of endogenous pyrogens.

Malaise

Fatigue

Headache

Anorexia

Treatment of Inflammation: drugs may decrease capillary permeability, reduce number of leukocytes & mast cells.

Types of Healing

Resolution: minimal tissue damage, cells can repair themselves.

Regeneration: damaged tissue is replaced by identical tissue.

Replacement: functional tissue replaced by scar or fibrous tissue.

1st Intention: wound is clean, edges are close together with minimal gap.

2nd Intention: large break in tissue, longer healing process with more scar tissue.

Scar Formation: fibroblasts proliferate, abnormal amount of collagen.

Hypertrophic: overgrowth of fibrous tissue, keloid.

Ulceration: blood supply around scar is impaired resulting in tissue breakdown.

Wound Staging

1. Partial thickness ulcer-red or pink ie. Sunburn.
2. Partial thickness ulcer-blister, scrape, abrasion.
3. Full thickness ulcer- through dermis.
4. Full thickness ulcer that includes muscle or bone.

Drainage

Translucent: clear & watery.

Serosanginous: clear w/ tinge of red/brown. Contains serum/blood thin & watery.

Exudate: creamy yellowish. Contains proteins & WBC's Thick.

Purulent: yellowish. Contains leukocytes and necrotic debris, thick.

Infected Pus: hues of yellow, green or blue. Contains pathogens, thick.

Venous Insufficiency	Clinical Presentation
Incompetent Valves	medial leg area
Inefficient Calf Pump	edema
Distended Capillary Bed	wet wound
Decreased Fibrolysis	scaring, red base
Fibrin Leakage	hemosiderin deposits(purple/brown on leg)
Trauma	Ulcer

Documentation of Pulses

Normal: 2+

Diminished: 1+

Absent: 0

Arterial Insufficiency: decreased arterial blood supply.

IgM:can activate compliment, natural antibodies ie. Involved in blood type

IgA:not in blood, is in tears, saliva & colostrums.

IgE

IgD

Compliment System:antigen-antibody complex, activated during immune rxn w/ IgG or IgM. Causes cell damage when activated, causes macrophages to release enzymes.

Immune Response

Primary: 1st exposure to antigen, 1-2 weeks needed for effective antibodies

Secondary:repeat exposure to same antigen, effective response in 1-3 days

Immunity: Innate-always present. Or acquired.

Hypersensitivity Reactions

Type 1 Hypersensitivity:allergic rxn, exposed to allergen causes development of IgE's,activate mast cells and causes inflammation. Ie. Hay fever, allergies, asthma

Type 2 Hypersensitivity:cytotoxic hypersensitivity. Antigen on cell membrane reacts w/ circulating IgG's, activates compliment, cells w/ antigen destroyed. Ie incompatible blood type exposure.

Type 3: Immune complex hypersensitivity-antigen & antibody combine forming immune complexes that cause inflammation & tissue destruction.

Type 4:Cell Mediated or delayed response by T-lymphocytes. No antibodies present. Ie. Tb test, contact dermatitis.

Immune System Malfunction

Hypersensitivity: full systemic immune response to non-noxious stimulus.

Asthma: central windpipe or airway disorder.

Interventions/Treatment: avoid triggers, medicate. Peak flow 50-80% of person's best signal moderate attack, >50% below best PF = major attack.

Autoimmune

Multiple Sclerosis: autoimmune demyelinization of nerves in brain &CNS

Classification

Clinically Isolated Syndrome-only suffer 1 attack.

MS-multiple attacks at least 1 month apart & damage to at least 2 separate CNS areas & r/o all other possible causes.

Tests & Measures: MRI, visual evoked potentials, CSF analysis.

Interventions: modify disease course, treat attacks, manage sx.

Myasthenia Gravis: antibodies destroy Ach receptors at NM junction.

Guillen Barre Syndrome: demyelination of peripheral nerves.

