Reproduction – parturition, neonatal physiology, lactation

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Parturition – control

- *Parturition requires myometrial contractions and cervical softening.*
- The **myometrium** is a functional syncytium (coupled by gap junctions) so contractions are coordinated. Contractions occur because action potentials cause Ca^{2+} influx; oestrogen-primed myometrium shows *pacemaker potentials*, which initiate APs if they are large enough. **Prostaglandins** liberate Ca^{2+} from intracellular binding sites. **Oxytocin** increases the rate of Ca^{2+} influx and lowers the excitation threshold of the myocyte.
- **Cervical softening.** The cervix has a high connective tissue content – collagen bundles in a proteoglycan matrix. Cervical softening occurs because there is a *loss of collagen* and an *increase in glycosoaminoglycans (GAGs)*, specifically keratan sulphate. (This does not bind collagen well; dermatan sulphate does, so the increased keratan:dermatan sulphate ratio may explain the "loosening" of the collagen.) **Prostaglandins** (PGE_2; $PGF_{2\alpha}$) increase cervical compliance.
- The endometrium is probably the most important site of prostaglandin (PG) synthesis. Remember that PGs are local hormones. The enzyme PLA_2 is critical in PG synthesis. The **oestrogen:progesterone ratio** controls both PG synthesis (high O:P → high PG synthesis) and also PG release: oestradiol → ↑oxytocin receptors in the endometrium → more PG release.
- Oxytocin is a peptide synthesized in hypothalamic neurons and transported along their axons to the posterior pituitary. It is released in response to tactile stimulation of the reproductive tract, esp. the cervix, and increases myometrial contractility (the Ferguson reflex). This reflex is facilitated by a high O:P ratio.
- **Relaxin** is a polypeptide hormone made by the corpus luteum and secreted throughout most of pregnancy. It causes relaxation of the interpubic ligament, so the pubic symphysis can separate to some extent. (This can cause considerable discomfort during pregnancy.) Its precise action in the human is not certain, but in addition to ligamentous relaxation it may also inhibit myometrial activity, inhibit oxytocin secretion and facilitate cervical softening. It therefore helps to maintain pregnancy but gets things ready for parturition.

Parturition – timing

- Human pregnancy normally lasts **40 weeks** *from the date of the last menstrual period.*[1] Forty weeks is "full term". Labour is considered premature if it occurs before 37 weeks' completed gestation; after 42 weeks pregnancy is considered prolonged. (Prematurity is not the same as low birth weight, though obviously premature babies tend to be small.)
- **In principal, both mother and fetus are capable of affecting timing.** A mare carrying a foal has a 340-day pregnancy; a mare carrying a mule has a 355-day pregnancy ⇒ fetal influence. But there is certainly maternal fine tuning: women deliver mostly at night; alpacas can postpone birth for up to 2 days in bad weather; rats deliver by day even if the day/night cycle has been shifted during pregnancy.
- **Goats:** pregnancy depends on corpus luteum for steroids. Fetal ACTH → fetal adrenal cortex → glucocorticoids → induce placental aromatizing enzymes → increased oestrogen synthesis from DHA → enhance placental production of $PGF_{2\alpha}$ → luteolysis → fall in progesterone → 'block' on myometrium removed → additional $PGF_{2\alpha}$ → contractions. In addition, the rising O:P ratio facilitates oxytocin release and the Ferguson reflex; since oxytocin stimulates PG release, a positive feedback system is established. There are additional mechanisms as well.
- **Sheep:** pregnancy depends on the placenta for steroids. As in the goat, the *fetal* HPA axis has a dominant role in timing: fetal hypophysectomy/adrenalectomy prolongs pregnancy (indefinitely) while injection of ACTH or corticosteroids into the fetus induce pregnancy. Fetal ACTH → fetal cortisol → placental enzyme activity changes such that placental progesterone is diverted into oestrogen synthesis → increased O:P ratio in the maternal circulation → $PGF_{2\alpha}$ made in the decidua and fetal membranes → contractions → oxytocin, etc.
- So in both the goat and the sheep, the fetus times the onset of parturition through the ultimate mechanism of $PGF_{2\alpha}$. How about the human?
- **Human: unclear.** Simple anencephaly (= no head ⇒ no CRH) does not necessarily prolong pregnancy; nor does fetal adrenal hypoplasia; infusion of ACTH or synthetic glucocorticoids does not induce parturition. (Nevertheless, cortisol does rise in late pregnancy.) It seems that the onset of labour is not critically dependent on fetal adrenal activity – and there are no clear and consistent changes in placental oestrogen synthesis at term. The O:P ratio does rise, but the change is neither abrupt nor universal. Might local steroid receptor changes be having the same effect? Unproven. $PGF_{2\alpha}$ levels do rise in amniotic fluid before labour, and rise throughout parturition. So the changes in cortisol and $PGF_{2\alpha}$ are the same as in sheep and goats, but the control mechanisms are unknown.

