Family medicine rotation notes

Topics I found them imp for mini OSCEs

- **HTN**
- **DM**
- **UTI**
- **Hyperlipidemia**
- **ECG (dx snd treatment)**
- **Subclinical hypothyroidism**
- **Goiter approach**
- **Hypothyroidism and hyperthyroidism differentials**
- **Headache types , and red flags**
- **Anemia differentials**
- **ABGs differentials**
- **Cranial nerves palsy**
- **Nail changes**
- **Jaundice differentials**
- **X-rays (chest :TB, pleural Effusion , pneumonia), abdomen:obstruction)**
- **Shingles**
- **Centor criteria**

Others (less imp)

Hep B serology, PFT , ulcers , BIRAD score

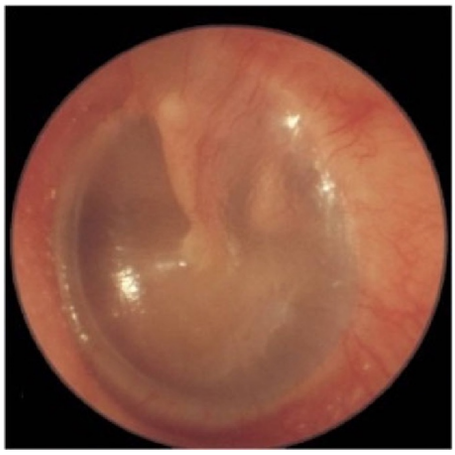
check the highlighted from from any source

Family medicine rotation notes

-B12 should be above 400
If the B12 is low we give the patient B12 injection 1000 mcg -1 every week for 2 month , then 1 every month

-Vitamin D deficiency treatment
One pill 50,000 every week for 2 month then 1 pill every month .

-normal tympanic membrane



Hyperlipidemia

Laboratory parameter	Optimal level
Total cholesterol	• < 200 mg/dL
Triglycerides	• < 150 mg/dL
LDL	• ~~< 100~~ 130 mg/dL
HDL	• ≥ ~~60~~ 55 mg/dL

Treatment of hypercholesterolemia in adults [11]

Indications for treatment [11]

Previous IHD, CAD, CVA, TIA

- Patients ≥ 20 years of age with clinical ASCVD: Consider high-intensity statin therapy. (3)
- Patients 20–75 years of age and LDL ≥ 190 mg/dL: high-intensity statin therapy (1)
- Patients 40–75 years of age and LDL 70–189 mg/dL: Treatment is based on the 10-year ASCVD risk. ACC Score
  - High (≥ 20%): high-intensity statin therapy
  - Borderline to intermediate (5–20%)
    - Review ASCVD risk-enhancing factors and consider moderate-intensity statin therapy depending on result.
    - If the benefit of treatment is unclear in patients with intermediate-risk, consider coronary artery calcium scoring.
- Patients 40–75 years of age with diabetes mellitus (2)
  - Initiate moderate-intensity statin therapy.

goal LDL ≤ 70

- Patients 20–39 years of age if LDL ≥ 160 mg/dL and family history positive for premature ASCVD: Consider statin therapy.

Treatment of hypertriglyceridemia in adults [11]		
	Definition	**Treatment**
Moderate hypertriglyceridemia	• Fasting or nonfasting TG 175–499 mg/dL	• In all patients > 20 years ◦ Recommend lifestyle changes to address obesity and metabolic syndrome. ◦ Manage associated conditions. ◦ Avoid triglyceride-raising medications (see "Etiology").
Severe hypertriglyceridemia	• Fasting TG ≥ 500 mg/dL	• Consider statin treatment in patients with intermediate to high 10-year ASCVD risk. • Recommend low-fat diet and alcohol avoidance. • If persistent, consider one of the following to reduce risk of pancreatitis: ◦ Omega-3 fatty acids, e.g., omega-3-acid ethyl esters DOSAGE ◦ Fibrates, e.g., fenofibrate DOSAGE or gemfibrozil DOSAGE

Tests that should be done with **high BP :**
CBC , KFT and electrolytes, urine analysis , LFT , TSH , A1C or fasting glucose , ECG , Echo , lipid profile .

Hyperlipidemia Screening :

- **Screening for lipid disorders**
  - Indications
    - All individuals 40–75 years of age [12][13] (Avg male : 35 , Female : 45)
    - Patients with a family history of familial hypercholesterolemia or premature ASCVD: Consider earlier screening. (20- 39)
  - Tests
    - Adequate in most cases: nonfasting lipid profiles
    - Nonfasting triglycerides > 400 mg/dL or if evaluating for familial lipid disorders: **fasting lipid profile**
- Assess ASCVD risk to guide treatment decisions, e.g., via the 2013 ACC/AHA pooled cohort equation

With **hyperlipidemia,** we should check **blood pressure** and **HbA1C and LFT for baseline**

Hyperlipidemia drugs

Low Intensity	moderate intensity	High Intensity
Simvastatin	Atorvastatin (20-40)	Atorvastatin (40 - 80)
	Rosuvastatin (10-20)	Rosuvastatin (20-40)

Urinary symptoms and diagnosis

Urinary Symptoms
" Dysurea
Urgency
frequency "
→ UTI Cystitis (usually No fever)
→ Urine analysis Culture (↑WBC & RBC)
→ treated empirically if not reccurent
non- pregnant : Bactrim (Trimethoprim – Sulfamethoxazol)
pregnant : Zinnat cefuroxime

UTI education
avoid Coffee
Drink water
Regular Urination
Good Hygine

Urinary Symptoms
+
Fever
+
Flank Pain
+
Renal angel tenderness
→ pyelonephritis → Urine analysis
Culture
CBC
CRP
KFT

Flank Pain
+
Urinary Symptoms
→ Renal Stones → Urine analysis
Culture
CBC
KFT
(↑ RBC) & return to Clinic

Urinary tract infection

Classification of urinary tract infections [9]		
		Details
By clinical presentation [10]	**Asymptomatic bacteriuria (ASB)**	• Significant bacteriuria without clinical features of UTI
	Urinary tract infection (UTI)	• Bacteriuria and clinical features of UTI
By location [11]	**Lower UTI**	• Infection of the bladder (**cystitis**), the most common location of UTIs • Often accompanied by urethritis (urethritis in isolation is suggestive of STI) • Can be associated with prostatitis in men
	Upper UTI	• Infection of the kidneys and ureter (pyelonephritis)
By severity [12][13][14]	**Uncomplicated UTI**	• Infection in nonpregnant, premenopausal women without further risk factors for infection, treatment failure, or serious outcomes [14]

	Complicated UTI (cUTI)	• Infection in patients with risk factors for infection, treatment failure, or serious outcomes, including:
		  ◦ Male sex [14]
		  ◦ Pregnancy
		  ◦ Postmenopause
		  ◦ Children with features of atypical pediatric UTI
		  ◦ Significant anatomical or functional abnormalities
		  ◦ Immunosuppression
		  ◦ Renal failure
		  ◦ Metabolic disorders (e.g., diabetes)
		• Infection associated with recent instrumentation or medical devices, e.g.:
		  ◦ Cystoscopy
		  ◦ Indwelling catheters
		  ◦ Drainage devices (e.g., ureteral stents, nephrostomy tubes)
		• Healthcare-associated UTIs (see below)

	Urosepsis	• UTI associated with a dysregulated immune response that can potentially lead to life-threatening organ dysfunction (See also "Sepsis.") [15]
By source of infection [16] [17]	**Community-acquired UTI**	• UTI acquired outside of a healthcare setting and/or UTI that manifests within 48 hours of hospital admission

	Healthcare-associated UTI	• UTI acquired in a healthcare setting
		• Among the most prevalent healthcare-associated infections [18]
		• **Nosocomial UTI**
		  ◦ Most common: **catheter-associated UTI** (CAUTI)
		  ◦ Can also occur secondary to urinary tract instrumentation

By frequency [10]	**Recurrent UTI**	• ≥ 3 episodes of symptomatic, culture-proven UTI in one year or ≥ 2 episodes in 6 months

Pathogens

Bacteria

- **Infection ascends** from the urethra to the bladder.
- Can ascend further to the ureters and the renal pelvises (see "Pyelonephritis")
- **Bacteria that cause UTI**
  - **Escherichia coli**: leading cause of UTI (approx. 80%) [1]
  - **Staphylococcus saprophyticus**: 2nd leading cause of UTI in sexually active women
  - Klebsiella pneumoniae: 3rd leading cause of UTI
  - Proteus mirabilis
    - Produces ammonia, giving the urine a pungent or irritating smell
    - Associated with **struvite stone** formation
  - Nosocomial bacteria: Serratia marcescens, Enterococci spp., and Pseudomonas aeruginosa are associated with increased drug resistance.
  - Enterobacter species
  - Ureaplasma urealyticum

Host-dependent factors

- **Structural or functional abnormalities of the urinary tract** [6]
  - Prevent bladder emptying and/or result in urinary stasis
  - Examples include:
    - Benign prostatic hyperplasia
    - Congenital malformations causing vesicoureteral reflux
    - Urinary bladder diverticulum
    - Neurogenic bladder
    - Urinary tract calculi
- **Sex**
  - Female individuals: anatomically predisposed because the urethra is shorter and anal and genital regions are in close proximity → bacteria spreading from the anal region → colonization of vagina → ascending UTIs [7]
  - Male individuals: higher risk in uncircumcised male infants [8]
- **Pregnancy**: hormonal changes during pregnancy → urinary stasis and vesicoureteral reflux → increased risk of UTIs
- **Postmenopause**: ↓ estrogen → ↓ vaginal lactobacilli → ↑ vaginal pH → ↑ colonization by E. coli [7]
- **Chronic constipation**: common cause of UTIs in children
- **Prior conditions**
  - Previous UTI [7]
  - History of kidney surgery
  - Immunosuppression
  - Diabetes mellitus
- **Medication**: recent use of antibiotics

Clinical features

- **Clinical features of lower UTI** [19][20]
  - Irritative **lower urinary tract symptoms** (LUTS)
    - Increased urinary frequency
    - Urinary urgency
    - Dysuria
  - Hematuria
  - Suprapubic tenderness
- **Clinical features of upper UTI** (pyelonephritis) [21]
  - Symptoms of lower UTI
  - **Fever**
  - Flank pain
  - Costovertebral angle tenderness
  - Fatigue/malaise
  - Nausea and vomiting
  - See "Clinical features" in "Pyelonephritis."
- **Additional features (special patient groups)** [19]
  - Male individuals: pain in the prostatic/perineal area
  - Children: See "Clinical features of pediatric UTI."
  - Older adults: delirium/acute confusion [22]

Urinalysis [24][25]

- **Indications**: **best initial test** for all patients
- **Procedure**: visual, chemical (dipstick), and microscopic examination of urine
- **Specimen collection method**
  - Clean-catch midstream sample: thought to reduce contamination with vaginal or skin flora
  - Straight catheterization of the bladder: may be considered if the risk of contamination is high
  - Suprapubic aspiration: no contamination if performed correctly but rarely used due to its invasive nature
- **Typical urinalysis findings of UTI** [9]
  - **Pyuria**: presence of white blood cells (WBCs) in the urine
    - **Positive leukocyte esterase**: an enzyme produced by WBC
    - ≥ 5 WBC/HPF or ≥ 8–10 WBC/mm^3 [24][25]
  - **Bacteriuria**: presence of bacteria in the urine [26]
    - Positive **urinary nitrites**: indicate bacteria that convert nitrates to nitrites (most commonly gram-negative bacteria; e.g., E.coli)
    - Direct visualization by gram stain (seldom performed)
- **Other findings**
  - Leukocyte casts may indicate pyelonephritis.
  - Micro- or macroscopic hematuria may be present.
  - Alkaline urine (pH > 8) and struvite crystals in sediment: indicate urease-producing organisms (e.g., Proteus, Klebsiella, Staphylococcus saprophyticus) [19][27]
  - The presence of squamous epithelial cells can be a sign of contamination.

Urine culture [24][9][14]

- **Indications**
  - Suspicion for complicated UTI or healthcare-associated UTI
  - Suspicion for pyelonephritis or urosepsis
  - Suspicion for uncomplicated cystitis with either of the following:
    - History of recurrent UTIs [10]
    - Equivocal urinalysis
    - Atypical symptoms
    - Concern for multiresistant pathogens, e.g., due to recent antibiotic use [15]
    - Age ≥ 65 years [28]
  - Follow-up cultures for test of cure in the following cases:
    - Nonresolving symptoms despite antibiotic treatment
    - Anatomic or functional abnormalities of the urinary tract
    - Continued pathological findings on urinalysis
- **Interpretation**
  - Cultures are considered positive if either of the following is present:
    - **Significant bacteriuria**: defined as **≥ 10^5 CFU/mL** in a clean-catch specimen [9][29]
    - Any organisms in a specimen obtained by suprapubic aspiration [24][30]
- **Typical colony findings**
  - E. coli: intensely pink on MacConkey agar
  - Klebsiella pneumoniae: viscous colonies
  - Serratia marcescens: often red in appearance
  - Proteus mirabilis: swarming motility pattern
  - Pseudomonas aeruginosa: blue-green pigment

> In patients with complicated or recurrent urinary tract infections, a urine culture should be obtained prior to initiating antibiotic treatment. False negative results are possible if a culture is obtained after the patient has received antibiotics.

Additional diagnostics [15][9][23]

- **Pregnancy test**: indicated in women of childbearing age
- **Testing for sexually transmitted infections (STIs)**
  - Indicated in patients with STI risk factors and/or symptoms of an STI
  - At-risk patients should be tested for Chlamydia trachomatis and Neisseria gonorrhoeae.
- **Blood tests**
  - Not routinely performed in patients with lower UTI
  - May be indicated to assess concomitant conditions (e.g., diabetes mellitus) and exclude differential diagnoses

Imaging [15][14][33]

Imaging is generally not indicated or helpful for the diagnosis of lower UTI, but it may be performed in select patients to rule out complicating factors (e.g., urinary tract obstruction) or if complicated pyelonephritis or urosepsis are suspected. For imaging indications and findings in upper UTI, see "Diagnostics" in "Pyelonephritis."

- **Indications may include**:
  - Suspected urinary tract obstruction
  - Severe illness (e.g., septic shock)
  - Early recurrence of UTI (within two weeks of appropriate treatment)
  - Persistent bacteriuria despite treatment
  - Recurrent complicated UTI
  - Men with febrile UTI
- **Modalities**
  - CT abdomen and pelvis with or without IV contrast
    - Most sensitive for initial imaging
    - Noncontrast CT is useful to diagnose urolithiasis.
    - IV contrast is indicated if complications (e.g., abscess) or other causes of obstruction are suspected.
  - Ultrasound of the kidneys and bladder
    - Perform if there are contraindications to contrast or radiation.
    - Useful for detecting hydronephrosis and measuring postvoid residual volume if an obstruction is suspected
  - Additional modalities include MRI abdomen and pelvis, voiding cystourethrography, and retrograde cystography.

