Line Language
Part I

Basic Arrhythmia Recognition
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Welcome to Line Language Part I– Basic Arrhythmia Recognition. We designed this course to carry the participant beyond ACLS to a detailed discussion of the most common cardiac arrhythmias seen in clinical practice. We will explore the anatomy, physiology and pathophysiology behind cardiac rhythm disturbances to help you understand why they look like they do and some brief discussion about therapeutic interventions in the acute and chronic phases of care.

**COURSE OBJECTIVES**

Upon completion of this course, the participant will be able to:

- **DISCUSS BASIC CARDIOVASCULAR A & P**
- **DESCRIBE THE LAYOUT OF ECG PRINTER PAPER**
- **DESCRIBE THE SYSTEMATIC APPROACH TO RHYTHM ANALYSIS**
- **IDENTIFY BASIC CARDIC DYSRHYTHMIAS TO INCLUDE:**
  - SINUS DYSRHYTHMIAS
  - JUNCTINAL DYSRHYTHMIAS
  - VENTRICULAR DYSRHYTHMIAS
  - AV BLOCKS
  - PACEMAKER RHYTHMS

**ANATOMY & PHYSIOLOGY**

Cardiac Gross Anatomy

The heart is a muscular organ roughly the size of an adult male fist located just left of the midline, behind the sternum and between the lungs in an area called the mediastinum. How big the heart is and its exact position in the chest will change as someone ages, which is one of the biggest reasons that an ECG is so valuable for more than just ischemic findings.

The heart is divided into four chambers, two atria and two ventricles, which are labeled according to the side they’re on. For the sake of physiology, it is better to consider each side of the heart rather the each type of chamber.

**Right Heart**

The right side of the heart is made up of, in anatomical order, the right atria, the tricuspid atrio-ventricular valve, the right ventricle and the pulmonic semilunar valve. This side is typically referred to as a low pressure system because its primary function is to receive blood from systemic...
circulation and pump it into the low pressure pulmonary circulation.

The right atria is thin walled chamber of the heart that receives this deoxygenated blood via the venae cavae. The right atria is separated from the right ventricle by the tricuspid atrioventricular valve and is under low pressure, usually 0-8 mmHg.

The tricuspid valve is the first of two atrioventricular valves that, as the name implies, separate the atria from the ventricles. This valve in particular prevents back flow, or regurgitation, of blood from the right ventricle back into the right atria during systole. The integrity of the tricuspid valve is crucial to providing good right ventricular stroke volume. The AV valves are attached to structures called chordae tendinæa that attach to papillary muscles in the ventricular walls that pull the valves open when the ventricle relaxes. In other words, this valve is opened by ventricular diastole.

The right ventricle is a slightly more muscular chamber than the atria, located below the tricuspid valve with the primary job of pumping blood into the pulmonary circulation where it can be oxygenated and returned to the body via the left side of the heart. Because the pulmonary system is under relatively low pressure, right ventricular pressures are usually between 15-30 mmHg systolic.

The last part of the right heart is the pulmonary semilunar valve, named so because the leaflets resemble a half moon shape. Blood leaving the right ventricle exits through this valve to enter the pulmonary artery and eventually the capillaries in the lungs to be oxygenated and circulated back to the left side of the heart. The pulmonary valve (sometimes called the pulmonic valve) prevents backflow of blood from the pulmonary artery into the right atria. This valve is opened by ventricular systole.

The Left Heart

The left side of the heart, in anatomical order, is made up of the left atria, the bicuspid AV or mitral valve, the left ventricle and the aortic semilunar valve. The left heart is typically referred to as a high pressure system because it collectively pumps blood against the systemic vascular resistance, more commonly known as the blood pressure.

The left atria is a thin walled chamber of the heart that receives oxygenated blood from the pulmonary system via the pulmonary veins. The left atria is separated from the right atria by the atrial septum, and from the left ventricle by the bicuspid or mitral atrioventricular valve and is under relatively low pressure, usually 2-15 mmHg.
The bicuspid, or mitral valve, is the atrioventricular valve that separates the left atria from the left ventricle. The integrity of this valve is especially important to preventing regurgitation of blood from the left ventricle back into the left atria. Since this valve essentially seals the high pressure left ventricle during systole, it is prone to chronic failure in patients with hypertension. The mitral valve has the same attachment mechanism as the tricuspid valve, and thus also opens during ventricular diastole.

The left ventricle is the most muscular chamber of the heart because it must eject its blood, called the stroke volume, against the greatest pressure. Therefore, left ventricular pressures are normally between 100‐140 mmHg. In patients with chronic hypertension, the left ventricle may become overly muscular, a condition call hypertrophy, which may lead to dysfunction and ECG changes.

The final component of the left heart is the aortic semilunar valve which is tasked with preventing regurgitation of blood from the aorta back into the left ventricle.

