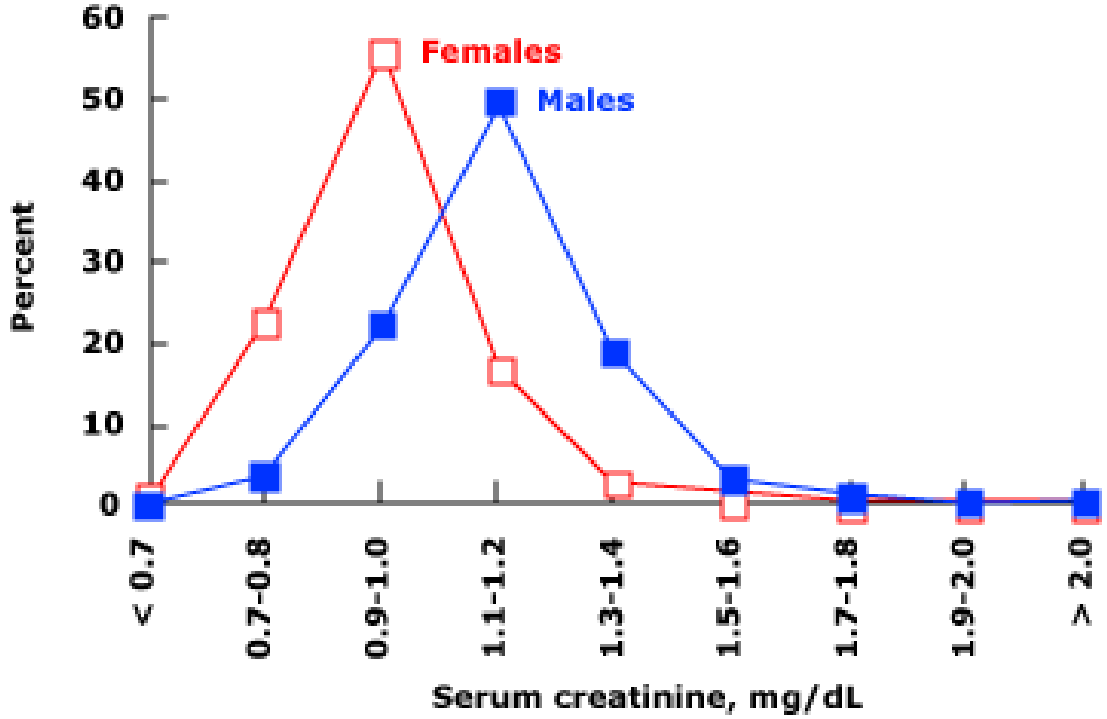
ΝΕΦΡΙΚΗ ΑΝΕΠΑΡΚΕΙΑ

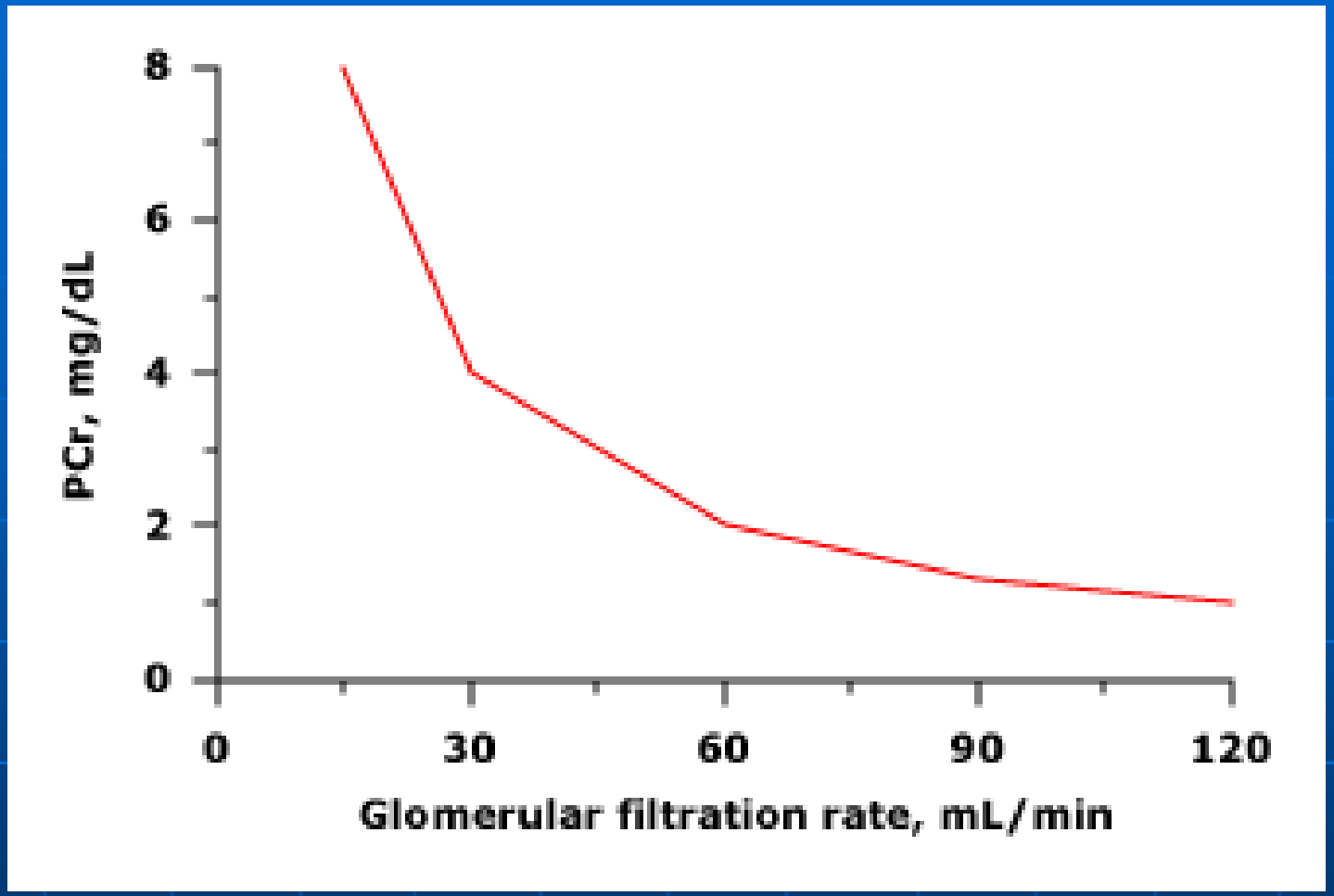
ΑΝΤΩΝΗΣ Ι. ΠΑΠΑΔΟΠΟΥΛΟΣ

ΕΠΙΚΟΥΡΟΣ ΚΑΘΗΓΗΤΗΣ
ΠΑΘΟΛΟΓΙΑΣ – ΛΟΙΜΩΞΕΩΝ

Δ΄ ΠΑΘΟΛΟΓΙΚΗ ΠΑΝΕΠ. ΚΛΙΝΙΚΗ
ΓΕΝΙΚΟ ΠΑΝΕΠΙΣΤΗΜΙΑΚΟ ΝΟΣΟΚΟΜΕΙΟ
«ΑΤΤΙΚΟΝ»

Distribution of serum creatinine





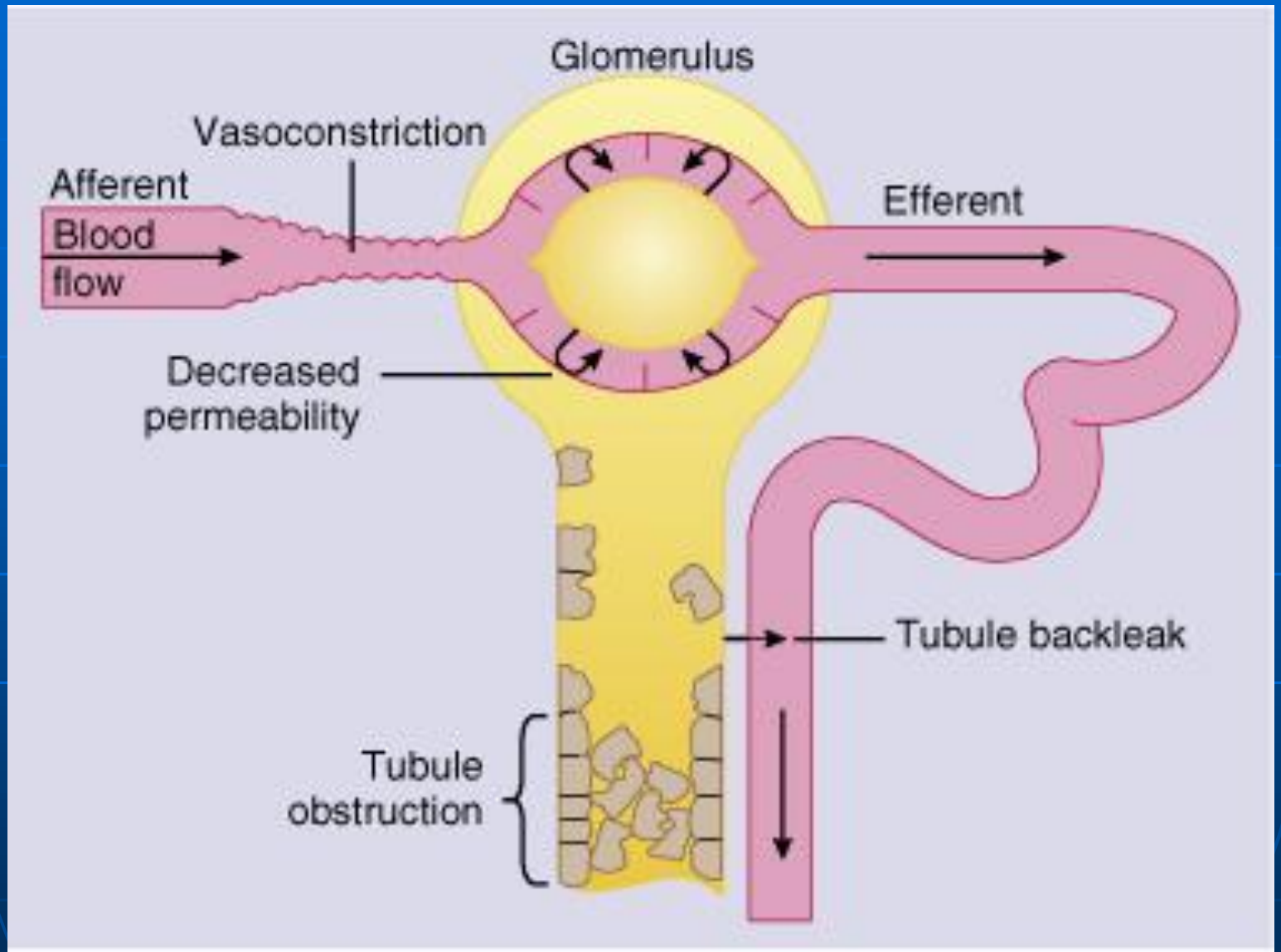
Idealized steady-state relationship between the plasma creatinine concentration (PCr) and the GFR. A fall in GFR decreases creatinine filtration and produces a proportionate rise in the plasma creatinine concentration.

ΟΞΕΙΑ ΝΕΦΡΙΚΗ ΑΝΕΠΑΡΚΕΙΑ

- ΤΑΧΕΙΑ ΠΤΩΣΗ GFR (ώρες / εβδομάδες)
- ΚΑΤΑΚΡΑΤΗΣΗ ΑΖΩΤΟΥΧΩΝ ΟΥΣΙΩΝ ΜΕΤΑΒΟΛΙΣΜΟΥ
- ΣΥΝΗΘΩΣ ΑΣΥΜΠΤΩΜΑΤΙΚΗ
- 50 % ΟΛΙΓΟΥΡΙΑ (< 400 ml ούρα / 24ωρο)

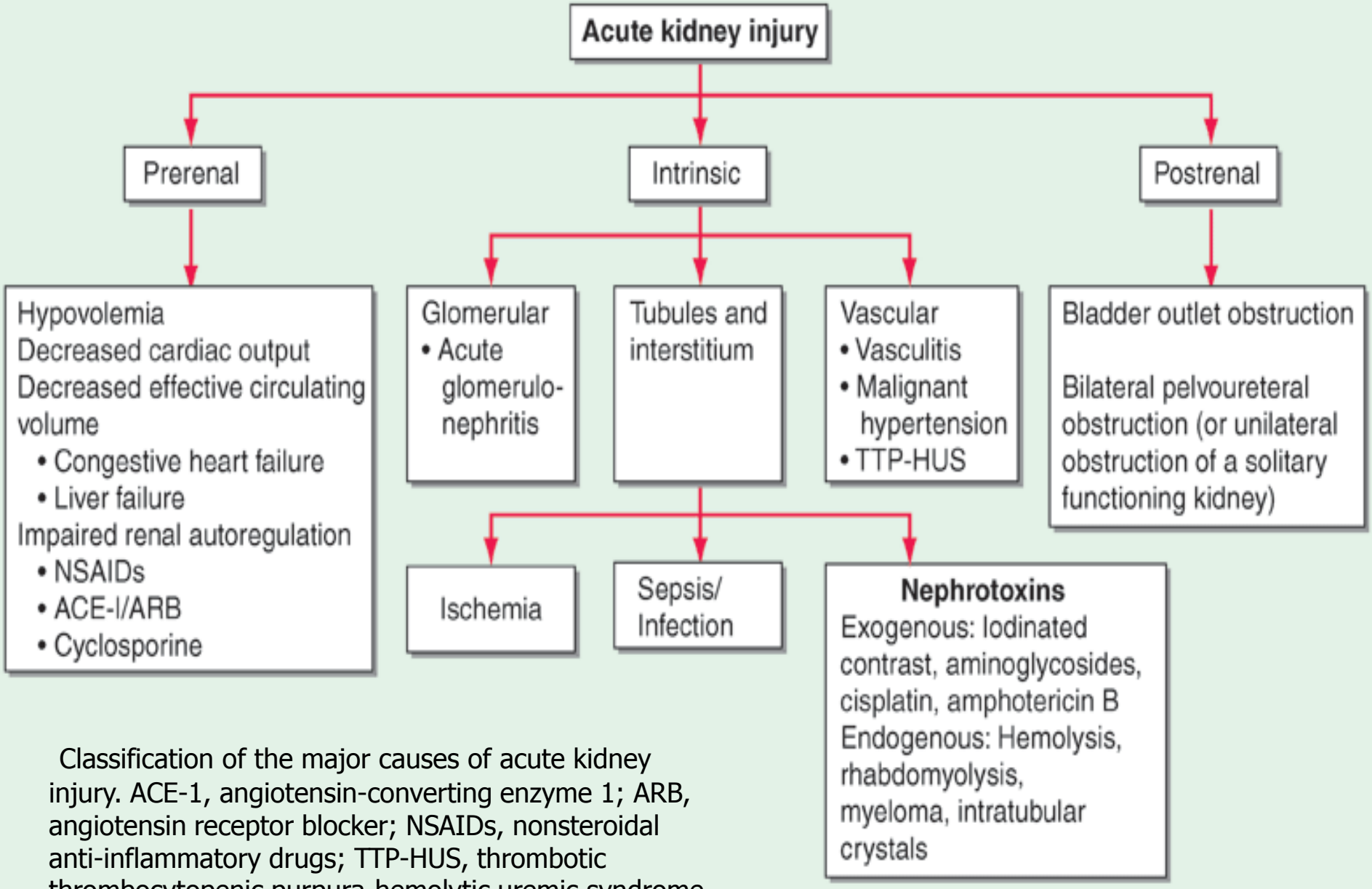
Οξεία νεφρική ανεπάρκεια

- Απότομη επιδείνωση της νεφρικής λειτουργίας
  - Μείωση του GFR
  - Αύξηση της κρεατινίνης
  - Ολιγουρία
    - <400 ml/24 ώρες ή <20 ml/hr ή <0.5 ml/kg hr
- Συχνότητα
  - 5% των εισαγωγών σε νοσοκομείο
  - 30% των ασθενών σε ΜΕΘ
- Πρόγνωση
  - >20% θνητότητα
  - Αναστρέψιμη στην πλειοψηφία αλλά 10-20% → αιμοκάθαρση



Αιτιολογία

- Προνεφρικά αίτια
  - Αφυδάτωση, αιμορραγία, μείωση κυκλοφορούντος όγκου
- Νεφρικά αίτια
  - Οξεία σωληναριακή νέκρωση
  - Νεφροτοξικά φάρμακα (ιωδιούχα σκιαγραφικά, μη στεροειδή αντιφλεγμονώδη, αμινογλυκοσίδες)
  - Σπειραματονεφρίτιδα
  - Ενδογενείς νεφροτοξίνες (μυοσφαιρίνη, ελαφρές αλυσίδες)
- Μετανεφρικά αίτια
  - απόφραξη



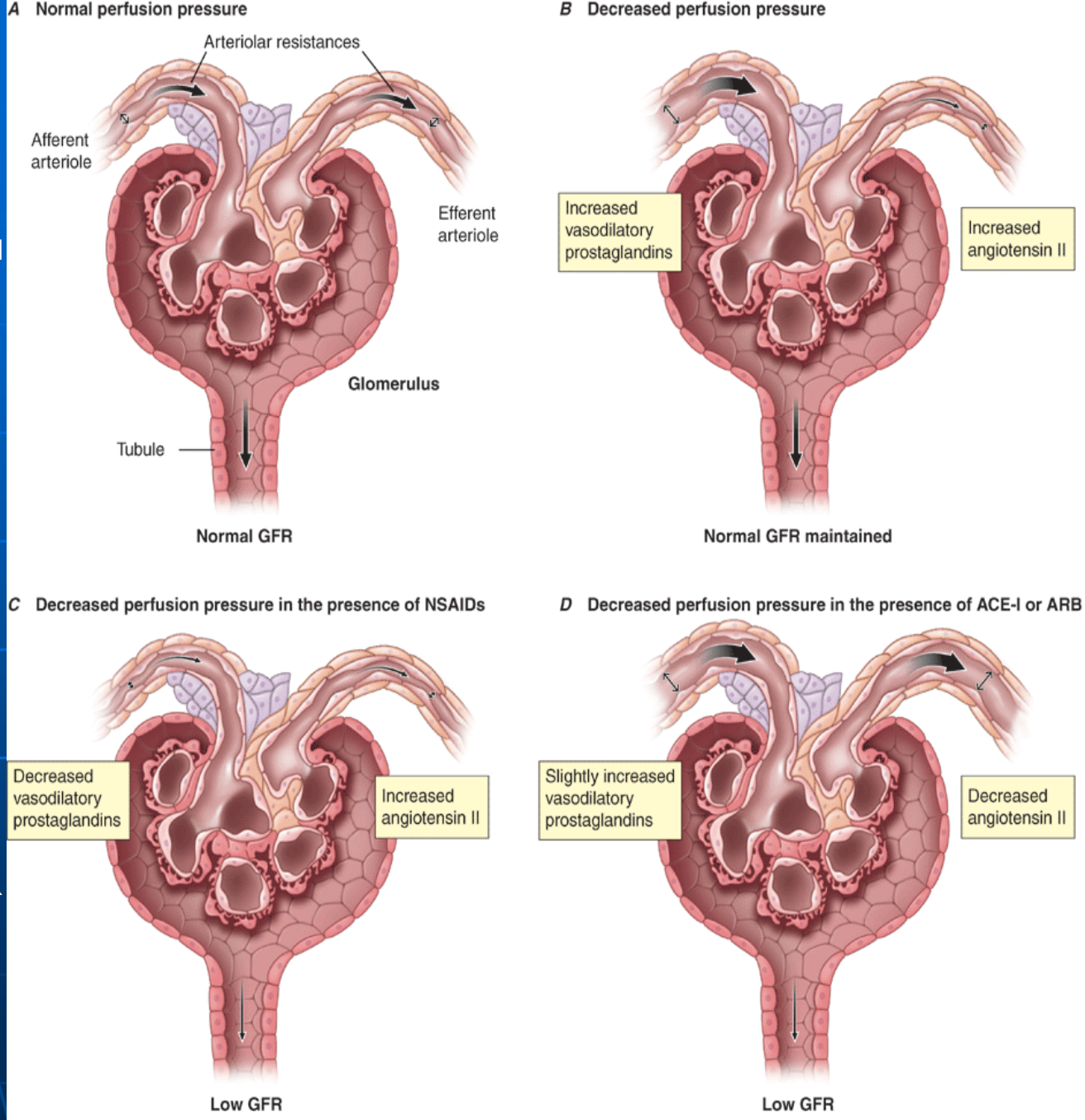
Classification of the major causes of acute kidney injury. ACE-1, angiotensin-converting enzyme 1; ARB, angiotensin receptor blocker; NSAIDs, nonsteroidal anti-inflammatory drugs; TTP-HUS, thrombotic thrombocytopenic purpura-hemolytic uremic syndrome.

Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 18th Edition: www.accessmedicine.com

Table 116–1 • CAUSES OF ACUTE RENAL FAILURE

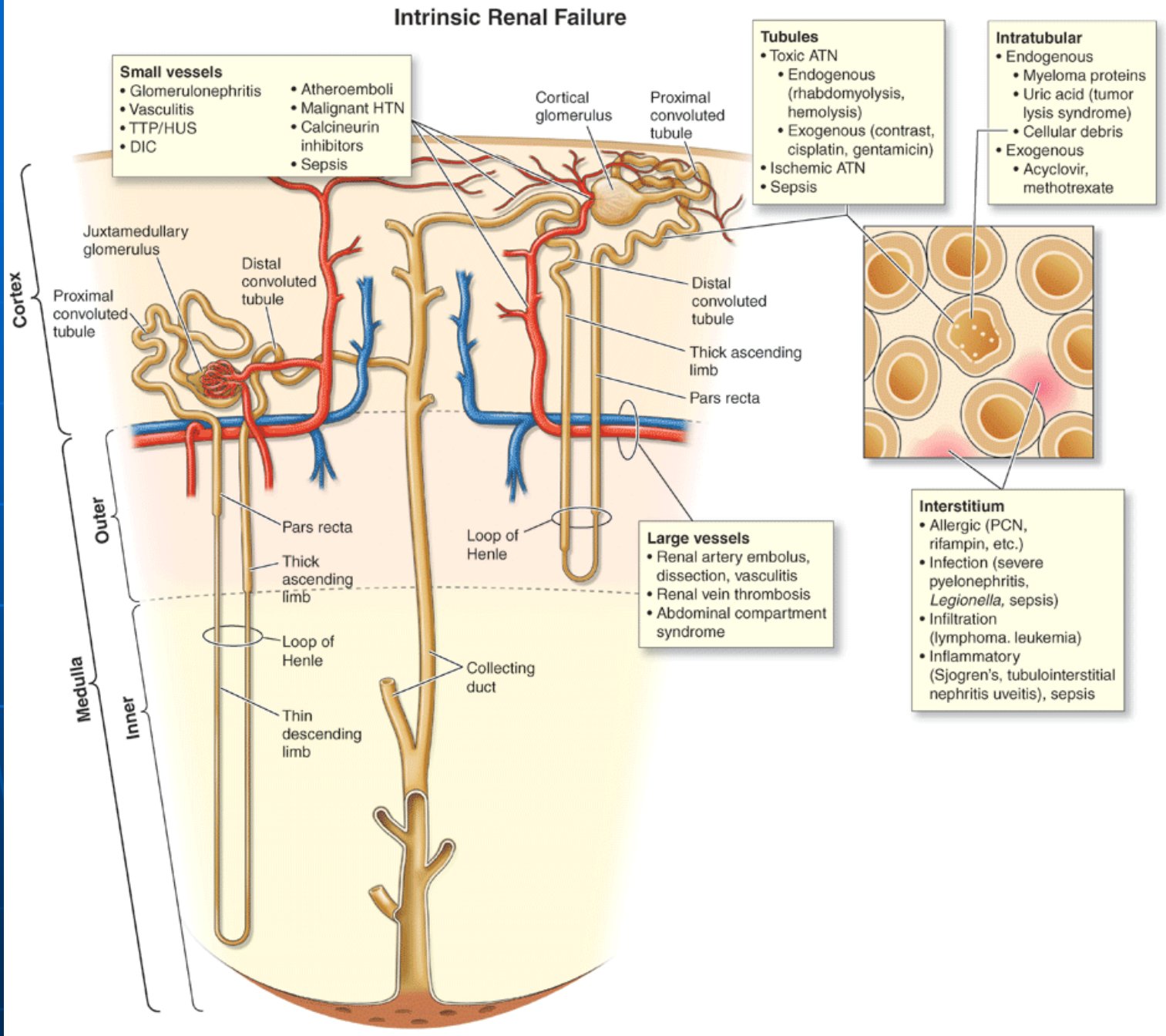
PRIMARY DISORDER	CLINICAL EXAMPLES
Prerenal	
Hypovolemia	Hemorrhage, skin losses (burns, sweating), gastrointestinal losses (diarrhea, vomiting), renal losses (diuretics, glycosuria), extravascular pooling (peritonitis, burns)
Ineffective arterial volume	Congestive heart failure, cardiac arrhythmias, sepsis, anaphylaxis, liver failure
Arterial occlusion	Bilateral arterial thromboembolism, thromboembolism of a solitary kidney, aortic or renal artery aneurysm
Postrenal	
Ureteral obstruction	Bilateral or in a solitary kidney (calculi, neoplasm, clot, retroperitoneal fibrosis, iatrogenic)
Urethral obstruction	Prostatitis, clot, calculus, neoplasm, foreign object
Venous occlusion	Bilateral or a solitary kidney (renal vein thrombosis, neoplasm, iatrogenic)
Intrarenal/Intrinsic	
Vascular	Vasculitis, microangiopathy, malignant hypertension, vasopressors, eclampsia, hyperviscosity states, hypercalcemia, iodinated radiocontrast agents
Glomerulars	Acute glomerulonephritis
Tubular injury	
Ischemia	Profound hypotension, postrenal transplant, vasopressors, microvascular constriction, sepsis
Endogenous proteins	Hemoglobinuria, myoglobinuria, light chain myeloma
Intratubular crystals	Uric acid, oxalate, sulfonamides, phenazopyridine
Tubulointerstitial inflammation	Interstitial nephritis caused by drugs, infection, radiation
Nephrotoxins	Antibiotics (aminoglycosides, cephaloridine, amphotericin B); metals (mercury, bismuth, uranium, arsenic, silver, cadmium, iron, antimony); solvents (carbon tetrachloride, ethylene glycol, tetrachloroethylene); iodinated contrast agents; antineoplastic agents (bleomycin, cisplatin)

Intrarenal Mechanisms for Autoregulation of the Glomerular Filtration Rate (GFR) under Decreased Perfusion Pressure and Reduction of the GFR by Drugs. Panel A shows normal conditions and a normal GFR. Panel B shows reduced perfusion pressure within the autoregulatory range. Normal glomerular capillary pressure is maintained by afferent vasodilatation and efferent vasoconstriction. Panel C shows reduced perfusion pressure with a nonsteroidal anti-inflammatory drug (NSAID). Loss of vasodilatory prostaglandins increases afferent resistance; this causes the glomerular capillary pressure to drop below normal values and the GFR to decrease. Panel D shows reduced perfusion pressure with an angiotensin-converting enzyme (ACE-I) inhibitor or an angiotensin receptor blocker (ARB). Loss of angiotensin II action reduces efferent resistance; this causes the glomerular capillary pressure to drop below normal values and the GFR to decrease. *(From N Engl J Med 2007;357:797-805; with permission.)*

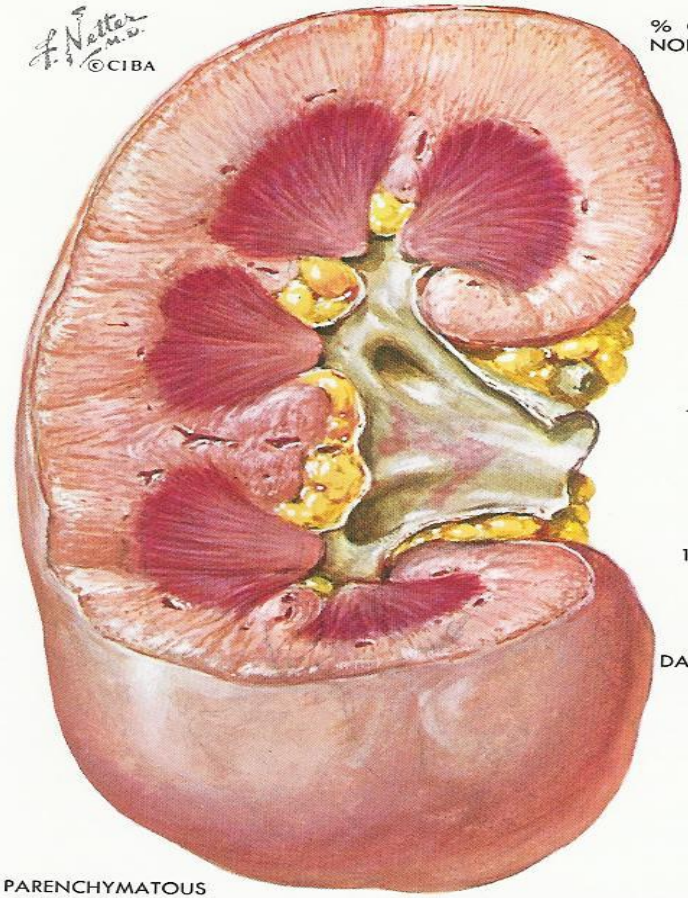


Major causes of intrinsic acute kidney injury.

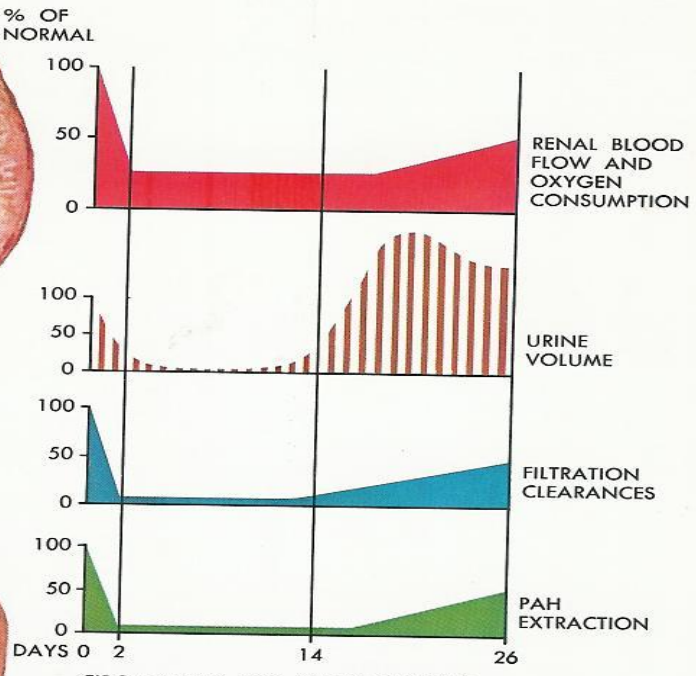
ATN, acute tubular necrosis; DIC, disseminated intravascular coagulation; HTN, hypertensive nephropathy; MTX, methotrexate; PCN, penicillin; TTP/HUS, thrombotic thrombocytopenic purpura/hemolytic uremic syndrome; TINU, tubulointerstitial nephritis-uveitis.



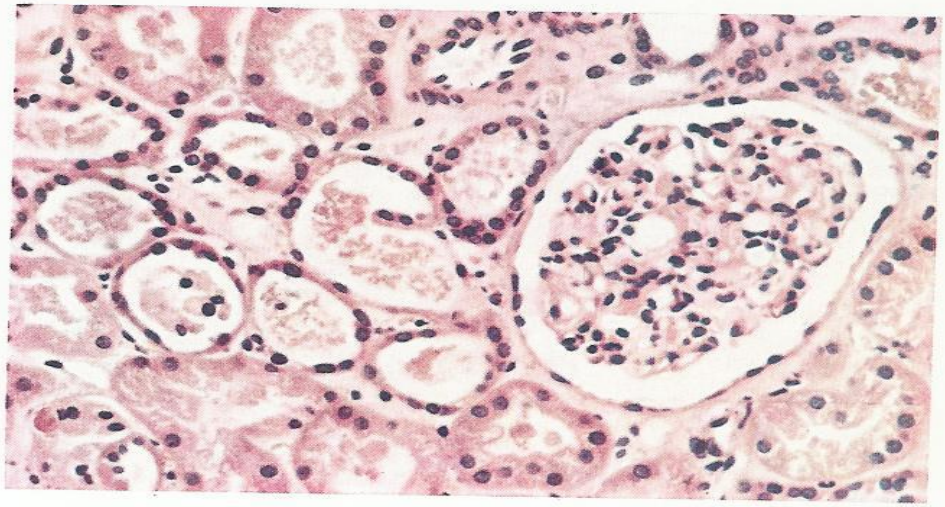
Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 18th Edition: www.accessmedicine.com



PARENCHYMATOUS ACUTE RENAL FAILURE: LARGE, PALE KIDNEY WITH THICK, PALE CORTEX AND DARK HYPEREMIC PYRAMIDS

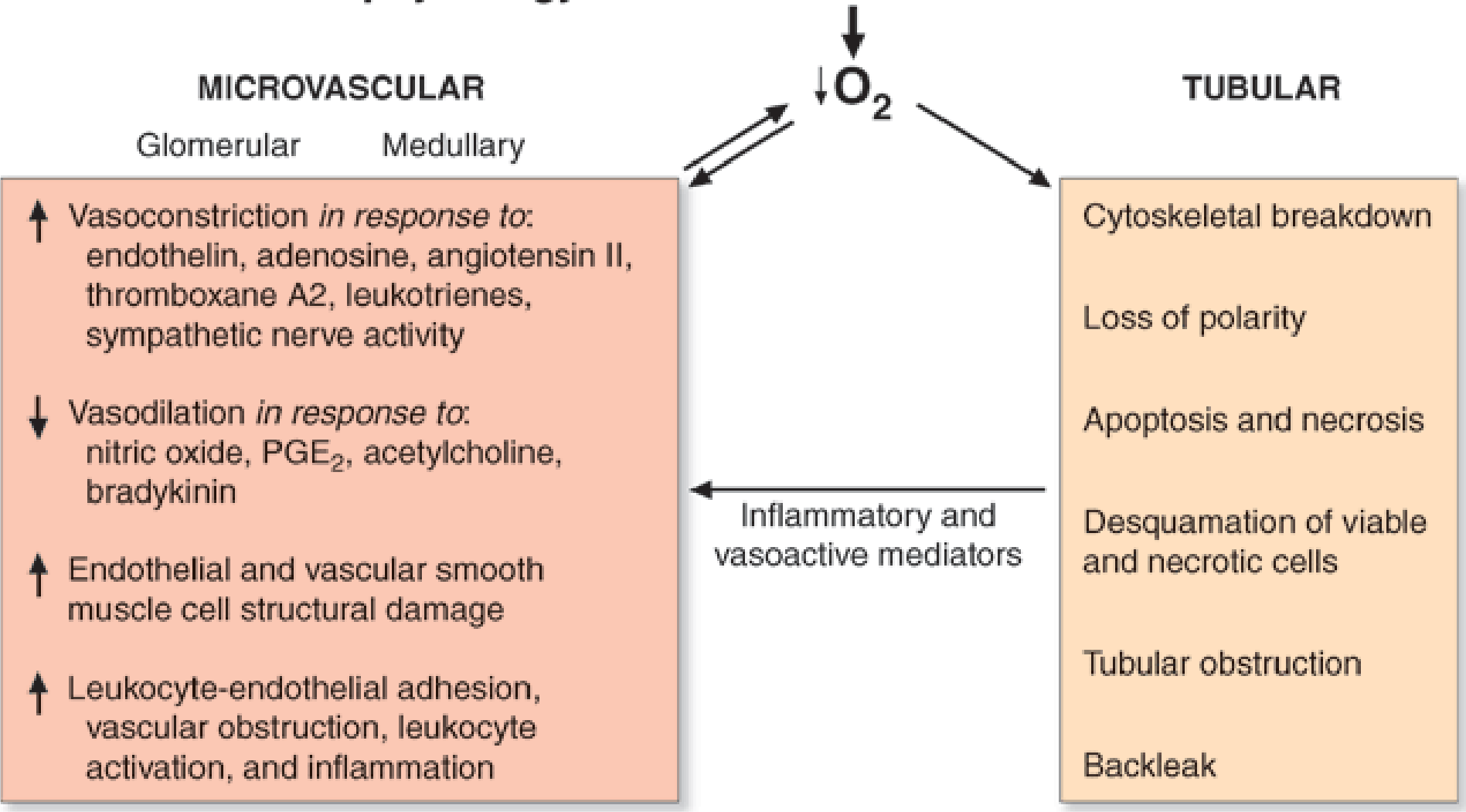


CIRCULATORY AND OTHER SELECTED FUNCTIONAL DISTURBANCES IN THE COURSE OF ACUTE RENAL FAILURE: URINE FLOW, FILTRATION CLEARANCES, AND PAH EXTRACTION ARE MUCH MORE AFFECTED THAN RENAL BLOOD FLOW AND OXYGEN CONSUMPTION



