Adult CCRN/CCRN-E/CCRN-K Certification Review Course: Cardiovascular, Part 1

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Disclosures

- The speaker has received honoraria from Edwards Lifesciences
Course Objectives

After the completion of this program, participants will be able to:
1. Identify the critical care needs that patients with single and multisystem organ abnormalities have in common
2. Outline assessment strategies and treatment options for common critical care needs
3. Review the pathophysiology of single and multisystem dysfunction, as well as the pharmacological management of each
4. Describe the AACN Synergy Model for Patient Care and the blueprint for the CCRN certification exam

Blueprint/Test Plan

CCRN Examination Blueprint/Test Plan
- Exam consists of 150 multiple choice questions
- 125 are scored
- 25 are used for item analysis for future questions
Clinical Judgment = 80%
- Cardiovascular 18%
- Pulmonary 17%
- Multisystem 14%
- Neurology, Musculoskeletal Behavioral/Psychosocial 13%
- Endocrine, Hematology Gastrointestinal, Renal/Genitourinary and Integumentary 18%

Professional Caring & Ethical Practice = 20%
- Advocacy/Moral Agency
- Caring Practices
- Collaboration
- Systems Thinking
- Response to Diversity
- Clinical Inquiry
- Facilitator of Learning

Testable Nursing Actions
- Normal/abnormal physical assessment
- Apply leads for ECG monitoring
- Monitor hemodynamic status
- Manage patients receiving CV medications
- Recognize indications for and manage patients with intraaortic balloon pump (IABP) or following percutaneous coronary intervention
- Recognize indications for and manage patients requiring
  - 12-lead ECG
  - Arterial line
  - Monitoring: pulmonary artery catheter (PAC)/central venous pressure (CVP)/mixed venous oxygen saturation
  - Defibrillation
  - Pacing: transcutaneous/transvenous
- Recognize signs and symptoms of cardiovascular emergencies, indicate interventions, and seek assistance as needed
Road Map: Cardiovascular System

- Review of hemodynamic parameters
- Manipulating hemodynamics, cardiovascular drugs
- Shock
- Heart failure
- Myocardial infarction
- Aortic aneurysms

Direct and Derived Hemodynamic Parameters

Cardiac Output

- Volume of blood ejected by the heart in 1 minute
- Formula: heart rate x stroke volume
- Normal: 4-8 L/min
Cardiac Index

- Formula: CO / BSA
- Normal: 2.5–4.3 L/m²/min
- Significance

Heart Rate x Stroke Volume

- Heart rate
  - Bradyarrhythmias
  - Sinus bradycardia, junctional rhythms, AV blocks
  - Consider if atrial kick is intact
  - Do not rely on blood pressure to determine stability

Effect of Heart Rate on Cardiac Cycle

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Systole</th>
<th>Diastole</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR 60/min</td>
<td>30%</td>
<td>70%</td>
</tr>
<tr>
<td>HR 90/min</td>
<td>45%</td>
<td>55%</td>
</tr>
<tr>
<td>HR 120/min</td>
<td>60%</td>
<td>40%</td>
</tr>
</tbody>
</table>
Stroke Volume

- Volume of blood ejected with each heartbeat
  - Normal: 60–100 mL/beat
  - Stroke volume index (SVI): 35–60 mL/m²
- Determinants
  - Preload
  - Afterload
  - Contractility

Preload

- Volume of blood in the ventricle at the end of diastole
- In practice: pressure generated by the volume of blood in the ventricle at the end of diastole
- How to assess?
  - RV: CVP RA pressure/RVEDVI
  - LV: PAOP/PAD/LA pressure/dynamic parameters: (SVV/PPV/SPV)

RVEDVI, right ventricular end-diastolic volume index; PAOP, pulmonary artery occlusion pressure
Preload

- CVP
  - Normal: 0–5 mmHg
  - Optimal: Varies—10 mmHg
- PAOP
  - Normal: 6–12 mmHg
  - Optimal: 14–18 mmHg in the critically ill patient
- RV EDP: Optimize using Frank–Starling curve
- Dynamic parameters: SVV/PPV/SPV: 10%–15%

Frank–Starling Principle

Preload

- Reducing preload
  - Diuretics
  - Vasodilators: Think venous bed
- Increasing preload
  - Volume
  - Vasoconstriction of venous bed
**Cardiovascular**

**Afterload**
- Definition
- Clinical implications

**Pressure**

- the ventricle must generate to open the semilunar valve and eject its contents
- Assessment of afterload
  - Left ventricle: Systemic vascular resistance (SVR)
  - Right ventricle: Pulmonary vascular resistance (PVR)

**Afterload**
- The higher the afterload, the greater the work
- Increases myocardial oxygen demands
- May decrease contractility
- RV more sensitive than the LV
- Some afterload is necessary to contribute to contractility
Cardiovascular Effects of Changes in Afterload

- Stroke volume: Reflects overall resistance or impedance to systolic ejection
  - Greatest resistance to flow in small arteries and arterioles
  - Formula: \( 80 \times (\text{MAP} - \text{RAP}) / \text{CO} \)
  - Normal: 800–1200 dynes/sec/cm\(^5\)
  - SVRI: 1970–3900 dynes/sec/cm\(^5\)/m\(^2\)

