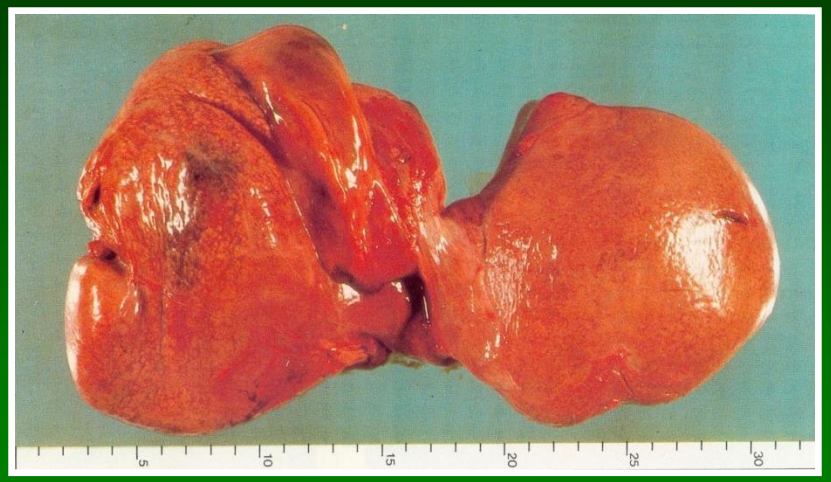
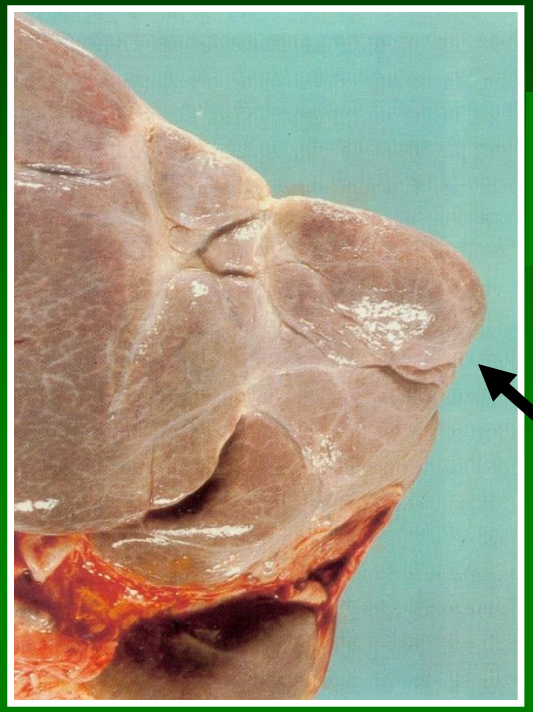
LECTURE 16

PATHOLOGY OF LIVER AND BILE DUCTS



LOBAR LIVER



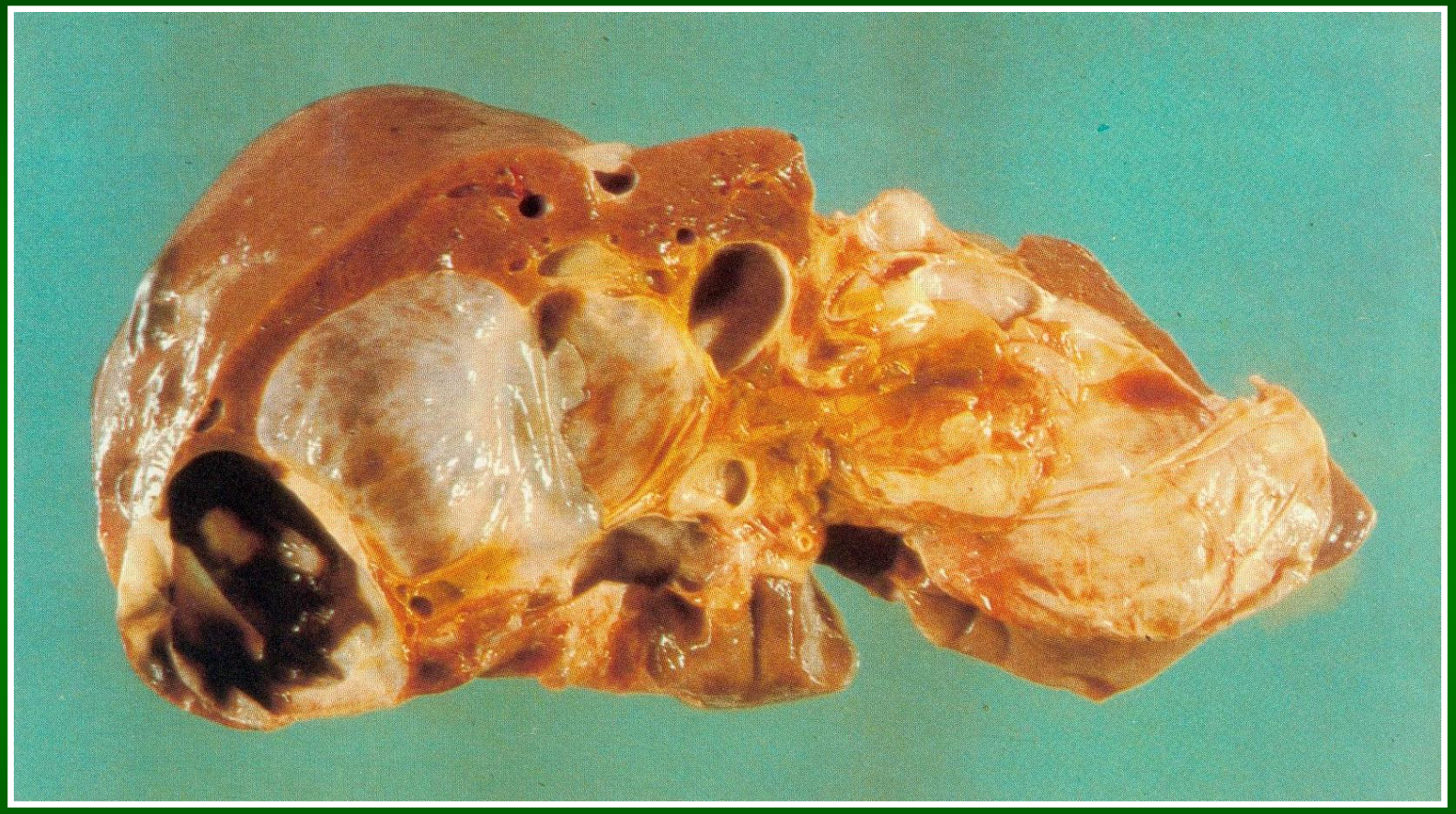
DISTURBANCES IN DEVELOPMENT

ADDITIONAL LOBE

DIAPHRAGMATIC SULCI

AGENESIA OF LEFT LOBE

LIVER PATHOLOGY



SIMPLE CYSTS OF LIVER

USUALLY SINGLE, RARELY NUMEROUS, USUALLY PARALLEL TO MULTICYSTIC KIDNEYS AND LUNGS

PATHOPHYSIOLOGY OF JAUNDICE (ICTERUS)

ICTERUS

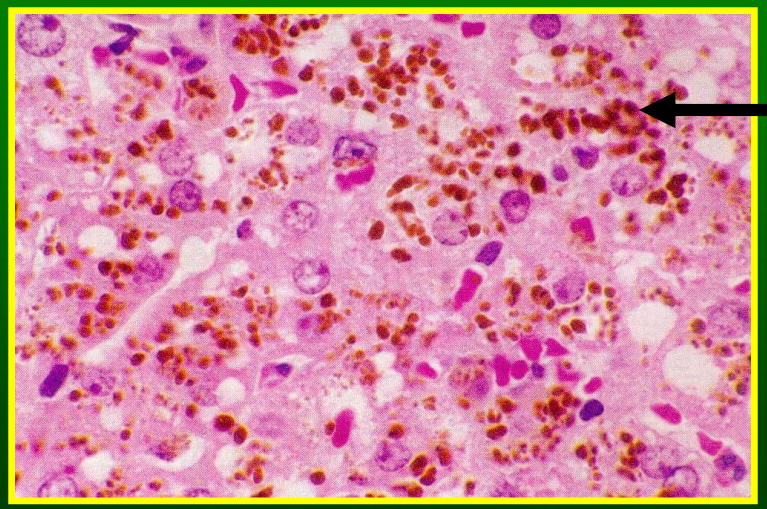
A. INCREASE IN PRODUCTION OF BILIRUBIN

B. DECREASE IN ACTIVITY OF GLUCURONYLTRANSFERASE IN HEPATOCYTES (GILBERT SYNDROME)

C. DISTURBANCES IN CONJUGATION OF BILIRUBIN WITH GLUCURONIC ACID

D. DISTURBANCES IN SECRETION OF BILIRUBIN TO BILE DUCTS (eg. DUBIN-JOHNSON SYNDROME); VARIANT – ROTOR SYNDROME

E. CRIGGLER-NAJJAR SYNDROME I AND II



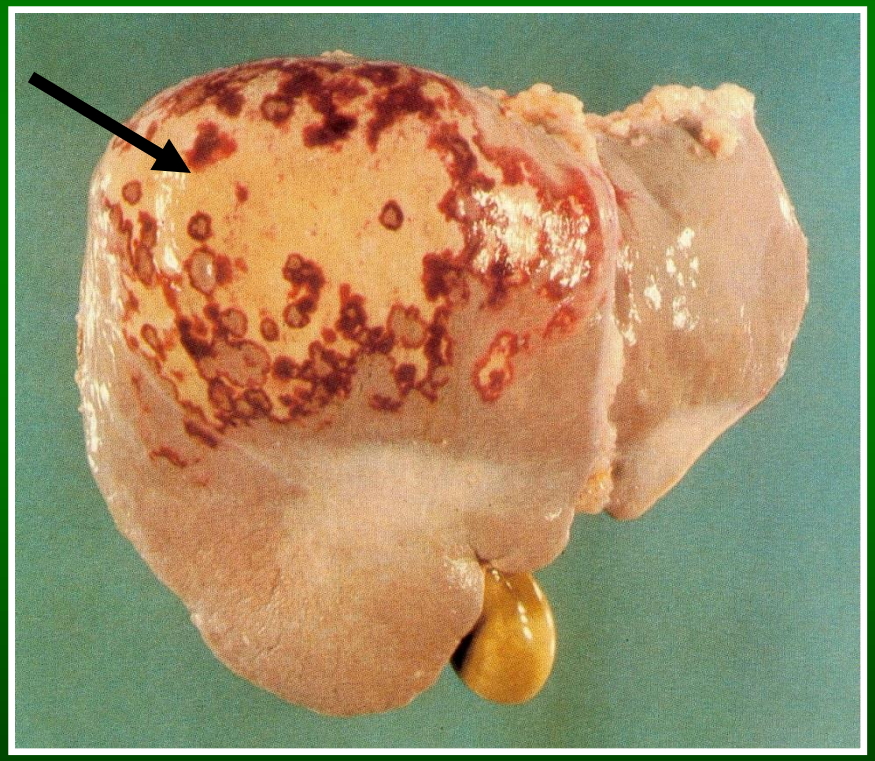
DUBIN-JOHNSON SYNDROME
bilirubin

DISTURBANCES IN CIRCULATION IN LIVER

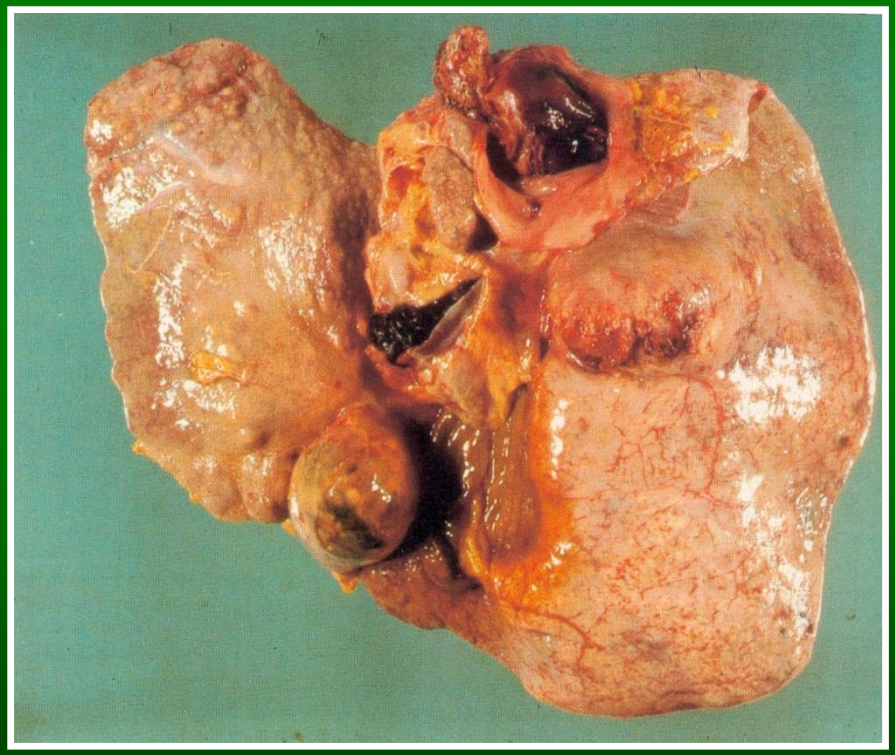
INFARCT

BUDD-CHIARI SYNDROME (THROMBOSIS OF HEPATIC VEINS)