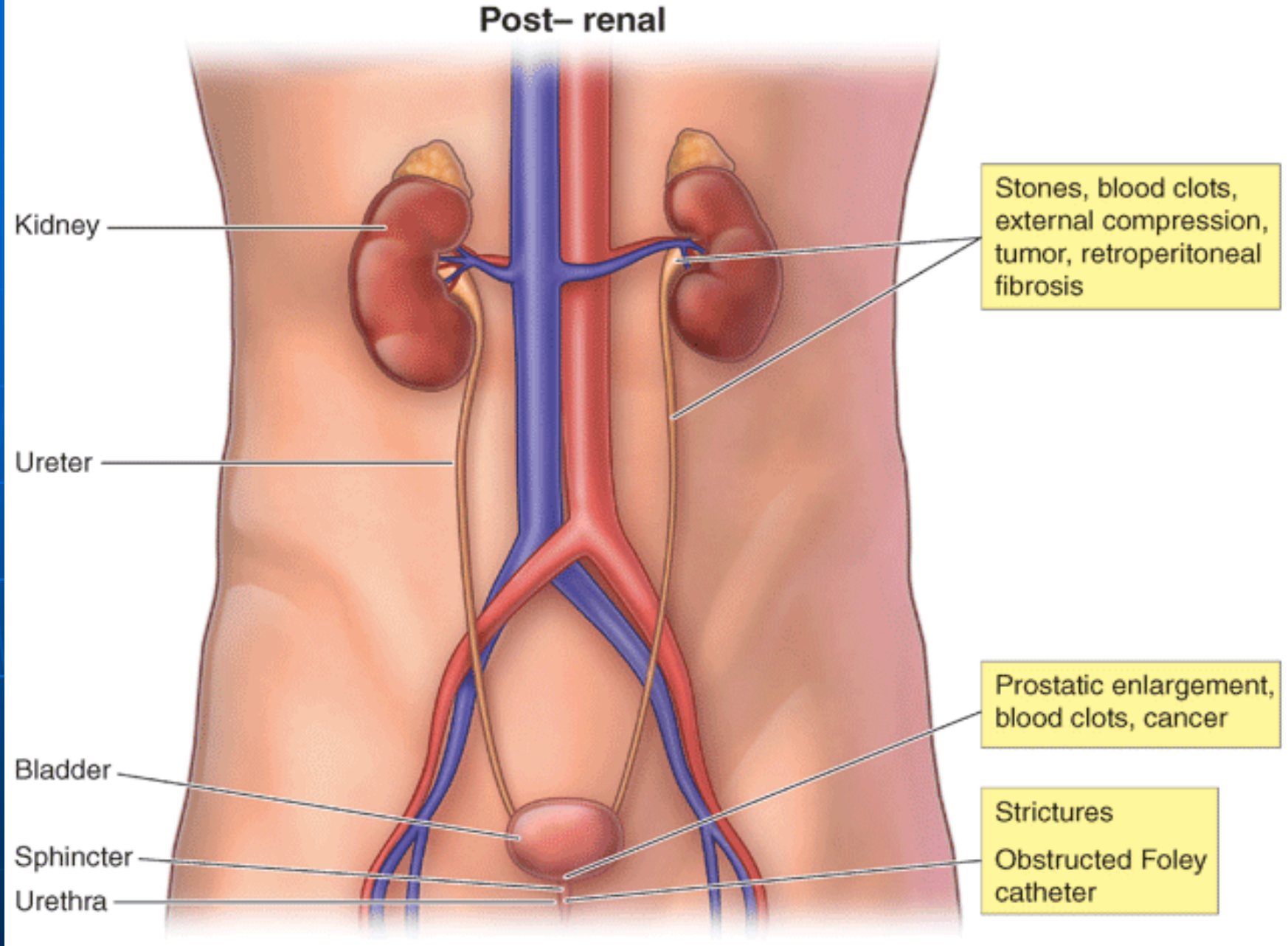
BIOPSY SECTION: GLOMERULUS NORMAL; DISTAL CONVOLUTED TUBULES DILATED, WITH FLATTENED EPITHELIUM, "PRETZEL–LIKE" DISTORTION, AND CONTAINING HEME CASTS (H. & E. STAIN)

Pathophysiology of Ischemic Acute Renal Failure



Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 18th Edition: www.accessmedicine.com

Post– renal



Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 18th Edition: www.accessmedicine.com

Διευρεύνηση

- Ιστορικό !!!
- Γενική εξέταση ούρων
- Υπερηχογράφημα νεφρών - κύστης
- Ηλεκτρολύτες, κρεατινίνη ούρων
- Ανάλογα με τις ενδείξεις
  - Ανοσολογικός έλεγχος
  - Αγγειογραφία νεφρού
  - Βιοψία νεφρού

Table 116–2 • SYSTEMATIC APPROACH TO DIAGNOSING THE CAUSE OF ACUTE RENAL FAILURE

1. Medical history: clinical setting, medications
2. Physical examination: postural changes in blood pressure and evaluation of hemodynamic status, skin rash, signs of systemic diseases
3. Urinalysis with evaluation of sediment
4. Chemical analysis of blood and urine: serum bicarbonate, potassium, uric acid, calcium, phosphorus, urine osmolality, urine and serum urea, creatinine, sodium
5. Bladder catheterization
6. Fluid-diuretic challenge
7. Radiologic studies to exclude obstruction: ultrasonography, CT scan, or retrograde pyelography
8. Renal biopsy

Table 116–3 • URINARY INDICES IN ACUTE RENAL FAILURE

LABORATORY TEST	PRERENAL	ACUTE TUBULAR INJURY
Urinary osmolality (mOsm/kg H_2O)	>500	<350
Urinary sodium (mEq/L)	<20	>40
Urinary/plasma creatinine ratio	>40	<20
Fractional sodium excretion*	<1	>1

$$*\frac{\text{Urine [Na]/serum [Na]}}{\text{Urine [creatinine]/serum [creatinine]}} \times 100.$$

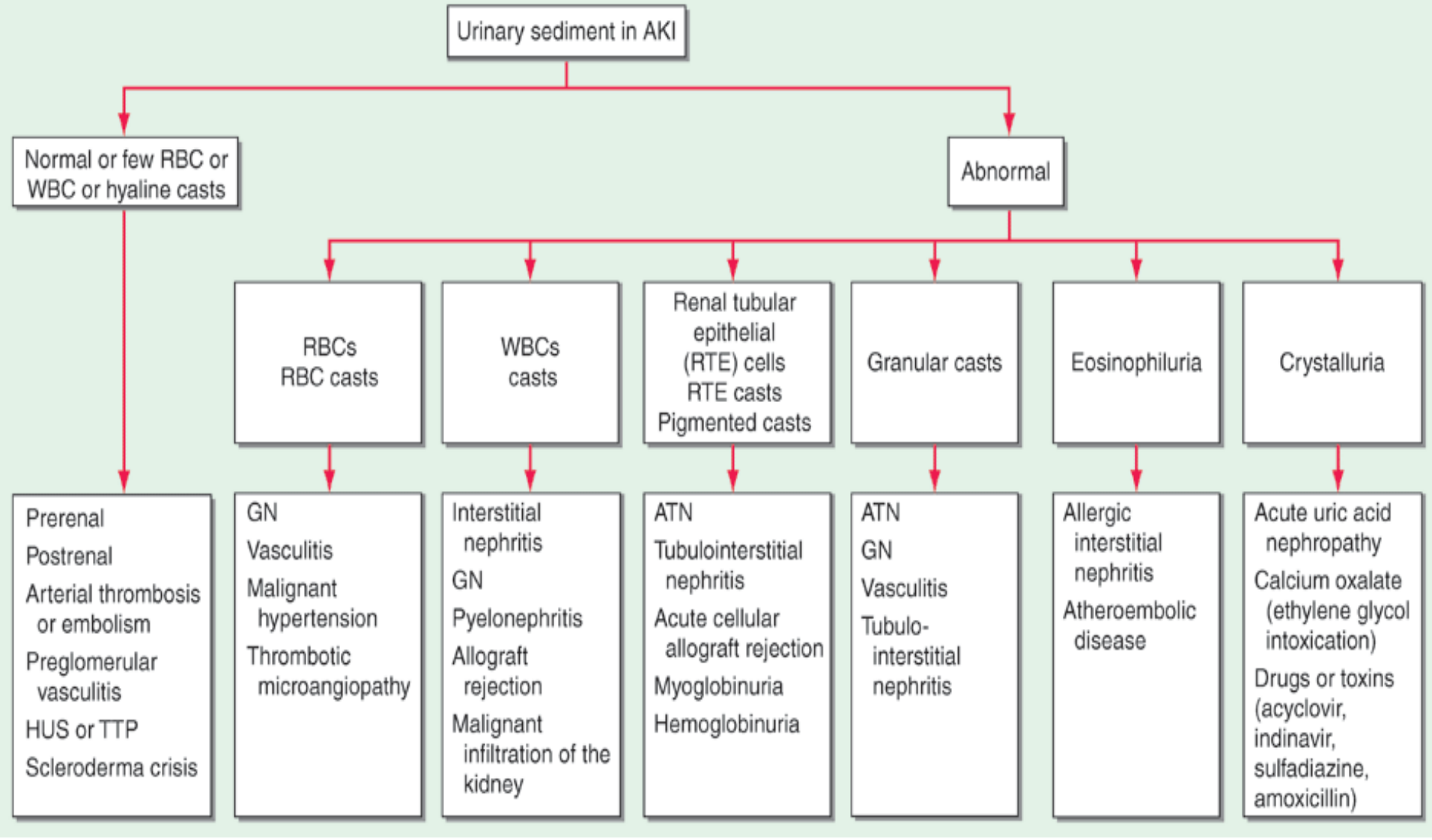
Table 116–4 • CONDITIONS ASSOCIATED WITH FRACTIONAL SODIUM EXCRETION LESS THAN 1% DESPITE INTRINSIC RENAL DAMAGE

Intense Intrarenal Vasoconstriction
Liver disease
Congestive heart failure
Norepinephrine administration
Severe burns, sepsis
Nonsteroidal anti-inflammatory drugs
Acute bilateral ureteral obstruction
Iodinated radiocontrast agents
Vascular Inflammation
Acute glomerulonephritis
Acute vasculitis
Renal transplant rejection

Table 116–5 • DIAGNOSTIC CLUES TO THE CAUSE OF ACUTE RENAL FAILURE

PRIMARY DISORDER	URINALYSIS	CLINICAL FINDINGS
Prerenal		
Hypovolemia	Hyaline casts, no RBC, or WBC, low FE_{Na}	Rapid weight loss, postural hypotension
Ineffective arterial volume	Hyaline casts, no RBC, or WBC, low Fe_{Na}	Weight gain, edema, normal or low blood pressure
Arterial occlusion	Hyaline casts, rare to many RBCs	Occasional flank or low back pain
Postrenal		
Ureteral obstruction	WBCs if infected, crystals or RBCs	Flank pain radiating into the groin
Urethral	WBCs and RBCs	Urethral pain
Venous occlusion	Proteinuria, hematuria	Occasional flank pain
Renal		
Vascular	Granular casts, proteinuria, RBCs and WBCs	Systemic illness suggesting vasculitis, hypertension
Glomerular	RBC casts, granular casts, RBCs, WBCs, proteinuria	Systemic illness, hypertension
Tubular	Granular casts, tubular cells, RBCs, WBCs	Hypotension, sepsis

FE_{Na} = fractional sodium excretion; RBC = red blood cell; WBC = white blood cell.



Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 18th Edition: www.accessmedicine.com

Interpretation of urinary sediment findings in acute kidney injury.

GN, glomerulonephritis; RTE, renal tubular epithelial

Επιπλοκές

- Υπερφόρτωση υγρών
- Ηλεκτρολυτικές διαταραχές
  - Υπερκαλιαιμία, υπερφωσφαταιμία
- Μεταβολική οξέωση
- Αναιμία
- Ουραιμία
- Πολυουρική φάση
- Δυσχέρεια χορήγησης φαρμάκων

Αντιμετώπιση

- Διόρθωση αναστρέψιμων παραγόντων
  - Αποκατάσταση ενδαγγειακού όγκου
  - Διακοπή νεφροτοξικών φαρμάκων
  - Απομάκρυνση ενδογενών νεφροτοξινών (π.χ. ουρικό οξύ, μυοσφαιρίνη)
- Ισοζύγιο υγρών
- Διουρητικά, περιορισμός ύδατος και αλατιού
- Διττανθρακικά (αν pH<7.2)
- Προσαρμογή δόσεων φαρμάκων

Ενδείξεις αιμοκάθαρσης

- Υπερκαλιαιμία
- Υπερφόρτωση όγκου (πνευμονικό οίδημα) χωρίς ανταπόκριση σε διουρητικά
- Επιδεινούμενη οξέωση
- Ουραιμικά συμπτώματα (επιληπτικές κρίσεις, εγκεφαλοπάθεια, περικαρδίτιδα)

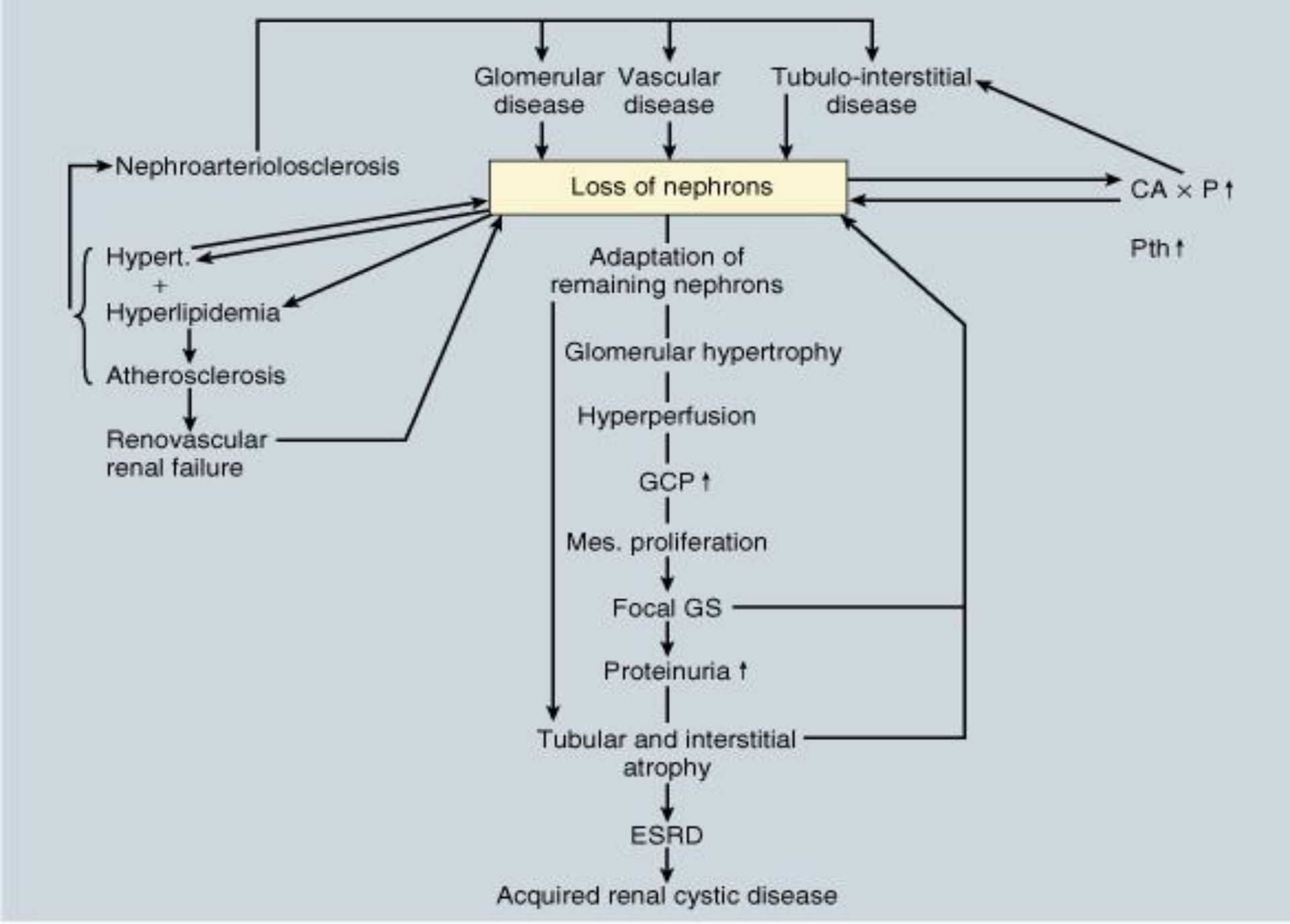
ΧΡΟΝΙΑ ΝΕΦΡΙΚΗ ΑΝΕΠΑΡΚΕΙΑ

- ΝΕΦΡΙΚΗ ΒΛΑΒΗ ΜΕ ΜΕΙΩΣΗ ΝΕΦΡΙΚΗΣ ΜΑΖΑΣ
- ΑΡΧΙΚΑ ΑΝΤΙΡΡΟΠΟΥΜΕΝΗ (λόγω υπερτροφίας και υπερλειτουργίας υπολειπομένων νεφρώνων)
- ΤΕΛΙΚΑ ΜΟΝΙΜΗ ΒΛΑΒΗ
- ΜΙΚΡΟΙ ΝΕΦΡΟΙ
- ΑΖΩΘΑΙΜΙΑ > 3 ΜΗΝΕΣ
- ΕΥΡΕΙΣ ΚΥΛΙΝΔΡΟΙ
- ΟΥΡΑΙΜΙΑ: σημεια και συμπτώματα ΧΝΑ, ανεξαρτήτως αιτίου

Χρόνια νεφρική ανεπάρκεια
Ορισμοί

- Παρουσία δεικτών νεφρικής βλάβης για > 3 μήνες ή
- GFR < 60 ml/min για > 3 μήνες

- GFR < 10-15 ml/min = τελικού σταδίου νεφροπάθεια (end stage renal disease ESRD)



