The Birth process imposes a significant and unique physical challenge to the neonate due to the necessity for adaptation to extra-uterine life and accomplishment of key life tasks necessary for survival. Such life tasks comprise independent breathing, the transition from fetal to neonatal circulation and the metabolic adaptation of thermoregulatory and glucose homeostasis. This unit summarises the main postnatal adaptive changes that occur at birth.

The fetal circulation

The figure below shows the fetal circulation with its temporary structures.


In utero the placenta acts as the organ of respiration for the fetus and is responsible for the oxygenation of blood and the elimination of waste products. The umbilical vein carries oxygenated blood from the placenta to the fetus.
Pulmonary vascular resistance and pressure in the pulmonary arteries is high encouraging blood to take the path of least resistance and cross through the foramen ovale and ductus arteriosus in a right-to-left direction across a low resistance ductus arteriosus into the descending aorta and then to the lower body including the kidneys, mesentery and placenta via the umbilical arteries. Only 10-12% of pulmonary artery flow passes to the lungs. Further detail on Fetal circulation can be seen in the Key Reading below.

**Post natal circulatory changes**

At birth the circulation alters in response to changes which take place in the lungs which become the primary organs of respiration. Birth and lung expansion results in an 8 to 10 fold increase in pulmonary blood flow (Blackburn, 2003). The pulmonary vessels respond to the increase in the oxygen content of the blood by vasodilating. This reduces pulmonary vascular resistance and encourages blood to flow to the lungs. Blood then returns to the left side of the heart through the pulmonary veins. This increases the pressure in the left side of the heart and closes the foramen ovale, facilitated by the lowering of right sided pressures. Reduction in pulmonary vascular resistance is primarily due therefore to lung aeration, increasing PaO2 and increased pulmonary vasodilators such as nitric oxide/endothelial-derived releasing factors (Blackburn, 2006). This process is also mediated by a surge in catecholamine release at birth (Khong, 2004).

Because of the lowered pulmonary vascular resistance, blood will travel to the lungs through the pulmonary arteries. The increased oxygen content of the blood encourages the ductus arteriosus to close and so the pattern of neonatal circulation is established.

The temporary structures will undergo changes during the first few weeks of life and will become obsolete. The foramen ovale becomes sealed with fibrin deposits during the first month of life. During this period some blood may continue to leak through the foramen ovale causing a murmur which disappears once the foramen ovale is finally sealed. The ductus arteriosus closes because of the increase in oxygen content in the blood and the drop in circulating prostaglandin levels (PGE2). This eventually becomes a ligament. The ductus venosus closes soon after birth because blood no longer passes through it. It becomes the ligamentum venosum.
**Thermal regulation in the fetus:** The fetus uses the placenta as a heat exchanger to maintain a stable intrauterine temperature. This is usually approximately 0.5 degrees celsius above maternal body temperature (Sherman et al, 2006) so that the normal fetal temperature ranges at 37.6°C to 37.8°C. The main factors influencing fetal temperature include the fetal metabolic rate, the heat exchanging capacity of the placenta and rate of blood flow in the intervillus spaces.

**Thermal regulation in the newborn baby:** Once the baby is born, they have to achieve thermal stability independently. In a healthy adult, thermoregulation takes place efficiently. Humans rely on a complex sensory system to detect changes in environmental temperature and respond appropriately to them. Temperature sensors are distributed widely throughout the body and transmit information to the hypothalamus and the cerebral cortex. This then stimulates an autonomic and a behavioural response keeping the body core temperature relatively stable.

