

The Heart and Cardiac Output

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Managing cardiac emergencies relies heavily on an ability to recognize, understand and respond to altered cardiac output. This point cannot be emphasized enough. By understanding the factors that influence cardiac output, memory work becomes unnecessary.

This chapter serves as a beginning in the process of becoming a competent cardiac care practitioner. The cardiac cycle is first covered. Terms such as atrial kick, systole and diastole are defined. Cardiac output is then defined and defended as the important concept that it is.

Using case studies, the parameters that influence cardiac output are presented. Starling's law, preload, and afterload are addressed with particular attention to their practical clinical use. This chapter focuses on the big picture. What is the heart's main purpose? The answer may surprise you.

"It's all about managing cardiac output!"

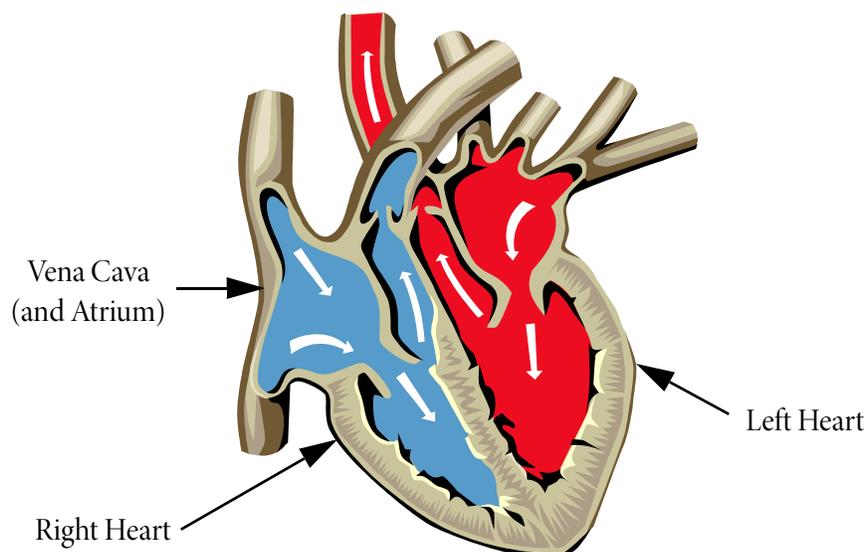
Not So Anonymous

The Cardiac Cycle

A complete cardiac cycle occurs with each audible 'lub-dub' that is heard with a stethoscope. During this heartbeat, both atria simultaneously contract followed soon after by the contraction of the ventricles. **Systole** is the contractile phase of each chamber while **diastole** is the relaxation phase. During the cardiac cycle, the atria and the ventricles each have periods of both systole and diastole.

The purpose of the cardiac cycle is to effectively pump blood. The right heart delivers deoxygenated blood to the lungs. Here oxygen is picked up and carbon dioxide is breathed off. The left heart delivers oxygenated blood to the body. Normally, the volume of blood ejected by the right ventricle to the lungs is about the same as the volume ejected by the left ventricle. A mismatch in volumes ejected by the ventricles (i.e. right ventricle pumps more blood than the left ventricle) can result in heart failure.

Figure 1.1 Route of Blood Flow Through the Heart



De-oxygenated blood enters the right side of the heart via the vena cava and is ejected through to the lungs where oxygen is replenished and carbon dioxide diffuses out to the lungs. Oxygenated blood enters the left side of the heart and is subsequently delivered to the body.

The synchronized actions of the atria and the ventricles are coordinated to maximize pumping efficiency. This sequence of events is worth considering. Rhythm disturbances can greatly impair this synchrony, resulting in a less effective cardiac cycle. For simplicity, we'll consider the events that lead to the ejection of blood from the right ventricle into the lungs beginning at the end of atrial diastole. These events mirror those of the left heart.

The tricuspid valve closes during ventricular systole - otherwise, it remains open. At end atrial diastole and ventricular diastole, an open tricuspid valve provides a channel between the right atrium and the right ventricle. As a result, blood flows into both the right atrium and the right ventricle simultaneously. The ventricle receives up to 85% of its blood volume during this period.

Prior to ventricular systole, the atrium contracts. Since the atrium is about 1/3 the size of the ventricle, an atrial contraction only contributes an additional 15-35% of blood volume to the ventricle. This 'topping up' of the ventricle by the atrium is called **atrial kick**. Note that the conclusion of atrial systole coincides with the end of ventricular diastole.



Atrial kick occurs as the atria contract prior to ventricular contraction. Atrial kick contributes 15-35% to the volume of blood in the ventricle. This extra volume in turn increases cardiac output by a similar 15-35%. **Note:** as we age, atrial kick tends to be a more significant contributor to cardiac output (closer to 35%). This is one reason that our older patients are more affected by rhythm disturbances such as atrial fibrillation (a quivering of the atria rather than a coordinated contraction) than our younger patients. Atrial fibrillation causes a complete loss of atrial kick.

After ventricular end-diastole, the ventricle enters systole and contracts forcefully. As the pressure within the ventricle increases, the tricuspid valve closes to ensure forward blood flow. Very soon after, the pulmonic valve opens as pressure within the ventricle becomes greater than pulmonary artery pressure. Blood is then ejected into the pulmonary arteries.

As blood is ejected, ventricular pressure falls. When ventricular pressure is below the pulmonary artery pressure, the pulmonic valve closes to prevent back flow of blood into the right ventricle. As mentioned in chapter one, the closure of the AV valves (tricuspid and mitral valves) normally produces the S_1 heart sound. The closure of the semilunar valves (pulmonic and aortic valves) produces the S_2 heart sound.

