

LECTURE 3



PIGMENTARY DEGENERATIONS

EXOGENOUS

PIGMENTATIONS

PNEUMOCONIOSES

ANTHRACOSIS (LUNGS, LYMPH NODES), TATUAGES

SILICOSIS

CHALICOSIS

SIDEROSIS

ASBESTOSIS

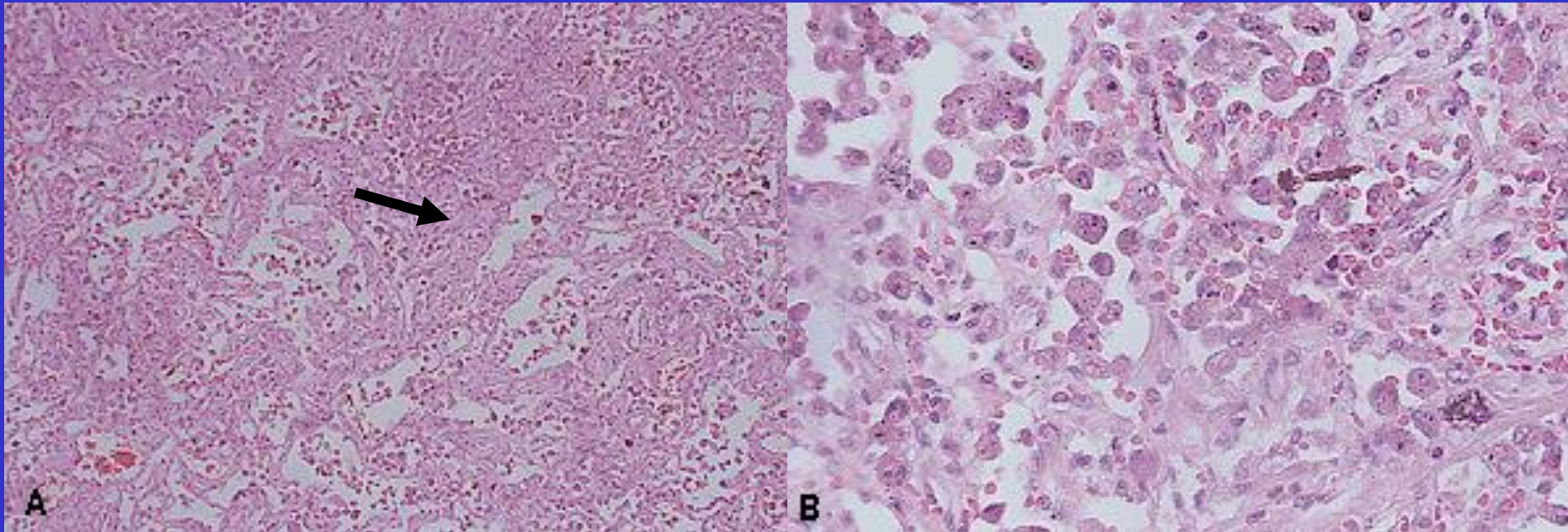
SATURNISMUS (PLUMBISMUS) – LEAD LINE (BLUISH LINE AT THE EDGE OF GUMS), INTESTINAL COLIC, ANAEMIA, NEUROPATHY

ARGYROSIS

PIGMENTARY DEGENERATIONS

EXOGENOUS PIGMENTATIONS

ASBESTOSIS



PULMONARY ASBESTOSIS

A. DIFFUSED INTRAPARENCHYMATOUS FIBROSIS

B. ASBESTOS DEPOSITS IN THE LUNG

ASBESTOSIS

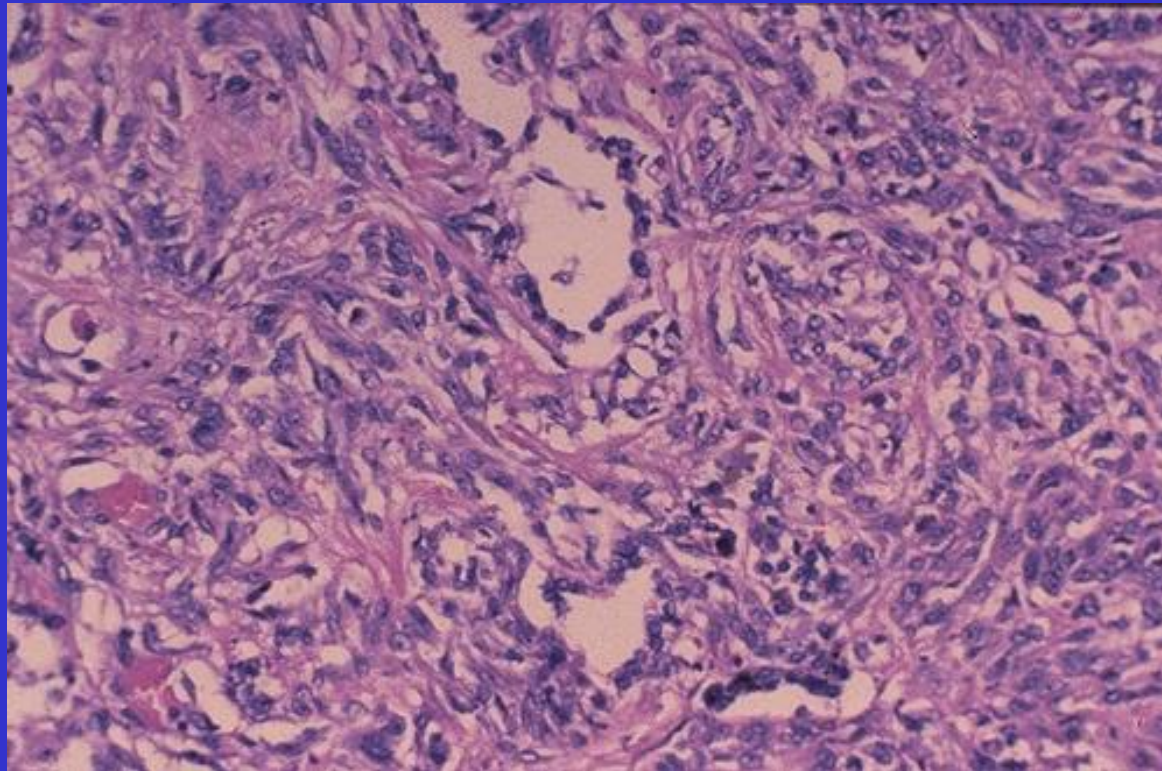


PLAQUES

MESOTHELIOMA

- Malignant mesothelioma arises from mesothelial lining of pleura, peritoneum, pericardium and tunica vaginalis –
- pleural mesothelioma is the most common of these

spindle cells or plump rounded cells forming gland-like configurations



MESOTHELIOMA

The dense white encircling tumor mass is arising from the visceral pleura and is a mesothelioma



MESOTHELIOMA - ETIOLOGY

- Smoking is not a risk factor
- Risk factors include: Asbestos exposure:
- Usually a prolonged latency period
- Studies do not show a linear dose / response relationship between asbestos exposure and malignant mesothelioma
- Radiation
- Erionite: very carcinogenic mineral fiber used in gravel roads
- SV40 virus (association is not clear)

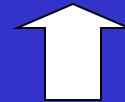
PIGMENTARY DEGENERATIONS

EXOGENOUS PIGMENTATIONS

ANTHRACOSIS OF THE LUNGS



MACROSCOPIC PICTURE

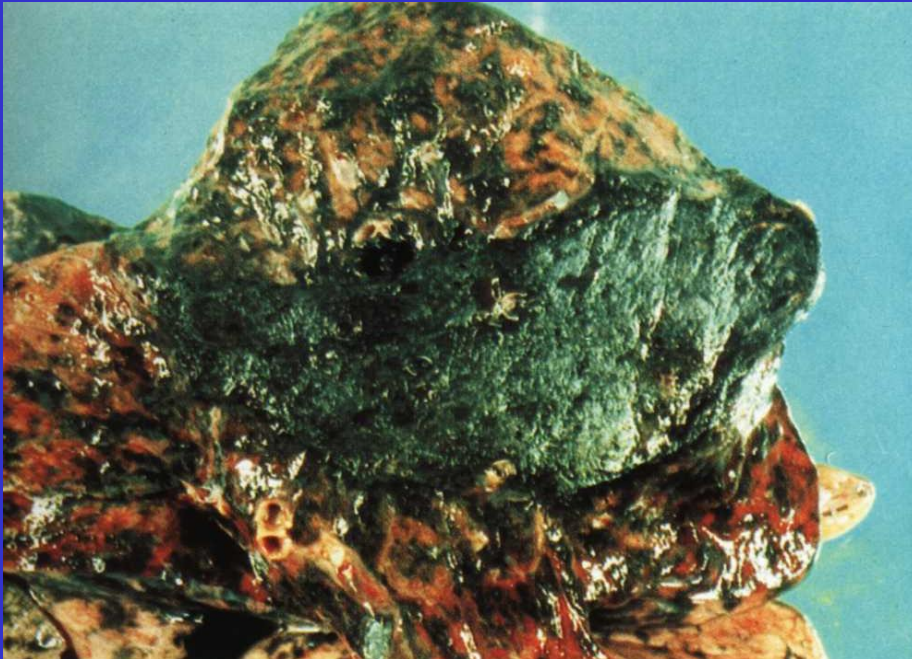


THIN PREPARATION FROM LUNG

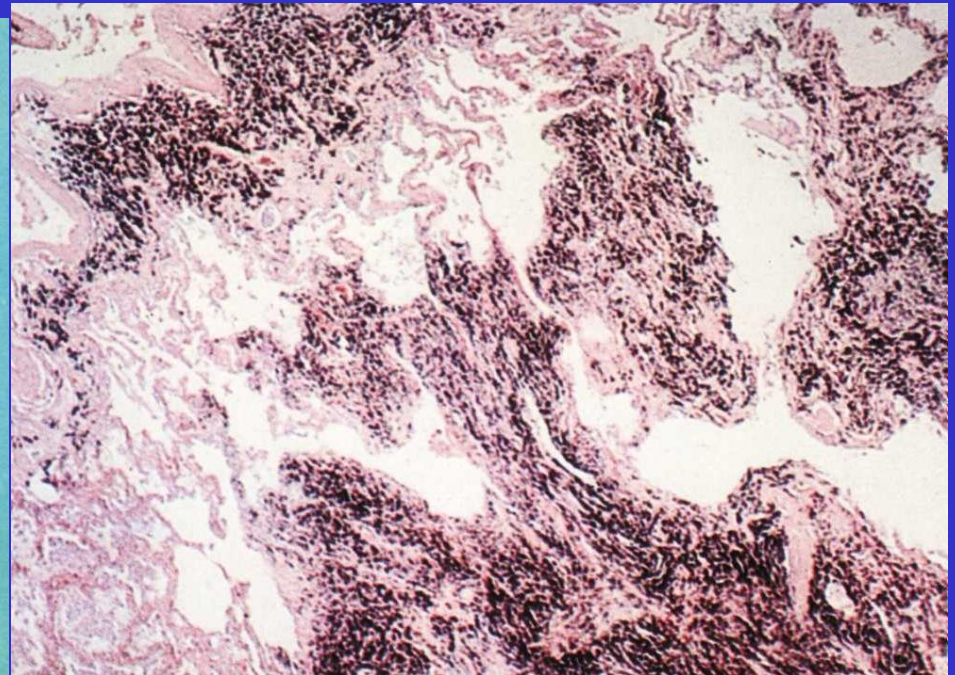
PIGMENTARY DEGENERATIONS

EXOGENOUS PIGMENTATIONS

PULMONARY SILICOANTHRACOSIS



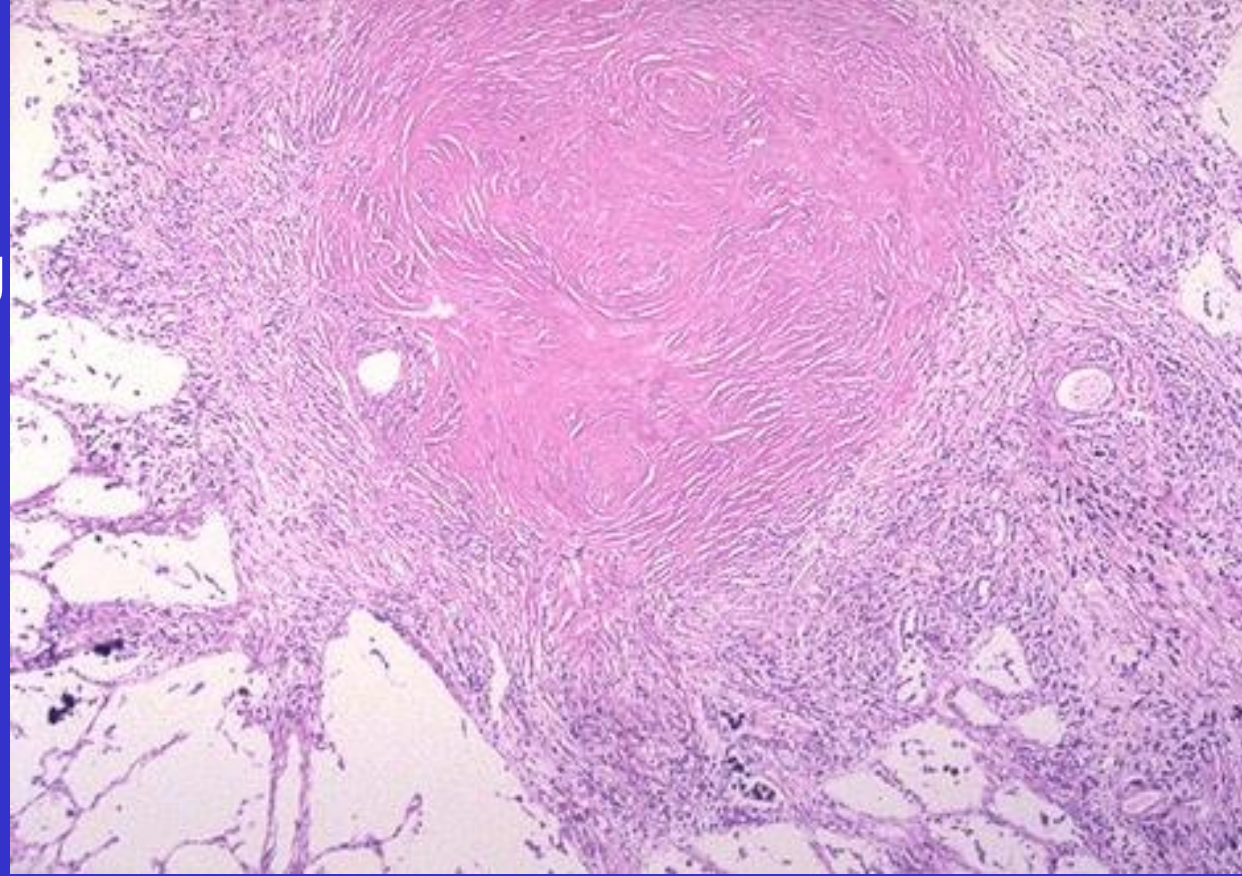
SILICOTIC NODULE IN LUNG



MICROSCOPIC PICTURE OF SILICOTIC FIBROSIS IN LUNG AND ANTHRACOSIS

SILICOTIC NODULE

composed mainly of bundles of interlacing pink collagen; there is a minimal inflammatory reaction.



