

Introduction to Electrocardiography

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The Genesis and Conduction of Cardiac Rhythm

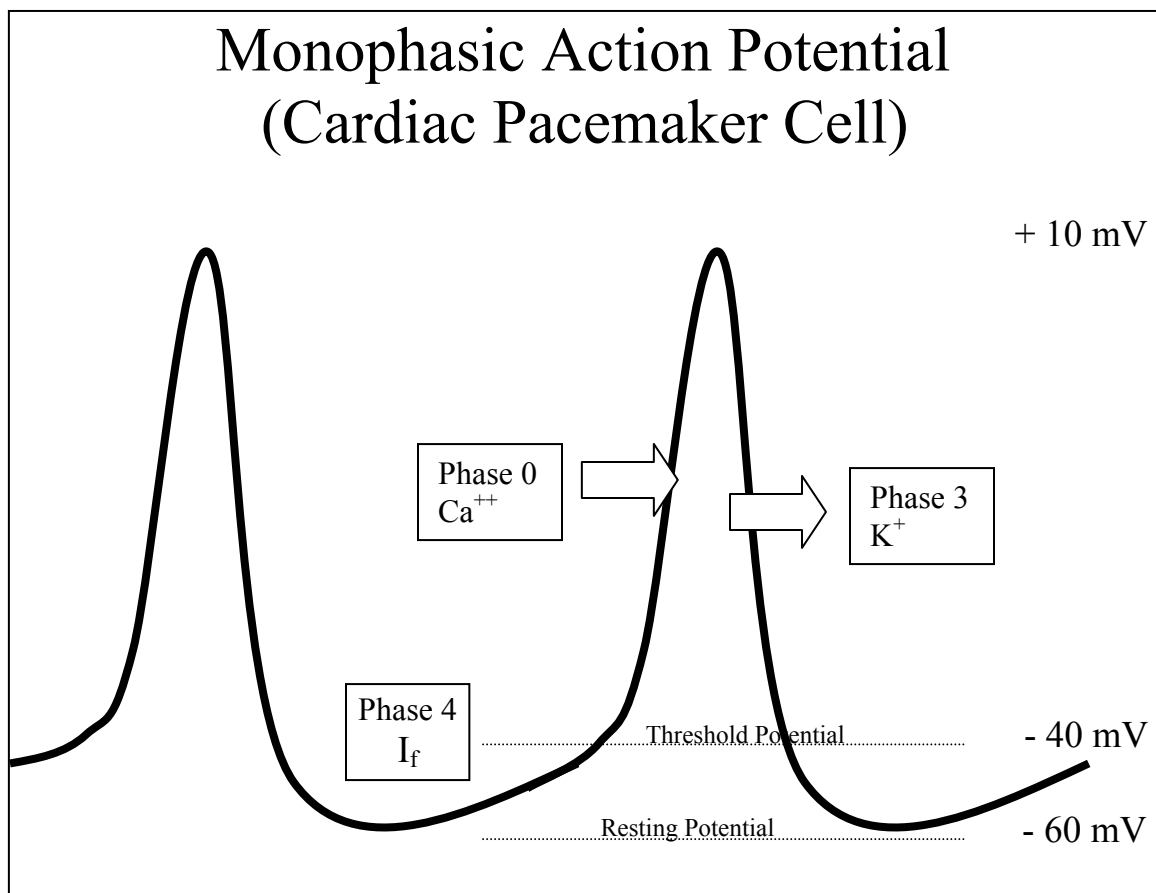
Automaticity is the cardiac cell's ability to spontaneously generate an electrical impulse (**depolarize**). Cells that are dedicated to the purpose of generating an impulse to maintain a heart rate commensurate with the body's need are called **pacemaker cells**. Cells normally capable of generating an impulse are the sinus node, cells of the AV node, the bundle of His and Purkinje cells. The lower the site of the latent pacemaker, the slower the intrinsic rhythm. The **sinus node** is located at the intersection of the superior vena cava and the right atrium laterally. While the sinus node cells have an intrinsic automaticity of their own, they are influenced by the sympathetic and parasympathetic nervous system. The former increases the heart rate while the latter slows the heart rate. During sleep and in highly trained athletes, the parasympathetic nervous system tone is elevated and the heart rate is slow. During exertion and any other condition that the sympathetic nervous system tone is high, the heart rate is elevated. Cardiac cells with automaticity displaying the fastest rate take over pacemaker cells with slower rates. The sinus node has the fastest intrinsic rate, other cells capable of generating a rhythm (automaticity) are termed latent pacemakers. They can take over pacing the heart if the sinus node becomes dysfunctional (sick sinus syndrome).

