Principles of Invasive Hemodynamics

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Purpose

The purpose of this course is to inform the healthcare provider about the fundamentals of hemodynamic monitoring and the devices used to evaluate these vital functions.

Learning Objectives

After successful completion of this course, you will be able to:

1. Discuss the indications for invasive hemodynamic monitoring.
2. Delineate hemodynamic values for pulmonary artery catheter, arterial line, and central venous pressure monitoring.
3. Describe three steps to ensure waveform accuracy.
4. Compare preload, afterload, and contractility when determining cardiac function.

Introduction

Hemodynamics is a term to describe intravascular pressure, oxygenation, and blood flow occurring within the cardiovascular system.

Hemodynamic monitoring is an essential part of critical care nursing. The primary goals of hemodynamic monitoring are to:

- Maintain adequate tissue perfusion by assessing the body's response to tissue oxygen demands
- Alert the healthcare team of an impending cardiovascular crisis before organ injury occur
- Evaluate immediate response to treatment modalities

Because organ blood flow cannot be directly measured; non-invasive and invasive mechanical methods must be used. These non-invasive and invasive methods include manual blood pressure, arterial blood pressure, central venous pressure, left atrial pressure, pulmonary wedge pressure, SVO^2, and cardiac output measurement. Using non-invasive and invasive methods provide quantitative information about vascular capacity blood volume pump effectiveness and tissue perfusion.

Indications:

The indications for hemodynamic monitoring include:

- All shock states (cardiogenic, neurogenic, anaphylactic, septic, and hypovolemic)
- Loss of cardiac function
- Decreased cardiac output

Hemodynamic Concepts:

The critical care nurse should recognize and understand the building blocks of hemodynamics. These building blocks include:

- Heart rate
- Stroke volume
- Cardiac output
- Blood pressure/Systemic vascular resistance/Pulmonary vascular resistance
- Central venous pressure/Pulmonary wedge pressure
- Determinants of stroke volume
    - Preload
    - Afterload
    - Contractility
- Mixed venous oxygen saturation (SVO_2)

Cardiac output (CO)

Cardiac output:
Cardiac output is the amount of blood that is pumped out of the ventricles in one minute. A normal cardiac output is between 4-8 L/minute. To determine cardiac output multiply heart rate X stroke volume. There is a direct relationship between cardiac output, heart rate, and

stroke volume; therefore, any change in heart rate and/or stroke volume can affect cardiac output.

Cardiac index:
Cardiac index (CI) is a hemodynamic measure that relates the cardiac output (CO) to body surface area (BSA). This relates heart performance to the size of the individual.

The cardiac index (CI) is usually calculated using the following formula:

$$CI = \frac{Cardiac\ Output\ (CO)}{Body\ Surface\ Area\ (BSA)} = \frac{Stroke\ Volume\ (SV) \times Heart\ Rate\ (HR)}{Body\ Surface\ Area\ (BSA)}$$

The unit of measurement is liters per minute per square meter (L/min/m2). The normal range of cardiac index (CI) at rest is 2.6-4.2 L/min per square meter.

Note! If the CI falls below 1.8 L/min/m2, the patient may be in cardiogenic shock.

Heart rate:
Not only is the rate important, the rhythm can cause hemodynamic imbalances. When the rhythm does not originate from the sinus node or the rate is too slow or too fast hemodynamics can be altered. For example, if a patient is in atrial fibrillation, there is a loss of atrial kick and heart rate variability that needs to be taken into consideration.

Stroke volume:
Stroke volume (SV) is the amount of blood pumped out of the ventricle with each contraction. A normal stroke volume is between 60 – 100 mL/beat. This volume can be altered when the heart rate is too fast or the rhythm is too irregular to allow complete filling of the ventricles. Additionally, stroke volume can be abnormal due to the cardiac muscle mass.

