Blood Vessels and Hemodynamics

Structure of Vessels

- Arterial walls have three layers
  - Tunica interna - simple squamous epithelial cells (endothelium) & internal elastic lamina
  - Tunica media - smooth muscles cells & elastic fibers
    - vasoconstriction and dilation under sympathetic ANS
    - vascular spasm
  - Tunica externa - elastic & collagen fibers & external elastic lamina in muscular arteries

Arteries

- Elastic (conducting) arteries - large proportion of elastic fibers and thin layer of smooth muscle
  - Serve as pressure reservoirs - reduces pulsatile pressure
  - Aorta, brachiocephalic, common carotid, subclavian, vertebral, pulmonary, & common iliac
- Muscular (distributing) arteries - thick layer of smooth muscle, fewer elastic fibers
  - Controllable diameter and thus controls distribution to tissues

Anastomoses

- Distal end of two or more vessels unite providing alternative pathways (collateral circulation)
- Can occur at any level of circulation

Arterioles

- Transition between three layer structure to two layers (endothelium and sparse smooth muscle)
- Play a major role in regulating blood flow to tissues and altering arterial pressure (vasoconstriction & dilation)

Capillaries

- Site of exchange between blood and tissues because of thin wall and high surface area
  - Single layer of endothelial cells and basement membrane
  - Density & flow of capillaries in tissue proportional to metabolic needs (high for muscles, liver, nerves)
- Precapillary sphincters regulate blood flow through individual capillaries
  - Thoroughfare channels provide bypass to capillaries

Capillary Structure

- Tight junction type - seal between endothelial cells causing all exchange through cells (brain)
- Continuous type - continuous with intercellular clefts
- Fenestrated type - “pores” in plasma membrane (70-100nm) allowing
movement of large molecules (kidney, intestines, endocrine glands)

- Sinusoid type - large intercellular clefts along with some specialized cells (liver, spleen, bone marrow)

**Venules**
- Transition between two layer structure and three layers

**Veins**
- Three layer structure
  - Thinner tunica interna & media and thicker tunica externa (lack elastic laminae)
  - Larger lumen diameter
- Venous valves to prevent backflow constructed of endothelial cells
  - Work in conjunction with skeletal muscle contraction and respiratory “pump”
  - When valves malfunction, expansion of venous wall - varicose veins
- Vascular sinus - veins with thin walls and no smooth muscle (coronary sinus)
- At rest, 60% of blood in veins (blood reservoirs in skin and digestive tract)
  - Compared with systemic capillaries holding 5% of blood
  - Can be reduced by venous vasoconstriction when needed elsewhere

**Movement Across Capillaries**
- Intercellular clefts
- Pinocytosis
- Across plasma membrane of endothelial cells
- Fenestrations

**Capillary Exchange**
- Diffusion - depends on concentration gradient, size and solubility
  - Water soluble molecules (glucose, amino acids & some hormones) through intercellular clefts & fenestrations
    - Not possible in blood-brain barrier
  - Lipid soluble molecules (triglycerides, some hormones, gases) through endothelial cells
- Vesicular transport
  - Large lipid insoluble molecules through transcytosis
- Bulk flow (filtration & reabsorption)
  - Movement of water and solutes (except large proteins) due to hydrostatic and osmotic pressure
    - Net filtration pressure (NFP)
    - Blood hydrostatic (35 mm Hg/16 mm Hg) vs. interstitial fluid hydrostatic pressure (0 mm Hg)
    - Blood colloid osmotic pressure (26 mm Hg) vs. interstitial fluid osmotic pressure (1 mm Hg)
    - NFP=(BHP+IFOP)-(BCOP+IFHP) arterial end=+10 mm Hg venous end=-9 mm Hg
– 85% of filtrate reabsorbed by capillaries, remaining by lymphatic capillaries

**Even More Capillary Exchange**
– Abnormal filtration or absorption leads to edema
  • Increased blood hydrostatic pressure - due to higher venous pressure
  • Decreased concentration of plasma proteins - lower blood colloidal pressure
  • Increased permeability of capillaries - more plasma protein pass through
  • Blockage of lymphatic vessels - postoperative or filarial worm parasite

