Maternal Physiology

Changes by System

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Plasma Proteins
Gastro-Intestinal System
Renal System
Neuro
Metabolic/Endocrine
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Aorto-Caval Syndrome

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Maternal Physiology

- pregnancy & ageing = most common altered physiological state which humans subjected to
- significant changes esp in:
  - CVS
  - resp
  - renal
  - endocrine
  - CNS
- pregnant woman also carries fetus with own physiological changes, growths & adaptations

Changes by System

Respiratory
(3rd trimester ⇒ term)
- volumes + capacities:
  - incr in:
    - VT - 40%
    - IC
    - IRV
  - decr in:
    - ERV + RV ⇒ ↓FRC ~25% ⇒ a shunt tendency
      ▼ cause by upwards displacement of diaphragm
  - No change:
    - TLC
    - VC
    - CV
- mechanics:
  - compliance:
    - Compliance lung = norm
    - compliance chest wall = ↓↓significantly
      ▼ overall C_T = slight ↓
  - Diffusing capacity = unchanged
  - Airway resistance:
    - ~unchanged or slight ↑ due to upper airway capillary engorgement
- ventilation:
  - ↑RR ~10%
  - ↑MV ~40% - progesterone influenced
    ▼ end of 3rd trimester to labour - ↓Vt due to mechanical problems. ↑RR to compensate
  - Dead space:
    - physiological = norm
    - alveolar dead space ↓ed (2nd to ↑CO)
  - V_A (alveolar ventilation) ↑70%
  - VO2 ↑20%
- gases:
  - PaO2:
    - 1st trim = ↑7-10mmHg
    - 3rd trim = ↑+/− 3mmHg
  - PaCO2 = ↓ to 30mmHg

By Adam Hollingworth
with low PaCO₂ would expect to see PaO₂ >100mmHg but limited due to ↑ed V/Q mismatch (15% compared to 3% in non pregnant)

- HCO₃ = ↓ to 20mmol/L
- pH 7.44 +/- full compensation
- P₅₀ = ↑s through pregnancy (from norm = 27)
  - 1st trim 27.8
  - 2nd trim = 28.8
  - term = 30mmHg
- due to ↑2,3 DPG ⇒ R shift

DO₂ = ↑10%

**Cardiovascular SYstem**

- blood:
  - ↑rbc 20%
  - plasma volume ↑45%
- leads to:
  - ↑TBV 48% ⇒ ↓HCT ⇒ enhances flow to uterus, kidney, breast, skin & compensates for loss with delivery
  - similar mechanisms with normovolaemic haemodilution as means to minimise rbc loss
    - dilutional ↓Hb ~120
- heart:
  - ↑SV 30%
  - ↑HR 15%
- ↑CO 30-40% fully developed at end of 2nd trim and continues until birth
- S3 heart sound common
  - systolic murmurs at left sternal edge common
  - diastolic murmurs are not common

(heart Sounds:
- S1 = beginning of systole. mitral/tricuspid forced shut
- S2 (A2, P2) = end of systole = closure of aortic/pulmon valves
- S3 = soon after S2. rapid vent filling after opening of A/V valves. can be norm in preg, athletes, young)
- S4 = before S1. atrial kick of blood into stiff ventricle. pathological)

- vasculature:
  - bp changes at max 2nd trim, then slowly return to norm levels at term
  - ↓SVR 15% ⇒ ↓MAP ~10mmHg
    - due to low resistance uterine circulation + prostaglandin effects on vasc tone
  - SBP ↓10mmHg
  - DBP ↓15-20mmHg ⇒ ↑ed pulse pressure
  - PulmonVR ↓15%
  - CVP & PCWP ~ normal
- CO distribution:
  - Uterine - ↑10% relative flow - mediated by corticotrophin releasing horomine
  - breast ↑2% - doubles through pregnancy
  - kidney, skin, brain, heart = all have ↑absolute flow, but unaltered proportional flow
- @ labour:
  - in 3rd stage labour CO ↑ed by 80% above pre labour values
- ECG:
• left axis dev
• T wave inversion III
• down sloping ST depression
• low voltage QRS

