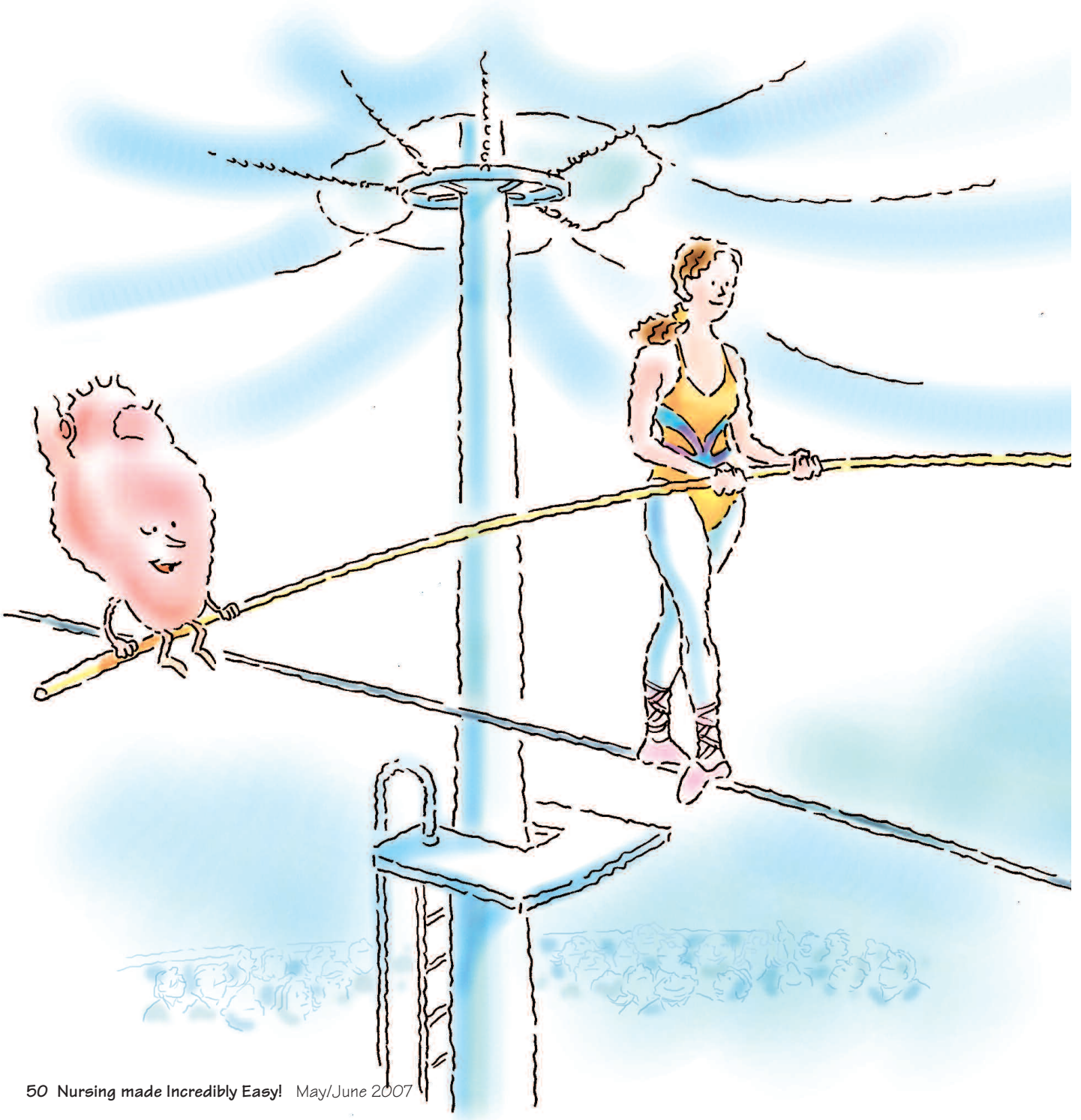
Putting an end to perfusion confusion



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Hemodynamic monitoring—just reading the words can spark a wave of confusion. What exactly are you measuring? And what in the world do all those acronyms stand for? Don't worry, we've got you covered. In this article, we'll help you make sense of this important topic—the *Incredibly Easy* way!

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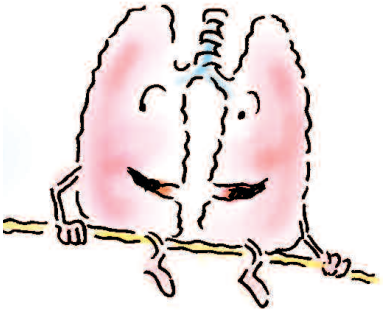
The author has disclosed that she has no significant relationships with or financial interest in any commercial companies that pertain to this educational activity.