**Drop of Blood**

One useful exercise, and a possible test question, to display understanding of cardiac anatomy is to trace a drop of blood from the time it enters the heart until it leaves. Fill in the missing blanks below using these anatomical features of the heart:

Pulmonary Vein → Pulmonary Artery → Aorta → Left Ventricle → Lungs → Right Atria → Left Atria → Mitral Valve → Right Ventricle → Pulmonary Valve → Tricuspid Valve → Aortic Valve → Pulmonary Artery → Aorta → Lungs → Left Atria → Right Ventricle → Mitral Valve → Tricuspid Valve → Pulmonary Valve → Aortic Valve

**Coronary Circulation**

Not only is the heart responsible for supplying blood to the rest of the body, but it must also circulate blood to itself via the coronary arteries. While coronary circulation is very complex, exams like the CCT only expect that you have a basic understanding of the major coronary arteries, the
ANATOMY & PHYSIOLOGY

Specific areas and anatomical structures that are supplied by each of these arteries and the like ECG changes indicative of occlusion or disease in the respective arteries.

It is important to note also that there are many physiologic variations in coronary circulation from person to person. Roughly 80% of the population has similar vascular structure in the heart, known as right heart dominance.

RIGHT CORONARY ARTERY
- SA NODE
- AV NODE
- RIGHT VENTRICLE
- INFERIOR WALL OF LEFT VENTRICLE
- POSTERIOR WALL OF LEFT VENTRICLE

LEFT ANTERIOR DESCENDING ARTERY
- ANTERIOR WALL OF LEFT VENTRICLE
- SEPTUM
- BUNDLE BRANCHES & HEMIFASCICLES
- MAJORITY OF PUMPING MASS
- LOWER LATERAL WALL

LEFT CIRCUMFLEX ARTERY
- UPPER LATERAL WALL
- SA NODE (45%)
- AV NODE (10%)
- POSTERIOR WALL (LHD)
ANATOMY & PHYSIOLOGY

Cardiac Conduction

In order for the heart to beat, like any other muscle, it must be stimulated by an impulse. In our other skeletal muscles, this impulse comes from our brain. But in cardiac contraction, this impulse comes from inside the heart itself through the cardiac conduction system. This system of specialized tissue embedded in the myocardium both generates and conducts the electrical stimuli necessary to make the heart muscle depolarize and in turn contract. The diagram below shows the name and relative position of the major parts of the conduction system.

- **Sinoatrial (SA) Node:** The primary pacemaker of the heart, the SA node is buried in the wall of the right atrium. In normal hearts, the impulse which triggers myocardial depolarization originates here and travels through the rest of the heart in a predictable time period. The normal intrinsic firing rate of the SA node is 60-100 beats per minute. This gives us the term we use frequently to describe the heart’s normal electrical function: Normal Sinus Rhythm.

- **Intranodal pathways & Bachmann’s Bundle:** From the SA node, the impulse is conducted via these pathways to the left atria (Bachmann’s) and through the rest of the right atria to the AV junction (intranodal pathways).

- **Atrioventricular (AV) Node/Junction:** The primary role of the AV junction is to delay the impulse coming from the SA node long enough for the ventricles to fill before allowing it to progress into the ventricular conduction system. The secondary function of the AV node is to act as a backup pacemaker for the heart should the SA node fail to function properly. This is where we get the term “junctional rhythm”. The intrinsic depolarization rate for the AV node is 40-60 beats per minute.
ANATOMY & PHYSIOLOGY

Cardiac Conduction

- **Bundle of His**: This main conduction channel descends directly from the AV junction into the superior ventricular septum for a very short distance before bifurcating into the right and left bundle branches.

- **Bundle Branches**: Because the left ventricle is so big, it requires additional conduction pathways to ensure a crisp depolarization and contraction. The right bundle branch conducts the impulses to the right side of the left ventricle as well as the right ventricle. The left bundle branch splits into the anterior and posterior hemifascicles in order to even better enervate the anterior and lateral left ventricular myocardium.

- **Purkinje Fibers**: The mass of the ventricular myocardium is laced with this network of conductive fibers that distribute the wave of depolarization evenly. This network is also capable of assuming a backup pacemaker role should the others fail to function. The intrinsic depolarization rate of the Purkinje system is 20-40 bpm.

Tracing the Current

Similar to the “drop of blood” exercise, the ability to trace an impulse from beginning to end is also important and may show up on an exam in some form or fashion. Fill in the anatomical order of depolarization from the beginning to the end using the terms below:

- Bundle of His
- Purkinje Fibers
- Bundle Branches
- Intranodal Pathways
- AV Junction
- Hemifascicles
- SA Node

_________→_________→_________→_________→_________→_________→_________.

Cardiac Tissue Layers

The cardiac tissue itself is divided into three layers. From outside to inside, there is the pericardium, the myocardium and the endocardium.
ANATOMY & PHYSIOLOGY

The pericardium itself is divided into the parietal pericardium which acts as a protective sac around the heart, and the visceral pericardium which lines the outside wall of the organ itself. The myocardium is the thick, muscular layer that provides the contractile force that pumps the blood. This is the layer most affected by a heart attack (hence the term “myocardial infarction”). The endocardium is the inner lining of the chambers of the heart that serves to protect the chambers and valves from chemical and osmotic changes.