Normally, the interior of cardiac cells are more negative than the exterior due to the distribution of the main intra and extracellular electrolytes; Na^+ , K^+ , Ca^{++} and Cl^- . The voltage differential between the interior and the exterior at rest (diastole) is called the **Resting Potential**. In myocardial cells, the interior is maintained more negative than the exterior by the extrusion of 3 Na^+ ions for every 2 K^+ ions pumped in by the Na^+/K^+ ATPase pump. The resting potential of sinus node cells is -50 to -60 mV, for ventricular muscle cells it is -80 to -90 mV. Cardiac cells endowed with automaticity depolarize spontaneously at a rate commensurate with the intrinsic rate of the cell. Once the membrane potential reaches a certain voltage it fires (depolarized) spontaneously. This voltage is called the **threshold potential**. For sinus node cells this threshold potential is approximately -40 mV. Compared with ventricular muscle cells, cells with automaticity spend little time at the resting potential beginning to depolarize soon after reaching their resting potential. The rate of spontaneous depolarization (automaticity) is dependent upon the slope of Phase 4 of the action potential (I_f), the level of the membrane resting potential and the level of the threshold potential (see figure below).

Movement of electrolytes across the impermeable cell membrane is through a number of **channels** (Na channels (I_{Na}), K channels (I_{K}), e.g.) that permit or prevent the movement of ions depending upon transmembrane voltage. The channel responsible for spontaneous depolarization (Phase 4) is called I_f . The **monophasic action potential (MAP)** describes

the changes in transmembrane potential plotted against time. The MAP of pacemaker cells such as the sinus node differs from the MAP of conduction and myocardial cells. Whereas calcium is the main electrolyte responsible for pacemaker cell depolarization, sodium is the main electrolyte responsible for depolarization of myocardial cells and cells dedicated to conduction of impulses.

The impulse generated in the sinus node depolarizes the surrounding atrium and conducts to the ventricle via the **AV node**. Conduction below the AV node and into the myocardial cells is via the specialized **His-Purkinje system**. These cells are specialized conduction cells and are localized to the **Right and Left Bundle Branches**. Cell-to-cell conduction across normal cells occurs across **gap junctions**. Within the gap junctions are different types of proteins that are responsible for cell-to-cell conduction of electrical impulses called **connexins**. Electrical conduction along the longitudinal axis of the cell is faster than it is perpendicular to its long axis.



The monophasic action potential of a cardiac cell endowed with automaticity. The rate of firing is dependent upon 1) the slope of Phase 4 (the steeper the faster the rate of depolarization), 2) the level of the resting membrane potential (the less depolarized the faster for any given phase 4 slope and firing threshold level) and 3) the level of the threshold potential (the closer to the resting potential, the faster the rate of spontaneous) depolarization