ALL HUMAN TISSUE is dependent on the heart-lung connection. The heart pumps blood to the body, and the lungs oxygenate that blood and remove carbon dioxide. Hemodynamic measurements describe the intravascular pressure and flow that occur when the heart muscle contracts and pumps blood through the body via the vascular system. The three elements that combine to keep the vascular system in working order are the heart (a pump), blood (volume for the pump to move), and vascular tone (resistance against the pump). So, basically, hemodynamic measurements relate the status of these elements.

Both noninvasive and invasive techniques can be used to determine a patient's hemodynamic status. Every time you take a patient's blood pressure, you're using a *noninvasive* hemodynamic monitoring device. The primary purpose of *invasive* hemodynamic monitoring is to detect and monitor changes in intravascular pressures and cardiac output, which allows for optimum patient management. Specialized catheters, intravenous (I.V.) tubing, and instruments are inserted into the vascular system to measure pressures.

In this article, I'll examine the components and interrelationship of the cardiovascular and pulmonary systems. Then I'll show you how to measure cardiac output and tissue oxygenation, with a focus on managing the hemodynamically compromised patient.

First, let's take a look at the essentials of the cardiac system.

Pump it up!

The cardiovascular system carries oxygen and nutrients in the blood to all cells in the body. The heart maintains adequate perfusion to organs and tissue systems by balancing volume and sustaining contractility. Blood circulation is a continuous, synchronized process. Blood returns to the right atrium from the superior and inferior vena cava and flows through the tricuspid valve into the right ventricle. As the heart muscle contracts, blood is sent through the pulmonary vasculature for oxygenation in the lungs.

Blood returns to the left atrium through the pulmonary veins and flows through the mitral valve into the left ventricle. It's ejected through the aortic valve, into the aorta, and then delivered to the rest of the body. Both the left and right ventricles contract almost simultaneously. Blood reaches capillary beds where oxygen is released to cells of organs and tissues. Veins then carry oxygen-poor blood back to the vena cava (see *Understanding the heart-lung connection*).

The events of the cardiac cycle include the following:

- ***Isovolumetric ventricular contraction.*** In

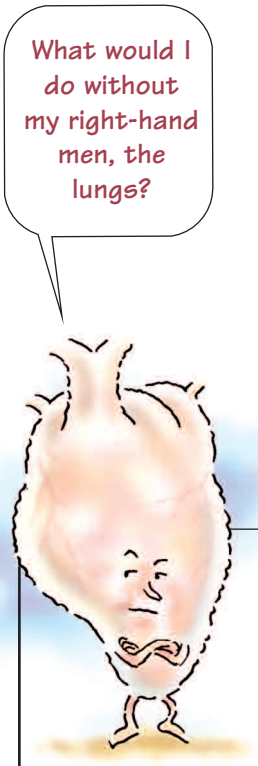
response to ventricular depolarization, tension in the ventricles increases. This rise in pressure within the ventricles leads to closure of the mitral and tricuspid valves. The aortic and pulmonic valves stay closed during the entire phase.

■ ***Ventricular ejection.*** When ventricular pressure exceeds aortic and pulmonary arterial pressure, the aortic and pulmonic valves open and the ventricles eject blood.

■ ***Isovolumetric relaxation.*** When ventricular pressure falls below the pressure in the aorta and the pulmonary artery, the aortic and pulmonic valves close. All valves are closed during this phase. Atrial diastole occurs as blood fills the atria.

