

Hemodynamics

Hemodynamics: The study of forces involved in blood circulation. It is used to assess cardiovascular function in critically ill or unstable clients. The goal of using hemodynamics is to evaluate cardiac and circulatory function as well as evaluate response to interventions.

Hemodynamic Parameters

- Heart rate
- Arterial blood pressure
- Central venous (right atrial) pressure (normal is 2-6 mm Hg)
- Pulmonary pressures (PA = 25/10 and PAWP = 4-12 mm Hg)
- Cardiac output (normal is 4-8 L/min)
- Assess preload (preload = pressure or stretch exerted on the walls of the ventricle by the volume of blood filing the ventricle at the END OF DIASTOLE.) End diastolic pressure tells us how well the pump is working!
- Assess afterload (afterload = the resistance to ventricular contraction. the pressure the ventricle has to overcome to open the aortic or pulmonic valve and push blood out of the ventricle.)
- Systemic vascular resistance = an estimate of afterload

How does it work?

Monitoring systems measure the pressure within a vessel/chamber. It converts this signal into an electrical waveform. The waveform is amplified and displayed on a monitor. It may be graphically recorded or displayed digitally, and I'm pretty sure the computer spits out a number of some kind.

NOTE: The graphic to the right shows an arterial line. You do not ever ever ever put any meds, blood or fluid into this line. It is only used to measure pressures. If air gets into this line, it is a BIG problem, so don't do it! Note that the tubing is very stiff vs the pliable tubing we use with fluid lines. The tubing receives a "bump" with each pulse of pressure → goes to the transducer → sends signal through an electrical wire → waveform on the monitor → pressure # displayed.

Intra-arterial Pressure Monitoring

This is very commonly used and is put in almost like an IV (doc has to do it though). It allows for direct and continuous monitoring of systolic, diastolic and mean arterial pressure...it is MUCH more accurate than a BP cuff. It is used to monitor the effects of vasoactive drugs...you have to watch for instant changes because these drugs cause changes very quickly. It can be used to obtain frequent samples for things like ABGs, cardiac markers, etc...

What Does an Arterial Line Tell Us?

- Accurate blood pressure
- Mean arterial pressure
- Reflects cardiac output...not the entire picture, but part of it
- Reflects systemic vascular resistance (SVR)
- Systolic pressure reflects pressure during ventricular contraction
- Diastolic reflects the arterial elasticity and the ability to maintain blood flow through capillaries.

What the Heck is is MAP Anyway?

Mean Arterial Pressure is the average pressure in the arterial circulation throughout the cardiac cycle. It reflects driving pressure or perfusion pressure. It is an indicator of tissue perfusion. You calculate MAP like this: $MAP = CO \times SVR$...or you can do it like this: $MAP = DBP + (SBP - DBP/3)$. A good MAP to shoot for is 65-80-ish or 70-90.. If MAP gets above 90 you start worrying about things like vasoconstriction and workload. If MAP gets too low, then you worry about tissue perfusion.

Central Venous Pressure (aka Right Atrial Pressure)

A Right Atrial line will tell us about volume and venous return. It is primarily used to measure fluid volume status. A catheter is inserted into the subclavian or internal jugular vein. It is positioned in the superior vena cava or just inside the RA. CVP can be measured in Cm of H₂O (older method, uses a manometer attached to a stopcock) and mm Hg (uses a computer).

Again, this system uses a pressure tubing that sends a signal through the transducer to a monitor which displays a waveform and reading. The dressings used for this are very specific and be aware that the line may not be sutured in place, so you will want to coil the line to prevent it from becoming dislodged if it gets tugged.

CVP Readings = 2-8 cm H₂O, or 2-6 mm Hg (can vary quite a bit in some pts)
 Low CVP = hypovolemia, shock states (GIVE FLUID)
 High CVP = vasoconstriction, pulmonary hypertension, right sided heart failure, raised intrathoracic pressure, fluid overload, cardiac tamponade (cardiac tamponade can happen quickly, just 70-80 ml will impact the heart and keep it from contracting adequately)..(DO NOT GIVE FLUID!)

