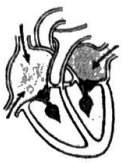
The Concepts of Hemodynamics

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

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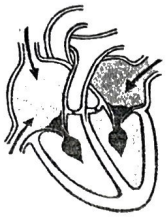
What can cause these kinds of bradycardias? The most common causes are:

- 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 (MVO_2) 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 MVO_2. 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.

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The last idea in the Frank-Starling Law is that you can overstretch the heart - by filling it so full of fluid that the muscle fibers can't contract back well if at all. This is what happens in congestive heart failure.

Compliance

Compliance is part of the stroke volume determination. It refers to the distensibility of the ventricular myocardium - or its ability to **stretch**. People with normal compliance have hearts that are able to stretch with volume loads - for example, you would do fine if you chugged a 20 ounce glass of water on a hot day - your heart would be able to accommodate that change.

People who have increased compliance, though, run the risk of overstretching and not being able to contract back well. Conditions that have increased compliance are:

- Congestive heart failure
- Dilated cardiomyopathy

On the flip side, people with decreased myocardial compliance don't stretch well to accommodate changes in load. People who have:

- a myocardial infarction,
- a stunned myocardium from surgery or trauma, or
- restrictive cardiomyopathy

may all have difficulty handling increased loads of fluid.

Afterload

The last concept is afterload. Afterload is how hard the heart (either the right or the left side) has to push to get the blood out. Afterload is also thought of as the resistance to flow or how **clamped** the blood vessels are.

Afterload is determined by:

- The compliance of the aorta
- Mass/viscosity of blood: how thick or thin it is
- Vascular resistance: whether the blood vessels are constricted or dilated
- Oxygen level: hypoxemia will cause vasoconstriction

Monitoring hemodynamics the old fashioned way

Long before there were monitors, cables, and lines, health care providers had to look, listen, and feel the patient to determine their hemodynamic status. Because all tissues are dependent on oxygenated blood, a deficiency in delivery will affect each organ system.

The "first things to go" when the blood circulation is not what it should be are the skin and the gut. Indicators of diminished blood supply to these organs are:

- Cool, clammy skin
- Pale, ashen, or cyanotic skin color
- Diminished bowel sounds
- Diarrhea or constipation
- Increased NG tube drainage

The "second things to go" are the kidneys and lungs. Failure to get blood to these organs has much more serious consequences:

- Increased respiratory rate and effort
- Shortness of breath
- Decreased PaO_2 on ABG or decreased SaO_2
- Crackles in the lungs from heart failure
- Decreased urine output
- Increased urine concentration
- Elevated BUN/creatinine/potassium

Finally, the brain and the heart are very greedy for oxygenated blood. They are the first and last organs to be perfused. When they fail, indicators may be:

- Decreased or altered level of consciousness
- Disorientation
- Slowly reacting pupils

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- Chest pain/pressure
- Tachy or brady-dysrhythmias, ectopy
- ST segment elevation

Monitoring Using Tools

Why do we sometimes choose to use invasive lines instead of monitoring the patient in non-invasive ways?

Well, our patients are not always straight-forward, textbook cases. They are complicated and complex and can be difficult to diagnose. Secondly, invasive monitoring can help determine what kinds of treatment should be started, as well as evaluating how well the treatment is working. And last, treatment for another problem may affect how the blood circulates; invasive line monitoring can also measure those effects.

There are four types of invasive lines:

1. Arterial line
2. CVP
3. Pulmonary artery catheter
4. Left atrial line

Of course, not everyone who is admitted to an ICU needs to have invasive arterial lines. Virtually all post-open heart surgery patients, however, will have at least an arterial line, if not a PA or CVP line in too. There are certain conditions where invasive monitoring is quite helpful:

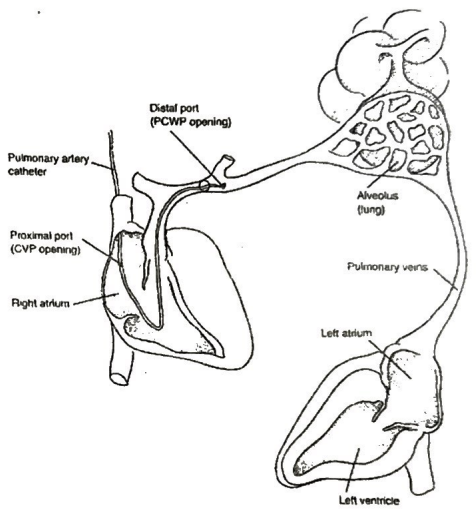
- Complicated MI
- Unstable MI with drug titration
- CHF/pulmonary edema
- Multisystem failure/ shock
- High risk cardiac patient for surgery or procedure
- High risk OB
- Respiratory failure

The Arterial Line

The arterial line transmits a systolic and diastolic pressure through a system that turns the pressure into an electrical waveform and a number. In short, the arterial line gives us a blood pressure. In cardiac surgery patients, the mean arterial pressure is followed, with a desired MAP between 60-80 mmHg.