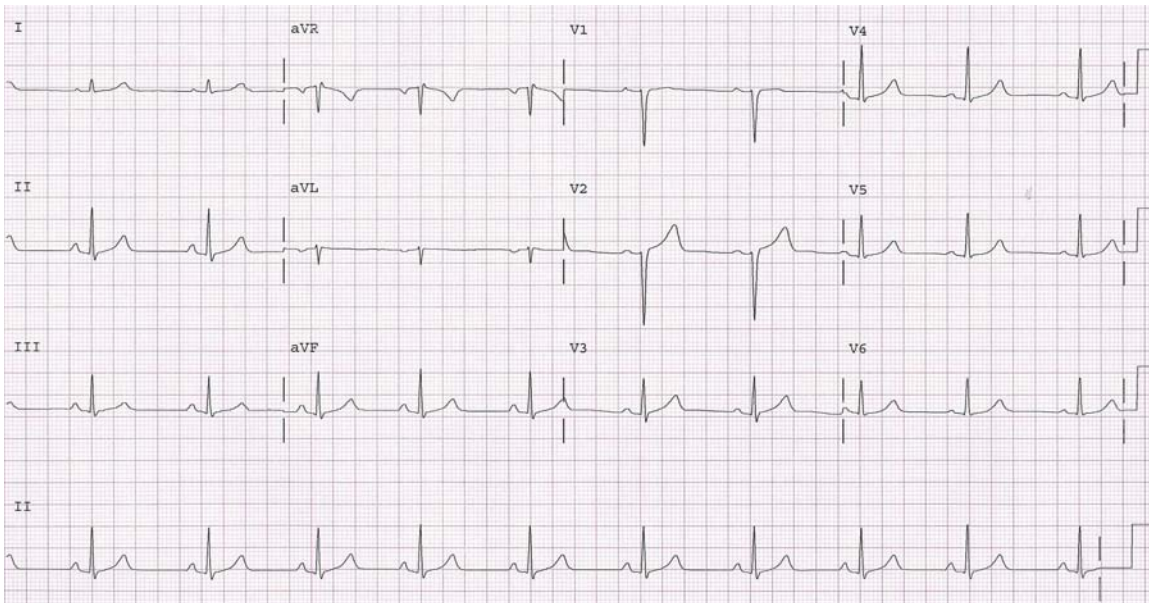
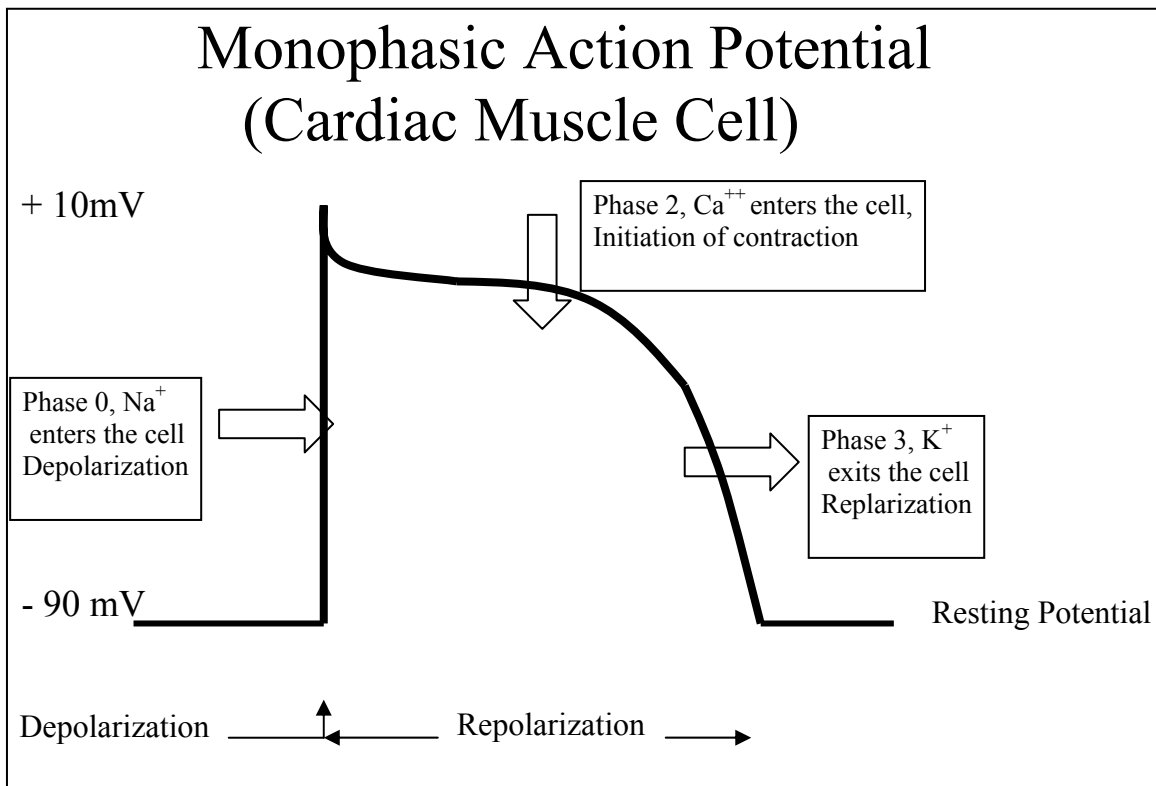


