The Six Second ECG

A Practical Guide to Basic and 12 Lead ECG Interpretation

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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

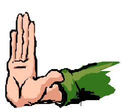
How to Use This Book

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.

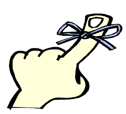
Each chapter is independent and can stand on its own. Read the book from cover to cover or jump around concentrating on what you need. Choose to complete the exercises and quizzes inside each chapter. Answers to the quizzes are provided at the base of the pages that contain the questions. Detailed annotated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

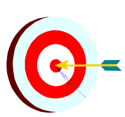


The 'stop' hand signal marks vital information often related to clinical practice.



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.

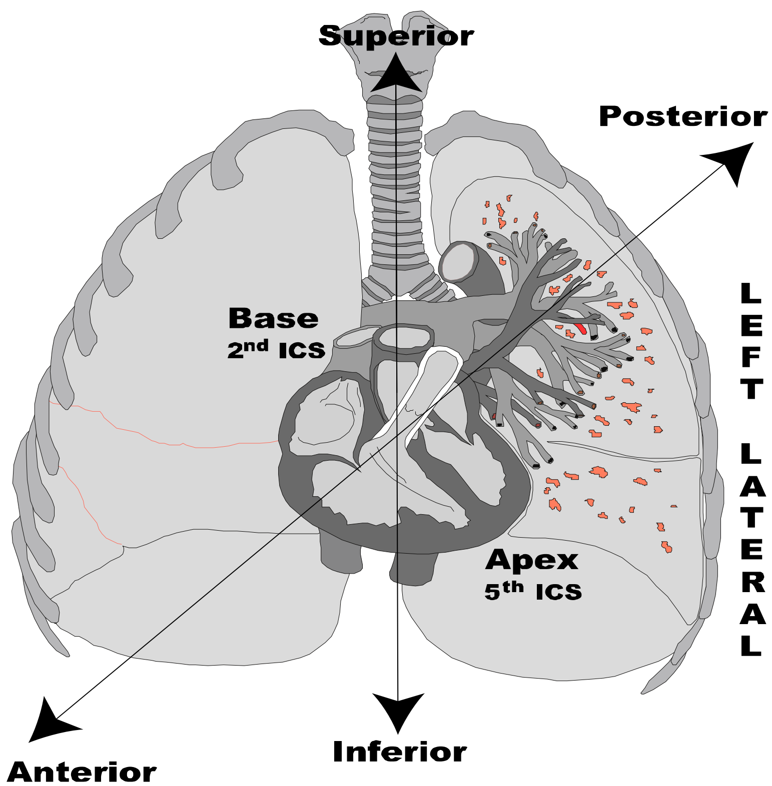
T.S. Eliot

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.

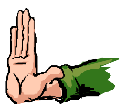
The inferior view of the heart includes the right ventricle and the heart's apex (left ventricle). About 40% of inferior MIs are right ventricular infarctions. The anterior and lateral (left lateral) views of the heart are of the left ventricle and left atrium.

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.

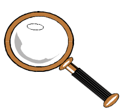
Layers

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.

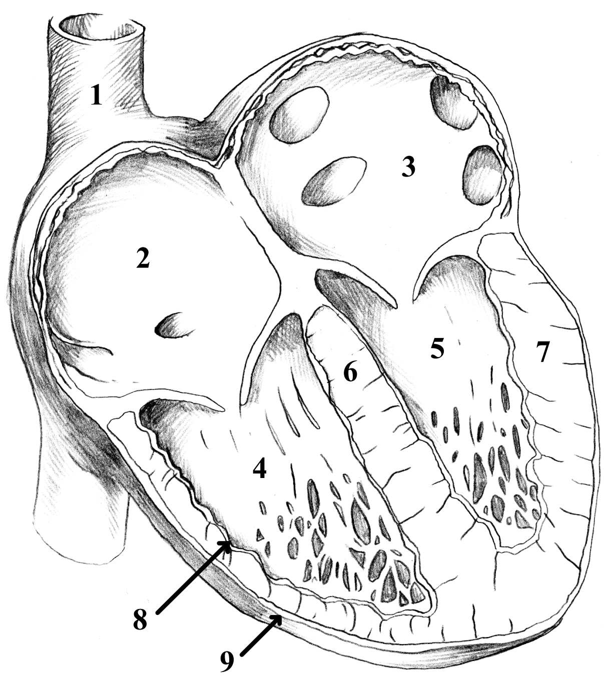
The heart is not just a pump but also an endocrine organ!

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



1. ____________________

2. ____________________

3. ____________________

4. ____________________

5. ____________________

6. ____________________

7. ____________________

8. ____________________

9. ____________________

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*

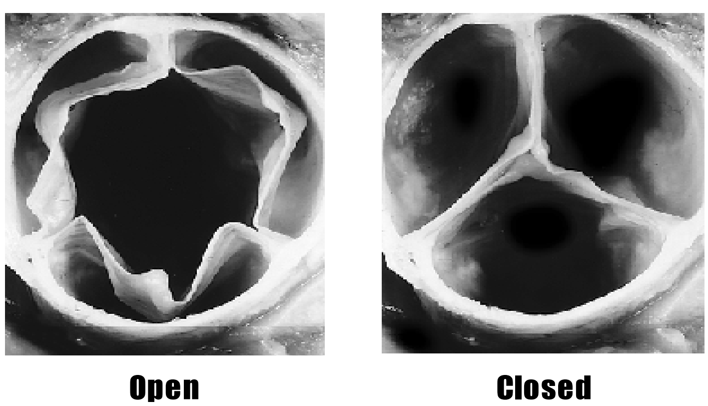
Heart valves ensure the forward flow of blood by closing off any back end routes. The atria do not share this advantage. The absence of valves between the venous system and the atria means that a small amount of blood is ejected back into the venous system with atrial contraction. With certain cardiac 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)

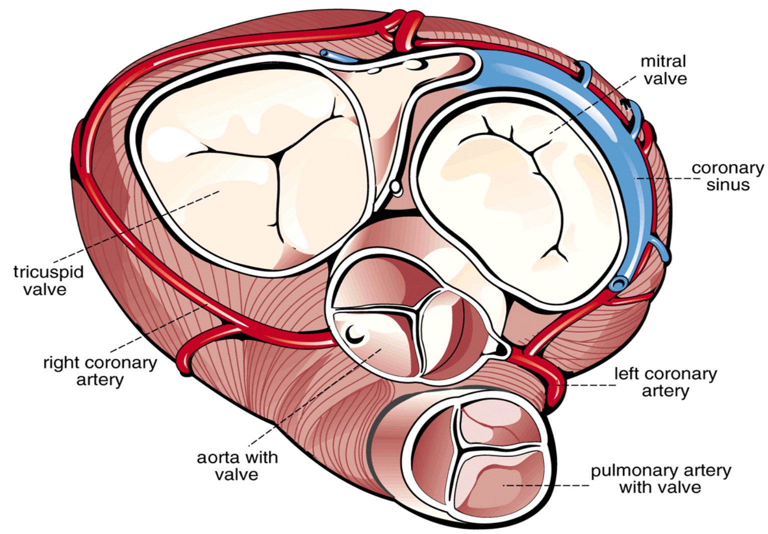


Figure 1.4 depicts the valves of the heart as viewed from above the heart.

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.

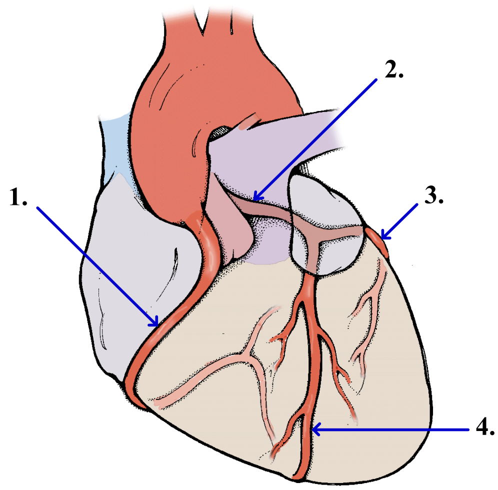
While heart murmurs may suggest valvular pathology, heart sounds also suggest normal function. The closing of the AV valves produce the classic **S1** sound, heard at the beginning of ventricle systole ('lub' of lub-dub). Subsequently, as the ventricles begin to relax (diastole), the semilunar valves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



1. ____________________

2. ____________________

3. ____________________

4. ____________________

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.

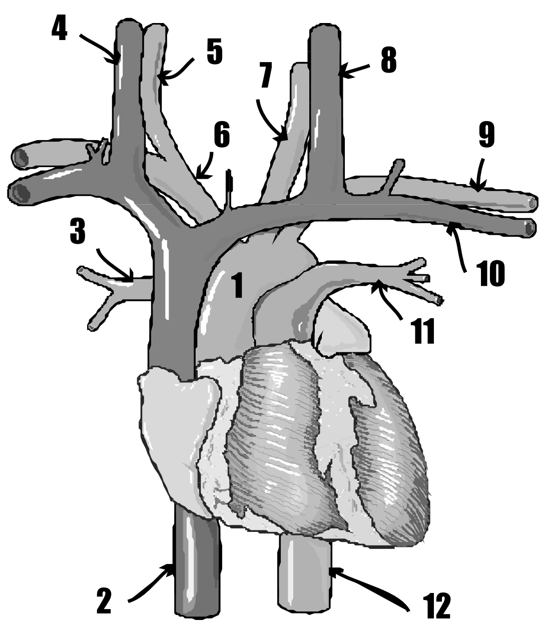
The **left main** begins at the left border of the aorta opposite the entrance to the RCA. The left main soon splits into 2 arteries: 1) the **circumflex** wraps around the surface of the left heart; and 2) the **left anterior descending** artery travels down the anterior surface of the left ventricle. 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.

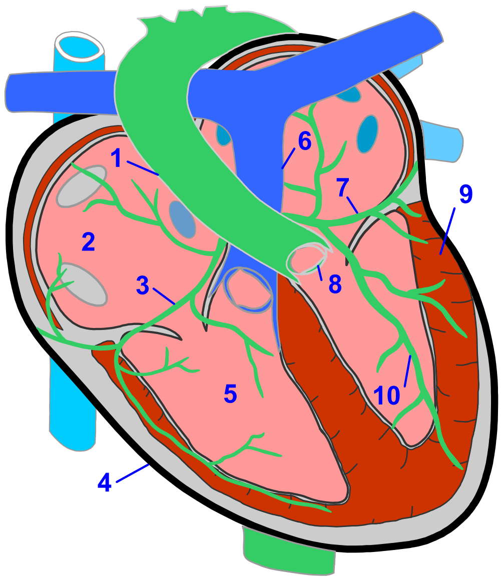
Valves act as gates in the flow of blood. They are located between the atria and ventricles as well as between the ventricles and the major arteries. The heart, being a specialized muscle, requires its own blood supply of oxygen and nutrients. This is provided by **coronary arteries**.

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.	Aortic Valve
2.	Epicardium
3.	Right Atrium
4.	Pulmonary Artery
5.	Right Ventricle
6.	RCA
7.	Aorta
8.	Myocardium
9.	LAD
10.	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!"

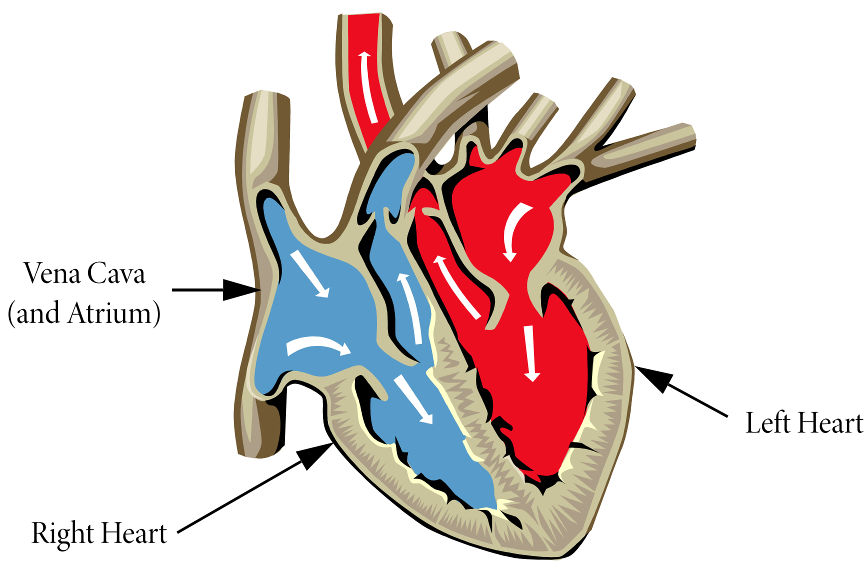
Not So Anonymous

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.

$\cancel{O_2}$ + Glucose = LACTIC ACID

$$\Downarrow$$

ONLY 2 ATP

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

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

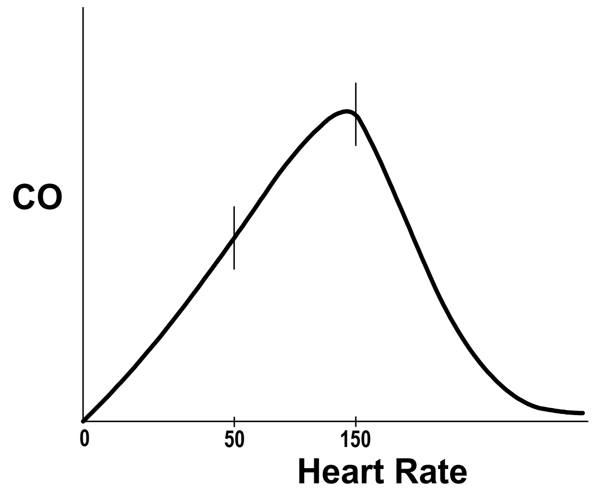
With this simplistic example, a 20% increase in heart rate (from 80 to 100/minute) yields a 20% increase in cardiac output (from 5200 ml to 6500 ml).

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



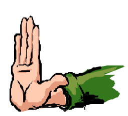
This graph illustrates the relationship between heart rate and cardiac output. As heart rate increases, so does cardiac output - to a point. Cardiac output tends to fall when heart rate surpasses 150/minute due to inadequate filling time. Low cardiac output states also occur with low heart rates (<50/minute). 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

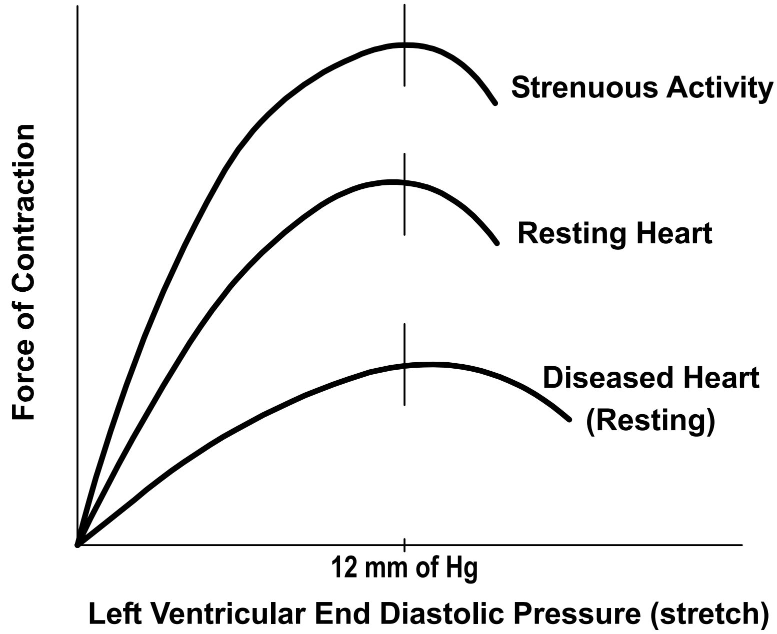


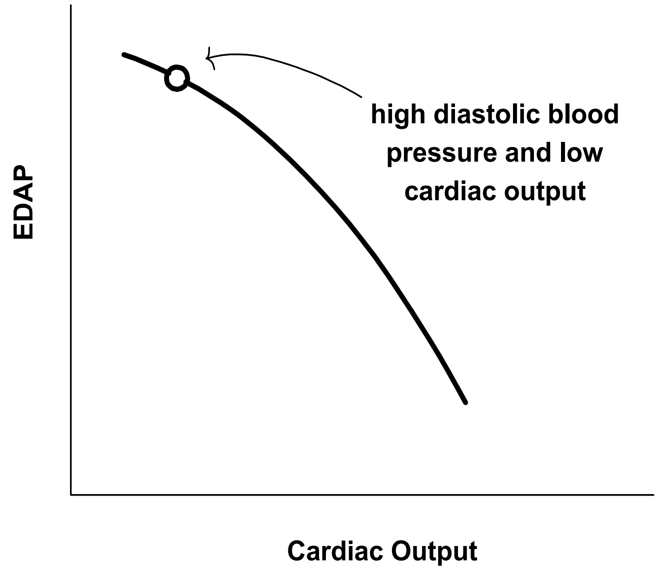
Figure 2.3 depicts the relationship between ventricular end diastolic pressure and contractility for a resting healthy heart, a resting diseased heart and a healthy heart during strenuous activity. Several points are evident here: ***1)*** *in general, the force of contraction (contractility) increases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

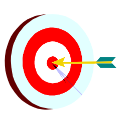
While systemic vascular resistance is not easily determined without a pulmonary artery catheter, diastolic blood pressure is easily measured. So while an accurate estimate of afterload is often not clinically practical, a patient's diastolic pressure provides a good indication of the resistance the left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

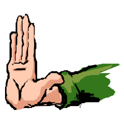
A heart rate of 100/minute helps to maintain an acceptable CO. Positioning Hank in semi-fowlers position further reduces the preload to his heart by using gravity i.e. blood pools in the abdomen and lower extremities rather than near his heart.

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*

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)

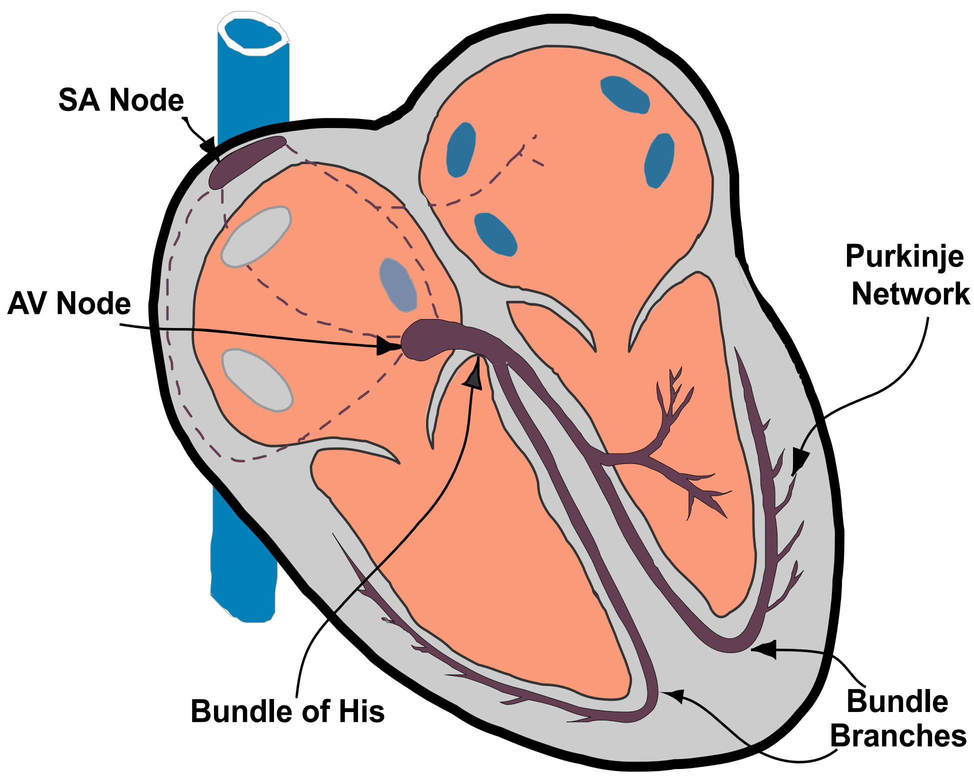
Electrical Overview

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

Pacemaker Site	Typical Rate/Minute
SA Node	60-100
AV Node	40-60
Bundle of His	40-60
Bundle Branches	30-40
Purkinje Network	20-40

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

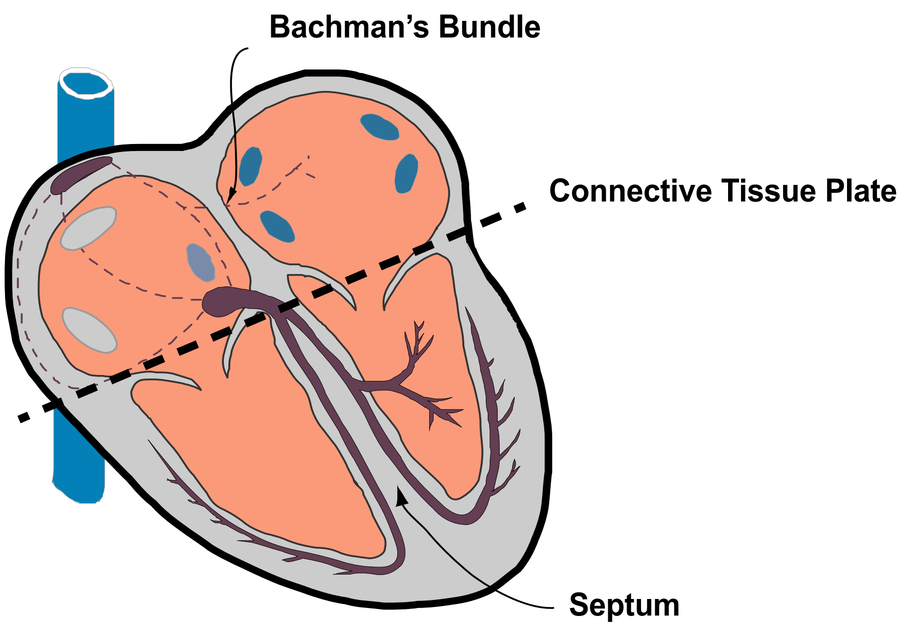


Figure 3.3 depicts the structures that form the cardiac skeleton, the septum and the plate of connective tissue that separates the atria from the ventricles. Connective tissue does not conduct electrical impulses, serving rather as an electrical insulator or barrier. To connect the left and right atria 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

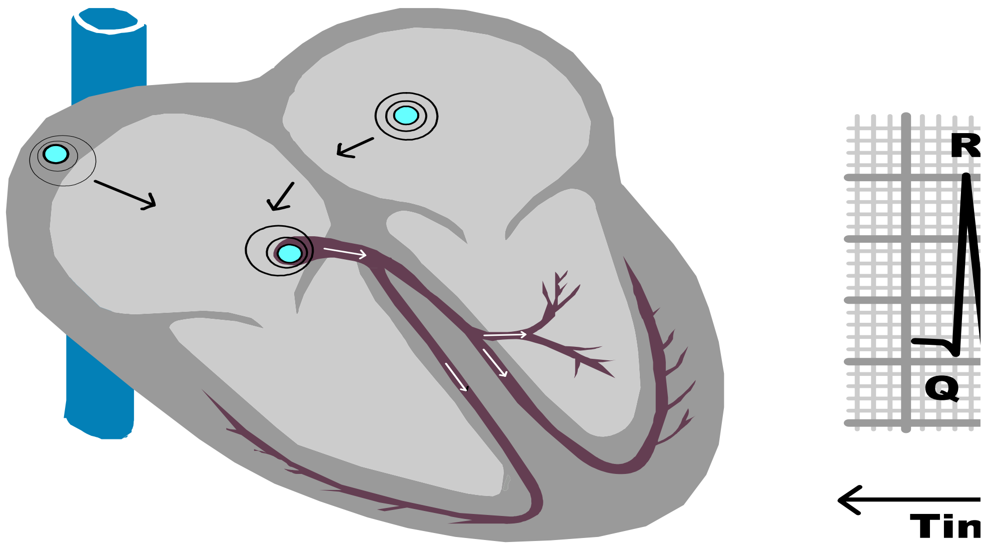


Figure 3.4 illustrates an impulse originating from the SA node, the atria or the junction. These supraventricular impulses are transmitted to the ventricles via the bundle branches, metaphorically referred to as the Autobahn. Since the Autobahn or bundle branches are used, the ventricles will depolarize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

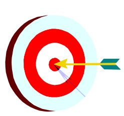
Asking the question, "Is the QRS wide or narrow" is an important step in ECG interpretation. A narrow QRS occurs when an impulse that originates from above the ventricles travels down the rapidly conducting bundle branches to depolarize the ventricles.

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

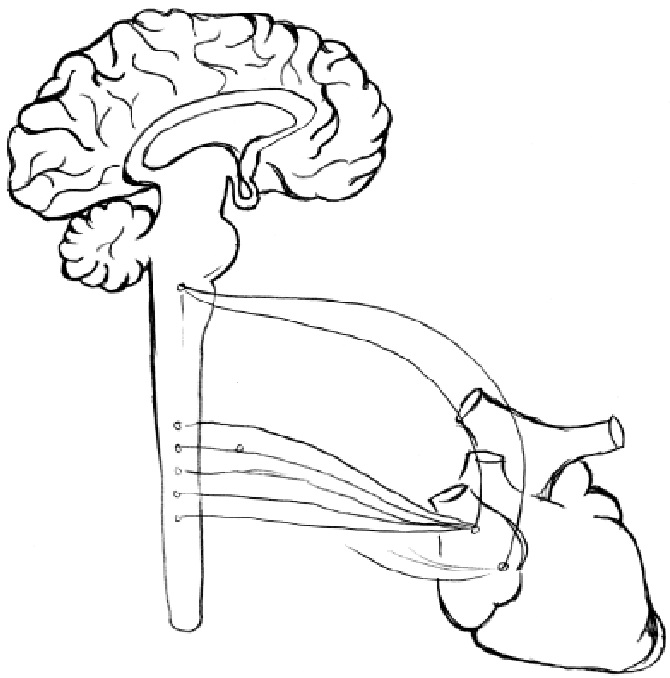
Heart rate, the numbers of beats or cardiac cycle per minute, is the result of three factors: intrinsic control by the heart's pacemakers, sympathetic stimulation and parasympathetic stimulation. The heart's pacemakers have their own intrinsic rate of impulse formation. For heart transplant recipients, 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.

4

An ECG Primer

Quick Look

The previous three chapters laid the ground work for the chapters that follow. In fact, two vital steps in rapid ECG interpretation have already been addressed: 1) Is the rhythm too fast or too slow? and 2) Is the QRS complex wide or narrow? ECG interpretation refers to both cardiac mechanics and the heart's electrics.

Except for a brief look at the QRS complex in the last chapter, this is our first foray into the realms of the ECG. On first glance, the ECG might appear formidable, mysterious and undecipherable. Don't believe it! This chapter, an ECG primer, quickly reveals the simplicity of the ECG.

This chapter begins with the cardiac monitoring system, particularly the three and five lead systems. Methods to determine heart rate are outlined and reinforced with practice exercises. The waveforms, intervals, segments and complexes of an ECG round out this chapter.

Reading an ECG can be fairly simple. The key is to find meaning in what you see on a cardiac monitor or on ECG paper. Making sense of the various components of the ECG is what this chapter is all about. Yes, it's about time!

I saw it, but I did not realize it.

Elizabeth Peabody

Cardiac Monitoring System

Since Dr. Willem Einthoven invented the first "electrokardiogram" in 1902, the electrical activity of the heart has been recorded. The ability to recognize a normal cardiac rhythm and recognize dysrhythmias didn't become common place in health care facilities until the 1960s.

Terminology
The electrocardiogram was initially called an **e**lectro**k**ardio**g**ram or EKG. While the term EKG continues to get a fair amount of use, "ECG" will be used in this book despite its similar sound to EEG. Also, the term arrhythmia is synonymous with dysrhythmia.

In the past 4 decades, great strides have been made in the recording and interpretation of electrocardiogram. Despite technological advances in cardiac diagnostics (i.e. electrophysiology studies), the electrocardiogram (ECG) remains a non-invasive, quick and effective diagnostic tool.

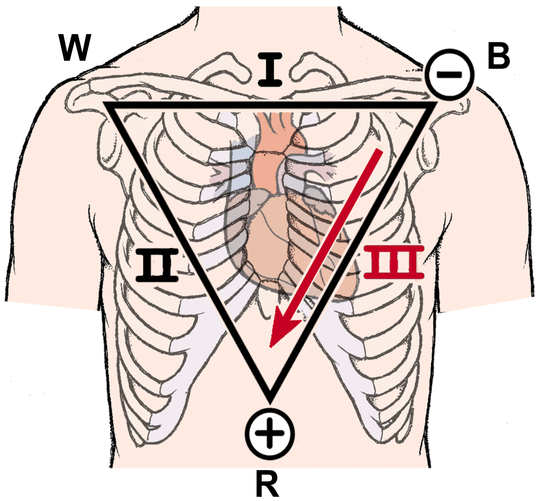
Your first exposure to an ECG might be compared to trying to make sense of a foreign language. Without any knowledge of the structure or rules, understanding is all but impossible. The good news is that the structures or components of an ECG can be quickly addressed. Also, the rules to understand and interpret an ECG are few and easily learned. The first step is to be able to capture electrical heart rhythms on a cardiac monitor and on paper.

The 3 Lead Wire ECG System (Einthoven's Triangle)

The three lead ECG has been around for some time. In fact, the three lead ECG recently celebrated its 100th birthday. While the five lead ECG is gradually becoming the norm, the three lead ECG continues to be used in emergency departments, telemetry monitoring, and during medical procedures. Three colored wires connect to three electrodes to form a triangle - Einthoven's triangle (see Figure 4.1 on page 65).

The three electrodes are colored white, black and red. These colors are not universal. Two coloring schemes exist for electrode placement, originating from two standards bodies: the American Heart Association (AHA) and the International Electrotechnical Commission (IEC). The coloring scheme followed in this book adheres to the standard advocated by the AHA (refer to Table 4.1 on page 65).

Figure 4.1 The 3 Lead Cardiac Monitoring System



W - white lead, always negative polarity

B - black lead, positive for lead I, negative for lead II

R - red lead, always positive polarity

Figure 4.1 depicts the standard three lead system that forms Einthoven's triangle. Note that while the red electrode is usually placed near the left lateral base of the chest, the electrical reference point for the red electrode tends to reside as shown. The arrow that is directed parallel to lead III represents a vector. If the wave of electrical depolarization moves parallel and in the same direction as this vector, the waveforms will be upright and the tallest in amplitude (this is covered later in this chapter).

For monitoring purposes with the three lead system, the white electrode is placed just below the clavicle (collarbone) on the right shoulder. When utilized - in leads I and lead II - the white electrode has a negative polarity. In accordance with the AHA, the end of the electrode cable is labelled "RA" for right arm.

Table 4.1 Electrode Location Standards of the AHA and the IEC

AHA (North America)		Location	IEC (Europe)	
Inscription	**Colour**		**Colour**	**Inscription**
RA	White	Right Arm	Red	R
LA	Black	Left Arm	Yellow	L
RL	Green	Right Leg	Black	N
LL	Red	Left Leg	Green	F
V1-6	Brown	Chest	White	C1-6

North America uses the AHA standards and Europe follows the IEC. Other regions are mixed. Knowing the standards in your region ensures correct electrode placement.

The red electrode, an electrode with positive polarity in lead in leads II and III, is connected below the left pectoral muscle near the apex of the heart. The end of the red electrode cable is usually labelled "LL" for left leg.

Electrodes are optimally placed directly on dry skin. Many electrode manufacturers stress: 1)shaving the skin if necessary; 2) removing dead skin cells by rubbing the area with a rough paper or cloth; 3) using electrodes from air tight packages; and 4) paying attention to expiry dates on the electrodes. While common practice may not place great importance on the later three items, these criteria may be especially useful when troubleshooting an unclear ECG tracing.

The black electrode is connected below the left clavicle near the shoulder. Often labelled "LA" for left arm, the black electrode switches polarity dependent on the lead chosen. With lead I, the black electrode becomes positive (white is always negative). The black electrode assumes a negative polarity in lead III.

Various mnemonics might help ensure correct lead placement. Two examples are:

White to the right. Red to the ribs. Black on top.

White to the right. Smoke (black) over fire (red).

Just the same, we can always just look for reference from the end of the electrodes and place them accordingly.

Electrodes are best connected to the skin in an area with minimal muscle activity. The cardiac monitor picks up any electrical activity, including any other muscle twitching in the vicinity. There is some question about whether the electrodes should be placed on bone, on muscle, under or over breasts. For dysrhythmia monitoring, electrodes should be optimally placed to get the clearest tracing. Changing electrode positions, though, by very little often changes the ECG. The key is consistency.

The three lead system provides three views of the heart. **Locating the positive electrode is crucial** to determining which area of the heart is viewed electrically. Metaphorically, the positive electrode serves as a mini-video camera aimed at the heart in the direction of the negative electrode. These leads and their corresponding electrodes do not sit right on the heart. Rather, their vantage points offer a surface, frontal view of the heart. Figure 4.2 depicts the regions viewed by each lead (next page).

Figure 4.2 The 3 Lead Views of the Heart

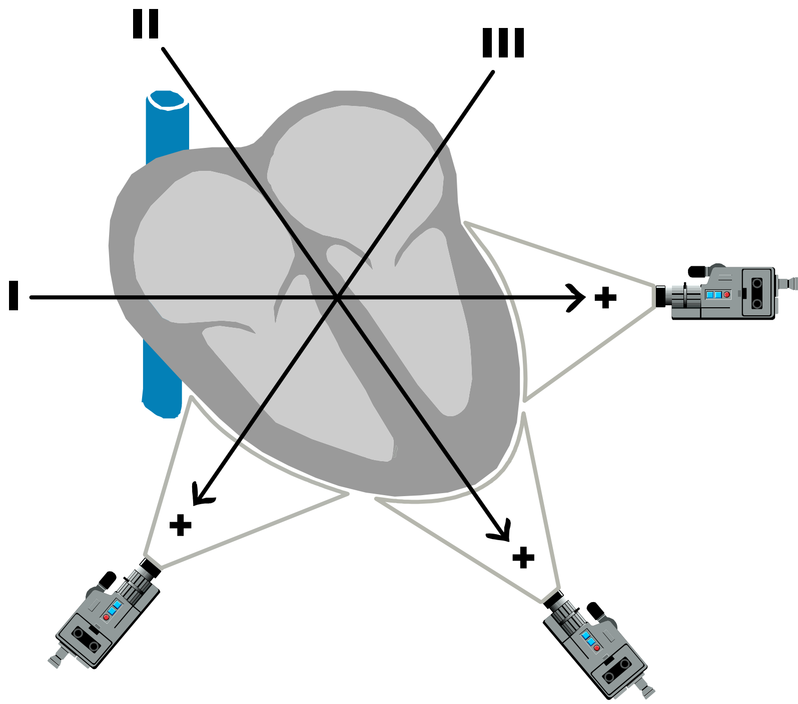


Figure 4.2 illustrates the views of the three lead system. Each of the leads view the heart from the perspective of the positive electrode towards the negative electrode.

Lead I provides a left lateral view of the heart. Perhaps the lead most often chosen for cardiac monitoring, lead II - an inferior lead - views the apex of the heart. Lead III also provides an inferior view.

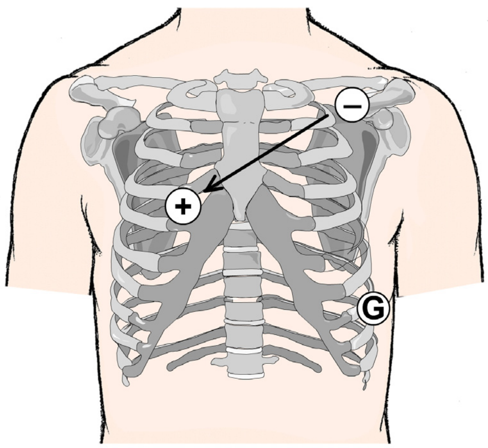
Table 4.2 The Chambers Viewed by the Three Lead System

Lead	Views	Heart Chambers
Lead I	Lateral	Left ventricle, left atrium
Lead II	Inferior	Left and right ventricle,
Lead III	Inferior	Right and left ventricles

Table 4.2 provides summarizes the chambers viewed by the three lead ECG system. Both leads II and lead III are inferior leads while lead I is a lateral lead. Each of the leads of the three lead system are bipolar leads since each lead has both a positive and a negative electrode. Note that the electrode that is not polarized (positive or negative) for each lead serves instead as a ground electrode (i.e. the black electrode with lead II).

Einthoven's three lead system is the most commonly used three lead cardiac monitoring system today. Some centers, though, choose to use a modified central lead (MCL_1) as part of the three lead system. This is sometimes also referred to as the modified *chest* lead. This lead is often used together with lead II to help distinguish between supraventricular and ventricular tachycardias.

Figure 4.3 Modified Central Lead (MCL_1)



The modified central lead, called MCL_1, is established with the cardiac monitor set to lead I, with the white electrode a negative polarity, the black electrode a positive polarity (the red electrode serves as the ground). Upon examining the 12 lead ECG in chapter 7, lead MCL_1 will be revealed to be similar to the precordial lead V_1. Note that lead V_1 is superior to lead MCL_1 as a diagnostic lead for dysrhythmias and for myocardial ischemia monitoring.

The modified central lead is created by placing the positive electrode at the 4th intercostal space just to the right of the sternum and the negative electrode below the left clavicle near the shoulder. The ground electrode (red) electrode can be placed anywhere on the body.

Whether the standard or a modified version of the three lead system is used, there is an important point to consider. Practically any lead will suffice for dysrhythmia monitoring. For tasks such as myocardial ischemia monitoring, though, each lead provides information *specific only to the region viewed*. For example, Lead I can provide signs of left ventricular ischemia, but only rarely signs of right ventricular ischemia.

For example, take a patient experiencing cardiac ischemia of the inferior region. In all leads, a sinus rhythm is identified. But evidence of ischemia - typically found in the inferior leads II and III, may be absent in lead I. If only lead I was monitored, this ischemic event would most likely be missed entirely (about 70-80% of all ischemic episodes occur with no symptoms). The bottom line: **the more lead views monitored, the better.**

The 5 Lead Wire ECG System

In the 1990s, research papers challenged the efficacy of lead II for dysrhythmia monitoring. One paper reported that supraventricular tachycardia (SVT) and ventricular tachycardia (VT) was correctly identified only about a third of the time when using lead II. If a 12 lead ECG was used, correct identification of either SVT or VT occurred 90% of the time. A single lead II continues to be commonly used today.

Lead V_1 of the 12 lead ECG was found to be the single best lead. Lead V_1 most often displayed the waveforms required to correctly distinguish between SVT and VT. Since lead MCL_1 is purported to provide a similar view to lead V_1, these two leads were compared. Lead V_1 came out on top, with ECG interpretation using lead MCL_1 arriving at an incorrect diagnosis for either SVT or VT about 22% of the time. Clearly, concurrent display of multiple lead views, including lead V_1, is advantageous.

Lead V_1 is addressed in more detail in chapter 7, The 12 Lead ECG. Lead V_1 is a unipolar lead that views the heart directly below. This lead is created by the combination of electrodes to simulate a negative pole at the anatomical centre of the heart, with the positive electrode located at the 4th intercostal space placed just right of the sternum. As a result, lead V_1 views the heart directly below the position of this positive electrode. In order to provide a true lead V_1, a 5 lead wire system is required.

Since the release of these findings, cardiac monitors have been developed with the ability to view multiple leads simultaneously. While 3 lead wire systems continue to be provided as standard equipment, most ECG monitors come with optional 5 lead wire systems.

These newer cardiac monitors come with several advantages. As mentioned, multiple lead views offer a more comprehensive electrical picture of the heart. For tasks such as ischemia monitoring, multiple lead views increase the likelihood that ischemic episodes are detected. For dysrhythmia monitoring, the case for multiple lead views is well established. To combine the old standard lead II with the preferred lead V1 requires at a 5 lead wire system, a useful option with the new cardiac monitors.

In addition, the multiple view monitor can provide a more balanced electrical representation of both the right and left sides of the heart. While lead II can provide a good view of the left ventricle (apex), lead V_1 can provide a sternum and a partial right-sided view of the heart. Several possible electrode configurations exist. One example of a 5 lead wire ECG schematic is provided in Figure 4.4 on the next page.

Figure 4.4 Electrode Placement for a Standard 5 Lead ECG System Using Lead V_1

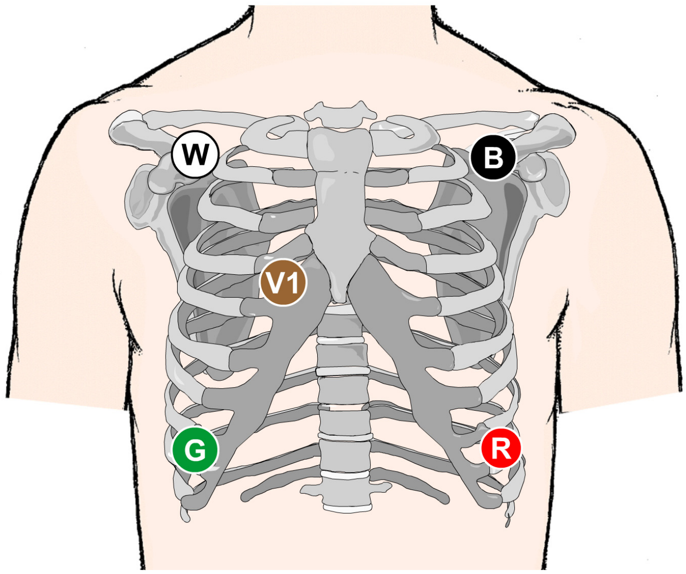
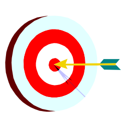


Figure 4.4 illustrates a standard 5 lead wire ECG configuration. The five electrodes includes four limb electrodes and one precordial chest lead (V_1). The limb leads are identical to the limb leads of a 12 lead ECG: right arm (RA), left arm (LA), right leg (RL) and left leg (LL).

Connecting a patient to a 5 lead wire ECG system is simple. The white, black and red electrodes are located in identical positions as with the 3 lead ECG. The green electrode is located opposite the red electrode. The brown precordial lead V_1 is located to the right of the sternal border at the 4th intercostal space (ICS). All but lead V_1 is created with four limb electrodes that would be placed on the wrists and ankles for the 12 lead ECG. The electrodes are placed on the torso for convenience.



The four limb electrodes - right arm, right leg, left arm and left leg - are commonly placed on the patient's wrists and ankles when taking a 12 lead ECG. For monitoring purposes, electrodes connected to the wrists and ankles are less than convenient as the lead wires would prove cumbersome. Instead, the limb electrodes are placed on the torso near the junction of each limb. This placement tends to reduce motion artifact caused by loose electrodes Since the limb electrodes in a 5 lead wire system are placed closer to the heart than the placement for a 12 lead ECG, the ECG rhythm from each lead system is seldom identical.

Other 5 lead electrode configurations are also used. For example, lead V_1 can be switched to lead V_5 to monitor the lateral left ventricle and atrium when suspecting a troublesome circumflex artery. The electrode for lead V_5 is placed at or just below the

5th intercostal space at the anterior axillary line (refer to Figure 4.5). Often the precordial lead used (V_{1-6}) depends on a patient's history and the chosen monitoring task (i.e. dysrhythmia and/or ischemia monitoring).

Figure 4.5 Electrode Placement for a Standard 5 Lead ECG System Using Lead V_5

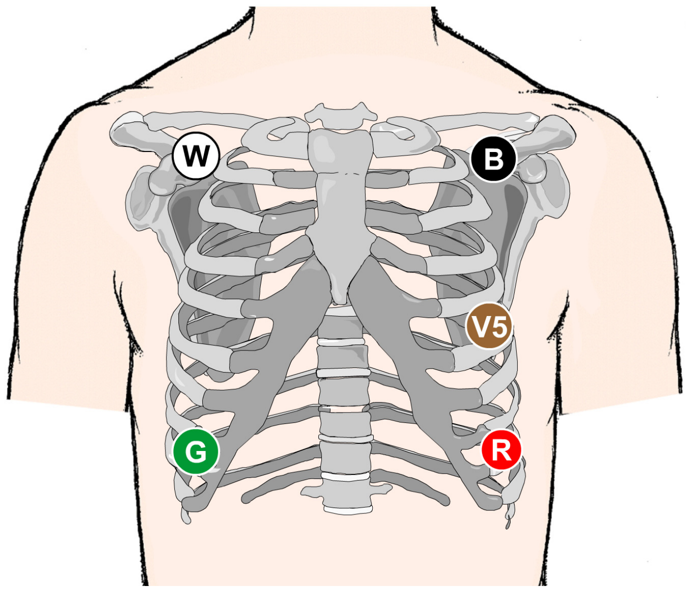


Figure 4.5 depicts a 5 lead wire ECG system that uses the four limb electrodes and the precordial lead V_5. Lead V_5 is positioned to monitor the lateral aspect of the left ventricle.