**Hemodynamics**
• Velocity of flow - inversely related to cross-sectional area of vessels
  – Aorta 3-5 cm² 40cm/sec
  – Capillaries 4500-6000 cm² 0.1 cm/sec
  – Vena cava 14 cm² 5-20 cm/sec
  – Approximately 1 min to flow through pulmonary and systemic circuit
• Volume of flow
  – \( F = P / R \) (blood pressure, vascular resistance)
  • Mean arterial BP = DBP + (SBP-DBP)/3
  • Systemic VR related to blood viscosity, vessel length and cross-section for all systemic vessels
  – Effects of dehydration and polycythemia, obesity, vasoconstriction/dilation

**Neural Control of BP and Flow**
• Cardiovascular center has major control effect
  – Compensation of brain blood flow when standing up (hydrostatic hypotension)
  – Redistribution during exercise (more to muscles)
  – Already discussed: control over HR and contractility with input from baroreceptors, chemoreceptors and proprioceptors
  – CV center also controls vasoconstriction-dilation through continuous stimulation of S-ANS via vasomotor nerves
  • Norepinephrine causes constriction in skin and visceral supply vessels (alpha adrenergic receptors)
  • Most veins constrict to move blood out of “reservoirs”

**Hormonal Control of BP & Flow**
• Effect on heart, vessel diameter, and blood volume
• Renin-angiotensin-aldosterone system (RAA)
  – Renin is an enzyme produced by kidney when BP or flow is low
  – Renin acts on precursor which ultimately becomes angiotensin II (a strong vasoconstrictor)
  – Angiotensin II stimulates aldosterone production by adrenal cortex (causes kidney to retain Na⁺; and water by osmosis)
• Epinephrine & norepinephrine from adrenal medulla increases HR, contractility and vasoconstriction in skin and abdomen, epinephrine causes vasodilation in
heart and skeletal muscle (beta adrenergic receptors)

- Antidiuretic hormone (ADH) from posterior pituitary increases water retention by kidneys and vasoconstriction during severe blood loss
- Atrial natriuretic peptide (ANP) from R atrium decreases electrolyte and water retention by kidneys and inhibit vasoconstriction

**Autoregulation**

- Localized regulation of blood flow within a region
  - e.g. flow to brain is constant, but localized flow depends on activity (O\textsubscript{2} and glucose demands)
- Physical factors
  - Temperature (heat dilates)
  - Myogenic effect in arterioles - stretching due to higher BP causes vasoconstriction (and vice versa)
- Chemical (vasoactive) mediators -
  - Released by cells depending on metabolic needs including WBCs, platelets, smooth muscle, endothelial cells and macrophages
  - Affect smooth muscle of arterioles and capillary sphincters

**Fainting (Syncope)**

- Caused by sudden temporary loss of consciousness due to cerebral ischemia
- Vasopressor syncope - sudden emotional stress
- Situational syncope - pressure stress due to coughing, urination or defecation
- Drug-induced syncope
- Orthostatic syncope - due to sudden standing
- Carotid sinus syncope - due to sinus stretch

**Shock**

- Inadequate CO supplying O\textsubscript{2} and nutrients to body cells
- Signs and symptoms - clammy, cool, pale skin; tachycardia; weak, rapid pulse; sweating; hypotension; decreased urinary output; thirst; and acidosis
- Hypovolemic shock due to reduced blood volume from hemorrhage or excessive fluid loss
- Also cardiogenic, vascular (due to vasodilation), and obstructive shock
- Stage I: compensated (non-progressive) shock
  - Negative feedback systems will correct including sympathetic ANS, RAA system, ADH system, and vasodilator mediators
- Stage II: decompensated (progressive) shock
  - Positive feedback system that activated with blood volume @ 75-85%, BP @ 40-50 mm Hg
  - Effects include: depressed cardiac activity, less vasoconstriction, increased capillary permeability, intravascular clotting, cellular destruction, acidosis
- Stage III: irreversible shock
Checking Circulation
• On your own
• Figure 21.18
Hepatic Portal Circulation
• Carries blood between two capillary networks from gastrointestinal tract to liver
• Enables direct nutrient utilization and blood detoxification
Fetal Circulation
• On your own
Disorders
• On your own
• Hypertension
• Aneurysm
• Coronary artery disease (CAD)
• Deep-venous thrombosis