THROMBOSIS OF PORTAL VEIN

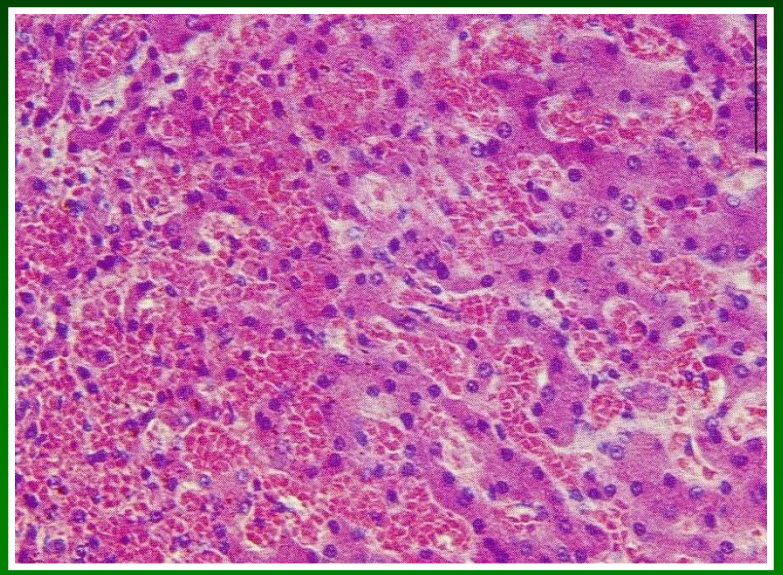


PALE INFARCT

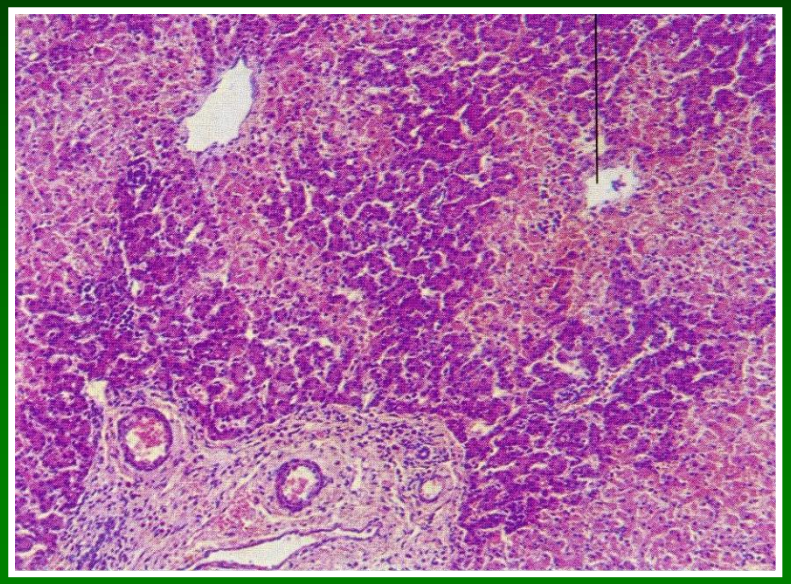


THROMBOSIS OF HEPATIC VEINS

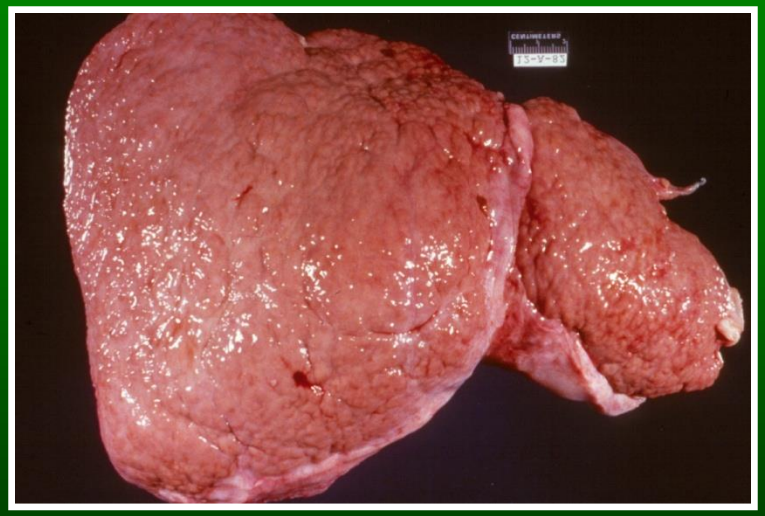
DISTURBANCES IN LIVER CIRCULATION



CHRONIC STASIS IN VEINS

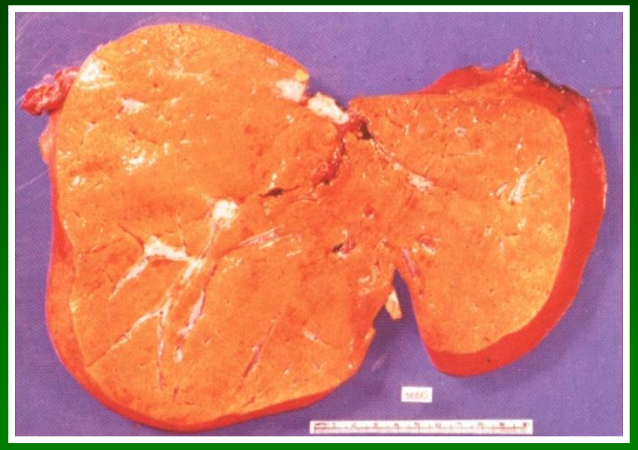


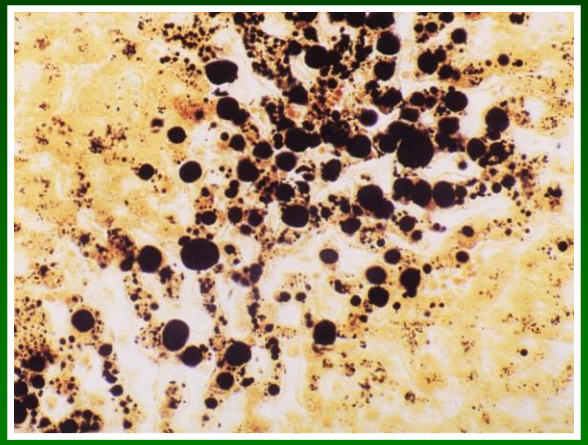
CENTRAL NECROSIS

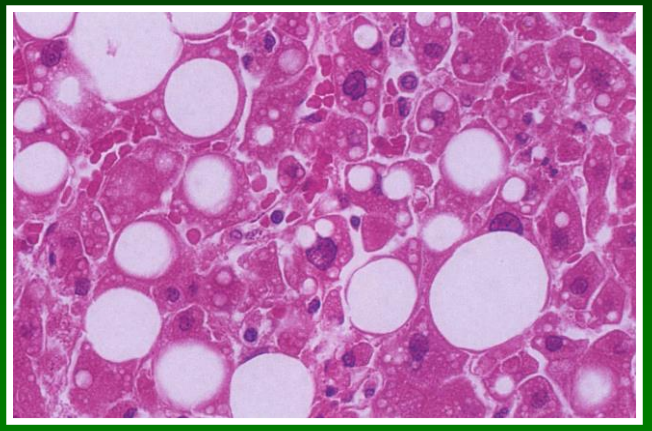


INDURATION

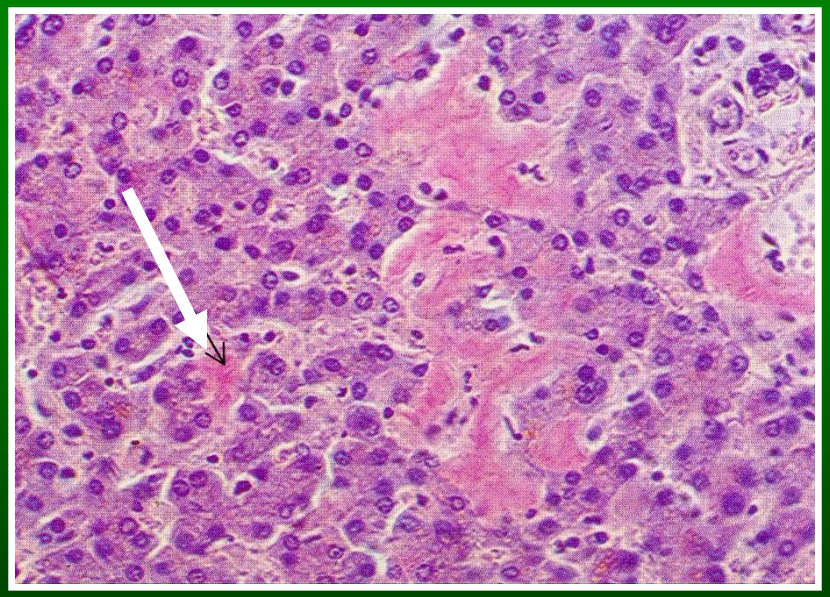
REGRESSIVE CHANGES IN LIVER

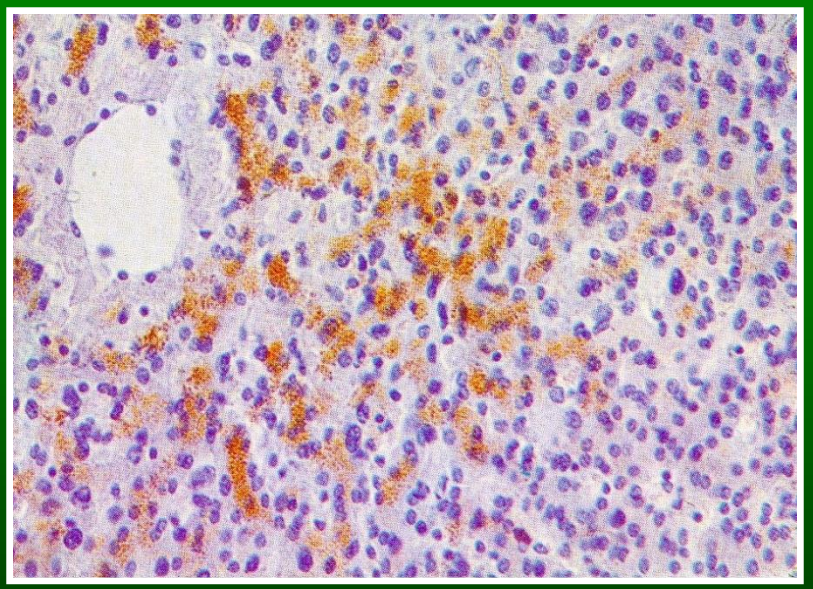






DIFFUSE ADIPOSE DEGENERATION – ANSERINE LIVER

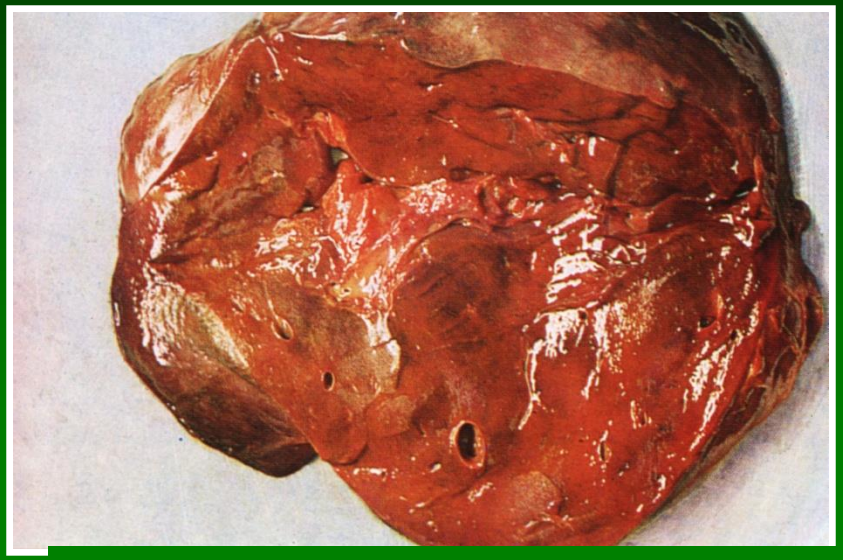




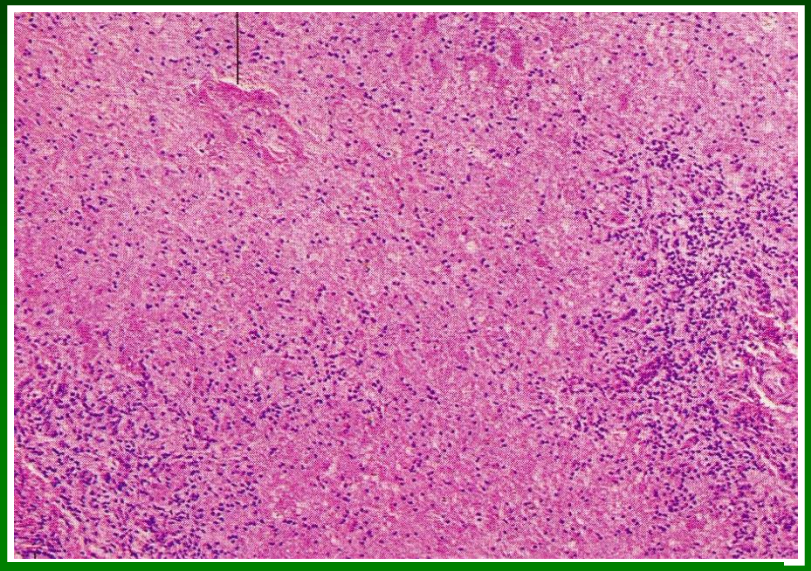
SECONDARY AMYLOIDOSIS

BROWN ATROPHY

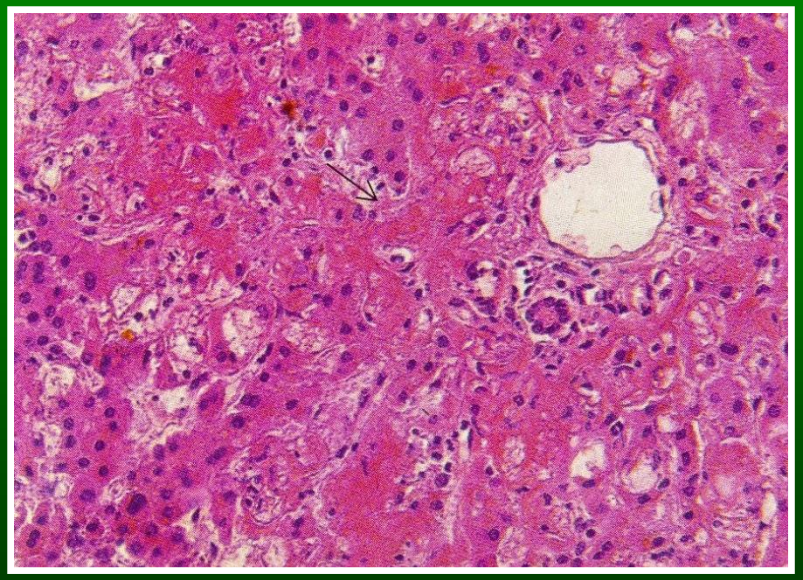
LIVER NECROSIS



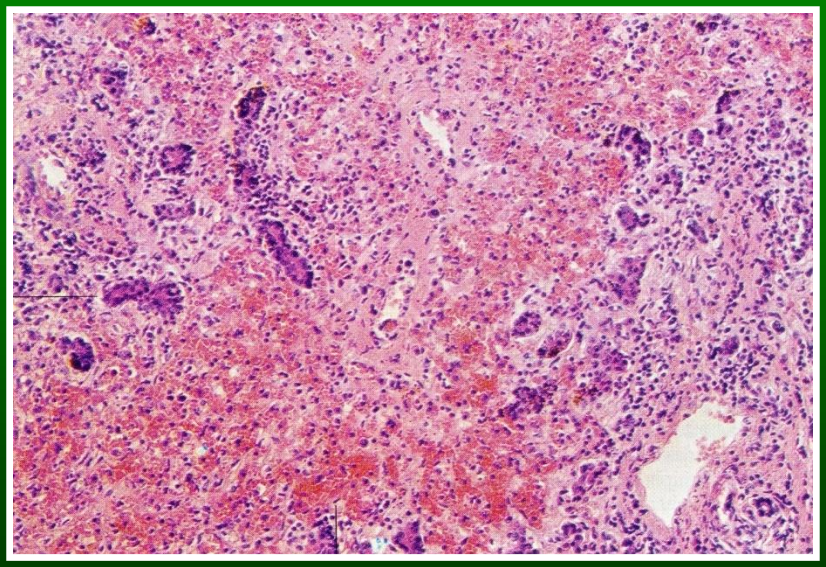
ACUTE YELLOW ATROPHY OF LIVER



ACUTE DIFFUSE MASSIVE NECROSIS



DISPERSE ACUTE NECROSIS



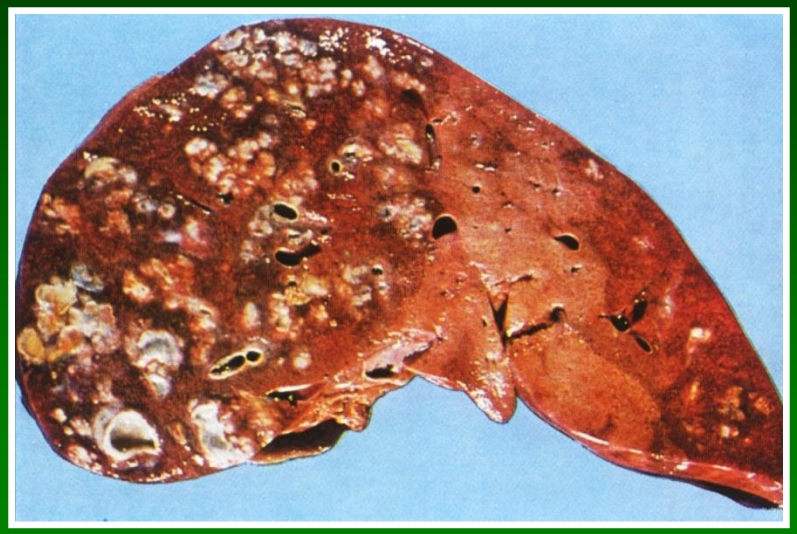
SUBACUTE MASSIVE NECROSIS

LIVER NECROSIS

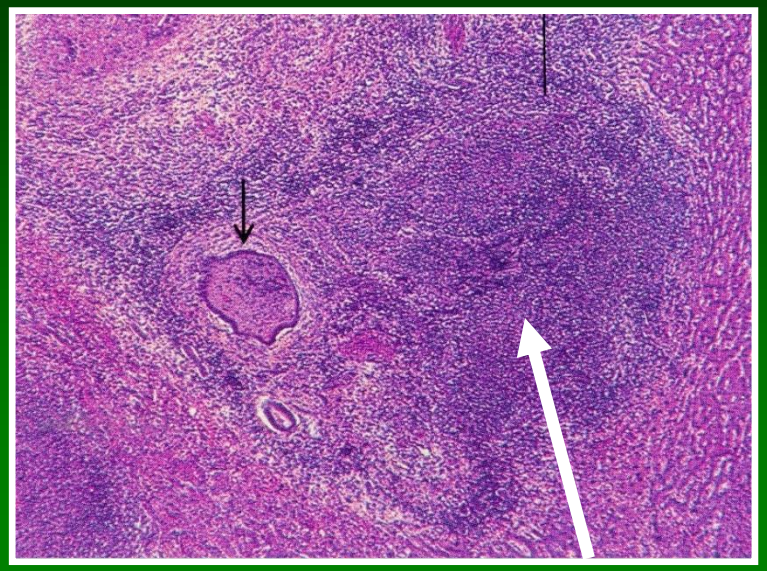
THE LIVER IS A GLAND WITH ENORMOUS REGENERATIVE ABILITY. A CONDITION FOR REGENERATION IS THE PRESERVATION OF THE FIBERS IN SUPPORTING TRABECULAE. NECROTIC HEPATOCYTES LYSE AND NEW ONES PROLIFERATE IN THEIR PLACE. THE STRUCTURE IS PRESERVED.

DIFFUSE NECROSIS WITH DESTRUCTION OF ELASTIC FIBERS CAUSES CHAOTIC REGROWTH, CONNECTED WITH FIBROSIS, AND RECONSTRUCTION OF THE LOBULAR STRUCTURE, AND THAT OF BLOOD VESSELS

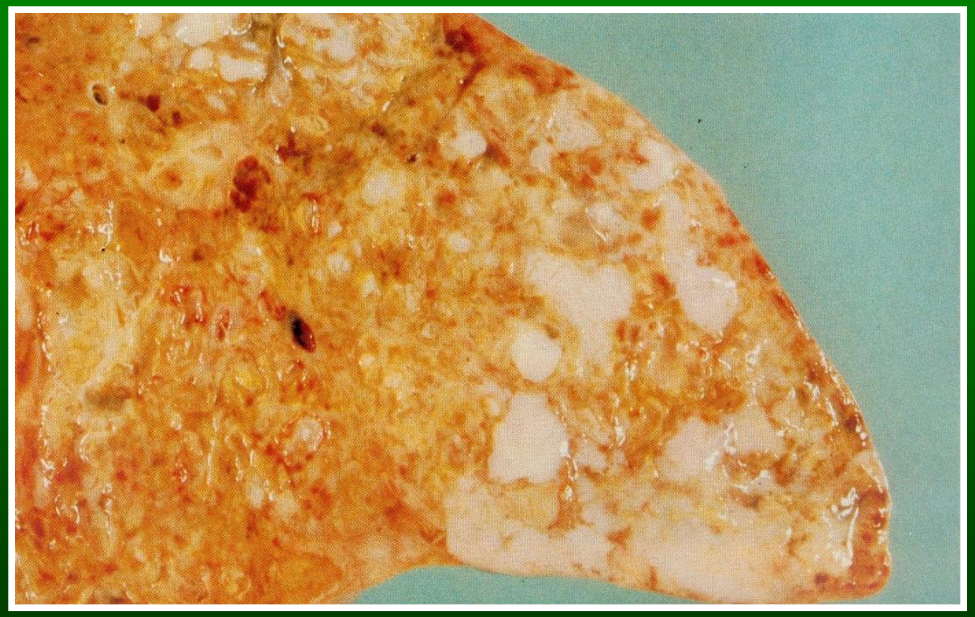
PURULENT INFLAMMATION OF LIVER



ABSCESSUS



SUPPURATIVE CHOLANGITIS



PURULENT INFLAMMATION OF LIVER DEVELOPS AS „METASTATIC" WITH BLOOD FROM THE LARGE VESSELS OR HEPATIC VEINS

VIRAL INFLAMMATIONS

HEPATITIS TYPE A – INCUBATION 14-45 DAYS

HEPATITIS TYPE B - INCUBATION 50-180 DAYS

HEPATITIS TYPE C – INCUBATION 5-10 WEEKS

MORPHOLOGICAL CHANGES IN EVERY TYPE OF HEPATITIS ARE SIMILAR. VIRUSES REPLICATE IN HEPATOCYTES. NECROSIS OF HEPATOCYTES IS THE CYTOTOXIC EXPRESSION OF VIRUS TYPE A OR THE ACTION OF ANTIBODIES OR SENSITIZED LYMPHOCYTES IN NON-CYTOTOXIC VIRUS TYPE B INFECTION.

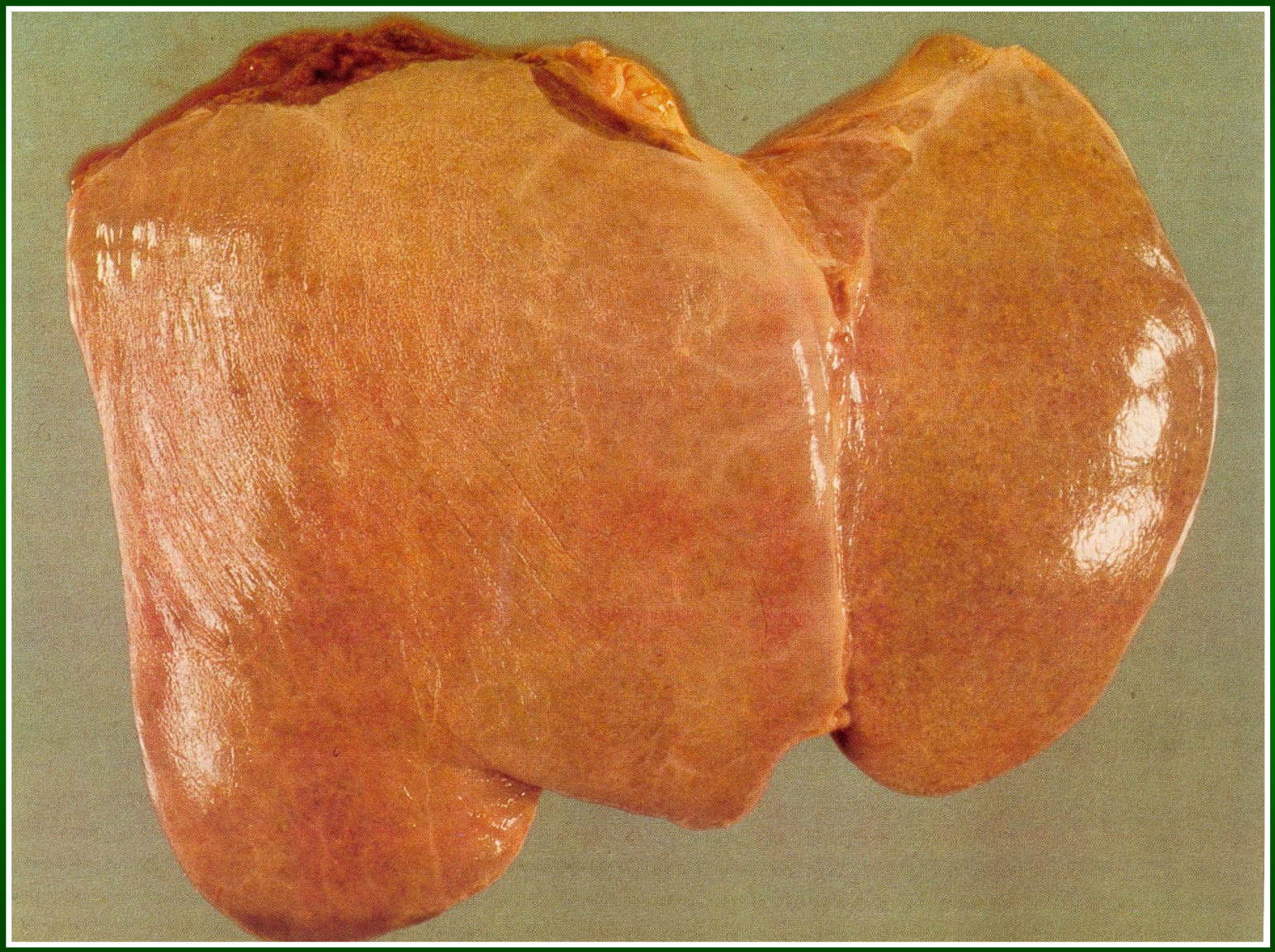
ACUTE PHASE WITH HEPATOCYTE NECROSIS – THE MORE DIFFUSE NECROSIS THE GREATER THE CLINICAL SYMPTOMS

DEATH OR RECOVERY (USUALLY)

