INTRODUCTION

In the largest single series exploring incidental surgeries during pregnancy, trimester-wise breakup is reported to be 42%, 35% and 23% during the first, second and third, respectively ***(Mazze and Källén, 2010).***

Acute abdomen is the term used to describe any serious acute intra-abdominal condition characterised by pain, tenderness, and muscular rigidity, and for which emergency surgery might be considered. In pregnancy, acute abdomen remains one of the most challenging diagnostic and therapeutic dilemmas even today in spite of recent advances in medical sciences ***(Augustin and Majerovic, 2009).***

Appendicitis, ovarian disorders (torsion or neoplasm) and trauma constitute the most common non-obstetric conditions requiring surgery during pregnancy. Obstetric patients presenting for neurosurgery or admitted in intensive care unit for various indications require appropriate changes in the management plans by the anaesthesiologist or intensivist ***(Marulasiddappa et al., 2014)***.

The estimated incidence of pregnant women requiring non-obstetric surgery is around 1-2% ***(Crowhurst, 2002)***.

The normal anatomical and physiological changes occurring in pregnancy lead to confusion in diagnosis and the

general tendency to delay the needed surgical intervention complicates the issues ***(Sivanesaratnam, 2010).***

The situation is even more difficult in places with underdeveloped healthcare systems. The incidence of acute abdomen during pregnancy is reported in literature to be about 1 in 500-635 pregnancies ***(Augustin and Majerovic, 2009).***

One of the major threats that may face pregnant females is trauma it is most prevalent during summer and in industrial (urban) areas ***(Marulasiddappa et al., 2014).***

Common risk (trauma predisposing) factors include environmental conditions such as heavy traffic and bad weather, and/or physical conditions such as intoxication, fatigue, or pregnancy itself. Trauma in pregnancy is currently a leading cause of non-pregnancy related maternal death, and maternal death remains the most common cause of fetal demise ***(Daponte et al., 2014)***.

The most common etiologies of trauma in pregnancy include transportation accidents, falls, violent assaults, and burn injuries ***(Shah and Kilcline, 2014)***.

Aim of the Study

This essay will highlight the important anaesthetic considerations and management of non obstetric emergencies of pregnant females.

PHYSIOLOGIC CHANGES DURING PREGNANCY AND DELIVERY

During pregnancy and the peripartum period, substantial changes in maternal anatomy and physiology occur secondary to: (1) changes in hormone activity, (2) increased maternal metabolic demands and biochemical alterations induced by the feto placental unit, and (3) mechanical effects of an enlarging uterus. These physiologic changes have a significant impact on anesthetic physiology, pharmacology, and management techniques required during pregnancy and present even greater implications for patients with comorbidities ***(Gaiser, 2009).***

Cardiovascular Changes:

Changes in the cardiovascular system occur throughout gestation and include (1) an increase in intravascular volumes and changes in hematology, (2) an increase in cardiac output, (3) a decrease in vascular resistance, and (4) the presence of supine hypotension. The following sections provide additional details of these changes.

Intravascular Volumes and Hematology

Maternal intravascular fluid volume begins to increase in the first trimester secondary to changes in the renin angiotensin-aldosterone system promoting sodium absorption

and water retention. These changes are likely induced by rising progesterone from the gestational sac.

Plasma protein concentrations accordingly decrease with a 25% decrease in albumin and 10% decrease in total protein at term compared with non pregnant levels. Consequently, colloid osmotic pressure decreases from 27 to 22 mm Hg over the time of gestation ***(Wu, udani etal 2011).***

At term, the plasma volume has increased approximately 50% above pre pregnancy values and the red cell volume has increased only approximately 25%. The greater increase in plasma volume creates a physiologic anemia of pregnancy, the hemoglobin (Hb) normally remains at 11 g/dL or greater even at term, and smaller values at any time during pregnancy are concerning for anemia ***(Cheek and Gutsche, 2002).***