PIGMENTARY DEGENERATIONS

ENDOGENIC PIGMENTATIONS

NON-HEMOGLOBINOUS DISCOLORATIONS

MELANIN

**PRODUCED BY MELANOCYTES, TRANSPORTED BY MELANOPHORES
PRESENT IN: SKIN, HAIR, CHOROID OF EYE, PIA MATER,**

MELANIN DEFICIENCY:

ALBINISM (CONGENITAL)

VITILIGO (ACQUIRED)

POLIOSIS

PIGMENTARY DEGENERATIONS

ENDOGENIC PIGMENTATIONS

VITILIGO



PIGMENTARY DEGENERATIONS

ENDOGENIC PIGMENTATIONS

NON-HEMOGLOBINOUS DISCOLORATIONS

MELANIN EXCESS

SOLAR IRRADIATION

ARSENIC MELANOSIS

SUPRARENAL MELASMA (ADDISON DISEASE)

MELASMA: OF PREGNANCY; IN CACHEXY

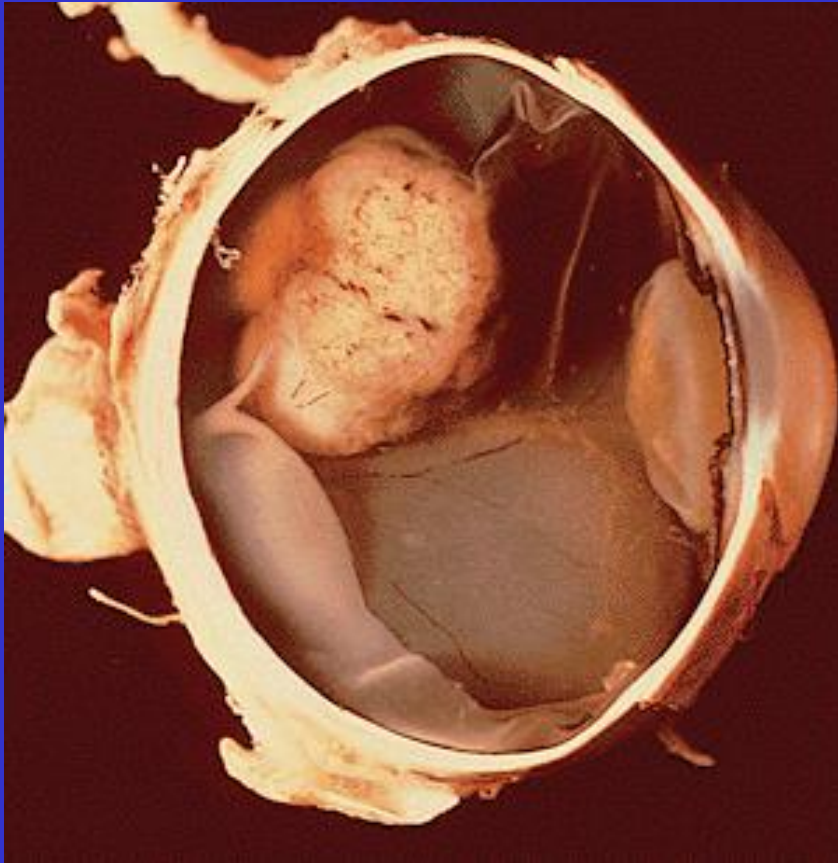
PIGMENTED NEVUS

MALIGNANT MELANOMA

PIGMENTARY DEGENERATIONS

ENDOGENIC PIGMENTATIONS

MALIGNANT MELANOMA



MALIGNANT MELANOMA OF THE EYE



**MALIGNANT MELANOMA
OF THE SKIN**

- **MELANOMA**

- Malignancy of melanocytes, predominantly in skin, but also eyes, ears, GI tract, leptomeninges, mucous membranes
- Only 4% of skin cancers but majority of skin cancer deaths
- Usually due to sun (UV light) exposure
- Incidence increasing worldwide

Clinical warning signs

Change in color of pigmented lesion

Enlargement of existing mole

Itching or pain in mole

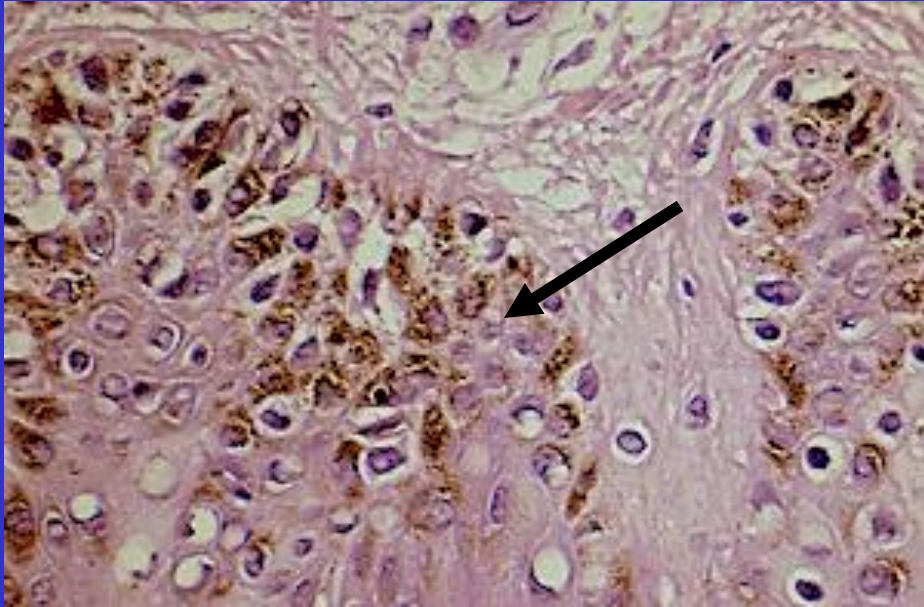
Development of new pigmented lesion in adult life

Irregular borders in pigmented lesion

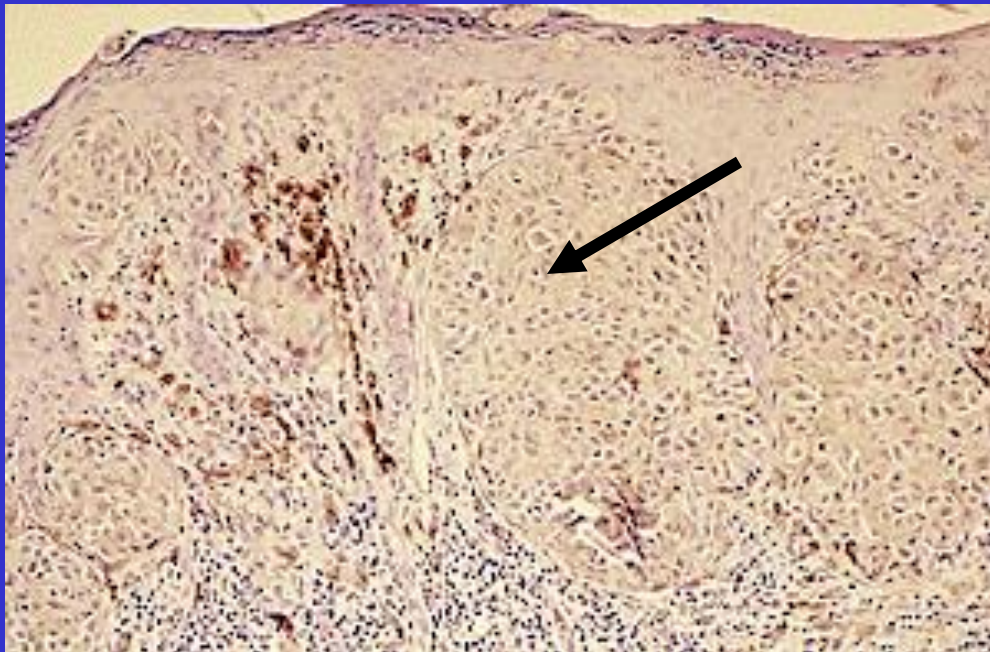
Variation of color in pigmented lesion

PIGMENTARY DEGENERATIONS

ENDOGENIC PIGMENTATIONS



**EPIDERMO-DERMAL
NEVUS PIGMENTOSUS**



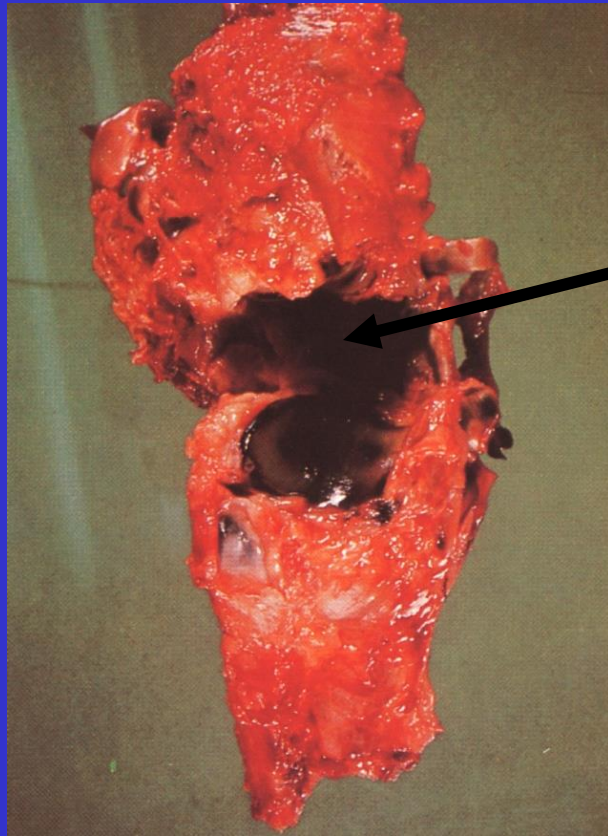
***MELANOMA
MALIGNUM***

DEGENERATIONS PIGMENTARY

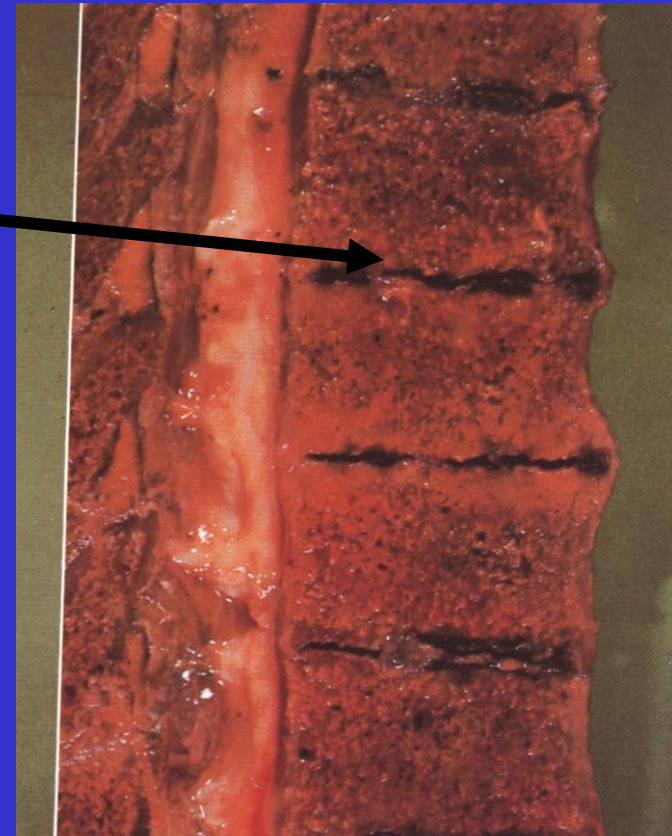
ENDOGENIC PIGMENTATIONS

NON-HEMOGLOBINOUS DISCOLORATIONS

ALKAPTONURIA (OCHRONOSIS, a symptom in alkaptonuria;
1908 r. GARROD - „inborn metabolic mistake” –
CONGENITAL ENZYMOPATHY)



