B. 8 Pulmonary circulation

a. Outline the vascular anatomy and structure of the pulmonary and bronchial circulations.

The pulmonary trunk arises from the right ventricle and branches into left and right pulmonary arteries. These pass posterolaterally to the main bronchi and follow them into the lungs. The pulmonary arteries give off multiple branches, generally following the bronchi so that bronchopulmonary segments have their own artery and bronchus without anastomosis.

Pulmonary capillaries line the walls of alveoli. They form a mesh in which the holes are smaller than the vessels themselves. The capillaries have very thin walls which are fused to the basement membrane of the alveolar epithelium.

Pulmonary veins drain oxygenated blood from the pulmonary capillaries. They are generally at the periphery of bronchopulmonary segments and drain adjacent segments. The large veins do accompany the bronchi and arteries and drain as two veins from each lung into the left atrium. The upper vein drains the upper lobe on each side and the lower vein the lower lobe. On the right, the middle lobe is drained by the upper vein.

The histology of the pulmonary arteries is different from systemic arteries of similar size. There is little smooth muscle tissue and a large amount of elastin in the artery walls. The walls overall are thin compared to the diameter of the vessels. This is consistent with the low pressures of the pulmonary circulation. The pulmonary veins are very thin-walled.

The pulmonary capillaries are lined with endothelial cells which share their basement membrane with that of the type I pneumocytes lining the alveolar air space.

The bronchial arteries arise from the thoracic aorta or from the upper intercostal arteries. There may be one or more on each side. Occasionally additional bronchial vessels arise from the descending aorta and travel in the pleural ligament. They follow the bronchi, forming a capillary plexus around the large bronchi, supplying the bronchial muscle coat and forming a second plexus in the mucosa. These plexi extend as far as the respiratory bronchioles where they anastomose with the pulmonary vessels.

The deep bronchial veins drain the bronchi within the lung and join the pulmonary veins. The superficial bronchial veins drain the bronchi near the hilum outside the pleura and join the azygous on the right and accessory hemiazygous or intercostal vein on the left.

The histology of the bronchial arteries is the same as that of other systemic arteries.

b. Describe the physiological features of the pulmonary circulation and compare them with those of the systemic circulation.

The pulmonary circulation differs from the systemic circulation in several major respects. The high-pressure side of the pulmonary circulation carries deoxygenated blood and the low-pressure side oxygenated blood. Typical pulmonary arterial pressures are much lower than in the systemic circulation:

<table>
<thead>
<tr>
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<th>Right (mmHg)</th>
<th>Left (mmHg)</th>
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<tbody>
<tr>
<td>Ventricular</td>
<td>25/0</td>
<td>120/0</td>
</tr>
<tr>
<td>Arterial</td>
<td>25/8</td>
<td>120/80</td>
</tr>
<tr>
<td>Capillary</td>
<td>12→8</td>
<td>30→10</td>
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<tr>
<td>Atrial</td>
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<td>8</td>
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There is no need for higher pressures in the pulmonary circulation as there is little variation in the hydrostatic pressure to be overcome and less need for preferential perfusion of one area over another. The difference in pressure is reflected in the histology of the arterial vessels.

Flow in the pulmonary vessels is dependent not only on the arterial and venous
pressures but also on the airway pressure and lung volume as West’s zones show. With high airway pressures or low venous pressure, flow is limited by the collapse of vessels where airway pressure exceeds blood pressure. At high lung volumes, the vessels are supported not only by blood pressure but by elastic forces within the lung parenchyma, allowing the effective pressure outside larger vessels to be less than intrathoracic pressure.

The volume within the pulmonary circulation is around 0.5 to 1.0 l, depending on posture, the cardiac cycle and airway pressure.

The pulmonary circulation has a number of metabolic functions which distinguish it from the systemic circulation. It is the site of synthesis of a number of hormones, including prostaglandins, histamine and kallikrein. It is the main site of conversion of angiotensin I to angiotensin II. It is a major site for removal from circulation of bradykinin, prostaglandins E₂ and F₂ₐ, leukotrienes, adenine nucleotides, serotonin, noradrenaline and acetylcholine. It is also the major site for removal of emboli from circulation and their fibrinolysis.

c. Explain the factors that affect pulmonary vascular impedance.

Impedance is the term for resistance under particular flow conditions. Resistance in a vessel equals pressure gradient per unit flow. Under normal resting circumstances, total pulmonary blood flow is about 6 l/min and the pressure drop from mean arterial to venous pressure is about 10 mmHg, giving a resistance of 1.7 mmHg/l/min (or about 100 dyne/cm²/s), which is about a tenth of systemic vascular resistance.

Pulmonary vascular resistance falls with an increase in arterial or venous pressure as any rise in capillary pressure causes increased distension and recruitment of capillaries and less resistance to flow. Similarly an increase in lung volume lowers large and medium vessel resistance as it increases the distending tension on the larger vessels. Capillary resistance is increased at high lung volumes as stretching of the alveolar walls compresses the capillaries. Thus total pulmonary vascular resistance is high at both very low and very high lung volumes and low between.

