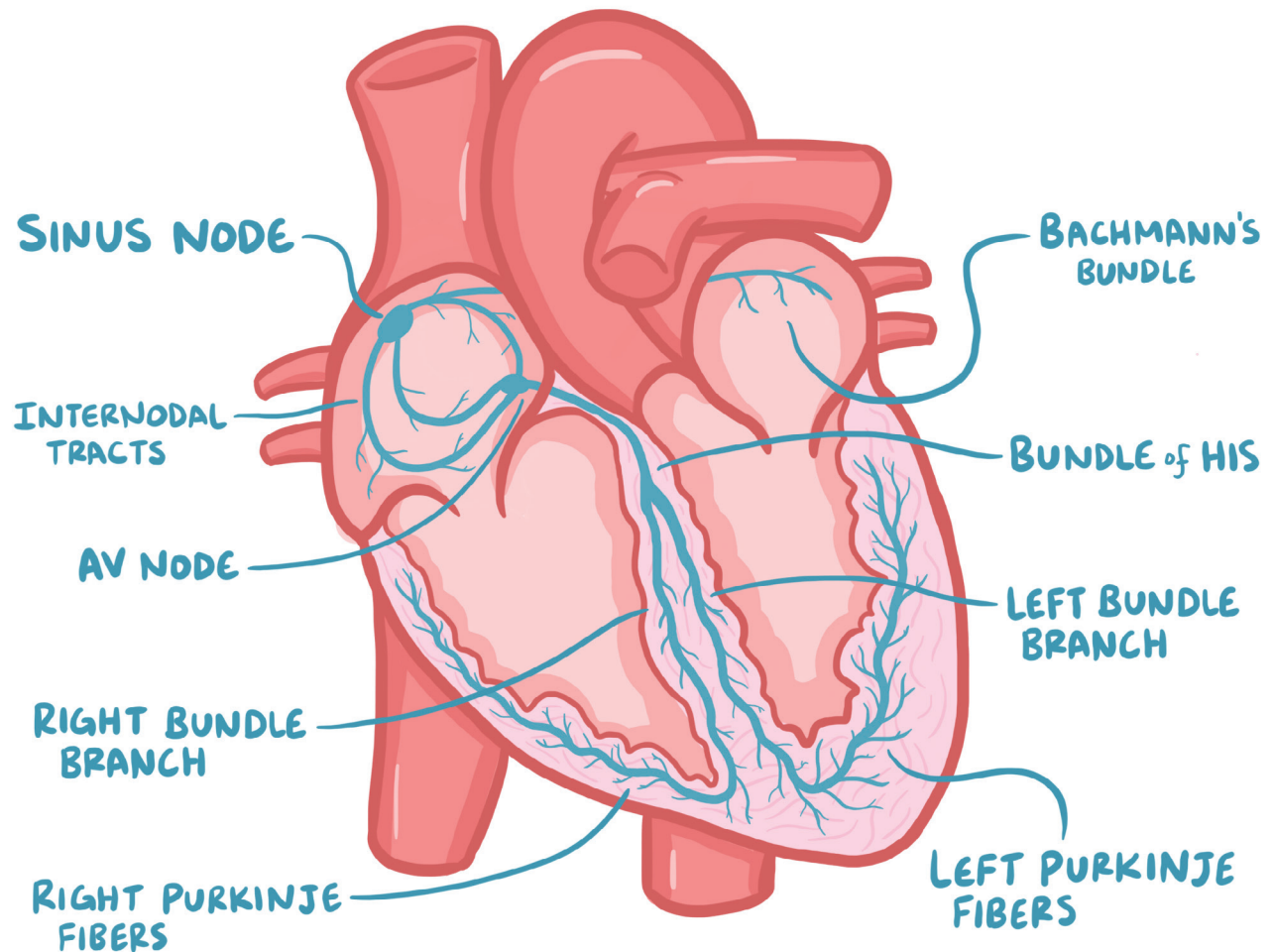


electrocardiography



ESSENTIALS



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ELECTROCARDIOGRAPHY ESSENTIALS

Edition: 1.03

The Osmosis Team

2021

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If you find any mistakes, let us know here:
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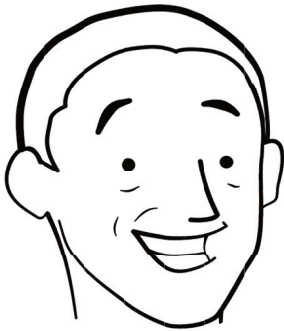


Fergus Baird
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Fergus Baird is a copywriter, editor and textbook designer at Osmosis, and dabbles in a little scriptwriting for the YouTube channel and textbooks as well.

Before moving to Canada, Fergus lived in a small village in Scotland. He earned his Master's degree in English literature at Concordia University in Montréal, writing his thesis on history and the graphic novel. Before joining Team Osmosis, Fergus worked as a gif curator, a movie subtitler, a meme master and a ghost writer, and spent a year teaching elementary school kids in Japan.

When he's not eating or cooking elaborate meals for his friends, Fergus spends his spare time playing video games and the theremin, drawing strange pictures, and consuming horror media in all its gruesome forms.



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Rishi is a pediatric infectious disease physician at Stanford University and serves as the Chief Medical Officer at Osmosis. He recently led the Khan Academy Medicine team, which put together a collection of videos and questions for students entering the health sciences.

With the help of his parents and teachers, Rishi completed high school and received his BS in Microbiology from UCLA by the age of 18. He completed his medical training at UCSF, pediatric residency at Boston Children's Hospital, and did an infectious disease fellowship at Children's Hospital Los Angeles. He earned his MPH in epidemiology at UCLA, and then spent two years chasing down infectious disease outbreaks for the Centers for Disease Control and Prevention, before beginning his work in online medical education.

In his spare time, Rishi enjoys watching his son torment the family dog, while eating nature's finest fruit—the raspberry!



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Tanner's the voice and illustrator behind the majority of Osmosis videos (as well as a few icons, graphics, and cartoons scattered around Osmosis). Tanner got both his undergrad and graduate degree in biomedical engineering from the University of Wisconsin-Madison (Go Badgers!), before moving out to Portland, OR and starting his engineering career.

After working in the cardiac rhythm management business for bit, he stumbled upon the two goofballs Rishi and Kyle, and began working alongside (well, technically under, but who's counting!) them producing openly accessible videos for Khan Academy Medicine.

In addition to being an enthusiast for science education and being halfway-decent with a drawing tablet, he also takes pride in his diverse collection of hats (including both beanies and baseball caps), his skills on the ultimate frisbee field, and his vast knowledge of the *Star Wars* universe.



Kyle Slinn
RN, MEd

Kyle is a project manager and instructional designer at Osmosis. He wears many hats, managing volunteers and contractors, designing future projects, producing videos, leading internationalization efforts, and designing Osmosis's textbooks. A jack-of-all-trades. Before Osmosis he worked as a project coordinator for Khan Academy Medicine where he managed the creation of videos, questions, and text-based articles for MCAT and NCLEX-RN students.

Kyle also worked in the pediatric intensive care unit as a nurse at the Children's Hospital of Eastern Ontario, in Ottawa, Canada. He received his Bachelors of Science in Nursing from the University of Ottawa and holds a Masters of Education in Distance Education from Athabasca University.

With his free time Kyle loves to go on adventures, hang out with friends, play board games, bake bread, and play musical instruments.

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Charlie writes content for Osmosis. He completed his BS in Biochemistry, Cell and Molecular Biology at Drake University. Next, he started a band and moved to New York City where he spent his days above ground as a research technician at Memorial Sloan Kettering Cancer Center, and his nights playing piano and ukulele in unfinished basements of Queens. He earned his MD at the University of Kansas School of Medicine and is currently a radiology resident at the University of Washington Medical Center.

Charlie believes that people are naturally curious and that it's the responsibility of educators to cultivate that curiosity with entertaining content presentations. In his free time, Charlie enjoys eating, reading science fiction, and performing random acts of kindness.

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Lyndsay copyedits for Osmosis. This means she helps scriptwriters convey meaning and tone through grammar, and she helps clarify content. Her favourite punctuation mark is the semicolon; the Oxford comma is a close second. As a teaching assistant, Lyndsay enjoys encouraging students to develop their own individual writing "voices." She is also working on a PhD in English Literature.

Lyndsay has enjoyed using her research and writing skills in new contexts while working for Osmosis. Reading scripts on a variety of topics has also given her a greater appreciation of how hard our bodies work to stay healthy.

In her free time, Lyndsay enjoys snuggling cats (particularly her sister Andrea's cat, Mortimer), doing crafts, and reading comic books.

ACKNOWLEDGMENTS

Creating accurate content is really hard. Thousands of eyeballs will look at our work for years to come and inevitably we'll have missed something, or something will go out of date. What's nice about our way of producing content is we get a lot of feedback really fast. Within 24 hours of releasing a video we've meticulously crafted and reviewed, we get thousands of views. In the rare instance there's a mistake, our audience is always quick to use the comment feature to point out where we went wrong. Yep, we're listening! We read every single comment across all of our videos. Whenever someone points out a factual inaccuracy, we update our video and rerelease it. Currently, YouTube doesn't allow editions of videos to exist. When we rerelease a video, we're effectively pulling the existing, well-established and easily searchable video out of circulation and putting a new, less searchable video in its place. There have been several times where our team has silently cried inside as we take down our most viewed video in the name of scientific accuracy. In the end, though, the result is that we can confidently say our videos (and textbooks) are factually accurate.

We'd like to thank the following people who have given us so much guidance as we've created our content.

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FOREWORD

Hi there! Welcome to Osmosis! I'm Shiv Gaglani, co-founder and CEO.

My co-founder Ryan and I started building Osmosis as medical students at Johns Hopkins. Despite access to world-class faculty, we knew there had to be a way to learn medicine more effectively once we left the classroom. What started as a simple learning app has evolved into the leading health education platform that empowers millions of current and future clinicians and caregivers with the best learning experience possible. .

Today, Osmosis is here to help you better understand and retain all of the information you need to succeed in your classes, on your exams, and in your career.

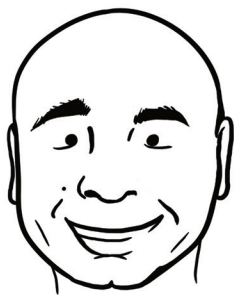
Being able to read an electrocardiogram is an essential skill for any clinician. Having a healthy heart means having a healthy heartbeat, and the best way to evaluate that is by looking at an ECG. We've designed this book to be a quick primer on the essentials of electrocardiography. By the time you've finished this book, you'll be familiar with the fundamentals of electrocardiography, and will be able to easily recognize the difference between a healthy QRS complex and an abnormal one, so that you can spot life-threatening conditions like ventricular fibrillation and myocardial ischemia.

While you may not be able to sleep with your head on this textbook and "learn by Osmosis," you can use the powerful & comprehensive Osmosis learning platform to watch our electrocardiography videos and actively quiz yourself with tens of thousands of associated multiple choice questions & flashcards.

As pioneers in medical and health education technology, Osmosis takes learning beyond textbooks and lectures by offering online educational video content that's simple, engaging, and informative. Osmosis.org has a library of over 2,000 videos covering pathology, physiology, pharmacology, and clinical practice, complete with questions, flashcards, and notes.

We encourage you to visit www.osmosis.org to learn more.

Best of luck Osmosing!



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CONTENTS

11	ELECTROCARDIOGRAPHY BASICS
17	NORMAL SINUS RHYTHM
27	AXIS
35	INTERVALS
41	QRS TRANSITION
44	CARDIAC HYPERTROPHY & ENLARGEMENT
49	CARDIAC INFARCTION & ISCHEMIA

57 SOURCES

59 INDEX

Electrocardiography Basics

TAKE NOTES
HERE!

osmosis.org/learn/ecg_basics

An electrocardiogram is also known as an ECG; the Dutch and German version of the word, *elektrokardiogram*, is shortened to EKG. It is a tool used to visualize, or “gram,” the electricity, or “electro,” that flows through the heart, or “cardio.” Specifically, a 12-lead ECG tracing shows how the depolarization wave, which is a wave of positive charge, moves during each heartbeat, by providing the perspectives of different sets of electrodes. This particular set of electrodes is called lead II; one electrode is placed on the right arm and the other on the left leg. Essentially, when the wave’s moving toward the left leg electrode, you get a positive deflection. This big, positive deflection corresponds to the wave moving down the septum (**Figure 1.1**).

To understand the basics, let’s start with an example of how we can look at the heart with only one pair of electrodes: a positive and a negative one (**Figure 1.2**; **Figure 1.3**). These electrodes detect the charge on the *outside* of the cell.

Remember, at rest, cells are negatively charged relative to the slightly positive outside environment; let’s make these cells red (**Figure 1.4**). When they depolarize, the cells become positively charged, leaving a slightly negative charge in the outside environment; let’s make these green (**Figure 1.5**). If we freeze this “wave of depolarization” as it’s moving through the cells, half the cells are negative, or depolarized, and half are positive and resting, so there’s a difference in charge across this set of cells (**Figure 1.6**). You can think of the charge difference as a *dipole*, because there are two electrical poles. We can draw this dipole out as an arrow, or vector, pointing towards the positive charge (**Figure 1.7**). The electrodes detect charge on the *outside* of the cell, so this points toward where the positive charge is, outside (**Figure 1.8**).

Figure 1.1

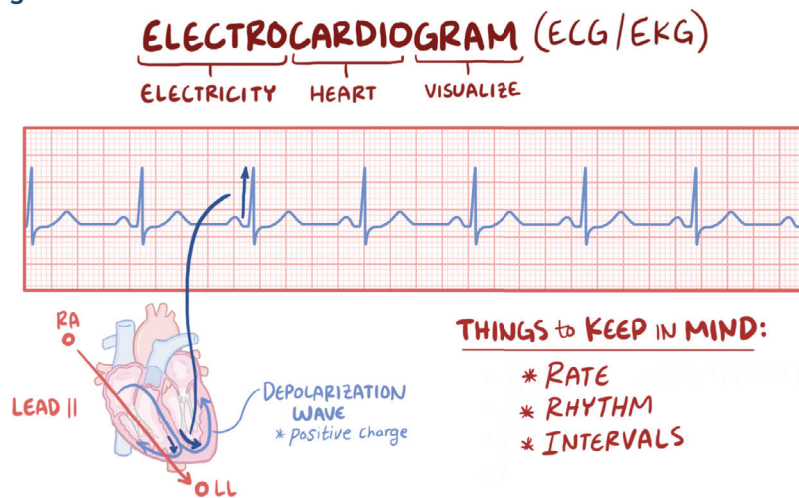


Figure 1.2

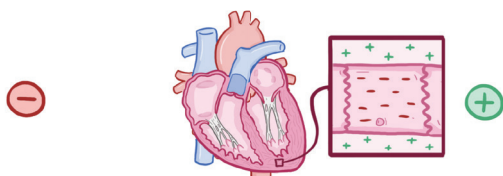


Figure 1.3

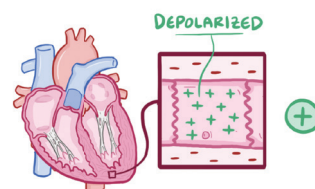


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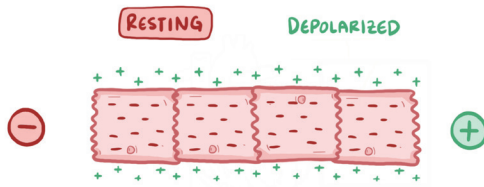


Figure 1.5

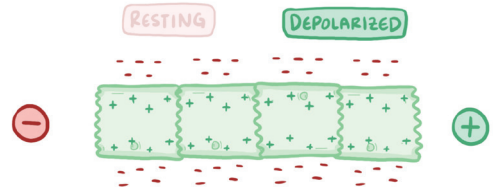


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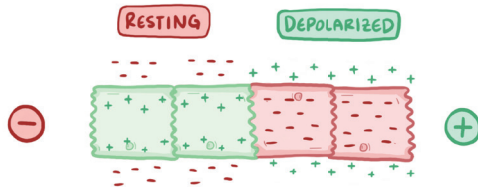


Figure 1.7

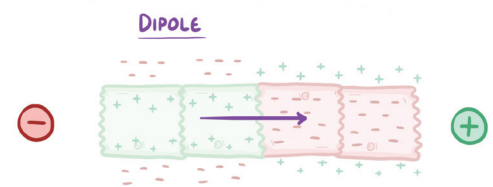


Figure 1.8

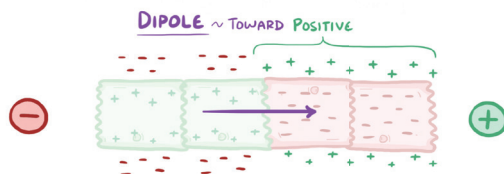


Figure 1.9

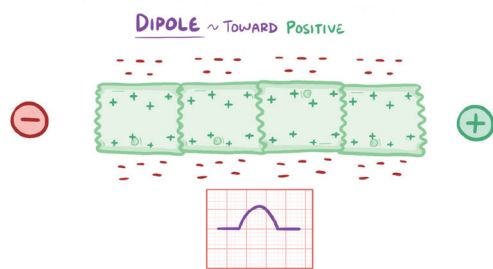


Figure 1.10

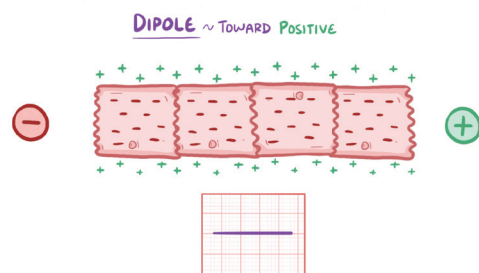


Figure 1.11

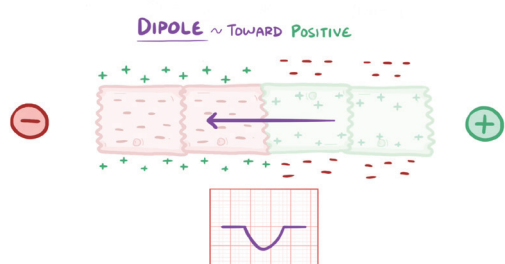


Figure 1.12

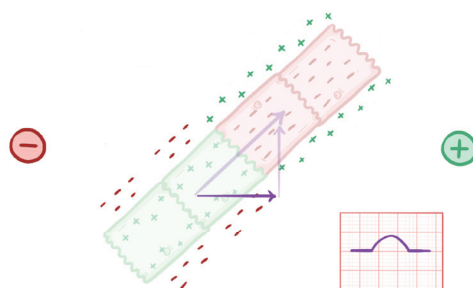
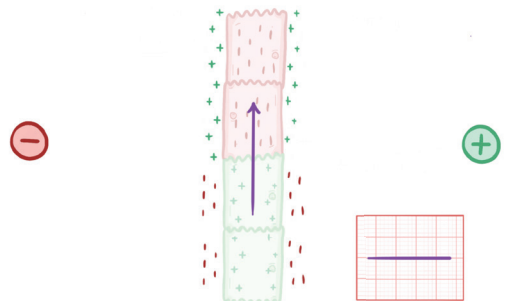


Figure 1.13



DEPOLARIZATION & DIPOLE VECTORS

Now, if there's a dipole vector pointing toward the positive electrode, then the ECG tracing shows it as a positive deflection; the bigger the dipole, the bigger the deflection (**Figure 1.9**). If we unpause this, then everything becomes depolarized. Since there's no *difference in charge*, there's no dipole, and thus no deflection (**Figure 1.10**). Moments later a wave of *repolarization* goes through and the cells become negative once again. Pausing halfway through again, now the dipole vector goes in the opposite direction and faces the negative electrode; this means that there will be a negative ECG tracing (**Figure 1.11**). Again, the bigger the dipole is, the bigger the negative deflection is.

Even though it'd be nice if the depolarization wave lined up perfectly with the electrodes, usually that's not the case. So, we simply look at the vector component parallel to that electrode. For example, let's say the depolarization happened this way, at an angle; then, we'd simply break the vector into two parts (**Figure 1.12**). The one we care about is going towards the positive electrode, which causes a deflection, even though it's a slightly smaller deflection than previously. In other words, the size of the deflection on the ECG tracing always corresponds to the magnitude, or size, of the dipole in the direction of the electrode. The perpendicular component isn't pointing at the electrodes, so it doesn't cause any deflection. In fact, if there's a depolarization wave that goes straight up, perpendicular to the positive and negative electrodes, there would be no deflection (**Figure 1.13**)!

LIMB & CHEST LEADS

In a standard ECG, there are 10 electrodes: four limb electrodes, with one each on the left arm, right arm, left leg, and right leg; and six precordial electrodes, V_1 through V_6 , that wrap around the chest. The right leg electrode is usually used as a neutral lead. The heart is a three-dimensional organ, so V_1 through V_6 line up in the transverse, or horizontal, plane of the heart. Each electrode is set up to detect any wave of positive charge coming towards it. These are collectively called the chest leads (**Figure 1.14**).

