INTRODUCTION

Maternal changes in pregnancy occur as a result of hormonal alterations, mechanical effects of the gravid uterus, increased metabolic and oxygen requirements, metabolic demands of the feto-placental unit, and haemodynamic alterations associated with the placental circulation. Such changes become more significant as pregnancy progresses, and they have major implications for anaesthetic management, especially in high-risk parturient ***(Apuzzio and David, 2010).***

Pregnancy and parturition are considered high-risk when accompanied by conditions unfavorable to the well-being of the mother and/or fetus. Maternal problems may be related to pregnancy, such as pre-eclampsia, eclampsia and other hypertensive disorders of pregnancy, or antepartum haemorrhage resulting from placenta previa or abruptio placentae. Prematurity; (gestation of less than 37 weeks), postmaturity; (42 weeks or more), intra-uterine growth retardation and multiple gestation are fetal conditions associated with risk. During labor and delivery fetal malpresentation; (breech, transverse lie), placental abruption, compression of the umbilical cord; (prolapsed nuchal cord), precipitous labor, or intrauterine infection (prolonged rupture of membranes) may increase the risk to the mother or the fetus ***(Banks and Levy, 2010).***

Because the high-risk parturient may have received a variety of drugs, anaesthesiologists must be familiar with potential interactions between these drugs and the anaesthetic drugs they plan to administer ***(Santos et al., 2011).***

The role of the anaesthetist in maternal resuscitation and management of life□threatening obstetric emergencies has been reviewed recently. The anaesthetist should also be an active participant in measures that might ameliorate isolated fetal compromise and even prevent the need for cesarean section ***(Thurlow and Kinsella, 2010).***

In general, the aim of anaesthetic management is maintenance of maternal cardiovascular function and oxygenation, maintenance and possibly improvement of utero-placental blood flow and creation of optimal conditions for a painless, atraumatic delivery of an infant without significant drug effects ***(Birnbach and Browne, 2010).***

Aim of the Essay

The aim of this essay is to spotlight the most important obstetric emergencies that occur during labour and how to deal with them throughout anaesthesia.

PHYSIOLOGICAL CHANGES DURING PREGNANCY

During pregnancy, there are major alterations in nearly every maternal organ system. These changes are initiated by hormones secreted by the corpus luteum and placenta. The mechanical effect of the enlarging uterus and compression of surrounding structures playing an increasing role in the second and third trimesters. This altered physiologic state has important implications for the anaesthesiologist caring for the pregnant patient ***(Santos et al., 2011).***

Cardiovascular Changes

The cardiovascular system (CVS) adjusts throughout pregnancy to meet the changes that occur. Where there is increased oxygen consumption to meet the metabolic demands of the growing fetus ***(Santos et al., 2011).***

Cardiac output (COP); increases from the fifth week of pregnancy and reaches its maximum levels at approximately 32 weeks, after which there is only a slight increase until labor, delivery and the postpartum period. Although increase in COP is due to an increase of both stroke volume and heart rate, the more important factor is stroke volume, which increases by 20% to 50% at term from nonpregnant values ***(Birnbach and Browne, 2011)***.

During labor, COP is further increased both from the sympathetic stimulation of pain, as will as the episodic autotransfusion of blood into central circulation from the contracting uterus. It is estimated that with each uterine contraction, 300-500 mL enters the maternal system. The greatest increase in COP occurs immediately after delivery when COP can be up to 80% above nonpregnant values. This again is attributed to autotransfusion from the contracted uterus. Prepregnancy COP levels are generally regained several weeks postpartum ***(Glosten, 2008).***

Blood volume; maternal blood volume increases markedly during pregnancy. Plasma volume increases from 40mL/kg before pregnancy to 70mL/kg during late pregnancy, and red blood cell volume increases from 25-30mL/kg. The latter increases at a slower rate than the former, accounting for the relative anaemia of pregnancy. Haemoglobin concentration, however, usually remains greater than 11gm/dL ***(Morgan et al., 2013).***

The autotransfusion adds 300-500mL to the central circulation with each uterine contraction. At term, maternal blood volume has increased by 1000-1500mL, in most women allowing them to easily tolerate the blood loss associated with delivery. Average blood loss during an uncomplicated vaginal delivery is 400-500mL. Blood loss during cesarean delivery varies widely, with 500-1400mL being reported ***(Glosten, 2008).***

Blood volume does not return to normal until 1-2 weeks after delivery. Because the increase in plasma volume is mainly due to water rather than colloid component, so, oncotic

pressure-pulmonary capillary wedge pressure gradient decreased significantly. Therefore, a pregnant female is more liable to develop pulmonary oedema ***(Morgan et al., 2013).***