Systemic Vascular Resistance

- Increased
  - Volume infusions
  - Peripheral vasoconstriction
  - Low CO states
  - Hypothermia
  - ↑ blood viscosity
  - Hypovolemia
  - Vasopressors
  - LV failure
  - Alpha-adrenergic agents

- Decreased
  - Diuretics
  - Vasodilators
  - Hyperdynamic phase of sepsis
  - Peripheral vasodilation
  - Loss of vasomotor tone

MAP, mean arterial pressure; RAP, right arterial pressure
Reduction of Afterload

- Use of vasodilators
  - Nitroglycerin
  - Nicardipine
  - Hydralazine (Apresoline)
  - Isosorbide dinitrate (Isordil)
  - Calcium channel blockers
  - Sodium nitroprusside
  - ACE inhibitors/angiotensin II (AII) receptor blockers

Increasing Afterload

- Use of pressors (ie, vasoconstrictors)
  - High-dose epinephrine
  - Phenylephrine (Neo-Synephrine)
  - Norepinephrine (Levophed)
  - High-dose dopamine
  - Vasopressin

Pulmonary Vascular Resistance

- Resistance or impedance to RV ejection
- Formula: $80 \times (MPAP – PAOP)/CO$
- Normal: $<250$ dynes/sec/cm$^5$
- $PVRI$: $255 – 285$ dynes/sec$^5$/m$^2$

MPAP: mean pulmonary artery; PAOP: pulmonary artery occlusion pressure; PVRI: pulmonary vascular resistance index
Pulmonary Vascular Resistance
- Increased
  - Hypoxia
  - Pulmonary edema
  - Acute respiratory distress syndrome
  - Pulmonary emboli
  - Congenital heart defects
  - Positive end-expiratory pressure
  - Pulmonary hypertension
  - Sepsis
  - Valvular heart disease
- Management
  - Vasodilator therapy
  - Correction of hypoxia
  - Prostaglandins
  - Prostacyclin

Contractility
- Ability of the heart to modulate its contractile performance independent of preload and afterload
- Assessment
  - Stroke work/stroke work index
  - Stroke volume index

Ventricular Contractility
- Stroke work/index
  - LVSWI: SVI (MAP – PAOP) x 0.0136
    - Normal: 50 – 62 gm·m²/m²/beat
  - RVSWI: SVI (MPAP – RAP) x 0.0136
    - Normal: 5 – 10 gm·m²/m²/beat
**Pulmonary Artery Pressure**

- Normal range: 15–25/6–12 mmHg
- Clinical significance
  - High readings
    - Pulmonary hypertension
      - Primary
      - Secondary
  - Low readings
    - Hypovolemia

**Pulmonary Artery Occlusion Pressure**

- Normal range: 6–12 mmHg
- Clinical significance
  - High readings
    - LV failure
    - Valvular heart disease
    - Cardiac tamponade
  - Low readings
    - Hypovolemia?

**Mean Arterial Pressure**

- Average pressure in circuit during systole and diastole
- Formula: \[ \frac{SBP + (2 \times DBP)}{3} \]
  - Normal: 70–105 mmHg
- Used to determine perfusion pressure of vital organs
  - <60 mmHg—perfusion deficits begin to occur
  - <40 mmHg—"cardiovascular collapse"
Mean Arterial Pressure
- Increased
  - Volume infusions
  - Peripheral vasoconstriction
  - Increased contractility
  - Hypervolemia
  - Vasopressors
- Decreased
  - Diuretics
  - Peripheral vasodilation
  - Inotropic therapy
  - Hypovolemia
  - Vasodilators

Mean Pulmonary Artery Pressure
- Average pressure in the pulmonary circuit during systole and diastole
- Formula: \[ \frac{SPAP + (2 \times DPAP)}{3} \]
- Normal: 10–20 mmHg
Question 1

Determinants of stroke volume are:

A. Heart rate, cardiac output, systolic BP
B. Preload, afterload, contractility
C. Cardiac index, diastolic BP, heart rate
D. Mean arterial pressure, cardiac output, heart rate

Question 1—Rationale

Determinants of stroke volume are:

B. Preload, afterload, contractility
   - Heart rate, cardiac output, systolic BP—Incorrect; HR and SBP can affect SV but do not determine SV. Stroke volume is a determinant of cardiac output.
   - Cardiac index, diastolic BP, heart rate—Incorrect; SVI determines CI. Diastolic BP and heart rate may affect SV
   - Mean arterial pressure cardiac output, heart rate—Incorrect; stroke volume is a determinant of cardiac output. Heart rate and MAP may affect stroke volume but are not a major determinant
Question 2

Afterload is defined as:
A. Decreased resistance  
B. Vasodilation  
C. Increased resistance  
D. Mean arterial pressure

Question 2—Rationale

Afterload is defined as:
C. Increased resistance—due to vasoconstriction of the vascular bed
   - Decreased resistance—incorrect; this is an increase in resistance
   - Vasodilation—incorrect; vasoconstriction is necessary for an increase in afterload
   - Mean arterial pressure—incorrect; does not give as much information about afterload