The Pulmonary Artery Catheter

The pulmonary artery (PA) catheter is also known as the Swan Ganz catheter. It will measure three different pressures directly and is the method to obtain the cardiac output, contractility, and afterload information.



The PA catheter is floated through a major vein into the superior or inferior vena cava. From there, it is placed into the pulmonary artery. It has a pressure sensor in the right atrium and another one in at the distal end of the catheter to measure the pulmonary artery pressure. When the balloon at the end of the catheter is inflated, pulmonary capillary wedge pressure is obtained to give information about the **left** side of the heart.

Right atrial pressure

The right atrial pressure (RAP) measures the venous return to the right heart. It is a **right heart preload** measurement.

The RAP is essentially the same pressure that is obtained with a CVP on a triple lumen catheter.

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Normally, the RAP will be between 2 and 6 mm Hg. It can be increased in:

- Fluid overload
- Cardiac tamponade
- Right heart dysfunction
- Pulmonary problems

The RAP is decreased with:

- Dehydration/volume loss
- Venodilation

Right ventricular pressure

This pressure is not documented routinely, as it should only be seen during the passage of the catheter. The normal value for RV pressure is 20-30 systolic/0-5 diastolic.

Pulmonary artery pressure

The pulmonary artery pressure (PAP) is measured at the distal end of the catheter. The normal values for PAP are 20-30 systolic/10-20 diastolic.

Increased pressures are found in:

- Atrial or septal defects
- Pulmonary hypertension
- LV failure
- Mitral stenosis

Pulmonary capillary wedge pressure

Often called the "wedge," this pressure is obtained when the balloon on the end of the PA catheter is inflated. This blocks off all pressures from the right side of the heart, allowing the pressure sensor at the tip of the catheter to see, indirectly, the pressures on the left side of the heart. It is a **left heart preload** measurement.

Normally, the PCWP is between 8 and 12 mm Hg. It is increased in:

- Fluid overload
- Mitral valve stenosis
- Aortic stenosis or regurgitation
- LV failure
- Constrictive pericarditis or tamponade

The PCWP is decreased with:

- Hypovolemia
- Vasodilation

The diastolic number of the PA pressure is often used instead of the PCWP, particularly in cardiac surgery patients. The PAD is always higher (by 1-4 mm Hg) than the PCWP, but is a close approximation.

Cardiac Output

The cardiac output can be measured by using the PA catheter. When the procedure has been finished, the computer will generate a bunch of numbers. Here are the numbers with the normal values and what they measure:

Cardiac output: 4-8 L/min

Cardiac index: 2.5 - 4.0 L/min/m^2 (keep > 2.1)

Stroke volume: 60-100 ml/beat *Contractility*

Systemic vascular resistance (SVR): 800-1200 dynes/s/cm^{-5} *Left heart afterload*

Pulmonary vascular resistance (PVR): 50-150 dynes/s/cm^{-5} *Right heart afterload*

The direct measurements from the PA catheter give information on the preload status of the heart.

RAP: 2-6 Hg *Right heart preload*

PCWP: 8-12 mm Hg *Left heart preload*

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Manipulation of Cardiac Output

Well, okay, now we've got all of these numbers - what do we do with them? Here's the overall idea:

We want to get blood to the tissues without beating the heart to death.

To optimize cardiac output, we need to optimize each of the components of cardiac output - the heart rate, preload, contractility, and afterload. Here are the strategies for the different components.

Heart rate

If the patient is in a non-compensatory tachycardia, such as atrial fibrillation, atrial flutter, supraventricular tachycardia (SVT), or paroxysmal atrial tachycardia (PAT), the first interventions are usually pharmacological:

- Beta blocking agents: such as atenolol, metoprolol, propranolol, sotalol, or esmolol
- Calcium channel blockers, such as diltiazem and verapamil
- Adenosine

If drugs don't work, or the patient is unstable, synchronized cardioversion is the treatment of choice.

Ventricular tachycardia is another non-compensatory tachycardia that should be treated **immediately.** If the patient is stable, lidocaine or amiodarone are the drugs of choice. A second line drug is procainamide. If drugs don't convert the v-tach, or if the patient is unstable, he/she is cardioverted.