The 5 lead wire ECG systems mentioned thus far provide 5 lead views. As mentioned earlier, a 12 lead ECG is a superior diagnostic tool both for dysrhythmia monitoring and for other tasks such as ischemia monitoring. While some cardiac monitor manufacturers include a 10 wire cable to enable real-time 12 lead ECG monitoring, these cables tend to be bulky and restrictive.

In an effort to reap the advantages of 12 lead views while keeping the monitoring system practical, reduced-lead set technologies have been developed using five or six lead wires. Phillips Medical Systems developed the EASI™ lead system using a 5-cable system to estimate a 12 lead ECG. General Electric Medical Systems developed the interpolated 12 lead system using a 6-cable system. Refer to Figures 4.6 and 4.7 on the next two pages for schematics on each of these reduced-lead set technologies.

The EASI™ lead system uses a 5 cable connector. The electrodes are configured as follows:

- the brown electrode (labelled **E** on the electrode) is located along the sternum between the white and red electrodes, horizontal with the 5th ICS;

- the red electrode (labelled **A** on the electrode) is located opposite the white electrode, at the left mid-axillary line at the 5th ICS;
- the black electrode (labelled **S** on the electrode) is located along the upper sternum below the sternal angle;
- the white electrode (labelled **I** on the electrode) is located at the right mid-axillary line at the 5th ICS;
- the green or ground electrode can be positioned at a convenient location anywhere on the torso.

The EASI^{TM} lead system has a well established track record, being utilized by the Zymed and Agilent cardiac monitors among others.

Figure 4.6 The EASI^{TM} Lead System

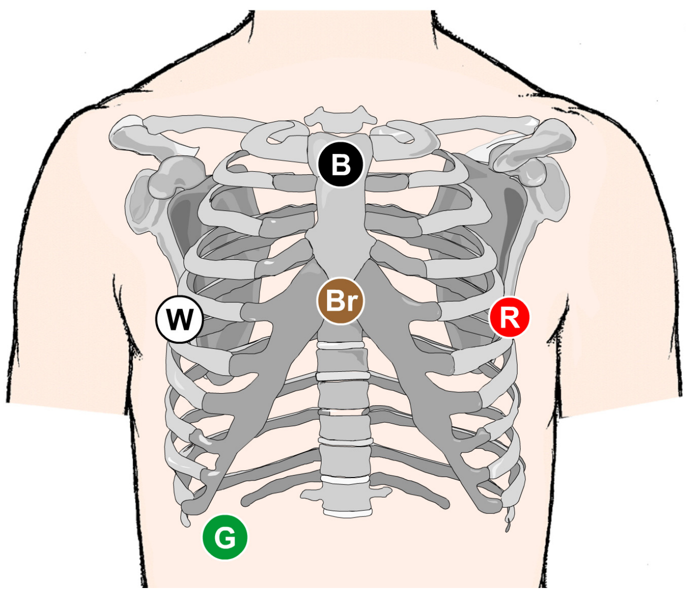


Figure 4.6 illustrates the EASI^{TM} lead system. The electrodes which are labelled according to the acronym EASI are colored as shown: B-black, Br-brown, W-white, R-red, and G-green.

The interpolated 6 wire ECG system can also deliver real-time 12 lead ECG monitoring. The electrode configuration is close to the standard 5 lead wire ECG system with four limb electrodes but with two precordial electrodes located at lead V1 and lead V5 (refer to Figure 4.7 on the next page). The interpolated lead system is offered with Marquette cardiac monitoring systems.

Figure 4.7 Interpolated 12 Lead System Using a 6 Wire Cable

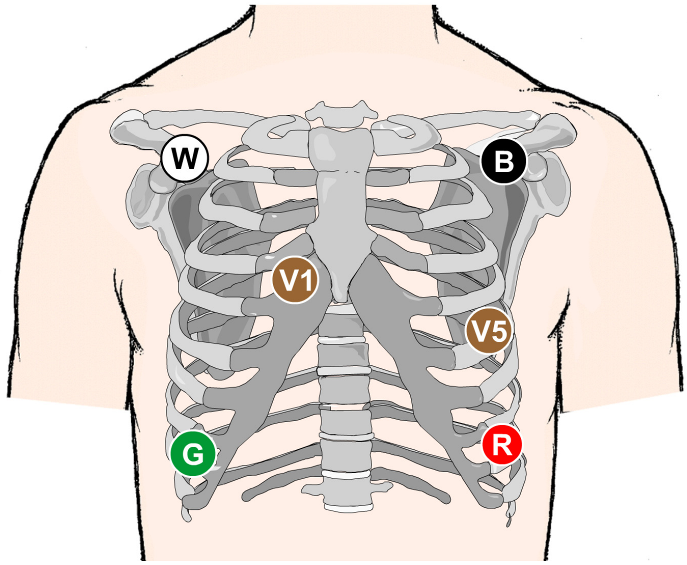


Figure 4.7 illustrates General Electric Medical Systems' interpolated 12 lead ECG cardiac monitoring system. This reduced lead set technology estimates a real-time 12 lead ECG system.

Regardless of the ECG lead system used, effective dysrhythmia and ischemia monitoring is possible only with quality ECG tracings. The use of fresh electrodes and the preparation of the patient's skin are all important steps to reduce motion artifact. Accurate placement of the ECG electrodes ensure consistent ECG readings that are worthy of comparison.

If at all possible, take advantage of multiple lead views including lead V_1. Multiple lead views, particularly the reduced-lead set 12 lead ECG, are superior to a single lead view whatever the purpose of ECG monitoring. Before moving ahead, challenge yourself to a flash quiz on the cardiac monitoring system.

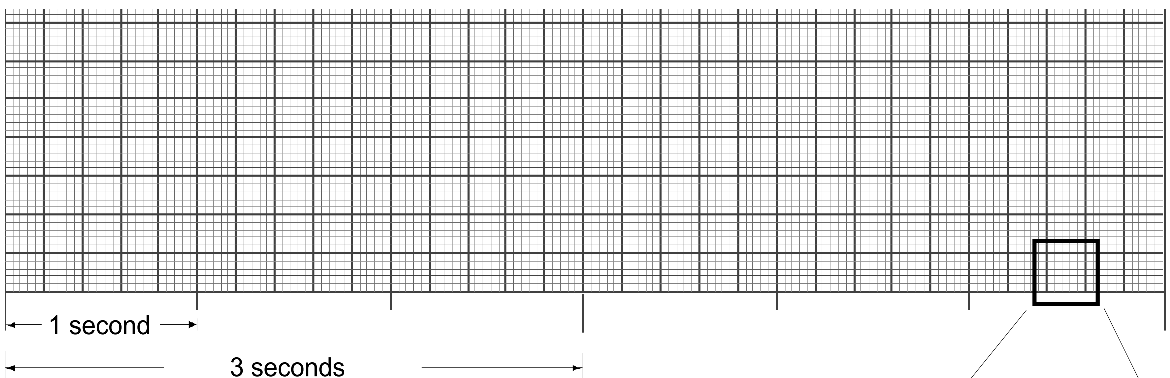
ECG Paper

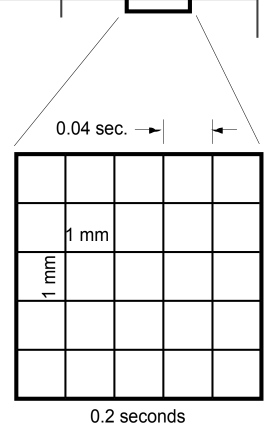
Cardiac monitors usually offer two mediums to help identify ECGs: the dynamic ECG viewed on the monitor's display screen and the static ECG printed on paper. While the screen of a cardiac monitor is useful for recognizing and interpreting rhythms, printing out the ECG on specialized paper provides a much more robust electrical picture of the heart. ECG paper allows for concise measuring of rates, intervals, segments and waveforms.

An ECG is a graphical display of electrical energy generated by the heart over time. ECG graph paper records cardiac electrical activity at a rate of 25 mm/second. The paper is divided into small 1 mm squares with thicker lines every 5 mm.

It follows then that the width of an ECG tracing is a **measurement of time**. For example, a horizontal accumulation of 25 small 1 mm squares measures electrical activity over one second (as does 5 large squares produced by the thick lines). A six second strip contains a cross-section of 150 small 1 mm squares (6 seconds x 25 small squares/second) or 30 large squares.

Figure 4.8 ECG Paper and the Measurement of Time





1 mm width = 1/25 second = 0.04 seconds = 40 milliseconds

5 mm width = 5/25 second = 0.20 seconds = 200 milliseconds

Figure 4.8 provides details of ECG paper. Note the larger hash marks that indicate 3 second intervals. Two 3 seconds intervals (6 seconds) is often used to determine heart rate.

Most brands of ECG paper also include hash marks that extend the thicker vertical lines to provide a visual reference for periods of one second and three seconds. This is depicted in Figure 4.8. These hash marks occupy either the top or bottom of the ECG paper strip.

The height (amplitude) measures electrical **voltage**. If calibrated properly, a waveform with a height of 10 mm equals 1 millivolt. The comparative height or depth of waveforms can yield significant insight about the heart (i.e. ischemia monitoring).

Flash Quiz 4.1

1. The 3 lead wire system is used to create which lead views (circle all that apply):

a) lead I
b) lead II
c) lead III
d) lead MCL_1
e) lead V_1

2. In North America, the right arm limb electrode is colored:

a) black
b) white
c) brown
d) red

3. In North America, the red cable/electrode has a (positive, negative) polarity.

4. With lead I, the cables (electrodes) used are the ____________________ cable and the ______________________ cable.

5. To minimize motion artifact, electrodes should (circle all that apply):

a) be taken from vacuum sealed packages
b) be moistened with saline prior to placement
c) be further reinforced with tape or glue
d) be placed on skin that has been cleared of dead cells and hair

6. Lead II is well established as the best lead for dysrhythmia monitoring.

True or False

7. Electrodes should never be placed over breast tissue or bone.

True or False

Answers: ***1.*** *a),b),c),d)* ***2.*** *b);* ***3.*** *positive;* ***4.*** *white, black;* ***5.*** *a),d);* ***6.*** *False;* ***7.*** *False;*

8. A white electrode is placed near the left shoulder below the clavicle. A black electrode is placed at the 4th intercostal space to the right of the sternum. In a 3 wire ECG system, the lead created is called:

a) lead I
b) lead II
c) lead III
d) lead MCL_1

9. The EASI reduced-lead set can provide 12 ECG views.

True or False

10. An ECG lead views the heart from the perspective of the (positive, negative) electrode looking towards the (positive, negative) electrode.

11. Each horizontal millimeter of ECG paper measures (circle all that apply):

a) 0.20 seconds
b) 0.04 seconds
c) 1/25 of a seconds
d) 0.1 millivolt

12. Each vertical millimeter of ECG paper measures (circle all that apply):

a) 0.04 seconds
b) 1 millivolt
c) 0.1 millivolt
d) amplitude of cardiac electrical activity

13. Thicker lines divide the ECG paper into larger (5 mm,10 mm,15 mm) squares that equal a period of (0.04, 0.01, 0.2, 1) seconds.

14. The speed standard for the printing of ECG paper is 25 mm per second.

True or False

15. If a QRS complex is present once every 5 large squares, the ventricles depolarize at a rate of (30/minute, 45/minute, 60/minute, 90/minute).

Answers: ***8.*** *d);* ***9.*** *True;* ***10.*** *positive, negative;* ***11.*** *b),c);* ***12.*** *c),d);* ***13.*** *5mm, 0.2;* ***14.*** *True;* ***15.*** *60/minute*

Lead Polarity and the Direction of Waveforms

Before we examine the waveforms expected on any ECG, a brief account of vector theory is required to make sense of these waveforms. A depolarizing wave moves through the myocardium *on average* along a trajectory or vector. A **vector** is a force moving in a direction symbolized by an arrow. The larger the force, the larger the arrow.

For example, an impulse initiated by the SA node moves towards the AV node and the left atrium. On average the depolarizing wave travels down and to the left. Atrial depolarization, then, has a vector that points down and towards the left. This average vector is the **electrical axis** of atrial depolarization (refer to Figure 4.9).

Figure 4.9 Vectors and the Electrical Axis

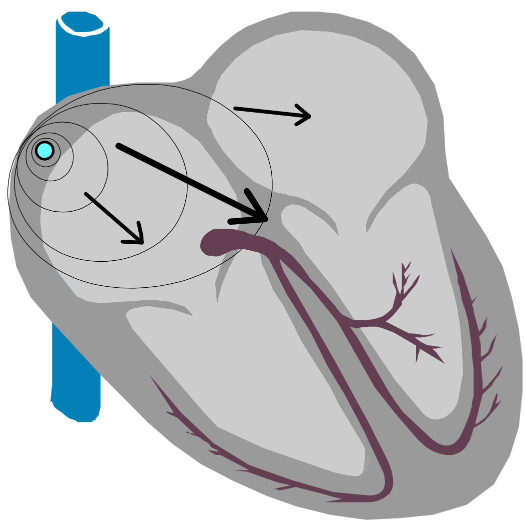


Figure 4.9 provides a graphical example of contributing electrical vectors (small arrows) that average to form the mean direction of depolarization, known as the electrical axis (large arrow). It is common for the electrical axis of the atria to point down and to the left.

The depolarization of the myocardium is represented on an ECG by a series of waveforms, one for atrial depolarization and soon after a larger waveform for ventricular depolarization. While an in depth discussion of each of these waveforms is forthcoming, an important characteristic shared by all ECG waveforms is the direction of a wave. Is the waveform upright or is it pointing downwards? A simple observation but one packed with significance.

Every lead view of an ECG has a positive electrode. As mentioned earlier, the heart is viewed electrically from the vantage point of the positive electrode. The positive electrode is important for another reason as well. **A depolarizing wave travelling towards the positive electrode produces an upright waveform.** This principle is pivotal in the quest to fully understand the ECG.

A depolarizing electrical wave that is directed towards a positive lead produces an upright waveform on an ECG. Conversely, an inverted waveform results when an depolarizing wave moves away from a positive lead.

Take an ECG tracing from the bipolar lead II. The positive red electrode is located near the apex of the heart. As a result, the apex of the heart is best viewed by lead II. Consider as well the depolarizing atrial wave (P wave) with respect to this positive red electrode. A depolarizing wave travelling from the SA node out to the left atrium and the AV node is directed towards the positive electrode in lead II. The P wave produced on lead II, then, would be upright (refer to Figure 4.10).

Alternately, an impulse originating from the AV junction depolarizes across the atria away from the positive red electrode. A resulting inverted P wave provides compelling evidence that this is a junctional rhythm initiated by the AV junction.

Figure 4.10 Atrial Depolarization, Vectors and the P Wave in Lead II

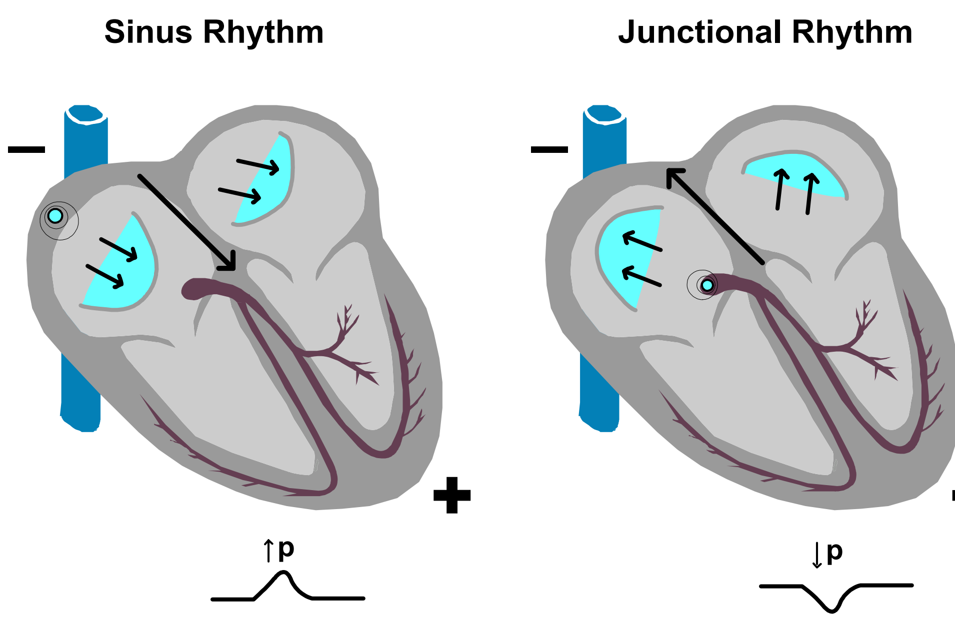


Figure 4.10 illustrates how the direction and shape of the P wave helps to locate the site of an originating supraventricular impulse. Lead II has its positive electrode near the heart's apex.

Ventricular depolarization moving towards a positive electrode also produces an upright waveform. The resulting waveform, though, is often more complex than the P wave produced by atrial depolarization. Ventricular depolarization (QRS complex) normally traverses three or four areas of the ventricles simultaneously thanks to the bundle branches.

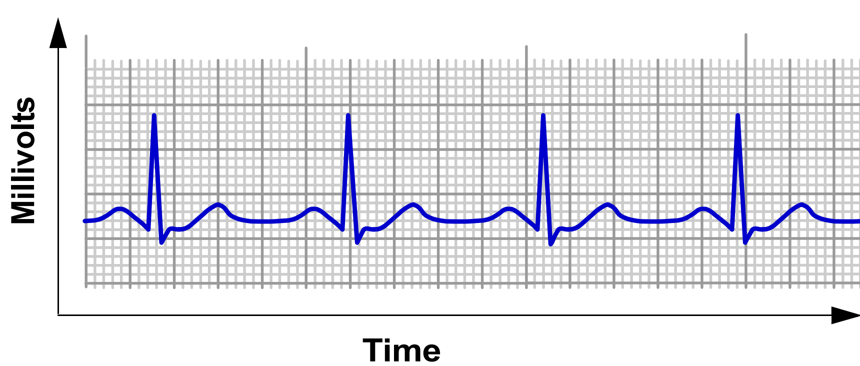
The QRS complex would likely be called just a Q wave if ventricular depolarization resulted from only one wave like what occurs in the atria. Kidding aside, the *direction* of the QRS complex is usually only considered with 12 lead ECG interpretation particularly when determining the electrical axis of ventricular depolarization (more on this in chapter seven). For single and dual lead views, only the *width* of the QRS complex is useful for rapid ECG interpretation.

The direction of the P wave, though, is an important consideration for rapid ECG interpretation. Based on a principle of vector theory, the P wave can provide insight into the location of an impulse that originates above the ventricles. Equipped as well with the knowledge that a narrow QRS complex is produced by a supraventricular impulse, the identification of supraventricular rhythms is definitely within reach.

Basic ECG Components

Having reviewed the cardiac monitoring system, ECG paper and vector theory, we are set to make sense of the basic ECG components. The ability to interpret an ECG relies heavily on a good understanding of these components.

Figure 4.11 The Scales of the ECG



An ECG is composed of a series of waves and lines usually ordered into some repeatable pattern. The waves and lines are displayed on either a two dimensional screen or on ECG paper. As mentioned earlier, the height of the tracing represents millivolts while the width of the ECG addresses an interval of time (see Figure 4.11).

Table 4.3 Normal and Abnormal Parameters of ECG Components

ECG Components	Normal Parameters	Abnormal Parameters	Causes of Abnormal Parameters
P Wave	Upright in most leads including lead II. Duration: < 0.11 seconds Amplitude: 0.5-2.5 sec.	Inverted Notched or tall	Junctional Rhythm Atrial rhythm, atrial hypertrophy
PR Interval	Duration: 0.12 - 0.20 sec.	Duration: shorter or longer than normal	Junctional rhythm, Wolff-Parkinson-White syndrome
Q Wave	Duration: <0.04 seconds Amplitude: <25% the amplitude of the R wave	Duration: 0.04 sec. or longer Amplitude: at least 25% the amplitude of the R wave	Myocardial infarction
QRS Complex	Upright, inverted or biphasic waveform Duration: < 0.11 seconds Amplitude: 1 mm or more	Duration: 0.11 second or more	Bundle branch block, ventricular ectopic i.e. PVC
QT Interval	Duration: less than 1/2 the width of the R-R interval	Duration: at least 1/2 the R-R interval	Long QT syndrome, cardiac drugs, hypothermia, subarachnoid hemorrhage Short QT associated with hypercalcemia
ST Segment	In line with PR or TP segment (baseline) Duration: shortens with increased heart rate	Deviation of 0.5 mm or more from baseline	Cardiac ischemia or infarction, early repolarization, ventricular hypertrophy, digoxin dip, pericarditis, subarachnoid hemorrhage
T Wave	Upright, asymmetrical and bluntly rounded in most leads Duration: 0.10-0.25 sec. Amplitude: less than 5 mm	Peaked, inverted, biphasic, notched, flat or wide waveforms	Cardiac ischemia or infarction, subarachnoid hemorrhage, left-sided tension pneumothorax, left bundle branch block, hyperkalemia, hypokalemia
U Wave	Upright Amplitude: < 2 mm	Peaked or Inverted Amplitude: > 2 mm	Hypokalemia, cardiomyopathy, ventricular hypertrophy, diabetes, digoxin, quinidine

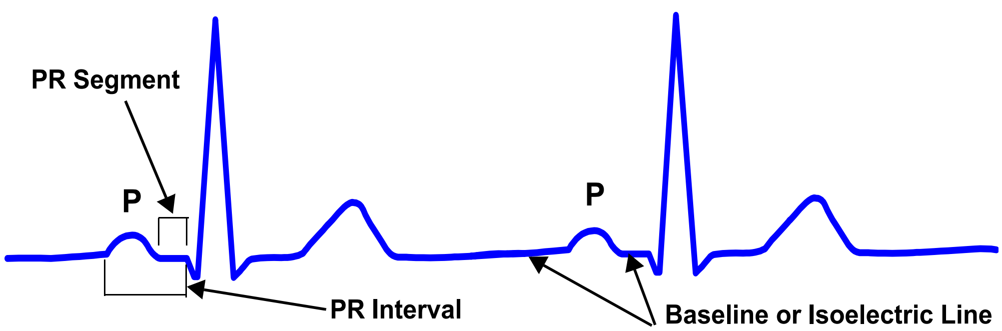
Table 4.3 outlines the parameters that define normal and abnormal ECG components. An incomplete list of possible causes of the abnormal waves, intervals and segments is included.

This section on ECG components addresses each of the waves, intervals and segments of an ECG in the order that they would appear. While waves are fairly self-explanatory, **intervals** measure time from the start of one wave to the start of another wave (an interval includes at least one wave) and **segments** measure time between waves (waves are not included in a segment). Table 4.3 outlines the parameters that are expected of normal and abnormal ECG waves, segments and intervals.

The P Wave, PR Segment and PR Interval

The **P wave** represents the depolarization of the right and left atria. The P wave begins with the first deviation from **baseline** and finishes when the wave meets the baseline once again. While the P wave is an electrical representation and not mechanical, a P wave strongly suggests that the atria have followed through with a contraction.

Figure 4.12 The P Wave, PR Segment and PR Interval



In Figure 4.12, several ECG components are labelled. Note that a waveform is produced when the electrical potential of cardiac cell membranes change. During atrial depolarization, the atrial cell membranes quickly become more positive producing a P wave. The baseline or isoelectric line represents nothing more than an absence of voltage change to the cardiac cells.

The **PR segment** is the line between the end of the P wave and the beginning of the QRS complex. The PR segment signifies the time taken to conduct through the slow AV junction. This delay allows for atrial kick. The PR segment also serves as a benchmark for the isoelectric line.

The **baseline** or **isoelectric line** is a reference point for the waves, intervals and segments. While the PR segment is often used as the baseline, the TP segment - between the end of the T wave and the beginning of the P wave - is now generally seen as a more accurate baseline. Note: with fast rates, the TP segment disappears, leaving you with the PR segment as the next best, and only, baseline.

The **PR interval** is measured from the start of the P wave to the start of the QRS complex. While it might appear obvious that this is indeed a PQ interval, a Q wave is not always present on an ECG tracing. For consistency, the term is PR interval has been adopted whether a Q wave exists or not.

The **PR interval** can provide clues to both the location of the originating impulse and the integrity of the conduction pathways of the heart. A PR interval longer than normal (greater then 0.2 seconds) suggests that conduction is abnormally slow through the AV junction. This phenomenon is called **first degree AV block**. A PR interval shorter than normal (less than 0.12 seconds) occurs commonly with **junctional rhythms** (the impulse begins somewhere in the AV junction) because part of the conduction pathway - the atria and part of the AV junction - is bypassed and thus shortened.

The PR interval covers the time taken for the impulse to travel from the SA node through the atria and the AV junction through to the Purkinje network. Most of the PR interval is taken by the slow conducting AV junction. **Changes to the PR interval often points to the AV junction** (see box above). A normal PR interval is 0.12-0.20 seconds, which is the equivalent to 3-5 small squares (3-5 mm) on ECG paper.

If an ECG shows P wave, QRS complex - P wave, QRS complex - P wave, QRS complex - atrial depolarization, ventricular depolarization until the cows come home, a rather important relationship between the atria and the ventricle is revealed. **If the P wave is consistently followed by a QRS complex across a consistent PR interval, this is strong evidence that the originating impulse is supraventricular.** A consistent PR interval is often sufficient to declare that this is a supraventricular rhythm.

The QRS Complex

ECG interpretation relies heavily on the QRS complex. The QRS complex represents the depolarization of the ventricles. The repolarization of the atria is also buried in the QRS complex.

The normal depolarization of the ventricles is illustrated in Figure 4.13 on the next page. Three distinct waveforms are often present in a normal QRS complex. These waveforms follow the pathways of ventricular depolarization. Depolarization of the ventricular septum proceeds first from left to right away from the positive electrode in lead II. This early depolarization causes a small downward deflection called a Q wave.

A Q wave is the first negative deflection of the QRS complex that is not preceded by a R wave. A normal Q wave is narrow and small in amplitude (see Table 4.3 on page 80). Note that a wide and/or deep Q wave may signify a previous myocardial infarction (MI). More on the signs of cardiac ischemia and infarction is addressed in the next section.

Figure 4.13 The QRS Complex, ST Segment and the T Wave

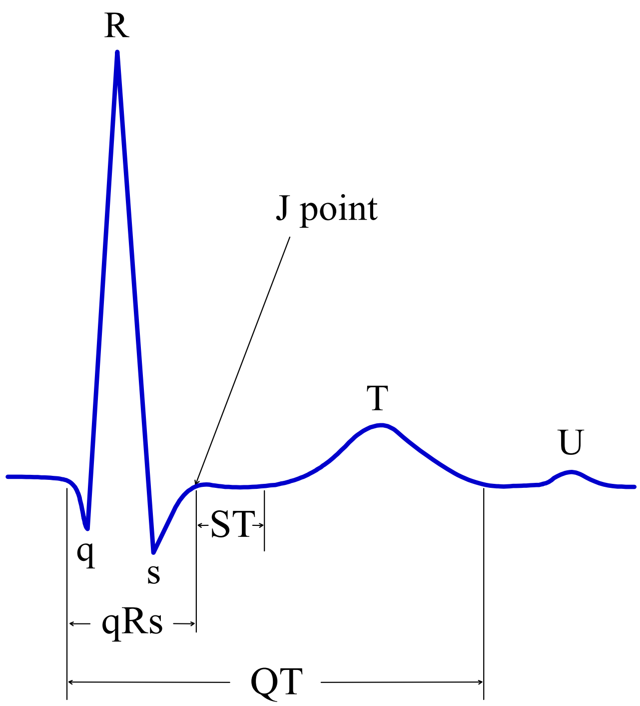


*Figure 4.13 depicts the component parts of the QRS complex. The QRS complex consists of a series of waves, the 'Q', 'R', and 'S' waves. The **'Q' wave** is the first negative deflection from baseline. The **'R' wave** is the first positive deflection above baseline. The **'S' wave** follows the 'R' wave with a negative deflection. A QRS complex may or may not have all three waveforms. The ST segment begins at the J point and continues to the beginning of the T wave.*

Following the depolarization of the interventricular septum, ventricular depolarization then progresses from the endocardium through to the epicardium across both ventricles producing an R wave and an S wave. An **R wave** is the first positive deflection of the QRS complex. An **S wave** is the first wave after the R wave that dips below the baseline (isoelectric line). The end of the S wave occurs where the S wave begins to flatten out. This is called the **J point**.

Why is the QRS complex so much larger than the P wave? The ventricles are about 3 times the size of the atria. The larger ventricle will produce a larger waveform.

The waveforms of the QRS complex are often labelled with lower and upper case letters. Large waves are labelled with upper case letters. Smaller waves that are less than half the amplitude of the large waveforms are labelled with lower case letters. The QRS complex #2 in Figure 4.14 would be correctly labelled an rS complex. Nevertheless, it is quite common to keep things simple, calling it a QRS complex regardless of the size and labels of its various waveforms.

Abnormal ventricular depolarization produces a QRS complex that often has additional waveforms. For example, a second positive deflection of a QRS complex *after an R wave* is labelled **R'** (R prime). Similarly, a second S wave that dips below the baseline after the R wave is labelled **S'** (S prime). Refer to Figure 4. 14 for an illustrated example. A downward **notch** in the R wave that does not dip below the baseline is simply called - yes - a notch in the R wave.

A narrow QRS complex occurs quickly over a period of less than 0.11 seconds (less than 3 mm in width). A narrow QRS occurs with normal ventricular depolarization that originates above the ventricles. Figure 4.14 provides a variety of different QRS complexes, produced by normal and abnormal ventricular depolarization. A normal, narrow QRS complex may be predominantly upright, predominantly inverted, completely inverted (called a QS complex) or biphasic (part upright, part inverted).

Figure 4.14 Various QRS Complex Morphologies

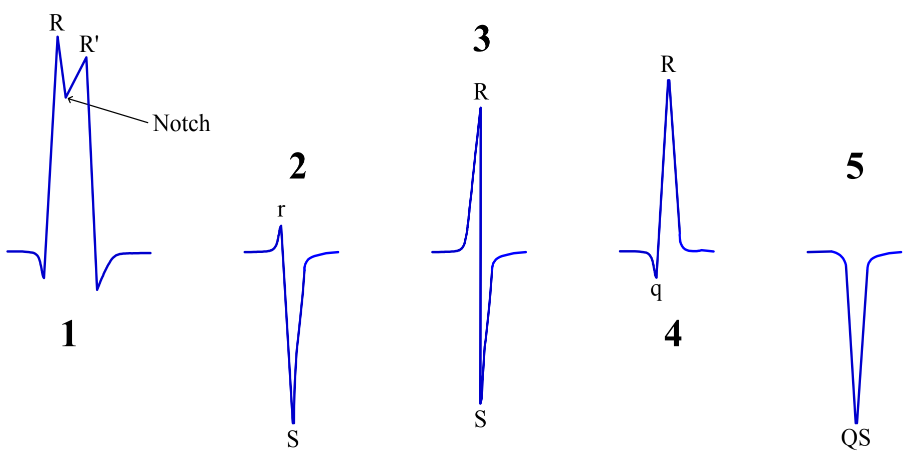


Figure 4.14 depicts several QRS complex shapes or morphologies. QRS complex #1 demonstrates the labelling convention for subsequent positive deflections above the baseline after the R wave. This second deflection is labelled R'. Note that a third upright deflection would be labelled R" (R double prime). QRS complex #2-4 are all normal QRS complexes of different shapes. QRS complex #3 is a biphasic QRS complex would be labelled RS. The QRS complex #5 is a QS complex.

While the direction of the QRS complex is generally not important with basic ECG interpretation, the width of the QRS complex is key. As mentioned in Chapter 3, **the width of the QRS complex often indicates the location of the originating electrical impulse**. This is a rather important point since the first and foremost word of an ECG interpretation is the location of impulse initiation.

For example, rhythms that come from the SA node are *sinus* rhythms, from the AV junction are *junctional* rhythms, and that originate from the ventricle are *ventricular* rhythms. Simple. If the QRS is narrow - taking very little time to occur - the cardiac rhythm originates from a supraventricular site. Quickly determining whether the QRS is narrow or wide is a vital step in rapid ECG interpretation.

The Q Wave and The QT Interval

As mentioned in the previous section, a normal Q wave represents a depolarization of the ventricular septum, which usually travels from left to right, towards the right ventricle. When present, a Q wave is the first downward deflection of the QRS complex. While ST segment deviation is a sign of present events, a prominent Q wave points to an MI that has already occurred, recently to some time ago. A prominent Q wave is like a tattoo - once you have one, it's pretty much yours for good.

Figure 4.15 The Normal Q Wave and QT Interval

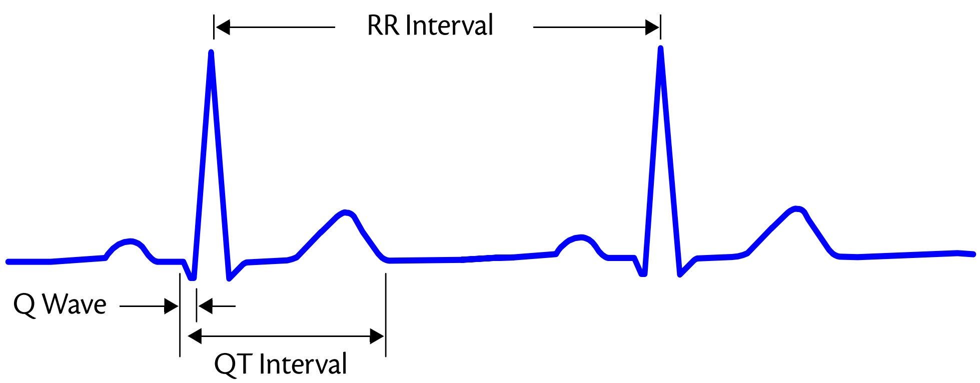
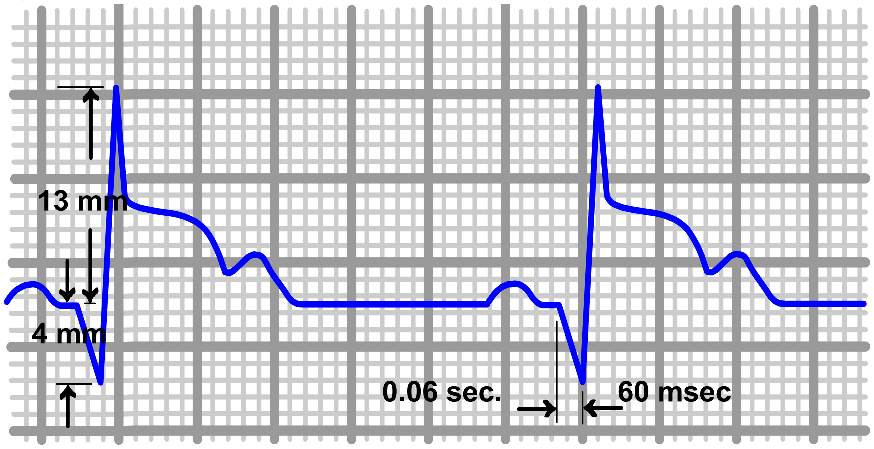


Figure 4.15 illustrates the Q wave, QT interval and the R-R interval. A normal QT interval should be less than half the R-R interval. A prolonged QT interval is associated with increased risk of R-on-T triggered lethal dysrhythmias.

A normal **Q wave** is usually no deeper than 2 mm and less than 1 small square in width (<0.04 seconds). An abnormal Q wave tends to get the most attention. A Q wave that is wider than 1 small square or at least 1/4 the height of the R wave is a significant marker of a myocardial infarction. In Figure 4.16, the Q wave is about 31% the height of the R wave (4/13 = 31%), making the Q wave prominent. The width of the Q wave is also significant with a width of 0.06 seconds. This Q wave is typical of an MI.

Figure 4.16 Prominent Q Waves



The Q waves of Figure 4.16 are abnormal in both depth and width, findings that point to a previous myocardial infarction.

The QT interval represents a complete ventricular cycle of depolarization and repolarization. The QT interval is measured from the beginning of the QRS complex to the end of the T wave. A QT interval should be less than 1/2 the R-R interval.

A **long QT interval** wider than 1/2 the R-R interval is a significant risk factor for developing hemodynamically unstable dysrhythmias such as ventricular tachycardia and torsades de pointes. A prolonged QT interval is also associated with a higher incidence of sudden death.

The concern around a longer QT interval centers around the possibility of the next QRS coming at the tail end of the T wave, called an R-on-T phenomenon. This phenomenon can potentially cause dangerous dysrhythmias such as torsades de pointes. Causes of prolonged QT intervals include long QT syndrome, antiarrythmics such as quinidine and procainamide, tricyclic antidepressants, and hypokalemia.

The ST Segment

Between the QRS complex and the T wave, lies the ST segment. The ST segment usually follows the isoelectric line. The ST segment represents early repolarization of the ventricles. Early repolarization includes a plateau phase where the cardiac cell membrane potential does not change.

During early repolarization, the positive ion potassium exits the cardiac cell while the positive ion calcium enters the cardiac cell, effectively negating any change in cell membrane potential. Because the cell membrane does not change its electrical potential, ECG leads do not record any electrical activity. As a result, the ST segment usually lies along the ECG baseline.

Determining where the ST segment begins is determined by the J point. The J point, the juncture of the QRS and the ST segment, defines the starting point of the ST segment. The J point marks where the QRS complex changes direction, forming a notch or bump in the ECG tracing. The ST segment is evaluated for any deviation from the ECG baseline 0.04 seconds (1 mm) after the J point.

While ST deviations may be a normal occurrence for a subset of the population, most often ST deviation is a sign of either myocardial ischemia, myocardial infarction and/or cardiac disease. **It makes sense, then, to report any finding of ST deviation from baseline in the ECG interpretation** i.e. sinus rhythm with ST depression.

Figure 4.17 ST Segment Deviations

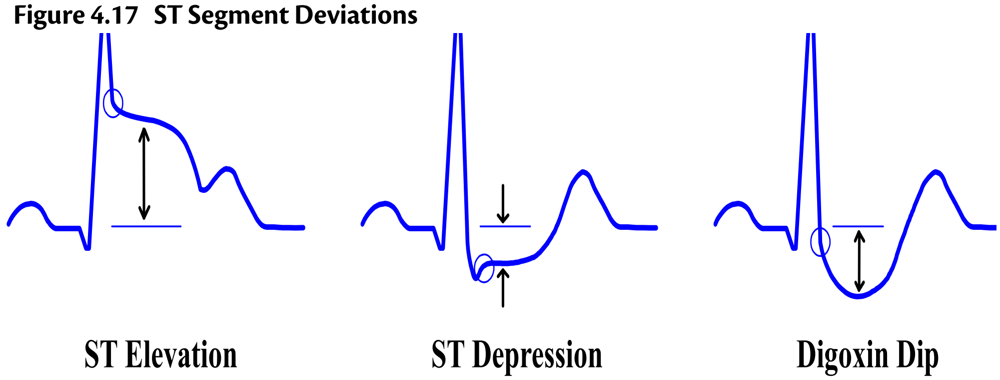
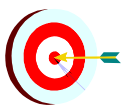


Figure 4.17 presents three examples of ST deviation. A digoxin dip is also called a "dig dip".

ST depression of 1 mm or more in **2 contiguous leads** (neighboring leads) is suggestive of myocardial ischemia, injury or infarction. **ST elevation** of 1 mm or more in 2 contiguous leads is highly suggestive of a myocardial injury or infarction. Note that ST changes (elevation or depression) are highly suggestive of current events - **the acute coronary events are happening now.**

The shape of the ST segment, if depressed, bears mention. The depressed ST segment often presents horizontal (see the center QRS complex of Figure 4.17), sloping downwards or sloping upwards. Although all morphologies can indicate myocardial ischemia, the horizontal and downward sloping depressed ST segments are the more likely morphologies that point to ischemic events.

Note that ST changes can occur from conditions other than myocardial ischemia. As mentioned, ST elevation and depression may be a benign finding, although uncommon. For example, ST depression that is concave in shape - called a **dig dip** - can occur for patients taking digitalis (see Figure 4.17 on the previous page) even at normal blood levels. A depressed and upward sloping ST segment can represent **ventricular hypertrophy**.



ST segment elevation is a common finding in young healthy adults of African descent. This phenomenon is attributable to normal **early repolarization** and not a result of cardiac disease. Using an ECG to diagnose a disease state in the absence of direct contact with the patient, a patient's clinical history and presentation is fraught with peril.

The presence of ST elevation in most views of a 12 lead ECG suggests **pericarditis**. Ventricular rhythms and supraventricular rhythms with **left bundle branch block** have wide and bizarre QRS complexes, making the detection of ST changes all but impossible.

Several conditions not linked to cardiac ischemia can produce ST changes. The bottom line: most ST changes indicate cardiac ischemia, requiring urgent treatment BUT every ECG interpretation is more robust when integrated with a patient's clinical status and history.

The T Wave

Expect a T wave to follow every QRS complex. The T wave is a graphic representation of the repolarization of the ventricle. The T wave is typically about 0.10 to 0.25 seconds wide with an amplitude less than 5 mm. While ventricular depolarization occurs rapidly producing a tall QRS complex, ventricular repolarization is spread over a longer interval, resulting in a shorter and broader T wave.

The T wave is normally slightly asymmetrical and is usually larger than the P wave (see Figure 4.13 on page 83). The T wave is normally upright in lead II. Note that as heart rates increase, the P wave (atria) and the T wave (ventricles) begin to share the same space on an ECG. The larger T wave often covers the P wave. Note that the T wave is rarely notched. A notched T wave may also contain a P wave trying to show itself.

If ventricular repolarization returns cell membrane voltage back to its pre-depolarization resting electrical voltage, then **shouldn't the wave produced by ventricular repolarization be opposite that of ventricular depolarization?** In other words, should the QRS complex and the T wave face opposite directions, upright and inverted. This is usually not the case.

Ventricular depolarization proceeds from the endocardium to the epicardium, essentially depolarizing the ventricles from the inside out. It follows that repolarization also occurs from the inside out, producing inverted T waves opposite in direction to the QRS complex. Instead, repolarization is delayed in endocardial cells, allowing the epicardium to repolarize first. **Repolarization normally proceeds opposite to depolarization, from the outside in. An upright T wave results.**

An inverted T wave can point to cardiac ischemia among other causes. Ischemia to the epicardium prolongs ventricular repolarization to this area. This extended repolarization of the epicardium removes the delay between the repolarization of the endocardium and the repolarization of the epicardium, with *re*polarization now following the sequence of *de*polarization. An inverted T wave results.

Abnormally shaped T waves can signify acute episodes of cardiac ischemia, electrolyte imbalances, and the influence of cardiac medications. For example, peaked T waves can occur early during periods of myocardial ischemia and infarction. Later, cardiac ischemia may cause the T wave to invert. Electrolyte imbalances can also affect the T wave. Hyperkalemia is often associated with peaked T waves. Hypokalemia can flatten the T wave. Quinidine can widen the T wave while digitalis can flatten the T wave.

Abnormally shaped T waves can also occur following injury to the lungs or the brain. While the physiology is not well understood, T wave inversion can occur with a left-sided tension pneumothorax. Peaked or inverted T waves have also been reported with brain injury, specifically subarachnoid hemorrhage.