Question 3

Two important events occur during diastole. They are:
A. Ventricular filling and coronary perfusion  
B. Ventricular contraction and coronary perfusion  
C. Shortening of protein filaments and coronary perfusion  
D. Ventricular depolarization and coronary perfusion
Question 3—Rationale

Two important events occur during diastole. They are:

A. Ventricular filling and coronary perfusion
   - Ventricular contraction and coronary perfusion—Incorrect; during systole, the ventricles are emptying and very little coronary blood flow occurs
   - Shortening of protein filaments and coronary perfusion—Incorrect; shortening of the protein filaments is myocardial contraction (systole: ventricle ejecting blood)
   - Ventricular depolarization and coronary perfusion—Incorrect; ventricular depolarization immediately precedes ventricular systole (or ejection)

Manipulating Hemodynamics

- Cardiovascular drugs

Inotropes

- Receptor-dependent vs phosphodiesterase inhibitors
Types of Receptors

- Beta receptors
- Alpha receptors
- Dopaminergic receptors

**Beta Receptors**

- $\beta_1$ receptors are found primarily in heart stimulation produces
  - $\uparrow$ Heart rate
  - $\uparrow$ Contractility
  - $\uparrow$ Conduction velocity through AV node
- $\beta_2$ receptors are found in the lungs and peripheral arterioles. Stimulation produces relaxation of smooth muscle
  - Vasodilation
  - Bronchodilatation
### Alpha Receptors

- $\alpha_1$ receptors are found primarily in the lungs and peripheral arterioles. Stimulation produces constriction of smooth muscle (i.e., vasoconstriction).
- $\alpha_2$ receptors are found primarily in the brain.

### Dopaminergic Receptors

- Found in the renal, mesenteric vascular beds.
- Stimulation produces vasodilation.

### Tissue Receptor Response Table

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Receptor</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart</td>
<td>Beta</td>
<td>Rate</td>
</tr>
<tr>
<td>SA node</td>
<td>Beta</td>
<td>Conduction velocity</td>
</tr>
<tr>
<td>Atria</td>
<td>Beta</td>
<td>Contractility</td>
</tr>
<tr>
<td>AV node</td>
<td>Beta</td>
<td>Conduction velocity</td>
</tr>
<tr>
<td>Ventricles</td>
<td>Beta</td>
<td>Refractoriness</td>
</tr>
<tr>
<td>Blood vessels</td>
<td>Beta</td>
<td>Rate</td>
</tr>
<tr>
<td>Skeletal muscle</td>
<td>Beta</td>
<td>Conduction velocity</td>
</tr>
<tr>
<td>Skin, mucosa, GI tract and kidney</td>
<td>Alpha</td>
<td>Vasodilation</td>
</tr>
<tr>
<td>Renal</td>
<td>Dopa, Alpha</td>
<td>Vasodilation</td>
</tr>
<tr>
<td>Mesentery</td>
<td>Dopa, Alpha</td>
<td>Vasodilation</td>
</tr>
<tr>
<td>Bronchial smooth muscle</td>
<td>Beta</td>
<td>Relaxation</td>
</tr>
</tbody>
</table>
Specific Agents

- Inotropes
- Vasodilators
- Beta blockers
- Calcium channel blockers

Dopamine

- Immediate precursor of norepinephrine
- Neurotransmitter in central and peripheral nervous system
- Decreases aldosterone secretion in adrenal cortex
- Inhibits thyroid-stimulating hormone and prolactin release
- Inhibits insulin secretion

Dopamine

- Titrate to achieve desired effects
  - 2–10 mcg/kg/min = ↑ contractility (β effects)
  - >10 mcg/kg/min = vasoconstriction (α effects)
Dopamine

- Indications
  - Shock states
  - Cardiogenic
  - Septic
  - Post cardiac surgery

- Side effects
  - Nausea, vomiting
  - Tachyarrhythmias
  - Supraventricular and ventricular
  - Profound vasoconstriction

NEVER infuse via a peripheral line!

Dobutamine

- Synthetic catecholamine directly stimulates
  - $\beta_1$ receptors
  - $\beta_2$ receptors
  - $\alpha$ receptors
  - ↑ contractility and heart rate with slight vasodilation
  - Can produce down-regulation of beta receptors

Dobutamine

- Titrate to achieve desired effects
- Dosage range: 2.5–20 mcg/kg/min
- $T_{1/2} = 2.5–3$ minutes
- Do NOT administer in alkaline solutions
**Dobutamine**

- **Indications**
  - CHF
  - Shock states
  - Cardiogenic
  - Septic
  - Stress testing

- **Side effects**
  - Increased heart rate
  - Dysrhythmias: ventricular and supraventricular

**Epinephrine**

- Cardiac effects mediated through β receptors
  - 0.005–0.02 mcg/kg/min = ↑ HR; + inotropic effects; vasodilation (↓ SVR)
- Vascular effects mediated through α receptors at high doses:
  - ↑ SVR; ↑ BP; renal artery vasoconstriction
  - β₂ stimulation → bronchodilation

