

**McHenry Western Lake County EMS System**  
**Paramedic, EMT-B and PHRN**  
**Optional Continuing Education**  
**2018 #12**  
**Understanding Preload and Afterload**

Cardiac output (CO) represents the volume of blood that is delivered to the body, and is therefore an important factor in the determination of the effectiveness of the heart to deliver blood to the rest of the body. In this CE we will be looking at the components that make up cardiac output and what we can do to alter them based on our field impression.

Cardiac output by definition is the amount of blood ejected by the heart in a minute. Two major factors contribute to the CO: the heart rate, which is the number of heart beats/minute and the stroke volume, which is the volume of blood that is ejected from the heart with each contraction.

$$\text{Cardiac Output} = \text{Heart Rate} \times \text{Stroke Volume}$$

For an adult, an average CO is about 5 – 8 liters of ejected blood per minute. With strenuous activity, an adult's CO can increase to an amazing 25 liters per minute to satisfy the body's demands for oxygen and nutrients.

## **HEART RATE**

Heart rate and cardiac output have a direct relationship. As heart rate increases, so does CO. A heart rate of 100/minute will almost always result in a higher CO than a heart rate of 80/minute. There are some limits. Heart rates of more than 150/minute are often associated with a reduced CO. This is due to inadequate filling times. When the heart rate is greater than 150 the heart is beating so fast that the ventricles do not have time to fill. With less blood volume, stroke volume and CO falls.

Conversely, if the heart rate is too low, CO falls quickly. With a slow heart rate (< 50) there is certainly adequate time for the ventricles to fill, the problem is the heart rate is just too slow.

In a healthy heart rates between 50 and 150 are associated with adequate CO. Patients with a significant cardiac history (congestive heart failure and/or myocardial infarction) may have a low stroke volume. Heart rates as high as 150 may be associated with cardiac ischemia and reduced CO. A bradycardia combined with an already low stroke volume could result in shock with a CO of only 2000ml. The more pronounced a patient's cardiac disease the narrower the range of heart rates the yield a sufficient CO.

## **STROKE VOLUME**

Two major factors that determine stroke volume include preload and afterload.

### **Preload**

Preload is the force that stretches the muscle fibers of the ventricle at the end of diastole — how much they are stretched just before contraction. The amount of blood present within the ventricles before contraction and the condition of the myocardium determine the stretch or preload of the heart muscle. The greater the volume of blood in a heart chamber, the greater the preload. As the blood volume in the left ventricle increases, the cardiac muscle stretches and, up to a point, ejects its volume more effectively. Ideally, an adequately filled and stretched left ventricle should briskly contract and snap like a rubber band to send blood on its way. However, a point can be reached at which this stretch is so extreme that output is diminished. The relationship between fiber stretch and the force of contraction is known as “Frank-Starling's Law of the Heart.”

Starling's Law states that, up to a point, the more a cardiac muscle is stretched in diastole (resting stage), the more forcefully it contracts in the next systole (ejection stage). Optimal CO is dependent upon blood volume, heart rate, and achieving the appropriate amount of stretch. For example, a hemorrhaging trauma victim may have a ventricle that is inadequately filled. Therefore CO is reduced because of two mechanisms: inadequate blood volume in the ventricle and a lesser force of contraction that is caused by less muscle fiber stretch. However, compensatory mechanisms are triggered by the sympathetic nervous system in response to shock — an increased heart rate and contractility as well as other mechanisms temporarily sustain CO. Replacing lost circulating volume supports these compensatory mechanisms by increasing preload, thus enhancing cardiac muscle stretch and subsequent contraction, optimizing the effect of the Starling mechanism.

For the patient in heart failure who is volume overloaded, the ventricle has the opposite problem. Increased ventricular volume raises pressure within the ventricles, thereby augmenting myocardial stretch or preload and subsequent contraction. Initially, this serves as a compensatory mechanism with cardiac function reaching the maximum beneficial stretch described in Starling's Law; thus CO is optimized. If fluid overload continues, the pressure within the ventricle rises beyond the point of beneficial stretch, leading to less effective cardiac contraction (the stretched rubber band without the snap) and decreasing CO.