Coagulation
• accelerated but compensated IV coagulability:
  • ↑factors 1 (fibrinogen may double), 7,8,9,10,12
  • ↓factor 13 and antithrombin-3
  • ↑fibrinolytic system -
    - this returns to normal post partum ⇒ hypercoagulable state immediate post partum
  • placental separation activates clotting
  • ↓platelets ~20%

Plasma Proteins
• total plasma proteins ↓ from 70 ⇒ 60g/L - mostly 2nd to ↓albumin concentration
• ↓plasma oncotic pressure: 288 ⇒ 277 = ↑risk of oedema formation (incl early post partum
• ↓plasma cholinesterase activity by 30% - not clinically important

Gastro-Intestinal System
• ↑propensity for passive regurg/reflux/heartburn:
  • stomach up & rotated 45deg to R
  • pylorus up
  • LES up - into thoracic cavity
  • ↑intragastric pressure
  • labour slows gastric emptying, THEN further slowed again with narcotics
    ⇝ via progesterone & ↓motilin level ⇒ ↓oesophageal & GIT peristalsis
    ⇝ although is some conflicting evidence - uncomplicated preg may have norm gastric emptying
  • ↑volume gastric contents & ↓pH - ↑gastrin secretion from placenta

Renal System
• ↑size of kidney - pelvis
• ↑size of ureters
• flows:
  • RPF ↑80%
  • GFR ↑50%
  ⇝ ⇒ ↓filtration fraction
• ↑glucose filtered (2nd to ↑GFR) ⇒ can exceed T_{max} Gluc ⇒ glucosuria
• ↑UTI incidence
• urea, creatinine, uric acid are ↓ed in preg ↓: a normal or slightly higher level may indicate significant ↓in renal function

Neuro
• ↓MAC - due to progesterone depressive effect on CNS
  • ↓40% for iso
  • ↓25% halothane
• Neuraxial blocks = need ↓25-30% dosing of LAs:
  • ↓volume of CSF
  • ↓volume epidural space
• ↑ sensitivity to LA’s

**Metabolic/Endocrine**

- ↑ VO2 ~20% → ↑ VO2 100% in active labour
- incr in CO > incr in VO2: A-V O2 difference ↓ by 25% → ↑ ed O2 returned to heart
  \[\Rightarrow \text{slight drop in Hb not of great importance}\]
- pregnancy = diabetogenic - due to relative insulin resistance
  - ↑ insulin secretion
  - ↑ human chorionic somatotrophin = ↑ ed insulin resistance
  \[\Rightarrow \text{HCS} \sim \text{GH} \text{ ie anti-insulin}\]
- thyroid hypertrophy 2nd to HCG + oestrogens
  - ↑ TBG
  - ↑ total T3/T4
  - but free T3/T4 & TSH = normal
- parathyroid:
  - ↑ PTH ⇒ ↑ vit D ⇒ +ve Calcium effects
  \[\Rightarrow \text{but see norm serum Ca - excess goes to foetus}\]
- pituitary:
  - ↑ ACTH ⇒ ↑ cortisol, ↑ aldosterone
  - ↑ prolactin
  - ↑ MSH
  - ↑ B-endorphin
- other:
  - ↑ oestrogen
  - ↑ progesterone