Figure 4.18 Normal and Abnormal T Waves

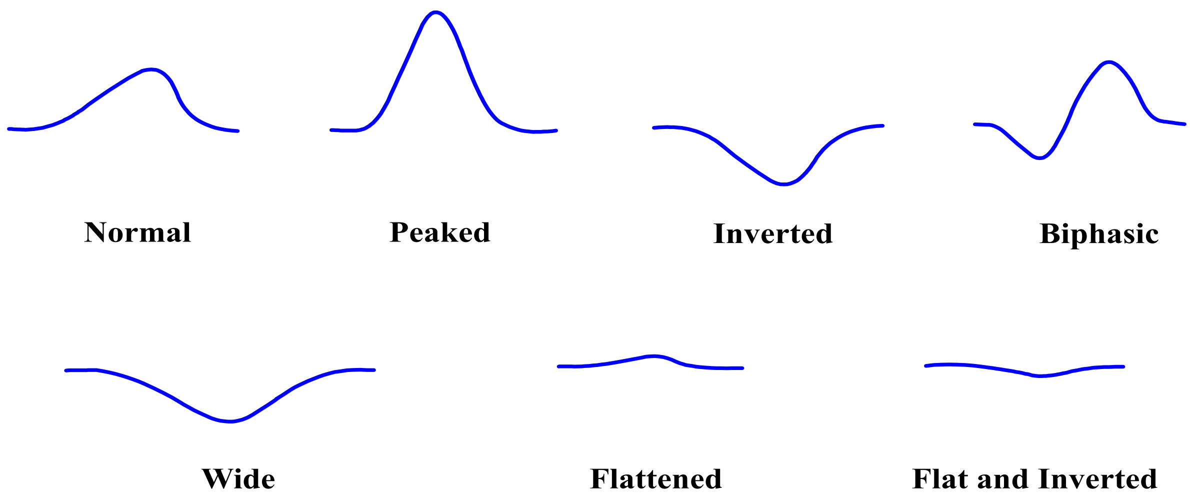


Figure 4.18 illustrates a variety of T waves, both normal and abnormal. A normal T wave is upright and slightly asymmetrical. During ischemic episodes, T waves may initially peak, then invert. Electrolyte imbalances such as hyperkalemia can cause the T wave to peak while hypokalemia is associated with flattened T waves. Certain medications such as quinidine can slow repolarization and widen the T wave while digoxin can flatten the T wave.

Abnormally shaped T waves are also commonly benign, muddying the clinical picture for practitioners. All morphologies of T waves, from normal to peaked to inverted are not uncommonly present in healthy individuals without any evidence of disease, cardiac or otherwise. This makes the T wave a weak sign for any diagnosis. The T wave must be placed along side other clinical evidence. Rarely would treatment be based solely on the shape of the T wave.

The U Wave

Occasionally, another wave -the **U wave** - is recorded immediately following the T wave and before the P wave. The U wave remains rather mysterious but is thought to represent a final stage of repolarization of unique ventricular cells in the midmyocardium. The U wave will most often orient in the same direction as the T wave with an amplitude less than 2 mm.

An abnormal U wave is inverted or tall with an amplitude of 2 mm or more. An abnormally tall U wave is associated with conditions such as hypokalemia, diabetes, ventricular hypertrophy, and cardiomyopathy. Cardiac medications such as digoxin and quinidine can also cause a tall U wave.

A series of waves, intervals and segments form the ECG. Knowing what to expect from each the these components prepares you to quickly recognize deviations from the norm. Before looking to the QRS complex and the R-R interval to determine heart rate, let's take a moment to differentiate between heart rate and pulse rate.

Heart Rate and Pulse Rate

Heart rate is the number of QRS complexes - the number of ventricular depolarizations - present in a minute. **Heart rate is not always the same as pulse.** Heart rate is a measurement of electrical activity while pulse ensures the perfusion of the blood to the target tissues.

Many have been caught depending on the cardiac monitor for vital information such as heart rate. For example, patients with electronic pacemakers may display heart rates twice the pulse rate. This occurs because the QRS complex produced after the pacer spike also may count as a separate QRS complex, thus doubling the heart rate.

A special case of the disparity between heart rate and pulse can occur with ventricular bigeminy. **Ventricular bigeminy** is a cardiac rhythm with PVCs every alternate QRS complex. If the PVCs do not have a corresponding pulse, the pulse rate would be equal to 1/2 the heart rate displayed on most cardiac monitors. A seemingly adequate heart rate of 70/minute may have a pulse of only 35/minute!

Another example of a serious disparity between heart rate and pulse occurs with premature ventricular complexes (called PVCs). The PVCs come early and cause short filling times. It follows then that the ventricles stretch minimally and subsequently contract ineffectively. The outcome: many PVCs fail to produce a perfusing pulse and the peripheral pulse is less than the heart rate displayed by the cardiac monitor (which includes both normal QRS complexes and PVC complexes).

The patient - not the monitor - is the gold standard.

This cannot be said enough. Most of us have been caught at one time or another relying on the cardiac monitor to the exclusion and great risk of the patient.

Heart rate is virtually always provided on the screen of a cardiac monitor. This number may offer some value but taking a patient's pulse is always good practice. Don't get burned.

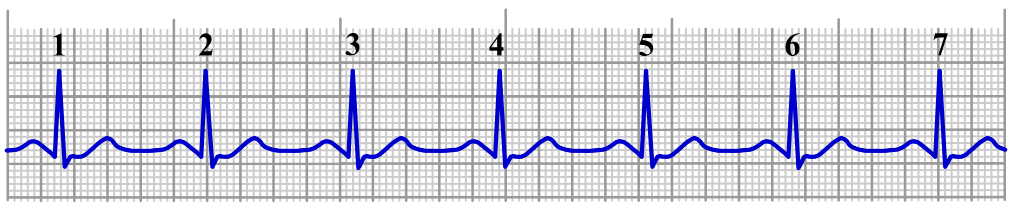
Calculating Heart Rate

Heart rate is a function of time. To measure heart rate from an ECG, a rhythm strip of ECG is required. As mentioned earlier, ECG paper provides a scale to measure time (25 mm = 1 second) including hash marks at either the top or bottom of the graph paper to indicate 1 second and/or 3 second intervals. Three methods for calculating heart rate are commonly used: the six second count, the triplicate method and the caliper method.

The Six Second Count

Perhaps the simplest and most common method to determine heart rates involves multiplying the number of QRS complexes found over six seconds by a factor of 10 to get the number of QRS complexes in a minute (60 seconds). Figure 4.19 provides an ECG over a six second period. What is the heart rate per minute for this rhythm strip?

Figure 4.19 The 6 Second Count Method to Determine Heart Rate.



7 QRS complexes in 6 seconds x 10 = 70 QRS complexes/minute

Simple stuff. This method - call it **the six second count** - works well whether the rhythm is regular or irregular. Mind you, if the rhythm is grossly irregular, a longer rhythm strip may produce a more accurate heart rate i.e. 15 seconds of QRS complexes multiplied by 4. Note that the six second count also works well for slow rhythms.

The Triplicate Method

The **triplicate method** is useful for measuring heart rate over shorter periods (less than 3 seconds) or for calculating heart rates of rapid tachycardias. This method is quick but not quite as accurate at measuring heart rate as the six second count method. **Note: the rhythm must be regular (consistent R-R interval) for the triplicate method to be of any use.**

Understanding the triplicate method calls on the fact that a large square on ECG paper equals 5 mm or 0.20 seconds (5 mm x 0.04 = 0.20 seconds or 1/5 of a second). Begin by finding an R wave that falls on a thicker vertical line in Figure 4.19 of the previous page (the second QRS complex). If the next R wave and every subsequent R wave occurred only one large square apart, how fast is the resulting heart rate?

1 QRS every 1/5 of a second = 5 QRS/second = 300 QRS/minute

Fast. If the R waves consistently arrived two large squares apart, the heart rate would be half of 300 at 150/minute. If the R waves are three large squares apart, the heart rate would be a third of 300/minute at 100/minute. Four large squares in between R waves equals a heart rate of 75/minute. A rate of 60/minute occurs with R-R intervals of five large squares.

Figure 4.20 Triplicate Method to Determine Heart Rate

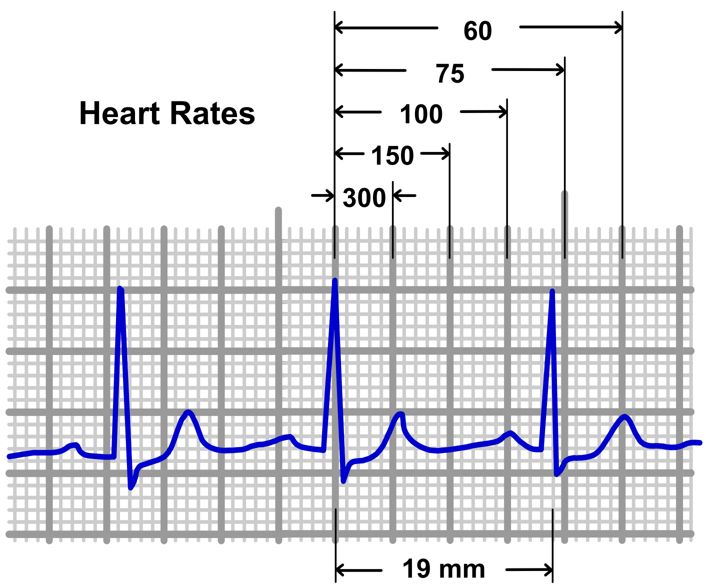


Figure 4.20 illustrates the triplicate method to estimate heart rate with only two QRS complexes. The triplicate method is particularly useful when measuring the heart rate of an underlying rhythm when frequent ectopic beats are also present. This method can be used only for rhythms with a consistent R-R interval. For rhythms with irregular patterns, the six second count is preferable for measuring heart rate.

In figure 4.20, the second QRS complex falls on a thick vertical line. The next R wave is between three large squares (thick lines) and four large squares away. The heart rate is estimated between 75/minute and 100/minute. Because the next R wave falls closer to the fourth thick line, the rate is closer to 75/minute. The heart rate is about 80/minute, a safe approximation from a treatment perspective. Would a heart rate of 77/minute or 85/minute be any more meaningful? Not likely.

The Caliper Method

If you did want an exact rate, you could count the number of millimeters across the R-R interval. This is often accomplished with the use of calipers. ECG paper records at a rate of 25mm/second which would total 1500 mm if allowed to print for a full minute (60 seconds x 25mm/second = 1500mm). Divide the total of 1500 by the R-R interval (in millimeters) to arrive at the number of QRS complexes per minute.

Revisiting Figure 4.20, the R-R interval is measured to be 19 mm.

1500 / 19 = 79 QRS complexes per minute

Heart Rate = 79/minute

An earlier approximation using the triplicate method of 80/minute wasn't far off the mark. Note that for regular rhythms (R-R interval is consistently the same), all three methods are effective in determining rate. **For irregular rhythms with fluctuating R-R intervals, the six second count is the only useful method for measuring heart rates.**

Summary

This chapter served as an ECG primer, outlining the cardiac monitoring system, ECG components and three methods to determine heart rate. The electrocardiogram is a graphical representation of cardiac electrical activity measured over time. The amplitude or height of waveforms reflects differences in voltage across the heart while the width of the ECG reflects intervals of time. Specialized paper records an ECG at a speed of 25 mm/second.

An ECG is recorded and displayed with a cardiac monitoring system. Ongoing monitoring is provided via 3 lead, 5 lead and modified 12 lead systems. The basic 3 lead ECG remains a common monitoring configuration, placing electrodes near the right shoulder (white), the left shoulder (black) and the lower left lateral area just below the ribs (red). There is a variety of 5 electrode systems that provide five lead views to as many as 12 lead views with the EASI™ or interpolated 12 lead systems.

The heart is viewed from the perspective of the electrode with the positive polarity, viewing the heart toward its negative counterpart. For example, lead II with the positive red electrode, looks up at the apex of the heart towards the white electrode (negative).

The positive electrode is also important in determining the direction of waveforms. Electrical activity that depolarizes towards the positive electrode produces an upward deflection.

The waveform produced by atrial depolarization is the P wave. The appearance of a P wave before a QRS complex strongly suggests atrial kick. Ventricular depolarization results in a QRS complex. A narrow QRS (rapid depolarization) occurs when rhythms are supraventricular in origin. The PR interval is the time between the beginning of atrial and ventricular depolarization. The T wave is the repolarization of the ventricles.

The ST segment and Q wave are also significant. Deflections of the ST segment more than 1 mm above or below the baseline in 2 contiguous leads is a diagnostic sign of cardiac ischemia and/or myocardial injury. While ST elevation is most commonly a sign of myocardial infarction (MI), ST depression can signify cardiac ischemia or myocardial infarction. The presence of a Q wave that is 1 mm wide and/or at least 25% the height of the QRS complex points to a completed MI.

The QT interval, the full ventricular cycle of depolarization and repolarization, should be less than half the R-R interval. Longer QT intervals are associated with R-on-T phenomenon yielding ominous rhythms such as ventricular tachycardia, torsades de pointes and ventricular fibrillation.

Heart rates and pulse rates can be quite distinct. The gold standard remains the pulse rate measured on the patient. Methods to determine heart rate include the six second count, the triplicate method and the caliper method. The preferred method often depends on the regularity and speed of the cardiac rhythm.

Chapter Quiz

1. Lead II, an inferior lead, views the apex of the heart.

True or False

2. Atria depolarization towards a positive electrode in lead II results in an upward P wave.

True or False

3. The rate by which the ECG normally prints on paper is 50 mm/second.

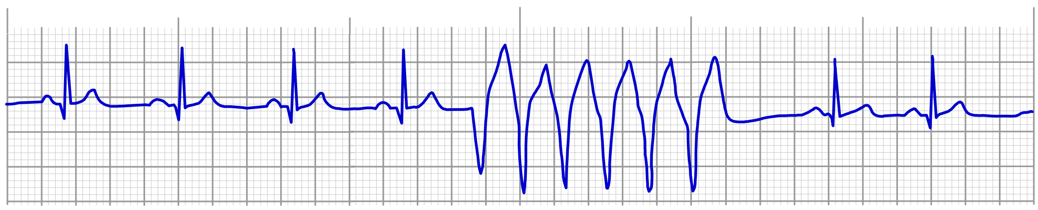
True or False

Answers: ***1.*** *True;* ***2.*** *True;* ***3.*** *False*

4. The QRS complex represents both ventricular depolarization and atrial repolarization.

True or False

Questions #5-8 refers to the six second ECG below.



5. The sinus rhythm for this ECG has a heart rate of _____/minute.

6. Would a patient with this ECG have an atrial kick during the burst of rapid ventricular beats?

Yes or No

7. The QT interval for the underlying sinus rhythm is normal.

True or False

8. The width of the QRS complex for the sinus rhythm is _______ seconds.

9. The coloring schemes for ECG lead wires are standardized by (circle all that apply):

a) American Heart Association (AHA)
b) American Medical Association (AMA)
c) International Electrotechnical Commission (IEC)
d) Manufacturers of cardiac monitors

10. Lead I views which area of the heart?

a) apex of the heart
b) lateral left ventricle
c) right ventricle
d) posterior right ventricle

11. The presence of ST depression in lead II and lead III points to cardiac ischemia or infarction to the (left lateral, inferior, septal) region of the heart.

Answers: ***4.*** *True;* ***5.*** *approximately 90/minute;* ***6.*** *No;* ***7.*** *True;* ***8.*** *0.06 seconds;* ***9.*** *a),c);* ***10.*** *b);* ***11.*** *inferior*

12. An ECG lead views the heart from the perspective of the positive electrode viewed towards the negative electrode.

True or False

13. Which of the following statements is **false** regarding dysrhythmia monitoring?

a) the more ECG lead views, the better
b) a 12 lead ECG can differentiate between ventricular tachycardia (VT) and supraventricular tachycardia (SVT) 90% of the time
c) the use of lead MCL_1 is associated with 22% more errors in differentiating VT and SVT than using lead V_1
d) lead II is the best lead view for dysrhythmia monitoring

14. A single lead view is often sufficient for dysrhythmia monitoring but seldom sufficient for cardiac ischemia monitoring.

True or False

15. According to the American Heart Association, the color configuration of the 5 lead ECG system is:

Left Arm (LA) ____________
Right Arm (RA) ____________
Left Leg (LL) ____________
Right Leg (RL) ____________
Lead V_1 or V_5 ____________

16. Lead II from a three lead system always appears identical to lead II of a 12 lead ECG.

True or False

17. Lead fingerprinting refers to:

a) based on a patient's history, choosing the ECG lead that monitors an area of the heart most likely to have ischemic episodes
b) the appearance of Q waves in ECG leads that view regions of the heart that have infarcted
c) the presence of two contiguous leads that display ST elevation, diagnostic of a myocardial infarction
d) smudging the ECG strip with your fingers

Answers: ***12.*** *True;* ***13.*** *d);* ***14.*** *True;* ***15.*** *LA-black, RA-white, LL-red, RL-green,* V_1 *or* V_5*-brown;* ***16.*** *False;* ***17.*** *a)*

18. The interpolated 12 lead ECG includes four (limb, precordial) electrodes and two (limb, precordial) electrodes.

19. A normal small Q wave is the result of:

a) atrial depolarization
b) interventricular depolarization
c) posterior ventricular depolarization
d) depolarization of septal region

20. A wave of depolarization that travels towards a lead's positive electrode, produces an upright waveform on an ECG.

True or False

21. Of the ECG components, an interval can include (circle all that apply):

a) wave(s)
b) segment(s)
c) voltage changes in the heart
d) all of the above
e) none of the above

22. Ventricular contraction and ventricular depolarization occur simultaneously.

True or False

23. The PR interval measures the period for the electrical impulse to travel:

a) from the SA node to the bundle of His
b) through the atria
c) from the SA node to the Purkinje network
d) across the AV junction

24. A consistent PR interval across a six second ECG strongly supports the claim that:

a) the rhythm originates at the AV junction
b) the rhythm originates above the ventricles
c) the rhythm originates from the SA node
d) ventricular depolarization progresses along a normal pathway

25. Ventricular contraction typically occurs at the same moment as the (QRS complex, ST segment, T wave).

Answers: ***18.*** *Limb, Precordial;* ***19.*** *d);* ***20.*** *True;* ***21.*** *d);* ***22.*** *)False;* ***23.*** *c);* ***24.*** *b);* ***25.*** *ST segment*

26. Characteristics of an ECG that suggest a myocardial infarction include (circle all that apply):

a) ST segment elevation in two contiguous leads
b) ST depression in two contiguous leads
c) inverted T waves
d) deep or wide Q waves
e) tall P waves

27. A normal QRS complex can be (circle all that apply):

a) upright
b) inverted
c) biphasic
d) wide

28. A QT interval longer than 1/2 the R-R interval increases the risk for which events to occur (circle all that apply):

a) R-on-T phenomenon
b) torsades de pointes
c) ventricular tachycardia
d) ventricular fibrillation

29. ST deviation is always a sign of cardiac ischemia.

True or False

30. Peaked T waves can signify (circle all that apply):

a) normal repolarization of the ventricles
b) cardiac ischemia
c) hyperkalemia
d) subarachnoid hemorrhage
e) left-sided tension pneumothorax
f) myocardial infarction
g) atrial hypertrophy

Answers: ***26.*** *all but e);* ***27.*** *all but d);* ***28.*** *all;* ***29.*** *False;* ***30.*** *all but g)*

Suggested Readings and Resources

Adams-Hamoda, M. et al. (2003). Factors to Consider When Analyzing 12-Lead Electrocardiograms for Evidence of Acute Myocardial Ischemia. American Journal of Critical Care (12) 1. Also found at:
http://www.aacn.org/pdfLibra.NSF/Files/Adamsfinal/$file/Adams.pdf

Cowley, M. (2002). A Practical Guide to ECG Monitoring and Recording. Found at http://www.mikecowley.co.uk/leads.htm.

Drew, Barbara J.(2002). Celebrating the 100th Birthday of the Electrocardiogram: Lessons Learned from Research in Cardiac Monitoring. American Journal of Critical Care (11) 4. Also found at
http://www.aacn.org/pdfLibra.NSF/Files/Drewjul02/$file/Drewfinal.pdf

Macfarlane, P.W. and Coleman, E. N. Resting 12-Lead ECG Electrode Placement and Associated Problems. Found at http://www.scst.org.uk/coleman/resting.htm.

Philips Medical Systems. (2002). EASI 12-Lead Algorithm. Found at:
http://www.medical.philips.com/main/products/resuscitation/assets/docs/algorithms/easi_12lead_technical_overview.pdf

Smith, Stephen W. (2002). The ECG in Acute MI. New York: Lippincott Williams and Wilkins

What's Next?

The ECG primer completed the core basics necessary to make sense of ECGs. It is time to put it all together. Chapter 5 weaves together the concepts and major points presented thus far into a simple four step method to rapidly interpret ECGs. This brief chapter does not delve into new material but rather quickly makes use of the four step method with ample opportunity for practice. Now that the ECG is seen for what it truly represents, ECG interpretation can be simple, fast and meaningful.

5

In Four Simple Steps

Quick Look

It is time to take stock of the concepts covered thus far. Chapter 1, *The Structures of the Heart*, provided a brief outline of cardiac anatomy. Chapter 2, *It's All About Cardiac Output*, addressed the parameters that affect cardiac output, including the consequences of heart rates that are either too fast or too slow.

In Chapter 3, *The Electrics*, the most important concept may be the special role the bundle branches have in facilitating rapid ventricular depolarization. This translated to, "A narrow QRS complex occurs only with supraventricular rhythms."

Chapter 4, *An ECG Primer*, covered the cardiac monitoring system, the components of an ECG and various methods to measure heart rates. Vector theory placed the P waves in a key position to differentiate between sinus, atrial and junctional rhythms.

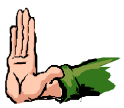
This chapter weaves these concepts into a simple, practical four-step method to rapidly interpret ECGs. It is time to bring it all together in an approach to ECG interpretation that is rooted in clinical practice.

And yet relation appears, a small relation expanding like the shade of a cloud on sand, a shape on the side of the hill.

Wallace Stevens from "Connoisseur of Chaos"

Overview

Most of the work necessary to arrive at a 4-step method for ECG interpretation is already complete. This four step method uses a couple assumptions. First, the 4-step method is largely based on interpreting a single lead strip i.e. lead II. Second, this method is definitely a bare bones approach to ECG interpretation. The simplicity of the 4-step method facilitates rapid ECG interpretation and a quick response to ominous ECG findings.



If you find that reading ECGs continues to be difficult after you complete this chapter, take the opportunity to go back and review the main points of the first four chapters. A second review may be all that is required. Often a concept that may prove elusive earlier, is fully captured on a second pass. The keys to meaningful ECG interpretation are definitely within reach.

Let's begin by taking a quick look at the four basic steps.

1. **Is the rate too fast (>150/minute) or too slow (<50/minute)?** If so, check the patient for signs and symptoms of poor cardiac output.

2. **Is the QRS wide or narrow?** A wide QRS strongly suggests a ventricular rhythm. A narrow QRS indicates a supraventricular rhythm.

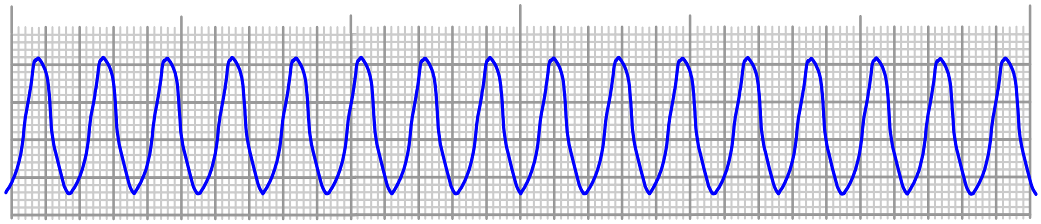
3. **Check the P waves.** The shape of the P wave points to the originating impulse site for supraventricular (SV) rhythms. Does the SV rhythm originate from the SA node, the atria or the AV junction?

4. **Is the R-R interval pattern regular or irregular?** An irregular pattern of the R-R intervals deserves closer inspection.

This 4-step method quickly identifies virtually every cardiac rhythm. Of course, some rhythms, particularly those with an irregular pattern, require a little more diligence. These will addressed separately in Chapter 6.

Before delving into each of these steps in more detail, perhaps taking a simple, brief look at an ECG, step-by-step, might demonstrate the merit of the 4-step method. A little success is a great motivator!

Figure 5.1 Sample Rhythm #1



1. Is the rate too fast or too slow? Using the triplicate method, the rhythm is clearly faster than 150/minute (an R-R interval less than 2 large squares). Using the six second count, the heart rate is 160/minute (16 QRS over six seconds x 10 = 160/minute). **Too fast!** Check the patient for signs and symptoms of poor cardiac output.

2. Is the QRS wide or narrow? The width of the QRS is greater than 3mm. The QRS is wide. **The rhythm most likely originates from the ventricles.**

3. Check the P waves. There are **no visible P waves** in lead II. If the patient was hemodynamically stable, taking a 12 lead ECG is warranted to look for P waves from several lead views (precordial lead V_1 is a good place to start). The absence of any notching of the QRS or T wave suggests an absence of any recognizable atrial activity.

Note that without any P waves, atrial kick is also probably absent. Compounded with the short filling time inherent to this rapid rate is a probable loss of atrial kick. Cardiac output is likely seriously compromised.

4. Is the pattern regular or irregular? It's a **regular rhythm**. Rhythm interpretation should be straightforward.

This is indeed a fast ventricular rhythm, called **ventricular tachycardia.** The rhythm originated in the ventricles with a rapid rate of 160/minute.

So how was that? Simple? A little choppy at first? If you found any of the steps difficult, please review the concepts in the first four chapters. Practice will most certainly also help. With the four step method, ECG interpretation can indeed be simple, fast and meaningful.

Rhythm Naming Conventions

A few naming conventions require mentioning before examining the 4-step method in detail. A **cardiac rhythm is usually named first by the location of the originating impulse**. For example, rhythms that begin in the SA node are called sinus rhythms.

Rhythms that originate from the AV junction are called junctional rhythms. As the sample rhythm in Figure 5.1 on the previous page indicates, ventricular rhythms originate in the ventricle.

Conventions for Naming Cardiac Rhythms
Usually, the first word of a rhythm is the location of the initiating impulse. This is followed by a descriptor of the rate - bradycardia, tachycardia or just label it a rhythm. Next, include any abnormal findings i.e. premature complexes or other abnormal ECG components. A rhythm might be called a **sinus** *tachycardia* with a *premature ventricular complex* (PVC). Sounds impressive, but the naming method is simple.

After first identifying the location of the generated impulse, **then classify it according to rate.** A rate faster than 100/minute (101/minute or faster) is a tachycardia. The term bradycardia applies to rhythms that are slower than the expected pacemaker rate range (see Table 5.1). Rates that are neither fast nor slow are often labelled rhythms i.e. sinus rhythm.

For example, a rhythm originating from the SA node with a heart rate less than 60/minute, is labelled a sinus *bradycardia*. A rhythm that originates from the SA node at a rate above 100/minute is called a sinus *tachycardia*. A rhythm that originates from the SA node at a rate of 60-100/minute is just called a sinus *rhythm*. Similarly, a rhythm originating from the AV junction with a rate of 46/minute is a junctional *rhythm* - not a junctional bradycardia - because a junctional rate is expected within this rate range.

Table 5.1 .Expected Rate of Impulse Formation for Select Pacemaker Sites

Pacemaker Site	Typical Rate
Sinus Node	60-100/minute
AV Junction	40-60/minute
Bundle of His	40-60/minute
Bundle Branches	30-40/minute
Purkinje Network	20-40/minute

Detailed in Chapter 3, The Electrics, various sites within the heart serve as pacemakers. While the sinus node is usually the dominant pacemaker, other pacemaker sites become active when faster pacemakers fail. Table 5.1 provides a range of expected rates of impulse formation for each of the pacemaker sites within the heart.

Lastly, **follow up with descriptors of any other noteworthy characteristics of the rhythm**. Begin with any abnormalities of the underlying main rhythm. Is there ST deviation? T wave inversion? Prominent Q waves? Complete labelling the rhythm by addressing any extra beats. Are there any premature beats?

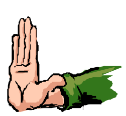
Take an ECG with a heart rate of 52/minute, narrow QRS complexes, inverted P waves, ST elevation and an extra premature beat that has a wide QRS complex. How would this rhythm be labelled? First, narrow QRS complexes with accompanying inverted P waves point to a supraventricular rhythm originating from the AV junction. The premature beat with a wide QRS complex suggests a premature ventricular complex (PVC). The rhythm is then labelled a *junctional rhythm with ST elevation and one PVC*.

When all is said, this rhythm sounds complicated and the person labelling the rhythm sounds quite brilliant. The steps, though, are simple.

The Four Step Method

Several methods for interpreting ECGs are available by such esteemed clinical educators as Aehlert, Connover, Dubin, Grauer, Huszar, Lewis, Marriott, Thaler and Walraven. Their books on ECG interpretation are referenced at the end of this chapter. Each presents a *unique* systematic approach to reading an ECG. Evidently, reading ECGs can be approached by various angles.

The Six Second ECG four step method focuses on simplicity, limited resources, a scarcity of time, and clinical relevance. Human resources are at a premium. Workload is increasing for most health care professionals. The available time available for any task, including ECG interpretation, is compressed. The four step method is typically completed in six seconds or less.



The great majority of cardiac rhythms can be identified with a single lead ECG, regardless of the ECG interpretation method chosen. There are instances where more than one name is possible for an ECG based on the clues provided by a single lead ECG. For these cases, multiple lead views may offer distinguishing characteristics not available with a single lead view, thus improving accuracy. The ability to differentiate between aberrantly conducted supraventricular tachycardia (supraventricular but with a wide QRS) and ventricular tachycardia is one example where multiple lead views can be beneficial.

While a thorough analysis of any ECG requires several lead views, a single lead ECG often suffices for the task of dysrhythmia interpretation. While many hospital units utilize multiple lead views to monitor a patient, single lead monitoring is also common. The four step method uses a single lead ECG to identify cardiac rhythms.

The Six Second ECG four step method is rooted in clinical practice, and successfully applied by health care professionals for more than a decade. The focus is rightly placed on the patient early on with the first step encouraging ongoing assessment for signs and symptoms of poor cardiac output for extreme heart rates (too fast or too slow). Perhaps at least as important, the four step method is reported to be practical, easily remembered and successfully used.

As a whole, the four step method is not superior to any other ECG methodology. It is simple and it works. Most likely, you will arrive at your own method for interpreting ECGs as your skills grow. You will likely include steps that tend to work for you and exclude those that don't. This is how the four step method evolved, with understanding and experience. Next is a detailed look at each of its four steps.

Step 1: Too Fast or Too Slow?

Quickly recognizing an excessively fast or slow heart rate is vital. As explained in Chapter 2, these extreme rates can lead to an inadequate cardiac output that requires urgent treatment. While rhythm interpretation is important, treatment for extreme unstable rates generally does not require knowledge of where the impulse originated.

Cardiac Output = Heart Rate x Stroke Volume

Heart rates over 150/minute are often associated with an inadequate filling time for older patients and for those with a significant cardiac history. As a result, stroke volumes tend to be poor. Even though the rate is high, when combined with a low stroke volume, the product (cardiac output) is low. With Figure 5.2 on the next page, notice how cardiac output can plummet as the heart rate increases above 150/minute.

Figure 5.2 Cardiac Output and Heart Rate

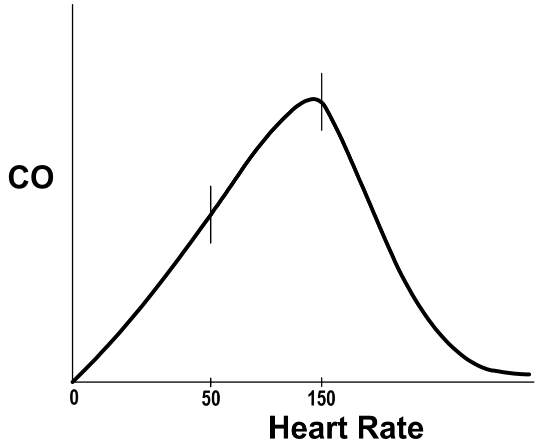


Figure 5.2 is a simplistic depiction of the relationship between heart rate and cardiac output (CO). Notice the low cardiac output values with heart rates above 150/minute or less than 50/minute.

Conversely, heart rates of less than 50/minute provide a lengthy filling time, thus maximizing stroke volume. Very slow rates, though, can compromise cardiac output. Even a good stroke volume of 80 ml when combined with a heart rate of only 30/minute yields a cardiac output of only 2400 ml, less than half an adult's average resting cardiac output of 5000 ml. The outcome for a patient with cardiac disease and a poor stroke volume is much more ominous (i.e. 40 ml x 40/min. =1600 ml).

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 poor cardiac output (shock).**

Low cardiac output is associated with signs and symptoms of shock: hypotension, shortness of breath, deteriorating level of consciousness, pale colour, cool extremities, and possibly cardiac ischemia. Signs and symptoms of shock require a rapid response that may include slowing rapid heart rates or speeding up heart rates that are too slow.

Of course, assessing for poor cardiac output *only* after heart rates are less than 50/minute or over 150/minute is overly rigid. Exceptions exist. If the patient has a history of cardiac illness i.e. coronary artery disease, myocardial infarction(s) or heart failure, expect lower stroke volumes that might require faster heart rates (60/minute or more) to produce an acceptable cardiac output.

For tachycardias and bradycardias that are the primary cause of poor cardiac output, treat the rhythm. These dysfunctional rhythms threaten the welfare of their host. Tachycardias less than 150/minute are rarely the cause of poor cardiac output. Rather, the increased rate is a compensatory response to a need for increased cardiac output i.e. hypovolemia, septicemia, pain, fever, exercise. For these slower tachycardias, treat the underlying cause. The rhythm is not the culprit.

Poor cardiac output can also occur with tachycardias at rates less than 150/minute. A diseased heart is often less compliant (more rigid), making it more dependent on sufficient preload and filling time to produce an effective stroke volume. As a result, fast rates of even 130/minute may be associated with inadequate filling times and poor cardiac output. **The more significant the cardiac history, the narrower the optimal heart rate range.**

Step 2: Is the QRS Wide or Narrow?

From a treatment perspective, steps #1 and #2 are necessary. Step #1 determines whether the patient is hemodynamically stable or not. Step #2 identifies the locus of the initiating impulse, important information for the medical management of rhythms. If the **QRS is narrow, the rhythm originates from a supraventricular site.** Otherwise, if the **QRS is wide, chances are, the rhythm is a ventricular rhythm.**

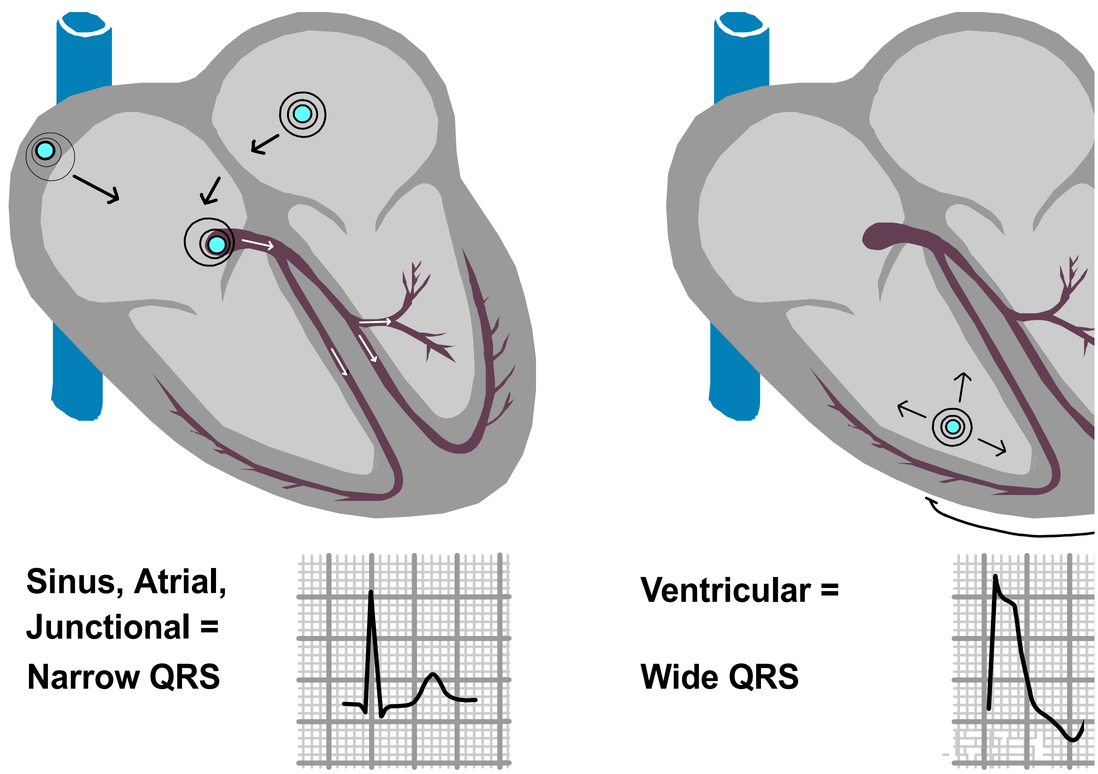
Remember that the ECG is a two dimensional plotting of voltage (height or amplitude) over time (width or length). The QRS complex is a representation of ventricular depolarization and to a much lesser extent atrial repolarization.

Please refer to Figure 5.3. With supraventricular rhythms, the wave depolarizing across the atria splits into at least three (as many as five) simultaneous waves depolarizing across the ventricles. This Autobahn (the bundle branches /Purkinje network) shortens both distance and the time taken for depolarization. A rapid depolarization results causing a narrow QRS of less than 0.12 seconds (less than 3 mm in width).

The depolarization of the ventricles are rapid when the impulse follows first the bundle branches and then the Purkinje network. The bundle branches take the one initiating supraventricular impulse and multiplies it into at least three simultaneous impulses. These impulses follow the right bundle branch (1 or 2 routes to the right ventricle) and the left bundle branch (2 or 3 routes called fascicles to the left ventricle). As a result, the one impulse wave across the atria becomes several waves across the ventricle. As a result, both the distance the waves need to travel and the time taken is dramatically decreased.

Supraventricular impulses ride the Autobahn (the bundle branches) causing rapid ventricular depolarization and a narrow QRS complex. But what of the wide QRS? What causes a QRS to be 3 mm or more in width? In other words, what slows down the depolarization of the ventricles?

Figure 5.3 Wide and Narrow QRS Complexes



Supraventricular rhythms with impulses that originate in the sinoatrial node (SA node), the atria or the AV junction in turn depolarize the ventricles via the multiple bundle branches that decrease both the distance and the time taken for each of the 3-4 depolarizing waves to travel. As a result, supraventricular rhythms are associated with rapid depolarization of the ventricles and a narrow QRS complex. Conversely, a wide QRS (more time taken to depolarize) often results from the one depolarizing wave initiated by a ventricular impulse that ***does not use*** *the bundle branches for rapid conduction.*

Ischemia and sympathetic stimulation can enhance a ventricle's automaticity, stimulating the ventricle to initiate an impulse before a sinus initiated wave reaches the ventricles. This solitary wave doesn't ride the Autobahn. Rather, this one wave must traverse both ventricles. The efficient autobahn is not utilized; instead, slower routes cross the ventricular myocardium. As a result, the distance and time taken to depolarize the ventricles are longer. A wide QRS of 3mm or more is produced.

Rhythms that are initiated in the ventricles have wide QRS complexes. A second characteristic of these ventricular rhythms is that the T wave is usually (though not always) facing the opposite polarity as the R wave.

A wide QRS complex is produced most often from an impulse that originates from within the ventricles. In fact, a wide QRS has a ventricular origin about 85% of the time in the general population. With those with known coronary artery disease, tachycardias with wide QRS complexes are indeed ventricular tachycardia almost 95% of the time. **As a general rule, if it looks like ventricular tachycardia, treat it like ventricular tachycardia.**

Again, exceptions exist. For about 15% of rhythms with wide QRS complexes, impaired bundle branch conductivity prolongs ventricular depolarization. Ischemia, infarction, and antiarrythmics can slow or block transmission of an impulse along these bundle branches. The resulting widening of the QRS complex is called **aberrant conduction**.

Slowed and aberrant ventricular conduction can also occur due to **Ashman phenomenon**. With premature beats, the His-Purkinje fibers may not completely repolarize before the next wave arrives. The ion channels may not yet be fully operational. The resulting depolarization is slower as is the conducting impulse resulting in aberrant conduction and a wider QRS. This phenomenon of an aberrantly conducted QRS occurring with premature or early beats is known as Ashman phenomenon.

Supraventricular rhythms with aberrant conduction can be recognized to be supraventricular in origin by the presence of a P waves before each QRS. In other words, a consistent PR interval implies a connection or relationship between the ventricles and supraventricular structures. **A consistent PR interval is a diagnostic criterion of a supraventricular rhythm.**

For faster rhythms, the T wave often overshadows the P wave making P wave detection difficult. Occasionally a P wave notches the T wave. A notched T wave strongly suggests the presence of a P wave.

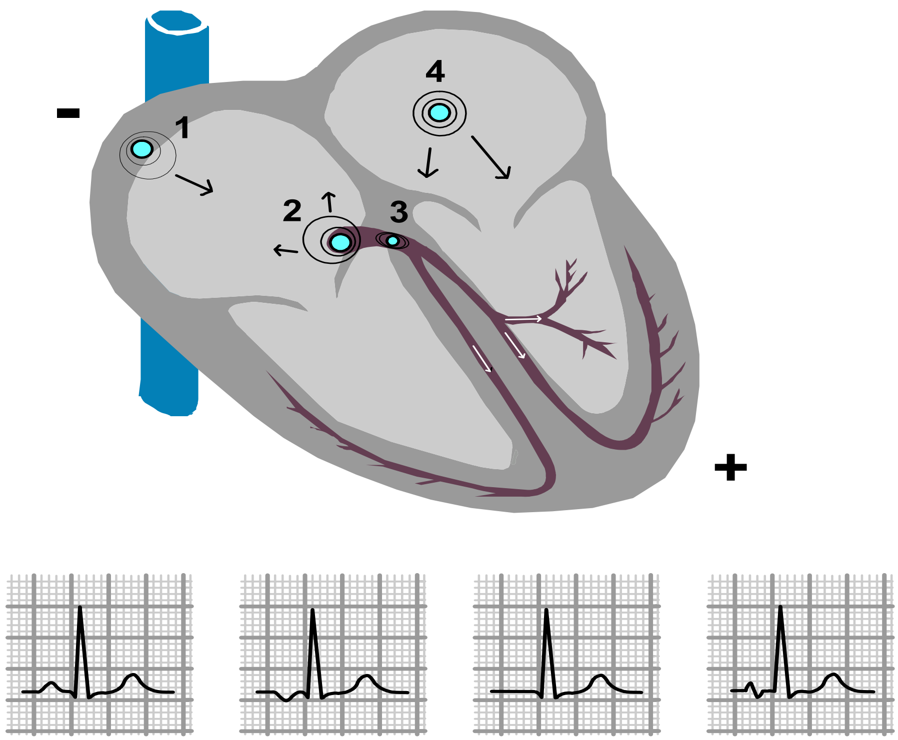
Bottom line: the impulse for a narrow QRS originates above the ventricles. A wide QRS requires a little more investigative work. A wide QRS is *almost always* caused by an impulse originating in the ventricles. The presence of a P wave before each wide QRS, though, strongly suggests a supraventricular rhythm with aberrant conduction.

Step 3: Check the P Waves

Whether the QRS is wide or narrow, looking for P waves is an important task. For a narrow QRS, the P waves and PR interval help us to specify whether the supraventricular rhythms is a sinus, atrial or junctional rhythm. We accomplish this based on vector theory outlined in Chapter 3. Electrical waves that move towards a positive electrode produce a positive deflection or wave. Inversely, if the electrical wave moves away from a positive lead a downward facing wave results

This vector principle is especially useful when inspecting P waves. Since the atria are depolarized with one wave, locating the initiating impulse can be quite simple. In Figure 5.4, for example, waves that initiate in the SA node (#1) travel across the atria in the direction of the positive red electrode in lead II producing an upright P wave.

Figure 5.4 The Shape of the P Wave in Lead II



In Figure 5.4, a sinus rhythm originates from the SA node (#1) with the resulting atrial depolarization being directed toward the positive red electrode typical of lead II. This results in an upward shaped P wave. When an impulse originates high in the AV junction (#2), atrial depolarization generally occurs in a direction away from the positive red electrode and an inverted P wave is seen. For rhythms that are initiated lower in the AV junction, often the atria and the ventricles depolarize simultaneously. While the QRS remains narrow (it is a supraventricular rhythm), the P wave is absent because it is buried in the much larger QRS complex. Impulses that originate in the atria produce a variety of non-symmetrical P waves, often with a biphasic shape (part of the wave up and the other directed downwards).