Empiric antibiotic treatment of uncomplicated lower UTIs

- **First-line treatment**
  - Nitrofurantoin for 5 days
  - Trimethoprim/sulfamethoxazole (TMP/SMX) for 3 days
  - Fosfomycin (single dose)
- **Second-line treatment**: beta-lactam antibiotics for 5–7 days
  - Aminopenicillins plus beta-lactamase inhibitors, e.g., amoxicillin/clavulanic acid
  - Oral cephalosporins, e.g., cefpodoxime, cefdinir, or cefaclor
- **Alternatives**: Consider fluoroquinolones, e.g., ciprofloxacin for 3 days for patients with previous infections with bacteria resistant to other drug classes. [46]

Antibiotic treatment of complicated lower UTIs [47][14]

- Antibiotic therapy must be adapted to culture results and is commonly given for **7–14 days**.
- Options for the initial empiric treatment of complicated lower UTIs include:
  - Fluoroquinolones PO or IV: e.g., ciprofloxacin or levofloxacin
  - Beta lactams
    - Second-generation or third-generation cephalosporins: e.g., ceftriaxone
    - Extended-spectrum penicillins: e.g., ampicillin/sulbactam
- Reasonable options if the pathogen is susceptible include: [49]
  - Nitrofurantoin
  - TMP/SMX
  - Fosfomycin (multiple doses)

Recurrent UTI [10][54]

Recurrent UTIs are common in women and are defined as ≥ 3 episodes of symptomatic, culture-proven UTI in one year or ≥ 2 episodes in 6 months. Management involves the implementation of preventive measures and antibacterial prophylaxis in addition to the antibiotic treatment of acute episodes.

Acute management

Whenever possible, obtain a urine culture for every episode prior to initiating antibiotic therapy.

- **Choice of antibiotic**
  - First recurrence: See "Antibiotic treatment for uncomplicated lower UTIs" and "Antibiotic treatment for complicated lower UTIs" for initial empiric regimens.
  - Frequent recurrences
    - Regimens should be tailored to the patient and prior culture results.
  - Antibiotics must be adapted to the current culture results once available.

Glycosuria	• Glucose in the urine • Occurs when blood glucose levels exceed 180 mg/dL (renal threshold for reabsorption of glucose)	• Diabetes mellitus
Ketonuria	• Ketones in the urine	• Diabetic ketoacidosis • States of starvation
Proteinuria	• **> 150 mg protein/day** in the urine	• Diabetic kidney disease • Hypertensive nephropathy • Glomerulonephritis (e.g., minimal change disease, focal segmental glomerulosclerosis) • Fever, intense exercise, dehydration • Multiple myeloma
Bacteriuria	• Bacteriuria: presence of bacteria in urine • Significant bacteriuria: **≥ 10^5 colony-forming units/mL** in midstream urinary sample	• Urinary tract infection

Pyuria	• White blood cells in the urine	• Urinary tract infection
		• **Sterile pyuria**
		  ◦ Acute tubulointerstitial nephritis
		  ◦ Glomerulonephritis (see nephritic syndrome)
		  ◦ Urogenital tuberculosis

Hematuria	• Red blood cells in the urine	• Benign prostatic hyperplasia • Urinary tract infection • Urolithiasis • Glomerulonephritis (see nephritic syndrome) • Polycystic kidney disease • Malignancy (e.g., bladder cancer, renal cell carcinoma)

Hemoglobinuria	• Hemoglobin in the urine	• Severe intravascular hemolysis
		  ◦ Microangiopathic hemolytic anemia
		  ◦ Paroxysmal nocturnal hemoglobinuria
		  ◦ G6PD deficiency
		  ◦ Malaria (especially Plasmodium falciparum)

Myoglobinuria	• Myoglobin in the urine	• Rhabdomyolysis

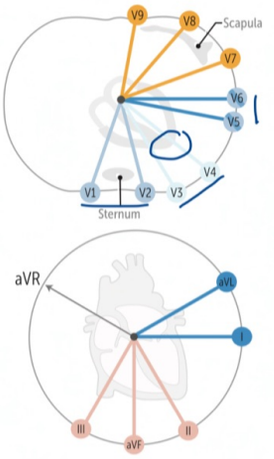
Partialy treated UTI

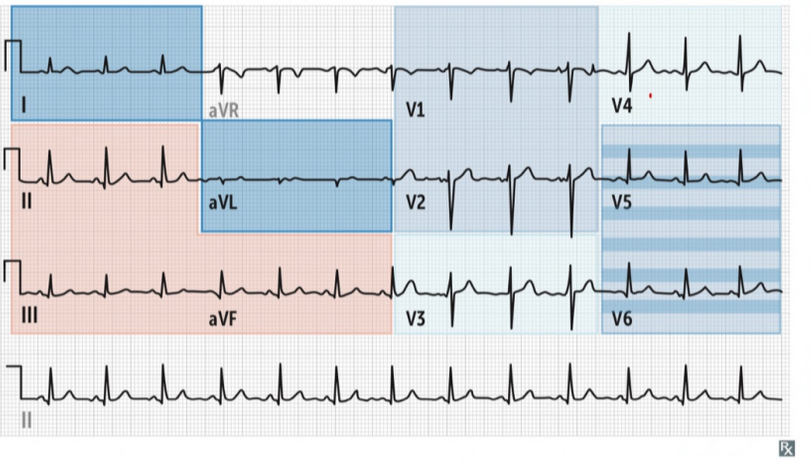
Esophegeal varices painless while mallory weiss syndrome is painful

ECG localization of STEMI

INFARCT LOCATION	LEADS WITH ST-SEGMENT ELEVATIONS OR Q WAVES
Anteroseptal (LAD)	V_1–V_2
Anteroapical (distal LAD)	V_3–V_4
Anterolateral (LAD or LCX)	V_5–V_6
Lateral (LCX)	I, aVL
InFerior (RCA)	II, III, aVF
Posterior (PDA)	V_7–V_9, ST depression in V_1–V_3 with tall R waves

LAD : septum, anterior
LCX : Lateral
Inferior : RCA
Posterior : PDA





Osteoporosis

Diagnosis

Osteoporosis is diagnosed in patients with a T-score ≤ -2.5 SD and/or a fragility fracture. [11]

Bone mineral density (BMD) assessment [11][12]

Indications

- Evaluation of suspected osteoporosis
- Screening for osteoporosis in asymptomatic high-risk individuals

Preferred modality: dual-energy x-ray absorptiometry

DXA measures BMD at the lumbar spine and hip/femoral neck using two x-ray beams. Findings are represented in terms of BMD scores that compare results to a reference population.

BMD scores [12][11]	
Postmenopausal women and men > 50 years of age	• BMD is calculated using the **T-score**. • **T-score ≤ -2.5 SD indicates osteoporosis** [11][12] • T-score -1 to -2.5 SD indicates osteopenia [11][12] • T-score ≥ -1 SD is normal [11][12]
All other individuals	• BMD is calculated using the Z-score.

Lab tests

- CBC, CMP, PTH, phosphate, and serum 25-hydroxyvitamin D
- 24-hour urine to measure calcium, creatinine, and sodium levels

Comprehensive metabolic panel

Abbreviation: CMP

A laboratory test that typically measures several components of the serum, including the concentrations of sodium, potassium, bicarbonate, chloride, creatinine, blood urea nitrogen, glucose, calcium, total protein, albumin, alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, and bilirubin.

Primary osteoporosis: Serum calcium, phosphate, and parathyroid hormone (PTH) levels are usually normal

Treatment

- Optimize calcium and vitamin D intake.
- Treat vitamin D deficiency.
- Encourage physical activity, including strength (resistance) and balance training.
- Avoidance or minimization of the following:
  - Tobacco use
  - Excessive alcohol consumption
  - Glucocorticoid use
- Bisphosphonates : in all patients , inhibit osteoclasts , Side effects : osteoarthritis in jaw , hypocalcemia, esophagitis

Oral bisphosphonates should be taken in the morning with plenty of water at least 30 minutes before food and other medication, and the patient should maintain an upright position for at least 30 minutes after intake to prevent esophagitis.

Estrogen is not approved for the treatment of osteoporosis in women; if estrogen is prescribed to a patient with a uterus, it should always be combined with progesterone therapy to reduce the risk of endometrial hyperplasia

Sinusitis : Inflammation of paranasal sinuses

Rhinosinusitis: simultaneous inflammation of the nasal mucosa and sinuses

Sinuses :
Maxillary (largest)
Sphenoidal
Frontal
Ethmoidal
-The nasal meatuses drain the sinuses

> A clinical diagnosis of chronic sinusitis should be confirmed with objective documentation of sinonasal inflammation, which can be accomplished via anterior rhinoscopy, nasal endoscopy, or CT scan

Classified into :
-Acute up to 1 month :

Most cases , usually by viral infection (rhino virus , parainfluenza , influenza) , bacterial infection (strep pneumonia, hemophilia influenza)

-Subacute 1-3 months
-Chronic > 3 months
Allergies (dust ..) , fungal infection (in immune compromised patients)
Chronic > hyperplasia > nasal polyps

Symptoms
Facial pain , pressure in the face , headache : due to mucus accumulation
Fever : due to bacterial infection
Changes in voice

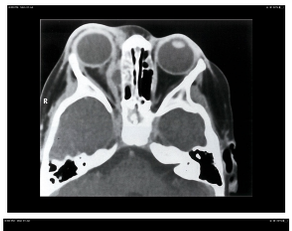
Diagnosis
Clinical Based on symptoms
Sometimes: CT scan or rhinos-copy

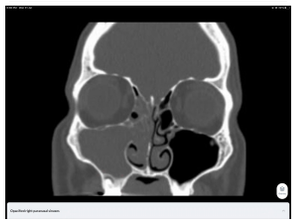
Imaging [6][17][14]

- **CT maxillofacial with or without IV contrast**
  - Findings may include signs of:
    - Rhinosinusitis: opacification, mucosal thickening, air-fluid levels, soft tissue swelling [15]
    - Complications: spread beyond the sinuses
    - Underlying causes of CRS: e.g., anatomic abnormalities, osteomeatal obstruction, polyposis
    - Aggressive fungal infection or neoplasm: e.g., osseous destruction, extrasinus extension, local invasion [6][13]
- **MRI with and without IV contrast**: can be used to evaluate for intracranial or intraorbital involvement or to differentiate polyps from tumors [6][14][17]
- **X-ray sinuses**
  - No longer recommended due to limited sensitivity and specificity
  - May show sinus opacification and air-fluid levels

Direct visualization

- **Indications**: evaluation of complicated rhinosinusitis, recurrent ARS, or CRS
- **Modalities**
  - Nasal endoscopy (preferred)
  - Anterior rhinoscopy
- **Findings**
  - Signs of rhinosinusitis: mucosal erythema, edema, osteomeatal blockage with purulent drainage [6]
  - Detection of structural abnormalities or masses, if present
  - Signs of fungal rhinosinusitis: necrosis





Treatment
Bacterial > Abx
Decongestants
Allergy > steroids
Nasal saline irrigation
Oral analgesics

	Adults
First-line treatment	• Amoxicillin • OR amoxicillin/clavulanate
Penicillin allergy	• Non-type I hypersensitivity to penicillin [6] ◦ Clindamycin [22] ◦ PLUS a third-generation cephalosporin • Type I hypersensitivity to penicillin ◦ Doxycycline ◦ OR a respiratory fluoroquinolone
Inpatient treatment	• Ampicillin/sulbactam • Levofloxacin • Moxifloxacin • Ceftriaxone • Cefotaxime

- If chronic or recurrent > open wall of sinus cavity by surgery

Modified Centor score [17][18][19]		
Criteria		**Points**
Age	3–14 years	+1
	15–44 years	0
	> 44 years	-1
Exudate or swelling on tonsils	Yes	+1
	No	0
Tender or swollen anterior cervical lymph nodes	Yes	+1
	No	0
Temperature > 100.4°F (38°C)	Yes	+1
	No	0
Cough	Absent	+1
	Present	0

Interpretation

- Score ≤ 1: no further diagnostic testing needed
- Score ≥ 2: Consider **rapid strep test** and/or throat culture.
- Score ≥ 4: **Consider** empiric antibiotic therapy **(controversial)** [2][10]

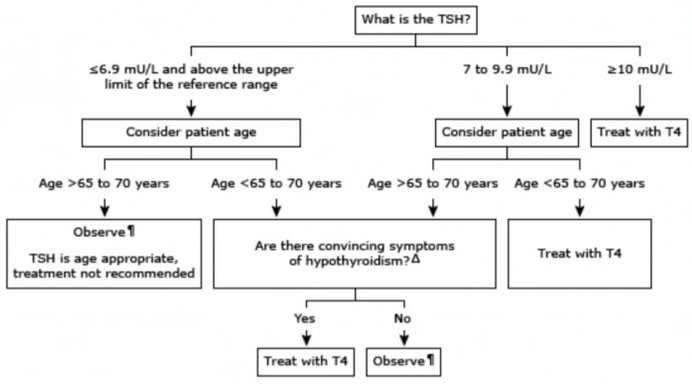
Think of **M-CENTOR** to remember the Modified Centor score criteria: **M** = Must be older than 3 years, **C** = Cough absent, **E** = Exudate on the tonsils, **N** = Node enlargement, **T** = Temperature elevation, **OR** = young OR old.

Empiric antibiotic therapy for patients with a modified Centor score ≥ 4 is **not** routinely recommended. [2]

Subclinical hypothyroidism

pregnancy
+TPO Antibodies

Indications for thyroid hormone replacement in nonpregnant adults with subclinical hypothyroidism*



TSH: thyroid-stimulating hormone; T4: levothyroxine; free T4: free thyroxine.

* Subclinical hypothyroidism is defined by a TSH above the normal reference range with a normal free T4, confirmed with repeat measurement.

¶ For patients not treated with T4, monitor TSH and free T4 initially at six months and, if stable, yearly thereafter.

Δ Convincing symptoms of hypothyroidism (eg, new or worsening fatigue, constipation, cold intolerance) or growing goiter.

Causes of subclinical hypothyroidism

Chronic autoimmune thyroiditis (risk factors: family history of autoimmune thyroid disease, personal or family history of associated autoimmune disorders, Down syndrome, Turner syndrome)
Persistent TSH increase in subacute thyroiditis, postpartum thyroiditis, painless thyroiditis
Thyroid injury – Partial thyroidectomy or other neck surgery, radioactive iodine therapy, external radiotherapy of the head and neck
Drugs impairing thyroid function – Iodine and iodine-containing medications (amiodarone, radiographic contrast agents), lithium carbonate, cytokines (especially interferon alfa), aminoglutethimide, ethionamide, sulfonamides, and sulfonylureas
Inadequate replacement therapy for overt hypothyroidism (inadequate dosage, noncompliance, drug interactions [iron, calcium carbonate, cholestyramine, dietary soy, fiber, etc.], increased T4 clearance [phenytoin, carbamazepine, phenobarbital, etc], malabsorption)
Thyroid infiltration (amyloidosis, sarcoidosis, hemochromatosis, Riedel's thyroiditis, cystinosis, AIDS, primary thyroid lymphoma)
Central hypothyroidism with impaired TSH bioactivity
Toxic substances, industrial and environmental agents
TSH receptor gene mutations; G-alpha gene mutations

TSH: thyroid-stimulating hormone; T4: thyroxine.

