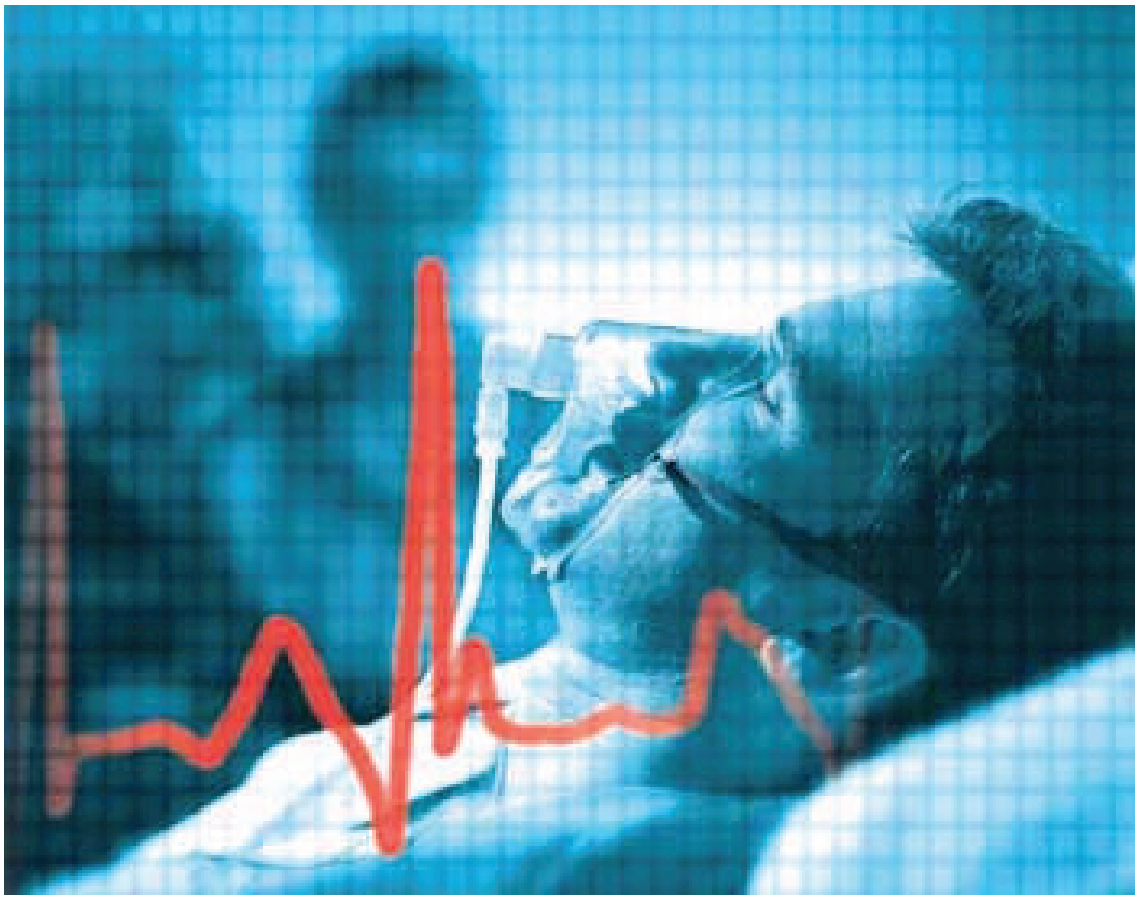
The Six Second ECG



A Practical Guide to Basic and 12 Lead ECG Interpretation

Tracy Paul Barill

The Six Second ECG

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Contents At a Glance

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Introduction

The ability to correctly interpret an electrocardiogram (ECG), be it a simple six second strip or a 12 lead ECG, is a vital skill in all critical care areas. Of all critical care skills, meaningful ECG interpretation may be the one skill that sets critical care practitioners apart from other clinicians.

The Six Second ECG is a practical guidebook designed for you to quickly and effectively interpret ECGs. Delivered in a no-nonsense candid style, each chapter builds on previous chapters. A simple and effective framework is presented that enables you to not only identify ECGs but to make sense of the ECG from a clinical perspective.

While ECG interpretation is well covered in several books, **The Six Second ECG** is unique in its persistent attention to the connections between ECG interpretation, cardiac physiology and clinical significance. A skilled practitioner connects the findings of an ECG to a patient's clinical condition and uses this information to decide upon an appropriate treatment strategy.

This Book is For You

If you want to *quickly identify* and to readily *make sense* of an ECG from a physiological and clinical perspective, the Six Second ECG is written for you. With an unwavering focus on understanding, the Six Second ECG is designed to help build career-long skills of ECG interpretation. Your time is far too precious to waste on imminently forgotten memory work.

It is, after all, the pattern on the fabric that holds the interest of most of us, rather than the threads.

Dr. Arnold M. Katz

How to Use This Book

This book is designed for the busy health care professional, one who needs to quickly address any informational gaps with the least amount of fuss. An expanded table of contents and index facilitates rapid navigation. Each chapter begins and ends with a chapter summary. As well, a "Quick Look" navigator appears on the first page of each chapter to facilitate a quick and focused reference to specific topics of interest.

Each chapter is independent and can stand on its own. Read the book from cover to cover or jump around concentrating on what you need. Choose to complete the exercises and quizzes inside each chapter. Answers to the quizzes are provided at the base of the pages that contain the questions. Detailed annotated answers are provided in Appendix B.

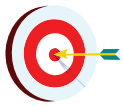
For the like-minded keener whose curiosity in the area of cardiology is almost insatiable, a list of additional resources are included at the end of each chapter. Several resources are freely available on the web. An abundance of resources are also included in the provided CD-ROM for off-line use.

Certain conventions such as the use of icons and gray text boxes have been used throughout the book to draw attention to tips, trivia, details and important points.

The 'stop' hand signal marks vital information often related to clinical practice.

The symbol of a string tied around the index finger is used as a reminder.

The icon of a magnifying glass marks supplementary explanations on various topics.



A symbol of an arrow on target signifies tips, trivia, and useful short-cuts.

Synonymous with the internet, this icon marks any supplemental resources.

Brief Synopsis

The Six Second ECG follows the order of a workshop of the same name. Seven chapters make up its contents. Over 140 illustrations and 300 quiz questions help to clarify the core content. A brief synopsis of the chapters and appendices follow.

Chapter 1: Chambers, Valves and Vessels is a brief account of the heart's anatomy.

Chapter 2: It's All About Cardiac Output is an introductory discussion on the dynamics of the heart as an effective pump. Concepts of the cardiac cycle and the parameters that determine cardiac output are brought together in case studies.

Chapter 3: The Electrics outlines the electrical pathways of the heart. Understanding the electrophysiology of the heart is a necessary foundation to make sense of an ECG.

Chapter 4: An ECG Primer introduces the cardiac monitoring system: ECG paper, the basic components of an electrocardiogram and methods to determine heart rate.

Chapter 5: In Four Simple Steps provides a step-by-step method for rapid ECG interpretation. In just four simple steps, systematically identify an electrocardiogram. Beyond just ECG interpretation, make sense of each ECG rhythm using several indicators from the ECG that potentially point to hemodynamic compromise. Practice exercises reinforce rapid ECG interpretation.

Chapter 6: The 12 Lead ECG reveals the advantages of multiple ECG lead views. Building on steps already established in earlier chapters, a simple method of 12 lead ECG interpretation is established. The primary use of the 12 lead ECG - to detect cardiac ischemia and infarction - is explored.

Part IV: Appendices

Appendix A: Glossary of Terms is a quick reference defining terms mentioned throughout this book.

The Author

I have been a critical care practitioner and educator for the past 22 years. My clinical experience - like many other critical care nurses - spans intensive care units, coronary care units, emergency rooms and flight nursing. I seem to have an insatiable curiosity for the cardiac domain compounded by a strong will to share this knowledge as an educator.

I have been privileged to travel to many regions of United States and Canada facilitating hundreds of ACLS courses, basic ECG and advanced ECG interpretation courses. Much of what I have learned is taken from discussions with talented course participants and fellow instructors.

I also develop web- based learning tools for health care professionals to augment skills learned in the classroom (found at www.skillstat.com). SkillStat's Six Second ECG simulator has been downloaded by more than 7 million health professionals.

Acknowledgements

This book would not have been possible without the invaluable suggestions of several colleagues. In particular, I am particularly grateful for the contributions of Michael Dare, Gaynor Burns, Cecelia L. Crawford, and SkillStat Learning Inc.. Their proofing and editing have greatly helped to shape this work. Any errors in grammar, spelling or content do not occur from any lack of effort, but remain my shortcomings.

The ongoing patience, support and encouragement from Janet, my spouse and our two sons -Kieran and Shane - have greatly helped this project go the distance.

Much of the book's content was formed during Six Second ECG workshops for nurses, medical students, paramedics, respiratory therapists and physicians. Thank you all for the many lessons you have taught me over the years. I hope that you recognize your feedback in these pages.

Let's Get Started!

This book was written to be straightforward and easy to read. Much effort has gone into eradicating errors in spelling, grammar and content. I expect that some may have snuck through, nevertheless. I greatly appreciate all feedback, corrections and questions via e-mail (ssecg@skillstat.com).

I sincerely hope that you grow in competence and confidence in ECG interpretation whether you are a novice or an experienced practitioner.

Tracy Barill

North Vancouver, British Columbia, Canada

1

Chambers, Valves and Vessels

Quick Look

If you could have it all with regards to electrocardiogram interpretation, what might that look like? Participants asked this question in ECG courses tend to want to quickly identify a cardiac rhythm strip competently and confidently.

But they also want to make sense of ECGs. To connect the rate, pattern and shape of the ECG with a patient's current clinical status. To recognize which cardiac rhythms are benign and which rhythms demand urgent attention. Some even want to be able to link components of an ECG to a patient's prognosis.

You can have it all. If you want to quickly identify cardiac rhythms, the last five chapters will suffice. If you want to put the whole picture together and make sense of ECGs, begin right here and work your way through. The journey's a bit longer but well worth it.

The first three chapters of this book provide the basics of cardiac anatomy and physiology. This chapter sets the stage, covering the anatomical structures of the heart. This may be just a good review. Let's begin.

In my beginning is my end.

