INTRODUCTION

Trauma complicates approximately 6-7% of all pregnancies which may be associated with maternal and fetal morbidity and mortality ***(Shah, 2003).*** The most common etiology for trauma in pregnancy are motor vehicle accidents, falls, violent assaults, and burn injuries. Management of the pregnant trauma victim requires a multidisciplinary approach in order to optimize outcomes for mother and fetus ***(Chulu, 2003).***

Anatomic, physiologic, changes, and existence of two lives should be considered while assessment, resuscitation, and stabilization of mother takes priority. Radiologic procedures should be used as indicated, minimizing fetal exposure to radiation.

The effective management of pregnant trauma victims (PTVs) requires the clinician to consider and understand the unique changes in anatomy and physiology of pregnancy. During the first trimester of pregnancy, the bony pelvis protects the uterus and the fetus from direct injury. The uterus enlarges into the peritoneal cavity after the 12^{th} week of pregnancy, protecting other maternal abdominal organs, although it now becomes more susceptible to injury. The pathophysiology and location of maternal injuries in pregnancy may significantly different from those that commonly occur in the nonpregnant women ***(Rudra, 2007).***

Anesthesiologists in practice at designated trauma centers are involved in the care of trauma patients beginning with airway maintenance and resuscitation in the emergency department (ED), proceeding through the operating room or to the intensive care unit (ICU). Anesthesia for trauma patient differs as most urgent cases may occur during off-hours, when the most experienced OR and anesthesia personnel may not be available. Patient information may be limited, and allergies, genetic abnormalities, and previous surgeries may create unexpected crises. Simple operations may become complicated, requiring special surgical and anesthesia equipment at short notice. Patients often have multiple injuries requiring complex procedures, which need to be prioritized during management. Occult injuries such as tension pneumothorax, can manifest unexpectedly. Successful management of these patients requires a good understanding of pathophysiology, supplemented by adequate preparation, and the ability to react quickly to the changing circumstances.

Equipment to facilitate difficult intubation like gum elastic bougie, intubating stylet, esophageal combitube, and laryngeal mask airway (LMA) should be readily available whenever emergency airway management is performed. The most recent advanced trauma life support (ATLS) guidelines suggest that practitioners providing emergency airway management should proceed with the method of intubation with which they are most proficient ***(KuczKowski, 2004)***.

The experienced anesthesiologist can play an important role along with the multidisciplinary team, regarding prioritization of different surgical procedures on the trauma victim. Aggressive resuscitation of the mother should take priority over concerns for fetal well being. The secondary effects of trauma result in an increase in the morbidity and mortality of pregnant trauma victims, an anesthesiologist can play a major role in preventing these secondary effects ***(Vanden, 2010).***

AIM OF THE WORK

The aim of the work to discuss Anesthetic Management of abdominal Trauma with Pregnancy which is the most common type of trauma in pregnancy.

ANATOMICAL AND PHYSIOLOGICAL CHANGES DURING PREGNANCY

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 consequences ***(Ostheimer, 2000).***

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, 2000).***

A) The respiratory System:

There are many profound changes with the respiratory system in women during pregnancy week by week.

The adaptations are controlled primarily by progesterone and take place in the early stages of pregnancy starting soon after the missed period.

Upper airway:

Histologic examination of the upper respiratory mucosa during pregnancy reveals hyperemia, glandular hyperactivity, increased phagocytic activity, and increased mucopoly-saccharide content ***(Toppozada, 1982)***. Pregnant women often experience nasal stuffiness and epistaxis, possibly as a result of these alterations. 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 ***(Mackenzie, 2013).***

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 (6.5-7.0mm) is recommended. Repeated attempts of laryngoscopy during management of difficult airway must be minimized to prevent obstructing airway edema ***(Lewin et al., 2000).***

Shape of chest:

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

1- An increase in the circumference of the lower chest wall (with increases in antero-posterior and the transverse diameters).

2- Elevation of the diaphragm (a cephalad displacement of approximately 4 cm to 5 cm).

3- A 50% widening of the costal angle.

