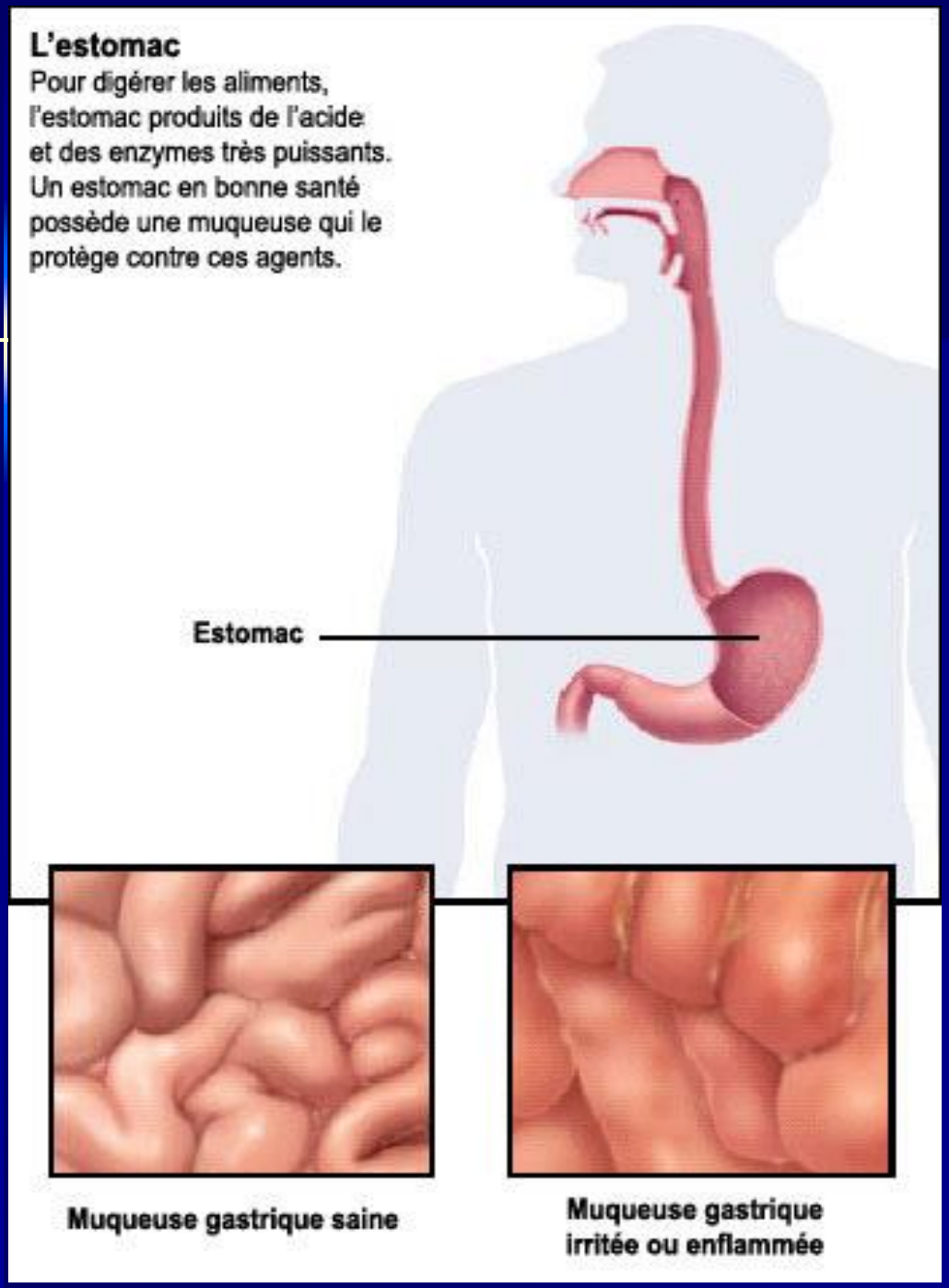
DEFINITION.

gastritis are acute or chronic inflammations, diffuse or focal, of the stomach mucosa, and sometimes of the other layers.

## Camadas da parede gástrica



# Microscopic images – normal and inflammation



# CLASSIFICATION

International Congress of Gastroenterology

(Sydney, 1990)

According to the clinical and evolutive picture:

A. **Acute**

B. **Chronic**

C. **Other types of gastritis**

# International Congress of Gastroenterology (Sydney, 1990)

According to the clinical and evolutive picture:

*A. Acute*

a) erosive – haemorrhagic

b) phlegmonous

# *B. Chronic*

A) type A - atrophic (autoimmune)

B) type B – microbial, produced by Helicobacter pylori

C) type C - chemical

- duodeno - gastric reflux
- drugs
- alcoholic

# *C. Other types of gastritis*

a) granulomatos
b) hypertrophic (Menetrier)
c) lymphocytic

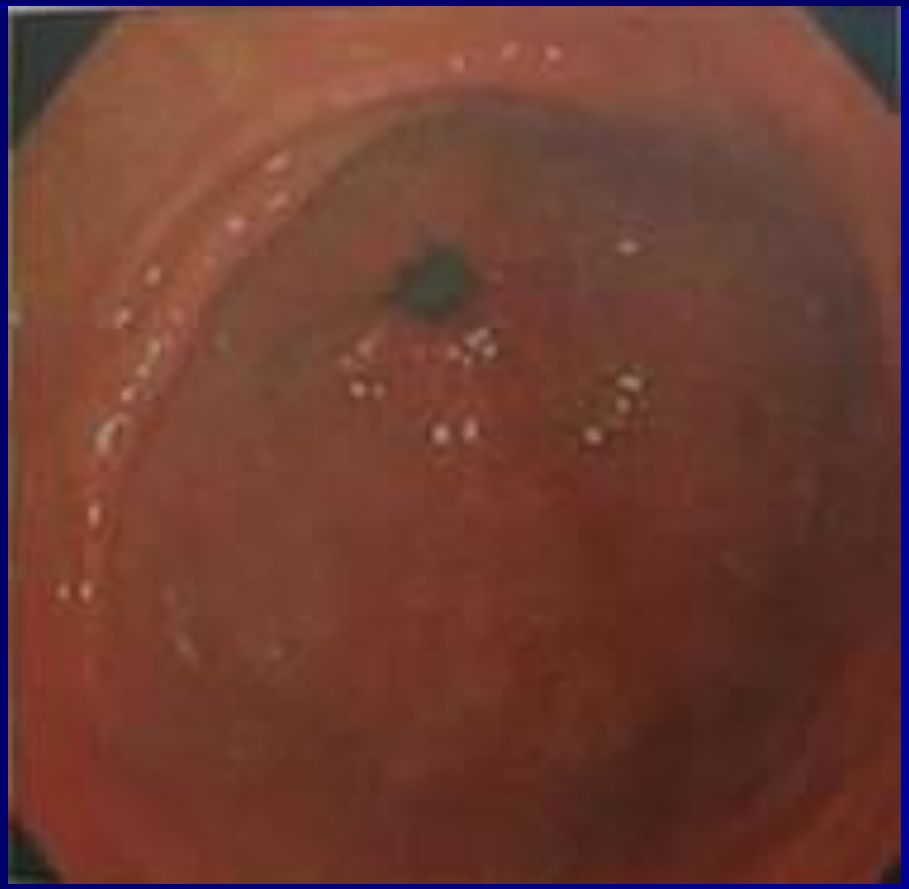
# According to the extension of endoscopic and histological lesions :

1. fundic gastritis (type A) localised in the body and fundic part of the stomach.
2. antral gastritis (type B) localised in the antrum, associated with infection with Helicobacter pylori.
3. multifocal gastritis (type AB), localised both proximal and antral.
4. Pangastritis involving the whole gastric mucosa.

# According to the endoscopic picture:

1. **erythematous – exudative gastritis** (usually acute) – characterized by erythematos zones of 2-3 mm, covered with white point shaped exudate, disseminated on mucosa.

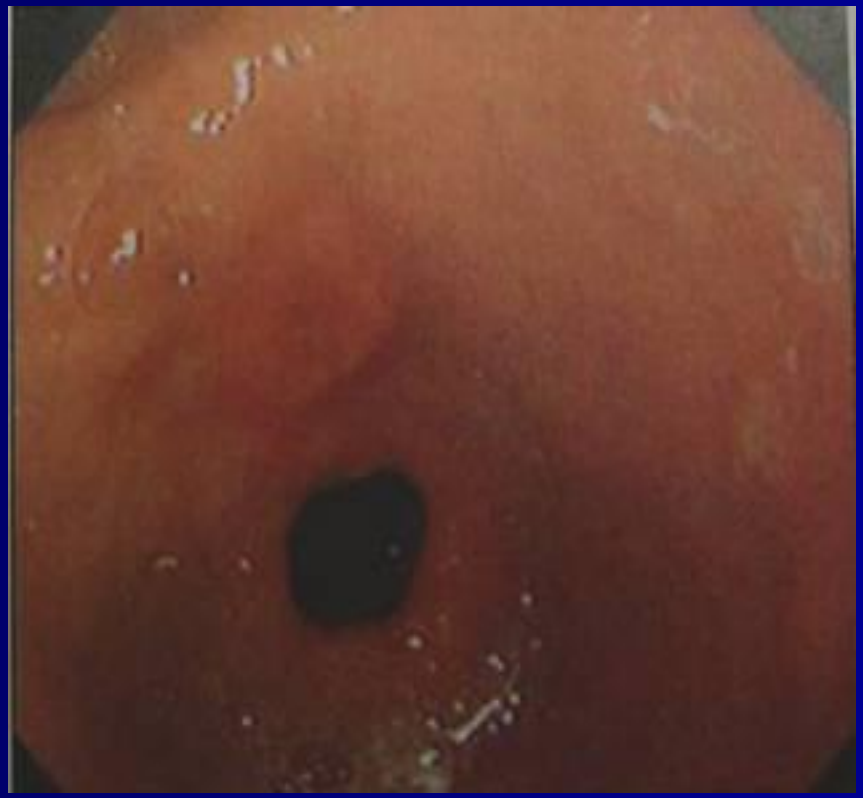
# Erythematos – exudative gastritis



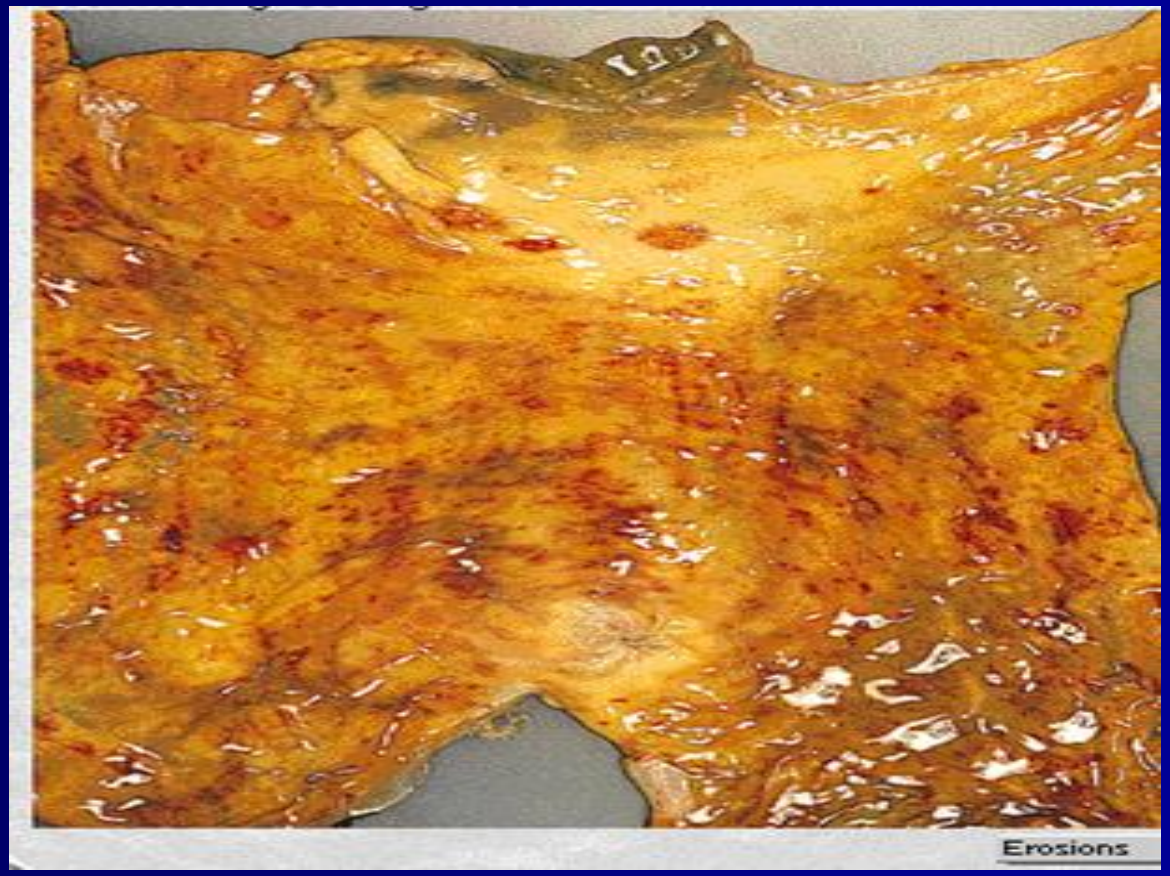
# 2. Maculo – erosive gastritis (acute lesion)

- characterized by erythematos spots 5-15 mm in diameter with superficial ulcerations covered with white to grey fibrino – leukocytar  detritus.
- The mucosa between the lesions is normal.

# Maculo – erosive gastritis



# Maculo – erosive gastritis

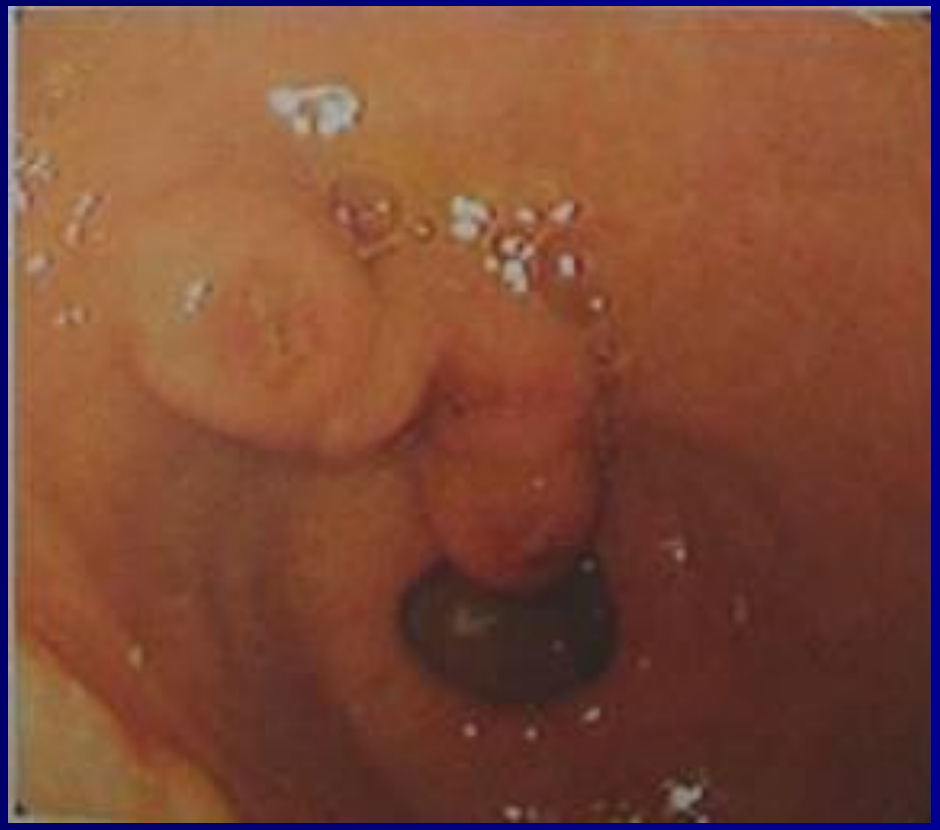


# 3. Papulo – erosive gastritis

- a chronic lesion, represented by protrusive zones 3-5 mm in diameter with a slight slope, their height is 3-4 mm over the level of mucosa.

- Sometimes these papulous lesions have an erosion in the centre, with haemorrhagic appearance .

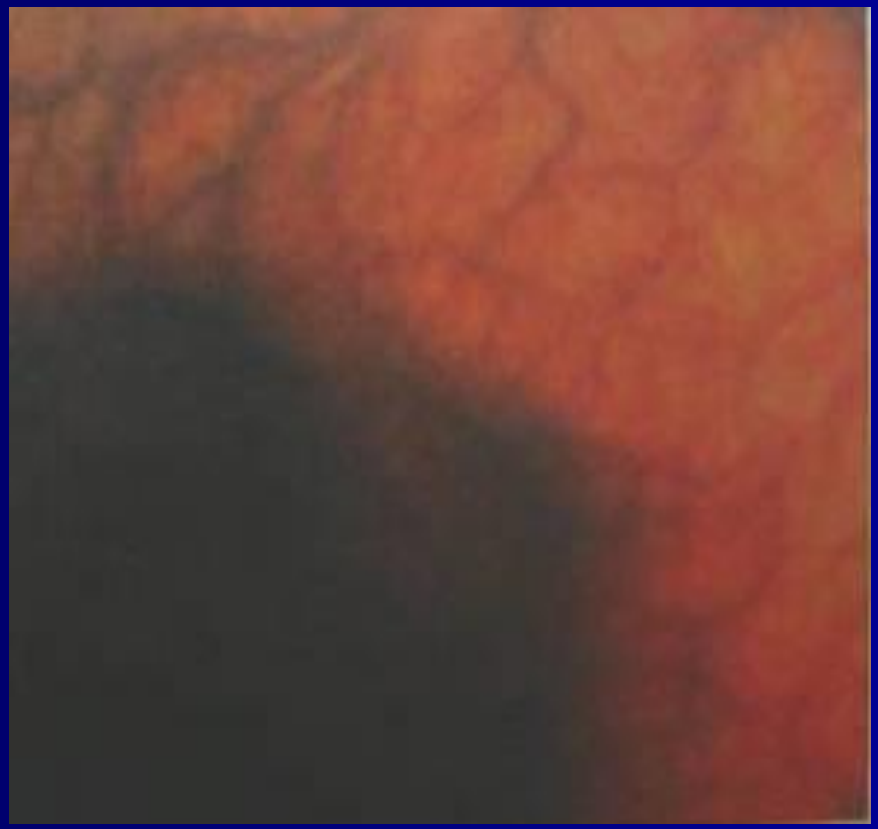
# Papulo - erosive gastritis



# 4. Atrophic gastritis

The appearance is with pale mucosa, without folders; the superficial vascularisation is very well seen.

# Atrophic gastritis



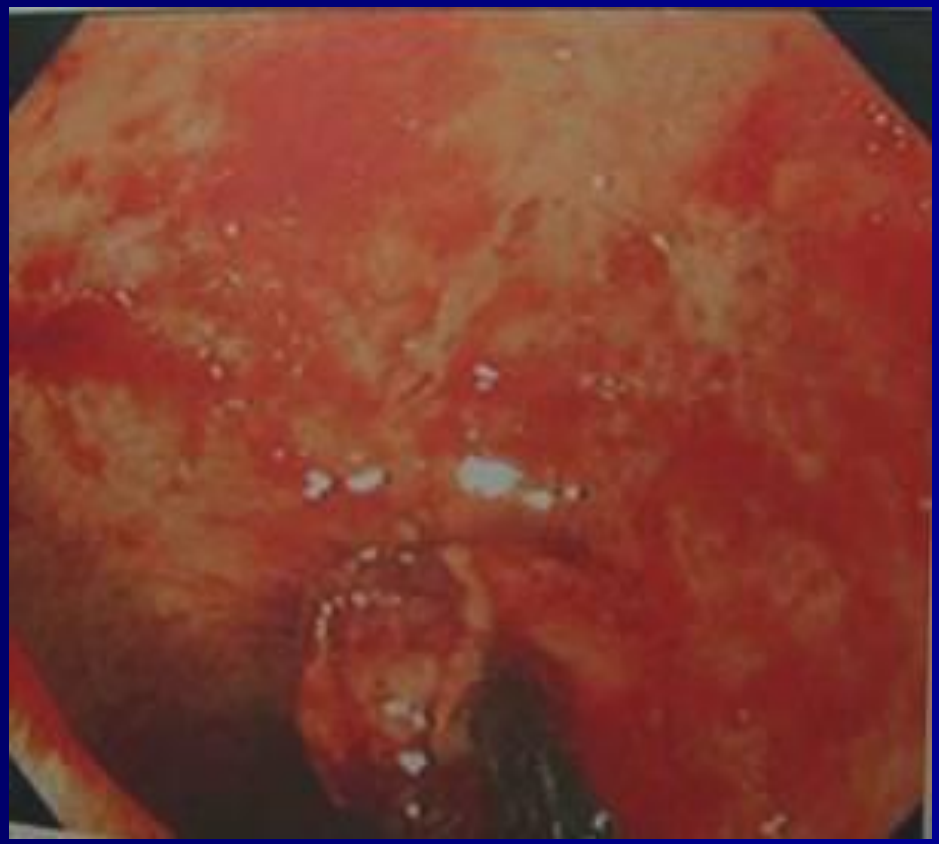
# 5. Hypertrophic gastritis

characterized by presence of high folders, more than 1 cm in height and 3-5 mm in width.

