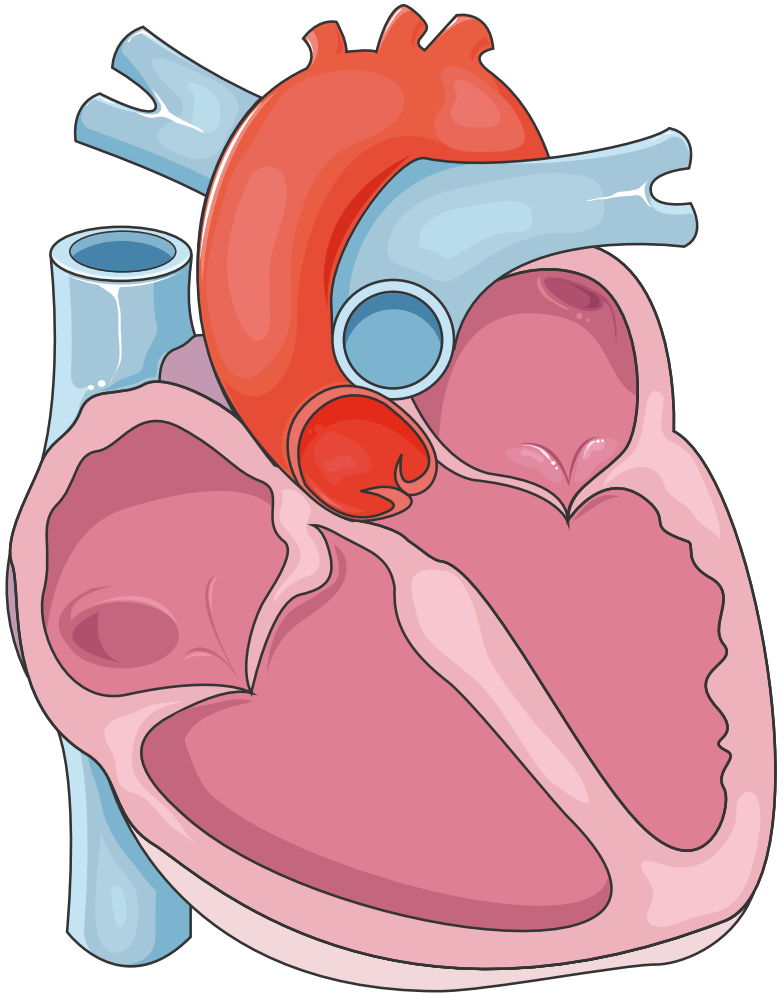
RISH ACADEMY's

Clinical Medicine

Flashcards



By T. Rishad

Rish Academy
Sri Lanka
www.rishacademy.com

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Dedication

To my family and friends, thank you for your love and support.

- Cardiovascular System Disorders
- Respiratory System Disorders
- Nervous System Disorders
- Endocrine System Disorders
- Gastrointestinal System Disorders
- Immune System Disorders
- Urologic System Disorders
- Women’s Health and Perinatal Disorders
- Dermatologic System Disorders
- Mental Health Disorders
- Musculoskeletal System Disorders
- Sensory System Disorders

AAA	abdominal aortic aneurysm
ABG	arterial blood gases
ABI	ankle-brachial index
ac	before meals
ACE	angiotensin-converting enzyme
AChE	acetylcholinesterase
AChR	acetylcholine receptor
ACLS	advanced cardiac life support
ACTH	adrenocorticotropic hormone
ADH	antidiuretic hormone
ADHD	attention-deficit/hyperactivity disorder
ADLs	activities of daily living
AED	antiepileptic drug
AF	atrial fibrillation
AFB	acid-fast bacillus
AGC	atypical glandular cells
AIDS	acquired immunodeficiency syndrome
ALL	acute lymphocytic leukemia
ALP	alkaline phosphatase
ALS	amyotrophic lateral sclerosis
ALT	alanine aminotransferase
AMI	acute myocardial infarction
AML	acute myelogenous (myeloblastic) leukemia
ANA	antinuclear antibody
ANP	atrial natriuretic peptide
anti-CCP	anticyclic citrullinated peptide
APAP	acetaminophen
aPTT	activated partial thromboplastin
ARDS	acute respiratory distress syndrome
AS	aortic stenosis
ASA	acetylsalicylic acid
ASC	atypical squamous cells
ASCA	anti–Saccharomyces cerevisiae antibody
ASC-US	ASC of undetermined significance
AST	aspartate aminotransferase
AV	atrioventricular
AVM	arteriovenous malformation
AVP	arginine vasopressin
BBB	bundle branch block
BCG	bacille Calmette-Guérin
BCP	birth control pills
BD	Buerger's disease
BHS	beta-hemolytic streptococci
bid	two times a day
BMI	body mass index
BMS	bone marrow suppression
BMT	bone marrow transplant
BNP	brain natriuretic peptide
BP	blood pressure
BPH	benign prostatic hyperplasia
bpm	beats per minute
BRM	biologic response modifier
BROW	barley, rye, oats, and wheat
BSA	body surface area
BSE	breast self-examination
BUN	blood urea nitrogen
BUN	blood urea nitrogen
BX	biopsy
C&S	culture and sensitivity
CA	coronary artery
Ca+	serum calcium
Ca++	calcium
CABG	cardiac artery bypass graft
CAD	coronary artery disease

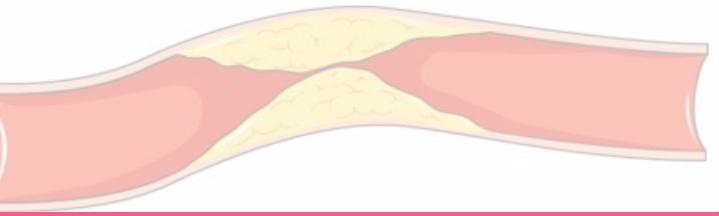
cap	*capsule*
CBC	*complete blood count*
CBI	*continuous bladder irrigation*
CBT	*cognitive behavioral therapy*
CD4	*T-helper cells*
CD8	*cytotoxic cells*
CEA	*carcinoembryonic antigen*
CFTR	*cystic fibrosis transmembrane regulator*
CHF	*congestive heart failure*
CIN	*cervical intraepithelial neoplasia*
CK	*creatine kinase*
CK-MB	*serum creatine kinase, myocardial bound*
CLL	*chronic lymphocytic leukemia*
CML	*chronic myelogenous leukemia*
CNS	*central nervous system*
CO	*cardiac output*
COMT	*catechol-O-methyltransferase*
COPD	*chronic obstructive pulmonary disease*
COX-2	*cyclooxygenase 2 inhibitors*
CPHSS	*Cincinnati Prehospital Stroke Scale*
CPK	*creatine phosphokinase*
CPM	*continuous passive motion*
CPR	*cardiopulmonary resuscitation*
CR	*controlled release*
CREST	*calcinosis, Raynaud's phenomenon, esophageal dysfunction, sclerodactyly, telangiectasia (cluster of features of systemic sclerosis scleroderma)*
CRP	*c. reactive protein*
CRS-R	*Conners Rating Scales–Revised*
CS	*cardiogenic shock*
CS	*cesaerean section*
CSF	*cerebrospinal fluid*
CSF	*colony-stimulating factor*
CT	*computerized tomography*
CV	*cardiovascular*
CVA	*cardiovascular accident*
CVC	*central venous catheter*
CVP	*central venous pressure*
CXR	*chest x-ray*
D5/0.9	*5% dextrose and normal NaCl saline solution (0.9% NaCl)*
D5/1/2/NS	*5% dextrose and half normal saline solution (0.45% NaCl)*
D5W	*5% dextrose in water*
DBP	*diastolic blood pressure*
Derm	*dermatology*
DEXA	*dual-energy x-ray absorptiometry*
DFV	*Doppler flow velocimetry*
DHT	*dihydrotestosterone*
DI	*diabetes insipidus*
DIC	*disseminated intravascular coagulation*
DISIDA	*diisopropyl iminodiacetic (scan) acid (cholescintigraphy)*
DJD	*degenerative joint disease*
DKA	*diabetic ketoacidosis*
	dL deciliter
DMARD	*disease-modulating antirheumatic drug*

DNA	*deoxyribonucleic acid*
DRE	*digital rectal examination*
DSM-IV-TR	*Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision*
DTR	*deep tendon reflexes*
DTs	*delirium tremens*
DVT	*deep vein thrombosis*
ECG	*electrocardiogram*
ECHO	*echocardiography*
ECMO	*extracorporeal membrane oxygenation*
ECT	*electroconvulsive therapy*
EEG	*electroencephalogram*
EENT	*eye, ear, nose, and throat*
EF	*ejection fraction*
EGD	*esophagogastroduodenoscopy*
ELISA	*enzyme-linked immunosorbent assay*
EMA-IgA	*immunoglobulin A antiendomysial*
EMG	*electromyography*
EMS	*emergency medical services*
Endo	*endocrine*
EP	*extrapyramidal*
EPS	*extrapyramidal symptoms*
ER	*extended-release*
ERCP	*endoscopic retrograde cholangiopancreatography*
ESR	*erythrocyte sedimentation rate*
ESRD	*end-stage renal disease*
ESWL	*extracorporeal shock wave lithotripsy*
ET-1	*endothelin-1*
ETOH	*ethal alcohol*
F and E	*fluid and electrolyte*
FAP	*familial adenomatous polyposis*
FBS	*fasting blood sugar*
FDA	*U.S. Food and Drug Administration*
FFP	*fresh frozen plasma*
FHT	*fetal heart tone*
FISH	*luorescence in situ hybridization*
G, g, gm	*gram*
GABA	*gamma-aminobutyric acid*
GABAB	*gamma-aminobutyric acid type B*
GABRB3	*GABAA receptor gene*
GB	*Guillain-Barré*
GERD	*gastroesophageal reflux disease*
GFR	*glomerular filtration rate*
GGT	*gamma-glutamyl transferase*
GH	*growth hormone*
GI	*gastrointestinal*
GnRH	*gonadotropin-releasing hormone*
GTT	*glucose tolerance test*
GU	*genitourinary*
GVHD	*graft-versus-host disease*
H&H	*hematocrit and hemoglobin*
H1N1	*hemagglutinin type 1 and neuraminidase type 1*
H2	*histamine 2*
H5N1	*hemagglutinin type 5 and neuraminidase type 1*
HAART	*highly active antiretroviral therapy*

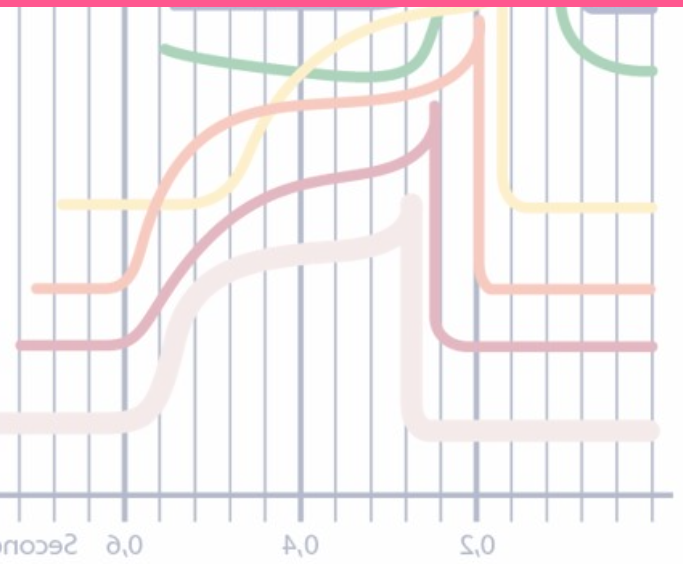
HAV	hepatitis A
HBV	hepatitis B
HCP	health-care professional
Hct	hematocrit
HCV	hepatitis C
HDL	high-density lipoproteins
HDV	hepatitis D
HELLP	hemolysis, elevated liver enzymes, low platelets
HEPA	high-efficiency particulate air
HER2	human EGF (epidermal growth factor) receptor 2
HEV	hepatitis E
Hgb	hemoglobin
HGSIL	high-grade squamous intraepithelial lesion
HIDA	hepatobiliary iminodiacetic (scan) acid (cholescintigraphy)
HIV	human immunodeficiency virus
HLA	human leukocyte antigen
HOB	head of bed
HPV	human papillomavirus
HR	heart rate
HRT	hormone replacement therapy
HTN	hypertension
HSIL	high-grade squamous intraepithelial lesion
HSV	herpes simplex virus
I&O	intake and output
ICD	implantable cardioverter defibrillator
ICP	intracranial pressure
ICS	intercostal space
IDM	infants of diabetic mothers
IgE	immunoglobulin E
IgG	immunoglobulin G
IL-1	interleukin 1
IL-8	interleukin 8
INR	international normalized ratio
IOL	intraocular lens
IOP	intraocular pressure
IVP	intravenous pyelogram
JNC 7	The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure
K+	potassium
KOH	potassium hydroxide
KS	Karposi's sarcoma
KUB	kidney-ureter-bladder
LDH	lactate dehydrogenase
LDL	low-density lipoprotein
LEEP	loop electrosurgical excision procedure
LFT	liver function tests
LLQ	left lower quadrant
LOC	level of consciousness
LP	lumbar puncture
LR	lactated Ringer's (solution)
LSIL	low-grade squamous intraepithelial lesion
LVAD	left ventricular assist device
MAO-B	monoamine oxidase-B
MELD	Model for End-Stage Liver Disease
MG	myasthenia gravis

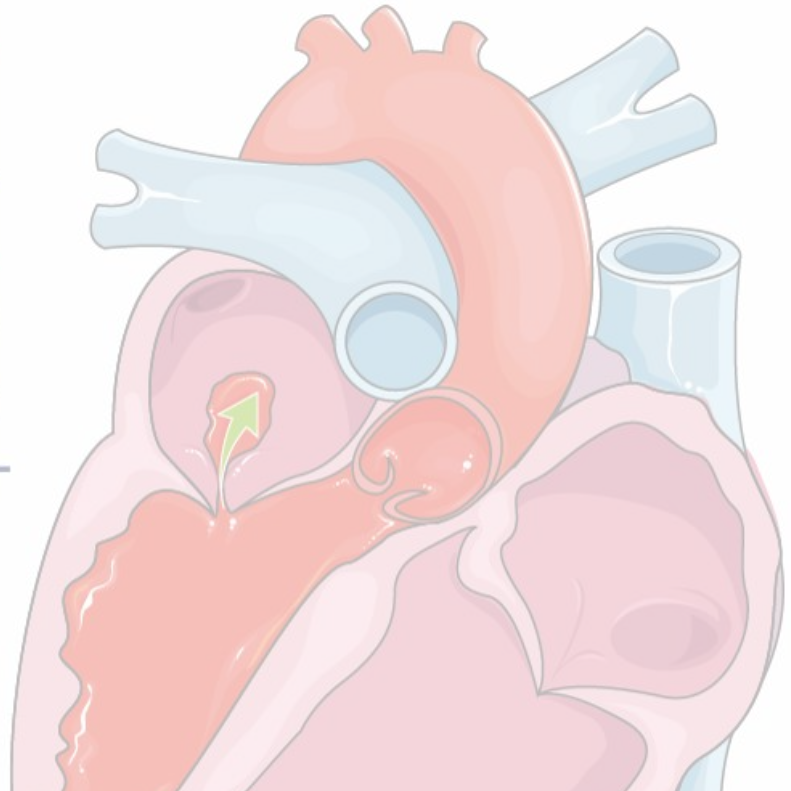
Mg+	magnesium
MgSO4	magnesium sulfate
MI	myocardial infarction
MM	multiple myeloma
MRgFUS	MR-guided focused ultrasound surgery
MRI	magnetic resonance imaging
NAA	nucleic acid amplification
NG	nasogastric
NGT	nasogastric tube
NK	natural killer
NMDA	N-methyl D-aspartate
NMJ	neuromuscular junction
NMS	neuroleptic malignant syndrome
NPO	nil per os (nothing by mouth)
NSAIDs	nonsteroidal antiinflammatory drugs
O2	oxygen
OCD	obsessive-compulsive disorder
OmpC	outer membrane porin C
ORIF	open reduction with internal fixation
OSHA	Occupational Safety and Health Administration
OTC	over-the-counter
PA	placenta abruption
PABA	para-aminobenzoic acid
PaCO2	partial pressure of carbon dioxide in alveolar gas
PAD	peripheral arterial disease
P-ANCA	perinuclear antineutrophil cytoplasmic antibody
PAO2	alveolar oxygen partial pressure
Pap	Papanicolaou
PCOS	polycystic ovarian syndrome
PCR	polymerase chain reaction
PD	Parkinson's disease
PD	peritoneal dialysis
PDA	patent ductus arteriosus
PE	pulmonary embolism
PEEP	positive end-expiratory pressure
PET	positron emission tomography
PFT	pulmonary function test
pH	potential of hydrogen
PIH	pregnancy-induced hypertension
PIPIDA	99mTc-para-isopropylac-(scan) etanilido-iminodiacetic acid (cholescintigraphy)
PND	paroxysmal nocturnal dyspnea
PP	placenta previa
PRBCs	packed red blood cells
PSA	prostate-specific antigen
PSV	peak systolic velocity
PT	prothrombin time
PUBS	percutaneous umbilical blood sampling
PUVA	psoralen ultraviolet A
PVC	premature ventricular contraction
PVR	peripheral vascular resistance
QFT-G	QuantiFERON-TB Gold
R/O	rule out
RA	rheumatoid arthritis
RAIU	radioactive iodine uptake
RBC	red blood cell
RD	Raynaud's disease

RF	rheumatoid factor
RFT	renal function tests
RLQ	right lower quadrant
ROM	range of motion
RSV	respiratory syncytial virus
RUQ	right upper quadrant
SA	sinoatrial
SAD	seasonal affective disorder
SARS	severe acute respiratory syndrome
SBP	systolic blood pressure
SCI	spinal cord injury
SDAT	senile dementia of the Alzheimer type
SERM	selective estrogen receptor modulator
SGA	small-for-gestational-age
SIADH	syndrome of inappropriate diuretic hormone
SJS	Stevens-Johnson syndrome
SLE	systemic lupus erythematosus
SNS	sympathetic nervous system
SOB	shortness of breath
SPECT	single-photon emission computed tomography
SPF	skin protection factor
SSRI	selective serotonin reuptake inhibitor
STD	sexually transmitted disease
T3	triiodothyronine
T4	tetraiodothyronine
T6	thoracic nerve pair 6
TB	tuberculosis
TEE	transesophageal echocardiogram
TEN	toxic epidermal necrolysis
TENS	transcutaneous electrical nerve stimulation
TG	thyroglobulin
THR	total hip replacement
TKR	total knee replacement
TN	trigeminal nerve
TNF	tumor necrosis factor
TNF-I	tumor necrosis factor inhibitors
TNF-α	tumor necrosis factor alpha
TNM	tumor-node-metastasis
TPN	total parenteral nutrition
TPO	thyroid peroxidase
TRAP	tremor, rigidity, akinesia criteria or postural instability bradykinesia, and postural instability
TSH	thyroid-stimulating hormone
tTG	antitransglutaminase
TUMA	transurethral microwave antenna
TURP	transurethral resection of the prostate
UC	ulcerative colitis
US	ultrasound
UTI	urinary tract infection
UV	ultraviolet
V/Q	ventilation/perfusion
VF	ventricular fibrillation
VT	ventricular tachycardia
WBC	white blood cell



Cardiovascular System Disorders





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Clinical Medicine Flashcards

- Clinical Clues to Diagnosis
- Pathophysiology

- Angina Pectoris
- Aortic Aneurysm
- Aortic Stenosis
- Atrial Fibrillation
- Buerger’s Disease
- Cardiogenic Shock
- Cardiomyopathy
- Congestive Heart Failure
- Coronary Artery Disease
- Deep Vein Thrombosis
- Graft-Versus-Host Disease
- Hypertension
- Leukemia
- Metabolic Acidosis
- Metabolic Alkalosis
- Multiple Myeloma
- Myocardial Infarction
- Myocarditis
- Pericarditis
- Peripheral Artery Disease
- Raynaud’s Disease
- Respiratory Acidosis
- Respiratory Alkalosis
- Rheumatic Endocarditis
- Varicose Veins
- Venous Stasis Ulcer
- Ventricular Fibrillation
- Ventricular Tachycardia

1 Angina Pectoris

- Chest pain referred to the jaw, neck, upper arms, and scapulae that is usually associated with activity, cold weather exercise, or smoking.
- Usually subsides with rest.

Pathophysiology

- The coronary arteries that feed the heart muscle become occluded with atherosclerotic plaque. Increased oxygen demands cannot be met because of narrowing and noncompliance to dilation. Ischemic pain results and is referred to the jaw, inner upper arms, sternum, and between the scapulae.
- Causative events include the 4 Es—eating a large meal, excitement, environment (very cold or very hot), and exercise—as well as smoking.
- Types include stable angina; variant angina (Prinzmetal's), unstable angina, which can easily lead to MI; and silent ischemia, usually experienced by older adults, that damages the heart without pain.

2 Aortic Aneurysm

- Abdominal pain, nausea, or fullness relieved by position change.
- Pulsating mass in the abdomen.
- Auscultation with the bell of the stethoscope for a bruit adjacent to the umbilicus.

Pathophysiology

- Bulging or ballooning of the aorta due to atherosclerosis, hypertension, chronic obstructive pulmonary disease, smoking, trauma, or congenital anomaly. Commonly found in the abdominal aorta (abdominal aortic aneurysm [AAA]). Tends to run in families with Marfan's syndrome.
- Types include fusiform, saccular, and dissecting.
- May be completely asymptomatic until it ruptures.

3 Aortic Stenosis

- Presence of a loud, harsh midsystolic, crescendo–decrescendo murmur that radiates to the side of the neck and down the left sternal border or apex.
- Heard loudest at the second right ICS.
- Low BP,
- Fatigue,
- Dizziness &
- Chest pain.

Pathophysiology

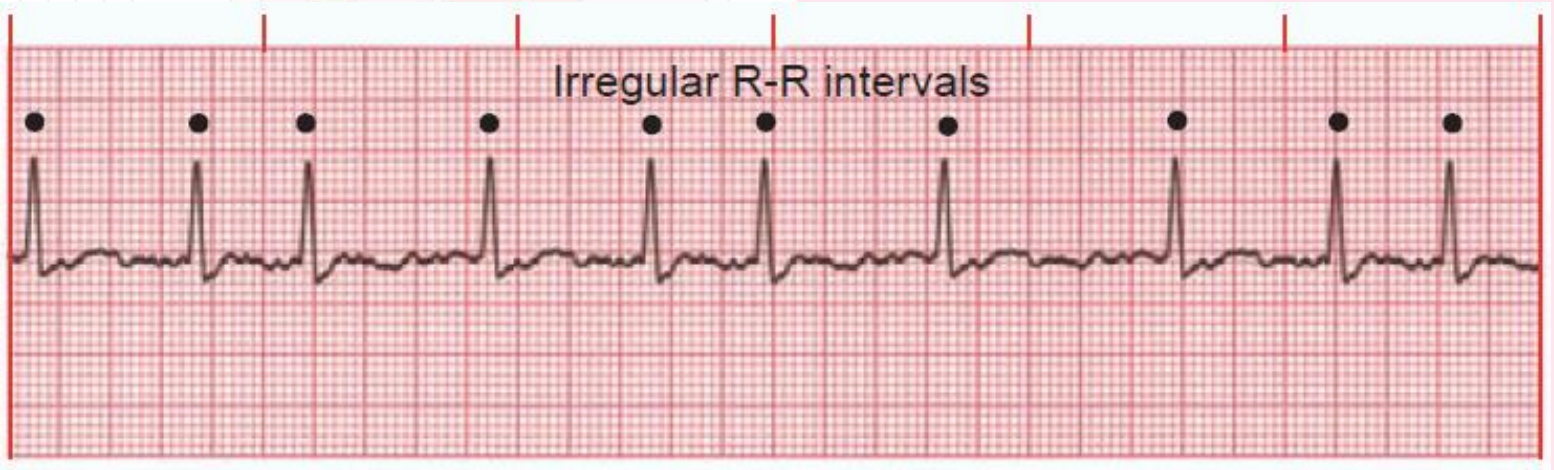
- AS develops from thickening, scarring, calcification, vegetation, or fusing of the flaps of the valve.
- Left ventricular hypertrophy occurs as the sympathetic nervous system is activated to compensate for low cardiac output. When compensatory mechanisms fail, heart failure results.

4 Atrial Fibrillation

- Palpitations
- Skipping heartbeats
- Vertigo

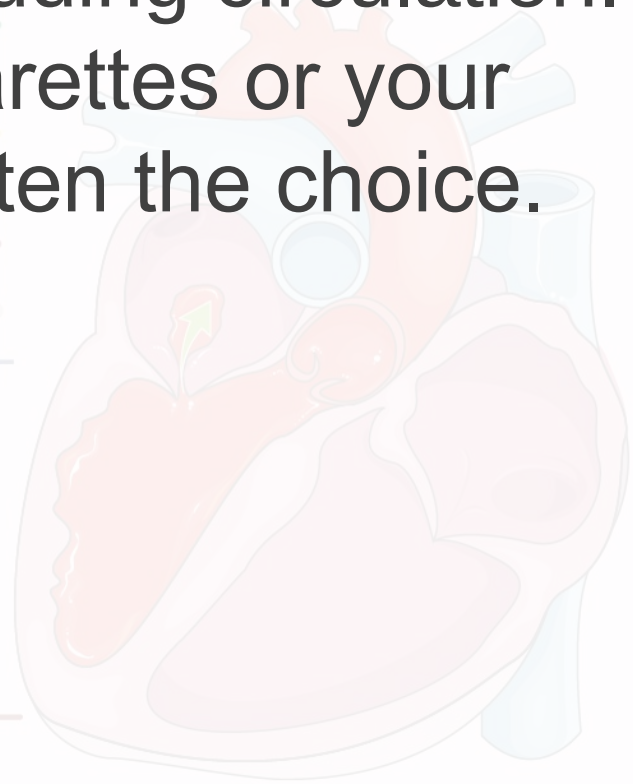
Pathophysiology

- Atrial fibrillation (AF), or quivering of the atria, is caused by repeated reentry of stimuli to the atrioventricular (AV) node.
- Loss of atrial kick.
- Stimulation of the sympathetic nervous system, as well as increasing age, illness (e.g., hyperthyroidism), and the stress of surgery may initiate AF.
- Types of AF include paroxysmal, persistent, permanent, and lone.



5 Buerger's Disease

- A disease of young men who smoke.
- Thrombi develop in the legs, occluding circulation.
- "Your cigarettes or your legs" is often the choice.

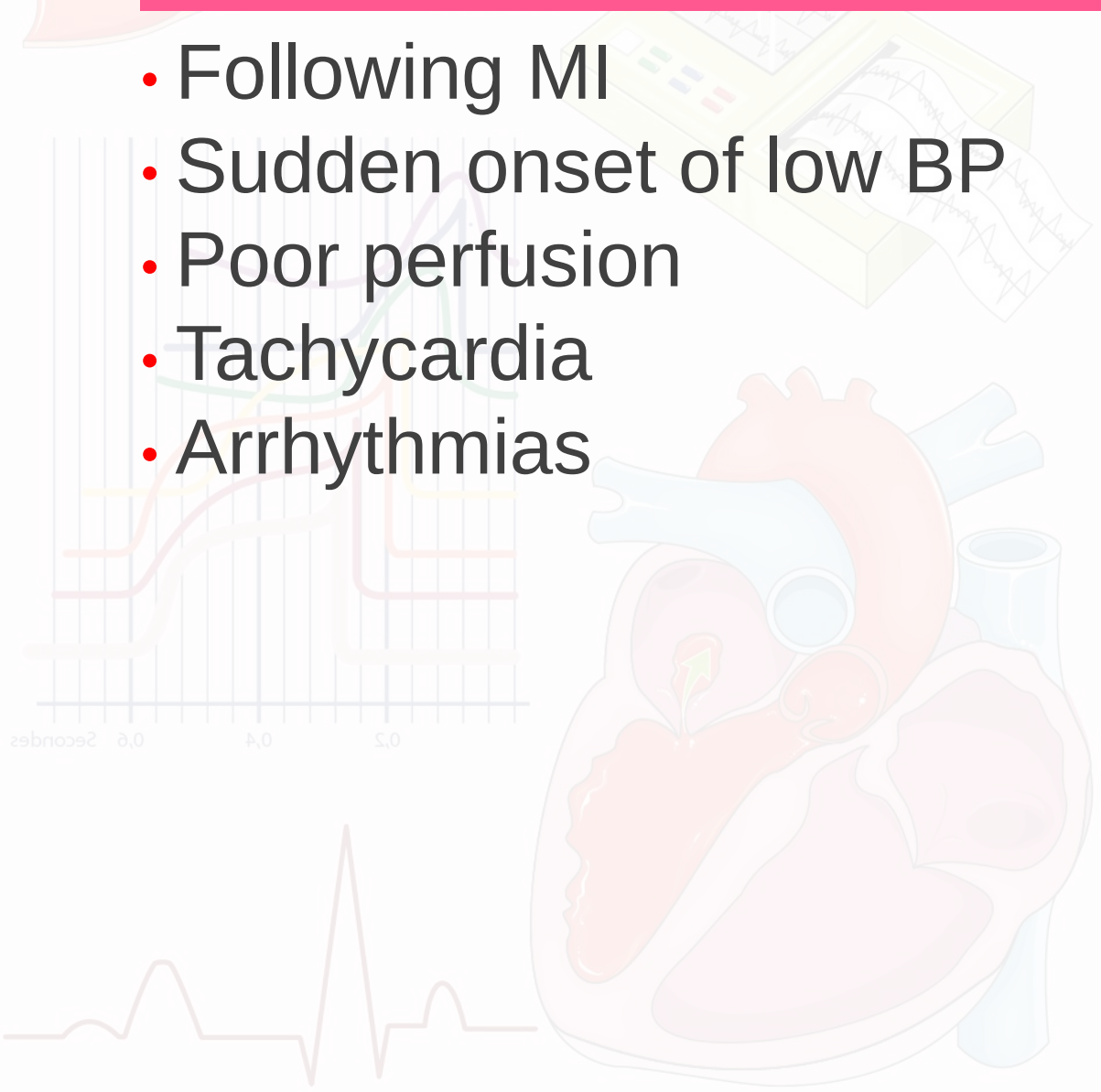


Pathophysiology

- BD also known as thromboangiitis obliterans is a disease of recurrent inflammation of the small and medium arteries of the legs that results in thrombus formation.
- Young men (aged 25–40) who smoke are affected. It is thought that substances in the tobacco products trigger an autoimmune response in these young men. Vasospasm and loss of arterial blood flow occurs.

6 Cardiogenic Shock

- Following MI
- Sudden onset of low BP
- Poor perfusion
- Tachycardia
- Arrhythmias



Pathophysiology

- AMI leads to decreased contractility of either the right or left ventricle, decreasing cardiac output to all body organ systems.
- CS may be caused by pericarditis and resulting cardiac tamponade.
- Stenosis of heart valves or sustained arrhythmia can cause CS.
- Drugs, used for preexisting hypertension, angina, or arrhythmias, may reach toxic levels and cause CS.

7 Cardiomyopathy

- Dyspnea
- Fatigue
- Edema of the ankles, and
- Possible atypical chest pain occurring with rest and not relieved with nitrates.
- MRI shows enlargement of the heart muscle or chambers.

Pathophysiology

- Enlargement of the heart muscle or chambers of the heart that causes heart failure.
- Major types: dilated and restrictive.
- Causes: Heredity, myocarditis, chronic alcohol or cocaine use, HIV, thiamine or zinc deficiencies, infections; or autoimmune disease.

8 Congestive Heart Failure

- Elevated BNP,
- Edema in the
- Extremities
- Shortness of breath
- Crackles and pleural effusion
- Jugular vein distention
- Hepatomegaly
- Splenomegaly

Pathophysiology

- The heart is a double pump. Any structural damage to the pump will cause heart failure.
- Left-sided heart failure causes backup of fluid in the lungs.
- Right-sided heart failure causes backup of fluid in the inferior and superior venae cavae.
- Preload becomes extensive and afterload is difficult to overcome because of ↑ PVR.

9 Coronary Artery Disease

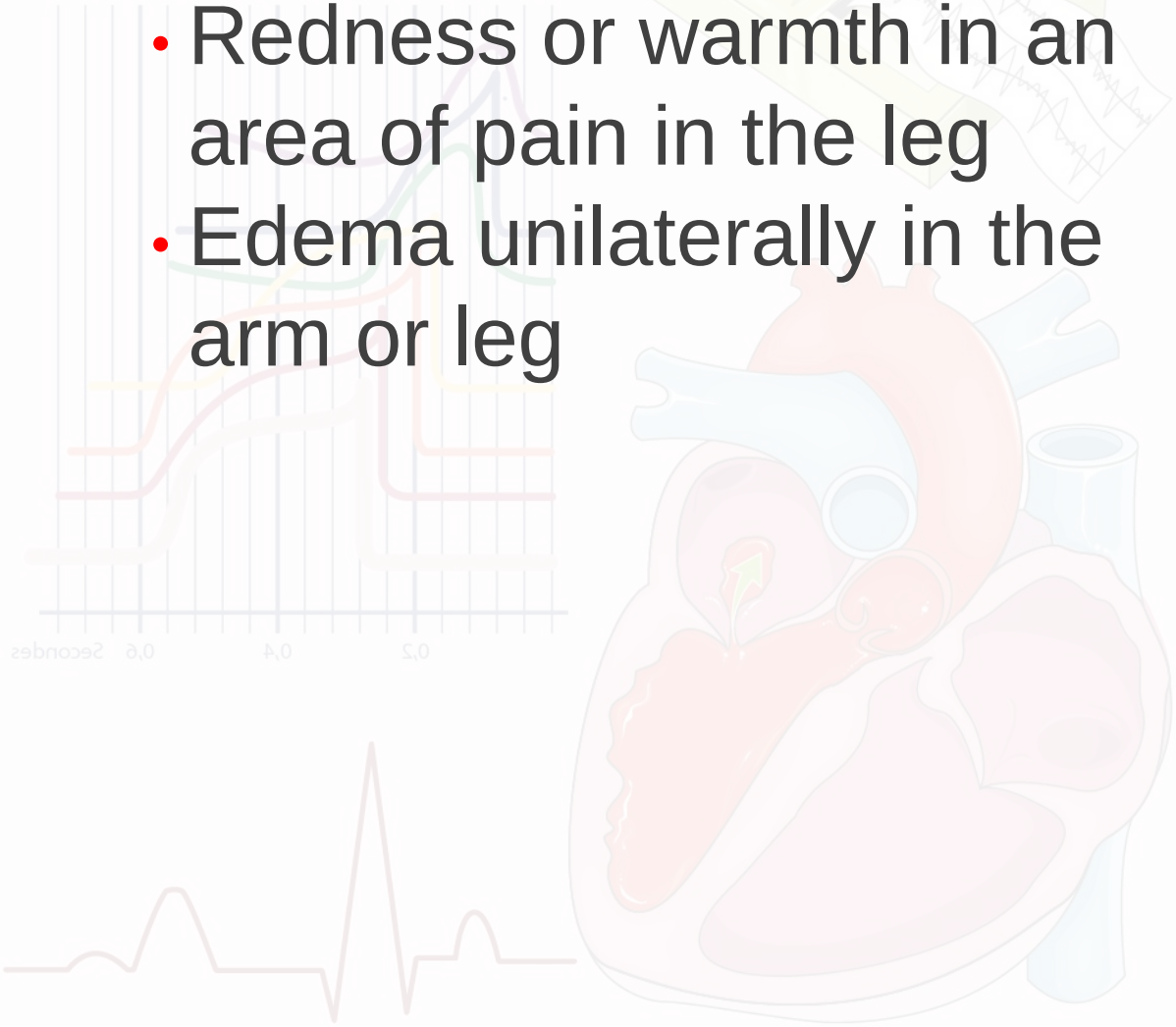
- Shortness of breath with activity in a client with risk factors for heart disease such as a history of
  - Elevated blood lipids
  - Smoking
  - Poor dietary habits
  - Sedentary lifestyle
  - Obesity

Pathophysiology

- CAD results in interruption of blood flow that can cause ischemia or infarction as a result of atherosclerosis.
- The inflammation attracts low-density lipoproteins (LDL) and binds them to the site. The triglyceride core of the LDLs is spilled into the underlayer of the intima. Macrophages envelop these fats and are now termed “foam cells.”
- This is the “fatty streak” seen in early stages of atherosclerosis. As the area enlarges, more LDL, macrophages, platelets, and smooth muscle fibers are drawn to the site and accumulate under the intima, narrowing the vessel.
- This causes reduced blood flow and higher blood pressure in the small coronary vessels.

10 Deep Vein Thrombosis

- Positive homans' sign
- Redness or warmth in an area of pain in the leg
- Edema unilaterally in the arm or leg



Pathophysiology

- Causes of DVT include venous stasis, vessel wall injury, and hypercoagulability. Perinatally, women are at increased risk because of excess clotting factors.
- Areas where blood flows more slowly, usually where veins are bending are more prone to DVT.
- Postsurgery clients are at greater risk due to ↓ activity.
- Septicemia resulting in hemolysis and dehydration can contribute to DVT.

11 Graft-Versus-Host Disease

- Approximately 31/2 months following solid organ, bone marrow, or stem cell transplant, damage to the epithelial cells of the skin, GI tract, and hepatocytes occurs from an immune attack initiated by the transplanted tissue.

Pathophysiology

- GVHD can occur following solid organ, bone marrow, or stem cell transplant. The graft cells recognize the host cells as foreign.
- Phase 1 of GVHD involves the host tissue that has been prepared for transplant by use of chemotherapy and radiation therapy. The injured tissue releases cytokines, which stimulate the host's CD4+ cells.
- In phase 2 of GVHD, activated CD4+ cells cause the graft to activate T killer cells and NK cells that mount an immune response against susceptible tissues of the host (epithelial tissue, GI tract, and hepatocytes).
- In phase 3 of GVHD, immune cells and cytokines begin to damage host tissues.

12 Hypertension

- BP readings of greater than 119 mm Hg systolic or greater than 79 mm Hg diastolic classify the client as prehypertensive.
- The client may have no symptoms or, in severe cases, headache and nosebleed.

Pathophysiology

- BP is determined by CO, which is determined by heart rate multiplied by the stroke volume. The heart rate can be affected by stimulation of the SNS responding to arterial baroreceptors that measure BP and by chemoreceptors that measure CO2 levels. Other mechanisms that alter BP include the renin-angiotensin-aldosterone system, exercise, emotions, and taking medications that cause vasoconstriction. High blood pressure damages the intima of arteries, making way for infiltration of macrophages, muscle fibers, cholesterol, and fatty acids that form atherosclerotic plaque.
- PVR is the resistance to blood flow through arterioles creating a high afterload.

13 Leukemia

- Low-grade fever
- Lymphadenopathy
- Bleeding tendency
- Infections
- Anemia.
- Bone marrow biopsy shows many immature WBCs.

Pathophysiology

- Leukemia can be acute or chronic and affect lymphocytes, monocytes, granulocytes, erythrocytes, and platelets. Due to a mutation in the stem cells of the bone marrow, immature WBCs (blasts), proliferate uncontrollably in the bone marrow, lymph tissue, and spleen. In the bone marrow, the immature and ineffective WBCs crowd the normal WBCs, RBCs, and platelets, greatly reducing their number.
- Types include ALL, AML, CLL, CML.

14 Metabolic Acidosis

- ABG shows pH of less than 7.35,
- PCO2 in the range of 35–45 mm Hg or decreasing to compensate, and
- HCO3_ of less than 22 mEq/L.

Pathophysiology

- Normal pH of the body is 7.35–7.45. ABG analysis diagnoses metabolic acidosis; pH is low, CO2 is within normal range or decreasing to compensate, and HCO3_ is low.
- Buffering systems are initiated by the body when the pH goes out of range. The first to react are cellular buffers. In metabolic acidosis, H+ are absorbed into the cells, causing a shift of K+ into the extracellular area.
- The lungs are the second buffering system to activate. When pH is low, CO2 is released through rapid and deep respirations.The kidneys are the last buffering system to activate; and it may take as long as 1–2 days for them to begin to affect pH. In metabolic acidosis, the kidneys secrete H+.
- Causes include diarrhea (loss below the waistlose base), CRF, lactic acidosis, salicylate poisoning, methanol and alcohol poisoning, paraldehyde poisoning, and diabetic ketoacidosis.

15 Metabolic Alkalosis

- ABG shows pH of greater than 7.45,
- PCO2 in the range of 35–45 mm Hg or rising to compensate, and HCO3_ of greater than 26 mEq/L.

Pathophysiology

- Normal pH of the body is 7.35–7.45. The ABG diagnoses metabolic alkalosis, pH is high, CO2 is within normal range or increasing to compensate, and HCO3_ is high.
- Buffering systems are initiated by the body when the pH goes out of range. The first to react are cellular buffers. In metabolic alkalosis, H+ are released from the cells, causing a shift of potassium ions (K+) into the cells.
- The lungs are the second buffering system to activate. When pH is high, CO2 is held by slow, shallow respirations.
- The kidneys are the last buffering system to activate, and it may take as long as 1–2 days for them to begin to affect pH. In metabolic alkalosis, the kidneys hold H+.
- Causes include persistent vomiting; gastrointestinal suction; diarrhea; and use of loop diuretics, antacids, licorice, glucocorticoids, and mineralocorticoids.

16 Multiple Myeloma

- Pathologic fractures from severe osteoporosis
- Bleeding tendency
- Infections
- Anemia affecting those in the fifth to seventh decades of life.

Pathophysiology

- Mutation of plasma cells (type of B-lymphocyte) that infiltrate the bone marrow, bone tissue, liver, spleen, lymph nodes, lungs, adrenal glands, kidneys, skin, and GI tract.
- MM has a poor prognosis.

17 Myocardial Infarction

- Severe chest pain that refers to the jaw, upper arms, neck, and scapula and is described as “crushing.”
- Accompanied by shortness of breath, elevated BP, and sweating.