CHRONIC PHASE

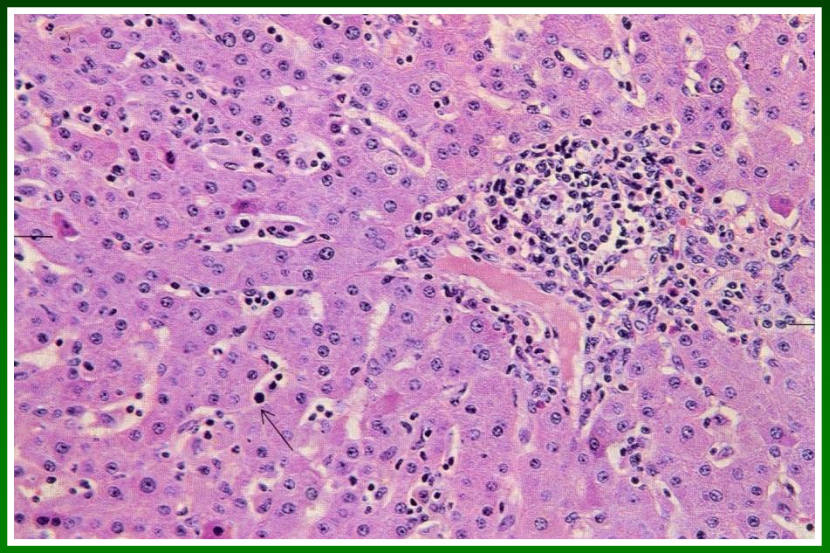
CHRONIC PERSISTENT HEPATITIS CHRONIC AGGRESSIVE HEPATITIS

VIRAL HEPATITIS

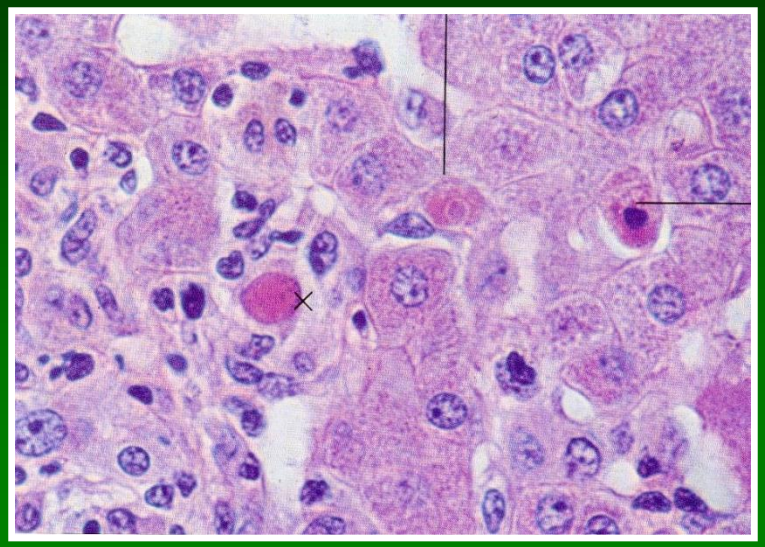


ACUTE HEPATITIS

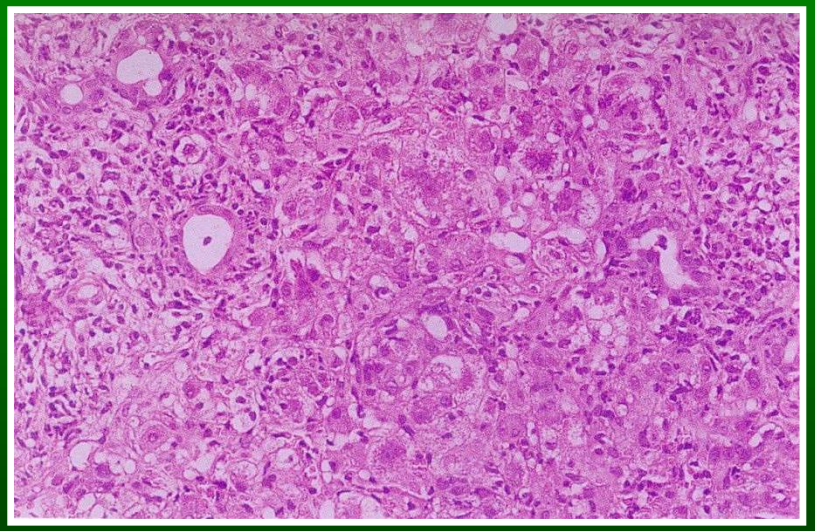
VIRAL HEPATITIS



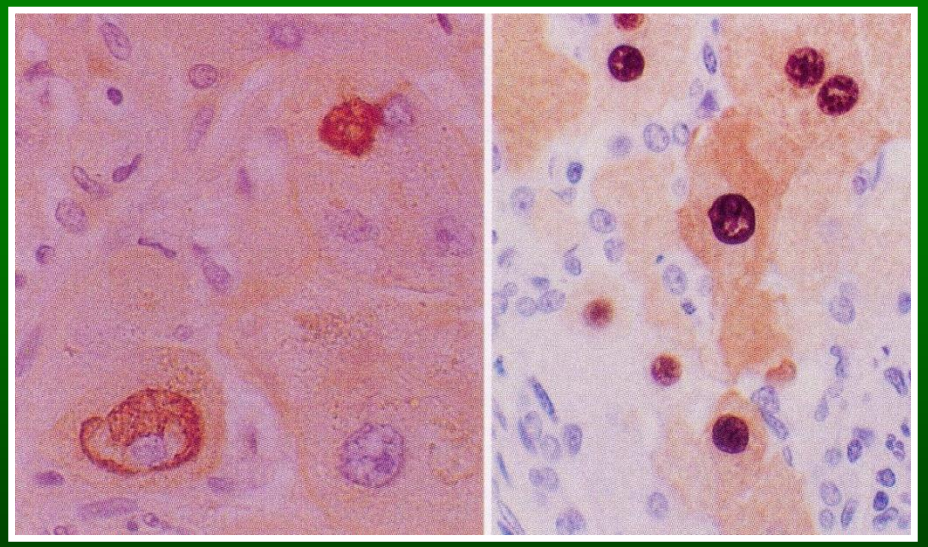
ACUTE HEPATITIS



ACUTE HEPATITIS

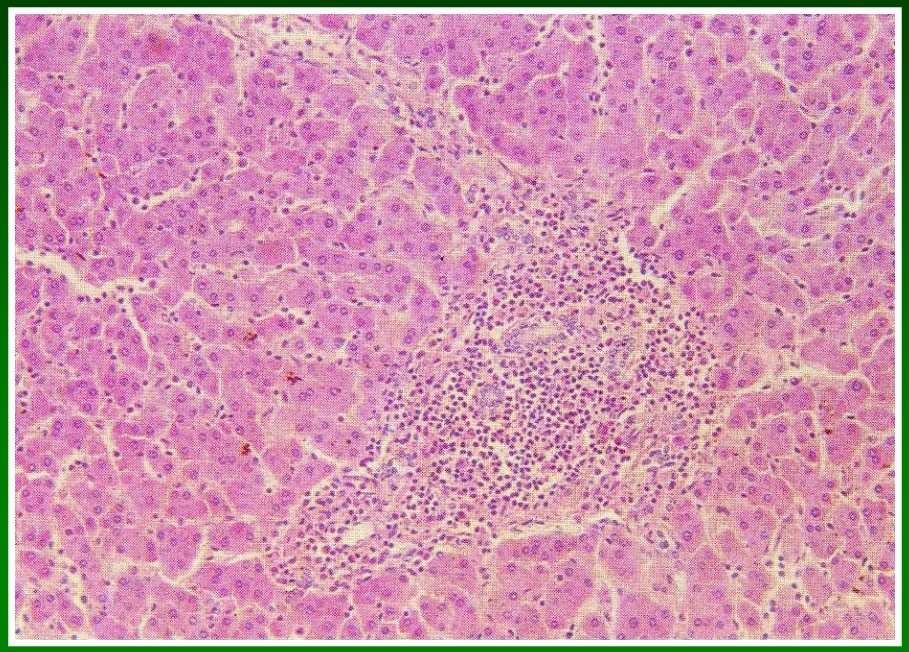


ACUTE HEPATITIS



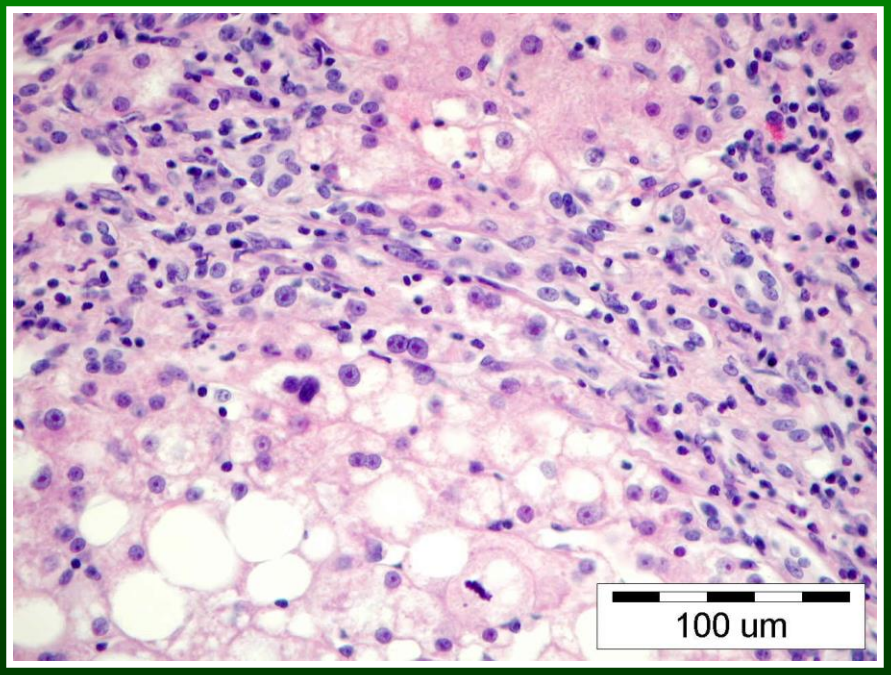
ANTIGEN HBs IN IMMUNOHISTOLOGICAL STAINING

