Physiology of pregnancy

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Abstract

During pregnancy, maternal physiology undergoes continual adaptation. These, often interlinked, changes affect all the body systems and are effected by the hormonal influences of the placenta and mechanical adaptations required to accommodate the growing fetus. These expected physiological changes can lead to decompensation in parturients with pre-existing co-morbidities or unmasking of pre-pregnancy disease. A sound knowledge of the expected maternal changes is essential to enable accurate interpretation of physiological and laboratory parameters and implementation of care plans to reduce complications.

Keywords Obstetrics; physiology; pregnancy

Royal College of Anaesthetists CPD Matrix: 1A01, 2A09, 2B06

Learning objectives

After reading this article, you should be able to:

- describe the changes to lung volumes during pregnancy at term
- chart and explain the physiological anaemia of pregnancy
- state by how much the cardiac output increases in the third trimester and by what mechanisms

Introduction

During pregnancy, maternal physiology undergoes continual adaptation. These, often interlinked, changes affect all the body systems and are effected by the hormonal influences of the placenta and mechanical adaptations required to accommodate the growing fetus. Knowledge of these changes is essential for safe anaesthetic practice.

Cardiovascular

Most cardiovascular changes occur early in pregnancy. Vascular smooth muscle relaxation occurs in response to increased circulating levels of progesterone, oestrogen and prostaglandins, leading to a reduction in systemic and pulmonary vascular resistance. Cardiac output gradually increases, eventually by up to 30–50% during the third trimester. The increase in cardiac output is as a result of an increase in heart rate and stroke volume, secondary to ventricular hypertrophy and increased end diastolic volume. Increases in stroke volume peak at around weeks 16–24.

Blood pressure at term is usually maintained, although there may be a transient reduction earlier in pregnancy with a widening of pulse pressure as diastolic pressure is more significantly affected than systolic. Parturients may also develop murmurs during pregnancy, these can be flow murmurs as a result of the increased cardiac output or tricuspid and mitral regurgitant murmurs due to dilatation of these valves.

As pregnancy progresses, diaphragmatic elevation leads to displacement of the heart upwards and to the left. This can result in ECG changes including leftward axis deviation and T wave inversion in the lateral leads and lead III. These changes do not, in themselves, have clinical significance but may mask further changes secondary to pathological processes. The increase in heart rate necessary to maintain the increased cardiac output may present as sinus tachycardia and may predispose to tachyarrhythmias.

Aorto-caval compression by the gravid uterus in the supine position can lead to profound hypotension. This is widely reported to occur from 20 weeks' gestation onwards. Compression of the inferior vena cava produces a reduction in preload and, therefore, cardiac output. The resultant increase in sympathetic tone causes vasoconstriction and diversion of blood flow through the vertebral and azygos veins, allowing maintenance of blood pressure. However, this adaptive mechanism is not present in up to 10% of parturients and those who have sympathetic blockade as a result of neuraxial anaesthesia. Regardless of the parturient's ability to compensate for aorto-caval compression, there can still be significant compromise of utero-placental perfusion. To prevent this, the uterus can be displaced by positioning the parturient with a left lateral tilt or, if this is not feasible, by manual displacement of the uterus.

Respiratory

The alterations in maternal respiratory physiology occur as a result of hormonal and biochemical effects on the central respiratory centre, via local effects on the respiratory smooth muscle or by mechanical effects of the maturing fetus.

Circulating progesterone stimulates the respiratory centre, leading to an increase in minute ventilation, primarily by an increase in tidal volume (by ~40%) and by an increase in respiratory rate (by ~15%). The resulting increase in alveolar ventilation can produce a decrease in arterial partial pressures of carbon dioxide ($PaCO_2$) and, consequently, a leftward shift of the oxygen-haemoglobin dissociation curve. However, maternal levels of 2,3-DPG also increase throughout pregnancy which leads to the overall rightward shift in the oxygen dissociation curve, facilitating oxygen transfer to the fetus. The increased minute ventilation results in a respiratory alkalosis that is partially compensated by increased renal bicarbonate excretion. Parturients therefore usually have a serum pH at the upper end of the normal range.