When the AV junction initiates an impulse - in premature junctional complexes or sustained junctional rhythms - the atrial depolarization is directed away from the positive red electrode producing an inverted wave. The location of the pacemaker within the junction will dictate whether the P wave will even be seen.

For example, if the firing locus is high in the junction, it is likely that the atria will depolarize before the ventricles and an inverted P wave will be present (see #2 in Figure 5.4). If the firing locus is further down in the junction, the ventricles and the atria may receive the impulse simultaneously, resulting in the large QRS burying the smaller P wave (see #3). As a rule, **if the QRS is narrow (it must be supraventricular) and the P waves are absent, a junctional rhythm is most probable.**

Atrial rhythms come with P waves that take shapes (also called morphologies) that appear to be a fusion of P waves typical of sinus and junctional rhythms. An atrial P wave is often notched, peaked or biphasic (see #4 of Figure 5.4 on the previous page). An atrial beat may come premature (a premature atrial complex or PAC) or may arrive in groupings that are sudden and often quite rapid called paroxysmal atrial tachycardia (PAT).

Besides P wave morphology, the presence of a P wave before each QRS complex is an important finding. The P wave represents the depolarization of the atria. A P wave that occurs a consistent period of time before the QRS complex implies a relationship between the atria and the ventricles. More specifically, **a consistent PR interval reveals that the rhythm is supraventricular in origin.**

Atrial depolarization (P wave) followed by ventricular depolarization (QRS) a consistent period thereafter yields a consistent PR interval, strong evidence for a connection between the atrial conduction system and the ventricular conduction system. A consistent PR interval occurs with supraventricular rhythms (sinus, atrial or junctional).

Perhaps as important, an irregular PR interval or the presence of lonely P waves (a P wave without a QRS complex immediately after) also speaks volumes. The presence of lonely P waves usually signify the presence of second or third degree AV block (AV blocks are addressed later in this chapter). The presence of an entirely chaotic PR interval across a ECG rhythm strip points to an absence of any relationship between the atria and the ventricles. This is known as atrioventricular (AV) dissociation.

Step 3 calls us to examine the P waves when interpreting an ECG. The shape of the P waves identifies whether atrial depolarization originates in the SA node, the atria or the AV junction. A consistent PR interval is strong evidence that the ventricles are driven by a supraventricular impulse. An irregular PR interval and/or lonely P waves points to various dysrhythmias.

Step 4: Is The QRS Pattern Regular or Irregular?

Regular rhythms can almost always be quickly identified with just steps #2 (Is the QRS narrow or wide?) and #3 (Check the P waves). Simple, basic and fast. These rhythms would include sinus rhythms, atrial rhythms, junctional rhythms and ventricular rhythms of varying rates. The irregular rhythms - those with pauses, extra beats or have a chaotic pattern - often demand closer inspection.

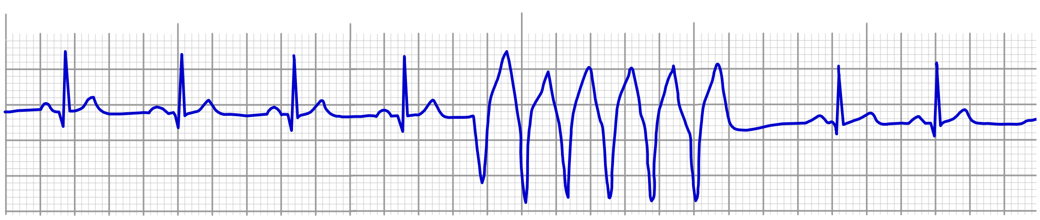
Checking for a regular rhythm pattern simply involves determining whether the R-R interval is consistent. This often can be accomplished with a quick visual snapshot of the rhythm. Are the QRS complexes evenly spaced?

Irregular rhythms should be printed on a rhythm strip of six seconds or longer. First, try to identify any periods of regularity. While the rhythm as a whole may be irregular, segments may be regular. These regular periods are often referred to as *the underlying rhythm*. Apply the four step method to the underlying rhythm first. When naming a rhythm, the underlying rhythm is identified before any irregular rhythm components.

Next study the segments of the rhythm that stand out as different. This may be early beats, periods where QRS complexes are absent, or other inconsistencies. Apply the 4-step method to these segments.

An example may help clarify this process. In Figure 5.5, the rhythm as a whole is not regular. The underlying rhythm begins and ends this rhythm. First apply the 4 step to the underlying rhythm. Using the triplicate method (see Chapter 4), the rate is somewhere around 75-100/minute. The QRS complex is narrow. The impulse originates above the ventricles. Where? The P waves are upright, a sign of a sinus focus. The underlying rhythm is a sinus rhythm.

Figure 5.5 A Rhythm Strip An With Irregular Pattern



Note the narrow, regular QRS complexes that begin and end this rhythm strip. Sinus rhythm is the underlying normal rhythm. The brief run of wide QRS complexes (ventricular tachycardia) stands out from the underlying rhythm. First name the underlying rhythm and finish with a description of any abnormal rhythm components. The rhythm in Figure 5.5 then would be identified as a sinus rhythm with a run of ventricular tachycardia.

The faster segment in the middle of this six second strip requires a second pass of the four step method. The rate is fast at about 240/minute. The QRS complexes are wide with no visible P waves. This is most likely a run of ventricular tachycardia.

An interpretation of this rhythm begins with the underlying rhythm followed by a description of any remaining rhythm features. You would call this rhythm a **sinus rhythm with a run of ventricular tachycardia**.

When rhythms have abnormal components that throw off the pattern of the rhythm, look for the parts of the rhythm that have a narrow QRS AND maintain a pattern. The age old task of picking out the "parts that are not the same", as the Sesame Street jingle goes, helps establish what is the main rhythm and what is not. **First identify the normal - or underlying - rhythm using the four step method. Then apply the four step method again to any of the remaining waveforms.**

Occasionally, the entire rhythm is chaotic, devoid of any pattern. This chaotic nature is typical of atrial fibrillation and ventricular fibrillation. Use the four step method to determine where the impulse initiates. At this point, a differential of possible rhythms is useful. An even closer inspection of the rhythm is often required.

Knowledge of select rhythms that fall outside the normal spectrum will prepare you for this eventuality. These special rhythms are not numerous, nor are they particularly difficult to interpret. These rhythms will be addressed shortly. But first, give the 4-step method a try with a few practice rhythms. You may find that rhythm interpretation can be fast and quite simple.

Applying the 4 Step Method: Practice Rhythms

The 4 step process is simple and fast. But does it work? Try a few six second rhythm strips for practice.

Practice Rhythm #1

Rate's OK

Narrow QRS

Upright P

Regular

1. Too fast? ___ Too slow? ___ Rates OK? ___

2. QRS wide or narrow? ________________.

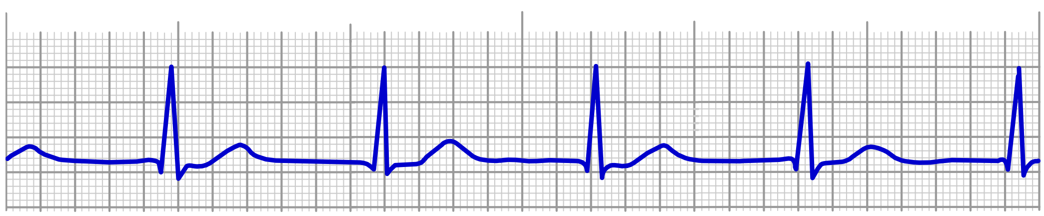
3. Check the P waves. The rhythm comes from the ________________________.

4. Is the pattern regular or irregular? ___________________.

The rhythm is _____________________________________.

Answer: The heart rate is about 90/minute, a safe rate, not a tachycardia nor a bradycardia. The QRS is narrow. It's a supraventricular rhythm. Where specifically in the top does the rhythm come? Check the P waves. The P waves are upright with a consistent PR interval. The impulse originated from the SA node. The pattern is regular. Since it originates from the SA node, this is a **sinus rhythm**.

Practice Rhythm #2



1. Too fast? ___ Too slow? ___ Rates OK? ___

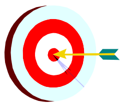
2. QRS wide or narrow? _____________________.

3. Check the P waves. The rhythm comes from the ________________________.

4. Is the pattern regular or irregular? ___________________.

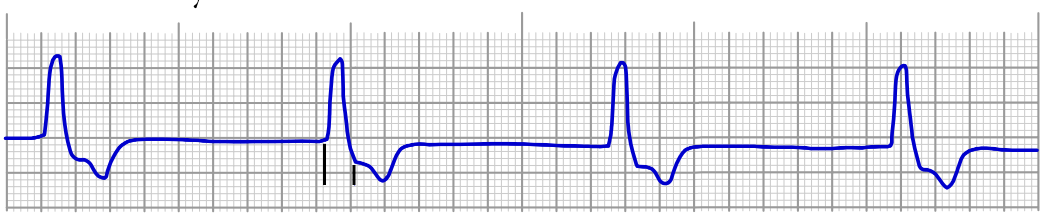
The rhythm is _____________________________________.

Answer: The heart rate is about 50/minute, an acceptable rate. The QRS is narrow making it a supraventricular rhythm. The P waves are absent. The pattern is regular. The only plausible explanation: the impulse comes from low in the AV junction (see Chapter 4 for more on the significance of P waves). This is a **junctional rhythm**. Since the heart rate is typical for the junction (40-60/minute), this rhythm is called a junctional rhythm.



Pacemaker sites can deliver impulses faster than typically expected. For example, pacemakers within the ventricles tend to generate rates of 20-40/minute. But these same pacemakers can, in certain circumstances, yield ventricular tachycardias (>100/minute). But what of rhythms that are not quite tachycardias but are faster than what is expected from a pacemaker site. These rhythms are called **accelerated rhythms**. For example, the junction usually fires at rates of 40-60/minute. A junctional rhythm with rates of 61-100/minute is called an accelerated junctional rhythm. Likewise, a ventricular rhythm with rates of 41-100/minute is an accelerated ventricular rhythm (or an accelerated idioventricular rhythm).

Practice Rhythm #3



1. Too fast? ___ Too slow? ___ Rates OK? ___

2. QRS wide or narrow? ________________________.

3. Check the P waves. The rhythm comes from the ___________________________.

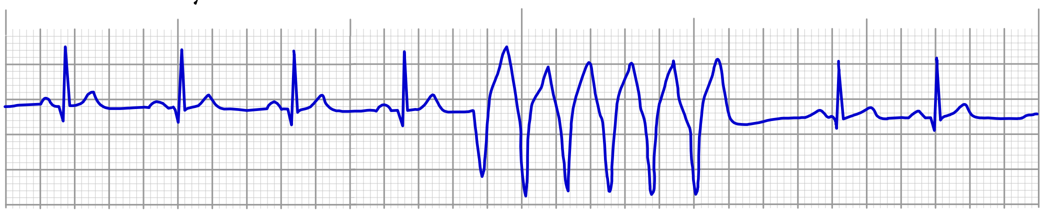
4. Is the pattern regular or irregular? ______________________.

The rhythm is ____.

Answer: The heart rate is about 40/minute. Too slow. The QRS is wide and the R wave is pointing opposite the T wave. Note: measure the width of the QRS from the beginning of the QRS to the end of the S wave, at the J-point. The pattern is regular, typical of a pacemaker site. P waves are absent.

The clues point to a **ventricular rhythm** (the ventricles normally fire at rates of 20-40/minute). This is also called an idioventricular rhythm (solely ventricular) or a ventricular escape rhythm (escaped from the dominance of the SA node).

Practice Rhythm #4



A. First apply the 4 step method to the underlying rhythm (narrow QRS complexes).

1. Too fast? ___ Too slow? ___ Rates OK? ___

2. QRS wide or narrow? ______________________.

3. Check the P waves. The rhythm comes from the ________________________.

4. Is the pattern regular or irregular? ____________________.

The underlying rhythm is ______________________________________.

Answer: The heart rate is about 90/minute. The QRS is narrow. The underlying rhythm has a regular pattern. It's a supraventricular rhythm. P waves are upright. Therefore, the impulse originates from the SA node. This is a **sinus rhythm**.

B. Next apply the 4 step method to the ectopic (extra) waveforms.

1. Too fast? ___ Too slow? ___ Rates OK? ___

2. QRS wide or narrow? ______________________.

3. Check the P waves. The rhythm comes from the ________________________.

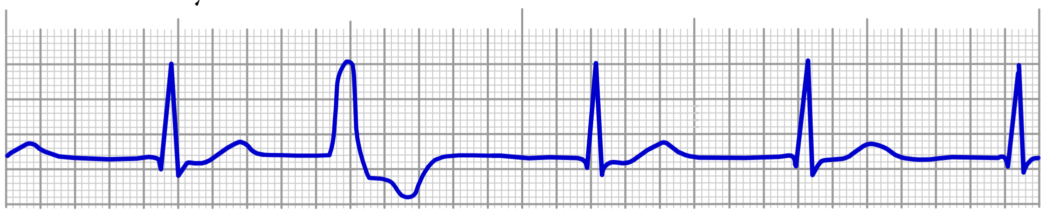
4. Is the pattern regular or irregular? ____________________.

The ectopics are ____________________________________.

Answer: The heart rate is about 240/minute. This is a tachycardia. The QRS is wide. Pattern is regular. It's most likely a ventricular rhythm. P waves are absent. This is a run of **ventricular tachycardia**.

What would the full descriptive name for this rhythm strip? This is a **sinus rhythm with a run of ventricular tachycardia**. Again, it sounds slick but the method is simple, quick, and effective.

Practice Rhythm #5



A. First apply the 4 step to the underlying rhythm (narrow QRS complexes).

1. Too fast? ___ Too slow? ___ Rates OK? ___

2. QRS wide or narrow? ______________________.

3. Check the P waves. The rhythm comes from the ________________________.

4. Is the pattern regular or irregular? ____________________.

The underlying rhythm is a ______________________________________.

Answer: The heart rate is about 50/minute. The QRS is narrow. The pattern is regular. Therefore, it's a supraventricular rhythm. P waves are absent.The rate is not a tachycardia and typical for the junction, making it a junctional rhythm - not an **accelerated junctional rhythm**

B. Next apply the 4 step to the ectopic (extra) waveforms.

1. Too fast? ___ Too slow? There's only 1 complex here.

2. QRS wide? Yes or No. If yes, go to step #4.

3. QRS narrow? Yes or No. If yes, you have a supraventricular rhythm.

4. Check the P waves. The rhythm comes from the ________________________.

The ectopic is a______________________________________.

Answer: The QRS is wide and it arrives earlier than expected (a premature complex). The impulse originates from the ventricle. P waves are absent. This is a **premature ventricular complex or PVC**.

This is a junctional rhythm with a PVC - not

an **accelerated junctional rhythm with one PVC.**

While tachycardias are any rhythms with rates over 100/minute, bradycardias are not so straight forward. **The term 'bradycardia' describes a heart rate less than the normal rate expected of the originating impulse site.** For example, a rate less than 60/minute is a bradycardia for the SA node and the atria. For rhythms originating from the junction which normally delivers impulses at 40-60/minute, a junctional bradycardia is less than 40/minute. Ventricular bradycardia (less than 20/minute) is also called an agonal rhythm for it is only rarely associated with a pulse.

The 4-step method follows a systematic, patient-centered approach to cardiac rhythms. Simply:

- **Is the heart rate too fast or too slow?** Check the patient for signs of poor cardiac output
- **Is the QRS wide or narrow?** A narrow QRS ensures that the rhythm is supraventricular. A wide QRS often occurs with ventricular rhythms
- **Check the P waves.** If the QRS is narrow, the shape of the P waves identifies the origin of the supraventricular rhythm (sinus, atria or junction). A consistent PR interval before each *wide* QRS complex suggests a supraventricular rhythm with aberrant ventricular conduction
- **Is the rhythm regular or irregular?** Regular rhythms are generally straightforward. Irregular rhythms often require a second look. Naming these irregular rhythms often require a familiarity with the cause and inner workings of these rhythms.

As mentioned earlier, several ECG interpretation methods are successfully employed in critical care units globally. The 4-step method advocated for **The Six Second ECG** has evolved to identify rhythms in six seconds or less while focusing on the patient's clinical picture. This step-by-step simple and easily learned approach tends to dispel the mystery of cardiac rhythms.

The five preceding practice rhythms were successfully identified using this four step method. The first three rhythms were regular sinus, junctional and ventricular rhythms. The fourth and fifth rhythms, while mostly regular, had irregular aspects that required a second pass of the four steps to arrive at an impressive, detailed interpretation The process, though, remains simple.

Rhythms Requiring Special Consideration

While all rhythms can be identified with the four step method, some rhythms do not follow typical naming conventions. These select rhythms are named according additional criteria beyond just the location of the initiating impulse. This additional criteria often describe the physiology of the rhythm (i.e. chamber fibrillation, AV nodal blocks, SA nodal failure). To equip you with the ability to identify the full range of ECG rhythms, this section explores each of these select rhythms.

Select Ventricular Rhythms

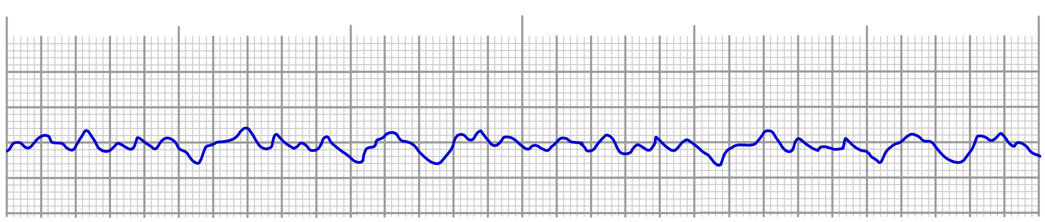
Ventricular rhythms are usually easy to recognize. Monomorphic ventricular tachycardia (often called just ventricular tachycardia or even VT), for example, is quickly identified with its rapid, regular and wide QRS complexes. Similarly, ventricular rhythm (also called idioventricular rhythm or ventricular escape rhythm) is a slow regular rhythm of wide QRS complexes at a rate of 20-40/minute. P waves are absent. Please refer to Chapter 4: An ECG Primer for examples.

Because of the lack of atrial kick (no P waves) and the tendency to occur at extreme rates, ventricular rhythms are often ominous, requiring an urgent response. While identifying ventricular rhythms with a regular pattern is often rudimentary, a few select ventricular rhythms merit special mention - ventricular fibrillation, torsades de pointes and asystole. These three ventricular rhythms have unique names and unique defining criteria.

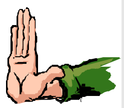
Ventricular Fibrillation

Ventricular fibrillation is the most common presenting rhythm for victims of sudden cardiac death. Rather than pumping as a cohesive unit, multiple sites in the ventricles fire simultaneously, effecting only a quiver. A resulting fibrillation rate is 350-600/minute.

Figure 5.6 Ventricular Fibrillation



The presence of chaotic ventricular fibrillatory waves of at least 3 mm in amplitude is often referred to as **coarse ventricular fibrillation**. It follows, then, that shallow ventricular fibrillatory waves (less than 3 mm in amplitude) is called **fine ventricular fibrillation**. The larger amplitude of coarse VF represents a higher degree of ventricular electrical activity, a positive indicator for successful defibrillation.



Note that what looks like ventricular fibrillation may also be a loose or unconnected lead wire. When considering the treatment for this rhythm - a high-energy electrical discharge across the heart - it should come as no surprise that ventricular fibrillation must be initially confirmed with a pulse/circulation check.

Cardiac output is non-existent with ventricular fibrillation. Without the benefit of a cardiac monitor, the patient appears lifeless: pulseless and without respirations. The definitive treatment for ventricular fibrillation is rapid defibrillation with ample current across the ventricular myocardium. For every minute, the likelihood of a successful resuscitation for a patient in VF falls by about 10%. Recognizing this rhythm and responding accordingly truly is a matter of life and death.

Asystole

Most students of dysrhythmia courses arrive with the claim that they are at least skilled in recognizing asystole. In fact, most of us have seen the proverbial straight line on countless television programs such as ER and movies such as Flatliners. Contrary to most of these story lines, a patient with asystole seldom regains a pulse.

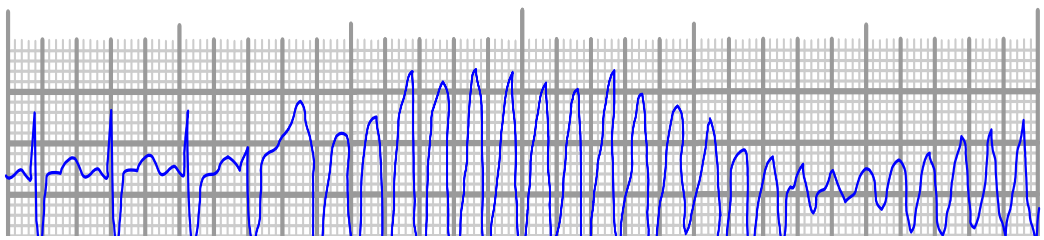
Asystole is characterized by the absence of any waveforms. In reality, asystole is seldom a bone straight line until the patient has been without a pulse for some time. It is good practice to check lead integrity to ensure that the rhythm seen is not another case of disconnected leads. Progress through all of the leads available through your cardiac monitor. Also, increase the gain or size to ensure that fine electrical activity is not present.

In the process towards asystole, the occasional occurrence of grossly wide and progressively irregular QRS complexes forms an **agonal rhythm**. The presence of an agonal rhythm is as serious as asystole, prompting many health care practitioners to consider stopping any further attempts at resuscitation.

Torsades de Pointes

Associated with long QT syndrome and any drugs that prolong the QT interval (quinidine, procainamide), Torsades de Pointes (TdP) is a polymorphic - many shapes - ventricular tachycardia. It can be differentiated from ventricular fibrillation in that TdP has an undulating pattern of increasing and decreasing amplitude. Ventricular fibrillation is chaotic, devoid of any pattern.

Figure 5.7 Torsades de Pointes



Torsades de Pointes is often associated with hemodynamic compromise. Notice in the prolonged QT interval present prior to the episode of TdP. Treatment includes discontinuing medications that may prolong the QT interval, administering magnesium and overdrive pacing.

Select Atrial Dysrhythmias

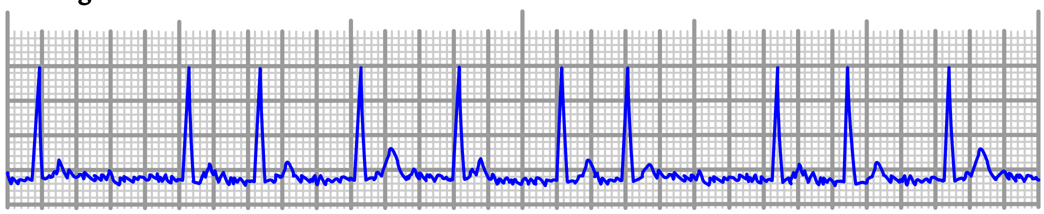
A few atrial rhythms fall outside the traditional naming conventions expected when using the 4 step method. The 4 step method will successfully identify the location of the initiating impulses. Nevertheless, the accepted names of these rhythms are unique. These rhythms also have unique defining characteristics. Rhythms outlined in this section include atrial fibrillation, atrial flutter and multifocal atrial rhythm.

Atrial Fibrillation

The most common supraventricular dysrhythmia is atrial fibrillation. About 1 in 5 people over the age of 50 will develop atrial fibrillation at some time in their lives. Mechanically, the atria are only quivering. Atrial kick is lost.

In an otherwise healthy population, atrial fibrillation is often symptom free. For the aged and those with cardiac illness, atrial fibrillation can be associated with hemodynamic compromise and cardiac ischemia. Generating further concern, the lack of an effective atrial contraction is associated with blood pooling and the potential development of a clot in the atria (particularly after 48 hours). The dislodging of this emboli can lead to a pulmonary embolism or a stroke.

Figure 5.8 Atrial Fibrillation



Recognizing atrial fibrillation usually requires only the ability to distinguish that the **rhythm is chaotic**. There is no pattern to this rhythm. If a rhythm has QRS complexes with no recognizable pattern **and** P waves are indistinguishable, rhythm is probably atrial fibrillation.

> A chaotic cardiac rhythm with QRS complexes (most often narrow) is most likely atrial fibrillation.

Using the 4 step method, the rhythm of figure 4.8 is not too fast or too slow. The rhythm is supraventricular. The absence of P waves and its chaotic nature could stump us. Instead of P waves, irregularly shaped fine fibrillation waves exist. Note fibrillation (atrial or ventricular) produces rates of 350-600/minute. With atrial rates this high, fortunately the AV junction ensures that many of these atrial depolarizations do not make it through to the ventricles.

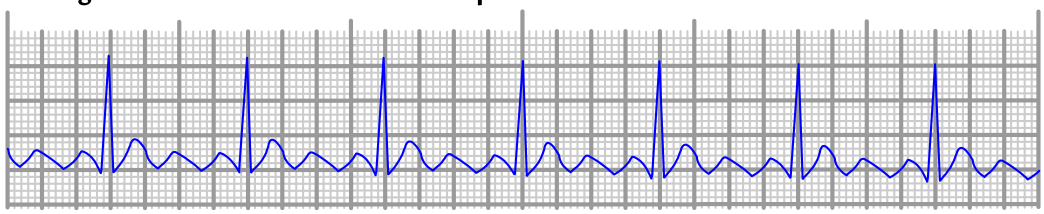
Atrial fibrillation with rates less than 60/minute is called atrial fibrillation with slow ventricular response. Atrial fibrillation with rates of 60-100/minute is called atrial fibrillation or atrial fibrillation with a controlled response. Atrial fibrillation with rates faster than 100/minute is called atrial fibrillation with uncontrolled response.

Atrial Flutter

Atrial flutter occurs from a rapid conducting loop (reentrant) within the atria that fires at rates between 260-340 minute. Typically, though, the atria flutter at rates close to 300/minute. This rate is faster than the AV junction's capacity (maximum is 240 impulses/minute). As a result, the AV junction conducts only the 2nd, 3rd or 4th impulse to the ventricle, usually in a consistent pattern.

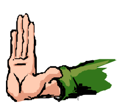
With only every 3rd or 4th impulse conducted, an abundance of P waves are seen between the QRS complexes. These flutter ('P') waves resemble the teeth of a saw. Flutter waves are easily seen at these slower ventricular rates.

Figure 5.9 Atrial Flutter with 4:1 Response



Since the atria flutter at rates of about 300/minute, a 2:1 conduction is the most common presentation of atrial flutter with ventricular rates of close to 150/minute. With 2:1 conduction, flutter waves do not usually appear (P waves are generally buried in either the QRS or the T wave). For any supraventricular rhythm with a rate close to 150/minute, consider atrial flutter.

If the patient is in atrial flutter, a vagal maneuver can slow the AV node, effectively increasing the number of visible flutter waves. Various vagal maneuvers are discussed in on page 43.



If a patient is in supraventricular tachycardia with a heart rate close to 150/minute, always consider the possibility that the rhythm is atrial flutter with a 2:1 block. Failure to detect this rhythm can lead to serious complications for those with Woolf-Parkinson-White syndrome or occasionally for those who have been in atrial flutter for more than 48 hours (risk of emboli).

Atrial flutter is often grouped with atrial fibrillation. Risk factors of atrial fibrillation can also apply to atrial flutter, though with less frequency. Atrial flutter also has a tendency to deteriorate into atrial fibrillation.

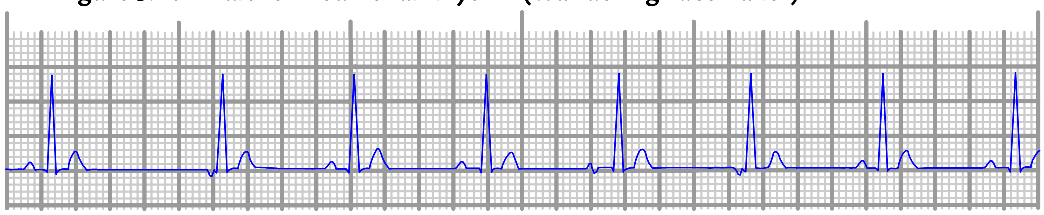
The 4 step process would probably take us to atrial flutter. The P waves are more indicative of an atrial locus. The appearance of more than one P wave between each QRS complex is a somewhat unique condition.

The simplest way to detect the number of flutter waves for every QRS complex is to complete some simple math. Determine the ventricular rate. If the atria are fluttering at rates close to 300/minute, divide the ventricular rate into 300 and round off your answer. For example, if the ventricle rate is 100/minute with flutter waves, the atria flutters 3 times for every (300/100) ventricular depolarization. You would call this rhythm atrial flutter with a 3:1 response.

Wandering Pacemaker and MAT

Various events cause increased automaticity throughout the atria which may lead to a phenomenon whereby several atrial foci compete for dominance. Called a multifocal atrial rhythm or wandering pacemaker, the 'P' waves can come from the SA node, the AV junction and anywhere in the atria. Three different P wave morphologies (shapes) are necessary to call this a multiformed atrial rhythm. If the rhythm is a tachycardia, it is called multifocal atrial tachycardia (MAT).

Figure 5.10 Multiformed Atrial Rhythm (Wandering Pacemaker)



A multifocal atrial rhythm is often an irregular rhythm. For MAT with rates greater than 140/minute, 'P' waves are difficult to differentiate from T waves. Rapid atrial fibrillation may look quite similar to rapid MAT. Note: 3 different 'P' wave shapes are necessary to call tachycardias MAT.

Multifocal atrial rhythm occurs with conditions such as chronic obstructive pulmonary disease (COPD), digitalis toxicity and atrial hypertrophy (commonly related to pulmonary hypertension associated with COPD).

Atrioventricular Blocks

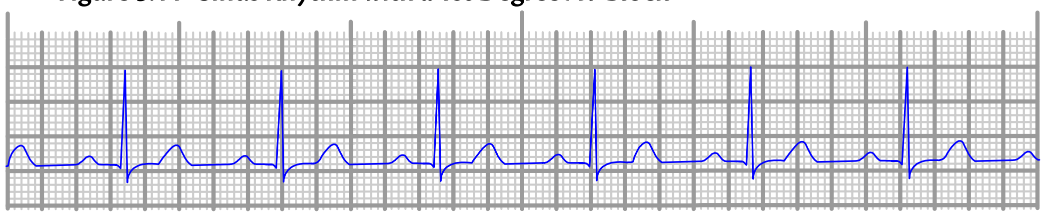
Atrioventricular blocks (AV blocks) result from a conduction disturbance at or just below the AV junction. The 4th step of the 4 step process prompts us to check the P waves and the PR interval. Abnormal PR intervals and lonely 'P' waves define the type of AV block.

From a clinical perspective, the severity of a block is similar to the severity of burns. The higher the degree of burn the more aggressive the treatment. Similar escalation in treatment is required for higher levels of AV blocks. The affects of 2nd degree type II and 3rd degree AV blocks on cardiac output can be much more significant than the affects of 2nd degree type I and 1st degree AV blocks.

First Degree AV Block

First degree AV block is simply conducts slower through the AV junction. First degree AV block can be a benign finding (particularly athletes). Other causes include ischemia, increased vagal tone, and the effects of medications that slow conduction across the AV junction: digoxin, calcium channel blockers, and beta blockers for example. As a result of the slowed junctional conductivity, the PR interval of first degree AV block is longer than normal (> 5 mm in width).

Figure 5.11 Sinus Rhythm with a 1st Degree AV Block



The significance of 1st degree block is revealed in its position in the naming of a rhythm. The underlying rhythm is identified first followed by descriptors of any abnormal components such as 1st degree AV block (see figure 4.11).

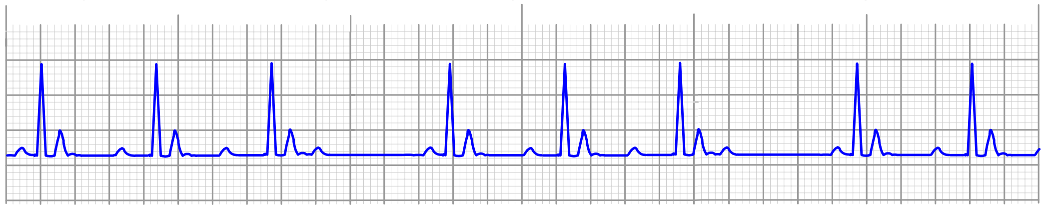
Second Degree AV Blocks

Second degree AV blocks have lonely P waves with a PR interval that confirms an atrioventricular association. There are 3 varieties of 2nd degree AV block: 2nd degree AV block Type I (sometimes called Wenckebach); 2nd degree AV block Type II (sometimes called Mobitz II); and a hybrid of the two called 2nd degree AV block 2:1 conduction.

Second Degree AV Block Type I

Metaphorically, picture the junction as a gate. In 2nd degree AV block type I, the gate slowly closes with the time taken for the impulse to get through the gate progressively getting longer. The PR interval progressively lengthens as a result. Eventually, the gate closes and the atrial wave is not carried to the ventricles - a lonely P wave results. With a dropped QRS, the gate then springs back open to begin the process again (PR interval begins narrow once again).

Figure 5.12 Second Degree AV Block Type I (Wenckebach or Mobitz Type I)



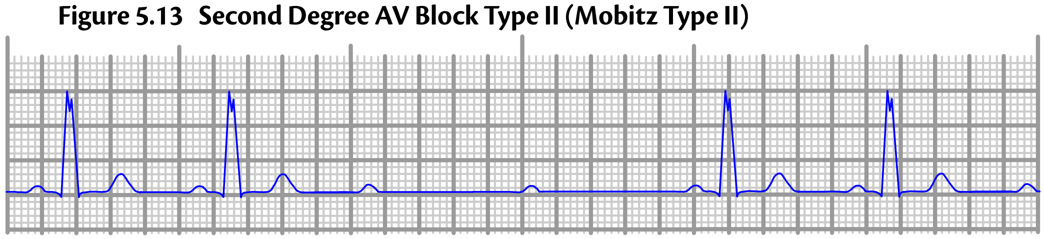
Second degree AV block type I is recognized by cycles of lengthening PR intervals that terminate a lonely P wave. This cycle repeats forming a pattern. Although the loss of every 3rd or 4th beat impacts cardiac output minimally, this rhythm does require monitoring. Aggressive treatment is usually not required.

Second degree AV block type I occurs at the AV node. Causes of second degree AV block are identical to first degree AV block. Typically, the patient is monitored, placed on oxygen and assessed for signs of cardiac ischemia.

Second Degree Block Type II

Second degree AV block type II is a significant dysrhythmia due to 1) its unpredictability; 2) its potential to cause consecutive dropped QRS complexes and 3) its tendency to progress to a complete heart block (third degree AV block). Second degree AV block is identified with lonely 'P' waves and a fixed PR interval. The metaphorical gate is either open or closed.

Figure 5.13 Second Degree AV Block Type II (Mobitz Type II)



Second degree AV block type II occurs at the level of the bundle branches (common) or at the bundle of His (much less common). This dysrhythmia often has includes aberrant conduction through the ventricles (bundle branch block - see figure 4.13). Causes of second degree AV block type II include cardiac ischemia and an anteroseptal myocardial infarction.

Second Degree AV Block with a 2:1 Conduction

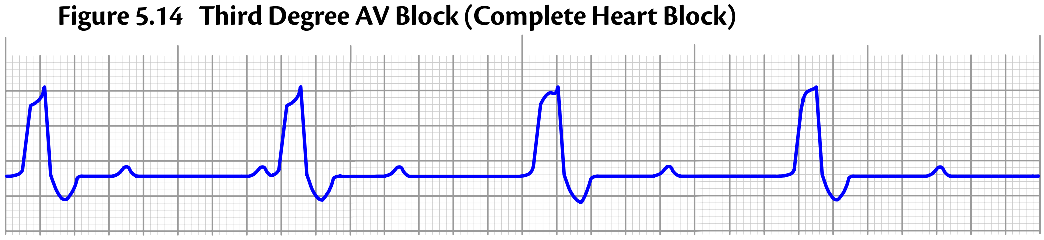
A hybrid form of second degree AV block is a block with 2:1 conduction where every second 'P' wave is a lonely 'P' wave. The PR interval is fixed. Cardiac output is often impacted (heart rate is reduced by 50%). This rhythm cannot be identified with certainty as a second degree AV block Type II or a special form of Type I. Instead, this rhythm is best called a second degree AV block with a 2:1 conduction.

Third Degree AV BLock

Third degree AV block (complete heart block) can occur at any part of the junction or further down in the bundle branches. Instances of third degree heart block occurring high in the AV node often have a narrow QRS complex (pacing below the block is provided by the bundle of His producing a narrow QRS complex). Third degree AV block at the bundle of His or the bundle branches, a wide QRS often results (pacing below the block is provided by the ventricles).

Note that third degree AV block with narrow QRS complexes may respond favorably to the use of a vagolytic medication such as Atropine. A third degree AV block with wide QRS complexes, though, is rarely responsive to Atropine. The Vagus nerve lands on the SA and AV node only. For AV blocks that are located below the junction (third degree and second degree type II), Atropine is not the treatment of choice.

Figure 5.14 Third Degree AV Block (Complete Heart Block)



With complete heart block, the atria and the ventricles are electrically severed. This is one form of atrioventricular dissociation. Because the atria and the ventricles are beating to two separate drummers, **the defining characteristics of third degree AV block are: 1) regular ventricular (and atrial) rhythms; and 2) chaotic PR interval.** This dysrhythmia often requires urgent attention (dependent on ventricular rate) with treatments that include transcutaneous pacing.

Electronically Paced Rhythms

An electronic pacemaker, or just pacemaker, delivers electrical current to the heart to stimulate depolarization. Pacemaker leads may connect with ventricular tissue and/or atrial tissue (permanent or transvenous pacemakers). Transcutaneous pacing delivers an electrical current through the chest wall to depolarize the ventricles.

Figure 5.15 Paced Ventricular Rhythm

Paced rhythms are fairly easy to detect because of the vertical spike present before the atrial ('P' waves) or ventricles depolarizations (QRS complexes). The spike represents the electrical impulse generated by the electronic pacemaker.

The wave immediately after the spike is the intended response to the initial electrical stimulus. If the spike is accompanied by a wave, the pacemaker is said to be **capturing**. Conversely, if spikes appear without accompanying waves, we have **loss of capture**.

Note that many older monitors will recognize the pacer spike as a QRS complex when determining rate, whether the spike captures the heart or not. As a result, many monitors do not alarm if the pacemaker is losing capture. Also, a monitor might provide a heart rate twice the pulse rate if both the pacer spike and the QRS complex are counted. Monitor your patient closely.

Pacer spikes should always have a waveform immediately afterwards. Failure to stimulate a depolarization is called **loss of capture**. Many older monitors will not inform you of any loss of capture. Again, always check the patient. The loss of capture of several complexes could result in poor cardiac output. Also, if the pacemaker is sensing the heart ineffectively, the pacemaker could deliver electrical impulses close to the T wave, potentially causing lethal dysrhythmias. Loss of capture or the presence of pacer spikes close to the T wave often requires an urgent response.

Summary

This chapter provided a 4 step method for fast and simple ECG interpretation. While most rhythms are rapidly and successfully identified using the 4 step process, select rhythms are named uniquely, rather than only by location of the initiating impulse and rate. Many of these rhythms were also outlined.

Interpreting an ECG can be a simple and fast process. The 4 step method works particularly well in the presence of cardiac emergencies, where rapid response is often vital. The 4 steps are first applied to the underlying rhythm and then applied with ectopic waveforms. The 4 steps are:

1. **Is the rate too fast (>150/minute) or too slow (<50/minute)?** If so, check the patient for signs and symptoms of shock.

2. **Is the QRS wide or narrow?** A wide QRS strongly suggests a ventricular rhythm. A narrow QRS indicates a supraventricular rhythm.

3. **Check the P waves.** The shape of the P waves points to where supraventricular rhythms originate - SA node, atria or the junction.

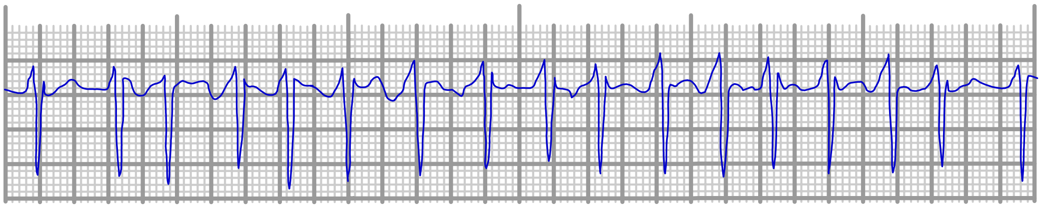
4. **Is the pattern regular or irregular?** An irregular pattern often requires closer inspection.

After determining the rhythm, a careful look at each of the ECG components increases the accuracy of any interpretation. For example, is the ST segment, PR interval or QT segment abnormal. Knowing the characteristics of select rhythms such as ventricular fibrillation, atrial fibrillation, atrial flutter and multiformed atrial rhythms facilitates a quick interpretation of these rhythms.

The ECG is a superior non-invasive diagnostic tool. It is best interpreted with the added perspective of a patient history and a physical assessment.

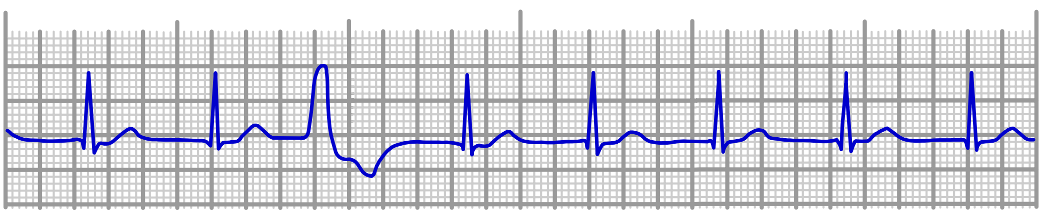
Chapter Quiz

1.This rhythm is called:
:



a) atrial flutter with 2:1 response
b) atrial fibrillation with uncontrolled response
c) ventricular tachycardia
d) junctional tachycardia

2. This rhythm is called:

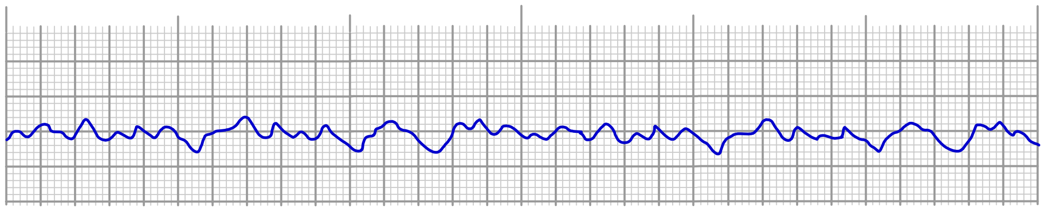


a) sinus rhythm with a PAC
b) junctional rhythm with a PJC
c) accelerated junctional rhythm with a PVC
d) sinus rhythm with a PVC

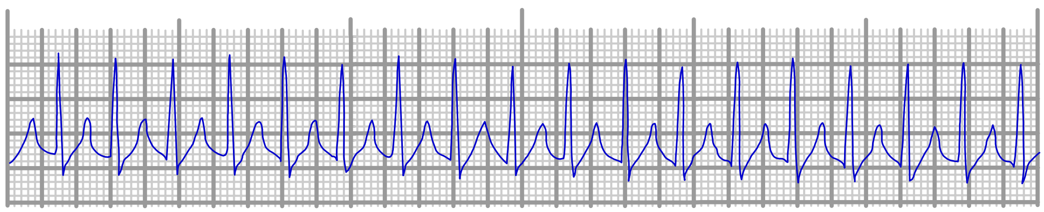
3. The QRS complex measures (.12 seconds,.08 seconds,.16 seconds) and the PR interval measures (.10 seconds,.12 seconds,.16 seconds,.20 seconds). This rhythm originates from the (supraventricular, ventricular) region of the heart.

Answers: **1.** b) The fast chaotic rhythm strongly suggests atrial fibrillation; **2.** c) A narrow QRS with absent P waves; **3.** 0.08 seconds, 0.20 seconds, supraventricular;

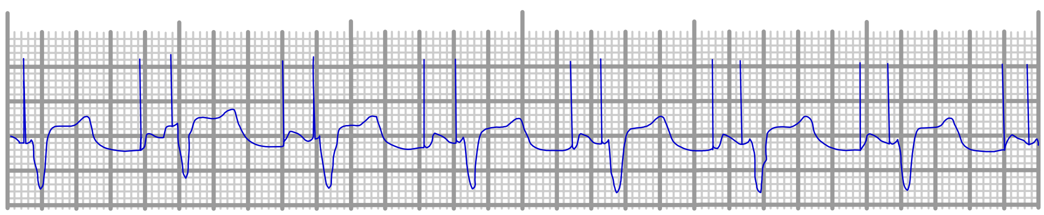
4. This rhythm is called ______________________________.