Pathophysiology

- When blood flow diminishes to the heart muscle, the sympathetic nervous system is activated, raising the blood pressure and heart rate. This increases the oxygen and glucose needs of the cardiac cells.
- Cardiac necrosis from lack of perfusion occurs centrally, surrounded by varying levels of ischemic tissue radiating outward from the site.
- Necrotic cardiac tissue will never resume its prior ability to contract but rather will form scar tissue.
- Damage can occur to the pacing system of the heart, causing lethal arrhythmias.

18 Myocarditis

- Fever,
- Chest pain, and
- Activity Intolerance.

Pathophysiology

- The myocardium is infiltrated by inflammatory cells leading to necrosis of muscle cells and fibrosis.
- Causes include viral, bacterial, protozoan, and fungal infections.
- Inflammatory and autoimmune causes or exposure to chemicals or toxins, and radiation therapy.
- Women who are pregnant, those undergoing radiation therapy to the chest area, and the elderly are also at risk.

19 Pericarditis

- Pericardial friction rub.
- Substernal radiating chest pain that increases in intensity with deep inspiration or lying flat.
- Pain is somewhat relieved by sitting upright and leaning forward.
- CBC and ESR may indicate inflammation or infection is present.

Pathophysiology

- Pericarditis is an inflammation of the pericardial sac. The pericardial sac is a fibrous tissue layer that surrounds the heart. Under normal circumstances, it contains and is bathed with approximately 25–50 mL of serous fluid. In pericarditis, the volume may increase to 1,500 mL.
- Many diseases, conditions, and drugs can inflame the pericardial sac.
- Hemopericardium may be caused by trauma and in-hospital procedures.

20 Peripheral Arterial Disease

- Symptoms occur late in the disease and include intermittent claudication in the calves associated with activity.
- Color changes in the legs, with hair loss and dry, flaky skin, may occur.

Pathophysiology

- PAD is caused by progressive narrowing of the lumen of the arteries by atherosclerotic plaque buildup.
- If arteries are totally occluded, necrosis and ulceration (gangrene) develop, and the limb is no longer viable.

21 Raynaud's Disease

- Vasospasm and vasoconstrictive ischemia of the tips of the nose, fingers, hands, feet, and toes when in contact with cold objects or cold temperatures.

Pathophysiology

- A disease of women, RD causes vasospasm and vasoconstrictive ischemia of the tips of the nose, fingers, hands, feet, and toes when in contact with cold objects or cold temperatures. Ischemia is followed by a period of hyperemia. Diagnosis is made when the ischemic attacks occur for 2 or more years.
- Endothelin 1 and angiotensin may be causative agents.
- Secondary RD is associated with autoimmune/ collagen disorders and persons with occupations that involve vibratory tools like jackhammers.

22 Respiratory Acidosis

- ABG shows pH of less than 7.45,
- PCO2 of greater than 45 mm Hg, and HCO3_ within range or rising to compensate.

Pathophysiology

- Normal pH of the body is 7.35–7.45. The ABG analysis diagnoses respiratory acidosis; pH is low, CO2 is high, and HCO3_ is within normal range or rising to compensate.
- Buffering systems are initiated by the body when the pH goes out of range. The first to react are cellular buffers. In respiratory acidosis, H+ are absorbed into the cells, causing a shift of K+ out of the cells.
- The lungs are the second buffering system to activate. When pH is low, CO2 is released through rapid and deep respirations.
- The kidneys are the last buffering system, and it may take as long as 1–2 days for them to begin to affect pH. In respiratory acidosis, the kidneys secrete H+.
- Causes include COPD, hypoventilation, sleep apnea, and drug use that suppresses respiratory function..

23 Respiratory Alkalosis

- ABG shows pH of greater than 7.45,
- PCO2 of less than 35 mm Hg, and
- HCO3_ within the range of 22–26 mEq/L or decreasing to compensate.

Pathophysiology

- Normal pH of the body is 7.35–7.45. The ABG analysis diagnoses respiratory alkalosis; pH is high, CO2 is low, and HCO3_ is within normal range or decreasing to compensate. Buffering systems are initiated by the body when the pH goes out of range. The first to react are cellular buffers. In respiratory alkalosis, H+ are released from the cells, causing a shift of K+ into the cells.
- The lungs are the second buffering system to activate. When pH is high, CO2 is held by slow, shallow respirations.
- The kidneys are the last buffering system to activate, and it may take as long as 1–2 days for them to begin to affect pH. In respiratory alkalosis, the kidneys hold H+.
- Causes include pain, anxiety, fever, CVA, tumor, and trauma.

24 Rheumatic Endocarditis

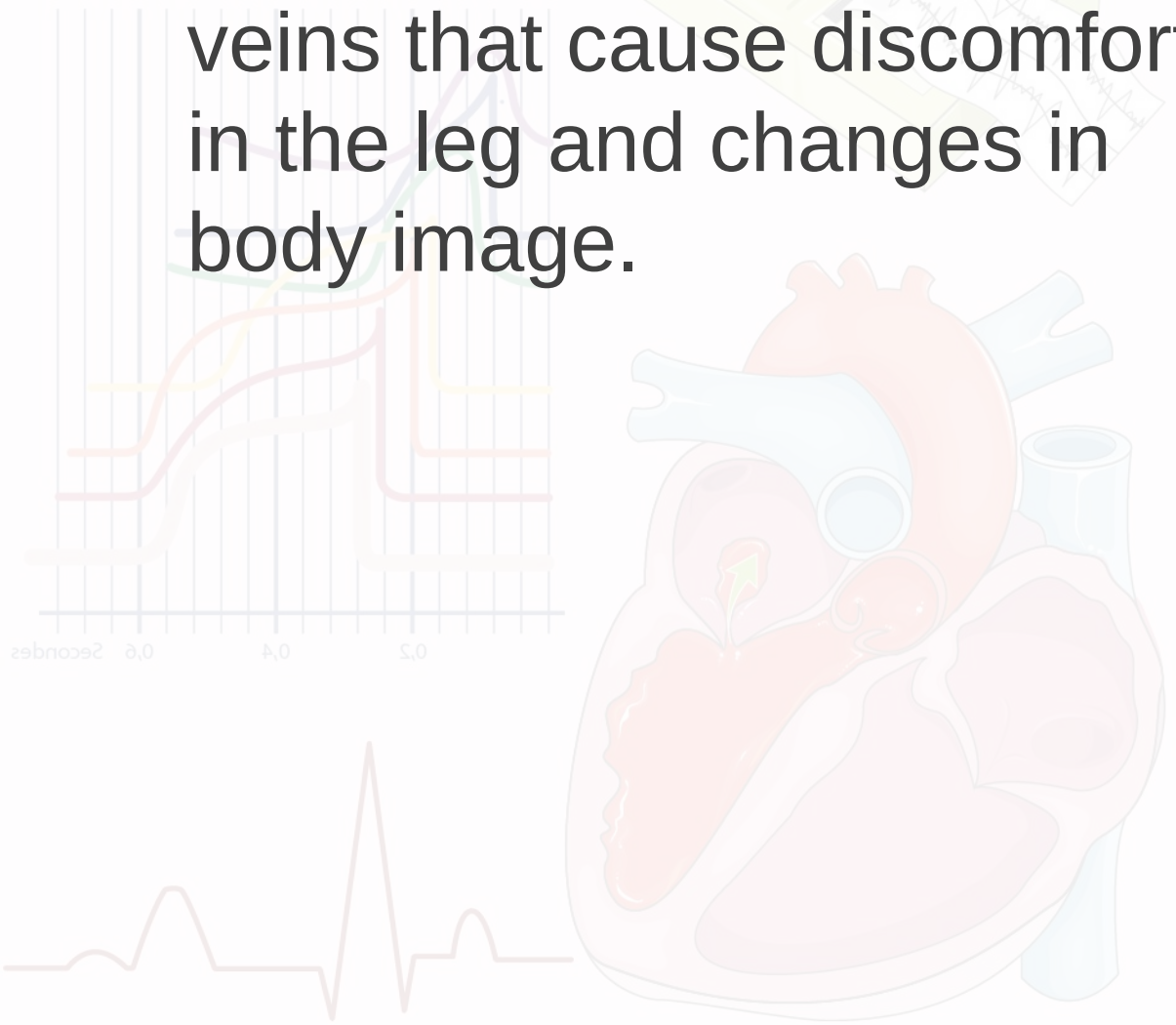
- Fever
- Chest pain
- Dyspnea
- Cough
- Arthritic symptoms
- Chorea and
- Ankle edema develop 2–3 weeks after strep. Throat (beta-hemolytic streptococci).

Pathophysiology

- BHS that cause throat infection or impetigo travel to the bloodstream, causing bacteremia. The BHS infect the heart typically 2–3 weeks after the initial infection. May occur in clients in childhood and recur as rheumatic endocarditis at any age.
- All layers of the heart are affected, with generalized inflammation of all heart structures.
- The endocardium is affected by vegetation deposited on the valves.
- The end result of cardiac structural anomalies is CHF.

25 Varicose Veins

- Visible, tortuous, bulging veins that cause discomfort in the leg and changes in body image.



Pathophysiology

- Venous return in the body is dependent on the muscular contractions of the skeletal muscle pump. Competency of the valves within the veins cause forward flow that is eventually returned to the heart.
- In pregnancy, the pressure of the fetus causes venous hypertension, and hormones make the valves less competent, which ↑ incidence of varicose veins of the legs and anus to occur.
- Superficial varicosities are more visible than more deeply located varicosities.

26 Venous Stasis Ulcer

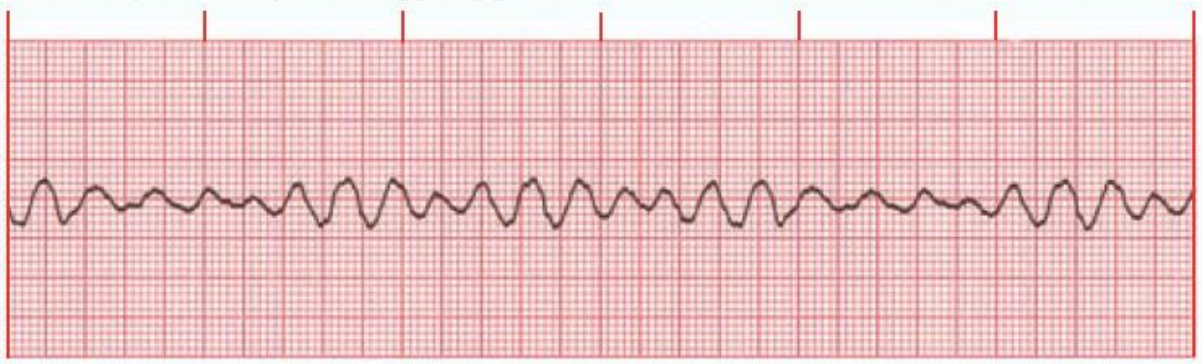
- Ulcer that occurs on the lower extremities in the presence of edema and brown, leathery skin.
- Described as “wet” and exudes a large amount of serous fluid.

Pathophysiology

- Blood is not returned efficiently to the heart and venous pressure ↑ in the lower extremities. The ↑ venous pressures cause backflow of blood into the capillary exchange beds and leakage of serous fluid containing wastes into the interstitial space.
- Edema in the interstitial space prevents capillary access for all cells and can be severe.
- Increased pressure in a vein causes a small rupture that becomes a deeper wound that cannot heal because of poor capillary access to inflammatory agents, oxygen, and glucose. The wound ulcerates because of inflammatory substances trapped in the subcutaneous tissue, damaging the valves in the veins and exuding serous fluid.

27 Ventricular Fibrillation

- Loss of consciousness,
- No peripheral pulses or blood pressure.



Pathophysiology

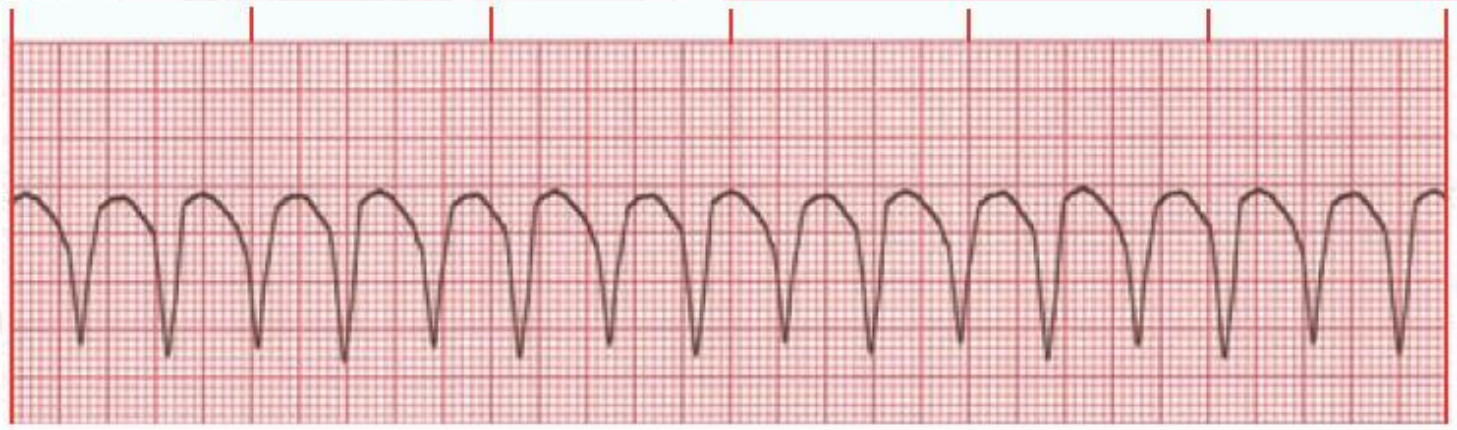
- VF is associated with CAD, MI, and structural or inflammatory cardiac conditions. It may be precipitated by antiarrhythmic drug administration, atrial fibrillation, cardioversion, and hypoxic states.
- VF causes include hyperkalemia and hypomagnesemia, cardiac catheterization and placement of pacemaker wires.
- Congenital conditions that predispose to VF include Marfan’s syndrome, tetralogy of Fallot, Kawasaki’s disease, long QT syndrome, and Wolff-Parkinson-White syndrome also predispose to VF.

28 Ventricular Tachycardia

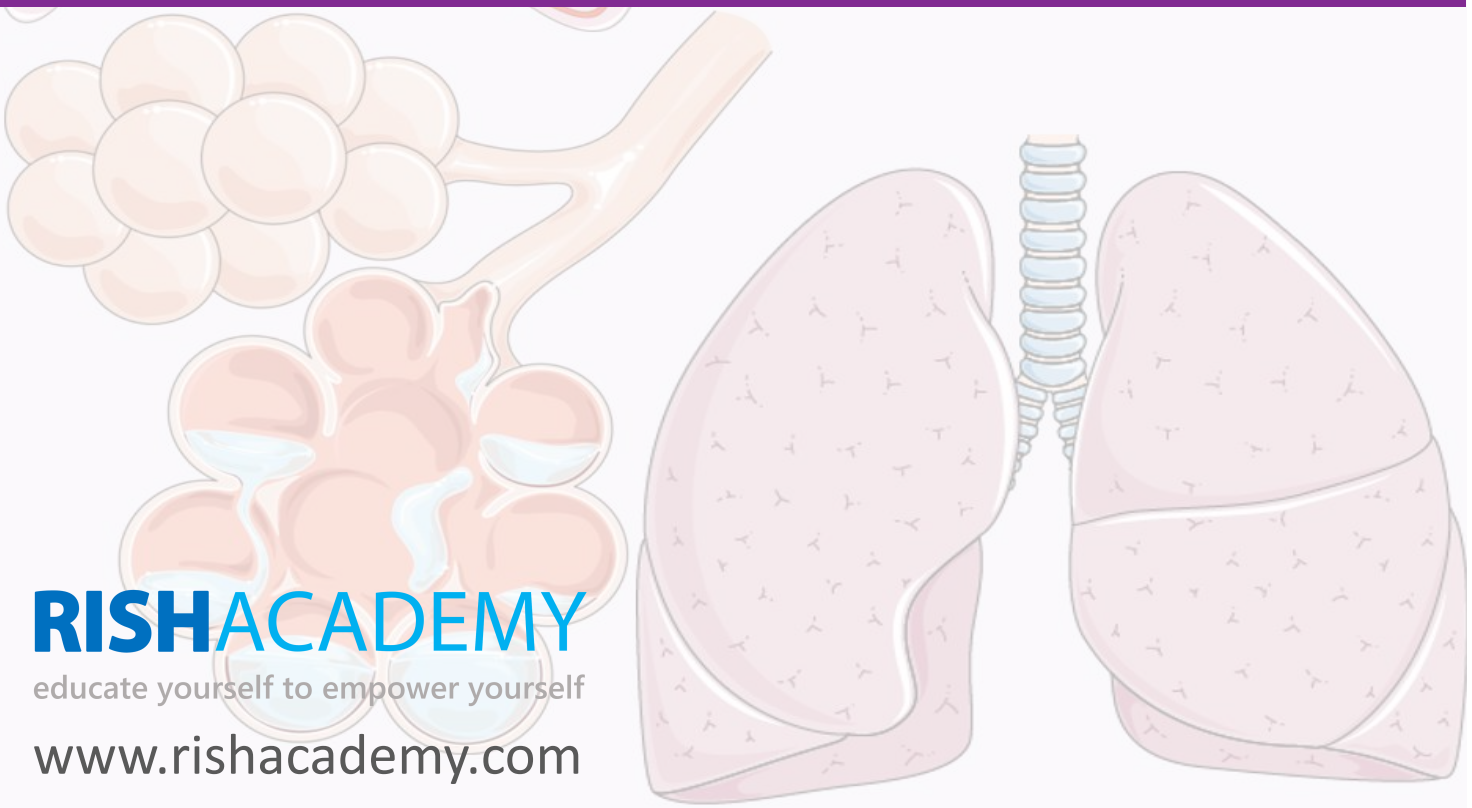
- Client may be lightheaded
- Unconscious and
- Pulseless.

Pathophysiology

- In ventricular tachycardia (VT), the ventricles replace the sinoatrial (SA) node as the pacemaker of the heart.
- PVCs often precede VT.
- VT may be caused by MI, myocardial irritability, and cardiomyopathy.
- Abnormally low levels of K+, Ca++, and Mg+; digoxin toxicity; RA, SLE, and respiratory acidosis.
- Cardiac catheterization and pacing wires.



Respiratory System Disorders



Clinical Medicine Flashcards

- Clinical Clues to Diagnosis
- Pathophysiology

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- Acute Respiratory Distress Syndrome (ARDS)
- Severe Acute Respiratory Syndrome (SARS)
- Asthma
- Bronchiectasis
- Chronic Bronchitis
- Cystic Fibrosis
- Emphysema
- Histoplasmosis
- Influenza
- Legionnaires' Disease
- Lung Cancer
- Mesothelioma
- Pleural Effusion
- Pneumonia
- Pneumothorax
- Pulmonary Embolism
- Sarcoidosis
- Tuberculosis (TB)

1 Acute Respiratory Distress Syndrome (ARDS)

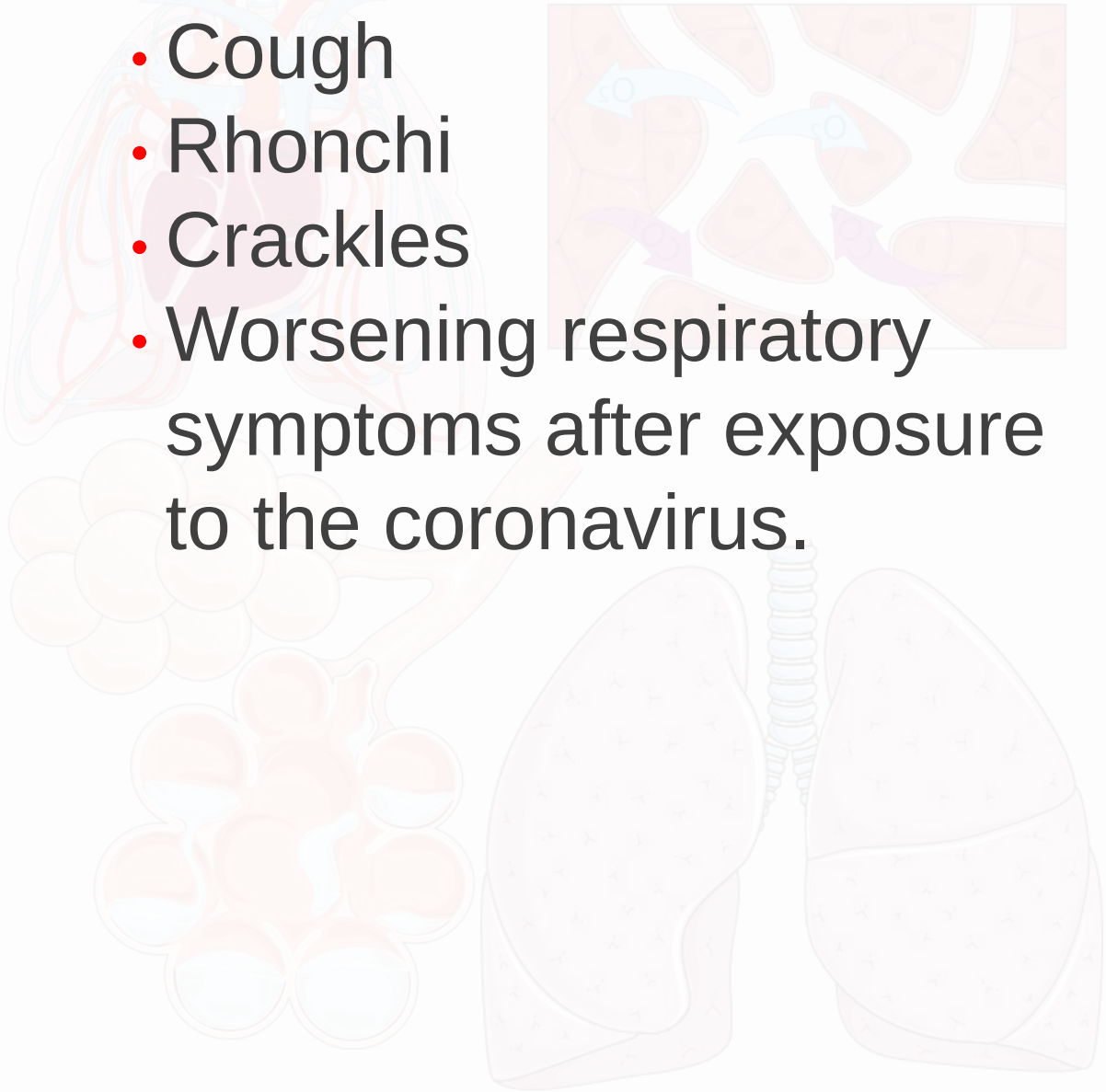
- Rhonchi and crackles on auscultation,
- ↓ O2 saturation after sepsis, near-drowning, or aspiration of gastric contents.

Pathophysiology

- Destruction of alveolar walls and capillary beds caused by stimulation of immune mediators that ↓O2 exchange by fibrosis and edema.
- Immune mediators are also stimulated by traumatic events.

2 Severe Acute Respiratory Syndrome (SARS)

- Cough
- Rhonchi
- Crackles
- Worsening respiratory symptoms after exposure to the coronavirus.



Pathophysiology

- Exposure to the coronavirus by droplet inhalation or contact. The SARS virus can live 6 hours on the hands.
- After contact with the SARS coronavirus, immune mediators cause inflammation, edema, and pneumonia by blocking gas exchange and resulting in filling of the alveoli with fluid.

3 Asthma

- Expiratory wheeze on auscultation
- Rapid onset
- Difficult expiration
- Nonproductive cough “chest is tight,”
- ↓ O2 saturation.

Pathophysiology

- Reactive inflammatory disorder associated with exposure to allergens, viral infection, pollution, smoking, or extremes of temperature.
- Chronic inflammation results in fibrosis and narrowing of bronchiole passageways, leading to air trapping, bronchospasm, and increased dead air space.
- Mast cells, active in inflammation, release histamine, prostaglandins, leukotrienes, and bradykinin.

4 Bronchiectasis

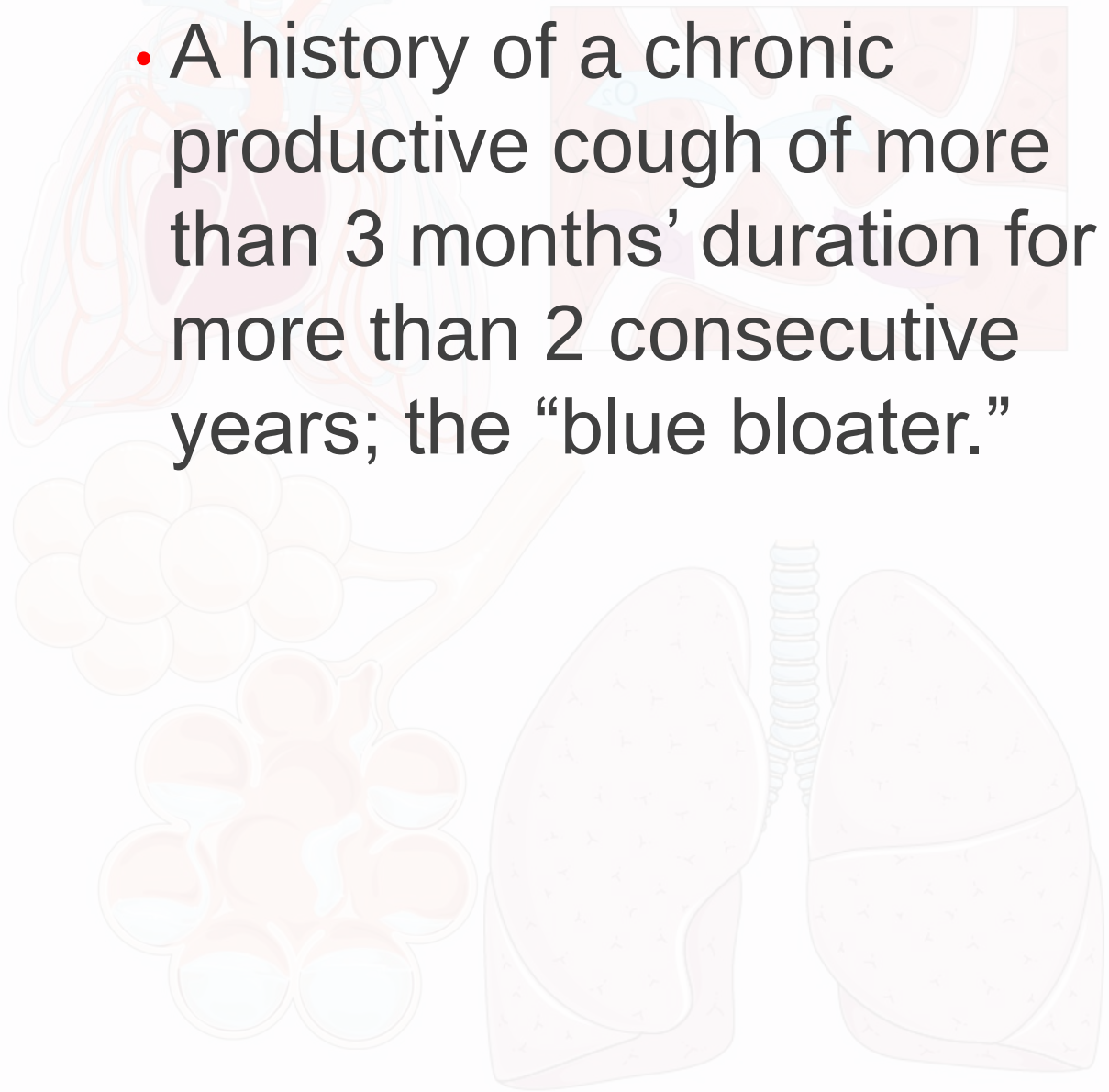
- Dyspnea
- Cyanosis
- Expectoration of large amounts of foul-smelling mucus
- ↓ RBC
- Weight loss
- Rhonchi heard on auscultation

Pathophysiology

- Chronic dilation of the bronchi and bronchioles due to inflammation.
- Inflammatory process destroys elasticity of smooth muscle in the airways.
- Chronic infections occur in dilated areas that retain mucus and obstruct airways.
- Can be localized or diffuse; associated with childhood diseases (e.g., measles), influenza, or tuberculosis.

5 Chronic Bronchitis

- A history of a chronic productive cough of more than 3 months' duration for more than 2 consecutive years; the "blue bloater."

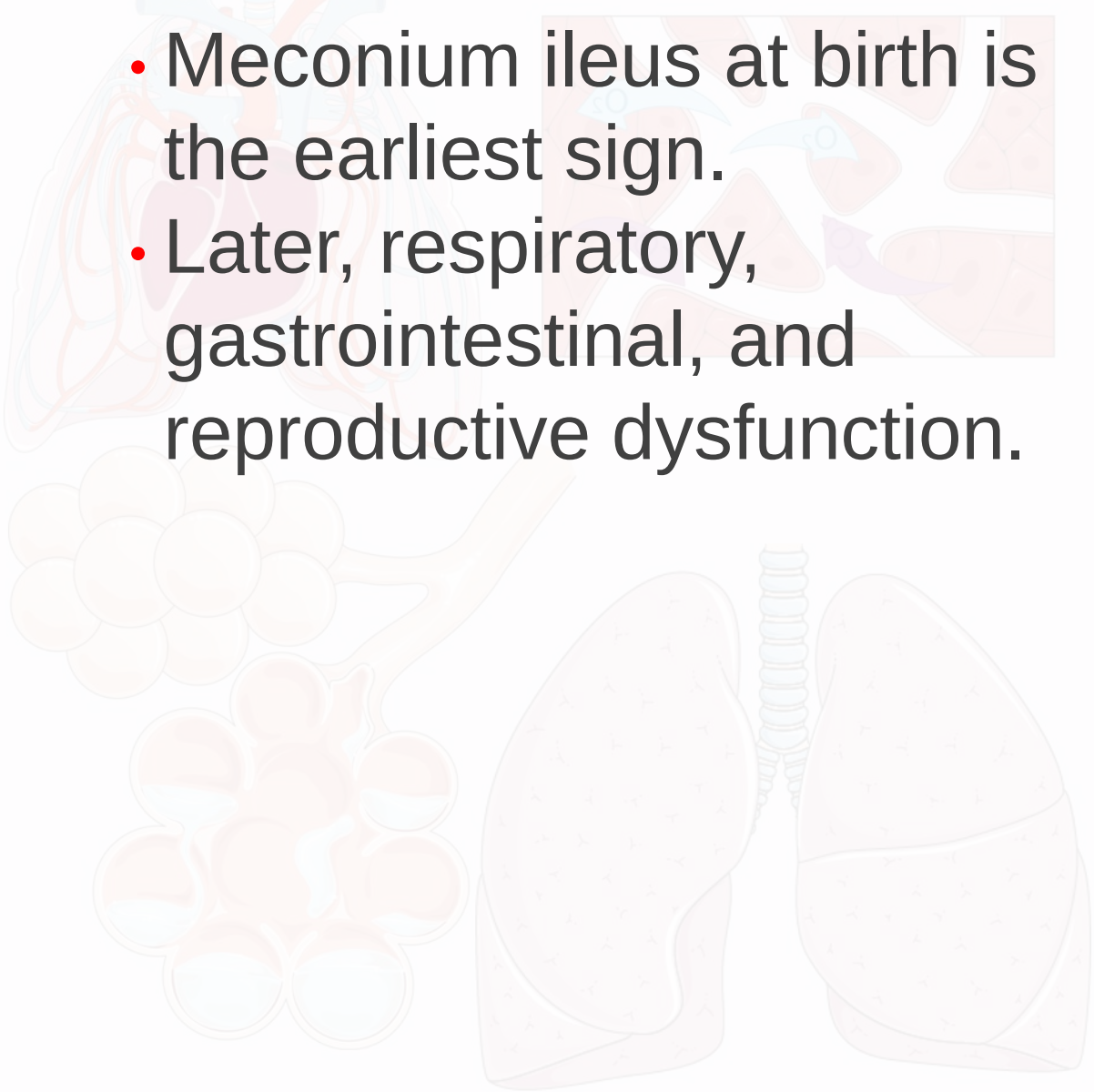


Pathophysiology

- Chronic Inflammation by IL-8 and cytokines of the mucous membrane lining the bronchi and bronchioles. Excess mucus is produced, and the mucociliary pump ceases to function properly, causing chronic congestion. Common in smokers.
- Chronic bronchitis results in a fibrotic, noncompliant airway and pulmonary hypertension.

6 Cystic Fibrosis

- Meconium ileus at birth is the earliest sign.
- Later, respiratory, gastrointestinal, and reproductive dysfunction.



Pathophysiology

- Autosomal recessive disorder that affects chromosome 7, which normally produces a protein CFTR that affects movement of Na+ and Cl– ions.
- All secretions of exocrine glands of the respiratory, gastrointestinal, and reproductive tracts become thick and obstruct normal flow.
- Sweat glands do not reabsorb sodium, so salt depletion in sweat can occur.

7 Emphysema

- Barrel or pigeon chest,
- Dyspnea,
- The “pink puffer,”
- ↑ PaCo2,
- Chronic respiratory acidosis
- Hypoxic respiratory drive.

Pathophysiology

- A chronic disorder in which the alveolar structures distend, lose elasticity, rupture, or coalesce, resulting in damage and destruction to the pulmonary capillary bed, air trapping, and increased dead air space.
- Cigarette smoking and an inherited deficiency of α1-antitrypsin are cocontributors to the disease. ↓ α1-antitrypsin results in elastase secreted from neutrophils that can digest elastin and other alveolar structures.

8 Histoplasmosis

- Cough
- Fever

Pathophysiology

- Systemic fungal disease caused by dimorphic fungus Histoplasma capsulatum.
- Organism grows in soil enriched with bird droppings. Fungal spores form that are then inhaled.
- Once at body temperature, fungal spores change to the yeast form in the alveoli. The yeast is then absorbed through the regional lymphatics and into the bloodstream. Cellular immunity occurs 2–3 weeks after infection.

9 Influenza

- Fever
- Myalgia
- Respiratory and gastrointestinal symptoms.

Pathophysiology

- A viral syndrome that is spread as aerosolized particles (airborne) and causes systemic inflammatory reactions of myalgia, fever, respiratory symptoms, and gastrointestinal symptoms.
- Strains are varied, and influenza vaccine is developed anew each season on the basis of identified strains. H1N1 influenza (swine flu) and H5N1 (bird flu) are relatively new strains causing concern.
- Influenza can easily become pandemic without vaccination.
- The very young, very old, and those with chronic disease are at most risk for death from complications.

10 Legionnaires' Disease

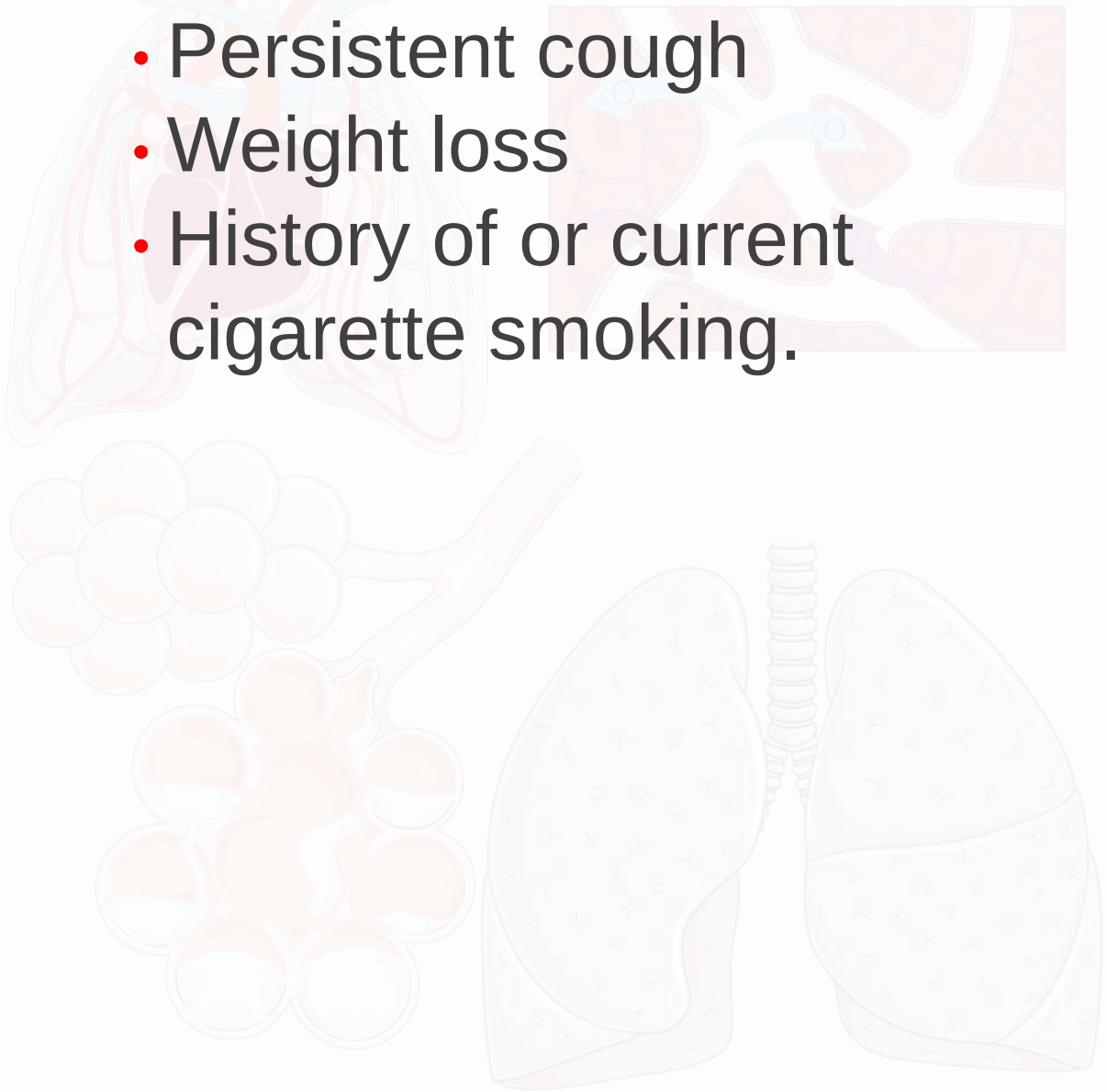
- Dry cough
- Myalgia
- Abnormal lung sounds
- Exposure to contaminated water droplets

Pathophysiology

- *Legionella* bacteria is inhaled from contaminated water supplies (e.g., from air conditioner vents, spas, respiratory equipment), causing pneumonialike symptoms. Thrives at temperatures from 90°–105° F.
- Headache, myalgia, fever, diarrhea. Incubation period of 2–10 days.
- Results in Legionnaire's disease or a lesser influenza-like illness known as Pontiac fever.

11 Lung Cancer

- Persistent cough
- Weight loss
- History of or current cigarette smoking.



Pathophysiology

- Exposure to chronic irritants or carcinogens cause cell mutation, resulting in oncogene stimulation and loss of genetic material from chromosome 3.
- Cells in early differentiation that mutate are more aggressive than more mature cells.
- Cancers are evaluated using the TNM method.
- Cancer cells divide more rapidly and are more metabolic than normal body cells. Secreting cancers cause damage to the body by hypersecretion.
- Cancer in the lung may be the primary site or a metastatic secondary site from a distant body area.

12 Mesothelioma

- Cough
- SOB
- History of asbestos exposure

Pathophysiology

- Mesothelia is a single layer of flat cells that line the pleural, peritoneal, and pericardial cavities. Exposure to asbestos through inhalation causes infiltration by the short asbestos fibers into these cells. Peritoneal infiltration is thought to occur by coughing up and swallowing the asbestos fibers.
- Cells mutate causing changing DNA, and activating oncogenes.

13 Pleural Effusion

- Dyspnea
- Diminished breath sounds over affected area
- Pleural friction rub

Pathophysiology

- The pleural space is the visceral and parietal lining of the outer lungs. Negative pressure or a vacuum exists in this space.
- If the lining becomes damaged or diseased, or experiences oncotic pressure changes (lung cancer, pulmonary tuberculosis [TB], lung abscess, congestive heart failure, ascites, chronic renal disease, chest trauma), the space loses its negative pressure and expands into a space that presses on the lung in that cavity.

