Selected topics from NSA Educational Webinars I

Physiology of Pregnancy and Anaesthetic Implications

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ABSTRACT: Series of physiological and anatomical adaptations occur in pregnancy, largely due to the actions of oestrogen and progesterone, to support fetal growth. These adaptations present unique challenges for anaesthetic management, especially during labor and delivery. This article reviews key physiological adaptations in cardiovascular, respiratory, gastrointestinal, and metabolic systems, and discusses their anaesthetic implications, focusing on optimized care for both mother and fetus.

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INTRODUCTION

Pregnancy begins following the implantation of a fertilized egg, which triggers numerous physiological changes to support maternal and fetal health. These changes, regulated mainly by pregnancy hormones, affect virtually every organ system and have critical implications for anaesthetic care.[1]

Figure 1: A pregnant woman

CARDIOVASCULAR SYSTEM ADAPTATION

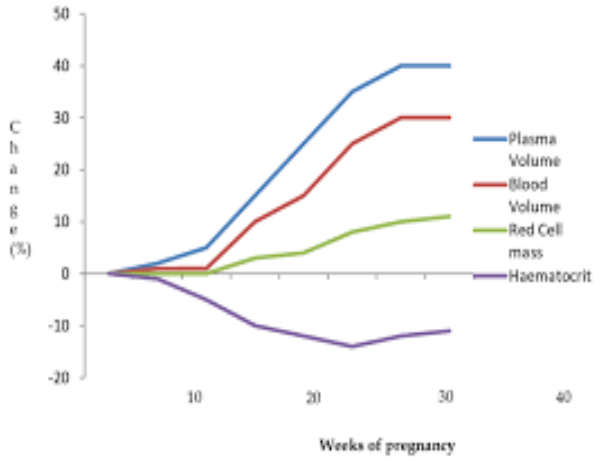

Figure 2: Haematologic changes in pregnancy

Blood volume: This increases progressively from 6-8 weeks, peaks at 32-34 weeks with little change thereafter. There is no evidence of circulatory overload in the healthy pregnant woman.Plasma volume increases by 40-50%, while red cell mass increases by 20-30%, resulting in haemodilution and decrease in haemoglobin concentration (Fig. 2). These facilitate

maternal and fetal exchange of respiratory gases, nutrients and metabolites. Furthermore, the impact of maternal blood loss at delivery is reduced via 'autotransfusion' of blood from the contracting uterus.[2]

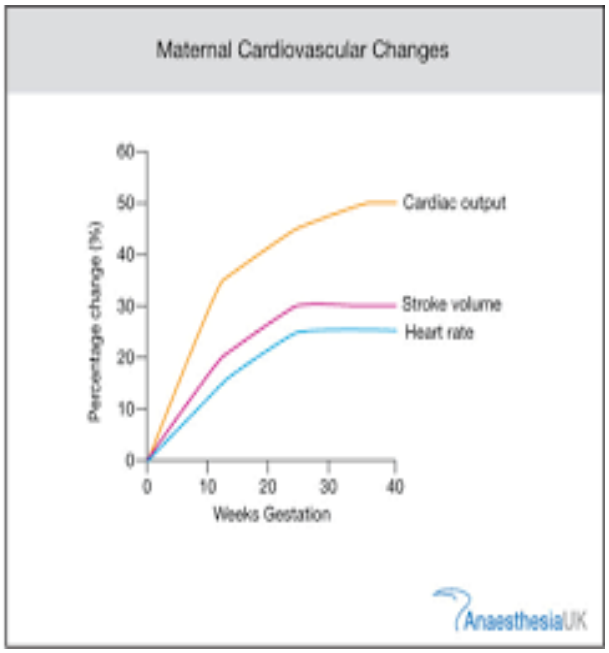

Figure 3: Maternal cardiovascular changes

Cardiac output (CO): In the first trimester, cardiac output is increased by 30-40% of the non-pregnant state. This is due to stroke volume increase (35%) and a more rapid heart rate (15%) (Fig. 3).

Systemic vascular resistance (SVR): This decreases steadily due to the development of a low resistance vascular bed (the intervillous space) and the vasodilatory effects of progesterone, oestrogens and prostacyclin. This fall in SVR contributes towards the hyperdynamic circulation. During labour, there is a further increase in CO which can be blunted with the institution of labour analgesia.[3]

Following delivery, autotransfusion compensates for the blood losses and increases cardiac output by 50% of pre-delivery values. At this point, stroke volume rises while heart rate falls. The left ventricular work of the heart is increased by 40%. This is a high risk period for parturients with cardiac disease.[2]

