Don't be puzzled by cardiovascular concepts

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A: Knowing the concepts of preload, afterload, and cardiac output is critical to understanding how the heart functions and how abnormal function of the heart affects your patient. This means that you must have a thorough understanding of these concepts in order to accurately assess and evaluate your patient's cardiac functioning. Medications are utilized to alter preload, afterload, and cardiac output to enhance the ability of the heart to work more efficiently. So you also need to be able to evaluate the effects of these medications.

Q: What's preload?

A: Preload is the degree of stretch in the ventricle at the end of diastole. Hemodynamic monitoring of central venous pressure (CVP) at the bedside evaluates right-sided heart functioning and provides a direct measurement of preload based on volume. Normal CVP ranges from 0 to 8 mm Hg.

Understanding preload, contractility, and afterload

If you think of the heart as a balloon, it will help you understand the concepts of preload, contractility, and afterload.

Preload

Blowing up the balloon

Preload is the stretching of muscle fibers in the ventricle. This stretching results from blood volume in the ventricles at end diastole. According to Starling's law, the more the heart muscles stretch during diastole, the more forcefully they contract during systole. Think of preload as the balloon stretching as air is blown into it: The more air, the greater the stretch.

Contractility

The balloon's stretch

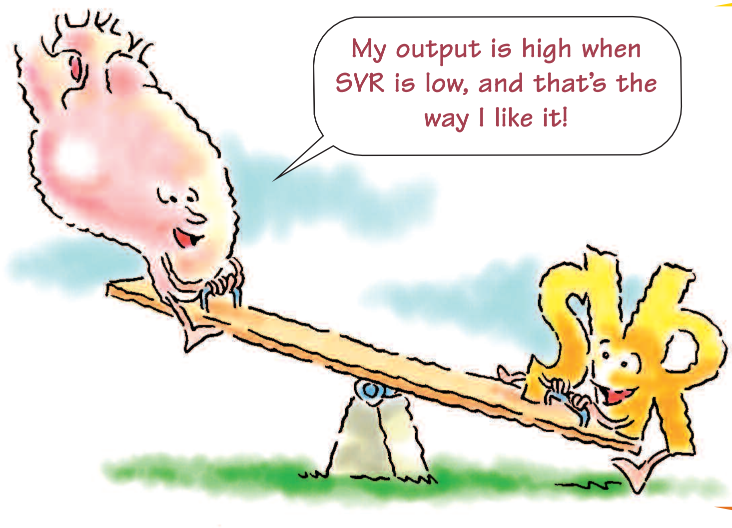
Contractility refers to the inherent ability of the myocardium to contract normally. Contractility is influenced by preload. The greater the stretch, the more forceful the contraction—or, the more air in the balloon, the greater the stretch and the farther the balloon will fly when air is allowed to expel.

Afterload

The knot that ties the balloon

Afterload refers to the pressure that the ventricular muscles must generate to overcome the higher pressure in the aorta to get the blood out of the heart. Resistance is the knot on the end of the balloon, which the balloon has to work against to get the air out.

Conditions that affect SVR



Conditions that can ***increase*** systemic vascular resistance (SVR) include:

- hypothermia
- hypovolemia
- stress response
- syndromes of low cardiac output.

Conditions that can ***decrease*** SVR include:

- anaphylactic and neurogenic shock
- anemia
- cirrhosis
- vasodilation.

But what does the concept of preload really mean? Let's start with Ernest Starling, who in 1914 developed the following law (known as Starling's law): Stretching the myocardial fibers during diastole will increase the force of contraction during systole. Myocardial fibers can be stretched by increasing ventricular diastolic volumes. The degree of fiber stretch, or preload, is determined by ventricular volume. The volume of blood contained within the ventricles during diastole depends on the amount of venous return. Venous return is dependent on circulating blood volume and venous tone. Increasing venous return consequently increases venous volumes and stretches the myocardial fibers. Preload can be a compensatory mechanism when cardiac output isn't adequate; however, it can also wreak havoc on the heart's ability to pump adequately.

It's easy to understand preload using the following analogy: Take a rubber band, pull it out halfway, and then release it. Now ask yourself: What happens to the force of the rubber band as it's exerted by the stretch? You should feel a strong forceful movement of the rubber band. If this were the heart, blood would've been propelled out and into the systemic circulation, maintaining cardiac output. If cardiac output is maintained, preload is adequate.

Now take the rubber band and stretch it out only a fourth of the way. Assess the distance pulled and the force at which the rubber band springs back. You should notice a weak or flimsy movement. If this were the heart, preload would be decreased.