Figure 1.14

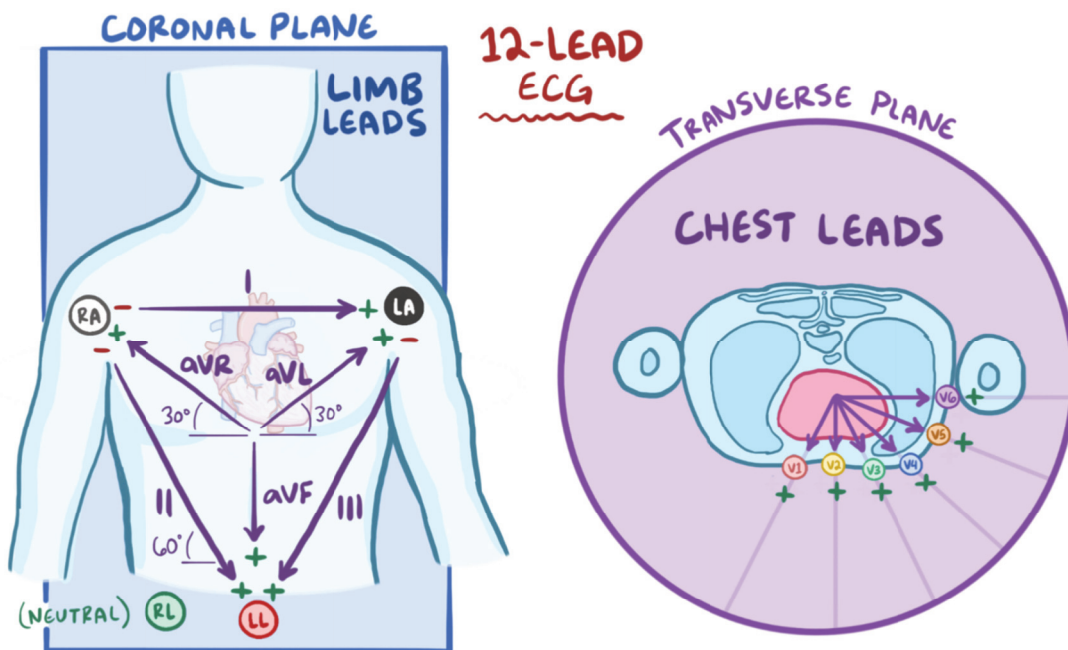


Figure 1.15

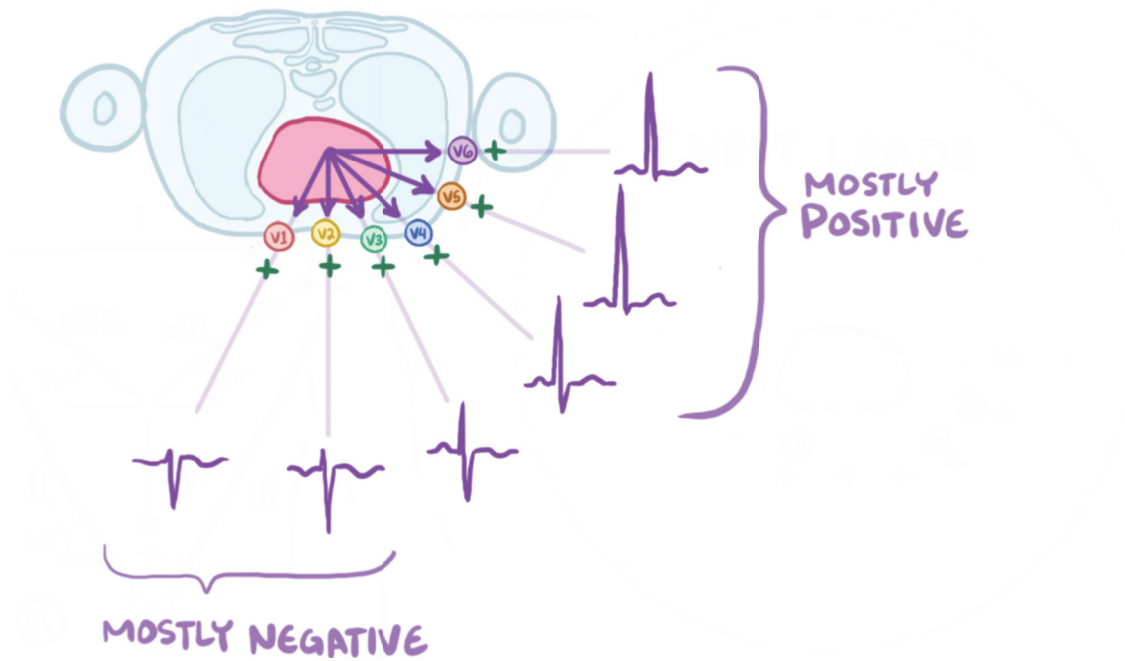


Figure 1.16

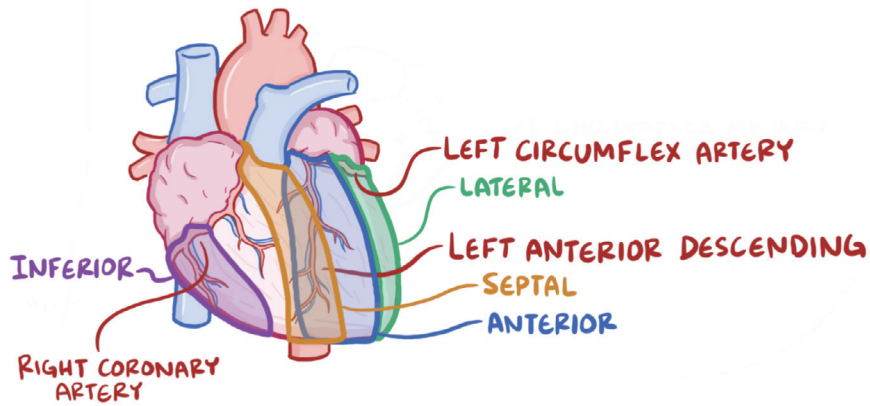
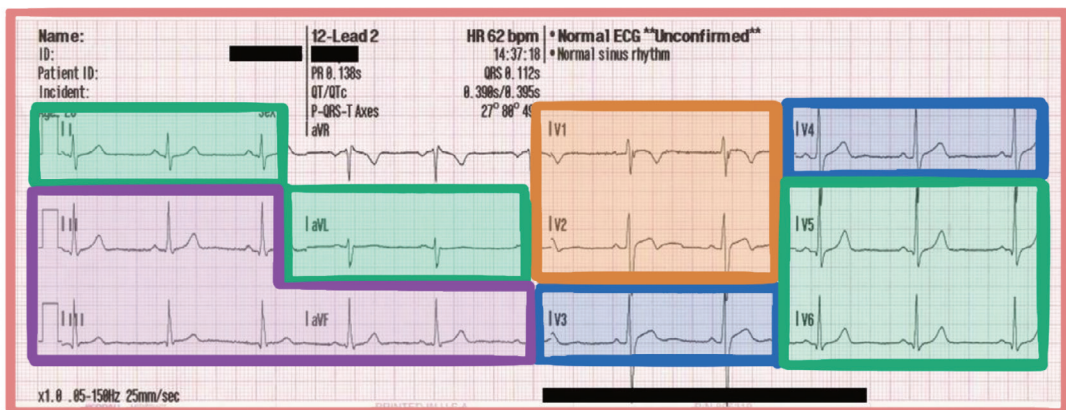


Figure 1.17



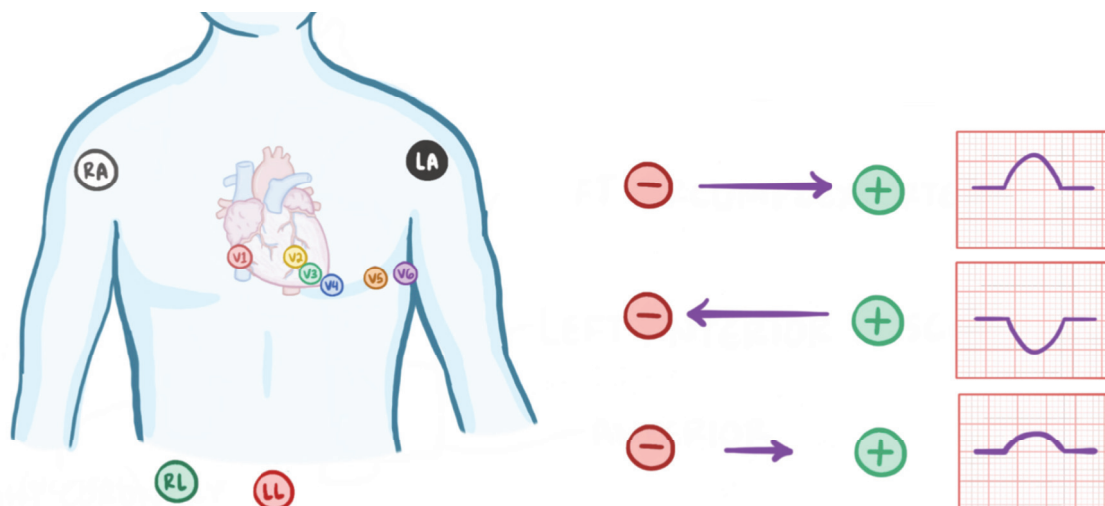
Meanwhile, in the coronal plane, the non-neutral leads are called augmented vector right, or aVR, on the right arm, and augmented vector left, or aVL, on the left arm. Both of these leads are represented as vectors that are 30 degrees up from the horizontal line. Augmented vector foot, or aVF, is on the left foot, which anatomically isn't straight down, but it's close enough that it ends up representing the vector facing straight down on the diagram. Just like the precordial electrodes, aVR, aVL, and aVF detect any positive deflection coming towards them. Now, in addition to these three limb leads, there are also bipolar limb leads called lead I, lead II, and lead III, which are recorded using two electrodes instead of just one. Lead I uses the RA as the negative pole and LA as the positive pole, forming a vector that goes to the left. Lead II uses aVR as the negative pole and aVF as the positive pole, forming a vector that goes to the +60 degree mark. Finally, lead III uses aVL as the negative pole and aVF as the positive pole, forming a vector that goes to the +120 degree mark. So, in total you've got six leads from the limb leads and six from the chest leads, which add up to 12 leads total; in other words, you have a 12-lead ECG.

THE VALUE OF DIFFERENT VIEWPOINTS

Having different views of the heart is important, because they make it possible for you to see how the wave of depolarization moves through the heart according to each viewpoint. For example, consider how the six chest leads, V₁ through V₆, register the depolarization wave form called the QRS complex. The very same depolarization wave might appear mostly negative in V₁ and V₂, isoelectric in V₃, and mostly positive in V₄, V₅, and V₆, because the exact direction and magnitude of the vectors are at different points in time. Similarly, each of the frontal plane leads produces its own perspective of the depolarization wave (**Figure 1.15**).

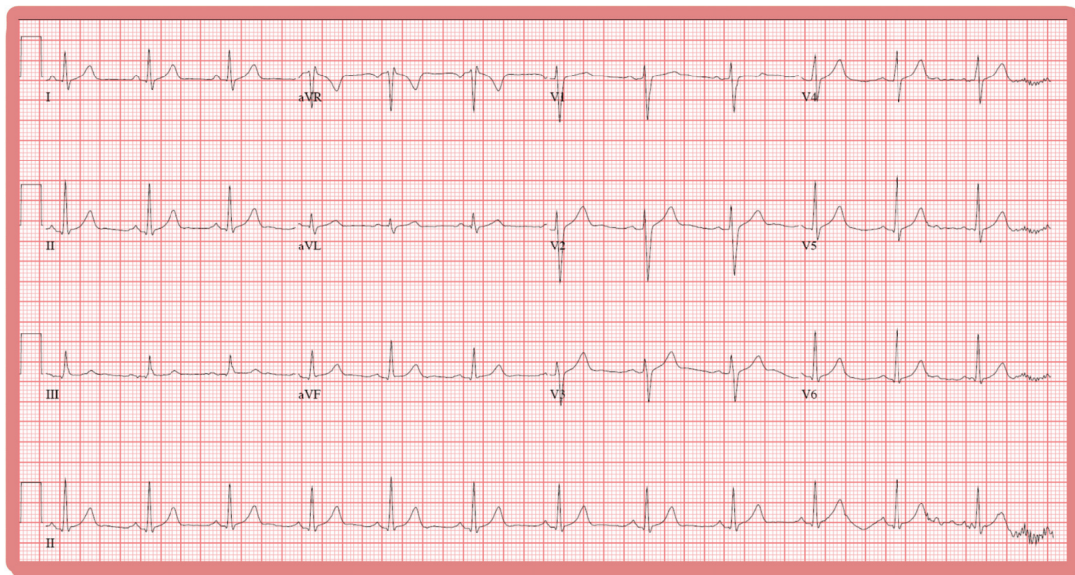
The limb leads and chest leads can be grouped based on the regions of the heart that they are nearest, as we've indicated in this color-coded chart (**Figure 1.16; Figure 1.17**). Problems in specific leads, or groups of leads, suggests that there may be a specific region of the heart affected by a disease. Leads II, III, and aVF are "inferior" leads because they're near the inferior wall of the heart, which receives blood from the right coronary artery. Leads I and aVL, along with two of the chest leads, V₅ and V₆, are considered "lateral" leads because they're near the lateral wall of the heart, which receives blood from the left circumflex artery. Finally, V₁ and V₂ are considered "septal" leads because they're nearest to the interventricular septum, and V₃ and V₄ are "anterior" leads because they're nearest the anterior wall of the heart. Both of the septal and anterior regions are served by the left anterior descending artery.

Figure 1.18 SUMMARY



All right, let's quickly recap. In a standard ECG there are 10 electrodes: four limb electrodes and six precordial electrodes that wrap around the chest. These electrodes are used to make 12 leads, each of which illustrates the movement of positive charge on the outside of heart cells. The ECG tracing shows a depolarization wave moving towards an electrode as a positive deflection, and one moving away as a negative deflection; each is proportional to the size of the dipole. The benefit of being able to see different views of the heart is that it makes it easier to see how the wave of depolarization moves, which provides valuable information about the heart's structure and function (**Figure 1.18; Figure 1.19**).

Figure 1.19



Normal Sinus Rhythm

osmosis.org/learn/ecg_normal_sinus_rhythm

To read an ECG, it's important to first understand what a normal sinus rhythm looks like (Figure 2.1). To do that, let's look at a single heartbeat on an ECG from the viewpoint of lead II.

DEPOLARIZATION

In a healthy heart, everything starts at the sinoatrial node, or SA node, which is a little patch of tissue in the wall of the right atrium full of pacemaker cells. When one of these pacemaker cells depolarizes, a wave of positive charge spreads outward; overall, it moves from the SA node towards the apex of the heart, so it aligns nicely with the lead II vector (Figure 2.2).

Figure 2.1

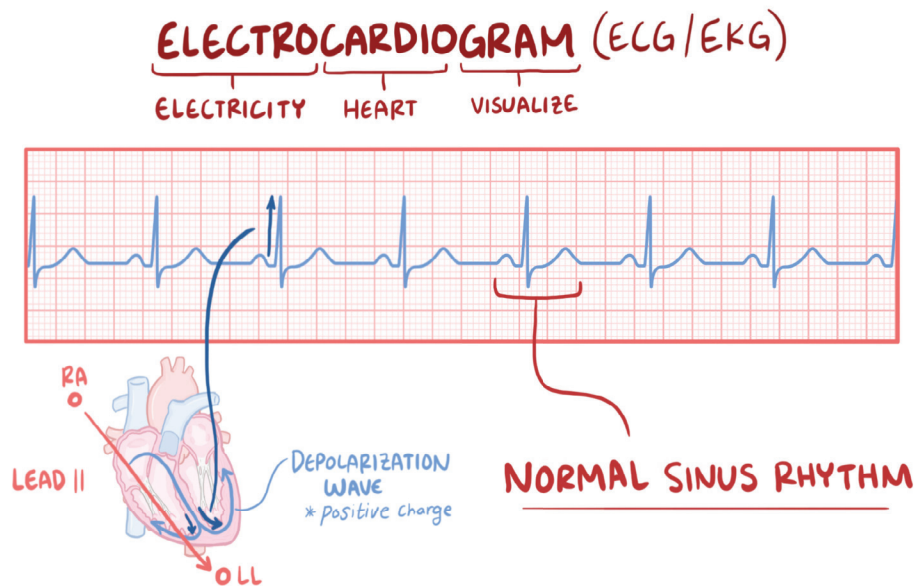


Figure 2.2

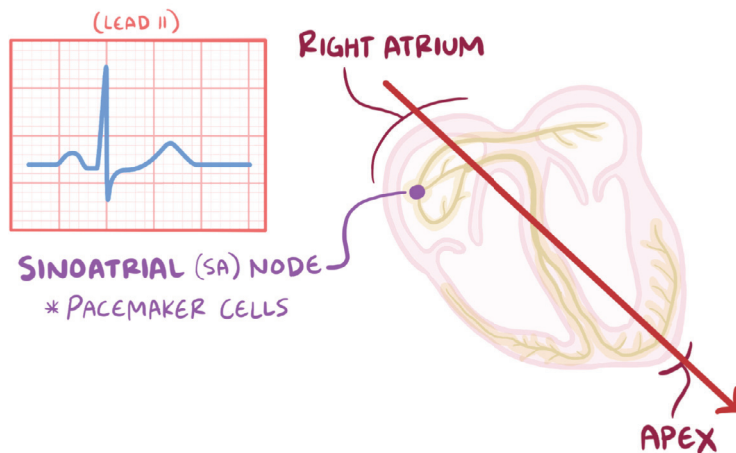


Figure 2.3

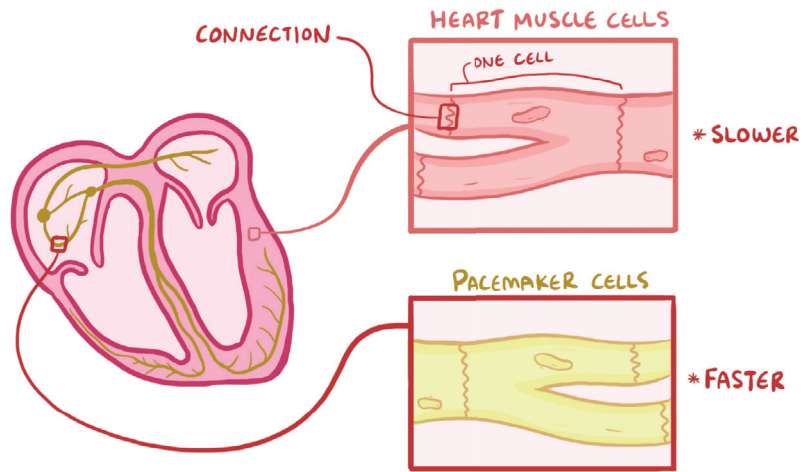


Figure 2.4

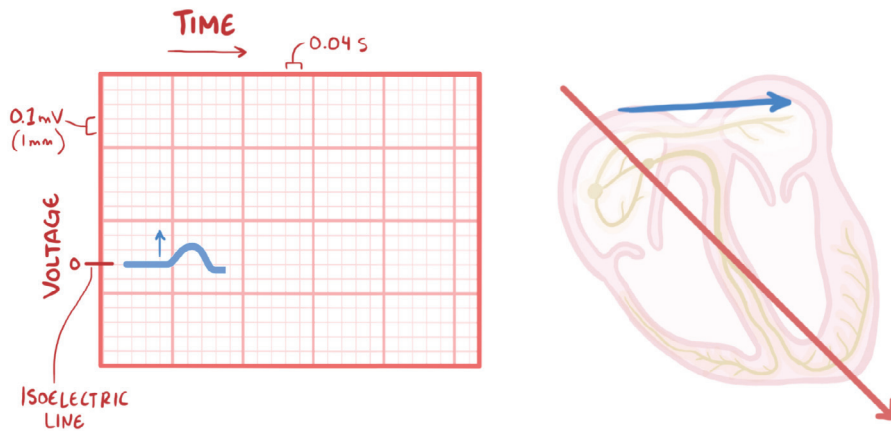
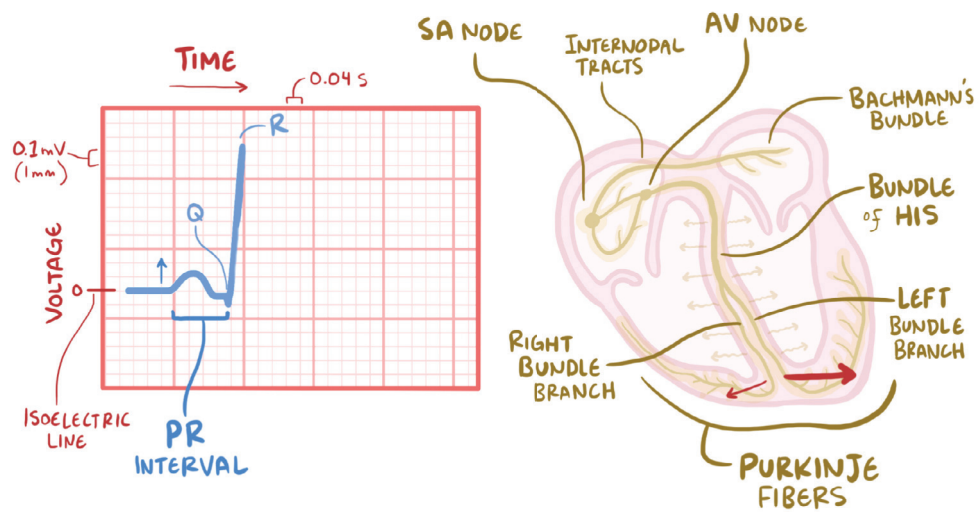


Figure 2.5



Now, the heart muscle cells are often described as having a functional syncytium because even though each one cell has its own cell membrane, the cells also have tiny connections, or openings, between them. That means that during a depolarization wave ions can flow right from one cell to the next. It's also important to note that the depolarization wave moves at two different speeds through the heart. In the pacemaker cells, which are special types of cardiomyocytes laid out like highways through the heart, it moves really fast. It moves more slowly through the rest of myocytes that do the contracting; the depolarization wave moves through these cells like a car travelling through small, congested streets (**Figure 2.3**).

READING DEPOLARIZATION ON AN ECG

The ECG measures out changes in time on the X-axis, where one small box is 0.04 s, and voltage on the Y-axis, with each small box equal to 0.1 mV, which is sometimes called 1 mm voltage. Zero is called the "isoelectric line." Every time there's positive voltage, there's an upward deflection *above* the isoelectric line, and every time there's negative voltage, there's a downward deflection *below* the isoelectric line. So, a depolarization wave starts in the SA node, then goes through *atrial intranodal tracts*, which is also called Bachmann's bundle, and travels over to the left atrium so that both atria basically depolarize together. The overall direction of that depolarization wave is in the same direction as the lead II vector, so that's considered positive voltage, and there's a positive deflection called the P wave (**Figure 2.4**).

Meanwhile, the signal also gets carried from the SA node to the atrioventricular node, or AV node; here, it gets delayed for a bit, which allows the atria to fully contract and fill the ventricles with blood. During this delay, there is no depolarization wave moving towards or away from lead II, so this appears as a flat line. The interval from the beginning of the P wave through this flat portion is called the PR interval. From there, the signal goes through the pacemaker cells that make up the bundle of His, and into the left bundle branch and right bundle branches. In addition to moving through the fast pacemaker cells, the signal also travels through the slow myocytes in the interventricular septum; this direction is slightly away from the lead II vector, because there is a lot of this tissue. This creates a tiny negative deflection on the ECG: a Q wave (**Figure 2.5**).

From there, the depolarization wave flows down into the Purkinje fibers. Since the largest vectors are the ones in the left ventricle, this is in the direction of lead II; this creates a large positive deflection on the ECG: an R wave. The apex of the heart depolarizes first, but then the wave moves back up to depolarize the top of the ventricles, which is in a direction away from lead II; this causes a slight negative deflection on the ECG: an S wave. Together, ventricular depolarization creates what is called the QRS complex (**Figure 2.6**).

