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Introduction

Trauma is the leading cause of death for women in the United States ages 34 years of age and younger. It is the primary cause of deaths not attributable to medical causes in pregnancy, complicating 6–8 % of pregnancies, and results in emergency surgery in up to 20 % of pregnant women with traumatic injuries [1–7]. Trauma-related deaths are often not included in maternal mortality reviews and the precise contribution of trauma to mortality rates is probably underestimated [8, 9]. Nevertheless, trauma accounts for up to 46 % of pregnancy-associated deaths, or greater than one million deaths annually worldwide [6, 10–12]. While pregnancy-related maternal deaths (those due to medical events during pregnancy) have declined, those due to injury have increased [1]. Causes include active-employment while pregnant, greater number of miles traveled in an automobile, and growing incidence of intimate partner violence.

Maternal injury can have serious consequences, including fetal loss, preterm rupture of membranes, preterm delivery, placental abruption, cesarean delivery, and stillbirth (see Fig. 16.1) [13–18]. One study estimated that as many as one-third of pregnant women hospitalized for trauma will deliver during their hospitalizations [19]. Actual fetal injury and loss

rates may be undercounted due to a lack of standardized reporting methods; for example, medical care for the initial episode of maternal trauma and subsequent fetal loss may occur at different medical centers; the fetal loss may occur after unreported maternal trauma; or the fetal loss may not be recorded because it occurred at less than 20 weeks gestation [20].

Trauma in pregnancy has been associated with younger age, less education, being unmarried, and it is more common among those who have used tobacco, alcohol, or illicit substances while pregnant [21, 22]. Data from the American College of Surgeons National Trauma Data bank indicate that alcohol and illicit substances are implicated in pregnancy-associated trauma in 12.9 % and 19.6 % of cases, respectively [3]. While pregnancy itself does not increase morbidity or mortality due to injury, it has been identified as an independent risk factor for trauma. This includes violent assaults aimed at causing fetal injury [23, 24].

Fetal outcomes after maternal trauma are poor, with mortality reported as high as 40–50 % [25]. Importantly, fetal morbidity and mortality can occur in the setting of insignificant maternal injury [25, 26], and severity scores for maternal injury do not accurately predict placental abruption or fetal death [27–29]. Consequently, it is essential that all women of childbearing age who experience trauma be evaluated for pregnancy, and if pregnant undergo fetal evaluation, even in the setting of minor injury.

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- Maternal death
- Maternal loss of consciousness
- Injury Severity Score > 15
- Pelvic fracture
- Failure to use seat belts
- Early gestational age
- Vaginal bleeding
- Severe hemorrhage
- Coagulopathy
- Serum lactate > 2 mEq/L

Fig. 16.1 Factors associated with trauma-associated fetal loss

Types of Trauma

Patterns of Injury

The risk for trauma increases as pregnancy progresses, with 10–15 % of injuries occurring in the first trimester and 50–54 % in the third trimester [30], and parturients are more likely to have abdominal rather than head injuries [31]. The uterus is protected within the bony pelvis until 12 weeks gestation, so chances of fetal injury are limited during the first trimester. The American College of Obstetricians and Gynecologists (ACOG) categorizes trauma into three categories: blunt abdominal injury, penetrating trauma, and pelvic fractures [32]. Maternal and fetal mortality may result from the injury itself or from indirect causes, such as maternal shock, disseminated intravascular coagulation, or acute respiratory distress syndrome.

Blunt Injury to the Abdomen

Blunt abdominal injury accounts for two-thirds of trauma cases in pregnancy. The force of the impact directly correlates with degree of maternal and fetal injury. As pregnancy progresses, the gravid uterus pushes abdominal contents upward, thus decreasing the risk of maternal bowel injury from a direct abdominal blow, although the risk of hepatic or splenic rupture and retroperitoneal hemorrhage remains.

With increasing gestational age, the uterus is at greater risk in abdominal injury. While the

amniotic fluid absorbs collision energy and prevents its direct transmission to the fetus, premature rupture of membranes can occur because the portion of membranes lying over the internal cervical os is unsupported by the uterine wall and this creates a potential site for tears. Placental abruption is common and it may occur within hours of injury or later.

Forceful direct impact and contrecoup injuries can contribute to traumatic rupture of the uterus itself. Clinical presentation of uterine rupture ranges from hemorrhagic shock with maternal collapse, to nonspecific abdominal discomfort. Fetal injuries following abdominal trauma are most commonly reported during the third trimester and while maternal mortality may be less than 10 %, fetal death rates approach 100 % [33].

Penetrating Trauma

There are few data on outcomes of penetrating injury during pregnancy. A retrospective study of abdominal injuries seen in a level 1 trauma center from 1996 to 2008 reported that blunt injuries occurred in 91 % whereas penetrating injuries accounted for only 9 % of patients [34]. Among penetrating injuries, 73 % were caused by gunshots. Maternal mortality did not differ between the two groups, but fetal mortality was 73 % following penetrating injuries, but only 10 % following blunt trauma. Overall, gunshot wounds are reported to cause fetal injury in 60–70 % and lead to death in 40–65 % of cases [4, 34, 35]. While gunshot wounds require a laparotomy to determine the full scope of injury, stab wounds do so only if the blade appears to have penetrated the peritoneum.

Pelvic Fracture

Pelvic fractures contribute to high maternal and fetal mortality. Medical treatment is complicated by pregnancy and there are increased risks of obstetrical complications. A literature review of 101 case reports of pelvic or acetabular fractures in pregnancy found that maternal and fetal deaths

did not correlate with the type of fracture (simple or complex), its location, or trimester of pregnancy [36]. Overall, fetal mortality was 35 % and maternal mortality 9 %. However, an institutional study of 148 motor vehicle accident (MVA)-related injuries at a level 1 trauma center, in which there were no maternal deaths, noted that mothers of five of the seven cases of fetal demise had sustained pelvic fractures [37]. The odds of fetal loss were 48 times higher if their mothers sustained a pelvic fracture, versus those who did not, and ten times more likely if the mother lost consciousness on impact [37]. Among the seven fetal deaths, only one was due to direct uterine trauma; the other six were the result of spontaneous abortion.

Pelvic fractures may lead to placental abruption in as many as 30 % of cases [38]. In one retrospective study of maternal fractures, patients who delivered during hospitalization had 15-fold increase in placental abruption and 20-fold increases in transfusions and stillbirths [39]. Those who were discharged and delivered subsequently had a 47 % increase in abruptions and 18 % increase in rate of preterm deliveries. In this study, women with pelvic fractures were the only group in which there was long-term increased risk of fetal death.

Causes of Maternal Trauma

Motor Vehicle Accidents

The overall incidence of motor vehicle accidents (MVA) has been estimated at 2.8 % [21], and MVAs may account for 34–70 % of all traumatic injuries during pregnancy [3, 16, 40, 41]. Maternal mortality following MVAs has been estimated at 1.4 and fetal mortality at 3.7 per 100,000 pregnancies, respectively [42]. Both maternal morbidity and mortality are substantially increased when seat belts are not used or placed incorrectly, and adverse outcomes have been reported in 100 % of women injured in MVAs who were not wearing seatbelts [43]. The use of alcohol and other intoxicants has

been implicated as a risk factor for MVAs during pregnancy, with up to 45 % of collisions involving maternal alcohol use [22, 44].

Seat belts can prevent maternal impact with the steering wheel in both front and rear collisions [45]. Many pregnant women fail to use or correctly place seatbelts [46], and one study reported that only half of patients indicated they received counseling about seatbelt use from their physicians [47]. Fear of injuring their fetus and belt discomfort have been reported as reasons for avoiding their use [43], and when used improperly, seatbelts can cause severe uterine and fetal injury. Yet, fetal deaths are three times more likely in MVAs when the mother is not wearing a seat belt [21]. Recommendations for their correct use, with or without airbag activation during MVAs, have been associated with improved fetal outcomes [48, 49]. Current guidelines recommend using seatbelts throughout pregnancy, with the lap belt portion placed under the abdomen and over the anterior superior iliac spines and pubic symphysis. The shoulder portion of the belt should be between the breasts with the belt as snug as comfort permits [32].

In high-speed automobile crashes, airbags and three-point seatbelts can be life saving, and one study reported they were protective only when vehicle speeds exceed 32–38 mph. There have been case reports of uterine rupture and placental abruption with airbag deployment [50, 51], however, a population-based study of airbag deployment during MVAs in pregnancy failed to find a statistically increased risk of poor fetal outcomes [52]. Another study of 42 pregnant women who were wearing three-point seat belts when they were involved in MVAs found that deployment of airbags further reduced the risk of adverse fetal outcomes [48].

Despite the absence of data regarding airbag injury risk, new manufacturing criteria for “advanced air bags” require supplemental restraint systems that are designed to accommodate children and women as well as standard-sized men. According to the National Highway Traffic and Safety Association, pregnant women should be at least 10 in. away from an airbag in

the dashboard or steering wheel and the seat should be pushed back or reclined as the abdomen grows during pregnancy.

During collisions, substantial mechanical forces are placed on the uterus. Both shear force failure (or strain) and tensile failure (by a contrecoup mechanism) have been implicated in uterine injury. The displacement of the uterus forward during a collision can generate negative forces and injure the opposite side of the uterus. In addition, upon impact the mother's torso folds over the abdomen, markedly increasing abdominal pressure. The combination of these two movements can cause placental abruption in addition to maternal injuries [53].

Falls

Falls are estimated to complicate 48.9 of 100,000 pregnancies, and they account for 22–52 % of all traumatic injuries in pregnancy [54, 55]. As many as one-in-four women will fall while pregnant [56, 57]. The likelihood of falling increases in the second and third trimesters when gait and balance are altered by shifts in a women's center of balance [58–60] and women are less able to stabilize themselves when their body position changes abruptly [61]. In one study of pregnant women hospitalized for falls, fractures were the most common injuries, followed by contusions and sprains [56]. This study also reported a 4.4-fold increase in preterm labor, an 8-fold increase in placental abruption, and a 2.9-fold increase in fetal hypoxia compared with those who had not fallen.

Assaults, Homicide, and Suicide

Assault during pregnancy is a leading cause of maternal and fetal deaths [62–66]. The prevalence of domestic violence (DV) or intimate partner violence (IPV) varies worldwide. In the United States, it occurs in 22.1 % of the women of childbearing age, although it is higher in specific groups [67, 68]. Pregnancy appears to be an independent risk factor for battery [23, 69].

Women who are abused while pregnant have a threefold risk of becoming homicide victims during the same pregnancy [61]. Among postpartum women 15–19 years old, the risk of homicide was 2.6-fold greater than that of women who had not been pregnant [70]. One study of mortality among pregnant women 15–44 years of age in New York City found that for injury-related deaths, 63 % were homicides and 13 % were suicides [12]. Another review of pregnancy-associated deaths in Maryland found that homicide was the leading cause of all maternal deaths [71].

Data from the multistate National Violent Death Reporting System (NVDRS) for 2003–2007 reveal that pregnancy-associated violent death mortality in women ages 15–54 accounted for a rate of 4.9 per 100,000 live births [62]. The homicide rate was calculated at 2.9 per 100,000 live births. Of the 139 homicides among women of childbearing age, 108 (77 %) occurred during pregnancy and the remainder within the first postpartum year. Women at extremes of age were at higher risk. Younger women were at greatest risk, with those 24 years and younger accounting for more than half (53.9 %) of pregnancy-associated homicides, but only 33.6 % of live births in reporting states. Women 40 years or older were also at elevated risk of homicide. In this sample, 59.1 % of homicides were due to intimate partner violence [62]. Another study, based on records of the New York City Office of the Chief Medical Examiner between 1998 and 2009, revealed that in 19 of 27 homicides among pregnant women, the victim and suspect were known to each other [72].

Violent abuse in pregnancy is associated with a 2.7-fold increase in preterm births and 5.5-fold increase in low birthweight infants [73]. Risk factors for DV/IPV include substance abuse, less education, low socioeconomic status, African-American race, unintended pregnancy, unmarried status, a history of DV/IPV prior to pregnancy, or witnessing violence by mother or intimate partner as a child [74]. Fetal mortality following intimate partner violence has been reported as high as 16 % [34].

Most studies have reported a lower incidence of suicide in pregnant versus non-pregnant

women [63]. According to NVDRS data for 2003–2007, maternal suicide accounted for two deaths per 100,000 live births [62]. In this survey, women over 40 years of age accounted for 17.0 % of pregnancy-related suicides but only 2.8 % of the live births. The homicide and suicide rates in this study were both higher than reported maternal death rates reported from common obstetric causes (hemorrhage/placenta previa, eclampsia/preeclampsia, and amniotic fluid embolism). Fetal or infant death, and substance abuse are maternal risk factors for attempting suicide in during pregnancy and postpartum [75, 76].

Burns and Electrical Injuries

Our understanding of burns in pregnancy is limited, as the reported incidence of burn injuries is low and burn victims are not consistently screened for pregnancy. Worldwide, approximately 7 % of women of reproductive age who are treated for burns are pregnant [77]. A prospective study of pregnant women admitted to a burn center in Iran over 9 years, found that larger total body surface area burned correlated with those that were self-inflicted. In this study, 27.45 % of burns were suicide attempts. Total body surface area burned and degree of burn are related to the extent of maternal and fetal injury. As the body surface area of injury approaches 40 %, mortality rates for mother and fetus approach 100 % [77, 78]. Sepsis complicating burn injury is a major contributor to maternal and fetal mortality [79].

A recent observational study used thromboelastography (TEG) to evaluate hypercoagulability in burn patients and noted a hypercoagulable state developing 1 week after the initial injury. Pregnant patients with burns should receive routine thromboprophylaxis as the hypercoagulable state of pregnancy may be exacerbated and deaths from pulmonary embolisms occur in the setting of burn injury [80].

Smoke inhalation during burn injury significantly increases maternal and fetal mortality [81, 82] due to oxygen depletion, carbon monoxide (CO) poisoning [82] and cyanide (CN) poisoning from combustion of synthetic products silk and wool [83]. Fetal hemoglobin has greater affinity

than maternal hemoglobin for CO, and fetal carboxyhemoglobin levels can reach as high as 15 %. The fetal effect of inhaled CO and CN poisoning depends on the gestational age of the fetus and combined exposure exhibits a synergistic effect.

CO poisoning can be treated with normobaric oxygen. Hyperbaric oxygen therapy has been used in pregnancy, but remains controversial. Prompt treatment of CN with intravenous hydroxocobalamin effectively removes CN, raises the threshold for lethal CO poisoning, and is superior to combined treatment with amyl nitrate, sodium nitrate, and sodium thiosulfate, all of which are contraindicated in pregnancy [83].

There are few reports of electrical injuries in pregnancy, and among reported cases there has been wide variance in the degree of injury. In one study of 15 cases of severe electrical injury during pregnancy, fetal mortality was 73 % [84]. A prospective study of minor electrical shocks from household appliances, however, found no difference in birth outcomes compared with controls [85]. The magnitude of the current appears to be related to the degree of fetal injury, as does trajectory through the uterus and conduction of the current to the fetus by amniotic fluid. In cases of severe electric shocks, resulting falls can lead to abdominal injury and placental abruption.

Poisoning

Case reports of poisoning in pregnancy are limited and primarily concern suicide attempts (see above) and inadvertent drug overdoses. One study found that pregnant women accounted for only 0.07 % of calls to a poison control center over 4 years [86]. If all women who sought help had received a pregnancy test, however, the number would likely have been higher [87]. No studies have examined the teratogenic risks of specific poison antidotes, and it is recommended that they not be withheld from pregnant women if there are clear medical indications [88].

Drug overdoses in pregnancy have been reported from both over-the-counter medications such as acetaminophen and prescription drugs.

Lead has been found to contaminate several naturopathic medications. Isolated case reports of accidental overdoses of hospital-delivered medications, such as epidural local anesthetics or misoprostol, have also been noted. Opioid-medications are responsible for the recent dramatic increase in overdose fatalities, with overall mortality rates three times higher in rural compared with metropolitan areas. A study of pregnancy-related deaths in Florida from 1999 to 2005 found that prescription drugs were detected in 54 % of cases, with opioids being the most commonly detected drug, followed by benzodiazapines. Among pregnant women who died, drug toxicity and motor vehicle accidents each accounted for one-third of the total deaths, followed by gunshot wounds in 14 % [89].