Bradycardia is a bit more straight forward. If the heart rate is too slow to support the cardiac output, it needs to speed up. The drug of choice is **atropine**. Epinephrine and dopamine may also be tried. Usually, though, if atropine doesn't work, the patient is either put on a transcutaneous **pacemaker** or has a transvenous pacemaker placed.

Contractility

If **low** contractility is the problem, the interventions take two paths: either to increase the stretch or to increase the strength.

Increasing the stretch goes right back to the Frank Starling Law - if you stretch further, you should contract back better. Crystalloids, such as normal saline, lactated Ringers, or D_5NS, will stay in the vascular space and will stretch the myocardium.

Colloids, such as albumin, Dextran, Hespan, fresh frozen plasma, or packed RBC's, are also used. Colloids are preferable in situations where there is edema, as they will act to pull fluid out of the "3rd space" and put it back into the vasculature.

Increasing the strength should start with fixing what's causing the decreased squeeze, such as correcting hypoxia, hypercapnia, and electrolyte imbalances. The squeeze can also be increased by giving positive inotropic drugs, like moderate dose **dopamine**, **dobutamine**, **epinephrine**, or **milrinone**.

Sometimes the problem is that the contractility is too **high** - the heart is squeezing harder than it has to. Because increased contractility increases the myocardial oxygen consumption, angina or MI may result. Drugs are used to decrease contractility:

- Beta blockers - acebutolol, atenolol, carteolol, labetalol, metoprolol, nadolol, oxprenolol, penbutolol, pindolol, propranolol, sotalol, timolol
- Calcium channel blockers - bepridil, diltiazem, felodipine, isradipine, nicardipine, nifedipine, verapamil

Preload

Problems with preload fall into two categories: there's too much or there's too little. If there is too much preload, furosemide or other **diuretics** are the first choice to "unload" the heart. **Renal dose dopamine** (2-5 mcg/kg/min) may be given. In special cases, **dialysis** may be indicated to remove excess fluid from the body.

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If there is not enough preload, it's simple - give some! The rule of thumb is to replace like-for-like. If they lost blood, give them PRBC's. If they've had diarrhea and vomiting for a week, give them crystalloids.

Most cardiac surgery patients are kept on the high end of preload with a RAP of 6-8 mm Hg and a PCWP of 12-14 mm Hg.

Afterload

Just like preload, afterload can either be too high or too low. If the afterload is too high - an SVR > 1200, the heart is working really hard to pump out its blood. Drugs are the first choice to decrease a high SVR:

- Nitroglycerin IV
- Nitroprusside IV
- If the patient is more stable, calcium channel blockers: diltiazem, felodipine, isradipine, nicardipine, nifedipine, verapamil

If pharmacologic management isn't doing the trick, an intra-aortic balloon may be inserted. The IABP helps to decrease SVR by deflating the balloon just before ventricular contraction, creating almost a "vacutainer" effect that allows the heart to work much less hard to get the blood out.

On the flip side, the SVR can be too low. A really low SVR indicates venous pooling, in which case, the preload drops. Vasoconstricting drugs can do the trick to increase SVR:

- Norepinephrine (Levophed®)
- Phenylephrine (Neosynephrine®)

Algorithm of Treatment

Please see the algorithm on the next page for recommended treatments to optimize cardiac output.

