

CRITICAL CARE NURSING COURSE  
BROOKE ARMY MEDICAL CENTER  
FORT SAM HOUSTON, TEXAS 78234

Hemodynamic Monitoring

Objectives

1. Terminal Learning Objective

Monitor the hemodynamics of the critical ill patient.

2. Enabling Learning Objectives

- a. Identify the uses and clinical indications for hemodynamic monitoring.
- b. Discuss cardiac output and its determinants (heart rate, stroke volume) to include normal values.
- c. Describe the effects of preload, afterload, and contractility upon cardiac output.
- d. Identify normal values and corresponding waveforms obtained from arterial central venous, and pulmonary arterial lines.
- e. Identify abnormal values and waveforms and the related nursing actions.
- f. Discuss/perform the steps the nurse must take to ensure accuracy of hemodynamic measurements to include the following:
  - (1) System set-up
  - (2) Zeroing
  - (3) Leveling
  - (4) Correct waveform identification
  - (5) Balloon inflation volume check
  - (6) Identification of end expiration
    - (a) Spontaneous breath
    - (b) Ventilator breath
  - (7) Appropriate cardiac output injectate volume
  - (8) Correct injection technique
  - (9) Cardiovascular profile analysis
- g. Discuss SVO<sub>2</sub> monitoring to include normal values, how measurement is obtained, and the significance of the value in the management of the patient.

## NOTES

### A. Uses of and Clinical Indications for Hemodynamic Monitoring

#### 1. The three uses of hemodynamic monitoring are

- a. To monitor cardiac output and its components of preload, afterload, and contractility in patients with abnormal cardiac output or the potential for abnormal cardiac output
- b. To evaluate changes in cardiac output from a patient's baseline
- c. To evaluate the effectiveness of treatments to increase cardiac output

#### 2. Clinical indications for hemodynamic monitoring include

##### a. Patients with actual decreased cardiac output or shock. Patients in

- (1) Hypovolemic shock from dehydration, burns, blood loss from trauma/surgery
- (2) Distributive shock from anaphylaxis, sepsis, or spinal cord injury
- (3) Obstructive shock from pulmonary emboli
- (4) Cardiogenic shock from MI or cardiomyopathy

##### b. Patients with the potential for decreased cardiac output

- (1) Patient's who are dehydrated, burned, have experienced blood loss from trauma or surgery, with spinal cord injury, MI, CHF or cardiomyopathy before shock occurs
- (2) Patients undergoing major surgical procedures who
  - (a) Have a history of pulmonary or cardiac disease
  - (b) Are having extensive abdominal surgery

##### c. Patients who are receiving acute treatments to maintain or optimize their cardiac output

## B. Cardiac Output and Its Determinants

1. Cardiac output - normal value of 4-8 lmp
  - a. Definition - the amount of blood pumped by the heart per minute
  - b. Calculation - Cardiac output = heart rate X stroke volume
2. Heart rate- normal value 60-100 beats per minute (bpm)
  - a. Can increase cardiac output by increasing the heart rate up to a certain point (about 120 bpm), with faster heart rates not enough time to fill the ventricles fully
  - c. Factors determining heart rate
    - (1) Temperature
    - (2) Metabolic rate (endocrine)
    - (3) Level of oxygen and carbon dioxide
    - (5) Level of catecholamines
  - d. Abnormal value effects on cardiac output
    - (1) Bradycardia < 60
      - ↓ number of contractions --> ↓ rate of pumping
    - (2) Tachycardia > 100
      - ↓ filling time --> ↓ volume of pumping
3. Stroke volume - normal value 60-130 ml
  - a. Definition - the amount of blood ejected by the left ventricle with one beat of the heart
  - b. Factors determining stroke volume
    - (1) Preload
    - (2) Afterload

(3) Contractility

d. Causes of abnormal values

(1) Decreased stroke volume (SV)

(a) Decreased preload

- 1) Vasodilation
- 2) Hypovolemia
- 3) Increased intrathoracic pressure
- 4) Cardiac tamponade
- 5) Stiff ventricle
- 6) Loss of atrial kick/atrial dysrhythmias
- 7) Increased heart rate