Envenomation injuries, caused by snakes, scorpions, spiders, jellyfish, and hymenoptera (bees, wasps, hornets, ants) are rare in pregnancy and treatment has been directed by case reports on non-pregnant subjects. Venom-specific approaches based upon supportive therapy and anti-venom administration is indicated to support the mother [90].

Maternal and Fetal Outcomes Following Trauma

Maternal mortality is directly linked to the severity of the traumatic injury. Traumatic head injuries, internal injuries, and hemorrhagic shock account for the majority of maternal deaths [16, 91, 92]. Pregnant women who are injured and deliver at the time of the initial trauma hospitalization experience worse outcomes. One retrospective analysis of hospital discharge records in California reported that pregnant women who delivered during their hospitalization for trauma had a 9-fold greater risk of placental abruption, a 42-fold greater risk of uterine rupture and 69-fold greater risk of maternal death compared with those who delivered during a subsequent admission [16]. Data are conflicting as to whether being pregnant during traumatic injury is associated with a survival advantage over non-pregnant women of childbearing age [31].

Obstetric complications of trauma include preterm labor and delivery, preterm premature rupture of membranes, placental abruption, fetomaternal hemorrhage, and uterine rupture. After 22–24 weeks gestation, preterm labor occurs in 25 % of trauma cases [4]. Most preterm deliveries occur after discharge from the initial trauma hospitalization. Calcium channel blockers such as nifedipine are widely used off-label for tocolysis for those patients who remain undelivered with preterm labor. Magnesium sulfate can be used for short-term tocolysis (5–7 days), and has been shown to have fetal neuroprotective effects in early preterm deliveries (<32 weeks) for all pregnancies [93, 94].

Abruption of the placenta occurs with 1.7 % of maternal injuries and in up to 40 % of severe injuries [95]. It is more common after blunt trauma [16]. Ultrasound is a relatively insensitive test for placental abruption and due to its delayed occurrence, continuous fetal monitoring is recommended for 6 h, even in the setting of minor trauma [96].

Uterine rupture is a rare consequence of trauma that has grave consequences for the fetus. Rupture occurs most commonly in rapid deceleration or compression injuries, and is typically found in patients with a previous uterine scar. Following uterine rupture, fetal mortality is almost universal and the maternal mortality rate is 10 % [97]. The risk of rupture increases with gestational age and with the severity of trauma. Abdominal pain, uterine tenderness, loss of abdominal shape, cessation of contractions, and maternal hemodynamic instability may be found. The uterus may rupture posteriorly if it is unscarred by previous surgery and typical findings on abdominal examination may be absent. Bladder injury is also associated with posterior ruptures, and blood or meconium may be found in the urine [98].

Of note, uterine rupture and ruptured membranes following blunt abdominal trauma are associated with amniotic fluid embolism (AFE) [99]. Signs and symptoms of AFE vary and include, shock, acute hypertension, seizure, respiratory distress, disseminated intravascular coagulation or cardiac arrest. These clinical findings in the setting of traumatic injury should prompt high suspicion of AFE [100].

Fetomaternal hemorrhage occurs in up to 30 % of pregnant trauma patients and is more common in those who sustain anterior trauma and have an anterior implanted placenta [101]. Hemorrhage can cause fetal anemia, arrhythmias, and exsanguination resulting in fetal death. In addition, mothers are at risk of Rh sensitization: as little as 0.01 mL of Rh-positive blood from the fetus can result in sensitization in Rh-negative women.

In the California study cited above, fetuses delivered during the initial trauma hospitalization had a 2-fold increase in premature delivery, a 4.6-fold increase in fetal death and a 3-fold increase in neonatal death [16]. Because the fetal head is in the pelvis near term, there is a risk of fetal skull fracture and brain injury with pelvic fractures. Even minor trauma during pregnancy can significantly increase the risk of preterm delivery, despite normal fetal monitoring and observation. Pregnant women who are discharged after hospitalization for trauma should still be considered at risk for the remainder of their pregnancies [26].

The Physiology of Pregnancy and Management of the Trauma Patient

The altered physiology of pregnancy and the fetal response to trauma affect both the severity of trauma and its treatment. Initial management is focused on maternal stabilization. Treatment must be guided by pregnancy-related changes in maternal physiology and how they affect trauma life support protocols. Physiologic changes in pregnancy involve alterations of the airway anatomy, gastrointestinal, respiratory, cardiovascular, and hematologic physiology that are particularly relevant in the trauma patient [102] and can influence the evaluation and treatment of traumatic injury (see Table 16.1). These changes allow for greater clinical compensation to trauma, but this can sometimes delay recognition of the extent of injury and hemorrhagic shock (see Fig. 16.2).

Airway Management

Airway management in normal pregnancy presents additional risks over the non-pregnant patient. Changes in oncotic pressure and increases in circulating blood volume lead to engorgement of the naso- and oropharyngeal mucosa and larynx, resulting in edema and friability of the upper airway and a predisposition to airway obstruction. Smaller endotracheal tubes (6–7 mm internal diameter) may be needed for intubation, and caution is indicated when inserting nasopharyngeal airways or endotracheal tubes. Soft tissue edema, enlargement of the tongue and breast tissue, and generalized weight gain can complicate laryngoscopy. A shorter laryngoscope handle may be needed to facilitate visualization of the airway structures.

Trauma can further complicate airway management. Fluctuating levels of consciousness as a result of intracranial injuries, alcohol or drug ingestion, hypoxia or shock, can lead to loss of airway reflexes. Specific injuries, such as facial fractures, burns, and cervical spine instability pose additional challenges.

In late-trimester pregnancies, difficult airway management should be anticipated and additional equipment made available, such as a stylet, gum elastic bougie, levered-laryngoscope, lightwand, intubating laryngeal mask airway (LMA), and fiberoptic or video laryngoscope. A study of maternal airway grades at 12 and 28 weeks found that the percent of Mallampati Grade IV airways, with only views of the hard palate and no view of soft palate or uvula, increased by 34 % [103].

In obese patients, an airway ramp can be made up of a rolled towel or blanket placed under the patient's upper back and head until horizontal alignment is achieved between the external auditory meatus and sternal notch. This has been found to be superior to the traditional "sniff" position that is created by placing a cushion under the patient's head and raising the occiput [104, 105]. In pregnant trauma patients, once cervical spine instability has been ruled out, ramped positioning may facilitate direct laryngoscopy.

Table 16.1 Physiologic changes of pregnancy

<i>Cardiovascular</i>			Clinical significance
Heart rate	Increased 15–20 bpm	75–95 bpm	Adaption to tolerate blood loss
Cardiac output	Increased 30–50 %	6–8 L/min	
Mean arterial blood pressure	Decreased 10 mmHg	Midtrimester 80 mmHg	
Systemic vascular resistance	Decreased 10–15 %	1,200–1,500 dyn/s/ cm ⁻⁵	
<i>Respiratory</i>			
Tidal volume	Increased 40 %	700 mL	
Minute ventilation	Increased 40 %	10.5 L/min	Respiratory alkalosis
Expiratory reserve volume	Decreased 15–20 %	550 mL	
Functional residual capacity	Decreased 20–25 %	1,350 mL	Rapid desaturation
<i>Blood gas</i>			
pH	Unchanged	7.4–7.45	
pCO ₂	Decreased	27–32 mmHg	
pO ₂	Increased	100–108 mmHg	
HCO ₃	Decreased	18–21 mEq/L	
<i>Hematologic</i>			
Blood volume	Increased 30–50 % 13–18 weeks	4,500 mL	
Erythrocyte volume	Increased 10–15 %		Dilutional anemia
Hemoglobin	Decreased 1–2 g/dL	9–11 g/dL	
Leukocytes	Increased up to 18 × 10 ⁹ Leukocytes/L 24–40 weeks	5,000–150,000/mm ³	
<i>Coagulation</i>			
Factors I, II, V, VII, IX, X, and XII	Increased		Hypercoagulable state

There are no studies that compare direct and video laryngoscopes in pregnant patients. In cases of neck trauma, there are conflicting data regarding decreased motion of the cervical spine using videolaryngoscopes [106, 107]. One study which randomized non-pregnant trauma patients to intubation with Glidescope[®] video laryngoscope or direct laryngoscopy with Macintosh blade found that the use of the Glidescope resulted in longer median intubation times without a mortality benefit [108]. Video laryngoscopes may have theoretical benefits in late-term pregnancy or in cases of maternal obesity, but there are no robust supporting data. Once intubation has been achieved, nasogastric decompression should be initiated to minimize the risk of aspiration. If intubation is impossible, a LMA may permit ventilation, although the risk

of aspiration remains. Some patients may require cricothyrotomy or tracheostomy.

Gastrointestinal Changes

While all trauma patients are at risk for aspiration, the risk is increased in pregnant patients due to progesterone-mediated relaxation of the lower esophageal sphincter, gastric tone and mobility. One case-controlled study of non-trauma patients reported an aspiration risk as high as 8 % [109]. Prolonged bag/mask ventilation in a trauma setting will increase the risk of aspiration. While pregnancy itself does not prolong gastric emptying, delays occur with obesity, labor, and the presence of pain, or opioid administration. In addition, many pregnant women may resort to

Fig. 16.2 Maternal physiologic changes associated with hemorrhage

- Compensated blood loss: 10–15% of blood volume (600 mL–900 mL)
 - Heart rate unchanged
 - Mean arterial pressure unchanged
- Mild blood loss: 20–25% of blood volume (1200 mL–1500mL)
 - Tachycardia (95–105 bpm)
 - Mean arterial pressure drops 10–15% (70–75 mmHg)
 - Vasoconstriction–cold, pale extremities, poor capillary refill
- Moderate blood loss: 25–35% of blood volume (1500 mL–2000 mL)
 - Tachycardia (105–120 bpm)
 - Mean arterial pressure drop 25–30% (50–60 mmHg)
 - Tissue hypoxia
 - Oliguria
 - Restlessness
- Severe blood loss: > 35% of blood volume (>2000 mL)
 - Tachycardia (>120 bpm)
 - Hypotension (mean arterial pressure <50 mmHg)
 - Tissue hypoxia
 - Oliguria
 - Altered consciousness
 - Coagulopathy–disseminated intravascular and/or trauma–associated

small frequent meals, increasing the likelihood of a full stomach when presenting with trauma.

Respiratory Changes

Weight gain and enlargement of the uterus during pregnancy cause decreased functional residual capacity (FRC) and can lead to rapid desaturation, further complicating airway management. Oxygen should be provided to all pregnant trauma patients, with early consideration of an oral, nasal, or endotracheal airway. Metabolic needs and oxygen consumption are high in pregnancy, both of which worsen hypoxia. Denitrogenation with 100 % oxygen must be performed prior to intubation. Maternal oxygen saturation should be maintained at ≥ 95 % in order to maintain a $\text{PaO}_2 > 70$ mmHg and optimize oxygen diffusion across the placenta. When maternal oxygenation falls below 60–70 mmHg, fetal oxygenation is compromised.

Maternal minute ventilation rate increases as a result of expanded tidal volume and progesterone-mediated stimulation of the medullary respiratory center that controls ventilatory drive. This results in lower carbon dioxide tensions of between 28 and 32 mmHg. There is

a compensatory excretion of bicarbonate to maintain an arterial pH of 7.40–7.45. These values need to be taken into account when interpreting blood gases and adjusting ventilator settings.

In trauma patients, ventilatory drive can be reduced following drug overdose, poisoning, alcohol ingestion, head injury, pneumothorax, hemothorax, lung, or chest wall injury. Successful management of these injuries may involve drainage of air or blood. In pregnancy, the thoracic anteroposterior diameter increases and the diaphragm moves 4 cm cephalad. If a thoracostomy procedure is needed, needle entry should be made one or two intercostal spaces higher than in non-pregnant patients to avoid injuring the diaphragm and abdominal organs.

Cardiovascular Changes

Many of the cardiovascular changes seen in pregnancy can complicate the evaluation and management of pregnant trauma patients. The enlarged uterus causes the heart to shift cephalad and to the left. The electrocardiogram (ECG) can show sinus tachycardia, left-axis deviation, non-specific ST-T changes, and inverted or flattened

T-waves. Q-waves may also be present in leads III and avF. Premature atrial and ventricular beats are common. As the body adjusts to expanded circulating volume and preload, the heart becomes hypertrophic and dilated with an enlarged left ventricular end-diastolic volume. Afterload, however, is reduced due to decreased peripheral vascular resistance. The heart rate and stroke volume begin to increase early and peak at 28–32 weeks' gestation. Heart murmurs, such as a pulmonary mid-systolic murmur and a supraclavicular murmur, may be present.

As the pregnant patient prepares for the blood loss of delivery, blood volume increases 50 % with a 30 % increase in red cell volume. The greater increase of plasma volume over erythrocyte count leads to a dilutional anemia resulting in hemoglobin values of 9–11 g/dL. Significantly, the pregnant patient can lose 2,000 mL of blood (30–40 % of blood volume) before she reveals changes in heart rate or blood pressure. As blood loss approaches 2,500 mL, rapid deterioration occurs [110]. These normal physiologic changes of pregnancy may provide better organ perfusion and maternal tolerance of “shock” state and may partially contribute to increased survival after traumatic injury [111].

Blood pressure is lower than normal in pregnancy due to the vasodilatory effects of progesterone and the low-resistance placental bed, which causes a decrease in peripheral vascular resistance that reaches its nadir at 28 weeks. Normal mean arterial blood pressures in pregnancy are 80 mmHg. By the second trimester, heart rates are mildly elevated by 15–20 bpm. Vascular remodeling and deterioration of the arterial media during pregnancy can predispose the patient to vascular aneurysms and injury. Spontaneous rupture of the aorta and coronary, vertebral, splenic, hepatic, gastric, and renal arteries has been reported in pregnancy independent of traumatic injury.

Engorgement of the pelvic vasculature during pregnancy increases the risk of retroperitoneal hemorrhage and hematoma following lower abdominal or pelvic trauma. At term, uterine blood flow accounts for approximately 20 % of cardiac output and may be up to 600 mL/min [112]. The placenta is a large and inelastic

vascular structure with high blood flow and low vascular resistance, and following trauma these changes can lead to rapid maternal and fetal exsanguination. Uterine perfusion is not autoregulated and is thus dependent on maternal mean arterial blood pressure. Fetal distress, due to inadequate placental perfusion, can be one of the first indicators of maternal hemodynamic deterioration.

By 18–20 weeks gestation, the enlarged uterus exerts pressure on the inferior vena cava which can restrict venous return. In the supine position, this can reduce cardiac output by up to 30 % and cause pallor, diaphoresis, nausea, vomiting, and hypotension. Inferior vena caval compression makes the saphenous and femoral veins less preferable for delivering medication, but lower extremity access is possible in emergencies. In order to release inferior vena caval pressure and promote venous return left uterine displacement with either a hip wedge, tilted backboard, or manual displacement, should be used during resuscitation.

Coagulation

Most procoagulant factors are increased in pregnancy. This adaptive mechanism can be beneficial in achieving hemostasis after delivery and in trauma. Nonetheless, venous stasis, dilation of the pelvic vessels, and endothelial damage accompanying trauma increase the risk of thromboembolism, and prophylaxis is indicated. Fibrinogen levels are normally higher in pregnancy, thus a low fibrinogen level (<100 mg/dL) can be an early indication of massive hemorrhage or disseminated intravascular coagulation. This finding can help guide transfusion therapy.

Field Intervention and Resuscitation

Management of trauma involves a multidisciplinary team of paramedics, nurses, emergency physicians, surgeons, obstetricians, and anesthesiologists. Evaluation and resuscitation should follow the Advanced Trauma Life Support (ATLS) guidelines for rapid assessment and

management of injury, which have been shown to decrease deaths during initial stages of resuscitation for all trauma patients [112, 113]. Modification of ATLS protocols for trauma in pregnancy may include supplemental oxygen, upper-extremity intravenous access, and left uterine displacement.