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[1] Dates always refer to the LMP, which is a useful clinical convention, but note that this means the true gestation (from ovulation) is 38 weeks.

Parturition – mechanism

I shall describe briefly the course of a human normal delivery. You don't need to know these details now, but you will have a quota of babies (5) to deliver in a couple of years' time…

- From ~36 weeks, the uterus occasionally contracts, weakly – **Braxton-Hicks contractions.** They can be felt – the uterus hardens. The head descends into the pelvis late in pregnancy, and "engages" there.
- **Cervical ripening** consists of softening, effacement (its length is obliterated) and dilatation.
- **Labour** begins when (a) uterine contractions are regular *and* (b) there is progressive dilatation of the cervix. Labour is divided into three stages.
- **First stage of labour.** From the onset of labour to full dilatation of the cervix (10 cm). Mean duration ~8h (primagravidae[2]) or ~5h (multiparous woman). The myometrium contracts, generating intrauterine pressures of 50–75 mmHg. The wave of excitation spreads down from the fundus of the uterus. As the muscle contracts, it also retracts – it does not relax back to its original length (*brachystasis*). The lower uterine segment is far less muscular than the rest; no point squeezing at the bottom.
- Pain relief in labour – nitrous oxide (tricky to use correctly because time to action is slow – need to inhale before it hurts), TENS (transcutaneous electrical nerve stimulation; doesn't work well for serious pain), intramuscular pethidine (mild opiate: if the baby gets some, it will come out doped up and with respiratory suppression, and will need naloxone before it starts to breathe), epidural anaesthesia (excellent but tricky to put in during contractions, involving as it does inserting a large needle temporarily into the epidural space; a plastic cannula is then left behind to infuse local anaesthetic around the nerve roots). Women who have pethidine and then an epidural have a tendency to fall asleep! Why?
- Monitoring in labour – cardiotocography is routine (monitors uterine contractions and fetal heart rate). More invasive monitoring, such as fetal scalp blood sampling, possible but avoided.
- The fetus is metabolically **vulnerable** during parturition: its own systems are not yet functioning, and maternal support is dwindling. Prolonged labour can result in fetal distress.
- **Second stage.** From full cervical dilatation to delivery of the fetus. Average duration 40 min (primagravidae) or 20 min (multiparae). The uterus continues to contract and force the fetus through the cervix. The head has been *flexing* continuously throughout labour. It *rotates internally* (usually from facing sideways to facing backwards). The head then *extends* as it passes through the perineum, and *rotates externally* to face sideways again. The shoulders appear (anterior, then posterior) and finally, the trunk is delivered by *lateral flexion*.
- At this point, the umbilical cord would be clamped[3] and cut, the baby slapped around a bit until it turns pink (operating on lungs now, so O_2 tension rises to normal levels) and makes some noise, dried, weighed, tagged, injected with vitamin K, etc.
- **Third stage.** From delivery of the baby to delivery of the placenta. About 15 min.
- The **puerperium** is the 6 weeks following birth (hence *puerperal fever*, now rare).

Parturition – pathology

A good deal can go wrong, and I won't cover it except to list the top causes of maternal mortality – hypertensive diseases (18%), pulmonary embolism (18%), anaesthesia (14%), amniotic fluid embolism (10%), abortion (8%), ectopic pregnancy (7%) and haemorrhage (7%). In the 1980s the absolute death rate was 8.6 per 100,000.

Perinatal mortality (stillbirths plus first-week neonatal deaths) are mainly due to congenital abnormalities (20%), low birth weight and asphyxia. Absolute rate is about 10 per 1000 births.

The Caesarean section rate in the UK is 5–13% of all labours[4] and the maternal mortality is 0.33/1000.

Neonatal physiology

You should cover fetal and neonatal physiology in some detail. This is a summary in essay form.

Sample essay; I wrote this in 1994.

"Discuss the changes that occur before and soon after birth to ensure survival of the neonate."

Introduction

The mammalian fetus is maintained in a tightly controlled and protected environment in the uterus. It receives oxygen and nutrients and disposes of its wastes through the placenta, so its own homeostatic systems are perturbed little. When it is born, however, it must be self-sufficient in many respects. The process of birth is the most dramatic time when many changes take place at once to ensure this, but many more subtle changes have been occurring for months and will continue to occur after birth.

Respiratory changes

For the lungs to function as a respiratory exchange surface, air spaces must develop. This begins to occur at 20 weeks of gestation, with the first alveoli at 22–23 weeks. They increase in number until one year after birth. Pulmonary blood vessels develop; they have thick walls *in utero* and remain constricted. Type II pulmonary epithelial cells produce surfactant, which reduces the work of breathing and prevents smaller air sacs collapsing into

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[2] Parity = # of pregnancies beyond 28 weeks; gravidity = total # of pregnancies (to any stage). The definition may have changed in line with the legal definition of fetal viability (= age of abortion) – now set at 24 weeks.
[3] It is possible to delay cord clamping and raise the placenta for 30 sec in premature babies, thus 'autotransfusing' them, raising their haematocrit and reducing oxygen therapy requirements.
[4] *Oxford Handbook of Clinical Specialties*, 1995

larger ones by keeping the surface tension low. Respiratory movements are present from 11 weeks of gestation: this is crucial in stimulating fibroblast and connective tissue damage by providing mechanical stress. Glucocorticoids and T_4 are also essential for structural and biochemical maturation.

During labour, the fluid present in the lungs starts to be absorbed, and this process continues after birth with the fluid leaving through pulmonary circulation and lymphatics. Further fluid production is inhibited as the elevated catecholamine concentration during labour reverses the action of chloride pumps in the pulmonary epithelium via a β-adrenergic effect (before birth, the pump pumps chloride into the lung spaces which takes water with it).

The first breath is the most visible sign of the neonate's health. The precise stimuli are known imprecisely, but it is known that cold stimulates breathing, as does release from facial immersion (the reverse of the diving reflex) and generalised arousal via the reticular activating system. The central chemoreceptors certainly play a part in regulating respiration after this, but do not cause the first breath as they cannot respond quickly enough; the peripheral chemoreceptors do *not* play a part (and neither do specific sensory inputs).

Circulatory changes

The respiratory changes described at birth lead to decreased pulmonary resistance. Mechanical effects include the lung expansion and filling with air; the increased pO_2 that occurs with air breathing causes pulmonary vasodilatation; and pulmonary ABP falls. Therefore the pulmonary blood flow increases from 35 ml/kg/min to 150–200 ml/kg/min.

Systemic vascular resistance increases markedly, because the low resistance "shunt" that is the placenta is lost. The umbilical vessels contract to minimize blood loss.

