## ECG

## **Electrocardiogram (ECG)**

When certain areas of the heart are polarized (eg. the auricle), certain other areas are not polarized (eg. the ventricle), creating a difference in electrical potential in different regions of the heart. As the cardiac impulse passes through the heart, electrical current also spreads from the heart into the adjacent tissues surrounding the heart.

If electrodes are placed on the skin on opposite sides of the heart, electrical potentials generated by the current can be recorded by a galvanometric setting attached to a recording device.



The record is called an electrocardiogram (ECG); the machine used to record the current is called an electrocardiograph.

The current is measured at certain points on the body surface by bipolar limb leads at the apices of **Einthoven's triangle** drawn around the area of the heart.

The two arms and the left leg form apices of a triangle surrounding the heart.

At least 15 cardiac cycles are taken from each of the three leads by a single selector switch.

6 standard chest leads may also be applied at specific points and recorded one at a time in addition to the limb leads.

**Einthoven's Law:** Einthoven's law states that if the electrical potentials of any two of the three bipolar limb electrocardiographic leads are known at any given instant, the third one can be determined mathematically by the vector sum of the first two.

From the ECG tracing, the following information can be determined:

the heart rate and the heart rhythm

whether there are "conduction abnormalities" (abnormalities in how the electrical impulse spreads across the heart)

whether there has been a prior heart attack

whether there may be coronary artery disease

whether the heart muscle has become abnormally thickened

### The Normal Electrocardiogram



Normal electrocardiogram.

Component/	Approx. Time	Causes or effects			
interval	(secs)				
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P wave	0.08 – 0.10	Depolarization (and contraction) of atrium			
QRS complex	0.08 - 0.10	Depolarization (and contraction) of ventricles			
T wave	0.12 – 0.16	Repolarization of ventricle			
PR interval	0.10 – 0.20	Wave travels through AV node, AV bundle , bundle branches and Purkinje fibres			
ST interval	0.08 - 0.12	Time for complete excitation of ventricle			
QT interval	0.30 – 0.35	Time required for complete excitation and recovery of ventricles			
TP interlude	0.25 – 0.35	Time from completion of ventricular repolarization to next atrial excitation			

The P wave is caused by electrical potentials generated when the atria depolarize before atrial contraction begins.

The QRS complex is caused by potentials generated when the ventricles depolarize ie. as the depolarization wave spreads through the ventricles.

Therefore, both the P wave and components of QRS complex are depolarization waves.

The T wave is caused by potentials generated as the ventricles recover from the state of depolarization. This process normally occurs in ventricular muscle 0.25 to 0.35 second after depolarization, and the T wave is known as a repolarization wave.

# Cardiac Arrhythmias and Their Electrocardiographic Interpretation

#### Tachycardia

The term "tachycardia" means fast heart rate, usually defined in an adult person as faster than 100 beats per minute.

The electrocardiogram is normal except that the heart rate is about 150 per minute instead of the normal 72 per minute.

The general causes of tachycardia include increased body temperature, stimulation of the heart by the sympathetic nerves, or toxic conditions of the heart.



#### **Bradycardia**

The term "bradycardia" means a slow heart rate, usually defined as fewer than 60 beats per minute.



#### **Sinoatrial Block**

In rare instances, the impulse from the sinus node is blocked before it enters the atrial muscle.

This phenomenon shows sudden cessation of P waves, with resultant standstill of the atria. However, the ventricles pick up a new rhythm, the impulse usually originating spontaneously in the atrioventricular (A-V) node, so that the rate of the ventricular QRS-T complex is slowed but not otherwise altered.



#### **Atrioventricular Block**

Conditions that can either decrease or block the rate of impulse conduction in the A-V bundle are:

1) Ischemia of the A-V node or A-V bundle fibres.

2) Compression of the A-V bundle by scar tissue or by calcification of the heart

3) Inflammation of the A-V node or A-V bundle eg. from myocarditis, caused by diphtheria, rheumatic fever etc.

#### Prolonged P-R (or P-Q) Interval-First Degree Block :

When the P-R interval increases to greater than 0.20 second, the P-R interval is said to be prolonged, and the patient is said to have first degree incomplete heart block.



#### Second Degree Block:

When conduction through the A-V bundle is slowed enough to increase the P-R interval to 0.35 to 0.45 second, the action potential is sometimes not strong enough to pass through the bundle into the ventricles.

