Introduction

Trauma is a significant cause of morbidity and mortality in women of childbearing age. Injury during pregnancy occurs in up to ٨% of pregnant women and is the most frequent cause of non-obstetric maternal and fetal mortality.

Anatomical and physiological changes during pregnancy influence patterns of injury and the patho-physiological responses of the patient to the injury.

Trauma from road traffic, falls and domestic violence are the most common causes of blunt trauma.

Other modes of trauma includes penetrating trauma, head injuries, thoracic trauma and burns.

Optimal management of the pregnant trauma patient requires a multidisciplinary approach. The anaesthetist and critical care physician play an important role in the fetomaternal care, from initial assessment, resuscitation and intraoperative management. Primary goals are resuscitation of the mother and maintenance of uteroplacental perfusion and fetal oxygenation by the avoidance of hypoxia,

hypotension, hypocapnia, acidosis and hypothermia. Recognizing and understanding the mechanism of injury, the factors that may predict fetal outcome, and the pathophysiological changes that can result from trauma, will allow early identification and treatment of fetomaternal injury. This in turn should improve morbidity and mortality.

Anatomical and Physiological Changes during Pregnancy

During pregnancy, changes occur in the maternal anatomy and physiology which are of such magnitude that they would cause alarm if they presented in other situations. Although many of these changes are beneficial and help women survive the stresses of child birth, e.g. increased blood volume enables tolerance of blood loss at delivery, on the other -hand, other changes such as decreased functional residual capacity have no obvious advantage and indeed place the parturient at increased risk of death ***(Ostheimer, ٢٠٠٠).***

It is not always possible to establish the etiology of changes in a particular physiological variable although those beginning in the first trimester of pregnancy are usually due to hormonal effect***;*** Progesterone, estrogen, human chorionic gonadotrophine (HCG) and prostaglandins are all present in excessive amount during pregnancy. As the uterus increases in size the mechanical effect of expanding intra-abdominal mass explains other physiological changes. The fetus, uterus and placenta present an increased metabolic demand to

the mother, although this does not adequately account for the major respiratory and cardio-vascular changes. Finally the utero-placental circulation acts as a low pressure arteriovenous shunt with the expected hemodynamic consequen-ces ***(Ostheimer, ٢٠٠٠).***

The anesthesiologist caring for the pregnant patient must understand these physiological changes to provide safe analgesia and anesthesia to mother and safe delivery of the fetus ***(Ostheimer, ٢٠٠٠).***

The respiratory System:

Upper airway: -

Anatomical changes which occur may render endotracheal intubation exceedingly difficult. The short neck and large breasts of an obese parturient can present an obstacle to insertion of the laryngoscope and visualization of the larynx ***(Mackenzi, ١٩٨٨).***

During pregnancy, capillary engorgement of mucosa occurs throughout the respiratory tract, potentially causing edema in the nasopharynx, larynx and trachea. Therefore manipulation of the upper airway requires extreme care. Suction of oropharynx, insertion of airway, and laryngoscopy may cause further edema and bleeding. Because of the area of vocal cords

may be swollen, a small cuffed endotracheal tube (٦.٥ - ٧.٠ mm) is recommended. Repeated attempts of laryngoscopy during management of difficult airway must be minimized to prevent obstructing airway edema ***(lewin etal, ٢٠٠٠).***

Shape of chest:

There are three important changes in the Configuration of the thorax during pregnancy:

١ An increase in the circumference of the lower chest wall (with increases in anteroposterior and the transverse diameters).

٢ Elevation of the diaphragm (a cephalad displacement of approximately ٤ cm to ٥ cm).

٣ A ٥٠% widening of the costal angle.

These changes peak around the ٣٧th week of Pregnancy and normalize within ٦ months after delivery ***(Pereira and Krieger, ٢٠٠٤).***

The major changes in pulmonary function are a progressive decline in expiratory reserve volume and a decrement in residual volume of ٧% to ٢٢%, which result in a reduction of the functional residual capacity (FRC) by ١٠% to ٢٥% close to term ***(Lapinsky et al,***

١٩٩٥).

