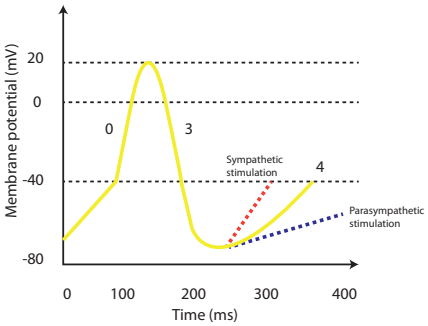
ELECTRICAL PROPERTIES OF THE HEART



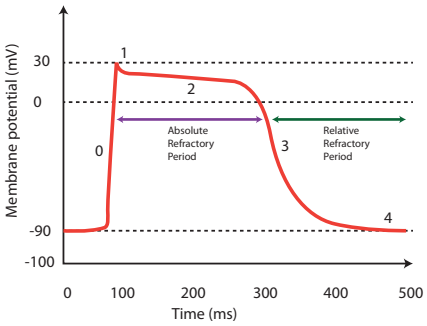
Ionic basis of the slow-response cardiac action potential The sino atrial node and the AV node have the same ionic basis although the AV node is slower. The adjacent diagram represents the SA node. In the slow response cardiac action potential there is no resting state; rather there is a pacemaker potential which generates cardiac autorhythmicity. Phases 1 and 2 (of the fast response action potential) are absent in the SA/AV node as there is no depolarisation plateau.

PHASE 0 Depolarisation is produced by the opening of voltage-gated calcium channels (L-Type) and inward movement of positive ions.

PHASE 1/2 are absent

PHASE 3 Repolarisation occurs as Ca^{2+} channels close and and K^{+} channels open. Efflux of K+ from within the cell repolarises the cell fairly rapidly.

PHASE 4 The pacemaker potential is produced by a fall in membrane potassium permiability and an increase in a slow inward current. The slow inward current consists of a voltage gated increase in calcium permiability (via T-Type channels) and activity of the electrogenic sodium-calium exchange system, driven by inward movement of calcium ions. This pacemaker activity brings the cell to threshold potential.



Ionic basis of the fast-response cardiac action potential Atrial and ventricular muscle and purkinje fibre action potentials differ from those in nerves as they are much longer in duration, with a distinct plateau phase when depolarisation is maintained.

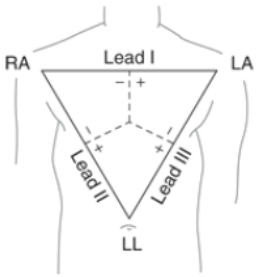
PHASE 0 The cell is rapidly depolarised from the resting membrane potential by a rise in sodium permiability via fast sodium channels. The slope is almost vertical. The the membrane is less negative then many sodium channels will be closed, thus the response will not be as quick.

PHASE 1 Repolarisation begins to occur as sodium channels close and potassium channels open.

PHASE 2 A plateau occurs owing to the opening of L-type Ca^{2+} channels which offset the action of K channels and maintains depolarisation. During this time no further depolarisation is posible, this represents the absolute refractory period.

PHASE 3 The L-type Ca^{2+} channels close and K efflux now causes repolarisation as seen before this accelerates through positive feedback. It is now possible to cause another depolarisation although the force of the contraction will be diminished. This the relative refractory period.

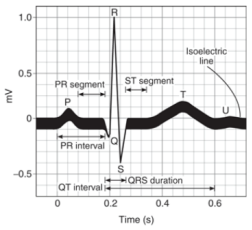
Cardiac excitation - contraction coupling Contraction of cardiac fibres is by the interaction of actin and myosin filaments in the presence of calcium. Tropomyosin lies in the groove and prevents interaction of the two, this action is modulated by the troponin complex which is activated by calcium (see previous figure). Similar to skeletal muscle contraction in cardiac muscle results from the temporary release of calcium from the sarcoplasmic reticulum. Unlike skeletal muscle the the SR Ca^{2+} release is triggered by the inward flow of Ca^{2+} across the cell membrane and the T-Tubules during the action potential. Cardiac muscle does not contract in the absence of calcium in the ECF. This form of excitation contraction coupling may be described as 'calcium triggered calcium release' and is an amplification process whereby the movement of a small amount of calcium into the cell causes a temporary release of a much larger amount of calcium from the SR. Increases in intracellular Ca increase the force of contraction. When the cardiac myocyte relaxes the sarcoplasmic reticulum actively takes up the calcium and sequesters it (lusitropy), the calcium which acted as a trigger is transported out of the cell by active and counter transport methods.