Now that the lungs are operating, the right-to-left blood shunt becomes undesirable and the foramen ovale closes. This is a purely mechanical effect: the opening of the pulmonary vasculature and cessation of umbilical flow reduce right atrial pressure while the sudden increase in pulmonary venous return increases left atrial pressure. This reverses the pressure gradient across the foramen ovale and the flap-like septum primum is pushed against the rigid septum secundum, closing the foramen ovale. Normally the septa fuse by 3 months after birth, but the foramen may remain technically patent throughout life without observable effects as long as it stays shut – this is the case in 10% of adults.

When the pulmonary circulation opens, the drop in pressure in the pulmonary trunk is thought to cause a slight reverse flow of oxygenated aortic blood through the ductus arteriosus. This has thick spiral muscle in its wall, and a metabolically active subintimal layer; the increased local oxygen tension induces it to close. This occurs within 10–15 hours of birth in infants born at term. The mechanism is uncertain: certainly the contraction involves Ca^{2+} (but that is hardly surprising!). It is possible that locally synthesized prostaglandins PGE_2 and PGI_2 maintain patency of the ductus arteriosus, and that prostaglandin levels are reduced by (a) increased lung blood flow, leading to increased prostaglandin clearance, and/or (b) inhibition of prostaglandin synthesis by oxygen.

In addition to the shift in the site of erythropoiesis (from yolk sac to liver, spleen and finally bone marrow) that is almost complete at term, the fetus alters its globin synthesis pattern around the time of birth. 50% of globin synthesis is α-globin throughout, but from 30 weeks of gestation to 12 weeks after birth the other 50% shifts from being primarily γ-globin to being primarily β-globin. Therefore the neonate switches from making HbF to HbA, which has a somewhat lower oxygen affinity. In fact, HbF is a relatively recent evolutionary adaptation, found only in primates. The switch occurs on a cell-by-cell basis in the bone marrow.

The fetal concentration of 2,3-DPG is also increased to the adult level, and the ductus venosus closes.

Renal changes

In the first 24–48 hours, the neonate shows a net loss of salt and water as it lowers its blood volume to a normal level. Glucocorticoid production leads to increased GFR and decreased urinary pH as acid excretion increases. Both GFR and tubular function increase over the first 2–3 years after birth.

Water conservation is a serious problem for babies. Their kidneys do not perform at adult levels; they are dependent on others to provide them with fluids; they have a high surface area/volume ratio; they breath shallowly and fast (much like a dog panting to lose heat by evaporation); food passes rapidly through the intestine, producing moist faeces; and their intracellular fluid volume is small so circulatory collapse is easily precipitated. Neonates have difficulty with both salt and water load. Furthermore, they need urea to concentrate the urine effectively, so malnutrition compounds water shortage. Milk serves as a rehydration therapy as well as a food; it is isotonic with sugar, and so there is an excess of water once the sugar has been metabolised.

Hepatic changes

Fetal glucocorticoids and growth hormone/placental lactogen cause deposition of glycogen reserves in the liver: these are essential to see the fetus through delivery and until it is first fed. The release of glycogen is triggered by higher levels of glucocorticoids which induce higher levels of enzymes for glycogenolysis and debranching, and β-adrenoceptors (all are low *in utero* to promote glycogen storage). In addition to this, catecholamine levels, glucagon and hepatic nerve activity increase at birth. Glucocorticoids also induce enzymes for gluconeogenesis and a hormone-sensitive lipase.

In utero, the fetus does not conjugate bilirubin in order that it may cross the placenta and be excreted in maternal bile; after birth, it must conjugate it for its own excretion. The increase in bilirubin level after delivery induces enzymes for its own conjugation. Many neonates show an increased rate of erythrocyte breakdown after birth, where blood volume is too high (this frequently occurs with late clamping of the umbilical cord). The iron is stored and lasts until the second half of the first year of life, but the bilirubin produced may lead to "physiological" hyperbilirubinaemia, visible as jaundice.

Thermal changes

Neonates are very vulnerable to variation in ambient temperature. Their thermoneutral zone (the temperature at which they can thermoregulate when naked purely by vascular changes) is between 32°C and 36°C (compare that with an adult, about 22–26°C).

They are vulnerable to heat loss because they have a high surface area/volume ratio; they are wet at birth, so lose heat by evaporation; they have little voluntary muscle activity and cannot shiver. To compensate for this, infants have a great deal of brown adipose tissue or BAT: between the scapulae, around blood vessels in the neck, in the axillae, in the mediastinum and around the kidneys and adrenals. It makes up 2–6% of body weight and is activated by noradrenaline. It generates heat either by uncoupling respiration from oxidative phosphorylation in mitochondria, or by a high level of sodium pumping and leakage; which is not clear. The neonate's BMR doubles in the first 10 days after birth, and BAT is responsible for much of this.

Infants are even more vulnerable to overheating. They have poor sweating mechanisms, and it takes a higher temperature to elicit sweating. They are susceptible to dehydration (see above), and the high surface area/volume ratio means that they gain heat rapidly with a high ambient temperature.

Immune system

The neonate has an immune system that is "competent but ignorant". There is some prenatal transfer of passive immunity from the mother: IgG is able to cross the placenta. After birth, there is no further transfer of immunity [1], but the mother is able to shield the neonate from pathogens by substances in her milk. This prevents the baby's immune system from being overwhelmed by pathogens. There is IgA in breast milk that is not absorbed by the baby's gut, but that forms a "coat of antigenic paint" that prevents pathogens from breaching the gut wall. This is the most effective form of antigenic protection in the adult, and no doubt it serves the neonate well. Also in milk is lysozyme, which will degrade some pathogens; lacto-

ferrin, which will sequester iron and thus inhibit bacterial growth (recall the neonate has iron stores so does not suffer as a result of this); bifidus factor (a nitrogenous carbohydrate that promotes growth of the organism *Bifidus* and prevents *Escherichia coli* colonization) and a variety of cells, including macrophages, that are able to survive for a time in the baby's gut.

Conclusion

Obviously the system works: by and large, new-born babies live. However, this is not to say that they are not extremely vulnerable to environmental stresses. Humans give birth to young that are highly immature by the standards of all other animals, and as their babies are so dependent, must provide a long period of parental care. It is perhaps the most extreme example of this reproductive strategy on the planet.

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[1] This may be wrong. Wraith (1994) states that IgG is specifically taken up from the gut into blood.

[~1,600 words]

- **Interesting facts: respiratory distress syndrome (RDS) in premature babies.** Lacking surfactant, the work of breathing is greatly increased and the alveoli collapse, leading to hypoxaemia. (Obviously dangerous of itself, hypoxia also predisposes to intra-cerebroventricular haemorrhage.) The development of artificial surfactant, simply squirted down an endotracheal tubes, together with advances in techniques of artificial ventilatory techniques, have improved survival dramatically.
- One method in the pipeline is the use of *liquid respiration:* the lungs are partially filled with a fluorocarbon that dissolves O_2 avidly. Mice can breathe like this (was shown for real in the film *The Abyss*) but diffusion distances limit gas exchange. Therefore the new method is to fill only the alveoli. Air is in the bronchi, so exchange distances are minimized. Why is this potentially useful? Because alveoli collapse at the point of *maximum surface tension* – the reason we need surfactant – and this is at the air–liquid boundary. By filling the alveoli with liquid, the boundary is moved into the bronchioles, which have cartilaginous support and do not collapse so easily. A few clinical trials have taken place (babies otherwise sure to die) and have sometimes worked; it remains to be seen if it catches on. The long-term effects of the fluorocarbon may be harmful. Watch this space…