(b) Decreased contractility

- 1) Hypoxemia
- 2) Acidosis
- 3) Decreased preload (i.e., Starling's Law)
- 4) Drugs that depress contractility

(c) Increased afterload

- 1) Increased systemic vascular resistance
- 2) Vasoconstriction
- 3) Aortic stenosis
- 4) Vasopressors

(2) Increased stroke volume

- (a) Increased preload
  - 1) Vasoconstriction
  - 2) Heart failure
  - 3) Hypervolemia
- (b) Increased contractility - usually a result of medication
- (c) Decreased afterload
  - 1) Vasogenic vasodilatation
  - 2) Drug induced vasodilatation

NOTE: Whatever affects stroke volume affects cardiac output the same way.

#### 4. Preload

- a. Definition - the volume or pressure in the ventricle at the end of diastole - just before systole
- b. The greater the volume of blood in the ventricle just prior to systole, the greater the stroke volume
- c. The preload of the right ventricle is known as right ventricular end diastolic pressure or RVEDP. Clinically measured as a right atrial ( RA) or central venous pressure (CVP). Normal value: 2-6 mm Hg
- d. The preload of the left ventricle is known as left ventricular end diastolic pressure LVEDP. Clinically measured as pulmonary artery wedge pressure or PAWP. Normal value for PAWP: 8-12 mm Hg
- e. Determinants of preload are
  - (1) The volume of blood delivered to the ventricle
    - (a) Blood volume
      - 1) Total blood volume
      - 2) Relative blood volume

- (b) Heart rate
- (c) Timing of atrial contraction
- (d) Ventricular compliance

5. Afterload

- a. Definition - the resistance to blood flow as it leaves the ventricles; resistance against which the ventricle ejects its volume
- b. Factors determining afterload
  - (1) Aortic valve competence
  - (2) Aortic compliance (stiffness)
  - (3) Systemic vascular resistance
- c. Clinically, afterload is measured as systemic vascular resistance (SVR).  
Normal value for SVR 800-1200 dynes/sec<sup>2</sup>

6. Contractility

- a. Definition - the force of the heart's contraction
- b. The greater the contractility, the greater the cardiac output; the lower the contractility, the lower the cardiac output
- c. Factors determining contractility
  - (1) Sympathetic nervous system activity
  - (2) Inotropic agents/negative inotropic agents
  - (3) Amount of functional heart muscle
  - (4) Nutrient supply (O<sub>2</sub>, CO<sub>2</sub>, Na<sup>+</sup>, K<sup>+</sup>, Ca<sup>+</sup>)
  - (5) Heart rate
- d. Causes of abnormal values
  - (1) Decreased contractility

- (a) Beta blockers
- (b) Calcium channel blockers
- (c) Hypoxemia
- (d) Acidosis
- (e) Myocardial ischemia/infarction

(2) Increased contractility

- (a) Catecholamines
- (b) Positive inotropic drugs: Digoxin, Epinephrine, Isuprel, Dopamine, Dobutamine

C. Introduction to Hemodynamic Monitoring: Arterial, CVP and PA Monitoring  
Basic Waveform Analysis

I. Arterial monitoring

a. Indications

- (1) When accuracy in BP measurement essential -- i.e., use of vasoactive drips
- (2) Patients who require frequent ABG's

b. Limitations

- (1) Care of the catheter influences the reading zeroing/calibrating equipment, position of patient/bed
- (2) Pressure varies in value in various locations

The more peripheral the A-line, the higher the systolic and lower the diastolic readings; the MAP remains the same

- (3) A-line pressures should be 5-20 mm HG higher than cuff measurements
- (4) If A-line pressure < cuff pressure reading or >5-20 mm Hg over cuff pressure measurement, one of the following is occurring

- (a) Equipment problem

- 1) BP cuff too small for the patient's arm --> erroneously high cuff reading
- 2) BP cuff too large for the patient's arm --> erroneously low cuff reading
- 3) A-line with poor dynamic response

