

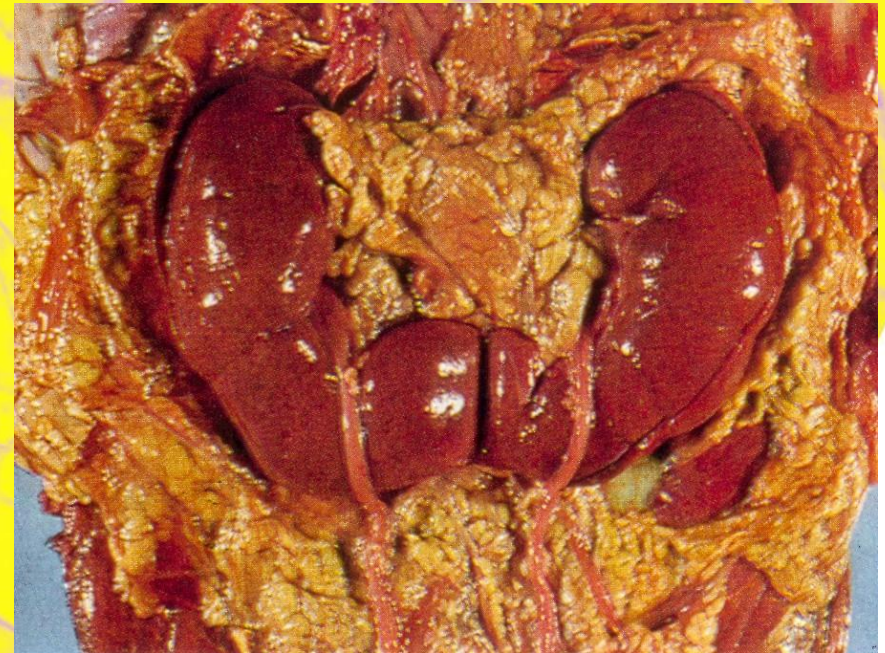
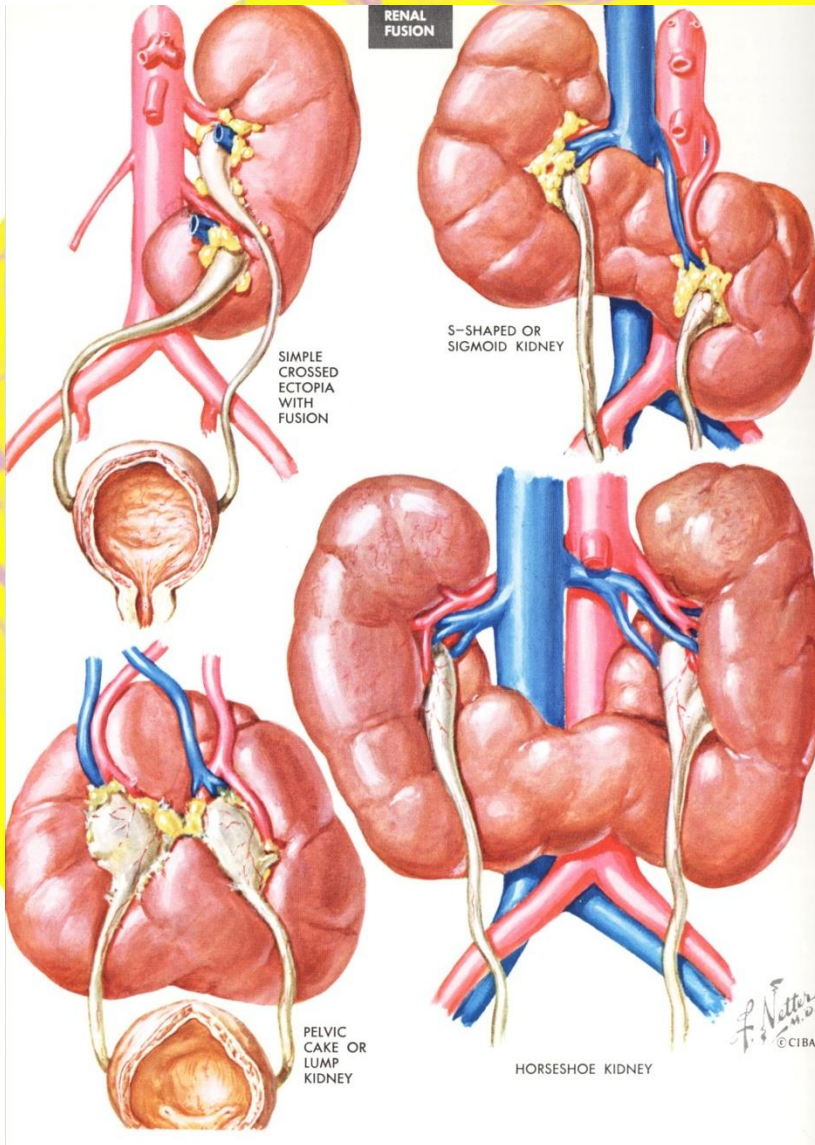
LECTURE 17



MARCELLO MALPIGHI
(1628-1694)

DEVELOPMENTAL DISTURBANCES

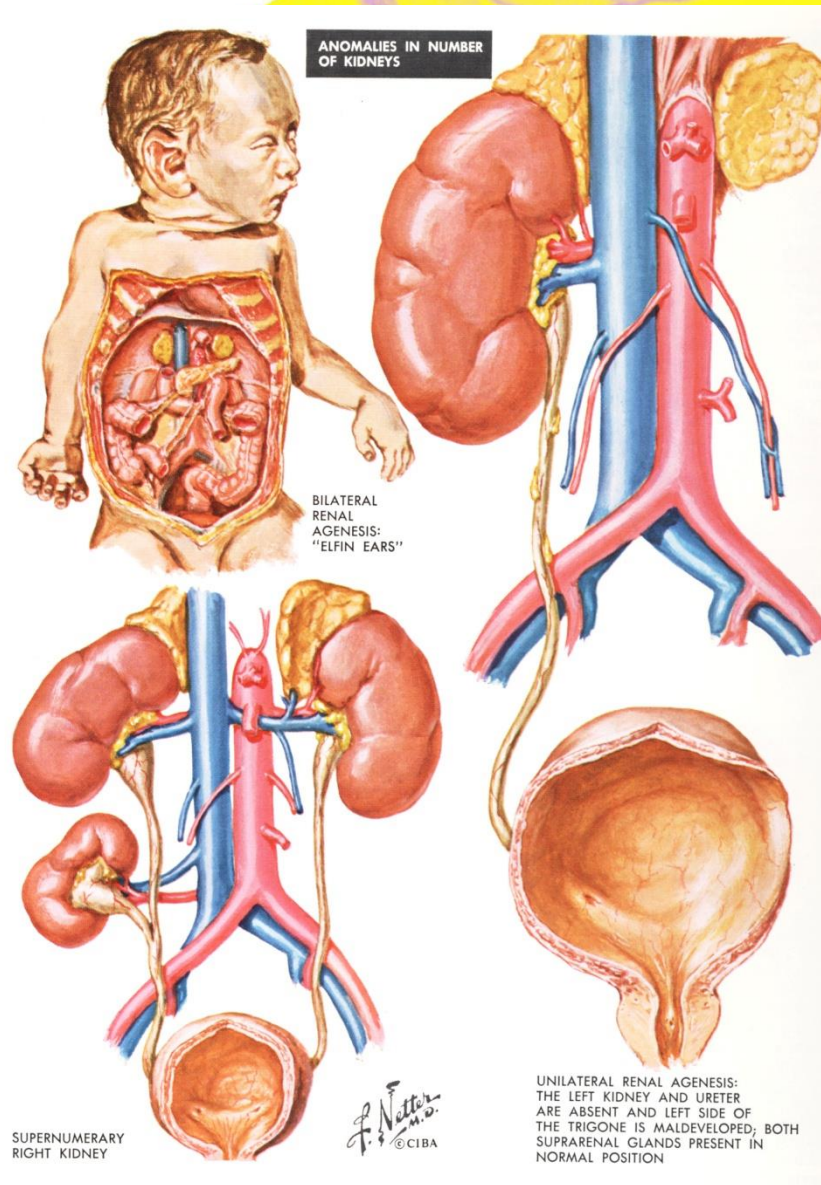
ARCUATED KIDNEY



DEVELOPMENTAL DISTURBANCES

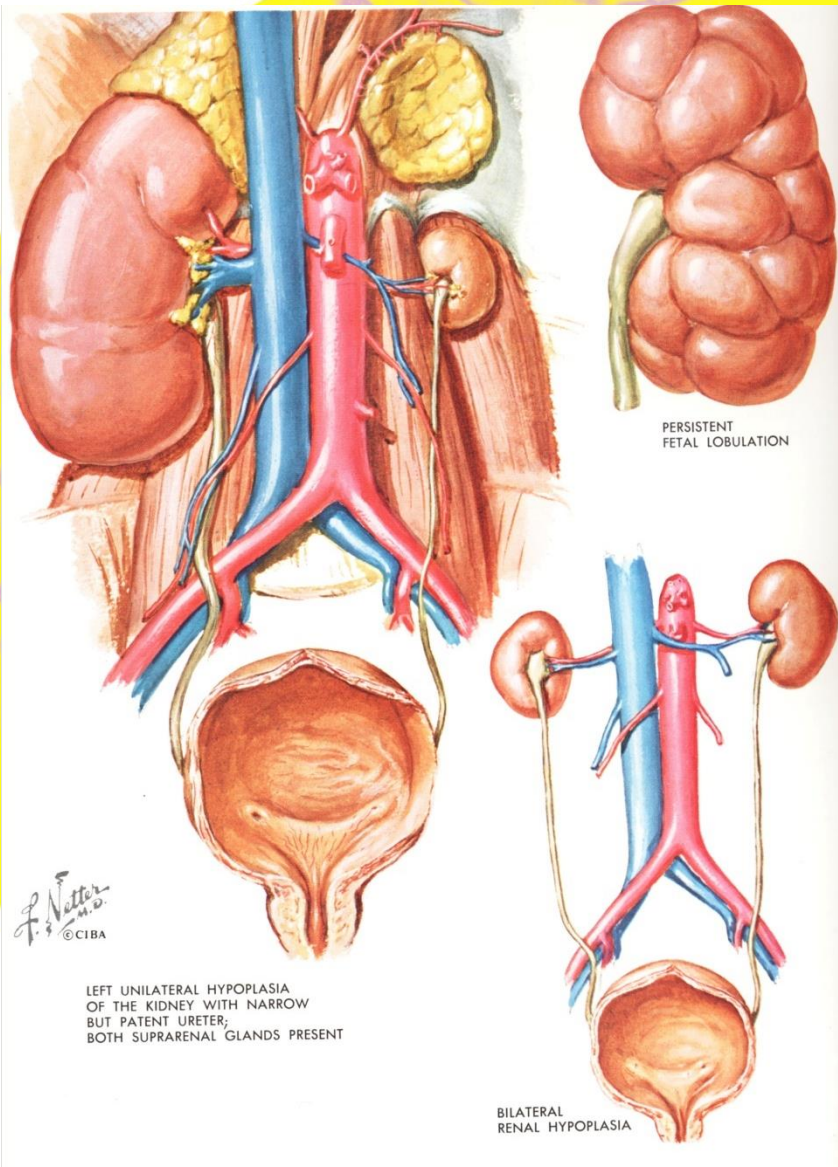
CONGENITAL LACK OF KIDNEY(S)

AGENESIA, APLASIA



DYSTOPIA OF KIDNEY (TRANSLOCATION) PELVIC RENAL DYSTOPIA

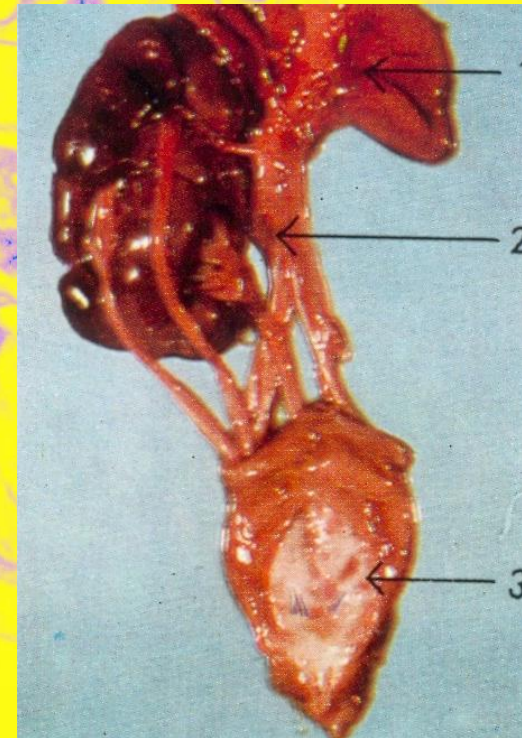
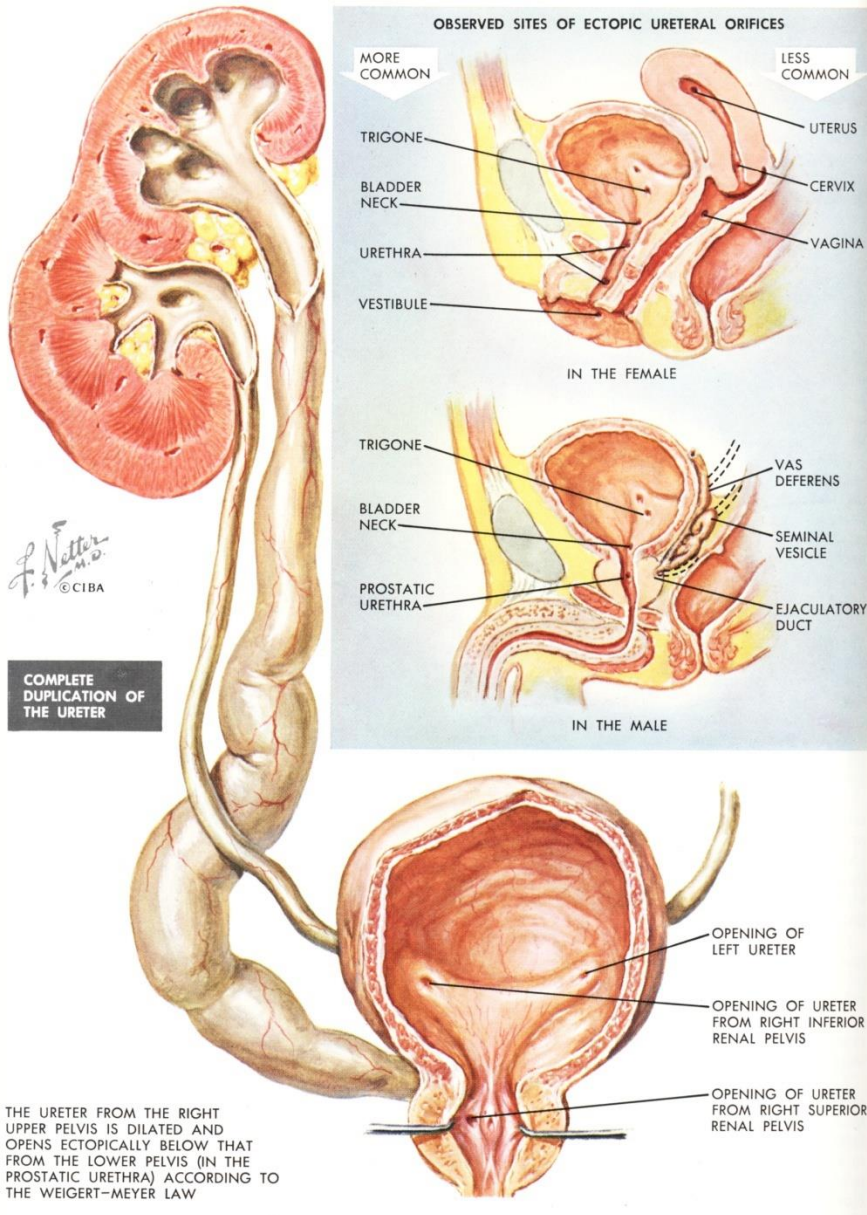
DEVELOPMENTAL DISTURBANCES