**Epinephrine**

- Beta effects = 0.005–0.02 mcg/kg/min
- Alpha effects = 1 mg IV push
- T₁/₂ = 2 minutes
Epinephrine

- **Indications**
  - Low output states
  - Cardiac arrest
  - Shock states
  - Asthma
  - Anaphylaxis

- **Side effects**
  - Restlessness, fear
  - Tachyarrhythmias
  - Severe hypertension → cardiovascular accident and angina
  - Hypokalemia
  - Hypophosphatemia

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Norepinephrine

- **Endogenous catecholamine—dose-dependent effects**
  - Low doses = beta stimulation
  - High doses = alpha stimulation

Norepinephrine

- **Dosage**
  - Start at 0.05–0.10 mcg/kg/min and titrate up
  - Or 2–4 mcg/min and titrate up
  - T1/2 = 2.0–2.5 min
  - Infiltration—Use phentolamine mesylate (Regitine) to block intense vasoconstriction

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Norepinephrine

- **Titrate via a central line to achieve desired effects; must weigh cost/benefit ratio**

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12/2015
Norepinephrine

- **Indications**
  - Hypotension
  - Cardiogenic shock (MI)
  - GI bleeding (lavage)

  **Never** infuse through a peripheral line!

- **Contraindicated in renal and mesenteric thrombosis**

- **Side effects**
  - Tachyarrhythmias
  - Headaches
  - Tremors
  - Restlessness
  - Severe hypertension

Phenylephrine (Neo-Synephrine)

- **Pure α stimulator**
- Effects are primarily vascular
  - ↑ SBP, ↑ DBP, ↑ PAP
  - Coronary and renal vasoconstriction
  - Indirectly releases norepinephrine from storage sites
  - At large doses, could stimulate β1 receptors, causing an increased heart rate
  - Can cause reflex bradycardia—mediated through vagus nerve

Phenylephrine

- **Initial dose:** 100–180 mcg/min to achieve desired effect
- **Maintenance dose:** 40–60 mcg titrated to maintain BP
- **Pressor effects**
  - Immediate
  - Lasts 15–20 minutes

Never infuse through a peripheral line!
Phenylephrine
- Side effects
  - Vasoconstriction
  - Hypertension
  - Bradycardia

*Never infuse through a peripheral line!*

Vasopressin
- Antidiuretic hormone
- Larger doses
  - α stimulator causing vasoconstriction
  - Does not have negative effect on myocardium
- Dosages
  - ACLS: 40 units—do not repeat
  - Infusion: 0.04 units per min

Milrinone
- Phosphodiesterase inhibitor
- Positive inotrope
- Minimal vasodilating effects
### Milrinone

**Administration**
- Loading dose: 50 mcg/kg undiluted over 10 minutes
- Infusion: Start at 0.5 mcg/kg/min and increase in increments of 0.375 mcg/kg
- Max infusion: 0.75 mcg/kg/min

**Indications**
- Low CO states
- Acute CHF
- Cardiomyopathy

**Side effects**
- Arrhythmogenic
- Headaches, tremors
- Thrombocytopenia
- Hypokalemia
- Hypotension
- Angina pectoris

### Nitroglycerin

**Direct vasodilator**
- Systemic and pulmonary venodilation → ↓ RV and LV afterload
- ↓ LV and RV filling pressures
- ↓ LV pressure-volume relationship
- ↓ aortic impedance
- Coronary artery dilation
- Improvement of ischemic zone
- May raise threshold for ventricular fibrillation

Cardiovascular, Part 1
Nitroglycerin

**Dosage/administration**
- Continuous infusion to achieve desired effects
- Start infusion rate at 10 mcg/min and increase in 10 mcg/min increments
- No upper limit of infusion except...?

**Indications**
- Chest pain r/t myocardial ischemia
- ↓ preload
- ↓ afterload

**Side effects**
- Hypotension
- Headache!
- Nitrate tolerance

Nitrate Tolerance

- Using nitrates → chronic vasodilation → activation of the renin angiotensin system → production of super oxides → inactivation of endogenous and exogenous nitric oxide
- Therefore, 12-hour, window-free time is important!
Sodium Nitroprusside

- Direct vasodilator
  - Balanced effects on arterial and venous beds
- In 10% of patients, can ↑ pulmonary shunt
  - Will see SpO₂ and PO₂ fall
- Can produce coronary steal syndrome

Usual dosages are 0.25–10 mcg/kg/min
Duration of action 1–5 minutes
Long-term monitoring (>48 hrs) should include serum thiocyanate levels
  - >10 mg/dL—significant (lab costs $100)
  - Infusion rates <3 mcg/kg/min are not associated with toxicity
  - Poor renal function increases risk for thiocyanate toxicity

Indications
- Severe heart failure with ↑ SVR
- Mitral regurgitation to ↓ afterload
- Low cardiac output syndrome with ↑ SVR
- Hypertensive crises

Contraindications
- Use with caution in patients with
  - Hypothyroidism
  - Hepatic/renal disease
  - Concomitant vasodilators

Use with caution in patients with

- Hypothyroidism
- Hepatic/renal disease
- Concomitant vasodilators
Sodium Nitroprusside Adverse Effects