Stroke volume components (SV)

Preload:
Preload is the volume required to STRETCH the cardiac muscle fibers in the atria and ventricles. The cardiac muscle stretches to accommodate the fluid in the chambers. Preload can also be considered filling pressures of the ventricles at end-diastole. Preload is measured as central venous pressure (CVP) for the right side and pulmonary artery wedge pressure (PAWP) for the left side of the heart. Normal CVP is 2-8 mm/Hg and PAWP is 4–12 mm/Hg. Factors that influence preload are:

Increases Preload	Decreases Preload
Increased circulating volume	Decrease circulating volume
Mitral valve insufficiency	Mitral valve stenosis
Tap vasoconstrictor use	Vasodilator use
Heart failure	Asynchrony of atria and ventricles
Aortic insufficiency	

Contractility:
Contractility is the strength of the cardiac muscle to push blood from the ventricles against the systemic vascular resistance/blood pressure of the patient. Any condition affecting the strength of the cardiac muscle, hypertrophy, will affect stroke volume. Factors that influence contractility are:

	Increases (SQUEEZE) Contractility	Decreases (RELAX) Contractility
Medications	Positive inotropes (digoxin, dopamine, dobutamine)	Negative inotropes (beta-blockers, calcium channel blockers, lidocaine, barbiturates)
Electrolytes	Hypercalcemia (calcium stimulates the heart to contract) Hyperglycemia	Decreases magnesium, sodium, calcium, increases potassium
O_2 & CO_2		Hypoxemia, acidosis, hypercapnia
Myocardial infarction/cardiomyopathy		"Stunned" heart, scar tissue, hypertrophied muscle
HR & Rhythm	Increased HR	Sustained tachycardia leads to decompensation
Autonomic nervous system	Sympathetic stimulation	Parasympathetic stimulation Vagal stimulation

Afterload:
Afterload is the RESISTANCE or pressure the ventricular heart muscle must overcome to open aortic valve and eject volume. Afterload is also known as systemic vascular resistance (SVR); and pulmonary vascular resistance (PVR), the resistance of blood flow through the pulmonary circulation. A normal SVR is 800 – 1200 dynes/sec/cm. A normal PVR is 100-250 dynes/sec/cm. An easy way to estimate increased or decreased afterload is by measuring blood pressure. Hypertension indicates increased SVR and hypotension indicates decreased SVR. Factors that increase afterload are:

Increases Afterload (increases vascular and pulmonary resistance)	**Decreases Afterload** (decreases vascular and pulmonary resistance)
Increased circulating volume	Decreased circulating volume
High blood pressure/high systemic vascular resistance	Septic shock – warm phase
Aortic stenosis	Aortic insufficiency
Myocardial infarction/cardiomyopathy	Decreased cardiac output
Polycythemia – increase blood viscosity	End-stage cirrhosis
Vasopressor use	Vasodilator use

Mixed venous oxygen saturation (SvO_2):
Mixed venous oxygen saturation is the measurement of oxygen delivery and oxygen consumption. Oxygen demand is the amount of oxygen the cells and tissues require for their own metabolism. Factors that influence SvO_2 are:

Increases SvO_2 (decreases body's demand for oxygen)	**Decreases SvO_2** (increases body's demand for oxygen)
Anesthesia	Cardiogenic shock
Chemical paralysis	Septic shock
Increased oxygen saturations	Decreased cardiac output
Hypothermia	Decreased hemoglobin
Increased cardiac output	Fever
Increased hemoglobin	Seizures
Sedation	Shivering

To better understand SvO_2, let's trace a red blood cell, called Ruby. As Ruby leaves the body through the left ventricle, it goes into the aorta with an oxygen saturation of 96-100%. She branches out with her other red blood cell family into other arteries where they deliver oxygen to the tissues and cells who need it. Then it continues to travel to the arterioles, capillaries, then venules and veins. It finally ends back up into the vena cava, right atrium, right ventricle, and then the pulmonary artery, where before it goes into pulmonary circulation we can take a continuous reading of how much oxygen the arteries used. The normal values of SvO_2 are between 60-80%.