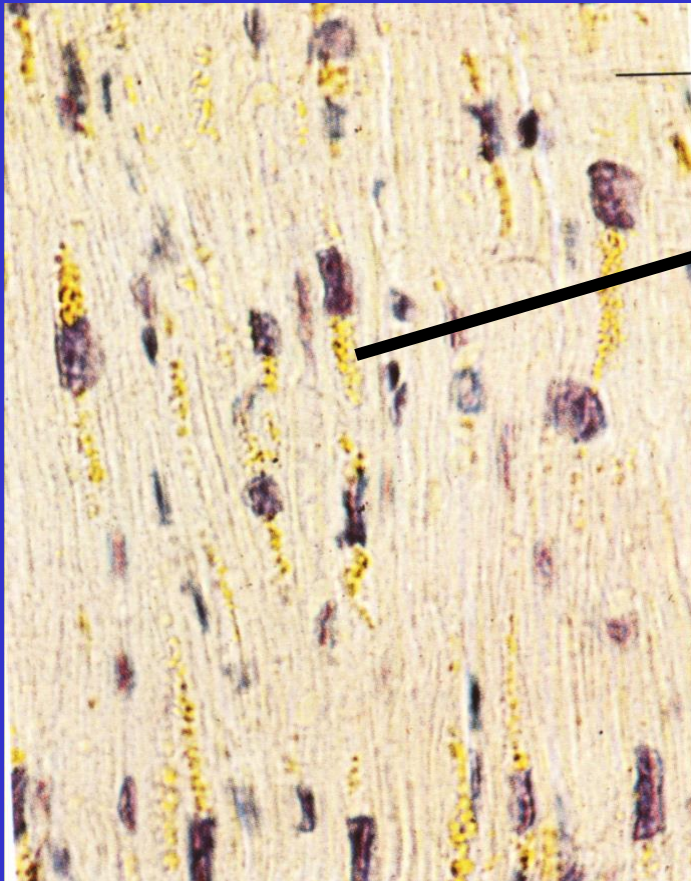
**BLACK
COLORATION
OF CARTILAGE**



PIGMENTARY DEGENERATIONS

NONHEMOGLOBINOUS DISCOLORATIONS

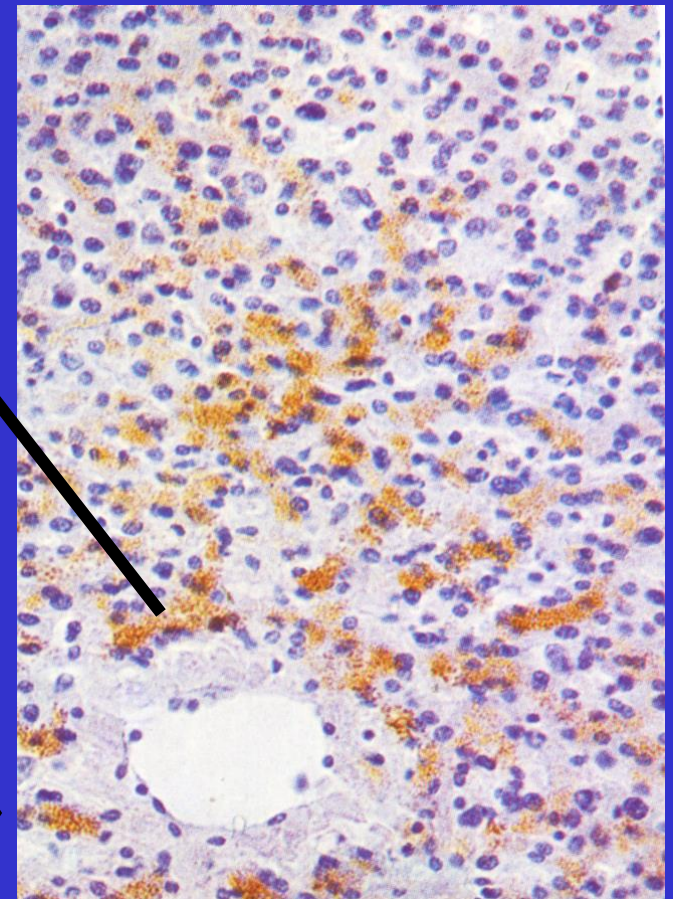
LIPOFUSCIN (LIPOCHROME)



**ATROPHIA
FUSCA
(BROWN
ATROPHY)**

**HEART
MUSCLE**

LIVER



PIGMENTARY DEGENERATIONS

ENDOGENIC PIGMENTATIONS

HEMOGLOBINOUS DISCOLORATIONS

HEMATOIDIN

(DOUBTS: exists or not ?)

1. Does not contain IRON
2. Originates during the period of three weeks
3. Originates without living cells in the center of large haemorrhage

PIGMENTARY DEGENERATIONS

ENDOGENIC PIGMENTATIONS

HEMOGLOBINOUS DISCOLORATIONS

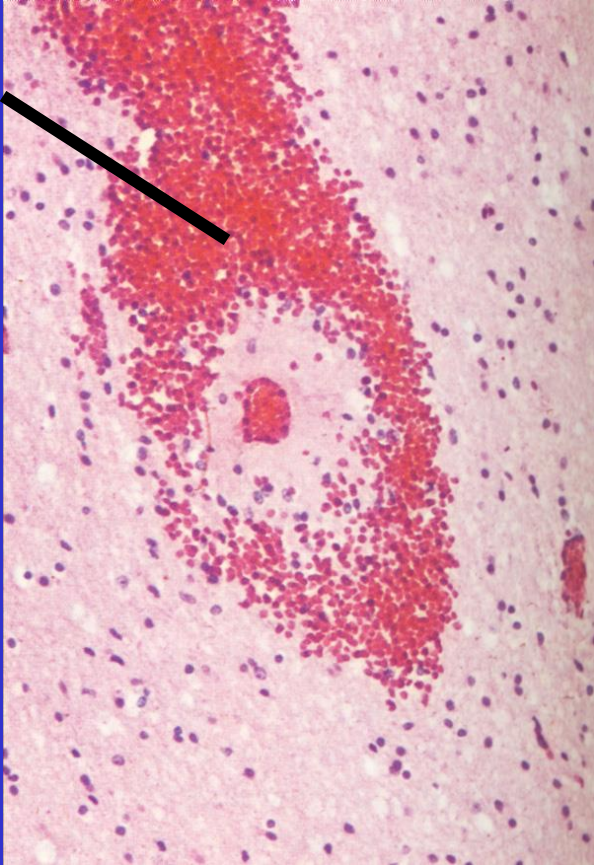
HEMOSIDERIN

- 1. JUNCTION OF IRON HYDROXIDE WITH PROTEIN**
- 2. ORIGINATES FROM IRON WHEN LEVELS OF APOFERRITIN ARE TOO LOW**
- 3. YELLOW-BROWN GRANULAR DISCOLORATION**
- 4. IRON POSITIVE IN STAINING**
- 5. ORIGINATES FROM ERYTHROCYTES WITHIN 5 DAYS**
- 6. OCCURS IN LIVING CELLS ONLY**

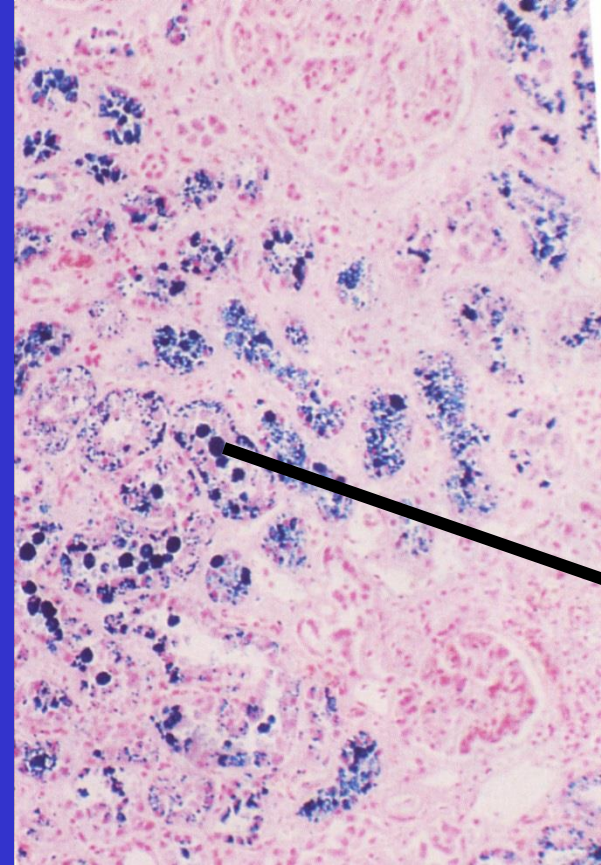
PIGMENTARY DEGENERATIONS

ENDOGENIC PIGMENTATIONS

HEMOGLOBINOUS DISCOLORATIONS



HEMOGLOBIN



HEMOSIDEROSIS OF KIDNEY

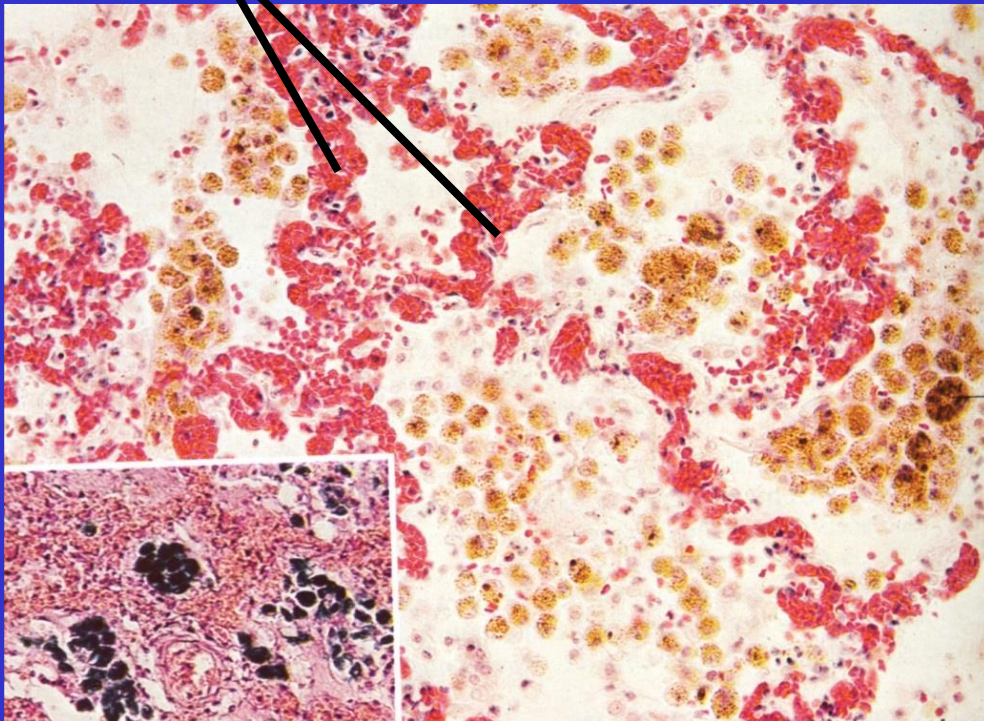
PIGMENTARY DEGENERATIONS

ENDOGENIC PIGMENTATIONS

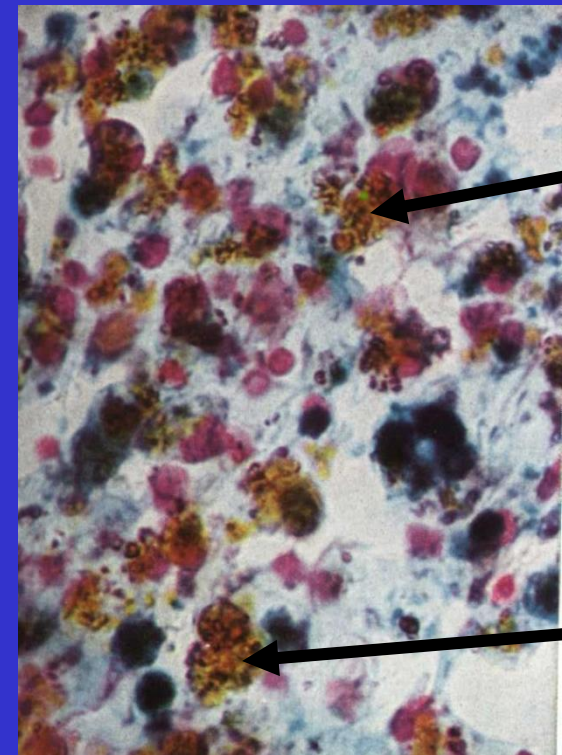
HEMOGLOBINOUS DISCOLORATIONS

HEMOSIDERIN

LOCAL HEMOSIDEROSIS



**CHRONIC VENOSTASIS
(CONGESTION) IN LUNG**



**OLD RED INFARCT MACROPHAGES
FILLED WITH HEMOSIDERIN**

PIGMENTARY DEGENERATIONS

ENDOGENIC PIGMENTATIONS

HEMOGLOBINOUS DISCOLORATIONS

HEMOSIDERIN

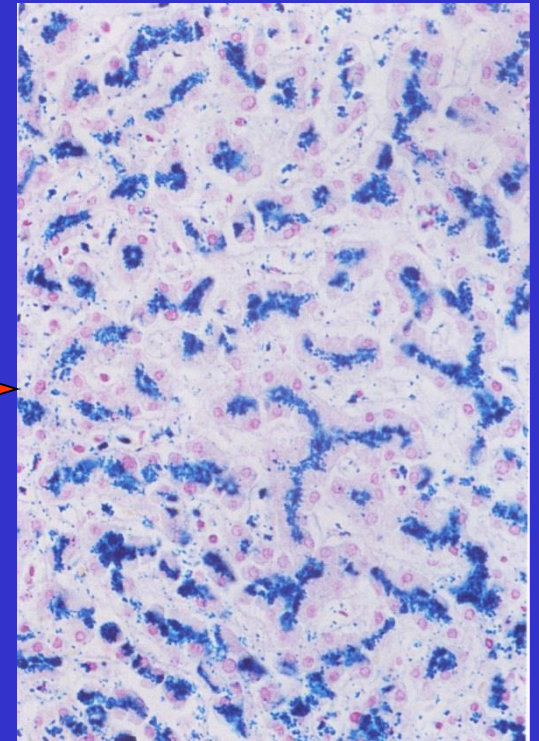
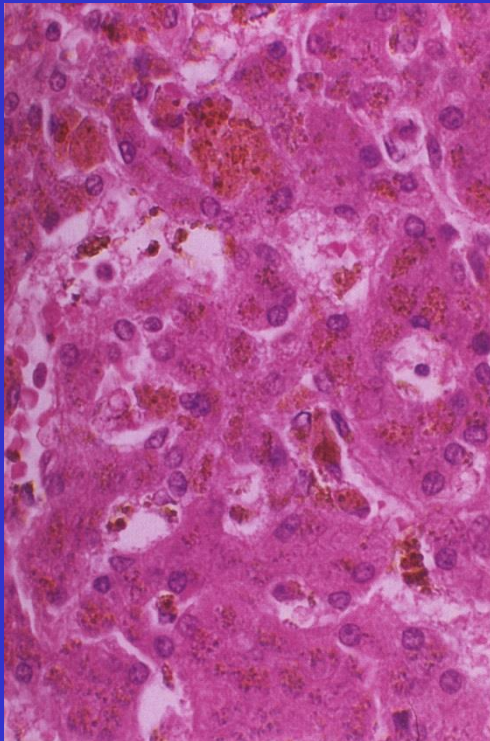
GENERALIZED HEMOSIDEROSIS