While ventricular systole ejects blood into either the pulmonary or systemic vascular systems, ventricular diastole is at least as important. Without a sufficient period of diastole, systole is ineffective. During diastole, the ventricles relax. But in relaxing, the ventricles open to regain their pre-contractile size, effectively dropping the chamber pressure below that of the vena cava. As a result, blood is drawn into the ventricle during ventricular (and atrial) diastole. Then the cardiac cycle begins again.

And this cardiac cycle is repeated over 100,000 times daily! Remarkable.

What is Cardiac Output?

This term ‘cardiac output’ has been used a few times already. What is cardiac output? Simply, **cardiac output** is the amount of blood ejected by the left ventricle in one minute. The left ventricle seems to get the lion’s share of attention perhaps because the body’s blood flow and pulse are provided by the left ventricle.

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

Some of us readily remember that cardiac output is calculated via the following formula:

Cardiac Output = Stroke Volume x Heart Rate

or

$CO = SV \times HR$

Cardiac output is a product of **heart rate** (beats per minute) and stroke volume. **Stroke volume** is the amount of blood ejected by the left ventricle with each contraction.

Let’s put this in perspective. What is your pulse rate? If a typical cardiac output is about 5000 ml (5 liters), what is your approximate stroke volume? For example, a patient named Mary has a pulse of 72/minute.

5000 = ____ (SV) X 72 (HR)

With a little math, Mary’s stroke volume is calculated to be about 70 ml.

SV = 5000 / 72 = 70 ml

Therefore, each time Mary’s left ventricle beats, it ejects about 70 ml of blood. Mary turns out to be about average when it comes to stroke volume. A typical stroke volume for adults is 50-80 ml. How about your stroke volume?

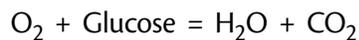
Why is Cardiac Output Vital?

Before we delve deeper into the particulars of cardiac output, it may be prudent to determine why cardiac output is vital to our well-being. Simply, cardiac output is intimately connected to energy production. Ample perfusion to the tissues yields an abundant energy supply. Poor tissue perfusion results in critical shortages of energy and often diminished function.

Blood, Oxygen and Aerobic Metabolism

An average adult has about 5-6 liters of blood (about 70 ml/kg). The blood serves many roles. Blood delivers nutrients and removes wastes. Blood also transports messengers such as hormones between sites, thus facilitating communication and responsiveness between various organs.

Paramount in importance, though, is the continuous flow of oxygenated blood. This flow is central to metabolism, the production of energy and other materials necessary for life. Energy production is synonymous with life. No energy...no life. Blood delivers oxygen and glucose to the tissues. One molecule of glucose is oxidized in the cell's mitochondria to produce 36 adenosine triphosphate molecules (ATP).



↓
↓
↓
36 ATP

Metabolism that utilizes oxygen is called **aerobic metabolism**. The above equation is the balance of the much abbreviated Krebs cycle. Any unsettled memories bubbling up? The point is that oxygen when combined with glucose produces *a substantial amount of energy*.



Note that ATP is the primary energy molecule for the body. Virtually every activity - thinking, movement, cardiac contraction, protein formation, etc. - requires ATP. Without a continuous production of ATP, each of these processes would cease.

Aerobic metabolism has by-products of water (H₂O) and carbon dioxide (CO₂). Water we can definitely use. In fact, about $\frac{2}{5}$ of body fluids come from aerobic metabolism, from what is burned (or oxidized) rather than what is drank. And carbon dioxide is readily breathed off at about 20 times the rate that oxygen diffuses into the bloodstream. Aerobic metabolism is incredibly efficient and effective.

Sufficient cardiac output is necessary to deliver adequate supplies of oxygen and nutrients (glucose) to the tissues. This translates to the conclusion that *cardiac output is directly related to energy production*. Low cardiac output will reduce energy levels.

For example, if your cardiac output fell to 3500 ml (about $\frac{2}{3}$ of normal) your oxygen - and hence your energy supply - would be decreased as well. Your brain with $\frac{1}{3}$ less energy may be less sharp, confused or even unconscious. Your muscles with $\frac{1}{3}$ less energy would feel weaker. In contrast, high cardiac output satisfies periods of high energy demand.

Anaerobic Metabolism

When energy demands surpass the supply of vital energy precursors such as oxygen, cells are left with the much less efficient anaerobic energy production - metabolism without oxygen. An insufficient supply of oxygen can occur due to hypoxia, obstructed blood vessels, anemia or low cardiac output conditions.

Anaerobic metabolism is not an efficient energy producer.



ONLY 2 ATP

Aerobic metabolism is clearly superior to anaerobic metabolism with regards to energy production. Anaerobic metabolism yields only 2 ATP. Also the production of acid (lactic acid) can alter the acid-base balance and hamper several vital intercellular chemical reactions.



Anaerobic metabolism can buy some time for activities that occur sporadically (i.e. sprinting or weight lifting). Anaerobic metabolism does not produce enough ATP to sustain the viability of cells that are engaged in rhythmic or continuous activity (i.e. myocardial cells).

We have all experienced the effects of anaerobic metabolism after over-engaging in a strenuous activity. The next day our muscles are painful. No, not stairs! Our blood vessels simply delivered insufficient amounts of oxygen and nutrients to satisfy the needs of these muscles. The muscles turned to anaerobic metabolism to boost the ATP supply. As a result, lactic acid accumulated in our tissues.

