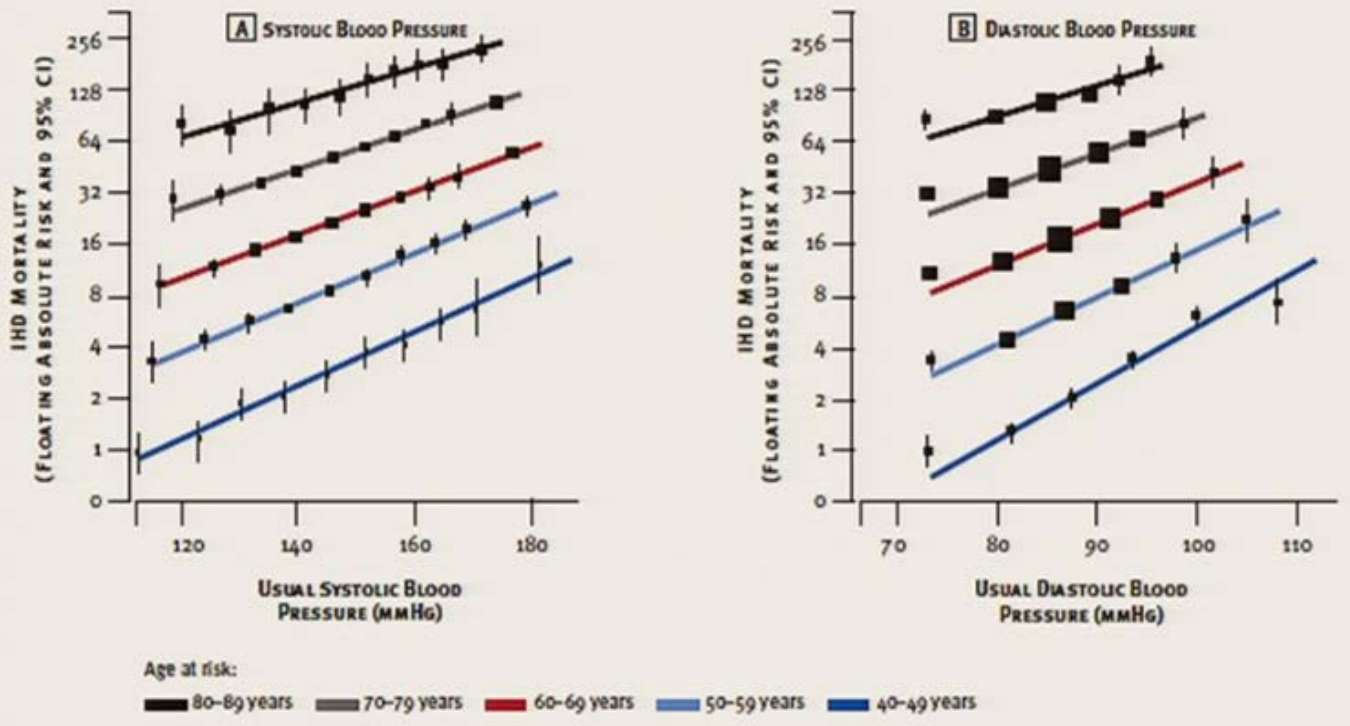
Renal Board Review

Brenda Shinar, MD

Question 1.

- Answer: A; Add chlorthalidone

Relationship Between Hypertension and Cardiovascular Mortality



Lewington S, et al. *Lancet* 2002

Intensive Review of Internal Medicine

Cleveland Clinic

Over 115/75 mm Hg, for every increase in systolic BP by 20 mm Hg or every increase in diastolic blood pressure by 10 mm Hg, the ***risk of cardiovascular death doubles!***

Treatment of Hypertension

- Make the diagnosis
  - Measure correctly
  - Ambulatory monitoring/Home monitoring
  - End organ damage/CV risks
- Look for modifiable risk factors
  - Obesity, sedentary lifestyle, alcohol use, drugs, dietary factors
- Think about secondary causes
  - RAS, OSA, endocrine, coarctation, renal disease
- Pick your medication(s)
  - First line for AA, Caucasian, underlying conditions
- Treat to goal
  - Add synergistic agents, avoid side effects

<u>Resistant hypertension:</u>

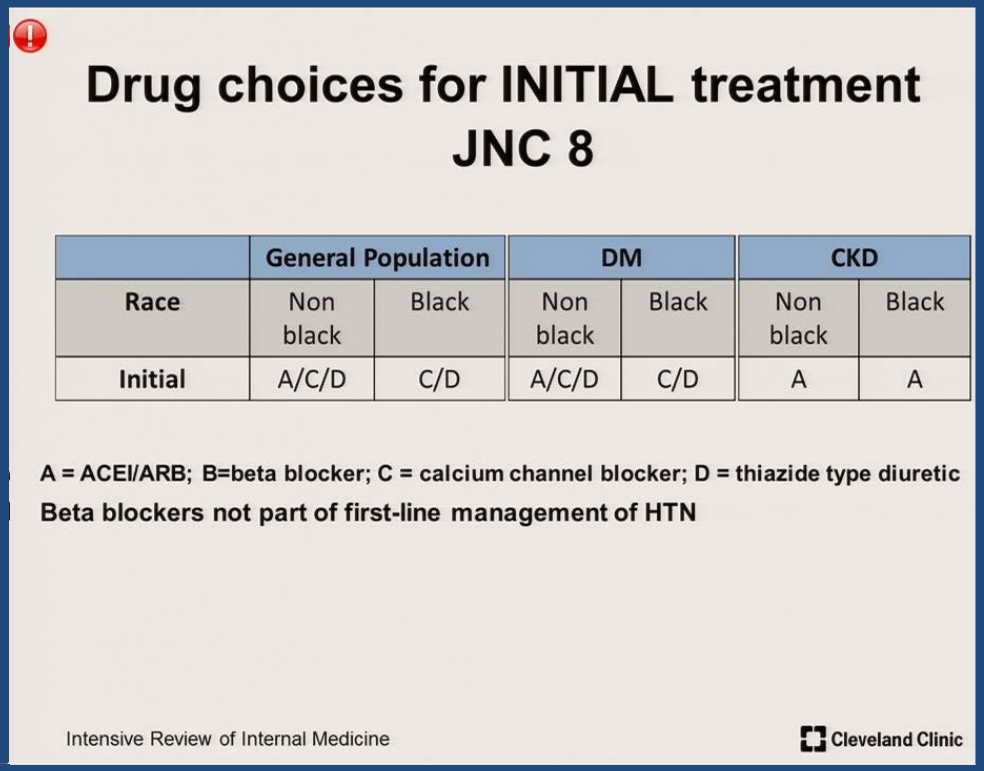
Blood pressure that is uncontrolled on 3 medications from different classes, at optimal dose, *one of which is a diuretic OR* controlled on 4 or more medications

- On lisinopril, nifedipine, atenolol
- ADD thiazide (GFR > 30) or loop diuretic (GFR <30)

Question 2.