What Do We Use CVP For?

Mostly, it is used to adjust fluid volume replacement. It is also useful to determine if patient needs vasopressor or fluids, or the right balance of both. So...if your pt's HR is 130, BP is 80/40, low MAP, CVP is 2 mm Hg...you will give FLUIDS (this is a volume problem) You do not want to give vasopressors to a pt who is hypovolemic...they cause vasoconstriction and since the heart arteries fill on diastole they won't get enough O₂. The heart will suffocate :-)

Nursing Decisions: Pt has a BP of 82/54, MAP 90, HR 130, CVP 12. What else do you want to assess?

- Urine output (very very important)
- Listen to lungs (if clear, it's just right-sided HF...if wet, it's left-sided HF or both)
- Peripheral circulation and skin signs
- Decide...is this a rate, pump or volume problem? This is a PUMP problem...we know this b/c it's not a volume problem (his CVP tells us this), and it's not a rate problem (the HR of 130 tells us this)...so give Lasix and Vasopressors.

Pulmonary Artery Pressure Monitoring

This is what you turn to if you need even more info...commonly called the Swan Ganz line. It utilizes a flow-directed balloon tipped catheter. It is used to evaluate left ventricular and overall cardiac function. You can also sample mixed venous blood, how cool is that? Using this line, we can evaluate a number of pressures: right atrium, right ventricle, pulmonary artery, left ventricle. Each of the four lumens exits into the heart or pulmonary artery at a different point, graduated along the catheter length.

- The Right Atrial Lumen (proximal lumen..BLUE): situated in right atrium and used for IV infusions, CVP measurement, withdrawal of venous blood samples, and injection of fluid for cardiac output determinants. This port is often described as the Right Atrial Port or the CVP Port.
- The Pulmonary Artery Lumen (distal lumen...YELLOW): located at the tip of the PA catheter and is situated in the pulmonary artery. It is used to record pulmonary artery pressures, withdrawal of blood samples and measurement of mixed venous gases. NEVER PUT ANYTHING IN THIS PORT!
- Balloon Lumen (RED): opens into a balloon at the end of the catheter. The balloon is inflated during catheter insertion after the catheter reaches the right atrium to assist in the forward flow of the catheter and to minimize right ventricular ectopy (abnormal heart beat) from the catheter tip. The balloon is also inflated to obtain the PAWP measurements when the PA catheter is correctly positioned in the pulmonary artery (wedged in place)
- Thermistor Lumen: This is a temperature sensor used to measure changes in blood temp.
- Additional Lumens: If continuous SVO₂ is needed, there is an additional lumen that exits at the tip of the catheter. You can also use catheters for cardiac pacing.

These catheters are placed via the SCV (most common), jugular vein, and femoral vein (last resort). Using one of these will give you the info you need to know if you have a rate, pump or volume problem. They better...they cost \$2500 - \$3000 apiece! Remind me to invent a medical device please.

Inserting the PA catheter

It is inserted into a central vein and threaded into the right atrium. The small balloon at the tip is inflated and the catheter is floated in with blood flow...it floats right on through the tricuspid and into the RV. It is then floated through the pulmonic valve and into the PA. The balloon can wedge the PA catheter into a smaller pulmonary artery and read transmitted

pressures from the LV...this is a PAWP, and your orders will indicate how often (usually 1x per shift). The whole process takes 30-45 minutes to complete.

What does a PA catheter tell us?

- We can assess LV function
- We can measure cardiac output
- We can calculate cardiac index
- Why do we care? This enables us to determine elevated PA pressure which tells us about heart failure. We can also determine contractility concerns.

Interesting Patho Tidbit!

CHF → Pulmonary Edema → Cardiogenic Shock. They are all part of the same process (pump failure), so basically CHF is a mild form of pulmonary edema, which is a moderate form of cardiogenic shock...no bueno!