Ischemia

Anaerobic metabolism becomes increasingly important during periods of ischemia. **Ischemia** results from an inadequate blood flow that fails to meet the oxygen demands (energy demands) of tissues. If tissues are subject to ischemia, they try to compensate by extracting more oxygen from the blood. Tissue groups such as muscle or the intestines typically use only a third of the oxygen available to them.

The heart is the exception, extracting about $\frac{3}{4}$ of the oxygen available to it through the coronary arteries. Because the heart does not have an abundance of extra oxygen available, it is extremely dependent on blood flow for sufficient oxygenation. With increased oxygen demand, the coronary arteries must dilate to increase this blood flow.

Table 1.1 Oxygen Extracted from Various Organs While The Body is at Rest

Organ	Extracted O ₂ as Percentage of O ₂ Available
Heart	75%
Kidney	20%
Skeletal Muscle	30%
Intestine	35%
Skin	8%

Note that the heart extracts most of the available oxygen from the blood even during periods when the body is at rest. The heart, then, has very little physiological reserve to respond to episodes of high energy demand. Rather, the heart depends almost entirely on increased coronary blood flow to satisfy high energy demand.

Low cardiac output can cause cardiac ischemia - perhaps more so for the heart than other organs because of the heart's already high rate of oxygen extraction (see Table 2.1). A vicious cycle ensues. Cardiac ischemia forces a shift towards anaerobic metabolism (2 ATP) from the much more efficient aerobic metabolism (36 ATP). With less energy available and increased intercellular acidity, the force of contraction weakens, causing a further reduction in stroke volume and cardiac output.

The bottom line is that cardiac output is intimately coupled with energy production. For the heart, low cardiac output may in turn cause ischemia. Cardiac ischemia weakens contractility, further impacting cardiac output. When caring for patients with cardiac ischemia, assess for signs and symptoms of poor cardiac output (shock).

For patients experiencing shock states, look also for cardiac ischemia. Cardiac ischemia and poor cardiac output states often occur simultaneously. These conditions can cascade further by causing various dysrhythmias (see chapter 5, Arrhythmogenesis). Poor cardiac output tends to cause an increase in catecholamines (i.e. norepinephrine), which, combined with cardiac ischemia, can trigger serious dysrhythmias such as ventricular tachycardia and ventricular fibrillation.

Flash Quiz 1.1

1. The contractile phase of the cardiac cycle is called _____. The relaxation phase of the cardiac cycle is called _____.
2. The right heart delivers (oxygenated, deoxygenated) blood to the (pulmonary circulation, systemic circulation).
3. An average cardiac output at rest is:
 - a) 3 litres
 - b) 4 litres
 - c) 5 litres
 - d) 10 litres
4. Heart valves ensure the forward flow of blood through the heart.

True or False

5. Cardiac output is the amount of blood ejected by the (atrium, ventricle) over (1 heart beat, 1 minute).

Answers:
1 minute

1. systole, diastole; 2. deoxygenated, pulmonary circulation; 3. c); 4. True; 5. ventricle,

6. Without atrial kick, cardiac output typically falls by:

- a) 5-10%
- b) 15-35%
- c) 50%
- d) 90-100%

7. Cardiac output is intimately connected to the body's ability to produce energy. A fall in cardiac output usually brings a fall in energy production.

True or False

8. Aerobic metabolism produces several adenosine triphosphate (ATP) energy molecules. How many ATP are produced from one glucose and one oxygen molecule?

- a) 2
- b) 12
- c) 24
- d) 36

9. By-products of aerobic metabolism include (circle all that apply):

- a) lactic acid
- b) water
- c) nitrogen
- d) carbon dioxide
- e) hydrogen peroxide

10. Which of the following tissue groups extract about 3/4 of the available oxygen from the blood supplied even while the body is at rest?

- a) heart
- b) skin
- c) skeletal muscles
- d) intestines
- e) skin
- f) brain

Answers: 6. b); 7. True; 8. d); 9. b), d); 10. a)

Parameters that Affect Cardiac Output

Cardiac output is the amount of blood ejected by the heart in a minute - the product of stroke volume and heart rate. Sufficient cardiac output is necessary to sustain life. Let's look further into the parameters affecting cardiac output.

Heart Rate

Generally speaking, heart rate and cardiac output have a direct relationship. As heart rate increases, so does cardiac output. As mentioned earlier, as energy demands grow (oxygen demands), cardiac output increases in kind. A heart rate of 100/minute will almost always result in more blood ejected per minute than a heart rate of 80/minute. Take a person with an average stroke volume of 65 ml.

$$\text{Heart Rate of 80/minute: CO} = \text{SV} \times \text{HR} = 65 \times 80 = \mathbf{5200}$$

$$\text{Heart Rate of 100/minute: CO} = \text{SV} \times \text{HR} = 65 \times 100 = \mathbf{6500}$$

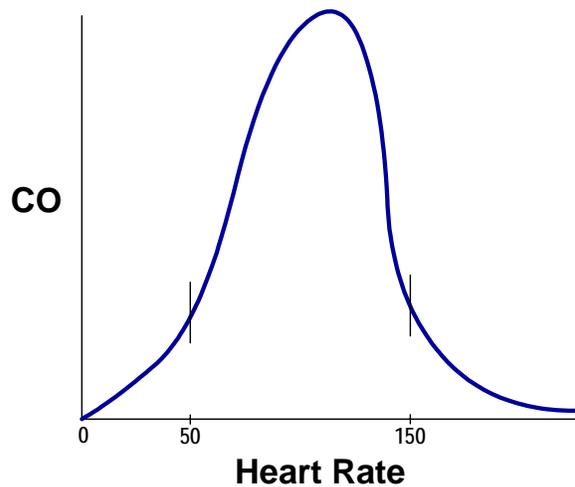
With this simplistic example, a 20% increase in heart rate (from 80 to 100/minute) yields a 20% increase in cardiac output (from 5200 ml to 6500 ml).