14 Pneumonia

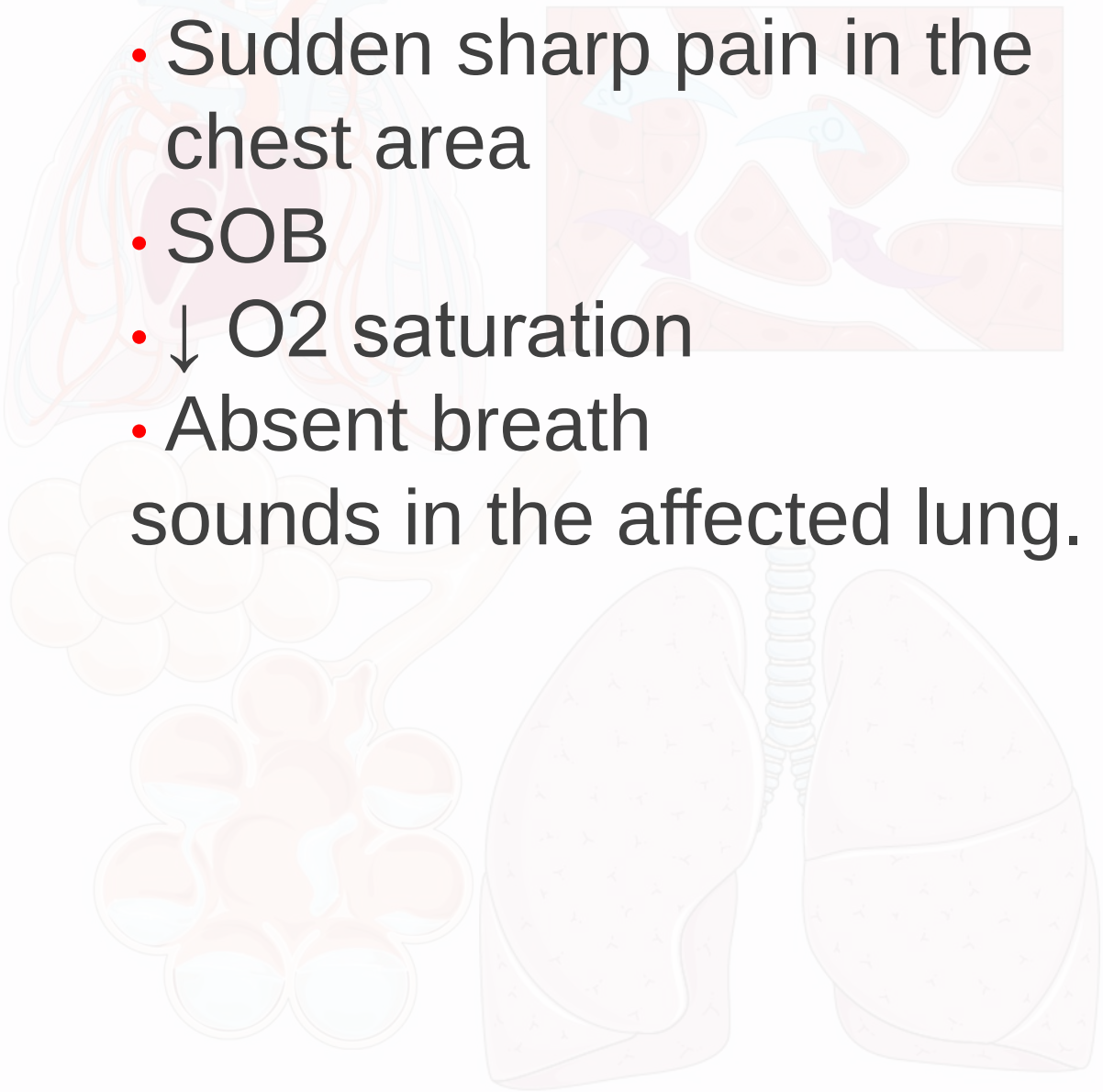
- Productive cough
- Chills
- Dyspnea
- Pain on inspiration

Pathophysiology

- Acute inflammation of lung tissue by inhalation of droplets containing viral particles, bacteria, fungi, parasites, or irritating chemicals.
- Inflammatory mediators in lung tissue cause edema and filling of alveoli with serous fluid and mucus.

15 Pneumothorax

- Sudden sharp pain in the chest area
- SOB
- ↓ O2 saturation
- Absent breath sounds in the affected lung.

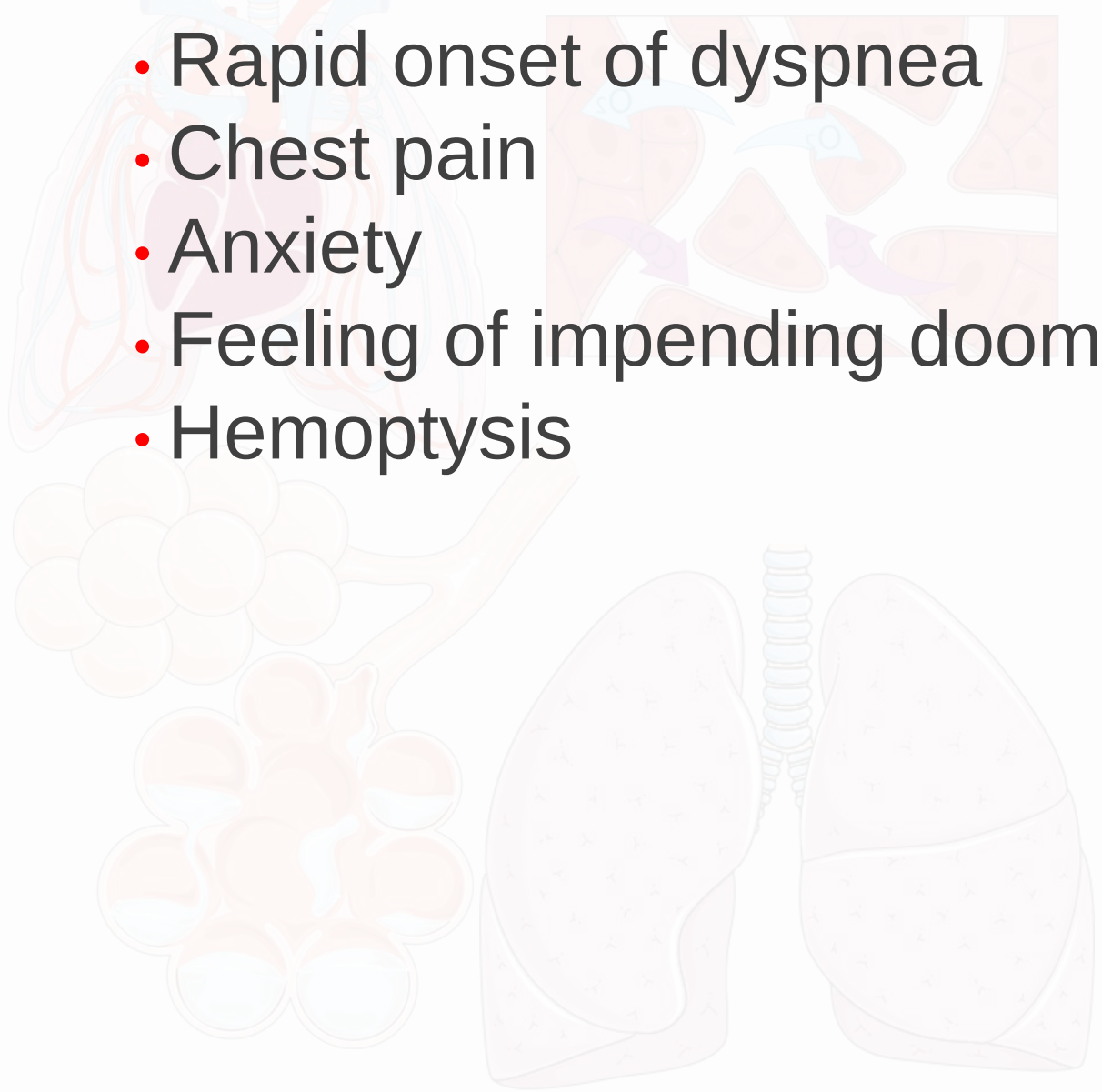


Pathophysiology

- Potential space created by the visceral and parietal pleura creates negative pressure in that area. Once breached by trauma or a pathologic event, negativity is lost and the potential space becomes an actual space that fills with air (pneumothorax) or blood (hemothorax).
- Positive pressure in the pleural space presses against the lung tissue, causing atelectasis ↓ gas exchange.

16 Pulmonary Embolism

- Rapid onset of dyspnea
- Chest pain
- Anxiety
- Feeling of impending doom
- Hemoptysis



Pathophysiology

- Thrombus formation in the deep veins from an ineffective cardiac pump; atrial fibrillation; the presence of increased clotting factors; or lack of movement of the musculoskeletal pump, delaying blood movement back to the heart.
- Emboli may also consist of air, fat, amniotic fluid, and bacteria.
- The thrombus occludes pulmonary circulation, impairing gas exchange.

17 Sarcoidosis

- Fever
- Myalgia
- Night sweats
- Anorexia
- Weight loss
- Fatigue with
- Progressive
- Lung noncompliance and
- SOB

Pathophysiology

- Granulomatous disorder primarily of the lungs, skin, eyes, and lymphatics thought to have a genetic link. Other organs affected are the heart, bones, joints, liver, and kidneys.
- Genetic clusters include mainly African Americans and Scandinavians. Environmental influences are considered as genetic triggers. Affects those 40 years of age and younger.
- Hilar lymphadenopathy occurs, then progresses to lymphocytic alveolitis. Skin lesions, peripheral lymphadenopathy, interstitial nephritis, iritis, hepatomegaly and splenomegaly can also occur.
- Symptoms and complications are related to malabsorption.
- Can result in pulmonary fibrosis or associated right-sided heart failure (cor pulmonale).

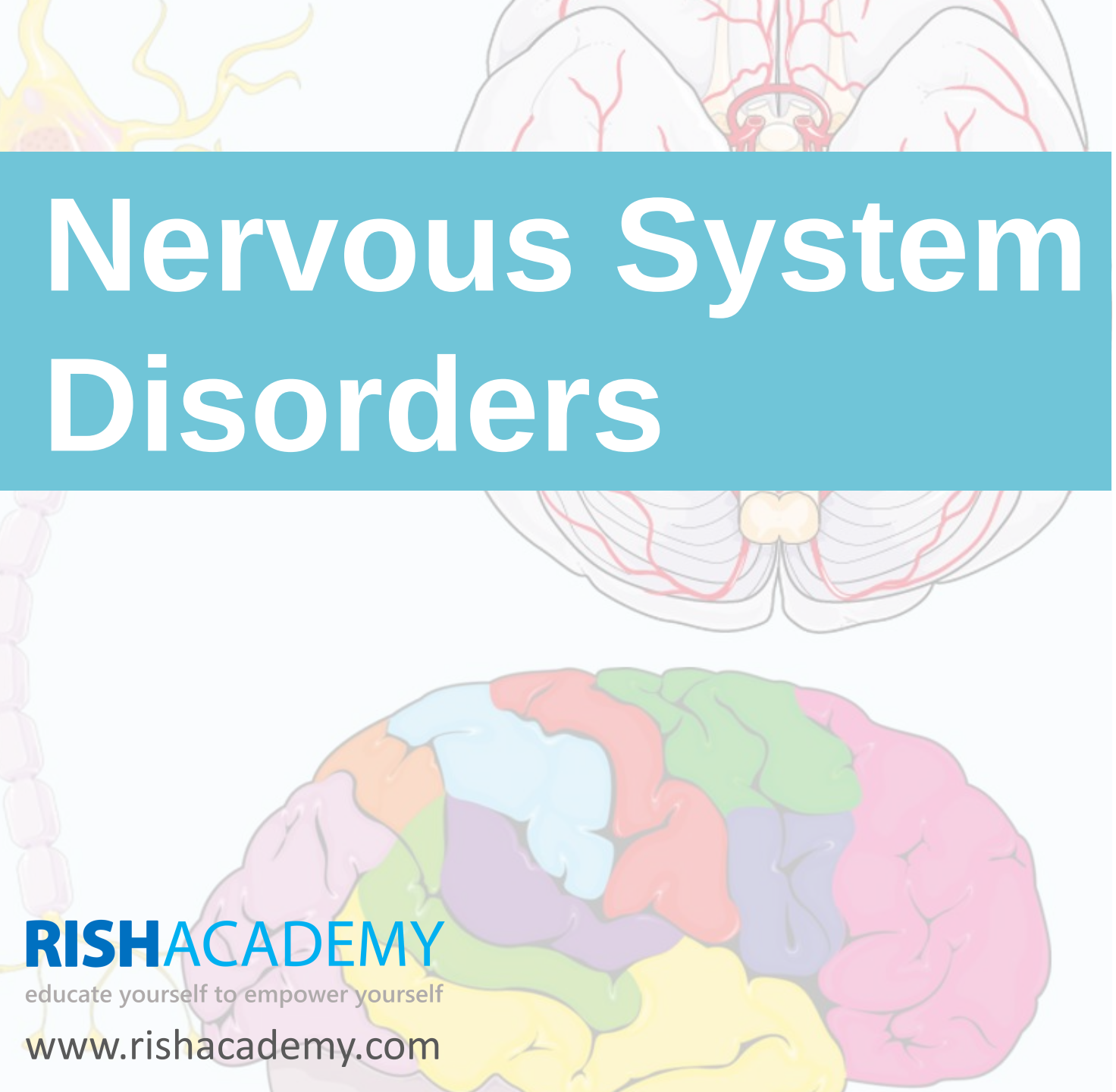
18 Tuberculosis (TB)

- Fatigue
- Sudden Weight loss
- Anorexia
- Night Sweats
- Low-grade fever
- Productive Cough
- Hemoptysis
- Chest pain
- Anxiety

Pathophysiology

- Mode of transmission: The tubercle bacilli are spread by the airborne route. The mycobacteriumcontaining droplet nuclei circulate in the air. A T-cell–mediated response occurs, walling off the lesion (Ghon tubercle), inactivating the disease.
- The Ghon tubercle affects the hilar region first. If the client becomes immunosuppressed, the Ghon necrose cavitates then may release the organism into the lung.

Nervous System Disorders



Clinical Medicine Flashcards

- Clinical Clues to Diagnosis
- Pathophysiology

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- Absence Seizures
- Alzheimer’s Disease
- Amyotrophic Lateral Sclerosis
- Atonic Seizures
- Autonomic Dysreflexia
- Bell’s Palsy
- Cerebral Aneurysm
- Cerebrovascular Accident
- Complex Partial Seizures
- Concussion
- Encephalitis
- Epidural and Subdural Hematoma
- Guillain-Barré Syndrome
- Huntington’s Disease
- Malignant Hyperthermia
- Meningitis
- Multiple Sclerosis
- Myasthenia Gravis
- Myoclonic Seizures
- Parkinson’s Disease
- Simple Partial Seizures
- Skull Fracture
- Spinal Cord Injury
- Spinal Shock
- Tonic-Clonic Seizures
- Trigeminal Neuralgia

1 Absence Seizures

- Usually a disease of children
- The child appears to be daydreaming and has no recall of the event.
- Abnormal EEG.

Pathophysiology

- Generalized seizures that may be due to alternating gamma-aminobutyric acid type B (GABAB) receptor–mediated inhibition alternating with glutamate-mediated excitation.
- Calcium channel abnormalities are also implicated in the genesis of absence seizures.

2 Alzheimer's Disease

- Short-term memory loss
- Forgetfulness
- Confusion
- Inability to recognize loved ones or self

Pathophysiology

- Neuropathologic findings in SDAT include amyloid plaques, neurofibrillary tangles, and synaptic and neuronal cell death.
- Degeneration occurs first in the hippocampus, (short-term memory), then damage spreads to the temporal area. Frontal damage causes personality changes and incontinence.
- Acetylcholine levels in the cerebral cortex become deficient.
- Ventricles of the brain become larger as the brain tissue is destroyed. The brain shrinks in size.

3 Amyotrophic Lateral Sclerosis

- Fasciculations and atrophy of muscle groups with progressive weakness
- A degenerative neuromuscular disease.

Pathophysiology

- Also known as Lou Gehrig's disease, ALS is a progressive neurodegenerative disease.
- The upper and lower motor neurons degenerate and form scar tissue, disrupting nerve transmission and leading to muscle atrophy.
- Results in swallowing and breathing difficulty.
- A genetic link is suspected as the cause. Onset is usually between ages 40 and 70 years and more prevalent in men than in women; survival varies from 3–10 years or more.

4 Atonic Seizures

- Sudden loss of muscle tone causing a “drop attack.”

Pathophysiology

- Classified as a generalized seizure, with juvenile onset lasting into adulthood. The cerebral cortex, subcortical, and spinal areas are believed to be involved in the development of generalized seizures. The electrical discharge from the cortex produces stimulation of the muscles through the motor centers.
- Evidence indicates that an abnormality exists among neurotransmitters (e.g., gammaaminobutyric acid [GABA]) or in calcium, potassium, or sodium channel activity.

5 Autonomic Dysreflexia

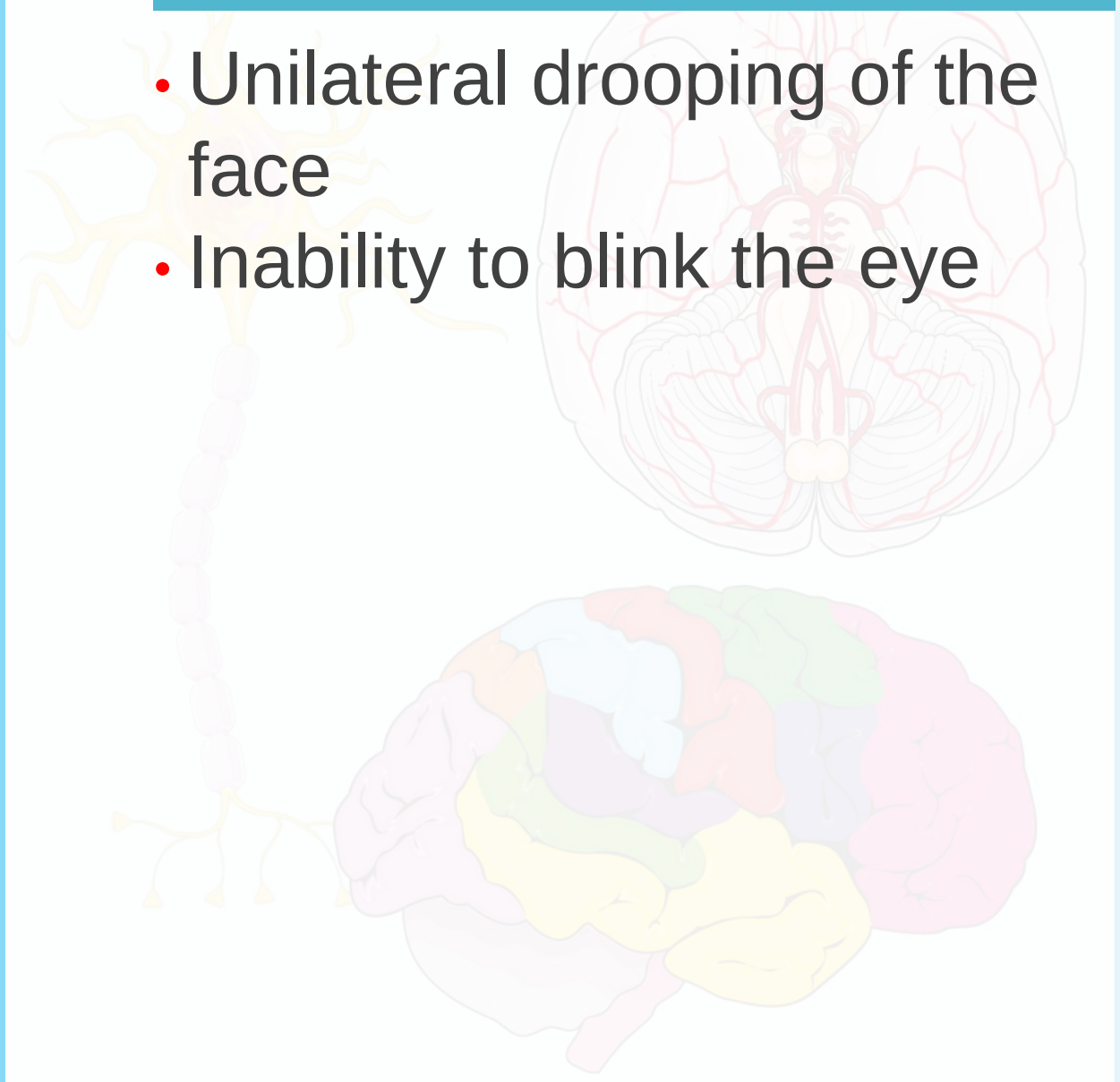
- In spinal cord injuries above T6
  - Sudden onset of headache
  - Nasal stuffiness
  - High BP
  - Flushed skin above the level of injury

Pathophysiology

- SCI above T6 receive peripheral sensory impulses from below the injury via the spinothalamic tract that stimulates a large sympathetic release of norepinephrine, dopamine β-hydroxylase, and dopamine. These neurotransmitters cause vasoconstriction (↑ BP) and skin pallor below the area of injury.
- The brainstem reacts to the ↑ BP by parasympathetic nervous innervation above the level of injury.
- Relieving the noxious stimuli stops the sensory signal and therefore the sympathetic response.

6 Bell's Palsy

- Unilateral drooping of the face
- Inability to blink the eye

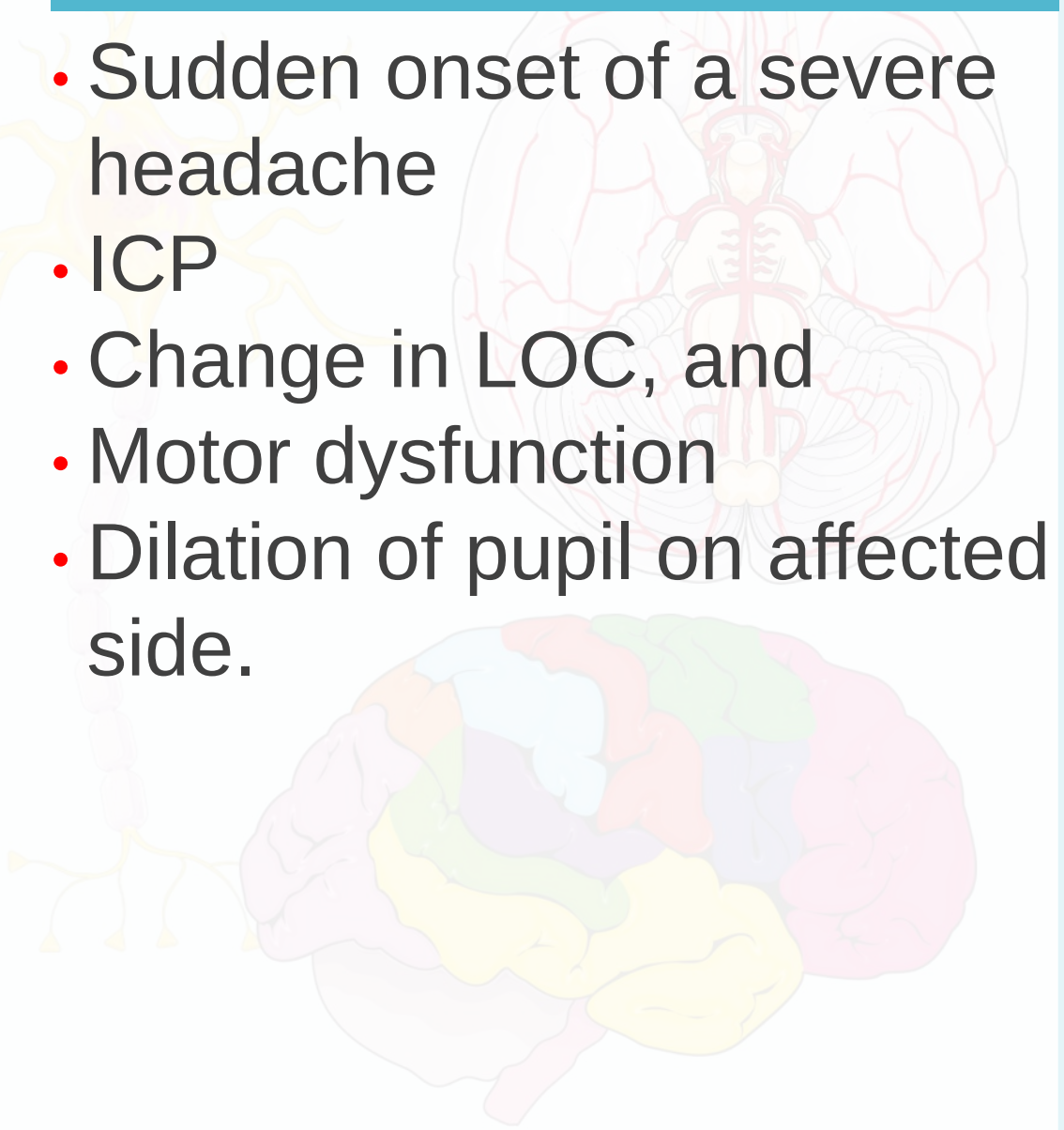


Pathophysiology

- Inflammation thought to be caused by autoimmune, viral, bacterial, or traumatic processes of the seventh cranial nerve (facial nerve), causing interruption of nerve transmission.
- Motor control is lost usually on one side of the face, although it can occur bilaterally (1%).
- More likely to occur in pregnancy, immune dysfunction (e.g., human immunodeficiency virus), or diabetes.

7 Cerebral Aneurysm

- Sudden onset of a severe headache
- ICP
- Change in LOC, and
- Motor dysfunction
- Dilation of pupil on affected side.

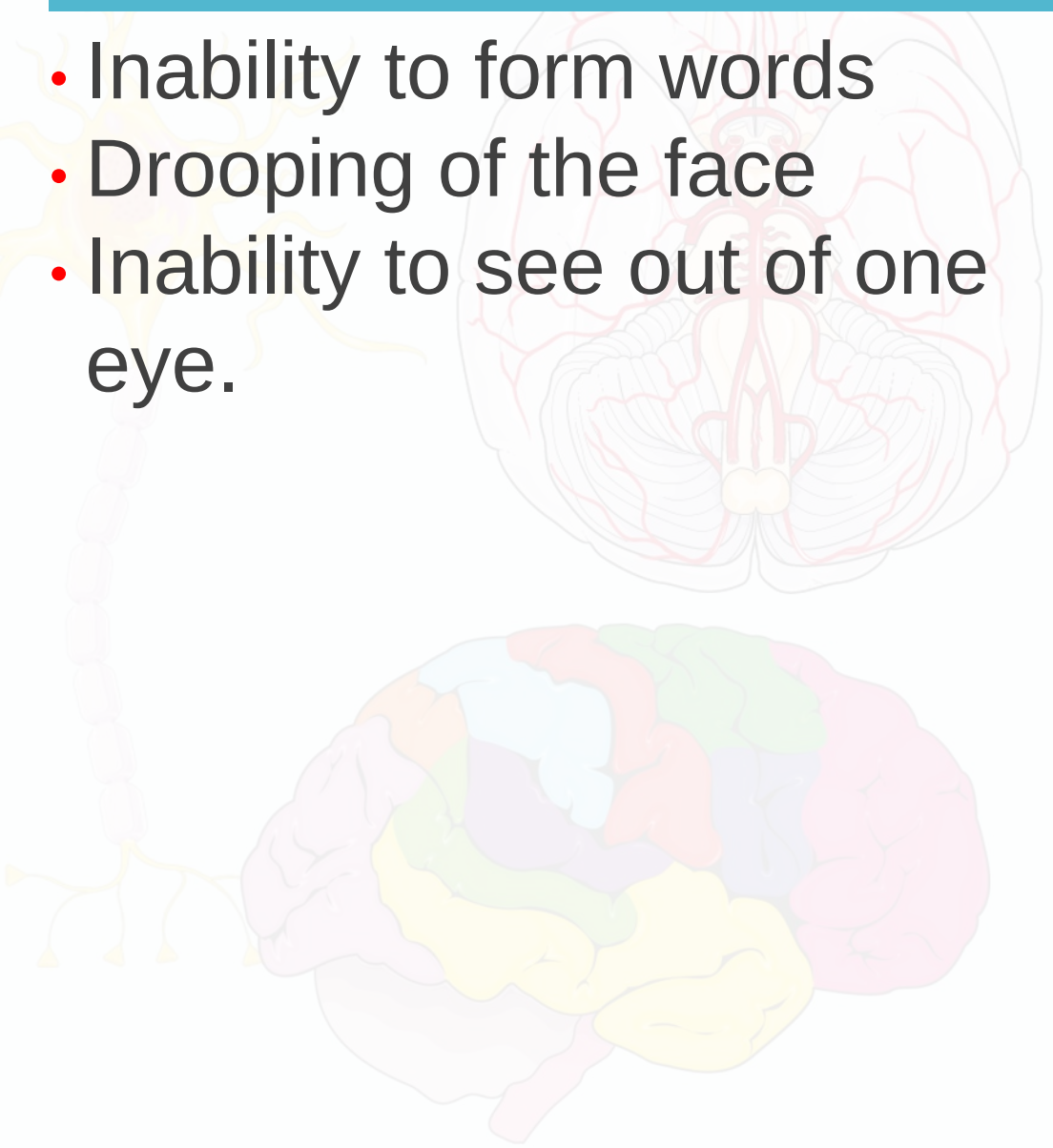


Pathophysiology

- The endothelial lining of vessel walls become damaged, lose elasticity, and become vulnerable to rupture. Contributing conditions include HTN, atherosclerosis, natural presence of arterial bifurcation, and congenital high-pressure areas such as AVMs.
- Ruptured aneurysms have abnormally high levels of inflammatory cell infiltration.

8 Cerebrovascular Accident

- Inability to form words
- Drooping of the face
- Inability to see out of one eye.



Pathophysiology

- Result of a thrombotic block to blood flow or bleeding into the brain that drastically diminishes blood flow to the neurons, causing the cerebrovascular accident (CVA).
- Injured cells fill up with free zinc ions that are believed to hasten their demise.
- Production of glutamate increases the metabolic needs of the already depleted neurons.
- Inflammation causes cerebral edema.

9 Complex Partial Seizures (Psychomotor)

- Staring, running away, picking at clothing, or standing still withnlip smacking or other socially awkward behavior.
- May lose consciousness.

Pathophysiology

- Complex partial seizures arise from one hemisphere of the brain, usually in the temporal lobe.
- May spread and become a generalized seizure.
- Neurotransmitter abnormalities, especially in gamma-aminobutyric acid (GABA), as well as calcium, potassium, or sodium channel abnormalities, may produce the abnormal electrical charge.
- Loss of consciousness occurs and may last 2–15 minutes.

10 Concussion

- A blow to the head resulting in changes in LOC.

Pathophysiology

- Also known as traumatic brain injury, concussion is caused by a nonpenetrating, or closed, head injury. Mechanisms of injury include acceleration injury, deceleration injury, or a combination of both. Rotational injuries cause traumatic shearing of the brain tissue. Most injuries are related to automobile accidents, but playing contact sports and falls are also frequent causes.
- After injury, the compromised cells require increased glucose in order to remain alive. However, with the onset of cerebral edema, the capillary bed may become displaced from the cells by fluid. With decreased blood flow, neuronal loss can continue to occur.

11 Encephalitis

- Inflammation of the brain leading to ICP
- most frequently caused by viruses (e.g., West Nile virus), parasites,
- toxins, bacteria, vaccines, or fungi.

Pathophysiology

- Neurons are damaged and inflamed, leading to cerebral edema and increased ICP.
- Causative agents are viruses, ticks, mosquitoes, parasites, toxins, bacteria, vaccines, or fungi. Those with a compromised immune system, the very young, and the very old are especially at risk.
- Herpes simplex virus may be the most common non-insect-borne cause of the disease.

12 Epidural and Subdural Hematoma

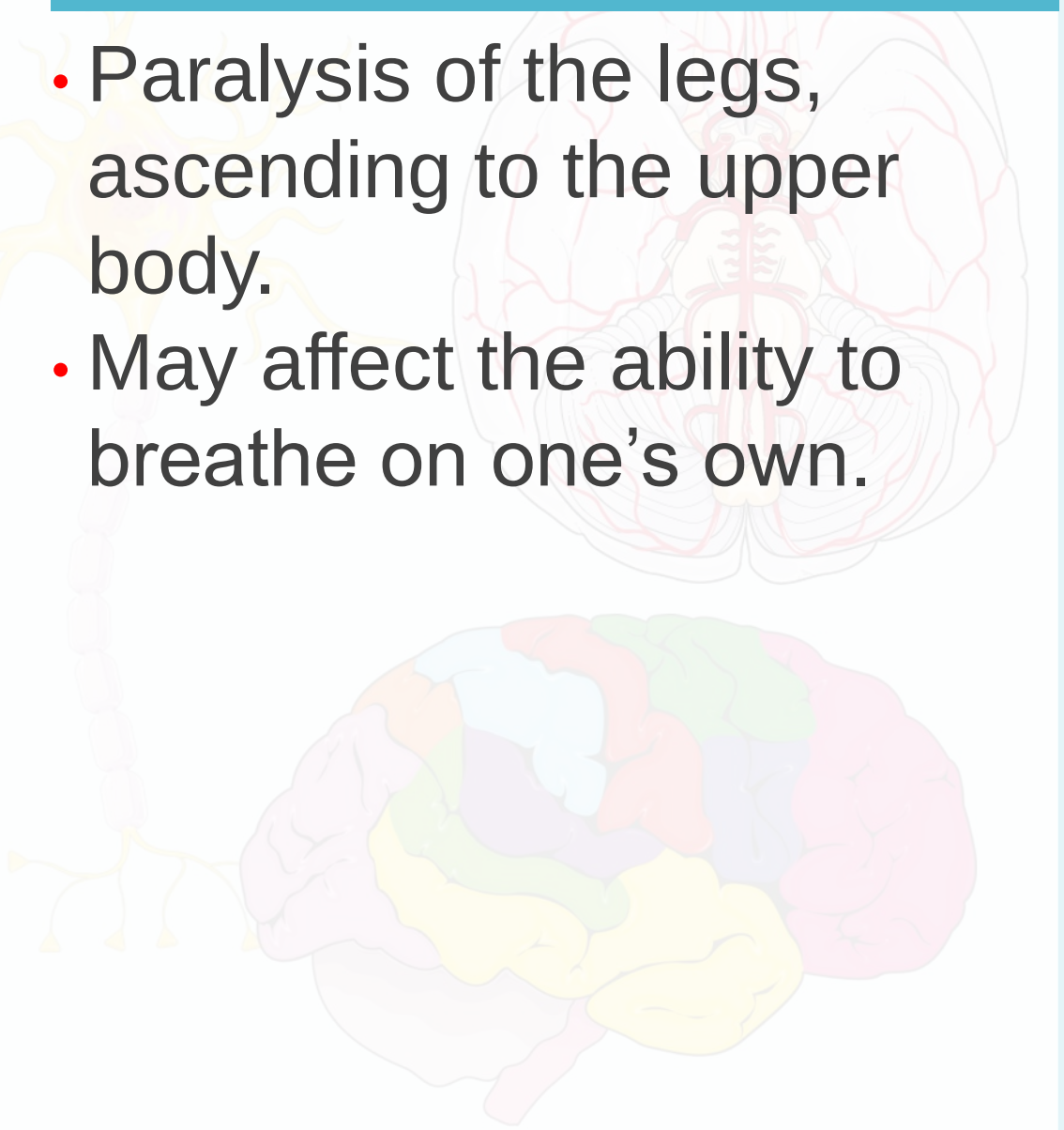
- Head injuries that may be arterial or venous in nature, causing ICP and change in LOC.
- May cause rapid ICP or be insidious, chronic, or become fatal.

Pathophysiology

- Traumatic brain injury that is nonpenetrating and caused by rotational injury, acceleration injury, deceleration injury, or both.
- Epidural hematomas are arterial, so symptoms are more severe due to rapid accumulation of blood above the dural layer.
- Subdural hematomas are venous, so symptoms may be more insidious.
- These hematomas may occur together.

13 Guillain-Barré Syndrome

- Paralysis of the legs, ascending to the upper body.
- May affect the ability to breathe on one's own.



Pathophysiology

- GB syndrome (inflammatory polyneuritis), is an inflammatory disorder characterized by a distinct progression of paralysis. GB often follows a viral infection. Occurs in those older than age 45 years and with higher frequency in Caucasians than in African Americans.
- Peripheral nerves are infiltrated by immune cells that lead to inflammation and demyelination of the axon. Paralysis begins in the legs and ascends. If the disease reaches the lungs, respiratory support is required. The plateau stage is the most severe, but it signals the end of the progression; remyelination occurs and the symptoms regress.
- A descending form of the disease exists.
- Miller-Fisher syndrome causes ataxia and extra ocular paralysis but no respiratory or sensory loss.

14 Huntington's Disease

- Onset of jerking movements of the upper extremities, face, and neck progressing to the rest of the body accompanied by progressive psychotic behavior.
- Genetic testing reveals mutation of the IT15 gene resulting in huntingtin protein.

Pathophysiology

- A disorder that causes a mutation in the IT15 gene that results in transcription of an abnormal protein called huntingtin protein. Abnormalities in DNA trigger cellular death. Cells affected are those that control motor and cognitive function.
- An autosomal dominant disorder; each offspring of an affected parent has a 50% chance of inheriting the disorder.
- Onset may be in childhood or in midlife.

15 Malignant Hyperthermia

- Temperature increase and muscle rigidity following exposure to anesthetics.
- Creatinine and BUN levels increase.
- Urine is brown in color.

Pathophysiology

- Autosomal dominant disorder in which exposure to a certain anesthetic agent causes temperature increase and muscle rigidity.
- Affects skeletal muscle tissue primarily. Free ionized calcium concentration can increase to damaging levels and cause multiple contractions of skeletal muscle. The temperature may rise to over 105°F from repeated contractions.
- The hypermetabolic state causes lactate formation, resulting in acidosis.

16 Meningitis

- Nuchal rigidity and pain as the meninges are stretched by moving the legs or flexing the neck to the chin.
- Turbid CSF with low glucose and high protein.

Pathophysiology

- The meninges include the dura matter, arachnoid layer, and pia matter; and surround the brain and spinal cord.
- Causative agents include bacteria, viruses, mycobacteria, fungi, amebas, cancer, and noninfectious sources. Entrance via the respiratory system is most common.
- Infections that occur close to the CNS, basilar fracture, CNS surgery or presence of an indwelling shunt, and blood-borne illnesses cause meningitis.
- TNF-α and IL-1 are major mediators of inflammation that increase permeability and transit of the causative agent through the blood–brain barrier.
- Inflammation causes ↑ ICP in meningitis.

17 Multiple Sclerosis

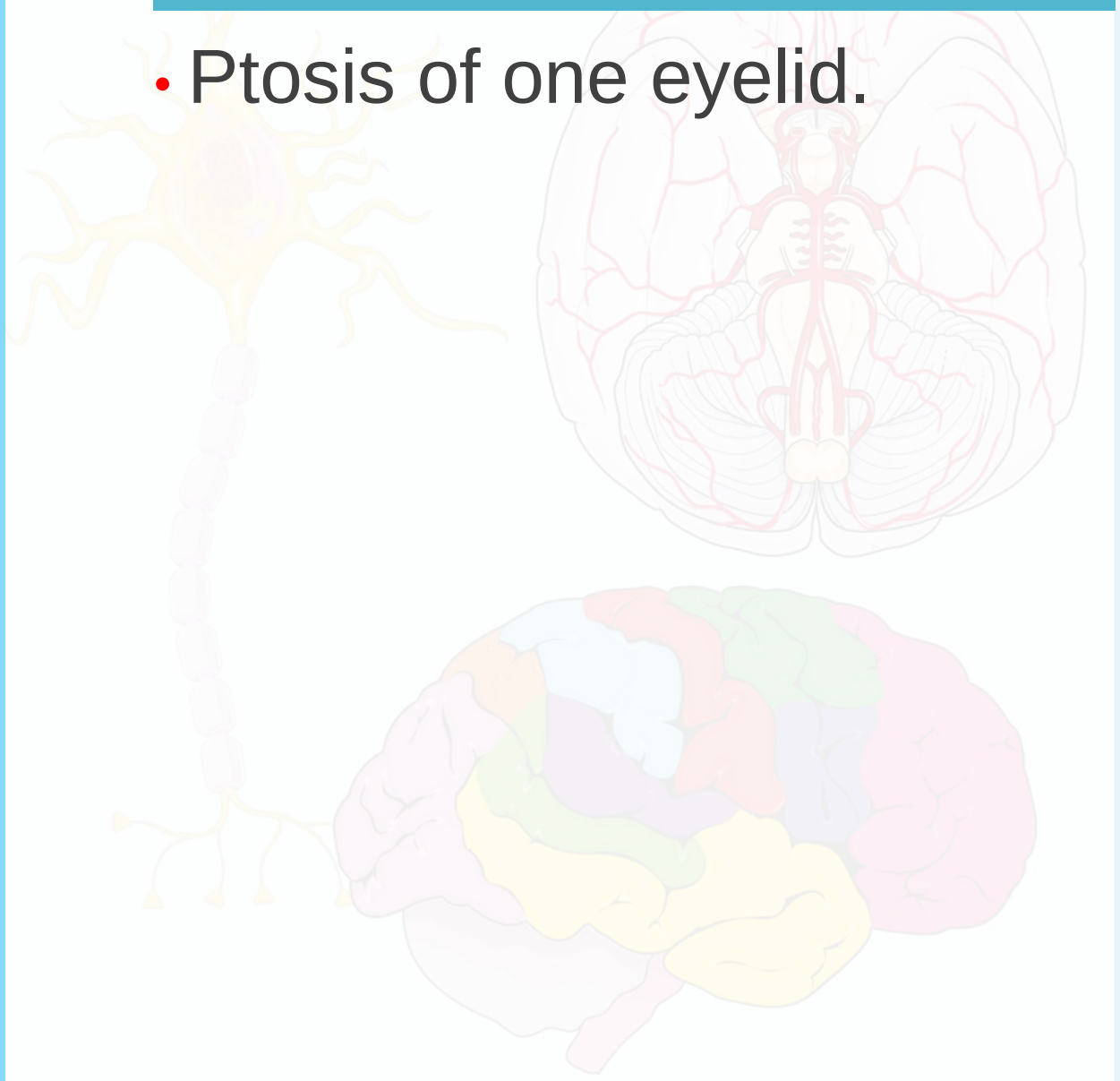
- Exacerbating and remitting periods of degenerating motor function.
- The MRI shows demyelination of the white matter of the brain.

Pathophysiology

- Exacerbating and remitting disease characterized by demyelination of brain white matter, damage to axons, and decreased number of oligodendrocytes in the CNS affecting young adults (ages 20–40) and women more than men.
- Autoimmune inflammatory disease involving cell-mediated (T-cell) and antibody (B-cell) activity.
- Exacerbation of symptoms can be caused by extreme heat or cold, fatigue, infection, stress, or pregnancy.

18 Myasthenia Gravis

- Ptosis of one eyelid.

