

MATERNAL PHYSIOLOGICAL CHANGES IN PREGNANCY

Respiratory

- ** overall there is ~ 50% loss of respiratory reserve,
 - a. increased $\dot{V}O_2$
 - b. decreased FRC
 - c. decreased CVS reserve
 - d. airway changes

- lung volumes change from about **5-10 weeks**, changes being increased by the *supine* position,

Alteration of Lung Volumes in Pregnancy		
Functional Residual Capacity	FRC	~ 20% decrease
Residual Volume	RV	
Expiratory Reserve Volume	ERV	
Vital Capacity	VC	unchanged
Inspiratory Reserve Volume	IRV	
Closing Volume	CV	
Total Lung Capacity	TLC	~ 5% decrease
Inspiratory Capacity	IC	~ 5% increase

- contrary to previous reports, the diaphragm is **not** splinted but moves freely throughout pregnancy
- the transverse and AP diameter of the chest increases to compensate for the elevation of the diaphragm
- as intra-abdominal contents increase, diaphragmatic breathing decreases in favour of thoracic excursion
- measurements of CV reveal that ~ 33% (up to 50% ASA) have airway closure during normal tidal ventilation, especially in the supine position
- factors which do increase CV are advancing age, smoking and lung disease
- airway closure increases atelectasis, shunt flow and V/Q mismatch,
 - increased $P_A O_2$ and $P_{A-a} O_2$ gradient

- there is capillary engorgement of the mucous membranes throughout the respiratory tract, causing swelling of the nasopharynx, oropharynx, larynx and trachea
- this may lead to difficulty in visualisation of the vocal cords, or to haemorrhage following manipulation of the airway

Obstetric Anaesthesia

Respiratory

- increased levels of **progesterone** ± oestrogen sensitise the respiratory centre to CO_2
- **dyspnoea** is experienced by ~ 60-70% of women early in pregnancy, when ventilation is only mildly increased

1. ↑ minute ventilation ~ 50% at term
 - i. ↑ tidal volume ~ 40%
 - ii. ↑ respiratory rate ~ 15% → ↓ **dead space** ventilation
2. ↑ **alveolar ventilation** ~ 70% above the nonpregnant state

- this is remarkable, in that it far exceeds,

- i. ↑ body weight ~ 20%
- ii. ↑ MRO_2 ~ 15-20%

- several studies have shown a decrease in **physiological dead space**, independent of tidal volume & rate changes
- this is believed to be due to more efficient mixing and distribution of ventilation, and possibly the increased CO of pregnancy → **decreased** ETCO_2 - PaCO_2 gradient
- pregnancy may actually be associated with a **negative** ETCO_2 - PaCO_2 gradient (CJA)

- progesterone → bronchodilatation and decreased airways resistance
- however, FEV_1 , MBC and diffusing capacity remain unchanged
- pulmonary **compliance** is decreased ~ 30%,

- i. C_L - unaltered
- ii. C_{CW} - decreased ~ 45%
- returns to normal immediately following delivery

- oxygen consumption increases ~ 20% during pregnancy, and up to 100% during labour
- the HbO_2 curve is shifted to the **right**, and arterial gas analysis reflects **chronic hyperventilation**

→ pH ~ 7.44
PaO₂ ~ 95-105 mmHg
PaCO₂ ~ 32 mmHg
HCO₃⁻ ~ 21 mmol/l

- PaO₂ tends to be high early, falling as FRC encroaches upon CV, and may be normal or slightly subnormal at term
- plasma HCO₃⁻ decreases to ~ 21 mmol/l to compensate for the $P_{a\text{CO}_2}$
- therefore, at term there is less **buffer reserve**, and metabolic acidosis readily develops
- during labour ventilation may increase 300% with marked maternal **hypocarbia & alkalaemia**,

→ P_{aCO2} ~ 20 mmHg
pH ~ 7.55

- as a result of this, between contractions women may hypoventilate, with periods of **hypoxaemia**
- this is one of the advantages of epidural anaesthesia & adequate pain relief

Respiratory - Importance for Anaesthesia

- a. intubation
 - mucosal bleeding
 - difficult intubation
- b. respiratory reserve
 - rapid onset of hypoxia*
 - high MRO_2 / low FRC \pm low PaO_2
 - decreased CO supine
- c. rapid gaseous induction
 - i. \downarrow MAC
 - \sim 25% halothane
 - \sim 40% isoflurane
 - ? progesterone, probably endorphins
 - ii. \downarrow FRC \rightarrow less dilution
 - iii. \uparrow MV \rightarrow rapid δ depth
- d. foetal effects
 - i. maternal respiratory alkalosis
 - umbilical/uterine vasoconstriction
 - \uparrow maternal HbO_2 affinity (relative not absolute, \uparrow DPG & curve \rightarrow R)
 - ii. maternal hypoventilation between contractions
 - 2^o to extreme hyperventilation with pain
 - epidural anaesthesia normalises PaO_2 & reduces MRO_2

NB: following adequate preoxygenation, the PaO_2 in apnoeic pregnant women falls
 \sim 80 mmHg/min more than in the nonpregnant state

Cardiovascular Changes

■ Blood Volume

i.	↑ plasma	~ 50%	∞	aldosterone & oestrogen
		~ 40	→	70 ml/kg
ii.	↑ RBC mass	~ 20-30%	→	dilutional ↓ [Hb] & haematocrit
		~ 25	→	30 ml/kg
iii.	↑ blood volume	~ 30-40%		
		~ 65	→	100 ml/kg

- ↑ RBC mass occurs slower than ↑ plasma, accounting for the relative **anaemia** of pregnancy
- administration of iron supplements results in a near normal [Hb]
- average blood loss during delivery,

i.	vaginal delivery	~ 200-500 ml
ii.	episiotomy	~ 150 ml
iii.	LUSCS + GA	~ 1000 ml
iv.	LUSCS + epidural	~ 600 ml

- **autotransfusion** of ~ 500 ml at delivery from the placenta into the maternal circulation
- this usually compensates, with the post-partum Hct. varying ± 5%

■ Cardiac Output

- increases by 8-10/52 gestation, reaching a peak at ~ 30/52, due to an increase in,

i.	↑ SV ~ 35%	
ii.	↑ HR ~ 15%	→ - CO ~ 40-50%

- CVP changes little, except during labor, or due to the effects of **aortocaval compression**

→ ↓ CO ~ 40% in in the **supine position**

- the pain and apprehension of labour increase circulating catecholamines and increase SV and CO a further 40-45% above prelabour values
- uterine contraction further augments CO by **autotransfusion**, with central blood volume increasing 10-25% with each contraction (200-300 ml)
- **BP** increases with each contraction as PVR increases together with the increases in CO
- during the **3rd stage**, **CO** increases ~ 80% above prelabour values (NB: MCQ)
- CO returns to normal by the 2nd postpartum week

NB: **aortocaval compression** is of major importance for foetal well-being

- most women do not become overtly hypotensive, "concealed caval compression"
- however, compensation is frequently inadequate
- ~ 10% of women become frankly hypotensive, "revealed caval compression"
- frequently with a superimposed **reflex bradycardia**, despite the inability to maintain peripheral vascular tone

■ Cardiac Work

- is increased, which may result in LVF when there is poor cardiac reserve
- changes found on clinical examination include,
 - a. an ejection systolic murmur
 - b. a loud split first heart sound
 - c. occasionally a soft diastolic murmur
 - d. the apex beat is displaced to the left
 - e. the ECG axis shifts to the left
 - f. the heart may appear enlarged on CXR

■ Blood Pressure

- a. \uparrow CO ~ 50%
- b. \downarrow TPR - uterine AV shunt & decreased viscosity
- c. \rightarrow slight *decrease in MAP*

NB: a high BP in pregnancy, except during labour, is *always abnormal*

- CVP and PAOP remain *normal* during pregnancy
- CVP increases 4-6 cmH₂O during contractions

■ Electrocardiogram LAD

- chamber volume and wall thickness increase during pregnancy
- upward displacement of the diaphragm and elevation of the heart
- arrhythmias occur more commonly during pregnancy, but are usually benign

■ Oxygen Flux

- *increases* despite the slight decrease in [Hb] and O₂ content, due to,
 - a. the marked increase in CO
 - b. hyperventilation and an increase in PaO₂ (early)
 - c. \uparrow 2,3-DPG \rightarrow HbO₂ dissociation curve to the *right*
 \rightarrow **P₅₀ ~ 30.4 mmHg** (cf. 26.7)

Cardiovascular - Importance for Anaesthesia

- patients undergoing spinal or epidural anaesthesia must,
 - a. be maintained in a lateral tilt position, with left uterine displacement
 - b. be adequately volume preloaded
- distension of the peridural venous plexus leads to,
 - a. decreased epidural space volume & LA requirements
 - b. increased risk of intravascular injection or catheter insertion, and subsequent LA toxicity
 - c. increased epidural space pressure, rendering the "hanging drop" technique unreliable
- regional anaesthesia attenuates some of the CVS changes which normally accompany labour, excepting those in the third stage which are not due to circulating catecholamines
- thus, epidural anaesthesia is recommended for any patient in whom an increase in myocardial work is undesirable

Blood Constituents

- there is a slight **decrease** in $[Na^+]$, $[K^+]$ and $[Cl^-]$
- albumin, globulins & total protein increase, but their plasma concentrations decrease
- the normal albumin/globulin ratio of $\sim 1.6/1 \rightarrow 1/1$ at term
- colloid osmotic pressure progressively decreases, parallel with the fall in serum albumin
- further decreases in COP occur in the postpartum period, irrespective of the mode of delivery or type of anaesthesia used
- thus, the preeclamptic patient, or those on tocolytic therapy are prone to the development of **pulmonary oedema**, despite near normal PAOP's
- changes in plasma protein binding may lead to drug toxicity, due to an increase in the unbound fraction
- pregnancy leads to a **hypercoagulable state**, due to,
 - a. \uparrow factors VII, VIII, X, XII (? IX)
 - b. \uparrow fibrinogen (I) and FDP's
 - c. \downarrow fibrinolytic activity - \downarrow levels of plasminogen activators
 - d. \downarrow antithrombin III

\rightarrow **increased risk of thromboembolic disease**

Uterine Circulation

- in the nonpregnant state, blood flow parallels the metabolic activity of the myometrium and endometrium, undergoing cyclic variations with menstruation
- during pregnancy, blood flow increases rapidly with the increasing uterus and foetus
 - **£20 fold increase**
- early in pregnancy the O₂ extraction of the uterus is low, therefore, some factor other than autoregulation increases flow ??oestrogen
- as the size and requirements of the foetus increase much greater than blood flow during pregnancy, the O₂ extraction ratio increases progressively with pregnancy

Central Nervous System

- LA requirements for subarachnoid or epidural anaesthesia are reduced in pregnancy
- this was initially thought to be due to the mechanical effects of raised intra-abdominal pressure and engorgement of the epidural venous plexus, resulting in decreased volumes of the subarachnoid and epidural spaces
- this however may not be the case, as similar effects are seen early in pregnancy, prior to major increases in abdominal contents
- possible causes include,
 - a. increased diffusion of LA to the receptor site
 - b. increased sensitivity of nerve fibres to LA
 - c. ? raised CSF progesterone levels
- valsalva manoeuvres during delivery may increase CSF and epidural pressures, markedly increasing the spinal spread of anaesthetic
- the MAC for the volatile agents is also reduced ~ 40% during pregnancy
- this was thought to be due to progesterone, however, studies in rats have shown **no correlation** between plasma progesterone and MAC reduction
- other studies indicate there may be an upregulation of the **endogenous opiate** pathways and the endorphin system

Renal

- the ureters and renal pelvis progressively dilate from the 12th week
 - increased incidence of **UTI's**
- \uparrow RBF and GFR $\sim 60\%$ at term $\propto \uparrow$ CO and blood volume
- aldosterone levels are slightly increased, as are TBW and sodium
- plasma osmolality is decreased, with an effective "resetting" of the threshold for ADH secretion
- despite this, fluid and electrolyte balance remains close to normal, partly due to glomerulo-tubular balance
- urine volume increases due to the need to excrete a greater mass of waste products
- both BUN and $[\text{Cr}]_{\text{pl}}$ decrease due to an **increased creatinine clearance**
- during third trimester there may be alterations of renal function due to aortocaval compression

Hepatic & GIT Function

- LFT's show general increase due to **enzyme induction**
- liver blood flow is not significantly altered and bilirubin levels are unaltered
- $\bar{\text{plasma cholinesterase}} \sim 30\%$ and remains low for several weeks postpartum
- this generally doesn't affect the response to suxamethonium, as there is a larger V_{dSS} for the drug, which compensates for the decreased clearance
- during labour there is a greatly increased risk of **aspiration** and Mendelson's syndrome due to,
 1. \downarrow gastric emptying
 - displacement of the pylorus
 - increased progesterone - antagonises **motilin***
 - pain, anxiety
 - narcotic analgesics
 2. \downarrow lower oesophageal sphincter (LOS) tone
 - anticholinergics - atropine, glycopyrrrolate
 - narcotics
 3. \uparrow gastric acidity
 - placental **gastrin** secretion
 4. \uparrow intragastric pressure
 - uterine contents
 - lithotomy
 5. \uparrow incidence of difficulty with intubation
 6. frequently require emergency anaesthesia in the "middle of the night" by junior staff
- there is argument concerning at what stage these changes occur*
- some workers have shown changes starting in early pregnancy, whereas others have found no change until ~ 34 weeks

Endocrine

- endocrine disease is rare in pregnancy, as pre-existing disease usually results in infertility
- **thyroid disease** is the commonest endocrinopathy to present during pregnancy, however this is difficult to diagnose
- earliest changes are increased levels of,
 - a. oestrogen
 - b. progesterone
 - c. β hCG
- normal pregnancy is associated with increases in the size of the,
 - a. thyroid
 - \uparrow BMR & PBI
 - FT_4 **normal** and patients remain **euthyroid**
 - difficult as pregnancy symptoms mimic thyroid disease
 - b. parathyroid
 - \uparrow PTH \rightarrow \uparrow Vit.D₃
 \uparrow Ca⁺⁺ absorption
 \downarrow Ca⁺⁺ excretion
 - plasma [Ca⁺⁺] remains **normal**, the increase supplies the foetus
 - c. anterior pituitary
 - \uparrow ACTH \rightarrow cortisol
aldosterone
prolactin
 - \uparrow MSH, β -END
 - d. adrenals

FOETAL PHYSIOLOGY

Placental Circulation		
Normal Values	Average at Maturity	Adult Comparison
Weight	500 g	
Lobules	200 - each multiple villi	
Diffusion Distance	3.5 μm	~ 0.5 μm - lung
Surface Area	3-4 m^2	~ 70 m^2 - lung
Blood Flow _{F/M}	300 ml/min $\sim 50\%$ of CO	600 ml/min
Blood Volume _M	150 ml - intervillous spaces	
RBC Transit Time	15 secs	
$P_M\text{O}_2$	50 mmHg	
$P_F\text{O}_2$	30 mmHg	

- the placenta is effectively the "foetal lung"
- the maternal portion is effectively a large blood sinus, or lake, into which project the foetal placental villi, which contain the small branches of the umbilical arteries and vein (see Ganong, fig. 32-17)
- O_2 , CO_2 and nutrient exchange occur across the cellular layers covering the villi, however these are thicker and less permeable than lung and exchange is less efficient

Foetal Circulation

- $\sim 55\%$ of the foetal CO supplies the placenta via the umbilical arteries, where the saturation, $\text{SafO}_2 \sim 60\%$
 - umbilical vein saturation, **$\text{SuvO}_2 \sim 80\%$** , c.f. 98% of maternal arterial blood
 - of this, the majority passes through the liver, a small fraction passing directly into the IVC via the ***ductus venosus***
 - the portal and systemic venous blood of the foetus, $\text{SvfO}_2 \sim 26\%$
 - the mixed venous blood in the **IVC** \rightarrow **$\text{SvfO}_2 \sim 67\%$**
 - most blood entering the RA from the IVC passes directly to the LA via the patent ***foramen ovale***
 - most of the blood entering the RA from the SVC passes into the pulmonary artery, then via the ***ductus arteriosus*** into the descending aorta
- \rightarrow net effect being the head receives the better oxygenated blood

Foetal Respiration

- the tissues of foetal and newborn mammals have high resistance to hypoxia
- three factors aid in foetal transfer of O₂
 - a. [Hb_F] ~ 50% greater than [Hb_A] → greater CaO₂ /ml
 - b. Hb_F binds 2,3-DPG less effectively than Hb_A → left shift
 - c. Hb_F-CO₂ → Hb_A-CO₂ → "double" Bohr effect

NB: Hb_F-O₂ dissociation curve lies above and to the left
Hb_F-P₅₀ ~ 19 mmHg Hb_A-P₅₀ ~ 30.4 mmHg (cf. 26.7)

- the total *diffusing capacity* at term for O₂,
 - a. across the placenta ~ 1.2 ml/O₂/min/mmHg
 - b. across the lung ~ 20 ml/O₂/min/mmHg
- the γ-chains of Hb_F have a neutral amino acid at positions 143, 146 cf. histidine in β-chains
→ the decreased foetal binding of 2,3-DPG
- the maternal 2,3-DPG level increases near term, increasing the maternal P₅₀, improving unloading of O₂ to the foetus
- Hb_A begins to appear around the 20th week of intrauterine life
- no Hb_F is formed after birth,
 - a. at birth Hb_A ~ 20%
 - b. 4 months Hb_A > 90%
- as CO₂ is 20 times more diffusible and concentration gradient is high, diffusion does not present a problem
- the maternal PaCO₂ is reduced by hyperventilation of pregnancy

Obstetric Anaesthesia

Other Placental Functions

- a. active nutrient absorption - where [F] > [M] → amino acids, Cr, PO₄
- b. metabolism - various drugs by MFO's and Plasma-ChE
- c. metabolic functions - stores protein, Fe⁺⁺, Ca⁺⁺
- acts ≡^t liver early, until foetal liver matures
- d. hormone synthesis - βhCG
- oestrogen
- progesterone
- hPL

Normal Values	Maternal	Foetal
Hb concentration	12 g/100 ml	18 g/100 ml
Blood flow	600 ml/min	300 ml/min
Uterine/Umbilical aa.		
• PaO ₂	95 mmHg	15 mmHg
• SaO ₂	97%	58%
• PaCO ₂	35 mmHg	48 mmHg
Uterine/Umbilical vv.		
• PvO ₂	33 mmHg	30 mmHg
• SvO ₂	50%	80%

Uterine Blood Flow

- average values range from 600-700 ml/min at term
- flow is governed by the usual relationship,

$$Q = \frac{\delta P_{a-v}}{R_{vascular}}$$

- anaesthesia may alter flow by affecting,
 - a. perfusion pressure
 - b. vascular resistance directly
 - c. vascular resistance indirectly through *uterine tone* & positioning
- vasopressors with pure α-adrenergic action will decrease uterine flow
- drugs such as *ephedrine* & phenylephrine, having a predominantly venous action increase perfusion by improving CO

Uterine Activity & Labour

Def'n: *labour* is the physiological process by which the products of conception are expelled from the uterus through the vagina

- this requires active uterine contraction and progressive effacement and dilatation of the cervix
- labour is normally divided into three stages,
 1. first stage - start of regular contractions to full dilatation
 2. second stage - full dilatation to delivery of the baby
 3. third stage - delivery of the placenta and membranes
- during active labour contractions occur ~ 3 minutely
- the average duration is ~ 1 minute
- intrauterine pressure increases to ~ **50-70 mmHg**, c.f. basal tone ~ 10 mmHg
- abnormal processes of labour can be classified as,
 - a. slow latent phase > 20 hrs primiparous
> 14 hrs multiparous
 - b. active phase arrest - no dilatation for 2 hrs
 - c. arrest of descent - no descent for 1 hr

Pain Pathways

- during first stage pain is due to uterine contraction and cervical dilatation
- impulses travel via visceral *sympathetic afferents* from **T₁₀-L₁**
- late first and second stage stretching of the perineum produces additional pain which is transmitted through the *pudendal nerves* to **S₂-S₄**
- ranks as one of the severest forms of pain recorded, described as *intolerable* by ~ 1/3 of parturients

Placental Transfer Drugs

- as for most biological membranes, transfer is governed by *Fick's Law* of diffusion,

$$Q_t = k \cdot (A/d) \cdot \delta(C_M - C_F)$$

- the factors of importance to anaesthesia include,
 - a. the maternal - foetal concentration gradient
 - b. uterine and umbilical blood flow
 - c. the physicochemical properties of the drug determining the *diffusion constant* (k)
- factors which produce a high value for k , or rapid diffusion, include,
 - a. low molecular weight < 500 D
 - b. low protein binding
 - c. high lipid solubility
 - d. low degree of ionisation
- virtually all drugs used for sedation in anaesthesia meet these criteria
- the neuromuscular blocking drugs, due to their high degree of *ionisation*, are not transferred across the placenta in significant concentrations
- factors which tend to increase maternal drug levels include,
 - a. administration of large doses
 - b. administration into highly vascular areas
 - c. use of drugs which are poorly metabolised
- there are a number of differences in the foetal circulation which help protect the foetus against high concentration of any drug, these include,
 - a. metabolism by the placenta
 - b. uptake by the foetal liver
 - c. dilution of umbilical venous blood in the IVC
 - d. dilution of umbilical venous blood in the right atrium
 - e. shunting of blood across the foramen ovale
- foetal liver microsomes have significant levels of **cytochrome P₄₅₀** and **NADP**
- cytochrome C reductase appears as early as week 14 of gestation
- although these are present in lower concentrations than in the adult, they can metabolise significant quantities of a number of drugs, including most *local anaesthetics*
- haemodynamic changes in either the mother or foetus may affect passive drug diffusion, such that drugs administered during contractions are less likely to reach the foetus in high concentrations

ANAESTHESIA FOR VAGINAL DELIVERY

Psychological Techniques

- these include,
 - a. hypnosis
 - b. "psychoprophylaxis" - Lamaze
 - c. acupuncture
 - d. TENS

- acupuncture and TENS have been investigated by a number of investigators with little success
- the use of TENS for analgesia after LUSCS has also been investigated and does not reduce maternal opiate requirements
- using a McGill Pain Questionnaire, Melzack *et al.* showed little difference in the pain scores between patients who did and did not receive extensive preparation, such as Lamaze
- although such patients generally require less analgesia, assessments of neonatal outcome have shown no distinguishable neonatal benefit

Systemic Medication

■ Sedative-Tranquilizers

- anxiety can generally be minimised by proper antenatal preparation and education, plus the support of attendant and partner
- in addition to anxiolysis, many of this group are also potent anti-emetics
 - a. Barbiturates
 - short to medium acting agents such as secobarbital, pentobarbital, & amobarbital are now virtually obsolete due to their *depressant* effects on the neonate
 - even with small doses the attention span of the neonate may be reduced for 2-4 days

 - b. Phenothiazine Derivatives & Hydroxyzine
 - promethazine & propiomazine may be useful to relieve anxiety
 - agents such as chlorpromazine, promazine & prochlorperazine possess greater α -adrenergic blocking action and are therefore less popular
 - hydroxyzine, though not chemically a phenothiazine, possesses similar anxiolytic properties to the former two agents and is also effective in controlling emesis

c. **Benzodiazepines**

- these may be used for,
 - i. anxiolysis
 - ii. as an adjuvant to narcotics
 - iii. as a premedicant to LUSCS
 - iv. or in the treatment of preeclampsia & eclampsia
- **diazepam** has been the most widely used agent, providing good maternal sedation with minimal foetal respiratory depression
- it crosses the placenta rapidly, with maternal and foetal levels being approximately equal within minutes of IV injection
- the neonate is capable of metabolising small amounts of the drug
- however, when the maternal dose exceeds 30 mg, significant amounts of both the parent drug and its metabolites persist for up to 1 week in the neonate
- the principal adverse effects are,
 - i. hypotonia
 - ii. lethargy
 - iii. decreased feeding
 - iv. hypothermia
- beat to beat variability of the foetal heart rate is reduced by even small doses (5-10mg)
- **no** adverse effects on acid-base or clinical status are encountered
- **lorazepam** has a much shorter half life and < 1% is metabolised to active metabolites
- nevertheless, even small doses (1-2mg) are associated with adverse effects on foetal neurobehaviour, feeding and respiration
- **midazolam**, differing pharmacokinetically from the other BZD's, may cause respiratory depression in the mother when administered rapidly IV
- thus, IV administration should be slow, in increments **£ 1mg**
- when used for induction of anaesthesia for LUSCS, more depression of the neonate has been reported
- another disadvantage of this agent is the profound **antegrade amnesia**, which may be undesirable during regional procedures

■ Ketamine

- low dose ketamine (0.25 mg/kg) can be used in lieu of inhalational agents to produce systemic analgesia
- at higher doses (1 mg/kg) it may be used as an induction agent instead of STP
- in both dose ranges uterine blood flow, uterine tone and neonatal status are **not** adversely affected
- at doses greater than 1 mg/kg neonatal **hypertonia** and low Apgar scores have been reported

■ Inhalational Agents

- the inhalational agents may be used in **subanaesthetic** concentrations to provide pain relief in the first and second stages of labour
- the level of relief is generally not as good as that provided by regional anaesthesia
- nitrous oxide is the most commonly used agent, though, methoxyflurane may also be used, as may enflurane or isoflurane
- the usual concentrations are,
 - a. nitrous oxide 30-70% in O₂
 - b. methoxyflurane 0.25-0.35%
 - c. enflurane 0.25-1.0%
 - d. isoflurane 0.2-0.7%

NB: Ostheimer states that the Briham & Women's hospitals have not used volatiles, for this purpose, in the last 20 years because of the risk of aspiration and the lack of therapeutic effectiveness

- at these levels, the volatiles are **not** associated with a reduction in uterine tone, strength of contractions, or responsiveness to oxytocics
- neonatal acid-base status, respiration, oxygenation, Apgar scores and neurobehaviour are not affected at these levels
- the total administration of methoxyflurane should be limited to < 15 ml
- with this dose F⁻ levels rarely exceed 25 µmol/l in the mother and 15 µmol/l in the neonate
- methoxyflurane should be avoided in patients with pre-existing renal disease
- 30 ml of non-particulate antacid should be administered prior to administration
- verbal contact should be maintained with the patient at all times, and at any sign of overdosage ventilated with 100% O₂
- factors which make overdosage & loss of airway reflexes more likely include,
 1. decrease in FRC ~ 20%
 2. increased alveolar ventilation ~ 70%
 3. decreased MAC ~ 40%

REGIONAL ANAESTHESIA

NB: this is by far the most commonly used technique for pain relief in labour

■ Advantages

1. good analgesia
 2. minimal impairment of maternal functioning
 3. less likelihood of drug induced impairment of the foetus
 4. less likelihood of aspiration pneumonitis in the mother
- the most common forms of regional anaesthesia include,
 - a. subarachnoid anaesthesia
 - b. epidural anaesthesia/analgesia
 - c. caudal anaesthesia/analgesia
 - d. paracervical block
 - e. pudendal nerve block
 - f. local perineal infiltration

Obstetric Anaesthesia

Subarachnoid Anaesthesia

- may be used to provide,
 - a. "saddle" block prior to a spontaneous vaginal delivery
 - b. "saddle" block prior to a mid/low-forceps delivery
 - c. anaesthesia for caesarean section ± epidural
 - d. anaesthesia for abdominal surgery ± epidural

- in general, subarachnoid doses of LA are ~ **70%** of those used in the nonpregnant state
- administration of LA during a contraction may lead to excessive spread of the solution and an unusually high block
- the incidence of post-dural puncture headache is significantly lower (~ 10 fold) with the use of 26G cf. 25G needles
- the use of smaller gauges (< 26G) does not significantly alter the headache rate
- sympathetic blockade is managed c.f. normal, with volume expansion ± ephedrine

- hyperbaric commercial solutions of LA suitable for spinal blockade, ie., xylocaine 5% and bupivacaine 0.5% **do not** contain adrenaline, as the catecols are unstable in dextrose solutions
- addition of adrenaline to these solutions by the anaesthetist is technically difficult and impractical

Approximate Doses for Local Anaesthetics¹

Local Anaesthetic	Saddle Anaesthesia < T ₁₂		Abdominal Procedures S ₅ -T ₄	
Xylocaine 5% Heavy	30-50 mg	0.6-1.0 ml	75-100 mg	1.5-2.0 ml
Bupivacaine 0.5% Heavy	7.5-10 mg	1.5-2.0 ml	15-17.5 mg	3.0-3.5 ml
Chincocaine 0.5% Heavy	2.5-5 mg	0.5-1.0 ml	8-10 mg	1.6-2.0 ml
Bupivacaine 0.5% Iso.			17.5-20 mg	3.5-4.0 ml
¹ non-pregnant values, therefore use ~ 70%				

Epidural Anaesthesia

- indications for the establishment of epidural anaesthesia in labour include,

- a. **maternal** - minimise stress response
 - i. pain relief
 - ii. pre-eclampsia
 - iii. cardiorespiratory disease
 - iv. other diseases requiring minimal stress
 - diabetes
 - cerebrovascular disease
- b. **foetal** - high chance of instrumental delivery
 - i. multiple foetuses
 - ii. large foetus
 - iii. malpresentation
 - iv. premature foetus
 - v. deformed/dead foetus
- c. **uterine** - normalisation of abnormal physiology
 - i. uterine hypertonicity / incoordinate action
 - ii. cervical dystocia
 - iii. placental insufficiency
 - iv. ?? trial of scar

- continuous epidural anaesthesia may be commenced once labour is well established; ie., cervical dilatation of 3-6 cm with strong contractions
- this may be established earlier if augmentation with oxytocin is anticipated
- prior to the block, volume loading with 500-1000 ml of balanced salt solution reduces the incidence of hypotension

■ Second Stage

- may be slightly prolonged due to the loss of the bearing down reflex to perineal distension, however, with appropriate tuition this can be minimised
- on average there is a ***prolongation*** of second stage, however, there is no detrimental effect upon the foetus in terms of cord pH's or the incidence of "distress" on CTG
- the incidence of forceps delivery is very institution/operator dependent

