ACLS
HOME REVIEW PACKET

Prior to class, please complete ACLS self-assessment test by accessing the student website: www.Heart.org/eccstudent and enter the code “ACLS15”. Bring completed pre-course material to class.

www.CPRguys.biz
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## ALGORITHMS

- BRADYCARDIA 29-30
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Perform the following assessments and actions during the stabilization, categorization and triage of the patient:

Use the acronym **VOMIT**

- Assess Vital signs
- Administer Oxygen
- Attach a Monitor
- IV or IO access
- Twelve lead and treatment

*Remember stable vs. unstable (drugs vs. electricity)*

How do you determine is a patient is stable or unstable?

Use the acronym **CASH**

- Chest pain (must be accompanied by one of the following)
- Alter mental status
- Shortness of breath
- Hypotension

If the patient has “CASH” they pay the electric bill= They get electricity

**Stable:** The term stable patient refers to a patient that is experiencing an arrhythmia or irregular heart rhythm but has vital signs that are within normal limits. We treat stable patients with pharmacology vs. electrical therapy. If drug therapy is ineffective we then turn to electrical therapy to treat the patient.

**Unstable:** The term unstable in relation to patients refers to patients that also have arrhythmias, but have vital signs that are considered unstable. For example chest pain, altered mental status, shortness of breath, and hypotension. If this patient is left untreated without intervention it may ultimately result in cardiac arrest and death. Patients that fall into the unstable classification are treated with electrical therapy. For example a patient who has bradycardia and is unstable the treatment would be to transcutaneously pace the patient (as per 2010 guidelines atropine may be used instead of directly transcutaneous pacing). In tachycardia we would synchronize cardiovert the patient. With unstable patients our first intervention is electrical therapy, if this is not effective we then turn to pharmacology.

Every patient with chest pain gets **MONA**=

- Morphine (**BP<100 systolic**)
- Oxygen
- Nitro
- Aspirin
Stroke

- Rapid identification and assessment of patients
- Utilize a stroke specific neuro check
- Example Cincinnati Prehospital Stroke Screen or Los Angeles Stroke Screen
- Rapid transport to closest appropriate facility
- Emergent head CT

Termination of Resuscitation Considerations

- Return of spontaneous circulation
- Valid DNR order exists
- Rescuer in danger or scene is unsafe
- Evidence of irreversible death
- Pre-hospital: approval from medical control
- Prolonged resuscitative efforts

If patient get a Return of Spontaneous Circulation (ROSC)

- Maintain O2 sat > 94%
- Consider advanced airway with waveform capnography
- Do not hyperventilate
- Treat hypotension when SBP <90 mm Hg
- IV/IO Bolus up to 2L or Vasopressor infusion
- Consider treatable causes (5 H’s and T’s)
- Consider Hypothermia if patient not following commands and no contraindications exist. Transfer pt to hospital with hypothermia capabilities.

H’s and T’s of ACLS

Knowing the H’s and T’s of ACLS will help prepare you for any ACLS scenario. Don’t forget your H’s and T’s. The H’s and T’s of ACLS is a mnemonic used to help recall the major contributing factors to pulseless arrest including PEA, Asystole, Ventricular Fibrillation, and Ventricular Tachycardia. These H’s and T’s will most commonly be associated with PEA, but they will help direct your search for underlying causes to any of arrhythmias associated with ACLS. Each is discussed more thoroughly below.

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<th>The H’s include</th>
<th>The T’s include</th>
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The H’s include:

**Hypovolemia**
Hypovolemia or the loss of fluid volume in the circulatory system can be a major contributing cause to cardiac arrest. Looking for obvious blood loss in the patient with pulseless arrest is the first step in determining if the arrest is related to hypovolemia. After CPR, the most important intervention is obtaining intravenous access/IO access. A fluid challenge or fluid bolus may also help determine if the arrest is related to hypovolemia.

**Hypoxia**
Hypoxia or deprivation of adequate oxygen supply can be a significant contributing cause to cardiac arrest. You must ensure that the patient’s airway is open, and that the patient has chest rise and fall and bilateral breath sounds with ventilation. Also ensure that your oxygen source is connected properly.

**Hydrogen ion (acidosis)**
To determine if the patient is in respiratory acidosis, an arterial blood gas evaluation must be performed. Prevent respiratory acidosis by providing adequate ventilation. Prevent metabolic acidosis by giving the patient sodium bicarbonate.

**Hyper-/hypokalemia**
Both a high potassium level and a low potassium level can contribute to cardiac arrest. The major sign of hyperkalemia or high serum potassium is taller and peaked T-waves. Also, a widening of the QRS-wave may be seen. This can be treated in a number of ways which include sodium bicarbonate (IV), glucose insulin, calcium chloride (IV), Kayexalate, dialysis, and possibly albuterol. All of these will help reduce serum potassium levels. The major signs of hypokalemia or low serum potassium are flattened T-waves, prominent U-waves, and possibly a widened QRS complex. Treatment of hypokalemia involves rapid but controlled infusion of potassium. Giving IV potassium has risks. Always follow the appropriate infusion standards. Never give undiluted intravenous potassium.

**Hypoglycemia**
Hypoglycemia or low blood glucose can have many negative effects on the body, and it can be associated with cardiac arrest. Treat hypoglycemia with IV dextrose to reverse low blood glucose. Hypoglycemia was removed from the H’s but is still to be considered important during the assessment of any person in cardiac arrest.

**Hypothermia**
If a patient has been exposed to the cold, warming measures should be taken. The hypothermic patient may be unresponsive to drug therapy and electrical therapy (defibrillation or pacing). Core temperature should be raised above 86 F (30 C) as soon as possible.

