Learning Objectives

Review the principles of the cardiovascular system as they relate to the effects of anesthetic drugs.

Describe the cardiovascular anatomy, physiology, and pathophysiology.

Describe the impact of selection of pharmacologic agents on patient comorbidity.

Continuous Professional Recertification

Crosswalk to Examination Preparation

Domain II: Applied Clinical Pharmacology (24%)

- II.A. Pharmacokinetics/pharmacodynamics/Pharmacogenetics of anesthetics and adjunct medications
  - II.A.1. Factors influencing distribution volume
  - II.A.2. Pharmacodynamics
  - II.A.3. Laboratory & diagnostic studies
  - II.A.4. Incidence, duration, benefits, adverse effects, observed responses, alternatives, and
  - II.A.5. S.A.E
  - II.A.6. Interpretation monitoring techniques
  - II.A.7. Adverse Pharmacological Reactions

Domain III: Human Physiology and Pathophysiology (24%)

- III.A. Cardiovascular
  - III.A.1. Normal anatomical structures and function
  - III.A.2. Physiologic processes and anesthetic considerations
  - III.A.3. Pathophysiologic disease processes and associated disorders

Brent Dunworth, CRNA, MSN, MBA
Physiology Concepts

Pressure-Volume Curves

Physiologic Changes

Pathophysiologic Changes

Pharmacology

Determinants of Ventricular Performance

- Systolic function
  - Ventricular ejection
- Diastolic function
  - Ventricular filling
- Equated with CO
  - \( CO = HR \times SV \)

Ventricular Performance

- Heart Rate
  - CO generally proportional
- Stroke Volume
  - Preload
  - Afterload
  - Contractility
Preload

- End diastolic volume
- Dependent on ventricular filling
- Starling Law
  - Relationship between CO and LVEDV

Determinants of Ventricular Filling

- Venous return
- Blood volume
- Distribution of blood volume
  - Posture
  - Intrathoracic pressure
  - Pericardial pressure
  - Venous tone
- Rhythm
- Heart Rate

Starling Law
Ventricular Compliance

- Early diastolic compliance reduction
  - Hypertrophy
  - Ischemia
  - Asynchrony

- Late diastolic compliance reduction
  - Hypertrophy
  - Fibrosis

- Extrinsic factors
  - Pericardial disease
  - Overdistention of contralateral ventricle
  - Pleural pressure
  - Tumors
  - Surgical compression
Ventricular Compliance

- Less compliant
- Normal
- More compliant

Ventricular and diastolic pressure

Ventricular and diastolic volume

Afterload

- Ventricular wall tension during systole

- Law of LaPlace
  - Larger radius → greater wall tension required to develop the same pressure

Law of LaPlace

Cylindrical Vessel

\[ T = PR \]

Spherical Vessel

\[ T = \frac{P}{R} \]
Law of Laplace

- About half as much tension
- Much less wall tension
- Very little wall tension
- Maximum wall tension

Same pressure in all regions according to Pascal's principle.

Afterload

- Arterial impedance to ejection
- SVR
  - Arteriolar tone

Cardiac output vs. Afterload

- Normal
- Decompensation
- Decompensation
- Normal
Afterload

Contractility

- Intrinsic ability to pump in the absence of changes in preload or afterload
- Dependent on intracellular calcium concentration during systole

Valvular Dysfunction

- Stenosis
  - AV valve (tricuspid, mitral)
  - Reduced stroke volume primarily by decreasing ventricular preload
  - Semilunar valve (pulmonary, aortic)
  - Reduced stroke volume by increased afterload
Valvular Dysfunction

- Insufficiency (regurgitation)
  - Reduced stroke volume due to regurgitant volume with every contraction
  - EDV can flow backward into atrium (or ventricle) during systole
  - Effective (forward) stroke volume decreased

Physiology Concepts

- Pressure-Volume Curves
- Physiologic Changes
- Pathophysiologic Changes
- Pharmacology

Cardiac Cycle
Diastole: Filling

Atrial Contraction

Systole: Isovolumetric Contraction
**Systole: Ejection**

- During systole, the heart contracts, propelling blood out of the ventricles.

