

PHYSIOLOGY CHANGES IN PREGNANCY

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RESPIRATORY - ANATOMICAL

- Capillary engorgement and oedema of upper airway
 - Worsened by PIH/PET
- Enlarged chest -> Difficult laryngoscopy with standard handles
- Diaphragm displaced cranially – but breathing more dependent on diaphragm than thorax
- ERV, RV, FRC -> decreased by 20% at term
 - Starts from middle of 2nd trimester
 - Reduced FRC causes airway closure in supine position
 - Pre-oxygenation less effective (Low FRC + ↑ O₂ consumption)
- Chest wall compliance reduced
 - Lung compliance unaffected

RESPIRATORY VARIABLES

Respiratory variable	Alteration in pregnancy
Functional residual capacity: FRC	↓ 30%
Forced expiratory volume in 1 s: FEV1	→ unchanged
FEV1/FVC	→ unchanged
Tidal volume	↑ 45%
Minute volume	↑ 20–50%
Respiratory rate	→ ↑ small increase

RESPIRATORY - PHYSIOLOGICAL

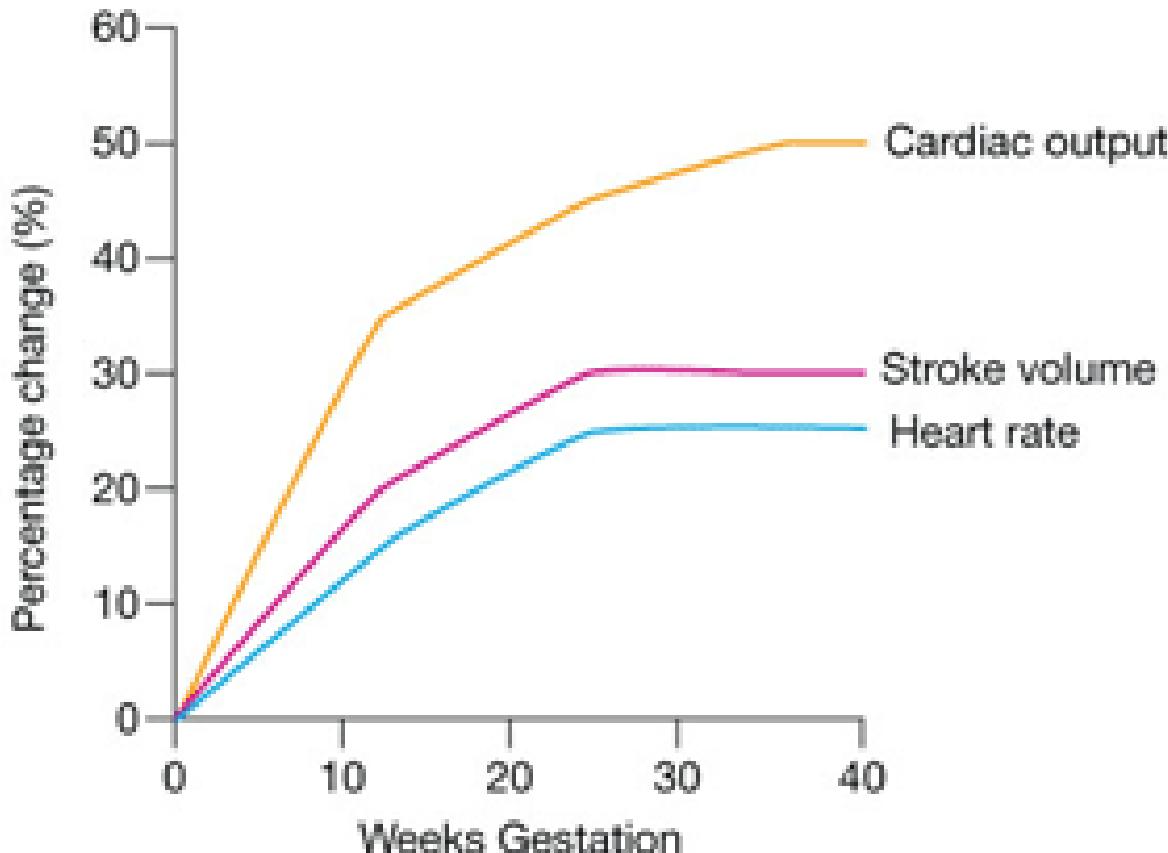
- Airway resistance reduced
 - Progesterone mediated smooth muscle relaxation
- RR ↑ by 15%, Vt by 40%
 - Progesterone mediated hypersensitivity to CO₂
- Alveolar ventilation higher (70% increase at term)
- Drop in PaCO₂ : 4.1 kPa by end of 1st trimester
 - Alkalosis compensated by drop in HCO₃
- Increased O₂ consumption (60% more)