The T’s include:

**Toxins**
Accidental overdose of a number of different kinds of medications can cause pulseless arrest. Some of the most common include: tricyclics, digoxin, beta blockers, and calcium channel blockers. Street drugs and other
chemicals can precipitate pulseless arrest. Cocaine is the most common street drug that increases incidence of pulseless arrest. ECG signs of toxicity include prolongation of the QT interval. Physical signs include bradycardia, pupil symptoms, and other neurological changes. Support of circulation while an antidote or reversing agent is obtained is of primary importance. Poison control can be utilized to obtain information about toxins and reversing agents.

Tamponade
Cardiac tamponade is an emergency condition in which fluid accumulates in the pericardium (sac in which the heart is enclosed). The buildup of fluid results in ineffective pumping of the blood which can lead to pulseless arrest. ECG symptoms include narrow QRS complex and rapid heart rate. Physical signs include jugular vein distention (JVD), no pulse or difficulty palpating a pulse, and muffled heart sounds due to fluid inside the pericardium. The recommended treatment for cardiac tamponade is pericardiocentesis.

Tension Pneumothorax
Tension pneumothorax occurs when air is allowed to enter the plural space and is prevented from escaping naturally. This leads to a buildup of tension that causes shifts in the intrathoracic structure that can rapidly lead to cardiovascular collapse and death. ECG signs include narrow QRS complexes and slow heart rate. Physical signs include JVD, tracheal deviation, unequal breath sounds, difficulty with ventilation, and no pulse felt with CPR. Treatment of tension pneumothorax is needle decompression.

Thrombosis (heart: acute, massive MI)
Coronary thrombosis is an occlusion or blockage of blood flow within a coronary artery caused by blood that has clotted within the vessel. The clotted blood causes an Acute Myocardial Infarction which destroys heart muscle and can lead to sudden death depending on the location of the blockage. ECG signs indicating coronary thrombosis are 12 lead ECG with ST-segment changes, T-wave inversions, and/or Q waves. Physical signs include: elevated cardiac markers on lab tests, and chest pain/pressure. Treatments for coronary thrombosis include use of fibrinolytic therapy, PCI (percutaneous coronary intervention). The most common PCI procedure is coronary angioplasty with or without stent placement.

Thrombosis (lungs: massive pulmonary embolism)
Pulmonary thrombus or pulmonary embolism (PE) is a blockage of the main artery of the lung which can rapidly lead to respiratory collapse and sudden death. ECG signs of PE include narrow QRS Complex and rapid heart rate. Physical signs include no pulse felt with CPR. Distended neck veins, positive d-dimer test, prior positive test for DVT or PE. Treatment includes surgical intervention (pulmonary thrombectomy) and fibrinolytic therapy.

Trauma
Trauma can be a cause of pulseless arrest, and a proper evaluation of the patients physical condition and history should reveal any traumatic injuries. Treat each traumatic injury as needed to correct any reversible cause or contributing factor to the pulseless arrest. Trauma was removed from the T’s but is still to be considered important during the assessment of any person in cardiac arrest.
Defibrillator

Most defibrillators used today are biphasic. Biphasic means that the electrical current travels from one paddle to the other paddle and then back in the other direction. The biphasic shock also requires less energy to restore normal heart rhythm and is believed reduce skin burns and cellular damage to the heart. When using a biphasic defibrillator in VF and/or pulseless VT, you will use a dose of 200 Joules to shock as opposed of using a much high dose of 360 Joules if you were using a monophasic defibrillator.

There are only 3 button on the defibrillator that pertains to the course:
- Defibrillator
- Pace
- SYNC

Manual external defibrillator

There are only 2 heart rhythms that you can defibrillate;
- Pulseless Ventricular Tachycardia
- Ventricular Fibrillation

Set the machine on defibrillator mode, set the Joules setting to 200 Joules (biphasic) (360 Joules for a monophasic), charge, and verify that there is no one near or touching the patient. (Note; verify there is no oxygen blowing over victims chest). Then push shock button and IMMEDIATELY resume CPR.

Transcutaneous pacing

Transcutaneous pacing (also called external pacing) is a temporary means of pacing a patient's heart during a medical emergency. It is accomplished by delivering pulses of electric current through the patient's chest, which stimulates the heart to contract. The most common indication for transcutaneous pacing is an abnormally slow heart rate. By convention, a heart rate of less than 60 beats per minute in the adult patient is called bradycardia. Not all instances of bradycardia require medical treatment. Normal heart rate varies substantially between individuals, and many athletes in particular have a relatively slow resting heart rate. In addition, the heart rate is known to naturally slow with age. It is only when bradycardia presents with signs and symptoms of shock that it requires emergency treatment with transcutaneous pacing. First attach pad, then a heart rate is selected, and current (measured in milliamps) is increased until electrical capture (characterized by a wide QRS complex with tall, broad T wave on the ECG) is obtained( pacer spike), with a corresponding pulse. Pacing artifact on the ECG and severe muscle twitching may make this determination difficult. It is therefore advisable to use another instrument (e.g. SpO2 monitor or bedside doppler) to confirm mechanical capture.

