déjà review

Internal Medicine

- Written by medical students who just aced the USMLE Step 2
- Rapid-fire, quick hit format for maximum retention
- All-inclusive, yet concise coverage of behavioral science
- Clinical vignettes chapter preps you for cases you'll see on the exam

REMEMBER WHAT YOU ALREADY KNOW

Sarvenaz Saadat

DEJA REVIEW™

Internal Medicine

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DEJA REVIEW™

Internal Medicine

Sarvenaz S. Saadat, MD

School of Medicine
University of California, Irvine
Irvine, California

Resident in Family Medicine
Kaiser Permanente
Woodland Hills Medical Center
Woodland Hills, California

New York Chicago San Francisco Lisbon London Madrid Mexico City
Milan New Delhi San Juan Seoul Singapore Sydney Toronto

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To my husband, for all his patience and encouragement. This book would not have been possible without you.

To our baby, while still in the womb, whose kicks, squirms and wiggles have brought smiles to my face daily. I hope that I can inspire you as much as you inspire me.

To my parents, for always inspiring me to do bigger and better things.

To my brother—keep reaching for the stars. You can achieve anything you put your mind to.

Contents

Contributors

Noah Craft, MD PhD, DTM&H
Assistant Professor
Department of Internal Medicine,
Division of Dermatology
Harbor-UCLA Medical Center
Los Angeles, California

Henry J. Feldman, MD
Medical Informatics,
Division of General Internal Medicine,
Department of Internal Medicine
NYU School of Medicine
New York

William Herring, MD, FACR
Vice Chairman and Radiology Residency
Program Director
Albert Einstein Medical Center
Philadelphia, Pennsylvania

Reviewers

Daniel Behroozan, MD
Department of Dermatology
University of California
Los Angeles, California

Paul Bellamy, MD
Department of Pulmonary and Critical
Medicine
Kaiser Permanente
Woodland Hills Medical Center
Woodland Hills, California

Jia-ling Chou, MD
Department of Hematology-Oncology
Kaiser Permanente
Woodland Hills Medical Center
Woodland Hills, California

Afshin Khatibi, MD
Department of Gastroenterology
Kaiser Permanente
Woodland Hills Medical Center
Woodland Hills, California

Rashmi Nadig
Department of Infectious Disease
Kaiser Permanente
Woodland Hills Medical Center
Woodland Hills, California

Pamela Nagami, MD

Department of Infectious Disease
Kaiser Permanente
Woodland Hills Medical Center
Woodland Hills, California

Braden Nago, MD
Department of Neurology
Kaiser Permanente
Woodland Hills Medical Center
Woodland Hills, California

Frederick Ziel, MD
Department of Endocrinology
Kaiser Permanente
Woodland Hills Medical Center
Woodland Hills, California

Student Reviewer

Mary M. Bonar
College of Osteopathic Medicine
Ohio University
Dayton, Ohio

Preface

The principles learned in*internal medicine* are the fundamental core principles applied in clinical medicine as well as the largest proportion of questions posed on the USMLE Step 2 exam. In order to do well both on the wards and on the Step 2 exam, you must have a solid foundation in these principles. This guide has been written as a high-yield resource, to endorse the rapid recall of the essential facts in a well-organized and efficient manner.

Organization

All concepts are presented in a question and answer format that covers the key facts on hundreds of commonly tested internal medicine topics that may appear on the USMLE Step 2 exam. The material is divided into chapters organized by internal medicine subcategories, along with a special chapter at the end that incorporated the material with their clinical presentation and relevance.

This question and answer format has several advantages:

• It provides a rapid, straightforward way for you to assess your strengths and weaknesses.

• It allows you to efficiently review and commit to memory a large body of information.

• It offers a break from tedious, convoluted multiple choice questions.

• The clinical vignettes incorporated expose you to the prototypical presentation of diseases classically tested on USMLE Step 2.

• It serves as a quick, last minute review of high-yield facts.

The compact, condensed design of the book is conducive to studying on the go, especially during any downtime throughout your day.

How to use this book

This text is intended to be used not only to study for the USMLE Step 2 examination but is also an essential tool while on the internal medicine and medicine subspecialty rotations, and during medical school. Remember, this text is not intended to replace comprehensive textbooks, course packets, or lectures. It is simply intended to serve as a supplement to your studies during your internal medicine clinical rotation and throughout your preparation for Step 2. We encourage you to begin using this book early in your third year to reinforce topics you encounter while on the wards. Also, it is recommended that you cover up the answers (rather than just reading both the questions and the answers) and quiz yourself or even your classmates. Carry the book in your white coat pocket so that you can easily access study material during down time. However you choose to study, we hope you find this resource helpful throughout your clinical years and during your preparation for USMLE Step 2. Best of Luck!

Sarvenaz S. Saadat, MD

Introduction

Important Lab Values

Blood Test	Value
Albumin, serum	3.2–5.5 g/dL
Alkaline phosphatase	26–110 IU/L

Ammonia, plasma	17–60 µmol/L
Amylase, serum	25–125 IU/L
Bilirubin	
Direct	0–0.2 mg/dL
Total	0–1.4 mg/dL
Calcium	9–10.6 mg/dL
Chloride	101–111 mEq/L
CO2	25–34 mEq/L
Cortisol, a.m.	6–28 µg/dL
Cortisol, p.m.	3–16 µg/dL
CPK	22–269 U/L
Creatinine	0.5–1.3 mg/dL
ESR, male	0–15 mm/h
ESR, female	0–20 mm/h
d–Dimer	< 0.5 µg/mL
Ferritin, male	23–233 ng/mL

Ferritin, female	10–1107 ng/mL
Folate	3–18.2 ng/mL
Glucose	70–115
Hemoglobin, male	13.5–16.9 g/dL
Hemoglobin, female	11.5–15 g/dL
Hematocrit, male	39.5–50%
Hematocrit, female	34–44%
Iron, male	49–181 µg/dL
Iron, female	37–170 µg/dL
LDH	91–180 IU/L
Lipase	4–24 IU/L
Magnesium	1.8–2.5 mg/dL
Osmolality, serum	278–305 mosmol/kg
Osmolality, urine	50–1200 mosmol/kg
Phosphorus	2.5–4.6 mg/dL

Platelets	150–450,000
Potassium	3.3–4.8 mEq/L
Pre-albumin	18–45 mg/dL
PSA, Age 0–39	0–1.4 ng/mL
PSA, Age 40+	0–2.8 ng/mL
Protein, total	6.7–8.2 g/dL
Reticulocyte count	0.5–1.5%
SGOT	10–42 U/L
SGPT	< 60 U/L
Sodium	135–145 mEq/L
T3 uptake	25–38%
T4 total	0.7–2.1 ng/dL
Transferrin	212–360 mg/dL
TSH	0.5–5.0 µIU/mL
Uric acid	2.6–7.2 mg/dL
WBC	4500–10,500

Writing Notes

Daily progress note: This should be in SOAP format.

Subjective: In this area you should report any overnight events, how the patient is feeling today, any complaints or problems the patient may be experiencing, and pertinent positives and negatives.

Objective: Any physical findings are reported in this section.

Vitals: temperature, max temperature, blood pressure, pulse, respiratory rate, oxygen saturation

Glucose (if patient is diabetic): Ins and Outs (Ins = IV fluids + po intake + any parenteral intake or blood products over 24 hours and Outs = urine output + stool + other [NG tube, chest tube, drains, emesis])

Physical examination:

General: Patients general appearance

HEENT (head, eyes, ears, nose, throat)

Cardiovascular

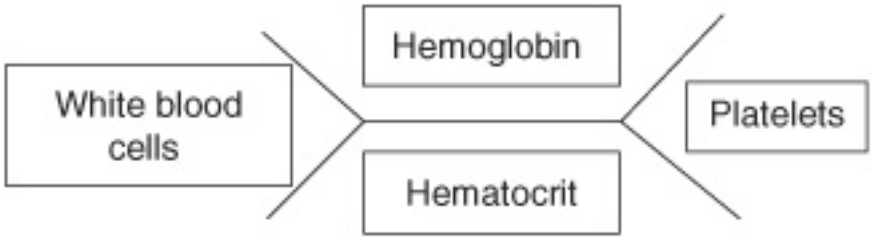
Pulmonary

Abdomen

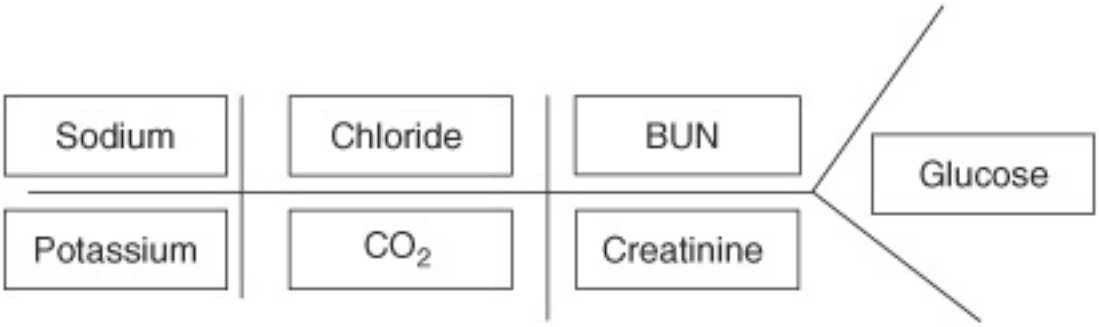
Extremities

Neurologic

Labs: Laboratory tests are reported here.



Complete blood count (CBC).



Chemistry 7.

Meds: Some people include a list of all the medication the patient is currently using.**Assessment** and**plan:** Write a summary of the patient, their problem(s) and possible differentials. Then write the plan for each problem.

X

Sign your note

Example

S: Patient has no complaints today. She is no longer short of breath and was able to

ambulate yesterday.

O: T: 36.8, Tmax 37°C, P: 70–85, BP: 128–148/68–80, RR: 20, O2sat: 95–100%, I/O: 1500/2000

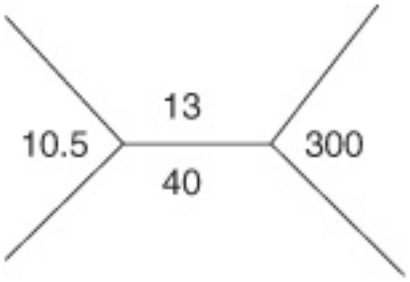
GEN: NAD (no apparent distress)

HEENT: PERRLA (pupils are equally round and reactive to light accommodation), EOMI (extraocular muscles are intact), NCAT (normocephalic atraumatic)

CV: RRR no M/R/G (regular rate and rhythm with no murmurs, rubs, or gallops) Pulm: CTA B (clear to auscultation bilaterally); no R/R/W (no rhonchi, rales, or wheezes)

Abd: S (soft)/NT (non-tender)/ND (non-distended)/NABS (normal abdominal bowel sounds)

Ext: no C (clubbing)/C (cyanosis)/E (edema); no calf tenderness



Labs: CBC.

Meds: Aspirin 81 mg daily

Albuterol nebs q4 hours

A/P: 35 y/o female with asthma exacerbation now improved and at baseline

1. Asthma: Patient improved with steroids and albuterol/atrovent treatments. Patient will be sent home with a medrol pack and albuterol inhaler. Patient will also be sent home with a steroid inhaler.

2. Disposition: Patient will be discharged home today with follow-up in 1 week.

Greta Student, MS III

History and Physical Examination

Chief complaint (CC): Main problem that the patient is here for (e.g., shortness of breath)

History of present illness (HPI): Include a chronologic history of the patient's problems and prior treatments for this problem as well as any other history that is pertinent. Describe symptoms in terms of onset, duration, quality of discomfort, setting, instigating and relieving factors.

Past medical history (PMH): Include the patient's medical history and be sure to ask about heart disease, hypertension, diabetes, cancer, and any other pertinent history. The patient's medication list can often serve as a clue since patients will sometimes forget to mention medical problems that they have.

Surgical history (SH): Include all operations a patient has as well as when and why.

Medication: List all the patient's medications as well as doses and frequency with which they are taken. Also ask the patient about any possible over-the-counter medications and alternative meds.

Allergies: Name all drugs the patient is allergic to and what happened when they took the drug.

NKDA means “no known drug allergies”

Family history (FH): This should include the health, medical problems of the patient’s family including parents, grandparents, siblings, and often, aunts, uncles, and cousins. Be sure to ask about heart disease, diabetes, hypertension, hyperlipidemia, and cancer.

Social history (SH): This section includes the patient’s marital status, occupation, exercise history, sexual history, diet, and tobacco use, drug use, and alcohol use.

Review of systems (ROS): Report all the pertinent positive and negative signs and symptoms that the patient reports (e.g., the patient denies any nausea, vomiting, diarrhea, chest pain, cough, travel history, …)

Physical examination: Include all pertinent organs and systems

Vital signs: Tmax, BP, HR, RR, O2saturation, Ins/Outs
General:
HEENT:
Neck:

Cardiovascular:
Pulmonary:
Abdominal:
Genitourinary:
Back:
Extremities:
Neurologic:

Labs and studies: Include all labs and studies that you have results for Assesment and plan: Write a summary of the patient’s problems and differential diagnoses as well as a plan

for each problem.

X

Sign your name at the bottom

Procedure Note

Whenever a procedure is done, a procedure note must be written in the chart. Always remember to get consent from the patient before a procedure is done. Below is an example.

Procedure Note:

Procedure: Biopsy of left lower abdominal macule

Indications: Rule out melanoma

Consent: The risks, benefits, and possible side effects of the procedure including but not exclusive of pain, bleeding, infection, and scar were explained to the patient who understands and wishes to have the procedure done.

Preparation: The area was prepped and draped in a sterile fashion.

Anesthesia: The area was anesthetized with 10 cc of 2% lidocaine solution using a 30-gauge needle.

Procedure: A wide excision (1 cm on each side) of the macule was done using a number-15 blade. There was minimal bleeding. The site of the excision was closed using 4-0 nylon sutures and the specimen was sent to pathology for examination.

Complications: The patient tolerated the procedure with no complications.

Greta Student, MS III

How to Write a Prescription

Patient name: _____ Medical record number_____
Address:_____ Phone #:_____DOB:_____

Rx: *Drug name, drug dose*
Dispense # : Write number here
Sig: Write instructions here
Refill:

X *Sign your name here* Date:_____

Example

Patient name: Ima N. Payne_____ Medical record number: 12345678
Address: 1111 Oak Street ; LA, CA Phone #: 222-2222 DOB:1/1/69_____

Rx: Famotidine 20 mg tablets
Dispense # : Sixty
Sig: Take two tablets by mouth twice daily
Refill: 1

X *Dr. Health* Date: 10/20/09

How to Admit a Patient

Admission Orders

Admit to:

Floor:

Service:

Medical student name:

Resident name:

Attending name:

Diagnosis:

Primary diagnosis:

Other diagnoses:

Condition:

Good, stable, fair, guarded, critical

Vitals:

Per routine (usually q2 hours in ICU and q4 hours on the floor)

q shift

q __ hours

Activity:

Ad lib

Bed rest

To chair

Ambulate bid

Bathroom privileges

Fall risk

Nursing:

Neuro check q__hours

Weigh daily

Pulse oximetry

Wound care

CALL MD for systolic blood pressure (SBP) > 165 or < 110; diastolic BP > 100 or < 60;

Pulse > 100, Temp > 38.5

etc.

Diet:

Regular

Diabetic

Low sodium

Low fat

Clear liquid

Soft

npo (nothing by mouth)

Ins and Outs: strict, per routine

IV fluids: e.g., D5½NS @ 100 cc/h

Drains: Foley, NG tube to suction, chest tube to suction

Medication:

Medication name, dose, route, frequency

Home medication should be written out

Antibiotics

Etc.

Special: These are things you will usually need to think about.

DVT prophylaxis

Pain medications

Antiemetics

Antipyretics

Allergies:

NKDA (no known drug allergies)

Penicillin

Sulfa

Etc.

Labs/studies:

CBC, electrolytes, BUN, Cr, ECG, radiology studies; other labs

Example

Admit to 2 North, Internal Medicine, Medical Student: Stew Dent;

Resident:

Dr. Smith; Attending: Dr. Bay

Diagnosis: Pneumonia

Condition: Fair

Vitals: Per routine

Activity: Bathroom privileges

Nursing: Pulse oximetry; call MD for systolic blood pressure (SBP) > 165

or < 110; diastolic BP > 100 or < 60; Pulse > 100, Temp > 38.5; Pulse ox < 90%

Diet: Regular

Ins and outs: Strict

IV fluids: D5NS@120 cc/h

Meds: Ceftriaxone 2 g IV q24 hours

Azithromycin 500 mg IV q24 hours

Tylenol 650 mg po q6 hours prn mild pain or Temp > 38.5

Special: Sequential compression stockings

Allergies: NKDA

Labs/studies: PA and lateral CXR; sputum culture/Gram stain; CBC;

electrolytes; BUN; Cr

Abbreviations You Should Know

AAA	abdominal aortic aneurysm
AAS	acute abdominal series
abd	abdomen
Abx	antibiotics
ac	before meals
ACLS	advanced cardiac life support
ACTH	adrenocorticotropic hormone
ADA	American Diabetes Association
ADH	antidiuretic hormone
ADL	activities of daily living
AFB	acid fast bacillus
AFP	alpha feto protein
AI	aortic insufficiency
AKA	above knee amputation

alk phos	alkaline phosphatase
ALL	acute lymphocytic leukemia
ALS	amytrophic lateral sclerosis
AMA	against medical advice
AMI	acute myocardial infarction
AML	acute myelogenous leukemia
ANA	antinuclear antibody
ant	anterior
AP	anteroposterior
ARDS	acute respiratory distress syndrome
ARF	acute renal failure
APTT	activated partial thromboplastin time
AR	aortic regurgitation
AS	aortic stenosis
ASA	aspirin
ASD	atrial septal defect

ASO	antistreptolysin O
ATN	acute tubular necrosis
AV	arteriovenous
AVN	atrioventricular node
B	bilateral
BBB	bundle branch block
BE	barium enema
BIB	brought in by
bid	two times per day
BKA	below knee amputation
BM	bowel movement; bone marrow
BPH	benign prostatic hypertrophy
BRBPR	bright red blood per rectum
BRP	bathroom privileges
BS	blood sugar; breath sounds

BUN	blood urea nitrogen
Bx	biopsy
c	with
Ca	calcium
CA	cancer, carcinoma
CABG	coronary artery bypass graft
CAD	coronary artery disease
cath	catheter
CBC	complete blood count
CBG	capillary blood gas
CC	chief complaint
CEA	carcinoembryonic antigen
CF	cystic fibrosis
CHF	congestive heart failure
CK-MB	creatinine kinase-myocardial band
CLL	chronic lymphocytic leukemia

CML	chronic myelogenous leukemia
CMV	cytomegalovirus
CN	cranial nerves
CNS	central nervous system
CO	cardiac output
c/o	complains of
COPD	chronic obstructive pulmonary disease
CP	chest pain
CPAP	continuous positive airway pressure
CPK	creatinine phosphokinase
CPR	cardiopulmonary resuscitation
CRF	chronic renal failure
C and S	culture and sensitivity
CSF	cerebrospinal fluid
CT	computerized tomography

CTAB	clear to auscultation bilaterally
CV	cardiovascular
CVA	cerebrovascular accident
CVAT	costovertebral angle tenderness
CVP	central venous pressure
CXR	chest x-ray
D5½NS	5% dextrose in half normal saline
D5W	5% dextrose in water
DA	dopamine
D/C	discharge, discontinue
Ddx	differential diagnosis
DI	diabetes insipidus
DIC	disseminated intravascular coagulation
DIP	distal interphalangeal joint
DJD	degenerative joint disease
DKA	diabetic ketoacidosis

DM	diabetes mellitus
DNR	do not resuscitate
DOA	dead on arrival
DOE	dyspnea on exertion
DT	delirium tremens
DTR	deep tendon reflexes
DVT	deep vein thrombosis
Dx	diagnosis
EBL	estimated blood loss
ECT	electroconvulsive therapy
EGD	esophagogastroduodenoscopy
EEG	electroencephalogram
EKG	electrocardiogram
EMG	electromyelogram
ENT	ears, nose, and throat

EOMI	extraocular muscles intact
ERCP	endoscopic retrograde cholangiopancreatography
ESR	erythrocyte sedimentation rate
ETOH	alcohol, ethanol
ETT	endotracheal tube
FB	foreign body
FBS	fasting blood sugar
f/c	fever and chills
FEV1	forced expiratory volume in 1 second
FFP	fresh frozen plasma
FH	family history
FRC	functional residual capacity
FTA-ABS	fluorescent treponemal antibody absorption (syphilis)
FTT	failure to thrive
f/u	follow-up

FUO	fever of unknown origin
FVC	forced vital capacity
fx	fracture
GC	gonococcus, gonorrhea
GERD	gastroesophageal reflux disease
GI	gastrointestinal
GU	genitourinary
HA	headache
HBsAg	hepatitis B surface antigen
HBV	hepatitis B virus
Hct	hematocrit
HDL	high-density lipoprotein
HEENT	head, eyes, ears, nose, throat
Hgb	hemoglobin
HIV	human immunodeficiency virus

HLA	histocompatablility locus antigen
h/o	history of
HO	house officer
HOB	head of bed
HPI	history of present illness
HSM	hepatosplenomegaly
HTN	hypertension
Hx	history
ICU	intensive care unit
I&D	incision and drainage
IDDM	insulin-dependent diabetes mellitus
IM	intramuscular
Ig	immunoglobulin
INH	isoniazid
I&O	intake and output
ITP	idopathic thrombocytopenic purpura

IVF	intravenous fluids
IVP	intravenous pyelogram
JVD	jugular venous distention
KUB	kidney ureter, bladder x-ray
LAD	left axis deviation (lymphadenopathy)
LAE	left atrial enlargement
LAP	left atrial pressure
LCM	left costal margin
LDH	lactate dehydrogenase
LLE	left lower extremity
LLL	left lower lobe
LLQ	left lower quadrant
LMN	lower motor neuron
LOC	loss of consciousness
LP	lumbar puncture

LR	lactated ringers
LUE	left upper extremity
LUL	left upper lobe
LUQ	left upper quadrant
LVH	left ventricular hypertrophy
m	murmur
MAO	monoamine oxidase inhibitor
MAP	mean arterial pressure
MCH	mean cell hemoglobin
MCHC	mean cell hemoglobin concentration
MCP	metacarpophalangeal joint
MCV	mean corpuscular volume
MEN	multiple endocrine neoplasia
MI	myocardial infarction
MRSA	methicillin-resistant*Staphylococcus aureus*
MS	mitral stenosis, multiple sclerosis

MVA	motor vehicle accident
MVI	multivitamin
NAD	no apparent distress
ND	nondistended
NG	nasogastric tube
NIDDM	non-insulin-dependant diabetes mellitus
NKDA	no known drug allergies
npo	nothing by mouth
NS	normal saline
NSAID	nonsteroidal anti-inflammatory drug
NSR	normal sinus rhythm
NT	non-tender
N/V	nausea and vomiting
OB	occult blood
OOB	out of bed

OR	operating room
PAC	premature atrial contraction
PAT	paroxysmal atrial tachycardia
PCWP	pulmonary capillary wedge pressure
PDA	patent ductus arteriosus
PE	pulmonary embolism
PEEP	positive end-expiratory pressure
PERRLA	pupils equally round and reactive to light
PFT	pulmonary function test
PMD	primary medical doctor
PMH	past medical history
PMI	point of maximal impulse
PMN	polymorphonuclear cell
PM&R	physical medicine and rehabilitation
PND	paroxysmal nocturnal dyspnea
po	by mouth

POD	post operative day
PR	per rectum
PRBC	packed red blood cells
PT	physical therapy, prothrombin time, pulmonary toilet
pt	patient
PTCA	percutaneous transluminal coronary angioplasty
PTH	parathyroid hormone
PTT	partial thromboplastin time
PUD	peptic ulcer disease
PVC	premature ventricular contraction
PVD	peripheral vascular disease
qAC	before each meal
qd	daily
qid	four times per day

qod	every other day
q4h	every 4 hours
RA	rheumatoid arthritis
RAD	right axis deviation
RAE	right atrial enlargement
RBC	red blood cells
RDW	red cell distribution width
RHD	rheumatic heart disease
RLE	right lower extremity
RLL	right lower lobe
RLQ	right lower quadrant
RML	right middle lobe
r/o	rule out
ROM	range of motion
ROS	review of systems
RR	respiratory rate

RRR	regular rate and rhythm
RT	respiratory therapy
RTA	renal tubular acidosis
RTC	return to clinic
RUE	right upper extremity
RUL	right upper lobe
RUQ	right upper quadrant
RVH	right ventricular hypertrophy
s	without
SBE	subacute bacterial endocarditis
SBO	small bowel obstruction
SBP	subacute bacterial peritonitis
SEM	systolic ejection murmur
SGOT	serum glutamic-oxaloacetic transaminase
SGPT	serum glutamic-pyruvic transaminase

SIADH	syndrome of inappropriate antidiuretic hormone
SL	sublingual
SLE	systemic lupus erythematosus
SOB	shortness of breath
s/p	status post
stat	immediate
subQ	subcutaneous
Sx	symptoms
tab	tablets
TB	tuberculosis
Temp	temperature
TIA	transient ischemic attack
TIBC	total iron-binding capacity
tid	three times per day
TKO	to keep open

TLC	total lung capacity
TPN	total parenteral nutrition
TSH	thyroid-stimulating hormone
TTP	thrombotic thrombocytopenic purpura
TURP	transurethral resection of the prostate
TV	total volume
Tx	treatment
UA	urinalysis
UGI	upper gastrointestinal
UMN	upper motor neuron
URI	upper respiratory infection
US	ultrasound
UTI	urinary tract infection
VC	vital capacity
VCUG	voiding cystourethrogram
VDRL	venereal disease research laboratory (syphilis

	test)
V/Q	ventilation perfusion scan
VSS	vital signs stable
WBC	white blood cells
WNL	within normal limits
y/o	years old

Common Formulas

Maintenance fluids per hour: 4:2:1 rule:

4 mL/kg up to 10 kg + 2 mL/kg from 11 to 30 kg + 1 mL/kg > 30 kg

Example:A person weighing 100 kg should get

(4 × 10) + (2 × 20) + (1 × 70) = 40 + 40 + 70 = 150 cc/h

Maintenance fluids over 24 hours: 100:50:20 rule
100 mL/kg up to 10 kg + 50 mL/kg from 11 to 30 kg + 20 mL/kg > 30 kg

Anion gap: Na – (Cl + HCO3)

Osmolality: 2Na + glucose/18 + BUN/2.8

$$\text{Fractional Na excretion } (FE_{Na}): \frac{\text{urine Na} \times \text{serum creatinine}}{\text{serum Na} \times \text{urine creatinine}}$$

Creatinine clearance, also known as glomerular filtration rate (GFR):

$$\frac{\text{urine creatinine} \times \text{urine volume in mL}}{\text{serum creatinine} \times \text{time in minutes}}$$

$$\text{Estimated creatinine clearance: } \frac{(140 - \text{age}) \times (\text{weight in kg}) \text{ (for females } \times 0.85)}{\text{serum creatinine} \times 72}$$

Corrected Na: Na + [(glucose – 100) × 0.016]

Corrected total calcium: [0.8 × (normal albumin – measured albumin)] + Ca

$$\text{Body water deficit: } \frac{0.6 \times \text{weight (kg)} \times (\text{patient Na} - \text{normal Na})}{\text{normal Na}}$$

Aa gradient: [(713 × FIO2) – (PaCO2/0.8)] – PaO2= 150 - (PaCO2/0.8)] – PaO2

Anion gap: Na – Cl + HCO3(normal value is between 8 and 12 mEq/L)

MAP (mean arterial pressure): diastolic BP + [(systolic BP – diastolic BP)/3]

Cerebral perfusion pressure: MAP – ICP (intracranial pressure)

Statistics

Sensitivity: This determines how well the test is able to detect disease.

$$\frac{\text{Number of patients with disease and positive test}}{\text{Total number with disease}}$$

Specificity: This determines how well the test detects the absence of disease.

$$\frac{\text{Number of patients without disease and negative test}}{\text{Total number without disease}}$$

$$\text{Odds ratio: } \frac{\text{Frequency of events in exposed}}{\text{Frequency of events not in exposed}}$$

$$\text{Relative risk: } \frac{\text{Incidence in exposed}}{\text{Incidence in unexposed}}$$

Patient with disease	Patients with disease
and	**and**
positive test	negative test
True positive	**False negative**
Patient**without** disease	Patient**without** disease
and	**and**
positive test	negative test
False positive	**True negative**

Common formulas statistics.

CHAPTER 1

The Basics

Quick Radiology

CHEST X-RAY

What is the first thing that you should check when evaluating a radiographic study?

Check the**name** of the patient as well as the date.

How can you determine if the chest x-ray (CXR) is adequate?

1. Penetration: Disk spaces can be seen without distinguishing the details of the spine.

2. Inspiratory effort: Diaphragm anteriorly should be below rib 5.

3. Rotation: Spinous processes of thoracic vertebrae should be midway between clavicles.

What is a postrrior anterior (PA) film?

Posterior anterior film. The x-ray is shot from the back of the patient to the plate in front of the patient

What is an anterior posterior (AP) film?

Anterior posterior film. The x-ray is shot from the front of the patient to the back of the patient.

When is an AP film appropriate?

A patient who is bed bound

How is the image altered in an AP film?

The heart appears large.

How should you approach reading a CXR?

Remember**A, B, C, D plus lungs and soft tissue**

Airway: Trachea should be midline.

Bones: Check for any bony defects, fractures, osteolytic lesions.

Cardiac: The heart should be less than ½ the width of the chest.

Diaphragm: There should be no blunting of the costophrenic angles.

No free air should be seen under the hemidiaphragm.

Lungs: Look for any nodules, opacification, bronchial markings.

Soft tissue: Look for any lesions, lymphadenopathy, masses.

Name the parts of the CXR shown below?

1. Sharp costophrenic angle

2. Right atrium

3. Hilum and main bronchus

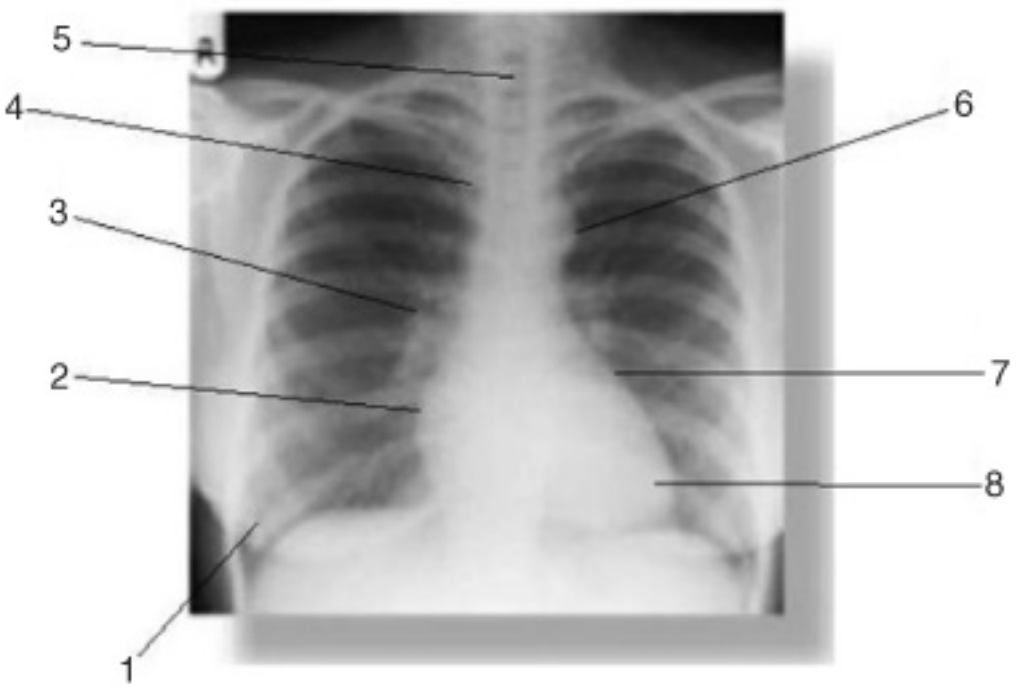
4. Superior vena cava

5. Trachea (midline)

6. Aortic arch

7. Left atrium

8. Left ventricle



(CXR reproduced with permission from William Herring, MD, FACR; Radiology Residency Program Director at Albert Einstein Medical Center in Philadelphia, PA;http://www.learningradiology.com)

OTHER RADIOLOGIC STUDIES

What is a kidneys, ureter, and bladder (KUB)?

X-ray which looks at the kidney, ureter, and bladder

What structures do computed tomographic (CT) scans visualize best?

CT scans visualize bone best and can identify acute bleeds.

What structures does a magnetic resonance imaging (MRI) visualize best?

Soft tissue

Name the radiographic study you would use to evaluate each of the following.

Biliary tract

Right upper quadrant ultrasound

Differentiate between loculated and unloculated pleural effusion

Lateral decubitus film—fluid that is loculated will not layer out

Carotid artery stenosis

Carotid ultrasound

Kidney stones

KUB

Stroke

MRI of the brain

Anterior cruciate ligament (ACL) tear of the knee

MRI of the knee

Name what each of the following radiographic findings is most commonly indicative of:

Flattened diaphragms

Chronic obstructive pulmonary disease (COPD)

Blunted costophrenic angles

Pleural effusion

Air outside pleural lines

Pneumothorax

Consolidation of lung parenchyma

Pneumonia

Dilated loops of small bowel

Small bowel obstruction

Air fluid levels

Small bowel obstruction

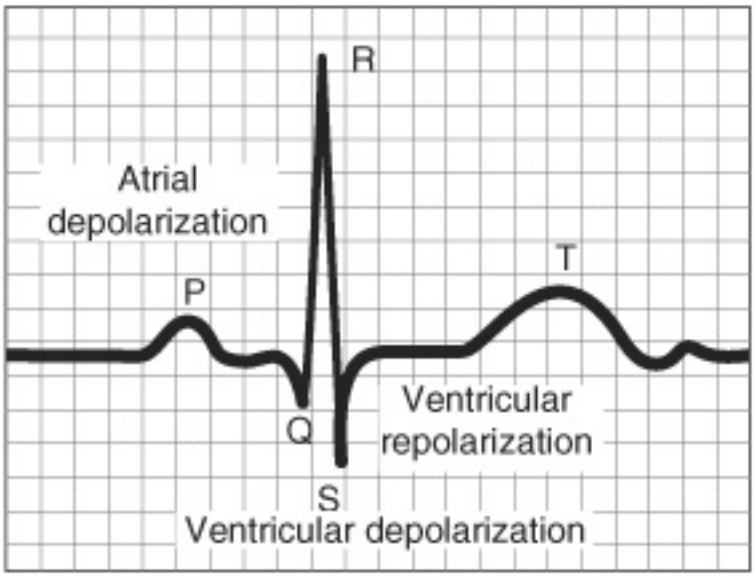


Figure 1-1 Parts of the EKG. (EKG used with permission of Dr. Henry Feldman fromhttp://students.med.nyu.edu/erclub/ekgexpl1.html)

Quick EKG Interpretation

Step 1: Calculate the rate (Fig. 1-2).

Rate = beats per minute.

The easy way to calculate the rate is**300/(# big boxes between 2 QRS complexes) or 300, 150, 100, 75, 60, 50.**

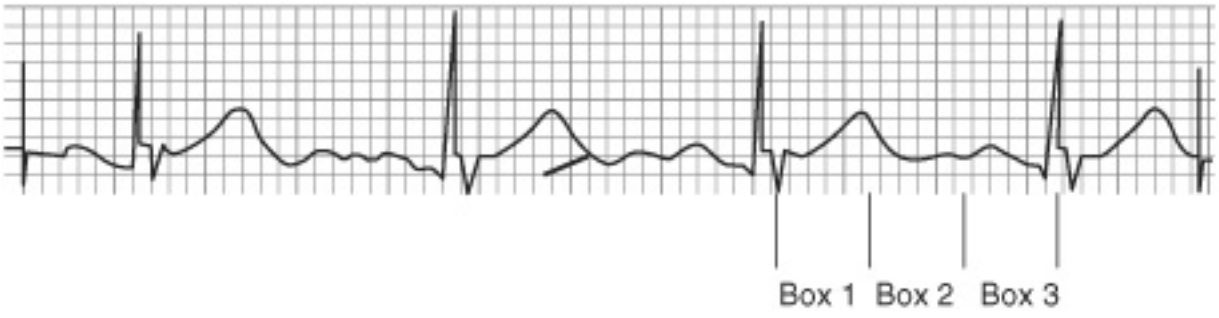


Figure 1-2(EKG used with permission of Dr. Henry Feldman

fromhttp://students.med.nyu.edu/erclub/ekgexpl1.html)

300/3 = 10.

In this example, the rate is about 100 beats per minute.

Step 2: Calculate the rhythm (Fig. 1-3).

Ask the question: Is there a P wave before each QRS? And, are the P waves of the same morphology? If yes, then the rhythm is sinus.

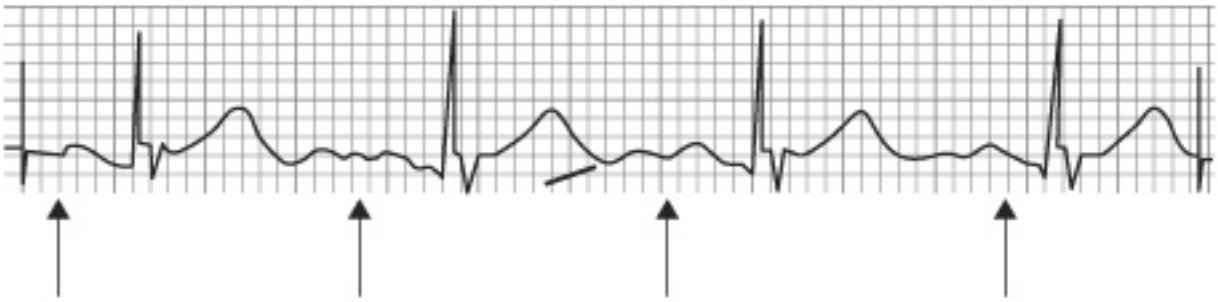


Figure 1-3(EKG used with permission of Dr. Henry Feldman fromhttp://students.med.nyu.edu/erclub/ekgexpl1.html)

In the example, there is a P wave of the same morphology before each QRS, which indicates that the patient is in sinus rhythm. If there were a lack of P waves or a disorganized rhythm, a differential diagnosis, which you will find in the cardiology chapter, would come into play.

Step 3: Determine the axis (Fig. 1-4).

Rules of thumb:

If I and aVF are positive, then axis is normal.

If I is positive and aVF is negative, check lead II.

If lead II is positive, then the axis is normal.

If lead II is negative, then there is left axis deviation.

If I is negative and aVF is positive, then there is right axis deviation.

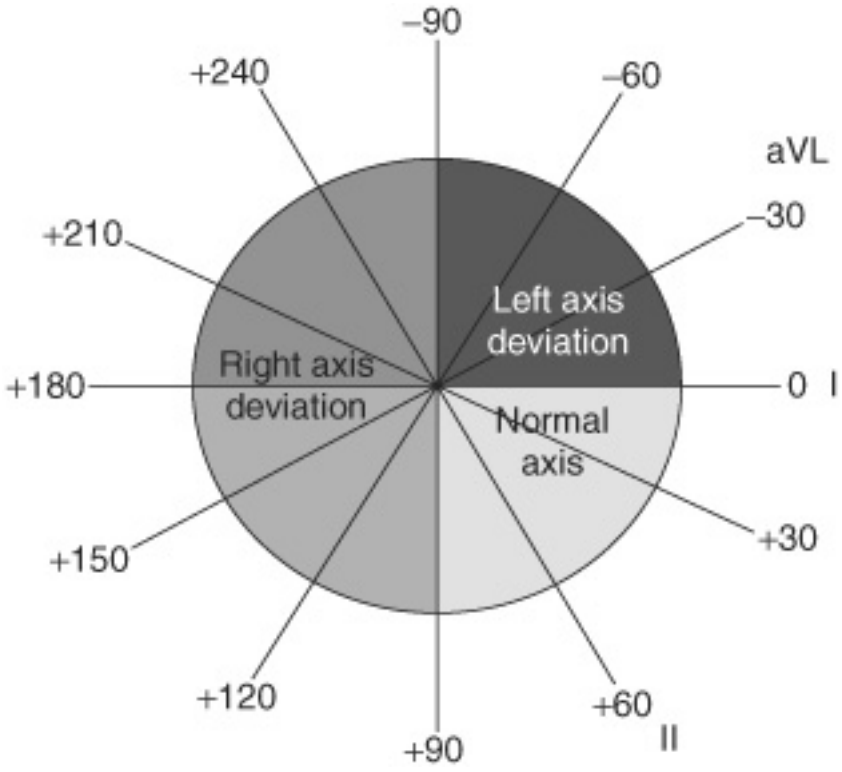


Figure 1-4

Step 4: Evaluate the intervals.

One large box = 0.20 seconds.

One small box = 0.04 seconds.

Normal measurements:

P wave < 0.11 seconds.

PR interval 0.12–0.2 seconds.

QRS interval < 0.12 seconds.

QT interval 0.33–0.47 seconds.

If PR < 0.12,**then** junctional rhythm or bypass tract.

If PR > 0.2,**then** atrioventricular (AV) block.

If QRS > 0.12,**then** either a left bundle branch block (LBBB), right bundle branch block (RBBB),**or** a nonspecific conduction delay.

Step 5: Check for hypertrophy.

Atrial hypertrophy

Right atrium:tall P waves in II, III, and aVF or V1or V2

Left atrium:notched P waves in limb leads

Ventricular hypertrophy

Left ventricular hypertrophy:height of S (mm) in V1+ height of R (mm) in V5> 35 mm

Right ventricular hypertrophy:height of R (mm)/height of S (mm) in V1> 1

Step 6: Look for ischemic changes:

ST elevation or depression.

T-wave inversion.

Q waves indicating old infarct.

Fluids and Electrolytes

FLUIDS

What percentage of body mass is water?

50 to 70%

In what two compartment is body water stored and what is the portion in each?

Intracellular ($^{2}/_{3}$); extracellular ($^{1}/_{3}$)

How is extracellular fluid separated?

Intracellular (¼); extravascular or interstitial (¾)

What percentage of body mass does intracellular water account for?

40%

What percentage of body mass does extracellular water account for?

20%

What percentage of body mass does blood account for?

About 7%

What physical examination signs can be used to assess volume status?

Skin turgor, mucous membranes, pulse, urine output, acute weight change

What are signs of hypovolemia?

Tachycardia, tachypnea, dry mucous membranes, decreased urine output, decreased blood pressure, decreased skin turgor

What is normal urine output in an adult?

30 cc/h

How do you calculate maintenance fluids per hour?

4/2/1 rule:

4 mL/kg (up to 10 kg); 2 mL/kg; (from 11 to 30 kg); 1 mL/kg >30 kg

How do you calculate maintenance fluids per day?

100/50/20 rule

100 mL/kg (up to 10 kg); 50 mL/kg (from 11 to 30 kg); 20 mL/kg (>30 kg)

What compromises each of the following IV fluids?

D5W

5% dextrose in water

D10W

10% dextrose in water

Normal saline (NS)

154 mEq Na, 154 mEq Cl

½ NS

77 mEq Na, 77 mEq Cl

¼ NS

39 mEq Na, 39 mEq Cl

Lactated ringers

130 mEq Na, 110 mEq Cl, 4 mEq K, 3 mEq Ca, 28 mEq lactate

What are the two most commonly used maintenance fluids?

D5½ NS or D5 ½ NS with 20 mEq K

What type of IV fluid should be given for fluid resuscitation?

NS or lactated ringers because they are isotonic

ELECTROLYTES

Hyperkalemia

What is the normal range for potassium?

3.5–5.0 mEq/L

What are the causes of hyperkalemia?

Increased load vs. decreased excretion:

Increased load:exogenous K+ ingestion, blood transfusion, tissue injury (rhabdomyolysis, burns), acidosis, hypoaldosteronism

Decreased excretion:renal failure, K+ sparing diuretics

What is pseudohyperkalemia?

Elevated K+ in a blood sample due to hemolysis

What are the signs and symptoms?

Muscle weakness, paresthesias, areflexia, bradycardia, respiratory failure, EKG changes

What are the characteristic EKG findings?

Peaked T-Waves

Prolonged PR interval, widening of QRS, P wave loss, U wave (Fig. 1-5)

Above what level are symptoms usually seen?

K+ > 6.5.

How is hyperkalemia treated?