Παθοφυσιολογία της εξέλιξης της χρόνιας νεφρικής νόσου

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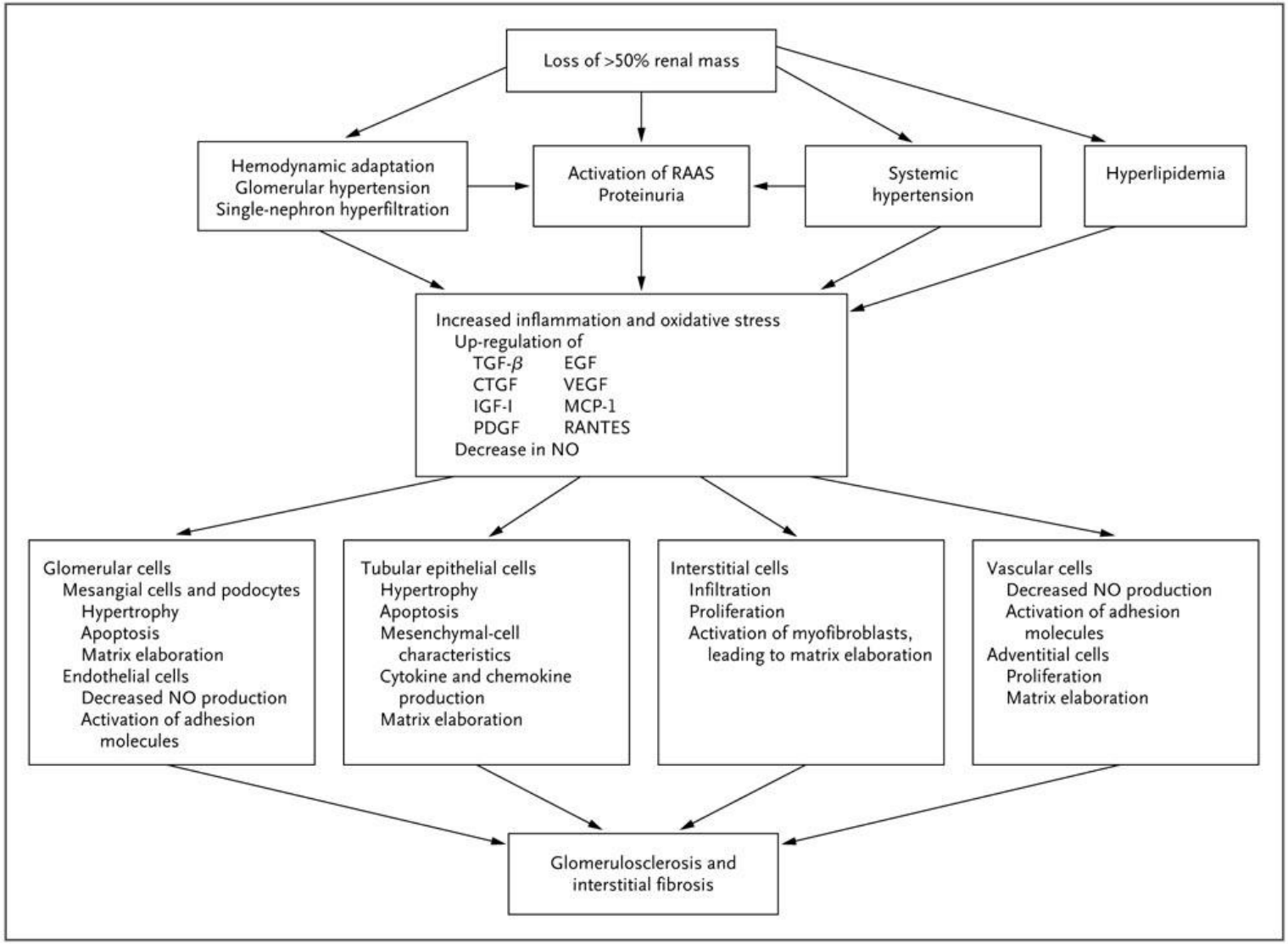
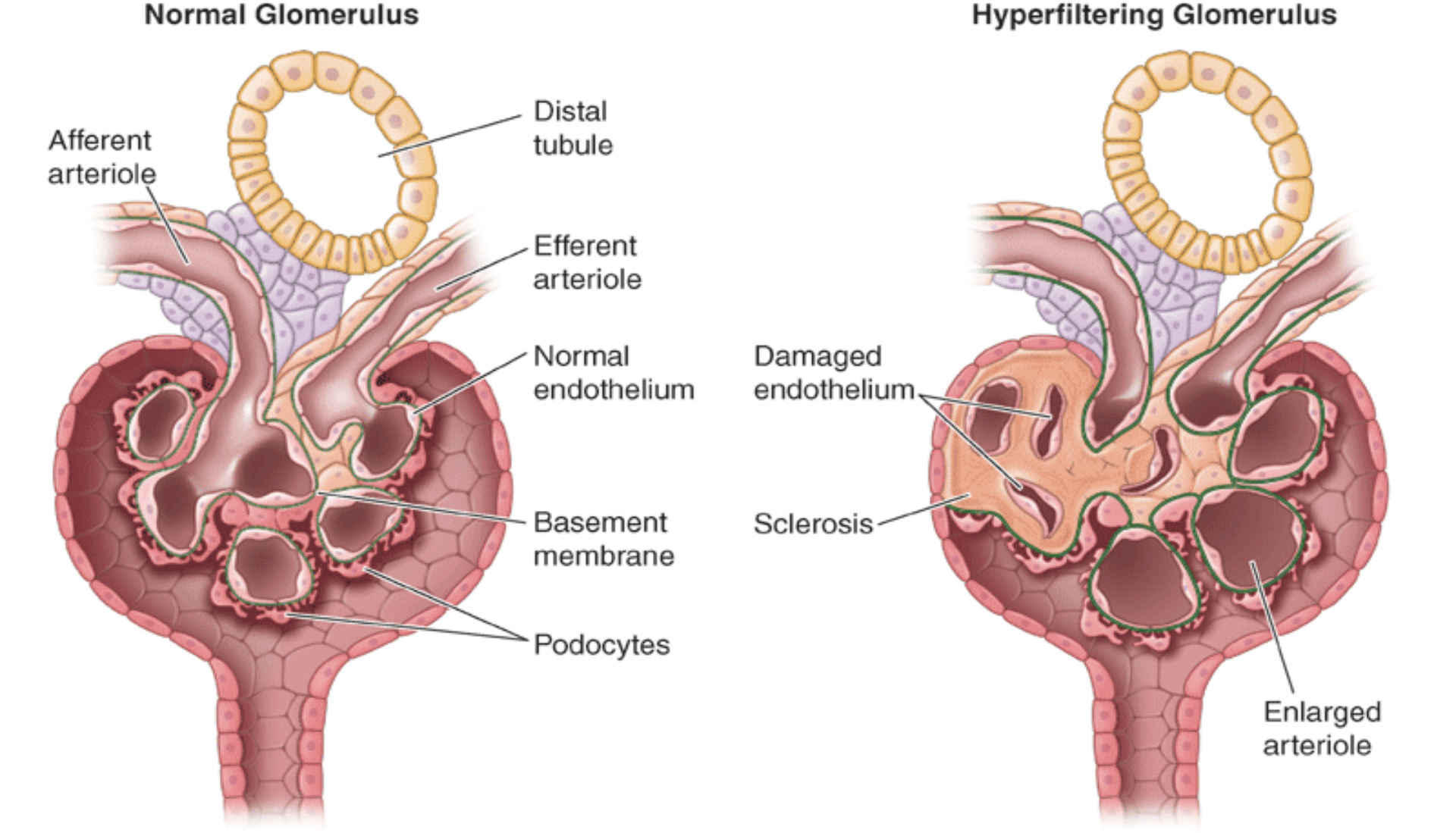
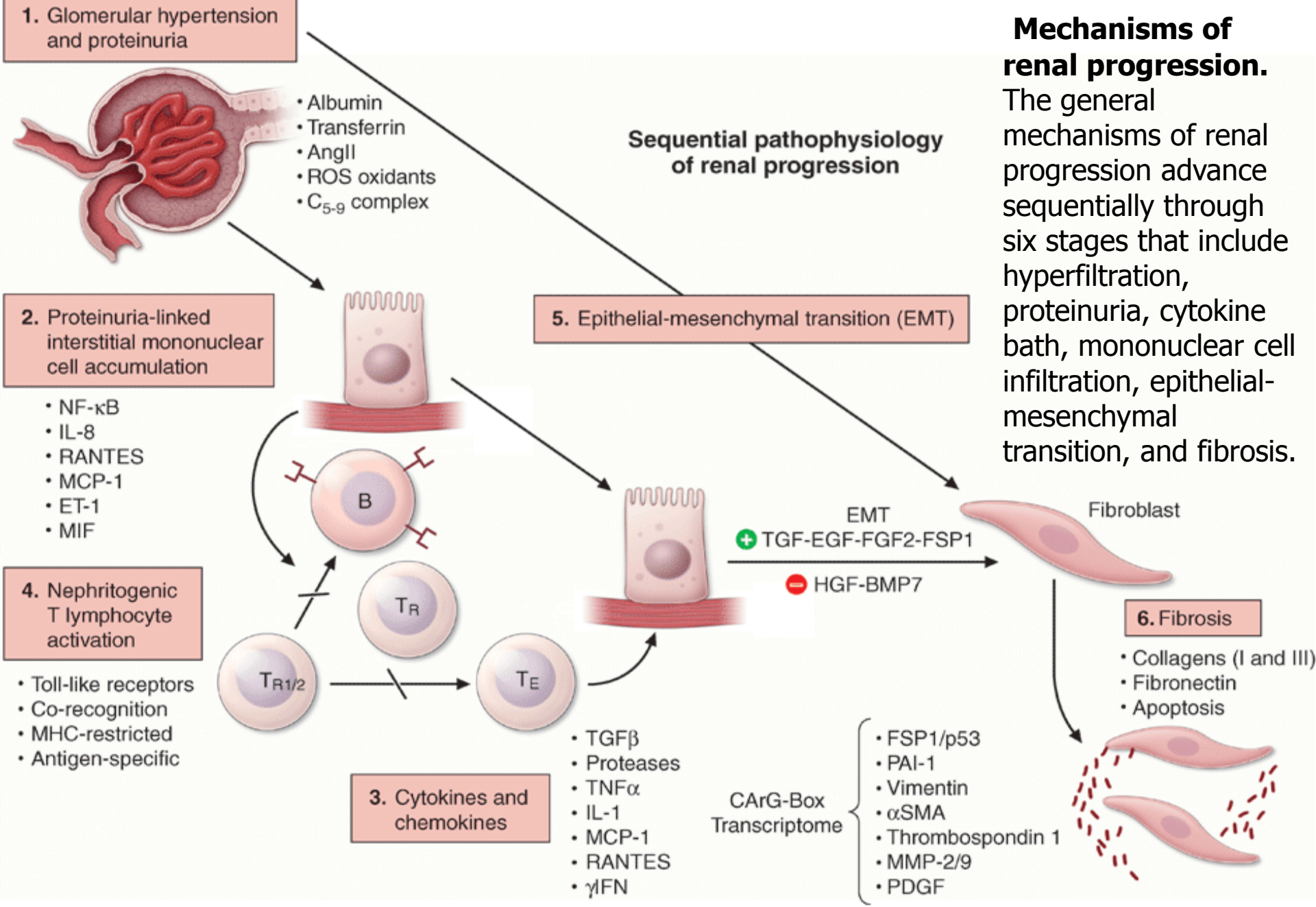


Abboud H and Henrich W. N Engl J Med 2010;362:56-65



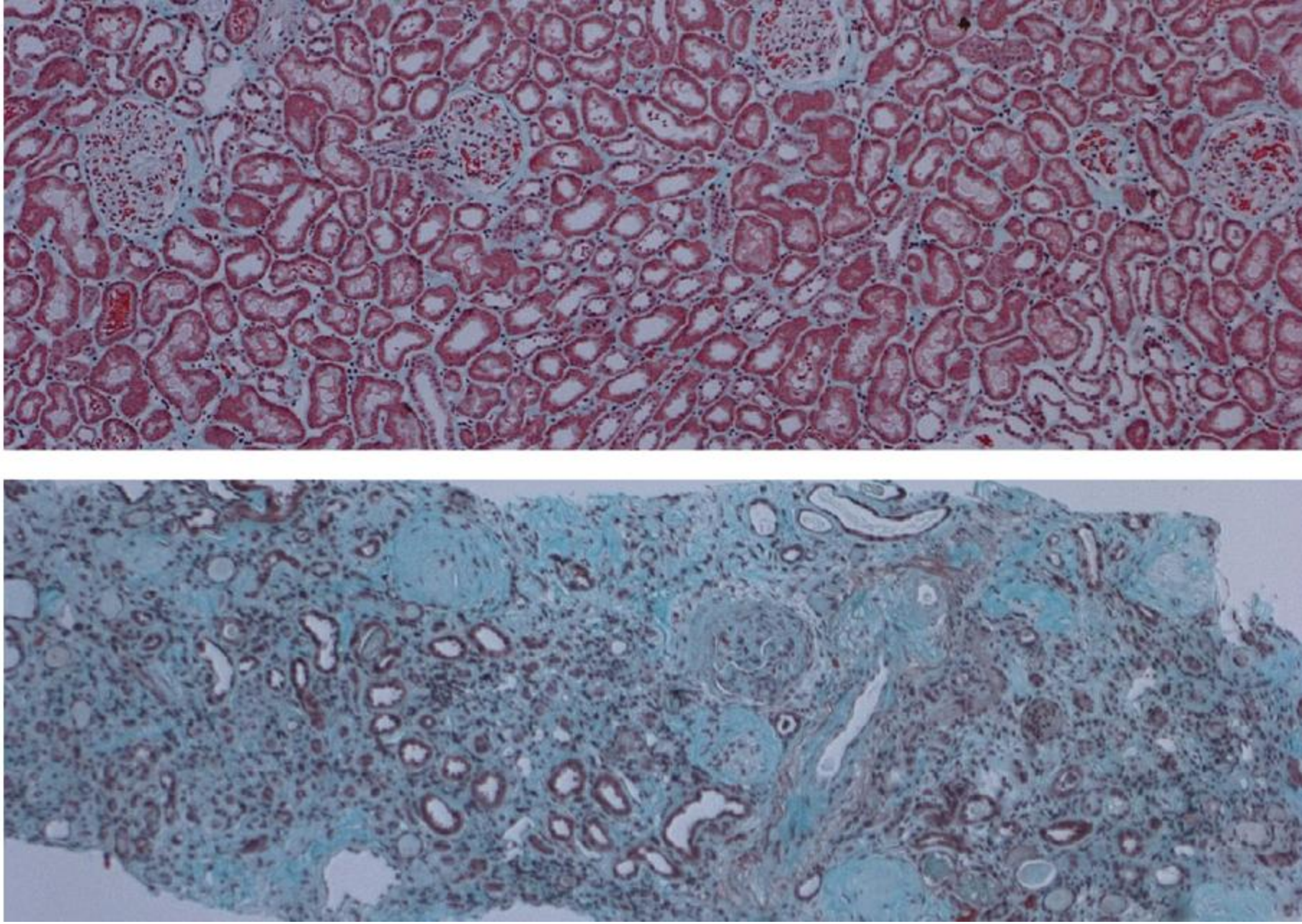
Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 18th Edition: www.accessmedicine.com

Left: Schema of the normal glomerular architecture. Right: Secondary glomerular changes associated with a reduction in nephron number, including enlargement of capillary lumens and focal adhesions, which are thought to occur consequent to compensatory hyperfiltration and hypertrophy in the remaining nephrons. (*Modified from JR Ingelfinger: N Engl J Med 348:99, 2003.*)

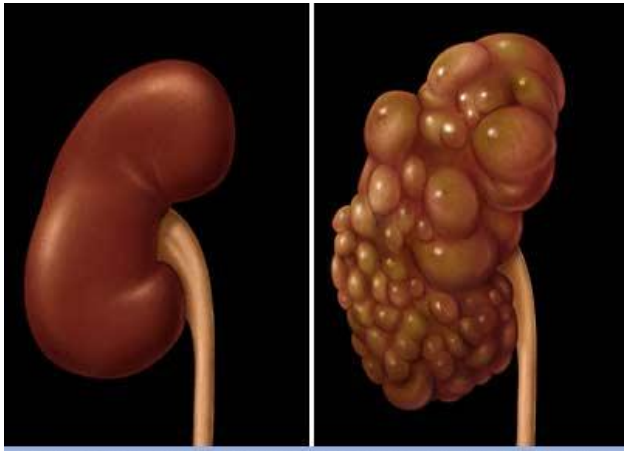


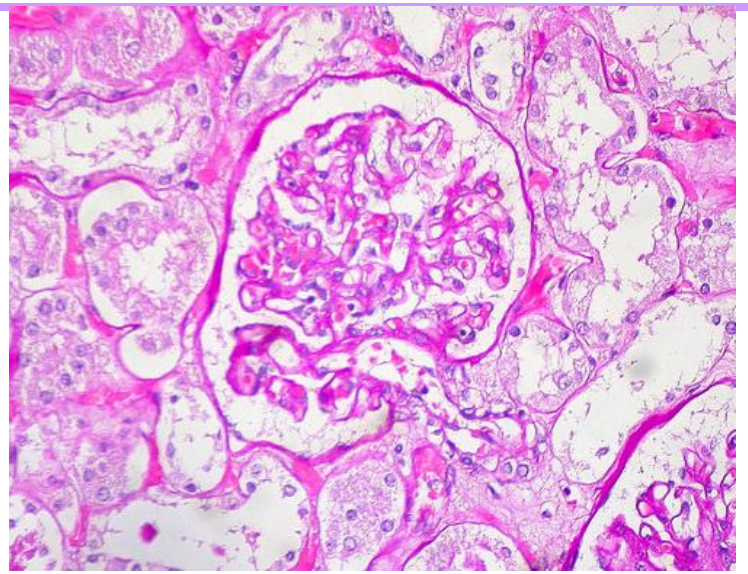
Mechanisms of renal progression. The general mechanisms of renal progression advance sequentially through six stages that include hyperfiltration, proteinuria, cytokine bath, mononuclear cell infiltration, epithelial-mesenchymal transition, and fibrosis.