Extracardiac anatomy

As stated before, the heart lies in a region of the body called the mediastinum. This region also contains the superior and inferior venae cavae, the aorta, the pulmonary artery and the pulmonary veins; collectively referred to as the great vessels. The venae cavae are the largest veins in the body, returning all the deoxygenated blood from systemic circulation to the heart via the right atria. The aorta is the largest artery, carrying all the oxygenated blood from the left ventricle to the rest of the body. The pulmonary artery takes blood from the right ventricle to the lungs for oxygenation while the pulmonary veins receive the blood from the lungs to return to the heart via the left atria. Changes in the tone or volume of any of these vessels can change how the heart functions by altering the pressure needed to fill or empty the chambers.

Physiology

There are two fundamentally important pieces of cardiac physiology to remember: electrophysiology and nervous system control. The first is concerned with how the myocardium itself functions while the second regulates how the heart can meet the body’s metabolic needs.

Cardiac electrophysiology explains how the myocardium responds to electrical stimuli to produce a physical contraction that will ideally pump blood.

The words most commonly used in association with electrophysiology are depolarization and repolarization, and they refer to the movement of electrolytes into and out of myocardial cells that change their chemical makeup and, ideally, cause them to contract and relax. The simple relationship between electrical and mechanical physiology is:

DEPOLARIZATION = CONTRACTION

REPOLARIZATION = RELAXATION
Some other words you should be familiar with pertaining to electrophysiology are:

- **Systole**: contractile phase of cardiac cycle resulting from depolarization.
- **Diastole**: relaxation phase of cardiac cycle during which repolarization is occurring and the ventricles are filling with blood.
- **Conductivity**: Ability of tissues or cells to carry an electrical impulse.
- **Excitability**: Ability of tissues or cells to respond to electrical impulse.
- **Automaticity**: Ability of tissues or cells to generate an electrical impulse.

Myocardial cells are unique in the body as they possess the characteristics of conductivity, excitability and automaticity. Essentially this means that any part of the heart muscle can act as the pacemaker, pathway or pump depending on the function of other areas.

Another word to remember is refractory. This means simple that the heart is not responding to the indicated therapy. In other words, if a patient is in cardiac arrest and remains in VFIB despite repeated countershocks, then we would say that they are refractory to defibrillation.

When it comes to electrical control and mechanical function, the ultimate goal is to pump enough blood to feed and oxygenate the body tissues. The collective blood required to do this called the cardiac output and is usually about 5-6 liters/minute in the average resting adult. The components of cardiac output are very simple to define and are expressed in this equation:

\[
\text{CARDIAC OUTPUT (CO)} = \text{STROKE VOLUME (SV)} \times \text{HEART RATE (HR)}
\]

The heart rate is self explanatory. The **stroke volume** is the amount of blood (in ml) ejected by the left ventricle with each contraction. The average adult male at rest has a stroke volume of about 70ml, which represents about 60% of the blood in the ventricle. This percentage is known as the **ejection fraction** and represents the efficiency of the heart at pumping the blood that fills it.

How the body regulates cardiac output involves the other central part of cardiac physiology: nervous system control. This piece is particularly important because it not only explains how we regulate our cardiac function internally, but also how we use medications to improve or inhibit those functions.
ANATOMY & PHYSIOLOGY

Because the heart is capable of generating its own impulses, it can function with little or no input from brain at all. However, the autonomic nervous system (ANS) goes a long way to regulate heart rate and vascular tone to produce optimal cardiac output based on metabolic needs. The ANS is further divided into the sympathetic and parasympathetic nervous systems. The sympathetic division, commonly referred to as “fight or flight” is responsible for increasing cardiac output in response to exercise or stressful situations. Increases in sympathetic tone cause an increase in heart rate and vasoconstriction to improve cellular oxygenation and the elimination of waste products from metabolism like carbon dioxide. Parasympathetic stimulation, commonly referred to “feed and breed” results in decreases in heart rate and vascular tone with increased blood flow to digestive and reproductive organs. The major nerve component of the parasympathetic division is the vagus nerve. Many actions and medications either incidentally or purposefully exert action on the vagus nerve to either stimulate or suppress its function.
The fundamentals of the diagnostic value of an ECG are based on, quite literally, the paper it is printed on. More specifically the paper, the size of the gridlines and how fast the paper comes out of the machine during the recording. This also means that, in order to do a quality rhythm analysis, the ECG strip must be printed and the complexes on a display screen are compressed and not to scale.