Hypothyroidism , hyperthyroidism differential diagnosis

Major causes of hypothyroidism

Primary hypothyroidism
Chronic autoimmune thyroiditis
Iatrogenic
Thyroidectomy
Radioiodine therapy or external irradiation
Iodine deficiency or excess
Drugs - thionamides, lithium, amiodarone, interferon alfa, interleukin-2, tyrosine kinase inhibitors, checkpoint inhibitor immunotherapy
Infiltrative diseases - fibrous thyroiditis, hemochromatosis, sarcoidosis
Transient hypothyroidism
Painless (silent, lymphocytic) thyroiditis
Subacute granulomatous thyroiditis
Postpartum thyroiditis
Subtotal thyroidectomy
Following radioiodine therapy for Graves' hyperthyroidism
Following withdrawal of suppressive doses of thyroid hormone in euthyroid patients
Congenital thyroid agenesis, dysgenesis, or defects in hormone synthesis
Central hypothyroidism
TSH deficiency
TRH deficiency
Generalized thyroid hormone resistance (some patients)*

TRH: thyrotropin-releasing hormone; TSH: thyroid-stimulating hormone.

* Refer to UpToDate content on clinical features of thyroid hormone resistance.

Causes of hyperthyroidism

Hyperthyroidism with a normal or high radioiodine uptake
Autoimmune thyroid disease
Graves' disease
Hashitoxicosis
Autonomous thyroid tissue (uptake may be low if recent iodine load led to iodine-induced hyperthyroidism)
Toxic adenoma
Toxic multinodular goiter
TSH-mediated hyperthyroidism
TSH-producing pituitary adenoma
Non-neoplastic TSH-mediated hyperthyroidism
Human chorionic gonadotropin-mediated hyperthyroidism
Hyperemesis gravidarum
Trophoblastic disease
Hyperthyroidism with a near absent radioiodine uptake
Thyroiditis
Subacute granulomatous (de Quervain's) thyroiditis
Painless thyroiditis (silent thyroiditis, lymphocytic thyroiditis)
Postpartum thyroiditis
Amiodarone (also may cause iodine-induced hyperthyroidism)
Checkpoint inhibitor-induced thyroiditis
Radiation thyroiditis
Palpation thyroiditis
Exogenous thyroid hormone intake
Excessive replacement therapy
Intentional suppressive therapy
Factitious hyperthyroidism
Ectopic hyperthyroidism
Struma ovarii
Metastatic follicular thyroid cancer

Major causes of hyperthyroidism according to the presence of a high or low radioiodine uptake. High uptake indicates increased new hormone synthesis by the thyroid, whereas low uptake indicates release of preformed hormone, exogenous ingestion, or extrathyroidal hormone synthesis.

TSH: thyroid-stimulating hormone.

Differentiation between histamine-mediated angioedema and bradykinin-mediated angioedema				
		Histamine-mediated angioedema [1][2]	Bradykinin-mediated angioedema [1][2]	
			Hereditary angioedema types I–III	Acquired angioedema
Onset		• Minutes	• Hours	
Duration		• 12–24 hours	• 24–48 hours	
Usual age		• Any	• < 20 years	• Acquired C1-INH deficiency: > 40 years • ACE inhibitor-induced: any
Family history		• Possible	• Common	• Uncommon

Cause/trigger [3][4]		• **IgE-mediated**
		  ◦ Food allergens
		  ◦ Drugs (e.g., NSAIDs and antibiotics)
		  ◦ Stinging insects
		  ◦ Latex
		• **Direct mast cell activation**
		  ◦ Drugs
		  ▪ NSAIDs
		  ▪ Opiates
		  ▪ Radiocontrast agents
		  ◦ Physical
		  ▪ Exercise
		  ▪ Temperature
		  ▪ Vibration
		  ▪ UV radiation
			• **Cause**: autosomal-dominant inheritance
			• **Trigger**
			  ◦ Estrogen fluctuation
			  ◦ Infection
			  ◦ Mental
			  ◦ Physical
				• **Acquired C1-INH deficiency**
				  ◦ Medications
				  ◦ Lymphoproliferative or autoimmune disorders
				• **ACE-inhibitor-induced**: ACE inhibitors [1]

Prodrome		• No	• Common	• Uncommon
Associated symptoms	Laryngeal edema	• Possible	• Common	
	Extremity or truncal edema			
	Gastrointestinal symptoms			
	Bronchospasm	• Common	• Uncommon	
	Urticaria			
	Hypotension			
Response to standard anaphylaxis treatment		• Good	• Poor	

Goiter workup

Evaluation of goiter in adults without obstructive symptoms

TSH: thyroid-stimulating hormone; T4: thyroxine; T3: triiodothyronine; TPO: thyroid peroxidase; TRAb: TSH-receptor antibodies.

* Non-autonomous focal areas of possible nodularity on thyroid scan (or exam) should be evaluated with ultrasound.

¶ We do not routinely obtain a thyroid ultrasound in patients with Hashimoto's thyroiditis. Ultrasound should be reserved for such patients with larger goiters, thyroid asymmetry, or a concern for thyroid nodularity.

Graphic 101377 Version 4.0

Causes of goiter in adults

Multinodular goiter
Iodine-deficiency goiter
Autoimmune/thyroiditis
Chronic autoimmune (Hashimoto's) thyroiditis
Painless thyroiditis
Subacute thyroiditis
Postpartum thyroiditis
Infectious thyroiditis
Ingestion of goitrogens
Iodine
Lithium carbonate
Foodstuffs (cassava, millet)
Thyroid infiltrative disease
Riedel's thyroiditis
Amyloid goiter
Histiocytosis
Cystinosis
Sarcoidosis
Toxic goiter
Graves' disease
Autonomously functioning thyroid adenoma
Thyroid cysts
Thyroglossal duct cysts
Thyroid adenomas
Thyroid carcinoma
Papillary carcinoma
Follicular carcinoma
Medullary carcinoma

BIRADS score

BI-RADS assessment categories

Assessment	Management	Likelihood of cancer
Category 0: Incomplete – Need additional imaging evaluation and/or prior mammograms for comparison	Recall for additional imaging and/or comparison with prior examination(s)	N/A
Category 1: Negative	Routine mammography screening	Essentially 0% likelihood of malignancy
Category 2: Benign	Routine mammography screening	Essentially 0% likelihood of malignancy
Category 3: Probably benign	Short-interval (6-month) follow-up or continued surveillance mammography	>0 but ≤2% likelihood of malignancy
Category 4: Suspicious	Tissue diagnosis*	>2 but <95% likelihood of malignancy
Category 4A: Low suspicion for malignancy		>2 to ≤10% likelihood of malignancy
Category 4B: Moderate suspicion for malignancy		>10 to ≤50% likelihood of malignancy
Category 4C: High suspicion for malignancy		>50 to <95% likelihood of malignancy
Category 5: Highly suggestive of malignancy	Tissue diagnosis*	≥95% likelihood of malignancy
Category 6: Known biopsy-proven malignancy	Surgical excision when clinically appropriate	N/A

BI-RADS: Breast Imaging-Reporting and Data System.

* Practice guidelines recommend biopsy for all BI-RADS 4 and 5 lesions. If there are clinical factors (eg, age, comorbidities, etc) for which the patient, in consultation with the clinician, chooses to defer biopsy, the reasoning should be documented in the medical record.

Jaundice

Classification of jaundice according to type of bile pigment and mechanism

Unconjugated hyperbilirubinemia
Increased bilirubin production*
Extravascular hemolysis
Extravasation of blood into tissues
Intravascular hemolysis
Dyserythropoiesis
Wilson disease
Impaired hepatic bilirubin uptake
Heart failure
Portosystemic shunts
Some patients with Gilbert syndrome
Certain drugs¶ – Rifampin, probenecid, flavaspadic acid, bunamiodyl
Impaired bilirubin conjugation
Crigler-Najjar syndrome types I and II
Gilbert syndrome
Neonates
Hyperthyroidism
Ethinyl estradiol
Liver diseases – Chronic hepatitis, advanced cirrhosis
Conjugated hyperbilirubinemia
Defect of canalicular organic anion transport
Dubin-Johnson syndrome
Defect of sinusoidal reuptake of conjugated bilirubin
Rotor syndrome

Conjugated hyperbilirubinemia (continued)
Extrahepatic cholestasis (biliary obstruction)
Choledocholithiasis
Intrinsic and extrinsic tumors (eg, cholangiocarcinoma, pancreatic cancer)
Primary sclerosing cholangitis
AIDS cholangiopathy
Acute and chronic pancreatitis
Strictures after invasive procedures
Certain parasitic infections (eg, *Ascaris lumbricoides*, liver flukes)
Intrahepatic cholestasis
Viral hepatitis
Alcohol-associated hepatitis
Non-alcohol-associated steatohepatitis
Chronic hepatitis
Primary biliary cholangitis
Drugs and toxins (eg, alkylated steroids, chlorpromazine, herbal medications [eg, Jamaican bush tea], arsenic)
Sepsis and hypoperfusion states
Infiltrative diseases (eg, amyloidosis, lymphoma, sarcoidosis, tuberculosis)
Total parenteral nutrition
Postoperative cholestasis
Following organ transplantation
Hepatic crisis in sickle cell disease
Pregnancy
End-stage liver disease

AIDS: acquired immunodeficiency syndrome.

* Serum bilirubin concentration is usually less than 4 mg/dL (68 mmol/L) in the absence of underlying liver disease.

¶ The hyperbilirubinemia induced by drugs usually resolves within 48 hours after the drug is discontinued.

Hepatitis B serology

Interpretation of the hepatitis B serologic panel

Tests	Results	Interpretation
HBsAg	Negative	Susceptible
anti-HBc	Negative	
anti-HBs	Negative	
HBsAg	Negative	Prior infection (inactive)
anti-HBc	Positive	
anti-HBs	Positive	
HBsAg	Negative	Immune due to hepatitis B vaccination*
anti-HBc	Negative	
anti-HBs	Positive	
HBsAg	Positive	Acutely infected
anti-HBc	Positive	
IgM anti-HBc	Positive	
anti-HBs	Negative	
HBsAg	Positive	Chronically infected
anti-HBc	Positive	
IgM anti-HBc	Negative	
anti-HBs	Negative	
HBsAg	Negative	Four interpretations possible¶
anti-HBc	Positive	
anti-HBs	Negative	

HBsAg: hepatitis B surface antigen; anti-HBc: hepatitis B core antibody; anti-HBs: hepatitis B surface antibody; IgM: immunoglobulin M; HBV: hepatitis B virus.

* Antibody response (anti-HBs) can be measured quantitatively or qualitatively. A protective antibody response is reported quantitatively as 10 or more milli-international units (≥10 mIU/mL) or qualitatively as positive. Postvaccination testing should be completed one to two months after the third vaccine dose for results to be meaningful.

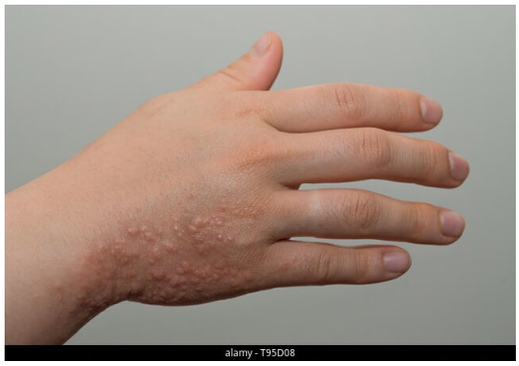
¶ Four interpretations:

1. Might be recovering from acute HBV infection.
2. Might have had prior infection and test not sensitive enough to detect very low level of anti-HBs in serum.
3. Might be susceptible with a false positive anti-HBc.
4. Might be undetectable level of HBsAg present in the serum, and the person is actually chronically infected.

Centers for Disease Control and Prevention, Hepatitis B information for health professionals: Interpretation of hepatitis B serologic test results. Available from the CDC website.

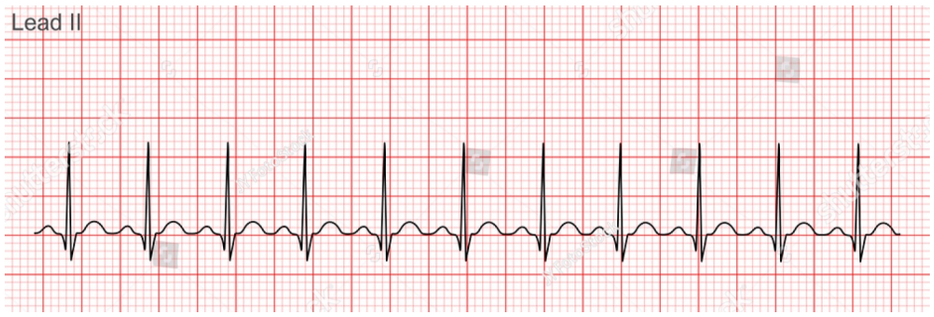
MiniOSCE questions

1- What is the diagnosis ? and what is the treatment ?



Shingles (follows specific dermatomes) , Acyclovir

2- follow the ECG below for 23 old female patient :



-What is the diagnosis?
Sinus tachycardia

-Order 3 labs .
CBC
Ferritin
TSH

-What will you ask her in the history ?
Chest pain , symptoms of hyperthyroidism, palpitations, caffeine intake

3- Male patients has a history of testicular cancer + give a chest - X-ray picture with changes ,what is the diagnosis ?
Lung metastasis

4-23 years female patient complains from pubic pain since **5 days** , give 3 differential diagnosis .
Pregnancy, cyclical pain , UTI

5-4-23 years female patient complains from pubic pain since **4 weeks** , give 3 differential diagnosis .
Pregnancy , recurrent ovarian cyst , pelvic adhesion disease

6-Picture of kidney , then Ask about the changes in this system in geriatric patients

Arrhythmias

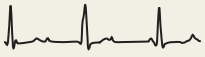
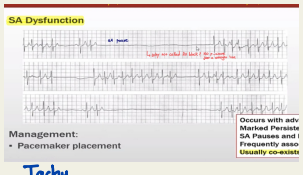
MNEMONIC

Management options for atrial fibrillation—

ABCD

Anticoagulate
β-blockers to control rate
Cardiovert/**C**alcium channel blockers
Digoxin (in refractory cases)