5. This rhythm is called ______________________________.

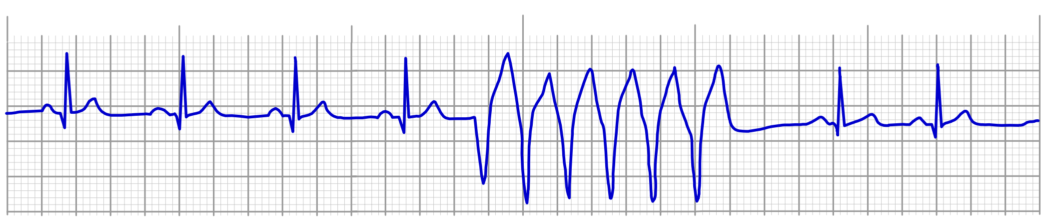


6. Identify the following rhythm:



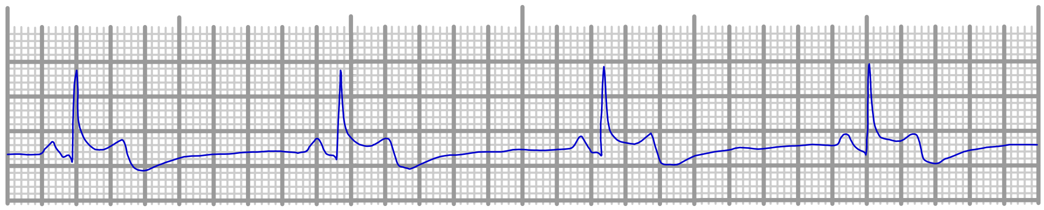
a) sinus rhythm with artifact
b) supraventricular tachycardia
c) ventricular pace rhythm
d) paced AV sequential rhythm

7. The following rhythm is ______________________________.

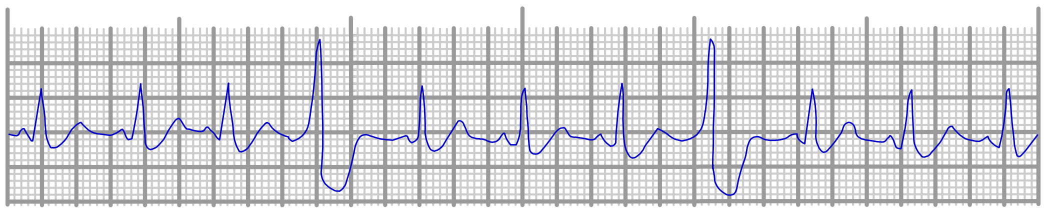


Answers: **4.** ventricular fibrillation; **5.** supraventricular tachycardia; **6.** d); **7.** sinus rhythm with a run of ventricular tachycardia;

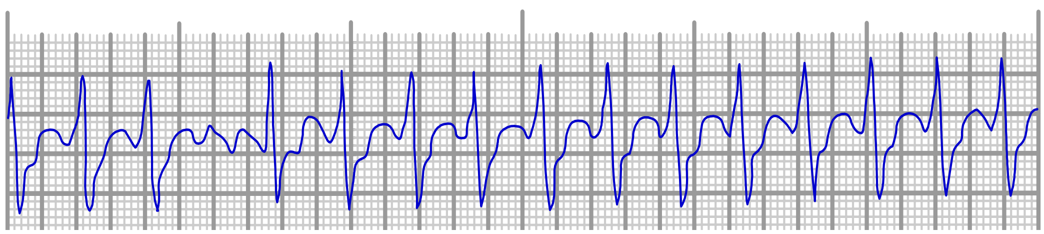
8. The following rhythm is ____.



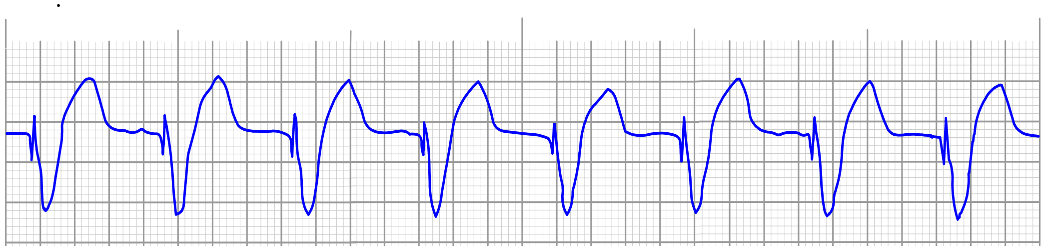
9. The following rhythm is ____.



10. The following rhythm is called____.



11. This rhythm is called:
:



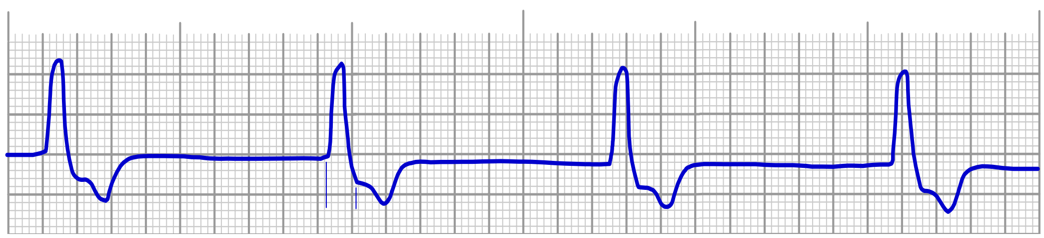
a) sinus rhythm with aberrant conduction
b) accelerated junctional rhythm
c) accelerated idioventricular rhythm
d) paced ventricular rhythm

Answers: **8.** sinus bradycardia with ST elevation; **9.** sinus tachycardia with ST depression and 2 unifocal PVCs; **10.** atrial flutter with a variable ventricular response; **11.**d)

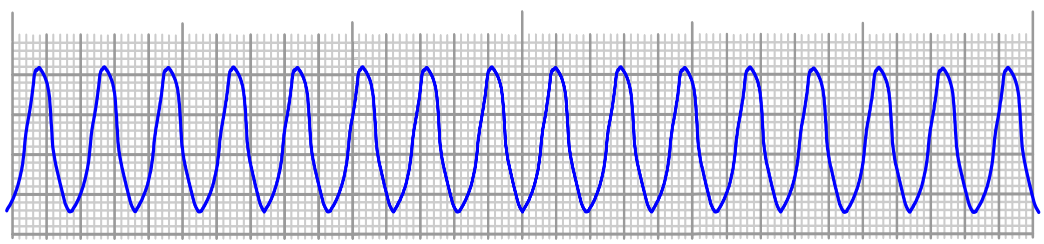
12. This rhythm is called:

a) sinus rhythm with a sinus exit block
b) accelerated idioventricular rhythm
c) second degree AV block type I
d) second degree AV block type II

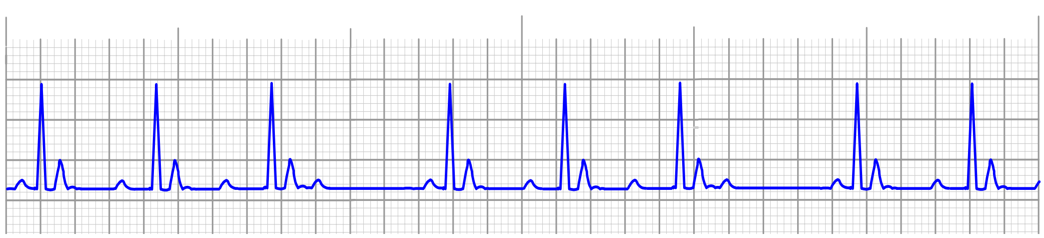
13. The QRS complex measures (.10 seconds,.16 seconds) This rhythm probably originates from the (supraventricular, ventricular) region of the heart. This rhythm is called a(an) ______________________________.



14. This rhythm is called ______________________________.

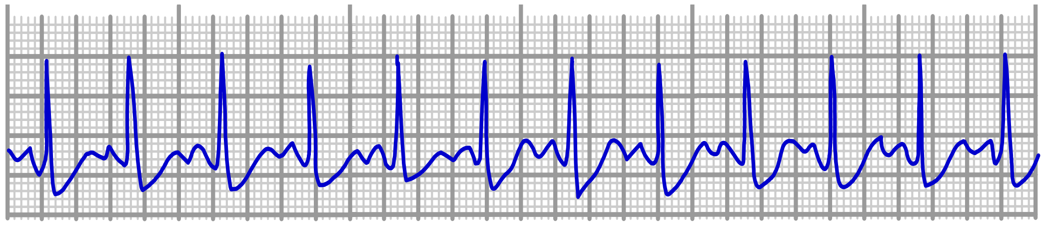


15. This rhythm is called ______________________________.



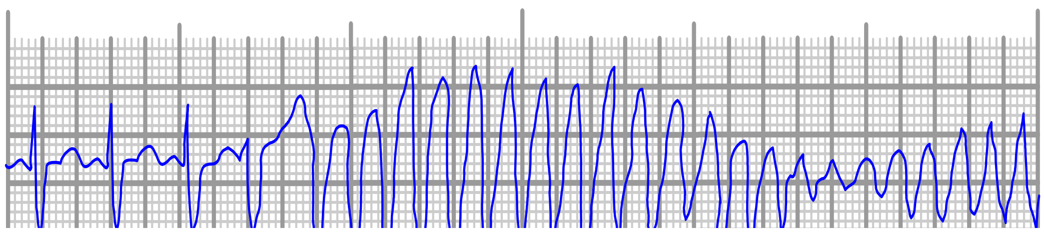
Answers: **12.** d); **13.** 0.16 seconds, ventricular, idioventricular (or ventricular escape rhythm); **14.** ventricular tachycardia; **15.**second degree AV block type I

16. This rhythm is best called:

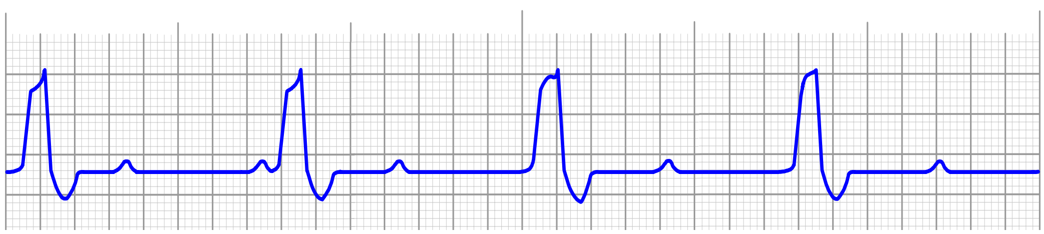


a) sinus rhythm
b) atrial fibrillation
c) sinus tachycardia
d) supraventricular tachycardia

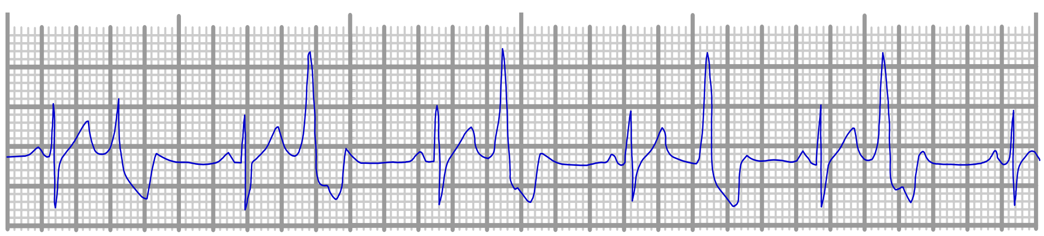
17. The following rhythm is ____.



18. The following rhythm is ____.

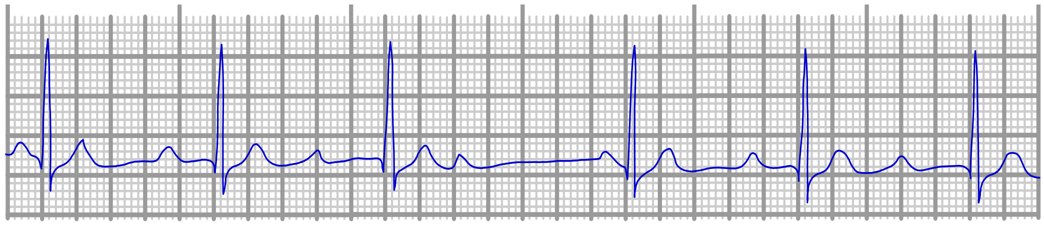


19. The following rhythm is ____.



Answers: **16.** sinus tachycardia with ST depression; **17.** torsades de pointes; **18.** third degree AV block (complete heart block); **19.**ventricular bigeminy

20. The following rhythm is ____.



Suggested Readings and Resources

Aehlert, Barbara. (2001). ECGs Made Easy. 2nd ed. New York: Mosby

Dubin, Dale. (2000). Rapid Interpretation of EKGs. 6th ed. Tampa, Florida: Cover Publishing

ECG Simulator. (2001) SkillStat Learning Inc. Found at http://www.skillstat.com

Grauer, Ken. (1998). A Practical Guide to ECG Interpretation. 2nd ed. New York: Mosby

Thaler, Malcolm S. (1997). The Only EKG Book You'll Ever Need. 2nd ed. New York: Lippincott - Raven

What's Next?

A single lead ECG is an effective and efficient method for continuous cardiac monitoring and rhythm interpretation. For a more comprehensive examination, the 12 lead ECG stands superior. In Chapter 6, the 12 lead ECG will be introduced. Ischemic and infarcted regions of the heart are mapped out. The advantages of certain lead views are highlighted. Also, a method to distinguish between left and right bundle branch blocks is provided.

Answers: **20.** second degree AV block type I

6

The 12 Lead ECG

Quick Look

This chapter completes our coverage of the ECG, expanding the mapping of the heart from a single lead view to 12, 15 or even 18 lead views. While a single (or dual) lead is usually sufficient to interpret and name rhythms, the 12 lead ECG is effective as a diagnostic tool for various cardiac conditions.

A 12 lead ECG *introduction* is the purpose of this chapter. This chapter should be considered a survival guide to making sense of the 12 lead ECG. Further resources to 12 lead ECG interpretation are listed at the end of this chapter.

This chapter focuses on the skills necessary to quickly identify cardiac ischemia, injury and infarction. As well, chamber enlargement, right and left bundle branch block detection are also included. Practice ECGs round out the discussion.

Much can be revealed with a 12 lead ECG. When analyzed with the benefits of previous 12 lead ECGs, a thorough physical assessment and a cardiac history, the 12 lead ECG is a powerful tool.

"The KEY to ECG interpretation is restraint. The key to restraint is using a systematic approach."

Ken Grauer, MD, F.A.A.F.P.

Electrode Placement

The 12 lead ECG provides 12 views of the heart. The 12 views are generated through only 10 electrodes. Four limb electrodes attach to the inner forearms and calves. The remaining 6 electrodes, labelled successively from V1 to V6, are attached to the chest to provide precordial views.

Figure 6.1 Placement of Electrodes for the 12 Lead ECG

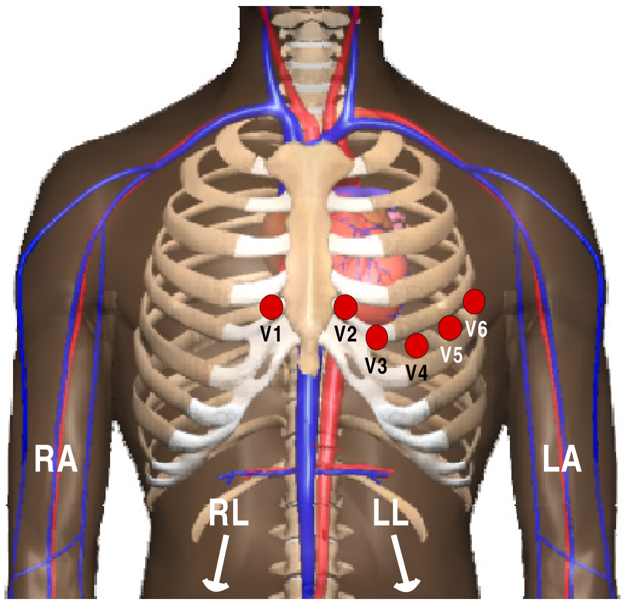
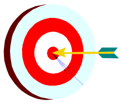


Figure 6.1 displays the locations for the 10 electrodes typical of a 12 lead ECG. Note that the positions for V5 abd V6 vary between institutions. The location is this depiction is for the lateral precordial leads V5 and V^ to follow the 5th intercostal space to the mid-axillary line. Another common configuration places V5 and V6 along the same horizontal axis as V4 with V6 again being in line with the mid-axillary line (though not along the 5th intercostal space). Both configurations are acceptable. The key point is that all 12 lead ECGs should be done using identical lead configurations to prevent the possible benign ECG changes often seen with different electrode placement.

The precordial leads are attached to the chest beginning with V1, placed at the 4th intercostal space (ICS) to the right of the sternum. Lead V2 mirrors V1, but to the left of the sternum. Lead V4 is placed along the mid-clavicular line at the 5th ICS. Lead V3 is placed between V2 and V4. Lead V6 is placed on the 5th ICS along the mid-axillary line (see Figure 6.1). For females, precordial leads V3-V5 should be placed under the left breast.



Lead V1 is situated close to the right ventricle. While technically a septal lead, lead V1 is a precordial chest lead used to differentiate right from left bundle branch block. Lead V1 is often the best lead to search for P waves. Because lead V1 monitors both the right and left sides of the heart, lead V1 is not sufficient to differentiate between left and right ventricular ischemic changes (lead V4R is a superior lead in this role).

Table 6.1 outlines the placement of the 10 electrodes. The location of the leads is an important consideration. For the 12 lead ECG to be an effective tool, electrode placement must be consistent for every 12 lead taken. A common practice is to mark the skin at each electrode (using a pen or marker) with the first 12 lead so that subsequent 12 leads can be compared accurately. Minor lead misplacements can significantly change the ECG waveforms making comparisons dubious at best.

Table 6.1 Electrode Placement for the 12 Lead ECG

Limb Lead Electrodes	Location	Precordial Lead Electrodes	Location
RA - right arm	near wrist on inside of right arm	**V1**	4th ICS to the right of the sternum
RL - right leg	inner right leg near the ankle	**V2**	4th ICS to the left of the sternum
LA - left arm	near wrist on inside of left arm	**V3**	between V4 and V3
LL - left leg	inner left leg near the ankle	**V4**	5th ICS along the midclavicular line
		V5	5th ICS between V4 and V6
		V6	5th ICS along the midaxillary line

Table 6.1 details the electrode placement for the 12 lead ECG. Note that only ten electrodes are used for twelve lead views.

12 Lead Views

The ten electrodes combine to provide 12 views of the heart. The limb leads combine to provide the three bipolar leads (leads I, II, and III) and three augmented voltage leads (aVR, aVL, and aVF). The three bipolar lead views of the heart have already been outlined in Chapter 3. The three "augmented voltage" leads - aVR (right), aVL (left lateral) and aVF (foot) - provide three additional surface views of the heart (see Table 6.2)

Table 6.2 .Lead Views for the Augmented Voltage Leads

Lead	Views
aVR	Upper right
aVL	Upper left
aVF	Inferior

Table 6.2 outlines the three views of the heart that are seen using the augmented leads. These lead views are mathematically created from the limb electrodes to view the heart from three distinct positions towards the centre of the heart. Therefore, the augmented voltage right (aVR) views the right atria and ventricle from the right shoulder (since the heart is generally situated more to the left of the mediastinum, aVR offers the most distant and least useful view of all the 12 leads). The augmented voltage left (aVL) views the left lateral aspect of the left ventricle from the left shoulder. The augmented voltage foot lead (aVF) views the inferior surface of the heart (left and right ventricle) from the feet. Together, the augmented voltage leads offer lateral and inferior views of the heart.

Of the 3 augmented voltage leads, the right lead, aVR, views the heart from the greatest distance (the heart sits more left of the sternum), and is usually ignored when reading a 12 lead ECG. The aVL lead provides another view of the left high lateral aspect of the heart. The aVF lead provides a true inferior view of the heart.

The remaining six views of the heart are provided by the precordial leads V1 - V6. The precordial leads wrap around to the left lateral aspect of the heart, providing septal, anterior and lateral views of the heart. Of all the precordial leads, only V1 views the right side of the heart. Lead V1 is also an excellent lead to check for P waves.

Figure 6.2 Bipolar and Augmented Voltage Lead Views

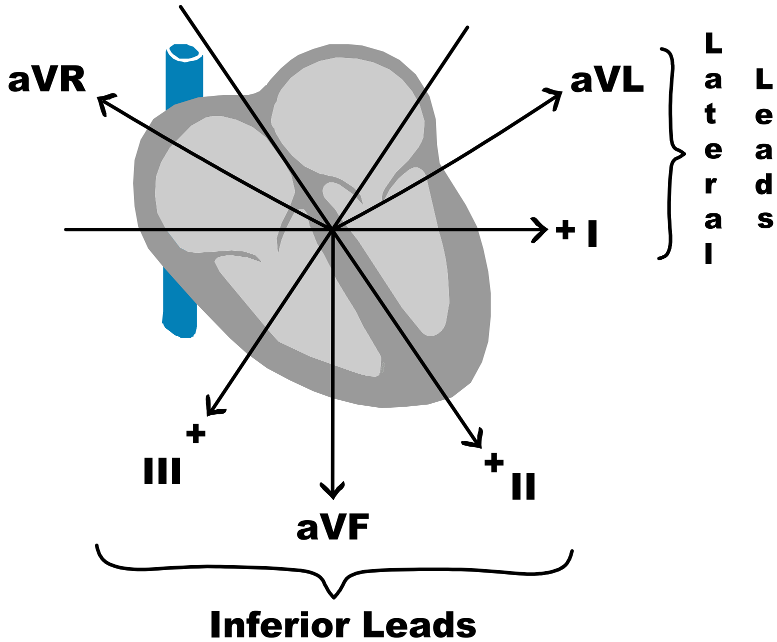


Figure 6.2 depicts the six lead views created from the four limb electrodes. Note that to diagnose a ST elevation myocardial infarction, two leads that view the same region of the heart - contiguous leads - must present with ST elevation of 1 mm or more. With two lateral leads and three inferior lead views, the lead views created by the limb electrodes alone provide significant diagnostic information. The two columns of lead views created by the limb electrodes are commonly found on the left half of a 12 lead ECG.

Virtually all of the precordial leads look directly at the left ventricle with the exception of V1 (see Figure 6.1), which tends to view the right and left ventricles. Leads V1 and V2 view the septum. Leads V3 and V4 provide anterior views of the left ventricle. Leads V5 and V6 give us left lateral views of the heart. Leads V2 and V4 are transitional leads, offering sharing perspective of neighbouring regions of the heart.

Let's take stock of the twelve lead views. This might seem daunting, since we have quickly moved from one or at best two lead views to twelve - a quantum jump from the previous five chapters. When the initial shock wears off, this is just not so. The key is to understand where is the positive and negative reference points for each lead view. The augmented voltage leads and the precordial chest leads all share a similar negative reference point - the theoretical centre of the three-dimensional heart. View this heart centre from each positive lead and yield the region each lead views.

For example, if using the centre of the heart as a negative polarity reference point, lead aVL views the heart from the left shoulder (electrode is placed on the left arm or shoulder). Imagining a tiny video camera from the left shoulder directed towards the centre of the heart, what surface region of the heart is viewed. Left lateral region (left ventricle) is correct. Similarly, what region of the heart is viewed when the video camera is positioned looking up from the feet (inferior view)?

1) Harkening back to Chapter 1, what chamber(s) of the heart are viewed from an inferior perspective?

The three bipolar leads may be slightly more complicated until you recall that the red electrode always has positive polarity and the white electrode always has negative polarity. In lead II (white to red electrode), the positive red looks up to the white visualizing the apex (inferior surface) of the heart along the way.

2) What surfaces of the heart are viewed by lead I and lead III?

Table 6.3 Lead Views for the Precordial Chest Leads

Views	Precordial Leads
Septal	V1, V2
Anterior	(V2), V3, V4
Lateral	(V4), V5, V6

*Table 6.3 provides an easy reference for the precordial chest leads (**SAL**). While the lead views provided by the limb electrodes (I,II,III,aVR,aVL,aVF) provide a frontal plane viewing the heart from inferior and lateral views, the precordial leads add anterior (anteroseptal) and lateral views. The precordial chest leads view the heart directly below their placement. Visualizing where the precordial leads are placed provide a quick and easy reference to what region of the heart each lead monitors (views).*

The three bipolar leads, the augmented voltage leads and the precordial leads map out the left side of the heart quite well. What about the right and posterior regions of the heart?

The 15 and 18 Lead ECG

The inferior leads show the inferior aspects of the heart - the inferior aspects of the right ventricle and the left ventricle. Much of the inferior view of the heart is of the right ventricle. Precordial leads placed along the right side of the chest help complete the picture of the right ventricle. Posterior views of the heart can be best seen via posterior leads placed below the scapula to the left of the spine.

Answers: **1.** left and right ventricles (only 40% of inferior myocardial infarctions occur to the right ventricle; the majority of inferior incidents are left ventricular); **2.** lead I - lateral view of the left ventricle; lead III - the inferior surface of the right and left ventricles

The addition of three right-sided precordial leads provides a complete view of the right ventricle. The addition of three posterior leads completes an 18 lead ECG. Note that most 12 lead ECG units do not provide the sixteen electrodes necessary for an 18 lead ECG, or the thirteen electrodes required for a 15 lead ECG. Instead, two separate 12 lead ECGs are taken - the first with standard lead placement and the second with the precordial electrodes repositioned to address the right and posterior aspects of the heart. The extra lead views of a 15 or 18 lead ECG are then labelled accordingly.

Figure 6.3 .Right-Sided Chest Leads to Begin an 18 Lead ECG

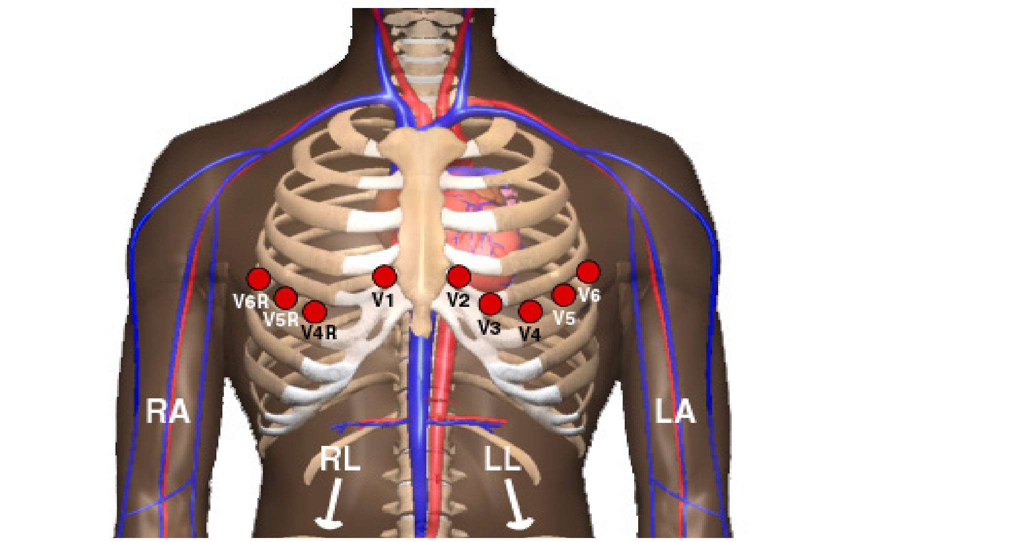


Figure 6.3 illustrates right-sided precordial chest leads. The placement of all three right-sided precordial chest leads (V4R,V5R,V6R) would provide an excellent view of the right ventricle but would not address possible posterior cardiac ischemic changes. Lead V4R is typically used in isolation with a 15 lead ECG because the inferior leads also offer right ventricular coverage. When inferior ischemic changes are seen (leads II, III, aVF), only one lead (V4R) then is needed to satisfy the diagnostic criterion of two leads displaying ST deviation of 1 mm of more in 2 contiguous leads. In other words, if inferior changes are already seen in the inferior leads, then a similar finding in lead V4R confirms right ventricular involvement. As a result, with a 15 lead ECG, leads V5R and V6R are instead placed below the scapula to the left of the spine and labelled V7, V8, or V9 to provide posterior coverage.

For example, to obtain a 15 lead ECG, first take a standard 12 Lead ECG. Then reposition lead V4 to the 5th intercostal space **right mid-clavicular line.** Reposition V5 and V6 along a horizontal plane even with V4R, below the scapula to the left of the spine. Print on this next 12 lead print out, labelling V4 as V4R, V5 as V8, and V6 as V7.

To capture an 18 lead ECG, the second captured 12 lead ECG mirrors the positioning of V4-V6 to the right side (labelled as V4R, V5R, and V6R). Leads V1 - V3 are repositioned to the back just below and between the scapula labelled V7 - V9. Lead V7 (using V1) is positioned level with V6 in the left posterior axillary line. Lead V8 (using V2) is level with V7 placed along the left mid-scapular line. Lead V9 (using V3) is level with leads V8 placed just left of the spine. Each lead is then labelled appropriately.

Is one 12 lead ECG sufficient? Well, yes and no. If the first 12 lead ECG reveals ST elevation in two contiguous leads (leads that view the same region of the heart), then subsequent 12 lead ECGs are used more to monitor the effectiveness of treatment than to make crucial decisions on appropriate treatment. Statistically, one 12 lead ECG will identify an acute myocardial infarction (AMI) for only 50% of those actually experiencing an AMI. Two 12 lead ECGs increase odds of AMI confirmation to 70%. A third 12 lead ECG (12 lead ECGs are ideally taken 15-20 minutes apart) increases AMI confirmation to 90%. Of course, that leaves 10% of those who are having an AMI without the diagnostic benefit of a 12 lead ECG. **Bottom line: one 12 lead ECG is seldom sufficient to rule out an AMI.**

Some centres routinely obtain 15 o 18 lead ECGs while most stick with the 12 lead ECG. For those who routinely rely on the 12 lead ECG, when should additional lead views be taken? A 15 or 18 lead ECG may prove useful with the following findings:

- **inferior lead (II,III,aVF) ischemic or infarct-related changes**
- **tall R waves in lead V1 with a narrow QRS complex** (lead VI usually presents with a deep S wave and very short R wave; tall R wave may signify mirror (reciprocal) view of deep posterior Q waves)
- **ST deviation with symptoms strongly suggestive of cardiac ischemia or infarction** (search for ST elevation in other views)
- **absence of ECG indicators with patient presentation typical of cardiac ischemia or infarction**

Especially in low population settings where percutaneous coronary interventions (PCI) such as angioplasty and arterial stents are not commonplace, within reason keep hunting for the ST elevation. Serial 15 or 18 lead ECGs are obtained to satisfy the criteria for thrombolytics (ST elevation of 1 mm or more in two contiguous leads).

Identifying Ischemia, Injury and Infarction

The ability to identify cardiac ischemia, injury and infarction is vital in the management of the majority of cardiac emergencies. Most sudden cardiac deaths are associated with an ischemic episode. Patient deaths due to an acute myocardial infarction (MI) typically occur within the first 2 hours of symptoms. The 12 lead ECG is a superior diagnostic tool to quickly identify various degrees of ischemia.

“Time is muscle” should prompt an immediate 12 lead ECG for any patient suspected of having cardiac ischemia. Identifying regions of the heart that are ischemic, injured and/or infarcting requires a close inspection for Q waves, ST deviation, dynamic T wave changes and even R wave progression. Refer to Chapter 4 for a review of ECG waveform indicators of cardiac ischemia and infarction. Table 6.5 on page 152 outlines abnormal waveform changes associated with myocardial infarctions.

In the search for signs of cardiac ischemia, identify both the abnormal waveforms as well as the region of the heart that is affected. For example, the presence of abnormal Q waves in leads II, III and aVF is sufficient evidence to confidently claim that the patient has had an inferior MI. Generally, abnormal findings must be present in two contiguous leads before arriving at a diagnosis. Note that a solitary abnormal Q wave is sufficient evidence of a previous MI of indeterminate age (in leads other than aVR).

Table 6.4 Lead Views and Locations of Myocardial Infarctions

Region of Infarction	Leads	Reciprocal Leads
septal	V1, V2	aVL, V7, V8, V9
anterior	(V2), V3, V4	II, III, aVF
lateral	(V4), V5, V6, I, aVL	V1, III
inferior	II, III, aVF, V1	aVL, I
right ventricle	V1, V3R, V4R, V5R, V6R, II, III, aVF, V9	I, aVL, V5, V6
posterior	V7, V8, V9	V1 and V2 (tall R waves), ST depression in V3 and V4

Table 6.4 organizes ECG leads into specific monitored regions of the heart. Note that V2 and V4 are considered transitional leads because they straddle two regions. Reciprocal changes - mirror changes that reflect the opposite surface of the heart - are also included for reference.

ECG indicators for cardiac ischemia and infarction such as prominent Q waves, ST segment deviation and T waves changes were addressed in Chapter 3. Dynamic T wave inversions often result from ischemic zones. The appearance of ST depression is typical of cardiac ischemia, injury and possibly even infarction. The presence of ST elevation of 1 mm or more in two contiguous leads (lead views proximate to each other) is diagnostic of an acute myocardial infarction.

Upon discovery of ischemic indicators, identify the affected region of the heart (see Table 6.4 above). Visually progress through other leads that also monitor the affected region to support your findings i.e. from lead II to leads III and aVF. This is a simple process of putting all the evidence together to complete the clinical picture. A brief exercise would help make all this theory more applicable to your everyday practice.

Practice Exercise 6.1

Figure 6.4 Practise 12 lead ECG for Exercise 6.1

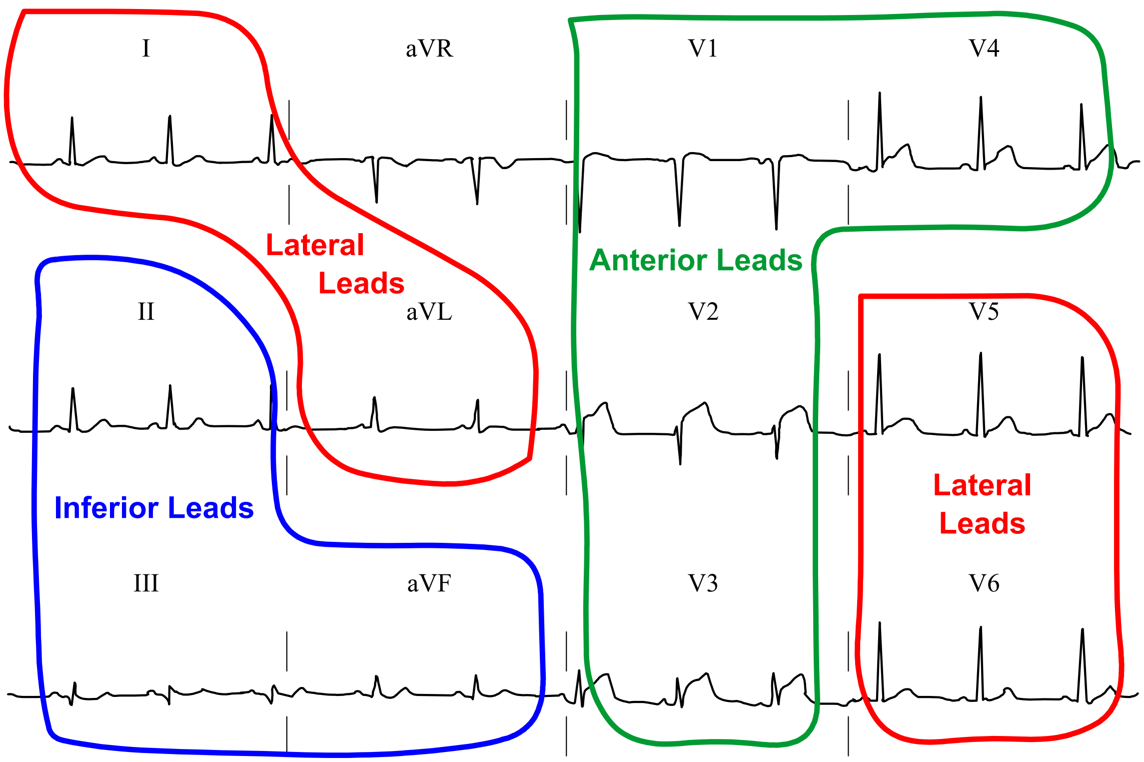


Figure 6.4 illustrates a typical four column layout of a 12 lead ECG. Each column is a 2.5 second recording (making a 12 lead ECG of four columns a 10 second ECG). First note how the lead views created by the four limb electrodes (I,II,III,aVR,aVL,aVF) are located in the first two columns on the left. The precordial chest leads are located in the two columns to the right. Second, note the three regions of the heart viewed by a 12 lead ECG: anterior view (left ventricle), lateral view (left ventricle) and inferior view (left and right ventricle). While the inferior "boot" and other shapes shown here are not normally included in a 12 lead ECG, having a quick reference for lead views helps make sense of the 12 lead ECG.

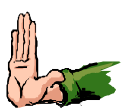
A method to systematically analyse this 12 lead ECG (Figure 6.4) has not been established as yet - this method will be presented shortly. Nevertheless, we already have the skills to make sense of this 12 lead ECG.

1. Looking at lead II, the QRS complexes are narrow with upright P waves. Since the reference grid is omitted here for simplicity, determining heart rate is challenging. But three QRS complexes are present in a 2.5 second period (each column represents 2.5 seconds). Twenty-four 2.5 second intervals make a minute (24 x 2.5 = 60 seconds). The heart rate with this ECG then is approximately 24 x 3 per minute or 72/minute. **This is a sinus rhythm**...simple, and conclusive.

2. Are there any indicators of cardiac ischemia or infarction? Look for prominent Q waves, ST segment deviation, and inverted T waves.

a) Scan quickly for ST segment deviation (most specific indicator for ischemia and infarction). If you typically read from left to right and from top to bottom, lead V1 is probably the first lead that you detect ST deviation - specifically ST elevation. Since lead V1 views the septal (anterior) region of the heart, look to the other septal and anterior leads (V2, V3 and V4). Two contiguous leads (view the same region of the heart) are necessary to diagnose an acute myocardial infarction. Leads V2, V3 and perhaps V4 also present with ST elevation (septal and anterior regions of the heart).

When combined with congruent clinical findings, a diagnosis of an anteroseptal myocardial infarction (MI) is strongly supported. Since the anteroseptal region of the heart involves the left ventricle, it is prudent to continue the investigation into the lateral region that is also occupied by the left ventricle. Is this an anteroseptal MI or the larger anterolateral MI? The lateral lead views (V5,V6,I,aVL) do not present with ST changes.



Pericarditis, an inflammation to the pericardial lining of the heart often attributed to infection, also produces ST elevation on a 12 lead ECG. In fact, because the pericarditis often affects the entire pericardial lining, ST elevation is often seen in most every lead of a 12 lead ECG. The treatment for pericarditis is very different than the treatment for a myocardial infarction. Mixing the two could have grave consequences. The presence of global ST changes (most leads) in a 12 lead should prompt a more complete history and physical assessment.

b) Scan all leads (except aVR - it is usually ignored) **for prominent Q waves and inverted T waves.** While T waves are upright in all leads, prominent Q waves (deep and wide) are present in the inferior leads III and aVF. Since prominent Q waves reflect a previous ST elevation MI of indeterminate age, this evidence supports an inferior MI of indeterminate age.

This is a sinus rhythm with strong evidence for an acute anteroseptal MI and a previous inferior MI of incriminate age.

Treatment often includes aspirin, beta blockers, Plavix and either fibrinolytics or percutaneous coronary interventions (i.e. angioplasty and a stent). Stroke volume of the affected left ventricle could be reduced, with pulmonary congestion possible. Agents to reduce preload may also become useful i.e. morphine, nitroglycerin and diuretics.

Identifying ischemia, injury and infarction with a 12 lead ECG is often straightforward. A ten second electrical picture of the heart may not offer the evidence necessary to decide on treatment. Serial ECGs are standard practice taken every twenty minutes while symptoms persist.

Reciprocal Changes

Picture this. A 12 lead ECG is taken on a patient who is admitted with crushing mid-sternal chest pain. On inspection, the patient is in a sinus tachycardia with a heart rate of 108/minute. five of the anterolateral leads (V3-V6, aVL) show ST elevation by as much as 4 mm. The inferior leads present with ST depression of as much as 4 mm and inverted T waves. Is this patient experiencing an anterolateral MI and further ischemia to the inferior region of the heart? Is it likely to have multiple ischemic regions at the same instant?

No, it is unlikely that two regions are simultaneously ischemic just as it is unlikely that two vessels are occluding at the same time. Instead, the ST depression on this 12 lead is called a reciprocal change. Occasionally, a lead view provides a mirror-like representation for the opposite surface of the heart. For example, ST elevation in anterior leads (V1-V4) may present as reciprocal changes in the posterior leads (opposite surface of the heart) as ST depression and possibly even T wave inversion.

Why hunt for ST elevation? First, myocardial infarctions (MI) are associated with 12 lead findings of ST elevation (55% of all MI), ST depression (35%) and even with normal or non-specific findings (10%). In hospitals without the ability to perform angioplasties and/or to insert stents into narrowed coronary arteries (percutaneous coronary interventions - PCI), the use of fibrinolytics is administered ONLY to patients experiencing an ST elevation MI (STEMI). Patients receiving fibrinolytics have a 25% reduction in morbidity and mortality. Unfortunately, only those experiencing a **STEMI** benefit from fibrinolytics. Fibrinolytics are not administered to those experiencing an MI with normal ECG findings or with ST depression because the risk of stroke associated with the fibrinolytics (about 2%) outweighs the possible advantages. **Hunt for ST elevation to identify those who would greatly benefit from fibrinolytics and save lives for your actions.**

Table 6.4 on page 145 provides a quick reference for expected reciprocal changes. A more apt way to search for reciprocal changes is to conceptualize opposite regions of the heart i.e. anterior and posterior, lateral and inferior. If ST depression is found in lateral leads, ST elevation might be found in the inferior leads (or in the right ventricular leads - V4R - if a 15 lead ECG is obtained).

This brings up an important consideration. Should a 12 lead ECG that only reveals ST depression in two or more leads be followed by a 15/18 lead ECG to hunt for possible ST elevation in mirror leads? Most often the answer is yes. Equipped with the knowledge that reciprocal changes exist and that findings of ST elevation are required to administer fibrinolytics, it is prudent to obtain a 15/18 lead ECG in an effort to search for ST elevation in these alternate lead views.

Possible reciprocal changes include ST depression and T wave inversion. Tall R waves in the anterior leads V1 and V2 (usually deep S waves dominate) may also be reciprocal changes that mirror deep Q waves in the posterior leads. Note that a right bundle branch block also typically presents with upright R waves in leads V1 and V2. The ability to determine bundle branch blocks is covered in the next section. But first, another practice exercise is warranted.