Overall oxygen delivery is not reduced by this anemia because of the subsequent increase in cardiac output. The additional intravascular fluid volume of approximately 1000 to 1500 mL at term helps compensate for the estimated blood loss of 300 to 500 mL typically associated with vaginal delivery and the estimated blood loss of 800 to 1000 mL that accompanies a standard cesarean section. After delivery, uterine contraction creates an auto transfusion of blood often in excess of 500 mL that offsets the blood loss from delivery. Leukocytosis is common in pregnancy unrelated to infection. Leukocytosis is defined as a white blood cell (WBC) count greater than 10,000

WBCs/mm3 of blood. In pregnancy, the normal range can extend to 13,000 WBCs/mm3. Neutrophilia increases at term and is exacerbated in labor, often to 34,000 WBCs/mm3. These changes revert to normal over 4 to 5 days after delivery ***(Wintrobe, 2009).***

Pregnancy is a hypercoagulable state with a marked increase in factor I (fibrinogen) and factor VII and lesser increases in other coagulation factors (see Table 1). Factors XI and XIII and antithrombin III are decreased, and factors II and V typically remain unchanged. These changes result in an approximately 20% decrease in prothrombin time (PT) and partial thromboplastin time (PTT) in normal pregnancy. Platelet count may remain normal or slightly decreased (10%) at term as a result of dilution. However, 8% of otherwise healthy women have a platelet count less than 150,000/mm3 ***(Othman et al., 2010).***

In the absence of other hematologic abnormalities, the cause is usually gestational thrombocytopenia, during which time the platelet count does not usually decrease to less than 70,000/mm3. However, this syndrome is not associated with abnormal bleeding. Gestational thrombocytopenia is due to a combination of hemodilution and more rapid platelet turnover and is a diagnosis of exclusion. Other more consequential diagnoses such as idiopathic thrombocytopenic purpura and HELLP syndrome (hemolysis, elevated liver enzyme levels, and low platelet count) must be excluded. Thromboelastography

(TEG) is a test of blood coagulation that examines the end result of coagulation and can provide information about clotting variables, including platelet function and the role of other coagulation factors ***(Wintrobe et al., 2009).***

At term gestation, TEG analysis reflects the hypercoagulable state with decreased time to start of clot formation (R), decreased time to specified clot strength (K), increased rate of clot formation (α), and increased clot strength (MA). Although the timing and degree of change in TEG analysis varies with each parameter, many of the changes begin to occur within the first trimester ***(Karlsson, Sporrong et al, 2012).***

(R) = reaction time; time of latency from start of test to initial fibrin formation (Initiation).

(K) = kinetics; time taken to achieve a certain level of clot strength (Amplification)

(α) = alpha angel; measures the speed at which fibrin build up and cross linking takes place, hence assesses the rate of clot formation (Thrombin burst).

(MA) = maximum amplitude; represents the ultimate strength of the fibrin clot (Overall stability of the clot).

Table (1): Changes in the cardiovascular system during pregnancy

Cardiovascular Parameter	Value at Term Compared With Nonpregnant Value
Intravascular fluid volume	Increased 35%-45%
Plasma volume	Increased 45%-55%
Erythrocyte volume	Increased 20%-30%
Cardiac output	Increased 40%-50%
Stroke volume	Increased 25%-30%
Heart rate	Increased 15%-25%
Systemic vascular resistance	Decreased 20%
Pulmonary vascular resistance	Decreased 35%
Central venous pressure	No change
Pulmonary capillary wedge pressure	No change
Femoral venous pressure	Increased 15%
Coagulation System	
Increased factors	I, VII, VIII, IX, X, XII, and von Willebrand factor
Decreased factors	XI, XIII, antithrombin III, and tPA
Platelets	Decreased 0%-10%
Clinical Studies	
Electrocardiography	Heart rate dependent decrease in PR and QT intervals Small QRS axis shift to right (first TM) or left (third TM) ST depression (1 mm) in left precordial and limb leads Isoelectric T-waves in left precordial and limb leads Small Q-wave and inverted T-wave in lead III
Echocardiography	Heart is displaced anteriorly and leftward Right-sided chambers increase in size by 20% Left-sided chambers increase in size by 10%-12% Left ventricular eccentric hypertrophy Ejection fraction increases Mitral, tricuspid and pulmonic valve annuli increase Aortic annulus not dilated Tricuspid and pulmonic valve regurgitation common Occasional mitral regurgitation (27%) Small insignificant pericardial effusions may be present