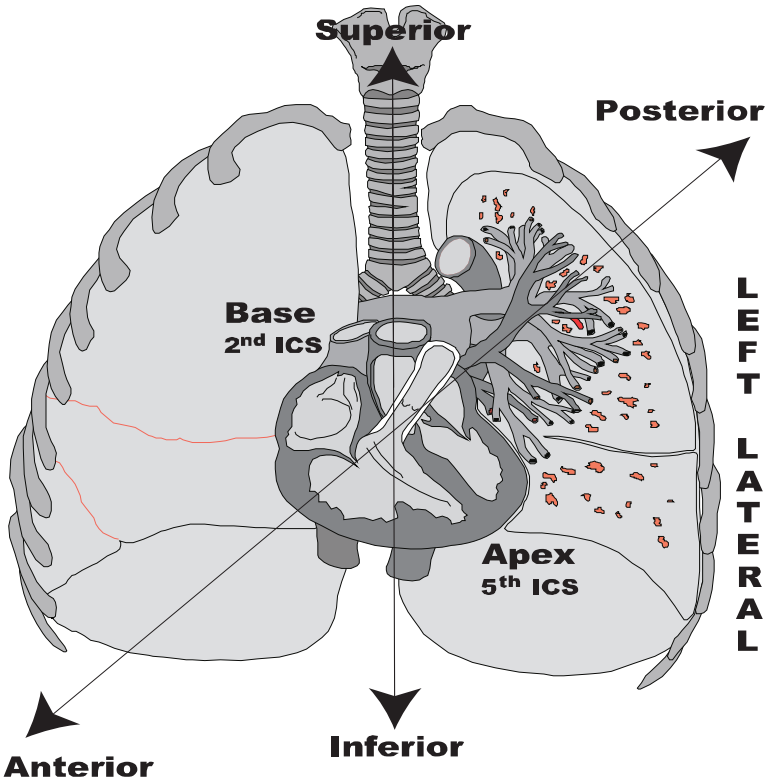
T.S. Eliot

Overview

The heart is a wondrous organ about the size of your fist, weighing in at less than a pound (about 400 grams). Each day, the adult heart beats over 100,000 times, delivering 7500 liters of blood to the tissues of the body. The heart is dynamic, ever sensitive and responsive to mechanical, chemical and electrical stimuli. It continuously fluctuates in rate and force in response to our physiologic and environmental needs.

Situated in the mediastinum directly behind the sternum, approximately 2/3 of the heart is left of the sternal border, resting on the diaphragm. The heart's apex is at the bottom of the heart pointing left near the 5th intercostal space (ICS). The base of the heart is located near the 2nd intercostal space to the right of the sternum.

Figure 1.1 Location and Views



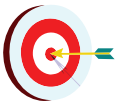
The heart is not positioned straight up. Rather, it sits on its right border (the base of the septum is pulled to the left) with the right chamber rotated anteriorly. Visualize the patient's right lateral border of the heart being pulled forward. In turn, this would bring the left border of the heart more posterior. Nevertheless, the larger left ventricle occupies the majority of the anterior, lateral and inferior surfaces of the heart.

Table 1.1 Views of the Heart

Perspectives	Heart Chambers
Anterior	Left Ventricle and Left Atrium
Left Lateral*	Left Ventricle and Left Atrium
Inferior**	Left and Right Ventricle
Right Lateral	Right Ventricle
Posterior	Left and Right Ventricle

**lateral MIs generally refer to left lateral MI*
*** only about 40% of inferior MIs are right ventricular infarctions*

So, when you are told that your patient is experiencing an anterior myocardial infarction (MI), what ventricle is most likely affected? How about an inferior MI? An anterolateral MI? Take a look at Figure 1.1 and Table 1.1.



A 12 Lead ECG provides a fairly good electrical picture of the left side of the heart. The right side of the heart is somewhat under served. If the 12 Lead suggests any pathology to the inferior view of the heart (left and right ventricle), 3 additional lead views should be added to map out the right lateral and posterior views. The resulting 15 lead ECG provides a more complete three dimensional picture of the heart.

The inferior view of the heart includes the right ventricle and the heart's apex (left ventricle). About 40% of inferior MIs are right ventricular infarctions. The anterior and lateral (left lateral) views of the heart are of the left ventricle and left atrium.

The Mechanical Structures of the Heart

The mechanical structures of the heart include the heart's layers, chambers, septum, valves, and the major vessels (including the coronary arteries). Each of these structures contribute to the effective ejection of blood - the primary purpose of the heart. The electrical components and pathways will be addressed separately in Chapter 3.

Layers

The heart is encased in two protective layers (refer to Figure 1.2 on the next page). The outer layer, the pericardial sac, covers the heart. It folds in on itself at the aorta forming the epicardial surface of the heart. Between these layers is a small amount of fluid that provides a non-stick surface between these layers.

Pericarditis, an infection within the pericardial sac, can cause increased friction between the inner surfaces of these layers. Chest discomfort is common. A friction rub, a sound similar to that produced by rubbing leather together - may also result. Note also that an accumulation of relatively small amounts of fluid (200 ml) in this pericardial sac - **pericardial effusions** - can straight jacket the heart's ability to contract. This condition called **cardiac tamponade** may result in little or no cardiac output.

The epicardium forms the outer layer of the heart. The myocardium forms the middle layer and the endocardium the innermost layer of the heart. The coronary arteries provide blood to the heart tissues, carrying blood first across the epicardium, then the myocardium and finally terminating in the endocardium.

The endocardium claims the dubious position as the terminus for the coronary arteries. Since the coronary arteries begin along the epicardial surface, enter the myocardium and terminate in the endocardium, myocardial ischemia rarely occurs without endocardial ischemia. While the endocardium is damaged in most every myocardial infarction, the epicardium's location in the blood flow hierarchy increases its safety factor.

The muscular myocardium is the thickest layer and the workhorse of the heart. It is composed of specialized muscle and electrical cells that are able to conduct an electrical impulse quickly and contract forcefully. The endocardium has a smooth inner surface to allow blood to flow easily through the heart's chambers. The heart's valves are part of the endocardium.

The endocardium releases hormones such as:

- endocardin, a substance that prolongs myocardial contraction;
- atrial natriuretic factor (ANF), released by the atria to oppose the activity of epinephrine, endothelin and the renin-angiotensin system
- brain natriuretic peptide (BNP) which is released by the ventricles upon ventricular distention having similar effects to ANF.

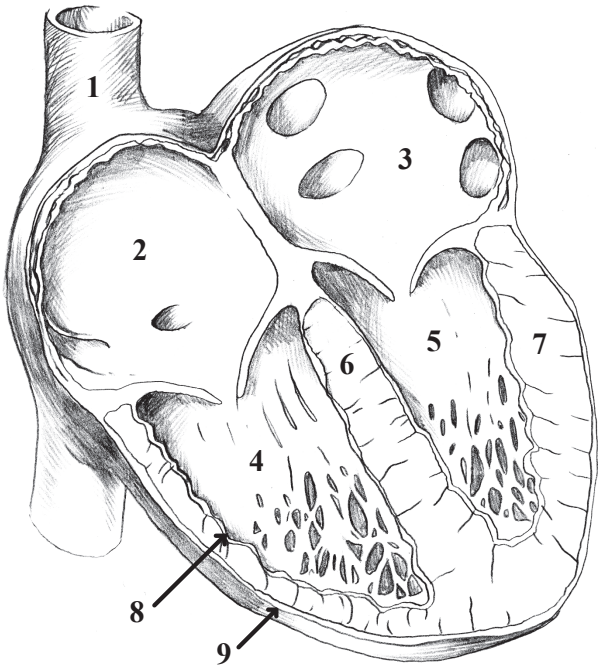
The heart is not just a pump but also an endocrine organ!

Chambers

The chambers of the heart are the main drivers within an intricate pathway, delivering blood to the lungs for gas exchange and enriching the body's cells with oxygen. The contracting and relaxing chambers facilitate varying pressure gradients that drive a resting cardiac output of five litres of blood per minute.

As the ventricles contract, the pressure in the ventricles overcomes the pressure of the aorta or pulmonary arteries, resulting in the valves opening and blood ejection. Similarly, as the ventricles relax and open, the resulting falling pressure created within the ventricles draws blood from the atria. Essentially, blood is sucked into the ventricle. In a healthy heart, approximately 65-85% of ventricular blood volume is provided during early diastole. Atrial diastole tops off the remaining 15-35% (atrial kick).

Figure 1.2 Chambers and Layers



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The heart consists of 4 chambers - 2 atria and 2 ventricles. The smaller atria are about 1/3 the size and volume of the ventricles. The left ventricle is the largest chamber of the heart, with about 3 times more muscle mass than the right ventricle. Both ventricles share a similar volume capacity. Due to the predominant size of the left ventricle, it is not surprising that 70% of all myocardial infarctions occur within the left ventricle.

Labels to figure 1.1: ***1.*** *superior vena cava;* ***2.*** *right atrium;* ***3.*** *left atrium;* ***4.*** *right ventricle;* ***5.*** *left ventricle;* ***6.*** *septum;* ***7.*** *myocardium;* ***8.*** *endocardium;* ***9.*** *epicardium*

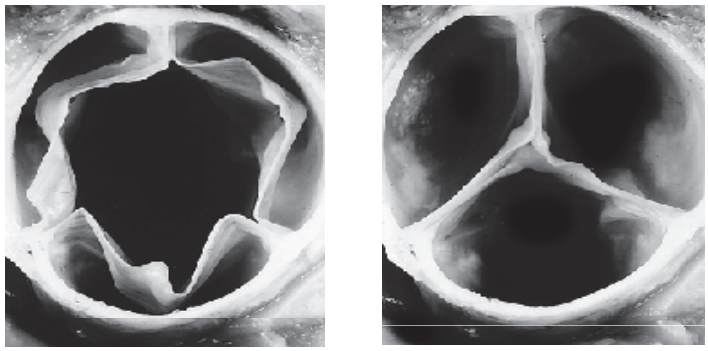
Heart valves ensure the forward flow of blood by closing off any back end routes. The atria do not share this advantage. The absence of valves between the venous system and the atria means that a small amount of blood is ejected back into the venous system with atrial contraction. With certain cardiac rhythms (i.e. 3rd degree AV Block, ventricular tachycardia and junctional rhythms), the timing of atrial contraction coincides with ventricular contraction and the closure of the AV valves (tricuspid and bicuspid). As a result, the atrial contraction delivers blood primarily back into the venous system causing the jugular veins to pulsate. The pulsations along the jugular veins are called **canon A waves**. This finding is sometimes useful when attempting to identify various challenging rhythms.

Discussions of the heart often refer to two hearts - a right and a left heart. Structurally, this is due to a thick layer of connective tissue called the septum that separates the left and right heart. Functionally, the right heart pumps deoxygenated blood to the lungs while the left heart pumps oxygenated blood to the body. When either the left or right side of the heart is unable to pump an adequate volume of blood, heart failure ensues that causes both decreased output and a backward volume buildup.