- CNS
  - Nervousness
  - Ataxia
  - Headaches
- Cardiac
  - Hypotension
  - Palpitations
- Cyanide poisoning
  - Impaired tissue oxygenation
  - Confusion
  - Hyperreflexia
  - Convulsions
  - Rx: cyanide poisoning kit

Nesiritide (Natrecor)

- Brain natriuretic peptide
  - Identical to endogenous BNP
- Effects
  - Vasodilation
  - Natriuresis
  - Diuresis

Nesiritide

- Usually start with a bolus dose followed by infusion
- Do not infuse through same line as other meds
- Side effect: hypotension
Nesiritide
- Incompatible with many medications
  - Insulin
  - Furosemide (Lasix), bumetanide (Bumex)
  - Heparin
- Do not infuse through heparin-coated catheters

Nicardipine (Cardene)
- Calcium channel blocker → vasodilation
- Indications
  - Hypertensive urgency/crisis
  - Afterload reduction
- Administration via infusion
  - 5–15 mg/hr—titrate for desired effect

Nicardipine
- T$_{1/2}$: 15–45 min; therefore, avoid rebound hypertension
- Side effects
  - Hypotension
  - Phlebitis
### Beta Blockers

- **General indications**
  - AMI
  - Prevention of sudden death
  - Attenuate ventricular remodeling?
  - Tachycardias
  - Supraventricular
  - Ventricular
  - Hypertension

- **Side effects**
  - AV blocks
  - Sinus bradycardia
  - Use with caution
  - Raynaud’s disease
  - COPD
  - Insulin-dependent diabetes mellitus

### Calcium Channel Blockers

- **Indications**
  - Hypertension
  - Supraventricular arrhythmias
  - MI
    - STEMI when beta blocker–intolerant
    - Non-STEMI possible

- **Side effects**
  - Some are stronger vasodilators, others are stronger AV blockers
  - Hypertension
### Receptor Dependent Inotropes