Newer strategies for pre-hospital treatment of trauma patients in the United States have been termed “load and go” or “scoop and run,” as opposed to the “stay and play” strategies of Germany and France. The US strategies provide patients with minimal life-saving treatment at the site of injury before rapid transfer to trauma centers. Current guidelines for field trauma indicate that patients at >20 weeks pregnant should be transported to the closest trauma center even if they fail to meet physiologic, anatomic, or mechanistic injury criteria for severe injury [114]. This avoids the under triage of pregnant women that would occur if only physiologic and anatomic triage criteria considerations were applied [115]. Injuries that are not significant to general patients can be serious to pregnant women and even minor injuries can lead to poor fetal outcomes [25, 116, 117]. This has been shown in a retrospective cohort study of approximately 10,000 deliveries that were associated with trauma [16]. Patients with non-severe injury scores had 7.7-fold increased risk of abruption, a 16-fold increase in uterine rupture, a 4.9-fold increase in maternal death, and a 2.7-fold increase in fetal death for non-severe injuries compared with uninjured patients.

Primary Survey

The primary survey summarizes the “ABCs” of resuscitation: immediate attention to “airway, breathing, and circulation.” The trauma protocol is completed with “D and E,” which refer to disability assessment and exposing the patient for identification of all injuries [6]. In the pregnant patient “D” should also prompt left uterine displacement (see below and Fig. 16.3).

The primary survey is designed to be efficient and begins with airway assessment, maintaining

inline cervical immobilization, ascertaining that the airway is free from obstruction and that airway reflexes are intact. Oral or nasal airways, or tracheal intubation may be necessary.

Respiratory effort and rate are determined in the spontaneously breathing patient, and high-flow oxygen can be given via a non-rebreather facemask to insure adequate oxygen delivery to mother and fetus. Hyperventilation is required for treating patients with maternal head injury and suspected increased intracranial pressure, but it may result in decreased uterine blood flow. When possible, carbon dioxide tension should be maintained within normal limits for pregnancy.

Blood pressure and peripheral pulses can be decreased by aortocaval compression. Left uterine displacement can be achieved with tilt of a spine board to 15° angle with a 6-in. diameter rolled towel (or bag of crystalloid fluid) or, once spinal injuries are ruled out, through manual uterine displacement [117].

Early volume replacement must be achieved to maintain placental perfusion and fetal well-being and needs to be adjusted to reflect the increased circulating volume of pregnancy. Sources of bleeding should be identified and controlled and blood pressure parameters kept within values normal for the gestational age of the pregnancy.

Venous access should be obtained with two large bore peripheral intravenous catheters in the upper extremities, particular in the setting of aortocaval compression, but may be difficult to obtain in hypovolemic shock. In pregnancy the internal jugular vein overlies the carotid artery to a greater degree than in non-pregnant patients, making the traditional landmarks technique more risky for carotid puncture [118]. Use of ultrasound and the Seldinger technique can help to guide needle placement. Needle puncture of the internal jugular vein is preferred over the subclavian vein because it is less frequently associated with hemo- or pneumothoraces. Obtaining venous access through the femoral veins can increase the risk of thromboembolism and sepsis and should only be used in emergencies. Direct visualization of the vessel with a cutdown

Airway

- Prepare for difficult intubation with airway edema and friability, aspiration risk, rapid desaturation
- Use smaller endotracheal tube (6–7)

Breathing

- If a chest tube is needed, place in 3rd or 4th intercostal space

Circulation

- Place 2 large bore IVs above diaphragm

Disability

- Consider eclampsia as cause of altered mental status

Displacement

- Displace uterus after 18–20 weeks to avoid aortocaval compression

Exposure

- Locate entry and exit wounds

Fig. 16.3 Primary survey in the pregnant trauma patient

technique can also be employed. Finally, the use of interosseous needles has been reported in a case of massive obstetric hemorrhage [119].

The brief evaluation for disability should focus on the patient's level of consciousness using the Injury Severity Score or the Glasgow Coma Scale. A more detailed evaluation of neurologic injury should also evaluate pupil size and reactivity, lateralizing signs, and level of spinal cord injury. Eclampsia should be considered as a reason for altered mental status or seizures. Injury scales are not useful in prospectively identifying those at risk for adverse fetal outcomes, as even minor injuries are associated with increased risk [14, 25, 26]. Indicators of maternal hypoperfusion and hypoxia, direct uterine injury, and maternal head injury have been repeatedly associated with poor fetal outcomes.

After immobilizing the cervical spine to maintain the airway and providing respiratory support and fluid resuscitation, the patient should be fully exposed and evaluated for any missed injuries. In the case of gunshot injury, it is mandatory to locate entry and exit wounds.

Fluid Resuscitation

In the past 10–15 years there has been a paradigm shift regarding the best strategies to resuscitate trauma patients before achieving definitive

surgical control of hemorrhage. Current pre-hospital trauma life support recommends 1–2 L of fluids be given in the field [120]. The most recent ATLS guidelines advocate aggressive crystalloid resuscitation as 3:1 replacement of estimated blood loss, with administration of fresh frozen plasma (FFP) and platelets (PLT) when one whole blood volume had been replaced or 1 unit FFP for every 5 units packed red blood cells (RBC) administered [113, 121].

The dilutional effects of crystalloid administration can affect coagulation function [122], and large volumes can lead to acidosis, interstitial edema, tissue swelling, dysfunction of the microcirculation, and impaired oxygenation [123]. Normal saline is isotonic with respect to extracellular fluid and large volumes can result in a hyperchloremic metabolic acidosis [124]. Concern about water and sodium overload has led to the notion of “small volume” resuscitation with hypertonic saline [125]. The early use of hypertonic saline for resuscitation, however, has not improved short- or long-term outcomes, and it is not recommended in traumatic brain injury [126]. Balanced salt solutions, such as Hartmann's or Ringer's solutions, are increasingly recommended for resuscitation; they are relatively hypotonic and use lactate, acetate, gluconate, or malate as anions [127]. A recent study has shown that in the setting of hemostatic transfusion, trauma patients who received restricted

crystalloid fluids <150 mL rather than standard fluid resuscitation had better survival [128].

Colloids interfere with coagulation more extensively than crystalloids by reducing fibrin polymerization. Albumin, prepared by fractionation and heat-treatment of blood, is the reference colloid solution. A comparison of the use of saline versus albumin (the SAFE Study) showed no significant difference between the two in ICU death rates at 28 days [129]. However, albumin was associated with increased deaths at 2 years in patients with traumatic brain injury due to increased intracranial pressure in the first weeks of treatment [130]. Hemodilution with albumin results in a coagulopathy that is more easily reversed with fibrinogen and factor XIII than that of synthetic colloids [131], but it is unclear whether specific groups of patients benefit more from albumin resuscitation compared with saline. Moreover, albumin is unlikely to be widely used given its cost and problems with storage.

Worldwide, hydroxyethyl starch (HES) solutions are the most commonly used semi-synthetic colloids. HES causes movement of plasma proteins into the interstitial space, decreases levels of factor VIII and von Willebrand factor (vWF), decreases the function of activated factor XIII, and inhibits platelet function [132]. These changes are more significant than those induced by crystalloid or colloid treatment [133]. The HES-induced coagulopathy can be reversed with fibrinogen and Factor XIII which together will improve fibrin polymerization. The use of HES in resuscitation is currently controversial, as meta-analyses of HES versus control fluids show adverse effects on renal function and trends toward increased mortality [134, 135].

Transfusion

Severe trauma often results in uncontrolled and noncompressible microvascular bleeding which can potentially lead to exsanguination. Resuscitation is a key component in trauma management, but trauma-associated coagulopathy is

still seen in approximately 40 % of patient deaths [136]. So-called *damage control resuscitation* strategies target conditions that worsen hemorrhage in these patients [137]. In 2005, The US Army Institute of Surgical Research proposed a resuscitation strategy for severely injured military personnel which minimizes the use of crystalloids and colloids and matches RBC transfusions to FFP and PLT in an effort to treat and prevent ongoing coagulopathy [138]. A large study showed that patients with greater FFP:RBC ratios ($\geq 1:2$) had a decrease in short-term and 30-day mortality without any increase in multi-organ failure [139].

Damage control resuscitation differs from conventional approaches by attempting to more aggressively correct coagulation and metabolic abnormalities in the assumption that coagulopathy is present early. This strategy includes the use of blood products over isotonic fluid for volume replacement, is permissive of some degree of hypotension, and provides early correction of coagulation disorders by using blood component therapy [140]. The goal of permissive hypotension is to achieve palpable radial pulses, with the caveat that patients with head injuries should maintain a systolic blood pressure of >110 mmHg [141, 142]. In addition, relative anemia is permitted in the early stages of resuscitation before hemostasis has been achieved.

Any extrapolation of data from non-pregnant trauma patients directly to pregnant trauma patients, or pregnant patients with massive hemorrhage (as occurs with placenta accreta) without taking into account the particular physiologic requirements of pregnancy should be viewed with caution, especially in light of the increased metabolic demands of pregnancy. In a study of postpartum patients admitted to an ICU with severe hemorrhagic shock, 51 % percent were found to have elevated serum levels of cardiac troponin I (cTnI) [143]. Factors associated with elevated troponins were hemoglobin of ≤ 6.0 g/dL on admission, systolic blood pressure of ≤ 88 mmHg or diastolic blood pressure of ≤ 50 mmHg, and transfusion of ≥ 9 units of RBC within 24 h. Electrocardiogram abnormalities have been noted in patients

- Minimize early crystalloid administration
- Early use of FFP with RBC in >1:2 ratio
- Early use of platelets
- Aggressive treatment of coagulopathy
 - antifibrinolytics—tranexamic acid
 - fibrinogen concentrate
 - prothrombin complex concentrate
 - recombinant Factor VIIa

Fig. 16.4 Damage control resuscitation strategies in the pregnant trauma patient

undergoing routine cesarean sections without massive hemorrhage [144].

In the event of urgent blood transfusion in pregnant patients, O-negative/Rh-negative blood should be used in order to prevent sensitization to Rho (D) factors and erythroblastosis fetalis in future pregnancies. Balanced administration of warmed RBCs, FFP, and PLT is warranted, guided by monitoring and treatment of the coagulation abnormalities often seen with massive hemorrhage. Frequent arterial blood gas sampling during transfusion is necessary to prevent acidosis and electrolyte abnormalities (see Fig. 16.4) [145].

The optimal dose and timing of FFP delivery to trauma patients remains controversial. Collective data indicate that an FFP:RBC ratio greater than 1:2 is associated with improved survival compared to one that is less than 1:2 [146–149]. Increased platelet administration to patients with massive hemorrhage has also been shown to increase survival [150].

Techniques which minimize the use of colloids and crystalloids to avoid dilutional coagulopathy and optimize the FFP:RBC:PLT ratio have been employed in the case of massively bleeding parturients with placenta accreta or extreme uterine atony. Considered together, 1 unit of RBC, one of FFP, and one pack of PLT have a hematocrit of 29 %, a platelet count of 85,000 cells/mL, and coagulation factor activity of 62 % [151]. Early use of cryoprecipitate and antifibrinolytic agents has also been advocated [152].

Massive transfusion is defined as the loss of more than half of the circulating blood volume in 3 h or an ongoing loss of 150 mL/min. If rapid transfusion devices are used to deliver blood products (such as the Belmont[®] Rapid Infuser

FMS 2000; Belmont Instrument Corporation, Billerica, MA which can deliver 1,000 mL/min, or the Level-1[®] H-1200 Fast Flow Fluid Warmer; Smiths-Medical, St. Paul, Minnesota), then point-of-care testing must be available to evaluate acidosis and electrolyte imbalances that can rapidly occur [151, 153].

Both RBC and FFP are stored in citrate-containing solutions. A healthy adult liver can metabolize the amount of citrate contained in 1 unit of RBCs administered every 5 min, but liver metabolism is adversely affected by hypotension and hypothermia. These rates of transfusion are often exceeded in the exsanguinating patient, and as a result the liver may be underperfused. Hypocalcemia, due to citrate toxicity, and hyperkalemia from RBCs can lead to cardiac arrest, and ionized calcium and potassium must be measured frequently during massive transfusion [145].

Other complications of large volume transfusion include acute respiratory distress syndrome (ARDS), transfusion-related acute lung injury (TRALI), and transfusion-associated circulatory overload (TACO), all of which are associated with FFP and PLT administration [154]. While there is compelling evidence supporting RBC and FFP in the early stages of damage control resuscitation, there are less data supporting the aggressive use of platelets in initial therapy. Evidence supporting blood replacement rather than using isotonic crystalloids is promising but not conclusive, and the optimal ratio for RBC:FFP:PLT is still under investigation [155–157]. Platelets should be administered if the platelet count falls below 50,000/ μg L. In the case of traumatic brain injury or known platelet dysfunction, a platelet count of 100,000/ μg L should be maintained [158].

Point of care viscoelastic coagulation monitoring, such as rotational thromboelastography (ROTEM[®]; Tem International, Munich, Germany) and thromboelastography (TEG[®]; Haemonetics, Braintree, MA), permits rapid assessment of clot formation, strength, and stability. This allows identification and guides treatment of specific deficits in the clotting cascade in order to reverse the effects of shock and

endothelial dysfunction while improving coagulation function [159–161].

Trauma-Induced Coagulopathy

The extent of coagulation abnormalities in trauma patients is a significant predictor of their prognosis. The pathophysiology of trauma-induced coagulopathy (TIC) differs from that of disseminated intravascular coagulation (DIC). Blood loss, localized consumption of coagulation factors, and hypoperfusion are contributors in TIC [162]. Hypoperfusion induces coagulopathy through activation of anticoagulation and fibrinolytic pathways [163, 164].

Hypothermia independently contributes to coagulopathy by causing platelet dysfunction, reduced factor activity, and initiating fibrinolysis [165]. Active rewarming with heated blankets, warmed IV fluids and products, and body cavity lavage can more rapidly correct hypothermia than the use of fabric or forced warm-air blankets.

Antifibrinolytics

Tranexamic acid (TXA) is a synthetic derivative of lysine, which competitively inhibits the activation of plasminogen to plasmin by binding to sites on each molecule and preventing hyperfibrinolysis. In a trauma coagulopathy model (using tissue plasminogen activator and tissue factor), TXA reversed hyperfibrinolysis and abnormal thromboelastogram findings induced by dilution with crystalloid, colloid, or HES [166].

TXA has been successfully used to minimize hemorrhage in a wide variety of operative settings. The randomized controlled CRASH-2 study of 20,000+ adult trauma patients found that administration of TXA reduced all-cause mortality and specifically reduced deaths due to bleeding [167]. A trial of TXA in patients with intracranial hemorrhage and traumatic brain injury was nested within CRASH-2. Investigators found no significant reduction in intracranial hemorrhage size, nor benefit or harm. Neither of these two studies found significant

increases in serious prothrombotic complications if TXA was administered within 3 h of injury [168]. A meta-analysis of the use of TXA in orthopedic surgery found significant reductions in blood loss and no increased risk of deep venous thrombosis [169].

TXA can be given orally in both the hospital and in damage control resuscitation in the field, and it has great potential to reduce postpartum hemorrhage worldwide [170]. The WOMAN Trial (World Maternal Antifibrinolytic Trial) is a double-blind placebo-controlled trial currently examining TXA for the treatment of postpartum hemorrhage [171]. There are as yet no specific reports of TXA treatment in pregnant trauma patients, although it has been used in cesarean section, postpartum hemorrhage, and metrorrhagia [172]. In a pregnant trauma patient, supplementation with tranexamic acid should be considered as part of efforts to reduce ongoing hemorrhage and coagulopathy.

Fibrinogen

Fibrinogen levels reach critically low levels (<100 mg/dL) before red cell transfusion is even necessary due to loss, dilution, increased breakdown, and insufficient synthesis. Small amounts of colloid administration (>1,000 mL) can impair fibrinogen polymerization [122]. There is evidence that fibrinogen supplementation helps manage trauma-induced coagulopathy. European recommendations call for fibrinogen repletion when levels reach 1.5–2.0 g/L [173]. Six units of FFP deliver roughly the same amount of fibrinogen as one bag of cryoprecipitate. As an alternative to cryoprecipitate, fibrinogen concentrate can be used and reconstituted with sterile water or saline.