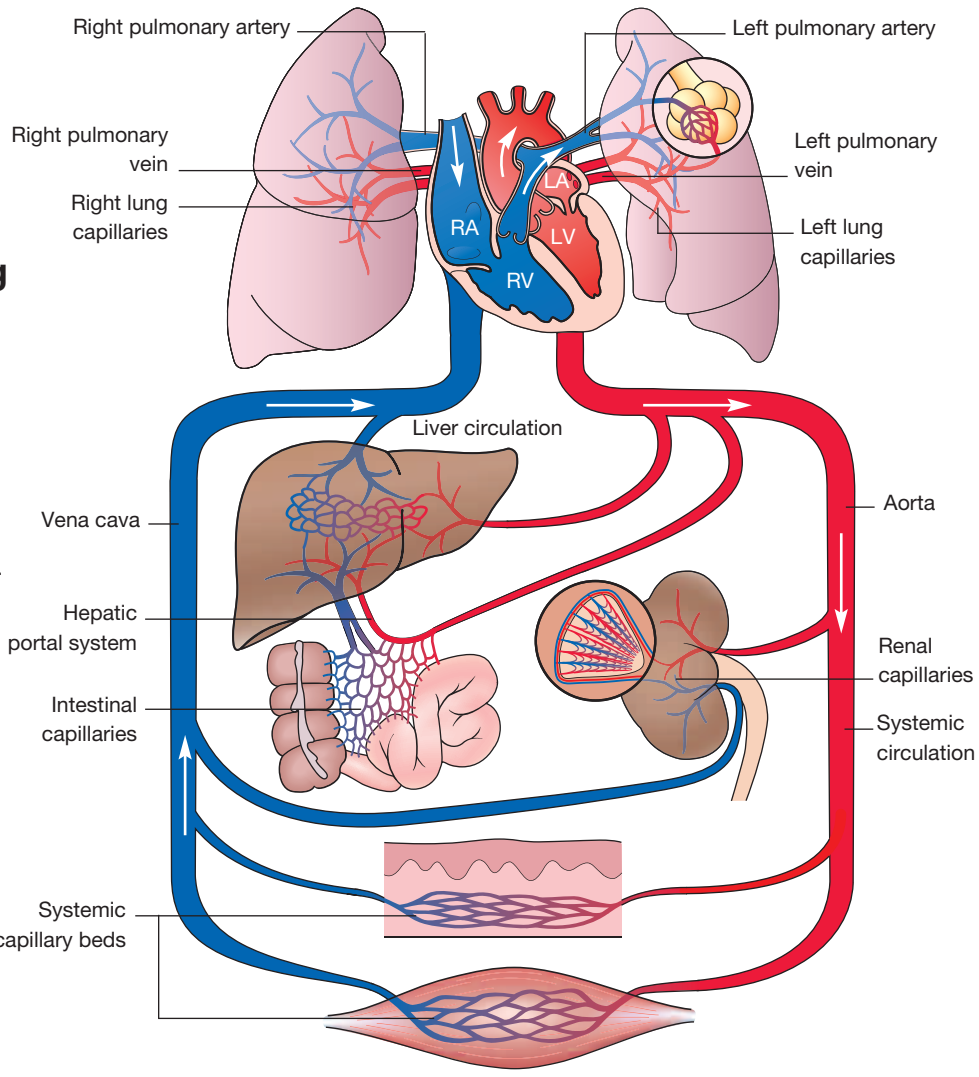
■ ***Ventricular filling.*** Atrial pressure exceeds ventricular pressure, which causes the mitral and tricuspid valves to open. Blood then flows passively into the ventricles. About 70% of ventricular filling takes place during this phase.

You may have heard the terms preload, contractility, and afterload mentioned in connection with the cardiac cycle, but do you know what they mean? Let's find out.



Understanding the heart-lung connection

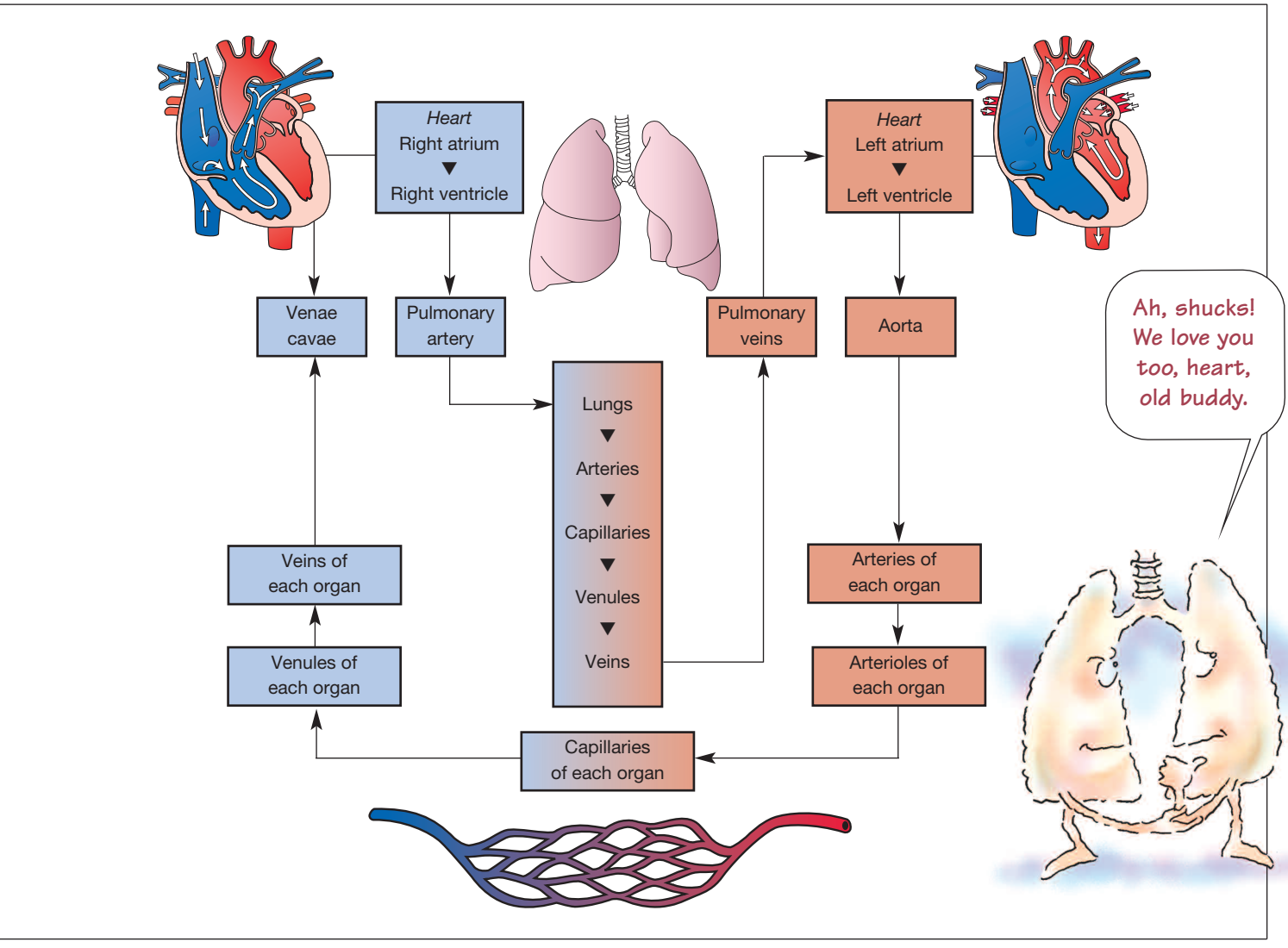
The illustration on the left traces the flow of blood through the heart, pulmonary system, and the rest of the body. Use the flowchart on the right to better understand the flow.