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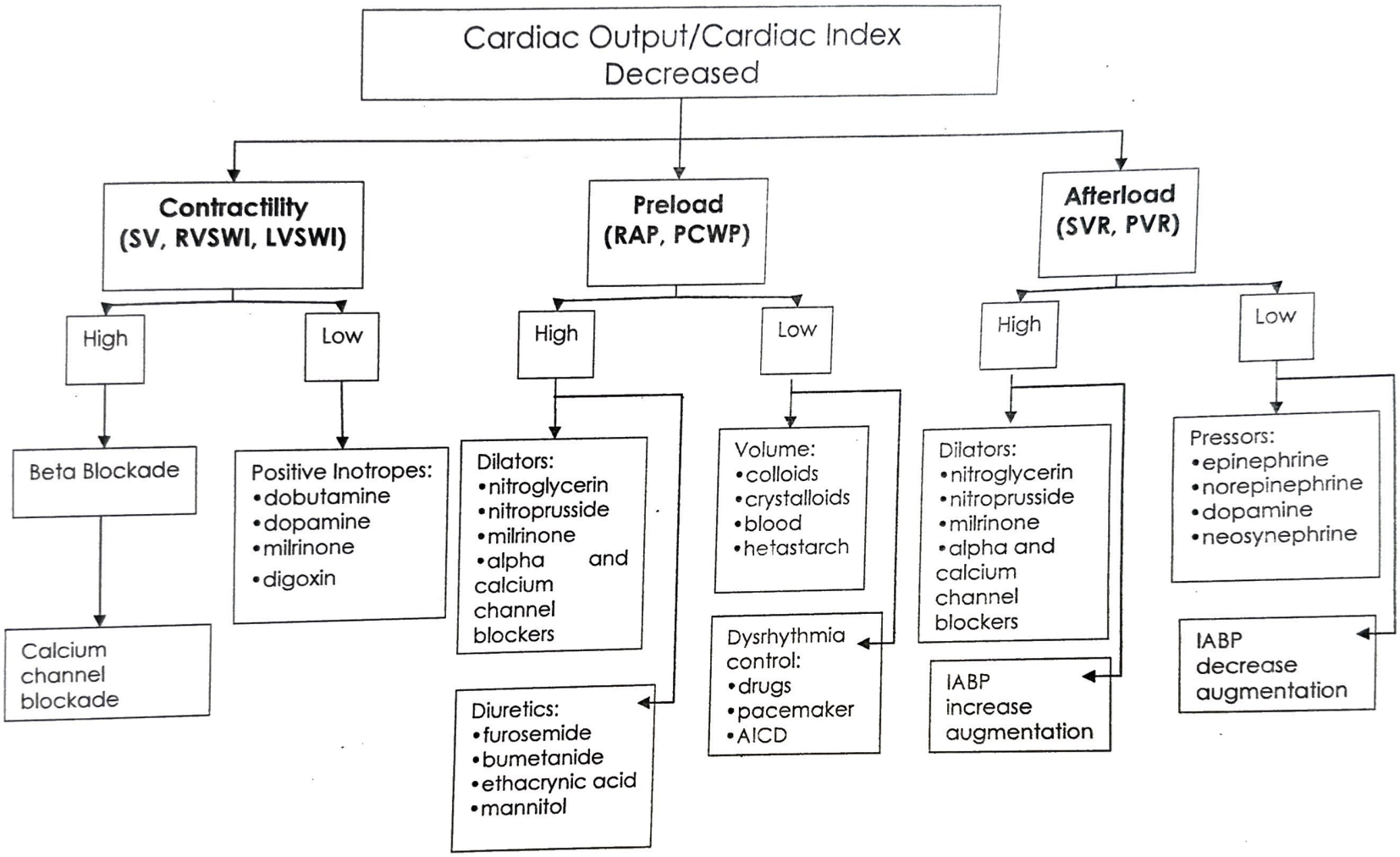

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Hemodynamic Pharmacology

Introduction

Now let's take a look at the medications that are used to manipulate cardiac output. First data is gathered to determine what aspects of the cardiac output need adjustment. Patient history, physical assessment, vital signs, and key hemodynamic parameters from the PA catheter all contribute to the assessment data. Once we've determined what the problems are, we are ready to determine which vasoactive drugs will benefit our patient. Remember, the primary goal is to optimize cardiac output. To do that, we need to optimize each of the components of cardiac output - the heart rate, preload, contractility, and afterload. In order to choose the appropriate medication, we need to keep in mind the physiological response that these medications produce.

Let's review a few relevant terms before we continue to discuss individual medications.

Inotropic: affects contractility

- A positive effect increases contractility
- A negative effect decreases contractility

Chronotropic: affects the heart rate

- A positive effect increases the heart rate
- A negative effect decreases heart rate

Dromotropic: affects conductivity

The hemodynamic effects of vasoactive drugs occur as a result of their interactions with receptors in the heart and vascular system. These receptors are:

Alpha-receptors:

- Are located in the blood vessels and cause vasoconstriction in most vessels, especially the arterioles.
- Increase afterload by causing peripheral vasoconstriction and increasing blood pressure.

Beta 1 receptors:

- Are located in the heart.
- Have inotropic, chronotropic, and dromotropic effects.

Because of their inotropic, chronotropic, and dromotropic effects, stimulation of the Beta 1 receptors increases contractility and heart rate, thus increasing cardiac output.

Beta 2 receptors:

- Are located in the bronchial and vascular smooth muscles.
- Cause bronchodilation, and vasodilatation.
- Reduce afterload.

Dopaminergic receptors:

- Are located in the renal and mesenteric artery bed.
- Dilate renal and mesenteric arteries.
- Reduce preload by inducing diuresis /natriuresis.