More realistically, stroke volume might also increase because catecholamine stimulation of the heart results in an increase in *both* heart rate and stroke volume. As a result, an increase in heart rate by 20% tends to increase cardiac output by more than 20%.

There is a definite limit to this logic. Heart rates of 260/minute are usually associated with signs and symptoms of shock, with a corresponding poor cardiac output. In fact, heart rates of more than 150/minute are often associated with a reduced cardiac output.

Why? Recall the importance of diastole in the cardiac cycle? During diastole, the blood is drawn into the ventricle. This takes time, referred to as “filling time.” Not too original a term but a very important parameter of cardiac output. Without an adequate **filling time**, the ventricle receives less blood. With less blood volume, stroke volume and cardiac output falls.

Figure 1.2 Cardiac Output and Heart Rate

This graph illustrates the relationship between heart rate and cardiac output. As heart rate increases, so does cardiac output - to a point. Cardiac output tends to fall when heart rate surpasses 150/minute due to inadequate filling time. Low cardiac output states also occur with low heart rates (<50/minute). Of course, this graph represents a significant generalization. Young and athletic people can have good cardiac outputs with heart rates greater than 150/minute and less than 50/minute. Those with cardiac disease often cannot tolerate heart rates as low as 50/minute or as high as 150/minute.

Conversely, if the heart rate is too low - say below 50/minute - cardiac output falls quickly. With slow heart rates (bradycardias) we certainly have adequate filling time. The ventricles have all the time they need to fill to the brim. Stroke volume is quite good. The problem is that there isn't a sufficient heart rate.

Another example is in order here. Let's continue with Henry. As Henry ages gracefully, unfortunately his sinus node begins to fail with a junctional escape rhythm resulting of only 40/minute. This long filling time might increase his stroke volume to 80 ml.

$$\text{CO} = \text{SV} \times \text{HR} = 80 \times 40 = \mathbf{3200 \text{ ml/minute}}$$

A cardiac output of 3200 could leave Henry feeling quite unwell.



As a general rule, **a patient with a heart rate that is too fast** (>150/minute - not enough filling time) **or too slow** (< 50/minute - not enough rate) **requires urgent assessment for signs and symptoms of shock**. Both extreme rates can be associated with inadequate cardiac output. Signs and symptoms of shock include shortness of breath, chest pain, hypotension, and an altered level of consciousness (due to hemodynamic compromise).

As a general rule, closely monitor patients with rates more than 150/minute **or** less than 50/minute for signs and symptoms of poor cardiac output. Exceptions do exist. For example, peak performance athletes have very efficient, larger hearts with higher resting stroke volumes than the average population. A stroke volume of 100/minute and a heart rate of 50/minute would yield an acceptable cardiac output of 5 liters.

On the other side of the continuum, patients with a significant cardiac history (i.e. myocardial infarction and/or congestive heart failure) may have a low stroke volume. Heart rates as high as 150/minute may be associated with cardiac ischemia and reduced cardiac output. A bradycardia of 50/minute combined with an already reduced stroke volume (i.e. 40 ml) could result in shock with a cardiac output of only 2000 ml!

The more pronounced a patient's history of cardiac illness, generally the narrower is the range of heart rates that yield sufficient cardiac outputs. Most of us have met the patient who becomes short of breath with minimal exertion i.e. walking to the bathroom. These patients are often restricted to limited activities due to a narrow range in acceptable heart rates that yield sufficient cardiac outputs (i.e. 65-100/min). For this patient, a heart rate over 95/minute could cause a drop in cardiac output.

Heart rate is an important factor in any physical assessment, as is collecting a cardiac history. The seriousness of a cardiac rhythm is intimately connected with each.

Stroke Volume

While heart rate is an undisputed contributor to cardiac output, stroke volume is the other major player. As heart rates vary to changes in cardiac output demand, so does stroke volume. Stroke volume - the amount of blood ejected with each beat - fluctuates with changes in preload, afterload, and catecholamine release.

Preload

The blood supply to the ventricle is often referred to as **preload**. Technically, the definition of preload is the volume or pressure in the ventricle at the end of diastole. Note that atrial kick offers much to preload, especially for those getting on in years (contributing up to 35% of cardiac output). Preload is connected to stroke volume and cardiac output via the Frank-Starling law.



Related to stroke volume is the term 'ejection fraction'. An **ejection fraction** is determined by an echocardiogram or via a pulmonary artery catheter. Ejection fraction is the percentage of volume ejected from the left ventricle. The left ventricle has about 100 ml of blood just before contraction. Of this 100 ml, about 50-80 ml is normally ejected from the heart with each beat (stroke volume). Therefore, about 50 to 80 percent of blood is ejected. This is a normal ejection fraction.