Smooth muscle contraction in the walls of pulmonary vessels plays some role in determining pulmonary vascular pressures, but is not as significant as in the systemic circulation. Vasconstrictors such as noradrenaline, serotonin and histamine increase pulmonary artery pressure.

d. Describe the control of pulmonary vascular tone.

Pulmonary arterioles respond autonomously to the P\textsubscript{O\textsubscript{2}} in adjacent alveoli. A P\textsubscript{O\textsubscript{2}} below 70 mmHg results in marked vasoconstriction, largely independent of arterial oxygenation. There is little constriction above 100 mmHg. The mediator of this response has not been identified, but endothelial cells normally produce NO which acts as a vasodilator and which can relieve vasoconstriction when inhaled in low concentrations.

This response serves to direct blood flow into the best oxygenated parts of the lung when there are areas which are poorly ventilated. At high altitude, where there is a prolonged and generalized reduction in alveolar P\textsubscript{O\textsubscript{2}}, widespread vasospasm causes a rise in pulmonary artery pressure and may result in right heart failure and acute mountain sickness.

In the transition from foetus to newborn, this response plays a major role in allowing the changeover from 15% of cardiac output going through the hypoxic foetal lung to 90% of cardiac output passing through a much more vasodilated pulmonary circulation in the newborn after the first few breaths (with 10% shunt).

Acidosis also results in some pulmonary vasoconstriction. There is sympathetic innervation of the pulmonary arterioles with increased sympathetic outflow causing vasoconstriction.

e. Outline the mechanisms which raise pulmonary vascular resistance and describe the circulatory effects of such a rise.
Pulmonary arterial tone is raised in response to the stimuli given above. Capillary resistance rises with lung volume.

Even if total pulmonary resistance rises (for example in living at high altitude), pulmonary blood flow must still remain equal to slightly less than total systemic blood flow. In the short term this requires increased work of the right ventricle and in the long term cardiac output tends to fall. There is commonly arterio-venous shunting of blood seen within the pulmonary circulation. In persistent pulmonary vascular obstruction (as in embolism), anastomosis with the bronchial circulation increases to provide perfusion to the embolized segment.

f. Describe the pulmonary circulation in the foetus and the newborn.

The foetal circulation is substantially different from the adult, primarily because of the difference in the source of oxygenation: the foetus obtains oxygenated blood from the placenta and the newborn from the lungs. Oxygenated blood returns from the placenta in the umbilical vein which joins the portal vein and then passes through the liver into the hepatic vein or bypasses the liver in the ductus venosus and passes directly into the IVC.

Much of the blood from the IVC passes through the foramen ovale into the left atrium and then into the systemic circulation. The remainder, together with blood from the SVC, passes into the RV and then into the pulmonary trunk. In the foetus, the pulmonary circulation is of a high resistance because of the lack of oxygen in the lungs and only about a third of the RV output passes through the lungs (12% of cardiac output), the remainder being diverted through the ductus arteriosus into the arch of the aorta and the systemic circulation.

Because of the high pulmonary resistance, the pressure in the pulmonary trunk is about 5 mmHg higher than that in the aorta. The parallel operation of the right and left ventricles allows them to have substantially different outputs with the left ventricle pumping 20% more blood.

About 75% of total cardiac output ends up in the descending aorta and the majority of this flows into the umbilical arteries (over 50% of cardiac output).

The oxygen saturation of haemoglobin in the foetus is much lower than in the newborn. In the umbilical vein, the blood is about 80% saturated, falling to 62% in the LV after mixing with other venous blood. This is the saturation of the blood perfusing the head and upper body. After mixing with blood from the ductus arteriosus, saturation falls to 58% for perfusion of the remainder of the body. Foetal haemoglobin ($\alpha_2\gamma_2$) has a higher affinity for oxygen than adult haemoglobin as it binds 2,3 DPG less strongly, so in the placenta oxygen is transferred from maternal to foetal haemoglobin at the same $P_{O_2}$.

After birth, pulmonary vascular resistance falls by 90% as air enters the airways. This results in a rapid fall in right heart pressures and reversal of flow passing through the ductus arteriosus which constricts and closes over several days. The foramen ovale closes functionally as the pressure gradient between LA and RA pushes the valve shut.

In response to the trauma at delivery, the umbilical arteries constrict distal to the superior vesical arteries, and the cord is usually clamped. The cessation of flow through the umbilical vein coincides with closure of the ductus venosus which has a sphincter mechanism. The closing of the placental circulation causes a sharp rise in systemic resistance and blood pressure.

In the weeks following these changes, the muscle lining of the pulmonary vessels thins and the left ventricular wall starts to thicken to a greater extent than the right. The foramen ovale, ductus arteriosus and ductus venosus are sealed with fibrous tissue and the circulation shows the characteristics of the adult circulation.