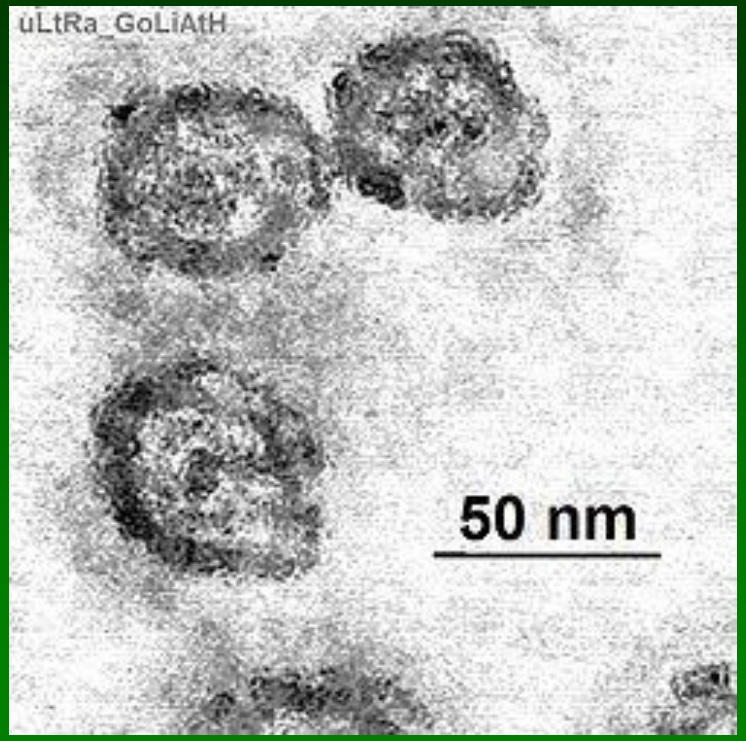
VIRAL HEPATITIS



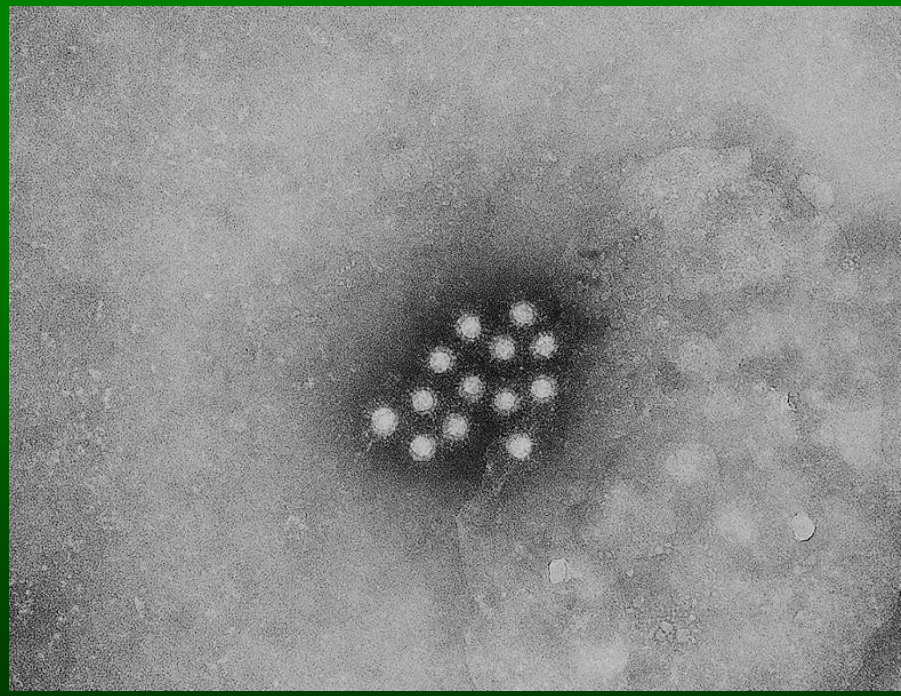
CHRONIC PERSISTENT HEPATITIS IN 80% OF CASES POSITIVE TEST FOR ANTIGEN HBs

CHRONIC AGGRESSIVE HEPATITIS



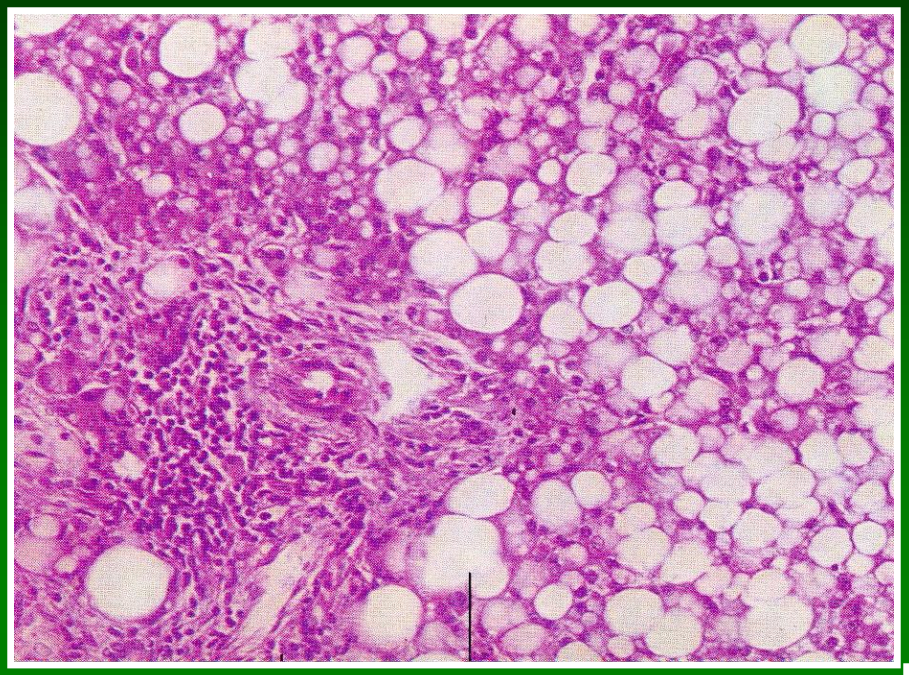


HEPATITIS VIRUS C



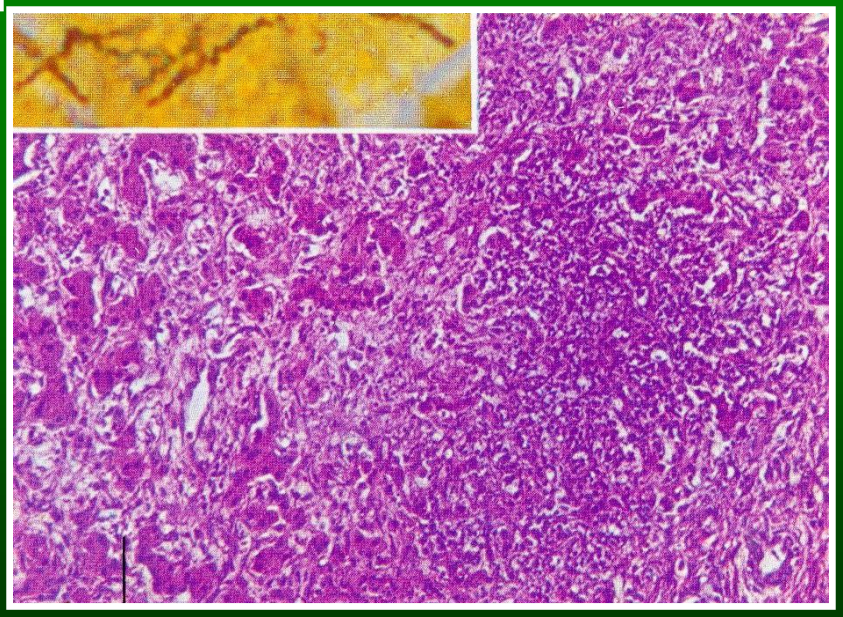
HEPATITIS VIRUS A

HEPATITIS

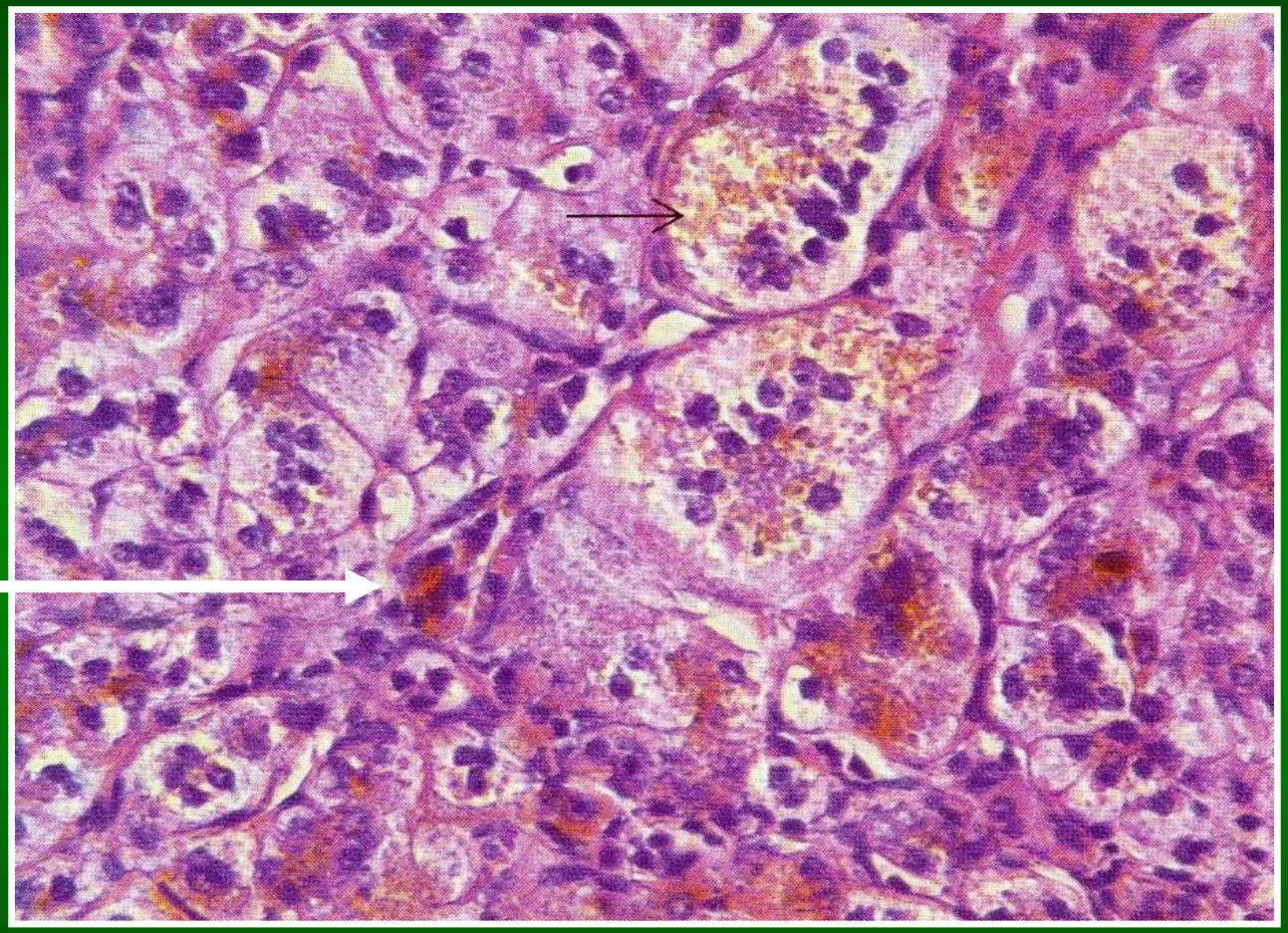


STEATOHEPATITIS
INFLAMMATION IN ALCOHOLICS WITH MALLORY BODIES

CONGENITAL INTERSTITIAL LUETIC HEPATITIS

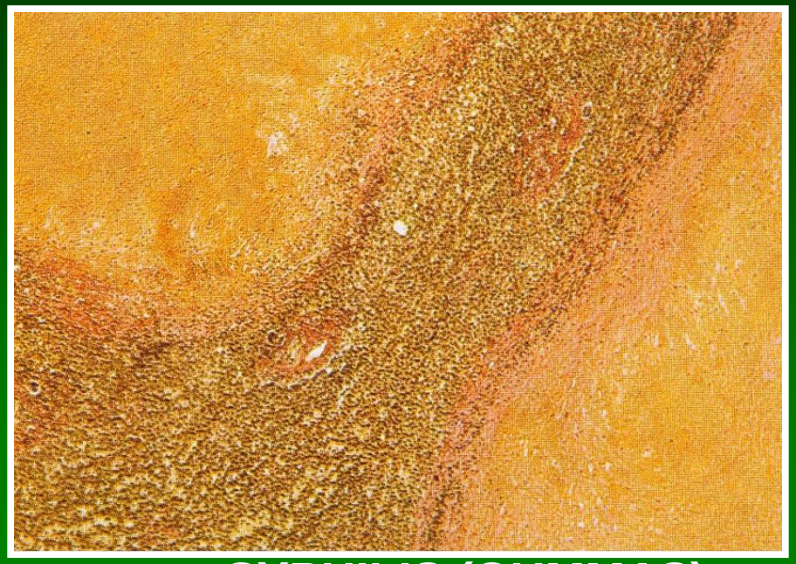


GIANT CELL INFLAMMATION IN LIVER

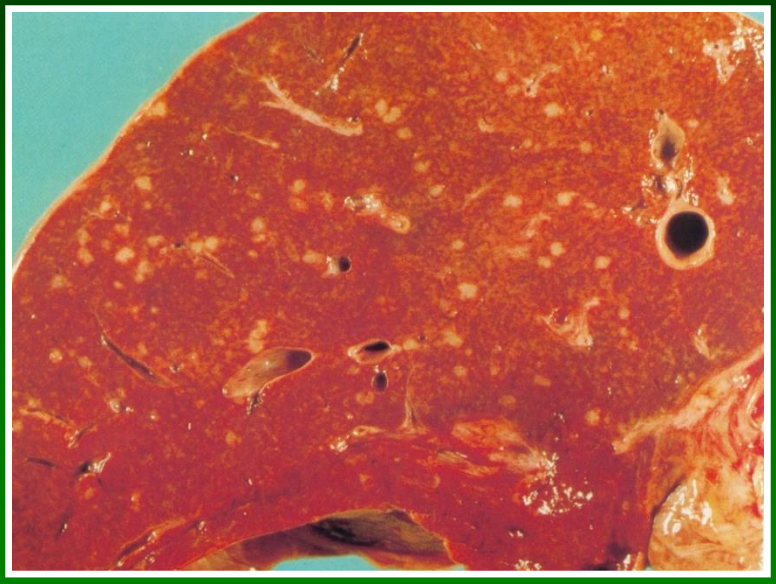


NEONATAL DISEASE (IT SHOWS SPECIFIC REACTIONS OF LIVER TO DIFFERENT PATHOGENS, ESPECIALLY VIRUSES). CLINICALLY OBSERVED OBSTRUCTIVE JAUNDICE. PATHOGNOMONIC IN HISTOLOGY ARE NUMEROUS GIANT CELLS WITH BILE PIGMENT. SOMETIMES FAMILIAL, LEADS TO CIRRHOSIS OF ORGAN AND DEATH IN MOST CASES.

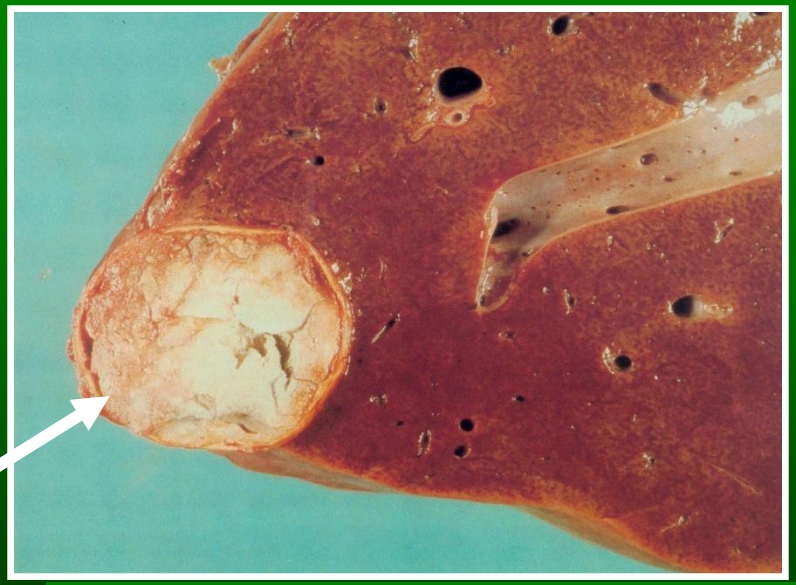
HEPATITIS



SYPHILIS (GUMMAS)



MILIARY TBC



ECHINOCOCCUS OF LIVER

AMEBIC ABSCESS

LIVER CIRRHOSIS

RECONSTRUCTION OF ORGAN WITH FORMATION OF AXIAL TUMORS (WITH CENTRAL VEIN AND BILE DUCT) AND NONAXIAL, AS WELL AS DISRUPTION OF BLOOD CIRCULATION THROUGH THE LIVER.

CIRRHOSIS RELATED TO ALCOHOL CONSUMPTION (LAENNEC, PORTAL, ALCOHOLIC, NUTRITIONAL, MICRONODULAR) - 30 – 60%

PIGMENTARY CIRRHOSIS:

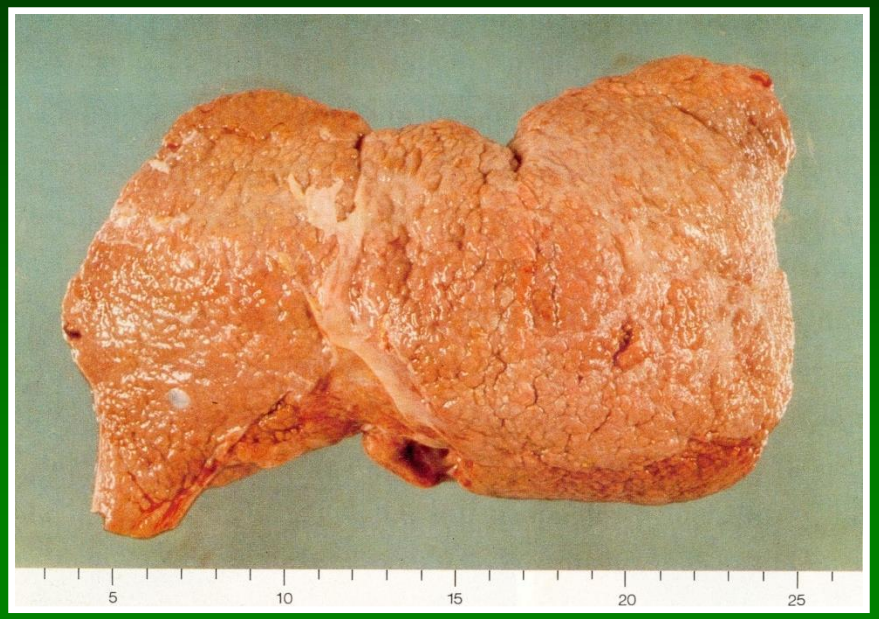
- **ACCOMPANIED BY HEMOCHROMATOSIS - 2%**
- **ACCOMPANIED BY WILSONS' DISEASE - VERY RARE**

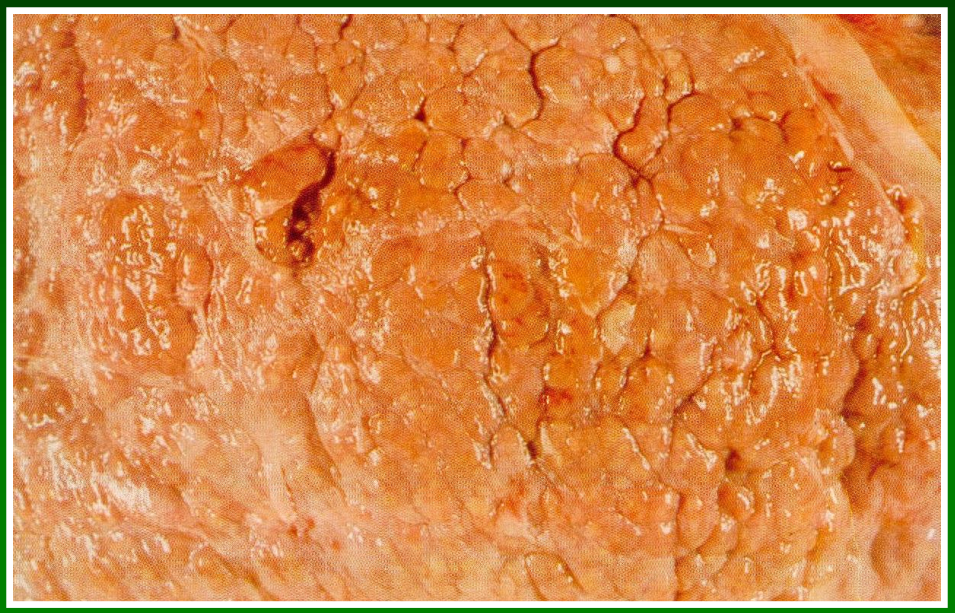
POST-NECROTIC CIRRHOSIS (MACRONODULAR) – 10-30%

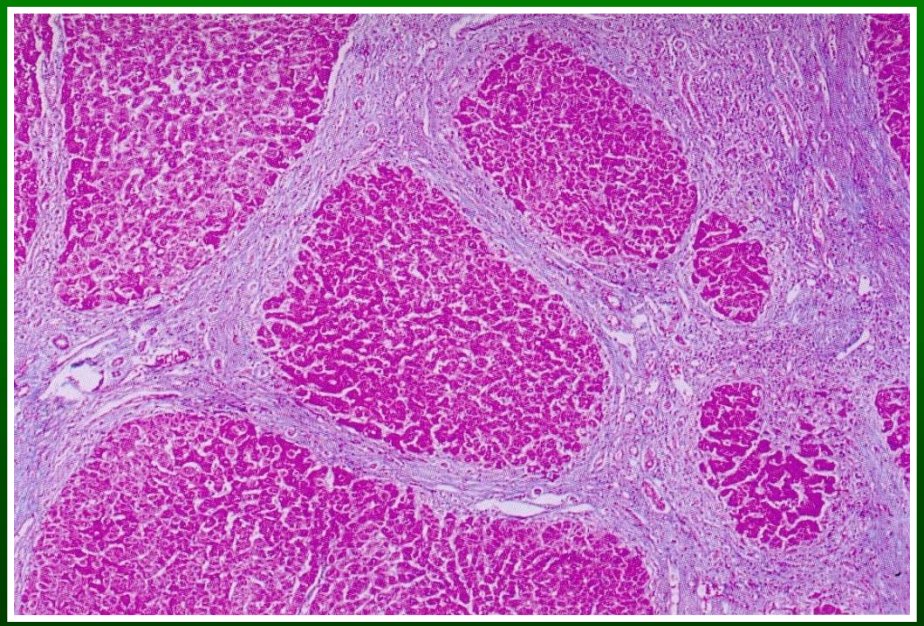
BILIARY CIRRHOSIS (PRIMARY AND PORTAL) - 10– 20%

OTHER FORMS 15-25%

LIVER CIRRHOSIS



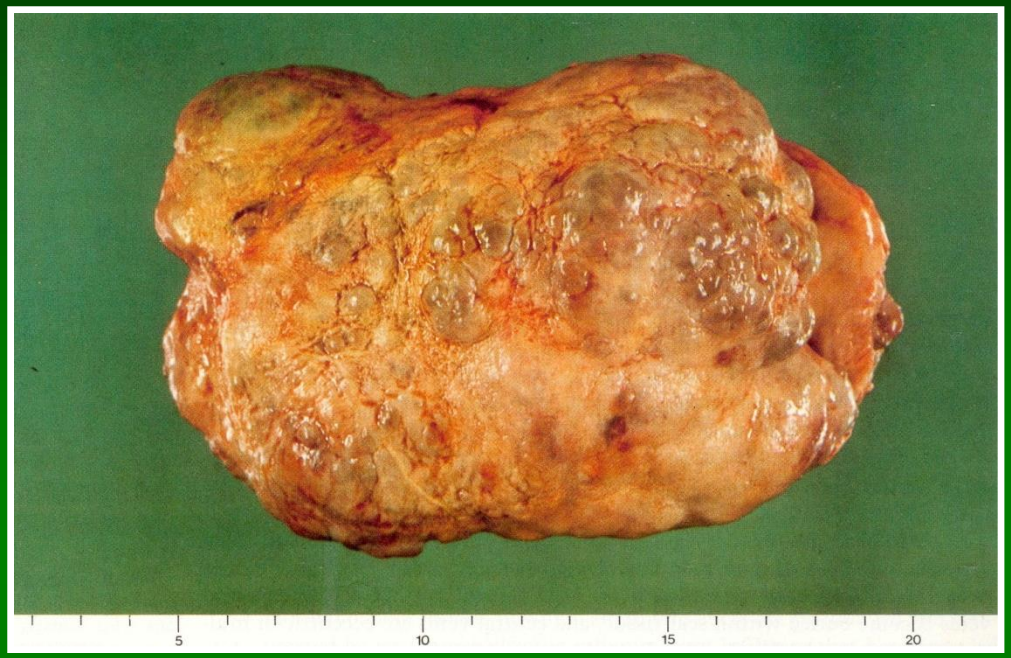


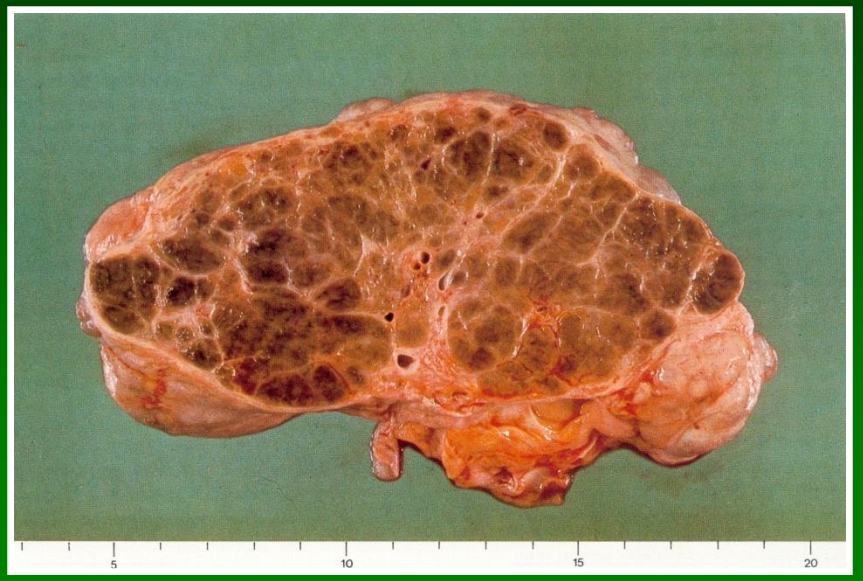


MICRONODULAR (ALCOHOLIC)