CONGENITAL HEMOCHROMATOSIS – AUTOSOMAL AND RECESSIVE

HEMOCHROMATOSIS OF LIVER

H&E

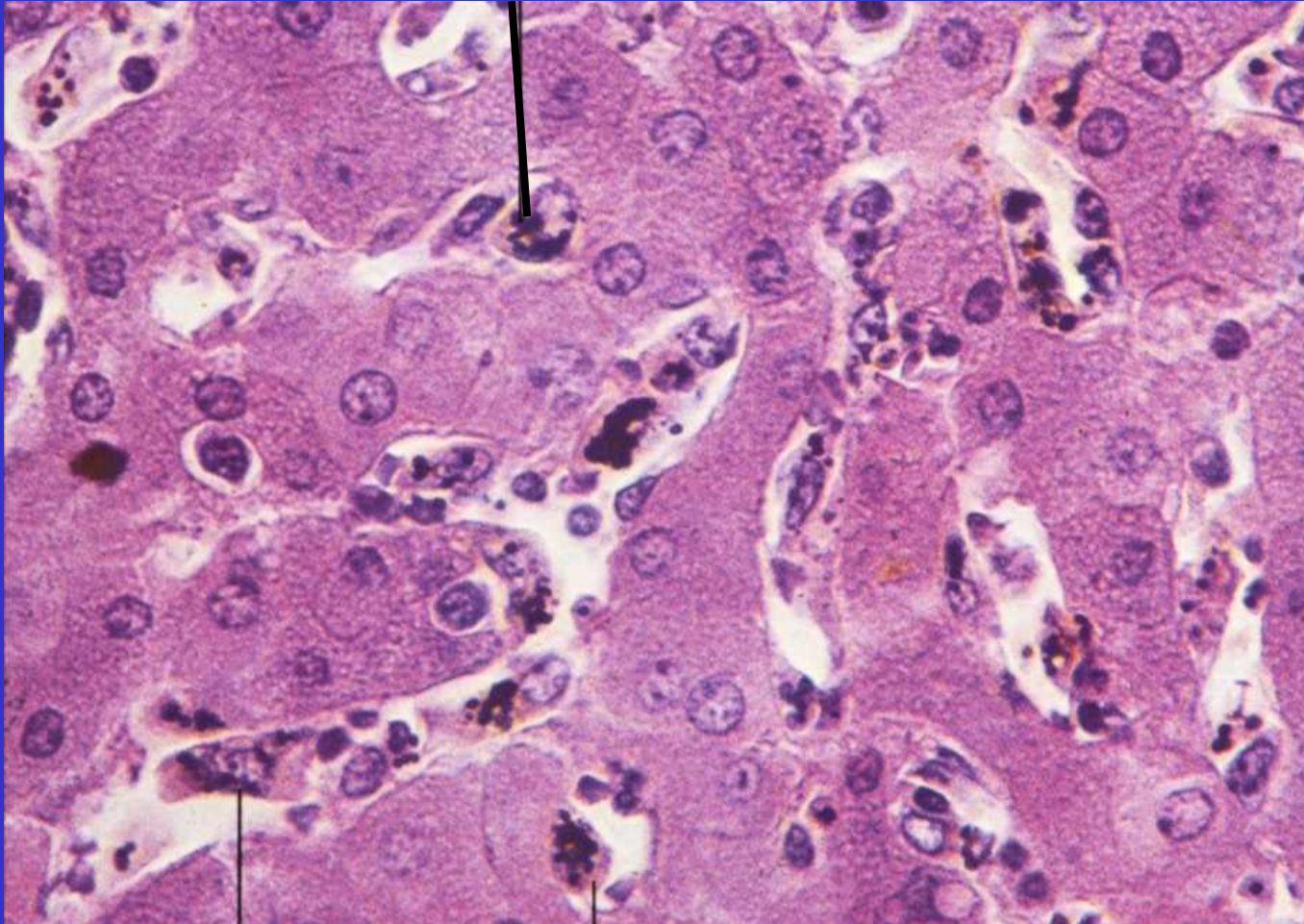
BERLIN BLUE



PIGMENTARY DEGENERATIONS

HEMOGLOBINOUS DISCOLORATIONS

MALARIAL DYE

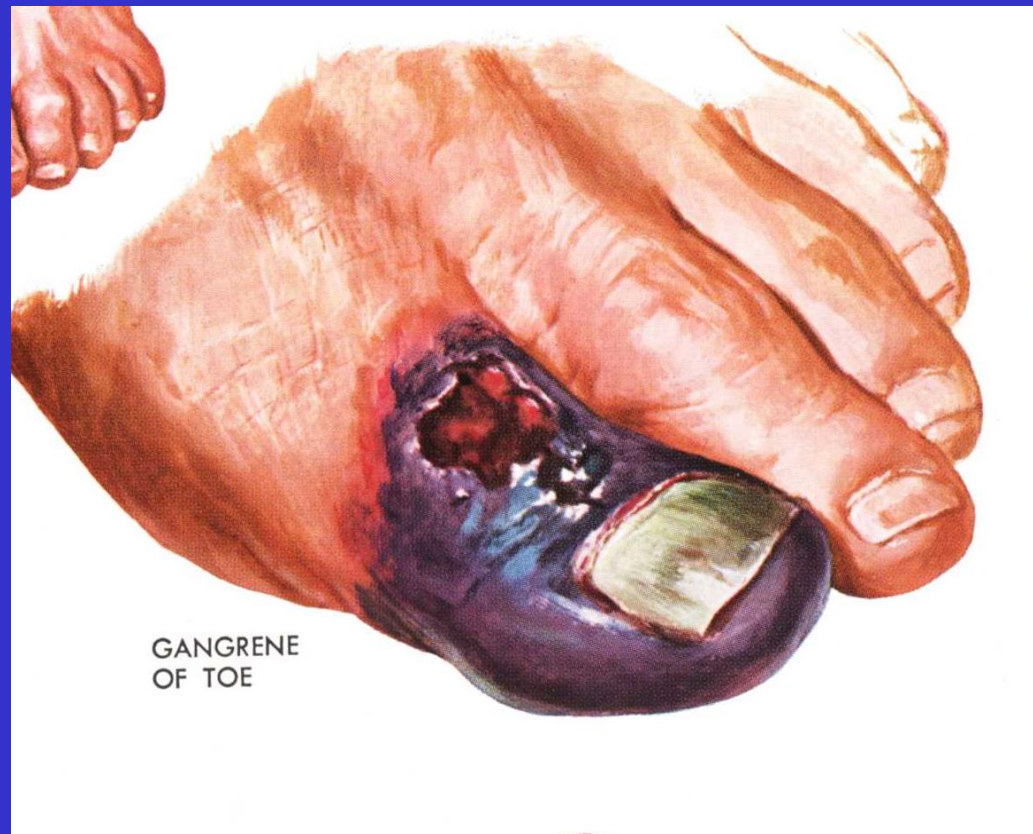


BROWN-BLACK MALARIAL DYE IN KUPFER CELLS

ENDOGENIC PIGMENTATIONS

HEMOGLOBINOUS DISCOLORATIONS

IRON SULPHIDE



GANGRENE OF FOOT AND TOE. BLACK COLOR OF NECROTIC TISSUE CAUSED BY IRON SULPHIDE THAT RESULTS FROM THE IRON IN HEMOGLOBIN

PIGMENTARY DEGENERATIONS

ENDOGENIC PIGMENTATIONS

HEMOGLOBINOUS DISCOLORATIONS

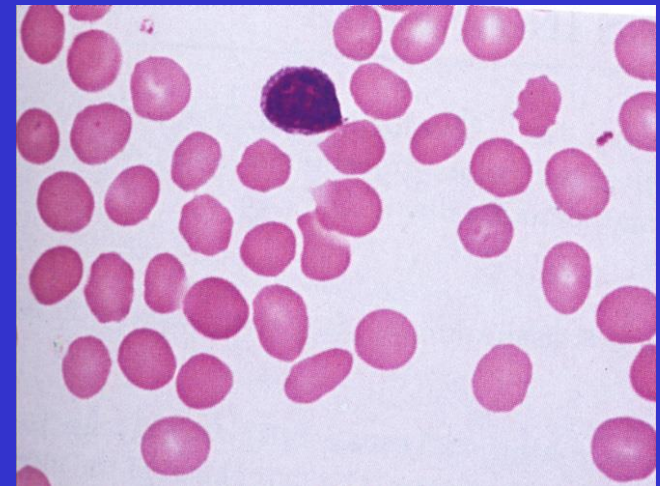
BILIRUBIN

HEMOLYTIC JAUNDICE (*ICTERUS*)

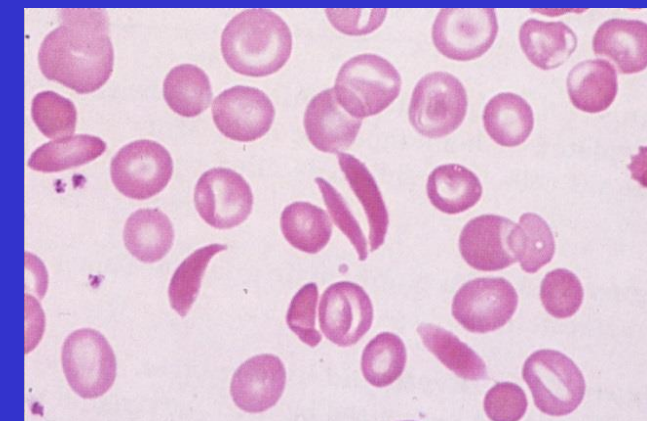


THALASSEMIA:

(IMPAIRED Hb SYNTHESIS – Hb IN CENTRE OF RBC; ERYTHROCYTES ARE SMALL AND PALE)



OVALOCYTIC ANAEMIA



DREPANOCYTIC ANAEMIA

PIGMENTARY DEGENERATIONS

ENDOGENIC PIGMENTATIONS

HEMOGLOBINOUS DISCOLORATIONS

BILIRUBIN

1. PARENCHYMATOUS JAUNDICE

DAMAGE OF LIVER CELLS

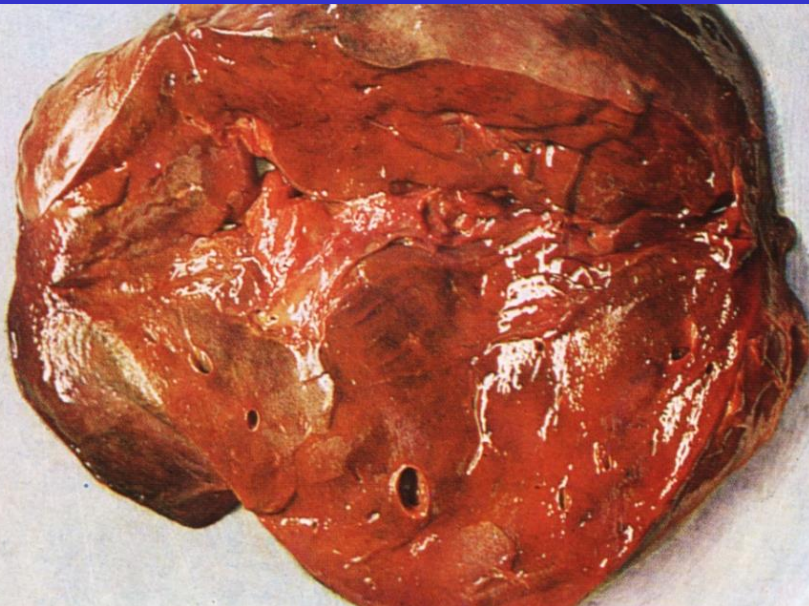
2. MECHANICAL JAUNDICE

HINDERING OR STOPPING OF BILE FLOW TO THE DUODENUM

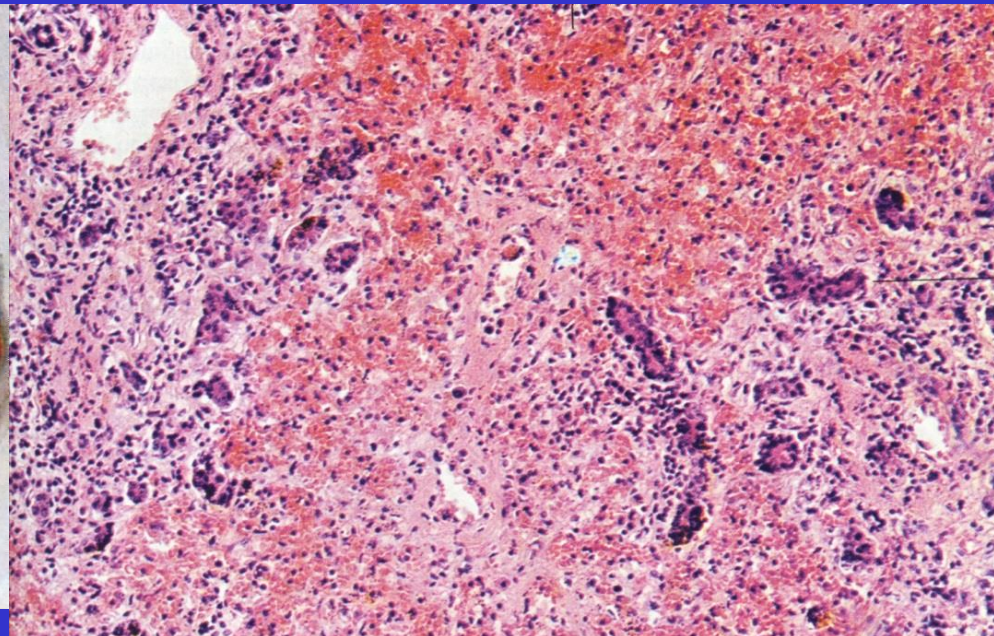
PIGMENTARY DEGENERATIONS
ENDOGENIC PIGMENTATIONS
HEMOGLOBINOUS DISCOLORATIONS
BILIRUBIN

