Explain in physiologic terms the effect of severe aortic stenosis on myocardial oxygen supply and demand.

**Background**

Aortic stenosis = pathological narrowing of aortic valve (AV) orifice

Classical clinical triad of dyspnoea, angina and syncope on exertion

<table>
<thead>
<tr>
<th>AS severity</th>
<th>AV area (cm^2)</th>
<th>Mean gradient (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>&gt; 1.5</td>
<td>&lt; 30</td>
</tr>
<tr>
<td>Moderate</td>
<td>1.0 – 1.5</td>
<td>30 – 50</td>
</tr>
<tr>
<td>Severe</td>
<td>&lt; 1.0</td>
<td>&gt; 50</td>
</tr>
</tbody>
</table>

*(European Society of Cardiology 2007)*

**Effect on myocardial O_2 supply**

Myocardial O_2 supply depends on:
- coronary blood flow → depends on coronary perfusion pressure and vascular resistance
- heart rate (thus ratio of time in systole:diastole)
- O_2 content of blood → depends on [Hb]

Coronary perfusion pressure for LV (or RV) = aortic pressure − LV (or RV) pressure

Severe AS → large pressure gradient across aortic valve → ↓*aortic pressure* + ↑*LV systolic pressure* → ↓LV (and RV) coronary perfusion pressure → ↓coronary blood flow

Severe AS → LV systole ejects turbulence jet of blood at high velocity → ↓*lateral flow* into coronary arteries (Venturi effect) → ↓coronary blood flow

Severe AS → LV hypertrophy over time → extrinsic compression of coronary arteries → ↓coronary artery radius → ↑*coronary vascular resistance* (Hagen-Poiseuille relationship) → ↓LV coronary blood flow

Severe AS → ↓CO and ↓MAP → compensatory sympathetic stimulation → ↑heart rate → ↓*diastole:systole ratio* → ↓coronary blood flow (affects LV > RV)

Severe AS may be associated with intestinal angiodysplasia (Heyde’s syndrome) → ↓*haemoglobin* → ↓myocardial O_2 supply

**Effects on myocardial O_2 demand**

Myocardial O_2 demand depends on:
- wall stress → which is proportional to ventricular pressure, radius, inversely proportional to thickness
- heart rate
Severe AS $\rightarrow$ ↑afterload $\rightarrow$ compensate by ↑ventricular pressure to maintain cardiac output $\rightarrow$ ↑wall stress $\rightarrow$ ↑O$_2$ demand

Severe AS $\rightarrow$ compensate by LV hypertrophy $\rightarrow$ ↓wall stress $\rightarrow$ ↓O$_2$ demand

Severe AS $\rightarrow$ eventually leads to decompensation $\rightarrow$ LV dilatation and failure $\rightarrow$ ↑LV radius $\rightarrow$ ↑wall stress $\rightarrow$ ↑O$_2$ demand

Severe AS $\rightarrow$ flow limitation with ↓cardiac output $\rightarrow$ compensate by ↑sympathetic tone $\rightarrow$ ↑heart rate + ↑inotropy $\rightarrow$ ↑O$_2$ demand

**Overall effects**

Severe AS results in both:
- decreased myocardial O$_2$ supply
- increased myocardial O$_2$ demand

$\therefore$ may result in myocardial ischaemia $\rightarrow$ clinically observed dyspnoea, angina and ventricular failure

**Examiner’s comments** – 45% of candidates passed this question

- first part of the answer was a description of aortic stenosis with extra marks for giving the measure of severity
- the main part of the answer interrelated the effects of increased myocardial work (Laplace Law) and decreased myocardial perfusion
- additional marks were awarded for discussing the varying effects of LV hypertrophy on both supply and demand
- marks are also awarded for mentioning how increased heart rate increase demand and decrease supply