Expand and contract

Preload is the stretching of muscle fibers in the ventricle at the end of diastole. It's determined by the amount of blood volume returning to the heart and filling the ventricles at the end of diastole (end-diastolic pressure). Increased preload causes an increase in stroke volume, ventricular work, and myocardial oxygen requirements.

Contractility is the ability of muscle fibers to stretch in response to volume and eject that volume into the pulmonary and systemic circulations. Starling's Law states that the force of contraction can be varied based on the length of the myocardial fiber just before contraction. The stretch of the myocardial fiber is influenced by the quantity of blood within the ventricle just before systole or the ventricular end-diastolic volume. The more the ventricle is filled with blood during diastole, the greater the volume of ejected blood during systolic contraction. However, there's a limitation to this law: Extended overstretching of the fibers decreases contractility. Therefore,



once the physiologic limit is reached, the force of contraction declines.

How stiff or distended the ventricles become (known as compliance) can alter the relationship between end-diastolic volume and end-diastolic pressure. A ventricle with *decreased* compliance (more stiff and less able to distend) won't be able to accommodate even a small increase in venous return to the heart. This increased volume will result in a greatly increased ventricular end-diastolic pressure. Causes of decreased compliance include myocardial fibrosis, myocardial ischemia, and restrictive cardiomyopathies. If the ventricle has *increased* compliance (less stiff and more able to distend), it can accept more volume, which means the end-diastolic pressure will be lower. Increased compliance can be seen in dilated cardiomyopathy.

Afterload is the pressure that the ventricle has to overcome to eject its contents into the aorta during systole. The right ventricle must eject blood through the pulmonic valve against the low pressure of the pulmonary circulation, or pulmonary vascular resistance (PVR). The left ventricle must eject blood through the aortic valve against the high pressure of the systemic circulation, or systemic vascular resistance (SVR). Increased afterload causes a decrease in ventricular compliance and stroke volume. The higher the afterload, the harder the heart has to work to eject its contents, resulting in increased myocardial oxygen demand.

How about cardiac output, stroke volume, and heart rate? Let's take a look at them next.

A handy equation

Cardiac output is the amount of blood that the heart pumps in 1 minute, measured in liters/minute. The formula for calculating cardiac output is: Cardiac output = heart rate x stroke volume. Normal cardiac output is 4 to 8 L/minute.

Stroke volume is the amount of blood ejected by the ventricle with each contraction. It's affected by preload, contractility, and afterload.

Heart rate is the number of times the heart beats in 1 minute, measured in beats/minute. If the heart beats too fast or too slow, cardiac output decreases.

Now, let's take a peak at the essentials of the pulmonary system.

I'm blue without you

The pulmonary system delivers oxygen to the bloodstream and removes excess carbon dioxide from the body. Effective respiration requires gas exchange in the lungs (external respiration) and in the tissues (internal respiration). Three external respiratory processes are needed to maintain adequate oxygenation and acid-base balance:

- ***ventilation***—gas distribution into and out of the pulmonary airways
- ***pulmonary perfusion***—blood flow from the right side of the heart, through the pulmonary circulation, and back to the left side of the heart
- ***diffusion***—gas movement from an area of greater concentration to an area of lesser concentration through a semipermeable membrane.

