The fetal circulation is substantially different from the adult, primarily because of the difference in the source of oxygenation: the fetus 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 fetus, 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 fetus 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. Fetal haemoglobin (α2γ2) has a higher affinity for oxygen than adult haemoglobin (P50 = 19 mmHg) as it binds 2,3 DPG less strongly, so in the placenta oxygen is transferred from maternal to fetal haemoglobin at the same P02. The Bohr and Haldane effects also operate at the placenta to facilitate transfer of oxygen from mother to fetus.

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 pressure gradient between LA and RA which pushes the foramen ovale shut. The rise in arterial oxygen tension and reversal of flow initiate constriction and closure of the ductus arteriosus (may be prostaglandin-mediated).

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 rises 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.

Hypoxia in the neonate can produce pulmonary vasoconstriction and raise right side pressures enough to cause the reopening of the ductus arteriosus and foramen ovale (“transitional circulation”). The shunting produced by this circulation tends to worsen and prolong desaturation despite administration of oxygen. It can be reversed with continued oxygen administration or by inhalation of NO.

**Neonatal heart**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR</td>
<td>120-160</td>
</tr>
<tr>
<td>Systolic</td>
<td>70-90 mmHg</td>
</tr>
<tr>
<td>MAP</td>
<td>45-65 mmHg</td>
</tr>
<tr>
<td>PAP labile (with hypoxia or hypercapnea)</td>
<td>CO 180-240 ml/min/kg</td>
</tr>
</tbody>
</table>
low compliance
dependent on preload and rate to maintain output
poor tolerance of hypovolaemia or bradycardia
equal muscularity of left and right ventricles
little sympathetic activity
bradycardia easily provoked
little change in vascular tone with spinal or caudal

Lungs
not viable before 24 weeks
inadequate surface area, inadequate surfactant, immature control
airway
jaw angle 140˚, high larynx (C3-4), long epiglottis, narrow cricoid
short trachea (4 cm), large nasal airway
first breath
results from stimulation at birth, ↑SVR from “zero” volume, requires >60 cmH₂O transpulmonary pressure
volume rises and transpulmonary pressure falls over first few breaths
control
high rate minimizes work of breathing
increased response to ↑PCO₂
unreliable response to hypoxia
transient apnoea is normal
true apnoea: >15 s, ↓PO₂, ↓HR
values
same as adult
TV 7 ml/kg, Vt 2.2 ml/kg, FRC 30 ml/kg, spf comp 0.05 /cmH₂O
different
VC 35 ml/kg (55), resistance 27 cmH₂O/l/s (1.6), RR 35 /min (14)
minute vol 125 ml/min/kg (60), O₂ uptake 6.8 ml/min/kg (3.3)
PₐO₂ 65-80 mmHg, PₐCO₂ 35 mmHg
low PₐO₂ due to CC>FRC (→ airway closure) and shunt
very compliant chest wall limits transpulmonary pressure
chest compliance 260 ml/cmH₂O
lung compliance 5 ml/cmH₂O

c. Explain temperature regulation in the neonate and how this differs from the adult.

d. Compare the physiological differences in organ function between the neonate and the adult.

Renal
immature at birth
GFR/SA 15 ml/m² at birth, 35 at 2 months, 70 at 2 years (adult)
concentrating ability 600 mOsm/l (1200 in adult)
protein synthesis is a major contributor to nitrogen metabolism
hepatic metabolism
conjugation less developed than phase 1 reactions

e. Explain the control of body fluids in the neonate and how the control and composition differ from the adult.