Prothrombin Complex

The use of prothrombin complex concentrate (PCC) has also been examined in trauma resuscitation. PCC is a formula of vitamin K-dependent clotting factors (II, VII, IX, and X) that are essential for thrombin formation. Reduced

thrombin formation generally occurs when blood losses exceed 150–200 % of estimated blood volume [174]. Experimental and clinical data suggest that PCC can be helpful in reversing trauma-induced coagulopathy. One study demonstrated that using fibrinogen concentrates with PCC in a goal-directed manner that was guided by thromboelastographic measurements from ROTEM[®] made it possible to treat coagulopathy without using FFP [175]. Should this finding be replicated in larger and randomized studies, it will allow resuscitation to be undertaken without the time delay associated with cross-matching, thawing, and transfusing FFP [176].

Recombinant Factor VIIa

The use of recombinant factor VIIa (rFVIIa, NovoSeven[®]; NovoNordisk, Copenhagen, Denmark) was shown to be safe and effective in reducing the amount of blood transfused in blunt trauma patients [177]. However, this two-armed double-blinded randomized placebo-controlled trial failed to show statistically significant differences in transfusion requirements for pelvic fractures or penetrating injuries. rFVIIa has been used to stem massive postpartum hemorrhage, prevent cesarean hysterectomy, and treat disseminated intravascular coagulation in pregnant patients [178–181]. As yet there are no robust data or guidelines for its broad use in obstetric patients. Existing hypothermia, acidosis, hypofibrinogenemia, and thrombocytopenia should be corrected before rFVIIa use is considered. It should not be used to compensate for inadequate transfusion and factor therapy, but may have a role to play along with timely and targeted coagulation factors, fibrinogen, and systematic antifibrinolytics in trauma-induced coagulopathy [182].

Anti-shock Garments

Anti-shock garments, such as Military Anti-Shock Trousers (MAST) or Pneumatic Anti-

Shock Garment (PASG), are currently not recommended for massive antepartum hemorrhage due to concerns about restricting pelvic blood flow and uterine perfusion. If they are used in the undelivered pregnant patient, only the lower extremity portion should be inflated. They are considered a Class III intervention in non-pregnant patients and current indications are: (1) to splint and provide control of bleeding from pelvic fractures; and (2) to stabilize patients with intra-abdominal trauma and severe hypovolemia during transport [183]. Indications for using anti-shock devices include signs of severe hemorrhagic shock with systolic blood pressure <80 mmHg, unconsciousness, and absent or weak radial pulses after uterine displacement. Leg compartments should be inflated to 50 mmHg and the patient reassessed. Further inflation may be required if shock persists. Sequential deflation should occur only in a hospital setting after upper-extremity intravenous lines are secure and definitive management of the causes of hemorrhage has occurred.

Anti-shock garments can reduce uterine perfusion and increase cardiac workload, and they are poorly tolerated in patients with mitral stenosis, congestive heart failure, or pulmonary hypertension [4]. Use of MAST may delay transportation to trauma centers and worsen the outcomes of thoracic and abdominal injuries [183]. Anti-shock garments are relatively contraindicated in obstetric trauma, but in low resource setting they may be useful as adjuncts in controlling severe postpartum hemorrhage or in cases of ruptured ectopic pregnancies [184]. Non-pneumatic Anti-Shock Garments (NASG) may be used to stabilize hypovolemic shock in postpartum hemorrhage. Each light and washable neoprene device has three segments to cover each leg and one to cover the pelvis. A third segment is provided with a foam compression ball to cover the abdomen. Using Velcro[®] closures, the garment provides 20–40 mmHg circumferential counterpressure to shunt blood to core organs. Special training is not required for its use, and uterine and vaginal procedures can be performed with the NASG in place [185, 186]. They have been shown to reduce internal iliac

Complete maternal history and/or exam

- Mechanism of injury
- Medications/allergies
- Last menstrual period
- Presence or absence of contractions
- Abdominal pain/vaginal bleeding/rupture of membranes

Fetal Evaluation

- Fetal Movement
- Check fetal heart rate—continuous fetal heart rate after 23–24 weeks for minimum 4–6 hours
- Biophysical profile and/or middle cerebral artery Doppler

Focused Assessment with Sonography in Trauma (FAST)

Labs and Imaging

Fig. 16.5 Secondary survey in the pregnant trauma patient

blood flow, indicating a mechanism for stabilizing uterine hemorrhage when all three compartments are deployed [187].

Secondary Survey

After confirming the hemodynamic stability of the mother and fetus, the universal secondary survey should be performed, with particular attention given to pregnancy-related findings: fetal well-being and placental injury (see Fig. 16.5).

Information should be elicited about the mechanism of injury; for example, the type of weapon used (if any), the use of drugs or alcohol or the use of seatbelts in motor vehicle accidents. A past medical and obstetric history should include the last menstrual period, current or previous pregnancy complications, and estimated gestational age.

Using McDonald's rule for uterine growth, at approximately 23–24 weeks the fundus can be palpated at the umbilicus and an injury at this gestational age should prompt fetal monitoring which should be continued for at least 4–6 h [96]. Continuous fetal monitoring is more sensitive in detecting placental abruption than ultrasonography, Kleihauer-Betke testing, or physical examination. A decision to cease fetal monitoring should be made in consultation with obstetricians, and should take into account the presence of uterine contractions, fetal well-being, and plans for operative delivery.

Uterine monitoring should commence when gestational age is >20 weeks. Recurrent uterine

contractions with cervical change suggest pre-term labor. More than four contractions per hour may signal placental abruption.

The secondary survey should involve a sterile speculum exam to look for vaginal lacerations or bony fragments, which may indicate pelvic fracture. If fluid is present, its pH should be determined; pH 7.0 indicates amniotic fluid and pH 5.0 indicates normal vaginal secretions. Fluid can also be evaluated for nitrazine color-change, ferning, and the presence of fetal fibronectin. In addition, newer bedside tests for insulin-like growth factor-binding protein-1 (IGFBP-1) and placental alpha-microglobulin-1 (PAMG-1) can be considered; they may be more accurate in detecting membrane rupture [188, 189]. If vaginal bleeding is present in a second or third trimester trauma patient, a vaginal exam should be deferred until placenta previa can be excluded by ultrasound. The exam should be postponed until a double set-up for emergency cesarean section is available.

Maternal and Fetal Monitoring

Standard noninvasive monitoring of the pregnant trauma patient includes pulse oximetry, electrocardiography, blood pressure monitoring, temperature measurement, together with monitoring of the fetal heart rate and uterine tocodynamometry when necessary. An indwelling urinary catheter should be inserted to measure hourly urine output. Invasive arterial monitoring is indicated if there is persistent hypotension,

hypoxia, or labile blood pressure; it provides a means for periodic arterial blood sampling and gas analysis. Pulmonary artery catheters are currently used less frequently in general critical care patients. In obstetric patients, they are more commonly used in those with pulmonary edema, known severe mitral or aortic stenosis, NYHA class III–IV disease in labor, intrapartum or intraoperative cardiac failure, shock or adult respiratory distress syndrome. In patients who are intubated, tidal volume, airway pressure, and end-tidal carbon dioxide should be monitored. If a volatile anesthetic agent is used, the end-tidal concentration should be observed. Monitoring of cardiac function through transesophageal echocardiography may also be useful.

The fetal heart rate tracing should be recorded in all pregnancies above 20 weeks and recorded continuously at viability. Fetal heart rate tracings which indicate distress (i.e., Category II or Category III [190]), or a low biophysical profile score should prompt suspicion of maternal hypovolemia, placental abruption, or fetomaternal hemorrhage.

Laboratory Tests

Initial laboratory tests in the pregnant trauma patient should include: complete blood count, basic metabolic panel with electrolytes and glucose, type and crossmatch, Rh status, coagulation profile, fibrinogen, liver function tests, blood lactate, toxicology screen, Kleihauer-Betke (KB) test, urinary protein, blood, bilirubin and glucose and urine osmolality or specific gravity. An arterial blood gas should be evaluated if respiratory function is compromised. All laboratory values should be measured against “normal” parameters for pregnant patients. Care must be taken in interpreting the results; for example, low platelets could be a sign of hypertensive diseases of pregnancy (see Fig. 16.6).

In the pregnant trauma patient, Rh typing is necessary. As little as 0.01 mL of fetal blood can cause sensitization in the Rh-negative mother [191]. The KB test can be used to quantify fetal

hemoglobin in the maternal circulation. A positive KB test (>0.01 mL of fetal RBC) has been associated with significant fetomaternal hemorrhage and preterm labor. All Rh-negative women should be treated with Rh-immune globulin within 72 h (300 μ g initially and an additional 300 μ g for each 30 mL of estimated fetomaternal transfusion). A positive KB test should be repeated in 24–48 h to investigate ongoing hemorrhage. In the future, anti-fetal hemoglobin (anti-HbF) flow cytometry may prove to be a more reliable and easily standardized test [192, 193].

While the utility of KB testing in Rh-positive patients has been questioned, it has been found to be a reliable independent predictor of preterm labor after trauma and the test should be obtained in all patients. Fetal middle cerebral artery Doppler testing may be considered when significant fetomaternal hemorrhage is suspected [194]. Fetal anemia can be rapidly detected and treated in cases where immediate delivery is not anticipated and lethal fetal hydrops prevented.

Imaging

The ATLS recommends radiographs of the cervical spine, chest, and pelvis. Concern about effects of ionizing radiation should not prevent medically indicated maternal X-rays. During the period of organogenesis (4–10 weeks), ionizing radiation is most likely to cause congenital malformations. A fetus is most susceptible to radiation-induced developmental delay from 10 to 17 weeks. Non-cancer fetal injuries diminish with increasing gestational age. Theoretical risks associated with radiation exposure at any time during pregnancy include an increased incidence of childhood leukemia (absolute risk $\sim 1:2,000$). Exposure to less than 5 rad (50 mGy), however, has not been associated with an increase in fetal anomalies or pregnancy loss and this is deemed a safe level throughout the gestation.

If multiple diagnostic studies are performed, consultation with a radiologist should be considered for calculating the estimated fetal exposure. The uterus should be shielded as much as possible and using a posterior-anterior exposure can

Fig. 16.6 Initial laboratory evaluation of the pregnant trauma patient

- Blood type, cross-match, Rh status
- Complete blood count (hemoglobin, white blood cell count, platelet count)
- Coagulation profile (prothrombin and partial thromboplastin time)
- Fibrinogen concentration and fibrinogen split products (D-dimer)
- Serum electrolytes
- Serum glucose level
- Liver function tests
- Serum amylase
- Serum lactate and urate
- Toxicology screen
- Arterial blood gas (pH, PaO₂, PaCO₂, bicarbonate, base deficit)
- Kleihauer–Betke test
- Urinary protein, blood, bilirubin, and glucose
- Urine specific gravity or urine osmolality

increase the distance from the anterior uterus to the radiation source [195]. If computed tomography is necessary, it can be performed with fewer slices, reduced current, or increased pitch. The interventional radiologist can use several techniques to minimize fluoroscopy time, decrease the fluoroscopy frame rates, and minimize image magnification [196]. Lead shields and internal shielding (with barium) should be used as much as possible [197]. Magnetic resonance imaging (MRI) and ultrasonography during pregnancy have not been associated with any adverse effects and there is no evidence of teratogenicity with gadolinium, a paramagnetic ion administered for contrast definition in MRIs [198].

Focused abdominal sonography for trauma (FAST) can be useful in patients who have experienced blunt trauma. FAST provides evidence of free fluid in four areas: the subxiphoid, the right and left upper quadrants, and the suprapubic area. In pregnant patients, it has a sensitivity of 80–83 % and specificity of 98–100 % in detecting intraperitoneal fluid [199, 200]. It can also be used to establish the diagnosis of an unknown pregnancy [201]. FAST can often reduce the need for multiple radiographic imaging studies, but it is of limited use in detecting maternal injuries such as arterial hemorrhage [202].

Bedside ultrasound and rapid computed tomography scans have largely rendered diagnostic peritoneal lavage unnecessary. In resource-limited settings, however, these tests can be performed safely using a supra-umbilical open technique [203, 204].

Specific Management Issues

Traumatic Brain Injury

As indicated above, resuscitation with crystalloids instead of colloids is preferable in traumatic brain injury. Specific techniques to elevate the head and minimize head and neck flexion encourage venous drainage and decrease elevated intracranial pressure. In pregnant trauma patients, hypoventilation should be avoided because it can decrease uterine blood flow by decreasing maternal cardiac output and blood pressure and by causing uterine vasoconstriction. PaCO₂ should be maintained within the normal range for pregnancy with a baroprotective ventilation strategy. Aggressive resuscitation to prevent hypotension and hypoxia is necessary to maintain perfusion of the brain and other vital organs.

Spinal Cord Injuries

Traumatic injuries to the spinal cord have implications for pregnant patients. Of the 12,000 women of childbearing age who sustain spinal cord injuries per year, 2,000 become pregnant in any given year [205]. Roughly 14 % of these women will have at least one pregnancy after being injured [206]. Should the spinal injury occur above the T5–T6 level, the patient is at risk of developing autonomic dysreflexia (AD) or hyperreflexia. In patients with this condition, noxious stimuli below the level of injury result

in unopposed sympathetic activity, piloerection, vasoconstriction, and pallor below the level of injury. Above the level of injury, unopposed parasympathetic activity can cause flushing, sweating, pupillary constriction, and nasal congestion. Without treatment, autonomic hyperflexia can lead to seizures, retinal hemorrhage, pulmonary edema, renal insufficiency, myocardial infarction, cerebral hemorrhage, and death [207].

A review of cases of spinal cord injury in pregnancy found rates for vaginal delivery, assisted vaginal delivery, and cesarean section were 37 %, 31 %, and 32 %, respectively [208]. Invasive hemodynamic monitoring may be indicated in these patients [209]. Initial management of autonomic dysreflexia includes elevating the head of the bed, loosening tight clothing, emptying bowel and rectum, and eliminating any triggering stimulus if possible [210]. Rapidly acting vasodilators, such as sublingual nitrates, oral clonidine, or topical nitropaste can be used in an outpatient setting and the medications can be changed to intravenous vasodilators or ganglionic blockers in an intensive care unit setting [211].

In labor and delivery, the pain of uterine contractions can be a stimulant for AD, which can be attenuated by administration of spinal, or epidural anesthesia. Further blood pressure management can be achieved with sodium nitroprusside or trinitroglycerin as needed [212, 213]. In most patients, confirmation of spinal anesthesia can be confirmed by the absence of a Babinski sign and the patellar tendon reflex and the loss of spasticity, although determining the exact level of block can be difficult [213]. Finally, additional care must be taken to prevent ascending urinary tract infections and thromboembolic events in pregnant women with spinal cord injuries [214].

Respiratory Failure and Extracorporeal Lung Support

Thoracic trauma and massive transfusion are independent risk factors for acute lung injury that may be refractory to conventional therapy. Approximately 4.6 % of all trauma patients develop adult respiratory distress syndrome

[154]. The use of extracorporeal membrane oxygenation (ECMO) in acute lung injury remains controversial, and data on its benefits compared with conventional treatment are limited. The use of ECMO in trauma is hindered by concerns over hemorrhage during cannulation in the presence of trauma-induced coagulopathy, contraindications to anticoagulation, decreased venous return as a result of abdominal packing in damage-control surgery, and risk of iatrogenic intracranial hemorrhage [215].

A retrospective analysis of ten non-pregnant trauma patients who were treated with either high-flow ECMO or interventional lung assist in one center showed survival in six out of ten patients, indicating that extracorporeal gas exchange can be considered as rescue therapy in adult trauma patients. In another 10-year retrospective analysis of chest trauma patients treated with ECMO, the overall survival rate was 79 % [216].