Blood falls during normal pregnancy. Systolic blood pressure falls by about 8% by mid-gestation, and then returns to normal by term. Diastolic blood pressure falls by 25% at mid-gestation but returns to normal at term. Vascular tone is more dependent upon sympathetic control than in the non-pregnant state, so that hypotension develops more readily and more markedly following sympathetic blockade upon institution of spinal or epidural anaesthesia. There is also a reduction in afterload and an increase in preload which, together with the increase in blood volume, may produce functional murmurs.[4]

Aortocaval Compression: Compression of the inferior vena cava (IVC) and aorta by the gravid uterus occurs during pregnancy, reducing cardiac output (Fig. 4a). The severity of this effect is dependent on patient position, gestational age, systemic blood pressure and presence of sympathetic block. IVC compression develops from 18 weeks gestation; the effect becoming maximal at 36-38 weeks.[2]

Supine Hypotension Syndrome

In the supine position, up to 8% of pregnant women experience supine hypotension syndrome – a large drop in blood pressure causing systemic signs of shock including pallor, sweating, nausea and syncope. Arterial hypotension can be observed in the lower extremities and in the uterine circulation. This can lead to fetal asphyxia and bradycardia.[5]

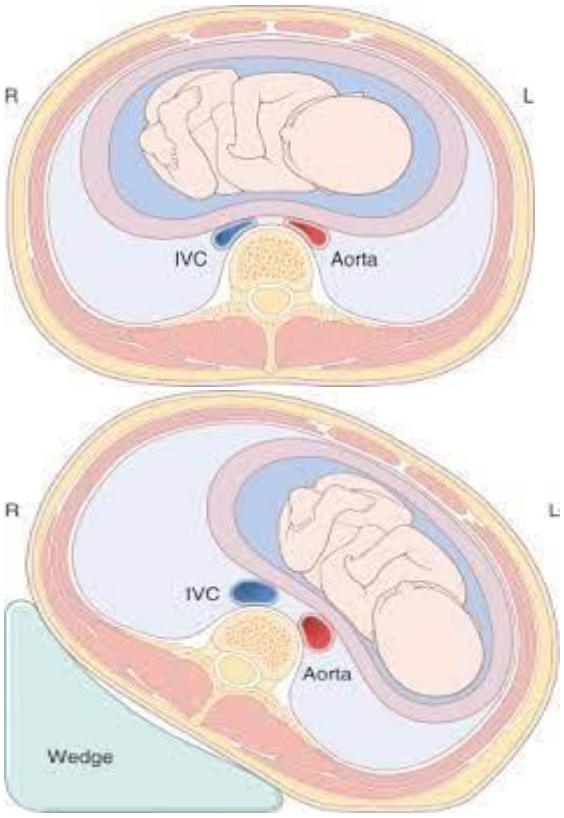

Figure 4a: Aortocaval compression
Figure 4b: Aortocaval compression relieved

CARDIAC SIZE, POSITION AND ECG

The heart is enlarged by both chamber dilation and hypertrophy. Dilation across the tricuspid valve can initiate mild regurgitant flow causing a normal grade I or II systolic murmur. Upward displacement of the diaphragm by the enlarging uterus moves the apex beat outwards and upwards. These changes lead to ECG findings of left axis deviation, sagging ST segments and T wave inversion or flattening in lead III.[4]

HAEMATOLOGICAL SYSTEM

Red cell mass increases by 20- 30%. Leukocyte counts usually remain within the upper limits of normal, though marked elevations develop and after parturition. Fibrinogen, total body and plasma levels of factors I, II, V, VII, VIII, IX, X and XII increase markedly. Factors XI, XIII and antithrombin III levels decrease. The number of platelets also increase, yet not above the upper limits of normal and this is combined with a decrease in fibrinolytic activity. All these changes tend to make pregnancy a relatively hypercoagulable state, preventing bleeding at delivery but increasing the risk of thromboembolic complications. At delivery there is increase in fibrinolytic activity, especially at the third stage, and a high concentration of plasminogen activators in the uterus, both of which may activate disseminated intravascular coagulopathy.[2]

PLASMA PROTEINS

Plasma albumin concentration is reduced but globulin and fibrinogen concentrations increase. Overall plasma protein concentration drops to 65-70g/L. This is associated with a decrease in total colloid osmotic pressure and an altered drug binding capacity. The plasma concentration of pseudocholinesterase decreases by 25%, there is an increase in erythrocyte sedimentation rate (ESR) and blood viscosity.[2]

RESPIRATORY SYSTEM

Hormonal changes lead to capillary engorgement and mucosal swelling of the nose, oropharynx, larynx, and trachea. Therefore, symptoms of nasal congestion, voice change and upper respiratory tract infection may prevail throughout gestation.[2] These symptoms can be exacerbated by fluid overload or oedema associated with pregnancy-induced hypertension (PIH) or pre-eclampsia. Also, manipulation of the airway can lead to profuse bleeding from the oropharynx. Endotracheal intubation can be difficult and a smaller than usual endotracheal tube may be required to fit through the larynx. Airway resistance is reduced, as a result of progesterone-mediated relaxation of the bronchial musculature. The gravid uterus causes elevation of the diaphragm and upward displacement of the lungs, but total lung capacity decreases only slightly because of compensatory increase in the transverse and anteroposterior diameters of the chest by 5-7cm, as well as flaring of the ribs (Fig 5).[4] These changes are brought about by hormonal effects that loosen ligaments. Breathing is more diaphragmatic than thoracic during gestation, as chest wall compliance is decreased, though lung compliance is relatively unaffected.