All over North America, ECGs are printed on the same type of paper in very similar formats. In the hospital or clinical setting, the paper is roughly letter size (8.5” x 11”) and prints in a landscape (horizontal) orientation similar to this page. The gridlines, or squares, on the paper like those on this page are of a specific size and can be used to rapidly measure things about the ECG. The horizontal (left to right) measurements are those of time. By counting the number of these blocks between certain waves, we can calculate things like heart rate (HR), PR interval, QT interval and QRS duration. These measurements are based on the size of the blocks and, perhaps most importantly, how fast the paper comes out of the machine while printing. Standard paper speed is:

25 mm/sec

This speed is necessary for the block measurements coming up, and the block measurements are necessary for the time and other numbers after that. Another value of having a standard paper speed is that some machines allow the technician to double the speed to 50 mm/sec, which will put the QRS complexes twice as far apart. This is useful in the case of ultrafast heart rates where rhythm regularity is a crucial assessment but is hard to determine because of the rate. This is a special feature not all machines are capable of and should only be used at the direction of the ordering provider.
MEASUREMENTS AND PAPER

Since the horizontal squares represent time, they can be measured in seconds or milliseconds. You may see either depending on your practice environment or how the ECG machine you use most prints measurements.

You can see that the gridlines on standard ECG paper are of two different shades, the small blocks are lighter while the larger boxes are heavier (see below).

The small, lighter boxes measure 0.04 seconds across at standard paper speed. The larger, heavier box, which is made of five small boxes, is 0.2 seconds in duration, again at standard paper speed.

The vertical boxes on the ECG paper represent measurements of voltage and amplitude and are not affected by the paper speed. We use these boxes to measure things like QRS amplitude, Q wave depth and perhaps most importantly, ST segment elevation and depression. The standard unit for these blocks are millimeters (mm), though they can also be expressed in millivolts (mV). The small, lighter boxes measure 1 mm or 0.1 mV. The larger, heavier box, which is made of five small boxes, is 5 mm or 0.5 mV in amplitude. Again, amplitude/voltage measurements are not affected by paper speed.
ECG LABELING

LABELING

Once the electrodes are applied and the ECG is printed, interpretation begins with simple recognition of the waves, or parts, of the ECG waveform. These are labeled the same regardless of whether you’re looking at a rhythm strip or a 12 lead ECG. Routine rhythm analysis requires you to be able to calculate different times such as the PR interval and QRS duration, so we will review the different waves as well as the normal measurements.

P-WAVE: Represents ____________________ First deflection from the baseline. It can be positive, negative, biphasic or absent.

Q-WAVE: Represents ____________________ First negative deflection after the P wave.

R-WAVE: Represents ____________________ First positive deflection after the P wave.

S-WAVE: Represents ____________________ First negative deflection after the R wave.

T-WAVE: Represents ____________________ Final deflection after the QRS complex. The T wave can be positive, negative, biphasic or appear absent.

Depending on the lead viewed, any or all of these waves may be present, and their specific appearance can give vital clues to underlying acute or chronic pathology.

ISOELECTRIC LINE: This baseline is the reference point from which we measure deviations such as elevation, depression and wave amplitudes. It is most accurately located by finding the bottom of the calibration spike at the beginning of each individual ECG.
ECG CURRENT, POLARITY, AND POINT OF VIEW

One of the most important concepts for reviewing ECG is comprehending how the machine represents the direction of current travel in each lead. In other words, each of the individual leads will “see” the electrical activity from a different point of view and present it accordingly on the printed ECG. These points of view are the result of the positive and negative poles of each lead and how the direction of travel of the electrical current relates to the positive (+) pole in each lead. This relationship can be most simply put this way:

**CURRENT TOWARDS A POSITIVE (+) ELECTRODE = POSITIVE ECG WAVE**

**CURRENT AWAY FROM A POSITIVE (+) ELECTRODE = NEGATIVE ECG WAVE**

This very simple idea, also called Mean Electrical Vector, is essential to helping us understand much of the diagnostic value of an ECG as well as assisting our efforts to troubleshoot the tracing and assure its validity; two things we’ll cover later in the course. First, let’s explore what a normal ECG looks like based on this idea.

**BI-POLAR LIMB LEADS**

The limb leads, I, II and III, make up what is commonly known as Einthoven’s Triangle. This is named for Dr. Willem Einthoven, a Dutch physiologist who pioneered and won the Nobel Prize for early work with ECG. By placing his arms and left leg in buckets of salt water, Dr. Einthoven was able to use a special machine to see and measure the heart’s electrical activity. These leads are labeled bi-polar because each lead has a positive and negative electrode.

**ECG CURRENT, POLARITY AND POINT OF VIEW**

Lead I: (+)____________________(-)______________

Lead II: (+)____________________(-)______________

Lead III (+)____________________(-)______________

![Einthoven’s Triangle](image)
ECG CURRENT, POLARITY, AND POINT OF VIEW

AUGMENTED VECTOR (aV) LEADS

The aV leads uses the existing electrodes from Einthoven’s Triangle to provide three additional views of the heart. These points of view are useful for confirming the diagnosis of STEMI, ventricular diagnosis or many other conditions. The positive pole for each lead is one of the limb electrodes. There is no physical negative electrode.

avR (+)____________________________

aVL (+)____________________________

aVF (+)____________________________

Einthoven’s Triangle

ELECTRODE PLACEMENT AND RHYTHMS

In order to assess a patient for things like STEMI, it is necessary to perform a diagnostic ECG with very precise electrode placement. For basic rhythm interpretation; however, they can be placed in such a way that the heart lies between them so it will generate a simple, single lead strip.

