Chapter 19: The Cardiovascular System: Blood Vessels

Most common route

heart → elastic arteries → muscular arteries → arterioles → metarterioles → capillaries → venules → veins

Alternative routes

• Portal system: blood flows through two consecutive capillary networks before returning to heart
  – hypothalamus - anterior pituitary
  – found in kidneys
  – between intestines – liver
• Anastomosis = point where 2 blood vessels merge
  • Arteriovenous shunt
    – artery flows directly into vein
    – fingers, toes, ears; ↓ heat loss, allows blood to bypass exposed areas during cold
  • Venous anastomosis
    – most common, blockage less serious
    – alternate drainage of organs
    – may be seen in skin of hands, arms, legs
• Arterial anastomosis
  – collateral circulation (coronary & cerebral circulation)

Resistance:

• Resistance is opposition to flow
• Produced by 3 factors:
  1) blood viscosity
  2) length of vessel
  3) diameter of vessel – resistance varies by 4th power of radius (= r^4) regulated by:
     a. vasoconstriction
     b. vasodilation
• The friction developed in the systemic circulation is called the “peripheral resistance (PR)” or “total peripheral resistance (TPR)”
• Important in Force-Flow relationship: \( \text{Flow} = \frac{\Delta P}{TPR} \)

Systemic Blood Pressure

• Measured at brachial artery of arm
• Systolic pressure: BP during ventricular contraction
• Diastolic pressure: BP during ventricular relaxation
• Normal value, young adult: 120/75 mm Hg
• Pulse pressure = (systolic – diastolic) = 120 – 75 = 45 mm Hg
Mean Arterial Pressure (MAP):
- measurements taken at intervals of cardiac cycle, best estimate: diastolic pressure + (1/3 of pulse pressure)
- varies with gravity: standing; 62 - head, 180 - ankle

Blood Pressure Changes With Distance

Abnormalities of Blood Pressure
- Hypertension = BP > 140/90 can weaken small arteries and cause aneurysms
- Hypotension = chronic low resting BP. causes: blood loss, dehydration, anemia

Arterial Elasticity
- Importance: expansion and recoil maintains steady flow of blood throughout cardiac cycle, smoothes out pressure fluctuations and ↓ stress on small arteries
- BP rises with age: arteries less distensible
- BP determined by cardiac output, blood volume and peripheral resistance

Vascular Contribution to Resistance:
- Arterioles
  - arterioles control amount of blood to various organs
  - short vessels connect arterioles to capillaries
  - muscle cells form a precapillary sphincter about entrance to capillary
- Precapillary sphincters in metarterioles control which beds are well perfused
  - only 1/4 of the capillaries are open at a given time

Regulation of BP and Flow
- Neural control
- Hormonal control
- Local control

Neural Control of BP and Flow: Baroreflex
- Changes in BP detected by stretch receptors (baroreceptors), in large arteries above heart
  - aortic arch
  - aortic sinuses (behind aortic valve cusps)
  - carotid sinus (base of each internal carotid artery)
- Autonomic negative feedback response
  - baroreceptors send constant signals to brainstem
BP causes rate of signals to rise, **inhibits** vasomotor center, ↓ sympathetic tone, vasodilation causes BP ↓

BP causes rate of signals to drop, **excites** vasomotor center, ↑ sympathetic tone, vasoconstriction and BP ↑

**Hormonal Control of BP and Flow: Angiotensin II**

- **Angiotensinogen** (prohormone produced by liver)
  - ↓ Renin (kidney enzyme – released by low BP & epinephrine)

- **Angiotensin I**
  - ↓ ACE (angiotensin-converting enzyme in lungs)
  - ACE inhibitors block this enzyme lowering BP

- **Angiotensin II** = very potent vasoconstrictor

**Hormonal Control of BP and Flow: other hormones**

- **Aldosterone**
  - promotes Na+ and water retention by the kidneys
  - increases blood volume and pressure

- **Atrial natriuretic factor** (↑ urinary sodium excretion)
  - generalized vasodilation

- **ADH** (water retention)
  - pathologically high concentrations, vasoconstriction

- **Epinephrine and norepinephrine effects**
  - most blood vessels: binds to α-adrenergic receptors, **vasoconstriction**
  - skeletal and cardiac muscle blood vessels: binds to β-adrenergic receptors, **vasodilation**