Lactation

Breast anatomy

- The mammary gland consists of many **lobes,** 15–20 in the human; each lobe consists of **lobules** interconnected by areolar tissue, blood vessels and ducts. Lobules are ovoid.
- In each lobule, clusters of **alveoli (acini)** open into **milk-collecting ducts,** which unite to form a larger **lactiferous duct** that drains a lobe. These ducts converge towards the **areola,** where they form dilatations called **lactiferous sinuses** that act as small milk reservoirs, before opening separately onto the **nipple** (papilla).
- The glandular lobes (the parenchyma) are connected by fibrous tissue and surrounded by loose areolar and adipose tissue.[5]
- Each alveolus consists of a single layer of epithelial cells that synthesize milk and secrete it into the alveolar lumen. There are **myoepithelial cells** between the basement membrane and the epithelial cells; these can contract to move milk from the alveoli to the ducts prior to ejection.
- Also opening onto the peripheral areola are small ducts from the sebaceous *Montgomery glands*, which probably have a lubricating function during suckling.
- In all species, nipples are found along the two *milk lines* (dictating the site of supernumerary nipples in humans!).

Development

- At birth, the breast consists of ducts but few alveoli. At puberty, under the influence of oestrogens, the lactiferous ducts sprout and branch to develop alveoli. As the menstrual cycle becomes established, oestrogen/progesterone cycles induce more (though limited) ductal–lobular–alveolar growth, and the breasts increase in size due to fat deposition and connective tissue growth. There can be considerable cyclical changes in breast volume.[6]
- During early pregnancy, oestradiol, progesterone (and insulin? prolactin?) induce hypertrophy of the ductular–lobular–alveolar system. Alveolar cells differentiate and by mid-pregnancy they contain considerable amounts of secretory material.

Milk

- **Milk fat** is synthesized in the ER of alveolar epithelial cells and forms membrane-bound lipid droplets. These reach the surface of the cell, bulge out and 'pinch off' some cytoplasm to be released, still enclosed in membrane, into the alveolar lumen. **Milk protein** is synthesized in vacuoles and exocytosed.
- **Prolactin** promotes these processes.

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[5] Breast malignancies nearly always arise in ductular or glandular epithelium, rather than stroma.
[6] This situation – of considerable breast development in the non-pregnant female – contrasts with other mammals, including primates, in whom breast development is only marked near the end of pregnancy.

- Milk contains a wide range of nutrients (water, electrolytes, carbohydrate, fat, amino acids and proteins, vitamins).
- The predominant carbohydrate is **lactose.** This promotes intestinal *Lactobacillus* growth and provides galactose for neural myelination. Lactose is formed (UDP-galactose + glucose → lactose) in the Golgi apparatus by lactose synthetase, which is a complex of *galactosyltransferase* ("protein A") and *α-lactalbumin* ("protein B"; whey protein).
- Milk proteins include casein, lactalbumin and lactoglobulin, plus immunoglobulins (IgA).
- In the first week following birth, the milk is yellowish and sticky – **colostrum,** or "witches' milk" – which is replaced by **mature milk** after 2–3 weeks. Colostrum contains less water-soluble vitamins, fat and lactose, but more protein (including IgG), minerals and fat-soluble vitamins (A,D,E,K). The caloric value of mature is higher.

Lactogenesis: the initiation of milk secretion

- As mentioned, the breasts are capable of milk secretion by 4 months of pregnancy, but they are not fully activated until after parturition. Prolactin and placental lactogen increase during pregnancy, but the breast is not responsive to them until **oestrogen and progesterone disappear,** which occurs with delivery of the placenta. After parturition, placental lactogen levels fall sharply, while prolactin levels fall more slowly – **prolactin** is maintained for the duration of lactation.
- There is an antagonistic relationship between progesterone and prolactin: for example, prolactin stimulates α-lactalbumin synthesis and progesterone inhibits it.
- Milk secretion will occur for 3–4 weeks after parturition in the absence of suckling, but is not copious or maintained beyond this unless suckling occurs.

Galactopoiesis: maintenance of milk secretion

- Suckling (nipple stimulation) induces the release of **prolactin** from the anterior pituitary. This is a neuroendocrine reflex. There is a therefore a burst of prolactin during suckling, determined by the strength and duration of suckling; in addition, the basal levels of prolactin are maintained by suckling. The amount of milk secreted is therefore controlled by the frequency/duration/intensity of suckling. When the baby suckles, it therefore causes secretion of milk for its *next* meal.