1. Protect cells by increasing membrane threshold: calcium (cardioprotective)

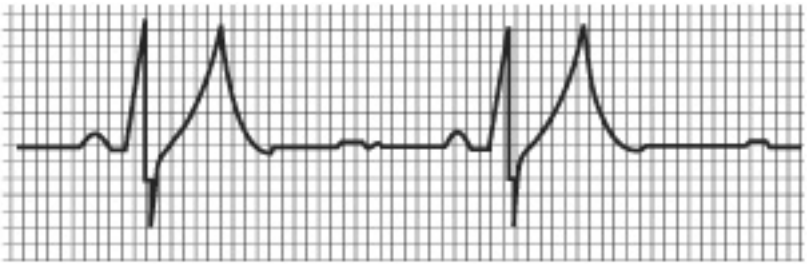


Figure 1-5 Peaked T-waves. (EKG used with permission of Dr. Henry Feldman fromhttp://students.med.nyu.edu/erclub/ekgguide.pdf)

2. Drive K+ into cells: sodium bicarbonate, insulin with glucose

3. Excrete K+: kayexalate (binds K+), furosemide, dialysis

What is the mnemonic for treatment of hyperkalemia?

C BIG K Drop

Calcium

Bicarbonate

Insulin

Glucose

Kayexalate

Dialysis

r

o

p

What acid-base disturbance can lead to hyperkalemia?

Acidosis

Hypokalemia

What are some causes of hypokalemia?

Vomiting, diarrhea, nasogastric (NG) tube suction, diuretic use (thiazides are a common culprit), insulin, amphotericin, hypomagnesemia

What are the signs and symptoms of hypokalemia?

Nausea, vomiting, weakness, paresthesias, hyporeflexia, ileus, digoxin sensitivity, v-tach, and EKG changes

What acid-base disturbance can cause hypokalemia?

Alkalosis

What are the characteristic EKG findings of hypokalemia?

T-wave depression, U waves

How is severe hypokalemia acutely treated?

IV KCl

What is the major side effect of IV potassium?

Burning sensation at IV site through which it is being administered

How can the burning be avoided when administering IV potassium?

Slow infusion usually not more than 10 mEq per hour

How can mild or chronic hypokalemia be treated?

Oral KCI supplementation or potassium-rich foods

What electrolyte level should be checked in a patient with hypokalemia?

Magnesium; hypomagnesemia can precipitate hypokalemia

What medication can be used to treat hypokalemia?

Potassium-sparing diuretic (e.g., spironolactone)

Hypercalcemia

What is the normal range for calcium?

9.0–10.6 (serum calcium)

What are the causes for hypercalcemia?

Mnemonics**CHIMPANZEES:**

Calcium supplementation

Hyperparathyroidism

Iatrogenic

Milk alkali syndrome

Paget disease

Addison disease

Neoplasm

Zollinger-Ellison syndrome

Excess vitamin A

Excess vitamin D

Sarcoidosis

What is a common iatrogenic cause of hypercalcemia?

Thiazide diuretics

What does the EKG look like?

Prolonged**PR** interval, short QT interval

What is the treatment?

IV hydration, loop diuretic (furosemide)

How is it treated in refractory cases?

Calcitonin, pamidronate, etidronate, glucocorticoids, plicamycin, dialysis

Hypocalcemia

What are the causes of hypocalcemia?

Renal failure, vitamin D deficiency, pancreatitis, diuretics, hypomagnesemia, parathyroidectomy

What can cause a pseudohypocalcemia?

Hypoalbuminemia

How can the true calcium level be calculated in hypoalbuminemia?

0.8 × (4 × albumin level) + calcium level = true calcium level

What are the two classic signs of hypocalcemia?

Trousseau and Chvostek signs

What is Trousseau sign?

Carpal spasm with arterial occlusion using a blood pressure cuff

What is Chvostek sign?

Facial spasm with tapping of the facial nerve

What are some other signs and symptoms of hypocalcemia?

Tetany, seizures, paresthesias, altered mental status, fatigue, weakness, EKG changes

What is the classic EKG finding with hypocalcemia?

Prolonged QT interval

What is the treatment for accurate hypocalcemia?

IV calcium gluconate

What is the treatment for chronic hypocalcemia?

Vitamin D with oral calcium tablets

Hypernatremia

What is the normal range for sodium?

135–145 mEq/L

What are the causes of hypernatremia?

Hypovolemia:decreased oral intake of water secondary to illness or altered mental status; increased water loss such as diuresis, vomiting, diarrhea, hyperaldosteronism

Hypervolemia:hypertonic fluid administration, excess ingestion of salt, Cushing syndrome, Conn syndrome

Isovolemia:diabetes insipidus, skin loss

How do you calculate water deficit in hypernatremia?

0.6 × weight (kg) × (measured Na/normal Na) - 1

What are the signs and symptoms of hypernatremia?

Seizure, coma, ataxia, lethargy, irritability, spasticity, edema

What is the treatment for hypernatremia?

Treatment is dependent on each of the following underlying causea of hypernatremia:

Hypovolemia: Replace fluid with isotonic saline. Replace ½ of water deficit in first 24 hours and ½ over the next 48–72 hours.

Hypervolemia: Loop diuretics to increase sodium excretion and fluid replacement with ½ NS.

Isovolemia: Fluid replacement with½ NS (½ water deficit in first 24 hours and ½ over next 48–72 hours). If patient has central diabetes insipidus, give vasopressin.

What is the risk of rapid correction of hypernatremia?

Cerebral edema

What is the maximum rate at which plasma osmolality can be corrected?

2 mOsm/kg/h

What is the maximum rate at which sodium concentration can be corrected safely?

1 mEq/L/h

Hyponatremia

What is the differential diagnosis of hyponatremia?

SeeFig. 1-6 .

What is pseudohyponatremia?

There is no true sodium deficit, but appears to be because the serum is occupied by lipids or protein.

What is factitious hyponatremia?

Normal total body sodium but decreased serum sodium because of an osmotic flow of water into serum secondary to excess glucose or mannitol in the serum.

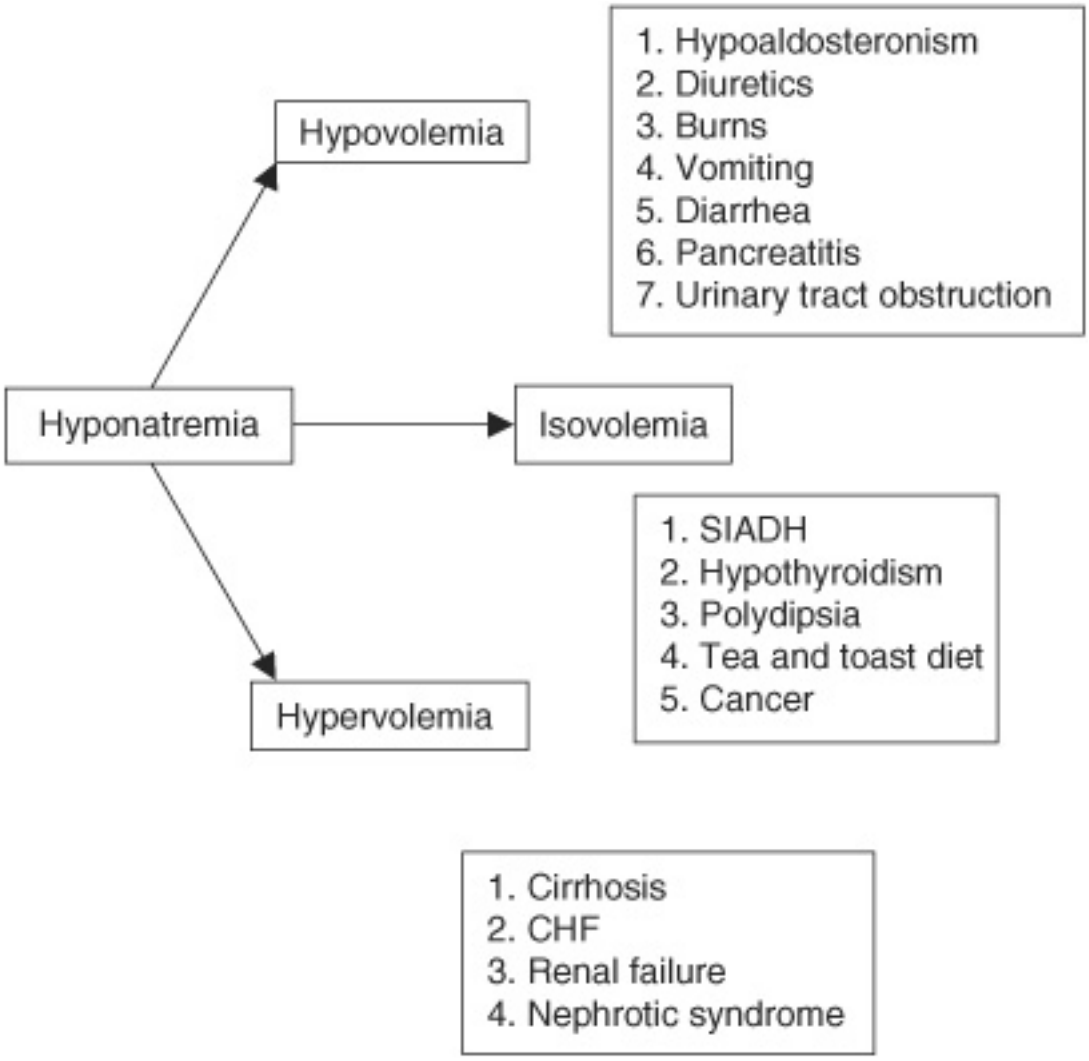


Figure 1-6

How is serum osmolality calculated?

2 × Na + blood urea nitrogen (BUN)/2.8 + glucose/18

How is hyponatremia evaluated?

SeeFig. 1-7 .

What are the signs and symptoms of hyponatremia?

Seizure, coma, lethargy, weakness, nausea, vomiting, ileus, altered mental status

What is the treatment for hypotonic hypovolemic hyponatremia?

Correct the underlying disorder and fluid resuscitation with IV normal saline (NS)

What is the treatment for hypotonic hypervolemic hyponatremia?

Fluid restriction. Diuretics like furosemide are helpful

What is the treatment for hypotonic isovolemic hyponatremia?

Treat the underlying cause. Fluid restriction.

What is the maximum speed at which hyponatremia can be safely corrected?

1 mEq/h

What can happen if sodium is corrected too quickly?

Central pontine myelinolysis

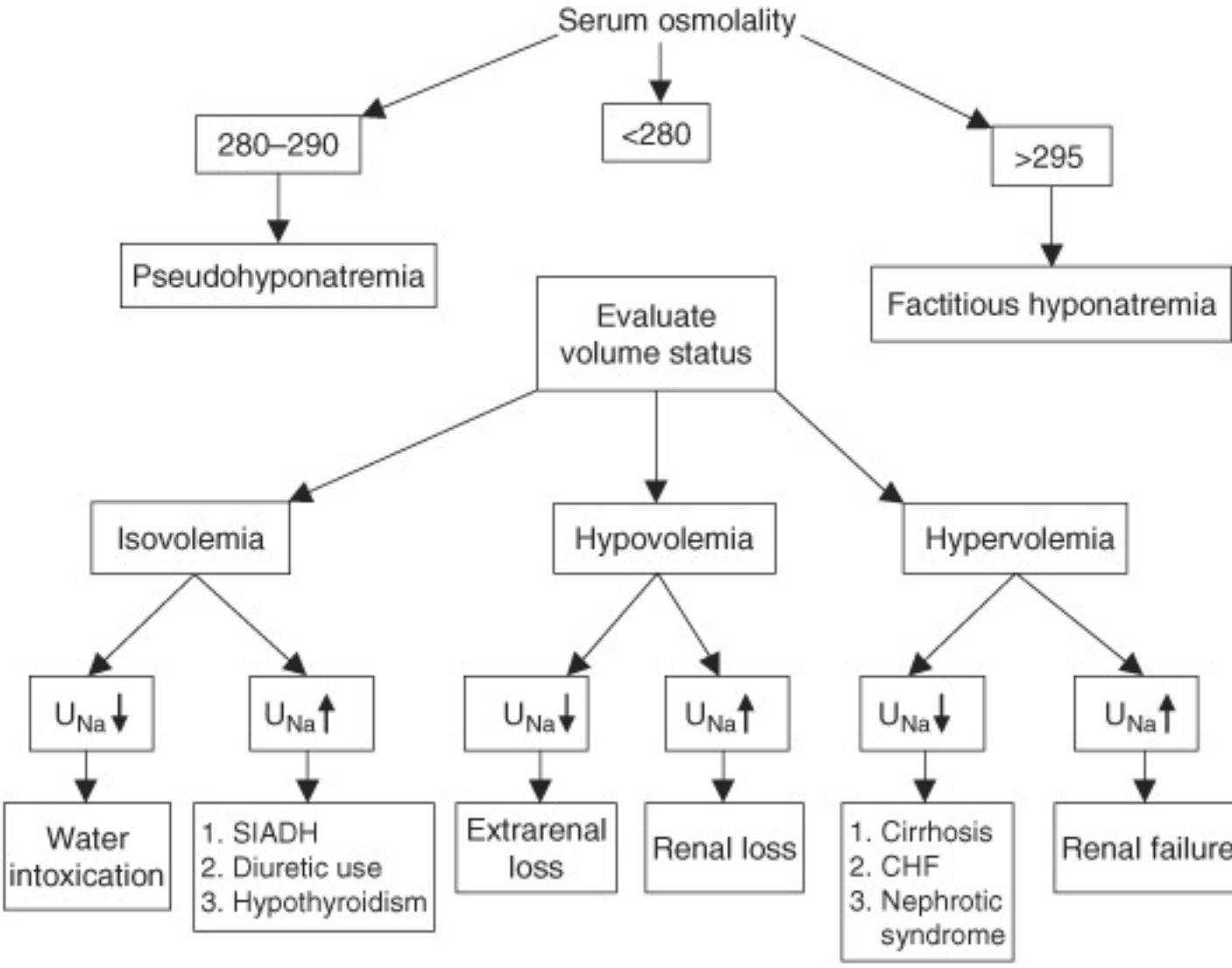


Figure 1-7

Hyperphosphatemia

What is the normal range of phosphate?

2.5–4.5 mg/dL

What are the most common causes of hyperphosphatemia?

Iatrogenic

What are other causes of hyperphosphatemia?

Hypoparathyroidism, hypocalcemia, renal failure, rhabdomyolysis

What are the signs and symptoms of hyperphosphatemia?

Heart block, ectopic soft tissue calcification

What is the treatment for hyperphosphatemia?

Aluminum hydroxide, a phosphate-binding agent; insulin and glucose; in severe cases—dialysis

Hypophosphatemia

What are the causes of hypophosphatemia?

Hyperparathyroidism, diuresis, decreased po intake, renal tubular acidosis, hypokalemia, hypomagnesemia, acetazolamide, glucose, and insulin

What are the signs and symptoms of hypophosphatemia?

Proximal muscle weakness, ataxia, rhabdomyolysis, paresthesias, hemolytic anemia, seizure

What is the treatment for hypophosphatemia?

Potassium phosphate of sodium phosphate supplementation

Hypermagnesemia

What is the normal range of magnesium?

1.5–2.5 mEq/L

What are the causes of hypermagnesemia?

Iatrogenic, renal failure, tumor lysis

What are the signs and symptoms of hypermagnesemia?

Weakness, fatigue, ? deep tendon reflexes, hypotension, paresthesias, coma

What is the treatment for hypermagnesemia?

Calcium gluconate and dialysis in refractory cases

Hypomagnesemia

What are the causes of hypomagnesemia?

Malabsorption, diarrhea, vomiting, NG tube suction, alcoholic patient, diuresis, hypokalemia or hypocalciuria induce hypomagnesemia, insulin and glucose administration

What are the signs and symptoms of hypomagnesemia?

Weakness, hyperreflexia, seizure, altered mental status, torsades de pointes, atrial fibrillation, hypokalemia and hypocalcemia refractory to replacement

What EKG changes would you expect to see in a patient with hypomagnesemia?

Prolonged QT and PR intervals, flattened T waves; may see torsades de pointes

What is the treatment for hypomagnesemia?

Magnesium sulfate IV

What other electrolyte abnormalities are related to hypomagnesemia?

Hypokalemia and hypocalcemia—if magnesium is low, these electrolyte abnormalities become refractory to treatment.

Name the electrolyte abnormality associated with the following EKG (Fig. 1-8).

Hyperkalemia with peaked T waves

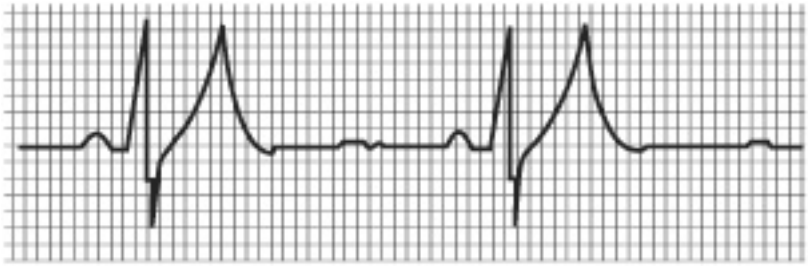


Figure 1-8 Peaked T-waves. (EKG used with permission of Dr. Henry Feldman fromhttp://students.med.nyu.edu/erclub/ekgguide.pdf)

Nutrition

Name the type of diet you would order for each of the following types of patients:

Patients who have no dietary restrictions	Regular diet
Patients with diabetes type I or II	Diabetic diet or American Diabetes Association (ADA) diet; be sure to specify the number of calories per day
Patients with renal failure or liver disorders	Protein-restricted diet; specify the amount of protein per day
Patients who do not have teeth or have difficulty with chewing and/or swallowing	Mechanical soft or pureed food

Patients with pancreatitis	npo (nothing by mouth)
Patients who are at risk for aspiration	npo
Patients with coronary artery disease	Low-fat diet
Patients who are being transitioned from npo to an oral diet	Clear liquids (includes clear broth, gelatin), then full liquids
Patients with syndrome of inappropriate antidiuretic hormone (SIADH)	Fluid-restricted diet; specify the amount of fluid per day

Normally, what is the daily protein requirement for an adult?

1 g/kg per 24 hours

Normally, what is the daily carbohydrate requirement for an adult?

35 kcal/kg per 24 hours

How many kilocalories (Kcal) in 1 gram of fat?

9 kcal

How many kcal in 1 gram of carbohydrate?

4 kcal

How many kcal in 1 gram of protein?

4 kcal

What lab test is used to determine chronic nutritional status?

Albumin, since the half-life is about 20 days

What lab test is used to determine acute nutritional change?

Prealbumin, since the half-life is about 3 days

Name the fat-soluble vitamins.

D, E, A, K (DEAK)

Where are the fat-soluble vitamins absorbed?

In the terminal ileum

Where is vitamin B12absorbed?

In the terminal ileum

What must bind B12in order for it to be absorbed?

Intrinsic factor

Where is intrinsic factor produced?

It is produced by the gastric parietal cells.

Name the effect on the body with each of the following deficiencies:

Vitamin B_{12} deficiency	Megaloblastic anemia
Zinc deficiency	Poor wound healing, dermatitis, alopecia
Vitamin C deficiency	Bleeding gums
Vitamin A deficiency	Poor wound healing
Vitamin K deficiency	Bleeding

What are the vitamin K-dependent clotting factors?

Factors 2, 7, 9, 10

What is TPN?

Total parenteral nutrition

What are the indications for TPN use?

npo for > 7 days

Pancreatitis

Anorexia

Enterocutaneous fistula

Ileus that is not resolving

Burn patients

Patients unable to take food by mouth

What are the three main components of TPN?

Amino acids, dextrose, fat

What percentage of TPN is fat?

10% (20% in the form of intralipid)

What percentage of TPN calories comes from dextrose?

50 to 70%

What percentage of total calories comes from fat?

30 to 50%

What percentage of total calories comes from amino acids (or protein)?

10 to 20%

How is basal energy expenditure (BEE) calculated in a male?

66 + (13.7 × weight [kg]) + (5 × height [cm])–(6.8 × age)

How is BEE calculated in a female?

65 + (9.6 × weight [kg]) + (1.8 × height [cm])–(4.7 × age)

What are the complications of TPN?

Fatty liver, acalculous cholecystitis, hyperosmolality, line infection, refeeding syndrome, cholestasis

What is refeeding syndrome?

Low potassium, phosphate, and magnesium after refeeding of a patient who was previously starving

What is PPN?

Partial parenteral nutrition

When would PPN be used?

In patients who can tolerate some nutrition orally and only need some supplementation

A patient who becomes jaundiced while on TPN or PPN most likely has what condition?

Cholestasis

Blood Products and Transfusions

What blood products are measured when checking a complete blood count (CBC)?

White blood cells, hemoglobin, hematocrit, platelets, red blood cells

Name the blood products described below:

Blood product that contains no platelets or clotting factors	Packed red blood cells (PRBC)
Contains red blood cell (RBC), white blood cells (WBC), plasma, platelets and can be used for an acute, heavy bleed	Whole blood
Used to replace clotting factors	Fresh frozen plasma (FFP)
Contains von Willebrand factor, factors VIII and XIII, and fibrinogen. Used in hemophilia A, fibrinogen deficiency, and von Willebrand disease	Cryoprecipitate
Used to replace low platelets	Platelets

Name the blood tests described below:

Tests the intrinsic coagulation pathway	Partial prothrombin time (PPT)
Tests the extrinsic coagulation pathway	Prothrombin time (PT)
Measures PT	International normalized ratio (INR)

What is the problem with using FFP in patients on Coumadin?

It will reverse the anticoagulation quickly; however, it is more difficult to get the patient back to a therapeutic level.

What else can be used to reverse anticoagulation in a patient on Coumadin?

Vitamin K

Which foods have vitamin K?

Leafy green vegetables

What is involved in normal coagulation?

Damage to the endothelium leads to platelet binding and aggregation, coagulation factors then help lay down fibrin to form and stabilize a clot.

What is a therapeutic INR level for a patient on Coumadin?

INR 2–3

When should you consider a blood transfusion in a normal, healthy patient?

When hemoglobin drops below**8**

When should you consider a blood transfusion in a patient with coronary artery disease?

When hemoglobin drops below**10**

How does 1 U of PRBC affect the hemoglobin and hematocrit?

1 U should increase the hemoglobin by 1g/dL and hematocrit by 3%.

What is the formula for converting hematocrit to hemoglobin?

Hematocrit ÷ 3 = hemoglobin.

What study should be ordered if you are considering transfusing a patient?

Type and cross

What is a type and cross?

The patient's RBCs are cross-matched to available donor blood for transfusion. In this process, the patient's serum is checked for preformed antibodies to the RBCs of the donor.

What is a type and screen?

The patient’s blood type and Rh antigen are determined and the donor’s blood is screened for common antibodies.

What blood type is considered the universal donor?

O

What blood type is considered the universal recipient?

AB

What are the two main complications of a blood transfusion that a patient should know about before consenting for a transfusion?

Possibility of acquiring an infectious disease and possibility of rejection

What is the most common cause of rejection during a blood transfusion?

Clerical error leading to ABO incompatibility.

What are the most common signs and symptoms seen of an acute rejection?

Fever, chills, tachycardia, shock, acute renal failure

What is the treatment of a rejection to a blood transfusion?

Stop the transfusion!IV fluid resuscitation and make sure the patient has good urine output. If urine output is not sufficient, furosemide (Lasix) can be administered.

After a transfusion, what would you expect to happen to the ionized calcium in the

blood?

It decreases because of the preservatives used to store blood.

What is the most common transfusion-related infection?

Hepatitis

What is the risk of infection with hepatitis B or hepatitis C from a blood transfusion?

1 in 50,000 U of blood

What is the risk of getting infected with human immunodeficiency virus (HIV) from a blood transfusion?

1 in 300,000 U of blood

How long can PRBCs be stored?

6 weeks

What is the life span of a RBC?

120 days

What is thrombocytopenia?

Platelet count < 200,000

At what platelet count is there a risk for spontaneous intercranial bleeding?

Platelet count < 20,000

In an actively bleeding patient or a patient who is preoperative, what should the platelet count be?

A minimum of 50,000

In what cases of thrombocytopenia are platelets not transfused?

Do not transfuse platelets in patients with thrombotic thrombocytopenic purpura (TTP), idiopathic thrombocytopenic purpura (ITP) and disseminated intravascular coagulation (DIC), because platelet transfusion will only perpetuate the problem. Platelets are only transfused if the patient is actively bleeding.

CHAPTER 2

Cardiology

HYPERTENSION

How is hypertension defined?

Prehypertension: 120–139/80–89

Stage 1: 140–159/90–99

Stage 2: >160/>100

What is the most common cause of hypertension?

90% is essential or idiopathic.

What are some secondary causes of hypertension?

1. Cardiovascular: coarctation of aorta, aortic regurgitation

2. Renal: renal artery stenosis, polycystic kidney disease, glomerular disease

3. Endocrine: eclampsia, pheochromocytoma, primary hyperaldosteronism (Cushing and Conn)

Define hypertensive urgency.

Systolic > 180, diastolic > 120**with*no* end organ failure**

Define hypertensive emergency.

Also known as malignant hypertension; systolic > 180, diastolic > 120;***with*end organ failure**

What are the signs and symptoms of malignant hypertension?

1. Change in mental status

2. Papilledema

3. Anuria (sign of renal failure)

4. Heart failure

5. New-onset neurological change

What is the treatment for malignant hypertension?

Nitroprusside or nitroglycerine

In malignant hypertension, by how much should the blood pressure be reduced in 1 hour?

Do notdecrease by more than ¼ within 2–6 hours, otherwise the patient will be at risk for a stroke.

How do you calculate mean arterial pressure (MAP)?

(2 × diastolic + systolic)/3

What hypertensive treatment is favorable for a patient with each of the following comorbidities?

1. No comorbidities	1. If they fail lifestyle modification for 6 months, add a thiazide diuretic.
2. Post-myocardial infarction (MI)	2. Beta-blocker and angiotensin -converting enzyme (ACE) inhibitor
3. Benign prostatic hyperplasia (BPH)	3. Alpha-blocker
4. Congestive heart failure (CHF)	4. ACE inhibitor
5. Osteoporosis	5. Thiazide diuretics (do not excrete calcium)
6. Diabetes	6. ACE inhibitor
7. African American	7. Calcium channel blocker, diuretic

What are the relative contraindications for each of the following treatments?

1. **ACE inhibitors**	1. Teratogenic in pregnancy, in renal artery stenosis, renal failure
2. **Beta-blocker**	2. Chronic obstructive pulmonary disease (COPD), asthma, diabetes, hyperkalemia
3. **Short-acting calcium channel blockers**	3. Prior MI, CHF
4. **Potassium (K)-sparing diuretics**	4. Renal failure (can lead to hyperkalemia)
5. **Thiazide diuretics**	5. Diabetes (can cause hyperglycemia)

Which two drugs are proven to reduce morbidity and mortality?

Beta-blockers and thiazide diuretics

HYPERLIPIDEMIA

When should a patient with no family history be screened for hyperlipidemia?

Men age 35; women age 45

How often should a patient with previously normal lipids be rechecked for hyperlipidemia?

Every 5 years

What should the low-density lipoprotein (LDL) level be in a patient with no or one risk factor(s) for coronary artery disease (CAD)?

<160

What is the goal LDL for a patient with known CAD?

<100

What is the goal LDL for patient with no known CAD but with two or more risk factors?

<130

What is a protective factory in terms of hyperlipidemia?

High-density lipoprotein (HDL) >60

What is the mechanism for each of the following lipid-lowering agents?

1. Statins	1. 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inhibitors; ↓ LDL ↑ HDL
2. Nicotinic acid	2. Decreases lipolysis and prevents cholesterol synthesis by the liver; ↓ LDL ↑ HDL
3. Fibrates	3. Reduces triglycerides in very low-density lipoprotein (VLDL) and chylomicrons; ↑ HDL ↓ triglycerides
4. Bile acid sequestrants	4. Bind bile acids in the gut; ↓ LDL

What side effect of statins should you screen for?

Elevation in alanine aminotransferase (ALT)

CORONARY ARTERY DISEASE

What is CAD?

Atherosclerosis leading to angina or MI

What are the risk factors for CAD?

Major

Family (hx) (MI before age 55 in a male or 65 in a female)

Diabetes

Smoking

Hyperlipidemia

Hypertension

Age: male >45, female >55

Minor

Obesity

Male sex

Postmenopausal female

Elevated plasma homocysteine

What lifelong treatment has been shown to decrease mortality in a patient with CAD?

Aspirin, beta-blocker, statin, ACE inhibitor

What is stable angina?

Substernal chest pain (may radiate as well to arms, jaw, and so forth) due to ischemia that occurs both predictably and reproducibly at a certain level of exertion and relieved with rest/nitrates

What are some classic electrocardiographic (EKG) findings in a patient with angina?

ST depression or T-wave inversion

What is the treatment for acute angina?

Sublingual nitroglycerin up to three doses

What is the long-term treatment for angina?

Nitrates, aspirin, beta-blocker, statin, smoking cessation

What is unstable angina?

Angina occurring more frequently, unrelieved by nitroglycerin, or occurs at rest

How do you evaluate a patient with unstable angina?

EKG, cardiac enzymes, and, once stable, a cardiac stress test to risk stratify; angiography may be necessary

How should a patient with unstable angina be treated?

Hospitalization and treatment with nitroglycerin, aspirin, beta-blocker, ACE inhibitor, statin, heparin drip or Lovenox while on a cardiac monitor

When is a coronary artery bypass graft (CABG) indicated?

Failure of medical treatment with severe three-vessel disease; multiple vessel disease in a diabetic patient; or >50% stenosis of the left main artery; proximal significant left anterior descending (LAD) coronary artery stenosis with left ventricular (LV) dysfunction

What is Prinzmetal angina?

Angina due to coronary vasospasm that is usually nonexertional but can be exertional. Angiography is normal in these patients.

What is an MI?

Myocardial necrosis caused by ischemia

What are the classic symptoms of an MI?

Crushing, substernal chest pain described as chest tightness or pressure. It can radiate to the left arm, neck, or jaw and can be associated with concomitant diaphoresis, shortness of breath, nausea, and vomiting.

What patients can present with nonclassic symptoms?

Diabetics and the elderly

What are the classic EKG changes associated with an MI?

ST elevation or depression, new left bundle branch block (LBBB), T-wave changes (Fig. 2-1aand2-1b)

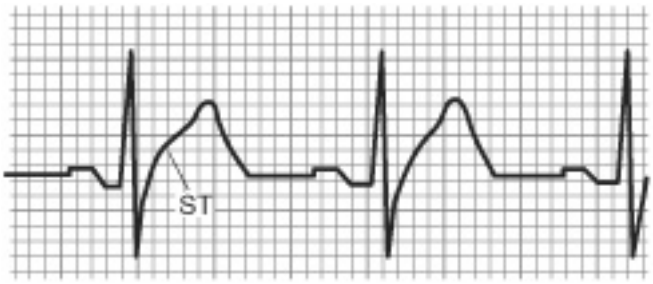


Figure 2-1a

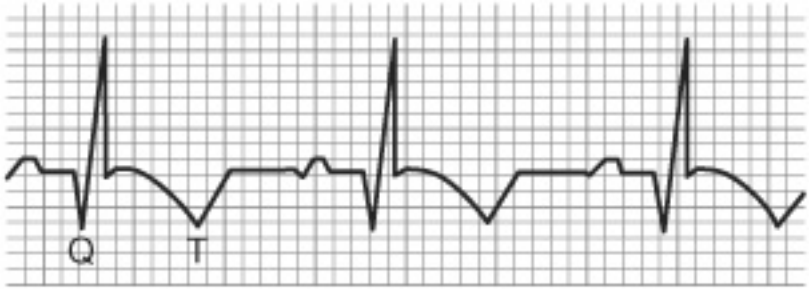


Figure 2-1b

What are the three different cardiac enzymes tested in a patient with chest pain?

Troponin, creatine kinase (CPK), and CK-MB (creatine kinase-MB)

How do the three cardiac enzymes differ in terms of elapsed time since an MI?

Cardiac Enzyme	Troponin	CPK	CK-MB
Rises	2–6 hours after injury	4–6 hours	Within 3–4 hours
Peaks	12–16 hours	24 hours	Varies
Stays elevated for	5–10 days	2–3 days	1–2 days

How often should the cardiac enzymes be done?

Repeat every 6–8 hours for a 24-hour period

What is the mnemonic for emergent treatment of an MI?

Be MONA:

Beta-blocker

Morphine

Oxygen

Nitroglycerin

Aspirin

When is thrombolysis indicated?

In an ST-elevation MI, within 12 hours of onset of chest pain

What are contraindications to thrombolytics?

Previous cerebral hemorrhage, known cerebral aneurysm or arterio-venous malformation (AVM), known intracranial neoplasm, ischemic stroke in the last 3 months, aortic dissection, active bleeding, significant closed head or facial trauma

What is a contraindication to the use of streptokinase specifically?

Cannot be used more than once in a 6-month period because of its immunogenicity

What are some possible post-MI complications?

New arrhythmias; Dressler syndrome; papillary muscle rupture; thromboembolism; CHF,

ventricular septal defect (VSD), myocardial rupture

What is Dressler syndrome?

An autoimmune process with the features of fever; pericarditis; elevated erythrocyte sedimentation rate (ESR) that occurs 2–4 weeks after an MI

What is the treatment of Dressler syndrome?

Nonsteroidal anti-inflammatory drugs (NSAIDs) and aspirin

What physical examination finding is indicative of a papillary muscle rupture?

New mitral regurgitation

ARRHYTHMIAS

Define each of the following types of heart block.

1. **First-degree**	1. **PR interval in > 0.2 seconds** but all atrial impulses are conducted
2. **Second-degree Mobitz type I**	2. Also known as Wenckebach; **PR intervals progressively increase until a beat is dropped**
3. **Second-degree Mobitz type II**	3. **PR intervals are fixed with intermittently dropped QRS complexes**
4. **Third-degree**	4. Also known as complete heart block; dissociation between atrial and ventricular activity; no relationship between P waves and QRS intervals

What is the treatment for each of the following types of heart block?

1. **First-degree**	1. No treatment required
2. **Second-degree Mobitz type I**	2. If caused by a drug, stop offending drug; may need a pacemaker if bradycardic
3. **Second-degree Mobitz type II**	3. Pacemaker, because it can progress to third degree block
4. **Third-degree**	4. Pacemaker

Name some medications that can lead to second-degree heart block?

Digoxin, beta-blockers, calcium channel blockers

What it the most common chronic arrhythmia?

Atrial fibrillation

What is Atrial fibrillation?

Irregularly irregular rhythm caused by disorganized electric activity of the atrium (Fig. 2-2)

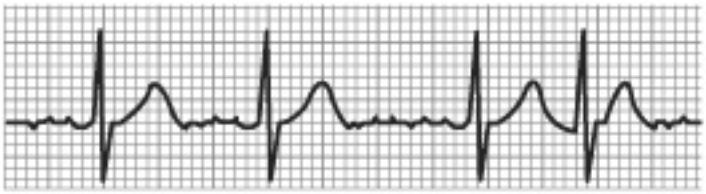


Figure 2-2

What is the mnemonic for some etiologies of Atrial fibrillation?

PIRATES: Pulmonary disease

Ischemia

Rheumatic heart disease

Anemia

Thyroid

Ethanol

Surgery, sepsis

What are some symptoms that patients with Atrial fibrillation complain of?

Fatigue, light-headedness, palpitations

What is the major complication of Atrial fibrillation if left untreated?

Embolization which often can lead to stroke

What are the treatments of Atrial fibrillation?

Rate controlwith beta-blocker, calcium channel blocker (diltiazem), digoxin

Antiarrhythmic agents(if failure to rate con-trol or symptomatic despite rate control)

Anticoagulationwith coumadin

In an unstable patient, synchronized cardioversion

What is atrial flutter?

Macroreentrant arrhythmia; atrial rates are typically between approximately 240 and 400

beats/min

What is the classic EKG pattern described in atrial flutter?

“Saw tooth” (Fig. 2-3)

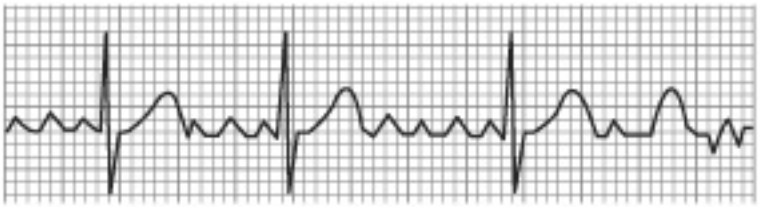


Figure 2-3

What is multifocal atrial tachycardia (MAT)?

Irregularly irregular rhythm caused by at least three sites of competing atrial activity.

What is the classic EKG finding in MAT?

At least three different P-wave morphologies (Fig. 2-4)

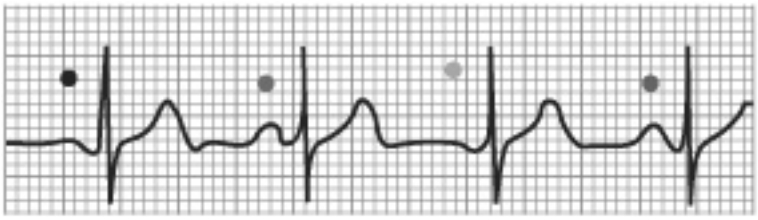


Figure 2-4

What medical condition is associated with MAT?

COPD

What is the treatment for MAT?

Treat the underlying cause.

What is a premature ventricular contraction (PVC)?

Ectopic beats of ventricular origin.

What is the typical EKG finding in PVCs?

Wide QRS with no P wave

What is ventricular tachycardia (VT)?

More than three consecutive PVCs; sustained VT must last >30 seconds

What is the possible complication of VT?

Ventricular fibrillation or cardiac arrest/hemodynamic collapse

What is the treatment for VT?

If the patient is asymptomatic and not hypotensive, treat with lidocaine or amiodarone; if the patient is hypotensive or pulseless, treatment is defibrillation.

What is ventricular fibrillation?

Disorganized electric activity of the ventricle (Fig. 2-5)

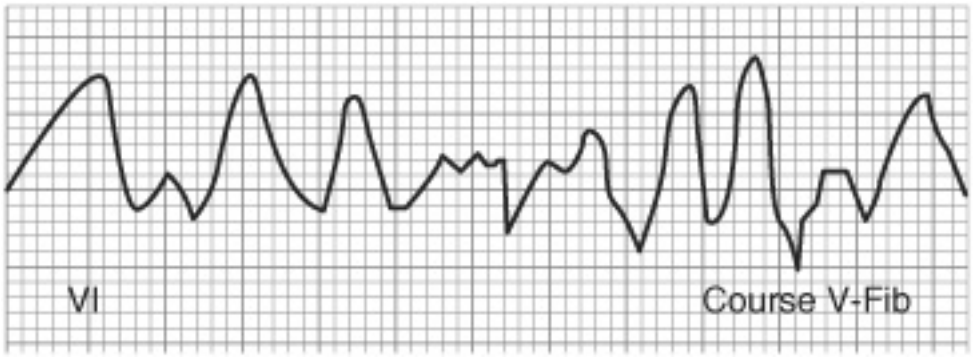


Figure 2-5

What is the treatment for ventricular fibrillation?

Emergent cardioversion

What is torsades de pointes?

Prolonged VT with rotation around the axis in a patient with a prolonged QT interval at baseline

What are the underlying causes of torsades de pointes?

Quinine, procainamide, intracranial bleed, tricyclics, phenothiazines, intracranial bleed, electrolyte abnormalities

What are the classic EKG findings in Wolff-Parkinson-White (WPW) syndrome?

“Delta” waves and short PR interval

What medications are contraindicated in WPW?

ABCD: adenosine, beta-blocker, calcium channel blocker, digoxin

How does digoxin toxicity present?

Supraventricular tachycardia (SVT) with atrioventricular (AV) block and yellow vision

CONGESTIVE HEART FAILURE

What is the definition of CHF?

Inability of the heart to pump enough blood to meet systemic demands. Left-sided heart failure (LHF) leads to pulmonary vascular congestion while right-sided heart failure (RHF) causes systemic venous congestion.

What are the underlying causes of CHF?

Myocardial ischemia, anemia, pulmonary embolism, endocarditis, cardiomyopathy, hypertension, pericarditis, cardiac dysrhythmias, thyrotoxicosis

What is the most common cause of RHF?

Left heart failure

What are the symptoms of RHF?

Hepatomegaly, jugular venous distension (JVD), ascites

What are the symptoms of LHF?

Orthopnea, S3gallop, paroxysmal nocturnal dyspnea, cough, diaphoresis, rales

What is classically seen on a chest x-ray (CXR) in a patient with CHF?

Pulmonary vascular congestion, enlarged heart

What are the treatments for CHF?

ACE inhibitor, diuretics, digoxin, calcium channel blocker, sodium-restricted diet, beta-blockers (but not in acute CHF)

What is second-line treatment for CHF?

Isosorbide and hydralazine if the patient can't tolerate an ACE inhibitor

Which medications have been shown to decrease mortality in CHF?

ACE inhibitor, beta-blocker, spironolactone, hydralazine + isosorbide (although less than ACE inhibitor)

Name the drug used in CHF with the following features?

1. **Reduced afterload**	1. ACE inhibitor
2. **Acute fluid retention**	2. Loop diuretics
3. **Positive inotropes**	3. Dobutamine, Dopamine, Digitalis

VALVULAR HEART DISEASES

What does the S1sound represent in a heart beat?

Closure of mitral and tricuspid valves

What does the S2sound represent in a heart beat?

Closure of the aortic and pulmonic valves

What is the most common valvular heart disease found in young women?

Mitral valve prolapse

What is the underlying etiology of mitral valve prolapse?

Idiopathic; genetic transfer via autosomal dominant gene; ischemic heart disease; Marfan; myxomatous degeneration of the mitral valve

What is the pathognomonic murmur heard in a mitral valve prolapsed?

Late systolic murmur and a mid-systolic click

Where is the murmur most audible?

Apex

What is the treatment for mitral valve prolapse?

No treatment is necessary.

What are the underlying etiologies of mitral stenosis?

Most commonly due to**rheumatic heart disease**

In what sex does mitral stenosis predominate?

Females

What are the signs and symptoms of mitral stenosis?

Dyspnea, orthopnea, cough, rales, hoarse voice, atrial fibrillation, hemoptysis

What is the underlying cause leading to the symptoms found in mitral stenosis?

Flow is decreased behind the mitral valve leading to left atrial enlargement and eventually heart failure.

Name the valvular heart diseases that cause a systolic ejection murmur.

Pulmonary stenosis, aortic stenosis

Name the valvular heart diseases that cause a pansystolic murmur.

Mitral regurgitation, tricuspid regurgitation

Name the valvular heart diseases that cause a diastolic murmur.

Aortic regurgitation

Name the valvular heart disease associated with each of the following.

1. **Systolic crescendo-decrescendo murmur at the second right intercostal space, which radiates to carotids**	1. Aortic stenosis
2. **Mid-diastolic murmur with an opening snap and rumble best heard at the left sterna border**	2. Mitral stenosis
3. **Holosystolic murmur that radiates to the axilla**	3. Mitral regurgitation
4. **High-pitched decrescendo diastolic murmur, louder if leading forward**	4. Aortic regurgitation
5. **Diastolic murmur louder with inspiration**	5. Tricuspid stenosis
6. **Holosystolic murmur at the left lower sternal border**	6. Tricuspid regurgitation
7. **Late-systolic murmur with mid-systolic click**	7. Mitral valve prolapse

CARDIOMYOPATHY

What are the three categories of cardiomyopathy?

Dilated, hypertrophic, and restrictive

What is the mnemonic for some etiologies of a dilated cardiomyopathy?

Alcohol abuse

Beriberi

Cocaine,**C** hagas disease,**C** oxsackie B

Doxorubicin

Idiopathic,**I** schemic,**I** nfectious

Name the type of cardiomyopathy associated with each of the following descriptions.

Symptoms of CHF, S_3 heart sound, enlarged balloon-like heart, atrial fibrillation, mitral regurgitation, systolic dysfunction	Dilated
Fifty percent of cases are genetically inherited via an autosomal dominant trait	Hypertrophic
Diastolic dysfunction as a result of ventricular enlargement and thickened septum, and systolic dysfunction as a result of LV outflow obstruction	Hypertrophic
Caused by radiation-induced fibrosis, endomyocardial fibrosis, amyloidosis, sarcoidosis, glycogen storage diseases	Restrictive
Syncope with exertion	Hypertrophic
Most common cause of sudden death in young adults	Hypertrophic
Mitral regurgitation, S_4 heart sound, systolic ejection murmur, large boot-shaped heart	Hypertrophic
Systolic dysfunction and left ventricular dilation are necessary to make the diagnosis	Dilated
Similar to constrictive pericarditis	Restrictive
Treatment includes cessation of all alcohol use	Dilated
Symptoms relieved with beta-blockers or calcium channel blockers	Hypertrophic
Treated with ACE inhibitor, beta-blocker, CHF-directed therapies, diuretics	Dilated

ENDOCARDITIS

What is endocarditis?

