Describe the blood-supply to the liver.

The liver serves as a blood reservoir (30ml per 100g, half of which may be mobilised in hypovolaemia), and receives 25% of cardiac output from a unique dual blood supply:

- **Hepatic arterial system**, which supplies about one-third of blood, but 40-50% of O₂. Hepatic arterial blood has an \( \text{SpO}_2 \) of ~98%, as would be expected. It is a high-pressure, high-resistance, high-flow system (average velocity 18cm.s\(^{-1}\)), with the capacity to autoregulate. Pulsatile.

- **Portal venous system**, which supplies the remaining two-thirds of blood. It is a low-resistance, low-pressure, low-velocity system (average flow 9cm.s\(^{-1}\)), with no capacity to autoregulate. Non-pulsatile, valveless. High Protein content.

The \( \text{SpO}_2 \) of portal venous blood varies depending on gut activity:
- In the resting gut, \( \text{SpO}_2 \) is ~85%
- In the active gut, \( \text{SpO}_2 \) is ~75%

**Functional anatomy**

- basic unit is the liver lobule
- consists of central vein in the middle of hexagon triad of hepatic artery, bile duct and portal vein
- bile canaliculi radiate out from central vein

**Physiology**

- total blood flow is 1500ml (25% of CO)
- has a capacitance function, storing 450mL (30ml/100gm) blood- utilised during hypovolaemia
- consists of 30% hepatic artery supply and 70% portal vein supply
- both contribute to oxygenation (hepatic artery 50%, portal vein 50%)
- the liver demonstrates variable oxygen extraction to adapt to changes in portal vein oxygenation

**Measurement**

- by indirect clearance methods such as indocyanine green

**Regulation of Flow**

- As with other organs, blood flow is autoregulated via intrinsic and extrinsic mechanisms, and may be affected by external factors.

**Intrinsic Autoregulation**

- **Myogenic autoregulation**
- **Hepatic arterial buffer response**
  - This is also known as the "hepatic artery-portal venous semi-reciprocal interrelationship".
  - Hepatic arterial resistance is proportional to portal venous blood flow, such that a reduction in portal venous flow causes a decrease in hepatic arterial resistance and increases hepatic arterial flow.
  - This is probably mediated by adenosine.

**Extrinsic Autoregulation**

- **Autonomic Nervous System**
  - Both the hepatic and portal vasculature have sympathetic innervation:
The hepatic artery has D-, β-, and α-adrenoreceptors. The portal vein has only α-adrenoreceptors. Activation of these receptors causes venoconstriction, reducing the compliance of the hepatic vasculature and mobilising up to 250ml of blood in times of sympathetic stress.

- **Endocrine and hormonal effects**

A number of substances affect portal flow:

<table>
<thead>
<tr>
<th>Hormone</th>
<th>Portal Vein Effect</th>
<th>Hepatic Artery Effect</th>
<th>Overall Effect on Flow</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenaline</td>
<td>Constriction</td>
<td>Constriction (α), then dilation (β)</td>
<td>Reduced</td>
</tr>
<tr>
<td>Glucagon</td>
<td>Dilation</td>
<td>-</td>
<td>Increased</td>
</tr>
<tr>
<td>Secretin</td>
<td>-</td>
<td>Dilation</td>
<td>Increased</td>
</tr>
<tr>
<td>VIP</td>
<td>-</td>
<td>-</td>
<td>Increased</td>
</tr>
<tr>
<td>Angiotensin II</td>
<td>Constriction</td>
<td>Constriction</td>
<td>Reduced</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>Constriction</td>
<td>Constriction</td>
<td>Reduced</td>
</tr>
<tr>
<td>PCO₂</td>
<td>Constriction</td>
<td>-</td>
<td>Reduced</td>
</tr>
</tbody>
</table>

**External Factors**

Flow in the hepatic vein is dependent on venous return:

- Increased venous return (e.g. negative-intrathoracic pressure) increases hepatic flow
- Decreased venous return (e.g. positive-pressure ventilation, tamponade, haemorrhage), reduces hepatic flow, and in extreme cases flow may only occur intermittently throughout the cardiac cycle
- **Feeding** increased GIT blood flow and indirectly increases hepatic flow
- **Exercise** reduces both portal vein and hepatic arterial flow

**Microvasculature**

Hepatic arterioles and portal venules form the **hepatic triad** with a bile canaliculi. Hepatic arterioles and venules anastomose to form **sinusoids**, which create a specialised low-pressure (~2mmHg) capillary system which drains into the central veins of the hepatic acinus.

This arrangement:

- **Optimises hepatic O₂ extraction**
  
  Increased hepatic O₂ demand is met by increasing O₂ extraction, rather than by increasing flow (as occurs in the heart).
- **Prevents shunting and retrograde flow**

**Examiner Comments:**

A correct description of the vascular anatomy; the contribution and composition of hepatic artery and portal vein flow to total hepatic flow and how this is regulated would be awarded with a pass. An answer that expanded on these main points received additional marks. The interdependence of hepatic artery and portal vein flow was not appreciated by any candidate. Either candidates knew the answer to this question or they did not. Some candidate(s) tried to guess at what the anatomy might be. This attracted no marks. Many candidates lacked sufficient knowledge to pass this question.

Syllabus I 2 d&g

No candidates (0%) passed this question.