Valves

Valves act as gates ensuring unidirectional blood flow. They are located between the atria and ventricles as well as between the ventricles and the major arteries. The atrioventricular (AV) valves lie between the atria and the ventricles of the right and left heart. The ventricles eject blood through semilunar valves composed of 3 cusps.

Figure 1.3 Semilunar Valve (aortic or pulmonic)



The aortic and pulmonic semilunar valves are pictured in Figure 1.3. The three leaves of the semilunar valves are billowed closed during ventricular diastole as arterial pressure becomes greater than the pressure within the ventricles. The semilunar valves ensure forward flow of arterial blood ejected from the ventricles.

The atria and ventricles are separated by the tricuspid valve (3 leaf) in the right heart and the bicuspid or mitral valve (2 leaf) in the left heart. Blood ejected from the ventricles pass through the semilunar valves (see Figure 1.3), the pulmonic valve into the pulmonary arteries and the aortic valve into the aorta. Pressure within a ventricle or artery catches the cusps of a valve - like a parachute - closing the valve and preventing back flow.

Figure 1.4 The Heart's Valves (superior view)

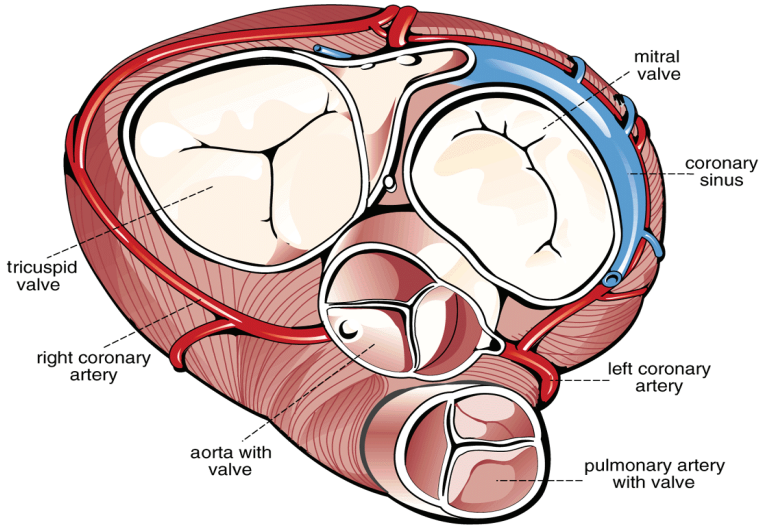


Figure 1.4 depicts the valves of the heart as viewed from above the heart.

The valves are composed of similar components: leaflets; annulus - a fibrous ring that encircles the valve; and chordae tendaneae – fibrous ligaments that connect to the papillary muscles. The papillary muscles flex when the ventricles contract to stabilize the AV valves. Note that an MI may weaken papillary muscles or rupture the chordae tendaneae, resulting in a heart murmur.

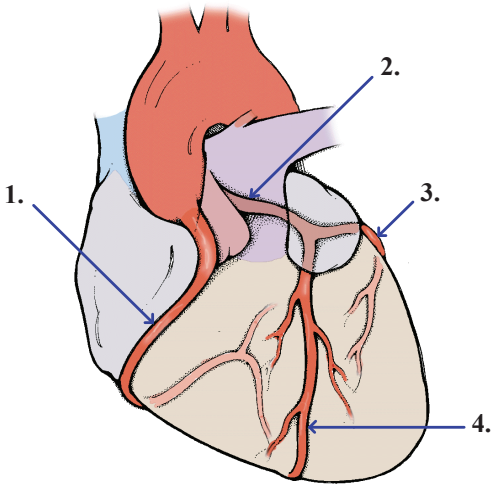
While heart murmurs may suggest valvular pathology, heart sounds also suggest normal function. The closing of the AV valves produce the classic **S1** sound, heard at the beginning of ventricle systole ('lub' of lub-dub). Subsequently, as the ventricles begin to relax (diastole), the semilunar valves close producing the **S2** heart sound ('dub').

Other causes of heart murmurs include age related changes to the valves such as the formation of calcium deposits and the stenosis of the valve leaflets or cusps. An impaired mitral valve, for example, could result in reduced blood volume being ejected from the left ventricle due to regurgitation of blood back into the atrium. This can eventually lead to left atrial hypertrophy and pulmonary hypertension.

Coronary Arteries

In order to beat over 100,000 times daily, the heart muscle requires a substantial blood and oxygen supply. The coronary arteries distribute the oxygen and nutrients necessary to provide energy to meet the workload demands of the heart. Even at rest, the cardiac cells extract 75% of the oxygen from the coronary arteries to meet energy demands. Essentially, the heart is entirely dependant on increased coronary artery blood flow to meet any increases in cardiac workload.

Figure 1.5 Coronary Arteries



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About 4-5% of the body's blood volume is contained by the heart's arteries and veins. This is a large volume considering that the heart comprises less than 1% of an adult's body mass.The heart's blood supply is provided mostly as the heart relaxes and dilates during diastole. This is unique - most organs receive pulsations of new oxygen-rich blood during cardiac systole (contractile phase of the heart).

The quantity of blood circulating through the coronary arteries is directly related to the coronary perfusion pressure, the difference between aortic diastolic pressure and central venous pressure (right atrial pressure). During events with increased central venous pressure and lower aortic diastolic pressure (i.e. right ventricular infarction) coronary perfusion often suffers.

Labels to figure 1.5: ***1.*** *RCA - right coronary artery;* ***2.*** *left main;* ***3.*** *circumflex;* ***4.*** *LAD - left anterior descending;*

The **right coronary artery** (RCA), sprouts off of the aorta superior to the aortic valve, primarily serving the right ventricle and the right atria. In about 50% of the population, the RCA branches early on to form the conus artery to further serve the right side of the heart. The RCA serves the right ventricle, the right atrium, the SA node (50-60% of people) and the AV node (90% of people). Note that the AV node and the Bundle of His are often served by both the RCA and the circumflex artery.

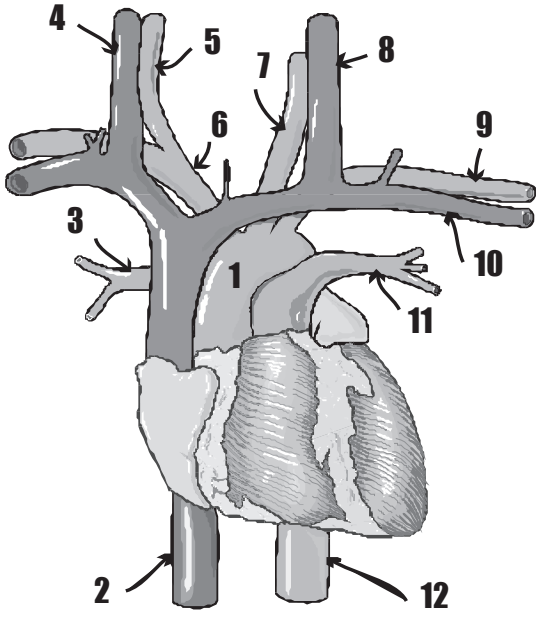
The **left main** begins at the left border of the aorta opposite the entrance to the RCA. The left main soon splits into 2 arteries: 1) the **circumflex** wraps around the surface of the left heart; and 2) the **left anterior descending** artery travels down the anterior surface of the left ventricle. The circumflex also serves the SA node (40-50% of people) and the AV node (10% of people).

The coronary veins exit into the right atrium via the coronary sinus. A one-way valve covers the coronary sinus, called the Thebesian valve (now this is definitely trivia).

Major Vessels

Several major vessels enter and exit the heart. The arteries carry blood away from the heart while the veins bring blood to the heart. While memorizing the major vessels is unnecessary, having a basic picture of the major vessels is clinically important.

Figure 1.6 Major Vessels



1. Aortic Arch
2. Inferior Vena Cava
3. Right Pulmonary Artery
4. Right Jugular Vein
5. Right Carotid Artery
6. Bracheocephalic Artery
7. Left Carotid Vein
8. Left Jugular Vein
9. Left Subclavian Artery
10. Left Subclavian Vein
11. Left Pulmonary Artery
12. Descending Aorta

The main vessel feeding the right heart is the vena cava. The right atrium also receives venous blood from the coronary sinus, the main venous return of the heart's blood supply.

Approximately 65% of blood volume is normally contained in the venous system. With increased energy demands, blood flow must increase. Table 1.2 outlines blood flow at rest and the changes in blood flow that occurs during strenuous activity. Sympathetic nervous system stimulation is responsible for the majority of the fluctuations in blood flow during exercise, with vasodilation and vasoconstriction occurring simultaneously to increase blood flow to the vital organs (i.e. brain, muscle).

The right ventricle ejects blood through the main branches of the left and right pulmonary arteries to the lungs. The left atrium receives its oxygen-rich blood supply via four main pulmonary veins. The left ventricle ejects blood into the aortic arch to the body. Within the arch, the coronary arteries branch off first followed by three main arteries that branch to the brain (carotids) and the upper thorax (subclavian artery).

Table 1.2 Blood Flow (BF) at Rest and During Exercise

Organ or Tissue	BF at Rest (cardiac output of 5000 ml)	BF with Exercise (volume)
Brain	650 ml	unchanged
Heart	200 ml	up to 3 times more
Muscle	1000 ml	up to 10 times more
Kidney	950 ml	reduced by 40%
Skin	400 ml	up to 4 times more
Abdomen	1200 ml	reduced by 50%
Other	600 ml	reduced by 30%

Note how the heart, skin and muscles receive significantly more blood flow while the abdomen and kidneys experience a reduction in blood supply. The skin's blood supply increases primarily to help release the excess heat yielded by increased energy use. The heart requires increased energy to meet the demands of an increased heart rate and increased stroke volume.

Note that the lion's share of blood volume is delivered to the muscles during exercise. During periods of cardiac ischemia, resting the muscles provide significant reductions to cardiac output demands - and cardiac oxygen demand - thus helping to minimize the extent of the ischemic episode.

Atrial Fibrillation and the Major Vessels

About 1 in 5 people over the age of 50 develop atrial fibrillation, a chaotic quivering of the atria. Blood velocity typically slows along the walls of the atria from the friction between the endocardium and the blood. As long as the atria rhythmically contract, the blood is propelled quickly forward. Without atrial contraction (i.e. atrial fibrillation), blood along the walls can slow significantly. After 48 hours, about 3-5% of people in atrial fibrillation will form a blood clot in the atria.

