Testable Nursing Actions

- Normal/abnormal physical assessment
- Apply leads for ECG monitoring
- Monitor hemodynamic status
- Manage patients receiving cardiovascular medications
- Recognize indications for and manage patients with an intra-aortic balloon pump (IABP)
- Manage patients following percutaneous coronary interventions (PCI)

- Recognize indications for and manage patients requiring:
  - 12-lead ECG
  - Arterial line
  - PAC/CVP/SvO2 monitoring
  - Defibrillation
  - Pacing: transthoracic/transvenous
- Recognize signs and symptoms of cardiovascular emergencies and indicated interventions; seek assistance as needed
Road Map: Cardiovascular System

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

Heart Failure

- Ventricles fail to pump adequate cardiac output to meet the metabolic requirements of the tissues
- May be due to:
  - Systolic dysfunction
  - Diastolic dysfunction
  - Or both

Heart Failure

- Ventricles fail to pump adequate cardiac output to meet the metabolic requirements of the tissues
- May be due to:
  - Systolic dysfunction
  - Diastolic dysfunction
  - Or both
Systolic Dysfunction
- Problem with ejection
- Falling cardiac output
- Low filling pressures

Diastolic Dysfunction
- Problems with ventricular filling
- Associated with:
  - High filling pressures
  - Pulmonary congestion

Cardiomyopathies
- Three types
  - Dilated
  - Hypertrophic
  - Restrictive
Dilated Cardiomyopathy

- Causes
  - Coronary artery disease
  - Viral
  - Chemotherapy
  - Pregnancy
  - Parasitic—eg, Chagas
  - Alcohol

Progression of Heart Failure

Hypertrophic Obstructive Cardiomyopathy

- Causes
  - Aortic stenosis
  - Congenital

- Management
  - Reduce outflow obstruction
  - Beta blockers
  - Other
Restrictive Cardiomyopathy

- Causes
  - Infiltrative diseases

- Management
  - Focus on symptom management
  - Halt disease progression

Congestive Heart Failure
Signs and Symptoms

**Left ventricular failure**

- ↑ Left ventricular end-diastolic pressure
- ↓ Cardiac output
- Sinus tachycardia
- ↓ Urine volume
- Cool skin
- Personality changes, confusion, lethargy
- Weak, tired
- Anorexia
- ↓ Exercise capacity

- Pulmonary signs and symptoms: dry, hacky cough; shortness of breath; dyspnea on exertion; paroxysmal nocturnal dyspnea
- Pulmonary edema: rales, crackles, frothy sputum, hemoptysis
Congestive Heart Failure
Signs and Symptoms

- ↑PAP
- ↓
  - Right ventricular failure
  - ↑right ventricular and right atrial pressures
  - ↑venous pressures
  - Distended neck veins (↑central venous pressure)
  - Dependent edema (pedal, sacral, ascites)
  - Hepatomegaly (positive hepatojugular reflux)

Congestive Heart Failure
Management/Target Goals

- Improve cardiac output/cardiac index
  - Rest
  - Pharmacologic interventions
    - Inotropes
    - Vasodilators to reduce preload and afterload
    - Beta blockers to prevent sudden cardiac death
    - ACE inhibitors to prevent ventricular remodeling
    - Digitals
    - Diuretics

Congestive Heart Failure
Management/Target Goals

- ECG monitoring
  - Sudden cardiac death is common

- Mechanical assist
  - IABP—reduces afterload and improves coronary perfusion
  - Left ventricular assist device—Bridge to recovery, transplant, or destination therapy

- Prior to discharge
  - Patient and family education
Acute Pulmonary Edema

- Caused by a marked increase in pulmonary capillary pressures resulting in leakage of fluid into the pulmonary interstitial spaces
- Etiologies
  - Left ventricular failure
  - Volume overload, often iatrogenic