HYPOPLASIA

DEVELOPMENTAL DISTURBANCES

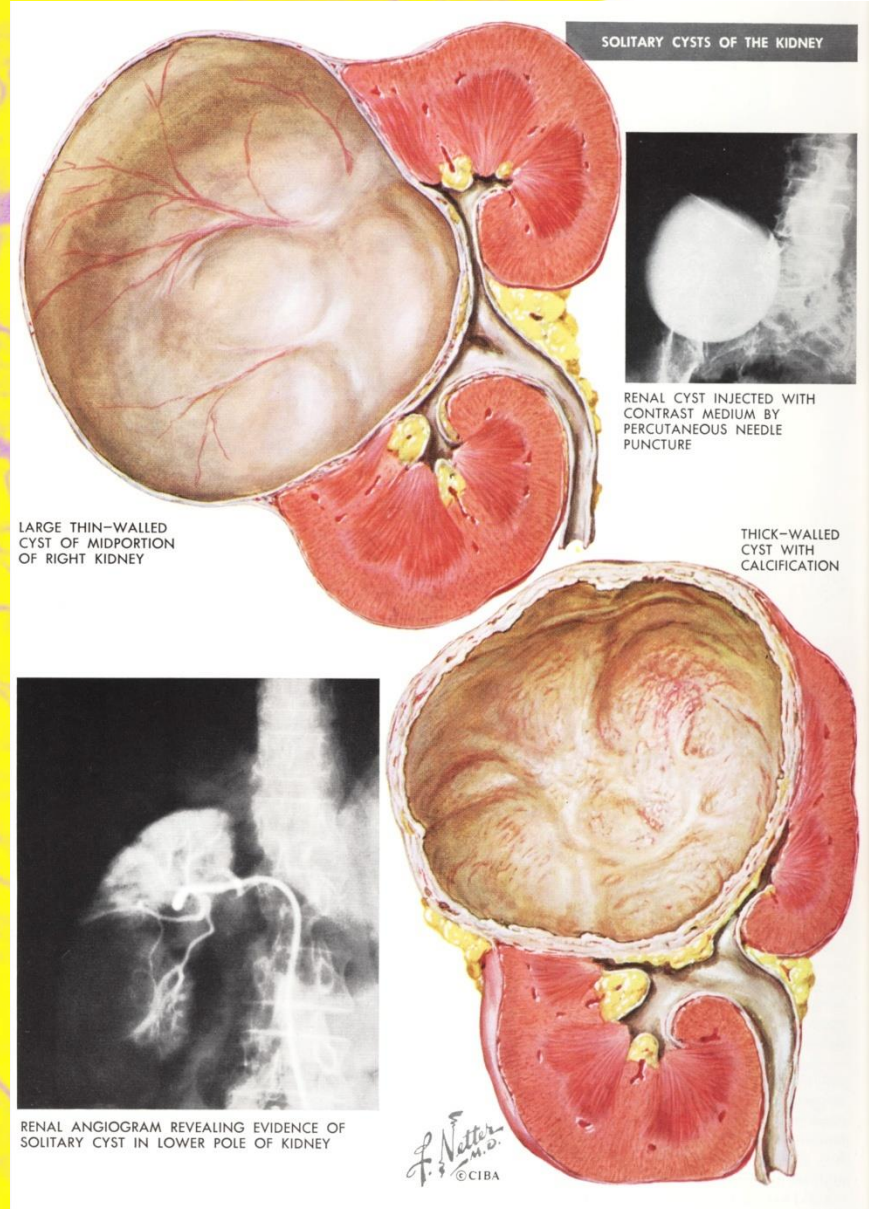
DUPLICATED PYELON AND URETER



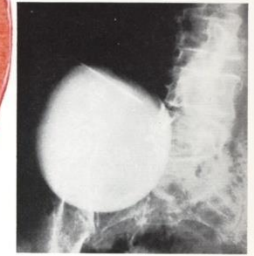
SOLITARY CYSTS



SIMPLE CYST



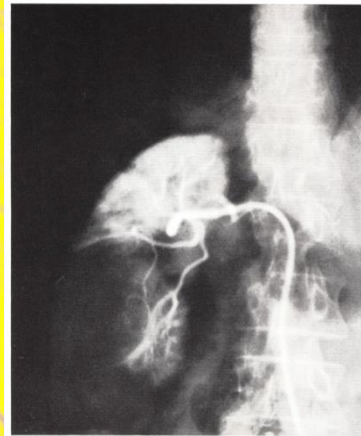
SOLITARY CYSTS OF THE KIDNEY



RENAL CYST INJECTED WITH CONTRAST MEDIUM BY PERCUTANEOUS NEEDLE PUNCTURE

LARGE THIN-WALLED CYST OF MIDPORTION OF RIGHT KIDNEY

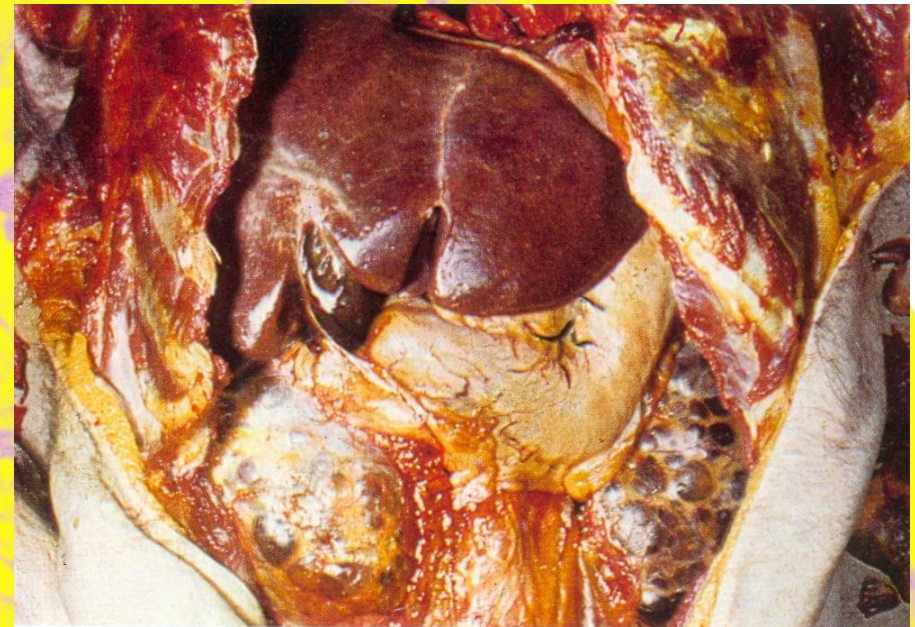
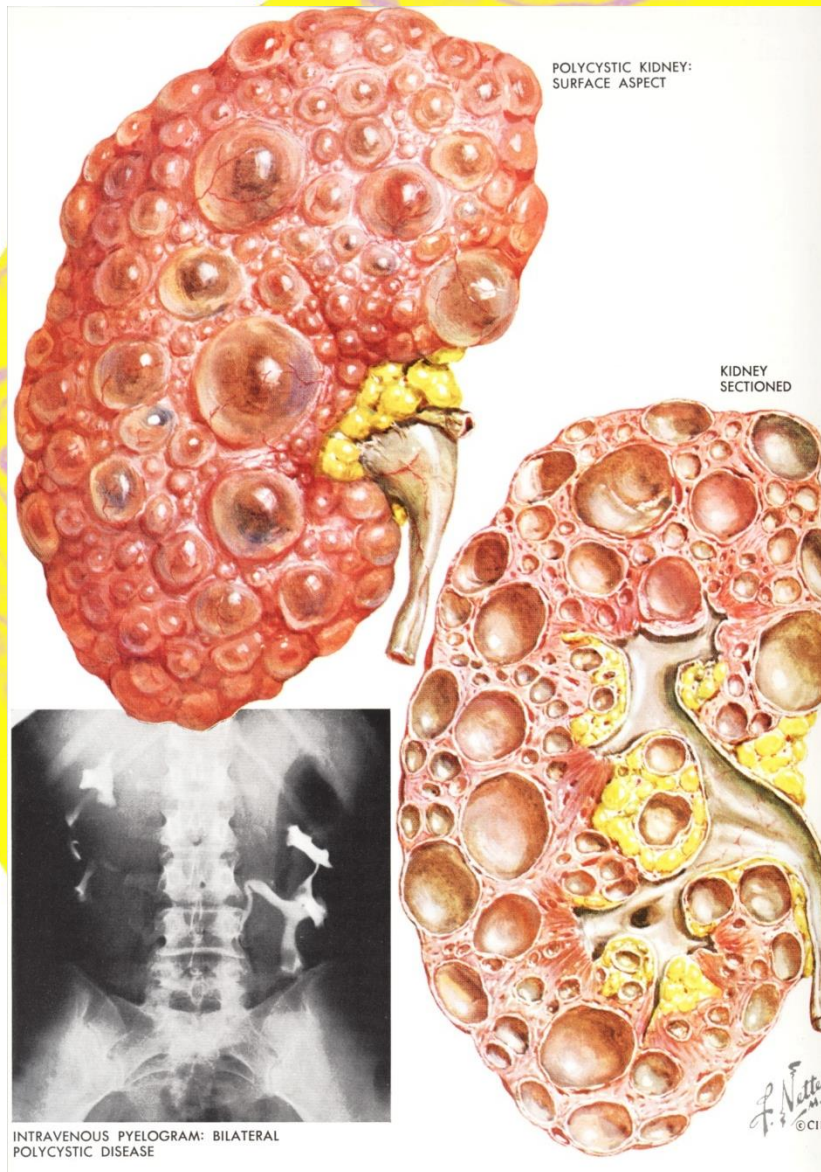
THICK-WALLED CYST WITH CALCIFICATION



RENAL ANGIOGRAM REVEALING EVIDENCE OF SOLITARY CYST IN LOWER POLE OF KIDNEY

F. Netter
M.D.
© CIBA

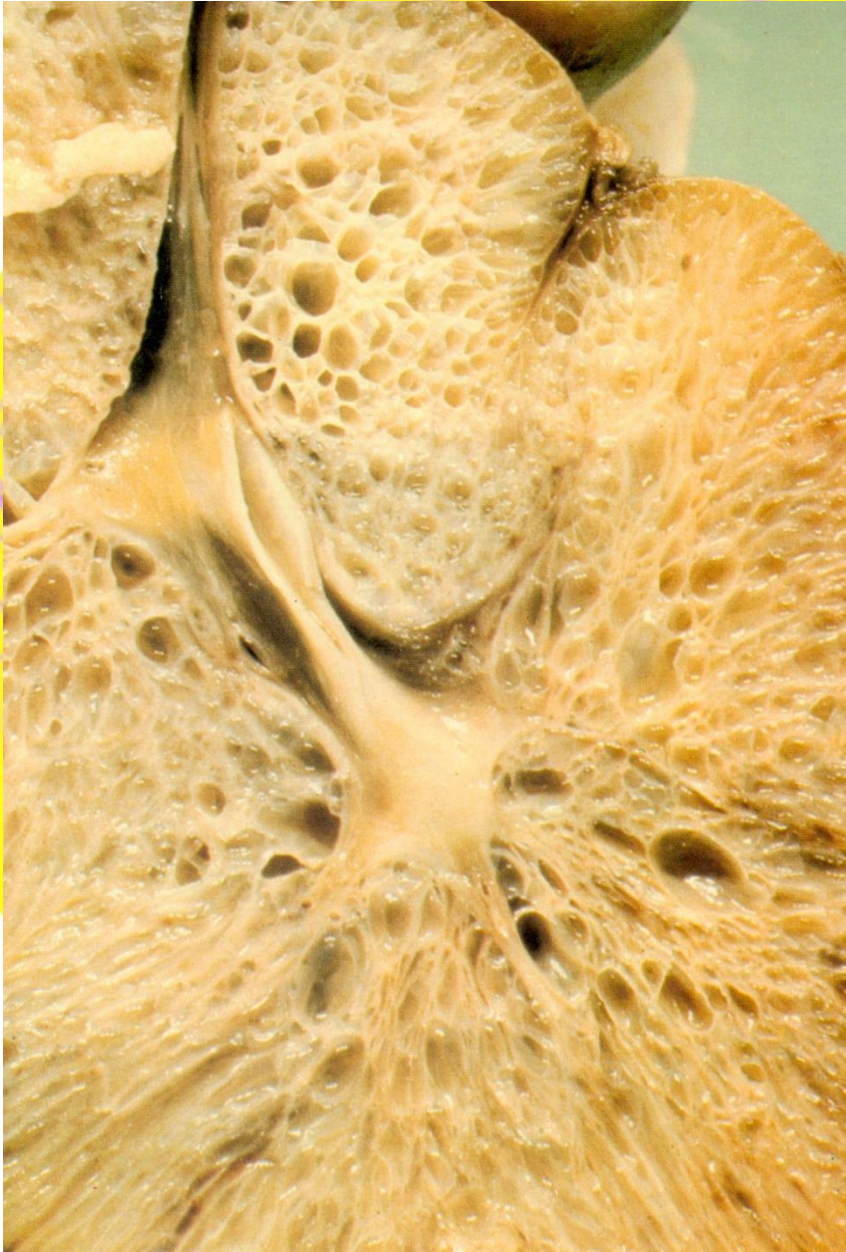
DEVELOPMENTAL DISTURBANCES



**POLYCYSTIC KIDNEY
(*RENES MACROPOLYCYSTICI*)**
TRIAD OF SYMPTOMS:
**SYMMETRIC TUMOR IN ABDOMEN,
HYPERTENSION, INFLAMMATION OF
RENAL PELVIS - PYELITIS**

DEVELOPMENTAL DISTURBANCES

MICROPOLYCYSTIC KIDNEYS



GENETIC DEFECT IN THE SHORT ARM OF CHROMOSOME 6 (RECESSIVE IN NEWBORNS AND DOMINANT IN ADULTS). DIFFERENT PATHOMORPHOLOGIC TYPES, ALSO WITH CYSTIC CHANGES IN LIVER

A circular inset showing a histological section of tissue. The tissue is stained with hematoxylin and eosin (H&E). The background is a pale pink, representing the extracellular matrix and connective tissue. Numerous small, dark purple-blue spots are scattered throughout, representing the nuclei of various cells, including inflammatory cells like leukocytes. Some larger, more complex structures are visible, possibly representing glandular or ductal structures. The overall appearance is one of active inflammation.

INFLAMMATIONS

NEPHRITIS

GLOMERULONEPHRITIS

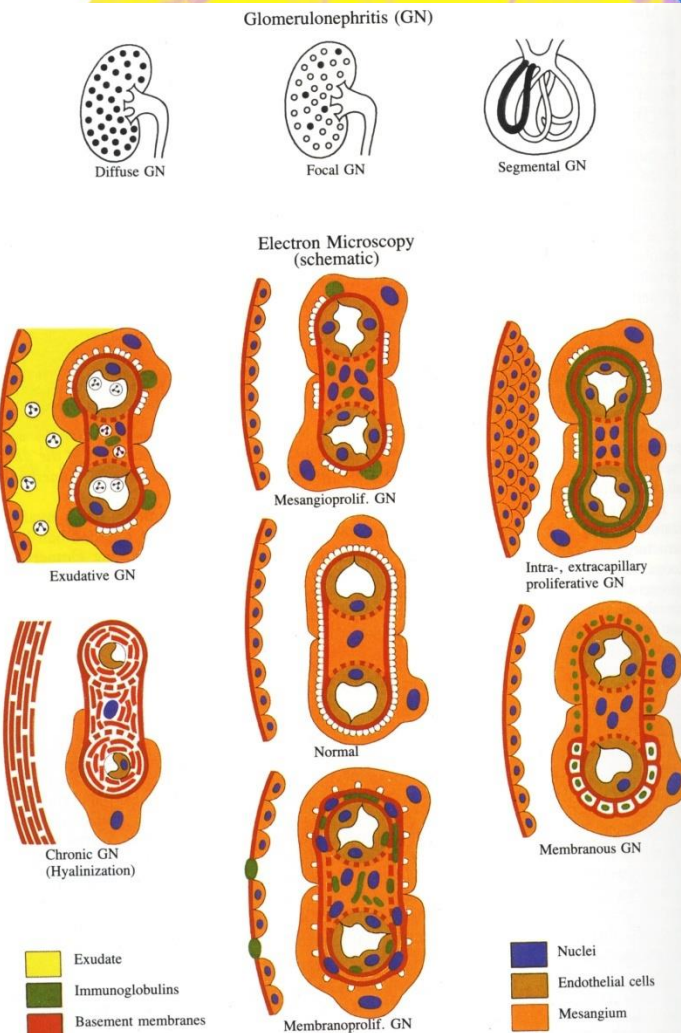


Fig 6-22.—Different forms of glomerulonephritis; electron microscopic features according to Bohle.

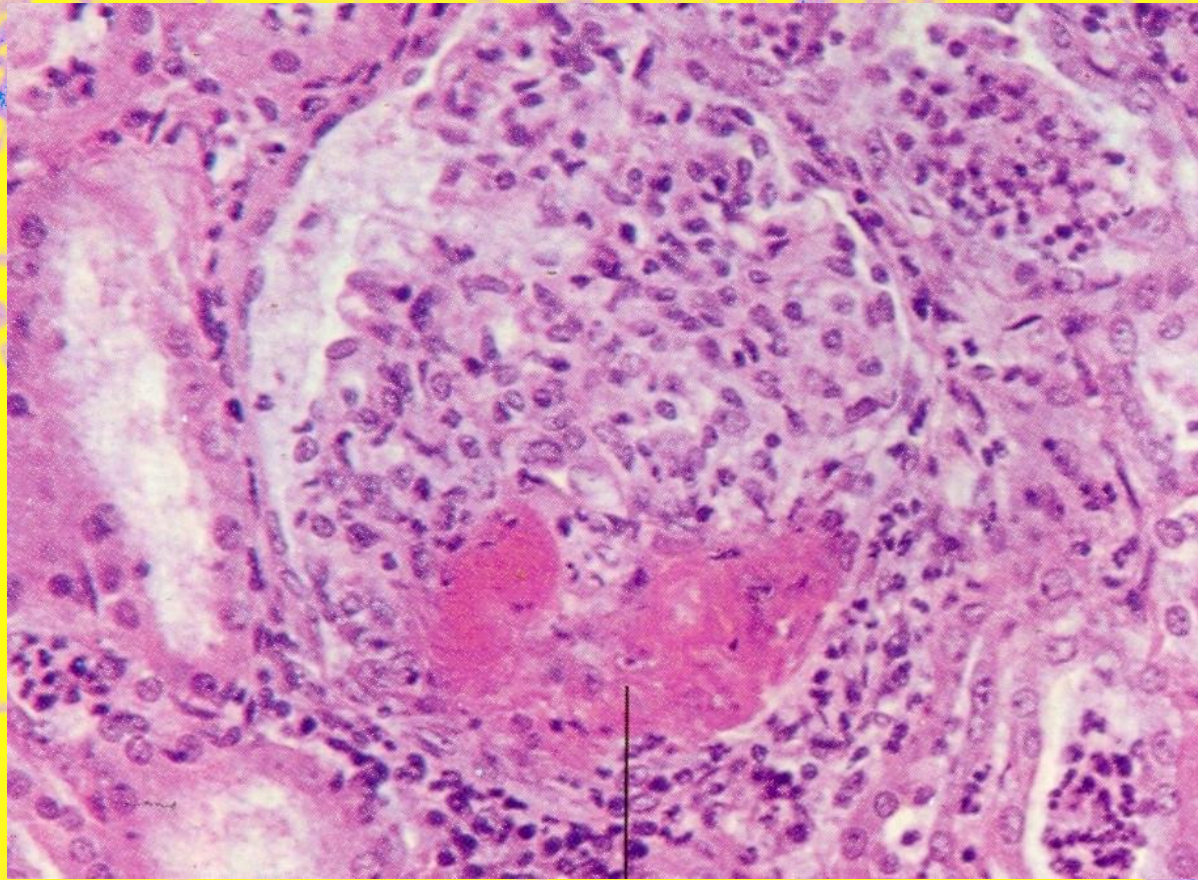
GLOMERULONEPHRITIS

FOCAL GN

DIFFUSE GN

NEPHRITIS

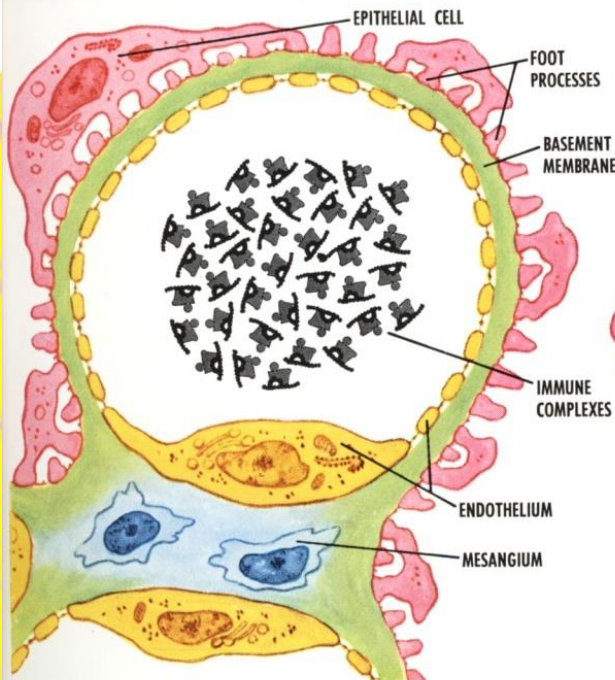
FOCAL GLOMERULONEPHRITIS



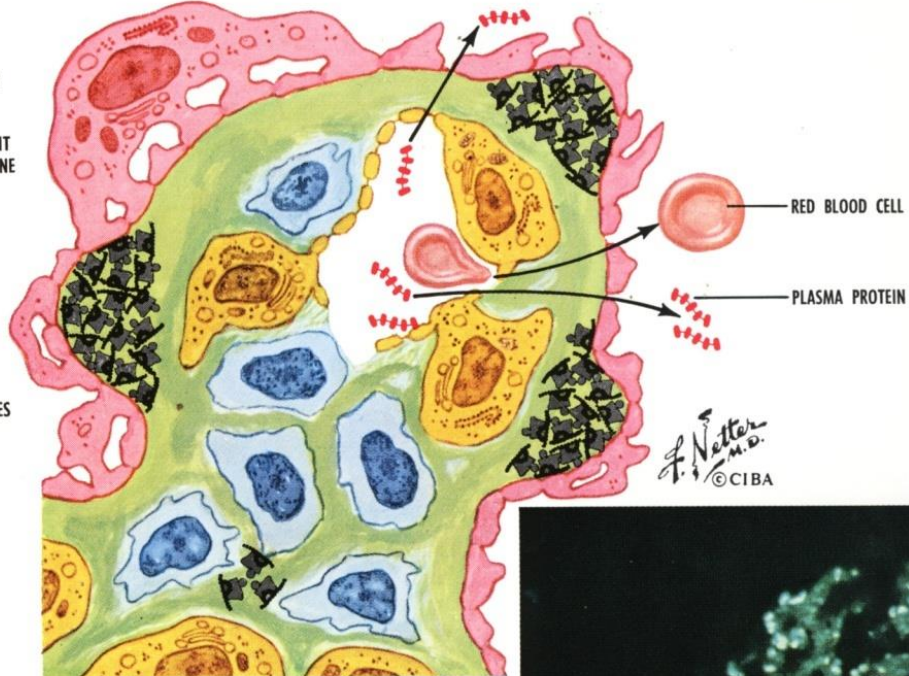
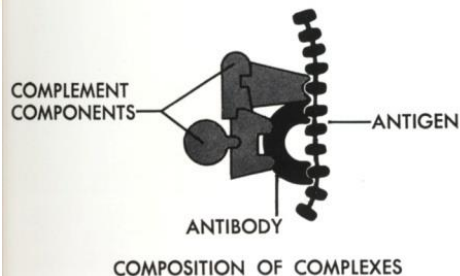
NEPHRITIS

GLOMERULONEPHRITIS

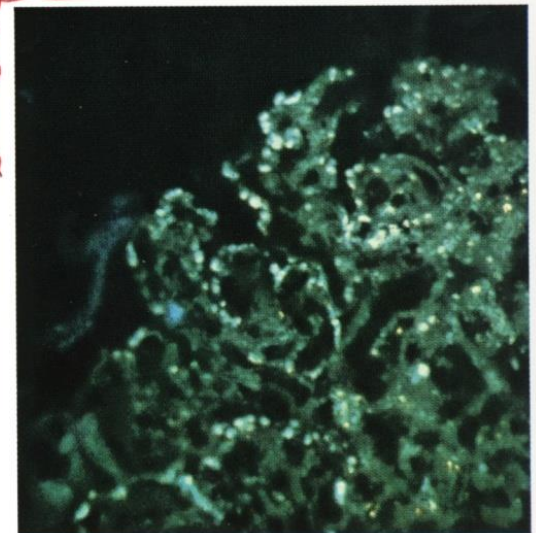
HYPOTHESIS OF PATHOGENESIS OF ACUTE GLOMERULAR INJURY BY CIRCULATING IMMUNE COMPLEXES (SCHEMATIC)



A: CIRCULATING IMMUNE COMPLEXES, FORMED ANYWHERE IN THE BODY, CONSISTING OF ANTIGEN, ANTIBODY, AND COMPLEMENT COMPONENTS, ARRIVE AT GLOMERULAR CAPILLARIES IN **LARGE AMOUNTS** OVER A SHORT PERIOD OF TIME



B: COMPLEXES PENETRATE ENDOTHELIUM AND BASEMENT MEMBRANE OF GLOMERULAR CAPILLARIES AND FORM LARGE ISOLATED DEPOSITS (HUMPS); FOOT PROCESSES FUSE; MESANGIAL AND ENDOTHELIAL CELLS SWELL AND PROLIFERATE, INVADING CAPILLARY LUMEN; FIBRILLAR BASEMENT MEMBRANELIKE MATERIAL (MESANGIAL MATRIX) IS DEPOSITED BETWEEN CELLS; INCREASED POROSITY OF CAPILLARY WALLS PERMITS ESCAPE OF PLASMA PROTEINS AND BLOOD CELLS, CAUSING PROTEINURIA AND HEMATURIA