If this clot is dislodged from the right atrium and floats to the lungs via the pulmonary arteries, a pulmonary emboli results. If a clot develops and moves from the left atrium, the aortic arch is next in line. Of the three main vessels of the arch, two of the three vessels target the brain. As expected, atrial fibrillation is a major risk factor for cerebral vascular accidents (stroke).

Having an understanding of the mechanical structures of the heart helps us make sense of both normal physiology and pathophysiology. Looking at the ramifications of atrial fibrillation is but one example.

Summary

In this chapter we have laid the ground work towards understanding electrocardiograms. The heart is a four-chamber (**2 atria and 2 ventricles**) pump. Its function is to deliver oxygen and nutrient rich blood throughout the body. The heart is often considered two hearts, the right and left heart. The septum is a fibrous barrier that serves as part of the heart's skeleton. The **septum also serves to** separate the right chambers from the left chambers of the heart.

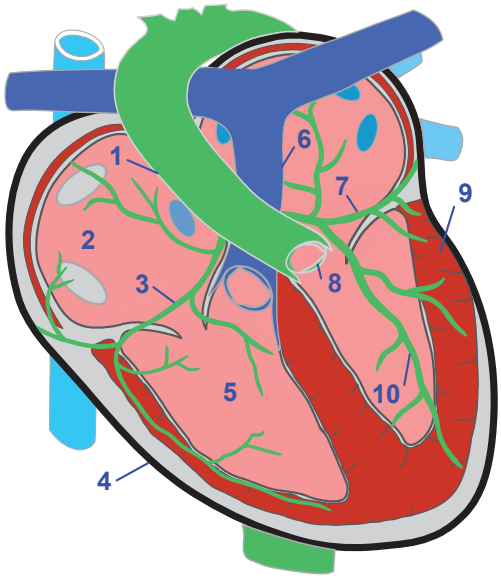
Valves act as gates in the flow of blood. They are located between the atria and ventricles as well as between the ventricles and the major arteries. The heart, being a specialized muscle, requires its own blood supply of oxygen and nutrients. This is provided by **coronary arteries**.

The major vessels of the heart include the vena cava, the pulmonary arteries, the pulmonary veins and the aorta. Together, the heart's mechanical structures synchronize efforts to satisfy the blood and oxygen requirements of the body.

Chapter Quiz

Try this chapter quiz to check whether you are 'anatomically sound'. Good luck.

1. Connect the labels with the appropriate number.



1. _____
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10. _____

Aortic Valve

Epicardium

Right Atrium

Pulmonary Artery

Right Ventricle

RCA

Aorta

Myocardium

LAD

Circumflex Artery

2. An inferior MI is usually a right ventricular infarction?

True or False

3. A posterior MI can result from an occlusion to the circumflex artery?

True or False

4. The heart is located in the center-left mediastinum between the _____ intercostal space (ICS) and the _____ ICS.

5. Coronary artery perfusion is increased with:

a) growing cardiac energy demands
b) sympathetic neural stimulation
c) widened differences between diastolic pressure and central venous pressure
d) all of the above

Answers: ***1.*** *A. aorta; B. RA; C. RCA; D. epicardium; E. RV; F. PA; G. circumflex; H. aortic valve; I. myocardium; J. LAD* ***2.*** *False* ***3.*** *True* ***4.*** *2nd, 5th* ***5.*** *d)*

6. The atria of the heart (circle all that apply):

a) respond to increased distention by releasing atrial natriuretic peptide to blunt the effects of epinephrine, endothelin and the renin-angiotension cascade
b) pump blood into a nearly empty ventricle
c) are roughly equal to the ventricles in volume and myocardial thickness
d) receive blood from the venous system
e) does not benefit from a valve to prevent atrial backflow during contraction

7. Blood flow to the lungs is roughly equal to the blood flow to the rest of the body.

True or False

8. The AV node and the Bundle of His receive blood from (circle all that apply):

a) the circumflex artery
b) the left anterior descending artery
c) the right coronary artery
d) all of the above

9. The endocardium (circle all that apply):

a) is continuous with the heart valves
b) begins to contract before the epicardium
c) receives blood supply from the distal aspect of the coronary arteries
d) has endocrine functions
e) often experiences ischemia prior to the epicardium
f) provides a smooth surface to facilitate blood flow
g) all of the above

10. While most of the body extracts only a quarter of the oxygen available, the resting heart extracts about (10%, 30%, 50%, 75%) of available oxygen to meet energy demands. This suggests that the heart is very dependent on (coronary artery perfusion, un-extracted oxygen reserves) during periods of high energy demand.

11. Pericarditis is an infection of the protective layers that encase the heart. Resulting inflammation and exudate can cause chest pain and a pericardial effusion.

True or False

12. The heart sounds typically heard with a stethoscope form a S_1 sound during the closure of the (AV valves, semilunar valves) and S_2 during the closure of the (AV valves, semilunar valves).

Answers: ***6.*** *a),d), e)* ***7.*** *True* ***8.*** *a), c)* ***9.*** *all of the above* ***10.*** *75%, coronary artery perfusion* ***11.*** *True* ***12.*** *AV valves, semilunar valves*

13. Tissues that experience increased blood supply during exercise and other high energy demand states include (circle all that apply):

a) heart
b) brain
c) skin
d) muscles
e) kidneys
f) abdomen

14. Atrial fibrillation is associated with increased risk of stroke after a period of (4 hours, 12 hours, 48 hours, 72 hours).

15. Most myocardial infarctions occur to the left ventricle.

True or False

Suggested Readings and Resources

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

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

HeartScape: The Anatomy of the Heart. (2001)
Web: http://www.skillstat.com/heartscapeDemo.html

The Heart: An Online Exploration. Web: http://sln.fi.edu/biosci/heart.html

What's Next?

Understanding the basic structures of the heart is vital to making sense of electrocardiograms. Chapter 2 builds on this knowledge, progressing step by step through the cardiac cycle and the many factors that affect cardiac output.

Answers: ***13.*** *heart, muscles, and skin* ***14.*** *48 hours* ***15.*** *True*

2

It's All About Cardiac Output

Quick Look

This chapter addresses the cardiac cycle and cardiac output parameters. Managing cardiac emergencies relies heavily on the ability to recognize, understand and respond to altered cardiac output. In an era where pulmonary artery lines are utilized less and less, the stalwart ECG continues to provide indicators about a patient's cardiac status.

Understanding the dynamics of cardiac output may not be necessary to identify dysrhythmias. In fact, Chapters 5-8 will suffice in this matter. Making sense of the ECG from a clinical perspective, however, requires a basic understanding of the parameters that govern cardiac output.

The ECG is a powerful tool in your assessment of a patient's cardiac status. The ECG might be likened to a window on the patient's heart, providing valuable detail not only about the electrical workings of the heart but also about the quality of the heart's ability to pump.

"It's all about managing cardiac output!"

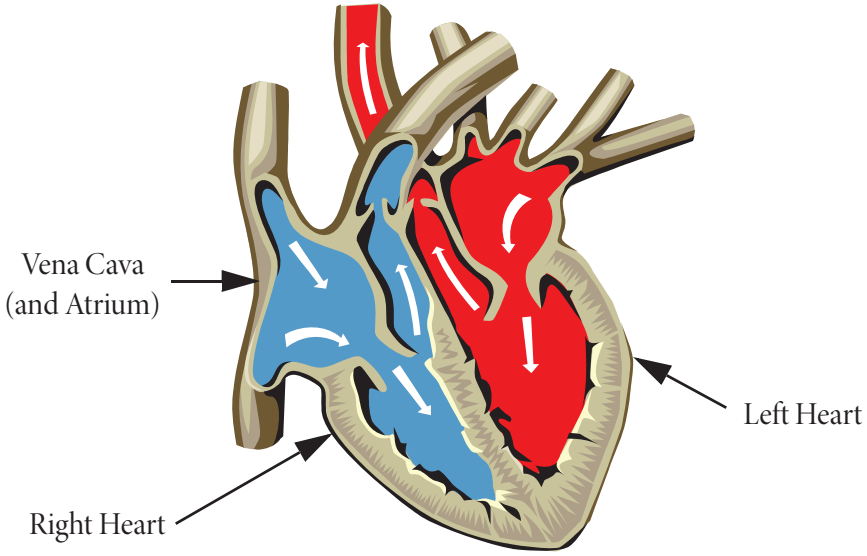
Not So Anonymous

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 2.1 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 additional15-35% 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 x 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).

$$O_2 + \text{Glucose} = H_2O + CO_2$$

$$\Downarrow$$

36 ATP

Metabolism that utilizes oxygen is called **aerobic metabolism**. The above equation is the balance of the much abbreviated Kreb's 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 continuos production of ATP, each of these processes would cease.

Aerobic metabolism has by-products of water (H_2O) and carbon dioxide (CO_2). Water we can definitely use. In fact, about $^2/_5$ 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.

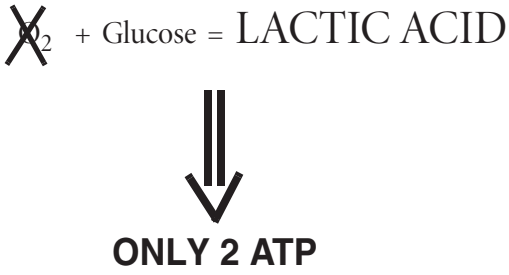
Sufficient cardiac output is necessary to deliver adequate supplies of oxygen and nutrients (glucose) to the tissues. This translates to the conclusion that *cardiac output is directly related to energy production*. Low cardiac output will reduce energy levels.

For example, if your cardiac output fell to 3500 ml (about $^2/_3$ of normal) your oxygen - and hence your energy supply - would be decreased as well. Your brain with $^1/_3$ less energy may be less sharp, confused or even unconscious. Your muscles with $^1/_3$ 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.



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 continuos 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 $^{3}/_{4}$ 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 2.1 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. 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 2.1

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

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.