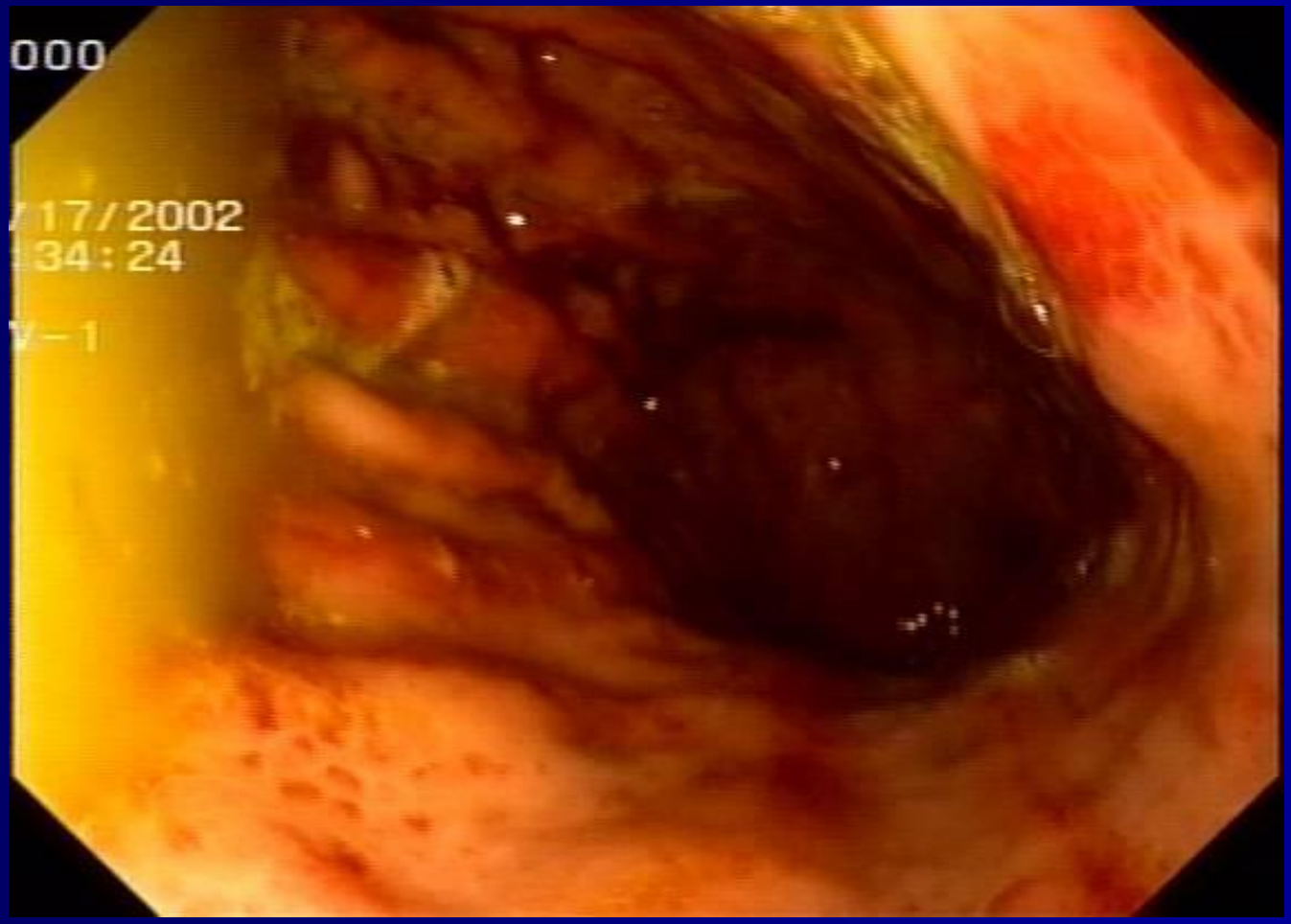
# 6. Haemorrhagic gastritis

in the faze of active bleeding, looks like circumscribed points or spots more or less extended, with haemorrhage.

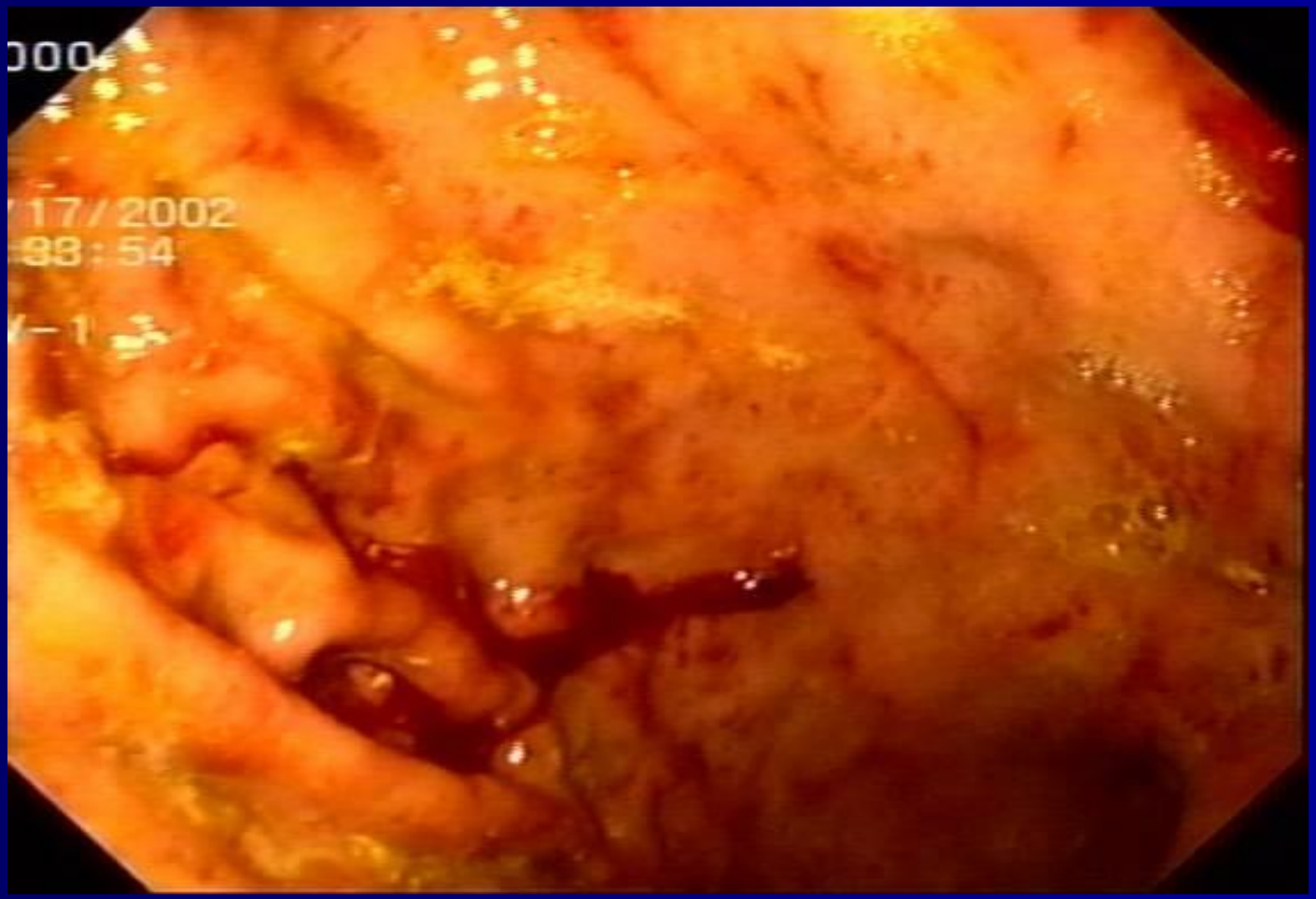
# Haemorrhagic gastritis



# Haemorrhagic gastritis



# Haemorrhagic gastritis



# Clinical and paraclinical syndromes in gastro-intestinal tract diseases

# Acute erosive – haemorrhagic gastritis

acute erosive gastritis is characterized by superficial ulcerations involving the layers up to muscular layer of mucosa.

# *Aetiology*

acute erosions can appear in a variety of etiological conditions:

- drug action (anti-inflammatory, prednisone, iron, cytostatics)
- Helicobacter pylori
- Alcohol
- local traumatism
- caustic substances
- duodeno – gastric reflux

8. Uraemia
9. Ischemia of mucosa
10. Polytraumatism
11. Extended surgery
12. Lesions of central nervous system
13. Extended combustions
14. Septicaemia
15. Shock (septic, hypovolemic, anaphylactic)
16. Acute respiratory failure
17. Acute renal failure
18. Hepatic failure.

# *Pathogenesis*

- The above etiological factors interact with diverse structures of the gastric mucosa.
- The etiological factors inhibit secretion by mucosa of prostaglandins, thus inhibiting secretion of bicarbonate and mucus.

- normally bicarbonate and mucus form the so-called „mucus – bicarbonate barrier”.
- in shock, gastric erosions appear mostly due to ischemia of mucosa.
- free radicals of oxygen, formed in conditions of ischemia under the action of anti-inflammatory drugs and alcohol induce lesions of gastric mucosa .

the etiological factors, together with HCl and pepsin, act on a mucosa having no means of defence, leading to development of gastric erosions.

# *Morphology of acute gastritis*

- acute medicamentos gastritis is manifested by multiple erosions, localized mainly in antral zone.

- alcoholic gastritis more frequently affects the antrum. Mucosa is hyperaemiated, fragile, with haemorrhages and oedema.

- acute gastritis with Helicobacter pylori is manifested endoscopically by congestion, sometimes acute localized erosions, more frequent in the antrum.

# *Clinical picture*
# the symptoms of acute erosive gastritis:

- Epigastric pain,
- Nausea
- Vomiting.

In the majority of patients the haemorrhage is occult, seen only by an examination of faeces for occult blood.

# *Diagnosis*

- Endoscopic examination: one can appreciate number of erosions and their extension (stomach, duodenum) and „signs” of haemorrhage.
- gastric erosions are covered with haematic crusts or fibrin. Mucosa is congested, oedemated and fragile when touched with gastroscope.

- Complications - haemorrhage

# Acute phlegmonos gastritis

- Found rarely, most frequent inpatients with immunodeficiency (AIDS, under cytostatic treatment, with organ transplant).

*Etiology*

- The bacteria associated with phlegmonos gastritis are: streptococci, *E.Coli, Proteus vulgaris, Clostridium perfringens*.

# *Pathogenesis*

- Purulent inflammation is more frequent found in submucosa.

*Morphopathology*

**the s**tomach is dilated, the gastric wall is thickened, infiltrated with pus, and gastric vessels are thrombosed.

# *Clinical picture*

- patients with phlegmonous gastritis have clinical manifestations of acute abdomen.
- pain initially is localized in epigastria, afterwards becomes diffuse.
- Also there could be nausea, vomiting, fever.

# *Diagnosis*

1. Radiological examination:

   Reveals presence of air inside the gastric wall (when the etiological agent is *Clostridium perfringens* , which forms gas). In case of perforation there are signs of pneumoperitoneum – air in abdominal cavity.

2. Bacteriological examination

   Reveals etiological agent.

# *Evolution. Complications*

- Evolution is severe.
- The most frequent complications are :

  peritonitis,

  septicaemia,

  superior digestive haemorrhage.

# Chronic gastritis

## Chronic bacterial gastritis (type B)

- Definition: inflammation of gastric mucosa (predominant in antral area) induced by Helicobacter Pylori.

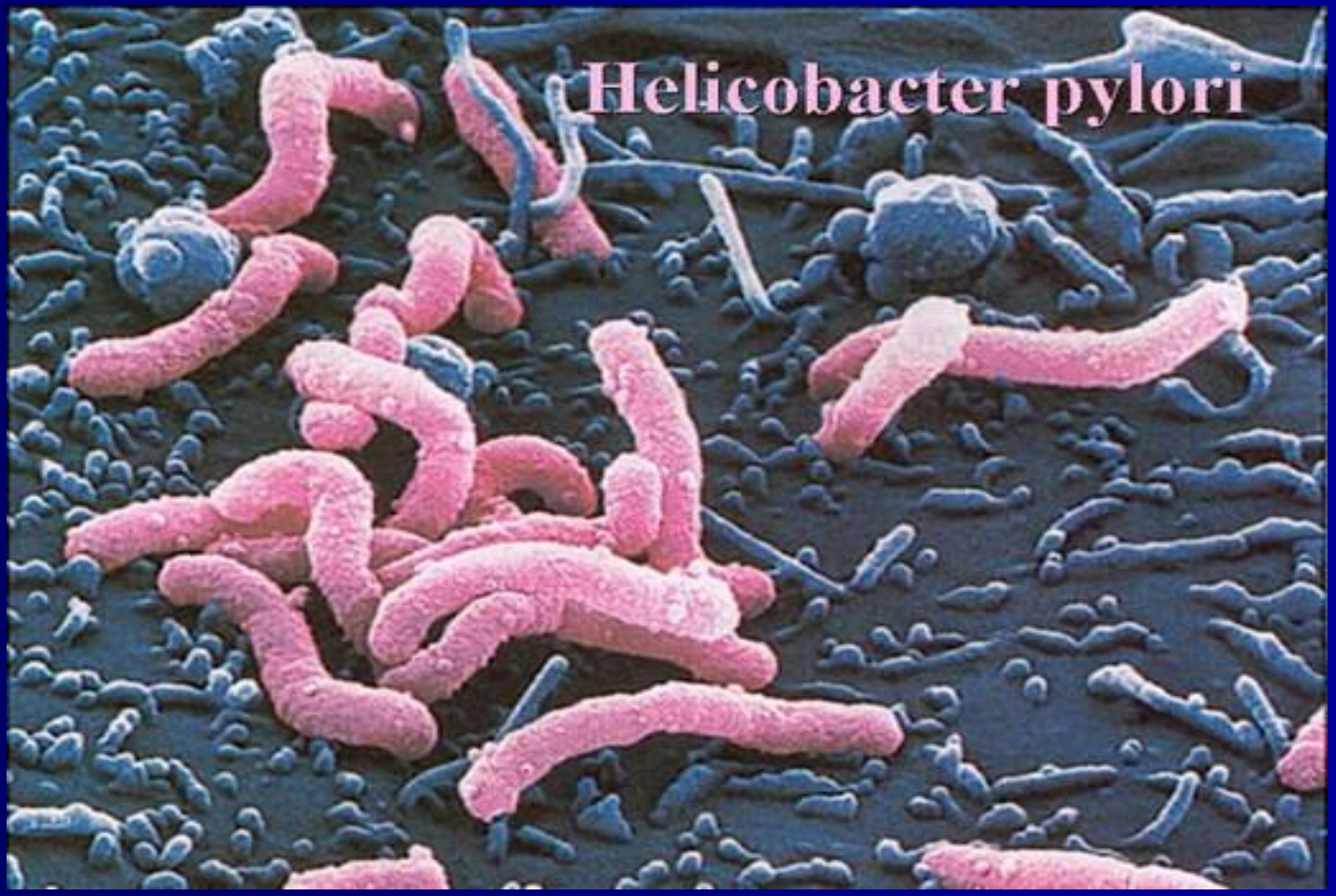
# *Aetiology*

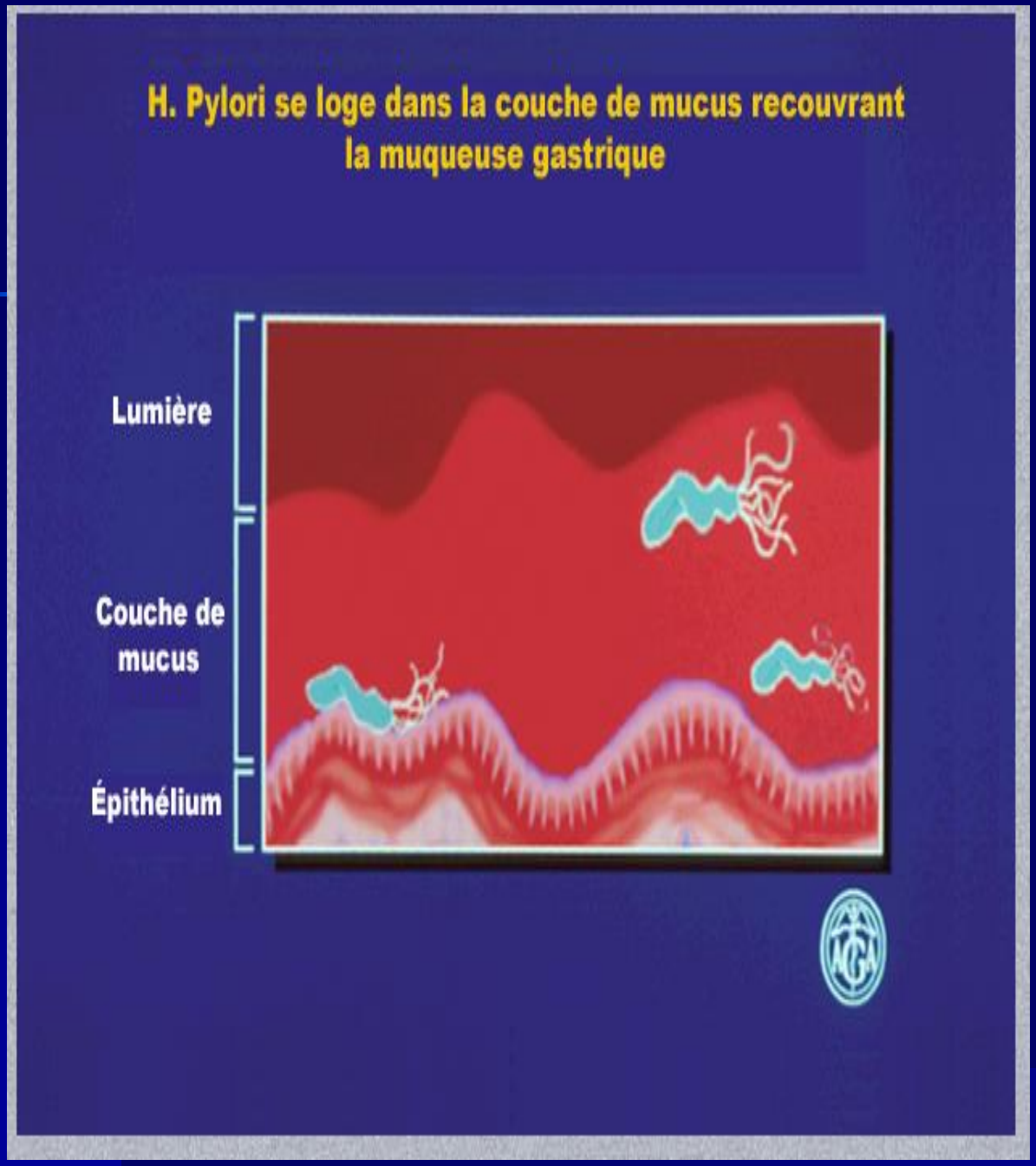
- The cause of the disease is HP.
- Bacteria is gram negative, spiral, localized in the stomach under the layer of mucus around the gastric crypts and between epithelial cells.
- The length of bacteria is 2-3 micron, and the diameter is 0,5 micron.
- Has a large enzymatic package: urease, catalase, protease, mucinase.

## H.Pylori est une bactérie flagellée et de forme spirale qui produit une uréase

- Bactérie Gram -, d'une grosseur de 0.5 - 1.0 microm x 2.5 - 4.0 microm
- L'uréase transforme l'urée en ammoniac et en diosyde de carbone
- Elle croît en 2 à 5 jrs à une température entre 35 - 37 Celsius dans un environnement microaérophile
- Sa culture en laboratoire requière un milieu spécial

# Helicobacter pylori






The enzymes are: urease, catalase, protease, mucinase.

# *Pathogenesis*

- The presence of bacteria in gastric mucosa implies participation of inflammatory cells (polymorphonuclears and mononuclears).
- Polymorphonuclears liberate free radicals of oxygen, which react with different structures of the cells.

- Hypo - or achlorhydria is transitory in HP infection.
- Bacteria generates ammonia at the surface of gastric epithelium.

- Seric gastrine is increased due to alkaline pH
- Pepsinogen I is markly increased in patients infected with HP
- Glycoproteins of gastric mucus are modified by bacterial proteases, thus, the protective function of gastric mucus is diminished.
- The bacteria also determines ischemia of gastric mucosa.

# Morphopathology

- There is congestion at the level of gastric mucosa, predominant in antrum, also acute or chronic erosions .
- after discovering the bacteria two histological terms appeared:

A. Active chronic gastritis

B. Inactive chronic gastritis

# A. **Active chronic gastritis is defined by:**

Presence in gastric mucosa of a reach infiltrate with polymorphonuclears.

# B. Inactive chronic gastritis is characterized by:

Presence in gastric mucosa of mononuclear cells, which dominate the inflammatory infiltrate

# *Clinical picture*

- epigastic pain – appears in 88% of patients.
- Nausea and vomiting are present in 60% of cases.
- The above symptoms persist months or even years, disappear only after treatment with antibiotics.

# *Diagnosis*

- Detection of circulating antibodies anti-IgG.
- Respiratory tests detect marked radioactive CO2, formed at decomposition of marked urea, administrated to the patient (by urease, produced by H. Pylori).
- Molecular method – appreciation of HP DNA in saliva, gastric juice, faeces.

- Ureasic test – due to the action of urease urea is decomposed into water, CO2 and ammonia, the latest could be detected with special devices during expiration of the patient.

- PH of gastric juice (FGDS) becomes alkaline due to accumulation of ammonia

- Endoscopic biopsy confirms infection in 90 % of cases.

# *Evolution*

- The inflammatory process is not limited to antrum, in time it is spread to the body of the stomach and sometimes to duodenum.

- Type B gastritis can evolve to gastric cancer .

# *Prophylaxis*

- Prophylaxis of HP infection presumes compliance to elementary hygienic rules, as the route of transmitting is oral one.
- In the hospital prevention of transmitting is done by adequate sterilisation of exploration instruments.

# Chemical gastritis

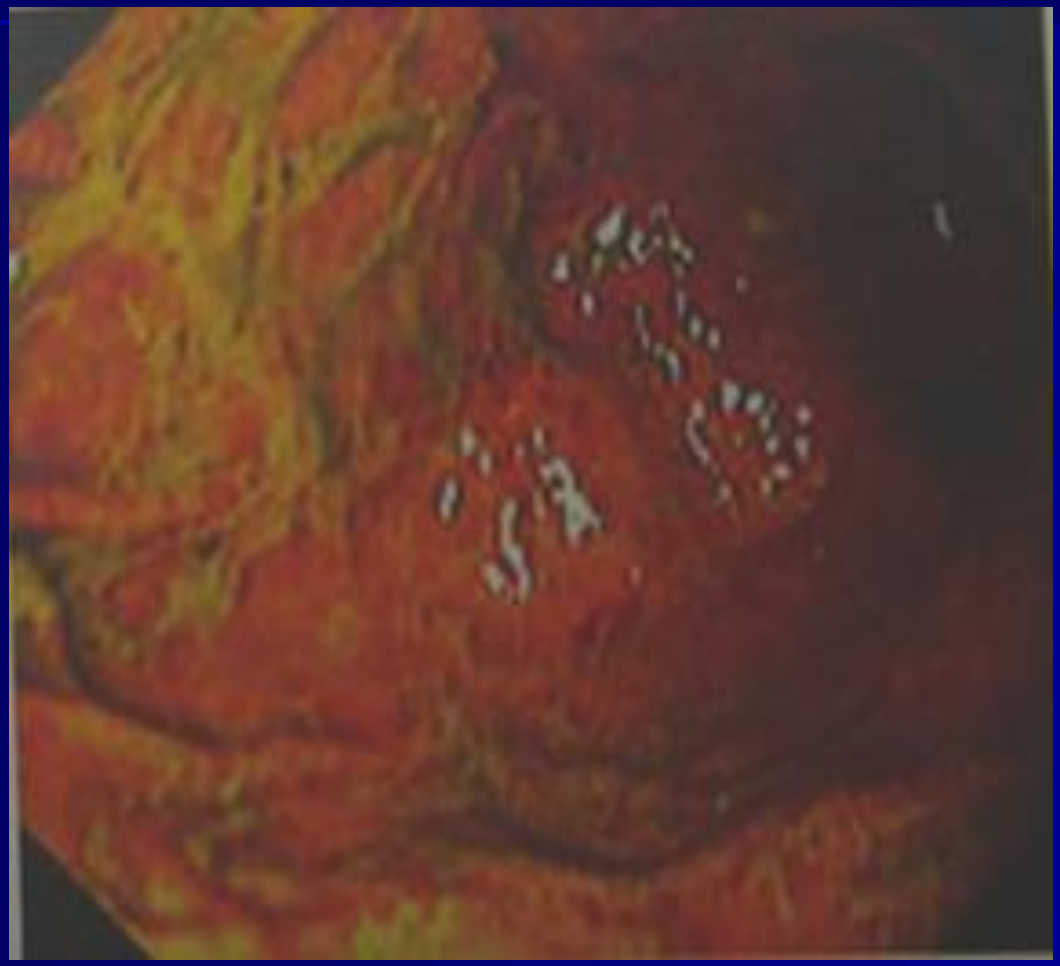
## *1. reflux - gastritis (type C)*

- Definition: is characterized by inflammation of gastric mucosa as a result of regurgitation of duodenal juice to the stomach.