TABLE 2.1-3. Bradyarrhythmias and Conduction Abnormalities

Type	Etiology	Signs/Symptoms	ECG Findings	Treatment
Sinus bradycardia acceptable in athletes	Normal response to cardiovascular conditioning Can also result from sinus node dysfunction, β-blocker or CCB excess; therefore, it is important to review medications	May be asymptomatic, but may also present with lightheadedness, syncope, chest pain, or hypotension 	Sinus rhythm Ventricular rate < 60 bpm narrow QRS Regular Slow HR Normal PR interval (3-5 small Boxes)	None if asymptomatic and rate > 40 bpm; atropine may be used to ↑ heart rate symptomatic: 1. 2. B. Agonist: Dopamine isoproterenol Pacemaker implant is the definitive treatment in severe cases
First-degree AV block	Can occur in normal individuals; associated with ↑ vagal tone, β-blocker or CCB use	Asymptomatic 	PR interval > 200 msec 0.2 sec = > 1 Large Square No Dropped QRS	Same as Sinus Bradycardia.
Second-degree AV block (Mobitz type I/ Wenckebach)	Drug effects (digoxin, β-blockers, CCBs) or ↑ vagal tone; right coronary ischemia or infarction	Usually asymptomatic 	Progressive PR lengthening until a dropped beat occurs; the PR interval then resets	Same None if asymptomatic Stop the offending drug Atropine as clinically indicated
Second-degree AV block (Mobitz type II)	Results from fibrotic disease of the conduction system or from acute, subacute, or prior MI	Occasionally syncope; frequent progression to third-degree AV block 	Unexpected dropped beat(s) without a change in PR interval Fixed Prolonged PR interval	Pacemaker placement (even if asymptomatic)
Third-degree AV block (complete)	No electrical communication between the atria and ventricles	Syncope, dizziness, acute heart failure, hypotension, cannon A waves 	P > QRS No relationship between P waves and QRS complexes Complete Dissociation QRS < Narrow → if from the Junction/ bundle of His Wide → if from the Ventricle	Pacemaker placement Medical Emergency
Sick sinus syndrome/ tachycardia-bradycardia syndrome	Heterogeneous disorder that leads to intermittent supraventricular tachyarrhythmias and bradyarrhythmias SVT in advanced Age	2° to tachycardia or bradycardia; AF and thromboembolism may occur → syncope, palpitations, dyspnea, chest pain, TIA, and/or stroke	 Tachy Brady Pause	Most common indication for pacemaker placement Anticoagulate in atrial fibrillation/flutter to prevent systemic emboli

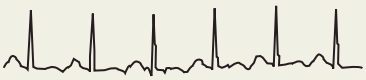
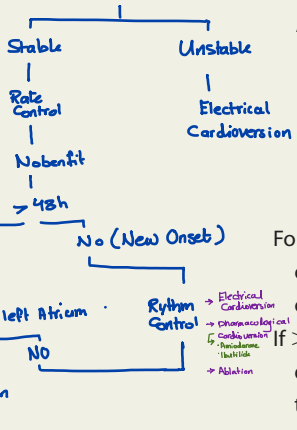
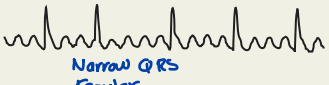
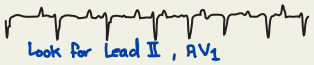
KEY FACT

Patients with persistent tachyarrhythmia (narrow- or wide-complex) causing hemodynamic instability should be managed with immediate synchronized cardioversion.

TACHYARRHYTHMIAS

Tables 2.1-4 and 2.1-5 outline the etiologies, clinical presentation, and treatment of common supraventricular and ventricular tachyarrhythmias.

TABLE 2.1-4. Supraventricular Tachyarrhythmias

Type	Etiology	Signs/Symptoms	ECG Findings	Treatment
		Atrial		
Sinus tachycardia	Normal physiologic response to fear, pain, and exercise Can also be 2° to hyperthyroidism, volume contraction, infection, or PE	Palpitations, shortness of breath  Narrow QRS Regular Tachy >100 ↙ P wave	Sinus rhythm Ventricular rate > 100 bpm	Treat the underlying cause β Blocker Ivabradine Ablation (End Game)
Atrial fibrillation (AF) Paroxysmal <48h Persistent >7 days Long Persistent >1 Year Permanent Forever	Acute AF—**PIRATES:** **P**ulmonary disease **I**schemia **R**heumatic heart disease **A**nemia/**A**trial myxoma **T**hyrotoxicosis or Hypothyroid **E**thanol Holiday Heart Syndrome **S**epsis Chronic AF—HTN, CHF Most often caused by ectopic foci within the pulmonary veins ← Re-entry	Often asymptomatic and incidental but can present with shortness of breath, chest pain, dizziness, fatigue, or palpitations. May present with congestive heart failure, cardiogenic shock, or devastating cerebrovascular accident Physical exam reveals an irregular pulse  Narrow QRS No P wave Irregular	No discernible P waves, with variable and irregular QRS response (Narrow)  Stable → Rate Control → Nebenfit → >48h → YES → TEE → thrombus in left Atrium → YES → Anticoagulation 3 weeks; NO → Rhythm Control (Electrical Cardioversion; Pharmacological; Cardioversion + Amiodarone + Flecainide; Ablation) Unstable → Electrical Cardioversion No (New Onset)	For chronic AF, initial therapy: Rate control with β-blockers (↓BP), CCBs, or digoxin (BP not affected → Good for Pt with Low BP) Anticoagulate with warfarin or novel oral anticoagulant (NOAC) for patients with CHA_2DS_2-VASc score ≥ 2 (0 – Platelet Only; 1 – female; 1 – for other Cause) AF/b w/: mechanical Valve; Severe mitral Stenosis; LVAD [Warfarin is a must] For unstable AF, or new-onset AF (of < 2 days) cardiovert If > 2 days or unclear duration, must get TEE to rule out atrial clot
Atrial flutter easy for Ablation	Circular movement of electrical activity around the atrium (macro circuit) at a rate of approximately 300 times per minute	Usually asymptomatic but can present with palpitations, syncope, and lightheadedness  Narrow QRS Regular No P wave	Regular rhythm; "sawtooth" appearance of P waves can be seen (Rarely.) The atrial rate is usually 240–320 bpm, and the ventricular rate is ~150 bpm	Anticoagulation, rate control, and cardioversion guidelines as in AF above Ablation is Favorable Here
Multifocal atrial tachycardia	Multiple atrial pacemakers or reentrant pathways; associated with COPD,* hypoxemia	May be asymptomatic. At least three different P-wave morphologies  Look for Lead II, AV_1	Three or more unique P-wave morphologies; rate > 100 bpm Irregular, Narrow QRS] like AF But there is P wave	Treat as AF ~~but avoid β-blockers because of chronic lung disease (if present)~~ Oxygenation (Best one) → Avoid electrical Cardioversion even if Unstable

(continues)

TABLE 2.1-4. Supraventricular Tachyarrhythmias *(continued)*

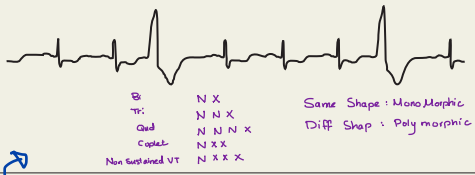
Type	Etiology	Signs/Symptoms	ECG Findings	Treatment
Atrioventricular Junction				
Atrioventricular nodal reentry tachycardia (AVNRT)	A reentry circuit in the AV node depolarizes the atrium and ventricle nearly simultaneously	Palpitations, shortness of breath, angina, syncope, lightheadedness	Rate 150–250 bpm; P wave is often buried in QRS or shortly after	Cardiovert if hemodynamically unstable. Vagal maneuvers (eg, carotid massage, Valsalva, ice immersion (dive reflex). IV Adenosine if vagal maneuver fails
Atrioventricular reentrant tachycardia (AVRT)	An ectopic connection between the atrium and ventricle that causes a reentry circuit Seen in WPW	Palpitations, shortness of breath, angina, syncope, lightheadedness	A retrograde P wave is often seen after a normal QRS	Except for WPW, same as that for AVNRT WPW listed separately below
Wolff-Parkinson-White (WPW) syndrome	Abnormal fast accessory conduction pathway from atria to ventricle (Bundle of Kent)	Palpitations, dyspnea, dizziness, and rarely cardiac death	Characteristic delta wave with widened QRS complex and shortened PR interval (see Figure 2.1-8)	Observation for asymptomatics Acute therapy is procainamide or amiodarone SVT gets worse after CCBs, BB or digoxin (dangerous in WPW). Radiofrequency catheter ablation is curative
Paroxysmal atrial tachycardia	Rapid ectopic pacemaker in the atrium (not sinus node)	Palpitations, shortness of breath, angina, syncope, lightheadedness	Rate > 100 bpm; P wave with an unusual axis before each normal QRS	Adenosine can be used to unmask underlying atrial activity by slowing down the rate

KEY FACT

Use the **CHA_2DS_2-VASc** scoring system to estimate stroke risk in atrial fibrillation, and anticoagulate with NOAC (eg, dabigatran, rivaroxaban, apixaban, and edoxaban) or warfarin (used with metal valves or mitral stenosis) for a score of 2 or more:

- **C**HF (1 point).
- **H**TN (1 point).
- **A**ge ≥ 75 (2 points).
- **D**iabetes (1 point).
- **S**troke or TIA history (2 points).
- **V**ascular disease (1 point).
- **A**ge 65–74 (1 point).
- **S**ex **c**ategory (female) (1 point).

TABLE 2.1-5. Ventricular Tachyarrhythmias

Type	Etiology	Signs/Symptoms	ECG Findings	Treatment
Premature ventricular contraction (PVC)	Ectopic beats arise from ventricular foci. Associated with hypoxia, fibrosis, ↓ LV function, electrolyte abnormalities, and hyperthyroidism	Usually asymptomatic but may lead to palpitations 	Early, wide QRS not preceded by a P wave PVCs are usually followed by a compensatory pause	Treat the underlying cause If symptomatic, give β-blockers or, occasionally, other antiarrhythmics
Ventricular tachycardia (VT)	Can be associated with CAD, MI, and structural heart disease	Nonsustained VT (lasts < 30 seconds) is often asymptomatic; sustained VT (lasts > 30 seconds) can lead to palpitations, hypotension, angina, and syncope Can progress to VF and death 	Three or more consecutive PVCs; wide QRS complexes in a regular rapid rhythm; may see AV dissociation	Cardioversion if unstable. Antiarrhythmics (eg, amiodarone, lidocaine, procainamide) if stable
Ventricular fibrillation (VF)	Associated with CAD and structural heart disease Also associated with cardiac arrest (together with asystole)	Syncope, absence of BP, no pulse 	Totally erratic wide-complex tracing	Immediate electrical defibrillation and ACLS protocol
Torsades de pointes	Associated with long QT syndrome, proarrhythmic response to medications, hypokalemia, congenital deafness, and alcoholism	Can present with sudden cardiac death; typically associated with palpitations, dizziness, and syncope 	Polymorphous QRS; VT with rates between 150 and 250 bpm	Give magnesium initially and cardiovert if unstable Correct hypokalemia; withdraw offending drugs

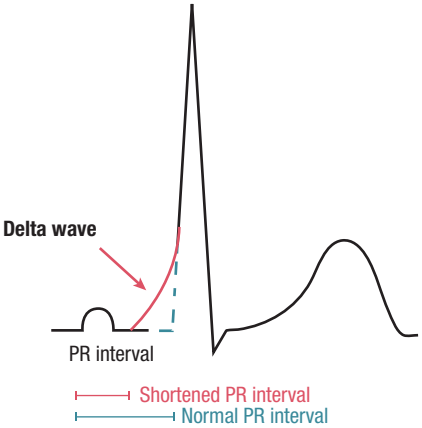
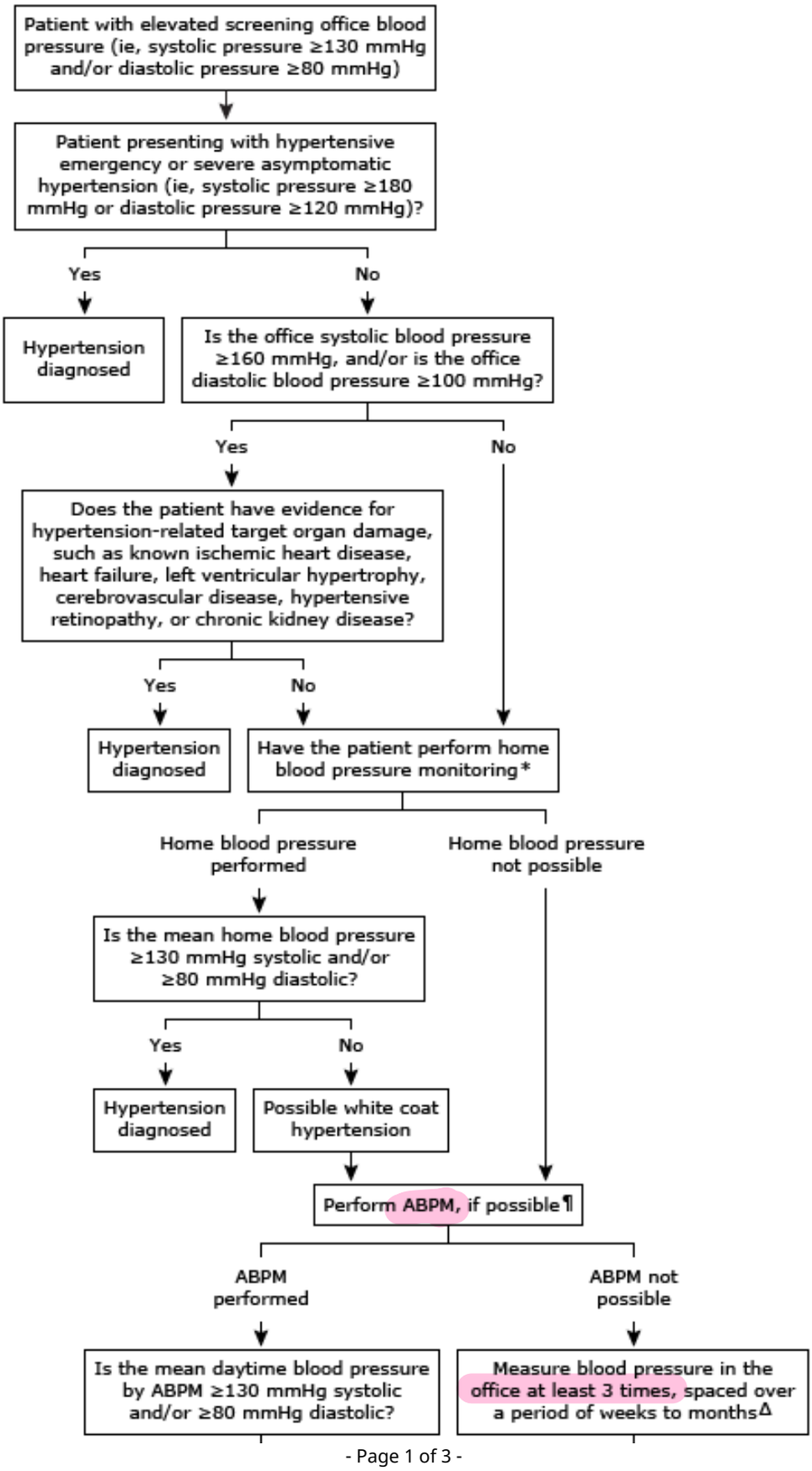
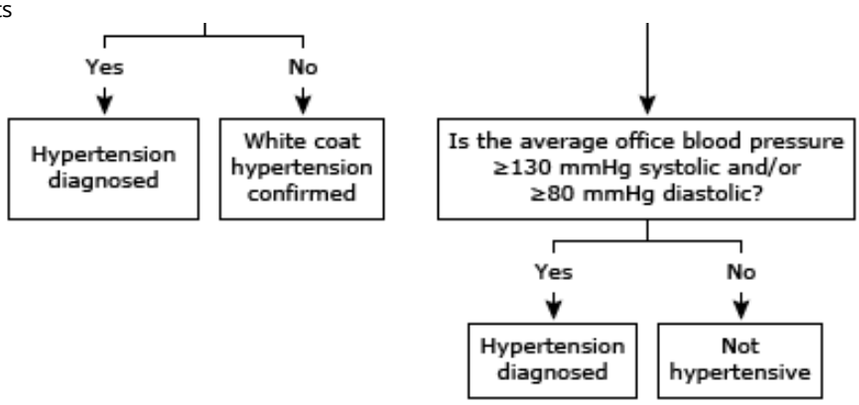


FIGURE 2.1-8. Ventricular tachyarrhythmias. Characteristic delta wave with widened QRS complex and shortened PR interval in WPW. (Reproduced with permission from USMLE-Rx.com.)