ARTIFACT TROUBLESHOOTING

There are many factors that can interfere with the quality of the ECG tracing and render it difficult or impossible to read. If this interference, called artifact, is too severe, the ECG may fail to print or the machine may fail to analyze or measure it properly. Causes of artifact include:

Somatic Tremor: This is simply the patient shaking. It can be caused by shivering from cold or anxiety, voluntary movement or involuntary movement from nervous conditions or ailments like Parkinson’s Disease.

Wandering Baseline: If the ECG displays a rhythmic variation or “wavy” baseline, the most likely cause is one of the electrodes for the affected lead is near the diaphragm. Slight movement of this electrode should fix this problem.

Electrical Interference: Sometimes called “60 cycle” or 60 mHz” artifact, this distortion is caused by an electrical source like the hospital bed or other medical equipment. It is visible on the ECG and a dark, wide and often blurry baseline. Turning off the offending equipment (unplug the bed) usually eliminates the interference.

Artifact in a single lead can frequently be traced back to a faulty wire, a dry or damaged electrode or poorly prepped skin.
RHYTHM ANALYSIS

Analyzing the ECG rhythm begins with assessing the heart rate and calculating the time relationships of several of the waves within the cardiac cycle.

Heart Rate: Several methods exist for calculating the heart rate if the machine calculation is not available.

6 second strip: Count the number of QRS complexes within 6 seconds of ECG strip. Multiply this number by 10. Six seconds of strip is 30 large horizontal blocks.

R to R: Count the number of small blocks (millimeters) between the peaks of two R waves (or S waves) Divide 1500 by this number. This method is derived from the printer paper speed of 25 mm/second which equals 1500 mm/minute.

Both methods are somewhat dependent on a grossly regular rhythm to accurately measure the heart rate. For irregular rhythms, a longer piece of strip may be necessary.

Intervals: The relationship between the waves within a single cardiac cycle are important for finding certain potentially dangerous conditions.

P-R Interval: The distance from the beginning of the P wave to the beginning of the R wave. This represents conduction time from the SA node through the AV junction. Normal PR interval is .12 to .20 seconds. Prolongation of the PR interval indicates delayed supraventricular conduction.

QRS Duration: The width of the QRS complex from the beginning of the Q (or R or S) wave to the J point. This represents the duration of ventricular depolarization. Normal QRS duration is .04 to .12 seconds. A QRS duration longer than normal indicates prolonged depolarization. This can be caused by an intraventricular conduction delay like a bundle branch block, or a more malignant condition like ventricular tachycardia.

QT/QTc: The time between the beginning of the QRS complex and the end of the T wave. This represents the total time required for the ventricular conduction cycle, both depolarization and repolarization. This measurement is physically related to the heart rate, so in order for it to be clinically relevant, it must be adjusted (corrected) for the heart rate. When the ECG prints from the machine, you will see two measurements: the QT and the QTc. The QT is the actual measured time as described. The machine will then apply a mathematical algorithm that accounts for the heart rate and calculates the QTc, or QT corrected. Normal QTc for heart rates between 60-100 is less than 440 ms. Prolonged QTc can result from medications or congenital disease and increases the risk of lethal dysrhythmia.
RHYTHM ANALYSIS

R-R Interval: As previously discussed, this is the time (or distance) from the peak of one R (or S) wave to the next. This measurement is useful for calculating the heart rate.

Identifying the Rhythm

It is important to use a systematic approach to analyzing and identifying the patient’s cardiac rhythm. It is equally important to use this approach EVERY SINGLE TIME so that important elements are not missed in the interpretation. It is easy to let a wildly abnormal feature such as extreme tachycardia, wide QRS or big ST elevation draw all of our attention, but as professionals we must concentrate on all the elements of the rhythm to provide the most accurate analysis.

SYSTEMATIC APPROACH

This is one example of a way to approach rhythm identification. If you have another method that incorporates all the elements, by all means stick with that.

1. Count the rate (atrial & ventricular)
2. Examine the QRS
3. Regular vs. irregular
4. Find the P wave (F/f wave)
5. Measure the PR interval
6. Determine the source
7. NAME IT!