(b) Patient problem

Patient in severe shock or hypothermia--> vasoconstriction causes lower indirect (cuff) pressure measurements; direct BP measurements can be 150 mm > indirect measurement

c. Pressures and waveforms

(1) Normal pressures and waveforms

- (a) Systolic pressure 90-140 mm Hg
- (b) Diastolic pressure 60-90 mm Hg
- (c) Mean arterial pressure
  - 1) Can calculate by  $\frac{\text{systolic} + 2(\text{diastolic})}{3}$
  - 2) Normal value 70-105 mm Hg

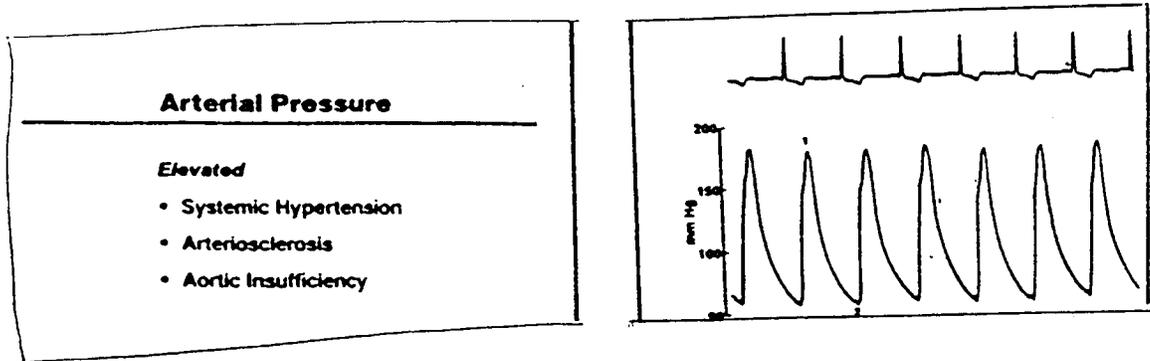
(2) Phases and correlation of mechanical events

- (a) Initial sharp upstroke (anacrotic limb) - represents rapid ejection of blood from the ventricle through the open aortic valve
- (b) Peak of anacrotic limb - represents systolic blood pressure
- (c) Dicrotic limb - blood flows into the periphery and pressure falls--> waveform begins downward trend
- (d) Dicrotic notch - pressure in the ventricle is less than pressure in aortic root--. aortic valve closes

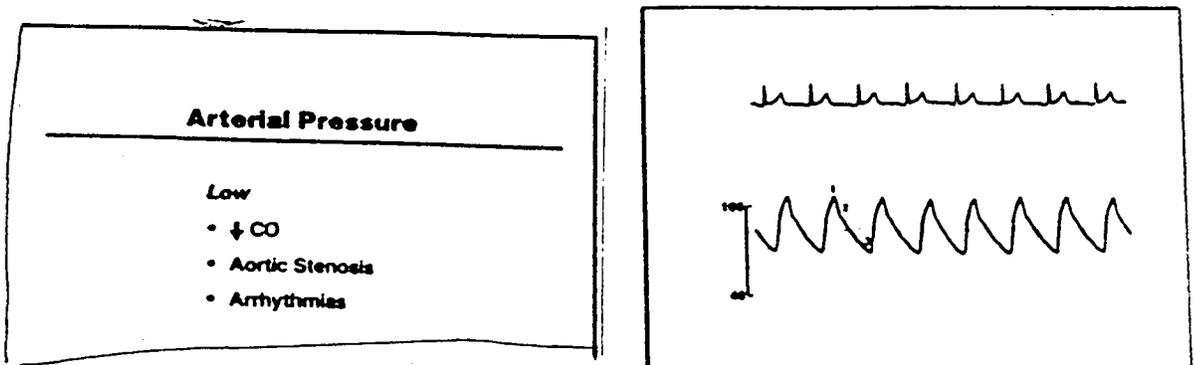
(3) Correlation with EKG

- (a) The peak systolic pressure wave will appear just after the QRS
  - (b) The dicrotic notch is associated with the end of the T wave
- (4) Abnormal pressures

Elevated Arterial Pressure Waveforms



Decreased Arterial Pressure Waveforms



- (a) Increased
  - 1) Systemic hypertension/vasoconstriction
  - 2) Atherosclerosis
  - 3) Aortic insufficiency
- (b) Decreased
  - 1) Low cardiac output
  - 2) Aortic stenosis