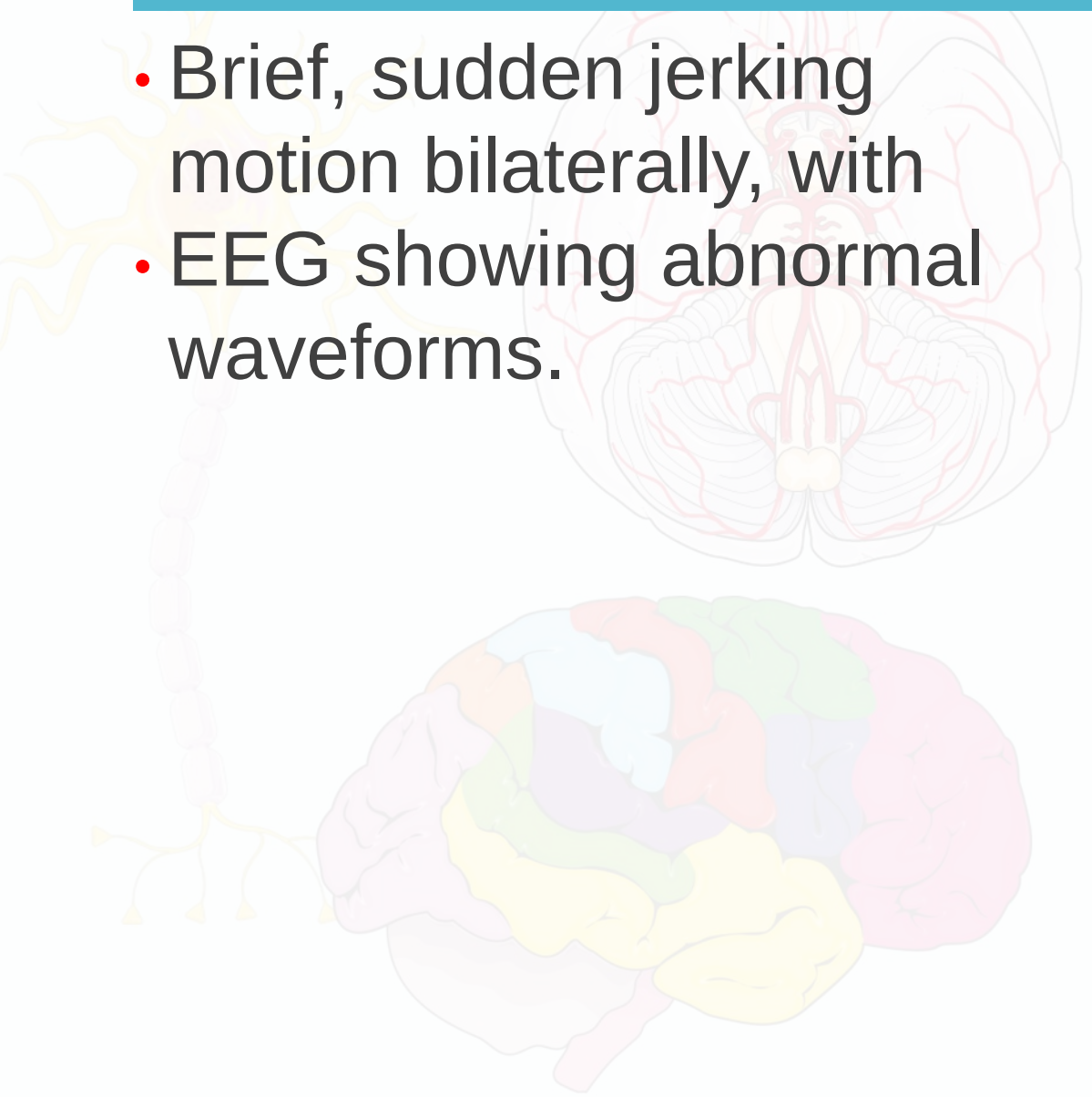


Pathophysiology

- Meaning “grave muscle weakness,” MG is an autoimmune disease that produces antibodies that attack AChR in the NMJ of skeletal muscles.
- ACh molecules are inactivated by the enzyme AChE, which is abundantly present at the NMJ.
- The disease involves periods of exacerbation and remission.

19 Myoclonic Seizures

- Brief, sudden jerking motion bilaterally, with
- EEG showing abnormal waveforms.

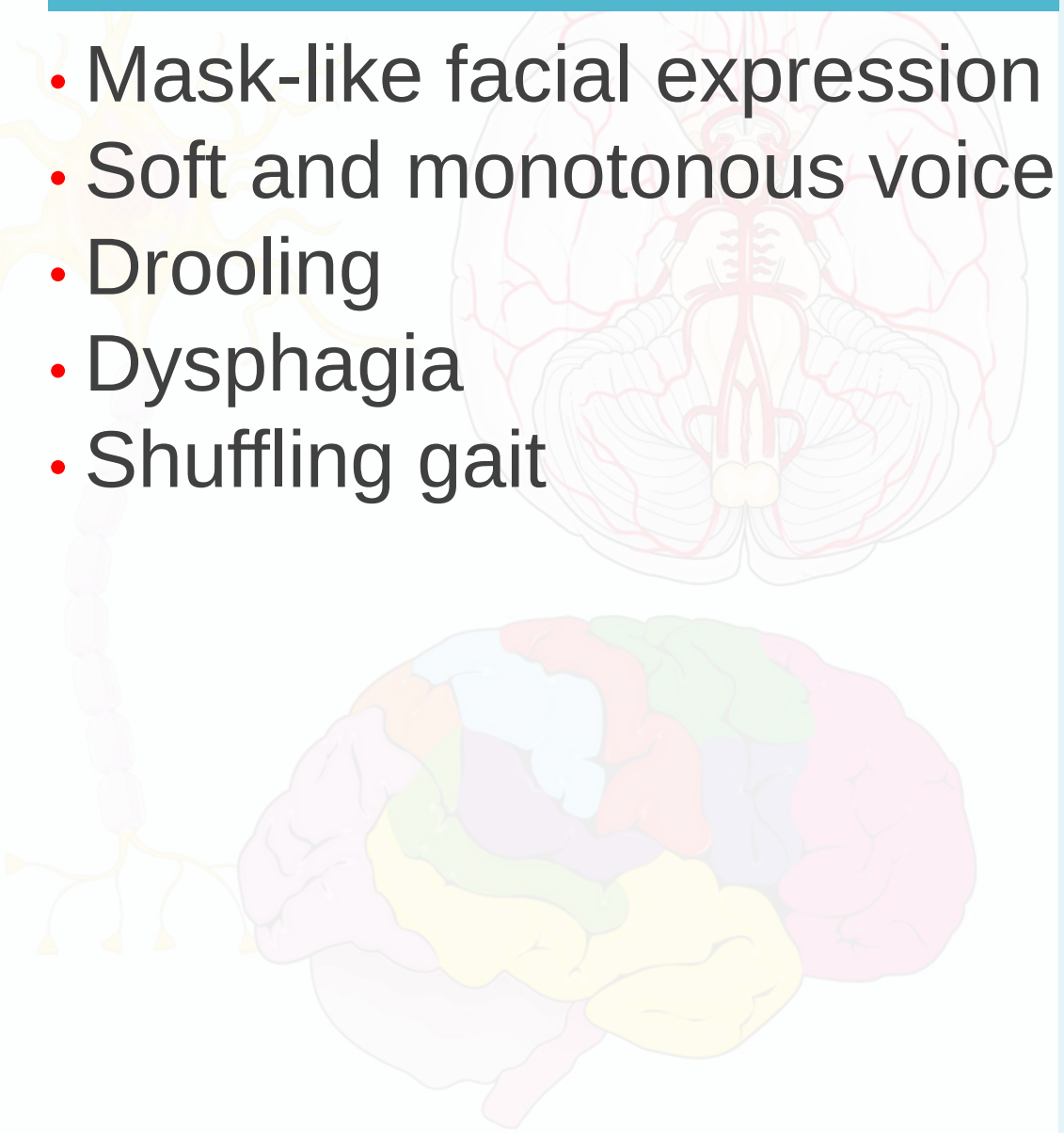


Pathophysiology

- A generalized seizure with juvenile onset. The cerebral cortex, subcortical, and spinal areas are believed to be involved in the development of myoclonic seizures. The electrical discharge from the cortex produces stimulation of the muscles through the motor centers.
- Acetycholine (excitatory), serotonin (inhibitory), and GABA (inhibitory) neurotransmitters are present in mismatched amounts in this disorder.
- The mechanism of myoclonic seizure disorder is related to restless leg syndrome.

20 Parkinson's Disease

- Mask-like facial expression
- Soft and monotonous voice
- Drooling
- Dysphagia
- Shuffling gait



Pathophysiology

- Under the cerebral cortex are interconnected areas of gray matter (basal ganglia), which are involved in controlling voluntary movement. Adjacent to the basal ganglia are cells of the substantia nigra that produce the neurotransmitter dopamine necessary to produce smooth and coordinated muscle movement. Death of cells in the substantia nigra leads to decreased levels of dopamine production, and impairment of EP tract.
- As dopamine levels decrease, acetylcholine levels increase.

21 Simple Partial Seizures (Focal)

- Lip smacking, picking at clothing, or chewing behaviors of which the client is unaware.
- Consciousness is not lost.

Pathophysiology

- Simple partial seizures, or focal seizures, arise from one hemisphere of the brain, usually in the temporal lobe.
- May spread to the parietal lobe, causing transient paresthesias of the body on the opposite side of the epileptogenic focus, usually beginning in the finger, arm, and hand and then spreading to the leg and face.
- Neurotransmitter abnormalities, especially in gamma-aminobutyric acid (GABA), as well as calcium, potassium, or sodium channel abnormalities, may produce the abnormal electrical charge.
- Simple partial seizures may spread and become a generalized seizure.

22 Skull Fracture

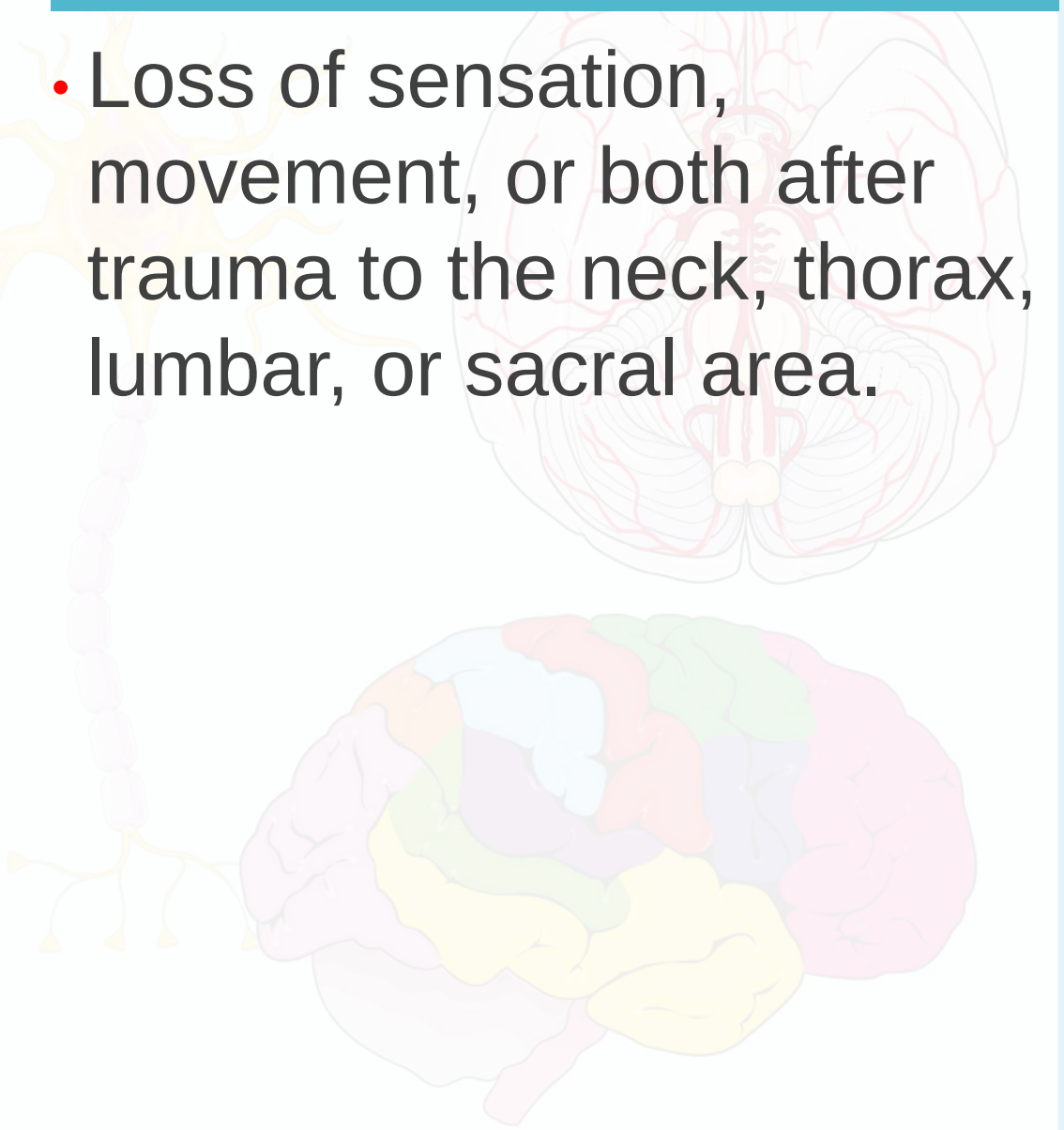
- Severe head trauma resulting in an area of open-skull injury that presents as
  - Crepitus
  - CSF leak, or a
  - Depressed area in the skull.

Pathophysiology

- Loss of integrity of the cranial bones and/or meninges causing damage to the underlying brain tissue and creation of an avenue of infection to the CNS.
- Types include linear, comminuted, depressed, compound, and basilar.
- Acute cerebral edema and ICP occur from neuronal damage, hemorrhage, inflammation, infection, potassium leaking to the extracellular space, and lactate buildup from glycolysis.

23 Spinal Cord Injury

- Loss of sensation, movement, or both after trauma to the neck, thorax, lumbar, or sacral area.



Pathophysiology

- Nerve fibers of the spinal cord are nonregenerative.
- Central cord syndrome results in weakness or paralysis that affects the upper extremities more than the lower extremities.
- Anterior cord syndrome, caused by trauma or ischemia results in weakness and decreased pain and temperature sensation below the damaged area.
- Posterior cord syndrome causes ataxia, but strength and sensation are preserved.
- Brown-Séquard syndrome results in paralysis on the affected side and sensation loss on the opposite side of injury.
- Cauda equina syndrome results in bowel and bladder dysfunction and some leg paresthesia.

24 Spinal Shock

- Period of time after SCI in which there is no motor or sensory transmission.
- Can last a day to several months.

Pathophysiology

- SCI causes a concussion-like injury to neurons known as spinal shock in which neurons below the level of the SCI are incapable of any sensory or motor transmission.
- Cytokines cause an inflammatory condition in the affected neurons.
- Phase 1 spinal shock is characterized by the absence of all reflex arcs below the SCI.
- Phase 2 spinal shock is characterized by the return of some of the reflex arcs, which signals the beginning of the end of spinal shock.
- Phase 3 and 4 are characterized by strong reflexes that occur with minor stimulation and may be followed by autonomic dysreflexia, hyperreflexia, and clonus.

25 Tonic-Clonic Seizures

- Presence of an aura followed by loss of consciousness with alternating cycles of stiffness and jerking movements lasting 1–2 minutes.

Pathophysiology

- Tonic-clonic seizures are generalized seizures.
- Pathology includes an area of hyperexcitable neurons. This is the epileptogenic focus.

26 Trigeminal Neuralgia

- Severe knife-like facial pain unilaterally in response to movement of the musculature of the face, a touch, or cool breeze.

Pathophysiology

- Vascular compression or other structural disorders of the vasculature cause inflammation of the fifth cranial nerve, or TN.
- Inflammation and compression cause demyelination and remyelination of the nerve. This abnormal myelination causes abnormal sensory discharge, felt by the client as intense pain.
- Trigger zones include the lips, upper or lower gums, cheeks, forehead, and side of the nose.

Endocrine System Disorders

Clinical Medicine Flashcards

- Clinical Clues to Diagnosis
- Pathophysiology

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- Addison’s Disease
- Cushing’s Syndrome
- Diabetes Insipidus
- Diabetes Mellitus Type 1
- Diabetes Mellitus Type 2
- Hyperpituitarism
- Hypopituitarism
- Hyperthyroidism
- Hypothyroidism
- Syndrome of Inappropriate Antidiuretic Hormone

1 Addison's Disease

- Tanned appearance to skin
- Low blood pressure,
- ↓ serum glucose,
- ↓ serum sodium (Na+),
- ↑ serum Potassium (K+)

Pathophysiology

- The adrenal cortex secretes hormones necessary to react to stress (physical or psychological). They include glucocorticoids, aldosterone, and sex hormones (sugar, salt, and sex).
- In primary hypofunction of the adrenal gland, the adrenal hormones are not secreted in adequate amounts; in secondary hypofunction of the adrenal glands, insufficient pituitary secretion of ACTH occurs.
- Primary Addison's disease may be autoimmune.
- Adrenalectomy may cause Addison's.

2 Cushing's Syndrome

- Moon face
- Buffalo hump
- Truncal obesity
- ↑ serum glucose
- ↓ potassium (K+)
- ↑ serum sodium

Pathophysiology

- Hormones secreted by the adrenal cortex are the body's stress hormones (glucocorticoids and mineral corticoids).
- Cushing's disease is caused by excess cortisol secretion related to excess ACTH secretion, while Cushing's "syndrome" is related to consumption of exogenous cortisol.
- May be caused by secreting tumor of the lungs or adrenal glands.

3 Diabetes Insipidus

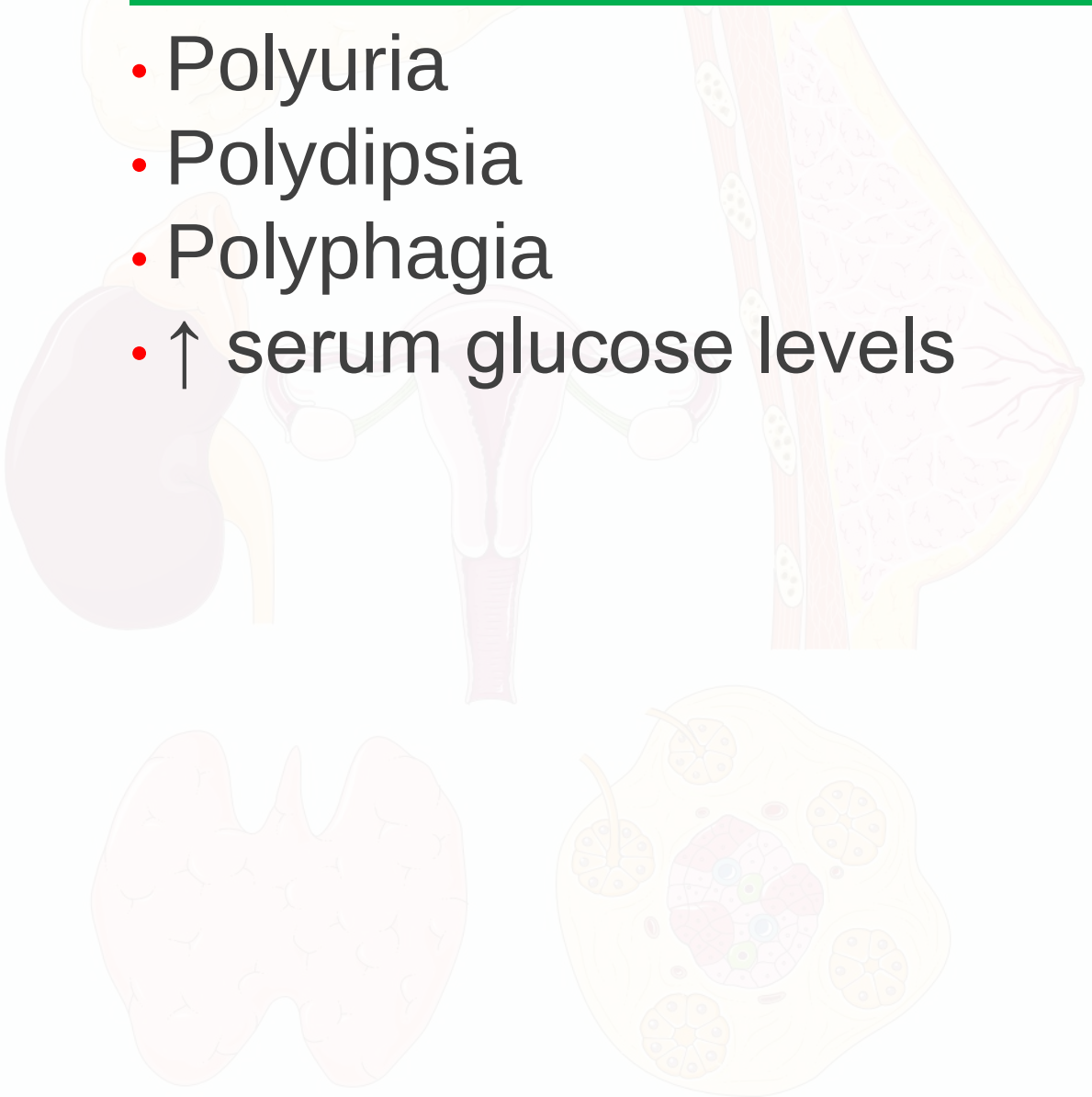
- Low specific gravity of urine <1.005
- Urinary output in excess of 5–15 liters daily

Pathophysiology

- ADH or AVP is secreted by the posterior pituitary gland and is responsible for reabsorption of water by the kidney.
- DI is caused by a deficiency of ADH and excess loss of water through urination. Urinary output can be in excess of 5–15 L daily.
- DI can be caused by drugs like lithium; surgical removal of the pituitary; and nephrogenic DI.
- Psychogenic DI (not true DI) is caused by a desire to drink large amounts of fluids (water intoxication); alcohol ingestion causes a temporary DI resulting in dehydration.

4 Diabetes Mellitus Type 1

- Polyuria
- Polydipsia
- Polyphagia
- ↑ serum glucose levels



Pathophysiology

- The pancreas secretes insulin from the beta cells (islets of Langerhans) in response to elevated blood glucose levels.
- In primary diabetes mellitus type 1, the beta cells are destroyed by an autoimmune reaction.
- In secondary diabetes mellitus type 1, the beta cells are damaged by cancer of the pancreas or other diseases like pancreatitis and cystic fibrosis.

5 Diabetes Mellitus Type 2

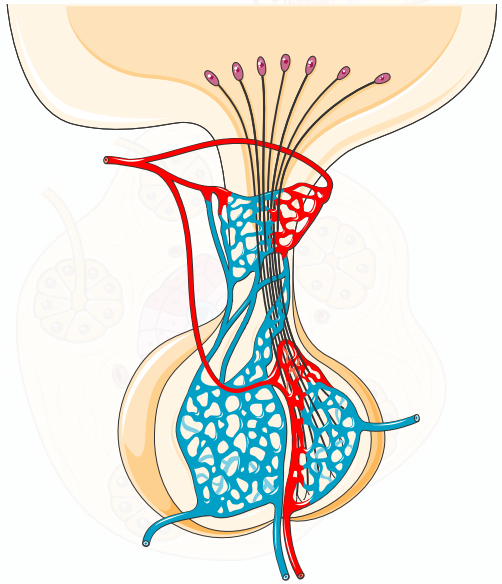
- Sedentary lifestyle
- Polyuria
- Polyphagia
- Polydipsia
- Elevated serum glucose

Pathophysiology

- Type 2 diabetes increases in incidence with obesity, poor diet, and sedentary lifestyle as the cells of the body become resistant to insulin.
- Genetic link (10 new gene variants that affect blood glucose and insulin levels have been identified); type 2 diabetes is affecting more children related to poor diet and obesity.

6 Hyperpituitarism

- Excess growth hormone
- Abnormal lipid level
- High blood glucose levels
- Adults experience arthritis
- Visual changes
- Enlarged hands & feet

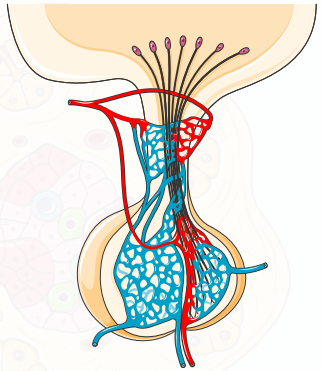


Pathophysiology

- Hyperfunction of the pituitary is almost always caused by an adenoma.
- GH, from the anterior pituitary, is secreted in large amounts, resulting in gigantism in children and acromegaly in the adult.
- Acromegaly is characterized by growth of bone, connective, and soft tissue.
- Hands and feet become enlarged; larynx enlarges; vertebral growth often results in kyphosis; teeth become displaced; enlargement and erosion of the sella turcica causes visual changes and headache.
- Metabolic alteration causes fats to become the initial energy burned, resulting in ketosis.
- GH–induced insulin resistance, along with glycogen release by the liver, causes DM.
- Other anterior pituitary hormones are inhibited.
- Fatty acid metabolism is altered causing atherosclerosis.
- Excess soft tissue of the soft palate cause sleep apnea.

7 Hypopituitarism

- Short stature in children accompanied by
  - Weakness
  - Low blood glucose
- Delayed sexuality
- Stunted growth of sexual organs.

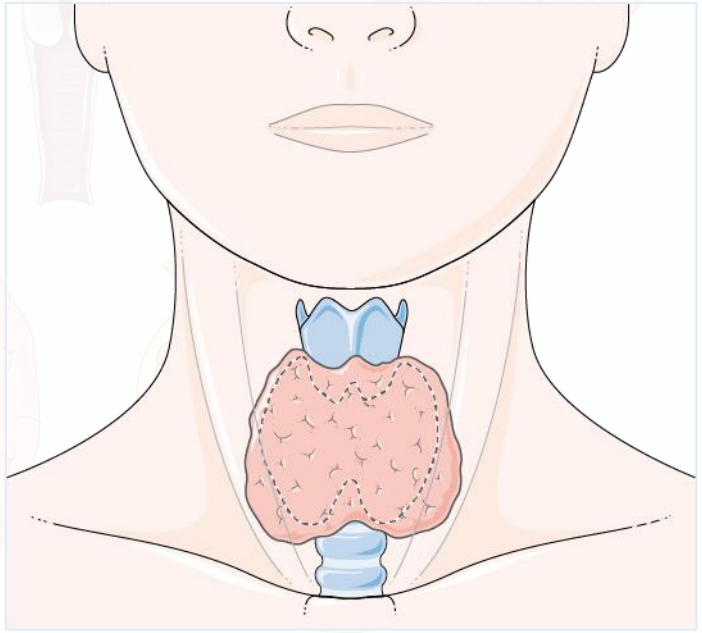


Pathophysiology

- Growth hormone (somatropin) is deficient related to an ablative pituitary tumor or failure of the gland to develop.
- Dwarfism, in the child, and mental slowness.
- In adults, decreased GH leads to central accumulation of body fat and related problems with cardiovascular health.

8 Hyperthyroidism

- Restlessness,
- Irritability,
- Heat intolerance,
- ↓ TSH,
- ↑ T3 and T4.

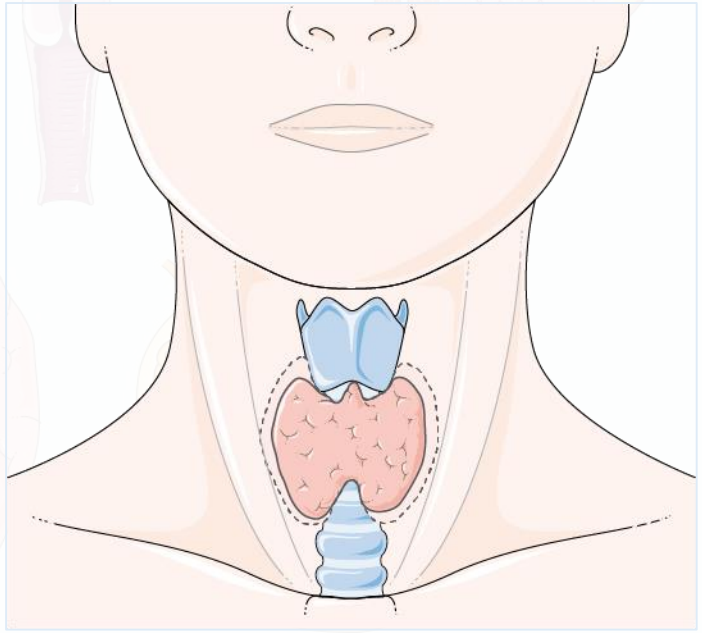


Pathophysiology

- The thyroid gland hormones are responsible for carbohydrate, protein, and fat metabolism required by the body cells and for calcium regulation (in tandem with the parathyroid glands).
- Primary hyperthyroidism (Graves' disease) is caused by excess thyroid hormone secretion (T3 and T4).
- Secondary hyperthyroidism is caused by hypersecretion of thyroid-stimulating hormone (TSH) by the pituitary gland.
- A thyroid tumor may also cause hypersecretion of thyroid hormones or TSH.
- Exposure to radiation is another causative factor.

9 Hypothyroidism

- Lethargy
- Mental slowness
- Menorrhagia
- ↑ TSH
- ↓ T3 and T4



Pathophysiology

- The thyroid gland is responsible for metabolism of carbohydrates, fats, and protein according to body requirements.
- Primary hypothyroidism (myxedema) occurs when the thyroid gland does not secrete adequate thyroid hormone.
- Secondary hypothyroidism is related to hyposecretion of thyroid-stimulating hormone (TSH) by the pituitary gland or overtreatment of hyperthyroidism.
- Low levels of thyroid hormone decrease metabolism in the body.
- Hashimoto’s thyroiditis is an autoimmune disorder that destroys thyroid tissue.

10 Syndrome of Inappropriate Antidiuretic Hormone

- High blood pressure
- Low serum osmolality
- Bounding pulse
- Seizures.

Pathophysiology

- SIADH occurs when ADH does not decrease in response to a low serum osmolality, leading to fluid overload.
- Frequently, SIADH is associated with cancers of the lung, pancreas, and Hodgkin’s disease.
- ADH may hypersecrete in the presence of head trauma or tumor or as a complication of diabetes insipidus treatment.

Gastrointestinal System Disorders

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Clinical Medicine Flashcards

- Clinical Clues to Diagnosis
- Pathophysiology

- Abdominal Hernias
- Appendicitis
- Bowel Obstruction
- Celiac Disease
- Cholecystitis
- Colon Cancer
- Crohn’s Disease
- Diverticulosis
- Esophageal Varices
- Gastric Cancer
- Gastritis
- Gastroesophageal Reflux Disease
- Hemorrhoids
- Hepatitis
- Histal Hernia
- Laënnec’s Cirrhosis
- Liver Cancer
- Pancreatic Cancer
- Pancreatitis
- Peptic Ulcer Disease
- Peritonitis
- Ulcerative Colitis

1 Abdominal Hernias

- Abdominal area that bulges out, especially when intra-abdominal pressure is ↑.
- Gentle pressure can cause reduction or popping back of the abdominal contents.

Pathophysiology

- A weakness in the abdominal wall allows the bowel or omentum, along with peritoneal tissue, to herniate outward. Weakened areas include the umbilical area, along the linea alba, incisional areas, and areas that have not completely closed after birth (the inguinal rings).
- Umbilical hernias are caused by incomplete closure of the umbilical orifice and commonly occur in children and obese clients.
- Ventral hernias are caused by weakness in the linea alba and are aggravated by obesity.
- Inguinal hernias are caused in both males and females by incomplete closure of the inguinal rings.
- Incisional hernias occur after underlying muscle is cut and scar tissue forms, weakening the area.

2 Appendicitis

- Shifting pain & Rebound tenderness at McBurney point
- ↑ WBC.

Pathophysiology

- Inflammation of the appendix by obstruction, usually by fecalith. The small stones may enter, causing more inflammation by exerting pressure and abrasiveness on the walls of the appendix.

3 Bowel Obstruction

- High-pitched bowel sounds
- Abdominal distention
- Pain.

Pathophysiology

- Mechanical obstruction occurs when a tumor or hard stool lodges in the intestine or when the bowel twists (volvulus) or telescopes within itself (intussusception). Pressure builds on the walls of the intestine, decreasing perfusion, which can lead to necrosis of the bowel.
- Adhesions from prior abdominal surgeries cause scar tissue causing mechanical obstruction.
- Paralytic obstruction results from a temporary cessation in nerve conduction.

4 Celiac Disease

- Bloating
- Diarrhea
- Rashes
- Anemia
- + Hydrogen breath test
- Malnutrition
- Failure to thrive

Pathophysiology

- Malabsorption disorder caused by antibody response to gluten or gliadin proteins in barley, rye, oats (some), and wheat (BROW) affecting up to 1% of U.S. population.
- Damages small intestinal villi; prevents fat, iron, calcium, and B-vitamin absorption.
- Genetic link that is not well understood, but human leukocyte antigen (HLA), of which the cell surface receptor type protein human leukocyte antigen (HLA-DQ) is one, is found in family members with a high incidence of celiac disease; pediatric and adult clients affected.

5 Cholecystitis

- Right upper quadrant pain that radiates to the right scapula.
- Murphy's sign is present.
- ↑ Amylase and bilirubin.

Pathophysiology

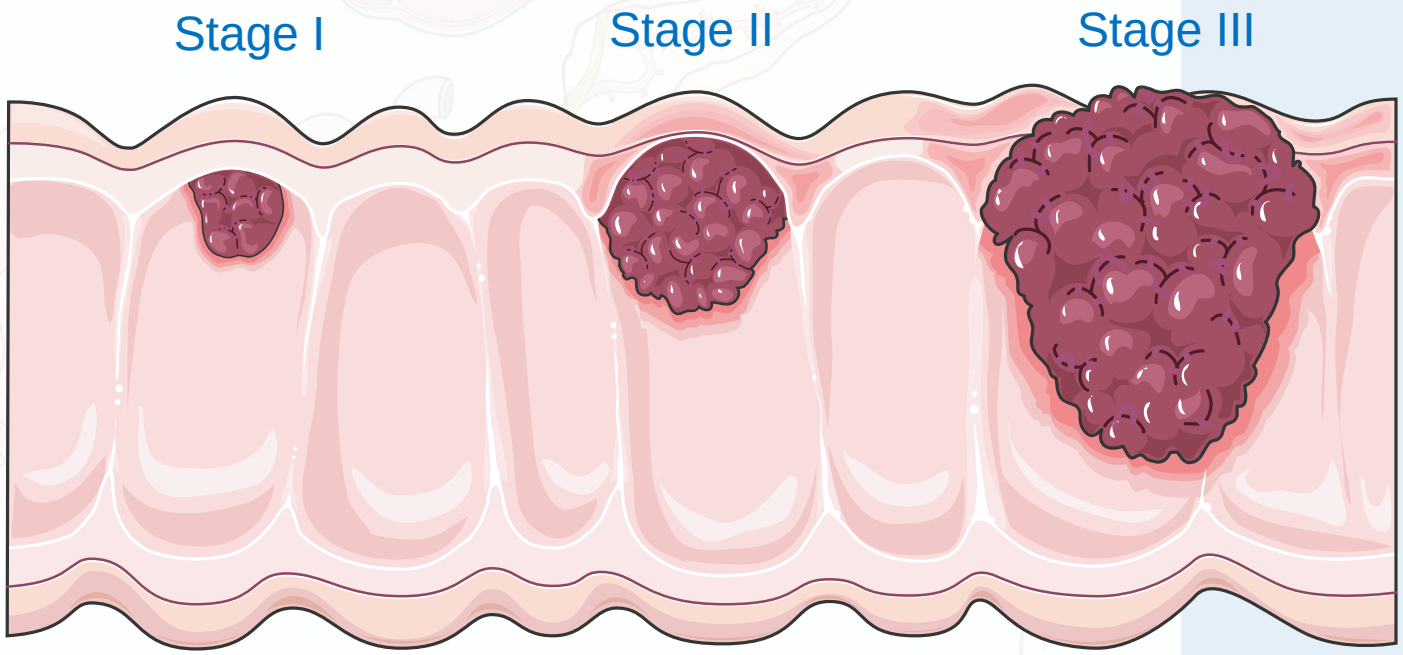
- Presence of gallstones causes mechanical obstruction of bile from the gallbladder. Stasis of bile attracts bacteria, which adds to the inflammation.
- Small gallstones enter the cystic duct and cause severe colicky pain as the duct's peristaltic waves press on the stone.
- The gallbladder becomes fibrotic and does not release bile effectively into the duodenum.
- Pressure of gallstones on the gallbladder walls can cause necrosis.

6 Colon Cancer

- Stool is positive for blood
- Change in bowel habits

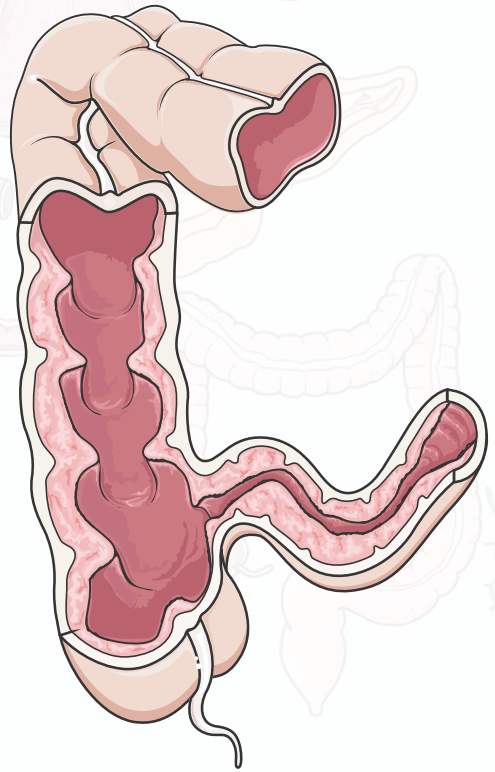
Pathophysiology

- Mutation of epithelial cells of the colon from the chronic irritation of inflammatory bowel disease, familial adenomatous polyposis (FAP; early onset of polyps in the colon that become malignant), removal of the gallbladder, increased fat in the diet, and ingestion of carcinogens.



7 Crohn's Disease

- Diarrhea
- Weight loss
- Cobblestone appearance in the small bowel.



Pathophysiology

- Inflammatory bowel disease affecting mostly women from adolescence to the third decade of life.
- Cobblestone appearance of the bowel wall related to interspersed areas of inflammation and healthy tissue; also called regional enteritis.
- Inflammation occurs mainly in the small intestine above the cecum and spreads proximally.
- Affects the submucosa, causing strictures, scarring, fissures, and fistulas.
- Disease involves both genetic and autoimmune factors.
- Crohn's disease affects the entire bowel wall.

8 Diverticulosis

- History of constipation
- Poor bowel habits.

Pathophysiology

- Small herniations of the sigmoid and descending colon that occur when pressure within the bowel and abdomen is high.
- Related to poor bowel habits, constipation, and straining at stool.
- When diverticula become inflamed by seeds or other residue entering them, diverticulitis results.

9 Esophageal Varices

- Oral hemorrhage in the presence of portal hypertension.

Pathophysiology

- Dilation of the veins of the esophagus occurs related to portal hypertension from chronic liver disease.
- The walls of the veins become thin and can spontaneously rupture and cause massive bleeding.
- Ingestion of fibrous or fried foods can scratch and rupture the varices.

10 Gastric Cancer

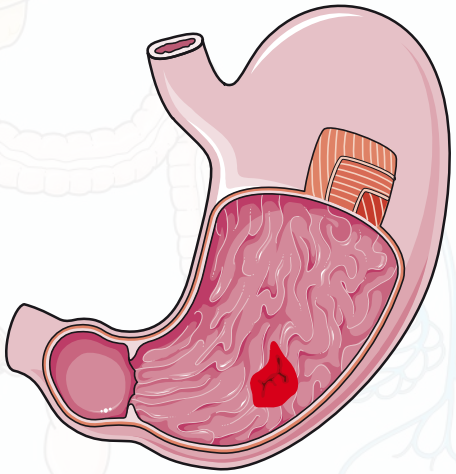
- Indigestion
- Anorexia
- Weight loss
- Nausea &Vomiting
- Pain relieved by antacids
- Anemia
- Melena

Pathophysiology

- Epithelial cells undergo mutation related to chronic irritation or exposure to carcinogens. Cells that are damaged must be replaced. The chance of a mutation occurring is proportional to the rate of new cell growth.
- Implicated causes are chronic or autoimmune gastritis; exposure to lead dust, grain dust, glycol ethers, or leaded gasoline; or a diet high in smoked fish or meats.

11 Gastritis

- Epigastric burning or discomfort associated with
  - Tobacco use
  - Alcohol ingestion
  - Stress
  - NSAID use.

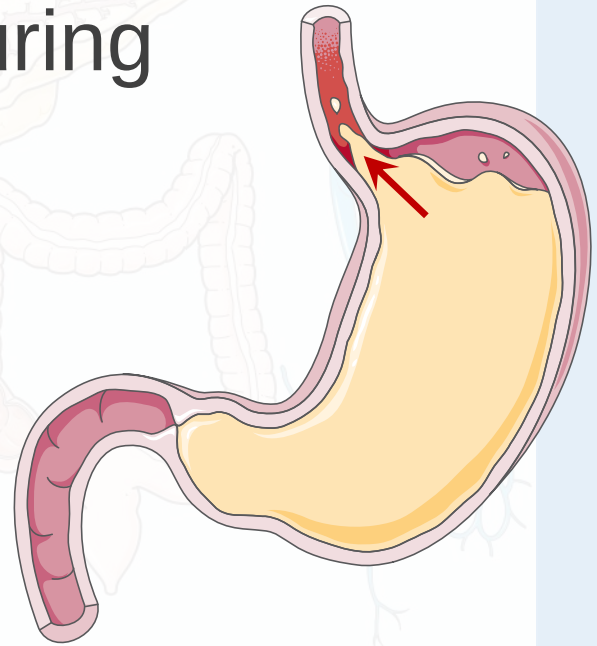


Pathophysiology

- Gastric mucus forms a physical and chemical barrier, protecting the epithelial cells lining the stomach and trapping bicarbonate between the mucus and the cells.
- Hydrogen production outpaces bicarbonate production during physiologic or psychological stress.
- Common gastric irritants include alcohol, stress, tobacco, caffeine, NSAIDs, Helicobacter pylori (H. pylori) bacteria, and shock.