1. PARENCHYMATOUS JAUNDICE

DAMAGE OF LIVER CELLS



ATROPHY OF LIVER



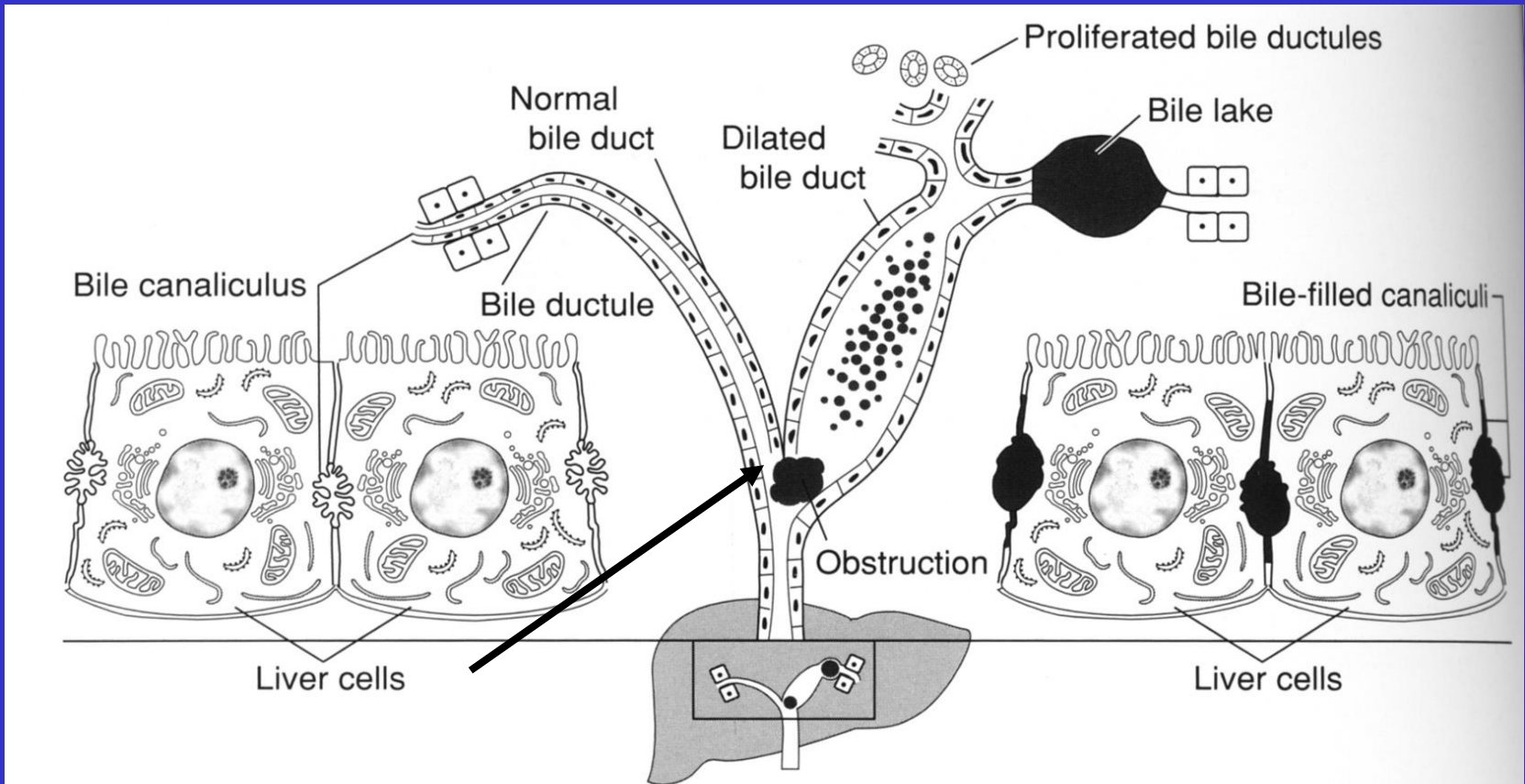
DIFFUSE NECROSIS OF LIVER

PIGMENTARY DEGENERATIONS

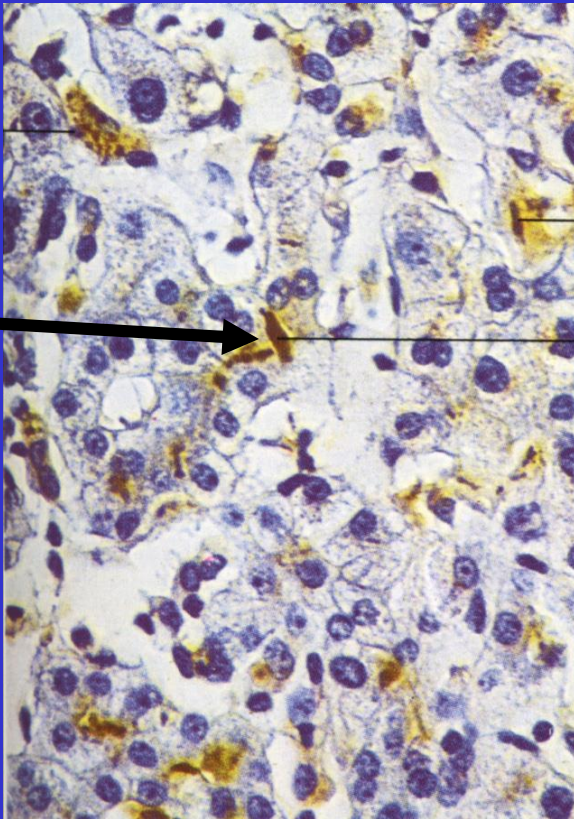
ENDOGENIC PIGMENTATIONS HEMOGLOBINOUS DISCOLORATIONS BILIRUBIN

2. MECHANICAL JAUNDICE

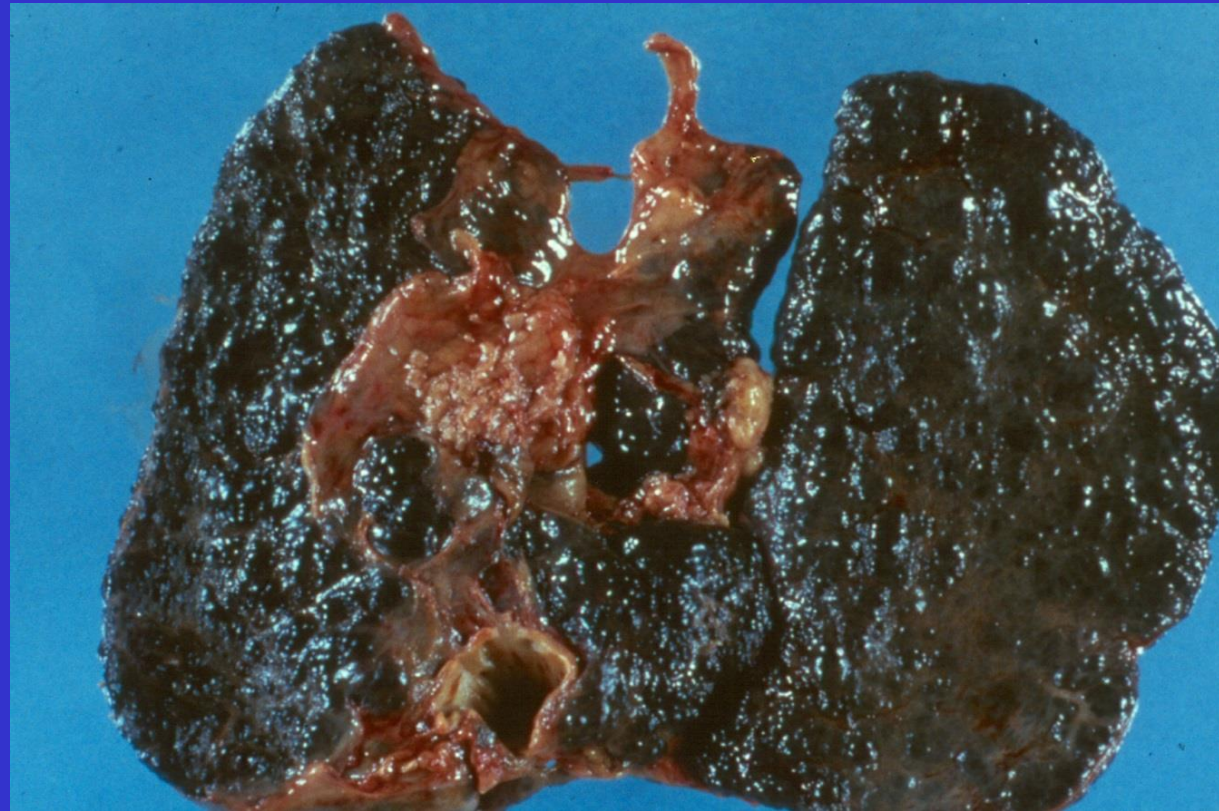
REDUCTION OR COMPLETE BLOCK OF FLOW OF BILE TO DUODENUM



PIGMENTARY DEGENERATIONS
ENDOGENIC PIGMENTATIONS
HEMOGLOBINOUS DISCOLORATIONS
BILIRUBIN
2. MECHANICAL JAUNDICE



CHOLESTASIS IN LIVER



BILIARY CIRRHOSIS OF LIVER

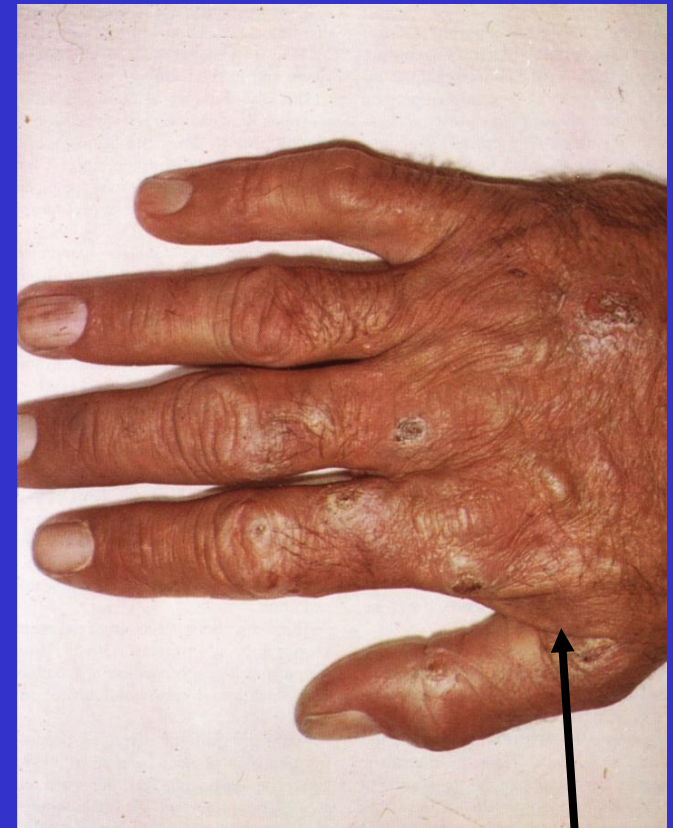
PORPHYRIAS

- group of diseases in which substances called porphyrins build up, negatively affecting the skin or nervous system, and sometimes other organs
- chest pain, abdominal pain, vomiting, constipation, confusion, fever, high blood pressure and high heart rate