**Diastole: Isovolumetric Relaxation**

- Diastole begins with the heart relaxing after ejection.
- Blood pressure decreases in the ventricles during this phase.

**Diastole: Ventricular Filling**

- Ventricular filling occurs as blood flows back into the ventricles through the atrioventricular valves.
- The diastolic compliance curve illustrates the relationship between pressure and volume.
### Key Points

**A**
- Mitral valve opens
- Ventricular filling

**B**
- EDV
- Ventricular contraction begins
- Systole

**C**
- AV opens
- Ejection

**D**
- AV closes
- ESV
- Isovolumetric relaxation
- Diastole

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### Physiology Concepts

**Pressure-Volume Curves**

**Physiologic Changes**

**Pathophysiologic Changes**

**Pharmacology**

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### Changes in Preload

- Physiologic Change

- Preload
  - End diastolic stress on ventricle
  - End diastolic fiber length
  - End diastolic volume
Preload Concepts

- When preload increases, EDV (filling) increases
- Preload decreases, EDV decreases
- Ventricle empties to the same point (same ESV); stroke volume changes

Increased Preload

Preload Reduction
Afterload Concepts

- When afterload increases, the ventricle empties less completely
- SV (EDV-ESV) decreases
- BP increases (shown as increased LV pressure as ejection begins)

Afterload Concepts

- When afterload decreases, the ventricle empties more completely.
- SV (EDV-ESV) increases
- Decreased BP (decreased LV pressure at ejection)

Changes in Afterload

- Physiologic
  - Increased
    - Increased pressure and Volume
  - Decreased
    - Smaller pressures and volumes
Contractility Concepts

- **Increase** (digitalis, calcium)
  - Ventricle empties more completely
  - ESV and EDV decrease ("shrinks")
  - SV increases
  - BP increases

- **Decrease** (heart failure)
  - Ventricle empties less completely
  - ESV and EDV increase ("dilates")
  - SV, BP decrease

Contractility Changes

Preload and afterload are constant

- Increase: higher pressure and smaller volumes
- Decrease: lower pressure; higher volumes

Physiology Concepts

<table>
<thead>
<tr>
<th>Pressure-Volume Curves</th>
</tr>
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<tbody>
<tr>
<td>Physiologic Changes</td>
</tr>
<tr>
<td>Pathophysiologic Changes</td>
</tr>
<tr>
<td>Pharmacology</td>
</tr>
</tbody>
</table>
Valvular Pathology

- Aortic Valve
  - Insufficiency
    - Acute
    - Chronic
  - Stenosis
- Mitral Valve
  - Insufficiency
  - Stenosis
- Hypertrophic Cardiomyopathy

Aortic Insufficiency

Etiologies

- Abnormalities of the Leaflets
  - Rheumatic, Bicuspid, Degenerative
  - Endocarditis
- Dilation of the Aortic Annulus
  - Aortic Aneurysm / Dissection
  - Inflammatory
    - Syphils
    - Giant Cell Arteritis
    - Collagen Vascular Disease
    - Amyloid Sporadictis
  - Inherited
    - Marfans
    - Osteogenis Imperfectis
Aortic Insufficiency

Pathophysiology of Aortic Regurgitation

Acute Chronic

Blood regurgitated into LV during diastole

Acute vs. Chronic AI

Eccentric Hypertrophy
Management of AI

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Goal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preload</td>
<td>Maintain preload</td>
</tr>
<tr>
<td>Afterload</td>
<td>Decrease; vasodilator beneficial</td>
</tr>
<tr>
<td>Rate</td>
<td>Prevent slow rate (&lt;80 bpm)</td>
</tr>
<tr>
<td>Contractility</td>
<td>Decreased in chronic</td>
</tr>
<tr>
<td>Oxygen balance</td>
<td>Increased demand as a result of increase in LV mass</td>
</tr>
<tr>
<td>Rhythm</td>
<td>Sinus</td>
</tr>
</tbody>
</table>

Fast, full, forward...