<table>
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<th>Indications/Actions</th>
<th>Dosages</th>
<th>Side/Adverse Effects</th>
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| **Dopamine**    | • Shock states: cardiogenic, septic; post-cardiac surgery  
• Immediate precursor of norepinephrine  
• Neurotransmitter in the central and peripheral nervous system  
• Decreases aldosterone secretion in the adrenal cortex  
• Inhibits TSH and prolactin release  
• Inhibits insulin secretion | Titrate the IV infusion to achieve desired effects  
• 2–10 mcg/Kg/min = ↑ contractility (beta stimulation)  
• >10 mcg/Kg/min = vasoconstriction (alpha stimulation) | • Nausea, emesis  
• Tachyarrhythmias (ventricular & supraventricular)  
• Profound vasoconstriction |
| **Dobutamine**  | • Synthetic catecholamine; directly stimulates the β1 receptors, β2 receptors, α receptors  
• Directly increases myocardial contractility and heart rate  
while modestly lowering peripheral vascular resistance  
• Will lose its effect during prolonged infusions due to down regulation of β receptors  
• Indications: congestive heart failure; shock states  
• Cardiogenic, septic | Titrate the infusion to achieve desired effects  
• Usual dosage range: 2.5–20 mcg/Kg/min.  
• Half-life: 2.5–3 minutes  
• Do not administer in alkaline solutions | • Dysrhythmias |
| **Epinephrine** | • Cardiac effects are mediated through β receptors;  
• 0.005–0.02 mcg/Kg/min = ↑ heart rate; ↓ inotropic effect, vasodilation → ↓ SVR  
• Vascular effects mediated through α receptors @ high doses; ↑ SVR, ↑ BP, renal artery vasoconstriction  
• β2 stimulation → bronchodilation  
• Indications: low output states, cardiac arrest, shock states, asthma, anaphylaxis | • 0.005–0.02 mcg/Kg/min = beta effects  
• Alpha effects: 1 mg IV push/via ET tube  
• Half-life = 2 minutes | • Restlessness, fear  
• Tachyarrhythmias  
• Severe hypertension → CVA, angina  
• Hypokalemia  
• Hypophosphatemia |
| **Norepinephrine** | • Naturally occurring catecholamine with effects that are dose dependent  
– low doses: β stimulation  
– higher doses: α stimulation  
• Indications: hypotensive states; cardiogenic shock (MI); GI bleeding | Titrate infusion via central line to achieve desired effect. Weigh cost/benefit ratio  
• Dosage/administration  
• Infusion rates 2–4mcg/min are suggested  
• Start at 0.05–0.1 mcg/kg/min and titrate up  
• Half-life = 2.0–2.5 min  
• If infiltration occurs, the drug will cause sloughing of tissue; use phentolamine (Regitine) to block the intense vasoconstriction | • Contraindicated in mesenteric and renal thrombosis  
• Side effects  
– Tachyarrhythmias  
– Headaches  
– Tremors  
– Restlessness  
– Severe ↑ BP |
| **Phenylephrine** (Neosynephrine) | • Pure α stimulator; effects are primarily vascular, causing vasoconstriction resulting in ↑ SBP and ↑ DBP, ↑ PAP. Coronary and renal arteries constrict. If vasoconstriction is severe, blood flow to the vital organs could decrease | Initial dose: 100–180 mcg/min to achieve desired effect  
• Maintenance infusion: 40–60 mcg/min titrated to maintain BP | Vasoconstriction  
Hypertension  
Bradycardia |
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• Vascular effects mediated through α receptors @ high doses: ↑ SVR, ↑ BP, renal artery vasoconstriction  
• β₂ stimulation → bronchodilatation  
• Indications: Low output states; cardiac arrest, shock states, asthma, anaphylaxis | • Pressor effects are immediate and will last 15–20 min  
• 0.005–0.02 mcg/Kg/min = beta effects  
• Alpha effects: 1 mg IV push/via ET tube  
• Half-life = 2 minutes | • Restlessness, fear  
• Tachyarrhythmias  
• Severe hypertension → CVA, angina  
• Hypokalemia  
• Hypophosphatemia |
| **Vasopressin (Pitressin)** | • Antidiuretic hormone  
• Larger doses: α stimulator causing vasoconstriction. Note: does not have negative effects on myocardium such as those caused by epinephrine | • Initial dose (ACLS): 40 units  
• Infusion: 0.04 units/min | Vasoconstriction Hypertension |
| **Milrinone (Primacor)** | • Positive inotrope with less peripheral vasodilating effects than amrinone  
• Indications: low cardiac output states; acute CHF; cardiomyopathy | • Loading dose: 50 mcg/Kg—slowly over 10 minutes (undiluted)  
• Infusion: 50/250 cc start @ .5 mcg/Kg/min. Increase in increments of .37 mcg/Kg/min, max of .75 mcg/Kg/min | • Arrhythmogenic: SVT, VT  
• Headaches, tremors  
• Thrombocytopenia |
| **Vasodilators** | • Systemic and pulmonary venodilation  
• Decreased left and right ventricular filling pressures  
• Decreased left ventricular pressure volume relationship  
• Decreased aortic impedance  
• Decreased right and left ventricular afterload  
• Dilation of coronary arteries  
• Improvement of ischemic zone | • Indications  
– Chest pain related to myocardial ischemia  
– Preload reduction  
– Afterload reduction  
• Dosage/administration: continuous infusion titrated to achieve desired effects. It is suggested that the infusion rate be started at 10 mcg/min and ↑ in 10 mcg/min increments until the desired effect is achieved | • Hypotension  
• Nitrate tolerance |
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| Sodium nitroprusside (Nipride) | • Direct vasodilator with balanced effect on the arteriolar and venous systems.  
• In 10% of patients can increase pulmonary shunt  
will see SpO₂ and PO₂ fall  
• Can produce coronary steal syndrome  
• Indications:  
  – Severe heart failure with ↑ SVR  
  – Mitral regurgitation to ↓ afterload and improve  
  – Forward flow out of the ventricle  
  – Low CO syndrome with ↑ SVR  
  – Hypertensive crises | • Usual dosages are 0.25–10 mcg/Kg/min  
• Duration of action: 1–5 minutes  
• Long-term administration of the drug should be monitored with serum thiocyanate levels.  
• Infusion rates of less than 3 mcg/Kg/min are not associated with toxicity  
• Serum thiocyanate levels >10 mg/dL are considered to be toxic. (Lab costs: $100)  
• Poor renal function increases the risk for thiocyanate toxicity antidote: sodium thiosulfate | • CNS effects = nervousness, twitching, ataxia, headaches  
• Cardiac effects = hypotension, palpitations  
• Cyanide poisoning = impaired tissue oxygenation, confusion, hyper-reflexia, convulsions  
• Contraindications: use with caution in patients with hypothyroidism, hepatic or renal disease as well as those patients receiving other antihypertensive drugs |
| Nesiritide (Natrecor) | • Brain natriuretic peptide – identical to endogenous BNP  
• Effects  
  – Vasodilation  
  – Natriuresis | • Usual dosage:  
  – Bolus: 2 mcg/Kg over 60 seconds  
  – Infusion: 0.01 mcg/min  
• Do not infuse though the same line with other medications | • Side effect:  
Hypotension—monitor BP closely  
• Incompatible with:  
  – Enalaprilat  
  – Insulin  
  – Lasix  
  – Heparin  
  – Hydralazine  
  – Bumex |
| Nicardipine (Cardene) | • Calcium channel blocker  
• Indication: Hypertension | • Usual dosage: Infusion: 0.1 mg/mL concentration. Titrate for effect | • Side effect:  
Hypotension |
ACE Inhibitors and Angiotensin II Receptor Blockers

Renin Angiotensin System

- Renin release
- Angiotensinogen
- Angiotensin I
- Converting enzyme
- Angiotensin II
- Vasoconstriction
- Na⁺ and H₂O retention
- Adrenal cortex
- Aldosterone secretion
- Angiotensin II production via other pathways?