Most of us have heard of the Frank-Starling phenomenon (often referred to as **Starling's Law** - Frank has somehow been left out over the years). Frank and then Starling demonstrated that as cardiac muscle fibers stretch, contraction becomes more forceful. In other words, the more the stretch of the heart's chambers, the more forceful the contraction (and indeed the greater the stroke volume).

What causes the heart's chambers to stretch? Blood filling into the chambers increase pressures causing fibers to stretch. Whether you refer to increased pressure or volume in a chamber as the cause of the stretch is probably not important. The key is that either way, you are referring to preload. More preload causes more cardiac fiber stretch and increased contractility.

Please refer to Figure 2.3: The Frank-Starling curve on the next page. The resting healthy heart depicts the varying contractility of the myocardium with respect to changes in ventricular end diastolic pressure (preload).

The slope of each curve is the key to this graph. Compare the healthy resting heart to the curves of both the diseased heart and the heart during strenuous activity. Notice how the effect of sympathetic stimulation (i.e. norepinephrine) during exercise results in a magnified effect of preload on contractility.

Compare the preload/contractility curve of the healthy heart with that of the diseased heart. While the healthy heart curves peak with a preload of about 12 mm of Hg, the diseased heart requires increased pressures to maximize contractility. The diseased heart depends more on preload than the healthy heart to drive an effective contraction.

Note that the higher the preload, the higher the myocardial workload. Therefore, high preload states (i.e. fluid overload) can make matters worse during ischemic episodes. And ischemia is one precursor to the development of serious dysrhythmias.

Figure 1.3 Frank-Starling Curve

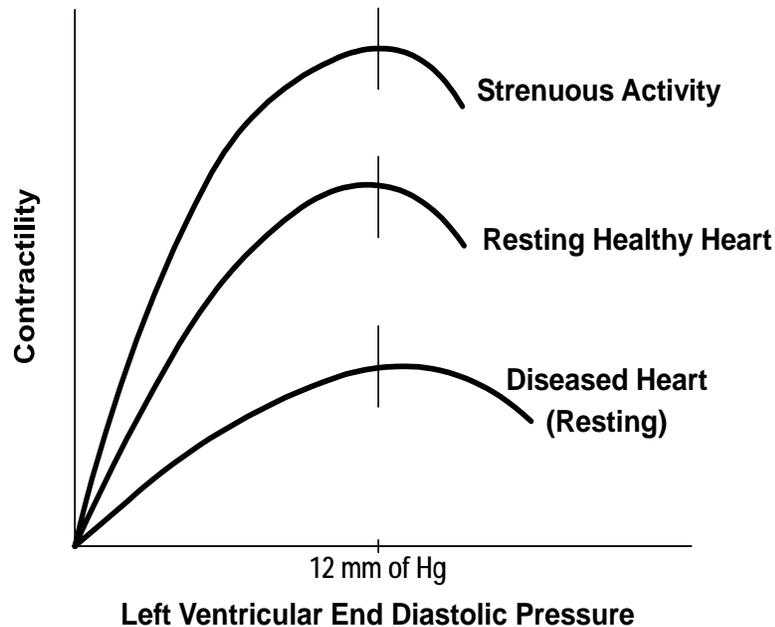
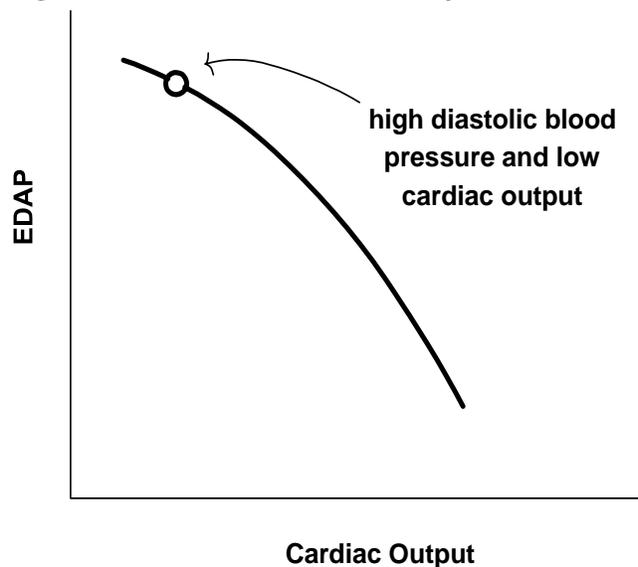


Figure 2.3 depicts the relationship between ventricular end diastolic pressure and contractility for a resting healthy heart, a resting diseased heart and a healthy heart during strenuous activity. Several points are evident here: **1**) in general, the force of contraction (contractility) increases as the pressure within the ventricles increase (increases in pressure and volume increase both cardiac fiber stretch and contractility); **2**) during strenuous activity, catecholamine release increases the force of contraction; **3**) for the diseased heart (i.e. cardiomyopathies), the force of contraction is impaired; **4**) increases in chamber pressure do not produce significant changes in contractility for the diseased heart; and **5**) there is a limit to the affect of ventricular end-diastolic pressures (VEDP) on contractility. With high VEDP, contractility begins to fall. In other words, with high VEDP, contractility and stroke volumes tend to decrease.

Afterload

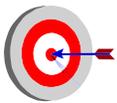
The resistance to the ejection of blood by the ventricle is called **afterload**. The left ventricle, for example, must create sufficient pressures during systole to overcome diastolic arterial pressure and systemic vascular resistance before any blood is ejected. While preload enhances contractility and stroke volume, high pressures in the *arterial* vessels during ventricular end diastole is inversely related to stroke volume (see Figure 2.4 on the next page).