Blood flow through the lungs is powered by the right ventricle. The right and left pulmonary arteries carry deoxygenated blood from the right ventricle to the lungs. In the lungs, the alveoli (tiny, thin-membraned air sacs) are responsible for gas exchange.

During gas exchange, oxygen from inhaled air diffuses into the blood through the alveolar and pulmonary capillary membranes as carbon dioxide diffuses in the opposite direction; carbon dioxide is then eliminated via exhalation. Oxygen is transported to the tissues on hemoglobin in the blood. Hemoglobin has a high affinity for oxygen and carries approximately 97% of

Hemodynamic parameters

Parameter	Normal value
Mean arterial pressure (MAP)	▪ 70 to 105 mm Hg
$MAP = \frac{Systolic + 2\ (Diastolic)}{3}$	
Central venous pressure; right atrium pressure (RAP)	▪ 2 to 6 cm H_2O; 2 to 8 mm Hg
Right ventricular pressure	▪ 20 to 30 mm Hg (systolic) ▪ 0 to 8 mm Hg (diastolic)
Pulmonary artery (PA) pressure	▪ 20 to 30 mm Hg (PA systolic) ▪ 8 to 15 mm Hg (PA diastolic) ▪ 10 to 20 mm Hg (PA mean)
Pulmonary artery wedge pressure	▪ 4 to 12 mm Hg
Cardiac output (CO)	▪ 4 to 8 L/minute
$CO = Heart\ rate \times Stroke\ volume$	
Cardiac index (CI)	▪ 2.5 to 4 L/minute/m^2
$CI = \frac{CO}{Body\ surface\ area}$	

Parameter	Normal value
Stroke volume (SV)	▪ 60 to 100 mL/beat
$SV = \frac{CO \times 1{,}000}{Heart\ rate}$	
Stroke volume index (SVI)	▪ 33 to 47 mL/beat/m^2
$SVI = \frac{CI \times 1{,}000}{Heart\ rate}$	
Systemic vascular resistance (SVR)	▪ 800 to 1,200 dynes/sec/cm^{-5}
$SVR = \frac{MAP - RAP}{CO} \times 80$	
Systemic vascular resistance index (SVRI)	▪ 1,900 to 2,400 dynes/sec/cm^{-5}/m^2
$SVRI = \frac{MAP - RAP}{CI} \times 80$	

cheat sheet

all oxygen in the body. The amount of oxygen bound to hemoglobin is measured as arterial oxygen saturation (SaO_2). A small portion of the oxygen dissolves in plasma, measured as the partial pressure of arterial oxygen (PaO_2).

The tissues of a healthy person can extract large quantities of oxygen from hemoglobin molecules with minimal effect on oxygenation. But in a person who's ill, a shift in the relationship between dissolved oxygen and hemoglobin-bound oxygen (oxyhemoglobin disassociation curve) can occur. This indicates a change in how oxygen is taken up at the alveolar level and how it's delivered to the tissue. A shift to the right of the curve signifies hemoglobin has less affinity for oxygen; however, it enhances oxygen delivery at the tissue level. Some factors influencing a right shift include decreased pH, increased partial pressure of arterial carbon dioxide (PCO_2), increased temperature, anemia (inadequate amounts of hemoglobin), hyperthyroidism, and chronic hypoxemia (PaO_2 less than 60 mm Hg).

With a shift to the left of the curve, there's an increased affinity for oxygen and a decrease in delivery. A shift to the left may indicate increased pH, decreased PCO_2, decreased temperature, and hypothyroidism.

Mixed venous oxygen saturation ($S\bar{v}O_2$) is another parameter used to evaluate hemodynamic stability. Blood from the pulmonary artery or right atrium can be analyzed to determine the amount of oxygen that has

been extracted by the tissues in relation to how much oxygen was supplied. Normal $S\bar{v}O_2$ is 75%. If the tissues have extracted more oxygen, such as can be seen with trauma, the percentage will be lower; if the tissues have extracted less oxygen, such as can be seen with sepsis, the percentage will be higher. The $S\bar{v}O_2$ level is higher in sepsis because the microcirculation is filled with clots, which prevents the tissues from extracting oxygen from the hemoglobin. The amount of oxygen bound to hemoglobin returning to the heart is, therefore, higher than normal in sepsis.

Every beat of my heart

So, how do we measure cardiac function? Cardiac status can be evaluated using noninvasive or invasive hemodynamic monitoring techniques. Let's take a closer look.

