Research Article

Predictive Value of Noninvasive Hemodynamic Parameters For Post-Induction Hypotensionin Pregnant Women With Placenta Previa: A Review Article

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Abstract

During pregnancy, anatomical and physiological changes occur to meet the increased metabolic needs, to permit appropriate development of fetus and to prepare the body for childbirth. The changes begin to occur early in the first trimester, peaking at the term or labour and revert to pre-pregnancy levels by a few weeks into the postpartum. Pregnancy induces significant cardiovascular adaptations beginning as early as the 8th week of gestation. Increased estrogen and progesterone levels cause peripheral vasodilation and reduced systemic vascular resistance, requiring an increase in cardiac output to maintain adequate blood pressure. Pregnancy causes several physiological changes in the respiratory system. Estrogen-induced capillary engorgement leads to edema of the nasal, oropharyngeal, and laryngeal mucosa, while enlargement of the thoracic cage increases chest dimensions. Increased metabolic demands result in higher carbon dioxide production and a rise in minute ventilation, reducing arterial carbon dioxide tension ($PaCO_2$) to 30–32 mmHg and producing a mild, compensated respiratory alkalosis with decreased serum bicarbonate levels

Key words: Hypotension, General Anaesthesia, Placenta Previa.

Introduction

Cardiovascular System

Pregnancy induces significant cardiovascular adaptations beginning as early as the 8th week of gestation. Increased estrogen and progesterone levels cause peripheral vasodilation and reduced systemic vascular resistance, requiring an increase in cardiac output to maintain adequate blood pressure [**1**]. Cardiac output rises through increases in both heart rate and stroke volume, enhancing blood flow to the uterus, kidneys, and skin to support fetal growth, waste elimination, and maternal thermoregulation [**2**]. Blood volume progressively increases from 6–8 weeks of gestation, with plasma volume expanding by 40–50% due to activation of the renin–angiotensin system. Left ventricular end-diastolic volume and ejection fraction also increase, while central venous and pulmonary capillary wedge pressures remain largely unchanged [**3**].

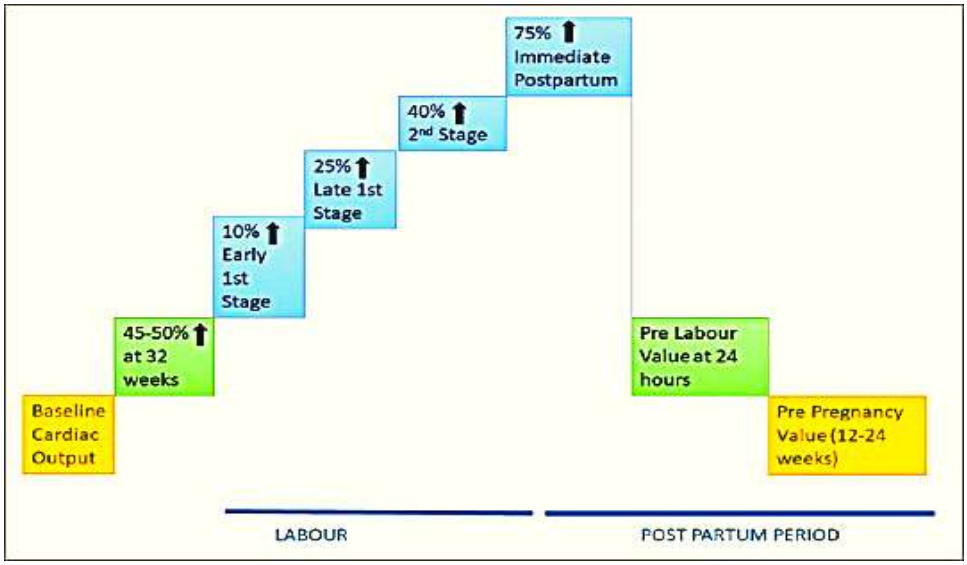

Fig [1] Graphical representation of changes in cardiac output during pregnancy, labor and postpartum. (↑: Increase) [3]

Anesthetic implications

Due to anatomical changes, the apical impulse is shifted laterally and cephalad to the fourth intercostal space. Increased blood volume provides some reserve for the normal blood loss during delivery (about 300–500 ml for vaginal delivery and 600–1000 ml for caesarean delivery) and peripartum hemorrhage. But due to this increase in blood volume, pregnant patients may not manifest the signs and symptoms of hypovolemia (tachycardia, hypotension, oliguria) till about 1500 ml of blood loss has occurred **[4].**