Figure 2.6

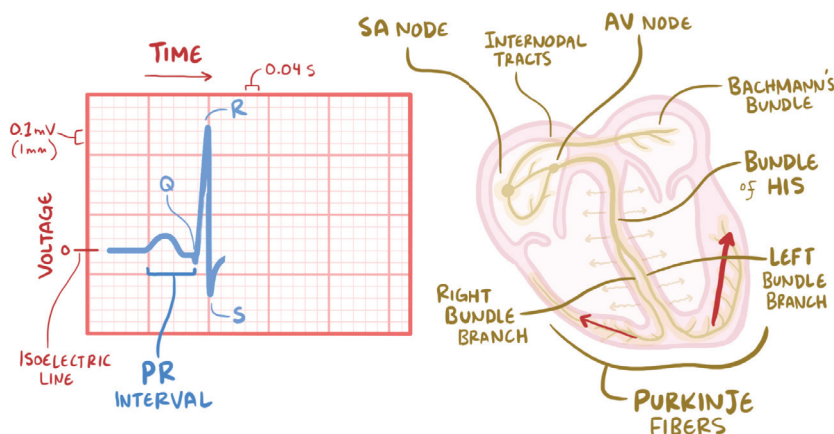
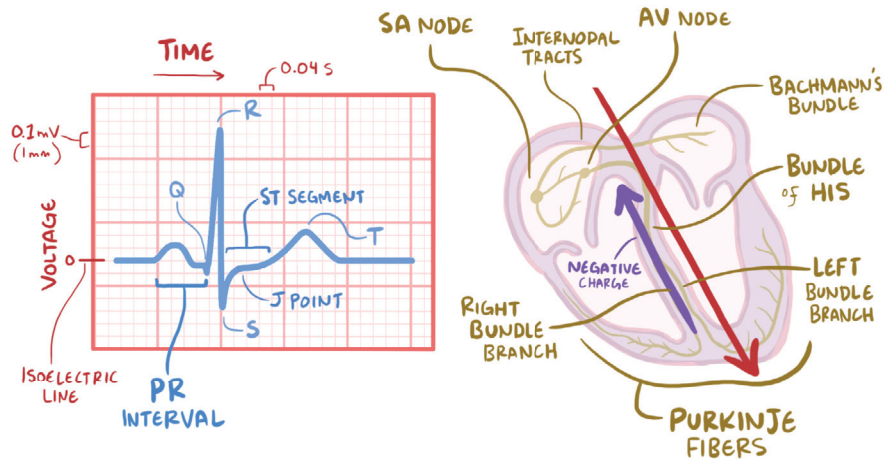


Figure 2.7



After tissue depolarizes, there is a brief period of time when there is no change in electrical activity; thus, the ECG goes back to the isoelectric line. The exact point at which it hits that line is called the J point. For a brief time, there is no net change in electrical activity; this is the ST segment. After that, the ventricles have to repolarize or become negative once more in order to reset themselves in preparation for another wave of depolarization.

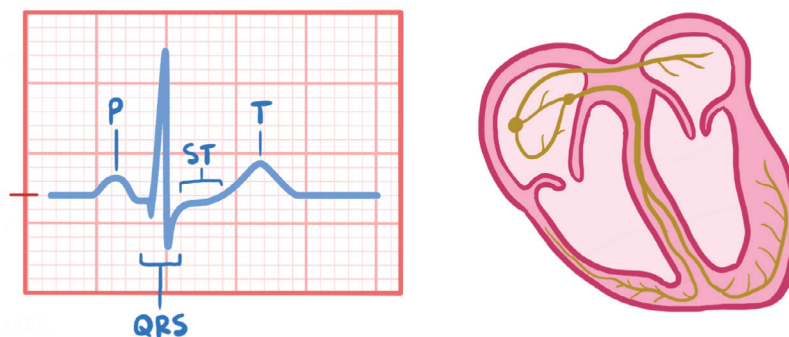
Now, *repolarization* moves in an overall direction upward, opposite the direction of lead II. You might think that it should be a negative deflection, but really it's a *positive* deflection, because this time it's a wave of *negative* charge; essentially, now when it's moving opposite to lead II, it's a positive deflection on ECG. This final wave is called the T wave (**Figure 2.7**).

The T wave is more spread out over time than the QRS complex because repolarization is a slower process that takes place at slightly different times for each cardiomyocyte, instead of happening all at once, as with depolarization. It's also worth mentioning that atrial repolarization doesn't usually show up at all on the ECG because the small vectors it creates get lost in much larger vectors created by the the QRS complex.

SUMMARY

All right, let's quickly recap. On an ECG, there's a P wave, which corresponds to the atrial depolarization wave, followed by an isoelectric line, when the depolarization wave goes through the AV node. After that, there's a QRS complex, which represents ventricular depolarization, followed by another brief isoelectric pause, called the ST segment. Finally, there's a T wave, which represents ventricular repolarization (**Figure 2.8**).

Figure 2.8



Rate & Rhythm

osmosis.org/learn/ecg_rate_and_rhythm

To read an ECG, there are a few key elements to keep in mind; one is to figure out the rate and rhythm (**Figure 3.1**). There are a couple of ways to figure out the heart rate on an ECG.

THE BOX METHOD

The first is called the “box method,” because you count the number of boxes between heartbeats. Each small box represents 0.04 seconds, and each big box is made up of five small boxes, so each big box is 0.2 seconds. To use this method, you count the number of small boxes between R waves, because R waves are tall, pointy, and easy to see in lead II of an ECG strip. You can find an R wave that has a peak that falls at the beginning of a box, and then count up how many boxes until the same point on the next R wave. Let’s say that there are four big boxes and one and a half small boxes between two R waves. In other words, there are $4 \times 5 + 1.5 = 21.5$ small boxes. This means that there’s $0.04 \text{ seconds} \times 21.5$, or .86 seconds, between heartbeats. Now, to get a result that’s a little more meaningful, we can take the inverse, which is one over 0.86 beats per second, or 1.16 beats per second. Now, there are 60 seconds in a minute, so if we multiply that by 60, we end up with 70 beats per minute—the heart rate (**Figure 3.2**)!

Now, if the distance between two R waves is exactly one big box, then the heart rate would be 300 beats per minute, which is *really* fast. If R waves are two big boxes apart, or 0.4 seconds apart, then the heart rate is 150 beats per minute. Three big boxes apart is 100 beats per minute, and four, five, and six big boxes apart is 75, 60, and 50 beats per minute respectively. Remembering these numbers makes it easier to make a rough estimate. For example, if there are three to four large boxes between R waves, then the heart rate must be between 75 and 100 beats per minute (**Figure 3.3**).

Figure 3.1

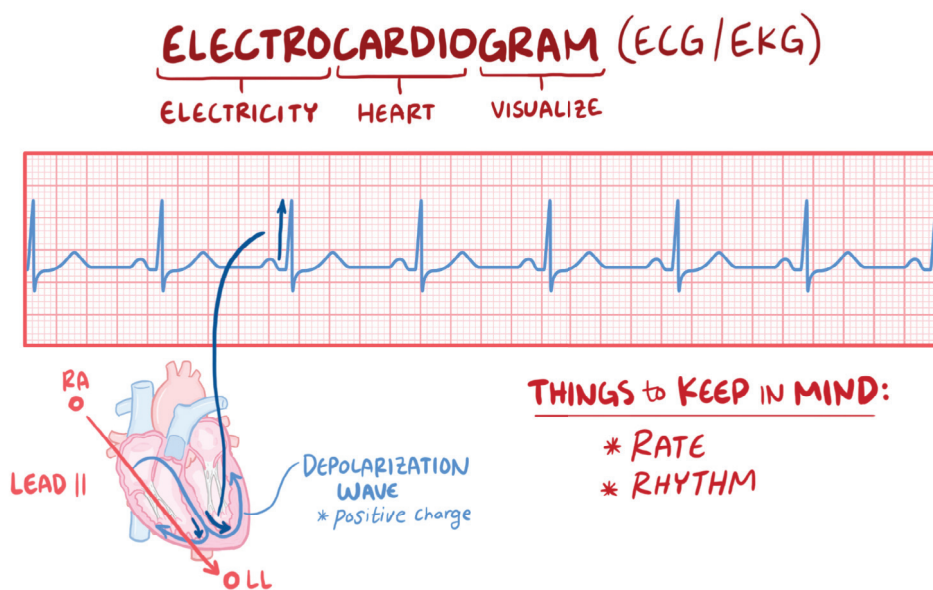


Figure 3.2

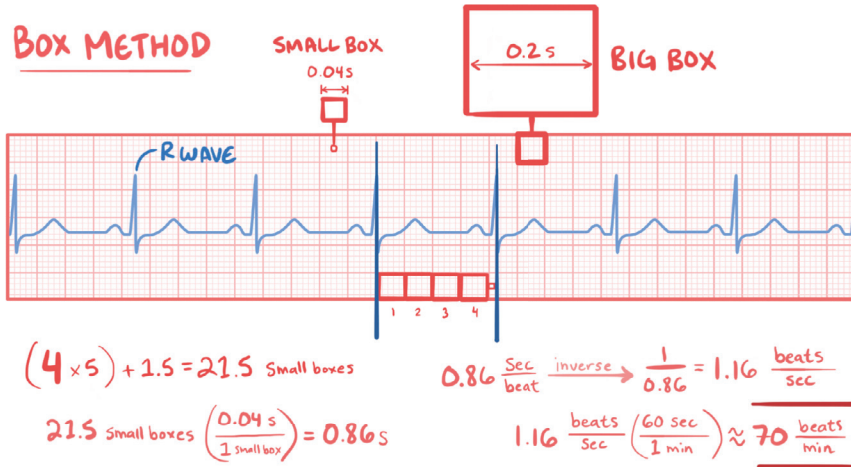


Figure 3.3

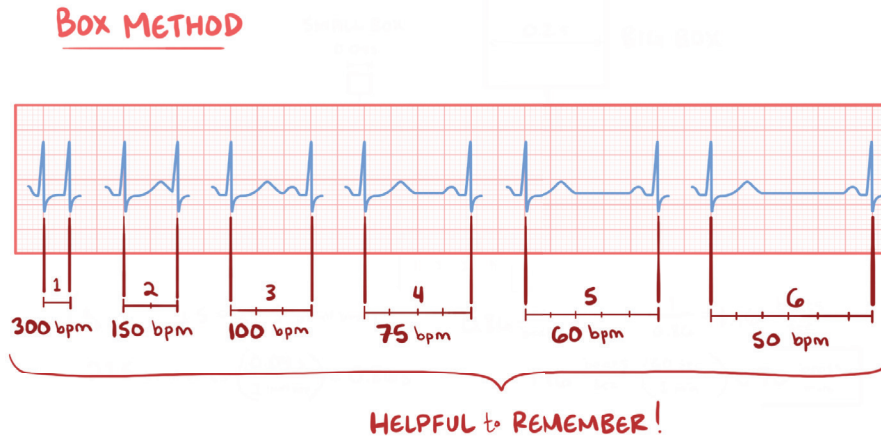


Figure 3.4

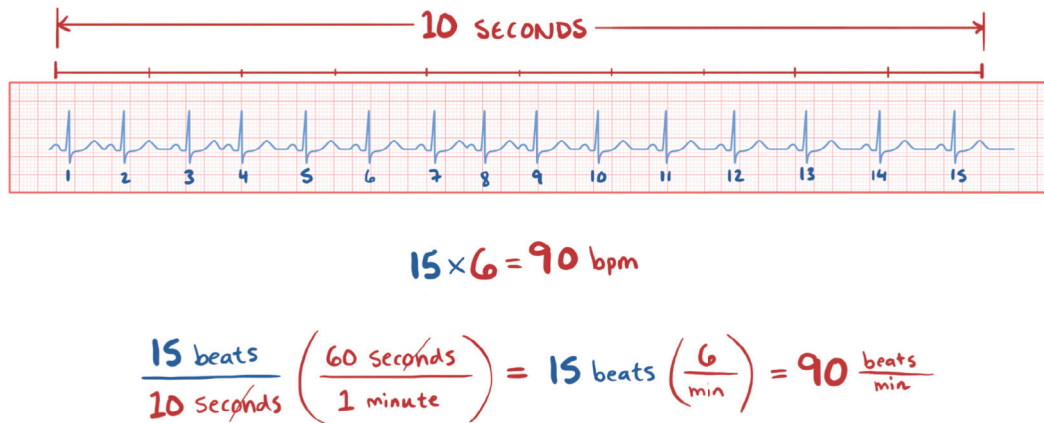
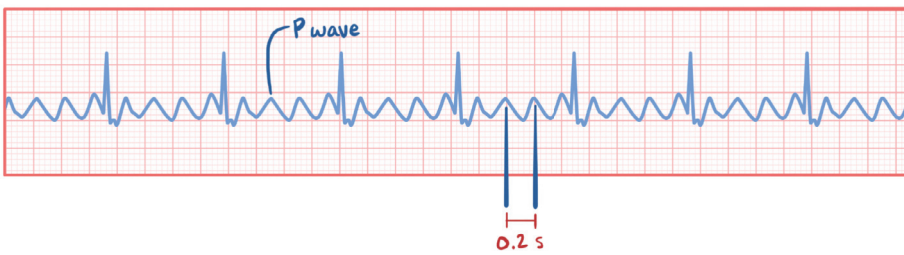


Figure 3.5

ATRIAL FLUTTER ~ ECTOPIC FOCUS in ATRIA
 * ATRIAL RATE 250-350 bpm



$$\frac{1 \text{ beat}}{0.2 \text{ sec}} \left(\frac{60 \text{ sec}}{1 \text{ min}} \right) = 300 \text{ bpm} = 1 \text{ BIG BOX}$$

NUMBER OF BEATS PER 10 SECONDS

Counting the number of beats in 10 seconds, the standard length of time on the rhythm strip portion of a 12-lead ECG is another method you can use to determine heart rate. In this case, we've got 15 beats. All you need to do is multiply this by six to get the heart rate, which would be 90 beats per minute. The reason this little trick works is that you've got 15 beats per 10 seconds, and again, to convert to *per minute* you multiply by 60 seconds in a minute. Looking at this, we see that $60 / 10 = 6$ per minute. So, $6 \times 15 = 90$ beats per minute (**Figure 3.4**).

IRREGULAR HEART RATES & RHYTHMS

If the heart rate is too slow or too fast, it could be because something other than the SA node is pacing the heart rate. For example, there could be atrial flutter, which is when an ectopic focus in the atria, such as an irritated atrial cell, starts to spontaneously fire between 250-350 depolarizations per minute; then, only one out of every few atrial depolarizations passes through to the ventricles.

To calculate the atrial rate, you can use the same method as before, except you look at P waves instead of R waves. If one P wave begins on a heavy line, and the next P wave begins on the next heavy line, or 0.2 seconds later, then again, you've got one beat for every 0.2 seconds; multiplying by the 60 seconds in a minute, you get 300 beats per minute. You could also remember that one big box is equal to 300 bpm (**Figure 3.5**).

Another situation is atrial fibrillation, which is when there are multiple ectopic foci in the atria that start firing all at once; the atrial rate can increase to 350-450 beats per minute. In this situation, only the occasional firing of an ectopic focus that happens to be near the AV node is able to make it through and down to the ventricles. The ectopic foci fire too quickly for the atria to be fully depolarized by any one of them, so there aren't any P waves on an ECG; rather, there are small waves that reflect waves of atrial depolarization that couldn't make it through the AV node. Of note, the 350-450 beats per minute reflects all the times the atrial foci fire per minute, and not the rate of ventricular depolarization. With atrial fibrillation, the ventricular rate is much lower than the atrial rate, but still over 120 beats per minute. It's also highly irregular, meaning that the distances between the QRS complexes are not equal (**Figure 3.6**).

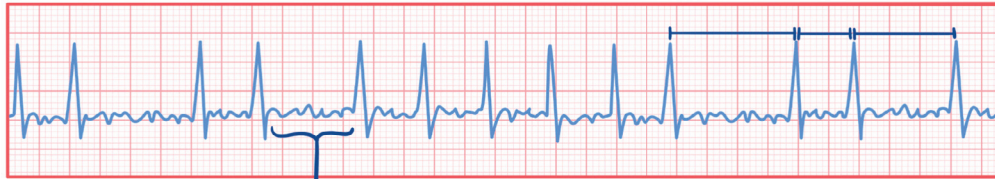
Normally on an ECG, one waveform's P wave, QRS complex, and T wave look just like the next one's, almost like they were copied and pasted one right after another. That's how a regular rhythm looks; the heart moves like a smooth dancer on rhythm. An irregular rhythm, on the other hand, is when there's any change in the timing of the QRS complexes (**Figure 3.7**).

Figure 3.6

ATRIAL FIBRILLATION ~ MULTIPLE ECTOPIC FOCI

* ATRIAL RATE 350-450 bpm

* VENTRICULAR RATE >120 bpm + HIGHLY IRREGULAR

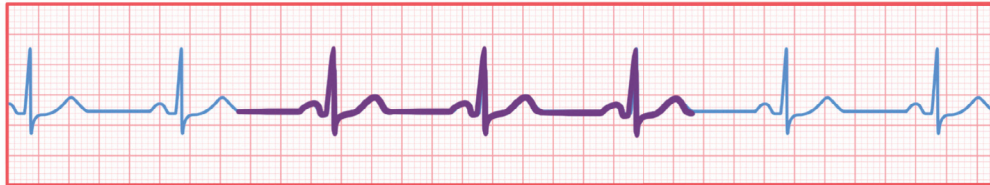


NO P WAVES

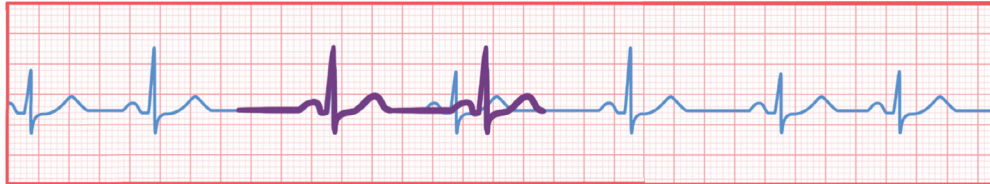
↳ SMALL WAVES of ATRIAL DEPOLARIZATION

Figure 3.7

*** REGULAR RHYTHM ***



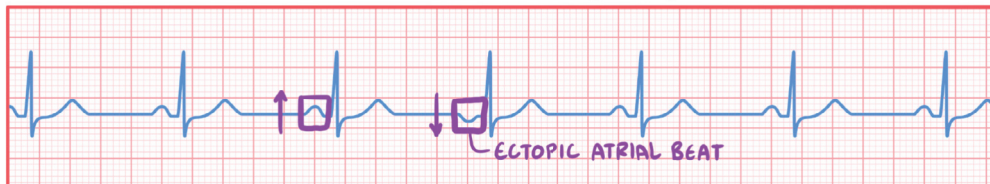
*** IRREGULAR RHYTHM ***



~ ANY CHANGE in TIMING of QRS COMPLEXES ~

Figure 3.8

*** ECG ABNORMALITIES ***



* DOES EVERY PART LOOK the SAME?

- ↳ P WAVE?
- ↳ QRS COMPLEX?
- ↳ T WAVE?

Figure 3.9

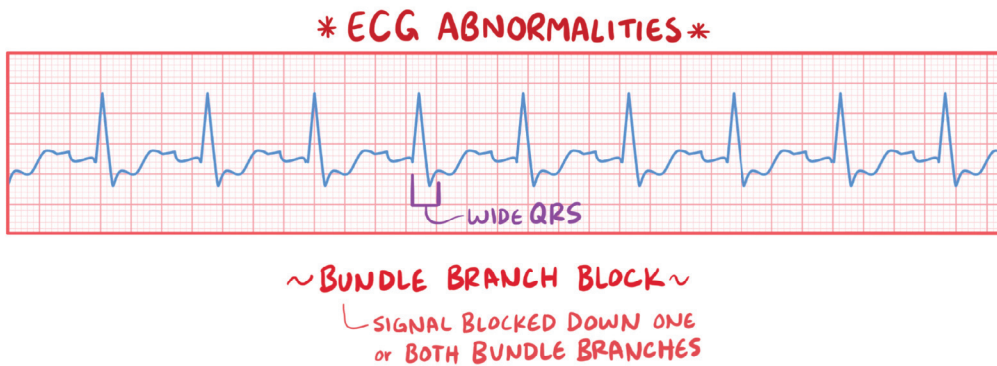
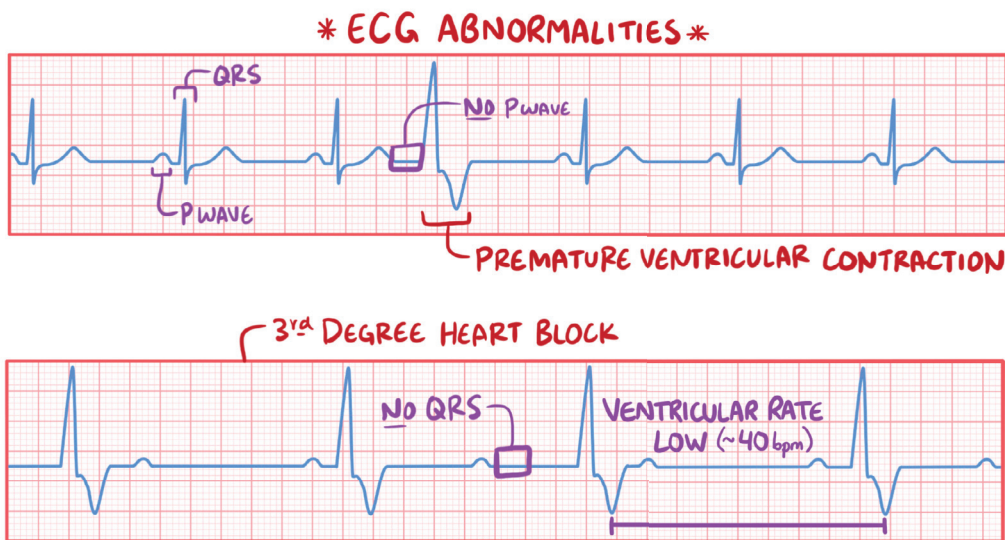


Figure 3.10



To help identify ECG abnormalities, it helps to ask if every part of the waveform, including the P wave, QRS complex, and T wave, looks exactly the same as the others. If not, it could mean that there's an ectopic beat, which means that it may have originated from an abnormal spot in the atria or ventricles. For example, this first P wave is deflected upwards and the second is deflected downward, which indicates that it's an ectopic beat (**Figure 3.8**).