These changes are secondary to the enlargement of the abdominal contents with upward displacement of the diaphragm. The reduction in FRC causes closure of small airways at the lung bases during normal tidal breathing which results in ventilation-perfusion mismatch and reduced gas exchange. Inspiratory capacity increases slightly. Total lung capacity decreases only minimally as the uterus enlarges. Overall, no significant change in peak flow rates, forced vital capacity, or forced expiratory volume in the first second (FEVI) is observed, the total pulmonary resistance is reduced by ٥٠% as a result of a decrease in airway resistance. Lung compliance does not change, but total respiratory compliance is decreased at term as a result of a reduction in chest wall compliance. Despite the significant increase in intra-abdominal pressure that is due to the enlarging uterus, the maximal inspiratory and expiratory pressures, as well as maximum transdiaphragmatic pressure do not change significantly ***(Pereira and Krieger, ٢٠٠٤)***.

Oxygen uptake increases about ٢٠ percent during pregnancy, owing to increased maternal metabolism and work of breathing and fetal metabolism ***(Clapp, ١٩٨٩)***.

Minute ventilation at term is increased about ٥٠ percent, mostly as a result of increased tidal volume, with only a slight increase in respiratory rate. As a result of increased alveolar ventilation at term, arterial carbon dioxide pressure usually decreases to about ٣٢ mm Hg, but arterial pH remains normal because of a compensatory decrease in serum bicarbonate (from ٢٦ to ٢٢ mEq/L). Partial pressure of arterial oxygen is slightly increased from the increased minute ventilation. The oxyhemoglobin dissociation curve shifts to the right during normal pregnancy (increased P٥٠) allowing a greater volume of oxygen to be unloaded to the tissues (fetus) at a given arterial oxygen pressure ***(Glosten, ٢٠٠٠).***

During the first trimester, the PaO_2 averages ١٠٥ mmHg to ١٠٧ mm Hg while sitting but decreases ٥ mmHg by the third trimester. Moving from the sitting to the supine position induces an average of ١٣ mm Hg decline in PaO_2. The alveolar to arterial oxygen tension difference while sitting increases from ١٤ mm Hg early in pregnancy to ٢٠ mm Hg at term ***(Pereira and Krieger, ٢٠٠٤)***.

Respiratory mechanics

The chest expands in the anteroposterior and

transverse diameters to compensate for the elevation of the diaphragm caused by upward pressure from the uterus ***(Bevan et al., ١٩٩٤)***.

The decrease in the functional residual capacity (FRC) is important because of its relationship to the closing volume (CV). When FRC is less than CV, the terminal airways close and alveoli become perfused but not ventilated, thus the deoxygenated blood is shunted and arterial hypoxemia occurs. This situation only occurs in about ٢٥% of parturients, most of whom are smokers and is present onlv when they are in the supine position. Closing volume increases with such factors as advanced age, smoking and lung diseases whereas FRC decreases with certain body position (lithotomy, Trendelenburg), obesity and general anesthesia. Thus, the likelihood of airway closure is substantially increased when any of these conditions is superimposed on pregnancy ***(Russell, ١٩٩١)***.

Gas exchange

Oxygen consumption increases by ١٠-٢٠% during pregnancy and further as much as ١٠٠% over normal during labor. This occurs in response to increased demand by the growing fetus, placenta and uterus and to

increased cardiac and respiratory work particularly during labor **(*Russell et al.,* ١٩٩٤).**

The oxy hemoglobin dissociation curve shifts to the right during normal pregnancy (increased P٥٠) allowing a greater volume of oxygen to be unloaded to the tissues (fetus) at a given arterial oxygen pressure .

Arterial blood gases reflect chronic hyperventilation with PaCO٢ level in the range of ٣٢-٣٤ mm Hg. By ١٢ weeks of gestation, this respiratory alkalosis is partially compensated for by renal excretion of bicarbonate, pH is slightly alkaline (٧.٤٤) and bicarbonate base excess and buffer base are all decreased. Serum bicarbonate decreases from ٢٦ to ٢٢mEq/L ***(Shanker et al., ١٩٩٧).***

Metabolic acidosis may develop during prolonged labor as lactic acid and pyruvate accumulate due to of lower than usual buffering capacity ***(Templeton and kelman, ١٩٩٩).***

During labor, particularly in the ١st stage and second stage, the pain from episodic uterine contractions produces increases in the maternal minute ventilation much as ٣٠٠% over that of non-pregnant women and oxygen consumption increases ٦٠% above prelabor

level. Maternal hypocarbia (arterial carbon dioxide pressure ٢٠ mm Hg or less) and alkalemia (pH ٧.٥٥) result. Hypocarbia (particularly in obese patients or those who have received parenteral opioids) can lead to hypoventilation between uterine contractions resulting in intermittent hypoxemia ***(Lewin etal, ٢٠٠٠).***

Cardiovascular Changes

The first trimester is a transition period between the nonpregnant and pregnant states during which changes in the cardiovascular system are clear ***(Chapman et al., ١٩٩٨)***.