Figure 1. Normal 12-lead electrocardiogram

The standard 12-lead electrocardiogram consists of 6 bipolar leads (3 limb and 3 augmented limb leads; I, II, III, aR, aVL and aVF) and 6 unipolar precordial leads (figure 1). The electrocardiogram records the heart's electrical activity. It reflects the sequence of

cardiac depolarization from atrial depolarization (P wave), atrioventricular conduction (the PR interval), ventricular depolarization (the QRS interval) and ventricular repolarization (the T wave). The bipolar limb leads record the heart's electrical activity in a coronal plane (a coronal plane is the plane that transects the body from ear to ear or shoulder to shoulder into a ventral and dorsal sections) (figure 3). Each lead has a positive and a negative pole. Activity is recorded from the positive pole. Electrical activity moving towards the positive pole inscribes a positive wave on the electrocardiogram, moving away from the positive pole inscribes a negative wave).

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Figure 2. Location of the electrocardiographic leads on the body surface

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Figure 3. Location and polarity of the limb leads I, II, and III

Limb leads I and aVL reflect the left ventricle's high lateral wall electrical activity whereas leads II, III and aVF the left ventricle's inferior wall electrical activity.

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Figure 4. The Precordial leads V1-V6. In the lower diagram the crescent shaped cavity is the right ventricle, the oval cavity is the left ventricle. Separating both is the septum

The unipolar precordial leads (V1-V6) record the heart's electrical activity along its transverse plane (figure 4). Precordial leads V1-V4 record activity in the interventricular septum and anterior left ventricular wall. Leads V5-V6 the lateral left ventricular wall.

The electrocardiogram is inscribed on an electrocardiographic strip that is divided into small and large squares (fig 5). These squares are used to determine the time interval and the amplitude of the electrocardiographic signal. Since interpreting the electrocardiogram relies on comparing measured intervals with validated standards, accurate interpretation of these intervals is crucial in establishing a correct diagnosis. The electrocardiogram is recorded at a sweep speed of 25 mm/sec. At that speed, the small, 1 mm squares (x axis) each correspond to 0.04 secs. (40 mseconds). Five small squares in width and amplitude form a large square. The width of the large square is 5 mm corresponding to 0.2 seconds (200 milli seconds). Five large squares correspond to 1 second. The amplitude of the recorded signal is measured on the y axis. At the outset of each electrocardiogram a 1 milli Volt calibration signal is inscribed to make sure that the amplitude of the recorded electrocardiogram complies with accepted standards. Each 1 mm on the y axis corresponds to 0.1 mV amplitude. Similar to the x axis, five small squares form a large square whose height corresponds to 0.5 milli Volts (mV).