While systemic vascular resistance is not easily determined without a pulmonary artery catheter, diastolic blood pressure is easily measured. So while an accurate estimate of afterload is often not clinically practical, a patient's diastolic pressure provides a good indication of the resistance the left ventricle must overcome (afterload). In general, the higher the diastolic pressure, the higher the afterload.

Figure 1.4 Afterload and Cardiac Output

As the resistance to the ejection of blood from the left ventricle increases, stroke volume tends to decrease as does cardiac output. Perhaps as important, cardiac workload increases with increases in afterload.

And the higher the afterload, the more difficult a job it is for the left ventricle to eject sufficient stroke volumes. Similar to preload, increased afterload causes increased myocardial workload, a factor to consider for those with advanced cardiac disease and/or cardiac ischemia.



The explanation for the walls of the left ventricle being three times the thickness of the walls of the right ventricle rests squarely with the concept of afterload. At birth, the wall thickness of the right and left ventricle are equal. Soon after birth, though, the pressures in the systemic circulation begin to surpass those of the pulmonary system. The lower pressures (typically about 24/8 mm Hg) of the pulmonary system mean a lower afterload for the right ventricle than the left ventricle. As a result, the muscle mass required of the right ventricle is also less than the left ventricle.

Afterload is also tied to cardiac hypertrophy. 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.

Applying Concepts of Cardiac Output Regulation

Cardiac output is a product of heart rate and stroke volume. We established that cardiac output (CO) is intimately tied to energy production. Many factors influence stroke volume: atrial kick, preload, afterload, filling time, Frank-Starling's Law, catecholamine stimulation and coronary ischemia. We also arrived at the conclusion that aerobic metabolism is quite preferable to anaerobic metabolism.

Table 1.2 Parameters That Affect Cardiac Output

Parameters that Increase Cardiac Output	Parameters that Reduce Cardiac Output
Heart rates between 50/minute and 150/minute*	Heart rates less than 50/minute or more than 150/minute*
Atrial kick	Lack of atrial kick
Adequate filling time	Inadequate filling time
Frank-Starling law - more myocardial stretch	Frank-Starling Law - less myocardial stretch
Increased preload (to a limit)	Reduced preload (to a limit)
Low afterload	High afterload

** As mentioned earlier, this heart rate range is a generous generalization. Variations in this range are person-specific. Athletes often enjoy a wider range while those with cardiac disease tend to have a narrower effective heart rate range.*



Heart rate and contractility are influenced by sympathetic innervation of the heart. Sympathetic innervation which releases epinephrine and norepinephrine, influences cardiac output through its alpha effect (peripheral vasoconstriction) and its beta 1 effect (increases heart rate and force of contraction). The alpha effect provides more preload by shunting blood to the core organs (including the heart). While the alpha effect can also increase afterload, sympathetic stimulation usually boosts cardiac output.

A case study might help to bring some life to these concepts.

Case: Hank, a 56 year old man, arrives in the emergency department via ambulance. He is pale and diaphoretic, reporting crushing chest pain. He is connected to a cardiac monitor, an intravenous access is started and oxygen is applied via nasal prongs at 4 liters/minute. A 12 lead ECG reveals that he is experiencing an anterolateral acute myocardial infarction (AMI).

1. An anterolateral AMI primarily affects which heart chamber? What coronary arteries serve this chamber? (answers below)

Vital signs are taken. While a brief history is taken, a children's aspirin is given for Hank to chew.

HR = 100/minute

BP = 160/110

RR = 26/minute

O₂ saturation = 95%

Hank has a history of angina and has been taking propranolol and a daily nitro patch. A recent angiogram showed 85% occlusion to his left anterior descending artery (LAD), 55% occlusion to his right coronary artery (RCA) and 60% occlusion to his circumflex artery. Findings from an echocardiogram done a month ago showed Hank had an ejection fraction of 55%. He is usually normotensive.

2. Would a blood pressure of 160/110 be optimal at this moment?

A blood pressure of 160/110 is not uncommon with an AMI. An abundance of sympathetic stimulation causes peripheral vasoconstriction, increased systemic vascular resistance (SVR) and often a higher blood pressure. Unfortunately, the high diastolic pressure also means a high afterload for the left ventricle.

Meanwhile, the left ventricle is currently under attack from ischemia. Most likely, the contractility of the left ventricle is impaired. A high afterload will only further reduce the pumping effectiveness of the left ventricle. As afterload increases, so does the workload and oxygen demand of the left ventricle. A reduction in afterload is a worthy treatment objective at this time.

Metoprolol IV, Nitroglycerin spray, and Morphine IV are administered.

Beta blockers (metoprolol and atenolol are the most commonly prescribed), nitroglycerin and morphine can reduce both preload and afterload. Beta blockers are very beneficial in reducing both morbidity and mortality of those having an AMI (25-40% reduction). Beta blockers reduce both heart rate and contractility. These dual

Answers: 1. left ventricle; left main, left anterior descending and circumflex coronary arteries 2. No. A diastolic pressure of 110 is high, representing a high afterload, potentially impairing stroke volume and increasing both myocardial workload and myocardial oxygen demand

actions reduce myocardial workload. Beta blockers limit the catecholamine stimulation of the heart and effectively decrease the incidence of troublesome dysrhythmias.

Hank's blood pressure comes down to 130/90. His lungs are auscultated. Crackles are heard to his bases bilaterally. This is a new finding.