Heart valve inflammation usually due to an infective cause

Name the most common causes of the following types of endocarditis:

Acute	Most commonly, *Staphylococcus aureus* (IVDA [intravenous drug abuse]); others : *Streptococcus pneumoniae, Neisseria gonorrhoeae*
Subacute	Most commonly, *Streptococcus viridans* (dental work);others: *Enterococcus, Staphylococcus*
Culture negative	**HACEK:** *Haemophilus influenzae* *Actinobacillus* *Cardiobacterium* *Eikenella* *Kingella*
Marantic	Previous rheumatic fever or cancer metastasis

What type of endocarditis is seen in systemic lupus erythematosus (SLE)?

Libman-Sacks endocarditis (LSE) caused by autoantibodies damaging heart valves

What are the signs and symptoms of endocarditis?

Fever, chills,**Janeway lesion, Roth spots, Osler nodes, splinter hemorrhages,** new murmur, conjunctival hemorrhages

What are Janeway lesions?

Dark hemorrhagic peripheral macules or nodules usually on palms and soles (Fig. 2-6)

What are Roth spots?

Retinal hemorrhages

What are Osler nodes?

Ouch! Painful nodules on fingers and toes (Fig. 2-7)

What are splinter hemorrhages?

Petechiae on fingernails (Fig. 2-8)

What is the most likely cause of right-sided endocarditis?

IVDA

What tests would you order to help diagnose endocarditis?

Three sets of blood cultures and an echocardiogram to look for vegetations

What valve is most commonly affected in endocarditis?

Mitral valve

What valve is most commonly affected in an IV drug user with infective endocarditis?

Tricuspid valve

What criteria are used to make the diagnosis of endocarditis?

Duke criteria

What are the major criteria?

1. Two positive blood cultures demonstrating the same organism

2. Positive echocardiogram

What are the minor criteria?

1. Predisposing condition such as a valvular heart abnormality, hypertrophic cardiomyopathy, congenital heart disease

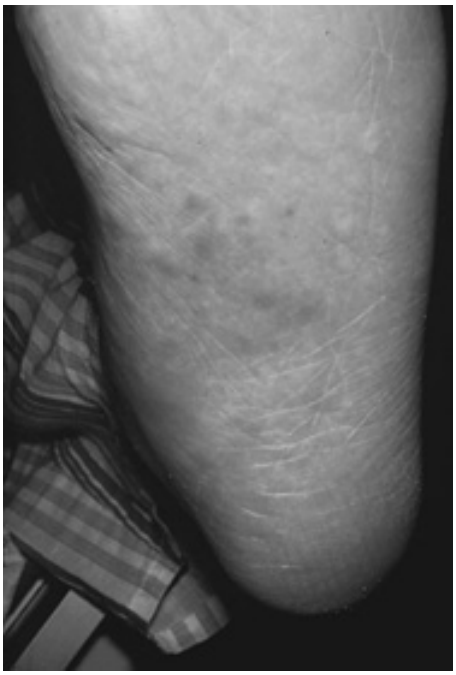


Figure 2-6 Janeway lesions. (Courtesy of the Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, Texas.

Reproduced, with permission, from Knoop KJ, Stack LB, Storrow AB:*Atlas of Emergency Medicine* . New York: McGraw-Hill, 1997: 348.)

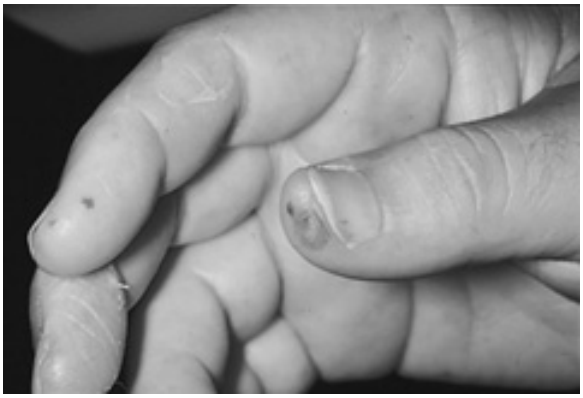


Figure 2-7 Osler nodes. (Reproduced, with permission, from Knoop KJ, Stack LB, Storrow AB:*Atlas of Emergency Medicine* . New York: McGraw-Hill, 1997: 349.)

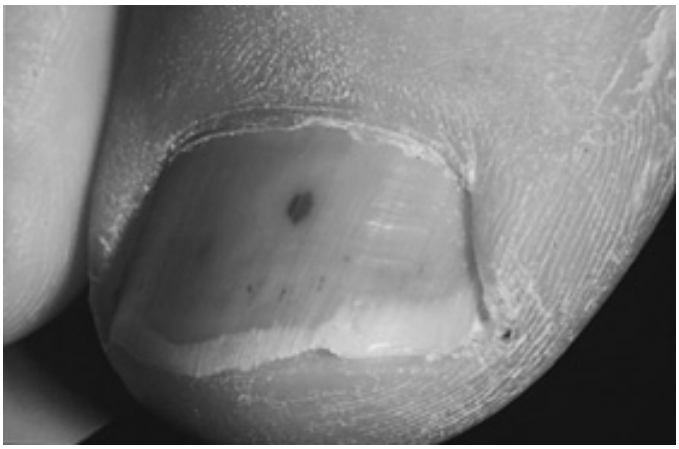


Figure 2-8 Splinter hemorrage. (Reproduced, with permission, from Knoop KJ, Stack LB, Storrow AB:*Atlas of Emergency Medicine* . New York: McGraw-Hill, 1997: 349.)

2. Documented temperature >38°C

3. Signs of embolic disease such as Janeway lesions, pulmonary emboli, cerebral emboli, hepatic or splenic emboli

4. Immunologic signs such as Roth spot, Janeway lesion

5. One positive blood culture

Before an organism is isolated and antibiotics can be tailored, what antibiotics should be initiated in a patient suspected to have endocarditis?

Aminoglycoside and a beta-lactam

How long should a patient with endocarditis be treated with antibiotics?

4–6 weeks

What antibiotic prophylaxis should be given to patients at risk for endocarditis?

Amoxicillin before dental procedures

RHEUMATIC FEVER

What infection causes rheumatic fever?

Group A streptococcal pharyngitis

Why does this infection cause rheumatic heart disease?

The antistreptococcal antibodies react with cardiac antigen.

What valve is most commonly affected in rheumatic heart disease?

Mitral valve

What serologic test could be used to confirm a prior streptococcal infection?

A positive antistreptolysino (ASO) antibody titer

What is the mnemonic for the five major criteria for rheumatic heart disease?

Jonescriteria

Joints (migratory polyarthritis)

Carditis (endocarditis, pericarditis, myocarditis)

Nodules (subcutaneous)

Erythema marginatum (serpiginous rash)

Sydenham chorea

What are the minor criteria for rheumatic heart disease?

PR-interval prolongation

Fever

Elevated ESR

Arthralgias

How should streptococcal pharyngitis be treated to prevent rheumatic heart disease?

Penicillin

PERICARDITIS

What is pericarditis?

Inflammation of the pericardium

What are some causes of pericarditis?

Infectious: viral, bacterial, fungal

Autoimmune: rheumatoid arthritis, SLE, scleroderma

Drugs HIP: hydralazine, isoniazid, procainamide (these are the same drugs that can lead to SLE-like reaction); radiation therapy

Trauma

Post-MI

Metastatic cancer

Uremia

What is pericarditis that occurs 2–4 weeks post-MI called?

Dressler syndrome

What are the classic symptoms of pericarditis?

Pleuritic chest pain that is relieved with sitting up and leaning forward

What is the pathognomonic physical exam finding of pericarditis?

Pericardial friction rub on auscultation of the heart during expiration

What are the classic EKG findings associated with pericarditis?

Diffuse ST elevations and PR depressions (usually in all or almost all leads) (Fig. 2-9)

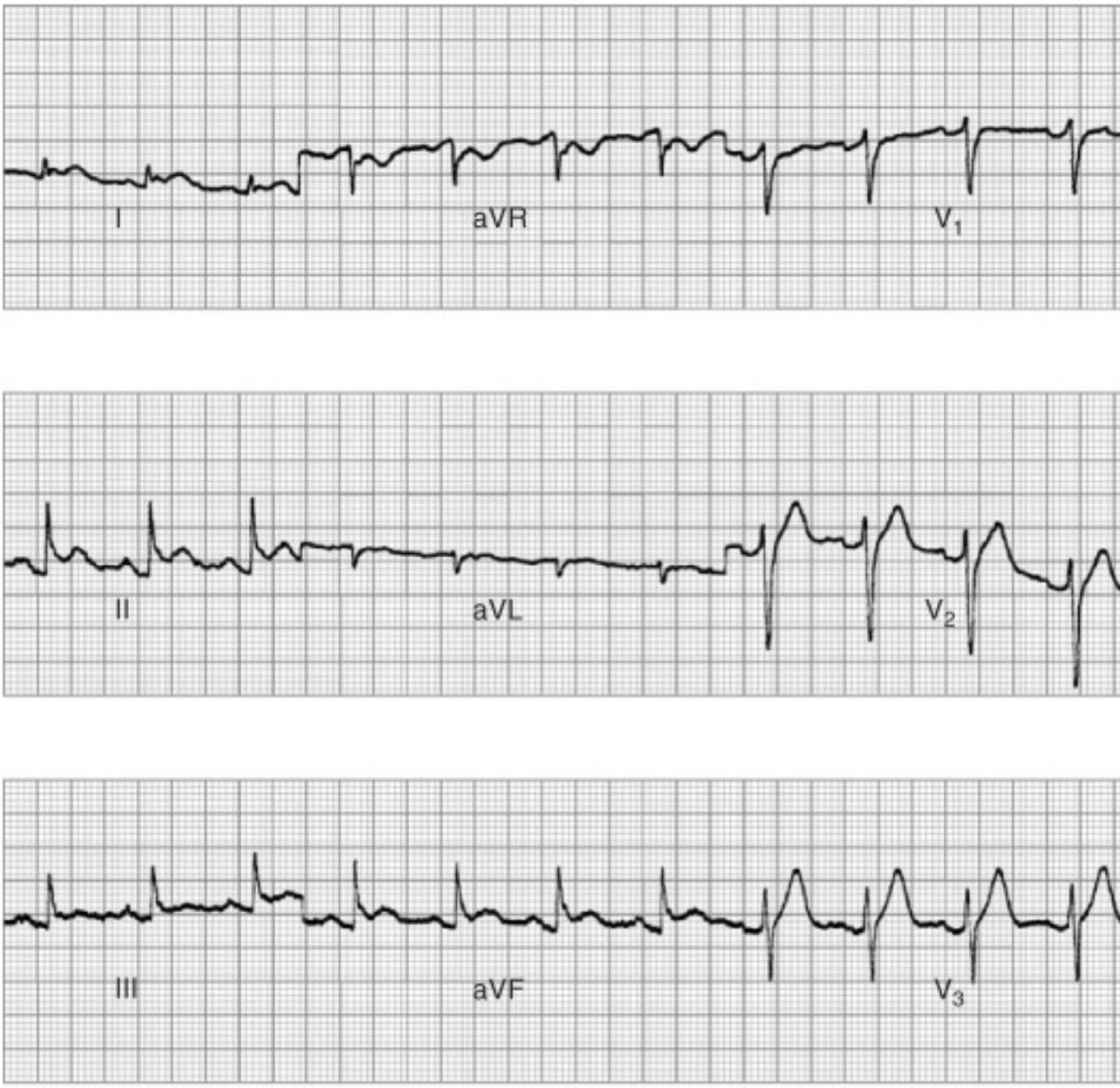


Figure 2-9 Classic EKG finding assosiated with pericardits. (Reproduced, with permission, from Stead LG et al:*First Aid for the Medicine Clerkship.* 2nd Ed. New York: McGraw-Hill, 2006:33. [Figure 2.1.4])

How can the diagnosis of pericarditis be confirmed?

Pericarditis is a clinical diagnosis, but an echocardiogram may show a pericardial effusion.

How is pericarditis treated?

The underlying cause should be addressed. NSAIDs to decrease inflammation; antibiotics for bacterial causes; steroids for autoimmune etiology; pericardiocentesis would be necessary for a large pericardial effusion

MYOCARDITIS

What is myocarditis?

Inflammation of the heart muscle

What is the most common viral cause of myocarditis?

Enterovirus infection (eg, Coxsackie B)

What are the causes of myocarditis?

Viral: Coxsackie A and B, HIV, EBV, HBV (hepatitis B), cytomegalovirus (CMV)

Bacterial: rheumatic fever, Lyme disease, meningococcus, mycoplasma

Parasitic: Chagas disease, toxoplasmosis, trichinella

Autoimmune: SLE, Kawasaki disease

Drugs

What are the signs and symptoms of myocarditis?

Precordial chest pain with signs of CHF

What does the EKG look like in a patient with myocarditis?

Nonspecific ST changes, dysrhythmias

How can a definitive diagnosis of myocarditis be made?

Myocardial biopsy

What is the treatment for myocarditis?

Treat CHF symptoms, dysrhythmias, and the underlying etiology. Steroids are contraindicated. In some cases, intravenous immunoglobulin (IVIG) is beneficial.

CARDIAC TAMPONADE

What is cardiac tamponade?

Pericardial fluid accumulation that causes impaired cardiac filling and thus leads to decreased cardiac output

What is Beck's triad?

Symptoms seen in cardiac tamponade:

1. Hypotension

2. Distant heart sounds

3. JVD

What are some other classic symptoms of cardiac tamponade?

Dyspnea, tachycardia, pulsus paradoxus

What is pulsus paradoxus?

> 10 mm Hg fall in blood pressure during inspiration

What is seen on EKG in a patient with pulsus paradoxus?

Electrical alternans—a beat-to-beat change in the height of the QRS complex

What study can help confirm the diagnosis of cardiac tamponade?

Echocardiogram will show a large pericardial effusion.

What is the treatment for pericardial tamponade?

Pericardiocentesis vs. pericardial window. IV fluids should also be given for volume expansion.

CHAPTER 3

Pulmonology

HYPOXIA

What is the mnemonic for the mechanisms of hypoxia?

CIRCULAR:

Circulatory

Increased oxygen requirement

Respiratory

Carbon monoxide poisoning

Underutilization

Low fraction of inspired oxygen (FIO2)

Anemia

Right-to-left shunt

What are the important respiratory causes of hypoxia?

Hypoventilation due to a decreased respiratory rate, decreased vital capacity, or ventilation/perfusion ratio (V/Q) mismatch

How can respiratory hypoxia be improved?

Supplemental oxygen and by treating the underlying cause

What is a common underlying cause of decreased respiratory rate?

Drugs: opiates

What are common underlying causes of increased respiratory rate?

Infection

Trauma

What are some reasons for decreased vital capacity?

Underlying lung disease, obstructive sleep apnea, deformities of the chest wall such as in severe scoliosis, muscle weakness

When is low FIO2mostly a problem?

High altitudes or closed spaces with no fresh air or fire

How does diffusion impairment cause hypoxia?

With circulatory impairment such as in heart failure or anemia, there is poor perfusion and, therefore, decreased blood transit time in the lungs causing decreased diffusion. Other reasons for diffusion impairment would be due to underlying lung pathology causing an increased diffusion pathway.

Give an example of hypoxia caused by underutilization.

When there is impairment of cytochrome due to toxins/poisons, such as with cyanide

What are the examples of increased requirement for oxygen?

Exercise, hyperthyroidism, infection

What are some examples of causes of V/Q mismatch?

Pulmonary embolism, underlying lung disease such as in lung cancer or chronic obstructive pulmonary disease (COPD), bronchospasm, pneumonia, pulmonary edema

Why is carbon monoxide poisoning a cause of tissue hypoxemia?

Carbon monoxide binds to hemoglobin and makes it unavailable for oxygen transport.

What is the clinical sign of carbon monoxide poisoning?

Cherry red lips and nails

What is an A-a gradient?

The difference in concentration between alveolar and arterial oxygen. It is a measure of gas exchange efficiency in the lung. The less the gradient the greater the oxygenation.

What do you expect the PCO2, and A-a gradient to be in each of the following causes of hypoxia:

See the table.

1. Hypoventilation

2. Right to left shunt

3. Low FIO2

4. V/Q mismatch

5. Diffusion defect

	Hypoventilation	R to L shunt	Low F_{IO_2}	V/Q mismatch	Impaired diffusion
P_{CO_2}	↑	↑	normal	↑	normal
A-a gradient	normal	↑	normal	↑	↑

What are the signs and symptoms of hypoxia?

Dyspnea, tachypnea, tachycardia (increased perfusion), clubbing of nails, and cyanosis of extremities

What is the treatment for most types of hypoxia?

Increased FIO2via oxygen administration while identifying and treating the underlying cause

What type of hypoxemia does not improve with increased FIO2?

A**right-to-left shunt** because there is no ventilation of the abnormal alveoli and, therefore, blood does not come in contact with oxygen.

How is hypoxemia secondary to high altitude treated?

Oxygen administration can help but the body adjusts and self-corrects within several weeks.

OBSTRUCTIVE PULMONARY DISEASES

What defines chronic obstructive pulmonary disease (COPD)?

As the name implies, it is defined by chronic obstruction to expiratory airflow such that the forced expiratory volume in 1 second/forced vital capacity (FEV1/FVC) is decreased

What are the two main forms of COPD?

Emphysema and chronic bronchitis

What is the male-to-female ratio of emphysema?

Male:female = 10:1

What defines emphysema?

Chronic obstructive expiratory airflow with**dilation of air spaces** caused by destruction of alveolar walls

What is the most common cause of emphysema?

Smoking

What type of emphysema does smoking cause?

Centrilobular, meaning that it affects the bronchioles (Hint: The “S” sound is in both smoking and centrilobular)

What causes panacinar emphysema?

Alpha-1-antitrypsin deficiency

What is the function of alpha-1-antitrypsin in the lung?

It protects the elastin in the lungs from proteolytic enzymes.

What are the pathognomonic symptoms associated with emphysema?

Pursed lip breathing(with prolonged expiratory phase),**barrel chest, hyperventilation;** classically described as a**"pink puffer,"** weight loss

What is seen on a chest x-ray (CXR) in a patient with emphysema?

Hyperinflation and hyperlucency of the lungs with flattening of the diaphragms; parenchymal bullae and subpleural blebs may be present; alveolar wall destruction

What do you expect to see in arterial blood gases (ABGs) in a person with early-stage emphysema?

Low PCO2and normal/low PO2

What is the long-term treatment for emphysema?

Smoking cessation!Home oxygen, bronchodilators, steroids; pneumococcal and flu vaccines should be offered

What defines chronic bronchitis?

Productive cough on most days during three or more consecutive months for two or more consecutive years

What is the difference in symptomatology in chronic bronchitis vs. emphysema?

Chronic bronchitis includes a persistent productive cough as well as more hypoxia than seen in emphysema, and patients are usually overweight.

What is the pathognomonic description of a person with chronic bronchitis?

“Blue bloater” because of CO2retention and hypoxia

What do you expect to see in an ABG in a person with chronic bronchitis?

High PCO2and low PO2, compensated respiratory acidosis

What are the potential complications associated with chronic bronchitis?

Right heart failure (cor pulmonale), polycythemia, pneumonia, hepatomegaly

What is the treatment for chronic bronchitis?

Treatments are the same as that for emphysema and include smoking cessation, oxygen therapy, bronchodilators, and steroids, and, also, some treatment with antibiotics in exacerbations.

What is the only treatment proven to extend life in COPD?

Oxygen therapy

How is bronchiectasis defined?

Pathological dilatation of bronchioles caused by chronic inflammation and wall structure destruction

What are some common etiologies of bronchiectasis?

Cystic fibrosis, tuberculosis (TB), lung abscess, toxin inhalation

What is the most common cause of hemoptysis?

Bronchiectasis

What is the underlying pathologic problem that results because of chronic dilatation of bronchioles?

The dilated bronchioles impede mucociliary clearance, favoring mucus pooling and colonization with bacteria and, therefore, further lung damage.

What are the most common pathogens that colonize the lung in an individual with bronchiectasis?

SHiP:

S*taphylococcus aureus*

H*aemophilus****i****nfluenzae*

Pseudomonas

How do you treat the organisms that most commonly infect the lung in bronchiectasis?

Third-generation cephalosporin

What are the signs and symptoms of bronchiectasis?

Halitosis, hemoptysis, chronic cough with sputum production

How can bronchiectasis be diagnosed?

High-resolution computed tomographic (CT) scan of the lungs will demonstrate bronchial dilatation as well as destruction.

What is the pathognomonic sign seen on CXR in a person with bronchiectasis?

Tram track lung markings

What is the treatment for bronchiectasis?

Antibiotics for infections, bronchodilators, oxygen, and, sometimes, lung transplant

How is asthma defined?

Reversible obstruction of airways secondary to airway inflammation, hypersecretion and, most importantly, bronchoconstriction that leads to a decreased peak flow and FEV1

What is intrinsic asthma associated with?

Exercise-induced or upper respiratory infection (URI)-induced asthma

What is extrinsic asthma associated with?

Asthma caused by**eosinophilia** or increased immunoglobulin E (Ig**E**) levels in response to**E** nvironmental antigens

When does asthma usually start and what is its usual course?

Asthma generally begins during childhood and usually resolves on its own by the early teenage years.

What is often the first symptom of asthma that a patient will often describe?

Nighttime cough (for some people this is the only symptom)

What are some of the major signs and symptoms of an acute asthma exacerbation?

Expiratory wheeze, shortness of breath, chest tightness, subcostal retractions, accessory muscle use, prolonged expiratory phase

What would spirometry show in an asthmatic?

Decreased FEV1

What would an ABG show in an asthma attack?

Hypoxia and respiratory alkalosis

How can it be confirmed that the wheezing is caused by asthma and not some other cause?

The wheezing resolves with bronchodilator therapy and the FEV1will increase by 10% or more.

What is a sign of impending respiratory failure in a case of asthma?

ABG that shows normalizing PCO2

What classic diagnosis should you think of if the complete blood count (CBC) of an

asthmatic demonstrates eosinophilia?

Churg-Strauss syndrome

What are the different categories of asthma, what are their symptoms (Sx), and what is the treatment for each?

See the table.

Asthma type	Symptoms	Treatment
Mild intermittent	< 2x per week and nighttime Sx < 2x per month	Short-acting beta-agonist (albuterol)
Mild persistent	> 2x per week and nighttime > 2x per month	Short-acting beta-agonist and low-dose steroid inhaler
Moderate persistent	Daily asthma with nighttime > 1x per week	Long-acting bronchodilator and medium-dose steroid as well as short-acting rescue as needed
Severe persistent	Continuous symptoms	Inhaled steroids and long-acting bronchodilators

What is the first-line treatment for an acute asthma exacerbation?

Oxygen, bronchodilators (includes beta-agonist and ipratropium [Atrovent]) and steroids

What is the second-line treatment for an acute asthma attack?

Subcutaneous epinephrine and $MgSO_4$

How can mild asthma refractory to aggressive beta-agonist therapy be treated?

Add an inhaled steroid

When is systemic corticosteroid therapy indicated in asthma?

Daily or continuous asthma that is refractory to beta-agonist and inhaled steroids

What are some alternative therapies in asthma?

Leukotriene inhibitors and cromolyn sulfate or allergic desensitization in extrinsic asthma

RESTRICTIVE LUNG DISEASE

What is the definition of a restrictive lung disease?

Unlike obstructive lung disease, the**FEV1/FVC is normal to high;** it is the**total lung capacity (TLC)** that**decreases.**

What are some examples of restrictive lung diseases?

Interstitial lung diseases, space-occupying lesions such as tumors; pleural effusions; neuromuscular diseases such as severe scoliosis, spinal cord trauma, and multiple sclerosis

What are some examples of interstitial lung diseases?

Anything that causes chronic lung injury such as asbestosis, acute respiratory distress syndrome (ARDS), coal mine dust, silicosis, berylliosis, chronic lung injury because of chronic infections

What is the pathognomonic description of an interstitial lung disease?

“Honeycomb lung”

What is the most common cause of atelectasis?

A postoperative patient who is nonambulatory for a long period of time

What types of chemotherapy can cause a restrictive lung disease?

Busulfan and bleomycin

PLEURAL EFFUSION

What is a pleural effusion?

Increased fluid in the pleural space

What are the two main types of pleural effusions?

Exudate and transudate

What are some common causes of exudative pleural effusions?

Infection such as pneumonia, malignancy, collagen vascular disease

What are some common causes of transudative pleural effusions?

Congestive heart failure (CHF), cirrhosis, nephritic syndrome

What is the underlying cause of fluid buildup in an exudate?

Increased capillary permeability

What is the underlying cause of fluid buildup in a transudate?

Decreased oncotic pressure (fluid backups)

How can a pleural effusion be evaluated?

Thoracentesis with analysis of cell counts, cultures, chemistries, and cytology

How can a pleural effusion be treated?

Treating the underlying cause and thoracentesis can be both diagnostic and therapeutic.

What lab tests should be sent in order to evaluate the pleural fluid?

Fluid and serum protein, glucose, lactate dehydrogenase (LDH); fluid culture and Gram stain; fluid cytology and cell count with differential and, additionally, you can send fluid amylase, AFB, ANA, RF, pH

What defines an exudative effusion?

If any of the following is true, the fluid effusion is considered exudative.

Pleural protein/serum protein > 0.5

Pleural LDH/serum LDH > 0.6

Pleural LDH > 200

What does it signify if the pleural fluid has > 10,000 WBCs with polymorphonuclear neutrophils (PMNs)?

Most likely a parapneumonic effusion

What is gross blood in the pleural fluid associated with?

Tumor or trauma

What can low glucose (glucose < 60) in the pleural fluid be associated with?

Tumor, empyema, rheumatologic etiology, parapneumonic exudate

What are high amylase levels in pleural fluid associated with?

Pancreatitis but can also be malignancy, or esophageal rupture

What percentage of pleural effusions caused by malignancy will have a fluid cytology that has malignant cells?

Only 40%

Summarize exudative vs. transudative analysis.

See the table.

Test	Exudate	Transudate
Pleural LDH/serum LDH	> 0.6	< 0.6
Pleural LDH	> 200	< 200
Pleural protein/serum protein	> 0.5	< 0.5
Gram stain	Bacteria present most likely secondary to pneumonia (PNA)	No bacteria
WBC	> 1000	< 1000
Glucose	< 60	> 60
Differential	Parapneumonic, malignancy, rheumatologic disease	CHF, pulmonary embolism (PE), cirrhosis, nephritic syndrome

COUGH

What is the definition of an acute cough?

Cough that has lasted < 3 weeks

What is the most common cause of an acute cough?

Postnasal drip

What are the most common causes of postnasal drip?

Sinusitis, allergic rhinitis, seasonal or environmental allergies, flu or cold

What is the preferred method of treatment of postnasal drip caused by allergies?

Antihistamine treatment and/or nasal corticosteroid

What is the preferred method of treatment of postnasal drip caused by the cold?

Antihistamine as well as a decongestant

What is sinusitis?

A bacterial infection of the sinuses

Which sinus is most commonly affected?

The maxillary sinus

What are the signs and symptoms of sinusitis?

Fever, tenderness to percussion over the sinuses, increased pain with bending forward, purulent nasal discharge, halitosis, headache

Define acute, subacute, and chronic sinusitis?

Acute sinusitis lasts < 3 weeks, subacute lasts between 21 and 60 days, and chronic sinusitis lasts > 60 days.

What most commonly causes acute sinusitis?

Viruses

What are the most common pathogens involved in acute bacterial sinusitis?

Streptococcus pneumoniae,

H. influenzae, and*Moraxella catarrhalis*

What is the treatment for acute sinusitis?

Viral rhinosinusitis does not require antimicrobial treatment. Nasal corticosteroids and decongestants are helpful. Studies have shown that steroids lead to faster symptom resolution. Bacterial causes should be treated with amoxicillin, augmentin, or bactrim for 1–2 weeks.

What are the potential complications secondary to chronic sinusitis?

Meningitis, osteomyelitis, orbital cellulitis, cavernous sinus thrombosis, abscess

What is the classic organism causing sinusitis in a diabetic?

Aspergillus causing mucormycosis

What is the definition of a chronic cough?

A cough lasting > 3 weeks

What are the three most common causes of chronic cough?

Postnasal drip, asthma, gastroesophageal reflux disease (GERD)

What medication class can cause a chronic cough?

Angiotensin-converting enzyme (ACE) inhibitors

ACUTE RESPIRATORY DISTRESS SYNDROME

What are the components of acute respiratory distress syndrome (ARDS)?

Refractory hypoxemia, decreased lung compliance, noncardiogenic pulmonary edema

What is the etiology of ARDS?

Endothelial injury secondary to aspiration, multiple transfusions, shock, sepsis, trauma

What are the criteria needed to diagnose ARDS?

1. Acute onset of respiratory distress

2. PaO2: FIO2ratio < 200 mm Hg

3. Bilateral pulmonary infiltrates on CXR

4. Normal capillary wedge pressure

What is the treatment for ARDS?

Treat the underlying disease and give adequate oxygen via mechanical ventilation

What is the overall mortality in ARDS?

About 50%

PULMONARY EMBOLISM

What is the most common etiology of a pulmonary embolism (PE)?

Dislodged deep vein thrombosis**(DVT)**

What are the risk factors for a DVT?

Virchow's triad:stasis (usually due to immobilization), hypercoagulable state, endothelial injury

What are the risk factors for a PE

Same risk factors as getting a DVT, as well as having a DVT, stroke, myocardial infarction (MI); recent surgery leading to immobilization.

What are some examples of hypercoagulable states?

Malignancy; protein C or protein S deficiency; antithrombin III deficiency; factor V

Leiden deficiency; hyperestrogen states such as pregnancy, oral contraceptive use, smoking

What is an important question to ask in the patient's history?

Ask if they have had any recent travel or other immobilization. Long trips cause people to be immobile for long periods of time and therefore have a greater risk for developing DVTs and therefore PEs.

What is the most common sign in a patient with a PE?

Sinus tachycardia

What are some of the common symptoms of PE?

Dyspnea, tachypnea, pleuritic chest pain, fever, unilaterally swollen and painful posterior lower extremity, cough, hemoptysis

What are the classic CXR findings in a PE?

Hampton's hump—wedge-shaped infarct

Westmark's sign—hyperlucency in the lung region supplied by the affected artery

What is the most common EKG finding in a PE patient?

Sinus tachycardia

What is the classic EKG finding in a PE patient?

S1Q3T3—S wave in lead I, Q wave in lead III, and inverted T wave in lead III (Fig. 3-1)

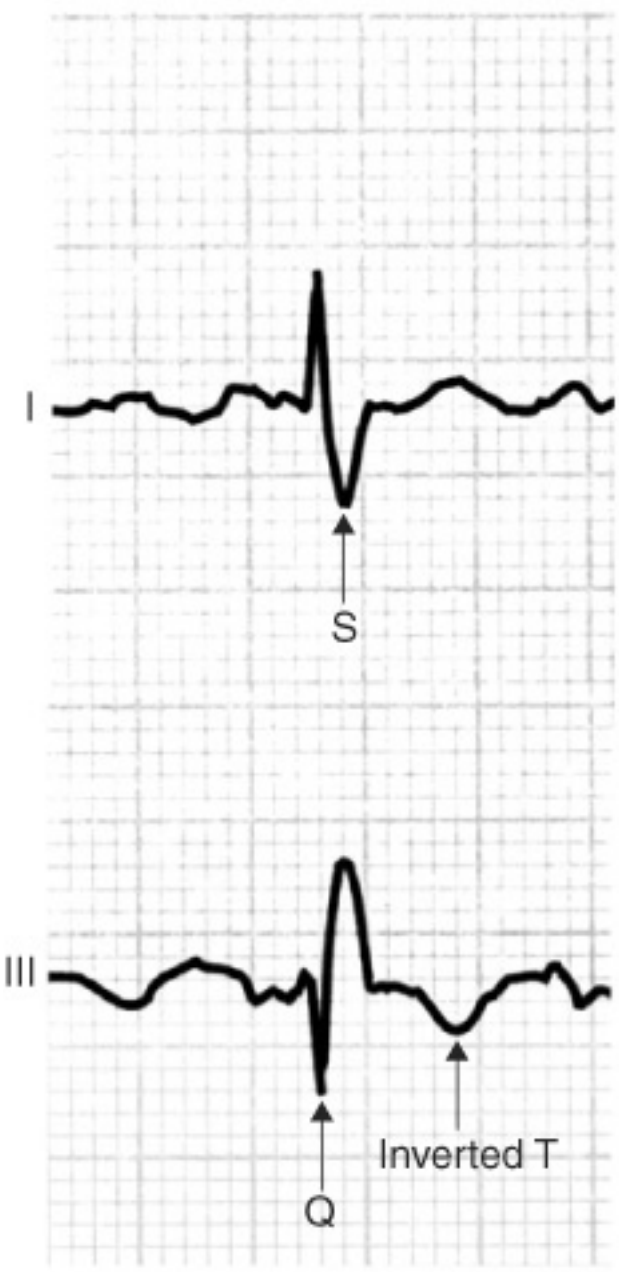


Figure 3-1 Pulmonary embolism S1Q3T3 pattern. (Reproduced with permission from Kaufman MS et al. First Aid for the Medicine Clerkship. New York: McGraw Hill; 2002:75;Figure 3-1)

What is the gold standard for diagnosis of PE?

Pulmonary angiography

What are some of the initial diagnostic techniques used to diagnose a PE?

CT pulmonary angiography or V/Q scan

What blood test can be done to rule out PE if it is not positive?

D-dimer

What diagnostic test can be done to rule out a DVT?

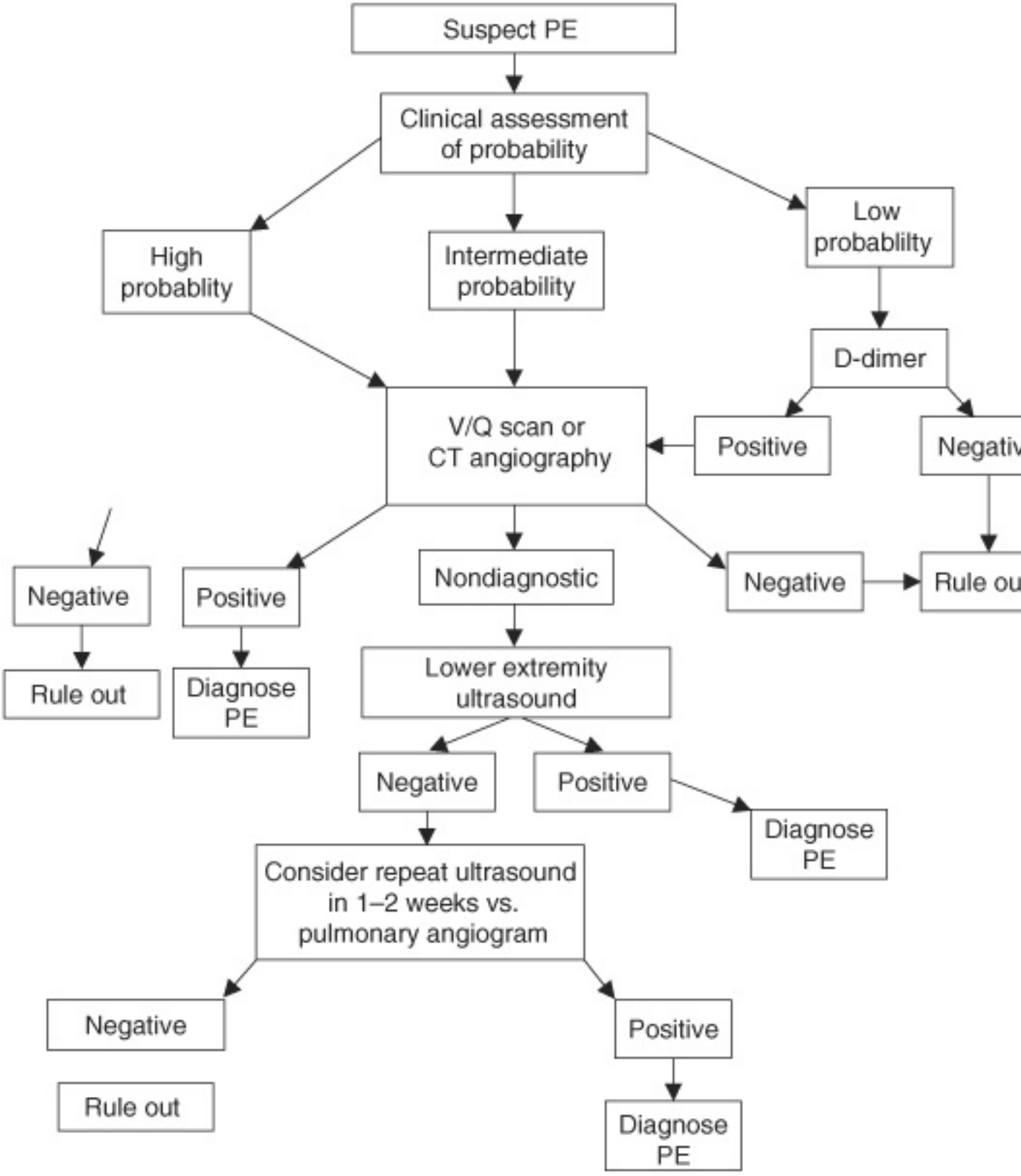
Lower extremity ultrasound (also known as duplex ultrasound)

What is the algorithm used to diagnose a PE, when one is suspected?

See algorithm on the next page.

What are the treatments for a PE?

Heparin or Iovenox (low-molecular-weight heparin) acutely, long-term treatment with coumadin or inferior vena cava (IVC) filter, or tissue-type



plasminogen activator (tPA) thrombolysis in massive PE

What needs to be done if coumadin is being started?

Heparin must be continued until coumadin becomes therapeutic since coumadin can cause a hypercoagulable state.

What is the therapeutic international normalized ratio (INR)?

INR of 2–3

PNEUMOTHORAX

A person with what body habitus is most likely to have a primary spontaneous pneumothorax?

Tall and thin male

What is the most likely etiology of a primary spontaneous pneumothorax?

Rupture of subpleural blebs

What are some risk factors for having a secondary spontaneous pneumothorax?

COPD, lung cancer, pneumonia, TB, HIV, cystic fibrosis, trauma

What are the signs and symptoms of a pneumothorax?

Sudden unilateral chest pain, dyspnea, and tachypnea

What is found on physical examination in a person with a pneumothorax?

Absent breath sounds on the side of the pneumothorax and hyperresonance to percussion

What is seen on CXR in a pneumothorax?

Absent lung markings on the side of the pneumothorax

What is the treatment of a spontaneous pneumothorax?

Oxygen is the mainstay of therapy, but if the pneumothorax is symptomatic, a tube thoracostomy may be indicated. Pleurodesis can be used to make the visceral and parietal pleura adhere to each other.

What is a tension pneumothorax?

A chest wall defect causes air to be trapped in the pleural space during expiration like a one-way valve (Fig. 3-2).

How is a tension pneumothorax treated?

This is a medical emergency. Treatment includes immediate needle decompression and chest tube placement after.

HEMOPTYSIS

What is the most common cause of hemoptysis?

Bronchiectasis

What are some other causes of hemoptysis?

Lung cancer (CA), pneumonia, bronchitis

What are the treatments for hemoptysis?

Place bleeding side down to protect the airway, oxygen as needed and in severe cases, bronchial artery embolization or intubation of the good lung

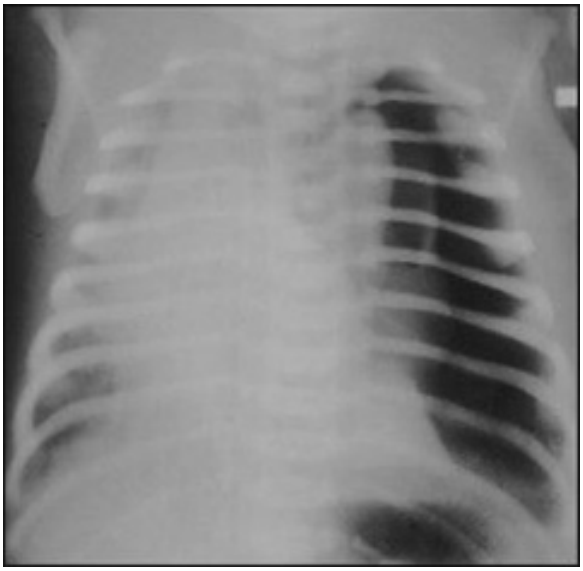


Figure 3-2 Tension pneumothorax. (Reproduced with permission from William Herring, MD, FACR; Radiology Residency Program Director at Albert Einstein Medical Center in Philadelphia, PA;http://www.learningradiology.com)

LUNG CANCER

What is the most common cause of cancer death in the United States?

Lung cancer

What is the most likely causative factor of lung cancer?

Smoking

What are some other causes of lung cancer?

Second-hand smoke, exposure to asbestos, nickel, arsenic, radon gas

What are the two main categories of lung cancer?

Small cell and nonsmall cell

What are the different types of lung tumors that are nonsmall cell lung cancers?

Large cell, adenocarcinoma, squamous cell, bronchoalveolar cell

Name the type of lung cancer associated with the following:

Central location	**S**mall cell, **s**quamous cell
Poor response to chemotherapy	Nonsmall cell
Treated with surgery	Nonsmall cell

Poorer prognosis	Small cell
Sensitive to chemotherapy	Small cell
Peripheral location	Nonsmall cell
Linked to smoking	**S**mall cell and **s**quamous cell
Not linked to smoking	Bronchoalveolar cancer, a type of adenocarcinoma
Secretes parathyroid hormone	Squamous cell
Associated with hypercalcemia	Squamous cell
Metastatic at diagnosis	Small cell
Secretes serotonin	Carcinoid tumor
Eaton-Lambert syndrome	Small cell
SIADH (syndrome of inappropriate antidiuretic hormone secretion)	Small cell
Cushing syndrome	Small cell associated with adrenocorticotropic hormone (ACTH) secretion
Asbestos exposure	Mesothelioma

What are some signs and symptoms of lung CA?

Chronic cough, hemoptysis, weight loss, night sweats, pneumonia (postobstructive), hoarseness, paraneoplastic syndrome

How is lung cancer diagnosed?

Usually a nodule or mass is seen on CXR or CT of the chest and is diagnosed with a**biopsy** usually done via bronchoscopy or CT-guided fine needle aspiration

What are some of the signs and symptoms of a carcinoid tumor?

Symptoms of carcinoid syndrome due to serotonin secretion, which include flushing, asthmatic wheeze, diarrhea

What is the diagnostic test for a carcinoid tumor?

Test for elevated urine 5-hydroxyindoleacetic acid (5-HIAA), a serotonin metabolite

How is carcinoid syndrome treated?

Serotonin antagonist

What is a Pancoast tumor?

Superior sulcus tumor

What paraneoplastic syndromes are associated with a Pancoast tumor?

Horner syndrome, superior vena cava syndrome

What is Pancoast syndrome?

Shoulder and arm pain secondary to the tumor compressing the eighth cervical nerve

Name the paraneoplastic syndrome associated with signs and symptoms described below.

Ptosis, myosis, anhydrosis	Horner syndrome
Facial and upper extremity swelling	Superior vena cava syndrome
Hyponatremia secondary to ectopic release of antidiuretic hormone (ADH)	SIADH
Low acetylcholine release leading to myasthenia gravis type symptoms	Eaton-Lambert syndrome

PNEUMONIA

What are some common signs and symptoms of pneumonia (PNA)?

Cough with purulent sputum, fever, chills, pleuritic chest pain

What are some common physical examination findings in a patient with pneumonia?

Decreased breathing sounds, crackles, egophony, dullness to percussion, tactile fremitus on the side of the pneumonia, fever

What studies should be ordered if a PNA is suspected?

CXR, CBC, sputum culture and Gram stain, blood culture (in hospitalized patient)

What do you see on a CXR in a patient with pneumonia?

Lobar consolidation (Fig. 3-3)

What would the CBC show?