Heart Rate of 80/minute: CO = SV X HR = 65 X 80 = **5200**

Heart Rate of 100/minute: CO = SV X HR = 65 X 100 = **6500**

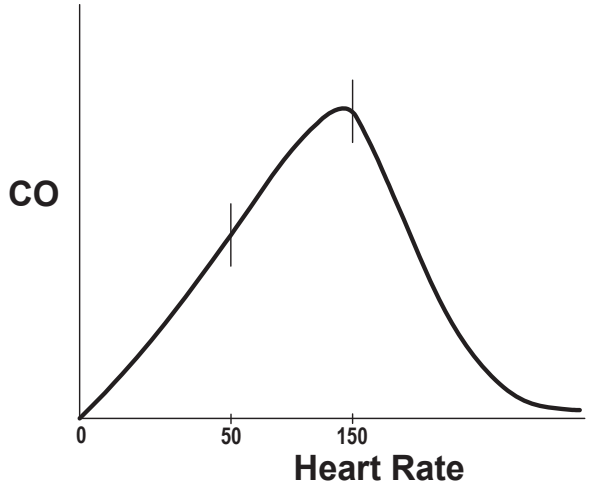
With this simplistic example, a 20% increase in heart rate (from 80 to 100/minute) yields a 20% increase in cardiac output (from 5200 ml to 6500 ml).

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 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 2.2 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.

CO = SV X HR = 80 X 40 = **3200 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 litres.

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 2.3 .Frank-Starling Curve

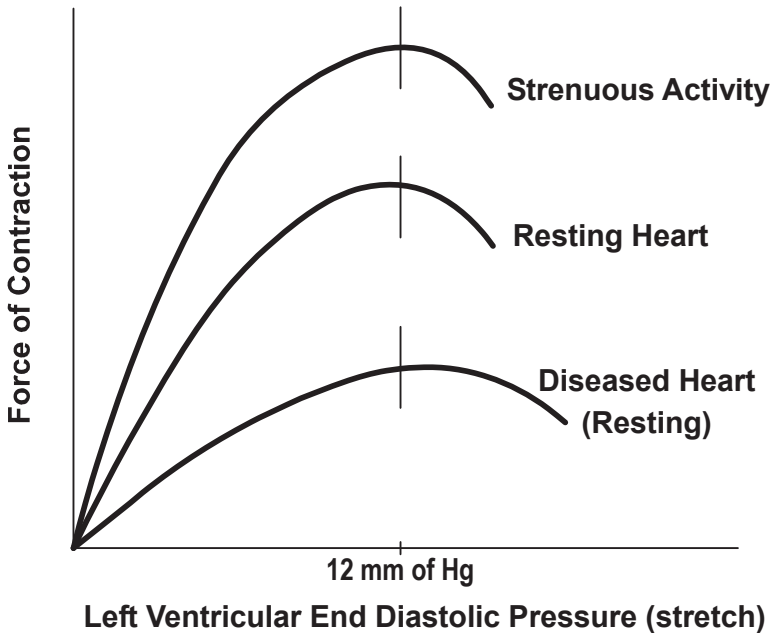


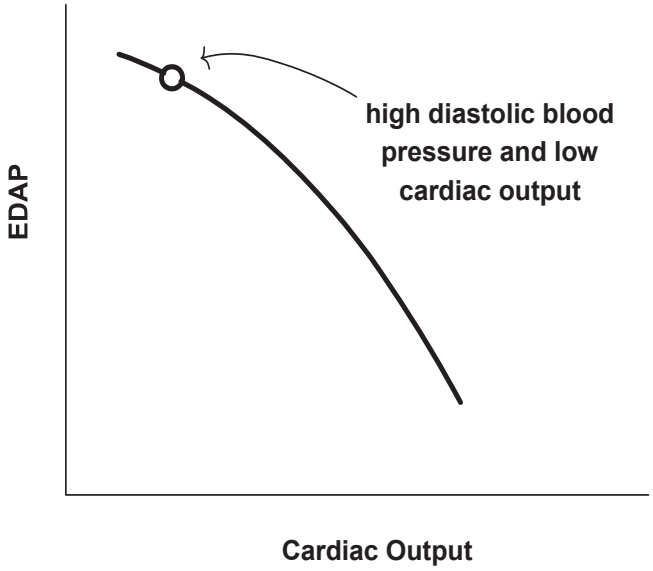
Figure 2.3 depicts the relationship between ventricular end diastolic pressure and contractility for a resting healthy heart, a resting diseased heart and a healthy heart during strenuous activity. Several points are evident here: ***1)*** *in general, the force of contraction (contractility) increases 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).

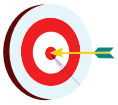
While systemic vascular resistance is not easily determined without a pulmonary artery catheter, diastolic blood pressure is easily measured. So while an accurate estimate of afterload is often not clinically practical, a patient's diastolic pressure provides a good indication of the resistance the left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

Figure 2.4 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 2.2 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 litres/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 propanolol and a daily nitropatch. 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.

Answers: ***3.*** *The left ventricle is beginning to fail with too much preload; back pressure to the lungs push fluids into the alveoli*

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/minute x 35 ml = 2800 ml/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 laid the ground work necessary to become skilled at making sense of ECGs. For example, extreme heart rates - too fast or too slow - often cause low cardiac output states. Cardiac ischemia and catecholamine stimulation can cause a variety of serious dysrhythmias. Understanding the heart 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 exasperates cardiac ischemia.

Being aware of the dynamics of cardiac output enables you to readily connect ECG interpretation with a patient's clinical picture, making you better able to care for and manage the patient experiencing an acute cardiac event. Is this not a much stronger position than simply being able to identify a cardiac rhythm? You bet it is.

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 increases 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

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

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

True or False

8. Patients with heart disease will most likely hemodynamically tolerate hearts 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 _____.

Answers: ***7.*** *True;* ***8.*** *False;* ***9.*** *a),b),c),f);* ***10.*** *True;* ***11*** *all but b);* ***12.*** *filling time, stroke volume. rate*

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 Readings and Resources

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

Animated Cardiac Cycle. (2003). University of Utah.
Web: http://medlib.med.utah.edu/kw/pharm/hyper_heart1.html

Cardiac Cycle. (2002). University of Kansas School of Nursing.
http://classes.kumc.edu/son/nurs420/unit4/cardiaccycle.html

Cardiac Output: Ever Wonder What Those Numbers Really Mean? (2002). Medical Education Consultants. Web: http://mededcon.com/card01.htm

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

What's Next?

This chapter established the importance of cardiac output and the parameters that affect cardiac output. The picture is not yet complete, though. The mechanics of the heart are truly at the whim of the heart's electrics. Chapter 3 continues to build a solid foundation of cardiac basics with an overview of the heart's electrical system. While the quality of cardiac output should be factored into any ECG interpretation, so should the electrophysiology of the heart.

Answers: ***18.*** *False;* ***19.*** *would, is;* ***20.*** *True*

3

The Electrics

Quick Look

The previous two chapters explored the mechanics of the heart, looking first at cardiac anatomy, then at the factors that affect cardiac output. Attention now turns to the heart's control centre, the electrical conduction system.

Without the innervation by the heart's electrical pathways, the heart muscle will simply not pump. An absence of cardiac electrical activity - the "flat line" seen on a monitor - is definitely not a good sign!

Starting with the SA node, each of the major electrical components of the heart are explored in this chapter. Attention is focused on pacemaker and non-pacemaker sites. The processes of depolarization and repolarization are briefly addressed. Finally, the autonomic control of heart rate is discussed.

While this may sound akin to discussing particle physics, the heart's electrics are much simpler and at least as interesting. There is much to be digested over the next dozen pages or so. Let's dig in.

There must be a seat...from which heat and life are dispensed to all parts...the heart is this place... I trust no one will deny.

William Harvey (1628)

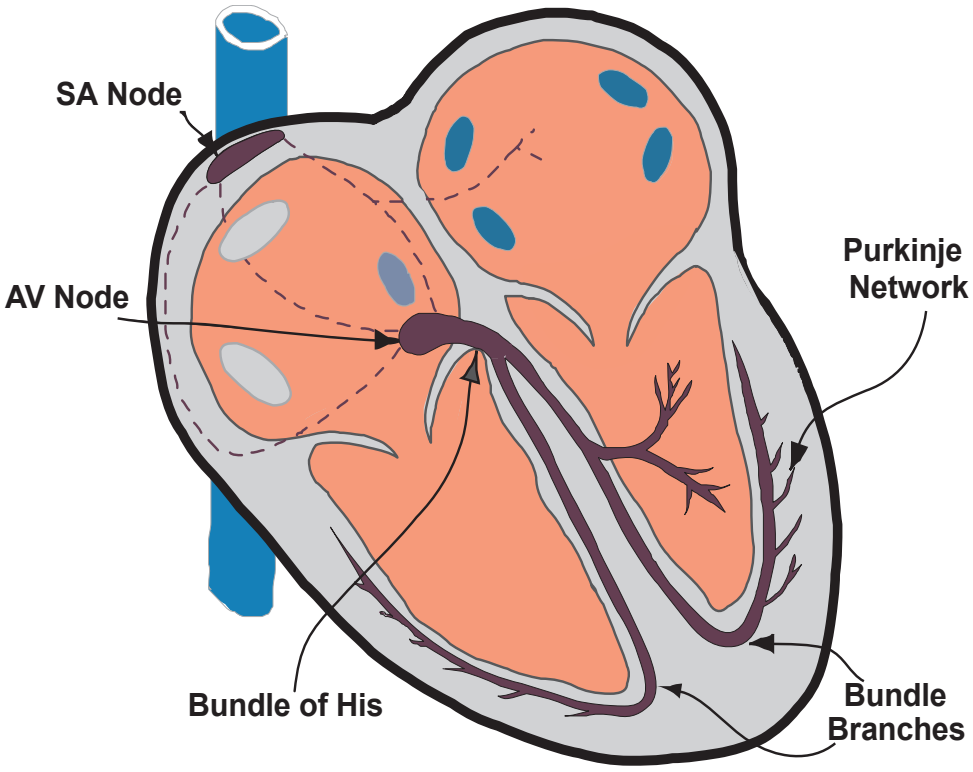
Electrical Overview

Late in the 1700s, physiologists realized that an electric stimulus causes muscles to contract. In the past 200 years, electrophysiologists have continued to uncover many secrets of cardiac electrophysiology. With these discoveries, an in depth understanding of cardiac electrophysiological events has led to insights into dysrhythmia interpretation and treatment.

New findings continue at a brisk pace, revealing the many evolutionary features of the heart.

The heart's electrical system is composed of five significant components: the SA node, the AV node, the Bundle of His, the Bundle Branches, and the Purkinje network. These electrical structures work as a cohesive interdependent team, conducting electrical impulses rapidly throughout the heart. Their location within the heart is depicted in the Figure 3.1 below.

Figure 3.1 The Heart's Electrical Pathway



A wave of depolarization normally begins with the SA node. This electrical wave from the SA node passes quickly across the atria, through the AV junction (the AV node and the Bundle of His) then across the ventricles via the bundle branches and the Purkinje network.