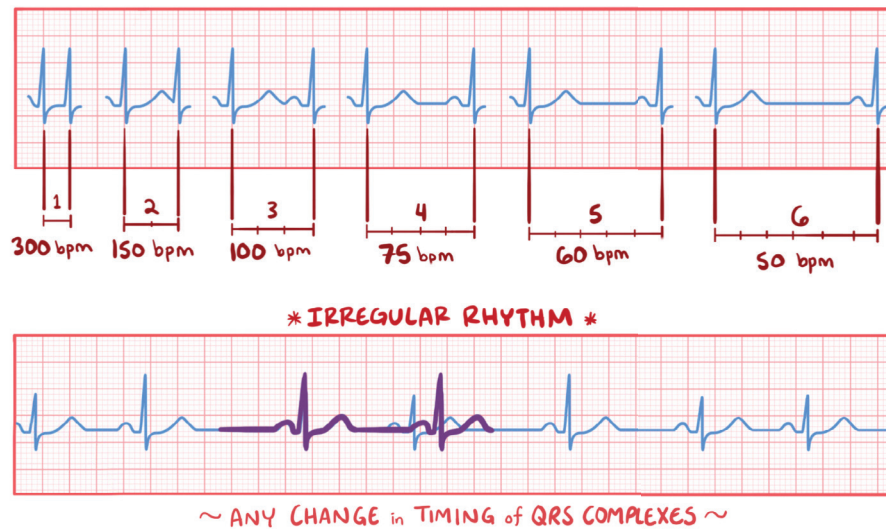
Alternatively, an odd-looking waveform may have originated from the normal spot, the sinoatrial node, but was thrown off course; this is what happens when there's a block somewhere. In this example, there's a bundle branch block, which describes when the signal can't go down one or both of the bundle branches; this usually results in these wide QRS complexes (**Figure 3.9**).

Next, check for changes in the sequence of the waves; changes most often involve the two depolarization waves, the P wave and the QRS complex. You can make sure that there is a P wave before every QRS complex, and a QRS complex after every P wave. For example, if there's a premature ventricular contraction like this, then there may be a QRS complex *without* a preceding P wave. On the flip side, if there's a third degree AV block, the atria and ventricles have completely separate electrical activity; kind of like two people dancing to different songs, at the same time. So in this case, there can be P waves that are not followed by QRS complexes, and the ventricular rate is usually low, around 40 beats per minute. (**Figure 3.10**).

SUMMARY

All right, let's quickly recap. One quick way to estimate the heart rate on an ECG is to remember that the heart rate is 300, 150, 100, 75, 60, or 50, depending on whether there's one, two, three, four, five, or six boxes between QRS complexes. To help identify an irregular rhythm, you can look at the waveforms and see if there is any variation in the timing of the QRS complexes (Figure 3.11).

Figure 3.11



Axis

osmosis.org/learn/ecg_axis

To read an ECG, there are a few key elements to keep in mind; one is to figure out the axis (Figure 4.1). The axis of an ECG is the average direction of electrical movement through the heart during a depolarization. Specifically, axis usually refers to the mean QRS vector, which is the size and direction of the depolarization wave as it moves through the ventricles. Normally, the QRS axis aims downward and to the left in relation to the body.

DEPOLARIZATION

So, if we simplify this heart a bit, normally, the sinoatrial node (SA node) sends an electrical signal that propagates outward through the walls of the heart and contracts both upper chambers (Figure 4.2). Then, that signal moves to the atrioventricular (AV node) where the signal is delayed for a split second, and then goes down into the ventricles, or lower chambers. Here, it moves down the bundle of His into the left and right bundle branches, and into each ventricle's Purkinje fibers, which causes them to contract as well (Figure 4.3).

Figure 4.1

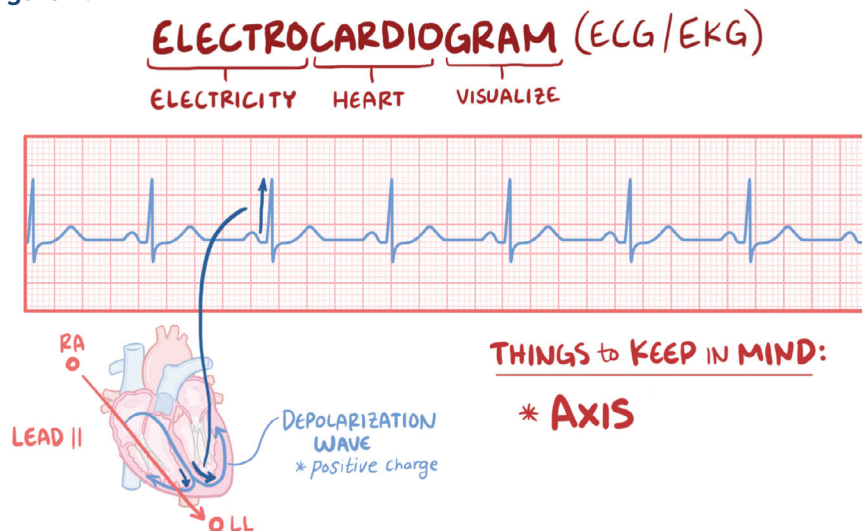


Figure 4.2

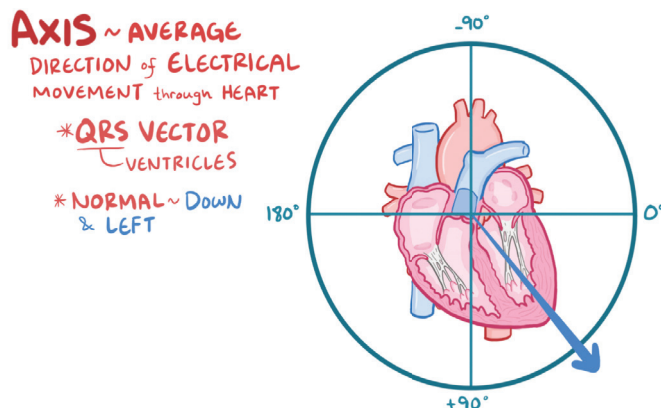


Figure 4.3

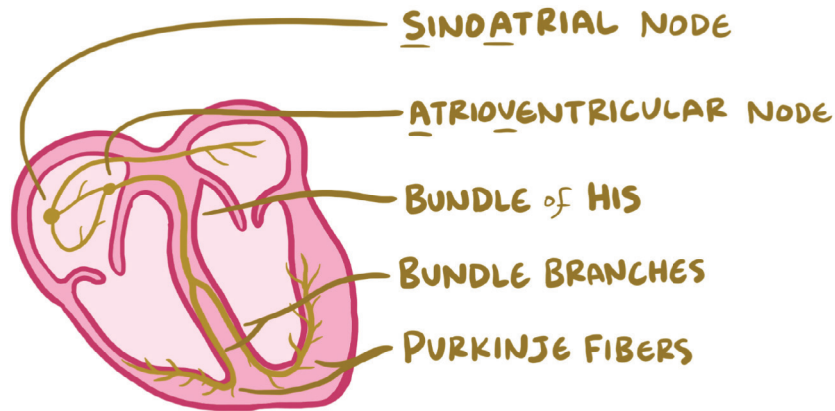


Figure 4.4

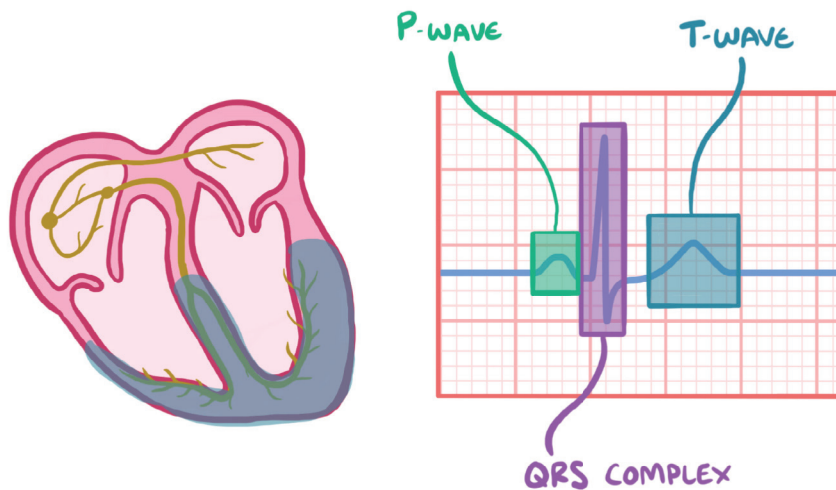
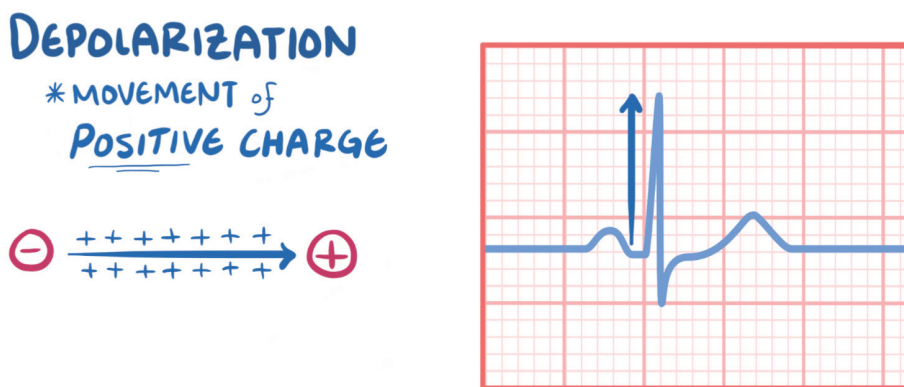


Figure 4.5



On an ECG, the atrial depolarization is seen as a P wave, the ventricular contraction is seen as a QRS complex, and the ventricular repolarization, a period of relaxation, is seen as a T wave (Figure 4.4). Keep in mind that a depolarization is caused by the movement of positive charge, so if this movement of positive charge is going toward the positive electrode, then it's captured as a positive deflection on an ECG (Figure 4.5).

Figure 4.6

AVERAGE QRS VECTOR

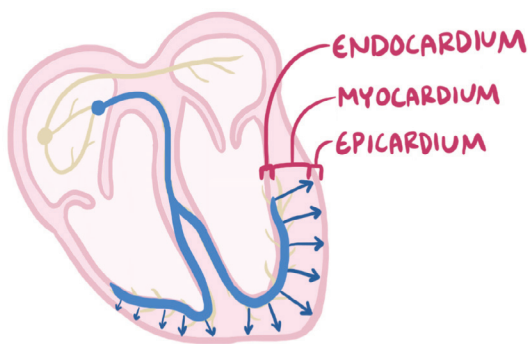
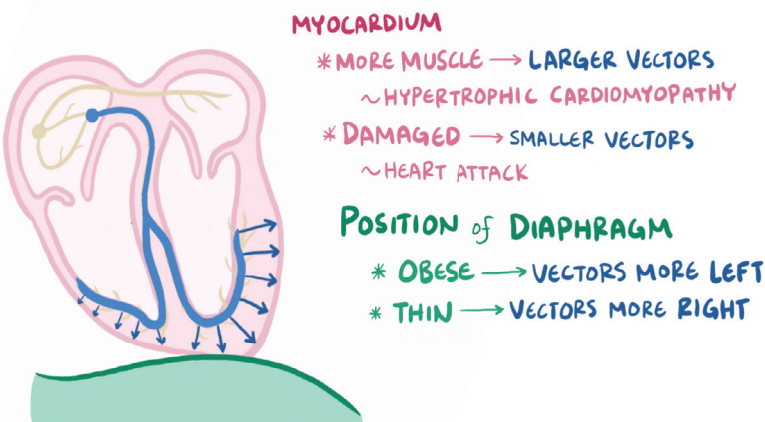


Figure 4.7

AVERAGE QRS VECTOR



MEAN QRS VECTOR

With this in mind, let's take a closer look at the mean, or average, QRS vector. After the depolarization wave arrives at the AV node, it travels down the interventricular septum and begins depolarizing the ventricles. The Purkinje fibers sit just below the endocardium, the innermost layer of the heart. After the endocardium, there's the myocardium, the cardiac muscle cells, and finally, the epicardium, which is the outer layer. Therefore, each Purkinje fiber initiates a depolarization vector that travels directly outward; it starts in the endocardium, goes through the myocardium, and ends in the epicardium (**Figure 4.6**). Because they transmit a depolarization wave so quickly, they all fire off pretty much in unison. The more muscle tissue in the myocardial layer that a vector has to travel through, the larger the size of the vector. So, as with hypertrophic cardiomyopathy, where the heart muscle gets thicker, you get bigger vectors.

However, if the heart tissue has been damaged, such as from a heart attack, then you have smaller vectors because the heart cells can't depolarize anymore. The position of the diaphragm can also affect vectors because it's usually sort of sitting right up against the heart. In people with obesity the diaphragm gets pushed upwards, rotating the heart further to the left. In thin people, the diaphragm lowers, rotating the heart a bit the other way (**Figure 4.7**).