Leukocytosis with a left shift

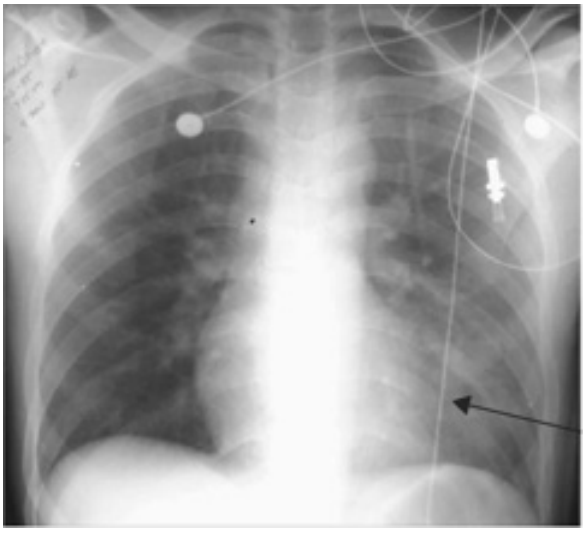


Figure 3-3 Pneumonia. (Reproduced with permission from William Herring, MD, FACR; Radiology Residency Program Director at Albert Einstein Medical Center in Philadelphia, PA;http://www.learningradiology.com)

Name the most common organism causing pneumonia in each of the following cases:

1. Community-acquired pneumonia	*S. pneumoniae*
2. Typical community-acquired pneumonia	*S. pneumoniae* and *Haemophilus influenzae*
3. Atypical community-acquired pneumonia	*Chlamydia, Legionella, Mycoplasma*
4. Hospital-acquired pneumonia	*Pseudomonas, S. aureus,* enteric gram-negative rods
5. Pneumonia in a patient with cystic fibrosis that easily develops resistance	*Pseudomonas*
6. Pneumonia after the flu	*S. aureus*
7. Atypical pneumonia in the young patient	*Mycoplasma*
8. Right upper lobe pneumonia in an alcoholic	*Klebsiella,* usually secondary to aspiration
9. Positive cold agglutinin test	*Mycoplasma*
10. Pneumonia in a butcher who sells rabbit meat	*Francisella tularensis*

11. Pneumonia in a person who likes to explore caves in the Ohio Valley	*Histoplasma*
12. Pneumonia in a person from southwestern United States	*Coccidioides immitis*
13. Pneumonia in a bird keeper	*Chlamydia psittaci*
14. Pneumonia that mimics TB, and is gram positive	*Nocardia*
15. Pneumonia in a person with a lot of air-conditioning exposure or aerosolized water	*Legionella*
16. Aspiration pneumonia in an alcoholic, a patient with dementia, or a person who became unconscious	Anaerobes
17. Pneumonia contracted from farm animals and called "Q fever"	*Coxiella burnetii*
18. Pneumonia with hyponatremia, LDH> 700, diarrhea, mental status change	*Legionella*

19. Fungus ball	*Aspergillus*
20. Rust-colored sputum	*S. pneumoniae*
21. Currant jelly sputum	*Klebsiella*
22. Three pneumonias in AIDS patients with CD4 count < 200	*Pneumocystis carinii, Histoplasma, Cryptococcus*
23. Pneumonia in AIDS patients with CD4 count < 50	*Mycobacterium avium*, cytomegalovirus (CMV)
24. Bilateral infiltrates on CXR	*Mycoplasma, Pneumocystis carinii* pneumonia (PCP)

What is the treatment for each of the following cases of pneumonia?

1. Typical pneumonia	Third-generation cephalosporin, macrolide, fluoroquinolone
2. Atypical pneumonia	Doxycycline, macrolide, quinolone
3. Anaerobic pneumonia	Clindamycin, metronidazole
4. *P. carinii*	Bactrim

What are the most common pathogens and treatments in each of the following cases?

1. Outpatient community-acquired pneumonia in a patient aged < 60	Organisms: *S. pneumoniae*, *Mycoplasma*, *Chlamydia pneumoniae*, *H. flu* (Haemophilus influenzae) Treatment: erythromycin, tetracycline, or azithromycin to also cover *H. flu*
2. Outpatient with age > 60 and with comorbidities such as CHF, COPD, diabetes, alcoholic, cirrhosis	Organisms: *S. pneumoniae, H. flu*, aerobic gram-negative rods such as *Klebsiella, Escherichia coli, Enterobacter, S. aureus, Legionella* Treatment: second-generation cephalosporin, amoxicillin, fluoroquinolone, erythromycin, or doxycycline for atypical pneumonia
3. Community-acquired pneumonia requiring hospitalization	Organisms: *S. pneumoniae, H. flu*, anaerobes, aerobic gram-negative rods, *Legionella, Chlamydia* Treatment: third-generation cephalosporin and azithromycin or doxycycline for atypical pneumonia
4. Community-acquired pneumonia requiring intensive care unit (ICU) admission	Organisms: *S. pneumoniae, H. flu, S. aureus*, gram-negative bacilli, *Legionella, Pseudomonas*

	Treatment: third-generation cephalosporin and azithromycin or doxycycline for atypical pneumonia
5. Nosocomial pneumonia	*Pseudomonas, S. aureus, Legionella,* gram-negative rods Treatment: third-generation cephalosporin, aminoglycoside, or piperacillin tazobactam and vancomycin if methicillin-resistant *S. aureus* (MRSA) suspected

TUBERCULOSIS

How does TB spread?

Air droplet transmission

Who is at high risk for becoming infected with TB?

Immunocompromised, foreign-born, homeless, prisoner, low-income communities, intravenous (IV) drug users

What are the common signs and symptoms of TB?

Productive cough, night sweats, weight loss, hemoptysis,fever, chills, chest pain,

How is latent TB detected?

Positive purified protein derivative (PPD) (tuberculin) skin test

What is considered a positive PPD?

> 15 mm in any person

> 10 mm in immunocompromised, IV drug user, foreign-born, prisoner, nursing home

resident, people who work in the medical field (that means you), underserved community

> 5 mm: HIV, abnormal CXR, close contact had TB

How is a positive PPD treated?

Isoniazid (INH) for 9 months with vitamin B6

What laboratory tests should be done when starting a patient on isoniazid (INH)?

Liver function tests (LFTs) because of possibility of hepatotoxicity

What part of the lung does primary TB usually affect?

Lower lobes

What is the radiographic finding of healed primary TB called?

“Ghon complex,” which is a calcified nodule with calcified lymph nodes (Fig. 3-4)

What is secondary TB?

Reactivation TB

Where is secondary TB usually found?

Lung apices

What is extrapulmonary TB?

TB that had disseminated to other organs

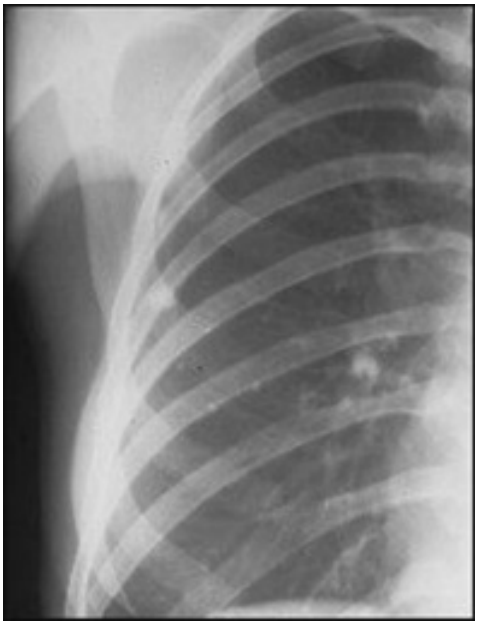


Figure 3-4 Tuberculosis. (Reproduced with permission from William Herring, MD, FACR; Radiology Residency Program Director at Albert Einstein Medical Center in Philadelphia, PA;http://www.learningradiology.com)

What is the most common extrapulmonary location for TB to spread?

Kidneys

What are other locations where extrapulmonary TB can be found?

Liver, central nervous system (CNS), vertebral bodies, psoas muscle, cervical lymph nodes, pericardium

What is TB of the vertebral bodies called?

Pott disease

What is cervical lymphadenopathy secondary to TB infection called?

Scrofula

How is active TB diagnosed?

Clinical symptoms, CXR, and sputum acid-fast stain and culture

What is seen on CXR in active TB?

Upper lobe infiltrates with scarring, cavitary lesions

What is the treatment for active TB?

Four-drug therapy for a minimum of 6 months (remember the mnemonic**RIPE**):

Rifampin

INH

Pyrozinamide

Ethambutol

CHAPTER 4

Neurology

CEREBROVASCULAR ACCIDENTS

What is a cerebrovascular accident (CVA)?

Sudden onset of neurologic deficit that is a result of cerebrovascular disease

What does TIA stand for?

Transient ischemic attack

What is a TIA?

A neurologic deficit that lasts**< 24 hours** and**resolves completely**

What is a stroke?

Focal neurologic deficit that results from infracted cerebral tissue

What does RIND stand for?

Reversible**I** schemic**N** eurologic**D** eficit

What is RIND?

Neurologic deficits that last > 24 hours and < 3 weeks

What are the two greatest risk factors for a stroke?

Hypertension and smoking

What are the three possible etiologies for a CVA?

Ischemic, hemorrhagic, or hypoperfusion (associated with hypotension)

What are the two etiologies for ischemia?

1. Thrombotic etiology which is secondary to atherosclerosis

2. Embolic etiology which is usually either cardiac in origin or from carotid arteries

What is the most common etiology of a CVA?

Ischemia

Name the term associated with each of the following:

Difficulty with expression of both written and spoken language as well as difficulty with comprehension	Aphasia
Difficulty with performing motor tasks	Apraxia (a patient with a**praxia can't practice**)
Difficulty with articulation	Dysarthria
CVA sequelae in which the patient speaks with fluency, however, without making sense, and comprehension is impaired and patient is unaware of the deficit	Wernicke aphasia (Wernicke is **wordy**)
CVA sequelae in which the patient has difficulty verbalizing what they want to express, comprehension is intac, and patient is aware of the deficit	Broca aphasia (Broca had **broken** language)
Infarct in the deep grey matter associated with hypertension and atherosclerosis	Lacunar infarct
Infarction that occurs in an area supplied by two major arteries and is usually a result of hypotension	Watershed infarct
Most common source of emboli that leads to a stroke	Carotid atheroma
Upper motor neuron deficit causing *flexion* of the upper extremities	Decorticate posturing
Lower motor neuron deficit causing *extension* of the upper extremities	Decerebrate

Describe the deficits caused by occlusion of the following arteries:

Middle cerebral artery (MCA) supplying the dominant hemisphere	Contralateral hemiparesis and hemisensory deficit, aphasia, homonymous hemianopsia
MCA supplying the nondominant hemisphere	Contralateral hemiparesis and hemisensory deficit, homonymous hemianopsia, confusion, apraxia, body neglect on contralateral side
Anterior cerebral artery (ACA)	Broca aphasia, contralateral weakness of lower extremity, incontinence
Posterior cerebral artery (PCA)	Homonymous hemianopsia with macular sparing, oculomotor nerve palsy, aphasia, and alexia if dominant hemisphere is affected
Posterior inferior cerebellar artery (PICA)	Vertigo, ataxia, contralateral pain and temperature disturbance, dysphagia, dysarthria, ipsilateral Horner syndrome (ptosis, miosis, anhidrosis)
Anterior inferior cerebellar artery	Deafness, tinnitus, ipsilateral facial weakness, gaze palsy
Ophthalmic artery	Amaurosis fugax (monocular blindness)

What is the first study to order if you suspect a stroke in a patient?

Computed tomography (CT) of head**without** contrast

What other studies can be done to further assess the patient?

Magnetic resonance imaging (MRI) to evaluate for subacute infarction or hemorrhage; carotid Doppler ultrasound to rule out carotid artery stenosis; echocardiogram to rule out embolic sources

What is the treatment for a TIA?

Patient should be started on aspirin.

What medication should the patient be started on if they fail aspirin?

Plavix

What other antiplatelet therapies are available other than aspirin?

Clopidogrel, ticlopidine, aggrenox

When would you consider a carotid endarterectomy?

If the patient had carotid artery stenosis > 70%

What is the treatment for a patient who had a cardioembolic stroke?

Anticoagulation with heparin or coumadin

What treatment improves outcome in a patient who present with an embolic stroke with symptoms beginning < 3 hours ago?

Tissue plasminogen activator (tPA)

What is a contraindication to tPA use?

Intracranial bleeding

How should hypertension be treated in a patient who acutely had a stroke?

Hypertension should not acutely be controlled tightly in order to allow for good cerebral perfusion.

Other than starting medications, what other long-term interventions should be taken in a patient with a history of stroke to prevent future infarctions?

Good diabetes control (improved HgA1c); control hypertension; smoking cessation; treat hyperlipidemia

SEIZURE DISORDERS

What is a seizure?

Excessive firing of cortical neurons leading to neurologic symptoms

What is the single most useful test to evaluate seizures?

Electroencephalogram (EEG)

What tests should be done on a patient suspected to have had a seizures?

Complete neurologic examination. Check for signs of incontinence, tongue lacerations, other injuries to the body to distinguish from syncope. Also check the following laboratory tests: complete blood count (CBC), electrolytes, calcium, glucose, oxygen level, liver function tests, blood urea nitrogen (BUN), creatinine (CR), rapid plasma reagin (RPR), erythrocyte sedimentation rate (ESR), urine toxicology screen. MRI and CT can also be done to rule out a mass.

What factors can increase the risk of having a seizure?

History of having a seizure in the past, central nervous system (CNS) tumor, CNS infection, trauma, stroke, high fever in children

What is Todd paralysis?

Postictal state in which there are focal neurologic deficits that last 24–48 hours; usually associated with focal seizures

Name the two types of generalized seizures.

Tonic-clonic and absence seizures

Name the seizure disorder described below:

Seizure that may involve motor, autonomic, or sensory functions with no loss of consciousness	Simple partial seizure
Elevated prolactin level in postictal state	Tonic-clonic seizure
Seizure that arises from one distinct region of the brain	Focal seizure
Most commonly involves the temporal lobe	Complex partial seizure
Also known as petit mal seizures	Absence seizures
Seizure involving both hemispheres with a loss of consciousness and postictal confusion	Generalized seizure
Loss of consciousness followed by muscle contractions and then symmetric jerking of extremities	Tonic-clonic seizures
Seizures lasting > 30 seconds or repetitive seizures lasting > 5 minutes with continuous altered level of consciousness	Status epilepticus
Often mimics "daydreaming" in children	Absence seizure
Associated with cyanosis and urinary incontinence	Tonic-clonic seizure
Seizure in which patient has an altered level of consciousness with	Complex partial seizures

auditory or visual hallucinations as well as repetitive motor actions and postictal confusion	
Also known as grand mal seizure	Tonic-clonic seizure
Can be caused by electrolyte imbalances, withdrawal from drugs or alcohol, infection (often in CNS), trauma	Status epilepticus
Impaired consciousness lasting only a few seconds	Absence seizures
EEG with three per second spike and wave	Absence seizure

Indicate the treatment for each of the following types of seizures:

Focal	Phenytoin, carbamazepine, valproic acid
Tonic-clonic	Phenytoin, carbamazepine, phenobarbital
Absence	Ethosuximide and valproic acid
Status epilepticus	This is a medical emergency. Start with the ABCs (airway, breathing, circulation). Benzodiazepine as well as loading dose of phenytoin administration are the next step in treatment, followed by intravenous (IV) sedatives (such as phenobarbital) if patient continues to seize.

What is the most significant side effect of each of the following antiseizure medications?

Phenytoin	Gingival hyperplasia
Valproic acid	Hepatotoxic; thrombocytopenia; neutropenia
Carbamazepine	Aplastic anemia
When can antiseizure medication be discontinued in a patient with a history of seizures?	No seizures for 2 years

MENINGITIS

What is the most common bacterial pathogen causing meningitis in adults?

Streptococcus pneumoniaecauses up to 60% of meningitis cases.

What two bacterial pathogens cause most cases of meningitis in young adults?

*S. pneumoniae*and***Neisseria meningitidis***

In what population does Group B streptococcus cause meningitis?

Neonates (most common cause of meningitis in neonates)

What three bacterial pathogens most commonly cause meningitis in neonates?

Listeria, Group B streptococcus, and*Escherichia coli*

In what adult population does*Listeria* cause meningitis?

Immunocompromised patients

What bacterial pathogen known to cause meningitis is now vaccinated against?

Haemophilus influenzae

What is the treatment for each of the bacterial pathogens in meningitis?

SeeTable 4-1 .

Table 4-1 Meningitis Organisms and Treatments

Organism	**Treatment**

S. pneumonia	Cefotaxime + vancomycin**or** ceftriaxone
N. meningitidis	Penicillin G**or** ceftriaxone
Listeria	Ampicillin + gentamicin
Group B streptococcus	Ampicillin
H. influenzae	Cefotaxime

What are the classic symptoms of meningitis?

Fever, headache with neck stiffness, photophobia, meningismus, Kernig sign, Brudzinski sign

What is meningismus?

Patient has difficulty touching their chin to their chest.

What is Kernig sign?

Patient has pain when extending the knee with the thigh at 90°.

What is Brudzinski sign?

Neck flexion causes involuntary flexion at the hip and knees.

What test is used to diagnose meningitis?

Lumbar puncture with cerebrospinal fluid (CSF) analysis including Gram stain, cultures

What would the CSF findings be in*bacterial* meningitis (seeTable 4-2 **)?**

Increased protein, decreased glucose, very elevated WBCs, elevated opening pressure, and elevated number of neutrophils

What would the CSF findings be in*viral* meningitis?

Normal protein and glucose, elevated WBC, normal or elevated opening pressure, increased lymphocytes

What would the CSF findings be in*fungal* meningitis?

Elevated protein, decreased glucose, elevated WBC, elevated opening pressure, increased lymphocytes (Table 4-2)

Table 4-2CSF Findings in Meningitis

Etiology	Protein	Glucose	WBC	Pressure	Differential
Bacterial	↑	↓	↑	↑	↑ Neutrophils
Viral	Normal	Normal	↑	Normal/↑	↑ Lymphocytes
TB/fungal	↑	↓	↑	↑	↑ Lymphocytes

What is the appropriate empiric treatment for meningitis in each of the following populations (see Table 4-3 **)?**

Neonates	Cefotaxime + ampicillin
1–3 months	Cefotaxime + vancomycin
Young adults	Ceftriaxone + vancomycin
Adults	Cefotaxime + vancomycin
Elderly, immunocompromised	Ceftriaxone + ampicillin

Table 4-3 Meningitis Treatment by Population

Population	Most Common Organisms	Treatment
Neonates (< 1 month)	**Group B strep tococcus;** *Listeria; E. coli*	Cefotaxime + ampicillin
1–3 months	*S. pneumoniae; H. influenzae; N. meningitidis*	Cefotaxime + vancomycin
Children; young adults; crowded living environments	*S. pneumoniae; N. meningitidis*	Ceftriaxone + vancomycin
Adults	***S. pneumoniae***	Cefotaxime + vancomycin
Elderly; immuno-compromised	*S. pneumoniae; Listeria*	Ceftriaxone + ampicillin

BRAIN TUMORS

What is the most common type of brain tumor?

Metastatic tumor

From what primary tumors do most metastatic brain tumors originate?

Lung cancer, breast cancer, melanoma, gastrointestinal (GI) tumors

Anatomically, where do most*adult* brain tumors tend to present?

Supratentorially

Anatomically, where do most*childhood* brain tumors tend to present?

Infratentorially

What are some common symptoms of brain tumors?

Headache (especially upon waking), vomiting, seizures, focal neurologic symptoms

How is a brain tumor diagnosed?

CT with contrast/MRI with gadolinium localizes the lesion and a biopsy is used to get the histologic class of the tumor.

What is the most common type of primary brain neoplasm?

Astrocytoma

What is the most common type of astrocytoma?

Glioblastoma multiforme

What is the prognosis of glioblastoma multiforme?

Poor prognosis. 5-year survival is < 5%.

Where do ependymomas usually arise?

In the fourth ventricle

In what population are ependymomas most common?

Children

What is the prognosis?

80% 5-year survival

What is the most common cranial nerve tumor?

Schwannoma

What cranial nerve does a schwannoma affect?

Cranial nerve VIII—vestibular division

What is the most common mesodermal tumor?

Meningioma

How are most brain tumors treated?

Surgical excision and radiation. Medulloblastomas also require chemotherapy and schwannomas are treated with surgery alone.

DEMYELINATING DISEASES

What is the most common demyelinating disorder?

Multiple sclerosis (MS)

Who is at higher risk for developing MS?

Those with a family history of MS, those who lived the first 10 years of their life in northern latitudes or temperate climates, female sex (incidence is 2:1 female:male)

What age is the peak age of MS presentation?

Age 20–40

What is the typical course of the disease?

Multiple progressive neurologic alterations that wax and wane and cannot be explained by a single lesion

What are some of the signs and symptoms of MS?

Limb weakness, paresthesias, optic neuritis,**nystagmus, scanning speech, intranuclear ophthalmoplegia,** vertigo, diplopia

What can be seen on MRI on a patient with MS?

MRI shows multiple, asymmetric, periventricular plaques with multiple areas of demyelination

What does the CSF show in an MS patient?

Oligoclonal bands; elevated IgG

What is the treatment for MS?

Steroids during acute episodes and interferon-ß to prolong remission

What is the other name for amyotrophic lateral sclerosis (ALS)?

Lou Gehrig disease

What is the underlying pathology in ALS?

Slowly progressive loss of upper and lower motor neurons in the CNS

What are the clinical signs and symptoms of ALS?

Asymmetric, progressive muscle weakness initially with fasciculations which present clinically as difficulty swallowing. Patients also have upper motor neuron and lower motor neuron signs on physical examination. They do not have bowel or bladder involvement.

Give examples of both upper and lower motor neuron signs?

Upper motor neuron signs: spastic paralysis, hyperreflexia, upgoing Babinski

Lower motor neuron signs: flaccid paralysis, fasciculations, downgoing Babinski

How is ALS diagnosed?

Clinically there should be a combination of upper motor neuron and lower motor neuron ? symptoms in three or more extremities. An electromyogram (EMG) will show widespread denervation and fibrillation potentials in at least three limbs.

What is the main treatment for ALS?

Supportive care

What do ALS patients ultimately die from?

Respiratory failure

What is Guillain-Barré syndrome?

An autoimmune, acute demyelinating disorder affecting the**peripheral nerves** (particularly

motor fibers)

What bacterial infection is Guillain-Barré syndrome associated with?

Campylobacter jejuni

What often precedes Guillain-Barré syndrome?

A bacterial infection causing diarrhea, specifically with*Campylobacter* , viral infection, or vaccination

Clinically, how does Guillain-Barré syndrome present?

Ascending paralysis. Symptoms usually begin with distal weakness and progress to proximal weakness with hyporeflexia and facial diplegia. It can eventually progress to paralysis of the diaphragm, leading to respiratory failure.

What tests would you do to diagnose Guillain-Barré syndrome?

Lumbar puncture and EMG

What would you see in the CSF after a lumbar puncture?

?? protein; normal cell count—this is known as**albuminocytologic dissociation**

What interventions should be undertaken in a patient with Guillain-Barré syndrome?

Monitor respiratory function very closely and**intubate** if needed. Medical treatment includes plasmapheresis and intravenous immunoglobulin (IVIG).

COGNITIVE DISORDERS

What is dementia?

A syndrome of global intellectual and cognitive deficits which are constant and progressive. Patients have no sensory abnormalities (no auditory or visual hallucinations)

What specific type of cognitive deficit is dementia usually characterized by?

Memory loss (Remember: de**mem**tia)

What types of cognitive impairments characterize dementia?

Impairments in memory, abstract thought, planning and organization as well as aphasia, apraxia and agnosia.

What are the causes of dementia?

Alzheimer; Parkinson, Huntington; seizure disorder; stroke; B12deficiency; thiamine deficiency; folate deficiency; alcoholism; head trauma (especially repetitive); neurotoxins; CNS infections such as syphilis; CNS malignancies; normal pressure hydrocephalus

Degenerative disorders (Alzheimer, Parkinson, Huntington) (Remember the mnemonic:**DEMENTIA**)

Electrolyte imbalances;**E** ndocrine

Mass effect

Epilepsy

Neurotoxins

Trauma

Infection

Alzheimer is most common (70%–80%)

Stroke

What tests would you order if you suspected dementia in a patient?

Head CT, CBC, electrolytes, B12; folate, rapid plasma region (RPR), thyroid-stimulating hormone (TSH), urine toxicology screen

What medical problem can mimic dementia?

Depression can present as pseudodementia.

What class of medications should be avoided in demented patients?

Benzodiazepines

What is the general treatment for dementia?

Supportive treatment. Patients should also learn to use environmental clues.

What is delirium?

Sudden and transient global cognitive deficits that**wax and wane.**

What specific clinical symptom distinguishes delirium from dementia?

Patients with delirium have sensory deficits which include**auditory and visual hallucinations.**

What are the symptoms of delirium?

Waxing and waning levels of consciousness and sensory disturbances. Patients are often found to be anxious, combative, have poor memory, and have decreased attention span.

What are the main etiologies of delirium?

HIDE:

Hypoxia

Infection

Drugs

Electrolyte abnormalities

What tests would you order if you suspected delirium?

CBC, electrolytes, glucose, TSH, urinalysis, chest x-ray (CXR), urine toxicology screen, pulse oximetry, possibly a head CT

What else would be very important to examine in a patient with delirium?

Patient's medication list

What two drug classes are often found to cause delirium?

1. Anticholinergics

2. Benzodiazepines

What is the most common infection leading to delirium in the elderly?

Urinary tract infection (UTI)

What is the main treatment for delirium?

Treat the underlying cause. Antipsychotics can be used to help control symptoms.

How can dementia be distinguished from delirium?

See Table 4-4 .

Table 4-4 Characteristics of Dementia versus Delirium

Dementia	Delirium
Constant cognitive deficits which are progressive over time	**Waxing and waning** of cognitive deficits (usually worse at night called **"sundowning"**
No audio/visual hallucinations	Hallucinations common
Deficits are irreversible	Deficits can be reversed if insulting factors removed
No alteration in the level of consciousness	Altered level of consciousness

What is the most common cause of dementia?

Alzheimer

What is found in the cerebral cortex in patients with Alzheimer?

Amyloid plaquesand**neurofibrillary tangles**

What is the most common symptom of Alzheimer?

Memory deficits

What can be seen on CT in a patient with Alzheimer?

Cortical atrophy

What genotype is Alzheimer associated with?

Apolipoprotein E

How can Alzheimer be diagnosed?

It is a clinical diagnosis because it can only be diagnosed definitively at autopsy

What medications can slow the cognitive decline in Alzheimer?

Anticholinesterase inhibitors: donepezil (Aricept), selegiline, tacrine

What is the underlying pathology in Parkinson disease?

Degeneration of dopaminergic neurons in the**substantia nigra**

What are the pathognomonic symptoms of Parkinson disease?

Cogwheel rigidity, resting tremor, bradykinesia, shuffling gait, mask-like faces,

postural instability

What are the treatment options for Parkinson disease?

Amantadine; Sinemet (levodopa/carbidopa); benztropine, selegiline, bromocriptine

What is the mechanism of amantadine and what symptom is it best for?

Blocks dopamine reuptake in presynaptic neurons and treats**bradykinesia** mainly in mild disease

What is the mechanism of Sinemet and what symptom does it best treat?

Sinemet is a combination of levadopa and carbidopa. Levodopa is converted into dopamine in the substantia nigra. Carbidopa is necessary because it cannot cross the blood-brain barrier and prevents levodopa metabolism by peripheral tissues. It is also best for treating bradykinesia.

Name some of the anticholinergic drugs?

Benztropine, trihexyphenidyl

What symptom of Parkinson disease do anticholinergics best treat?

Tremor

What is the mechanism of selegiline?

Monoamine oxidase (MAO) inhibitor which blocks dopamine metabolism

What is the mechanism of bromocriptine?

Dopamine agonist

What kind of genetic pattern does Huntington disease follow?

Autosomal dominant

On what chromosome is the genetic alteration found and what is the genetic defect?

Chromosome 4; triple repeat of CAG

In what age range does Huntington disease usually present?

Between 30 and 50 years of age

What is the underlying pathophysiology of Huntington disease?

Atrophy of the caudate nucleus

What are the typical signs and symptoms of Huntington disease?

Choreiform movements, dementia, schizophreniform changes,ataxic gait

What is the treatment for Huntington disease?

Supportive treatment. Antipsychotics can be used as needed for psychotic symptoms.

What is the problem in Wilson disease?

Defect in copper metabolism

What are the symptoms of Wilson disease?

Tremors and rigidity as well as psychiatric changes such as manic depression; patients have parkinsonian features

What is the pathognomonic physical examination finding in Wilson disease?

Kayser-Fleischer ring around the cornea

How is Wilson disease diagnosed?

Elevated serum ceruloplasmin

What is the treatment for Wilson disease?

Penicillamine with pyridoxine (vitamin B6) and zinc. Liver transplantation is the final treatment if patient fails medical therapy.

HEADACHE

What is the most common type of headache?

Tension headache

What are signs and symptoms of a tension headache?

Bilateral, bandlike, dull, most intense at neck/occiput, worsened with stress

What psychiatric disorder is it most commonly associated with?

Depression

What is the most common age group with this type of headache?

Between 20 and 50 years of age

What type of headache is characterized of rhinorrhea being unilateral, stabbing, retro-orbital pain, ipsilateral lacrimation, ptosis, and nasal congestion?

Cluster headache

What type of headache is characterized by photophobia, nausea, aura, and being unilateral?

Migraine headache

What are some common triggers for migraines?

Menstruation, stress, foods, alcohol

What type of headache is associated with jaw claudicating?

Temporal arteritis (usually a unilateral temporal headache with temporal artery tenderness)

What is the predilection for temporal arteritis?

Female > Male

What are the risks of temporal arteritis?

Optic neuritis and blindness if not treated

What is it associated with?

Polymyalgia rheumatica

How is it diagnosed?

Must do a temporal artery biopsy; elevated ESR is just a screening test

What is it associated with?	Polymyalgia rheumatica
How is it diagnosed?	Must do a temporal artery biopsy; elevated ESR is just a screening test

What is the treatment for temporal arteritis?

Steroids

INTRACRANIAL BLEEDING

What is "the worst headache of my life"?

Subarachnoid hemorrhage (SAH)

What is the most common cause of SAH?

Head trauma

What is the most common underlying cause of a spontaneous SAH?

Atrioventricular (AV) malformation

How is an SAH diagnosed?

CT scan shows subarachnoid blood (dark); lumbar puncture shows bloody CSF with xanthochromia; cerebral angiography can be done to find berry aneurysms.

What is the immediate treatment for an SAH?

Goal is to decrease intracranial pressure (ICP). Give nimodipine to decrease chance of vasospasm, raise the head of the bed, and administer IV fluids.

What is the second-line treatment for an SAH?

Surgical evacuation of blood via burr holes

What is a berry aneurysm?

Outpouching of vessels in the circle of Willis, usually at bifurcations (looks like a berry)

What medical condition is a berry aneurism associated with?

Polycystic kidney disease

What is a symptom of berry aneurysm rupture?

Third nerve palsy

What is the most common location for a berry aneurysm?

Anterior communicating artery (30%), followed by posterior communicating artery, then middle cerebral artery

What type of hemorrhage is associated with a lateral skull fracture?

Epidural hematoma

What artery is involved in an epidural hematoma?

Middle meningeal artery

What is the sequence of events in an epidural hematoma?

The patient has a lucid interval lasting from minutes to hours followed by a loss of consciousness and hemiparesis.

What can cause a “blown” pupil in a patient with an epidural hematoma?

Uncal herniation

What is seen on CT in a patient with an epidural hematoma?

Biconcave (lens shaped) hyperdensitythat does**not** cross the midline.

What is the treatment for an epidural hematoma?

Surgical evacuation of the hematoma via burr holes

What vessels are involved in a SAH?

Bridging veins

In what population are subdural hematomas most common?

The elderly and alcoholics

What is the course of events in a subdural hematoma?

Patient can have symptoms similar to dementia since mental status changes and hemiparesis can present subacutely.

What is seen on CT in a patient with a subdural hematoma?

Crescent-shaped, concave hyperdensitythat may cross the midline

VERTIGO

What type of vertigo is characterized by horizontal nystagmus?

Peripheral vertigo

What type of vertigo is characterized by vertical nystagmus?

Central vertigo

What is the most common cause of vertigo?

Benign positional vertigo

What are the signs of benign positional vertigo?

Sudden, episodic vertigo that occurs with quick head movement and lasts for**seconds**

How is benign positional vertigo diagnosed?

Dix-Hallpike maneuver—have the patient go from sitting to the supine position while quickly turning the head to the side

What is the etiology of Meniere disease?

Excess endolymph causes dilation of the membranous labyrinth

What is the triad of symptoms?

1. Tinnitus

2. Hearing loss

3. Episodic vertigo lasting**hours**

What does audiometry show in Meniere disease?

Low-frequency pure-tone hearing loss

What is the treatment for Meniere disease?

Low salt intake and acetazolamide. If acute, you could use antihistamines, anticholinergics, or antiemetics. Surgery may be necessary.

What type of vertigo follows a viral respiratory illness?

Viral labyrinthitis

How long does the vertigo last?

Days to weeks

What is the treatment for viral labyrinthitis?

Meclizine

CHAPTER 5

Gastroenterology

ESOPHAGEAL DISORDERS

What is dysphagia?

Difficulty swallowing

What is odynophagia?

Pain with swallowing

How does oropharyngeal dysphagia present?

More difficulty initiating the swallowing of liquids than solids

How does esophageal dysphagia present in terms of swallowing?

Difficulty swallowing both liquids and solids

What are the causes of oropharyngeal dysphagia?

Neurologic disorders (muscular, cranial nerve diseases), Zenker diverticulum,

thyromegaly, sphincter dysfunction, oropharyngeal cancers

What is a Zenker diverticulum?

Pharyngeal or esophageal outpouching of the muscular wall. Pulsion diverticulum secondary to pressure from swallowing.

What are the signs and symptoms of Zenker diverticulum?

Halitosis, neck mass on the left, dysphagia, aspiration

How is Zenker diverticulum diagnosed?

Clinical palpation of a left-sided neck mass or a barium swallow

What is the treatment for Zenker diverticulum?

Cricopharyngeal myotomy or surgical excision

What are the causes of esophageal dysphagia?

1. Mechanical obstruction: esophageal cancer, Schatzki ring, peptic stricture

2. Problem with esophageal motility: achalasia, diffuse esophageal spasm, or scleroderma

How do symptoms of mechanical dysphagia differ from dysphagia secondary to motility problems?

Patients with mechanical dysphagia have more difficulty with solids than liquids whereas motility disorders cause difficulty with both solids and liquids.

What is the most common motility disorder often seen in patients with scleroderma?

Esophageal hypomotility

What defines achalasia?

Loss of esophageal peristalsis with an inability of the lower esophageal sphincter to relax due to ganglionic loss of**Auerbach plexus.**

What is the diagnostic feature seen on barium swallow in a patient with achalasia?

"**Bird's beak**" appearance (dilation of the proximal esophagus with narrowing of the distal esophagus)

What would manometry demonstrate in a patient with achalasia?

Increased pressure at the lower esophageal sphincter with no relaxation with swallowing

How is achalasia treated?

Balloon dilatation, sphincter myotomy, local botulinum toxin

What is the diagnostic feature seen on barium swallow in a patient with diffuse esophageal spasm?

"**Corkscrew pattern**"

What is the treatment for diffuse esophageal spasm?

Nitroglycerin, calcium channel blockers

What is Schatzki ring?

Narrowing of the lower esophageal ring

What is Plummer-Vinson syndrome?

Esophageal webs, atrophic glossitis, and dysphagia associated with iron deficiency anemia

GASTROESOPHAGEAL REFLUX DISEASE

What are the underlying causes of gastroesophageal reflux disease (GERD)?

Incompetent lower esophageal sphincter, obesity, hiatal hernia, pregnancy, decreased esophageal motility, delayed gastric emptying

What are the signs and symptoms of GERD?

Postprandial epigastric (chest) burning worse in supine position, cough, hoarse voice, regurgitation

How is GERD diagnosed?

It is a clinical diagnosis.

What is the treatment for GERD?

First-line treatment is lifestyle modification; weight loss; avoidance of instigating foods such as caffeine, fatty foods; avoid eating right before going to sleep Second-line treatment: H2 blockers Third-line treatment: If H2 blockers fail, try proton pump inhibitors (PPIs) Last resort: Nissen fundoplication

What can be the long-term effects of chronic GERD?

Barrett esophagus, peptic stricture, and esophageal cancer

What is Barrett esophagus?

Transformation of normal squamous epithelium to columnar epithelium

What is the risk with Barrett esophagus?

10% lifetime risk of transforming into esophageal adenocarcinoma

GASTRITIS

What is gastritis?

Inflammation of the gastric mucosa

Name the type of gastritis that matches the statement below:

Gastritis most likely to be found in the fundus	Type A
Gastritis most likely to be found in the antrum of the stomach	Type B
Associated with autoimmune causes, achlorhydria, pernicious anemia	**Type A**
Most common cause is nonsteroidal anti-inflammatory drug (NSAID) use	Type B
Can be caused by *Helicobacter pylori* infection	Type B
Associated with risk for peptic ulcer disease and gastric cancer	Type B

What are the signs and symptoms of gastritis?

May be asymptomatic; otherwise symptoms are epigastric pain, weight loss, nausea, vomiting, hematemesis,**coffee ground emesis**

How is gastritis diagnosed?

Endoscopy

What is the treatment for gastritis?

It depends on the etiology.

If caused by*H. pylori* —triple therapy with PPI, two antibiotics, and bismuth compound

If caused by NSAID use—discontinue NSAID use; start sucralfate, PPI, or H2 blocker

If caused by stress—intravenous (IV) H2 blocker

If caused by pernicious anemia—vitamin B12treatment

PEPTIC ULCERS

What are the two types of peptic ulcers?

Duodenal ulcer and gastric ulcer

Which type of ulcer is more common?

Duodenal ulcers are twice as common

What is the underlying pathology in a patient with a duodenal ulcer?

Most have increased acid production

How does the underlying pathology of gastric ulcers differ from that of duodenal ulcers?

Gastric ulcers are not caused by increased acid production. Patients are more likely to have decreased mucosal protection.

What bacterial infection is found in 90% of patients with duodenal ulcers?

H. pylori

What percentage of gastric ulcers are associated with*H. pylori* infection?

70%

What test can determine if a patient is infected with*H. pylori?*

Stool*H. pylori* antigen, urea breath test, serum IgG test

What is the drawback of the*H. pylori* blood test?

It does not indicate an**active** infection. It will be positive even if the patient was infected in the past and is not currently infected. The test also has a low sensitivity.

What are the two most common causes of peptic ulcer disease?

*H. pylori*infection and frequent NSAID use

What are the risk factors for a peptic ulcer?

Smoking, significant alcohol use, frequent NSAID use, significant physiologic stress (examples are surgery, trauma, burns), and hypersecretory states

Name three hypersecretory states.

Zollinger-Ellison syndrome, multiple endocrine neoplasia type I (MEN I), and antral G cell hyperplasia

What are the signs and symptoms of a duodenal ulcer?

Burning epigastric pain that is usually 2–3 hours postprandially; relieved by food or antacids; pain may radiate to the back; pain awakens patient at night; nausea and sometimes vomiting; hematemesis/melena if patient has a gastrointestinal (GI) bleed

What are the signs and symptoms of a gastric ulcer?

Same as that for a duodenal ulcer except that pain is greater with meals, so patients often lose weight

What tests would you order if you suspected a peptic ulcer?

Complete blood count (CBC) to make sure patient is not anemic; upper GI endoscopy or upper GI series;*H. pylori* screening

What should be ruled out in a patient with a gastric ulcer?

Malignancy

How can malignancy be ruled out?

A biopsy of the ulcerated region should be done during endoscopy

What is a gastric ulcer in a burn patient called?

Curling ulcer

What is a gastric ulcer in a patient with central nervous system (CNS) damage called?

Cushing ulcer

How is peptic ulcer disease treated?

Avoidance of instigating factors such as smoking and NSAIDs; H2 blockers or PPIs mucosal protectors such as bismuth; and antibiotics if the patient is infected with*H. pylori*

How is an*H. pylori* infection treated?

Triple therapy:PPI + bismuth compound + two antibiotics for 14 days

For example: omeprazole + amoxicillin (or metronidazole) + clarithromycin + bismuth compound

What are some complications of peptic ulcer disease?

Hemorrhage, obstruction, perforation

When would you suspect a perforated duodenal ulcer?

Severe epigastric pain that radiates to the back

What studies would you order if you suspected a perforated ulcer?

Abdominal series or upper GI series with contrast (**do not use barium**)

What would you expect to see on an abdominal series if there was a perforated ulcer?

Free air under the diaphragm

What is the treatment for a perforated ulcer?

npo (nothing by mouth), IV fluids, antibiotics, emergent surgery

What are the typical symptoms of gastric outlet obstruction?

Nausea, vomiting, weight loss, distended abdomen

What is the most serious complication of a posterior duodenal ulcer?

Erosion into the gastroduodenal artery can lead to a massive hemorrhage.

What symptoms could be a red flag for a gastric malignancy?

Early satiety with weight loss

What are the risk factors for gastric cancer?

Diets with high nitrosamines or salt content, history of chronic gastritis, low fiber diets

What blood group type is more likely to develop gastric cancer?

Type A

In what part of the stomach is gastric cancer usually found?

In the antrum of the stomach

What is the most common type of gastric cancer?

Adenocarcinoma

Name the physical findings associated with metastatic gastric cancer described below:

Palpable supraclavicular lymph node	**Virchow node**
Hard lymph nodule palpable at the umbilicus	**Sister Mary Joseph sign**
Palpable ovarian mass that originates from the metastasis of signet ring cells	**Krukenberg tumor**
Lymph node that can be palpated on a rectal examination due to metastasis to the pouch of Douglas	Blumer shelf

What is the most fatal form of gastric cancer?

Linitis plastica (diffusely infiltrating gastric cancer)

GI BLEED

What are signs of an upper GI bleed?

Hematemesis, coffee ground emesis, melena (black, tarry stools), bright red blood per rectum (BRBPR) only if the bleed is very brisk

What are the six main causes of upper GI bleeds?

PAGE ME!

1. Peptic ulcer

2. Atrioventricular (AV) malformation

3. Gastritis

4. Esophageal varices

5. Mallory-Weiss tear

6. Esophagitis

What is a Mallory-Weiss tear?

Small esophageal tear usually near the gastroesophageal (GE) junction that is caused by vomiting or retching

What blood tests would you order in a patient you thought may have a

CBC (look for anemia, platelet abnormality), blood urea nitrogen (BUN)

GI bleed?

(fresh bleeding may lead to elevated BUN), prothrombin time (PT), partial thromboplastin time (PTT), international normalized ratio (INR), bleeding abnormalities

What is the best diagnostic test in a patient with upper GI bleed?

Endoscopy

How are bleeding varices treated?

Ligation or injection of vessels with sclerosing or vasoconstrictive agents

How should all GI bleeds be treated?

Emergency airway, breathing, circulation (ABCs) as well as IV fluid resuscitation, gastric lavage and nasogastric (NG) tube if needed

What are the signs of a lower GI bleed?

BRBPR, maroon or dark red stool, anemia

What are the six most common causes of lower GI bleeding?

1. Diverticulosis

2. AV malformation

3. Hemorrhoids

4. Colitis

5. Colon cancer

6. Colonic polyps

What is the most common cause of a major lower GI bleed in a patient over age 60?

Diverticulosis

What physical examination and imaging study would you do on a patient with suspected lower GI bleed?

Alwaysdo a rectal examination; colonoscopy

If no clear source is found, what other studies can be done?

Endoscopy to rule out an upper GI source, tagged red blood cell (RBC) scan; arteriography, gastric lavage; barium enema (but not if there is acute blood loss)

COLON

What is a true diverticulum?

Colonic herniation involving the full thickness of bowel wall

What is a false diverticulum?

Colonic mucosal herniation through the muscular layer which is acquired

Which type of diverticulum is more common?

False

In what part of the colon are diverticula most commonly found?

Sigmoid

What is diverticulosis?

Presence of multiple diverticula in the colon

What is thought to be an important risk factor for the development of diverticulosis?

Low-fiber diet

Why do diverticula bleed?

Diverticula which are inflamed erode through an artery and cause profuse bleeding that usually subsides on its own.

What is the treatment for diverticulosis?

Increase of fiber in diet and decrease of obstructing foods such as seeds and fatty foods

What is diverticulitis?

“Itis” implies inflammation. Diverticulitis is inflammation of a diverticulum secondary to infection.

What is the most common symptom of diverticulitis?

Most are asymptomatic, but the most common presenting symptom is**left lower quadrant abdominal pain.**

What are other signs and symptoms of diverticulitis?

Constipation, fever, elevated white blood cells (WBCs), bleeding is much less common than with diverticulosis

What are the four serious complications of diverticulitis?

1. Perforation through the bowel wall causing peritonitis

2. Fistula formation

3. Abscess

4. Obstruction

How do patients who develop a colovesicular fistula present?

Multiple urinary tract infections (UTIs)

What is the best imaging test to diagnose diverticulitis?

Computed tomography (CT) of the abdomen and pelvis

What studies are contraindicated in diverticulitis?