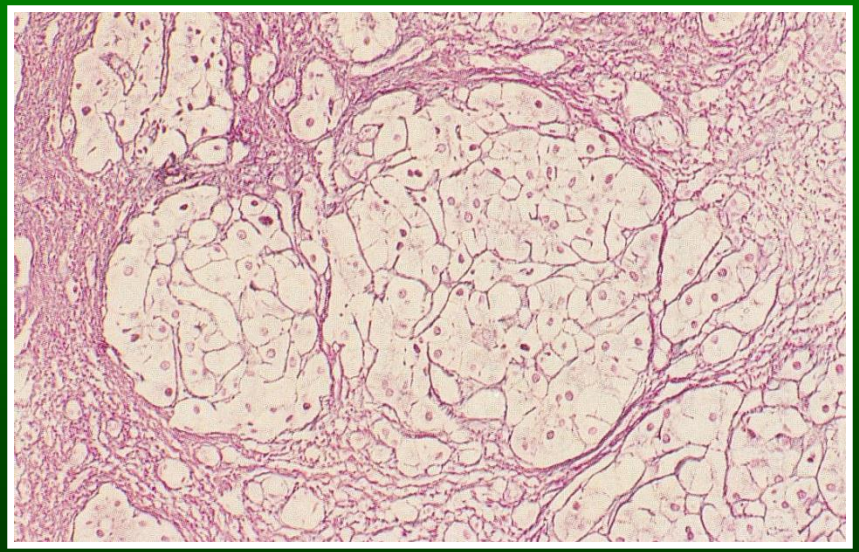
CIRRHOSIS

LIVER CIRRHOSIS





MACRONODULAR (POSTNECROTIC) CIRRHOSIS

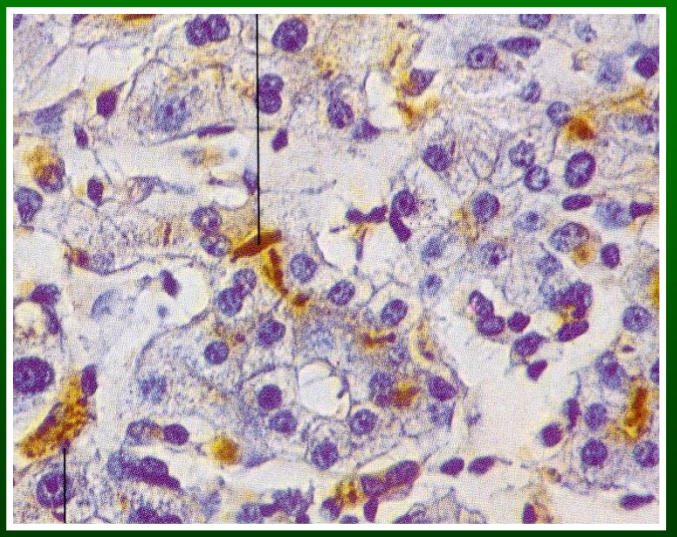


REGENERATING NON-AXIAL TUMORS ARE RESULT OF THE DESTRUCTION OF STRUCTURAL FIBERS

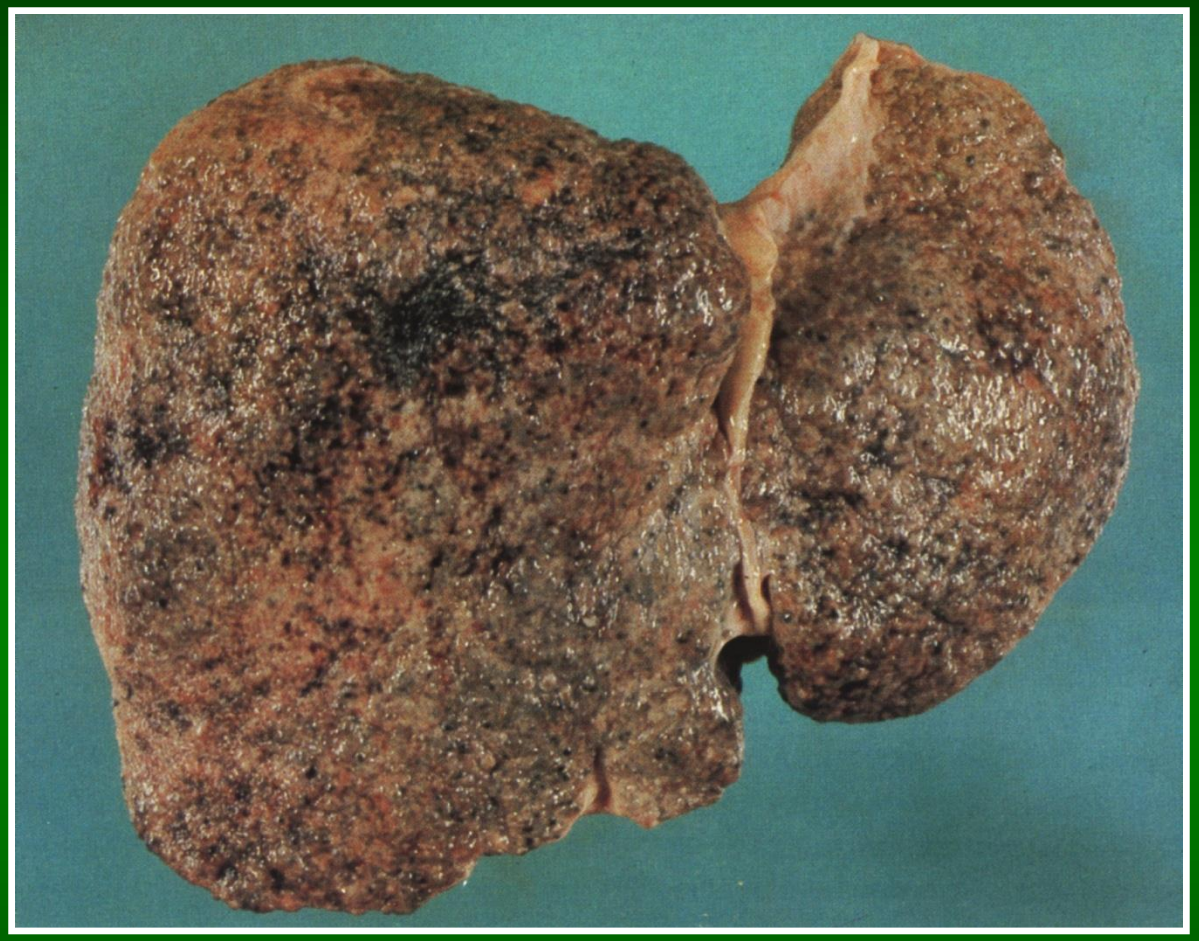
LIVER CIRRHOSIS

BILIARY
CIRRHOSIS

CHOLESTEROL IN LIVER

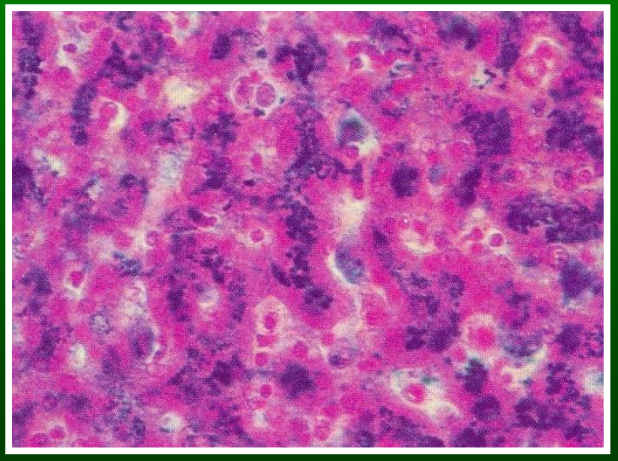


LIVER CIRRHOSIS



PIGMENTARY CIRRHOSIS

HEMOCHROMATOSIS



LIVER HEMOCHROMATOSIS (IRON DEPOSITS)

• HEMOCHROMATOSIS

- **HISTORICALLY, THE TERM HEMOCHROMATOSIS**
- **WAS USED TO REFER TO WHAT IS NOW MORE**
- **SPECIFICALLY CALLED HEMOCHROMATOSIS TYPE 1.**
- **CURRENTLY, HEMOCHROMATOSIS (WITHOUT**
- **FURTHER SPECIFICATION) IS MOSTLY DEFINED AS**
- **IRON OVERLOAD WITH A HEREDITARY/PRIMARY**
- **CAUSE OR ORIGINATING FROM A METABOLIC**
- **DISORDER. HOWEVER, THE TERM IS CURRENTLY**
- **ALSO USED MORE BROADLY TO REFER TO ANY**
- **FORM OF IRON OVERLOAD THUS REQUIRING**
- **SPECIFICATION OF THE CAUSE**
  FOR EXAMPLE: *hereditary hemochromatosis*.

HEMOCHROMATOSIS

CLINICAL PRESENTATION

- **Organs commonly affected by hemochromatosis are LIVER, HEART and ENDOCRINE GLANDS**

CAUSES

- **The causes can be distinguished between primary (hereditary or genetically determined) and less frequent secondary cases (acquired during life). People of CELTIC (IRELAND, SCOTLAND) origin have a particularly high incidence of whom about 10% are carriers of the gene and 1% sufferers from the condition.**

PROGNOSIS

- **A third of those untreated develop HEPATOCELLULAR CARCINOMA**

COMPLICATIONS OF LIVER CIRRHOSIS

COLLATERAL CIRCULATION – VARICES OF ESOPHAGUS, ANUS, MEDUSA HEAD AND ITS COMPLICATIONS: HEMORRHAGES

HYPERSPLENISM

COAGULATION DISTURBANCES (LACK OF PROTHROMBIN AND OTHER BLOOD CLOTTING FACTORS, PLATELETS)