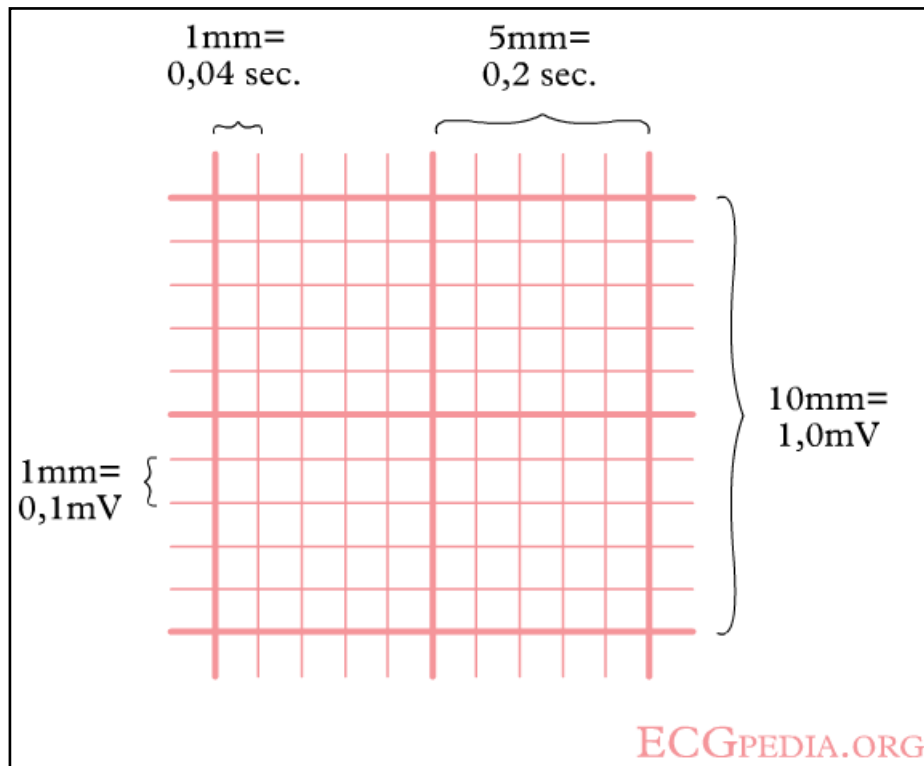


Figure 5. Electrocardiographic grid

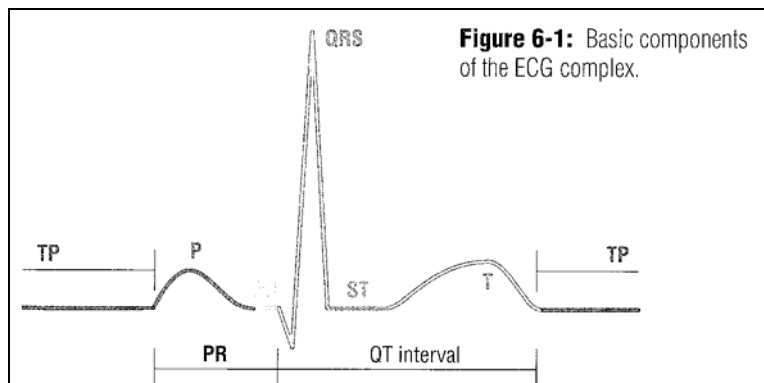


Figure 6. P, QRS and T waves. PR and QT intervals. See below.

Depolarization of the atria (right and left) is reflected in the P wave. Atrial abnormalities lead to changes in the duration, amplitude and morphology of the P wave. P waves are seen when there is organized atrial activity. P waves disappear in *atrial fibrillation* to be replaced by an irregular, low amplitude and high frequency baseline activity (figure 9). In *atrial flutter* the P waves are replaced by high frequency, rapid and regular continuous baseline activity (flutter waves) at rates of 250-350 bpm giving the saw-tooth or pike-fence appearance (figure 10).

The PR interval is measured from the beginning of the P wave until the beginning of the QRS complex (figure 6). This interval represents the time it takes for the atria to depolarize and the impulse to conduct to the ventricles via the AV node and the His / Purkinje system until ventricular depolarization begins (the QRS complex). The PR interval should not exceed 0.2 secs. (200 milliseconds or 5 small squares on standard electrocardiographic paper). In patients with *first degree AV block* the PR interval is in excess of 0.2 seconds. In *second degree AV block* one appropriately timed P wave fails to conduct. In *third degree AV block* P waves no longer conduct to the ventricles. If, in third (complete) heart block the ventricular escape (rescue rhythm) is junctional (high) the QRS complex is narrow (< 0.12 seconds or < 3 squares), if the escape is low, the rate is slower and the QRS width is ≥ 0.12 seconds. A PR interval of < 0.12 seconds is seen in *preexcitation* (figure 11).