Synchronized electrical cardioversion

Synchronized electrical cardioversion is used to treat hemodynamically significant supraventricular (or narrow complex) tachycardias, including atrial fibrillation and atrial flutter. It is also used in the emergent treatment of wide complex tachycardias, including ventricular tachycardia, when a pulse is present. To perform synchronized electrical cardioversion two electrode pads are placed on the chest of the patient. A synchronizing function allows the cardioverter to deliver a reversion shock, by way of the pads, of a selected amount of electric current over a predefined number of milliseconds at the optimal moment in the cardiac cycle which corresponds to the R wave of the QRS complex on the ECG. Timing the shock to the R wave prevents the delivery of the shock during the vulnerable period (or relative refractory period) of the cardiac cycle, which could induce ventricular fibrillation.
ACLS Algorithms and Their Primary Drugs

Vent. Fib./Tach.
Epinephrine
Vasopressin (removed from algorithm per 2015 ACLS Guidelines)
Amiodarone
Lidocaine
Magnesium

Asystole/PEA
Epinephrine
Vasopressin
Atropine (removed from algorithm per 2010 ACLS Guidelines)

Bradycardia
Atropine
Epinephrine
Dopamine

Tachycardia
Adenosine
Diltiazem
Beta-blockers
Amiodarone
Digoxin
Verapamil
Magnesium

Acute Coronary Syndromes
Oxygen
Aspirin
Nitroglycerin
Morphine
Fibrinolytic therapy
Heparin
Beta-Blockers

Acute Stroke
tPA-tissue plasminogen activator
Glucose (D50)
Labetalol
Nitroprusside
Nicardipine
Aspirin
PHARMACOLOGY

Vasopressors vs. Anti-dysrhythmic

Vasopressors:
A vasopressor is a medication that produces vasoconstriction and a rise in blood pressure. The vasopressors that can be used in the treatment of VF/Pulseless VT are epinephrine and/or vasopressin. Epinephrine is primarily used for its vasoconstrictive effects. Vasoconstriction is important during CPR because it will help increase blood flow to the brain and heart. Vasopressin is also used for its vasoconstrictive effects and has been shown to have effects similar to those of epinephrine.

Anti-dysrhythmic:
Antiarrhythmic medications are a group of pharmaceuticals that are used to suppress abnormal rhythms of the heart (cardiac arrhythmias), such as atrial fibrillation, atrial flutter, ventricular tachycardia, and ventricular fibrillation. Drugs such as Amiodarone, Lidocaine, Procainamide and Magnesium are antiarrhythmic medications that are used in the pulseless arrest algorithm.

ACLS DRUGS:

Epinephrine
(VASOPRESSOR)-- Indicated in cardiac arrest. Increases heart rate, increase contractility and peripheral vascular resistance. Standard Arrest Dose: 1 mg IV q3-5 min (1:10,000). E-T Tube: 2.0-3.0 mg diluted in 10cc NS. Also for profound bradycardia and/or hypotension as a Drip: 2-10 mcg/minutes.

Amiodarone
(ANTIARRHYTHMIC)-- Used for both atrial and ventricular arrhythmias. Indicated for shock-refractory VT/VF Arrest Dose: 300 mg IVP, consider repeating with 150 mg IVP in 3-5min. Tachycardia Dose: 150 mg over 10 min. Max 2.2 mg/24 hr. Side effects are Bradycardia Hypotension. **Useful in controlling rate of atrial fibrillation and atrial flutter with WPW.

Procainamide
(ANTIARRHYTHMIC)-- Used in a wide variety of arrhythmias. Non-arrest dose: 20-50mg/min. Maximum dose of 17 mg/kg (1.2 gm for 70 kg patient Note: may cause torsade’s de pointes with QRS widening and prolong QT intervals. Drip: 1 Gm./250cc are at 1-4 mg/min. **Useful in controlling rate of atrial fibrillation (with rapid pulse) in WPW.

Lidocaine
(ANTIARRHYTHMIC)-- Used for ventricular arrhythmias. An alternative to Amiodarone for VT/Vfib Dose: 1.0 – 1.5 mg/kg IV total-3.0 mg/kg. Drip: 1Gm/250cc at 1-4mg/min.

Magnesium Sulfate
(ANTIARRHYTHMIC) Indicated in cardiac arrest-- Torsade’s de Pointes. Indicated for torsade’s de pointes with a pulse and life threatening arrhythmias due to digitalis toxicity. Dose: 1-2 Gm. (2-4 ml of a 50% solution) diluted. IVP in full arrest. 1-2g in 50 to 100 ml of DSW over 5-60 minutes in non-arrest.
**Adenosine**
Short half-life (<10 seconds). Slows A-V node conduction. First drug for most forms of narrow complex PSVT. Does not convert atrial fibrillation and/or flutter. Dose: 6mg rapid IV push, followed by 20ml NS push, may repeat twice after 1-2 min at 12mg IVP.

**Atropine**
Decreases vagal reflex, accelerates the rate of sinus node, may be useful in AV Block at the nodal level. Bradycardia dose: 0.5mg IVP q3-5 minutes – total 0.04mg/kg. (Max dose 3 mg)

**Dopamine**
Useful in the treatment of cardiogenic shock, hypotension, symptomatic bradyarrhythmias. 10mcg/kg/min = (cardiac dose) CAUTION: May increase myocardial oxygen requirements or worsen ischemia. Drip: 400mg in 250cc = 1600mcg/cc

**Sodium Bicarb**
Not recommended for routine use in cardiac arrest patients. Adequate ventilation and CPR, not bicarbonate, are the major “buffer agents” in cardiac arrest. May be used with responsive acidosis, prolonged arrest, and certain drug overdoses. Dose: 1mEg/kg IV. Given when the patient has been pulse less for a duration of time exceeding 10 mins.