From mid-second trimester, expiratory reserve volume (ERV), residual volume (RV) and functional residual capacity (FRC) progressively decrease with an approximately 20% decrease at term.[1,5] Tidal volume increases by 40% and respiratory rate increases by 15% (2-3 breaths.min^{-1}). This results in progressive increase in minute ventilation which starts soon after conception and peaks at 50% above normal levels around the second trimester. Deadspace remains unchanged, thus, alveolar ventilation is about 70% higher at the end of gestation.[1] Arterial and alveolar carbon dioxide tensions are decreased by the increased ventilation (Fig 5). An average PaCO2 of 4.3kPa (32mmHg) and arterial oxygen tension of 13.7kPa (105mmHg) persist. Compensatory decreases in serum bicarbonate prevents development of alkalosis.

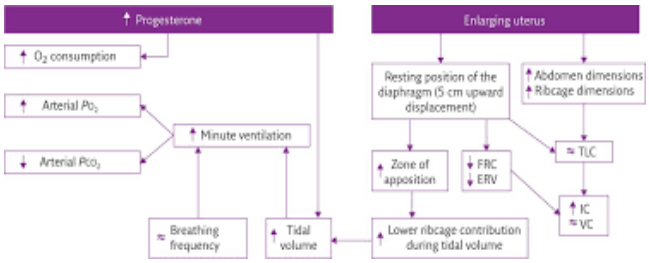

Figure 5: Respiratory system changes

ANAESTHETIC IMPLICATION

General anaesthesia is not the routine choice for caesarian section (enlarged breasts and oedema of the airway make intubation more difficult). There is increased oxygen consumption and decreased reserve due to the reduced functional residual capacity, which can result in a rapid fall in arterial oxygen tension during apnoea. This occurs despite careful maternal positioning and preoxygenation.[1] The increased minute ventilation combined with decreased functional residual capacity hastens inhalational induction or changes in depth of anaesthesia when breathing spontaneously. Failed

intubation rate increases to 1 in 250 and is worse in obese and pre-eclamptic patients. There is also an increased risk of aspiration because of the gastrointestinal changes.[2]

GASTROINTESTINAL SYSTEM

The enlarging uterus causes a gradual cephalad displacement of the stomach and intestines. At term, the stomach is vertical rather than its normal horizontal position, resulting in increased intragastric pressure, change in the angle of the gastroesophageal junction and increased oesophageal reflux.[2] The intragastric pressure is normally 7-8cmH_2O. It rises to 13-17cmH_2O in pregnancy and can increase up to 40cmH_2O with multiple gestation and in the obese. As early as 8-10 weeks of gestation, there is increased gastric volume and decreased pH, with delayed gastric emptying only occurring during labour and delivery.[4,5] The pregnant woman is a 'full stomach' patient with increased risk of aspiration during most of gestation. Aspiration of acidic gastric contents results in chemical pneumonitis (Mendelson's syndrome).[4]

ANAESTHETIC IMPLICATION: The danger of aspiration is almost eliminated when regional anaesthesia or inhalational analgesia is used. A nonparticulate oral antacid, 30ml of 0.3 Molar sodium citrate or bicarbonate, should be given immediately prior to induction of general anaesthesia to all pregnant women. For those requiring general anaesthesia, supine position with a left lateral tilt will minimise further increase in intragastric pressure (Fig. 4b).[2] Rapid sequence induction (RSI) and intubation of the trachea with a cuffed endotracheal tube should be employed (preoxygenation prior to induction then no positive pressure ventilation prior to insertion of the endotracheal tube and cricoid pressure [Sellick's maneouvre] during induction and maintained until endotracheal tube placement in the trachea has been confirmed).[2]

METABOLISM: All metabolic functions increase with carbohydrate metabolism showing the most dramatic changes. Hormones such as human placental lactogen, progesterone, prolactin and cortisol, together with reduced liver enzyme activity of glucokinase and phosphofructokinase, result in an insulin resistant state.[5] Healthy women counteract this by increasing insulin production, however, women with gestational diabetes are unable to do this.By 15 weeks of gestation, maternal blood glucose levels after an overnight fast are considerably lower than in the non-gravid state.[3] This is due to expanded maternal blood volume, increased placental transfer of glucose and loss through the kidneys because of a low renal threshold.