These changes include changes in cardiac output, heart rate, ECG and blood pressure.

Cardiac output:

Cardiac output increase starts from ٥-٦ weeks gestation ***(Chapman et al., ١٩٩٨)***.

It reaches its maximum level at approximately ٣٢ weeks, after which there is only a slight increase until the labor and the postpartum period. Approximately ٥٠% of the increase in cardiac output occurs by the ٨th week of pregnancy. Although this increase in cardiac output is due to an increase in both stroke volume and heart rate, the more important factor is stroke volume, which increases by ٢٠% to ٥٠% from non pregnant values ***(Glosten, ٢٠٠٠)***.

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, ٣٠٠ to ٥٠٠ mL enters the maternal system. The autotransfusion can increase COP and central blood volume by ١٠% to ٢٥% above nonpregnant values. The greatest increase in COP occurs immediately after delivery when COP can be up to ٨٠% above nonpregnant values. This again is attributed to autotransfusion from the contracted uterus. Prepregnancy COP levels are generally regained several weeks postpartum, and the normal nonpregnant blood volume is reached by about ٢ weeks postpartum ***(Glosten, ١٩٩٨).***

Heart rate:

Increases in heart rate contribute primarily to early changes in cardiac output, as most of the ١٠-١٥bpm pregnancy-induced increase in heart rate occurs during the first trimester.

Some studies have reported a continuous increase in heart rate throughout the first trimester **(Robson et al., ١٩٩١**), whereas others have documented substantial increases by ٦ - ٨ weeks gestation with no significant changes thereafter ***(Clapp et al., ١٩٨٨)***.

ECG changes:

There are both size and position changes which can lead to changes in ECG appearance. 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 displace-ment of the diaphragm by the enlarging uterus causes the heart to shift to the left and interiorly, so that the apex beat is moved outwards and upwards. These changes lead to common ECG findings of left axis deviation, sagging ST segments and frequently inversion or flattening of the T-wave in lead III ***(Ciliberto and Marx, ١٩٩٨)***.

Blood pressure:

The placenta modulates the production and release of specific hormones, including progester-one, estrogens (estradiol, estriol, and estrone), and human chorionic gonadotropnin ***(Speroff et al., ١٩٩٩)***.

Estrogen increases maternal cardiac dimensions ***(Hart et al, ١٩٨٥)***.

There is also nitric oxide production which may contribute to reduced peripheral vascular resistance (PVR) and enhanced endothelial function. HCG may lower PVR by attenuating vascular responsiveness to

angiotensin II in mesenteric and uterine resistance arteries ***(Hermsteiner et al, ٢٠٠٢)***.

Reduced PVR contributes to a slight decrease in mean arterial pressure, which is evident as early as ٦ weeks gestation ***(Spaanderman et al, ٢٠٠٠)***.

Systemic arterial pressure is never increased during normal gestation. In fact, by midpregnancy, a slight decrease in diastolic pressure can be recognized. Pulmonary arterial pressure also maintains a constant level. However, vascular tone is more dependent upon sympathetic control than in the non pregnant state, so that hypotension develops more readily and more markedly consequent to sympathetic blockade following spinal or extradural anesthesia. Central venous and brachial venous pressures remain unchanged during pregnancy, but femoral venous pressure is progressively increased due to mechanical factors ***(Ciliberto and Marx, ١٩٩٨)***.

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 ***(Glosten, ٢٠٠٠)***.

Aortocaval Compression:

From mid-pregnancy, the enlarged uterus compresses both the inferior vena cava and the abdominal aorta when the patient lies supine. Obstruction of the inferior vena cava reduces venous return to the heart leading to a fall in cardiac output by as much as ٢٤% towards term ***(Ciliberto and Marx, ١٩٩٨)***.

This is called the supine hypotension syndrome, which is characterized by hypotension associated with pallor, sweating, or nausea and vomiting. Turning the patient on her side typically restores venous return from the lower body and corrects the hypotension in such instances ***(Morgan et al., ٢٠٠٦)***.

In the unanesthetised state, most women are capable of compensating for the resultant decrease in stroke volume by increasing systemic vascular resistance and heart rate. There are also alternative venous pathways; the paravertebral and azygos systems. During anesthesia, however, these compensatory mechanisms