NOTE: A high PAWP (> 20) indicates pulmonary edema. What is the Tx for that? Lasix to get the fluid off, Nitro to decrease workload, dobutamine to increase contractility. Normal PAWP is 4-12 mm Hg.

Wedge Pressure is increased:

- LV failure, pulmonary edema
- Pericardial tamponade
- Cardiogenic shock

Wedge Pressure is decreased:

- Hypovolemia
- Hemorrhagic shock

Nursing Care of Patients with Hemodynamic Monitoring

- Calibrate the system once per shift to maintain accurate readings
- Keep the transducer at the mid-axillary line
- Measure all pressures between breaths
- Maintain 300 mm Hg of pressure on the flush solution at all times
- Monitor pressure trends rather than individual readings
- Treat the pt instead of the monitor
- Obtain CXR before infusing IV fluids through central lines. Failure to do this means fluids could infiltrate and close off the airway.
- Set alarm limits carefully....never off!
- Use aseptic technique to prevent infection
- Assess and document appearance of insertion site every shift
- Change IV solutions q 24 hours
- Change dressing q 48 hours

- Change tubing q 72 hours
- Label solution, tubing and dressing with date
- Untangle and organize all lines at beginning of each shift (can take up to an hour)
- Thoroughly flush lines after drawing blood samples
- Assess pulse and perfusion distal to the monitoring line site
- Secure all connections and stopcocks
- Ensure that electrical equipment is grounded
- Loosely restrain affected extremity

- Keep tubing free of kinks and tension (be super careful when transferring, turning)

Complications of Pressure Lines

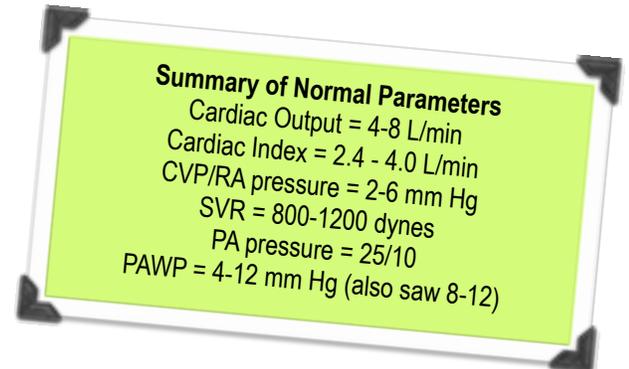
- Risk of infection, so use occlusive dressing or Biopatch

Normal pressures:
Pulmonary Pressure = 25/10
PAWP = 4-12, also saw 8-12 in same PPT

- Thrombosis/emboli...an air embolism may occur when the balloon ruptures, or a clot on the end of the catheter could cause an embolus
- Catheter wedges permanently...this is an emergency! Call the DOC! Can cause pulmonary infarction
- Ventricular irritation: occurs if catheter migrates back into the RV.
Catheter Fling/Catheter Whip can cause V-tach.

Educational Resources for Hemodynamic Monitoring

- Pulmonary artery catheter education: <http://www.pacep.org>
- American Association of Critical Care Nurses. Essentials of Critical Care Education (ECCO)



States of Shock

(altered tissue perfusion)

Concept of Shock

Shock is a complex set of signs and symptoms that is considered a syndrome. It is a sequence of changes d/t a disparity between oxygen supply and oxygen demand that is related to either a rate, pump or volume problem. The big picture is altered tissue perfusion.

Four Classifications of Shock

- Hypovolemic (d/t fluid volume loss, vasodilation)
- Transport (d/t loss of hgb, carbon monoxide poisoning)
- Obstructive (a mechanical barrier i.e. cardiac tamponade, tension pneumo)
- Cardiogenic shock (pump failure)

Stages of Shock

- Initial shock
- Compensatory shock
- Progressive shock
- Refractory shock (end stage, super bad news)

Clinical S/S of Shock (these are the general signs, each type has specifics)

- Usual signs used to assess shock include
 - BP (varies with age and health status of pt)
 - HR (can be masked by anxiety, pain or fever)
 - Mentation (can be altered by alcohol, head injury or drugs)
 - Urine output (neuro-endocrine responses)

Stage 1: Initial Shock

Initial shock may go unnoticed...you have to have a high index of suspicion and know the pt's etiology.