Impedance cardiography (ICG) is a noninvasive way to collect hemodynamic data when assessing a patient with heart failure, hypertension, or dyspnea. An ICG device consists of a monitor with four dual electrodes that are applied to the patient's neck and thorax. The portions farther from the heart send the electrical current and those closer to the heart measure changes in the current during the cardiac cycle to determine cardiac output, stroke volume, SVR, and contractility. This technique is safe and painless, and it can be used at a single point in time or for continuous assessments. It's contraindicated in patients with septic shock, very recent sternotomy, shivering, tachyarrhythmia, or those with an intra-aortic balloon pump in place.

Most invasive hemodynamic monitoring systems work pretty similarly, and they usually have three components: a transducer, an amplifier, and a recorder/monitor. The

(hemo)Dynamic nursing interventions

Cardiac output monitoring

- Monitor the patient for signs and symptoms of inadequate perfusion, including restlessness, fatigue, changes in level of consciousness, decreased capillary refill time, diminished peripheral pulses, and pale, cool skin.
- Record the patient's cardiac output, cardiac index, and other hemodynamic values and vital signs at the time of measurement. Also note the patient's position during measurement.

PAP and PAWP monitoring

- Inform the patient that he'll be conscious during catheterization and he may feel temporary local discomfort from the administration of the local anesthetic. Catheter insertion takes about 30 minutes.
- After catheter insertion, you may inflate the balloon with a syringe to take PAWP readings.
- If fever develops when the catheter is in place, inform the health care provider; he may remove the catheter and send the tip to the lab for culture.
- Make sure stopcocks are properly positioned and connections are secure. Loose connections may introduce air into the system or cause blood backup, leakage of deoxygenated blood, or inaccurate pressure readings. Also make sure the lumen hubs are properly identified to serve the appropriate catheter ports.

Arterial blood pressure monitoring

- Explain the procedure to the patient, including the purpose of arterial pressure monitoring.
- After catheter insertion, observe the pressure waveform to assess arterial pressure.
- Assess the insertion site for signs of infection, such as redness and swelling. Notify the health care provider immediately if you note these signs.
- Document the date and time of catheter insertion, the catheter insertion site, the type of flush solution used, and the type of dressing applied.

transducer converts the fluid waves generated by blood flow into electrical signals that are presented numerically by electronic monitoring equipment. The amplifier, which increases the size of the signal from the transducer, is located inside the bedside monitor. A recorder or monitor displays the signal and records information. For tips on how to manage monitoring systems, see *(hemo) Dynamic nursing interventions*.

The ***thermodilution method*** is one such invasive hemodynamic monitoring technique used to evaluate the cardiac status of critically ill patients and those suspected of having cardiac disease. In this method, a quantity of solution that's cooler than the patient's body temperature is injected at timed intervals into the right atrium through the proximal port of a thermodilution pulmonary artery (PA) catheter (such as a Swan-Ganz catheter). The injectate mixes with the blood as it travels through the right ventricle into the pulmonary artery, and a thermistor on the end of the catheter registers the difference between the temperature of the injected fluid and the temperature of the blood as it reaches the thermistor (see *A closer look at the thermodilution method*). A computer then plots the temperature change over time as a curve and calculates flow on the area under the curve.

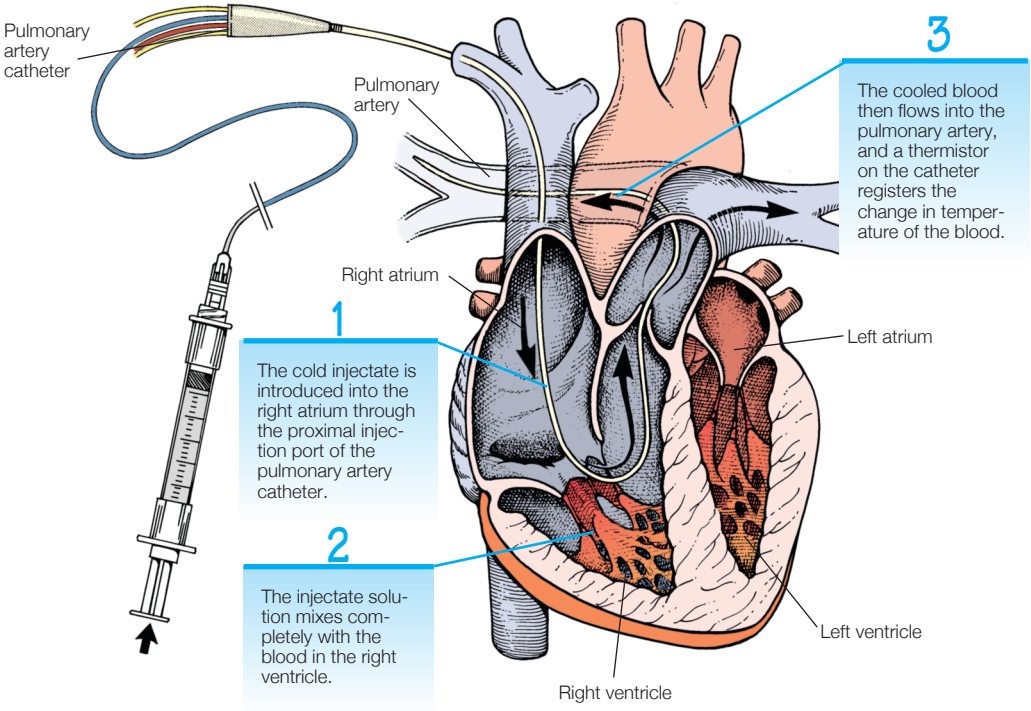
A normal waveform (4 to 8 L/minute) shows a sharp upstroke followed by a smooth curve and a slightly prolonged downslope. When cardiac output is high, the injectate is carried more quickly through the heart and a smaller curve is seen. When output is below normal, the curve is larger because more time is required for the temperature to return to baseline.