- Answer: D; Lisinopril

Treating Stage 1 hypertension in African Americans



Drug choices for INITIAL treatment JNC 8

	General Population		DM		CKD	
Race	Non black	Black	Non black	Black	Non black	Black
Initial	A/C/D	C/D	A/C/D	C/D	A	A

A = ACEI/ARB; B=beta blocker; C = calcium channel blocker; D = thiazide type diuretic

Beta blockers not part of first-line management of HTN

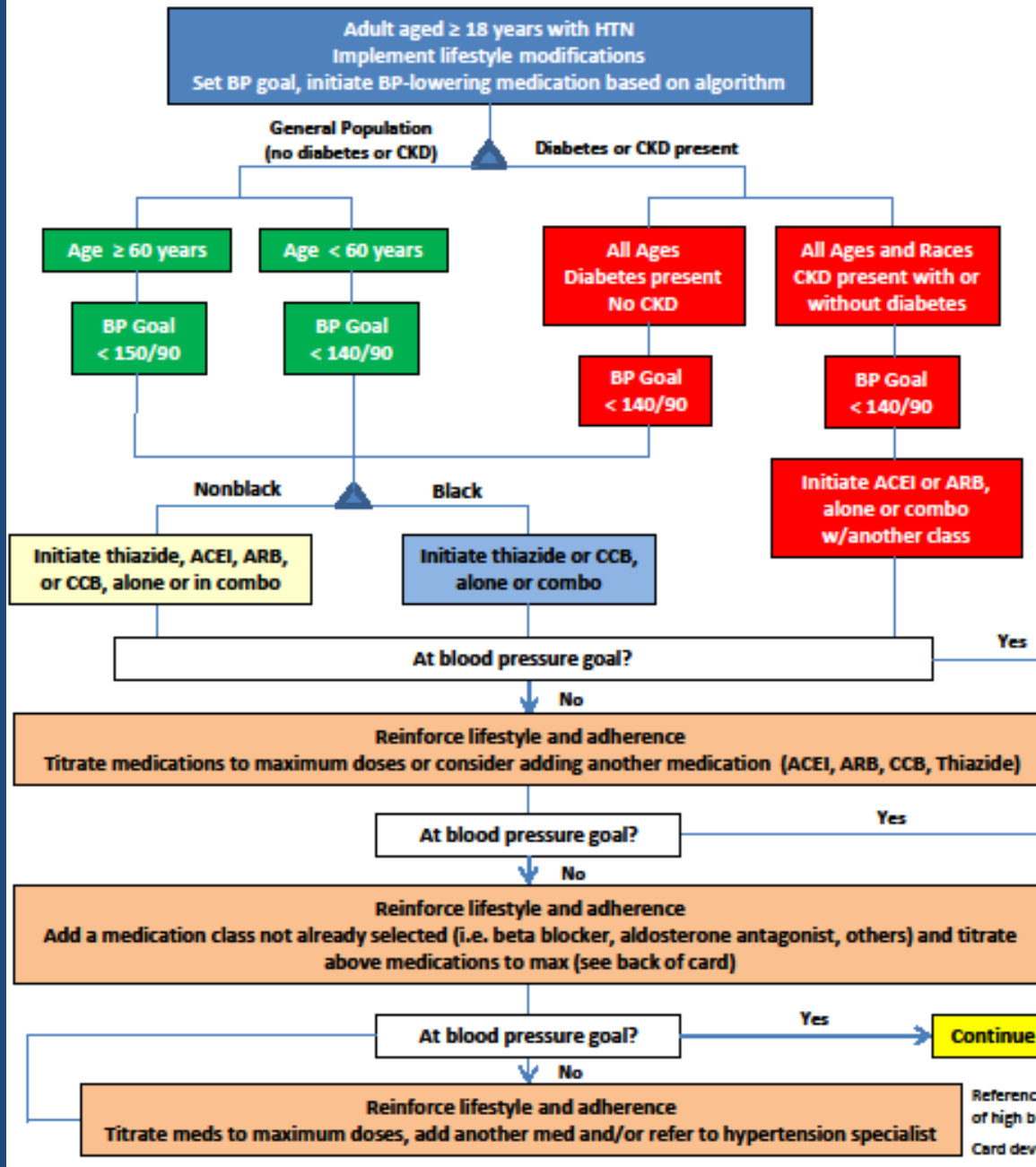
Intensive Review of Internal Medicine

Cleveland Clinic

Beta blockers are never first line antihypertensive drugs!

- **Thiazide diuretics and calcium channel blockers (amlodipine and diltiazem) are first line treatment for blood pressure management *even in the setting of diabetes for black patients!***
- ***Black patients with CKD can start with ACEI as first line!***

JNC 8 Hypertension Guideline Algorithm



Initial Drugs of Choice for Hypertension

- ACE inhibitor (ACEI)
- Angiotensin receptor blocker (ARB)
- Thiazide diuretic
- Calcium channel blocker (CCB)

Strategy	Description
A	Start one drug, titrate to maximum dose, and then add a second drug.
B	Start one drug, then add a second drug before achieving max dose of first
C	Begin 2 drugs at same time, as separate pills or combination pill. **Initial combination therapy is recommended if BP is greater than 20/10mm Hg above goal**

Lifestyle changes:

- Smoking Cessation
- Control blood glucose and lipids
- Diet
  - ✓Eat healthy (i.e., DASH diet)
  - ✓Moderate alcohol consumption
  - ✓Reduce sodium intake to no more than 2,400 mg/day
- Physical activity
  - ✓Moderate-to-vigorous activity 3-4 days a week averaging 40 min per session.

Reference: James PA, Ortiz E, et al. 2014 evidence-based guideline for the management of high blood pressure in adults: (JNC8). JAMA. 2014 Feb 5;311(5):507-20

Card developed by Cole Glenn, Pharm.D. & James L Taylor, Pharm.D.

Question 3.

- Answer: D; Continue clinical observation

Manage HTN in a patient who is over age 60.

Classification

Blood Pressure Classification	SBP mmHg	DBP mmHg
Normal	<120	and <80
Prehypertension	120–139	or 80–89
Stage 1 Hypertension	140–159	or 90–99
Stage 2 Hypertension	≥160	or ≥100

Goals of Therapy
Recommendations from recent guidelines

	JNC 8 panel	ASH/ISH	JNC 7
General	<140/90	<140/90	<140/90
Elderly	<150/90 *(≥ 60 y)*	<150/90 *(≥80 y)*	
DM	<140/90	<140/90	<130/80
CKD	<140/90	<140/90	<130/80

Question 4.

- Answer: B: Plasma aldosterone-plasma renin activity ratio

Understand the differential diagnosis of resistant hypertension

Definition of resistant hypertension:

The failure to reach blood pressure goal despite the concurrent use of 3 antihypertensive agents of different classes at maximal tolerated doses, with one of the agents being a diuretic. (JNC 7)

OR

Patients whose blood pressure is controlled on 4 or more agents (AHA)

Approach to Resistant HTN

- Establish diagnosis
- Is there white coat hypertension?
- Is the patient adherent to medical program?
- Is the patient taking interfering substances?
- Is there an identifiable (secondary) cause?
- Optimize and intensify pharmacologic therapy
- Refer to hypertension specialist
- Experimental therapies? (currently none FDA approved…)

Indications for Further Evaluation for Secondary Causes

- Abnormal labs - hypercalcemia, hypokalemia with kaliuresis…
- Abrupt onset
- Age < 30 or > 55 years
- Resistant hypertension
- Systolic-diastolic bruits in epigastrium or lateralizing over a kidney

Primary Hyperaldosteronism:
60%+ of patients have NORMAL K level

- ALDOSTERONE HIGH
- RENIN LOW
- AR RATIO >25 is suggestive BUT NOT diagnostic
- Metabolic alkalosis and hypokalemia MAY OR MAY NOT be present

- 8 AM draw
- OFF spironolactone or eplerenone for 6 weeks
- Possibly off ACEI

- NO CONFIRMATION test needed:
  - Spontaneous hypokalemia
  - Undetectable renin level
  - Aldosterone >30 ng/dL

Question 5.

- Answer: D; Sevelamer

CURRENT CHRONIC KIDNEY DISEASE (CKD) NOMENCLATURE USED BY KDIGO

CKD is defined as abnormalities of kidney structure or function, present for > 3 months, with implications for health. CKD is classified based on Cause, GFR category (G1-G5), and Albuminuria category (A1-A3), abbreviated as CGA.