The QRS complex represents ventricular depolarization. Since the left ventricular myocardial mass and thickness are considerably greater than that of the right ventricle, most of the QRS complex represents left ventricular depolarization (figure 6). Not all QRS complexes have all three components. The first negative deflection of the QRS complex is the Q wave, the first positive deflection the R wave and the second negative deflection the S wave. The normal QRS interval is < 0.12 seconds (three small squares). The major causes of QRS abnormalities are myocardial infarction, ventricular hypertrophy and infra nodal conduction abnormalities (right bundle branch block, left bundle branch block, interventricular conduction delay).

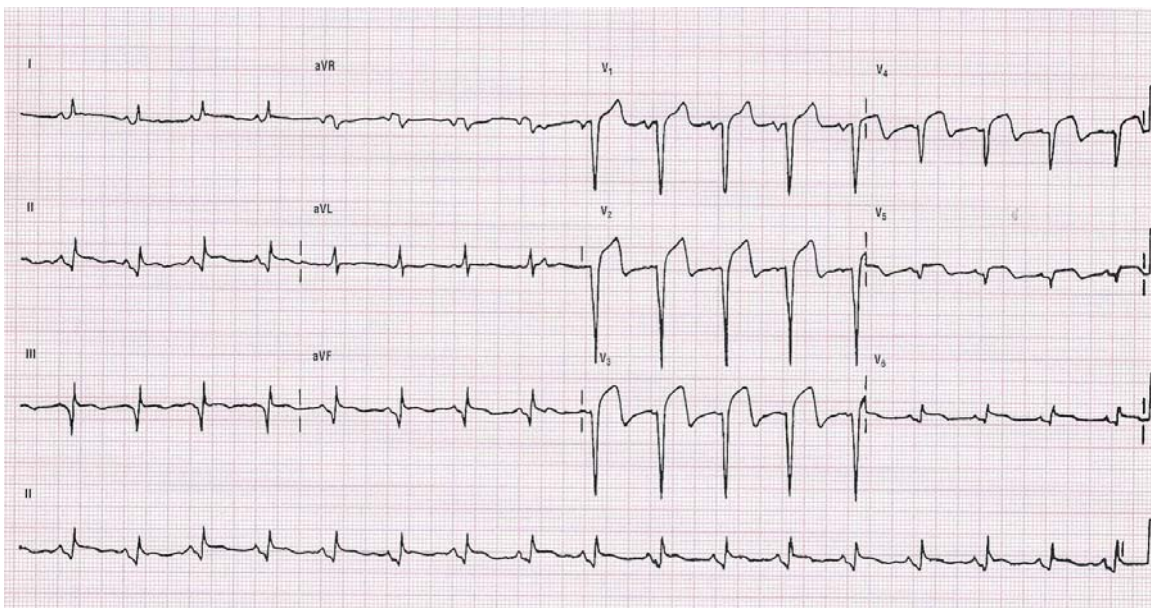


Figure 7. Marked ST-segment elevation in precordial leads V1-V5 reflecting injury of the underlying myocardium. The pronounced Q waves seen in the same leads reflect myocardial necrosis the result of a myocardial infarction. The location of the abnormalities point to the anterior septum and anterior wall.

The importance of the ST-segment rests on the changes (elevation or depression relative to the TP segment) in this segment seen with coronary artery disease. Patients presenting with a clinical picture consistent with an acute coronary syndrome (chest pressure,

shortness of breath) and display new or dynamic shifts in the ST-segment places them in a higher risk category. If the ST-segment rises in at least two contiguous leads the patient is probably suffering from an ST elevation myocardial infarction (STEMI) the result of total occlusion of the culprit coronary vessel and should undergo immediate revascularization (thrombolysis or percutaneous catheter intervention) (figure 7).