3) Dysrhythmias

4) Distributive shock/vasodilatation

d. Complications of A-line

(1) Arterial thrombosis

(2) Infection

(3) Bleeding

2. Central venous pressure monitoring

a. Indications

(1) Can provide adequate data for fluid management in most young patients with normal cardiac function

(2) Measurement of RV preload

(3) Can be used to assess intravascular volume status, adequacy of venous return and administration of IV fluids/medications

(4) Trends and responses to fluid challenges more important than absolute values

b. Limitations

(1) Hypovolemia - if venous tone increases or compromised myocardial function exists (CHF), the CVP may not fall proportionate to the amount of fluid lost. A minimal CVP response to rapid infusions of fluids is characteristic of hypovolemia. A rapid rise in CVP suggests that the patient has adequate blood volume or poor right ventricular function.

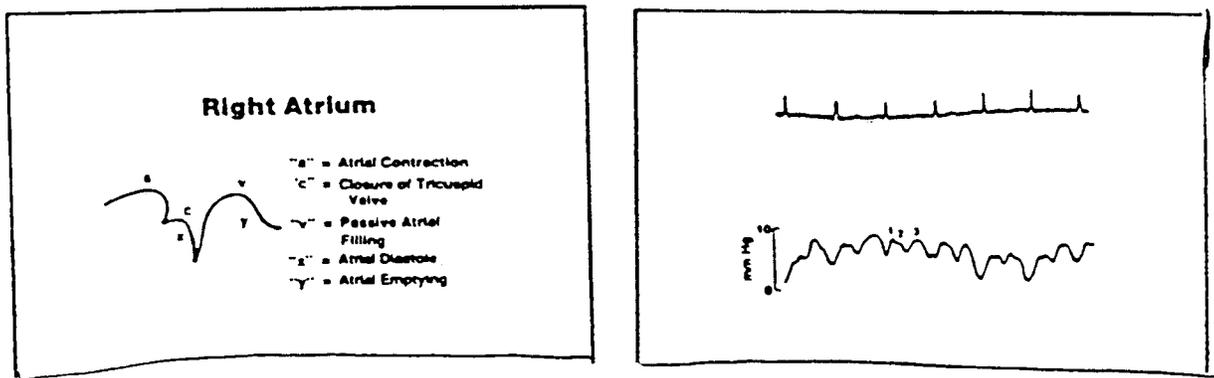
(2) If the patient is receiving positive pressure ventilation, the CVP may be falsely elevated

(3) If the patient has hemopneumothoraces; cardiac tamponade, or abdominal distention, CVP will not accurately reflect fluid status

(4) If integrity or patency of system is compromised, CVP measurements will not be accurate

c. Pressures and waveforms

- (1) Normal 2-6 mm Hg measured on the mean
- (2) Phases and correlation of mechanical events; three positive waves (a, c, v) followed by three negative waves (x, x', y)
  - (a) "A" waves corresponding atrial systole
  - (b) "C" waves - can be distinct, a notch on the "a' wave or absent, corresponds to the increase in pressure in the atrium caused by closure of the AV valve
  - (c) "X" descent - decrease in pressure corresponding to atrial relaxation
  - (d) "X1" descent - negative wave following "c" wave corresponding to downward pulling of ventricular septum during systole
  - (e) "V" wave - increase in atrial pressure caused by RA filling and RV systole which causes leaflets of AV valve to bulge backward into RA
  - (f) "Y" descent - follows "v" wave; is produced by opening of AV valve and emptying of atrium into ventricle



- (3) Abnormal pressures
  - (a) Increased
    - 1) RV failure

- 2) Tricuspid stenosis and regurgitation
- 3) Cardiac tamponade
- 4) Constrictive pericarditis
- 5) Pulmonary hypertension - primary and secondary
- 6) Chronic LV failure
- 7) Volume overload

(b) Decreased

- 1) Hypovolemia
- 2) Distributive shock

d. Complications

- (1) Related to placement - pneumothorax
- (2) Related to maintenance - infection

3. Pulmonary arterial monitoring