Here's how this works.

Using sterile technique, the physician inserts the catheter through the right or left internal jugular or subclavian vein, and then threads it through the right atrium, past the tricuspid valve to the right ventricle, and past the pulmonic valve to the pulmonary artery. To maintain a patent system, the distal and proximal ports are connected to a pressurized fluid system, which is calibrated to atmospheric pressure at the site of the transducer and the level of the phlebostatic axis. Without this system, blood would back up into the catheter. Each transducer receives a physiologic signal from the patient, which is transmitted as an electrical

A closer look at the thermodilution method

This illustration shows the path of the injectate solution through the heart during thermodilution cardiac output monitoring.



signal to the monitoring system. The monitor displays a waveform for each connection.

The proximal lumen opens into the right atrium. The low-pressure right atrium produces a waveform consistent with the cardiac cycle. Three positive deflections—a, c, and v—correlate with atrial contraction, upward movement of the tricuspid valve during ventricular contraction, and right atrial filling. Normal right atrium pressure (RAP) is 2 to 8 mm Hg.

As the catheter is passed through the right ventricle, the waveform amplifies in size secondary to the increased pressure. This rapid rise in pressure corresponds to early ventricular systole. When ventricular pressure exceeds the pulmonary artery pressure, the pulmonic valve opens and blood is ejected into the pulmonary circulation. Normal right ventricular systolic pressure is 20 to 30 mm Hg. Normal right ventricular end-diastolic pressure is equivalent to RAP.

The catheter is then advanced into the pulmonary artery. The distal port allows measurement of the pulmonary artery pressure (PAP). (Blood can also be drawn from this port to measure a mixed venous sample.) Closure of the pulmonic valve after systole causes right ventricular pressure to fall, but PAP remains high. Also, PA diastolic pressure is higher than right ventricular diastolic pressure because of pulmonary valve closure. Closure of the pulmonic valve generates the dicrotic notch seen on the pulmonary artery waveform and indicates the onset of diastole. Normal PA systolic pressure is 20 to 30 mm Hg. Normal PA diastolic pressure is 8 to 15 mm Hg. Normal pulmonary mean arterial pressure is less than 20 mm Hg.

Left-sided cardiac pressures are determined by obtaining pulmonary capillary wedge pressure (PCWP). This pressure reflects the function of the left atrium and the left ventricle. Because the mitral valve is open during diastole, the PA diastolic pressure can be representative of the left ventricular end-diastolic pressure. However, occlusion of a branch within the pulmonary artery with balloon inflation gives a more accurate reflection of this pressure in a healthy patient. In this way, the effects of pressure from the right side of the heart and pulmonary circulation don't influence the measurement. The low-amplitude wedge waveform is between 4 and 12 mm Hg under normal conditions.

If a patient has heart failure, the PA systolic pressure, PA diastolic pressure, and PCWP will be elevated because blood backflows into the pulmonary vasculature due to left ventricular failure. If the PA systolic and diastolic pressures are elevated but the PCWP is normal, the patient has a pulmonary issue that's causing the elevated pressures. This is commonly seen in acute respiratory distress syndrome.

Supply and demand

Now, how's tissue oxygenation measured? Again, noninvasive and invasive techniques can be used.

Pulse oximetry is a noninvasive way to measure peripheral oxygen saturation (Spo_2) by using two light-emitting diodes (LEDs) that send red and infrared light through a pulsating arterial vascular bed, such as in the fingertip or the earlobe. A photodetector slipped over the finger or earlobe measures the transmitted light as it passes through the vascular bed. It detects the relative amount of color absorbed by arterial blood, and then calculates the Spo_2 value. The Spo_2 value can be used to estimate the amount of oxygen molecules bound to hemoglobin molecules.