There is only one case report of successful ECMO therapy in a pregnant trauma patient who developed ARDS [217]. However, pumpless and pump-driven extracorporeal lung support systems have been used to treat pregnant women with ARDS due to influenza and pneumonia, with successful maternal and neonatal outcomes [218–221]. ECMO has also been employed in cases of massive thromboembolism/amniotic fluid embolism and peripartum cardiomyopathy [222–224]. Recognizing the need to maintain aortic blood flow during medical procedures, a technique for venous ECMO cannulation has been described for left uterine displacement during late pregnancy [225]. Extracorporeal lung support should be considered as salvage therapy in pregnant patients with severe thoracic trauma and acute lung injury.

Analgesia

All pregnant trauma patients should receive adequate analgesia. Pain causes high levels of circulating catecholamines, which can reduce placental blood flow. Opioids cause a reduction in fetal heart rate variability and the obstetrician

and neonatal team should be informed whenever they are given. Remifentanyl, an ultra short-acting synthetic opioid rapidly metabolized by nonspecific plasma and tissue esterases, can be used if imminent delivery is suspected and avoidance of neonatal respiratory depression deemed is a priority.

Nonsteroidal anti-inflammatory medications should generally be avoided because of their effects on platelet and renal function. They are also relatively contraindicated late in gestation because of risk of fetal ductus arteriosus closure. Intravenous acetaminophen may be used to minimize total opiate doses. Regional anesthetic techniques can be used in cases where there are no coagulation abnormalities.

Thromboprophylaxis

Trauma patients in general are at risk of thrombotic events. Orthopedic injuries and immobility independently contribute to elevated risk for blood clots. Recent research indicates patients develop a hypercoagulable state 48 h after blunt injury to abdominal organs and that fibrinogen plays an important role in clot strength [226, 227]. Specific venous thromboembolic prophylaxis regimens have not been established in non-pregnant trauma patients and routine thromboprophylaxis after trauma remains debated. Coagulation changes of pregnancy, however, predispose women to thromboembolism even without injury. All pregnant trauma patients should receive thromboembolic prophylaxis after hemostasis is obtained. Unfractionated heparins and low molecular weight heparin are too large to cross the placenta and are safe for the fetus.

Antibiotic and Tetanus Prophylaxis

All patients with traumatic injuries should receive antibiotic prophylaxis. If emergency laparotomy is necessary, antibiotics should cover streptococcal, staphylococcal, clostridial, and polymicrobial infections [20]. If massive transfusion is required, care must be taken to

administer antibiotics at intervals that are sufficient to maintain adequate tissue levels.

In addition to antibiotics, patients who have tetanus-prone wounds should be given 0.5 mL of tetanus toxoid if they have not received a booster dose within the past 5 years. If they have never been immunized with tetanus toxoid and have a high-risk injury, they should receive an additional 500 units of tetanus immunoglobulin intramuscularly [30]. A tetanus-prone wound is an injury or burn that requires surgical intervention due to a treatment delay greater than 6 h. Injuries with a significant degree of devitalized tissue, puncture-type injuries (particularly when contaminated with soil or manure, as might be found with farm equipment), and animal bites are also at risk. Wounds containing foreign bodies, compound fractures, and injuries in patients who have systemic sepsis are prone to tetanus infection.

Pregnancy-Related Management Issues

Hypertensive disease in pregnancy is typically marked by elevated blood pressure and proteinuria. A patient with preeclampsia may have a normal or an unexpectedly elevated blood pressure in the setting of significant blood losses related to injury. In a pregnant woman with trauma, a near normal blood pressure in association with proteinuria, abnormal liver enzymes, elevated serum uric acid and otherwise unexplained thrombocytopenia should suggest the possibility of underlying preeclampsia. Seizures after traumatic brain injury in a pregnant patient with the same findings should prompt consideration of eclampsia, and intravenous magnesium sulfate therapy should be initiated. In trauma patients with suspected preeclampsia, blood pressure should be supported at a level that maintains adequate uterine perfusion and relative hypotension should be avoided.

Fetal Delivery

Delivery of the fetus may be required in the setting of placental abruption, uterine rupture,

maternal shock, or fetal intolerance to maternal trauma surgery. The fetal heart rate is a sensitive indicator of placental perfusion and a fetal heart rate tracing that is not reassuring (i.e., Category II or Category III) or does not recover with maternal resuscitation and uterine displacement should prompt immediate delivery. Continued maternal instability in the face of ongoing resuscitation or cardiac arrest is also an indication for fetal delivery. Relief of aortocaval compression by delivery will increase cardiac output by approximately 60–80 %, decrease oxygen requirements, improve ventilation, and make cardiopulmonary resuscitation more effective [228].

Cardiac Arrest and Perimortem Cesarean Delivery

The global incidence of maternal cardiac arrest is unknown due to lack of reliable reporting and differences in standards of perinatal care. Maternal cardiac arrest is rare, but it appears to have increased in incidence from 2.20 to 2.37 per 100,000 maternities according to the most recent data from the United Kingdom's Confidential Enquiry into Maternal Deaths [228]. Following cardiac arrest, the American Heart Association (AHA) recommends urgent operative delivery within 4 min. Perimortem cesarean section is defined as cesarean delivery initiated after maternal arrest [230].

In developing countries, hemorrhage and sepsis are the most frequent contributors to maternal cardiac arrest and death. Cardiac disease is now the most common cause of maternal arrest in developed countries, exceeding hemorrhage, thromboembolism, and sepsis. Direct causes of cardiac arrest include eclampsia, hemorrhage, thromboembolism, and amniotic fluid embolism. Indirect causes include underlying cardiac disease, sepsis, malignancy, and trauma. Anesthetic causes such as airway failure and local anesthetic toxicity may be contributing factors.

Resuscitation efforts during pregnancy should take into account the physiologic changes of pregnancy (see Fig. 16.7) [231]. Because aortocaval compression reduces cardiac output,

thoracic compressions need to be given with the uterus displaced 15–30°. Wedges, such as the Cardiff Resuscitation Wedge, have been designed to produce an adequate degree of tilt, but they are seldom available [232]. Manual displacement of the uterus may be superior to tilting the entire patient or raising the right hip to achieve left uterine displacement, and can be easily accomplished [233–236]. The force of compressions needs to be increased when the patient is no longer supine. When the body is tilted at 27° for uterine displacement, the force of adequate chest compressions is reduced to 80 % of that when the patient is flat. Chest compressions should be performed 2–3 fingers above the xiphoid in the midsternum to avoid injury to the uterine fundus, liver, or spleen.

Paddle placement for defibrillation must account for enlarged breasts, but thoracic impedance remains unchanged and normal defibrillator current settings can be used [237]. Similarly, standard doses of ACLS drugs should be used; the benefits of restoring maternal circulation outweigh the risk of uteroplacental vasoconstriction. However, the volume of drug distribution and drug metabolism differs in pregnancy, and higher doses should be considered if standard doses do not yield an adequate response [238].

In cases of cardiac arrest, cesarean delivery should be considered when the fetus is estimated to be beyond 20 weeks gestation and the uterus is palpable at the umbilicus. It is vital that CPR be continued during cesarean section. Fetal delivery during maternal arrest has been shown to improve overall maternal and fetal outcomes. Maternal and fetal survival rates have been reported to be 72 % and 45 %, respectively, in cases of non-traumatic maternal cardiac arrest [239]. A review of maternal cardiac arrests noted that 12 out of 20 mothers who experienced cardiac arrest had improved hemodynamics or a return to spontaneous circulation following urgent cesarean section [240]. Evacuation of the uterus relieves aortocaval compression, provides autotransfusion of the uterine blood, decreases maternal metabolic requirements, and improves ventilation.

Prompt delivery of the fetus increases the likelihood of intact neurologic function in

- Chest compressions
 - Hand placement mid–sternum, 2–3 finger–breadths higher
 - Uterus displaced after 18–20 weeks with wedge, manual displacement, or tilted backboard
- Defibrillation
 - Paddle placement unchanged
 - Current unchanged
- Drugs
 - Doses unchanged
- Delivery
 - Plan for perimortem cesarean delivery within 4 minutes

Fig. 16.7 Cardiopulmonary resuscitation

neonates [241, 242]. Data from patients who experienced cardiac arrest after amniotic fluid embolism show that 98 % of fetuses had intact neurologic function if delivered within 5 min, 83 % had intact neurologic function if delivered within 6–15 min, but none had neurologic function if delivered 36+ min after maternal amniotic fluid embolism [243]. Unfortunately, in cases of traumatic hypovolemic cardiac arrest, fetal outcomes are likely to be worse because the fetus has already suffered prolonged hypoxia prior to the maternal arrest.

An analysis of case reports of maternal cardiac arrest indicates that a 4-min time frame for emergency hysterotomy was not met in 93 % of cases, yet the neonatal survival rate was still 50 %, and this included cases in which the fetus was delivered 10 min after the arrest began [244]. This analysis also indicated that cesarean delivery during maternal arrest provided clear improvement in maternal hemodynamics in only 31.7 % of cases, which may reflect prolonged arrest times prior to initiation of cesarean section. Currently, one-third of women who die during pregnancy remain undelivered at time of death. It is unclear whether cesarean section during maternal arrest might increase the number of viable fetuses who would otherwise have remained undelivered [245].

Where cesarean delivery during arrest should be performed is subject to debate. In a mannequin-based study of simulated maternal arrest, maternal transport impaired the quality of the resuscitation [246]. During cardiac arrest, hemorrhage is minimal and definitive surgical hemostasis and antibiotic therapy can be

completed in the operating room after spontaneous circulation is restored. If resources are available emergency cesarean sections should be performed where the arrest occurs.

In cases of maternal arrest with return to spontaneous circulation, therapeutic hypothermia may be considered if coagulation parameters are normal. The AHA recommends considering therapeutic hypothermia in the undelivered patient with continuous fetal monitoring [116]. Occasionally resuscitation is successful, but the patient has irreversible brain damage and remains undelivered. Several case reports show that such patients may deliver viable fetuses even though they sustained their injuries as early as the 15th week of pregnancy [247–249].

Anesthetic Management

The anesthetic care of the pregnant trauma patient combines the principles of trauma resuscitation with the anesthetic management of pregnant women undergoing non-obstetric surgery. Uteroplacental perfusion and maternal hemodynamics must be maintained in order to optimize maternal and fetal outcomes. The specific anesthesia techniques employed will depend on the nature of the patient's injuries.

Induction and Intubation

All pregnant trauma patients should receive anti-acid prophylaxis if possible prior to intubation.

After denitrogenation with 100 % oxygen, a rapid sequence induction with cricoid pressure is preferred, if conditions permit. Airway management in pregnancy and trauma poses increased risks and additional airway equipment should be available. Pregnant patients >18–20 weeks gestation should be placed in left uterine displacement to prevent aortocaval compression and optimize placental perfusion.

Most drugs for induction are considered safe for use in pregnant trauma patients. The choice of induction agents includes etomidate or ketamine. In hypotensive patients with ongoing hemorrhage, they provide better blood pressure support than thiopental or propofol. Ketamine should be avoided in patients with traumatic head injuries because it may increase intracranial pressure. It can also cause myocardial depression in patients with severe hypovolemia and in large doses it has caused increased uterine tone in pregnant ewes [250]. Opiates such as remifentanyl or fentanyl can be used to supplement the induction agent. Neuromuscular blockade can be achieved with succinylcholine or rocuronium. Once endotracheal intubation has been achieved, an oro- or nasogastric tube should be passed to decompress the stomach. When muscle tone has recovered, muscle relaxation can be re-initiated and maintained with a nondepolarizing muscle relaxant guided by peripheral nerve monitoring.

Maintenance of Anesthesia

A balanced anesthetic technique using a volatile agent, opioids, and neuromuscular blockade is favored to maintain maternal hemodynamics during trauma surgery. If a volatile anesthetic is contraindicated because of hypotension, an opiate such as fentanyl and an antianxiolytic agent should be used until a volatile agent can be safely administered. Nitrous oxide should be limited in trauma cases in which there is a possibility of pneumothorax. In its absence, nitrous oxide can be considered to help minimize the use of volatile agents in postpartum patients, thus reducing the risk of uterine atony and ongoing hemorrhage.

If the fetus is between 20 and 23 weeks fetal heart rate should be evaluated prior to induction and after surgery is completed. If the fetus greater than 23 weeks, intraoperative fetal monitoring can detect fetal distress that might indicate a need for urgent cesarean section. Continuous fetal monitoring is contingent on adequate facilities, the availability of trained personnel to interpret the heart rate tracing in the operating room, and an obstetrical team that is immediately ready for cesarean delivery.

If a cesarean delivery is performed, uterotonic agents should be immediately available. An oxytocin infusion should be started after the fetus is delivered. A slow intravenous drip is preferred to a bolus dose because rapid infusion can cause vasodilation and hypotension. In order to minimize ongoing bleeding, high-concentrations of volatile anesthetic agents should be avoided. Additional uterotonic agents such as prostaglandin E1, prostaglandin F2 alpha, and methyl ergonovine may be used if oxytocin alone proves insufficient.

Intraoperative fluid therapy should be guided by the preoperative fluid status, preoperative and intraoperative blood losses, maternal hemodynamics, urine output, and additional information from transesophageal echocardiography (TEE), or central venous pressure monitoring. Early treatment of hemorrhage and coagulopathy is vital. If massive hemorrhage occurs, intraoperative red blood cell salvage can be achieved with a Cell Saver[®] (Haemonetics Corporation, Braintree, Massachusetts), with care to avoid collection of amniotic fluid. A rapid infuser should be used to assist transfusion management.

Conclusion

The global burden of trauma mortality is greater than the mortality burden attributed to HIV/AIDS, malaria, and tuberculosis combined. In the United States, maternal mortality from traumatic injury exceeds direct pregnancy-related causes of death.

Motor vehicle accidents and intimate partner violence remain leading causes of maternal trauma. Providing information on

- Transport and stabilize in trauma center
 - protect brain and spinal cord
- Monitor maternal vital signs and fetal heart rate
- Restore maternal tissue perfusion and oxygenation
 - resuscitate with FFP and RBC transfusion in ratio > 1:2
- Prevent and treat coagulopathy
 - prevent hypothermia
 - early use of antifibrinolytics, fibrinogen concentrate, prothrombin complex, rFactorVIIa
- Optimize uteroplacental blood flow and fetal well-being
 - uterine displacement
 - supplemental oxygen
- Prevent preterm labor
 - tocolysis
- Prepare for emergency cesarean delivery
 - Fetal deterioration with gestation > 23–24 weeks
 - maternal deterioration with fetus > 23–24 weeks
 - maternal arrest

Fig. 16.8 Management strategies for pregnant trauma patients

seatbelts during prenatal visits can help prevent injury related to absent or improper seat belt use. Similarly, routine questions about substance and alcohol use, and exposure to intimate partner violence can enable appropriate referral and assistance.

Maternal and fetal outcomes of trauma are often poor and fetal outcomes do not correspond with severity of maternal injury. All women of childbearing age who experience trauma should be evaluated for possible pregnancy. Pregnant women who are discharged following their initial injury should be considered high-risk patients for the remainder of their pregnancy.

Pregnancy involves significant alterations in maternal physiology that directly influence the evaluation and management of trauma (see Fig. 16.8). Cardiovascular and hematologic changes clinically compensate for hemorrhage, but can delay recognition of the extent of injury. Airway, cardiovascular, and respiratory changes affect the use of ATLS protocols. Uterine displacement to relieve aortocaval compression must be established for all patients with pregnancies of 18–20 weeks or greater.

Fluid resuscitation and blood transfusion should be guided by maternal needs and blood pressure supported at levels that optimize fetal well-being. All pregnant patients who require emergency transfusion should receive O-negative/Rh-negative blood to avoid Rho sensitization. Trauma-induced coagulopathies should be aggressively treated and may require use of tranexamic acid, fibrinogen, or prothrombin complex concentrate.

All pregnant women with trauma should be evaluated for fetomaternal hemorrhage. Rh immune globulin should be administered if fetomaternal hemorrhage is suspected. The fetal heart rate should be recorded for all pregnancies above 20 weeks. Continuous fetal heart rate monitoring should be initiated for pregnancies greater than 23–24 weeks and it should be maintained for at least 6 h during the trauma hospitalization.