PORTAL HYPERTENSION

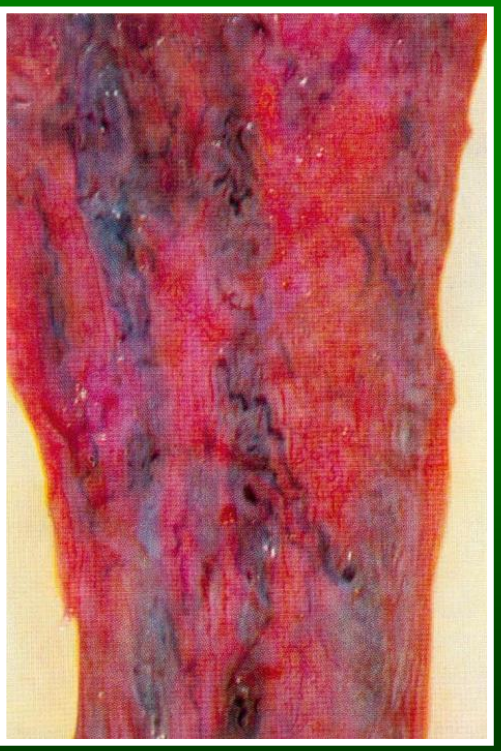
EDEMAS AND TRANSUDATES

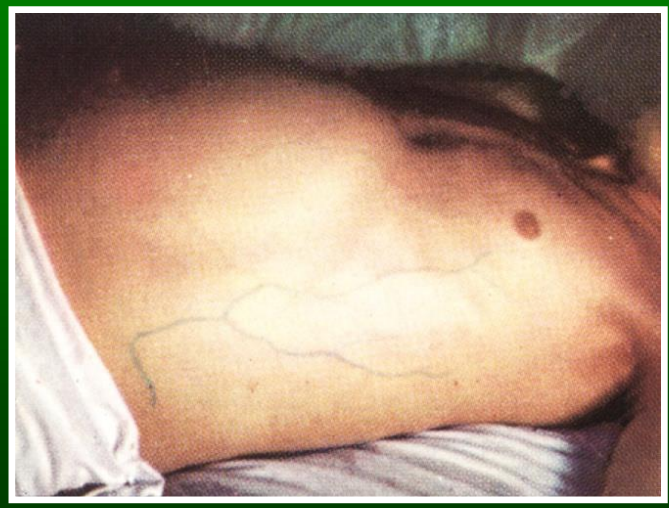
RELATIVE HYPERALDOSTERONISM

HYPERESTROGENISM

PRIMARY CANCER

COMA

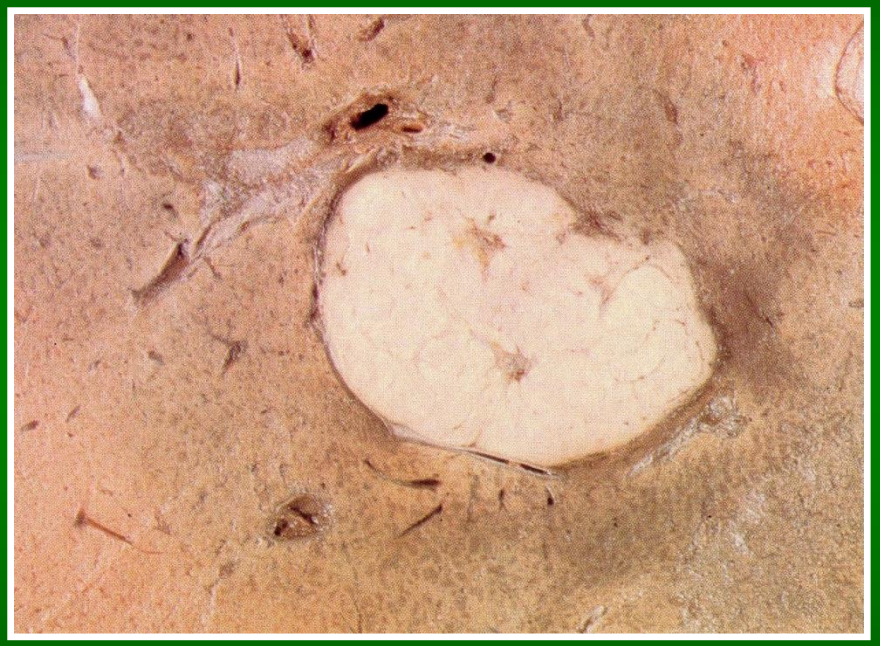




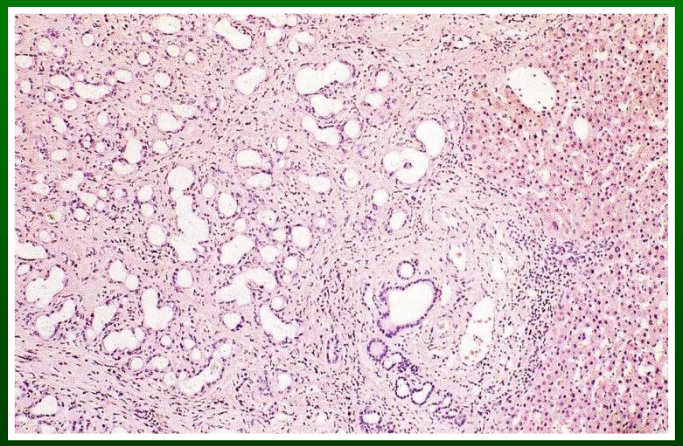
TUMORS OF THE LIVER

CAVERNOUS HEMANGIOMA

MOST COMMON
NON-EPITHELIAL
HEPATIC TUMOR

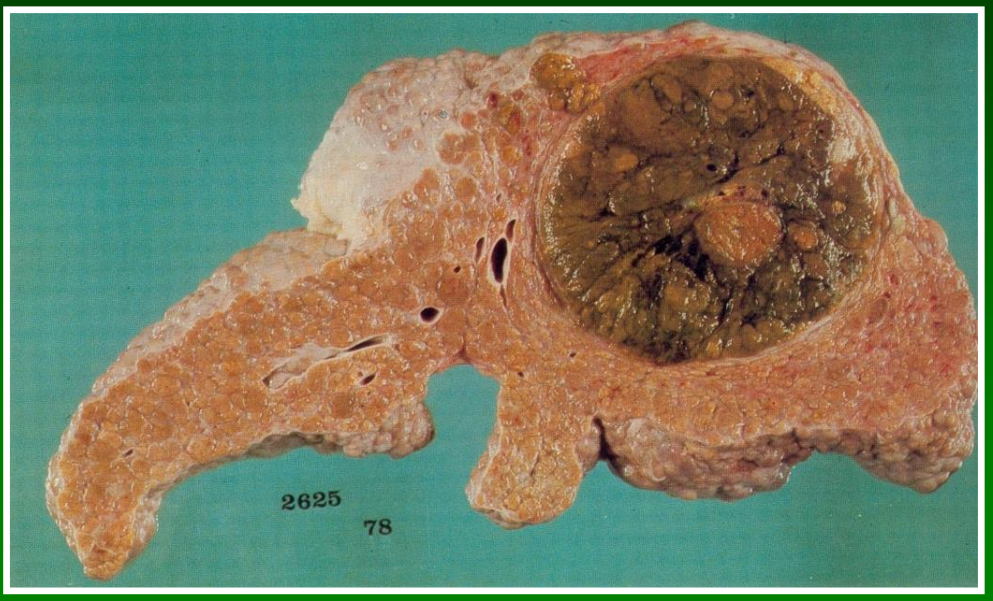


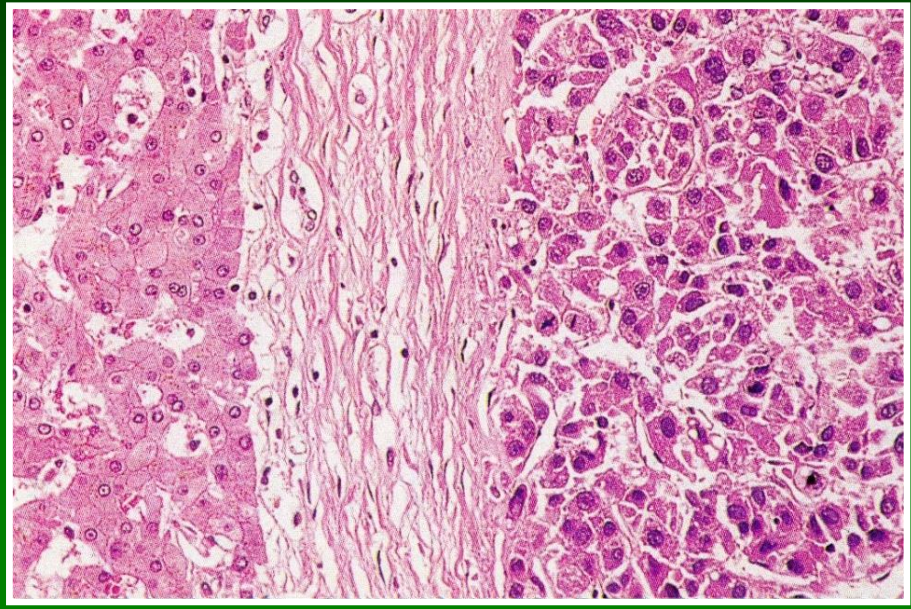
HEPATOCELLULAR ADENOMA

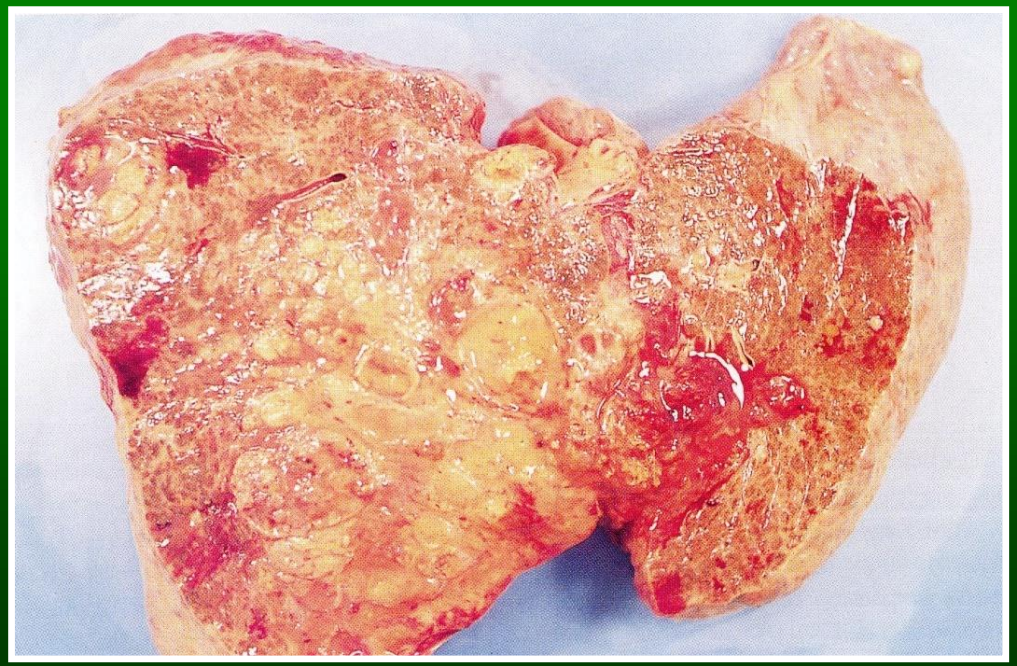


CHOLANGIOMA

HEPATIC TUMORS



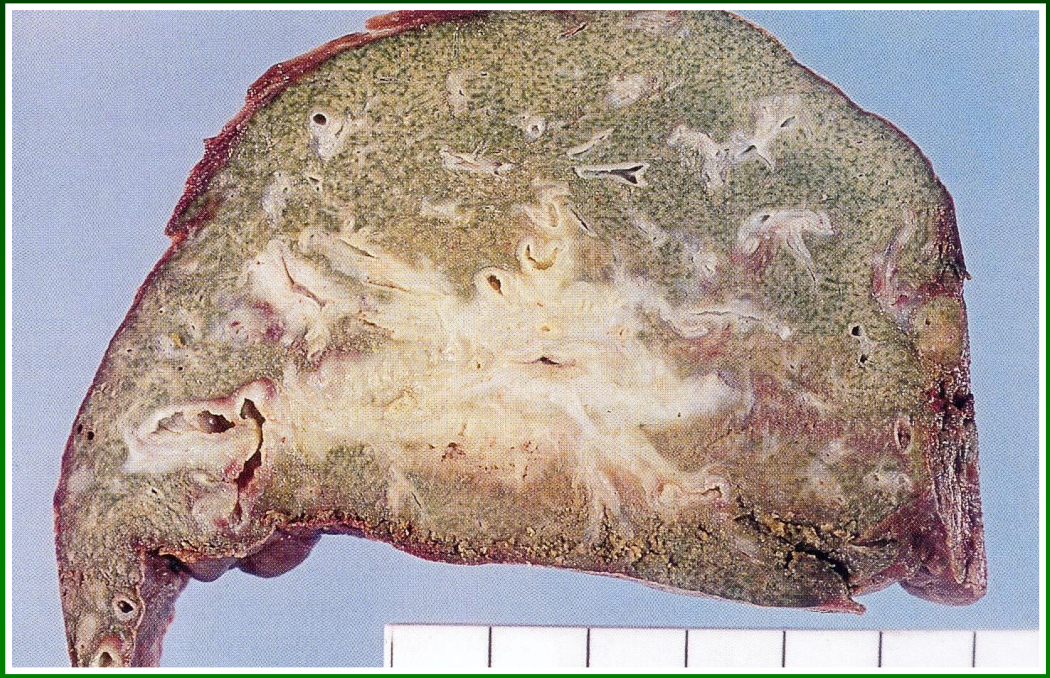


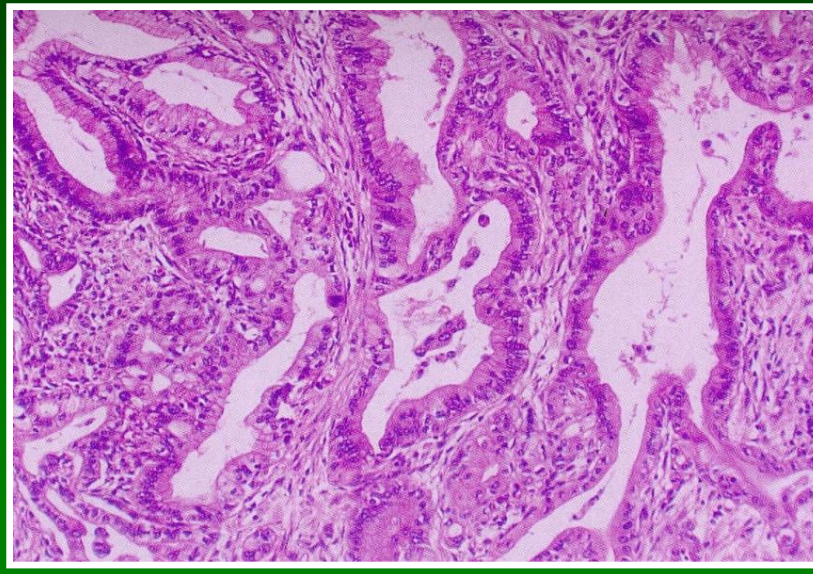


HEPATOCARCINOMA

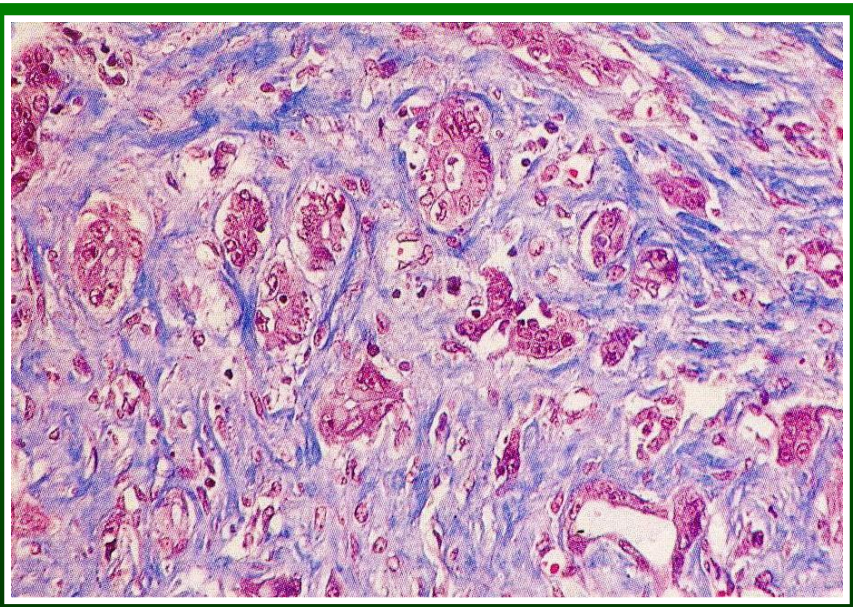
TUMOR USUALLY DEVELOPS IN A CIRRHOTIC LIVER
IMPORTANT: NUMEROUS PRIMARY FOCI

TUMORS OF LIVER



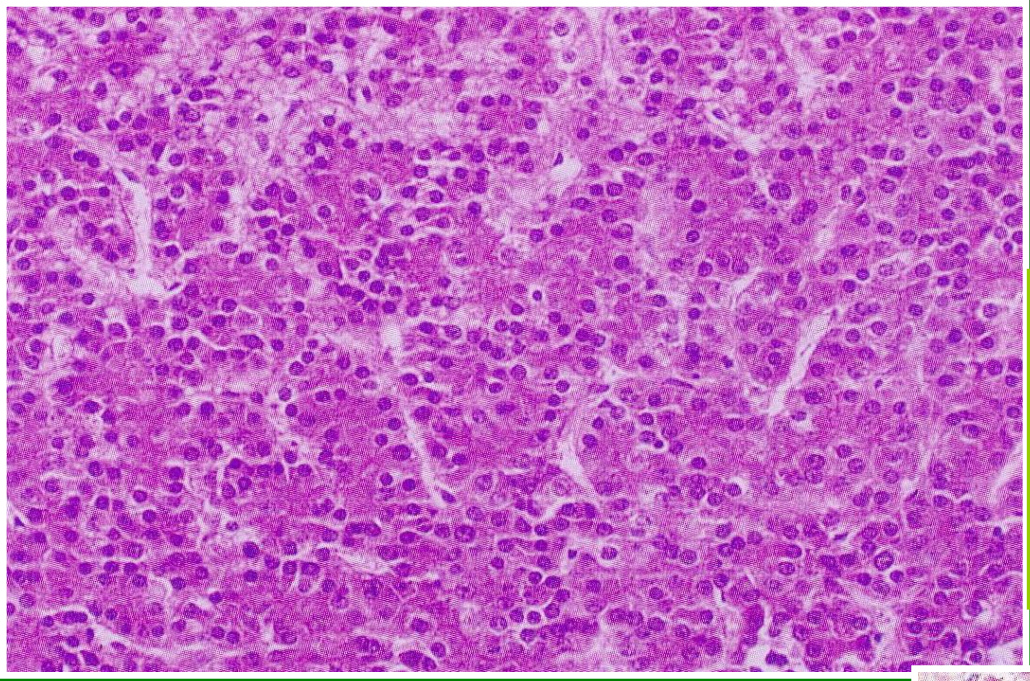


CHOLANGIOCARCINOMA



ADENOCARCINOMA, MUCUS-PRODUCING CELLS (PINK)

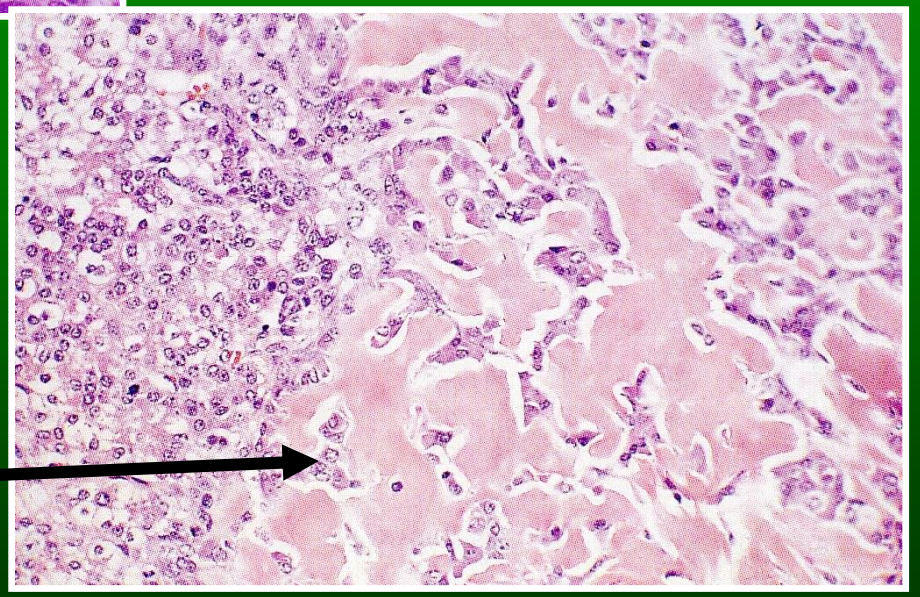
TUMORS OF LIVER



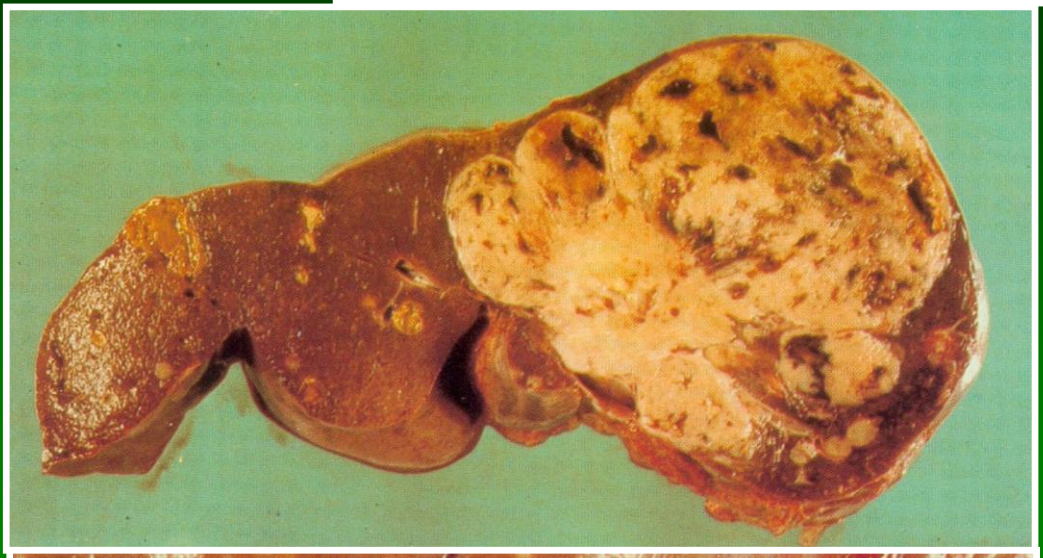
HEPATOBLASTOMA

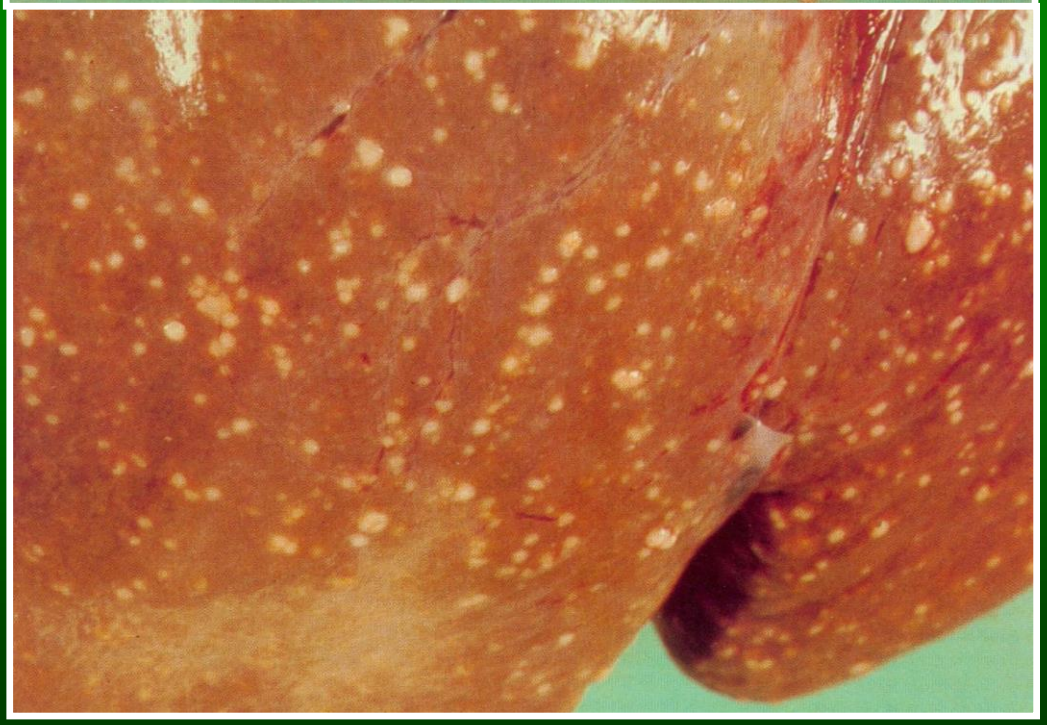
ONE OF THE MOST COMMON TUMORS IN CHILDREN BELOW THE 2ND YEAR OF LIFE WITH OTHER DEVELOPMENTAL ANOMALIES. MANY ANOMALIES IN CHROMOSOME 2 AND 20.

MESODERMIC STRUCTURES COMMONLY SEEN IN HEPATOBLASTOMA – EXAMPLE: OSTEOID TISSUE



MALIGNANT TUMORS OF LIVER



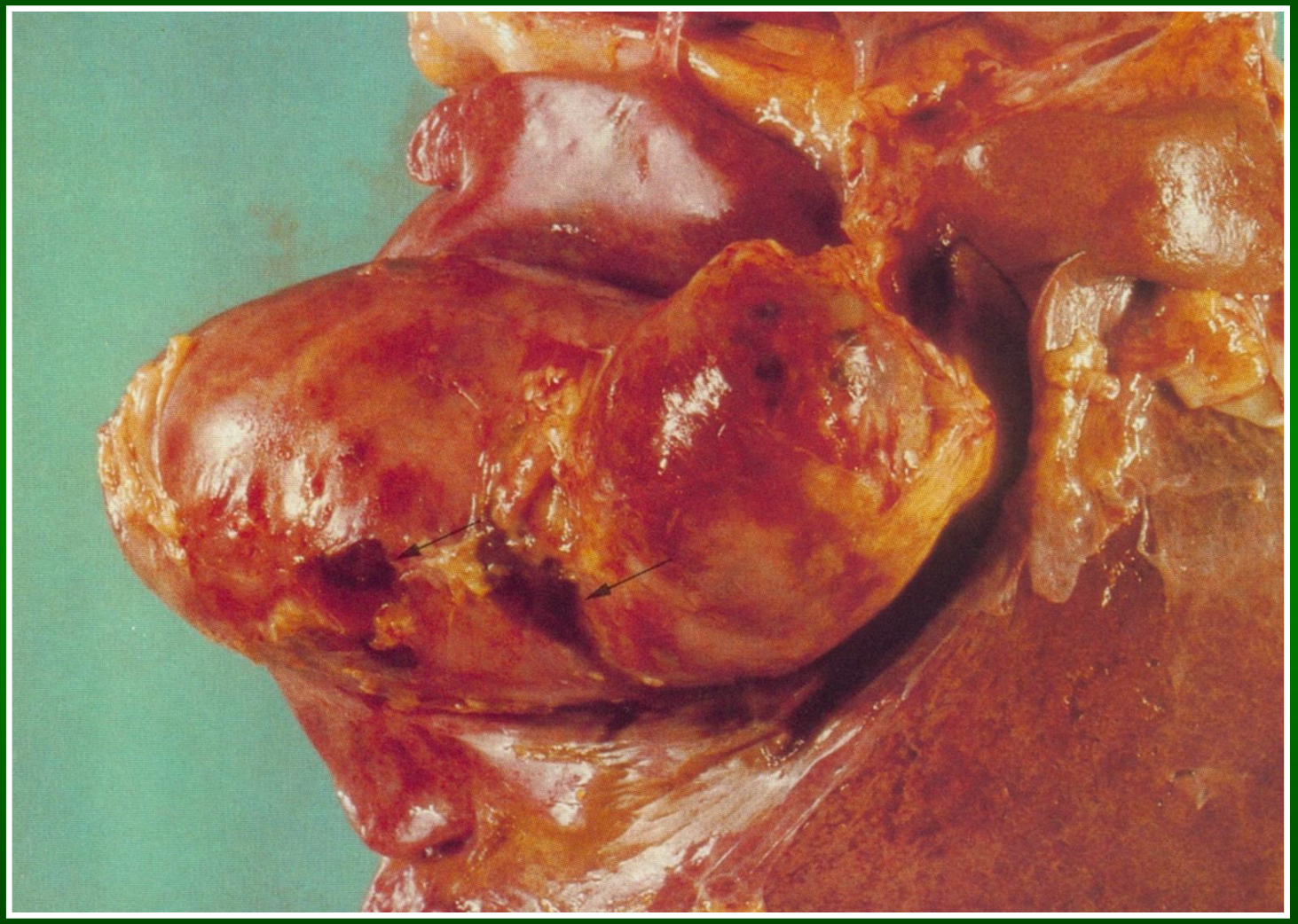


REMEMBER !!

PRIMARY CANCER OF LIVER IS VERY RARE CASE. MOST COMMON ARE METASTASES TO THE LIVER.