Diagnosis of hypertension in adults





ABPM: ambulatory blood pressure monitoring; AOBPM: automated office blood pressure monitoring.

* Home blood pressure must be performed adequately in order for the measurements to be used for diagnosis and management. To be adequate: The accuracy of the home device should be verified in the clinician's office; the patient should measure their blood pressure while seated (with feet flat on the floor), with arm supported (such as on a table), and after several minutes of rest; the blood pressure should be measured at different times per day and over a series of multiple days. A common strategy is to have the patient measure their blood pressure twice daily (once in the morning and once in the evening) for 7 days. Readings from the first day are discarded, and the remaining 12 measurements are averaged. Home blood pressure should not be used for diagnosis and management if it cannot be performed adequately. Adequate home blood pressure should be possible in most cases. Inexpensive devices to measure blood pressure at home are available over the counter. Alternatively, such devices can be borrowed (eg, provided by the clinic). Only rarely are such devices unavailable or unaffordable.

¶ ABPM is performed by having the patient wear, typically for 24 hours, an electronic blood pressure device that automatically measures the blood pressure, usually every half-hour during the day and hourly at night. We use the mean daytime value to determine the presence of hypertension. ABPM is possible if it is available in the clinic or via an external vendor and if it can be paid for by the patient's insurance or by the patient.

Δ Blood pressure measured in the office may vary according to the manner in which it is obtained. If blood pressure in the office is to be used for the diagnosis of hypertension (rather than using out-of-office blood pressures), we suggest performing unattended AOBPM (using a device that can average multiple readings while the patient sits alone in a room). Unattended AOBPM may provide a

measurement that is 5 to 10 mmHg less than a manual measurement (ie, with a stethoscope). Office blood pressure must be performed with proper technique (eg, patient given time to rest, seated with feet flat on the floor, use of multiple measurements, appropriate-sized cuff placed on bare arm, etc). Office blood pressure measured with improper technique **should not** be used for diagnosis and management of hypertension. Refer to UpToDate topics on measurement of blood pressure for details of proper technique.

Graphic 105050 Version 7.0

TABLE 2.1-18. Lipid-Lowering Agents

CLASS	EXAMPLES	EFFECT ON LIPID PROFILE	ADVERSE EFFECTS
HMG-CoA reductase inhibitors (statins)	Atorvastatin, simvastatin, lovastatin, pravastatin, rosuvastatin	↓ LDL, ↓ triglycerides	↑ LFTs, myositis, warfarin potentiation
Lipoprotein lipase stimulators (fibrates)	Gemfibrozil	↓ triglycerides, ↑ HDL	GI upset, cholelithiasis, myositis (especially in combination with statins), ↑ LFTs, pancreatitis
Cholesterol absorption inhibitors	Ezetimibe	↓ LDL	Diarrhea, abdominal pain; can cause angioedema
Niacin	Niaspan	↑ HDL, ↓ LDL	Skin flushing (can be prevented with ASA, due to ↑ prostaglandins), paresthesias, pruritus, GI upset, ↑ LFTs
Bile acid resins	Cholestyramine, colestipol, colesevelam	↓ LDL	Constipation, GI upset, LFT abnormalities, myalgias; can ↓ absorption of other drugs from the small intestine
Proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors	Evolocumab, alirocumab (injectable medications taken every 2–4 weeks)	↓↓ LDL	Injection-site swelling, rash, muscle/limb pain, backache

- If ASCVD risk ≥7.5% after initial treatment, statins may be considered.
- **Prevention of pancreatitis:** Patients with severe hypertriglyceridemia (especially triglycerides ≥1000 mg/dL) benefit from fibrate therapy to prevent pancreatitis.

HYPERTENSION

HTN is the most common disease in the United States and the key risk factor in MI and stroke. Stage 1 HTN is defined as an SBP ≥130 to 139 mm Hg or a diastolic blood pressure (DBP) ≥80 to 89 mm Hg. Measurements are based on an average of ≥2 readings obtained on ≥2 occasions separated in time in adults (see Table 2.1-19 for classifications). HTN is classified as primary or essential without an identifiable cause and secondary when an identifiable cause exists.

at least one week between them

PRIMARY (ESSENTIAL) HYPERTENSION

Hypertension that has no identifiable cause and represents ~95% of cases.

Risk factors:

- Nonmodifiable: Increasing age, male sex, Black race, family history.
- Modifiable: High-salt diet, alcohol (amount varies), obesity, sedentary lifestyle.

KEY FACT

PCSK9 inhibitors are a new class of LDL-lowering drugs. They significantly increase hepatic clearance of LDL. Indicated in familial hypercholesterolemia and statin-resistant or statin-intolerant patients with severe hyperlipidemia.

TABLE 2.1-19. Definitions of Blood Pressure Values for Adults (>18 years) and General Practice Guidelines

STAGE	BP (MM HG)	GENERAL PRACTICE GUIDELINES
Normotensive	SBP <120 and DBP <80	Routine follow-up and continued promotion of a healthy lifestyle
Prehypertensive	SBP 120–129 and DBP <80	Lifestyle modifications recommended with routine follow-up[a]
Stage I hypertension	SBP ≥130–139 or DBP ≥80–89	Lifestyle modifications recommended to all patients[b] Lifestyle modifications + medication(s) for high-risk patients[c]
Stage II hypertension	SBP ≥140 or DBP ≥90	Lifestyle modifications + medication(s) for all patients
Severe hypertension	SBP >180 or DBP >120 (no generally agreed upon BP values)	Severe hypertension + no end-organ damage = hypertensive urgency Severe hypertension + end-organ damage = hypertensive emergency Treatment goal for both: 25% reduction in BP from baseline or <160/100 mm Hg (see associated Key Fact for more details)

[a]Lifestyle modifications (listed in order of effectiveness): Weight loss > DASH diet > Exercise > Restricting salt intake > alcohol limitation.

[b]Lifestyle modifications alone are usually given a trial period of 3–6 months for lower-risk patients before medications are considered.

[c]High-risk patients: Heart failure, coronary artery disease, chronic kidney disease, diabetes, age >65 (debated), ASCVD 10-year risk >10%.

Note: Medication follow-up: Start new medication (1 month) → medication working (3–6 months). If medication not working → Follow-up 1 month OR change dose ± medication.

Isolated systolic hypertension [17][18]

- **Definition**: elevated SBP (≥ 140 mm Hg) with DBP within normal limits (≤ 90 mm Hg)
- **Etiology** [17]
  - Most common: decreased arterial elasticity and compliance due to aging
  - May also be secondary to increased cardiac output due to:
    - Anemia
    - Hyperthyroidism
    - Chronic aortic regurgitation
    - AV fistula
- **Clinical features**
  - Often asymptomatic
  - Signs of increased pulse pressure: e.g., head pounding, rhythmic nodding, bobbing of the head in synchrony with the heartbeat
  - Symptoms of hypertension
- **Diagnostics**
  - Assess for secondary causes.
  - See "Diagnosis of hypertension" for details on diagnostic testing.
- **Treatment**
  - Recommend lifestyle changes for managing hypertension.
  - Start pharmacological treatment of hypertension.
    - First-line medication: thiazide diuretics or dihydropyridine calcium antagonists [18]
    - Treatment goal: SBP < 140 mm Hg

Patients with isolated systolic hypertension have a high risk of renal dysfunction and cardiovascular events, e.g., myocardial infarction, stroke.

Patients with masked hypertension have a similar risk of stroke, cardiovascular disease, and all-cause mortality to those with sustained hypertension

Resistant hypertension: hypertension that remains uncontrolled (≥ 130/80 mm Hg) despite treatment with ≥ 3 antihypertensives OR requires ≥ 4 medications to be controlled

History/PE/Complications

- Majority of patients are asymptomatic ("silent killer") and found on routine screening.
- Symptomatic patients exhibit end-organ damage as HTN ↑ atherosclerosis + ↑ arteriosclerosis:
  - **Eyes:** Early (AV nicking, cotton wool spots) → later (hemorrhage, exudates, papilledema). See Figure 2.1-22.
  - **Central nervous system (CNS):** Encephalopathy, stroke (intracerebral hemorrhage, lacunar, ischemic, TIAs).
  - **Cardiovascular:** CAD → MI, LV hypertrophy → CHF, peripheral arterial disease (PAD), aortic aneurysms/dissections.
  - **Kidney:** Arteriosclerosis of glomerulus → nephrosclerosis, ↓ GFR/dysfunctional tubules → failure. See Figure 2.1-23.

Interpretation of blood pressure readings [1][16]		
	In-office blood pressure	Out-of-office blood pressure
Sustained hypertension	• Elevated	• Elevated
White coat hypertension (isolated clinic hypertension)	• Elevated	• Normal
Masked hypertension (isolated ambulatory hypertension)	• Normal	• Elevated

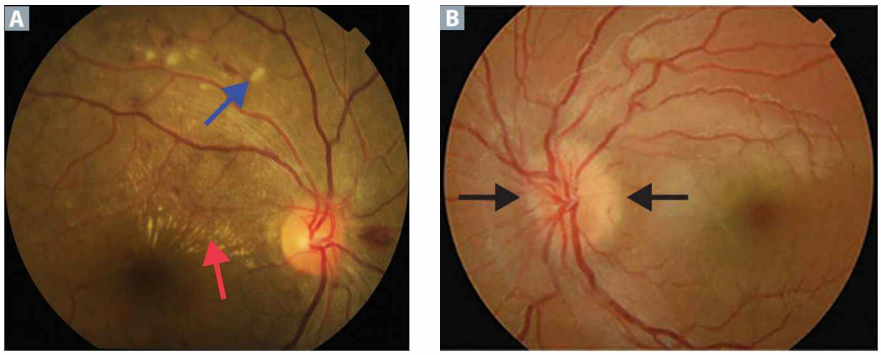


FIGURE 2.1-22. Hypertensive retinopathy. **(A)** Cotton wool spots *(blue arow)* and hard exudates *(red arrow)*. **(B)** Papilledema *(black arrows)*. (Adapted with permission from Diallo JW, Méda N, Tougouma SJ, et al. Intérêts de l'examen du fond d'œil en pratique de ville: bilan de 438 cas [Interests of the examination of the fundus in general practice: review of 438 cases]. *Pan Afr Med*. 2015;20. doi:10.11604/pamj.2015.20.363.6629. B: Adapted with permission Kanonidou E, Chatziralli I, Kanonidou C, Parava M, Ziakas N. Unilateral optic disc edema in a paediatric patient: Diagnostic dilemmas and management. Case Rep Med. 2010;2010:529081. doi:10.1155/2010/529081.).

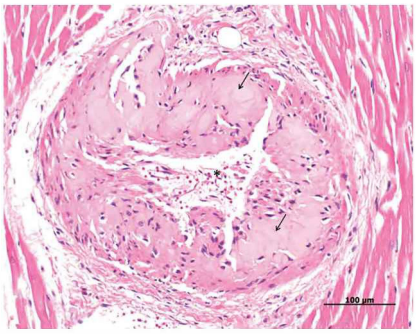
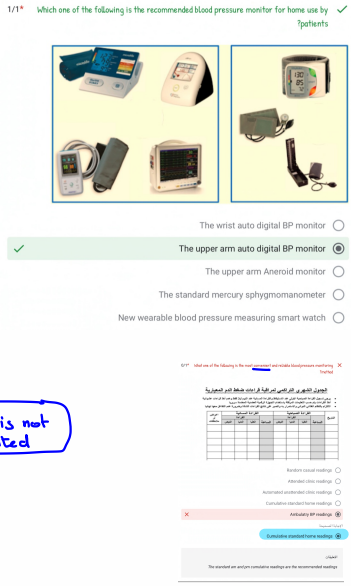


FIGURE 2.1-23. Hyaline arteriolosclerosis occurs secondary to plasma protein leakage into the endothelium due to essential hypertension or diabetes mellitus. (Reproduced with permission from Sostaric-Zuckermann IC, Borel N, Kaiser C, et al. Chlamydia in canine or feline coronary arteriosclerotic lesions. *BMC Res Notes*. 2011;4:350. doi: 10.1186/1756-0500-4-350.)

Diagnosis

- **Measurement:** Never diagnose HTN on one reading unless severe HTN or end-organ damage is present. Measurements are based on an average of ≥2 readings obtained on ≥2 occasions separated in time (days, weeks) in adults. **Ambulatory BP monitoring is the gold standard.** Ambulatory BP and outpatient BP (with home BP monitors) monitoring help exclude white coat hypertension (elevated BP only in clinic) as a cause of HTN. Serial clinic BP checks can also be used.
- **New-onset HTN:** Once primary HTN is diagnosed, the next best step is to **screen for complications and comorbid conditions**, which include HbA1c or fasting glucose, lipid panel, chemistry panel (serum Cr, blood urea nitrogen [BUN], K), ECG (screen for LV hypertrophy, Q waves for previous MI), and a urinalysis (protein).
- **Secondary cause of HTN:** If suspected, order appropriate tests (see secondary HTN key fact).



Treatment

- **Best initial treatment:** Lifestyle modifications listed in order of effectiveness: **Weight loss** (in overweight people) > **DASH diet** > exercise > restricting salt intake > alcohol limitation (if patient has refractory HTN, limiting alcohol may be the answer). Depending on patient, usually tried for 3 to 6 months before drugs are considered.
- **Best initial medications:** Choice of thiazide diuretics (usually), ACEs/ARBs, or dihydropyridine CCBs unless a compelling indication exists (Tables 2.1-20 and 2.1-21).
- **General management goals:** BP should be lowered to <130/80 mm Hg in most patients with hypertension as tolerated. Although 70% of patients are controlled with one drug, 90% are controlled with two to three drugs. For example, if a thiazide diuretic alone doesn't control HTN, consider adding an ACEI/ARB, a CCB, or a β-blocker (depending on the patient/compelling indications).

MNEMONIC

Initial Drug Treatment of HTN—
TAC

T (Thiazide diuretics usually initial choice)
ACEIs/ARBs
CCBs

Q

A patient is diagnosed with new-onset primary HTN. The physician recommends lifestyle modifications with a focus on weight loss, with follow-up scheduled 3 months later to evaluate the need for medications. What is the next best step in evaluating this patient?