12 Gastroesophageal Reflux Disease (GERD)

- Chest pain or severe burning occurring within an hour of eating.
- Discomfort is worse when lying down after meals and may occur during the night.



Pathophysiology

- Meals that are large, high-fat, spicy, or consumed concurrently with alcohol cause relaxation of the lower esophageal sphincter, allowing hydrochloric acid and pepsin present in gastric contents to reflux, or pass back, into the esophagus.
- The esophagus is easily damaged by acidic gastric contents, resulting in inflammation, edema, and scarring over time.

13 Hemorrhoids

- History of constipation
  Frank blood on the stool
- Painful anal lesions

Pathophysiology

- Varicosities of the veins of the anus related to increased intra-abdominal pressure.
- Occur during pregnancy as the weight of the fetus compresses the inferior vena cava, causing congestion of the veins in the anus (as well as the legs).
- Poor bowel habits and constipation contribute to the etiology.
- Internal hemorrhoids occur above the internal sphincter; external hemorrhoids occur below the external sphincter.

14 Hepatitis

- Lethargy & Malaise
- Low-grade fever
- Right upper quadrant pain
- Jaundice
- Elevated ALT & AST levels
- Headache
- Anorexia

Pathophysiology

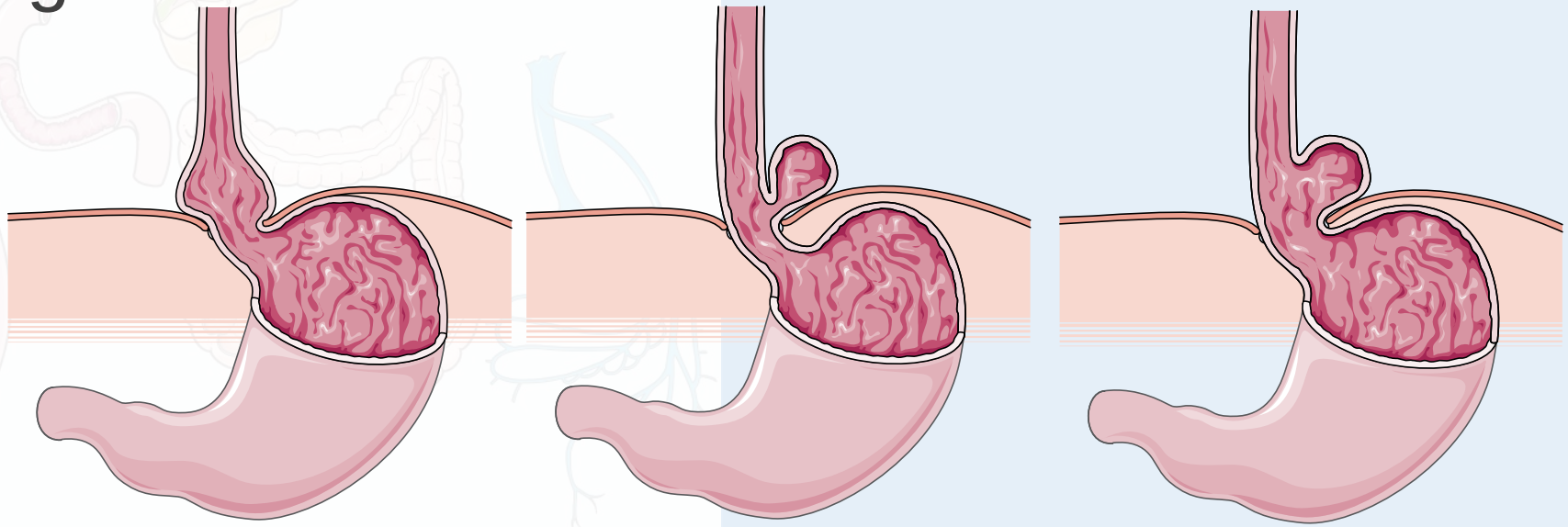
- Inflammation of the liver caused by direct cellular injury and secondary injury by the immune response; those with a lesser immune response may become carriers rather than infected.
- Hepatitis A (HAV): Spread by the oral-fecal route.
- Hepatitis B (HBV; often coexistent with hepatitis D [HDV]): Spread by blood and body fluids.
- Hepatitis C (HCV): Spread by contact with contaminated blood, IV drug use, unprotected sex.
- Hepatitis E (HEV): Spread by contaminated water.

15 Hiatal Hernia

- Burning
- Chest pain
- Heartburn,
- Dysphagia
- GERD
- A feeling of fullness.

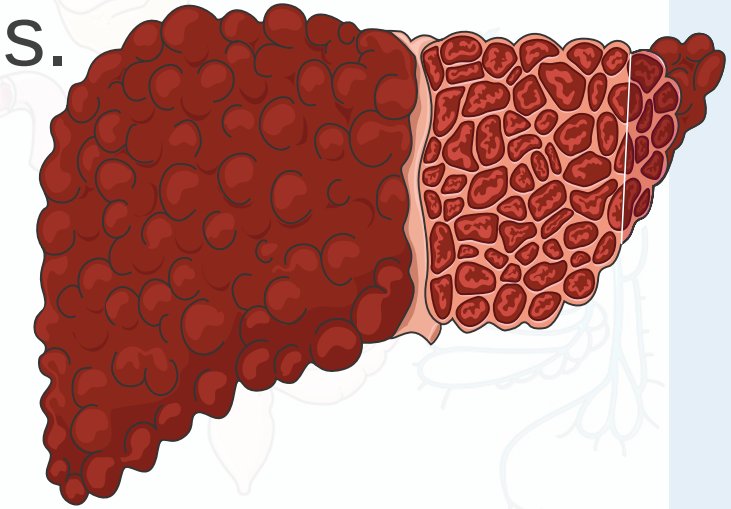
Pathophysiology

- A weakness in the hiatus of the diaphragm coupled with intra-abdominal pressure forcing protrusion of the stomach and esophagus upward through the hiatus.
- Hiatal hernias include the “sliding” type and the “rolling” type.



16 Laënnec's Cirrhosis

- Chronic condition associated with alcohol consumption.
- Elevated ALT & AST
- Ascites
- Edema in the lower extremities.



Pathophysiology

- Chronic heavy consumption of alcohol causes inflammation of the cells of the liver.
- Fatty infiltration of the liver occurs related to decrease in fatty acid oxidation and increase in gluconeogenesis. The liver enlarges first.
- The stellate cells to produce fibrous connective tissue and becomes resistant to blood flow from the portal vein; portal hypertension and ascites result.

17 Liver Cancer

- Elevated ALT & AST
- Ascites
- Edema in the lower extremities
- High bilirubin levels

Pathophysiology

- Chronic irritation of hepatocytes or surrounding parenchyma causes mutation of cells.
- The liver is a common site of mastastasis.

18 Pancreatic Cancer

- Weight loss
- Anorexia
- ↑ amylase, lipase, and bilirubin.
- ↑ vitamin D intake may be preventative.

Pathophysiology

- Mutation of cells in the pancreas occurs from genetic factors or chronic irritation.
- Tumors are most commonly found in the head of the pancreas and are large.
- Metastasis by direct extension to the stomach, gallbladder, liver, and duodenum occurs rapidly.
- Tumors in the body of the pancreas metastasize rapidly via blood and lymph.

19 Pancreatitis

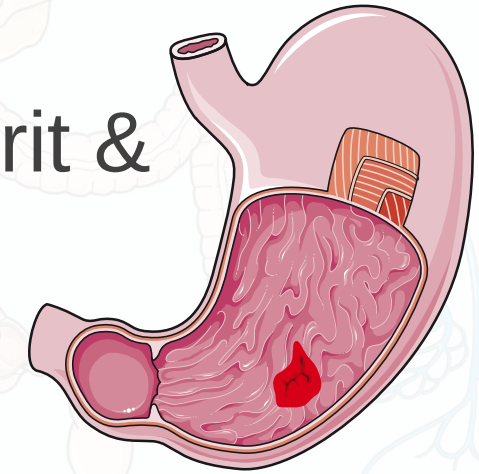
- Severe midline abdominal pain that radiates to the flank, spine & back
- Pain worsening with extension of the legs or ingestion of food.
- Elevated ALT & AST, amylase, lipase & glucose

Pathophysiology

- The outlet of the pancreas may become blocked due to inflammation, mechanically (gallstones), or by the digestive enzymes being prematurely activated while they are still in the pancreas.
- Protease causes dilation and permeability of the capillaries, allowing fluid to move from the pancreas to the retroperitoneal space. If fluid loss is severe, shock may occur.
- Protease initiates a chain reaction of inflammation that results in conversion of prothrombin to thrombin, causing DIC.

20 Peptic Ulcer Disease

- Gnawing, burning pain in either the midepigastric area 2–4 hours after meals or the left epigastric area with meals.
- Weight loss and presence of melena.
- Low hematocrit & hemoglobin

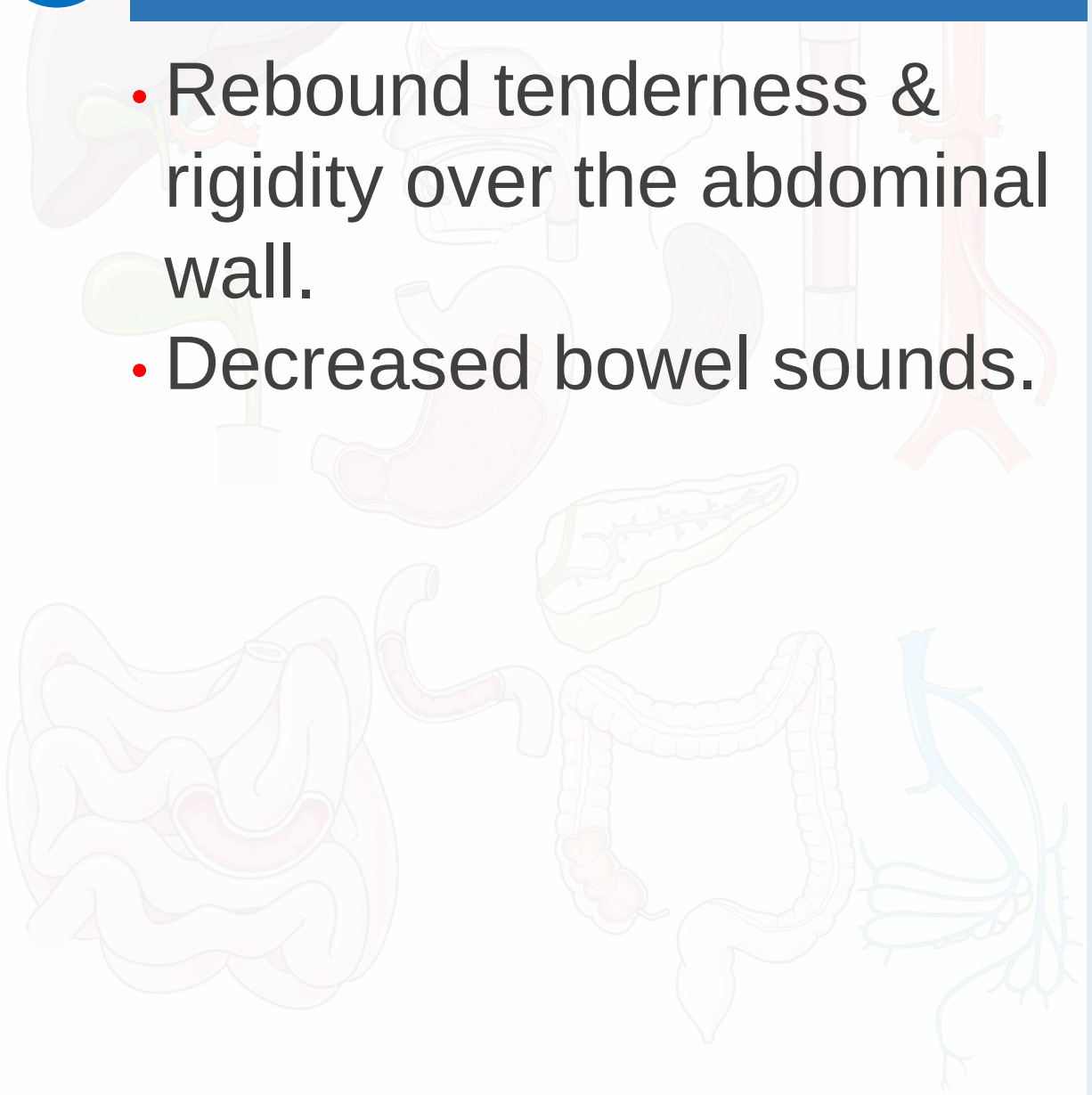


Pathophysiology

- Eighty percent of all peptic ulcer disease is caused by Helicobacter pylori (H.pylori) infection that causes inflammation and erosion of the mucosal barrier in the stomach.
- Gastric mucus provides a physical and chemical barrier, protecting the epithelial cells lining the stomach and trapping bicarbonate between the mucus and the cells.
- Hydrogen production outpaces bicarbonate production during physiologic or psychological stress.
- Common gastric irritants or contributors to gastritis include alcohol, stress, tobacco, NSAIDs, H. pylori bacteria, and shock.

21 Peritonitis

- Rebound tenderness & rigidity over the abdominal wall.
- Decreased bowel sounds.



Pathophysiology

- Inflammation of the sterile peritoneal cavity by introduction of bacteria via invasive procedures, open bowel surgeries, or perforation of intraabdominal organs whose normal flora contain bacteria.

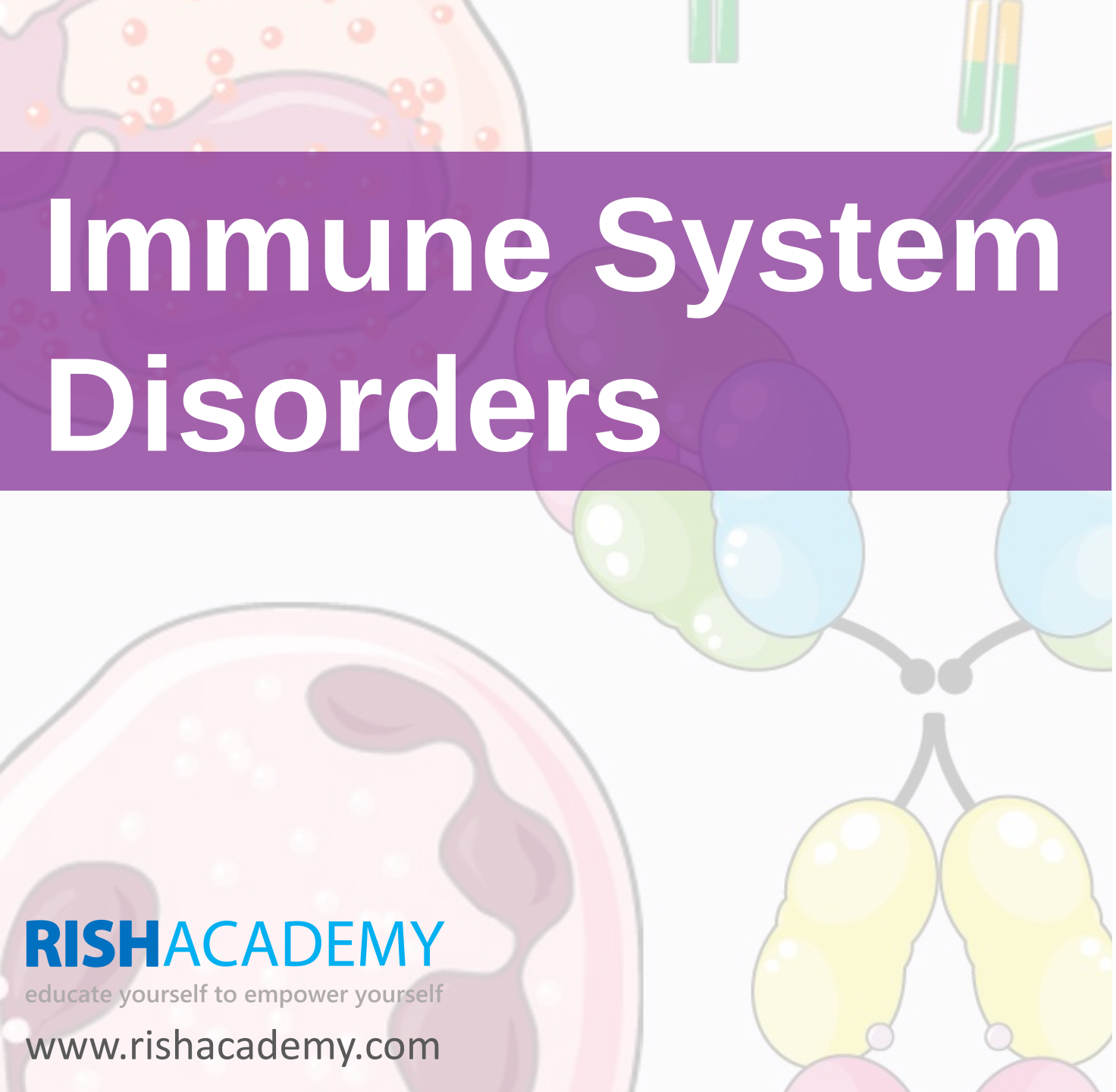
22 Ulcerative Colitis

- Daily passage of six or more bloody mucus stools associated with abdominal pain.

Pathophysiology

- Inflammation and hemorrhage in small areas of the mucosal layer of the colon cause abscesses to form (crypt abscesses). The necrotic areas slough off, causing ulcer formation that extends to the submucosal layer of the bowel.
- Blood in the colon causes hypertonicity of the bowel contents and acts as a laxative.
- Pseudopolyps (ragged edges of the mucosal layer).
- Incidence is greatest in the second, third, and sixth decades of life, a genetic link exists; probable autoimmune disease.
- Lesions begin in the rectum and spread proximally.

Immune System Disorders



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Clinical Medicine Flashcards

- Clinical Clues to Diagnosis
- Pathophysiology

- Acquired Immunodeficiency Disease
- Anaphylaxis
- Hashimoto’s Thyroiditis
- Kaposi’s Sarcoma
- Scleroderma
- Sjögren’s Syndrome
- Systemic Lupus Erythematosus

1 Acquired Immunodeficiency Disease

- Lymphadenopathy
- Night sweats
- Presence of rare opportunistic illness
- T-helper (CD4) cells <500 cells/mm3,
- T-killer (cytotoxic) (CD8) cells <375 cells/mm3
- Change in the CD4/CD8 ratio (normal 0.9–1.9),
- Measurable viral load

Pathophysiology

- Macrophages process foreign antigens and present antigenic material to the T-helper cells (CD4).
- The CD4 transfer this information to the T and B lymphocytes. In HIV, a retroviral particle (RNA strand) wrapped in a glycoprotein coat (gp120 receptor) with p24 viral protein invades the CD4 cell. The CD4 cell and macrophage are the immune cells affected and destroyed.
- Once the CD4 cell count drops below 200 cells/mm3, the client is diagnosed with AIDS. Other diagnostic criteria include the presence of an opportunistic infection.

2 Anaphylaxis

- Sudden onset of
  - Wheezing,
  - Edema of airway,
  - Hypotension,
  - Tachycardia,
  - Feeling of impending doom and anxiety.

Pathophysiology

- Severe type I hypersensitivity reaction in which IgG antibodies attached to mast cells, previously sensitized to an antigen, are reactivated. The most common antigenic material is derived from foods or insect stings.
- Chemical mediators are released, the most common of which are histamine, proteases, chemotactic factors, leukotrienes, prostaglandin D, cytokines, and interleukins 1, 3, 4, 5, and 6). These mediators cause vasodilation and fluid shift from the intravascular to the interstitium.

3 Hashimoto's Thyroiditis

- Goiter
- Periods of insomnia
- Anxiety
- Muscle and joint aches
- Weight changes
- Hair loss
- Fertility problems that are mixed symptoms of hyper- and hypothyroidism.
- Tsh may be normal or elevated.
- T3 and t4 are low.

Pathophysiology

- Usually a disease of older women with a history of autoimmune disease.
- Autoantibodies are produced to fight TSH. TSH is not destroyed and instead binds with its receptors in the thyroid gland, causing symptoms of hyperthyroidism.
- As the thyroid gland becomes infiltrated with lymphoid tissue and plasma cells it enlarges and hypothyroidism occurs.
- Episodic hyperthyroidism can occur, so symptoms may swing back and forth from hyperthyroidism to hypothyroidism.

4 Kaposi's Sarcoma

- Red-to-purple macules, papules, and nodules seen in persons with AIDS.
- First seen usually on the mucous membranes.

Pathophysiology

- A rare lymphatic malignancy of the endothelial, rather than connective, tissue characterized by red-to-purple macules, papules, or nodules.
- Lesions are first seen on the skin or mucous membranes but may involve the internal organs.
- A rare cancer commonly related to AIDS.
- In patients with AIDS, KS is believed to be sexually acquired by infection with the human herpes virus 8.

5 Scleroderma

- Taut face without wrinkles
- Calcium deposits.
- + ELISA for ANA & other antibodies.
- Raynaud's phenomenon

Pathophysiology

- An autoimmune disease.
- Women are affected more often than men; disorder often has periods of exacerbation and remission.
- The skin, connective tissue, and internal organs are affected. Insoluble collagen is overproduced and deposited in the skin and other organs, causing inflammation. Inelastic rather than supple edema results. A common finding is "stone face," which is the result of this hardening of the skin.
- There is a strong association (95%) with Raynaud's phenomenon.

6 Sjögren's Syndrome

- Blurred vision
- Thick secretions
- Decreased sense of taste
- Dysphagia
- Xerostomia
- Dry nasal membranes
- Antiribonucleoprotein serum antibodies & + rheumatoid factor in the absence of rheumatoid arthritis

Pathophysiology

- Autoimmune illness in which the lacrimal and salivary glands are attacked by autoantibodies and T lymphocytes.
- Can occur alone or with other autoimmune diseases.
- Occurrence is mainly seen in older women.
- Sjögren's syndrome is associated with a 40%–60% increase in the chance of developing non-Hodgkin's lymphoma.

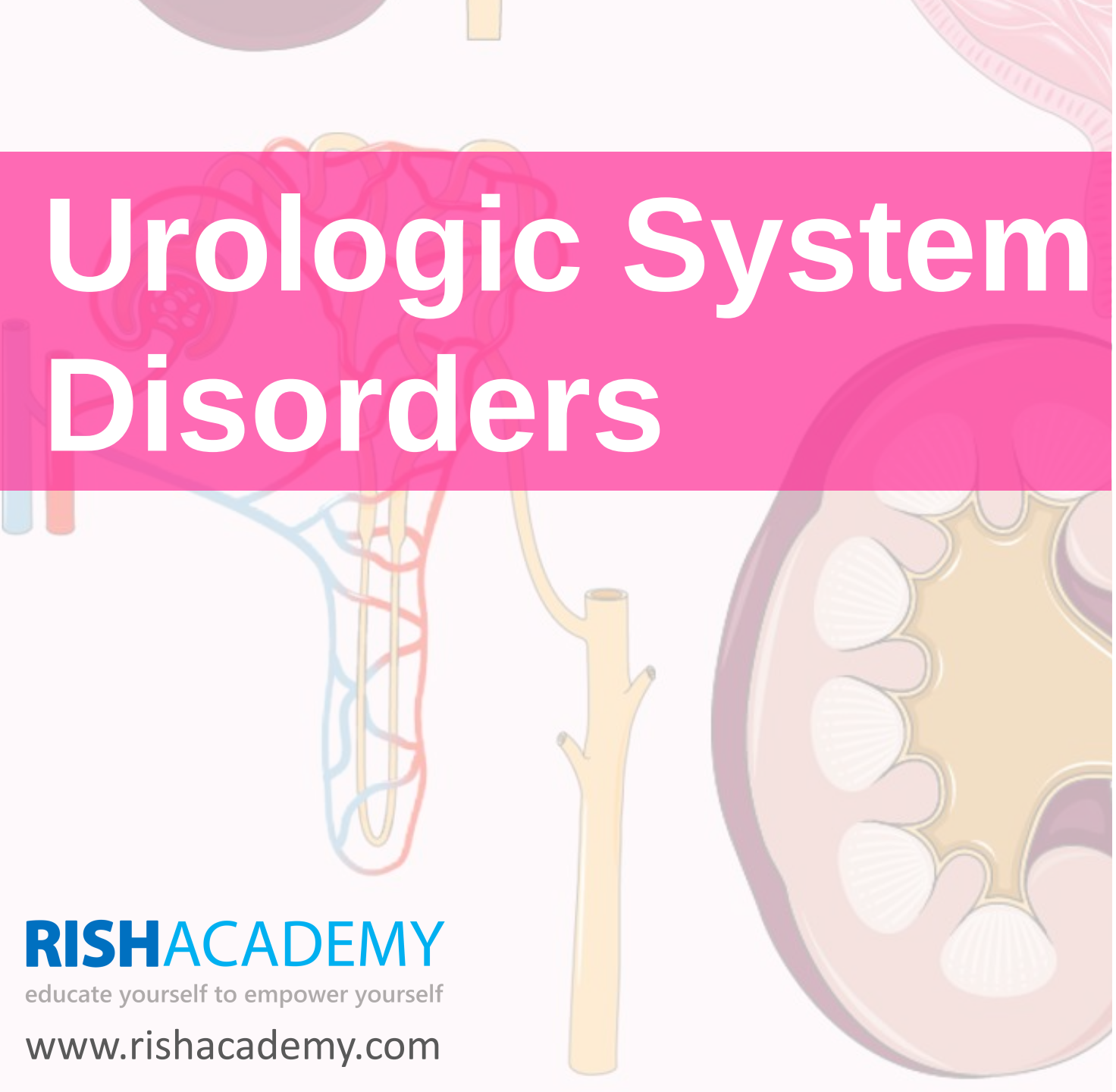
7 Systemic Lupus Erythematosus

- Butterfly rash
- Arthritis
- Malaise
- Raynaud's phenomenon
- Peripheral neuropathy
- Change in vision & renal status
- Round lesions on head cause hair loss

Pathophysiology

- Immune system antibodies attack "self." Females are affected more than males. A hereditary predisposition exists.
- Discoid lupus causes skin plaques that tend to occur on the face, ears, and hair. Wherever they appear, the area is inflamed and becomes scarred. Alopecia results in affected areas in the hair.
- Systemic lupus erythematosus (SLE) causes changes in the dermatologic, cardiovascular, musculoskeletal, hematologic, gastrointestinal, renal, and ophthalmologic systems—all related to inflammation from overreaction of the immune system.

Urologic System Disorders



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Clinical Medicine Flashcards

- Clinical Clues to Diagnosis
- Pathophysiology

- Acute Renal Failure
- Benign Prostatic Hyperplasia
- Bladder Cancer
- Chronic Renal Failure
- Epididymitis
- Glomerulonephritis
- Hydronephrosis
- Nephrotic Syndrome
- Overactive Bladder
- Polycystic Kidney Disease
- Prostate Cancer
- Pyelonephritis
- Renal Artery Stenosis
- Renal Calculus
- Rhabdomyolysis
- Urethritis
- Urinary Tract Infection

1 Acute Renal Failure

- Azotemia, anuria, or oliguria.
- Precipitated by severe hypotension, use of diagnostic contrast dyes, or structural damage to nephrons.
- Elevated K+ and decreased Na+ in serum.
- Elevated creatinine and BUN.

Pathophysiology

- Acute damage to nephrons associated with severe hypotension, use of contrast dyes, or damage to skeletal muscle fibers that accumulate in the nephron tubules.
- Three stages: The oliguric stage (less than 400 mL/24 hr), lasting 2 weeks (better prognosis) to several months (poor prognosis). The diuretic phase, characterized by a normal output of low-quality urine lasting up to a month. The recovery phase, which may last up to 1 year. The quality of urine in this phase improves, but full recovery is not guaranteed.
- Prerenal conditions are those that decrease perfusion of the kidneys. Intrarenal failure includes incidents that damage the nephrons.
- Postrenal failure is caused by obstruction, resulting in hydronephrosis.

2 Benign Prostatic Hyperplasia

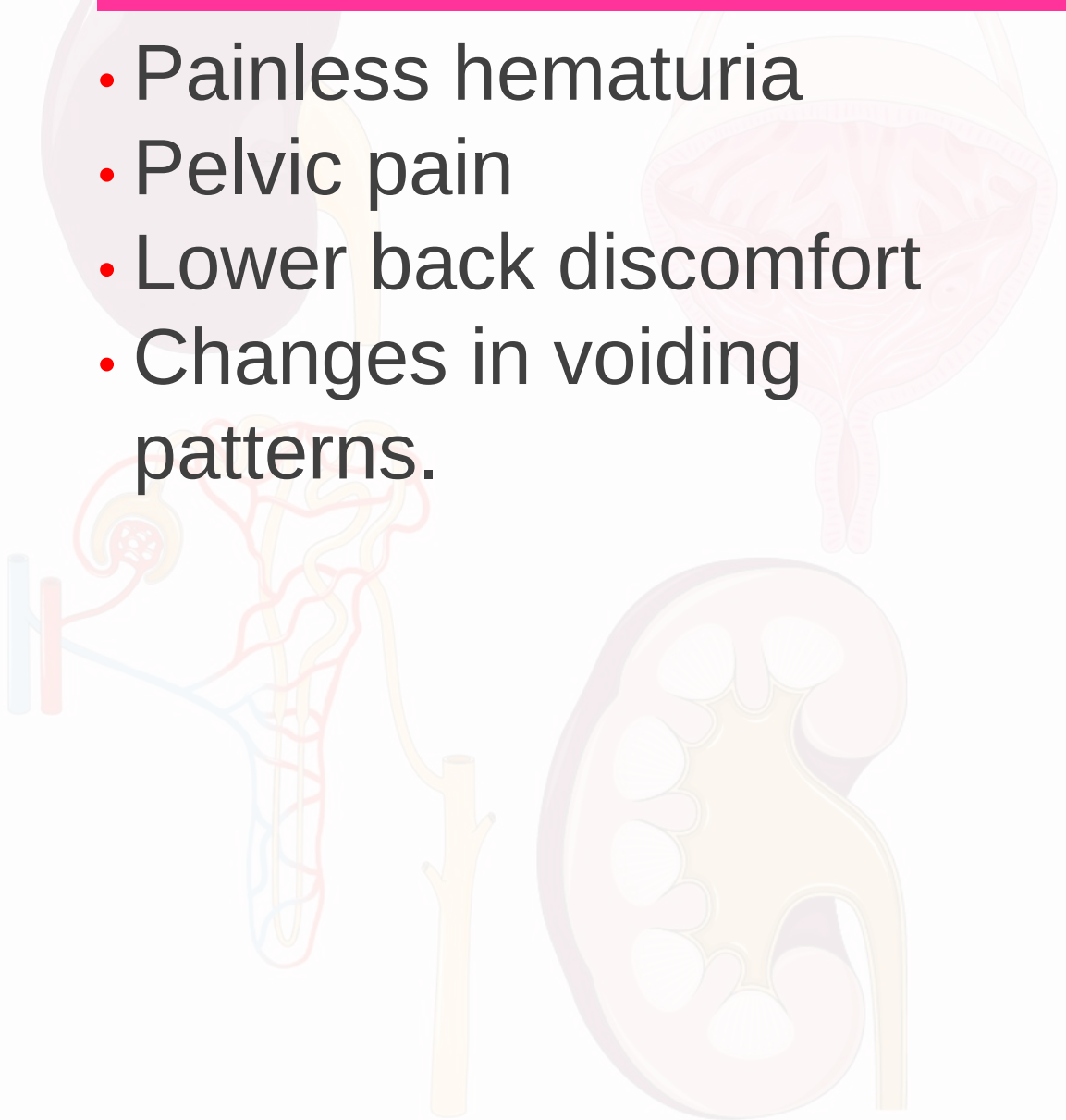
- Difficulty starting or maintaining urinary stream
- Dribbling of urine
- Urgency & frequency
- Men approaching or in the fifth decade of life.

Pathophysiology

- Enlargement of glandular tissue in the periurethral area of the prostate under the influence of testosterone, particularly DHT.
- Estrogen is also implicated, as it makes the gland more susceptible to DHT.
- The prostatic urethra narrows as the prostate gland enlarges, causing partial, or eventually total, obstruction of urine outflow from the bladder.

3 Bladder Cancer

- Painless hematuria
- Pelvic pain
- Lower back discomfort
- Changes in voiding patterns.



Pathophysiology

- More common in middle-aged males than in females.
- Strong association with cigarette smoking.
- Exposure to industrial pollutants (e.g., aniline dyes).
- The tumor-node-metastasis (TNM) method of staging the cancer determines prognosis and treatment.
- Over time, dysplastic changes occur in the urothelium. With chronic irritation, these areas of dysplasia are replaced by malignant cells. The cells may form small cancers that remain in the urothelium or may become invasive and metastatic to the liver, lungs, and bones.

4 Chronic Renal Failure

- History of
  - Diabetic nephropathy,
  - Hypertension,
  - Glomerulonephritis, or
  - An autoimmune disease (systemic lupus erythematosus [SLE])

Pathophysiology

- Gradual destruction of the nephrons and reduction of GFR. Acute renal failure, diabetic nephropathy, and hypertension are the most common causes, but abnormalities of the kidney, autoimmune disorders, and chronic infection or cancer are also causes.

5 Epididymitis

- Painful inflammation of the back of the testes.
- The scrotum is erythematous

Pathophysiology

- Infection and inflammation of the epididymis, the tube along the back side of the testes in which sperm mature and are stored, can be the result of several events.
- In older men, regurgitation of urine from excessive bladder pressure when trying to urinate in the presence of an enlarged prostate can force urine into the vas deferens to the epididymis, causing infections with bacteria such as Escherichia coli.
- Infections with sexually transmitted organisms occur with frequency in young, sexually active males.
- Congenital structural abnormalities in young children predispose them to infection.
- Trauma results from excessive pressure exerted on the epididymis.

6 Glomerulonephritis

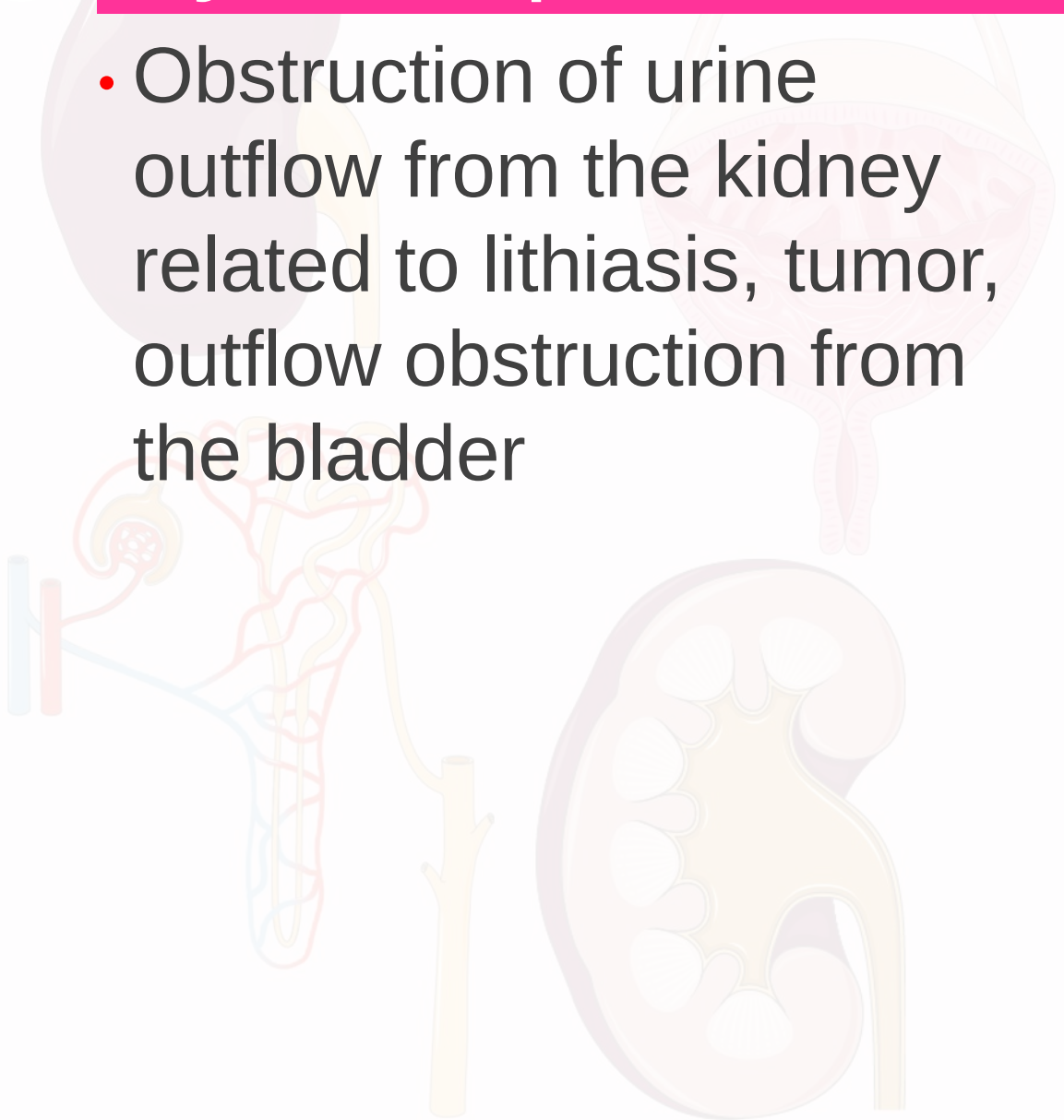
- Hypertension
- Oliguria
- Smoky, frothy urine
- Urinalysis shows RBCs casts, and protein.

Pathophysiology

- The glomerulus is formed from tufts of arteriolar capillaries fed by an afferent arteriole and drained by an efferent arteriole that have thin basement membrane composed of a proteinous matrix and a layer of epithelial cells with footlike outpouches. Blood plasma is forced through these thin structures by a pressure gradient into Bowman's capsule and the renal tubule.
- A number of toxins, diseases, and organisms can cause inflammation and damage to this basement membrane.
- In poststreptococcal infection, antigens are deposited in the basement membrane of the glomerulus. When antigen/antibody complexes form, the immune system destroys them, setting up large areas of inflammation and damage to surrounding structures.

7 Hydronephrosis

- Obstruction of urine outflow from the kidney related to lithiasis, tumor, outflow obstruction from the bladder



Pathophysiology

- Unilateral or bilateral swelling of the renal capsule from regurgitant urine related to outflow obstruction. Because the renal capsule is fibrous, internal functional structures (nephrons) are destroyed.
- Causes may include renal system lithiasis; tumors of the kidneys, ureters, or bladder; enlargement of the prostate; or stricture of the urethra.
- May occur with continuous bladder irrigation (CBI) if a clot obstructs outflow of irrigant and urine or with an obstructed Foley catheter.

8 Nephrotic Syndrome

- Elevated LDL cholesterol and triglyceride
- Proteinuria, frothy urine from protein and lipids,
- Decreased immunoglobulins lost in urine.
- Massive edema.

Pathophysiology

- Nephrotic syndrome is an umbrella term encompassing disorders that result from glomerular damage. Damage to the basement membrane results in loss of blood components that would otherwise remain in circulation.
- Large amounts of protein and immunoglobulins are lost in the urine. Hyperlipidemia and hypertriglyceridemia occur as the liver responds to the low protein levels. Triglycerides and LDL are also lost in the urine, to some extent adding to the frothy appearance.
- Protein loss causes loss of intravascular fluid into the interstitial spaces, but low glomerular filtration rate still results in hypertension.

9 Overactive Bladder

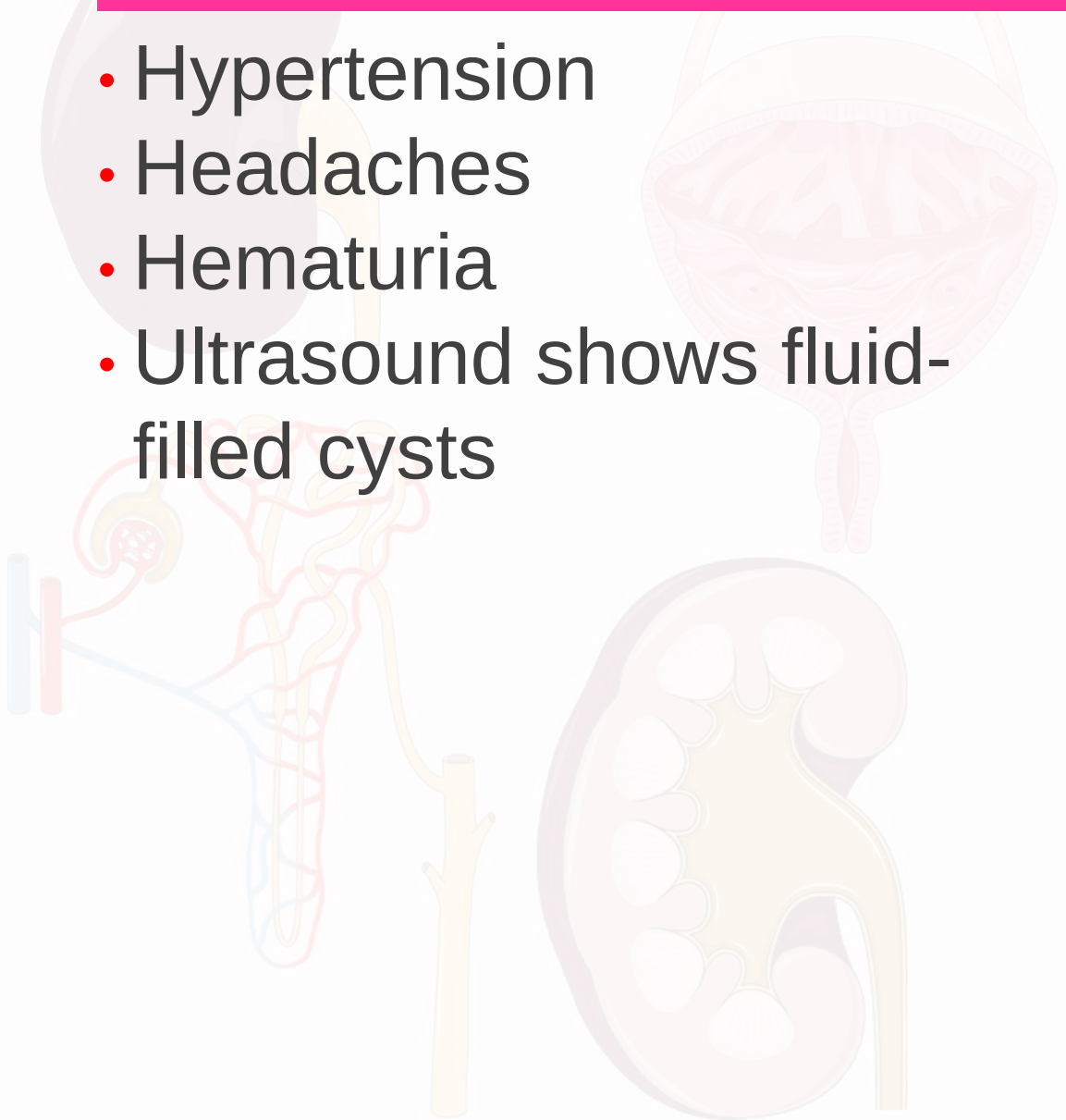
- Urgency
- Frequency
- Stress incontinence related to autonomic and structural anomalies of the bladder.