PATHOLOGY OF BILE DUCTS

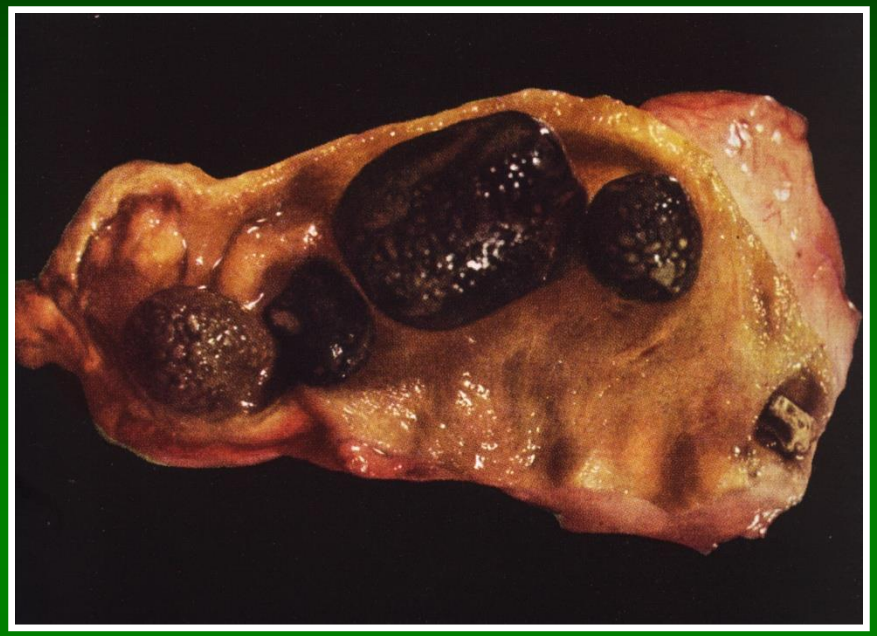
CHOLECYSTITIS

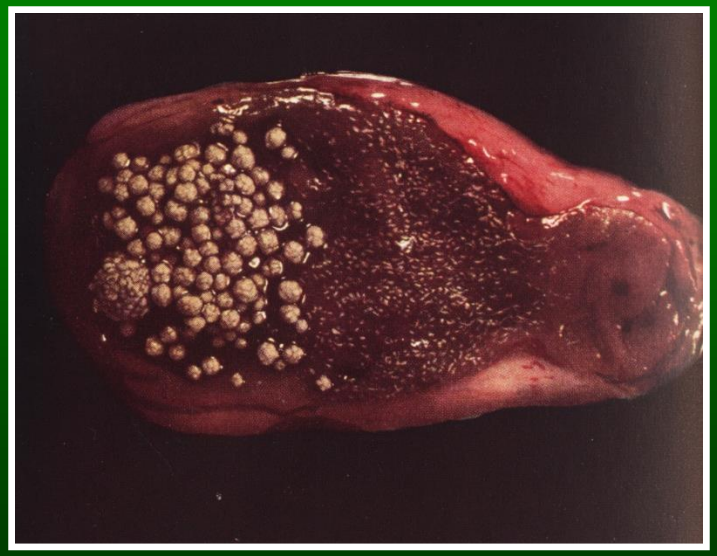


ACUTE, GANGRENOUS INFLAMMATION OF THE GALL BLADDER -
CHOLECYSTITIS ACUTA GANGRAENOSA

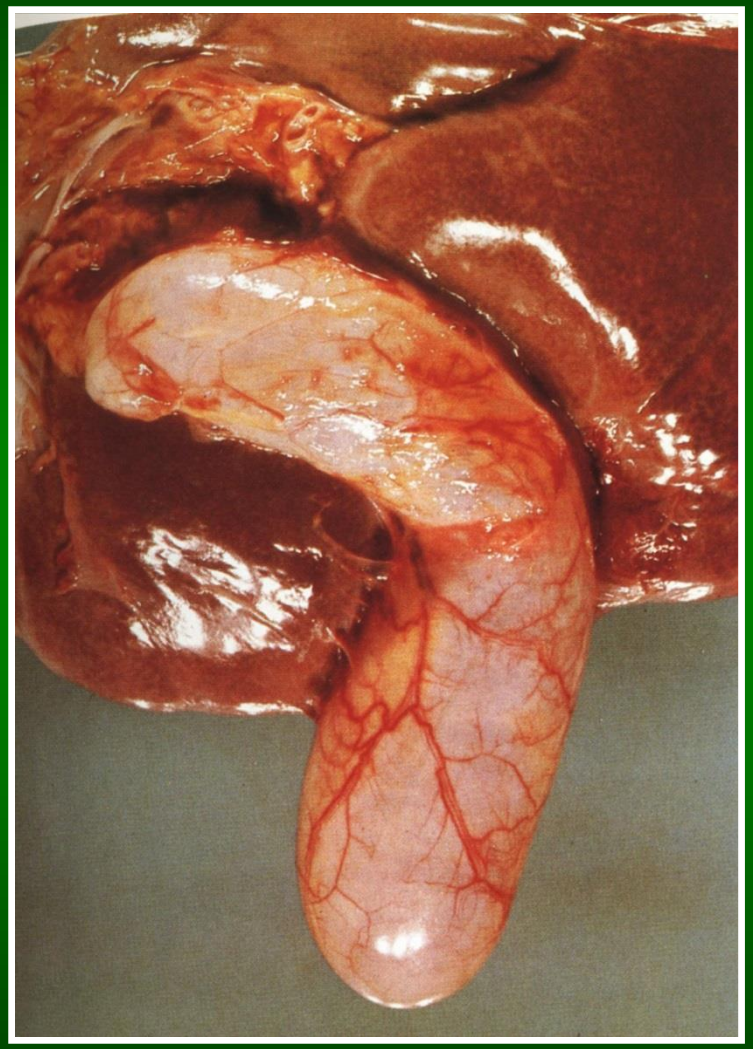
PATHOLOGY OF BILE DUCTS

CHOLELITHIASIS





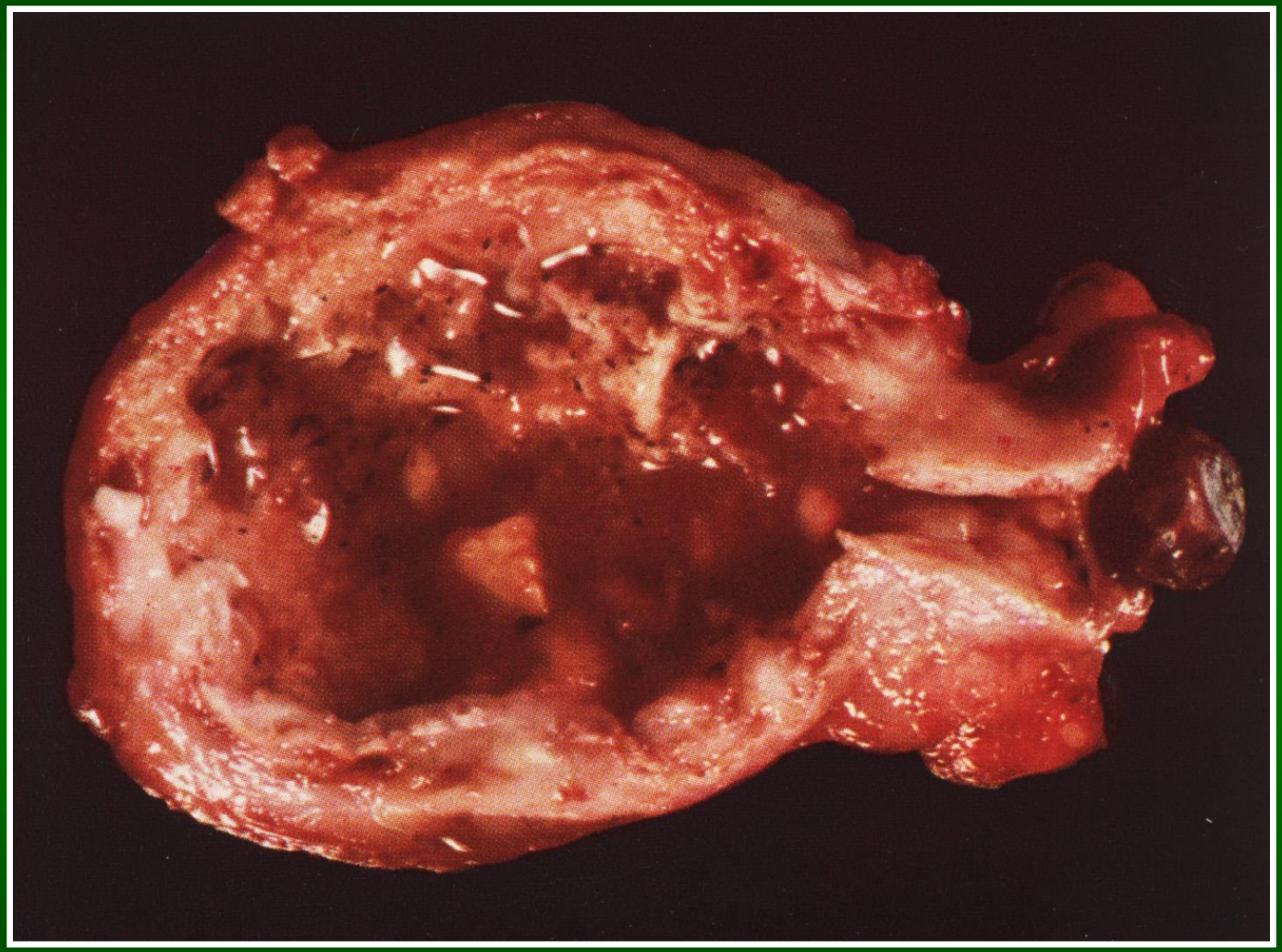
CHOLECYSTO
-LITHIASIS



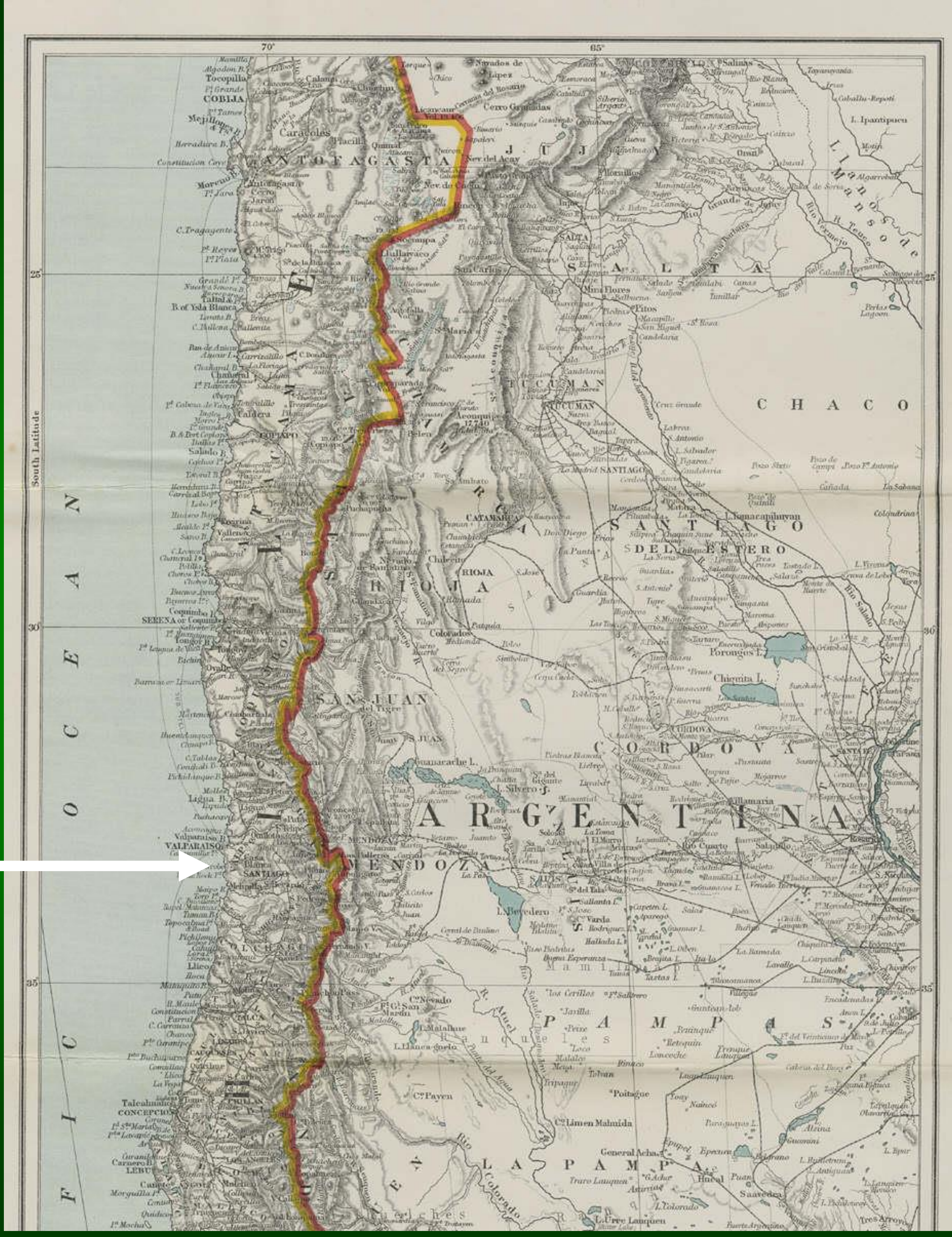
GALL BLADDER HYDROPS

PATHOLOGY OF BILE DUCTS

GALL BLADDER CANCER (CARCINOMA)



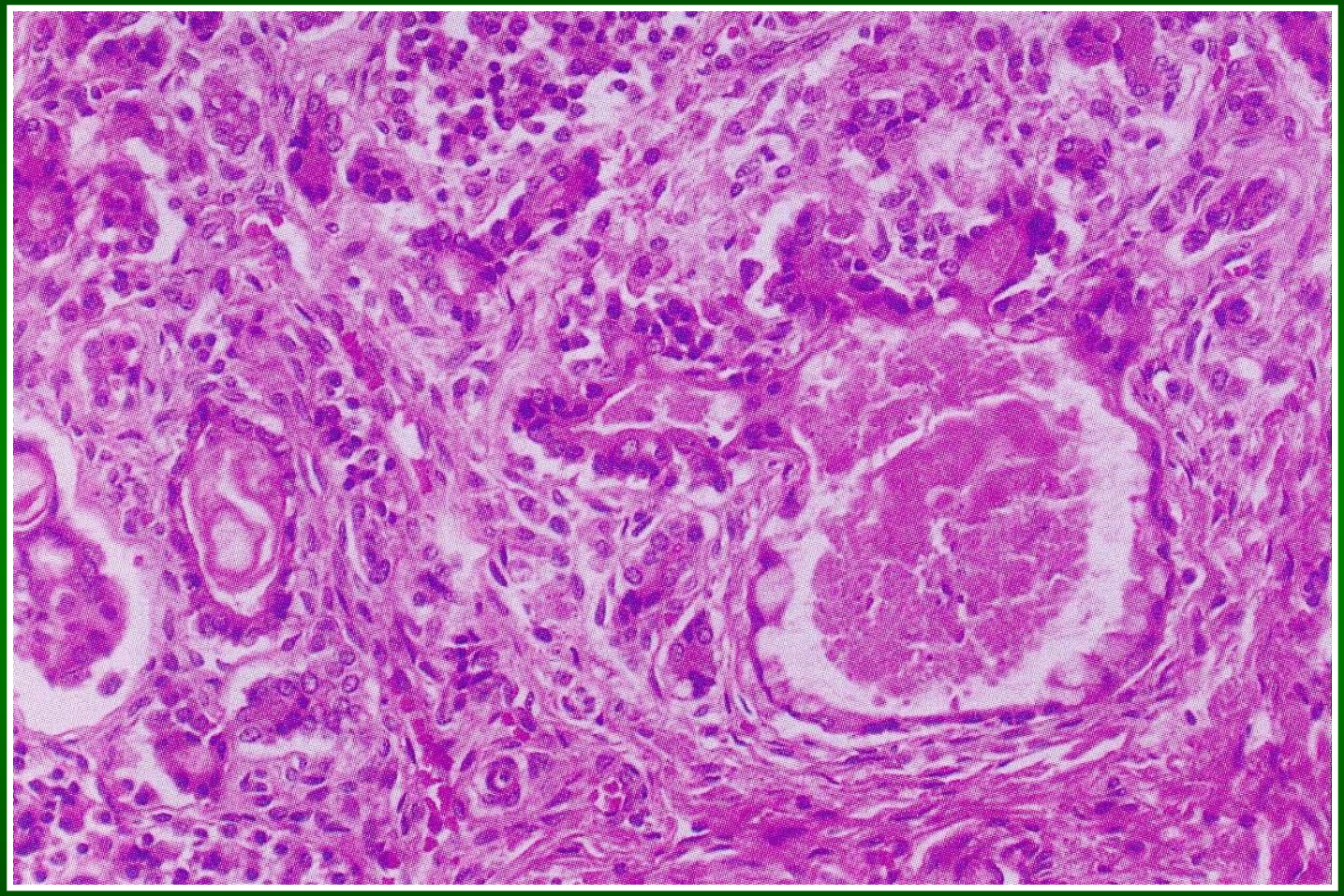
CANCER OF GALL BLADDER IS THE MOST COMMON PLACE OF TUMOR OF BILE TRACT. OCCURS IN WOMEN TWICE AS OFTEN AS IN MEN, EVEN MORE OFTEN OCCURS IN GALL BLADDER WITH STONES !!!



70°
65°
ANTOFAGASTA
JUJUY
SALTA
TUCUMAN
CHACO
CATAMARCA
SANTIAGO DEL ESTERO
RIOJA
SAN JUAN
CORDOVA
ARGENTINA
MENDOZA
SANTIAGO
VALPARAISO
TALCA
CONCEPCION
PAMPAS
LA PAMPA
OCEAN
South Latitude
25°
30°
35°

PATHOLOGY OF PANCREAS

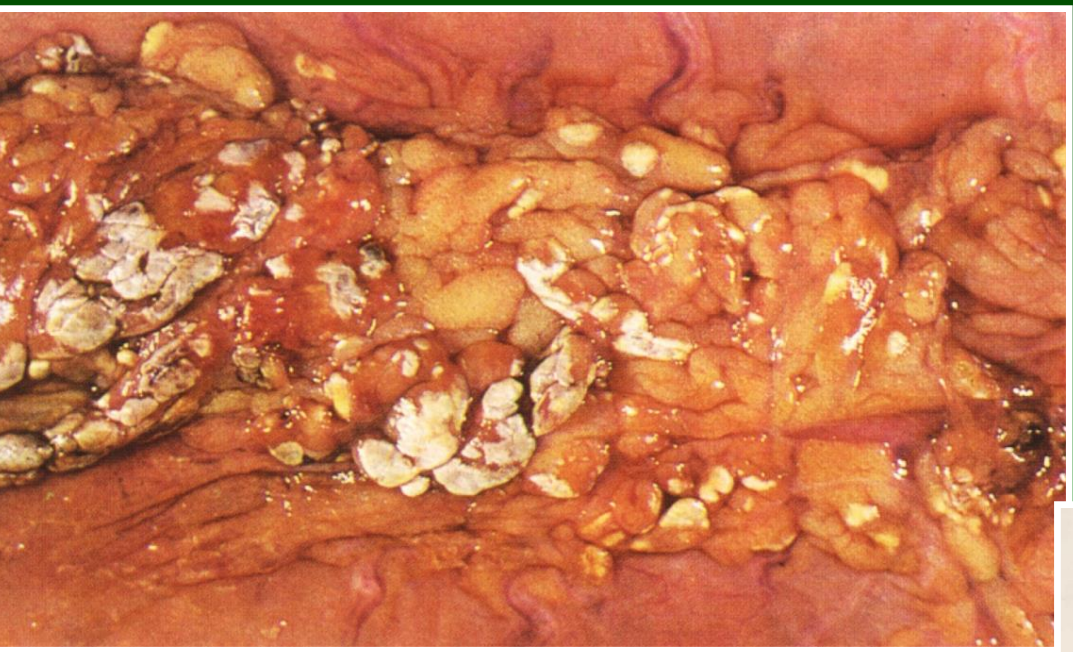
FIBROCYSTIC DISEASE OF THE PANCREAS - MUCOVISCIDOSIS



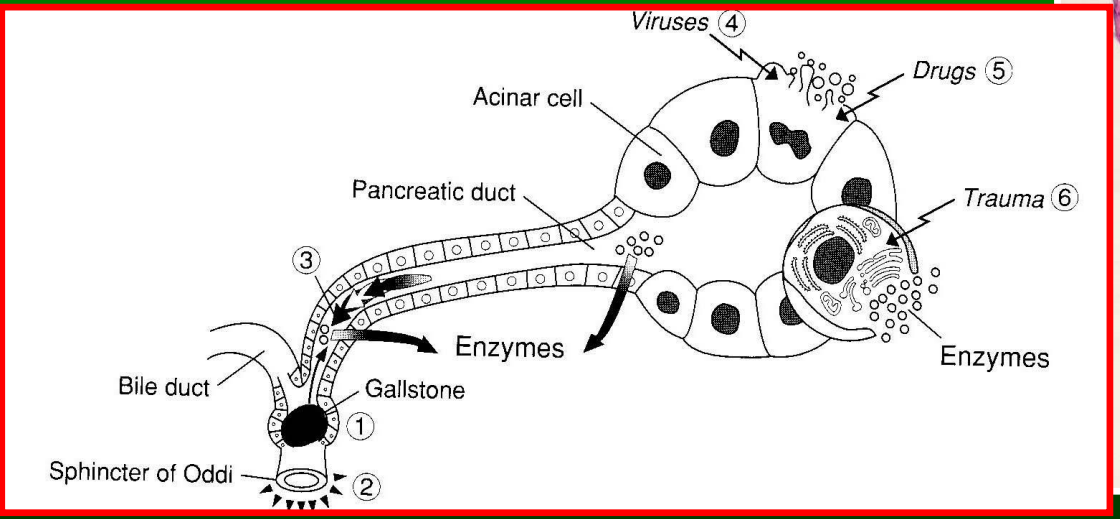
DISTURBANCES IN THE PRODUCTION OF MUCUS, OBSTRUCTION OF TRACTS BY DENSE MUCUS, DILATION OF DUCTS AND FIBROSIS OF ORGAN. GENETIC DISORDER, RECESSIVE TRAIT.

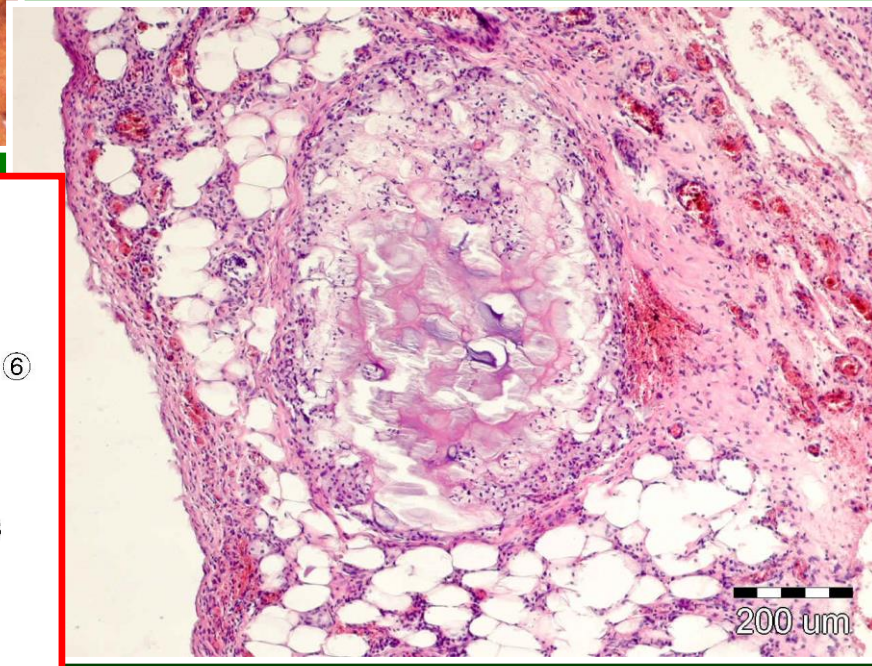
PATHOLOGY OF PANCREAS

ACUTE PANCREATITIS



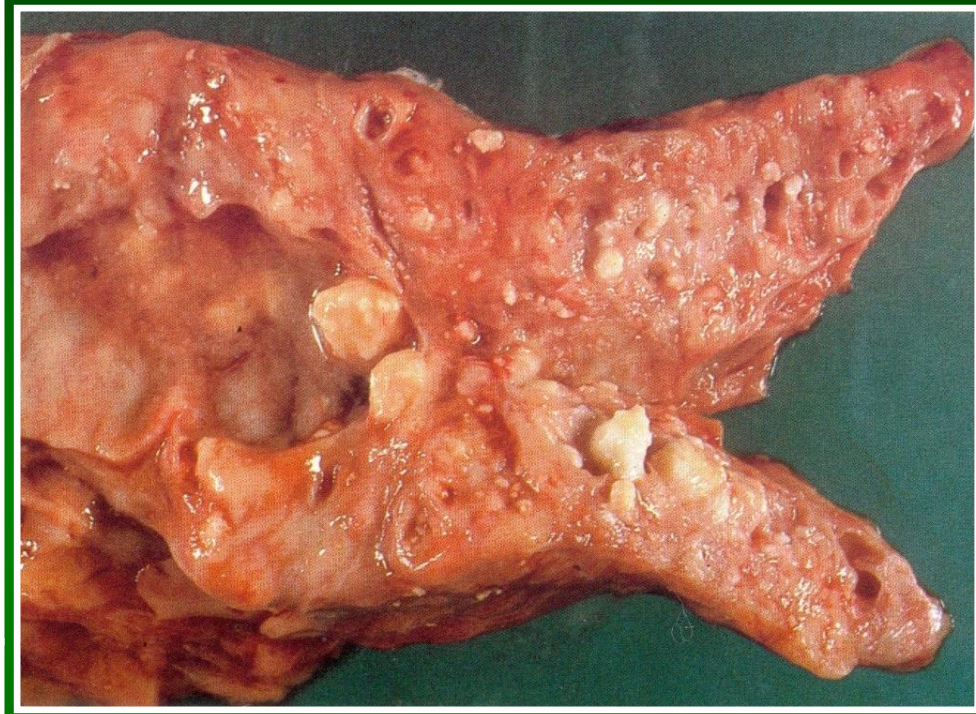
ACUTE PANCREATITIS OCCURS SUDDENLY WITH SIGNIFICANT SHOCK SYMPTOMS.
MORPHOLOGICALLY: ENZYMATIC NECROSIS, CIRCULATORY CHANGES, DIFFERENT REASONS (SEE SCHEME)

