

## Q21 Define preload and describe the determinants of preload (March 2010)

Preload is defined as the stretch on myocardial fibres just prior to contraction. The Frank Starling mechanism states that the force of cardiac contraction is dependent on the initial stretch of the myocytes. Alterations in myocyte length affect the calcium sensitivity of the myofilaments and also the degree of interaction between myosin and actin filaments. Hence, higher preload results in an increase in initial fibre length, increasing the force of contraction (up to a point - beyond an optimal fibre length, contraction actually becomes impaired).

Determinants of preload include:

- **CARDIAC FACTORS**
  - **Heart rate** → the slower the heart rate the greater the filling time of diastole and the greater the preload
  - **Atrial contraction** → a change in atrial contraction due to sympathetic stimulation or increased filling (via Starling) will affect ventricular filling and hence preload. Atrial arrhythmias (eg, AF) will affect atrial filling and emptying and may result in loss of the atrial kick component of diastole, which gives the ventricle ~30% of its filling volume.
  - **Ventricular compliance** → the greater the compliance, the greater the filling at a given pressure and hence greater preload. Compliance varies with volume; it is greatest at low volume and smallest at high volumes
  - **Afterload** → changes in afterload of the left ventricle will alter stroke volume by changing end systolic volume. This will lead to a secondary change in preload.
  - **Valvular disease** → mitral or tricuspid stenosis will reduce ventricular filling during diastole and thus reduce preload. Aortic or pulmonary stenosis will increase the afterload on the ventricle and may result in a reduction in ejection fraction, thus increasing the LVESV and hence preload.
  - **External compression** → eg, large pericardial effusion, tamponade or space occupying lesion will reduce ventricular filling

### SYSTEMIC FACTORS

- **Venous return** → changes in VR (due to alterations in intravascular volume, venous compliance, the gradient favouring venous return, skeletal muscle pump action, the effects of gravity, or intrathoracic pressure) will change the volume of blood returning to the right atrium and thus the ventricular preload
- **Changes in aortic pressure** → will affect afterload as above