Pathophysiology

- Overactive bladder is thought to be caused by excessive parasympathetic impulses to the detrusor muscle of the bladder, initiating the micturition response.
- Also, structural anomalies resulting from pelvic relaxation syndrome decrease the angle of the bladder, causing undue pressure on the neck of the bladder and abnormal stretch of the transitional cells, which again triggers the micturition response.
- Neurogenic causes may include chronic neurologic illnesses (e.g., multiple sclerosis) that unintentionally stimulate motor function and the micturition reflex arc, making the bladder more active.

10 Polycystic Kidney Disease

- Hypertension
- Headaches
- Hematuria
- Ultrasound shows fluid-filled cysts



Pathophysiology

- Hereditary disorder causing cystic formation in the cortex or medulla of the kidney.
- Cysts may develop from pressure buildup in the tubules and can progress to the entire kidney.
- Glomerular filtration rate (GFR) decreases.
- Stasis of fluid in the cysts predisposes to repeated urinary tract infection (UTI).
- Persons with this hereditary disease are at high risk for aneurysms in the brain and diverticulosis related to body system formation during the embryonic period.

11 Prostate Cancer

- Late symptoms include
  - Signs of urinary obstruction
  - Pain in the lumbar or hip area
  - Weight loss
  - Weakness
- Urine outflow may be impaired

Pathophysiology

- Prostatic glandular cells mutate and grow under the influence of testosterone and DHT.
- Prostate cancer late in life is usually slow growing (↓ testosterone levels).
- Metastatic spread into other urinary and reproductive structures is through lymph and blood vessels.
- The TNM system is used to grade the cancer and make a prognosis.

12 Pyelonephritis

- Chills
- Fever
- Tenderness over the costovertebral angle
- Dysuria
- Elevated WBC

Pathophysiology

- Usually an ascending urinary tract infection (UTI) caused by a failure of the “washout” mechanism of urine and protective mucin gel. Causative agents are usually Escherichia coli and, to a lesser extent, Staphylococcus aureus.
- Kidney pelvis structures may be damaged by ongoing infection, leading to nephron damage and renal failure.

13 Renal Artery Stenosis

- Onset of severe hypertension in the absence of
  - Glomerular disease
  - Renal failure or
  - Pheochromocytoma

Pathophysiology

- Hypertension occurs when the renal artery becomes narrowed and incapable of transmitting blood to the kidney. The response is activation of the renin-angiotensin-aldosterone mechanism to increase vasoconstriction, further increasing the blood pressure.
- Young women usually develop renal stenosis from fibromuscular dysplasia; older adults develop it from chronic atherosclerotic disease.

14 Renal Calculus

- KUB or US shows one or more masses in the kidneys, ureters, or bladder
- Renal colic in the flank that radiates downward
- Nausea
- Vomiting
- Costovertebral tenderness

Pathophysiology

- Men are affected more than women, and stone formation is usually unilateral. Once stones have formed, repeated formation is likely.
- Irritation of the epithelial cells that line the tubules.
- Dehydration causes more solute to be present in the urine.
- Persons prone to stone formation may lack inhibitor proteins and stones may recur.
- Small stones (<5 mm) usually are passed in the urine.

15 Rhabdomyolysis

- Azotemia
- Edema
- Hypertension
- Hematuria
- Arrhythmias
- Common causative drugs are cholesterol lowering agents

Pathophysiology

- Results from crush injuries (compartment syndrome), the toxic effect of drugs or chemicals on skeletal muscle, extremes of exertion, sepsis, shock, electric shock, and severe hyponatremia.
- Lipid-lowering drugs (e.g., statins, niacin, and/or fibrates) are among the commonly prescribed drugs that cause damage to skeletal muscle fibers that are released into the bloodstream and accumulate in renal tubules.

16 Urethritis

- Dysuria, blood in the urine or ejaculate in a male.
- Discharge from the urethra.
- History of unprotected sex
- In women, pelvic pain

Pathophysiology

- More common in men but can occur in women; also characterized by inflammation and colonization of the urethra by Escherichia coli, Neisseria gonorrhoeae, Chlamydia trachomatis, herpes simplex, or cytomegalovirus.
- Infectious agents may ascend and affect the prostate and infiltrate the lymph nodes in the groin area. In women, these agents can ascend to infect the pelvic area and may be a cause of infertility.

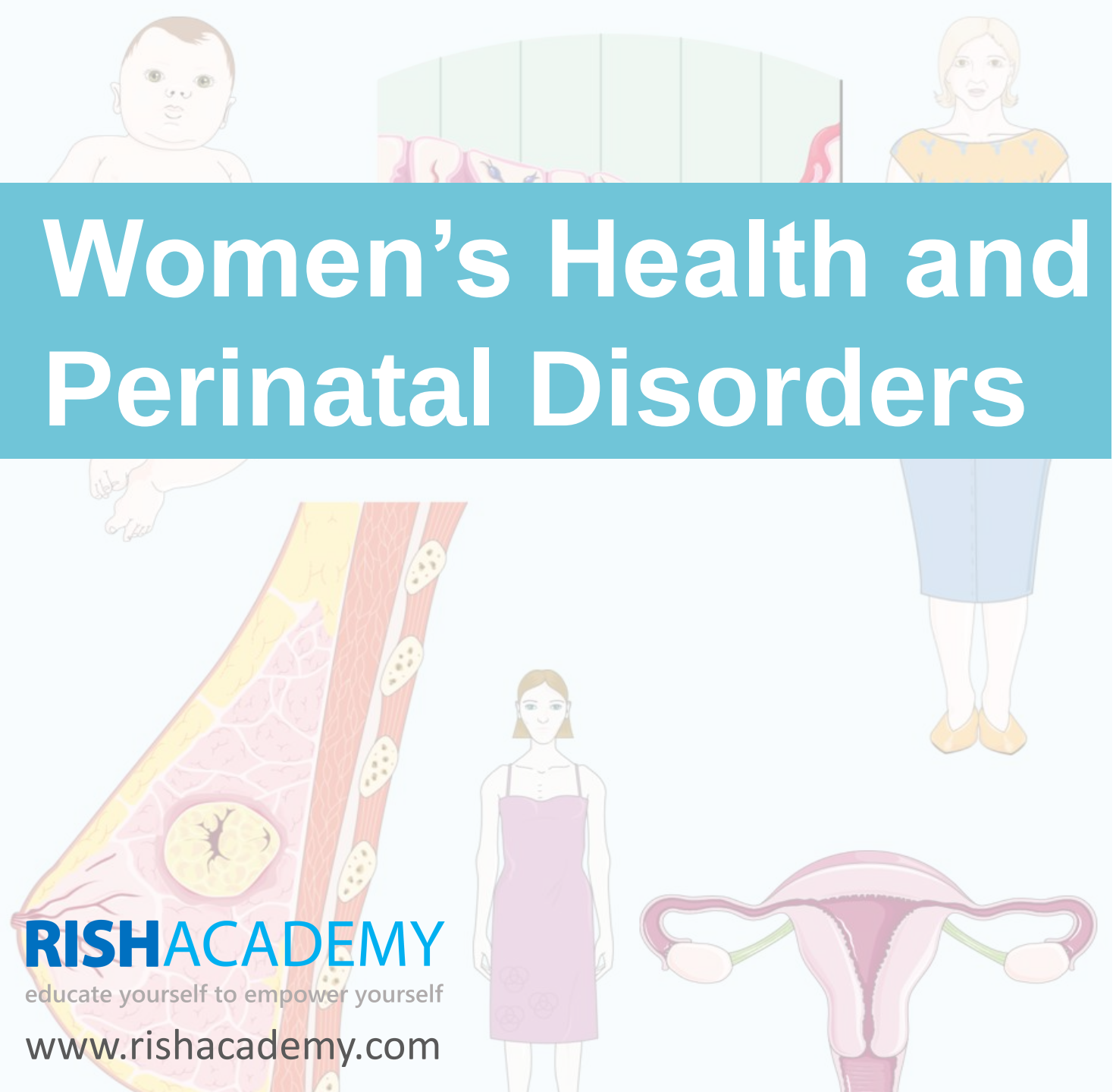
17 Urinary Tract Infection

- Urinary frequency
- Urgency
- Dysuria
- Bacterial count of >100,000/mL of urine

Pathophysiology

- Occurs more frequently in women because of anatomy and age-related structural changes.
- UTIs can also occur as a result of obstructive disease, invasive therapies, and incontinence issues.
- Most UTIs (95%) are caused by contamination and ascension in the urethra by normal flora from the rectum.
- Causative agents are Escherichia coli; Staphylococcus saprophyticus; and to a lesser extent Klebsiella species, Proteus mirabilis, Staphylococcus aureus, and Pseudomonas aeruginosa.
- The normal mucin-surface glycosaminoglycans are overwhelmed and bacteria become adherent to bladder surfaces.
- Soap in bathwater causes UTIs in children.

Women's Health and Perinatal Disorders



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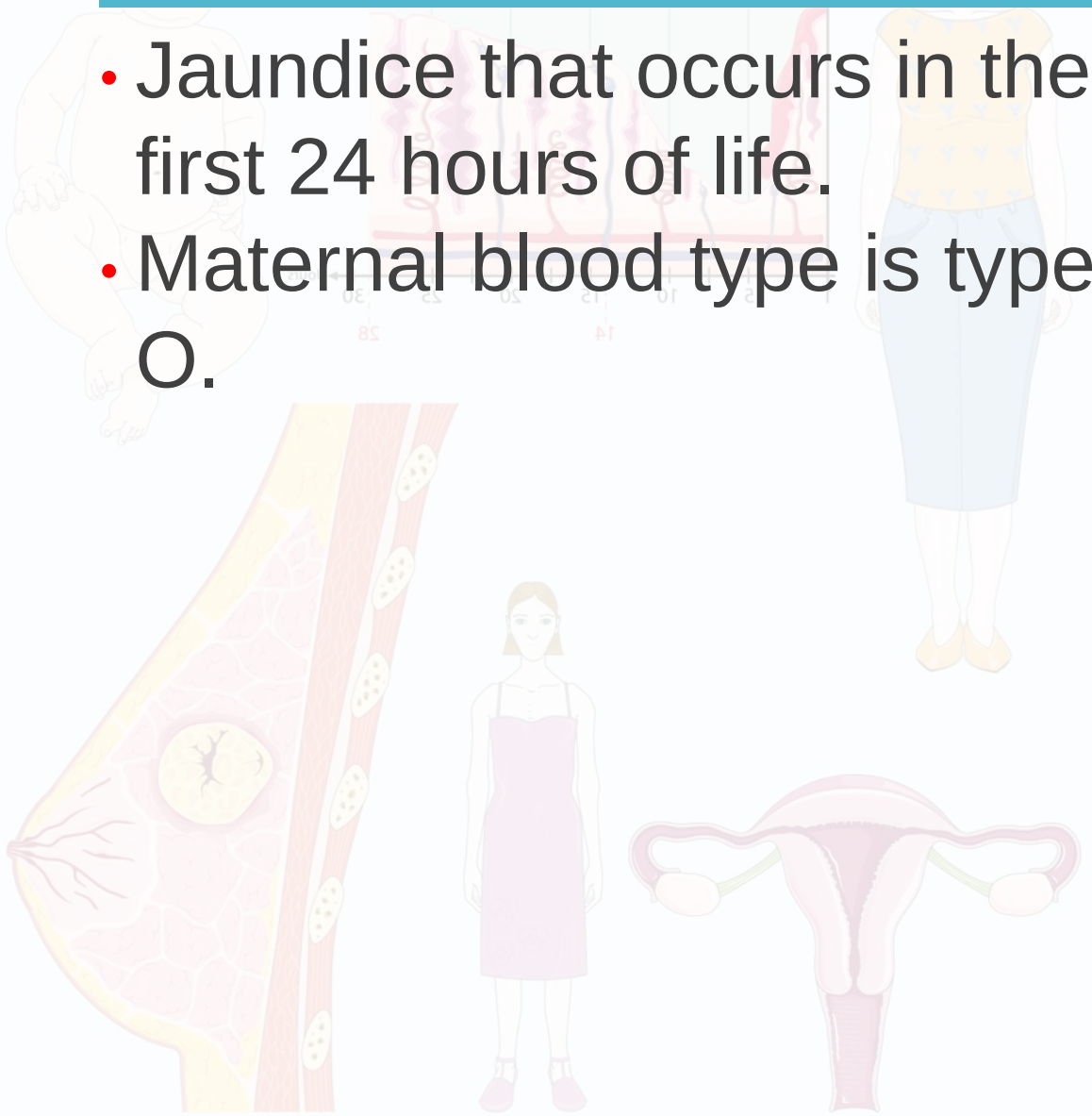
Clinical Medicine Flashcards

- Clinical Clues to Diagnosis
- Pathophysiology

- ABO Blood Type Incompatibility
- Breast Cancer
- Cervical Cancer
- Leiomyomas
- Meconium Aspiration Syndrome
- Neonatal Sepsis
- Patient Ductus Arteriosus
- Persistent Fetal Circulation
- Placenta Abruption
- Placenta Previa
- Polycystic Ovarian Syndrome
- Pregnancy-Induced Hypertension
- Rh Incompatibility

1 ABO Blood Type Incompatibility

- Jaundice that occurs in the first 24 hours of life.
- Maternal blood type is type O.

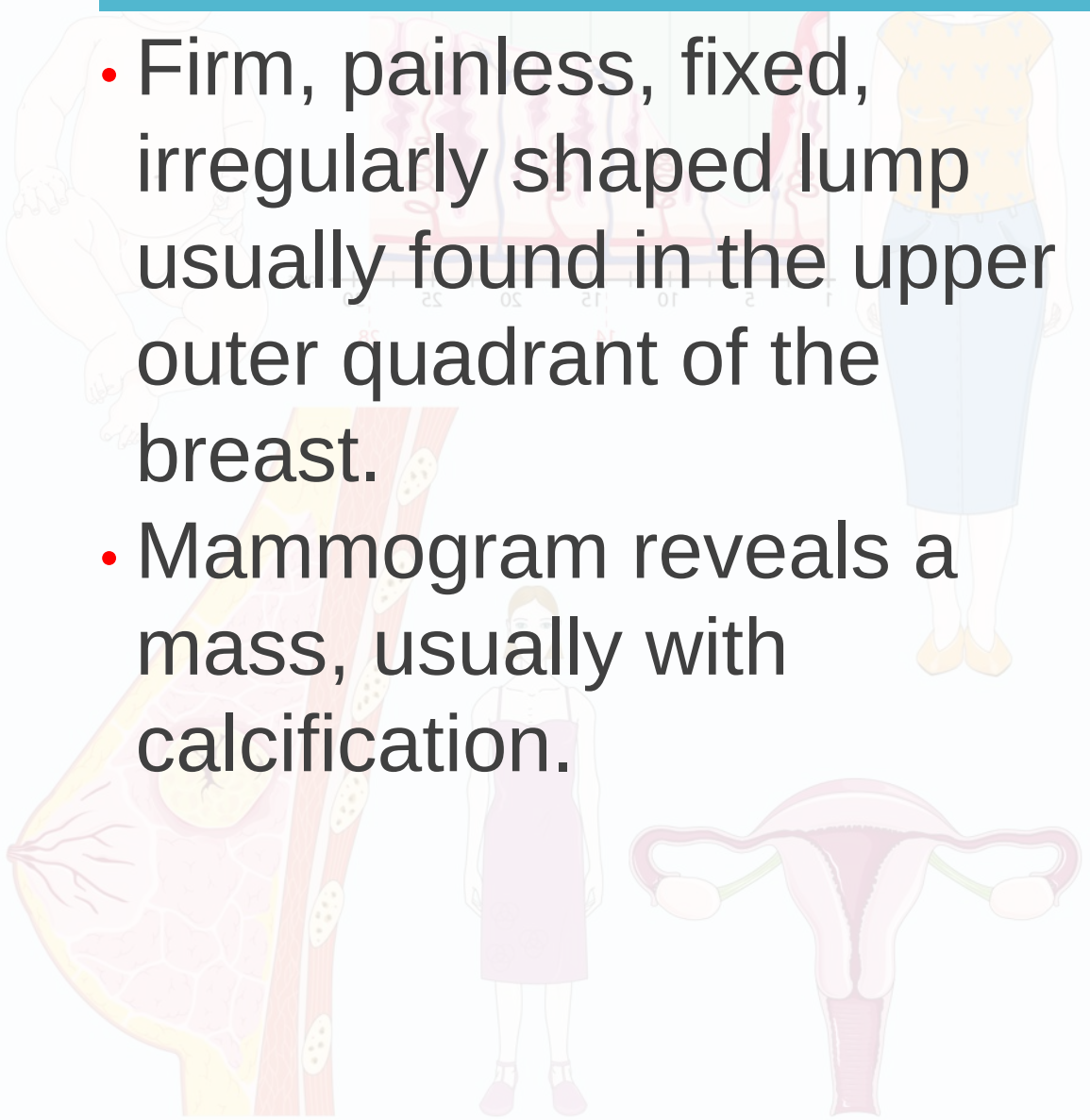


Pathophysiology

- Mothers with type O blood carrying fetuses with type A, B, or AB blood are at risk for having a neonate with an ABO incompatibility problem if their antibodies are IgG antibodies.
- IgG antibodies readily cross the placenta and begin to hemolyze the fetal red blood cells (RBCs).
- The fetal/neonatal liver is too immature to process the bilirubin produced from the RBC breakdown, and this results in early-onset jaundice.

2 Breast Cancer

- Firm, painless, fixed, irregularly shaped lump usually found in the upper outer quadrant of the breast.
- Mammogram reveals a mass, usually with calcification.

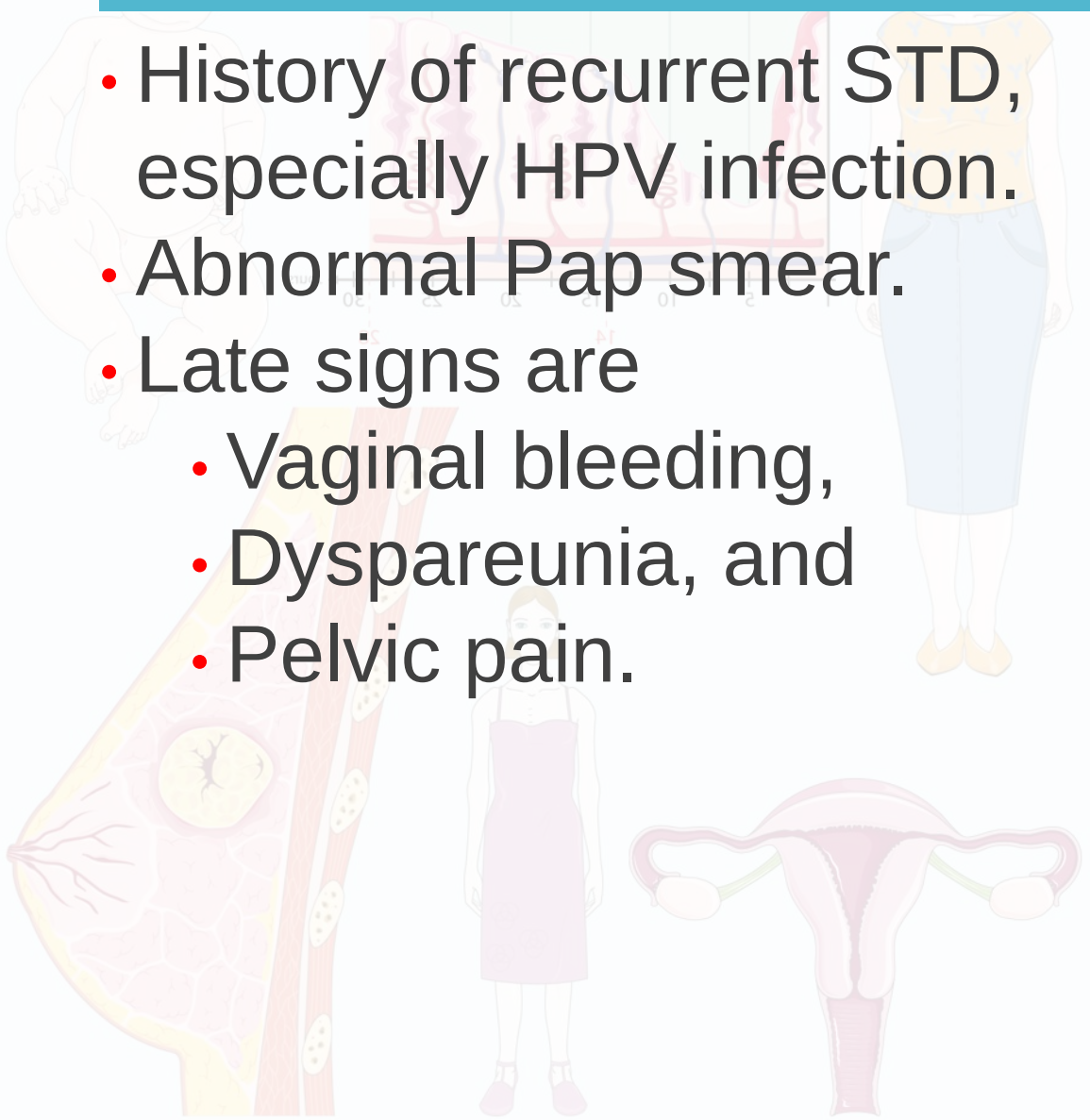


Pathophysiology

- Overstimulation of estrogen.
- TP53 mutation, (protection against tumor growth).
- Genetic anomalies (e.g., BRCA1, BRCA2, TP53) increase the risks of developing breast cancer and ovarian cancer.
- Women develop cancer more than men.
- Risk factors are ↑ age, ↓ immunity, HRT, personal or family history of breast cancer, high-fat diet, alcohol intake, early menarche, late menopause, no pregnancy or late pregnancy, and no or short breastfeeding.

3 Cervical Cancer

- History of recurrent STD, especially HPV infection.
- Abnormal Pap smear.
- Late signs are
  - Vaginal bleeding,
  - Dyspareunia, and
  - Pelvic pain.

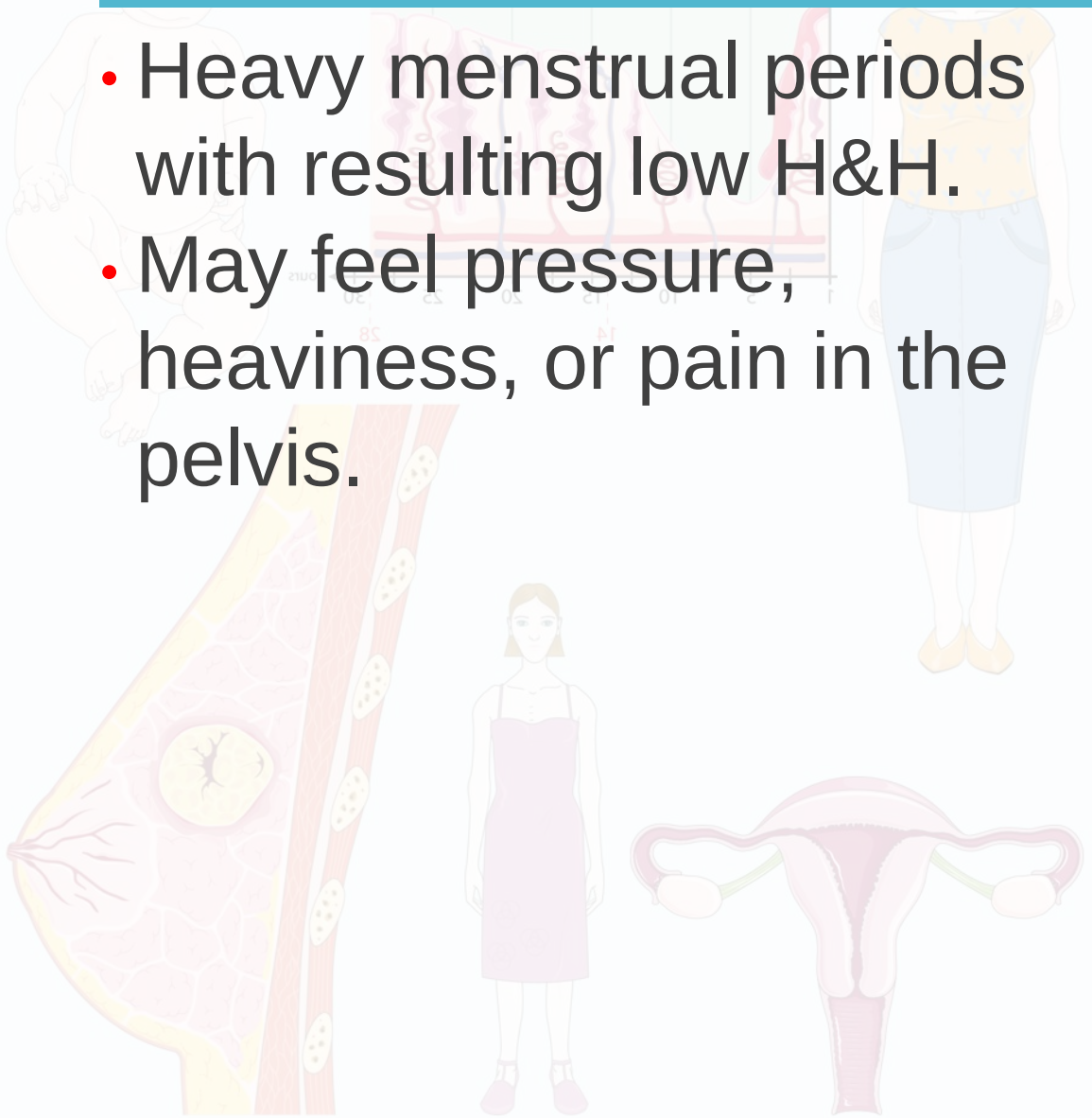


Pathophysiology

- Squamous cells on the outer cervix, chronically irritated, undergo dysplasia from antigenic or infectious material from STD, especially HPV, or multiple sexual partners. Glandular cells on the uterine side of the cervix can undergo dysplasia from chronic irritation of smoking, infection with HIV that lowers immunity, or having several pregnancies.
- LSIL, and HSIL are squamous cells (afffected by HPV) that are likely to progress to cancer. CIN followed by the numbers 1, 2, or 3 describes the thickness of the lining of the cervix that contains abnormal cell growth.
- Pap smear results and grade (low or high) guide treatment regimen.

4 Leiomyomas

- Heavy menstrual periods with resulting low H&H.
- May feel pressure, heaviness, or pain in the pelvis.

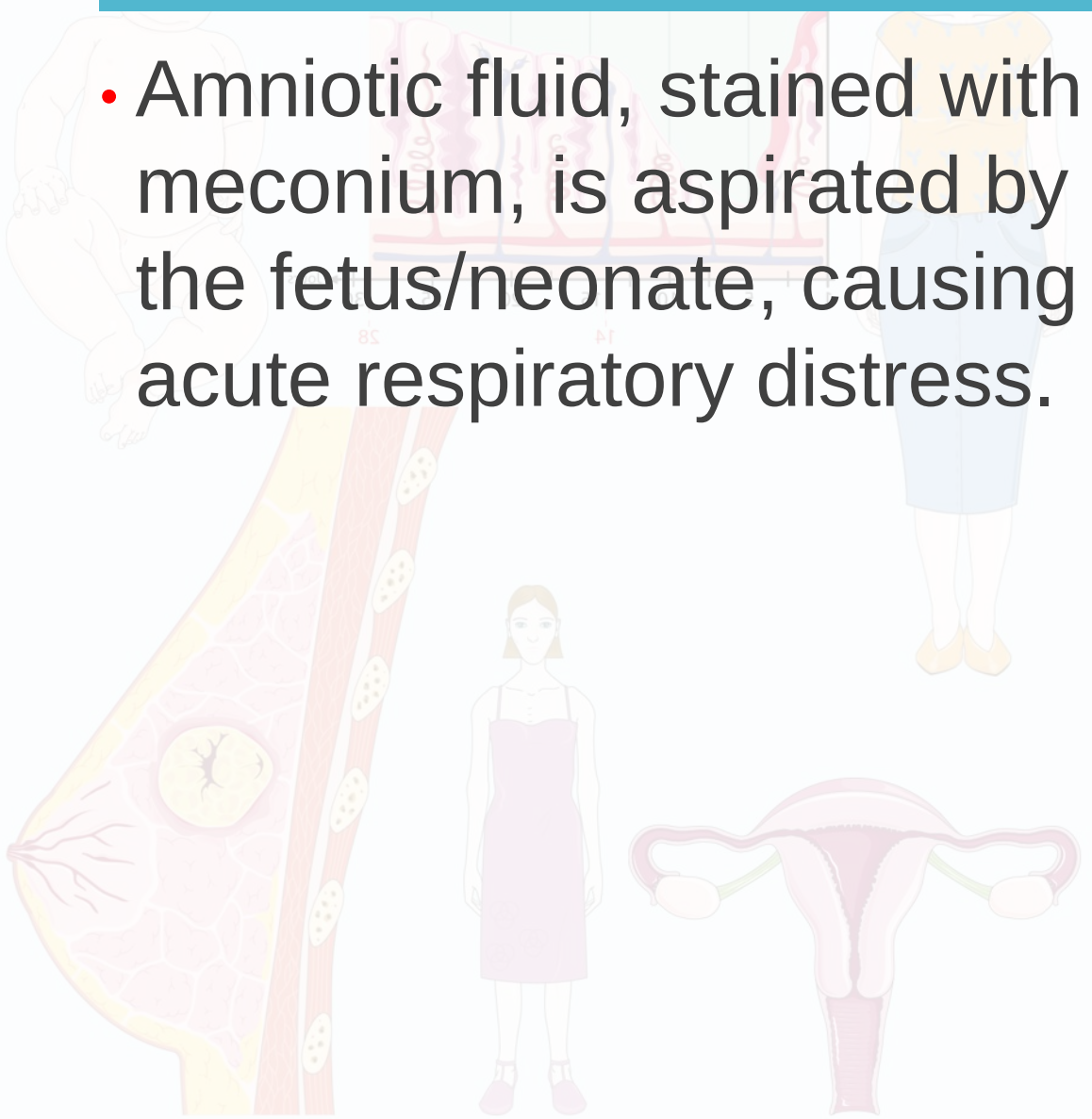


Pathophysiology

- Estrogen is dominant in the proliferative phase of the menstrual cycle and acts on the endometrial layer and is also responsible for proliferation of abnormal uterine cells that grow under its influence.
- Benign tumors or growths that usually occur in the corpus of the uterus. Subserosal myoma (forms under the outer serous layer of the uterus and may become pedunculated); intramural myomas (grow within the myometrium) and submucosal myomas (endometrial layer and may cause excessive menstrual bleeding).
- Fibrous connective tissue surrounds the body of the tumor.

5 Meconium Aspiration Syndrome

- Amniotic fluid, stained with meconium, is aspirated by the fetus/neonate, causing acute respiratory distress.

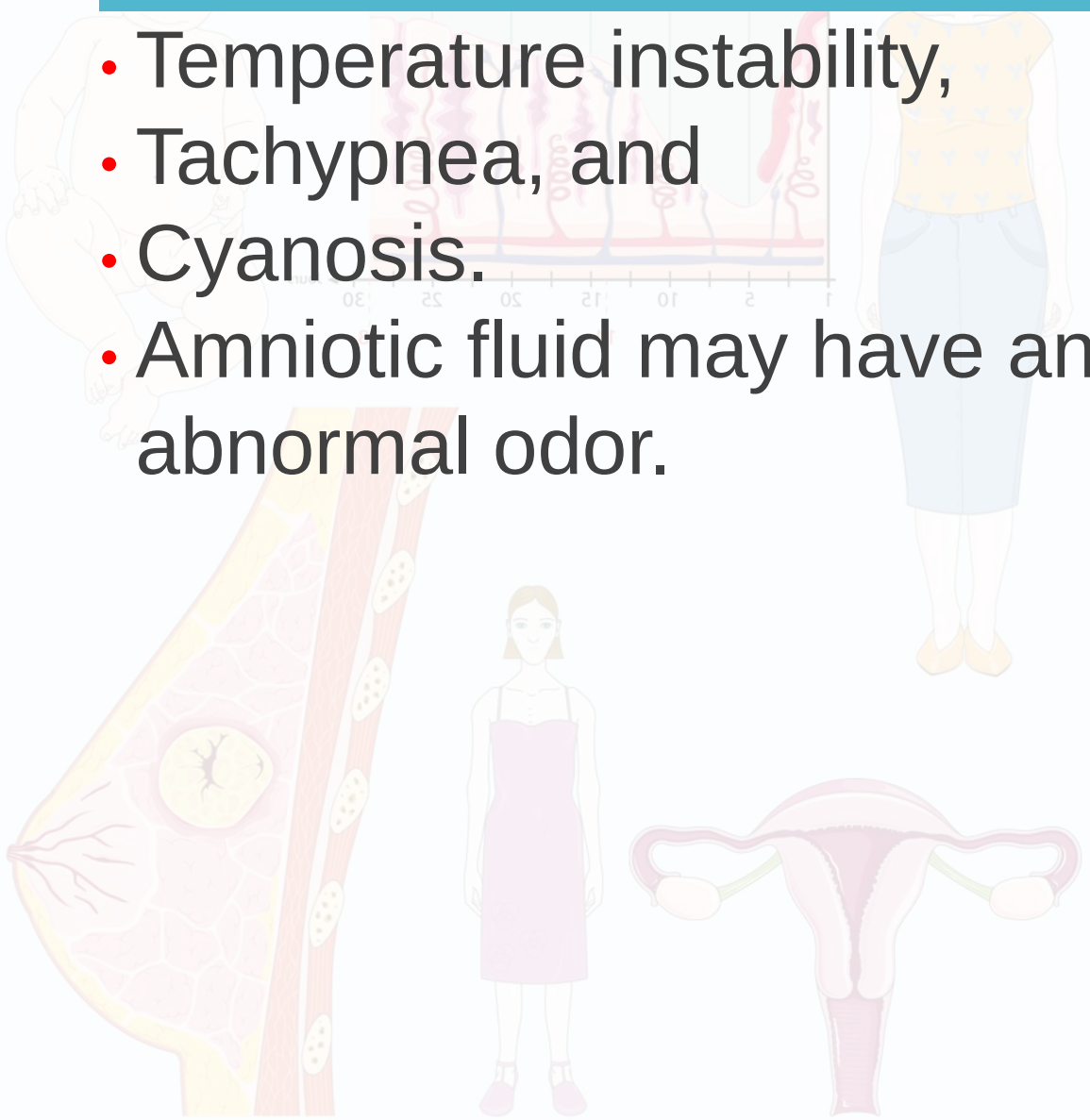


Pathophysiology

- Causes include intrauterine stress, such as PIH, postmaturity (aging placenta), intrauterine hypoxia and asphyxia, and infection.
- Fetal stress may produce increased intestinal peristalsis, anal sphincter relaxation, and expulsion of meconium into the amniotic fluid.
- Fetus breathing in utero, or with the first few breaths of air after delivery, causes aspirated meconium-stained fluid to enter the lungs.
- Alveoli of the lungs can be infiltrated by the meconium, causing atelectasis or the blockage of bronchiolar passages.

6 Neonatal Sepsis

- Temperature instability,
- Tachypnea, and
- Cyanosis.
- Amniotic fluid may have an abnormal odor.

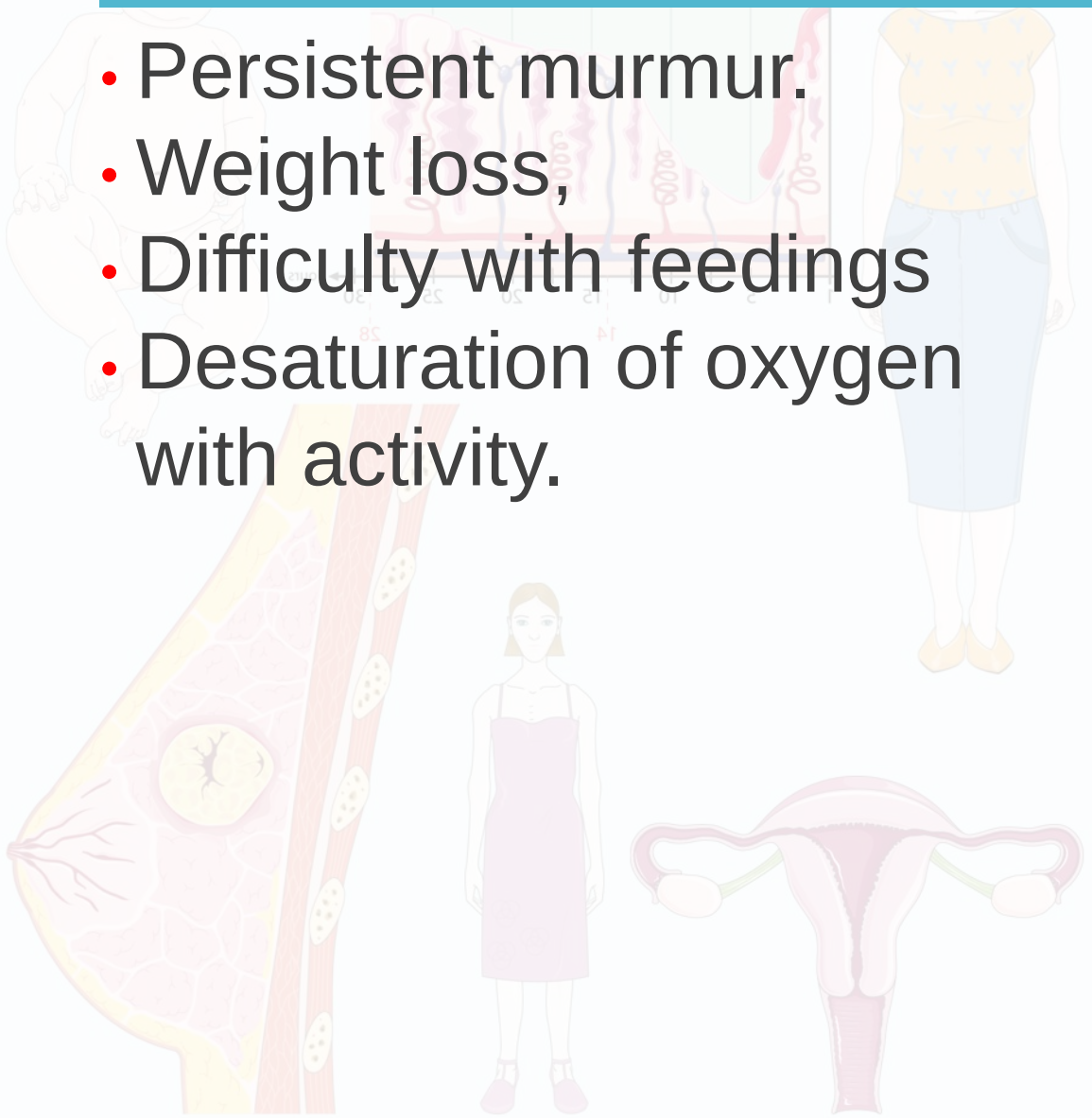


Pathophysiology

- Usually an ascending infection caused by Escherichia coli, Listeria monocytogenes, or group B streptococci related to premature rupture of membranes, maternal chorioamnionitis, or premature birth in which immunity is severely limited.
- Sepsis may occur as a result of invasive therapies (e.g., umbilical catheters; fetal surgery).

7 Patent Ductus Arteriosus

- Persistent murmur.
- Weight loss,
- Difficulty with feedings
- Desaturation of oxygen with activity.