Fibromyalgia: generalized musculoskeletal pain > 3 months, multiple tender points affecting all 4 quadrants, 11/18 points, 4Kg force painful

Rheumatiod Arthritis:autoimmune destruction of joints affects multiple

ELECTROLYTES

Intracellular electrolytes: potassium, phosphate, magnesium
Blood electrolytes: sodium, calcium, less extent bicarbonate

EXCESS/DEFICIENT ELECTROLYTE	CAUSES of	EFFECTS of
HypoNatremia	Excessive sweating, vomiting, diarrhea, insufficient aldosteerone, kidney failure, excessive water-intake	Impaired nerve conduction, fatigue, mm cramps, Abdom issues, decreased Osmotic pressure in ECF- THUS fluid into cells
HyperNatremia	Insufficient ADH, loss of thirst mechanism, watery diarrhea, rapid respiration,	*Fluid shift out of cells,* weakness, dry tongue mucous membranes, increased BP
HypoKalemia	Diarrhea, diuresis, excessive aldosterone, low dietary intake, Insulin forces K+ into cell	Cardiac Dysrythmias, interference with neuromm junc, decreased dig. Tract motility
HyperKalemia	Renal failure, deficit aldosterone, leakage of K+ from ICF into ECF, prolonged acidosis (H+ replaces)	Cardiac dysrythmias, mm weakness common progressing to paralysis, respiratory arrest
HypoCalcemia	Hypothyroidism, malabsorption syndrome, deficient serum albumin, increased serum pH	Increased permeability/ --excitability of nerve membranes,spont stim of skeletal mm, *Tetany, weak Heart contractions*
HyperCalcemia	Uncontrolled release from bones - demineralization from immobil. -increased intake	Depressed neuromm activity -interference with ADH function -*increased strength cardiac contractions*
HypoMagnesmia	Malabsorption of assoc with alcoholism, use of diuretics	Neuromuscular hyperirritability - heart arrhythmias
HyperMagnesmia	Renal failure	Depressed neuromm funct

GENETIC DISORDERS	**CAUSE(S)**	**CHARACTERISTICS**
Angelman syndrome	X-linked, lose "bit" of chromosome	Flat head. PROTUDING TONGUES odd bouts of Laughter, Balance disorders
Cri du Chat	Missing part chromosome 5, mutation at	Hi "CAT-like" cry, webbed toes & fingers, DOWNward slant to wide set eyes, skin tags ant. ears
Downs syndrome	Trisomy 21	Flat face, Upward slanted eyes, single DEEP crease palm of hand, HYPOtonia (low muscle tone),
Fragile X syndrome	Fragile x retardation protein	Large head w/ **prominent forehead**, boys develop long face, tactile defensiveness
Neurofibromatosis	Autosomal dominant,	Esp. effects nervous syst and skin, birthmarks called **café-au-lait**, **freckles** in armpits and groin, purplish RUBBERY lesions on skin
GENETIC DISORDERS	**CAUSE(S)**	**CHARACTERISTICS**
Prader Willi syndrome	Chromosome XV	Extreme hunger/overeating, obsessed w FOOD, temper tantrums, violent outbursts, **@ 1 Y.O. become ravenously hungry**
		Broad nasal bridge, **PROTRUDING**

-collapse of lung caused by: obstructed airway, compression (tumor), increased surface tension preventing expansion
-small areas asymptomatic, large areas –dyspnea and chest pain

-Pleural effusion – "hydrothorax"

-fluid in pleural cavity à protiens and WBCs follow, respond to inflammation

-Incr pressue in pleural cavity, layers separate, prevent expansion

-presents with incr RR and HR, cyclic chest pain

-Pneumothorax

-air in chest cavity, lung collapse
-open – air enters through hole in chest cavity;
-closed – air in chest cavity from rupture on inside

-tension – air allowed to enter cavity, no natural way to remove it

-Adult Respiratory Distress Syndrome

-rapid, shallow resp, incr HR, confusion

-caused by shock, sepsis, burns, multiorgan failure

Diff Dx

- Sputum

-yellow-green = infection

-rusty-dark = pneumonia

-purulent and foul odor = bronchiectasis

-Breathing

-labored breathing – obstruction

-wheezing, whistling – obstruction of small airways

-stridor – high crowing noise – obstruction of small airways

Patho 4

Digestive Disorders

Digestive System Overview

5 layers of gut wall (inner to outer): mucosa, submucosa, circular muscle layer, longitudinal muscle layer, serosa

*Peristalsis (involuntary contractions) occurs in circular and longitudinal smooth muscle layers