PORPHYRIAS

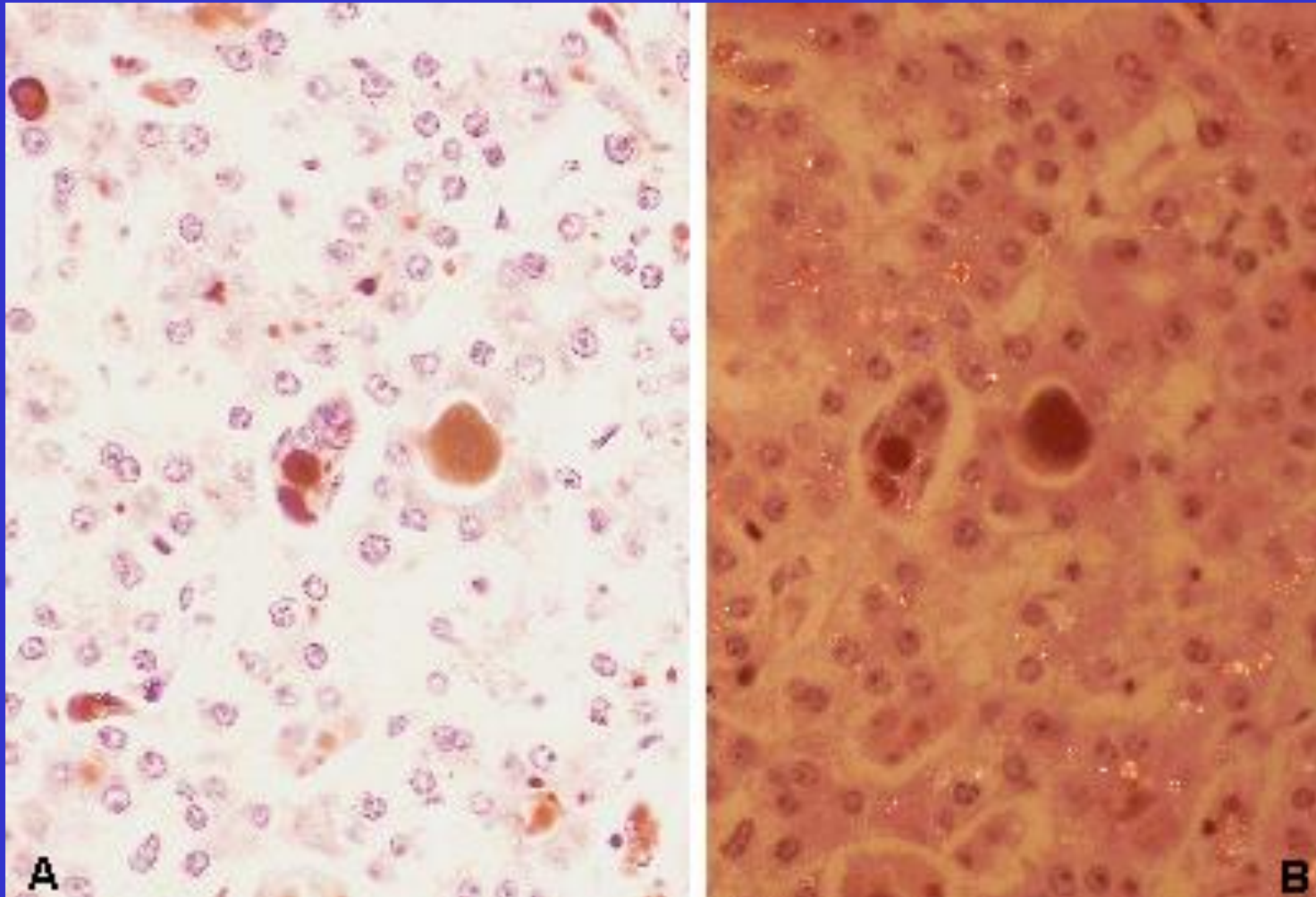


RED PORPHYRIN ILLUMINESCENCE
OF TEETH IN ULTRAVIOLET LIGHT
(405 nm)



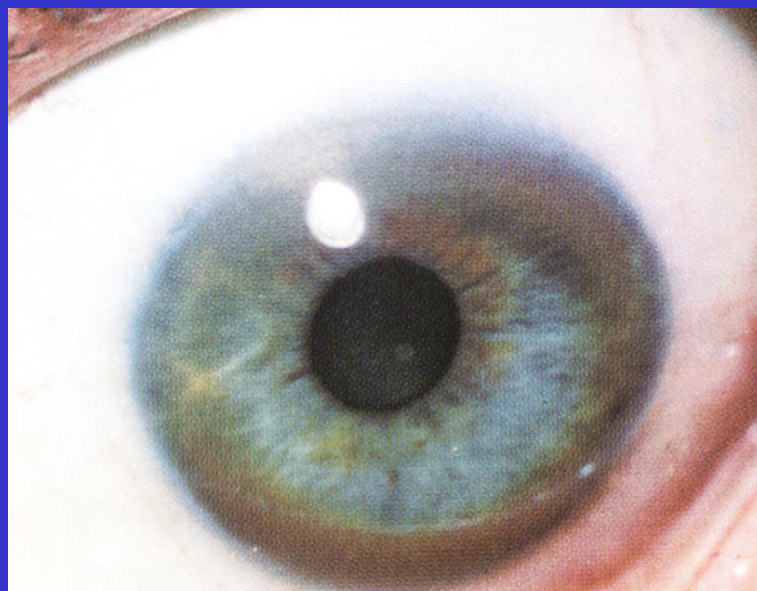
PORPHYRIA CUTANEA
TARDA (ENGLISH: PCT)

PORPHYRIAS



PROTOPORPHYRIA ERYTHROPOETICA A. BILE CYLINDERS (CHOLESTASIS) AND BROWNISH DYE IN HEPATOCYTES B. IN POLARIZED LIGHT

COPPER BALANCE DISORDER



KAYSER-FLEISCHER RING

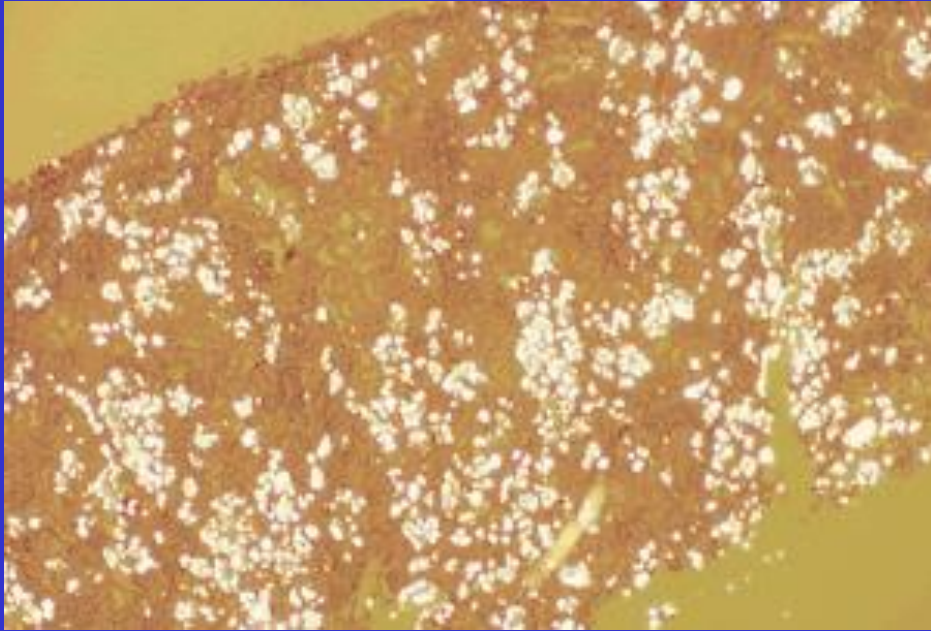
1. CERULOPLASMIN DEFICIENCY
2. GENETICALLY DETERMINED DISEASE
3. Cu IN CORNEA (KAYSER – FLEISCHER RING)
- 4., Cu DEPOSITS IN HEPATOCYTES



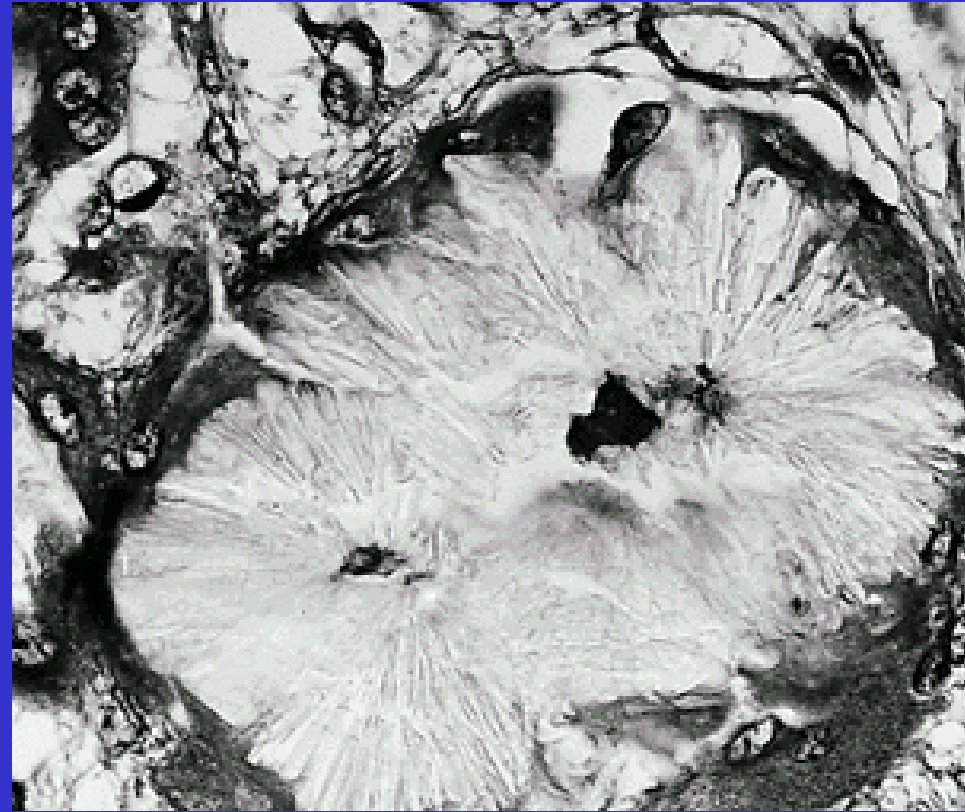
PIGMENTED CIRRHOSIS OF THE LIVER

DISTURBANCES IN MINERAL BALANCE

OXALOSIS (CALCIUM OXALATES)



**OXALATE CRYSTALS IN LUMEN
OF RENAL CANALICULI IN THE
COURSE OF ETHYLENE GLYCOL
POISONING (SLIDE VIEWED IN
POLARIZED LIGHT)**

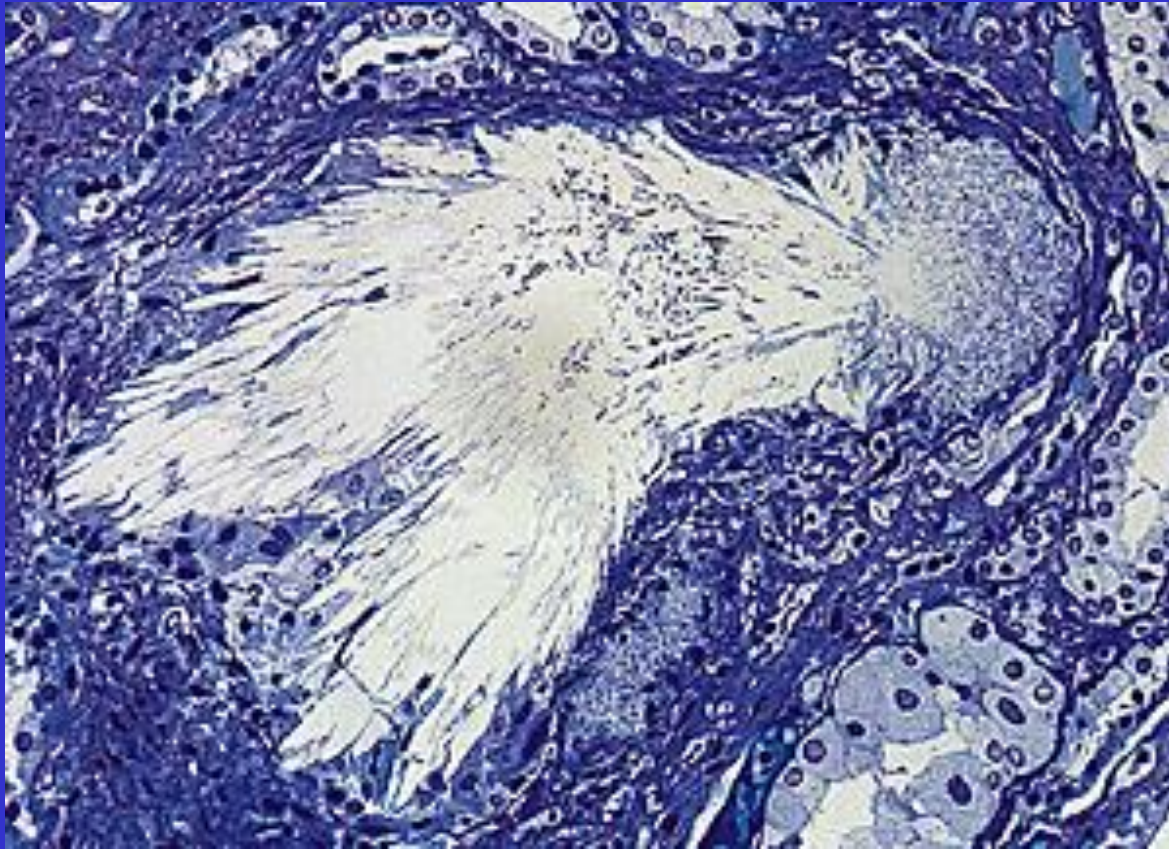


**CHARACTERISTIC VIEW OF
OXALATE IN A BROOM
SHAPE**

DISTURBANCES IN MINERAL BALANCE

URATE

URIC ACID SALTS PRODUCTION AS A RESULT OF DEAMINATION AND
PURINE OXYGENATION



PAIN:

**PODAGRA
(HALLUX)**

CHIRAGRA (HAND)

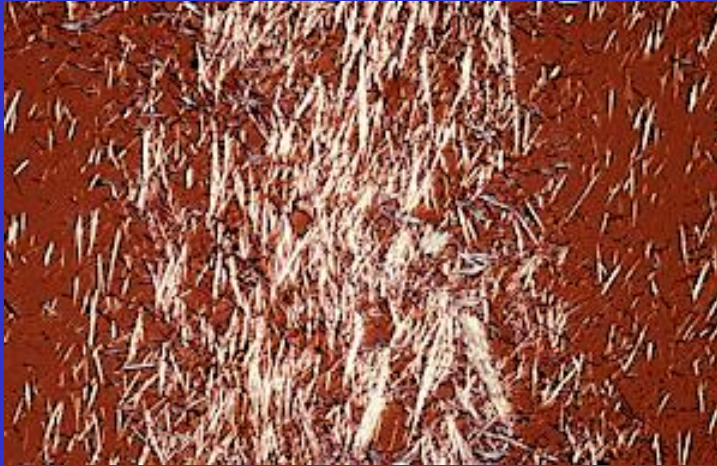
GONAGRA (KNEE)