Practice Exercise 6.2

Figure 6.5 Practise 12 lead ECG for Exercise 6.2

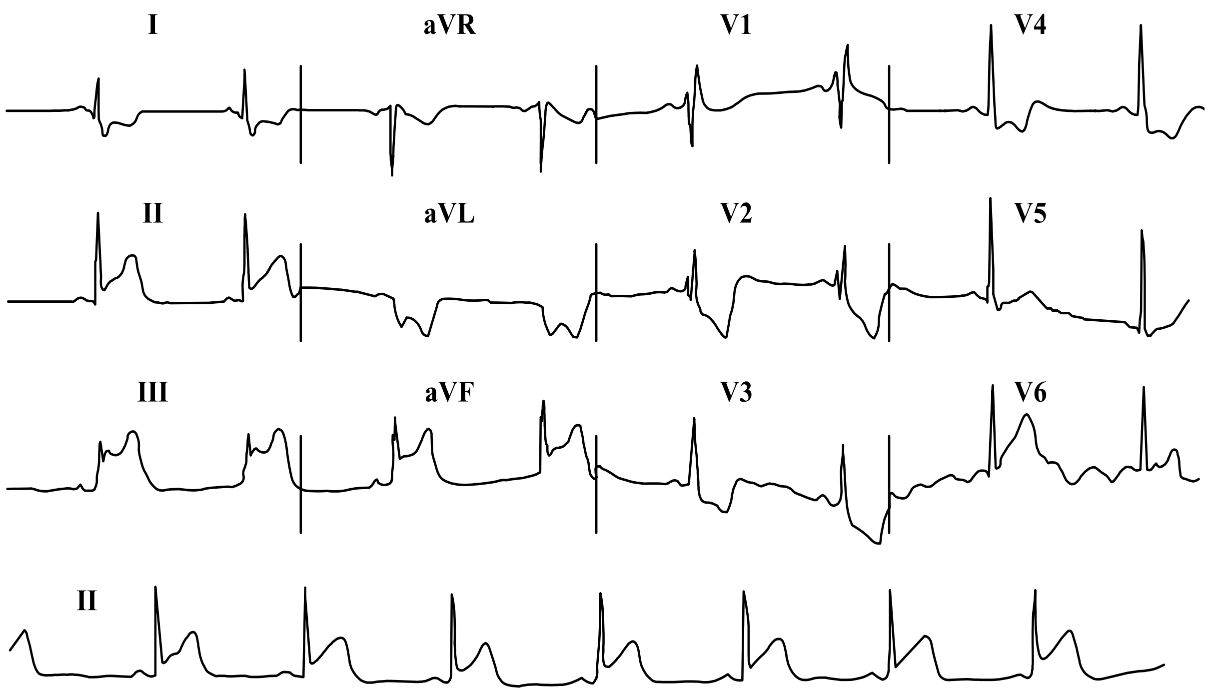


Figure 6.5 is a 12 lead ECG that includes an additional reference lead tracing that persists through the 12 lead tracing. Typically this reference lead tracing is of lead II or V1.

Let's use the knowledge and skills formed thus far to make sense of this 12 lead ECG.

1. Looking at the reference lead II provided at the bottom of this 12 lead ECG, the QRS complexes are narrow with upright P waves and ST elevation. Only two QRS complexes are seen in any of the bipolar leads of the first column. Using the same formula as exercise 6.1, the heart rate is approximately 48/minute (24 x 2 = 48). **This is a sinus bradycardia with ST elevation**...again simple, and conclusive.

2. Are there any indicators of cardiac ischemia or infarction? Look for prominent Q waves, ST segment deviation, and inverted T waves.

a) Scan quickly for ST segment deviation. Well ST elevation has already been established in lead II. Since lead II is an inferior lead, progress to the other inferior leads (III, aVF). ST elevation is present in all the inferior leads, strongly supporting the diagnosis of an **acute inferior myocardial infarction**. If this patient does not have any contraindications (i.e. recent surgery, active bleeding, previous stroke), fibrinolytics are warranted.

But hold on. The presence of ST elevation in most leads can point to the presence of pericarditis - not myocardial infarction. Administering fibrinolytics to a patient experiencing pericarditis can have disastrous effects. It is prudent to examine all the leads to rule out this possibility. Of course, examining all the leads (with the exception of aVR) will often yield a more comprehensive electrical picture.

On closer examination, the anterior leads and some of the lateral leads provide reciprocal changes of ST depression (leads V1-V4, I and aVL). These are indeed reciprocal changes rather than further signs of another ischemic region of the heart.

If you recognized upright R waves in leads V1 and V2, these findings are likely attributed to a right bundle branch block. This topic will explored in the next section.

b) Scan all leads (except aVR - it is usually ignored) **for prominent Q waves and inverted T waves.** While inverted T waves are present, the lead views are probably providing reciprocal changes (anterior and some lateral leads). Prominent Q waves (deep and wide) are not present.

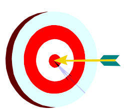
This is a sinus bradycardia with strong evidence for an acute inferior MI.

Since an inferior MI could involve either the left or right ventricle (seldom both), a 15/18 lead ECG is warranted to help differentiate. The evidence of ST evidence in V4R, for example, would clarify the diagnosis of a right ventricular infarction. Reflecting back to Chapter 2, hemodynamic management of a right-sided MI is quite distinct from a left-sided MI. Overabundant preload is common with a left-sided MI (treat with nitroglycerin and morphine for example) whereas insufficient preload is often an issue with a right-sided MI (treat with a fluid bolus if blood pressure is low).

Bundle Branch Blocks

Normal ventricular depolarization begins with the septal fascicle of the left bundle branch (causing a Q wave) followed by a simultaneous depolarization of the remaining ventricular walls via the right and left bundle branches. The left bundle branch splits into the septal, anterior and posterior fascicles. A damaged conduction system can lead to the blockage of any or all of these bundle branches (or fascicles).

A bundle branch block reduces the speed by which the ventricles depolarize, resulting in a wide QRS complex (>.12 seconds or 3 mm). Supraventricular rhythms with a bundle branch block (with its wide QRS complex) can appear to be ventricular rhythms, especially for rapid rhythms where P waves are difficult to identify. Fortunately, left bundle branch block (LBBB) and right bundle branch block (RBBB) are easily determined with a 12 lead ECG.



An incomplete block of the anterior or posterior fascicle of the left bundle branch is called a hemiblock. A hemiblock has a normal QRS duration of less than 0.12 seconds (unless a RBBB coexists). Left anterior hemiblock (LAHB) is diagnosed if the net QRS deflection in lead II is negative (deeper S wave than height of R wave). About 98% of all hemiblocks are anterior hemiblocks.

Both bundle branch blocks cause the ventricles to depolarize out of sync. The ventricle with the intact bundle branch depolarizes before its counterpart. As a result, two R waves form, an R wave and R prime resulting in a notch in the QRS complex.

Figure 6.6 RBBB versus LBBB

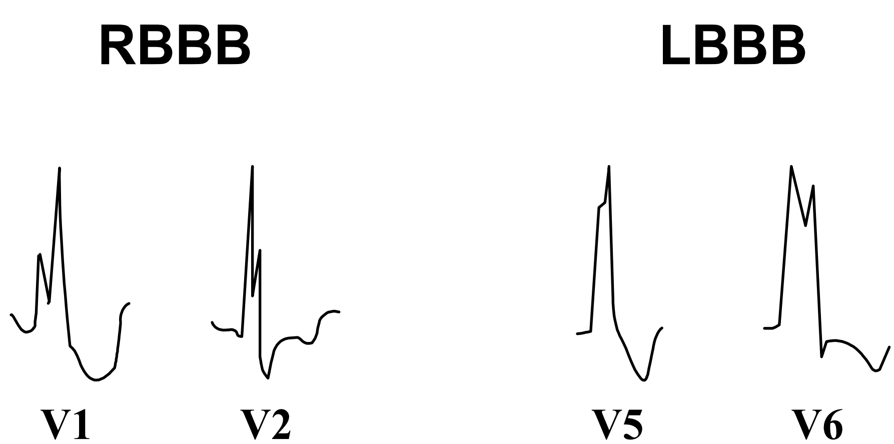


Figure 6.6 provides a quick reference for right and left bundle branch block.

A bundle branch block must satisfy two criteria - a wide QRS complex and a notch in the QRS complex. To distinguish between a RBBB and a LBBB, first make certain that the rhythm is indeed supraventricular (P waves before each QRS) and that the QRS complex is wide (at least 0.12 seconds in duration).

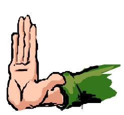
A left bundle branch block (LBBB) is best distinguished using leads V5 or V6 due to their close proximity to the left ventricle. If a supraventricular rhythm has wide and notched QRS complexes in leads V5 and/or V6, then a LBBB is evident. Similarly, a supraventricular rhythm with a right bundle branch block (RBBB) is diagnosed with a wide and notched QRS complex in leads V1 and V2 (closest to the right ventricle).

Occasionally, the QRS complex meets only one of the criteria (i.e. wide but not notched or notched but not wide). If not a hemiblock (see caption on previous page), this is commonly called an **intraventricular conduction delay**.

Both left and right bundle branch blocks are commonly associated with ST depression and T wave inversion (with or without ischemia). This makes the identification of cardiac ischemia difficult. In addition, a LBBB often presents with ST elevation in leads V1-V3 (and other leads), making the identification of an acute MI almost impossible in the presence of a LBBB. With clinical symptoms congruent with an MI, the appearance of **a new onset LBBB is considered equivalent to a STEMI.**

The Systematic Analysis of a 12 Lead ECG

The previous sections - many would claim the entire book to this point - have laid the foundation for a systematic approach to make sense of the 12 lead ECG. Equipped with a carefully formed clinical impression of the patient, a systematic approach to 12 lead ECG interpretation makes the 12 lead ECG a reliable assessment tool. Mindful of false positives and false negatives, the patient's old 12 lead ECG serves as an invaluable reference that greatly increases the likelihood of a correct interpretation.



While an ECG is a superior diagnostic tool, experienced practitioners are witness to false positive and false negative ECGs. A **false positive ECG** occurs when ECG findings are abnormal for a patient that is quite healthy. An example of a false positive is the presence of ST elevation that is a benign **early repolarization** - more common with young athletes. Conversely, a **false negative ECG** does not indicate the presence of cardiac disease that is subsequently established. The bottom line: a 12 lead ECG is a tool best used in conjunction with previous ECGs, a thorough physical assessment and an in depth cardiac history.

The systematic ECG interpretation of a 12 lead ECG takes a bit more time than the six seconds necessary to correctly name most single lead rhythm strips. While many excellent approaches to 12 lead interpretation exist (please refer to the end of this chapter for a brief list of resources), all methods include certain key points that are incorporated into a four-step system for 12 lead ECG analysis. Deja vu?

1. Begin with the four-step method of ECG interpretation to identify the rhythm.

Using lead II, is the rhythm too fast or too slow? If so, check the patient for poor cardiac output. Are the QRS complexes wide or narrow? Check the P waves. If the ECG rhythm is irregular, identify the causes of the irregular pattern.

2. Hunt for indicators of cardiac ischemia and infarction.

Q - prominent Q waves (25% the height of the R wave and/or 1 mm in width)
ST segment deviation (of 1 mm or more in two leads with similar lead views)
T wave changes such as T wave inversion, peaked T waves and biphasic T waves

Once you find an ischemic indicator in a single lead view, immediately complete the picture by proceeding to other leads that share the same lead view. For example, if lead I shows ST elevation, proceed visually to leads aVL, V5 and V6 - all lateral leads.

Distinguish between signs of infarction and reciprocal ECG changes.

3. Determine if a right or left bundle branch block is present.

The presence of a new left bundle branch block accompanied by symptoms of an acute myocardial infarction is diagnostic. Both left and right bundle branch blocks also change the morphology (shape) of the ECG to resemble ischemic changes with **and without** the presence of cardiac ischemia. Again, an old ECG is quite useful here.

For example, the symptoms typical of cardiac ischemia are also typical of a long list of medical conditions such as cholecystitis and gastroesophageal reflux disease. Do not be fooled into moving down the cardiac ischemia road to the complete exclusion of other possibilities. Do not allow a chronic left or right bundle branch block together with their accompanying ST elevation or depression move you into a one-dimensional treatment plan. Rule out the presence of bundle branch blocks.

For most clinicians, a generally reliable interpretation can be provided after only these first three steps.

4. Revisit the waveforms, mapping normal and abnormal findings to regions of the heart in a systematic fashion, all the while evaluating:

P waves, looking for large and notched morphologies (i.e. atrial hypertrophy)
Q waves - normal or abnormal
R and **S** waves for amplitude (also to determine R wave progression and electrical axis)
ST segment deviation (possible ventricular strain or digoxin dip)
T wave changes for signs of electrolyte imbalance
U wave may suggest electrolyte imbalance

Note the easy to remember **PQRSTU** progression to complete your descriptive analysis.By completing your 12 lead ECG systematically, more subtle findings are rarely missed.

Table 6.5 Normal and Abnormal Characteristics of ECG Waveforms

Waveforms	Normal Characteristics	Abnormal Characteristics (AC)	Possible Cardiac Conditions with AC
P wave	rounded; 0.5-2.5 mm in height; <0.11 seconds in duration; upright in leads I and II	peaked, wide or notched; height >2.5 mm flat	atrial hypertrophy; congestive heart failure; COPD; valvular disease hyperkalemia and hypomagnesemia
Q wave	normal in lead aVR; <0.04 seconds in duration; less than 25% the height of the R wave	>0.04 seconds in duration; 25% the height of the R wave or more	evolved myocardial infarction (MI)
R wave progression	progressive increase in the amplitude and dominance of the R wave (larger than S wave) in leads V3 or V4	large R waves in lead V1 and V2 late R wave progression to leads V5 or V6	acute posterior or high lateral (MI) acute anterior myocardial infarction
ST segments	J point level with or less than 1 mm deviated from the isoelectric line	> 1mm deviation from the isoelectric line measured 0.04 seconds after the J point	cardiac ischemia, injury or infarction; ventricular hypertrophy; digoxin

Waveforms	Normal Characteristics	Abnormal Characteristics (AC)	Possible Cardiac Conditions with AC
T waves	normally inverted in lead aVR; slightly asymmetrical; 5 mm in amplitude or less in leads I, II, and III	inverted T waves peaked T waves flat	myocardial ischemia; vent. hypertrophy early cardiac ischemia; hyperkalemia hypomagnesemia
U waves	rounded; <2 mm in amplitude; smaller than the T wave	> 2 mm in amplitude	hypokalemia; hypomagnesemia

Table 6.5 provides a non-exhaustive list of cardiac conditions that are associated with a variety of abnormal ECG waveforms. Arriving at a useful interpretation of a 12 lead ECG requires more diligence than the rapid interpretation of a six second rhythm strip. In many ways, you become a detective, synthesizing data from both the ECG and the patient to come to a more complete understanding.

Only for those with a keen interest in cardiology and a generous volume of 12-lead ECGs, the fourth step can alert you to (**but not confirm**) potential cardiac disease. This last step, while not diagnostic, can yield valuable clues when combined with a well established history. Further tests must be ordered to confirm these concerns.

The successful application of the first three steps is a solid foundation for the identification of cardiac ischemia and infarction - the most common use of a 12 lead ECG. While being redundant, it again is worth mention: for many, if not most practitioners, the fourth step is unnecessary. Begin to utilize step four only after the first three steps become a well established skill set.

The availability of previous ECGs, a thorough clinical assessment, and an established patient history are all necessary for a definitive 12 lead ECG interpretation. The moment is ideal to put the first three steps of 12 lead ECG interpretation into practice.

Flash Quiz 6.1

Case Study 6.1: A 68 year old man presents via ambulance with persistent shoulder, neck and jaw pain that began about one hour previous after physical exertion. He is placed in a monitored bed. Vital signs are taken including an oxygen saturation. Oxygen is then administered via nasal prongs at 2 litres/minute. An intravenous drip is initiated at a rate to keep the vein open. Meanwhile a 12 lead ECG is obtained. The first 12 lead ECG is normal. Repeat 12 lead ECGs are ordered.

1. With persistent symptoms, at least three 12 lead ECGs should be taken:

a) 20 minutes apart
b) one hour apart
c) 3 hours apart
d) 6 hours apart

2. Serial 12 lead ECGs are ordered because a single 12 lead ECG confirms only:

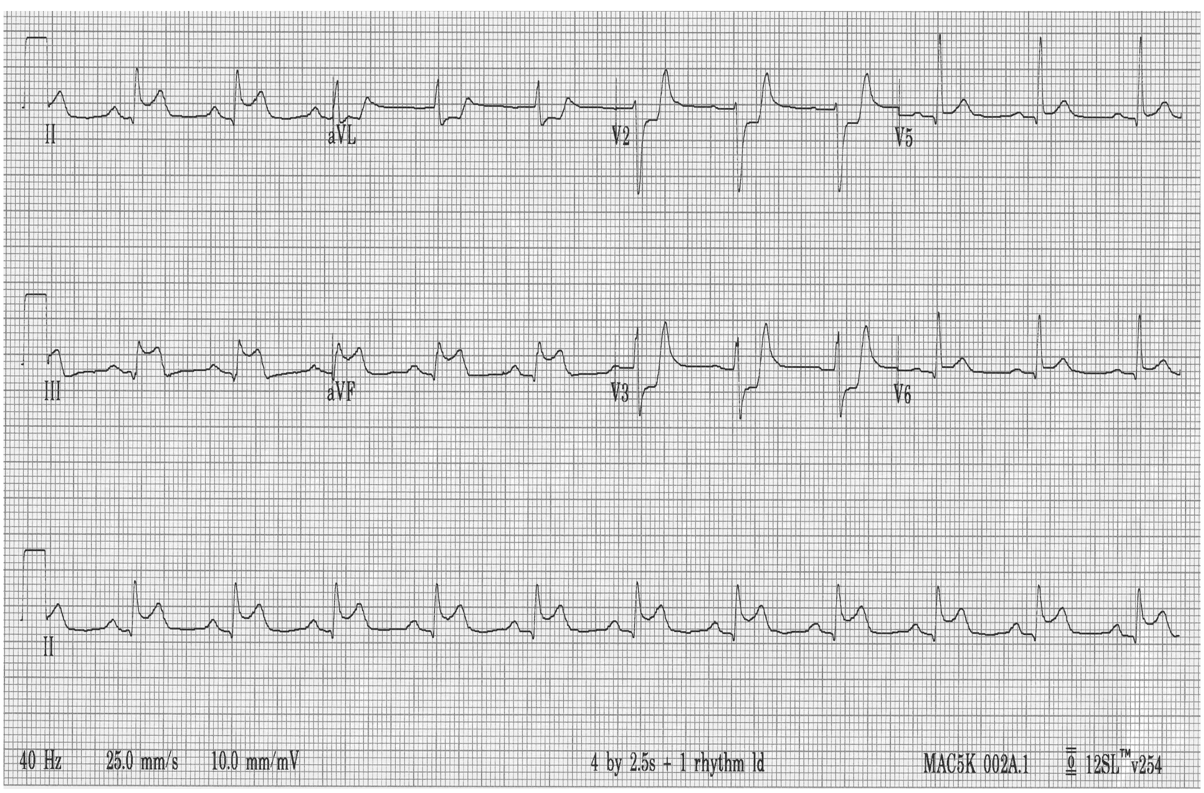
a) 50% of myocardial infarctions
b) 70% of myocardial infarctions
c) 80% of myocardial infarctions
d) 90% of myocardial infarctions

3. A finding of ST depression always suggests cardiac ischemia and not infarction.

True or False

The second 12 lead ECG is presented in Figure 6.7.

Figure 6.7 Second ECG in Series for Case Study 6.1



Answers: **1.** a) **2.** a); **3.** False;

4. The ECG rhythm in lead II shown in the 12 lead ECG in Figure 6.7 is:

a) sinus bradycardia with ST depression
b) sinus rhythm with ST elevation
c) sinus rhythm
d) sinus tachycardia with ST elevation

5. Significant elevations in the ST segment are present in the:

a) inferior leads
b) septal leads
c) anterior leads
d) lateral leads

6. Significant depression of the ST segment are NOT present in the:

a) inferior leads
b) septal leads
c) anterior leads
d) lateral leads

7. Prominent Q waves are not seen in this 12 lead ECG.

True or False

8. After inspecting the ECG waveforms of V1, V2, V5, and V6, the following is found:

a) right bundle branch block
b) left bundle branch block
c) first degree AV block
d) normal ventricular conduction

9. The correct interpretation for this 12 lead ECG is:

a) sinus rhythm with signs that support a diagnosis of an acute anterior MI
b) sinus rhythm with signs that support a diagnosis of an acute inferior MI
c) sinus rhythm with signs that support a diagnosis of acute anterolateral ischemia
d) sinus rhythm with signs that support a diagnosis of unstable angina

Answers: **4.** b); **5.** a); **6.** a); **7.** True; **8.** d); **9.**b); **10.** a) and b);

10. If this is an inferior myocardial infarction, the advantages of a 15 or 18 lead ECG are (circle all that apply):

a) to differentiate between a left and right ventricular infarction
b) to establish whether to be cautious in the administration of nitroglycerin
c) to meet the criteria for the administration of fibrinolytics
d) to determine if the patient should receive aspirin

This ends the questions that relate to Case Study 6.1.

11. A 12 lead ECG is obtained from a patient presenting with sudden onset mid-sternal chest pain. After inspecting a 12 lead ECG, you collect the following findings: ST elevation in leads I, aVL, V3-V6, prominent Q waves in leads II and III. Left and right bundle branch blocks are ruled out. Your interpretation is;

a) signs of anterolateral ischemia with evidence of an inferior MI of indeterminate age
b) acute anterior STEMI with evidence of an inferior MI of indeterminate age
c) acute anterolateral STEMI with evidence of an inferior MI of indeterminate age
d) acute anterolateral NSTEMI with evidence of an anterior MI of indeterminate age

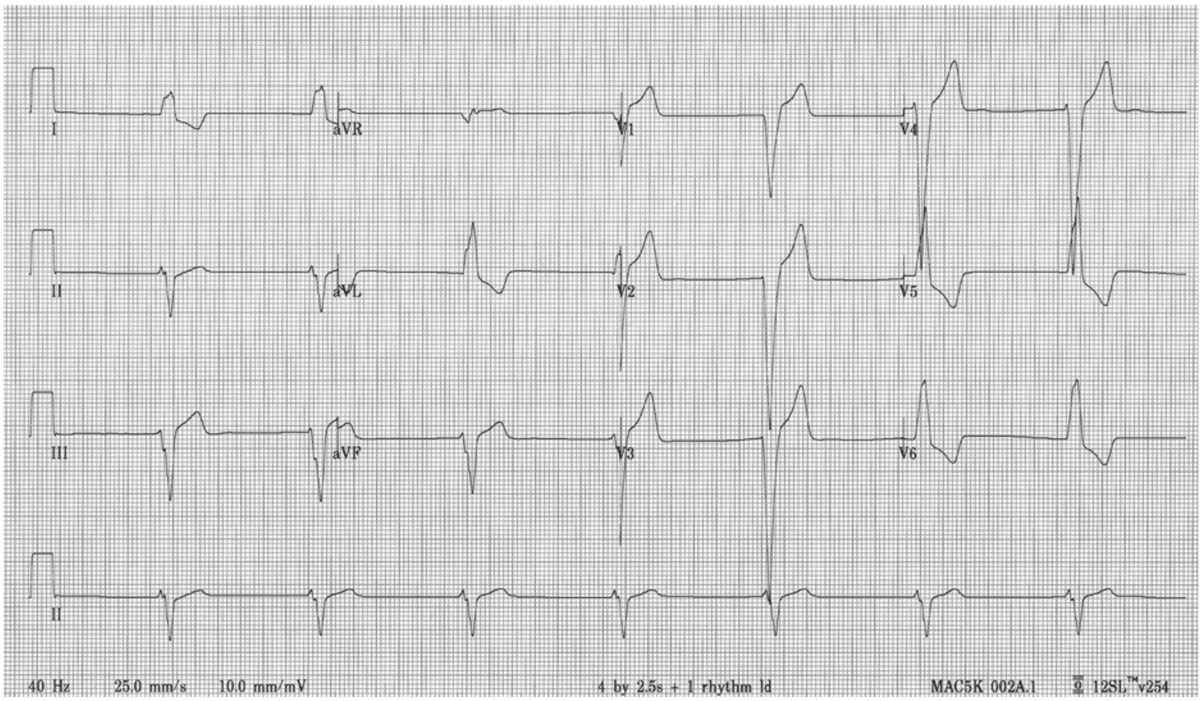
12. A 12 lead ECG shows ST elevation of 2 mm with most all leads. This strongly suggests (a global myocardial infarction, pericarditis, anterior myocardial infarction).

13. Myocardial infarctions are always identified with either ST elevation or depression.

True or False

Answers: **11.** c); **12.** pericarditis; **13.** False;

Figure 6.8



Questions 14-18 refer to Figure 6.8.

14. The ECG rhythm in lead II shown in this 12 lead ECG is:

a) sinus bradycardia
b) junctional rhythm
c) sinus rhythm
d) junctional rhythm with aberrant conduction

15. Significant elevations in the ST segment are present in the:

a) inferior leads
b) septal leads
c) anterior leads
d) lateral leads

16. Significant depression of the ST segment are present in the:

a) inferior leads
b) septal leads
c) anterior leads
d) lateral leads

Answers: **14.** d); **15.** c); **16.** d);

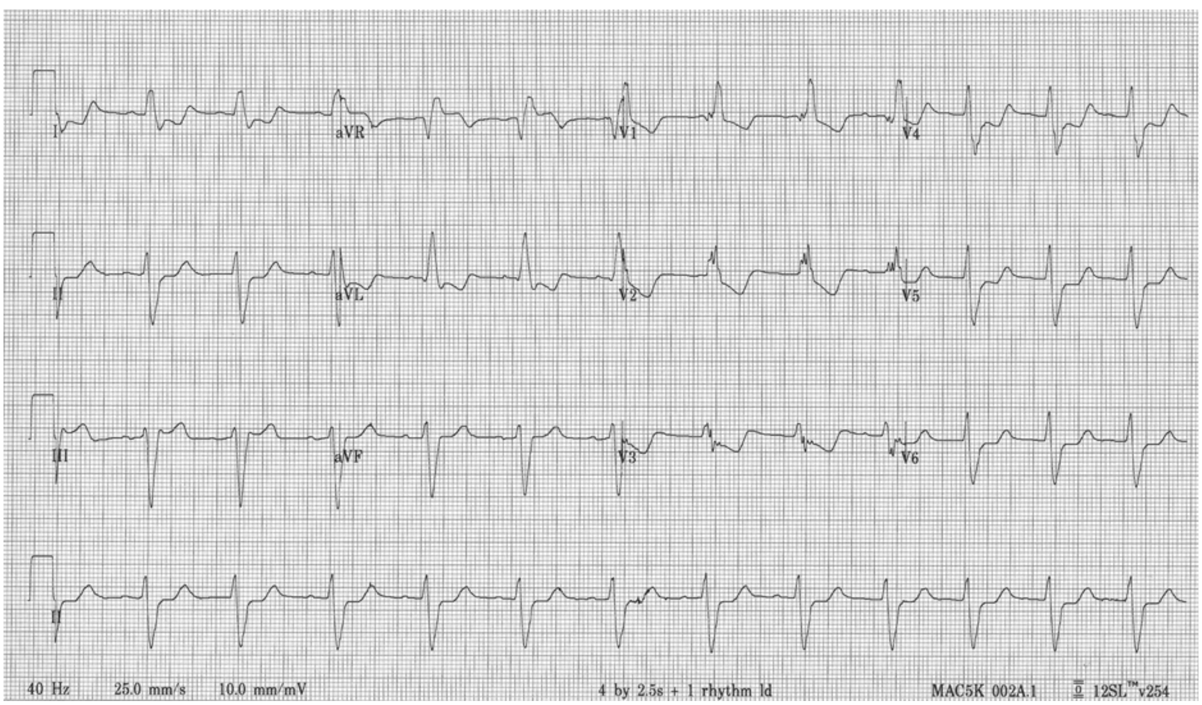
17. After inspection of leads V1, V2, V5 and V6, the following is established:

a) right bundle branch block
b) left bundle branch block
c) an anterior MI
d) lateral ischemia

18. The changes seen in this 12 lead ECG are more typical of a left bundle branch block than an anterolateral MI.

True or False

Figure 6.9



Questions 19-20 refer to Figure 6.9 on the previous page.

19. The rhythm represented in this 12 lead ECG is:

a) sinus rhythm with aberrant conduction and ST depression
b) idioventricular rhythm
c) sinus rhythm with ST depression
d) junctional rhythm with aberrant conduction

Answers: **17.** b); **18.** True; **19.** a);

20. Your interpretation of this 12 lead ECG includes (circle all that applies):

a) right bundle branch block
b) left bundle branch block
c) global ischemia
d) pericarditis

Identifying the QRS Axis*

Many novice practitioners of 12 lead ECG interpretation find the next two topics - identifying the QRS axis and assessing for R wave progression - challenging and somewhat confusing. Fortunately, these skills only rarely impact the acute management of a patient in a cardiac crisis. Hence, the next two topics are considered optional skill sets in the systematic analysis of a 12 lead ECG. If you are keen to delve into these non-essential ECG realms, the next two topics are definitely within reach.

Background

If the identification of the QRS axis is often considered a non-essential skill, is there any merit to establishing a QRS axis? The 12 lead ECG becomes a more robust assessment tool when QRS axis is utilized in a systematic analysis. A QRS axis can help identify ventricular and atrial hypertrophy, correlate QRS deviation with the extent of a myocardial infarction and even help differentiate between supraventricular tachycardia with aberrant conduction and ventricular tachycardia. A QRS axis has merit.

Let's begin the discussion of QRS axis with a brief review of the QRS complex.

Revisiting the QRS Complex

The QRS complex provides a surprising amount of information about ventricular depolarization: the speed of depolarization, the electrical force or voltage involved and the direction of the depolarizing wave across the ventricles.

The QRS complex is produced by ventricular depolarization. The width of the QRS is a function of the time taken for the ventricles to depolarize. The height or amplitude of the QRS is a function of the electrical force or voltage of the monitored region of the

Answers: **20.** a)

* this concept is considered optional in the systematic analysis of a 12 lead ECG

heart. The orientation of the QRS complex (i.e. upright, downward or diphasic waveform) is a function of whether the depolarizing wave is directed towards or away from the positive electrode of each lead.

With lead II, the positive electrode resides close to the apex of the heart. Since the depolarizing wave moves towards this positive red electrode, the resulting QRS complex will also be positive (upright). Conversely, with a positive electrode located on the right shoulder as in lead aVR, the QRS complex would be inverted since the ventricles typically depolarize away from the right shoulder.

Figure 6.10 Calculating Net Deflection of the QRS Complex

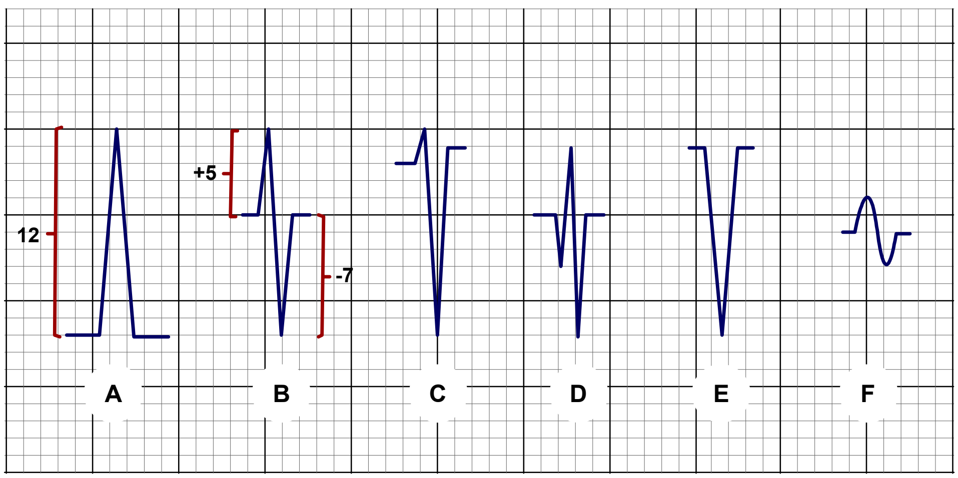


Figure 6.10 outlines the steps needed to determine whether a QRS complex is upright, inverted or neutral. For QRS complexes such as 'A' and 'E', the answer is straightforward. For the other QRS complexes (B-D, F), the net deflection of the QRS complex must be calculated. Take QRS complex 'B'. The R wave is 5 mm in height and the S wave is 7 mm in depth leaving a net deflection of +5-7 or -2. The QRS complex 'B' has a negative net deflection of -2.

While often a quick look is sufficient to determine whether the QRS complex is upright or inverted, occasionally a QRS complex requires a simple calculation to arrive at a net deflection. Figure 6.10 outlines the steps required to arrive at a net deflection. The orientation (up or down) of the QRS complex 'D' is not easily established. The net deflection would equal the sum of the separate three deflections: Q wave of -3 mm, an R wave of +4 mm and an S wave of -7 mm = net deflection of -6 mm. Calculating net QRS deflection is a well-utilized exercise in identifying the QRS axis.

Net Deflection in Figure 6.10: **A.** 12 mm; **B.** -2 mm; **C.** -8 mm; **D.** -6 mm; **E.** -12 mm; **F.** 0 mm

The Mean Vector and the QRS Axis

It is time to introduce the term **vector**. A vector is an arrow that represents the size and direction of a force. The larger the force the larger the arrow. Now take the small vectors from Figure 6.11 that represent the direction and size of the electrical force of ventricular depolarization. When adding these small vectors, the resulting size and direction of the overall ventricular depolarization could be represented by the large arrow. The **direction of an average (mean) vector is called the electrical axis**. The overall direction of ventricular depolarization is called the **QRS axis**.

Figure 6.11 Vectors and Ventricular Depolarization

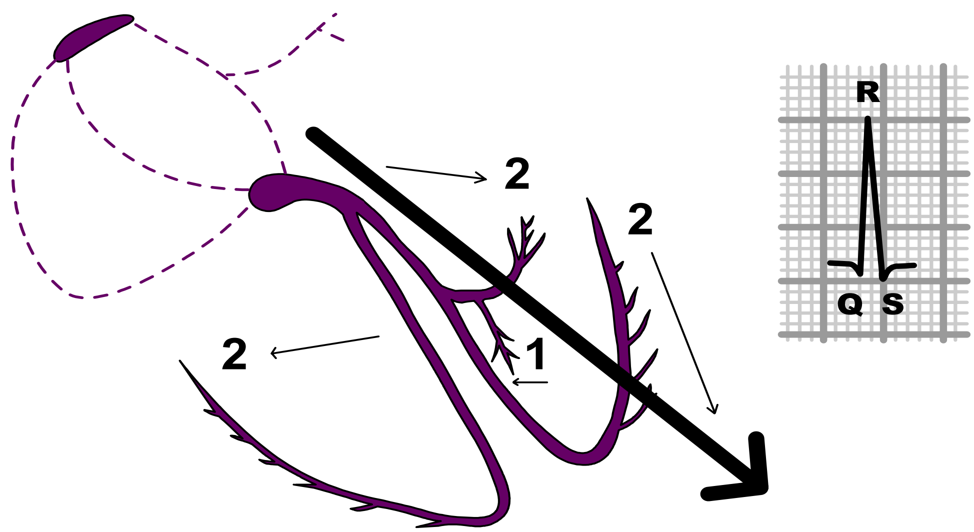
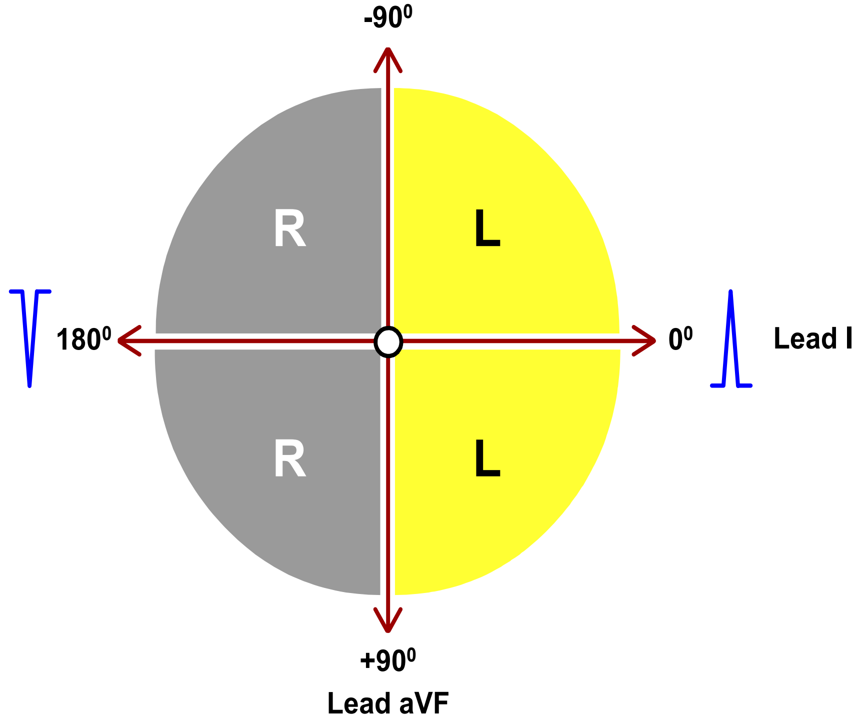


Figure 6.11 illustrates the direction of normal depolarization of the ventricles from the AV node out to the apex of the heart. Strictly speaking, each of the four depolarizing waves move in different directions. The septal fascicle of the left bundle branch depolarizes the septal region from the left septum towards the right ventricle (#1), away from the positive electrode in lead II. This early depolarizing wave away from the positive electrode produces a small Q wave. Both ventricles then simultaneously depolarize (#2). While the right ventricle depolarizes away from the positive electrode (in lead II), the electrical current of the much thicker left ventricular myocardium dominates the overall direction of ventricular depolarization thus producing an upward QRS complex.

With an electrocardiogram, the mean vector (QRS axis) of ventricular depolarization is best identified by comparing various QRS complexes. The lead views taken from the limb electrodes (I, II, III, aVR, aVL, aVF) are particularly useful since together these six leads provide a six point mapping of the frontal plane (see Figure 6.16).

Figure 6.12 Patient's Left and Right QRS Axis

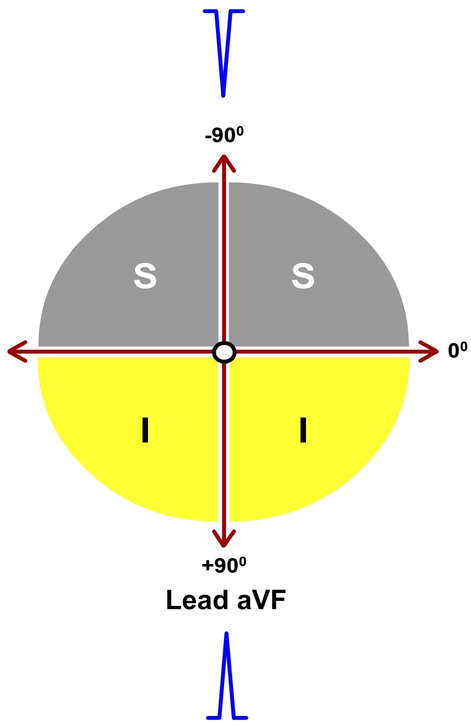


Lead I effectively identifies whether the QRS axis is pointing to the patient's left (L-lighter shade) with an upward QRS complex or pointing to the patient's right (R-darker shade) and away from the positive electrode in lead I resulting in an inverted QRS complex.The tail of a QRS axis is the AV node (effectively the centre of the heart).

Perhaps most useful are leads I and lead aVF because these leads divide the frontal plane into four quadrants (see Figure 6.12). Lead I, the bipolar lead between the right and left shoulder with the left shoulder a positive electrode, provides a horizontal reference point of 0^0 (zero degrees). With the AV node serving as the tail of the mean QRS axis, a QRS complex in lead I effectively divides the QRS axis that is pointing to the patient's left from a QRS axis facing to the patient's right (see Figure 6.11).

Lead aVF, with its positive electrode at the feet, is perpendicular to lead I, forming an angle of $+90^0$ (90 degrees). A QRS complex in lead aVF effectively divides the QRS axis that is pointing inferior from a QRS axis facing superior (Figure 6.13). An upright QRS complex in lead aVR signifies a downward facing QRS axis. An inverted QRS complex signifies that the QRS axis is moving away from the feet, facing superiorly.

Figure 6.13 Inferior and Superior QRS Axis



Lead aVF effectively identifies whether the QRS axis is pointing down or inferior (I-lighter shade) with an upward QRS complex or pointing up or superior (S-darker shade) and away from the positive electrode in lead aVF resulting in an inverted QRS complex.

Identifying QRS Axis Deviation

A normal QRS axis is directed towards the inferior left quadrant formed by leads I and aVF ($0\text{-}90^0$). This is illustrated in Figure 6.14 on the next page. Using leads I and aVF, a normal QRS axis can be quickly identified. If the QRS complex in lead I is generally upward and the QRS complex in lead aVF is generally upward, then the QRS axis must be directed into the patient's left inferior quadrant.

Most clinicians are quite satisfied to find the QRS axis in the normal left inferior quadrant. The 'rub' though is with a deviated QRS axis. Left or right QRS axis deviation provides evidence for such conditions as chamber hypertrophy and myocardial infarction. Fortunately, identifying QRS deviation is as simple as identifying a normal QRS axis.

Figure 6.14 The Quadrant of a Normal QRS Axis

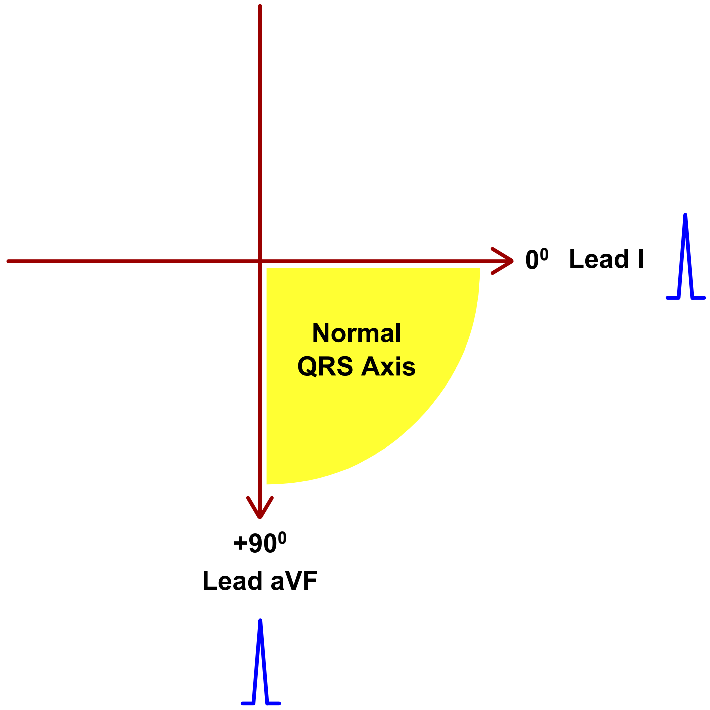


Figure 6.13 depicts the expected quadrant for a normal QRS axis. The QRS axis is normally directed down and to the left. Note that if the QRS complexes of both leads I and aVF are upright, then ventricular depolarization must be directed both towards the feet (aVF) and towards the patient's left (lead I).

Left QRS axis deviation occurs with the QRS axis facing the patient's upper left quadrant. Conversely, right QRS axis deviation points the QRS in the bottom right quadrant (see Figure 6.15). Identification of an abnormal QRS axis involves the same steps as used to identify a normal QRS axis. Essentially, check the QRS complexes in leads I and aVF and plot out the direction of the depolarizing wave (QRS axis).

As a general rule, a QRS axis often shifts away from the an infarcted area. A QRS axis moves towards chamber hypertrophy i.e. left ventricular hypertrophy.

Example: A patient with a long history of hypertension has a 12 lead ECG with a positive (upward) QRS complex in lead I and a negative (downward) QRS complex in lead aVF. Identify the QRS axis.

Step 1: A positive QRS in lead I is associated with a leftward pointing QRS axis (depolarizing toward the positive left shoulder electrode of lead I). The QRS axis is directed toward the left quadrants. Whether the QRS is directed toward the upper left or the lower left quadrant is determined by the lead aVF.

Step 2: A negative QRS complex in lead aVF is associated with a QRS axis that is facing away from the feet, towards the upper quadrants. The QRS axis then is directed towards the upper left quinacrine. This is called left QRS axis deviation.