**Diltiazem**
Used to control ventricular rate in A-fib/ A-flutter or after adenosine or in SVT. Dosage: 0.25 mg/kg (over 2mins, e.g. 20mg) second dose: .35mg/kg (over 2mins, e.g. 25mg)
Cardiac Anatomy

The heart could be described as being 2 pumps. One pump (right side) sends blood to your lungs to be oxygenated and to remove waste products (CO2) and the other pump (left side) sends the blood around the systemic circulation to oxygenate all the cells in the body. The heart weighs between 7 and 15 ounces (200 to 425 grams) and is a little larger than the size of your fist. It is located between your lungs in the middle of your chest, behind and slightly to the left of your breastbone (sternum).

The heart has 4 chambers. Two upper chambers are called the left and right atria, and the two lower chambers are called the left and right ventricles. The septum (a wall of muscle) separates the left and right atria and the left and right ventricles. The left ventricle is known as the largest and strongest chamber in your heart with enough force to push blood through the aortic valve and into your body.

The heart chambers have valves which assist in the transport of blood flow through the heart. These are:

- The tricuspid valve regulates blood flow between the right atrium and right ventricle.
- The pulmonary valve controls blood flow from the right ventricle into the pulmonary arteries, which carry deoxygenated blood to your lungs to oxygenated.
- The mitral, or bicuspid valve lets oxygenated blood from your lungs pass from the left atrium into the left ventricle.
- The aortic valve opens the way for oxygenated blood to pass from the left ventricle into the aorta, your body's largest artery, from here the blood is distributed to whole of your body.
The electrical system in your heart controls the speed of your heartbeat. Your heart has three main components to the system, these consist of:

- S-A node (sinoatrial node)
- A-V node (atrioventricular node)
- Purkinje system

The S-A node, also called the "natural pacemaker", of your heart because it controls your heart rate. The S-A node is made of specialized cells located in the right atrium of the heart. The S-A node creates the electricity that makes your heart beat. The S-A node normally produces 60-100 electrical signals per minute — this is your heart rate.

The A-V node is a bundle of cells between the atria and ventricles. The electrical signals generated by the S-A node are "caught" and held for milliseconds before being sent onto the bundle of HIS (HIS Purkinje system).

HIS purkinje system is in your heart's ventricles. Electricity travels through the His-purkinje system to make your ventricles contract. The electricity from the A-V node hits the bundle of HIS before being directed into the right and left bundle branches and finally into the purkinje fibres that are located in the cardiac muscle. This stimulates the ventricles to contract.

**The Electrical Pathway**

- **STEP 1.** The S-A node creates an electrical signal
- **STEP 2.** The electrical signal follows natural electrical pathways through both atria. The movement of electricity stimulates the atria to contract, which pushes blood into the ventricles.
- **STEP 3.** The electrical signal reaches the A-V node. There, the signal pauses to give the ventricles time to fill with blood
- **STEP 4.** The electrical signal spreads through the His-purkinje system. The movement of electricity causes the ventricles to contract and push blood out to your lungs and body.

The name for the steps above is known as the cardiac cycle which lasts for 0.8 seconds.
1. Atrial systole = 0.1 second
2. Ventricular systole = 0.3 seconds
3. Diastole = 0.4 seconds

- Systole refers to the contraction of the cardiac muscle
- Diastole refers to the relaxation of the cardiac muscle

**Nervous Control of the Heart**

Although the S-A node sets the basic rhythm of the heart, the rate and strength of its beating can be modified by two auxiliary control centres located in the medulla oblongata of the brain.

- One sends nerve impulses down **accelerator nerves**.
- The other sends nerve impulses down a pair of **vagus nerves**

**Accelerator Nerves**

The accelerator nerves are part of the sympathetic branch of the autonomic nervous system. They increase the rate and strength of the heartbeat and thus increase the flow of blood. Their activation usually arises from some stress such as fear or exertion. The heartbeat may increase to 180 beats per minute. The strength of contraction increases as well so the amount of blood pumped may increase to as much as 25-30 litres/minute.

**Vagus Nerve**

The vagus nerves are part of the parasympathetic branch of the autonomic nervous system. They, too, run from the medulla oblongata to the heart. Their activity slows the heartbeat.

Pressure receptors in the aorta and carotid arteries send impulses to the medulla which relays these impulses back by way of the vagus nerves to the heart. Heartbeat and blood pressure diminish.
The electrocardiogram (ECG) is a diagnostic tool that measures and records the electrical activity of the heart in detail. Being able to interpretate these details allows diagnosis of a wide range of heart problems.

**ECG Electrodes**

*Skin Preparation:*

Clean with an alcohol wipe if necessary. If the patients are very hairy – shave the electrode areas.

**ECG standard leads**

There are three of these leads, I, II and III.

Lead I: is between the right arm and left arm electrodes, the left arm being positive.  
Lead II: is between the right arm and left leg electrodes, the left leg being positive.  
Lead III: is between the left arm and left leg electrodes, the left leg again being positive.

**Chest Electrode Placement**

- V1: Fourth intercostal space to the right of the sternum.  
- V2: Fourth intercostal space to the Left of the sternum.  
- V3: Directly between leads V2 and V4.  
- V4: Fifth intercostal space at midclavicular line.  
- V5: Level with V4 at left anterior axillary line.  
- V6: Level with V5 at left midaxillary line. (Directly under the midpoint of the armpit)

**ECG Leads - Views of the Heart**

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<th>Chest Leads</th>
<th>View</th>
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<td>V1 &amp; V2</td>
<td>Right Ventricle</td>
</tr>
<tr>
<td>V3 &amp; V4</td>
<td>Septum/Lateral Left Ventricle</td>
</tr>
<tr>
<td>V5 &amp; V6</td>
<td>Anterior/Lateral Left Ventricle</td>
</tr>
</tbody>
</table>

The ECG records the electrical activity that results when the heart muscle cells in the atria and ventricles contract.

