Describe the cardiovascular effects of a sudden increase in afterload.

Afterload is the sum of forces, both elastic and kinetic, opposing ventricular ejection

- This definition is a bit wordy but avoids using the words "resistance" and "impedance", which are strictly defined in physics (and crudely applied in medicine), and may be leapt on by the cruel examiner
- Afterload = left ventricular wall tension required to overcome resistance to ejection (impedance to ejection of blood from the heart into the arterial circulation).
- Afterload represents all the factors that contribute to total myocardial wall stress (or tension) during systolic ejection

Determinants of Afterload
Afterload is equal to ventricular wall stress, which is given by the equation:

$$\theta \propto \frac{P \times r}{T}$$

where:

- $\theta$ is ventricular wall stress
- $P$ is ventricular transmural pressure
- $r$ is ventricular chamber radius
- $T$ is ventricular wall thickness

Similar to Laplace, $P = 2T/r$, where $T$=tension. Stress = Tension/Thickness. $\theta \propto PR/T$

Effects of sudden increase in afterload can be demonstrated using a Left ventricular PV loop:

- End-Systolic Volume is increased, causing a reduction in stroke volume
- Initially, Left atrial pressure decreases
- Increased End systolic volume leads to secondary increase in end diastolic volume, hence increasing ventricular filling
- This secondary increase in preload enables the ventricle to contract with greater force (Frank-Starling mechanism) which partially offsets the reduction in stroke volume
- In patients with impaired left ventricular function, the decreased in stroke volume cannot be compensated
• ventricular end-systolic pressure: increases
• ventricular end-diastolic pressure: increases

• cardiac output (HRxSV) – remains the same
  o Initial drop in SV is compensated by increased pre-load
  o In a failing heart, the drop in SV causes subsequent stimulation of barceptors, which in turn causes increased heart rate and can potentially return cardiac output to normal
• Increase in afterload → Anrep → Small ↑Contractility to compensate
  o Mechanism: ↑AL → sustained ↑stretch (prolonged isovolumetric contraction) → ↑ Ca induced Ca release → ↑ contractility
  o Purpose: ↑AL → ↓SV and ↑ ESV
• myocardial oxygen demand and myocardial work: Increased Afterload will increase the pressure during contraction, hence increasing MVO2 for internal work. This might be partially offset by the reduction in external work (due to decreased stroke volume) depending on the cause of the increased afterload
  
• coronary blood flow is autoregulated to remain normal

Examiner Comments:
21% of candidates passed this question.
It was expected the answer would start with a definition of afterload and then proceeded to indicate what effects this increase in afterload would have on ventricular end-systolic pressure, ventricular end-diastolic pressure, left atrial pressure, cardiac output, myocardial oxygen demand and myocardial work, coronary blood flow and systemic blood pressure.
Most candidates who failed to pass this question submitted answers that were just too brief, only including a small subset of the material required. Very few candidates included any mention of myocardial oxygen demand or myocardial work or the impact upon the cardiac output. A number of candidates included a detailed description of the Sympathetic Nervous System and the Renin-Angiotensin system, material which was not asked for. There were quite a number of incorrect perceptions about what effect a sudden increase in afterload would have on the systemic blood pressure. Candidates who mentioned the baroreceptor response and the stretch receptor response where rewarded with additional credit.