CARDIOVASCULAR

- Hyperdynamic circulation
 - ↓SVR – hormone mediated
- But: $BP = CO(HR \times SV) \times SVR$
 - Need to maintain placental perfusion
 - CO needs to ↑
- Cardiac output increases: up to 50% by 3rd Trimester
- SV by 35%
 - Increased blood volume (↑EDV), and cardiac muscle mass
- HR by 15-25%
 - Oestrogen/ progesterone -> vasodilation -> Drop in SVR

- Upward displacement of diaphragm -> Apex moved anterior and to left
 - ECG – Left axis deviation, Depressed ST segments/inversion
- Auto-transfusion in 2nd stage
- **Avoid dehydration!**

Maternal Cardiovascular Changes



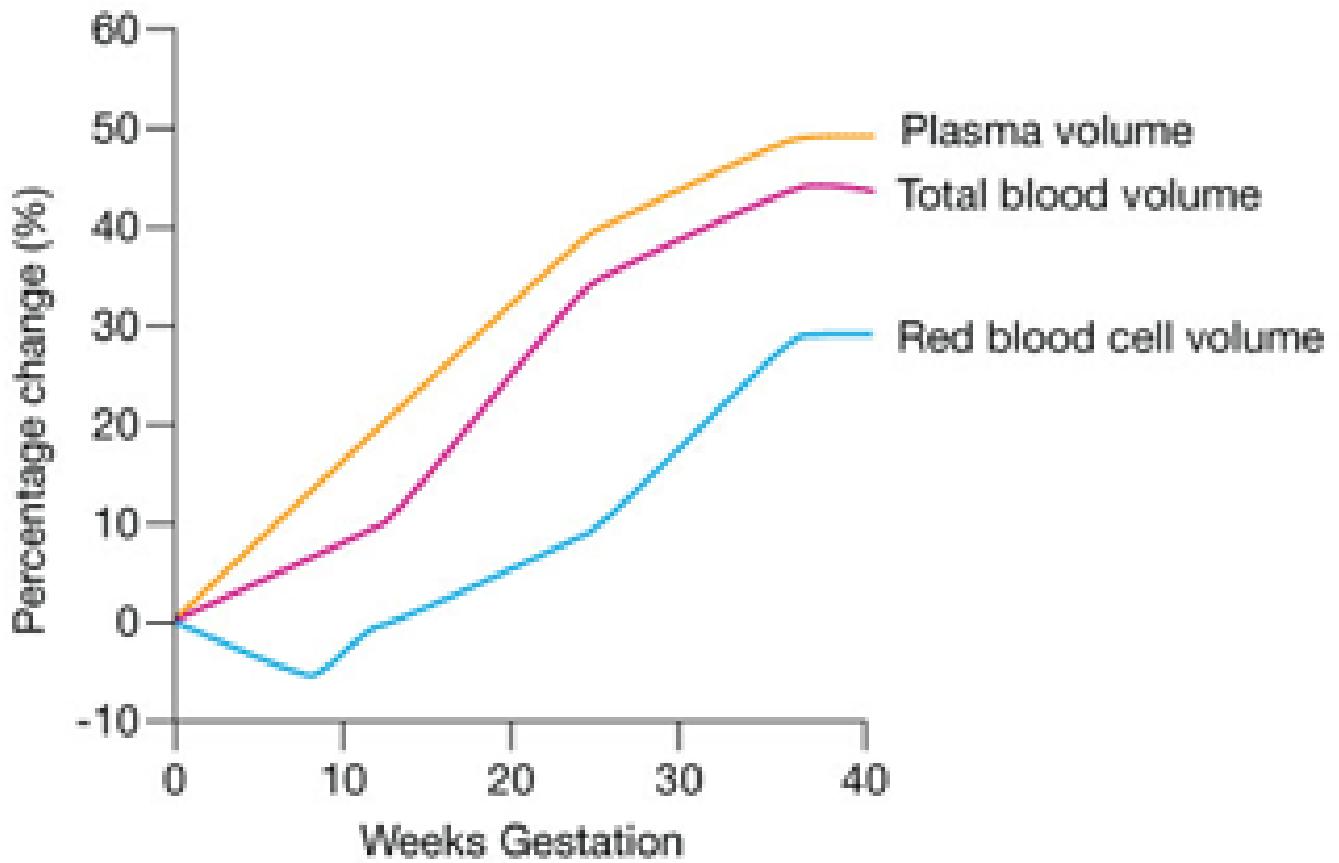
AORTOCAVAL COMPRESSION

- Compression of IVC and lower Aorta by gravid uterus
- Compression of IVC
 - Reduce venous return -> reduce preload -> reduce CO -> drop in BP
- Compression of Aorta
 - Reduction of uteroplacental and renal blood flow
- Some compensation by ↑ SVR/HR in unanaesthetised mothers
- ↑ Blood return by paravertebral and azygous systems
- **Remember to wedge!**

HAEMATOLOGY

- Blood volume increases (from 8 to 32 weeks gestation)
 - To promote placental efficiency
- Plasma volume ↑ by 45%
 - Oestrogen/progesterone mediated action of RAAS in the kidneys
- Red cell mass ↑ by 20 – 30%
 - Renal erythropoietin
- Resulting in effectively haemodilution -> Physiological anaemia of pregnancy
- Increased blood volume reduce impact of maternal blood loss at delivery
- WCC rises throughout pregnancy and peaks after delivery

Maternal Intravascular Volume Changes



COAGULATION