Figure 4.8

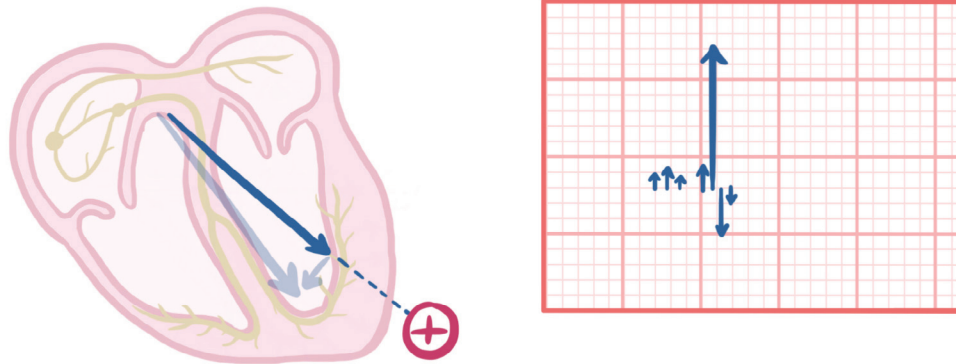


Figure 4.9

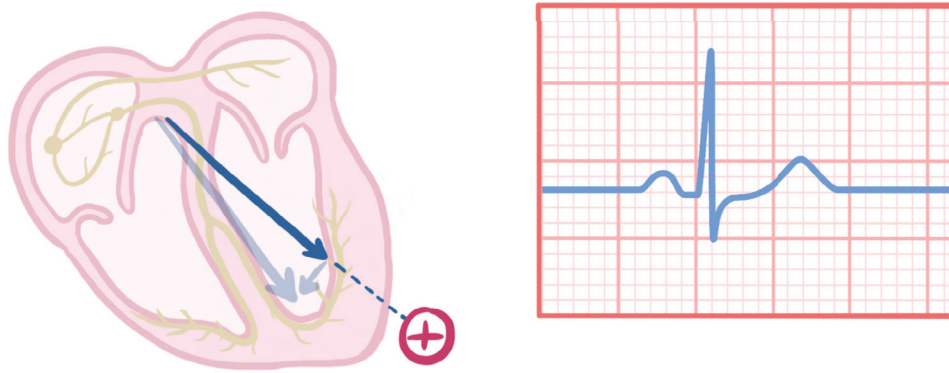


Figure 4.10

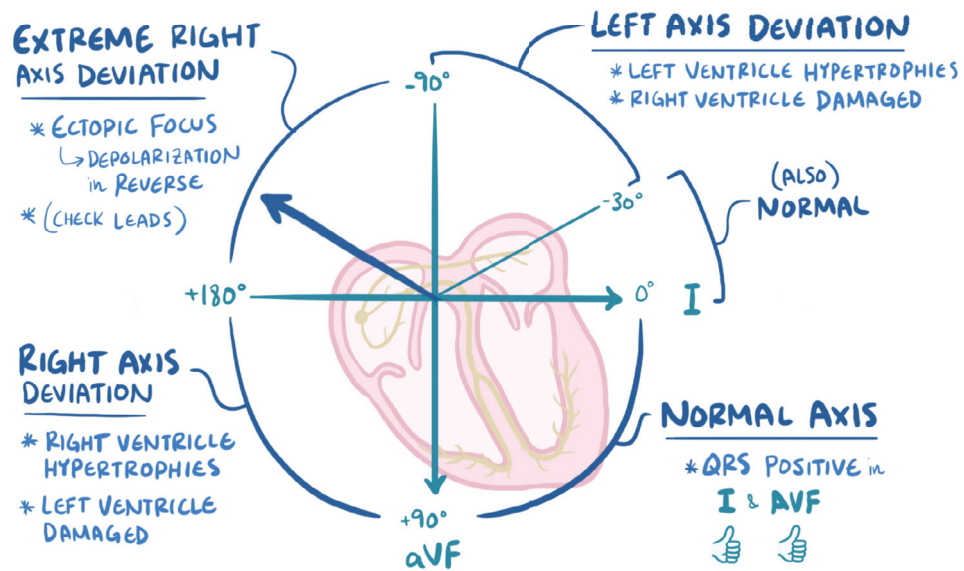


Figure 4.11

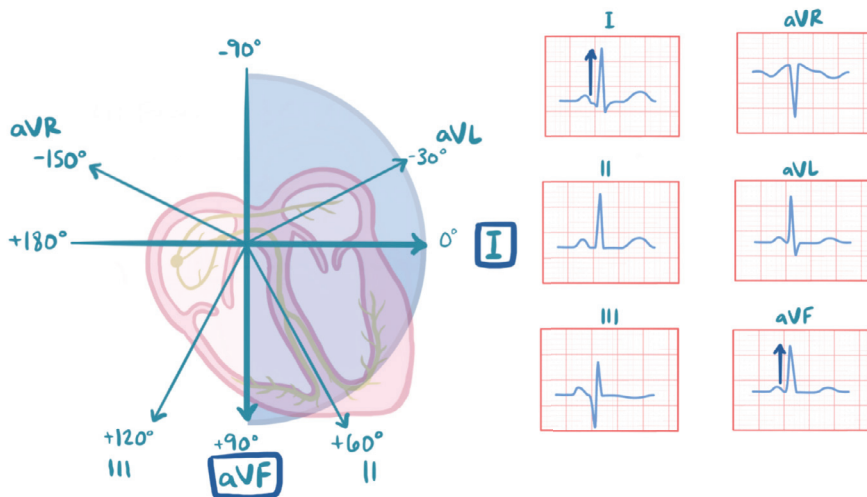


Figure 4.12

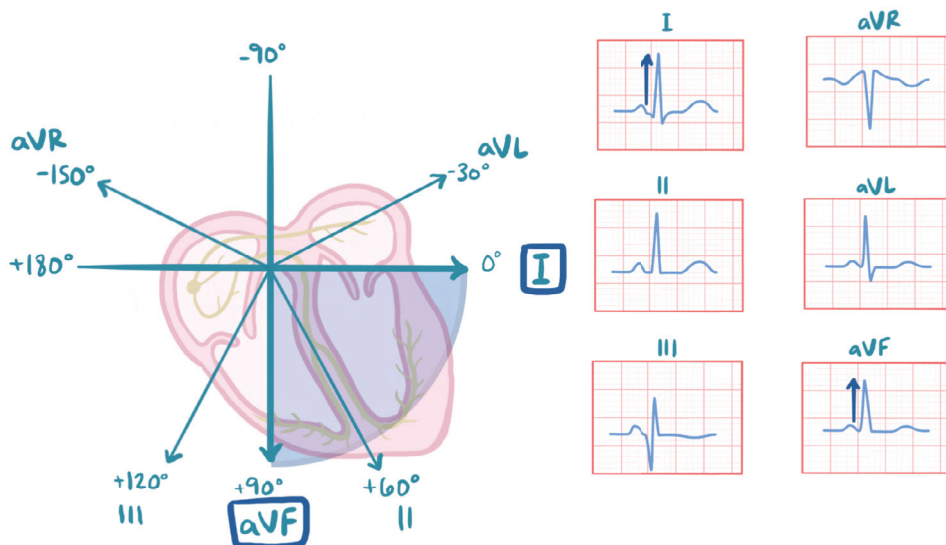


Figure 4.13

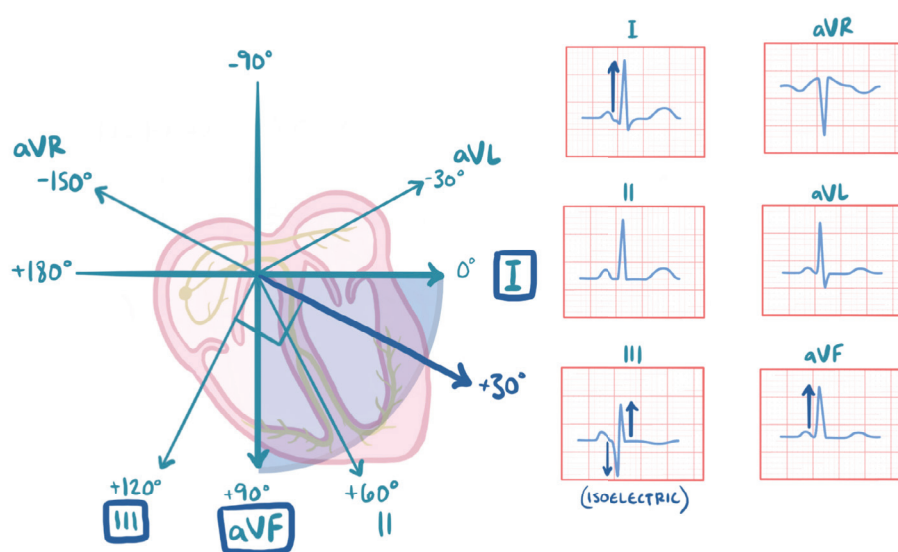


Figure 4.14

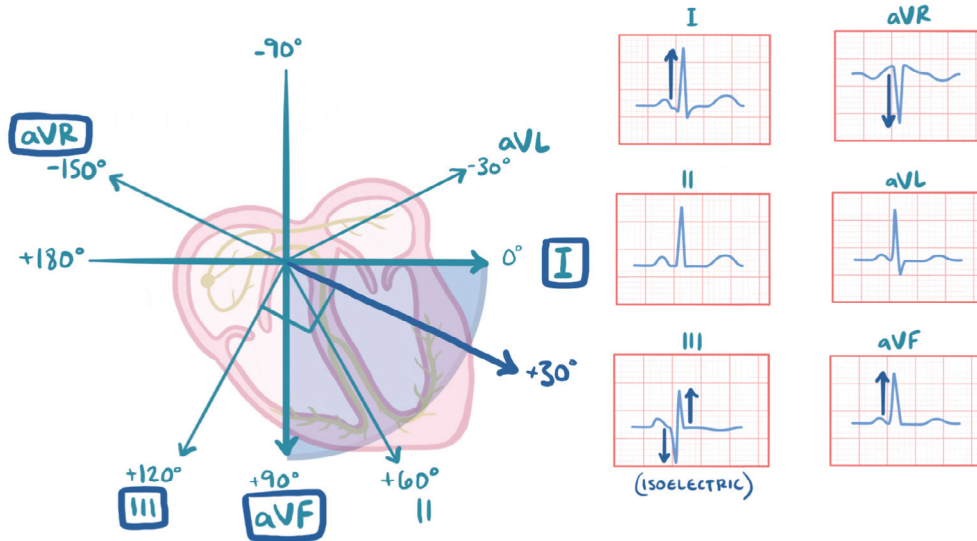


Figure 4.15

CHEST LEADS

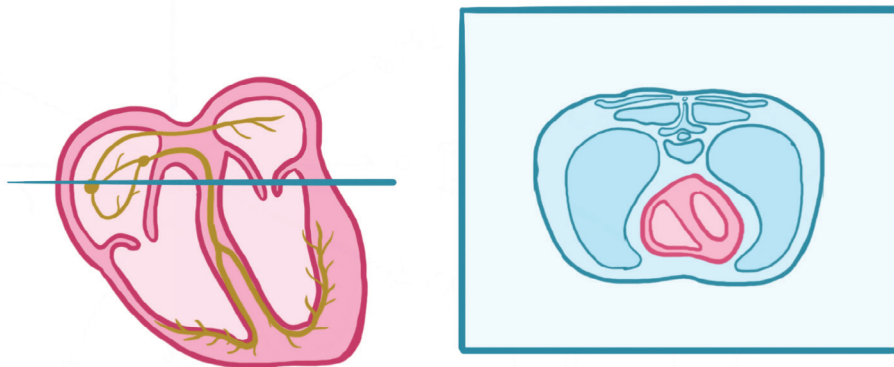


Figure 4.16

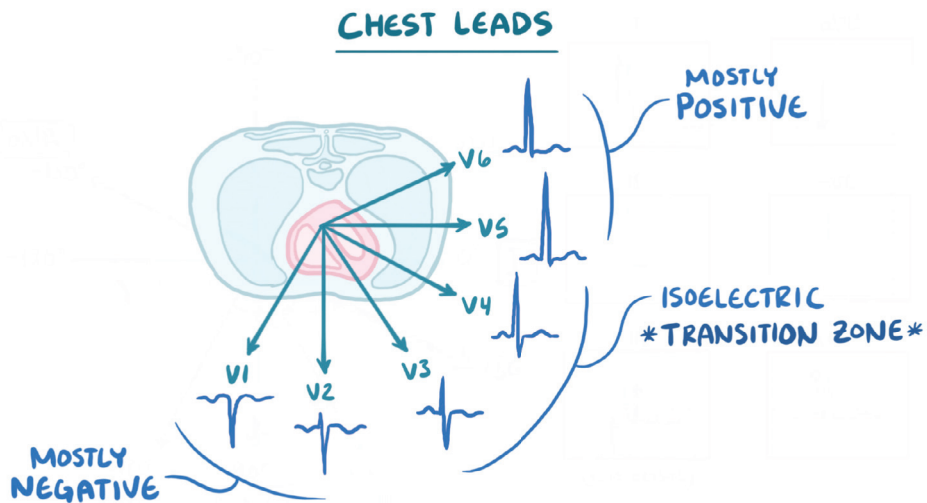
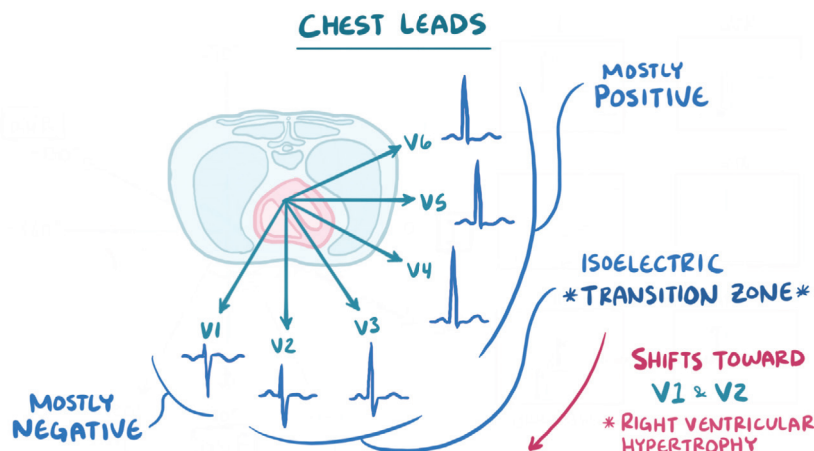


Figure 4.17

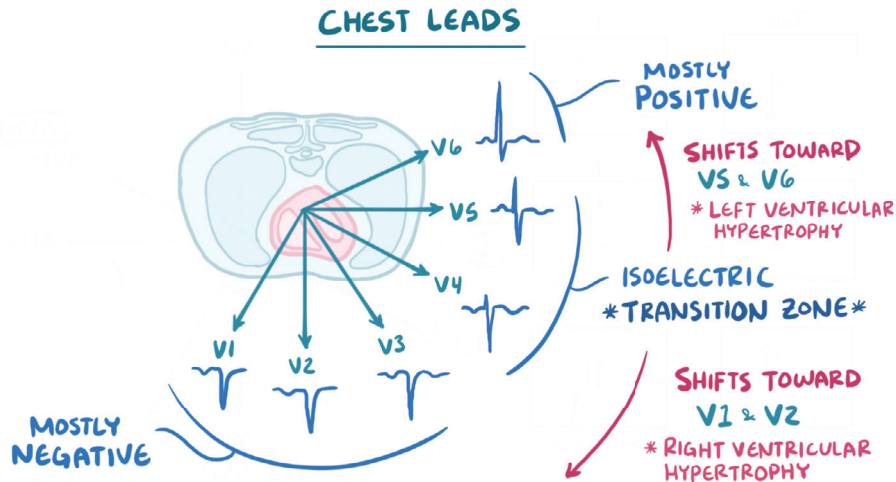


When everything is taken into consideration and all of the individual vectors are added up, there's one overall representative vector arrow, which starts from the AV node and points in one direction through the ventricles (**Figure 4.8**). Now, vectors can be broken down into two perpendicular vector components, so if you look at the component pointing at the positive lead electrode, that's what is recorded on the ECG. Therefore, when you plot these vectors over the course of ventricular depolarization, the QRS complex emerges (**Figure 4.9**).

All right, to figure out the direction of the axis we can start by looking at lead I and aVF. Lead I moves from right to left across the heart, so anything pointing to the left will be positive in lead I; in contrast, aVF points downward, so any vector pointing downward will be positive in aVF. So, looking at our overall vector from before, it's pointing both down and to the left, and both are positive. Thus, it's in the bottom left quadrant between 0 and +90 degrees; that's a normal axis. If it's up in lead I and aVF, you can imagine it's like seeing two thumbs up, which means that everything is okay! If the vector is positive in lead I and negative in aVF, then it's in this quadrant; it could be normal if it's between 0 and -30 degrees. However, if it's between -30 and -90, it could be considered left axis deviation. Left axis deviation can happen when the left ventricle hypertrophies, or when the right ventricle becomes damaged and loses healthy tissue. If the opposite happens, and the vector is negative in lead I and positive in aVF, then it's in this quadrant between +90 and +180 degrees; we call this right axis deviation. Right axis deviation can happen when the right ventricle hypertrophies, or when the left ventricle is damaged and loses healthy tissue. Finally, if the vector is negative in both lead I and aVF, then it's a very rare case called extreme right axis deviation between -90 and +180 degrees. This can sometimes happen when there's an ectopic focus that causes depolarization to start from the bottom of the ventricles, and travel in the reverse direction. It's also a good idea to double check that the leads are placed correctly (**Figure 4.10**).

You can figure out the QRS vector within 30 degrees by looking at someone's ECGs. Specifically, to do so, you'll look at the six leads, I, II, III, aVR, aVL, and aVF, which correspond to 0 degrees, +60 degrees, +120 degrees, -150, -30, and +90, respectively (**Figure 4.11**). Here, lead I is clearly positive, which means that the axis is somewhere over here, and lead aVF is positive; this puts the axis in the lower left quadrant between 0 and +90 degrees, which is a normal axis (**Figure 4.12**). To be more precise, it looks like lead III is the closest to being isoelectric, which means equal positive and negative deflections; therefore, lead III must actually run *perpendicular* to the QRS vector, so 120 degrees subtract 90 degrees is the +30 degree line (**Figure 4.13**). Notice that this happens to sit on the same line as aVR, but in the opposite direction (**Figure 4.14**). We would think that this vector should look *negative* in aVR, and it does!

Figure 4.18

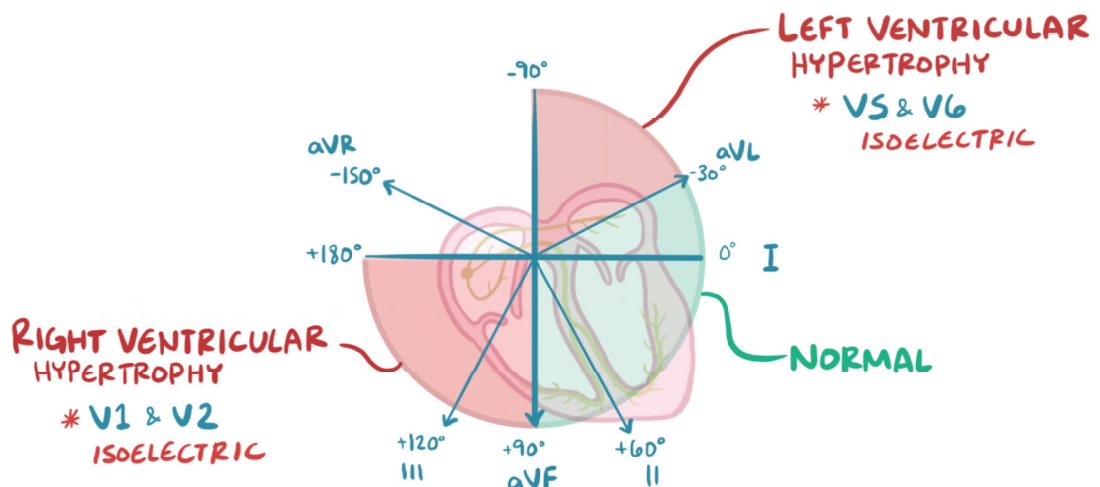


Now, let's switch gears and talk about the chest leads, which essentially view the heart in a different plane (Figure 4.15). Looking down at the heart we have leads V_1 , V_2 , V_3 , V_4 , V_5 , and V_6 . Normally, the QRS complex is negative in leads V_1 and V_2 , isoelectric in lead V_3 and V_4 , which is called the transition zone, and positive in leads V_5 and V_6 (Figure 4.16). If the transition zone shifts toward V_1 or V_2 , and looks isoelectric instead of negative, it suggests that the heart may be rotated to the person's right, which can happen if the right ventricle is hypertrophied (Figure 4.17). On the other hand, if leads V_5 or V_6 look isoelectric instead of positive, it suggests that the heart may be rotated to the person's left, which can happen if the left ventricle is hypertrophied (Figure 4.18).

SUMMARY

All right, let's quickly recap. A normal heart's axis is between -30 and $+90$ degrees. Right ventricular hypertrophy can cause the axis to be between $+90$ and $+180$, and can sometimes cause the V_1 and V_2 chest leads to appear isoelectric. Left ventricular hypertrophy can cause the axis to be between -30 and -90 , and can sometimes cause the V_5 and V_6 chest leads to appear isoelectric (Figure 4.19).

Figure 4.19



Intervals

osmosis.org/learn/ecg_intervals

When reading an ECG, there are a few key elements to keep in mind; one of them is looking at the intervals (Figure 5.1). In a typical waveform, there's a P wave, QRS complex, and T wave. In addition, there are certain intervals, including the PR interval, the QRS complex itself, and the QT interval.

Figure 5.1

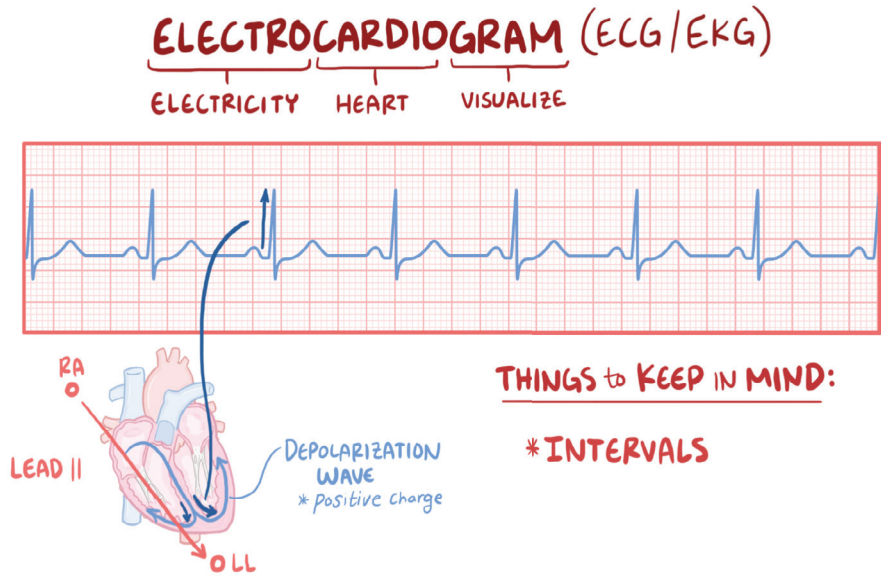


Figure 5.2

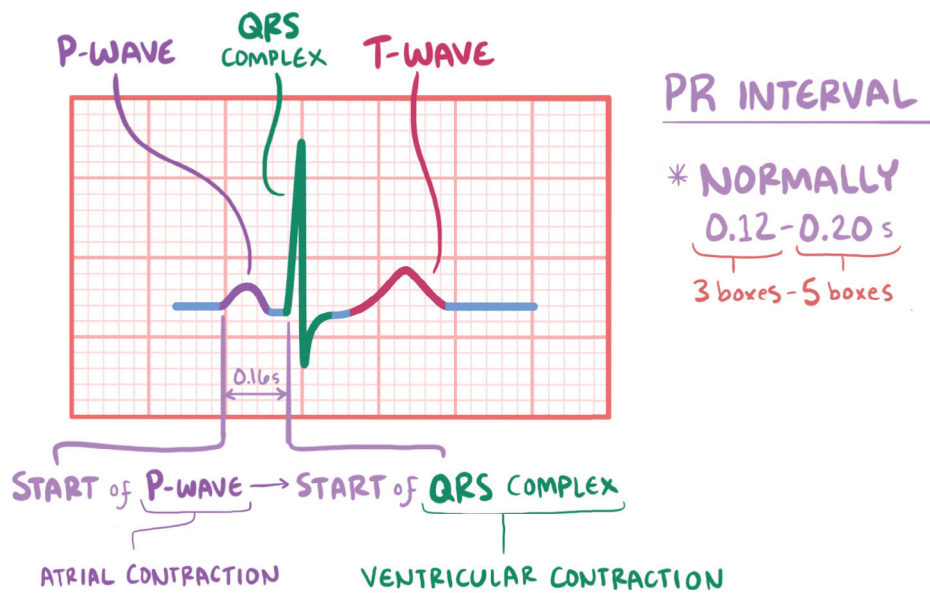
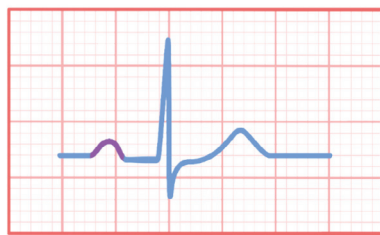


Figure 5.3



*CHANGES in PR INTERVAL
└ ATRIAL ECTOPIC FOCUS
 * FARTHER → LONGER

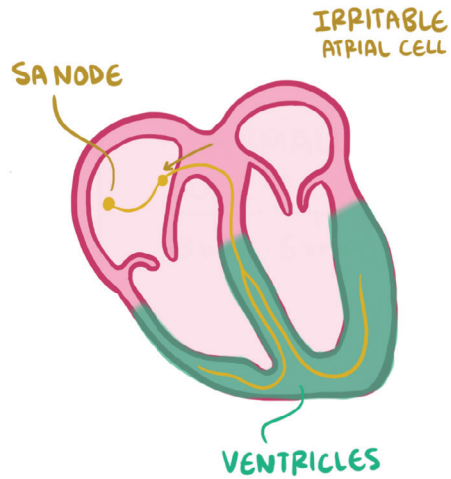
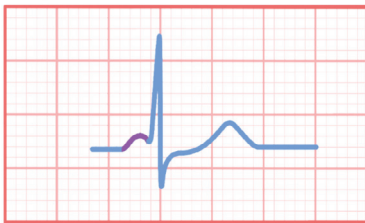


Figure 5.4



*CHANGES in PR INTERVAL
└ ATRIAL ECTOPIC FOCUS
 * FARTHER → LONGER
 * CLOSER → SHORTER
└ FIRST DEGREE HEART BLOCK

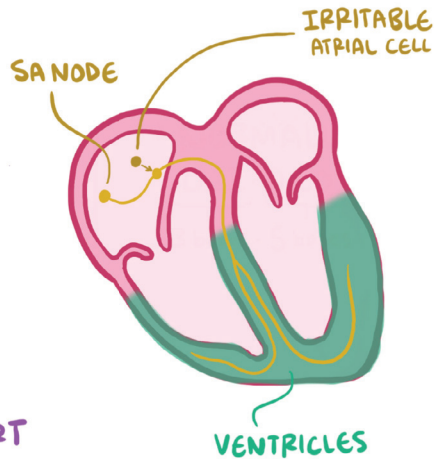
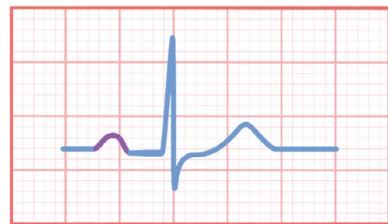


Figure 5.5



*CHANGES in PR INTERVAL
└ ATRIAL ECTOPIC FOCUS
 * FARTHER → LONGER
 * CLOSER → SHORTER
└ FIRST DEGREE HEART BLOCK ~ > 0.2 s

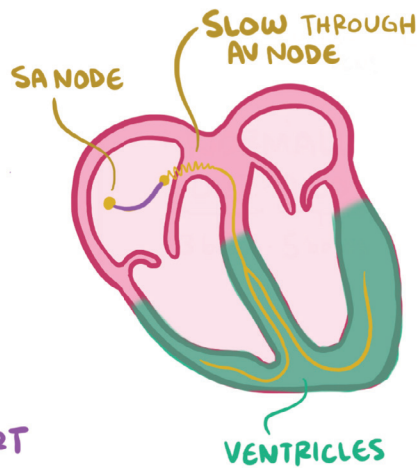
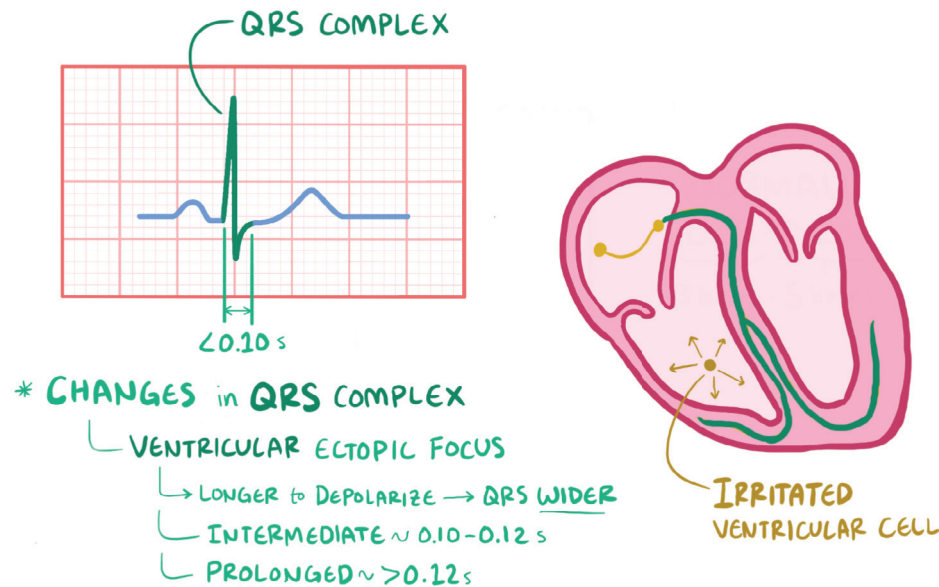


Figure 5.6



PR INTERVAL

The PR interval spans from the beginning of the P wave to the beginning of the QRS complex, and it represents the time from the beginning of atrial depolarization to the beginning of ventricular depolarization. It's normally 0.12-0.20 seconds, which is three to five little boxes, since each little box is 0.04 seconds. Therefore, the PR interval shown is about four boxes or 0.16 seconds (Figure 5.2).

Any deviation in the normal depolarization pathway from the SA node to the ventricles can change the PR interval. For example, consider if the atria are depolarized by an ectopic atrial focus, such as an irritable atrial cell outside of the SA node. If it was farther away from the AV node, it'd result in a *longer* PR interval, because the signal has to travel a greater distance (Figure 5.3).

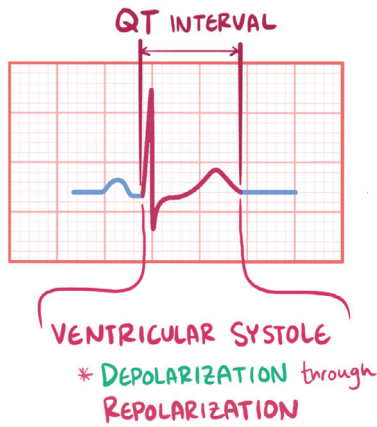
Alternatively, if it was really *close* to the AV node, the PR interval might be super short. Another example is first degree heart block, which is when the electrical signal travels more slowly through the AV node than it normally does, causing the PR interval to lengthen beyond 0.2 seconds (Figure 5.4).

QRS COMPLEX

The QRS complex represents depolarization of the ventricles; it's normally less than 100 milliseconds or two and a half little boxes. Just like the PR interval, the QRS duration can differ in its path from the AV node to the ventricles (Figure 5.5).