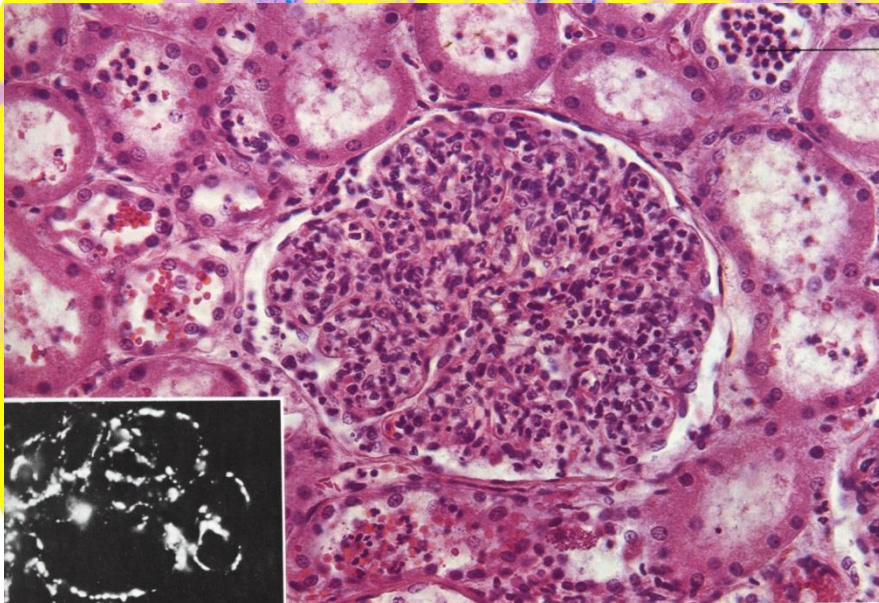


IMMUNOFLUORESCENT PREPARATION, ACUTE GLOMERULONEPHRITIS: IRREGULAR LUMPY DEPOSITS OF GAMMA GLOBULIN AND COMPLEMENT, RESEMBLING EXPERIMENTAL ACUTE IMMUNE COMPLEX DISEASE

NEPHRITIS

GLOMERULONEPHRITIS

DIFFUSE ACUTE GLOMERULONEPHRITIS



**SIGNIFICANTLY INCREASED NUMBER
OF NUCLEI IN THE GLOMERULUS,
MOSTLY BECAUSE OF NEUTROPHILS**

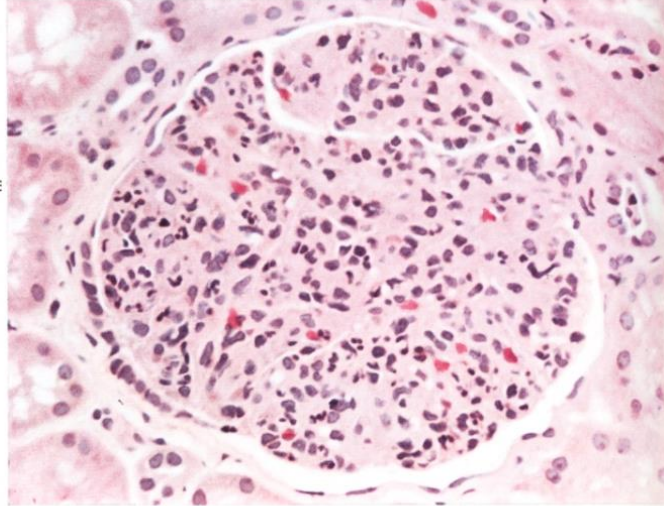


**MACROSCOPY OF ACUTE
NEPHRITIS (LARGE RED
KIDNEY)**

NEPHRITIS

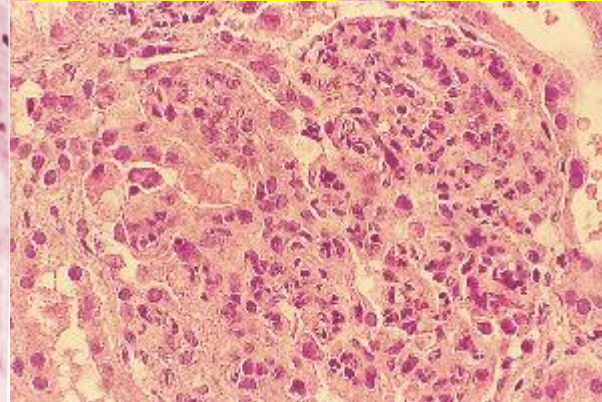
DIFFUSE GLOMERULONEPHRITIS

ACUTE GLOMERULONEPHRITIS

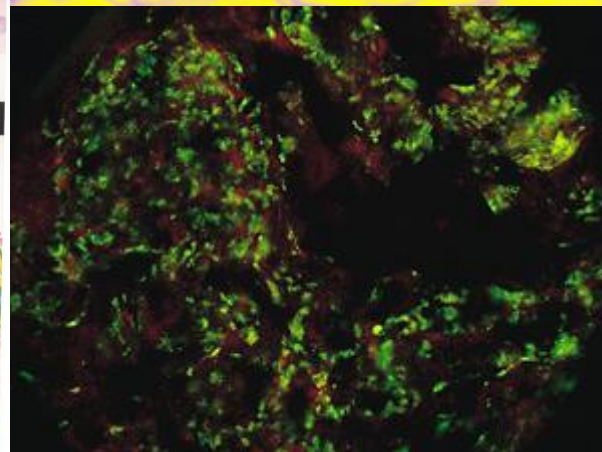


GLOMERULUS WITH GREATLY INCREASED CELLULARITY AND MESANGIAL MATRIX AND ALMOST COMPLETE OBLITERATION OF CAPILLARY LUMINA; THE CELLS ARE CHIEFLY MESANGIAL IN TYPE, WITH SOME POLYMORPHONUCLEAR LEUKOCYTES AND A FEW EOSINOPHILS

F. Netter
© CIBA

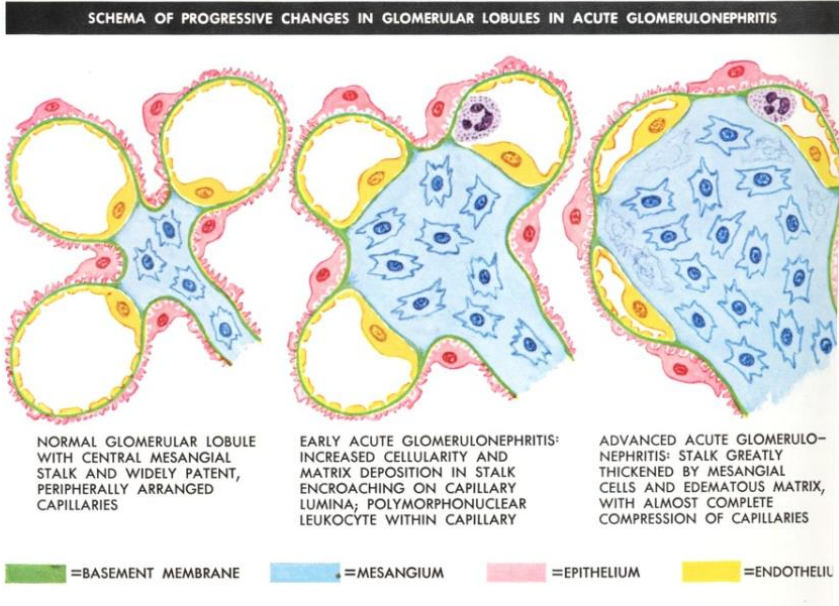


SIGNIFICANTLY INCREASED NUMBER OF NUCLEI IN GLOMERULUS, MOSTLY BECAUSE OF NEUTROPHILS



GRANULAR DEPOSITS OF ANTIGEN-ANTIBODY COMPLEX - COMPLEMENT COMPLEX

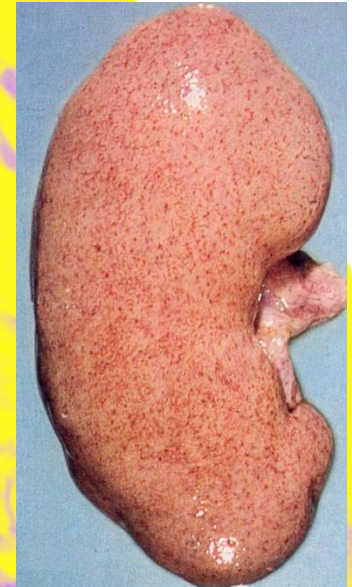
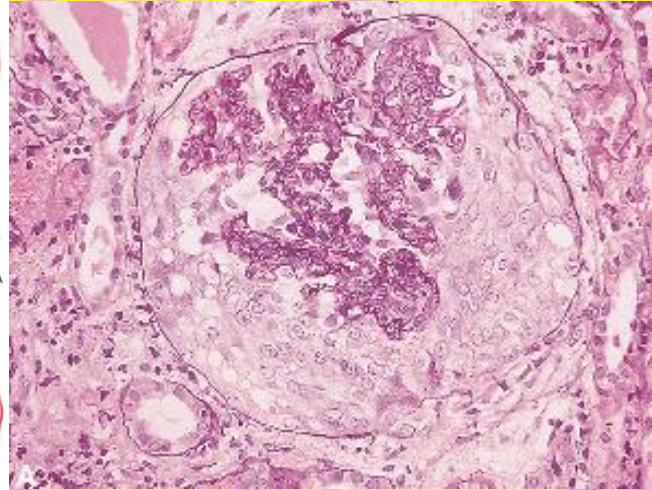
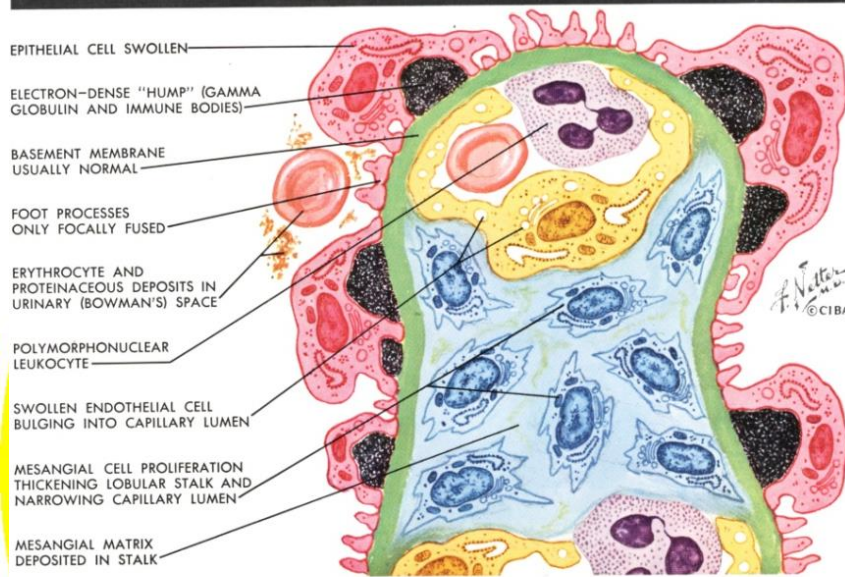
ACUTE GLOMERULONEPHRITIS (POST STREPTOCOCCAL)



NEPHRITIS

DIFFUSE GLOMERULONEPHRITIS

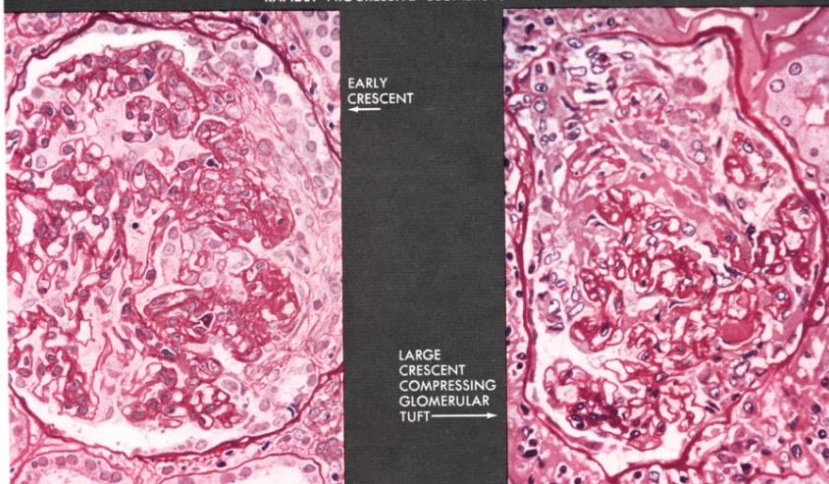
ACUTE GLOMERULONEPHRITIS: ELECTRON MICROSCOPIC FINDINGS



**CHARACTERISTIC
PROLIFERATION OF
CRESCENT PODOCYTES IN
BOWMAN'S CAPSULE**

**LARGE, YELLOW
KIDNEY**

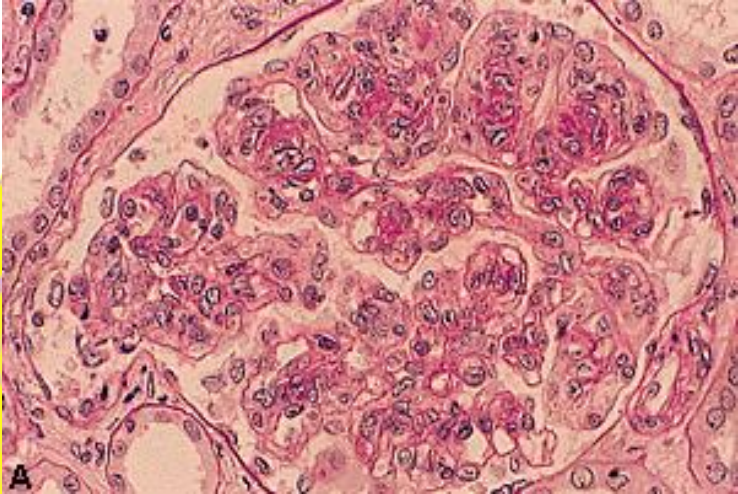
RAPIDLY PROGRESSIVE GLOMERULONEPHRITIS



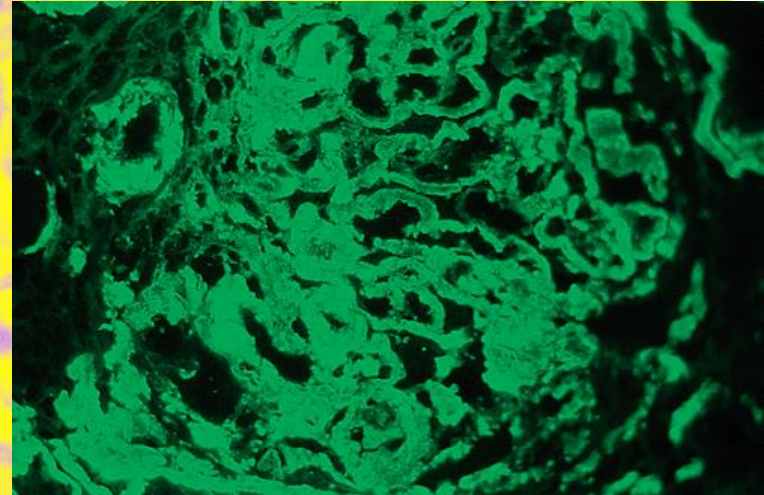
**FULMINATING
GLOMERULONEPHRITIS (RAPIDLY
PROGRESSIVE)**

NEPHRITIS

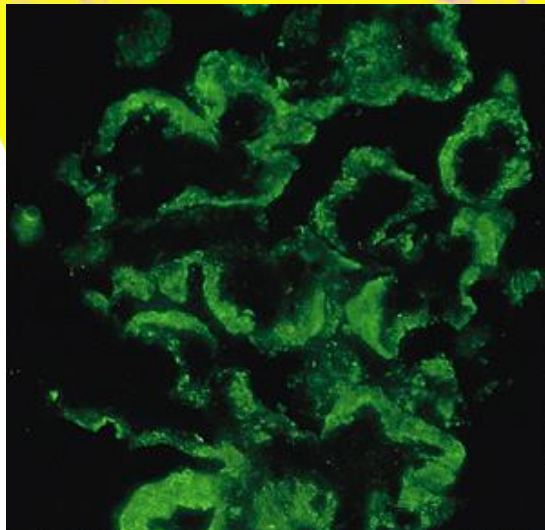
MESANGIOCAPILLAR GLOMERULONEPHRITIS



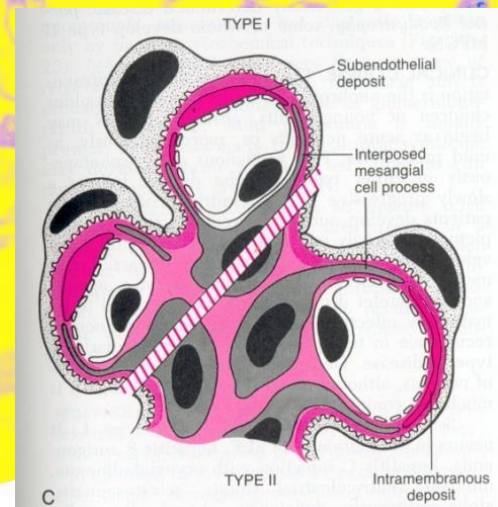
DOUBLE CONTOUR OF CAPILLARY WALLS



**MESANGIOCAPILLAR GLOMERULONEPHRITIS
TYPE II: DIFFUSE DEPOSITS IN CAPILLAR AND
MESANGIAL WALLS (ANTI- C3)**

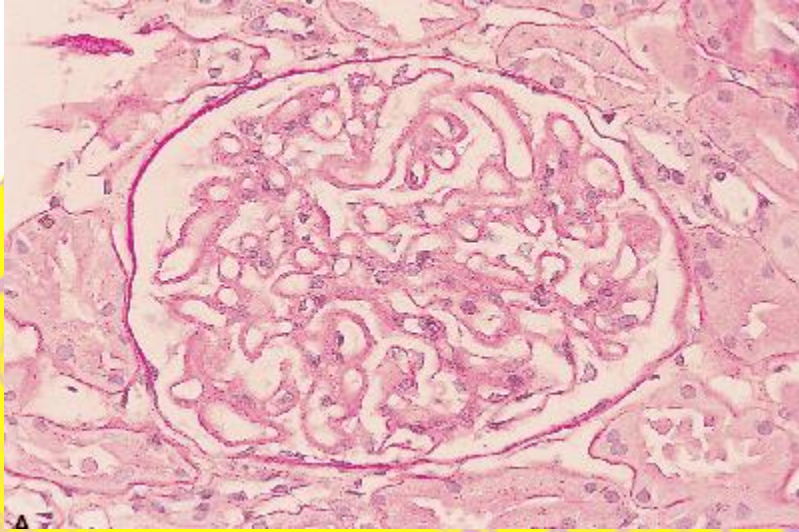


**MESANGIOCAPILLAR
GLOMERULONEPHRITIS
TYPE I: PRESENCE OF
FOCAL DEPOSITS IN
MEZANGIUM
(ANTI-C3)**

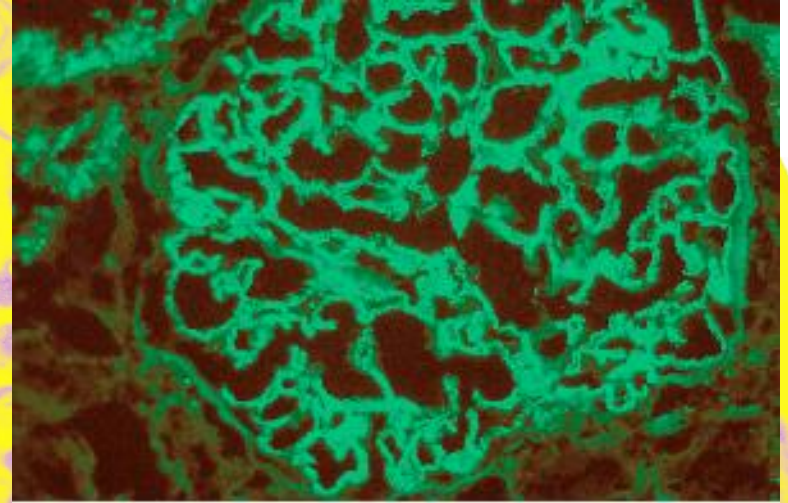


NEPHRITIS

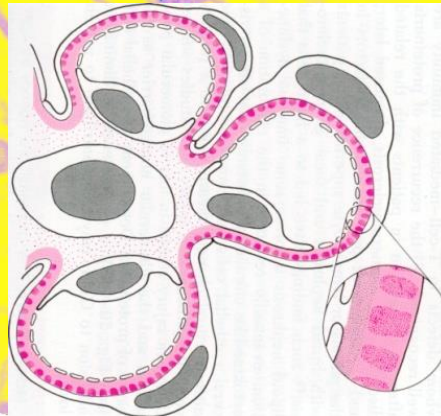
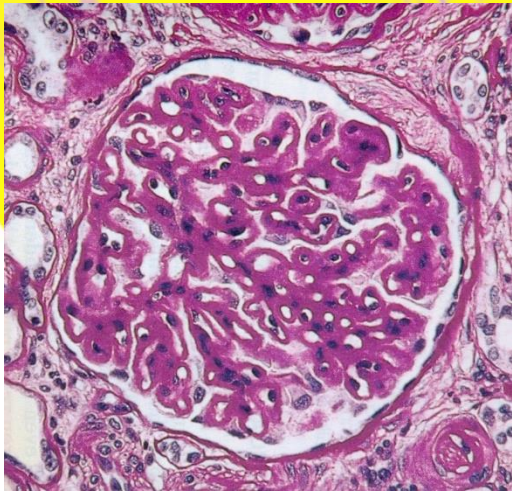
MEMBRANOUS GLOMERULONEPHRITIS