A small increase in PaO_2 occurs during the third trimester (to ~14 kPa), however as the parturient approaches term, the

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Lung volume changes by term

Lung volume	Effect at term compared to pre-pregnancy
Expiratory reserve volume	Decrease 20–30%
Functional residual capacity	Decrease 20%
Tidal volume	Increase 30–40%
Residual volume	Decrease 20%
Vital capacity (VC)/Forced expiratory volume in 1 second (FEV_1)	Unchanged

Table 1

increase in cardiac output is unable to keep up with the increased oxygen demands of the gravid uterus and fetus, and the PaO_2 falls (to <13.5 kPa). By term there is a 60% increase in CO_2 production and oxygen consumption.

During labour there is a further increase in respiratory rate which results in an acute fall in $PaCO_2$. This increases the affinity of maternal haemoglobin for oxygen and, combined with an increase in metabolic rate and oxygen consumption, can compromise oxygen delivery to the fetus.

Expected changes to lung volumes at end term are shown in Table 1.

The presence of the gravid uterus can have a 'splinting effect' on the diaphragm. This is initially compensated for by increases in the transverse and anterior-posterior diameters of the chest, facilitated by increased ligamentous laxity which allows flaring of the lower rib cage. However, inspiration remains largely a function of diaphragmatic movement and, by term, functional residual capacity (FRC) is reduced by approximately 20%. This, combined with greater oxygen consumption, renders parturients more prone to desaturation during induction of general anaesthesia. Airway management in obstetrics can also be more complex due to anatomical issues (enlarged breasts and an increase in chest wall diameter), mucosal oedema, dilation of small vessels and increased mucosal friability. A more difficult laryngoscopy view, compounded by rapid desaturation and urgency of the surgery results in a challenging environment.

The importance of optimum pre-oxygenation and positioning is essential prior to embarking on general anaesthesia.

The concomitant increase in minute volume and decrease in FRC, speeds up the 'wash-in' of volatile anaesthetic agents. Adequate alveolar concentrations are therefore achieved more quickly on institution of inhalational anaesthesia.

The balance between bronchodilatation (effected by prostaglandin E_2 and progesterone) and bronchoconstriction (effected by PFG2a, decreased RV and decreased $PaCO_2$) determines airway resistance.

Diffusing capacity may increase in early pregnancy although this is not clinically significant.

Haematological

The peripartum period sees widespread adaptations in the haematological system with a marked increase in risks including anaemia, thromboembolism and consumptive coagulopathies (Table 2).

Increased secretion of aldosterone (by activation of the renin–angiotensin axis) results in an increase in total body water and, consequently, plasma volume. Erythropoiesis also increases by around 30%. These changes are illustrated in Figure 1. The resulting dilution of red cell mass (physiological anaemia of pregnancy) is reflected by a reduction in haematocrit.

During labour, each contraction 'squeezes' blood back into the circulation. After delivery, approximately 500 ml of blood is returned into the circulation. While most parturients tolerate this without adverse effect, it can contribute to decompensation in those with existing cardiac disease. Plasma volume has been demonstrated to revert to that of pre-pregnancy within 6 days of delivery.

Alterations in the haematological system at term pregnancy

Increased	Unchanged	Decreased
Blood volume 30–45%	Mean corpuscular haemoglobin concentration	Haematocrit 35–45%
Plasma volume 45%	Lymphocyte/T-cell (although function reduced)	Plasma protein 10–14%
Red cell mass 33%	Bleeding time	Plasma oncotic pressure (haemodilution)
White cell count 8%		Antithrombin III
Clotting factors (I, VII, VIII, X, XII, prekallikrein, von Willebrand factor, thrombin		Platelets
Activated partial thromboplastin time, prothrombin time		
Fibrinogen levels 50–80%		
Renal erythropoietin/Reticulocyte count		
RBC 2,3-diphosphoglycerate (rightward shift in oxygen-haemoglobin dissociation curve)		
Serum albumin concentration		
Venous hydrostatic pressure		
Erythrocyte sedimentation ratio		
Serum lipids 40–60%		

Table 2