TABLE 2.1-20. Compelling Indications for Treatment of Primary Hypertension

IF HISTORY OF...	...THEN BEST INITIAL TREATMENT(S)
Prior myocardial infarction, coronary artery disease, compensated heart failure, atrial fibrillation, hyperthyroidism	β-blockers are a high-yield answer for exams + practice (others may include CCBs, ACE/ARBs, diuretics, and aldosterone antagonists, *depending on condition*)
CKD, proteinuria, diabetes	ACEIs or ARBs (renal protective in diabetics)
Benign prostatic hyperplasia	α-Blockers → smooth muscle relaxation of blood vessels (vasodilation) + bladder/prostrate
Osteoporosis Black race[a]	Thiazides → blocks Na-Cl reabsorption in DCT →↑ calcium reabsorption
Current pregnancy	"**H**e **L**ikes **M**y **N**eonate" **H**ydralazine, **L**abetalol (use first), **M**ethyldopa, **N**ifedipine
Asthma	ARBs (NOT ACEIs because bradykinin → cough), CCBs, thiazide diuretics, cardioselective (β_1) β-blockers

[a]Controversial "salt-sensitive HTN" in Black individuals. CCBs are equally first line.

TABLE 2.1-21. Major Classes of Antihypertensive Agents, Mechanism of Actions, and Adverse Effects

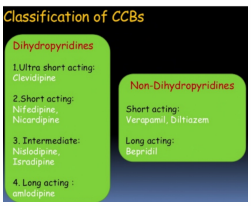
CLASS	AGENTS	MECHANISM OF ACTION	ADVERSE EFFECTS
Thiazide diuretics	Hydrochloro**thiazide,** chlorthalidone, metolazone. "Thiazides get it **done** = chlorthali**done**." "Get in the **zone** with a thiazide = metola**zone**."	Thiazides → block Na^+/Cl^- reabsorption in DCT → Na^+/H_2O excretion → ↓ BP.	Elevate blood levels (hyper**GLUC**) of **G**lucose, **L**ipids, **U**ric acid, **C**alcium. So, caution use in diabetes, ↑ TGs, ↑ uric acid → gout, hypercalcemia. Causes metabolic alkalosis, ↓ K+, can lead to hyponatremia (promotes Na^+ loss with no change to medullary gradients' osmolarity that drive H_2O reabsorption → if patient ↑ H_2O intake → hyponatremia).
Loop diuretics	Sulfonamides: furosemide, torsemide, bumetanide. Non-sulfonamide: ethacrynic acid.	**Loops** → block $Na^+/K^+/2Cl^-$ pump in thick ascending **loop** of Henle → ↓ medullary osmotic gradients → ↑↑↑ Na^+/ H_2O excretion → ↓ BP.	Metabolic alkalosis, ↓ K^+, ↓ Ca^{2+}, ↓ Mg^{2+}, ↑ uric acid → gout, ototoxicity (all + ↑ ↑ risk ethacrynic acid). "Loop earrings hurt your ears." Sulfonamides → rash + acute interstitial nephritis.

(continues)

A

Once primary HTN is diagnosed, the next best step is to screen for complications and comorbid conditions, which include HbA1c or fasting glucose, lipid panel, chemistry panel (serum Cr, BUN, K), ECG (screen for LV hypertrophy, Q waves for previous MI), and a urinalysis (protein).

TABLE 2.1-21. Major Classes of Antihypertensive Agents, Mechanism of Actions, and Adverse Effects (continued)

CLASS	AGENTS	MECHANISM OF ACTION	ADVERSE EFFECTS
K^+-sparing diuretics	**S**pironolactone, **T**riamterene, **E**plerenone, **A**miloride, **K**$^+$-sparing = **STEAK**.	Spironolact**one** + eplerenone → aldoster**one**-R inhibitors. Triamterene + amiloride → inhibit epithelial Na^+ channels.	Hyperkalemia + metabolic acidosis. Spironolactone mimics/blocks testosterone + progesterone effects → gynecomastia (men) + amenorrhea (women).
β-blockers	Cardioselective (β_1- > β_2-blockers) are mostly in ***first*** part of alphabet **(A–M)**: **A**tenolol, **A**cebutolol (partial agonist), **B**etaxolol, **B**isoprolol, **E**smolol, **M**etoprolol ("**ABEAM**"). **N**on-**Z**elective ($\beta_1 = \beta_2$ blockers) are mostly in ***second*** part of alphabet **(N–Z)**: **N**adolol, **P**indolol (partial agonist), **P**ropranolol, **T**imolol. Nonselective β- and α-blockers ($\beta_1 = \beta_2 \geq \alpha_1 > \alpha_2$) have a modified suffix (instead of "-olol"): carved**ilol**, labet**alol**.	Block β_1 on heart + kidney → ↓ HR, cardiac contractility, renin release → ↓ effective circulating volume, CO → ↓ BP. Block β_2 on lungs + liver → bronchoconstriction + ↓ portal blood flow (useful to treat portal hypertension in cirrhosis patients).	Caution in obstructive lung diseases (nonselective agents → bronchospasm), decompensated HF (↓ mortality in compensated HF), heart block (block β_1 → bradycardia), diabetes (commonly used but can mask hypoglycemia), depression. Sleep disturbances (insomnia), fatigue, erectile dysfunction, ↓ HDL, ↑ TGs. Overdose treatment: glucagon.
ACEIs + ARBs + related agents	ACEIs: end in **pril** (lisino**pril**, capto**pril**). Please celebrate A**pril** as national ACEI month. ARBs: end in **sartan** (losartan, valsartan). Entresto = valsartan/sacubitril (inhibits neprilysin). Aliskiren = direct renin inhibitor.	ACEI: inhibit angiotensin I → angiotensin II conversion = vasodilation + ↓ aldosterone → ↑ Na^+/H_2O excretion → ↓ BP. ARBs: inhibit angiotensin II receptor → same as earlier. Neprilysin inhibition → ↑ angiotensin II (pair w/ ARB) + ↑ natriuretic peptides (A/BNP). Aliskiren inhibits renin → ↓ angiotensinogen conversion → angiotensin I → ↓ angiotensin II + aldosterone → diuresis + ↓ BP.	ACEI → ↑ bradykinin → cough + angioedema. ARBs have less risk. ACEI, ARBs, Aliskiren → ↓ GFR (acute renal failure) as efferent arteriole vasoconstriction controlled by angiotensin II. ↓ aldosterone → ↑ K^+. Renal teratogens, cause rash. *Entresto's main side effects = ARBs.
CCBs 	Dihydropyridine CCBs (amlod**ipine**, nifed**ipine**) = more selective for vascular smooth muscle. Nondihydropyridine CCBs (diltiazem, verapamil) = more selective for cardiac muscle.	Block voltage-dependent L-type Ca^{2+} channels in cardiac (↓ HR, contractility) + vascular smooth muscle (↓ BP) to varying degrees.	Gingival hyperplasia. Dihydropyridines → vasodilation → headache, flushing, peripheral edema, reflex tachycardia (may coadminister β-blocker). Nondihydropyridines: ⊖ inotropes → precipitate HF, AV block, ↓ HR/contractility, verapamil (constipation, ↑ prolactin).

(continues)

TABLE 2.1-21. Major Classes of Antihypertensive Agents, Mechanism of Actions, and Adverse Effects (continued)

CLASS	AGENTS	MECHANISM OF ACTION	ADVERSE EFFECTS
α_1-Antagonists	Common **-osin** suffix: praz**osin**, doxaz**osin**, teraz**osin**, alfuz**osin**, tamsul**osin**.	Block α_1 → inhibits smooth muscle contraction in vasculature (↓ BP) + bladder/prostate (↑ urine flow).	Although any anti-HTN agent can cause postural hypotension, these agents are notorious. Headache, dizziness, reflex tachycardia (may coadminister β-blocker). Tamsulosin = uroselective = less ↓ BP.
α_2-Agonists	Methyldopa (a drug of choice in pregnancy), clonidine.	Stimulate presynaptic CNS α_2 receptors → ⊖ feedback → ↓ norepinephrine → ↓ BP.	Clonidine → ↓ sympathetic response → somnolence, ↓ HR, ↓ respirations, miosis. Dry mouth and severe rebound HTN with sudden dose stoppage → ↑↑↑ sympathetic response. Methyldopa: direct Coombs ⊕ warm autoimmune hemolytic anemia, sedation, drug-induced lupus ⊕), anti-histone Abs, hyperprolactinemia.
Vasodilators	Hydralazine (a drug of choice in pregnancy), minoxidil.	Hydralazine → ↑ NO → ↑ cGMP → arteriolar vasodilation (↓ BP). Minoxidil → opens K^+ channels → vasodilation (↓ BP).	Hydralazine: drug-induced lupus ⊕ anti-histone Abs, fluid retention, reflex tachycardia (may coadminister β-blocker). Minoxidil = rogaine = hypertrichosis.
Hypertensive emergency agents	β-blockers (labetalol or esmolol), CCBs (clevidipine or nicardipine), hydralazine, enalapril, nitroprusside and fenoldopam (explained here).	Nitroprusside → ↑ NO → ↑ cGMP → vein/arteries dilate → ↓ BP. Fenoldopam D1 agonist, arteries dilate, especially in kidneys → maintenance renal perfusion + ↑ Na^+/H_2O excretion → ↓ BP.	Nitroprusside prolonged use → cyanide poisoning → inhibition ETC → severe lactic acidosis. Fenoldopam → vasodilation → headache, flushing, nausea.

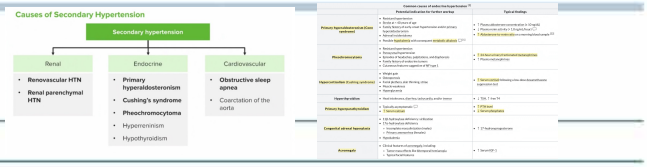


TABLE 2.1-22. Subset of Identifiable Causes of Secondary Hypertension

CATEGORY	DEFINITION
Cardiovascular causes	Coarctation of the aorta, aortic regurgitation, pre-eclampsia/eclampsia (vascular issue of placental spiral arteries)
Obstructive sleep apnea	Hypoxia → ↑ sympathetic tone → systemic + later pulmonary hypertension
Drug-induced	Birth control pills = most common cause in young women; exogenous glucocorticoids, stimulants (cocaine), decongestants (contain sympathomimetic agents), TCAs/SNRIs (block norepinephrine reuptake), nicotine, caffeine, NSAIDs (↓ renal prostaglandin synthesis → ↓ GFR → ↑ Na^+/H_2O retention)
Endocrine causes	Hyperaldosteronism (eg, Conn's syndrome), hypercortisolism (eg, Cushing disease), pheochromocytoma (eg, MEN 2A/B), congenital adrenal hyperplasia, thyroid (hyperthyroidism and hypothyroidism), hyperparathyroidism, acromegaly (↑ growth hormone/IGF-1)
Renal causes	Renal artery stenosis = most common (atherosclerosis > fibromuscular dysplasia) overall cause; chronic kidney disease also very common cause; glomerular diseases (eg, glomerulonephritis, diabetic nephropathy); polycystic kidney disease

SECONDARY HYPERTENSION

HTN that occurs secondary to an identifiable cause (see Table 2.1-22). Identifiable causes of HTN account for a minority (~5%) of cases. Patients should be worked up for secondary causes of HTN if they are younger (age <35), have a severely elevated or refractory-to-treatment BP, or a specific sign indicating a secondary cause.

HYPERTENSIVE EMERGENCY/URGENCY

Presentation

The features of severe HTN, hypertensive urgency, and emergency are compared in Table 2.1-23.

Treatment

Goal: A 25% reduction in BP from baseline or <160/100 mm Hg for both hypertensive urgency and emergency. Medications: eg, labetalol, captopril.

- **Hypertensive urgency:** Lower to goal within 24 hours with oral medications.
- **Hypertensive emergency:** ↓ mean arterial pressure (MAP) by ~20% within first hour, not exceeding 25% in 24 hours. Do NOT lower too quickly or to normal BP, as autoregulation of BP cannot adjust quickly enough → ischemia (eg, stroke, MI). Classically occurs in a patient with long-standing HTN who stops medications.
- **Medications:** Any IV antihypertensive medication can be acceptable, as the specific drug available is not as important as proper dosing of it to manage BP to goal. Drugs commonly used: β-blockers (labetalol or esmolol), CCBs (clevidipine or nicardipine), D1 agonist (fenoldopam), hydralazine, enalapril, **nitroprusside** (prolonged use → cyanide poisoning → inhibition of electron transport chain [ETC] → severe lactic acidosis).

TABLE 2.1-23. Presentation of Hypertensive Urgency and Emergency

CONDITION	DEFINITION
Severe hypertension	SBP >180 or DBP >120 mm Hg
Hypertensive urgency	Severe hypertension + no end-organ damage* (perhaps mild headache)
Hypertensive emergency	Severe hypertension + end-organ damage[a]

[a] End-organ damage is defined as any of the following manifestations:
- **CNS:** Encephalopathy (confusion), stroke, retinal hemorrhage (blurry vision), ↑ intracerebral pressure (papilledema).
- **Cardiovascular:** Acute coronary syndromes, angina, dyspnea, heart failure, aortic dissection, microangiopathic hemolytic anemia (MAHA) (endothelial injury → thrombus → MAHA; mostly historical).
- Key pathologic finding: **Hyperplastic arteriosclerosis** → widespread ischemia.
- **Renal:** Acute kidney injury, hematuria, proteinuria.

MNEMONIC

Causes of secondary hypertension

CODER

Cardiovascular cause
Obstructive sleep apnea
Drug induced
Endocrine causes
Renal causes

KEY FACT

Hypertensive emergency is diagnosed based on the presence of hypertension-induced end-organ damage, NOT a specific BP measurement. Generally, however, severe hypertension has an SBP >180 mm Hg or DBP >120 mm Hg present.