Colonoscopy, contrast enema

What is the treatment for diverticulitis?

npo, IV fluids, antibiotics to cover anaerobes and enteric organisms

What is the treatment for recurrent bouts of diverticulitis?

Elective sigmoid colectomy

How would you treat an abscess secondary to diverticulitis?

CT or ultrasound-guided percutaneous drainage

How do you treat obstruction or perforation secondary to diverticulitis?

Surgical resection of affected bowel with a colostomy that is usually temporary

What is the most common nosocomial enteric infection?

Clostridium difficile

What can a*C. difficile* infection lead to?

Pseudomembranous colitis

What antibiotic is classically associated

Clindamycin

What are the symptoms of*C. difficile* infection?

Diarrhea and abdominal cramping/pain

How is a*C. difficile* infection diagnosed?

*C. difficile*stool toxin, stool leukocytes

How is a*C. difficile* infection treated?

Stop the offending agent and treat with po metronidazole or vancomycin.

How is pseudomembranous colitis confirmed?

On colonoscopy or sigmoidoscopy, a yellow plaque adherent to the colonic mucosa can be seen.

What is volvulus?

Twisting of the bowel around the mesenteric base

What is the most common location of volvulus?

Sigmoid colon

What is the second most common location of volvulus?

Cecum

What are the symptoms of a volvulus?

Painful, distended abdomen; high-pitched bowel sounds; tympany on percussion

What is the classic sign of volvulus on an abdominal series?

Dilated loops of bowel with a**kidney bean** appearance

What is the sign of volvulus on a barium enema?

Bird’s beak appearance with the beak pointing to the area where the rotation has occurred

What is the treatment for volvulus?

Sigmoidoscopy or colonoscopy is usually therapeutic for decompression.

What is the second most common cancer causing death in the United States?

Colon cancer

What are the risk factors for colon cancer?

Family history

Low-fiber diet

Familial adenomatous polyposis (FAP)

Hereditary nonpolyposis colorectal cancer

High-fat diet

Colonic adenomas

Age > 50

Inflammatory bowel disease

What are the general signs and symptoms of colon cancer?

Weight loss, fatigue, iron deficiency anemia in a male > 50 years of age is colon cancer (CA) until proven otherwise; GI bleed, constipation, distended abdomen secondary to obstruction, pencil thin stools

How do the symptoms of right-sided-and left-sided colon cancer differ?

Left-sided colon cancer presents as constipation.

Right-sided colon cancer presents as anemia secondary to blood loss.

What are the recommendations for colon cancer screening?

Starting age 50, a colonoscopy every 10 years or a sigmoidoscopy every 5 years with annual digital rectal and hemoccult examination.

How are the screening recommendations different in patients with a family history of colon cancer?

Start screening 10 years prior to the age that the family member was diagnosed with cancer.

How is colon cancer diagnosed?

Biopsy of the lesion on colonoscopy/sigmoidoscopy

What laboratory marker can be used to help follow the progression of colon cancer and its treatments?

Carcinoembryonic antigen (CEA)—but it cannot be used as a screening test

How is colon cancer staged and what is the prognosis of each stage?

TNM (tumor node metastasis) classification (Table 5-1aand5-1b)

Table 5-1a Colon Cancer Staging

Staging of Primary Tumor	Nodal Involvement	Metastasis
Tis: Carcinoma in situ	N0: No regional node involvement	M0: No metastasis
T1: Tumor invades submucosa	N1: Metastasis in one to three regional lymph nodes	M1: Distant metastasis present
T2: Tumor invades muscularis propria	N2: Metastasis in four or more regional lymph nodes	
T3: Tumor invades the subserosa or into the nonperitoneal pericolic or perirectal tissues		
T4: Tumor perforates the visceral peritoneum or directly invades other organs		

Table 5-1b Colon Cancer Prognosis Based on Staging

Stage	T	N	M	Approximate 5-year prognosis
Stage 0	Tis	N0	M0	> 90%
Stage I	T1	N0	M0	> 90%
	T2	N0	M0	
Stage II A	T3	N0	M0	70%–85%
II B	T4	N0	M0	55%–65%
Stage III A	T1, T2	N1	M0	45%–55%
III B	T3, T4	N1	M0	20%–35%
III C	Any T	N2	M0	
Stage IV	Any T	Any N	M1	< 5%

What is the treatment of colon cancer?

Surgical resection; radiation therapy (if rectal cancer), and chemotherapy for stages B and C

INFLAMMATORY BOWEL

What is ulcerative colitis (UC)?

Inflammatory bowel disease that affects the colon

What classic symptom is associated with UC?

Bloody diarrhea

What other serious symptom can sometimes occur with UC?

Toxic megacolon

Where are lesions found in UC?

Large intestine only

Where do the lesions usually first appear?

Rectum

How do lesions spread in UC?

Proximally from the rectum

How is UC diagnosed?

Colonoscopy with biopsy

What is seen on colonoscopic biopsy in a patient with UC?

Crypt abscess; distorted cells

How is the mucosa of the colon described in a patient with UC?

Friable mucosa with erosions and erythema

On biopsy, what is the depth of involvement of the lesions?

Mucosa and submucosa only

What is ulcerative proctitis?

A subtype of UC in which only the rectum is involved

What is the treatment for each of the following severities of UC:

Distal colitis (mild)?	**Mesalamine**
Moderate colitis?	Mesalamine + **sulfasalazine** ± corticosteroids
Severe colitis?	IV corticosteroids + cyclosporine; unresponsive cases require resection
Fulminant colitis?	Broad spectrum antibiotics, surgery

What is Crohn disease?

Inflammatory bowel disease that affects the GI tract; there could be an infectious etiology

What part of the GI tract can Crohn disease involve?

From the mouth to the rectum, but often with rectal sparing

What is the classic symptom of Crohn disease?

Bloodyor**watery diarrhea** (although the diarrhea does not always have to be bloody)

What are some other physical examination findings in Crohn disease?

Fistulas, fissures, fever, abdominal pain

How is Crohn disease diagnosed?

Colonoscopy and biopsy

How are the lesions classically spread in Crohn disease?

There are**skip lesions,** which means that there is no contiguous spread. The lesions are disseminated through the entire colon.

What is the depth of the lesions on biopsy?

Lesions go through all layers—they are**transmural.**

On physical examination, what type of lesion is often found in the mouth of a patient with Crohn disease?

Aphthous ulcer

What is the mnemonic to remember Crohn disease?

The old,**fat Crohn skipped** over the**cobblestone.**

What is the treatment for Crohn disease?

Sulfasalazine, corticosteroids; for unresponsive patients, try mercaptopurine, azathioprine, infliximab

What are the differences between UC and Crohn disease?

(Table 5-2)

Name six extraintestinal manifestations of both UC and Crohn disease?

1. Erythema nodosum

2. Pyoderma gangrenosum

3. Uveitis

4.**Ankylosing spondylitis**

5. Primary sclerosing cholangitis

6. Arthritis

Table 5-2 Crohn Disease versus Ulcerative Colitis

Crohn Disease	Ulcerative Colitis
Lesions in small and large intestine	Lesions only in large intestine
Rectal involvement uncommon	Rectal involvement common
Transmural	Submucosa/mucosa only
Skip lesions	Lesions are contiguous
Fissures and fistulas common	No fissures or fistulas
Lower risk for colon cancer	High risk for colon cancer

DIARRHEA

What is the definition of diarrhea?

Daily stool weighing > 200 g

What are the most common causes of bacterial and parasitic bloody diarrhea?

wh**Y CaSES**

Y*ersinia*

C*ampylobacter*, cholera

S*higella*

E*scherichia coli*,***E****ntamoeba histolytica*

S*almonella*

What is the treatment for bacterial bloody diarrhea?

Ciprofloxacin or bactrim

What are viral causes of bloody diarrhea?

Rotavirus and Norwalk virus

What is the treatment for bloody diarrhea caused by a virus?

IV fluids

What is the treatment for parasitic bloody diarrhea?

Metronidazole

What studies would you order in a patient with bloody diarrhea?

CBC, stool for ova and parasites, stool for fecal leukocytes, stool culture

What acid-base disorder can you expect to see in a patient with severe diarrhea?

Metabolic alkalosis

MALABSORPTION DISORDERS

Name the malabsorption disorder described below:

Gluten-induced enteropathy	Celiac sprue
Caused by tropical infection	Tropical sprue
Protein losing enteropathy with large gastric folds seen on barium swallow	Menetrier disease
Most common malabsorptive disorder of adulthood	Lactase deficiency
Caused by infection with *Tropheryma whippelii*, a gram-negative rod	Whipple disease
Affects the jejunum	Tropical sprue
Diagnosed with antigliadin IgG and IgA antibodies, endomysial antibody, antireticulin antibody; and small bowel biopsy shows blunting of intestinal villi	Celiac sprue
Periodic acid-Schiff (PAS) + macrophages in intestines	Whipple disease

Classic rash of dermatitis herpetiformis	Celiac sprue
Causes signs and symptoms of folic acid deficiency including cheilosis, glossitis, stomatitis	Tropical sprue
Flatulence after consumption of lactose-containing products	Lactase deficiency
Signs and symptoms include hyperpigmentation, arthralgias, rash, diarrhea, endocarditis, ophthalmoplegia, memory deficits, and altered mental status	Whipple disease
Avoidance of wheat, rye, and barley will help treat the disorder	Celiac sprue
Treated with penicillin	Whipple disease

PANCREAS

What is pancreatitis?

Inflammation of the pancreas

What are the two most common causes of pancreatitis?

Alcoholic pancreatitis and gallstone pancreatitis

What is the mnemonic for the causes of pancreatitis?

I GET SMASHED

Idiopathic

Gallstones

Ethanol

Trauma

Steroids

Mumps

Autoimmune

Scorpion bites

Hyperlipidemia

Endoscopic retrograde cholangiopancreatography (ERCP)

Drugs (such as thiazide diuretics)

What are the signs and symptoms of pancreatitis?

Epigastric pain that radiates to the back;nausea, vomiting, decreased bowel sounds, fever

What is Grey Turner sign?

Ecchymoses seen on the patient flank in hemorrhagic pancreatitis

What is Cullen sign?

Periumbilical ecchymosis seen in hemorrhagic pancreatitis

What laboratory findings are consistent with pancreatitis?

? amylase,? lipase, hypocalcemia

What would you expect to see on an abdominal x-ray?

Sentinel loop or colon cutoff sign

What is a sentinel loop?

Dilated bowel or air fluid levels near the pancreas

What is the colon cutoff sign?

Transverse colon distended with no colonic gas distal to the splenic flexure

What is the best study to evaluate pancreatitis?

Abdominal CT

What test should be ordered if there is a suspicion of gallstone pancreatitis?

Right upper quadrant (RUQ) ultrasound

What is the treatment for pancreatitis?

npo, NG tube for ileus or vomiting, IV fluid hydration, and treat the underlying cause

What do we use to determine the prognosis of a patient with pancreatitis?

Ranson criteria (predicts risk of mortality based on risk factors)

What are Ranson criteria on admission?

Remember the mnemonic**GA LAW**

Glucose > 200

Age > 55

Lactate dehydrogenase (LDH) > 350

Aspartate aminotransferase (AST) > 250

WBC > 16,000

What are Ranson criteria after 48 hours?

Remember the mnemonic:**C & HOBBS**

Calcium < 8

Hematocrit (Hct) drop > 10%

Oxygen < 60 mm

BUN > 5

Base deficit > 4

Sequestration of fluid > 6 L

How is the risk of mortality calculated based on Ranson criteria?

< 3 risk factors: 1% mortality

3–4 risk factors: 16% mortality

5–6 risk factors: 40% mortality

7–8 risk factors: close to 100% mortality

BILIARY TRACT

What is cholelithiasis?

Gallstones

What are the four classic risk factors for cholelithiasis?

Female, fat, fertile, and forty

What is the most common type of stone?

Cholesterol stone

What other type of stone can be found?

Pigment stone

What is the predisposition to pigment stones?

Hemolytic anemia or hemoglobinopathies

Which type of stone is radiopaque?

Pigment stones

What are the common signs and symptoms of cholelithiasis?

RUQ pain, nausea, and vomiting especially after a fatty meal

What is the most specific and sensitive test to diagnose cholelithiasis?

RUQ ultrasound

When should cholelithiasis be treated?

Only if the patient is symptomatic

What is the treatment for cholelitihiasis?

Elective cholecystectomy

What is cholecystitis?

Gallbladder inflammation secondary to infection caused by an obstructing stone

What bacteria cause cholecystitis?

KEEEP

K*lebsiella*

***E**.coli*

Enterococcus

Enterobacter

Pseudomonas

What are the symptoms of cholecystitis?

Prolonged RUQ pain, fever,nausea, vomiting, referred pain to subscapular region on the right +**Murphy sign**

What is Murphy sign?

Acute pain and inspiratory arrest with deep palpation of RUQ during inspiration

How is cholecystitis diagnosed?

RUQ ultrasound will show gallstones, gallbladder wall thickening, and pericholecystic fluid, and sonographic Murphy's sign

What imaging study should be performed if the ultrasound results are equivocal?

Hepatobiliary iminodiacetic acid (HIDA) scan

What is the treatment for cholecystitis?

npo, IV fluids, IV antibiotics (third-generation cephalosporin + aminoglycoside + metronidazole, cholecystectomy

What pain medicine has historically been referred to as being more appropriate to treat pain from cholecystitis and why?

Demerol because morphine is thought to cause spasm of the sphincter of Oddi; however, this is not always done in clinical practice

What is choledocholithiasis?

Gallstones in the common bile duct

What are the signs and symptoms of choledocholithiasis/cholangitis?

Jaundice secondary to obstruction, RUQ pain, Murphy's sign, hypercholesterolemia,? alkaline phosphatase, ? bilirubin, ? alanine aminotransferase (ALT)

What is the treatment for choledocholithiasis?

1. ERCP with papillotomy and stone removal

2. Common bile duct exploration at time of surgery

What are the complications of choledocholithiasis?

Ascending cholangitis and pancreatitis

What is ascending cholangitis?

Bacterial infection of the biliary tract secondary to obstruction

What is the most common organism causing cholangitis?

E. coli

What is Courvoisier sign?

Gallbladder enlargement with jaundice secondary to carcinoma of the head of the pancreas leading to a firm palpable gallbladder

What are the classic symptoms of ascending cholangitis?

Charcot triad:

1. Jaundice

2. Fever

3. RUQ tenderness

OR

Reynold pentad (Charcot triad+altered mental status and shock)

What are the laboratory findings consistent with ascending cholangitis?

? WBC, ? alkaline phosphatase, ? direct bilirubin, ? ALT

How is ascending cholangitis definitively diagnosed?

ERCP or percutaneous transhepatic cholangiogram (PTC)

What is the treatment for ascending cholangitis?

npo, IV fluids, IV antibiotics (ampicillin + aminoglycoside + metronidazole), and ERCP to remove stones

What is primary sclerosing cholangitis?

Chronic inflammation and fibrosis of the biliary tree

What is a common medical diagnosis that patients with sclerosing cholangitis also have?

UC

LIVER

What is cirrhosis?

Chronic hepatic injury leading to fibrosis, necrosis, and nodular regeneration

What is the most common cause of cirrhosis?

Alcoholism

What are some nonalcoholic causes of cirrhosis?

Alpha-1 antitrypsin deficiency, hemochromatosis, primary or secondary biliary cirrhosis, Wilson disease, hepatitis B, hepatitis C

What are the signs and symptoms of cirrhosis?

Jaundice, ascites, asterixis, bleeding, edema, hepatomegaly, encephalopathy, palmar erythema, spider angiomata on the abdomen

What is asterixis?

Downward flapping of hands when held in a dorsiflexed position

Why do cirrhotic patients get ascites?

Because they have low albumin.

How can the ascite be treated?

Spironolactone and paracentesis

What is a major complication of ascites?

Spontaneous bacterial peritonitis (SBP)

What is the most common organism causing SBP?

E. coli

What is the most classic sign of SBP?

Rebound abdominal tenderness in a patient with ascites

How is SBP diagnosed?

Paracentesis with fluid sent for cell count and Gram stain, culture, and sensitivity

What are the diagnostic criteria for SBP?

Neutrophil count > 250 or positive Gram stain or culture

What is the treatment for SBP?

Third-generation cephalosporin with albumin

Why do cirrhotic patients tend to bleed?

PT is elevated and platelets are low

What is the treatment for cirrhosis?

Stop alcohol consumption, multivitamin including thiamine and B12, nutrition

What marker can detect an alcohol binge?

Gamma-glutamyltransferase (GGT)

What is portal hypertension?

Elevated portal vascular resistance secondary to presinusoidal, postsinusoidal, or sinusoidal obstruction

Presinusoidal: portal vein thrombosis, schistosomiasis

Postsinusoidal: hepatic vein thrombosis, right heart failure

Sinusoidal: cirrhosis

Internationally, what is the most common cause of portal hypertension?

Schistosomiasis

What are the classic physical examination findings in a patient with portal hypertension?

CHASE:

Caput medusa

Hemorrhoids

Ascites

Splenomegaly

Esophageal varices

What are the treatments for portal hypertension?

Decrease portal pressure with propranolol; transjugular intrahepatic portosystemic shunt (TIPS); last resort is a liver transplant

What is a common cause of hematemesis in a patient with portal hypertension?

Varicial bleeding

How is a variceal bleed diagnosed?

Esophagogastroduodenoscopy (EGD)

What is the treatment for a variceal bleed?

IV fluids, fresh frozen plasma (FFP), vasopressin, sclerotherapy on banding or the varices, balloon tamponade, propranolol (although not acutely given)

What are some treatments for hepatic encephalopathy?

Lactulose to decrease absorption of ammonia, neomycin, and protein-restricted diet

What is hepatorenal syndrome?

Patients with advanced hepatic disease develop acute renal failure.

How is hepatorenal syndrome diagnosed?

Elevated BUN/creatinine (CR), hyponatremia, oliguria, hypotension, and urine Na< 10

What are the three different etiologic categories of hepatitis?

1. Viral

2. Alcoholic

3. Toxin-induced (Tylenol)

Name the hepatitis viruses transmitted via the fecal-oral route.

Hepatitis A and E

Name the hepatitis viruses transmitted via blood and sexual contact.

Hepatitis B, C, D

Name the only DNA hepatic virus.

Hepatitis B

Which hepatitis viruses have a chronic carrier state?

Hepatitis B, C, D

Which hepatitis viruses have a vaccine available?

Hepatitis A and B (and D)

How can you detect an acute hepatitis A infection?

Anti-hepatitis A virus (HAV) IgM

How can you detect immunity to hepatitis A?

Anti-HAV IgG

How is hepatitis A treated?

It is a self-limiting disease.

Which disease state do each of the following hepatitis B markers detect?

HBsAg (hepatitis B surface antigen)	Active hepatitis or carrier
HBeAg	Chronic hepatitis that is highly infective
HBcAg	Early infection
Anti-HBc IgM	Acute infection (1.5–6 months)
Anti-HBe	Very low infectivity
Anti-HBs	Immune state
Anti-HBc IgG	Remote infection from 6 months to 1 year ago

What can be given to a patient exposed to hepatitis B to prevent infection?

Hepatitis B immunoglobulin (HBIG)

What is the treatment for a person infected with hepatitis B?

Interferon, lamivudine, adefovir

When is the window period for hepatitis B?

The time when HBsAg has become undetectable but HBsAb is not yet detectable

What is the worst complication of hepatitis B?

Hepatocellular carcinoma

Which hepatitis virus carries the highest risk of developing into hepatocellular carcinoma?

Hepatitis B

What is the treatment for a person infected with hepatitis C?

Interferon + ribavarin

Which hepatitis virus must have concomitant infection with hepatitis B?

Hepatitis D

CHAPTER 6

Hematology–Oncology

ANEMIA

What are the three basic categories of anemia?

1. Microcytic (mean corpuscular volume [MCV] <80)

2. Macrocytic (MCV>100)

3. Normocytic (MCV between 80 and 100)

Match the following anemias with their correct category (microcytic, macrocytic, or normocytic):

SeeTable 6.1 .

Iron deficiency anemia

Thalassemia

Folate deficiency

Sideroblastic anemia

Anemia of chronic disease

Lead poisoning

B12deficiency

Chronic renal failure

Table 6-1 Etiologies of Different Types of Anemia

Microcytic (Mnemonic: LISTS)	Macrocytic	Normocytic
Lead poisoning	B_{12} deficiency	Chronic disease
Iron deficiency	Folate deficiency	Renal failure
Sickle cell anemia		Aplastic anemia
Thalassemias		Spherocytosis
Sideroblastic		Autoimmune destruction
		Mechanical destruction

MICROCYTIC ANEMIAS

What is the most likely etiology of iron deficiency anemia?

In women of childbearing age, it is most likely because of menses; in children it is usually a dietary deficiency; and in the elderly it is colon cancer until proven otherwise.

A 68-year-old man with iron deficiency anemia presents to your clinic and denies any hematochezia or melena. What is the first thing you would do?

Screen for colon cancer (iron deficiency anemia in the older population is cancer until proven otherwise.)

What are the symptoms for iron deficiency anemia?

Pallor, tachycardia, easy fatigability,**Pica, esophageal webs**

What is the triad for Plummer-Vinson?

1. Microcytosis

2. Atrophic glossitis

3. Esophageal webs

What are the laboratory findings seen in iron deficiency anemia?

? iron, ? ferritin, ?**total iron-binding capacity (TIBC)**

(**Think:**Since there is less iron in the body, there is greater capacity for binding iron.)

How is iron deficiency anemia treated?

Ferrous sulfate

What is sideroblastic anemia?

Anemia caused by a disorder of the porphyrin pathway leading to ineffective erythropoiesis

What drugs commonly cause sideroblastic anemia?

Isoniazid; chloramphenicol, copper chelators, lead

What are some other causes of sideroblastic anemia?

Alcoholism, heredity

What are laboratory findings?

?**Iron,** ? ferritin, ?**TIBC**

How is it diagnosed?

Iron stain of bone marrow shows ringed sideroblasts

What is the treatment?

Withdraw the offending agent, if one is identified, and give pyridoxine (B6)

What type of anemia is sickle cell anemia?

Microcytic

What kind of genetic inheritance pattern does sickle cell anemia exhibit?

It is an autosomal recessive disorder

What causes “sickling” of red blood cells (RBCs)?

Hemoglobin S tetramer polymerizes when RBCs are deoxygenated

What are some signs and symptoms of sickle cell anemia?

Pain crisiscaused by vaso-occlusion

Infarcts of the lungs, kidneys, bone, spleen

Intravascular hemolysis

Osteomyelitis caused by*Salmonella*

Aplastic anemia from parvovirus B19 infection

Myocardiopathy

“Fish mouth” vertebrae

What kind of infection are sickle cell patients with an autosplenectomy at risk for?

Infection with**encapsulated bacteria** which include pneumococcus, meningococcus, and*Haemophilus influenzae*

What can intravascular hemolysis lead to in children?

Gallstones

How is sickle cell anemia diagnosed?

Hemoglobin electrophoresis shows hemoglobin S.

How is sickle cell treated?

Remember the mnemonic HOP:

Hydroxyurea—to prevent pain crises

Oxygen—to prevent sickling of cells

Pneumococcal vaccine

What are thalassemias?

Hereditary diseases in which there is a decreased production of globins causing a decrease in the production of hemoglobin

What causes alpha-thalassemia?

A decrease in the alpha-globin chain production. There are four alpha alleles and anywhere from one to all four of these alleles may be affected.

Match the alpha-thalassemia to the correct number of affected alleles and all the matching characterstics.

See Table 6.2 .

α-Thalassemia minor	One affected allele	Hemoglobin Barts (β_4 hemoglobin)
Carrier	Two affected alleles	Mild microcytic anemia
Hydrops fetalis	Three affected alleles	Asymptomatic, no anemia
Hgb H disease	Four affected alleles	Fetal demise
		Intraerythrocytic inclusions

Table 6-2 Alpha-thalassemia

	Alleles	Characteristics
Carrier	One allele affected	Asymptomatic
α-Thalassemia minor	Two alleles affected	Mild microcytic anemia
Hgb H disease	Three alleles affected	Intraerythrocytic inclusions
Hydrops fetalis	All four alleles affected	Barts; fetal demise

In what ethnicity is alpha-thalassemia most likely to be found?

More common in Asians. Also seen in people of Mediterranean and African decent.

What causes beta-thalassemia?

A decrease in the synthesis of one or both of the beta-chains (there are two beta-chains in hemoglobin.)

In what ethnicities is beta-thalassemia most likely to be found?

African and Mediterranean descent

Match the description below to the correct beta-thalassemia:

Missing both beta chains	Beta-thalassemia major
Missing one beta chain	Beta-thalassemia minor
Asymptomatic	Beta-thalassemia minor
Splenomegaly, frontal bossing, iron overload	Beta-thalassemia major
Treatment is folate supplementation	Beta-thalassemia major
Avoid oxidative stress	Beta-thalassemia minor
Electrophoresis shows increased fetal hemoglobin (Hgb F)	Beta-thalassemia major

How is beta-thalassemia definitively diagnosed?

Through gel electrophoresis.**Thalassemia major will have increased levels of Hgb F** as well as very decreased Hgb A; while thalassemia minor will have normal levels of Hgb F with somewhat decreased Hgb A.

MACROCYTIC ANEMIAS

What are five different etiologies of macrocytic anemia?

1. Folate deficiency

2. Vitamin B12deficiency

3. Alcoholism

4. Liver disease

5. Hypothyroidism

Where is vitamin B12absorbed?

In the terminal ileum

What factor is needed for vitamin B12absorption?

Intrinsic factor

What are the signs and symptoms of vitamin B12deficiency?

Neurologic symptomssuch as ataxia, parasthesias, demyelination of corticospinal tract and dorsal columns. Memory problems can also develop.

What is the most common cause of vitamin B12deficiency?

Pernicious anemia

What is the underlying pathology in pernicious anemia?

In pernicious anemia, there is a decreased production of intrinsic factor because the gastric parietal cells are destroyed by autoantibodies; there is atrophic gastritis.

How is pernicious anemia diagnosed?

? Methylmalonic acid

? Homocysteine levels

Abnormal Schilling test(not used as much any more)

What are other causes of vitamin B12deficiency?

Malabsorption because of resection of the terminal ileum or gastric resection, celiac sprue, Crohn disease, infection with*Diphyllobothrium latum* or*Giardia lamblia* . Rarely, B12deficiency is due to hypoalimentation. This can be seen in strict vegetarians or alcoholics.

How is vitamin B12deficiency treated?

Vitamin B12supplementation

What foods contain folic acid?

Green leafy vegetables

What is the most common cause of folate deficiency?

Hypoalimentation

What are other causes of folate deficiency?

Pregnancy, tropical sprue, hemolytic anemia, long-term treatment with bactrim

What can folate deficiency in pregnancy cause?

Neural tube defects in the developing fetus

How can the diagnosis of folate deficiency be differentiated from that of B12deficiency?

Normal methylmalonic acid

? Homocysteine levels

No neurologic symptoms

NORMOCYTIC ANEMIA

What are the most common causes of normocytic anemia?

Anemia of chronic disease, aplastic anemia, renal disease, hemolytic anemia, acute blood loss

How is anemia of chronic disease diagnosed?

? Iron; ?**TIBC,** normal ferritin

Why does renal failure cause anemia?

Erythropoietin is produced by the kidneys, and in chronic renal failure, erythropoietin levels are low.

What is the treatment of anemia in a patient with renal failure?

Erythropoietin supplementation

What is aplastic anemia?

Bone marrow failure leading to**pancytopenia**

Name six different etiologies of aplastic anemia.

1. Parvovirus B19 in the presence of sickle cell anemia

2. Hepatitis

3. Chloramphenicol

4. Benzene

5. Radiation therapy

6. Idiopathic

How is aplastic anemia diagnosed?

Normocytic, normochromic pancytopenia; hypocellular bone marrow in a bone marrow biopsy

What are the two main treatments for aplastic anemia?

Bone marrow transplant; immunosuppression

What is the most common enzyme deficiency that causes hemolytic anemia?

G6PD deficiency

Why is G6PD important?

It is part of the hexose monophosphate pathway which reduces glutathione which is used to protect RBCs against oxidative damage

How is G6PD deficiency genetically transferred?

It is sex linked

In what ethnicities is G6PD deficiency most common?

Sephardic Jews, Mediterraneans, Middle Easterners, Africans, Asians

What are the signs and symptoms of G6PD deficiency?

Signs of hemolysis which include dark urine, jaundice, weakness, pallor, abdominal and back pain due to mesenteric/renal ischemia, hepatosplenomegaly

What can trigger an attack in a patient with G6PD deficiency?

Infection,**fava beans,** dapsone, sulfa drugs, primaquine, nonsteroidal anti-inflammatory drugs (NSAIDs)

What is the pathopneumonic G6PD deficiency diagnostic feature?

Peripheral smear shows**Heinz bodies, schistocytes**

What is the treatment for anemia caused by G6PD deficiency?

It is usually self-limited. Remove inciting factors such as drugs. Transfuse only in very severe cases.

What two infections are associated with cold autoimmune hemolytic anemia?

*Mycoplasma*pneumonia and mononucleosis

Cold autoimmune hemolytic anemia is mediated by which immunoglobulin (lg)?

IgM

How is cold autoimmune hemolytic anemia diagnosed?

A positive cold agglutinin test or positive indirect Coombs test

How is cold autoimmune hemolytic anemia treated?

Staying warm as well as immunosuppresives

What general lab results would be seen in hemolytic anemia?

Unconjugated bilirubinemia, hemoglobinuria, elevated urine urobilinogen

COAGULOPATHIES

What does partial thromboplastin time (PTT) measure?

Intrinsic pathway

What does prothrombin time (PT) measure?

Extrinsic and common pathway

Which pathway does heparin affect?

Intrinsic pathway

Which pathway does warfarin affect?

Extrinsic pathway

What are some causes of PT elevation?

Warfarin treatment, vitamin K deficiency, liver disease

What are the many causes of thrombocytopenia?

Two categories:

1. ? Destruction/sequestration Platelet disorders: TTP, ITP, DIC, HUS Splenomegaly Drugs (heparin, aspirin, chemotherapy)

2. ? Production Leukemia Liver disease/alcohol Aplastic anemia

At what platelet level does significant bleeding begin?

20,000

At what platelet level is a patient at risk for an intercranial bleed?

10,000

At what platelet level is there an increased risk for bleeding?

50,000

Name the platelet disorder associated with the following features:

Autoimmune-mediated platelet destruction; often occurs after a viral infection in children and is self-limited, can be chronic in adults	ITP
Triad of thrombocytopenia, hemolytic anemia, and acute renal failure	HUS
Often in children with bloody diarrhea infected by *Escherichia coli*	HUS
Pentad of fever, anemia, thrombocytopenia, renal failure, and neurological changes	TTP; (Note: **FAT RN**—Fever, **A**nemia, **T**hrombocytopenia, **R**enal failure, **N**eurological changes)
Seen in adenocarcinoma, trauma, septic shock, leukemia	DIC
Often associated with human immunodeficiency virus (HIV), malignancy, autoimmune disorders, pregnancy	TTP
Petechiae and purpura over trunk and limbs	TTP, ITP, DIC
Caused by the deposition of abnormal von Willebrand factor (vWF) multimers	TTP

Describe how each of the following platelet disorders can be diagnosed:

TTP	Pentad of **F**ever, **A**nemia, **T**hrombocytopenia, **R**enal failure, **N**eurologic changes in addition to peripheral smear with **schistocytosis, helmet cells;** ↓ haptoglobin, ↑ lactate dehydrogenase (LDH); may have ↑ blood urea nitrogen/creatinine (BUN/CR), ↑ unconjugated bilirubin
ITP	Diagnosis of exclusion; no fever as in TTP; no schistocytosis on peripheral smear; positive Coombs test
DIC	↑ **Fibrin split products,** ↑ **D-dimer,** ↓ fibrinogen, ↑ PT/PTT, ↓ hematocrit
HUS	Stool is hemoccult positive, ↑ BUN/CR, peripheral smear with schistocytosis, helmet cells; clinically different from TTP because there is no change in mental status

What is the treatment for each of the following platelet disorders?

TTP	Plasmapharesis or intravenous immunoglobulin (IVIG) are first-line treat-ments. Splenectomy in refractory cases. Platelet transfusion is contraindicated because it just causes more consump-tion of platelets and more symptoms.
ITP	Corticosteroids are first-line treatment; second line is IVIG, splenectomy, or cyclophosphamide; platelet transfusion to stop bleeding.
DIC	Treat underlying cause. Platelet trans-fusion and fresh frozen plasma (FFP) can be given to stop bleeding as first line and aminocaproic acid as second line.

What is the most common genetic coagulopathy?

von Willebrand factor deficiency

How is vWF deficiency inherited?

Autosomal dominant pattern

What are the signs and symptoms of vWF deficiency?

Easy bruisability as well as mucosal and gastrointestinal (GI) bleeding

How is vWF deficiency diagnosed?

Normal PT/PTT,? bleeding time,?**factor VIII antigen,** normal platelet count, ? ristocetin platelet study

What is the treatment for vWF deficiency

Desmopressin (DDAVP) in mild cases; severe cases need factor VIII concentrate, cryoprecipitate for bleeding

Name the hemophilia described below:

X-linked recessive	Hemophilia A
Autosomal recessive	Hemophilia B
Factor IX deficiency	Hemophilia B
Factor VIII deficiency	Hemophilia A
Christmas disease	Hemophilia B

What are the clinical signs and symptoms of the hemophilias?

Hemarthroses;bleeding with minimal trauma, multiple ecchymoses

How are the hemophilias diagnosed?

? PTT, normal PT and normal bleeding time,normal vWF; factor VIII deficiency in hemophilia A and factor IX deficiency in hemophilia B

What is the treatment for each of the hemophilias?

Hemophilia A: factor VIII concentrate Hemophilia B: factor IX concentrate

What treatment can be given to a patient with hemophilia A prior to a surgical procedure?

Desmopressin—It increases the production of endogenous factor VIII

LEUKEMIAS

What are the signs and symptoms of leukemia?

Pallor, fatigue, anemia, infection, petechiae

Name the type of leukemia described below:

Proliferation of immature blast cells	Acute leukemias
Proliferation of mature, differentiated cells	Chronic leukemias
Associated with benzene	Acute myelogenous leukemia (AML)
Most common leukemia in *children*	Acute lymphoblastic leukemia (ALL)
Most common leukemia in *adulthood*	AML
Bimodal distribution	Acute leukemias
90% have the Philadelphia chromosome t(9; 22)	Chronic myelogenous leukemia (CML)
30% have the Philadelphia chromosome t(9; 22)	ALL
Auer rods, Sudan black positive, myeloperoxidase positive	AML
Terminal deoxynucleotidyl transferase (TdT) positive	ALL
Blast crisis	CML

What is the peak age of ALL?

Age 3–4 (most common cancer in children)

What are the subtypes of ALL?

L1, L2, L3

What subtype is most common in children?

80% are L1

Of the adult cases of ALL, what subtype is most common?

L2

What is the L3 subtype morphologically identical?

Burkitt lymphoma

How is ALL diagnosed?

Peripheral blood smear with increased blast cells and**TdT+, periodic acid-Schiff positive (PAS+), CALLA+**

What is the treatment plan for ALL?

Induction with chemotherapy (4–5 drugs)

Consolidation

Maintenance—radiation or low-dose chemotherapy

What is a poor prognostic factor in ALL?

Presence of Philadelphia chromosome

What is the treatment in patients that have the presence of the Philadelphia chromosome?

Bone marrow transplant

What is the prognosis of ALL in children?

80% remission

What is the prognosis in adults?

30% remission

What is the most common leukemia in adults?

AML

At what ages does AML peak?

15–39

What are the subtypes of AML?

M1–M7

M1–M3 granulocyte differentiation

M4–M5 monocytic precursors

M6 erythroblasts

M7 megakaryocytes

What hematologic disorder is the M3 subtype associated with?

DIC

How is AML diagnosed?

Peripheral blood smear with increased blast cells. Myeloblasts are**myeloperoxidase+, Auer rod+, Sudan black.**

What is the treatment for AML?

Induction with daunorubicin and cytarabine; add all-trans retinoic acid for M3 subtype

Consolidation—continue chemotherapy

Maintenance

What is the prognosis in adults with AML?

Those under age 60 have about a 70% – 80% remission rate.

What age group does chronic lymphocytic leukemia (CLL) affect?

65 and older

Which blood cell type does CLL mainly affect?

B cells

How is CLL usually diagnosed?

Bone marrow infiltrated with lymphocytes, lymphocytes express**CD5 protein,** lymphocytosis on complete blood count (CBC)

What is the progression of the disease?

Very slow progression

What is the treatment for CLL?

Supportive therapy because early therapy does not prolong life. Later there are the COP and CHOP regimens. COP: cyclophosphamide, vincristine, prednisone/prednisolone; CHOP: same as COP plus doxorubicin.

What age group does CML most commonly affect?

40–60 years of age

What carcinogenic agent might CML be associated with?

Prior exposure to radiation

What are the unique signs and symptoms of CML?

Abdominal pain/fullness, anorexia, diaphoresis, bone pain

What chromosomal abnormality is CML associated with?

90% have the Philadelphia chromosome.

What is the Philadelphia chromosome?

Translocation of the*ABL* gene from chromosome 9 to*BCR* gene on chromosome 22

How is CML diagnosed?

90% have the Philadelphia chromosome; peripheral blood smear shows increased myeloblasts, basophils, and white blood cells. Low leukocyte alkaline phosphatase.

What are the different phases of CML?

1. Chronic phase: hepatosplenomegaly and increase in WBCs.

2. Accelerated phase: platelet and RBC decrease while patient develops symptoms of night sweats, fever, bone pain, and weight loss.

3. Blastic phase: acute phase of the disease; blood and marrow are rapidly filled with proliferating blast cells.

What is a blast crisis?

Acute phase of the disease in which the blood and marrow are rapidly filled with proliferating blast cells; this takes about 3–4 years to develop and death is usually within 3–6 months

What is the treatment for CML?

Bone marrow transplant is main treatment. Hydroxyurea and interferon alfa can reduce WBC count. Chemotherapy is for patient who cannot have bone marrow transplant.

What is the prognosis after a bone marrow transplant in CML?

About 60% of patients go into remission.

What can CML progress to?

AML

Which type of leukemia has peripheral leukocytes with tartrate-resistant acid phosphatase and cytoplasmic projections?

Hairy cell leukemia

What is the treatment for hairy cell leukemia?

Interferon alfa, splenectomy

LYMPHOMA

Name the type of lymphoma (Hodgkin lymphoma vs. non-Hodgkin lymphoma [NHL]) described below:

Bimodal distribution—peaks in the thirties and seventies, more common in women	Hodgkin lymphoma
Bimodal distribution, more common in men	NHL
Mediastinal lymphadenopathy, contiguous spread	Hodgkin lymphoma
B cells transform to become malignant	Hodgkin lymphoma
Mostly originate from B cells but could also involve T cells	NHL
Peripheral lymphadenopathy, noncontiguous spread	NHL
Associated with Epstein-Barr virus (EBV) infection	NHL—Burkitt lymphoma
Associated with HIV	NHL
Symptoms worse with alcohol consumption	Hodgkin lymphoma
Reed-Sternberg cells	Hodgkin lymphoma
Bone marrow with "starry sky" appearance	NHL—Burkitt lymphoma

What are the four subtypes of Hodgkin lymphoma?

1. Nodular sclerosing

2. Lymphocyte predominating

3. Mixed cellularity

4. Lymphocyte dependent

What is the most common type of Hodgkin lymphoma?

Nodular sclerosing

Which of the four subtypes of Hodgkin lymphoma has the worst prognosis?

Lymphocyte dependent

What clinical feature distinguishes Hodgkin lymphoma from NHL

Adenopathy is regional rather than systemic.

What are the symptoms of Hodgkin lymphoma and what are they called?

“B” symptoms—fever, night sweats, malaise, weight loss

How is Hodgkin lymphoma diagnosed?

Lymph node biopsy will show**Reed-Sternberg cells.**

What are the next steps to be taken after a biopsy determines a lymphoma is present?

Chest x-ray (CXR) to see extent of involvement as well as possible bone marrow biopsy and computed tomographic (CT) scan.

What is the staging of Hodgkin’s lymphoma?

Stage 1: one lymph node

Stage 2: two or more lymph nodes on the same side of the diaphragm

Stage 3: involvement on both sides of the diaphragm

Stage 4: dissemination to organs and tissues

What is the treatment for Hodgkin lymphoma?

Radiation therapy for localized disease (stages 1 and 2) and chemotherapy for more extensive disease (stages 3 and 4)

What chemotherapy regimens are most commonly used?

ABVD: adriamycin, bleomycin, vincristine, dacarbazine

MOPP: meclorethamine, oncovin, procarbazine, prednisone

How are the different types of NHL characterized?

Low-, intermediate-, and high-grade

Name the most common subtypes of NHL?

Low-grade: follicular small cleaved cell

Intermediate grade: diffuse large cell lymphoma

High-grade: lymphoblastic lymphoma

Burkitt lymphoma: American type and African type

Name the subtype of NHL described below.

High-grade lymphoma more common in children	Burkitt's lymphoma
Burkitt lymphoma with jaw involvement	African Burkitt lymphoma
Burkitt lymphoma with abdominal involvement	American Burkitt
Translocation involving *BCL2* gene	Follicular small cleaved cell
Can involve the GI tract as well as the head and neck	Diffuse large cell lymphoma
Can involve the central nervous system (CNS) and bone marrow	Lymphoblastic lymphoma
Derived from thymic T cells	Lymphoblastic lymphoma

How is NHL diagnosed?

Biopsy of lymph node

What are the next diagnostic studies to consider after the biopsy?

A CXR, CT scan, bone marrow biopsy to determine the extent of the disease

What is the prognostic factor in NHL?

Histologic subtype is a more prognostic factor than the extent of spread of disease.

How is the adenopathy in NHL described?

Painless adenopathy

What is the treatment for NHL?

Radiation and chemotherapy depending on subtype

MYELOPROLIFERATIVE DISEASES

What are myeloproliferative diseases?

A number of diseases in which there is excessive production of differentiated myeloid cell lines

What can the myeloproliferative diseases transform into?

Acute leukemias

What is polycythemia vera?

A myeloproliferative disorder in which there is excess production of**ALL** blood cell lines

What are the different etiologies of polycythemia vera?

It can be a primary disorder which is idiopathic in nature or it can be secondary to hypoxia, dehydration, low erythropoietin production, and smoking.

What is the peak of onset of polycythemia vera?

Age > 60

In what sex is polycythemia vera most commonly seen?

Males

What are the signs and symptoms of polycythemia vera?

Pruritis after showering, epistaxis, plethora, blurred vision, splenomegaly, gout, basophilia, headache, retinal hemorrhages, cerebrovascular accidents (CVA), gastric ulcers

How is polycythemia vera diagnosed?

On CBC there is pancytopenia. Patient may have low erythropoietin and low erythrocyte sedimentation rate (ESR).

What is the treatment for polycythemia vera?

Serial phlebotomy to decrease the volume of blood; hydroxyurea to suppress excess blood cell production; aspirin to thin the blood

What is a possible long-term complication that occurs in about 20% of patients with polycythemia vera?

Fibrosis of the bone marrow

What is essential thrombocytosis?

Disease in which there is an idiopathic increase of platelets to > 5 × 105cells/μL

What are the clinical signs and symptoms of essential thrombocytosis?

Burning and throbbing hands and feet as well as splenomegaly

What are the main treatments for essential thrombocytosis?

Platelet exchange, hydroxyurea, anagrelide

What is idiopathic myelofibrosis?

Disorder in which there is extensive extramedullary hematopoiesis with proliferation of the

megakaryocytes in the bone marrow

What is the pathopneumonic sign of myelofibrosis?

Peripheral smear shows**tear drop cells.**

What is the treatment for myelofibrosis?

The prognosis is poor and the treatment is mainly supportive.

What is multiple myeloma?

Malignant disease of plasma cells which produce**monoclonal immunoglobulins or light chains**

What is the ratio of white to African American who have multiple myeloma?

1:2

What can be seen on an x-ray of a patient with multiple myeloma?

Lytic lesions(“punched out” areas of bone)

What are the signs and symptoms of multiple myeloma?

Bone pain, pathological fractures due to**lytic lesions;** anemia, hypercalcemia, renal failure

What is the triad that is often seen in multiple myeloma?

1. Anemia

2. Back pain

3. Renal failure

How can multiple myeloma be diagnosed?

24-hours urine collection followed by urine protein electrophoresis (UPEP) and serum protein electrophoresis (SPEP). These studies will demonstrate free kappa and lambda light chains known as**Bence Jones proteins,** and monoclonal elevation of one cell line. There will be an “M-spike” (or a peak) in the SPEP if there is whole antibody made. There will be an “M-spike” in the UPEP if light chains only are made. To make the diagnosis there should be a spike in the SPEP or UPEP as well as one of the following: lytic lesions, Bence Jones proteinuria, or increased plasma cells in the bone marrow.