Frank-Starling Law:
The Frank-Starling Law states, "*The greater the stretch, the greater force of the next contraction*" (Hodges, 2005).

To better understand the Frank Starling Law, let's use the example of the rubber band being the heart's muscle. The less the rubber band is stretched the less it will propel in the air. The further the rubber band is stretched the greater the force and it will propel in the air further.

As this relates to preload, if there is too little preload (stretch) as in hypovolemia, the heart will not have enough stretch to propel the blood (cardiac output) through the body. If volume increases, the more preload (stretch) the heart will have and will contract with more force.

In the same manner if the rubber band is continually, excessively stretched over time it will lose it elasticity. The heart muscle will wear out and lead to heart failure.

Invasive Hemodynamic Monitoring Devices

The three most common monitoring devices are:

- Arterial lines
- Central venous lines
- Swan-Ganz lines/PA lines

Each of these devices require a pressurized fluid system, transducer, monitoring cable, monitor, confirm and leveling device. To ensure the accuracy of these devices, the critical care nurse must that the fluid source has an adequate amount of fluid, the tubing is flushed and no bubbles remain in the tubing, all connections are tight, and the transducer has been leveled, zeroed, and square wave tested at the beginning of each shift.

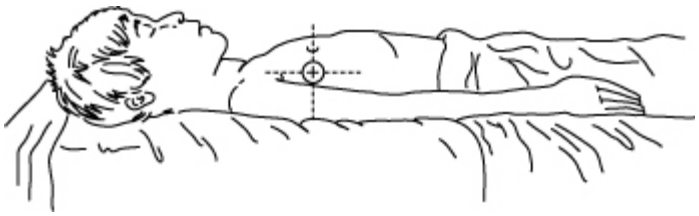
Note: be sure to know your hospital's policy regarding the type of fluid, heparinized or heparin free, used to flush the hemodynamic monitoring devices.

Hemodynamic Accuracy

There are three steps to hemodynamic accuracy. These are, leveling, zeroing, and the square wave test. These three methods should be utilized every shift with all hemodynamic monitoring devices.

Leveling
The phlebostatic axis is the anatomical area which the nurse should LEVEL to the stopcock. The phlebostatic axis is located at the 4th intercostal space, mid-axillary line. This is the location of the right atrium. Best practice is to always use a leveling device. DO NOT 'eye-ball' the phlebostatic axis when leveling; for every 10 cm above or below the phlebostatic axis 7 .4 mmHg will be added or subtracted to the pressure (Deranged Physiology, 2015a).

If the head of the bed is increased or decreased this will change the location of the phlebostatic axis, so re-level and zero for accuracy.



Zeroing
Zeroing is defined as the use of atmospherics pressure as a reference standard against which all other pressures are measured or a process which confirms that atmospheric pressure results in a zero reading by the measurement system. This ensures the pressure values we see on the monitor are only those values which are reflected in the vessel or heart.

After leveling the transducer to the phlebostatic axis, then turn the stop-cock off to the patient and remove the cap. Be certain not to contaminate the cap. Press the 'zero' button on the monitor and wait for the number '0' to appear. Replace the cap and return the stop-cock to its original position.
(Deranged Physiology, 2015a)

Note: Be sure to check the manufacturer's recommendations regarding removing the cap during zeroing. Changes in the manufacturing of transducers have made cap removal unnecessary with some devices.

Hemodynamic Accuracy

Square Wave Testing
Square wave test is done by fast-flushing the system for 1-2 seconds and noting the waveform or the 'square wave' producing a waveform that rises sharply, plateaus, and drops off sharply, and the oscillations that immediately follow the square wave.