Diuretics: Decreasing Preload

According to the algorithm on page 8, preload is one of the three factors affecting cardiac output. When preload is too high (i.e., high RAP and PCWP), medications are used to reduce the preload. Medications that are used to reduce preload are diuretics and vasodilators.

Furosemide (Lasix®) and Bumetanide (Bumex®)

Lasix® and Bumex® are diuretics that act on the loop of Henle in the kidney. These medications decrease the preload by increasing urine output, thus reducing the work of the heart. They are used for acute pulmonary edema, CHF, peripheral edema, and HTN. Monitor serum electrolytes such as potassium closely because potassium is depleted as the patient diureses.

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Expected Hemodynamic Outcome for Lasix® and Bumex®

Heart rate	Preload (RA, PAW, PAD)	Contractility	Afterload (SVR)	Blood pressure
	↓			

Dopamine: Renal-Dose and Low-Dose

Dopamine will cause different vasoactive effects depending on the dose infused. At low doses, dopamine stimulates the dopaminergic receptors in the arteries located in the kidneys, abdomen, heart and brain. The vasodilatation of renal and mesenteric arteries causes an increase in urine output and results in a decreased preload. This is referred to as "renal-dose dopamine."

Renal-dose dopamine is used for patients who have a high preload or low urine output. This often occurs in patients with CHF.

Receptors stimulated by renal and low-dose dopamine

Alpha (Vaso-constriction)	Beta 1 (Heart rate and contractility)	Beta 2 (Vaso-dilatation)	Dopaminergic (Renal dilation)
			✓

Renal-dose dopamine is generally considered to be in the range of approximately 0.5-2mcg/kg/min.

When further reduction in preload is needed, dopamine may be titrated up into the "low dose" range. Low dose dopamine is approximately 2-5mcg/kg/min. and is titrated by 1-2 mcg/kg/min every 5-10 min. Low dose dopamine will have little to no effect on the heart rate or blood pressure.

Expected hemodynamic outcome for renal and low dose dopamine

Heart rate	Preload (RA, PAW, PAD)	Contractility	Afterload (SVR)	Blood pressure
	↓			

Vasodilators: Decreasing Afterload and Preload

Vasodilators are used to reduce afterload and, to a somewhat lesser extent, reduces preload. Reducing afterload decreases the amount of squeeze needed to circulate blood, conserving myocardial O_2 consumption. Reducing preload involves getting rid of excess fluid in the system. This decreases the amount of blood in the right atrium prior to contraction of the atria during the cardiac cycle. The two medications discussed here are nitroprusside and nitroglycerine.

Nitroprusside or Nipride

Nipride is used for hypertensive emergencies to reduce blood pressure.

Nipride relaxes the arterial and venous smooth muscles causing vasodilatation thus decreasing afterload. The primary effect is arterial vasodilatation. This is a very potent medication and can quickly drop the BP and the SVR (afterload). Monitor closely when making any dose changes.

This medication works very quickly, which means when you start the medication there are immediate effects. The half-life is so short that when you stop the medication its effects are gone.

The dosage range is 0.5 to 5 mcg/kg/min. and the average dose is 2 mcg/kg/min. Start nipride at 0.5mcg /kg/min and titrate by 0.25-0.5 mcg/kg/min every 2 min to achieve the desired parameters determined by the physician. Nipride may be titrated to B/P or SVR.

Use a volumetric infusion pump to administer this medication. Nipride may be infused in a peripheral IV. As a vasodilator, nipride will not cause tissue damage upon infiltration as can occur with vasopressors such as dopamine.

Do not flush or bolus in the IV site of nipride because the blood pressure will drop immediately. Nipride tends to deteriorate in the presence of light. To protect nipride from light, the container should be covered with foil, or another opaque material and

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discarded after 12 hours. An arterial line is beneficial for continuously monitoring the blood pressure.

At high doses or with long term therapy (>72 hours), nipride converts to thiocyanate and may result in cyanide toxicity. Do not exceed 10mcg/kg/min. for longer than 10 minutes or it will increase the risk for cyanide toxicity. If infused at this high of a rate it must be turned off because of the potential for cyanide toxicity.

One of the risks associated with nipride administration is a precipitous drop in blood pressure. Sometimes dopamine is needed to keep the blood pressure in a therapeutic range, while providing afterload reduction.

Expected hemodynamic outcome for nipride

Heart rate	Preload (RA, PAW, PAD)	Contractility	Afterload (SVR)	Blood pressure
	↓		↓↓	↓↓

Nitroglycerin

Nitroglycerin is used for patients with angina and hypertension.

This medication is a venodilator; dilating the venous system. When the veins are dilated blood moves more easily through the system, reducing the pressure needed to circulate the blood around. Coronary perfusion will increase, and both preload and afterload will decrease.