- Atrial contractions show up as the P wave.  
- Ventricular contractions show as a series known as the QRS complex.  
- The third and last common wave in an ECG is the T wave. This is the electrical activity produced when the ventricles are recharging for the next contraction (repolarizing).  
- Interestingly, the letters P, Q, R, S, and T are not abbreviations for any actual words but were chosen many years ago for their position in the middle of the alphabet.  
- The electrical activity results in P, QRS, and T waves that are of different sizes and shapes. When viewed from different leads, these waves can show a wide range of abnormalities of both the electrical conduction system and the muscle tissue of the hearts 4 pumping chambers.
**ECG Interpretation**

The graph paper that the ECG records on is standardized to run at 25mm/second, and is marked at 1 second intervals on the top and bottom. The horizontal axis correlates the length of each electrical event with its duration in time. Each small block (defined by lighter lines) on the horizontal axis represents 0.04 seconds. Five small blocks (shown by heavy lines) is a large block, and represents 0.20 seconds.

Duration of a waveform, segment, or interval is determined by counting the blocks from the beginning to the end of the wave, segment, or interval.

P-Wave: represents atrial depolarization - the time necessary for an electrical impulse from the sinoatrial (SA) node to spread throughout the atrial musculature.

- Location: Precedes QRS complex
- Amplitude: Should not exceed 2 to 2.5 mm in height
- Duration: 0.06 to 0.11 seconds

P-R Interval: represents the time it takes an impulse to travel from the atria through the AV node, bundle of His, and bundle branches to the Purkinje fibres.
- Location: Extends from the beginning of the P wave to the beginning of the QRS complex
  Duration: 0.12 to 0.20 seconds.

![QRS wave](image)

QRS Complex: represents ventricular depolarization. The QRS complex consists of 3 waves: the Q wave, the R wave, and the S wave.

- The Q wave is always located at the beginning of the QRS complex. It may or may not always be present.
- The R wave is always the first positive deflection.
- The S wave, the negative deflection, follows the R wave.
- Location: Follows the P-R interval
  Amplitude: Normal values vary with age and sex
  Duration: No longer than 0.10 seconds

![Q-T interval](image)

Q-T Interval: represents the time necessary for ventricular depolarization and repolarization.

- Location: Extends from the beginning of the QRS complex to the end of the T wave (includes the QRS complex, S-T segment, and the T wave)
Duration: Varies according to age, sex, and heart rate

T Wave: represents the repolarization of the ventricles. On rare occasions, a U wave can be seen following the T wave. The U wave reflects the repolarization of the His-Purkinje fibres.

- Location: Follows the S wave and the S-T segment
- Amplitude: 5mm or less in standard leads I, II, and III; 10mm or less in precordial leads V1-V6.

Duration: Not usually measured

S-T Segment: represents the end of the ventricular depolarization and the beginning of ventricular repolarization.

- Location: Extends from the end of the S wave to the beginning of the T wave
- Duration: Not usually measured

The ECG and Myocardial Infarction

During an MI, the ECG goes through a series of abnormalities. The initial abnormality is called a hyperacute T wave. This is a T wave that is taller and more pointed than the normal T wave.
The abnormality lasts for a very short time, and then elevation of the ST segment occurs. This is the hallmark abnormality of an acute MI. It occurs when the heart muscle is being injured by a lack of blood flow and oxygen and is also called a *current of injury*.

An ECG can not only tell you if an MI is present but can also show the approximate location of the heart attack, and often which artery is involved. When the ECG abnormalities mentioned above occur, then the MI can be localized to a certain region of the heart. For example, see the table below:

<table>
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<tr>
<th>ECG leads</th>
<th>Location of MI</th>
<th>Coronary Artery</th>
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<td>Inferior MI</td>
<td>Right Coronary Artery</td>
</tr>
<tr>
<td>V1-V4</td>
<td>Anterior or Anteroseptal MI</td>
<td>Left Anterior Descending Artery</td>
</tr>
<tr>
<td>V5-V6, I,aVL</td>
<td>Lateral MI</td>
<td>Left Circumflex Artery</td>
</tr>
<tr>
<td>ST depression in V1, V2</td>
<td>Posterior MI</td>
<td>Left Circumflex Artery or Right Coronary Artery</td>
</tr>
</tbody>
</table>
Rhythms

Normal Sinus Rhythm

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate - (60-100 bpm)
- QRS Duration - Normal
- P Wave - Visible before each QRS complex
- P-R Interval - Normal (<5 small Squares. Anything above and this would be 1st degree block)
- Indicates that the electrical signal is generated by the sinus node and travelling in a normal fashion in the heart.

Sinus Bradycardia

A heart rate less than 60 beats per minute (BPM). This in a healthy athletic person may be 'normal', but other causes may be due to increased vagal tone from drug abuse, hypoglycaemia and brain injury with increase intracranial pressure (ICP) as examples

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate - less than 60 beats per minute
- QRS Duration - Normal
- P Wave - Visible before each QRS complex
- P-R Interval - Normal
- Usually benign and often caused by patients on beta blockers
Sinus Tachycardia

An excessive heart rate above 100 beats per minute (BPM) which originates from the SA node. Causes include stress, fright, illness and exercise. Not usually a surprise if it is triggered in response to regulatory changes e.g. shock. But if there is no apparent trigger then medications may be required to suppress the rhythm.