(Cheek and Gutsche, 2002)

Cardiac Output

By the end of the first trimester, maternal cardiac output typically increases approximately 35% above pre pregnancy values and continues to increase 40% to 50% above non pregnant values by the end of the second trimester, where it remains throughout the third trimester. This increased cardiac output is secondary to increases in both stroke volume (25% to 30%) and heart rate (15% to 25%). Labor further increases cardiac output, which fluctuates with each uterine contraction. Increases above prelabor values of 10% to 25% occur during the first stage and 40% in the second stage. The largest increase in cardiac output occurs immediately after delivery, when cardiac output can increase by 80% to 100% more than prelabor values ***(Iwasaki, 2002).***

This abrupt increase is secondary to the autotransfusion from the final uterine contraction, reduced vascular capacitance from loss of the intervillous space, and decreased lower extremity venous pressure from release of the aortocaval compression. This large fluctuation in cardiac output presents a unique postpartum risk for patients with cardiac disease, especially those with fixed valvular stenosis and pulmonary hypertension. Cardiac output returns toward prelabor values approximately 24 hours postpartum and decreases substantially toward prepregnant values by 2 weeks postpartum, with complete return to non pregnant levels between 12 and 24 weeks after delivery ***(Iwasaki, 2002).***

Systemic Vascular Resistance

Although cardiac output and plasma volume increase, systemic blood pressure decreases in an uncomplicated pregnancy secondary to a reduction in systemic vascular resistance. Although affected by positioning and parity. systolic, diastolic, and mean blood pressure may all decrease 5% to 20% by 20 weeks gestational age and then gradually increase toward non pregnant values as the pregnancy progresses. Diastolic arterial blood pressure decreases more than systolic arterial blood pressure, central venous and pulmonary capillary wedge pressures do not change during pregnancy, despite the increased plasma volume, because venous capacitance increases ***(Iwasaki, 2002).***

Aortocaval Compression

Aortocaval compression by the gravid uterus as a result of supine positioning is associated with a decrease in systemic blood pressure. Supine hypotension is experienced by nearly 15% of women at term (defined as a decrease in mean arterial pressure > 15 mm Hg with an increase in HR > 20 beats/minute) ***(Kinsella et al., 2012).***

And is often associated with diaphoresis, nausea, vomiting, and changes in mentation. This group of symptoms is known as supine hypotension syndrome. At term, the inferior vena cava is almost completely occluded in the supine position with the return of blood from the lower extremities through the

epidural, azygos, and vertebral veins that become engorged (Fig 1).