■ Test Dose

- after placement of the catheter a **test dose** should be given, as negative aspiration does not exclude intravascular or subarachnoid placement
- the test dose should be such that,
 - a. SA injection
 - will result in a rapid onset of spinal block
 - not exceeding the upper thoracic dermatomes
 - i. lignocaine 75 mg = 5 ml / 1.5%
 - ii. bupivacaine 12.5 mg = 5 ml / 0.25% → ~ T₂
 - b. IV injection
 - will result in a tachycardia of 20-30 bpm, and
 - a slight increase in BP within 1 minute → lignocaine 1.5% + adrenaline
- ideally this should contain a vasoactive agent, ie. adrenaline, as the volume of LA used to recognise SA placement of the catheter is usually too small to recognise IV placement
- Moore & Batra (Anesth.1981) demonstrated that 3 ml of local anaesthetic with **adrenaline** 1:200,000 (15 µg) is an effective test dose in premedicated **surgical** patients
- Ostheimer recommends use of this technique for establishment of epidural anaesthesia for elective LUSCS in the **non-labouring** parturient, or for any other major regional anaesthetic
- criticisms of the use of **adrenaline** as a test agent in **active labour** include,
 - a. a high incidence of **false positives**, both in pregnant and nonpregnant individuals
 - b. the possibility of **false negatives** in mothers with highly variable baseline heart rates
 - Chestnut *et al.* (Anesth 1988) bupivacaine 15 mg/adrenaline 12.5 µg epidurally
 - 5 of 10 showed increases in HR > 25 bpm -10/+5 minutes of injection
 - c. the possibility of adverse effects on **uterine blood flow** and foetal well being
 - Wallis *et al.* (Anesth 1976) decrease in uterine artery blood flow in ewes
 - Leighton *et al.* (Anesth 1987) 15 µg IV to 10 pregnant patients & NaCl controls
 - 2 FHR tracing showed distress (not significant) but lasted 10-12 minutes
 - d. β-mimetic effects resulting in decreased **uterine tone**
 - Matadial & Cibils (Am J. O&G 1976) significant reduction in uterine activity
- alternatives, such as sub-convulsant doses of the LA's rely on subjective symptoms in the mother and carry the risk of being convulsant in a proportion of the population
- the use of other catecols, such as isoprenaline which has a greater effect on maternal HR with minimal effect on uterine perfusion, require preparation by the anaesthetist which is impractical

Obstetric Anaesthesia

- the decrease in uterine perfusion after **IV injection** of 3-4 ml of 1:200,000 adrenaline is transient and similar in duration and degree to that produced by a uterine contraction (RDM)
 - this is **not** seen with epidural injection of this quantity of adrenaline
 - the incidence of **false positives** can be reduced by,
 - a. injecting a repeated test dose where the results are equivocal (re IV placement), and
 - b. injecting between contractions when the maternal HR is more stable
 - Ostheimer recommends **not** using adrenaline in established labour, and a 3-5 ml test dose, with a maximum dose of **any solution ~ 5 ml**
 - even if placement of the catheter is confident, management should include,
 - a. **incremental injection** - irrespective of test dose results
*"every dose is a test dose !!"
 - b. **lateral tilt** position to prevent supine hypotension
 - c. regular BP measurement - 2 minutely for the first 10 minutes, then
- 10-15 minutely for the duration of blockade
 - d. prompt management of **hypotension**
 - i. ↓ MAP ~ 20-30%
 - ii. ↓ SAP < 90 mmHg, or
 - iii. maternal symptoms →
 - left lateral tilt
 - IV fluid bolus
 - high flow O₂ by face mask
 - 10-20% of Trendelenburg
 - ± ephedrine 5-15 mg IV if no response
 - 5. monitoring with a foetal cardiotocograph
-
- if unilateral analgesia results then administration of further solution with the sensitive side dependent may be successful

Continuous Epidural Infusions

■ Advantages

1. reduced fluctuations in the level of pain relief
2. with the use of dilute solutions the amount of motor blockade is reduced
3. fewer hypotensive episodes
4. no requirement for repeat test dosing or frequent monitoring once the block is established

NB: however, infusions do require that the patient is examined at regular intervals to adjust the infusion rate

■ Complications

- a. overdosage & high blockade
- b. segmental blockade
- c. subarachnoid catheter migration
- d. intravascular migration
*this is unlikely to produce any side-effects, except *loss of analgesia*
- e. infection

• the addition of opioids to the infused solution has allowed further reduction of the LA concentration with adequate analgesia

• **fentanyl** is commonly combined with bupivacaine,

- a. loading dose - bupivacaine 0.25%
- fentanyl 50 µg
- b. infusion - bupivacaine 0.0625-0.125% + fentanyl 1-3 µg/ml
~ 6-14 ml/hr infusion rate
- c. bolus "top-up's" - bupivacaine 0.125-0.25% 5 ml
- required to re-establish block level with infusion increases

NB: higher concentrations of fentanyl require caution in prolonged labours, Hughes (CJA) recommends bupivacaine 0.125% & fentanyl 1µg/ml

NB: "standard solution" 10 mls bupivacaine 0.5%
 2 mls fentanyl
 50 ml total volume with normal saline
→ **bupivacaine 0.1% & fentanyl 2 µg/ml**

Caudal Anaesthesia

- patients are positioned either prone or lateral, the landmarks are the sacral cornua
- Miller describes doing a PR after placement of the needle to protect against anterior placement of the needle (!?!)
- as for lumbar epidural placement there is an increased risk of intravascular or intrathecal placement with this technique during pregnancy
- the volume required to attain a block to T₁₀ varies from 10-20 ml
- **lumbar** catheter placement may be preferable to caudal placement for the following reasons,
 1. T₁₀-L₁ analgesia is easily obtained early in labour, when sacral analgesia is not required
 2. a lower total dose of anaesthetic is required
 3. pelvic muscles tend to retain their tone and rotation of the foetal head is more easily accomplished
 4. despite the increased risk of dural puncture, lumbar placement is frequently technically easier
- just prior to delivery, or for instrumental delivery, the caudal approach has the advantage of a more rapid onset of perineal anaesthesia and relaxation

Choice of Local Anaesthetic

- ideal properties for a LA during labour include,
 - a. lack of toxicity - mother, foetus or neonate
 - b. effective analgesia / anaesthesia
 - c. duration of action appropriate for use - vaginal delivery
- instrumental delivery
- LUSCS
 - d. minimal muscle relaxation when used for analgesia
 - e. profound muscle relaxation when used for operative procedures
- NB:** clearly these requirements cannot be met by a single agent, therefore a range of anaesthetics are used, according to the situation

■ Neurobehavioural Studies

- high serum levels of local anaesthetic may cross the placenta and cause depression of the neonate
- the factors determining this transfer are,
 - a. maternal & foetal protein binding
 - b. placental blood flow
 - c. solubility of the agent in foetal tissues
 - d. the presence of *foetal acidaemia* → "ion trapping"

NB: at normal concentrations, this is only clinically significant for *mepivacaine*

Scanlon et al 1974

- reported that *lignocaine* was associated with abnormal neonatal neurobehavioural function
- infants of mothers who had received continuous epidural infusions had significantly lower muscle strength & tone cf. a control group
- however, their study did not separate lignocaine and *mepivacaine*
- 2/3 received the later, with umbilical cord levels ~ 3-4x lignocaine
- they still concluded that lignocaine caused adverse effects
- the same group performed a similar study with *bupivacaine* and *chloroprocaine*, and concluded that they had no adverse effects
- subsequent studies have failed to support this accusation

Abboud et al

- compared lignocaine, bupivacaine and chloroprocaine with a control group and found no adverse effects on the *early neonatal neurobehavioural score* (ENNS)

Kileff et al

- LUSCS with bupivacaine 0.5% and lignocaine 2.0% and found *no* differences in the ENNS

■ Bupivacaine Cardiotoxicity

- seizures, presumably from accidental intravascular injection have been associated with cardiac arrests and difficult resuscitation ± death
- Moore & coworkers in the largest clinical survey reported,
 - a. seizures 2° to LA's were frequently associated with *hypoxia* or *acidosis*
 - b. subsequently reported cases of bupivacaine induced convulsions managed with early ventilation with 100% O₂, *without* subsequent cardiac arrest
 - c. 21,000 administrations of bupivacaine, with 23 cases of convulsions not associated with cardiac arrest or neurological sequelae
- studies in awake sheep show that lignocaine and bupivacaine produce similar CNS toxicity when administered rapidly IV
- in the absence of,
 1. hypoxia or hypercarbia
 2. respiratory or metabolic acidosis
 3. hyperkalaemia, or
 4. hypotension→ serious cardiac arrhythmias occurred following bupivacaine, but *not* lignocaine
- subsequent studies have shown bupivacaine cardiotoxicity is enhanced in the presence of hypoxia, hypercarbia or acidosis
- voltage clamp experiments with lignocaine and bupivacaine in guinea pig papillary muscles show,
 - a. both agents block fast inward Na⁺ channels
 - b. both reduce $\delta V/\delta t$ of phase 0 to a similar degree
 - c. lignocaine dissociates from the channel ~ 1 sec
 - d. bupivacaine dissociation takes ~ 5x that of lignocaine
 - e. *frequency-dependent block* accumulates with bupivacaine, even at slow heart rates→ bupivacaine is ~ **16x** as toxic as lignocaine to the myocardium
- NB:** as bupivacaine is ~ 4x as potent as lignocaine, this gives it a *relative toxicity ratio* ~ **4 times**
- the net effect is that bupivacaine is potentially cardiotoxic at ~ 1.0 mg/kg if injected intravascularly
- the majority of cases of toxicity have been reported with 0.75% solution, which is no longer recommended for obstetric use in the USA

■ Chloroprocaine Neurotoxicity

- this is an **ester-linked** LA, with a rapid onset and short duration of action
- because of its rapid hydrolysis in plasma (maternal $t_{1/2} \sim 21s$) very little drug reaches the placenta, and systemic toxicity is low
- there are several case reports of persistent neurological damage, with prolonged sensory or motor deficits after accidental **spinal administration**
- there are a very small number following "uncomplicated" epidural anaesthesia
- there are essentially 2 proposed mechanisms,
 - a. the addition of **sodium bisulphite** and the **low pH** of the solution
→ this is no longer commercially available
 - b. SA injection of large volumes results in increased I-CSF pressure and, combined with systemic hypotension, decreases spinal cord perfusion pressure
- RDM suggests that management of accidental spinal administration of large volumes of LA should be managed, in addition to ABC, with aspiration of as much of the LA containing CSF as is practicable

■ Addition of Adrenaline

- suggested reasons for the addition of adrenaline include,
 - a. decreased rate of systemic **absorption**
 - b. longer **duration** of anaesthesia
 - c. intensification of **motor blockade**
- generally for obstetric use, except for the **test dose**, this is **not warranted** as the total amount of drug used is small and the absence of need for motor blockade
- also, β -mimetic effects of systemically absorbed adrenaline may **decrease** uterine activity and prolong labour
- anaesthesia for LUSCS varies, in that more profound motor blockade is required and the bolus administration of LA is greater

Obstetric Anaesthesia

Spinal Anaesthesia

- may be used for "saddle block" immediately prior to delivery, or for instrumental delivery
- may be used for LUSCS alone, or in conjunction with an epidural technique, with or without added opioid (morphine)
- the incidence of post-spinal headache may be reduced with the use of,
 1. small bore needles (25-26G)
 2. insertion of "cutting" needles with the bevel parallel to the dural fibres
 3. those with a "pencil-point" (Whitacre or Sprott)
- care should be taken not to administer local immediately prior to a contraction, as subsequent a Valsalva may result in excessive cephalad spread

Recommended Dosages - Subarachnoid			
	T ₄ to S ₅	T ₁₀ to S ₅	Duration
Bupivacaine 0.5% heavy	10-12.5 mg 2.0-2.5 ml	6-8 mg 1.2-1.6 ml	80-120 min
Lignocaine 5% heavy	70-90 mg 1.4-1.8 ml	30-50 mg 0.6-1.0 ml	45-75 min

Factors Influencing the Height of Blockade ¹		
Clinically Important	Less Important	Clinically Insignificant
<ul style="list-style-type: none"> • dose (mg) of LA² • baricity of solution³ • position of patient • level of insertion 	<ul style="list-style-type: none"> • patient height⁴ • patient age • configuration of the spinal column • bevel direction Sprott/Whitacre • volume of CSF 	<ul style="list-style-type: none"> • weight • gender • barbotage • speed of injection • CSF - composition <li style="padding-left: 20px;">- circulation <li style="padding-left: 20px;">- pressure • use of vasoconstrictors
¹ Steinstra & Green, Reg Anesth 16:1, 1991 - Study was <i>not</i> in pregnant patients ! ² Russel, Reg Anesth 1991, serial dilutions of 15 mg bupivacaine → same level of anaesthesia ³ Ostheimer, the addition of opioids lowers baricity but <i>does not</i> appear to affect spread ⁴ Norris found no difference using bupivacaine with age, height or BMI Hartwell et al. found no correlation with height, weight, or BMI; but did with the C ₇ -S ₅ distance		

■ Failure to Aspirate CSF

1. needle is not in the CSF
2. small calibre impedes CSF flow
3. CSF pressure may be too low → confirm by aspiration
4. blockage of the bevel by a nerve root → rotate bevel 90° steps
5. a plug of tissue may occlude the needle end
6. needle may be only partially through the posterior dura, or may be against the anterior dura

Contraindications to Central Neuraxis Anaesthesia

- a. patient refusal
- b. infection at the site of needle insertion or of the meninges
- c. coagulopathy
- d. uncorrected hypovolaemia
- e. documented allergy to local anaesthetics
- f. raised intracranial pressure
- g. relative contraindications
 - i. inability to remain still for performance of the block
 - ii. pre-existing spinal/neurological disease
 - iii. high risk of haemorrhage
 - placenta praevia
 - placenta accreta
 - iv. fixed output cardiac disease

Complications - Epidural

- a. hypotension
- b. incomplete / segmental anaesthesia
- c. accidental dural puncture & post-dural puncture headache
- d. backache
- e. total spinal anaesthesia
- f. local anaesthetic toxicity & convulsions
- g. vasopressor induced hypertension
- h. nerve injury
- i. infection / abscess
- j. haematoma

Complications - Spinal

- a. hypotension
- b. excessive spread & high spinal anaesthesia
- c. post-dural puncture headache
- d. incomplete anaesthesia*
- e. nerve injury* **rare* with spinal below L₂
- f. infection*

"Total Spinal" Anaesthesia

- subarachnoid injection of an epidural dose of local anaesthetic will result in,
 - a. unconsciousness
 - b. apnoea
 - c. hypotension & bradycardia
 - d. **dilated** pupils - brainstem hypoperfusion → fixed
- immediate management should include,
 - a. airway maintenance & immediate endotracheal intubation
 + O₂ and IPPV (frequently required for ~ 2-3 hours)
 - b. Trendelenberg position
 - c. IV fluids
 - d. atropine & ephedrine ± catecholamines
 - e. assessment of foetal well-being ± emergent delivery
- the requirement for **sedation** is usually minimal, with most patients having no recall of the events
- more gradual onset of symptoms may be seen with subdural catheter placement, or with catheter migration following initial insertion

■ Vasopressor Induced Hypertension

- usually only a problem with the combination of vasoactive agents and **ergot** derivatives
- especially α-agonists, such as methoxamine / metaraminol / phenylephrine, in conjunction with ergonovine and methylergonovine
- if severe this may require treatment with chlorpromazine, trimethaphan, phentolamine, or SNP

■ Nerve Injury

- direct mechanical injury is extremely rare as pressure on the nerve elicits extreme pain and prompts its withdrawal
- other potential complications such as epidural haematoma, infections (epidural or caudal abscess, meningitis), or chemical contamination of the SA or epidural space are also extremely rare
- nerve injury to the mother is usually not related to anaesthesia but to,
 - a. compression of the lumbrosacral trunk between the foetal head and the sacrum
 - b. kinking or compression of the,
 - i. femoral nerve
 - ii. lateral femoral cutaneous nerve, or
 - iii. common peroneal nerve

* usually related to the *lithotomy* position
- recovery from these compressive neuropathies usually requires **12-16 weeks**

Post-Dural Puncture Headache

■ Clinical Features

- postural headache, often severe, relieved upon assuming the supine position
- aggravated by coughing, straining, or sudden movements
- may have associated **reflex spasm** of the cervical muscles may result in neck pain, or ocular or auditory dysfunction
- the average time of onset is within the first **48 hours** post-puncture
- duration ranges from hours to months (untreated), however most resolve after ~ 4 days

■ Differential Diagnosis

- i. simple headache
- ii. meningitis
- iii. CVA - subdural or subarachnoid haemorrhage
- infarction (cortical vein thrombosis)
- iv. hypertensive crisis
- v. migraine
- vi. metabolic disturbance
- vii. psychogenic * post-partum

■ Pathophysiology

- a. **CSF loss**
 - epidural space pressure ~ 0 erect or supine → gradient ~ 40-50 cmH₂O erect
 - multiple studies support the correlation between reduction in CSF **volume** & PDPH
 - downward traction on dural sinuses, vessels, nerves and the tentorium
 - this results in referred pain,
 - i. above the tentorium → **trigeminal nerve** → the front of the head
 - ii. below the tentorium → cranial nn. IX & X → the occiput
 - iii. pain in the upper neck & shoulders is referred by C₁₋₂₋₃
 - b. **reflex cerebral vasodilatation** (Monroe-Kelly effect)
 - stimulates perivascular stretch receptors, similar to migraine
- certain populations are at greater risk for PDPH, these include,
 - a. younger patients
 - b. females * excluding the obstetric population
 - c. pregnant or post-partum ~ 2x increase due to,
 - i. repeated Valsalva manoeuvres during labour
 - ii. intravascular volume depletion
 - dehydration during labour
 - blood loss with delivery
 - post-partum diuresis
 - the incidence is remarkably lower in the > 60 age group, possible reasons being,
 - a. lower CSF pressure & decreased CSF circulation
 - b. cerebral vessels less reactive to volume change
 - c. alteration of connective tissue composition with age
 - ? earlier closure of the dural defect
 - factors influencing the incidence of PDPH include,
 - a. use of small calibre needles
 - b. alignment of the needle bevel with the dural fibres
 - c. angle of penetration of the dura * midline > lateral
 - d. bevel design * Quincke > Whitacre
 - the incidence of headache is directly related to the needle gauge & type, (Ostheimer)
 - a. Epidural tap ~ 60-70%
 - b. Quincke 26G ~ 6.4%
 - c. Quincke 27G ~ 2.7%
 - d. Whitacre 25G ~ **1.3%** ?? Sprott

■ Needle Diameter

- many studies have correlated needle diameter & PDPH
- Phillips *et al.* (1968) 10,440 subarachnoid anaesthetics, 25/26G, with an incidence of ~ 3.5%
- Vandam & Dripps (1956) in a similar size study, 82% were 20/22G, overall incidence ~ 11%

■ Bevel Direction

- Green (1926) postulated that aligning the bevel parallel to the dural fibres would result in a smaller defect & therefore reduce the CSF leak
- Franksson & Gordh (1946) confirmed this by autopsy studies, showing ~ 2x the number of severed fibres with perpendicular entry
- Mihic (1985) confirmed this in a clinical study, with respective incidences of PDPH of 16.1 versus 0.24%
- some suggest identification of the *epidural space* in this manner in case accidental dural puncture occurs, however, rotation of the needle is then required

■ Bevel Design

- as an extension of the above, conical point needles which result in minimal cutting of the dura appear to be associated with a lower incidence

■ Needle Angle

- Hatfalvi (1977) reported 600 cases with a 20G needle and a zero incidence of PDPH
- he attributed this low incidence to the *paramedian approach*
- in vitro studies of human dura show that puncture at 30° results in less CSF loss cf. 60-90°

■ Patient Position

- an overly flexed position may result in an extended dural tear due to tension on the dura
- several studies with patients in "a comfortable, neutral position" have shown a significantly reduced frequency of PDPH

■ Agents Used

- Vandam & Dripps (1956) concluded that the LA used had no relationship to PDPH incidence
- this is still generally accepted, however, a recent review suggested that certain agents may be associated with an increased incidence,

→ lignocaine > bupivacaine > tetracaine/procaine

- other workers have noticed a *lower* incidence with the addition of fentanyl or adrenaline

■ Continuous Subarachnoid Anaesthesia

- most studies suggest that the incidence may actually be quite low, despite the use of large catheters (18G)
- possible reasons include occlusion of the dural rent by the catheter during use, and stimulation of an inflammatory reaction which results in earlier closure of the dura
- the use of microcatheters (32G) may reduce the incidence further

■ Management

- the incidence of unintentional dural puncture during *epidural* is ~ 1-2%, with ~ **70%** PDPH
- when unintentional dural puncture occurs, the needle/catheter should be resited at an adjacent space, with the catheter directed **away** from the punctured space
- establishment of epidural blockade at a different level may be associated with a lower incidence of PDPH, presumably due to raised epidural pressure and decreased loss of CSF
- if LOR to *air* is being used, the volume injected should be minimal, as accidental subarachnoid introduction and pneumatoencephalocele will produce a severe headache
- the subsequent establishment of blockade should be carefully **fractionated**

- **conservative management** is appropriate as PDPH is usually self-limiting, and often only mild to moderate in intensity
 - a. **bed rest**
 - having the patient lie flat will **not** prevent PDPH and is **no longer** recommended
 - this may actually increase the incidence of nausea, delay the diagnosis of PDPH and lead to thromboembolic disease
 - it does have a role **after** the diagnosis of PDPH is made
 - b. adequate fluid hydration
 - c. simple analgesics
 - d. abdominal binders & posture changes to increase venous return are poorly tolerated and generally ineffective

- **caffeine** has been suggested by some workers to decrease the incidence and severity of PDPH
- the mechanism of action is in reducing vasodilatation, as in the management of migraine

- **saline infusion** (40-60 ml) will reduce the severity of PDPH, however the increase in epidural pressure is usually transient and the headache frequently recurs

- **autologous blood patch** is indicated for severe headache or failed conservative management
- first described by Gormley (1960) and has a success rate > 90%
- failure of ABP and performance of a second procedure has a higher success rate ~ 95%
- under aseptic conditions, ~ 15 ml of blood is removed by venipuncture and placed immediately into the epidural space, preferably at the level of the puncture
- QVH, part of the blood sample is sent for culture
- if the site of the leak is uncertain, due to multiple punctures, then the **lowermost** space should be used, due to the tendency to cephalad spread
- **backache** may occur, but is usually mild and limited to ~ 48 hours
- no cases of infection, arachnoiditis, or cauda equina syndrome have been reported
- epidural anaesthesia performed at a later date may be associated with a higher incidence of incomplete block or missed segments, though, there are no controlled studies to support this
- the use of prophylactic blood patch is controversial
- older studies showed that prophylactic or early ABP (< 24 hours) **did not** decrease the incidence
- however, more recent studies have found administration of 15-20 ml through the catheter to be highly effective in reducing the incidence of headache
- however, no large scale study has demonstrated the efficacy of prophylactic ABP

Epidural & Intrathecal Narcotics

- spinal opioids vary from local anaesthetic agents, due to,
 - a. the lipid solubility of the agent
 - rate of onset and duration of analgesia
 - degree of cephalad spread, segmental nature
 - b. the absence of sympathectomy
 - c. the absence of motor blockade
 - "selective spinal analgesia"
 - d. side effects
 - nausea & vomiting
 - drowsiness, dizziness
 - pruritis
 - urinary retention
 - respiratory depression

NB: all side effects can be reversed by *naloxone*

- due to the lack of haemodynamic and motor effects, there was initial enthusiasm for their use in labour
- this has largely diminished due to,
 - a. inadequate *analgesia* for instrumental delivery/episiotomy
 - b. frequent occurrence of the listed *side effects*
 - c. the requirement for special *monitoring* of the mother

- the lipid soluble agents provide good analgesia following LUSCS, but this is usually of short duration ~ 3-5 hours
- intrathecal morphine 0.1-0.25 mg, or epidural morphine 5 mg, administered at the time of delivery may provide analgesia for up to 24 hours
- pruritis, nausea and vomiting occur frequently but are easily treated with naloxone 0.2-0.4 mg, *without* significant loss of the analgesic effect
- respiratory depression is a rare complication at these doses, but does require the mother be monitored for 24 hours

Effects of Epidural Anaesthesia on Labour

- in the very early stages, **latent labour**, any analgesia may slow the progression of labour
- studies of established **first stage** and the rate of cervical dilatation show no significant effect
- several studies have shown short-term (< 30-60 minutes) inhibition of uterine activity, principally associated with the use of **adrenaline** containing solutions
- the **second stage** of labour is prolonged, possibly due to,
 - a. loss of the "bearing-down" reflex
 - b. loss of pelvic tone and delayed rotation of the foetal head
 - c. generation of lower intra-abdominal pressures
- the American College of Obstetrics and Gynecology guidelines for **second stage** are,
 1. nulliparous < 2 hours without regional anaesthesia
< 3 hours with a regional anaesthetic
 2. multiparous < 1 hours without regional anaesthesia
< 2 hours with a regional anaesthetic

NB: these are guidelines only, and individual cases should be managed according to assessment of **foetal well-being**

■ Operative Deliveries

- cf. vaginal delivery, **LUSCS** is associated with an increase in maternal **mortality ~ 2-4 fold**
 - a. a half of these are due to complications of the **operative procedure**
 - b. the remainder to **obstetric complications** leading to operative delivery
- maternal **morbidity** is significantly increased, ranging from ~ **10-80%**
- Amirikia *et al.* (Am J. O&G 1981), 9718 cases of LUSCS with ~ 28% morbidity, most commonly,
 - i. endometritis
 - ii. UTI
 - iii. anaemia
 - iv. wound infection
 - v. ? fever
- the majority of studies show no adverse effects on acid-base, Apgar scores, or later IQ testing for **low-cavity forceps** delivery
- there are conflicting reports about mid-cavity deliveries, the consensus being that they may still be applied judiciously in select circumstances by experienced operators
- comparing the data from multiple trials, there are 3 trends,
 1. large difference between frequency of operative intervention *6-93%
 2. women having epidurals have between **2-10 fold** increase in operative delivery
 3. **primiparous** women have a higher incidence irrespective of regional anaesthesia
 - since more primips have LEA, studies not differentiating are hard to assess !

■ Factors Supporting Increased Incidence

- a. prolongation of second stage
 - i. inability to feel contractions and loss of urge to push
 - ii. muscle weakness associated with anaesthesia
- b. perineal and pelvic muscle relaxation
 - i. instrumental delivery is better tolerated and easier to perform or teach
 - ii. increased incidence of malpresentation due to relaxation of pelvic "sling"

- several studies showing increased incidence of OP presentation, up to 3 fold
- possibly 2° to muscle relaxation, or to selection bias, more OP's requesting analgesia due to greater pain
- many of the older studies were with bupivacaine 0.5% and profound motor blockade
- numerous studies showing **no increase** with the use of lower concentrations of LA
- other prospective studies do show an increased incidence but no adverse neonatal effects

NB: Thorpe, (Kansas, 1993), randomised controlled trial with some minor limitations, showed a 3x increase in operative delivery with epidural use, bupivacaine 0.25%

■ Factors Opposing Increased Incidence

- several institutions have shown a steadily decreasing incidence of operative delivery, while the incidence of LEA is steadily rising
- O'Driscoll (Dublin), increase incidence of LEA from 5% to 40% with **no increase** in operative delivery, however, no other institutions have come close to this institutions figures and they certainly **do not** apply to practice in Australia (especially QVH)
- in some women experience severe pelvic & perineal pain and are unable to push effectively, but may do so with the conduction of LEA
- similarly, some women with incoordinate uterine action may be improved by LEA, possibly due to the reduction in circulating catecholamines
- Philipsen & Jensen (1989) in a randomised, controlled trial with pethidine versus LEA, showed no difference between the 2 groups with respect to,

1. first or second stage duration
2. incidence of instrumental delivery

NB: however, LEA provided better pain relief

- studies assessing the effectiveness of allowing the epidural to wear-off for second stage show no significant decrease in the incidence of operative delivery
- one study by Chestnut *et al.* actually showed a statistically significant increased incidence

■ Factors Associated with Instrumental Delivery

1. institution and obstetrician preference / convenience
2. unresolved foetal distress
3. failure to progress, prolonged second stage, "maternal exhaustion"
4. teaching hospital practice

General Anaesthesia

- most common reason for GA during vaginal delivery is the requirement for *uterine relaxation*,
 - a. intrauterine manipulation for internal podalic version
 - b. complete breech extraction
 - c. tetanic uterine contraction during breech delivery, prior to delivery of the head
 - d. manual removal of the placenta
 - e. replacement of an inverted uterus
 - f. instrumental delivery in 2^o stage
 - i. with acute foetal distress, where no epidural catheter is in place
 - ii. when regional is contraindicated
- most texts state that immediately following manipulation and delivery, volatile agents should be ceased and oxytocic agent administered
- this is essentially outdated, 0.5 MAC of volatile agent will not increase the incidence of PPH and will significantly reduce the incidence of awareness

ANAESTHESIA FOR CAESAREAN SECTION

- the choice of anaesthesia for LUSCS depends upon,
 - a. the reason for the operation
 - b. the degree of urgency of the operation
 - c. the preference of the patient
 - d. the preference of the anaesthetist
 - e. the preference of the surgeon

- the advantages of **regional anaesthesia** include,
 - a. an awake mother
 - b. minimal risk of aspiration
 - c. minimal neonatal drug effects
 - d. decreased maternal blood loss
 - e. ?? lower incidence of DVT
 - f. with catheter techniques
 - titration to effect
 - postoperative pain relief

- the advantages of **general anaesthesia** include,
 - a. more rapid induction
 - b. less associated hypotension & cardiovascular instability
 - c. better control of the airway and ventilation

Regional Anaesthesia for Caesarean Section

1. Routine history, examination, investigation and consent.
Careful explanation to patient & partner.
2. Premedication with non-particulate oral antacid within 1 hour of surgery.
3. Check anaesthetic machine in accordance with Faculty guidelines.
Draw-up emergency drugs prior to arrival of the patient.
Check intubation trolley for appropriate equipment, ie. difficult intubation.
4. Maintain lateral tilt & left uterine displacement prior to positioning for blockade.
Check baseline haemodynamic observations.
5. Establish IV access and commence volume loading with crystalloid/colloid.
 - if using spinal anaesthesia, then add **ephedrine 30 mg** to 1000 ml crystalloid
6. Administer supplemental oxygen via face mask.

7. Spinal Blockade

- i. use the smallest calibre needle possible, preferably "pencil-point"
- ii. local anaesthetic - **bupivacaine 0.5%** ~ **2.0-2.5 ml** (10-12.5 mg)
- lignocaine 5% ~ 1.4-1.8 ml (70-90 mg)
- iii. optional opioid - **morphine** ~ **0.1-0.25 mg**
- fentanyl ~ 10-25 µg

8. Epidural Blockade

- i. local anaesthetic - lignocaine 2% + adrenaline
- bupivacaine 0.5%
 - ii. optional opioid - fentanyl ~ 50-100 µg
- morphine ~ 5 mg (following delivery)
 - iii. optional pH adjustment ~ 1 ml of bicarbonate to 10 ml lignocaine
~ 0.1 ml of bicarbonate to 10 ml bupivacaine
 - iv. **adrenaline** may be added to a maximum of 1:200,000
 - v. **test dose** ~ 3-4 ml with adrenaline 1:200,000
- observe closely for 3-5 minutes re SA/IV administration
 - vi. 5 ml fractional administration to ~ 20 ml, titrate to the T₄ dermatome
* initial administration may be either through the needle or the catheter
 - vii. supplement with ~ ½ the original dose at 30 minutes
9. Maintain lateral tilt & left uterine displacement prior to delivery.
10. Monitor SpO₂, ECG and patient comfort.
Check HR/BP frequently (~1-3 min), then ~ 5 minutely following delivery.
Treat **hypotension** (> 30% fall or systolic < 100 mmHg) promptly:
- i. ensure left uterine displacement
 - ii. IVT crystalloid or colloid (blood transfusion if required)
 - iii. ephedrine ~ 5-10 mg boluses, repeated q5m prn
 - iv. metaraminol ~ 0.5-1.0 mg boluses, repeated q5m prn
11. Administer **syntocinon 10^U** with delivery of the infant.
12. Manage overt anxiety, or patchy blockade with,
- i. reassurance & explanation to the patient
 - ii. fentanyl ~ 20-40 µg prn
 - iii. ketamine ~ 0.25 mg/kg
 - iv. midazolam ~ 0.5-1.0 mg prn
13. If analgesia is inadequate, then proceed to GA with standard precautions.
14. If supplemental analgesia is required following delivery of the infant, then administer,
- i. supplemental spinal/epidural opioid
 - ii. epidural bupivacaine 0.125-0.25% with or without opioid
15. If spinal or epidural morphine has been administered, then admit to a high dependency nursing area for 24 hours.