Acute Pulmonary Edema

- Characterized by abrupt onset of respiratory distress, including:
  - Tachypnea
  - High Fowler's position
  - Productive cough: frothy sputum, often pink

Acute Pulmonary Edema

- Diagnosis
  - Chest x-ray
  - Arterial blood gases
  - Falling PO₂, SpO₂
  - Hemodynamics
    - Elevated pulmonary artery occlusion pressure (PAOP) — often >28 mmHg
  - Clinical presentation
- Management
  - Oxygen; may require intubation with ventilatory support
  - Diuretics
  - Treat the cause (eg, heart failure, rhythm disturbances, etc.)
Which of the following applies to systolic failure?

A. Problem with ventricular filling
B. Association with a low CO/CI
C. “Filling” pressures will be high initially
D. Elevated PAP

**Question 1—Rationale**

Which of the following applies to systolic failure?

B. Association with a low CO/CI—The problem is with impaired ventricular contractility causing the cardiac output/cardiac index to be low
  - Problem with ventricular filling—The problem is with impaired contractility
  - “Filling” pressures will be high initially—Filling pressures are low-to-normal, initially
  - Elevated PAP will be low, initially
Question 2

Beta blockers are important for patients with heart failure because:

A. The heart rate should be increased
B. Ventricular fibrillation/ventricular tachycardia are the most common causes of death in these patients
C. They further reduce myocardial contractility
D. None of the above

Question 2—Rationale

Beta blockers are important for patients with heart failure because:

B. Ventricular fibrillation/ventricular tachycardia are the most common causes of death in these patients—Beta blockers are the only class of drugs that have been shown to reduce sudden cardiac death due to ventricular fibrillation/ventricular tachycardia
  - Beta blockers slow the heart rate, they do not increase it
  - Contractility may actually improve due to recovery of ventricular function

Question 3

IABP produces which of the following effects?

A. Increases ventricular afterload
B. Decreases coronary perfusion
C. Decreases coronary and cerebral perfusion
D. Decreases ventricular afterload
**Question 3—Rationale**

IABP produces which of the following effects?

D. Decreases ventricular afterload—The IABP increases diastolic blood pressure, which increases coronary perfusion. The balloon deflates just before aortic valve opening, reducing afterload

- Increases ventricular afterload—The balloon deflates a split second before the aortic valve opens. This creates a 40 mL vacuum, which lowers afterload
- Decreases coronary perfusion—Rather, IABP increases coronary perfusion
- Decreases coronary and cerebral perfusion—The balloon is inflated during diastole, increasing diastolic blood pressure and coronary and cerebral perfusion

**Question 4**

Why are ACE inhibitors frequently ordered for patients with heart failure?

A. To prevent atrial fibrillation
B. To prevent the ventricular remodeling process following acute myocardial infarction
C. To increase preload
D. To prevent sudden cardiac death

**Question 4—Rationale**

Why are ACE inhibitors frequently ordered for patients with heart failure?

B. To prevent the ventricular remodeling process following acute myocardial infarction—ACE inhibitors block the production of angiotensin II, which is believed to contribute to ventricular remodeling and development of a dilated cardiomyopathy

- To prevent atrial fibrillation—ACE inhibitors do not have antidysrhythmic properties
- To increase preload—ACE inhibitors will actually help reduce preload by preventing sodium and water reabsorption in the distal renal tubule
- To prevent sudden cardiac death—These agents have no effect on sudden cardiac death
Acute Coronary Syndromes

- Spectrum of coronary artery disease (atherosclerotic process) that includes:
  - Unstable angina
  - Non-ST-elevation myocardial infarction (NSTEMI)
  - ST-elevation myocardial infarction (STEMI)

Unstable Angina

- *Stable* angina is defined as angina that has not changed in any way for a period of >4 weeks
  - Intensity of pain, exertion factors, etc., have remained the same
- *Unstable* angina means that some aspect of the angina is changing
  - Chest pain occurs with less exertion or at rest
  - Intensity of pain is more severe
  - May require more nitroglycerin for relief