- Affects balance of coagulation and fibrinolysis -> Hypercoagulable state
 - 10x increase in VTE risk
- All clotting factors (except FXI and FXII) and fibrinogen levels increase
- Increased Antithrombin III and fibrin degradation products
- Platelet
 - Increased production, BUT count is lower as dilution + consumption
 - Function is normal

GASTROINTESTINAL

- Increased gastric volume + delayed emptying = ↑ Reflux
- Gravid uterus displaces stomach upwards and increases pressure
- Don't forget: H₂ blockers, prokinetics, Sodium citrate

HEPATIC

- Changes in liver function
 - Elevated GGT, ALT, LDH can occur
- ALP ↑ by up to 3x
 - Placental production
- Spider naevi and palmer erythema – can occur in normal pregnancy
- Plasma cholinesterase fall by 25%
 - Prolonged block with sux (rarely significant)

RENAL

- ↑ Renal plasma flow, ↑ GFR progressively (50-60% higher at term)
 - Caused by increased CO
- Increased clearance of: Urea, creatinine, urate, Bicarbonate
- Plasma osmolality falls
- Mild glycosuria/ proteinuria are possible
- More prone to UTI
- Possible obstruction of ureters by gravid uterus

ENDOCRINE

- Increased insulin production, but increased insulin resistance
 - Facilitates placental glucose transfer
- Gestational diabetes -> maternal hyperglycaemia -> foetal hyperglycaemia
-> Foetal hyperinsulinism
 - Maternal insulin doesn't cross placenta -> foetus makes own insulin
 - Insulin is main growth hormone for foetus -> large babies -> ↑ assisted deliveries
- Thyroid gland ↑ size (up to 20%)
 - Transient hyperthyroidism
 - Can exacerbate pre-existing thyroid disease