However, in a newborn baby, there are a number of problems which must be overcome in order to achieve such thermal stability. At birth, the intrauterine heat reservoir and heat exchange through the placenta is lost. Therefore, they must adapt to this new environment by a process known as non-shivering thermogenesis. This involves lipolysis of brown fat deposits or adipose tissue. When the temperature drops, a catecholamine surge (norepinephrine) stimulates the mitochondria in the brown fat to produce heat utilises many enzymes consuming oxygen and glucose. The final stage of this oxidative process involves the protein Thermogenin and results in marked production of heat. In healthy newborns, thermoregulation will not pose a problem if they are kept warm and dry straight from delivery by a combination of simple but effective measures such as drying, immediate skin-to-skin contact, wrapping and maintaining an optimum room temperature. However, if a newborn is allowed to become cold then the brown fat stores will deplete and the newborn will become hypoxic and hypoglycaemic (See Unit 3C).
Glucose homeostasis in the fetus; In fetal life glucose provides almost half of the total energy requirements. Glucose diffuses across the placenta. The rate of diffusion depends on the concentration of glucose in the maternal circulation. If this is high then glucose diffuses across more rapidly. Insulin is produced by the fetus as it cannot cross the placenta. This tends to happen late in pregnancy. Production of insulin increases in response to higher levels of fetal blood glucose. The effect of this is to stimulate the uptake of glucose into muscle and adipose tissue. This contributes to the rapid fetal growth that takes place in the last trimester of pregnancy.

Blood glucose homeostasis and changes at birth; At birth, the baby has to switch from a state of net glucose uptake (from the mother via the placenta) to independent glucose metabolism. Birth is a period of stress for the baby. This results in mobilisation of glycogen stores - part of this is due to the increased levels of catecholamines in the neonatal circulation. As a result, the baby’s blood glucose levels fall. Plasma glucagon levels rise in response to the lowered blood glucose and this triggers the process of gluconeogenesis. Provided a normal term baby is kept warm and fed within the first few hours of birth, they should be able to control their own blood glucose concentrations. This is due to their ability to mount protective responses to falls in blood sugar and increased availability of alternative substrate or fuel for the brain if glucose is low. The most abundant alternative fuel in early neonatal life are ketone bodies which are produced by fatty acid oxidation (Hawdon, 2005). The newborn therefore mobilises glucose to meet energy requirements by secreting glucagon and catecholamines while insulin is suppressed which causes the blood glucose levels to rise at approximately three to four hours of age (Aylott, 2006).

Fluid Balance changes at birth In fetal life, there is interplay between the urine, amniotic fluid and lung fluid production to maintain adequate fluid balance and lung tissue growth. Urine production begins in the kidney and continually circulates into the amniotic fluid from 9-12 weeks gestation. Amniotic fluid originating from maternal blood is also continually swallowed and absorbed via the digestive tract. This passes as waste products which cross the placenta to be discarded with any excess water excreted by the fetal kidneys in to the amniotic fluid. Fluid balance undergoes significant postnatal adaptive changes at birth. All newborns undergo extra-cellular fluid contraction after delivery which shows as a diuresis following a natriuresis.

Conclusion; Thermal and glucose homeostasis together with the ability to breathe normally without assistance are critical physiological functions that are closely interrelated (Aylott, 2006). A change or difficulty in one of these variables affects the other. The features of the compromised neonate in early life and the interplay between these functions will be covered in Unit 3C. Good neonatal care can reduce the likelihood of complications and facilitate successful adaptation to extrauterine life.
KEY READING

http://www.embryology.ch/anglais/pcardio/umstellung01.html#fetalkreislauf

http://www.frca.co.uk/Documents/Foetal%20circulation.pdf

http://en.wikipedia.org/wiki/Adaptation_to_extrauterine_life

http://www.frca.co.uk/Documents/Foetal%20circulation.pdf


http://www.infantgrapevine.co.uk/journal_article.html?RecordNumber=5647&number=15


Aylott, M. (2006b) The Neonatal energy triangle part 2; Thermoregulatory and respiratory adaptation Paediatric Nursing. 18(7):38-43


Blackburn ST (2003) Maternal, Fetal and Neonatal Physiology; A Clinical perspective Philadelphia; Saunders


Hawdon JM (2005) Blood Glucose levels in infancy- clinical significance and accurate measurement Infant, 2, 2, 24-27


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