There will be an atrial P wave but no QRS-T wave, and it is said that there are "dropped beats" of the ventricles. This condition is called second degree heart block.



#### Complete A-V Block (Third Degree Block):

When the condition causing poor conduction in the A-V node or A-V bundle becomes severe, complete block of the impulse from the atria into the ventricles occurs.

In this case, the ventricles spontaneously establish their own signal, usually originating in the A-V node or A-V bundle.

Therefore, the P waves become dissociated from the QRS-T complexes and there is no relation between the rhythm of the P waves and that of the QRS-T complexes as the ventricles have "escaped" from control by the atria, and they are beating at their own natural rate, usually controlled by signals generated in the A-V node or A-V bundle.

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#### Stokes-Adams Syndrome and Ventricular Escape

In some patients with A-V block, the total block comes and goes; that is, impulses are conducted from the atria into the ventricles for a period of time and then suddenly impulses are not conducted.

Each time A-V conduction ceases, the ventricles often do not start their own beating until after a delay of 5 to 30 seconds.

Then some part of the Purkinje system beyond the block, usually in the distal part of the A-V node beyond the blocked point in the node, or in the A-V bundle, begins discharging rhythmically at a rate of 15 to 40 times per minute and acting as the pacemaker of the ventricles. This is called **ventricular escape**.

Because the brain cannot remain active for more than 4 to 7 seconds without blood supply, most patients faint a few seconds after complete block occurs because the heart does not pump any blood for 5 to 30 seconds, until the ventricles "escape."

After escape, however, the slowly beating ventricles usually pump enough blood to allow rapid recovery from the faint and then to sustain the person. These periodic fainting spells are known as the **Stokes-Adams syndrome**.

#### **Ventricular Fibrillation**

Ventricular fibrillation results from cardiac impulses that have gone berserk within the ventricular muscle mass, stimulating first one portion of the ventricular muscle, then another portion and eventually feeding back onto itself to re-excite the same ventricular muscle over and over-never stopping.

When this happens, many small portions of the ventricular muscle will be contracting at the same time, while many other portions will be relaxing. Thus, there is never a coordinate contraction of all the ventricular muscle at once, which is required for a pumping cycle of the heart.

When the normal cardiac impulse in the normal heart has traveled through the extent of the ventricles, it has no place to go because all the ventricular muscle is refractory and cannot conduct the impulse farther.



Therefore, that impulse dies, and the heart awaits a new action potential to begin in the atrial sinus node.

There are three different conditions that can cause this impulse to continue to travel around the circle, that is, to cause "re-entry" of the impulse into muscle that has already been excited.

This is called a "circus movement."

1) if the pathway is too long, by the time the impulse returns, the originally stimulated muscle will no longer be refractory and the impulse will continue again (eg. in dilated hearts).

2) if the length of the pathway remains constant but the velocity of conduction becomes decreased (eg. due to blockage of Purkinje system or ischemia of muscle).

3) the refractory period of the muscle might become greatly shortened. A shortened refractory period commonly occurs in response to various drugs or after repetitive electrical stimulation.



Initiation of fibrillation in a heart when patches of refractory musculature are present and continued propagation of fibrillatory impulses in the ventricle

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One of the most important features of fibrillation is the division of impulses. When a depolarization wave reaches a refractory area in the heart, it travels to both sides around the refractory area. Thus, a single impulse becomes two impulses.

In this way, many new wave fronts are continually formed by chain reactions until, finally, there are many small depolarization waves traveling in many directions at the same time. This irregular pattern of impulse travel causes many circuitous routes for the impulses to travel, greatly lengthening the conductive pathway.

In ventricular fibrillation, the electrocardiogram shows no tendency toward a regular rhythm of any type.

#### **Atrial Fibrillation**

The mechanism of atrial fibrillation is identical to that of ventricular fibrillation, except that the process occurs only in the atrial muscle mass.

A frequent cause of atrial fibrillation is atrial enlargement resulting from heart valve lesions that prevent the atria from emptying adequately into the ventricles. The dilated atrial walls provide long conductive pathway as well as slow conduction, both of which lead to atrial fibrillation.



In the electrocardiogram, there are no P waves or only a high-frequency, very low voltage wavy record. The QRS-T complexes are normal but their timing is irregular.