Renin Angiotensin System

- Endocrine
- Tissue
  - Exists in many systems, including cardiac cells
  - Responsible for ventricular remodeling process occurring with MI or CHF
  - Angiotensin II (AII) production via other pathways?
Cardiovascular, Part 1

Ventricular Remodeling
- Following AMI, necrotic tissue is replaced with scar tissue. The myocardial wall becomes much thinner.
- AII is thought to produce hypertrophy of the normal cells adjacent to the scar tissue.
- Over time, the infarcted area begins to bulge outwardly during systole.
- Additionally, the myocytes begin to slide apart, leading to development of a dilated cardiomyopathy.

ACE Inhibitors
- Block conversion (converting enzyme) of angiotensin I to angiotensin II.
- End with "pril" (example: captopril).
- Indications:
  - Hypertension
  - MI
  - CHF
- Major side effects:
  - Cough
  - Angioedema
  - Common in females
  - More common in black females
  - Renal insufficiency

Angiotensin Receptor Blockers
- Directly block the AII receptors on the cell membrane.
- Cough is not a side effect.
- Effects are equal to ACE inhibitors.
Question 4

Which of the following are pressor agents?

A. Dobutamine, milrinone, and dopamine
B. Norepinephrine, phenylephrine, and sodium nitroprusside
C. Vasopressin, dobutamine, and dopamine
D. Norepinephrine, phenylephrine, and vasopressin

Question 4—Rationale

Which of the following are pressor agents?

D. Norepinephrine, phenylephrine, and vasopressin
- Dobutamine, milrinone, and dopamine—Incorrect; dobutamine and milrinone often produce vasodilation. Dopamine can be used as a pressor agent
- Norepinephrine, phenylephrine, and sodium nitroprusside—Incorrect; sodium nitroprusside is a vasodilator
- Vasopressin, dobutamine, and dopamine—Incorrect; dobutamine has slight vasodilation effects
Question 5

What is the major effect of inotropes?
A. Inhibit phosphodiesterase
B. Facilitate transport of calcium into the cell
C. Impair myocardial contractility
D. Prolong ventricular diastole

Question 5—Rationale

What is the major effect of inotropes?

B. Facilitate transport of calcium into the cell—cAMP production enhances calcium influx into the cell
- Inhibit phosphodiesterase—Incorrect; only milrinone inhibits phosphodiesterase
- Impair myocardial contractility—Incorrect; calcium influx increases myocardial contractility
- Prolong ventricular diastole—Incorrect; inotropes have no effect on the length of diastole

Question 6

Which of the following should always be infused through a central line?
A. Dopamine
B. Nitroglycerin
C. Dobutamine
D. Milrinone
Question 6—Rationale

Which of the following should always be infused through a central line?

A. Dopamine; dopamine causes stimulation of alpha receptors with marked localized vasoconstriction if infused through a peripheral line and infiltration occurs
   • Nitroglycerin—Incorrect; nitroglycerin causes vasodilation
   • Dobutamine—Incorrect; dobutamine’s beta 2 effects override the alpha 1 effects of the drug, causing vasodilation
   • Milrinone—Incorrect; milrinone will cause some vasodilation

Question 7

A major concern associated with sodium nitroprusside infusion lasting >72 hours is:

A. Marked peripheral vasoconstriction
B. Development of thiocyanate toxicity
C. Depressed myocardial contractility
D. Development of sinus bradycardia

Question 7—Rationale

A major concern associated with sodium nitroprusside infusion lasting >72 hours is:

B. Development of thiocyanate toxicity; sodium nitroprusside infusions >48 hours should be monitored by thiocyanate levels. Levels >10 mg/dL are considered to be clinically significant. If the patient is symptomatic, the antidote is sodium thiosulfate
   • Marked peripheral vasoconstriction—Incorrect; the vasodilating effects of the drug are not reduced with prolonged infusions
   • Depressed myocardial contractility—Incorrect; there is no direct effect on myocardial contractility
   • Development of sinus bradycardia—Incorrect; there is no effect on the SA node
Shock

- State that develops when there is inadequate tissue perfusion
  - Cells deprived of oxygen
  - Conversion to anaerobic metabolism
  - Production of lactic acid and acidosis

Cardiogenic Shock

- State in which the myocardium is unable to pump adequate cardiac output to maintain perfusion and meet metabolic demands of the body
Cardiogenic Shock Etiologies
- Loss of >40%–50% viable myocardium (most common cause)
- Cardiomyopathies
- Others
  - Hypovolemia
  - Metabolic dysfunction
  - Vasomotor dysfunction
  - Microcirculatory dysfunction
- Mechanical problems
  - Perforated intraventricular septum
  - Papillary muscle dysfunction/rupture
  - Myocardial rupture
  - Valvular heart disease
  - Post-op low CO syndrome

Cardiogenic Shock
- Pathophysiology
  - Marked ↓ CO: CI = <1.8 L/m²
  - Usual compensatory response is ↑ed SVR
    - If not, MAP will fall → ↓ coronary blood flow and worsening myocardial ischemia

Cardiogenic Shock
- If the compensatory mechanisms are working
  - ↑ SVR and ↑ catecholamine release
  - ↑ afterload    ↑ contractility
    - ↑ ischemia
    Contributing to a downward spiral
      and, ultimately, death
Cardiogenic Shock
- Left ventricular end-diastolic volume and left ventricular end-diastolic pressure
- Continue to ↑ further ↑s afterload
  - Limits filling of endocardial vasculature → endocardial ischemia
  - ↑ LVEDP is reflected back into pulmonary vasculature → ↑ PA pressures → pulmonary edema → arterial hypoxemia → cellular acidosis
- RV ischemia and failure follow