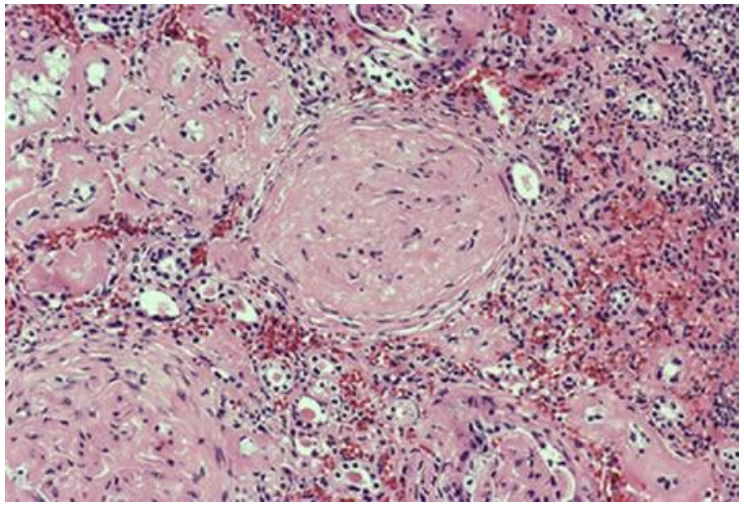
Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 18th Edition: www.accessmedicine.com

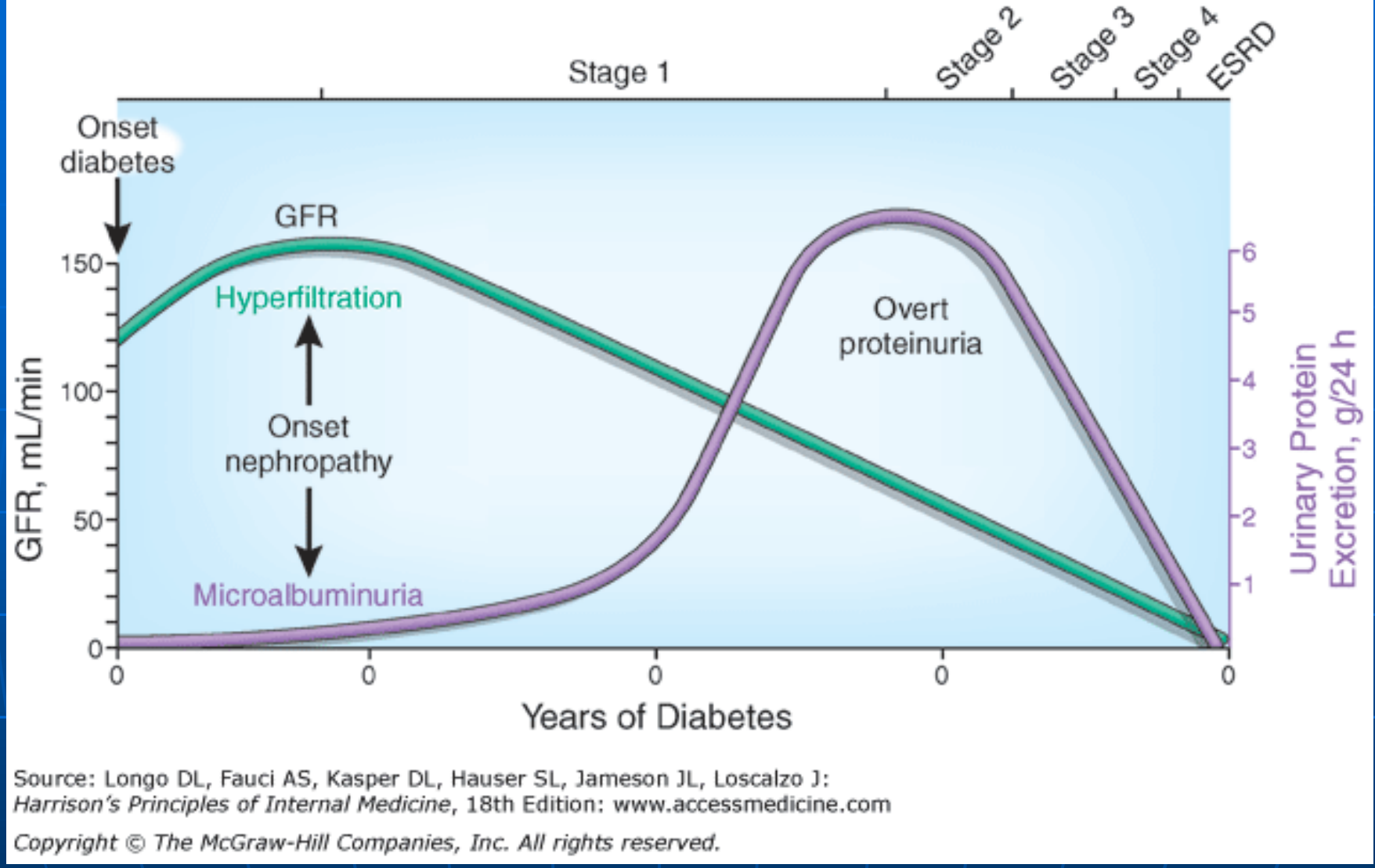


Low-power photomicrograph of a normal kidney showing normal glomeruli and healthy tubulointerstitium without fibrosis. Right: Low-power photomicrograph of chronic kidney disease with sclerosis of many glomeruli and severe tubulointerstitial fibrosis (*Masson trichrome, × 40)*









Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 18th Edition: www.accessmedicine.com

Progression of chronic renal injury. Although various types of renal injury have their own unique rates of progression, one of the best understood is that associated with type I diabetic nephropathy. Notice the early increase in glomerular filtration rate, followed by inexorable decline associated with increasing proteinuria. Also indicated is the National Kidney Foundation K/DOQI classification of the stages of chronic kidney disease.

Table 10. Stages of Chronic Kidney Disease

Stage	Description	GFR (mL/min/1.73 m^2)
1	Kidney damage with normal or ↑ GFR	≥90
2	Kidney damage with mild ↓ GFR	60–89
3	Moderate ↓ GFR	30–59
4	Severe ↓ GFR	15–29
5	Kidney failure	<15 (or dialysis)

Chronic kidney disease is defined as either kidney damage or GFR <60 mL/min/1.73 m^2 for ≥3 months. Kidney damage is defined as pathologic abnormalities or markers of damage, including abnormalities in blood or urine tests or imaging studies.

Κύρια αίτια χρονίας νεφρικής ανεπάρκειας

Table 2. Major Causes of Severe Chronic Kidney Disease.*

Cause	Percent of Cases†
Diabetes mellitus	44.9
Type 1	3.9
Type 2	41.0
Hypertension	27.2
Glomerulonephritis	8.2
Chronic interstitial nephritis or obstruction	3.6
Hereditary or cystic disease	3.1
Secondary glomerulonephritis or vasculitis	2.1
Neoplasms or plasma-cell dyscrasias	2.1
Miscellaneous conditions‡	4.6
Uncertain or unrecorded cause	5.2

* Data are from the U.S. Renal Data System.[3]
† The percentages are based on the incidence of reported end-stage renal disease according to the primary diagnosis.
‡ Examples of miscellaneous conditions are irreversible acute kidney injury and nephropathy associated with the acquired immunodeficiency syndrome.

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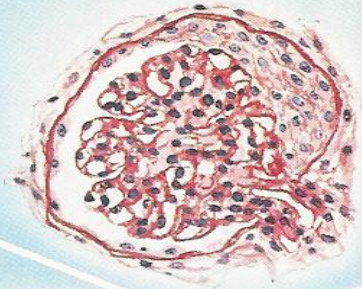
ETIOLOGY OF CHRONIC RENAL FAILURE

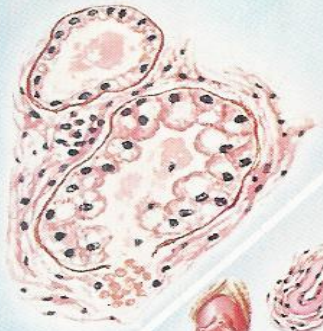
1: PRIMARILY GLOMERULAR DISEASE

ACUTE GLOMERULONEPHRITIS
ANTIGLOMERULAR BASEMENT MEMBRANE DISEASE
CHRONIC GLOMERULONEPHRITIS
FOCAL GLOMERULONEPHRITIS
GOODPASTURE'S SYNDROME
INTERCAPILLARY GLOMERULOSCLEROSIS
RAPIDLY PROGRESSIVE GLOMERULONEPHRITIS



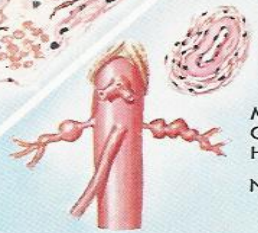
2: PRIMARILY TUBULAR DISEASE

CHRONIC HYPERCALCEMIA
CHRONIC POTASSIUM DEPLETION
FANCONI SYNDROME AND VARIANTS
HEAVY METAL POISONING (LEAD, CADMIUM, etc.)



3: VASCULAR DISEASE

ISCHEMIC DISEASE OF KIDNEYS, CONGENITAL OR ACQUIRED
BILATERAL RENAL ARTERY STENOSIS; BILATERAL FIBROMUSCULAR HYPERPLASIA
MALIGNANT PHASE OF ESSENTIAL HYPERTENSION
NEPHROSCLEROSIS



4: INFECTIONS

CHRONIC PYELONEPHRITIS
TUBERCULOSIS

5: OBSTRUCTIVE DISEASE

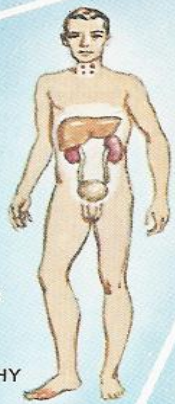
UPPER
CALCULI
NEOPLASMS
RETROPERITONEAL FIBROSIS
LOWER
CONGENITAL ANOMALIES OF BLADDER NECK AND/OR OF URETHRA
PROSTATIC ENLARGEMENT
URETHRAL STRICTURE

6: COLLAGEN DISEASE

DIFFUSE SYSTEMIC SCLEROSIS (SCLERODERMA)
DISSEMINATED (SYSTEMIC) LUPUS ERYTHEMATOSUS
POLYARTERITIS NODOSA

7: METABOLIC RENAL DISEASE

AMYLOIDOSIS
CHRONIC PHENACETIN OVERDOSAGE
GOUT WITH HYPERURICEMIC NEPHROPATHY
PRIMARY HYPERPARATHYROIDISM
MILK–ALKALI SYNDROME
SARCOIDOSIS



8: CONGENITAL ANOMALIES OF KIDNEYS

HYPOPLASTIC KIDNEYS
MEDULLARY CYSTIC DISEASE
POLYCYSTIC KIDNEYS

Table 117–1 • CAUSES OF CHRONIC RENAL FAILURE

Diabetic glomerulosclerosis*
Hypertensive nephrosclerosis
Glomerular disease
Glomerulonephritis
Amyloidosis, light chain disease*
SLE, Wegener's granulomatosis*
Tubulointerstitial disease
Reflux nephropathy (chronic pyelonephritis)
Analgesic nephropathy
Obstructive nephropathy (stones, BPH)
Myeloma kidney*
Vascular disease
Scleroderma*
Vasculitis*
Renovascular renal failure (ischemic nephropathy)
Atheroembolic renal disease*
Cystic diseases
Autosomal dominant polycystic kidney disease
Medullary cystic kidney disease

*Systemic disease involving the kidney.
BPH = benign prostatic hypertrophy; SLE = systemic lupus erythematosus.

Table 40. Potential Risk Factors for Susceptibility to and Initiation of Chronic Kidney Disease

Clinical Factors	Sociodemographic Factors
Diabetes	Older age
Hypertension	US ethnic minority status: African American, American Indian, Hispanic, Asian or Pacific Islander
Autoimmune diseases	Exposure to certain chemical and environmental conditions
Systemic infections	Low income/education
Urinary tract infections	
Urinary stones	
Lower urinary tract obstruction	
Neoplasia	
Family history of chronic kidney diseases	
Recovery from acute kidney failure	
Reduction in kidney mass	
Exposure to certain drugs	
Low birth weight	

Κλινική εικόνα

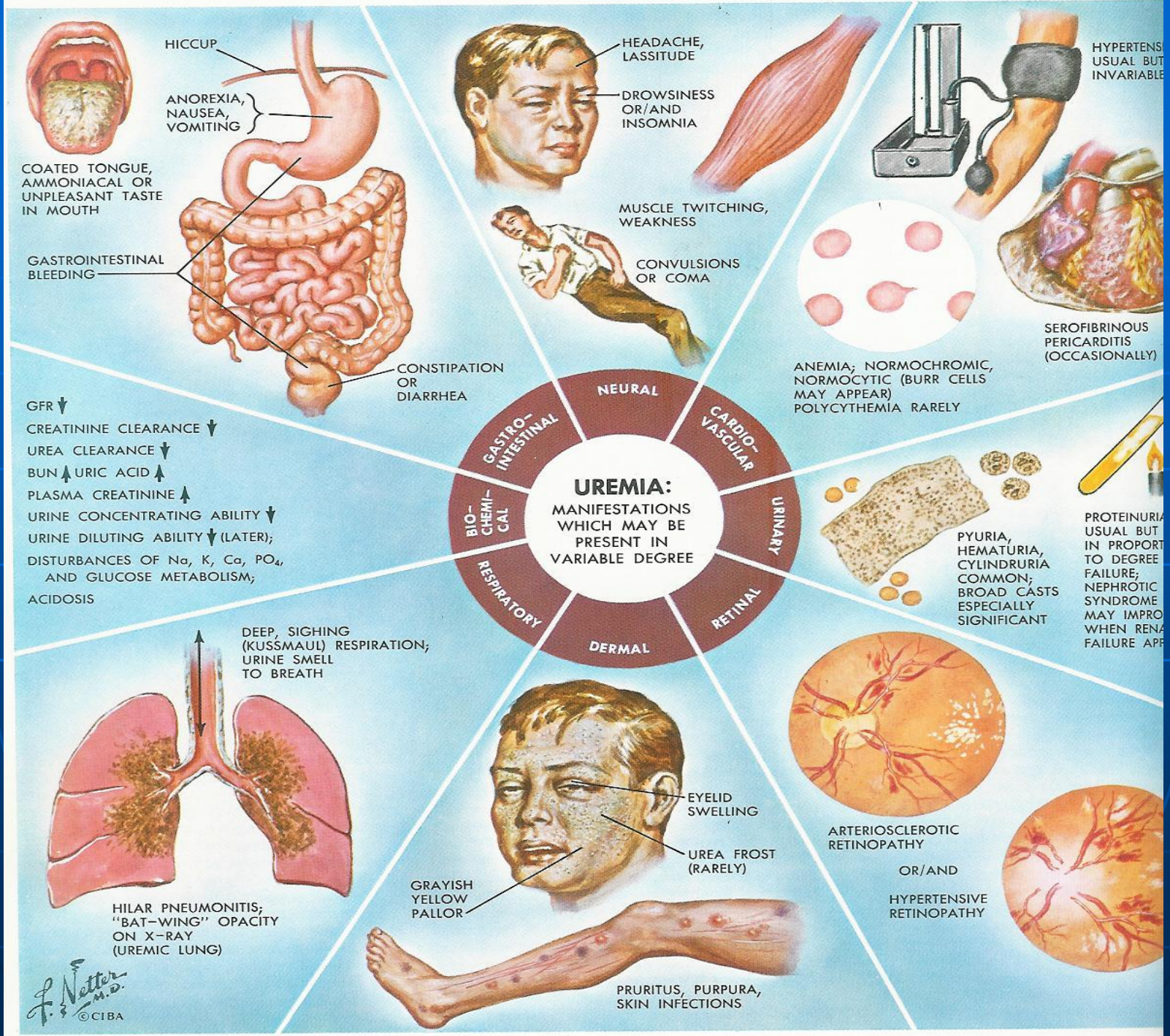
- Μη ειδικά συμπτώματα
- Συνήθως όταν GFR < 15
- Κακουχία, εύκολη κόπωση, αδυναμία
- ΓΕΣ: ανορεξία, ναυτία, έμετος, λόξυγγας, μεταλική γεύση
- Νευρολογικά: ευερεθιστότητα, αϋπνία, διαταραχές συγκέντρωσης, σύνδρ. ανήσυχων ποδιών (restless legs), μυικοί σπασμοί
- Κνησμός
- Παραισθησίες

- Ουραιμική απόπνοια
- Υπέρταση
- Καρδιομεγαλία, πνευμονικό οίδημα, ήχος περικαρδιακής τριβής

Table 117–2 • FEATURES OF CHRONIC RENAL FAILURE

Early
Hypertension
Proteinuria; elevated BUN or SCr
Nephrotic syndrome
Recurrent nephritic syndrome
Gross hematuria
Late (GFR <15 mL/min, BUN >60 mg/dL) ("uremia")
Cardiac failure
Anemia
Serositis
Confusion, coma
Anorexia
Vomiting
Peripheral neuropathy
Hyperkalemia
Metabolic acidosis

BUN = blood urea nitrogen; GFR = glomerular filtration rate; SCr = serum creatinine.


HICCUP
ANOREXIA, NAUSEA, VOMITING
COATED TONGUE, AMMONIACAL OR UNPLEASANT TASTE IN MOUTH
GASTROINTESTINAL BLEEDING
CONSTIPATION OR DIARRHEA
HEADACHE, LASSITUDE
DROWSINESS OR/AND INSOMNIA
MUSCLE TWITCHING, WEAKNESS
CONVULSIONS OR COMA
HYPERTENS
USUAL BUT
INVARIABLE
ANEMIA; NORMOCHROMIC, NORMOCYTIC (BURR CELLS MAY APPEAR)
POLYCYTHEMIA RARELY
SEROFIBRINOUS PERICARDITIS (OCCASIONALLY)
GFR
CREATININE CLEARANCE
UREA CLEARANCE
BUN
URIC ACID
PLASMA CREATININE
URINE CONCENTRATING ABILITY
URINE DILUTING ABILITY (LATER);
DISTURBANCES OF Na, K, Ca, PO4, AND GLUCOSE METABOLISM;
ACIDOSIS
NEURAL
CARDIO-VASCULAR
URINARY
RETINAL
DERMAL
RESPIRATORY
BIO-CHEMI-CAL
GASTRO-INTESTINAL
UREMIA:
MANIFESTATIONS WHICH MAY BE PRESENT IN VARIABLE DEGREE
PYURIA, HEMATURIA, CYLINDRURIA COMMON; BROAD CASTS ESPECIALLY SIGNIFICANT
PROTEINURIA
USUAL BUT
IN PROPORT
TO DEGREE
FAILURE;
NEPHROTIC
SYNDROME
MAY IMPRO
WHEN RENA
FAILURE APP
DEEP, SIGHING (KUSSMAUL) RESPIRATION; URINE SMELL TO BREATH
HILAR PNEUMONITIS; "BAT-WING" OPACITY ON X-RAY (UREMIC LUNG)
EYELID SWELLING
UREA FROST (RARELY)
GRAYISH YELLOW PALLOR
PRURITUS, PURPURA, SKIN INFECTIONS
ARTERIOSCLEROTIC RETINOPATHY
OR/AND
HYPERTENSIVE RETINOPATHY
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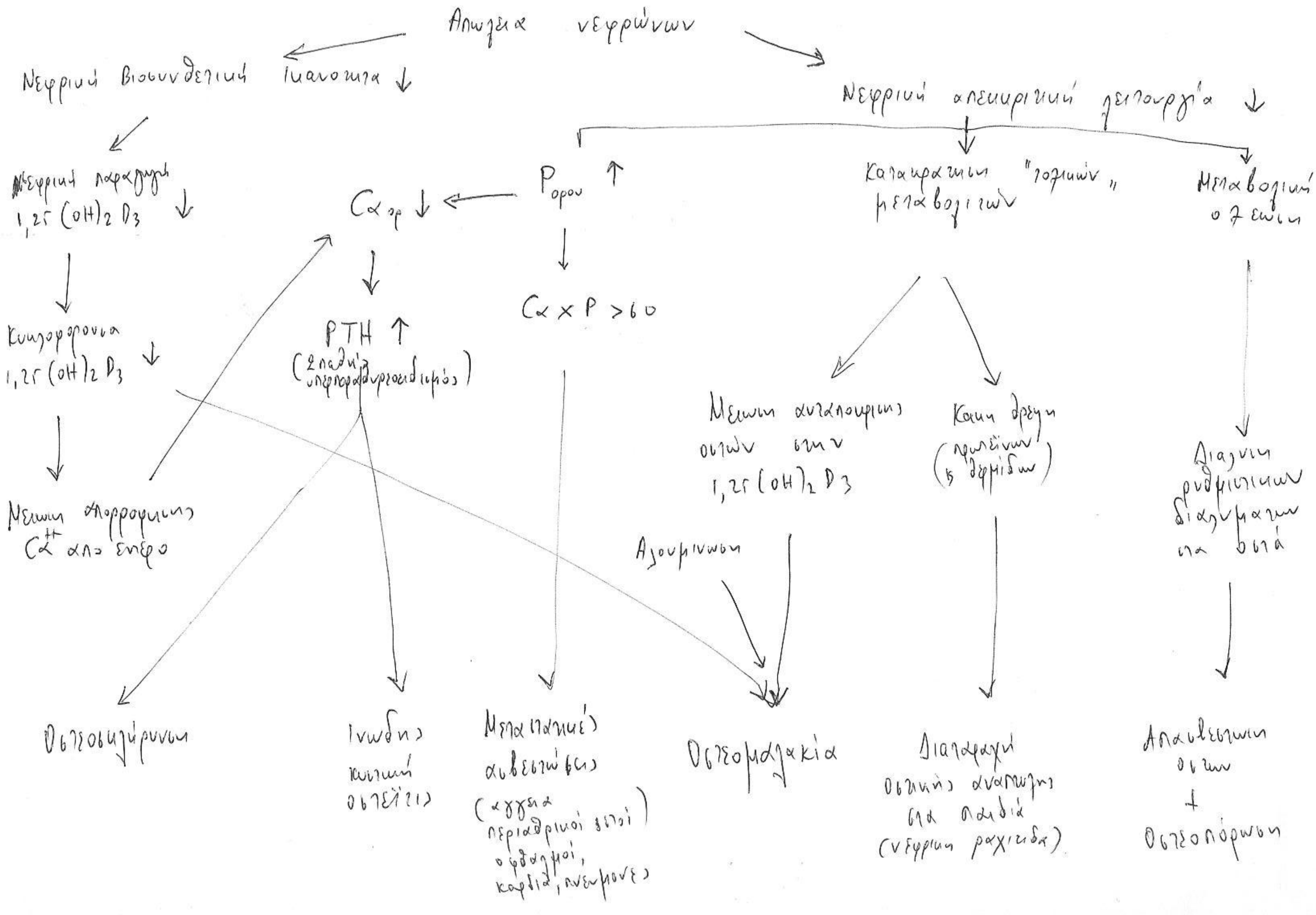
Correlation between urinary patterns and renal disease