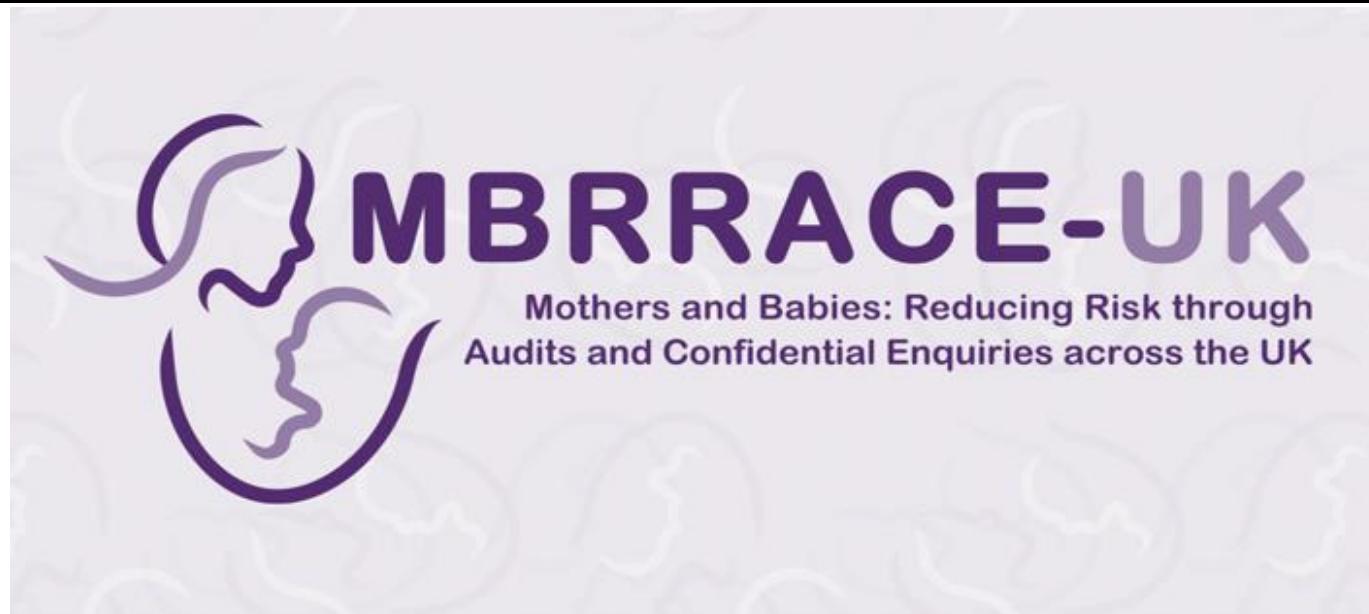
NEURO/ ANAESTHESIA

- Engorged epidural venous plexus
- LA
 - Increased sensitivity to LA centrally
 - Increased cephalad spread of spinal -> as increased abdominal pressure
- GA
 - Need lower MAC (30%)
 - Increased ventilation -> delivers more gas into alveoli
 - Other risks as previously discussed
 - “Full stomach”

REFERENCES

- Physiology of pregnancy: Clinical anaesthetic implications
 - BJA Education April 2014
- Anaesthesia UK

MBRACE REPORT



- Surveillance data on maternal deaths from 2014-16.
- Confidential Enquiry reports on deaths from psychiatric causes, deaths due to thrombosis and thromboembolism, malignancy and homicides, and morbidity from major obstetric haemorrhage.

Key messages from the report 2018



In 2014-16 **9.8 women** per 100,000 died during pregnancy or up to six weeks after childbirth or the end of pregnancy.

Most women who died had multiple health problems or other vulnerabilities.



Balancing choices:

Always consider individual **benefits** and **risks** when making decisions about pregnancy



Things to think about:



Many medicines are **safe** during pregnancy

Continuing medication or preventing illness with vaccination may be the best way to keep both mother and baby healthy - ask a specialist



Be body aware - some symptoms are normal in pregnancy but know the **red flags** and always seek specialist advice if symptoms persist

Black and Asian women have a higher risk of dying in pregnancy

White women 1 8/100,000

Asian women 2x 15/100,000

Black women 5x 40/100,000

Older women are at greater risk of dying

Aged 20-24 1 7/100,000

Aged 35-39 2x 14/100,000

Aged 40 or over 3x 22/100,000



Overweight or obese women are at higher risk of blood clots including in early pregnancy

KEY POINTS

- statistically non-significant increase in the overall maternal death rate in the UK between 2011–13 and 2014–16
- Assessors judged that 28% of women who died and 12% of women surviving with major obstetric haemorrhage had good care.
- However, improvements in care may have made a difference to the outcome for 38% of women who died and 74% of women with major obstetric haemorrhage who survived

- Maternal deaths from direct causes are unchanged with no significant change in the rates between 2011–13 and 2014–16.
- Thrombosis and thromboembolism remain the leading cause of direct maternal death during or up to six weeks after the end of pregnancy.
- Maternal suicide is the third largest cause of direct maternal deaths occurring during or within 42 days of the end of pregnancy. However, it remains the leading cause of direct deaths occurring within a year after the end of pregnancy