For example, if an ectopic ventricular focus, such as an irritated ventricular cell, fires off, the resulting depolarization wave will move slowly from muscle cell to muscle cell, instead of traveling quickly through the electrical conduction system. Therefore, it takes a longer time to depolarize the ventricles, and the QRS is wider. It's considered intermediate if it's 100 to 120 milliseconds, and prolonged if it's over 120 milliseconds, or three little boxes (Figure 5.6).

Figure 5.7



* NORMAL ~ LESS THAN (ABOUT) HALF a CARDIAC CYCLE

* ABNORMALLY LONG

@ 60 BPM

↳ > 440 ms (MEN)

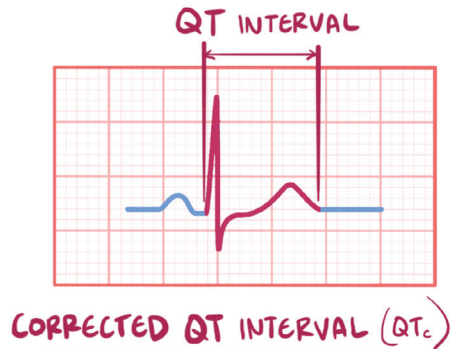
↳ > 460 ms (WOMEN)

@ 90 BPM

↳ 400 ms ~ ~~NORMAL?~~

CHANGES with RATE!

Figure 5.8



* BAZETT'S FORMULA

$$QT_c = \frac{QT (ms)}{\sqrt{\frac{R-R (sec)}{1 sec}}} = \frac{400 ms}{\sqrt{\frac{0.66 s}{1 s}}}$$

* ABNORMALLY LONG

@ 60 BPM

↳ > 440 ms (MEN)

↳ > 460 ms (WOMEN)

@ 90 BPM

↳ 400 ms

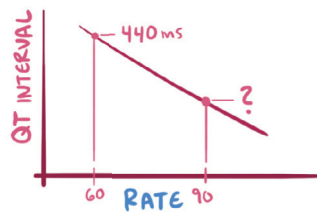
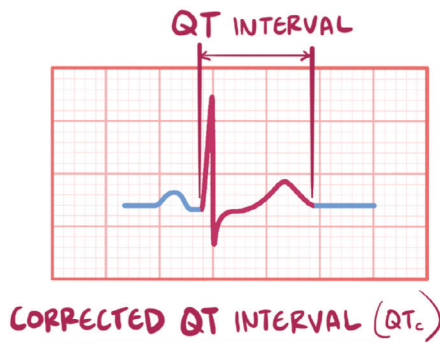


Figure 5.9



* BAZETT'S FORMULA

$$QT_c = \frac{QT (ms)}{\sqrt{\frac{R-R (sec)}{1 sec}}} = \frac{400 ms}{\sqrt{\frac{0.66 s}{1 s}}} = \frac{400 ms}{0.81} = \underline{\underline{493 ms}}$$

* ABNORMALLY LONG

@ 60 BPM

↳ > 440 ms (MEN)

↳ > 460 ms (WOMEN)

@ 90 BPM

↳ 400 ms (QT)

↳ 493 ms (QTc) > 440 ms

LONG!

Figure 5.10

FACTORS AFFECTING QT INTERVAL

SHORTEN

- * HYPERCALCEMIA

LENGTHEN

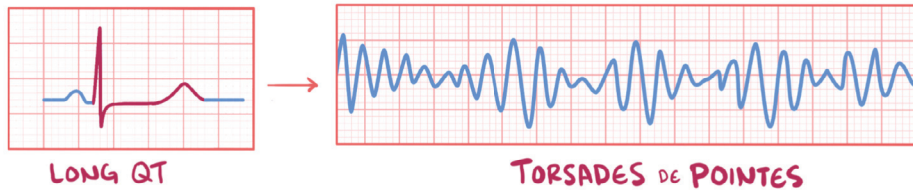
- * HYPOCALCEMIA

- * MEDICATIONS

↳ AMIODARONE ~ AFFECTS ION CHANNELS

- * INHERITED

↳ MUTATIONS in LQT1, LQT2, LQT3



QT INTERVAL

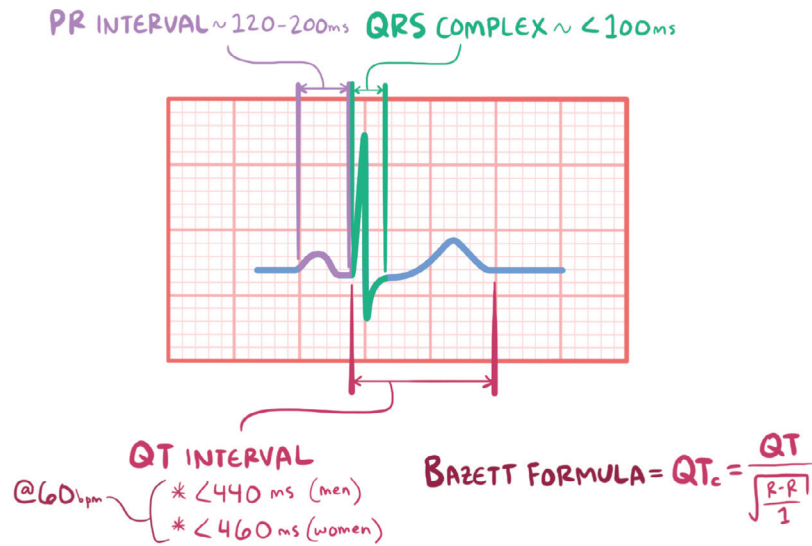
The QT interval spans from the beginning of the QRS complex to the end of the T wave. It represents ventricular systole, which is the entire span from depolarization through repolarization. Normally, the QT interval should be less than half of a cardiac cycle. In fact, for a heart rate of 60 beats per minute, the QT interval is generally considered to be abnormally long when it's greater than 440 milliseconds in men, or 460 milliseconds in women. If you measure someone's QT interval at a different rate, say 90 beats per minute, and it was 400 milliseconds, you might think that that'd be considered normal; however, you can't really use these values to compare to the normal QT interval at 60 beats per minute, or bpm, because the QT interval changes depending on the rate (**Figure 5.7**).

As rate increases, the QT interval decreases; thus, at 60 bpm, the abnormal cutoff might be 440 ms, whereas at 90 bpm, it's likely something lower. So, what we have to do is find the *corrected* QT interval, or QT_c , at the different rate. Then, you can compare it to the QT interval at 60 beats per minute. Even though there are several formulas you can use, Bazett's formula is probably the simplest, where the corrected QT interval equals the QT interval in milliseconds, divided by the square root of the R to R interval in seconds, divided by one second. As a side-note, this formula is usually expressed *without* the "divide by 1 second" part, but the astute observer will notice that the units won't work out if you leave it out (**Figure 5.8**).

Let's use our 90 bpm and 400 milliseconds QT interval from before, and imagine the person is male. If we plug in 400 for QT and 90 beats per minute, or 0.66 seconds per beat, we have a QT of 400 milliseconds divided by the square root of 0.66 seconds over 1 second, which is 400 milliseconds divided by 0.81, which is unitless, and we get a corrected QT interval of 493 milliseconds. Now we can compare this sum to the value at 60 bpm. We see that the corrected QT_c is actually *greater* than 440 ms, which means that this measured QT interval of 400 ms at 90 bpm is indeed abnormally long (**Figure 5.9**)!

Finally, several factors can affect the QT interval. Hypercalcemia, or too much calcium in the blood, typically shortens the QT interval; hypocalcemia, or too little calcium in the blood, lengthens it. The QT interval can also be prolonged by medications, such as amiodarone, which is an antiarrhythmic that affects cardiomyocyte ion channels, or caused by inherited long QT syndromes, where there are mutations in genes, including LQT1, LQT2, or LQT3. One of the most feared complications of having a prolonged QT interval is a type of ventricular tachycardia called torsades de pointes, which can lead to sudden cardiac death; here, the ECG shows a "twisting of the points" (**Figure 5.10**).

Figure 5.11



SUMMARY

All right, let's quickly recap. An ECG waveform has a PR interval that's usually 120 to 200 milliseconds, a QRS complex that's less than 100 milliseconds, and a QT interval that's less than 440 milliseconds in men and 460 milliseconds in women; these values are for rates at 60 bpm. Bazett's formula can be used to find the corrected QT interval, which you can use to compare rates other than 60 bpm to the normal rate (**Figure 5.11**).

QRS Transition

osmosis.org/learn/ecg_qrs_transition

To read an ECG, there are a few key elements to keep in mind; one is to figure out the QRS transition (Figure 6.1). The chest leads, V_1 through V_6 (Figure 6.2), “view” the heart through the horizontal plane. Each one has its own slightly different “view,” which will have a mostly positive deflection if a depolarization wave is moving towards it, and a mostly negative if the depolarization wave is moving away from it.

QRS VECTOR & TRANSITION ZONE

The QRS transition zone refers to where the QRS complex switches from being mostly negative to mostly positive, from the point of view of the chest leads. The QRS transition usually happens in lead V_3 or V_4 , depending on factors such as chest lead placement and the exact anatomy of a person’s heart (Figure 6.3). So, the QRS transition tells us when the overall QRS vector is aligned in the direction of the chest leads; it’s a way of understanding what’s happening to the QRS axis in the horizontal plane.

Figure 6.1

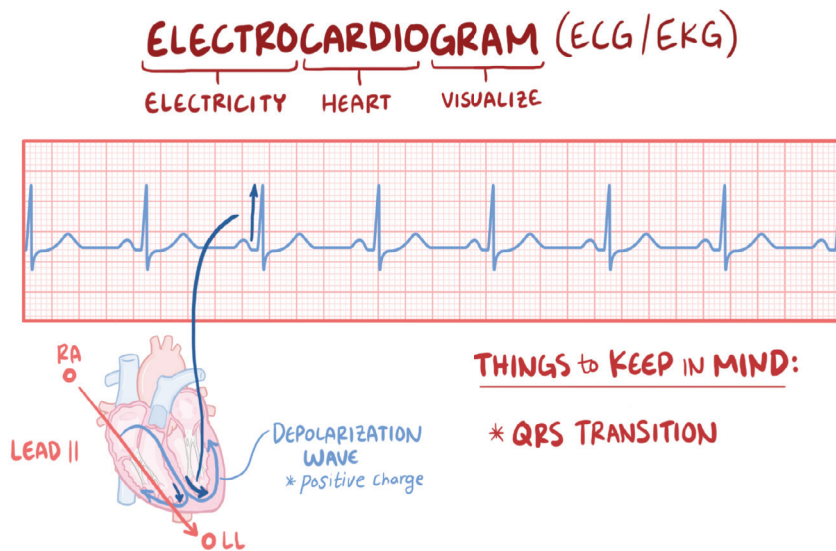


Figure 6.2

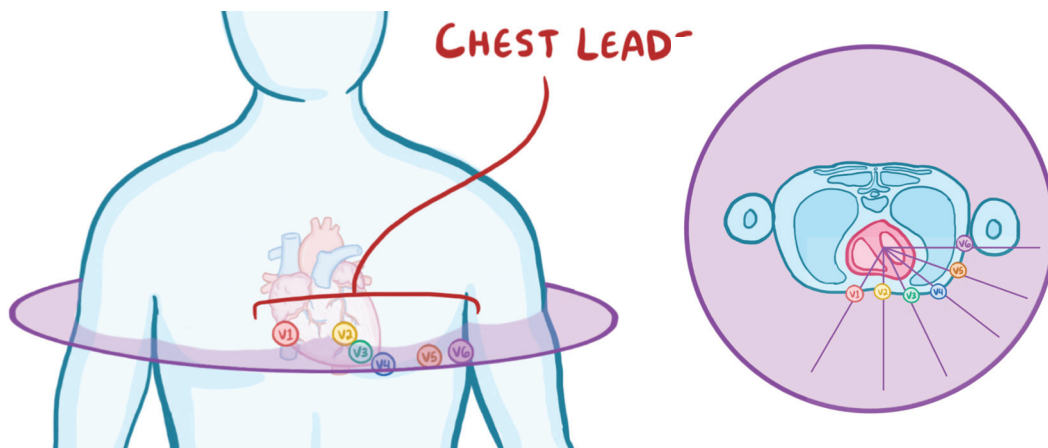


Figure 6.3

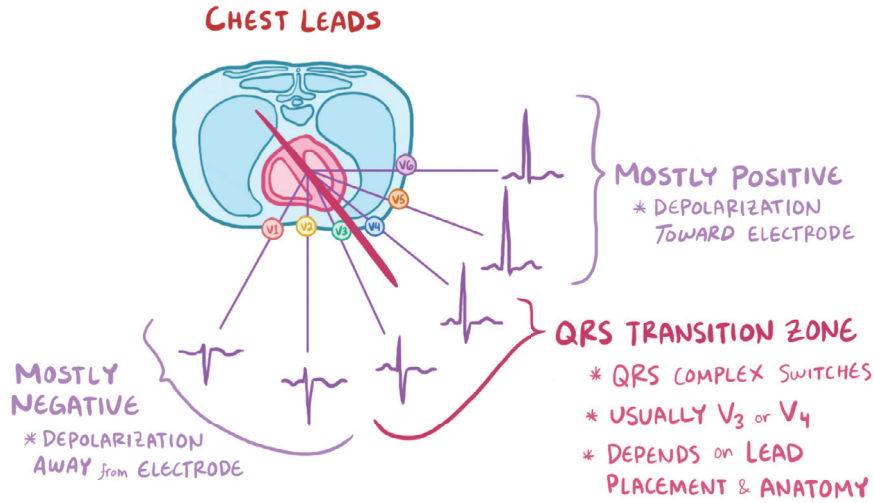


Figure 6.4

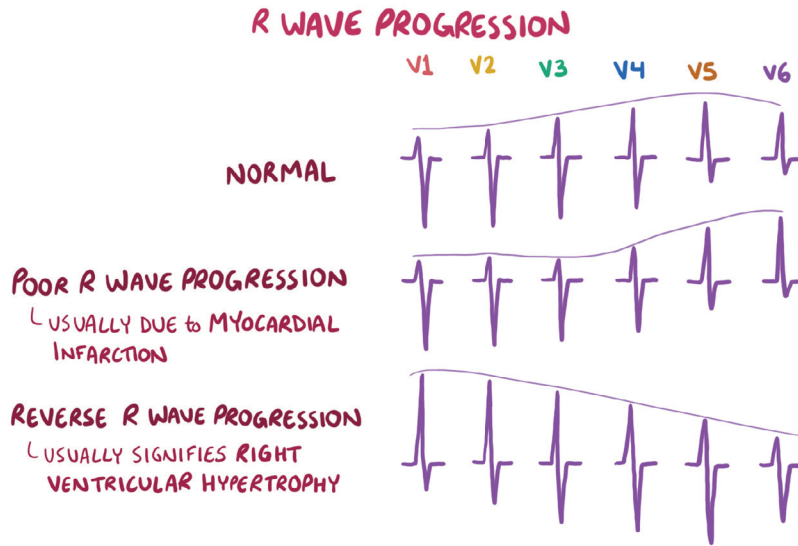


Figure 6.5

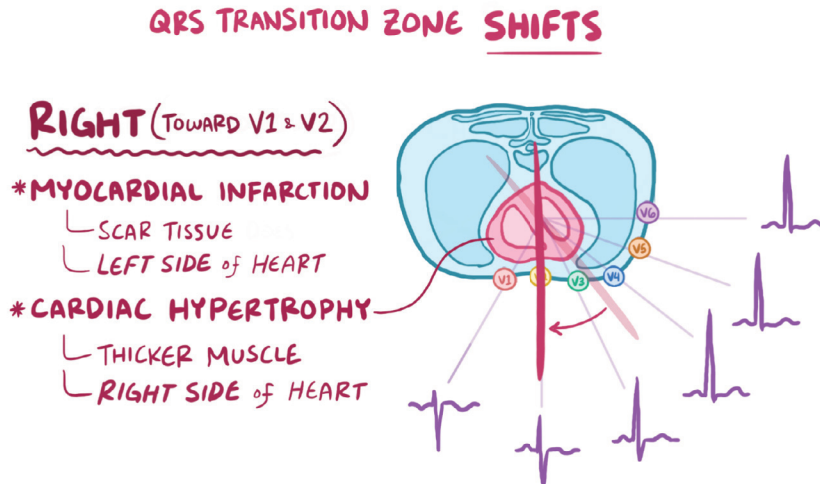
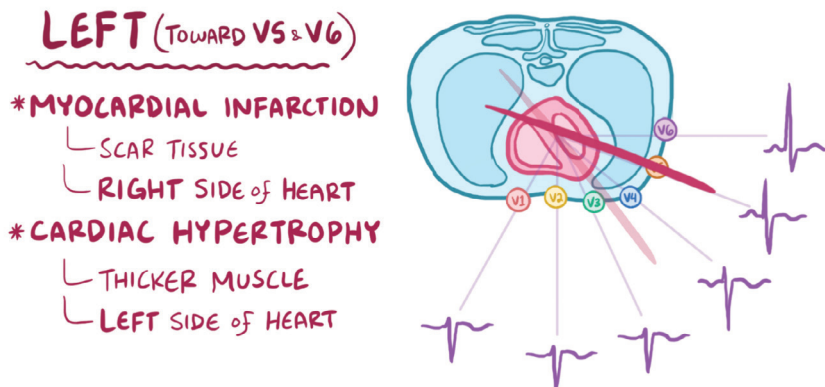


Figure 6.6 **QRS TRANSITION ZONE SHIFTS**



R WAVE PROGRESSION

Another way to look at QRS transition is to look at R wave progression. Normally, the R wave is small in V₁, and progressively grows until V₃ or V₄, only to become smaller again towards V₆. One abnormal ECG finding is *poor R wave progression*, which is when the R wave doesn't grow as it should until V₄; it's usually because of myocardial infarction. There's also *reverse R wave progression*, which is when the R wave is tall in V₁ and V₂; this usually signifies right ventricular hypertrophy (Figure 6.4).

CASE STUDY

If something alters the heart's overall QRS vector, then the QRS transition zone can shift to the right, towards V₁ and V₂. For example, a myocardial infarction leads to the formation of scar tissue that can't depolarize. Generally speaking, the QRS transition zone will shift away from a region of scar tissue, because it no longer contributes to the overall QRS vector. So in order for it to shift right, the infarction would have to be on the left side of the heart. Consider the following: a blockage in the left circumflex artery can cause infarction of the posterior wall of the left ventricle, which would lead to a rightward rotation of the QRS transition zone, toward V₁ and V₂ (Figure 6.5). Another example would be cardiac hypertrophy, because a thicker muscular wall contributes more to the overall QRS vector; in order for this to be a shift to the right, it would have to be right ventricular hypertrophy (Figure 6.5). In contrast, there can also be a shift to the left, toward V₅ and V₆; this would be caused by a myocardial infarction on the right side of the heart, or hypertrophy on the left. (Figure 6.6).

SUMMARY

All right, let's quickly recap. The QRS transition zone, in the horizontal plane, is where the QRS complex switches from being mostly negative in V₁ and V₂ to mostly positive in V₅ and V₆; it normally happens in lead V₃ or V₄. The QRS transition zone rotates away from previous myocardial infarctions, and toward hypertrophied tissue (Figure 6.3).

Cardiac Hypertrophy & Enlargement

osmosis.org/learn/ecg_cardiac_hypertrophy_and_enlargement

There are lots of things to look for when reading an ECG, including figuring out if part of the heart has undergone hypertrophy or enlargement (**Figure 7.1**). *Hypertrophy* means that a heart's muscular wall has increased in thickness. *Dilation* refers to an increase in the volume of the chamber. The term *enlargement* is generally used when both hypertrophy and dilation occur together; this is what typically happens in the atria. In contrast, the ventricles often undergo hypertrophy without dilation (**Figure 7.2**). An ECG can show evidence of hypertrophy and enlargement in all of the heart's four chambers. Let's go through them one by one.

ATRIAL ENLARGEMENT

Normally, atrial depolarization produces a pretty normal-looking symmetric P wave. In right atrial enlargement, all of that extra right atrial muscle tissue results in a large P wave in leads V_1 and V_2 that is often over 1.5 mm, as well as in the inferior leads, which are leads II, III, and aVF, that is often over 2.5 mm. One reason why right atrial enlargement develops is that there can be a stenotic, or narrowed, tricuspid valve that makes it more difficult for the atria to eject blood into the ventricles. In response, the right atrium enlarges (**Figure 7.3; Figure 7.4**).

In left atrial enlargement, the left atrium has extra muscle tissue that results in a P wave with two peaks in lead II. The entire wave stretches out over 110 ms, and has a gap of over 40 ms separating the two peaks. In lead V_1 , the P wave is biphasic, which means that it looks like a hill with a valley alongside it. However, this can also be seen in healthy individuals. The key difference between a normal and pathologic biphasic P wave in V_1 is that, with left atrial enlargement, the negative portion is usually more than 1 mm deep, and lasts for more than 40 ms. Left atrial enlargement also develops from a stenotic valve, but this time it's the mitral valve that causes the left atrium to get bigger (**Figure 7.5**).