These changes peak around the 37th week of Pregnancy and normalize within 6 months after delivery ***(Pereira and Krieger, 2004).***

The major changes in pulmonary function are progressive decline in expiratory reserve volume and a decrement in residual volume from 70% to 22%, which result in a reduction of the functional residual capacity (FRC) by 10% to 25% close to term ***(Lapinsky et al., 2000).***

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 50% 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, 2004).***

Oxygen uptake increases about 20 % during pregnancy, owing to increased maternal metabolism and work of breathing and fetal metabolism. Minute ventilation at term is increased about 50 %, 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 32 mmHg, but arterial pH remains normal because of a compensatory decrease in serum bicarbonate (from 26 to 22 mEq/L). Partial pressure of arterial oxygen is slightly increased from the increased minute ventilation. The oxy-hemoglobin dissociation curve shifts to the right during normal pregnancy (increased P50) allowing a greater volume of oxygen to be unloaded to the tissues (fetus) at a given arterial oxygen pressure ***(Glosten, 2000)***.

During the first trimester, the PaO_2 averages 105mmHg to 107 mmHg while sitting but decreases 5 mmHg by the third trimester. Moving from the sitting to the supine position induces an average of 13 mmHg decline in PaO_2. The alveolar to arterial oxygen tension difference while sitting increases from 14 mmHg early in pregnancy to 20 mmHg at term ***(Pereira and Krieger, 2004).***

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., 1994).***

The decrease in (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 25% of parturients, most of whom are smokers and is present only when they are in the supine position. (CV) 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 & Chambers, 1991).***

Gas exchange:

Oxygen consumption increases by 10-20% during pregnancy and further as much as 100% 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., 1994).***

Arterial blood gases reflect chronic hyperventilation with $PaCO_2$ level in the range of 32-34 mm Hg. By 12 weeks of gestation, this respiratory alkalosis is partially compensated for by renal excretion of bicarbonate, pH is slightly alkaline (7.44) and bicarbonate base excess and buffer base are all decreased. Serum bicarbonate decreases from 26 to 22mEq/L ***(Shanker et al., 1997).***

Metabolic acidosis may develop during prolonged labor as lactic acid and pyruvate accumulate due to of lower than usual buffering capacity ***(Templeton and Kelman, 1999).***

During labor, particularly in the 1st stage and second stage, the pain from episodic uterine contractions produces increases in the maternal minute ventilation much as 300% over that of non- pregnant women and oxygen consumption increases 60% above prelabor level. Maternal hypocarbia (arterial carbon dioxide pressure 20mm Hg or less) and alkalemia (pH 7.55) 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 et al., 2000).***

Preoxygenation with 100% oxygen for 3 minutes; induce anesthesia with a fast-acting IV agent, followed by a muscle relaxant, with cricoid pressure and intubation, and inflate the cuff immediately is indicated for pregnant female during labour ***(Faure, 2006).***

B) Cardiovascular Changes

Pregnancy is associated with vasodilation of the systemic vasculature and the maternal kidneys. The systemic vasodilation of pregnancy occurs as early as at 5 weeks and therefore precedes full placentation and the complete development of the uteroplacental circulation ***(Chapman, 1998).*** In the first trimester, there is a substantial decrease in peripheral vascular resistance, which decreases to a nadir during the middle of the second trimester with a subsequent plateau or slight increase for the remainder of the pregnancy ***(Mahendru, 2014).*** The decrease is ≈35% to 40% of baseline. Systemic vascular resistance increases to near-prepregnancy levels postpartum, and by 2 weeks after delivery, maternal hemodynamics have largely returned to nonpregnant levels. Increased vascular distensibility, or compliance, has been observed in normal human pregnancy starting in the first trimester. Systemic vascular resistance increases to near-prepregnancy levels postpartum. Vasodilation of the kidneys

results in a 50% increase in renal plasma flow and glomerular filtration rates by the end of the first trimester. This results in decreases in serum creatinine, urea, and uric acid values ***(Cheung, 2013).***