What is the treatment for multiple

Chemotherapy in addition to calcitonin and allopurinol as needed for hypercalcemia and elevated uric acid respectively.

CHAPTER 7

RHEUMATOLOGY

ARTHROPATHIES

What is rheumatoid arthritis (RA)?

An autoimmune symmetric inflammatory arthritis

What HLA type is RA associated with?

HLA-DR4

In what sex is RA more common?

Females

What classical physical examination findings can be found in RA?

Boutonniere deformity; swan neck deformity; ulnar deviation; pain in the**proximal interphalangeal (PIP) and metacarpophalangeal (MCP) joints,** rheumatoid nodules

What are the seven diagnostic criteria for RA?

1. Morning stiffness > 1 hour

2. Three or more joints with arthritis

3. One hand joint with arthritis

4. Symmetric arthritis

5. Rheumatoid nodules

6. Elevated serum rheumatoid factor (RF)

7. Radiographic changes

How many of the criteria must be positive for a diagnosis of RA?

Four

What is a boutonniere deformity?

Hyperextension of distal interphalangeal (DIP) and flexion of PIP joints (Fig. 7-1)

What is a swan neck deformity?

Flexion of DIP and extension of PIP joints (Fig. 7-2)

What laboratory findings could you expect in a patient with RA?

? RF, ?erythrocyte sedimentation rate (ESR)

What is the treatment for pain associated with RA?

First-line: nonsteroidal anti-inflammatory drugs (NSAIDs) to decrease inflammation

Second-line: corticosteroids

Figure 7-1 Boutonniere deformity. (Reproduced, with permission, from Wilson FC, Lin PP.*General Orthopedics* . New York: McGraw-Hill, 1997:413.)

What disease modifying agents are available for patients with RA?

Methotrexate, hydroxychloroquine, gold compounds

What are some newer biologic agents used to treat RA?

Infliximab, etanercept, abetacept, rituxan

What is the most common type of arthritis?

Osteoarthritis (OA)

What is the underlying cause of OA?

Mainly wear and tear of the joints

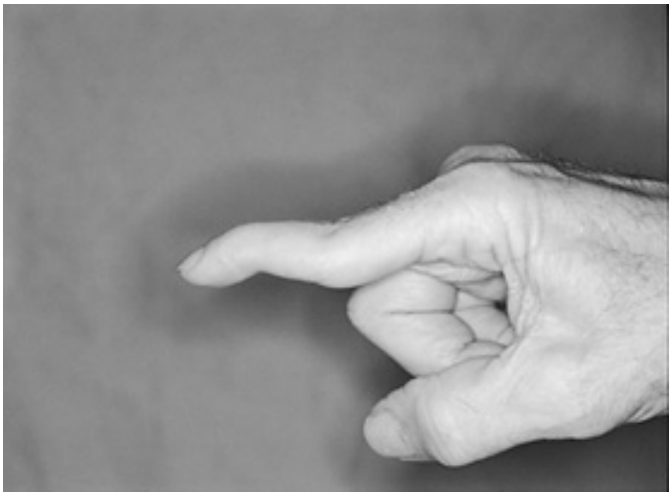


Figure 7-2 Swan neck deformity. (Reproduced, with permission, from Knoop KJ, Stack LB, Storrow AB. Atlas of Emergency Medicine. New York: McGraw-Hill, 1997:291.)

What are the two classic physical examination findings in OA?

Heberden noduleswhich affect the DIP joints

Bouchard nodeswhich affect the PIP joints (Fig 7-3)

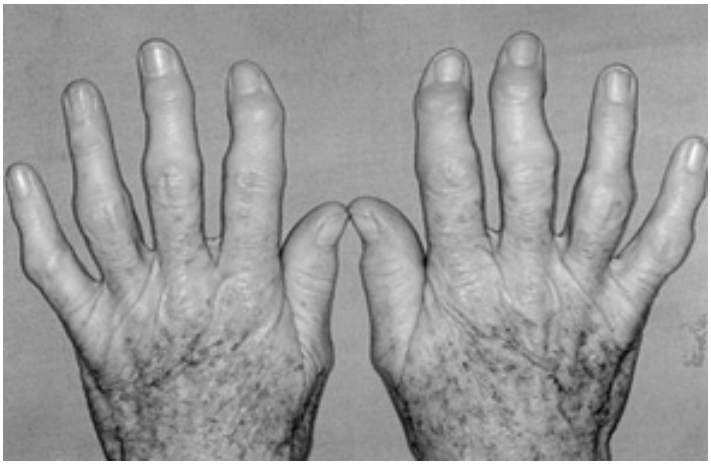


Figure 7-3 Bouchard nodes. (Reproduced, with permission, from Knoop KJ, Stack LB, Storrow AB. Atlas of Emergency Medicine. New York: McGraw-Hill, 1997:291.)

How do the symptoms of OA differ from RA?

Morning stiffness resolves within 30 minutes; outer joints of the hand are mainly affected (DIP joints in addition to MCP and PIP joints).

What are the x-ray findings seen in OA?

Narrowed joint spaces,**osteophyte formation**

What is the treatment for OA?

NSAIDs to relieve pain; muscle strengthening exercises; steroid joint injection; last resort is joint replacement

What is gout?

Arthropathy caused by urate crystal deposit in a single joint

What are the etiologies of gout?

Decreased uric acid excretion (high-protein diet, alcohol use, diuretic use) or increased uric acid production (genetic diseases, hemolysis, cancer)

What are the signs and symptoms of

Acute pain accompanied by redness**gout?** and swelling of a joint

What is the most common joint to be affected?

First metatarsophalangeal joint

What is podagra?

Inflammation of the first metatarsophalangeal joint of the foot which is of sudden onset

What are tophi?

Aggregates of gouty crystal and giant cells secondary to chronic gout

What is the classic radiographic finding in advanced gout?

“Rat-bite” appearance

How is gout diagnosed?

Fluid aspirated from the joint would reveal**needle** -shaped monosodium urate crystals with**negative birefringence**

How is acute gout treated?

Colchicine and NSAIDs for pain

What is used for maintenance therapy of gout?

Allopurinol to prevent production; probenecid to increase excretion; low-protein diet; refrain from alcohol

What is pseudogout?

Deposition of calcium pyrophosphate crystal deposits in joints

What does joint fluid aspiration in pseudogout demonstrate?

Positively birefringent rhomboid crystals

What is the treatment for pseudogout?

NSAIDs

Name the autoimmune disorder which is characterized by sacroiliitis, with fusion of adjacent vertebral bodies.

Ankylosing spondylitis

What HLA type is ankylosing spondylitis associated with?

HLA-B27

What joint is always affected in ankylosing spondylitis?

Sacroiliac joint

What is the classic x-ray finding seen with ankylosing spondylitis?

Bamboo spine(Fig. 7-4)

What other disorder is ankylosing spondylitis associated with?

Ulcerative colitis

SYSTEMIC DISORDERS

What are the signs and symptoms of systemic lupus erythematosus (SLE)?

Fatigue, malaise, malar rash, arthralgias, pericarditis, endocarditis, neurologic symptoms, polyarthritis

What is the sex distribution of SLE?

90% female predominance

How is SLE distributed based on race?

Black > white

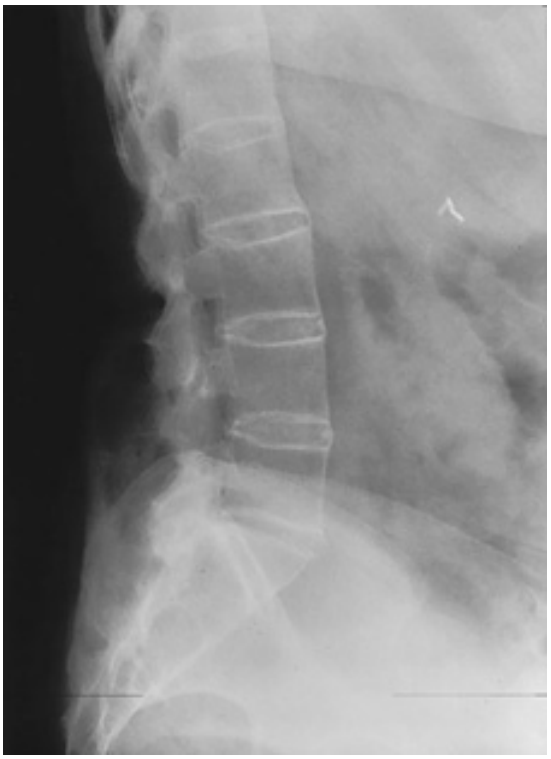


Figure 7-4 Bamboo spine. (Reproduced, with permission, from Wilson FC, Lin PP.*General Orthopedics* . New York: McGraw-Hill, 1997:454.)

What is the mnemonic for diagnosing SLE?

DOPAMINE RASH:

Discoid rash: raised, erythematous circular rash with scale

Oral ulcers

Photosensitivity

Arthritis > 2 joints

Malar rash: butterfly rash on cheeks

Immunologic criteria: anti-Sm Ab, anti-double stranded DNA, false-positive venereal disease research laboratory (VDRL) test

Neurologic symptoms: seizures, psychosis

ESR elevated (not part of the 11 criteria)

Renal disease

Antinuclear antibody (ANA) positive

Serositis: pericarditis, pleurisy

Hematologic disorder: hemolytic anemia, leukopenia, thrombocytopenia, lymphopenia

How many of the criteria must be present to make the diagnosis of SLE?

Four or more

What is the pathomnemonic heart disorder seen in SLE patients?

Libman-Sacks endocarditis (LSE)

What autoantibodies is most sensitive for SLE?

ANA (it is not specific)

Which autoantibody is most specific for SLE?

Anti-double stranded-DNA (very high titers are associated with renal involvement), anti-

SM antibody

What other autoantibodies are associated with SLE?

Anti-La antibody

Anti-Ro antibody

What are lupus anticoagulant and anticardiolipin associated with?

Thrombosis, central nervous system (CNS) lupus, thrombocytopenia, valvular heart disease, fetal loss

What serologies can be falsely positive in patients with SLE?

Rapid plasma reagin (RPR)/VDRL

Anticardiolipin can cause a falsely elevated result with which lab test?

Elevated partial thromboplastin time (PTT), but in reality SLE patients are more likely to develop blood clots

What are the treatments for SLE?

Avoid sun exposure, NSAIDs for joint pain, systemic steroids, immunosuppressives such as cyclophosphamide in refractory cases with more advanced development of disease

How is drug-induced lupus different from SLE?

Symptoms resolve with discontinuation of the drug*and***anti-histone antibody positive**

What drugs are known to cause drug-induced SLE?

SIQ CHaMP:

Sulfasalazine

Isoniazid (INH)

Quinidine

Chlorpromazine

Hydralazine

a

Methyldopa, minocycline

Procainamide, penicillamine

What is the most common drug to cause lupus-like symptoms?

Procainamide

What autoimmune disorder is characterized by systemic fibrosis secondary to excess collagen and extracellular matrix production?

Scleroderma

What are the signs and symptoms of scleroderma?

Tight, thick skin; Raynaud phenomenon; dysphagia; renal artery fibrosis; pulmonary hypertension secondary to fibrosis; telangiectasias

What is a milder form of scleroderma called?

CREST syndrome

What does CREST stand for?

Calcinosis

Raynaud phenomenon

Esophageal dysmotility

Sclerodactyly

Telangiectasias

What laboratory test is 80% sensitive for CREST syndrome?

Anticentromere antibody

What laboratory test is highly specific to scleroderma?

Anti-Scl-70 antibody

What is the treatment for scleroderma?

CAPS:

Calcium channel blocker

Ace inhibitor (captopril)

Penincillamine

Steroids

What systemic disease is characterized by noncaseating granulomas in the lung?

Sarcoidosis

What race is more predisposed to sarcoidosis?

African Americans

What are some findings associated with sarcoidosis?

GRUELING

Granulomas

RA

Uveitis

Erythema nodosum

Lymphadenopathy

Interstitial fibrosis

Negative TB test

Gamma-globulinemia

What renal problem is associated with sarcoidosis?

Nephrolithiasis because of hypercalciuria

What is the most important component of diagnosing sarcoidosis?

Transbronchial biopsy showing noncaseating granuloma

What is seen on a chest x-ray (CXR) of a patient with sarcoidosis?

Bilateral hilar adenopathy with perihilar calcifications

What classic laboratory findings are seen in sarcoidosis?

Hypercalcemiaand ?**angiotensin-converting enzyme (ACE)**

What is the treatment for sarcoidosis?

Symptomatic treatment and cholinergic drugs

What autoimmune disorder is associated with the following triad: keratoconjunctivitis sicca, xerostomia, and arthritis?

Sjögren syndrome

What HLA type is Sjögren syndrome associated with?

HLA-DR3

What type of cancer are patients with Sjögren syndrome at high risk for?

Lymphoma

What autoantibodies is Sjögren syndrome associated with?

Anti-single stranded (SS)-A (Ro) and anti-SS-B (La)

What is the treatment for Sjögren syndrome?

Corticosteroids

Name the syndrome associated with the following: conjunctivitis, uveitis, urethritis, and asymmetric arthritis.

Reiter syndrome

What is the mnemonic used to remember the associated findings of Reiter syndrome?

“Can’t see. Can’t Pee. Can’t climb a tree.”

Can’t see: conjunctivitis, uveitis

Can’t pee: urethritis

Can’t climb a tree: arthritis

What HLA type is Reiter syndrome associated with?

HLA-B27

What are the two forms of Reiter syndrome?

1. Sexually transmitted

2. Postinfectious:*Campylobacter* ,*Yersinia, Salmonella, Shigella*

What will a urethral culture often grow out in a patient with Reiter syndrome?

Chlamydia trachomatis

What is the treatment for Reiter syndrome?

Doxycycline to cover for*Chlamydia* and NSAIDs for pain

What is the autoimmune syndrome associated with the following: aphthous ulcers, genital ulcers, arthritis, uveitis, psychiatric symptoms

Behçet syndrome

MUSCLE DISORDERS

What is polymyositis?

Autoimmune disease which causes proximal muscle weakness

How is polymyositis different from dermatomyositis?

Dermatomyositis includes rash as a symptom, whereas with polymyositis there is no rash.

What sex is more likely to have polymyositis?

Females are twice as likely.

What are the signs and symptoms of polymyositis?

Symmetric proximal muscle weakness,dysphonia, and dysphagia; patients have difficulty standing up from a chair or brushing their hair

What are the classic signs of dermatomyositis?

Symmetric proximal muscle weakness,**heliotropic periorbital rash, shawl sign** (erythematous macules on shoulders and upper back),**Gottron papules** (violacious papules on DIP joints)

What autoantibody is associated with polymyositis and dermatomyositis?

Anti-Jo-1

What are the four criteria for polymyositis?

1. ? Creatine phosphokinase (CPK)

2. Proximal muscle weakness

3. Low-amplitude potentials and fibrillations on electromyogram (EMG)

4. ? Muscle fiber size on muscle biopsy

What is the treatment for polymyositis and dermatomyositis?

Corticosteroids and methotrexate or cyclophosphamide in refractory cases

What is myasthenia gravis?

Autoimmune disease in which autoantibodies block the postsynaptic acetylcholine receptors preventing acetylcholine from binding leading to muscle weakness

What are the two peak incidences of myasthenia gravis?

Women: second to third decades of life

Men: fifth to sixth decades of life

What can myasthenia gravis be associated with?

Thymomas or other autoimmune diseases

What are the signs and symptoms of myasthenia gravis?

Muscle weakness and increasing fatigue with use, proximal muscle weakness, ptosis, diplopia, dysphagia

What is the test used to diagnose myasthenia gravis?

Edrophonium test (Tensilon test)

How does the test work?

Edrophonium inhibits acetylcholinesterase allowing for higher levels of acetylcholine to be available to stimulate receptors and, therefore, if the patient has myasthenia gravis,

edrophonium administration will lead to improved muscle strength.

What is the treatment for myasthenia gravis?

Pyridostigmine and acetylcholinesterase inhibitor as well as steroids

What is the pathology in Lambert-Eaton syndrome?

There are autoantibodies to presynaptic calcium channels

How does Lambert-Eaton syndrome differ from myasthenia gravis?

Increased muscle use improves symptoms making muscles stronger

VASCULITIS

Small and medium vessel vasculitis with no pulmonary involvement that often presents as abdominal pain and is associated with hepatitis B antigenemia and perinuclear antineutrophil cytoplasmic antibodies positive (p-ANCA +)	Polyarteritis nodosa (PAN)
Medium vessel arteritis with prominent pulmonary findings and associated with eosinophilia and asthma	Churg-Strauss disease
Granulomatous vasculitis mainly of the upper and lower respiratory tract that often presents with hemoptysis and can lead to glomerulonephritis	Wegener granulomatosis
Cytoplasmic antineutrophil cytoplasmic antibody (c-ANCA) positive	Wegener granulomatosis

Medium and large vessel arteritis that is most commonly seen in young Asian individuals	Takayasu arteritis
Arteritis that is characterized by loss of pulses in arms and carotids, Raynaud's phenomenon, and signs of ischemia such as blindness	Takayasu arteritis
Also known as giant cell arteritis and affects the temporal artery	Temporal arteritis

CHAPTER 8

Nephrology

ACUTE RENAL FAILURE

What is azotemia?

A high level of urea or other nitrogen-containing compounds in the blood usually secondary to renal failure

What is uremia?

Elevated levels of urea in the blood usually secondary to renal failure

What characterizes uremic syndrome?

Uremic syndrome is CRF that has effects on multiple organs and systems.

Cardiovascular: hypertension

Pulmonary: pleural effusions, pulmonary edema

Central nervous system (CNS): asterixis, clonus

Hematology: anemia because of low erythropoietin; increased bleeding time

Gastrointestinal (GI): nausea; vomiting

Metabolic: acidosis, electrolyte imbalances (especially hyperkalemia), hypocalcemia (lack of vitamin D), azotemia

What is acute renal failure (ARF)?

Newly increased azotemia with an increase in blood urea nitrogen (BUN) and creatinine

What are the three categories of acute renal failure?

1. Prerenal

2. Renal

3. Postrenal

What causes prerenal ARF?

Low perfusion

What are examples of prerenal causes of ARF?

1. Congestive heart failure (CHF)

2. Volume loss

3. Hypotension

4. Sepsis

5. Burns

6. Low blood flow to the kidneys (renal artery stenosis [RAS])

What is the underlying cause of intrinsic ARF?

Injury to the nephron due to ischemia or toxins

What is the most common cause of intrinsic renal failure?

Acute tubular necrosis (ATN)

What are some other causes of intrinsic ARF?

1. Acute interstitial nephritis (AIN)

2. Glomerulonephritis (GN)

3. Ischemia

4. Vasculitis

What are some causes of postrenal acute renal failure?

Obstruction caused by:

1. Kidney stones

2. Enlarged prostate (BPH)

3. Tumors such as bladder cancer (CA), cervical CA, prostate CA

What are some signs and symptoms of ARF caused by uremia?

Asterixis, nausea, vomiting, anemia, pericarditis, pruritis, urea crystals on the skin ("uremic frost"), fatigue, oliguria

What are some signs and symptoms of ARF not caused by uremia?

Metabolic acidosis

Hyperkalemia ? arrhythmias

Fluid overload ? pulmonary edema, CHF, hypertension

Anemia 2° to low-erythropoietin secretion

Hypertension 2° excess renin secretion

What defines oliguria?

Urine output of < 400 cc/24 hours

What tests would you initially order to evaluate for ARF?

Urine/serum electrolytes; urine/serum BUN/CR; urinalysis including urine osmolality

What is FENa?

FENastands for fractional sodium excretion and is the best diagnostic test to help discriminate between the different types of ARF.

How is FENacalculated?

$$FE_{Na} = \frac{(\text{urine Na}/\text{plasma Na})}{(\text{urine creatinine}/\text{plasma creatinine})} \times 100\%$$

How do you distinguish between prerenal, renal, and postrenal ARF?

SeeTable 8-1 .

Table 8-1ARF: Laboratory Differences Between Prerenal, Renal, and Postrenal Etiologies

Study	Prerenal	Renal	Postrenal
FE_{Na}	< 1%	> 2%	> 4%
Urine Na	< 20	> 20	> 40
BUN/CR	> 20	< 15	> 15
Urine osmolality	> 500	< 350	< 350

Name the type of ARF associated with the following urinary sediment findings:

Red cell casts	GN
Urine eosinophils	AIN
White blood cell (WBC) casts	AIN
Granular casts	ATN

What are the causes of ATN?

There are two categories:

1. Ischemic: shock, trauma, hypoxia, hemorrhage, sepsis

2. Toxic: medications, rhabdomyolysis (which causes myoglobinuria), IV contrast

What medications classically cause ATN?

Contrast

Lithium

Aminoglycosides

Amphotericin

Pentamine

How is ATN treated?

Remove insulting agent, IV fluids to maintain urine output, IV diuretic therapy to increase urinary output and prevent overload, protein-restricted diet, close monitoring of electrolytes; dialysis if needed

What are the causes of AIN?

Inflammation of the renal parenchyma caused by:

1. Medications: diuretics, nonsteroidal anti-inflammatory drugs (NSAIDs), penicillin

2. Infection: cytomegalovirus (CMV), Epstein-Barr virus (EBV), toxoplasmosis, syphilis

3. Systemic diseases: Sjögren syndrome, sarcoidosis

Name the cause of ARF classically indicated by the following serologic tests:

Anti-neutrophil cytoplasmic antibodies + (ANCA +)	Wegener, polyarteritis nodosa, other vasculitis
Antiglomerular basement membrane antibody (anti-GBM)	Goodpasture syndrome
How is AIN treated?	Treatment is the same as ATN

CHRONIC RENAL FAILURE

What is chronic renal failure (CRF)?

Progressive loss of nephrons

What is the most common cause of CRF?

Diabetes

What can be used to measure the severity of CRF?

Glomerular filtration rate (GFR); the lower the GFR the worse the renal function

How is GFR estimated?

Creatinine clearance is approximately equal to GFR

How is creatinine clearance calculated?

Urine creatinine × urine volume in mL/serum creatinine × time in minutes

$$\text{Estimated creatinine clearance} = \frac{(140 - \text{age}) \times (\text{weight in kg}) \text{ (for females} \times 0.85)}{\text{serum creatinine} \times 72}$$

In CRF, there is decreased synthesis of what two entities?

Vitamin D and erythropoietin

What electrolyte abnormalities are seen in CRF?

Hyperkalemia

Hypocalcemia

Hyperphosphatemia

What is the treatment for chronic renal failure?

Dialysis: either hemodialysis or peritoneal dialysis

How should medications prescribed to patients in CRF be adjusted?

They should be renally dosed.

What are the indications for dialysis?

Remember the mnemonic:

Acidosis

Electrolyte abnormalities

Ingestion of toxins

Overload of fluid

Uremic symptoms

How is vascular access achieved in a hemodialysis patient?

Either an arteriovenous fistula that is usually placed in the forearm or a shunt between a vein and artery

How is peritoneal dialysis achieved?

Patient gets a permanent catheter in the peritoneum and the peritoneum is used as a membrane through which dialysis is achieved. Dialysis fluid is infused rapidly, then allowed to stay in the peritoneal cavity for several hours, then drained and new fluid infused.

What kind of infection are patients on peritoneal dialysis classically at risk for?

Bacterial peritonitis

How is bacterial peritonitis treated?

Intraperitoneal vancomycin or antibiotics based on culture sensitivity

GLOMERULONEPHROPATHIES

What is nephrotic syndrome?

Nephroticsyndrome is glomerular damage leading to**proteinuria (>3.5 gram/d)**

What are other defining features of nephrotic syndrome?

Hypoalbuminemia, generalized edema, hyperlipidemia, hypercoagulable state (because of loss of protein C and S), immunocompromised state

What is nephritic syndrome?

Glomerular disease leading to syndrome of hematuria, edema, and often hypertension (HTN)

How can urinary cholesterol be identified?

If urine is seen under polarized light, there will be “maltese crosses.”

What are some causes of nephrotic syndrome?

1. Minimal change disease (MCD)

2. Focal segmental glomerulosclerosis

3. Membranous glomerulonephritis

4. Membranoproliferative glomerulonephritis

What are the other names for minimal change disease?

Nil disease, lipoid nephrosis

Name the nephritic syndrome associated with each of the following:

Loss of epithelial foot processes seen under electron microscopy	Minimal change disease
Idiopathic etiology	Minimal change disease
Most common primary cause of nephritic syndrome	Membranous glomerulonephritis
Two forms, Type I is slowly progressive and Type II has autoantibodies against C3 and is more rapidly progressive	Membranoproliferative glomerulonephritis
Associated with refractory HTN	Focal segmental glomerulosclerosis
Frequently recurs	Minimal change disease
Granular deposits of IgG and C3	Membranous glomerulonephritis
Often seen in children	Minimal change disease
Presents in young, black men with refractory hypertension	Focal segmental glomerulosclerosis
Associated with HIV, IV drug abuse, sickle cell anemia	Focal segmental glomerulosclerosis
"Spike and dome" on histology due to excess basement membrane	Membranous glomerulonephritis
Slowly progressive disease with minimal response to corticosteroid therapy	Membranous glomerulonephritis
Does not progress to chronic renal failure	Minimal change disease
Associated with hepatitis, systemic lupus erythematosus (SLE), syphilis, malaria, penicillamine, gold salts, CA	Membranous glomerulonephritis

What is the main treatment for each of the following:

Minimal change disease	Corticosteroids
Focal segmental glomerulosclerosis	Corticosteroid with cyclophosphamide (prognosis is poor)
Membranous glomerulonephritis	Corticosteroids, can add cyclophosphamide in refractory cases
Membranoproliferative glomerulonephritis	Corticosteroids. Plasmapharesis can be added.

Name the systemic diseases that can lead to nephritic syndrome.

SLE, sickle cell anemia, HIV, diabetes, multiple myeloma

What is nephritic syndrome?

Glomerulonephropathy also known as glomerulonephritis in which there is acute-onset hematuria, azotemia, hypertension, edema, and mild proteinuria

What is classically seen on microscopy in nephritic syndrome?

Red blood cell (RBC) casts

Name the five types of glomenrulonephritis.

1. Poststreptococcal glomerulonephritis (PSGN)

2. Rapidly progressive glomerulonephritis

3. Mesangial proliferative glomerulonephritis

4. Membranoproliferative glomerulonephritis

5. IgA nephropathy

Name the nephritic syndrome associated with the following:

Follows group A beta-hemolytic *Streptococcus* or another infectious agent	PSGN
Henoch-Schönlein purpura	IgA nephropathy
Self-limiting disease	PSGN, Henoch-Schönlein purpura
Also known as crescentic glomerulonephritis	Rapidly progressive glomerulonephritis
Goodpasture disease	Rapidly progressive glomerulonephritis
Often diagnosed with elevated ASO titer	PSGN
Buerger disease	IgA nephropathy
Coarse, granular IgG or C3 deposits	PSGN
Smooth, linear IgG deposits	Rapidly progressive glomerulonephritis
Anti-GBM antibody disease	Rapidly progressive glomerulonephritis

What is the most common glomerulonephropathy?

Buerger disease

What is Goodpasture disease?

Glomerulonephritis with pneumonitis

When is the peak incidence of Goodpasture disease?

Males in the second decade of life

What is the most common presenting symptom of Goodpasture disease?

Hemoptysis

URINARY TRACT

What is nephrolithiasis?

Kidney stones

What are the classic signs and symptoms of nephrolithiasis?

Back pain or flank pain that radiates to groin, nausea, vomiting, microscopic vs. gross hematuria

What is the most common type of kidney stone?

Calcium pyrophosphate

What is the underlying etiology?

Hypercalciuria

What is the treatment for calcium pyrophosphate stones?

Hydration and thiazide diuretics; lithotripsy if stone is too large to pass

What is the second most common type of kidney stone?

Ammonium magnesium phosphate

What is another name for ammonium magnesium phosphate stones?

Struvite stones

What are the underlying bacterial etiologies of ammonium magnesium phosphate stones?

Proteus, *Pseudomonas*, *Providencia*, or*Staphylococcus saprophyticus*

How are struvite stones treated?

Treat the underlying infection and lower the urinary pH

Which type of stone is radiolucent?

Uric acid stones

What disorders are often an underlying cause of uric acid stones?

Gout or myeloproliferative disease

How are uric acid stones treated?

Raise urinary pH

Which type of stone is radiopaque?

Calcium pyrophosphate and ammonium magnesium phosphate

How is nephrolithiasis diagnosed?

Plain films can identify radiopaque stones. Renal ultrasound (US) can visualize hydronephrosis; computed tomography (CT) scan can visualize small stones. IV pyelogram is the gold standard for diagnosis, however.

What is the most common pathogen in urinary tract infections (UTIs)?

Escherichia coli

What is the mnemonic for common pathogens causing UTIs?

KEEPS:

Klebsiella

E. coli

Enterobacter

Proteus

S. saprophyticus

What are the signs and symptoms of UTI?

Urinary urgency, frequency; burning with urination; hematuria; sense of incomplete bladder emptying

How is a UTI diagnosed?

Urinalysis can demonstrate a high number of WBCs, positive leukocyte esterase, positive nitrites, and moderate to large number of bacteria.

What is the indication of a contaminated urinalysis?

Many epithelial cells or many types of bacteria present

Other than urinalysis, what test should be ordered in a patient suspected to have a UTI?

Urine culture, Gram stain, and sensitivity

What is the first-line treatment for UTI?

Bactrim

What would you suspect in a patient with urinary frequency, burning on urination, costovertebral angle tenderness as well as fever and chills?

Pyelonephritis

What is the treatment for pyelonephritis?

po or IV antibiotics

ACID-BASE DISORDERS

What are the normal lab values for each of the following components of an arterial blood gas (ABG):

pH?	7.35–7.45
$PaCO_2$?	35–45
PaO_2?	80–100
HCO_3?	21–27
O_2 saturation?	95–100
Base excess?	−2 to +2

How is anion gap calculated and what is a normal range?

Na - (Cl + HCO3) Normal range is 9 to 14.

What is the definition of metabolic acidosis?

? pH with ? HCO3

What is Winter's formula?

It determines if there was appropriate compensation in the setting of metabolic acidosis.

$1.5 \times (HCO3-) + 8 +/- 2 = PCO2$

What are the causes of anion gap metabolic acidosis?

MUD PILES:

Methanol,**M** etformin

Uremia

DKA (diabetic ketoacidosis)

Paraldehyde

INH (isoniazid), iron tablets

Lactic acidosis

Ethanol

Salicylates

How is the etiology of the metabolic acidosis determined?

Check for ketonuria

Which of the etiologies are present with and without ketonuria?

SeeTable 8-2 .

Table 8-2 Anion Gap Metabolic Acidosis Etiologies

Ketonuria Present	Ketonuria Absent
DKA	Lactic acidosis
Paraldehyde ingestion	Methanol
Isopropyl alcohol ingestion	Ethylene glycol
Starvation	Salicylate poisoning

What are the causes of normal anion gap metabolic acidosis?

Renal tubular acidosis, diarrhea, exogenous acid ingestion

What is the treatment for metabolic acidosis?

Correct the underlying cause

What is the definition of respiratory acidosis?

Hypoventilation causing ? PaCO2and ? pH

What is the treatment for respiratory acidosis?

Treat the underlying cause and mechanical hyperventilation can help to release some CO2

What is the definition of metabolic alkalosis?

? pH, ? plasma bicarbonate, and compensatory $PaCO_2$

What are the underlying causes of metabolic acidosis?

Vomiting, diarrhea, nasogastric (NG) tube suction for prolonged period, diuretic use, hypomagnesemia, hypokalemia, licorice, tobacco use, Cushing syndrome, RAS

What is the treatment for metabolic acidosis?

Treat the underlying cause. These patients are usually volume depleted so rehydration is needed. Replete potassium and magnesium as needed.

What is the definition of respiratory alkalosis?

Hyperventilation causing ? arterial pH, ? PCO_2, ? serum bicarbonate

What is the treatment of respiratory alkalosis?

Decrease rate of breathing

RENAL ARTERY STENOSIS

What are the classic findings in renal artery stenosis (RAS)?

Hypertension with hypokalemia

What are the underlying causes of RAS?

Atherosclerosis or fibromuscular dysplasia

What is the more common cause of RAS in females?

Fibromuscular dysplasia

What is in the differential diagnosis when a patient has the classic finding of hypertension with hypokalemia?

Conn hyperaldosteronism vs. secondary hyperaldosteronism due to renal artery stenosis

How can a patient be screened for RAS?

Captopril stimulation test: if the patient has RAS, captopril will induce an increase in renin; however, in Conn syndrome renin will not increase

How is RAS diagnosed?

Renal angiography

How is RAS treated?

Angioplasty and, in some cases, surgery

CHAPTER 9

Endocrinology

DIABETES

What is the pathophysiology of type 1 diabetes?

Insulin deficiency due to autoinflammatory destruction of pancreatic B cells

What is the pathophysiology of type 2 diabetes?

Insulin resistance and relative insulin deficiency

What is the age of onset of type 1 and type 2 diabetes?

Type 1 usually begins in childhood/adolescence and type 2 usually begins in adulthood

Which of the two types of diabetes has a stronger genetic factor?

Type 2 diabetes(seems counterintuitive)

What are the early symptoms of diabetes?

“The three polys”: polyuria, polydipsia, and polyphagia;**and** weight loss

What are chronic complications of diabetes?

Retinopathy, nephropathy, neuropathy, cerebrovascular disease, coronary artery disease (CAD)

What type of fatal fungal infection can diabetics get?

Mucor, especially**sinusitis** (Note: They love to ask this on the boards!)

What is the histologic description of*Mucor?*

Nonseptate hyphae with branching at 90° (looks like the letter**M**)

What are the diagnostic criteria for diabetes?

Both types of diabetes are diagnosed based on the same criteria.

Fasting glucose over 125 two separate times

Random glucose over 200 with symptoms of diabetes

Or

Glucose tolerance test over 200

What is the treatment for type 1 diabetes?

Insulin replacement. Since these individuals do not have insulin, hypoglycemics will not work.

For each of the following types of insulin, describe the peak and duration of action:

Insulin lispro	Peak 15–30 minutes; duration 3–4 hours
NPH insulin	Peak 8–12 hours; duration 18–24 hours
Insulin glargine	No peak; duration < 24 hours

Define each of the following complications of insulin treatment:

Somogyi effect	Nocturnal hypoglycemia causing elevated morning glucose due to release of counterregulatory hormones; treat with less insulin
Dawn phenomenon	Early morning hyperglycemia secondary to nocturnal growth hormone (GH) release

What is the first-line treatment for type 2 diabetes?

Metformin

In what patients would metformin be avoided?

In patients who have compromised kidney function; causes lactic acidosis

How do we believe metformin works?

Increases sensitivity to insulin

Give an example of each of the following classes of hypoglycemic agents, how they work, and major side effects:

Sulfonylureas	Example: glipizide, glyburide (Note: **Start** with GL or end with IDE) How it works: increased insulin secretion by B cells Side effects: hypoglycemia and teratogenic (except glyburide)
Thiazolidinediones	Examples: rosiglitazone (Avandia), pioglitazone (Actose) (end with glitazone) How it works: increases sensitivity to insulin (Note: The **zone** for sensitivity to insulin is increased) Side effects: hepatitis so patients on this class of drugs should have liver enzymes monitored for first year that they are on the drug

When is it most appropriate to treat a type 2 diabetic with insulin?

Refractory to oral hypoglycemic agents

What medication slows the progression of nephropathy in diabetes?

Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs)

Other than medication, what other therapy is important in diabetes?

Nutrition education

What is HgA1c?

Blood marker of glucose control over the last 3 months. HgA1c < 7 is ideal.

What preventative measures are recommended to minimize diabetic complications?

1. Lipid control (low-density lipoprotein [LDL] < 100, TG < 150)

2. BP control < 130/80

3. HgA1C < 7.0

4. Foot checks

5. Check for microalbuminuria, proteinuria

6. Annual funduscopic examination

What is the appropriate treatment in a diabetic patient with microalbuminuria?

ACE inhibitor or ARB

What is the major complication of type1 diabetes?

Diabetic ketoacidosis (DKA)

What are the signs and symptoms of DKA?

Severe hyperglycemia (glucose often >500), ketoacidosis, hyperkalemia, fruity breath, slow deep breaths, abdominal pain, dehydration, lethargy

What are slow deep breaths in DKA called?

Kussmaul hyperpnea

What is the most important treatment in DKA?

Intravenous (IV) fluid hydration(usually with normal saline)

What are the other treatments in DKA?

Insulin drip. Add potassium if potassium is low or normal**and** add glucose when blood sugar reaches 250 because insulin needs to be continued to be given despite normal glucose until ketones are no longer present.

What are the most severe complications of DKA treatment?

Cerebral edema or cardiac arrest due to hyperkalemia

What is the major complication of type 2 diabetes?

Hyperosmolar hyperglycemic nonketotic (HHNK) coma; although on rare occasions type 2 diabetics can also go into DKA

What are some of the signs and symptoms of HHNK?

Hypovolemia, hyperglycemia (glucose can be >1000),**no ketoacidosis,** renal failure, altered mental status, seizure, disseminated intravascular coagulation (DIC); often precipitated by acute stress such as trauma or infection

The difference between this and DKA is that in HHNK there is no ketoacidosis

What is the treatment for HHNK?

The mortality is >50%; as a consequence, immediate treatment is urgent. Treatment includes rapid IV fluid resuscitation; insulin and potassium are usually needed earlier than in DKA because the intracellular shift of plasma potassium during therapy is accelerated in the absence of acidosis

PITUITARY

What hormones are secreted from the anterior pituitary?

Follicle-stimulating hormone (FSH), luteinizing hormone (LH), adrenocorticotropic hormone (ACTH), thyroid-stimulating hormone (TSH), prolactin, GH (Note:**FAST P:G)**

What hormones are secreted from the posterior pituitary?

Antidiuretic hormone, oxytocin, vasopressin

What is the action of each of the following hormones?

FSH	Spermatogenesis in males, ovarian follicle growth in females
LH	Testosterone secretion in males and ovulation in females
ACTH	Stimulates adrenal cortex to make cortisol, aldosterone, and sex hormones
TSH	T3 and T4 production as well as thyroid gland maturation
Prolactin	Milk production (lactation)
GH	Insulin-like growth factor secretion causing protein and fat metabolism
Antidiuretic hormone (ADH), vasopressin	Production of concentrated urine by sodium and water retention
Oxytocin	Uterine contractions, milk letdown

What is the most common type of pituitary tumor?

Prolactinoma

What type of tumor is a prolactinoma?

A pituitary adenoma which secretes prolactin

What are the two mechanisms by which a prolactinoma causes symptoms?

1. Endocrine effect: due to hyperprolactinemia

2. Mass effect: pressure of the tumor on surrounding tissues

What are some signs and symptoms of a prolactinoma?

Headache, diplopia, hypogonadism, amenorrhea, gynecomastia, galactorrhea,

hypopituitarism

What cranial nerve (CN) can be affected by a prolactinoma?

CN III

How is a prolactinoma diagnosed?

Magnetic resonance imaging (MRI)/computed tomography (CT)

What is the first-line treatment for a prolactinoma?

Dopamine agonist such as bromocriptine

What are other treatment options?

Surgical resection or radiation therapy if tumor is very large or refractory to medical treatment

Other than a prolactinoma, what are other causes of hypopituitarism?

Sheehan syndrome (postpartum pituitary necrosis), hemochromatosis, neurosyphilis, tuberculosis (TB), surgical destruction of pituitary

What disorder is seen with elevated levels of GH?

Acromegaly

What is the most likely underlying cause of acromegaly?

Pituitary adenoma secreting GH

When must there be an elevation in GH in order for acromegaly to result?

Elevated levels of GH must be present after epiphyseal closure

What results if there is excess GH secretion before epiphyseal closure?

Gigantism

What are the signs and symptoms of acromegaly?

Coarse facial features, large hands and feet, large jaw, deepening of voice, decreased peripheral vision due to compression of optic chiasm, hyperhidrosis

How is acromegaly diagnosed?

MRI/CT demonstrating pituitary tumor or nonsuppressible GH postoral glucose challenge and elevtated IGF-1 (insulin-like growth factor)

What are the treatment options for acromegaly?

Surgery or radiation of pituitary tumor, or medical treatment with octreotide or somatostatin, which blocks GH or dopamine agonists

What malignancy are patients with acromegaly at increased risk for?

Colon cancer

THYROID

What is hyperthyroidism?

Increased secretion of thyroid hormones

In what sex is hyperthyroidism more common?

Ten times more common in women than men

What is the most common cause of hyperthyroidism?

Graves disease (80%–90% of U.S. cases)

What are some other causes of hyperthyroidism?

Plummer disease (toxic adenoma); toxic multinodular goiter; subacute thyroiditis; amiodarone therapy

What are some of the signs and symptoms of hyperthyroidism?

Heat intolerance, weight loss, exophthalmos,tachycardia, anxiety, palpitations, atrial fibrillation, tremor, sweating, fatigue, weakness, diarrhea, increased reflex amplitude

What is Graves disease?

Autoimmune disease causing hyperthyroidism. It is due to antibody stimulation of TSH receptors causing excess secretion of free thyroid hormone.

What are the two symptoms only seen in Graves disease?

1. Pretibial myxedema

2. Infiltrative ophthalmopathy

What is pretibial myxedema?

Pruritic, nonpitting edema found on shins that usually remits spontaneously

What is infiltrative ophthalmopathy?

Exophthalmos that may not resolve despite treatment of Graves disease most likely due to autoimmune damage in extraocular muscles

How is Graves disease diagnosed?

All hyperthyroidism is diagnosed via measurement of TSH, free T4, and free T3. In Graves disease, since there is excess stimulation of the thyroid gland causing increased production of thyroid hormone, laboratory tests show high levels of free T4 and free T3, and low levels of TSH (because of negative feedback) (Table 9-1). Also, a radioactive iodine uptake scan should be done. If uptake is low, then thyroiditis or medication-induced hyperthyroidism is considered.

Table 9-1 Thyroid Function Evaluation

Hyperthyroid	TSH	Free T4	TRH
Graves disease	↓	↑	↑
Pituitary tumor	↑	↑	↓
Plummer disease	↓	↑	↑
Hypothyroid			
Primary	↑	↓	↑
Secondary	↓ or normal	↓	↓
Tertiary	↓ or normal	↓	normal
Hashimoto	↓ or normal	↑	↑ or normal

Abbreviation: TRH, thyrotropin-releasing hormone.

What is another name for toxic multinodular goiter?

Plummer’s disease

What is the underlying cause of hyperthyroidism in Plummer disease?

Multiple thyroid nodules develop autonomous T4 secretion and, therefore, more T4 is released.

How is Plummer disease diagnosed?

Radioactive iodine uptake tests show “hot” nodules with the rest of the gland being “cold”; also, clinically, nodules can sometimes be felt.

What is another name for subacute thyroiditis?

de Quervain thyroiditis

What are the signs and symptoms of subacute thyroiditis?

Prodrome of viral urinary tract infection (UTI) followed by rapid onset of thyroid swelling and**tenderness** as well as hyperthyroid symptoms that can later turn into a hypothyroid state.

What is the treatment for de Quervain thyroiditis?

Usually self-limiting, but asprin and corticosteroids may be indicated to control inflammation

What are the treatment options for a hyperthyroid state?

1. Medication: propylthiouracil (PTU) or methimazole

2. Radioactive iodine ablation

3. Surgery: subtotal thyroidectomy

What is the first-line treatment for Graves disease?

Radioactive iodine ablation

What is radioactive iodine ablation?

Radioactive iodine is concentrated in the gland and destroys tissue

What are the possible sideeffects of radioactive iodine ablation?

Hypothyroidism; thyrotoxic crisis secondary to the release of thyroid hormone into the blood stream

What is the mechanism by which PTU works?

It inhibits the peripheral conversion of T4 to T3, decreases iodine uptake, decreases T4 synthesis

Do patients need to be on therapy for the rest of their lives?

No. After a 1–2 year course of treatment about 50% no longer need to be treated.

What is the potential side effects of PTU?

Leukopenia, rash, nausea

What other adjunctive treatment is given to patients with hyperthyroidism?

Beta-blocker, usually propranolol, to control symptoms

What is the most serious complication of hyperthyroidism?

Thyroid storm

What can induce thyroid storm?

Infection, surgery, trauma, abrupt stop of antithyroid medication, serious acute medical problems such as cerebrovascular accident (CVA) or myocardial infarction (MI)

What are the signs and symptoms of thyroid storm?

Exaggerated symptoms of hyperthyroidism are tachycardia, high output**congestive heart failure (CHF),** abdominal pain, fever, altered mental status (ultimately coma)

What is the mortality rate of thyroid storm?