If pregnant women require diagnostic imaging, care must be taken to shield the uterus. Following trauma, they should also receive thromboprophylaxis, antibiotics, and tetanus prophylaxis when indicated.

If a pregnant trauma patient suffers a cardiac arrest after 20 weeks gestation, chest

compressions must be performed with uterine displacement. As soon as the arrest occurs, a cesarean section for fetuses >23–24 weeks should be initiated within 4 min. Cesarean delivery of nonviable or dead fetuses should also be considered as it can improve maternal hemodynamics and prompt return to spontaneous circulation.

Anesthesiologists may have more knowledge of the pregnant patient than other members of a trauma team. As such, they can play a critical role in integrating their understanding of the physiologic changes of pregnancy with the life support and trauma protocols needed to reduce morbidity and mortality in pregnant women who experience trauma.

References

- Mirza FG, Devine PC, Gaddipati S. Trauma in pregnancy: a systematic approach. *Am J Perinatol.* 2010;22(7):579–86.
- Pearlman MD. Motor vehicle crashes, pregnancy loss and preterm labor. *Int J Gynaecol Obstet.* 1997;57(2):127–32.
- Ikossi DG, Lazar AA, Morabito D, et al. Profile of mothers at risk: an analysis of injury and pregnancy loss in 1,195 trauma patients. *J Am Coll Surg.* 2005;200:49–56.
- Mattox KL, Goetzl L. Trauma in pregnancy. *Crit Care Med.* 2005;33(Suppl):S385–9.
- El Kady D. Perinatal outcomes of traumatic injuries during pregnancy. *Clin Obstet Gynecol.* 2007;50:582–91.
- Chames MC, Pearlman MD. Trauma during pregnancy: outcomes and clinical management. *Clin Obstet Gynecol.* 2008;51:398–408.
- Aboutanos SB, Aboutanos MG, Dompkowski D, et al. Predictors of fetal outcome in pregnant trauma patients: a five-year institutional review. *Am Surg.* 2007;73:824–7.
- Horon IL. Underreporting of maternal deaths on death certificates and the magnitude of the problem of maternal mortality. *Am J Public Health.* 2005;95:478–82.
- Horon IL, Cheng D. Effectiveness of pregnancy check boxes on death certificates in identifying pregnancy-associated mortality. *Public Health Rep.* 2011;126:195–200.
- Fildes J, Reed L, Jones N, et al. Trauma: the leading cause of maternal death. *J Trauma.* 1992;32:643–5.
- Harper M, Parsons L. Maternal deaths due to homicide and other injuries in North Carolina: 1992–1994. *Obstet Gynecol.* 1997;90:920–3.
- Dannenber AL, Carter DM, Lawson HW, et al. Homicide and other injuries as causes of maternal death in New York City, 1987 through 1991. *Am J Obstet Gynecol.* 1995;172:1557–64.
- Pearlman MD, Tintinalli JE, Lorenz RP. A prospective controlled study of outcome after trauma during pregnancy. *Am J Obstet Gynecol.* 1990;162:1502–7.
- Schiff MA, Holt VL, Daling JR. Maternal and infant outcomes after injury during pregnancy in Washington state from 1989 to 1997. *J Trauma.* 2002;53:939–45.
- Pak LL, Reece EA, Chan L. Is adverse pregnancy outcome predictable after blunt abdominal trauma? *Am J Obstet Gynecol.* 1998;179:1140–4.
- El-Kady D, Gilbert WM, Anderson J, et al. Trauma during pregnancy: an analysis of maternal and fetal outcomes in a large population. *Am J Obstet Gynecol.* 2004;190:1661–8.
- Schiff MA, Holt VL. Pregnancy outcomes following hospitalization for motor vehicle crashes in Washington state from 1989 to 2001. *Am J Epidemiol.* 2005;161:503–10.
- Weiss HB, Songer TJ, Fabio A. Fetal deaths related to maternal injury. *JAMA.* 2001;286:1863–8.
- Kuo C, Jamieson DJ, McPheeters ML, et al. Injury hospitalizations of pregnant women in the United States, 2002. *Am J Obstet Gynecol.* 2007;196:161.e1–6.
- Hill C. Trauma in the obstetrical patient. *Womens Health.* 2009;5:269–85.
- Hyde LK, Cook LJ, Olson LM, et al. The effect of motor vehicle crashes on adverse fetal outcomes. *Obstet Gynecol.* 2003;102:279–86.
- Patteson SK, Snider CC, Meyer DS, et al. The consequence of high-risk behaviors: trauma during pregnancy. *J Trauma.* 2007;62:1015–20.
- Gazmararian JA, Lazorick S, Spitz AM, et al. Prevalence of violence against pregnant women. *JAMA.* 1996;275:1915–20.
- Tinker SC, Reefhuis J, Dellinger AM, et al. National Birth Defects Prevention Study. Epidemiology of maternal injuries during pregnancy in a population-based study, 1997–2005. *J Womens Health (Larchmont).* 2010;19:2211–8.
- Fischer PE, Zarzaur BL, Fabian TC, et al. Minor trauma is an unrecognized contributor to poor fetal outcomes: a population-based study of 78,552 pregnancies. *J Trauma.* 2011;71:90–3.
- Sperry JL, Casey BM, McIntyre DD, et al. Long-term fetal outcomes in pregnant trauma patients. *Am J Surg.* 2006;192:715–21.
- Schiff MA, Holt VL. The injury severity score in pregnant trauma patients: predicting placental abruption and fetal death. *J Trauma.* 2002;53:946–9.

28. Biester EM, Tomich PG, Esposito TJ, et al. Trauma in pregnancy: normal revised trauma score in relation to other markers of maternofetal status—a preliminary study. *Obstet Gynecol.* 1997;176:1206–12.
29. Baerga-Varela Y, Zietlow SP, Bannon MP, et al. Trauma in pregnancy. *Mayo Clin Proc.* 2000;75:1243–8.
30. Tweddle CJ. Trauma during pregnancy. *Crit Care Nurs Q.* 2006;29:53–67.
31. Shah KH, Simons RK, Holbrook T, et al. Trauma in pregnancy: maternal and fetal outcomes. *J Trauma.* 1998;45:83–6.
32. American College of Obstetricians and Gynecologists. ACOG educational bulletin Obstetric aspects of trauma management. Number 251, September 1998 (replaces Number 151, January 1991, and Number 161, November 1991). *Int J Gynaecol Obstet.* 1999;64:87–94.
33. Romero VC, Pearlman M. Mortality due to trauma. *Semin Perinatol.* 2012;36:60–7.
34. Petrone P, Talving P, Browder T, et al. Abdominal injuries in pregnancy: a 155-month study at two level I trauma centers. *Injury.* 2011;42:47–9.
35. Theodorou DA, Velmahos GC, Souter I, et al. Fetal death after trauma in pregnancy. *Am Surg.* 2000;66:809–12.
36. Leggon RE, Wood GC, Indeck MC. Pelvic fractures in pregnancy: factors influencing maternal and fetal outcomes. *J Trauma.* 2002;53:796–804.
37. Aboutanos MB, Aboutanos SZ, Dompkowski D, et al. Significance of motor vehicle crashes and pelvic injury on fetal mortality: a five year institutional review. *J Trauma.* 2008;65:616–20.
38. Cannada LK, Pan P, Casey BM, et al. Pregnancy outcomes after orthopedic trauma. *J Trauma.* 2010;69:694–8.
39. El Kady D, Gilbert WM, Xing G, et al. Association of maternal fractures with adverse perinatal outcomes. *Am J Obstet Gynecol.* 2006;195:711–6.
40. Lavery JP, Staten-McCormick M. Management of moderate to severe trauma in pregnancy. *Obstet Gynecol Clin North Am.* 1995;22:69–90.
41. Tinker SC, Reefhuis J, Dellinger AM, et al. Epidemiology of maternal injuries during pregnancy in a population-based study, 1997–2005. *J Womens Health.* 2010;19:2211–8.
42. Kvarnstrand L, Milsom I, Lekander T, et al. Maternal fatalities, fetal and neonatal deaths related to motor vehicle crashes during pregnancy: a national population-based study. *Acta Obstet Gynecol Scand.* 2008;87:946–52.
43. Pearlman MD, Philips ME. Safety belt use during pregnancy. *Obstet Gynecol.* 1996;88:1026–9.
44. Schiff M, Albers L, McFeely P. Motor vehicle crashes and maternal mortality in New Mexico: the significance of seat belt use. *West J Med.* 1997;167:19–22.
45. Motozawa Y, Hitosugi M, Abe T, Tokudome S. Effects of seat belts worn by pregnant drivers during low-impact collisions. *Am J Obstet Gynecol.* 2010;203:62.e1–8.
46. Metz T, Abbott JT. Pregnancy after motor vehicle crashes with airbag deployment: a 30-case series. *J Trauma.* 2006;61:658–61.
47. Sirin H, Weiss HB, Sauber-Schatz EK, et al. Seat belt use, counseling and motor-vehicle injury during pregnancy: results from a multi-state population-based survey. *Matern Child Health J.* 2007;11:505–10.
48. Klinich KD, Flannagan CAC, Rupp JD, et al. Fetal outcome in motor-vehicle crashes: effect of crash characteristics and maternal restraint. *Am J Obstet Gynecol.* 2008;198:450.e1–9.
49. Astarita DC, Feldman B. Seat belt placement resulting in uterine rupture. *J Trauma.* 1997;42:738–40.
50. Fusco A, Kelly K, Winslow J. Uterine rupture in a motor vehicle crash with airbag deployment. *J Trauma.* 2001;51:1192–4.
51. Schultze PM, Stamm CA, Roget J. Placental abruption and fetal death with airbag deployment in a motor vehicle accident. *Obstet Gynecol.* 1998;92(Pt 2):719.
52. Schiff MA, Mack CD, Kaufman RP, et al. The effect of airbags on pregnancy outcomes in Washington State. *Obstet Gynecol.* 2010;115:85–92.
53. Mendez-Figueroa H, Dahlke JD, Vrees RA, et al. Trauma in pregnancy: an updated systematic review. *Am J Obstet Gynecol.* 2013;209:1–10.
54. Kady E, Gilbert WM, Xing G, et al. Maternal and neonatal outcomes of assaults during pregnancy. *Obstet Gynecol.* 2005;105:357–63.
55. Connolly AM, Katz VL, Bash KL, et al. Trauma and pregnancy. *Am J Perinatol.* 1997;14:331–6.
56. Schiff MA. Pregnancy outcomes following hospitalization for a fall in Washington state from 1987 to 2004. *BJOG.* 2008;115:1648–54.
57. Dunning K, Lemasters G, Bhattacharya A. A major public health issue: the high incidence of falls during pregnancy. *Matern Child Health J.* 2010;14:720–5.
58. Lymberly JK, Gilleard W. The stance phase of walking during late pregnancy; temporospatial and ground reaction force variables. *J Am Podiatr Med Assoc.* 2005;95:247–53.
59. Butler EE, Colon I, Druzing ML, et al. Postural equilibrium during pregnancy: decreased stability with an increased reliance on visual cues. *Am J Obstet Gynecol.* 2006;195:1104–8.
60. Fries EC, Hellebrandt FA. The influence of pregnancy on the location of the center of gravity, postural stability, and body alignment. *Am J Obstet Gynecol.* 1943;46:374–80.
61. McCrory JL, Chambers AJ, Daftary A, Redfern MS. Dynamic postural stability during advancing pregnancy. *J Biomech.* 2010;43:2434–9.
62. Palladino CL, Singh V, Campbell J, et al. Homicide and suicide during the perinatal period: findings from the National Violent Death Reporting System. *Obstet Gynecol.* 2011;118:1056–63.

63. Shadigian E, Bauer ST. Pregnancy-associated death: a qualitative systematic review of homicide and suicide. *Obstet Gynecol Surv.* 2005;60(3):183–90.
64. Krulewicz CJ, Pierre-Louis ML, de Leon-Gomez R, et al. Hidden from view: violent deaths among pregnant women in the District of Columbia, 1988–1996. *J Midwifery Womens Health.* 2001;46(1):4–10.
65. Sachs BP, Brown DA, Driscoll SG, et al. Maternal mortality in Massachusetts. Trends and prevention. *N Engl J Med.* 1987;316(11):667–72.
66. Ho EM, Brown J, Graves W, et al. Maternal death at an inner-city hospital, 1949–2000. *Am J Obstet Gynecol.* 2002;187(5):1213–6.
67. Thaden P, Thoennes N. Extent, nature and consequences of intimate partner violence: findings from the national violence against women survey. Washington, DC: US Department of Justice; 2000.
68. Truman JS. National criminal victimization survey: criminal victimization, 2010. Washington, DC: US Department of Justice, Bureau of Justice Statistics; 2011. <http://www.bjs.gov/content/pub/pdf/cv10.pdf>.
69. Ribe JK, Tegatz JR, Harvey CM. Blows to the maternal abdomen causing fetal demise: report of three cases and a review of the literature. *J Forensic Sci.* 1993;38:1092–6.
70. Dietz PM, Rochat RW, Thompson BL, et al. Differences in the risk of homicide and other fatal injuries between postpartum women and other women of childbearing age: implications for prevention. *Am J Public Health.* 1998;88:641–3.
71. Horon IL, Cheng D. Enhanced surveillance for pregnancy-associated mortality—Maryland, 1993–1998. *JAMA.* 2001;285:1455–9.
72. Lin P, Gill LR. Homicides of pregnant women. *Am J Forensic Med Pathol.* 2011;32:161–3.
73. Wiencrot A, Nannini A, Manning SE, et al. Neonatal outcomes and mental illness, substance abuse, and intentional injury during pregnancy. *Matern Child Health J.* 2012;16:979–88.
74. Chang J, Berg CJ, Saltzman LE, et al. Homicide: a leading cause of injury deaths among pregnancy and postpartum women in the United States, 1991–1999. *Am J Public Health.* 2005;95:471–7.
75. Ghandi SG, Gilbert WM, McElvy SS, et al. Maternal and neonatal outcomes after attempted suicide. *Obstet Gynecol.* 2006;107:984–90.
76. Schiff MA, Grossman DC. Adverse perinatal outcomes and risk for postpartum suicide attempt in Washington state, 1987–2001. *Pediatrics.* 2006;118:e669–75.
77. Maghsoudi H, Samnia R, Garadaghi A, et al. Burns in pregnancy. *Burns.* 2006;32:246–50.
78. Guo SS, Greenspoon JS, Kahn AM. Management of burn injuries during pregnancy. *Burns.* 2001;27:394–7.
79. Chama CM, Na'aya HU. Severe burn injury in pregnancy in northern Nigeria. *J Obstet Gynaecol.* 2002;22:20–2.
80. Van Haren RM, Thorsen CM, Valle EJ, et al. Hypercoagulability after burn injury. *J Trauma Acute Care Surg.* 2013;75:37–43, discussion 43.
81. Rode H, Millar AJ, Cywes S, et al. Thermal injury in pregnancy—the neglected tragedy. *S Afr Med J.* 1990;77:346–8.
82. Karimi H, Momeni M, Rahbar H. Burn injuries during pregnancy in Iran. *Int J Gynaecol Obstet.* 2009;104:132–4.
83. Roderique JD, Gebre-Giorgis AA, Stewart DH, et al. Smoke inhalation injury in a pregnant patient: a literature review of the evidence and current best practices in the setting of a classic case. *J Burn Care Res.* 2012;33:624–33.
84. Fatovich DM. Electric shock in pregnancy. *J Emerg Med.* 1993;11:175–7.
85. Einarson A, Bailey B, Inocencion G, et al. Accidental electric shock in pregnancy: a prospective cohort study. *Am J Obstet Gynecol.* 1997;176:678–81.
86. Rayburn W, Aronow R, DeLancey B, et al. Drug overdose during pregnancy an overview from a metropolitan poison control center. *Obstet Gynecol.* 1984;64:611–4.
87. Perrone J, Hoffman RS. Toxic injections in pregnancy: abortifacient use in a case series of overdose patients. *Acad Emerg Med.* 1997;4:206–9.
88. Bailey B. Are there teratogenic risks associated with antidotes used in the acute management of the poisoned pregnant woman? *Birth Defects Res A Clin Mol Teratol.* 2003;67:133–40.
89. Hardt N, Wong TD, Burt MJ, et al. Prevalence of prescription and illicit drugs in pregnancy-associated non-natural deaths of Florida mothers, 1999–2005. *J Forensic Sci.* 2013. doi:10.1111/1556-4029.12219.
90. Brown SA, Seifert S, Rayburn WF. Management of envenomations during pregnancy. *Clin Toxicol (Phila).* 2013;51:3–15.
91. Brookfield KF, Gonzalez-Quintero VH, Davis JS, et al. Maternal death in the emergency department from trauma. *Arch Gynecol Obstet.* 2013;288(3):507–12. doi:10.1007/s00404-013-2772-5.
92. Crosby WM. Traumatic injuries in pregnancy. *Clin Obstet Gynecol.* 1983;26:902–12.
93. American College of Obstetricians and Gynecologists. Committee on Obstetric Practice Safety for Maternal-Fetal Medicine. Committee Opinion No. 573: magnesium sulfate use in obstetrics. *Obstet Gynecol.* 2013;122:727–8.
94. Doyle LW, Crowther CA, Middleton P, et al. Magnesium sulphate for women at risk of preterm birth for neuroprotection of the fetus. *Cochrane Database Syst Rev.* 2009;(1):Art. No.: CD004661. doi:10.1002/14651858.CD004661.pub3.
95. Ali J, Yeo A, Gana TJ, et al. Predictors of fetal mortality in pregnant trauma patients. *J Trauma.* 1997;42:782–5.
96. Barraco RD, Chiu WC, Clancy TV, et al. Practice management guidelines for the diagnosis and management of injury in the pregnant patient: the EAST practice management guidelines work group. *J Trauma.* 2010;69:211–4.