Preload is also known as left ventricular end-diastolic pressure (LVEDP). End-diastole represents the moment in the cardiac contraction-relaxation cycle when the ventricle contains the greatest volume of blood, just before it contracts and ejects its volume. The volume and the stretch or amount of tension placed on the heart muscle at that point determines LVEDP.

Several factors influence preload, including the distribution of blood within the body, total blood volume, sympathetic stimulation, the presence and force of atrial contraction (called the "atrial kick") and natriuretic peptides.

Blood distribution refers to the allocation of blood within the body (and in this case, the ventricles) at any specific time. The venous system can be thought of as a large reservoir that can hold blood in the peripheral circulation or return it back to the heart, depending on the state of vasodilation or vasoconstriction. For example, a drug that dilates the venous system, such as nitroglycerin (NTG), reduces preload by causing a greater volume of the blood to remain in peripheral circulation. Conversely, when higher blood pressure is needed, sympathetic stimulation causes vasoconstriction, which increases peripheral blood return to the central circulation augmenting CO. Gravity affects this distribution of blood. For example, elevating the legs of a supine patient redistributes blood to core organs such as the heart and brain when blood pressure is low. This position increases venous return by adding to the volume in the left ventricle, which stretches the cardiac muscle and enhances preload, and raises CO (and potentially, blood pressure).

When too much blood is distributed to a diseased left ventricle with poor muscle tone, it may become overstretched. This condition is known as left ventricular dysfunction. In left ventricular dysfunction, ventricular contraction is not forceful enough to eject its volume of blood with each contraction. You can help patients with left ventricular dysfunction by using gravity to redistribute blood to the lower extremities. You can do this by encouraging patients to dangle their legs from the side of the bed. Reducing venous return in this way lessens preload and decreases the work of the heart. Many patients with heart failure who develop lung congestion caused by increased preload learn this principle on their own. They find that sleeping in a recliner, elevating the head of the bed, or resting on multiple pillows alleviates symptoms and allows them to breathe easier. They are able to breathe easier because blood is redistributed, decreasing the volume that the heart must handle as preload.

Total blood volume is the common pool of blood available for distribution throughout the body; too little or too much can adversely affect preload. For example, blood loss from trauma may reduce preload by having less blood available to stretch the ventricle. Thus, a simple fluid bolus often improves the patient's cardiac status. On the other hand, a patient may have more blood in the body than the heart can handle, causing the ventricle to

overstretch, as happens with heart failure. Administering Nitroglycerin can reduce the volume of blood returning to the heart by vasodilation and lessen preload so the heart doesn't have to work so hard. Years ago, one treatment for fluid overload associated with heart failure was to therapeutically phlebotomize or "bleed" a patient to lessen the volume stretching the myocardium by decreasing total blood volume. Sympathetic stimulation can enhance preload by causing blood vessels to constrict, which increases blood return to the left ventricle. This stimulation also increases heart rate, ultimately improving CO. However, if the myocardium is injured, a faster heart rate can overwork the heart and increase its oxygen demand, causing further myocardial ischemia and injury.

Atrial contraction occurs just before the valves between the atria and ventricles close and is commonly referred to as "atrial kick." This action enhances ventricular preload by contributing up to 30% more volume to the ventricles at the end of diastole. When dysrhythmias, such as atrial fibrillation, occur and normal atrial contraction is absent, this added volume is lost.

When atrial and ventricular chamber pressures increase, endogenous peptides, called atrial natriuretic peptides (ANP) and B-type natriuretic peptides (BNP) are released to reduce preload and afterload. These peptides cause selective vasodilation and decrease sodium reabsorption, thereby decreasing preload and afterload.