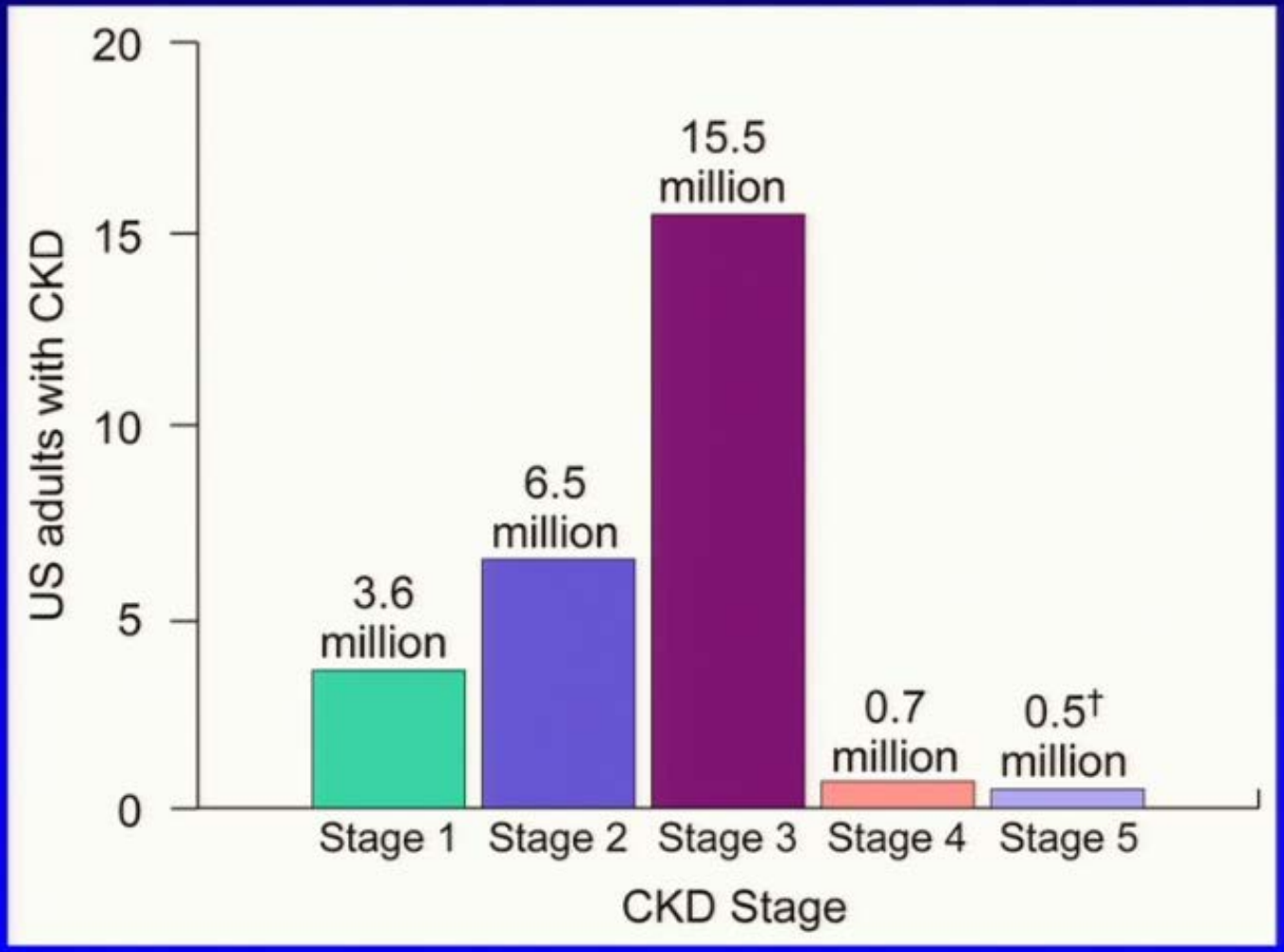
Prognosis of CKD by GFR and albuminuria category

Prognosis of CKD by GFR and Albuminuria Categories: KDIGO 2012

GFR categories (ml/min/ 1.73 m^2) Description and range				Persistent albuminuria categories Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30-300 mg/g 3-30 mg/mmol	>300 mg/g >30 mg/mmol
	G1	Normal or high	≥90			
	G2	Mildly decreased	60-89			
	G3a	Mildly to moderately decreased	45-59			
	G3b	Moderately to severely decreased	30-44			
	G4	Severely decreased	15-29			
	G5	Kidney failure	<15			

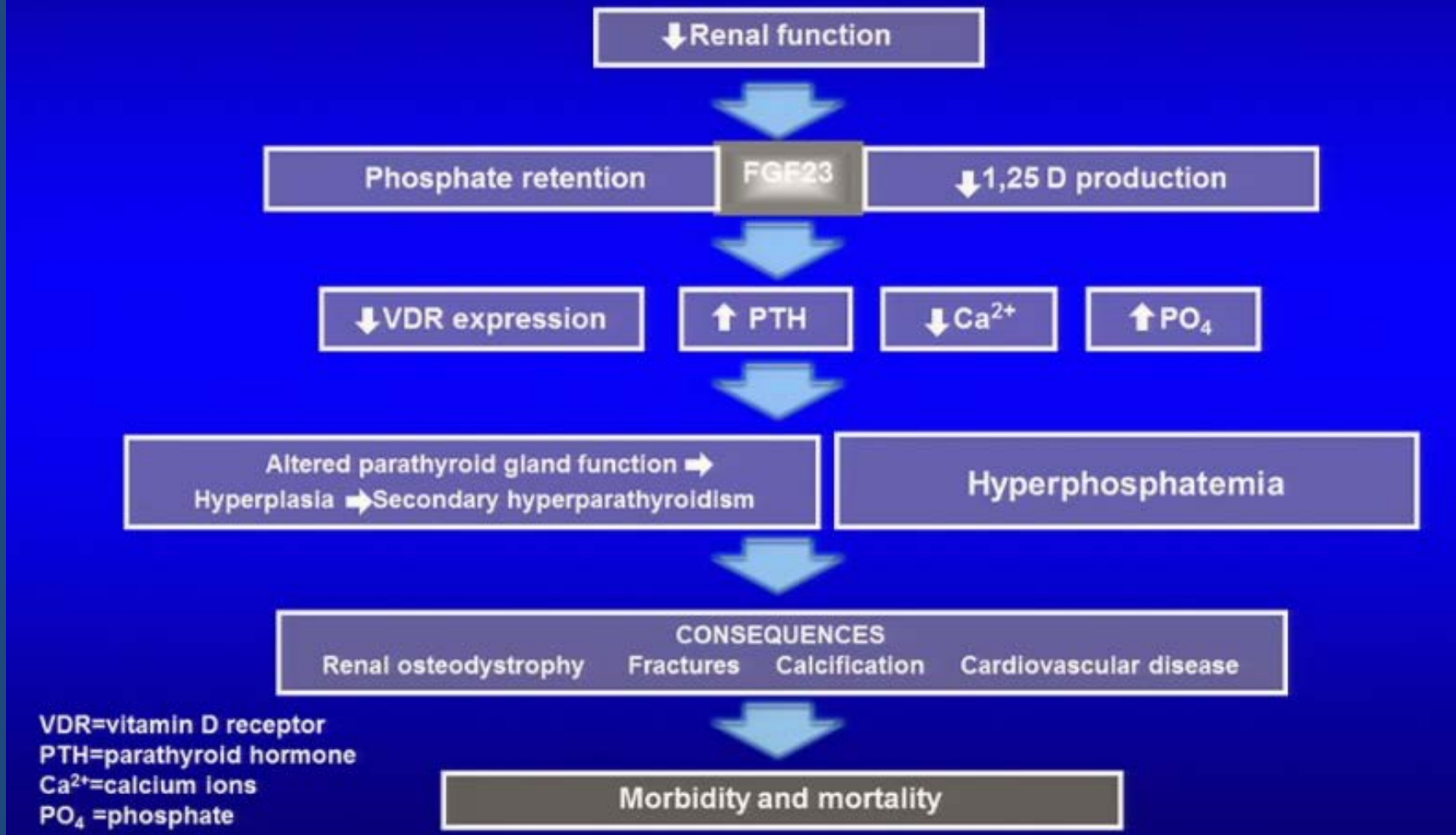
Green: low risk (if no other markers of kidney disease, no CKD); Yellow: moderately increased risk; Orange: high risk; Red, very high risk.

US Prevalence of CKD Stages



Adapted from Coresh J, et al. Prevalence of CKD in the US *JAMA*. Nov. 7, 2007;298:17.