97. Pearlman M, Tintinalli J. Evaluation and treatment of the gravida and fetus following trauma during pregnancy. *Obstet Gynecol Clin North Am.* 1991;18(2):371–80.
98. Shah AJ, Kilcane BA. Trauma in pregnancy. *Emerg Med Clin North Am.* 2003;21:615–29.
99. Ellingsen CL, Eggebø TM, Lexow K. Amniotic fluid embolism after blunt abdominal trauma. *Resuscitation.* 2007;75:180–3.
100. Kramer JS, Rouleau J, Siu S, et al. Amniotic fluid embolism in a pregnant and peripartum patient. *Crit Care Med.* 2005;33(10 Suppl):S354–61.
101. Pilkington S, Carli F, Dakin MJ, et al. Increase in Mallampati score during pregnancy. *Br J Anaesth.* 1995;74(6):638–42.
102. Collins JS, Lemmens HJ, Brodsky JB, et al. Laryngoscopy and morbid obesity: a comparison of the “sniff” and “ramped” positions. *Obes Surg.* 2004;14(9):1171–5.
103. El-Orbany M, Woehlk M, Salem MR. Head and neck position for direct laryngoscopy. *Anesth Analg.* 2011;113(1):103–9.
104. Robitaille A, Williams SR, Tremblay MH, et al. Cervical spine motion during tracheal intubation with manual in-line stabilization: direct laryngoscopy versus GlideScope® videolaryngoscopy. *Anesth Analg.* 2008;106:935–41.
105. Maruyama K, Yamada T, Kawakami R, et al. Randomized cross-over comparison of cervical-spine motion with the AirWay Scope or Macintosh laryngoscope with in-line stabilization: a video-fluoroscopic study. *Br J Anaesth.* 2008;101:563–7.
106. Yeatts DJ, Dutton RP, Hu PF, et al. Effect of video laryngoscopy on trauma patient survival: a randomized controlled trial. *J Trauma Acute Care Surg.* 2013;75:212–9.
107. Quinn AC, Milne D, Columb M, et al. Failed tracheal intubation in obstetric anaesthesia: 2 yr national case-control study in the UK. *Br J Anaesth.* 2013;110:74–80.
108. Gress Jr FC. Uterine vascular response to hemorrhage during pregnancy, with observations on therapy. *Obstet Gynecol.* 1966;27(4):549–54.
109. John PR, Shiozawa A, Haut ER, et al. An assessment of the impact of pregnancy on trauma mortality. *Surgery.* 2011;149:94–8.
110. Wilkening RB, Mescha G. Fetal oxygen uptake, oxygenation, and acid-base balance as a function of uterine blood flow. *Am J Physiol.* 1983;244(6):H749–55.
111. Advanced Trauma Life Support (ATLS) for doctors. Chicago: American College of Surgeons Committee on Trauma; 2012. <http://www.facs.org/trauma/atls/index.html>.
112. Shackford SR, Hollingworth-Fridlund P, Cooper GF, et al. The effect of regionalization upon the quality of trauma care as assessed by concurrent audit before and after institution of a trauma system: a preliminary report. *J Trauma.* 1986;26(9):812–20.
113. Sasser SM, Hunt RC, Faul M, et al. Center for Disease Control and Prevention (CDC). Guidelines for field triage of injured patients. Recommendations of the national expert panel on field triage, 2011. *MMWR Recomm Rep.* 2012;61(RR-1):1–20.
114. Brown JB, Stassen NA, Bankey PE, et al. Mechanism of injury and special consideration criteria still matter: an evaluation of the national trauma triage protocol. *J Trauma.* 2011;70:38–44, discussion 44–5.
115. Vanden Hoek TL, Morrison LJ, Shuster M, et al. Part 12: cardiac arrest in special situations: 2010 American heart association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation.* 2010;122(18 Suppl 3):S829–61.
116. Siddiqui N, Goldszmidt E, Haque SU, Carvalho JCA. Ultrasound simulation of internal jugular vein cannulation in pregnant and nonpregnant women. *Can J Anaesth.* 2010;57:966–72.
117. Chatterjee DJ, Bukunola B, Samuels TL, et al. Resuscitation in massive obstetric haemorrhage using an intraosseous needle. *Anaesthesia.* 2011;66(4):306–10.
118. NAMET. PHTLS Trauma First Response. St. Louis, MO: Mosby/JEMS; 2011.
119. Practice guidelines for perioperative blood transfusion and adjuvant therapies: an updated reported by the American Society of Anesthesiologists Task force on Perioperative Blood Transfusion and Adjuvant Therapies. *Anesthesiology.* 2006;105:198–208.
120. Mittermayr M, Streif W, Haas T, et al. Hemostatic changes after crystalloid or colloid fluid administration during major orthopedic surgery: the role of fibrinogen administration. *Anesth Analg.* 2007;105:905–17.
121. Thorsen K, Ringdal KH, Strand K, et al. Clinical and cellular effects of hypothermia, acidosis and coagulopathy in major surgery. *Br J Surg.* 2011;98:894–907.
122. Morgan TJ, Venkatesh B, Hall J. Crystalloid strong ion difference determines metabolic acid-base change during acute normovolaemic haemodilution. *Intensive Care Med.* 2004;30:1432–7.
123. Bulger EM, May S, Kerby JD, et al. Out-of-hospital hypertonic resuscitation after traumatic hypovolemic shock: a randomized controlled trial. *Ann Surg.* 2011;253:431–41.
124. Cooper DJ, Myles PS, McDermott FT, et al. Prehospital hypertonic saline resuscitation of patients with hypotension and severe traumatic

- brain injury: a randomized controlled trial. *JAMA*. 2004;291:1350–7.
127. Myburgh JA, Mythen MG. Resuscitation fluids. *N Engl J Med*. 2013;369:1243–51.
 128. Duke MD, Guidry C, Guice J, Stuke L, et al. Restrictive fluid resuscitation in combination with damage control resuscitation: time for adaptation. *J Trauma Acute Care Surg*. 2012;73:674–8.
 129. The SAFE Study Investigators. A comparison of albumin and saline for fluid resuscitation in the intensive care unit. *N Engl J Med*. 2004;350:2247–56.
 130. The SAFE Study Investigators. A comparison of albumin and saline for fluid resuscitation in the intensive care unit. Saline or albumin for fluid resuscitation in patients with traumatic brain injury. *N Engl J Med*. 2007;357:874–84.
 131. Winstedt D, Hanna J, Schött U. Albumin-induced coagulopathy is less severe and more effectively reversed with fibrinogen than is synthetic colloid-induced coagulopathy. *Scand J Clin Lab Invest*. 2013;73(2):161–9.
 132. Hartog CS, Kohl M, Reinert K. A systemic review of third-generation hydroxyethyl starch (HES 130/0.4) in resuscitation: safety not adequately addressed. *Anesth Analg*. 2011;112:635–45.
 133. Caballo C, Escolar G, Diaz-Ricart M, et al. Impact of experimental haemodilution on platelet function, thrombin generation and clot firmness: effects of different coagulation factor concentrates. *Blood Transfus*. 2013;11:391–9.
 134. Haase N, Perner A. Hydroxyethyl starch for resuscitation. *Curr Opin Crit Care*. 2013;19(4):321–5.
 135. FDA Safety Communication: Boxed Warning on increased mortality and severe renal injury, and additional warning on risk of bleeding, for use of hydroxyethyl starch solutions in some settings. June 24, 2013. <http://www.fda.gov/biologicsbloodvaccines/safetyavailability/ucm358271.htm>.
 136. Kauvar DS, Wade CE. The epidemiology and modern management of traumatic hemorrhage: US and international perspectives. *Crit Care*. 2005;9 Suppl 5:S1–9.
 137. Duchesne JC, McSwain NE, Cotton BA, et al. Damage control resuscitation: the new face of damage control. *J Trauma*. 2010;69:976–90.
 138. Holcomb JB, Hess JR. Early massive trauma transfusion: state of the art. *J Trauma*. 2006;60:1.
 139. Holcomb JB, Wade CE, Michalek JE, et al. Increased plasma and platelet to red blood cell ratios improves outcome in 466 massively transfused civilian trauma patients. *Ann Surg*. 2008;248:447–58.
 140. Holcomb JB, Jenkins D, Rhee P, et al. Damage control resuscitation: directly addressing the early coagulopathy of trauma. *J Trauma*. 2007;62:307–10.
 141. Dries DH. Hypotensive resuscitation. *Shock*. 1996;6:311–6.
 142. Doreide E, Deaken CD. Pre-hospital fluid therapy in the critically injured patient—a clinical update. *Injury*. 2005;35:1001–10.
 143. Karpati PCJ, Rossignol M, Pirot M, et al. High incidence of myocardial ischemia during postpartum hemorrhage. *Anesthesiology*. 2004;100:30–6.
 144. Palmer RM. Postpartum hemorrhage is not the only setting for maternal myocardial ischemia. *Anesthesiology*. 2004;101:1035–7.
 145. Sihler KC, Napolitano NM. Complications of massive transfusion. *Chest*. 2010;137:209–20.
 146. Duchesne JC, Hunt JP, Wahl G, et al. Review of current blood transfusion strategies in a mature level I trauma center: we were wrong for the last 60 years? *J Trauma*. 2008;65:272–6.
 147. Teixeira PG, Inaba K, Shulman I, et al. Impact of plasma transfusion in massive transfused trauma patients. *J Trauma*. 2009;66:693–7.
 148. Murad MH, Stubbs JR, Gandhi MJ, et al. The effect of plasma transfusion on morbidity and mortality: a systematic review and meta-analysis. *Transfusion*. 2010;50:1370–83.
 149. Johansson PI, Oliveri R, Ostrowski SR. Hemostatic resuscitation with plasma and platelets in trauma. A meta-analysis. *J Emerg Trauma Shock*. 2012;5:120–5.
 150. Holcomb JB, Zarzabal LA, Michalek JE, et al. Trauma Outcomes Group: increased platelet: RBC ratios are associated with improved survival after massive transfusion. *J Trauma*. 2011;71(2 Suppl 3):S318–28.
 151. Ho KM, Leonard A. Concentration-dependent effect of hypocalcaemia on mortality of patients with critical bleeding requiring massive transfusion: a cohort study. *Anaesth Intensive Care*. 2011;39:46–54.
 152. Snegovskikh D, Clebone A, Norwitz E. Anesthetic management of patients with placenta accreta and resuscitation strategies for associated massive hemorrhage. *Curr Opin Anaesthesiol*. 2011;24:274–81.
 153. Ho KM, Leonard A. Risk factors associated with hypomagnesemia in massive transfusion. *Transfusion*. 2011;51(2):270–6.
 154. Chaiwat O, Lang JD, Vavilala MS, et al. Early packed red blood cell transfusion and acute respiratory distress syndrome after trauma. *Anesthesiology*. 2009;110:351–60.
 155. Stansbury LG, Dutton RP, Stein DM, et al. Controversy in trauma resuscitation: do ratios of plasma to red blood cells matter? *Transfus Med Rev*. 2009;23:255–65.
 156. Allen SR, Kashuk JL. Unanswered questions in the use of blood component therapy in trauma. *Scand J Trauma Resusc Emerg Med*. 2011;19:5.
 157. Nascimento B, Callum J, Rubenfeld G, et al. Clinical review: fresh frozen plasma in massive bleedings—more questions than answers. *Crit Care*. 2010;14:202.
 158. Spahn DR, Cerny V, Coasts TJ. Management of bleeding following major trauma: a European guideline. *Crit Care*. 2007;11:R17.
 159. Grasseto A, De Nardin M, Ganzerla B, et al. ROTEM[®] guided coagulation factor concentrate therapy in trauma: 2-year experience in Venice, Italy. *Crit Care*. 2012;16:428.