MEMBRANOUS GN – LINEAR THICKENING OF BASEMENT MEMBRANE



MEMBRANOUS GN – GRANULAR DEPOSITS IN BASEMENT MEMBRANE (THICKENING OF BASEMENT MEMBRANE, ANTI-IgG)



MEMBRANOUS GN (SCHEME)

NEPHRITIS

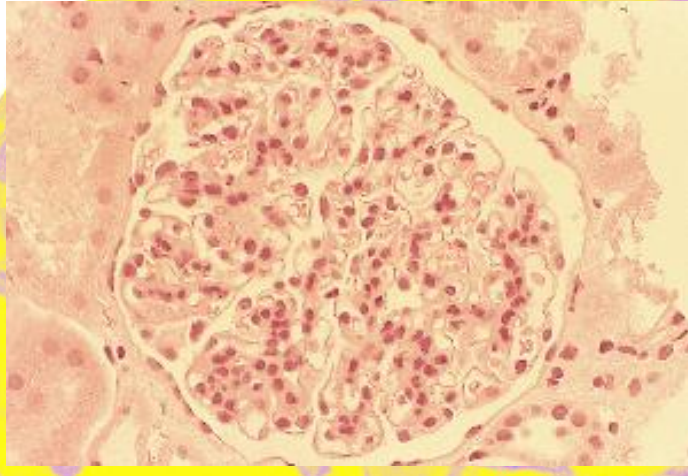
DIFFUSE GLOMERULONEPHRITIS

PATHOLOGY OF GLOMERULONEPHRITIS

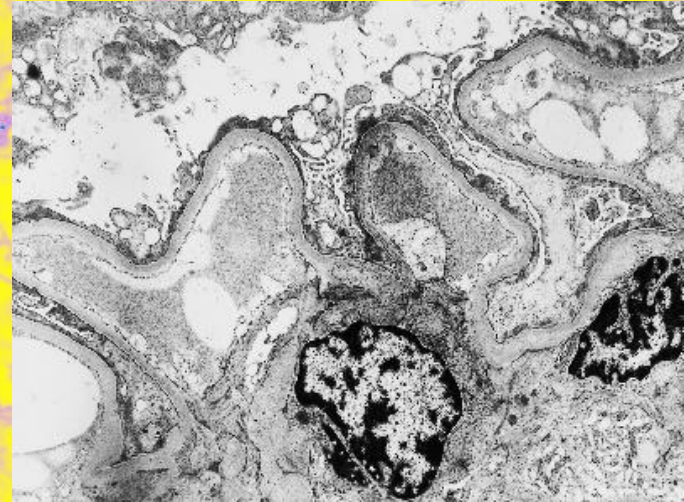
- 1. PROTEINURIA OVER 3,5 g/ 24h**
- 2. DECREASE IN PROTEIN LEVELS IN PLASMA:
EDEMA AND TRANSUDATES**
- 3. HYPERLIPIDEMIA INCLUDING INCREASE IN
CHOLESTEROL LEVELS**

NEPHRITIS

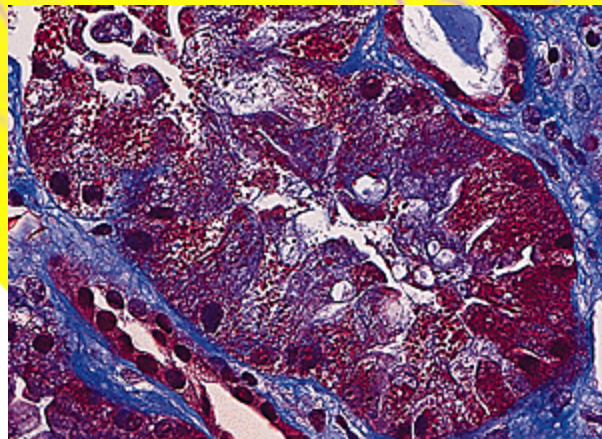
SUBMICROSCOPIC GLOMERULONEPHRITIS – MINIMAL CHANGE



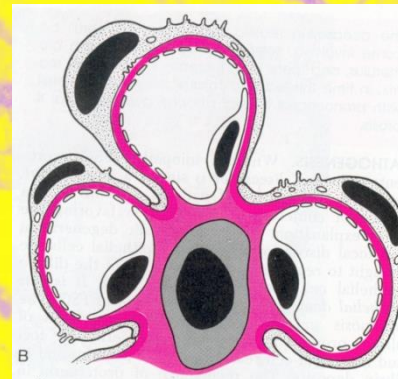
**INSIGNIFICANT INCREASE IN
MESANGIUM MATRIX**



**ELECTRON MICROSCOPY SHOWS CHANGES IN
STRUCTURE OF PODOCYTES – MERGING OF
PODOCYTE ON BASEMENT MEMBRANE**



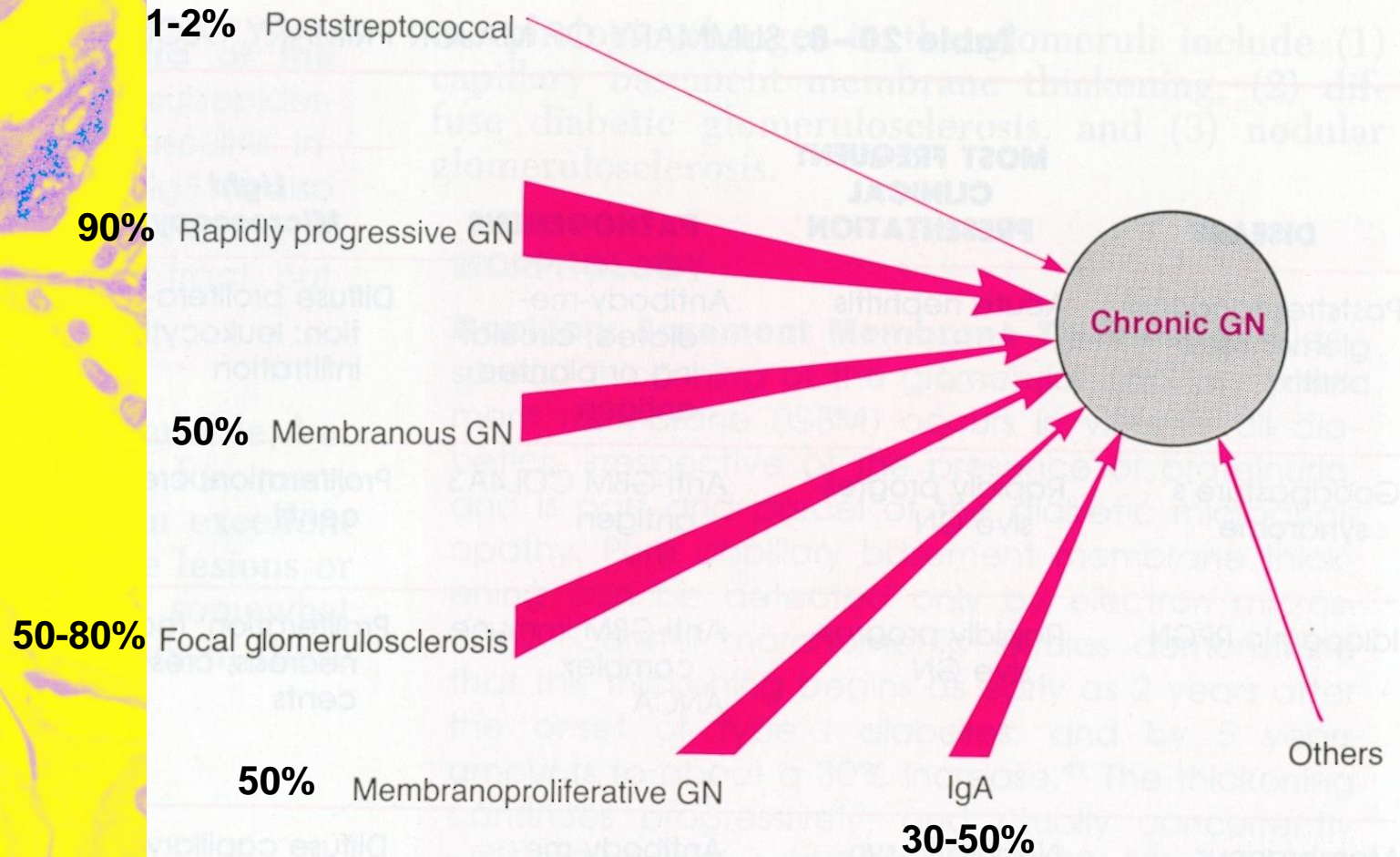
**ACCUMULATION OF LIPID DROPLETS IN
EPITHELIUM OF CANALICULI**



**SUBMICROSCOPIC
GN (SCHEME)**

NEPHRITIS

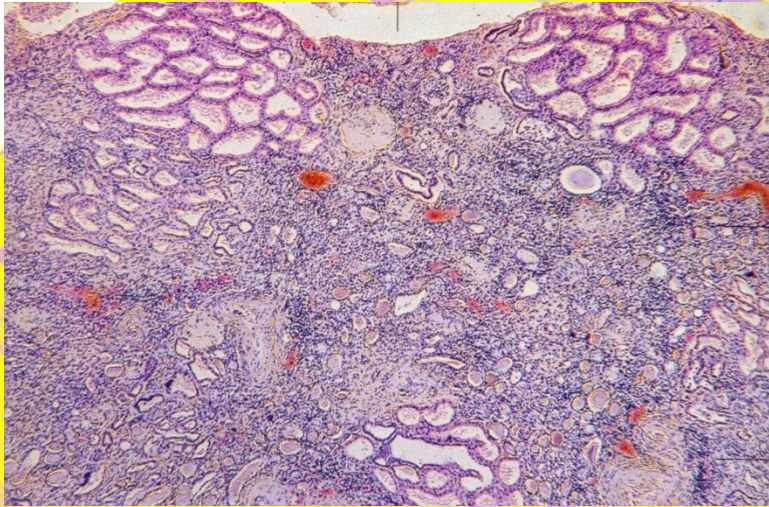
CHRONIC GLOMERULONEPHRITIS



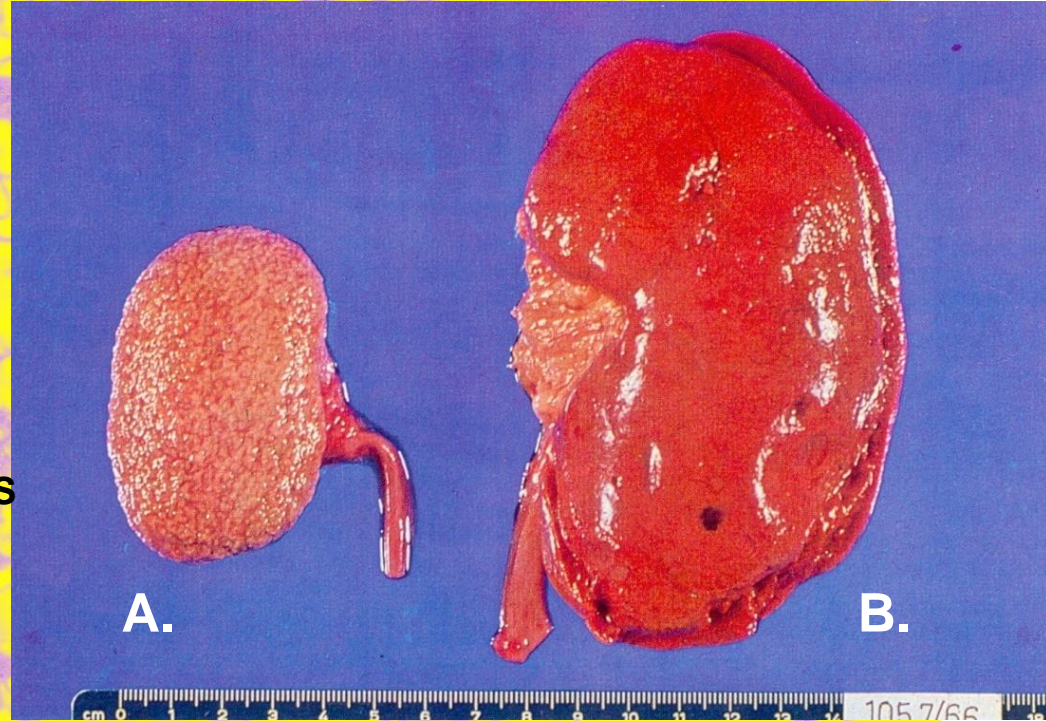
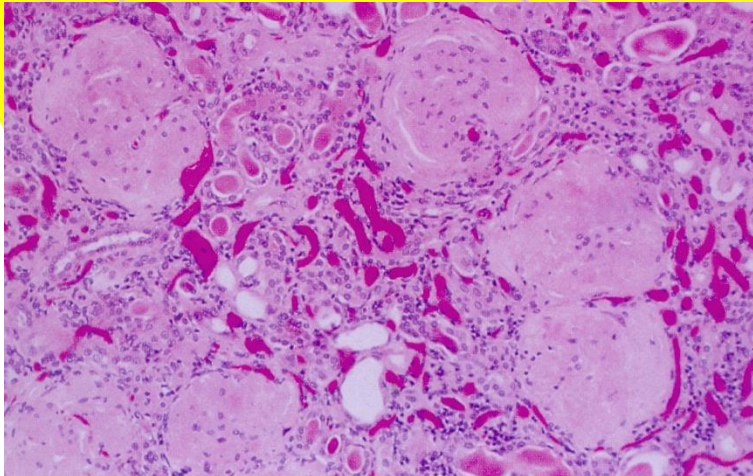
% OF GNs THAT ENDS AS A CHRONIC GN

NEPHRITIS

CHRONIC GLOMERULONEPHRITIS



**KIDNEYS IN CHRONIC GLOMERULONEPHRITIS
- MICROSCOPY**

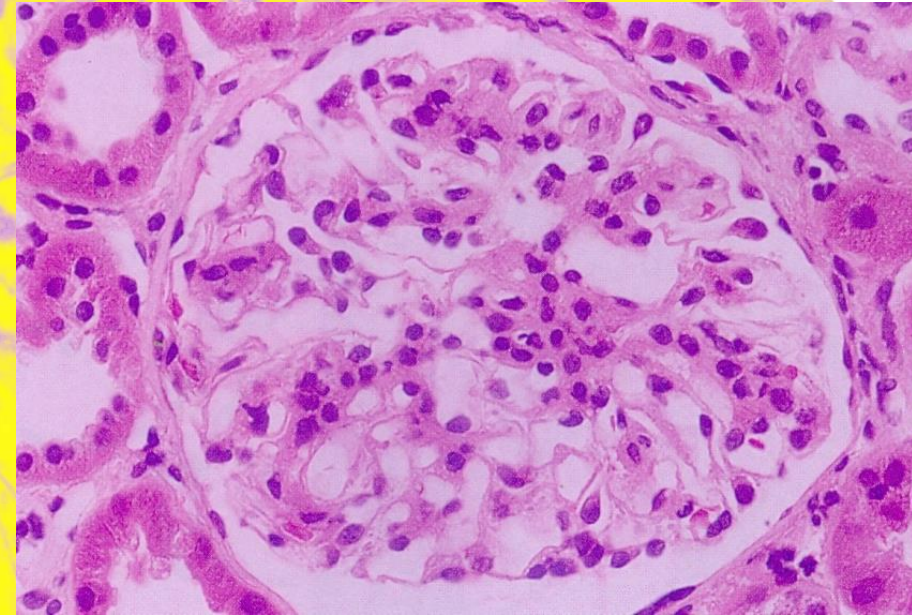
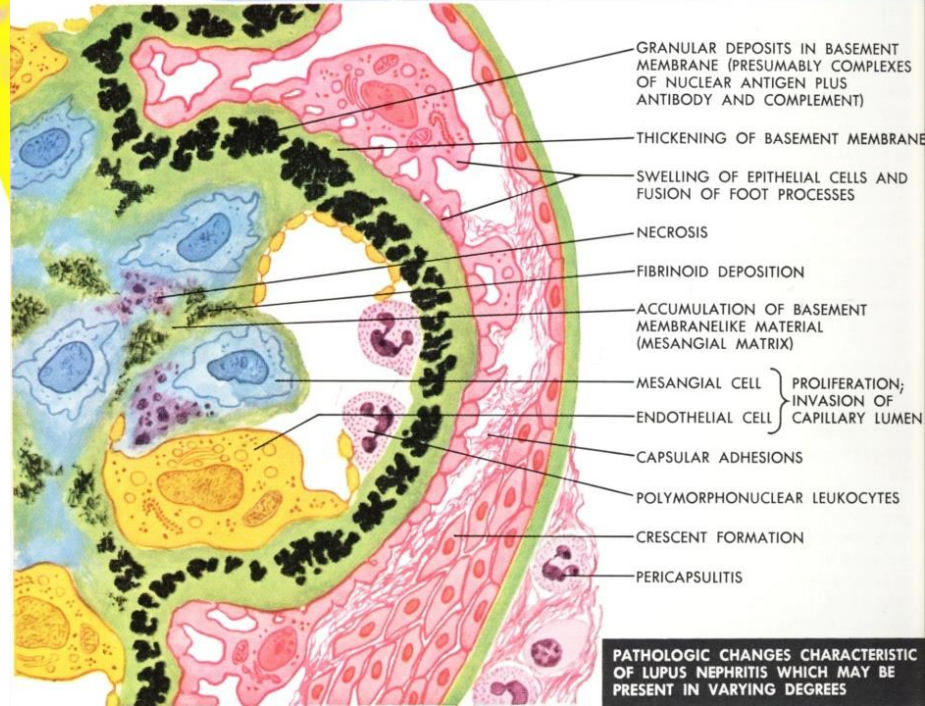
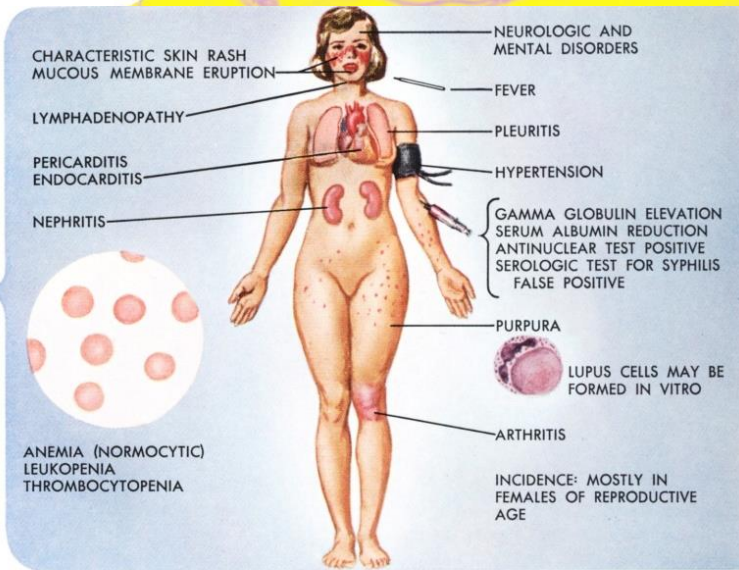


**A. KIDNEY IN CHRONIC
GLOMERULONEPHRITIS AND
B. NORMAL KIDNEY**

DAMAGE OF GLOMERULI IN SYSTEMIC DISEASES

LUPUS ERYTHEMATOSUS

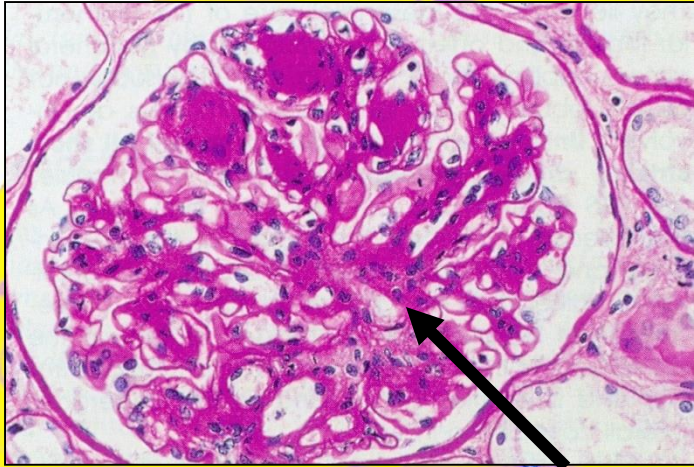
Netter
© CIBA



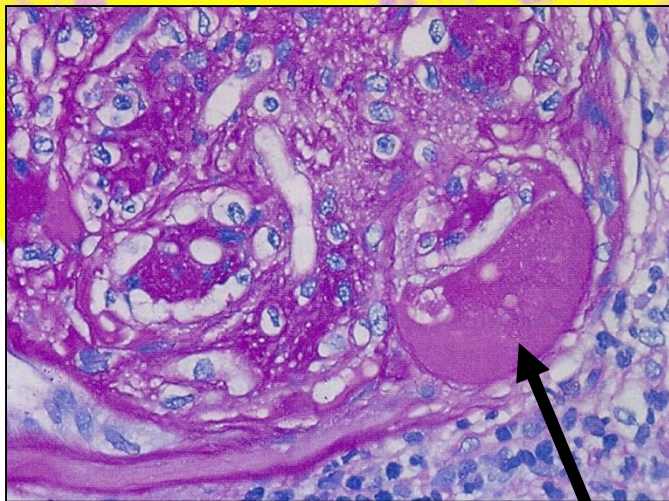
II CLASS OF LE – SMALL CHANGES WITH PRODUCTION OF MESANGIUM. ACCORDING TO WHO: 6 CLASSES OF CHANGES IN GLOMERULI IN LE