An invasive way to measure $S\bar{v}o_2$ is to use a special ***fiberoptic thermodilution PA catheter*** to continuously monitor oxygen delivery to tissues and oxygen saturation by tissues. A drop in $S\bar{v}o_2$ occurs as oxygen demand exceeds oxygen consumption. At rest, a healthy patient uses about 25% of available oxygen (remember, normal $S\bar{v}o_2$ is 75%). Under duress, the body's need for oxygen increases. When oxygen demand increases, the body responds by increasing

cardiac output to increase oxygen delivery, such as in hypovolemia, ventricular dysfunction, and shock. If the cardiovascular system is also stressed, the body's ability to meet oxygen demand is compromised.

Conditions that *decrease* $S\bar{v}O_2$ include cardiogenic shock, decreased cardiac output, decreased hemoglobin level, hyperthermia or fever, decreased SaO_2 levels, seizures, septic shock, and shivering.

Conditions that *increase* $S\bar{v}O_2$ include anesthesia use, chemical paralysis, increased SaO_2 levels, hypothermia, sepsis, increased cardiac output, increased hemoglobin level, and sedation.

How to treat hemodynamic instability

Problem	Treatment
Decreased preload	Volume expanders: crystalloids (such as normal saline solution and lactated Ringer's solution) or colloids (albumin or blood products)
Decreased systemic vascular resistance (SVR)	Vasoconstrictors: norepinephrine, dopamine (10 to 20 mcg/kg/min), vasopressin (0.04 units/min), or neosynephrine
Decreased preload; increased SVR (as compensation)	Volume replacement
Decreased preload; decreased SVR	Volume replacement and vasoconstrictors
Increased preload	Diuretics: furosemide or bumetadine
Increased SVR	Vasodilators: nitroglycerine or nitroprusside
Increased preload; increased SVR	Diuretics and vasodilators
Decreased cardiac output	Dobutamine

(hemo)Dynamic patient care

If your patient has congestive heart failure, acute respiratory distress syndrome, or sepsis or if he's experienced myocardial infarction, trauma, or open-heart surgery, his hemodynamic status should be evaluated. So, what are some things you need to do if you're monitoring a hemodynamically compromised patient? Let's see.

If your patient's cardiac output is low, ask these questions:

- What are the filling pressures of the ventricles? Pressures in the right atrium and ventricle and the left ventricle indicate volume status.
- Are the ventricular filling pressures normal, high, or low? Elevated filling pressures indicate fluid overload.
- What's your patient's heart rate? A low heart rate could be contributing to the low cardiac output in the presence of normal volume status. A faster heart rate could indicate that the heart is attempting to compensate for a low volume status.

Evaluating PVR further defines treatment. In the presence of pulmonary hypertension, the right ventricle must pump against higher opposition. Dilation is one effect of this increased workload on the right ventricle over time. Increased PVR may mean that the patient requires additional volume to meet the preload needs of the left ventricle, even if filling pressures are within the normal range (remember Starling's Law). Factors that *increase* PVR include vasoconstricting drugs, hypoxemia, acidemia, hypercapnia (high partial pressure of arterial carbon dioxide [$PaCO_2$]), and atelectasis. Factors that *decrease* PVR include vasodilating drugs, alkalemia, hypocapnia (low $PaCO_2$), and strenuous exercise.

If your patient's blood pressure is elevated in the presence of low cardiac output, the SVR is most likely elevated as well. Conditions that can *increase* SVR include hypothermia, hypovolemia, cardiogenic shock, and stress response. The left ventricle may not be able to provide adequate cardiac output to the systemic circulation if resistance is high. To lessen the work on the left

ventricle, systemic resistance needs to be reduced. This can be accomplished by infusing volume expanders if the preload is adequate, or vasodilators if the preload and SVR are elevated, such as in cardiogenic shock.

In the presence of hypotension and low SVR, as seen with sepsis, cardiac function may be hyperdynamic. Other conditions that can *decrease* SVR include anaphylactic and neurogenic shock, anemia, cirrhosis, and vasodilation. Pressor agents, like norepinephrine, vasopressin, dopamine, and neosynephrine, that vasoconstrict the vascular system are needed to tighten the vascular bed and improve blood pressure and stabilize cardiac output.

For more information, see *How to treat hemodynamic instability*.

The heart-lung connection

The cardiac and pulmonary systems are elaborate and their interdependence is total. For this reason, all hemodynamic parameters need to be evaluated concurrently to achieve the best outcome for your patient. Determining whether oxygenation is compromised is the primary consideration when evaluating your patient's cardiovascular status. Ultimately, the goal is to ensure that oxygen delivery is meeting the needs for oxygen consumption to prevent tissue death. ■

Learn more about it

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