Q1 March 2009

Relate the surface ECG to the events of the cardiac cycle. Describe how the PR, QRS, and QT intervals may be prolonged by the action of drugs.

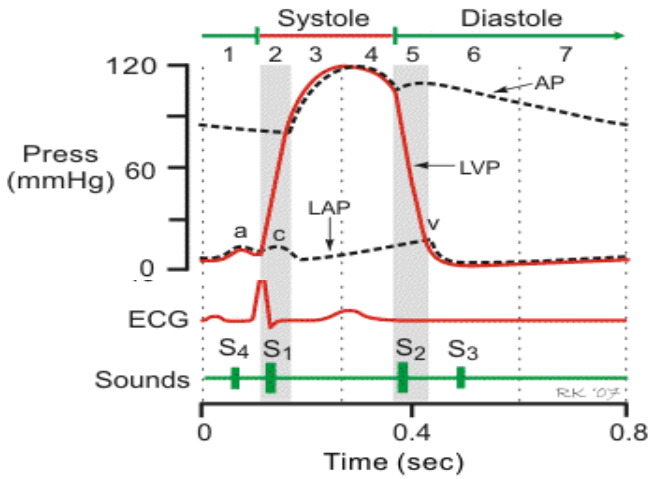

Diagram modified from CV Physiology website: http://www.cvphysiology.com/Heart%20Disease/HD002.htm

Phase 1 - Atrial contraction

Begins with the p wave, which represents electrical depolarisation of the atria. PR interval (normal duration 0.12-0.2s) includes conduction through the AV node. Last phase of diastole.

Phase 2 - Isovolumetric contraction

Begins with the QRS cycle, which represents ventricular depolarisation. Peak of the R wave corresponds to beginning of LV contraction. Coincides with closure of AV valves (1st heard sound), and rapid rise in ventricular pressure, without any change in volume.

Phase 3 - Rapid ejection

Aortic and pulmonary valves open, resulting in rapid ejection of blood.

Phase 4 - Reduced ejection

~200ms after QRS and the beginning of ventricular contraction, ventricular repolarisation occurs (T wave). Repolarisation causes a decline in ventricular ejection rate.

Phase 5 - Isovolumetric relaxation

Aortic and pulmonary valves close (2nd heart sound) as intraventricular pressure decreases.

Phase 6 - Rapid filling

Intraventricular pressures fall below atrial pressures, and the AV valves open, causing ventricular filling.

Phase 7 - Reduced filling

Intraventricular pressure rises and rate of filling falls. Cycle returns to Phase 1 with atrial depolarisation.

Interval	Drug	Mechanism
PR	Digoxin	Increased vagal activity, augmenting direct AV nodal slowing (increased refractory period)
QRS	Amiodarone	Inhibits inward Na^+ and Ca^{2+} flux, causing decreased depolarisation speed
QT	Amiodarone	Inhibits K^+ outflux from cell, causing delayed repolarisation