DAMAGE OF GLOMERULI IN SYSTEMIC DISEASES

DIABETIC GLOMERULOPATHY

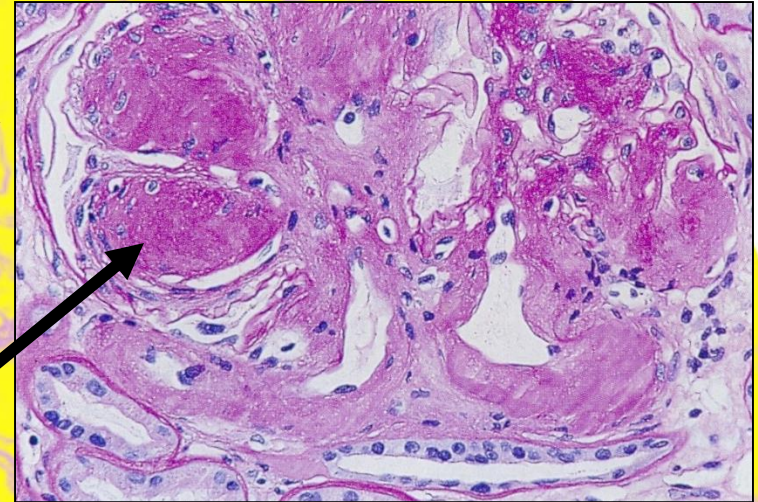


DIFFUSE CHANGES IN MESANGIUM

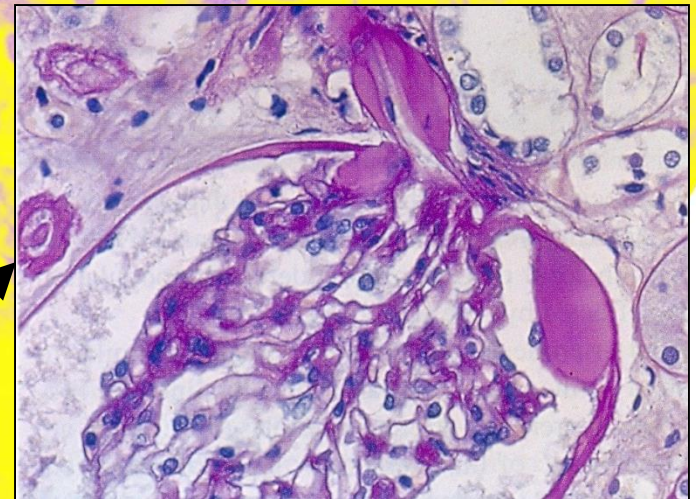


FIBRINOUS CAPS

**GLOMERULAR
CHANGES IN
DIABETES**



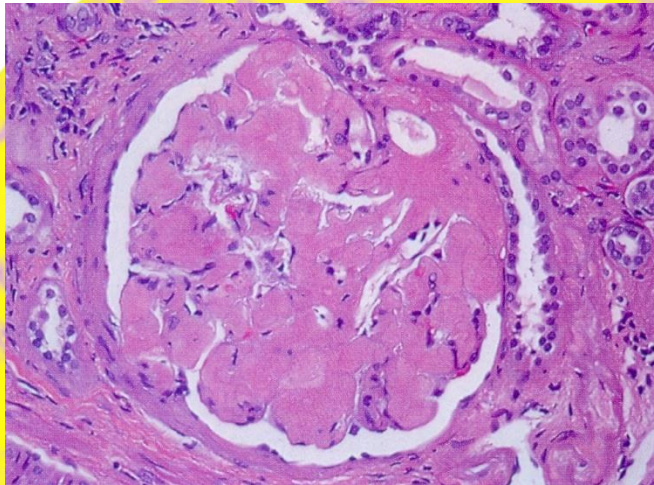
**INTERCAPILLARY GLOMERULOSCLEROSIS
KIMMELSTIEL-WILSON**



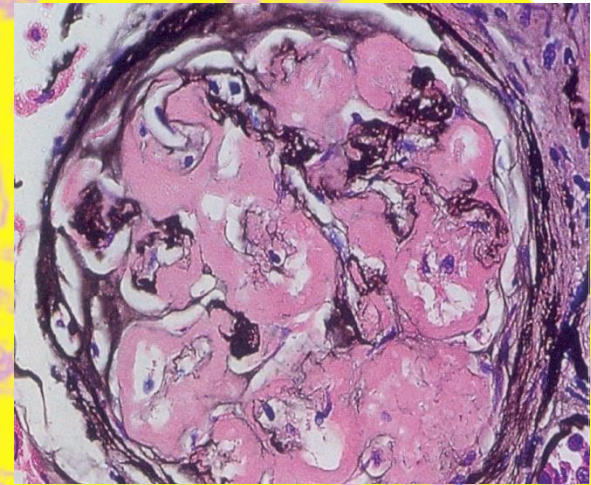
SCLEROSIS OF ARTERIOLES

DAMAGE OF GLOMERULI IN SYSTEMIC DISEASES

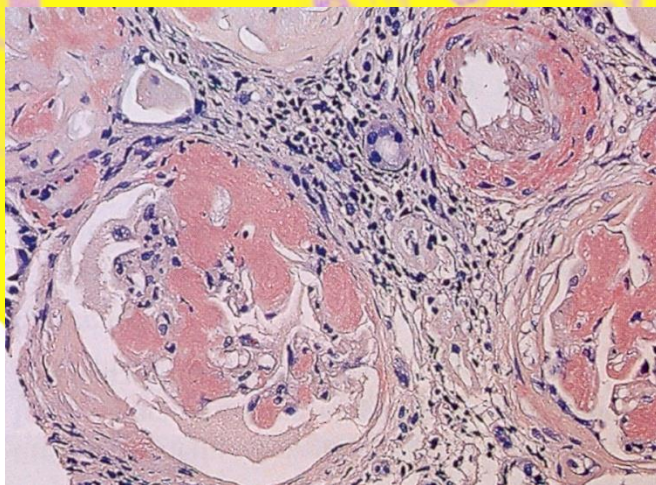
AMYLOIDOSIS



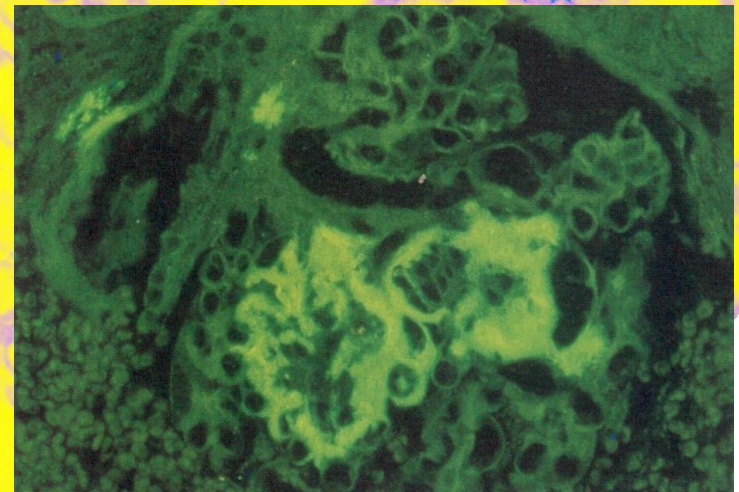
AMYLOID DEPOSITS IN GLOMERULI (HE)



AMYLOID DEPOSITS IN GLOMERULI (silver)



AMYLOID DEPOSITS IN GLOMERULI (Kongo red)

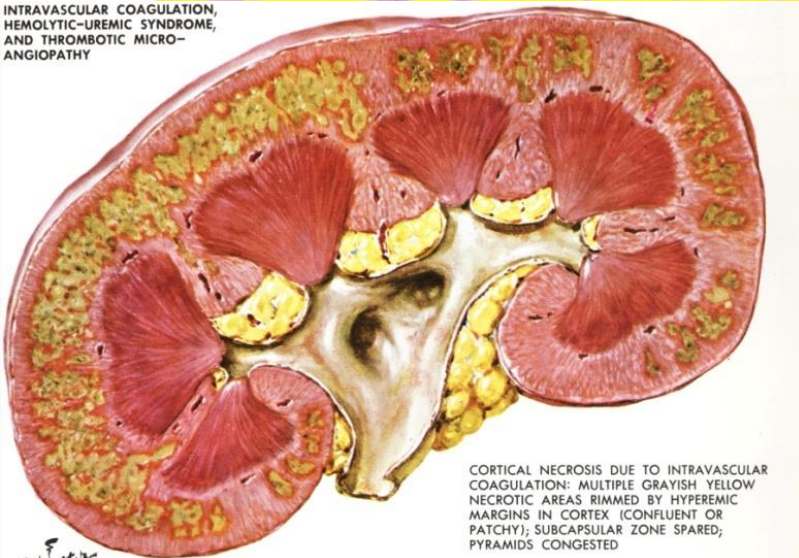


**AMYLOID DEPOSITS IN
GLOMERULI (fluorescent
microscopy)**

ACUTE NECROSIS OF RENAL CORTEX

THE MOST COMMON FORM OF KIDNEY DAMAGE, RELATED TO INTRAVASCULAR COAGULATION. A COMPLICATION IN THE 3rd TRIMESTER OF PREGNANCY (ABLATION OF THE PLACENTA), SEPTIC ANEMIA, BURNING, TRAUMA, INTOXICATION, CHANGES ARE SYMMETRICAL AND IRREVERSIBLE.

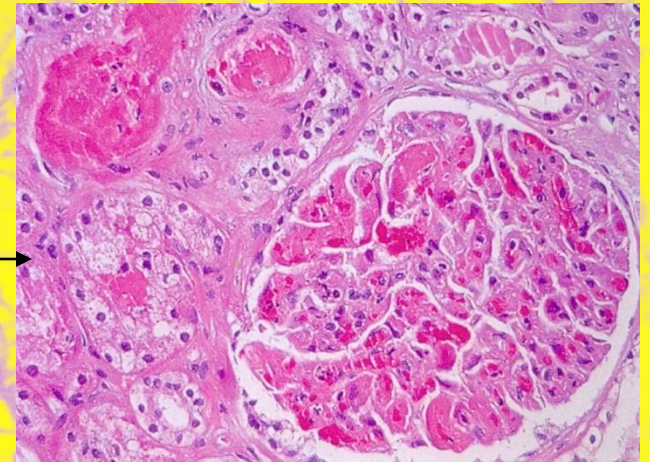
INTRAVASCULAR COAGULATION,
HEMOLYTIC-UREMIC SYNDROME,
AND THROMBOTIC MICRO-
ANGIOPATHY



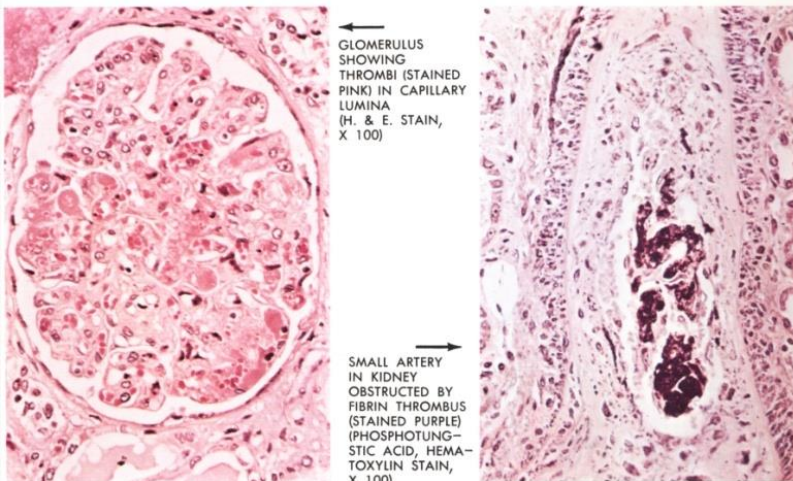
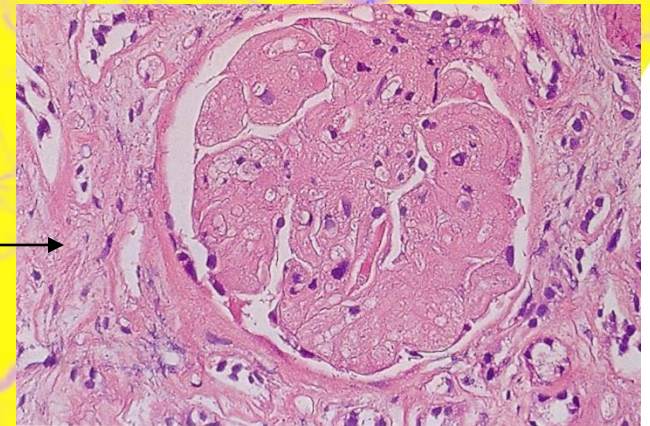
CORTICAL NECROSIS DUE TO INTRAVASCULAR COAGULATION: MULTIPLE GRAYISH YELLOW NECROTIC AREAS RIMMED BY HYPEREMIC MARGINS IN CORTEX (CONFLUENT OR PATCHY); SUBCAPSULAR ZONE SPARED; PYRAMIDS CONGESTED

F. Netter
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THROMBOTIC MICROANGIOPATHY



NECROTIC GLOMERULUS



←
GLOMERULUS
SHOWING
THROMBI
(STAINED
PINK) IN
CAPILLARY
LUMINA
(H. & E. STAIN,
X 100)

→
SMALL ARTERY
IN KIDNEY
OBSTRUCTED BY
FIBRIN THROMBUS
(STAINED PURPLE)
(PHOSPHOTUNG-
STIC ACID, HEMA-
TOXYLIN STAIN,
X 100)

(TUBULO)-INTERSTITIAL NEPHRITIS

**GROUP OF DISEASES THAT ARE HISTOLOGICALLY
CHARACTERISTIC (LESIONS OF CANALICULI AND STROMA)**

INFECTIOUS FACTORS

ACUTE BACTERIAL INFLAMMATION OF KIDNEYS

CHRONIC NEPHRITIS

OTHER INFECTIONS (VIRAL, PARASITIC)

TOXINS

DRUGS

ACUTE ALLERGIC INTERSTITIAL NEPHRITIS

NEPHRITIS BECAUSE OF THE OVERUSE OF ANALGESICS

HEAVY METALS

LEAD, MERCURY, CADMIUM

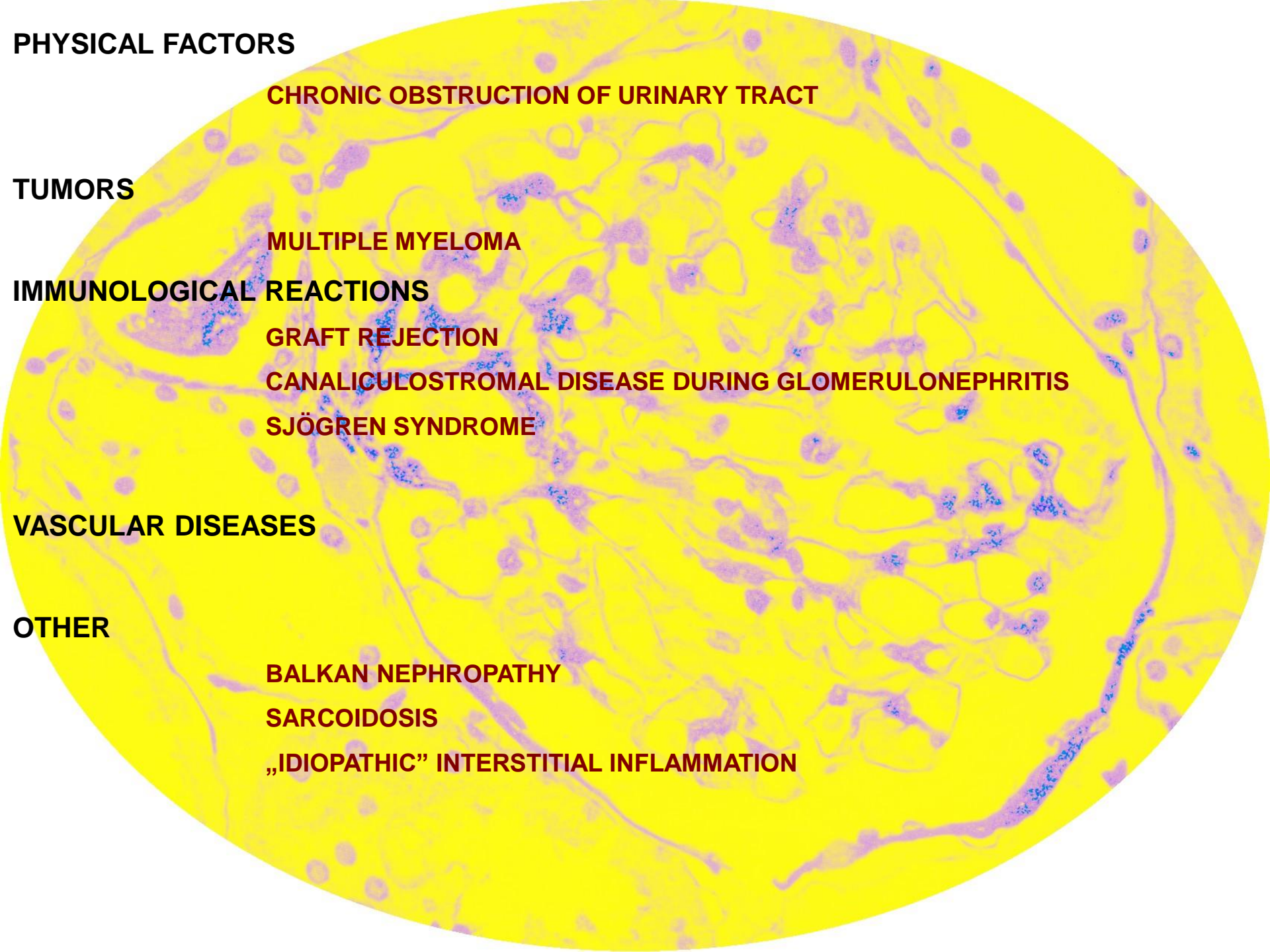
METABOLIC DISEASES

URIC NEPHROPATHY

CALCIUM NEPHROPATHY

HYPOKALEMIC NEPHROPATHY

OXALATE NEPHROPATHY



PHYSICAL FACTORS

CHRONIC OBSTRUCTION OF URINARY TRACT

TUMORS

MULTIPLE MYELOMA

IMMUNOLOGICAL REACTIONS

GRAFT REJECTION

CANALICULOSTROMAL DISEASE DURING GLOMERULONEPHRITIS

SJÖGREN SYNDROME

VASCULAR DISEASES

OTHER

BALKAN NEPHROPATHY

SARCOIDOSIS

„IDIOPATHIC” INTERSTITIAL INFLAMMATION

PYELONEPHRITIS

PYELONEPHRITIS IS A DISEASE MAINLY INVOLVING THE STROMA AND RENAL PELVIS

INFECTION OF URINARY TRACT

VERY COMMON INFECTION OF THE BLADDER OR KIDNEY OR BOTH

ETIOLOGY AND PATHOGENESIS OF PYELONEPHRITIS

BACTERIAL FACTORS (85% GRAM-NEGATIVE BACTERIA FROM INTESTINES)

DESCENDING INFECTIONS (HEMATOGENIC)

ASCENDING INFECTIONS

**FACTORS FAVORING COLONIZATION OF URINARY TRACT BY BACTERIA:
ANATOMICAL FACTORS e.g., SHORT, STRAIGHT URETHRA IN WOMEN,
INSTRUMENTATION OF THE URINARY TRACT e.g., CATHETERIZATION,
OBSTRUCTIONS, REFLUX, IMMUNOLOGICAL MECHANISMS.**

ACUTE PYELONEPHRITIS

ACUTE PYELONEPHRITIS IS A PURULENT FORM OF INFLAMMATION CAUSED BY BACTERIAL INFECTION



METASTATIC ABSCESSUS

ACUTE PYELONEPHRITIS



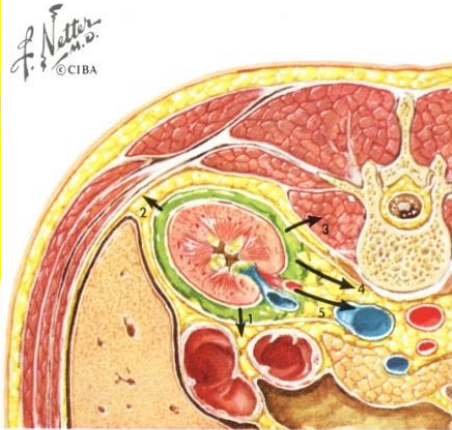
CARBUNCLE
OF KIDNEY



ARTERIOGRAM: NUMEROUS VESSELS AROUND MASS
SURROUNDED BY HOMOGENEOUS BLUSH IN UPPER
POLE OF KIDNEY, SUGGESTIVE OF INFLAMMATORY
PROCESS (CARBUNCLE); ALSO MARKED LUMBAR SCLIOSIS