As an electrical impulse is initiated and then conducted over the heart, affected cardiac cells undergo an ionic shift, called **depolarization**. The interior of cardiac cells at rest have a negative charge relative to the outside of the cell. During depolarization, positive ions enter the cell, changing the cell's polarity. Soon after, the cells experience a series of ionic shifts that return the cell to its resting state. This subsequent process is called **repolarization**. Contraction of cardiac cells is initiated during repolarization.

The cells that initiate and then conduct this impulse can be split into two groups, pacemaker cells and non-pacemaker cells. **Pacemaker cells** have the ability to self-initiate an electrical impulse. This relative independence from the body enables the heart to continue beating even if removed from the body (up to 20 minutes). The SA and the AV nodes contain groups of pacemaker cells. **Non-pacemaker cells** conduct the impulse to neighboring cells but usually do not initiate an impulse (i.e. atrium).

The electrical activity of the heart, then, is dependent on pacemaker cells to initiate each wave of depolarization. Normally, this depolarizing wave passes across the cells of the atria, through the AV node, and the Bundle of His. The bundle branches carry this wave through to the Purkinje network where the wave enters the ventricular endocardium, the myocardium and the epicardium in that order. The atria and the ventricles depolarize then contract *from the inside out*.

A wave of repolarization follows. Normally, the repolarization of the endocardium is delayed, so the wave of repolarization begins in the epicardium, proceeds through to the myocardium and finishes at the endocardium - opposite the direction of depolarization.

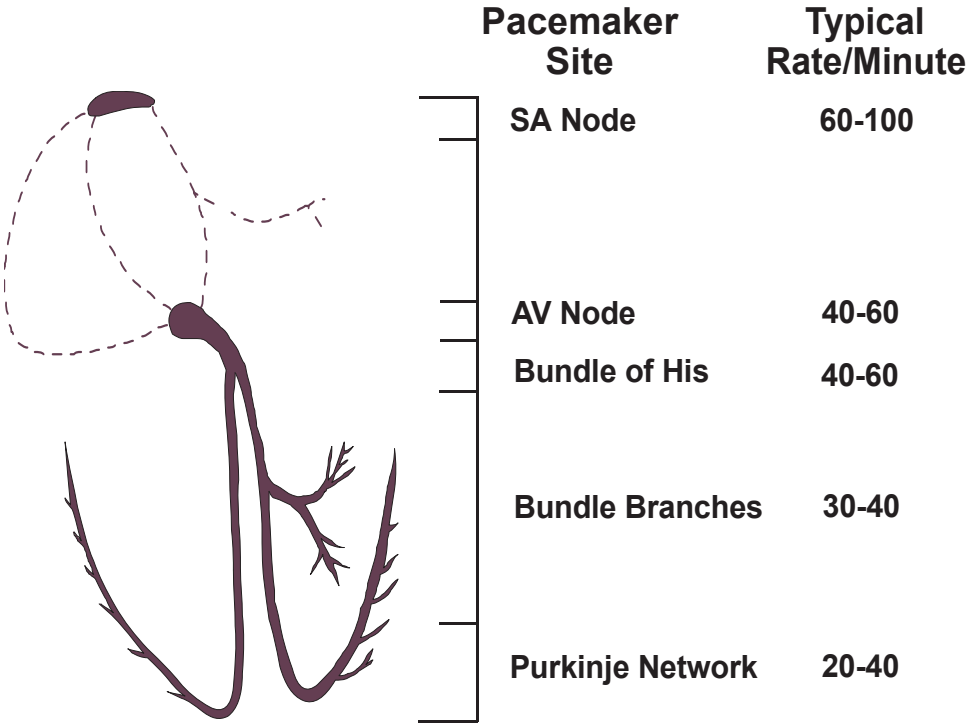
SA Node

The **SA node**, usually the dominant pacemaker, is located in the right atrium at the opening of the superior vena cava. The SA (sinoatrial) node is a clump of hundreds of specialized cardiac cells that have the ability to self-initiate an electrical impulse. This pacemaking ability, called **automaticity**, makes the SA node a pacemaker site in the heart.

As a general rule, the site in the heart that is able to self-generate the quickest rate, **RULES** the heart. This site is almost always the sinoatrial node (SA node). Thus, the SA node is often called the dominant pacemaker. If an ectopic site (site other than the SA node) begins to fire faster than the SA node, the ectopic site tends to drive the heart.

The SA node initiates an electrical impulse at a rate faster than other pacemaker sites (see Figure 3.2). In the heart, the pacemaker which fires at the quickest pace takes control of heart rate. This is why the SA node is the "dominant pacemaker". A cardiac rhythm that originates from the SA node is called a sinus rhythm.

Figure 3.2 Pacemaker Sites and Normal Rates



These are typical heart rates from various pacemaker sites. Heart rates can vary, though, for each site above and below the range specified. Note that the typical pacemaker rate decreases as the distance from the SA node increases. Lower pacemakers serve as "back-up" in case higher pacemakers fail. The bundle branches and the Purkinje network (both from the ventricles) typically provide an exceptional slow heart rate that is often associated with poor cardiac output. Note also that the absence of atrial activity results in a loss of atrial kick, impacting an already low cardiac output further.

The SA node normally generates electrical impulses at 60-100 /minute. This rate tends to increase with sympathetic stimulation (norepinephrine and epinephrine) and slows with parasympathetic stimulation (acetylcholine and the Vagus nerve). Therefore, Vagal stimulation can slow the SA node to rates below 60/minute causing a sinus **bradycardia**. Sympathetic stimulation can cause rapid sinus rhythms called sinus **tachycardias**. The control of heart rate is addressed in more detail later in this chapter.

Atrial Conduction and the AV Junction

Once the SA node initiates an electrical impulse, the resulting electrical wave moves across the right and left atria. The atrial septum serves as an electrical insulator. Bachman's Bundle tunnels through the atrial septum to continue the electrical wave across the left atrium. The wave takes approximately 3/100 of a second to cross the atria and arrive at the AV node.

The atrioventricular (AV) node is a rounded bulbar structure of specialized cells similar to the SA node. The AV node also has intrinsic automaticity, with the ability to serve as a pacemaker in case of SA nodal failure. The AV node usually does not initiate impulses, though, as its intrinsic firing rate is normally 40-60 /minute, slower than the SA node. Remember, the fastest pacemaker site rules.

Figure 3.3 Connective Tissue Structures and Electrical Conduction

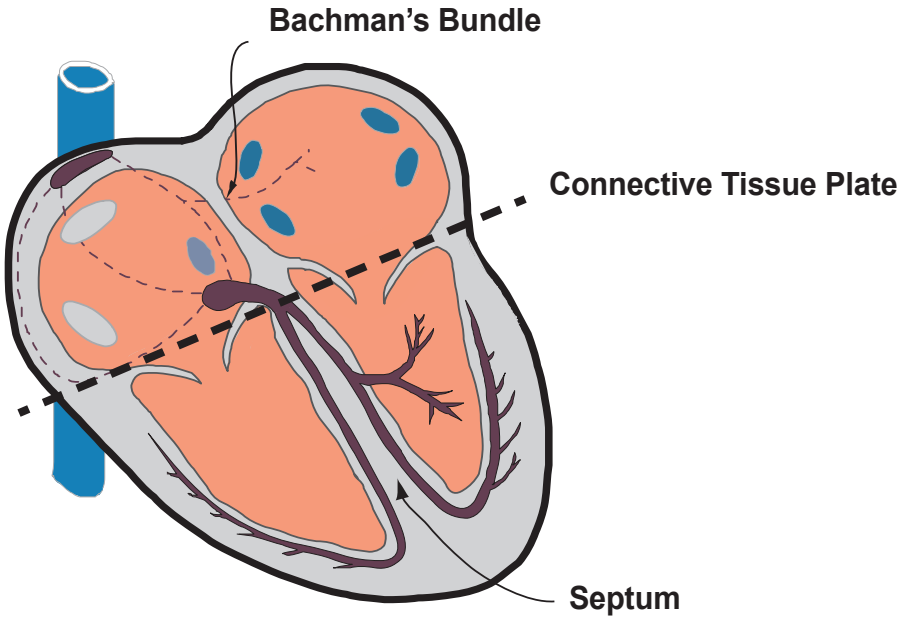


Figure 3.3 depicts the structures that form the cardiac skeleton, the septum and the plate of connective tissue that separates the atria from the ventricles. Connective tissue does not conduct electrical impulses, serving rather as an electrical insulator or barrier. To connect the left and right atria electrically, ***Bachman's bundle*** *burrows through the atrial septum. The* ***Bundle of His*** *performs a similar function, connecting the atria electrically with the ventricles. Note that without the Bundle of His, supraventricular impulses would not be transmitted through to the ventricles. The ventricles would then be dependent on their own slow intrinsic pacemakers.*

The AV node has a second important role. The AV node and the bundle of His slows impulse conduction to allow the atria time to contract prior to ventricular contraction. In other words, the AV junction provides the time delay for an atrial kick. The time taken to cross the small AV junction is 10-12/100 of a second (a significantly lengthy period for such a small structure).

The **Bundle of His** serves as an electrical connection between the atria and the ventricles, traversing the fibrous plate that separates the atrial and ventricular electrical systems. The AV node and bundle of His form the AV junction (sometimes just called the junction).

Note that the AV junction, atria and SA node are the three main supraventricular (located above the ventricles) electrical sites. This is an exceptionally simple and important distinction. As you will soon discover, in order for an impulse to be transmitted down the bundle branches, the impulse must be supraventricular in origin.

The Ventricular Conduction System

The ventricles' electrical system is exceptionally efficient. To produce a forceful, coordinated contraction, the electrical wave must travel quickly through the large ventricles. Knowing that the atria depolarize over 3/100 of a second, how long would depolarization take to crest across the ventricles (three times the size of the atria)?

Venture a guess? The electrical wave crosses the ventricles in a mere 1/100 of a second. The wave moves like lightning! The question is: How is this rapid conduction accomplished?

Table 3.1 .Duration of Depolarizing Waves

Structure	Duration of Depolarizing Wave
Atria	0.03 seconds (3/100)
AV Junction	0.10-0.12 seconds (10-12/100)
Ventricles	0.01 seconds (only 1/100)

The time taken for depolarization is somewhat counterintuitive. The depolarizing wave takes the longest duration moving through the small AV junction (and thus allowing for atrial kick) and the shortest time covering the large ventricles.

