Frank-Starling Law of the Heart

• Relationship between EDV, contraction strength, and SV.
• Intrinsic mechanism:
  – Varying degree of stretching of myocardium by EDV.
  – As EDV increases:
    • Myocardium is increasingly stretched.
    • Contracts more forcefully.
  – As the ventricles fill, the myocardium stretches; so that the actin filaments overlap with the myosin at the edges of the A band.

Frank-Starling Law of the Heart

• Allows more force to develop.
• Explains how the heart can adjust to rise in TPR.

Extrinsic Control of Contractility

• Contractility:
  – Strength of contraction at any given fiber length.
• Depends upon sympathoadrenal system:
  – NE and Epi produce an increase in contractile strength.
  • + inotropic effect:
    – More Ca²⁺ available to sarcomeres.
Negative Feedback Control of Blood Pressure by Baroreceptor Reflex

Negative Feedback Control of Blood Volume and Osmolality
Regulation of Blood Volume by the Kidney

- Formation of urine begins by filtration of plasma through glomerular capillary pores.
- Volume of urine excreted can be varied by changes in reabsorption of filtrate.
  - Adjusted according to needs of body by action of hormones.

Regulation by ADH

- Released by posterior pituitary when osmoreceptors detect an increase in plasma osmolality.
- Dehydration or excess salt intake:
  - Produces sensation of thirst.
  - Stimulates H₂O reabsorption from urine.

Regulation by Aldosterone

- Steroid hormone secreted by adrenal cortex.
- Mechanism to maintain blood volume and pressure through absorption and retention of Na⁺ and Cl⁻:
  - Stimulates reabsorption of NaCl.
  - Indirectly increases H₂O reabsorption.
  - Does not dilute osmolality.
- Release stimulated:
  - During salt deprivation.
  - Reduced blood volume and pressure.
Renin-Angiotension-Aldosterone System

- When blood pressure and flow are reduced in renal artery, juxtaglomerular apparatus secretes renin.
- Renin converts angiotensinogen to angiotensin I.
- Angiotensin I is converted to angiotensin II by ACE.
- Angiotensin II:
  - Powerful vasoconstrictor.
  - Stimulates production of aldosterone.
  - Stimulates thirst.

Atrial Natriuretic Peptide (ANP)

- Produced by the atria of the heart.
- Stretch of atria stimulates production of ANP.
  - Antagonistic to aldosterone and angiotensin II.
  - Promotes Na⁺ and H₂O excretion in the urine by the kidney.
  - Promotes vasodilation.
Hypertension (HTN)

- Blood pressure in excess of normal range for age and gender.
  - > 140/90 mm Hg.
- Primary or essential hypertension:
  - Is the result of a complex or poorly understood process.
- Secondary hypertension:
  - Is a result of a known disease process.

Dangers of Hypertension

- Silent killer:
  - Patients are asymptomatic until substantial vascular damage occurs.
    - Atherosclerosis.
- Increases afterload.
  - Increases workload of the heart.
    - Congestive heart failure.
- Damage cerebral blood vessels.
  - Cerebral vascular accident (stroke).

Treatment of Hypertension

- Modification of lifestyle:
  - Cessation of smoking.
  - Moderation in alcohol intake.
  - Weight reduction.
  - Programmed exercise.
  - Reduction in Na⁺ intake.
  - Diet high in K⁺.
Treatment of Hypertension (continued)

• Medications:
  – Diuretics:
    • Increase urine volume.
  – Beta-blockers:
    • Decrease HR.
  – Calcium antagonists:
    • Block Ca²⁺ channels.
  – ACE inhibitors:
    • Inhibit conversion to angiotensin II.
  – Angiotension II-receptor antagonists:
    • Block receptors.

Circulatory Shock

• Hypovolemic shock:
  – Circulatory shock that is due to low blood volume.
  – Decreased CO and blood pressure.
  • Bleeding, dehydration, and burns.
• Compensations:
  – Baroreceptor reflex:
    • Tachycardia.
    • Vasconstriction to GI, skin, kidneys, and muscles.
  – Kidneys stimulate production of renin-angiotensin-aldosterone system.
    • Vasconstriction.
    • Increase in ADH.

Other Causes of Circulatory Shock

• Anaphylactic shock:
  – Severe allergic reaction.
  – Widespread release of histamine.
  – Vasodilation.
• Neurogenic shock:
  – Rapid fall in BP.
    • Sympathetic tone is decreased.
• Cardiogenic shock:
  – Cardiac failure.
    • CO inadequate to maintain perfusion.
Congestive Heart Failure

- CO is insufficient to maintain the blood flow required by the body.
  - Increased venous volume and pressure.
- Caused by:
  - MI (most common cause).
  - Congenital defects.
  - Hypertension.
  - Aortic valve stenosis.
  - Disturbances in electrolyte concentrations.
    - $K^+$ and $Ca^{++}$.
- Compensations similar to those of hypovolemic shock.
- Treated with medications:
  - Digitalis, vasodilators, and diuretics.