Pathway of Chronic Kidney Disease - Mineral and Bone Disorder (CKD-MBD)



VDR=vitamin D receptor
PTH=parathyroid hormone
Ca^{2+}=calcium ions
PO_4 =phosphate

MRB 001 2009

Treat hyperphosphatemia in a patient with CKD

KDIGO guidelines 2016

2016 REVISED KDIGO CKD-MBD Recommendations
3.2.1. In patients with CKD Stages 3a-5D with evidence of CKD-MBD and/or risk factors for osteoporosis, we suggest BMD testing to assess fracture risk if results will impact treatment decisions. (*2B*)
3.2.2. In patients with CKD Stages 3a-5, it is reasonable to perform a bone biopsy if knowledge of the type of renal osteodystrophy will impact treatment decisions. (*Not Graded*)
4.1.1. In patients with CKD Stages 3a-5D, treatments of CKD-MBD should be based on serial assessments of phosphorus, calcium and PTH levels, considered together. (*Not Graded*)
4.1.2. In patients with CKD Stages 3a-5D, we suggest lowering elevated phosphorus levels towards the normal range. (*2C*)
4.1.3. In adult patients with CKD Stages 3a-5D, we suggest avoiding hypercalcemia (*2C*). In children with CKD Stages 3a-5D, we suggest maintaining serum calcium in the age-appropriate normal range. (*2C*)

1. Phosphorus-restricted diet
2. Phosphorus binders
   - Calcium-containing P04 binders:
     - Calcium carbonate (Tums)
     - Calcium acetate (PhosLo)
     - Appropriate for all hypo-calcemic patients and normocalcemic patients without vascular calcification or adynamic bone disease
   - Noncalcium-containing P04 binders:
     - Sevelamer (Renagel, Renvela)
     - Lanthanum (FosRenal)
     - Appropriate for hypercalcemic patients and patients with vascular calcification, adynamic bone disease, or patients on 1,25-OH vitamin D supplements

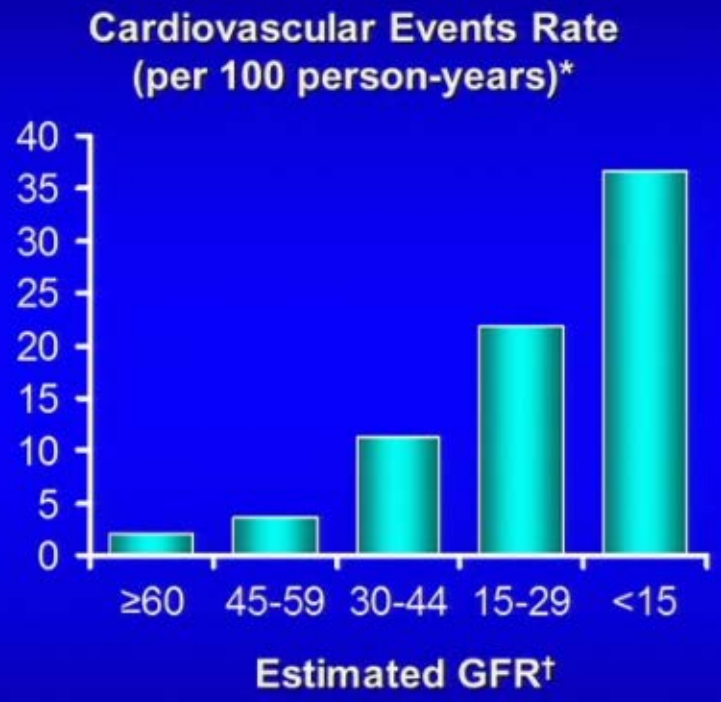
Management of Anemia in CKD

1) Rule out other causes of Anemia – bleeding, nutritional deficiences.

2) Once CKD cause of Anemia is established – Evaluate for iron deficiency – check Iron SAT and Ferritin , consier supplemental Fe (oral or IV) as needed, and Evaluate Hgb response

3) If anemia persists consider ESA's – Do Not Over-treat!!! Hgb >10 is current goal.

Renal Function vs Death, Cardiovascular Events, or Hospitalization Endpoints



N= 1,120,295

*Age-standardized rates

Go et al. *N Engl J Med.* 2004;351:1296-1305.

Question 6.

- Answer: C: Measure urine chloride level

Evaluate a Patient with Hypokalemic Metabolic Alkalosis

URINE CHLORIDE < 15 (LOW)=

Chloride Responsive (90%)

- Low effective circulating volume
- Volume depleted (orthostatic, hypotensive)
- NORMAL increase in renin, angiotensin, aldosterone
- Urine chloride LOW <15
- Cannot replace K until volume is replaced (GIVE sodium CHLORIDE)

URINE CHLORIDE >15 (HIGH) =

Chloride Unresponsive (10%)

- Hypertensive
- Hypervolemic
- ABNORMAL increase in aldosterone (Primary hyperaldo) or renin (Secondary hyperaldo)
- Urine chloride HIGH >15

Diagnosis of Metabolic Alkalosis

Once it has been determined that a patient has metabolic alkalosis, the etiology is usually obvious from the history. If there is no pertinent history, then one can assume that the alkalosis is due to one of the three most common causes: 1) vomiting, 2) diuretics, 3) mineralocorticoid excess. To differentiate between these conditions, it is usually helpful to measure the **urinary chloride concentration**.

In causes of metabolic alkalosis associated with a reduction in the ECV, there will be a stimulus for avid Na and Cl reabsorption to replenish extracellular volume. In these setting **urinary Cl should be expected to be very low, less than 25 meq/L**.

Urinary Na is not a reliable measure of extracellular volume in this setting because if the alkalosis is such that not all of the filtered HCO3- can be reabsorbed, then some will be excreted with Na and the urinary Na may be high. Thus, it may appear that the volume status is euvolemic or hypervolemic when it is not.

If the urinary Cl is low, indicating a hypovolemic state, then administration of NaCl and water to replenish the extracellular volume should stop the stimulus for aldosterone production and in turn should lead to appropriate excretion of excess HCO3- and improvement of hypokalemia. Thus, leading to correction of the metabolic alkalosis. Such causes of metabolic alkalosis are said to be **saline responsive.** See table below.

In contrast, states of **mineralocorticoid excess are associated with an expanded volume** and sometimes hypertension. The **urinary Cl will be high (> 40 meq/L).** In these patients, administration of saline would further expand the extracellular volume and worsen hypertension. It would not correct the alkalosis which is primarily due to hypokalemia. Such causes of metabolic alkalosis are said to be **saline resistant**.

Urine Cl < 25 meq/L Saline Responsive	Urine Cl > 40 meq/L Saline Unresponsive
Vomiting or nasogastric suction	Primary mineralocorticoid excess
Diuretics	Exogenous Alkali load
Posthypercapnia	Barrter's or Gitelman's syndrome
Cystic Fibrosis	Severe Hypokalemia (K< 2.0)
Low chloride intake	

Causes of saline resistant metabolic alkalosis can further be distinguished based on whether or not the patient is hypertensive. Mineralocorticoid excess states tend to be associated with hypertension while exogenous alkali load, Barrters and Gitelman's syndrome are associated with normal blood pressure.

Question 7.

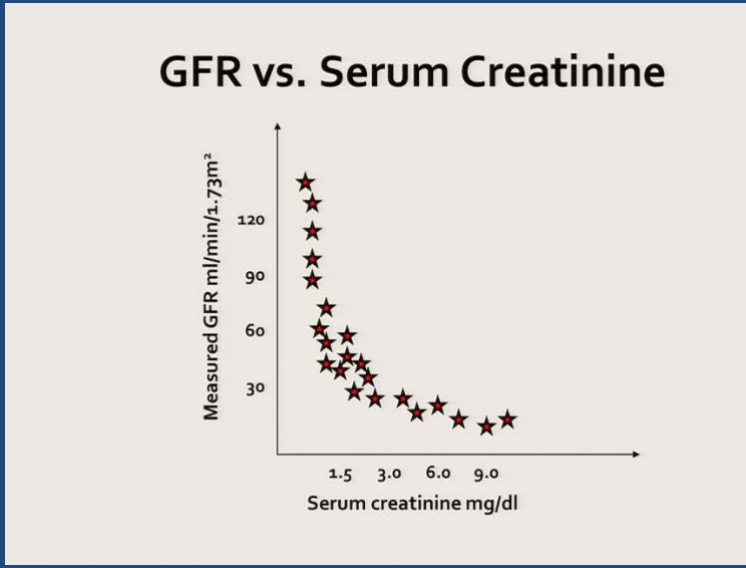
- Answer: E, Estimated GFR of < 15mL/min

