Question 19

A. false B. true C. false D. false E. true

Mitral stenosis is usually the result of rheumatic fever with a distorted and partly fused valve secondarily calcifying. Slow deterioration with dyspnoea, pulmonary oedema, chest pain, palpitations and haemoptysis occurs. Left atrial pressure is chronically raised and pulmonary hypertension occurs. Atrial contraction will contribute 30% of ventricular filling and if atrio-ventricular pacing is needed a long P-R interval will help filling of the ventricle. Cardiac output will usually not be helped by afterload reduction in the setting of a normal blood pressure since the obstruction is at mitral valve level. Pulmonary vascular resistance is a serious problem with right ventricular failure being a risk. If pulmonary vascular resistance increases the right ventricle may further distend and the inter-ventricular septum intrude on left ventricular function. Due to the pulmonary hypertension the pulmonary diastolic pressure will often be considerably above the pulmonary wedge pressure.

Reference: Hensley. The practice of cardiac anaesthesia. Little, Brown. Anaesthetic management for the treatment of valvular heart disease. Also Update in Anaesthesia No 14

Question 20

A. true B. false C. false D. true E. true

In aortic stenosis the normal aortic valve area decreases from $3cm^2$ to less than $1cm^2$. Without increased left ventricular systolic pressures the blood flow across the valve is dependent on the pressure gradient. With compensatory hypertrophy of the left ventricle the aortic valve gradient will increase. However later in the disease as the left ventricle dilates and further fails the left ventricular valve gradient will fall as cardiac output falls. A relatively slower heart rate is important to allow adequate time for left ventricular filling and emptying. The increased impedance to left ventricular emptying is at valve level and so changes in systemic vascular resistance will not signicantly affect left ventricular emptying. However a decrease in systemic vascular resistance may lead to critical reductions in myocardial perfusion. Episodes of myocardial ischaemia should be treated by firstly increasing systemic perfusion pressure. Vasodilators such as nitrates should be used with extreme caution if at all.

Reference: Hensley. The practice of cardiac anaesthesia. Little, Brown. Anaesthetic management for the treatment of valvular heart disease.

Also Update in Anaesthesia No 14

Answers to Self Assessment Questions

Question 1 - Answer

Prolonged late decelerations on the CTG are abnormal and signify probable fetal distress. This is progressive fetal asphyxia that if uncorrected will lead to permanent central nervous system damage or death. Fetal acidosis should be confirmed by performing a fetal scalp pH. This procedure should not, however, delay the institution of intra-uterine fetal resuscitation (IUFR) which should begin immediately. IUFR consists of specific measures aimed to increase the delivery of oxygen to the placenta and the umbilical blood flow, in order to reverse fetal hypoxia and acidosis. The mother should be examined quickly to exclude maternal hypoxia or shock or placental separation (placental abruption) which would require additional specific therapy. The following management should then be instituted immediately:

- Turn the syntocinon off.
- Turn the mother into the left lateral position and if there is no improvement try the right lateral position or the knee chest position in case cord compression is the cause.
- Administer high flow oxygen via a tight fitting Hudson mask with a reservoir bag.
- Infuse 1000mls Hartmann's solution or normal saline rapidly.
- Treat a low blood pressure if it exists. Fluids and vasopressors may be required after epidural analgesia.
- Tocolysis (stopping uterine contractions). Terbutaline 250 micrograms subcutaneously or GTN spray sublingually (2 puffs repeated up to 3 times). [not if placental abruption or antepartum haemorrhage]

If fetal acidosis is confirmed and the fetal heart rate trace does not improve with the above measures a caesarian section will be necessary.

The Physiology of Normal Oxygen Transport to the Fetus

The delivery of oxygen to the organs of the fetus requires oxygen delivery to the maternal side of the placenta (intervillous spaces), placental transfer of oxygen to the fetal blood in the chorionic villi by passive diffusion and an intact fetal circulation.

Oxygen delivery to the placenta. Placental blood flow is determined by the perfusion pressure (arterial pressure - venous pressure) and the resistance to blood flow. Oxygen delivery is defined as placental blood flow multiplied by the arterial oxygen content (haemoglobin concentration multiplied by the arterial oxygen saturation). Branches of the uterine arteries supply the intervillous spaces and the blood returns to the maternal circulation via the uterine veins. The branches of the uterine arteries are maximally dilated during late pregnancy and therefore placental oxygen delivery is close to maximum at this time provided that the mother has a normal haemoglobin concentration, normal oxygen saturations and a normal perfusion pressure.