Myocardial Infarction

- STEMI vs NSTEMI
Myocardial Infarction

- ECG changes are secondary to occlusion of vessel

Indicative changes

Normal (baseline)

Current of Ischemia
Necrosis/Infarction
Pathological "Q-wave"
- Width >0.038 seconds
- Depth >25% of accompanying "R" wave

Indicative Changes
- Current of ischemia/primary T-wave inversion
- Current of injury/ST-segment elevation
- Current of necrosis/pathological Q-wave
Primary Sites for MIs

- Squau
- Anterior
- Inferior
- Posterior
- Lateral

Vessel involved
- Right coronary artery

Indicative changes
- II, III, AVF

Reciprocal changes
- I, AVL

Inferior MI

- Vessel involved
  - Right coronary artery
- Indicative changes
  - II, III, AVF
- Reciprocal changes
  - I, AVL
Anterior MI

- Vessel involved
  - Left anterior descending (LAD)
- Indicative changes
  - V2, V3, V4
- Reciprocal changes
  - II, III, AVF

Anterior wall

Often associated with anterior MI

- Vessel involved
  - LAD
- Indicative changes
  - V1, V2
- Reciprocal changes
  - V5, V6

Septal MI
Lateral MI

- Vessel involved
  - Circumflex
- Indicative changes
  - I, AVL, V5, V6
- Reciprocal changes
  - II, III, AVF
Posterior MI

- Vessel involved
  - Right coronary artery, if dominant
- Indicative changes
  - V7, V8, V9
- Reciprocal changes
  - V1, V2, V3
Non-STEMI

- ST-segment depression over involved area of muscle
  - Major predictor of mortality
  - 40%-50% will develop STEMI within hours
- Occasionally T-wave changes alone
  - Best prognosis
  - >2.0 mm suggests critical stenosis of LAD coronary artery
Non–STEMI

ST depressed or elevated

Acute Management

- Aspirin
- Beta blocker
- Immediate reperfusion
  - Fibrinolytic (screen for contraindications)
  - Primary PCI
GP IIb IIIa Inhibitors

- Include:
  - Abciximab (ReoPro)
  - May be able to reverse with platelets
  - Eptifibatide (Integrilin)
  - Dose adjusted for renal insufficiency

- Screen as you would for a fibrinolytic
- Observe for onset of:
  - Bleeding
  - Thrombocytopenia

Postreperfusion Monitoring

- Observe for acute closure
- ST-segment changes are an early indicator
- ≥80% have silent ischemia

Discharge Medications

- Aspirin
- Beta blocker
- ACE inhibitor if ejection fraction <40%
Question 5
Changes that indicate a STEMI include:
A. Tall peaked P-wave, PR-segment elevation
B. Widened QRS duration >0.12 seconds
C. T-wave inversion, ST-segment elevation, pathological Q-wave
D. Prolonged PR interval

Question 5—Rationale
Changes that indicate a STEMI include:
C. T-wave inversion, ST-segment elevation, pathological Q-wave
   • Tall peaked P-wave, PR-segment elevation—These changes are not indicative of MI
   • Widened QRS duration >0.12 seconds—A myocardial infarction can cause the QRS duration to widen, resulting in a bundle branch block, but this is not an early indicative change
   • Prolonged PR interval—The PR interval may become prolonged with ischemia of the AV node, but this is not a primary indicative change
Question 6
Indicative changes for an inferior MI may be found in leads:

A. II, III, AVF
B. V2, V3
C. V2, V3, V4
D. V5, V6, I, aVL

Question 6—Rationale
Indicative changes for an inferior MI may be found in leads:

A. II, III, AVF—These leads have their positive recording electrode on the left leg, which means the electrode directly faces the inferior wall of the left ventricle
   • V1, V2—These leads “look” at the intraventricular septum
   • V2, V3, V4—These leads reflect the anterior wall of the left ventricle
   • V5, V6, I, aVL—These are the lateral leads