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate - More than 100 beats per minute
- QRS Duration - Normal
- P Wave - Visible before each QRS complex
- P-R Interval - Normal
- The impulse generating the heartbeats are normal, but they are occurring at a faster pace than normal. Seen during exercise

Supraventricular Tachycardia (SVT) Abnormal

A narrow complex tachycardia or atrial tachycardia which originates in the 'atria' but is not under direct control from the SA node. SVT can occur in all age groups.

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate - 140-220 beats per minute
- QRS Duration - Usually normal
- P Wave - Often buried in preceding T wave
- P-R Interval - Depends on site of supraventricular pacemaker
- Impulses stimulating the heart are not being generated by the sinus node, but instead are coming from a collection of tissue around and involving the atrioventricular (AV) node
Atrial Fibrillation

Many sites within the atria are generating their own electrical impulses, leading to irregular conduction of impulses to the ventricles that generate the heartbeat. This irregular rhythm can be felt when palpating a pulse

Looking at the ECG you'll see that:

- Rhythm - Irregularly irregular
- Rate - usually 100-160 beats per minute but slower if on medication
- QRS Duration - Usually normal
- P Wave - Not distinguishable as the atria are firing off all over
- P-R Interval - Not measurable
- The atria fire electrical impulses in an irregular fashion causing irregular heart rhythm

Atrial Flutter

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate - Around 110 beats per minute
- QRS Duration - Usually normal
- P Wave - Replaced with multiple F (flutter) waves, usually at a ratio of 2:1 (2F - 1QRS) but sometimes 3:1
- P Wave rate - 300 beats per minute
- P-R Interval - Not measurable
- As with SVT the abnormal tissue generating the rapid heart rate is also in the atria, however, the atrioventricular node is not involved in this case.
1st Degree AV Block

1st Degree AV block is caused by a conduction delay through the AV node but all electrical signals reach the ventricles. This rarely causes any problems by itself and often trained athletes can be seen to have it. The normal P-R interval is between 0.12s to 0.20s in length, or 3-5 small squares on the ECG.

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate - Normal
- QRS Duration - Normal
- P Wave - Ratio 1:1
- P Wave rate - Normal
- P-R Interval - Prolonged (>5 small squares)

2nd Degree Block Type 1 (Wenckebach)

Another condition whereby a conduction block of some, but not all atrial beats getting through to the ventricles. There is progressive lengthening of the PR interval and then failure of conduction of an atrial beat, this is seen by a dropped QRS complex.

Looking at the ECG you'll see that:

- Rhythm - Regularly irregular
- Rate - Normal or Slow
- QRS Duration - Normal
- P Wave - Ratio 1:1 for 2,3 or 4 cycles then 1:0.
- P Wave rate - Normal but faster than QRS rate
- P-R Interval - Progressive lengthening of P-R interval until a QRS complex is dropped
2nd Degree Block Type 2

When electrical excitation sometimes fails to pass through the A-V node or bundle of His, this intermittent occurrence is said to be called second degree heart block. Electrical conduction usually has a constant P-R interval, in the case of type 2 block atrial contractions are not regularly followed by ventricular contraction.

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate - Normal or Slow
- QRS Duration - Prolonged
- P Wave - Ratio 2:1, 3:1
- P Wave rate - Normal but faster than QRS rate
- P-R Interval - Normal or prolonged but constant

3rd Degree Block

3rd degree block or complete heart block occurs when atrial contractions are 'normal' but no electrical conduction is conveyed to the ventricles. The ventricles then generate their own signal through an 'escape mechanism' from a focus somewhere within the ventricle. The ventricular escape beats are usually 'slow'.

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate - Slow
- QRS Duration - Prolonged
- P Wave - Unrelated
- P Wave rate - Normal but faster than QRS rate
- P-R Interval - Variation
- Complete AV block. No atrial impulses pass through the atrioventricular node and the ventricles generate their own rhythm.
Bundle Branch Block

Abnormal conduction through the bundle branches will cause a depolarization delay through the ventricular muscle, this delay shows as a widening of the QRS complex. Right Bundle Branch Block (RBBB) indicates problems in the right side of the heart. Whereas Left Bundle Branch Block (LBBB) is an indication of heart disease. If LBBB is present then further interpretation of the ECG cannot be carried out.

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate - Normal
- QRS Duration - Prolonged
- P Wave - Ratio 1:1
- P Wave rate - Normal and same as QRS rate
- P-R Interval - Normal

Premature Ventricular Complexes

Due to a part of the heart depolarizing earlier than it should

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate - Normal
- QRS Duration - Normal
- P Wave - Ratio 1:1
- P Wave rate - Normal and same as QRS rate
- P-R Interval - Normal
- Also you'll see 2 odd waveforms, these are the ventricles depolarising prematurely in response to a signal within the ventricles.(Above - unifocal PVC's as they look alike if they differed in appearance they would be called multifocal PVC's, as below)
Junctional Rhythms

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate - 40-60 Beats per minute
- QRS Duration - Normal
- P Wave - Ratio 1:1 if visible. Inverted in lead II
- P Wave rate - Same as QRS rate
- P-R Interval - Variable

Ventricular Tachycardia (VT) Abnormal

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate - 180-190 Beats per minute
- QRS Duration - Prolonged
- P Wave - Not seen
- Results from abnormal tissues in the ventricles generating a rapid and irregular heart rhythm. Poor cardiac output is usually associated with this rhythm thus causing the pt to go into cardiac arrest. Shock this rhythm if the patient is unconscious and without a pulse
Ventricular Fibrillation (VF) Abnormal

Disorganised electrical signals cause the ventricles to quiver instead of contract in a rhythmic fashion. A patient will be unconscious as blood is not pumped to the brain. Immediate treatment by defibrillation is indicated. This condition may occur during or after a myocardial infarct.