CORTICAL
ABSCESSUS



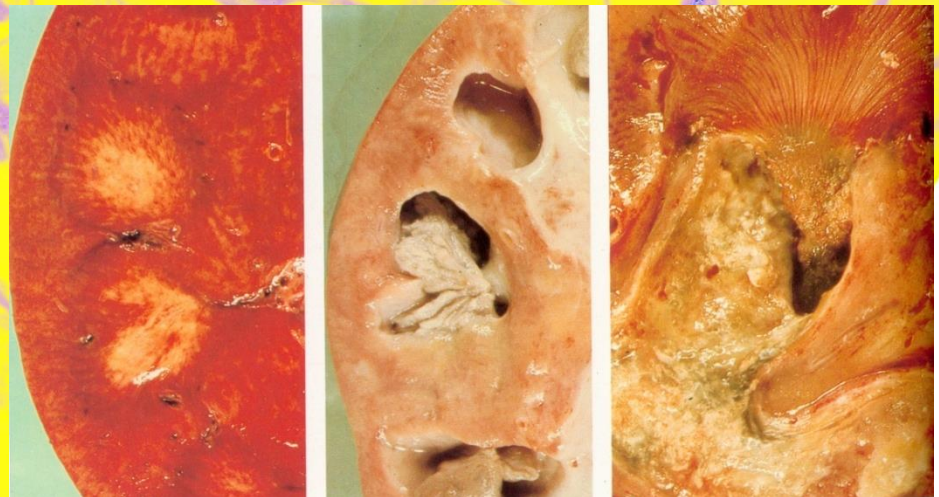
PERIRENAL ABSCESS

ROUTES OF SPREAD:

1=THROUGH RENAL FASCIA (OF GEROTA) TO RETROPERITONEAL
TISSUES; 2=TO FLANK; 3=TO PSOAS MUSCLE; 4=TO MIDLINE
(PREVERTEBRAL); 5=TO INFERIOR VENA CAVA; 6=TO RETROHEPATIC
AND SUBPHRENIC AREAS; 7=THROUGH DIAPHRAGM TO PLEURAL
CAVITY; 8=TO PELVIC RETROPERITONEAL TISSUES



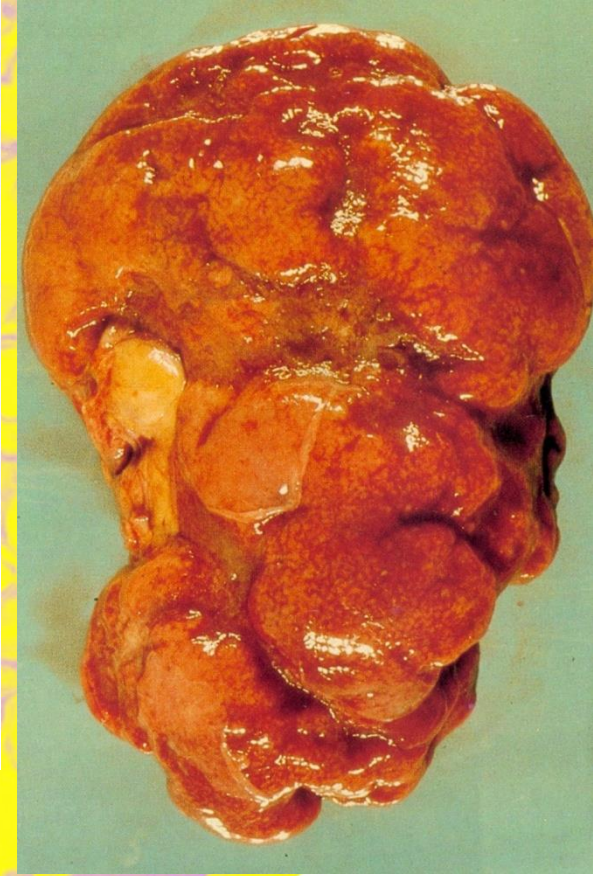
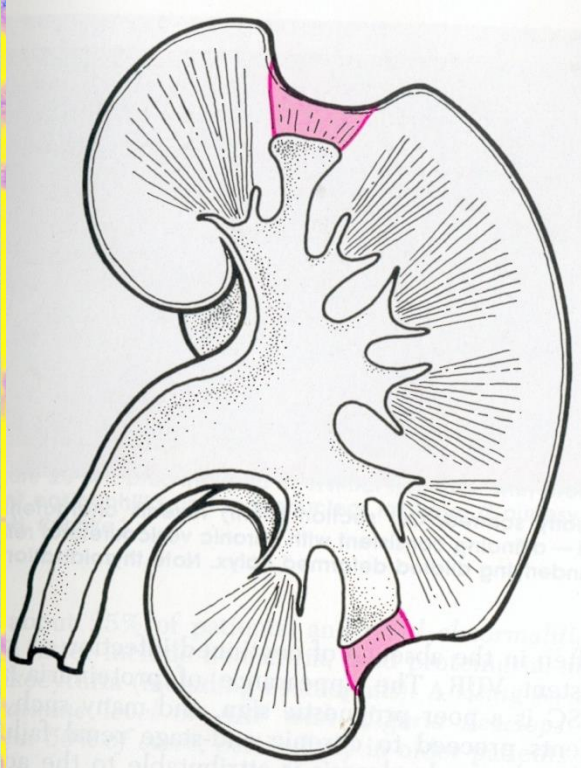
PERI- AND PARANEPHRITIC
ABSCESSUS



DIFFERENT PHASES OF NECROSIS

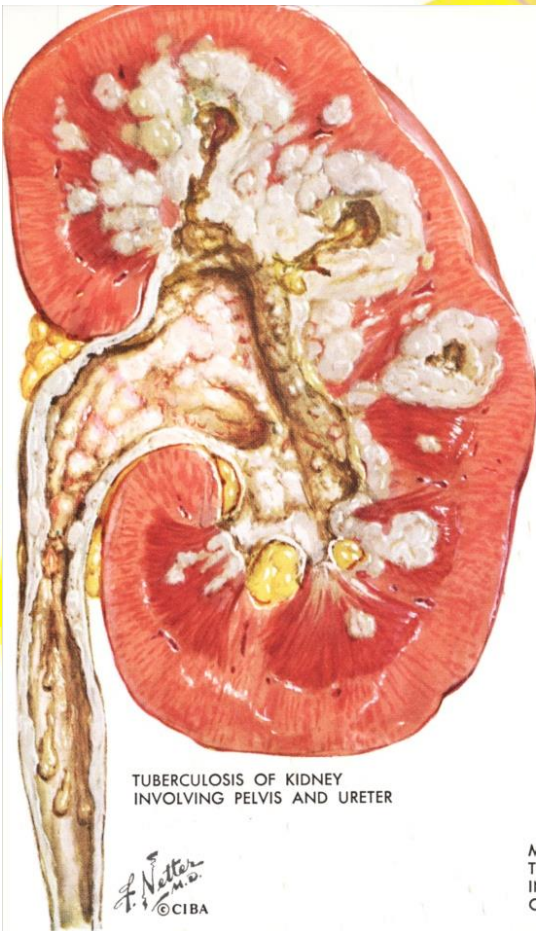
CHRONIC PYELONEPHRITIS

CHRONIC PYELONEPHRITIS IS AN INFLAMMATORY PROCESS IN WHICH BACTERIA ARE NOT ALWAYS PRESENT



MACROSCOPIC PICTURE OF CHRONIC PYELONEPHRITIS – CHANGE OF SHAPE (!)

TUBERCULOSIS OF KIDNEYS



TUBERCULOSIS OF KIDNEY INVOLVING PELVIS AND URETER

F. Netter M.D.
© CIBA

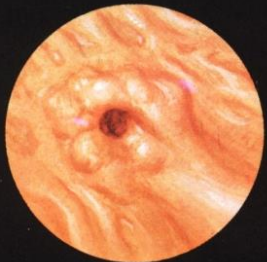


INTRAVENOUS PYELOGRAM: RENAL TUBERCULOSIS
DISTORTION OF COLLECTING SYSTEM AND
DILATATION OF URETER, MOST MARKED ON LEFT

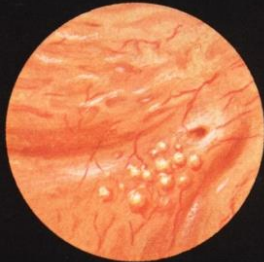


MILIARY
TUBERCULOSIS
IN KIDNEY
OF A CHILD

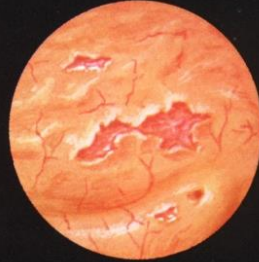
TUBERCULOSIS OF BLADDER, CYSTOSCOPIC VIEWS



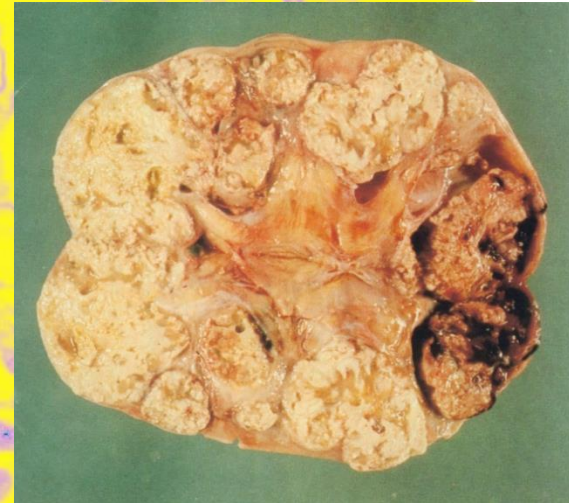
RETRACTION OF URETERAL ORIFICE
("GOLF HOLE" ORIFICE) WITH EDEMA
AND RIGIDITY OF SURROUNDING WALL



TUBERCLES IN PROXIMITY
TO URETERAL ORIFICE

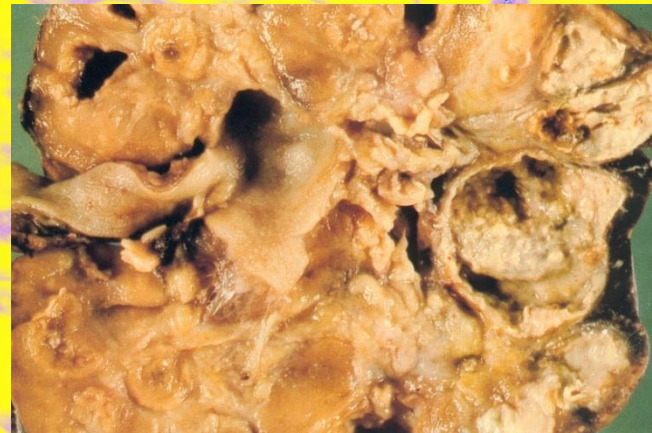


LARGE AND SMALL TUBERCULOUS
ULCERS IN URINARY BLADDER



CASEOUS TUBERCULOSIS

SURGICAL FORMS OF TBC IN KIDNEYS

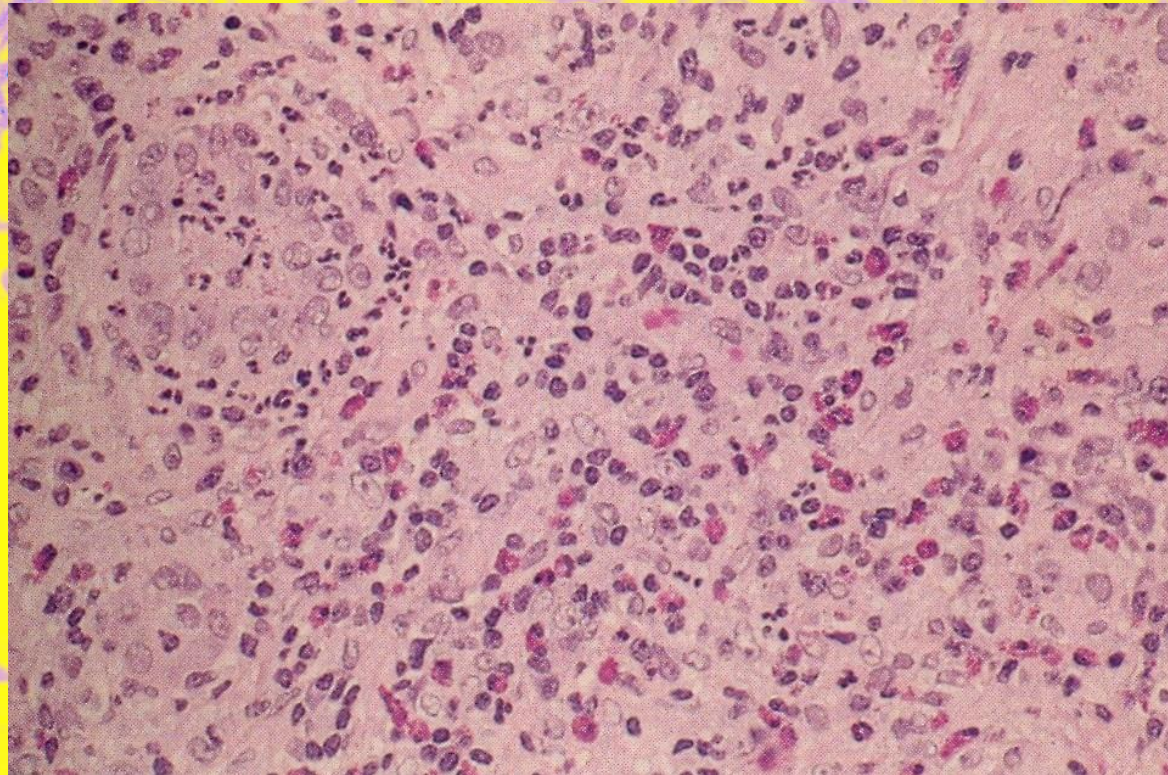


CAVERNOUS TUBERCULOSIS



INTERSTITIAL INFLAMMATION INDUCED BY DRUGS AND TOXINS

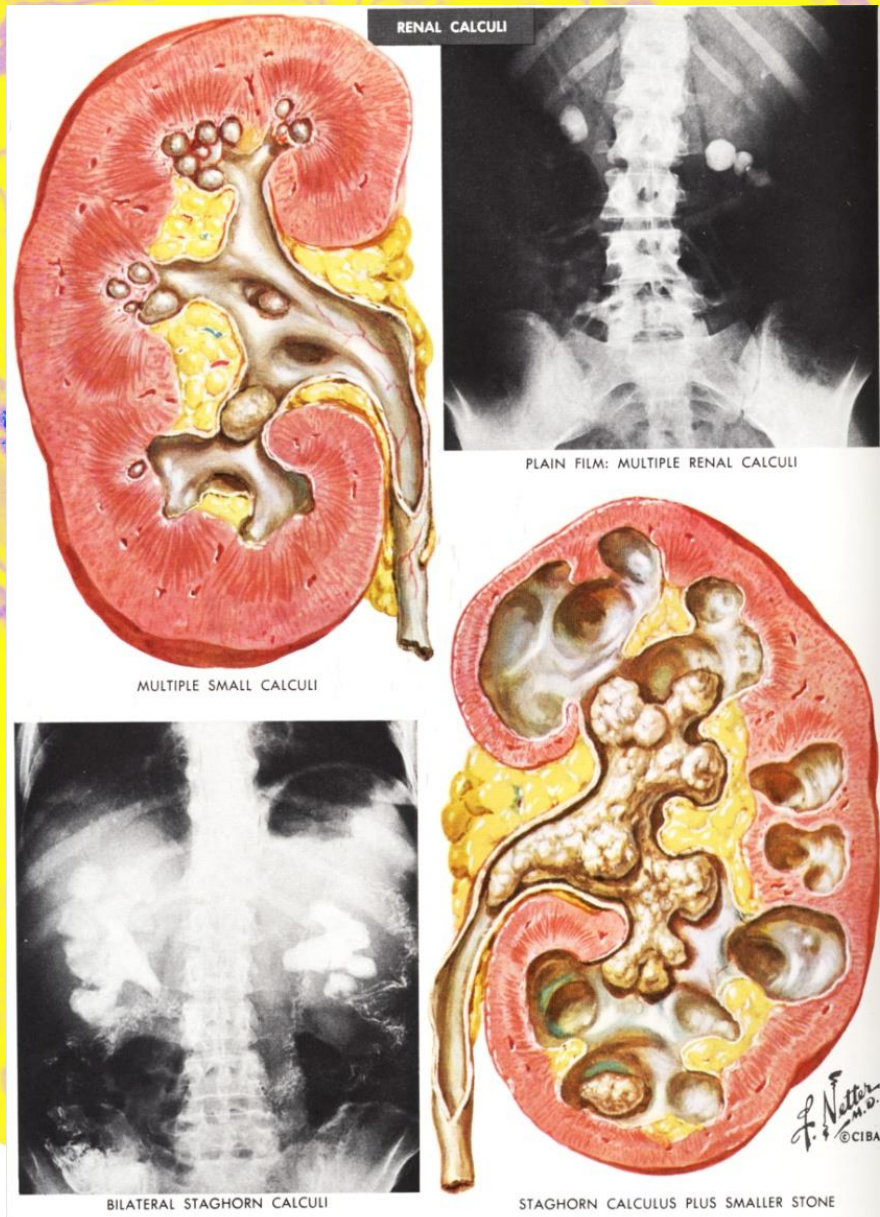
**DESTRUCTION OF KIDNEYS INDUCED BY SULPHONAMIDES, SYNTHETIC
ANTIBIOTICS (e.g., PENICILLIN), DIURETICS, NONSTEROID ANTI-
INFLAMMATORY DRUGS**



**INFLAMMATORY INFILTRATION IN INTERSTITIAL NEPHRITIS (MONONUCLEAR
CELLS WITH SIGNIFICANT INVOLVEMENT OF EOSINOPHILS)**

CALCIUM NEPHROPATHY

DURING HYPERCALCEMIA (HYPERPARATHYROIDISM, MULTIPLE MYELOMA, BONE METASTASES, HYPERVITAMINOSIS D) - NEPHROLITHIASIS

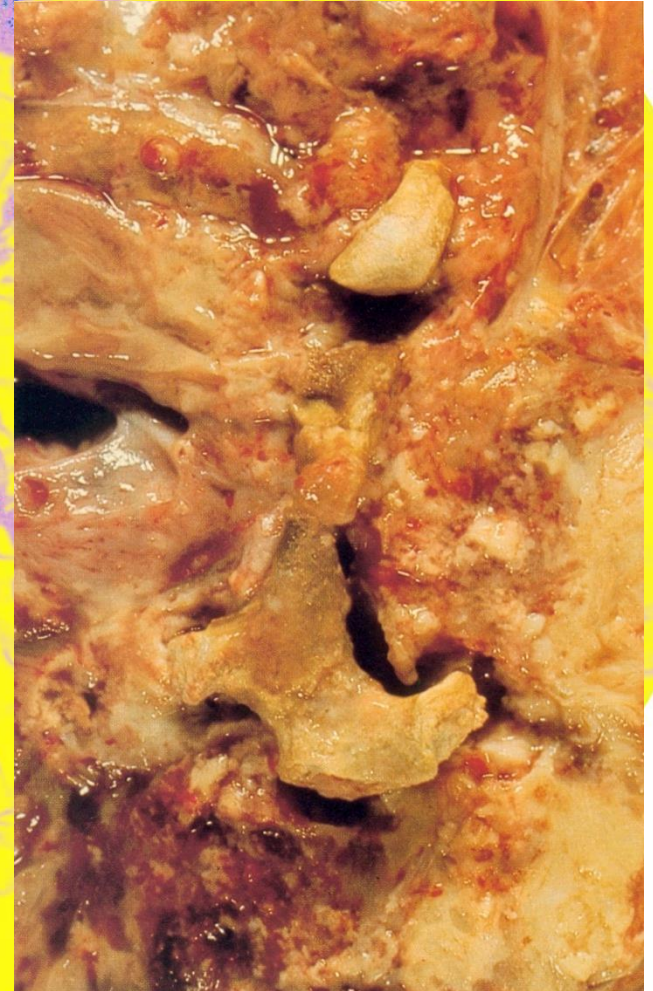
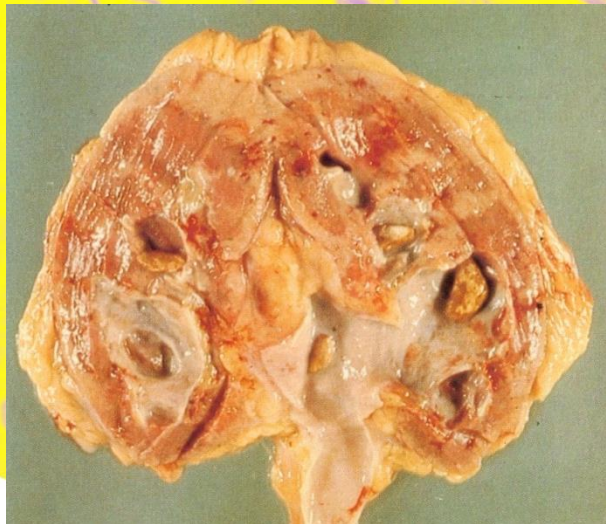