Plasma concentrations of alkaline phosphatase increase three-fold in pregnancy due to placental production.

Protein synthesis is reduced leading to reduced plasma cholinesterase concentrations. This may cause prolongation of neuromuscular block after administration of succinylcholine but this is rarely clinically significant.[2]

RENAL SYSTEM ADAPTATIONS

Renal plasma flow and glomerular filtration rate (GFR) increase progressively from the first trimester. At term, both are 50-60% higher than in the non-pregnant state. Creatinine clearance is elevated. Blood urea and serum creatinine are reduced by 40%.[4] The increase in GFR may overwhelm the ability of the renal tubules to reabsorb, leading to glucose and protein losses in the urine (Table 1). Mild glycosuria (1-10g/day) and/or proteinuria (up to 300mg/day) can occur in normal pregnancy. The activities of the renin-angiotensin-aldosterone system and progesterone are increased.[2] These lead to sodium and water retention, conservation of potassium and reduced plasma osmolality. After the 12th week of gestation, atony of the renal calyses and ureters can result from effect of progesterone. The enlarging uterus can compress the ureters as they cross the pelvic brim, obstructing flow and leading to further dilatation. These changes may contribute to the frequency of urinary tract infections during pregnancy.[2]

Table 1: Renal system changes in pregnancy

Increased	**Decreased**
Renal plasma flow	Renal tubular reabsorption
Glomerular filtration rate	Plasma urea concentration
Urine volume	Plasma creatinine concentration
Glycosuria	
Proteinuria	
Creatinine clearance	

CENTRAL NERVOUS SYSTEM (CNS)

Aortocaval compression by the gravid uterus causes epidural veins to become engorged, decreasing the volume of the epidural space, hence, solutions injected

into the epidural space will spread more extensively.[1] Cerebrospinal fluid (CSF) pressure is increased by aortocaval compression and by uterine contractions in labour. Constituents of CSF and its specific gravity are unchanged. Sympathetic nervous system activity increases and is maximal at term. The effect is largely on the venous capacitance vessels of the lower limbs, and counteracts IVC compression by the gravid uterus. Hence, sympathetic block due to spinal or epidural anaesthesia can result in a marked decrease in blood pressure in pregnant women compared with non-pregnant patients.[2]

ANAESTHETIC IMPLICATIONS

Local anaesthetic doses for epidural and spinal anaesthesia are reduced due to:

- decreased volumes of epidural and subarachnoid spaces
- increased nerve fibre sensitivity to local anaesthetics
- decreased $PaCO_2$ leading to reduced buffering capacity, therefore, local anaesthetics remain as free bases for longer.

Minimal alveolar concentration (MAC) of inhalational agents is reduced by 40%, possibly due to increased progesterone levels.[2]

MUSCULOSKELETAL SYSTEM

During pregnancy, there is general ligamentous relaxation due to placental production of the hormone relaxin. Relaxin widens pubic symphysis and increases joint mobility. As the uterus enlarges, lumbar lordosis is enhanced and often results in low back pain.[4]

WEIGHT GAIN

Weight increases by 10-12 kg in pregnancy due to increases in body water and fat, the fetus, the placenta, amniotic fluid, uterine and breast enlargement. Human placental lactogen secretion is responsible for some of these changes.[3]

CARDIOPULMONARY RESUSCITATION (CPR) IN PREGNANCY

This entails the same basic principles of resuscitation as in any patient; however, certain modifications are necessary to account for the physiologic changes that occur during the pregnancy.[6] These include left uterine displacement (LUD) to relieve the compression of the inferior vena cava and aorta by the gravid uterus (Fig. 4b), intubation using RSI with cricoid pressure and timely perimortem cesarean section (PMCS) within 5 minutes of cardiac arrest if no return of spontaneous circulation (ROSC), and resuscitation is deemed unsuccessful. The PMCS is performed if the gestational age is at least 20 weeks or the gravid uterus is evident.[7]

HIGH RISK GESTATION

Refers to one in which the mother or the fetus has an increased risk of adverse outcomes. This includes

- maternal age <16 or >35 years
- Chronic medical conditions; hypertension, diabetes mellitus, cardiovascular or renal disease, thyroid disorder, and haemoglobinopathies.
- Pregnancy induced hypertension
- Rhesus iso-immunization
- Multiple gestation
- Post-term pregnancy
- Previous cervical incompetence

SUMMARY

A good understanding of the physiological changes in pregnancy is essential in the management of the well woman, as well as those women who have pre-existing medical condition(s). This gives the anaesthetist the ability to provide quality perioperative care to the pregnant woman. Knowledge of principles of CPR in a parturient in the health care team is crucial to expedite positive outcomes for both the mother and the fetus.

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