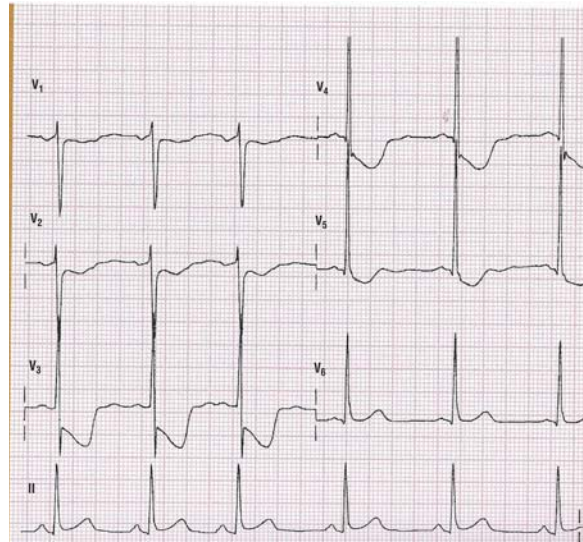


Figure 8. The ST-segment changes seen here consist of downsloping depression consistent with ischemia. A patient presenting with the clinical picture of an acute coronary syndrome and ST-segment depression would NOT be a candidate for thrombolysis.

The QT interval represents ventricular repolarization. The significance of this component of the electrocardiogram is in its duration. Prolongation of the QT interval may predispose to polymorphic ventricular tachycardia known as torsade de pointe. The normal QT interval is 0.45 seconds in males, 0.46 seconds in females. Prolongation of the QT interval may be congenital (long-QT interval), a cause of sudden cardiac death, or acquired (potassium channel blocking drugs, sotalol for example).

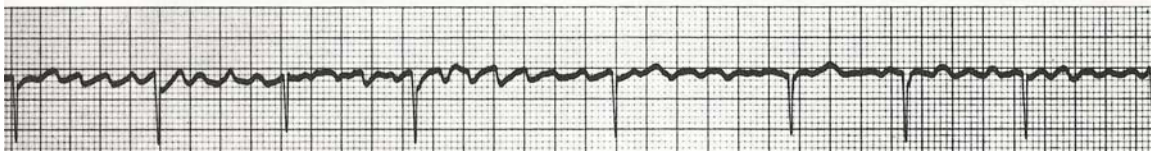


Figure 9. Atrial fibrillation

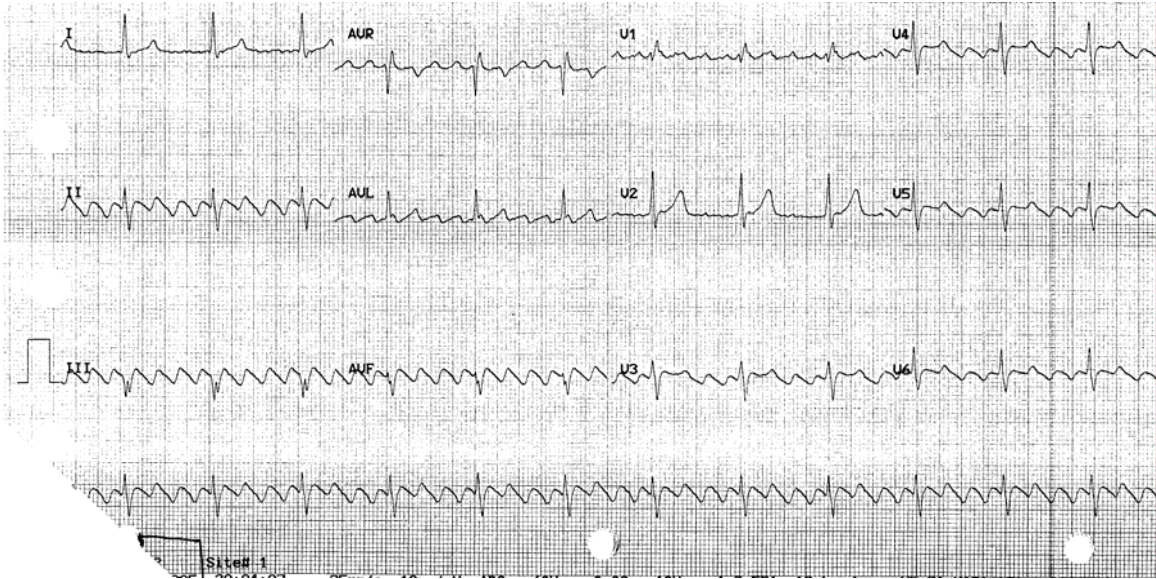


Figure 10. Atrial flutter



Figure 11. Preexcitation; Note the short PR interval and the “delta” wave