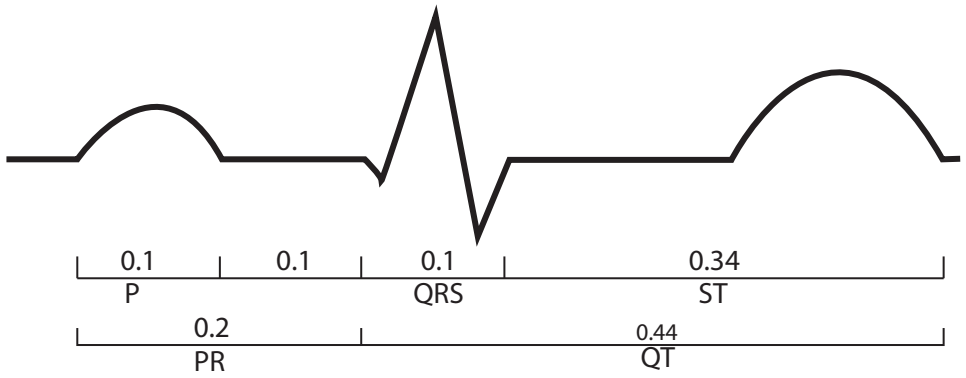
JULY 2007
VIVA 3

Explain the physiological basis of the ECG

"Please draw, label and provide time intervals for the ECG"



"Describe the electrodes and what they measure"

Measures the difference between two electrodes
Forms Einthoven's triangle
Central terminal of wilson is the sum of the three limb leads and is the centre of the triangle

«Discuss the changes noted with the following electrolytes and hormones»

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 i ncreases 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.