There are three types of square waves; optimally damped system, over-dampened system, and under-dampened system:

1. Optimally dampened system correlates with a properly working system and does not require any interventions.
    a. When looking at the waveform on the monitor the nurse should see one or two oscillation waves after the square wave.
2. Over-dampened waveform is the most common and this may be caused from not enough pressure in the pressure bag, flush bag is empty, blood clots or air bubbles in the non-compliant tubing.

    a. When looking at the waveform on the monitor the nurse should see only one oscillation wave and then a flat line after the square wave.
3. Under-dampened waveform will overestimate the systolic pressure and underestimate the diastolic pressure; however, the mean arterial pressure will remain accurate.
    a. When looking at the waveform on the monitor the nurse should see multiple oscillation waveforms after the square wave.

(Deranged Physiology, 2015b)

For more information and images regarding square wave testing go to the following website: http://www.derangedphysiology.com/main/core-topics-intensive-care/haemodynamic-monitoring/Chapter%201.1.4/arterial-line-dynamic-response-testing

Arterial Line:

The arterial line is used to monitor systemic blood pressure/systemic vascular resistance. The radial artery is most commonly used for arterial line placement. However, other arteries such as the brachial, axillary, or femoral may be used depending on patient condition and physician's preference. This line should be used for monitoring only. Infusion of fluids other than the Flushing fluid, should not be done.

When inserting a radial arterial line, an Allen's Test should be performed to ensure there is blood supply to the hand if the radial artery becomes occluded.

Allen's Test Procedure

1. Raise the patient's hand and ask them to make a fist. Occlude both radial and ulnar arteries by applying pressure with your fingers.
2. Instruct the patient to clench their fist a few times and take note of blanching or paleness that will occur.
3. Release pressure from the ulnar artery, while maintaining pressure on the radial artery. If the ulnar artery is patent, you will see the hand color return (then release pressure from the radial artery).
4. If it takes more than 5-10 seconds for color to return after you have released pressure from the ulnar artery, the Allen's Test is considered negative and that radial artery should not be used.

(Segen's Medical Dictionary, 2012)

Central Venous and Pulmonary Artery Catheters

The CVP and PA catheters are essential to measure preload and other cardiac functions. Both lines are placed into a central vein, internal jugular, subclavian, or femoral locations. The CVP line is most commonly used and measures right atrial pressures or preload. The PA catheter is utilized when more information is required; such as left heart preload and cardiac output. Besides monitoring hemodynamic parameters these lines may be used for fluid and medication infusions. Radiographic confirmation of catheter tip placement should be done after insertion

of these lines. It is important to know your facility's policy on informed consent for these lines. In some institutions consent for these lines is implied on the admission consent for treatment.

Because the PA line is not utilized as frequently as the CVP or arterial line this module will go into more detail about the PA line.

Pulmonary artery catheter:
PA catheters are indicated for patients with cardiogenic shock or congestive heart failure; in the presence of respiratory failure to assist in improving oxygenation delivery; multiorgan failure, and when vasoactive medications and fluid management are essential for positive outcomes.

There are various types of PA catheters available to the practitioner. These catheters may have ports for temporary pacing, continuous cardiac output monitoring, and mixed venous oxygen saturation monitoring. The type of catheter used is based on patient condition and physician preference (www.edwards.com, n.d.).

The most common type is a catheter that has four lumens.

- The proximal port is for monitoring CVP/right atrial pressures (injectate port for cardiac output).
- The PA distal port is used to monitor PA pressures (systolic, mean, and diastolic).
- The balloon inflation port is used for inflating and deflating the balloon during insertion as well as when the PCWP is being measured. The balloon maximum inflation is 1.5 mL of air, though at times less air is needed to gain the desired result. ONLY use the syringe the manufacturer included in the kit and NEVER fill this balloon with fluids.
- The thermistor connector is used for connecting it to the monitor cable for measurement of cardiac output/cardiac index and blood temperatures. It assists in measuring the change in blood temperature.
- If other ports exist then these are used for additional monitoring, fluid infusion, or for inserting a pacemaker electrode.