Question 7
NSTEMI is characterized by:

A. ST-segment elevation and T-wave inversion
B. Normal troponin I levels
C. ST-segment depression and T-wave inversion
D. Absence of symptoms
Question 7—Rationale

NSTEMI is characterized by:

C. ST-segment depression and T-wave inversion—Common ECG changes associated with a NSTEMI include ST-segment depression and/or T-wave inversion. There is no progression to the development of a Q-wave

- ST-segment elevation and T-wave inversion—Non-ST-segment MI is not associated with ST-segment elevation. However, the T wave may be inverted.
- Normal troponin I levels—The troponin levels will be elevated
- Absence of symptoms—The patient will have signs and symptoms similar to those patients who present with ST-segment MI

Question 8

STEMI patients should have the following:

A. ECG ≤60 minutes of arrival to the ED
B. Initiation of abciximab on arrival
C. Blood cultures ≤30 minutes of arrival
D. Primary coronary intervention ≤90 minutes of arrival

Question 8—Rationale

STEMI patients should have the following:

D. Primary coronary intervention ≤90 minutes of arrival to the ED—Once the STEMI is confirmed in the ED, the patient should undergo a primary PCI ≤90 minutes of arrival

- ECG ≤60 minutes of arrival to the ED—The ECG should be obtained ≤30 minutes of arrival to the ED
- Initiation of abciximab on arrival—There is no indication for initiation of a GP IIb IIIa inhibitor on arrival
- Blood cultures ≤30 minutes of arrival—This does not apply
Question 9

Mr. Jones arrived in the ED complaining of chest pressure and shortness of breath for the past 2 hours. The ECG demonstrated 5 mm ST-segment elevation in leads V2, V3, and V4. He was taken immediately to the cardiac catheterization lab, where a 100% occlusion of the proximal LAD coronary artery was discovered. Angioplasty was performed and a stent deployed. Following the procedure, he was taken to the recovery area. Mr. Jones is likely to receive which medications to assure patency of his coronary artery?

A. Dobutamine
B. Clopidogrel
C. Atropine
D. Dopamine

Question 9—Rationale

B. Clopidogrel—Clopidogrel, along with aspirin, will be given not only during the patient's hospital stay but also for several months thereafter.
- Dobutamine—Dobutamine is a positive inotrope, does not affect the clotting system in any way.
- Atropine—Atropine has no effect on the clotting cascade and will not assure patency of the vessel.
- Dopamine—Dopamine has no effect on the clotting cascade and will not assure patency of the vessel.

Question 10

Careful monitoring of Mr. Jones after transfer to the ICU would include:

A. ST-segment monitoring
B. Arterial blood gases every 12 hours
C. Biomarker levels every 4 hours x 48 hours
D. White blood cell count
Question 10—Rationale

Careful monitoring of Mr. Jones after transfer to the ICU would include:

A. ST-segment monitoring—ST-segment is monitored for acute closure of the target vessel
   • Arterial blood gases every 12 hours—Not applicable unless the patient demonstrates signs of respiratory insufficiency
   • Biomarker levels every 4 hours x 48 hours—Usually ordered daily to trend over time
   • White blood cell count—Not applicable unless there is a suspicion of concomitant infection

Complications of MI

- Heart failure
- Cardiogenic shock
- Arrhythmias
- Mechanical complications
  - Papillary muscle rupture/dysfunction → mitral regurgitation
  - Cardiac tamponade
  - Perforated intraventricular septum

Acute Mitral Regurgitation

- Papillary muscle dysfunction/rupture
- Acute onset loud systolic murmur
- Abrupt decline of BP and cardiac output/cardiac index
- Development of large “V” wave in PAOP tracing
Papillary Muscle Rupture