3. Why are Hank's lungs wet?

A region of Hank's left ventricle is infarcting. The infarcted (dead) tissue has ceased to contract at all. Around this infarct zone is an ischemic zone (the penumbra) which is not able to contract optimally. The result - compounded by a high afterload - is a reduced stroke volume. Before this AMI, Hank could quite comfortably pump about 55% of the blood from his left ventricle (ejection fraction). Not now.

For the sake of this example, let's say that Hank's ejection fraction has been reduced to 35%. This would mean that his stroke volume would be about 35 ml. But what about the pumping ability of his right ventricle? It has not been damaged. It can most likely maintain a 55% ejection fraction. Picture the right ventricle pumping out 55 ml with each beat while the left ventricle is able to only pump out 35 ml. Hank has a serious mismatch problem. This is known as left-sided heart failure.

Hank has too much blood supply for his left ventricle, otherwise known as too much preload. Blood volume collects within the pulmonary vessels, increasing hydrostatic pressure. Elevated pressures in the pulmonary circulation can result in fluid being pushed into the alveoli. Crackles to the lung bases soon become audible.

Cardiac management should then include reducing his preload. By lessening Hank's blood volume (and the blood return to the heart), the right ventricle's preload will also fall. This, in turn, decreases both the stretch of the right ventricle and its force of contraction (Frank-Starling law). The goal: a more evenly matched right and left stroke volume.

Lasix IV, Morphine and Nitroglycerin are administered.

Note that Lasix reduces fluid volume through diuresis. Lasix, morphine and nitroglycerin also cause vasodilation, shifting more blood to the periphery and away from the heart to reduce preload.

4. Why is Hank's heart rate at 100/minute?

It is no surprise that Hank's heart rate sits at 100/minute. First, he definitely has an abundance of epinephrine circulating due to both the pain and the fear he is experiencing. From a CO perspective, if his heart rate remained at 80/minute, his CO would have plummeted to only 2800 ml ($80/\text{minute} \times 35 \text{ ml} = 2800 \text{ ml}/\text{minute}$), more than a third less than his resting cardiac output.

A heart rate of 100/minute helps to maintain an acceptable CO. Positioning Hank in semi-fowlers position further reduces the preload to his heart by using gravity i.e. blood pools in the abdomen and lower extremities rather than near his heart.

Hank's blood pressure is now 130/80. His pain has lessened. He receives a second IV to prepare for thrombolytics. Blood work is drawn. Oxygen saturations increase from 95% to 98% as the crackles to his lung bases resolve.

Much of his care revolves around 2 simple objectives:

INCREASE OXYGEN SUPPLY AND REDUCE OXYGEN DEMAND.

Hank recovers from this event. His ejection fraction will probably never return to its pre-infarct value. His resting cardiac output is lower now than before his AMI. As a result, he may have less energy for daily activities. He continues to take lasix twice daily and restricts his fluids intake. Hank must now adjust to living with poor left ventricular function.



As a general rule, a patient experiencing a left ventricular infarction - anterior, lateral or anterolateral MI - should be managed with particular attention to preload. Fluids should be administered cautiously. Medications that reduce preload and afterload can be very therapeutic: nitroglycerin, morphine and lasix for example. Also, routinely assess for left ventricular failure: lung congestion, falling blood pressure, increased breathing rate and falling oxygen saturations.

This case study reveals how the medical management of cardiac output parameters is vital for a person experiencing cardiac ischemia. Note that aspirin, beta blockers and thrombolytics are the three pillars in the treatment of most AMI events.

Answers: 4. *sympathetic response to pain and fear; a falling stroke volume is often compensated by an increased heart rate to maintain an acceptable CO*

Summary

In this chapter we began laying the ground work necessary to effectively manage cardiac emergencies. Understanding the heart's dynamics and its role in maintaining homeostasis often draws the conclusion, "It's all about cardiac output".

The cardiac cycle and the regulation of cardiac output was explored. Energy production is directly tied to blood (oxygen and nutrients) supply. Low cardiac output often results in insufficient energy production. The effective and efficient aerobic metabolism (using oxygen and producing 36 ATP) is replaced with anaerobic metabolism (without oxygen and only 2 ATP produced) during periods of ischemia.

The amount of blood pumped to the body each minute is called cardiac output. Cardiac output is a product of how much blood the left ventricle pumps with each contraction (known as stroke volume) and heart rate.

A number of factors govern cardiac output. The more the heart's muscle fibers stretch, the more forceful the contraction (more blood = more stretch = more pumped out with each beat). This is called Frank-Starling's Law. Catecholamine stimulation (sympathetic nervous system and the adrenals) increases both stroke volume and heart rate to increase cardiac output.

Three conditions impact blood flow to the ventricles. The more time provided for filling the ventricles (diastole or filling time) results in more blood in the chambers. Also, the greater the blood supply that is returning to the heart (preload), the faster the chambers will fill. Atrial kick tops up the ventricles, accounting for 15-35% of cardiac output.

Generally rates of 50-150/minute are associated with an acceptable cardiac output. Heart rates of less than 50/minute provide sufficient stroke volume but often an insufficient heart rate results in poor cardiac output. Rates of greater than 150/minute provide rapid heart rates but insufficient filling times and poor stroke volume.

Cardiac disease most often involves the parameters that govern cardiac output. For example, chronic afterload causes chamber enlargement and possibly even heart failure. Atrial fibrillation can reduce cardiac output by as much as 35% with the loss of atrial kick. Increased catecholamine release, increased preload and afterload exacerbates cardiac ischemia.