Nitroglycerin may be infused in a peripheral IV. Use a volumetric infusion pump. The usual dosage starts at 5-10 mcg/min. The range is 10-200 mcg /min. Titrate nitroglycerine by 10 mcg every 5 minutes to achieve the goal blood pressure or relief of chest pain.

The dosage of nitroglycerin may also be based on the patient's weight. The normal range is 0.5 –1.5 mcg /kg/min. Titrate by 0.1 –0.2 mcg /kg every 5 minutes.

It is common to have complaints of a headache because of the dilation happening in the cerebral vasculature. Acetaminophen is usually effective for headaches caused by nitroglycerine.

Expected hemodynamic outcome for Nitroglycerin

Heart rate	Preload (RA, PAW, PAD)	Contractility	Afterload (SVR)	Blood pressure
	↓↓		↓	↓

Positive Inotropes: Improving Contractility

Contractility or "squeeze" is the third component that affects cardiac output. Drugs that have beta 1 effects increase the contractility o the heart.

Dobutamine

Dobutamine is used for patients experiencing heart failure, cardiogenic shock, and sometimes following cardiac surgery for patients requiring intravenous inotropic support.

Receptors stimulated by dobutamine

Alpha (Vaso-constriction)	Beta 1 (Heart rate and contractility)	Beta 2 (Vaso-dilatation)	Dopaminergic (Renal dilation)
Minimal	✓	✓	

Dobutamine increases cardiac contractility (a positive inotropic effect) because of the beta 1 effect of the medication. By increasing the squeeze of the heart or contractility, it will help to increase the blood pressure and cardiac output.

These same beta 1 effects also increase heart rate and may lead to an increased myocardial oxygen demand due to tachycardia.

Dobutamine also has beta 2 effects which cause vasodilation, resulting in a decrease in afterload / systemic vascular resistance. Dobutamine may also decrease PAW or preload to the L side of the heart.

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The usual dosage is 2.5 to 20 mcg/kg/min. Start at 2 mcg/kg/min. Titrate dobutamine by 1-2 mcg/kg/min every 5- 10 minutes to obtain the desired B/P.

Dobutamine should be infused in a central line because it does have some alpha (vasoconstrictive) effects. There may be tissue necrosis if infiltration occurs. Use a volumetric infusion pump. An arterial line is beneficial for continuously monitoring the blood pressure.

Expected hemodynamic outcome for dobutamine

Heart rate	Preload (RA, PAW, PAD)	Contractility	Afterload (SVR)	Blood pressure
↑	↓	↑ ↑	↓	↑

Milrinone (Primacor®)

Milrinone is used for low cardiac output due to poor contractility. This is commonly seen in patients with acute, decompensated right heart failure. Milrinone is a good medication for CHF as a short-term therapy.

Receptors stimulated by milrinone

Alpha (Vaso-constriction)	Beta 1 (Heart rate and contractility)	Beta 2 (Vaso-dilatation)	Dopaminergic (Renal dilation)
none	***none***	***none***	***none***

Milrinone is a good medication for improving contractility but the mechanism of action is different than the medications we have already discussed. It does not stimulate the alpha or beta cells. Milrinone increases the cyclic-AMP concentrations in the cell which improves contractility and vasodilatation (afterload reduction).

While milrinone increases contractility and decreases afterload, it has minimal effect on the heart rate. This means that it increases cardiac output without an increase in heart rate or oxygen consumption.

Recommended dosage is a 50 mcg/kg load over 10 minutes, followed by a maintenance dose of 0.375 to 0.75 mcg/kg/min. The dose will vary depending on renal function. Use a volumetric infusion pump. Because milrinone does not cause vasoconstriction, it may be infused peripherally.

Expected hemodynamic outcome for milrinone

Heart rate	Preload (RA, PAW, PAD)	Contractility	Afterload (SVR)	Blood pressure
	↓	↑ ↑	↓	

Dopamine: Medium-Dose

Remember "renal-dose" dopamine? When the dose of dopamine is increased the beta 1 and 2 receptors are stimulated causing an increase in heart rate, contractility and, to a lesser extent, vasodilatation.

A medium dose of dopamine is used to achieve a positive inotropic effect in patients with heart failure. Heart rate also increases. When used in this fashion, the effects are similar to dobutamine.

Receptors stimulated by medium-dose dopamine

Alpha (Vaso-constriction)	Beta 1 (Heart rate and contractility)	Beta 2 (Vaso-dilatation)	Dopaminergic (Renal dilation)
	✓	✓	

The medium-dose range is about 5-10 mcg/kg/min.