**OMAGRA
(SHOULDER)**

**URATE CRYSTALS DESTROYING RENAL CANALICULI WITH
ACCOMPANYING INFLAMMATION**

DISTURBANCES IN MINERAL BALANCE

DIATHESIS URICA (GOUT)



**URIC ACID CRYSTALS IN
CARTILAGINOUS TISSUE**



**MICROSCOPIC PICTURE
(TOPHUS)**



**TOPHUS OF LITTLE FINGER
A. X-RAY PICTURE,
B. MACROSCOPIC PICTURE**



DISTURBANCES IN MINERAL BALANCE

CALCIUM

ABSORBED IN THE INTESTINES, EXCRETED BY KIDNEYS, LIVER, PANCREAS, STOMACH. STORED MOSTLY IN BONES

FACTORS CONTROLLING CALCIUM BALANCE:

VITAMIN D

PARATHORMONE

STEROID HORMONES

CALCITONIN

DISTURBANCES IN MINERAL BALANCE

DISTURBANCES IN CALCIUM BALANCE: ENDOCHONDRAL OSSIFICATION AND ITS DISORDERS

Resting cartilage
Stem cells

Proliferating cartilage
Cell division

Vesicular cartilage
Matrix formation

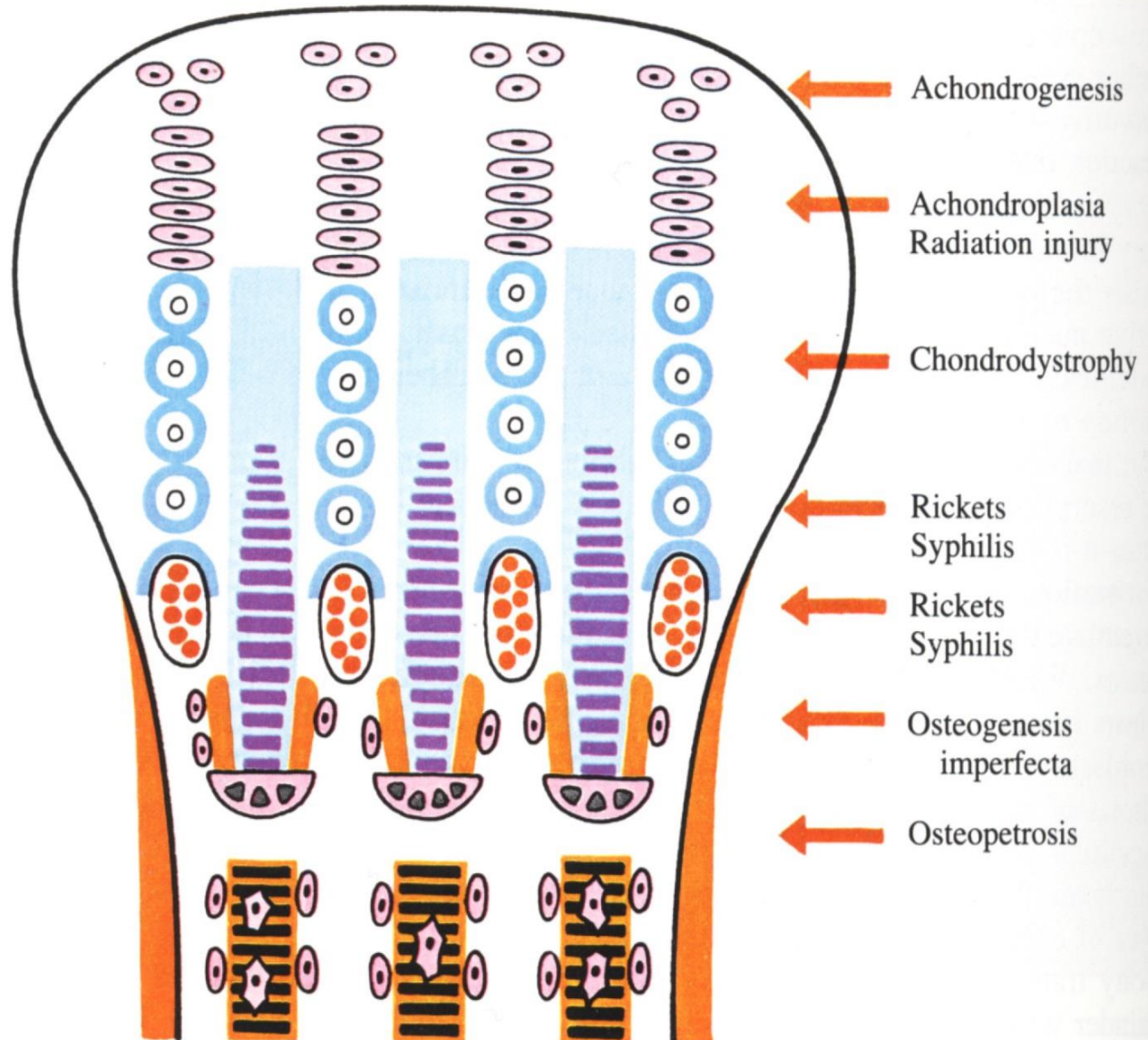
Zone of calcification

Proliferating zone

Primary spongiosa
Osteoid formation

Osteoclastic
bone remodeling

Secondary spongiosa



RICKETS

- Defect in matrix mineralization due to Vitamin D disturbance (deficiency, abnormal metabolism or calcium deficiency)
- Causes accumulation of unmineralized bone matrix
- Rickets: children with irregular, broadened, cup shaped epiphyseal growth plates around knee and wrist

DISTURBANCES IN MINERAL BALANCE

RACHITIS - RICKETS

VITAMIN D DEFICIENCY

STIGMATA RACHITIS

CRANIOTABES (soft, thinned skull bones)

CAPUT QUADRATUM

ROSARIUM RACHITICUM (rachitic rosary)

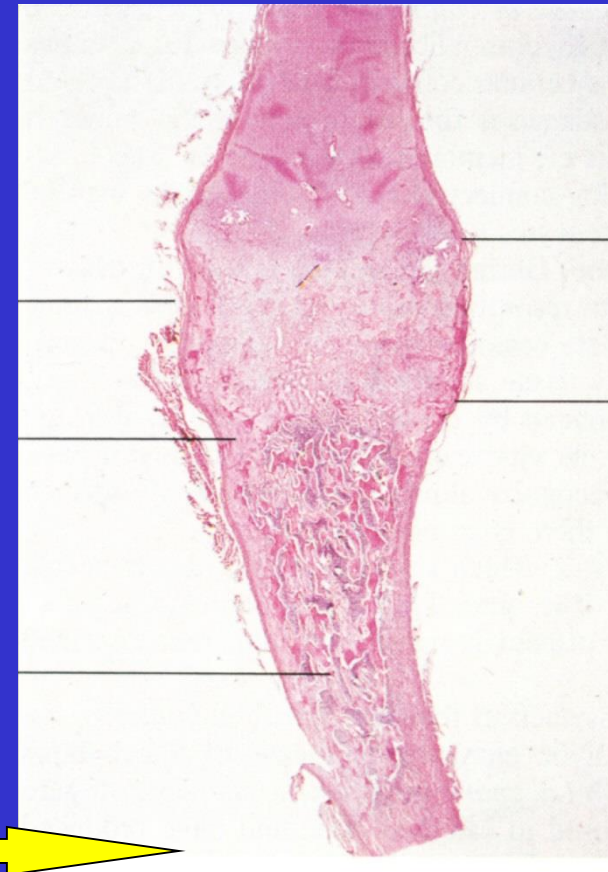
PECTUS GALINACEUS ET PECTUS INFUNDIBULARIS

SCOLIOSIS, KYPHOSIS, LORDOSIS, GIBBUS

PELVIS PLANUS (flattened pelvis)

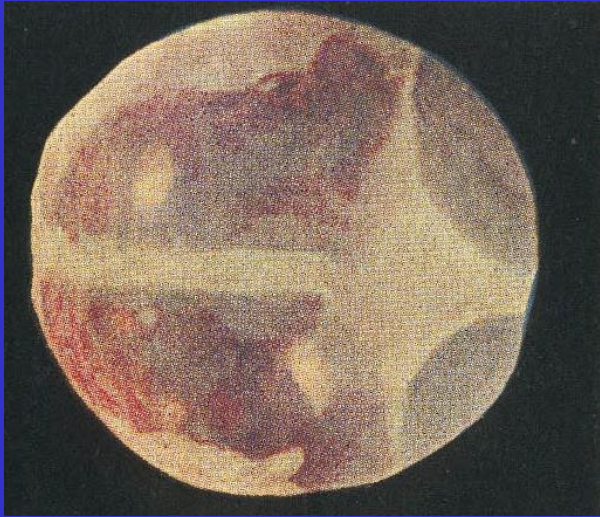
GENUA VARA ET GENUA VALGA

Hypocalcemia, a low level of calcium in the blood can result in tetany – uncontrolled muscle spasms

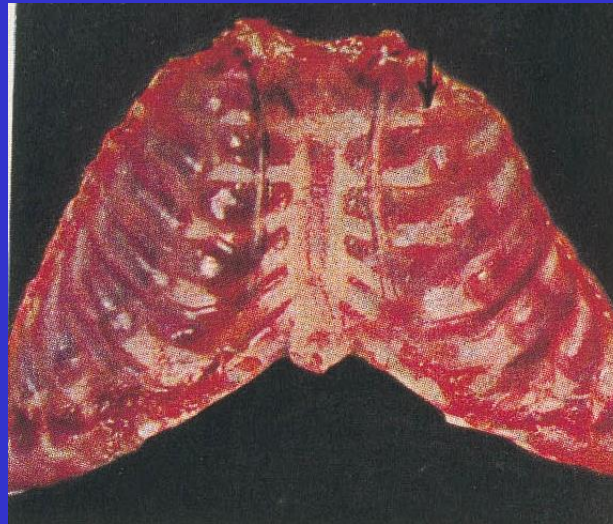


DISTURBANCES IN MINERAL BALANCE

RICKETS - RACHITIS



**FONTANELLES AND SUTURES
DELAYED CLOSURE**



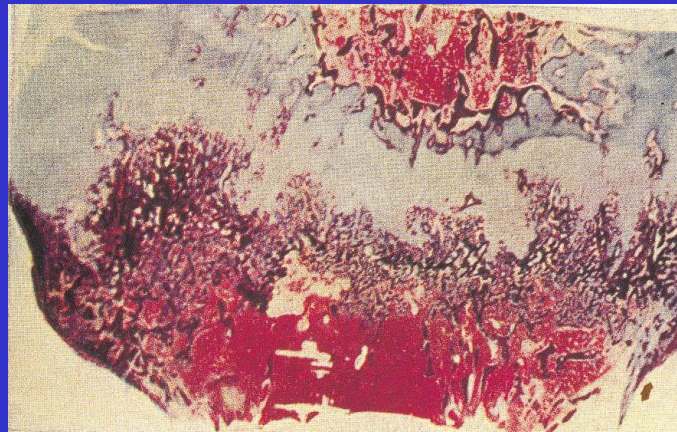
RACHITIC ROSARY



**SCOLIOSIS AND
FLATTENED
PELVIS**



**WIDENING OF STatural ZONE AND FORMATION OF
RACHITIC BRACELETS**



**STIGMATA
RACHITIS**

HYPERCALCEMIA – CALCIUM NEPHROPATHY CAUSED BY THE OVERDOSAGE OF VITAMIN D, DISTURBANCES IN EXCRETION (KIDNEY MALADY), PRIMARY HYPERPARATHYROIDISM

CALCIUM NEPHROPATHY

ETIOLOGIC OR ASSOCIATED CONDITIONS

HYPERVITAMINOSIS D



MILK-ALKALI
SYNDROME



HYPERPARATHYROIDISM

HYPERTHYROIDISM

SARCOIDOSIS

MALIGNANCIES,
WITH OR
WITHOUT
BONE
INVOLVEMENT

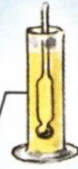
BONE DISSOLUTION;
DISUSE ATROPHY;
PAGET'S DISEASE

IDIOPATHIC
(IN INFANTS)

F. Netter
M.D.
© CIBA

CLINICAL FINDINGS

DECREASED URINARY
CONCENTRATING ABILITY



↓
POLYURIA
POLYDIPSIA

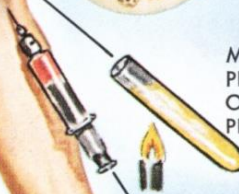
INCREASED SODIUM AND
CHLORIDE EXCRETION

(POTASSIUM REABSORPTION
AND ACID EXCRETION MAY
BE IMPAIRED)

HEMATURIA
AND PYURIA
MAY OCCUR

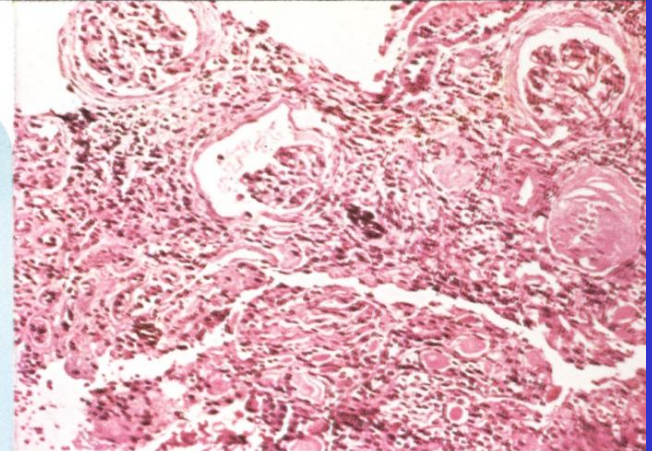


MILD
PROTEINURIA
OFTEN
PRESENT

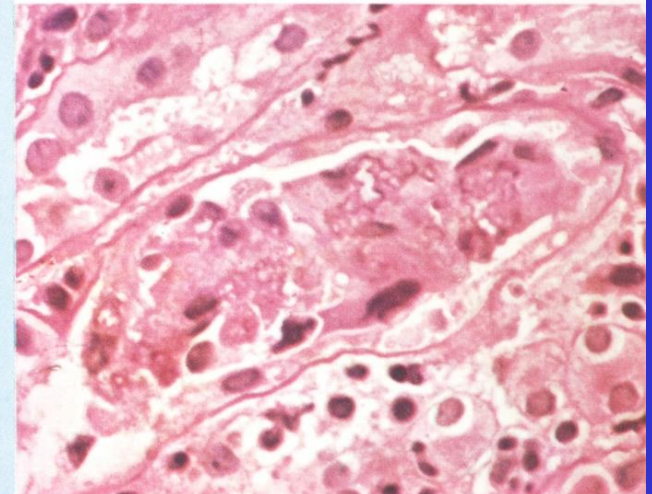


IN SEVERE CASES,
AZOTEMIA

↓
ANOREXIA
VOMITING
STUPOR
COMA



CALCIUM NEPHROPATHY: PERIGLOMERULAR FIBROSIS AND VARYING DEGREES OF GLOMERULAR HYALINIZATION MULTIFOCAL CALCIUM DEPOSITS; INTRATUBULAR PROTEIN MATERIAL SIMULATING THYROIDIZATION OF PYELO-NEPHRITIS (H. AND E. STAIN, X 100)