Up to 50%

What is the initial treatment for thyroid storm?

It is an emergency so think of the ABCs:

Airway stabilization

Breathing/oxygen administration

Circulation (check pulse/blood pressure [BP]) and start IV fluids

After primary stabilization of the patient, what is the medical management of thyroid storm?

Beta-blocker, PTU, or methimazole. Tylenol for fever, cold iodine about 2 hours after PTU, and glucocorticoids.

What are the signs and symptoms of hypothyroidism?

Cold intolerance, fatigue, lethargy, weakness,**constipation, weight gain,** arthralgias, hoarse voice, skin is dry, coarse, and with nonpitting edema, loss of outer third of eyebrows, delayed relaxation phase of deep tendon reflexes

What is primary hypothyroidism?

Thyroid gland dysfunction

What are some examples of primary hypothyroidism?

Hashimoto thyroiditis, thyroid ablation or neck radiation therapy in the past, subacute thyroiditis, iodine excess or deficiency, medication-induced

What medication can cause hypothyroidism?

Lithium

What is the most sensitive lab test for primary hypothyroidism?

Elevated TSH

What other lab results are present in primary hypothyroidism?

Low T3 and T4

What is Hashimoto thyroiditis?

Painlesschronic autoimmune thyroid inflammation of autoimmune etiology

What laboratory results can help diagnose Hashimoto thyroiditis?

Elevated antithyroglobulin and antimicrosomal antibody titers

What is subacute thyroiditis?

Tender,enlarged thyroid; often post-viral infection can begin with hyperthyroid symptoms, then hypothyroid symptoms

How can you distinguish Hashimoto from subacute thyroiditis?

On clinical examination, in Hashimoto the thyroid gland is**not** tender to palpation but in subacute thyroiditis it is**tender** to palpation.

How can Graves disease and increased Hashimoto's thyroiditis be distinguished?

Radioactive iodine uptake is with Graves and**decreased** with Hashimoto.

What is secondary hypothyroidism?

Hypothyroidism caused by pituitary dysfunction?

What are some examples of secondary hypothyroidism?

Sheehan syndrome, pituitary neoplasm, TB

What is Sheehan syndrome?

Postpartum pituitary necrosis

What lab results indicate a secondary hypothyroidism?

Low to normal TSH as well as**normal thyrotropin-releasing enzyme (TRH),** low levels of T3 and T4

What is tertiary hypothyroidism?

Deficiency of TRH

What is an example of tertiary hypothyroidism?

Hypothalamic radiation

Other than TSH, TRH, T3, T4, what other abnormal lab tests may be found in a hypothyroid patient?

Elevated serum cholesterol (TG, LDL, total cholesterol); elevated aspartate aminotransferase (AST) and alanine aminotransferase (ALT); anemia; hyponatremia

What is the treatment for hypothyroidism?

Levothyroxine

What is subclinical hypothyroidism?

Elevated TSH levels but with normal thyroid hormone levels and with no clinical symptoms

What is the life-threatening complication of hypothyroidism called?

Myxedema coma

What are the signs and symptoms of myxedema coma?

Severe lethargy or coma, hypothermia, areflexia, bradycardia

What causes myxedema coma?

Prolonged cold exposure, infection, sedatives, narcotics, trauma, or surgery

What is the treatment for myxedema coma?

This is an emergency, so start with ABCs (airway, breathing, circulation); IV fluids, steroids, levothyroxine, treat any precipitating causes

What is the initial appropriate workup of a thyroid mass?

Fine needle biopsy and TSH

What other studies are done to workup a thyroid mass?

Thyroid ultrasound to determine the number and sizes of masses; and thyroid technetium 99m scan

What is a hot nodule and a cold nodule on a thyroid scan?

Hot nodule indicates a hyperactive nodule and is**less** likely to be malignant. A cold nodule indicates a hypoactive nodule that is**more** likely to be malignant

What is the most common type of thyroid cancer?

Papillary cancer

What is the prognosis for papillary cancer?

85%, 5-year survival

What is seen on pathology?

Psammomabodies, Orphan Annie nucleus

Which type of thyroid carcinoma is associated with multiple endocrine neoplasia types 2 and 3 (MEN 2 and 3)?

Medullary cancer

What can be used to monitor medullary carcinoma?

Calcitonin, because it is a calcitonin-secreting tumor

Which type of thyroid carcinoma has the worst prognosis?

Anaplastic cancer

In what patient population is anaplastic carcinoma usually found?

Older patients

What is the 5-year prognosis for anaplastic carcinoma?

0% survival at 5 years

Which thyroid cancer has the second worst prognosis?

Medullary cancer

Which thyroid carcinoma often has metastasis to the bone and lungs?

Follicular cancer

Name the tumors that are part of each of the MEN syndromes?

MEN 1: Wermer syndrome. three P's:**prolactinoma, parathyroid, pancreatoma**

MEN 2: Sipple syndrome:**pheochromocytoma, medullary thyroid, parathyroid**

MEN 3: same as MEN 2B:**pheochromocytoma, medullary thyroid, mucocutaneous neuromas**

PARATHYROID

What is primary hyperparathyroidism?

Increased secretion of parathyroid hormone (PTH)

What is the most common cause of primary hyperparathyroidism?

Adenoma is the most common cause; however, other etiologies include hyperplasia, carcinoma, MEN 2 or 3

What does elevated PTH cause?

There is an ultimate increase in serum calcium (**hypercalcemia**) because PTH leads to increased vitamin D hydroxylation and, therefore, increased calcium resorption as well as decreased resorption of phosphate (**hypophosphatemia**). Calcium levels are also increased because of increased osteoclastic activity**(osteoporosis)** .

What are the signs and symptoms of hyperparathyroidism?

Same as those for hypercalcemia. "**Stones, maons, groans, and psychiatric overtones.**" Because of the osteoclastic activity it can also lead to osteoporosis.

What EKG finding could you expect with hyperparathyroidism?

Shortened QT, because of hypercalcemia

How is hyperparathyroidism diagnosed?

Hypercalcemia, hypophosphatemia, hypercalciuria, and PTH level

What other differential diagnoses should be considered with hypercalcemia?

Neoplasm, sarcoidosis, thiazide diuretic treatment, Paget disease, vitamin D intoxication, milk alkali syndrome, myeloma

What is the acute medical treatment for hyperparathyroidism?

Asymptomatic patients with calcium levels below 13 should just be watched. However,

symptomatic patients or those with higher calcium levels should be treated with furosemide and bisphosphonates to decrease bone resorption and prevent osteoporosis. Calcitonin can be used as well.

What long-term treatment must be considered in hyperparathyroidism?

Surgical treatment. Adenomas should be removed. In hyperplasia, all four parathyroids are removed and a small piece is placed usually near the sternocleidomastoid for functionality.

What are the most common complications of parathyroidectomy?

Hoarseness because of damage of the recurrent laryngeal nerve and hypocalcemia

What is secondary hyperparathyroidism?

Increased PTH secretion secondary to chronic renal failure or vitamin D deficiency

What is hypoparathyroidism?

Decreased PTH

What are the causes of hypoparathyroidism?

Idiopathic, secondary to surgery or neck irradiation, DiGeorge syndrome, hypomagnesemia

Why does hypomagnesemia lead to hypoparathyroidism?

Because magnesium is necessary for the parathyroid to secrete PTH

In what conditions is low magnesium seen?

Syndrome of inappropriate secretion of antidiuretic hormone (SIADH), pancreatitis, alcoholism

How is hypoparathyroidism diagnosed?

Hypocalcemia, hyperphosphatemia, low PTH

What are the signs and symptoms of hypoparathyroidism?

Same as that for hypocalcemia: perioral paresthesias, tetany, seizures, Trousseau sign, Chvostek sign, anxiety

What EKG findings could you expect in hypoparathyroidism?

Prolonged QT interval because of the hypocalcemia

What is Trousseau sign?

Carpal spasm with arterial occlusion with BP cuff

What is Chvostek sign?

Spasm of the facial nerve upon tapping

How is hypoparathyroidism treated?

Emergently treat with IV calcium, then treat with vitamin D and oral calcium for maintenance treatment

ADRENALS

What are the two main parts of the adrenal gland and what is the secretory product of each part?

The adrenal cortex and the adrenal medulla make up the adrenal gland. The cortex secretes aldosterone, cortisol, and sex hormones and the medulla secretes the catecholamines including epinephrine and norepinephrine.

What is the function of aldosterone?

Kidney resorption of sodium and secretion of potassium and hydrogen ions

What is Addison disease?

Primary adrenal insufficiency caused by the destruction of the adrenal cortex leading to a deficiency in both mineralocorticoids as well as glucocorticoids

What is secondary adrenal insufficiency?

Decreased secretion of ACTH by the pituitary gland; the adrenal gland is functional

What is the cause of tertiary adrenal insufficiency?

Decreased hypothalamic function

What is the most likely etiology of Addison disease?

Autoimmune destruction of the adrenal gland

What are some other causes of Addison disease?

TB, amyloidosis, sarcoidosis, HIV, adrenal hemorrhage secondary to DIC or trauma, Waterhouse-Friderichsen syndrome, congenital adrenal hyperplasia, metastasis to the adrenals

What is Waterhouse-Friderichsen syndrome?

Endotoxin-mediated adrenal hemorrhage usually caused by meningococcemia

What is the most likely cause of secondary adrenal insufficiency?

Hypothalamic-pituitary axis disturbance, usually by sudden cessation of exogenous corticosteroids, which leads to decreased ACTH secretion

What are some other causes of secondary adrenal insufficiency?

Pituitary infarction, Sheehan syndrome, pituitary adenoma

What are some signs and symptoms of Addison disease?

Because of low aldosterone and cortisol there are hyponatremia, hyperkalemia, pica (craving for salt), weakness, anorexia, hypotension, nausea, vomiting, hyperpigmentation

What are the diagnostic findings in primary adrenal insufficiency?

Hyperpigmentation,? ACTH, ? cortisol and aldosterone response to ACTH challenge

What is the test used to diagnose adrenal insufficiency?

ACTH (Cortrosyn) test in which a dose of ACTH is given to the patient and then serum cortisol levels as well as serum ACTH levels are measured.

Primary adrenal insufficiency: ?**cortisol**levels in response to ACTH and ?**aldosterone**levels

Secondary adrenal insufficiency: ?**cortisol**levels in response to ACTH and**normal aldosterone** levels

How is the diagnosis of secondary adrenal insufficiency distinguished from primary adrenal insufficiency?

No hyperpigmentation,?cortisol response, ?ACTH

What kind of metabolic disturbance is seen in primary adrenal insufficiency?

Metabolic acidosis due to aldosterone and cortisol deficiency and, therefore, lack of secretion of hydrogen ions

What is the treatment for adrenal insufficiency?

Glucocorticoid replacement. Extra glucocorticoids should be given in times of physical stress such as infection. You should instruct patients to taper off this extra replacement slowly as to prevent an adrenal crisis.

What is Cushing syndrome?

A term used to describe the symptoms caused by hypercortisolism

How is Cushing*syndrome* different from Cushing*disease?*

Cushing disease refers to a type of Cushing syndrome caused specifically by ACTH hypersecretion by the pituitary

What are the different causes of hypercortisolism?

1. Exogenous glucocorticoids

2. Pituitary hypersecretion of ACTH

3. Hypersecretion of cortisol due to adrenal hyperplasia/neoplasm

4. Ectopic ACTH production such as with small cell lung carcinoma

What is the most common cause of Cushing syndrome?

Exogenous corticosteroids

What is the most common cause of endogenous hypercortisolism?

Cushing**disease** (pituitary hypersecretion of ACTH)

What are the signs and symptoms of Cushing syndrome?

Buffalo hump,moon facies, truncal obesity, striae, virilization/menstrual disorders, hyperglycemia, hypertension, hypokalemia, immune suppression, osteoporosis, hirsutism, acne

What tests are used to diagnose hypercortisolism?

24-hour urine-free cortisol and the dexamethasone suppression tests, ACTH level, diurnal cortisol variation

What is the dexamethasone suppression test?

First a low dose of dexamethasone is given and cortisol is measured. If cortisol is not elevated then Cushing is ruled out; if it is elevated then a high-dose dexamethasone suppression test is done and ACTH is measured. If ACTH is decreased then the pituitary has good feedback and, therefore, it must be an adrenal etiology. However, if the ACTH is high or normal then it is probably ectopic ACTH; and if it is only partially suppressed, then the pituitary is the etiology.

Dexamethasone ? ? ACTH (ectopic/pituitary)

?ACTH (adrenal)

What are some other studies to consider to localize the lesion in hypercortisolism?

A CT scan can look for an adrenal mass and an MRI can look for a pituitary mass.

What is the treatment for hypercortisolism?

Treat the underlying cause. If it is a resectable tumor, tumor resection with postoperative glucocorticoids. In nonresectible tumors, medical therapy with ketoconazole, mitotane, metyrapone, aminoglutethimide. If the etiology is exogenous glucocorticoids, taper off the glucocorticoids and eventually stop

What is Conn syndrome?

Primary hyperaldosteronism

What is the etiology of Conn syndrome?

Either hyperplasia of the zona glomerulosa or aldosterone-producing adenoma

What are the signs and symptoms of Conn syndrome?

Hypertension,muscle cramps, palpitations, polyuria, polydipsia, hypokalemia

What percent of hypertensive patients have Conn syndrome?

1%–2%

What are some of the laboratory findings in Conn syndrome?

?Na, ?Cl, ?K (muscle cramps, palpitations), ?renin-angiotensin feedback, metabolic alkalosis

How is Conn syndrome diagnosed?

Captopril stimulation test:captopril (an ACE inhibitor) is administered and then serum renin and aldosterone levels are measured. ?aldosterone and ?**renin** confirm the diagnosis

What is the renin level in Conn syndrome?

Low renin

What other study can help in the diagnosis of Conn syndrome?

CT demonstrating an adrenal nodule or hyperplasia

What is the treatment for Conn syndrome?

Adrenal adenoma: resection of tumor; unilateral adrenal hyperplasia: unilateral adrenalectomy; bilateral adrenal hyperplasia: spironolactone (potassium-sparing diuretic) or ACE inhibitor to control blood pressure

What is secondary hyperaldosteronism?

Elevated aldosterone levels due to elevated renin levels secondary to renal ischemia in CHF, renal artery stenosis, shock, renal tumor.

How is secondary hyperaldosteronism diagnosed?

?**Renin**

What can be measured to differentiate primary from secondary hyperaldosteronism?

Renin(this is very important)

What is the treatment for secondary hyperaldosteronism?

Treat the hypertension with a potassium-sparing diuretic, a beta-blocker, and treat the underlying cause

What is a pheochromocytoma?

Tumor of the adrenal**medulla** that produces excess**catecholamines**

What percentage of people with hypertension have a pheochromocytoma?

0.5%

What are the possible etiologies for a pheochromocytoma?

MEN 2 or 3, von Hippel-Lindau disease, Recklinghausen disease, neurofibromatosis

What are the five P's of pheochromocytoma?

1. Pain (headache)

2. Pressure

3. Perspiration

4. Palpitation

5. Pallorand hypertension

What is the most common sign of a pheochromocytoma?

Hypertension

What is the diagnostic test for a pheochromocytoma?

Urine screen for elevated**VMA** (vanillylmandelic acid), a urine catecholamine; as well as elevated urine and serum epinephrine and norepinephrine levels

What other test can be done to localize a pheochromocytoma?

A CT scan can identify a**suprarenal mass** (adrenal mass).

What are some other laboratory findings in a pheochromocytoma?

Hyperglycemia, hypercalcemia, polycythemia

What is the "rule of 10's" for a pheochromocytoma?

10% malignant

10% bilateral

10% extrarenal

10% familial

10% in kids

10% multiple tumors

10% calcified

What must be ruled out in a patient with a pheochromocytoma?

MEN type II or III

What is the treatment for a pheochromocytoma?

In operative cases preoperative alpha-blockers and beta-blockers, then surgical resection

In inoperable cases**phe** noxybenzamine**or phe** ntolamine

Why treat with preoperative alpha-blockers and beta-blockers?

To prevent unopposed vasoconstriction and thus, volume depletion

BONES

What is osteoporosis?

Reduction in bone mass leading to increased risk of fracture

What are the risk factors for osteoporosis?

Female, postmenopausal or low estrogen state, hypercortisolism, hyperthyroidism, calcium deficiency, low physical activity, smoking, ACE inhibitors

What are the typical fractures that occur in osteoporosis?

Hip, vertebrae, and Colle fractures

How is osteoporosis diagnosed?

Dual-energy x-ray absorptiometry (DEXA) scan which shows low bone density or an incidental fracture in the elderly

What are the treatments for osteoporosis?

Bisphosphonates, calcitonin, selective estrogen receptor modulators, calcium

How much calcium should be taken daily?

1500 mg daily with vitamin D

What is the calcitonin most useful for?

Treating bone pain; however, it cannot be used chronically because the effects wear off

What are some examples of selective estrogen modulators?

Tamoxifen, raloxifene

What do the selective estrogen modulators increase the risk for?

Thromboembolism

What is osteomalacia?

Vitamin D deficiency in adults

What is osteomalacia called in children?

Rickets

What are the signs and symptoms in children?

Pigeon breast, craniotabes(thin skull bones),**rachitic rosary** (chostocondral thickening)

How is osteomalacia diagnosed?

Low levels of vitamin D as well as diffuse osteopenia on x-ray

How is osteomalacia treated?

Vitamin D supplementation

What is Paget disease of the bone?

Localized hyperactivity of the bone which leads to disordered bone matrix being replaced with soft, enlarged bone

What is the etiology of Paget?

Unknown, but some think it may be viral

What are the signs and symptoms of Paget disease of the bone?

Hearing loss(impingement of cranial nerve [CN] VIII), multiple fractures, bone pain, high-output cardiac failure,**increased hat size**

What is the typical finding on x-ray?

Hyperlucent area surrounded by hyperdense border-sclerotic lesions

How is Paget diagnosed?

Elevated alkaline phosphatase, sclerotic lesionson bone scans/x-rays

What are the complications associated with Paget disease of the bone?

Pathologic fractures, high-output cardiac failure, hearing loss, kidney stones, sarcoma, spinal cord compression

What is the treatment for Paget disease?

Most patients do not need treatment; however, patients with complications associated with Paget's, are treated with bisphosphonates as first line and calcitonin as second line.

CHAPTER 10

Infectious Disease

HIV/AIDS

What does HIV stand for?

Human immunodeficiency virus

What is HIV?

A retrovirus that destroys CD4 cells

How is HIV transmitted?

Sexual contact, blood products, mother to child in HIV positive mothers, needle stick injury

How is acquired immunodeficiency syndrome (AIDS) defined?

CD4 count < 200 or evidence of an AIDS defining condition or T-helper cell < 200%

Describe the life cycle of HIV?

gp120 bind CD4 molecule ? gp41 molecule helps HIV to fuse with host cell ? HIV RNA released into host cell ? reverse transcriptase converts viral RNA into DNA ? viral DNA translocates into nucleus and viral DNA fuses with host DNA ? host cell transcribes the integrated DNA ? mRNA is translated into HIV polypeptides which are cleaved by viral proteases ? new virus particles assemble to create a new virus cell

How is an HIV infection diagnosed?

A positive enzyme-linked immunosorbent assay (ELISA) for HIV is then confirmed with a Western blot assay

How is HIV ruled out?

A negative ELISA for HIV

What marker is used to follow the*extent* of disease?

CD4 count

What can be used as a marker of disease*progression?*

Viral load (it will tell how well the treatment is working)

What are the signs and symptoms of acute HIV?

Flu-like symptoms that can later subside

Name the complications associated with each of the following CD4 counts:

> 500	Multiple episodes of vaginal candidiasis; lymphadenopathy
< 400	Pneumonia, pulmonary TB, oral candidiasis, shingles, Kaposi, non-Hodgkin lymphoma
< 200	*Pneumocystis carinii* pneumonia (PCP), wasting, dementia
< 100	*Cryptococcus* or toxoplasmosis infections
< 50	Mycobacterium avium complex (MAC), central nervous system (CNS) lymphoma, cytomegalovirus (CMV), cryptosporidiosis

When should antiretroviral therapy be initiated?

At CD4 counts < 350

What is the antiretroviral therapy called?

Highly active antiretroviral therapy (HAART) therapy

What does HAART therapy usually include?

Two nucleoside analogues and a protease inhibitor

Name the medical management that should be initiated for each of the following CD4 counts:

CD4 < 200	Start prophylaxis against PCP pneumonia and toxoplasmosis with Bactrim
CD4 < 100	Start prophylaxis against MAC with clarithromycin or azithromycin
CD4 < 50	Start prophylaxis against fungal infections with fluconazole

Name the AIDS-related opportunistic infection/complication associated with the following:

Presents as nonproductive cough	PCP pneumonia
Vascular nodules on the skin	Kaposi sarcoma
Most common cause of AIDS death in the United States	Disseminated MAC
Most common fungal infections in HIV	Candidiasis
Most common cause of meningitis in AIDS	*Cryptococcus*

Presents as painless progressive vision loss	CMV retinitis
Painful vesicular eruptions	Shingles
Human herpes virus (HHV)-6, 8	Kaposi sarcoma
Bilateral interstitial infiltrates on chest x-ray (CXR)	PCP pneumonia
Ring enhancing lesion on head computed tomography (CT)	Toxoplasmosis
Perivascular hemorrhages and exudates on funduscopic examination	CMV
Elevated alkaline phosphatase	MAC

What is the treatment for each of the following opportunistic infections?

PCP	Bactrim + glucocorticoids
Toxoplasmosis	Pyrimethamine + sulfadiazine
MAC	Clarithromycin + ethambutol
Cryptococcus	Amphotericin B + fluconazole
CMV	Ganciclovir, foscarnet
Shingles	Acyclovir
Esophageal candidiasis	Fluconazole, ketoconazole
Herpes simplex virus (HSV)	Acyclovir, foscarnet

SEXUALLY TRANSMITTED DISEASES

Which sexually transmitted disease (STD) is caused by the spirochete*Treponema pallidum?*

Syphilis

Name the stage of syphilis associated with the following:

Painless chancre (ulcer) near the area of contact that often heals spontaneously	Primary syphilis
Fever, malaise, lymphadenopathy, maculopapular rash on soles and palms, condylomata lata	Secondary syphilis (1–2 months after infection)
Positive serology but asymptomatic and < 1 year of infection	Early latent
> 1 year of infection with possibly positive serology	Late latent
Gummas, tabes dorsalis, Argyll Robertson pupil, aortitis, aortic regurgitation, aortic root aneurysm	Tertiary syphilis

What are gummas?

Rubbery granulomatous lesions in CNS, aorta, heart, skin, bone

What is tabes dorsalis?

Posterior column degeneration

How is syphilis diagnosed?

Four possible tests

1. VDRL/rapid plasma reagin (RPR)-rapid test, however nonspecific blood test (eg, can be falsely positive in systemic lupus erythematosus [SLE]).

2. Dark-field microscopy would show motile spirochetes.

3. EIA (enzyme immunoassay): tests for antitreponemal IgG; can be used to screen for syphilis.

4. FTA-ABS/MHA-TP (fluorescent treponemal antibody/microhemagglutination assay—*T. pallidum*): sensitive and specific; it remains positive for life.

What is the treatment for syphilis?

Penicillin; doxycycline or tetracycline can be given to penicillin-allergic patients (but not for CNS disease)

Which STD often coexists with gonorrhea?

Chlamydia

How can*Chlamydia* present?

Asymptomatic, cervicitis, urethritis, salpingitis or pelvic inflammatory disease (PID)

What are the signs and symptoms of*Chlamydia* infection with PID?

Mucopurulent discharge with adnexal pain

What is Fitz-Hugh-Curtis syndrome?

Complication of gonorrhea or*Chlamydia* in which there is perihepatic inflammation and fibrosis.

What is lymphogranuloma venereum?

Systemic disease caused by the*Chlamydia* L serotype causing painful lymphadenopathy called buboes

What is the treatment for*Chlamydia* infection?

Doxycycline or azithromycin

What sexually transmitted disease is caused by a gram-negative diplococcus?

Neisseria gonorroheae

What is a major complication of gonorrhea?

PID

On what type of medium is gonorrhea diagnosed?

Thayer-Martin

How is gonorrhea treated?

Third-generation cephalosporin with concomitant treatment of*Chlamydia*

How is PID diagnosed?

Cervical motion tenderness plus at least one of the following: positive Gram stain; fever; elevated WBCs, tubo-ovarian abscess; pus on culdocentesis

What is the most common cause of vaginitis?

Bacterial vaginosis caused by*Gardnerella*

What are the signs and symptoms of vaginitis?

Vaginal itching, burning, bad odor, discharge, and dyspareunia

What is the classic odor associated with bacterial vaginosis?

Fishy odor =**positive Whiff test**

How is bacterial vaginosis diagnosed?

Clue cells (epithelial cells coated with bacteria) on wet mount

How is bacterial vaginosis treated?

Metronidazole

Which type of vaginitis is caused by a flagellated, motile protozoan?

Trichomonas

What are the classic symptoms of*Trichomonas* infection?

Fishy odor of discharge and**strawberry cervix**

How is*Trichomonas* treated?

Patient and partner are treated with metronidazole

Which type of vaginitis is associated with a cheesy white discharge?

Candida(also known as yeast infection)

How is candidiasis diagnosed?

Pseudohyphae on wet mount

How is a*Candida* infection treated?

Nystatin cream or oral diflucan

Which types of human papillomavirus (HPV) are associated with cervical cancer?

16, 18, 31, 45, 51, 52, 53

What is the new vaccine approved to protect against cervical cancer?

Gardasil

Who can get the vaccine?

Females aged 12–26

When should a female start getting Pap smears?

Age 18 or first sexual activity, whichever comes first

How often should a Pap smear be done?

If a patient has had three normal consecutive Pap smears, they can get them every 3 years.

SEPSIS

What is sepsis?

An infection that causes systemic inflammatory response syndrome (SIRS)

What is septic shock?

Sepsis-induced hypotension

What type of bacteria causes shock secondary to exotoxin-induced fluid loss?

Gram-positive bacteria

What type of bacteria causes shock secondary to endotoxin-induced vasodilatation?

Gram-negative bacteria

What are some of the signs and symptoms of sepsis?

Fever, hypotension, tachycardia, tachypnea, disseminated intravascular coagulation (DIC), increased cardiac output

What is the treatment of sepsis?

Intravenous (IV) fluids, antibiotics to treat infection, vasopressors, remove potential sources of infection such as Foley catheter, sometimes steroids

OSTEOMYELITIS

What is osteomyelitis?

Bone infection

What are the two main routes of bone infection?

Direct spread from soft tissue infection or hematogenous seeding

What type of patients are predisposed to getting osteomyelitis by direct spread?

Diabetics, people with peripheral vascular disease, deep soft tissue injuries

What is the most common organism causing osteomyelitis?

Staphylococcus aureus

What is the most common cause of osteomyelitis in a patient with sickle cell anemia?

Salmonella

What are the two most common causes of osteomyelitis in a patient who is an IV drug user?

Pseudomonas,S. aureus

What is the most common cause of osteomyelitis in a patient with a deep foot puncture wound?

Pseudomonas

What are the signs and symptoms of osteomyelitis?

Fever, bone pain, warmth, swelling, erythema of overlying skin, with limited range of motion of the area affected

What is the classic finding on x-ray?

Periosteal elevation; lytic lesion

What is the gold standard diagnostic technique to evaluate osteomyelitis?

Magnetic resonance imaging (MRI)

What is the treatment for osteomyelitis?

Appropriate IV antibiotics for 4–6 weeks

What are possible complications of osteomyelitis?

Chronic osteomyelitis, sepsis, septic arthritis, squamous cell carcinoma secondary to a draining sinus tract

CHAPTER 11

Dermatology

TERMINOLOGY

Name the primary dermatologic skin lesion described below:

Flat, nonpalpable area of discoloration < 1 cm in diameter	Macule
Elevated, palpable skin lesion < 1 cm in diameter	Papule
Elevated skin lesion > 1 cm in diameter	Plaque
Fluid-filled lesion < 0.5 cm in diameter	Vesicle
Fluid-filled lesion > 0.5 cm in diameter	Bullae
Circumscribed, elevated pus-filled lesion	Pustule
Circumscribed, elevated area of edema that occurs transiently	Wheal

Circumscribed, elevated solid lesion > 0.5 cm	Nodule
Red-purple, nonblanching, pinpoint lesion due to hemorrhage into the skin	**Petechiae**
Red-purple, nonblanching lesion > 0.5 cm in diameter	Purpura (also known as bruise)
Blanchable lesion due to dilated blood vessel	Telangiectasia
Flat-topped thickening of skin usually due to prolonged scratching	Lichenification
Type of lesion seen in any type of thrombocytopenia	Petechiae

SKIN CANCERS

What is the most common type of skin cancer?

Basal cell carcinoma (BCC)

What are the three main characteristic of a BCC seen on physical exam?

1. Pearly papule

2. Telangiectasias

3. Traslucent border

What is the classic description of a BCC?

“Rodent ulcer”(Fig. 11-1)

Figure 11-1 Rodent ulcer. (Courtesy of Dr. Noah Craft, MD, PhD.)

What is the skin cancer most likely to cause death?

Melanoma

What are the risk factors for BCC (Fig. 11-2)?

Sun exposure, fair skin, radiation therapy

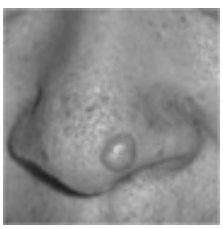


Figure 11-2BCC-pearly papule. (Courtesy of Dr. Noah Craft, MD, PhD.)

Where are BCCs most commonly found?

Sun exposed skin, i.e., head, neck, hands

How is a BCC diagnosed?

Biopsy

What is the treatment?

Excision

What is the prognosis?

Prognosis is excellent because this cancer rarely metastasizes.

What is second most common skin cancer?

Squamous cell carcinoma (SCC)

What is the precursor lesion to SCC?

Actinic keratosis (also known as solar keratosis)

What are the characteristics of an actinic keratosis on physical exam?

Red, scaly, rough patches usually found in sun-exposed area of skin

How are actinic keratoses treated?

Cryotherapy for a small number of lesions, topical 5-FU (an antimitotic agent) for large areas on face and scalp, or imiquimod cream

What are the risk factors for developing a SCC?

Sun exposure, fair skin, radiation therapy, xeroderma pigmentosa, exposure to arsenic, immunosuppression

Where are SCCs most commonly found?

Sun-exposed areas of skin, i.e., head, neck, hand

How is SCC diagnosed?

Biopsy shows “keratin pearls” in the dermis

What is the treatment?

Excision; radiation in cases where surgery is not an option

What is the prognosis?

Prognosis is very good. They metastasize more often than BCC but not as often as melanoma.

What is the type of skin cancer most likely to be found in younger age groups?

Melanoma

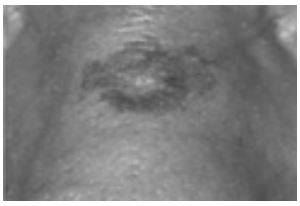


Figure 11-3 Melanoma. (Courtesy of Dr. Noah Craft, MD, PhD.)

What characteristics are most suggestive of melanoma?

Asymmetry

Borders are irregular

Colors vary

Diameter is > 6 mm (larger than a pencil eraser)

Enlarged over time (growing)

Elevation

What are the risk factors for melanoma?

Sun exposure (particularly childhood sunburn), fair skin, family history

How is melanoma diagnosed?

Excisional or incisional biopsy shows melanocytes with atypia; do not do a shave biopsy

What is the most important prognostic factor for melanoma?

Depthof invasion or thickness of melanoma; the deeper the lesion the worse the prognosis

What is Breslow classification?

Breslow classification:staging is done by measuring the depth of the lesion in millimeters

What is Clarke classification?

Clarke classification:staging is done by determining the penetration of the lesion in relation to the layers of the dermis

Which classification scheme is most predictive of survival?

Breslow classification (Fig 11-4)

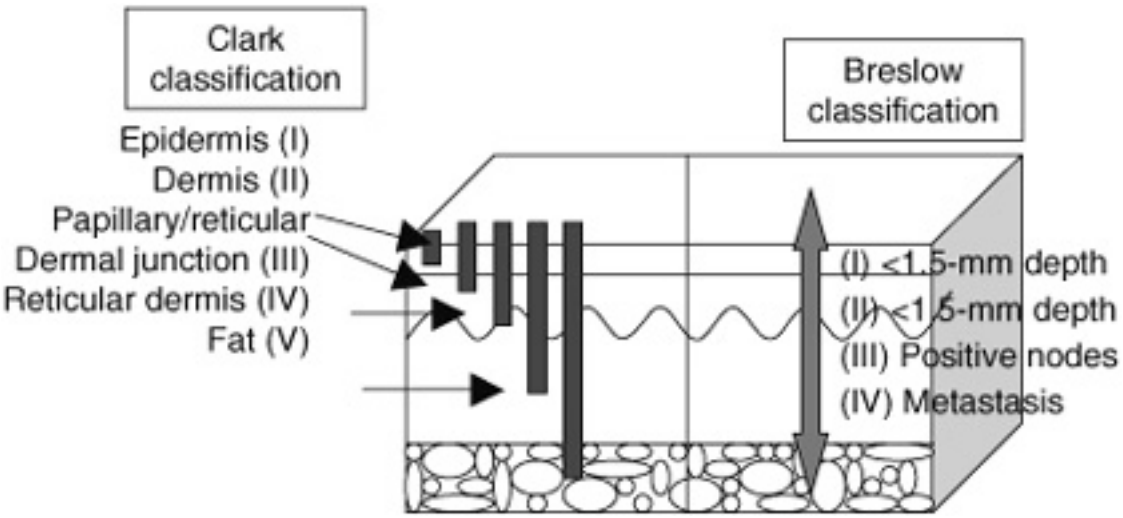


Figure 11-4 Clark and Breslow classification schemes.

Name the different types of melanoma. (Fig. 11-3)

1. Superficial spreading melanoma

2. Nodular melanoma

3. Lentigo maligna

4. Acral lentiginous

Name the type of melanoma described below:

Most common type of melanoma	Superficial spreading
Melanoma associated with worst prognosis	Lentigo maligna
Usually found on the head/neck of elderly patient	Lentigo maligna
Melanoma associated with the best prognosis	Acral lentiginous

Type of melanoma common in African Americans	Acral lentiginous
Found on palms, soles, nail beds, mucous membranes	Form of lentigo maligna that is in radial phase of growth; noninvasive
Hutchinson freckle	Acral lentiginous

What is the treatment for melanoma?

Excision; chemotherapy if metastasis is suspected

What is the type of skin cancer associated with HIV?

Kaposi sarcoma

Which herpes virus is associated with Kaposi?

Human herpes virus (HHV) 8

What are the clinical findings of Kaposi?

Red/purple macular or papular nodules on skin, mucous membranes, and viscera (especially lungs, gastrointestinal [GI] so it may present as shortness of breath)

What is the treatment?

Treat human immunodeficiency virus (HIV); treat lesions if they cause discomfort; intralesional vinblastine; radiation; chemotherapy

What is mycosis fungoides?

Cutaneous T-cell lymphoma

What is the leukemic phase of the disease called?

Sézary syndrome

What are the clinical findings of mycosis fungoides?

Chronic progressive eczema unresponsive to treatment

PSORIASIS

What do psoriatic lesions look like?

Pink plaques with silvery-white scale (Fig. 11-5)

Where are psoriatic plaques classically found?

On the elbows and knees (extensor surfaces)

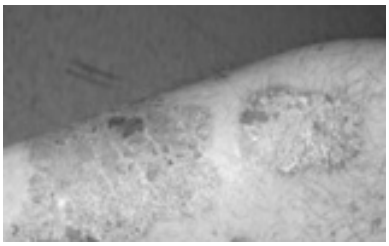


Figure 11-5 Psoriasis. (Courtesy of Dr. Noah Craft, MD, PhD.)

What other clinical findings can psoriasis be associated with?

1. Fingernail pitting

2. Oncholysis (separation of distal nail plate from nail bed)

3. Psoriatic arthritis (rheumatologic factor negative)

Which joints do psoriatic arthritis most commonly affect?

Distal interphalangeal (DIP) joints

What is Köbner phenomenon?

Psoriatic lesions that occur at the site of injury

What is Auspitz sign?

Pinpoint bleeding at sites where overlying scale is removed

How is psoriasis affected by season?

Psoriasis is**w** orse in**w** inter and better in summer because sunlight improves lesions

How is psoriasis treated?

See Table 11-1 .

Table 11-1 Management of Psoriasis

Topical (mild dose)	**Systemic** (severe dose)
Emollients	Systemic steroids
Steroids	Narrow band UV-B
Coal tar	Retinoids
Vitamin D analogs (Calcipotriene)	Methotrexate
	Cyclosporine
	Biologics

What blood tests should be done on patients taking methotrexate?

Complete blood count (CBC) to monitor for bone marrow suppression; liver function test to check for hepatotoxicity; renal function tests

What blood test should be done on patients taking cyclosporine?

Renal function tests due to the risk of nephrotoxicity

BLISTERING DISEASES

Name the blistering diseases described below:

Flaccid bullae that rupture easily Pemphigus vulgaris (PV)

Tense bullae that do not rupture easily	Bullous pemphigoid
Autoimmune blistering disorder	Both PV and bullous pemphigoid are autoimmune
Blistering disorder more likely to affect 40 to 60-year-olds	PV
Blistering disorder most likely to affect the elderly	Bullous pemphigoid
Immunofluorescence shows a "tombstone" pattern surrounding epidermal cells	PV
Immunofluorescence shows a linear band along the basement membrane	Bullous pemphigoid
Blistering disease that is more likely to be fatal	PV (PV is vulgar because it is fatal; that's why we see tombstones on biopsy)
Nikolsky sign	Pemphigus vulgaris

What is the Nikolsky sign?

Sloughing of epidermis with gentle pressure

What is the treatment for blistering diseases?

Oral steroids and antibiotics if infection

VECTOR-BORNE DISEASES

Which vector-borne illness is caused by*Rickettsia rickettsii?*

Rocky Mountain spotted fever (RMSF)

What are the symptoms?

Fever, headache, rash, myalgias, nausea, photophobia (Note:**R** ash**M** yalgias**S** evere headache**F** ever)

What kind of rash is it?

Maculopapular

How does the rash spread?

The rash spreads centrally. It starts at the wrists and ankles and spreads to the palms, soles, and trunk. (Note: The rash**WRAPS: WR** ists**A** nkles**P** alms**S** oles)

In what months is it likely to be seen?

April through September

In what regions is this illness found?

It is an illness of the western hemisphere; mainly southeastern states (North/South Carolina, Tennessee, Oklahoma); rare in the Rocky Mountains

How is RMSF diagnosed?

Usually a clinical diagnosis with a history of being outdoors or tick bite; clinical test results are slow and it is important to start treatment immediately

What is the most specific and sensitive clinical test for RMSF?

Indirect fluorescent antibody assay

What are some clinical tests to diagnose RMSF?

Serologies for*R. rickettsii* ; Weil-Felix test, biopsy showing necrotizing vasculitis

What is considered the best treatment for RMSF?

Doxycycline

How would you treat patients that are pregnant, young, or have severe illness?

Chloramphenicol

What is the major side effect of chloramphenicol to watch for?

Aplastic anemia

What vector-borne illness is caused by*Borrelia burgdorferi?*

Lyme disease

What is this transmitted by?

Ixodes deer tick

What are the symptoms?

Fever, headache, myalgias, photophobia, rash, myocarditis

What is the classic rash called and how does it spread?

Erythema chronicum migrans—erythematous annular plaques at the sites of tick bites expand with central clearing (Note: Looks like a target)

How is this rash different from that seen in RMSF?

It does not involve the palm and soles; usually rash is on trunk, extremities, axilla, inguinal regions

In what months is Lyme disease usually seen?

May through September

In what region of the United States is it mostly found?

Northeast

How is Lyme disease diagnosed?

Clinically and confirmed by polymerase chain reaction (PCR) or skin biopsy for*B. burgdorferi* (spirochete)

What is the treatment?

Penicillin, doxycycline

What are the potential complications if treatment is delayed?

Cardiac: carditis, atrioventricular (AV) block

Neurologic: meningitis, encephalitis, Bell palsy

FUNGAL INFECTIONS

Name the fungal infection described below.

Scaly, erythematous, pruritic, ring-shaped plaque with elevated borders and central clearing on the body	*Tinea corporis*
Previous symptoms found on the scalp	*Tinea capitis*
Thickened, yellow fingernails or toenails	*Onychomycosis*
Erythematous, scaly plaques with satellite pustules in intertriginous areas	*Candida*
Cottagecheese-like plaques on oral mucosa	Oral thrush
Sharply demarcated hypopigmented macules on face and trunk; more prominent in summer months	*Tinea versicolor* (also known as *Pityriasis versicolor*)

What is the causative agent of*tinea versicolor?*

*Pityrosporum ovale*also known as*Malassezia furfur*

How are these infections diagnosed?

KOH (potassium hydroxide) preparation

What is the "classic finding" on KOH preparation for*T. versicolor?*

Termed "spaghetti and meatballs"(Note: The spaghetti is the hyphae and the meatballs are the yeast)

What is seen in the KOH preparation of*Candida?*

Satellite scrapings show budding yeast and**pseudohyphae**

What is seen in KOH preparation of*T. corporis?*

Hyphae

What is the treatment for each of the following?

T. corporis	Topical antifungals (imidazoles) Systemic antifungals (griseofulvin, azoles, terbinafine) if unresponsive to topicals

BACTERIAL AND VIRAL INFECTIONS

What is the causative agent of*acne* vulgaris?

Propionibacterium acnescause inflammation of the pilosebaceous unit

What is the term used for a "blackhead?"

Open comedone

What is the term used for a "whitehead?"

Closed comedone

What are the topical treatments for acne?

Mild acne: use topicals alone Benzoyl peroxide, retinoic acid, erythromycin, or clindamycin, and antiseptics

What are the oral treatments for acne?

Use in moderate to severe cases (cystic acne)

Oral tetracyclines (doxycycline), erythromycin, clindamycin

Isotretinoin in very severe cases

What is the warning that female patients should receive*before* being placed on an isotretinoin (Accutane)?

Female patients should be put through the “I Pledge” system and be told that they should**not** become pregnant while taking this drug because it will cause severe fetal abnormalities.

What is cellulitis?

Subcutaneous, soft tissue infection with classic signs of inflammation. Area of skin is shiny and poorly demarcated and borders are not elevated

What are the classic signs of inflammation?

Red(rubor)

Hot(calor)

Painful(dolor)

Swollen(tumor)

What are the most common causative agents of cellulitis?

*Staphylococcus*and*Streptococcus*

What is the term used to describe a superficial spreading cellulitis?

Erysipelas

What is the most common causative agent?

Streptococcus pyogenes

What patients are at high risk for cellulitis?

Immunocompromised patients (Note: If diabetic with tender, erythematous rash on lower extremity,**think** cellulitis)

How is the diagnosis confirmed?

Gram stain with gram-positive cocci

How is it treated?

Penicillin or cephalosporin (cephalexin)

If penicillin- or methicillin-resistant*Staphylococcus aureus* (MRSA)-allergic, use vancomycin or clindamycin

What are the signs and symptoms of*folliculitis?*

Erythematous pustules in areas of hair growth especially in beard region

What is the most common causative agent?

S. aureus

What is the most common causative agent of “hot tub” folliculitis?

Pseudomonas

What is the treatment?

Keep area clean, if severe can use fluoroquinolone

What is a furuncle?

A collection of puss in one hair follicle

What is a carbuncle?

A collection of puss in multiple hair follicles

What is an*abscess?*

Localized collection of pus “walled off” by a cavity formed by the surrounding tissue

What is the most common causative agent?

S. aureus

What is the abnormal lab value seen?

High white blood cell (WBC) count

What is the treatment for an abscess, carbuncle, and furuncle?

Incision and drainage, Keflex may be added if needed

What is*impetigo?*

Superficial skin infection

What is the characteristic description of impetigo?

Honey-crusted lesion

What is the treatment?

Keflex, clindamycin; if MRSA, Keflex will not be effective

What are the most common causative agents?

S. aureus(children) or*S. pyogenes* (adults)

What is erythrasma?

An**eryth** ematous rash along major skin folds (e.g., axilla, groin)

In what patient population is it most commonly found?

Diabetics

What is the causative agent?

Cor*ynebacterium*

How is it diagnosed?

Under Wood lamp there is**cor** al red fluorescence; KOH preparation is negative

What is the treatment?

Erythrasma is treated with

Erythromycin

What is the term used to describe a plugged apocrine sweat gland that has become infected?

Hidradenitis suppurativa

In what regions of the body is it usually found?

Axilla and groin

What is the treatment?

Surgical debridement and antibiotics

What is the term used to describe an infection of the skin surrounding the nail plate?