Stomach

Gastrin cells (G cells): initiated by food entering stomach, stimulates chief and parietal cells

Intrinsic factor (parietal cells): needed for absorption of vitamin B12

HCL (parietal cells): activates pepsinogen, creates optimal pH ~2, denatures proteins

Pepsinogen (chief cells): pepsin not activated until pH of 6

Liver "Metabolic factory of the body"

1)Storage of nutrients 2) Maintains blood glucose 3) Blood reservoir 4) Produces bile, plasma proteins, blood clotting factors, cholesterol/lipoproteins 5) Metabolic processes (detoxification, conversions)

Glucose -> Glycogen = Glycogenesis (when glycogen supply low)

Protein, Fat -> Glycogen = Glyconeogenesis

Glycogen -> Glucose = Glycogenolysis (maintains blood glucose levels)

Pancreas

Exocrine (secreting digestive enzymes and electrolytes) and endocrine organ

Trypsin, chymotrypsin, carboxypeptidase-break proteins

Ribonuclease-break nucleic acids

Pancreatic amylase-break starch

Lipase-break lipids

GI tract

Ileum=major site of **nutrient** absorption, villi (folds of mucosa)

Large intestine=**fluid and electrolyte** reabsorption, movement slow to allow absorption of water, vitamin K synthesis (essential for blood clotting)

Neural and hormonal control

PNS (vagus mainly): increased motility and secretions, SNS: inhibits GI activity

Gastrin: increases gastric motility and promotes stomach entering, Secretin: decreases gastric secretions, Cholecystokinin: inhibits gastric emptying

Upper GI Tract Disorders (includes differential diagnosis)

Disorder	Description	Causes
Dysphagia	Difficulty swallowing	1) Neurological deficit

Release epinephrine and aldosterone

Trousseau's sign- positive sign would be tremors and twitching bc of nerve or muscle irritability (Non specific sign)

Chvostek's sign-elicit this by having the patient relax face and then the therapist taps the facial nerve, watch for twitch of mouth or side of face

Addison's Diseases (hypoadrenal)- autoimmune, skin changes color to a slight grey

Cushing's Syndrome (hyperadrenal)- too much glucocorticoids in system, can cause muscle wasting, bone demineralization, and ligaments to be lax, might see buffalo hump on back (does NOT feel like fluid)

Diabetes (fasting plasma glucose >126mg/dL)

Action of insulin-when insulin gets to cell, it makes glucose transporters close to membrane (Below 100 mg/dL is normal for FPG)

Type 1=autoimmune (insulin producing B cell destruction), typically under age 20

Risk factors: sibling has Type 1, parents have type 1

Type 2=obesity (insulin resistance...receptor not binding to insulin as well, pancreas intact), hyperglycemia develops slowly, may have genetic predisposition, 85-90% of all diabetes

Risk factors: overweight, over 45 yrs old, **inactive,** women who had a baby over 9ilbs, low HDL

Gestational=associated with type 2, glucose intolerance w/ pregnancy, increased risk of diabetes later on, if continues >6 weeks after pregnancy...no longer GDM

Acute Hyperglycemia

Early signs: blood sugar >180mg/dL, blurred vision, thirsty, ketones,dry skin, increased urination, tired

Late signs: blood sugar >240mg/dL, nausea/vomiting, deep/rapid breathing, large ketones in urine, fruity breath, some diabetes pts don't sense changes bc of neuropathy

Acute Hypoglycemia

Caused by: over treatment w/ insulin, missed meal, exercising when insulin peaking, stress

Early signs: tachycardia**,** hunger, headache, dizziness, sweating, shaking, pale skin, tingling around mouth,

Late signs: slurred speech, confusion, sudden moodiness, clumsy or jerky movements, seizures, pass out

Treatment: Act quickly! Test blood sugar after attempting to raise it with 15 grams of fast acting sugar...if <70 repeat, if >70 eat meal/snack (always assume they will drop 50 mg/dL while exercising)

Obesity **Nursing/Allied health professions have greater risk of injury due to rising obesity trends**

Underweight = <18.5 Normal weight = 18.5-24.9 Overweight = 25-29.9 Obesity = BMI of 30 or greater

Subcutaneous vs. Intra-abdominal fat

Subcutaneous fat needed for thermal control