Chapter 11: Trauma in Pregnancy

Objectives:

Upon completion of this topic, the participant will be able to:

A. Discuss anatomic and physiologic alterations of pregnancy that affect initial management of the injured pregnant patient.

B. Discuss mechanisms of injury unique to the pregnant patient and fetus.

C. Outline the priorities and method of evaluating the injured pregnant patient.

D. Outline indications for surgical intervention unique to the injured pregnant patient.

E. Recognize the possibility of isoimmunization and its early treatment in Rh-negative mothers following trauma.

F. Recognize indications for obstetric intervention and consultation in the injured pregnant patient.
I. Introduction

Pregnancy causes major physiologic changes and altered anatomic relationships involving nearly every organ system of the body. These changes of structure and function may influence the evaluation of the traumatized pregnant patient by altering the signs and symptoms of injury, as well as the results of diagnostic laboratory tests. Pregnancy may also affect the patterns of injury or severity of injury. Treatment priorities for an injured pregnant patient remain the same as for the non-pregnant patient. However, resuscitation and stabilization should be modified to accommodate the unique anatomic and physiologic changes of pregnancy. The physician attending a pregnant trauma victim must remember that he does, in fact, have two patients. A thorough understanding of this special relationship between a pregnant patient and her fetus is essential if the best interests of both are to be served. The best treatment for the fetus is the provision of optimum treatment for the mother. Monitoring and evaluation techniques should allow not only assessment of the mother but also of the fetus. The use of roentgenograms, if indicated during critical management, should not be withheld because of the pregnancy. A qualified surgeon and obstetrician should be consulted early in the evaluation of the pregnant trauma patient.

II. Anatomic and Physiologic Alterations of Pregnancy

A. Anatomic

The uterus remains an intrapelvic organ until the 12th week of gestation, when it begins to rise out of the pelvis and encroach on the peritoneal cavity. By 20 weeks, the uterus is at the umbilicus. At 36 weeks, it reaches its maximal supraumbilical extent - the costal margin. During the last two to eight weeks of gestation, the fetus slowly descends as the fetal head engages the pelvis. As the uterus enlarges, it reduces the confines of the intraperitoneal space, restricting the intestines to the upper abdomen. Likewise, the intrauterine environment gradually changes from very protective to very vulnerable.

During the first trimester, the uterus is a thick-walled structure of limited size, confined within the safety of the bony pelvis. During the second trimester, the uterus leaves its protected intrapelvic location, but the small fetus remains mobile and cushioned by a relatively generous amount of amniotic fluid. The amniotic fluid itself could be a source of amniotic fluid embolism and disseminated intravascular coagulation following trauma. By the third trimester, the uterus is large and thin walled. The head is usually fixed in the pelvis with the remainder of the fetus exposed above the pelvic brim. The placenta reaches its maximum size by 36 to 38 weeks and is devoid of elastic tissue. This lack of placental elastic tissue predisposes to shear forces between the placenta and uterine wall leading to such complications as abruptio placentae. The placental vasculature is maximally dilated throughout gestation, yet it is exquisitely sensitive to catecholamine stimulation. Direct trauma to the placenta or uterus may reverse the normal protective hemostasis of pregnancy by releasing high concentrations of placental thromboplastin or plasminogen activator from the myometrium. All of these changes make the uterus and its contents more susceptible to injury, including penetration, rupture, abruptio placentae, and premature rupture of membranes.
B. Hemodynamic

1. Cardiac output

After the 10th week of pregnancy, cardiac output is increased by 1.0 to 1.5 liters per minute. This increased output is greatly influenced by the maternal position as term nears. Vena cava compression in the supine position may decrease cardiac output by 30% to 40%. As maternal blood volume decreases due to trauma, placental blood flow is preferentially reduced.

2. Heart rate

Heart rate increases throughout pregnancy. During the third trimester, it reaches a rate of 15 to 20 beats per minute more than in the nonpregnant state. This change in heart rate must be considered in interpreting the tachycardic response to hypovolemia.

3. Blood pressure

Pregnancy results in a 5- to 15-mm Hg fall in systolic and diastolic pressures during the second trimester. Blood pressure returns to near-normal levels at term. Some women may exhibit profound hypotension (supine hypotensive syndrome) when placed in the supine position. This condition is relieved by turning the patient to the left lateral decubitus position.

4. Venous pressure

The resting central venous pressure (CVP) is variable with pregnancy, but the response to volume is the same as in the nonpregnant state. Venous hypertension in the lower extremities is normal during the third trimester.