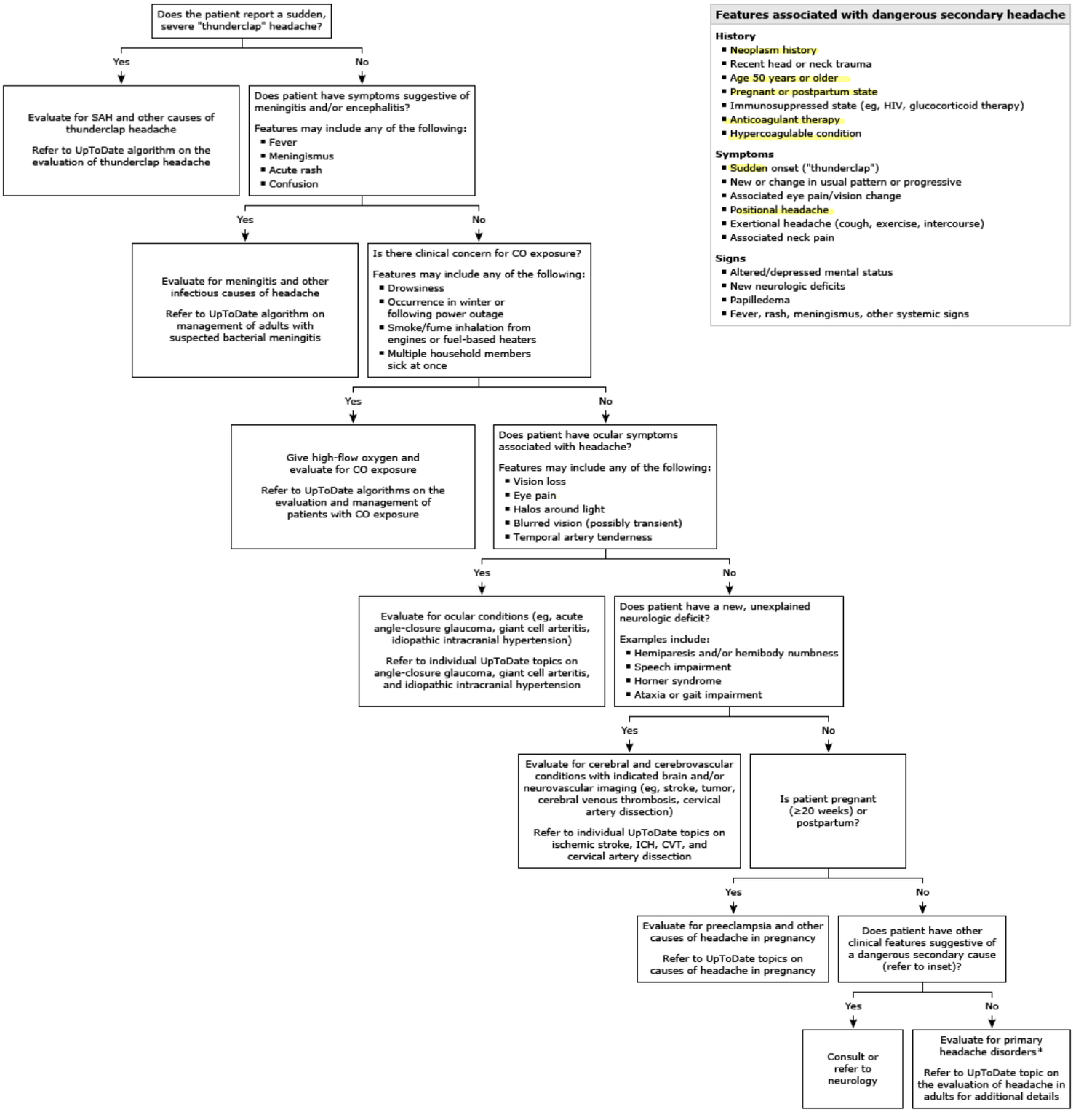
KEY FACT

Cyanide poisoning classically presents with hypertensive emergency after nitroprusside therapy with altered mental status + widespread features of tissue hypoxia. Immediately initiate antidote(s):
- Hydroxocobalamin (first-line)
- Sodium thiosulfate (coadminister with hydroxocobalamin)
- Sodium or amyl nitrate

Q

A 20-year-old man presents with an initial BP of 150/85 mm Hg, and repeat measurement yields 147/85 mm Hg. The patient's potassium level is 3.2 mg/dL. What is the next best diagnostic step?

Emergency evaluation of the adult with new nontraumatic headache



The evaluation of new-onset nontraumatic headache involves assessing for secondary (eg, structural, inflammatory) causes as well as identifying primary headache syndromes.

SAH: subarachnoid hemorrhage; CO: carbon monoxide; ICH: intracranial hemorrhage; CVT: cerebral venous thrombosis; HIV: human immunodeficiency virus.

* Primary headache syndromes include migraine and related conditions, tension-type headaches, trigeminal autonomic cephalalgias (cluster headache, paroxysmal hemicrania, short-lasting unilateral neuralgiform headache attacks, and hemicrania continua), and less common primary headache disorders (eg, new persistent daily headache, primary cough headache, primary exercise headache). Refer to UpToDate topics for additional details.

Praimary Headeche

Migraine

- Most common primary headache
- Subtypes
  - Migraine without aura
    - Triggers
      - Menses
        - 1 to 2 days prior to onset of bleeding, often persisting for up to 3 days into bleeding
        - pure menstrual migraine — migraine at the time of menstruation only
      - Cheese
      - Alcohol
      - Caffeine withdrawal and overuse
      - Nitrate food — processed meats
      - Wether changes
      - many migraine attacks occur without identifiable triggers
    - Dx
      - At least 5 attacks
      - Lasts for 4-72 hrs without tx
      - At least 2 of these :
        - Unilateral
        - Throbbing
        - Worse with activity
        - Moderate to severe pain
      - At least 1 of these :
        - Nausea/ vomiting
        - Photophobia and phonophobia
  - Migraine with aura
    - classic migraine or complicated migraine
    - Caused by cortical spreading depression — Hyperpolarization followed by depolarization
    - oestogen-based contraceptives is contraindicated — increased cardiovascular risk and stroke
    - Aura
      - Unilateral
      - Last for 5 -60 min
      - Gradual
      - Reversible
      - acephalgic migraine — Aura without headache
      - Types
        - Visiual
          - fortification — zigzag lines off the central vision, usually spreading gradually
          - scotoma
        - Sensory
          - paresthesias
          - tingling
          - pins-and-needles sensation
        - Motor
          - hemiplegic migraine — Sporadic or familial
          - gradual onset of weakness
- Complications
  - Status migranous
    - migraine lasts for more than 72 hours
    - Caused by abortive medication overuse
    - Tx
      - I.V treatment
      - oral steroids
      - ketorolac and diphenhydramine + antiemetics
  - Chronic migrane
    - 15 days/month for more than 3 months
    - at least 8 days of headache consistent with typical migraine
    - Usually have medication overuse headache — Using abortive tx more than twice a week
- Treatment
  - Abortive
    - NSAIDS
    - Triptans
      - serotonin 1b/1d agonists
      - Low risk of serotonin syndrome
    - Caffeine
  - Prophylactic
    - at least a month before assuming them ineffective
    - onabotulinum toxin A
    - CGRP antagonists (Erenumab)

Antihypertensives	Antiseizure Drugs	Antidepressants
Metoprolol	Sodium valproate	Amitriptyline
Propranolol	Topiramate	Venlafaxine
Timolol		

Medications in bold have level A evidence for efficacy. Medications in italics have level B evidence for efficacy.

  - Adjuvant — antiemetics — prochlorperazine and metoclopramide

Tension

- Stress or ordinary headache
- 2nd most common primary headache
- Episodic or chronic
- Not lead to significant disability
- Treatment
  - Abortive
    - NSAID — moderate to severe pain
    - Aspirin
    - Paracetamol
  - Preventive
    - Antidepressants
      - First:amitryptiline
      - 2nd :mirtazapine and venlafaxin
    - Muscle relaxants — tizanidine
  - Adjuvant
    - Biofeedback
    - physical therapy
- Chronic tension — >15 daus per month

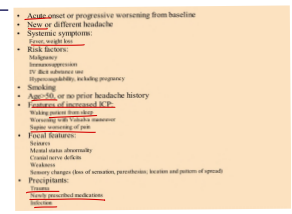
Trigeminal autonomic cephalgia

- 3rd most common
- Unilateral pain associated with cranial autonomic symptoms
- autonomic symptoms
  - Conjuctival injection or lacrimation
    - Can be Both in cluster
    - Not both in SUNA
    - Both in SUNCT
  - Eyelids edema
  - Ptosis , miosis or both
  - Nasal congestion
  - Rhinorhhea
  - fullness in the ear
  - Forehead and facial sweating or flushing
- Subtypes
  - Cluster
    - recurrent attacks for weeks to months
    - followed by remission for months to years
    - During attack > restless and pacing
    - episodic or chronic
    - Triggers
      - Alcohol
      - Histamine
      - nitroglycerine
    - Chronic cluster — intractable cluster headaches with less than 1 month of remission before the recurrence of symptoms
    - Treatment
      - Abortive
        - 100% O2 12-15 litter
        - Triptans
        - Occipital nerve block — In chronic cluster
        - dihydroergotamine
      - Preventive
        - First: Verapamil
        - glucocorticoids
        - 2nd :Lithium and topiramate
    - with horner syndrome
  - Short-lasting unilateral neuralgiform headache attacks
    - SUNCT — Both conjunctival injection and lacrimation
    - SUNA — With autonomic symptoms as above
    - Autonomic symptoms at the same side of headache
    - episodic or chronic
    - with lesion in the posterior fossa
    - Chronic — lasting more than a year, or for less than a year but less than 1 month of remission
    - Treatment
      - Abortive — Intravenous lidocaine
      - Preventive
        - topiramate, gabapentin, and lamotrigine
        - Occipital nerve blocks
  - Hemicrania
    - Subtypes
      - Attacks last between 2 and 30 minutes but can recur within a day
        - Episodic paroxysmal hemicranias — Recurrent attacks separated by at least one pain-free month
        - Chronic paroxysmal hemicranias — Recurrent attacks without remission, or less than 1 month of remission
      - Hemicrania continua — lasting for more than 3 months
    - with lesion in the posterior fossa
    - Treatment — Indomethacin is the definitive treatment

Presented with xmind

Infos

- Pain is due to involoment of pain sensitive structures in the head
- No biomarker available for headache
- Primary headache
  - Pt usually have normal neurological exam
  - Dx by hx and px
- cervicalgia — Associated with pain of cervical muscle or occipital ridge
- occipital Tinel sign — tenderness or tingling when palpating near the occipital protuberance along the occipital nerve
- cortical dysfunction occurs with venous sinus thrombosis
- Opioids are not effective in headache tx
  - secondary opioid use disorder
  - MOH
- Red flags



Secondary headeche causes

- Cerebral hemorrhage
- Ischemic strokes
- Cerebral thrombosis
- Vasculitis
  - Primary
  - 2ndary
- Giant cell arteritis
- encephalitis and meningitis
- Intracranial neoplasms
- autoimmune conditions such as sarcoidosis and lupus
- Traumatic causes
- Intracerebral pressure disorders
  - Intracranial hypertension
  - Intracranial hypotension
- Medication
  - MOH — when patients use an abortive therapy 10 or more times per month for more than 3 months with an increase in headaches
  - Drugs
- Head and neck disorders
  - Sinusitis
  - Temporomandibular joint disorder
- Trigeminal neuralgia

Horner syndrome

BOX 4-1. Etiology of Horner's Syndrome

First-order (or central):
- Hypothalamic: infarcts, tumor
- Mesencephalic: stroke
- Brainstem: ischemia (Wallenberg syndrome), tumor, hemorrhage
- Spinal cord: syringomyelia, trauma

Second-order (or preganglionic):

Cervicothoracic cord/spinal root trauma
- Cervical spondylosis
- Pulmonary apical tumor: Pancoast tumor

Third-order (or postganglionic):
- Superior cervical ganglion (tumor, iatrogenic)
- Internal carotid artery: dissection, trauma, thrombosis, tumor
- Base of skull: tumor, trauma
- Middle ear problems
- Cavernous sinus: tumor, inflammation (Tolosa–Hunt syndrome), aneurysm, thrombosis, fistula

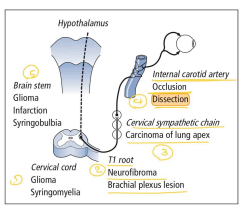
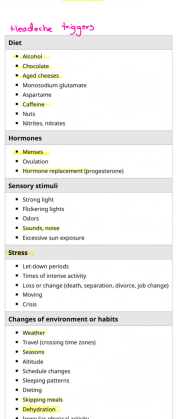


Figure 4.8 Causes of Horner's syndrome – classified according to the site of the lesion along the sympathetic pathway from the hypothalamus to the eye.

TABLE 10-1. Key Features of Primary Headache Disorders

	Episodic Migraine	Episodic Tension	Episodic Cluster	Paroxysmal Hemicrania	SUNCT and SUNA
Sex	Female > male	Female > male	Male > female	Female > male	Male > female
Location	Unilateral > bilateral	Bilateral (band around the head)	Unilateral (behind or around the eye)	Unilateral (behind or around the eye)	Unilateral (behind or around the eye)
Quality	Throbbing, pulsatile	Dull pressure or tightening (vice-like)	Stabbing, burning, boring	Stabbing, burning, throbbing	Stabbing, burning
Severity	Moderate to severe	Moderate	Severe	Severe	Severe
Attack duration	4–72 h	30 min–7 d	15–180 min	2–30 min	1 s–10 min
Attack frequency	Variable	Variable	From 1 every other day to 8/d	>5/d to 40/d	From 1/d to 200/d
Autonomic	No	No	Yes	Yes	Yes



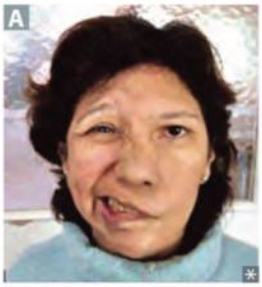
Cranial nerve refl xes

REFLEX	AFFERENT	EFFERENT
Accommodation	II	III
Corneal	V_1 ophthalmic (nasociliary branch)	Bilateral VII (temporal and zygomatic branches—orbicularis oculi)
Cough	X	X (also phrenic and spinal nerves)
Gag	IX	X
Jaw jerk	V_3 (sensory—muscle spindle from masseter)	V_3 (motor—masseter)
Lacrimation	V_1 (loss of reflex does not preclude emotional tears)	VII
Pupillary	II	III

Common cranial nerve lesions

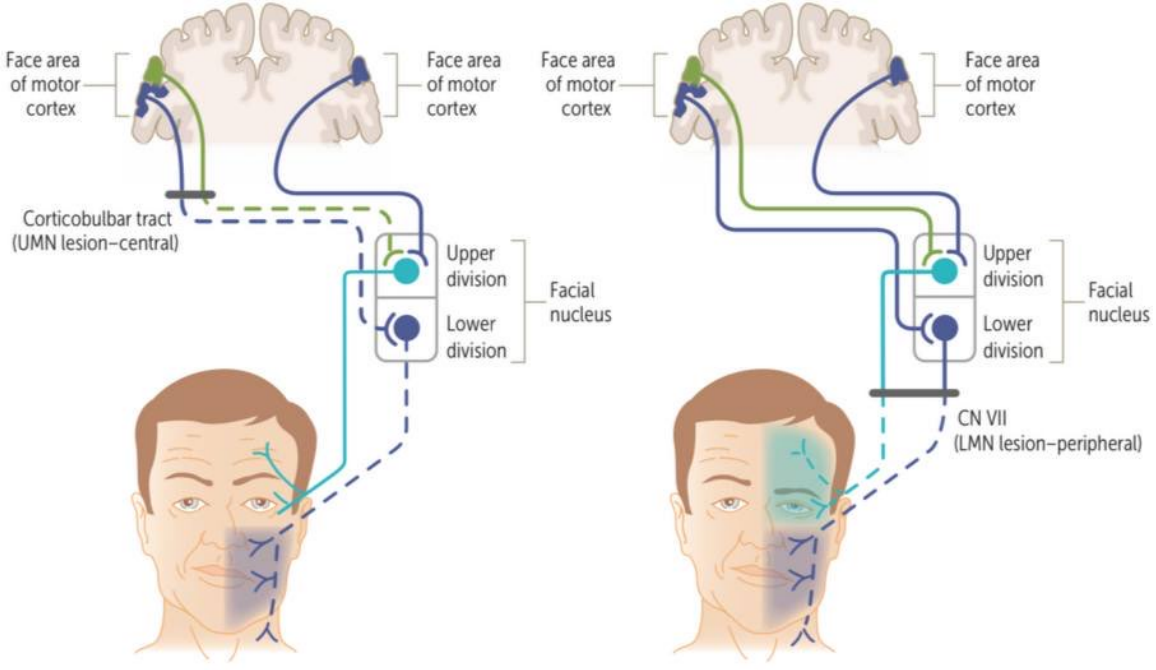
CN V motor lesion	Jaw deviates **toward** side of lesion due to unopposed force from the opposite pterygoid muscle.
CN X lesion	Uvula deviates **away** from side of lesion. Weak side collapses and uvula points away.
CN XI lesion	Weakness turning head to contralateral side of lesion (SCM). Shoulder droop on side of lesion (trapezius).
CN XII lesion	LMN lesion. Tongue deviates **toward** side of lesion ("lick your wounds") due to weakened tongue muscles on affected side.