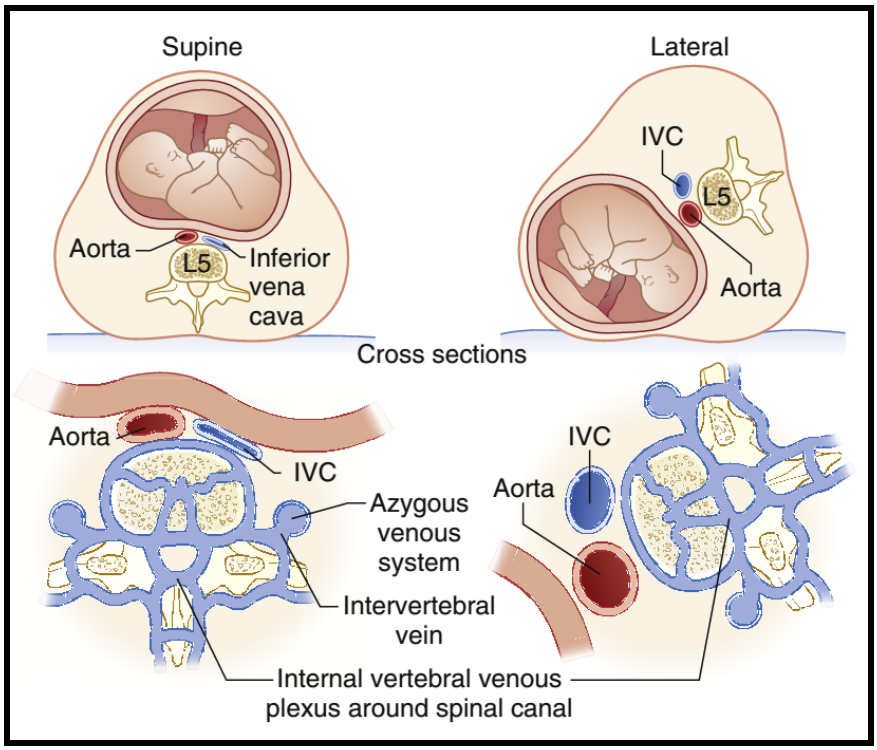

Figure (1): Cross-sectional views of aortocaval compression from the gravid uterus in the supine position with loss of compression in the lateral position ***(Kinsella et al., 2012).***

Also, significant aortoiliac artery compression occurs in 15% to 20% of pregnant women. Vena caval compression in the supine position causes a decrease in both stroke volume and cardiac output of 10% to 20% (see Fig.1, *B*), and may exacerbate venous stasis in the legs and thereby result in ankle edema, varices, and increased risk for venous thrombosis.

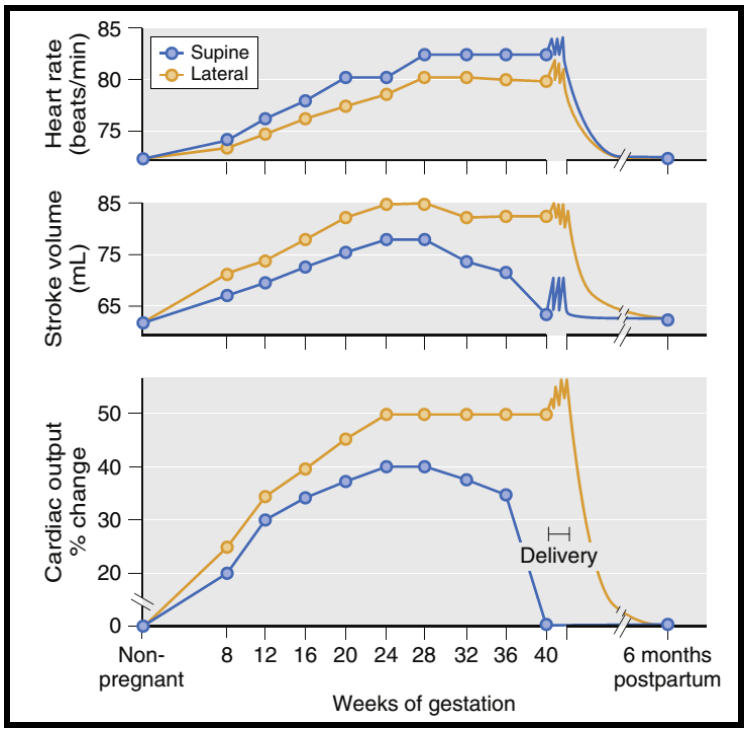

Figure (2) ***(Kinsella et al., 2012).***

Most pregnant women have compensatory adaptations that reduce the supine hypotension despite aortocaval compression. One compensatory response is a reflexive increase in peripheral sympathetic nervous system activity. This increase in sympathetic activity results in increased systemic vascular resistance and permits arterial blood pressure to be maintained despite the reduced cardiac output. Consequently, the reduced sympathetic tone from neuraxial or general anesthetic techniques impairs the compensatory increase in vascular resistance and exacerbates the impact of hypotension from supine positioning. Compression of the lower abdominal aorta by the gravid uterus reduces arterial pressure

in the lower extremities; however, decreases in systemic blood pressure as measured in the maternal arms are not reflective of this change ***(Eckstein et al., 2011).***