Placental transfer of oxygen. In the placenta, chorionic villi project into the large 'lakes' of maternal blood in the intervillous spaces and contain fetal capillaries. These chorionic villi are perfused by the umbilical arteries and the blood returns to the fetal circulation via the umbilical vein. The placental transfer of oxygen is a passive process from maternal blood, with a relatively

high PO_2, to the fetal capillaries with a low PO_2. Fetal umbilical venous PO_2 is relatively low (35mmHg) compared to maternal arterial PO_2 (100mmHg when breathing air). This is thought to be due to the structural characteristics of the placenta (it functions as a concurrent exchange system rather than a countercurrent exchange system), poor matching of fetal and maternal blood flow in certain areas of the placenta (analogous to shunt and ventilation/perfusion mismatch in the lung) and because of the high oxygen consumption of the placenta itself.

Fetal circulation. An adequate fetal oxygen delivery is still possible despite the low umbilical venous PO_2 because of a number of factors. The haemoglobin concentration is high (17-19g/dl), the cardiac index high (350mls/m^2/min) and the haemoglobin dissociation curve is shifted to the left compared to the adult because of the presence of haemoglobin F. This means that despite the low PO_2 in the umbilical vein the haemoglobin is 75-80% saturated. The fetal circulation is also designed such that the best oxygenated blood from the umbilical vein is directed via the ductus venosus to the inferior vena cava and via the foramen ovale to the left side of the heart and then to the head and neck of the fetus. The less well oxygenated blood from the superior vena cava enters the right ventricle and then enters the aorta via the ductus arteriosus distal to the left subclavian artery. The less well oxygenated blood is therefore diverted to the trunk and lower body of the fetus.

Effect of uterine contractions on oxygen transport. Active contractions during labour generate intra-uterine pressures of 45-50mmHg which compress the uterine veins and decrease arterial blood flow. This causes a reduction in the PO_2 of the blood in the intervillous spaces and the fetal oxygen saturations decline about 7% to a low point about 90-120sec after the peak of the contraction. Recovery occurs over a similar period of time. When oxygen delivery is borderline contractions may cause a marked fall in fetal oxygenation and fetal bradycardia. This is seen as a late deceleration on the CTG. When oxygen delivery is severely impaired, oxygenation fails to recover between contractions and a sustained bradycardia results.

Pathological Conditions Causing an Inadequate Oxygen Delivery to the Fetus

- **Maternal hypoxia**
- **Maternal hypovolaemia / hypotension**
- **Aortocaval compression**. The pregnant uterus may compress the inferior vena cava and aorta within the abdomen. This is usually worst when the mother is lying on her back but can occur in other positions to. Caval compression decreases venous return and cardiac output and may result in maternal symptoms of hypotension. Isolated aortic compression does not produce maternal symptoms but will also result in a decrease in fetal oxygen delivery.
- **Uterine hyperstimulation**. This is defined as a contraction frequency of more than one in every 2 minutes and does not allow recovery of fetal oxygenation between contractions. As already explained a normal contraction frequency can cause distress to a fetus without physiological reserve.
- **Placental separation/abruption**
- **Pre -eclampsia**
- **Umbilical cord compression**. This is most obvious when the cord prolapses into the vagina but it may also be compressed in the uterus. If compression is severe enough to cause fetal hypoxia the bradycardia follows contractions but the timing is not constant (variable decelerations).
- **Fetal haemorrhage**
- **Regional analgesia**. Sympathetic blockade will worsen any tendency to supine hypotension. Some of the changes in fetal heart rate pattern seen after regional analgesia may be due to an increase in uterine activity.

*[Ref: Thurlow JA and Kinsella SM. Intrauterine resuscitation: active management of fetal distress. International journal of Obstetric Anaesthesia (2002)**11**,105-116]*

Question 2 - Answer

Left lower lobe collapse (left diaphragm not seen and double shadow along left heart border). Initial treatment would include humidified oxygen and physiotherapy to re-expand the lung. If this was not successful a bronchoscopy could be performed. Antibiotics should be prescribed.

Question 3 - Answer

1) Chronic subdural haematoma
1) Acute extradural haematoma
2) Subarachnoid haemmorhage