Facial nerve lesions



Bell palsy is the most common cause of peripheral facial palsy A. Usually develops after HSV reactivation. Treatment: glucocorticoids +/– acyclovir. Most patients gradually recover function, but aberrant regeneration can occur. Other causes of peripheral facial palsy include Lyme disease, herpes zoster (Ramsay Hunt syndrome), sarcoidosis, tumors (eg, parotid gland), diabetes mellitus.

	Upper motor neuron lesion	Lower motor neuron lesion
LESION LOCATION	Motor cortex, connection from motor cortex to facial nucleus in pons	Facial nucleus, anywhere along CN VII
AFFECTED SIDE	Contralateral	Ipsilateral
MUSCLES INVOLVED	Lower muscles of facial expression	Upper and lower muscles of facial expression
FOREHEAD INVOLVEMENT	Spared, due to bilateral UMN innervation	Affected
OTHER SYMPTOMS	Variable; depends on size of lesion	Incomplete eye closure (dry eyes, corneal ulceration), hyperacusis, loss of taste sensation to anterior tongue



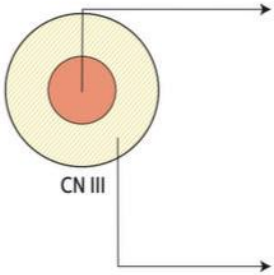
Cranial nerve III, IV, VI palsies

CN III damage

CN III has both motor (central) and parasympathetic (peripheral) components.

Common causes include:

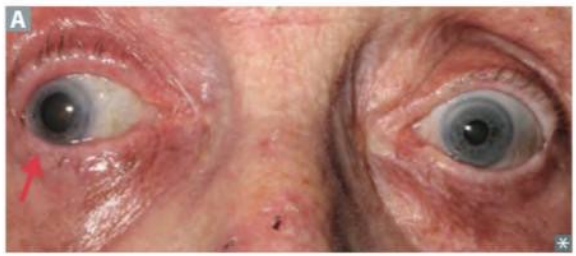
- Ischemia → pupil sparing (motor fibers affected more than parasympathetic fibers)
- Uncal herniation → coma
- PCom aneurysm → sudden-onset headache
- Cavernous sinus thrombosis → proptosis, involvement of CNs IV, V_1/V_2, VI
- Midbrain stroke → contralateral hemiplegia



Motor output to extraocular muscles—affected primarily by vascular disease (eg, diabetes mellitus: glucose → sorbitol) due to ↓ diffusion of oxygen and nutrients to the interior (middle) fibers from compromised vasculature that resides on outside of nerve. Signs: ptosis, "down-and-out" gaze.

Parasympathetic output—fibers on the periphery are first affected by compression (eg, PCom aneurysm, uncal herniation). Signs: diminished or absent pupillary light reflex, "blown pupil" often with "down-and-out" gaze A.

Motor = middle (central)
Parasympathetic = peripheral

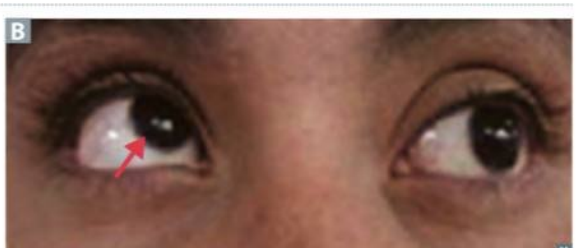


CN IV damage

Pupil is higher in the affected eye B.

Characteristic head tilt to contralateral/unaffected side to compensate for lack of intorsion in affected eye.

Can't see the floor with CN IV damage (eg, difficulty going down stairs, reading).



CN VI damage

Affected eye unable to abduct C and is displaced medially in primary position of gaze.

Excretion of Potassium

90% By kidney

Chronic Kidney disease

70 % By Kidney

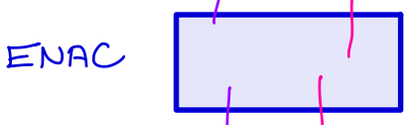
30% By GIT

Constipation a major cause to ↑

M.acidosis , Hyperkalemia

Distal tubules → K^+, H^+

Na, Water → Hypertension



Hypoaldosterone → Na^+ Water

Hyperaldosterone → K^+ H^+] metabolic Alkalosis Hypokalemia

Important

RTA 1,2 → Acidosis , Hypokalemia

RTA 4 → Acidosis , Hyperkalemia

Primary ↓Renin ↑ Aldosterone

2nd ↑ Renin ↑ Aldosterone

Hypertkalemia

medical Emergency
Can Be completely Asymptomatic

Causes

↑ Intake < oral / I.V

Tumor lysis Syndrome

Rhapdomyalisis

CKD → M.C metabolic acidosis

Insulin Difficiency

Beta Blockers

Severe excerise

Hypoaldosteronism

RTA4 → acidosis w/ Hyperkalemia

NSAIDS

ARBS

Cyclosporin

Spironolactone → aldosterone Antagonist

amiloride → work on Enac Directly

Arrythmias

Hyperacute Twave

Prolonged QRS

Cardiac Arrest

management

Calcium Gluconate

Others

Insulin w/ DSW

HCO_3^-

Diuritics (loop) Lasix

Resins

B- agonist

Dialysis → Severe not Responding to medical managment

pseudoHyperkalemia

Fist Clenching

صولح يعني لرحت للاب

Hypokalemia

Replacement
- >3 oral
- <3 I.V
  - No more than 10 mmol/hour
  - Rapid changes can leads to death

Normal Intake = 100 mEq

3.5 - 5.3
mainly Intracellular
<3:300 <2:600 mmol

Perodic Paralysis (AD)
Young Asian man
thyrotoxicosis
- Without stress
  Normal K+
- After excersice
  Carb meal
  Insulin
  Epinephren
  stress
  → Sudden onset Hypokalemia + Paralysis

GI Losses

Vomiting → Cause Hypokalemia By 2nd Hyperaldosterone

Diarrhea → loss of Bicarb → m.acidosis
loss of K+ → Hypokalemia

Renal Loss

Thiazides
Furosimide

RTA 1,2

↑cortisol in High level will mimic Aldosterone effect (Cushing)

Licorich

Barter

Furosimide Like

Gitelman

Thiazide Like

Low Blood Pressure

more Sodium Loss بحفز ال Enac →
- Loss of Sodium
- Compensation
  - ↑Na يرجع يحبس ويرفع بس بالبقي
  - ↓H+, K+
  - Hypokalemia
- Net Sodium more

m.acidosis
Hypokalemia
- Diarrhea laxitisve
- RTA 1,2

اي سبب بالاسفل
↓
alkalosis
Hypokalemia
- High Renin
  ↑BP
  ↓
  Aldosterone
  - *Diuritic abuse
  - *Barter
  - *Gitlman
- Low Renin
  Low or normal BP
  ↓

Normal PH 7.40

Normal $PaCO_2$ 40 ± 15

Normal HCO_3 24

طول ما الفرق 15 يعني

Compensated

Compensation is not a disorder

metabolic acidosis + metabolic Alkalosis

Next Step in metabolic acidosis → Anion GAP

Anion gap = $Na - (HCO_3^- + Cl^-)$

>12

N 8 - 12

High Anion GAP

Low Anion GAP

acidosis , Hypokalemia

↓

Urine Anion GAP

$(Na + K^+) - Cl^-$

(-) → Proximal RTA Diarrhea

(+) → Distal Proximal RTA

Cl in urine is HHyCl

Diarrhea vs RTA

Diarrhea	dRTA
History	History
Urine pH < 5.5	Urine pH > 5.5
Negative urine anion gap	Positive urine anion gap

Check arterial pH

pH < 7.35 — Acidemia

Pco_2 > 44 mm Hg — Respiratory acidosis

Hypoventilation [causes]
- Airway obstruction (OSA, Aspiration)
- Acute lung disease (PE, Pneumonia)
- Chronic lung disease (COPD)
- Opioids, sedatives
- Weakening of respiratory muscles (MS, Guillain Barre)

HCO_3^- < 20 mEq/L — Metabolic acidosis

Check anion gap = $Na^+ - (Cl^- + HCO_3^-)$

> 12 mEq/L — ↑ Anion gap

GOLDMARK:
- Glycols (ethylene glycol, propylene glycol)
- Oxoproline (chronic acetaminophen use)
- L-lactate (lactic acidosis)
- D-lactate (exogenous lactic acid)
- Methanol (and other alcohols)
- Aspirin (late effect)
- Renal failure
- Ketones (diabetic, alcoholic, starvation)
- Iron
- Isoniazid
- Starvation

8–12 mEq/L — Normal anion gap

HARDASS
- Hyperchloremia/hyperalimentation
- Addison disease
- Renal tubular acidosis
- Diarrhea
- Acetazolamide
- Spironolactone
- Saline infusion
- Conduit

Cause	Mechanism	Diagnosis	Treatment
Methanol	Ingestion	Methanol level	Fomepizole/dialysis
Uremia	Renal failure	BUN/Cr	Dialysis
DKA	Insulin deficiency	Serum ketones	Insulin, fluids
Propylene Glycol	Ingestion	Propylene glycol level	Dialysis
Iron	Ingestion	Serum iron level	Deferoxamine
Lactic Acidosis	Hypoperfusion	Serum lactate	Restore perfusion
Ethylene Glycol	Ingestion	Ethylene glycol level	Fomepizole/dialysis
Salicylates	Aspirin overdose	Aspirin level	Urine alkalization

$$\text{Osmolarity} = 2[Na] + \frac{\text{Glucose}}{18} + \frac{\text{BUN}}{1.5}$$

Osmolar GAP = measured osmolarity − calculated Osmolarity
normally ≤ 10
> 10 : methanol, mannitol, ethylene Glycol

pH > 7.45 — Alkalemia

Pco_2 < 36 mm Hg — Respiratory alkalosis

Hyperventilation (Normal RR = 12–20)
- Anxiety/panic attack
- Hypoxemia (eg, high altitude)
- Salicylates (early)
- Tumor
- Pulmonary embolism
- Pregnancy

Parameter	Change
P_{O_2} (alveoli, artery)	Decrease
Respiratory rate	Increase
Carbon dioxide	Decrease
pH	Increase

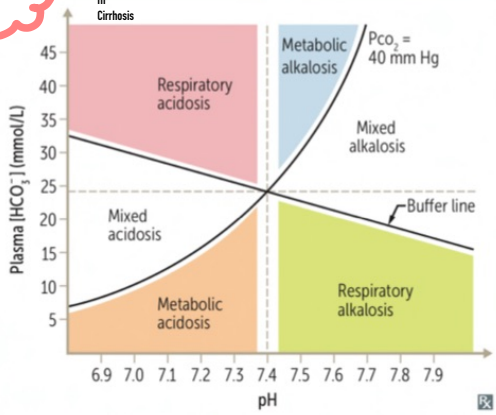
HCO_3^- > 28 mEq/L — Metabolic alkalosis

Na^+ Reabsorption ↙ ++ By Aldosterone
ENaC → H^+, K^+ Loss

Check urine Cl^-

> 20 mEq/L — Saline-resistant
- Hyperaldosteronism
- Bartter syndrome
- Gitelman syndrome

< 20 mEq/L — Saline-responsive
- Vomiting
- Recent loop/thiazide diuretics
- Antacids



Milk alkali Syndrome

↑ Ca → ↑ Base
→ Polyuria → Volume Contraction → ↑ RAAS

Liddle Syndrome
- ↑ activity of ENaC in Juxtaglomerular
- Treated By amiloride

SAME
- ↓ Aldosterone
- ↑ HTN
- Hypokalemia
- Metabolic Alkalosis

SAME → Cortisol → Cortisone (✗ 11-β Hydroxysteroid dehydrogenase)
Treated By: Amiloride, Spironolactone + Dexamethasone

Licorice → glycyrrhetinic Acid
Inhibit the same enzyme

Nail changes

3.4 The nails in systemic disease

Nail changes	Description of nail	Differential diagnosis
Beau's lines	Transverse grooves (see Fig. 3.7B)	Sequella of any severe systemic illness that affects growth of the nail matrix
Clubbing	Loss of angle between nail fold and nail plate (see Fig. 3.8)	Serious cardiac, respiratory or gastrointestinal disease (see Box 3.5)
Leuconychia	White spots, ridges or complete discoloration of nail (see Fig. 3.7C)	Trauma, infection, poisoning, chemotherapy, vitamin deficiency
Lindsay's nails	White/brown 'half-and-half' nails (see Fig. 12.7)	Chronic kidney disease
Koilonychia	Spoon-shaped depression of nail plate (see Fig. 3.7D)	Iron deficiency anaemia, lichen planus, repeated exposure to detergents
Muehrcke's lines	Narrow, white transverse lines (see Fig. 12.6)	Decreased protein synthesis or protein loss
Nail-fold telangiectasia	Dilated capillaries and erythema at nail fold (see Fig. 14.13B)	Connective tissue disorders, including systemic sclerosis, systemic lupus erythematosus, dermatomyositis
Onycholysis	Nail separates from nail bed (see Fig. 3.7A)	Psoriasis, fungal infection, trauma, thyrotoxicosis, tetracyclines (photo-onycholysis)
Onychomycosis	Thickening of nail plate with white, yellow or brown discoloration	Fungal infection
Pitting	Fine or coarse pits in nail (see Fig. 3.7A)	Psoriasis (onycholysis, thickening and ridging may also be present), eczema, alopecia areata, lichen planus
Splinter haemorrhages	Small red streaks that lie longitudinally in nail plate (see Fig. 4.5B)	Trauma, infective endocarditis
Yellow nails	Yellow discoloration and thickening (see Fig. 14.13C)	Yellow nail syndrome

3.5 Causes of clubbing

Congenital or familial (5–10%)

Acquired

- Thoracic (~70%):
  - Lung cancer
  - Chronic suppurative conditions: pulmonary tuberculosis, bronchiectasis, lung abscess, empyema, cystic fibrosis
  - Mesothelioma
  - Fibroma
  - Pulmonary fibrosis
- Cardiovascular:
  - Cyanotic congenital heart disease
  - Infective endocarditis
  - Arteriovenous shunts and aneurysms
- Gastrointestinal:
  - Cirrhosis
  - Inflammatory bowel disease
  - Coeliac disease
- Others:
  - Thyrotoxicosis (thyroid acropachy)
  - Primary hypertrophic osteoarthropathy

Anemia

Physiologic Classification of Anemia