First, the specialized bundle branches and Purkinje network facilitate this rapid conductivity (refer to Figure 3.1 "The Heart's Electrical Pathway" on page 54). With the atria, only one wave is propagated. Via the bundle branches, this impulse is split into at least **three simultaneous waves**, thus reducing the distance each wave must travel. Less distance equates to less time. As a result, the time taken to depolarize the ventricles is reduced considerably.

Second, the bundle branches and Purkinje network are composed of Purkinje fibers, specialized cardiac cells that are tailored for fast conductivity. These rapidly conducting cells carry the impulses through connective tissue, reaching contractile cardiac tissue at the distal ends of the Purkinje network. This encapsulated electrical network is extremely efficient, rapidly carrying a depolarizing wave throughout the ventricles.

Note that the speed of contraction translates directly into the force of contraction. The faster that the ventricles can depolarize and subsequently contract, the greater the force of contraction. A greater force of contraction increases both stroke volume and cardiac output. Force of contraction is referred to as **contractility**.

Why is the speed of ventricular depolarization important? As mentioned in the box above, the faster the depolarization, the greater the force of contraction. But there is another good reason to take the time to fully grasp what causes the ventricles to depolarize with varied speeds.

We established that an electrical wave envelops the ventricles very quickly **IF** the bundle branches and the Purkinje network are utilized. This is comparable to getting off the back country roads and racing down the freeway. The rapidly conducting bundle branches could be called the Autobahn* of the heart.

Where is the only location to ramp onto the Autobahn of the heart? Remember that the bundle branches are largely encapsulated in connective tissue. The impulse must have travelled through the bundle of His to arrive at the bundle branches (the Autobahn). This is the only entry point to the Autobahn.

Where are we going with this? If the impulse travels through the bundle of His, then it originated in either the bundle of His or above the bundle of His (i.e. the AV node, the atria or the SA node). Simply stated, for a *rapid* wave of depolarization to envelop the ventricles, the impulse must originate above the ventricles.

Let's repeat this for effect. **In order for rapid depolarization of the ventricles to occur, the impulse must originate from a supraventricular site.**

**The Autobahn is an ultra-fast freeway that connects several countries in western Europe.*

Here's the crunch. On an electrocardiogram - an ECG - the QRS complex is often narrow. We are jumping ahead a bit here, but a QRS complex represents ventricular depolarization. An ECG is the graphical representation of the electrical activity of the heart, with the horizontal axis (width) of the ECG being a measurement of time. A narrow QRS then equates to rapid ventricular depolarization, taking very little time. A **narrow QRS, then, occurs when the impulse originates above the ventricles**.

Figure 3.4 Ventricular Conduction Speed and the QRS Complex

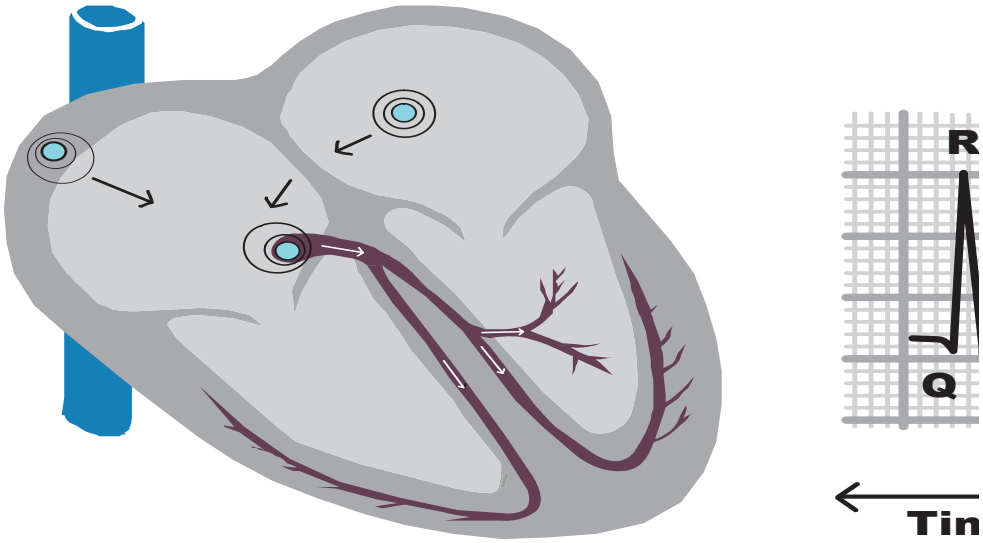


Figure 3.4 illustrates an impulse originating from the SA node, the atria or the junction. These supraventricular impulses are transmitted to the ventricles via the bundle branches, metaphorically referred to as the Autobahn. Since the Autobahn or bundle branches are used, the ventricles will depolarize rapidly, resulting in a narrow QRS complex.

Consider for a moment an ectopic impulse originating in the right ventricle. Instead of three simultaneous depolarizing waves, one wave depolarizes the right and then the left ventricle. Of course, more distance is covered by the one wave, taking more time. Instead of the Autobahn, the wave travels the back country roads. With more time taken for ventricular depolarization, a wide QRS results.

When interpreting an ECG, the location of the originating impulse for each beat is quite important. In fact, the naming of most cardiac rhythms begin with the site that the impulse originated. For example, a rhythm that consistently originates from the sinoatrial node is called a sinus rhythm. If a beat originates from the AV junction, it is called a junctional beat.

Therefore, based on what we know about the QRS and the speed of depolarization, a narrow QRS occurs with supraventricular rhythms. A wide QRS complex is commonly associated with ventricular rhythms. This is perhaps the most important step in

identifying cardiac rhythms. Is the QRS wide or narrow? The width and shape of the QRS is addressed in more detail in the next two chapters. As it stands, though, you are already equipped to differentiate between supraventricular and ventricular rhythms.

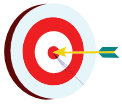
Asking the question, "Is the QRS wide or narrow" is an important step in ECG interpretation. A narrow QRS occurs when an impulse that originates from above the ventricles travels down the rapidly conducting bundle branches to depolarize the ventricles.

We sneaked ahead a little to look at one aspect of the ECG. It is important, though, to integrate your knowledge of the heart's electrical structures with the skill of ECG interpretation. Understanding why a QRS is narrow is much better than memorizing the particulars of every cardiac rhythm.

Controlling Heart Rate

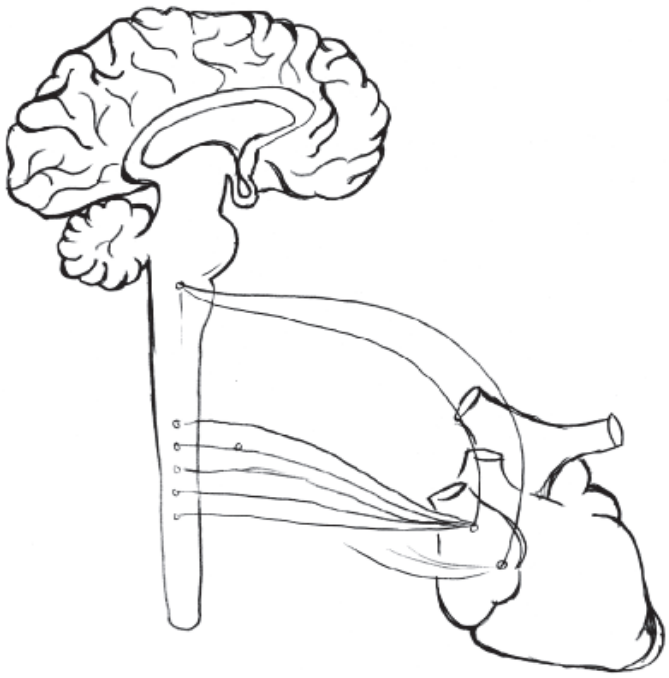
Heart rate, the numbers of beats or cardiac cycle per minute, is the result of three factors: intrinsic control by the heart's pacemakers, sympathetic stimulation and parasympathetic stimulation. The heart's pacemakers have their own intrinsic rate of impulse formation. For heart transplant recipients, without the benefit of cardiac innervation, this is a welcome phenomenon. Physiologists have determined that the SA node would beat at a rate of about 100/minute without any other influences.

Typical heart rates, though, range across a much wider continuum due primarily to the influence of the autonomic nervous system. Influence from the sympathetic nervous system increases heart rate and the speed of conductivity. Catecholamines released from the adrenal glands (i.e. epinephrine) can also produce a similar effect.



Terminology
The autonomic nervous system has dramatic effects on the cardiovascular system. When regards to the heart, sympathetic stimulation can yield a positive **chronotropic** (rate) effect, increasing heart rate. In contrast, the Vagus nerve produces a negative chronotropic effect slowing the heart rate. The Vagus nerve also slows the conductivity across the AV node. This is called a negative **dromotropic** (speed of conduction) effect. Sympathetic stimulation - particularly beta 1 stimulation - causes a positive **inotropic** (force of contraction) response, meaning that the force of contraction has increased. The medication Dopamine is known as a positive inotrope because it has the effect of increasing cardiac contractility.

Figure 3.5 The Autonomic Nervous System and the Heart



While sympathetic stimulation innervates most of the heart, parasympathetic stimulation via the Vagus nerve is more specific, innervating the SA and AV nodes.

The main effect of the catecholamines epinephrine and norepinephrine is an increased cardiac output. The alpha effect, vasoconstriction, shunts blood away from the periphery and improves preload and afterload (see Chapter 2). Coronary and cerebral perfusion are also enhanced. The beta 1 effects of epinephrine increase cardiac output by increasing both heart rate and stroke volume (contractility).

Unfortunately, beta 1 stimulation also increases myocardial oxygen demand. Beta 1 stimulation is also responsible for dysrhythmia generation. It is not surprising that beta blockers, which tend to slow heart rate, reduce cardiac contractility, decrease myocardial workload and decrease dysrhythmias are one of the most important medical treatments for myocardial infarctions (AMI). The morbidity and mortality of an AMI is reduced by as much as 40% with early beta blocker administration.

Parasympathetic innervation produces the opposite effect, slowing the rate of impulse formation by the SA node and slows conductivity through the AV nodes. Parasympathetic influence occurs via the Vagus nerve (acetylcholine). Vagal stimulation is also known to decrease the contractility of the atria as well as cause peripheral vascular dilation.