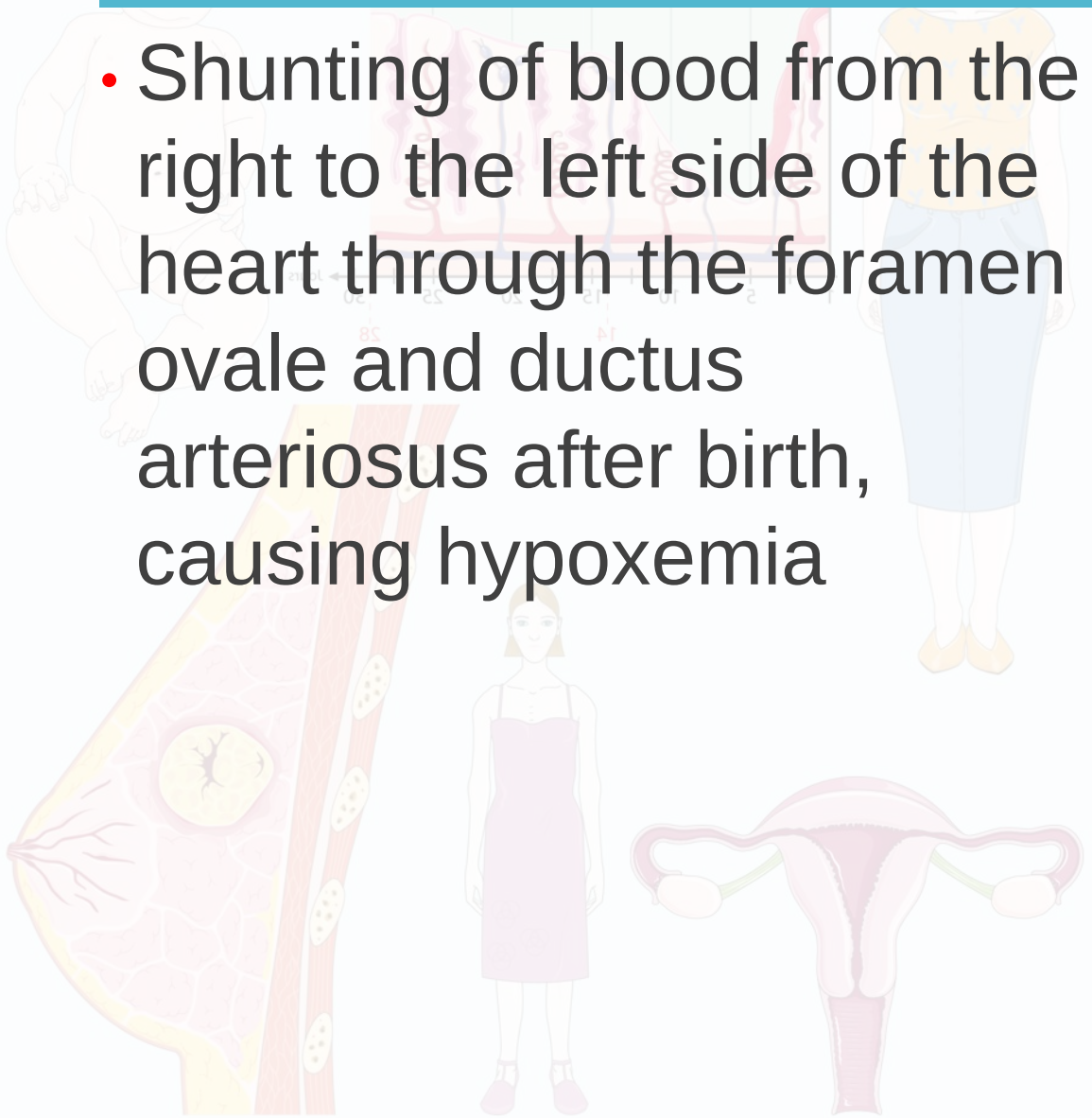


Pathophysiology

- Antenatal blood circulation includes three shunts one of which is ductus arteriosus, which shunts oxygenated blood from the pulmonary artery to the aorta (a right-to-left shunt).
- A PDA following birth will become a left-to-right shunt related to pulmonary vascular resistance causing excessive blood flow to the pulmonary area and left atria, as well as left ventricle congestion and decreased systemic flow via the aorta.
- Occurs in preterm infants; girls are affected more often than boys; more likely to occur in neonates with Downs syndrome or those exposed to rubella during gestation.

8 Persistent Fetal Circulation

- Shunting of blood from the right to the left side of the heart through the foramen ovale and ductus arteriosus after birth, causing hypoxemia

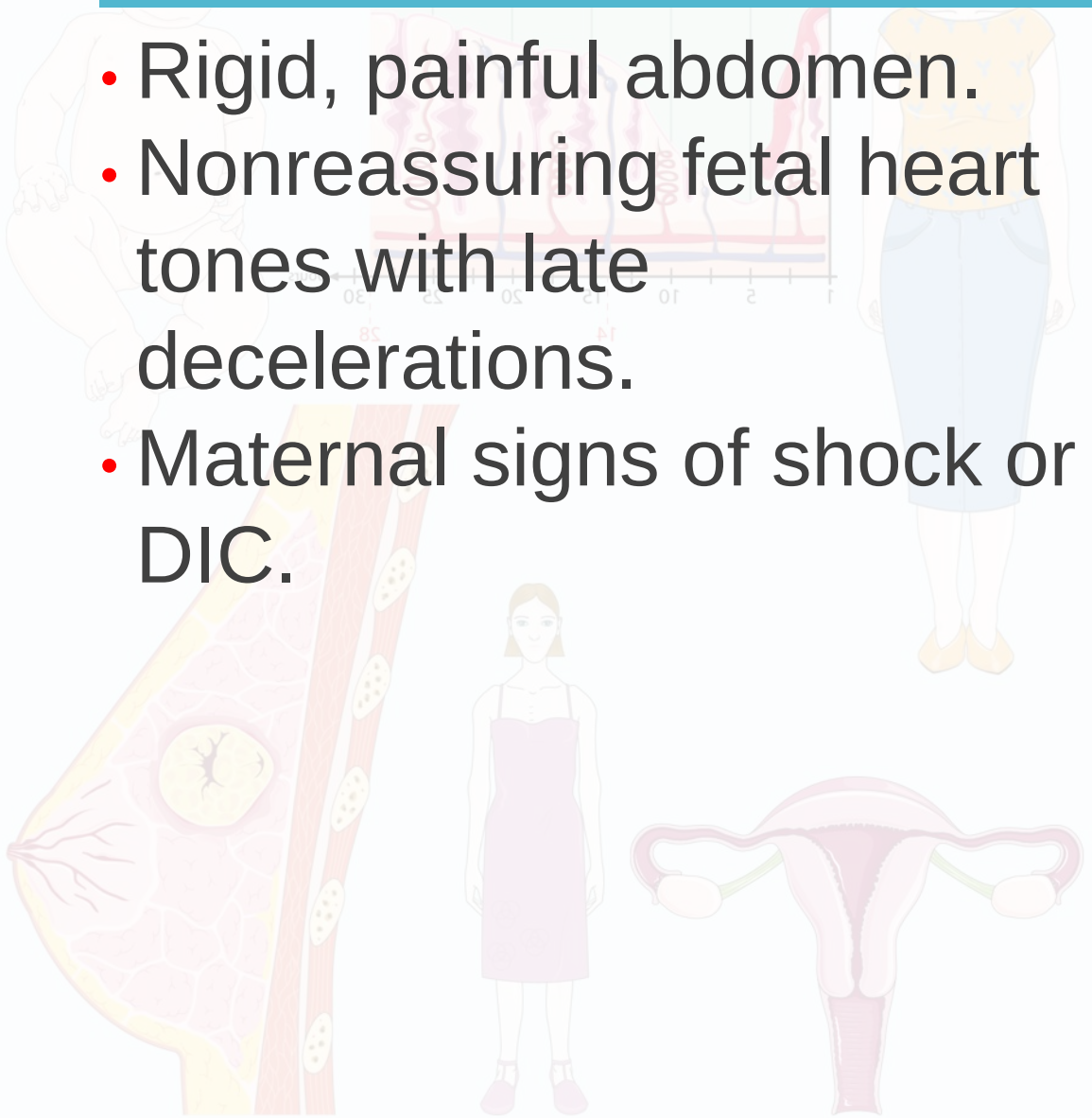


Pathophysiology

- Antenatal blood circulation includes three shunts. The lungs are bypassed by two shunts, the foramen ovale, and the ductus arteriosus. The last is the ductus venosus that bypasses the liver.
- Following birth and adequate ventilation pressures, the foramen ovale closes and the ducts collapse.
- Hypoxia and high carbon dioxide levels increase vasoconstriction in the lungs causing pulmonary hypertension that interferes with closure of the shunts and PFC occurs in small-for-gestationalage (SGA) neonates, infants of diabetic mothers (IDM), and those with a traumatic delivery.

9 Placenta Abruption

- Rigid, painful abdomen.
- Nonreassuring fetal heart tones with late decelerations.
- Maternal signs of shock or DIC.



Pathophysiology

- Placenta abruption (PA), the sudden dislodgment of the placenta from the uterine wall, is classified according to type and severity:
- Grade 1 PA (vaginal bleeding with mild uterine tenderness and mild uterine tetany, where only 10%–20% of the placenta is detached).
- Grade 2 PA (uterine tenderness and uterine tetany, with or without uterine bleeding, fetal distress, or maternal shock where 20%–50% of the placenta is detached).
- Grade 3 PA (severe uterine tetany and maternal shock, fetal demise is imminent or has occurred, maternal or fetal DIC, and more than 50% of the placental surface is detached).

10 Placenta Previa

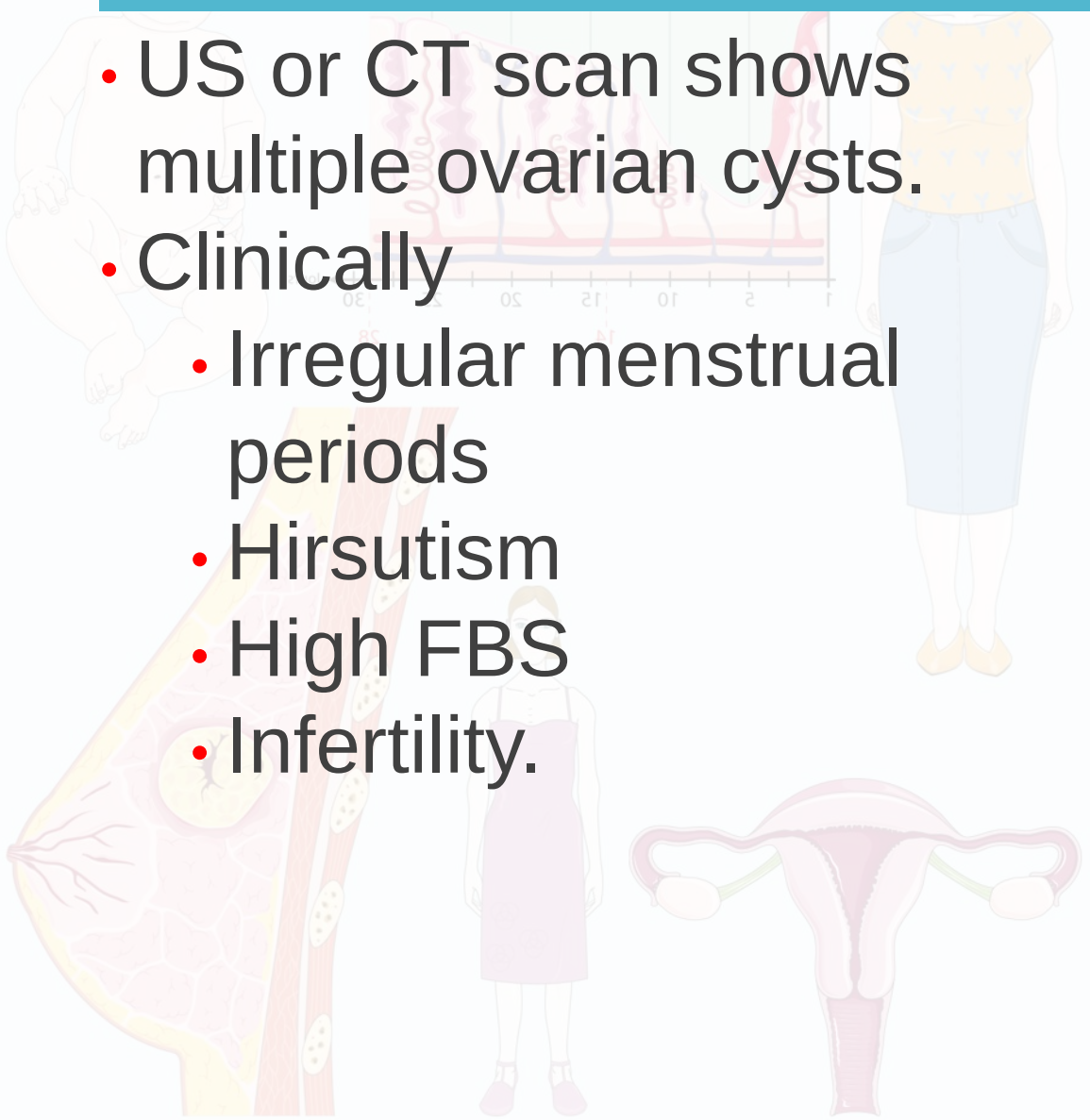
- Painless, frank red vaginal bleeding at or after 20 weeks' gestation.

Pathophysiology

- In PP, the placenta implants in the lower rather than the upper uterine area. When the cervix begins to dilate and move up the uterine wall in preparation for delivery of the fetus, the placenta may dislodge due to traction, causing bleeding and decreased oxygen delivery to the fetus.
- There are three types: PP centralis (total or complete PP) in which the placenta has been implanted in the lower uterine segment and completely covers the internal cervical os; PP lateralis (low marginal implantation) and PP marginalis (partial or incomplete PP).
- Risk factors include uterine scarring, previous pregnancy or cesarean section, Asian ethnicity, smoking, and age over 35 years.

11 Polycystic Ovarian Syndrome

- US or CT scan shows multiple ovarian cysts.
- Clinically
  - Irregular menstrual periods
  - Hirsutism
  - High FBS
  - Infertility.

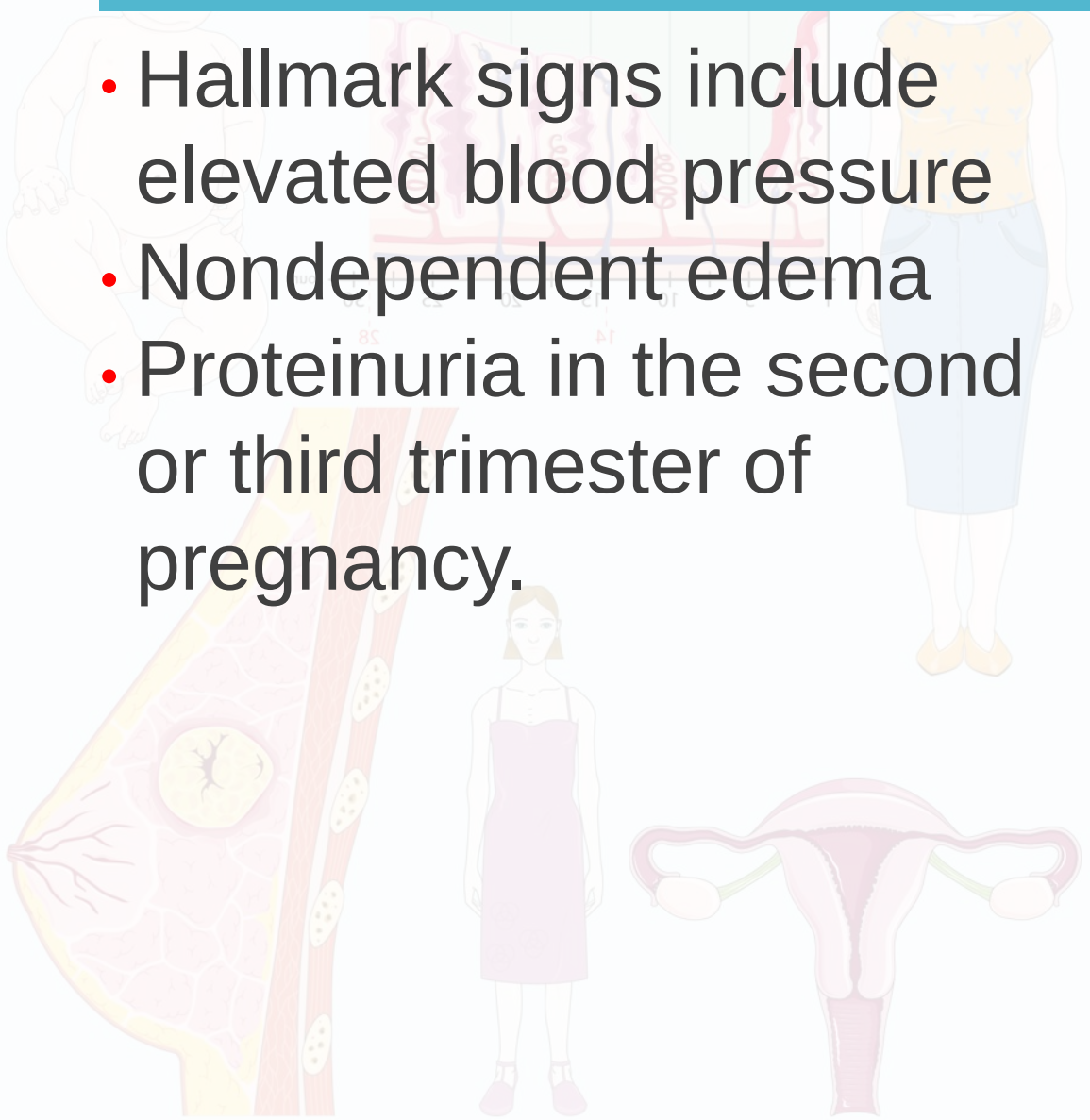


Pathophysiology

- PCOS is a genetically linked female endocrine disorder that results in chronic anovulation, hyperinsulinemia that triggers androgen hormone release, type 2 DM, lipid abnormalities, hirsutism and thinning scalp hair, infertility, and ovarian cysts.
- Etiology is unknown. Also known as Stein- Leventhal syndrome.

12 Pregnancy-Induced Hypertension

- Hallmark signs include elevated blood pressure
- Nondependent edema
- Proteinuria in the second or third trimester of pregnancy.

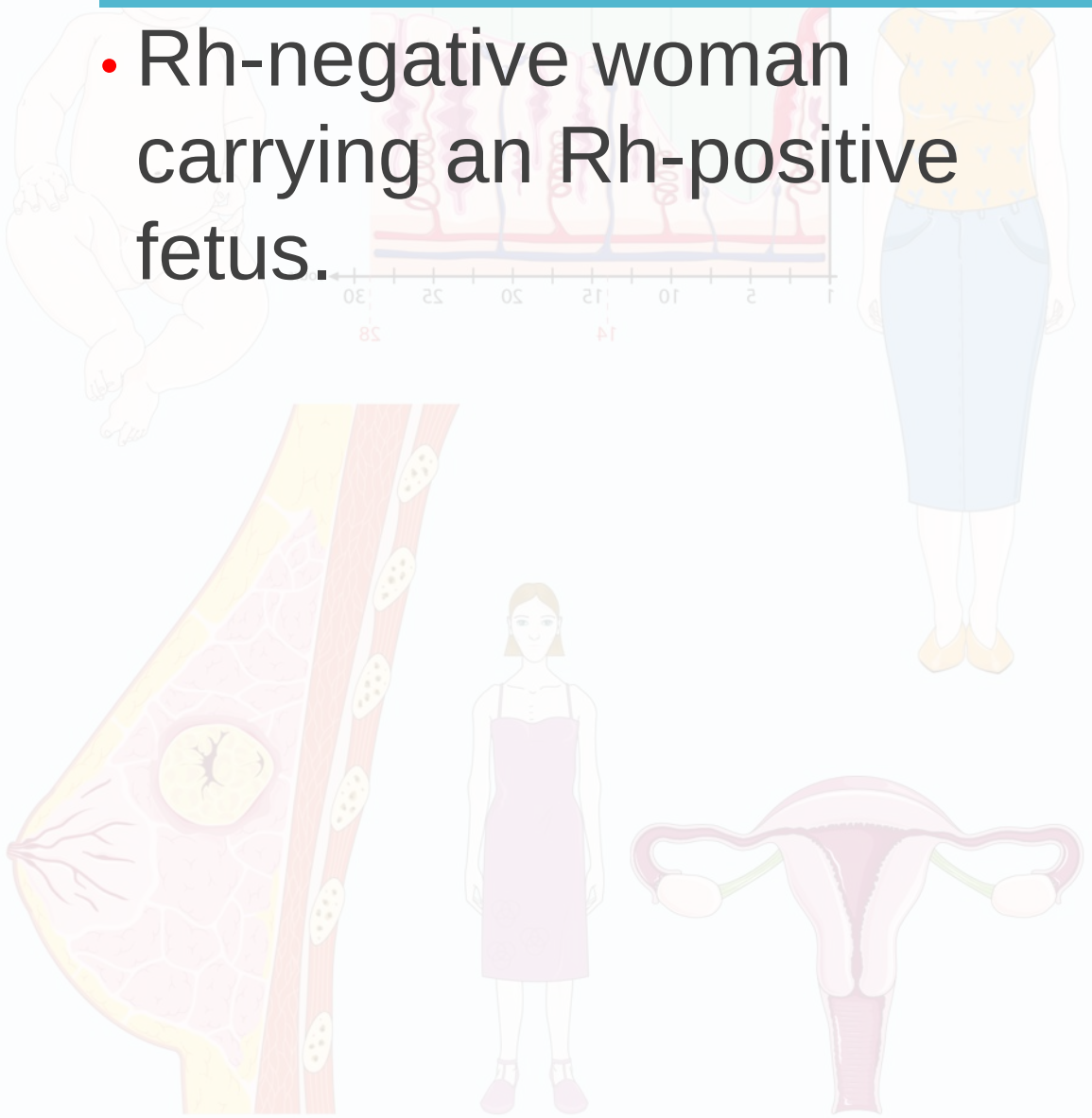


Pathophysiology

- PIH, also known as preeclampsia, may be caused by a vasospastic disorder of the placenta leading to a endothelial dysfunction and placental release of factors such as sFlt-1.
- Endothelial dysfunction leads to capillary permeability, resulting in nondependent edema, weight gain, pulmonary edema, hemoconcentration, edema in the retina, and edema in the brain tissue.
- BP ↑ is caused by an abnormal response to angiotensin II and epinephrine, and an imbalance among prostaglandins, prostacyclin, and thromboxane A2, resulting in vasoconstriction and vasospasm.
- Proteinuria is the result of HTN and subsequent damage to vessel walls.
- Severe PIH is characterized by HELLP syndrome.

13 Rh Incompatibility

- Rh-negative woman carrying an Rh-positive fetus.



Pathophysiology

- A pregnant woman with no Rh antigen, known as Rh0(D), on her RBCs is Rh negative. Additionally, there are no antibodies against the Rh antigen in her serum. If the fetus she is carrying is Rh positive, there should not be antibodies produced that cross the placenta. However, at delivery of the fetus, when maternal and fetal bloods finally mix, the maternal immune system is activated to produce anti-Rh antibodies. If no treatment is provided, the next Rh-positive fetus the woman carries will have its RBCs attacked by the anti-Rh antibodies, producing a condition called erythroblastosis fetalis, a potentially fatal condition in which most or all of the fetal RBCs are destroyed.

Dermatologic System Disorders

Clinical Medicine Flashcards

- Clinical Clues to Diagnosis
- Pathophysiology

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- Acne Vulgaris
- Cellulitis
- Contact Dermatitis
- Eczema
- Herpes Zoster
- Herpes simplex
- Impetigo
- Paronychia
- Pediculosis Capitis
- Psoriasis
- Rosacea
- Scabies
- Skin Cancer
- Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis
- Superficial and Partial-Thickness Burns
- Full-Thickness Burns
- Tinea
- Verruca

1 Acne Vulgaris

- Whitehead, blackheads, or cysts on the face, neck, upper back, chest, and shoulders, usually in adolescents, but can occur in adults.

Pathophysiology

- Skin disorder of the sebaceous glands and their hair follicles.
- Androgens, stress, strong soaps or cosmetics, or genetic factors cause an increase in sebum secretion.
- Pilosebaceous ducts are blocked with accumulated debris causing inflammation and bacterial infiltration.
- Lesions may be on face, neck, chest, upper back, and shoulders.

2 Cellulitis

- Patchy erythema and
- edema in the extremities in the absence of DVT.
- May be more prevalent in those with diabetes or peripheral vascular disease.

Pathophysiology

- Bacteria infiltrate skin, bypassing normal skin barriers, and release toxins in the subcutaneous tissue. Comorbid illnesses like diabetes and peripheral vascular disease increase the incidence.

3 Contact Dermatitis

- Patterned skin eruption after contact with an irritant or allergen

Pathophysiology

- Irritant contact dermatitis is caused by chemicals like detergents; allergic contact dermatitis is a cell-mediated type IV hypersensitivity reaction to an allergen (poison ivy, tape, and jewelry are some examples).
- Areas affected may show mild erythema or vesicles and bullae and are referred to as eczema.

4 Eczema

- Presence of a vesicular rash that may occur on any skin surface, including under the eyes.
- Asthma is often a comorbid illness.

Pathophysiology

- Type I hypersensitivity disorder, genetically linked, with a family history of hay fever, asthma, or atopic dermatitis.
- Infantile forms are vesicular, cheeks are pale, and Dennie-Morgan folds may be present under the eyes. Adults usually have dry leathery areas that are either more or less pigmented than surrounding tissue and appear in the antecubital and popliteal areas.
- The pruritic lichenified (dry, leathery) lesions can spread to the hands, feet, eyelids, and neck.

5 Herpes Zoster

- "Dew drops on a red leaf."
- Painful vesicular lesions unilaterally occurring along sensory nerve pathways.

Pathophysiology

- Caused by varicella zoster virus, identical to that which causes chickenpox.
- The herpes virus lies latent in the nerve tissue and reactivates with stress or decreased immunity or in clients with a malignancy or an injury to the spine or cranial nerve.
- Breakouts follow the course of sensory nerves, dermatomes, or cranial nerves and occur unilaterally. Eruptions last may last from 5 days to 5 weeks.
- Even when the lesions are gone, a postherpetic neuralgia exists and persists.

6 Herpes Simplex

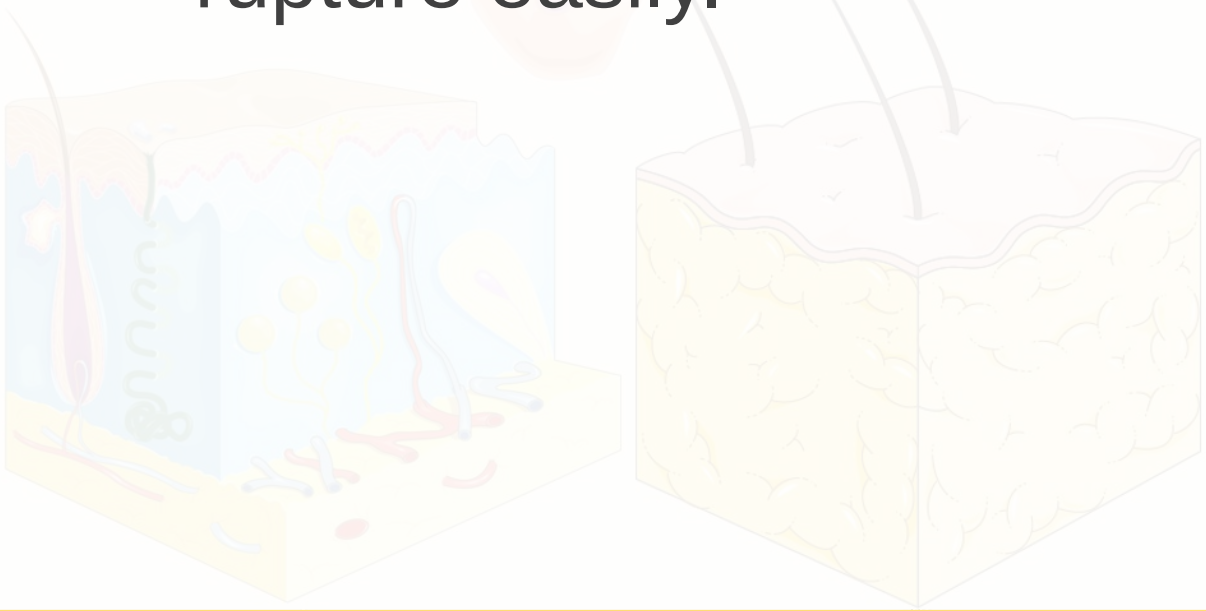
- Painful, itchy, vesicular lesion on the lips or in the nose.
- May also occur in the genital area.

Pathophysiology

- Caused by the herpes simplex virus (HSV). There are two types: HSV-I occurs above the waist, and HSV-II occurs below the waist.
- Occurs through direct contact, respiratory droplet, or fluid exposure. After exposure, the virus lies dormant in the nerve ganglia where the immune system cannot destroy it. Stress of any kind can cause the virus to reactivate.

7 Impetigo

- Honey-colored crusts on the lips mouth, nose, hands, or perineum; usually in children.
- Shallow vesicles that rupture easily.

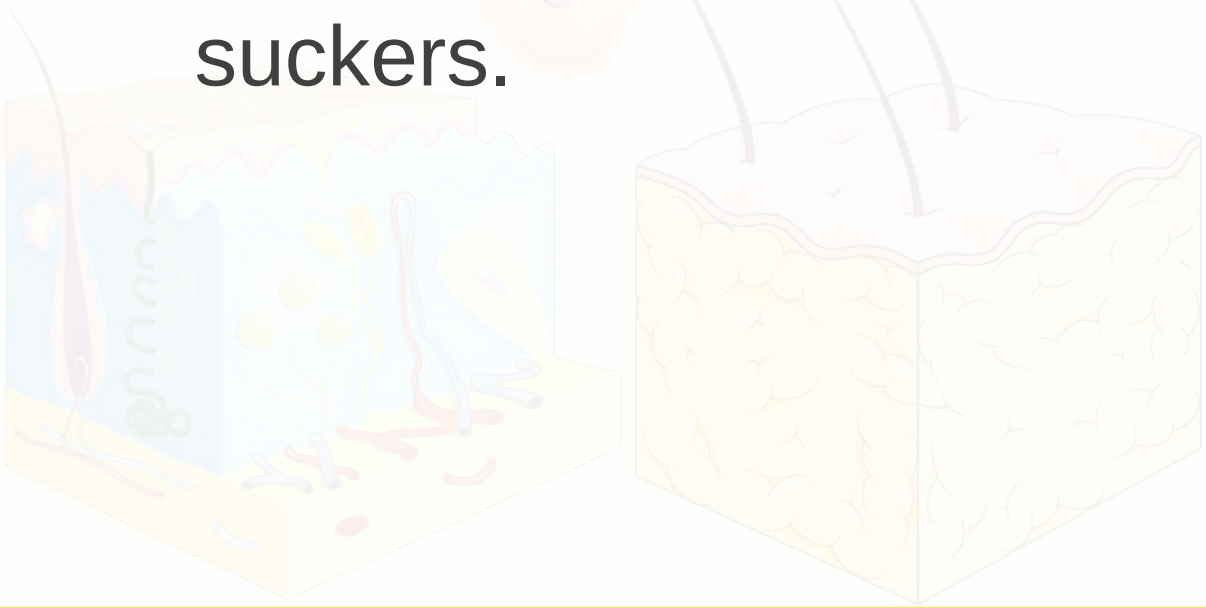


Pathophysiology

- Superficial bacterial infection of the skin caused by staphylococci, streptococci, or both.
- Vesicles or bullae are preceded by discolored spots.
- Vesicles or bullae rupture, leaving honey-colored crusts.
- Infection spreads outward.

8 Paronychia

- Erythema around the base of the nailbed in persons who frequently, as a result of employment, have their hands in water or are nailbiters or thumb suckers.

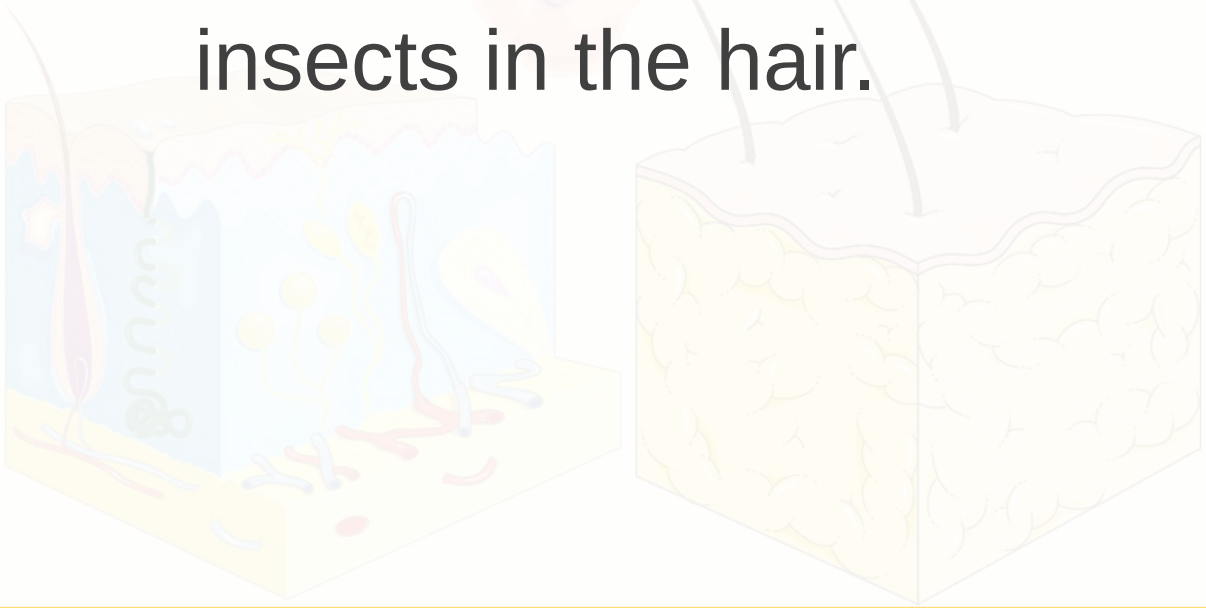


Pathophysiology

- Acute or chronic condition in which the protective barrier between the nail and the nail fold is breached by bacteria or fungus, causing erythema and pain.
- Can occur in persons whose hands are often in water and in those who engage in thumb sucking or nail/cuticle biting.

9 Pediculosis Capitis

- Itching of the scalp.
- Magnifying glass assessment reveals white or translucent eggs adhered to hair shaft near the skin or movement of insects in the hair.

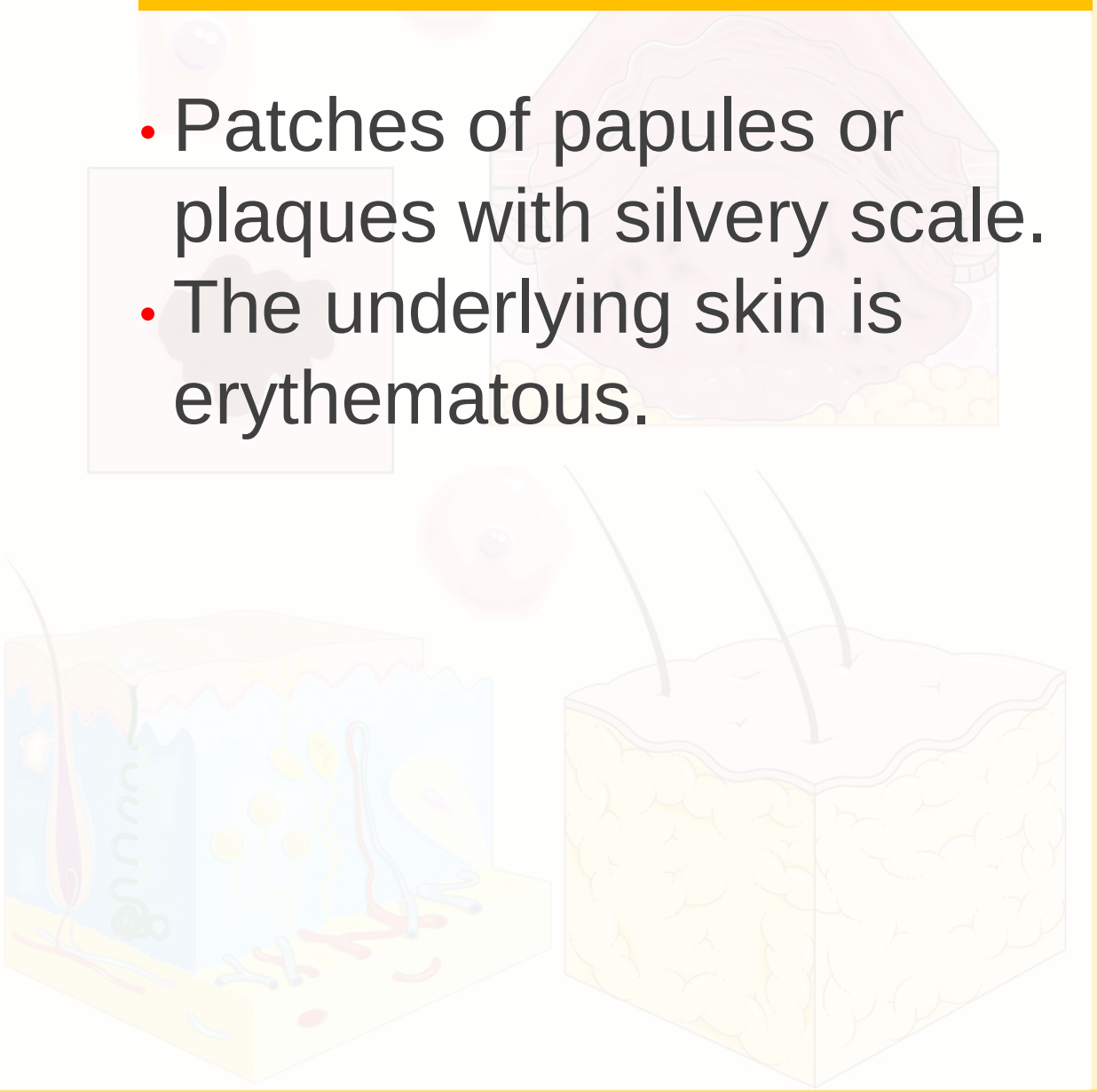


Pathophysiology

- Infestation of the head by Pediculus humanus capitis (head louse).
- Female louse lays eggs at night on a hair shaft close to the skin; eggs appear white or clear.
- Eggs hatch 7–10 days later.
- Itching of the scalp is produced by the insects crawling and by their saliva on the scalp. Lice bite and feed on human blood. They are approximately 2 mm in length.

10 Psoriasis

- Patches of papules or plaques with silvery scale.
- The underlying skin is erythematous.



Pathophysiology

- T cell–mediated autoimmune disorder characterized by silvery scale on an erythematous base. Abnormal growth of keratinocytes and dermal blood vessels.
- Precipitating factors may be any that stimulate the T-cell lymphocytes (e.g., trauma, stress, infections, and medications).
- Histologic studies show increased epidermal cell turnover; white blood cells (WBCs) are found in the stratum corneum.

11 Rosacea

- Client's skin appears flushed.
- States that sun exposure, eating or drinking hot foods or liquids, and alcohol make the condition worse.

Pathophysiology

- Chronic inflammatory process that often coexists with acne that looks like blushing.
- Thought to be caused by leakage of fluid and inflammatory mediators into the dermis. Inflammation may persist because of bacterial infiltration in the area.
- The main types of rosacea are the following:
- Telangiectatic (marked by the appearance of spidery blood vessels on affected skin).
- Papulopustular (bumpy/pustular lesions).
- Phymatous (nasal scarring and deformity).
- Ocular (involving the lids, lashes, or conjunctiva).
- The condition is common, especially in persons of Northern European ancestry. It usually is noted first between the ages of 30 and 50.
- Women are affected more often than men.

12 Scabies

- Grayish brown pruritic threadlike lesions with black dot at the end between fingers, toes, axillae, groin, buttocks, and abdominal areas.

Pathophysiology

- Impregnated female scabies mite burrows under the skin and lays eggs. An inflammatory response occurs 30–60 days after initial contact.

13 Skin Cancer

- Skin lesion with
  - Asymmetry
  - Irregular borders
  - Color changes
  - >6 mm diameter

Pathophysiology

- Mutation of cells of the skin that occurs from chronic exposure to the sun or other irritants.
- Basal cell cancer (the most common; cells in the lowest layer of the epidermis), squamous cell (cells of the middle layer of the epidermis), or melanoma (the most deadly form; melanocytes in the bottom layer of the epidermis).
- Cells lose normal functional properties and can metastasize to other organs (melanoma).

14 Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis

- Flu-like symptoms or macular rash after starting sulfa drugs, antibiotics, or antiepileptics.

Pathophysiology

- Altered drug metabolism causes a T cell–mediated reaction in the keratocytes.
- A macular rash spreads rapidly and forms vesicles and bullae in the epidermis and in the mucous membranes, which necrose and slough.
- Stevens-Johnson syndrome (SJS) involves 10% of body surface area, while toxic epidermal necrolysis (TEN) involves 30%.

15

Superficial and Partial-Thickness Burns

- Redness of the skin resembling sunburn or redness and mottling of the skin with blister formation after contact with a thermal source.

Pathophysiology

- Thermal or chemical injury to skin, the extent of which is expressed as body surface area.
- Superficial (first-degree) burns are likened to sunburn and affect only the epidermis. Partialthickness (second-degree) burns involve the epidermis and some portion of the dermis. Second-degree burns are characterized by blister formation.
- Deep second-degree burns take longer to heal and may cause scarring.
- First- and second-degree burns are painful because of intact free nerve endings for sensory pain transmission.

16 Full-Thickness Burns

- Tough, leathery, or charred skin surface that is brown, tan, red, or black.
- The skin does not blanch and is painless.

Pathophysiology

- Thermal, chemical, or electrical source destroys all layers of the skin (third-degree burns).
- Severe edema from protein loss and increased capillary permeability.

17 Tinea

- Reddened lesions that have a scaly appearance.
- Lesions may form circular areas, reddened raised areas, or, between toes, crevasses that can be deep and bloodless.

Pathophysiology

- Tinea infections or dermatophytoses occur when skin is impaired by exposure to a moist environment.
- Infections may occur through direct contact with infected humans, animals, or objects.
- Superficial mycotic infections include:
  - Tinea pedis.
  - Tinea capitis.
  - Tinea corporis.
  - Tinea versicolor.
  - Tinea cruris.
  - Tinea unguium.
  - Tinea barbae.