Paronychia

What are the most common infective agents?

*Staphylococcus*or*Streptococcus*

What is the treatment?

Warm compress, incision and drainage (ID) if purulent, keflex if severe

What is herpes simplex?

Recurrent, painful vesicular eruptions in groups due to the herpes simplex virus (HSV) infection

Where are the lesions most commonly found?

Oral-labial region or genitals

What form of the virus is most commonly found at each of the regions above?

HSV 1—oral-labial

HSV 2—genital

(Note:**Think from top to bottom—type 1 then type 2) (**Fig. 11-6**)**

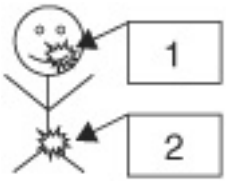


Figure 11-6

How is it diagnosed?

Tzanck smear—positive for HSV when multinucleated giant cells are seen

What is the treatment?

Acyclovir ointment reduces duration but does not prevent recurrence. Oral acyclovir

reduces frequency and recurrence.

What is herpes zoster?

Also known as shingles;

An acute, dermatomal vesicular eruption caused by the reactivation of latent varicella zoster that has been dormant in the sensory root ganglion

What is the typical history of symptoms?

Day 1: dermatomal pain (no lesions), can also present with fever, malaise

Day 3–5:**unilateral** grouped vesicles along a dermatome

Day 5–10: crust formation

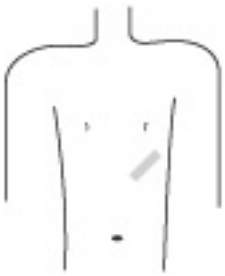


Figure 11-7

Which nerves are most commonly involved?

Thoracic nerves

What test is used to confirm the diagnosis?

Tzanck smear—multinucleated giant cells revealed (same as with herpes simplex)

What is the term used to describe herpes infection of the geniculate ganglion which leads to vesicles forming on the external auditory meatus?

Ramsay Hunt syndrome (RHS)

What can happen if RHS isn't treated rapidly?

It could extend to meningitis. It can also lead to facial paralysis and hearing loss.

What is the treatment for herpes zoster?

Oral acyclovir within 3 days of infection; immunocompromised patient: IV acyclovir

Analgesia to all patients (**it hurts**)

What are potential complications of herpes zoster?

1. Superficial infection of affected area

2.**Postherpetic neuralgia** (may last for years)

3. V1 (primary visual area) involvement can lead to corneal scarring

PIGMENTARY DISORDERS

Name the pigmentary disorders described below:

Discrete areas of hypopigmentation due to melanocyte loss	Vitiligo
Hypopigmentation due to tyrosinase deficiency—melanocytes present	Albinism
Dark hyperpigmented plaques on flexor surfaces and intertriginous areas	*Acanthosis nigricans*

What autoimmune disorder is associated with vitiligo?

Thyroid disease

What are the potential treatments for vitiligo?

Vitiligo cannot be cured because it is autoimmune in nature, but skin grafting or total depigmentation are options, also psoralen–UV-A (PUVA) and narrow band UV-B

What are the characteristics seen in albinism?

White skin and hair, red eyes, translucent iris, impaired vision with**nys** tagmus

What are albinos predisposed to?

Skin cancer

In what patient population is acanthosis nigricans seen?

Obese patients and patients with diabetes

What can acanthosis nigricans be a sign of?

It may indicate the presence of a malignancy

HYPERSENSITIVITY REACTIONS

What is Henoch-Schönlein purpura?

An IgA small vessel hypersensitivity vasculitis in which immune complexes lodge in small vessels resulting in inflammation, fibrinoid necrosis, and**palpable purpura.** Patients have a hypersensitivity reaction to antigens in immune complex.

What is seen on physical examination?

Palpable purpura,usually of the lower extremities and buttocks

Lesions may be crusted because of tissue necrosis

Patients also present with abdominal pain, pruritis, fever, and malaise

What is palpable purpura?

Nonblanchable, red papules

What patient population is Henoch-Schönlein purpura usually seen in?

Children

What are the criteria for diagnosis of a hypersensitivity vasculitis according to the American College of Rheumatology?

Three of the following must be present:

Meds taken at onset of disease

Age > 16 at onset of disease

Palpable purpura

Maculopapular rash

Eosinophils seen on biopsy

What is Henoch-Schönlein purpura associated with?

*Streptococcus*infection and penicillin use

What can it potentially progress to and why?

Rarely it progresses to glomerulonephritis because IgA deposits in glomeruli

What is the treatment?

Treat the underlying cause; systemic corticosteroids; immunosuppressives in serious cases but often self-limiting

What is erythema multiforme?

Immune complex hypersensitivity reaction to various causative agents

What are the various causes of erythema multiforme?

Half of all cases are idiopathic but other causes are:

Infections

Bacterial (*Streptococcus, Mycoplasma*)

Viral (herpes simplex, hepatitis A or B)

Fungal

Drugs: nonsteroidal anti-inflammatory drugs (NSAIDs), penicillin, sulfonamides, thiazide diuretics, barbiturates, phenytoin

Malignancy

Collagen vascular disease

What is the pathopneumonic lesion?

Erythematous**target lesions** with red center and dark outer ring in many different shapes (that's why it is called multiforme) (Fig. 11-8)

Figure 11-8 Target lesions. (Courtesy of Dr. Noah Craft, MD, PhD.)

Where are lesions mostly found?

On the palms, soles, and extremities

What forms can the lesions take?

Many forms—vesicles, papules, bullae

What is the treatment?

Treat the underlying cause. Stop any drugs causing the reaction or treat any underlying infection.

What is Stevens-Johnson syndrome?

A severe form of erythema multiforme with systemic symptoms as well as**mucous membrane** involvement (oral mucosa and conjunctiva); < 10% of body; potentially fatal (Fig. 11-9)

Figure 11-9 Stevens-Johnson syndrome. (Courtesy of Dr. Noah Craft, MD, PhD.)

What is the treatment?

Remove/treat causative agent; systemic corticosteroid therapy; treat skin lesions as burns; immune globulin intravenous (IGIV) potentially helpful

What can Stevens-Johnson syndrome progress to?

Toxic epidermal necrolysis (TEN)

How is TEN different from Stevens-Johnson?

> 30% of body surface area with full- thickness skin necrosis; higher risk of being fatal

What happens to the target lesions in TEN?

Lesions become confluent, tender, erythematous, and become bullae. There is eventual loss of the epidermis.

What is "positive" sign for TEN?

Nikolsky's sign—Gentle manual pressure leads to sloughing off of epidermis.

What is the treatment?

Remove/treat causative agent (acyclovir to prevent herpes recurrence); fluid and electrolyte replacement; systemic corticosteroids; IGIV may be helpful

What is erythema nodosum?

An inflammation of subcutaneous fat

What is the etiology?

Most cases are idiopathic

Other causes:

Drugs: oral contraceptives, sulfonamides

Infections:*Streptococcus* , TB, leprosy,*Chlamydia*

Autoimmune: inflammatory bowel disease, Behçet, sarcoidosis, rheumatic fever, pregnancy

In what patient population is it most commonly seen?

Young women between the ages of 15 and 30

What is seen on physical examination?

Erythematous nodules on lower legs(Note: Nodules are bilateral but not symmetric

Occasionally found on forearms or other areas with fat) (Fig. 11-10)

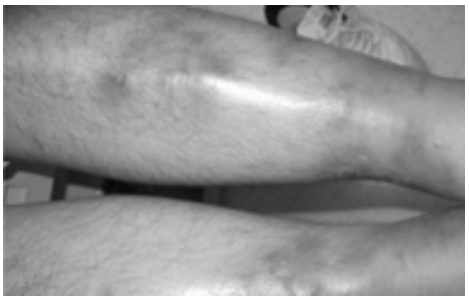


Figure 11-10 Erythema nodosum. (Courtesy of Dr. Noah Craft, MD, PhD.)

How is the diagnosis confirmed?

CBC, CXR, throat culture, antistreptolysin-O

What is the treatment?

Treat the underlying cause as well as anti-inflammatories for pain and leg elevation

What is pityriasis rosea? And what is the sequence of eruption?

A self-limiting maculopapular pruritic rash with central scale that begins as a**single herald**

patch on the trunk, then followed by a generalized rash of pink scaly patches within 2 weeks of the initial eruption. Caused by HHV 7. (Fig. 11-11)

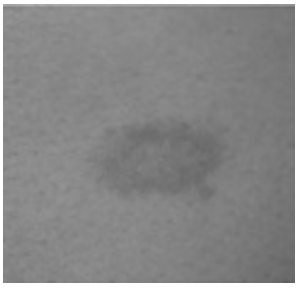


Figure 11-11 Herald patch in pityriasis rosea. (Courtesy of Dr. Noah Craft, MD, PhD.)

What is the pattern of distribution of the generalized rash?

Christmas tree patternon the back (Fig. 11-12)

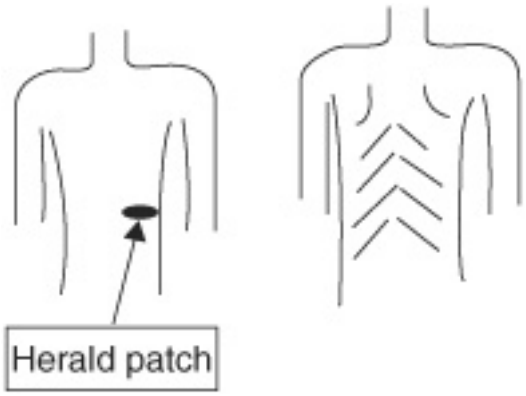


Figure 11-12 Herald patch and Christmas tree pattern.

In what season is this most commonly seen?

Spring

In what patient population does it most commonly present?

Children and young adults

What is the treatment?

Treatment is symptomatic only; it usually self-resolves in 6–8 weeks; however, sunlight helps.

Symptomatic treatment includes antihistamines, topical corticosteroids, and calamine lotion

What is scabies?

An infection by the*Sarcoptes scabiei* mite which causes an extremely pruritic papular rash. Lesions are contagious.

What should you look for on physical examination if you suspect scabies?

Burrows in webs of finger, toes, and other intertriginous areas

How is it diagnosed?

Microscopic identification of the*S. scabiei* mite in skin scraping

What is the treatment?

Permethrin 5% cream to entire body for 8 hours, then repeat 1 week later. Wash all linens. Antihistamines can help with pruritis.

How long can postscabies pruritis last after treatment?

6–8 weeks after treatment

Who should be treated?

Patient with scabies and all close contacts

CHAPTER 12

Clinical Vignettes

A 45-year-old female who recently had surgery for a thyroid cancer develops perioral paresthesias, confusion, and muscle weakness. An EKG was performed and it demonstrated a prolonged QT interval. What is the most likely reason for this woman's symptoms?

Hypocalcemia

A 30-year-old African-American female presents to your office complaining of a photosensitive skin rash over her nose and cheeks as well as fever and poly-arthritis. She reports no pain. You listen to her heart and hear a murmur. Based on the previous findings, what do you think is the most likely cause of the murmur?

Libman-Sacks endocarditis (Mnemonic: SLE causes LSE)

A 60-year-old alcoholic female presents with severe back pain with nausea and vomiting. Abdominal x-ray shows a sentinel loop. What is the most likely diagnosis?

Pancreatitis

A patient diagnosed with leukemia has Auer rods on blood smear. What type of leukemia does he have?

Acute myelogenous leukemia (AML)

A young boy presents to the dentist and is found to have excessive bleeding. Laboratory tests are performed and he is found to have a prolonged bleeding time with normal prothrombin time (PT)/partial prothrombin time (PTT). What is the diagnosis of choice?

von Willebrand disease

A 58-year-old female presents with acute renal failure of unknown etiology. Urinalysis shows Bence Jones proteinuria and she is found to have a monoclonal gammopathy. What is the diagnosis?

Multiple myeloma

A 70-year-old male in the intensive care unit (ICU) on total parenteral nutrition (TPN) for 10 days develops jaundice. Liver function test demonstrates a total bilirubin of 12. What is the most likely cause of his hyperbilirubinemia?

Cholestasis caused by parenteral nutrition

A 60-year-old female is found to be hypotensive on pressors with minimal improvement. Her chest x-ray (CXR) demonstrates an enlarged heart that resembles a water bottle. What would be the test you would order to make the diagnosis?

Echocardiogram. This patient most likely has a pericardial effusion.

A 50-year-old male who had a myocardial infarction (MI) approximately 3 weeks prior presents with fever and elevated erythrocyte sedimentation rate (ESR). What is the most likely diagnosis?

Dressler syndrome

You are called by the nurse about a hospitalized patient with a blood pressure of 100/60. You go to evaluate the patient and on examination she has distant heart sounds and jugular venous distention (JVD). You order an EKG and you notice that the height of the QRS complex varies from beat to beat. What is your diagnosis?

Cardiac tamponade

An otherwise healthy medical student gets his annual purified protein derivative (PPD) (tuberculin) as required by medical school; 48 hours later he goes to have it read and it measures 10 mm. What would you tell this student about the results?

He has a positive PPD and needs to be treated with 6–9 months of isoniazid (INH).

A 30-year-old presents with fatigue for several months. She has also had multiple urinary tract infections (UTIs) over the past year. You order a complete blood count (CBC) with a peripheral smear. The smear shows Auer rods and 52% myeloblasts. What is the diagnosis?

Acute myelocytic leukemia

A 60-year-old male presents to your office for a physical examination. He has no past medical history, does not drink or smoke and currently takes no medications. His physical examination is benign except that he appears somewhat pale. His CBC shows a hemoglobin of 11 and the mean corpuscular volume (MCV) is 70. He has a low ferritin, low serum iron, and elevated total iron-binding capacity (TIBC). What is

your next step?

Screen for colon cancer—iron deficiency anemia is colon cancer until proven otherwise.

Your 16-year-old patient comes to your office because his friends told him that he looks "yellow." He has no past medical history and is not taking any medications. He denies any recent antibiotic use. He does mention that he has felt fatigued over the past 2 days. He also says that he tried Indian food for the first time a few days ago. He had a really tasty bean dish. You order a CBC and his hemoglobin is 8. What is the diagnosis?

Glucose-6-phosphate dehydrogenase (G6PD) deficiency

An HIV patient with a CD4 count of 198 comes to see you for follow-up on his HIV. What new antibiotic would you initiate?

Bactrim as prophylaxis against*Pneumocystis carinii* pneumonia (PCP)

A 55-year-old alcoholic male is brought in to the emergency room (ER) for altered mental status. He is found to have a pulse oxygen of 85%. A stat CXR is done and the patient is found to have a large right upper lobe consolidation. He is reported to have a "currant jelly" sputum. What is the most likely organism?

*Klebsiella*secondary to an aspiration pneumonia

A 25-year-old female presents to your office complaining of diarrhea, weight loss, and heart palpitations. What initial test would you order?

Thyroid-stimulating hormone (TSH) and T4 (Think: Hyperthyroidism)

A 70-year-old male presents with renal failure. During your history and physical on

your review of systems you discover that he has been having bone pain and weight loss over the past several months. On your initial laboratory assessment you find that your patient is hypercalcemic, has rouleaux formation and has Bence Jones proteins in his urine. A serum protein electrophoresis demonstrates an "M" spike. You order an x-ray and find he has "punched out lesions." What is the most likely diagnosis?

Multiple myeloma

A patient presents to the ER with symptoms of nausea, vomiting, and fatigue. He tells you that over the past few months he has had a significant amount of weight loss. His sister, who has come to the hospital with him, says that she has noticed that recently his skin has become very tanned as well. You question the patient about recent sun exposure and he tells you that he has had very little since he is an accountant and is indoors most of the day. His laboratory tests reveal that he is hyponatremic and hyperkalemic. What diagnostic test would you order for this patient?

Plasma adrenocorticotropic hormone (ACTH) level to evaluate for Addison disease

A patient with a history of IV drug abuse presents to the hospital with high fever and chills. On physical examination you hear a new murmur. Blood cultures are drawn and are positive×2 with*Streptococcus viridans* . What is the most likely diagnosis?

Endocarditis. The tricuspid valve is most likely involved.

Unfortunately, your patient has been diagnosed with lung cancer. He has been feeling very weak and fatigued for the past few days and develops an altered level of consciousness. A CT scan was done and fortunately there are no metastases to the brain. Electrolytes show that he has a sodium of 125. His glucose is within normal limits. What test would you order next to confirm your suspected diagnosis?

Urine electrolytes to confirm the most likely diagnosis of SIADH (syndrome of

inappropriate antidiuretic hormone)

A nursing home patient who is alert and oriented presents with severe hyponatremia. Your colleague treats the patient with hypertonic saline and is able to correct his sodium within 7 hours. Subsequently, the patient becomes unresponsive and unarousable. Your colleague does not know what happened. What would you tell her was the cause of her patient's rapid alteration in mental status?

The patient has central pontine myelinolysis. Hyponatremia should never be corrected too quickly for this reason.

A patient in renal failure complains of chest pain. Her potassium is 6.5. A stat EKG shows peaked T waves. What would be the initial treatment that should be given?

Calcium gluconate to protect the heart

Your next patient in clinic is a 75-year-old white male visiting for a routine physical. He mentions that he has noticed a lesion on the ridge of his ear. You take a look at it and find it is pearly in appearance and has some telangiectasias. What is the most likely diagnosis?

Basal cell carcinoma

A sexually active 18-year-old male presents with a hot, swollen, severely painful right knee for the past 2 days. He denies any history of trauma to the joint that he can recall. What is the next step in diagnosis?

Arthrocentesis. Most likely organism is*Neisseria gonorrhoeae* .

A 77-year-old female complains of severe joint pain over the past several years. On her hands you notice some nodules on her proximal interphalangeal joints (PIP).

What are these nodules called?

Bouchard nodes

A 45-year-old woman presents to the ER complaining of dyspnea and chest pain. She just came back from a cross-country road trip. She also tells you that she had one episode of hemoptysis. The nurse takes her vitals. They are: Tm: 37.8; BP: 130/90; pulse: 110; respiratory rate: 28; and oxygen saturation of 88%. You examine the patient and find that her left calf is swollen and tender. What is the most likely diagnosis for this patient's shortness of breath?

Pulmonary embolism from a deep venous thrombosis (DVT) in her left lower extremity

A 20-year old patient presents with altered level of consciousness. His parents report that he has been very thirsty recently. A serum glucose is 849. What test could you order to differentiate between type 1 and type 2 diabetes?

C-peptide. It would be missing in type 1 diabetics.

A 45-year-old obese female presents with a 2-day history of nausea, vomiting, and abdominal pain. On examination the patient has right upper quadrant pain. You suspect cholecystitis so you order a right upper quadrant ultrasound. The test is equivocal. What is your next step in management?

This patient needs a hydroxy iminodiacetic acid (HIDA) scan

A 70-year-old male with a 35 pack per year history of smoking presents with dyspnea on exertion. The patient has a chronic dry cough and his voice sounds very hoarse. Physical examination demonstrates decreased breath sounds, a hyper-resonant chest, and distant heart sounds. A CXR reveals flattened diaphragms. What is the diagnosis?

Chronic obstructive pulmonary disease (COPD)

A 25-year-old male presents with acute right knee pain. The patient denies any history of trauma but does report fever and chills. He also tells you that over the last week he has had pain in multiple joints as well. He admits to you that he is sexually active with multiple partners and does not like to use any protection. On physical examination, the knee is swollen, erythematous, and painful. He has a rash on his palms. You tap the joint and the fluid demonstrates gram-negative diplococci. What is the diagnosis?

Gonococcal arthritis

An 84-year-old male with a past medical history significant for hypertension, hyperlipidemia, and diabetes presents with left-sided paralysis. He is admitted to the hospital for further workup. In the next 15 hours, his symptoms resolve. What is the most likely diagnosis?

Transient ischemie attack (TIA)

A 19-year-old male presents to your clinic complaining of a "rash" on his knees and elbows. He says that he has used moisturizer on it with no improvement. On physical examination, you find silvery white scaly patches on his elbows and knees. You also notice that he has pitting of some of his fingernails. What is the most likely diagnosis?

Psoriasis

A young male presents with a 3-month history of night sweats, fatigue, and 15-lb weight loss. He has noticed that he has a single, nontender cervical lymph node that does not seem to be resolving. He did mention that his symptoms seem worse with alcohol consumption. A CBC demonstrates leukocytosis. A lymph node biopsy

demonstrates binucleated giant cells (Reed-Sternberg cells). What is the diagnosis?

Hodgkin lymphoma

A patient presents with altered mental status with petechiae on the lower extremities. The patient has a temperature of 38.3°C, blood pressure of 110/80. The following are the patient's labs: CBC: WBC 10, hemoglobin 10, hematocrit 26, and platelets 50. Electrolytes demonstrates hyperkalemia and blood urea nitrogen/creatine (BUN/CR) of 40/2.5. The patient has an elevated lactate dehydrogenase (LDH) and unconjugated bilirubin. What is the diagnosis?

Thrombotic thrombocytopenic purpura (TTP)

An 18-year-old athlete presents with an erythematous, pruritic skin eruption in the intertriginous region. A KOH scraping demonstrates hyphae. What is the diagnosis?

Tinea cruris

An HIV patient presents with purple colored macules and nodules on his skin. It is caused by human herpesvirus 8 (HHV 8). What is the diagnosis?

Kaposi sarcoma

Index

Page numbers followed by *f* or *t* indicate figures or tables, respectively.

A

A-a gradient

Abdominal radiograph, in pancreatitis

Abscess

Absence seizure

ACA(anterior cerebral artery)

Acanthosis nigricans

ACE (angiotensin-converting enzyme)

ACE (angiotensin-converting enzyme) inhibitors.*See* Angiotensin-converting enzyme (ACE) inhibitors

Acetazolamide

Acetylcholinesterase inhibitors

Achalasia

Acid-base disorders

Acidosis

ACL(anterior cruciate ligament) tear

Acne vulgaris

Acquired immunodeficiency syndrome (AIDS).*See also* Human immunodeficiency virus

Acral lentiginous melanoma

Acromegaly

Actinic keratoses

Acute interstitial nephritis (AIN)

Acute lymphoblastic leukemia (ALL)

Acute myelocytic leukemia

Acute myelogenous leukemia (AML)

Acute renal failure

Acute respiratory distress syndrome (ARDS)

Acute tubular necrosis (ATN)

Acyclovir

Addison disease

Adenocarcinoma

esophageal

Albumin

Albuminocytologic dissociation

Aldosterone

Alkalosis

ALL(acute lymphoblastic leukemia)

Allopurinol

Alpha-1-antitrypsin deficiency

Alpha-blockers

Alpha-thalassemia

Aluminum hydroxide

Alzheimer dementia

Amantadine

Amaurosis fugax

American Burkitt lymphoma

Aminocaproic acid

Aminoglycosides

for endocarditis

for pneumonia

AML(acute myelogenous leukemia)

Ammonium magnesium phosphate stones

Amoxicillin

Amphotericin B

Ampicillin

Amyloid plaques

Amyotrophic lateral sclerosis (ALS)

Anaplastic carcinoma, thyroid

Anemia

of chronic disease

etiologies

macrocytic

microcytic

Anterior cruciate ligament (ACL) tear

Anterior inferior cerebellar artery

Anterior pituitary

Anterior posterior (AP) film

Anti-ds-DNA

Anti-La antibody

Anti-Ro antibody

Anti-Scl-70 antibody

Anti-seizure medications

Anti-SM antibody

Antibiotics

for endocarditis

in HIV/AIDS

for meningitis

for pneumonia

for syphilis

for ulcerative colitis

Anticentromere antibody

Anticholinesterase inhibitors

Antidiuretic hormone (ADH)

Antinuclear antibody (ANA)

Antiretroviral therapy

Aortic regurgitation

Aortic stenosis

AP (anterior posterior) film

Aphasia

Aplastic anemia

Apolipoprotein E

Apraxia

ARDS (acute respiratory distress syndrome)

Arrhythmias

Arterial blood gases

in asthma attack

in emphysema

normal values

Arteriovenous fistula

Arthropathies

Ascending cholangitis

Ascites

Aspergillus

Aspirin, after transient ischemic attack

Asterixis

Asthma

Astrocytoma

Atelectasis

ATN (acute tubular necrosis)

Atrial fibrillation

Atrial flutter

Atrioventricular malformation

Auerbach plexus

Auspitz sign

Axis, of EKG

Azathioprine

Azithromycin

Azotemia

B

Bacterial vaginosis

Bactrim

for bacterial diarrhea

for pneumonia

for urinary tract infection

Bamboo spine

Barrett esophagus

Basal cell carcinoma (BCC)

Basal energy expenditure

Beck’s triad

Behçet syndrome

Bence Jones proteins

Benign positional vertigo

Benign prostatic hyperplasia (BPH)

Benzodiazepines, in status epilepticus

Benztropine

Berger disease

Berry aneurysm

Beta-agonists

Beta-blockers

for congestive heart failure

contraindications

for dilated cardiomyopathy

for hypertension

in hyperthyroidism

preoperative, in pheochromocytoma

Beta-lactam antibiotics

Beta-thalassemia

Bile acid sequestrants

Biliary tract, radiographic study

“Bird’s beak” appearance

on barium enema

on barium swallow

Bisphosphonates

in hyperparathyroidism

for osteoporosis

Blackhead

Blast crisis

Bleomycin

Blistering diseases

Blood products

Blood transfusions

Blood types

Blood urea nitrogen (BUN), in gastrointestinal bleeding

Bloody diarrhea

Blumer shelf

Bone marrow transplant

for acute lymphoblastic leukemia

for chronic myelogenous leukemia

Borrelia burgdorferi

Bouchard nodes

Boutonniere deformity

BPH (benign prostatic hyperplasia)

Bradykinesia

Brain tumors

Breslow classification, melanoma

Broca aphasia

Bromocriptine

Bronchiectasis

Bronchodilators, for asthma exacerbation

Brudzinski sign

Buffalo hump

Bullae

Bullous pemphigoid

Burkitt lymphoma

Busulfan

C

CABG (coronary artery bypass graft)

esophageal

in HIV/AIDS

oral

vaginal

Captopril stimulation test

Carbamazepine

Carbidopa

Carbohydrates

Carbon monoxide poisoning

Carbuncle

Carcinoembryonic antigen (CEA)

Carcinoid tumor

Cardiac enzymes

Cardiac tamponade

Cardiomyopathy

in multiple sclerosis

Cerebrovascular accident (CVA)

Charcot triad

Chest x-ray (CXR)

in congestive heart failure

in emphysema

evaluation

parts

in pneumonia

positions

in pulmonary embolism

in sarcoidosis

in tuberculosis

Chlamydia

Chlamydia psittaci

Chlamydia trachomatis

Chloramphenicol

Cholangitis

Cholecystitis

Choledocholithiasis

Cholelithiasis

Cholera

Cholestasis

Chronic bronchitis

Chronic lymphocytic leukemia (CLL)

Chronic myelogenous leukemia (CML)

Chronic obstructive pulmonary disease (COPD)

Chronic renal failure

Churg-Strauss disease

Churg-Strauss syndrome

Chvostek sign

Ciprofloxacin, for bacterial diarrhea

Cirrhosis

CK-MB (creatine kinase-MB)

Clarithromycin

Clarke classification, melanoma

Clindamycin

*Clostridium difficile*infection and

for pneumonia

Clopidogrel (Plavix)

Clostridium difficile

Cluster headache

Coagulopathies

Coccidioides immitis

Coffee ground emesis

Cognitive disorders

Colchicine

Cold autoimmune hemolytic anemia

Colicovesicular fistula

Colon cancer

Comedone

Complete blood count (CBC)

components

in gastrointestinal bleeding

in peptic ulcer

in pneumonia

Complex partial seizure

Computed tomography (CT)

in Alzheimer dementia

in diverticulitis

in epidural hematoma

in pancreatitis

Coxiella burnetii

Creatine kinase (CPK)

Creatine kinase-MB (CK-MB)

Creatinine clearance

CREST syndrome

Crohn disease

Cromolyn sulfate

Cryoprecipitate

Crypt abscess

Cryptococcus

CSF.*See* Cerebrospinal fluid

CT.*See* Computed tomography

Cullen sign

Curling ulcer

Cushing disease

Cushing syndrome

Cushing ulcer

CVA(cerebrovascular accident)

CXR.*See* Chest x-ray

Cyclophosphamide, for polymyositis and dermatomyositis

Cyclosporine

adverse effects

for psoriasis

for ulcerative colitis

Cytomegalovirus

D

D-dimer test

D5W

D10W

Dawn phenomenon

de Quervain thyroiditis

hypertension treatment in

sinusitis in

treatment

type 1

type 2

Diabetes insipidus

Diabetic ketoacidosis (DKA)

Dialysis

for chronic renal failure

for electrolyte disturbances

Diarrhea

Diastolic murmur

Diets

Diffuse esophageal spasm

Diflucan

Digitalis, for congestive heart failure

Digoxin

for congestive heart failure

contraindications

Digoxin toxicity

Dilated cardiomyopathy

Disseminated intravascular coagulation (DIC)

Diuretics

for congestive heart failure

contraindications

for dilated cardiomyopathy

for hypertension

Diverticula

Diverticulitis

Diverticulosis

Dix-Hallpike maneuver

Dobutamine

Donepezil

Dopamine

Doxycycline

for*Chlamydia*

for pneumonia

for Rocky Mountain spotted fever

for Sjögren’s syndrome

for syphilis

Dressler syndrome

Drug-induced lupus

Duke criteria, endocarditis

Duodenal ulcer

DVT (deep vein thrombosis)

Dysarthria

Dysphagia

E

in myocarditis

parts

in pericarditis

in pulmonary embolism

in pulsus paradoxus

in ventricular fibrillation

Electroencephalogram (EEG)

Electrolyte imbalances

hypercalcemia

hyperkalemia

hypermagnesemia

hypernatremia

hyperphosphatemia

hypocalcemia

hypokalemia

hypomagnesemia

hyponatremia

hypophosphatemia

Emphysema

Endocarditis

causes

clinical vignette

definition

diagnosis

symptoms

treatment

Entamoeba histolytica

Enterobacter

Enterococcus

Enteroviruses

Enzyme immunoassay (EIA)

in cholangitis

in diarrhea

hemolytic uremic syndrome and

in neonatal meningitis

in spontaneous bacterial peritonitis

in urinary tract infections

Esophageal candidiasis

Esophageal disorders

Esophageal varices

Essential thrombocytosis

Ethambutol

Ethosuximide

Etidronate

Extracellular fluid

Extrapulmonary tuberculosis

Exudative pleural effusion

F

Factitious hyponatremia

Factor VIII concentrate

Fat

Fat soluble vitamins

Fibrates

Fistula

arteriovenous

colicovesicular

Fitz-Hugh-Curtis syndrome

Fluconazole

Fluid-restricted diet

Fluids

Fluoroquinolones, for pneumonia

Focal segmental glomerulosclerosis

Focal seizure

Folate deficiency

Follicle-stimulating hormone (FSH)

Follicular cancer, thyroid

Folliculitis

Foscarnet

Fractional sodium excretion (FENa)

Fractures, in osteoporosis

Francisella tularensis

Fresh frozen plasma (FFP)

FTA-ABS/MHA-TP

Fungal infections

Furosemide

Furuncle

G

Glipizide

Glomerular filtration rate

Glomerulonephritis

Glomerulonephropathies

Glucocorticoids.*See* Corticosteroids

Glucose-6-phosphate dehydrogenase (G6PD) deficiency

Glyburide

Gold compounds

Gonococcal arthritis

Gonorrhea

Goodpasture disease

Goodpasture syndrome

Gottron papules

Gout

Graves disease

Grey Turner sign

Group Astreptococcal pharyngitis

Group B*Streptococcus* , in meningitis

Growth hormone (GH)

Guillain-Barré syndrome

Gummas

H

H2 blockers

HACEK organisms

Haemophilus influenzae

in bronchiectasis

in meningitis

in pneumonia

in sinusitis

Hairy cell leukemia

Hampton hump

Hashimoto thyroiditis

Headache

in brain tumor

types

Heart block

Heart murmurs

Heberden nodules

Heinz bodies

Helicobacter pylori

Hemodialysis

Hemolytic anemia

Hemolytic uremic syndrome (HUS)

Hemophilia

Hemoptysis

in bronchiectasis

causes

treatment

Henoch-Schonlein purpura

Heparin

coagulation and

for pulmonary embolism

Hepatic encephalopathy

Hepatitis

Hepatobiliary iminodiacetic acid (HIDA) scan

Hepatocellular carcinoma

Hepatorenal syndrome

Herpes simplex virus (HSV)

Herpes zoster (shingles)

Hgb A1c

Hidradenitis suppurativa

High-density lipoprotein (HDL)

Hutchinson freckle

Hydralazine

Hydroxychloroquine

Hyperaldosteronism

Hypercalcemia

Hypercoagulable states

Hypercortisolism

Hyperkalemia

Hyperlipidemia

Hypermagnesemia

Hyperosmolar hyperglycemic nonketotic coma (HHNK)

Hyperparathyroidism

Hyperphosphatemia

Hyperpigmentation

Hypersensitivity reactions

Hypertension

causes

definition

treatment

treatment in stroke

Hypertensive emergency

Hypertensive urgency

Hyperthyroidism

Hypertrophic cardiomyopathy

Hypervolemia

Hypoalbuminemia

Hypocalcemia

Hypoglycemic agents

Hypokalemia

Hypomagnesemia

Hyponatremia

I

Inflammatory bowel disease

Infliximab, for Crohn disease

Insulin

Insulin glargine

Insulin lispro

Interferon

for hairy cell leukemia

for hepatitis C

International normalized ratio (INR)

Interstitial lung disease

Intracellular fluid

Intracranial bleeding

Intravenous fluids

Intrinsic factor

Ipratropium, for asthma exacerbation

Iron deficiency anemia

Isoniazid (INH)

Isosorbide, for congestive heart failure

Isotretinoin

Ixodes deer tick

J

Janeway lesions

Jones criteria, rheumatic heart disease

K

Kaposi sarcoma

Kayser-Fleisher ring

Kernig sign

Ketoconazole

Kidney stones

Klebsiella

in pneumonia

in urinary tract infections

Koebner phenomenon

Krukenberg tumor

KUB (kidney, ureter, bladder) radiograph

Kussmaul hyperpnea

L

Lactase deficiency

Lactated ringers

Lacunar infarct

Lambert-Eaton syndrome

Legionella

Lentigo maligna

Leukemias

Leukotriene inhibitors

Levodopa

M

Malignant hypertension

Mallory-Weiss tear

Manometry

Marantic endocarditis

Mean arterial pressure (MAP)

Meclizine

Medullary cancer, thyroid

Medulloblastoma

Melanoma

Membranoproliferative glomerulonephritis

Membranous glomerulonephritis

MEN (multiple endocrine neoplasia)

Ménétrier disease

Meniere disease

Meningioma

for bacterial vaginosis

for*Clostridium difficile* infection

for parasitic diarrhea

for pneumonia

for*Trichomonas*

Microcytic anemias

Middle cerebral artery (MCA)

Migraine headache

Minimal change disease

Mitral regurgitation

Mitral stenosis

Mitral valve prolapse

Moraxella catarrhalis

Mucor

Mucormycosis

Multifocal atrial tachycardia (MAT)

Multiple endocrine neoplasia (MEN)

Multiple myeloma

Multiple sclerosis

Murmurs

Murphy sign

Myasthenia gravis

*Mycobacterium avium*complex (MAC)

Mycoplasma

Mycosis fungoides

Myelofibrosis

Myeloproliferative diseases

Myocardial infarction (MI)

complications

diagnosis

symptoms

treatment

Myocarditis

Myxedema, pretibial

Myxedema coma

N

Neisseria gonorrhoeae

Neisseria meningitidis

Nephritic syndrome

Nephrolithiasis

Nephrotic syndrome

Neural tube defects

Neurofibrillary tangles

Neuropathy, in diabetes

Nicotinic acid

Nikolsky sign

Nitroglycerin

Nitroprusside

Nocardia

Nodule

Non-Hodgkin lymphoma

Nonsmall cell lung cancer

Nonsteroidal anti-inflammatory drugs (NSAIDs)

gastritis and

for gout

peptic ulcers and

for rheumatoid arthritis

for systemic lupus erythematosus

Normal saline (NS)

Normocytic anemias

Norwalk virus

NPH insulin

Nutrition

Nystagmus

Nystatin cream

O

Odynophagia

Oliguria

Onychomycosis

Ophthalmic artery

Ophthalmopathy, infiltrative

Opportunistic infections, in HIV/AIDS

Oral thrush

Osler nodes

Osmolality, serum

Osteoarthritis

Osteomalacia

Osteomyelitis

Osteoporosis

Oxytocin

P

P wave

PA(posterior anterior) film

Packed red blood cells (PRBCs)

Paget disease of bone

Pamidronate

PAN (polyarteritis nodosa)

Panacinar emphysema

Pancoast syndrome

Pancreatitis

Pansystolic murmur

Pap smears

Papillary cancer, thyroid

Papillary muscle rupture

Papule

Paracentesis

Paraneoplastic syndromes

Parathyroid disorders

Parathyroidectomy

Parkinson’s disease

Paronychia

Partial parenteral nutrition (PPN)

Partial prothrombin time (PPT)

Partial thromboplastin time (PTT)

Pelvic inflammatory disease (PID)

Pemphigus vulgaris

Penicillamine

Penicillin

Peptic ulcers

Pericardial effusion

Pericarditis

Peritoneal dialysis

Peritonitis

in peritoneal dialysis

spontaneous bacterial

Pernicious anemia

Petechiae

Phenobarbital

Phenytoin

Pheochromocytoma

Philadelphia chromosome

Phosphate

Pica

Pigmentary disorders

Pioglitazone

Piperacillin tazobactam

Pituitary gland

disorders

hormones

Pityriasis rosea

Pityrosporum ovale

Plaque

Platelet disorders

Platelet levels

Platelet transfusions

Plavix. (clopidogrel)

Pleural effusion

Plicamycin

Plummer disease

Portal hypertension

Postrenal acute renal failure

Posterior anterior (PA) film

Posterior cerebral artery (PCA)

Posterior inferior cerebellar artery

Posterior pituitary

Postherpetic neuralgia

Postnasal drip

Poststreptococcal glomerulonephritis

Potassium

Potassium-sparing diuretics

Pott disease

PPD (purified protein derivative)

Prealbumin

Pregnancy, folate deficiency in

Premature ventricular contraction (PVC)

Prerenal acute renal failure

Pretibial myxedema

Prinzmetal angina

Procainamide

Prolactin

Prolactinoma

Propionibacterium acnes

Propythiouracil (PTU)

Protein

Protein-restricted diet

Proteinuria

Proteus

Prothrombin time (PT)

Proton pump inhibitors

Pseudogout

Pseudohyperkalemia

Pseudohypocalcemia

Pseudohyponatremia

Pseudomembranous colitis

Pseudomonas

in bronchiectasis

in folliculitis

in osteomyelitis

in pneumonia

Psoriasis

Psoriatic arthritis

Pulmonary angiography

Pulmonary embolism

diagnosis

EKG findings

risk factors

signs and symptoms

treatment

Pulmonary stenosis

Pulsus paradoxus

Purified protein derivative (PPD)

Purpura

Pustule

Pyelonephritis

Pyrazinamide

Pyridostigmine

Pyrimethamine

Q

QRS complex

R

anemia in

chronic

diet

Respiratory acidosis

Respiratory alkalosis

Restrictive cardiomyopathy

Restrictive lung disease

Reversible ischemic neurologic defect (RIND)

Reynold pentad

Rheumatic fever

Rheumatic heart disease

Rheumatoid arthritis

Ribavirin

Rickets

Rickettsia rickettsii

Rifampin

Rocky Mountain spotted fever

Rosiglitazone

Rotavirus

Roth spots

S

S1sound

S2sound

Salmonella

in diarrhea

in osteomyelitis

Sarcoidosis

Sarcoptes scabiei

Scabies

Schatzki ring

Schistosomiasis

Schwannoma

Scleroderma

Sclerosing cholangitis

Scrofula

Seizure disorders

Selective estrogen receptor modulators

Selegiline

Sentinel loop

Sepsis

Septic shock

Serum osmolality

Sexually transmitted diseases

Sézary syndrome

Shawl sign

Sheehan syndrome

Shigella

Shingles (herpes zoster)

SIADH (syndrome of inappropriate diuretic hormone)

Sickle cell anemia

Sideroblastic anemia

Simple partial seizure

Sinemet

Sinus tachycardia, in pulmonary embolism

Sinusitis

Sipple syndrome

SIRS (systemic inflammatory response syndrome)

Sister Mary Joseph sign

Sjögren syndrome

Skin cancers

Skin lesions

SLE (systemic lupus erythematosus)

Small bowel obstruction

Small cell lung cancer

Sodium

Somogyi effect

Spironolactone

for ascites

for congestive heart failure

Splenectomy, for hairy cell leukemia

Splinter hemorrhages

Spontaneous bacterial peritonitis

Squamous cell carcinoma (SCC)

lung

skin

Staphylococcus aureus

in abscess

in bronchiectasis

in cellulitis

in endocarditis

in folliculitis

in impetigo

methicillin-resistant

in osteomyelitis

in pneumonia

Staphylococcus saprophyticus

Statins

Status epilepticus

Stevens-Johnson syndrome

Streptococcus pneumoniae

in endocarditis

in meningitis

in pneumonia

in sinusitis

Streptococcus pyogenes

Streptococcus viridans

Streptokinase

Stroke (cerebrovascular accident)

Struvite stones

Subacute thyroiditis

Subarachnoid hemorrhage

Subdural hematoma

Sulfadiazine

Sulfasalazine

Sulfonylureas

Superior vena cava syndrome

Swan neck deformity

Syndrome

Syndrome of inappropriate diuretic hormone (SIADH)

Syphilis

Systemic inflammatory response syndrome (SIRS)

Systemic lupus erythematosus (SLE)

Systolic ejection murmur

T

Tabes dorsalis

Tacrine

Takayasu arteritis

Tamoxifen

Telangiectasia

Temporal arteritis

Tensilon test

Tension headache

Tension pneumothorax

Tetracycline

for acne vulgaris

for syphilis

Thalassemias

Thayer-Martin medium

Thiazide diuretics

Thiazolidinediones

Thrombocytopenia

Thrombocytosis, essential

Thrombolysis

Thrombotic thrombocytopenic purpura (TTP)

Thyroid cancer

Thyroid disorders

Thyroid nodule

Thyroid-stimulating hormone (TSH)

Thyroid storm

Ticlopidine

Tinea capitis

Tinea corporis

Tinea cruris

Tinea versicolor

Tissue-type plasminogen activator (tPA)

contraindications

in pulmonary embolism

after embolic stroke

Todd paralysis

Tonic-clonic seizure

Tophi

Torsades de pointes

Total parenteral nutrition (TPN)

Toxic epidermal necrolysis (TEN)

Toxic megacolon

Toxoplasmosis

Tram track lung markings

Transient ischemic attack (TIA)

Transudative pleural effusion

Treponema pallidum

Trichomonas

Tricuspid regurgitation

Tricuspid stenosis

Trihexyphenidyl

Tropical sprue

Troponin

Trousseau sign

TTP (thrombotic thrombocytopenic purpura)

Tuberculosis

Type 1 diabetes.*See also* Diabetes

Type 2 diabetes.*See also* Diabetes

Type and cross

Type and screen

Tzanck smear

U

Ulcerative colitis

Ulcerative proctitis

Universal donor

Universal recipient

Unstable angina

Upper motor neuron signs

Uremia

Uremic syndrome

Uric acid stones

Urinary cholesterol

Urinary tract infections (UTIs)

Urine

V

V/Q mismatch

V/Q scan

Vaginitis

Valproic acid

Valvular heart diseases

Vancomycin

for*Clostridium difficile* infection

for meningitis

Vasculitis

Vasopressin

VDRL/rapid plasma reagin (RPR)

Vector-borne diseases

Ventricular fibrillation

Ventricular hypertrophy

Ventricular tachycardia (VT)

Vertical nystagmus

Vertigo

Vesicle

Viral labyrinthitis

Virchow node

Vitamin Adeficiency

Vitamin B12

absorption

deficiency

Vitamin C deficiency

Vitamin K

Vitamins

Vitiligo

Volume status, assessment

Volvulus

von Willebrand disease

von Willebrand factor (VWF) deficiency

W

Warfarin

coagulation and

fresh frozen plasma and

for pulmonary embolism

reversal

Water deficit

Waterhouse-Friderichsen syndrome

Watershed infarct

Wegener granulomatosis

Weil-Felix test

Wermer syndrome

Wernicke aphasia

Westmark sign

Wheal

Whiff test

Whipple disease

Whitehead

Wilson disease

Winter formula

Wolff-Parkinson-White (WPW) syndrome

Y

Yersinia

Z

Zenker diverticulum

Zinc deficiency

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