Table 3.2 Cardiovascular Receptor Sites and Responses to the Autonomic Nervous System

Autonomic Nervous System	Principal Chemicals	Receptors (Effects)	Sites Affected	Site Response
Sympathetic	catecholamines (norepinephrine, epinephrine)	**Alpha** (constriction)	abdominal, peripheral, coronary blood vessels	arterial and venous constriction
		Beta 1 (increases heart rate, enhances contractility, increases cardiac irritability)	cardiac muscle	increased heart rate and strengthened force of contraction
		Beta 2 (dilation of bronchioles)	bronchioles	bronchioles dilates
Parasympathetic	acetylcholine	cholinergic receptors	SA node, AV node, atria, coronary vessels	slows rate, conductivity and weakens atrial contraction; dilates coronary vessels

Table 3.2 presents a summary of the cardiac effects of the autonomic nervous system. Parasympathetic and sympathetic innervation tend to produce opposite effects. Sites that experience both parasympathetic and sympathetic innervation, such as the SA and AV nodes, are constantly being pulled in different directions. For example, with significant Vagal stimulation (parasympathetic nervous system), the heart rate tends to slow. If Vagal stimulation is blocked by the administration of a vagolytic medication such as Atropine, the sympathetic nervous system exerts its effect, driving the heart rate up once again.

Note that whereas sympathetic stimulation tends to blanket the heart, parasympathetic stimulation is limited primarily to the SA node, the atria and the AV node. During episodes of rapid heart rates that originate in above the ventricles (supraventricular tachycardias), Vagal stimulation can often slow or even terminate the fast rhythm by slowing the conductivity through the AV node.

Several techniques can cause **Vagal stimulation**: the Valsalva maneuver or "bearing down", carotid sinus massage, immersing a person's head in cold water, and even a change of position. Perhaps not as popular is the digital anal sweep. Note that all forms of Vagal stimulation can slow heart rates significantly.

Vagal stimulation can also produce very slow heart rates called **bradycardias**. For athletes, regular exercise tends to increase vagal tone with resting heart rates often being in the forties or fifties. Excess Vagal stimulation for people with structural heart disease can produce disastrous consequences, causing bradycardias that may be associated with hemodynamic compromise. Even periods of asystole - an absence of heart rate - can be caused by Vagal innervation.

Table 3.2 outlines the various effects of the sympathetic and parasympathetic nervous systems. Heart rate is the product of several competing drives. Behind the parasympathetic and sympathetic innervation is the body's need to quickly respond to internal and external stressors to keep its steady state. The ability of the heart to react quickly with changes in heart rate is a major factor in holding this state of homeostasis.

Summary

In this chapter we completed what we began in Chapter 2: review the anatomy and physiology of the heart. Understanding the inner workings of the heart is vital to ECG interpretation and to responding effectively to acute cardiac events. Of course, this review has been a simple, high level review. Hurst's The Heart presents an *overview of the heart* in a short 2600 pages! Nevertheless, we have covered the essentials necessary to our work at hand.

This chapter is an abbreviated description of the heart's electrical system. The mechanical aspects of the heart are intimately connected to the heart's electrical system. A dysfunctional electrical system often negatively impacts the heart's effectiveness as a pump. For example, atrial fibrillation results in the loss of atrial kick.

The SA (sinoatrial) node has the ability to self-initiate an electrical impulse. This ability, called automaticity, makes the SA node a pacemaker site for the heart. The fact that the SA node normally fires at rates greater than other pacemakers (60-100/minute) makes the SA node most often the dominant pacemaker.

The AV node also has the ability to initiate impulses, serving as a back-up in the case of SA nodal failure. The AV node significantly slows down the transmission of the electrical wave of depolarization, providing time for atrial kick prior to ventricular contraction.

The bundle of His carries the impulse from the AV node in the atria to the bundle branches in the ventricle. The bundle of His and the AV node, called the AV junction, can serve as a pacemaker at 40-60 beats/minute.

The bundle branches and the Purkinje network facilitate rapid depolarization throughout the ventricles. These electrical structures also can self-initiate impulses if necessary with typical rates of 20-40/minute. We referred to the bundle branches/Purkinje fibers as the Autobahn of the heart. Impulses that originate above the ventricles are associated with a narrow QRS complex.

Lastly, heart rate control was explored. Heart rate is the product of three factors: intrinsic impulse formation, sympathetic and parasympathetic stimulation. Sympathetic stimulation increases heart rate and contractility whereas parasympathetic stimulation slows heart rate and reduces contractility. Measuring heart rate is a useful sign when assessing a person's state of homeostasis.

Chapter Quiz

1. The Vagus nerve stimulates (circle all that apply):

a) the SA node
b) the AV node
c) the ventricles
d) the atria

2. Normally, the dominant pacemaker of the heart is the SA node.

True or False

3. Number the following structures in the expected order of electrical transmission beginning with the normal dominant pacemaker.

___ AV Node
___ SA Node
___ Bundle of His
___ Purkinje Network
___ Bundle Branches

4. The primary role of the bundle of His is to electrically connect the atria with the ventricle.

True or False

Answers: ***1.*** *a),b),d);* ***2.*** *True;* ***3.*** *SA node, AV node, Bundle of His, Bundle branches, Purkinje network;* ***4.*** *True*

5. The (sympathetic, parasympathetic) nervous system uses the chemical norepinephrine. The action of norepinephrine on the SA node is to (increase, decrease) its rate of firing. (Automaticity, Synchronicity) is the property of cells to self-initiate an electrical impulse.

6. The layers of the heart depolarize in what order? (Number the layers in order)

___ Epicardium
___ Endocardium
___ Myocardium

7. Ventricular depolarization occurs most rapidly if the impulse has a (ventricular, supraventricular) origin.

8. A QRS complex represents:

a) atrial depolarization
b) atrial repolarization
c) ventricular depolarization
d) ventricular repolarization

9. Because hearts that are transplanted are not innervated by either the parasympathetic or sympathetic nervous systems, an electronic pacemaker is required to keep the heart beating.

True or False

10. The (epicardium, endocardium) experiences a delay in repolarization. This causes the (epicardium, endocardium) to begin the process of repolarization.

11. The following structures are supraventricular (circle all that apply):

a) bundle branches
b) SA node
c) bundle of His
d) AV node
e) Purkinje network

12. The SA node is located near the juncture of the (right, left) atrium and the _____.

Answers: ***5.*** *sympathetic, increase, automaticity;* ***6.*** *endocardium, myocardium, epicardium;* ***7.*** *supraventricular;* ***8.*** *ventricular depolarization;* ***9.*** *False;* ***10.*** *endocardium, epicardium;* ***11.*** *b),c),d);* ***12.*** *right, superior vena cava;*

13. Depolarization is the same as contraction.

True or False

14. Pacemaker cells are normally located in the:

a) SA node
b) AV node
c) atrial myocardium
d) bundle branches
e) Purkinje network
f) bundle of His
g) ventricular myocardium

15. At rest, the interior of cardiac cells have a (positive, negative) polarity as compared with the cell exterior.

16. The atrioventricular (AV) junction serves the following functions:

a) protects the ventricles from overly rapid atrial rates
b) back up pacemaker during periods of SA nodal failure
c) slows conduction to allow time for atrial kick
d) the pathway between the atria and the ventricles
e) all of the above

17. The SA node is a cluster of hundreds of specialized cells that possess the ability to initiate impulses. An area of the heart that shares very similar cells is the:

a) the atria
b) the AV node
c) bundle branches
d) the ventricles

18. The skeleton of the heart is composed of connective tissue. This connective tissue occupies much of the septum as well as a plate that separates the atria from the ventricles. Because this connective tissue does not conduct electrical impulses, conducting cells burrow through to connect each of the heart's chambers. The structure that connects the atria electrically to the ventricles is called the:

a) SA node
b) Bachman's bundle
c) AV node
d) bundle of His

Answers: ***13.*** *False;* ***14.*** *a),b),d),e),f);* ***15.*** *negative;* ***16.*** *e);* ***17.*** *b);* ***18.*** *d);*

19. Pacemaker sites furthest from the SA node tend to produce the slowest rates.

True or False

20. The depolarization of the ventricles progress very rapidly due to (circle all that apply):

a) specialized Purkinje fibers
b) simultaneous depolarization of several waves across the ventricles
c) the bundle branches (Autobahn of the heart)
d) the muscle cells are able to contract much faster than skeletal fibers

21. Normally, more time is taken to depolarize the AV junction than the rest of the heart combined.

True or False

22. An impulse that originates in the myocardium tends to depolarize the ventricles (slower, faster) than if the impulse originated from a supraventricular focus.

23. Heart rate is influenced by (circle all that apply):

a) intrinsic ability of the pacemaker cells
b) automaticity
c) sympathetic innervation
d) parasympathetic innervation
e) epinephrine released from the adrenals

24. A cardiac rhythm that originates from the SA node is called a (circle all that apply):

a) sinus rhythm
b) atrial rhythm
c) supraventricular rhythm
d) ventricular rhythm

25. The cardiac effects of the sympathetic nervous system include (circle all that apply):

a) positive chronotropic effect
b) negative dromotropic effect
c) positive inotropic
d) proarrhythmic
e) positive dromotropic
f) negative chronotropic

Answers: ***19*** *True;* ***20.*** *a),b),c);* ***21.*** *True;* ***22.*** *slower;* ***23.*** *all of the above;* ***24.*** *a),c);* ***25*** *a),c),d),e)*

26. Vagal stimulation can help slow rapid ventricular dysrhythmias.

True or False

27. Examples of Vagal stimulation include (circle all that apply):

a) anal stimulation
b) sudden change of body position
c) face immersed in ice-cold water
d) carotid sinus massage
e) deep pain
f) vomiting

28. Supraventricular tachycardias can be slowed or terminated with _____ _____.

29. Sympathetic stimulation may be advantageous during periods of (low cardiac output, ischemia) and increase morbidity during episodes of (low cardiac output, ischemia).

30. Beta blockers reduce morbidity and mortality for those experiencing an acute myocardial infarction by as much as (10%, 20%, 30%, 40%).

Suggested Readings and Resources

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

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

HeartScape: The Anatomy of the Heart. SkillStat Learning Inc.
Web: http://www.skillstat.com/heartscapeDemo.html

The Heart: An Online Exploration. Web: http://sln.fi.edu/biosci/heart.html

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

Answers: ***26*** *False;* ***27.*** *all of the above;* ***28.*** *Vagal stimulation;* ***29.*** *low cardiac output, ischemia;* ***30.*** *40%*

What's Next?

It's time to apply your knowledge of normal cardiac anatomy and physiology to the skills of ECG interpretation. The next chapter is a primer on the components of an ECG. Learn about the cardiac monitoring system, ECG waveforms, segments and complexes. Also become skilled at quickly determining heart rate. Proficiency in rapid ECG interpretation is definitely within reach.