*Aetiology*

- Duodenal reflux is the principal cause of gastritis and has a triple origin: biliary, pancreatic and intestinal with aggressive action on different structures of mucosa.

# Gastritis due to duodeno - gastric reflux



# *Pathogenesis*

Duodenal reflux appears in 2 conditions:

- Absence of pyloric barrier (surgical)
- Incompetence of pyloric sphincter

biliary acids act as detergents, washing out the layer of mucus from the surface of stomach epithelium

- Tripsin realizes proteolysis of glycoproteins from gastric mucus

- The modified stomach epithelium allows retrodiffusion of H+ to interstitium and leads to tissue acidosis.

  in acid medium pepsinogen is transformed to pepsin, thus injuring the mucosa.

# *Clinical picture*

- Post- food ingestion pain in epigastria, refractary to antiulcer drugs.
- biliary nausea and vomiting are frequently met in reflux gastritis

# *Morphology*

- Histologically there is vasodilation and congestion; interstitial oedema; reduced number of inflammatory cells.

# *Diagnosis*

- endoscopic and histological examination are mandatory diagnostic procedures for reflux-gastritis.

  The reflux can be determined by:
- biochemical analysis of gastric juice (biliary acids, bilirubin ... Etc.)
- PH of gastric juice increases.

# *Evolution. Complications*

- chronic superficial postgastrectomy gastritis potentially evolves to chronic atrophic gastritis
- The most severe complication is gastric cancer.

# Alcoholic gastritis

- Ethanol represents an aggressive factor for gastric mucosa.

***Pathogenesis***

- 70% Alcohol produces in 30 – 45% of cases necrosis of epithelial cells.
- Capillaries are thrombosed with erythrocyte and platelet thrombi.

# *Morphology*

- mucosa is congested, friable with chronic erosions.
- Histological – subepithelial haemorrhage.

# *Clinical picture*

- Epigastric pain, nausea, vomiting after ingestion of alcohol .
- superior digestive haemorrhage sometimes could be a manifestation of alcoholic erosive gastritis.

# *Diagnosis*

- Is based on the above clinical picture and anamnestic data.
- The major complication is superior digestive haemorrhage.

# Medicamentos gastritis

- medicamentos gastritis is a cause of use of nonsteroid and steroid anti-inflammatory drugs.

- Could be a result of other drugs: tetracycline, potassium salts, cytostatic drugs.

# *Pathogenesis*

- The severity of lesions is correlated to the dosage used.
- The lesions appear mostly in the stomach (93%), and more rare in duodenum ≈ 45%.

# *Morphology*

- endoscopic and histological lesions are similar to those in reflux - gastritis

*Clinical picture*

- epigastric pain,
- nausea,
- vomiting.

# *Diagnosis*

- Anamnestic data.
- The most frequent complication is superior digestive haemorrhage.

# Chronic atrophic autoimmune gastritis (type A)

- Definition: atrophy of mucosa, located in the body and fundic part of the stomach, associated with pernicious anaemia and other autoimmune diseases.

# *Aetiology*

1. Etiological factors

**Exogenous:**

- Aggressive food: spicy, irritant
- Drugs
- Physical factors: cold or hot food
- Bacterial factors: HP, viruses, parasites.
- Alcohol
- Smoking

# Endogenous:

- Age over 50 years
- Pathological conditions: general – diabetes mellitus, hypo- or hyperthyroidism, chronic heart failure, liver cirrhosis, chronic renal failure, chronic pulmonary diseases.

# *Pathogenesis*

- The disease is autoimmune.
- Presence of anti parietal cell antibodies (parietal cells secrete a protein, which becomes an antigen), of anti-intrinsic factor antibodies: association of other autoimmune diseases (Hashimoto thyroiditis, Adison disease).

- The etiological factor triggers an inflammatory process like in chronic superficial gastritis at the level of gastric body, which afterwards evolves in the majority of cases to atrophy.

- Disappearance of parietal cells leads to hypo- or achlorhydria

- chronic gastritis type A can finally in some patients be associated with Biermer anaemia (decreased level of seric Vit B12) .

# *Morphology*

- gastritis is localized at the level of gastric body and gastric fundus
- Endoscopically mucosa has signs of atrophy: low folds, pale mucosa

# *Clinical picture*

Complaints:

- loss of appetite,
- nausea,
- balonation after food ingestion.

Could be associated with pernicious anaemia

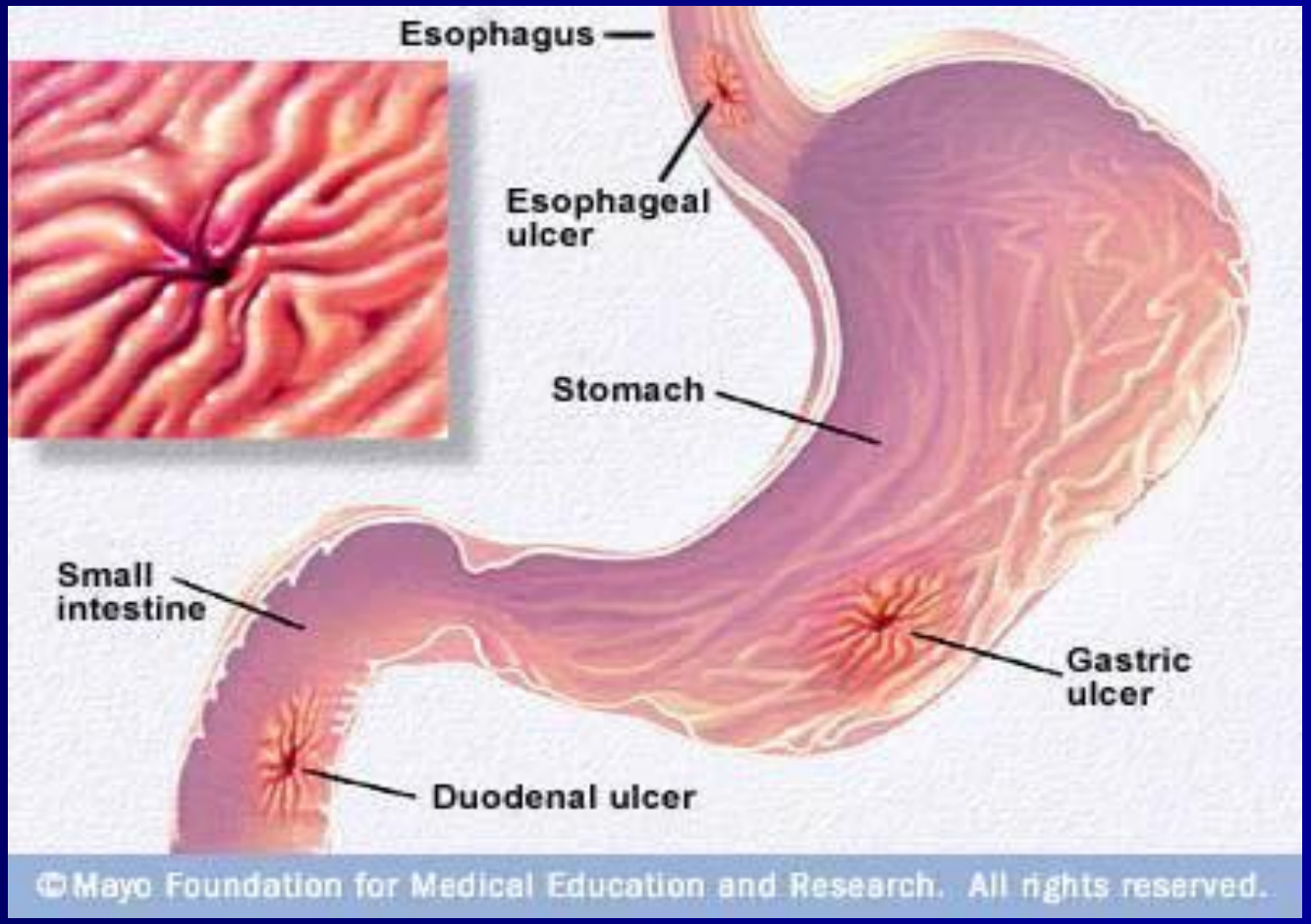
# *Diagnosis*

- endoscopic and histological examination  are essential for diagnosis. PH decreases
- B12 deficient anaemia (macrocytes), Joli corpuscles and Kebot rings.
- The most frequent complication is gastric adenocarcinoma.

# Gastric and duodenal ulcer

Ulcer is a defect of gastric or duodenal mucosa, passing in depth the muscular layer and surrounded by acute or chronic type inflammatory infiltrate.

# Gastric and duodenal ulcer



# *Ulcerogene factors*

1. Hydrochloric acid is increased mostly in duodenal ulcer. There is an increase of number of parietal cells and of vagal tonus, hypersecretion of histamine and gastrine, and also pepsinogen I.
2. Duodeno – gastric reflux:

- biliary salts
- pancreatic secretion
- intestinal secretion

# *Protective factors*

1. Mucus, which contains glycoproteins
2. Bicarbonate
3. Prostaglandins E2, F2ą
4. cells of epithelium gastric

5. The epidermal growth factor is a hormone which has an antiulcerative effect, by acceleration of cell maturation and stimulation of cell proliferation. This factor acts on parietal cells by inhibition of HCl secretion.

6. Blood microcirculation of gastric and duodenal mucosa, which represents nutrition income and the degree of oxygenation.

- In patients with gastric and duodenal ulcer all these factors of protection are decreased.

# *Pathogenesis*

1. Genetic factor: male more frequent. Blood group O increases the risk for ulcer.
2. Infection with Helicobater pylori:

- Urease – hydrolyses urea into ammonia, protects bacteria from acid medium and also has a cell toxicity, modifying the physicochemical properties of gastric mucus.

- Mucinases and peptidases interact with glycoproteins from gastric mucus and change them.
- Phospholipase – acts on phospholipids of cell membrane of gastric epithelium

Thus, there is an alteration of gastric mucosa barrier.

3. Smoking: increases secretion of HCl, increases synthesis of pepsinogene, decreases secretion of gastric mucus, decreases synthesis of stomach and duodenum prostaglandins.

4. Alimentation: there are a lot of theories and diet schemes for the treatment of ulcer, but no one had proved its consistence.

5. Stress
6. Anti-inflammatory drugs: act on different structures of gastric mucosa, inhibiting synthesis of endogenous prostaglandins and increasing secretion of HCl.

# *Morphology*

- Gastric ulcer is most frequently localized on small curvature, but it could be localized anywhere.
- The dimensions a variable.
- Duodenal ulcers are more frequent
- Microscopically there is an periulcerous  inflammatory infiltrate

# Chronic peptic ulcer

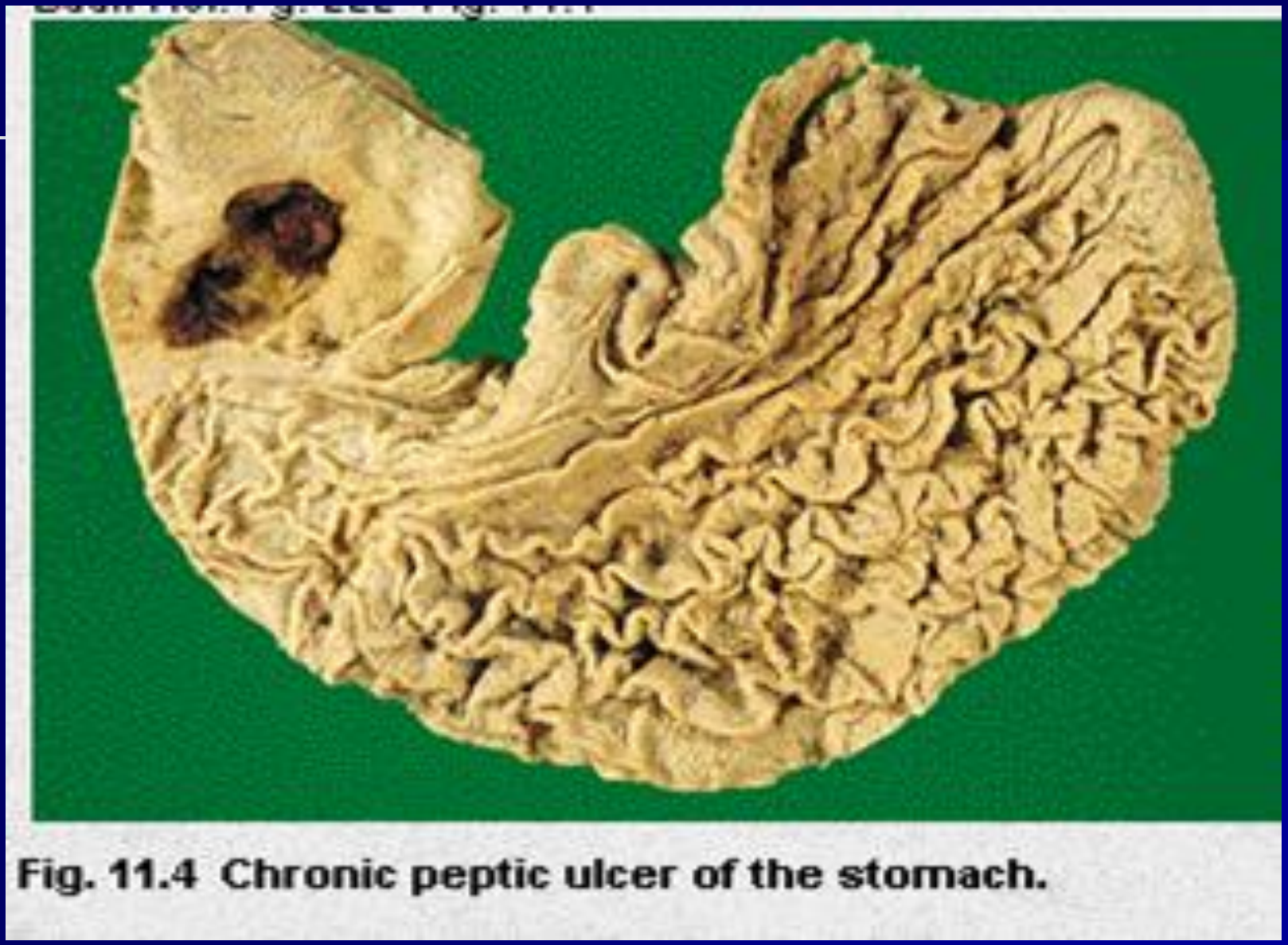


Fig. 11.4 Chronic peptic ulcer of the stomach.

# *Clinical picture*

- pain in epigastric region
- in duodenal ulcers pain appears late after food ingestion (1,5 -2 hours, sometimes 4-5 hours), sometimes there could be hungry or nocturnal pain.

# in gastric ulcer

- pain appears in 30min – 1 hour after food ingestion. The character of the pain is variable: burning, feeling of compression or nibble, feeling of emptiness.

Localization:

- epigastria
- Posterior $T_{11}$-$T_{12}$, $L_1$, $L_2$ paravertebral (Boas points)
- in supraumbilical region

# Characteristics of pain

Irradiation of pain - posterior (more frequent in duodenal ulcer),

Duration is de 30min – 1 hour,

Relief - after ingestion of alkaline food.

Periodicity – in patients with duodenal ulcer: spring and autumn (conditioned by psychical and endocrine factors; alimentary factors).

# Other complaints:

- Nausea and acid vomiting, after which pain is relieved.

- Weight loss is present especially in patients with gastric ulcer (≈2-3 kg) and could be explained by deficient alimentation due to fear of pain

- Heartburn is frequent – 60-80% of cases. The cause is reflux of acid content to the inferior third of oesophagus.

- Eructation– with air;
- Hypersalivation
- Nausea – more frequent in gastric ulcer.
- Vomiting – the cause is pylorosmasm. pain and nausea precede vomiting and sometimes disappear after vomiting. The emetic masses contain food, have an acid smell at the beginning, which then change to bile character.
- The appetite is preserved in duodenal ulcer, rarely decreased.
- Constipation – the cause is hypervagotonia.

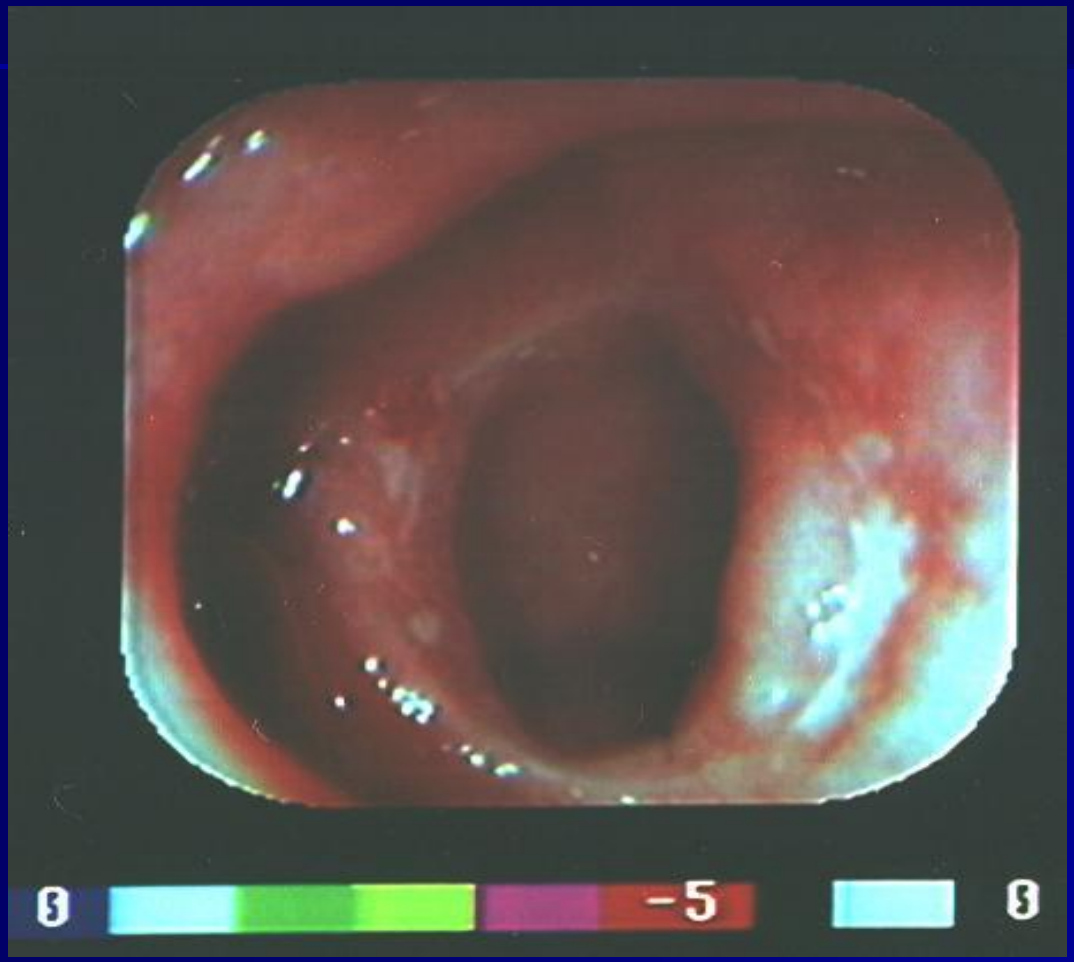
# *Physical examination*

- The patients could be of asthenic constitutional type with prominent cheeks, wet and cold extremities.
- examination of abdomen – there could be a tenderness by palpation: in gastric ulcer 2-3 cm above umbilicus and in duodenal ulcer - 1 cm above umbilicus and to the right.
- Also there could be a muscular defence in these regions.

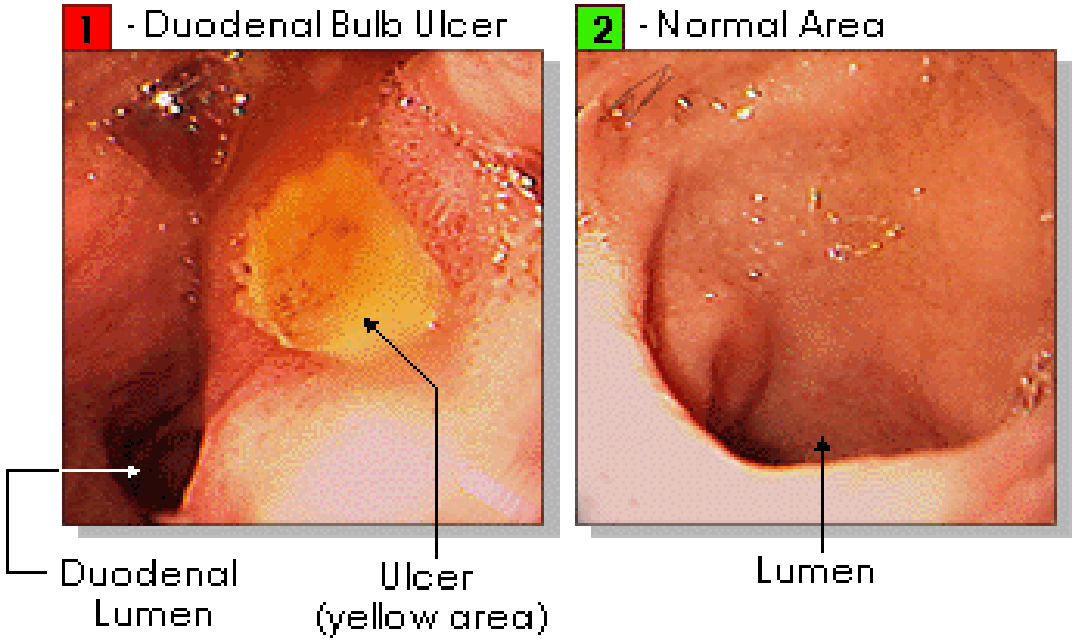
# *Diagnosis*

- Endoscopic – se evidențiază craterul ulceros de diverse forme și dimensiuni, covered with o membrană alb-surie de fibrină. Se studiază  profunzimea ulcerului, mucosa din jurul ulcerului.
- Aceste caracteristici pot fi și in cancer gastric, deaceea e necesar de efectuat și biopsia din marginea ulcerului for diagnosis diferențial.

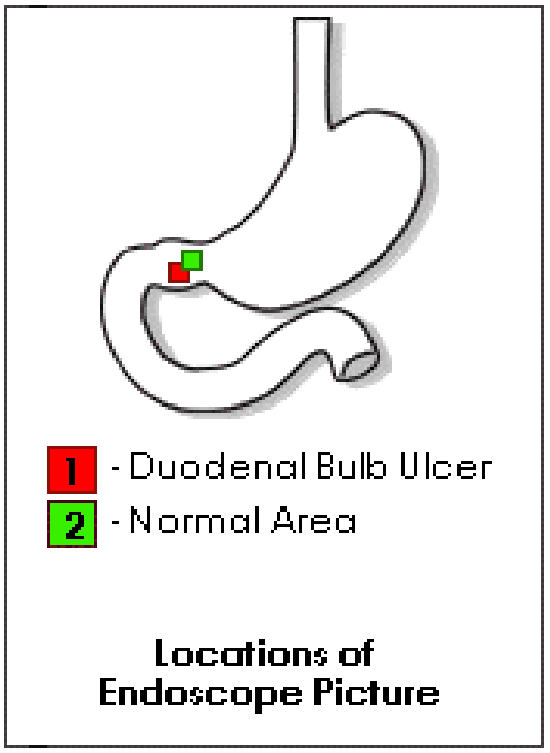
# Ulcer of circumferential shape



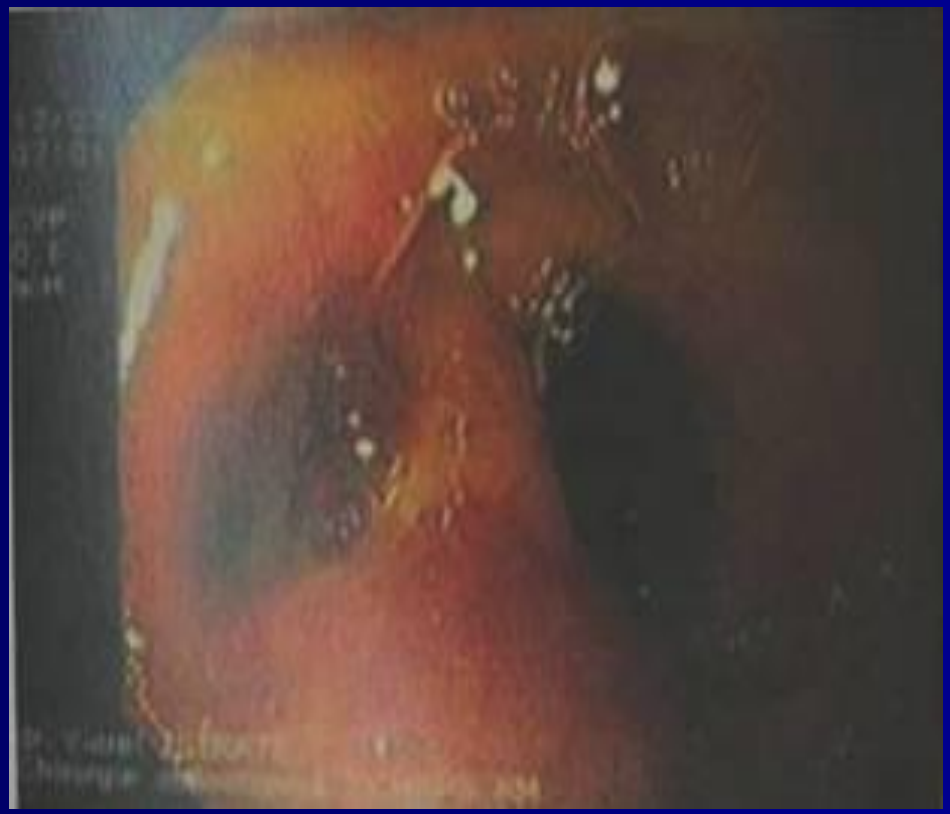
# Endoscopy Findings



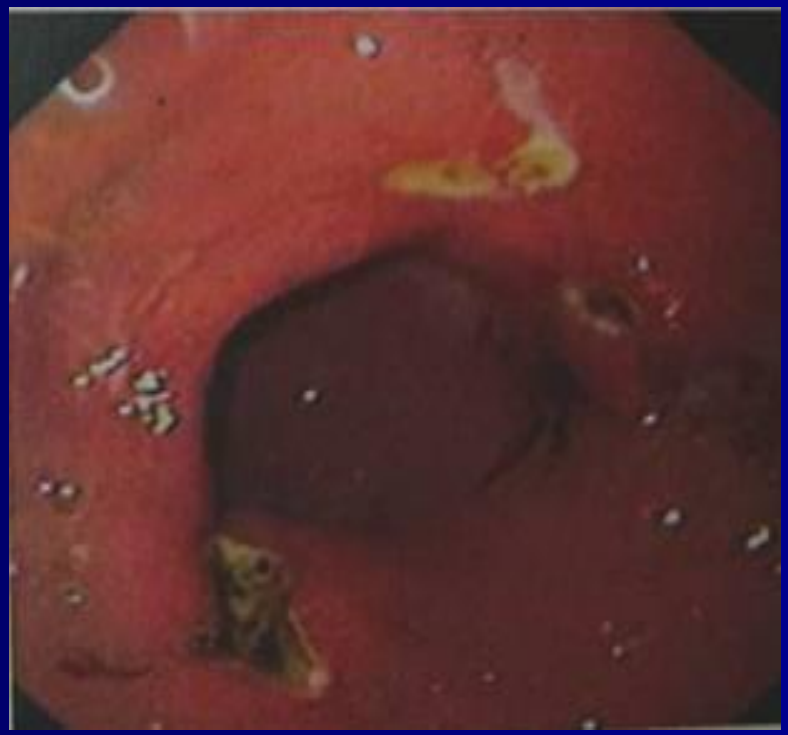

**Endoscopic view of duodenal bulb ulcer. View is from the pylorus, looking aborally into the bulb. The yellow area represents FIBRIN.**



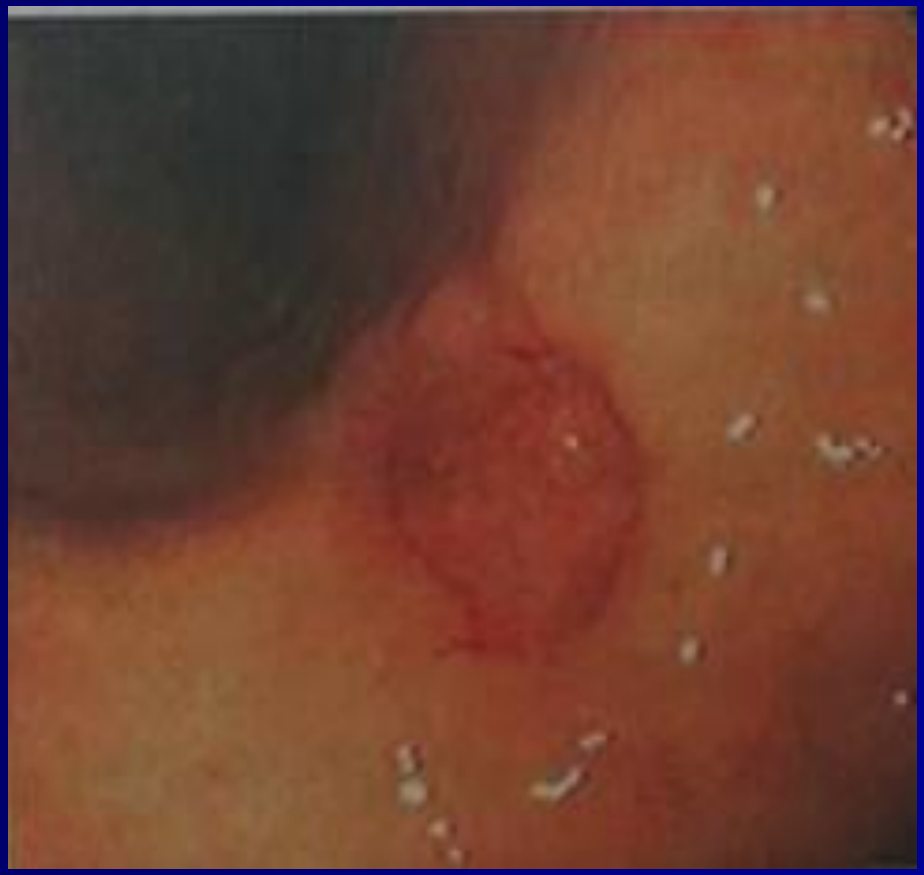
# Chronic bulbar ulcer



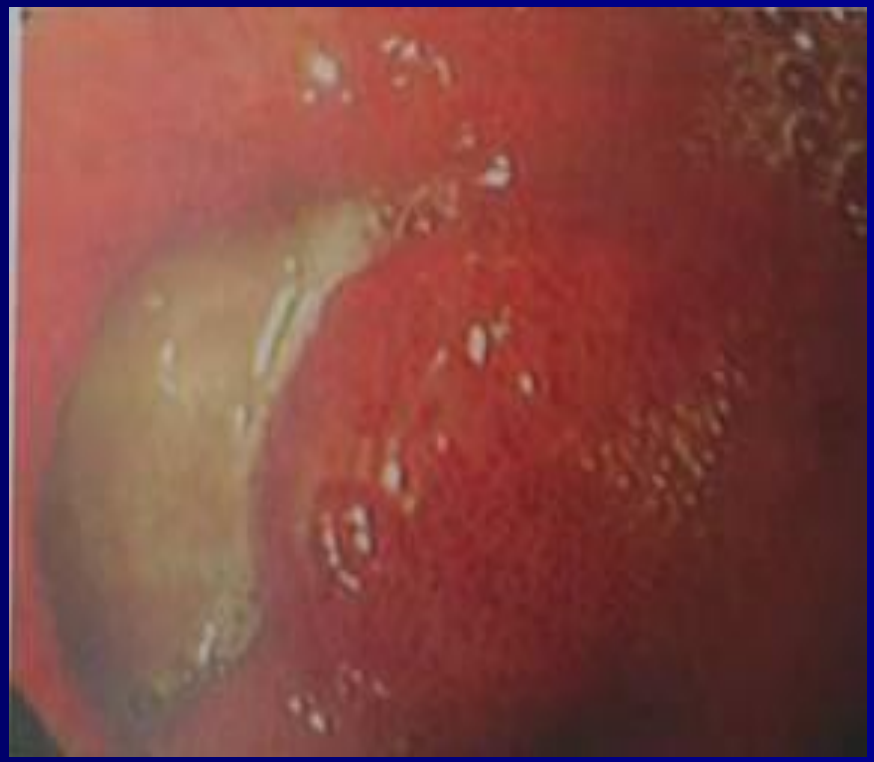
# Irregular gastric niche, acute ulcerations



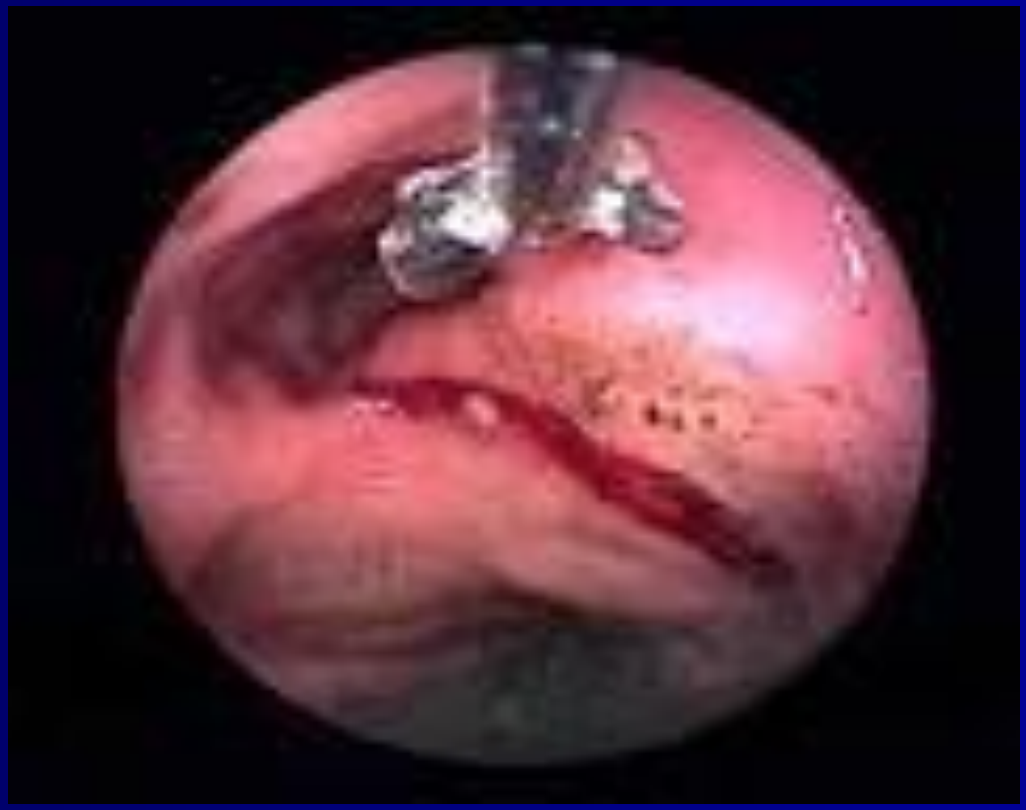
# Rounded gastric niche



# Oval duodenal niche



# Gastric biopsy



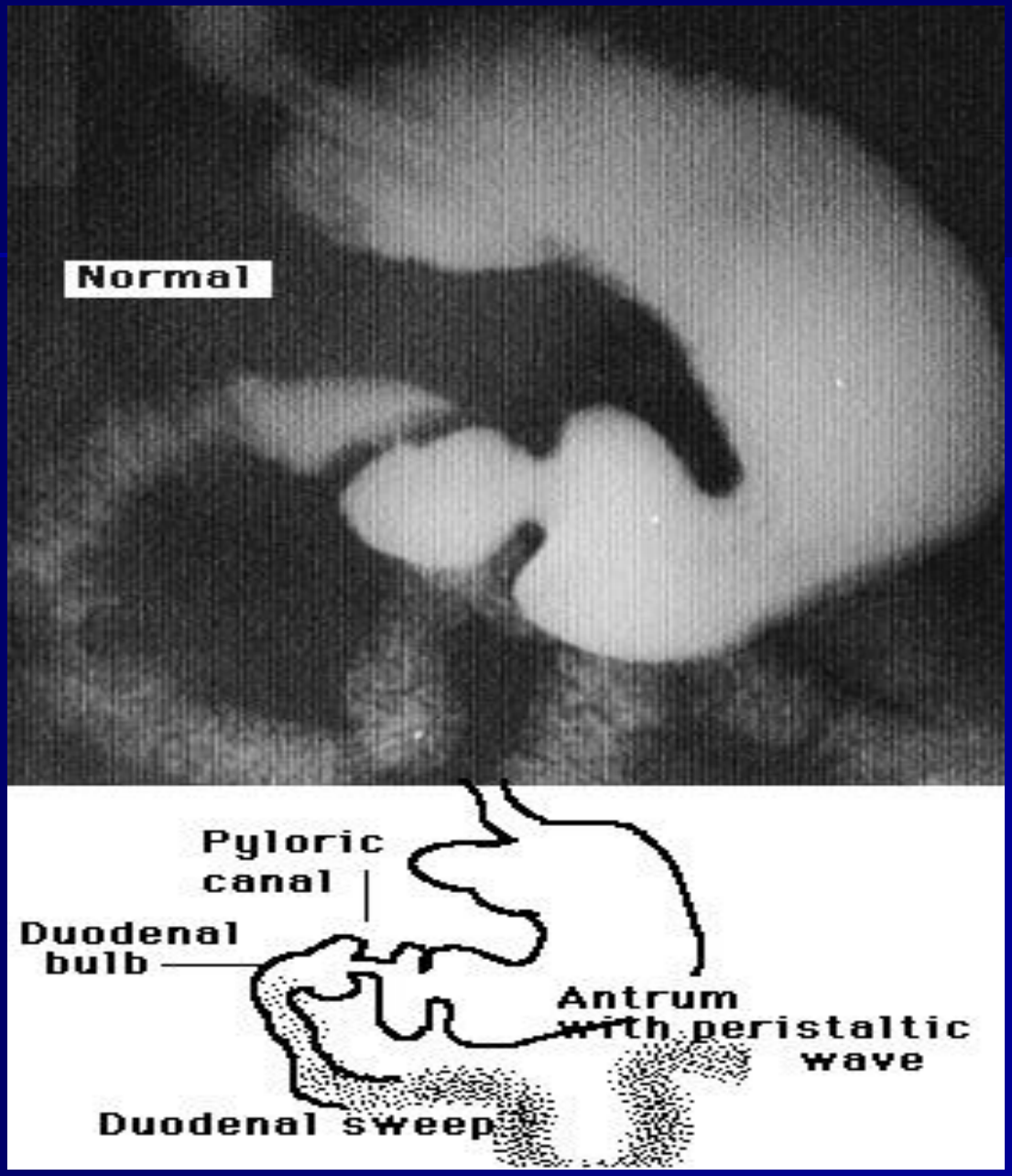
I. Radiological examination:

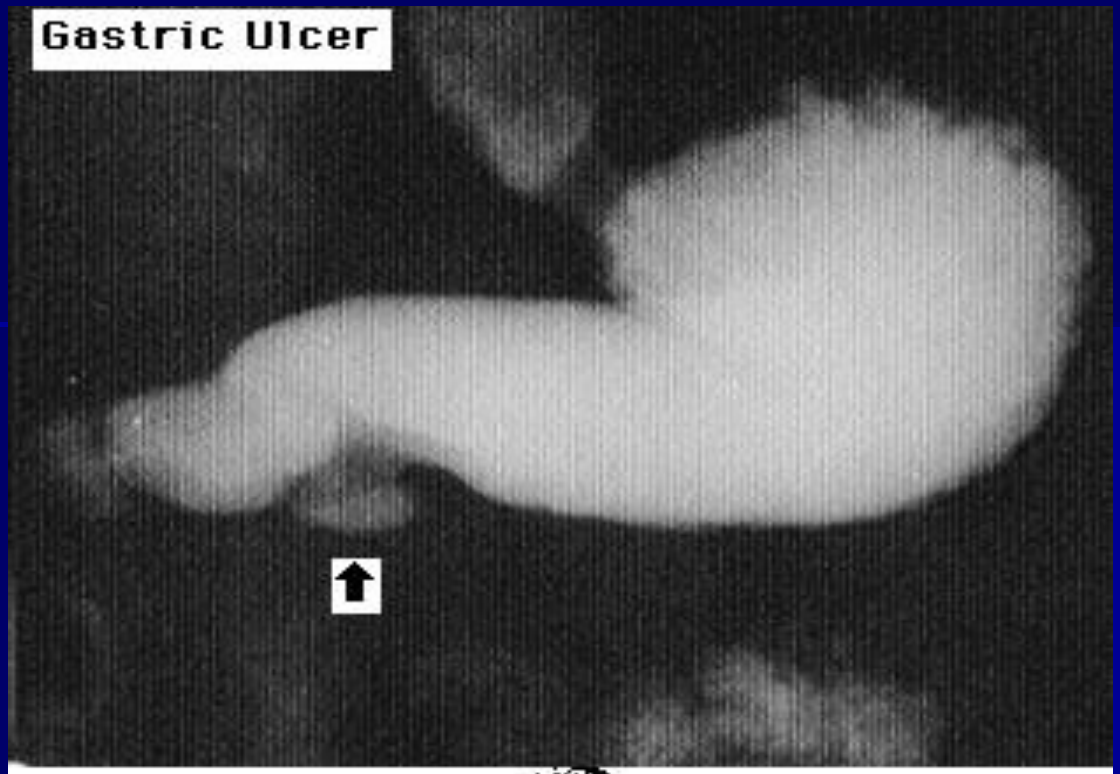
Direct signs:

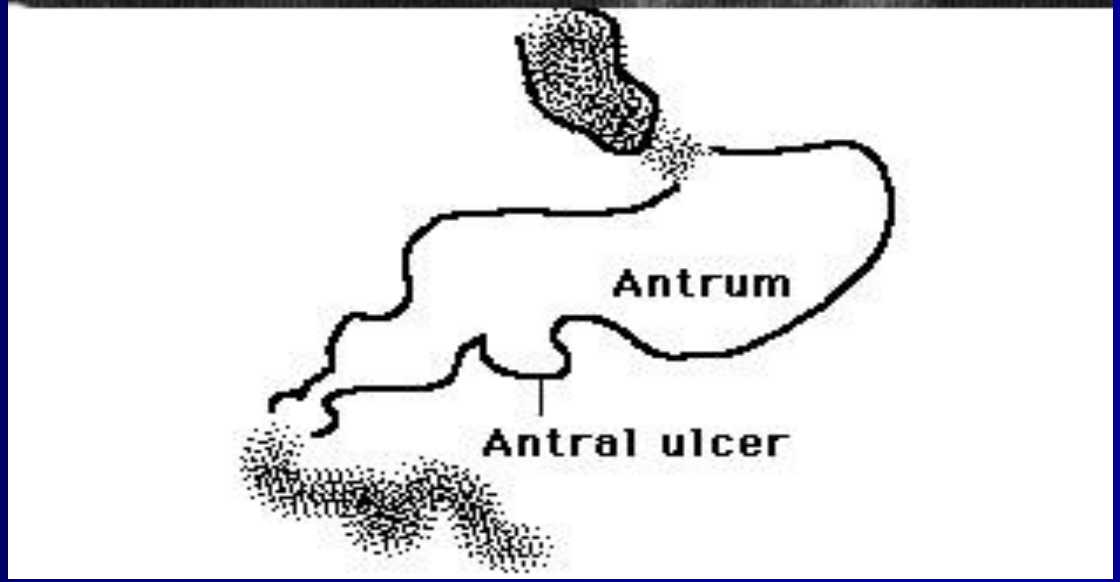
- Presence of niche (on small curvature - 85% of cases, on big curvature or pyloric region - 15% of cases)
- Duodenal niche.

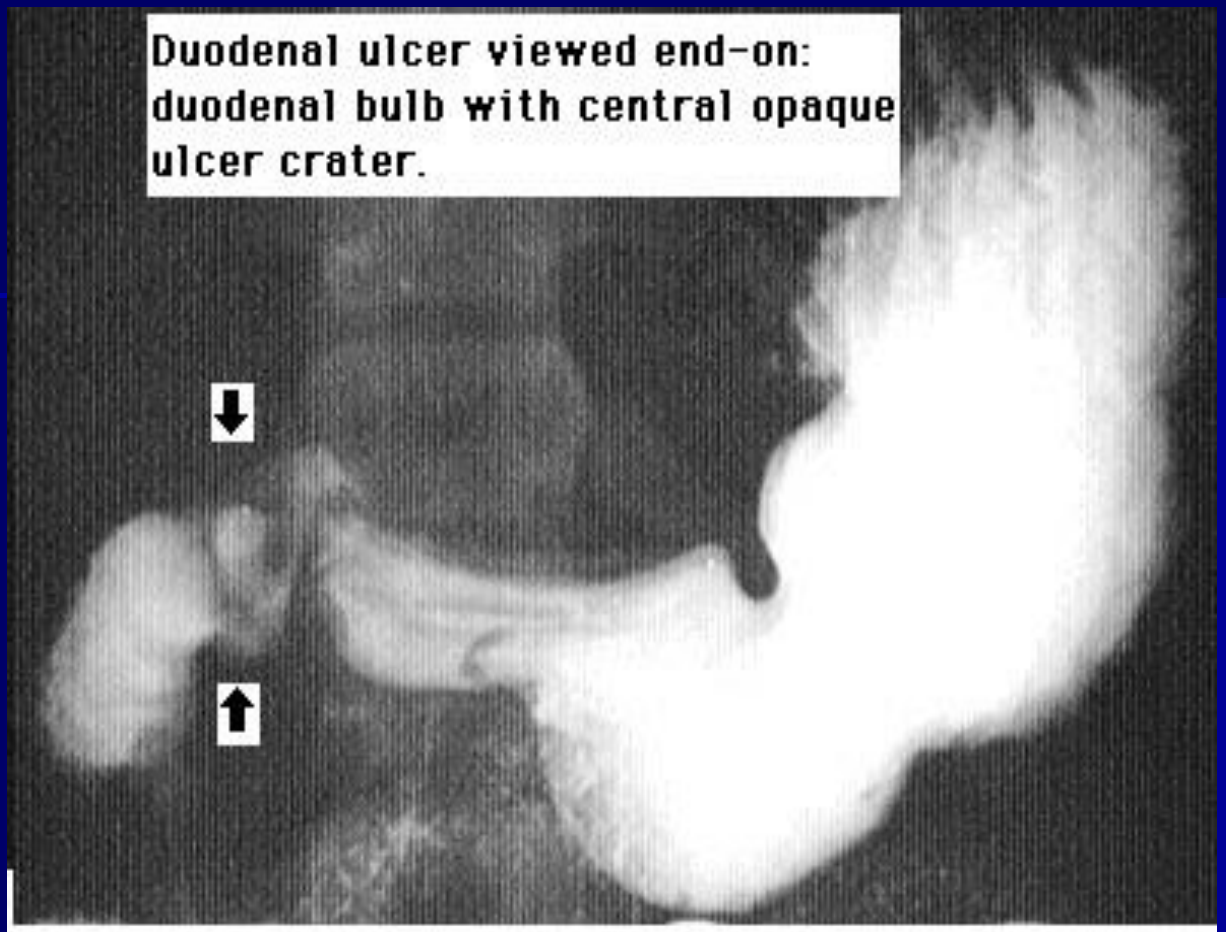
Indirect signs:

- spastic incisure on the opposite wall
- Deformation of duodenal bulb
- Pyloric spasm


Normal
Pyloric canal
Duodenal bulb
Antrum with peristaltic wave
Duodenal sweep

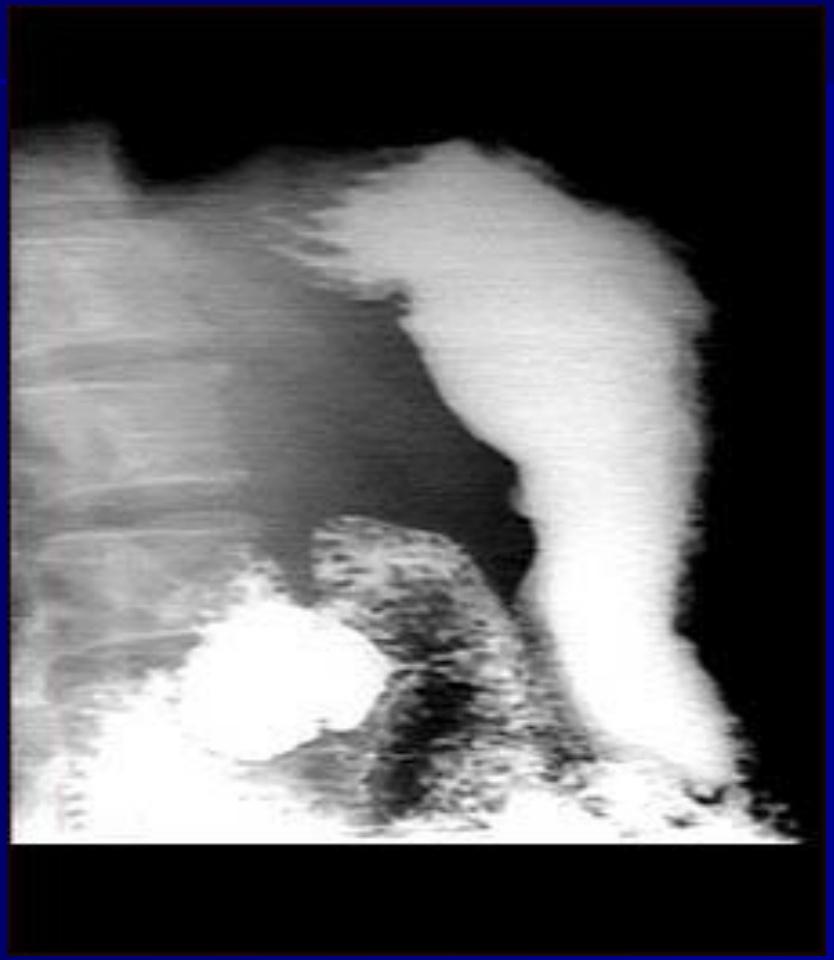

Gastric Ulcer


Antrum
Antral ulcer

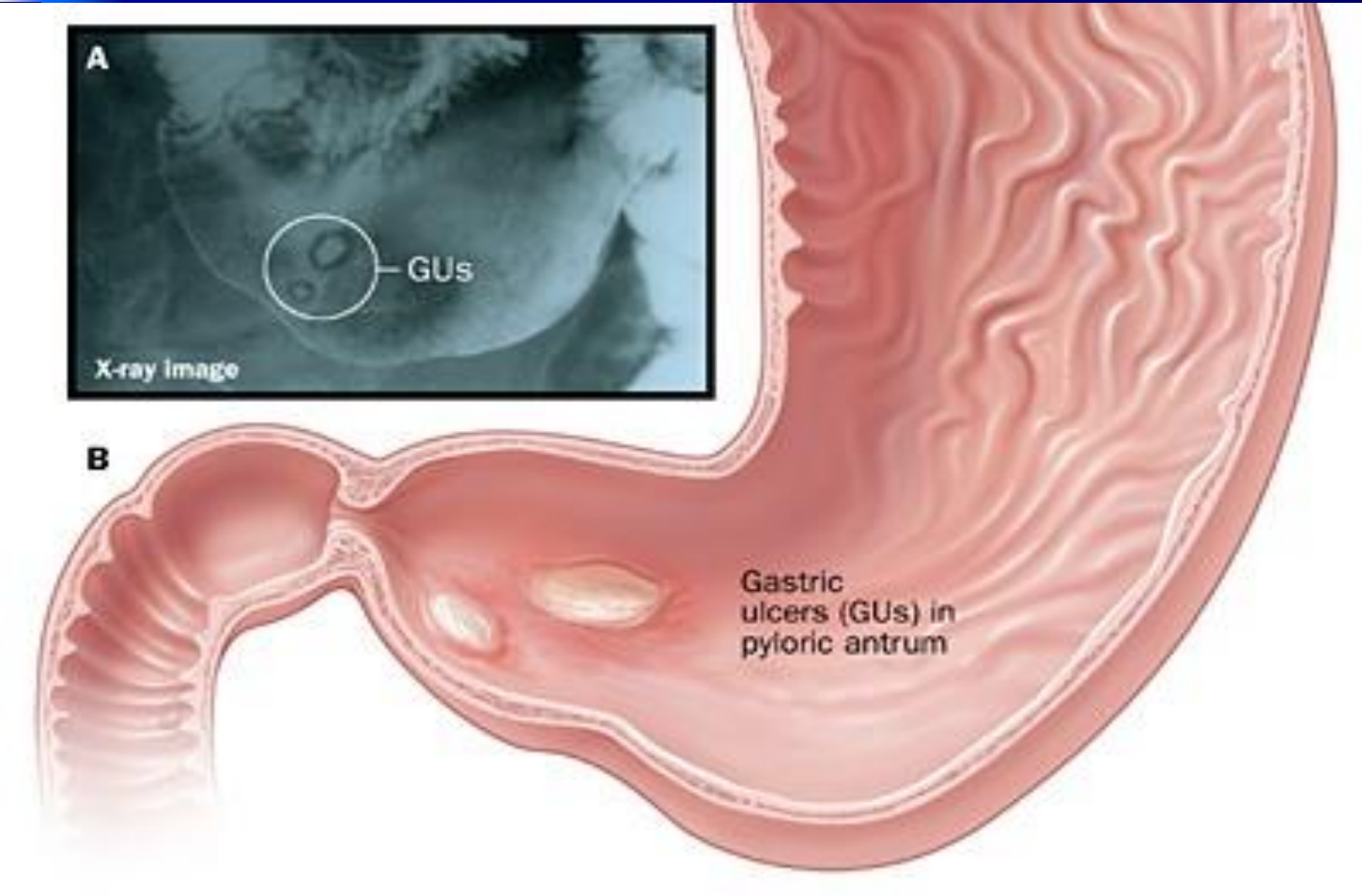



Edematous tissue around ulcer projects into the duodenal lumen and prevents barium from accumulating in the area. This is demonstrated by the dark area surrounding the ulcer.

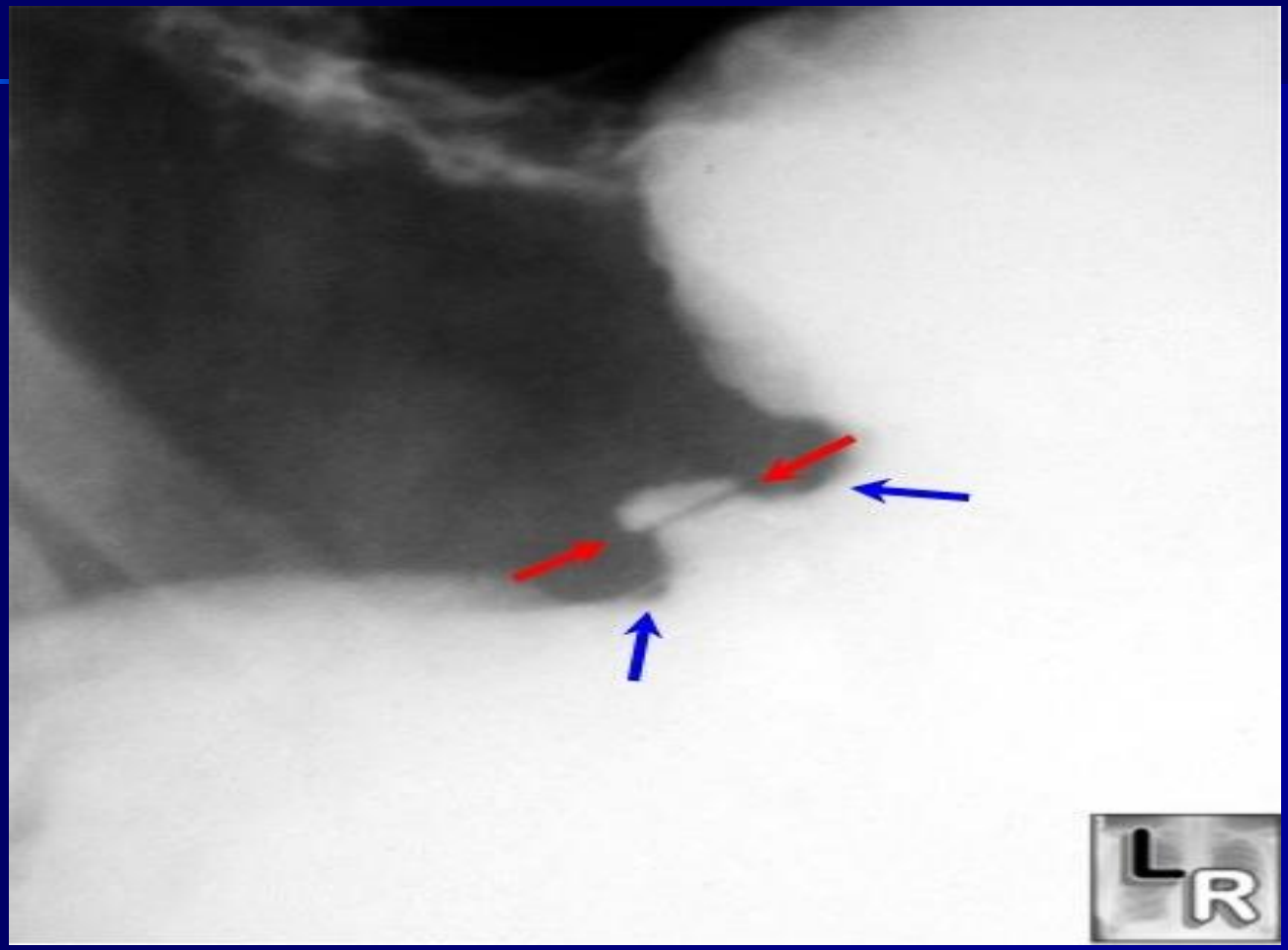
# Gastric ulcer



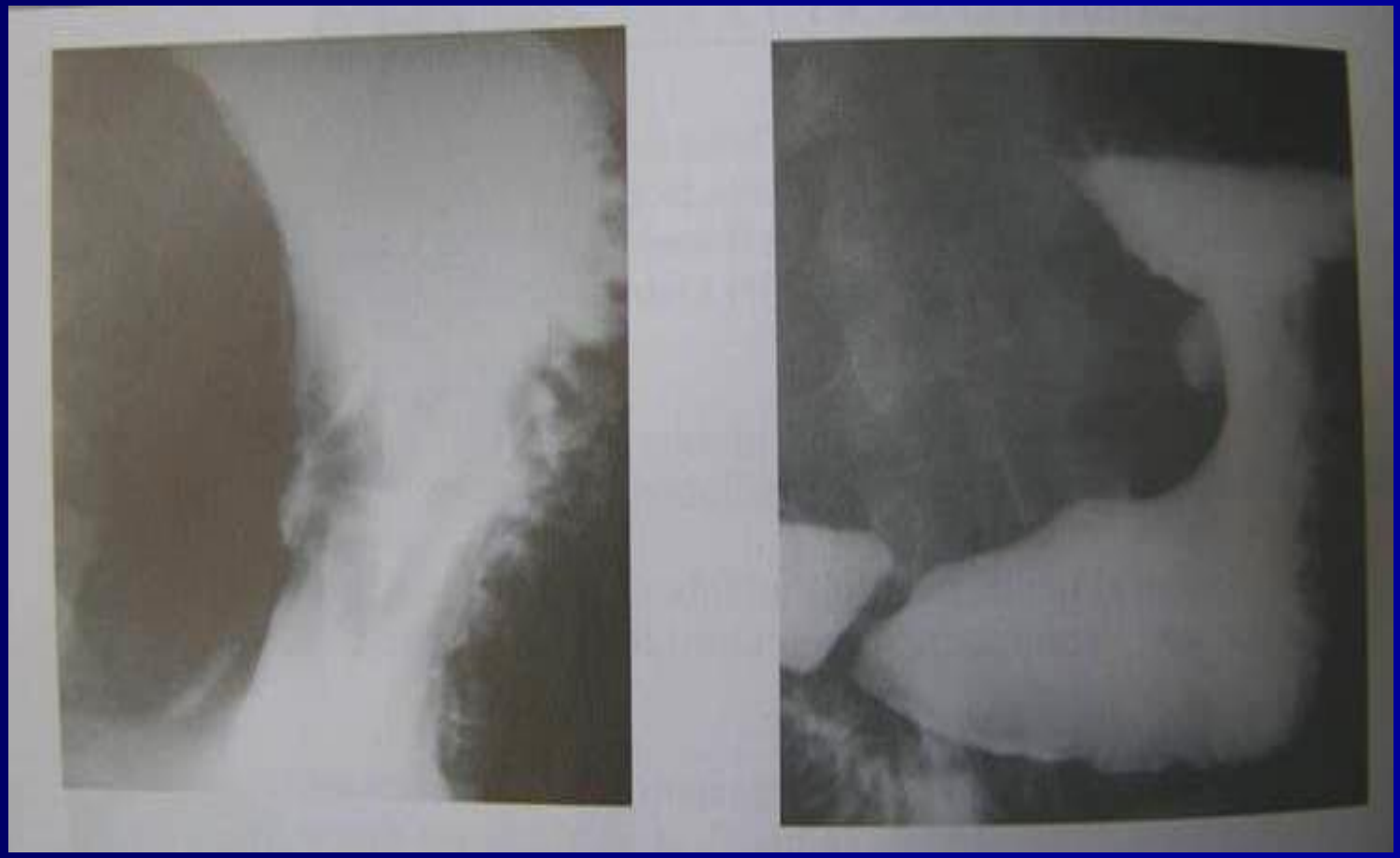
# Radiological examination ulcer



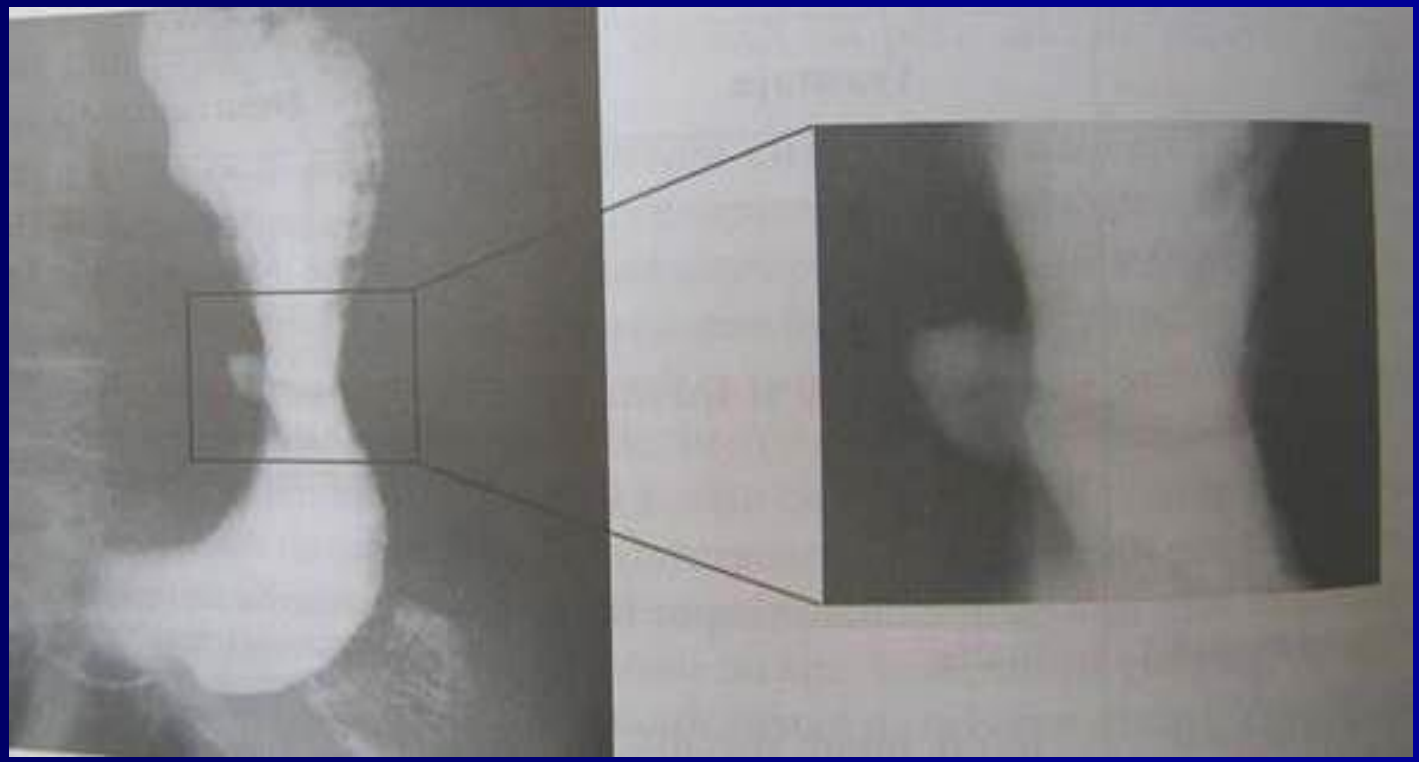
# Gastric ulcer



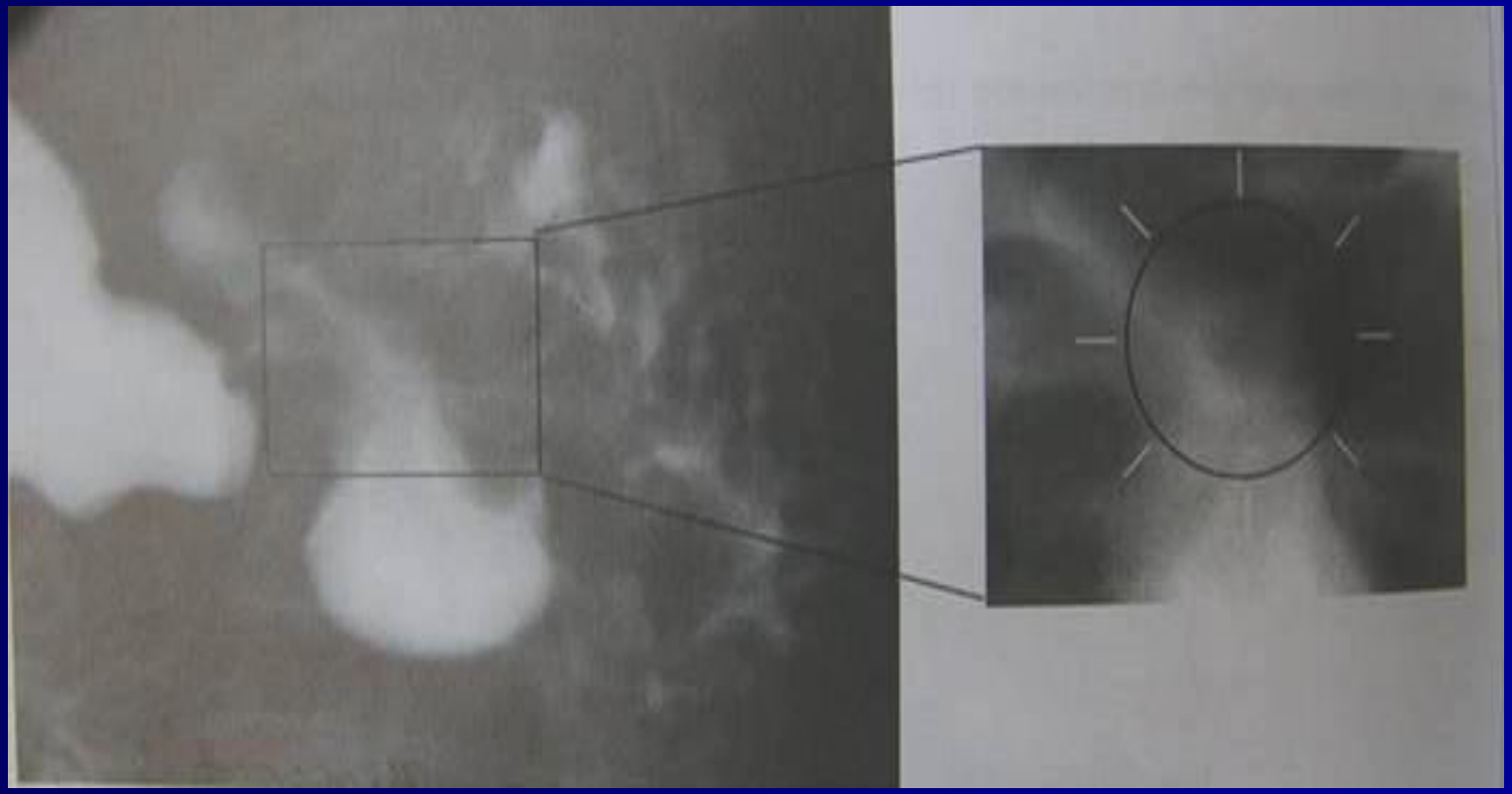
# Malign gastric niche



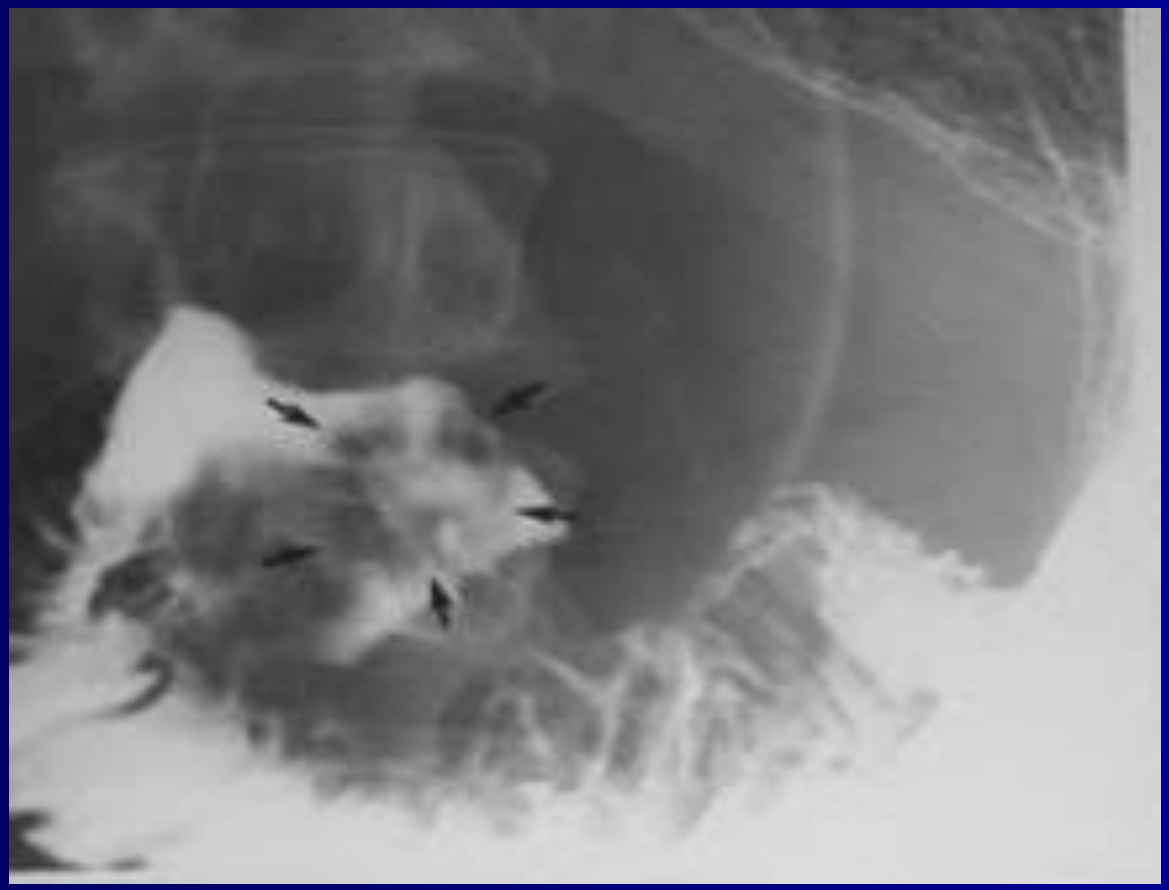
# Gastric niche



# Duodenal ulcer



# Duodenal ulcer



# II. Examination of gastric secretion:

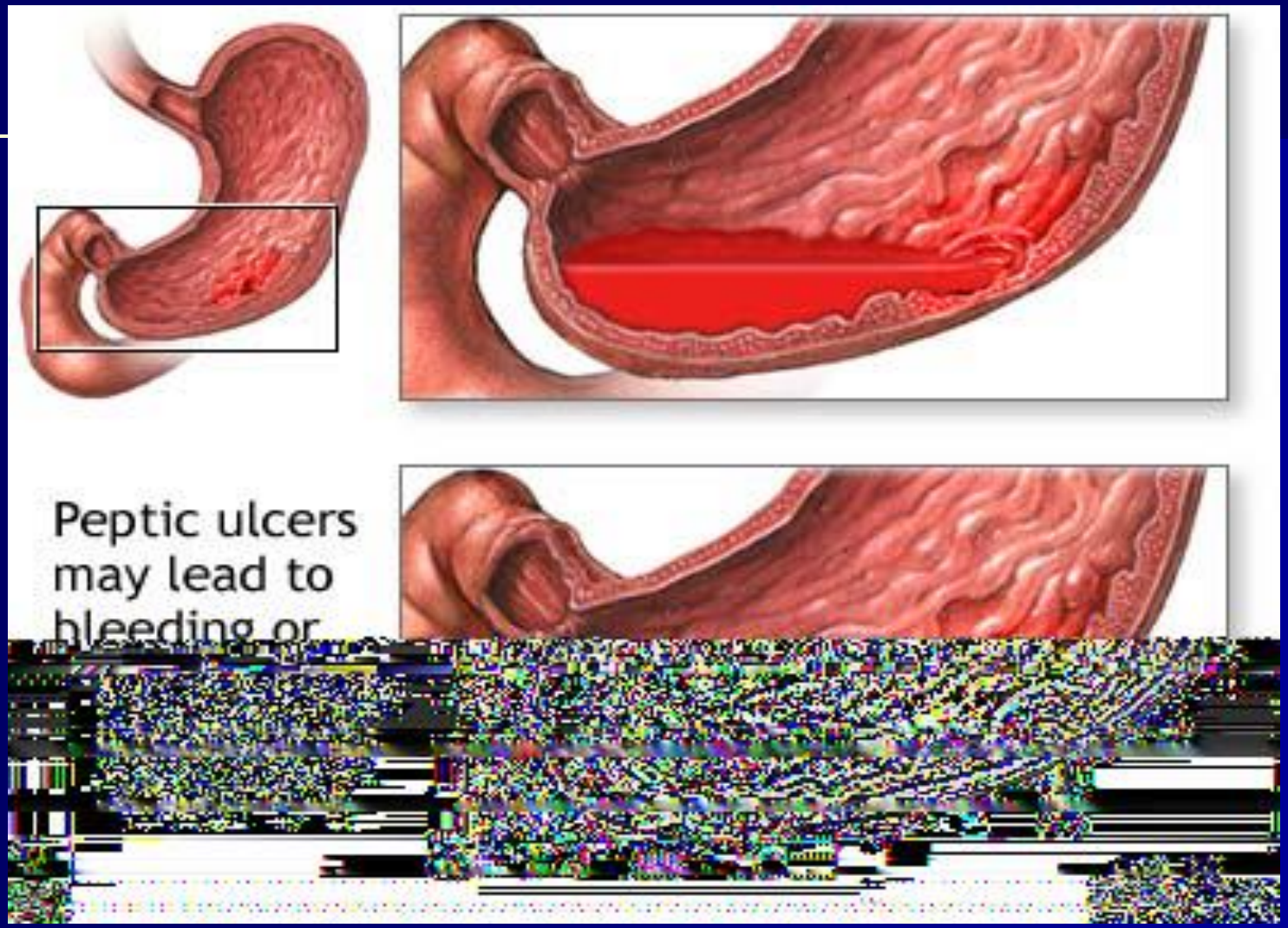
- in duodenal ulcer 50% of cases are associated with hyperacidity
- in gastric ulcer there is a normal or decreased acidity
- Evidence of Helicobacter pylori

# Complications

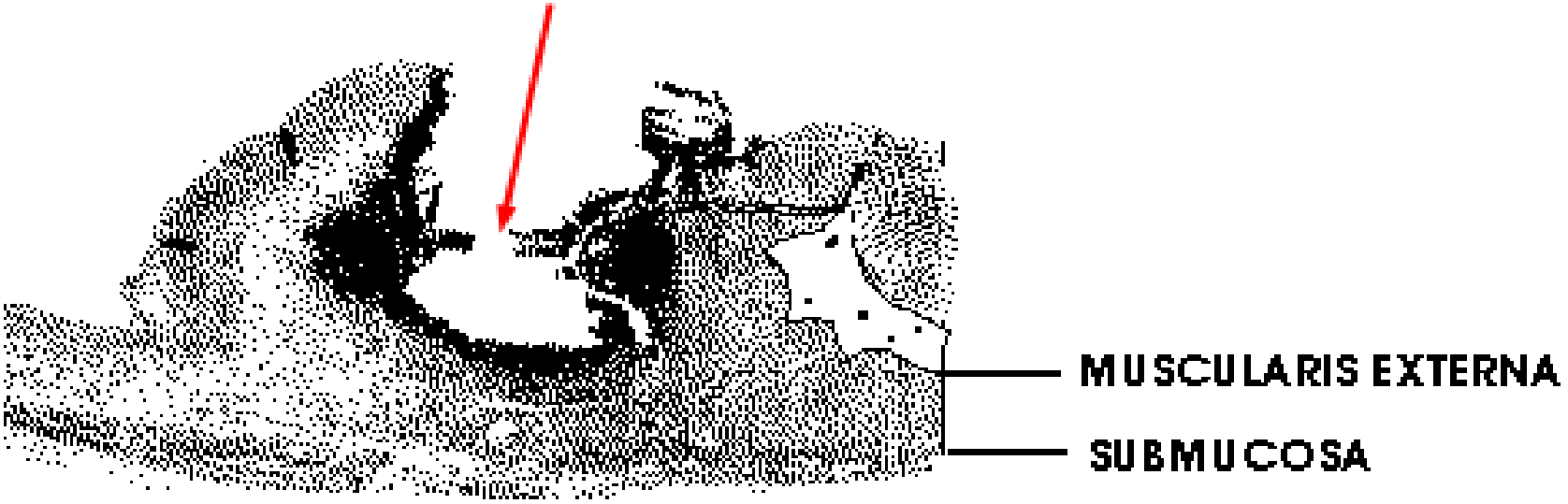
1. Haemorrhage – vomiting with „coffee ground”, melena. Signs of acute anaemia.
2. Perforation
3. Penetration
4. Pyloric stenosis

- Perforation leads to overflow of gastric content into peritoneal cavity

  There is a very intense pain in epigastria (as a dagger shot)
- Penetration – the overflow of gastric content is blocked by the adjacent to the stomach or duodenum organs : peritoneum, pancreas, liver, biliary ducts, colon.

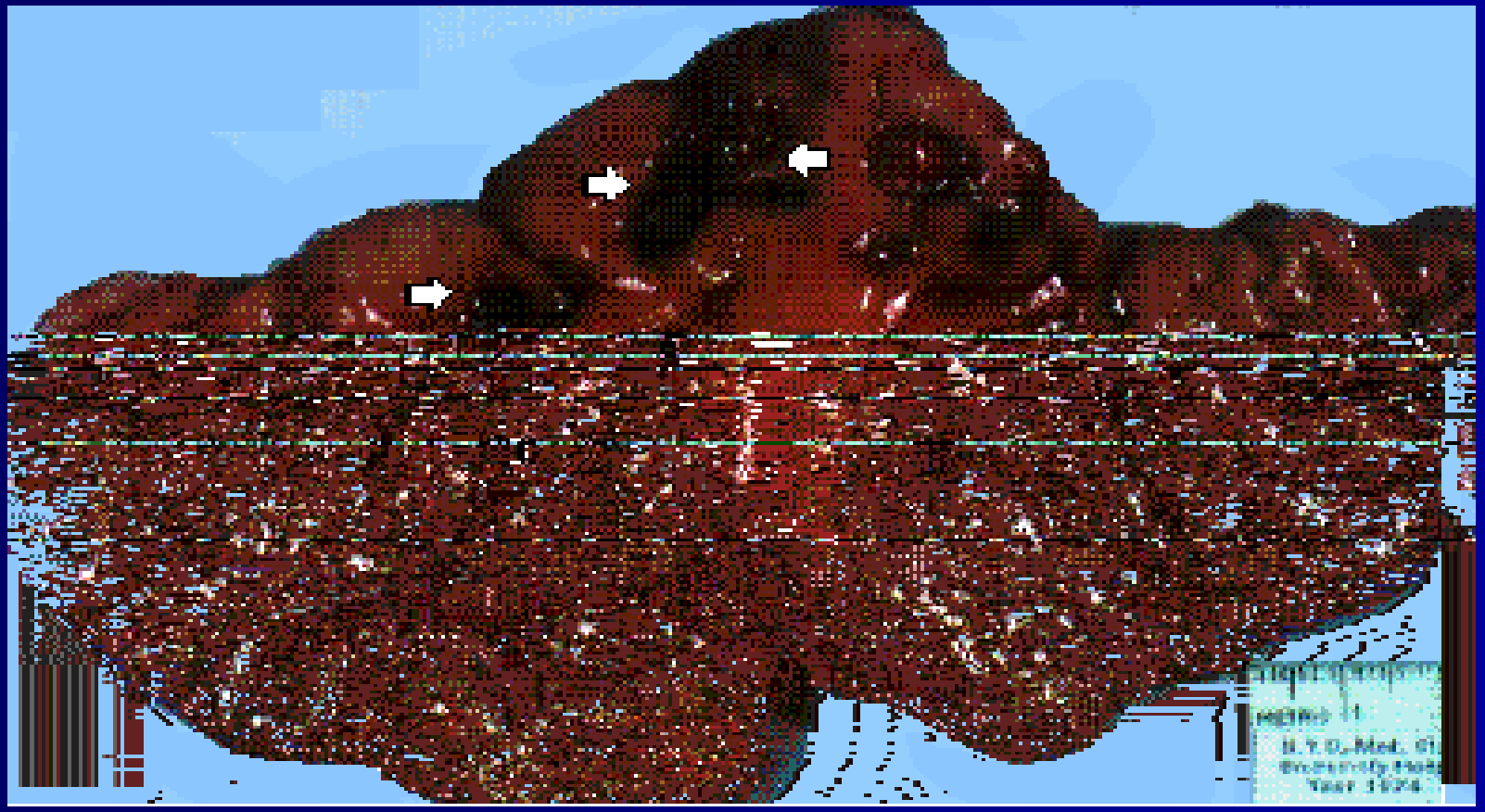

Peptic ulcers
may lead to
bleeding or

Ulcer crater filled with blood.




Damage to blood vessels, such as occurs in a bleeding ulcer, can lead to HEMORRHAGE. This is a serious complication of ulcer disease.

# Ulcer - haemorrhages

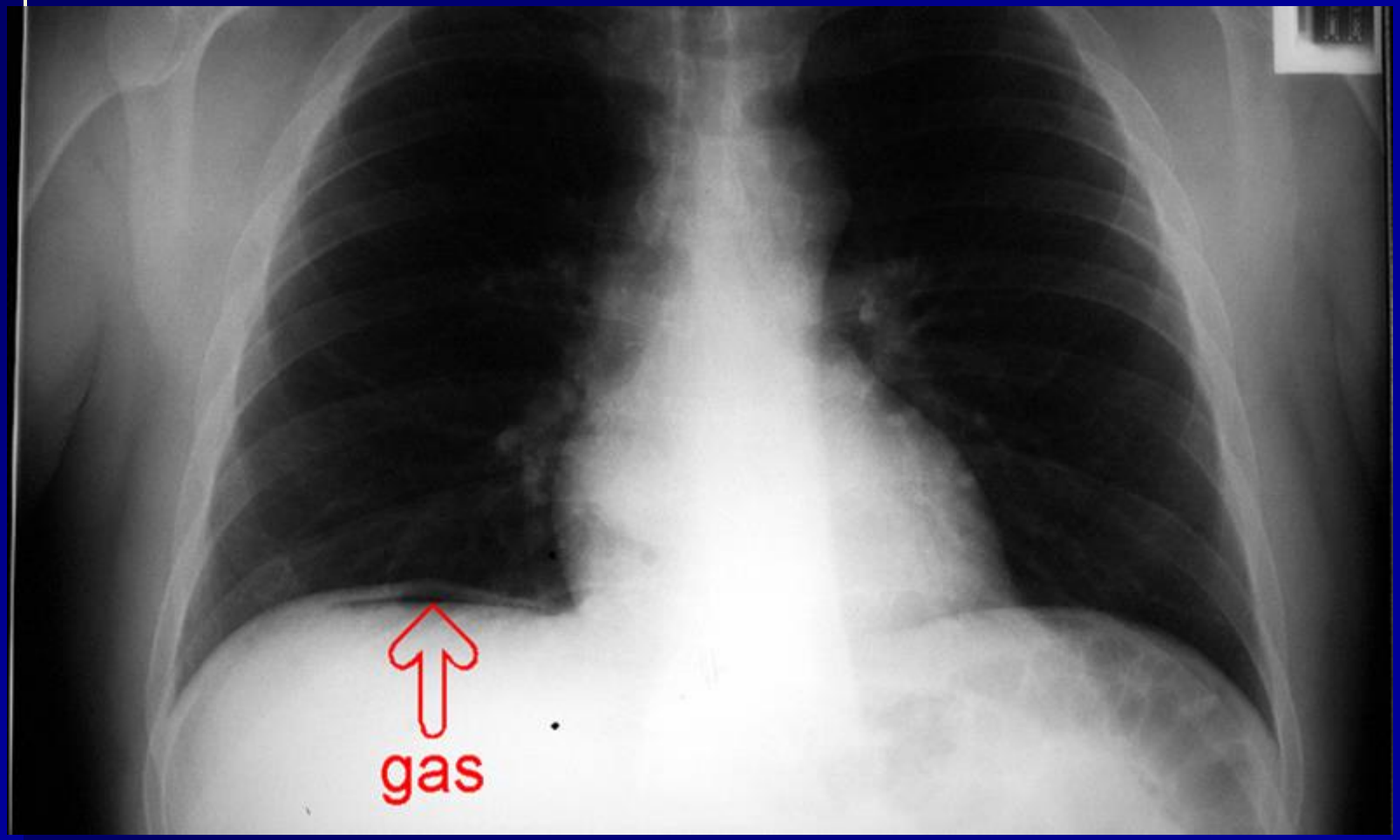


# Radiologic examination in perforation reveals:

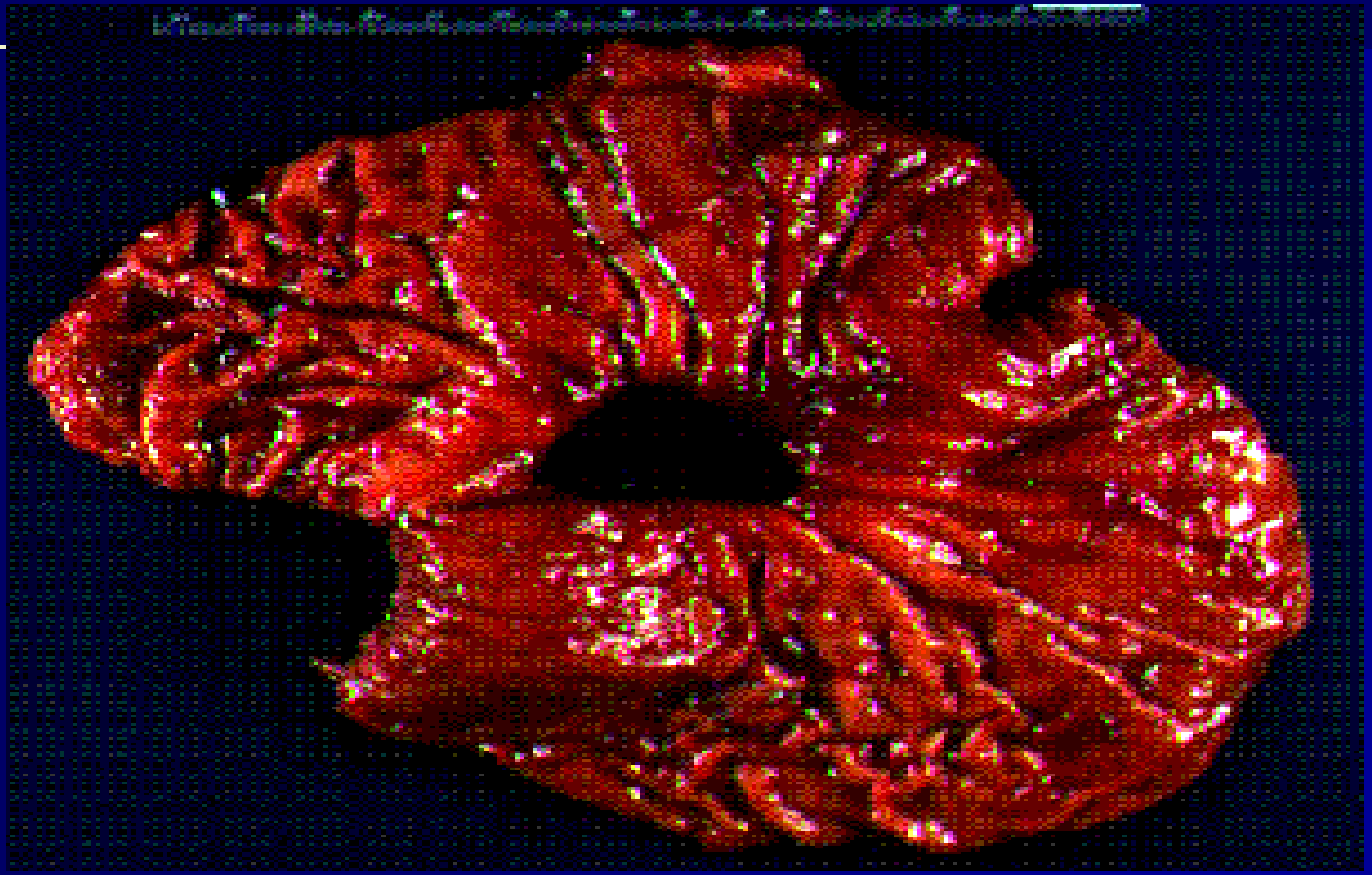
- Presence of air in peritoneal cavity.

- radiologic examination with barium and endoscopic examination in these cases is contraindicated – risk of air inflow.

# Perforated ulcer



# Perforated ulcer



# Pyloric stenosis

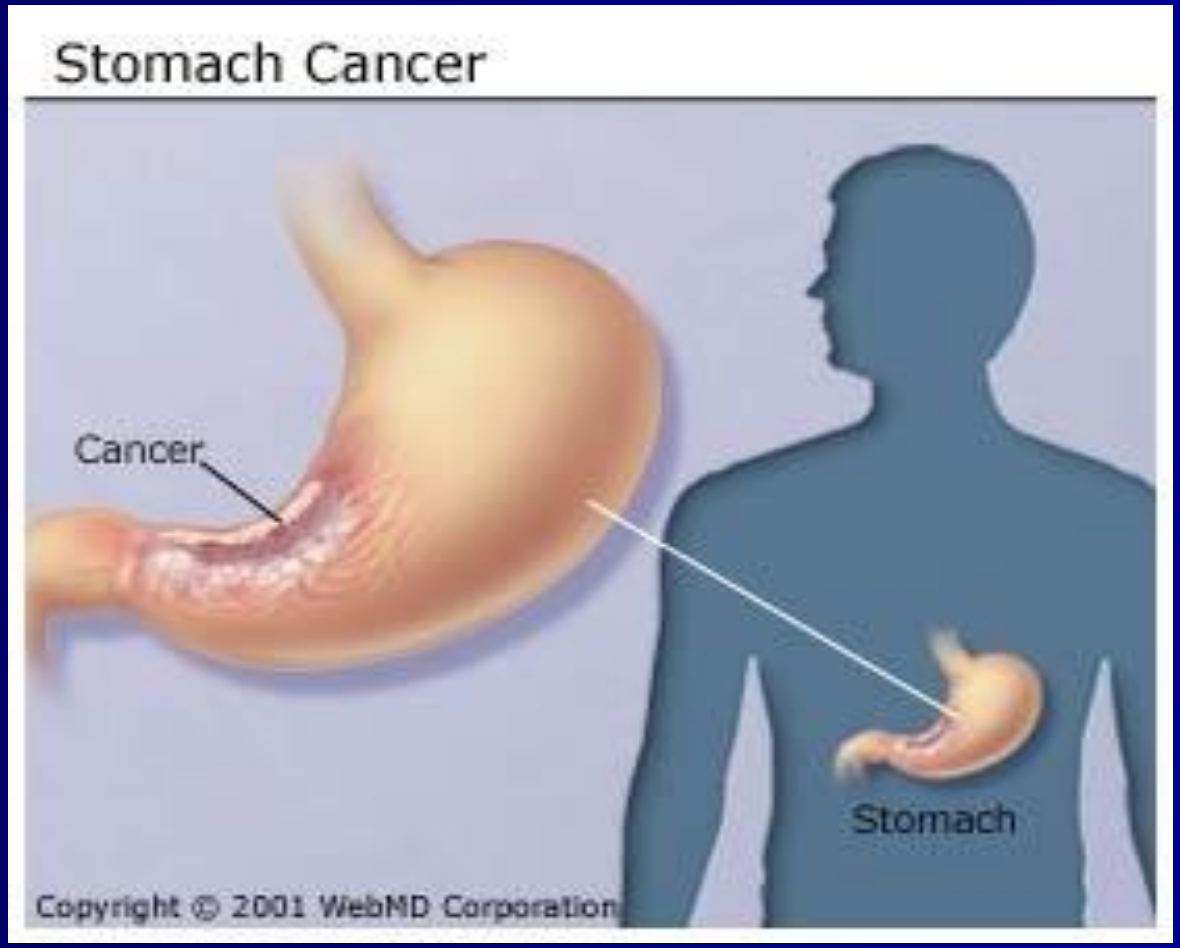
Cause – narrowing after ulcer healing, periulcerous oedema, pyloric spasm.

The main symptom is vomiting (with residues of previously ingested food)

Weight loss

Pyloric stenosis is diagnosed endoscopically.

# Gastric cancer



# Gastric cancer

Macroscopically gastric cancer is presented in one of following forms:

- an ulcerated protrusive formation, prominent in lumen
- infiltrative form with narrowing of gastric lumen
- polypoid aspect
- tumour with superficial extension

# Gastric cancer is classified according to extension of the lesion:

- incipient
- advanced

# *Aetiopathogenesis*

The cause of cancer is unknown. The following risk factors are supposed to be involved:

- hereditary (blood group A)
- alimentary (nitrate, salt, meat, fish abuse)
- Gastric precancerous conditions: atrophic gastritis, gastric polyps, immunodeficient conditions, infection with HP

# *Clinical picture*

Incipient gastric cancer is asymptomatic or is manifested with nonspecific dyspeptic phenomena.

Clinical examination is normal.

Advanced gastric cancer is manifested with:

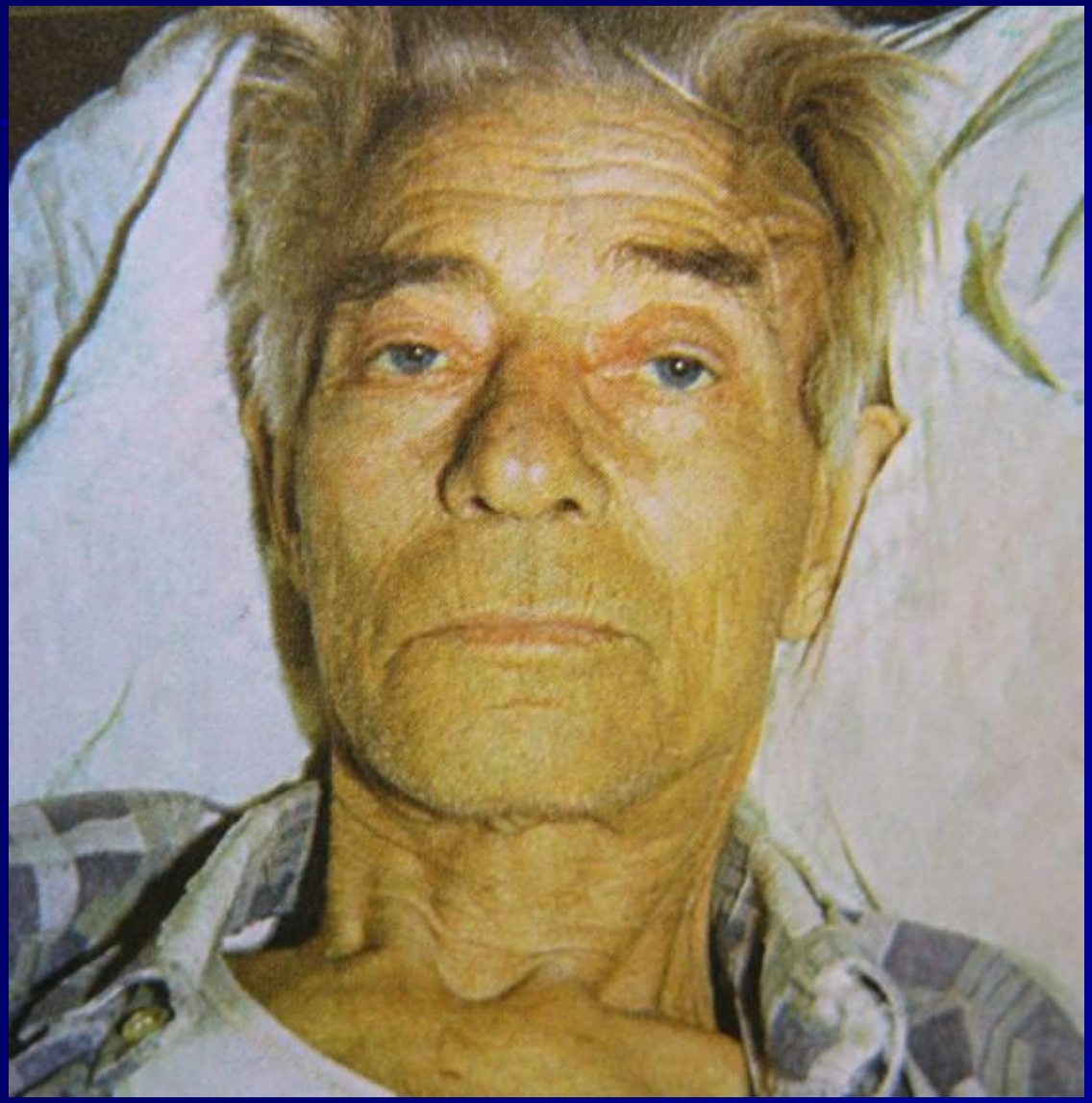
- permanent epigastric pain,
- nausea, vomiting,
- loss of appetite (sometimes selective anorexia for meat, which produce bitter taste due to aminoacids and urea),
- fatigue, weight loss.

- If metastases in liver - jaundice, ascitis.
- If metastases in lungs – dyspnoea.
- If metastases in vertebrae - pain in vertebral column
- If metastases at the level of central nervous system – hear ache, dizziness.
- If metastases in ovaries – pain in the inferior abdomen

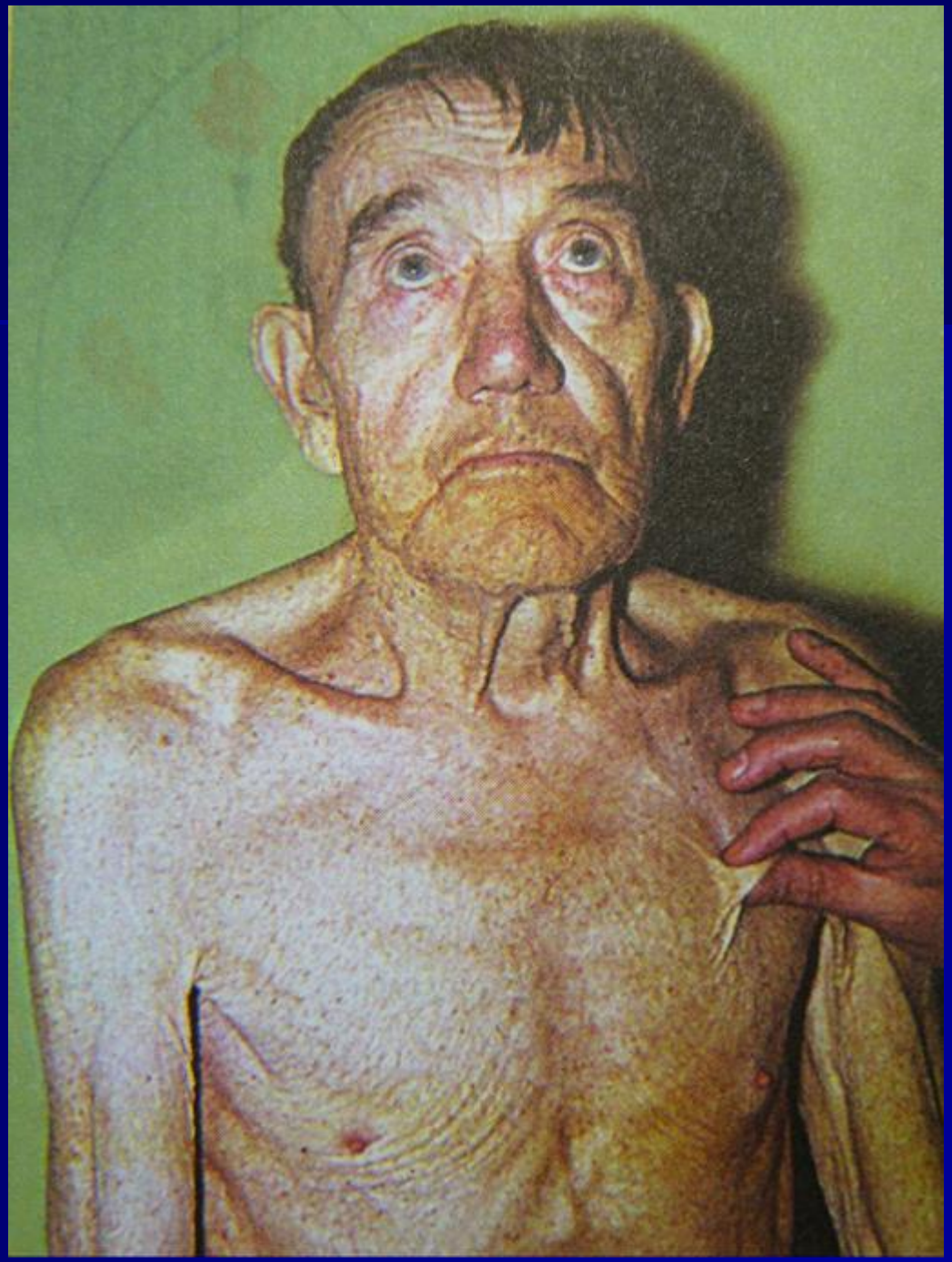
# Clinical examination

- Pale skin,
- Enlarged left supraclavicular lymph nodes (Virchow sign), left axillary lymph nodes, prerectal lymph nodes (Straus sign)
- Hepatomegaly if hepatic metastases

# Gastric cancer, Mt liver, anaemia



**Gastric cancer** anaemia, cachexia



- gynaecological examination could reveal increase of ovaries due to metastasis (Krukenberg tumour, usually bilateral).

## Complications of gastric cancer

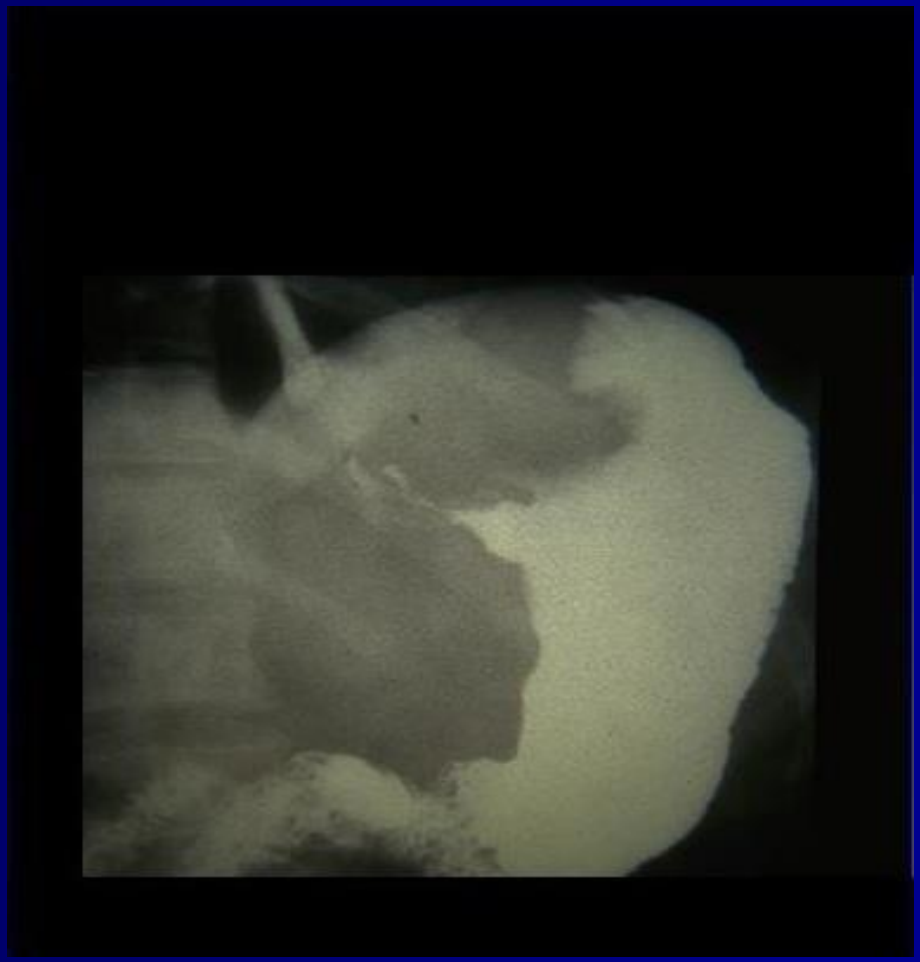
- superior digestive haemorrhage
- insufficient gastric evacuation in stenosing antro-pyloric lesions
- oesophagean syndrome with dysphagia in stenosing cardio-tuberozitar cancer
- gastro-colonic fistula (foul vomiting)

# *Paraclinical exploration*

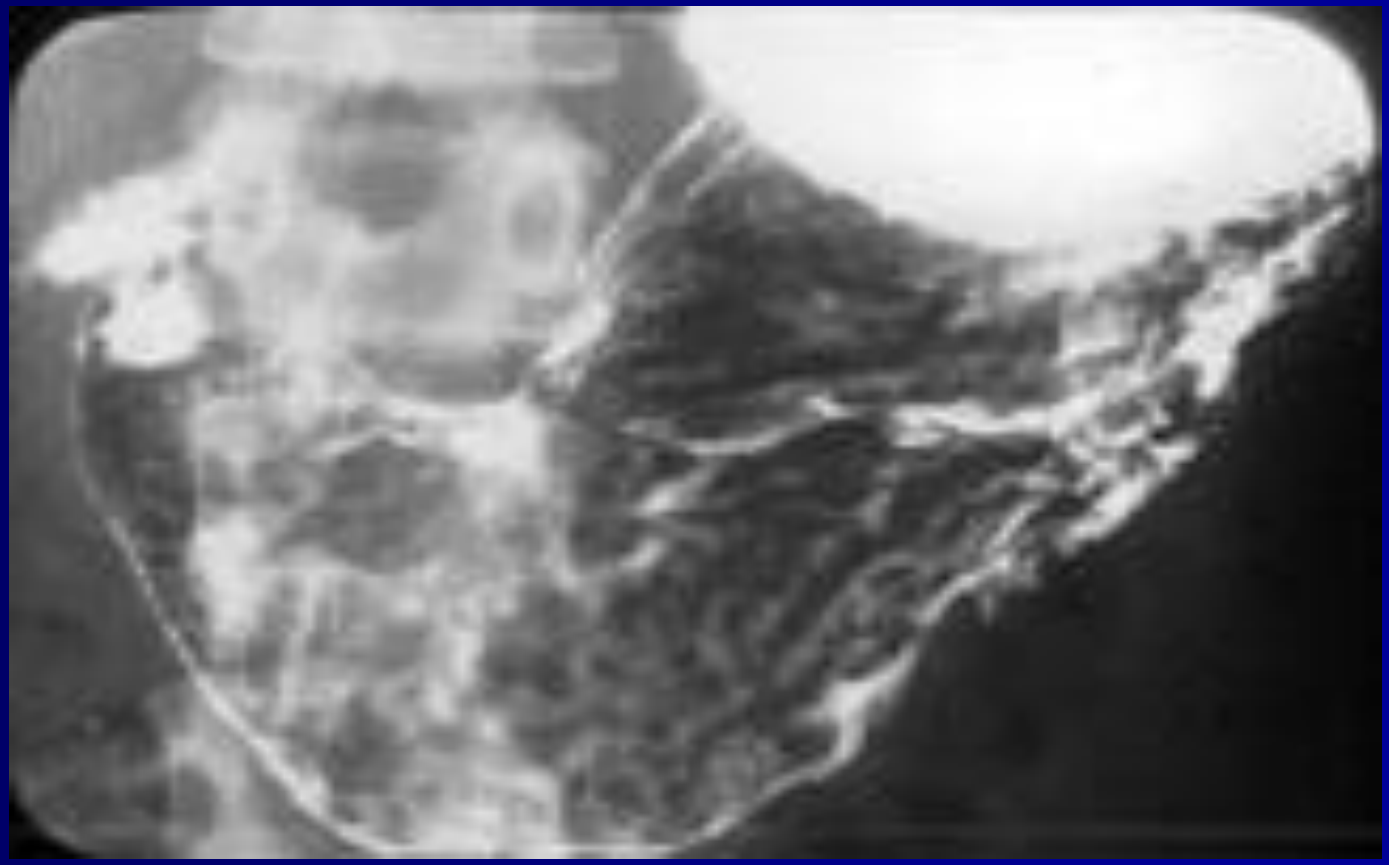
Radiologic examination with barium:

- proliferative form looks like a filling defect;
- ulcerative form – looks like a niche, usually unregulated (the niche has a large implantation basis, the gastric wall around it could be infiltrated, rigid, and the gastric folders do not reach the niche).
- infiltrative form - looks like segmental rigidity of the whole stomach.

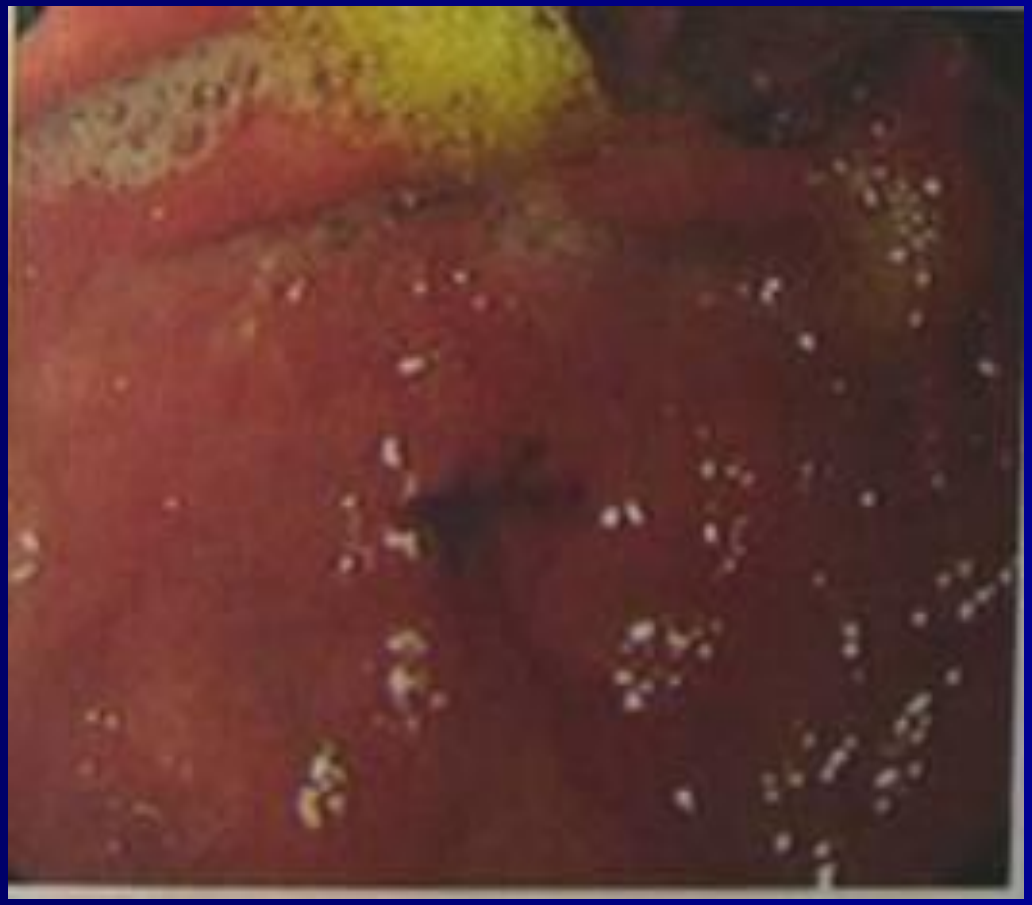
# Gastric cancer (fundus)



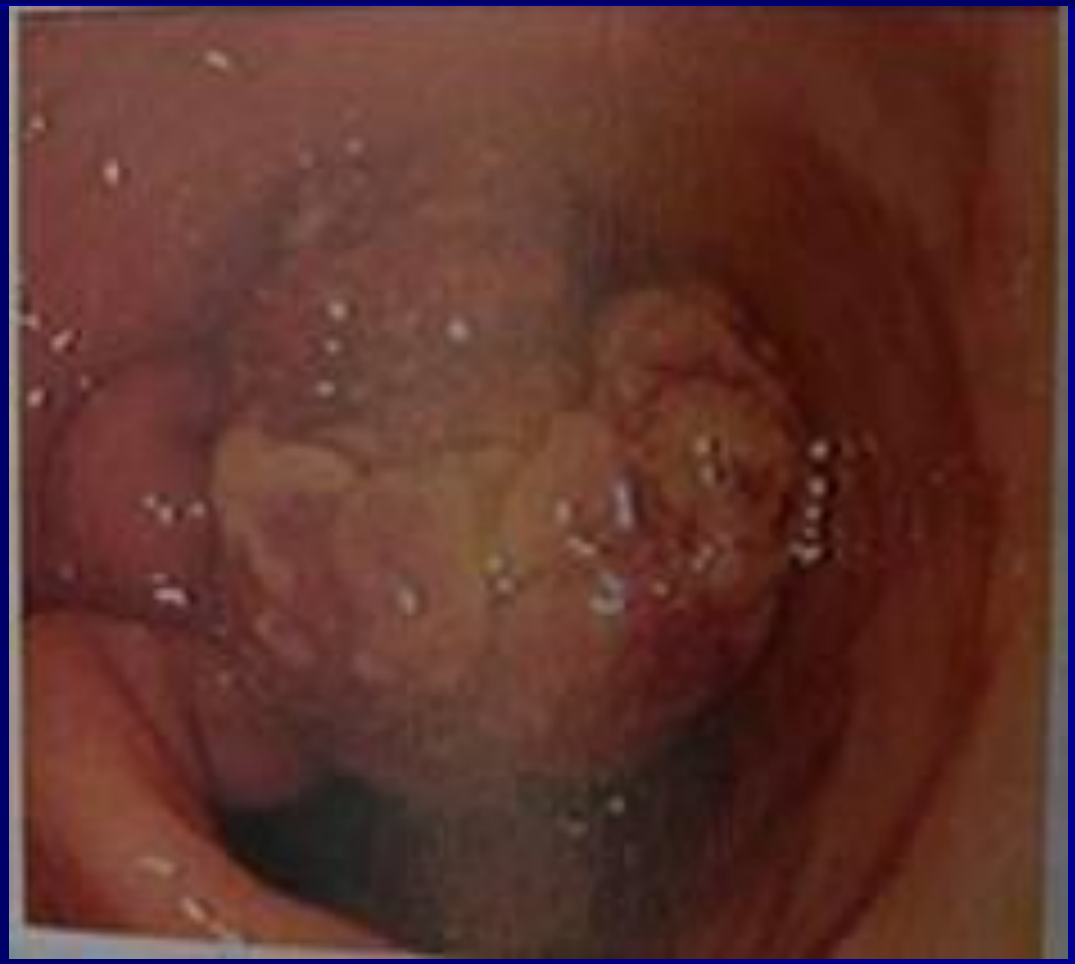
# Gastric carcinoma



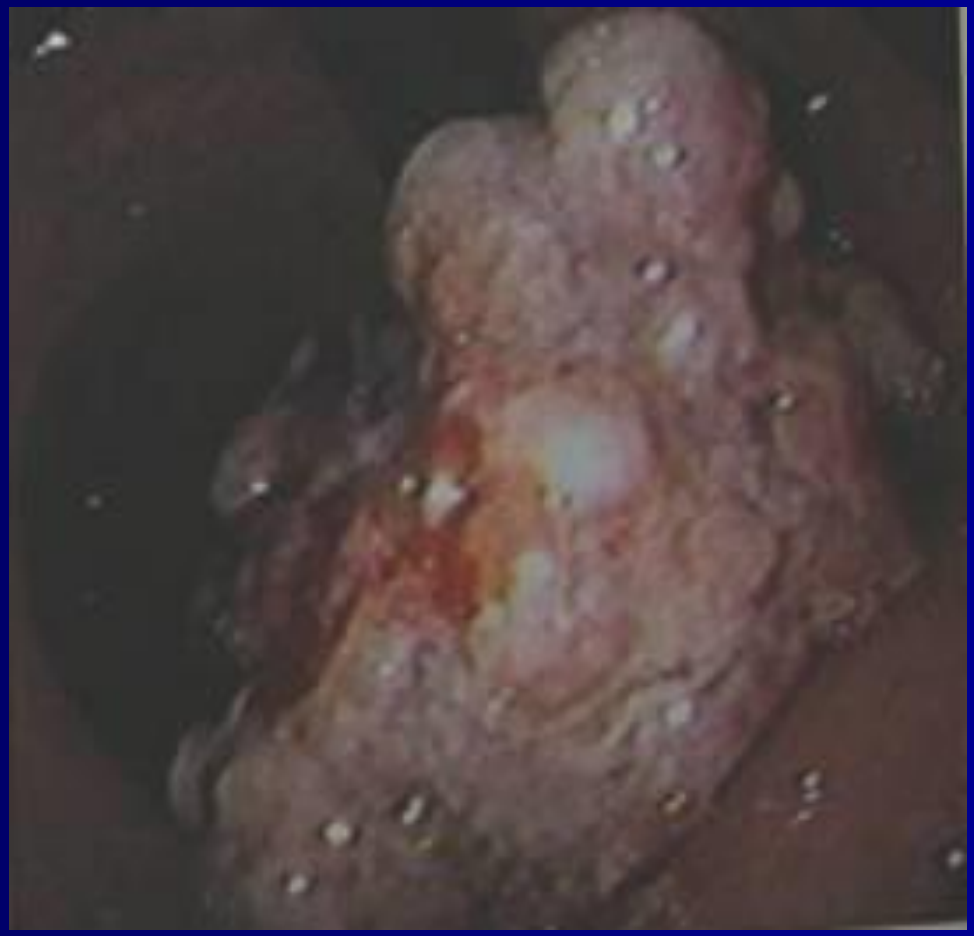
# Antral infiltrative-stenosing gastric cancer



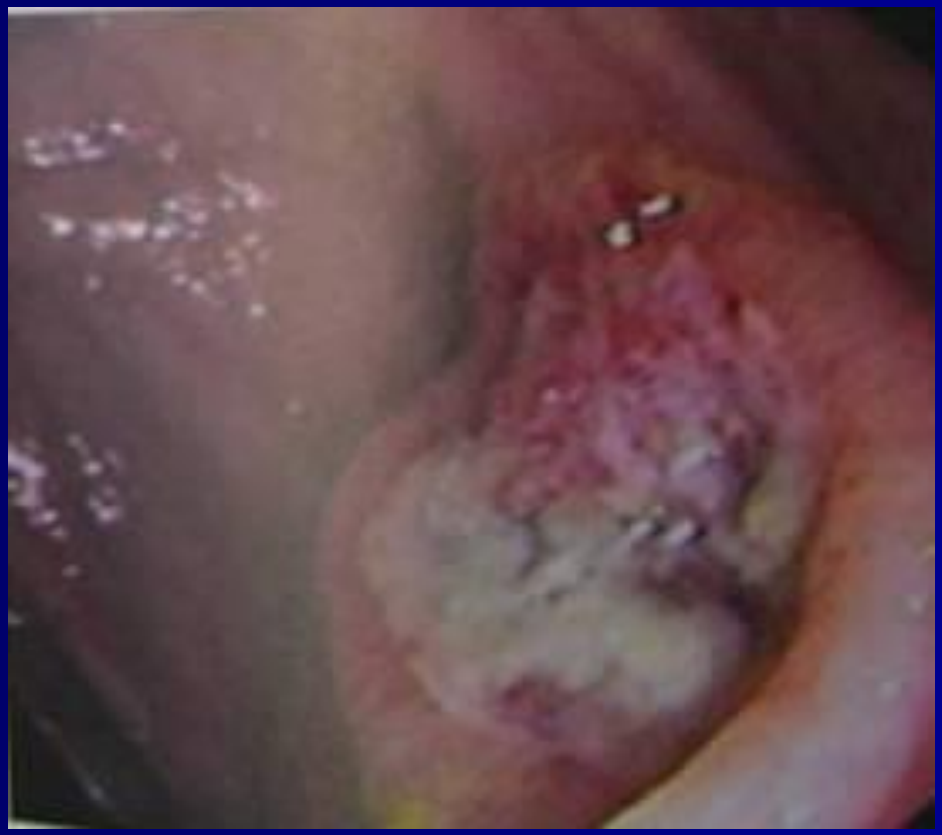
# Polypoid gastric tumor



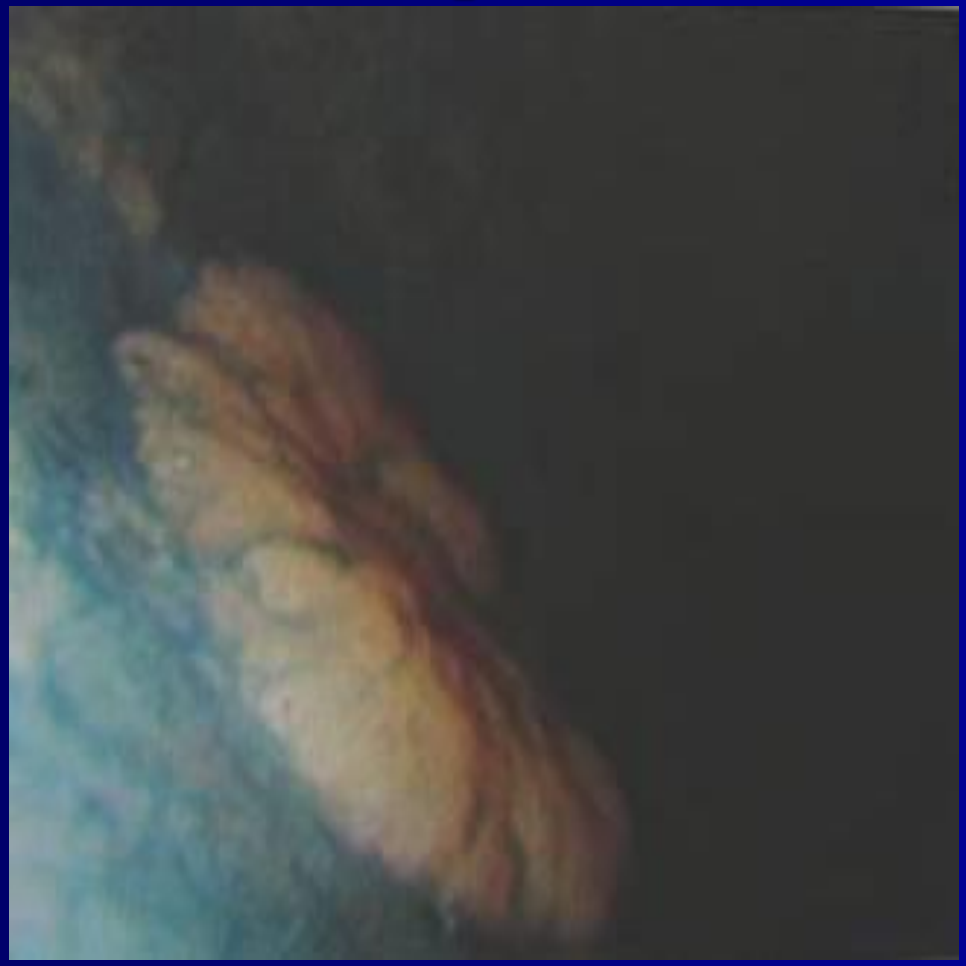
# Vegetating gastric cancer



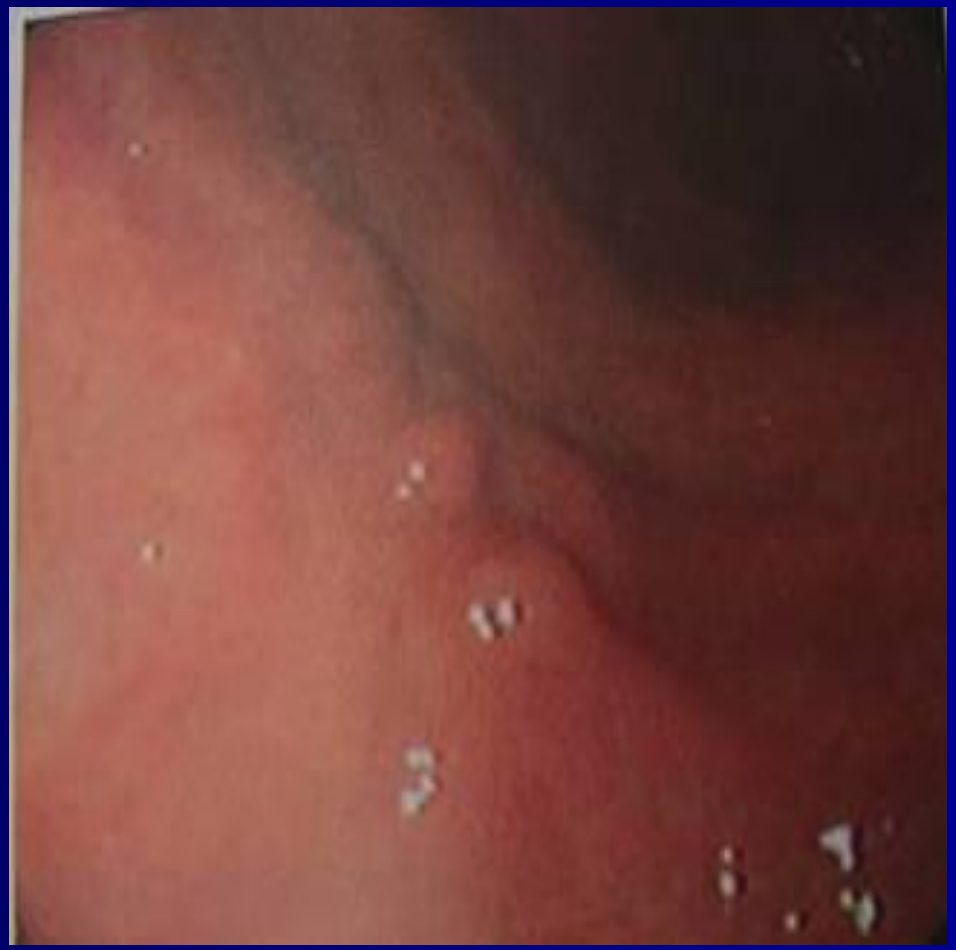
# Ulcerated gastric cancer



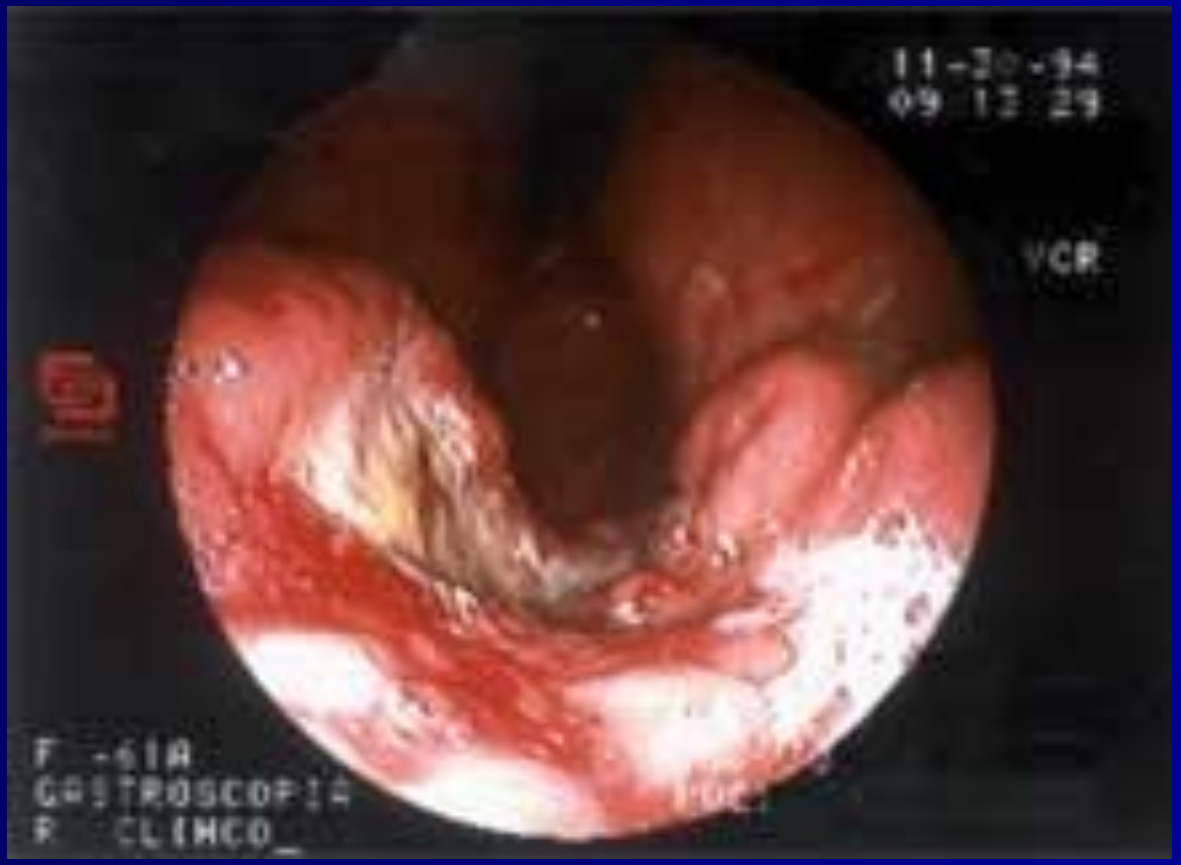
# Gastric cancer (chromoendoscopy with indigocarmin)



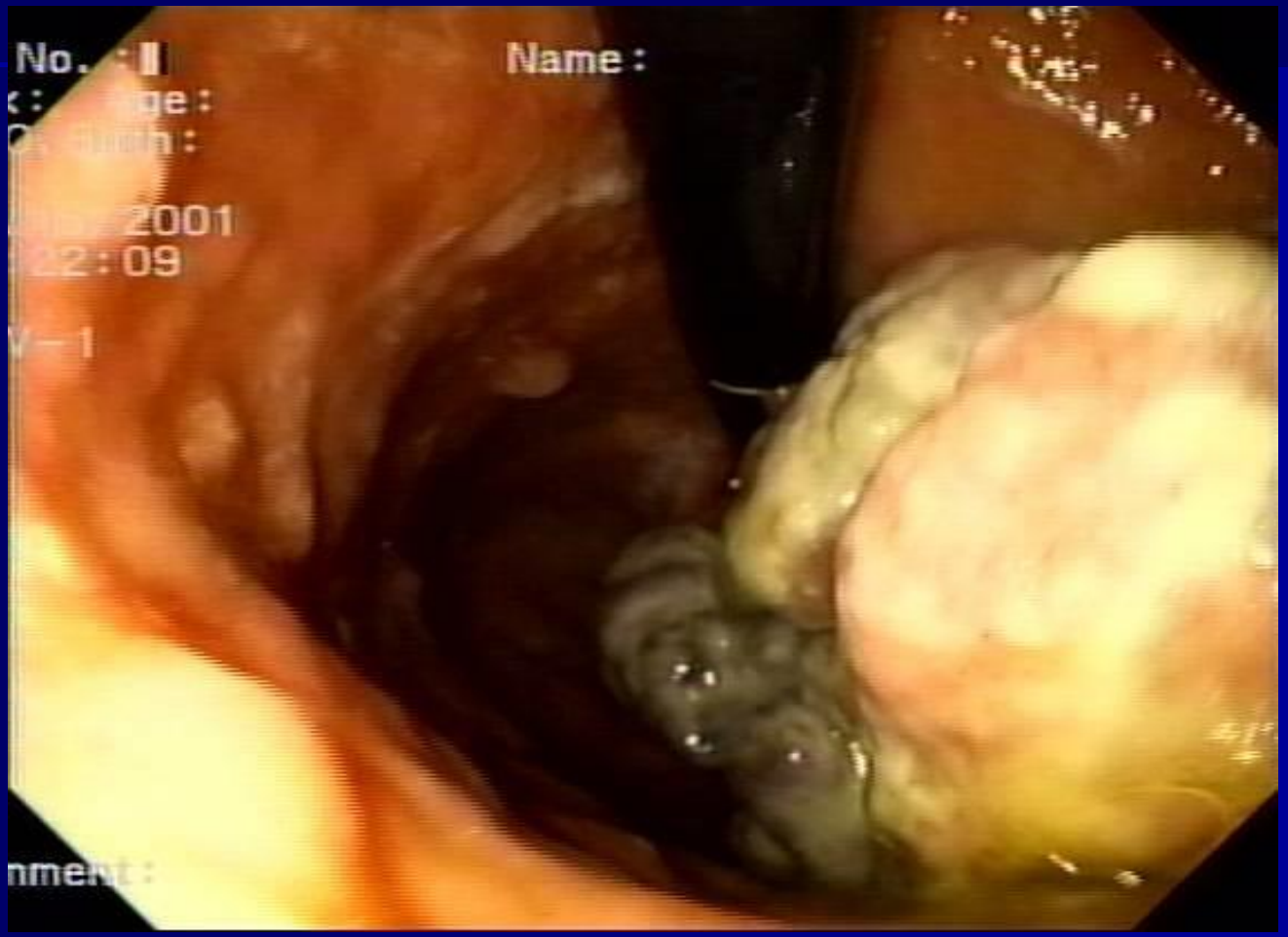
# Gastric cancer, early stage



# Gastric cancer



# Gastric cancer



- Endoscopic examination with biopsy and cytological examination is the method allowing the correct diagnosis.

# Histo cancer