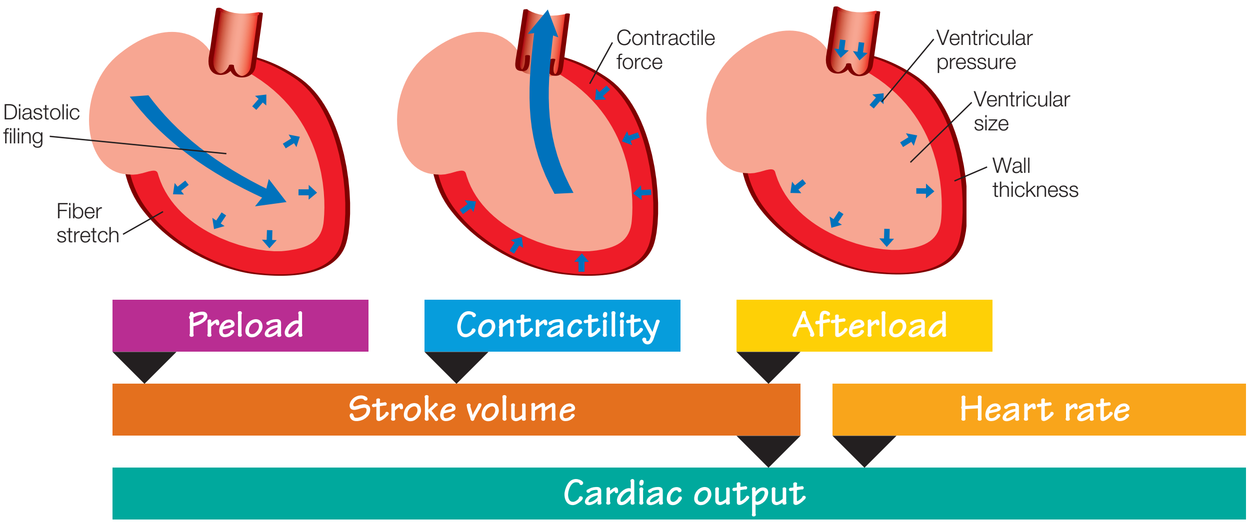
Lastly, take the same rubber band and stretch it as far as you can. The extreme stretching may cause the rubber band to break. Similarly, the heart muscle can only be stretched so far before it loses its ability to pump effectively. For another analogy you can use, see *Understanding preload, contractility, and afterload*.

Q: What about afterload?

A: Afterload is the amount of tension that the ventricle must overcome to open the aortic valve and eject blood into the systemic circulation. Also known as systemic vascular resistance (SVR), afterload reflects changes in the radius of the arterioles. The arterioles are resistant vessels because they constrict or relax. Normal SVR is 700 to 1,500 dynes/seconds/cm^{-5}.

An elevation in SVR may affect the emptying of the left ventricle, causing a drop in cardiac output. Conversely, if SVR decreases,

Influences on stroke volume and cardiac output



the pressure that the left ventricle must exert in order to open the aortic valve will lessen. This means that the heart pumps more efficiently and cardiac output increases (see *Conditions that affect SVR*).

Q: And what's cardiac output?

A: Cardiac output is the volume of blood flowing from the systemic and pulmonary circulation per minute. The formula for cardiac output is: stroke volume x heart rate. What's stroke volume, you ask? It's the amount of blood ejected with each contraction that's determined by three factors: preload, contractility, and afterload (see *Influences on stroke volume and cardiac output*). And heart rate is the rate at which the heart beats in 1 minute. Normal cardiac output ranges from 4 to 8 L/minute. Here's an example: If a patient's stroke volume is 70 mL with each contraction and his heart rate is 80 beats/minute, his cardiac output is 5,600 mL/minute (or 5.6 L/minute).

Q: Now, how do the concepts of preload and afterload relate to pharmacologic management?

A: If your patient's preload is high, several medications can be utilized to decrease the volume returning to the right side of the heart, such as diuretics and nitroglycerin. Diuretics facilitate the excretion of sodium, chloride, and water by the kidneys, decreasing volume and then preload. Nitroglycerin decreases preload by means of vasodilation.

There are several drugs that can be used to affect afterload, or SVR, such as angiotensin-converting enzyme (ACE) inhibitors and sodium nitroprusside. ACE inhibitors block the conversion of angiotensin I to angiotensin II, which results in vasodilation. Peripheral vasodilation causes a decrease in SVR. When afterload is decreased, cardiac output will increase because the left ventricle meets less resistance and can pump blood more efficiently.

In conclusion, health care professionals must be aware of the concepts of preload, afterload, and cardiac output because understanding these concepts helps promote comprehensive nursing care and optimize patient outcomes. ■

Learn more about it

Hemodynamic Monitoring Made Incredibly Visual! Philadelphia, Pa., Lippincott Williams & Wilkins, 2007:13-15.

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