5. Electrocardiographic changes

The axis may shift leftward by approximately 15 degrees. Flattened or inverted T waves in leads III, AVF, and the precordial leads may be normal. Ectopic beats are increased during pregnancy.

C. Blood Volume and Composition

1. Volume

At 34 weeks' gestation, plasma volume increases by 40% to 50%. A smaller increase in red blood cells (RBC) volume occurs, resulting in a decreased hematocrit (physiologic anemia of pregnancy). In late pregnancy, a hematocrit of 31% to 35% is normal. Blood volume overall increases by 48%. With hemorrhage, otherwise healthy pregnant women may lose 30% to 35% of their blood volume before exhibiting symptoms.
2. Composition

The white blood cell (WBC) count increases during pregnancy to a high of 20,000/mm³. Levels of serum fibrinogen and many clotting factors are elevated. Prothrombin and partial thromboplastin times may be shortened but bleeding and clotting times are unchanged. The serum albumin level falls to 2.2 to 2.8 g/dL during pregnancy, causing a drop in serum protein levels by approximately 1.0 g/dL. Serum osmolarity remains at about 280 mOsm/L throughout pregnancy.

D. Respiratory

Minute ventilation increases primarily as a result of an increase in tidal volume. This is attributed to an increased level of progesterone - a known respiratory stimulant. Hypocapnea (PaCO₂ of 30 mm Hg) is therefore common in late pregnancy. Although the forced vital capacity (FVC) fluctuates slightly during pregnancy, it is largely maintained throughout pregnancy due to equal and opposite changes in inspiratory capacity (which increases) and residual volume (which decreases). Anatomic alterations in the thoracic cavity appear to account for the decreased residual volume that is associated with diaphragmatic elevation with increased lung markings and prominence of the pulmonary vessels seen on chest roentgenogram.

The FEV₁/FVC does not change significantly during pregnancy, suggesting no obstruction to air flow.

Oxygen consumption is usually increased during pregnancy so that maintenance of adequate arterial oxygenation is particularly important in the resuscitation of the injured pregnant patient.

E. Gastrointestinal

Gastric emptying is greatly prolonged during pregnancy, and the physician should assume that the stomach is full. Therefore, early nasogastric tube decompression is particularly important in order to avoid aspiration of gastric contents. The intestines are relocated to the upper part of the abdomen and may be shielded by the uterus. Position of the patient's spleen and liver are essentially unchanged by pregnancy.

F. Urinary

The glomerular filtration rate and the renal plasma blood flow increase during pregnancy. Levels of creatinine and serum urea nitrogen (BUN) fall to approximately one half of normal pregnancy levels. Glycosuria is common during pregnancy. Excretory urography reveals a physiologic dilatation of the renal calyces, pelves, and ureters outside of the pelvis.

G. Endocrine

The pituitary gland gets 30% to 50% heavier during pregnancy. Shock may cause necrosis of the anterior pituitary, resulting in pituitary insufficiency.
H. Musculoskeletal

The symphysis pubis widens by the seventh month (4 to 8 mm). The sacroiliac-joint spaces also increase. These factors must be considered in interpreting roentgenograms of the pelvis.

I. Neurologic

Eclampsia is a complication of late pregnancy that may mimic head injury. Eclampsia should be considered if seizures occur with or without hypertension, especially if hyperreflexia is present.

III. Mechanisms of Injury

Mechanisms of injury are similar to those in the nonpregnant patient. However, certain differences must be recognized in the pregnant patient.

A. Penetrating Injury

As the gravid uterus increases in size, the remainder of the viscera is relatively protected from penetrating injury, while the likelihood of uterine injury increases. The dense uterine musculature can absorb a great amount of energy from penetrating missiles and decreases the missile velocity and transfer of energy to other viscera. Also, the amniotic fluid and conceptus contribute to slowing of the penetrating missile. The resulting low incidence of associated maternal visceral injuries accounts for the generally excellent maternal outcome in penetrating wounds of the gravid uterus.

B. Blunt Injury

The amniotic fluid acts as a buffer to direct fetal injury from blunt trauma. Direct injuries may occur when the abdominal wall strikes an object such as the dashboard or steering wheel or is struck by a blunt instrument. Indirect injury of the fetus may occur from rapid compression, deceleration, contra-coup effect, or a shearing force.