Heart rate; changes in heart rate are extremely difficult to reliably quantify, but it is thought that the approximately 20% increase in heart rate is present by the 4th week of pregnancy. Tachyarrhythmias are more common, especially later in pregnancy as a result of both hormonal and autonomic factors ***(Duvekot and Peeter, 1994).***

Blood pressure; vasodilator effect of prostaglandin, estrogen, prostacycline and progesterone causes; decreased systemic vascular resistance by 20% therefore, diastolic blood pressure is decreased by 20%, but little change in systolic blood pressure occurs due to increased blood volume and cardiac output. Decreased pulmonary vascular resistance by 34%, but no changes in pulmonary artery pressure or pulmonary capillary wedge pressure occurs due to increased blood volume and cardiac output. Decreased tone in capacitant veins but no change in central venous pressure due to increased blood volume and cardiac output ***(Glosten, 2008).***

Blood pressure increases during painful labor. Additional stresses are imposed by uterine contractions, which cause, in effect, an autotransfusion. After delivery, the same autotransfusion occurs. In addition to an increase in central blood volume, obstruction of the vena cava is relieved. As a result, there is a marked increase (up to 80% of prelabor values) in stroke volume and cardiac output immediately postpartum.

Patients with limited cardiac reserve may experience cardiac failure at this time ***(Glosten, 2008).***

Despite the increase in blood volume and cardiac output, the parturients at term are susceptible to hypotension, when in the supine position. When the patient is supine, the gravid uterus partially or completely compresses the aorta and inferior vena cava, leading to decreased venous return, decreased cardiac output, hypotension, and reduced uterine blood flow, *figure (1)* ***(Eckstein and Marx, 1974).***

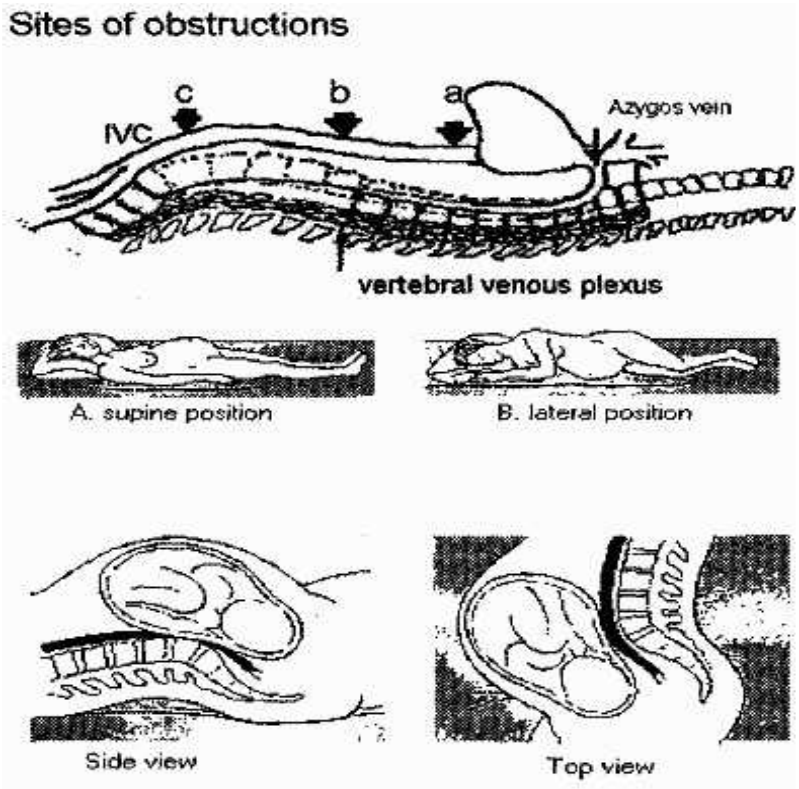

Figure (1): Diagram of the caval venous system and its connections with the vertebral and azygous systems. Commonest sites of compression of the inferior vena cava (IVC) are: (a) suprahepatic in lordotic position; (b) uterus at term; and (c) pressure at pelvic brim in exaggerated lordosis and term pregnancy ***(Bromage, 1990).***