General Anaesthesia for Caesarean Section

• GA is the technique of choice for LUSCS in the following circumstances,

1. RA is *contraindicated*
 - i. patient refusal
 - ii. documented allergy to local anaesthetics
 - iii. uncorrected maternal hypovolaemia, or severe hypotension
 - iv. coagulopathy
 - v. septicaemia, local sepsis
 - vi. acute neurological disease - infection, inflammation, demyelination, ↑ ICP
 - vii. severe anaemia
 - viii. afterload / preload dependent CVS disorders
2. acute foetal distress, no epidural in place
3. maternal preference following informed discussion

NB: at the Briham & Women's hospital, the number of deliveries under GA has decreased from 24% in 1982-83 to 8% in 1989-90

■ Preoperative Medications

- i. antacid - 30 ml *non-particulate*, 30-60 minutes pre-induction
- ii. H₂-blockers - orally the night before, or
- orally or IV 1 hour prior to induction
- iii. metoclopramide - may be beneficial in the setting of a recent meal
- iv. anticholinergics - not routinely required
 - *glycopyrrolate* ~ 0.1-0.2 mg is preferable as doesn't cross the placenta
- v. sedatives - avoided due to the risk of neonatal depression

■ Induction Agents

- i. thiopentone
 - rapidly crosses the placenta
 - peak UV within 1 minute of maternal peak plasma
 - ≤ 4 mg/kg pregnant body weight (PBW)
 - foetal brain levels low due to hepatic ER & dilution
 - * **no** advantage in delaying delivery to allow redistribution
- ii. ketamine
 - generally not used due to psychomimetic properties
 - first choice in severe asthma or hypotension/hypovolaemia
 - ≤ 1 mg/kg_(PBW) there are no adverse foetal effects
- iii. relaxants
 - minimal placental transfer in normal doses
 - normal metabolism of succinylcholine
- iv. inhalational agents
 - N₂O transfer is time dependent, ≤ 50% for ≤ 15 min prior to delivery OK
 - volatile agents at ≤ 0.7 MAC don't cause excessive sedation or increase bleeding

ASPIRATION PNEUMONITIS

- John Hunter provided the first scientific description, while giving evidence at a murder trial in 1781 (brandy & cats)
- James Simpson (1848) also indicted aspiration, rather than chloroform, as the cause of the first anaesthetic death of Hannah Greener
- Winternitz (1920) first described *acid aspiration* and Hall (1940) associated this with obstetrics
- *Curtis and Mendelson* (1946) described,
 1. 66 cases of gastric aspiration in obstetric patients undergoing GA for vaginal delivery
 2. an experimental aspiration syndrome in rabbits with acidic versus neutral fluids
 3. the development of pulmonary oedema and CXR changes
 4. the beneficial effects of neutralisation of the stomach contents
- the true *incidence* is difficult to determine, as not all cases are diagnosed and/or reported
- further, the incidence is small, therefore multicentre data is required
- reported figures include,
 - a. all anaesthetic deaths ~ 1-20%
 - b. anaesthesia related deaths ~ 0.008-0.2 per 1,000 cases
 - c. "silent" regurgitation ~ 4-26% of all general surgical cases
subsequent aspiration ~ 10-20% of these
 - d. Olsson *et al.* (1986) - 185,358 anaesthetics
- 38 cases of aspiration
~ 4.7 : 10,000 → **1 : 2,131**
~ **15 per 10,000 obstetric patients**
 - e. subsequent *mortality* - reported ranges of 3-70%
~ **5%** is the most recent & reliable figure

Physiology

- swallowing occurs in **3 stages**,
 - a. voluntary - movement of food into the pharynx by the tongue
 - b. pharyngeal - stimulation of receptor areas around the pharynx
- upward movement of the soft palate to protect the nares
- medial movement of the palatopharyngeal folds
- covering of the laryngeal inlet by the epiglottis
- upward and anterior movement of the larynx
 - c. oesophageal - relaxation of the upper oesophageal sphincter
- contraction of the superior constrictor muscles of the pharynx
- oesophageal peristalsis & relaxation of the LOS
- neural centres are located in the reticular substance of the medulla and lower pons
- sensory input travels in the trigeminal and glossopharyngeal nerves, motor via V, IX, X, XII

Obstetric Anaesthesia

- the *stomach* performs 3 functions,
 1. storage
 2. mixing with digestive juices
 3. propulsion
- it normally accommodates 1000-1500 ml of food, but may contain up to 6000 ml
- contractions occur ~ 20 seconds, with periodic stronger contractions responsible for emptying
- a multitude of factors *delay* the rate of gastric emptying,
 - a. hyper/hypo-osmolar contents
 - b. high calorie solids > low calorie solids > liquids
 - c. acid within the duodenum & cholecystokinin
 - cf. gastrin, motilin and parasympathetic agents which increase emptying
 - d. pain, anxiety, opioids and labour
 - e. disease states, ie. inflammatory bowel disease, diabetes, hypothyroidism, peptic ulcer disease, electrolyte disorders, etc.
- usually, in a calm patient, fluids pass into the duodenum within 2 hours and solids 4-6 hours
- *gastric secretion* continues to add volume to the gastric contents, ≤ 200 ml/hr,
 - a. interdigestive phase ~ 1 ml/min
 - b. digestive phase ~ 3-4 ml/min

Def'n: *regurgitation* is the process whereby gastric contents *passively* flow through the gastro-oesophageal sphincter (lower oesophageal sphincter, LOS) into the oesophagus and the pharynx

- when the larynx is incompetent then "silent" *aspiration* can then occur
- the pressure at the LOS is usually greater than intragastric pressure, preventing regurgitation
- the mechanism of action is multifactorial,
 - a. anatomical sphincter - doesn't exist *per se*, muscle is similar above & below
 - b. physiological sphincter - tonic contraction of the circular smooth muscle fibres
 - c. flap valve[§]
 - d. diaphragmatic action[§]
 - e. mucosal valve[§] [§]these act in unison
 - f. mechanical factors - lowermost 2-3 cm of the oesophagus are intra-abdominal
- despite these factors reflux does occur, but is rarely seen at intragastric pressures < 20 cmH₂O
- both (f) and reflex rises in sphincter tone act to prevent reflux during coughing/straining, when intragastric pressures may exceed 60 cmH₂O
- this reactive increase also accompanies the increases in pressure seen with suxamethonium

Obstetric Anaesthesia

- numerous drugs affect the competency of the LOS,
 - a. **decrease** LOS tone
 - i. anticholinergics: atropine, scopolamine, glycopyrrolate
 - ii. opioids: morphine, pethidine, fentanyl
 - iii. benzodiazepines: diazepam, midazolam
 - b. **increase** LOS tone
 - i. gastrokinetics: metoclopramide, ? cissapride
 - ii. hormones: motilin, gastrin, PGE₂
 - iii. acetylcholinesterase inhibitors
 - iv. increased **alkalinity** of gastric contents (MCQ)

Def'n: **vomiting** is the reflex action by which intragastric contents are **actively** expelled from the stomach, through the oesophagus and oropharynx, involving the use of both voluntary and involuntary muscles

- the **vomiting centre** in the medulla receives input from,
 - a. the CTZ in the area postrema
 - b. the vestibular & olfactory apparatus, and other cortical areas
 - c. almost the entirety of the GIT via the vagus nerve
- motor responses are mediated via,
 - a. the cranial nerves V, VII, IX, XII
 - b. phrenic and spinal motor nerves to the diaphragm and abdominal muscles
- the usual sequence of events in vomiting may include,
 - a. prodromal tachycardia, sweating, tachypnoea, salivation and a sensation of nausea
 - b. elevation of the hyoid bone & larynx, opening the crico-oesophageal sphincter
 - c. closure of the glottis
 - d. elevation of the soft palate, closing the posterior nares
 - e. respiration is held in *mid-inspiration*
 - f. simultaneous strong downward contraction of the diaphragm & abdominal muscles
→ rapid elevation of intragastric pressure
 - g. relaxation of the LOS & reverse peristalsis commences, allowing expulsion of the gastric contents

Risk Factors

1. depressed level of *consciousness*
 - hypotension, hypoxia, hypercapnia
 - metabolic encephalopathy, coma
 - ETOH, drug overdosage, *anaesthesia*
 - CVA
 - epilepsy
 - trauma
 - cardiac arrest
 2. impaired *airway reflexes*
 - *drugs* (CNS, NMJ, local anaesthesia)
 - *intubation/extubation*
 - motor neurone disease
 - CVA
 - elderly
 - local disease, post-surgical
 3. increased *regurgitation*
 - *pregnancy*
 - hiatus hernia
 - obesity
 - bowel obstruction
 - NG tube
 - oesophageal disease
 - LOS dysfunction
(scleroderma, oesophageal achalasia)
- an *at risk* has been defined as a patient having,
 1. a gastric volume > **0.4 ml/kg** ~ 25 ml
 2. a gastric pH < **2.5**
 - these limits were based on a paper by *Roberts and Shirley (1974)*, who used unpublished data from Rhesus monkeys
 - more recent work by *Raidoo (1988)* found that a much greater volume (~ 0.8 ml/kg) was required to produce classic Mendelson's syndrome
 - further, these animal models involve the *direct installation* of acid into the lungs, whereas clinical aspiration is almost never associated with the aspiration of the entire stomach contents
 - subsequent studies of non-lethal aspiration support the concept that *pH* is the more important factor

Obstetric Anaesthesia

- **pregnancy** is unique among the risk factors for aspiration as the risk is **multifactorial**,
 1. delayed gastric **emptying**
 - displacement of the pylorus
 - increased progesterone (antagonises *motilin*)[§]
 - pain, anxiety, narcotic analgesics
 2. increased gastric **acidity**
 - placental **gastrin** secretion
 3. decreased **LOS tone**
 - anticholinergics (atropine, glycopyrrolate)
 - narcotics
 - ?? loss of the cardio-oesophageal angle
 4. increased **intra gastric pressure**[§]
 - mechanical effects of the uterus (some have found an increase in the 1st trimester)
 - lithotomy

NB: **barrier pressure** (gastric - oesophageal) is normal, except in those who experience "heartburn"

- there is argument concerning at what stage these changes occur [§]
- some workers have shown changes starting in early pregnancy, whereas others have found no change until ~ 34 weeks
- there is equal lack of agreement as to when the risk decreases following delivery
- numerous studies showing up to 75% of patients having gastric contents > 0.4 ml/kg & pH < 2.5, up to **48 hrs** post-delivery
- however, this is no different from the general surgical population & the definition of "at risk" requires further clarification

NB: however, aspiration remains the major cause of maternal morbidity and mortality

Prevention

1. nil orally
 - some would argue this increases gastric volume/acidity
 - prevention of prolonged fasting with regular fluid intake
2. non-particulate antacids
3. H₂ blocking agents
4. anticholinergic agents
 - however, these reduce tone of the LOS
5. metoclopramide
6. head-up position
7. avoid general anaesthesia
8. rapid sequence induction of anaesthesia
9. cricoid pressure
10. endotracheal intubation with a cuffed ETT
11. awake extubation
12. alert, well trained recovery room staff

Obstetric Anaesthesia

- **nil orally** has traditionally meant 6 hours for solids and 4 hours for fluids
- there are a number of recent articles which challenge this philosophy,
 - a. Miller *et al.* - BJA 1983
 - light breakfast 2-3 hours preoperatively (coffee & toast)
 - * no difference in gastric volume/pH from overnight fast
 - not done in **pregnant** patients
 - b. Maltby *et al.* - CJA 1987, 1988, 1988
 - 100 ml of water, OJ, or coffee 2-3 hours preoperatively
 - no difference in gastric volume/pH versus overnight fasting
 - * a significant percentage "at risk" in both groups
 - risk considerably reduced if given oral **ranitidine** with fluids
- while it is reasonable to allow elective surgical patients fluids within 2-4 hours of anaesthesia, solids should be withheld due to the far greater damage caused from **particulate aspiration**, regardless of pH
- whether this liberation should be extended to emergency patients has not been defined, but would seem unwise on theoretical grounds
- Lewis and Crawford (BJA 1987) showed that this is **not appropriate** for obstetric patients,
 - a. 3 groups - controls, tea, and tea plus toast
 - b. residual gastric volumes- 33, 65 and 73 ml respectively
- however, all of these studies have used **NG tube aspiration** as the means of gastric volume measurement
- there is good evidence that patients may have large residual volumes following aspiration, and this is the reason prophylactic aspiration is not routinely recommended
- routine administration of **antacid** significantly raises the gastric pH, however does not decrease the risk of aspiration of **particulate** matter
- most antacids are themselves suspensions of particulate matter and will result in a chemical pneumonitis if inhaled
- **non-particulate** antacids, such as **sodium citrate** are therefore preferable
- administration throughout labour is not warranted, as they exert their effect within 15-20 minutes
- sodium citrate is effective for 1-3 hours
- administration of **histamine H₂ blockers** (cimetidine, ranitidine & famotidine) will reduce gastric acidity and volume prior to anaesthesia require, however these require,
 - a. 1-2 hours orally
 - b. 45-60 minutes IV/IM
- the half-life of cimetidine ~ 2 hours and it is excreted by the kidney within 24 hours
- the half-life increases to ~ 3-4 hours in renal failure & the dose should be reduced
- **ranitidine** has a duration of action ~ 8 hours, and it is equally effective as cimetidine in increasing gastric pH and reducing volume

Obstetric Anaesthesia

- there have been numerous studies comparing dosages and routes of administration for both of these agents,
 - a. both are equally effective when given orally h.s./a.m. prior to elective surgery
 - b. there is no need for IV/IM administration in elective surgery
 - c. single dose R_x with cimetidine
 - 7.5 mg/kg effective in 95% of children
 - 10 mg/kg effective in 100% of children
 - d. both are more effective than anticholinergic agents, or clear antacids; cimetidine/antacid is *less* effective as the absorption of cimetidine is impaired
 - e. the effect on volume may be less than the effect upon pH
 - f. neither agent affect contents already in the stomach; ie., clear antacids may be preferable in emergency situations
- **complications** with the use of H_2 -receptor blockers include,
 - a. bradycardia, hypotension & rarely cardiac arrest with rapid IV administration (mainly in seriously ill patients & cimetidine >> ranitidine)
 - b. potentiation of H_1 -receptor effects in *asthmatic* patients, especially those taking theophylline
 - c. rare reactions
 - dry mouth
 - gynaecomastia
 - diarrhoea
 - d. altered drug metabolism & elimination;
 - i. inhibition of cytochrome P_{450}
 - *both agents, but cimetidine >> ranitidine
 - ii. interference with GIT absorption
 - raised gastric pH delays absorption of weak bases
 - iii. competition for renal excretion
 - both actively excreted by PRT, therefore compete with other drugs
 - procainamide and flecanide
 - iv. altered plasma protein & tissue binding
 - cimetidine decreases the V_{dss} of labetalol, lignocaine, imipramine & pethidine
 - unknown whether tissue or plasma binding is responsible
 - however, plasma protein binding of cimetidine is ~ 20%
 - v. decreased hepatic blood flow
 - suggested by earlier studies using indocyanine uptake which is inaccurate
 - later studies indicate *no* substantial change in hepatic blood flow
- most of the above work has been pharmacokinetically and not clinically based
- therefore it is recommended to monitor only those drugs which have a narrow therapeutic margin, such as warfarin, theophylline and phenytoin

- H₂-receptor blocker/drug interactions of note include,
 1. **local anaesthetics**
 - most studies are of ***lignocaine*** IV as an antiarrhythmic agent
 - cimetidine may decrease lignocaine clearance ~ 30-40%, with increases in plasma concentration of up to 70%
 - studies using lignocaine as a regional LA (epidural) have been equivocal
 - ranitidine may be safer in these circumstances
 - studies on the effects on bupivacaine are also equivocal
 2. **suxamethonium**
 - cimetidine may prolong the duration of action (Kabam *et al.* - from a mean ~ 8.6 to ~ 20 minutes)
 - this effect has not been reproduced by other workers with either agent
 - plasma cholinesterase activity has been shown to be unaffected by either agent
 - the original study also used ***metoclopramide*** & there is now some data to suggest that this may decrease plasma cholinesterase activity ??
 3. ***b*-blockers**
 - propranolol & metoprolol clearance is decreased by cimetidine but not ranitidine
 4. **Ca⁺⁺ channel blockers**
 - cimetidine enhances the plasma concentrations of nifedipine and diltiazem following oral administration
 - no effect seen with ranitidine
 5. ***theophylline***
 - cimetidine decreases the plasma clearance but ranitidine does not
 - however, there have been case reports of significantly elevated plasma levels after administration of ranitidine
 - one of the few interactions which has been shown to be ***clinically significant***
 6. ***warfarin & phenytoin***
 - effects are similar to those for theophylline, ie. cimetidine >> ranitidine
 - again there have been clinically significant case reports
 7. ***benzodiazepines***
 - cimetidine decreases the plasma clearance of diazepam but ranitidine does not
 - elevated plasma levels of midazolam have been reported with both agents, though, no alteration of clearance was observed

- the H₂-blockers are not routinely indicated in ***obstetric practice***, except where the mother has a history of,
 - a. severe reflux - actually a large percentage of women
 - b. morbid obesity
 - c. peptic ulcer disease
 - d. day cases for VTOP

Obstetric Anaesthesia

- **metoclopramide** acts both centrally and peripherally to increase gastric emptying, increase the tone of the LOS, and act as an antiemetic,
 - a. centrally it antagonises the effects of **dopamine**
 - b. peripherally it stimulates the release of **ACh**,
 - i. increasing gastric and SI motility
 - ii. increasing tone of the LOS
 - iii. decreasing tone of the pylorus and duodenum
- its efficacy, either as a sole agent, or in combination with a H₂ blocker is questionable
- studies have shown variably either no effect, or a synergistic decrease in gastric volume
- no adverse foetal or neonatal effects have been described in the small number of studies done
- **tardive dyskinesia** and parkinsonian symptoms have occurred in the elderly, and in the young given large doses
- providing there are no obvious indicators that intubation will be difficult, **rapid sequence induction**, is the technique of choice for induction of general anaesthesia;
 - a. denitrogenation with 100% O₂ for 3 minutes, or at least 4 VC breaths (Gold *et al.*)
 - b. administration of a sleep dose of thiopentone ~ 4 mg/kg
 - c. application of cricoid pressure **as** consciousness is lost, until inflation of the ETT cuff and confirmation of endotracheal placement
 - d. rapid establishment of paralysis,
 - i. suxamethonium 1.5 mg/kg, unless contraindicated
 - ii. vecuronium or atracurium using the "priming principle"
 - e. endotracheal intubation & **confirmation**,
 - i. direct vision if possible
 - ii. ETCO₂ for ³ **6 breaths**
 - iii. breath sounds & chest movement * unreliable
 - iv. SpO₂ * changes occur too late
- **cricoid pressure** (Sellick's manoeuvre) should always be employed and maintained until endotracheal intubation is **confirmed**
- maternal mortality studies in Britain indicated that in 11 cases of maternal mortality due to aspiration, CP had been applied incorrectly, or had been discontinued prior to intubation
- correctly applied, CP should prevent virtually all cases of regurgitation/aspiration, as it will withstand oesophageal pressures of up to ~ 100 cmH₂O
- the use of IPPV prior to endotracheal intubation should be **avoided** as this may result in inflation of the stomach and an increased risk of regurgitation
- if intubation is not possible, then CP should be continued throughout assisted ventilation
- some state CP should be removed if the patient vomits due to the risk of oesophageal rupture
- **vomiting** does not occur in the paralysed patient & rupture is exceedingly unlikely
- silent aspiration can still occur around the ETT cuff, but the volume is usually small
- extubation should be performed awake, when the patient has control over laryngeal reflexes

FAILED ENDOTRACHEAL INTUBATION

- this is the leading cause of anaesthetic related maternal **mortality**
- in Australia, Holland (AIC, 1984) ~ **69%** of anaesthesia related deaths were airway catastrophes
- the Confidential Enquiry into Perioperative Deaths (UK 1987) found that 1 in 3 deaths attributable solely to anaesthesia was due to failure to intubate the larynx
- however, this report did not include obstetric patients, in whom the **incidence** of failed intubation is much higher,
 - a. general surgical population ~ **1 : 2,303**
~ 0.04%
 - b. obstetric population ~ **1 : 300** (~ 8x ↑ risk)
~ 0.33%
- reports of Confidential Inquiries into Maternal Deaths in England and Wales found that,
 - a. **13%** of all maternal deaths were associated with anaesthesia (1982-84)
 - b. **41%** of deaths arising from anaesthesia related to intubation difficulties (1973-1984)
- postulated reasons for this include,
 - a. presence of full dentition
 - b. increased incidence of laryngeal and pharyngeal oedema
 - due to an effect of **oestrogen** on the ground substance of connective tissue
 - increase in total body water, with capillary engorgement of the mucosa of the nasal passages, pharynx and larynx
 - increased size and decreased mobility of the tongue
 - **preeclamptic** patients in particular develop widespread soft tissue oedema
 - c. presence of large pendulous breasts
 - d. failure to allow adequate time for paralysis with suxamethonium
 - e. incorrectly applied, or overenthusiastic cricoid pressure may distort the larynx
 - f. most emergency obstetric anaesthesia is carried out by junior anaesthetic staff
- physical characteristics associated with difficulty with intubation include,
 - a. short muscular necks
 - b. a receding mandible - micrognathia
- obtuse mandibular angles
 - c. an increased alveolar-mental distance (anterior depth of mandible)
 - d. a prominent maxilla with protruding upper incisors
 - e. poor mobility of the - neck
- temporomandibular joints
 - f. long high-arched palate with a narrow mouth

Airway Assessment

NB: it is axiomatic that careful preoperative assessment which predicts airway difficulty is the best method of avoiding disaster

■ Clinical Assessment

- even the most exhaustive assessment **does not** predict all cases
- one of the most useful bedside examination predictive of difficult intubation is that described by **Mallampati et al.** and modified by Samssoon and Young*
- assessment is made with the patient sitting upright and the head in the neutral position, with the mouth fully open and the tongue extended, **without** phonation,

Mallampati Classification - Class	
Class 1	• faucial pillars, soft palate and uvula visible
Class 2	• faucial pillars and soft palate visible • but uvula obscured by the tongue
Class 3	• only the soft palate is visible
Class 4*	• the soft palate is not visible

NB: this classification has been correlated with the **grading** system, at laryngoscopy, of **Cormack and Lehane**,

Cormack and Lehane - Grading	
Grade I	• full exposure of the glottis
Grade II	• visualisation of the posterior commissure of the glottis
Grade III	• no exposure of the glottis • corniculate cartilages visible
Grade IV	• no exposure of the glottis, or of the corniculate cartilages

- the Mallampati classification predicts ~ 50% of difficult airways, and has a high incidence of **false positives**
- Tham *et al.* showed that posture has little effect upon the grading, thus assessment in the **supine** position is equally predictive and can be used in emergency situations

Obstetric Anaesthesia

- due to the unreliability of the Mallampati grading, *Wilson et al.* analysed 20 different patient factors and airway measurements, however only 5 correlated with difficult intubation

Wilson - Risk Sum Analysis		
Risk Factor	Level	Description
Weight	0	< 90 kg
	1	90-110 kg
	2	> 110 kg
Head & neck movements	0	above 90°
	1	about 90°
	2	below 90°
Jaw movements	0	IG ¹ ≥ 5 cm or SLux ² > 0
	1	IG < 5 cm and SLux = 1
	2	IG < 5 cm and SLux < 0
Mandibular recession	0	normal
	1	moderate
	2	severe
Buck teeth	0	normal
	1	moderate
	2	severe
Total Score	0-10 points	> 2 <i>potentially difficult</i>
¹ IG: incisor gap ² SLux: maximum forward protrusion of the lower incisors beyond the upper incisors		

NB: total score > 2 → 75% of difficult intubations
false positive rate > 12% (unacceptably high)

- *Oates et al.* compared these two systems prospectively in 675 patients,
 - they were **unable** to predict > 50% of difficult intubations with either system
 - the predictive **power** of each was similar but poor
 - Wilson ~ 8.9%
 - Mallampati ~ 4.4%
 - there was an unacceptable **false positive** rate with both systems
 - they preferred Wilson's score as there was less inter-observer variation

NB: Wilson's group concluded that it was not possible to identify the majority of difficult patients without the false positive rate increasing unacceptably, and it was therefore necessary to practice *failed intubation routines*

- another measure of airway difficulty is the *thyromental distance* (from the notch of the thyroid cartilage to the mental prominence with the neck fully extended),
 - i. > 6.5 cm - airway problems are unlikely
 - ii. 6-6.5 cm - without other anatomical abnormalities
→ intubation is likely to be difficult but possible
 - iii. < 6.0 cm - laryngoscopy is likely to be impossible

■ Radiological Assessment

- radiological indicators (Bellhouse) of difficulty with laryngoscopy include,
 - a. ratio of the effective mandibular length to the posterior depth of the mandible > 3.6
 - b. reduction of the atlanto-occipital distance (occiput to spinous process of C₁)
 - c. reduction of the C₁-C₂ interspace
 - d. increased anterior-posterior thickness of the tongue
- radiological examination is seldom performed and somewhat impractical for routine use
- CT and MRI scanning may provide excellent assessment of abnormal anatomy, as with upper airway tumours

■ Flow Volume Loops

- of the currently available pulmonary function tests, these allow the easiest recognition of airway obstruction
- obstructions can be identified as fixed, intrathoracic or extrathoracic
- limited by the requirement for patient cooperation, observer training, and the distortion by concurrent airways disease

Assessment of Airway

1. **history**
 - i. letters etc. re previous difficult intubation
 - ii. previous anaesthetic records
2. **examination** → "**MOUTHS**"
 - i. **M**andible
 - thyromental distance > 6 cm, 3 "finger-breadths"
 - alveolar-mental distance < 2 cm
 - "receding", length - subluxation
 - obtuse mandibular angles
 - ii. **O**pening
 - incisor gap > 4 cm (Wilson > 5 cm)
 - iii. **U**vula
 - Mallampati grades I-IV - as per Samssoon & Young
 - iv. **T**eeth
 - prominent upper incisors, "buck" teeth
 - solitary uncisors, nuisance teeth
 - loose teeth
 - crowns, caps, plates & dentures
 - v. **H**ead
 - flexion, extension, lateral flexion & rotation
 - vi. **S**ilhouette
 - obesity
 - Dowager's hump
 - "no neck", neck masses
 - craniofacial anomalies
3. **investigations**
 - i. direct awake laryngoscopy
 - ii. indirect laryngoscopy
 - iii. fluoroscopy
 - iv. XRays (Bellhouse)
 - effective mandibular length
 - atlanto-occipital distance
 - C₁-C₂ interspace
 - anterior-posterior thickness of the tongue
 - v. CT scan
 - tracheal deviation, luminal diameter
 - intrathoracic trachea

Detection of Oesophageal Intubation

■ Clinical Evaluation

- **visualisation** of the tube passing through the larynx would seem adequate in experienced hands
- however, this has been shown to be unreliable with inexperienced staff and the cords cannot always be seen
- **auscultation** of breath sounds has long been known to be an **unreliable** sign of tube placement
- there are numerous reports of "normal" chest expansion and breath sounds despite oesophageal intubation
- auscultation is actually more accurate in detecting **endobronchial** intubation
- **Anderson** (1989) in a blinded study found that,
 1. abdominal movements were judged normal in 36/40 cases when an oesophageal tube was ventilated
 2. condensation in the ETT was seen in 34/40 of the same group
- visualisation of the carina and tracheal rings at fibre-optic bronchoscopy is usually reliable

■ Exhaled Carbon Dioxide

- theoretically there should be no expired CO₂ from the stomach
- however, if there has been excessive **mask ventilation** prior to intubation then the concentration of CO₂ in the stomach may be close to that of the alveoli
- Ping (1987) reported a virtually normal ETCO₂ waveform following oesophageal intubation
- however, after 3 inspiratory cycles this rapidly fell to zero
- another device which utilises CO₂ is the **Fenem detector**, which is a dome shaped device which is purple in room air and turns **yellow** in the presence of CO₂
- this has the advantage of lightweight and portability, therefore may be useful outside of the operating theatre

NB: the effectiveness of ETCO₂ is not decreased in light of these reports, however, a minimum of **6 breaths** should be observed to ensure endotracheal placement

■ Oesophageal Detector Device

- first described by **Wee** (1988) and consists of a 60 ml catheter-tip syringe with a standard 15 mm tracheal tube fitting at the distal end
 - a. aspiration from the oesophagus results in its collapse and return of the plunger
 - b. aspiration from the trachea simply removes ventilatory air
- as the stomach may act like a "lung" when over-inflated, the device should be used prior to attempted ventilation
- **Nunn** modified this device by attaching an Ellick's evacuator bulb to the tracheal tube connector
- in prospective trials these have proved **> 99% accurate** and are inexpensive and simple to use

■ Transtracheal Illumination

- the use of an illuminated introducer was first described by MacIntosh in 1952
- other workers have used lighted stylets placed within the ETT to confirm endotracheal placement
- transillumination not occurring with oesophageal placement of the tube

■ Improved Patient Monitoring

- the ASA closed claims analysis judged that better monitoring would have prevented an adverse outcome in **72%** of the claims for adverse respiratory events
- SpO₂ and ETCO₂ were suggested by the reviewers in 98% of cases
- minimum monitoring standards apply equally to the induction room & operating theatre
- NB: faculty policy document

Management of the Known Difficult Airway

- most anaesthetists would elect to intubate the trachea under sedation and local anaesthesia
- the main sensory nerves to the larynx are cranial nerves V, IX, and X, which cannot be blocked directly, therefore surface anaesthesia ± nerve blocks is the only option
- those nerves which are accessible to blockade are the
 - a. superior laryngeal
 - percutaneously at the greater horn of the hyoid bone
 - runs submucosal in the pyriform fossa and may be blocked by topical anaesthetics
 - b. recurrent laryngeal - transmucosally following a transtracheal injection of LA
- introduction of an ETT may be achieved by methods from *above* or *below* the cords