160. Schöchl H, Maegele M, Solomon C, et al. Early and individualized goal-directed therapy for trauma-induced coagulopathy. *Scand J Trauma Resusc Emerg Med.* 2012;20:15.
161. Schöchl H, Schlimp CJ. Trauma bleeding management: the concept of goal-directed primary care. *Anesth Analg.* 2013. doi:10.1213/ANE.0b013e318270a6f7.
162. Fries D, Innerhofer P, Schobersberger W. Time for changing coagulation management in trauma-related massive bleeding. *Curr Opin Anaesthesiol.* 2009;22:267–74.
163. Brohi K, Singh J, Heron M, et al. Acute traumatic coagulopathy. *J Trauma.* 2003;54:1127–30.
164. Brohi K, Cohen MH, Gantner MT, et al. Acute coagulopathy of trauma: hypoperfusion induces systemic anticoagulation and hyperfibrinolysis. *J Trauma.* 2008;65:1211–7.
165. Wolberg AS, Meng ZH, Monroe III DM, et al. A systematic evaluation of the effects of temperature on coagulation enzyme activity and platelet function. *J Trauma.* 2004;56:1221–8.
166. Kostousov V, Wang YW, Cotton BA, et al. Influence of resuscitation fluids, fresh frozen plasma and fibrinolytics on fibrinolysis in a thromboelastography-based, in-vitro, whole-blood model. *Blood Coagul Fibrinolysis.* 2013;24:489–97.
167. CRASH-2 Trial Collaborators. Effects of TXA on death, vascular occlusive events, and blood transfusion in trauma patients with significant haemorrhage (CRASH-2): a randomized placebo-controlled trial. *Lancet.* 2010;376:2–32.
168. CRASH-2 Trial Collaborators. Effect of TXA in traumatic brain injury: a nested randomized, placebo controlled trial. CRASH-2 Intracranial Bleeding Study. *BMJ.* 2011;343:d3795. doi:10.1136/bmj.d3795.
169. Huang F, Wu D, Ma G, et al. The use of tranexamic acid to reduce blood loss and transfusion in major orthopedic surgery: a metaanalysis. *J Surg Res.* 2014;186(1):318–27. doi:10.1016/j.jss.2013.08.020.
170. Rappold JF, Pusateri AE. Tranexamic acid in remote damage control resuscitation. *Transfusion.* 2013;53:96S–9.
171. Shakur H, Elbourn D, Gülmezoglu M, et al. The WOMAN Trial World Maternal Antifibrinolytic Trial: tranexamic acid for post-partum haemorrhage: an international randomized, double blind placebo controlled trial. *Trials.* 2010;11:40. doi:10.1186/1745-6215-11-40.
172. Sentürk M, Cakmak Y, Yildiz G, et al. Tranexamic acid for cesarean section: a double-blind placebo-controlled randomized clinical trial. *Arch Gynecol Obstet.* 2013;287:641–5.
173. Levy JH, Szlam F, Tanaka KA, et al. Fibrinogen and hemostasis: a primary hemostatic target for the management of acquired bleeding. *Anesth Analg.* 2012;114:261–74.
174. Hiippala ST, Myllylä GJ, Vahtera EM. Hemostatic factors and replacement of major blood loss with plasma-poor red cell concentrates. *Anesth Analg.* 1995;81:360–5.
175. Sørensen B, Spahn DR, Innerhofer P, et al. Clinical review: prothrombin complex concentrates—evaluation of safety and thrombogenicity. *Crit Care.* 2011;15:201.
176. David JS, Godier A, Dargaud Y, et al. Case scenario: management of trauma-induced coagulopathy in a blunt trauma patient. *Anesthesiology.* 2013;119:191–200.
177. Boffard KD, Riou B, Warren B, et al. Recombinant factor VIIa as adjunctive therapy for bleeding control in severely injured trauma patients: two parallel randomized, placebo-controlled, double-blind clinical trials. *J Trauma.* 2005;58:8–15.
178. Kobayashi T, Nakabayashi M, Yoshioka A, et al. Recombinant activated factor VII (rFVIIA/NovoSeven®) in the management of severe postpartum haemorrhage: initial report of a multicentre case series in Japan. *Int J Hematol.* 2012;95:157–63.
179. Huber AW, Raio L, Alberio L, et al. Recombinant human factor VIIa prevents hysterectomy in severe postpartum hemorrhage: single center study. *J Perinat Med.* 2011;40:43–9.
180. Seoud M, Cheaib S, Birjawi G, et al. Successful treatment of severe retroperitoneal bleeding with recombinant factor VII in a woman with placenta percreata invading into the left broad ligament: unusual repeated antepartum intra-abdominal bleeding. *J Obstet Gynaecol Res.* 2010;36:183–6.
181. Nohira T, Osakabe Y, Suda S, et al. Successful management by recombinant activated factor VII in a case of disseminated intravascular coagulopathy caused by obstetric hemorrhage. *J Obstet Gynaecol Res.* 2008;34:623–30.
182. Fries D. The early use of fibrinogen, prothrombin complex concentrate, and recombinant-activated factor VIIa in massive bleeding. *Transfusion.* 2013;53:91S–5.
183. Frank LR. Is MAST in the past? The pros and cons of MAST usage in the field. *FEMS.* 2000;25:38–41, discussion 44–5.
184. Pease CS, Magrina JF, Finely BE. The use of MAST suit in obstetrics and gynecology. *Obstet Gynecol Surv.* 1984;39:416–22.
185. Miller S, Martin HB, Moris JL. Anti-shock garment in post-partum haemorrhage. *Best Pract Res Clin Obstet Gynecol.* 2008;22(6):1057–74.
186. Sutherland T, Downing J, Miller S, et al. Use of the non-pneumatic anti-shock garment (NASG) for life-threatening obstetric hemorrhage: a cost-effectiveness analysis in Egypt and Nigeria. *PLoS One.* 2013;8(4):62282. doi:10.1371/journal.pone.0062282.
187. Lester F, Stenson A, Meyer C, et al. Impact of the Non-pneumatic Antishock Garment on pelvic blood flow in healthy postpartum women. *Am J Obstet Gynecol.* 2011;204:409.e1–5.
188. Abdelaziz A. Fetal Fibronectin (Quick Check fFN test®) for detection of premature rupture of fetal

- membranes. *Arch Gynecol Obstet.* 2013;287(2):205–10.
189. van der Ham DP, van Teefelen AS, Mol BW. Prelabor rupture of membranes: overview of diagnostic methods. *Curr Opin Obstet Gynecol.* 2012;24(6):408–12.
 190. Macones GA, Hankins GD, Spong CY, et al. The 2008 National Institute of Child Health and Human Development workshop report on electronic fetal monitoring: update on definitions, interpretation, and research guidelines. *Obstet Gynecol.* 2008;112(3):661–6.
 191. Muehnhch MV, Baschat AA, Reddy UM, Mighty HE, Weinder CP, Scalea TM, Harman CR. Kleihauer-Betke testing is important in all cases of maternal trauma. *J Trauma.* 2004;57(5):1094–8.
 192. Kim YA, Makar RS. Detection of fetomaternal hemorrhage. *Am J Hematol.* 2012;87(4):417–23.
 193. Chambers E, Davies L, Evans S, et al. Comparison of haemoglobin F detection by the acid elution test, flow cytometry and high-performance liquid chromatography in maternal blood samples analyzed for fetomaternal haemorrhage. *Transfus Med.* 2012;22(3):199–204.
 194. Cosmi E, Rampon M, Saccardi C, Zanardo V, Litta P. Middle cerebral artery peak systolic velocity in the diagnosis of fetomaternal hemorrhage. *Int J Gynaecol Obstet.* 2012;117(2):128–30.
 195. Nguyen CP, Goodman LH. Fetal risk in diagnostic radiology. *Semin Ultrasound CT MRI.* 2012;33:4–10.
 196. Sadro C, Bittle M, O'Connell K. Imaging the pregnant trauma patient. *Ultrasound Clin.* 2011;6:97–103.
 197. Yousefzadeh D, Ward M, Reft C. Internal barium shielding to minimize fetal irradiation in spiral CT: a phantom simulation experiment. *Radiology.* 2006;239:751–8.
 198. Webb JA, Thomsen HS, Morcos SK. Members of Contrast Media Safety Committee of European Society of Urogenital Radiology (ESUR). The use of iodinated and gadolinium contrast media during pregnancy and lactation. *Eur Radiol.* 2005;15:1234–40.
 199. Ma OJ, Mateer JR, DeBehnke DJ. Use of ultrasonography for the evaluation of pregnant trauma patients. *J Trauma.* 1996;40(4):665–8.
 200. Goodwin H, Holmes JF, Wisner DH. Abdominal ultrasound examination in pregnant trauma patients. *J Trauma.* 2001;50:689.
 201. Bochicchio GV, Haan J, Scalea TM. Surgeon-performed focused assessment with sonography for trauma as an early screening tool for pregnancy after trauma. *J Trauma.* 2002;52(6):1125–8.
 202. Sadro C, Bernstein MP, Kanal KM. Imaging of Trauma: part 2, abdominal trauma and pregnancy—a radiologist's guide to doing what is best for the mother and baby. *AJR Am J Roentgenol.* 2012;199:1207–19.
 203. Rothenberger DA, Quattlebaum FW, Zabel J, et al. Diagnostic peritoneal lavage for blunt trauma in pregnant women. *Am J Obstet Gynecol.* 1977;129(5):479–81.
 204. Scorpio RJ, Esposito TJ, Smith LG, et al. Blunt trauma during pregnancy: factors affecting fetal outcome. *J Trauma.* 1992;32(2):213–6.
 205. National Spinal Cord Injury Statistics Center. UAB spinal cord injury info sheet #15. Birmingham, AL: University of Alabama; 2009. https://www.nscisc.uab.edu/PublicDocuments/fact_figures_docs/Facts%202012%20Feb%20Final.pdf. Updated February 2012. Accessed 12 Oct 2013.
 206. Ghidini A, Simonson MR. Pregnancy after spinal cord injury: a review of the literature. *Top Spinal Cord Inj Rehabil.* 2011;16(3):93–103.
 207. Furlan JC. Autonomic dysreflexia—a clinical emergency. *J Trauma Acute Care Surg.* 2013;75:496–500.
 208. Hughes SJ, Short DJ, Usherwood MM, et al. Management of the pregnant woman with spinal cord injuries. *Br J Obstet Gynaecol.* 1991;98(6):513–8.
 209. Krassioukov A, Warburton DE, Teasell R, et al. A systematic review of the treatment of autonomic dysreflexia after spinal cord injury. *Arch Phys Med Rehabil.* 2009;90(4):682–95.
 210. Katz VL, Thorp Jr JM, Cefalo RC. Epidural analgesia and autonomic hyperreflexia: a case report. *Am J Obstet Gynecol.* 1990;162(2):471–2.
 211. Periera L. Obstetric management of the patient with spinal cord injury. *Obstet Gynecol Surv.* 2003;58(10):678–87.
 212. Owen MD, Stiles MM, Opper SE, et al. Autonomic hyperreflexia in a pregnant paraplegic patient. Case report. *Reg Anesth.* 1994;19(6):415–7.
 213. Takatsuki A, Ohtsuka M. Clinical trial of a method for confirming the effects of spinal anesthesia in patients with spinal cord injury. *J Anesth.* 2012;26(6):914–7.
 214. Camune BD. Challenges in the management of pregnant women with spinal cord injury. *J Perinat Neonatal Nurs.* 2013;27(3):225–31.
 215. Biderman P, Einav S, Fainblut M, et al. Extracorporeal life support in patients with multiple injuries and severe respiratory failure: a single-center experience. *J Trauma Acute Care Surg.* 2013;75:907–12.
 216. Ried M, Bein T, Philipp A, et al. Extracorporeal lung support in trauma patients with severe chest injury and acute lung failure: a 10-year institutional experience. *Crit Care.* 2013;17:R110. <http://ccforum.com/content/17/3/R110>.
 217. Plotkin JS, Shah JB, Lofland GK, et al. Extracorporeal membrane oxygenation in the successful treatment of traumatic adult respiratory distress syndrome: case report and review. *J Trauma.* 1994;37(1):127–30.
 218. Cunningham JA, Devine PC, Jelic S. Extracorporeal membrane oxygenation in pregnancy. *Obstet Gynecol.* 2006;103:792–5.
 219. King PT, Rosalion A, McMillan J, et al. Extracorporeal membrane oxygenation in pregnancy. *Lancet.* 2000;356:45–6.

220. Robertson LC, Allen SH, Konamme SP, et al. The use of extra-corporeal membrane oxygenation in the case of a pregnant woman with severe h1N1 2009 influenza complicated by pneumonitis and adult respiratory distress syndrome. *Int J Obstet Anesth.* 2010;19:443–7.
221. Grasselli G, Bombino M, Patroniti P, et al. Use of extracorporeal respiratory support during pregnancy: a case report and literature review. *ASAIO J.* 2012;58:281–4.
222. Ho CH, Chen KB, Liu SK, et al. Early application of extracorporeal membrane oxygenation in a patient with amniotic fluid embolism. *Acta Anaesthesiol Taiwan.* 2009;47(2):99–102.
223. Weinberg L, Kay C, Liskaser F, et al. Successful treatment of peripartum massive pulmonary embolism with extracorporeal membrane oxygenation and catheter-directed pulmonary thrombolytic therapy. *Anaesth Intensive Care.* 2011;39(3):486–91.
224. Smith IJ, Gillham MJ. Fulminant peripartum cardiomyopathy rescue with extra-corporeal membranous oxygenation. *Int J Obstet Anesth.* 2009;18(2):186–8.
225. Ngatchou W, Ramadan AS, Van Nooten G, et al. Left tilt position for easy extracorporeal membrane oxygenation cannula insertion in late pregnancy patients. *Interact Cardiovasc Thorac Surg.* 2012;15:285–7.
226. Chapman BC, Moore EE, Barnett C, et al. Hypercoagulability following blunt solid abdominal organ injury: when to initiate anticoagulation. *Am J Surg.* 2013;206:917–23. doi:10.1016/j.amjsurg.2013.07.024.
227. Harr JN, Moore EE, Ghasabyan A, et al. Functional fibrinogen assay indicates that fibrinogen is critical in correcting abnormal clot strength following trauma. *Shock.* 2013;39:459. doi:10.1097/SHK.0b013e3182787122.
228. Hill CC, Pickinpaugh J. Trauma and surgical emergencies in the obstetric patient. *Surg Clin North Am.* 2008;88:282–7.
229. Nelson-Piercy C. Chapter 9: Cardiac disease in Center for Maternal and Child Enquiries (CMACE). Saving mother's lives: reviewing maternal deaths to make motherhood safer: 2006–08. The eighth report on confidential enquiries into maternal deaths in the United Kingdom. *BJOG.* 2011;118:1–203.
230. Hui D, Morrison LJ, Windrim R, et al. The American Heart Association 2010 guidelines for the management of cardiac arrest in pregnancy: consensus recommendations on implementation strategies. *J Obstet Gynaecol Can.* 2011;33:858–63.
231. Morris S, Stacey M. Resuscitation in pregnancy. *BMJ.* 2003;327:1277–9.
232. Rees GA, Willis BA. Resuscitation in late pregnancy. *Anaesthesia.* 1988;43:347–9.
233. Mathur D, Leon SB. Perimortem caesarean section: a review of the anaesthetist's nightmare. *Trends Anaesth Crit Care.* 2013;3(6):327–30. <http://x.doi.org/10/10/16/j.tacc.2013.05.002>.
234. Jeejeebhoy FM, Zelop CM, Windrim R, et al. Management of cardiac arrest in pregnancy: a systematic review. *Resuscitation.* 2011;82:801–9.
235. Suresh MS, LaToya MC, Munnur U. Cardiopulmonary resuscitation and the parturient. *Best Pract Res Clin Obstet Gynaecol.* 2010;24:383–400.
236. Kundra P. Manual displacement of the uterus during Caesarean section. *Anaesthesia.* 2007;62:460–5.
237. Nanson J, Elcock D, Williams M, et al. Do physiological changes in pregnancy change defibrillation energy requirements? *Br J Anaesth.* 2001;87:237–9.
238. Farinelli CK, Hameed AB. Cardiopulmonary resuscitation in pregnancy. *Cardiol Clin.* 2012;30:453–61.
239. Morris Jr JA, Rosenbower TH, Jurkowich GJ, et al. Infant survival after cesarean section for trauma. *Ann Surg.* 1996;223:481–8.
240. DePace NL, Betesh JS, Kotler MN. "Postmortem" cesarean delivery with recovery of both mother and offspring. *JAMA.* 1982;248:971–3.
241. Katz VL, Dotters DH, Droegmueller W. Perimortem cesarean delivery. *Obstet Gynecol.* 1986;68:571–6.
242. Katz VL, Balderston K, DeFreest M. Perimortem cesarean delivery: were our assumptions correct? *Am J Obstet Gynecol.* 2005;192:1916–20.
243. Clark SL, Hankins GD, Dudley DA, et al. Amniotic fluid embolism: analysis of the national registry. *Am J Obstet Gynecol.* 1995;172(4 Pt 1):1158–67.
244. Einav S, Kaufman N, Sela HY. Maternal cardiac arrest and perimortem cesarean delivery: evidence or expert-based? *Resuscitation.* 2012;83:1191–200.
245. Center for Maternal and Child Enquiries (CMACE). Saving mother's lives: reviewing maternal deaths to make motherhood safer: 2006–08. The eighth report on confidential enquiries into maternal deaths in the United Kingdom. *BJOG.* 2011;118:1–203.
246. Lipman SS, Wong JY, Arafeh J, et al. Transport decreases the quality of cardiopulmonary resuscitation during simulated maternal cardiac arrest. *Anesth Analg.* 2013;116:162–7.
247. Bernstein IM, Watson M, Simmons GM, et al. Maternal brain death and prolonged fetal survival. *Obstet Gynecol.* 1989;74:734–77.
248. Mallampalli A, Powner DJ, Gardner MO. Cardiopulmonary resuscitation and somatic support of the pregnant patient. *Crit Care Clin.* 2004;20:747–61.
249. Field DR, Gates EA, Creasy RK, et al. Maternal brain death during pregnancy: medical and ethical issues. *JAMA.* 1988;260:816–22.
250. Craft Jr JB, Coaldrake LA, Yonekura ML, et al. Ketamine, catecholamines, and uterine tone in pregnant ewes. *Am J Obstet Gynecol.* 1983;146(4):429–34.