Expected hemodynamic outcome for medium-dose dopamine

Heart rate	Preload (RA, PAW, PAD)	Contractility	Afterload (SVR)	Blood pressure
↑		↑ ↑	↑	↑

Inotropes/ Vasopressors: Increasing Afterload

Many times critically ill patients have problems with low blood pressure. To ensure that all the body's

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organs are being perfused properly, it is necessary to keep the blood pressure in an acceptable range.

Vasopressors are used to increase the blood pressure by constricting the arterial blood vessels. Constricting the vascular system also increases the afterload and cardiac output. Vasopressors are used to manage such situations as severe hypotension and cardiac arrest.

In general, all vasoactive medications should be given in a central IV port. If a vasoconstrictor infiltrates, tissue necrosis may occur. If this happens, notify the physician immediately. The recommended treatment is Phentolamine, infiltrated subcutaneously at the extravasation site.

Vasoconstrictors clamp down or shunt blood away from the periphery in order to give blood to the more important organs and systems. Monitor the circulation to the extremities as part of your routine assessments. Dusky and/or mottled extremities that are cool or cold to the touch can occur with peripheral vasoconstriction. There are times when the need to maintain BP outweighs the need to maintain good circulation to the extremities. The physician will try to balance these needs as much as possible.

Use a volumetric infusion pump. An arterial line is beneficial for continuously monitoring the blood pressure.

Dopamine: High-dose (10-20 mcg/kg/min)

High-dose dopamine is reserved for the treatment of severe hypotension that is not related to hypovolemia.

Receptors stimulated by high-dose dopamine

Alpha (Vaso-constriction)	Beta 1 (Heart rate and contractility)	Beta 2 (Vaso-dilatation)	Dopaminergic (Renal dilation)
✓			

In high doses, dopamine stimulates the alpha-receptors causing vasoconstriction. This effect tends to override the other effects that occur at lower doses (including the vasodilator effect).

When high-dose dopamine clamps down on the blood vessels, internal organs are not perfused as well and the "renal effect" is lost; urine output may decrease. Systemic vascular resistance increases and the amount of squeeze needed to circulate the blood also increases. Couple that with an increase in heart rate and it is easy to see why there is also an increase of myocardial oxygen demand.

A dopamine infusion is usually started at 5 mcg/kg/min and is titrated at 1-2 mcg/kg/min. every 5-15 minutes until adequate results are achieved.

When you are administering high doses of dopamine, you may want to consider using norepinephrine (Levophed®) in addition to dopamine, or as an alternative.

Expected hemodynamic outcome for high-dose dopamine

Heart rate	Preload (RA, PAW, PAD)	Contractility	Afterload (SVR)	Blood pressure
↑	↑	↑	↑	↑↑

Norepinephrine (Levophed®)

Levophed® is used for profound hypotension caused by such conditions as myocardial infarction, septicemia, transfusion reactions and drug reactions.

Receptors stimulated by Levophed®

Alpha (Vaso-constriction)	Beta 1 (Heart rate and contractility)	Beta 2 (Vaso-dilatation)	Dopaminergic (Renal dilation)
✓	Minimal		

Levophed® is a potent vasoconstrictor used to increase blood pressure. The primary effects are alpha-adrenergic effects, resulting in vasoconstriction. Used mainly for pressor effects indicated in severe hypotension secondary to low peripheral resistance.

When using Levophed®, always correct hypovolemia first. Vasoconstriction when the "tank" is dry will

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not increase blood pressure. Levophed is an especially good medication for septic shock.

The loading dose for Levophed® is 8-12 mcg/min., followed by a maintenance dose of 2-4 mcg/min. The therapeutic dosage range is 2-12 mcg/min. Titrate by 1-2 mcg every 5-10 minutes to achieve the desired effect.

Levophed® has the potential to cause end-organ renal damage with prolonged use due to vasoconstriction. Because of the possibility of causing extreme hypertension, monitor vital signs closely.

Expected hemodynamic outcome for Levophed®

Heart rate	Preload (RA, PAW, PAD)	Contractility	Afterload (SVR)	Blood pressure
			↑	↑

Phenylephrine (Neosynephrine®)

Phenylephrine is used in the management of hypotension caused by shock, anesthesia or hypersensitivity reactions to drugs.

Receptors stimulated by phenylephrine

Alpha (Vaso-constriction)	Beta 1 (Heart rate and contractility)	Beta 2 (Vaso-dilatation)	Dopaminergic (Renal dilation)
✓			

Phenylephrine has a powerful effect on the alpha-receptors causing potent vasoconstriction, although it is not as potent as Levophed®. phenylephrine completely lacks the chronotropic and inotropic effects on the heart.