Know how to appropriately dosing medications in acute kidney injury

Estimation of Creatinine Clearance

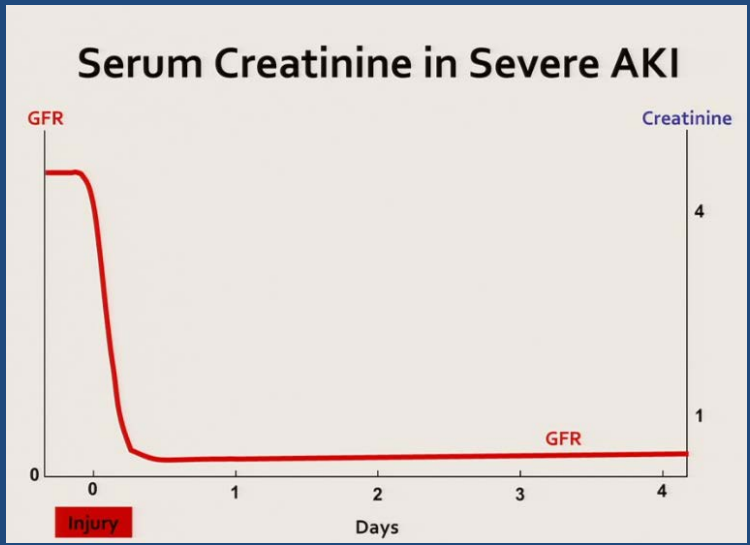
1. Crockcoft-gault
2. MDRD
3. CKD-EPI

All equations require steady-state, stable serum creatinine to be valid.



Epidemiology
AKI Hospital Admissions

Etiology	N	% of total
ATN	337	45
Prerenal	158	21
Obstructive	75	10
Glomerulonephritis	23	3
AIN	15	2
Vasculitis	11	2
Vascular	8	1



Question 8.

- Answer: B: Hypokalemic distal (type 1) renal tubular acidosis

Diagnose hypokalemic distal (type 1) renal tubular acidosis

	Type 1 RTA (distal)	Type 2 RTA (proximal)	Type 4 RTA (distal)
Chloride	↑	↑	↑
Bicarbonate	↓	↓	↓
Potassium	↓	**NL**	↑
Urine pH	**High (> 5.5)**	**Low (<5.5)** except with bicarb load	**Low (<5.5)**
Etiologies	**C**hronic hepatitis ***A**mphotericin B* **T**oluene **L**ithium **S**jogren's; **S**LE	**M**ultiple Myeloma **M**etal poisoning *Acetazolamide*	**D**iabetes mellitus **S**ickle cell *Spironolactone*
Associations	**Nephrolithiasis** due to hypercalcuria		

Question 9.

- Answer: B: Chlorthalidone

Manage hypercalciuria in a patient with nephrolithiasis

- Hypercalciuria:
  - >250 mg/24 hours WOMEN
  - >300 mg/24 hours MEN
  - > 200 mg/liter
- WORSENED by:
  - High sodium diet
  - High animal protein diet
  - Loop diuretics
- DO NOT advise a calcium restricted diet, as this increases GI absorption of oxalate, increasing oxaluria
- DO ADVISE:
  - Thiazide diuretic
  - Fluids > 2 liters/day
  - Low purine diet
  - Low sodium diet

Question 10.

- Answer: C; Oral sodium bicarbonate

Treating metabolic acidosis in CKD

Problem List:

- 58 year-old woman
- HTN, CKD stage 3b d/t analgesics
- Amlodipine
- Low serum bicarb, pH 7.36 , AG of 12
- Mild asymptomatic NAGMA

Non-AG metabolic acidosis:

- Renal (RTAs) – impaired acid excreation
- GI (diarrhea) – loss of bicarb
- Medications: Lithium, TPN

Our patients history suggests Type IV RTA

1. H/o analgesic nephropathy
2. Normal AG MA
3. Hyperkalemia

Treatment goal:

- Serum bicarb goal 23-29 which reduces risk of CKD progression
- Bicarbonate administration will help correct the acidosis.

- **Dose: 0.5 to 1mEq/kg/day**

Question 11.

- Answer: A; AL amyloidosis

Diagnose Multiple Myeloma as a cause of acute kidney injury

Clinical Features of MM:

- Anemia (NCNC) (80%)
- Bone pain (70%)
- Recurrent infections
  - 25% presenting
  - 75% during disease
- Renal complications (50%)
- Hypercalcemia (25%)
- Renal failure (25%)

Renal Complications:

- Tubular Damage
  - Adult Fanconi's syndrome
  - RTA Proximal Type 2
- Cast Nephropathy *
  - Proteinaceous casts clog the tubules resulting in tubule atrophy
- Glomerulopathy
  - Light chain disease deposition
  - Resulting in *albuminuria*!
- Exquisite sensitivity to IV contrast!

*most common

Amyloidosis: In an apple-green nutshell

Primary AL Amyloidosis

- Monoclonal protein related to plasma cell dyscrasia
- SPEP/IFE, UPEP/IFE, free light chains
- Bone marrow positive for plasma cell proliferation
- Monoclonal proteins infiltrate tissues; heart, kidney, GI tract, pleura, nerves (peripheral and autonomic)
- Kidney involvement may result in nephrotic proteinuria (albumin)

Secondary AA Amyloidosis

- ***Polyclonal protein*** related to inflammatory state; rheumatologic or infectious
- Juvenile RA, anklyosing spondylitis, psoriatic arthritis, IBD
- Familial Mediterranean Fever (60% of cases in Turkey)
- NEGATIVE SPEP/IFE etc for monoclonal proteins
- Kidney disease may result in nephrotic proteinurea (albumin)

Hereditary ATTR Amyloidosis

- *Mutation in the transthyretin gene* making abnormal proteins that infiltrate organs
- Cardiac and neurologic involvement more common than renal involvement
- NEGATIVE SPEP/IFE etc for monoclonal proteins
- Rare to have kidney involvement

Question 12.

- Answer: A: Interstitial nephritis

Diagnose Acute Interstitial Nephritis

Clinical Presentation:

- Fever, rash, eosinophilia, and elevated creatinine (10%)
- UA: WBC, WBC casts
- NEGATIVE CULTURE (sterile pyuria)
- Eosinophiluria (sensitive but not specific)
- < 2 gm/ 24 hr proteinuria

Causes: Drugs, Infections, Illnesses

- Antibiotics (B-lactam, cephalosporins, quinolones, Bactrim, rifampin)
- NSAIDS * (nephrotic proteinuria) with minimal change disease on biopsy
- Thiazides
- Proton Pump Inhibitors
- Phenytoin
- Allopurinol
- 5-aminosalicylates

Question 13.

- Answer: D; Continued current therapy

Diagnose and manage infection-related glomerulonephritis

- 24 y/o man IVDU
- H/o previous staph endocarditis with prolonged antibiotics
- Recent dx of MRSA endocarditis on IV vanco
- Worsening renal function

Laboratory Studies

Creatinine 2.8

Active UA with protein, RBCs and RBC casts

Low C3, nL C4

Negative cryoglobulins

Renal U/S with Doppler: normal kidneys

DDx:

- Acute interstitial nephritis
- Drug induced nephrotoxicity
- Infection related GN
- Septic emboli

Clinical picture and labs suggest infection related GN.

Treatment is to continue treating the infection.

Manage Post-Infectious Glomerulonephritis (PIGN)

- MANY bacteria, viruses and parasites can cause PIGN
- Most common nephritogenic strains of strep and staph
- Rapid onset of hypertension, oliguria, erythrocyte casts, and edema, LOW C3

Pathophysiology:

- Immune complex-mediated disease
- Complexes deposit in glomerulous and active complement, recruiting inflammatory cells and causing proliferative GN
- Common in non-strep infections (antigen is the infection)

Why isn't Vancomycin the culprit?

Drug induced tubular toxicity occurs 7 -10 days after Abx initiation and there is usually no cells in urine sediment.

Question 14.