**Anatomical Changes**

- physical effects include:
  - engorgement of epidural veins: uterine enlargement ⇒ vena caval compression
    \[\Rightarrow \text{↑ ed risk of IV cannulation}\]
  - engorged vertebral foraminal veins:
    - contiguous with epidural veins
    - leads to ↑ length of action of epidural LA
    \[\Rightarrow \text{one of pathways for egress of anaesthetic agent from epidural space}\]
- ↓ ed CSF in thoracolumbar region:
  - caused by:
    - enlarged epidural veins
    - ↑ ed intrabdo pressure of pregnancy
  - explains need for ↓ ed dose in spinals
- progressive ↑ of lumbar lordosis:
  - causes changes:
    - pelvis rotates on long axis of spine (ant pelvic tilt) ⇒
      \[\text{• Tuffer’s line (intercrest line) slightly higher due to ↓ ed flexion of Lx spine}\]
      \[\text{ie may be L3-4 interspace rather then L4-5}\]
    - ↓ ed space between adjacent Lx spinous processes ⇒ difficulty using midline approach
    - apex of Lx lordosis shifted caudad with ↓ ed Tx kyphosis - influence spread of intrathecal solutions
    - labour pain makes it harder to assume ideal position for performing technique
hormonal effects include:
  - softening of ligaments:
    - esp ligamentum flavum - more difficult to feel needle move through
  - ↓specific gravity of CSF ⇒ ↓ed dose required for spinal

**Anaesthetic Significance of Physiological Changes**

**Resp**
- difficult airways:
  - x8 ↑: 0.05 to 0.4%
  - obese
  - large engorged breasts
  - short neck
  - larynx slightly cephalad +/- ant angulated
  - swollen mucosa - worse in pre-eclampsia - use smaller ETT/avoid nasal
- ↑hypoxia risk:
  - ↓FRC & ↑VO₂
  - thus ↑chance of DI and ↑hypoxia risk ⇒ ↑↑risk GA dramatically
- anaesthetic changes:
  - ↓MAC - ?progesterone
  - faster induction with
    - insoluble volatiles: ↓FRC
    - soluble volatiles: ↑V_A
  - pre-oxygenation shorter due to smaller FRC ie ~3mins or 3-5 VC breaths
- hyperventilation:
  - avoid as PaCO <24mmHg ⇒ ↓uterine perfusion

**CVS**
- healthy term pt will tolerate up to 1.5L blood loss
- CO remains high 1st few hrs post partum (up to 80% > prelabour)
  - impt in preg pts with cardiac lesions eg valves, LVOT obstruction
- if norm Hb seen must think low volume state:
  - pre-eclampsia
  - HTN
  - diuretics
- venodilation +/- ⇒ ↑incidence accidental epidural vein puncture
- oxytocin & 5% dex +/- ⇒ fluid overload
- maternal bp <90 systolic with neuraxial technique = concern
  - ↓placental perfusion as system not autoregulated

**GIT**
- ↑aspiration risk in GA:
  - pain/opioids/emotional stress ⇒ delayed gastric emptying
  - hormonal effects: progesterone ⇒ ↑gastrin, ↓motilin
mass effects: ↑intra-gastric pressure, distortion of LES angle/position
\[\therefore\] full stomach precautions from 19/40 ⇒ 48hrs post partum

**Renal**

- normal or slight high levels of creat/urea ⇒ likely significant renal impairment

**Aorto-Caval Syndrome**

- supine gravid uterus ⇒ IVC compression
  - only in 15% pts due to
    - collateral flows: paravertebral, azygous, ovarian veins
    - baro-reflexs - needing intact sympathetic n.s.
  - see:
    - initially: dramatic ↓venous return
    - then: ↑afterload ⇒ further ↓CO
- usually problem of late pregnancy but can see earlier:
  - multiple pregnancies
  - polyhydraminos
  - obesity
- signs:
  - early: anxiety, sweating, nausea
  - late: profound hypotension
- Rx:
  - prevent ie no mother with regional should be allowed supine
  - OT: tilt 15deg L or use wedge
**Uteroplacental Physiology**