The usual dose of phenylephrine starts at 100-180 mcg/min then decreases to 40-60 mcg/min once stabilized. The usual dose range is 20-200 mcg/min.

Like Levophed®, phenylephrine also has the possibility of causing end organ damage with prolonged use due to vasoconstriction.

Expected hemodynamic outcome for phenylephrine

Heart rate	Preload (RA, PAW, PAD)	Contractility	Afterload (SVR)	Blood pressure
			↑	↑

Epinephrine (Adrenaline)

Epinephrine is naturally occurring hormone secreted by the adrenal glands. A sympathomimetic, it imitates almost all the actions of the sympathetic nervous system (the "fight or flight" response). This medication is commonly used intravenously during cardiac arrest, and as an infusion for severe hypotension.

Receptors stimulated by epinephrine

Alpha (Vaso-constriction)	Beta 1 (Heart rate and contractility)	Beta 2 (Vaso-dilatation)	Dopaminergic (Renal dilation)
✓	✓	✓	

Epinephrine has mixed effects on the receptors of the sympathetic nervous system. It stimulates alpha, beta 1 and beta 2 receptors.

The results of alpha stimulation are vasoconstriction and an increase in blood pressure. An increase in contractility and heart rate occur as a consequence of beta 1 stimulation. Myocardial oxygen demand is increased as a result.

The usual dose is 1-8 mcg/min. or 0.01-0.05 ug/kg/min. Titrate epinephrine by 1mcg/min or 0.01 mcg/kg every 5 minutes. Monitor blood pressure every 5 minutes.

Expected hemodynamic outcome for epinephrine

Heart rate	Preload (RA, PAW, PAD)	Contractility	Afterload (SVR)	Blood pressure
↑		↑	↑	↑

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vasopressin (Pitressin®)

Vasopressin is most commonly used for vasodilator shock, GI bleeding, and organ donor management.

Receptors stimulated by vasopressin

Alpha (Vaso-constriction)	Beta 1 (Heart rate and contractility)	Beta 2 (Vaso-dilatation)	Dopaminergic (Renal dilation)	Other
none	***none***	***none***	***none***	✓

Vasopressin is a naturally occurring anti-diuretic hormone most commonly used in the treatment of diabetes insipidus. The hormone is released in the presence of a low blood volume and has direct effect on V1 vascular smooth muscles receptors causing constriction. When vasopressin is administered at higher doses, vasoconstrictive effects occur due to vasopressin's action on vasopressin receptors.

The usual dosage is quite low, at about 0.04 units /min. with the usual dosage range of 0.01-0.1 units/min. Vasopressin is not usually titrated; rather it is left at a set dose. If blood pressure management is indicated, it is best to use other medications.

Higher doses have been used with GI Bleeding, although there is some evidence that the higher dose may increase mortality.

Be careful in the transcription and administration of this medication. Because of the small dosages, decimal point errors are common when orders are written and transcribed. A single decimal point mistake results in a dose that is TEN TIMES what was intended.

Expected hemodynamic outcome for vasopressin

Heart rate	Preload (RA, PAW, PAD)	Contractility	Afterload (SVR)	Blood pressure
				↑

Applying Concepts to Case Examples

Now let's take a look at the different medications you will use to manage your patient's hemodynamic status. Please fill in your answers in the spaces provided. Compare your answers to those at the end of the case studies.

Case 1:

Bob has an Anterior Wall Myocardial Infarction (AWMI) with sustained hypotension **SBP < 85** and a **low cardiac output** due to **poor contractility.** There is an **elevated preload** to the left side of the heart (PAW: pulmonary artery wedge), and an **elevated afterload** (SVR: systemic vascular resistance).

In order to manage Bob's hemodynamics each component should be addressed.

Bob's preload is elevated
In order to optimize Bob's preload you may want to use a._____.

Bob also has an elevated afterload (SVR).
In order to reduce the resistance the heart has to work against, afterload reducers or _____are a good choice.

In order to help Bob with the low contractility, a _____ _____medication would be beneficial.

Case 2

Your next patient, Joe, is admitted with a necrotic bowel. He has a fever of 39.8° C and a WBC of 26,000. His cardiac output is very high (9.6 L /min) and his **B/P is very low:** 68/ 40. His **afterload or SVR is low:** 480.

In order to maintain adequate arterial pressure and organ perfusion _____are needed.

Answers: (Case 1) diuretic, vasodilators, positive inotropic. (Case 2) vasoconstrictors or pressors

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