**Local Control of BP and Flow**

- **Metabolic theory of autoregulation**
  - tissue inadequately perfused, wastes accumulate = vasodilation

- **Vasoactive chemicals**
  - substances that stimulate vasomotion; histamine, bradykinin

- **Reactive hyperemia**
  - blood supply cut off then restored

- **Angiogenesis - growth of new vessels**
  - regrowth of uterine lining, around obstructions, exercise, malignant tumors
  - controlled by growth factors and inhibitors

**Capillary Exchange - Diffusion**

- **Most important mechanism**

- **Lipid soluble substances**
  - steroid hormones, O₂ and CO₂ *diffuse easily*

- **Insoluble substances**
  - glucose and electrolytes must pass through channels, fenestrations or intercellular clefts

- **Large particles - proteins, held back**
Capillary Exchange - Filtration and Reabsorption
• Opposing forces
  – blood (hydrostatic) pressure drives fluid out of capillary
    • high on arterial end of capillary, low on venous end
  – colloid osmotic pressure (COP) draws fluid into capillary (same on both ends)
    • results from plasma proteins (albumin) - more in blood
    • oncotic pressure = net COP (blood COP - tissue COP)
• Capillary filtration at arterial end
• Capillary reabsorption at venous end
• Variations
  – location (glomeruli - devoted to filtration
  – activity or trauma releases histamine & ↑ filtration

Causes of Edema
• ↑ Capillary filtration (↑ capillary BP or ↑ capillary permeability)
  – poor venous return
    • congestive heart failure - pulmonary edema
    • insufficient muscular activity
  – kidney failure (water retention, hypertension)
  – histamine causes vasodilation and makes capillaries more permeable
• ↓ Capillary reabsorption
  – hypoproteinemia (oncotic pressure ∝ blood albumin) cirrhosis, famine, burns, kidney disease
• Obstructed lymphatic drainage

Mechanisms of Venous Return
• Pressure gradient
  – 7-13 mm Hg venous pressure towards heart
    • venules (12-18 mm Hg) to central venous pressure (~5 mm Hg)
• Gravity drains blood from head and neck
• Thoracic pump
  – inhalation - thoracic cavity expands (pressure ↓) abdominal pressure ↑, forcing blood upward
  – central venous pressure fluctuates
    • 2mmHg - inhalation, 6mmHg - exhalation
    • blood flows faster with inhalation
• Cardiac suction of expanding atrial space
• Skeletal muscle pump in the limbs (exercise & posture important)
  – venous pooling occurs with inactivity
  – venous pressure not enough force blood upward
  – with prolonged standing, CO may be low enough to cause dizziness or syncope
    • prevented by tensing leg muscles, activate skeletal m. pump
  – jet pilots wear pressure suits

Circulatory Shock
• Any state where cardiac output insufficient to meet metabolic needs, 2 types:
- cardiogenic shock - inadequate pumping of heart (MI)
- low venous return (LVR) shock - 3 principle forms

LVR shock

• Hypovolemic shock - most common
  - loss of blood volume: trauma, bleeding, burns, dehydration
• Obstructed venous return shock - tumor or aneurysm
• Venous pooling (vascular) shock
  - Caused by long periods of standing, sitting or widespread vasodilation
    • Neurogenic shock - loss of vasomotor tone, vasodilation from emotional shock to brainstem injury
    • Septic shock - bacterial toxins trigger vasodilation and ↑ capillary permeability
    • Anaphylactic shock - severe immune reaction to antigen, histamine release, generalized vasodilation, ↑ capillary permeability

Responses to Circulatory Shock

• Compensated shock
  - homeostatic mechanisms may bring about recovery
  - ↓ BP triggers baroreflex and production of angiotensin II, both stimulate vasoconstriction
  - if person faints and falls to horizontal position, gravity restores blood flow to brain; quicker if feet are raised
• Decompensated shock: above mechanisms fail and life-threatening positive feedback loops occur
  - ↓ CO → myocardial ischemia and infarction → ↓ CO
  - slow circulation → disseminated intravascular coagulation → slow circulation
  - ischemia and acidosis of brainstem → ↓ vasomotor tone, vasodilation → ↓ CO → ischemia and acidosis of brainstem