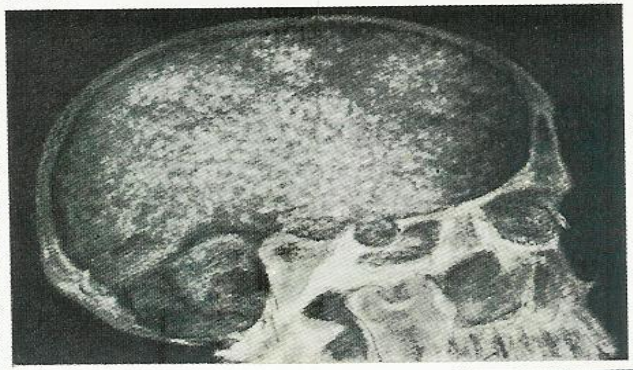
Urinary pattern	Renal disease
Hematuria with red cell casts, dysmorphic red cells, heavy proteinuria, or lipiduria	Virtually diagnostic of glomerular disease or vasculitis
Multiple granular and epithelial cell casts with free epithelial cells	Strongly suggestive of acute tubular necrosis in a patient with acute renal failure
Pyuria with white cell and granular or waxy casts and no or mild proteinuria	Suggestive of tubular or interstitial disease or urinary tract obstruction
Hematuria and pyuria with no or variable casts (excluding red cell casts)	May be observed in acute interstitial nephritis, glomerular disease, vasculitis, obstruction, and renal infarction
Hematuria alone	Varies with the clinical setting
Pyuria alone	Usually infection; sterile pyuria suggests urinary tract tuberculosis or tubulointerstitial disease
Few cells with little or no casts or proteinuria (normal or near-normal)	In acute renal failure, prerenal disease, urinary tract obstruction, hypercalcemia, myeloma kidney, some cases of acute tubular necrosis, or a vascular disease with glomerular ischemia but not infarction (scleroderma, atheroemboli); in chronic renal failure, nephrosclerosis, urinary tract obstruction, and tubulointerstitial disease

ΠΑΘΟΓΕΝΕΙΑ ΟΣΤΙΚΗΣ ΝΟΣΟΥ ΣΕ ΧΝΑ

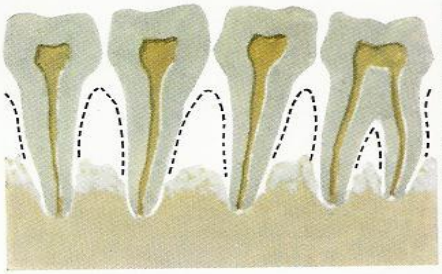


BONE MANIFESTATIONS OF SECONDARY HYPERPARATHYROIDISM IN CHRONIC RENAL DISEASE

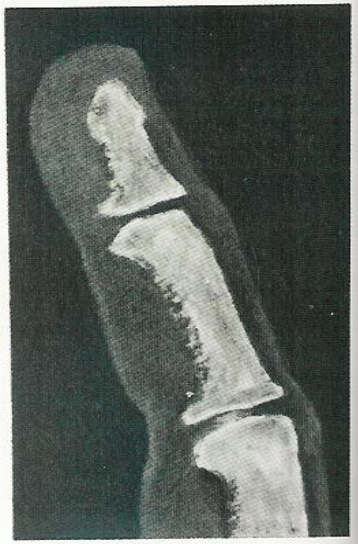
"BANDED SCLEROSIS" OF SPINE, SCLEROSIS OF UPPER AND LOWER MARGINS OF VERTEBRAE WITH RAREFACTION BETWEEN. NOTE COMPRESSION FRACTURE



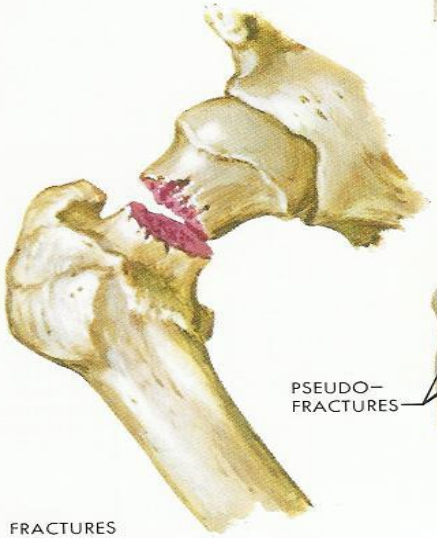
SPOTTY DEMINERAL–IZATION OF SKULL



LOSS OF LAMINA DURA OF TEETH (BROKEN LINES INDICATE NORMAL CONTOURS)

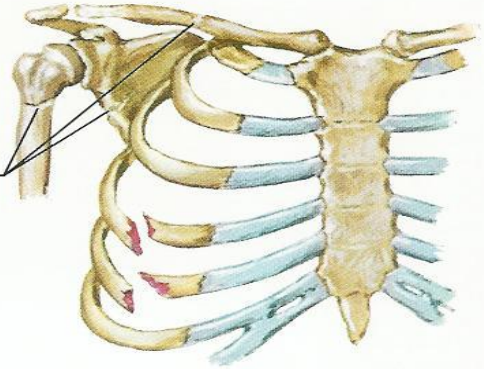


SUBPERIOSTEAL RESORPTION OF PHALANGES (CHIEFLY ON PALMAR ASPECT OF MIDDLE PHALANX)

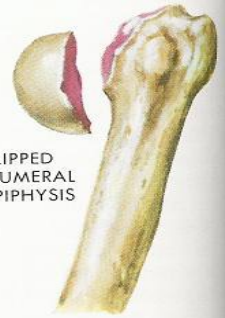


FRACTURES OF LONG BONES

PSEUDO–FRACTURES



RESORPTION OF LATERAL END OF CLAVICLE; RIB FRACTURES



SLIPPED HUMERAL EPIPHYSIS

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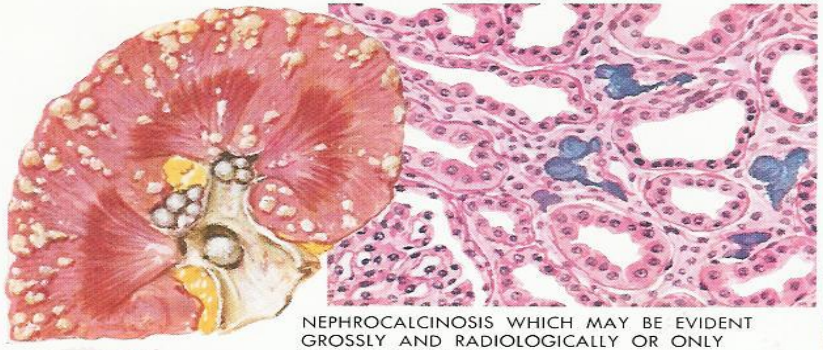
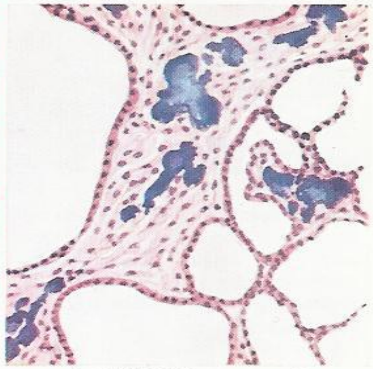
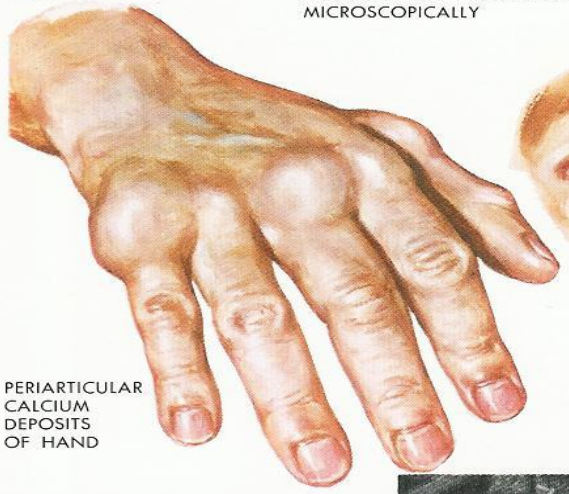
VASCULAR AND SOFT TISSUE CALCIFICATION IN SECONDARY HYPERPARATHYROIDISM OF CHRONIC RENAL DISEASE



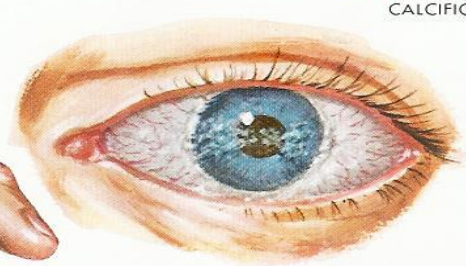
NEPHROCALCINOSIS WHICH MAY BE EVIDENT GROSSLY AND RADIOLOGICALLY OR ONLY MICROSCOPICALLY



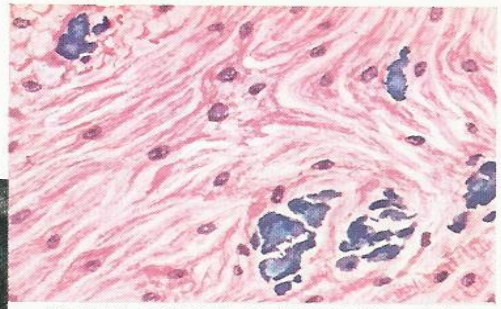
CALCIFICATION IN LUNG



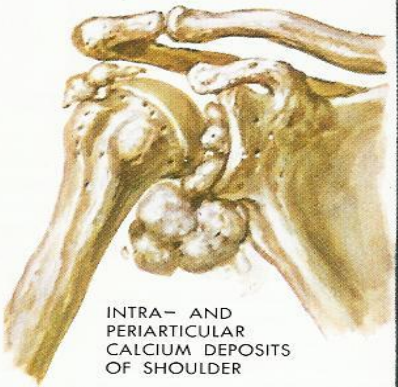
PERIARTICULAR CALCIUM DEPOSITS OF HAND



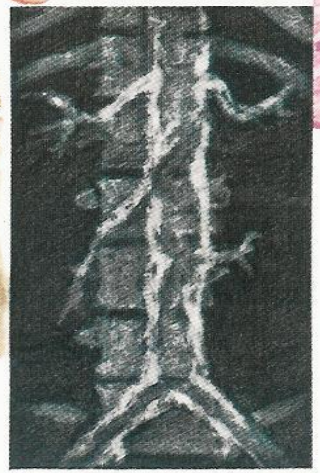
BAND KERATOPATHY, LIMBUS KERATOPATHY, AND/OR CALCIUM DEPOSITS IN CONJUNCTIVA WITH CONJUNCTIVITIS



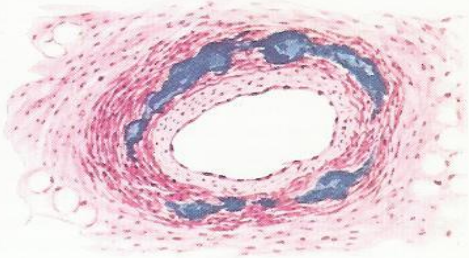
CALCIUM DEPOSITS IN CONDUCTION SYSTEM OF HEART WHICH MAY CAUSE SERIOUS OR FATAL ARRHYTHMIAS



INTRA– AND PERIARTICULAR CALCIUM DEPOSITS OF SHOULDER



CALCIFICATION OF AORTA AND/OR OTHER LARGE VESSELS



MEDIAL CALCIFICATION OF CORONARY AND/OR OTHER SMALL ARTERIES

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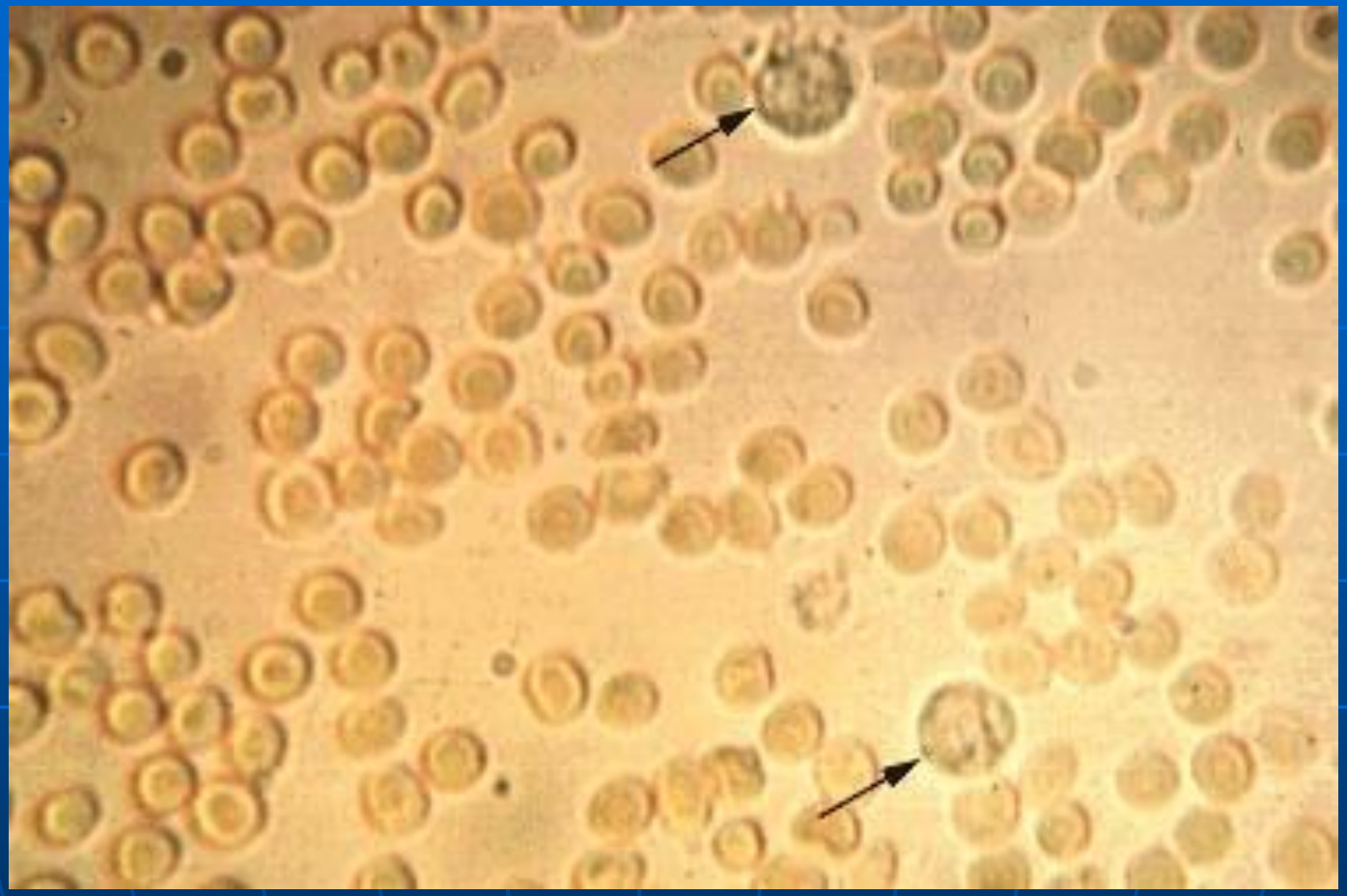
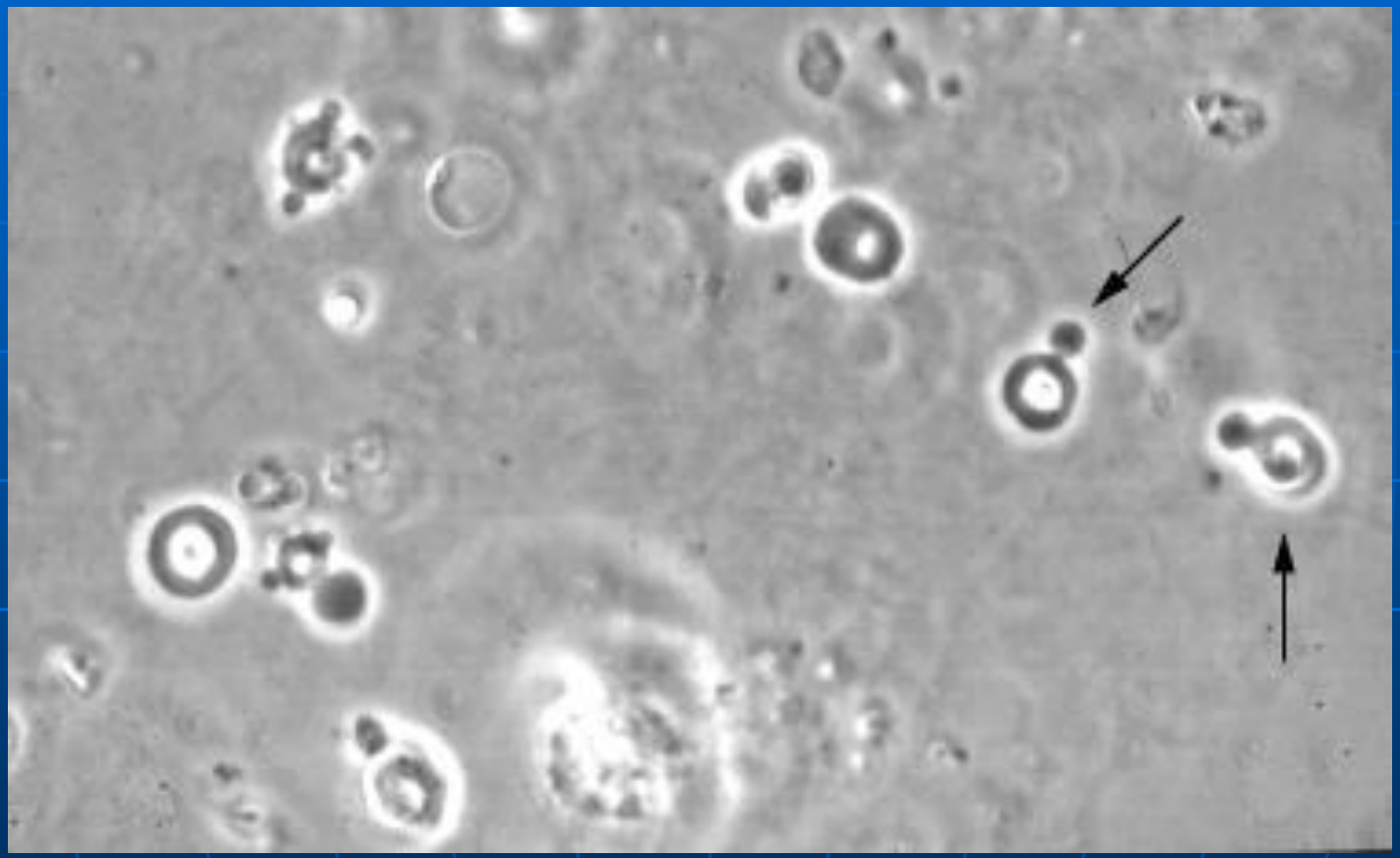
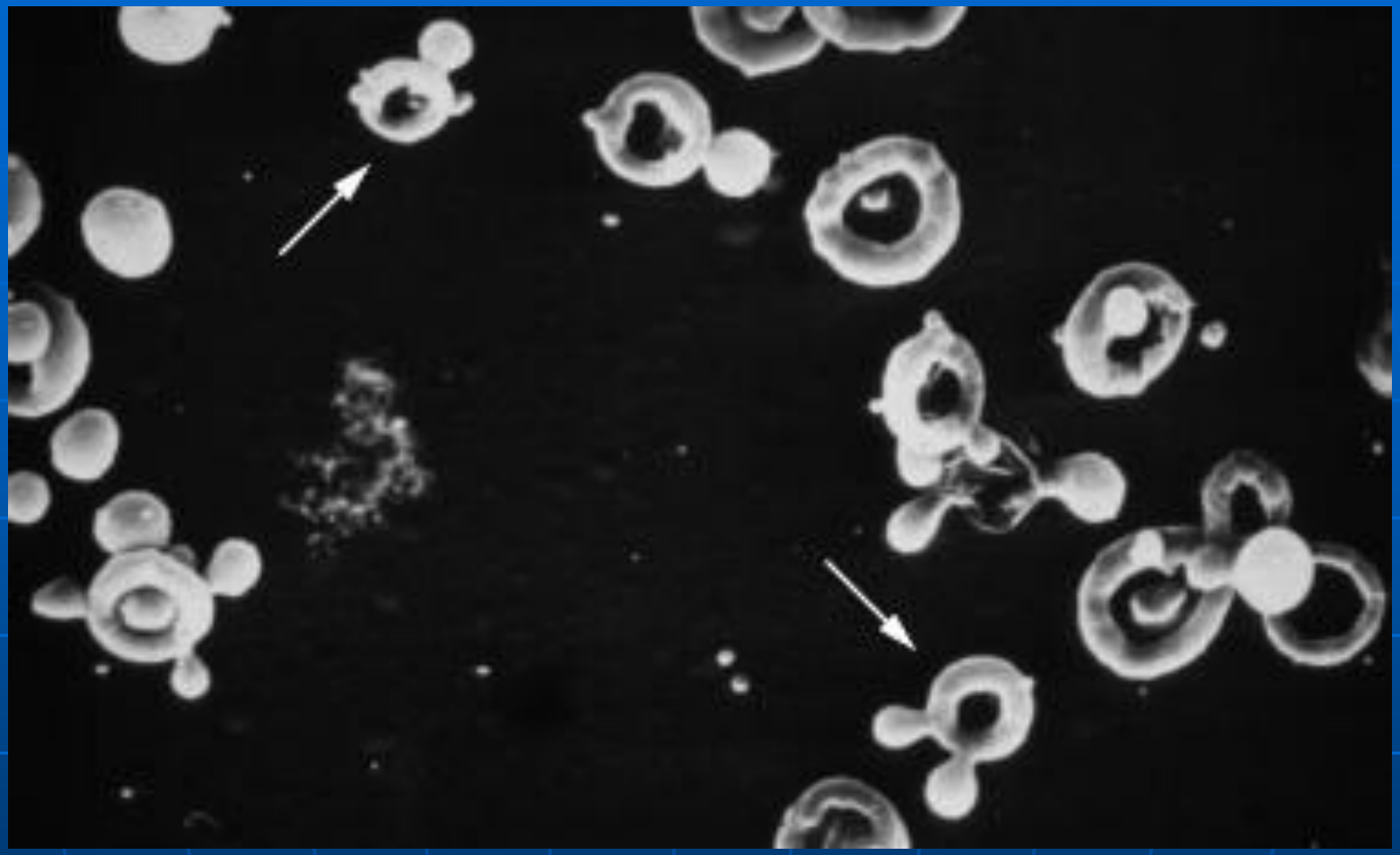
soft tissues such as the kidneys, lungs, joints, bursae, calcium deposition will not be a feature in this type