Aortic Stenosis

- Etiology & Incidence
  - 38% Congenital bicuspid
  - 33% Degenerative calcification
  - 24% Postinflammatory fibrocalcific (Rheumatic Heart Disease)
  - 2% Unicommissural aortic valve
  - 1% Hypoplastic
Aortic Stenosis

Pathophysiology

1. Angina
   - Elevated LVEDP decreases perfusion pressure
   - Myocardial hypertrophy increases demand
   - Median survival: 5 years

2. Syncope
   - Inability to increase CO and meet reduced SVR demands
   - Median survival: 3 years

3. Heart Failure
   - Elevated LVEDP
   - Elevated LA pressure
   - Pulmonary venous congestion
   - Median survival: 2 years
AVA: Rate of Flow vs. Systolic Pressure

Aortic Stenosis

About same LV volume, much greater pressure

Management of AS

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Goal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preload</td>
<td>Increase to distend the stiff ventricle</td>
</tr>
<tr>
<td>Afterload</td>
<td>Decrease is hazardous</td>
</tr>
<tr>
<td>Rate</td>
<td>Normal to Slow (increase in rate can produce ischemia)</td>
</tr>
<tr>
<td>Contractility</td>
<td>Decreased to normal</td>
</tr>
<tr>
<td>Oxygen balance</td>
<td>Increased demand as a result of increase in LV mass</td>
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</table>
Mitral Regurgitation

- Etiologies
  - Alterations of the Leaflets, Commissures, Annulus
  - Rheumatic
  - MVP
  - Endocarditis
  - Alterations of LV or LA size and Function
    - Papillary Muscle (Ischemic, MI, Myocarditis, DCM)
    - HOCM
    - LV Enlargement - Cardiomyopathies
    - LA Enlargement - from MR
      - MR begets MR

Decreased ventricular volume during isovolumetric contraction
Mitral Regurgitation

- Increased LV emptying; retrograde flow into LA

Management of MR

<table>
<thead>
<tr>
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<tr>
<td>Preload</td>
<td>Normal to increased</td>
</tr>
<tr>
<td>Afterload</td>
<td>Decreased, vasodilator beneficial</td>
</tr>
<tr>
<td>Rate</td>
<td>Prevent slow rate (&lt;80 bpm)</td>
</tr>
<tr>
<td>Contractility</td>
<td>Decreased from rheumatic or coronary artery disease</td>
</tr>
<tr>
<td>Oxygen balance</td>
<td>Increased demand as a result of increased mass</td>
</tr>
<tr>
<td>Rhythm</td>
<td>Sinus</td>
</tr>
<tr>
<td></td>
<td>• Atrial fibrillation tolerated if rate is controlled</td>
</tr>
</tbody>
</table>

Mitral Stenosis

- Normal MV area ≈ 4.4 cm² |
- Symptoms begin at ≈ 2 cm² |
- Critical MS ≈ 1 cm²
Mitral Stenosis

- **Etiology**
  - 99% postinflammatory (Rheumatic)
  - 1% congenital

- **Pathophysiology**
  - LA hypertension
  - Pulmonary interstitial edema
  - Pulmonary hypertension
  - LA stretch and atrial fibrillation
  - Limited LV filling and cardiac output

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Mitral Stenosis

- **Symptoms**
  - Dyspnea
  - Pulmonary venous congestion
  - Fatigue
  - Diminished cardiac output
  - Inability to tolerate volume
  - Inability to tolerate increased HR
  - Decreased filling
  - Increased LA pressure / PV congestion
  - Hemoptysis

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Mitral Stenosis

- **Less LV filling**
Management of MS

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Goal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preload</td>
<td>Normal to increased</td>
</tr>
<tr>
<td>Afterload</td>
<td>Maintain; increase is poorly tolerated</td>
</tr>
<tr>
<td>Rate</td>
<td>About 80 bpm; tachycardia and bradycardia decrease CO</td>
</tr>
<tr>
<td>Contractility</td>
<td>Normal</td>
</tr>
<tr>
<td>Oxygen balance</td>
<td>Normal</td>
</tr>
<tr>
<td>Rhythm</td>
<td>Sinus</td>
</tr>
<tr>
<td></td>
<td>• Atrial fibrillation tolerated if ventricular rate &lt;80 bpm</td>
</tr>
</tbody>
</table>

Hypertrophic Obstructive Cardiomyopathy (HOCM)

Hypertrophic Cardiomyopathy

- **Etiology**
  - No identifiable cause
  - Evidence of myocardial fiber disarray