Hemodynamic Subsets

<table>
<thead>
<tr>
<th>CI ≥ 2.2 L</th>
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<td>PAOP ≤ 18 mmHg (warm and dry)</td>
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<tr>
<td>PAOP ≤ 18 mmHg (cool and dry)</td>
<td>PAOP ≥ 18 mmHg (cold and wet)</td>
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</table>

Management Goals
- Improve oxygen transport
  - Cardiac output
  - Oxygen content
    - Hgb
    - SaO₂
- Maintain ventilation
- Maintain/improve nutrition
- Decrease O₂ demand
- Prevent complications
Management Goals
- Pharmacological
- Inotropes
- Vasodilators
- Mechanical support
  - IABP
  - Ventricular assist device (VAD)
  - ECMO
- Surgical
  - Revascularization
  - VAD as destination therapy
  - Transplant

Review Questions

Question 8
A patient in cardiogenic shock has the following hemodynamic profile:
BP: 90/56 HR: 110 CO/CI: 1.4/0.8
PA: 36/20 PAOP: 18 SVR: 3000 RAP: 10
The patient is receiving an infusion of dobutamine 10 mcg/kg/min and epinephrine 0.02 mcg/kg/min. You would be most concerned about:
A. BP, CO/CI, PA
B. CO/CI, SVR
C. BP, SVR, CO/CI, CVP
D. All of the above
Question 8—Rationale

The patient is receiving an infusion of dobutamine 10 mcg/kg/min and epinephrine 0.02 mcg/kg/min. You would be most concerned about:

B. CO/CI, SVR; the high SVR has increased the workload of the heart so much that the ventricle is unable to contract effectively, causing the low CO/CI

- BP, CO/CI, PA: Incorrect: The marginal BP and slightly elevated PA pressure are not enough to impact the significantly reduced CO/CI
- BP, SVR, CO/CI, CVP: Incorrect: Again the marginal BP and the slightly elevated CVP are not significant enough to impact the CO/CI
- All of the above: Incorrect

Question 9

A patient in cardiogenic shock has the following hemodynamic profile:

BP: 90/56 HR: 110 CO/CI: 1.4/0.8
PA: 36/20 PAOP: 18 SVR: 3000 RAP: 10

The patient is receiving an infusion of: dobutamine 10 mcg/kg/min and epinephrine 0.02 mcg/kg/min. Which of the following interventions would be appropriate?

A. Reduce afterload with sodium nitroprusside
B. Elevate blood pressure with epinephrine
C. Reduce preload by giving a diuretic
D. Improve renal blood flow with dopamine 10 mcg/kg/min

Question 9—Rationale

The patient is receiving an infusion of: dobutamine 10 mcg/kg/min and epinephrine 0.02 mcg/kg/min. Which of the following interventions would be appropriate?

A. Afterload reduction with sodium nitroprusside; lowering the SVR should reduce myocardial workload and improve myocardial contractility, thereby increasing cardiac output/index

- Elevate blood pressure with epinephrine: Incorrect: Increasing the BP is not the primary issue. If afterload is reduced, the heart will contract more efficiently, thereby increasing CO/CI, and the BP will increase
- Reduce preload by giving a diuretic: Incorrect: The CVP of 10, sometimes used as an indicator of preload, is not significantly elevated
- Improve renal blood flow with dopamine 10 mcg/kg/min. Incorrect: Dopamine at 10 mcg/kg/min will produce significant alpha stimulation and vasoconstriction, thereby increasing afterload further and worsening the situation.
Question 10

Which of the following, accompanied by decreased intravascular volume, are hemodynamic signs of hypovolemic shock?

A. ↓ preload; ↑ afterload; ↑ cardiac output
B. ↓ preload; ↓ afterload; ↓ cardiac output
C. ↑ preload; ↑ afterload; ↓ cardiac output
D. ↓ preload; ↑ afterload; ↓ cardiac output

Question 10—Rationale

Which of the following, accompanied by decreased intravascular volume, are hemodynamic signs of hypovolemic shock?

D. ↓ preload; ↑ afterload; ↓ cardiac output; the ↓ intravascular volume causes the ↓ preload and ↓ CO/O. Afterload (vasoconstriction) increases in response to the low CO/O.

- ↓ preload; ↑ afterload; ↑ cardiac output: Incorrect. Hypovolemic shock is associated with a lower circulating blood volume; therefore, the cardiac output will be lower.
- ↓ preload; ↓ afterload; ↓ cardiac output: Incorrect. Hypovolemic shock is characterized by a reduced preload and cardiac output, but afterload will be increased due to vasoconstriction to drive blood flow to the vital organs.
- ↑ preload; ↑ afterload; ↓ cardiac output: Incorrect. While afterload is increased and the cardiac output is low, preload will be low due to the reduction in circulating blood volume.

Summary
REFERENCES – Cardiovascular:


