OBSTETRIC PHARMACOLOGY

(a) ***To explain the physiological consequences of pregnancy and its pharmacological implications.***

Pharmacokinetic implications of physiological changes associated with pregnancy:

Absorption	Oral - N/V and heartburn common → ↓ absorption - ↓ gastric emptying only during labour (due to pain, anxiety, opioids) → ↓ absorption - ↓ gastric motility 2° to intestinal compression → ↑ gastric absorption but ↓ intestinal absorption IM/SC/transdermal - ↑ skin blood flow 2° to ↑ C.O. (by 30-40%) and ↓ SVR → ↑ absorption IV - ↑ C.O. → ↑ onset of action Neuraxial - ↓ epidural space 2° to engorged epidural veins → ↓ spinal and epidural doses Inhalational - Progesterone-mediated ↑ MV (by 50-70%) 2° to ↑ TV and MV, and ↓ FRC (by 20%) → ↑ FA/FI ratio (or uptake) of inhaled anaesthetic agents - ↑ C.O. → opposes effect of ↑ MV
Distribution	- ↑ TBW/ECF (by 50%) → ↑ V_D (esp for polar/ionized drugs (Eg. NMBD)) - ↑ body fat % → ↑ V_D and ↑ sequestration of lipid soluble drugs (Eg. propofol) - ↓ plasma protein 2° to dilutional effect a/w ↑ TBW/ECF: o (i) Esp ↓ albumin → results in ↑ free % of acidic drugs (Eg. STP, propofol) → thus, ↓ dose required and ↑ transplacental transfer of drug. Note – this is further exacerbated late in pregnancy when ↑ FFA that competes with acidic drugs for binding to remaining plasma albumin o (ii) ↓ A1AGP (by 30%) → ↑ free % of basic drugs (Eg. LA, β blockers) → thus, ↓ dose required and ↑ transplacental transfer of drug - Ionisation → mild respiratory alkalosis (pH 7.42) 2° to ↑ MV → ↑ transplacental transfer of basic drugs as they will have ↑ % in unionized form → also ↑ ion trapping of drug in more acidotic foetal circulation
Metabolism	- Progesterone:oestrogen ratio o Progesterone → induces hepatic enzymes → ↑ metabolism o Oestrogen → inhibits hepatic enzymes → ↓ metabolism - ↓ plasma cholinesterase (30%) → ↓ metabolism of SCh (Nb. but ↑ prolonged effect of SCh is offset by ↑ in its V_D) - Placenta has enzymes similar to liver → metabolises drugs also - Foetal liver have functioning CYP450 and can metabolise drugs too → but are poor conjugators (Ie. drugs pass back into maternal circulation for conjugation)
Excretion	- ↑ RBF/GFR (50%) → ↑ clearance/↓ elimination t ½ of water-soluble drugs - ↑ MV/↓ FRC → ↑ washout of volatile agents

Pharmacodynamic implications of physiological changes associated with pregnancy:

- MAC of inhaled anaesthetic agents ↓ 40% (due to progesterone/β-endorphins)
- ↓ induction dose of STP by 35% (due to ↑ Vd and elimination)
- ↑ sensitivity to vecuronium (as ED_{50} ↓ by 50%)
- Effect on SCh relatively unchanged (↓ metabolism 2° to ↓ plasma cholinesterase levels offset by ↑ Vd)

- ↓ LA dose (by 25-30%) required in neuraxial blocks due to → (i) ↑ spread of LA (esp at 2nd TM due to distension of EDV), (ii) ↑ sensitivity of nerve fibres to LA, and (iii) ↑ diffusion of LA to membrane receptor sites
- ↓ responsiveness to vasopressors (Eg. ephedrine)
- ↑ hypotension associated with regional blockade (due to ↑ SNS tone during pregnancy)
- ↑ dose of anti-coagulants (Eg. heparin, warfarin) due to ↑ CFs

(b) ***To describe the pharmacology of oxytocic agents with special reference to oxytocin derivatives, ergot derivatives and prostaglandins.***

"Oxytoxic agent" – A drug that can stimulate uterine contraction → used to induce/augment labour or prevent/treat PPH post-delivery

Types of oxytoxic agents:

(1) Oxytocin derivatives:
- (a) Syntocinon:
  - Overview – Synthetic oxytocin (which is a naturally-occurring 9 a.a. polypeptide produced by hypothalamus → secreted by posterior pituitary)
  - Presentation – Clear colourless solution for IV/IM (5-10 units/mL)
  - Indications:
    - (i) Induce/augment labour at term (20 mU/mL at IVI of 5 mL/hr, titrate to effect)
    - (ii) Uterine contraction post-delivery to ↓ PPH (IV bolus of 5 units +/- infusion at 5 units/hr)
    - (iii) Aid lactation
  - Mechanism of action
    - Binds to oxytocin receptors (GPCR – Gq) on myometrium → opens membrane-bound Ca^{2+} channels → ↑ Ca^{2+} influx which stimulates Ca^{2+} release from sarcoplasmic reticulum → ↑ IC [Ca^{2+}] causes ↑ force and frequency of uterine contractions
    - Oestrogen induces expression of oxytocin receptors during pregnancy → uterus becomes ↑ sensitive to oxytocin as pregnancy progresses 2° to receptor upregulation
  - Pharmacokinetics:
    - Absorbed parentally only (IM, IV, intranasal) → inactivated orally by GI peptidases
    - Inactivated by hepatic, renal and placental oxytocinase (aminopeptidase made during pregnancy)
    - Short duration of activity (elimination ½ 5-12 mins) → requires IV infusion
  - Pharmacodynamic effects:
    - Dose-dependent ↑amplitude and frequency of uterine contraction BUT with no ↑ in basal resting tone (Nb. risk of uterine rupture if excessive)
    - Breast myoepithelial contraction → milk ejection
    - ↓ SVR (30%) 2° to direct VSMC relaxation → causes flushing, headaches, hypotension (↓ MAP, SBP/DBP), and reflex tachycardia and ↑ C.O.
    - Minor ADH effects at high doses (due to V2R binding in renal tubules) → risk of ↓ Na^+/water intoxication (esp with hypotonic IVF) 2° to H_2O reabsorption in CD
    - ↑ PAP/PVR (esp in PHT)
    - Other side-effects – anaphylaxis, arrhythmias (prolongs QTc), and foetal hypoxia/acidosis if given prior to delivery
  - Notable issues:
    - Should not be administered in same line as blood/plasma products → due to plasma hydrolysis/inactivation of drug
    - ↑ dose of SCh
- (b) Carbetocin:

- Synthetic peptide anaologue of oxytocin → has a more prolonged effect (elimination t ½ 4-10x longer; lasts 60 mins) but is less potent than syntocinon
- Given parentally only (IV or IM 100 mcg)

(2) Ergot derivatives:
- Overview:
  - Ergots are naturally-occurring alkaloid produced by fungus which grows on grain
  - Commonly used ergot derivative in obstetrics → Ergometrine
  - "Syntometrine" → ergometrine 0.5 mg combined with oxytocin 5 units
- Indications:
  - Contract uterus post-delivery to ↓ PPH (IV 0.2 mg very slowly; IMI 0.5 mg)
  - Note – Cannot be used to induce/augment labour → b/c it causes sustained ↑ in resting/basal uterine tone
- Mechanism of action – Unknown
- Pharmacokinetics:
  - Given IV/IM → onset in 40 secs IV (10 mins IM) with 2-3 hrs duration of action
  - Hepatic metabolism
- Pharmacodynamic effects:
  - ↑ uterine contractions (esp sustained ↑ in basal/resting uterine tone with high doses) → Nb. risk of violent abnormal contractions a/w uterine rupture
  - Significant arterial vasoconstriction due to (i) direct VSMC contraction and (ii) vasomotor ctr depression (prevents reflex vasodilation) → causing:
    - ↑ SVR → ↑ systemic BP by > 30-40 mmHg → HTN (50%)
    - ↑ PVR → ↑ PAP
    - Risk of myocardial ischaemia (2° to coronary artery spasm and HTN), APO (2° to ↑ afterload), CVAs, seizures and retinal detachment
  - Highly emetogenic (75%)
  - Bradycardia due to ↑ vagal tone
- Contraindications – Preexisting HTN (esp PET), PHT and cardiac disease

