Q7 March 2009
Define afterload. Describe the factors that can affect left ventricular afterload.

Afterload is the force per unit of cross-sectional area (i.e. wall stress) that opposes the shortening of the ventricular cardiac muscle i.e. the resistance that the ventricle has to overcome in order to eject blood during systole.

Therefore a certain amount of wall tension needs to be generated in the cardiac muscle before contraction and ejection of blood will occur. Wall tension in the ventricle is used as an estimation of afterload. Wall tension is given by the equation:

$$\text{Wall tension} \propto \frac{\text{Ventricular pressure} \times \text{Ventricular radius}}{\text{Ventricular wall thickness}}$$

Ventricular radius
Increased ventricular radius (ventricular dilation) causes increased wall tension, and therefore increased afterload.

Ventricular wall thickness
Increased wall thickness (ventricular hypertrophy) decreases wall tension, as the more sarcomere units, the less tension experienced by each at a given pressure and radius.

Ventricular pressure
Increased systemic vascular resistance increases afterload by increasing aortic pressure. This increases the intraventricular pressure that needs to be generated in order to open the aortic valve and eject blood during systole.

Aortic stenosis causes increased afterload, as the ventricle has to generate increased intraventricular pressure to overcome the transvalvular pressure gradient and eject blood during systole.

Decreased pleural pressure causes increased LV afterload, as decreased pressure around the heart means the left ventricular myocytes must develop greater tension to generate the required intraventricular pressures to eject blood during systole. Positive pressure ventilation will increase pleural pressure and decrease afterload.

Other
Increased blood viscosity increases afterload, as the sluggish flow of blood resists ejection by the left ventricle.