Compensatory mechanisms include increased sympathetic tone and development of collateral routes (paravertebral veins to azygos vein) to improve venous return during obstruction of the vena cava. Caval compression also increases uterine venous back pressure, which further decreases uterine blood flow. If the compensatory mechanisms are enough, they maintain blood pressure and no symptoms appear. This called concealed caval occlusion. If the compensatory mechanisms are not enough (e.g., during general anaesthesia or regional anaesthesia), cardiac output and blood pressure decrease and symptoms as nausea, vomiting, dizziness, anxiety and fetal hypoxia (causing fetal acidosis and bradycardia) appear. This is called revealed caval occlusion ***(Datta, 2011).***

Compression of the aorta is not associated with maternal symptoms in a healthy parturient, but it may be associated with decreased uteroplacenta1 perfusion ***(Marx, 1974).***

Chronic partial caval obstruction in the third trimester predisposes to venous stasis, phlebitis, and oedema of the lower extremities ***(Birnbach and Browne, 2011).***

The changes in blood volume and cardiac output usually have clinical implications for parturients who have concomitant cardiac disease, but they may also have an impact on healthy parturients. Many pregnant patients will complain of symptoms suggestive of cardiovascular disease at term, including

shortness of breath, palpitations, dizziness, oedema, and poor exercise tolerance ***(Cole and St John, 1989).***

Physical examination of the patient may also be abnormal when compared with the prepregnant state, with auscultation commonly revealing a wide loud split first heart sound, third heart sound, and a soft systolic ejection murmur caused by increased blood flow and vasodilatation. A few patients develop small, asymptomatic pericardial effusions ***(Morgan et al., 2013).***

Pregnancy has numerous effects on cardiac evaluation, including changes in the electrocardiogram, e.g., changes in the axis of the heart to the left and nonspecific ST, T, and Q wave changes and benign arrhythmias ***(Lang and Borow, 1991).*** The position of the heart is usually altered by the elevated diaphragm at term gestation resulting in the appearance of an enlarged heart in echocardiogram. Although these minor changes occur in healthy pregnant women at term, symptoms and signs such as chest pain, syncope, severe arrhythmias, systolic murmur more than grade 3, or diastolic murmur suggest severe disease and warrant further investigations. A gradual return to the prepregnancy blood volume occurs at 6-9 weeks postpartum ***(Morgan et al., 2013)*** (Table 1).

Table (1): Cardiovascular changes in pregnancy.

Parameter	Changes	Amount (%)
Heart rate	Increased	20-30
Stroke volume	Increased	20-50
Cardiac output	Increased	30-50
Contractility	Variable	±10
Central venous pressure	Unchanged	-
Pulmonary capillary wedge pressure	Unchanged	-
Systemic vascular resistance	Decreased	Midtrimester 1 0 -15 mm Hg, then rises
Systemic blood pressure	Decreased	30
Pulmonary vascular resistance	Decreased	-
Pulmonary artery pressure	Slightly decreased	-

(Birnbach et al., 2011)

Haematologic System

By term, blood volume increases by up to 45% whereas red cell volume increases by only 30%. This differential increase leads to" physiologic anaemia" of pregnancy with an average haemoglobin and haematocrit of 11.6 g/dL and 35.5% respectively. However, oxygen transport is not impaired by this relative anaemia because the mother's body compensates for it by increased cardiac output, increased partial pressure of arterial oxygen, and rightward shift in the oxyhaemoglobin dissociation curve. The leukocyte count remains 8,000-l0,000/mm3 throughout pregnancy ***(Conklin, 1991).***

A state of hypercoagulability exist in pregnancy, with increased levels of most coagulation factors (Table 2).

Table (2): Coagulation factors in pregnancy.

Factor	Change
II	Unchanged
VII	Increased+++
VIII, IX, X, XII	Increased
XI	Reduced
Fibrinogen	Increased+++
Platelets	Stable

(Birnbach et al., 2011)

Fibrinogen and factor VII are markedly increased, whereas the other factors increase to lesser extent. This increase in coagulation factors has been verified by thromboelastography and is probably a protective adaptation to lessen the risks associated with the acute haemorrhage that occurs at delivery. This hypercoagulable state, however, may lead to thromboembolism, which remains one of the leading causes of maternal mortality ***(Sharma et al., 1997).***

The platelet count remains unchanged throughout most of pregnancy, but it may be slightly reduced in vivo. The platelet count increases in the postpartum period, probably because of activation of haemostasis at the time of delivery. It has been suggested that obstetric management of parturients with stable platelet counts above 50,000 should not be different from that of normal parturients ***(Lottan et al., 2009).***

Respiratory System

To accommodate the increased oxygen demand and requirement for carbon dioxide elimination, pregnancy is associated with an increase in the respiratory minute volume and work of breathing. Oxygen content decreases slightly because of the lesser haematocrit. Oxygen delivery increases because of the increase in cardiac output and a shift to the right in maternal oxyhaemoglobin dissociation curve. Also respiratory changes result in increase in oxygen supply ***(Kamban et al., 1983).***

The most notable change in maternal lung capacities and volumes occurs in functional residual capacity, which decreases 15-20% by term (Table 3 and 4).

