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

↳ 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
- P_{50} = ↑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 normovoluaemic 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 cinically 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
  ↳ ∴ slight drop in Hb not of great importance
- pregnancy = diabetogenic - due to relative insulin resistance
  - ↑insulin secretion
  - ↑human chorionic somatotrophin = ↑ed insulin resistance
    ↳ HCS ~ GH 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
    ↳ 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
    ↳ ↑ed risk of IV cannulation
  - engorged vertebral foraminal veins:
    - contiguous with epidural veins
    - leads to ↑length of action of epidural LA
      ↳ = 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) ⟹
        - Tuffer's line (intercrest line) slightly higher due to ↓ed flexion of Lx spine
        - 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 cepehalad +/- 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
- ∴ full stomach precautions from 19/40 ⟹ 48hrs post partum

Renal

- normal or slight high levels of creat/urea ⟹ likely significant renal impairement

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

By Adam Hollingworth

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

- $DO_{2\ Plac} = CaO_2 \times Q_{UA}$

- $Q_{UA} = U_{PP}/U_{VR}$
- where $U_{PP} = U_{AP} - U_{VP}$

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

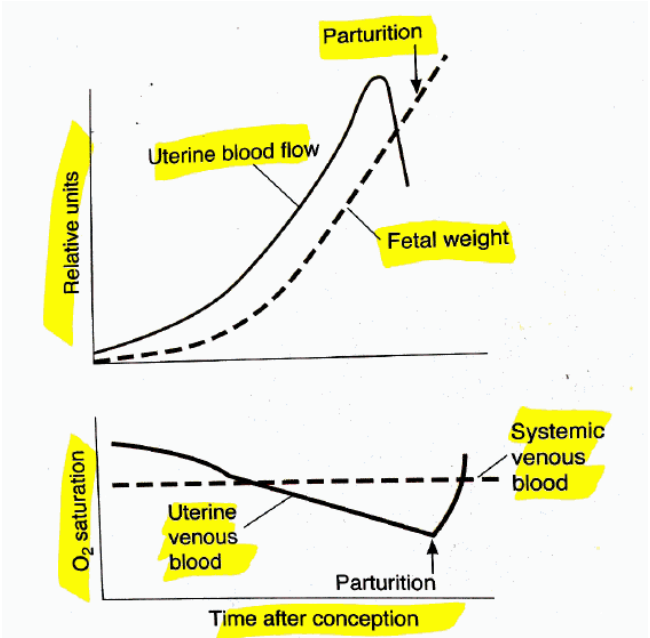


FIGURE 34–15 **Changes in uterine blood flow and the amount of O_2 in uterine venous blood during pregnancy.** (After

- 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.
PaO2	100	40	15	30
PaCO2	30	45	55	40
SO2	98	75	40	80
p50	26			18
CaO2	16	12	10	16
CaCO2	48	52	44	37

- 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 ⟹ ↓pH ⟹ R shift curve ⟹ ↑O2 release
  - foetal side: CO2 is lost ⟹ ↑pH ⟹ L shift curve ⟹ ↑O2 uptake
- CO2:
  - maternal hypervent ⟹ ↑ed gradient for CO2 diffusion from foetus ⟹ mum
  - double Haldane effect:
    - maternal side: blood deoxygenated ⟹ ↑CO2 carrying capacity
    - foetal side: blood oxygenated ⟹ ↑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 ⟹ ↑maternal skin flow

Immunologic

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

Metabolic

- produces:

- glycogen
- cholesterol
- fatty acids
- enzymes

Endocrine

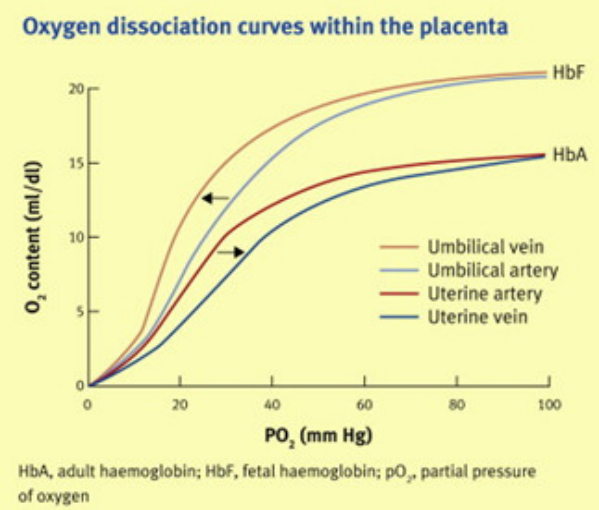
- produces:
  - HCG (human chorionic gonadotropin)
    - maintains corpus luteum in early preg
  - Human placental lactogen (hPL), also called human chorionic somatomammotropin (HCS):
    - ~ GH
    - impt in regulating glucose availability for foetus by altering maternal CHO, protein, fat metab
    - 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