Figure 7.1

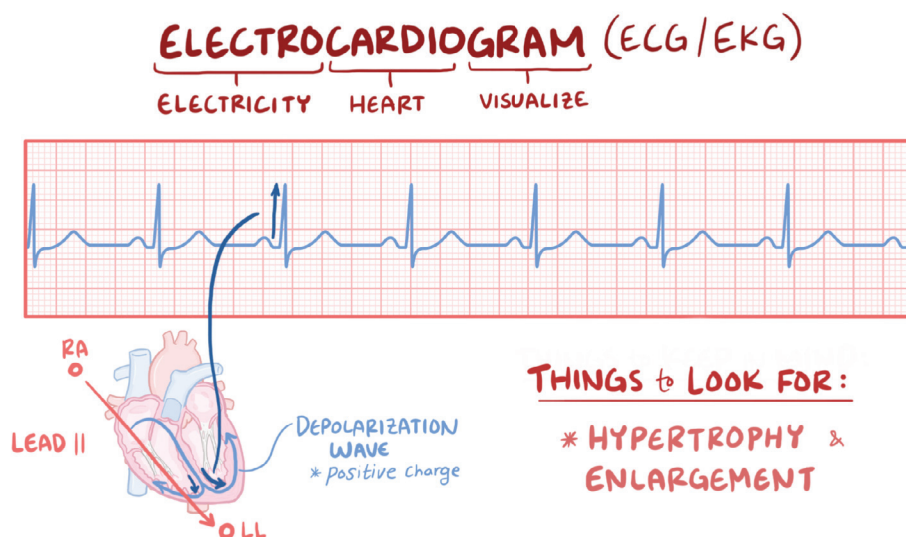


Figure 7.2

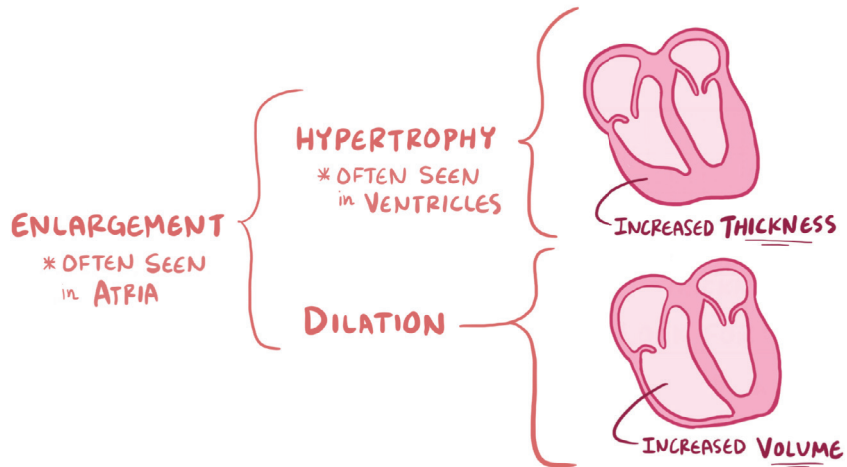


Figure 7.3

RIGHT ATRIAL ENLARGEMENT

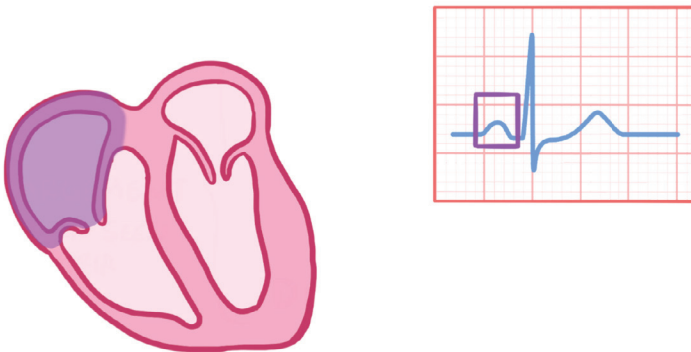
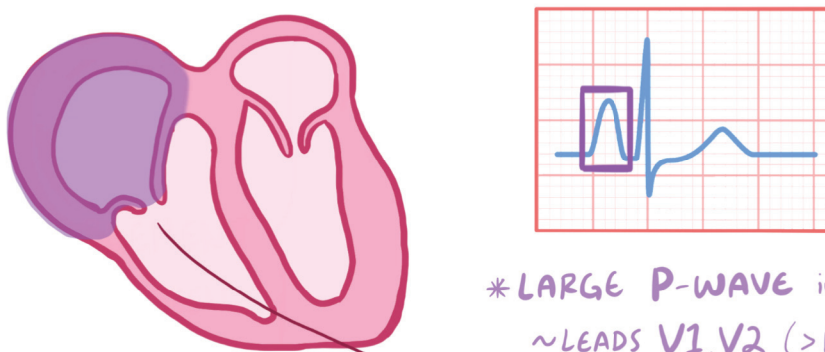


Figure 7.4

RIGHT ATRIAL ENLARGEMENT



CAUSE ~ STENOTIC (TRICUSPID VALVE)
* DIFFICULT to EJECT BLOOD

* **LARGE P-WAVE** in...
~ LEADS V1, V2 (>1.5mm)
~ LEADS II, III, aVF (>2.5mm)

Figure 7.5

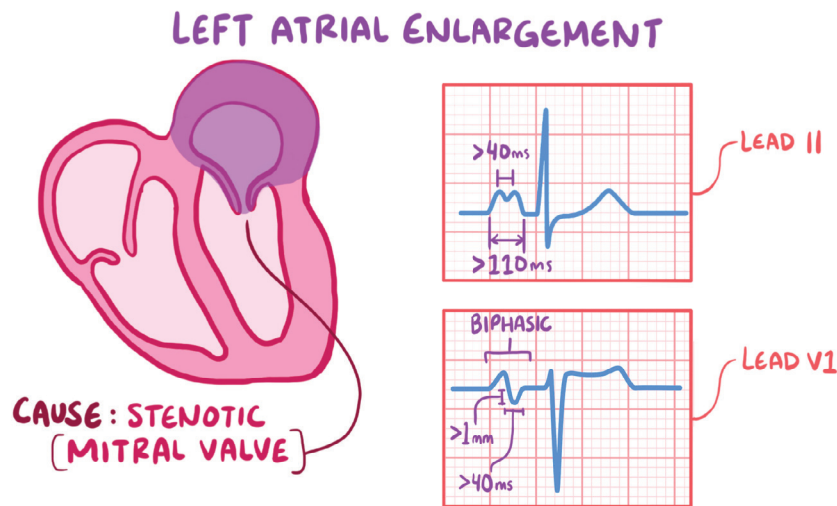
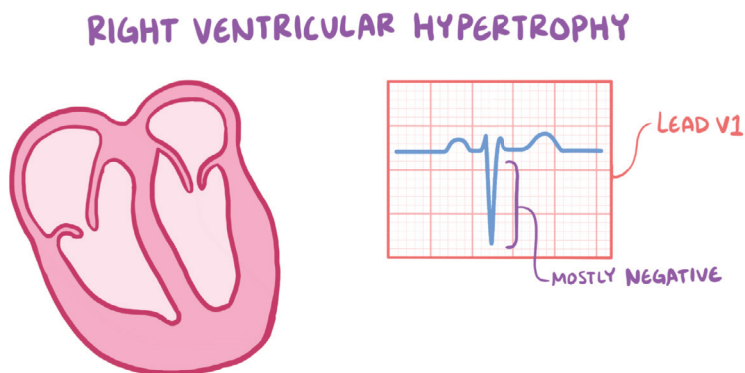


Figure 7.6



VENTRICULAR HYPERTROPHY

Normally, the QRS complex is mostly negative in lead V_1 , because the large left ventricle, which carries the greatest amount of muscle tissue, is oriented down and away from this electrode (Figure 7.6).

In right ventricular hypertrophy, the thicker right ventricle helps to counterbalance the left ventricle and makes lead V_1 more positive. Specifically, it makes the R wave bigger, which is the upward deflection of the QRS complex. In fact, a dominant R wave in V_1 is a classic sign of right ventricular hypertrophy; it's defined as an R wave that is over 7 mm tall, or 7 little boxes, and is larger than the S wave, making the R/S ratio > 1 . Another classic sign of right ventricular hypertrophy is a dominant S wave in V_5 or V_6 , meaning that it's over 7 mm deep, or 7 little boxes, and bigger than the R wave, making the R/S ratio < 1 . Typically, there's also right axis deviation in right ventricular hypertrophy that results in an axis of 110 degrees or more (Figure 7.7). In right ventricular hypertrophy the QRS complex is $<120\text{ms}$, meaning that it's not longer than normal. This is important because it means that the changes can't be the result of something like a right bundle branch block, which would cause a prolonged QRS complex. Right ventricular hypertrophy can develop for a number of reasons. A common cause is pulmonary hypertension, which makes sense because the right ventricle has to build up muscle to push blood into the lungs (Figure 7.8).

Figure 7.7

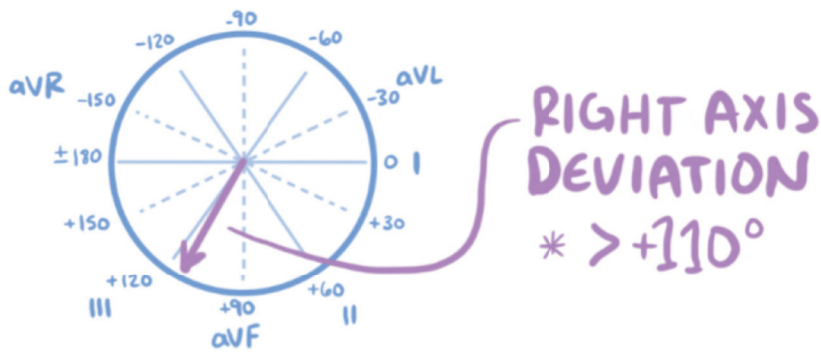
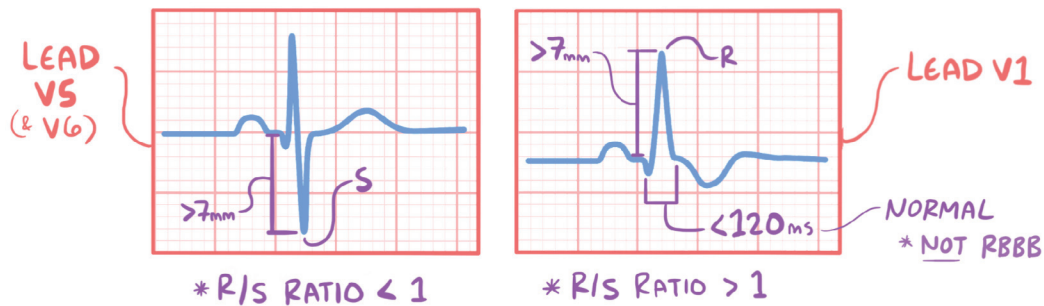


Figure 7.8

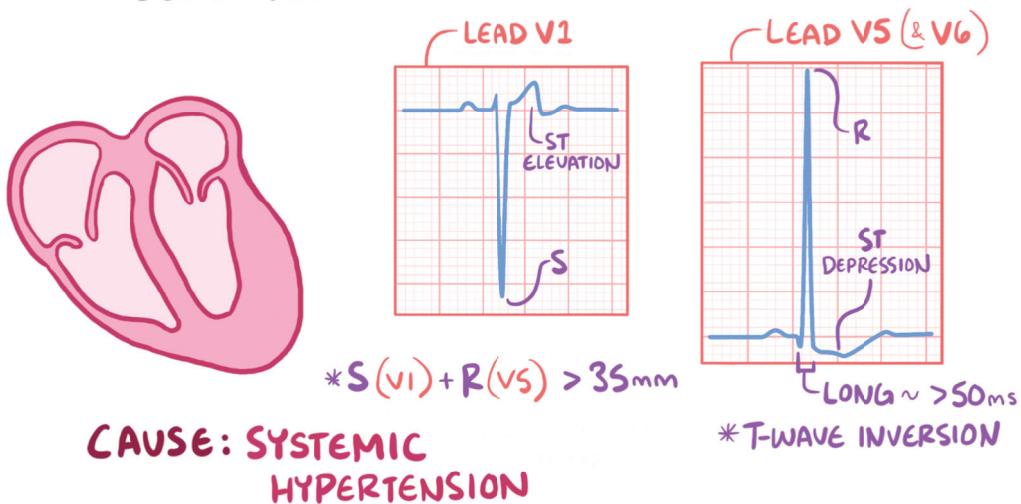
RIGHT VENTRICULAR HYPERTROPHY



CAUSE: PULMONARY HYPERTENSION

Figure 7.9

LEFT VENTRICULAR HYPERTROPHY

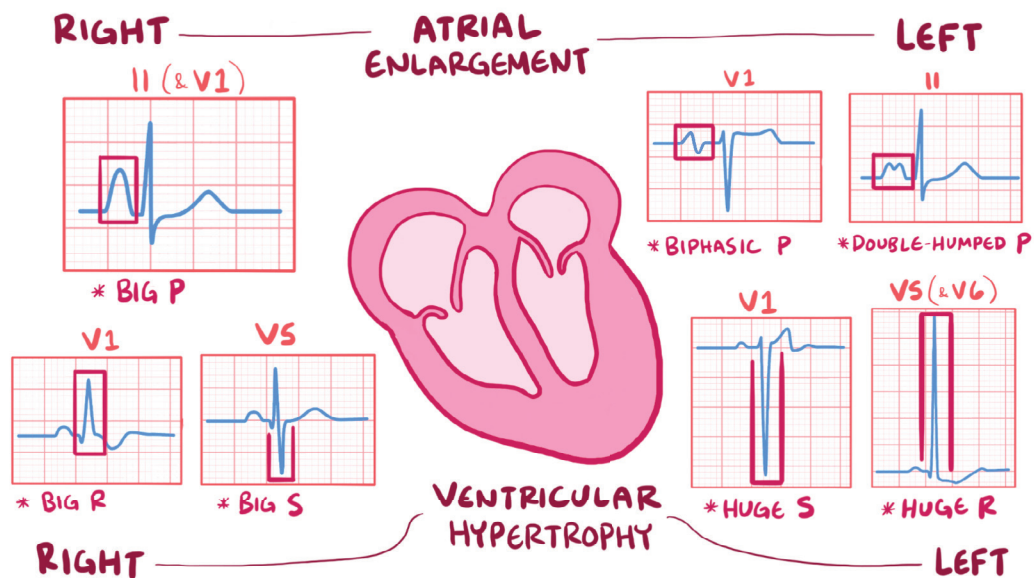


In left ventricular hypertrophy, the pattern is almost the opposite of what happens in right ventricular hypertrophy. Lead V_1 has even more positive charge traveling away from it than usual, making the S wave really deep. Meanwhile, lead V_5 and V_6 are located on the side of the left ventricle, and therefore have an enormous R wave. The most commonly used voltage criteria to identify left ventricular hypertrophy is that the sum of the S wave in V_1 and the tallest R wave in V_5 or V_6 has to be over 35 millimeters, or 35 little boxes. Additional criteria include having an R wave that goes on for longer than usual, typically over 50 ms, and ST elevation in V_1 , ST segment depression, and T wave inversion in V_5 or V_6 . These are signs that the left ventricle is straining during muscle contraction. Left ventricular hypertrophy commonly develops due to systemic hypertension, or elevated systemic blood pressure, because the left ventricle needs more muscle so that it can eject blood against higher pressures (**Figure 7.9**).

SUMMARY

All right, let's quickly recap. Right atrial enlargement shows a big P wave in lead II and V_1 , whereas left atrial enlargement has a biphasic P wave in lead V_1 , with a negative component greater than 1 mm deep and lasting more than 40 ms, as well as a double-humped P wave in lead II. Right ventricular hypertrophy shows a big R wave in V_1 and a big S wave in V_5 and V_6 , whereas left ventricular hypertrophy shows the opposite: an enormous S wave in V_1 and a large R wave in V_5 and V_6 that add up to over 35 mm (**Figure 7.10**).

Figure 7.10



Cardiac Infarction & Ischemia

osmosis.org/learn/ecg_cardiac_infarction_and_ischemia

To read an ECG, there are a few key elements to keep in mind; one is to figure out if part of the heart is suffering from ischemia or has undergone an infarction (Figure 8.1). The term *ischemia* means that blood flow to tissue has decreased, which results in *hypoxia*, or insufficient oxygen in the tissue. *Infarction* goes one step further, because it means that blood flow has been completely cut off, which results in necrosis, or cellular death. This typically happens if blood flow has been cut off for about 20 minutes (Figure 8.2).

In the heart, ischemia and infarction can be transmural, affecting the entire thickness of the myocardium, or subendocardial, affecting just the innermost part of the myocardium, the part just beneath the endocardium. Out of the four chambers of the heart, the ECG is most sensitive to transmural and subendocardial ischemia or infarction in the left ventricle because that's the chamber with the thickest walls; in other words, it has the most cardiac tissue (Figure 8.3).

Figure 8.1

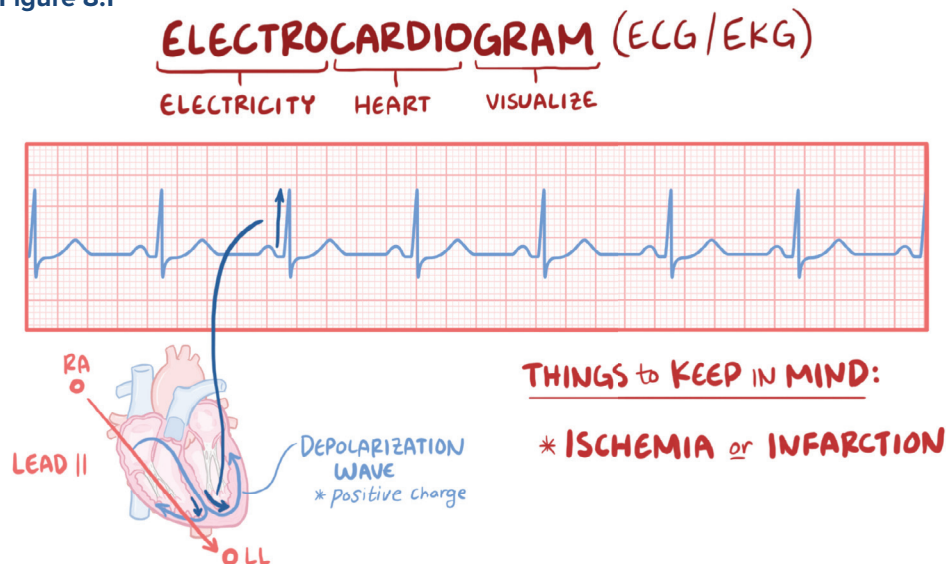


Figure 8.2

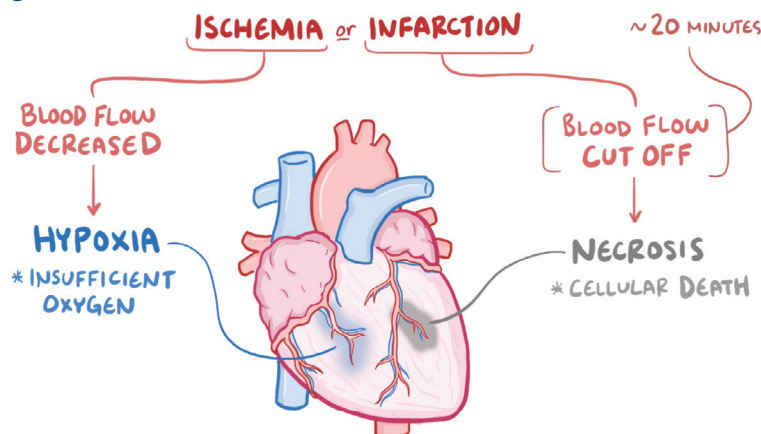


Figure 8.3

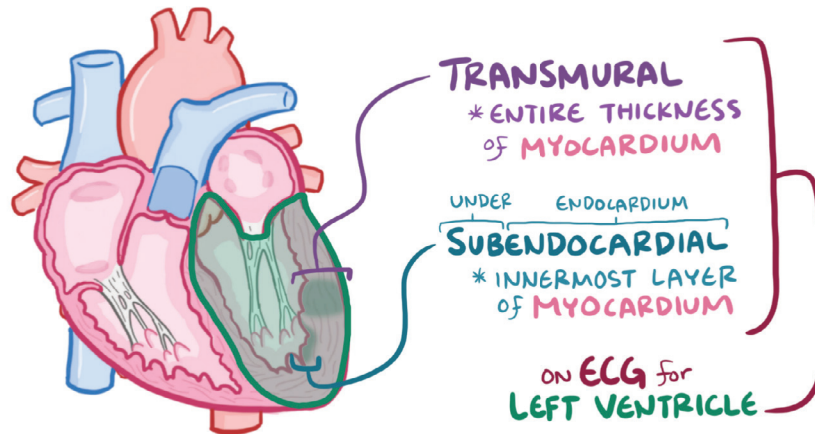


Figure 8.4

SUBENDOCARDIAL ISCHEMIA

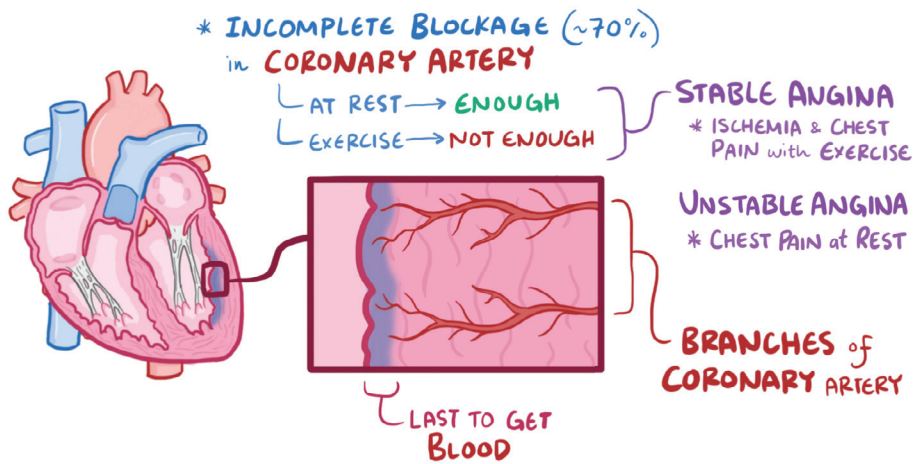


Figure 8.5

SUBENDOCARDIAL ISCHEMIA

* ST DEPRESSION *
└ J-POINT DOWN by ≥ 0.5 mm



SUBENDOCARDIAL ISCHEMIA

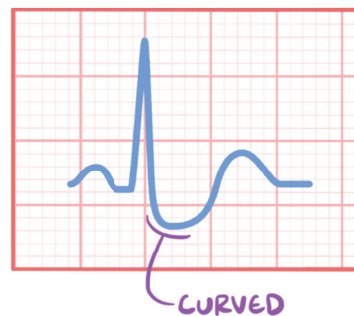
Subendocardial ischemia might happen when there's incomplete blockage, let's say 70%, in a coronary artery. In this situation, there's enough blood flowing through to meet the demand of the myocardium when the person is at rest, but during exercise, there's not enough to meet the increased demand of the myocardium. Therefore, the subendocardial tissue becomes ischemic because it's the last bit of tissue to receive blood from the tiny branches of the coronary arteries as they make their way through the ventricular wall, from outside to inside. This condition is called stable angina, because there's ischemia that causes chest pain with exercise, and the pain disappears with rest. Unstable angina can also occur with incomplete blockage of a coronary artery, but in this case, chest pain appears at rest (**Figure 8.4**).

When there's subendocardial ischemia in a region, it causes ST depressions in the corresponding lead on the ECG. An ST depression describes when the J point, which is where the QRS complex meets the ST segment, goes down by at least 0.5 mm or $\frac{1}{2}$ of a small box. The ST depression can be upward sloping, downward sloping, or horizontal (**Figure 8.5**). In subendocardial ischemia, ST depressions are usually widespread, and often affect leads I, II, and V_4 to V_6 . With such widespread involvement, it's difficult to determine which coronary artery caused the ST depression. As a side note, if the ST segment is depressed and curved, it may be due to the *digitalis effect*, which happens when the patient takes the medication digoxin, rather than being due to subendocardial ischemia (**Figure 8.6**).