Milk removal and the milk ejection reflex (MER)

- Nipple stimulation also causes **oxytocin** synthesis and release from the posterior pituitary. This of benefit in the *current* meal: oxytocin causes contraction of alveolar myoepithelial cells, thereby inducing expulsion of milk into the ducts (milk 'let-down'). This causes an increase in intramammary pressure, and can cause milk to spurt from the nipple. The MER can also be conditioned (e.g. to a baby's cry), while the prolactin release cannot and requires nipple stimulation.
- Since cervical/vaginal stimulation also causes oxytocin release (see Parturition, above), milk ejection can occur during coitus in lactating women. Apparently farmers blow air into cow's vaginae for this reason!
- The MER is quite vulnerable to inhibition by stress. Mechanism unclear.
- Babies suck (airtight seal, negative pressure) but they also compress the nipple to remove the milk already in it (allowing more milk to enter between 'sucks') which is simpler.

Other points

- After lactation ceases, it takes ~3 months for the mammary gland to involute. This is largely due to mechanical factors: the accumulation of milk damages the alveoli and compresses capillaries to cause alveolar hypoxia. Desquamated epithelium is phagocytosed, so the lobular/acinar structures regress and the ductular system again dominates.
- Lactation can be suppressed clinically by **bromocriptine,** a dopamine agonist (recall that dopamine suppresses prolactin synthesis). Why might this be desirable?
- Menstrual cycles and fertility returns more slowly in lactating than non-lactating women following birth (probably because prolactin suppresses the initiation of cyclic gonadotrophin release – may be due to decreased pituitary sensitivity to GnRH). Normal reproductive function returns by 3–6 months, but *menstruation is a poor indicator of fertility and lactation is an unreliable method of contraception.*

Parental behaviour

- Terms to know: *altricial* young are born naked, blind and highly dependent on their parents (e.g. mice, rabbits). *Semi-altricial* young have hair and sight at birth but poor motor skills (e.g. carnivores, primates). *Precocious* young can, to some extent, fend for themselves (e.g. cows, dolphins).
- Maternal behaviour may be divided into *preparation* during gestation (e.g. nest-building, buying cots), *care and protection* of the young and *lactation* after birth, and behaviour associated with progressive independence (associated with *weaning* but lasting much longer in the human).
- This area is complex and varies considerably with species.

- Hormones are not required for the display of maternal behaviour in **rats** – the presentation of pups can be sufficient – but they contribute, because post-partum females respond much quicker than other females.
- The factors determining acceptance of particular young have been examined in detail in **mice and sheep,** including the way the smell of an individual pup is 'stamped in'. Vaginal stimulation of oestrogen-primed sheep (as occurs during birth) leads to acceptance of a lamb and maternal behaviour for a period of ~2 hours, by causing changes in the olfactory bulb. This is useful for farmers (oestrogen + vibrator → ewes accept orphan lambs).
- Is **human** mother–infant bonding different following vaginal and Caesarean birth different for such reasons?
- **Both infant and mother** contribute to mother–infant bonding. Fairly obvious thing to say. Babies maintain eye contact with the mother, perceive facial expressions and respond to them, cry, suckle, derive reinforcement from warm/cuddly/moving things, etc. Even newborn babies can recognise and respond to faces (they can see, but are short-sighted). Mothers have a characteristic pattern of behaviour to newborns; they pick it up, gaze at its face, stroke it, start breast-feeding etc. Infants that display abnormal patterns of behaviour, such as crying and showing avoidance responses when picked up, can affect the parents' behaviour adversely.
- Early social interaction between infant and parents can have long-lasting effects on both in a range of altricial species. Precise effects of poor "bonding" unclear in humans (much speculation!) but probably v. important.