- **Treatment**
  - Medical management
  - Surgical myomectomy
### Hypertrophic Cardiomyopathy

**Diagram:**
- Chamber gets small
- Small volume
- High Pressure

### Management of Hypertrophic Cardiomyopathy

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Goal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preload</td>
<td>Normal to high</td>
</tr>
<tr>
<td>Afterload</td>
<td>High</td>
</tr>
<tr>
<td>Rate</td>
<td>Rate &lt;80 bpm</td>
</tr>
<tr>
<td>Contractility</td>
<td>Decreased</td>
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<td>Increased demand as a result of increased LV mass</td>
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<td>Sinus rhythm</td>
</tr>
</tbody>
</table>

### Physiology Concepts

- Pressure-Volume Curves
- Physiologic Changes
- Pathophysiologic Changes
- Pharmacology
Causes of Hypotension

- **Hypovolemia**
  - Hemorrhage
  - Dehydration
- **Blood volume redistribution**
  - Postural changes
- **Reduced cardiac output**
  - Acute heart failure
  - Chronic heart failure
- **Reduced systemic vascular resistance**
  - Loss of sympathetic tone
  - Vasodilation (septic shock, anaphylaxis)

Treatment of Hypotension

**GOAL: RAISE ARTERIAL BLOOD PRESSURE**

- **Increase cardiac output**
  - Increase blood volume
  - Stimulate contractility
- **Increase systemic vascular resistance**
  - Vasoconstrictor drugs

Contractility

**Sympathomimetic**

- Stimulate heart (beta receptors)
- Vascular smooth muscle contraction (alpha receptors)

- **Side effects**
  - Increase in myocardial oxygen demand
  - Cardiac arrhythmia
  - Angina, ischemia
### Contractility

<table>
<thead>
<tr>
<th>Drug</th>
<th>Receptor Selectivity</th>
<th>Clinical Use</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epinephrine</td>
<td>$\beta_1 = \beta_2 &gt; \alpha_1$*</td>
<td>Anaphylactic shock; cardiogenic shock; cardiac arrest.</td>
<td>Increased pressor cardiac stimulation and vasodilation, which turn to vasoconstriction at high doses.</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>$\beta_1 = \beta_2 &gt; \alpha_1$</td>
<td>Severe hypotension; septic shock.</td>
<td>Reflex bradycardia masks direct stimulatory effects on atrioventricular node.</td>
</tr>
<tr>
<td>Dopamine</td>
<td>$\beta_1 &gt; \beta_2 &gt; \alpha_1$*</td>
<td>Acute heart failure; cardiogenic shock and acute renal failure.</td>
<td>Biosynthetic precursor of norepinephrine; stimulates norepinephrine release.</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>$\beta_1 &gt; \beta_2 &gt; \alpha_1$</td>
<td>Acute heart failure; cardiogenic shock; refractory heart failure.</td>
<td>Net effect is cardiac stimulation with modest vasodilation.</td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>$\beta_1 = \beta_2$</td>
<td>Bradycardia and electromechanical block.</td>
<td>Net effect is cardiac stimulation and vasodilation with little change in pressure.</td>
</tr>
</tbody>
</table>

### Alpha agonists

- **Phenylephrine**
  - **Side effects**
    - Reflex bradycardia
    - Increase afterload (increased myocardial workload)
    - Angina, ischemia

- **Vasopressin**
  - **Potent vasoconstrictor ($V_1$ receptor)**
    - **Side effects**
      - Bronchoconstriction
      - Water intoxication, hyponatremia
      - Increases myocardial oxygen demand (increased afterload)
      - Coronary artery constrictor
Phosphodiesterase inhibitors

- Phosphodiesterase breaks down cAMP
- cAMP is an important second messenger in cardiac muscle contraction
- Breakdown prevention increases cardiac inotropy, chronotropy, and dromotropy (conduction velocity).

**Systemic circulation**
- Vasodilation
- Increased organ perfusion
- Decreased SVR
- Decreased arterial pressure

**Cardiopulmonary**
- Increased contractility and rate
- Increased stroke volume and EF
- Decreased ventricular preload
- Decreased PCWP

Clinical Decision Making

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