- diffusion barrier = 3.5um (vs 0.5um of lung)
- surface area = 16m$^2$ (vs 50-60 m$^2$ in lung)
- foetus needs high DO$_2$ to grow which met by:
  - ↑ uterine art flow = ↑ed to 600ml/min near term (x20↑)
  - HbF -
    - ↑O2 affinity (P50 = 18mmHg)
    - 2α,2γ
    - @bith = 80% of Hb; @6months <5%)
  - foetus has ↑ed Hb ~17g/dl
  - double Bohr effect

- DO$_2$ Plac = CaO$_2$ x QUA
- QUA = UPP/UVR
- where UPP = UAP – UVP

- absolute uterine blood flow↑ by x20 during pregnancy
- during pregnancy ↑ o2 extraction from uterine blood ⇒ ↓SvO2 uterine venous blood

- uterine flow = directly pressure dependant:
  - uterine vessels without stimulation = max vasodilated
  - (↓ but can vasoC to external factors eg SNS, catecholamines, hypocarbia)
  - not autoregulated
- ∴ drop in MAP is poorly tolerated compared to regions with autoregulation ie coronary, brain
**Normal Values**
(arteries (uterine or umbilical) always flow towards uterus, veins always away from it)

- **maternal circulation:**
  - Uterine A.
  - Uterine V.
  - Umbilical A.
  - Umbilical V.

<table>
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<td>CaCO2</td>
<td>48</td>
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- Uterine artery:
  - blood flow 600ml/min
- Umbilical artery:
  - blood flow back to placenta ~300ml/min

**Placental Functions**
(TIME = transport, Immunologic, Metabolic, endocrine)

**Transport**
(see next page - transplacental gas exchange)
- oxygen - double Bohr effect seen:
  - maternal side: maternal blood gains CO2 $\Rightarrow$ ↓pH $\Rightarrow$ R shift curve $\Rightarrow$ ↑O2 release
  - foetal side: CO2 is lost $\Rightarrow$ ↑pH $\Rightarrow$ L shift curve $\Rightarrow$ ↑O2 uptake
- CO2:
  - maternal hypervent $\Rightarrow$ ↑ed gradient for CO2 diffusion from foetus $\Rightarrow$ mum
  - double Haldane effect:
    - maternal side: blood deoxygenated $\Rightarrow$ ↑CO2 carrying capacity
    - foetal side: blood oxygenated $\Rightarrow$ ↑unloading of CO2
- delivery of nutrients ie glucose, aa’s, lipids
- waste removal - urea, bilirubin
- water + electrolyte delivery/exchange
- heat transfer:
  - foetus = 1deg warmer than mum
  - ↑heat returned to maternal circ $\Rightarrow$ ↑maternal skin flow

**Immunologic**
- protects foetus from infection:
  - IgG Antibodies only class able to cross placenta
    $\Rightarrow$ provide immunity for few months post birth
- protects foetus from rejection by mother

**Metabolic**
- produces:
Endocrine
- produces:
  - HCG (human chorionic gonadotropin)
    - maintains corpus luteum in early preg
  - Human placental lactogen (hPL), also called human chorionic somatomammotropin (HCS):
    - ~ GH
    - imp in regulating glucose availability for foetus by altering maternal CHO, protein, fat metab
    - insulin antagonist
    - stims erythropoiesis
  - oestriol
  - progesterone:
    - made by corpus luteum in 1st trim
      - then by placenta rest of preg
  - other: gastrin, somatomedin, human chorionic thyrotropin, placental corticotrophin

Transplacental Gas Exchange
- Bohr effect = an ↑ in PaCO2 will ↓ affinity of Hb for O2 (ie a R shift of OHDC) and vice versa
- Haldane Effect = as Hb is deoxygenated, its affinity for CO2 ↑s and vice versa

Explanation
- Ficks Law of diffusion: O2 & Co2 diffuse along their concentration gradients across the placental barrier ie O2 mother ⇒ fetus; CO2 fetus ⇒ mother:
  - area 16m2
  - diffusion constant D
  - conc gradients - as above & note mat hyperventilation
  - thickness of placental barrier 3.5um
- diffusion gradient for Co2 foetus ⇒ mother is ↑ed by maternal hyperventilation (mat PaCo2 = 30mmHg)
- diffusive transfer is enhanced by double Bohr & Double Haldane effects
  - double Bohr effect seen:
    - maternal side: maternal blood gains CO2 ⇒ ↓pH ⇒ R shift curve ⇒ ↑O2 release
    - foetal side: CO2 is lost ⇒ ↑pH ⇒ L shift curve ⇒ ↑O2 uptake
- double Haldane effect:
  - maternal side: blood deoxygenated $\Rightarrow$ ↑CO2 carrying capacity
  - foetal side: blood oxygenated $\Rightarrow$ ↑unloading of CO2

- other factors influencing gas exchange:
  - high foetal Hb conc = 170
  - high affinity of HbF for O2 (p50 18mmHg)


By Adam Hollingworth

Maternal Physiology - 12

Pain in Labour

First Stage Labour Pain

• pain via afferents from:
  - cervix
  - lower uterine segment
  - (not uterine body:
    - needs co inflammation eg chronic pain
    - pregnancy ⇒ ↓ afferents from here downregulate)
• ∴ do not feel pain from uterine distension
• cervix innervation = dual (∴ chance of referred pain):
  - endocervix & lower uterine segment (1st stage)
    - = nerve cell bodies in thoracolumbar dorsal root ganglion (DRG) T10-L1
  - vaginal cervix & upper vagina (2nd & 3rd stage only)
    - = nerve cell bodies in sacral DRG
    - C fibres
    - innervation pattern not affected by pregnancy
    - mediators incl substance P & CGRP
• 1st stage pain afferent pathway:
  - C fibres
  - paracervical region
  - hypogastric nerve & plexus
  - lumbar sympathetic chain
  - T10-L1 DRG nerve cells
  - visceral C fibre termination:
    - enter in dorsal horn
    - terminate in loose network of fibres in deep dorsal & ventral horns
      ⇐ including crossing midline to contralat side
      ⇐ explains non specific localisation of visceral pain
  - ascending tracts:
    - contralat ant spinothalamic tract ⇒ somatosensory cortex
    - spinoreticular & spinomesencephalic tract ⇒
      - areas of vigilance (reticular formation)
      - cardioresp centre (NTS, caudal medulla)
      - reflex descending inhibition (PAG, nucleus raphe magnus, cerebellum)
• (somatic afferents = localised pain: traditional C & A-delta somatic afferents enter dorsal horn &
  terminate in ipsilateral lamina I & II)
• diffuse termination explain why for visceral pain intrathecal fentanyl > morphine
  ⇐ ↑ed lipophilic ∴ can penetrate deeper into cord connections
• ∴ to achieve pain relief:
  - peripheral blockade ⇒
    - paracervical,
    - paravertebral sympathetic nerve,
    - epidural T10-L1
  - spinal cord blockade
  - should use lipophilic drug to enable deep penetration
2nd Stage Labour Pain

- same as 1st but with additional afferents from:
  - cervix (vaginal surface - as described above)
  - vagina
  - perineum
- afferents are somatic:
  - via pudenal nerve DRG (S2-S4)
- pain caused by:
  - distension
  - ischaemia
  - frank injury - stretching or surgical incision
- ∴ to achieve pain relief:
  - as stage 1
  - extension of epidural blockade T10-S4
  - pudendal nerve block

Role of Sensitisation

- amplification of pain signalling seen in labour
- cervical ripening associated & labour itself due to local inflam products
- long term oestrogen exposure ⇒ ↑sensitivity of nociceptors
- ∴ effect of periph sensitisation of cervical afferents:
  - Braxton Hicks contractions - prior to onset of labour inflam process may be as powerful as labour contractions but are painless
  - pain may ↑ with progress through process of labour due to sensitisation
  - inflam mediators may provide new targets for pain control