(3) Prostanglandins:
- (1) PGF2α (Dinoprost)
  - Synthetic PG used to treat PPH → only injectable PG available (diluted with N/S and given via intramyometrial injection (0.5-1 mg) – Nb. but NOT given IV!)
  - Mechanism of action – ↑ IC [Ca^{2+}] due to ↑ Ca^{2+} release from SR and ↑ Ca^{2+} entry into cells → ↑ MLCK activity → ↑ myometrial contraction
  - Causes uterine contraction
  - Side-effects – N/V, diarrhoea, severe vasoconstriction (↑ SVR → systemic HTN; ↑ PVR → PHT), bronchoconstriction, arrhythmias, APO, uterine rupture, seizures
- (2) PGE1 (Misoprostol)
  - Used to induce labour, ripen cervix or treat PPH → given PO, S/L, rectally or intra-vaginally (as a pessary)
  - Mechanism of action – As PGF2α
  - Causes uterine contractions and dilates/thins cervix
  - Side-effects – N/V, fever, vasodilation (↓ SVR → hypotension a/w reflex tachycardia and ↑ C.O.), bronchodilation, uterine rupture
  - Note – Safer to use in pts with asthma or PHT
- (3) PGE2 (Dinoprostone)
  - Used to induce labour and ripen cervix (limited use in PPH as it is not thermostable) → given as vaginal pessary or cervical gel
  - Similar mechanism of action, uterine/cervical effects and side-effects as PGE1

(c) ***To describe the pharmacology of tocolytic agents with particular reference to beta 2 agonists, calcium antagonists, magnesium, inhalational anaesthetics, nitrates and NSAIDs.***

"Tocolytic agent" – A drug that can inhibit labor by retarding or ceasing uterine contractions → used to treat premature labour to allow foetus to gain size/maturity prior to delivery

Types of tocolytic agents:

(1) Beta-2 agonists:
- Types – Salbutamol, Terbutaline, Ritodrine
- Use – 2nd line agent for acute tocolysis of preterm labour
- Mechanism of action – Stimulates membrane β2 receptors (GPCR – Gs; ↑ cAMP) on uterine myometrial cells → ↓ IC [Ca^{2+}] → results in uterine relaxation
- Side-effects:
    - Maternal – CVS effects (tachycardia, hypotension, arrhythmias, angina, APO), tremor, hyperglycaemia, hypokalaemia (esp if hyperventilating)
    - Foetal – Tachycardia, hyperglycaemia, hyperinsulinaemia (risk of delayed hypoglycaemia)
- Anaesthetic issues:
    - GA: Avoid halothane (↑ arrhythmogenicity to catecholamines), avoid hyperventilation (↓ K^{+}), avoid ketamine
    - RA: Ephedrine is less effective; phenylephrine more effective

(2) Calcium channel blockers:
- Type – Nifedipine (PO/SL 10-20 mg every 4-6 hrs)
- Use – 1st line agent for acute tocolysis of preterm labour
- Mechanism of action – Inhibits membrane L-type VG Ca^{2+} channels → inhibits Ca^{2+} entry causing ↓ IC [Ca^{2+}] → relaxes uterine myometrium
- Side-effects – Vasodilation (causing flushing, headaches, hypotension a/w reflex tachycardia/↑ C.O.), peripheral oedema, conduction defects (esp AV heart block if used with Mg^{2+}), N/V, myocardial depression

(3) NSAIDs:
- Type – Indomethacin (PO/PR 50 mg; then PO 25 mg every 4-6 hrs)
- Use – Not used for acute tocolysis → less commonly used due to side-effects
- Mechanism of action – Reversible and non-specific inhibitor of COX → ↓ PG synthesis from arachidonic acid (esp PGF2α and PGE2 that stimulate uterine contraction)
- Side-effects:
    - Maternal – Abnormal platelet function, renal impairment, gastritis, reflux, HTN
    - Foetal (can cross placental barrier) – Premature closure of DA, necrotizing enterocolitis, foetal oliguria (a/w oligohydramnios), ICH and bronchopulmonary dysplasias
    - Note – Has longer t ½ in foetus (12 hrs vs 2 hrs in mother)

(4) Magnesium (Mg^{2+}):
- Use – Also a 1st line agent for acute tocolysis of preterm labour (IV 4 g (over 20 mins), then IVI 1-4 g/hr (aim serum [ ] 1.7-3.5 mmol/L))
- Mechanism of action – Competes with Ca^{2+} at membrane L-type VG Ca^{2+} channels and low-affinity Ca^{2+} binding sites on SR membrane → ↓ IC [Ca^{2+}] → uterine relaxation
- Side-effects less cf. other tocolytics:

    - CVS effects (transient hypotension, chest pain, palpitations, pulmonary oedema)
    - CNS effects (blurred vision, sedation, abnormal NMJ function)
    - N/V
    - Respiratory depression and CVS collapse with toxicity
- Anaesthetic considerations:
    - Potentiates effects of ND NMBD
    - MAC sparing (by 20%)
    - Attenuates vasopressor effects

(5) Nitrates:
- Type – Glyceryl trinitrate (GTN) → IV 50-100 mcg or S/L 400 mcg spray; Amyl nitrate
- Uses – Potent and rapid uterine SM relaxant used esp in breech deliveries and extraction of retained placenta under RA
- Mechanism of action – NO is liberated from GTN via a thiol-dependent pathway (nitrate reductase) in liver and VSMC → NO activates guanylyl cyclase and↑ IC [cGMP] → ↓ IC [Ca^{2+}] → uterine relaxation
- Side-effects – Hypotension (with reflex tachycardia), flushing, headaches, pulmonary oedema

(6) Inhalational anaesthetic agents:
- All agents (EXCEPT N_2O) cause uterine relaxation with MAC > 0.5 (BUT atonic uterus → ↑ PPH risk)
- Mechanism of action – Uterine relaxation caused by ↓ IC [Ca^{2+}] due to (i) Blockade of uterine myometrial VG Ca^{2+} channels, and (ii) ↑ NO production in surrounding VSMC

(7) Competitive oxytocin antagonist (Atosiban):
- Mechanism of action – Competitively inhibits oxytocin receptor
- Not available in Australia yet
- Few maternal side-effects and no foetal effects (as it does NOT cross the placenta)

Important to note – Uterine muscle has the following innervation:
- α1 receptor – stimulation causes ↑ resting tone and strength of contractions (Eg. high dose adrenaline, NAd, phenylephrine)
- β2 receptor – stimulation causes↓ resting tone and strength of contractions (Eg. low dose adrenaline, salbutamol, isoprenaline)
- Cholinergic – stimulation causes ↑ strength of contractions only (Eg. neostigmine)

(d) ***To explain the factors which influence the transfer of drugs across the placenta to the foetus.***

Most drugs are transferred across the placental barrier via "simple passive diffusion" (Ie. transferred down their [ ] gradients without use of energy) → rate of transfer follows "Fick's Law of Diffusion":

$$J = \frac{D \times A \times dC}{dT}$$

Where:

- (1) [ ] gradient of drug across placental barrier (dC) – determined by:
  - (i) Maternal-foetal arterial $[\ ]_{GRADIENT}$ of drug in its free and unionized form – ↑ $[\ ]_{GRADIENT}$ of drug in its free/unionized form → ↑ diffusion rate of drug
  - (ii) Maternal (uteroplacental) and foetal (umbilical) blood flows – ↑ blood flows cause ↑ $[\ ]_{GRADIENT}$ drug (particularly lipid-soluble drugs (Eg. LA, fentanyl) >> water-soluble drugs (Eg. morphine)) → ↑ diffusion rate of drug
  - (iii) Protein binding/dissociation rate
    - Drugs with ↑ protein binding in maternal blood will have ↓ % drug in free form → ↓ $[\ ]_{GRADIENT}$ across placenta → ↓ rate of diffusion to foetal circulation
    - Foetal content of plasma protein is low (esp A1AGP > albumin) → thus have ↑ % drug in free form (esp basic > acidic drugs) → ↓ $[\ ]_{GRADIENT}$ across placenta → ↓ rate of diffusion
  - (iv) Metabolism of substance by placenta – Placental metabolism causes ↓ $[\ ]_{GRADIENT}$ → ↓ diffusion rate of drug
- (2) Surface area of placental barrier (A) – Exchange area = 1.8 m^2 (Nb. total villous area = 16 m^2) → factors that ↓ placental SA will ↓ diffusion rate of drug
- (3) Thickness of placental barrier (dT) – 3.5 um → factors that ↑ placental thickness will ↓ diffusion rate of drug
- (4) Diffusion capacity of substance by placenta (D) – determined by drug's:
  - (a) MWT – ↑ MWT of drug will ↓ diffusion rate of drug
  - (b) Lipid solubility – ↑ lipid soluble drug will have ↑ diffusion rate
  - (c) Degree of ionization
    - (i) Unionised drugs have ↑ transplacental transfer than ionised drugs
    - (ii) Maternal/foetal blood pH and pKa of drug → determines "Ion trapping"
      - Weak bases (Eg. LA) have a ↑ % unionized form in mother (pH 7.4) → cross placental barrier into foetal circulation → as foetal blood is more acidotic (pH 7.3; esp with foetal distress), drug will have ↑ % ionized form and become trapped in foetal circulation
      - Weak acids (Eg. STP) have ↓ % unionized form in maternal blood → trapped in maternal circulation