Looking at the ECG you'll see that:

- Rhythm - Irregular
- Rate - 300+, disorganised
- QRS Duration - Not recognisable
- P Wave - Not seen
- **This patient needs to be defibrillated!! QUICKLY**
- 
- **Asystole - Abnormal**

Looking at the ECG you'll see that:

- Rhythm - Flat
- Rate - 0 Beats per minute
- QRS Duration - None
- P Wave - None
- **Carry out CPR!!**
Myocardial Infarct (MI)

Looking at the ECG you'll see that:

- Rhythm - Regular
- Rate - 80 Beats per minute
- QRS Duration - Normal
- P Wave - Normal
- S-T Element does not go isoelectric which indicates infarction

<table>
<thead>
<tr>
<th>ECG Component</th>
<th>Time(sec)</th>
<th>Small Squares</th>
</tr>
</thead>
<tbody>
<tr>
<td>P Wave</td>
<td>0.10</td>
<td>up to 2.5</td>
</tr>
<tr>
<td>PR Interval</td>
<td>0.12 - 0.20</td>
<td>2.5-5.0</td>
</tr>
<tr>
<td>QRS</td>
<td>0.10</td>
<td>1.5-2.5</td>
</tr>
</tbody>
</table>
Marriages and Heart Blocks

I figured I would share with you all something I made up while I was in paramedic school learning ECGs. It’s an easy way to memorize the different heart blocks and how to recognize them. It all revolves around a story about a couple named “P” and “QRS” (clever huh?). Different stages of their relationship will represent the blocks.

Normal Sinus
P and QRS are in the honeymoon phase of their marriage. They are always together and never leave each others sight. Life is good for P and QRS!

First Degree AV Block
P and QRS are getting irritated with each other. While they still remain true to the relationship, they are keeping their distance for now.

Second Degree AV Block (Type I)
P and QRS have entered the viscous cycle of breaking up and getting back together. They get together, they start fighting and keeping their distance, and they break up again.

Second Degree AV Block (Type II)
P and QRS have decided to get back together again. However, P is still quite disgruntled and has decided to go out every night or two and fool around on QRS. You can probably imagine what comes next!

Third Degree AV Block
As you probably saw coming, QRS caught P cheating and moved out. They have filed for divorce and are completely separated. There is absolutely no relationship between P and QRS now.

The End
Cardiac Arrest Algorithm

ASYSTOLE/PEA (PULSELESS ELECTRICAL ACTIVITY)

VENTRICULAR FIBRILLATION (VF)

VENTRICULAR TACHYCARDIA (VT)

TORSADES DE POINTES
Adult Cardiac Arrest

Shout for Help/Activate Emergency Response

1. Start CPR
   - Give oxygen
   - Attach monitor/defibrillator

2. VF/VT
   - Yes: Rhythm shockable?
   - No: Asystole/PEA

3. Shock

4. CPR 2 min
   - IV/IO access

5. CPR 2 min
   - Rhythm shockable?
   - Yes: Shock
   - No: CPR 2 min

6. CPR 2 min
   - Epinephrine every 3-5 min
   - Consider advanced airway, capnography

7. CPR 2 min
   - Rhythm shockable?
   - Yes: Shock
   - No: CPR 2 min

8. CPR 2 min
   - Amiodarone
   - Treat reversible causes

9. CPR 2 min
   - IV/IO access
   - Epinephrine every 3-5 min
   - Consider advanced airway, capnography

10. CPR 2 min
    - Rhythm shockable?
    - Yes: Shock
    - No: CPR 2 min

11. CPR 2 min
    - Treat reversible causes

12. CPR 2 min
    - If no signs of return of spontaneous circulation (ROSC), go to 10 or 11
    - If ROSC, go to Post-Cardiac Arrest Care

CPR Quality
- Push hard (≥2 inches [5 cm]) and fast (≥100/min) and allow complete chest recoil
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Rotate compressor every 2 minutes
- If no advanced airway, 30:2 compression-ventilation ratio
- Quantitative waveform capnography
  - If Petco2 < 10 mm Hg, attempt to improve CPR quality
- Intra-arterial pressure
  - If relaxation phase (diastolic) pressure < 20 mm Hg, attempt to improve CPR quality

Return of Spontaneous Circulation (ROSC)
- Pulse and blood pressure
- Abrupt sustained increase in Petco2 (typically ≥ 40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Shock Energy
- Biphasic: Manufacturer recommendation
  (eg, initial dose of 120-200 J; if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered)
- Monophasic: 360 J

Drug Therapy
- Epinephrine IV/IO Dose: 1 mg every 3-5 minutes
- Vasopressin IV/IO Dose: 40 units can replace first or second dose of epinephrine
- Amiodarone IV/IO Dose:
  - First dose: 300 mg bolus
  - Second dose: 150 mg

Advanced Airway
- Supraglottic advanced airway or endotracheal intubation
- Waveform capnography to confirm and monitor ET tube placement
- 8-10 breaths per minute with continuous chest compressions

Reversible Causes
- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypo-/hyperkalemia
- Hypothermia
- Tension pneumothorax
- Tamponade, cardiac
- Toxins
- Thrombosis, pulmonary
- Thrombosis, coronary