RECOMENDATIONS

- Obstetric haemorrhage – surgical plans for placental malpresentations
- VTE – Risk scoring
 - Obesity (\uparrow risk)
- Mental health services
 - continuity of care
- Women from vulnerable groups
 - alert symptoms/signs of domestic violence
- Women with malignancy
 - Suspected cases to be Ix in same manner as in non-pregnant population
 - ?postpone pregnancy
 - VTE

Figure 2.1: Direct and indirect maternal mortality rates per 100,000 maternities using ICD-MM and Previous UK classification systems; rolling three year average rates 2003–2016

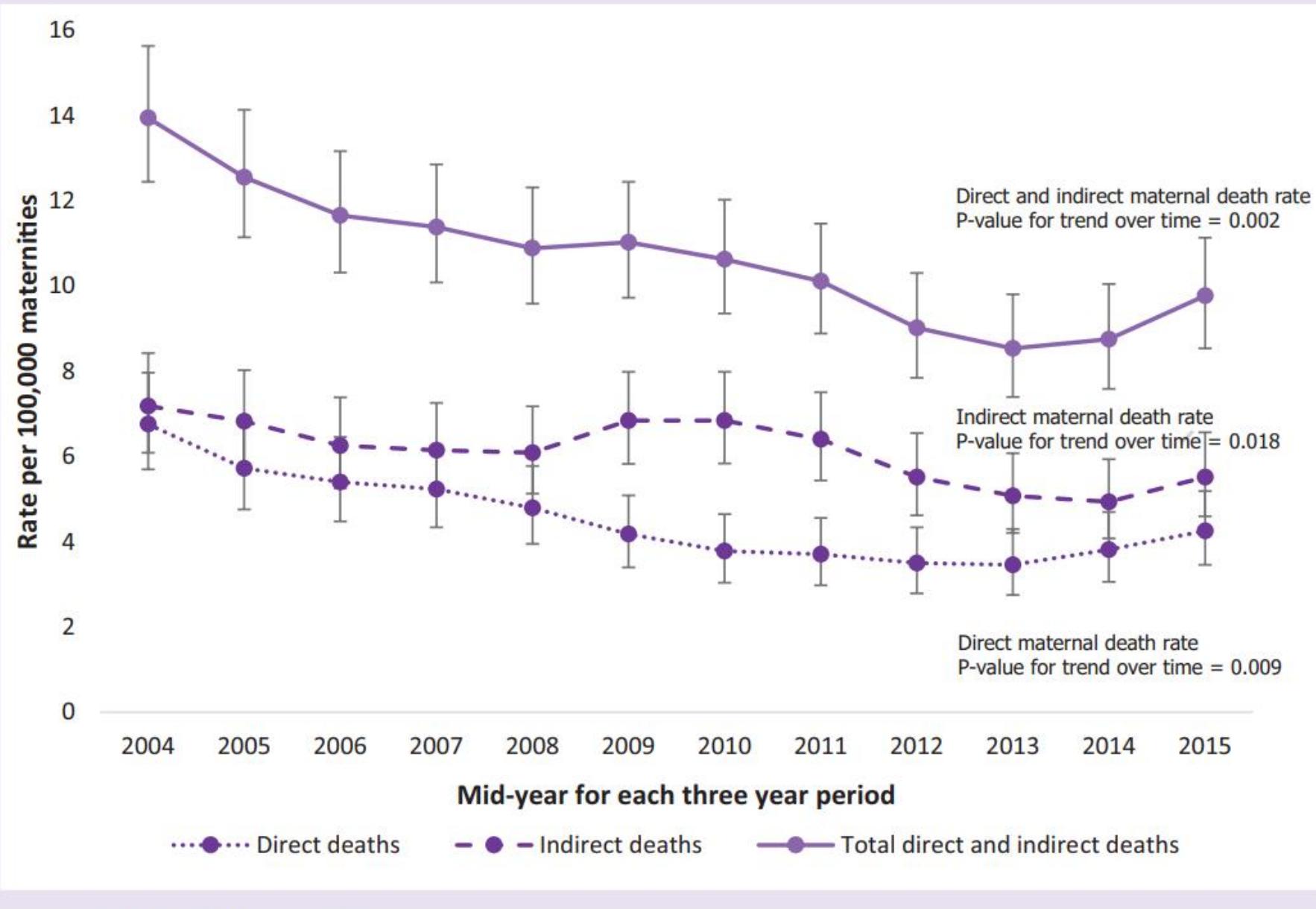
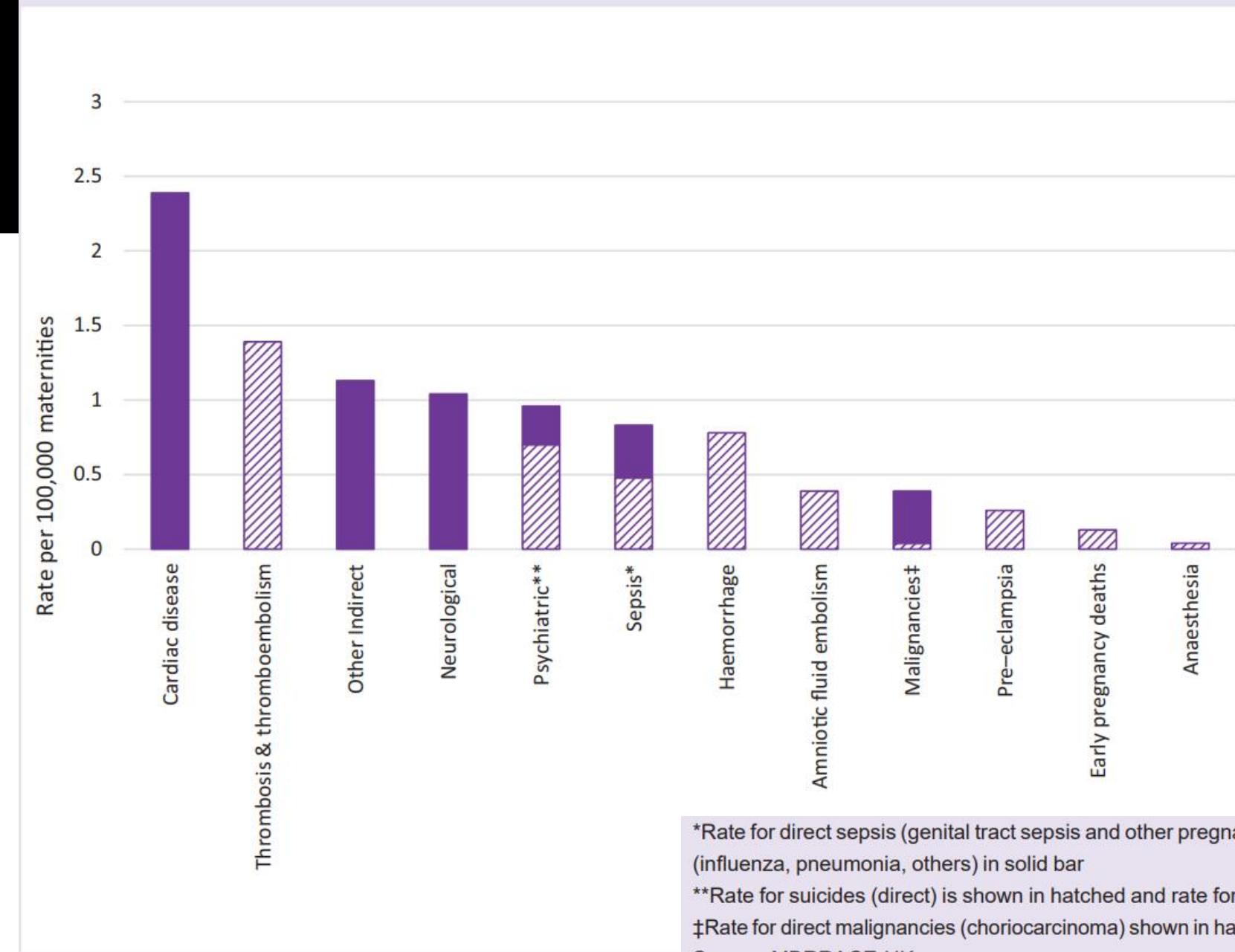


Figure 2.3: Maternal mortality by cause 2014-16



Hatched bars show direct causes of death, solid bars indicate indirect causes of death;

QUESTIONS?

Thank you