The ECG Electrodes are the sites at which an electrical potential is measured, while ECG leads record the difference in potentials between two electrodes. Standard surface electrodes (right and left arm, right and left leg, and the six precordial electrodes) measure the electrical potential at a site. Leads may unipolar or bipolar. Bipolar leads, which include I, II and III measure the difference between two surface electrodes, and drawn together they form Einthoven's triangle. The central terminal of Wilson, is calculated from the average voltage of the limb leads. This idealized site is meant to represent a reference at the center of Einthoven's triangle where total current is zero. From this reference point the unipolar leads; aVR, aVL and aVF plus the chest leads are calculated.

	Normal Durations		
Intervals	Average	Range	Events in the Heart during Interval
PR Interval	0.18	0.12-0.20	Atrial depolarisation and conduction through the AV node
QRS duration	0.08	up to 0.10	Ventricular depolarisation and atrial repolarisation
QT interval	0.40	up to 0.44	Ventricular depolarisation and subsequent repolarisation
ST interval (QT minus QRS)	0.32	....	Ventricular repolarisation (during T wave)

PR is actually from the start of the PR segment to the start of the QRS. The PR shortens as the heart rate increases.



Factors which influence cardiac electrical activity

Sodium a fall in plasma Na^{+} may be associated with low voltage ECG complexes.

Potassium in the setting of hyperkalaemia the most common finding is tall T waves which is a manifestation of abnormal repolarisation. At higer levels paralysis of the atria and prolongation of the QRS complexes can occur. Ventricular arrhythmias may develop. The resting membrane potential of muscle fibres decreases as the extracellular K^{+} concentration increases. The fibres eventually become unexcitable and the heart stops in diastole. In the setting of hypokalaemia causes prolongation of the PR interval, prominent U waves, and occasionally late T-Wave inversion in precardial leads.

Calcium hypercalcaemia enhances myocardial contractility. There is shortening of the QT interval due to a shorter ST segment. In experiments large doses of calcium prevents the heart from relaxing and the heart stops in systole (calcium rigor) however calcium levels are rarely significant in the clinical setting. Hypocalcaemia causes prolongation of the ST segment and consequently the QT interval.

Magnesium Hypomagnesiumaemia results in several ECG changes and may be a result of concurrent hypokalaemia or its actions on several cardiac membrane channels including those responsible for calcium and poassium. Changes seen include Widening of the QRS complex and peaking of T waves have been described with modest magnesium loss, while more severe magnesium depletion can lead to prolongation of the PR interval, progressive widening of the QRS complex, and diminution of the T wave.

Adenosine Adenosine receptors exist in both atrial and nodal tissues and activate the K^{+} current which transiently hyperpolarises the cell. This has little effect on in atrial tissue (already at -90mV) but drives the SA and AV nodal tissue further from their threshold and therefore slows its rate. It also antagonises adenylyl cyclase reduces intracellular Ca^{2+} and also slows conduction. The result is transient AV node block which is used in supraventricular tachycardias to restore sinus rhythm.

Sympathetic Stimulation acts via noradrenaline at the β1 receptors. It increases heart rate by increasing the rate of phase 4 depolarisation (see figure top left). This is through increased Na^{+} influx during phase four. It also increases inward Ca^{2+} influx which increases conduction through the AV node, decreasing the PR interval. This is known as the positive dromotropic effect.

Parasympathetic Stimulation is based on acetylcholine acting on muscarinic receptors which results in the opposite effects of sympathetic stimulation, decreasing HR by reducing Na^{+} influx and therefore extending phase four duration in the slow response myocytes and decreasing Ca^{2+} influx which slows conduction through the AV node.

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