Being aware of the dynamics of cardiac output enhances your ability to recognize and respond acute cardiac events.

Chapter Quiz

1. Increased preload usually corresponds to increased contractility (force of contraction).

True or False

2. A typical stroke volume for a healthy adult is:

- a) 15-35 ml
- b) 35-50 ml
- c) 50-80 ml
- d) 80-110 ml

3. During periods of ischemia, cells must turn to anaerobic metabolism. With anaerobic metabolism, energy produced from a glucose molecule is only: (2, 12, 24, 36) ATP.

4. Pressure within the ventricle must overcome the arterial diastolic pressure before the semilunar valves open and blood is ejected.

True or False

5. An increase in afterload tends to increase stroke volume and cardiac output.

True or False

6. Cardiac ischemia can cause (circle all that apply):

- a) a decrease in contractility
- b) decrease in energy production
- c) increased intercellular acidity
- d) dysrhythmias
- e) all of the above

7. Acidosis impairs intercellular chemical reactions, potentially leading to cellular death.

True or False

Answers: 1. True; 2. c); 3. 2 ATP; 4. True; 5. False; 6. e); 7. True

8. Patients with heart disease will most likely hemodynamically tolerate heart rates below 50/minute and above 150/minute.

True or False

9. Which of the following factors tend to increase cardiac output? (Circle all that apply)

- a) gradually increasing heart rates up to 150/minute
- b) presence of atrial kick
- c) increased preload
- d) increased afterload
- e) decreased preload
- f) decreased afterload
- g) heart rate of 40/minute that allows for increased ventricular filling time

10. Cardiac ischemia and catecholamine stimulation is often a lethal combination, causing serious dysrhythmias such as ventricular fibrillation and ventricular tachycardia.

True or False

11. Beta blockers therapy is commonly used for those experiencing an acute myocardial infarction. Beta blocker therapy have several theoretical benefits such as (circle all that apply):

- a) decrease preload
- b) increase afterload
- c) reduce myocardial oxygen demand
- d) reduce heart rate
- e) decrease contractility
- f) limit catecholamine stimulation of the heart
- g) antiarrhythmic properties

12. Rapid heart rates can cause a low cardiac output due to insufficient _____ which significantly reduces _____. Overly slow heart rates have long ventricular filling times and adequate stroke volumes but not enough _____.

13. An acute anterior myocardial infarction can result in left sided heart failure. Treatment is often directed at:

- a) reducing afterload
- b) reducing preload
- c) increasing afterload
- d) increasing preload

Case Study for Questions 14-20: John is a 84 year old man who arrives in the emergency department with shortness of breath and vomiting. His oxygen saturations are 95%, heart rate is 90/minute, breathing rate is 26/minute and blood pressure is 110/70 mm Hg. John is visibly anxious. A 12 lead ECG is taken.

The findings of the 12 lead ECG point to an inferior myocardial infarction. Since the 12 lead provides a good view of the left heart but not the right heart, a 15 lead ECG (3 more leads over the right side of the chest and the back) is done. The 15 lead ECG confirms that John is experiencing a right ventricular infarction.

14. Larger myocardial infarctions usually cause a reduction in stroke volume from pre-infarction values. How would a large right ventricular infarction (RVI) affect the preload (blood supply) to the left ventricle?

- a) reduce preload
- b) increase preload
- c) no effect on preload
- d) none of the above

15. Should medications such as morphine, lasix and nitroglycerin be routinely administered to John?

Yes or No

16. Large right ventricular infarctions often are associated with low blood pressures. This hypotensive state is best treated by:

- a) inotrope medication (increase the contractility of the heart)
- b) reducing afterload
- c) reducing preload
- d) fluid bolus intravenously

17. If a 500 ml fluid bolus was given to John, this would (increase, decrease) his preload. This would have an effect on the right ventricle explained by the Frank-Starling law as (increasing, decreasing) myocardial fiber stretch and (increasing, decreasing) the stroke volume of the right ventricle.

Answers: 13. b); 14. a); 15. No; 16. d); 17. increase, increasing, increasing

18. The hemodynamic management of left and right ventricular infarctions is identical.

True or False

19. Since John remains normotensive with a blood pressure of 110/70 mm Hg, he (would, would not) benefit from beta blocker therapy. Since beta blockers also reduce contractility, this (is, is not) an important consideration when prescribing beta blockers for those with a right ventricular infarction.

20. The 12 lead ECG has a vital role to play in the diagnosis and hemodynamic management of myocardial infarctions.

True or False

Suggested Reading and Resources



Alexander, W. et al. (2001). *Hurst's the Heart*. 10th ed. New York: McGraw-Hill

Cardiac Output. Web: <http://www.ebme.net/arts/cardop/>

Cardiac Output: Ever Wonder What Those Numbers Really Mean? Web: <http://mededcon.com/card01.htm>

Katz, A.M. (2001). *Physiology of the Heart*. 3rd ed. London: Lippincott

Linappa, V. & Farey, K. (2000). *Physiological Medicine*. New York: McGraw-Hill

What's Next?

Understanding cardiac output parameters is necessary to make sense of acute cardiac events. The next two chapters switch gears from recognizing and understanding cardiac emergencies to the interventions commonly employed in the management of acute cardiac events. Chapter 2 outlines the rationale and the step-by-step procedures of electrical interventions. Chapter 3 covers the equipment and techniques of airway management.