- Mild tachycardia
- Mild tachypnea
- Normal BP, may trend downward a little
- Normal urine output
- Slightly cool extremities (hands/feet)
- Pt may be anxious

Stage 2: Compensatory Shock

Neuro endocrine responses are triggered by decreased tissue perfusion. The SNS gets activated and all kinds of stuff happens... \uparrow HR, \uparrow RR, \uparrow Glycolysis at the liver, \downarrow urine output, \downarrow peristalsis, vasoconstriction to \uparrow BP (via renin-Ang pathway), diaphoresis.

So, who sends out that message to get this chain of events started?

- Right atrium: stretch receptors
- Baroreceptors: decreased blood volume
- Chemoreceptors: alterations in pH

Stage 3: Progressive Shock

Progressive shock occurs when compensatory mechanisms cannot restore homeostasis. At this point, you have almost lost the battle. Your pt needs to be intubated to decrease WOB! It is characterized by:

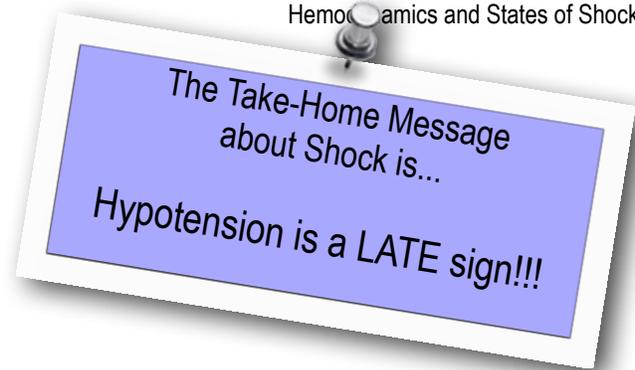
- continued low blood flow
- poor tissue perfusion
- inadequate oxygen delivery
- buildup of metabolic wastes (lactic acid)

$$CO \text{ (L/min.)} = SV \text{ (cc/beat)} \times HR \text{ (beats/min.)}$$

Preload | Afterload
Contractility

Progressive Shock, cont'd

- multiple organ dysfunction (MODS)
- start seeing hypotension
- cold
- altered LOC
- acidic pH
- tachycardia, tachypnea
- urine output below 30 ml/hour



Refractory Shock

At this point, you probably won't be able to save the pt. Tissue perfusion is SEVERELY compromised at this point and cell destruction is extensive. It is called "refractory shock", because the condition is refractory to treatment, meaning it does not improve with treatment. So, you'll have profound hypotension despite potent vasoactive drugs and/or fluids and your pt will be hypoxemic despite oxygen therapy (lungs not getting perfused). You may see bradycardia at this point...the body is giving up.

Etiology of Shock States...what causes it?

Recall that there are four types of shock...hypovolemic, transport, obstructive, cardiogenic.

Hypovolemic Shock

- Fluid loss d/t hemorrhage, dehydration, burns (there is not enough fluid in the bucket)
- Vasodilation d/t neurogenic shock, anaphylactic shock, septic shock (the bucket is too big for the fluid that is there)

S/S of Hypovolemic Shock

- The S/S are related to the degree of volume depletion
- Skin cool, capillary refill delayed
- BP low, patient orthostatic (looking for 10% change in HR, BP when changing position)
- Tachycardia
- Urine output is low
- PAWP is low
- CVP is low (0-2)
- SVR is elevated (as a compensatory mechanism to keep BP up...d/t vascular response)

Neurogenic Shock

In this type of shock, the bucket is too big...it is a VOLUME problem! It is d/t a loss of sympathetic innervation and often happens to people with spinal cord injuries at C3, C4, C5.