Consequently, even without maternal symptoms, uterine and placental blood flow may be substantially decreased from supine aortocaval compression, even with a healthy term pregnancy, prolonged maternal hypotension can significantly decrease uterine blood flow and lead to progressive fetal acidosis ***(Rosen, 2002).***

Therefore, supine positioning is avoided during use of neuraxial techniques for labor analgesia and cesarean deliveries. Reducing the compression of the inferior vena cava and abdominal aorta with left tilt mitigates the degree of hypotension and helps maintain uterine and fetal blood flow. This is accomplished by positioning the patient laterally or by elevating the right hip 10 to 15 cm with a blanket, wedge, or table tilt ***(Othman et al., 2010).***

Increased venous pressure below the level of compression of the inferior vena cava diverts venous blood return from the lower half of the body via the paravertebral venous plexuses to the azygos vein. Flow from the azygos vein enters the superior vena cava to bypass the obstruction and maintain venous return to the heart. Dilation of the epidural veins in pregnancy may make unintentional placement of the epidural catheter more likely. An unintended intravenous bolus

of local anesthetic can have significant Consequences on the cardiovascular and central nervous systems with the possibility of complete hemodynamic collapse, seizures, and death. A small, nontoxic test dose is thus employed to decrease the likelihood of unrealized intravascular placement with initiation of neuraxial blockade ***(Rosen, 2002).***

The cardiovascular changes of a normal pregnancy are significant. In cardiac auscultation an accentuated first heart sound (S1) can be heard, with an increased splitting noted from dissociated closure of the tricuspid and mitral valves. A third heart sound (S3) is often heard in the final trimester, and a fourth heart sound (S4) can also be heard in a few pregnant patients as a result of increased volume and turbulent flow. Neither the S3 nor S4 has clinical significance. In addition, a systolic ejection murmur is typically heard over the left sternal border and is secondary to mild regurgitation at the tricuspid valve from the annular dilation associated with the increased cardiac volume. Table 1 details the effects of pregnancy on the electrocardiogram and echocardiography. Women who present with chest pain, syncope, high-grade flow murmurs, and clinically significant shortness of breath or severe arrhythmias should undergo appropriate diagnostic investigation and referral ***(Othman et al., 2010).***

Respiratory system changes

Pregnancy results in significant alterations in (1) the upper airway, (2) lung volumes and minute ventilation, and (3) O2 consumption and metabolic rate (Table 2).

Table (2): Changes in the respiratory system at term

Pulmonary Parameter	Value Near Term Compared With Nonpregnant Value (%)
Minute ventilation	Increased 45-50
Tidal volume	Increased 40-45
Respiratory rate	Increased 0 to 15
Lung Capacities and Associated Volumes	
Total lung capacity	Decreased 0-5
Vital capacity	No change
Inspiratory capacity	Increased 5-15
Inspiratory reserve volume	Increased 0-5
Tidal volume	Increased 40-45
Functional residual capacity	Decreased 20
Expiratory reserve volume	Decreased 20-25
Residual volume	Decreased 15-20
Oxygen Consumption	
Term	Increased 20
Labor (first stage)	Increased 40 above prelabor value
Labor (second stage)	Increased 75 above prelabor value
Respiratory Measures	
FEV_1	No change
FEV_1/FVC	No change
Closing capacity	No change

(Cheek and Gutsche, 2002)