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Bradycardia Algorithm

SINUS BRADYCARDIA

1ST DEGREE HEART BLOCK

2ND DEGREE TYPE 1 HEART BLOCK

2ND DEGREE TYPE 2 HEART BLOCK

3RD DEGREE HEART BLOCK
BRADYCARDIA
Heart rate <60 bpm and inadequate for clinical condition

1. Maintain patent airway; assist breathing as needed
2. Give oxygen
3. Monitor ECG (identify rhythm), blood pressure, oximetry
4. Establish IV access

Signs or symptoms of poor perfusion caused by the bradycardia?
(eg, acute altered mental status, ongoing chest pain, hypotension or other signs of shock)

4A. Observe/Monitor

Perfusion Adequate

- Prepare for transcutaneous pacing; use without delay for high-degree block (type II second-degree block or third-degree AV block)
- Consider atropine 0.5 mg IV while awaiting pacer. May repeat to a total dose of 3 mg. If ineffective, begin pacing
- Consider epinephrine (2 to 10 μg/min) or dopamine (2 to 10 μg/kg per minute) infusion while awaiting pacer or if pacing ineffective

Perfusion Poor

5. Prepare for transvenous pacing
6. Treat contributing causes
7. Consider expert consultation

Reminders
- If pulseless arrest develops, go to Pulseless Arrest Algorithm
- Search for and treat possible contributing factors:
  - Hypovolemia
  - Hypoxia
  - Hydrogen ion (acidosis)
  - Hypo-/hyperkalemia
  - Hypoglycemia
  - Hypothermia
  - Toxins
  - Tamponade, cardiac
  - Tension pneumothorax
  - Thrombosis (coronary or pulmonary)
  - Trauma (hypovolemia, increased ICP)
Tachycardia Algorithm

SINUS TACHYCARDIA

SUPRAVENTRICULAR TACHYCARDIA (SVT)

ATRIAL FIBRILLATION

ATRIAL TACHYCARDIA
Adult Tachycardia
(With Pulse)

1. Assess appropriateness for clinical condition.
   Heart rate typically ≥150/min if tachyarrhythmia.

2. Identify and treat underlying cause
   - Maintain patent airway; assist breathing as necessary
   - Oxygen (if hypoxemic)
   - Cardiac monitor to identify rhythm; monitor blood pressure and oximetry

3. Persistent tachyarrhythmia causing:
   - Hypotension?
   - Acutely altered mental status?
   - Signs of shock?
   - Ischemic chest discomfort?
   - Acute heart failure?

4. Synchronized cardioversion
   - Consider sedation
   - If regular narrow complex, consider adenosine

5. Wide QRS? ≥0.12 second
   - Yes
   - IV access and 12-lead ECG if available
   - Consider adenosine only if regular and monomorphic
   - Consider antiarrhythmic infusion
   - Consider expert consultation
   - No

6. Antiarrhythmic Infusions for Stable Wide-QRS Tachycardia
   - Procanamide IV Dose:
     - 20-50 mg/min until arrhythmia suppressed, hypotension ensues, QRS duration increases >50%, or maximum dose 17 mg/kg given.
     - Maintenance infusion: 1-4 mg/min. Avoid if prolonged QT or CHF.
   - Amiodarone IV Dose:
     - First dose: 150 mg over 10 minutes. Repeat as needed if VT recurs.
     - Follow by maintenance infusion of 1 mg/min for first 6 hours.
   - Sotalol IV Dose:
     - 100 mg (1.5 mg/kg) over 5 minutes. Avoid if prolonged QT.

Doses/Details

Synchronized Cardioversion
- Initial recommended doses:
  - Narrow regular: 50-100 J
  - Narrow irregular: 120-200 J biphasic or 200 J monophasic
  - Wide regular: 100 J
  - Wide irregular: defibrillation dose (NOT synchronized)

Adenosine IV Dose:
- First dose: 6 mg rapid IV push; follow with NS flush.
- Second dose: 12 mg if required.
ROSC

(Return of Spontaneous Circulation)
Adult Immediate Post-Cardiac Arrest Care

1. Return of Spontaneous Circulation (ROSC)

2. Optimize ventilation and oxygenation
   - Maintain oxygen saturation ≥94%
   - Consider advanced airway and waveform capnography
   - Do not hyperventilate

3. Treat hypotension (SBP <90 mm Hg)
   - IV/IO bolus
   - Vasopressor infusion
   - Consider treatable causes
   - 12-Lead ECG

5. Consider induced hypothermia

4. Follow commands?
   - Yes
   - STEMI OR high suspicion of AMI
   - No
   - Advanced critical care

Doses/Details

Ventilation/Oxygenation
Avoid excessive ventilation. Start at 10-12 breaths/min and titrate to target $\text{PETCO}_2$ of 35-40 mm Hg. When feasible, titrate $\text{FiO}_2$ to minimum necessary to achieve $\text{SpO}_2$ ≥94%.

IV Bolus
1-2 L normal saline or lactated Ringer’s. If inducing hypothermia, may use 4°C fluid.

Epinephrine IV Infusion:
0.1-0.5 mcg/kg per minute (in 70-kg adult: 7-35 mcg per minute)

Dopamine IV Infusion:
5-10 mcg/kg per minute

Norepinephrine IV Infusion:
0.1-0.5 mcg/kg per minute (in 70-kg adult: 7-35 mcg per minute)

Reversible Causes
- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypo-/hyperkalemia
- Hypothermia
- Tension pneumothorax
- Tamponade, cardiac
- Toxins
- Thrombosis, pulmonary
- Thrombosis, coronary

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