Table (3): Maternal respiratory changes at term.

VARIABLE	AVERAGE CHANGE
Volumes and capacities	
Total lung capacity	0 to–5%
Inspiratory lung capacity	+5%
Functional residual capacity	-15–20%
Expiratory reserve volume	–20%
Residual volume	–20%
Vital capacity	No change
Closing volume	No change

(Glosten, 2008)

Table (4): Maternal respiratory changes at term.

Variable	Average changes
Mechanics	
Minute ventilation	+50%
Alveolar ventilation	+70%
Tidal volume	+40%
Respiratory rate	+15%
Dead space	No change
Airway resistance	–36%
Total pulmonary resistance	–50%
Total compliance	–30%
FEV_1	No change
Diffusing capacity	–5%
Blood gases	
Arterial PCO_2	–10 mm Hg
Serum bicarbonate	–4 mEq/L
Arterial pH	No change
Arterial PO_2	+10 mm Hg
Oxygen consumption	+20%

(Glosten, 2008)

Tidal volume increases by 40%, with half of this increase occurring during the first trimester. The transverse and anteroposterior diameter of the chest increases to compensate for the elevation of the diaphragm from the gravid uterus ***(Prowse and Gaensler, 1980).***

Concomitantly, there is an increase in inspiratory reserve volume (IRV), so total lung capacity (TLC) slightly changed. In most parturients, a decreased FRC does not cause problems, but those with pre-existing alterations in closing volume as a result of smoking, obesity, or scoliosis may experience early airway

closure leading to hypoxemia as pregnancy advances ***(Santos et al., 2011)***.

Measurements of closing volume confirm that in the supine position, one-third of parturients have airway closure during normal tidal ventilation and are therefore at risk for developing atelectasis and an increased oxygen alveolar-arterial gradient. Closing capacity (CC), however, remains unchanged. The resulting decrease in the FRC/CC ratio causes faster small-airway closure when lung volume is reduced; thus, parturients can desaturate at a much faster rate than non-pregnant women can ***(Chan and Gin, 1995).***

The Trendelenburg and supine positions also exacerbate the abnormal relationship between closing volume and FRC. Residual volume and FRC quickly return to normal after delivery. Progesterone-induced relaxation of bronchiolar smooth muscle decreases airway resistance, whereas lung compliance remains unchanged. Minute ventilation increases from the beginning of pregnancy to a maximum of 50% above normal at term. Increased minute volume result in a decrease in PCO2 but arterial pH is normalized by a decrease in serum bicarbonate ***(Prowse and Gaensler, 1980).***

Because dead space does not change significantly, alveolar ventilation is increased by 70% at term. After delivery, as blood progesterone levels decline, ventilation returns to normal within 1-3 weeks ***(Santos et al., 2011).*** During labor, particularly in the late first stage and second stage, the pain

from episodic uterine contractions produces corresponding increases in maternal minute ventilation (as much as 300% over that of nonpregnant women) and oxygen consumption. Maternal hypocarbia (arterial carbon dioxide pressure = 20 mm Hg or less), and alkalemia (pH ≥7.55) results. Hypocarbia can lead to hypoventilation between uterine contractions, resulting in intermittent hypoxemia (particularly in obese patients or those who have received parenteral opioids). Epidural analgesia eliminates these pain-induced increases in oxygen consumption and minute ventilation and the accompanying hyperventilation-hypoventilation cycle ***(Hägerdal et al., 1983).***

The increased extracellular fluid and vascular engorgement in respiratory tract and oropharynx may compromise the upper airway, causing oedema of the oropharynx, larynx, and trachea ***(Birnbach and Browne, 2011).***

Gastrointestinal System

There is increase in the risk of aspiration associated with anaesthesia ***(Birnbach and Browne, 2011).*** Regardless of the number of hours after last food intake, all parturients are considered to have a full stomach ***(Datta, 2010).***

The pain of labor, anxiety, belladonna alkaloids may delay gastric emptying and promote emesis. These changes may be caused by the effects of placentally derived gastrin and, which also increases the secretion of gastric acid, and so increases the gastric volume ***(Glosten, 2008)***.