Cardiovascular Regulation

- Regulation of hemodynamics occurs via local autoregulation, neural control and hormones.
Autoregulation of Blood Flow

Local regulation of blood flow occurs by vasoconstriction and vasodilation.

Both occur at the site of arteriolar branches, usually into capillary beds (pre-capillary sphincters).

Smooth muscle contraction closes lumen of the blood vessel.

Response is a direct effect of conditions within local environment.

- Local vasodilation can be caused by:
  - Uptake of $O_2$ (local decrease) by local cells and release of $CO_2$ (local increase).
  - Fall in pH.
  - Injury / inflammation.

- Local vasoconstriction can be caused by:
  - Decrease in temperature.
  - Release of various chemicals in response to trauma (shock).
  - Response to certain localized activities (digestion – vs – exercise, etc).
Neural Control of Blood Flow

- Cardiac centers and the vasomotor centers within medulla oblongata are responsible for monitoring and regulating cardiovascular activities.
  - Carioacceleratory center (sympathetic excitation to increase cardiac output).
  - Cardioinhibitory center (parasympathetic inhibition of cardiac output).
  - Vasomotor centers cause vasodilation (inhibition) or vasoconstriction (excitation).
- Cardiovascular centers detect changes in pH, blood pressure, and dissolved gas concentrations.
- Two types of “transducers” sense these parameters:
  - Baroreceptors (pressure sensors)
  - Chemoreceptors (chemical sensors).
The Baroreceptor Reflexes

- Autonomic reflexes that adjust cardiac output and peripheral resistance to regulate and maintain normal blood pressures.

- Baroreceptors are situated at:
  - Aortic sinuses (immediately distal to aortic valve).
  - Carotid sinuses (within carotid arteries of neck).
  - Right atrial walls.
Chemoreceptor Reflexes

- Found in aortic arch and carotid sinuses.
- Additional chemoreceptors in medulla oblongata monitor CSF composition.
- Activation occurs via a rise in CO$_2$ or drop in pH.
- Produces activation of cardioacceleratory and vasomotor centers.
- Increases cardiac output and vasoconstriction.
Hormones and Cardiovascular Function

- **Endocrine system provides acute and chronic regulation.**
- **Short term:**
  - Epinephrine released immediately upon activation of adrenal medulla.
- **Long term:**
  - **Angiotensin II:**
    - Produced via enzymatic reaction initially catalyzed by renin produced by kidneys in response to lower pressure.
    - Causes powerful vasoconstriction and elevation of blood pressure.
    - Activates other hormones that act to retain water.
  - **Antidiuretic Hormone**
    - Also responds to increase in plasma solute concentration.
    - Water retention at kidneys; Vasoconstriction.
  - **Erythropoietin:**
    - Released by kidneys in response to decreased $\text{O}_2$ or blood pressure.
    - Increases RBC production.
  - **Atrial Natriuretic Peptide**
    - Released by cells in RA in response to **increased** blood pressure.
    - Responds to increased venous filling by promoting loss of Na+ and water at kidneys, inhibiting sympathetic activation of adrenal medulla, and vasodilation.
Cardiovascular Response to Exercise

- Cardiac output at rest ranges from 4.5 – 6 L/min.
- Exercise produces substantial increase.
- Effects of exercise:
  - Vasodilation at skeletal muscle vasculature.
  - Increase in venous return.
  - Increase in cardiac output as a function of the Frank-Starling law and as a reflex response to atrial stretching.
  - Arterial pressure are therefore maintained despite decrease in downstream resistance.
  - Advanced stage of exercise produces sympathetic stimulation.
  - Produces vasoconstriction for non-essential vasculature.
  - Blood supply to brain remains unaffected.
  - Chronic exercise produces mild to moderate hypertrophy of cardiac muscle.
  - Significant chronic exertion coupled with genetic predisposition can cause hypertrophic cardiomyopathy.
Response to Hemorrhage

Hemorrhage \(\rightarrow\) significant loss of blood volume.

Short term responses:

- Baroreceptor reflexes stimulate cardiac output and initiate peripheral vasoconstriction.
- Mobilization of the venous reserve.
- Sympathetic stimulation of heart rate.

Longer term responses:

- Decline in capillary blood pressure reverses pressure gradient and moves interstitial fluid into capillaries.
- Hormones (ADH, aldosterone) released to promote fluid retention at kidneys.
- Erythropoietin released to stimulate RBC formation.
- Thirst reflexes triggered.

Significant blood loss (> 35%) leads to circulatory shock.