- Answer: B; Ethylene glycol poisoning

Diagnose ethylene glycol poisoning

AGMA ← Toxic alcohol → NAGMA

Methanol

Sources: windshield wiper fluid, moonshine, perfumes
Effects: formic acid causes vision changes (blindness), “snowfield vision” (toxic to mitochondrial of retina and optic nerve), basal ganglia effects and pancreatitis
Labs: Anion gap + Osmolar gap

Isopropyl Alcohol

Sources: rubbing alcohol/hand sanitizer
Effects: hemorrhagic gastritis, fruity breath
Labs: Osmolar gap ONLY (no anion gap), ketones

Ethylene Glycol

Sources: antifreeze
Effects: oxalic acid causes cardiac and neuro effects
Labs: Anion gap + Osmolar gap, calcium oxalate crystals deposit in renal tubules, elevated creatinine, false lactic acid elevation from blood gas analyzer (serum lactate is normal)

Ethylene Glycol Poisoning

Work up:

- Check blood gas and electrolytes
- If +AGMA, check ketones, lactic acid and calculate osmolar gap

Osmolar gap =
measured – calculated osmolarity

Calculated osmolarity= (Na x 2) + (BUN/2.8) + (Glucose/18) = 287,
Measured= 314 - 287 = 27 gap.

>10 suggest the presence of an osmotically active substance
>20 is usually due to toxic alcohol ingestion, >50 for sure!

Treatment:

- Fomepizole (blocks alcohol dehydrogenase and autometabolizes)
- Ethanol administration is complicated by inebriation, erratic metabolism and hypoglycemia
- HD removes alcohol and toxic metabolites
- Dialyze if high levels of toxic alcohols, severe AGMA, electrolyte disturbances

Question 15.

- Answer: A: Cryoglobulinemia associated with Hepatitis C

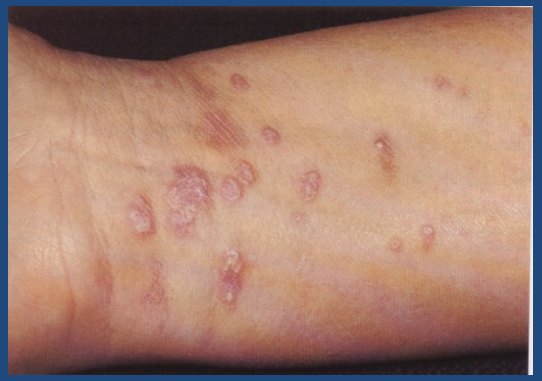
Diagnose Hepatitis C virus-associated membranoproliferative glomerulonephritis due to cryoglobulins

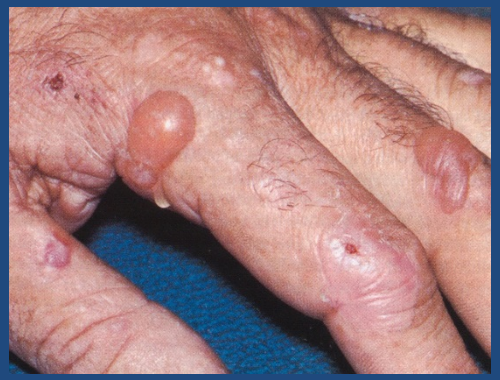
- Occurs in up to 20% of patients with chronic Hepatitis C
- Presents as membranoproliferative gnitis or mixed cryoglobulinemia (skin , kidney, and nerve involvement)

EXTRA-HEPATIC MANIFESTATIONS OF HEPATITIS C INFECTION:

1. Membranoproliferative GNitis
2. Mixed Essential Cryoglobulinemia
3. Lichen Planus
4. Porphyria Cutanea Tarda

- 1/3 relapsing dz with progression to ESRD
- Low complement (C4)
- + Rheumatoid factor
- TREAT Hep C virus

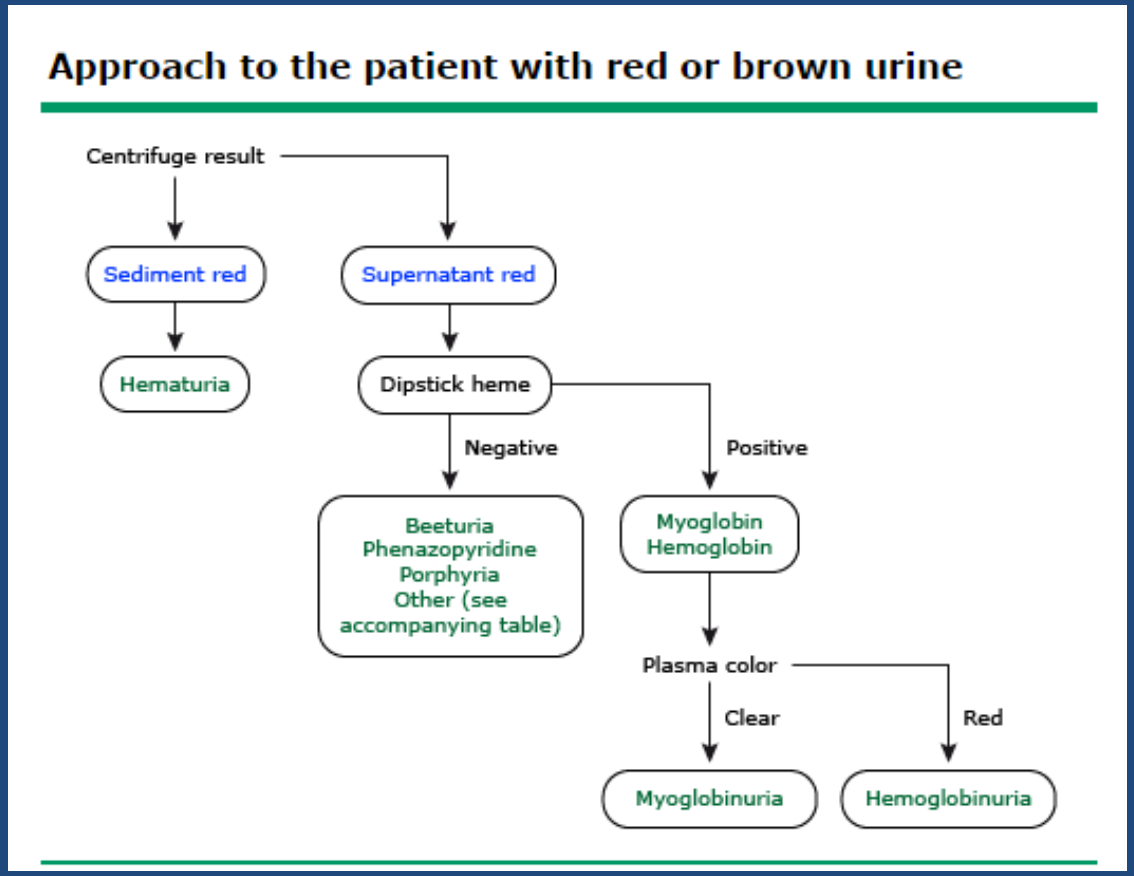




Question 16.

- Answer: C; Hemoglobinuria

Evaluation of Red Urine



Hemoglobinuria due to Intravascular hemolysis:

- Microangiopathic hemolytic anemia
- Transfusion reactions
- Mechanical shear from valves
- Infections
  - (Clostridial sepsis, Malaria)
- Paroxysmal Nocturnal Hemoglobinuria (PNH)
- Hypotonic fluid infusions
- Exposure to compounds with high oxidant potential (copper poisoning, Wilson's disease)

Question 17.

- Answer:B; HIV antibody test

Diagnose Focal Segmental Glomerulosclerosis:

the most common renal cause of nephrotic syndrome in US (36-80%)

Primary Disease:

Podocyte damage similar to minimal change dz

Secondary Disease Associations:

- Morbid obesity
- Heroin use
- HIV
- Vesicoureteral reflux

Risk factors for progression to ESRD:

- Black race
- >2 gm/24 hr proteinuria
- Low GFR
- BMI>27
- Hypertension

Clinical Manifestations:

- Acute onset of nephrotic syndrome with hematuria, renal failure and hypertension in primary disease;
- Asymptomatic subnephrotic to nephrotic proteinuria in secondary disease

Management:

- Immunosuppression with steroids or calcineurin inhibitors in primary disease
- ACEI +/- ARB in primary and secondary disease
- BP goal < 125/75 mm Hg

Question 18.