Note – Measurement of placental transfer of drugs:

- Determined by the "foeto-maternal (FM) ratio" or "UV/MV ratio" → ratio of [drug] in umbilical blood to maternal blood at time of birth
- So – ↑ UV/MV or FM ratio = ↑ placental transfer

(e) ***To outline the potential effects on the foetus and neonate of drugs administered during pregnancy.***

Safety of drugs administered during pregnancy:

Pregnancy category A	Drugs which have been taken by a large number of pregnant women and women of childbearing age without an increase in the frequency of malformations or other direct or indirect harmful effects on the fetus having been observed
Pregnancy category B1	Drugs which have been taken by only a limited number of pregnant women and women of childbearing age, without an increase in the frequency of malformation or other direct or indirect harmful effects on the human fetus having been observed
Pregnancy category B2	Like B1 except – Studies in animals are inadequate or may be lacking, but available data show no evidence of an increased occurrence of fetal damage
Pregnancy category B3	Like B1 except – Studies in animals have shown evidence of an increased occurrence of fetal damage, the significance of which is considered uncertain in humans
Pregnancy category C	Drugs which, owing to their pharmaceutical effects, have caused or may be suspected of causing, harmful effects on the human fetus or neonate without causing malformations. These effects may be reversible.
Pregnancy category D	Drugs which have caused, are suspected to have caused or may be expected to cause, an increased incidence of human fetal malformations or irreversible damage. These drugs may also have adverse pharmacological effects.
Pregnancy category X	Drugs that have such a high risk of causing permanent damage to the fetus that they should NOT be used in pregnancy or when there is a possibility of pregnancy

Note – Teratogen exposure in 1st 2-3 weeks post-conception usually results in spontaneous loss → b/t 30-70 days is peak time where teratogenicity is most likely!

Effect of anaesthetic agents on foetus/neonates during pregnancy:

- IV induction agents:
  - (i) Propofol
    - Rapid placental transfer with significant foetal uptake (as lipophilic, low MWT, non-ionised and flow-dependent uptake) → FM ratio 0.7-0.85
    - Not metabolised in foetus → undergoes rapid clearance back into maternal circulation (Ie. transfers back across placenta)
    - Foetal impact mixed → may or may not ↓ APGAR/neurobehavioural scores
  - (ii) STP
    - Rapid placental transfer with significant foetal uptake (as lipophilic, low MWT, non-ionised and flow-dependent uptake) → FM ratio 0.4-1.1
    - Minimal foetal metabolism
    - Foetal impact minimal (Ie. ↓ APGAR/neurobehavioural scores) if dose < 7 mg/kg
  - (iii) Ketamine
    - Rapid placental transfer and foetal uptake due to moderate lipid solubility