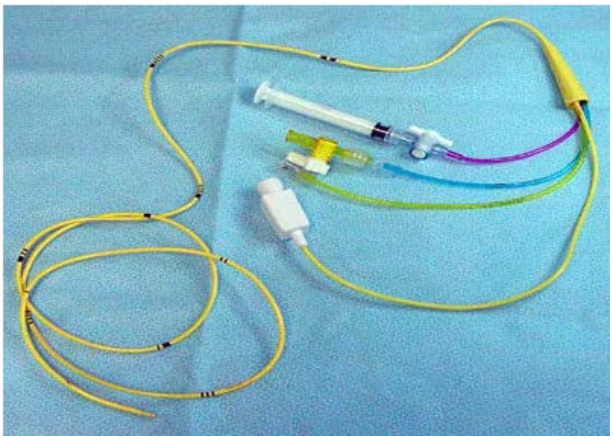
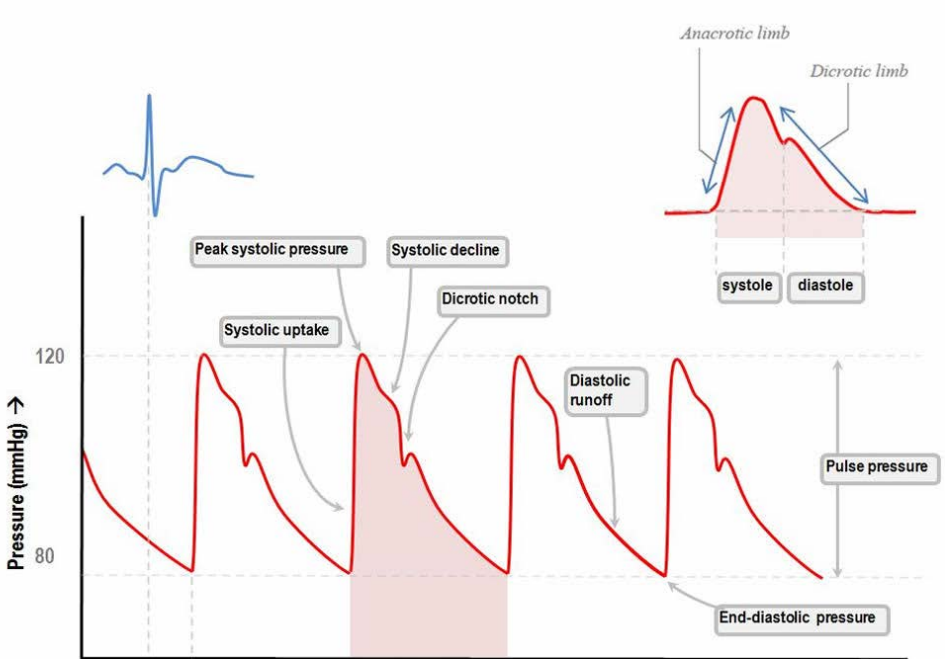


Image courtesy of Federal Drug Administration, 2013

Know and follow your institution's procedure for insertion, maintenance, and discontinuation of a PA catheter.