Mitral valve

Large “V” Waves Associated with Mitral Insufficiency

Cardiac Tamponade
- Falling blood pressure and cardiac output/cardiac index
- Narrowing pulse pressure
- Sinus tachycardia
- Distended neck vein, elevated central venous pressure
- Equilibration of pressures (diastolic plateau)
- Muffled heart tones
- Pulseless electrical activity
Perforated Intraventricular Septum

- Abrupt drop in blood pressure, cardiac output/cardiac index
- Loud holosystolic murmur
- Insertion of pulmonary artery catheter
  - Look for oxygen step-up from right atrium to right ventricle
  - Presence of large "V" wave in PAP tracing

Perforated Intraventricular Septum

- Note large "V" wave in wedge pressure tracing

Valvular Heart Disease

- Mitral valve
  - Insufficiency
  - Association with large V wave in pulmonary artery wedge pressure waveform
  - Elevated PAP
  - STenosis
  - Pulmonary artery wedge pressure not helpful—falsely elevated
  - PAP elevated

- Aortic valve
  - Insufficiency
  - Left ventricular volume overload
  - STenosis
    - Develop left ventricular hypertrophy
    - Volume-dependent
    - Onset of atrial fibrillation can be catastrophic due to the loss of atrial kick
    - Elevated PAP

12/2015
Coronary Bypass Surgery

- Approaches
  - Minimally invasive
  - Sternotomy
- Use of cardiopulmonary bypass
  - On pump
  - Off pump

Postoperative Management

- Assess hemodynamic stability
  - Hemodynamic assessment
  - Titrating infusions
  - IABP
  - Electrolyte status
  - Hypokalemia
  - Hypomagnesemia
- Cardiac arrhythmias
- Ventilatory status
  - ABG
  - Early extubation protocol (if appropriate)

Postoperative Management

- Pain control can be challenging
  - Use of local anesthetics and delivery systems
  - Use of epidurals and PCA pumps
  - Incisional care
  - Activity progression
  - ICU length of stay
    - 1 day
Early Complications

- Coagulopathies
- Excessive bleeding
- Cardiac tamponade
- Electrolytes—potassium, magnesium
- Respiratory failure/atelectasis
- Renal insufficiency/acute tubular necrosis
- Cardiogenic shock
- Stroke

Peripheral Arterial Disease (PAD)

- Etiology
  - Atherosclerosis—may have history of stroke, coronary artery disease, and/or hypertension

Peripheral Arterial Disease

- Signs and symptoms
  - Pain—especially with elevation
  - Pale, mottled with rubor with dependence of extremity
  - Ulcers/gangrene
  - Hair loss, skin is thin and shiny
  - Weak or absent peripheral pulses
  - Sluggish capillary refill
Assessing the 6 Ps

- Pain
- Pallor
- Paresthesias
- Pulselessness
- Paralysis
- Poikilothermia

Diagnostic Studies—PAD

- Doppler duplex
- Ankle-brachial index (ABI)
  - Used as a screening tool
  - Ankle systolic blood pressure divided by systolic blood pressure in the arm to derive an index
  - Peripheral angiography
- ABI scoring
  - Normal: 0.9–1.3 (pressure normally higher in the ankle)
  - ABI <0.9: positive for PAD
  - ABI <0.4 indicates severe ischemia

Management—PAD

- Treatment
  - Thrombolysis
  - Thrombectomy
  - Percutaneous angioplasty
  - Endovascular stent graft
- Clinical implications
  - Assess for palpable pulses
  - Limbs are warm and pink, with good capillary refill
  - No signs of bleeding
  - Pain is absent
Aortic Aneurysms

- Most common and most lethal event involving the aorta
- If not treated, the mortality rate is:
  - >50% within 48 hours
  - 80% within 2 weeks
- Most common cause of death:
  - Rupture
  - Dissection