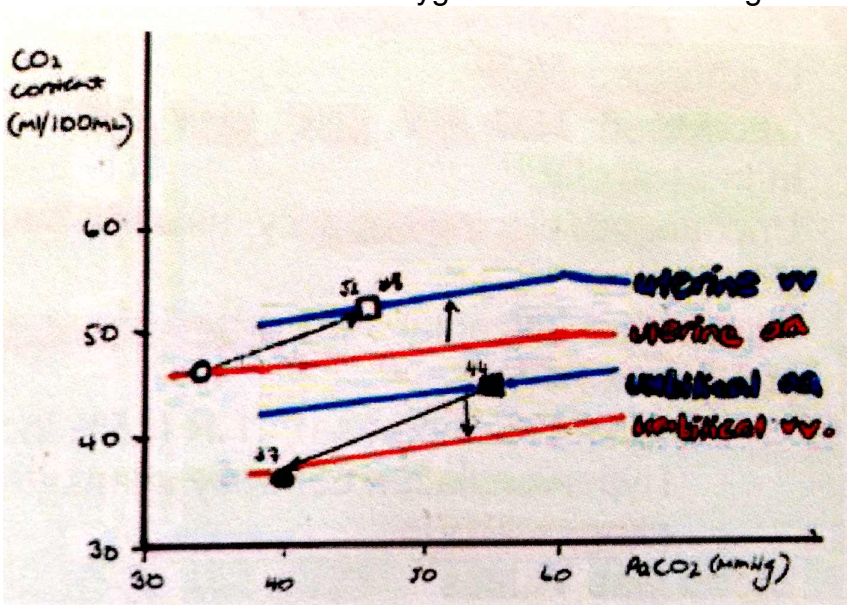
- Bohr effect = an ↑ in PaCO2 will ↓affinity of Hb for O2 (ie a R shift of OHDC) and vice versa
- Haldane Effect = as Hb is deoxygenated, its affinity for CO2 ↑s and vice versa

Explanation

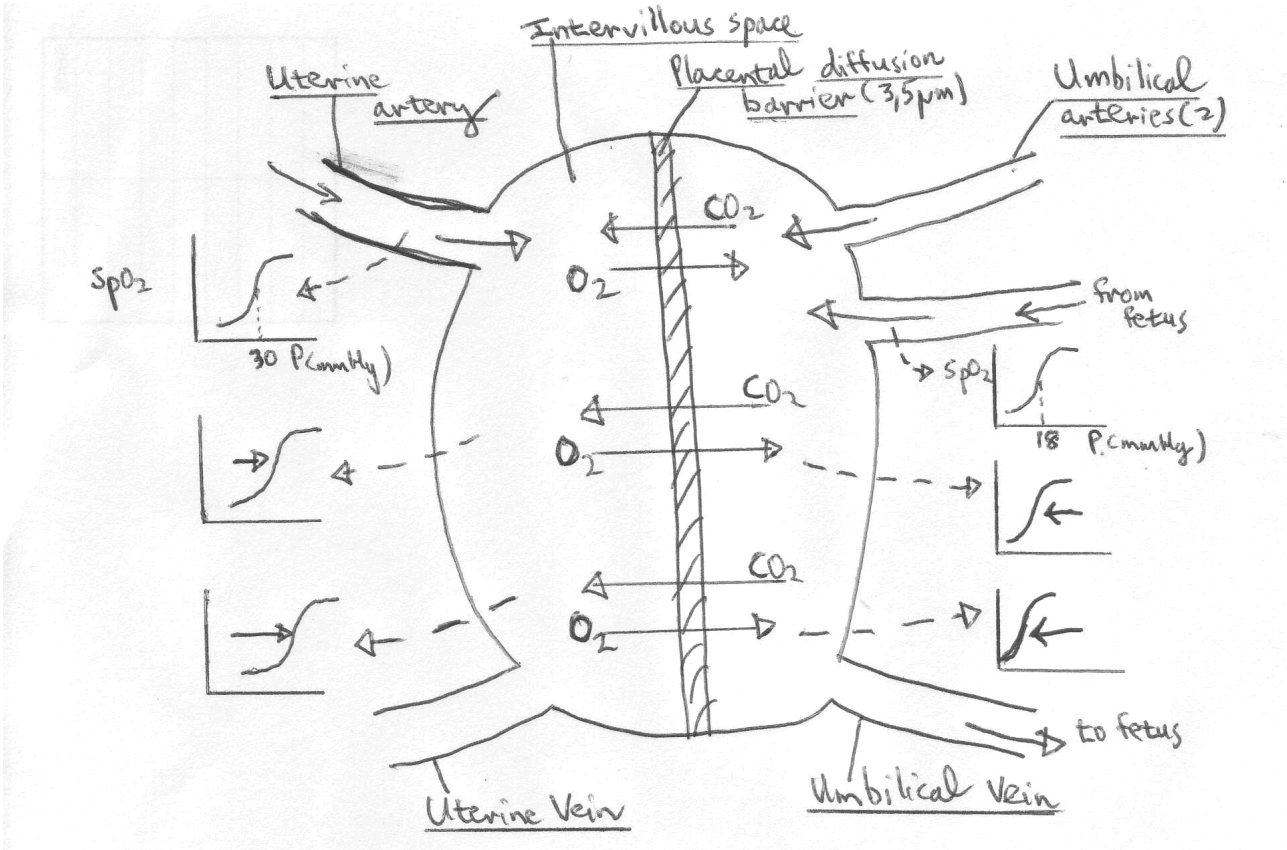
- Ficks Law of diffusion: O2 & Co2 diffuse along their concentration gradients across the placental barrier ie O2 mother ⟹ fetus; CO2 fetus ⟹ mother:
  - area 16m2
  - diffusion constant D
  - conc gradients - as above & note mat hyperventilation
  - thickness of placental barrier 3.5um
- diffusion gradient 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 ⟹ ↑CO2 carrying capacity
  - foetal side: blood oxygenated ⟹ ↑unloading of CO2



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



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