CALCIUM NEPHROPATHY

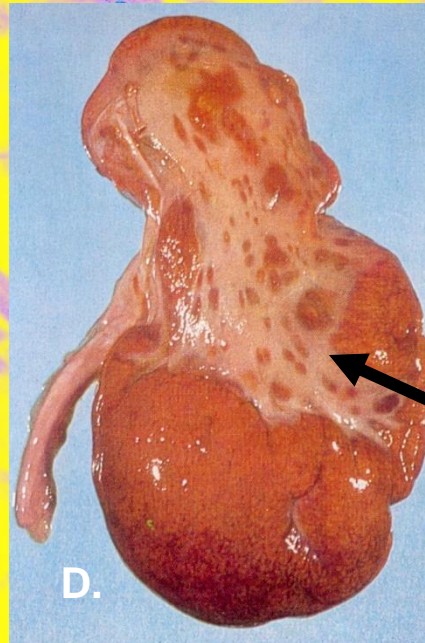
**DURING HYPERCALCEMIA (HYPERPARATHYROIDISM, MULTIPLE MYELOMA, BONE METASTASES, HYPERVITAMINOSIS D)
NEPHROLITHIASIS**



STONES IN KIDNEY



CIRCULATION INFARCT



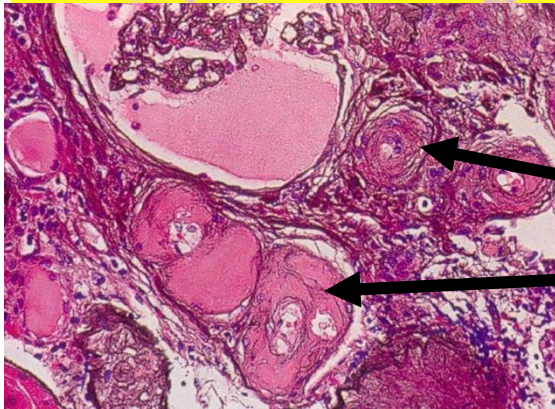
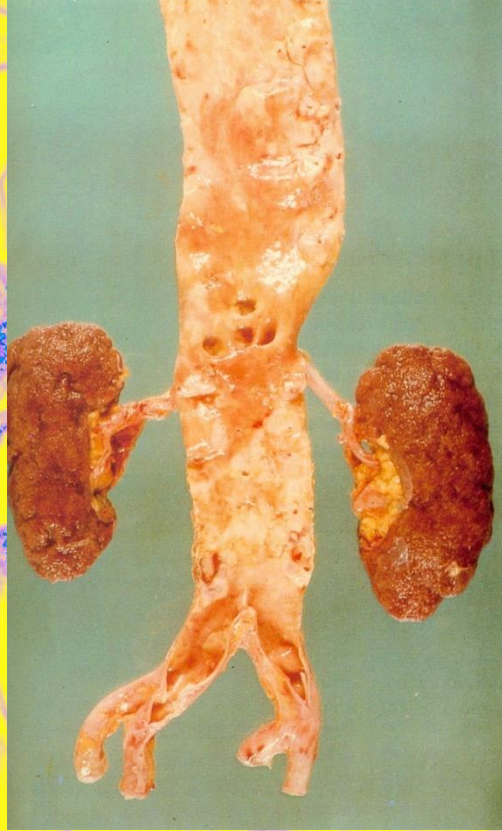
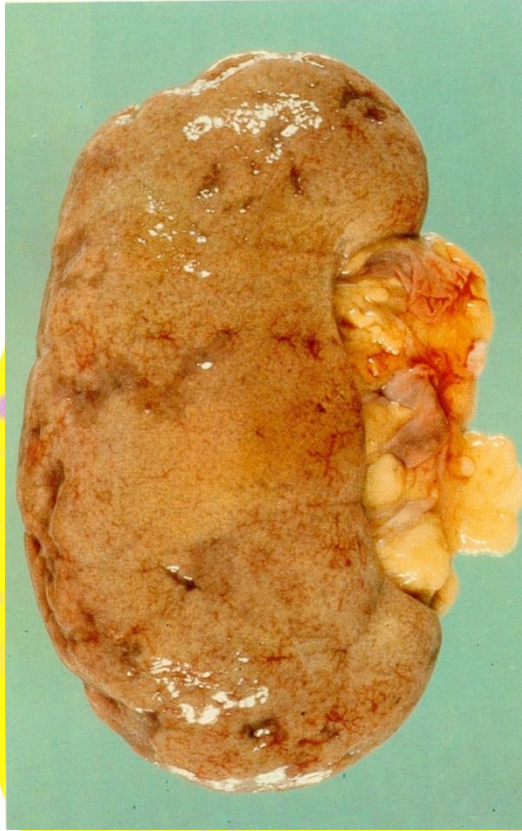
**A. RECENT PALE
INFARCT WITH
DISTINCT RED BORDER**

**B. MICROSCOPIC
PICTURE OF INFARCT**

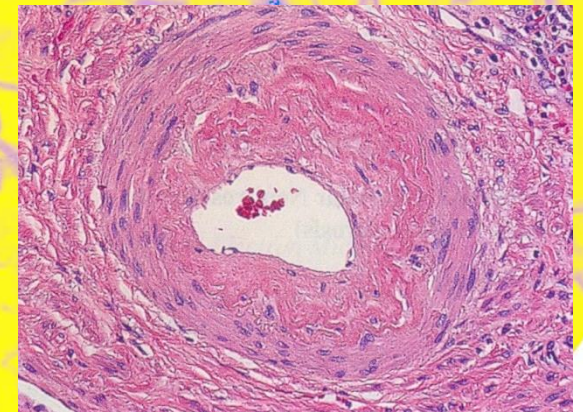
**C. INFARCT IN
ORGANIZATION**

**D. DIFFUSE POST-
INFARCT SCAR IN
KIDNEY**

VASCULAR PROBLEMS



**HYALINIZATION OF
ARTERIOLEAR WALLS**

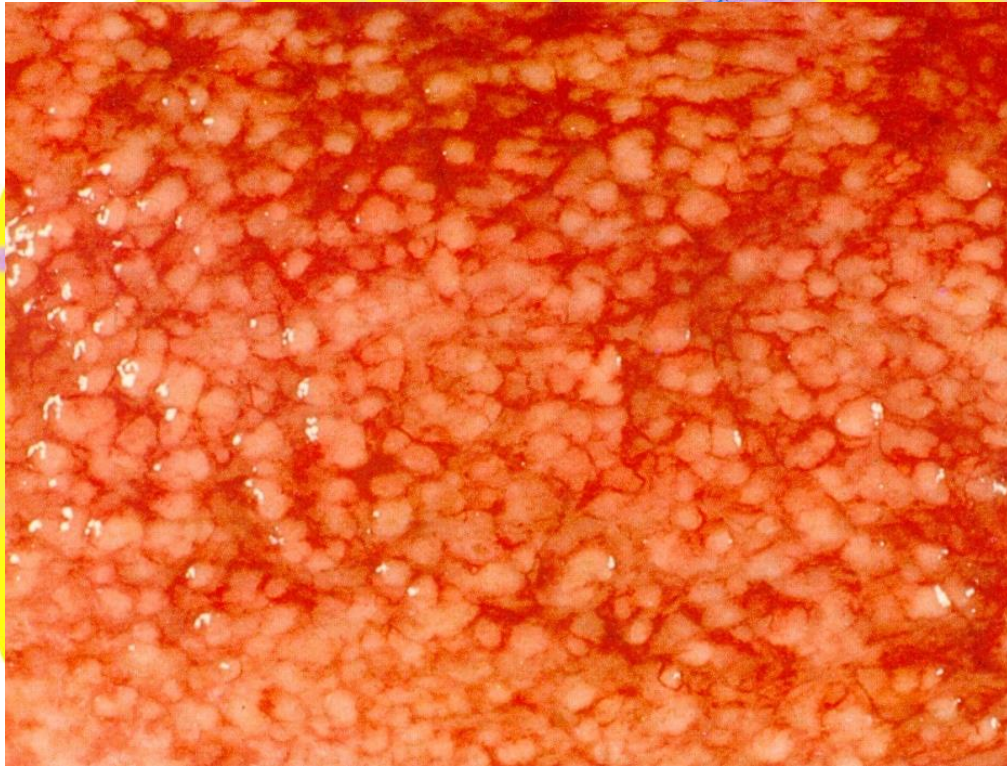


**HARDENING OF ARTERIAL WALLS
DUE TO MILD HYPERTENSION**

CHANGES IN LARGE ARTERIES

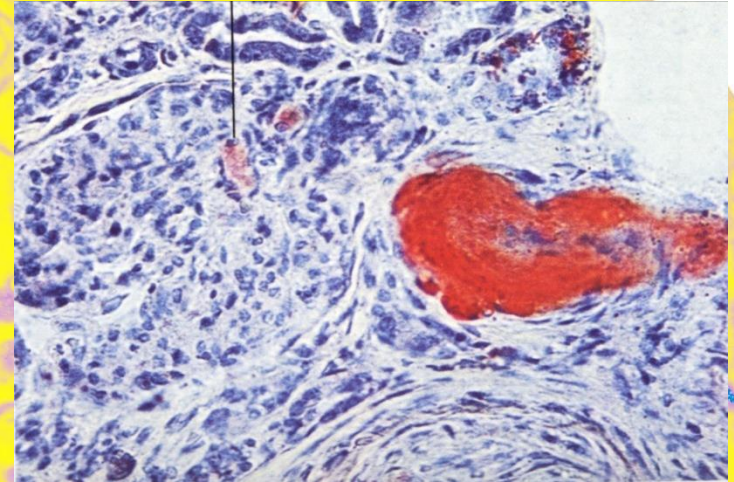
VASCULAR PROBLEMS

ARTERIOLOSCLEROTIC NEPHROCIRRHOSIS

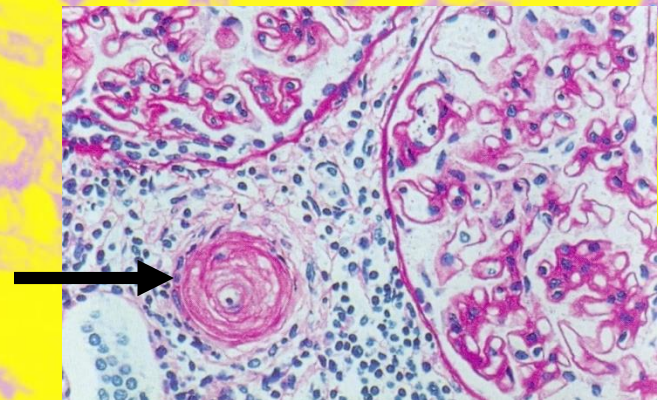


**MACROSCOPIC PICTURE OF SURFACE
OF KIDNEY (GRANULAR)**

CHANGES OF ARTERIOLES



HYALINIZATION OF ARTERIOLE



ARTERIOLOSCLEROSIS

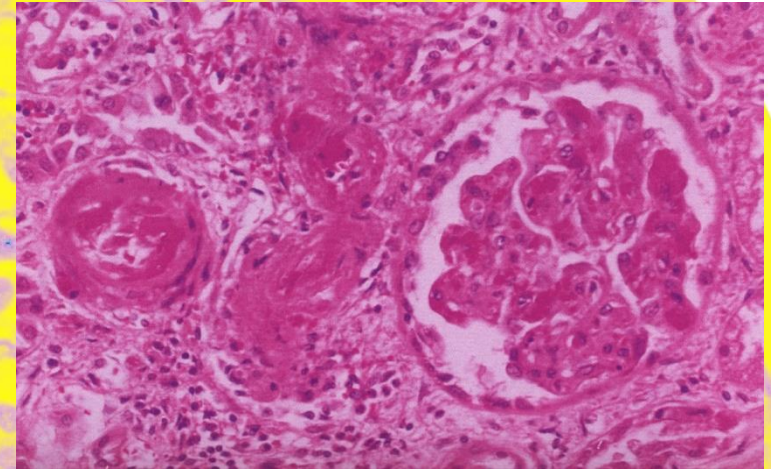
VASCULAR PROBLEMS

MALIGNANT ARTERIOLOSCLEROTIC NEPHROCIRRHOISIS

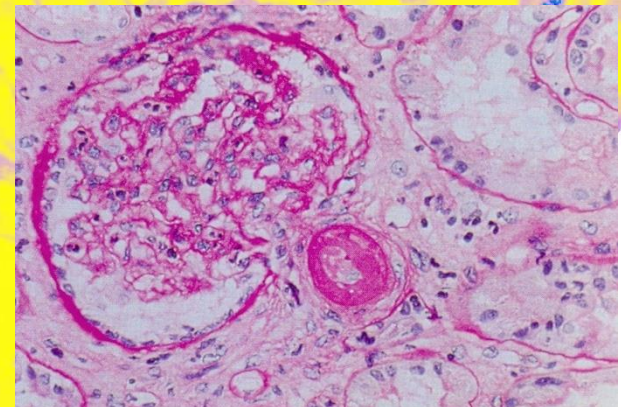
MALIGNANT PHASE OF HYPERTENSION



**MACROSCOPIC PICTURE
GRANULAR SURFACE OF KIDNEY**



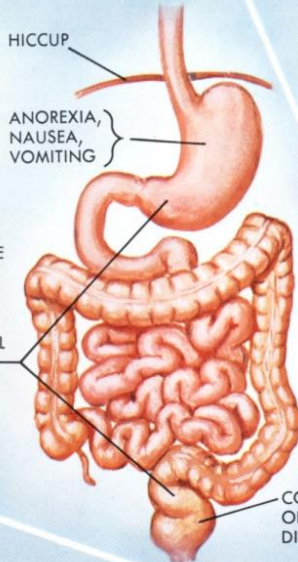
**FIBRINOID NECROSIS IN
ARTERIOLES**



UREMIA



COATED TONGUE,
AMMONIACAL OR
UNPLEASANT TASTE
IN MOUTH



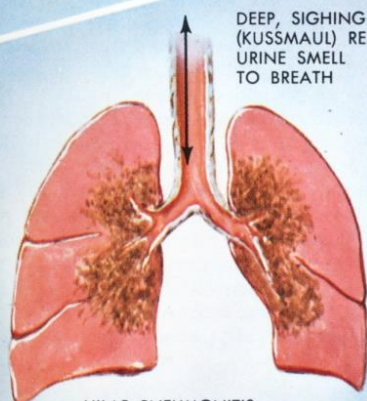
HICCUP

ANOREXIA,
NAUSEA,
VOMITING

GASTROINTESTINAL
BLEEDING

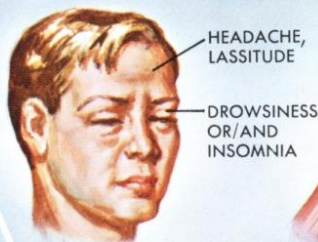
CONSTIPATION
OR
DIARRHEA

GFR ↓
CREATININE CLEARANCE ↓
UREA CLEARANCE ↓
BUN ↑ URIC ACID ↑
PLASMA CREATININE ↑
URINE CONCENTRATING ABILITY ↓
URINE DILUTING ABILITY ↓ (LATER);
DISTURBANCES OF Na, K, Ca, PO_4 ,
AND GLUCOSE METABOLISM;
ACIDOSIS



HILAR PNEUMONITIS;
"BAT-WING" OPACITY
ON X-RAY
(UREMIC LUNG)

DEEP, SIGHING
(KUSSMAUL) RESPIRATION;
URINE SMELL
TO BREATH



HEADACHE,
LASSITUDE

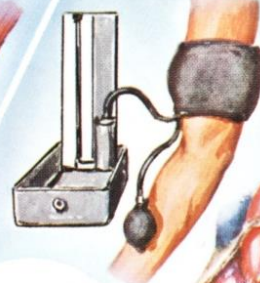
DROWSINESS
OR/AND
INSOMNIA

MUSCLE TWITCHING,
WEAKNESS

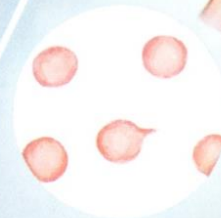
CONVULSIONS
OR COMA

NEURAL
GASTRO-
INTESTINAL
BIO-
CHEMI-
CAL
RESPIRATORY
DERMAL

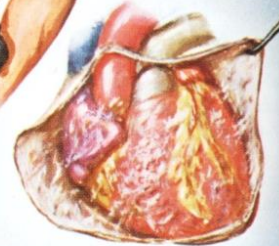
UREMIA:
MANIFESTATIONS
WHICH MAY BE
PRESENT IN
VARIABLE DEGREE



HYPERTENSION
USUAL BUT NOT
INVARIABLE



ANEMIA; NORMOCHROMIC,
NORMOCYTIC (BURR CELLS
MAY APPEAR)
POLYCYTHEMIA RARELY



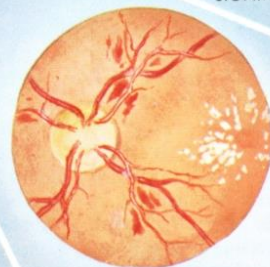
SEROFIBRINOUS
PERICARDITIS
(OCCASIONALLY)



PYURIA,
HEMATURIA,
CYLINDRURIA
COMMON;
BROAD CASTS
ESPECIALLY
SIGNIFICANT

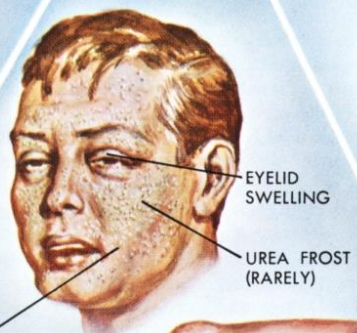
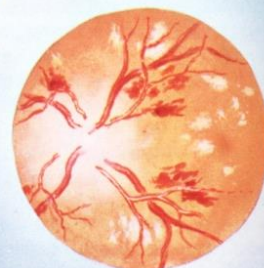


PROTEINURIA
USUAL BUT NOT
IN PROPORTION
TO DEGREE OF
FAILURE;
NEPHROTIC
SYNDROME
MAY IMPROVE
WHEN RENAL
FAILURE APPEARS



ARTERIOSCLEROTIC
RETINOPATHY

OR/AND
HYPERTENSIVE
RETINOPATHY



EYELID
SWELLING

UREA FROST
(RARELY)

GRAYISH
YELLOW
PALLOR



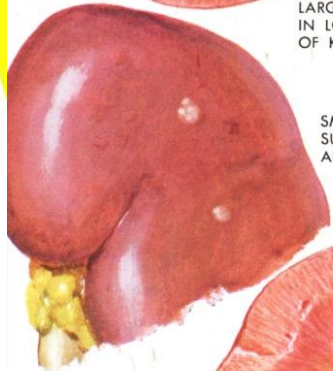
PRURITUS, PURPURA,
SKIN INFECTIONS

TUMORS OF KIDNEY

BENIGN TUMORS OF THE KIDNEYS



LARGE ADENOMA
IN LOWER POLE
OF KIDNEY

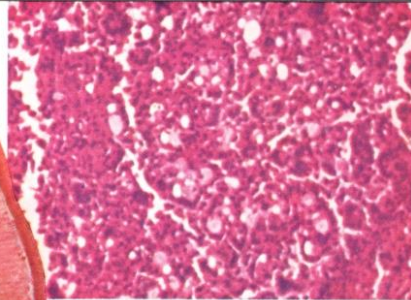


SMALL
SUBCAPSULAR
ADENOMAS

F. Netter
M.D.
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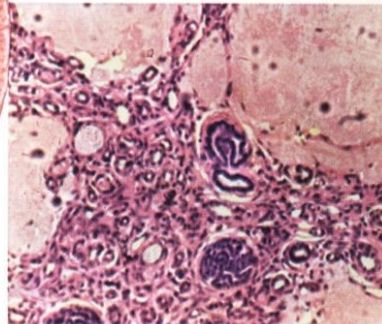
HEMANGIOMA
WITH HEMORRHAGE
(CLOT) IN CALYCES
AND PELVIS