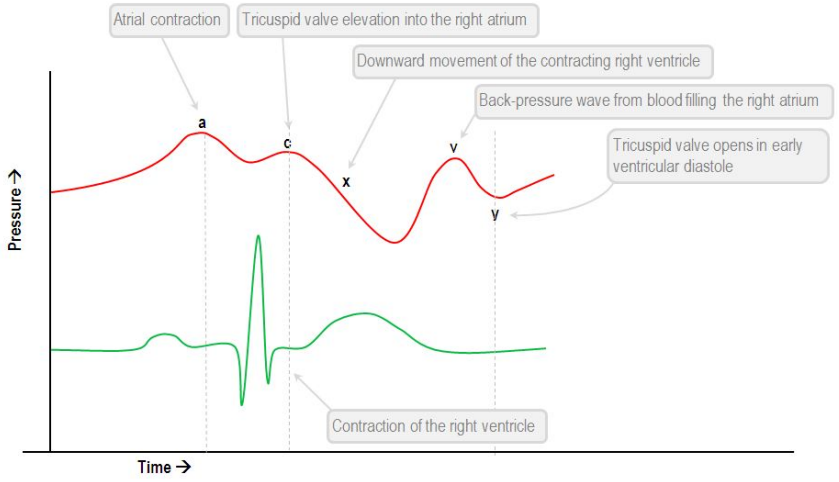
Waveforms

Arterial Line



Deranged physiology, 2015c

CVP



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PA

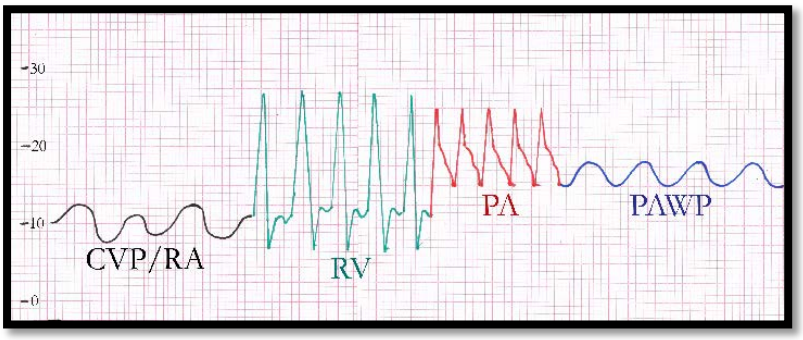

Image courtesy of Federal Drug Administration, 2013

Case Study: Introduction

A 76-year-old was admitted to the medical floor with two days of nausea and vomiting. He has a history of diabetes, myocardial infarction five years ago, with a drug-eluting stent placed in his right coronary artery (RCA). At that time, he had an ejection fraction (EF) of 40%.

On admission is vital signs were:

Day 1	1400
BP	100/60
HR	124
RR	22
SaO2	93%

- Physician ordered 0.9% NS to infuse at 125 mL/hour x 12 hours, then a maintenance infusion at 75 mL/hour
- Labs were drawn
- CXR was completed
- Cardiac telemetry shows sinus tachycardia
- All home medications were held
- Ondansetron (Zofran) 4mg IV every six hours was ordered
- Patient remains NPO
- No supplemental oxygen is ordered

Day 2	0200
BP	88/52
HR	130
RR	28
SaO_2	92%

12 hours after admission the patient's blood pressure was dropping, heart rate was elevated, and as saturations had dropped. You review the patient's labs and find the following significant values.

- White count: 14,000, no left shift
- Potassium: 5.2
- BUN/creatinine: 22/2

Based on these findings you anticipate the physician will order which of the following:

1. Oxygen
2. Fluid bolus
3. Vasoconstrictors

If you selected oxygen and fluid bolus you are correct. However, to know if vasoconstrictors are necessary what else might you want to know?

Urine output, respiratory status, cardiac output, and/or peripheral perfusion? A thorough physical assessment will help ensure the practitioner has all the information needed to order the appropriate treatment.

Your assessment shows that the patient is not in respiratory distress, his prior 12-hour intake and output was 1500 mL/50mL, and his extremities are cool to the touch with weak pulses. The practitioner orders a 500 mL 0.9% NS bolus and to continue the fluids at 125 mL/hour after the bolus is completed.

An hour after the bolus was infused the patient's vital signs and assessment have not improved. You notify the practitioner and receive orders for another 500mL 0.9% NS bolus. The bolus is infused and at 0500 his vital signs are as follows:

Day 2	0400	0500
BP	90/48	120/82
HR	140	140
RR	24	24
SaO2	92%	90%

What are your concerns?

Although the blood pressure has improved, the patient's heart rate remains elevated and the saturations continue to fall.