18 Verruca

- Pink or light pink growths that cluster on skin structures. Occasionally, these lesions are flattened and found on the facial area.

Pathophysiology

- Benign papillomas, or warts, are caused by human papillomaviruses (HPVs) that are spread through cracks in the skin; genital warts are spread by sexual contact.
- Various types are commonly seen:
  - Verruca vulgaris (common warts).
  - Verruca filiformis are found on the eyelids, face, and neck and project from the skin.
  - Verruca plana (flat warts).
  - Verruca plantaris (plantar warts).
  - Condyloma acuminata (genital warts).
  - These growths usually clear in time, but the immune system reacts very slowly to their presence.

Mental Health Disorders

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Clinical Medicine Flashcards

- Clinical Clues to Diagnosis
- Pathophysiology

- Alcoholism
- Attention Deficit-Hyperactivity Disorder (ADHD)
- Borderline Personality Disorder
- Conversion Disorder
- Depression
- Mania
- Bipolar Disease
- Dissociative Amnesia
- Generalized Anxiety Disorder
- Obsessive-Compulsive Disorder
- Panic Disorder
- Phobias
- Posttraumatic Stress Disorder
- Schizophrenia

1 Alcoholism

- Smell of alcohol on breath
- Ataxia
- Slurred speech
- Inappropriate affect; or shaking if abstinent

Pathophysiology

- Alcoholism is genetically linked.
- Alcohol is very lipid-soluble and enters the brain easily. Once there, it acts on GABA receptors, promoting a depressant and pleasurable effect. The action of other drugs (e.g., heroin) on the opioid and dopaminergic centers is similar to that of alcohol, cross-addictions occur.

2 Attention Deficit-Hyperactivity Disorder (ADHD)

- Child or adult with
  - Difficulty focusing
  - Finishing projects
  - Listening to instructions and
  - Sitting still who also shows emotional lability

Pathophysiology

- PET scans show decreased metabolic activity in the frontal lobes and basal ganglia; EEG readings show ↓ wave activity in the same area.
- PET scans show ↑ metabolism in the primary sensory and sensorimotor areas. There is no specific lesion. ADHD is believed to be an error in myelination.
- Affects boys and men more than girls and women.
- Inability to wait, impatience, bursts of anger, and an inability to sit still; difficulty finishing projects, focusing, and following directions and often appears to be staring off into space.

3 Borderline Personality Disorder

- Substance abuse; impulsive, “needy” behavior.
- Self-destructive behavior (suicide attempts) for attention.

Pathophysiology

- The personality develops as a normal part of neurophysiology, coupled with environmental factors. Components of the client’s genetic framework react to what is external, creating the outer and inner persona.
- Changes in the prefrontal cortex may be responsible for the personality changes exhibited by those with personality disorders. Affects women more than men.

4 Conversion Disorder

- Somatization of anxiety that results in paralysis, blindness, or other physical symptoms for which no medical explanation can be found.
- The client seems indifferent to the loss of function.

Pathophysiology

- A somatoform disorder in which neurologic symptoms (e.g., blindness, paralysis, loss of touch) may occur as a result of anxiety. According to the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision (DSM-IV-TR), symptoms cannot be intentional or explained by any medical tests.
- Impulses to the brain are misinterpreted or rerouted by an anxiety response, resulting in perceptual abnormalities. An anxiety- or stressproducing event precedes onset of the conversion disorder. Women are affected more than men.
- Neurotransmitters affected in this disorder are serotonin and norepinephrine.

5 Depression

- Persistent sadness hopelessness
- Feelings of guilt
- Inability to concentrate
- Decreased interest in daily activities
- Changes in appetite
- Insomnia or excessive sleep and
- Recurrent thoughts of death or suicide

Pathophysiology

- Changes in brain tissue metabolism and blood flow, particularly in the prefrontal cortex (decreased) and the amygdala (increased).
- Changes in the ability of receptors to bind with neurotransmitters (e.g., serotonin, norepinephrine); increase in reuptake of neurotransmitters before they can bind with receptors and increased destruction of neurotransmitters by monoamine oxidase, which deaminates serotonin and norepinephrine.
- Less ability to handle stress related to altered hypothalamus-pituitary-adrenal system.

6 Mania

- Mental disorder characterized by excessive excitement, restlessness, delusions of grandeur, and poor judgment.

Pathophysiology

- According to the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision (DSM-IV-TR), the client must have experienced at least three persistent episodes of grandiose thoughts, excessive need to speak characterized by flight of ideas, decreased need for sleep, poor judgment, and irritability.
- Imbalance in levels of norepinephrine, serotonin, dopamine, and hormones.

7 Bipolar Disorder

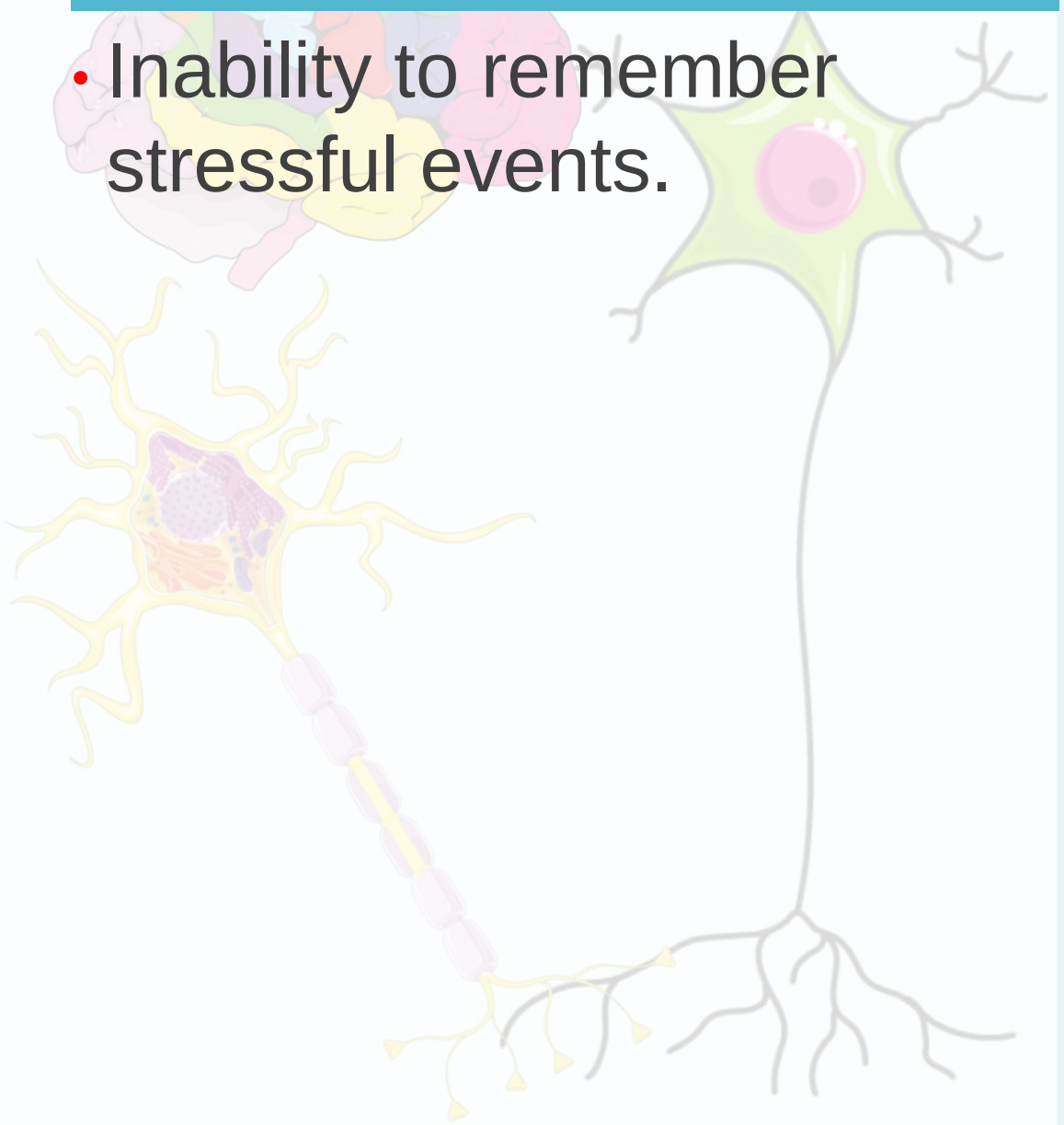
- Cycling through periods of depression and mania.
- Rapid cycling (four episodes per year) indicates a more severe illness.

Pathophysiology

- Changes in brain tissue metabolism and blood flow, particularly in the prefrontal cortex (decreased) and the amygdala (increased). Strong genetic link, and women are affected more than men.
- Imbalance in neurotransmitters. Epinephrine and norepinephrine are increased in the manic phase, and serotonin and norepinephrine are decreased in the depressive phase.
- Less ability to handle stress (hypothalamuspituitary- adrenal system).
- Sleep disturbances related to neurotransmitter imbalances.

8 Dissociative Amnesia

- Inability to remember stressful events.



Pathophysiology

- A dissociative disorder is caused by a traumatic occurrence. The areas of the brain associated with memory recall and storage (the limbic and hippocampal areas) may be traumatized by childhood events or by unbearable events later in life.
- According to the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision (DSM-IV-TR), the client must have experienced at least two occurrences of amnesia for an event as well as impaired social or familial processes.
- Repression.

9 Generalized Anxiety Disorder

- Excessive worry or anxiety that cannot be controlled, causing interference with normal activities of daily living.
- Symptoms must have occurred for at least 6 months.

Pathophysiology

- Anxiety is produced by stimulation of the autonomic nervous system. Neurotransmitters involved in the anxiety response include gammaaminobutyric acid (GABA), serotonin, epinephrine, and norepinephrine. A prolonged, abnormal fight-or-flight response occurs to normal stimuli.

10 Obsessive-Compulsive Disorder

- Rituals are performed a specific number of times and in a specific sequence to decrease unpleasant thoughts.

Pathophysiology

- According to the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision (DSM-IV-TR), an obsession involves recurrent, intrusive, and persistent thoughts, impulses, or images that cause excessive anxiety. The obsession is known to be irrational yet cannot be ignored. Attempts to suppress the obsession become rituals known as compulsions.
- The DSM-IV-TR defines a compulsion as a repetitive act or ritual.
- The client with obsessive-compulsive disorder (OCD) spends a great deal of time on the ritualistic behavior.
- There is a genetic predisposition for OCD.

11 Panic Disorder

- Sudden feeling of impending doom, going crazy, unreality, and fear accompanied by
- Palpitations
- Numbness of the arms
- Chest discomfort anddizziness

Pathophysiology

- Symptoms are recurrent.
- The cycle of panic is attributable to “fear of the fear.” Dreading an attack brings one on.
- Physical symptoms are related to the sympathetic and adrenal systems.
- Several hypotheses exist as to cause: a disorder in serotonin sensitivity, hypersensitivity to catecholamines, sensitivity to lactate, decreased inhibition to GABA, hypersensitivity in neuroanatomy producing abnormal signals for fight or flight, and genetics.

12 Phobias

- Irrational fear of an object, place, situation, thing, or person that causes avoidance behaviors.

Pathophysiology

- According to the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision (DSM-IV-TR), phobia development is strongly associated with anxiety disorders. A phobia occurs when an object, place, situation, thing, or person causes a sympathetic nervous system (autonomic) response that results in anxiety. The trigger of anxiety becomes a phobia.
- A phobia can become so severe that all social contact is lost.
- A simple phobia is one associated with fear of common things (e.g., spiders, heights).

13 Posttraumatic Stress Disorder

- Acute anxiety and distress related to flashbacks or memories of a traumatic event.

Pathophysiology

- Severe psychological distress after traumatic events (e.g., war, criminal assault, accidents, natural disasters, rape).
- The amygdala of the brain is hyperactive in PTSD.
- Activation of the amygdala causes activation of the autonomic nervous system, and the adrenal system. The sympathetic nervous system produces many of the symptoms of PTSD, which are prolonged by the adrenal hormones.
- According to the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision (DSM-IV-TR), symptoms must last at least 1 month. Onset may occur at any time after the traumatic event.

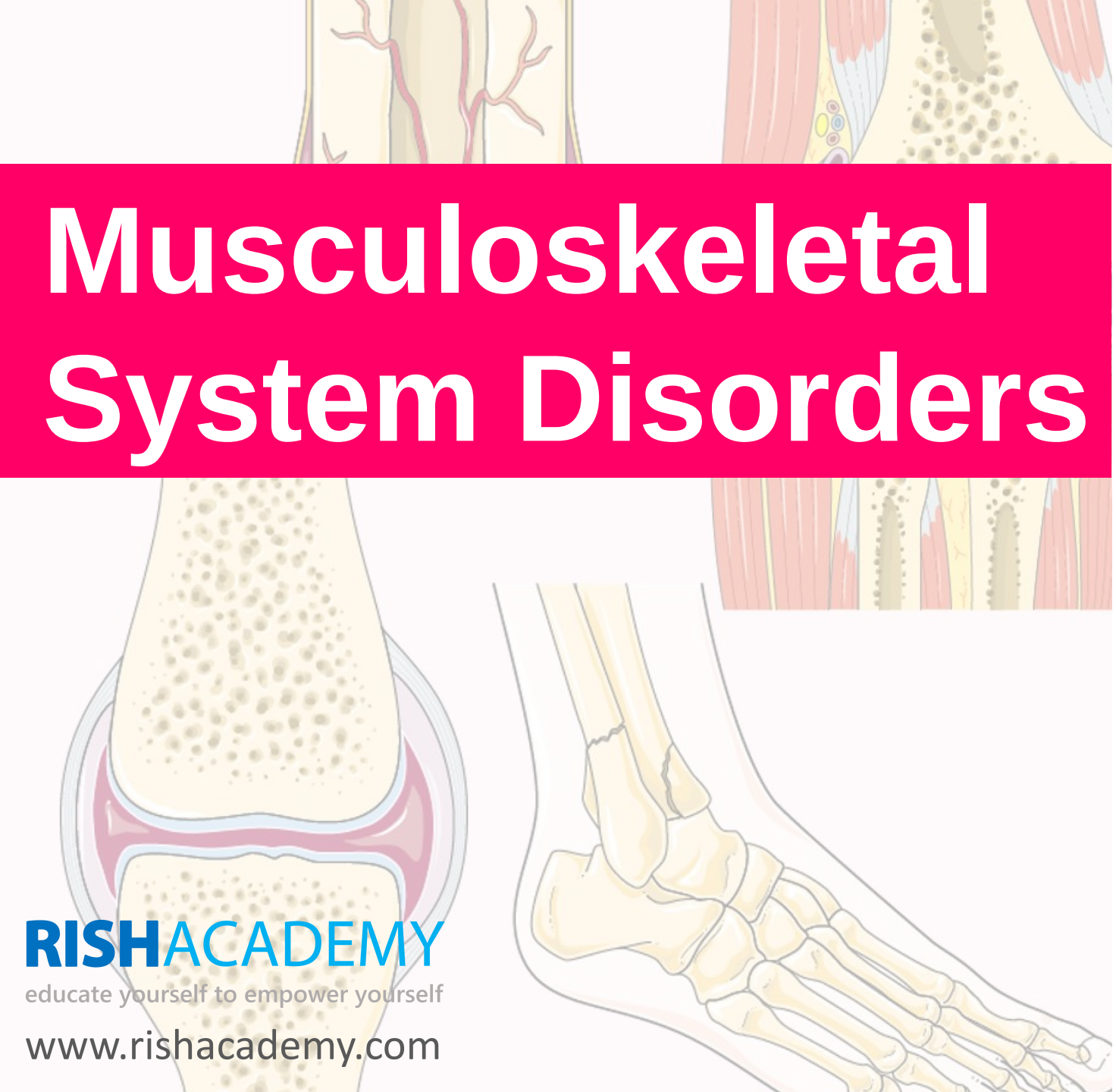
14 Schizophrenia

- Often described by the 4 As (autism, avolition, anhedonia, and associative looseness).
- Schizophrenia means “split mind,” with a chasm occurring between the client and the environment.
- High dopamine levels are present.

Pathophysiology

- The neurotransmitter dopamine is excessively abundant. Changes in brain metabolism.
- Genetic links are not as strong as once thought but still place relatives at greater risk of developing schizophrenia (onset adolescense; early adulthood).
- Alterations in perception and thought, including delusions (fixed thoughts) and hallucinations (auditory is the most common but can involve all the senses).
- Difficulty with expression of thought.

Musculoskeletal System Disorders



Clinical Medicine Flashcards

- Clinical Clues to Diagnosis
- Pathophysiology

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- Compartment Syndrome
- Fracture
- Gouty Arthritis
- Herniated Nucleus Pulposus
- Osteoarthritis
- Osteomalacia
- Osteomyelitis
- Osteoporosis
- Paget’s Disease
- Rheumatoid Arthritis
- Sprain
- Total Joint Replacement

1 Compartment Syndrome

- Neurovascular assessment of injured area usually by crush injury or fracture
- Severe pain
- Pallor
- Pulselessnessa
- Paresthesi
- Paralysis
- Coolness to the touch.

Pathophysiology

- In a traumatic injury (e.g., fractures caused by automobile accident or crush injury), soft tissue swelling occurs. The soft tissue in this case is the muscle. Every muscle compartment is surrounded by connective tissue called fascia.
- Fascia compresses the swelling tissue, causing loss of vascularity to tissue and nerves. The muscle tissue is essentially being strangled in its own covering.
- Less problematic compartment syndrome is seen in exertional compartment syndrome and stress fracture.

2 Fracture

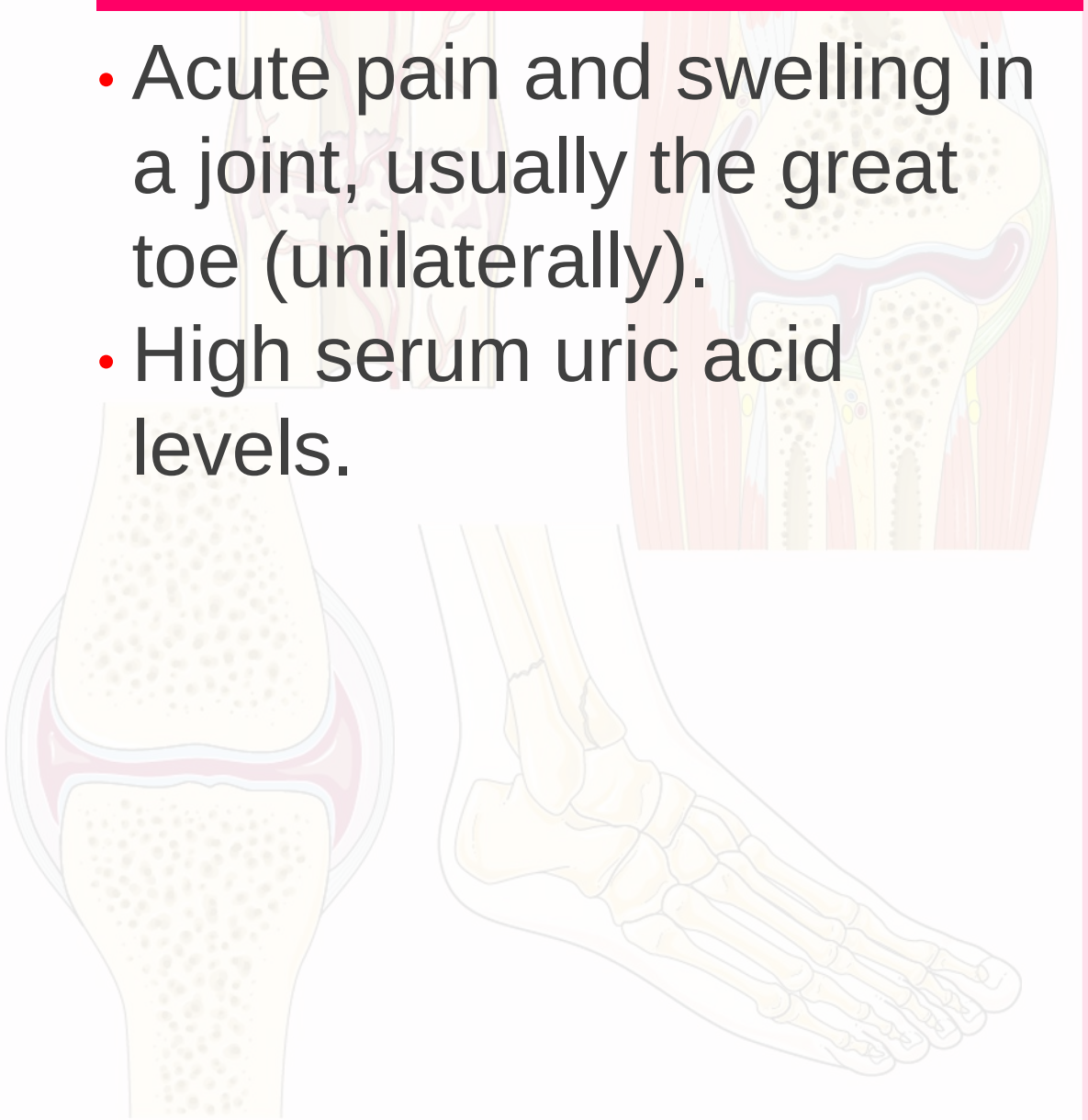
- Tenderness, pain, or deformity over an area of injury
- Crepitation may be heard or felt
- Range of motion is decreased

Pathophysiology

- Healthy bone is living tissue that is dynamic in nature. Osteoclasts resorb bone, while osteoblasts lay down new bone. In this way, healthy bone is always remodeled.
- A fracture is a disruption in the bone structure caused by trauma or pathology.
- Closed fractures do not disrupt the integrity of the skin; open fractures are called compound because they break through the skin as well as disrupt the integrity of the bone.
- Fracture types are comminuted, impacted, greenstick, oblique, longitudinal, and transverse.
- Fractures heal by forming a blood clot at the site and attracting cells to the site. The fracture site is known as a callus at week 1, and by week 6, osteoclasts have resorbed dead bone and osteoblasts have remodeled the site. Complete healing is usually in 1 year.

3 Gouty Arthritis

- Acute pain and swelling in a joint, usually the great toe (unilaterally).
- High serum uric acid levels.



Pathophysiology

- In gouty arthritis, uric acid crystals are deposited in the joints and other connective tissues. The concentration in synovial fluid is higher than in plasma, so the crystals cause excessive inflammation in the joint. Joints affected are usually those at the distal area of the body, as uric acid crystals are affected by gravity. Called “the rich man’s disease” because many of the foods that contain purines are considered those consumed by the wealthy.

4 Herniated Nucleus Pulposus

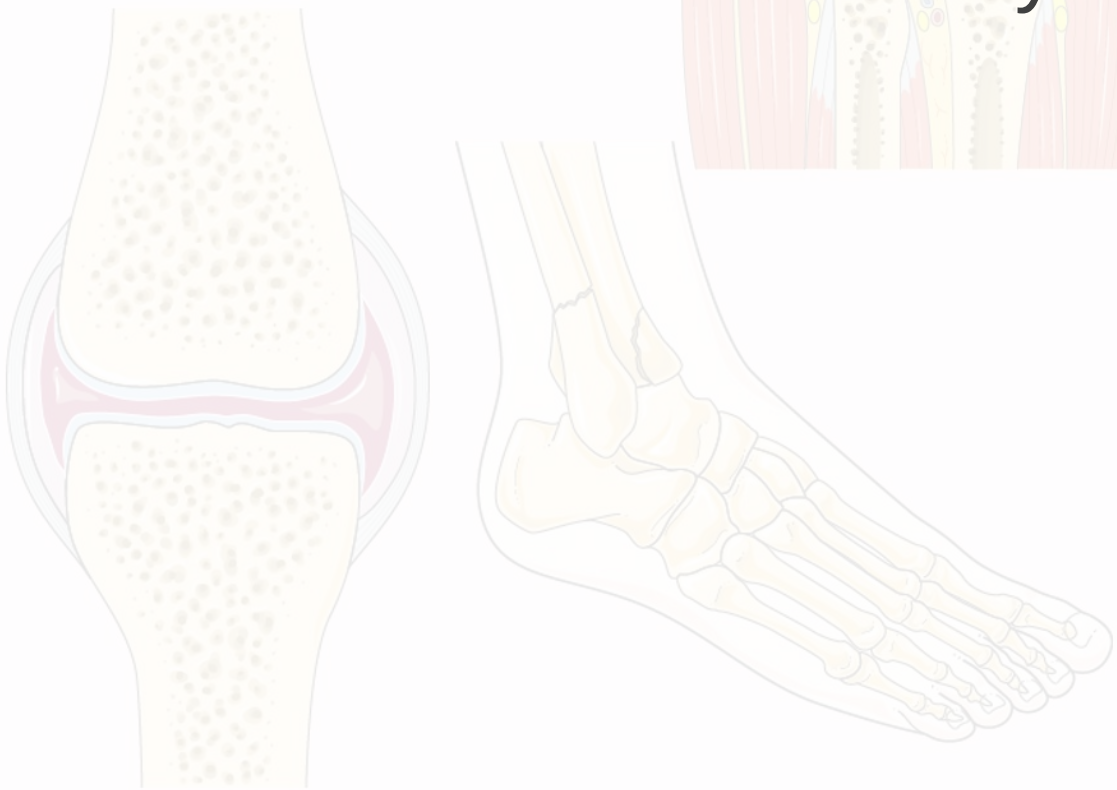
- Pain and numbness in the arm
- Headaches on the affected side (cervical)
- Pain and numbness radiating down the sciatic nerve in the leg (lumbar)

Pathophysiology

- The vertebrae have cushions or intervertebral disks between them to absorb shock and to keep the nerve roots away from the boney areas.
- Disks can herniate out of the normal position, and the annulus fibrosus tears. The inner portion (nucleus pulposus) pushes outward and places pressure on a nerve root.
- The most common sites are the cervical and lumbar areas.

5 Osteoarthritis

- Pain and stiffness in the weightbearing joints and the vertebral column due to wear and tear or obesity.



Pathophysiology

- The matrix of cartilage is composed of chondrocytes imbedded in proteoglycan molecules, which are large and osmotic, drawing fresh synovial fluid into the joint.
- With excess wear and tear, the chondrocytes become inflamed and release inflammatory mediators (cytokines), causing a cascade of events that includes formation of protease, which break down the proteoglycan molecules. Eventually, the cartilage becomes worn and misshaped. Streaks and dents in the cartilage become cracks. Synovial fluid leaks into the underlying bone, causing cysts. The underlying layer of the cartilage can no longer be an effective shock absorber. Bone spurs form.

6 Osteomalacia

- Softening of bone causing bowed legs in children and soft or brittle bones in adults.
- Bone pain is often present with muscle weakness.

Pathophysiology

- Bone mineralization is diminished because of lack of calcium or vitamin D.
- Vitamin D absorbed by exposure of the skin to sunlight must be activated by two organs, first the liver and then the kidney. Any disorders of these organs decreases vitamin D availability.
- The disease is more prevalent in women because of their increased need for calcium.
- Osteomalacia is seen more frequently in persons with low sun exposure.

7 Osteomyelitis

- Increased temperature with pain and inflammation over the affected bone
- Elevated WBC and ESR.
- Bone biopsy positive for infection.

Pathophysiology

- Bone infection with microorganisms that can occur in compound fracture as well as in surgical intervention (direct inoculation), spread from surrounding tissue (e.g., cellulitis [contiguous spread]), and infection of the bone from sepsis (hematogenesis).
- When the bone becomes infected, the inflamed area forms an abscess that impairs blood flow to the intramedullary area. Bone death occurs, the periosteum peels away from the ostium, and dead bone (sequestrum) forms. The sequestrum can fall from the bone, causing more pressure and decreased blood flow to other boney areas.
- Sinuses commonly form that allow pus and debris to escape from the bone to the outer skin.

8 Osteoporosis

- Loss of bone density (by DEXA scan) seen mainly in females who weigh less than 140 lb at menopause and have never used estrogen-replacement therapy.
- The mnemonic is ABONE (A = age, B = bulk, ONE = one never on estrogen).

Pathophysiology

- Healthy bone is living tissue that is dynamic in nature. Osteoclasts resorb bone, while osteoblasts lay down new bone. In this way, healthy bone is always remodeled. Bone remodeling occurs under the influence of hormones and from normal body movement and weight-bearing activities.
- At menopause, estrogen withdrawal causes inflammatory mediators and immune cells that encourage the differentiation of osteoclasts and increase their life span. Osteoblasts, or bone creators, are less active.
- As osteoporosis progresses, this trabecular framework is diminished and may be totally resorbed. Bone density decreases.

9 Paget's Disease

- Enlarged bone mass & deformity of the femur, skull, vertebrae, or pelvis
- Usually in older adults
- Increased serum ALP,
- Increased urine hydroxyproline (measured by pyrilinks and osteomark)
- Increased urine and serum calcium.

Pathophysiology

- A disease of older adults in which osteoclastic activity is followed by an exaggerated response by osteoblasts resulting in enlargement of bone.
- There are three phases: (1) the active phase (reabsorption); (2) the mixed phase (osteoblast activity); and (3) the inactive phase, in which the osteoblastic phase has exceeded the osteoclastic activity.
- Increased vasculature around the bones.
- The femur, skull, vertebrae, and pelvis are most often affected.

10 Rheumatoid Arthritis

- Swan neck deformity or ulnar deviation of the metacarpophalangeal joints.
- Fatigue, flu-like symptoms may accompany exacerbations of
- The disease
- Elevated ESR
- RF & CCP antibodies present

Pathophysiology

- Rheumatoid arthritis is a systemic disease known to be genetic and autoimmune in nature. Women are affected more than men.
- Rheumatoid factor (RF) antibodies react with IgG, forming immune complexes in the body and in the synovial joints.
- Granulocytes phagocytize the immune complexes and release toxins into the tissue and into the joints.
- Synovitis occurs as well as increased formation of blood vessels in the synovial walls, which contributes to production of vascular pannus.
- The area of inflammation is “walled off” in an attempt to heal injured tissue causing more immobility and destruction.

11 Sprain

- Pain,
- Swelling &
- Heat
- Around a recently injured joint
- Usually the ankle.

Pathophysiology

- The ligaments, the strong connective tissue bands that secure bone to bone, are affected. The most likely joint is the ankle.
- The ligaments may be slightly torn or completely torn with disconnection from and a piece of the bone attached to the torn ligament.

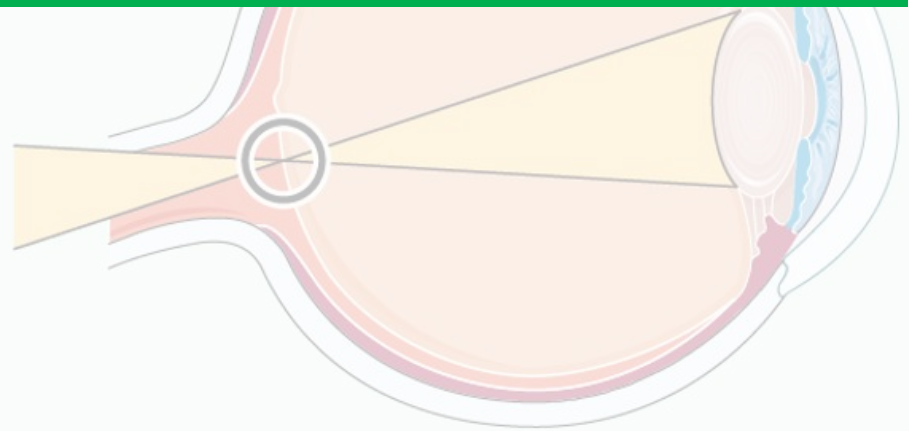
12 Total Joint Replacement

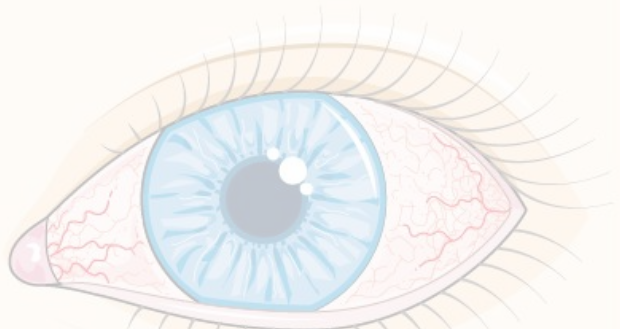
- Replacement of the femoral head and placement of an acetabular cup (THR) (hip), or replacement of the femoral and tibial ends of the knee by metal and the knee cap by a button (TKR).

Pathophysiology

- Persons with problems like chronic osteoarthritis pain, avascular necrosis of the femoral head, or systemic lupus erythematosus (SLE) cannot perform activities of daily living (ADLs) and require the joint be replaced by prosthetic devices.

Sensory System Disorders





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Clinical Medicine Flashcards

- Clinical Clues to Diagnosis
- Pathophysiology

- Acoustic Neuroma
- Acute Angle-Closure Glaucoma
- Cataracts
- Conductive and Sensorineural Hearing Loss
- Diabetic Retinopathy
- Labyrinthitis
- Macular Degeneration
- Mastoiditis
- Ménière’s Disease
- Otitis Externa
- Otitis Media
- Otosclerosis
- Primary Open-Angle Glaucoma
- Retinal Detachment

1 Acoustic Neuroma

- Hearing loss
- Headache (wakes the client or is worse with sneezing or coughing)
- Facial numbness
- Balance problems
- Tinnitus.

Pathophysiology

- Benign tumor of the Schwann cells of cranial nerve VIII.
- Linked with neurofibromatosis type 2.

2 Acute Angle-Closure Glaucoma

- Unilateral redness and pain in the eye
- Headache, nausea, and vomiting
- Client may see halos in the visual field around lights
- Tonometry measurement may exceed 50 mm Hg

Pathophysiology

- Highest risk group is Asian or Inuit women over age 45 years or persons with nearsightedness.
- In glaucoma, the anterior chamber experiences outflow problems, with fluid and pressure increases. Because it is an enclosed fibrous capsule, the eye is unable to swell without causing pressure on important structures like the choroid retina and optic nerve.
- Causes the outflow area of the iris/corneal angle to become narrow because of bunching of the iris as the pupil dilates. Prolonged pupil dilation, can cause an episode of mild or emergent severity. A mild episode may be relieved by sleep and relaxation.
- Trauma to the eye can also produce the same type of symptoms, which create a medical and surgical emergency.

3 Cataracts

- Difficulty driving at night because of excessive glare.
- Opacity of the lens on ophthalmologic examination.

Pathophysiology

- Opacity of the lens can occur at any age, including congenitally. However, most cataract formation occurs over age 40 years and most commonly in the elderly. Types include subcapsular; nuclear; and cortical.
- In nuclear (age-related) cataract formation, the center and outer areas of the lens start to produce more protein strands that begin to aggregate in the center portion of the lens and form strata by folding. As strata forms, the center portion of the lens opacifies and yellows as the protein fibers accumulate.
- Women who take HRT are at a greater risk, and women who take HRT and consume significant amounts of alcohol are at an even greater risk.
- Exposure to UV light is another risk factor.

4 Conductive and Sensorineural Hearing Loss

- Acquired or congenital inability to discriminate sound, resulting in impaired hearing.
- History of ototoxic drug use.
- Weber’s and Rinne’s tests are abnormal.

Pathophysiology

- Hearing loss is a common problem in the elderly, but it may occur at any age.
- Efficient hearing is accomplished by appropriate conduction of sound into the inner ear. Conduction of sound is accomplished by vibration of the tympanic membrane that is connected to the malleus. The malleus, incus, and stapes transmit the vibration to the oval window of the inner ear. The oval window vibrates and causes movement of endolymph within the cochlea that stimulates sensorineural receptors within the cochlea. Transmission to the acoustic nerve sends information to the brain for interpretation.
- Conductive hearing loss can result from increased cerumen, foreign bodies in the ear canal, cysts, tumors, otosclerosis, or stiffened or scarred tympanic membrane.
- Sensorineural hearing loss occurs through damage to sensory nerves caused by complications of infections, use of ototoxic drugs, neuromas, arteriosclerosis, chronic exposure to noise, and aging.

5 Diabetic Retinopathy

- Those with a history of poorly controlled diabetes experience gradual central visual field changes that can progress to flashing lights and cessation of vision (retinal detachment).
- On ophthalmoscopic examination, cotton-wool spots and tortuous, dilated vessels are seen.

Pathophysiology

- Background retinopathy is caused by microaneurysms that form on the retinal capillaries and leak blood. The client may experience visual changes caused by inflammation.
- The preproliferative stage of retinopathy is characterized by edema of the retina with blocked and infarcted blood flow.
- The proliferative stage of retinopathy is characterized by twisting of vessels, with neovasculature growing into the optic disk and obscuring the retina. The neovasculature leaks easily. Traction may occur as a result of the twisting and leaking of these vessels and cause retinal detachment.
- Disorder has a genetic link.

6 Labyrinthitis

- Vertigo
- Tinnitus.
- Weber’s and Rinne’s tests indicate conductive or sensorineural hearing loss.
- Fever, elevation in WBCs, nausea, and vomiting may occur.

Pathophysiology

- Inflammation of the inner ear caused by bacterial or viral microorganisms that enter the inner ear from the middle ear, meninges, or bloodstream.
- Serous labyrinthitis can occur after toxic intake of alcohol or drugs.
- Diffuse suppurative labyrinthitis is caused by acute or chronic otitis media, mastoiditis, or mastoid surgery.

7 Macular Degeneration

- Most commonly, a gradual, age-related loss of central, near, or color vision.

Pathophysiology

- The central fovea of the retina is rich in cones (color vision) and is responsible for clear central vision.
- Risk factors include being an older (75 years) female, white, a smoker; having hyperlipidemia; and consuming little antioxidant-containing foods.
- Dry or atrophic age-related macular degeneration causes pigmental changes in the fovea, which can be visualized on examination. Drusen (pale yellow spots) appear on the macula, showing areas that no longer function. This is the most common type.
- Wet or exudative age-related macular degeneration occurs when vitreous fluid and/or blood leak under the macula. The onset of this type is sudden, and it may be treated with an argon laser, as in retinal detachment.

8 Mastoiditis

- Pain behind the ear, with
- Fever and chills,
- Usually after ear or sinus infection.

Pathophysiology

- Inflammation of the mastoid sinuses, usually as a result of the spread of infection from acute otitis media.
- Occurs rarely because of the availability of antibiotics for otitis media.
- Causative organisms usually are the same as those causing otitis media (e.g., Streptococcus species, Haemophilus influenzae, Staphylococcus aureus), although on some occasions, mycobacteria or fungi may cause the disease.
- Chronic infection of the frontal sinuses may cause this secondary infection.

9 Ménière's Disease

- Ear fullness
- Tinnitus
- Vertigo
- Sweating, nausea, and vomiting may occur.
- Movement of the head makes symptoms worse.

Pathophysiology

- Excessive endolymph in the compartment of the inner ear, possibly from a blockage of endolymph reabsorption.
- Recurring episodes of hearing loss, tinnitus, vertigo, and aural fullness, often resulting in gradually progressive hearing loss.
- Exacerbations may occur suddenly and last for as long as 24 hours.
- When one ear is affected, the other ear will become involved in approximately 50% of cases.
- Injury, infections, endocrine disorders, and vascular disorders may be causative.

10 Otitis Externa

- Pain in the external auditory canal.
- The canal may swell shut.
- Pain is elicited by pressure on the tragus.
- Often called “swimmer’s ear.”

Pathophysiology

- Infection or inflammation of the external auditory canal caused by a contact allergy, an acute bacterial infection, or a fungal infection.
- Diabetics and immunosuppressed clients may experience invasion of the infection into the base of the skull, resulting in deep bone infection.

11 Otitis Media

- Fever and pain in the ear.
- Otoscopic examination reveals a reddened and swollen tympanic membrane.
- Usually associated with colds and allergies.

Pathophysiology

- Otitis media is common in infants and children and results in accumulation of fluid in the middle ear because their short, horizontal eustachian tubes allow exudates from colds and allergens access to the inner ear.
- Causative microorganisms are viruses and bacteria.
- Other risks include respiratory infections, daycare attendance, lower socioeconomic status, exposure to secondhand smoke or wood-burning stoves, allergies, excessive use of a pacifier, and feeding with a propped bottle.

12 Otosclerosis

- Progressive hearing loss, especially with low or soft tones.
- Rinne’s test for bone conduction is normal, but Weber’s test shows lateralization to the most affected ear.
- Tinnitus may be evident.

Pathophysiology

- The cause of this condition is unknown, and is more common in women (worsens in pregnancy).
- The condition may begin in the adolescent years and occurs bilaterally.
- Due to chronic inflammation in the inner ear, bone remodeling by the osteoclasts and osteoblasts occurs, causing excessive spongy bone growth around the stapes and the oval window, resulting in ankylosis and conductive hearing loss.

13 Primary Open-Angle Glaucoma

- Bilateral, usually painless loss of vision.
- May see halos around objects
- Experience mild aching in the eyes or headaches.
- Tonometry measurement ≥20 mm Hg.

Pathophysiology

- This is the most common type of glaucoma, with insidious onset in persons older than 35 years of age. The only risk factors are black race, trauma to the eye, and chronic use of corticosteroids by any route.
- In glaucoma, the anterior chamber experiences outflow problems, with fluid and pressure increases on the choroid layer, the retina, and optic nerve.
- In the darkly pigmented eye, iris pigment may flake off and occlude the iridocorneal angle.

14 Retinal Detachment

- Visual abnormalities of seeing flashing lights or sparks, floaters
- Loss of peripheral vision, & eventually nothing, like a curtain falling over the visual field.

Pathophysiology

- Disorders in vision, like myopia, may predispose the peripheral retina to come away (traction) from the choroid layer.
- Rhegmatogenous detachment can occur during intraocular surgery if traction is applied to the retina, causing vitreous fluid to flow into a hole between the retina and choroid layer, resulting in detachment. Trauma to the head and eyes may cause this type of detachment.
- Exudative or serous detachment occurs in persons with hypertension or intraocular tumors in which serous fluid leaks between the retina and choroid.

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