Understanding Adult Hemodynamics

A Primer for:
Cardiovascular Surgery and
Hemodynamic Monitoring

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Understanding Adult Hemodynamics: A Primer for Cardiovascular Surgery and Hemodynamic Monitoring

Introduction/Purpose Statement

Aristotle started investigating it in the 4th century B.C.; Galen in the 2nd century A.D. continued to investigate it. The heart, blood vessels, and concepts of hemodynamics were intriguing to Harvey, Malpighi and van Leeuwenhoek in the 1600's. So what exactly is hemodynamics? **Heme** means "blood", and **dynamus** means "movement," so hemodynamic means the movement of blood.

We care about the movement of blood, and monitor it, because how the blood moves through the body will determine how the tissues are replenished with oxygen and nutrients and are able to excrete end-products of metabolism.

The purpose of this home study program is to give a brief introduction to hemodynamic monitoring - how we do it, what the numbers mean, and how we can optimize the movement of blood in the body. You’ll also learn about a variety of pharmacologic strategies that are used to improve cardiac output.

- **CV Surgery Class**
  All patients undergoing cardiovascular surgery will have some sort of hemodynamic monitoring. If you are unfamiliar with hemodynamic monitoring, you should read this primer to be able to understand content presented in the CV Surgery class.

- **Hemodynamic Monitoring Class**
  This primer was developed to give you a starting point in learning how to manage patients with hemodynamic monitoring. This primer can be used as either a stand-alone educational activity or as an introduction to the "Hemodynamic Monitoring" class.

Target Audience

This home study was designed for the novice critical care or telemetry nurse; however, other health care professionals are invited to complete this packet.

Content Objectives

1. Identify non-invasive indicators of hemodynamic status.
2. List three indications for invasive hemodynamic monitoring.
3. Describe the relationships among preload, contractility, compliance, afterload, and cardiac output.
4. Describe pharmacologic strategies that manipulate heart rate, preload, contractility, and afterload to improve cardiac output.

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**Contact Hour Information**

<table>
<thead>
<tr>
<th>For completing this <strong>Home Study and evaluation</strong>, you are eligible to receive:</th>
<th>2.0 MN Board of Nursing contact hours / 1.66 ANCC contact hours</th>
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The Concepts of Hemodynamics

The end-all and be-all of hemodynamic monitoring is the cardiac output. The cardiac output (CO) is the amount of blood ejected from the ventricle in one minute. This amount of blood is adequate to supply the body tissues with oxygenated blood.

Normally, the cardiac output is between 4-8 liters of blood every minute. Imagine an organ the size of your fist pumping out 2-4 Coke bottles of blood every minute!

Two components multiply to make the cardiac output: the heart rate and the stroke volume.

\[ CO = SV \times HR \]

Of course, different sized folks need different amounts of blood circulating. An 80-pound little old lady needs less blood than a 350-pound linebacker, right? To even things out a little bit, there is a calculation called the "cardiac index."

The cardiac index (CI) is the cardiac output adjusted for body surface area. It should be between 2.5 - 4.2 liters of blood per minute per square meter of surface area.

Heart rate

The first component of the cardiac output is the heart rate. The heart rate and stroke volume should work like a teeter-totter. If one goes up, the other should go down, and vice versa. This is the concept of the compensatory heart rate.

The most common change in the heart rate to compensate is for it to go faster (become tachycardic) because of low stroke volume or increased tissue oxygen needs.

Causes of compensatory tachycardia are:

- Hypovolemia from dehydration, bleeding, loss of fluid
- Low blood pressure
- Anxiety, fear, pain, and anger cause the sympathetic nervous system to release endogenous and exogenous catecholamines
- Fever
- Exercise

There are limitations to the compensation that tachycardia can provide: heart rates above 180 beats/min in a normal heart, or above 120 in a diseased heart, are too fast to compensate. If the stroke volume continues to decline, the heart rate can only increase so much to balance cardiac output.

On the other hand, the heart can go more slowly (become bradycardic) to compensate for a high cardiac output or high blood pressure. This can be seen with seasoned athletes with "strong pumps," who often have heart rates in the 40's-60's at rest.

Beyond the compensatory tachy- or brady-cardias, there are those rhythms that hurt the hemodynamic state of the patient.

Sinus tachycardias that are > 180 in the normal heart or > 120 in the diseased heart are not compensatory anymore because the heart can't fill adequately with blood to pump out. Other dysrhythmias have the same problem, but an additional one: they lose 20% of their cardiac output because their atria are not contracting in sync with the ventricles. These rhythms are:

- Atrial tachycardia
- Uncontrolled atrial flutter/atrial fibrillation
- Junctional tachycardia
- SVT
- Ventricular tachycardia

Bradycardias that present problems to the hemodynamic standing of the patient are:

- Junctional rhythm
- 2nd degree AV block, type II
- 3rd degree AV block
- Idioventricular rhythm

What can cause these kinds of bradycardias? The most common causes are:

- Myocardial infarction
• Vagal stimulation (bearing down)
• Beta blocking and calcium channel blocking agents

Stroke volume
The stroke volume is the amount of blood ejected with each ventricular contraction. Kinda makes sense, doesn't it? The amount of blood per beat X the number of beats in a minute.

Three main factors determine stroke volume: **contractility, preload, and afterload.**

Contractility
Contractility is the force and velocity with which ventricular ejection occurs, independent of the effects of preload and afterload. Huh? Think of contractility as the "squeeze."

Contractility increases (the heart squeezes harder) from:
• The fight or flight response from fear, anxiety, stress, pain, hypovolemia
• Exercise

The bad thing about increased contractility is that although it increases stroke volume, it will also increase the demand of oxygen by the heart (MVO₂). This can be hazardous in someone with heart disease. The prime example: the guy who has the heart attack while shoveling snow -- all the exercise increased his heart rate and his contractility and his heart couldn't handle the extra work.

Decreased contractility decreases stroke volume and MVO₂. The causes might be:
• hypoxia
• hypercapnia
• metabolic acidosis
• hyperkalemia
• hypocalcemia
• myocardial infarction
• cardiac surgery

Preload
Preload is the amount of blood in a ventricle before it contracts. It's the "gas in the tank." Preload is also known as "filling pressures."

Preload is determined by:
1. The total circulating blood volume: how much blood is actually in the blood vessels?
2. The distribution of vascular volume: where is the blood and fluid? In the blood vessels, in the cells, or in the "3rd space?"
3. Atrial systole: are the atria contracting in sync with the ventricles? If they are not, there is a decrease in preload by 20%.

There is a theory that helps to explain how preload and contractility are related: the Frank Starling Law. This is what it says --

Imagine that you have a rubber band in your hands that you are stretching. If you stretch out the rubber band out about three inches, it will contract back pretty well.

Now imagine that you stretch out your rubber band just an inch or so. What will happen now? It won't contract back very fast or with very much force. This is what happens when a person becomes hypovolemic - they don't have a lot of stretch, so they don't have a lot of squeeze.

Next, if you stretch out your rubber band six-eight inches, it will contract back more strongly and faster. This is the principle behind a fluid challenge or fluid flush. If you give someone a little fluid to stretch their heart, they should squeeze back harder.

Please return to previous page to purchase this home study program.