Sinus Rhythm/Dysrhythmias

Those rhythms that originate in the SA, or sinoatrial node, feature a normal, rounded and upright P wave in lead II and are collectively referred to as “sinus”. Within this classification you will find normal sinus rhythm, sinus bradycardia, sinus tachycardia, sinus arrhythmia and sinus arrest. Each of these rhythms has unique characteristics, but all assume the rhythm originates in the SA node, is grossly regular, has a P wave for every QRS and the QRS is narrow (with the exception of bundle branch block which will be covered later and does not disqualify a rhythm from being sinus)
RHYTHM ANALYSIS

SYSTEMATIC APPROACH

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Normal Sinus Rhythm Criteria:

- Ventricular Rate 60-100
- Atrial Rate same
- Regular rhythm
- P wave present, rounded and upright in Lead II
- PR interval .12-.20 seconds
- QRS duration: .04-.12 seconds

Sinus Bradycardia Criteria:

- Ventricular Rate <60
- Atrial Rate same
- Regular rhythm
- P wave present, rounded and upright in Lead II
- PR interval .12-.20 seconds
- QRS duration: .04-.12 seconds
Sinus Tachycardia Criteria:

- Ventricular Rate >100 (usually less than 150)
- Atrial Rate same
- Regular rhythm
- P wave present, rounded and upright in Lead II
- PR interval .12-.20 seconds
- QRS duration: .04-.12 seconds

Sinus Arrhythmia Criteria:

- Ventricular Rate 60-100 (may fall outside that range briefly, but returns)
- Atrial rate same
- Irregular rhythm, usually within the respiratory cycle
- P wave present, rounded and upright in Lead II
- PR interval .12-.20 seconds
- QRS duration: .04-.12 seconds
Sinus Arrest Criteria:

- Ventricular Rate 60-100 (sometimes less)
- Atrial Rate same
- Regular rhythm except at time of arrhythmia
- P wave present, rounded and upright in Lead II (absent on missed beat)
- PR interval .12-.20 seconds
- QRS duration: .04-.12 seconds

Atrial Dysrhythmias

Some rhythms may look similar to a sinus rhythm but actually originate from somewhere else in the atria or near the AV junction. These are usually referred to as “atrial”, “supraventricular” or “junctional” and feature inverted, biphasic or absent P waves. Instead of P waves, they may feature either flutter waves (atrial flutter) or fibrillation waves (atrial fibrillation) which lend their characteristic names. These rhythms are usually regular, with the exception of atrial fibrillation, which is by criteria irregular. These rhythms include: wandering atrial pacemaker, premature atrial contractions, atrial/supraventricular tachycardia, atrial fibrillation with fast and slow ventricular response, and atrial flutter with variable conduction

Wandering Atrial Pacemaker

Sometimes the primary pacemaker of the heart will shift between the SA node and one or more ectopic foci somewhere in the atria. This results in an irregular appearance of the P waves from one beat to the next and often a somewhat irregular rhythm. The P waves will appear upright, inverted, biphasic and sometimes absent.
Wandering Atrial Pacemaker Criteria:

- Ventricular Rate 60-100 (sometimes less)
- Atrial Rate same
- Irregular rhythm with periods of regularity
- P waves change shape and direction, but usually precede every QRS
- PR interval .12-.20 seconds
- QRS duration: .04-.12 seconds

Premature Atrial Contractions

Sometimes an irritable focus in the atria will generate an impulse of sufficient amplitude to depolarize the myocardium outside the intrinsic rhythm. These complexes occur irregularly and usually appear similar to the underlying sinus rhythm since they originate within the atria. They can occur singularly or in pairs (couplets) or in regular proportion to normal beats such as every other beat (bigeminy) or every third beat (trigeminy). If the ectopic beats have more than one shape, this means they originate from different places within the atria and are said to be multifocal.
PAC Criteria

- Ventricular Rate 60-100 (sometimes less)
- Atrial Rate same
- Regular rhythm except at time of ectopy, followed by compensatory pause
- P waves normal for intrinsic, different morphology for ectopic beat
- PR interval .12-.20 seconds
- QRS duration: .04-.12 seconds

Atrial/Supraventricular Tachycardia

Tachycardias that originate in the atria but outside the SA node are referred to as either atrial or supraventricular tachycardia. Sometimes the word paroxysmal is used if the tachycardia begins and terminates spontaneously without intervention.

Atrial/Supraventricular Tachycardia Criteria

- Ventricular Rate >100 (usually greater than 180, sometimes as fast as 300)
- Atrial Rate same
- Regular rhythm
- P waves abnormal and often buried
- PR interval .12-.20 seconds if visible
- QRS duration: .04-.12 seconds

Supraventricular Tachycardia

HR__________________
Atrial Fibrillation

Atrial fib is a characteristically irregular rhythm that originates from multiple foci in the atria, usually at or around the pulmonary veins. It is quite common, especially in diseased hearts, and becomes dangerous when the rate of ventricular conduction increases. This is known as rapid ventricular response, or RVR for short. Well controlled atrial fibrillation is usually said to have slow or normal ventricular response.