Seat belts decrease maternal injury and death by preventing ejection. However, the type of restraint system affects the frequency of uterine rupture and fetal death. The use of a lap belt alone allows forward flexion and uterine compression with possible uterine rupture. Also a lap belt worn too high could produce uterine rupture because of direct force transmission to the uterus on impact. The use of shoulder restraints improves fetal outcome presumably because of the greater surface area over which the deceleration force is dissipated as well as the prevention of forward flexion of the mother. Therefore, determination of the type of restraint device worn by the pregnant patient is important in the overall assessment.

IV. Severity of Injuries

Severity of maternal injuries determines not only maternal but also fetal outcome. Therefore, the method of treatment is also dependent on the severity of maternal injuries. All pregnant patients with major injuries require admission to a facility with surgical obstetrical
capabilities since there is a 24% maternal mortality rate and 61% fetal mortality rate in this group of patients. Eighty percent of females admitted to the hospital in hemorrhagic shock have an unsuccessful fetal outcome. Even the pregnant patient with minor injuries should be carefully observed since occasionally even minor injuries are associated with such complications as fetomaternal hemorrhage (the presence of fetal RBCs in the maternal circulation). Fetal injuries usually tend to occur in late pregnancy, the most common being skull fractures and intracranial hemorrhage, although any injury could occur.

V. Diagnosis and Management

A. Initial Assessment

1. Patient position

Uterine compression of the vena cava reduces venous return to the heart, thereby decreasing cardiac output and aggravating the shock state. Elevated caval pressures below the point of compression can lead to extension of placental separation. Therefore, unless a spinal injury is suspected, the pregnant patient should be transported and evaluated on her left side. If the patient is in a supine position, the right hip should be elevated and the uterus should be displaced manually to the left side to relieve pressure on the inferior vena cava.

2. Primary survey

Follow the ABCs and administer supplemental oxygen. If ventilatory support is required, consideration should be given to hyperventilating the patient. Because of the increased intravascular volume and the rapid contraction of the uteroplacental circulation shunting blood away from the fetus, the pregnant patient can lose up to 35% of her blood volume before tachycardia, hypotension, and other signs of hypovolemia occur. Thus, the fetus may be "in shock" and deprived of vital perfusion, while the mother's condition and vital signs appear stable. Crystalloid fluid resuscitation and early type-specific blood administration are indicated to support the physiologic hypervolemia of pregnancy. Avoid administering vasopressors to restore maternal blood pressure, because these agents further reduce uterine blood flow, resulting in fetal hypoxia.

B. Secondary Assessment

The secondary survey should follow the same pattern as in the nonpregnant patient. Indications for diagnostic peritoneal lavage are the same and this may be conducted safely if the incision is made in the midline well above the fundus of the uterus. The examination of the patient should include an assessment of uterine irritability, fundal height and tenderness, fetal heart tones, and fetal movement. Use a Doppler ultrasound stethoscope or fetoscope to auscultate fetal heart tones. Pay careful attention to the presence of uterine contractions suggesting early labor; or tetanic contractions accompanied by vaginal bleeding, suggesting premature separation of the normally implanted placenta. The evaluation of the perineum should include a formal pelvic examination. The presence of amniotic fluid in the vagina, evidenced by a pH of 7 to 7.5, suggests ruptured chorio-amniotic membranes. Cervical effacement and dilatation, fetal presentation, and the relationship of the fetal presenting part to the ischial spines should be noted. Because vaginal bleeding in the third
trimester may indicate disruption of the placenta and impending death of the fetus, an obstetrician ideally should carry out the vaginal examination or be called immediately if blood is coming from the cervical os. The decision regarding an emergency cesarean section should be made in conjunction with an obstetrician.

Admission to the hospital is mandatory in the presence of vaginal bleeding, uterine irritability, abdominal tenderness, pain or cramps, evidence of hypovolemia, changes in or absence of fetal heart tones, or leakage of amniotic fluid. Care should be provided at a facility with appropriate fetal and maternal monitoring and treatment capabilities. **The fetus may be placed in jeopardy even with apparent, minor maternal injury.**

C. Monitoring

1. Patient

If possible, the patient should be monitored on her left side after physical examination. Monitoring of the CVP response to fluid challenge is extremely valuable in maintaining the relative hypervolemia required in pregnancy.

A correlation between maternal serum bicarbonate level and fetal outcome has been suggested. Therefore, it may be useful to monitor the maternal serum bicarbonate level in addition to other hemodynamic parameters. It should be noted that serum bicarbonate level may be depressed in these patients when large volumes of normal saline are infused. The requirement for saline infusion itself may be a reflection of severity of injury and blood loss opposed to the bicarbonate itself.