ADENOMA
OF KIDNEY

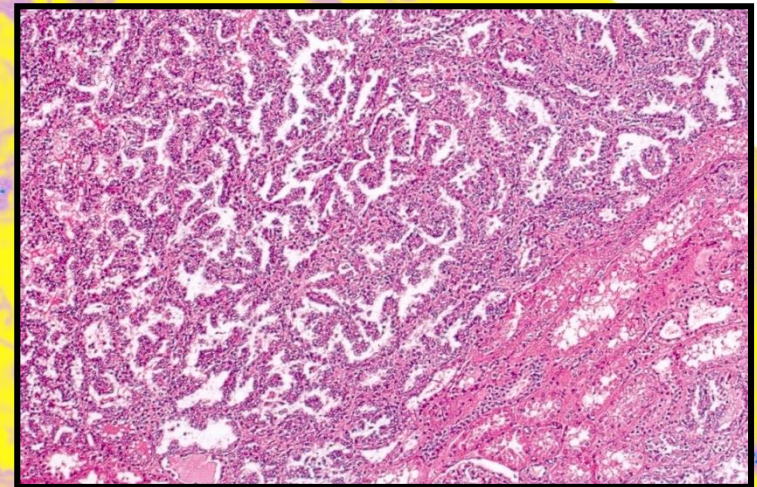


INTRAVENOUS PYELOGRAM: DISTORTION OF
LOWER CALYCES OF LEFT KIDNEY BY MASS
WHICH PROVED TO BE AN ADENOMA

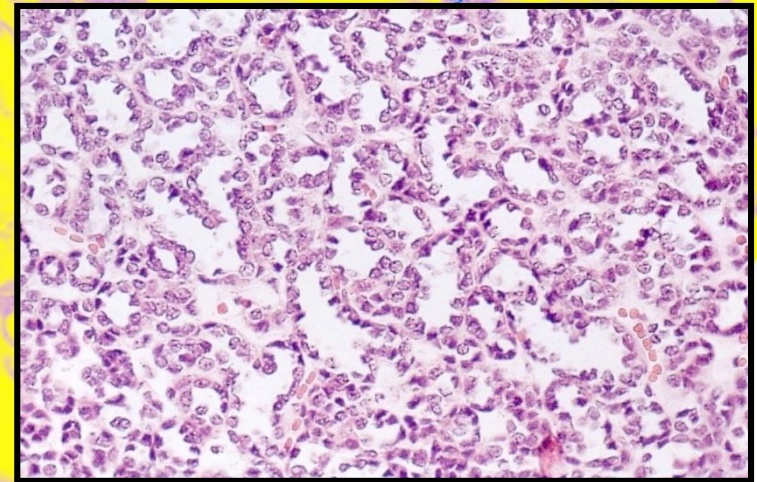


HAMARTOMA OF KIDNEY

BENIGN



PAPILLARY ADENOMA



METANEPHRIC ADENOMA

TUMORS OF KIDNEYS IN ADULTS

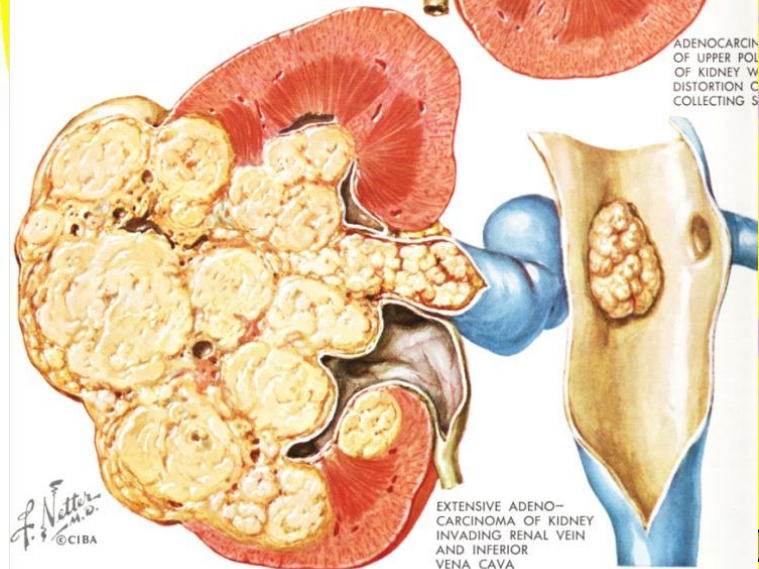
CLEAR CELL ADENOCARCINOMA



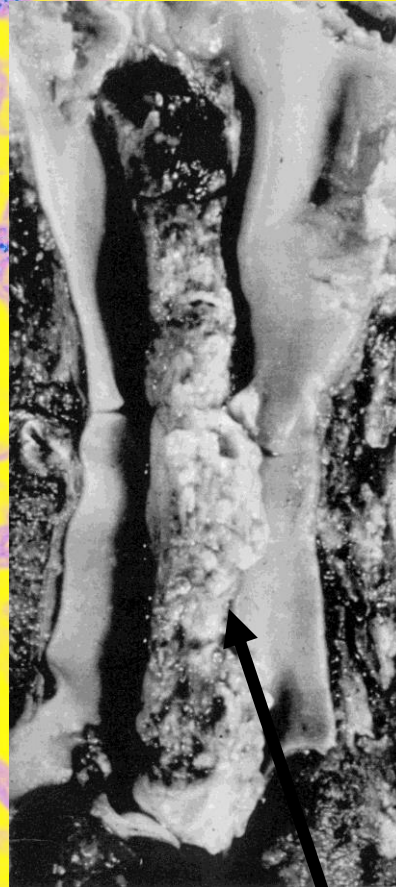
SELECTIVE RIGHT RENAL ARTERIOGRAM SHOWING TYPICAL TUMOR VESSEL PATTERN CHARACTERISTIC OF ADENOCARCINOMA (HYPERNEPHROMA)



ADENOCARCINOMA OF UPPER POLE OF KIDNEY WITH DISTORTION OF COLLECTING SYSTEM



EXTENSIVE ADENOCARCINOMA OF KIDNEY INVADING RENAL VEIN AND INFERIOR VENA CAVA



INFILTRATION OF VENA CAVA INFERIOR



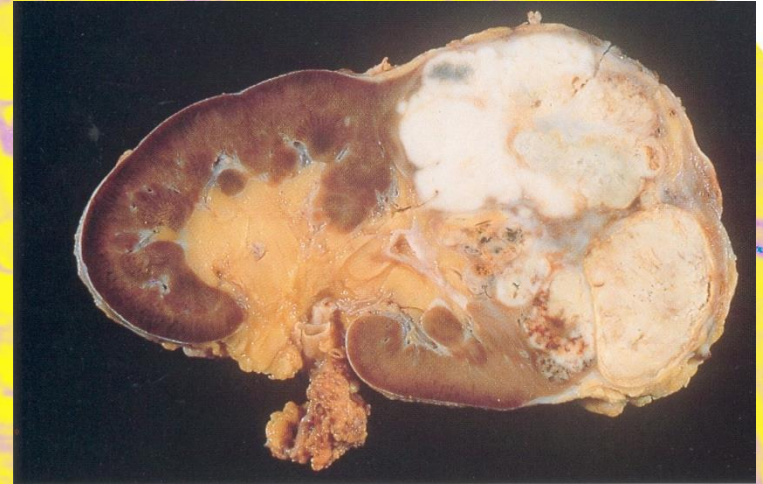
NEOPLASTIC THROMBOSIS IN PULMONARY VEINS

TUMORS OF KIDNEYS IN ADULTS

CLEAR CELL ADENOCARCINOMA



**EARLY STAGE: TUMOR WITH
DISTINCT BORDERS**



WHITE SARCOMATOUS TISSUE



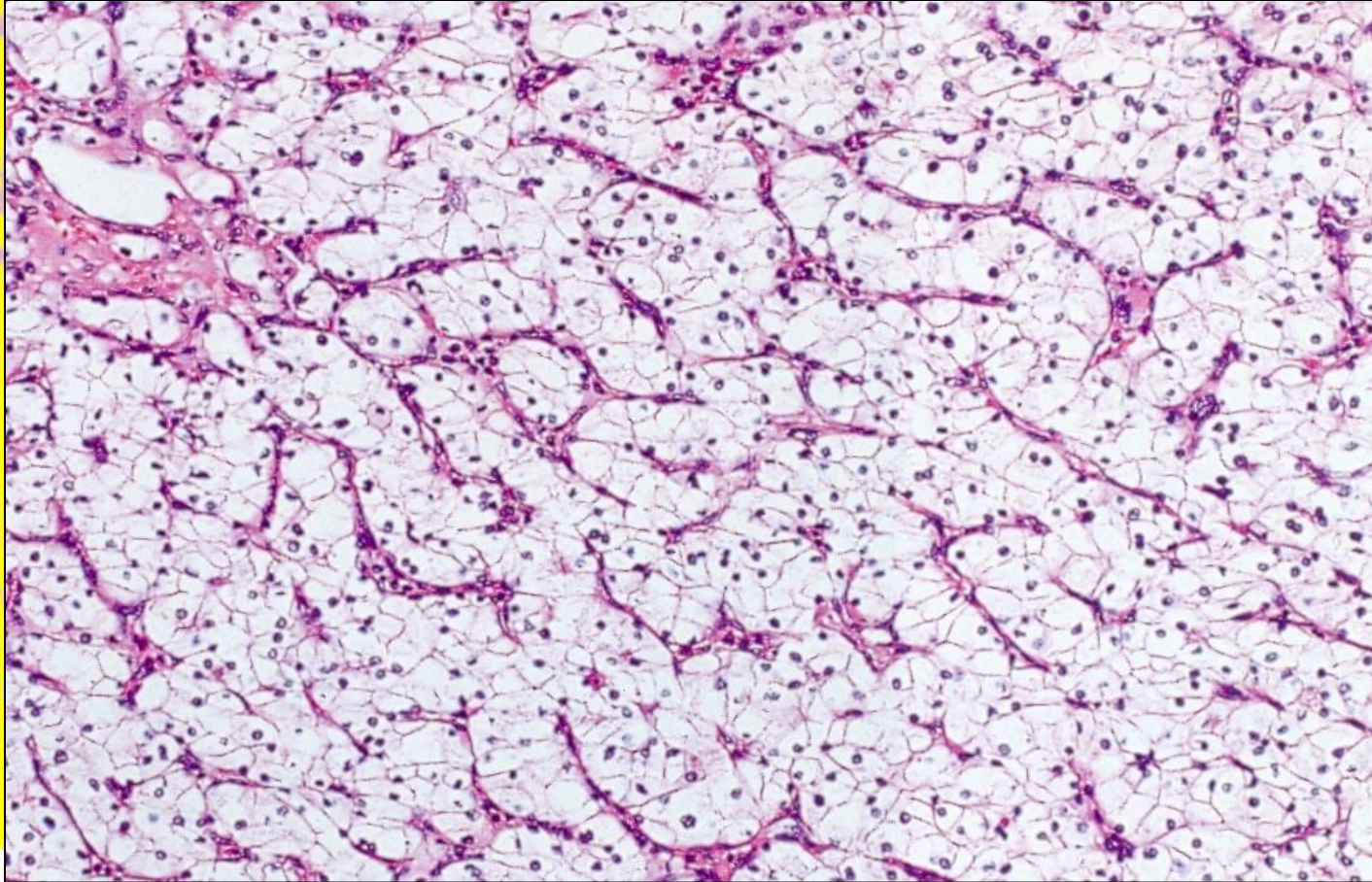
**TYPICAL PICTURE OF ADVANCED TUMOR
WITH INFILTRATION TO RENAL VEIN**



CYSTIC FORM OF CANCER

TUMORS OF KIDNEYS IN ADULTS

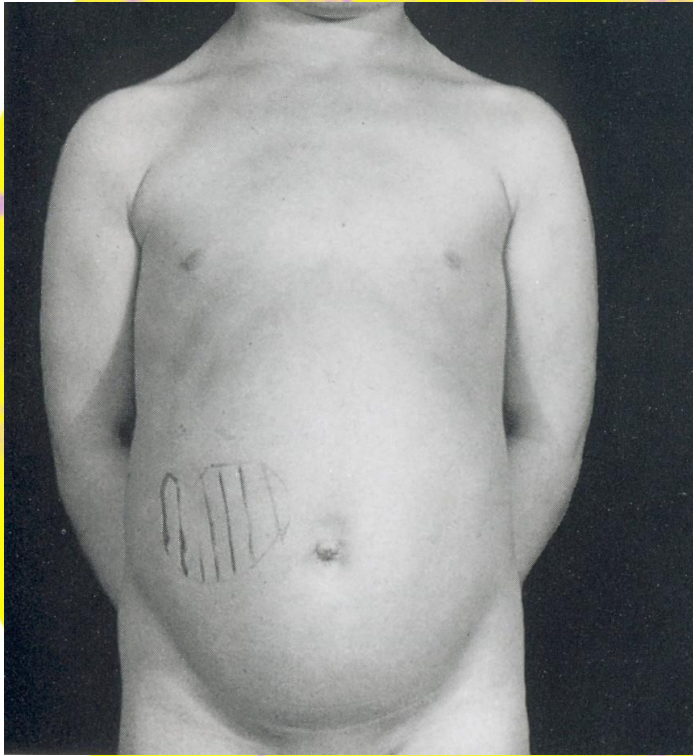
CLEAR CELL ADENOCARCINOMA



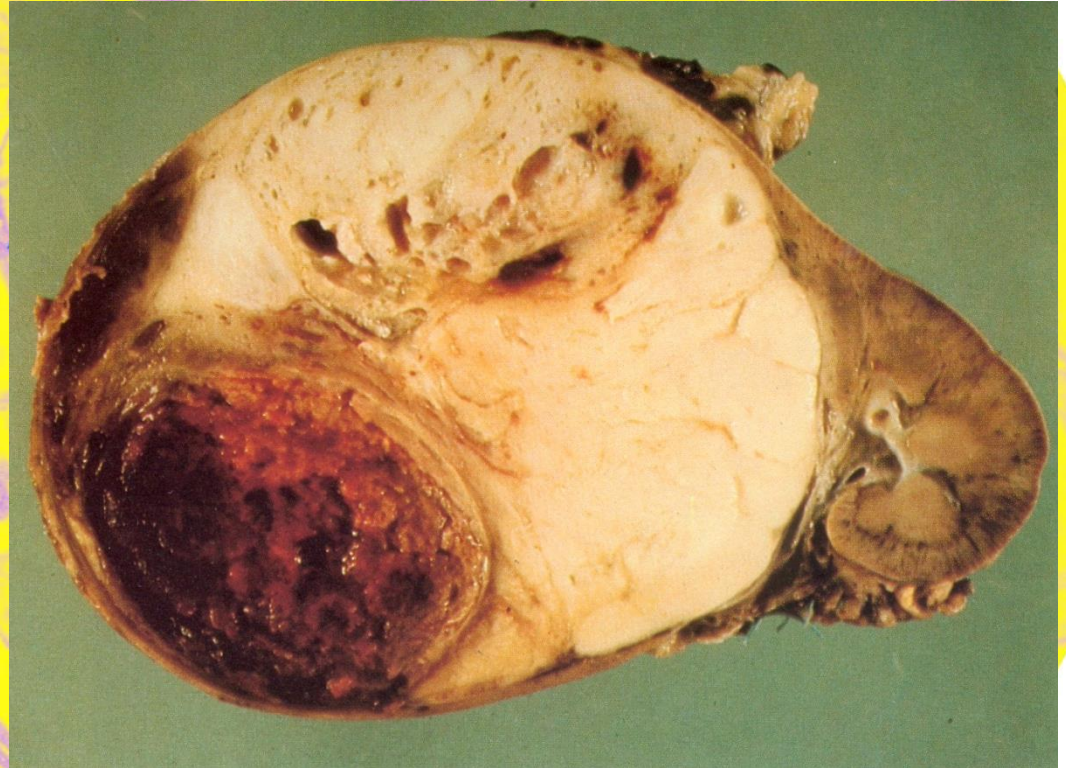
**CLASSICAL MICROSCOPIC PICTURE OF CLEAR CELL
ADENOCARCINOMA**

TUMORS IN CHILDREN

NEPHROBLASTOMA



**ASYMMETRICAL TUMOR IN
ABDOMEN**

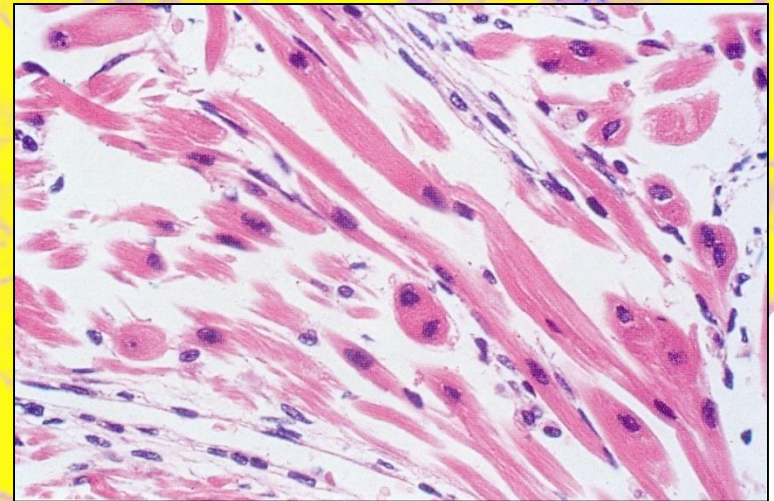
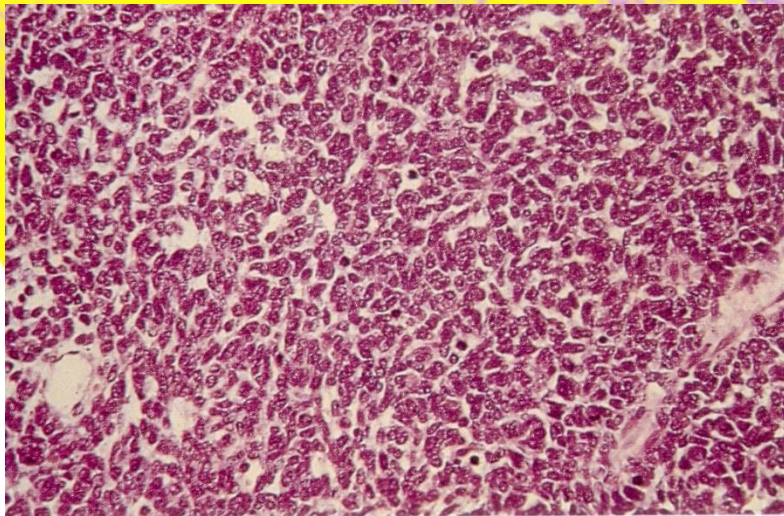
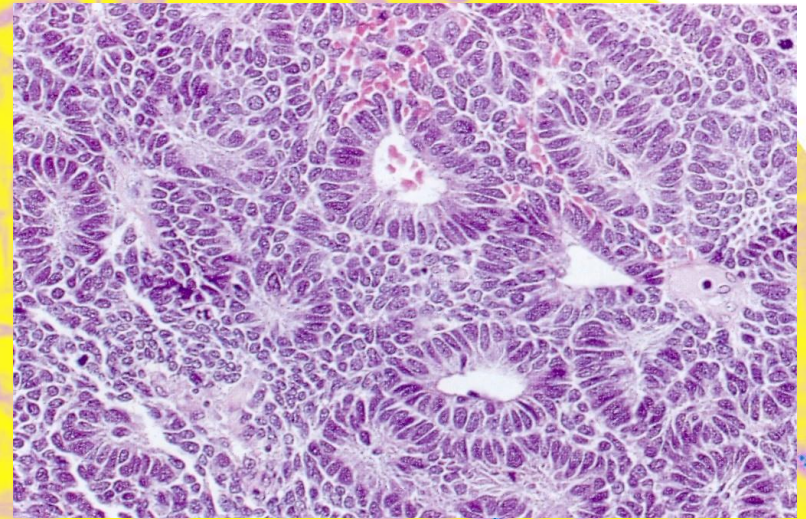
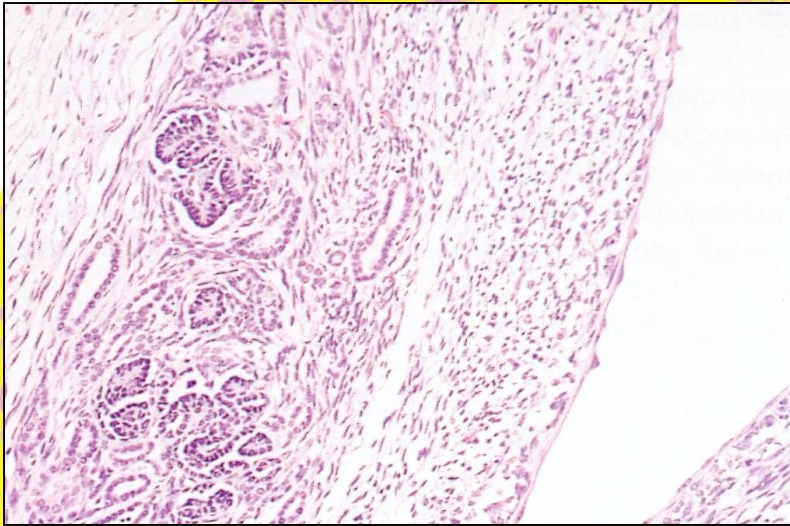


**MACROSCOPIC PICTURE OF TUMOR WITH FOCAL
HEMORRHAGIC NECROSIS**

**USUALLY ONE-SIDED TUMOR. 60% DEVELOP BEFORE THE 3RD YEAR OF LIFE. GENETIC
DELETION IN THE SHORT ARM OF THE 11TH CHROMOSOME.**

TUMORS IN CHILDREN

NEPHROBLASTOMA



THERE IS EMBRYONIC TISSUE IN STRUCTURE OF TUMOR, RARELY PRIMITIVE BLASTEMA, EPITHELIAL AS WELL AS MESODERMAL STRUCTURES – SMOOTH AND STRIATED MUSCLES, FIBROBLASTS AND CARTILAGE

THANK YOU