Acute Aortic Dissection

- Medial degeneration
  - Age
  - Marfan syndrome
  - Idiopathic
- Aortitis
  - Bacterial
  - Mycotic aneurysm
- Hypertension
- Atherosclerosis
- Trauma
- Smoking
- Hyperlipidemia
- Iatrogenic
- Complication of aortic surgery
Types—Acute Aortic Dissection

- True
  - All layers involved
- False
  - Partial or complete disruption of aortic wall with blood contained within the adventitial layer

Description—Acute Aortic Dissection

- Shape
  - Fusiform
  - Saccular
- Location
  - Ascending
  - Transverse
  - Descending

Location—DeBakey System

Acute Aortic Dissection

- Type I
  - Originates in ascending aorta, propagates at least to the aortic arch and often beyond it distally
- Type II
  - Originates in and is confined to the ascending aorta
- Type III
  - Originates in descending aorta, rarely extends proximally
Location—Acute Aortic Dissection

- Involves ascending aorta
- Surgical emergency
- DeBakey type I and II
- Ascending aorta not involved
- Type III DeBakey

Diagnostic Tests: Acute Aortic Dissection

- Rule out myocardial infarction
- CT scan
- MRI
- Transesophageal echocardiogram

Signs and Symptoms: Acute Aortic Dissection

- Ascending aorta
  - Chest pain, aortic insufficiency, congestive heart failure
- Transverse aorta
  - Dyspnea, stridor, hoarseness, cough
  - Chest pain
  - Jugular venous distention (less common)
- Descending aorta
  - Back or chest pain
Treatment: Acute Aortic Dissection

- Medical management
- Surgical repair

Medical Management: Acute Aortic Dissection

- Goals
  - Reduce left ventricular contractility and velocity of blood flow
  - Reduce systolic blood pressure to lowest possible level
  - Type B—medical management first

Medical Management: Acute Aortic Dissection

- Relief of pain is the most important clinical sign
- Vigilant monitoring of blood pressure
  - Especially as patient increases activity level
  - Assess for postural hypotension
- Educate patient and family about meds
Medical Management: Acute Aortic Dissection

- Morphine sulfate to control pain
- Combination of beta blockers and vasodilators to lower systolic blood pressure
  - Start beta blocker before vasodilator
  - Reduce systolic blood pressure to 100–120 mmHg or lower if patient complains of pain

Surgical Repair: Acute Aortic Dissection

- Type A—surgical emergency
- Routine ICU care
- Special emphasis on management of hypertension
- Monitor spinal fluid pressures
  - Goal: spinal pressure ≤8–10 mmHg
- Drain spinal fluid to maintain
- Neuro assessments
  - Central
  - Peripheral

Review Questions
Question 11

An important feature to differentiate ventricular septal rupture from acute mitral regurgitation is:

A. Abrupt decrease of blood pressure, cardiac output/cardiac index
B. Abrupt onset of systolic murmur
C. Oxygen step-up between right atrium and right ventricle
D. Appearance of "V" wave distorting PAOP waveform

Question 11—Rationale

An important feature to differentiate ventricular septal rupture from acute mitral regurgitation is:

C. Oxygen step-up between right atrium and right ventricle—Ventricular septal rupture is associated with a left-to-right shunt, causing arterial blood from the left ventricle to mix with blood in the right ventricle. This increases the oxygen saturation of the blood in the right ventricle.
- Abrupt decrease of blood pressure, cardiac output/cardiac index—A VSD and mitral regurgitation will present with these signs
- Abrupt onset of systolic murmur—A VSD and mitral regurgitation are associated with a systolic murmur
- Appearance of "V" wave distorting PAOP waveform—Both will produce a "V" wave that will distort the PAOP waveform

Question 12

What is the advantage of a tissue valve over a mechanical valve?