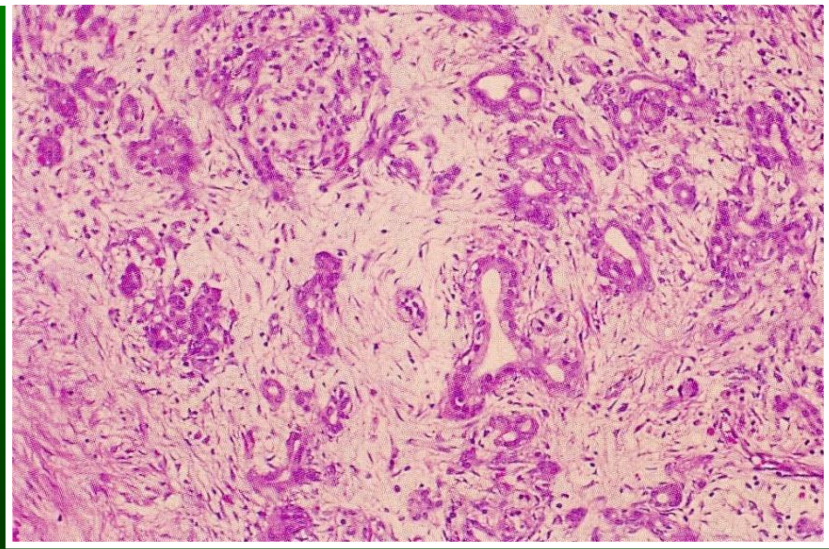




PATHOLOGY OF PANCREAS

CHRONIC PANCREATITIS

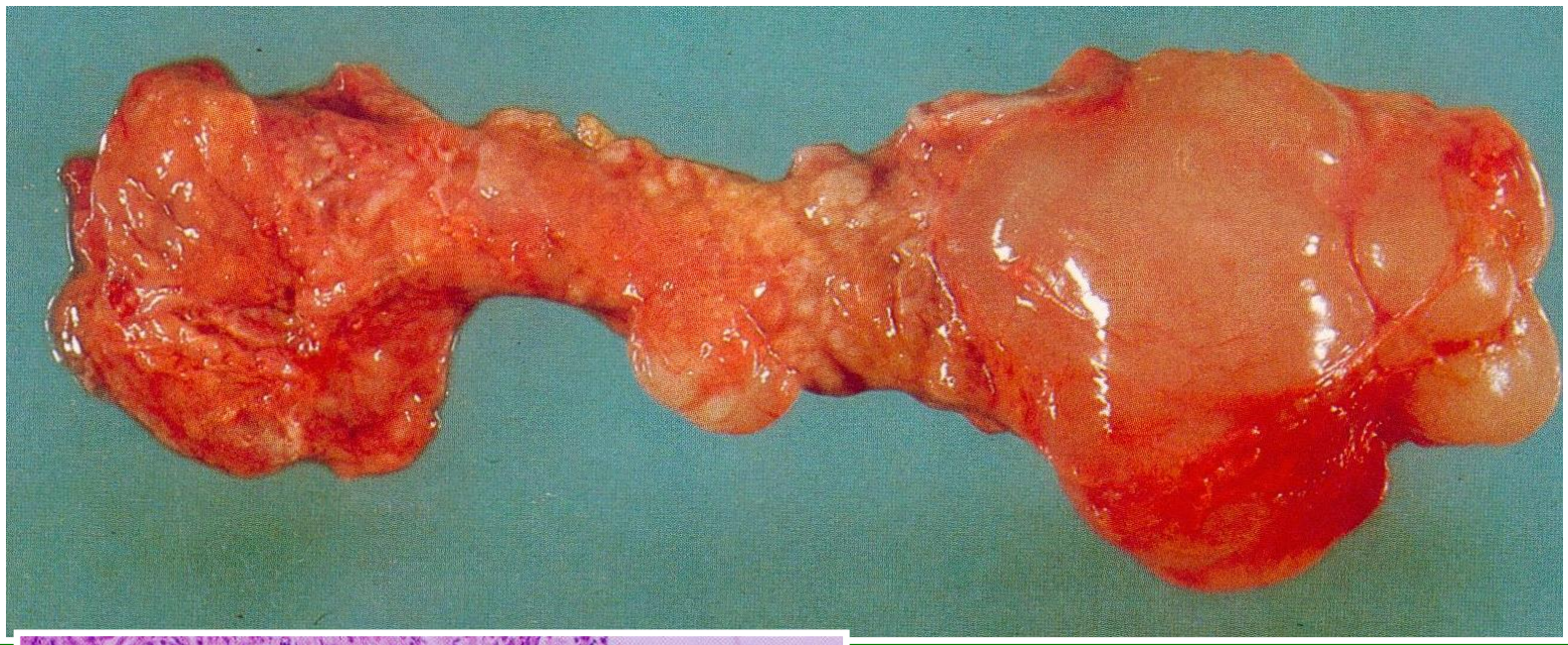


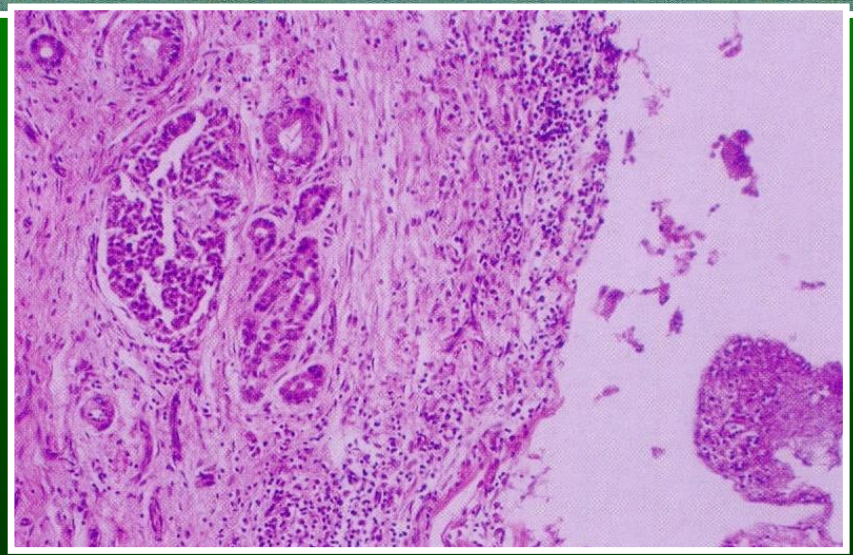


CHRONIC PANCREATITIS LEADS TO FIBROSIS OF ORGAN, SIGNIFICANT REDUCTION IN SECRETION. SOMETIMES INVOLVES PANCREATOLITHIASIS. ADVANCED CHANGES ARE QUALIFIED AS A PRECANCEROUS CONDITION

PANCREAS PATHOLOGY

CYSTS OF PANCREAS – TRUE AND PSEUDOCYSTS

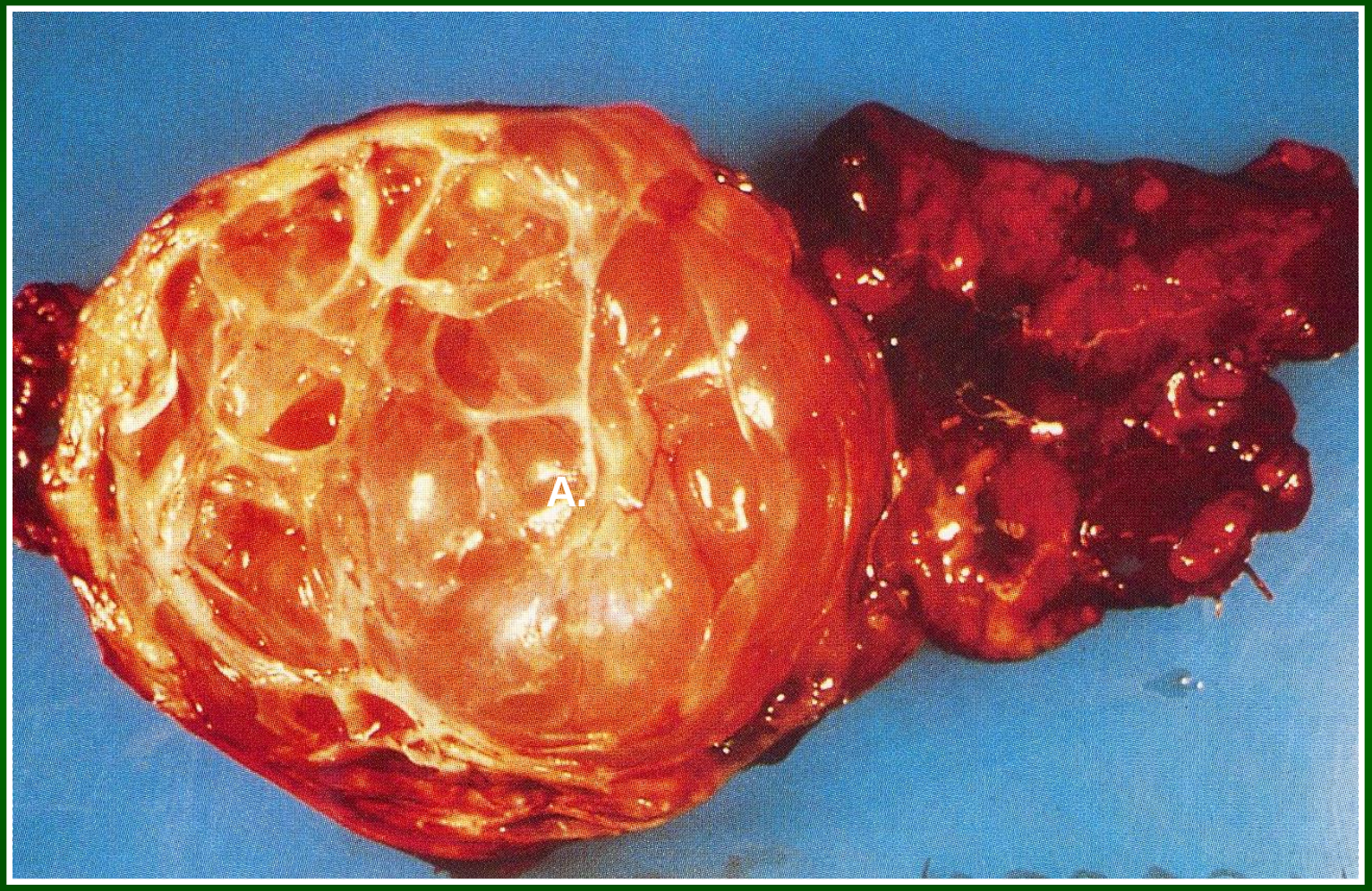




TRUE CYSTS OCCUR IN THE PANCREAS (WITH EPITHELIAL LINING) AND PSEUDOCYSTS (WITHOUT LINING) ARE USUALLY THE RESULT OF THE REABSORBTION OF PREVIOUS PANCREATITIS

PATHOLOGY OF PANCREAS

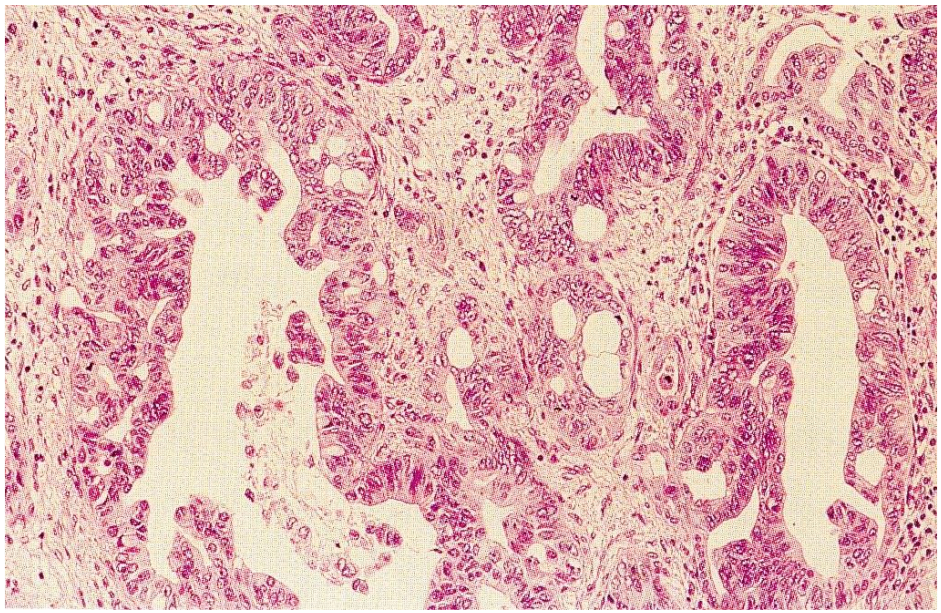
TUMORS OF PANCREAS

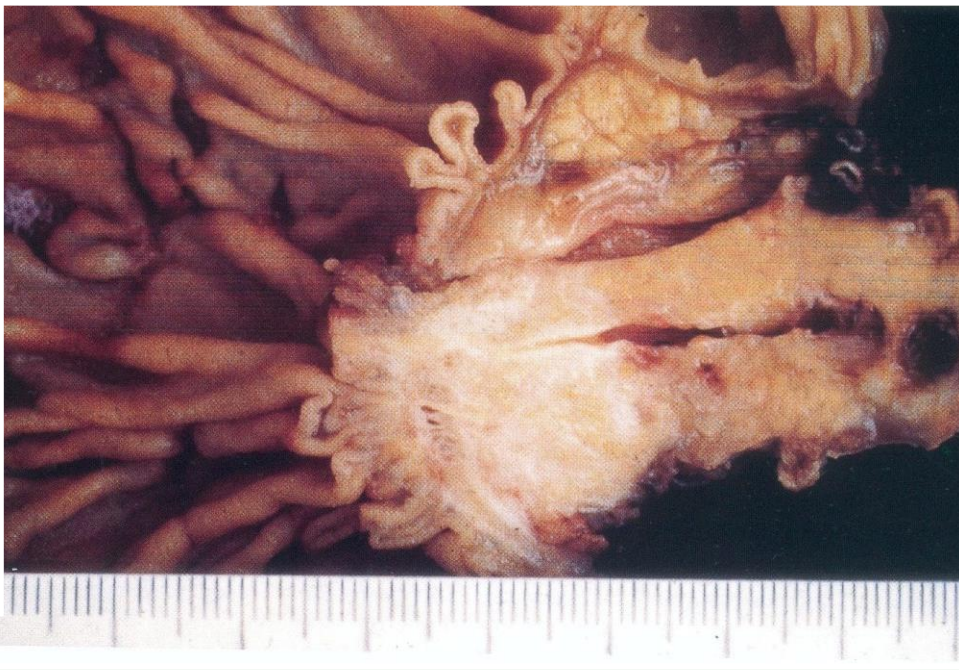


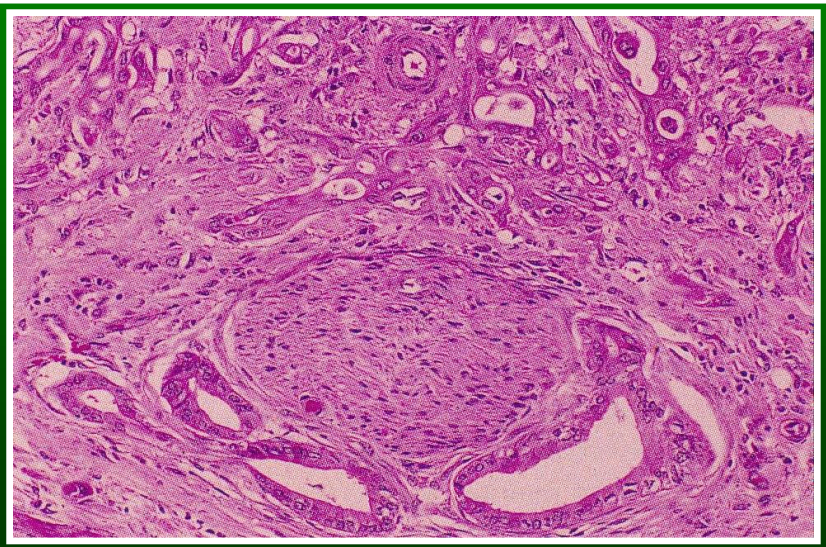
THE MOST COMMON BENIGN TUMORS OF THE PANCREAS ARE MUCINOUS AND SEROUS CYSTADENOMAS

PATHOLOGY OF PANCREAS

TUMORS OF PANCREAS







DIFFERENT FORMS OF ADENOCARCINOMA OF THE PANCREAS

PATHOLOGY OF PANCREAS

TUMORS OF PANCREAS

REMEMBER !!!

PROGNOSIS IN PANCREATIC CANCER IS ALWAYS POOR!!!

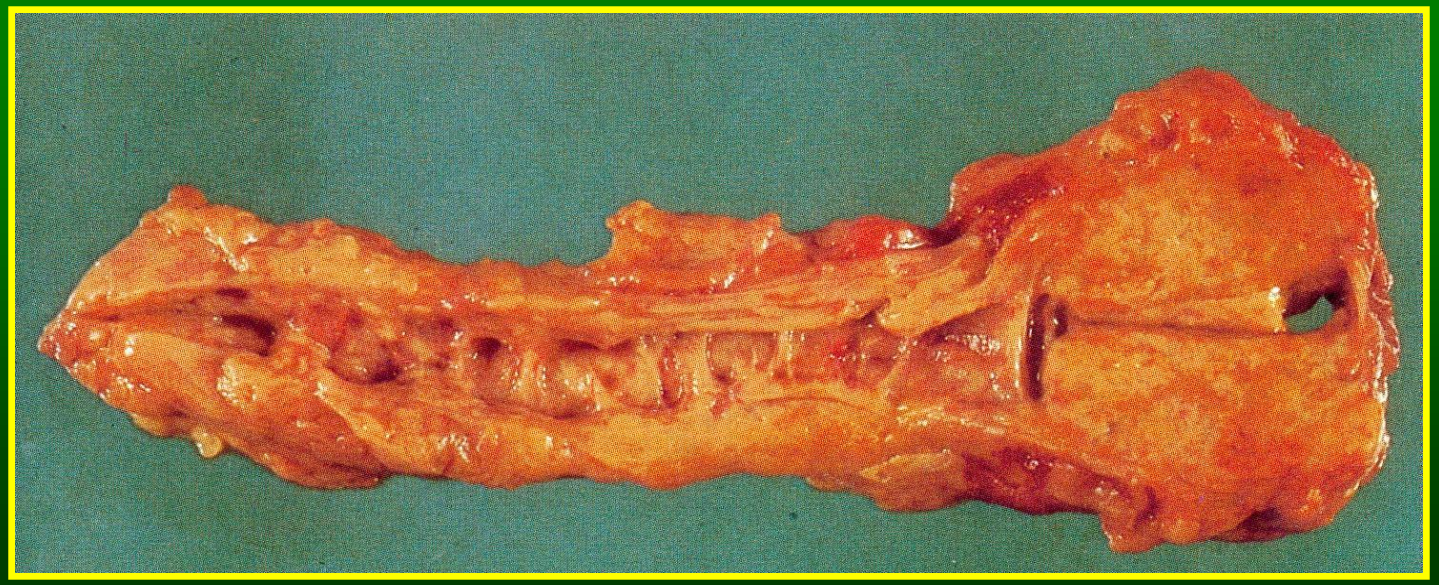
DIFFUSE SURGICAL PROCEDURES (EXCISIONS) ARE THE BEST WAY OF TREATMENT

LOCALIZATION OF THE TUMOR DECIDES THE SYMPTOMS:

CANCER OF HEAD (*CAPUT*) OF PANCREAS – INCREASING OBSTRUCTIVE JAUNDICE

CANCER OF BODY OF PANCREAS – PAIN IN MIDABDOMEN – PAIN OF ROOTLETS INCREASING IN THE SUPINE POSITION

CANCER OF TAIL OF PANCREAS - DIABETES




THANK YOU