Atrial Fibrillation Criteria:

- Ventricular Rate <60 to >100 (dependent on ventricular response)
- Atrial Rate 300-1200
- Irregular rhythm (often characterized as irregularly irregular)
- P wave absent, replaced by fibrillation waves
- PR interval absent
- QRS duration: Usually .04-.12 seconds, but may widen with poor conduction
Atrial Flutter

Atrial Flutter originates from a single focus inside the atrial that depolarizes faster than the SA node and this acts as the primary pacemaker. The hallmark of atrial flutter is the “sawtooth” appearance of the flutter waves occurring in relatively predictable proportion to the QRS complexes. A flutter is often named by this conduction ratio (i.e: Atrial flutter with a 4:1 conduction)

Atrial Flutter Criteria:

- Ventricular Rate <60 to >100 (dependent on ventricular response)
- Atrial Rate 240-300, sometimes greater
- Regular rhythm if the conduction ratio is constant
- P wave absent, replaced by flutter waves
- PR interval absent
- QRS duration: Usually .04-.12 seconds, but may widen with poor conduction

Junctional Dysrhythmias

Junctional rhythms or complexes originate from the AV junction either because of irritability/ischemia or because the primary pacemaker, the SA node, had failed to depolarize causing the AV junction to act at the escape pacemaker for the heart. This dysrhythmias include premature junctional contractions (PJC), junctional escape rhythm, accelerated junctional rhythm and junctional tachycardia.
Premature Junctional Complexes

- Ventricular Rate: Underlying rhythm
- Atrial Rate: Same
- Regular rhythm outside of ectopic beat
- P wave normal for underlying rhythm, absent or inverted for ectopy
- PR interval .12-.20 seconds
- QRS duration: Usually .04-.12 seconds for underlying rhythm, PJC may feature widened QRS if unusually conducted.

Sinus with PJC

Junctional Rhythm Criteria

- Ventricular Rate: 40-60
- Atrial Rate: 0
- Regular rhythm
- P wave absent or inverted
- PR interval absent or shortened
- QRS duration: Usually .04-.12 seconds for underlying rhythm, may be slightly widened.
Junctional Tachycardia/Accelerated Junctional Rhythm Criteria:

- Ventricular Rate: 60-150
- Atrial Rate: Absent
- Regular rhythm
- P wave inverted or absent
- PR interval <.12 seconds if present
- QRS duration: Usually .04-.12 seconds, may be slightly widened.

Ventricular Rhythms

Because of delayed conduction, rhythms of ventricular origin feature a widened QRS and are usually either fast (V tach), slow (ideoventricular) or disorganized (V fib). At times, an irritable focus (or multiple foci) will generate ectopic beats called premature ventricular contractions. If the ventricles have taken over as the pacemaker due to failure of the SA and AV nodes to depolarize, this is known as ventricular escape. P waves may be visible, but they will have no relationship to the underlying ventricular rhythm. Sometimes only P waves are visible without conducted QRS complexes, a rhythm known as ventricular standstill.
Premature Ventricular Contraction (PVC) Criteria:

- Ventricular Rate: Underlying rhythm
- Atrial Rate: Underlying rhythm
- Rhythm consistent with underlying, irregular on the ectopic beat
- P wave consistent with underlying, absent on PVC
- PR interval consistent with underlying, absent on PVC
- QRS duration consistent with underlying, > .12 seconds on PVC.

Sinus with bigeminy and couplet PVC
HR__________________

Atrial fib with multifocal PVC
HR__________________
Ventricular Escape Rhythm (idioventricular) Criteria:

- Ventricular Rate 20-40
- Atrial rate usually absent or completely unrelated to ventricular rhythm
- Regular rhythm
- P wave absent or hidden.
- PR interval absent
- QRS duration: >.12 seconds

Ventricular Tachycardia Criteria:

- Ventricular Rate 120-150
- Atrial rate usually absent or completely unrelated to ventricular rhythm
- Regular rhythm
- P wave absent or hidden.
- PR interval absent
- QRS duration: >.12 seconds
Ventricular Fibrillation Criteria

- Ventricular Rate Unknown
- Atrial rate Unknown
- Grossly Irregular rhythm
- P wave absent or hidden.
- PR interval absent
- QRS duration: unknown

Ventricular Standstill Criteria:

- Ventricular Rate absent
- Atrial rate usually 60-100
- Regular atrial rhythm
- P wave usually normal
- PR interval absent
- QRS duration absent
AV Blocks

When an impulse is generated normally from the SA node, it conducts through the AV junction down to the ventricles in a predictable fashion. If this conduction is delayed or fails all together, this is known as an atrioventricular (AV) block. There are several different grades of AV block, designated by degrees and each more severe that the preceding one. These blocks include a 1°, 2° Type I, 2° Type II and complete or 3° heart block.