A. A tissue valve lasts longer
B. A tissue valve prevents the need for lifelong anticoagulant therapy
C. A mechanical valve has a higher incidence of atrial fibrillation
D. A tissue valve is easier to implant
Question 12—Rationale
What is the advantage of a tissue valve over a mechanical valve?
B. A tissue valve prevents the need for lifelong anticoagulant therapy—
   Tissue valves do not require long-term anticoagulant therapy. Mechanical
   valves do because thrombus material develops on the prosthetic valve
   • A tissue valve lasts longer—Mechanical valves have been shown to last
     longer than tissue valves
   • A mechanical valve has a higher incidence of atrial fibrillation—Atrial
     fibrillation is common in patients with valvular heart disease
   • A tissue valve is easier to implant

Question 13
Nursing priorities for patients immediately after cardiac surgery include:
A. Maintain hemodynamic stability
B. Prevent hyperkalemia
C. Quickly warm the patient’s body temperature to 38°C
D. Keep blood glucose levels <90 mg/dL

Question 13—Rationale
Nursing priorities for patients immediately after cardiac surgery include:
A. Maintain hemodynamic stability—Immediate post-op management focus
   is on titration of inotropes, vasodilators, and pressors to optimize
   myocardial performance
   • Prevent hyperkalemia—Serum potassium and magnesium levels are
     monitored to prevent hypokalemia and hypomagnesemia
   • Quickly warm the patient’s body temperature to 38°C—Rapid
     warming often leads to the patient’s temperature climbing out of
     control. Slower warming to 37°C is preferred
   • Keep blood glucose levels <90 mg/dL—Target blood glucose levels
     range between 120 mg/dL and 180 mg/dL
Question 14

JR is a 48-year-old male who arrived in the ED complaining of chest pain. His blood pressure is 186/110 mmHg. He is diaphoretic and pale. ECG is normal. A CT scan of the chest reveals a type B thoracic aneurysm. JR is admitted to the ICU for closer monitoring. What order would you expect to be the priority for medical management of this patient?

A. Esmolol infusion to lower systolic blood pressure to 120 mmHg
B. Dopamine at 5 mcg/kg/min to support renal perfusion
C. Dobutamine 2.5 mcg/kg/min to increase cardiac output/cardiac index
D. Intermittent positive pressure breathing (IPPB) treatments to improve gas exchange

Question 14—Rationale

JR is a 48-year-old male who arrived in the ED complaining of chest pain. His blood pressure is 186/110 mmHg. He is diaphoretic and pale. ECG is normal. A CT scan of the chest reveals a type B thoracic aneurysm. JR is admitted to the ICU for closer monitoring. What order would you expect to be the priority for medical management of this patient?

A. Esmolol infusion to lower systolic blood pressure to 120 mmHg—it is imperative that systolic blood pressure be lowered to prevent rupture of the aneurysm  
   - Dopamine at 5 mcg/kg/min to support renal perfusion—This may elevate systolic blood pressure  
   - Dobutamine 2.5 mcg/kg/min to increase cardiac output/cardiac index—There is no issue with cardiac output/cardiac index  
   - IPPB treatments to improve gas exchange—There is no clinical indication for this

Question 15

Following surgical repair of a thoracoabdominal aortic aneurysm, a patient is at risk to develop:

A. Hypercoagulable disorder  
B. Paralysis of lower extremities  
C. Hyperactive bowel sounds  
D. Paralysis of the recurrent laryngeal nerve
Following surgical repair of a thoracoabdominal aortic aneurysm, a patient is at risk to develop:

B. Paralysis of lower extremities—After repair of a thoracoabdominal aortic aneurysm, a patient is at risk to develop ischemia of organ systems that receive blood supply from the aorta. The spinal cord is at highest risk
- Hypercoagulable disorder—Rather, such patients are more likely to bleed
- Hyperactive bowel sounds—Patients are at risk for the development of an ischemic bowel
- Paralysis of the recurrent laryngeal nerve—This might occur with an aneurysm involving the transverse aorta, but not the thoracic abdominal aorta distal to the left subclavian artery