- Massive vasodilation, decreased SVR
- Pooling of blood in vessels
- Decreased venous return
- Decreased CVP, PAWP and CO
- HR is also decreased...PNS has taken over!

Anaphylactic Shock

- Occurs in response to severe allergic rxn
- Massive amounts of vasoactive substances are released from mast cells
- This causes vasodilation!
- There is also increased capillary permeability
- Increased size of intravascular compartment
- Hypovolemia occurs from maldistribution (decreased venous return and decreased CO)
- Your pt will show up hypotensive, tachycardic, with wheezes and hives. Other S/S are:
 - itching, cutaneous flushing, urticaria
 - throat swelling/fullness, wheezes
 - anxiety, tightness in chest
 - decreased LOC

Septic Shock

Septic shock is a systemic response to infection that involves massive vasodilation. It is caused by invading organisms of all types, but is most often gram-negative (UTI, VAP, H1N1). It triggers a complex series of cellular and humoral events. The organisms release endotoxins that lead to vasodilation and leakiness of vessels. Cytokines are also released which also produces vasodilation and leaky vessels. This pt needs 2 large bore IVs and fast fluid!

S/S Septic Shock

- Temp of > 38 or less than 36 if immunocompromised, child, elderly
- HR > 100
- RR > 20
- PaCO₂ > 32
- WBC > 12 or < 4 with 10% bands (note that the body will sacrifice RBCs for the sake of making more WBCs...this is called "anemia of chronic disease")
- Decreased SVR (normal is 800-1200)
- Decreased CVP, PCWP

Transport Shock States

Transport shock has to do with diminished oxygen carrying capacity that is d/t either anemia or carbon monoxide. CM is an odorless, tasteless gas. It rapidly binds to Hgb to form carboxyhemoglobin, taking up receptor sites for oxygen. It also interferes with release of oxygen from Hgb. Blood will be pink and red ("Coma" book example)

S/S Transport Shock

- Caused by anemia or hemorrhage
 - Low Hct and Hgb
 - Pressures may be normal
 - Tachycardia is usually present to compensate
- Carbon monoxide poisoning
 - HA, malaise, nausea
 - Loss of memory and personality changes
 - Elevated carboxyhemoglobin (>70%)
 - Tx= lots and lots of O₂ to knock the CM off the receptor sites

Obstructive Shock

Obstructive shock occurs when there is a mechanical barrier to blood flow d/t a pulmonary embolism, tension pneumo or cardiac tamponade. Note that cardiac tamponade can lead to PEA (pulseless electrical activity)...the heart is sending out impulses but it's not pushing anything around.

S/S Obstructive Shock

- Pulsus paradoxus (SBP ↑ on exhalation, drops on inspiration)
- Distant heart sounds (muffled)
- CVP, PAWP are both elevated
- Becks triad: Elevated CVP, decreased BP, muffled heart tones
- Dyspnea if PE or tension pneumo
- Pleuritic pain and cough with hemoptysis

Cardiogenic Shock

This is a pump problem! The heart basically fails as a pump. It can be caused by right-sided or left-sided heart failure (what's the most common cause of left-ventricular failure? right ventricular failure!)

- Extensive myocardial infarction
- Cardiomyopathy
- Valve dysfunction
- Papillary muscle rupture

cardiogenic shock, cont'd

- Left-sided heart failure
 - hypoperfusion and pulmonary congestion
 - dyspnea, crackles
 - distant heart sounds
 - elevated PAWP
 - low CO/CI
- Right-sided heart failure
 - systemic venous congestion
 - peripheral edema
 - elevated CVP, JVD
 - normal or low PAWP