1° AV Block Criteria:

- Ventricular Rate usually 60-100
- Atrial rate same
- Regular rhythm
- P wave usually normal
- PR interval >.200 seconds
- QRS duration 0.04—0.12 seconds

2° Type I AV Block (Wenckebach) Criteria:

- Ventricular Rate usually 60-100
- Atrial rate slightly greater than ventricular
- Irregular rhythm
- P wave usually normal
- PR interval >.200 seconds and variable, getting longer until a complete depolarization is missed
- QRS duration 0.04—0.12 seconds
2° Type II AV Block Criteria:

- Ventricular Rate usually 60-100
- Atrial rate slightly greater than ventricular
- Irregular rhythm
- P wave usually normal
- PR interval >.200 seconds and usually fixed, occasionally dropping a complete ventricular depolarization
- QRS duration 0.04—0.12 seconds

3° AV Block (Complete Heart Block) Criteria:

- Ventricular Rate dependent on escape pacemaker
- Atrial rate usually 60-100
- Usually regular rhythm
- P wave usually normal but not related to QRS
- PR interval completely unrelated to QRS, irregular
- QRS duration dependent on escape pacemaker
Asystole Criteria:

- Ventricular Rate absent
- Atrial rate absent
- No rhythm
- P wave absent
- PR interval absent
- QRS duration absent

PACEMAKERS

No discussion of cardiac rhythm is complete without mention of artificially generated rhythms, or pacemakers. Implantable pacemaker/defibrillators are indicated if the patient has the potential for lethal heart rhythms that require fixed or demand pacing and/or defibrillation. The cause can either be organic or a result of other treatment like SA nodal ablation.

Pacemakers are programmed based on what chamber they pace, what chamber they sense, what action they take when the sensor is activated, and what variable effect the pacemaker may exert on the heart rate.

Programming language:

Pacing— An impulse generated at a specific voltage via the lead wire to stimulate a depolarization.

Sensing— The pacemaker’s ability to sense native electrical activity or the absence thereof.

Response to Sensing: Indicates the pacemaker’s response to sensed activity, either to inhibit pacing or to trigger it. This chart illustrates the common options:
Common Pacing Modes:

AAI—Paces and senses in the atria. If there is native atrial activity, the pacing is inhibited.

VVI—Paces and sense in the ventricle. If there is native ventricular activity, the pacing is inhibited.

DDD—Dual chamber pacing and sensing. The most common pacing mode as it can sense and respond to impulses in the atria or the ventricles.

P wave Synchronous Pacing: This special mode is used for patient with intact SA node function who suffer from AV nodal disease. In this mode, a pacing impulse is triggered by the normal P wave, causing ventricular depolarization.

Pacing Failure:

Pacemakers usually either fail to sense or fail to pace appropriately in one of these patterns.

Oversensing: The pacemaker senses native activity when there is none and thus fails to pace when needed. (Oversensing = Too little pacing)
Undersensing: The pacemaker does not sense native activity when present, resulting in asynchronous pacing when it is not needed. (Undersensing = Too much pacing)

Pacing Output Failure: Pacemaker fails to generate an impulse when needed.

Failure to Capture: Pacemakers generates an impulse, but the heart fails to respond with a contraction.

Battery Failure: A weakening or otherwise failing battery can result in low voltage pacer spikes that do not capture, causing bradycardia. Conversely, it can also result in runaway pacing from a failure to sense and modulate the heart rate.
MAKING IT WORK

At first, trying to remember where all the electrodes go, much less all the different rules and criteria can be overwhelming. Just remember that even the physicians for whom all of these diseases and procedures are named for at one time, long ago, acquired and interpreted their first ECG, just like you. They didn’t know, they had to learn, they had to use cheat sheets to remember what they needed to know. But they learned the same way you will, by doing it every single day.

Another example I use frequently are firemen and sailors. In order to do their jobs, they have to tie knots of all different kinds for many purposes. They learn to do this by having rope in their hands EVERY SINGLE DAY. The way you’ll learn how to acquire and interpret ECGs is to do them and read them every day. Run them on every patient you can, share them with your coworkers, get them from the hospitals, make friends with cardiologists and cath lab techs.

The internet is full of free ECG resources, a list of which I’ve included below. These are the best for learning from different teachers and clinicians about different ways to look at each ECG. Also, search the web for ongoing research about new applications for ECG and the evidence based medicine which drives prehospital, emergency and acute care.

Finally, some of the best advice I was ever given is to “always show the ECG to someone smarter than you”. No matter how simple or normal or non-specific an ECG looks, there are almost always hidden clues that a more experienced eye may see that you haven’t learned yet. Seek out those people who can help you get the most of this incredibly useful, versatile and vital diagnostic test.

INTERNET RESOURCES
The ECG Wave Maven: http://ecg.bidmc.harvard.edu/maven/mavenmain.asp
Prehospital 12 Lead: http://ems12lead.com/
Dr. Smith’s 12 Lead blog: http://hqmeded-ecg.blogspot.com/
REFERENCES


ABOUT THE INSTRUCTOR

Michael C. Berrier is has been active in public safety since 1987, and has been working and teaching as a paramedic for the past 20 years. He has worked in rural/suburban 911, fire service, the offshore oilfield, EMS administration and most recently as a critical care transport paramedic in North Carolina.

Michael holds certifications from North Carolina, The National Registry of EMT’s, University of Maryland- Baltimore County, Cleveland Clinic, Board for Critical Care Transport Certification and the Society of Critical Care Medicine.