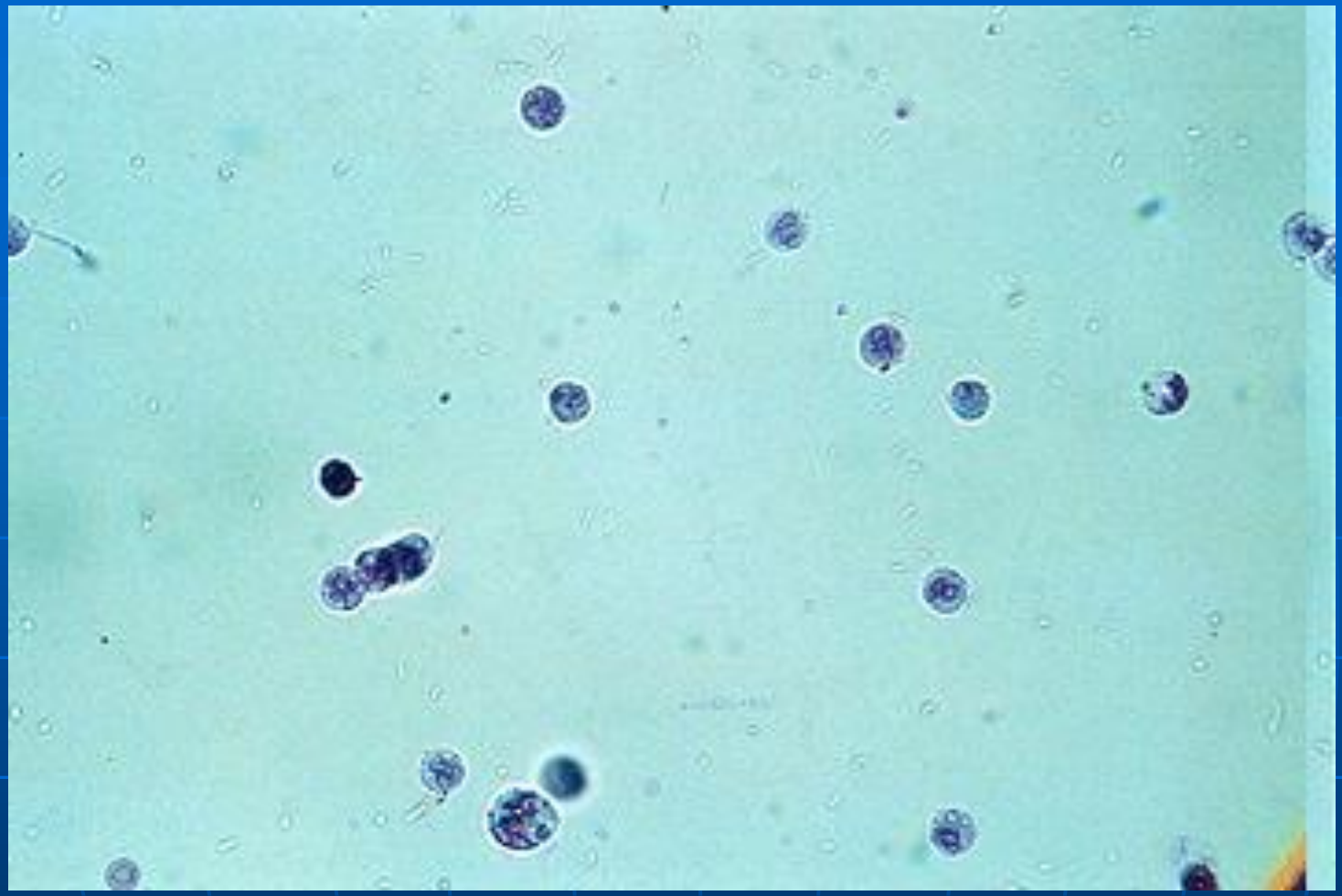
Urine sediment showing many red cells and an occasional larger white cell with a granular cytoplasm (arrows). The red cells have a uniform size and shape, suggesting that they are of nonglomerular origin. Courtesy of Harvard Medical School.



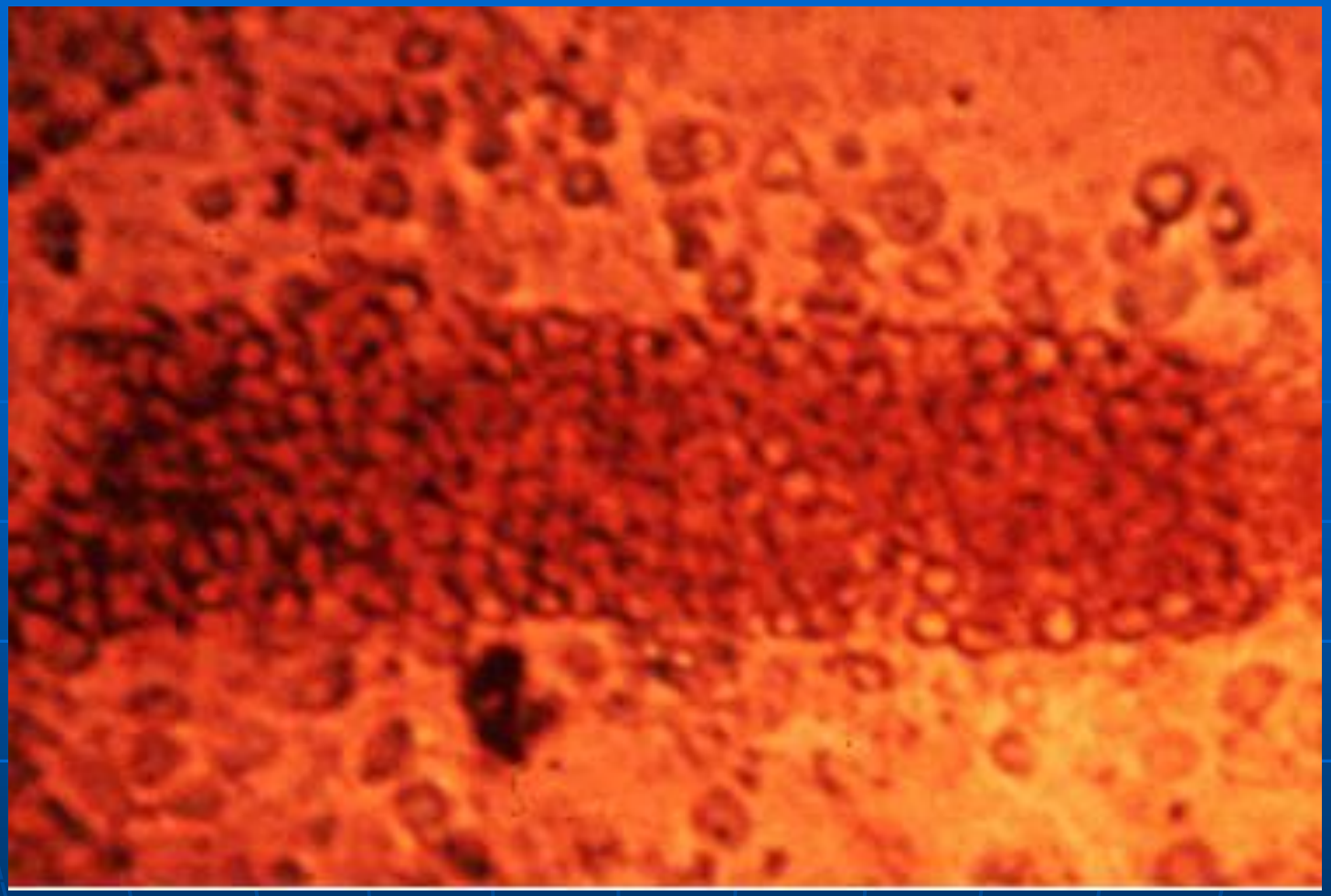
Phase contrast microscopy showing dysmorphic red cells in a patient with glomerular bleeding. Acanthocytes can be recognized as ring forms with vesicle-shaped protrusions (arrows). Courtesy of Hans Köhler, MD.



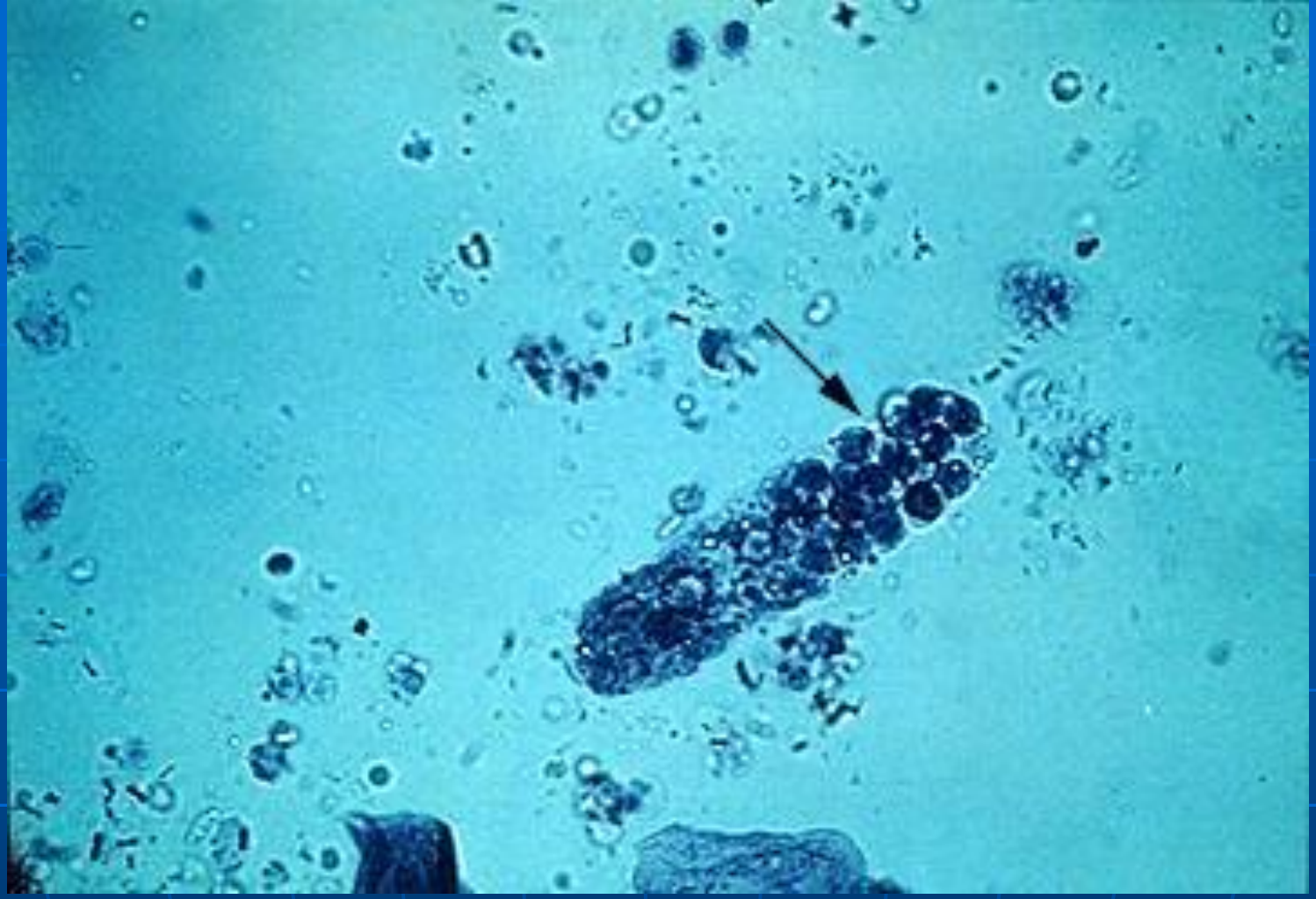
Scanning microscopy showing dysmorphic red cells in a patient with glomerular bleeding. Acanthocytes can be recognized as ring forms with vesicle-shaped protrusions (arrows). Courtesy of Hans Köhler, MD.