■ Newer Laryngoscope Blade Designs

- these devices require practice and training, cf. the use of a normal blade
- the Bellhouse blade is one example, which is a straight blade bent at 45° in the middle
- the lamp is 2cm from the end and a prism may be attached for extremely difficult cases
- although excellent reports have been claimed, there are no comparative studies

■ Blind Nasal Intubation

- relies on favourable anatomy of the patient, correct positioning of their head and a number of techniques to locate the laryngeal inlet
 - a. listening for breath sounds at the 15mm connector
 - b. insertion of a stethoscope tube down the ETT lumen and listening for breath sounds
 - c. attachment of a capnograph to the end of the ETT
- disadvantages of this technique include,
 - a. trauma to the nasal passages, pharynx, larynx or oesophagus
 - this is ***relatively contraindicated in pregnancy***
 - b. reliance on operator experience
 - c. high incidence of oesophageal intubation

■ Gum Elastic Bougie

- first described by ***Macintosh*** in 1949
- where the vocal cords cannot be seen, there are 3 mechanisms of placement,
 1. the tracheal rings may be felt as "clicks" as the bougie slides in (~ 90%)
 2. the bougie may be held-up at the small bronchi, whereas the entire length will pass into the oesophagus (~ 100%)
 3. the patient may cough
- use of a "hollow" type bougie may allow attachment of a capnograph to the end
- successful passage of an ETT over the bougie may be facilitated by,
 - a. rotating the tube 90° anticlockwise
 - longest aspect of the ETT bevel is furthest away from the post. commissure
 - less likely to impact on the right vocal cord
 - b. leaving the laryngoscope in place

■ Fibre-Optic Intubation

• where a difficult intubation is known or highly suspected then awake *fibre-optic intubation* is inherently safer, however may be exceedingly difficult in the presence of,

- a. bleeding - GIT or upper airway
- b. airway oedema * pregnancy, especially preeclampsia
- c. abnormal anatomy or upper airway masses

• *disadvantages* of this technique include,

- a. frequently prolonged *time* requirement (up to 3x as long)
- b. similar, but prolonged *hypertensive* response to intubation
- c. difficulty in maintaining an airway and *oxygenation* with the scope in place
 - especially if the procedure is done under GA or heavy sedation
- d. use of a reasonable sized ETT through the *nasal* passages
 - minimum size depends upon external diameter of scope
- e. difficulty in ETT placement when the *oral route* is used

• these later factors, plus the frequent requirement for rapid induction of anaesthesia in obstetrics makes this a less useful approach

■ Rigid Bronchoscope

- although the cords may not be visible with a standard laryngoscope, they are frequently visible with a Negus bronchoscope, due to the different angle of approach
- this may then be used for insertion of a bougie

■ Light Wand

- this is essentially a flexible light source, inserted through an ETT to its distal end
- intubation is then performed "blind" in a darkened room, inspecting the cricothyroid membrane for transillumination
- insertion into the oesophagus will not result in illumination of the anterior larynx

■ Laryngeal Mask Airway

- first described by *Brain (1983)*, who subsequently described its use in 3 cases of failed intubation
- numerous case reports have emerged since confirming its usefulness in this scenario, including its use during emergency caesarean section
- during the later cases *cricoid pressure* was maintained throughout
- clearly it is most suited to elective cases where there is minimal risk of aspiration and no need to paralyse and ventilate the patient, though ventilation is possible
- use of the LMA may also be inappropriate in patients with abnormal upper airway anatomy, eg. those with tumours or foreign bodies, as insertion may be unsuccessful

• *advantages* of the LMA include,

- a. high rate of successful insertion in inexperienced hands
- b. successful insertion and ventilation in patients in whom the larynx is not visible
- c. use as a blind guide for endotracheal placement of either,
 - i. a size 6.0 ETT
 - ii. a bougie and subsequent insertion of a larger ETT
 - iii. a fiberoptic scope (with subsequent sacrifice of the LMA)
- d. allows maintenance of an airway and oxygenation
 - i. prior to emergence from anaesthesia in aborted attempts
 - ii. during placement of a spinal anaesthetic, emergency LUSCS
 - iii. during anaesthesia for LUSCS, extreme foetal distress, failed intubation

• *limitations* of the LMA, during non-life-threatening situations, include,

- a. an insertion *failure rate* ~ 4%
- b. limitation of *IPPV* ≤ 2 kPa (15 cmH₂O)
- c. failure to protect the lower respiratory tract from *aspiration*

NB: there are several case reports of aspiration during LMA use

■ Retrograde Intubation

- all of these are variations of the original description by **Waters** in 1963,
 - a. a Tuohy epidural needle is inserted through the **cricothyroid** membrane
 - b. position is confirmed by aspiration of air through a saline filled syringe
 - c. a catheter is inserted through the needle and emerges through the mouth or nose
 - d. an appropriately sized ETT is then guided along the catheter until it rests against the exit of the catheter from the larynx
 - e. with continued application of pressure to insert the ETT, the catheter is cut and the ETT advanced into the larynx, helping to lessen the chance of posterior displacement
- there are various forms of this technique, generally known as guided "blind" retrograde oral or nasal intubation
- the major difficulty with the technique is passage of the ETT past the epiglottis, tending to be more difficult from the oral route
- a variation was published by **Shanther** (1992 - BJA) using the **cricotracheal** approach
- the claimed advantages of this technique include,
 - a. the penetrating needle does not enter the cavity of the larynx and the cricothyroid arteries are avoided, therefore there is less risk of haemorrhage
 - b. this level is usually cephalad enough to avoid the thyroid isthmus
 - c. the entry into the airway is lower, allowing passage of more of the ETT into the larynx prior to cutting the catheter, reducing the chance of posterior displacement
 - d. traction on the catheter is transmitted to the solid cricoid cartilage, whereas following cricothyroid puncture traction may result a vertical tear of the cricothyroid membrane, or in pressure against the thyroid cartilage with haemorrhage and subcutaneous emphysema

■ Oesophageal Airways

- other devices used for blind insertion during failed intubation include,
 - a. oesophageal obturator airway
 - b. oesophageal gastric tube airway
- however,
 1. at least 19 cases of **oesophageal rupture** have been reported, most being fatal
 2. **endotracheal placement** occurs in ~ 4-10% of cases, and at least 9 deaths resulted
 3. a number of studies have indicated ventilation may be inadequate

Emergency Anaesthesia for Difficult Intubations

- *optimise* the following factors,

1. patients *clinical condition*
 - ABC
 - ability to protect own airway
2. anaesthetic *equipment*
 - tubes, laryngoscope blades, assist devices
 - LMA, minitracheostomy set
 - fiberoptic instrumentation
3. *expertise*, anaesthetic & surgical
 - most senior available anaesthetist
 - ENT or general surgeon for tracheotomy

- lessen the *risk of aspiration*,

1. allow time for the stomach to empty \pm metoclopramide
 - this assumes there is no GIT pathology and surgery can be delayed
2. a nasogastric tube may be passed to empty the stomach contents (unreliable)
3. reduce stomach acidity by the administration of antacids and/or H₂-blockers

- there are two generally accepted options to this problem,

1. *awake intubation* under local anaesthesia
2. *tracheotomy* under local anaesthesia, followed by intubation and GA

NB: the choice between these should be that which provides the *safest conditions* for the patient.

- Ovassapian (1989) argued that the upper airway could be anaesthetised in a patient at high risk for aspiration, as the lower respiratory tract would remain sensitive and capable of expelling foreign material

- this opinion is *not universally accepted*

- the problem case of the emergency LUSCS for foetal distress in a patient known to have a difficult airway is probably best performed under *spinal anaesthesia*

- failing this, then it would "probably" be safe to anaesthetise the upper airway, providing the time required did not place the foetus at undue risk

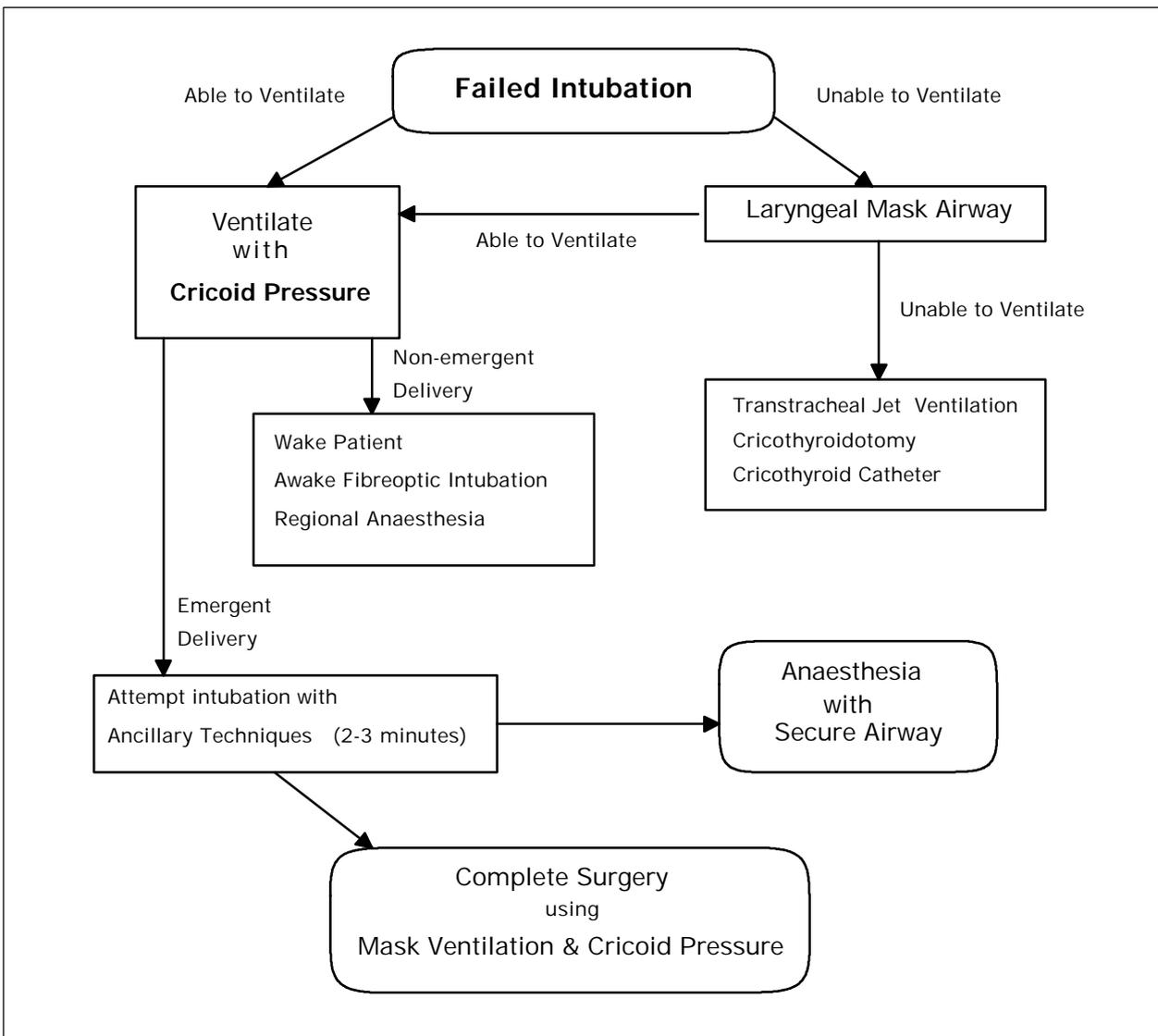
- regardless of the means of endotracheal placement, *confirmation* of correct placement is essential

Transtacheal Ventilation

- inability to ventilate the patient via face or laryngeal mask leaves this as the only alternative
- there are 3 generally accepted methods of achieving this end,
 1. **needle cricothyroidotomy**
 - reviewed by Benumof & Scheller (1989) who concluded that transtacheal jet ventilation was the **most efficient** means of ventilation in these circumstances
 - adequate ventilation may be achieved by either,
 - i. a Sanders injector at ~ 40-50 bpm ~ 2.5-3.0 bar
 - ii. a 3-way tap, cricothyroid cannula and non-compliant tubing with O₂ at 15 l/min, or using the O₂ flush from the anaesthetic machine
 - if the patient can exhale through the larynx, the risk of **barotrauma** is reduced
 - failure of this may require a 2nd cannula through the cricothyroid membrane
 - attachment of the anaesthetic circuit to the cannula & manual ventilation has been described, however, ventilation is less efficient & CO₂ will rise
 - however, **oxygenation** may be maintained pending definitive airway management
 - methods for connection include,
 - i. connecting the male end of a 3mm ETT adaptor directly to the cannula
 - ii. connecting the male end of a plungerless 3 or 5 ml syringe to the cannula, then inserting the end of a 8 or 10 mm ETT adaptor into the empty syringe barrel
 - **complications** of transtacheal jet ventilation include,
 - i. barotrauma
 - up to 14.4% in one series
 - subcutaneous emphysema
 - mediastinal emphysema & pneumothorax
 - ? VAE
 - ii. trauma due to catheter placement
 - iii. migration of the catheter
 - iv. the effects of dry gases
 - v. permissive hypercapnoea
- 2. **minitracheostomy**
 - difficult under elective conditions, especially in inexperienced hands
 - complications include submucosal haematomas and cricoid cartilage injury
 - surveys in major training hospitals reveal that only a small percentage of anaesthetists have had any experience with these devices
 - there are essentially 2 types available
 - i. those which allow ventilation through the tube, and
 - ii. those with narrow lumens, which require jet ventilation and a patent larynx for exhalation
 - generally these **do not** protect the airway
- 3. **surgical cricothyroidotomy**

Failed Intubation Drill

- factors which increase the associated risk of this event include,
 - a. an inexperienced anaesthetist
 - b. emergency anaesthesia, especially late at night
 - c. the presence of a full stomach
 - d. *pregnancy*
- as difficult intubation cannot be predicted in 100% of cases, most reviewers agree the best approach is for trainee staff to undergo regular practice of a *failed intubation drill*
- the essence of all failed intubation drills is,
 - a. oxygenation of the patient, by airway maintenance and ventilation, and
 - b. the prevention of aspiration of gastric contents, through positioning and continued application of cricoid pressure



PREGNANCY INDUCED HYPERTENSION

■ Incidence

- a. 5-10% of all pregnancies
- b. highest in primigravidas, though, the *prevalence* is greater in multiparous
- c. 0.05-0.2% of all deliveries progress to *eclampsia* (1:500-1:2,000)
- d. PIH is implicated in,
 - i. ~ 20% of maternal deaths
 - ii. ~ 6-10% of perinatal deaths

■ Categories

1. **gestational hypertension / proteinuria**
 - i. hypertension without proteinuria
 - ii. proteinuria without hypertension
 - iii. proteinuric hypertension → *preeclampsia*
 - developing *after* the 20th week of gestation
 - may occur earlier if associated with *hydatidiform mole*
2. **chronic hypertension**
 - i. incidentally associated with pregnancy
 - ii. aggravated by pregnancy or preeclampsia
3. **unclassified hypertension**
 - found later than 20 weeks where the past hypertensive status is unknown
4. **eclampsia**
 - any form of hypertension accompanied by *fitting*

Preeclampsia

- *gestational hypertension* is defined as,
 1. BP > 140/90 mmHg * on at least 2 occasions, > 6 hours apart, or
 2. increased BP above pre-pregnancy levels
 - i. > 30 mmHg systolic
 - ii. > 15 mmHg diastolic
- preeclampsia exists if any *two* of the following are present,
 1. gestational hypertension
 2. proteinuria > 300 mg/l per 24 hours
 3. oedema

Obstetric Anaesthesia

• *severe preeclampsia* is denoted by *any* of the following,

1. systolic BP > 160 mmHg
diastolic BP > 110 mmHg * on at least 2 occasions, > 6 hours apart
2. proteinuria > 5g / 24 hours * or rapidly increasing
3. oliguria < 400 ml / 24 hours
4. cerebral or visual disturbance
5. cyanosis or pulmonary oedema
6. epigastric pain

NB: *eclampsia* being the presence of any degree of hypertension, or proteinuria, with the occurrence of *convulsions*

■ Aetiology

• unknown, though, it is generally agreed the underlying disorder relates to *utero-placental ischaemia*

- a. genetic
- b. acquired immune factors
 - immune reaction to *trophoblastic* tissue → placental vasculitis & ischaemia
 - more common amongst,
 - i. nulliparas - no previous exposure to trophoblast
 - ii. conditions with a large mass of trophoblast
 - hydatidiform mole
 - multiple pregnancy
 - diabetes
 - Rh incompatibility
- c. abnormal (placental) activation of renin-angiotensin-aldosterone
- d. imbalance in prostaglandin synthesis
 - i. angiotensin II / PGE₂ imbalance (PGE = vasodilator secreted by placenta)
 - ii. **PGI₂ / TXA₂ imbalance** - Walsh (Am.J. O&G, 1985)
 - behind the advent of the "CLASP" (collaborative low dose aspirin) trial
- e. nutritional disorders / deficiencies
- f. a combination of these

• the disease appears to have both a *geographical* and *socioeconomic* distribution

• the incidence being far greater in,

- a. developing countries
- b. immigrant populations in developed countries
- c. lower socioeconomic groups in developed countries

Pathophysiology

NB: the majority of lesions are due to *occlusive vascular spasm* of arterioles

■ Central Nervous System

- a. normal total CBF, however, there is *focal* vasospastic ischaemic injury
 - MRI, CT and angiographic evidence
 - postmortem studies → haemorrhagic necrosis around thrombosed precapillaries
 - petechial haemorrhages are common after the onset of convulsions
 - EEG shows diffuse slowing (δ/θ)
 - ~ 50% of preeclamptic women
 - ~ 75% of eclamptic women
- b. generalised oedema - does occur but usually 2° to seizures, not a 1° event
 - * papilloedema occurs rarely
- c. headache, visual disturbance, other focal neurological signs
- d. irritability, hyperreflexia
- e. sensitive to central depressant drugs
- f. *convulsions*
 - i. frequently have prodromal signs, but may be completely unheralded
 - ii. often intractable leading to status epilepticus
 - iii. ≥ 30% occur *post-partum* * most of these within the first 48 hours
 - iv. may occur with only minimal increase in BP, cf. hypertensive encephalopathy
- g. *intracranial haemorrhage* * leading cause of maternal death (~ 50%)

■ Cardiovascular

- a. increased total body water
- b. reduction in blood volume
 - ~ 10 mild disease
 - ~ 30-40% in severe cases
- c. decreased RBC mass * masked by haemoconcentration
- d. arteriolar vasoconstriction
 - i. increased SVR * PVR remains ~ normal
 - ii. increased sensitivity to vasoactive drugs
 - iii. increased LSVWI ~ 25% show suboptimal myocardial function
- e. increased vascular permeability
 - i. fluid shifts from intravascular to extravascular space
 - responsible for haemoconcentration and the Hct. ~ normal
 - ii. hypoproteinaemia
 - iii. generalised oedema

Obstetric Anaesthesia

- f. decreased CO * one study suggested normal to increased, however, they used MgSO₄ first
- g. CVP / PCWP low to normal
 - i. the reduction in CVP generally correlates with the severity of hypertension
 - ii. there may be marked discrepancy between CVP & PCWP

NB: the initial presentation of low PCWP & CI, plus high SVR & HR responds well to initial *volume expansion*, with elevation in filling pressures and CI, and a reduction in SVR & HR; subsequent infusion of hydralazine resulted in further reduction in SVR without a reduction in PCWP (Finster)

■ Respiratory

- a. frequently none
- b. facial and laryngeal oedema * difficulty with *intubation*
- c. increased shunt and V/Q mismatch
 - i. pulmonary arteriolar resistance is normal or low
 - ii. increased δP_{A-aO_2} may occur, usually associated with a *rise* in PCWP
 - iii. represents **LV dysfunction** more than ARDS
- d. aspiration during eclamptic seizures
- e. pulmonary oedema common at autopsy in fatal cases

■ Renal

- a. decreased ERBF and GFR
- b. swelling of glomerular endothelial cells and luminal narrowing by *fibrinoid* deposition
- c. increased glomerular permeability to large molecules
- d. **proteinuria** → may reach nephrotic levels > 10-15 g/24 hrs
- e. decreased clearance of **uric acid**, proportional to severity of hypertension
 - i. normal ~ 0.18-0.21 mmol/l ~ 3-3.5 mg/dl
 - ii. mild PIH > **0.24 mmol/l** ~ 4.0 mg/dl
 - iii. severe PIH > **0.45 mmol/l** ~ 7.5 mg/dl
- f. decreased clearance of urea & creatinine in more severe cases ~ 30-50%
- g. **oliguria** * frequently due to **hypovolaemia & ↓ RBF**
- h. acute renal failure requiring dialysis,
 - i. overenthusiastic hypotensive therapy
 - ii. haemoglobinuria associated with HELLP
 - iii. good prognosis if remainder of disease is appropriately managed

■ Hepatic

- a. hepatic swelling & epigastric pain
 - i. subcapsular haemorrhages
 - ii. ischaemic hepatic necrosis
 - iii. fibrinoid deposits in venous sinusoids
- b. spontaneous hepatic rupture * rare, but mortality 55-75%
- c. ascites
- d. HELLP * elevated liver enzymes

■ Coagulation

- a. thrombocytopenia ~ 20-30% of preeclamptic patients
 - usually in the range 100-150,000 / mm³
 - * associated **platelet dysfunction** ~ 10-25%
- b. DIC ~ 7% of all cases, more common with **abruption**
- c. HELLP
 - haemolysis, elevated liver enzymes, low platelets
 - associated with a poor maternal & foetal outcome
 - no relationship between hypertension & HELLP

NB: recent studies indicate the SBT is **not** a reliable test of clotting (Rodgers & Levin, 1990, Sem. Thrombosis Haemostasis)

■ Foeto-Placental Unit

- a. decreased uterine and placental blood flow ~ 50-70%
- b. premature placental aging with,
 - i. increased infarcts
 - ii. uterine hypertonicity
 - iii. increased sensitivity to oxytocic drugs
- c. increased frequency of,
 - i. intrauterine growth retardation < 25th percentile
 - ii. intrauterine foetal death ≤ 23%
 - iii. premature labour
 - iv. placental abruption ~ 15x normal frequency
 - v. LUSCS
 - vi. neonatal hypoglycaemia & hyperbilirubinaemia

Management - General

NB: the cure for preeclampsia begins with delivery of the foetus, early delivery has been stressed as crucial in avoiding serious sequelae, all other treatment modalities are supportive only !

• conditions which mandate the **delivery** of the foetus, regardless of the gestational age, include

1. severe hypertension, persisting after 24-48 hours of treatment
2. presence of the HELLP syndrome
3. acute renal failure
4. eclampsia

NB: further delay rarely improves foetal survival and is detrimental to the mother

• standard **obstetric management** includes,

1. decreasing CNS irritability / control of convulsions
2. blood pressure control
3. improving end-organ perfusion → urine output ~ 1 ml/kg/hr
4. correction of coagulopathy if present

■ CNS Irritability

a. MgSO₄

- i. anticonvulsant * administered parenterally **is not** an anticonvulsant itself
 - effect is due to potent **cerebral vasodilatation**
 - also blocks NMDA glutamate receptors ?protective
 - cleared by the kidney, therefore monitor level & [Cr]_{pl}
 - therapeutic blood levels ~ **2-4 mmol/l**
 - loading dose ~ 40-80 mg/kg
 - maintenance ~ 1.0-2.0 g/h
 - ampoules 10 mmol/5 ml ~ 2.5g
- ii. muscle paralysis - NMJ blockade is a **linear** function of plasma [Mg⁺⁺]
 - decreases release of ACh from motor neurones
 - decreases the sensitivity of the motor endplate
 - decreases the excitability of the muscle membrane
 - * diaphragmatic paralysis at ³ **7 mmol/l**
- iii. cardiovascular
 - reduces SVR and increases CO, ± reflex tachycardia
 - increased conduction time
 - ↑PR, ↑QRS and ↑QT duration ≥ 5 mmol/l
 - decreased discharge rate of S-A node
 - may abolish digitalis induced VPC's
 - hypotension, conduction disturbances ± CHB

- iv. uterine / foetal
 - reduces uterine hypercontractility → *tocolytic*
 - crosses the placenta & may cause *foetal hypotonia*
- b. other anticonvulsants
 - i. diazepam
 - * treatment of choice for acute seizure control
 - ~ 5-10 mg increments until effective
 - continuous infusions result in too much sedation
 - ii. *phenytoin*
 - more popular for prophylaxis due to lack of sedation
 - side effects → rash, nausea and blurred vision
 - therapeutic levels ~ 40-100 µmol/l
 - loading dose ~ 10 mg/kg in 100 ml, over 20 minutes
+ 5 mg/kg in 100 ml, 2 hours later
 - maintenance ~ 200 mg po/iv q8h, 12 hrs post-loading

NB: 1. MgSO₄ produces better control of convulsions cf. diazepam, diazepam & pentazocine, diphenyl hydantoin, or epanutin
2. MgSO₄ was superior to diazepam for foetal wellbeing
3. one study showed the best perinatal outcome with diphenyl hydantoin

■ Control of Hypertension

- wide debate as to what represents adequate monitoring in severe preeclampsia
- good data to show that CVP may not reflect PCWP and there may be associated LV dysfunction

NB: James states that "as volume loading is frequently necessary in these patients, a CVP line represents minimum monitoring in any patient with severe PET"

- where hypertension is marked, and a potent vasodilator is to be used, then direct arterial BP monitoring is advisable
- the case for PA catheters is less clear, Clark & Cotton 1988 recommended their use in,
 - 1. hypertension unresponsive to conventional doses of hydralazine
 - 2. pulmonary oedema
 - 3. oliguria unresponsive to volume challenge

NB: but the majority of patients could be managed *without* central catheterisation

- as the condition is one of vasospasm and there is usually associated volume depletion, most authorities recommend *gradual* reduction in BP to slightly *supranormal* values
- rapid, or profound reductions in MAP may have adverse effects upon both mother and foetus
- arteriolar vasodilators are the most popular agents, but provision of adequate volume expansion must be instituted *prior* to their use

- methods for controlling hypertension include,
 - a. bed rest and hospitalisation
 - b. avoidance of aortocaval compression
 - c. **dihydrallazine** ~ 5 mg IV q20m (max ~ 10 mg)
 - * maximal effect is in ~ 15 minutes, \ 20 minute intervals
 - increases RBF, CO, **HR** and uterine blood flow
 - d. **nifedipine**
 - as yet not widely used in PE
 - 10 mg SL q20m, up to 30 mg has been recommended
 - ? possible beneficial effects on platelet count & renal function
 - e. **b-blockers**
 - esmolol produces adverse effects upon foetal sheep
 - * adrenergic blockade and hypoxaemia
 - **labetalol** has been used successfully in one series, but caution is required if the foetus is premature
 - f. **methyldopa**
 - usually used for chronic hypertension
 - long safe history in pregnancy up to 1-3 g/day in divided doses
 - may cause drowsiness, depression & postural hypotension
 - g. diazoxide
 - used less frequently, only for refractory hypertension
 - hypotension is unpredictable and inhibits uterine activity
 - * use **small doses** ~ 30-50 mg and monitor closely
 - * maternal collapse & death have followed standard 300 mg doses
 - h. trimethaphan
 - absence of significant cerebrovasodilatory effects
 - ? therefore may actually be detrimental
 - metabolised by plasma ChE & prolongs suxamethonium
 - doesn't cross the placenta
 - may decrease venous return → reflex tachycardia
 - i. nitroprusside
 - limited to short-term control of refractory hypertension
 - * fears of cyanide toxicity in the foetus
 - j. nitroglycerine
 - predominantly a venodilator & less effective with volume loading
 - may drop preload with high SVR & myocardial dysfunction
 - possible risk of maternal rises in ICP
 - k. diuretics
 - * used in the past but now generally **condemned**
 - exacerbate the existing volume deficit, despite oedema

■ Renal

- despite the presence of **oedema** and **oliguria**, the use of diuretics is not recommended
- **volume expansion**, arteriolar dilatation with a slightly supranormal MAP are required
- the usual aim is for a urine output ~ 1 ml/kg/hr
- the use of low dose **dopamine** has not been widely investigated in PE
- it could be expected to be beneficial and there are a small number of positive case reports
- the increased sensitivity of the mother to catecholamines needs to be remembered
- the use of **nifedipine** following delivery to enhance renal output has been described

■ Respiratory

- involvement is generally minimal, with the greatest problem being oedema of the upper airway and the potential difficulty in intubation
- pulmonary oedema is usually the result of overenthusiastic volume loading
- ARDS is *uncommon*

■ Other Systems

- *thrombocytopenia* is common, though, rarely severe
- requirement for platelet transfusion is very uncommon
- low grade DIC similarly rarely requires active treatment
- management of liver dysfunction is purely supportive
- the rare complication of liver rupture requires emergency surgery for haemorrhage control

Anaesthetic Management

NB: the role of anaesthetic management includes,

1. pain relief during labour
2. anaesthesia for LUSCS
3. intensive management of life-threatening complications
4. consultive help in the routine obstetric management

■ Lumbar Epidural Anaesthesia LEA

- epidural anaesthesia is recognised as the method of choice
- considerations prior to establishment of LEA include,
 1. *intravascular volume status*
 - hypotension was the principal objection in the past
 - studies vary in volume & initial choice of fluid, but volume expansion should be commenced *prior* to starting the block
 - i. ~ 500-1000 ml of crystalloid or colloid
 - ii. CVP \pm PCWP in the presence of oliguria or pulmonary oedema, etc.
 - Hodgkinson *et al.* in 1980 showed that LUSCS under epidural was associated with far less haemodynamic disturbance
 - analgesia with 10 ml bupivacaine 0.25% \rightarrow ~ 75% *increase* in intervillous blood flow
 - a *sequential* technique, raising the block in stages, rather than by a single bolus is generally preferred

2. **treatment of hypotension**
 - rapid administration of IV fluids
 - maintenance of left uterine displacement
 - **ephedrine** is safe, as it does not adversely affect uterine blood flow
 - α -agonists should be avoided as they may further decrease UBF
3. **adrenaline supplementation**
 - controversial due to the increased sensitivity of these patients
 - most studies have shown **no** adverse effect when administered properly into the epidural space
 - Heller & Goodman (1986) argued that only the β -effects were seen
 - however, many still avoid its use due to this risk of intravascular administration
4. **presence of coagulopathy**
 - routine placement results in inadvertent puncture of an epidural vein ~ 1-10%
 - epidural haematoma are rare, the majority being associated with vascular abnormalities, increasing age, anticoagulants, antiplatelet therapy and trauma
 - at least half are said to occur spontaneously during normal activity
 - Ramanathan (Anes. 1989) found that the platelet count only correlated with the SBT when the platelet count is $< 100,000 / \text{mm}^3$
 - general recommendations for LEA, (Douglas 1991 - CJA)
 - i. contraindicated if the platelet count is $< 50,000 / \text{mm}^3$
 - ii. contraindicated in the presence of the HELLP syndrome
 - iii. may be **justified** if the platelet count is $50-100,000 / \text{mm}^3$, and the bleeding time is normal - ie., the benefit $>$ risk
 - iv. is almost certainly safe if the platelet count is $> 100,000 / \text{mm}^3$
 - v. is probably safe if the patient is on low-dose **aspirin** therapy, providing there is no evidence of bruising or undue bleeding from venipuncture sites
 - vi. all patients with Plts $< 100,000 / \text{mm}^3$ should have a SBT performed
 - other haematologists would argue that the SBT is a useless investigation and that significant bleeding is exceedingly unlikely with Plts $> 50,000 / \text{mm}^3$
 - however, there are **no studies** to say this is a safe practice
5. **drug metabolism**
 - total body clearance of lignocaine is reduced and "normal" doses may result in accumulation & higher blood levels than in normotensive patients

■ Subarachnoid Anaesthesia

- if LEA was not used for labour, and an anaesthetic is required for vaginal delivery, then **low** subarachnoid anaesthesia (saddle block) may be performed
- the precautions are as for LEA above
- generally not recommended for LUSCS, due to the rapid onset of haemodynamic alteration, which may be more difficult to control
- some studies have actually suggested that PE patients are actually less sensitive to sympathetic blockade, due to their high levels of circulating vasopressors (catechols, angiotensin)

■ General Anaesthesia

- common indications for GA in the setting of PIH include,
 1. patient refusal of LEA
 2. presence of coagulopathy
 3. foetal distress requiring rapid delivery
 4. eclampsia

- problems associated with GA specific to PIH include,
 1. difficulty in maintenance of the **airway** and tracheal intubation
 - consider awake intubation in the presence of gross facial oedema or stridor
 - usual precautions for difficult intubation
 2. excessive **CVS response** to endotracheal intubation
 - increases in MAP up to 30%
 - potentially exceed the upper limits of cerebral autoregulation
 - large increases in mean PAP and PCWP
 - methods for attenuating this should aim at decreasing MAP ~ 20-25%,
 - i. supplemental thiopentone
 - ii. lignocaine ~ 1.5 mg/kg
 - iii. fentanyl ~ 3-5 µg/kg
 - iv. labetalol
 - v. vasodilators - SNP, trimethaphan, GTN
 3. **postoperative** management
 - i. a high proportion of **seizures** commence postoperatively
 - ii. associated fall in plasma oncotic pressure & propensity for **pulmonary oedema**

- **ketamine** and the **ergot alkaloids** should be avoided due to their pressor response
- in the presence of MgSO₄ the use of muscle relaxants requires cautious doses and monitoring

OBSTETRIC HAEMORRHAGE

NB: this is the major cause of maternal **mortality**
significant bleeding occurs in ~ 3% of all pregnancies

1. **placental causes**
 - i. placenta praevia[§]
 - ii. abruption placentae[§] § 50-70% of all **anteartum haemorrhage**
 - iii. placenta accreta / increta / percreta
 - iv. retained placenta
 - v. advanced ectopic pregnancy
2. **uterine & cervical causes**
 - i. uterine atony
 - ii. uterine rupture
 - iii. uterine inversion
 - iv. cervical or vaginal lacerations
 - v. uterine or cervical abnormalities * polyps, tumours, varicosities
 - vi. trauma
3. **coagulopathy**
 - i. DIC - intrauterine foetal death
 - chorioamnionitis
 - amniotic fluid embolism
 - placental abruption
 - ii. preeclampsia, HELLP syndrome
 - iii. coexisting haematological disease
 - iv. drugs

Placenta Praevia

- implantation of the placenta in the lower uterine segment, either partially or completely overlying the cervical os, classified as **total**, **partial** or **marginal**
- Clark (1985) reported an overall incidence ~ 0.3%, being increased by,
 - i. advanced maternal age ~ 3x increase over 35 years
 - ii. previous LUSCS * 10% with ≥ 4 previous sections
 ~ 0.26% without prior section
- classically presents as **painless** vaginal bleeding & accounts for 1/3 of all third trimester bleeding
- diagnosed on routine prenatal ultrasound with ≥ 95% accuracy
- maternal mortality has decreased to < 1%, but foetal mortality may be as high as 20%

■ Anaesthetic Management

- if the patient presents at *term*, then
 1. if haemodynamically stable and not actively bleeding, the diagnosis should be confirmed by ultrasound
 2. in the absence of ultrasound, examination should be performed in the operating theatre, with adequate preparation for emergency LUSCS

- in addition to the standard preparation for LUSCS, preoperative preparation should include,
 1. 2 large peripheral IV lines (14-16G)
 2. G&M blood
 - 2-4 units available in theatre
 - blood warmer & pressure bags available

- if the patient is not bleeding and presents for elective LUSCS, then *regional anaesthesia* is appropriate, providing provisions are made for the potential for increased blood loss
- if the patient is known placenta praevia, has had a prior LUSCS, and is therefore at increased risk of *placenta accreta* and *caesarean hysterectomy*, regional anaesthesia may still be used

- in the advent of *severe haemorrhage*, either at examination or at LUSCS, induction of GA may be required, if so,
 1. preoxygenation with 6 x VC breaths cf. usual 3-4 minutes
 2. standard RSI with cricoid pressure
 3. *ketamine* may be preferable to thiopentone for induction
 - if severely hypovolaemic / hypotensive then only small doses (0.5 mg/kg)
 - if in hypovolaemic shock then left uterine displacement is imperative for resuscitation
 4. maintain 100% O₂ until delivery of the foetus & control of haemorrhage
 - N₂O or volatile agents should only be added if the BP is adequate
 - volatile agents will result in uterine relaxation and may increase bleeding
 5. following delivery, add N₂O & opioid supplementation, decrease/cease volatile agents

- following delivery, patients are at greater risk of *post-partum haemorrhage*, as the lower uterine segment has less contractile capability
- routine use of a syntocinin infusion
- transfer and observation in a high dependency nursing area

Placenta Accreta

- placenta accreta, increta and percreta are conditions of **abnormal placentation**, frequently associated with **placenta praevia**

1. placenta accreta - villi attach directly to the myometrium, without decidua basalis
- there is **no invasion** of the myometrium
2. placenta increta - there is **invasion into** the myometrium
3. placenta percreta - there is **invasion through** the myometrium
± invasion of adjacent structures

- variable incidence, but has been reported as high as 1:2,562 pregnancies

- the aetiology is unknown, but there are a number of predisposing factors,

- i. placenta praevia * incidence of ~ 5% (Clark)
- ii. prior LUSCS * incidence of ~ 24% with 1 previous section
- iii. prior manipulation of the uterus - D&C, myomectomy, etc.
- iv. congenital malformations of the uterus
- v. uterine tumours
- vi. multiparity
- vii. ? smoking

- the principal risk is that of **haemorrhage**

- failing conservative management may require **hysterectomy**

- diagnosis during vaginal delivery, 2° to haemorrhage, requires standard ABC management,

1. supplemental O₂
2. IV access * G&M, rapid infusion equipment, blood warmers, etc.
3. crystalloid / colloid resuscitation
4. request for assistance & preparation of theatre
5. administration of antacid ± metoclopramide
6. brief history / examination *assessment of the airway
7. if a functioning epidural is in situ, then this may be continued providing,
 - i. intravascular volume & BP can be maintained
 - ii. the patient is capable of protecting their airway

NB: otherwise a GA with RSI should be administered

- in the patient with placenta praevia & previous section, or in those with ultrasound evidence of placenta accreta, regional anaesthesia is permissible providing there is no suggestion of placenta increta or percreta

- actually difficult to tell on ultrasound, \ most would elect for GA

Abruptio Placentae

- is the premature separation of an abnormally implanted placenta, after the 20th week of gestation
- the incidence varies from 0.5-2.5%, and this constitutes 1/3 of antepartum haemorrhages
- maternal mortality is < 3%, however perinatal mortality is high, up to 60% in some studies
- associated factors include,
 1. hypertensive disorders of pregnancy
 2. chronic hypertension
 3. multiparity
 4. uterine abnormalities
 5. previous abruption
 6. premature rupture of the membranes
- vaginal bleeding is variable & frequently underestimates the degree of total loss, blood tracking back into the myometrium and broad ligaments → **concealed abruption**
- diagnosis is made clinically or by ultrasound

■ Complications

1. hypotension ± haemorrhagic shock
 2. coagulopathy (DIC) ~ 20-40% of severe abruptions
 3. acute renal failure ~ 1-4%
 4. pregnancy induced hypertension ~ 50% of severe abruptions
 5. postpartum haemorrhage
 6. ischaemic organic necrosis
- DIC occurs 2° to the release of **tissue thromboplastin** from necrotic placental tissue, with activation of circulating **plasminogen**
 - this leads to activation of fibrinolysis and a consumptive coagulopathy, with reduction in platelets, fibrinogen and other clotting factors, leading to,
 1. hypofibrinogenaemia - decreased factors V and VIII
 2. thrombocytopenia
 3. prolongation of the APTT & INR
 4. increased fibrinogen degradation products
 5. widespread capillary damage and increased permeability

■ Anaesthetic Management

- clearly this depends upon the severity of the abruption
- assessment of circulatory status and stability are of prime importance
- in addition to the usual management of obstetric haemorrhage, patients require assessment and management for DIC
- as for other causes of antepartum haemorrhage, regional anaesthesia may be appropriate if intravascular volume can be maintained
- contraindications include any presence of coagulopathy,
 - i. platelet count $< 100,000 / \text{mm}^3$
 - ii. abnormal bleeding time, PT or APTT
 - iii. hypofibrinogenaemia or raised FDP's

- if the abruption is severe and/or foetal distress is present, then emergency GA as for placenta praevia is required
- **ketamine** can result in increased uterine tone and worsen the abruption & foetal distress
- if DIC is present patients may require additional blood products, including,
 - i. fresh whole blood - if available
 - ii. FFP, cryoprecipitate, platelets

- postpartum haemorrhage can occur 2° to uterine atony & DIC
- fibrin split products can inhibit the effect of oxytocics on the uterus, worsening uterine atony
- DIC usually resolves with removal of the placenta and foetus

Retained Placenta

- occurs in ~ 1% of all vaginal deliveries
- management requires manual exploration of the uterus, which requires analgesia \pm anaesthesia
- if regional anaesthesia is in place, this may be continued but requires extension to T₁₀
- if an epidural is not in place, depending upon the patient, the options include,
 - i. GA with RSI and endotracheal intubation
 - ii. subarachnoid anaesthesia
 - iii. monitored IV sedation

- **ketamine** should be avoided as it increases uterine tone and may make exploration difficult
- adequate IV access should be present
- prior to anaesthesia, a CBP and coagulation profile should be checked if possible

Uterine Rupture

- may occur ante / intra / postpartum, with an incidence of 0.08-0.1%
- maternal mortality varies from 5-60% if rupture is 2° to prolonged labour
- overall foetal mortality ~ 50%
- complete rupture is associated with sudden onset of severe abdominal pain and hypotension
- partial rupture can present as mild abdominal pain, shoulder tip pain (2° to blood), vaginal bleeding, or peritonitis
- associated maternal factors include,
 1. uterine scar ~ 0.5% with a lower uterine scar
 ~ 2.5% with a vertical scar
 2. previous difficult delivery
 3. rapidly progressive, tumultuous labour
 4. prolonged labour with excessive use of oxytocics
 5. weakness of uterine muscle - multiparity
 - multiple gestation
 - polyhydramnios
 6. cephalopelvic disproportion
 7. trauma 2° to - forceps
 - external version
 - intrauterine manipulation
- anaesthetic management is as for other causes of antepartum haemorrhage/hypovolaemia
- emergency GA is required unless there is a functioning epidural in situ and haemodynamic stability can be maintained

Uterine Atony

- this is a major cause of *postpartum haemorrhage*, occurring in ~ 2-5% of all vaginal deliveries
- maternal mortality is < 1% and associated factors include,

1. multiparity
2. large birth weight infant, multiple gestation, polyhydramnios
3. excessive use of oxytocics, prolonged labour
4. retained placenta
5. uterine inversion
6. chorioamnionitis
7. previous history of uterine atony

■ Management

1. intravascular resuscitation, oxygen
2. oxytocics & manual compression ± packing
* rapidly administered oxytocin may cause vasodilatation ± hypotension
3. intramyometrial prostaglandins
4. preparation for possible emergency laparotomy (as above)
5. laparotomy & ligation of internal iliac arteries
6. gravid hysterectomy

Uterine Inversion

- this is a rare cause of postpartum haemorrhage, but can be associated with massive haemorrhage and high maternal mortality
- classified as first, second or third degree and can occur acutely, before the cervical ring has contracted, subacutely, or chronically (> 4 weeks postpartum)
- factors associated with an increased incidence,
 - i. fundal implantation of the placenta
 - ii. antepartum use of MgSO₄
 - iii. uterine atony

- patients present with pain and haemorrhage ± shock, which is often underestimated
- management requires provision for rapid transfusion
- regional blockade may be adequate for acute inversion, prior to contraction of the os
- subsequent management requires uterine relaxation with GA with high dose volatile, the later mandating adequate intravascular resuscitation
- oxytocics should **not** be administered prior to version, as they will result in close of the os and exacerbate haemorrhage
- primiparous patients generally have greater blood loss, average estimates being ~ 1800 ml
- if the placenta is still adherent, then the uterus should be replaced prior to separation, as removal from the inverted uterus will result in profound bleeding
- complications include,
 - a. reinversion
 - b. sepsis
 - c. urinary retention
 - d. anaemia
 - e. pituitary necrosis - Sheehan's syndrome

Gravid Hysterectomy

- gravid, caesarean hysterectomy, or hysterectomy within 24 hours of delivery is the treatment of choice for postpartum haemorrhage refractory to other forms of treatment (any cause)
- **placenta praevia** and **placenta accreta** are the 2 most common causes, followed by uterine rupture and atony
- due to the increasing number of LUSCS deliveries, and the association between these and placenta accreta, there are an increased number of patients at risk for emergent hysterectomy
- in the study by Clark, 82% of patients with placenta previa/accreta and a uterine scar underwent gravid hysterectomy, cf. 58% of those without a uterine scar
- there is an increased morbidity/mortality due to the increased length of the procedure and associated blood loss ~ 2000-5000 ml, cf. ~ 1500 ml for elective caesarean section
- 96% of patients require a transfusion

VAGINAL BIRTH AFTER CAESAREAN SECTION

- between 1970 to 1984 in the USA, the LUSCS rate increased from 5.5% to 18%
- the commonest indication for section was a previous section, and in 1985 it was the most commonly performed major operation in the USA
- retrospective studies indicated that *trial of scar* (TOS) could safely be tolerated in,
 1. patients with a lower segment, transverse incision
 2. patients with more than 1 previous LUSCS incision
 3. those with oxytocic induction or augmentation of labour
- the overall success of TOS is 60-80%,
 - a. up to 90% when the indication for LUSCS was breech presentation
 - b. 55-65% for cephalopelvic disproportion or failure to progress
- the rationale for TOS is a decrease in maternal and foetal morbidity & mortality with vaginal delivery, cf. elective repeat caesarean section,
 - a. maternal factors
 - i. 30-50% increase in blood loss
 - ii. risk of postoperative thrombophlebitis (2-10% versus 0.1-0.25%)
 - iii. bladder (0.31%) and ureteral (0.09%) injury
 - iv. risk of wound infection
 - v. longer hospital stay (4.5 versus 2.5 days)
 - b. decrease in perinatal mortality rate (PMR) from ~ 40 to 20 : 1000
- there are 2 types of uterine separation associated with TOS,
 - a. ***incomplete rupture***
 - a relatively benign event, not associated with increased maternal/foetal morbidity
 - usually an incidental finding at postdelivery VE or at caesarean section
 - occurs with an incidence of 0.5-3.0% with TOS
 - b. ***complete rupture***
 - associated with increased maternal/foetal morbidity
 - incidence ~ 0.3-0.5% with TOS (0.125-0.3% spontaneous)
 - contributes to 1-4.7 : 1000 perinatal deaths (same cf. elective LUSCS)
 - therefore, rupture doesn't significantly contribute to the PMR from TOS

Epidural Analgesia and TOS

- the "classical" signs of uterine rupture include,
 1. pain between contractions
 2. a severe tearing sensation followed by cessation of contractions
- the majority of cases are now diagnosed by *foetal distress* on continuous foetal heart monitoring,
 - a. severe variable decelerations
 - b. prolonged decelerations
- data from Ostheimer, 14 studies between 1980-89, total 10,967 patients having TOS,
 - a. 41 complete uterine ruptures ~ 0.37% overall incidence
 - b. 1,623 had CEA ~ 0.86% incidence (~ 3x, but not significant)
~ 0.25% without epidural analgesia
 - c. abdominal pain alone, or in association with foetal distress was present in only 9/41 or 22% of cases
 - d. foetal distress alone was the presenting finding in 50% with CEA, and 61% without
 - e. if abdominal pain is going to present with rupture, then it is likely there will be "breakthrough pain" despite the presence of epidural analgesia, "*epidural sieve*"
 - f. there was *not* a significant difference in frequency with the use of oxytocin
 - g. CEA *does not* have a negative effect upon the success rate for vaginal delivery

■ Recommendations

1. sensory analgesia with either intraspinal opioids, or sensory anaesthesia with local anaesthetics, with or without opioids, should be the goal for pain relief
2. the lowest effective / acceptable concentration of local anaesthetic should be used, with or without opioids
 - bupivacaine 0.0625-0.125% & fentanyl 1-2 µg/ml at 8-12 ml/hr
3. continuous foetal heart monitoring should be employed
4. support for emergency caesarean section should be available

PREMATURITY

Def'n: birth of an infant between the 20th and 37th weeks of gestation
distinct from a *small for gestational age* infant → < 10th percentile

- the incidence ranges from 7-8% in the USA
- it accounts for ~ 80% of all *perinatal deaths*, either directly or indirectly
- morbidity & mortality tends to be greater than for the SGA infants
- the aetiology of the normal timing of labour is uncertain, but may involve,
 - a. upregulation of the myometrium to circulating oxytocin
 - b. increased oxytocin receptor numbers
 - c. prostaglandin synthesis
 - d. progesterone, oestrogen and catecholamines

■ Clinical Significance

- a. **maternal factors**
 - i. previous history of premature labour or abortion
 - ii. systemic disease - diabetes, hyperthyroidism, CVS disease
 - iii. trauma, surgery
 - iv. coitus
 - v. low socioeconomic status
 - vi. drug abuse - smoking, cocaine, ? ETOH
 - vii. general anaesthesia
- b. **uterine factors**
 - i. premature ROM
 - ii. incompetence of the cervix
 - iii. malformations, tumours, retained IUCD
 - iv. overdistension - polyhydramnios, multiple gestation
- c. **placental abnormalities** - praevia, abruption
- d. **foetal problems**
 - i. congenital malformations
 - ii. infections
 - iii. growth retardation, IUD

■ Obstetric Problems

- a. breech presentation ~ 25% cf. 3% normally
- b. maternal haemorrhage * associated problems
 - i. placenta praevia
 - ii. abruptio placentae
 - iii. uterine atony from residual tocolytic agents
- c. prolapsed cord / foetal distress
- d. infection
 - association of prematurity with premature ROM
 - signs may be obscured by administration of **glucocorticoids** to enhance lung maturity

■ Tocolysis

- absolute contraindications,
 - a. significant maternal haemorrhage
 - b. acute foetal distress
 - c. chorioamnionitis
 - d. eclampsia / severe preeclampsia
 - e. foetal anomaly incompatible with life, or IUD
- relative contraindications,
 - a. mild/moderate preeclampsia
 - b. maternal disease
 - i. cardiovascular
 - ii. endocrine - uncontrolled diabetes or hyperthyroidism
 - iii. renal disease
 - c. foetal growth retardation
- reasons for tocolytic therapy,
 - a. **transfer** to a high risk obstetric unit
 - b. enhancement of foetal **lung maturity**
 - < 34 weeks production of **surfactant** by type II pneumocytes is insufficient
 - maternal glucocorticoids **may** decrease IRDS, mechanism unknown
 - requires 24 hours for effect & effective within 7 days of delivery
 - undesirable side-effects in PIH, diabetes, and possibly infection, which may be increased by the β -adrenergic tocolytics

■ Premature Rupture of the Membranes

- 80-90% of such patients will proceed into premature labour within 7 days
- risk of delaying these patients is the development of *chorioamnionitis*,
 - a. 4 fold increase in
 - i. IRDS
 - ii. neonatal sepsis
 - iii. intraventricular haemorrhage
 - b. prophylactic antibiotic therapy has been shown to reduce these complications
 - c. tocolytic therapy in **not** more effective than expectant management
 - d. relative contraindication to administration of glucocorticoids

■ Anaesthetic Considerations

- a. labour and vaginal delivery in the event of failed tocolysis
 - b. elective caesarean section for maternal / foetal wellbeing
 - c. emergent caesarean section for foetal distress
- in addition to the usual requirements,
 1. to maintain maternal safety
 2. avoidance of foetal asphyxia, and
 3. provision of maternal comfort
 - during vaginal delivery it is important to inhibit the maternal "bearing-down" reflex, and thus a potentially traumatic delivery
 - uncontrolled, precipitous delivery increases the risk of *intracranial haemorrhage*
 - the preterm infant is also less tolerant of *asphyxia*, which markedly increases the risk of,
 1. respiratory distress syndrome
 2. intraventricular haemorrhage
 3. necrotising enterocolitis
 - however, premature labour is associated with a higher incidence of placental abruption, placenta praevia, prolapsed cord and infection
 - therefore, a trial of premature labour is frequently associated with foetal distress and the need for urgent LUSCS

Obstetric Anaesthesia

- the premature infant is more susceptible to the depressant effects of anaesthetic agents,
 - a. immaturity of the lungs, liver and kidneys, with slower metabolism and excretion
 - b. decreased plasma protein binding, plus increased bilirubin competing for protein binding → increased free fraction
 - c. greater permeability of the foetal blood-brain barrier
 - d. greater total body water & lower fat content
 - e. tendency to acidaemia, with the potential for "ion-trapping" of local anaesthetics
 - *though, the significance of these in humans has recently been questioned

■ Vaginal Delivery

- CEA is the anaesthetic of choice, as it provides the usual advantages,
 1. control of maternal pain & anxiety
 2. reduction in catecholamine levels
 3. decreased hyperventilation / hypoventilation
 4. preservation of maternal awareness & airway reflexes
 5. anaesthesia for instrumental delivery
 6. ready extension to anaesthesia for emergency LUSCS

plus

 7. it obviates the need for systemic opioids & other anaesthetic, to which the neonate is more sensitive
 8. relaxes the pelvic floor & reduces reflex expulsive efforts, thereby allowing a more controlled delivery
 9. there are few interactions between this and residual tocolytic therapy
- the usual guidelines / precautions apply, with the addition of,
 - a. due to the increased sensitivity to hypoxia, maternal hypotension should be treated earlier and more aggressively
 - b. diminution of reflex motor activity at the end of 2nd stage is desirable, in contrast to normal vaginal delivery, preservation of motor strength being less important

■ Caesarean Delivery

- a. emergency delivery for foetal distress
- b. electively for malpresentation, multiple gestation
- c. in the extremely premature to prevent birth trauma

NB: whenever time permits, **regional** anaesthesia is preferable to GA

- maternal **hypotension** is a greater risk with T₄ anaesthesia, cf. T₁₀ for vaginal delivery, and volume loading should commence prior to blockade
- careful hydration is required in patients having been on **b-adrenergic tocolytics**, due to the increased risk of **pulmonary oedema**
- hypotension is more common following rapid sympathectomy, therefore epidural is preferable to spinal anaesthesia, time permitting
- subarachnoid anaesthesia may be safely administered, providing meticulous attention is paid to volume status
- where foetal distress is present and an epidural is in situ, American texts recommend 2-chloroprocaine as the agent of choice, as the rapid plasma hydrolysis & lack of foetal ion-trapping reduce the likelihood of foetal toxicity

- **general anaesthesia** is usually recommended where there is foetal distress and regional anaesthesia is either contraindicated (time, infection, coagulopathy, etc.) or refused
- term infants delivered under GA are more likely,

- a. to be depressed at birth
- b. have low 1 minute apgars, and
- c. require active resuscitation

NB: these problems are amplified in the premature neonate, plus anaesthesia may be complicated by the recent use of tocolytic agents (see over) therefore,

1. induction-delivery time should be kept to a minimum
2. volatile concentrations should be kept to a minimum
3. neonatal oxygenation & ventilation should be employed to enhance elimination

NB: the use of minimal volatile agent & opioids is applicable, except where prematurity is such that neonatal intubation is to be undertaken, when "anaesthesia" of the neonate may be desirable

Obstetric Anaesthesia

Side Effects of Tocolytic Agents		
Drug	Maternal Effects	Foetal Effects
β-agonists	anxiety, nervousness nausea and vomiting hyperglycaemia hypokalaemia metabolic (lactic) acidosis hypotension, tachycardia chest pain, tightness arrhythmias pulmonary oedema, CCF	tachycardia foetal hyperglycaemia neonatal rebound hypoglycaemia increased free fatty acids foetal asphyxia (large doses) <ul style="list-style-type: none"> • maternal hypotension • increased uterine resistance
MgSO ₄	anxiety, nervousness nausea and vomiting flushing drowsiness blurred vision sensitivity to muscle relaxants pulmonary oedema, CCF	hypotonia drowsiness decreased gastric motility hypocalcaemia
Phosphodiesterase inhibitors	tachycardia, arrhythmias narrow therapeutic index hypotension tremor nausea & vomiting hyperglycaemia hypokalaemia	tachycardia hyperglycaemia rebound hypoglycaemia
Prostaglandin synthetase inhibitors	GIT irritation inhibition of platelet function reduction in factor XII depressed immune function	? decreased uterine blood flow
Diazoxide	tachycardia hypotension hyperglycaemia	decreased uterine blood flow hyperglycaemia tachycardia
Ethanol	CNS depression disorientation, agitation, headache nausea and vomiting hypotension gastric hypersecretion & acidity hypoglycaemia metabolic acidosis	foetal & neonatal CNS depression neonatal respiratory depression hypotonia metabolic acidosis hypoglycaemia temperature instability gastric irritation & vomiting foetal alcohol syndrome (withdrawal)

Tocolytic Agents

- currently the most commonly used agents are,
 - i. β_2 -adrenergic agonists
 - ii. $MgSO_4$
 - iii. prostaglandin synthetase inhibitors*
 - iv. Ca^{++} channel blockers* * less common
- ethanol has essentially been abandoned due to its undesirable side-effects
- similarly, the PDE inhibitors such as theophylline, have a narrow margin of safety and are less frequently used
- the common mechanism of action of these agents is a reduction in *intracellular calcium*, either by a direct effect or by an increase in cAMP

■ Beta-Adrenergic Agonists

- salbutamol, terbutaline and ritodrine are the commonly used agents
- although they are β_2 -*selective*, they still have significant β_1 activity which accounts for most of the undesirable effects
- tachycardia, often > 120 bpm, is common but will usually subside if anaesthesia can be delayed for >30 minutes after the last dose
- if GA is required, agents which exacerbate tachycardia (ketamine), or predispose to arrhythmias (halothane) should be avoided
- extreme tachycardia should be controlled with a short acting β -blocker (esmolol, atenolol, labetalol) prior to induction
- drug induced tachycardia makes judgement of anaesthetic depth & intravascular volume status difficult
- *pulmonary oedema* is seen in **5%** of patients receiving β -adrenergic tocolysis
- the cause of this is not completely understood, but risk factors include,
 1. pre-existing cardiac disease
 2. multiple gestation
 3. anaemia
 4. inappropriate resuscitation & overhydration
 5. prolonged therapy with β -agonists
 6. hypokalaemia
 7. sepsis
 8. ? combined therapy with $MgSO_4$
- concurrent use of glucocorticoids is no longer thought to be a risk factor
- the balance of opinion currently favours a *noncardiogenic* origin for this, in the absence of pre-existing cardiac disease

- **hypoxic pulmonary vasoconstriction** is impaired during β -adrenergic therapy, and this may account for the observation that **hypoxia**, out of proportion to the degree of pulmonary oedema is frequently seen
- peripheral vasodilatation will exacerbate the sympathectomy of epidural or spinal anaesthesia
- in the presence of pulmonary oedema, cautious volume expansion should be employed prior to blockade
- subarachnoid anaesthesia is relatively contraindicated under these circumstances
- if any doubt exists, then IVT should be governed by CVP or PCWP
- emergency anaesthesia for florid pulmonary oedema obviously requires GA with RSI \pm PEEP
- these patient may require postoperative ventilation \pm invasive monitoring in ICU

- in the setting of hypotension due to significant vasodilatation, rapid infusion of crystalloid is relatively contraindicated
- vasopressors with predominantly a β -effect, such as **ephedrine**, will have reduced efficacy and may exacerbate the tachycardia
- recent human studies have shown that in the setting of maternal hypotension, the use of virtually **pure α -agonists** (phenylephrine, metaraminol), **does not** produce any adverse neonatal outcome,

1. Apgar scores
2. acid-base balance
3. neurobehavioural examination

NB: these studies were in **term** pregnancies, still to be validated in preterm

- metabolic side-effects include,
 - i. increased glycogenolysis, lipolysis, and gluconeogenesis
 - ii. **hyperglycaemia** and raised plasma insulin
 - may require additional insulin in the diabetic
 - may be exacerbated by glucocorticoids for foetal lung maturation
 - neonatal **rebound hypoglycaemia** may be severe
 - iii. intracellular shift of K^+ , with **hypokalaemia**
 - normal total body stores, therefore no treatment is required
 - hyperventilation / alkalaemia will enhance hypokalaemia
 - hypoventilation / acidaemia increase arrhythmias

- myocardial ischaemia and infarction have been associated with β -adrenergic use, however are exceedingly rare in the absence of heart disease

- relative **contraindications** include,
 - i. asthma
 - ii. uncontrolled hypertension
 - iii. severe PIH
 - iv. significant cardiac disease (AS, MS, IHSS)
 - v. unstable diabetes mellitus
 - vi. hyperthyroidism
 - vii. history of migraine headaches

Magnesium Sulphate

- decreases uterine activity by membrane and intracellular competition with Ca^{++}
 - efficacy is comparable to that of ritodrine, though many side-effects are similar,
 - a. decrease in MAP * decreased PVR
 - b. tachycardia * reflex, not direct chronotropic cf. β -agonists
 - c. depression of myocardial contractility
 - d. myocardial conduction blockade
 - e. neuromuscular junction blockade
 - i. decreased ACh release from motor neurone
 - ii. decreased sensitivity of the motor endplate to ACh
 - iii. decreased excitability of the muscle membrane
 - **not** reliably antagonised by administration of Ca^{++}
 - f. postpartum uterine atony and haemorrhage
 - g. nausea, vomiting, flushing, drowsiness and blurred vision
 - toxicity is far more likely in the presence of abnormal **renal function**
 - in the absence of toxic plasma levels, the CVS effects are thought to generally be **less** than those of the β -agonists,
 1. pulmonary oedema is seen less frequently
 2. tachycardia is seldom significant
- NB:** however, some animal work suggests that Mg^{++} blunts the compensatory haemodynamic response to **haemorrhage** to a greater extent
- \ **hypotension** should be treated promptly with volume/ephedrine
- in the absence of a specific contraindication, regional anaesthesia for vaginal delivery and LUSCS is preferable, providing attention is paid to volume status
 - if general anaesthesia and nondepolarising muscle relaxants are used, administration should be titrated against a TOF response
 - the interaction between Mg^{++} and suxamethonium is unpredictable
 - however, as concerns for the airway far outweigh those of prolonged duration of action, the usual dose should be used

Clinical Manifestations of Hypermagnesaemia	
Plasma Level	Clinical Features
2.0-4.0 mmol/l	<ul style="list-style-type: none"> • anticonvulsant ?? • sedation • mild vasodilatation • increased AV & <i>intraventricular conduction</i>
~ 5.0 mmol/l	<ul style="list-style-type: none"> • loss of <i>monosynaptic reflexes</i> (DTR's) • increase in PR & QRS duration • hypotension • respiratory centre depression
~ 6.0 mmol/l	<ul style="list-style-type: none"> • NMJ blockade, severe weakness
6.0-8.0 mmol/l	<ul style="list-style-type: none"> • respiratory paralysis
8.0-12.0 mmol/l	<ul style="list-style-type: none"> • cardiac arrest (asystolic)

Calcium Channel Blockers

- *nifedipine* is the commonly employed agent and use is increasing
- found to be as effective as ritodrine but with fewer maternal and foetal side-effects
- Ferguson (1990) found doses sufficient for tocolysis had minimal CVS effects,
 1. decrease in MAP ~ 4% (~ 14%)
 2. tachycardia < 10% (~ 40%) (*ritodrine effect)
 3. mild negative inotropic effects are usually offset by baroreflex compensation
- recent doppler flow studies show there is no significant effect upon uteroplacental circulation, providing MAP is preserved (cf. original work with verapamil & hypotension)
- the metabolic effects of nifedipine are similarly mild compared to ritodrine,
 1. mild haemodilution 2° to vasodilatation
 2. plasma K⁺ remains unaltered
 3. glucose is minimally elevated cf. ritodrine * preferable in *diabetes*
- concomitant use of Mg⁺⁺ and has resulted in 1 documented case of difficulty swallowing
- theoretically additive effects but animal data is conflicting & no other case reports
- like other tocolytic therapy, may result in *uterine atony & PPH*
- this may be unresponsive to oxytocin or PGF_{2α}
- in the absence of overdosage and significant hypotension, both regional and GA are safe
- caution with the use of volatile agents is required due to augmentation of the negative inotropic effects and abolition of baroreflex compensation

Prostaglandin Synthetase Inhibitors

- PG's soften the cervix and stimulate the uterus to contract during labour, term & preterm
- prostaglandin synthetase inhibitors (PSI's) have been proven to be effective for the inhibition of preterm labour in multiple clinical trials
- maternal side-effects are *minimal* cf. other forms of tocolytic therapy,
 - i. GIT irritation - N&V, gastric erosion ± peptic ulcer
 - ii. impaired *platelet function*
 - iii. mildly impaired *renal function*
 - iv. suppression of low-grade fever ± masking of infection
 - v. oligohydramnios with prolonged use (indomethacin)
 - * has actually been used to treat idiopathic polyhydramnios

NB: widespread use is currently limited by potential adverse foetal effects, principally premature closure of the *ductus arteriosus*

- PG's are normally responsible for maintaining the patency of the duct in foetal life
- animal data suggests that the risks are greatest in late gestation
- a short course of indomethacin prior to 34 weeks gestation is probably safe for the foetus

NB: however, Moise (1990) demonstrated significant placental transfer and echocardiographic evidence of *premature duct closure* following short, low-dose therapy as early as 26 weeks

- of greater concern is the reversible inhibition of platelet TXA₂ by indomethacin
- platelet function may be impaired with administration within 24 hours, cf. aspirin within 10 days due to permanent inhibition
- most authors regard PSI usage as a *relative* rather than an absolute contraindication to regional blockade
- there have been 2 recent case reports of *profound hypertension* in women with PIH, treated with β-blockers, following administration of indomethacin for preterm labour

MULTIPLE GESTATION

- twins occur in ~ 1% of all pregnancies, triplets in ~ 0.01%
- increased maternal morbidity and mortality due to an increased incidence of,
 - a. preeclampsia / eclampsia
 - b. placental abruption & APH
 - c. premature ROM
 - d. preterm labour
 - e. uterine atony & PPH
 - f. obstetric trauma
 - g. operative delivery* both vaginal & LUSCS
- NB:** cf., singleton pregnancies, **caesarean delivery** \uparrow **50%**
perinatal mortality \sim **4-6x**
- multiple gestations accounts for ~ 10% of all perinatal mortality,
 - a. prematurity ~ 40% deliver before 37 weeks
 - b. congenital anomalies
 - c. polyhydramnios
 - d. umbilical cord entanglement*
 - e. umbilical cord prolapse* * greater for **monozygous** twins
 - f. intrauterine growth retardation
 - g. twin-twin transfusion*
 - vascular communications in **monochorionic placentas**
 - intrauterine & neonatal mortality up to 80%
 - h. malpresentation
- antepartum death occurs in 1 twin in ~ 0.5-6.8%
- morbidity in the other twin is common
- DIC may develop in the mother or surviving foetus

Anaesthetic Considerations

- multiple gestation exaggerates all of the physiological changes in pregnancy, especially,
 - a. **respiratory changes** - decrease in FRC and TLV, \uparrow MRO₂
 - b. tendency to relative or actual **anaemia**, cf. singletons
 - c. tendency to **aortocaval compression**, due to foetal mass and polyhydramnios
 - d. **gastric acid** production (gastrin from placenta) & LOS dysfunction

NB: renal, hepatic and CNS changes are similar to those in singleton pregnancies
- the high incidence of preterm labour increases the likelihood of recent **tocolytic** use
- neither regional nor general anaesthesia is clearly superior for abdominal delivery
- improved uterine relaxation with the volatile agents may aid in intrauterine manipulation for malpresentation
- there are clear benefits for the use of **epidural anaesthesia**, even though labour may be prolonged and the incidence of forceps delivery increased,
 1. the biochemical status of both twins is improved
 2. perineal relaxation and maternal cooperation decrease foetal and maternal trauma
 3. rapid conversion to abdominal delivery

NB: abdominal delivery of the 2nd twin is required in ~ 4-8% where the 1st is delivered vaginally

■ Labour

- CEA is the preferred method for pain relief in labour, with attention to,
 - a. adequate hydration prior to blockade
 - b. frequent monitoring & prompt treatment of hypotension
 - c. continuous foetal heart monitoring
 - d. when lower concentration solutions are used during labour, a top-up with a stronger solution prior to delivery is advisable due to the high incidence of operative delivery

■ Vaginal Delivery

- although CEA does facilitate external or internal version of the second twin, it **does not** provide uterine relaxation
- therefore, provision for the administration of a GA must always be considered
- oxygen should always be administered to the mother to maximise foetal wellbeing

■ Abdominal Delivery

- LEA is probably preferable to subarachnoid anaesthesia, due to the propensity for aorto-caval compression, rapidity of sympathetic blockade & risks of hypotension with the later

BREECH DELIVERY

- three major concerns for anaesthesia,
 1. prematurity
 2. risk of traumatic delivery
 3. coexisting foetal / maternal illness or abnormality
- overall ~ 70-84% of vaginal deliveries are successful
- **cord prolapse** occurs in ~ 2% of frank breeches at term
- CEA has both advantages and disadvantages,
 - a. usually does not prolong the 1st stage if initiated during **active labour**
 - b. the 2nd stage may be prolonged, however, full breech extraction and perinatal morbidity are **not increased**
 - c. relaxation of the pelvic floor may lessen trauma to the foetal head
 - d. surgical anaesthesia can rapidly be achieved in the event of foetal distress

■ Labour & Vaginal Delivery

- CEA is preferable for the above reasons
- administration of a lipid soluble **opioid** to the solution may decrease the "rectal pressure" frequently experienced during breech presentation
- the use of opioids should however be limited with a premature infant
- some advocate "delaying" blockade of the sacral segments, thus preserving the effectiveness of the maternal "pushing" effort
- GA following RSI is indicated if uterine relaxation is required for full breech extraction
- doses up to 2MAC are required and there is no difference in the relaxation afforded by halothane, enflurane or isoflurane
- following delivery volatile should be discontinued and an opioid based technique employed
- should incision of the cervix be required for extraction, blood loss may be severe and frequently occurs into the pelvis
- therefore, vaginal losses **underestimate** the degree of haemorrhage

■ Caesarean Delivery

- this may be accomplished with either CEA or GA, however, provision for administration of GA must be available if uterine relaxation is necessary
- **external version** may be attempted prior to the onset of labour, however this carries the risks of,
 - a. foetomaternal haemorrhage
 - b. placental abruption
 - c. umbilical cord compression
 - d. intrauterine foetal death

AMNIOTIC FLUID EMBOLISM

- rare cause of maternal mortality
- first reported by Meyer in 1926, then subsequently in animal work by Warden in 1927
- clinical importance established by Steiner and Lushbaugh in 1941
 - a. incidence ~ 1:8,000 to 1:80,000
 - b. **mortality** ~ **86%**
 ~ 25-50% within the first hour
 - c. aetiology
 - i. predisposing factors - advanced maternal age
 - multiple pregnancies (majority > 3)
 - foetal macrosomia
 - short duration labour with intense contractions
 - oxytocic stimulation (intact membranes)
 - ii. associated factors - foetal demise (~ 40%)
 - meconium stained liquor
 - amniotomy, amniocentesis
 - PIH
 - placenta praevia & placental abruption (~ 50%)
 - LUSCS
 - pregnancy with an IUCD
 - uterine rupture or cervical tears

■ Pathophysiology & Clinical Picture

- most cases occur during a vigorous labour, frequently with oxytocic stimulation
- the association with oxytocics is probably incidental, given their widespread use and the rarity of AFE
- classical descriptions are of the unheralded onset of **shock, cyanosis, & coagulopathy**, typically in a multiparous patient

- the 2 life-threatening consequences of AFE are,
 1. **cardiopulmonary collapse**
 - acute pulmonary hypertension cor pulmonale & RVF
 - V/Q mismatch, hypoxia, hypercarbia & acidaemia, further increasing PVR
 - decreased LV preload & LV output, with peripheral vascular failure
 2. **DIC**
 - aetiology disputed
 - potent thromboplastic activity of amniotic fluid, deposition of fibrin clots and activation of fibrinolysis → hypofibrinogenaemia & coagulopathy
 - the thromboplastic activity of trophoblastic tissue is well known and may play an integral role
 - amniotic fluid collected during labour has greater toxicity in animal models, however, the exact mediator is uncertain - ? PG's, leukotrienes
 - toxicity of amniotic fluid is greatly dependent upon the *particulate content*
 - this is especially true for meconium, ? *anaphylactoid* response, however,
 - i. *absence* of pruritis, urticaria & bronchospasm
 - ii. requires "sensitisation", evidence for which is frequently lacking
- the most significant pathological findings are those in the lungs,
 1. pulmonary oedema ~ 70%
 2. alveolar haemorrhage
 3. pulmonary embolisation with amniotic materials
- prodromal symptoms of AFE include the sudden onset of,
 - a. chills & shivering
 - b. sweating, anxiety
 - c. coughing, followed by signs of respiratory distress

NB: these are followed by shock, cardiovascular collapse and convulsions
- respiratory difficulty is manifest by,
 - a. cyanosis & tachypnoea*
 - b. bronchospasm (?)
 - c. pulmonary oedema

NB: *2° to *hypoxia*, which is also the cause of the *convulsions*

- the definitive diagnosis is usually made at autopsy, however additional diagnostic aids include,
 - a. CXR
 - enlargement of the RA & RV
 - prominent proximal PA (cf. embolism)
 - pulmonary oedema (not seen in PTE, cf. the former)
 - b. Lung scan
 - isolated perfusion defects
 - c. CVC catheter
 - initially raised pressures 2° to pulmonary hypertension
 - later pressures may be low 2° to haemorrhage
 - d. CBP / Coag's
 - coagulopathy & anaemia later
 - cf. the normal procoagulant state of pregnancy

■ Differential Diagnosis

- a. pulmonary thromboembolism
 - more common postdelivery
 - chest pain is a more common finding
- b. air embolism
 - similar except for doppler / auscultation
- c. aspiration of gastric contents
 - temporal relationship to general anaesthesia
- d. eclamptic convulsions
 - presence of hypertension & proteinuria
- e. local anaesthetic toxicity
 - temporal relationship & dose administered
- f. acute LVF
 - presence of pre-existing heart disease
- g. cerebrovascular accident
 - no cyanosis or coagulopathy
- h. haemorrhagic shock
 - abruptio placentae, placenta praevia
 - ruptured uterus

Anaesthetic Management

- no specific therapy has been proven effective, management is supportive only
 - a. **respiratory support**
 - i. high FiO₂ by face mask
 - ii. CPAP
 - iii. intubation & ventilation
 - 100% O₂
 - ± PEEP, depending upon cardiac output
 - iv. treatment of bronchospasm
 - b. **cardiovascular support**
 - i. left uterine displacement
 - ii. IV access
 - 2 large gauge cannulae
 - iii. CVP / PCWP guided volume resuscitation
 - pulmonary oedema is variably ascribed to excessive volume therapy
 - iv. inotropic support of MAP
 - v. treatment of pulmonary vasospasm ? inhaled NO
 - c. **treatment of DIC**
 - i. fresh whole blood, or packed cells plus FFP
 - ii. cryoprecipitate
 - several reports of improvement (? fibronectin)
 - * fibrinogen may act only to perpetuate coagulation
 - iii. platelets
 - iv. heparin
 - * controversial, not routinely recommended
 - d. other
 - i. uterine massage and oxytocic stimulants (oxytocin ± methylergonovine)
 - ii. PG's for control of uterine haemorrhage
 - * may result in **bronchospasm** and/or **pulmonary hypertension**
 - iii. **aprotinin** for control of lysis prior to delivery
 - * aprotinin doesn't cross the placenta & EACA is teratogenic

THROMBOEMBOLIC DISEASE

■ Incidence

- ~ 0.05-1.8% of pregnancies (1:50-2000)
- ~ 5x more common during pregnancy or postpartum period
- ~ 3-6x more common *postpartum* cf. antepartum
- ~ 3x more common with LUSCS cf. vaginal delivery
- ~ 12% risk of repeat episode in the same pregnancy
- ~ 5-10% risk during subsequent pregnancies

■ Aetiology

- * three classically described factors,

1. *vessel wall trauma*
2. *venous stasis*
 - increased venous distensibility during first trimester
 - aortocaval compression from the second trimester
 - bed rest post-partum and with complications of pregnancy
3. *altered coagulation status*
 - increases in all plasma coagulation proteins, except XI & XIII
 - decreased fibrinolytic activity
 - * depressed levels of plasminogen activators
 - * increased concentration of soluble fibrin-fibrinogen complexes
 - decrease in antithrombin III, especially with hereditary ATIII deficiency
 - neither the platelet count, nor platelet adhesiveness is increased

NB: other risk factors,

- i. increased maternal age
- ii. obesity
- iii. caesarean delivery
- iv. prolonged bed rest
- v. oestrogen therapy to suppress lactation
- vi. blood group other than *type O*
- vii. * antithrombin III deficiency (autosomal dominant)

■ Pathophysiology

- physiological disturbance depends upon,
 - a. the quantity and size of the embolus
 - b. the site of obstruction
 - c. the presence of pre-existing cardiopulmonary disease
- clinical syndromes range from,
 - a. asymptomatic
 - i. small multiple emboli traversing the pulmonary arteries, with subsequent lysis and no adverse haemodynamic consequences
 - ii. isolated small emboli producing subsegmental obstruction, with subclinical alteration of V/Q matching
 - b. chronic recurrent pulmonary emboli
 - with pulmonary hypertension, leading eventually to cor pulmonale and RVF
 - c. moderate-large single, or multiple emboli
 - i. clinically symptomatic - tachycardia, dyspnoea, mild fever, chest pain
 - ii. significant V/Q mismatch - hypoxaemia, \uparrow A-a gradient
 - iii. increased RV afterload / decreased LV preload
 - d. massive embolus
 - rapid onset of dyspnoea, hypoxaemia, chest pain, hypotension, RVF and/or sudden death
- the principal physiological derangement's with significant embolism are,
 - a. increased RV afterload
 - b. increased V/Q mismatch
 - i. increased V_D \rightarrow dyspnoea & tachypnoea
 - ii. increased Q_S
 - due to loss of *surfactant* and local mediator release
 - * *hypoxaemia* is frequently *not* totally corrected by O_2 administration
 - c. decreased LV preload and CO with systemic hypotension
- right coronary blood flow does *not* usually decrease following PTE, rather it increases due to autoregulation

Diagnosis

- many of the clinical signs of DVT can be present in normal pregnancy
 - a. **venography**
 - where PTE is suspected is the most sensitive & specific test
 - suboptimal for detecting deep femoral or pelvic vein thromboses
 - there may be false positives with external vein compression, or poor technique
 - b. **doppler sonography**
 - sensitivity ~ 90% and is most useful for popliteal, femoral and pelvic thrombi
 - however, far less sensitive at detecting thrombi below the knee
 - because of collateral venous channels ~ 50% of small calf thrombi are missed
 - c. impedance plethysmography
 - d. thermography
 - e. fibrinogen scanning (^{125}I)
 - * contraindicated in pregnancy (\rightarrow foetal thyroid)
 - * similarly C/I in lactating mothers
 - f. radionuclide venography ($^{99\text{m}}\text{Tc}$) - low risk to the foetus
 - ~ 90% sensitivity
 - g. **lung V/Q scan**
 - safe during pregnancy, though, $^{99\text{m}}\text{Tc}$ should be used with uterine shielding ??
 - a perfusion defect with normal ventilation is adequate for treatment
 - serious morbidity occur in ~ 2-4% of those having angiography
 - h. pulmonary angiography - usually avoided due to the radiation hazard to the foetus

Clinical Presentation

- the first sign of DVT may be PTE, and the manifestations of PTE may be nonspecific or absent,
 - i. shortness of breath, dyspnoea, cough \pm haemoptysis
 - ii. chest pain, substernal tightness
 - iii. apprehension, altered sensorium
 - iv. sweating
 - v. syncope
 - vi. tachycardia
 - vii. CXR:
 - usually normal
 - diminished vascular markings ("cut-off" sign)
 - elevated hemidiaphragm
 - pleural effusion
 - viii. ECG:
 - sinus tachycardia frequently the only abnormality
 - RV strain: RAD, tall R in V_1 , rarely $S_1-Q_3-T_3$
 - arrhythmias

NB: CXR & ECG are frequently *normal*, their main use is to rule out other pathology

Obstetric Anaesthesia

- if the patient is on **heparin** at the time of labour, management is simpler,
 1. heparin **does not** cross the placenta, thus the risk of foetal haemorrhage is low
 2. the half-life is short, so if delivery is not anticipated for > 4-6 hours, there is no need to reverse the anticoagulation
 3. if emergency LUSCS is required the heparin can be acutely reversed with protamine
 - approximate dose → **protamine ~ 1 mg / 100^U heparin**
- because of the ease of management, some advocate the use of heparin in these patients up to the time of delivery (150-250^U/kg q12h)
- **thrombolytic agents** are presently contraindicated during pregnancy
- tPA may be associated with a lower risk of haemorrhagic complications and may be useful under these circumstances
- **surgical management** is also limited during pregnancy, procedures used including,
 1. femoral vein or vena caval interruption
 2. thrombectomy
 3. embolectomy * ~ 80% mortality in nonpregnant patients

Anaesthetic Management

- management is dependent upon when PTE develops, ie. perinatal, during labour, vaginal or caesarean delivery, or in the postpartum period
- excepting acute resuscitation, the main concern of anaesthesia is the presence of **anticoagulation**
- there have been a number of reports looking at central neuraxis techniques,
 1. Owens *et al.* - Anesth & Anal. 1986
 - 33 cases of spinal haematoma following lumbar puncture or spinal anaesthesia
 - 6 cases with anaesthesia & 27 following diagnostic lumbar puncture
 - 40% (13) had received anticoagulant therapy (heparin, warfarin or both)
 2. Odoom & Sih - Anaesthesia 1983
 - over 1000 epidural blocks in 950 patients for vascular surgery
 - all received oral anticoagulation preoperatively & the majority received heparin intraoperatively
 - ~ 10% suffered postoperative backache, but there was no clinical evidence of epidural haematoma, or neurological complications
 3. Rao & El-Etr - Anesthesiol. 1981
 - total of 3164 epidural and 847 continuous spinal anaesthetics
 - all patients received heparin **after** the start of anaesthesia, with the ACT at twice the baseline
 - they reported **no** incidence of epidural haematoma
 - however, procedure abandoned if "bloody tap", **∴ not applicable** to pregnancy

Obstetric Anaesthesia

- the incidence of epidural vein cannulation has been reported between 1-10%
- even in patients on "*mini-dose*" *heparin* there are no prospective case reports which provide assurance that central neuraxis techniques are safe
- there may be some advantage for spinal anaesthesia for those at risk of PTE,
 1. Modig *et al.* - Anesth & Anal. 1983
 - compared the incidence of DVT and PTE in total hip replacement patients using GA or epidural anaesthesia continued for 24 hours postoperatively
 - GA group → DVT ~ 67% and PTE ~ 33%
 - CEA group → DVT ~ 13% and PTE ~ 10%
 2. McKenzie *et al.* - BJA 1985
 - incidence of DVT following repair of femoral neck fractures
 - reduced from ~ 76% to ~ 40% with subarachnoid anaesthesia
 3. a similar benefit for general or thoracic surgical procedures has **not** been demonstrated
- the major effect is probably the increase in **limb blood flow** secondary to sympathetic blockade
- in contrast to spinal anaesthesia, GA definitely reduces limb blood flow
- there may also be a reduction in blood viscosity due to **haemodilution** with volume expansion
- Writer (in James book: "Obstetric Anaesthesia: the Complicated Patient") makes the following recommendations,
 1. restrict regional techniques to those receiving heparin no more frequently than b.d.
 2. prior to performance of the block the APTT & PT must be normal
 3. utilise the left lateral position to avoid aortocaval compression and distension of the epidural veins
 4. use a midline technique, as a lateral approach is more likely to lacerate an epidural vein
 5. abandon the procedure if a bloody tap occurs
- further recommendations include,
 1. allow the block to wear off at intervals to allow neurological assessment
 2. should anticoagulation be instituted following placement of the catheter, then this should be left in place until anticoagulation is reversed, or normalised
 3. following catheter removal, frequent neurological assessment is required
 4. should a clinically detectable epidural haematoma occur, resolution is **unlikely** without surgical intervention

NB: Cousins & Bromage state that decompression within **12 hours** offers the best chance of recovery of normal function

VENOUS AIR EMBOLISM

• **precordial doppler** can detect as little as **0.1 ml** of intracardiac air & the correlation with TEE during caesarean section is ~ 100%

• the **incidence** of Doppler VAE during LUSCS is reported from,

- a. 11 to 66% for epidural anaesthesia
- b. 28 to 71% for general anaesthesia

NB: this may occur at any stage throughout the procedure, however is most likely to occur during **hysterotomy**, or repair of hysterotomy

■ Aetiology

- a. pneumoperitoneum for laparoscopy or hysteroscopy
- b. LUSCS - especially with exteriorisation of the uterus
- c. surgery involving major veins * classically sitting neurosurgery
- d. central venous cannulation
- e. pump infusions - CPB
- haemofiltration / dialysis
- f. orthopaedic surgery - especially THR

■ Contributing Factors

- a. **venous pressure gradient**
 - gradients as small as $-5 \text{ cmH}_2\text{O}$ have been associated with significant entrainment
 - exteriorisation of the uterus increases this gradient & distends collapsed veins
 - decreased CVP,
 - i. absolute hypovolaemia from haemorrhage (placenta praevia or abruption)
 - ii. prolonged labour with NPO status
 - iii. pregnancy induced hypertension
 - iv. regional anaesthesia with inadequate volume expansion
- b. **posture**
 - routine positioning with lateral tilt produces a gradient $\geq -10 \text{ to } -15 \text{ cmH}_2\text{O}$
- c. **volume & rate** of entrainment
 - small volumes entrained slowly are usually asymptomatic
 - i. $\geq 0.5 \text{ ml/kg/min}$ → results in symptoms
 - ii. $\geq 2 \text{ ml/kg/min}$ → generally fatal
- d. presence of an ASD * probe patent foramen ovale in ~ 10-25%

■ Associated Problems

- a. increased PA pressure
- b. increased alveolar dead space and P_{A-aO_2} gradient
- c. acute RV failure
- d. systemic hypotension and tachycardia
- e. hypoxia
- f. arrhythmias, cardiac arrest
- g. systemic embolisation * coronary or cerebral

NB: the most likely cause of rapid death following massive embolisation is mechanical obstruction to RV outflow

■ Monitoring

- a. oesophageal stethoscope ~ 1.8 ml/kg/min
- b. systemic hypotension ~ 0.7 ml/kg/min
- c. ECG/tachyarrhythmias ~ 0.6 ml/kg/min
 - NB: ~ 0.5 ml/kg/min required for clinical symptoms
- d. End Tidal CO₂ ~ 0.42 ml/kg/min
- e. PA pressure rise ~ 0.42 ml/kg/min
- f. continuous CVP ~ 0.4 ml/kg/min
- g. **doppler precordial stethoscope** ~ **0.02 ml/kg/min** (1.5 ml/min)
- h. transoesophageal echocardiography ~ 5-10x more sensitive than doppler

■ Clinical Presentation

- massive VAE, with EMD, hypotension, hypoxaemia and cardiac arrest is **infrequent**
- only ~ 1% of maternal deaths are attributed to VAE
- the routine picture is less profound, presumably due to the slow rate of entrainment
- the aetiology of **chest pain** during LUSCS is unclear & probably multifactorial, however,
 - a. 20-50% of women **with** doppler VAE will complain of chest pain
 - b. < 2% **without** doppler VAE will complain of chest pain
 - c. SpO₂ < 92% has been reported in up to 25% with doppler VAE
 - d. **dyspnoea** is present in 20 to 40% with doppler VAE

NB: dyspnoea and SpO₂ < 92%, without doppler VAE is unusual

■ Management

- given the low incidence of significant morbidity or mortality during LUSCS, the use of precordial doppler in all cases would seem excessive
- however, cases at risk should be considered,
 - a. hypovolaemic patients
 - b. pre-existing intracardiac shunt

- symptomatic doppler evident VAE, or strongly suspected clinical VAE should be managed as follows,
 - a. prevent further air embolisation
 - flood the operative field with saline if practicable
 - return the uterus to the abdominal cavity
 - b. 100% FiO₂ (ie. cease N₂O)
 - c. usual recommendation is **right lateral** position, however,
? maintain left lateral tilt, as this decreases chance of RV outflow obstruction
 - d. IV volume expansion
 - e. place a **multiorifice CVC** line and attempt to aspirate air
 - f. if cardiovascular collapse occurs → immediate delivery of the baby
 - g. drugs
 - pulmonary vasodilators
 - inotropes / vasoconstrictors ??
 - antiarrhythmics
 - h. others
 - i. thoracotomy
 - ii. intracardiac needle aspiration
** must get RV & always get a **pneumothorax**

NB: any patient who becomes **comatose**, or fails to waken following GA, should have a CT head to exclude cerebral air embolism, as prompt management with **hyperbaric oxygen** is indicated

CARDIAC DISEASE

Valvular Heart Disease

- the prevalence of heart disease during pregnancy ranges from 0.4 to 4.1%
- where possible, patients with surgically correctable lesions should have these performed prior to pregnancy
- all patients with valvular lesions should have *antibiotic prophylaxis* prior to operative procedures

Mitral Valve Prolapse

- prolapse of one or more of the MV leaflets (usually posterior) into the LA during systole
- estimated to have an incidence of ~ 5% in the general population and up to **20%** in pregnancy
- clinical features include,
 - a. a midsystolic snap
 - b. a late systolic, bruit best heard at the apex
 - c. classically thin and tall patient, who may possess other Marfanoid features
- the diagnosis is usually suspected from auscultation and confirmed by echocardiography
- the aetiology is unknown, but thought to involve autosomal dominant inheritance, with reduced male expression
- few are symptomatic and even fewer are on medical therapy, usually for TIA's or frequent PCV's
- sudden death is commonly discussed but exceedingly rare and results from arrhythmia
- if severe these patients are managed as for mitral regurgitation (see below)

Mitral Regurgitation

- the second most common valvular anomaly during pregnancy
 - a. soft S₁ and a widely split S₂
 - b. holosystolic murmur, best heard at the apex, radiating to the axilla
- the increased blood volume of pregnancy is usually helpful for the MR patient
- in chronic MR, in contrast to acute MR,
 - a. the LA dilates to accommodate the regurgitant volume
 - b. LA stretch may result in AF
 - may precipitate pulmonary oedema, with a maternal mortality ~ 17%
 - may result in systemic embolisation
 - c. the LV dilates and hypertrophies with the LA
 - d. LVEDP increases
 - e. LVEF decreases

Obstetric Anaesthesia

- techniques which decrease afterload are preferable, as they decrease the regurgitant fraction and increase forward cardiac output
- therefore, CEA is the preferred anaesthetic technique
- as these patients are **preload dependent**, they require
 - a. adequate volume expansion prior to establishment of regional block
 - b. absolute avoidance of aortocaval compression
- if **general anaesthesia** is to be used, then the following principals apply,

NB: "full, fast and loose"

- a. factors decreasing the regurgitant fraction,
 - decreasing afterload
 - vasodilators
 - regional anaesthesia
- b. factors increasing the regurgitant fraction,
 - increased afterload
 - increased SNS tone (pain, hypoxia, hypercarbia, acidosis)
 - slow HR
 - N₂O
- c. volume expansion prior to induction, and prompt replacement of blood loss

Mitral Stenosis

- this is the most common of the **rheumatic** valvular lesions in pregnancy

■ Pathophysiology

- a. diastolic pressure gradient LA-LV determined by
 - mitral valve area
 - mitral valve flow
 - b. increased LAP, pulmonary venous pressure ± pulmonary oedema
 - c. passive, reversible pulmonary hypertension, increased PVR
 - irreversible pulmonary hypertension later
 - d. decreased CO
 - e. reduced LV filling & **LV dysfunction**
- causes of sudden deterioration include,
 - i. AF
 - ii. fever, infection
 - iii. exercise
 - iv. pregnancy
 - v. SBE

■ Symptoms

- a. dyspnoea, orthopnoea, PND
- b. acute pulmonary oedema
- c. haemoptysis - may be severe
- d. recurrent respiratory infection
- e. fatigue - decreased CO, development of PAH
- f. chest pain ~ 10%
- g. systemic thromboembolism

■ Clinical Signs

- a. malar flush, peripheral cyanosis
- b. small volume pulse \pm AF
- c. normal JVP, loss of 'a' wave
- d. heart:
 - tapping apex beat
 - palpable RV impulse & loud P₂
- e. auscultation:
 - opening snap
 - mid-diastolic rumble (supine \pm left lateral)
 - loud S₁ \pm S₃

■ Investigations

- a. ECG:
 - P mitrale \pm AF
 - RV hypertrophy (PAH) \pm "strain"
- b. CXR:
 - enlarged LA
 - pulmonary venous congestion
 - Kerley B lines \pm pulmonary oedema
 - large pulmonary outflow tract
 - mitral valve calcification (lateral - below diagonal)
- c. Echocardiography
 - assessment of severity
 - exclusion of atrial myxoma
 - LA size and presence of thrombus
 - LV size and function
 - RA/RV size & function
- d. Catheterisation
 - MV area & pressure gradient
 - PVR and pulmonary hypertension
 - LV function
 - coronary artery anatomy
 - other valvular lesions

■ Clinical Assessment of Severity

- a. systolic BP and pulse volume
- b. severity of dyspnoea
- c. signs of PAH
 - RV heave & loud P₂
 - elevated JVP
- d. murmur
 - duration of murmur ∝ degree of stenosis
 - interval between S₂-OS (↓ = higher LAP & worse)
- e. loud S₁ and OS represent *pliable valve*
- f. CXR:
 - calcification
 - LAH, LVH, PA prominence
 - CCF

Treatment

■ Medical

- i. SBE prophylaxis
- ii. AF
 - digoxin ± quinidine
 - cardioversion
- iii. systemic emboli
 - warfarin
- iv. dyspnoea
 - diuretics, fluid restriction, low Na⁺ diet

■ Surgery

- i. commisurotomy
- ii. valve replacement

■ Anaesthetic Considerations

- Ostheimer recommends using a *continuous epidural* technique, raising the sensory level of anaesthesia slowly, thereby avoiding cardiovascular changes
- this requires adequate, but cautious volume expansion prior to establishment of blockade and prompt treatment of hypotension with vasopressors (ephedrine / metaraminol)
- this technique would seem relatively contraindicated due to the requirement to maintain preload & afterload
- therefore, the emphasis is on *gradual* onset of blockade
- one shot spinal anaesthesia is absolutely contraindicated

- Chesley claims that the mortality of parturients with MS is equal to that of those who have never conceived
- *atrial fibrillation* is a common sequelae of MS and *does not* result in an increased mortality in either pregnant or nonpregnant patients (Sullivan & Ramanathan NEJM 1985)

■ Anaesthetic Considerations - General

NB: "full, slow and tight"

- a. primary goal is to maintain a slow HR
 - rates > 110 are poorly tolerated
 - avoid anticholinergics, sympathomimetics, vasodilators (reflex)
 - with recent onset AF consider DC cardioversion, or digoxin to control rate
- b. relatively fixed CO
 - i. maintain SVR, avoid vasodilatation
 - ii. maintain preload
 - within the constraints of pulmonary congestion
 - rapid infusions may precipitate AF or acute pulmonary oedema
- c. avoid pulmonary vasoconstriction
 - hypoxia, hypercarbia, acidosis

AORTIC STENOSIS

- a. **aetiology**
 - rheumatic
 - congenital bicuspid valve
 - calcific or degenerative
- b. **pathophysiology**
 - i. normal valve area ~ 2.5 - 3.5 cm²
 - ii. fixed low output state
 - iii. chronic pressure overload
 - concentric LVH
 - LV failure / decompensation
 - iv. increased LV mass
 - v. LV / aortic root pressure gradient
 - vi. decreased ejection fraction and CO
 - vii. increased LVEDP, eventually increased LAP
 - viii. increased PCWP
 - ix. eventually pulmonary hypertension
- c. **symptoms** * late onset and indicate *severe* stenosis
 - i. angina
 - life expectancy ~ 5 yrs
 - ~ 50% have CAD
 - ii. effort syncope
 - life expectancy 3-4 yrs
 - eventually LVF ± arrhythmias
 - iii. SOBOE
 - life expectancy 2 yrs

■ Physical Examination

- a. pulse - regular, slow upstroke, plateau, small volume
- b. BP - narrow pulse pressure
- c. heart - LV impulse + presystolic lift (S_4)
 - sustained, basal systolic thrill
 - harsh SEM → carotids
 - decrease in A_2/P_2 + reverse splitting
 - * normal heart size until late

NB: AS + *cardiomegaly* → AI, MI, CCF & severe end-stage disease

■ Problems

1. the murmur may disappear with the development of LVF
2. the pressure gradient is low with LVF
3. in the elderly
 - murmur is often louder at the apex/LSE
 - arteriosclerosis obscures pulse changes
 - other causes of LVF are common

■ Investigations

- a. ECG: - SR, LVH ± strain, LBBB ~ 10%
- b. CXR:
 - normal heart size
 - convex LV border
 - dilated ascending aorta (post-stenotic)
 - valve calcification
- c. Echo:
 - AV disorganisation, LVH
 - LV size and contraction
 - LA size
 - **not** good at quantifying severity
- d. Catheterisation:
 - assessment of LV function and other valves
 - coronary anatomy

Catheter	AV gradient	AV size
normal	~ 0 mmHg	2.5-3.5 cm ²
mild	0-25 mmHg	1.2-2.0 cm ²
moderate	25-50 mmHg	0.8-1.2 cm ²
severe	> 50 mmHg	< 0.8 cm ²

■ Medical Treatment

- a. SBE prophylaxis
- b. digoxin & diuretics for LVF
- c. balloon dilatation
- d. vasodilators are **contraindicated**, except in severe LVF
- e. cardioversion for sudden onset AF

■ Anaesthetic Considerations

NB: "full, normal rate & tight"

- these patients tolerate the increase in plasma catecholamines better than MS/MI patients
- again, Ostheimer will use a slow onset epidural block, though, decreases in afterload are contraindicated
- spinal anaesthesia remains contraindicated
- **hypotension** should be treated aggressively, using predominantly α_1 -agonists (metaraminol) rather than ephedrine
- some recommend GA using thiopentone/volatile/N₂O/vecuronium
- irrespective of the technique used, the following are relevant,
 - a. good IV access * 2 x 16G or larger cannulae
 - b. ECG with II + V₅ to monitor for ischaemia
 - c. prevent ischaemia * avoid AF, loss of atrial contribution to LV filling
 - avoid tachycardia/bradycardia
 - avoid decrease SVR
 - d. maintain - **sinus rhythm**, HR ~ 70-80 bpm
 - preload and SVR

Aortic Regurgitation		
	Acute	Chronic
Aetiology:	<ul style="list-style-type: none"> • SBE • aortic dissection • traumatic 	<ul style="list-style-type: none"> • rheumatic • syphilis • Marfan's • SBE • RA, psoriasis, Reiter's • UC, Crohn's, ankylosing sp. • myxomatous degeneration
Symptoms:	<ul style="list-style-type: none"> • abrupt onset • pulmonary oedema • cardiogenic shock 	<ul style="list-style-type: none"> • asymptomatic period • palpitations • fatigue • SOBOE • angina (5-10%)
Signs:	<ul style="list-style-type: none"> • rapid low volume pulse • hypotension • <i>normal</i> heart size • soft or absent S₁ • loud S₃ • EDM (soft) 	<ul style="list-style-type: none"> • 'water hammer' pulse • low diastolic pressure • LV enlargement • EDM at LSE • ESM with high CO • apical MDM (Austin Flint)
ECG:	<ul style="list-style-type: none"> • normal ± ischaemia 	<ul style="list-style-type: none"> • LVH
CXR:	<ul style="list-style-type: none"> • LVE, pulmonary oedema • dilated aorta 	<ul style="list-style-type: none"> • increased LV & aortic shadow

• predominance of AI / AS determined by,

1. pulse characteristic
2. pulse pressure
3. heart size

• clinical severity is determined by,

- a. pulse character - bounding, collapsing, bisferens
- b. BP - systolic > 140 & diastolic < 60
- c. cardiomegaly
- d. LV heave
- e. Austin-Flint murmur
- f. ECG - LVH & strain
- g. loudness of the murmur is *not* a useful guide
- h. assessment of severity is via *echocardiography* and *catheterisation*

■ Anaesthetic Management

NB: "full, dilated and fast"

- a. maintain a HR > 80 bpm, with a low SVR
- b. avoid bradycardia & vasoconstriction
- c. regional anaesthesia, with sympathetic blockade, is the technique of choice
- d. if hypotension develops, *ephedrine* is the agent of choice, as it will tend to increase the HR in addition to vascular tone

IDIOPATHIC HYPERTROPHIC SUBAORTIC STENOSIS

■ Features Include

- a. hypertrophic cardiomyopathy
- b. marked asymmetrical septal hypertrophy
- c. autosomal dominant inheritance (~ 50% familial)

■ Pathophysiology

- a. anatomical septal hypertrophy
- b. markedly reduced LV compliance
- c. increased LAP
- d. hypercontractile LV
- e. dynamic subaortic muscular stenosis
- f. systolic anterior motion of anterior MV leaflet, occasionally with MI

■ Symptoms

- a. exertional angina
- b. effort syncope
- c. palpitations
- d. SOBOE

■ Clinical Signs

- a. sharp upstroke, often bifid pulse
- b. ESM at the LSE and apex, increased by valsalva manoeuvre
- c. MR in 50%
- d. normal S_1 and normal or split S_2
- e. $\pm S_3$ and S_4

■ Complications

- a. sudden death, syncope
- b. arrhythmias
- c. LVF *murmur decreases markedly
- d. angina

■ Exacerbating Factors

- a. increased contractility
 - sympathomimetics / endogenous catecholamines
 - digoxin & (+)'ve inotropes
 - tachycardia
- b. reduced preload
 - ↓ LVEDV & LVESV with outflow obstruction
 - hypovolaemia
 - venodilators (GTN)
- c. reduced afterload
 - ↓ LVESV & ↑ fibre shortening
 - vasodilators
 - regional sympathectomy (*spinal / epidural)

■ Factors Decreasing Dynamic Obstruction

- a. decreased contractility
 - β adrenergic blockers
 - Ca^{++} entry blockers
 - volatile anaesthetics
- b. increased preload
 - hypervolaemia (cf. increased volume of pregnancy)
 - bradycardia
- c. increased afterload
 - vasoconstrictors
 - metaraminol, phenylephrine

■ Investigations

- a. ECG: - LVH + strain changes
 - septal Q-waves simulate AMI
 ± LA hypertrophy
- b. CXR: - often no LVF or cardiomegaly
- c. Echo: - anterior septal hypertrophy
 - ratio of septum:free wall ~ **1.5:1**
 ± increase in size of LA

■ Treatment

- a. β -adrenergic blockade
 - usually administered to all pregnant patients chronically
 - reduce contractility, decrease outflow obstruction
 - the slower HR allows increased diastolic filling which increases the LVEF
- b. Ca^{++} entry blockers
- c. ? diuretics
- d. management of arrhythmias * amiodarone *not* digoxin
- e. partial surgical resection of the septum

■ Anaesthetic Considerations

- as for MS & AS, Ostheimer believes slow onset epidural anaesthesia, instituted early in labour is not contraindicated, cf. spinal anaesthesia
- he also recommends IABP and CVP monitoring, and observation in ICU for 24 hours postdelivery
- general anaesthesia considerations are,

NB: "*full, slow and tight*"

- a. SBE prophylaxis
- b. avoid - AF, tachycardia
 - falls in venous return or SVR
 - increases in contractility
- c. maintain - slow HR, low contractility
 - high venous return & high SVR

ARRHYTHMIAS

- pregnancy is associated with an increased incidence of *benign arrhythmias* (PAC's / PVC's)
- investigation should focus on contributing factors, coexisting cardiac disease and the patient's haemodynamic status

Guide to Antiarrhythmics in Pregnancy				
Drug	Route	Application	Use in Pregnancy	Comments
Lignocaine	IV	VT, VF digoxin toxicity	safe	• toxic doses and foetal acidosis → accumulation and CVS depression in the neonate
Quinidine	oral	PAT	relatively safe	• high doses may lead to premature labour • rarely neonatal thrombocytopenia
Procainamide	oral, IV	termination & prophylaxis in PAT	relatively safe	• maternal ANF & "lupus" syndrome chronically
Phenytoin	oral, IV	digoxin toxicity resistant VF/VT	not recommended ? OK acutely for digoxin toxicity	• "foetal hydantoin syndrome" • bleeding disorders
Amiodarone	oral, IV	SVT, VT, VF	not recommended	
Verapamil	oral, IV	SVT, chronic AF	probably safe	• IV may cause hypotension & foetal distress
Digoxin	oral, IV	SVT, chronic AF	safe	• monitor plasma levels • adjust dose with quinidine
Propranolol	oral, IV	atrial & ventricular tachyarrhythmias, chronic AF	relatively safe	chronically associated with <ul style="list-style-type: none"> • IUGR, premature labour • neonatal hypoglycaemia • bradycardia • respiratory depression

■ Sinus Arrhythmias

- HR normally increases 10-20% during pregnancy, with acute increases with labour and tocolytic therapy
- in the absence of hypoxia, hypotension, anaemia, or fever, tachycardia requires no therapy
- *bradycardia* is rare in pregnancy in the absence of organic cardiac disease
- other underlying conditions include hypothyroidism, coronary artery disease, cardiomyopathy, or drug effects

Supraventricular Tachycardias

- a. ***premature atrial contractions***
 - frequency increased in pregnancy & more likely to cause symptoms of anxiety etc.
 - usually benign, often related to stress, fatigue, caffeine or alcohol consumption
 - in rheumatic heart disease they may herald the onset of atrial flutter/fibrillation

- b. ***paroxysmal atrial tachycardia*** ± block
 - rapid reentry rhythm, beginning & terminating with a PAC
 - HR ranges from 140-220 bpm
 - increased susceptibility in pregnancy, or increased frequency of paroxysms
 - usually well tolerated haemodynamically, unless underlying CVS disease
 - ***sinus massage*** may help differentiate from,
 - i. sinus tachycardia - no effect or gradual slowing
 - ii. flutter with 2:1 block - increased degree of block
 - massage may be therapeutic in PAT converting the rhythm to sinus
 - R_x edrophonium, metaraminol, digoxin, verapamil, β-blockade
 - PAT + block = ***digoxin toxicity*** → check levels and treat hypokalaemia

- c. ***multifocal atrial tachycardia***

- d. ***atrial flutter***
 - uncommon in pregnant women
 - flutter waves seen at 220-340 bpm, with ventricular response ~ 150 bpm
 - sinus massage will increase the degree of block & make diagnosis easier
 - hyperthyroidism, chronic pulmonary disease & organic heart disease are common
 - therapy should be directed at the underlying condition and slowing the ventricular rate
 - R_x digoxin, verapamil, quinidine, procainamide

- e. ***atrial fibrillation***
 - rare in pregnancy, except in patients with mitral valve disease, cardiomyopathy, IHD, chronic obstructive pulmonary disease, pulmonary embolism, hyperthyroidism
 - ventricular rates ~ 140-200 untreated and 90-110 in chronic cases
 - with chronic cases, left atrial thrombus and systemic embolism is a major risk
 - anticoagulation should be considered in these patients with heparin (not warfarin)
 - R_x digoxin, verapamil, DC cardioversion for acute onset or if unstable

Ventricular Arrhythmia's

- a. ***premature ventricular contractions***
 - isolated, asymptomatic PVC's require no therapy
 - symptomatic PVC's in patients without underlying CVS disease are best managed by removal of precipitating factors, such as alcohol or caffeine
 - echocardiography may be useful to rule-out mitral valve prolapse or asymmetrical septal hypertrophy
 - in the presence of underlying CVS disease, PVC's may herald the onset of LVF
 - serum lignocaine levels with CEA range from 2-4 µg/ml, which is comparable to therapeutic infusions
 - though not recommended as the primary therapy, this may be useful in labour

- b. ***ventricular tachycardia***
 - rare in pregnancy, but does occur more frequently in those with frequent PVC's
 - usually have underlying CVS disease, IHD, mitral valve disease, cardiomyopathy, valvular heart disease, mitral valve prolapse, asymmetrical septal hypertrophy, congenital long QT syndrome
 - R_x = DC cardioversion if unstable, lignocaine for stable, slower patients
bretylum and phenytoin are second line agents
correction of underlying abnormalities (hypo-K⁺/Mg⁺⁺, hypoxia)
quinidine or procainamide are used for recurrent or chronic VT

- c. ***ventricular fibrillation***
 - though rare, this is the most common cause of maternal death
 - R_x = immediate DC cardioversion & advanced life support
failure to respond mandates immediate caesarean section
 - both cardioversion and defibrillation have been used successfully in pregnancy
 - other than transient foetal arrhythmias, foetal outcomes have been good
 - upward & lateral displacement of the mediastinum requires more lateral paddle placement

Conduction Abnormalities

■ Bundle Branch Blocks

- are rare in pregnancy, right being more common than left
- usually secondary to underlying CVS disease,
 - a. cardiomyopathy
 - b. coronary artery disease
 - c. valvular heart disease
- in the absence of underlying disease, no specific therapy is required

■ Wolff-Parkinson White Syndrome

- characterised by a short PR interval, prolonged QRS and a δ -wave
- these patients are more likely to experience arrhythmias during pregnancy
- usually reentrant rhythms, ventricular rates up to 200 bpm, through an aberrant pathway (bundle of Kent most common)

NB: $R_x =$ DCCV for unstable rhythms
procainamide & β -blockers for chronic therapy

- **digoxin** may increase conduction through the aberrant pathway & is contraindicated

■ Atrio-Ventricular Block

1. **1st Degree** = prolonged PR interval (> 0.20 s)
 - may be transient due to vagal tone, secondary to drugs (digoxin, β -blockers), or due to AV nodal disease
 - requires no specific therapy
2. **2nd Degree**
 - i. **Mobitz I** = progressive lengthening PR interval, followed by a dropped beat
 - caused by disease *within* the AV node
 - rarely causes significant bradycardia, or progresses to higher degree block
 - associated with digoxin, increased vagal tone, inferior MI, or myocarditis
 - requires observation only
 - ii. **Mobitz II** = fixed PR interval (long) with regular dropped beats
 - caused by disease *below* the AV node
 - ventricular rates can be quite slow, with dyspnoea, syncope & fatigue
 - frequently progresses to a higher level of block
 - permanent pacemaker insertion is frequently required
3. **3rd Degree** = complete AV dissociation
 - ventricular rates are frequently 40-50 bpm
 - rare in women of childbearing age, usually associated with rheumatic heart disease, inferior MI, acute myocarditis & congenital heart block
 - patients with congenital block frequently have associated VSD's
 - R_x = permanent pacemaker insertion

CONGENITAL HEART DISEASE

Left to Right Shunts

- common causes are ASD, VSD and PDA
- all result in **increased** pulmonary blood flow, for as long as left heart pressures exceed right
- eventually results in progressive pulmonary hypertension, RVH and failure
- pregnancy related increases in blood volume, HR and CO may be tolerated with small shunts
- progression to right to left shunt, **Eisenmenger's complex**, may be exacerbated by the decrease in SVR seen in pregnancy

■ Anaesthetic Considerations

- a. baseline investigations - FBE, MBA₂₀
- ECG, CXR, and echocardiogram
 - b. endocarditis prophylaxis
 - c. IV fluid precautions * paradoxical embolism
 - d. monitoring
 - i. ECG - prone to arrhythmias, especially ASD
 - ii. SpO₂ - all lesions, but esp. those at risk of **shunt reversal**
 - iii. IABP * low threshold, any symptomatic patient
 - iv. PA catheter - evidence of pulmonary hypertension
- symptomatic CCF
- shunt reversal
 - e. maintain preload & afterload - volume preloading where required
- prompt replacement of blood loss
- avoidance of aortocaval compression
 - f. avoid raised PVR * avoid hypoxia, hypercarbia, acidosis
- for labour and delivery, CEA may be employed, however,
 1. cautious volume loading should be employed
 2. loss of resistance should employ **saline**, not air
 3. the sensory level should be raised slowly, and
 4. hypotension should be aggressively treated * α_1 -agonists preferably

Right to Left Shunts

- commonly include tetralogy of Fallot, transposition of the great arteries, tricuspid atresia
- most of these will have been surgically corrected, the most common R-L shunt seen in the childbearing years is a congenital L-R shunt with Eisenmenger's syndrome
- many patients who have had a "functional" repair with no residual symptoms will tolerate pregnancy with minimal increased risk
- for these patients prophylaxis against endocarditis is the principal concern
- uncorrected or "palliated" patients are at high risk throughout pregnancy and have an increased morbidity / mortality
- the decrease in SVR, which may be maximal immediately postpartum, increases shunt flow
- stress and pain with labour may lead to increases in PVR

■ Anaesthetic Considerations

- | | | |
|----|------------------------------|--|
| a. | baseline investigations | - FBE, MBA ₂₀
- ECG, CXR, and echocardiogram |
| b. | endocarditis prophylaxis | |
| c. | IV fluid precautions | * paradoxical embolism |
| d. | monitoring | |
| | i. ECG, SpO ₂ | |
| | ii. IABP & CVP | * almost all cases |
| e. | maintain preload & afterload | - volume preloading where required
- prompt replacement of blood loss
- avoidance of aortocaval compression
* left uterine displacement |
| f. | supplemental O ₂ | - but minimal effect with large shunt (> 30%) |
| g. | avoid raised PVR | * avoid hypoxia, hypercarbia, acidosis |

- there is controversy over both the best method of pain relief during labour and anaesthesia for operative delivery
- if CEA is employed, the same considerations cf. L→R shunts apply, however, any decrease in SVR may be detrimental
- for labour, local / opioid mixtures are preferable due to the lesser sympathetic blockade
- any evidence of hypotension should be treated with α_1 -agonists (metaraminol)
- if general anaesthesia is chosen, then the use of volatile agents should be limited
- usual rapid sequence induction may be poorly tolerated, and a carefully performed regional technique may be safer despite the theoretical problems with afterload

COARCTATION OF THE AORTA

- usually located just distal to the left subclavian artery
- associated conditions include cerebral aneurysms and other cardiac conditions
- most will have been surgically corrected prior to pregnancy, however in uncorrected lesions,
 1. maternal mortality ~ 3-9%
 2. foetal mortality ~ 20%
- cardiac output is rate limited, and although bradycardia is poorly tolerated, tachycardia may also result in LV decompensation
- due to the limited SV, the increased metabolic demands of pregnancy can only be met by an increase in HR
- the progressive decrease in SVR may be poorly tolerated due to reflex tachycardia & CCF
- **aortic rupture** & dissection are possible distal to the stenosis, due to increased turbulent flow
- labour and delivery do not appear to increase the chance of rupture, although alterations of aortic anatomy have been documented in pregnancy
- most reported deaths from rupture have occurred **prior** to labour / delivery
- patients with surgically corrected lesions may undergo labour and delivery without increased risk

■ Anaesthetic Considerations

- a. maintain "normal" HR & SVR
- b. PA catheter and IABP monitoring
 - patients with symptoms of CCF or aneurysmal dilatation of the aorta
 - IABP pre & post-stenosis if severe disease
- c. if a regional technique is chosen, then decreases in afterload and reflex increases in HR should be avoided

MYOCARDIAL INFARCTION

- in an otherwise normal pregnancy this is a rare event ~ 84 reported cases since 1922
- this corresponds to an incidence of ~ 1:10,000
- the coronary anatomy of 30% of these cases was delineated, either angiographically or PM,
 - a. atherosclerosis found in ~ 40% but not severe disease
 - b. **thrombus formation & spasm** are the 1° cause of AMI in pregnancy
 - c. during pregnancy the mortality ~ 30%

■ Factors Affecting Risk

- a. increasing maternal age
 - elevated cholesterol
 - hypertension
 - ? work related stress
- b. increase in women smoking
 - ? decreasing recently
 - accentuates hypercoagulable state
- c. drug abuse
 - cocaine & coronary spasm
- d. cardiorespiratory changes of pregnancy
 - most changes are maximal from 32 weeks on & this corresponds with increased risk
 - majority of MI's occur in the third trimester & death is twice as likely
 - i. increased demand
 - increases in CO, HR, blood volume and MRO_2
 - stress of labour → CO may be 2-3x nonpregnant levels
 - ii. decreased supply
 - increased Q_s and arterial **desaturation**
 - decreased FRC & IRV decreases reserve
 - chronic mild hyperventilation increases 2,3-DPG with **left shift**
 - lowered SVR decreases mean diastolic pressure and LV blood flow
 - thrombus formation 2° to the **hypercoagulable** state of pregnancy
 - coronary spasm 2° to **renin** release from the chorion during ischaemia

■ Medical Management

- a. relieve ongoing ischaemia and limit extension of infarction
 - continuous NIBP/IABP, SpO_2 and foetal heart monitoring
 - maximise oxygenation
 - Ca^{++} blockers, nitrates, β -blockers & opioids have **no** adverse foetal effects
 - Ca^{++} blockers are especially useful due to the **vasospastic** component
 - cardioselective β -blockers are associated with fewer adverse foetal effects

- b. manage complications of AMI *especially arrhythmias, CCF
 - CCF is best managed with afterload reduction
 - reports that *captopril* may be teratogenic & limited use precludes routine use
 - SNP is relatively contraindicated due to potential *thiocyanate* toxicity
 - digoxin is safe during pregnancy & first choice for SVT's
 - lignocaine accumulates in the foetus but no lasting adverse effects demonstrated
- c. **anticoagulation**
 - i. low dose *heparin* for all patients
 - ii. prevention of systemic emboli
 - large anterior infarcts
 - CKMB ≥ 160
 - CPK ≥ 8 times normal
 - presence of AF or ventricular aneurysm
 - iii. * warfarin is better than heparin, but contraindicated in pregnancy
 - teratogenesis, CNS defects, retroplacental & foetal intracranial haemorrhage
- d. **thrombolytic therapy** - the role has not been established
- e. percutaneous angioplasty or surgical revascularisation
 - only if there is ongoing ischaemia and a large segment of myocardium is at risk
- f. **caesarean delivery**
 - any ongoing foetal hypoxia unresponsive to resuscitative measures
 - balance between avoiding the immediate 2 week post-infarct period, due to the risk of arrhythmias & haemodynamic instability, and the increasing stresses of pregnancy on the myocardium, with the risk of rupture, aneurysm etc.

■ Anaesthetic Management

- a. maximise myocardial O₂ supply / demand
- b. avoid
 - tachycardia, hypotension, hypertension
 - pain, anxiety, shivering
- c. monitoring
 - ECG, SpO₂, NIBP/IABP, CVC \pm PA catheter
- d. regional analgesia / anaesthesia
 - decreased LV afterload / preload
 - blocks cardioaccelerator fibres & inhibits sympathetically mediated vasospasm
 - decreases the surgical stress response & avoids the stress of intubation
 - avoids the problems of a high dose opioid GA
- e. high dose opioid general anaesthesia
 - requires prolonged intubation of both mother & infant
 - usually restricted for decompensated patient, or those with mixed valvular lesions in addition to AMI
 - in unstable patients, retaining the ability to manipulate SVR may provide for greater haemodynamic stability
 - control of ventilation may maximise VO₂ and remove pharmacological constraints

ASTHMA

- a. incidence ~ 1:20 persons
~ 1% of pregnant women
~ 10-15% of these will require hospitalisation
~ 50% will have no change in their asthma with pregnancy
~ 25% will improve & 25% worsen
- b. conditions associated with maternal asthma
 - preterm delivery, low birthweight infants & perinatal death occur more frequently
 - haemorrhage, PIH, requirement for induced labour also more common
- c. factors which affect asthma in pregnancy
 - reduction in FRC ~ 20%
 - increased $\dot{V}O_2$
 - increased progesterone - increased RR & MV

Management

- NB:**
1. the aim of therapy is to prevent bronchospastic episodes & the subsequent maternal and foetal hypoxia
 2. although some drugs may have adverse effects upon the foetus, there is generally *less risk* than if exposed to repeated episodes of *hypoxia*

■ *b-Sympathomimetics*

- little data relating to teratogenicity
- high doses used for tocolysis may result in tachycardia, hypotension, & pulmonary oedema
- *albuterol* has been associated with an increased incidence of uterine haemorrhage during spontaneous abortion

■ *Steroids*

- safety is undetermined, some studies showing an association with stillbirth, IUGR and cleft palate, while others show no such association
- the systemic effects of *inhaled steroids* are minimal in nonpregnant patients
- if systemic steroids are required, then use the minimal effective dose, or alternate day therapy
- given IV they take several hours for effect, therefore if required in an acute attack they should be administered early
- the mechanism of action is believed to be *direct bronchodilatation*, in addition to inhibition of synthesis of chemical mediators of inflammation

■ Theophylline

- narrow therapeutic range, ∴ need to monitor & difficulty maintaining therapeutic plasma levels
- clearance is unchanged, or **reduced** in pregnancy
- no apparent teratogenic side effects, however is a potent tocolytic and may **prolong labour**
- crosses the placenta easily and may result in,
 - a. decreased foetal HR variability
 - b. transient tachycardia in the newborn ~ 10% even with normal plasma levels
- the elimination half-life is prolonged in neonates

■ Cromolyn Sodium

- not a bronchodilator & not efficacious in all patients
- difficult to predict those patients who will benefit from use, however, no adverse effects have been observed during pregnancy

■ Antihistamines

- in premature infants, there is a strong association with **retrolental fibroplasia** & maternal antihistamine use in the last 2 weeks of pregnancy

Labour and Delivery

- CEA is the preferred technique for the severe patient
- benefits are decreased stress, anxiety and hyperventilation which may all precipitate asthma
- anti-asthmatic therapy should continue throughout labour with the understanding that,
 1. it will prolong labour & decrease uterine contractility
 2. predispose to PPH
- for LUSCS, the stimulus of intubation and IPPV, together with the associated risks of barotrauma, are avoided
- if GA is required, some would use **ketamine** instead of thiopentone in the severe patient
- when used in conjunction with aminophylline it **does not** increase the incidence of **arrhythmias** but does reduce the **seizure threshold**
- emergence reactions may be severe but are usually adequately managed with small doses of benzodiazepines
- dTC, metocurine and atracurium should probably be avoided due to **histamine release**
- the potent inhaled agents are equally effective as bronchodilators

ENDOCRINE DISEASE

Thyroid Disease

NB: this is one of the most common endocrine disorders of pregnancy

■ Nontoxic Goitre

- a. may increase in size due to relative **iodine deficiency**
 - increased GFR and renal excretion
- b. clinical manifestations
 - dyspnoea, altered phonation
 - dysphagia
- c. anaesthesia
 - * potential for intubation difficulty
 - regional anaesthesia by choice

■ Hyperthyroidism

- a. aetiology
 - Graves' disease, or **diffuse toxic goitre** most common
 - toxic nodular goitre, toxic multinodular goitre
 - hydatidiform moles, choriocarcinoma
- b. manifestations
 - * usual signs frequently seen in euthyroid patients
 - tachycardia, systolic ejection murmur
 - heat intolerance, increased skin temperature
 - diarrhoea, nervousness, weight loss (obscured by pregnancy)
 - eye changes; exophthalmos, lid lag / retraction
 - hyperemesis gravidarum may be the 1st sign
- c. investigations
 - difficult as oestrogen increases **thyroxine binding globulin**
 - plasma thyroxine elevated & T₃-uptake in hypothyroid range

■ Anaesthesia

- elective surgery should be postponed until rendered **euthyroid**
- if hyperthyroid, avoid sympathomimetic agents & ensure an adequate depth of anaesthesia prior to surgical stimulation
- plasma catecholamines are **not** increased and the circulatory response is not due to increased sensitivity, however, they will exacerbate changes
- adequate premedication, ie. a benzodiazepine, is desirable
- anticholinergic agents should be avoided due to tachycardia & inhibition of heat loss
- the thiobarbiturates have antithyroid properties & are OK for induction, though, the antithyroid effect has not been demonstrated clinically
- ketamine and pancuronium are generally contraindicated
- CEA has the advantage of blocking adrenal and cardiac sympathetic innervation
- a potential problem is hypotension requiring pharmacological support

- superficial and deep cervical plexus blockade, combined with local infiltration is useful for thyroid surgery in the pregnant patient
- however, the addition of adrenaline may result in systemic effects
- β -blockers are generally useful for controlling the manifestations during surgery
- the increased MRO_2 above the normal pregnant state further complicates the reduction in FRC & tendency to arterial desaturation

■ Thyroid Storm

- a. manifestations
 - hyperpyrexia, tachycardia, AF, CVS instability \pm collapse
 - severe dehydration, anxiety, altered consciousness
 - * may mimic MH
- b. anaesthesia
 - same cf. hyperthyroidism

■ Hypothyroidism

- a. aetiology
 - rare in term pregnancy
 - associated with an increase in *spontaneous abortion*
 - usually iatrogenic, surgery or radioactive iodine therapy
- b. manifestations
 - fatigue, cold intolerance, cool dry skin, coarse hair
 - hoarseness, constipation
 - delayed DTR's, decreased mentation
 - oedema, cardiomegaly, CCF, pleural \pm pericardial effusions
 - mild anaemia, hypercholesterolaemia, accelerated atherosclerosis
 - low voltages & sinus bradycardia on ECG

■ Anaesthesia

- more sensitive to opioids, sedatives and anaesthetic agents
- hypoxic ventilatory drive is diminished
- hypercapnic ventilatory drive is decreased in myxoedema, though not in hypothyroidism
- these factors may combine to predispose to *respiratory failure*
- metabolism of drugs, especially opioids is delayed
- the bradycardia, decreased contractility & CO delay induction with IV agents but speed induction with volatiles
- typically have reduced intravascular volume, therefore are at greater risk of hypotension from blood loss or sympathectomy
- thus prehydration is advised & ephedrine is useful for treating hypotension
- NMJ blockade may be prolonged with standard doses
- they are prone to develop *hypothermia*
- impaired free water clearance may result in hyponatraemia
- hypoglycaemia may develop 2^o to thyroid hormone replacement

Diabetes

a. this is actually the **most common** medical problem encountered in pregnancy

Def'n: pathophysiology

- placental insufficiency → major problem
 - ~ **35-45%** decrease blood flow
 - decrease worse with poor control (high HbA_{1c})
- HbA_{1c}
 - poor carrier of oxygen
 - maternal P_{aO2} inversely related to levels
- ketoacidosis
 - now a rare entity with good perinatal control
 - remains a significant cause of neonatal mortality
 - uncontrolled infection, steroids & β-mimetics for prematurity
- susceptibility to infection
- accelerated atherosclerosis
- autonomic neuropathy
- associated conditions
 - pregnancy induced hypertension
 - premature labour
 - abruptio placentae
 - macrosomia
 - major foetal congenital malformations
 - rebound neonatal hypoglycaemia

Classification*	
Class A	<ul style="list-style-type: none"> • abnormal CHO tolerance nonpregnant • no insulin requirement before / during pregnancy
Class B	<ul style="list-style-type: none"> • duration of diabetes < 10 years
Class C	<ul style="list-style-type: none"> • duration of diabetes 10-20 years
Class D	<ul style="list-style-type: none"> • duration of diabetes > 20 years
Class F	<ul style="list-style-type: none"> • associated with diabetic nephropathy
Class R	<ul style="list-style-type: none"> • associated with retinitis proliferans
Class T	<ul style="list-style-type: none"> • associated with renal transplant
Class H	<ul style="list-style-type: none"> • requiring insulin and associated with coronary artery disease
* modified from White	

■ Anaesthetic Management

Def'n: labour & delivery

- a. advantages of CEA,
 - decrease plasma *catecholamine* levels, which may further decrease placental perfusion
 - reduces maternal *lactate* production, and therefore foetal acidosis
 - provides ready provision for *instrumental delivery* which is required more frequently
- b. *hypotension* in these patients should be treated aggressively
- c. a second IV line containing a non-dextrose solution should be available for rapid administration

Def'n: caesarean delivery

- a. Datta & Brown (1977) found a higher incidence of foetal acidosis in infants of diabetic mothers
- b. in a subsequent study by Datta *et al.* using CEA, *foetal acidosis* was found to relate to both,
 - the severity of the *diabetes*
 - the duration and severity of *hypotension*
- c. the generation of foetal acidosis is multifactorial,
 - increased lactate production from the hypoxic placenta
 - increased placental glycogen, increasing lactate production
 - hyperglycaemia, in the presence of hypoxia further increases lactate production
 - foetal hyperglycaemia may be associated with an increased O₂ utilisation
- d. in 1982, Datta *et al.* repeated their study, using *spinal anaesthesia*, plus,
 - tightly controlled maternal BSL levels * 80-120 mg/dl
 - non-dextrose containing volume expansion - Hartmann's solution
 - aggressive management of hypotension - MAP > 100 mmHg
- e. neonatal pH's *did not* differ significantly from non-diabetic groups
- f. either spinal or epidural anaesthesia are suitable for diabetic patients, providing the above guidelines are followed
- g. as for other disease states, CEA may be preferable due to the slower onset of hypotension
- h. for *general anaesthesia*, there are a number of important factors,
 - increased *gastric stasis*
 - "stiff-joint syndrome" in juvenile onset diabetics
 - autonomic neuropathy
 - decreased insulin requirement immediately postoperatively
 - i. determination of BSL's in recovery

Adrenal Disorders

■ Adrenocortical Insufficiency

- or **Addison's disease**, is the most common adrenal abnormality in pregnancy
- may result from,
 1. 1° destruction of the adrenal
 - autoimmune
 - tumour
 - haemorrhagic necrosis
 - infection (TB)
 2. 1° hypofunction of the pituitary
 - tumour, infection (TB), sarcoid
 - haemorrhagic necrosis
 3. 2° suppression of the pituitary
 - exogenous steroid administration
 - steroid secreting tumours
- presentation may be indolent, or acute life threatening collapse
- clinical features include,
 - i. weakness, fatigue, vomiting, diarrhoea, abdominal pain
 - ii. excess pigmentation
 - iii. hypotension ± hypovolaemia
 - iv. biochemical abnormalities
 - mild **hyponatraemia**, hypoosmolality
 - **hyperkalaemia** (Na^+/K^+ ratio < 25:1)
 - **hypoglycaemia**
 - mildly elevated urea
 - mild anion gap **acidosis**
- may be better tolerated in pregnancy due to foetal → maternal transfer of **glucocorticoids**
- the post-partum diuresis & dehydration may precipitate an **adrenal crisis**
- anaesthetic considerations include,
 1. correction of hypovolaemia
 2. steroid supplementation
 3. correction of biochemical abnormalities
 - i. glucose supplementation
 - ii. Na^+/K^+ balance
 4. increased susceptibility to
 - drug induced myocardial depression
 - muscle paralysis
 5. decreased responsiveness to catecholamines
 6. monitoring
 - + IABP and CVP

■ Phaeochromocytoma

- rare in pregnancy, but high maternal / foetal mortality
- symptoms include,
 - i. anxiety, palpitations, tachyarrhythmias
 - ii. headache, diaphoresis, blurred vision
 - iii. heat intolerance
 - iv. excessive weight loss
 - v. paroxysmal or sustained hypertension, usually **not** associated with proteinuria
 - vi. episodic attacks triggered by
 - uterine contractions
 - foetal movements
 - changes in posture

- prolonged α -adrenergic stimulation results in,
 - i. decreased plasma volume with elevation of the haematocrit
 - ii. reflex hypotension / tachycardia

- the safety of adrenergic blocking drugs has not been established, however foetal survival is undoubtedly improved with their use
- well controlled patients may be delivered vaginally, or by LUSCS at term followed by excision of the tumour
- the α -blocking agents **phentolamine** and **phenoxybenzamine** are combined with **b-blockade** to prevent the reflex tachycardia
- β -blockade may result in decreased foetal HR and increased uterine contractility
- avoidance of factors which increase catecholamine release,
 1. hypoxia, hypercarbia and acidosis
 2. hypotension *poorly controlled epidural anaesthesia

- Cousins & Rubin reported delivery under light GA + epidural with the claimed advantages of,
 1. improved CVS stability by deafferentation of the operative site
 2. sympathetic blockade
 3. systemic effects of local anaesthetics (lignocaine)

- in contrast to deep general anaesthesia, myocardial contractility is better preserved and the coronary and peripheral circulations are better able to respond to catecholamines following tumour removal

- drugs known to have a pressor or tachycardic response should be avoided,
 - i. droperidol
 - ii. anticholinergics
 - iii. succinylcholine
 - iv. histamine releasing agents - dTC, atracurium, ? morphine
 - v. pancuronium
 - vi. halothane *ventricular arrhythmias
- SNP (? reflex tachycardia), GTN, prazosin, MgSO₄ and phentolamine have all been advocated for intraoperative control of hypertension
- patients with pre-existing cardiomyopathy may require infusion of catecholamines following tumour removal, pending up-regulation of receptors and decay of sympathetic blockade
- in addition to standard a GA, minimum monitoring should include,
 - i. CUD
 - ii. CVP
 - iii. IABP

■ Cushing's Syndrome

- ovulation and pregnancy are rare with this condition
- increased incidence of spontaneous abortion, stillbirths, and premature labour
- effects 2° to excess circulating glucocorticoids, common caused by,
 - i. **iatrogenic steroid administration** = most common
 - ii. pituitary adenoma ~ 80% (of remainder)
 - iii. ectopic ACTH ~ 15%
 - iv. adrenal adenomas, hyperplasia
- classical features may closely resemble normal pregnancy,
 - i. weight gain, truncal obesity, plethoric face, hirsutism, bruising, striae
 - ii. weakness, osteoporosis, poor wound healing
 - iii. hypertension
 - iv. psychosis
 - v. hypernatraemia, hypokalaemia and hyperglycaemia
- objectives of management,
 1. control of hypertension
 2. normalisation of volume status - spironolactone
 3. correction of biochemical abnormalities - hyperglycaemia
- hypernatraemia/hypokalaemia
 4. perioperative steroid supplementation
 5. potential for airway difficulty

Hyperparathyroidism

- **parathyroid adenoma** is the commonest cause in pregnancy
 - normal pregnancy is associated with increased PTH secretion and Vit.D₃ activity
 - however, serum Ca⁺⁺ levels are normally **decreased**,
 1. increased foetal demands
 2. increased RBF/GFR and renal excretion
 3. hypoalbuminaemia of pregnancy → ↓ total Ca⁺⁺ > ↓ ionised Ca⁺⁺
- NB:** serum Ca⁺⁺ levels may be **normal** in the hyperparathyroid patient
- labour and delivery usually proceed uneventfully
 - there is an increased incidence of,
 1. stillbirths, spontaneous abortion, premature labour
 2. neonatal tetany

■ Clinical Manifestations

1. anorexia, hyperemesis
2. generalised weakness, malaise, lethargy
3. polyuria, polydipsia (nephrogenic DI)
4. hypertension
5. constipation

■ Complications

1. renal calculi
2. pancreatitis
3. psychiatric disorders
4. hypercalcaemic crisis
 - mental deterioration ± coma
 - arrhythmias, CCF
 - renal failure

■ Anaesthetic Considerations

1. maintenance or normovolaemia and renal output
2. ECG monitoring
 - increased Ca⁺⁺ & **decreased QT** interval
 - arrhythmias
3. osteoporosis and risk of pathological fractures
4. unpredictable response to NMJ blocking drugs, ∴ **monitor**

Hypoparathyroidism

- rare in pregnancy, usually resulting from unintentional parathyroidectomy at thyroid surgery
- borderline cases may present due to increased demands of the foetus

■ Clinical Manifestations

1. fatigue, lethargy
2. paraesthesias, numbness
3. muscle weakness, tetany
4. carpopedal spasms, laryngeal stridor, convulsions
5. altered mental status
6. dry, rough skin, patchy hair loss, cataracts
7. **long QT** and decreased myocardial contractility

■ Anaesthetic Considerations

1. plasma ionised Ca^{++} can be rapidly reduced by **respiratory alkalosis**
2. CEA may prevent the hyperventilation associated with labour
→ decreased likelihood of **tetany**

Pituitary Disorders

- postpartum pituitary necrosis, **Sheehan's syndrome**, following shock or haemorrhage, is the commonest cause of anterior pituitary insufficiency
- the clinical picture relates to the degree of damage, and relates to the deficiency of hormones secreted by the ovaries, adrenal gland, and thyroid gland
- the presentation may be insidious, with the first sign being failure of **lactation** with breast involution
- more dramatic presentation may follow hypoadrenalism & hypothyroidism

HEPATIC DISEASE IN PREGNANCY

NB: the most common cause of hepatic dysfunction in pregnancy is *viral hepatitis*

Cholestasis of Pregnancy

- *intrahepatic cholestasis*, with deposition of bile acids in the skin & pruritis
- ? increased sensitivity to bile acids 2° to *oestrogen* production
- increased risk of *prematurity* (~ 50%) and foetal death
- foetal distress may occur in up to 30%, with a caesarean rate of 30-60%
- clinical manifestations include,
 - a. *pruritis*
 - classical presenting symptom
 - beginning in the third trimester
 - involving the palms, soles of the feet and the trunk
 - b. dark urine, mild jaundice, light stools
 - c. *prothrombin time* is usually normal
 - may be increased with vitamin K malabsorption
 - this occurs 2° to *cholestyramine* used to alleviate the jaundice
 - a coagulation profile should be ordered prior to regional anaesthesia
 - d. increased risk of *postpartum haemorrhage*

Acute Fatty Liver of Pregnancy

- aetiology is unknown, but there may be some link to *tetracyclines*
- maternal & foetal morbidity / mortality in the untreated patient ~ 80-90%
- immediate delivery has reduced *maternal mortality ~ 10-33%*
- *foetal mortality* remains high due to a high incidence of stillbirths
- untreated the disease may progress to,
 - a. fulminant hepatic failure & encephalopathy
 - b. DIC with uncontrolled GIT bleeding
 - c. death
- the incidence is higher in *young primiparas* giving birth to twins or male infants
- typically presents between the 36-40th weeks of gestation, clinical symptoms including,
 - a. headache, fatigue, malaise
 - b. severe persistent vomiting
 - c. diffuse abdominal pain, or right upper quadrant pain
 - d. jaundice & fever occur in ~ 50% of patients
 - e. mild hypertension & peripheral oedema suggest PIH
 - f. there is usually evidence of DIC
 - g. plasma electrolyte & glucose abnormalities
 - h. encephalopathy & coma occur late

■ Hepatic Involvement in Other Conditions

1. PIH, preeclampsia, eclampsia ~ 50% abnormal LFT's
2. HELLP syndrome

RENAL DISEASE

- normal physiological changes in pregnancy,
 - a. RBF & GFR increase ~ 50% above baseline
 - b. BUN & creatinine **decrease**, reflecting the changes in GFR
 - c. tubular reabsorption increases in proportion to the filtered load to maintain water & sodium balance
 - however, it is not unusual to see **glycosuria**, amino-aciduria & **proteinuria**
 - serum **uric acid** levels decrease significantly in normal pregnancy & are a sensitive marker of **tubular function**
 - d. dilatation of the collecting system extends to the pelvic brim
 - this represents a functional dead space of ~ 200 ml
- there is minimal data relating renal function to maternal / foetal outcome
- in the absence of associated factors, such as hypertension or proteinuria,
 - a. creatinine → - lower in pregnancy, laboratory dependent
 - i. female ~ 45-95 $\mu\text{mol/l}$
 - ii. pregnancy ~ 30-80 $\mu\text{mol/l}$
 - b. creatinine < 140 $\mu\text{mol/l}$ - no adverse effect
 - c. creatinine > 200 $\mu\text{mol/l}$ - decreases the likelihood of conception
~ 20% deliver prior to 36 weeks
- increased stillbirths, IUGR, neonatal deaths

- NB:**
1. levels > 200 $\mu\text{mol/l}$ may be associated with a decrease in maternal renal function which does **not reverse** after the pregnancy
 2. the presence of **hypertension** may be the most important determinant of maternal & foetal outcome
 3. the majority of renal disease occurs in females after their childbearing years, however, there are a number of conditions which may affect young females

■ Glomerular Disease

- may result from infection, inflammation, or systemic diseases such as SLE or diabetes
- frequently accompanied by **hypertension & proteinuria**, with an associated incidence of **preeclampsia ~ 50%**
- **nehrotic syndrome** commonly results from glomerular disease, however, the commonest cause of this de novo in pregnancy is preeclampsia
- **hypertension** is the most common medical complication of pregnancy
- concurrent hypertension may result in further deterioration of renal function
- diastolic pressures > 85 mmHg require differentiation, though, this may be difficult
- long term management aims for gradual pressure reduction and both hydralazine and methyldopa have been used with safety
- β -blockers have also been used but cause IUGR and neonatal bradycardia

■ Acute Renal Failure

- occurs with an incidence of ~ 1:10,000
- usually is related to late complications of pregnancy,
 - a. causes of maternal haemorrhage
 - placental abruption
 - placenta praevia
 - other causes
 - b. preeclampsia / eclampsia
 - c. postpartum HUS
 - d. amniotic fluid embolism
 - e. progression of pre-existing renal disease
- factors requiring consideration during anaesthesia include,
 1. pericardial effusion, CCF
 2. pulmonary infiltrates / oedema
 3. CNS depression with uraemia
 4. platelet dysfunction, anaemia

■ Renal Transplant

- factors relevant to management include,
 1. natural history of the primary renal disease
 2. current renal function ~ 25% suffer deterioration in pregnancy
 3. time of conception relative to the time of surgery
 4. immunosuppressive drugs used for rejection control
 - predisposition to infection in the mother
 - premature rupture of the membranes
 - foetal malformation, IUGR
 - adrenal insufficiency
 - neonatal lymphopaenia within the first few weeks
- maternal and foetal outcome is good in ~ 70% who are otherwise healthy
- the risk of **prematurity** may be as high as 45%

■ Anaesthetic Considerations

- CEA remains the method of choice for most patients, providing no coagulopathy is present
- generally results in **increased** uterine and renal perfusion, providing hypotension is avoided
- relatively contraindicated with long-term β -blockers, due to risk of hypotension/bradycardia
- general anaesthesia should be avoided unless regional contraindicated, due to the reduction of 30-50% in RBF/GFR with the volatile agents

HAEMATOLOGICAL DISEASES

Haemoglobinopathies

■ Thalassemia

- mixed disorders resulting in the diminution of production of the protein chains of HbA₁
 - a. α -thalassaemia - thalassaemia minor
 - b. β -thalassaemia - autosomal codominant
 - i. heterozygous - β -thalassaemia minor
- half normal haemoglobin
 - ii. homozygous - β -thalassaemia major (Cooley's anaemia)
- most patients with α -thalassaemias and β -thalassaemia minor require *no treatment*
- patients with **β -thalassaemia major** have,
 1. little or no production of β -globin
 2. **anaemia** - average Hct < 20%
 3. multiple transfusions with the potential for **iron overload**,
 - i. cardiomyopathy
 - ii. CCF & arrhythmias
 - iii. pancreatic dysfunction - bronze diabetes
 - iv. hepatic dysfunction - cirrhosis
 - v. hepatomegaly & splenomegaly
 4. skeletal malformations in association with **marrow hyperplasia**
 - spinal anomalies make regional anaesthesia difficult

■ Sickle Cell Disease

- most common in Negroes and people of Mediterranean descent
- heterozygotes (sickle cell trait) are usually asymptomatic
- single gene mutation encoding for β -globin, substituting **valine** for **glutamate** at position 6



1. polymerisation of Hb under conditions of low P_{O₂} or acidosis
2. RBC distortion, decreased plasticity and decreased survival time
→ **hyperbilirubinaemia & anaemia**
3. **vaso-occlusive** phenomena - splenic infarction, sepsis
- pulmonary infarction, infiltrates, chest pain
- CVA's
- avascular bone necrosis

Disorders of Coagulation

- normal pregnancy is associated with a ***hypercoagulable state***,
 1. increased factors I, VII, VIII, IX, X
 2. expansion of blood volume
 3. platelet count remains normal but increased TXA₂ with increased aggregation
 4. reduction in proteins S & C, and plasminogen activators

- **Iatrogenic Coagulopathy**
 - usually patients with prior veno-occlusive disease or with prosthetic heart valves
 - these patients are usually on warfarin but are changed to heparin during pregnancy due to the teratogenic effects of the former
 - some would continue warfarin as it is more effective prophylaxis in the presence of prosthetic heart valves, converting to heparin only for labour and delivery

- **Idiopathic Thrombocytopenic Purpura**
 1. autoimmune disorder due to ***antiplatelet antibodies***
 - commonest autoimmune haematological disorder in pregnancy
 2. accelerated platelet destruction
 - thrombocytopenia
 - splenomegaly
 3. transplacental Ab passage
 - ~ 50% have platelet counts < 100,000
 - risk of ICH during vaginal delivery
 - * no good evidence LUSCS is safer
 - usual management includes ***steroids*** and ***splenectomy***
 - high dose IgG has been used for resistant cases and acute deteriorations at delivery
 - regional anaesthesia may be used providing assessment of bleeding time is normal

- **Von Willebrand Disease**
 - autosomal dominant, variable penetrance mode of inheritance, characterised by,
 1. reduced factor VIII activity
 2. impaired aggregation of platelets & prolonged bleeding time

 - most patients increase factor VIII levels with pregnancy and deliver normally
 - if factor VIII levels/activity remain below 25% then consideration for cryoprecipitate or FFP at the time of delivery should be given

NEUROLOGICAL DISORDERS

■ Chronic Back Pain

- ligamentous strain from the lumbar lordosis of pregnancy results in back pain in ~ 50%, frequently with radicular symptoms in the legs
- there is **no evidence** that regional anaesthesia will exacerbate these problems
- those having had previous back surgery may be technically more difficult and there is a greater likelihood of,
 - i. a "patchy" block
 - ii. accidental dural puncture
- for LUSCS in these people spinal may be a better choice

■ Multiple Sclerosis

- relatively common disease in young people, **incidence ~ 1:2000**
- results in **demyelination** within the CNS, it **does not** involve the peripheral nerves
- characterised by unpredictable **relapsing course** and the incidence of exacerbation in the first 3 postpartum months is ~ **3x** the nonpregnant population
- several studies have implicated anaesthesia as causing exacerbation in the postoperative period in the nonpregnant patient
- however, the numbers in these studies have been small and the relationship to anaesthesia unclear
- other conditions in the perioperative period are known precipitants, eg. **pyrexia**, surgery itself
- later studies in both obstetric and nonobstetric patients have shown no significant increase with spinal or epidural anaesthesia

NB: there is **no evidence** that women who receive epidural or spinal anaesthesia have a higher relapse rate, however, the patient should be warned there is a higher relapse rate in the peripartum period, **irrespective** of the use of anaesthesia

■ Epilepsy

- increased risk of complications,
 - i. prematurity
 - ii. preeclampsia
 - iii. obstetric haemorrhage
 - iv. uterine hypotonia
- these may be 2° to medications or to the seizures themselves
- LEA & regional techniques are safe
- if GA is required, then avoid ketamine & enflurane
- phenytoin & phenobarbitone interfere with vit.K metabolism & require coagulation studies

■ Myasthenia Gravis

- a. history
 - course of the disease
 - previous surgery, thymectomy, plasmapheresis
 - daily muscle strength & functioning
 - bulbar involvement
 - presence of CAL
- b. medications
 - anticholinesterase dosage
 - immunosuppressives
- c. lung function tests
 - spirometry
 - arterial gas analysis if indicated
- d. optimisation of condition prelabour / anaesthesia
- e. regular assessment during labour for changing anticholinesterase requirement
 - peak flows
 - sequential vital capacity estimations
- f. LEA *preferred technique where possible
 - slowly raised sequential block is preferable
 - avoidance of amino-ester agents due to prolongation of action
 - establish LEA early in the course of labour
- g. GA
 - avoid CNS depressant medication
 - increased risk of aspiration
 - unpredictable response to neuromuscular blocking agents, usually,
 - i. increased sensitivity to nondepolarising agents
 - ii. resistance to suxamethonium - ED₉₅ up to 2.5x normal
 - duration of action of SCh is prolonged
 - requirement for elective postoperative ventilation

■ Spinal Cord Injury

- patients with lesions below T₁₀ will experience pain during labour
- those with lesions above T₆ are prone to autonomic hyperreflexia
- regional anaesthesia may be difficult technically, but is the method of choice
- usually only low concentrations of LA are required
- however, hypertension may be persistent, requiring treatment with vasodilators or emergent delivery of the baby under GA

■ Migraine Headache

- 70% of migraine sufferers are women in the childbearing years
- no studies indicating an adverse effect of LEA
- administration of ephedrine to those taking ergot preparations may produce hypertension

AUTOIMMUNE DISEASE

- mechanisms of immune host damage include,
 1. circulating antigen-antibody activation of C', T-cells and macrophages (type II)
 2. Ag-Ab immune complex deposition, with subsequent tissue damage (type III)
 3. cell mediated, sensitised T-cell destruction of tissue (type IV)
- immunosuppression at the level of the foetoplacental unit allows ongoing pregnancy
- **progesterone** inhibits T-lymphocyte function
- several protein species have been isolated and appear to be associated with exacerbation/remission of a number of autoimmune diseases,
 1. pregnancy-associated globulin 2-PAG
 - also called pregnancy associated plasma protein A (PAPP-A)
 - immunosuppressive glycoprotein affecting C' and lymphocyte transformation
 2. pregnancy zone protein PZP
 3. uromodulin
 - immunosuppressive glycoprotein isolated from urine which inhibits T-cell and macrophage function

Rheumatoid Arthritis

- chronic, systemic, non-organ-specific autoimmune disease of unknown aetiology
- ? infectious synovitis that induces antigenic change which stimulates autoimmune systemic response
- Ab's are formed against a myriad of gamma globulins and EBV related antigens
- frequency in young women ~ 3x young males
- rarely occurs during, and is generally suppressed by pregnancy, possibly due to increased concentrations of circulating **cortisol**
- may relapse severely in the post-partum period (~ 2-4/12)
- lactation appears to prolong remission

■ Anaesthetic Considerations

1. airway problems
 - i. mandibular hypoplasia
 - ii. TMJ synovitis / arthritis
 - may have growth failure in juvenile RA with micrognathia
 - iii. cervical spine instability, fusion, subluxation
 - iv. cricoarytenoid arthritis
 - v. laryngeal rotation

2. cardiovascular
 - i. pericardial effusion, pericarditis
 - ii. rheumatoid nodules - valvular
- epicardial or myocardial
 - iii. coronary arteritis & focal interstitial myocarditis (rarely)
3. respiratory
 - i. CAL
 - ii. pleuritis
 - iii. interstitial fibrosis
 - iv. nodular lung disease - Caplan's disease
 - v. pneumonitis
 - vi. pulmonary arteritis - PAH rare
 - vii. intrapulmonary rheumatoid nodules (spontaneous rupture)

Systemic Lupus Erythematosus

• Ab formation against,

1. intranuclear
 - i. single and double stranded DNA
 - ii. Sm1 ribonucleoprotein
2. lymphocytes, erythrocytes
3. neurons
4. gamma globulins

• *anticardiolipin antibody* is one of many phospholipid AB's present in SLE (20-65%)

■ Clinical Features

- a. incidence ~ 4-250 / 100,000
~ 8-10:1 F:M ratio
- b. peak onset 2nd-4th decades
- c. ~ 20% of cases are diagnosed at the onset of pregnancy during ***routine screening***
- d. severity & frequency of exacerbation increases in ~ 50% of pregnancies
- e. usual presentation in pregnancy
 - i. fever, general malaise
 - ii. symmetric arthritis
 - iii. myalgias and muscle weakness
- f. occasional "preeclampsia-like" syndrome, with hypertension, proteinuria & oedema

■ Anaesthetic Considerations

1. CVS
 - i. pericardial effusions ~ 25%
 - ii. acute or constrictive pericarditis
 - iii. tachyarrhythmias
 - iv. Libman-Sacks endocarditis & valvular malfunction (check Echo preop.)
 - v. rarely AMI
2. respiratory ~ 75%
 - i. interstitial pneumonitis
 - ii. fibrinous pleuritis ± bilateral pleural effusions
 - iii. acute pulmonary vasculitis, or advanced arteriosclerosis
 - iv. focal alveolar haemorrhages
 - v. bronchopneumonias
 - vi. massive pulmonary haemorrhage - rarely
3. renal
 - i. lupus nephritis ~ 50%
 - ii. nephrotic syndrome
 - iii. chronic renal failure
4. other
 - i. **coagulopathy** - raised APTT due to circulating ***lupus anticoagulant***
- either a phospholipid or an anticoagulant Ab
 - ii. anticardiolipin - thrombocytopaenia (but increased adhesiveness)
- arterial thromboses
(CVA, gangrene, MI, avascular necrosis of bone)
- venous thromboses & intravascular clot formation
 - iii. Raynaud's phenomenon ~ 15%

■ Foetal & Neonatal Considerations

1. increased incidence of premature labour, miscarriage & stillbirth
 - especially with antiphospholipid Ab's
 - survival may be improved by antiplatelet or anticoagulant drugs
2. neonatal lupus syndrome
 - i. cytopaenias
 - ii. discoid rash
 - iii. cardiac conduction abnormalities

Idiopathic Thrombocytopenic Purpura

- common autoimmune disorder of pregnancy, F:M ratio ~ 3:1
- often asymptomatic, or may present with bleeding diathesis
- **CNS haemorrhage** is the most serious consequence
- anti-platelet Ab's passively cross the placenta and result in varying degrees of **neonatal thrombocytopenia**
- mothers can be treated with steroids and gamma-globulin, however their response **does not** correlate with that of the foetus
- controversy exists as to whether LUSCS is safer than NVD for the baby, due to the risks of IVH with vaginal delivery