Figure 6.15 The Quinacrine of Right and Left QRS Axis Deviation

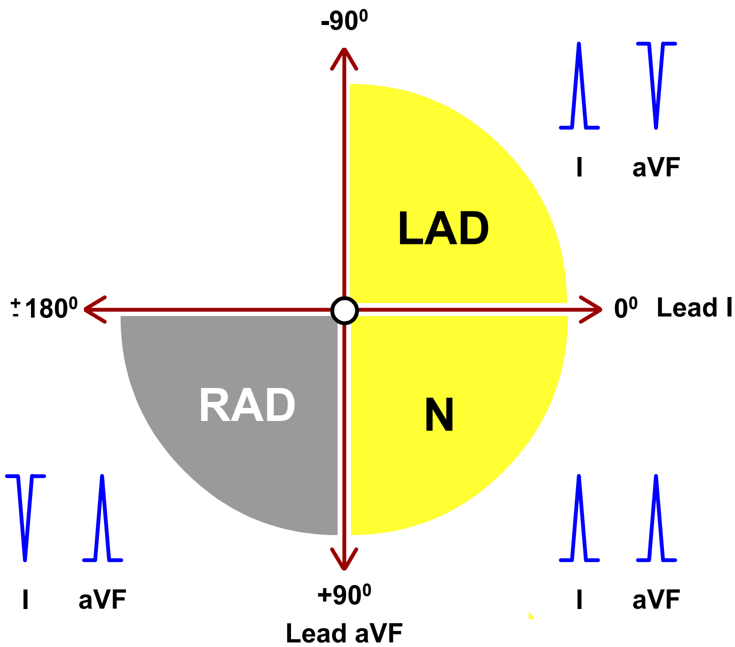


Figure 6.15 labels the abnormal quadrants of left axis deviation (LAD) and right axis deviation (RAD). The orientation of the QRS axis (i.e. positive or negative) for leads I and aVF are provided for reference. Note that the unlabelled quadrant between -90^0 and +/- 180^0 is commonly referred to as 'No Man's Land' or the 'Northwest Quinacrine'. A QRS axis directed at this quadrant is an indeterminate axis.

How is left QRS axis deviation related to a long history of hypertension? In Chapter 2, chronically high afterload was one explanation for left ventricular hypertrophy. Earlier in this section, the dominance of the large left ventricle was the rationale for a normal left facing QRS axis. But what if the left ventricle became even larger, dominating the right ventricle further? The QRS axis would deviate more to the left. A finding of left QRS axis deviation is associated with left ventricular hypertrophy.

Flash Quiz 6.2

A little practice should bring this skill home.

1. A 68-year-old man on the medical ward has chronic emphysema. A routine 12 lead ECG reveals an inverted QRS complex in lead I and an upright QRS complex in lead aVF. What is the QRS axis? Does this QRS axis fit the medical diagnosis?

2. Two day post-anterolateral myocardial infarction, a patient's 12 lead ECG shows a right QRS axis deviation. Does this make sense?

3. A 12 lead ECG shows an upright QRS complex in lead I and an inverted QRS complex in lead aVF. The QRS axis supports a suspected (left ventricular infarction, right ventricular infarction).

4. A 72-year-old patient with congestive heart failure is on fluid restrictions and diuretics among a host of other cardiac medications. A routine 12 lead ECG shows an upright QRS complex in leads I and aVF. Is the QRS axis typical of congestive heart failure?

Answers

1. The QRS axis is directed to the right lower quadrant, signifying right axis deviation. Chronic emphysema, a form of chronic obstructive lung disease (COPD), can cause pulmonary hypertension and a resulting right ventricular hypertrophy. Right ventricular hypertrophy 'pulls' the QRS axis to the right.

2. An anterolateral myocardial infarction results in the loss of myocardial tissue from the left ventricle. If the loss of left ventricular tissue is substantial, the left ventricle can lose some of the dominance normally held with the QRS axis. The QRS axis could potentially deviate to the right lower quadrant.

3. The QRS axis is directed to the patient's upper left quadrant - left axis deviation. The loss of right ventricular muscle (right ventricular myocardial infarction) makes the left ventricle more dominant 'pulling' the QRS axis into the upper left quadrant.

4. The QRS axis faces the normal lower left quadrant. The QRS axis of patients with cardiac disease is commonly directed towards the normal lower left quadrant. The presence of cardiac disease may shift the QRS axis without deviating the axis out of the normal quadrant.

The QRS Axis Using All Leads of the Frontal Plane

The ability to be more specific with QRS axis deviation requires a quick scan of all the limb leads (I, II, III, aVR, aVL, aVF). Using these standard and augmented voltage leads, a closer approximation can be made of the QRS axis allowing for the identification of shifts in the QRS axis within each quadrant.

A more specific QRS axis is established with a simple two step process. The six leads created by the four limb electrodes are ideal when calculating the QRS axis since these leads (aVR, aVL, aVF, I, II, III) provide six directions within the frontal plane. The six surface leads provide a full 360 degree reference system, separated by 30 degree intervals (see Figure 6.16).

By studying the amplitude and direction of the QRS complex in each of these six leads, the QRS complex with the greatest amplitude (upright or downward facing) has a QRS axis that is near parallel the lead chosen. The lead with the QRS complex that is diphasic (positive deflection is equal the negative deflection) is perpendicular the QRS axis.

Of the 6 frontal leads (I, II, III, aVR, aVL, and aVF), the lead with the largest QRS (height up or down) is approximately **parallel** to the electrical axis of the ventricles. The lead that has diphasic QRS complexes with equal amplitudes facing up and down is **perpendicular** to the electrical axis. For example, if lead II has the largest QRS, expect lead aVL (which is perpendicular to lead II) to have biphasic QRS complexes of equal amplitudes up and down.

For example, if after scanning the six leads in question, lead II is found to have the largest amplitude, the QRS axis of the ventricles is parallel to lead II. If the QRS complex in lead II is predominantly upright, then the QRS axis is also moving in the direction of the positive electrode in lead II - in this case, the red electrode. These findings would support a QRS axis that is +60 degrees, a normal QRS axis.

Figure 6.16 Reference System to Identify the QRS Axis

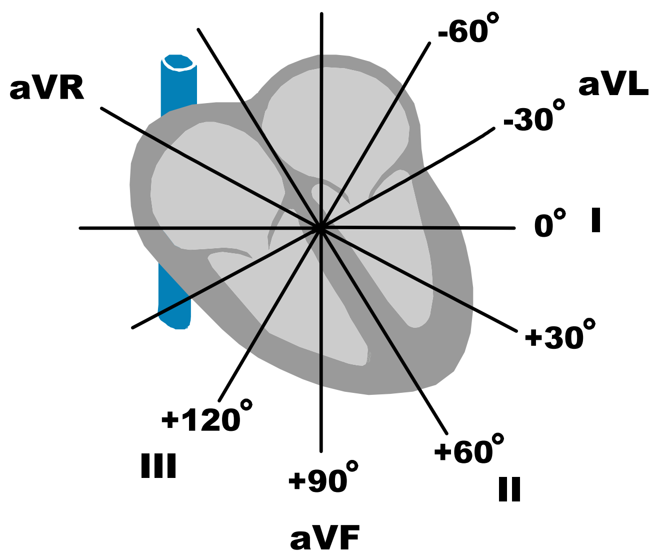


Figure 6.16 provides the 360^0 reference schematic created by the six limb leads. Upon first glance, this is somewhat overwhelming. With practice, this diagram becomes a simple tool. Begin with leads I and aVF. The coordinates for these leads are already well established with lead I the starting point of 0^0 and aVF perpendicular (90^0)to lead I. The standard limb leads (I,II,III) form Einthoven's equilateral triangle (Chapter 3) with 60^0 separating each lead. Hence lead II is 60^0 from lead I and lead III is a further 60^0 from lead II (120^0 from lead I). The augmented voltage leads also form the frontal plane with these three leads (aVR,aVL,aVF) covering a full 360^0. Each of these leads then are separated by 120^0. Lead aVL occupies a QRS axis of -30^0. The opposite end of lead aVR provides the +60^0 point.

Table 6.6 lists possible conditions that can cause deviations of the QRS axis.

Table 6.6 Cardiac Conditions and Electrical Axis Deviation

Electrical Axis Deviation	Cardiac Conditions
Right Axis Deviation - (more than +90 degrees)	pulmonary hypertension, right ventricular hypertrophy, right bundle branch block, normal for children, high lateral MI
Left Axis Deviation - (more negative than -30 degrees*)	left ventricular hypertrophy, inferior MI, left bundle branch block
Bizarre (+150 to -90 degrees)	limb lead misplacement, dextrocardia, occasionally with ventricular tachycardia

*The prevailing opinion is that left axis deviation only be placed on a QRS axis with a deviation of more than 30^0. Many healthy elderly patients show a QRS axis between 0^0 and -30^0. For the sake of simplicity, left axis deviation of more than 0^0 can be considered significant.

Identifying a QRS axis is a rather simple exercise. Making sense of your results requires the ability to place all clinical information in context. How does this electrical axis compare with previous ECGs? What is the patient's cardiac history? Having the skill to identify a QRS axis can help build a more complete clinical picture.

R Wave Progression*

Leads of the frontal plane (I, II,III,aVR, aVL, aVF) were the focus of the last section in identifying the QRS axis. The phenomenon of R wave progression utilizes the remaining six precordial chest leads. The chest leads provide information on the sagittal plane from the anterior surface across to the lateral surface of the heart.

Notice in Figure 6.17 how the R wave in lead V1 is small (predominant S wave), with the R wave progressively increasing in amplitude in leads V4 to V5. The R wave (upright wave of the QRS complex) is the dominant wave by lead V3. This gradual increase in the size of the R wave is called a **normal R wave progression**.

Figure 6.17 A Normal 12 Lead ECG

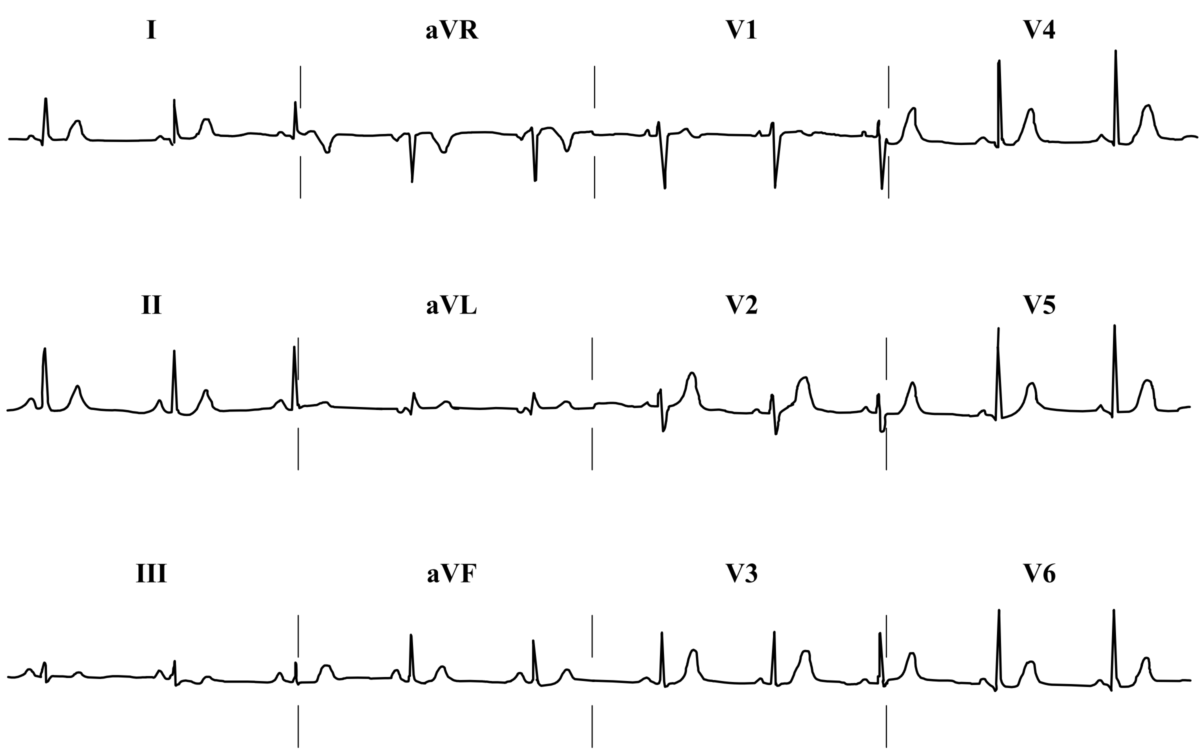


Figure 6.17 is a normal 12 lead ECG. Note the gradual increase in the size of the R wave across the precordial leads with the R wave becoming dominant by V3 or V4.

*optional topic not vital to basic 12 lead ECG interpretation

Abnormal R wave progression can occur with acute myocardial infarctions and right ventricular hypertrophy. Large, dominant R waves in leads V1 and V2 may indicate posterior or lateral myocardial infarction and right ventricular hypertrophy. Poor R wave progression (i.e. not until leads V5 or V6) may signal an anterior infarction.

Because the heart is a three dimensional organ, each of the waveforms may reflect or mirror an opposite region of the heart (reciprocal leads). For example, large R waves in leads V1 and V2 (septal, anterior) may reflect prominent Q waves present in posterior or lateral lead ECGs.

In line with QRS axis deviation, abnormal R wave progression does not stand on its own with sufficient strength to form a diagnosis. Placed with other findings, though, abnormal R wave progression may help support a diagnosis when bolstered by other findings.

Atrial Enlargement and Ventricular Hypertrophy*

Our account of the 12 lead ECG is rounded out with a brief discussion of the ECG criteria that constitute chamber enlargement. The atria can enlarge and the ventricles can hypertrophy as a compensation to increased resistance attributed to a high afterload. Enlarged heart chambers can be suspected - but not confirmed - with a 12 lead ECG. A superior tool for this purpose is the echocardiogram.

Atrial Enlargement

Atrial enlargement is best analysed using lead II. A P wave should always be upright in lead II (see Chapter 3). A normal P wave begins with the depolarization of the right atrium and completes with the depolarization of the left atrium. A notched P wave in lead II with increased amplitude to the latter aspect of the P wave (left atrium) suggests **left atrial enlargement**. Right atrial enlargement is reflected by a tall upright P wave of more than 2.5 mm in leads II, III and/or aVF.

Right atrial enlargement is often caused by chronic lung disease such as pulmonary hypertension and COPD. Left atrial enlargement arises from left ventricular failure and mitral valve disease. Criteria for atrial enlargement is illustrated in Figure 6.18 on the next page.

Figure 6.18 P Wave Morphology in Lead II and Atrial Enlargement

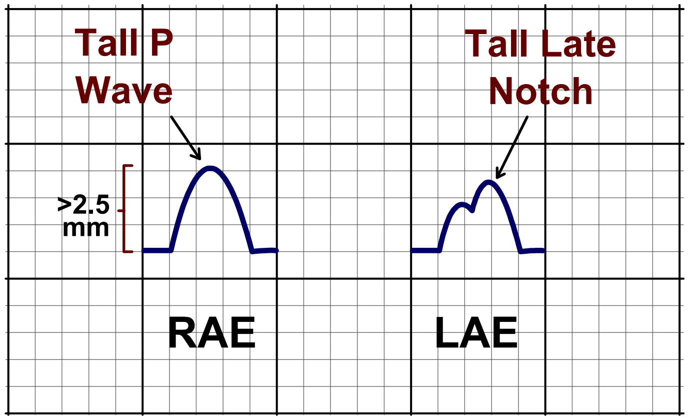


Figure 6.18 provides the ECG criteria for atrial enlargement. Right atrial enlargement (RAE) is suspected with a tall P wave of more than 2.5 mm in lead II. Left atrial enlargement (LAE) is suspected with a tall terminal notch on P waves in lead II, III or aVF.

Ventricular Hypertrophy

The ECG criteria for ventricular hypertrophy are more complicated and less conclusive. Ventricular hypertrophy is reflected in QRS axis deviation towards the hypertrophied ventricle, increased amplitude in the QRS complex, altered R wave progression, and possibly signs of ventricular strain - ST depression and T wave inversion.

A reversed R wave progression (large V1 and V2 with S waves in V4-V6) and right electrical axis deviation are common findings with right ventricular hypertrophy (RVH). Similar to right atrial enlargement, RVH occurs with lung disease and pulmonary valve dysfunction.

Left ventricular hypertrophy is reflected in taller than normal R waves in leads V5 and V6. One method involves adding the amplitude of the S wave in lead V1 or V2 to the amplitude of the R wave in V5 or V6. If the amplitudes add to 35 mm or more, strongly suspect ventricular hypertrophy. Also, left ventricular hypertrophy may produce ST depression and T wave inversion (ventricular strain) in the lateral leads V5 and V6.

Ventricular Strain

With severe ventricular hypertrophy, the myocardium can thicken to such a degree that the blood supply to the subendocardium (inner lining of the heart just inside the endocardium) can diminish. As a result, the endocardium is particularly susceptible to hypoxia.

While uncomplicated ventricular hypertrophy affects ventricular depolarization and the QRS complex (i.e. tall R waves), severe ventricular hypertrophy can also affect ventricular repolarization as seen with ischemic changes to the ST segment and T wave. Called a ventricular strain pattern, severe ventricular hypertrophy can cause ST segment depression and T wave inversion (see Figure 6.19).

Figure 6.19 Ventricular Strain

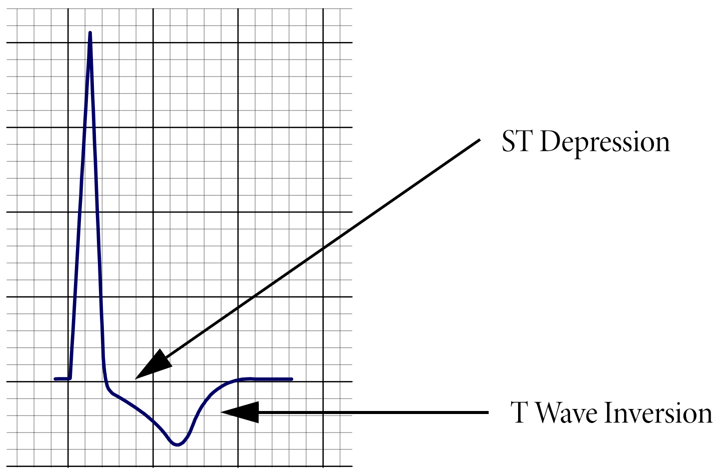


Figure 6.19 depicts the QRST ventricular strain pattern. Note the tall R wave that is typical of ventricular hypertrophy. A ventricular strain pattern includes ST depression and T wave inversion typically in leads with tall R waves.

Not surprisingly, a right ventricular strain pattern may be seen in leads closest to the right ventricle - leads V1 and V2. Left ventricular strain, if present, is usually evident in the lateral leads (I, aVL, V5 and V6).

As mentioned earlier, an echocardiogram should be ordered to prove or disprove 12 lead ECG findings that only *suggest* possible hypertrophies. The value to being aware of these criteria includes the ability to recognize that ST depression and T wave inversion are not always signs of cardiac ischemia. After all, a 12 lead ECG is performed much more frequently than an echocardiogram. Evidence that supports chamber enlargement or hypertrophy could offer some insight into a patient's current clinical status, prompting further investigations.

Summary

This chapter completes our coverage of the ECG, expanding the mapping of the heart from a single lead view to as much as 18 lead views. This chapter focused on the skills necessary to quickly identify cardiac ischemia, injury, and infarction. Abnormal waveforms - prominent Q waves, ST deviation, T wave inversion, QRS axis deviation or altered R wave progression - can point to myocardial ischemia and infarction. The ability to map these findings to regions of the heart is crucial.

Most of us have come to realize that there are exceptions to any rule and electrocardiography has its fair share of exceptions. Bundle branch blocks produce changes to the ECG that mimic ischemic and infarcted zones. Criteria to differentiate between right and left bundle branch blocks were included to rule out the possible influence of bundle branch blocks on the morphology of the waveforms that make up a 12 lead ECG.

A systematic analysis of the 12 lead ECG is necessary to consistently arrive at a well-informed clinical impression. A four step method begins with interpreting the ECG rhythm. Then hunt for signs of cardiac ischemia and infarction. Next rule out the presence of a right or left bundle branch block as the cause of seemingly pathological changes to the 12 lead ECG. For the willing, complete the systematic analysis by revisiting each of the waveforms for additional signs for chamber enlargement, ischemia and infarction.

Lead views of the frontal plane (I, II, III, aVR, aVL, aVF) are required in the identification of the QRS axis. Two simple methods were presented, using either quadrants or angles to describe the direction of the QRS axis. Left and right axis deviation, while not a conclusive diagnostic indicator, can point to a variety of cardiac conditions.

The precordial chest leads (V1-V6) typically produce the phenomena of R wave progression. The amplitude of the R wave normally becomes dominant over the depth of the S wave by lead V3 or V4. Poor or late R wave progression may signify an acute anterior MI. Also, large R waves in V1 and V2 may point to an acute posterior MI. The ability to identify a QRS axis and R wave progression are optional skills not vital to basic 12 lead ECG interpretation. Criteria for atrial enlargement are simpler and more conclusive than the criteria for ventricular hypertrophy.

An echocardiogram is a superior tool in diagnosing chamber enlargement and hypertrophy. Much can be revealed with a 12 lead ECG. When analyzed together with the previous 12 lead ECGs, a thorough physical assessment and a cardiac history, the 12 lead ECG is a powerful tool.

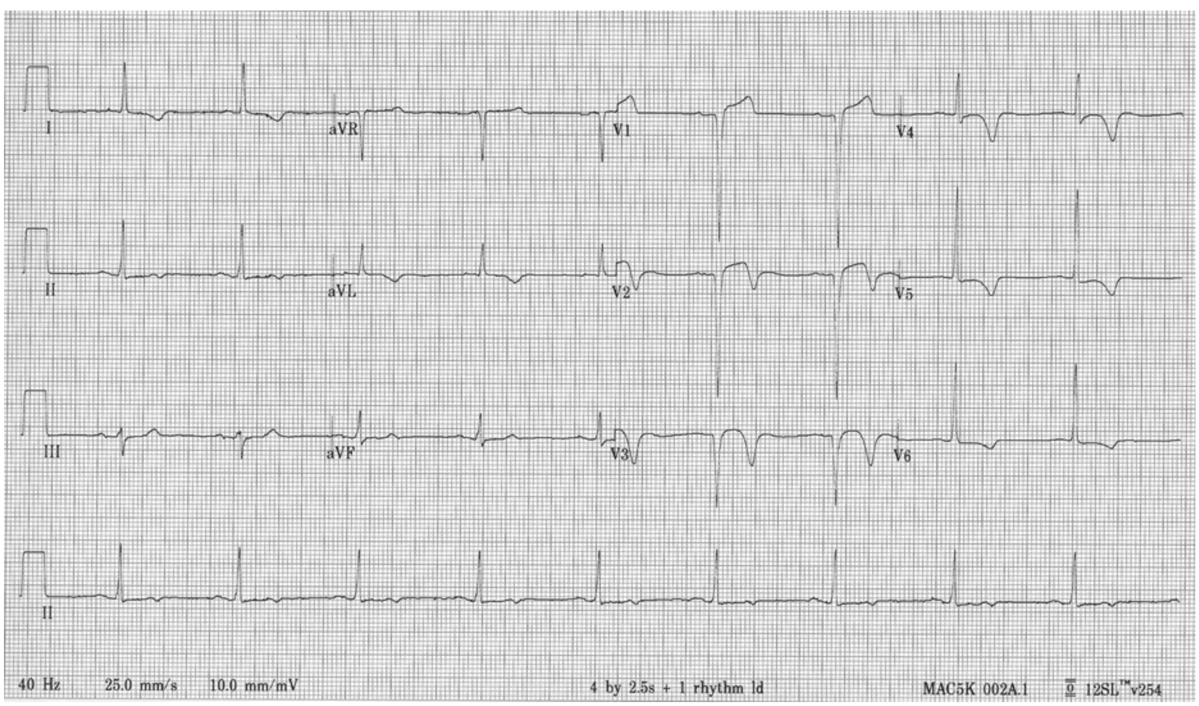
Chapter Quiz

1. Electrical depolarization that is directed towards a positive electrode provides a (positive deflection, negative deflection) on an electrocardiogram.

2. The presence of ST elevation in anterior, lateral and inferior lead views of a 12 lead ECG points to:

a) imminent death
b) pericarditis
c) global myocardial infarction
d) left bundle branch block

Figure 6.20 12 Lead ECG for Questions 3-6.



Questions #3-6 refer to Figure 6.20.

3. This 12 lead ECG shows ST elevation in the:

a) anterior leads
b) lateral leads
c) inferior leads
d) posterior leads

Answers: **1.** positive deflection; **2.** b); **3.** a)

4. This 12 lead ECG shows prominent Q waves identifying a myocardial infarction in:

a) the anterior leads
b) the lateral leads
c) the inferior leads
d) no lead views of this 12 lead ECG

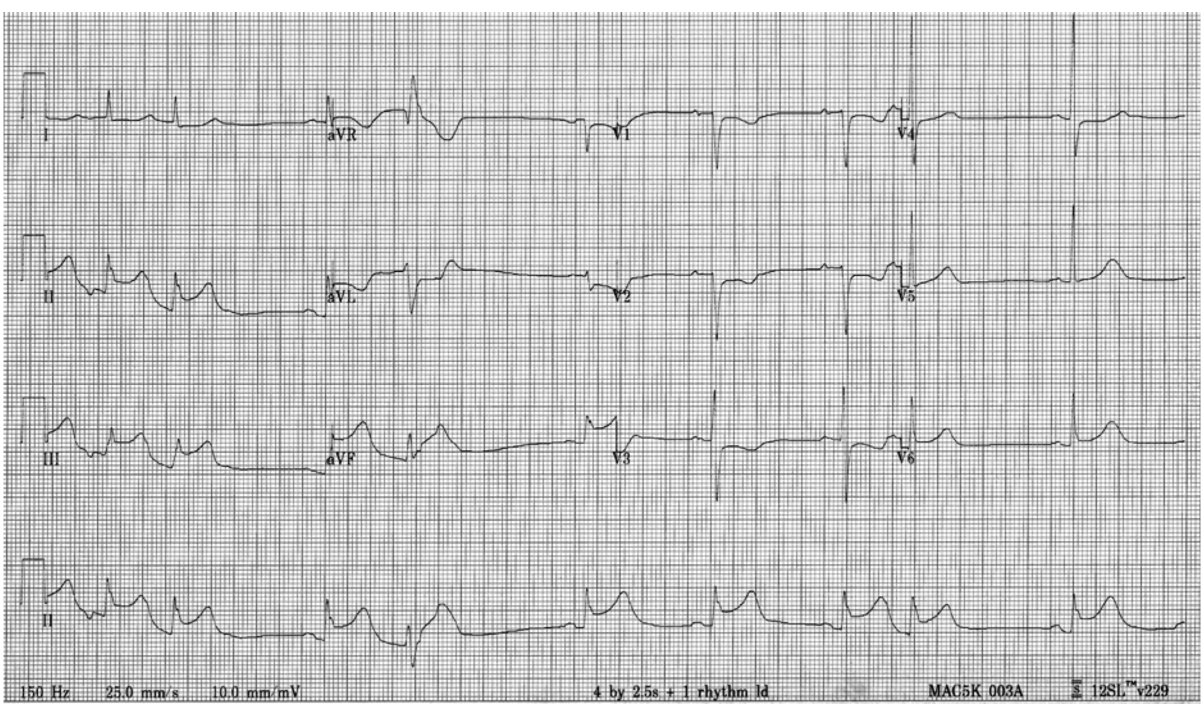
5. Abnormal inverted T waves (usually only in lead aVR) are present in leads (circle all that apply):

a) anterior leads
b) lateral leads
c) inferior leads
d) posterior leads

6. This 12 lead ECG shows:

a) anterolateral ischemia
b) pericarditis
c) posterior MI with reciprocal changes in the anterolateral leads
d) evolving anterior MI with signs of ischemic changes to the lateral leads

Figure 6.21 12 Lead ECG for Questions 7-13.



Answers: **4.** a); **5.** a), b); **6.** d);

Questions #7-13 refer to Figure 6.21.

7. The ECG rhythm provided by lead II at the bottom of this 12 lead ECG is:

a) atrial fibrillation with ST elevation
b) sinus rhythm with ST elevation and two PACs and one PVC
c) junctional rhythm with multifocal PVCs
d) wandering pacemaker with ST elevation

8. Looking at lead I, this patient (is, is not) in first degree AV block.

9. The diphasic P waves in lead V1 are a normal finding with this lead view.

True or False

10. This 12 lead ECG shows ST elevation in the:

a) anterior leads
b) lateral leads
c) inferior leads
d) posterior leads

11. This 12 lead ECG shows prominent Q waves identifying a myocardial infarction in:

a) the anterior leads
b) the lateral leads
c) the inferior leads
d) no lead views of this 12 lead ECG

12. Abnormal inverted T waves (usually only in lead aVR) are present in leads (circle all that apply):

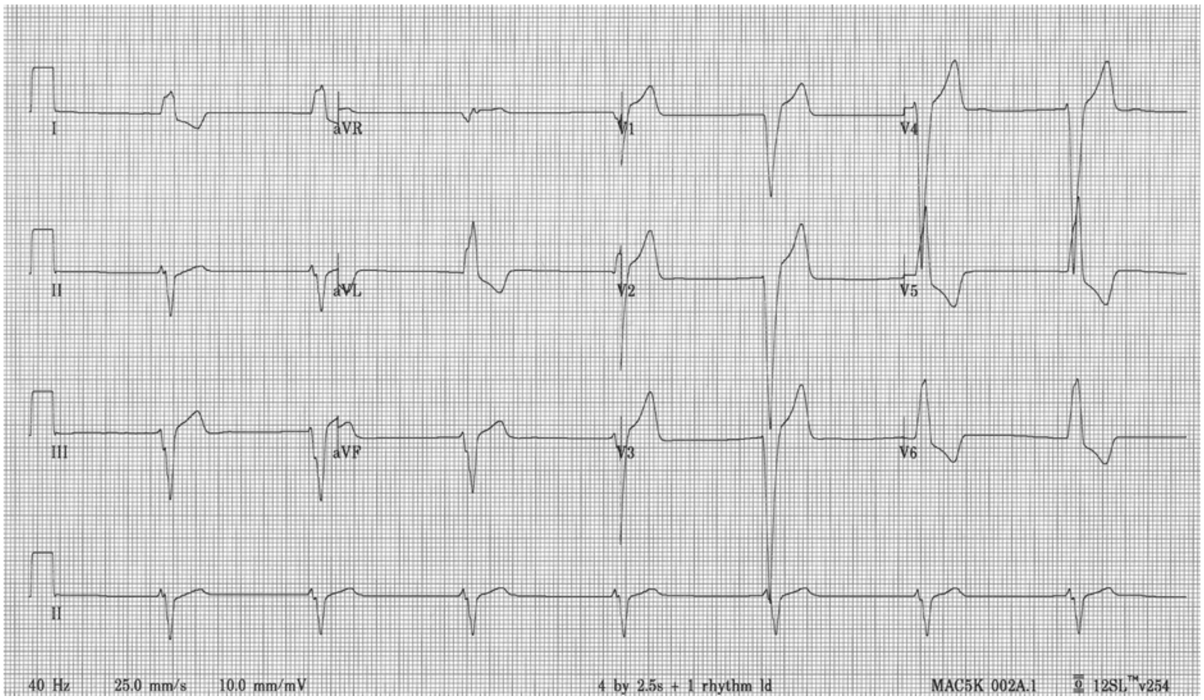
a) anterior leads
b) lateral leads
c) inferior leads
d) posterior leads

13. This 12 lead ECG shows:

a) inferior MI with reciprocal changes to the anterior leads
b) inferior MI with anterior ischemia
c) an old inferior MI
d) evolving anterolateral MI

Answers: **7.** b); **8.** is; **9.**True; **10.** c); **11.** d); **12. a); 13.** a);

Figure 6.22 12 Lead ECG for Questions 14-20.



Questions #14-20 refer to Figure 6.22.

14. The ECG rhythm provided by lead II at the bottom of this 12 lead ECG is:

a) sinus bradycardia
b) sinus rhythm with ST elevation
c) junctional rhythm
d) idioventricular rhythm

15. When looking for P waves, often the best lead views are:

a) lead V1 and lead II
b) lead V1 and lead V6
c) lead II and lead III
d) the bipolar leads (I, II, III)

16. This 12 lead ECG shows ST elevation in the (circle all that apply):

a) anterior leads
b) lateral leads
c) inferior leads
d) there is no ST elevation

Answers: **14.** c); **15.** a); **16.**a),c)

17. This 12 lead ECG shows prominent Q waves identifying a myocardial infarction in:

a) the septal leads
b) the lateral leads
c) the inferior leads
d) no lead views of this 12 lead ECG

18. Abnormal inverted T waves (usually only in lead aVR) are present in leads (circle all that apply):

a) the septal leads
b) the lateral leads
c) the inferior leads
d) no lead views of this 12 lead ECG

19. This 12 lead ECG includes the presence of (a left bundle branch block, a right bundle branch block, normal QRS complexes in every lead view).

20. This 12 lead ECG shows a(n):

a) anterior and inferior signs of a myocardial infarction with lateral reciprocal changes
b) junctional rhythm with a left bundle branch block
c) sinus rhythm with a right bundle branch block
d) anterior MI

21. A left bundle branch block alone can cause changes in an ECG that are commonly associated with myocardial ischemia and infarction.

True or False

22. Lead II views the apex of the heart.

True or False

23. The inferior leads of the heart are:

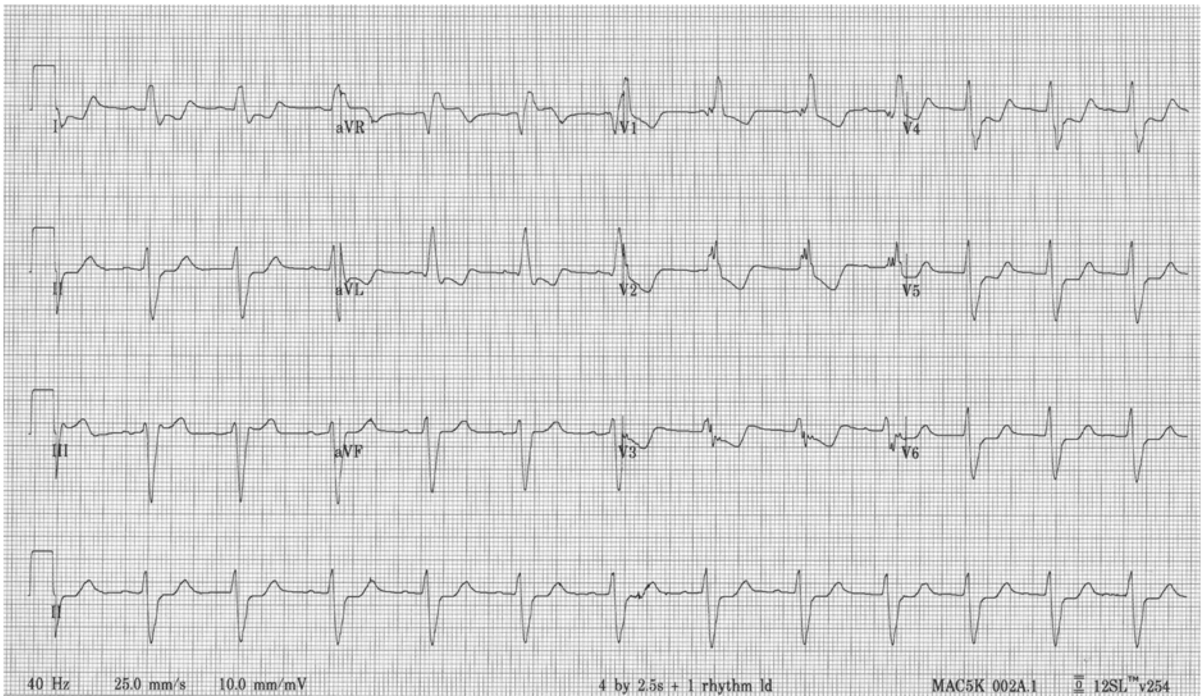
a) I, aVL
b) V3-V6
c) I, II, III
d) I, II, aVF

24. ST depression in leads V1-V3 may be a reciprocal change for a posterior STEMI.

True or False

Answers: **17.** a); **18.** b); **19.**left bundle branch block; **20.** b); **21.** True; **22.** True; **23.**d); **24.** True

Figure 6.23 12 Lead ECG for Questions 25-28.



Questions #25-28 refer to Figure 6.23.

25. The ECG rhythm provided by lead II at the bottom of this 12 lead ECG is:

a) sinus bradycardia
b) sinus rhythm
c) junctional rhythm
d) accelerated idioventricular rhythm

26. Abnormal inverted T waves (usually only in lead aVR) are present in leads (circle all that apply):

a) the anterior leads
b) the lateral leads
c) the inferior leads
d) no lead views of this 12 lead ECG

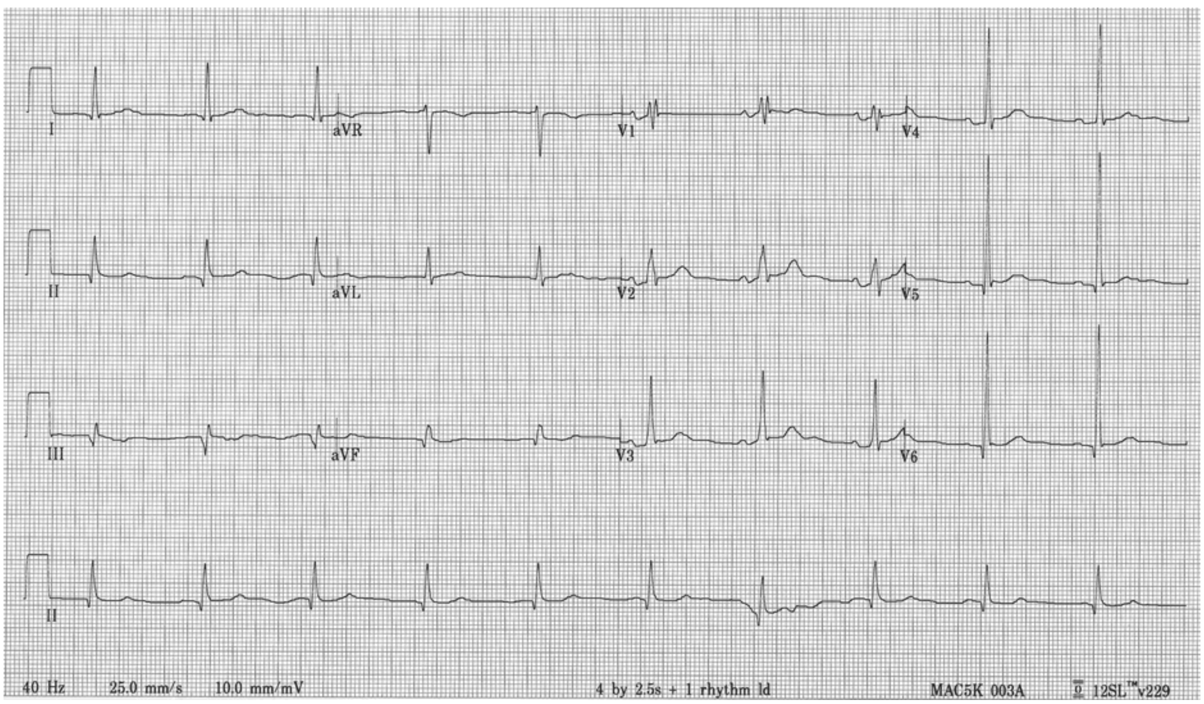
27. This 12 lead ECG includes the presence of (a left bundle branch block, a right bundle branch block, normal QRS complexes in every lead view).

Answers: **25.** b); **26.** a); **27.**right bundle branch block;

28. This 12 lead ECG is best interpreted as a:

a) anterolateral non-STEMI
b) junctional rhythm with a left bundle branch block
c) sinus rhythm with a right bundle branch block
d) right ventricular infarction

Figure 6.24 12 Lead ECG for Questions 29-33.



Questions #29-33 refer to Figure 6.24.

29. The ECG rhythm of this 12 lead ECG is:

a) sinus tachycardia
b) sinus rhythm
c) junctional rhythm
d) accelerated junctional rhythm

30. This 12 lead ECG includes the presence of (a left bundle branch block, a right bundle branch block, normal QRS complexes in every lead view).

31. The lead that provides the best view of P waves in this 12 lead ECG is (lead I, lead II, lead V1, lead V5).

Answers: **28.** c); **29.** b); **30.** right bundle branch block; **31.** lead V1

32. This 12 lead ECG shows prominent Q waves identifying a myocardial infarction in:

a) the anterior leads
b) the lateral leads
c) the inferior leads
d) no lead views of this 12 lead ECG

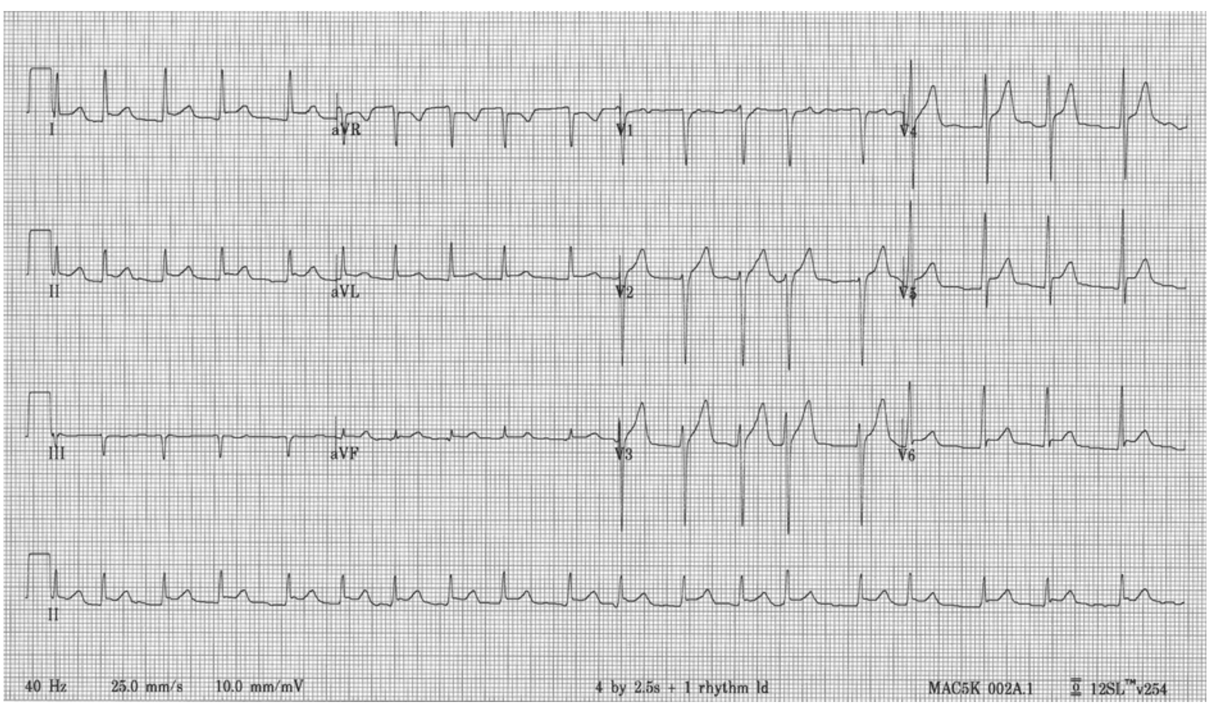
33. This 12 lead ECG is best interpreted as a:

a) right bundle branch block with evidence of an inferior MI of indeterminate age
b) left bundle branch block
c) sinus rhythm with a right bundle branch block
d) right ventricular infarction

34. While ST changes and T wave inversion are often signs of myocardial ischemia and infarction, these changes could also be produced by (circle all that apply):

a) ventricular hypertrophy
b) left bundle branch block
c) right bundle branch block
d) early repolarization

Figure 6.25 12 Lead ECG for Question 35.