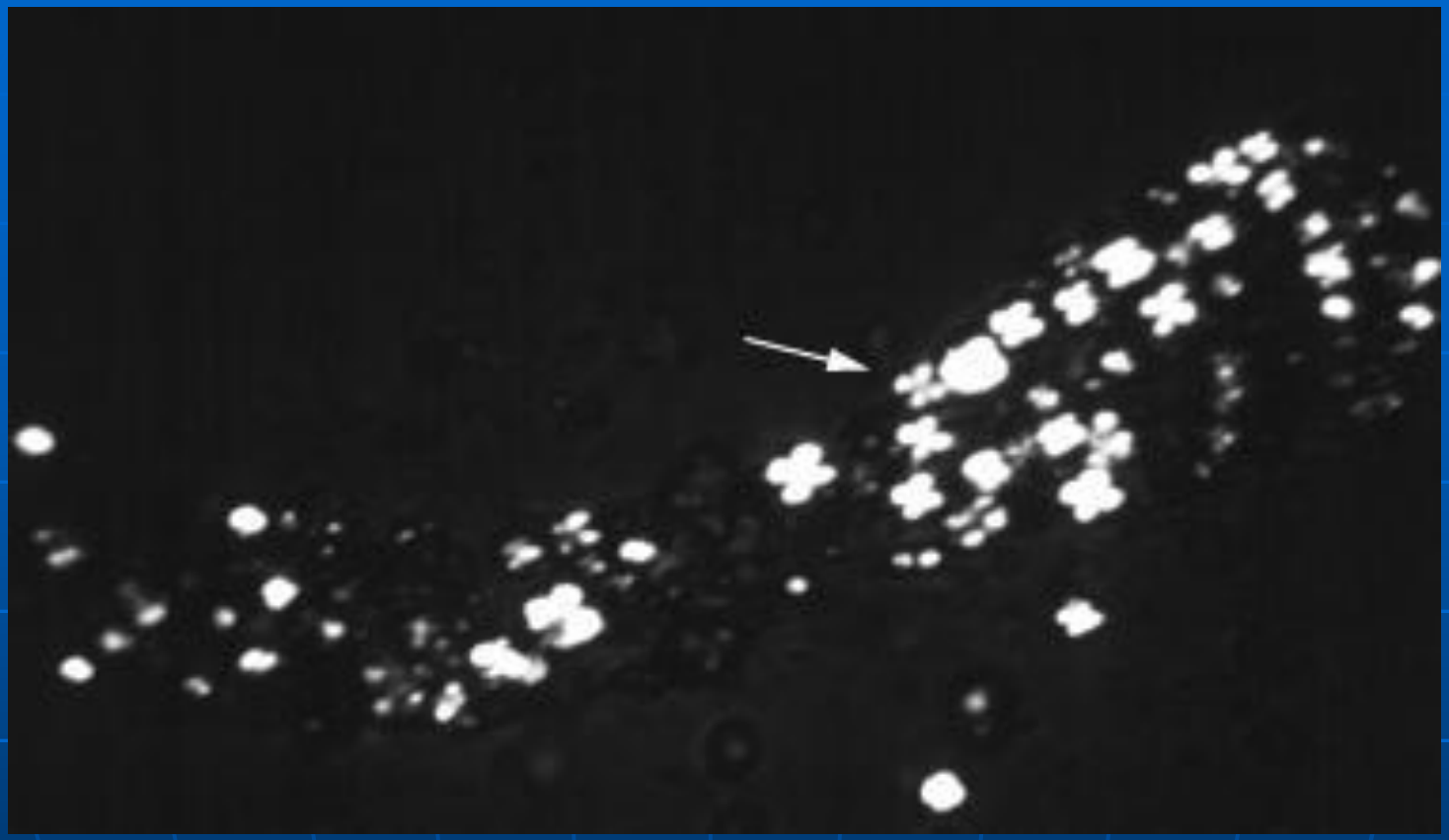
White blood cells in the urine sediment with nuclei and granular cytoplasm. Courtesy of Frances Andrus, BA, Victoria Hospital, London, Ontario.



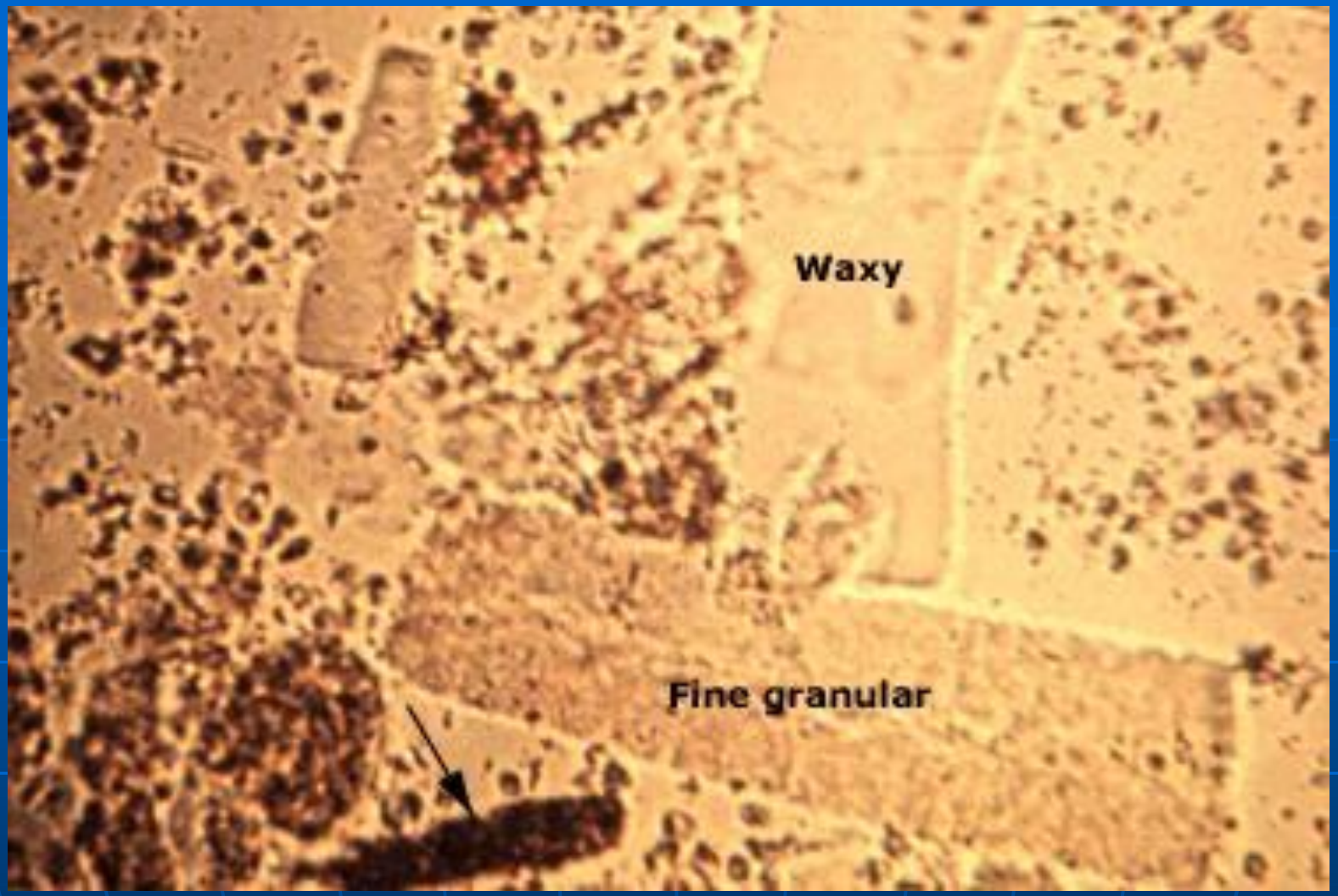
Urine sediment showing free red cells and a red cell cast that is tightly packed with red cells. It is more common for red cell casts to have fewer red cells trapped within a hyaline or granular cast. Red cell casts are virtually diagnostic of glomerulonephritis or vasculitis. Courtesy of Harvard Medical School.



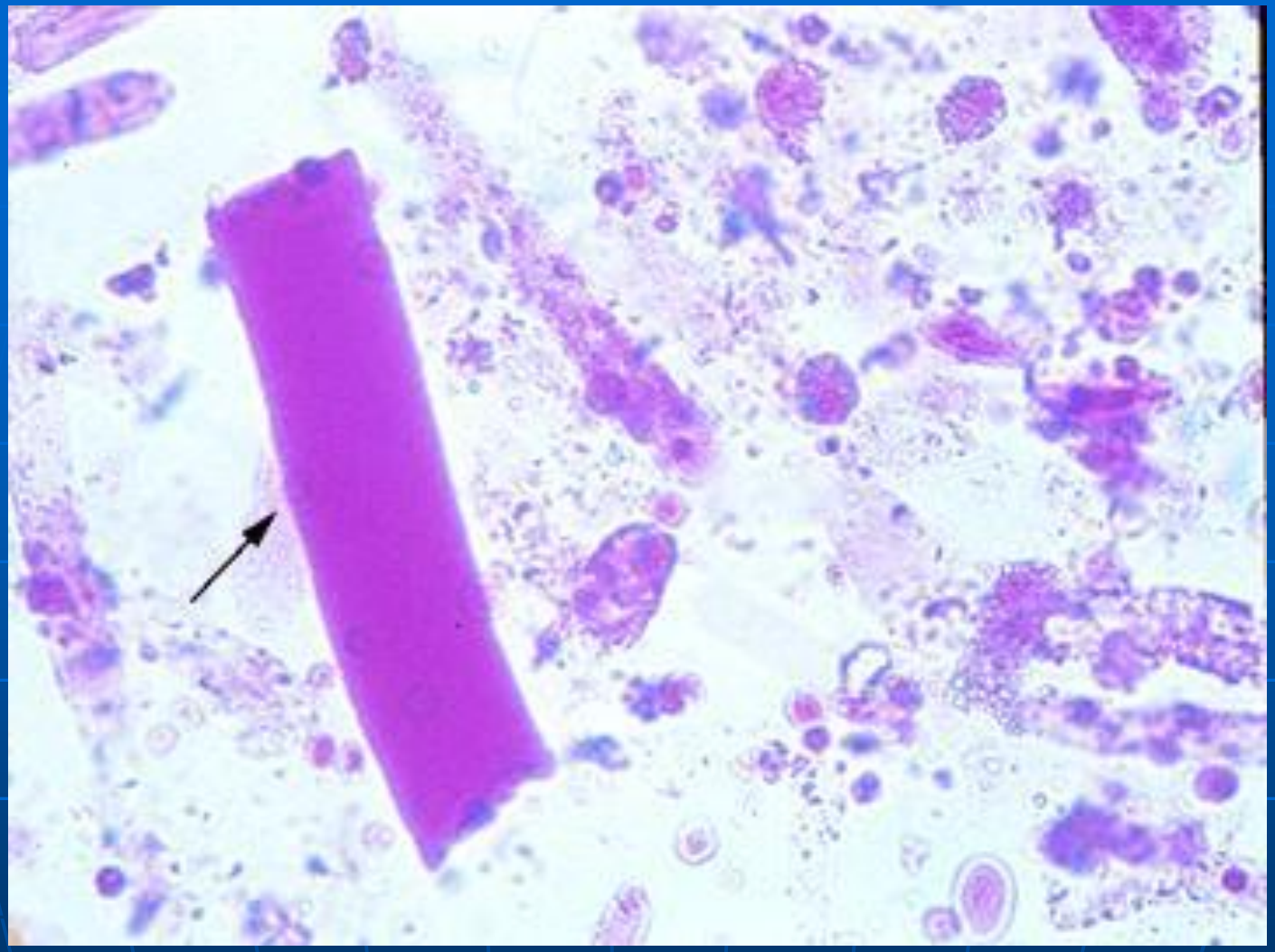
White cell cast in which blue stained white cells (arrow) are contained within a granular cast. Courtesy of Frances Andrus, BA, Victoria Hospital, London, Ontario.



Urine sediment showing fatty cast under polarized light. The fat droplets have a characteristic "Maltese cross" appearance (arrow). Courtesy of Harvard Medical School.



Urine sediment showing waxy and fine and coarse (arrow) granular casts. The broader casts are thought to form when there is stasis (due to advanced renal failure) in the wider collecting tubules into which many nephrons drain. Courtesy of Harvard Medical School.



Urine sediment showing a waxy cast (arrow) and many small fine granular casts. Note the cast outline and its amorphous appearance. The high optical density, smooth surface, and blunt ends of waxy casts, which appear to represent degenerated cellular or granular casts, allows them to be distinguished from hyaline casts. The latter are composed solely of precipitated Tamm-Horsfall mucoprotein.

Εργαστηριακός έλεγχος

- Ουρία, κρεατινίνη
- Προηγούμενες εξετάσεις
- Αποκλεισμός αναστρέψιμων παραγόντων
  - Λοίμωξη, απόφραξη, υποογκαιμία, νεφροτοξίνες, υπέρταση, καρδιακή ανεπάρκεια

Απεικονιστικός έλεγχος

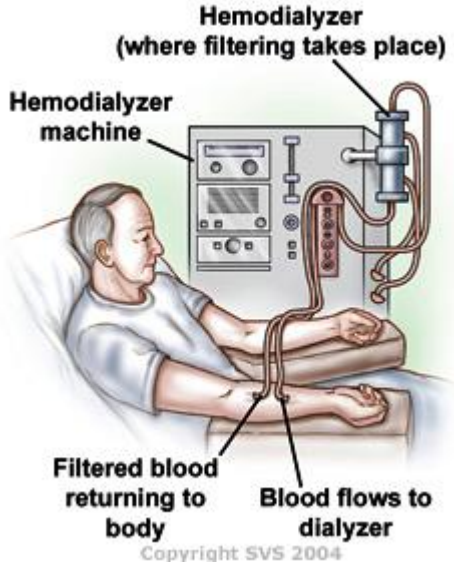
- Υπερηχογράφημα νεφρών
  - Μικροί ρικνοί νεφροί (<10 cm)
  - Φυσιολογικό ή αυξημένο μέγεθος σε
    - Πολυκυστική νόσο
    - Διαβητική νεφροπάθεια
    - Αμυλοείδωση
    - Αποφρακτική νεφροπάθεια

Επιπλοκές

- Υπερκαλιαιμία
- Μεταβολική οξέωση
- Καρδιαγγειακές επιπλοκές (κίνδυνος 10-200 x)
  - Αρτηριακή υπέρταση
  - Περικαρδίτιδα
  - Συμφορητική καρδιακή ανεπάρκεια
- Αιματολογικές επιπλοκές
  - Αναιμία
  - Διαταραχή πηκτικότητας
- Νευρολογικές επιπλοκές
  - Ουραιμική εγκεφαλοπάθεια
  - Περιφερική νευροπάθεια
- Διαταραχές μεταβολισμού
  - Νεφρική οστεοδυστροφία
  - Ινώδης κυστική οστεΐτιδα
  - Δευτεροπαθής υπερπαραθυρεοειδισμός

Αντιμετώπιση

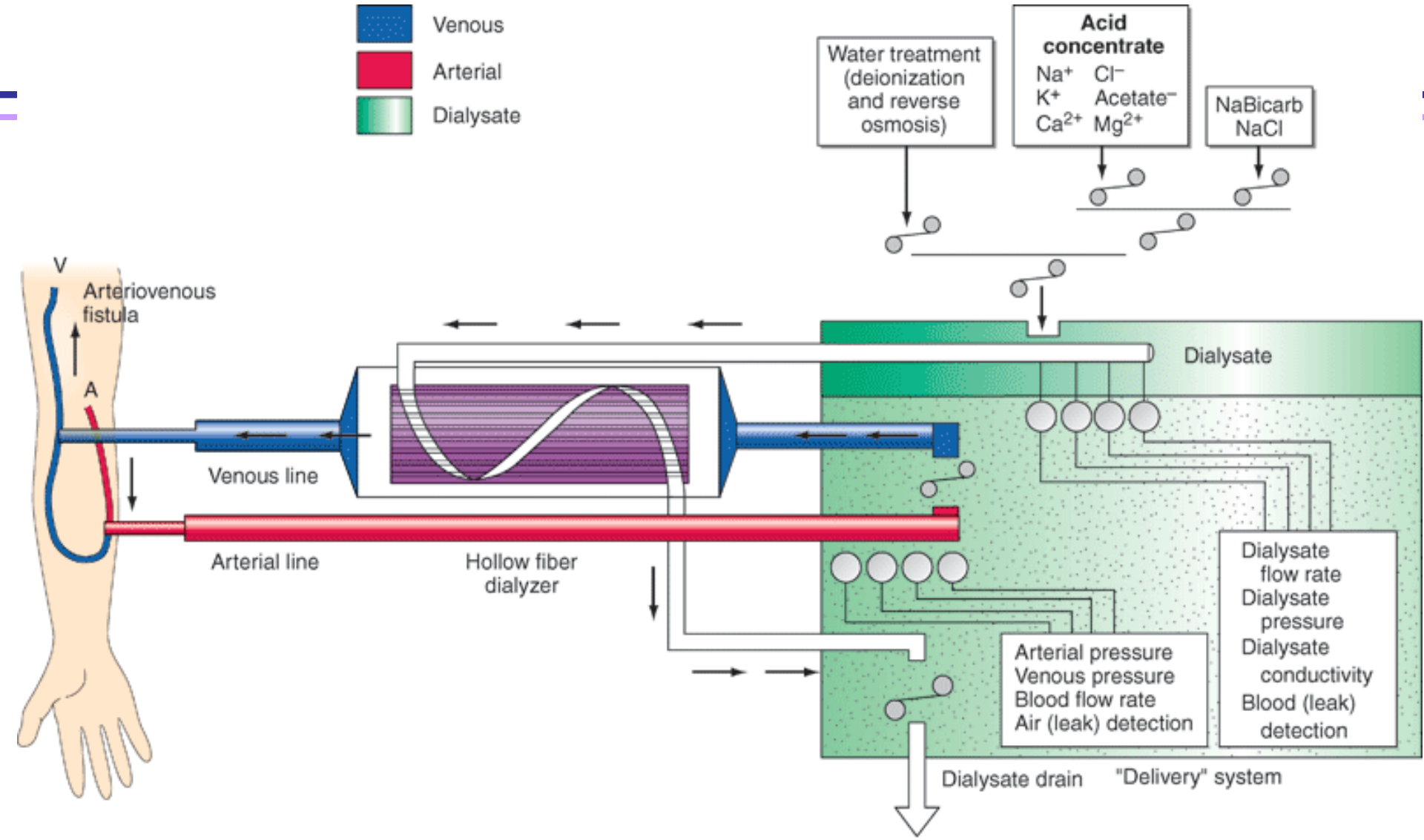
- Δίαιτα
  - πρωτεϊνών
  - ύδατος και αλατιού
  - Καλίου, φωσφόρου, μαγνησίου
- Φαρμακευτική αγωγή
  - Διουρητικά (της αγκύλης)
  - α-ΜΕΑ
  - Φωσφοροδεσμευτικά (άλατα Ca)
  - Ερυθροποιητίνη
- Αιμοκάθαρση – περιτοναϊκή κάθαρση
- Μεταμόσχευση



Ενδείξεις αιμοκάθαρσης

- Ουραιμικά συμπτώματα (περικαρδίτιδα, εγκεφαλοπάθεια, διαταραχή πηκτικότητας)
- Σοβαρή μεταβολική οξέωση (pH < 7.20)
- Σοβαρή εμμένουσα υπερκαλιαιμία
- Εμμένουσα υπερφόρτωση υγρών
- Νευρολογική συμπτωματολογία (επιληπτικές κρίσεις ή νευροπάθεια)
- GFR < 10-15 ml/min (Cr 6-8 mg/dl)

ΑΙΜΟΚΑΘΑΡΣΗ



Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 18th Edition: www.accessmedicine.com

Πρόγνωση

- Σημαντική αύξηση θνητότητας
- Αναμενόμενη επιβίωση στην ηλικία 55-64 ετών
  - Αιμοκαθαιρόμενοι:5 έτη
  - Υγιείς: 22 έτη
- Αίτια θανάτου
  - Καρδιακή νόσος 48%
  - Λοίμωξη 15%
  - Αγγειακή εγκεφαλική νόσος 6%
  - Κακοήθεια 4%

Καρδιαγγειακή νόσος σε ασθενείς με χρόνια νεφρική ανεπάρκεια