2. Fetus

Fetal distress can occur at any time and without warning. Although fetal heart rate can be determined with any stethoscope, the fetal heart rate and rhythm is best monitored continuously using the ultrasonic Doppler cardioscope. The fetus should be monitored continually to ensure early recognition of fetal distress. Inadequate accelerations of fetal heart rate in response to fetal movement, and/or late or persistent decelerations of fetal heart rate in response to uterine contractions indicate fetal hypoxia.

Indicated radiographic studies should be performed, because the benefits certainly outweigh potential risk to the fetus. However, unnecessary duplication of films should be avoided.

D. Definitive Care

In addition to the spectrum of injury found in a nonpregnant patient, trauma during pregnancy may cause uterine rupture. The uterus is protected by the bony pelvis in the first trimester, but it becomes increasingly susceptible to injury as gestation progresses. Traumatic rupture may present a varied clinical picture. Massive hemorrhage and shock may be present or only minimal signs and symptoms may be present.
Roentgenographic evidence of rupture includes extended fetal extremities, abnormal fetal position, or free intraperitoneal air. Suspicion of uterine rupture mandates surgical exploration.

Placental separation from the uterine wall (abruptio placentae) is the leading cause of fetal death after blunt trauma. Abruptio can occur following relatively minor injuries especially late in pregnancy. With separation involving 25% of the placental surface, external vaginal bleeding and premature labor may begin. Larger areas of placental detachment are associated with increasing fetal distress and demise. Other than external bleeding, signs and symptoms may include abdominal pain, uterine tenderness, uterine rigidity, expanding fundal height, and maternal shock. Uterine ultrasonography will frequently demonstrate the lesion.

With extensive placental separation or with amniotic fluid embolization, widespread intravascular clotting may develop causing depletion of fibrinogen, other clotting factors, and platelets. This consumptive coagulopathy may emerge rapidly. In the presence of life-threatening amniotic fluid embolism and/or disseminated intravascular coagulation, uterine evacuation should be accomplished on an urgent basis.

Consequences of fetomaternal hemorrhage include not only fetal anemia and death, but also isoimmunization if the mother is Rh-negative. Since as little as 0.01 mL of Rh-positive blood will sensitize 70% of Rh-negative patients, the presence of fetomaternal hemorrhage in an Rh-negative mother should warrant Rh immunoglobulin therapy. This should be undertaken early in consultation with and under the direction of an obstetrician. Although a positive Kleihauer-Betke test (a maternal blood smear allowing detection of fetal RBCs in the maternal circulation) indicates fetomaternal hemorrhage, a negative test does not exclude minor degrees of fetomaternal hemorrhage that are capable of sensitizing the Rh-negative mother. Where this test is readily available, increasing ratios of fetal to maternal RBCs demonstrated in sequential maternal blood smears may be used as an index of increasing fetomaternal hemorrhage. All pregnant Rh-negative trauma patients should be considered for Rh immunoglobulin therapy unless the injury is so minor or remote from the uterus as to make fetomaternal hemorrhage unlikely. In situations where there is doubt as to the severity of the injury or the presence of fetomaternal hemorrhage, then the traumatized pregnant Rh-negative patient should receive Rh immunoglobulin therapy. Three hundred micrograms of Rh immunoglobulin therapy is required for every 30 mL of fetomaternal hemorrhage. In 90% of cases the volume of fetomaternal hemorrhage is less than 30 mL. Immunoglobulin therapy should be instituted within 72 hours of injury.

The large, engorged pelvic vessels that surround the gravid uterus can contribute to massive retroperitoneal bleeding after blunt trauma with associated pelvic fractures.

Initial management is directed at resuscitation of the pregnant patient and stabilization of her condition because the fetus' life at this point is totally dependent on the integrity of the mother's. Fetal monitoring should be maintained after satisfactory resuscitation and stabilization of the mother's condition.

Obstetric consultation is necessary for the care of the fetus.
VI. Summary

Important and predictable anatomic and physiologic changes occur during pregnancy that may influence the evaluation and treatment of the injured pregnant patient. Vigorous fluid and blood replacement should be given to correct and prevent maternal as well as fetal hypovolemic shock. A search should be made for conditions unique to the injured pregnant patient, such as blunt or penetrating uterine trauma, abruptio placentae, amniotic fluid embolism, isoimmunization, and premature rupture of membranes. Attention also must be directed toward the second patient of this unique duo - the fetus - after its environment has been stabilized. A qualified surgeon and obstetrician should be consulted early in the evaluation of the pregnant trauma patient.