General Treatment of Shock

- Optimize oxygen delivery (IV fluids to restore optimal tissue perfusion, restore preload, increase CO)
 - Oxygen delivery via NRB or ET tube...pt will get NRB even if O2 sats are currently fine
 - IV fluids
 - Crystalloids (NS or LR) and a small amount of colloid (albumin) to help mobilize fluid from 3rd space
 - Packed RBCs to increase oxygen carrying capacity if pt is anemic
 - Inotropic medications if the pt has a pump problem (dobutamine)
 - Increases contractility
 - Increases SV
 - You will titrate the dobutamine to C.O/perfusion, NOT to BP.
 - Pt may also get some lasix if they have too much fluid on board
 - Reduce afterload with some drugs and decrease the workload of heart (need Swan line!)
 - Vasodilate
 - Improve CO and oxygen delivery
 - Monitor BP carefully (meds are very potent)
 - Vasopressors (used with sepsis...Norepi/Levophed) NOTE: The bucket must be full before using these!
 - Acts on smooth muscle of blood vessels
 - Affects preload and afterload
 - Mimics SNS
 - Increases SVR and BP
- Decrease oxygen consumption (decrease total body work, decrease pain/anxiety, decrease temp)
 - Decrease total body work (WOB, pain and anxiety, temp)
 - Decrease oxygen demands
 - ventilation decreases work of respiratory muscles
 - neuromuscular blocking agents
 - propofol induces deep sedation
 - sometimes you will induce a coma to treat the pt!

More Specific Treatments

- Treating Hypovolemic Shock
 - Fill up the bucket!
 - Identify and control the source of fluid loss
 - Two large bore IVs
 - Crystalloids and colloids
 - Maybe blood if warranted
 - Assess for improvement in HR, BP and urine output...in that order.
 - HR improves after about 300 ml, BP improves in about 500 ml, and urine output after 1 L
- Treating Neurogenic Shock
 - Maintain spinal stability is key...need to get the SNS to work again
 - Optimize oxygen delivery
 - Preload should be restored with IV fluids
 - Vasopressors to support BP

- Watch for bradycardia and treat as warranted
- Treating Anaphylactic Shock
 - Maintain airway
 - Support BP via fluids and vasopressors/epinephrine (0.4 mg of 1:1000 SubQ)
 - Histamine blockers (Benadryl, Tagemet, Pepcid)
 - Bronchodilators
 - Steroids
- Treating Transport Shock
 - Restore oxygen carrying capacity
 - Give RBCs for anemia
 - Oxygen at high flow for carbon monoxide poisoning
- Treating Obstructive Shock
 - Goal: remove the mechanical barrier
 - chest tube or needle decompression of tension pneumo
 - pericardiocentesis for tamponade
 - Heparin, IVC filter or thrombolytic therapy for PE
 - Observe for PEA
- Treating Cardiogenic Shock
 - Goal: decrease myocardial oxygen demand; improve oxygen supply
 - Fluids
 - Inotropics (dobutamine...increases cardiac output without causing vasoconstriction. It decreases the workload of the heart, but still increases oxygen demand as it stimulates the B-receptors to increase contractility and stroke volume)
 - Airway management
 - Relief of pain/anxiety to decrease SNS response and decrease oxygen consumption
 - Hemodynamic monitoring to titrate meds to accomplish the goal of decreasing SVR, increasing CO.
 - Diuretics...use with hemodynamics...at the very least a CVP number is needed
 - Vasopressors
 - IAB P (Intra aortic balloon pump)...a cardiac assist device that augments the pumping action of the heart, can be used temporarily if pt needs revascularization or valve replacement.
 - Revascularization

SURVIVING SEPSIS CAMPAIGN GOALS!

- Administer abx within 1 hour of order
- Colloid or crystalloids until CVP 8-12
 - Maintain MAP > 65
- Urine output > 0.5ml/kg/hr
- Central venous O2 sat > 70%
 - Hgb > 7.0 g/dl
- Blood glucose < 150 mg/dL

INITIAL RESUSCITATION OF SEPSIS

- 500 - 1000 ml crystalloids/30 mins
- +300-500 ml colloids/30 mins
- Repeat fluids to maintain rec CVP, MAP
- Vasopressors (norepi, dopamine)
- Arterial catheter
- Give vasopressors through central line
- Corticosteroids

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