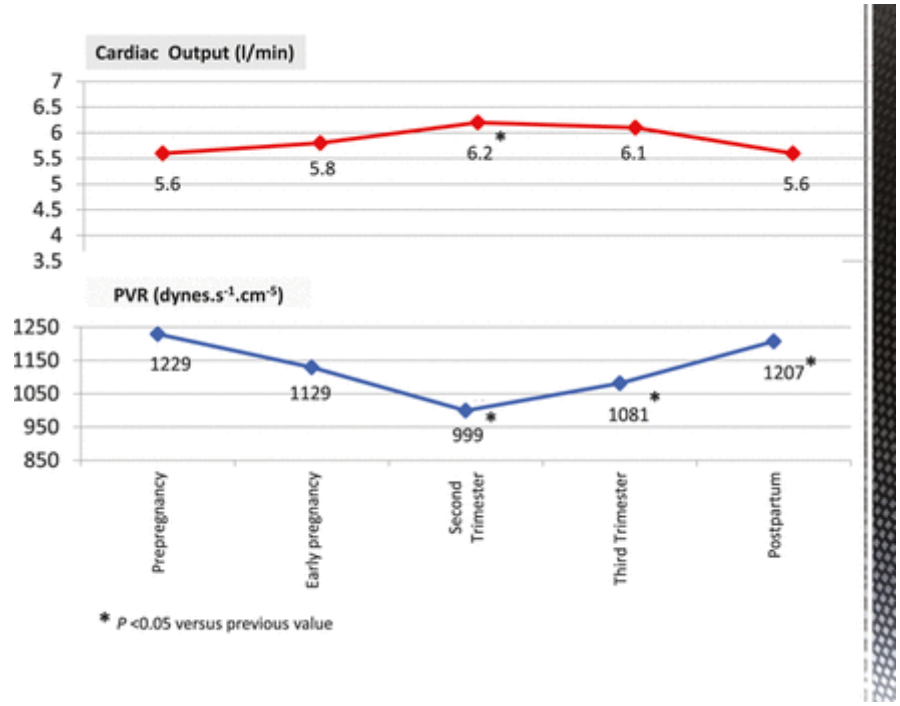

Figure (1): Detailed hemodynamics were longitudinally studied in 54 women with normal pregnancies preconception and then at 6, 23, and 33 weeks during pregnancy and 16 weeks postpartum. Radial artery waveforms were obtained with a high-fidelity micromanometer; a central waveform was generated with a validated central transfer function; and mean arterial pressure (MAP) was determined with integrated software. Cardiac output (CO) was assessed with a noninvasive, validated inert gas rebreathing technique, and peripheral vascular resistance (PVR) was calculated from the formula PVR=MAP (mmHg)×80/CO (L/min). The reciprocal relationship of CO and PVR in pregnancy is demonstrated. CO increases from preconception to the second trimester and then falls to the preconception level 16 weeks postpartum. The PVR fell significantly by the second trimester (a 19% fall), followed by an increase in the third trimester and a return to preconception levels by 16 weeks postpartum. Figure created from the data of Mahendru et al***. (Cheung, 2014).***

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., 1998).***

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

Cardiac output (COP):

COP increase starts from 5-6 weeks gestation ***(Chapman et al., 1998).***

It reaches its maximum level at approximately 32 weeks, after which there is only a slight increase until the labor and the postpartum period. Approximately 50% of the increase in COP occurs by the 8th week of pregnancy. Although this increase in cardiac output is due to an increase in both stroke volume and heart rate, the most important factor is stroke volume, which increases by 20% to 50% from non pregnant values ***(Glosten, 2000).***

During labor, COP is further increased both from the sympathetic stimulation of pain, as well as the episodic autotransfusion of blood into central circulation from the contracting uterus. It is estimated that with each uterine contraction, 300 to 500 mL enters the maternal system. The autotransfusion can increase COP and central blood volume by 10% to 25% above nonpregnant values. 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, and the normal nonpregnant blood volume is reached by about 2 weeks postpartum ***(Glosten, 1998).***

Heart rate:

Increases in heart rate contribute primarily to early changes in cardiac output, as most of the 10-15bpm 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, whereas others have documented substantial increases by 6 - 8 weeks gestation with no significant changes thereafter ***(Clapp et al., 2000).***

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 displacement 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, 1998).***

Blood pressure:

The placenta modulates the production and release of specific hormones, including progesterone, estrogens (estradiol, estriol, and estrone), and HCG ***(Speroff et al., 1999).***

Estrogen increases maternal cardiac dimensions. 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., 2002).***

Reduced PVR contributes to a slight decrease in mean arterial pressure, which is evident as early as 6 weeks gestation ***(Spaanderman et al., 2000).***

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,