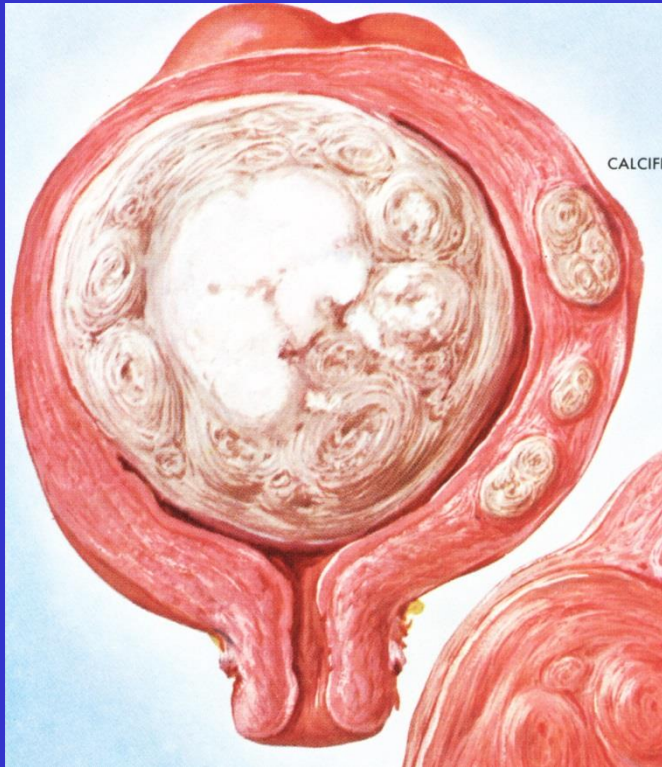


RENAL TUBULE SHOWING INTRALUMINAL ACCUMULATION OF CALCIUM AND CELLULAR DEBRIS (PAS STAIN, X 400, ENLARGED)

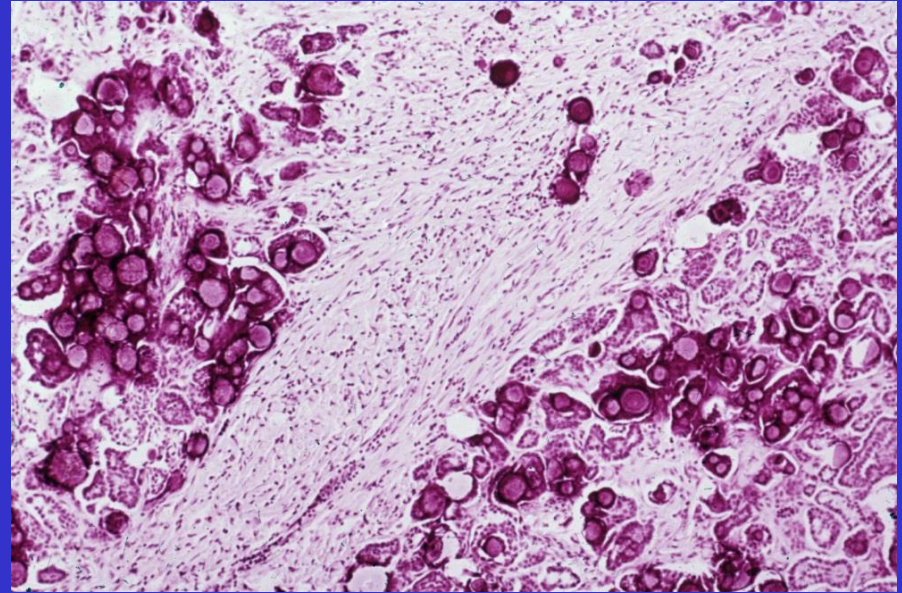
CALCIUM NEPHROPATHY

- Clinical features of macroscopic nephrocalcinosis (the form most commonly seen) may include the following:
 - Renal colic, Hematuria, Passage of urinary stones, Urinary tract infection, Polyuria and polydipsia
 - Hypertension, Proteinuria
 - In Dent disease, loss of low-molecular-weight proteins, hypercalciuria, and nephrolithiasis
 - Microscopic pyuria
 - Renal failure

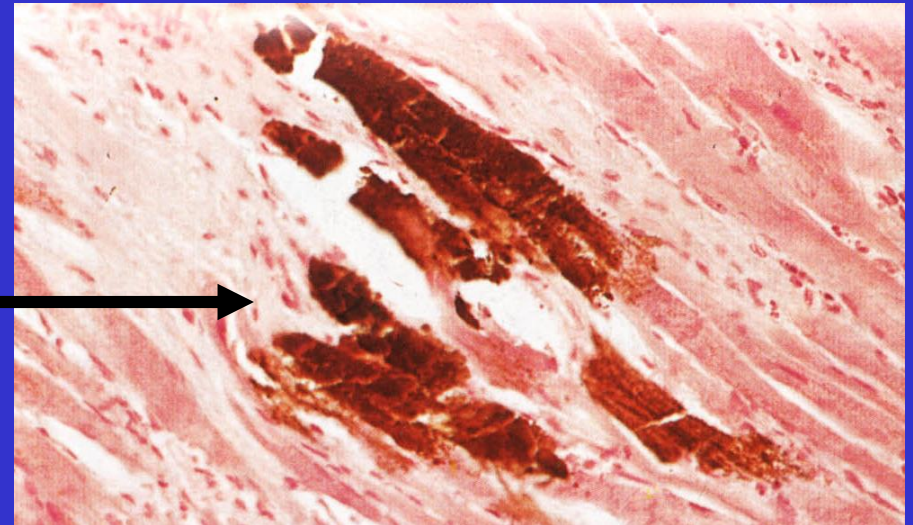
DISTURBANCES IN MINERAL BALANCE



**CALCIFICATION IN:
A. UTERINE LEIOMYOMA**



IN B. SEROUS OVARIAN CANCER



CALCIFICATION IN CARDIAC MUSCLE

**DYSTROPHIC
CALCIFICATION**

DISTURBANCES IN MINERAL BALANCE

CALCULOSIS - LITHIASIS

CHOLELITHIASIS

**HEPATOLITHIASIS, CHOLEDOCHOLITHIASIS,
CHOLECYSTOLITHIASIS**

UROLITHIASIS

**NEPHROLITHIASIS, URETEROLITHIASIS, UROCYSTOLITHIASIS,
URETHROLITHIASIS**

SIALOLITHIASIS

PANCREOLITHIASIS

PNEUMOLITHIASIS

ARTERIOLITHS, PHLEBOLITHS

LITHOPEDION (PETRIFIED FETUS)

LITHIASES - CALCULOSIS



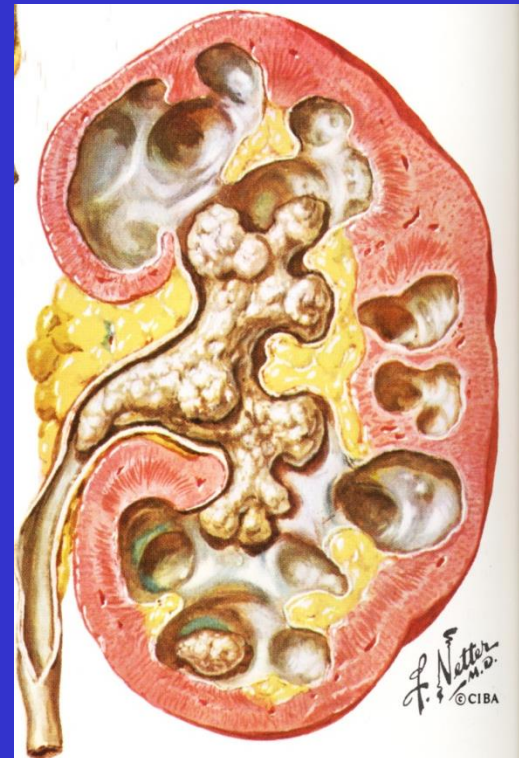
GALL STONES



PANCREOLITHIASIS

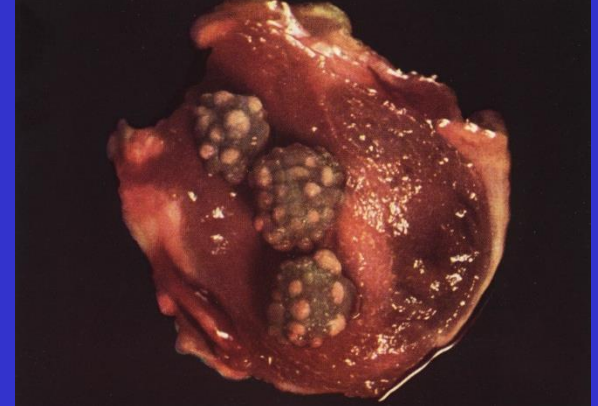
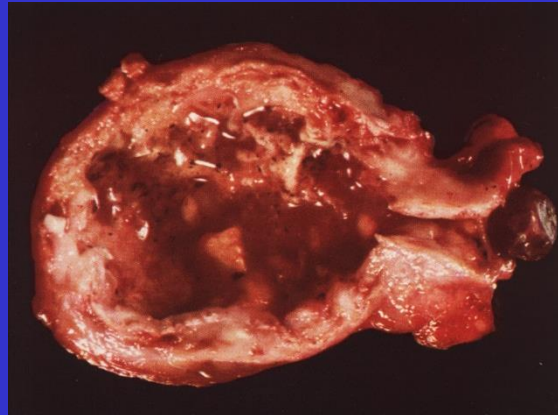
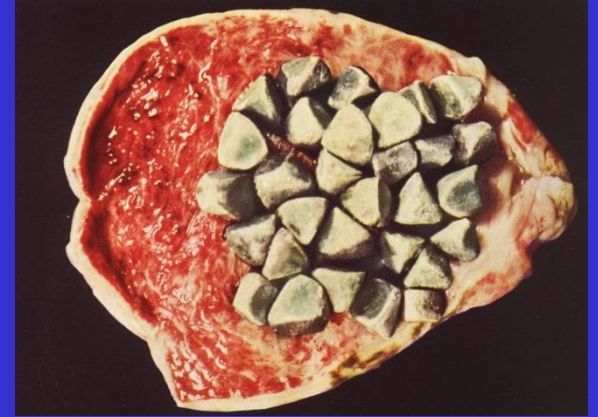
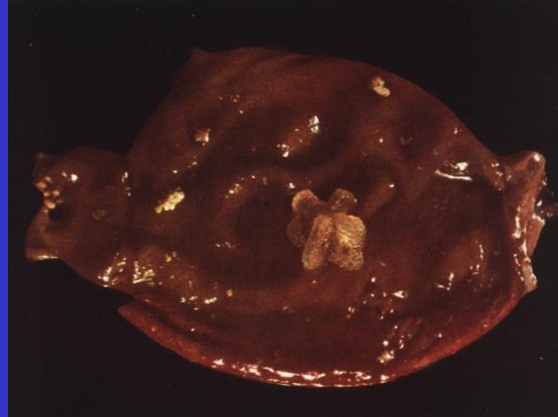
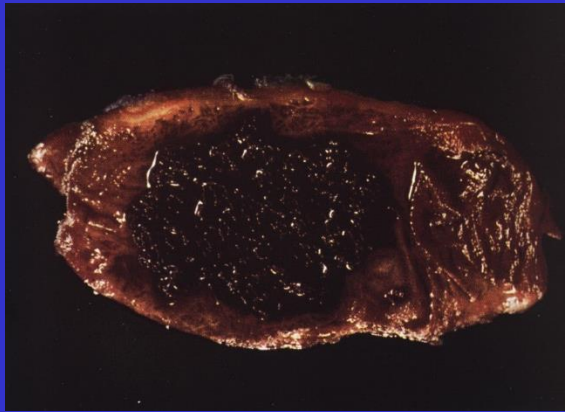
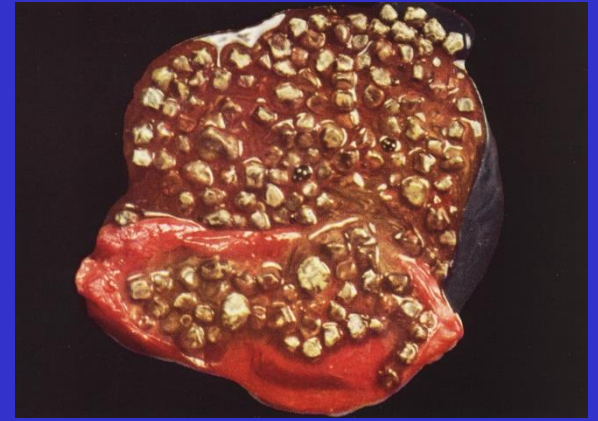


SIALOLITHIASIS



NEPHROLITHIASIS

CHOLECYSTOLITHIASIS (STONES)





REX ET DOMINA