The practitioner makes rounds at 0630. You inform the practitioner that in addition to the above findings the patient is complaining of shortness of breath. On auscultation, the physician hears crackles bilaterally. Due to a history of a prior MI and an EF of 40%, the patient was transferred to the cardiac intensive care unit for additional monitoring. In addition, 12-Lead ECG, ECHO and cardiac enzymes were ordered.

A central line was placed in the right internal jugular (IJ) and can arterial line is inserted in the right radial artery. What steps would you take to ensure the accuracy of the readings?

- Tubing and transducer flushed without bubbles in tubing
- Transducer leveled at the phlebostatic axis
- Transducer and monitor zeroed
- Square wave form test completed

The initial readings of the CVP were 18-20. His blood pressure is 110/70. An echocardiogram was completed and showed a decrease in ejection fraction to 20%. The 12-Lead showed some ischemia in the anterior leads, which correlates to the coronary arteries which supply blood to the left ventricle.

What do you think is happening and what is the practitioner likely to order?

The patient appears to be fluid overloaded/elevated preload and the heart's ability to move the fluid efficiently is decreased. Because the patient is having respiratory difficulties and on auscultation rales are heard in the bases of the lungs; you anticipate diuretics.

The practitioner orders furosemide 40 mg IV x 1, keep NPO, and decrease the IV fluids to 25 mL/hour.

It is now 1400 on day two of his hospitalization. The 40 mg of furosemide was administered at 0800 and the patient voided 400 mL. His vital signs are:

Day 2	400
BP	100/52
HR	120
RR	26
SaO2	88%

The practitioner orders arterial blood gases every four hours as well as hourly vital signs and assessments; to be called if the mean arterial pressure (MAP) falls below 70 mmHg.

Case Study: Cardiologist Examination

The patient was seen by the cardiologist and because of his ischemia and history of an MI and stent placement five years ago, he will take the patient to the cardiac catheterization lab for a right and left heart catheterization. The patient had a PA catheter inserted for additional monitoring.

Let's continue to learn about the hemodynamics to better interpret what the numbers mean clinically.

	Normal
CO	4-8
CI	2.6-4.2
CVP	6-10
PCWP	4-12
PVR	100-200
SVR	800-1200
HR	60-100

Test Yourself

Day 2	2100
CO	3.4
CI	1.7
CVP	16
PCWP	16
PVR	93
SVR	1800
HR	80

Using the reference table above, why would the SVR & PCWP still be high and CO/CI low?

a. Effects of cardiogenic shock
b. Hypothermia
c. Blood loss from CABG

If you selected cardiogenic shock you are correct. Signs of cardiac shock include an elevated SVR and PCWP with a low CO/CI. Remember, the gold standard value for detecting cardiogenic shock is a CI of 1.7 or less.

Case Study: Dobutamine and Nitroglycerin Drips

The practitioner orders a dobutamine drip as well as a nitroglycerin drip. He explains that the dobutamine drip will increase the CO/CI and the nitroglycerin drip will decrease the SVR and PCWP (afterload) of the left ventricle. Some effects of the dobutamine drip are increased oxygen consumption and potential for dysrhythmias. Nitroglycerin drip may decrease the blood pressure; so slow titration is warranted.

Look at the hemodynamics two hours after the start of the dobutamine and nitroglycerine drips.

BEFORE			AFTER	
Day 2	**2100**		**Day 2**	**2300**
CO	3.4		**CO**	5.2
CI	1.7		**CI**	2.6
CVP	16		**CVP**	10
PCWP	16		**PCWP**	12
PVR	285		**PVR**	185
SVR	1800		**SVR**	1000
HR	80		**HR**	84

As you can see the dobutamine increased the cardiac output/index and the nitroglycerine decreased the PCWP and CVP to normal values. The patient continued to progress through the night; the dobutamine and nitroglycerine drips were weaned off and the PA catheter was discontinued.

Day 2	2100
CO	5.2
CI	2.6
CVP	10
PCWP	8
PVR	128
SVR	1000
HR	84

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