Answers: **32.** c); **33.** a); **34.**a), b), c), d);

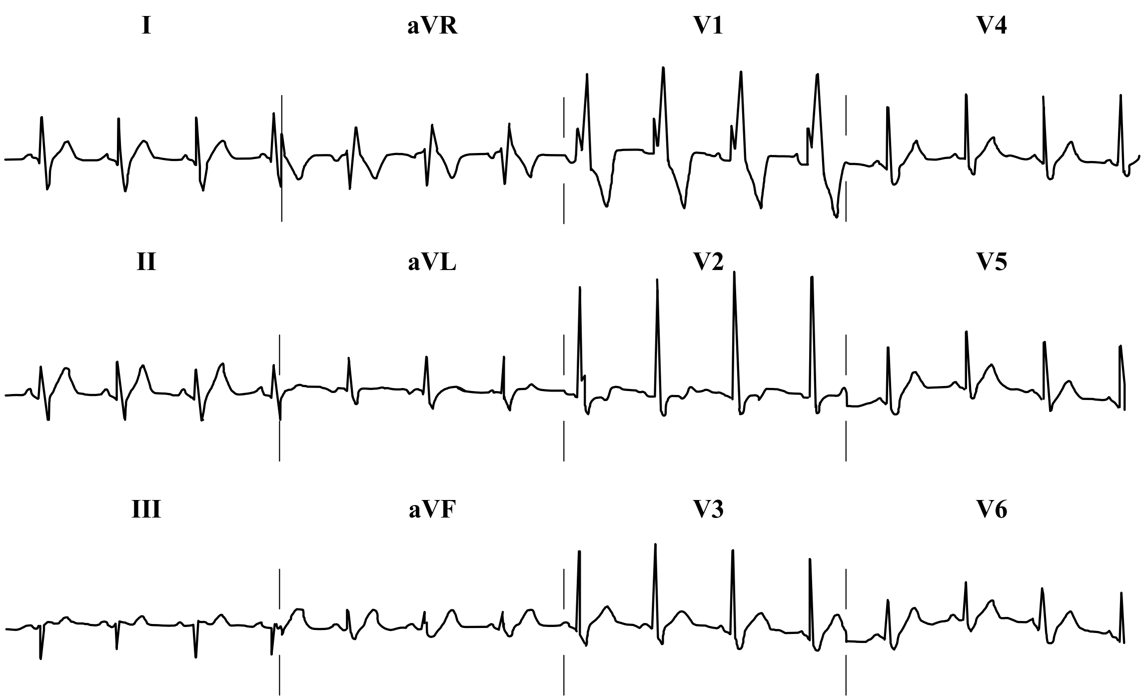
Questions #35 refers to Figure 6.25.

35. This 12 lead ECG interpretation would include the terms (circle all that apply):

a) sinus rhythm
b) early repolarization (or pericarditis if symptomatic)
c) atrial fibrillation
d) possible old inferior MI

36. The following 12 lead ECG in Figure 6.26 presents with a (LBBB, RBBB).

Figure 6.26 12 Lead ECG for Question #36



The next 14 questions are optional for those who are interested in QRS axis, chamber hypertrophy and R wave progression.

37. A 12 lead ECG shows an upright QRS complex in leads I and aVF. The QRS axis is directed toward:

a) the normal lower left quadrant
b) the normal lower right quadrant
c) the upper left quadrant
d) the upper right quadrant

Answers: **35.** b), c), d); **36.** RBBB; **37.** a)

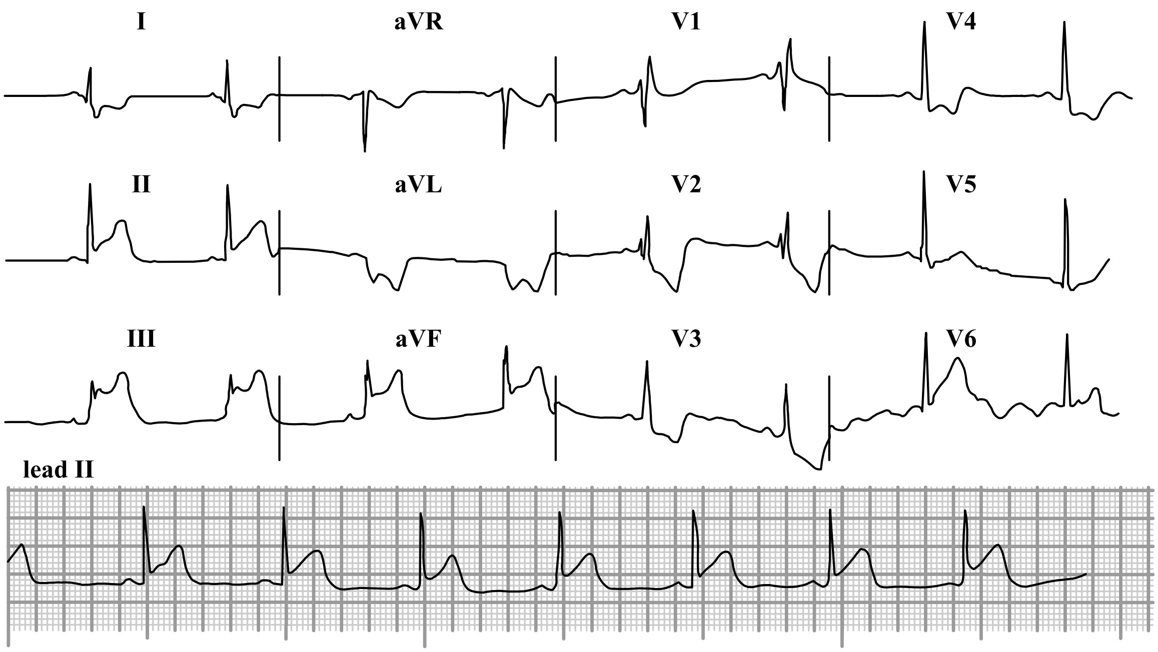
38. A normal QRS axis is -60 degrees along lead III.

True or False

39. A tall R wave in leads V1 and V2 can be caused by an acute posterior MI and by right ventricular hypertrophy.

True or False

Figure 6.27 12 Lead ECG.



Questions #40-45 refers to Figure 6.27.

40. The ECG rhythm is called a ______________________________.

41. The QRS axis is close to ________________ degrees.

42. Q waves? No___ Yes___ If yes, in which leads? ______________________

43. R wave progression is (normal, abnormal).

44. ST deviation? No___ Yes___ If yes, ST elevation in leads ______________.
ST depression in leads _________________________.

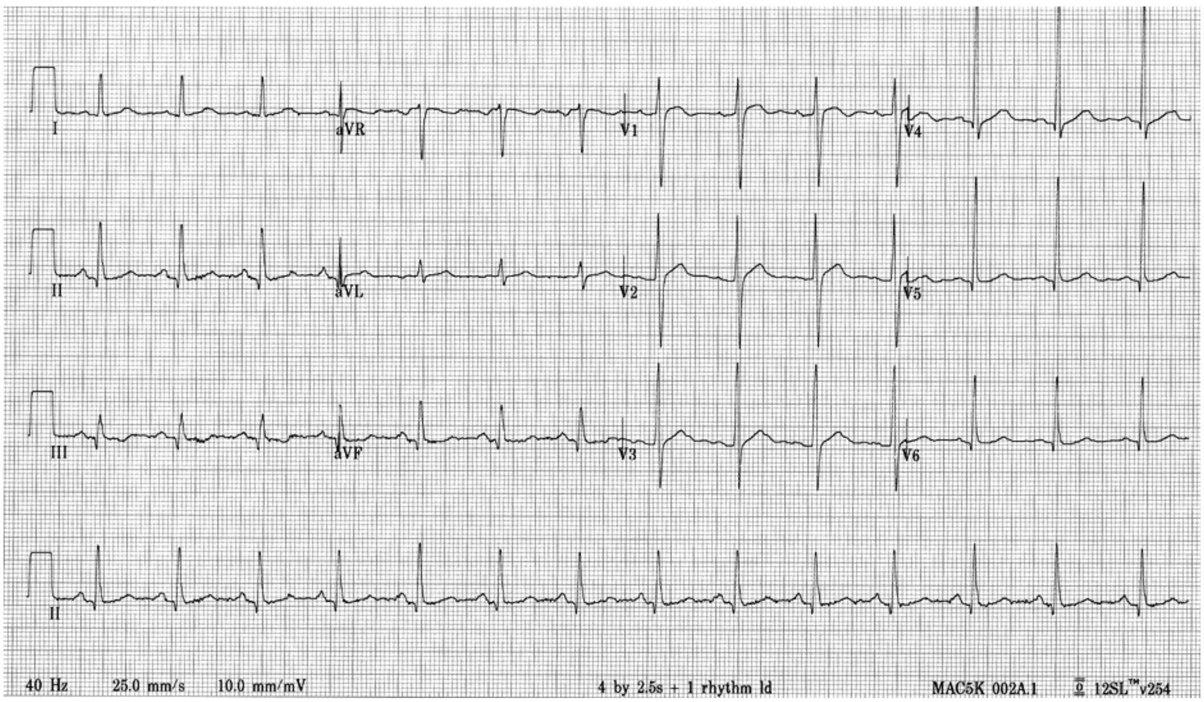
45. T wave changes? No___ Yes___
If yes, T wave inversion in leads _________________________.

Answers: **38.** False; **39.** True; **40.** sinus rhythm with ST elevation; **41.** 60 degrees; **42.** No; **43.** normal**; 44.**II, III, aVF; anterolateral; **45.** Anterolateral leads (V1-V4, leads I, aVL)

46. Your interpretation: ________________________________

47.Which leads are showing reciprocal changes? ______________

Figure 6.28 12 Lead ECG for Questions 48-50.



Questions #48-50 refer to Figure 6.28.

48. The ECG rhythm of this 12 lead ECG is:

a) sinus tachycardia
b) sinus rhythm
c) junctional rhythm
d) accelerated junctional rhythm

49. An echocardiogram is ordered for this patient to further investigate for the presence of (left ventricular hypertrophy, anterior ischemic changes, pericarditis) - already supported by findings on the 12 lead ECG.

50. Deep Q waves in the inferior leads is strongly associated with an old inferior myocardial infarction of indeterminate age.

True or False

Answers: **46.** acute inferior MI with a RBBB; **47.** anterolateral; 48. b); **49.** left ventricular hypertrophy; **50.** True;

Suggested Readings and Resources

Aehlert, Barbara. (2001). ECGs Made Easy. 2nd ed. New York: Mosby

Dubin, Dale. (2000). Rapid Interpretation of EKGs. 6th ed. Tampa, Florida: Cover Publishing

Grauer, Ken. (1998). A Practical Guide to ECG Interpretation. 2nd ed. New York: Mosby

Mariott, H. and Conover, B. (1998). Advanced Concepts in Arrhythmias. 3rd ed. New York: Mosby

O'Grady, S. (2001). Prehospital 12-Lead ECG. Redmond, Washington: Physio-Control Corporation. Found at http://www.physiocontrol.com/documents/12Lead.pdf

Thaler, Malcolm S. (1997). The Only EKG Book You'll Ever Need. 2nd ed. New York: Lippincott-Raven

Yanowitz, Frank W. (2001). The Alan E. Lindsay ECG Learning Centre in Cyberspace.Found at http://medlib.med.utah.edu/kw/ecg/index.html

What's Next?

Well, that's up to you. Practice, practice, practice. Gaining additional experience interpreting ECGs will build your knowledge and skills. Know that this is an introductory account of basic and 12 lead ECGs. Much more is available on this topic...much, much more. If you are hungry for more, check out the resources mentioned above. As well, the internet is a wonderful resource with several sites that are dedicated to ECG interpretation from the simple to the advanced.

Glossary

A

Absolute Refractory Period:
period when the cardiac cells cannot depolarize irrespective of the strength of the electrical impulse

Accelerated Junctional Rhythm:
cardiac rhythm that originates from the AV junction with a rate of 60-100/minute; QRS complex is most often narrow with P waves that are absent or inverted; PR interval is often short

Accelerated Idioventricular Rhythm:
cardiac rhythm that originates from the ventricular with a rate of 40-100/minute; QRS is wide with P waves absent

Accessory Pathway:
alternative connecting pathway between the atria and the ventricles (beside the Bundle of His); resulting syndrome is called Woolf-Parkinson-White syndrome

Actin:
one of two types of fibres that lie parallel to each other; with the influx of calcium into the cell the fibres bind together and shorten, causing contraction

Action Potential:
the electrical activities of a cell from depolarization to repolarization; 5 phases (0-4) take place largely involving the sodium, potassium and calcium ions

Aerobic Metabolism:
energy production through oxygenation (with oxygen) yielding 32 ATP molecules from the interaction of one glucose and one oxygen molecule

Anaerobic Metabolism:
energy production without oxygen with only 2 ATP produced as well as lactic and pyruvic acid

Afterload:
the pressure that the ventricle (right or left) must overcome to eject blood (i.e. the left ventricle pumps against aortic diastolic pressure and systemic vascular resistance

Agonal Rhythm:
a fatal dysrhythmia with rates usually less than 20/minute and common widening of the QRS; also referred to as the dying heart

Alpha Stimulation:
the peripheral vasoconstriction that results from circulating catecholamines (epinephrine & norepinephrine)

Amplitude:
the height or depth of waves and complexes of an ECG in millimetres; represents millivolts where 10 mm is 1 millivolt with a properly calibrated monitor

Angina:
chest discomfort – usually pressure or tightness – that results from a relative poor oxygen supply to cardiac tissue (ischemia)

Anterograde Conduction:
forward conduction of the electrical impulse across the atria & ventricles; the expected route of electrical conduction from top to bottom

Aorta:
begins with the aortic valve, the largest main vessel that carries oxygenated blood from the ventricles to the body

Apex (of heart):
the bottom of the heart (inferior aspect) located usually around the 5th intercostal space

Arrhythmia:
technically the absence of rhythm, arrhythmia is commonly used interchangeably with the term dysrhythmia

Artery:
main vessels carrying blood from the heart; the arteries have minimal elasticity and contain approximately 20% of the blood supply

Asynchronous Pacemaker:
pacemaker (non-demand mode) fires irrespective of the person's intrinsic firing; note that the risk for R-on-T phenomena and resulting ventricular tachycardia/fibrillation is higher than synchronous pacing (demand mode)

Asystole:
absence of electrical activity demonstrated by a straight ECG line

Atria:
right and left atria (1/3 volume and muscle mass of the ventricles) pump blood to the ventricles

Atrial Fibrillation:
one of the most common dysrhythmias, the atria have a host of sites that are concurrently firing at a combined rate of 350-600/minute; since the junction allows maximum 240 impulses through per minute, the result is a chaotic rhythm most often with a narrow QRS; note that without effective pumping from the atria, atrial kick is lost and the risk of blood clotting in the atria is relatively high

Atrial Flutter:
a rapid atrial rhythm (approximately 300/minute in the atria) caused by a re-entry loop within the atria; since the junction is unable to conduct 300 impulses/ minute (max=240), the junction often allows every 2nd, 3rd, or 4th impulse through; resulting ventricular rate is approximately 150, 100, and 75/min. respectively

Atrial Kick:
the contraction of the atria prior to ventricular contraction causes an increased volume and stretch to the ventricles – resulting in increased force of contraction and increased stroke volume (Starling's Law); this extra stroke volume increases cardiac output by 10-35%

Atrial tachycardia:
a fast rhythm with rates commonly 150-240/minute; QRS complex is most often narrow; rhythm is usually generated from a re-entry loop – often making use of an accessory bundle and the AV node

Atrioventricular valve:
the valves that connect the atria to the ventricles; the tricuspid valve resides in the right side of the heart; the bicuspid or mitral valve connects the left atria to the left ventricle

Automaticity:
a cardiac cell's (usually pacemaker cells such as the SA node, AV node or His-Purkinje network) ability to self-initiate an impulse; note that abundant catecholamines and/or ischemia enhances automaticity – non-pacemaker cells may become pacemaker cells

Autonomic Nervous System:
involuntary nervous system consisting of the sympathetic and parasympathetic nervous systems

AV junction:
conducts the impulse through the fibrous plate that separates the atria and the ventricles; consists of the AV node and the Bundle of His; functions also to slow the conduction speed to allow for atrial conduction prior to ventricular conduction (atrial kick); also serves as a pacemaker if the SA node fails to fire

AV node:
is located in the inferior aspect of the right atria; functions to slow the conduction speed to allow for atrial conduction prior to ventricular conduction (atrial kick); also serves as a pacemaker if the SA node fails to fire

AV Dissociation:
the atria and the ventricles are firing independently of each other (i.e. complete heart block and ventricular tachycardia)

AV Sequential Pacemaker:
a dual chamber pacemaker that fires first in the atria, then in the ventricle similar to normal cardiac functioning – allowing for atrial kick

B

Base (of heart):
the top of the heart located approximately at the 2nd intercostal space

Beta Stimulation:
with adrenergic (sympathetic) stimulation, Beta 1 and Beta 2 responses occur; Beta 1 effects are increased heart rate and increased force of contraction resulting in increased cardiac output; Beta 2 effect is bronchial dilation

Biphasic (diphasic):
a wave that includes both an upright (positive) and downward (negative) deflection

Bipolar Lead:
the standard three lead system that forms Einthoven's triangle with the white electrode (right shoulder), black electrode (left shoulder) and red electrode (left lower abdomen) forming three bipolar leads (positive lead and negative lead) - Lead I (white to black) – Lead II (white to red) – Lead III (black to red)

Bradycardia:
a heart rate slower than a pacemaker's regular intrinsic rate – commonly thought as less than 60/minute for sinus rhythms; less than 40/minute for junctional rhythms

Bundle Branches:
the Bundle of His terminates in the right and left bundle branches, insulated rapidly conducting electrical pathways that connect with the Purkinje network and thus begin depolarizing waves across the ventricles; the left bundle branch splits into three smaller branches called fascicles

Bundle Branch Block:
since the bundle branches are insulated – they are encapsulated with a fibrous sheath – an obstacle to conduction in any bundle (i.e. ischemia or infarct) results in the impulse not carried through to the ventricle; as a result, the depolarizing wave from the other bundle branch must travel further to depolarize the remaining ventricle; due to the extra distance for the wave to travel, more time is taken to depolarize and the QRS is wider than normal

Bundle of His:

part of the AV junction, the Bundle of His conducts the impulse through the fibrous plate that separates the atria and the ventricle; the Bundle of His is also a pacemaker, firing at 40-60/minute

Burst:
a sudden group of 3 or more ectopic beats or complexes (also called a run or a salvo)

C

Calcium Ion Channel:
channel that allows the influx or efflux of calcium: the influx of calcium particularly in phase 2 of the action potential enables cellular contraction

Capture:
effective depolarization of the atria and/or the ventricles by an artificial pacemaker

Cardiac Arrest:
the absence of pulse and respiration – clinical death

Cardiac Output:
the amount of blood pumped out of the ventricle in a minute (most often refers to the blood pumped by the left ventricle)

Cardiac Tamponade:
excess fluid between the parietal and pleural layers surrounding the heart restrict the contraction of the heart; early signs of cardiac tamponade may be a narrowing range between systolic and diastolic blood pressures and elevated jugular venous distension

Cardioversion:
more accurately called synchronous cardioversion, an electrical current is applied to the heart when the ventricles are depolarizing (R wave as opposed to firing on the T wave which could produce lethal dysrhythmias – R-on-T phenomena) to produce a period of asystole with the objective of allowing the SA node to become the pacemaker

Catecholamines:
the chemicals of the sympathetic nervous system – epinephrine, norepinephrine, and dopamine

Cholinergic:
pertaining to the actions of the parasympathetic nervous system; chemical involved in producing cholinergic responses is acetylcholine

Chordae Tendaneae:
tendons that connect the cardiac valves to the papillary muscles to prevent valvular collapse

Chronotropic:

refers to a physiological response that involves the heart rate: note that a positive chronotrope increases the heart rate

Circumflex Artery:
branches from the left main artery and wraps the left ventricle travelling between the left ventricle and the left atria along the epicardial surface of the heart

Compensatory Pause:
refers to the period between two sinus beats with a premature complex in between. If the period in between the QRS complexes is twice the normal R-R interval, a compensatory pause has occurred; the presence of a compensatory pause strongly suggests that the premature complex in between originates from the ventricle

Complete Heart Block (3rd Degree):
a cardiac rhythm that occurs when the junction (or possibly bilateral bundle branches) does not conduct the impulse from the atria to the ventricles; a pacemaker below the block must then begin firing to sustain cardiac output; thus, the atria and the ventricle fire independently

Complex:
a collection of waveforms (i.e. QRS complex and the ECG complex)

Conductivity:
the ability of a cell to receive and transmit an electrical impulse

Congestive Heart Failure:
may occur from an overabundance of preload, high afterload and/or a damaged left ventricle; the overall effect is a backup of blood within the lung vasculature causing fluid to cross into the alveoli

Contractility:
a muscle cell's ability to shorten or contract through the action of actin and myosin - mediated by the calcium ion; the faster the influx of calcium, the more forceful the contraction

Coronary Sinus:
the common venous outlet into the right atria that drains the heart's venous system (from the coronary arteries)

Couplet:
the presence of two consecutive premature complexes (i.e. 2 consecutive PVCs are called a ventricular couplet)

D

Defibrillation:
for those in pulseless ventricular tachycardia (VT), ventricular fibrillation or polymorphic VT; an asynchronous application of an electrical current to the heart to depolarize all cells not in absolute refractory period – effectively producing a short period of asystole, hopefully followed by a sinus rhythm

Delta wave:
a slurring of the upstroke of the QRS produced by early depolarization (pre-excitation) of a ventricle via an accessory pathway (see Woolf-Parkinson-White Syndrome)

Demand Mode (Pacemaker):
the synchronous pacing of the heart by an artificial pacemaker; the pacemaker fires only if the heart does not initiate an intrinsic impulse within a defined time interval

Depolarization:
the rapid influx of positive ions (sodium and/or calcium) into a cell – depolarization is necessary for contraction to occur

Dextrocardia:
a relatively rare phenomena, with the heart located on the right side of the thorax (rather than the typical left thorax position)

Diastole:
the phase of relaxation during the cardiac cycle; occurs for the atria and the ventricles; blood enters the heart's chambers and the coronary arteries during diastole; note that diastole is as important as systole – the negative pressure created by chamber relaxation and opening draws blood into the chamber; diastole lasts approximately twice the period of systole (for heart rates up to 150-170/minute)

Diphasic (biphasic):
a wave that includes both an upright (positive) and downward (negative) deflection

Dromotropic:
refers to a physiological response that involves the conduction speed through the AV junction; note that a positive dromotropic effect increases the speed of conduction

Dual Chamber Pacemaker:
an artificial pacemaker fires first in the atria, then in the ventricle similar to normal cardiac functioning, thus allowing for atrial kick

Dyskinetic:
a sub-optimal contracting myocardium due to ischemia, infarction and other cardiac diseases such as cardiomyopathies

Dysrhythmia:
used interchangeably with arrhythmia, refers to any abnormal rhythm – not normal sinus rhythm or sinus tachycardia

E

ECG:
electrocardiogram; also called an EKG; a representation of electrical voltage measured across the chest over a period of time

Ejection Fraction:
the percentage of blood volume ejected from the ventricle; for example, if blood volume in the left ventricle at the end of diastole is 100 ml., and 80 ml. is the stroke volume ejected, then the ejection fraction is 80% or 0.8

EKG:
electrocardiogram; also called an ECG

Ectopic:
a depolarizing wave that originates anywhere outside of the SA node

Einthoven's Triangle:
Dr. Einthoven was a physiologist, who in the late 1800s, first established the 3-lead cardiac monitoring system; the three lead system maps the electrical workings of the heart with a triangular lead system (Leads I,II,III)

Electrocardiogram:
the graphical representation of cardiac electrical activity – voltage (mV – y axis) measured over time (x axis)

Endocardium:
the smooth innermost layer of the heart covers the inner chambers and the cardiac valves

Epicardium:
the external layer that covers the heart – also called the visceral layer of the heart; between the visceral layer and the outer parietal layer is the pericardial sac; note that the coronary arteries travel along the epicardium before burrowing into the myocardium

Escape Interval:
the time interval between a heart's intrinsic impulse and an impulse generated by an artificial pacemaker

Escape rhythm:
a cardiac rhythm that arises when higher – faster – pacemaker sites fail to initiate an impulse; for example, if the SA node fails to fire, the junction most often begins to fire at a rate of 40-60/minute – called a junctional escape rhythm

Excitability:
a cell's ability to respond to an impulse by depolarizing

Extrasystole:
another term for a premature complex

F

Fascicle:
with regards to the heart, a fascicle is a smaller branch of the left bundle branch – two fascicles serve the left ventricle (one of which divides again); the three fascicles ensures rapid depolarization across the entire left ventricle

First Degree AV Block:
a characteristic of a junction with reduced conduction speed - an extended PR interval results >.20 seconds; when identifying 1st degree heart block, first mention the underlying rhythm, then that a 1st degree AV heart block is present

Fixed Rate Pacemaker:
an artificial pacemaker that fires irrespective of impulses initiated by the heart; risk of R-on-T phenomena and resulting lethal dysrhythmias is possible

H

Heart Rate:
the number of QRS complexes per minute; note that heart rate may not equal perfused pulse rate

His-Purkinje System:
the electrical network that includes the Bundle of His, the Bundle Branches and the Purkinje fibres

Hyperkalemia:
blood potassium level higher than normal limits

Hypertension:
blood pressure above acceptable limits – above 90 mm of Hg diastolic and above 140 mm of Hg systolic

Hypokalemia:
blood potassium level lower than normal limits

Hypotension:
blood pressure below acceptable levels – commonly below 90 mm of Hg systolic

Hypoxia:
a sub-therapeutic blood oxygen level resulting in markedly reduced energy production and the formation of lactic and pyruvic acid (anaerobic metabolism)

I

Idioventricular Rhythm (IVR):
a cardiac rhythm with the impulse originating in the ventricles and an absence of atrial activity (no P waves); heart rate is 20-40/minute; cardiac output is often poor with this slow rate and no atrial kick

Infarction:
the necrosis of tissue; acute myocardial infarction involves the acute death of myocardial cells

Inotropic:
refers to the contractility of the heart (often produced from sympathetic nervous system stimulation; note that a stimuli that leads to increased contractility is called a positive inotrope, stimuli that reduces the force of contraction is called a negative inotrope

Interpolated PVC:
a PVC that occurs when the next expected QRS complex is to occur; the rhythm pattern is not interrupted

Intrinsic:
characteristic or property natural to the heart and its structures

Ischemia:
insufficient supply of oxygen to meet the oxygen demands of tissue

Isoelectric Line:
also called the baseline, the straight line that is present when no electrical activity is present

Interval:
a period measured on rhythm strip paper that measures a wave and a segment; the distance measured is equal to time taken as an ECG is voltage over time; a PR interval for example is measured from the beginning of the P wave to the beginning of the QRS (includes the PQ segment)

J

J point:
a small notch in the QRS where the ST segment begins

Joule:
the unit of energy used for defibrillation and cardioversion; technically a joule is equal to power (voltage X current) times duration in seconds

Junction:
connects the atria to the ventricle and slows the impulse conduction speed sufficiently to allow for atrial kick; the junction consists of the AV node and the Bundle of His; the junction is a supraventricular structure

Junctional Escape Rhythm:
a cardiac rhythm that occurs as a backup pacemaker when the sinus node fails to initiate an impulse; the junction typically fires at 40-60/minute; the P wave is either absent or inverted

Junctional Rhythm:
a cardiac rhythm that occurs as a backup pacemaker when the sinus node fails to initiate an impulse; the junction typically fires at 40-60/minute; the P wave is either absent or inverted

Junctional Tachycardia:
a cardiac rhythm that occurs when the junction typically fires at >100/minute;

L

Left Anterior Descending Artery:
a major artery that serves the left ventricle, travelling along the left anterior epicardial surface of the heart

Lown-Ganong-Levine (LGL) Syndrome:
a type of pre-excitation syndrome similar to Woolf-Parkinson-White with the accessory pathway connecting the atria to the distal portion of the Bundle of His, thus bypassing much of the junction; note that a Delta wave is not present; characteristic signs include a shortened PR interval and a normal QRS complex

M

Mediastinum:
the region of the thorax behind the sternum and in front of the spine – includes the heart, esophagus, trachea and the major vessels attached to the heart

Microvasculature:
the network of smaller vessels – particularly the arterioles, the capillaries and the venules that serve regions of tissue

Milliampere:
unit of electrical current: artificial pacemakers use current to initiate depolarization of the heart

Mobitz:
a physiologist who in the 1920s discovered the 2nd degree AV heart blocks – Type I and Type II; note that the physiologist Wenckebach is reputed to accomplish similar discoveries 15-20 years earlier

Monomorphic:
similarly shaped; suggests that the waves or complexes originate from the same focus

Multifocal Atrial Tachycardia (MAT):
often associated with atrial hypertrophy, pulmonary hypertension and COPD, MAT is a tachycardia diagnosed by the presence of 3 or more P waves of differing morphologies

mV:
millivolt; the ECG is usually calibrated to display 1mV = 10mm of amplitude

Myocardium:
the muscle layer of the heart; the middle layer that is responsible for contraction of the heart

Myocardial Infarction:
the necrosis or death of myocardial tissue due to insufficient supply of oxygen to the infarcted region

Myosin:
one of two types of fibres that lie parallel to each other; with the influx of calcium into the cell the fibres bind together and shorten, causing contraction

N

Nodal:
commonly refers to the junction; for example, junctional rhythm is also known as nodal rhythm; may also refer to the sinoatrial node

Non-Compensatory Pause:
when the QRS complex following a premature complex occurs before expected; occurs with PACs (a full compensatory pause occurs with a PVC)

Normal Sinus Rhythm:
also known as regular sinus rhythm or sinus rhythm, this cardiac rhythm is not a dysrhythmia; sinus rhythm originates in the sinoatrial (SA) node with a rate of 60-100/minute; P waves are upright in most leads and the QRS is most often narrow

O

Overdrive Pacing:
paces the heart faster than its non-paced rate to entrain the heart – once the heart is dominated by the firing of the artificial pacemaker, the heart rate is brought down into a range <100/minute; eventually, the artificial pacemaker stops firing at rates where the sinus node begins firing

Oxygen Saturation:
the percentage of sites of circulating hemoglobin that are bound to oxygen – oxyhemoglobin; most people have normal oxygen saturation of 95% or higher

P

P wave:
a wave generated from the depolarization of the atria; the P wave is upright when originating from the SA node

PR interval:
the time and interval measured from the beginning of the P wave to the beginning of the QRS; should be called the PQ interval; normal PR interval is 0.12-0.20 seconds

Paced Interval:
the interval of time between artificially paced complexes

Paced Rhythm:
a cardiac rhythm that is generated by current provided by an artificial pacemaker; vertical pacemaker spikes precede the waves

Pacemaker:
an electronic pulse generator that stimulates depolarization of the atria and/or the ventricles

Pacemaker Generator:
the main battery unit as well as the electronics for the pacemaker; often resides under the skin inferior to the clavicle

Pacemaker Spike:
the telling sign that the rhythm is a paced rhythm; since the pacemaker spike is vertical, virtually no time was spent – this suggests strongly that the spike was initiated artificially and not organically (within the heart); note that a pacemaker spike must be followed by a wave – a spike and an immediate wave is called a captured beat – spike without an immediate wave is called loss of capture

Papillary Muscles:
small muscles that attach to the inner wall of the ventricles and to the chordae tendaneae (which in turn are connected to the heart's valves)

Parasympathetic Nervous System:
the involuntary nervous system that invokes a cholinergic response; for the heart, the Vagus nerve stimulates the SA node and the AV node, slowing the rate of impulse formation and the speed of conduction

Paroxysmal Atrial Tachycardia (PAT):
atrial tachycardia with a witnessed sudden onset; rhythm is regular with a rate of 150-240/minute

Paroxysmal Supraventricular Tachycardia (PSVT):
broad category for rapid rhythms originating outside of the ventricles that occur suddenly; rapid supraventricular rhythms include sinus tachycardia, junctional tachycardia, atrial tachycardia, atrial fibrillation with fast response and atrial flutter with fast response

Pericardium:
the sac that surrounds the heart (composed of the visceral and parietal layers); the pericardium is filled with approximately 30-50 ml. of surfactant fluid – this reduces the surface tension within the sac and allows it to open and close freely with cardiac contraction and relaxation

Phase 0 (action potential):
phase of depolarization: for myocardial cells the slow sodium channels open, and then, if threshold potential is reached, fast sodium channels open; note that cells of the SA & AV node are calcium dependent for depolarization

Phase 1 (action potential):
depolarization has now completed; repolarization begins with the efflux of potassium from the cell

Phase 2 (action potential):
potassium leaves the cell while calcium enters the cell resulting in an electrical plateau; the calcium entering the cell is significant because it initiates the cell's contraction

Phase 3 (action potential):
potassium leaves the cell resulting in the cell becoming increasingly negative; at 60-70 mV, the cell's fast sodium channels begin to be receptive to causing another depolarization – a period known as relative refractory period or the supranormal period

Phase 4 (action potential):
the polarized state of the cell with resting negative potential of approximately –90 mV; early in this phase, the sodium-potassium ATP pump restores the cell to its initial state

Polymorphic:
refers to waves that change in shape – usually refers to QRS complexes that have more than one impulse site

Potassium Ion Channel:
selective ion channel that allows potassium to enter or exit the cell

Pre-excitation:
depolarization of the ventricle(s) is begun via an accessory pathway (other than the AV junction) resulting in an early initiation of a QRS and possibly a Delta wave; meanwhile, the junction and the His-Purkinje network is responsible for depolarizing the remaining unaffected ventricles and also therefore for the remainder of the QRS

Preload:
technically the end-diastolic pressure of either the left or right ventricles; simplified as the blood volume supplied to the left or right ventricles; note that the more volume or preload, the greater the myocardial stretch and forceful the contraction; increased preload most often results in increased cardiac output

Premature Complex:
a QRS complex that comes earlier than expected

Premature Atrial Complex:
also known as a PAC, an ectopic firing within the atria results in an early QRS

Premature Junctional Complex:
also known as a PJC, an ectopic firing within the junction results in an early QRS

Premature Ventricular Complex:
also known as a PVC, an ectopic firing within the ventricle results in an early QRS that is: 1) wider than normal (>.12 seconds); 2) with a compensatory pause; and 3) often the R wave is pointing opposite to the T wave

Pulseless Electrical Activity:
also known as PEA, is the paradoxical combination of an organized rhythm without a palpable pulse; note that rapid ventricular tachycardia, ventricular fibrillation and asystole are rhythms that are not included within the PEA designation

Purkinje Network:
a matrix of fibres located throughout the myocardium that connects the impulse from the Bundle Branches to the myocardial tissue

Q

QRS Complex:
the electrical representation of ventricular depolarization; the atrial repolarization is also a part of the QRS

QT interval:
the time taken from the beginning of ventricular depolarization to the end of ventricular repolarization; this interval begins with the onset of the QRS to the end of the T wave; a normal QT interval should be less than or equal to half the R-R interval; note that long QT intervals are associated with increased incidence of R-on-T phenomena and resulting episodes of ventricular tachycardia or ventricular fibrillation

R

Rate:
heart rate; calculated by counting the number of QRS complexes in six seconds and multiplying by 10; rate is also determined by measuring the number of large squares between two R waves; i.e. –1 large square = heart rate of 300/minute

Re-entry:
one of several causes of dysrhythmias, the re-entry loop often results in tachycardias; the re-entry loop requires an area with slowed speed of conduction, an proximal area with regular conduction speed and cells that are receptive to depolarizing; the loop that occurs becomes self-sustaining and rapid, thus dominating the heart

Regular Sinus Rhythm:
also known as normal sinus rhythm or sinus rhythm, this cardiac rhythm is not a dysrhythmia; sinus rhythm originates in the sinoatrial (SA) node with a rate of 60-100/minute; P waves are upright in most leads and the QRS is most often narrow

Relative Refractory Period:
in phase 3 of the action potential, once the cell repolarizes to threshold potential to –90 mV, the cell's ion channels are once again receptive to depolarization; this period is also called the supernormal period - minimal impulse energy is required to stimulate sufficient ion transfer to reach threshold potential once again and cause depolarization

Repolarization:
follows depolarization, involving the return to a pre-depolarization state; the myocardial cell's electrical potential returns from +30 mV to its polarized state of –90 mV; the ions potassium, calcium and sodium are largely involved; note that contraction of the myocardial cell occurs during repolarization

Retrograde Conduction:
conduction along the electrical pathway opposite in direction from what occurs in sinus rhythm

Rhythm:
often refers to a discernible pattern in time or distance between QRS complexes and/or P waves

Right Coronary Artery:
branches from the aorta near the aortic valve and serves the right ventricle, the AV node (90% of the time), and part of the posterior aspect of the left ventricle

ROSC
return of spontaneous circulation

Run (burst, salvo):
a sudden group of 3 or more ectopic beats or complexes (also called a burst or a salvo)

S

ST Depression:
the downward placement of the ST segment greater than 1 mm from the isolectric line; suggestive of cardiac ischemia, but also may point to ventricular hypertrophy, digoxin use, and pericarditis among other possibilities

ST Elevation:
the upward placement of the ST segment greater than 1 mm from the isolectric line; suggestive of cardiac infarction or ischemia

ST Segment:
segment (line) between the end of the QRS complex and the beginning of the T wave; the end of the QRS is marked by the J point

Salvo:
a sudden group of 3 or more ectopic beats or complexes (also called a burst or a run)

Second Degree AV Block Type 1:
a cardiac rhythm characterized by occasional P waves without a QRS (lonely Ps) and an observable pattern of progressive lengthening of the PR interval followed by a P wave without a QRS (dropped QRS) – the PR interval then shortens followed by a repeat in the pattern

Second Degree AV Block Type 2:
a cardiac rhythm characterized by occasional P waves without a QRS (lonely Ps) and a fixed PR interval; 1 lonely P wave may be present or consecutive lonely P waves – this is a serious rhythm with potential significant effects on cardiac output

Segment:
a line between waves

Semilunar Valves:
valves located at the exit route of the ventricles; for the right ventricle, connects with the pulmonary artery; for the left ventricle, connects with the aorta

Sensitivity:
a function of an artificial pacemaker to sense intrinsic electrical activity of the heart

Sick Sinus Syndrome:
more common with the elderly, the matrix of fibrous tissue around the SA node becomes denser, eventually affecting the ability of the SA node to transmit an impulse consistently to the atria; characteristic signs include periods of sinus bradycardia following by periods of sinus tachycardia

Sinoatrial Node (SA node):
located in the upper right atrium near the inlet for the superior vena cava, serves as the predominant pacemaker for the heart with rates usually of 60-100/minute

Sinus Arrhythmia:
a cardiac rhythm that originates from the SA node without the usual regular rhythm indicative of sinus rhythm; this rhythm is common for children and for elderly adults; it presents as a narrowing of the R-R interval during inspiration and a widening R-R interval during expiration; note that P waves are upright in most leads and the QRS is narrow (unless a Bundle Branch Block is coexisting)

Sinus Bradycardia:
a cardiac rhythm that originates from the SA node with a rate less than 60/minute

Sinus Exit Block:
a non-conducted (blocked) sinus impulse exhibited by a pause with a length equal to twice the normal R-R interval

Sinus Arrest (Pause):
a failure of the sinus node to initiate an impulse; a sign of a diseased sinus node

Sinus Rhythm:
a cardiac rhythm that originates from the SA node with rate of 60-100/minute

Sinus Tachycardia:
a cardiac rhythm that originates from the SA node with rates most often 101-180/minute

Sodium Ion Channel:
a variety of fast and slow channels that are responsible for depolarization in the myocardial cell

Starling's Law (Frank-Starling):
established about a century passed by a physiologist Dr. Starling and two colleagues, the law states that the more the cardiac fibres stretch (within limits – with increased preload), the more forceful the contraction – and the greater the stroke volume and the cardiac output

Stroke Volume:
the amount of blood ejected by either the right or left ventricle with one beat (contraction)

Supraventricular:
located above the ventricle – includes the Bundle of His, AV node, atria and the SA node

Sympathetic Nervous System:
the involuntary nervous system that provides alpha, Beta 1 and Beta 2 responses as stimulated by catecholamines – epinephrine, norepinephrine and dopamine; combined responses include increased heart rate, more forceful contraction, peripheral vasoconstriction and bronchial dilation

Synchronous Pacemaker:
the synchronous pacing of the heart by an artificial pacemaker; the pacemaker fires only if the heart does not initiate an intrinsic impulse within a defined time interval

Syncope:
a symptom of lightheadedness or dizziness

Systole:
a contraction phase of the cardiac cycle; systole takes about 1/3 of the time of the cardiac cycle (2/3 of the time is taken by diastole)

T

T wave:
the wave that arrives after the QRS; is a graphical presentation of ventricular repolarization

Tachycardia:
a cardiac rhythm with a rate above 100/minute; for example, if the impulse originates from the atria with a rate of 160/minute, the rhythm is called an atrial tachycardia

Third Degree AV Block:
also known as complete heart block, the supraventricular impulse is blocked at the junction or high in the bundle branches; as a result, the myocardium above the block depolarizes independently of the myocardium below the block; characteristics of this rhythm include PR intervals that are chaotic, a R-R interval that remains equal and a P-P interval that remains equal; this is a serious rhythm due to the tenuous nature of the ventricular pacer – the slow ventricular rate is often associated with a poor cardiac output and the ventricular pacemaker can slow further to a stop

Torsades de Pointes:
means the twisting of the points, a polymorphic ventricular tachycardia characterized by periods of increasing QRS amplitude followed with a period of reduced QRS amplitude – then the pattern repeats again; often associated with effects of cardiac drugs or with low magnesium blood levels; often preceded by long QT intervals

Threshold:
the minimum level of electrical current necessary for an artificial pacemaker to stimulate depolarization of the heart

Threshold Potential:
the electrical potential point across the cell membrane where the cell's channels open to cause depolarization; for example, in the heart's myocardial cells, the fast sodium channels open if the electrical potential across the cell's membrane is –60 to –70 mV (note polarized state = -90 mV) – the fast sodium channels opening leads to depolarization; in the SA and AV nodes, the threshold potential is approximately –40 mV

Transmural Infarction:
a full thickness myocardial infarction through all three layers of the heart – epicardium, myocardium and endocardium

Troponin:
a large protein found within the muscle cells that is involved in the process of contraction; presence of abundant Troponin in blood plasma is strongly suggestive of muscle necrosis

U

U wave:
a wave that follows the T wave and precedes the P wave; its significance remains somewhat unknown, but it may be the repolarization of the Purkinje fibres; a U wave taller than 2 mm may suggest hypokalemia or that the person is on digoxin or quinidine

Unipolar Lead:
nine leads of a 12 lead ECG are unipolar leads; the unipolar leads view the heart directly from the electrode's vantage point; for example, V1 sits over the right ventricle and provides a view of the right ventricle directly beneath this lead

V

Vagal Maneuver (vagal response):
a maneuver that stimulates the Vagus nerve to be stimulated; possible maneuvers include a Valsalva maneuver (bearing down), ice thrust in ice water, anal stimulation, and carotid massage; baroreceptors in the carotid arteries respond to increased blood pressure (and increased cardiac output) by stimulating the Vagus nerve to fire; this slows down the firing rate of the SA node and also slows down the speed of conduction of the junction thus decreasing blood pressure and cardiac output

Veins:
blood vessels that carry blood to the heart

Ventricular Bigeminy (Trigeminy):
the presence of PVCs every second beat (Bigeminy) or every third beat (Trigeminy)

Ventricular Escape Rhythm:
otherwise known as idioventricular rhythm, is a cardiac rhythm with only ventricular complexes appearing at a rate of 20-40/minute; occurs in the absence of impulse initiation of either the SA node or the AV node; can be considered the last bastion of the pacemakers, although the ventricles are not considered dependable pacemakers (with the low rates, lack of atrial kick and probable low cardiac output)

Ventricular Fibrillation:
a chaotic unorganized cardiac rhythm with several firing regions within the ventricles; the result is uncoordinated ventricular activity and NO cardiac output – a lethal dysrhythmia; fine ventricular fibrillation defines an amplitude less than 3 mm; coarse ventricular fibrillation has amplitudes of 3 mm or more

Ventricular Tachycardia:
is a cardiac rhythm characterized by rapidly occurring wide QRS complexes with absent or infrequent P waves evident; the absence of atrial kick, the lack of filling time, and the tendency for ventricular tachycardia to change to ventricular fibrillation makes this a serious – and often lethal – dysrhythmia

Ventricles:
the larger chambers of the heart (3 times the volume and muscle thickness than the atria), responsible for the pumping of blood to the lungs and the rest of the body

W

Wandering Pacemaker:
also referred to as multiformed atrial rhythm, is a dysrhythmia with at least 3 different P waves originating from the SA node, atria and/or the junction

Waveform:
a wave of an ECG

Wenckebach:
a physiologist of the late 19th and early 20th century, who is responsible for the discovery of the Second Degree AV Block Type I and Type II

Woolf-Parkinson-White Syndrome (WPW):
pre-excitation syndrome characterized by the early depolarization of the ventricle(s) via an accessory pathway (other than the AV junction) resulting in an early initiation of a QRS and possibly a Delta wave; due to the presence of the accessory pathway, re-entry phenomena is possible with resulting rhythms such as atrial tachycardia; note that a Delta wave is present in only about 5% of people with WPW; those with a Delta wave and in atrial fibrillation or atrial flutter are at increased risk of sudden cardiac death - avoid medications that slow conductivity through the AV node such as digoxin, adenosine, calcium channel blockers and beta blockers

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