- Answer: B: Membranous nephropathy

Diagnose Membranous Nephropathy:

the second most common *renal* cause of nephrotic syndrome

Primary disease:

- Immune complexes of IgG react with antigens in the outer aspect of GBM
- Antibody to PLA2

Secondary disease associations:

- Malignancy
- Hep B and C
- NSAIDS

Risk factors for progression to ESRD:

- Male gender
- Age>50
- Persistent proteinuria > 4g/24 h over 6 months
- Declining GFR

Clinical manifestations:

- Nephrotic syndrome with preserved GFR
- Renal vein thrombosis
- Hypercoagulability

Management:

- ACE and/or ARB
- BP goal < 125/75 mm Hg
- Persistent proteinuria > 4g/24 hr
- Cyclophosphamide, corticosteroids, or calcineurin inhibitor with possible need for rituxan

Question 19.

- Answer: B; Hyperglycemia

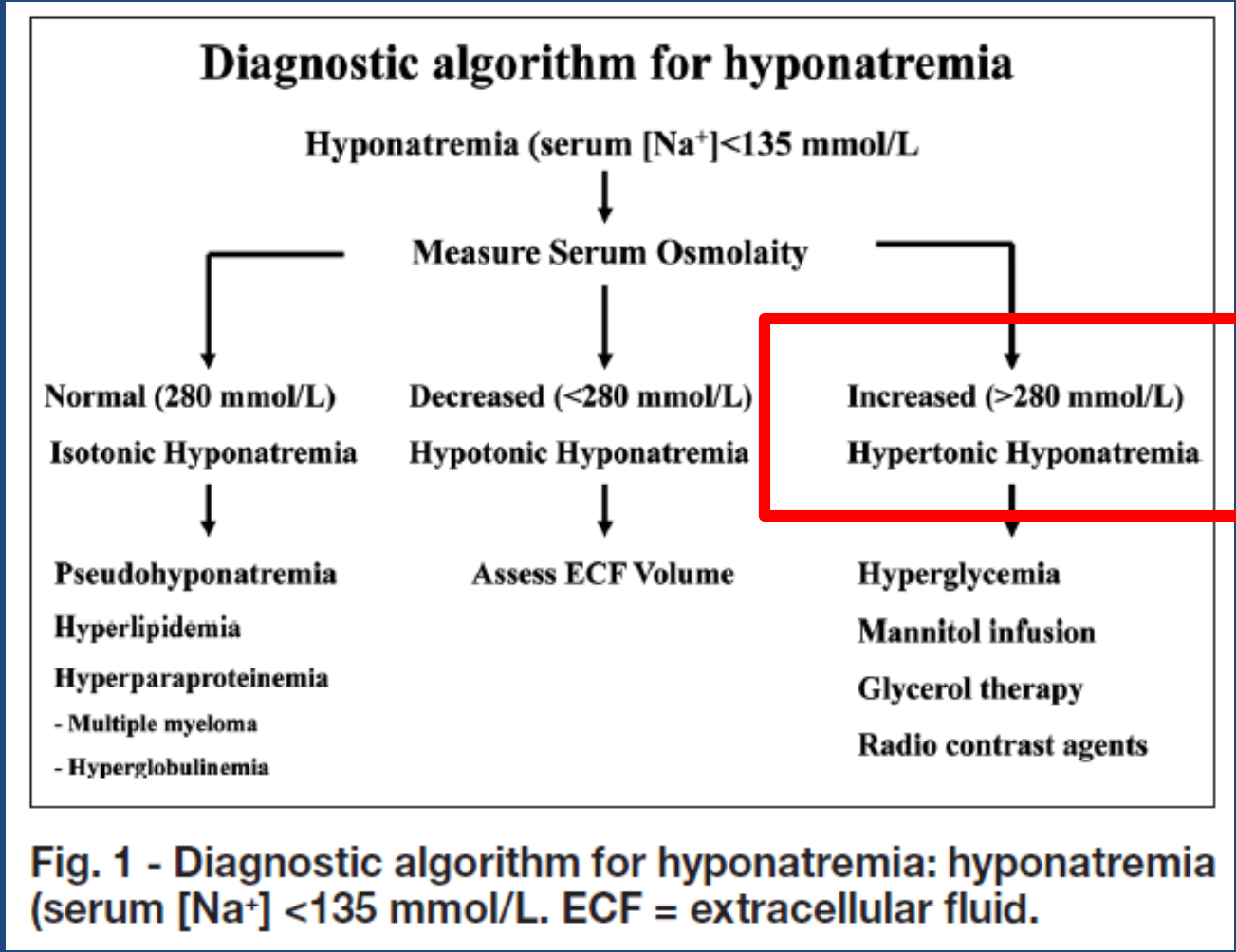


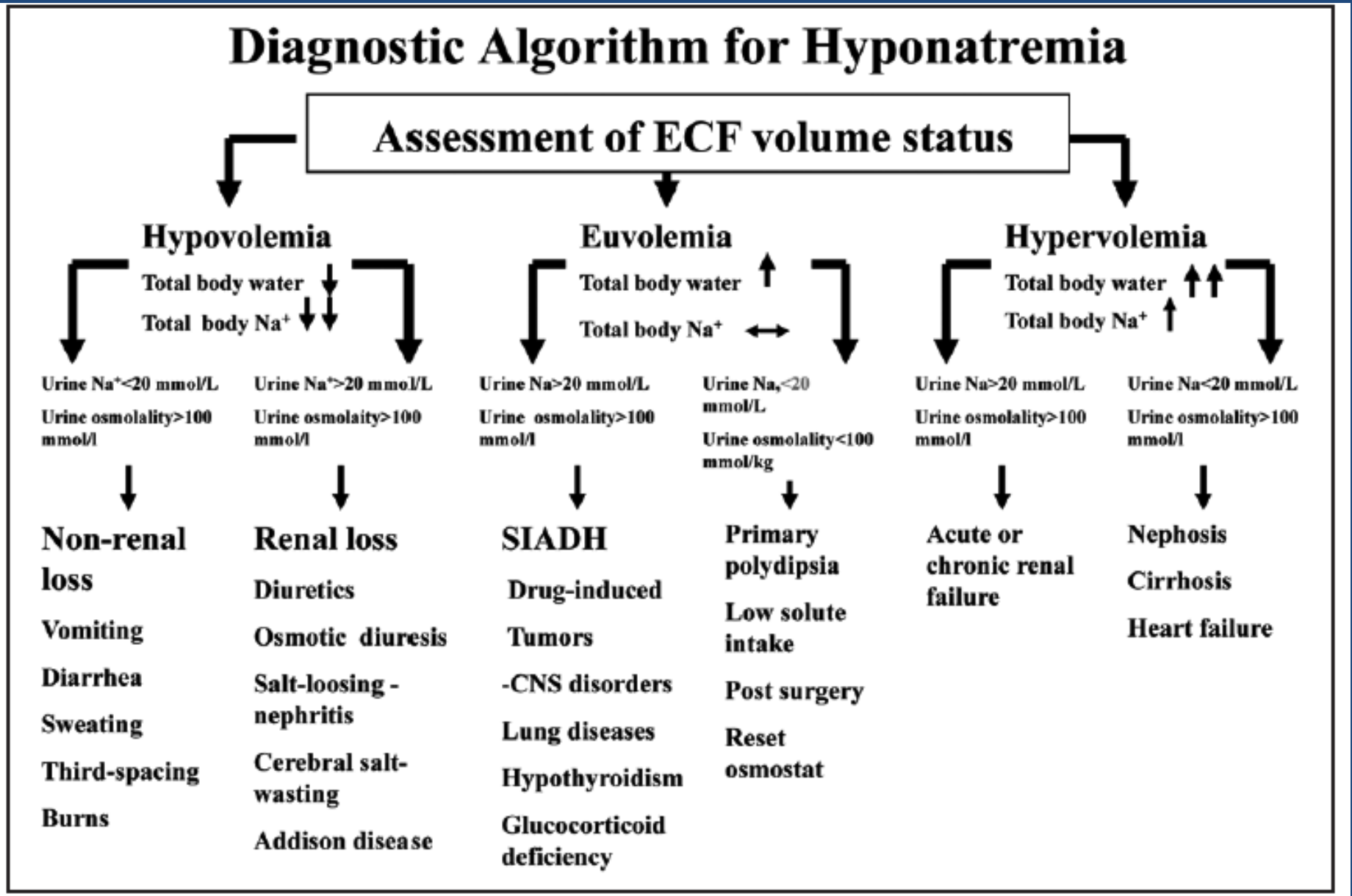
Fig. 1 - Diagnostic algorithm for hyponatremia: hyponatremia (serum [Na+] <135 mmol/L. ECF = extracellular fluid.