Figure 8.6

DIGITALIS EFFECT

*From TAKING DIGOXIN



SUBENDOCARDIAL INFARCTION

On the other hand, subendocardial infarction occurs if the coronary artery remains blocked for around 20 minutes, and the subendocardial cells begin to die, which results in necrosis. With necrosis, the subendocardial cells leak cardiac enzymes, like troponins and CK-MB, into the blood. With a subendocardial infarction, there is no ST elevation on the ECG, so this is called a non-ST elevation myocardial infarction, or an NSTEMI for short. Instead, the ECG can show ST depression and T wave inversion. (**Figure 8.7**).

On an ECG, both unstable angina and NSTEMIs may show ST depression and T wave inversions. The difference is that with an NSTEMI, cardiac enzyme levels in the blood are elevated, whereas with unstable angina, the damage resolves in a short amount of time, so there is no cardiomyocyte death - and therefore no elevated cardiac enzymes. With an NSTEMI, the ST depressions look similar to the ones in subendocardial ischemia. The T wave inversions are usually symmetrical, and are at least 1 mm, or 1 little box, deep. They are most noticeable in the chest leads, but can also appear in the limb leads. They also have to occur in at least two contiguous leads. For example, lead V_2 and V_3 are contiguous, but leads V_3 and V_5 are not contiguous. There can also be a dominant R wave, which means that the R wave has a higher amplitude than the S wave. Now, it turns out that a little bit of T wave inversion can be normal in leads III, aVR, and V_1 , but any sign of T wave inversion in leads V_2 to V_6 is abnormal (**Figure 8.8**).

Figure 8.7

SUBENDOCARDIAL INFARCTION

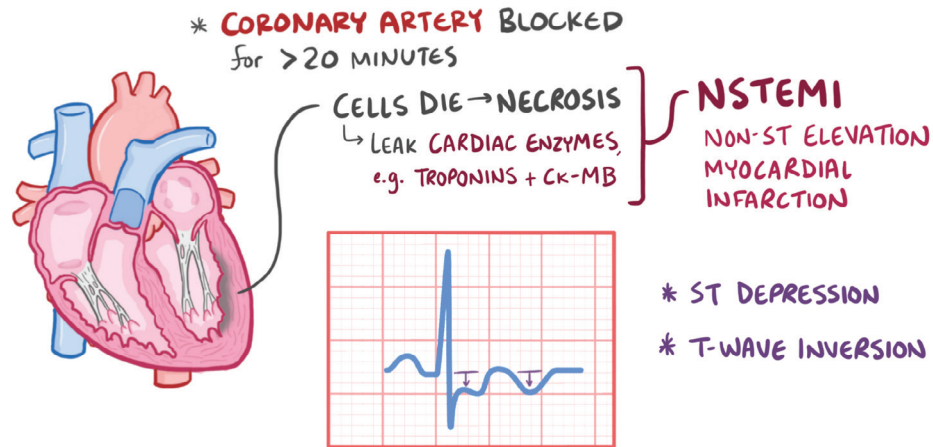
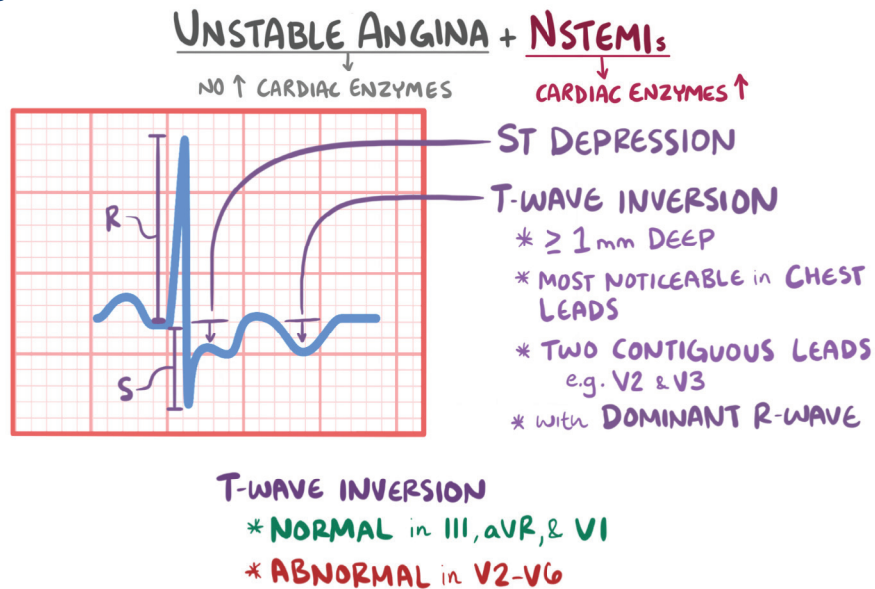


Figure 8.8



TRANSMURAL ISCHEMIA

Next, there's transmural ischemia. When a coronary artery is narrowed from an atherosclerotic plaque buildup over time, there are two parts to the plaque: a hard fibrous cap and the soft cheese-like interior. As blood flows past the plaque, the fibrous cap can sometimes rip. That cheesy interior is exposed and considered thrombogenic, meaning that blood clots form on it very quickly; these can quickly stop blood flow (**Figure 8.9**)! Transmural ischemia can occur secondary to vasospastic angina, also called Prinzmetal angina. This is when a coronary blood vessel spasms and narrows, cutting off blood flow to that cardiac region. Vasospastic angina occurs independently of traditional risk factors, like atherosclerosis, and can be triggered by tobacco, alcohol, or cocaine use (**Figure 8.10**).

Figure 8.9

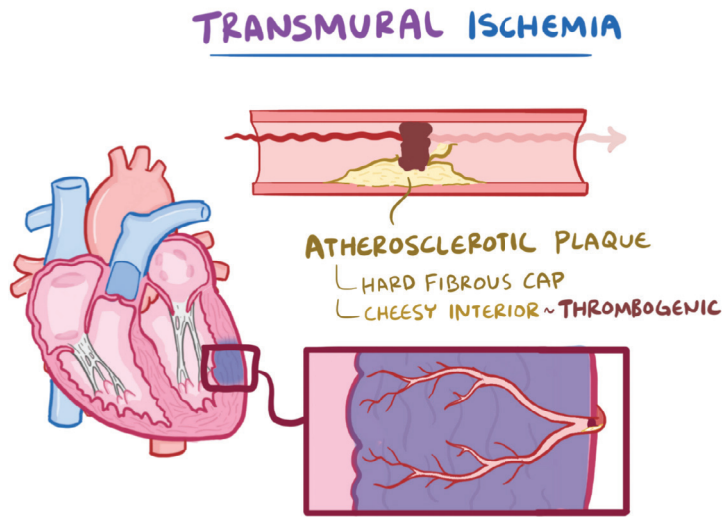
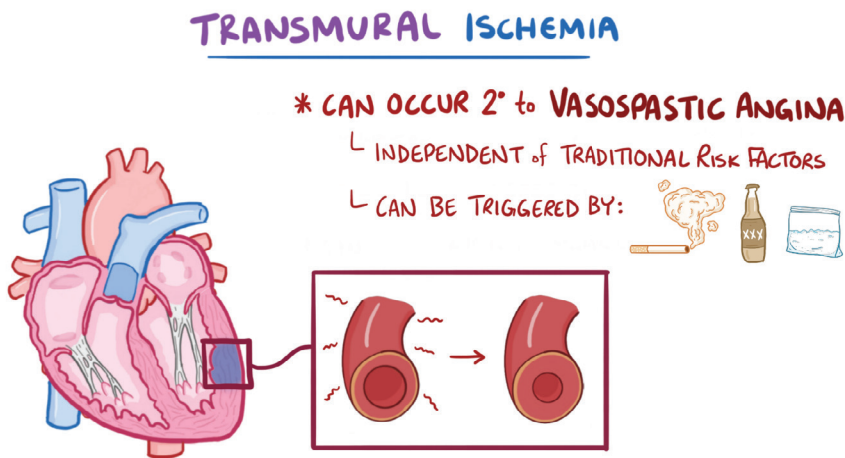


Figure 8.10



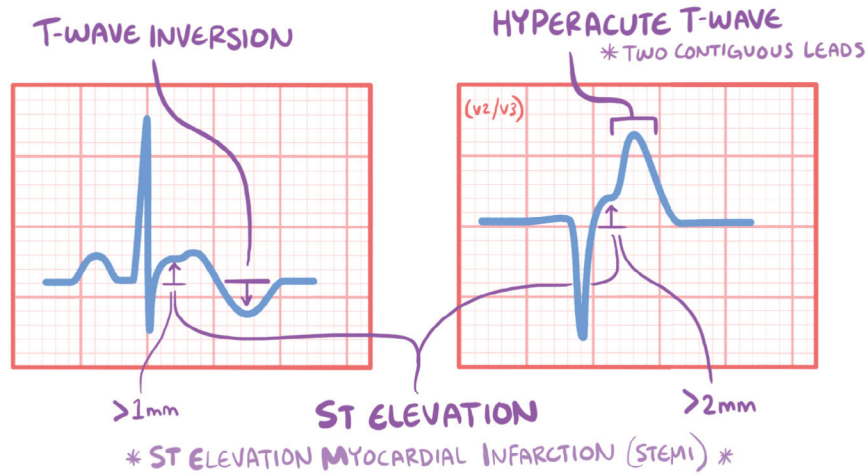
TRANSMURAL INFARCTION

Transmural infarction occurs when a coronary artery becomes completely blocked for greater than about 20 minutes, which causes transmural necrosis. Within minutes, a transmural infarction leads to ECG changes. The most common change is T wave inversions, but there might also be hyperacute T waves, which are large asymmetric T waves that appear in at least two contiguous leads. The classic sign of transmural infarction is ST elevation; when this happens, it's called an ST elevation myocardial infarction, or STEMI. On ECG, the ST elevation at the J point must be over 1 mm in any two contiguous leads, except V_2 or V_3 , where it has to be over 2 mm (**Figure 8.11**).

On an ECG, both a STEMI and vasospastic angina can cause ST elevation; however, with vasospastic angina, the elevation is transient. Another key difference is that, with a STEMI, the ischemia is so severe that damaged heart cells die - resulting in myocardial necrosis - and leak out certain enzymes, like troponin and CK-MB.

Figure 8.11

TRANSMURAL INFARCTION



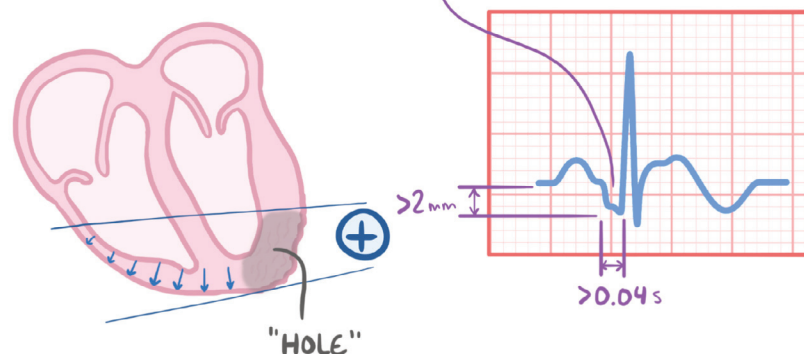
ST elevation can also be caused by other conditions, such as left ventricular hypertrophy and pericarditis. For example, a physically fit 20-year-old with a viral illness, who gradually develops chest pain and is found to have ST elevation on ECG, is more likely to have pericarditis than an ST elevation myocardial infarction.

Besides ST elevation and T wave inversions, a transmural infarction can also cause pathologic Q waves to appear on the ECG. Normally, depolarization in the ventricle spreads from the endocardium to the epicardium; most of this wave ends up pointing toward the positive electrode, which means a positive deflection. However, infarcted tissue doesn't conduct electricity, which means that the electrode essentially sees through the infarcted tissue, as if it was a hole, to the *other* ventricular wall. Therefore, the electrode sees the depolarization wave that moves through the healthy wall on the opposite side of the heart. Because those waves are mostly moving away from the positive electrode, you end up with a big, negative pathologic Q wave. So, pathologic Q waves are waves that are longer than 0.04 seconds, or one small box, and over 2 mm, or two small boxes, deep (**Figure 8.12**).

Figure 8.12

TRANSMURAL INFARCTION

* PATHOLOGIC Q-WAVES



The location of the transmural infarction can be identified based on the lead that has pathologic Q waves. Septal wall infarctions show changes in leads V₁ and V₂, whereas anterior wall infarctions show changes in leads V₃ and V₄. Anterolateral wall infarctions show up in leads I, V₃ through V₆, and aVL. Small subendocardial infarctions don't cause pathologic Q waves because some of the affected wall still conducts electricity. Also, the aVR lead is oriented in a way that normally produces Q waves, so it's not a reliable lead to look for pathologic Q waves. Finally, pathologic Q waves can also result from things other than transmural infarction, like a left bundle branch block or Wolff-Parkinson-White syndrome (Figure 8.13).

After a transmural myocardial infarction, the ECG gradually begins to look more normal. During the first two days there might be T wave inversion, ST segment elevation, and pathologic Q waves. Around day three, the ST segment is often back at baseline. After a few weeks or months, the T wave inversion may start to improve as well, but pathologic Q waves can linger for much longer. That's why it's always important to compare ECGs to previous ones to look for new changes (Figure 8.14).

Figure 8.13

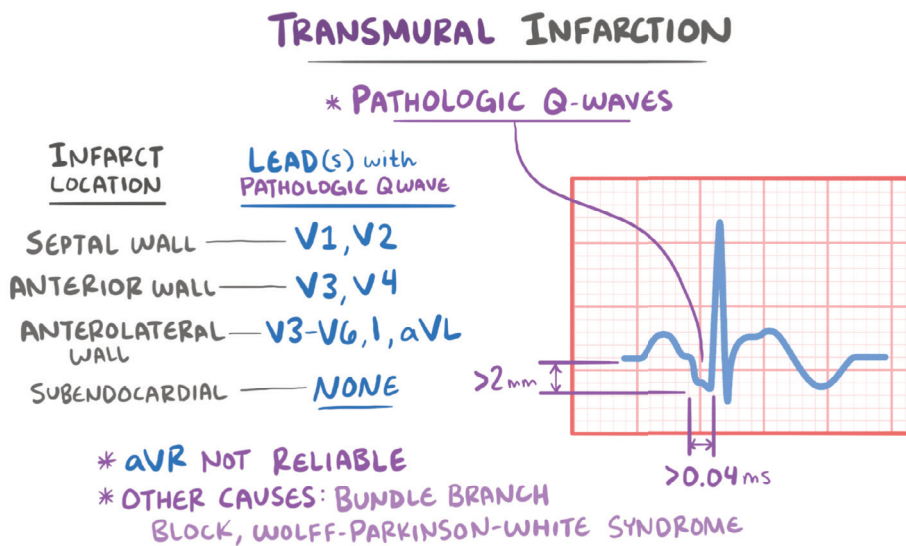
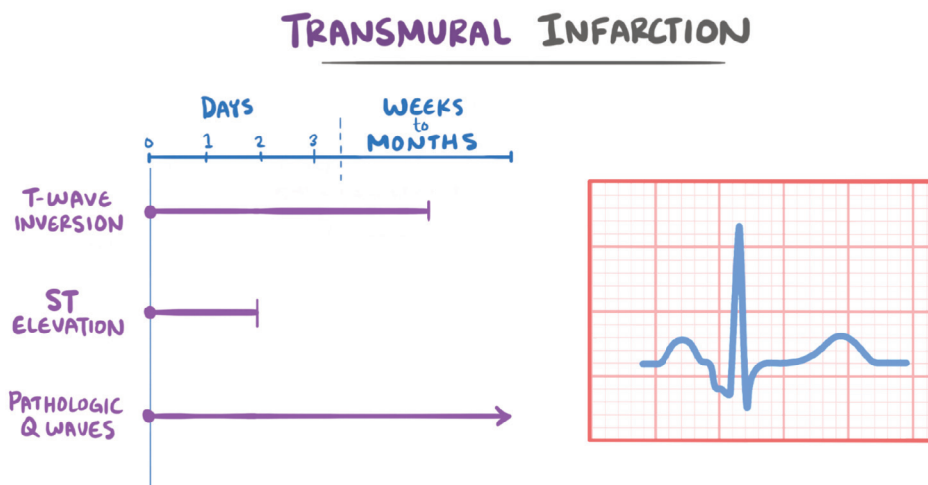


Figure 8.14



SUMMARY

All right, let's quickly recap. Subendocardial ischemia can reflect stable or unstable angina. Subendocardial infarction causes an NSTEMI. Stable angina, unstable angina, and NSTEMIs are associated with ST depressions and T wave inversions on an ECG; the difference is that, with NSTEMIs, there are elevated cardiac enzymes like CK-MB and troponins in the blood. Transmural ischemia can reflect vasospastic angina, whereas transmural infarction leads to a STEMI; this causes ST elevation on an ECG. The key difference is that with vasospastic angina, the ST elevation is transient, and with a STEMI, there are elevated cardiac enzymes in the blood. Additionally, with a STEMI, the ECG can show T wave inversions, hyperacute T waves, and pathologic Q waves (**Figure 8.15**).

Figure 8.15

SUBENDOCARDIAL		TRANSMURAL	
ISCHEMIA	INFARCTION	ISCHEMIA	INFARCTION
~ STABLE/UNSTABLE ANGINA	~ NSTEMI ↑ CARDIAC ENZYMES	~ VASOSPASTIC ANGINA	~ STEMI ↑ CARDIAC ENZYMES
~ ST DEPRESSIONS ~ T-WAVE INVERSIONS		ST ELEVATION	
		~ ST ELEVATION is TRANSIENT	~ T-WAVE INVERSIONS ~ HYPERACUTE T WAVES ~ PATHOLOGIC Q WAVES

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INDEX

Numbers in red indicate the term is defined on those pages.

A

amiodarone 39–40
anterior leads **15**
atrial depolarization 37–40, 44–48
atrial fibrillation **23**
atrial flutter **23**
atrial intranodal tracts. *See* Bachmann's bundle
atrioventricular node 19, 23–26, 27, 29–34, **33–34**, 37–40
augmented vector foot **15, 33–34**, 44–48
augmented vector left **15**, 33–34, 55
augmented vector right **15**, 33–34, 51, 55
AV block 25–26
aVF. *See* augmented vector foot
aVL. *See* augmented vector left
AV node. *See* atrioventricular node
aVR. *See* augmented vector right
axis **27**, 41–43, 51

B

Bachmann's bundle 19
Bazett's formula **39**
biphasic **44**
bipolar limb leads **15**
box method **21**
bundle branch block **25**
bundle of His 19, 27–34

C

chest leads **13**, 41–43
CK-MB 51–56
coronary artery vasospasm 52–53, 56
corrected QT interval 39–40

D

depolarization wave **11**, 11–16, 19–20, 28, 41–43, 54
diaphragm 29
digoxin 51
dilation **44**
dipole **11**

E

ectopic beat **25**
ectopic focus 23–26, 33–34, 37
electrode 46–48, 54
endocardium **29**, 49–56
enlargement **44**
epicardium **29**, 54
extreme right axis deviation **33**

F

first degree heart block **37**
functional syncytium **19**

H

hypertension 48
hypertrophic cardiomyopathy 29
hypertrophy 43, **44**, 46–48

I

inferior leads **15**
intervals **35–40**
ischemia **49**
isoelectric line **19**, 20

J

J point **20**, 50–51, 53

L

lateral leads **15**
lead I 15–16, 33–34, 51–55
lead II 11, 15–16, 17–20, 21–26, 33–34, 44–48, 51–55
lead III 15–16, 33–34, 44–48, 51–55
left atrial enlargement. *See* enlargement
left axis deviation **33**
left bundle branch 19–20
left bundle branch block 55
left ventricular hypertrophy 34, 54.
See hypertrophy
limb electrodes **13**
long QT syndrome 39

M

mean QRS vector **27**
myocardial infarction 43, **49–56**
myocardium **29**, 49–56

N

necrosis **49**, 51–53
non-ST elevation myocardial infarction **51–52**, 56

Index
NSTEMI. *See* non-ST elevation myocardial infarction

P

pacemaker cells 17–20, **19**
pericarditis 54
plaque **51**
precordial electrodes **13**
PR interval **19**, 35–40
pulmonary hypertension 46
Purkinje fibers 19–20, 27–34
P wave **19**, 20, 23–26, 28–34, 35–40, 44–48

Q

QRS complex **19–20**, 23–26, 28–34, 35–40, 41–43, 46–48, 51–56
QRS transition **41–43**
QRS transition zone **41**, 43
QRS vector 29–34, 33–34
QT interval 35–40
Q wave **19**, 54–56

R

rate and rhythm **21–26**
repolarization wave 13, 20
right atrial enlargement. *See* enlargement
right axis deviation **33**, 46–48
right bundle branch 19–20
right bundle branch block 46–48
right ventricular hypertrophy 34, 43.
See hypertrophy
R wave **19**, 21–26, 46–48, 51
R wave progression 43

S

SA node. *See* sinoatrial node
septal leads **15**
sinoatrial node **17**, 19–20, 23–26, 25, 27, 37–40
stable angina 50–**51**, 56
ST depression 50, **51**, 52, 56
ST elevation 48, 53–56
ST-elevation myocardial infarction **53–56**
STEMI. *See* ST-elevation myocardial infarction
stenotic tricuspid valve **44**
ST segment **20**
ST segment depression 48, 50, **51**, 52, 56
ST segment elevation 53–56

subendocardial infarction **51**–52,
55–56
subendocardial ischemia 50–**51**, 56
S wave **19**, 46–48, 51–52

T

thrombogenic **51**
torsades de pointes **39**
transition zone **34**
transmural infarction **53**–56
transmural ischemia **52**–53, 56
troponin 51–53, 56
T wave **20**, 23–26, 28–34, 35–40,
39–40, 48
T wave inversion 51–56

U

unstable angina 50–**51**, 56

V

V₁ 13–16, 34, 41–43, 44–48, 51–55
V₂ 15–16, 34, 43, 44–48, 51–55
V₃ 15–16, 34, 41–43, 51–55
V₄ 15–16, 34, 41–43, 51–55
V₅ 15–16, 34, 43, 46–48, 51–55
V₆ 13–16, 34, 41–43, 46–48, 51–55
ventricular depolarization 37–40

W

Wolff-Parkinson-White syndrome 55

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