        - Dose > 1 mg/kg → neonatal depression, muscular hypertonicity, ↓ APGAR scores
- Inhaled anaesthetic agents:
    - Readily transferred across placental (FM ratio 0.9 for isoflurane, 0.8 for N_2O, 0.87 for halothane) → no neonatal depression
    - No blood loss if < 0.5 MAC, uterine relaxation (and blood loss) if > 0.5 MAC, uterine arterial dilation (and good foetal perfusion) if > 1.5 MAC
- NMBD:
    - Very slow transplacental transfer as NMBDs are ionized (FM ratio 0.07 for atracurium, 0.11 for vecuronium, 0.16 for rocuronium, 0.19 for pancuronium)
    - Nil issue with use of SCh
- Benzodiazepines:
    - Midazolam preferred to diazepam → ↓ incidence of "floppy infant syndrome (Ie. foetal hypotonia, lethargy, poor feeding)
- Narcotics:
    - Rapid placental transfer and significant foetal uptake (as lipophilic, poorly protein bound), and accumulation in foetus (due to reduced metabolism/excretion) → Nb. limit foetal exposure with neuraxial administration (vs IM/IV)
    - Issues – Foetal respiratory depression and ↓ neurobehvioural scores
    - Types:
        - (i) Pethidine – Commonly used for labour analgesia → large amts cross into foetus as it is highly lipid soluble (exposure highest 2-3 hrs after IMI) → it is metabolized to norpethidine (less lipid soluble), but due to ↓ foetal clearance both pethidine and norpethidine accumulate (t ½ 16 hrs can be prolonged by 3x) → issues with foetal respiratory depression and ↓ neurobehvioural scores
        - (ii) Morphine – ↓ placental transfer cf. pethidine (due to ↑ H_2O solubility)
        - (iii) Fentanyl – Lipid soluble like pethidine but a/w ↓ foetal issues (esp if EDB dose < 100 mcg)
        - (iv) Remifentanyl – No neonatal issues as it is rapidly metabolized by foetus
- LAs:
    - Generally cross placenta readily (depends on pKa and protein binding of LA agent)
    - Issues – Foetal acidosis/distress due to ↓ UBF a/w hypotension and ↑ uterine contractions (loss of tocolytic effect of catecholamines from sympathectomy) from regional block → can lead to ↑ ion trapping of LA in foetus
    - Types:
        - (i) Lignocaine (FM ratio 0.5-0.7; PB 64%)
        - (ii) Bupivacaine (FM ratio 0.25-0.45; PB 95%)
        - (iii) Ropivacaine (FM ratio 0.2; PB 95%)
- NSAIDs:
    - Avoid due to premature closure of ductus arteriosus and foetal oliguria (causing oligohydramnios)

<u>Effect of non-anaesthetic agents on foetus/neonates during pregnancy</u>:

- Warfarin is contraindicated during pregnancy (but heparin is OK as it does not cross placenta)
- Anticonvulsants (Eg. phenytoin, valproate, Etc.) can cross placenta → foetal congenital abnormality rate of 4-8%
- Antidepressants OK
- Anti-HTN agent (hydralazine is preferred)

(f) ***To outline the potential effects on the neonate of drug administration in association with lactation.***

Overview of drug passage in breast milk:

- Most drugs given to a lactating mother are secreted in breast milk → generally at low [ ] that are not usually of clinical significance to the neonate
- The amount drug detected in breast milk is a variable fraction of maternal blood level, which is proportional to the oral maternal dose → so the resulting dose in breast milk is ~ 1-2% of the maternal dose
- Lactation is not fully established in 1st few days post-partum → so drugs given in this period (cf. later on) have minimal transfer into breast milk (Ie. neonate receives small volumes of colostrum containing little drug)

Adverse neonatal effects due to drug exposure with lactation:

- (i) Effect of toxic drugs (Eg. cytotoxic drugs, substances of abuse)
- (ii) Neonatal allergy to drug
- (iii) Toxicity in mother (Ie. due to OD or organ impairment) → cause toxicity in neonate

Factors determining drug passage from mother to baby with lactation:

- (i) Maternal drug absorption
- (ii) Maternal drug metabolism
- (iii) Passage of drug into breast milk → occurs via passive diffusion (lipid solubility, MWT, degree of ionization, degree of protein binding of drug) +/- active secretion
- (iv) Oral absorption by infant → influenced by solubility in GIT and hepatic first-pass metabolism of infant

Therapeutic points to consider when prescribing drugs while lactating:

- (i) Avoid drugs that are have contraindicated or concerns during lactation (esp cytotoxic drugs, lithium, amiodarone, prolonged diazepam use, ?metoclopramide, amphetamines, Etc.)
- (ii) Avoid long-acting drugs (Eg. long elimination t ½) and/or drugs that are highly excreted in breast milk
- (iii) Consider withholding medication (if appropriate)
- (iv) Consider alternate routes of drug administration (Eg. inhaled, topical) that minimize systemic absorption
- (v) Avoid breast feeding when plasma drug levels are high
- (vi) Take drugs after feeding
- (vii) Consider temporarily withholding feeding

> Important to note – Lactating patient undergoing anaesthesia should be advised to (i) provide a 24 hr supply of expressed milk to be stored pre-operatively for feeding post-operatively, and (ii) express/discard milk in the 1st 24 hrs post-operatively