Patient has hypertonic hyponatremia!

Correction factor: 1.6mEq/L decrease in the serum sodium level per every 100mg/dL of plasma glucose over 100mg/dL.

Treatment: fluids and treat hyperglycemia.

Algorithm for hypotonic hyponatremia



Manage asymptomatic hypotonic hyponatremia

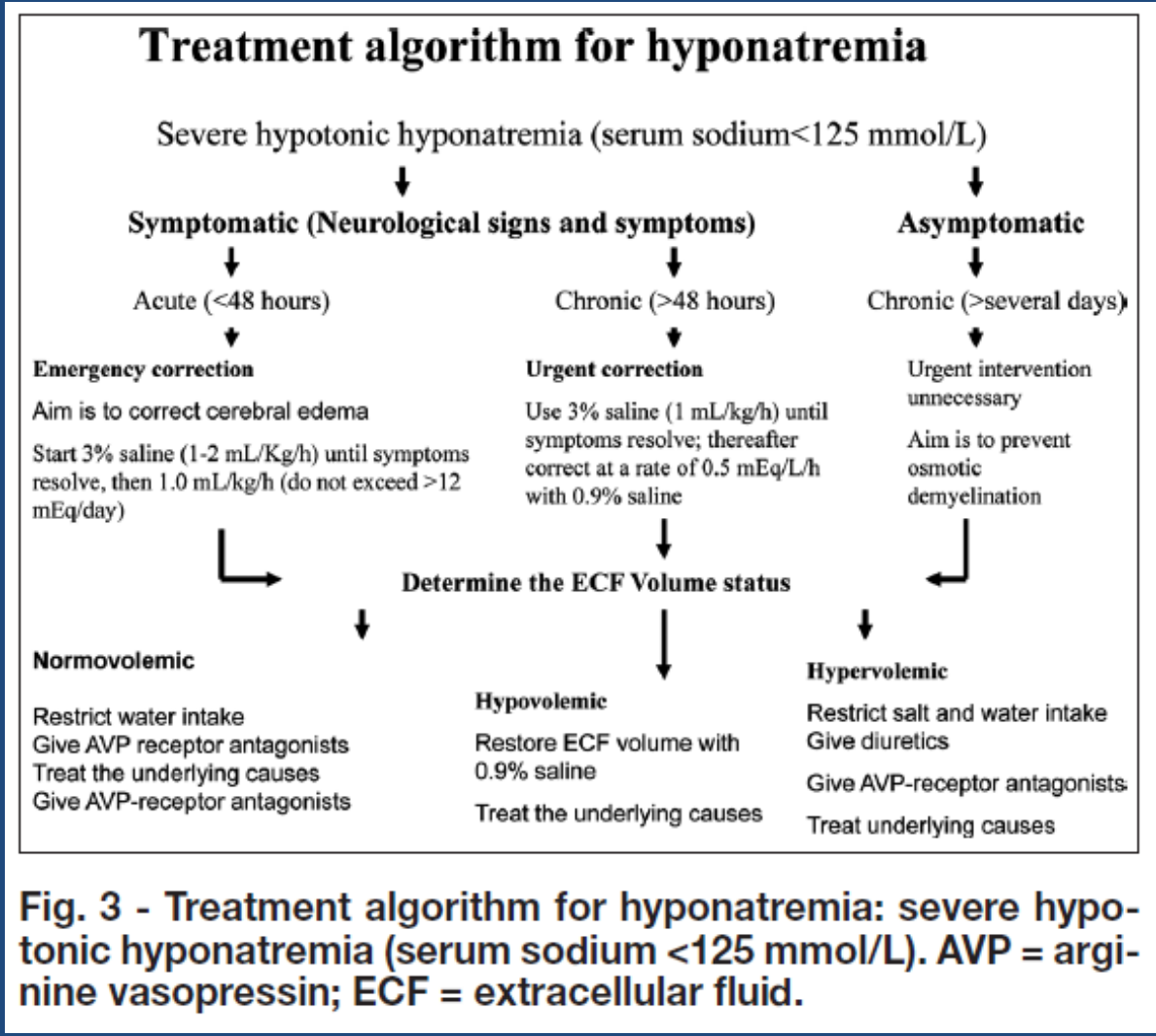


Fig. 3 - Treatment algorithm for hyponatremia: severe hypotonic hyponatremia (serum sodium <125 mmol/L). AVP = arginine vasopressin; ECF = extracellular fluid.

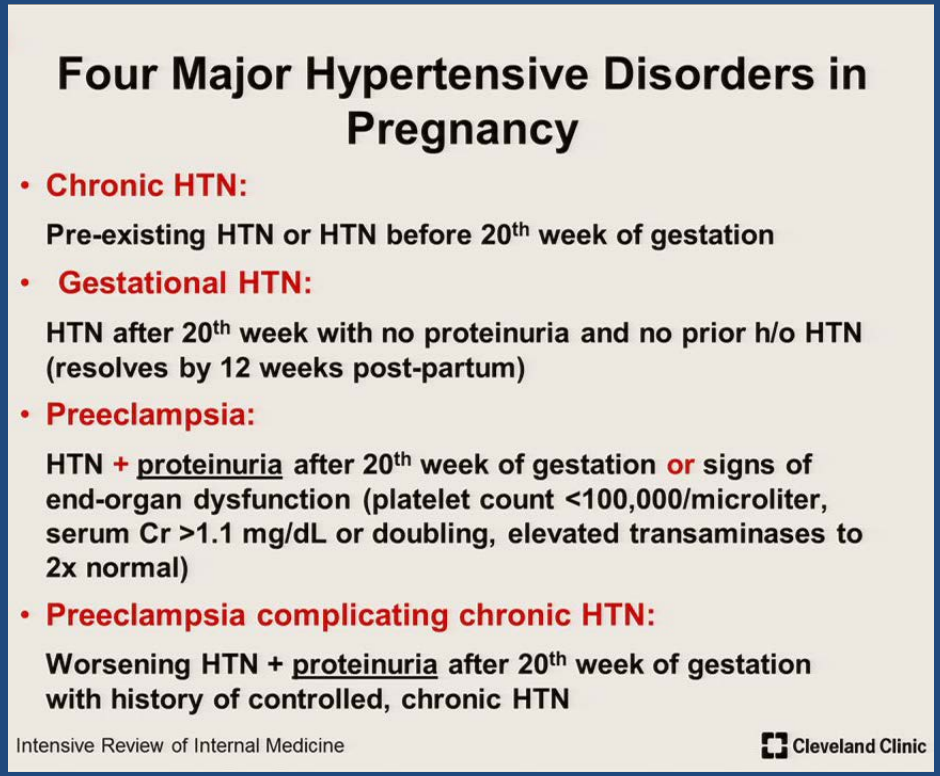
Question 20.

- Answer: B; Substitute labetalol for lisinopril

Manage HTN in a woman of childbearing age

Normal physiology in pregnancy

- Blood pressure lowers
- Plasma volume increases
- GFR increases
- Renal pelvis, calices, and ureters dilate
- Increased risk for pyelo
- Hyperventilation causes resp alkalosis with increased renal bicarb excretion



Pearls in Pregnancy "20 weeks":

ACEI, ARB, and Renin-Inhibitors are CONTRAINDICATED in pregnancy!!