### **Afterload**

Afterload is the resistance to ventricular ejection. Afterload is also defined as all the factors that influence ventricular wall tension during systolic ejection. Sources of resistance include blood pressure, systemic vascular resistance (SVR), and the condition of the aortic valve. When arterial vasoconstriction raises SVR, as in shock, or the aortic valve is very tight or stiff, as in aortic stenosis, the ventricle must generate a tremendous amount of pressure — or afterload — to overcome that resistance. It's like opening a door against a strong wind — it takes a lot of energy. This increases the workload of the heart that may result in ischemia or injury to myocardial tissue.

Sympathetic stimulation causes vasoconstriction of certain arteries, arterioles, and veins, thereby raising blood pressure. This increases cardiac workload. The ventricle now has to generate enough tension to raise the pressure within the ventricle above the pressure in the aorta to force the aortic valve open. Only then can the ventricle eject its contents. Imagine that you have a 60 cc syringe with a 25-gauge needle on the end and you are trying to eject the contents of the syringe as quickly as possible. It takes a tremendous amount of force to empty the contents of the syringe because the small diameter of the needle acts as resistance to flow.

Aortic stenosis can be congenital or occur after infections such as rheumatic fever or with aging as calcium deposits on valve leaflets. All of these conditions have the effect of creating an obstruction to the outflow of blood from the left ventricle. Consider the energy required to open a window that has been painted shut versus a window that freely opens. Valves open because the pressure generated on one side of the valve (left ventricle) exceeds the pressure on the other side (aorta). A stenotic valve creates a great deal of resistance to ejection, causing afterload to rise dramatically.

To open the aortic valve and eject blood, the ventricle has to overcome the resistance of the arterial blood pressure and resistance caused by the valve. Therefore, patients with chronic, untreated hypertension or aortic stenosis develop left ventricular hypertrophy in response to the high afterload. The same phenomenon occurs in skeletal muscles when a person undertakes a weight-lifting program. As the resistance to chamber contraction increases, the chamber adapts to this increased workload with the accumulation of increased fibre within the myocardial cells. This makes the cells stronger but also bulks up the cells, ultimately resulting in chamber hypertrophy. Unfortunately, these thicker chamber walls can be associated with additional complications such as decreased contractility, reduced stroke volume and cardiac dysrhythmias.

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Post Test

Name: \_\_\_\_\_

Date: \_\_\_\_\_

Dept: \_\_\_\_\_

Level of Practice: \_\_\_\_\_

1. Increased preload usually corresponds to increased contractility (force of contraction).
  - A. True
  - B. False
2. Normal cardiac output for a health adult is:
  - A. 1 – 3 L/minute
  - B. 2 – 4 L/minute
  - C. 5 – 8 L/minute
  - D. 7 – 9 L/minute
3. Complete the following formula for cardiac output.  
Cardiac Output = \_\_\_\_\_ X \_\_\_\_\_
4. Two major factors that determine stroke volume include \_\_\_\_\_ and \_\_\_\_\_.
5. \_\_\_\_\_ is the resistance to ventricular ejection.
6. Patients with healthy hearts will most likely hemodynamically tolerate heart rates between 50 and 150 beats per minute.
  - A. True
  - B. False
7. Which of the following factors tend to increase cardiac output? (Circle all that apply)
  - A. Increased preload
  - B. Decreased preload
  - C. Increased afterload
  - D. Decreased afterload
8. “Atrial kick” is lost in atrial fibrillation, explain how that effects preload.
9. Explain Nitroglycerin’s effect on preload.
10. Increase afterload increase/decreases the workload of the myocardium.

If you are NOT a member of the McHenry Western Lake County EMS System, Please include your address on each optional quiz turned into our office. Our mailing address is: Northwestern Medicine – McHenry Hospital EMS, 4201 Medical Center Drive, McHenry, Illinois 60050. We will forward to your home address verification of your continuing education hours.

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