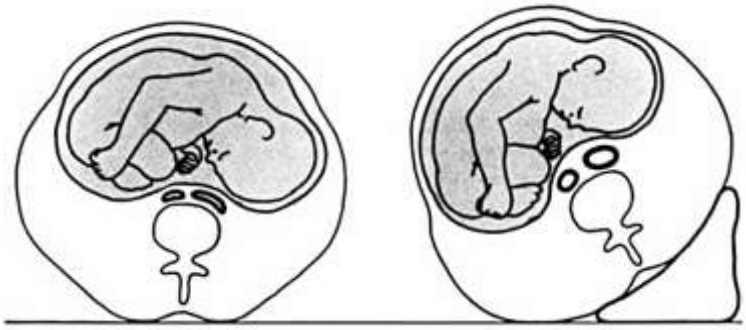

Fig. [2]. Aortocaval compression. **[4]**

Respiratory System

Pregnancy causes several physiological changes in the respiratory system. Estrogen-induced capillary engorgement leads to edema of the nasal, oropharyngeal, and laryngeal mucosa, while enlargement of the thoracic cage increases chest dimensions. Increased metabolic demands result in higher carbon dioxide production and a rise in minute ventilation, reducing arterial carbon dioxide tension ($PaCO_2$) to 30–32 mmHg and producing a mild, compensated respiratory alkalosis with decreased serum bicarbonate levels [**5**]. Alveolar ventilation increases arterial oxygen tension (PaO_2), enhancing maternal oxygenation and fetal oxygen delivery. Despite diaphragmatic elevation by the enlarging uterus, diaphragmatic excursion increases to maintain effective ventilation, although functional residual capacity decreases during late pregnancy [5].

Anesthetic implications

Mallampati classification worsens during pregnancy and more so during labour [4]. Upper airway changes, enlarged breasts and obesity can make intubation difficult during pregnancy. Laryngoscopes with short handles, smaller diameter endotracheal tubes and ramp position at the head end might be needed in difficult scenarios. Nasotracheal intubation should be avoided as there is an increased risk of nasal bleed during pregnancy [5].

Hematological and Immune System

Pregnancy is associated with a greater increase in plasma volume (40–50%) than red blood cell mass (approximately 20%), resulting in physiological anemia, which reduces blood viscosity and facilitates uteroplacental blood flow. Leukocyte count gradually increases, mainly due to neutrophils, reaching approximately 15,000/mm^3 during pregnancy. Although neutrophil function is relatively impaired, increasing the severity of some infections, pregnant women are not generally more susceptible to infections. Immunoglobulin levels (IgA, IgG, and IgM) and autoantibody production remain largely unchanged [6].

Anesthetic Implications

Routine platelet count assessment is not required before neuraxial anesthesia in healthy pregnant women. However, in patients with severe preeclampsia, platelet count should be evaluated within 6 hours before epidural placement or catheter removal because of the increased risk of epidural hematoma associated with thrombocytopenia.

Assessment of platelet function may also be beneficial, and thromboelastography (TEG) can provide rapid evaluation of overall coagulation status in acute clinical settings [7].

Gastrointestinal System

Pregnancy primarily affects gastrointestinal motility rather than secretory or absorptive functions. Progesterone-induced relaxation of the lower esophageal sphincter, together with anatomical displacement of the esophagus, predisposes pregnant women to gastroesophageal reflux [8]. Gastric emptying is generally unchanged during pregnancy but becomes delayed during labor and the immediate postpartum period. Reduced gastrointestinal motility also slows esophageal and intestinal transit, contributing to constipation, while nausea and vomiting occur in approximately 80% of pregnancies. Most gastrointestinal changes resolve within 1–2 days postpartum [8].

Anesthetic implications

No alteration is required in the fasting guidelines. Gastric emptying is delayed in pregnant and peripartum females receiving systemic or neuraxial opioids. There is an increased risk of aspiration of gastric contents due to increased intra-abdominal pressure and a low LOS tone. The risk is increased during GA and intubation. Important steps in prevention include preference to neuraxial techniques and use of aspiration prophylaxis. If GA is indicated, rapid sequence induction is recommended. In uncomplicated labour, moderate amount of clear fluids is recommended (9).

Nervous System

Cerebral blood flow is increased due to a decrease in cerebrovascular resistance. Permeability of the blood–brain barrier increases. There is an increase in threshold to pain at full term and in labour probably due to increased levels of plasma endorphins and progesterone. Due to the compression of the IVC by the gravid uterus, dilatation of the epidural venous plexus occurs. There is an increase in epidural fat and decrease in epidural free space and spinal cerebrospinal fluid (CSF) volume [10]. CSF pressure remains unchanged during pregnancy but is increased during uterine contractions and bearing down. There is more dependence on sympathetic nervous system for maintenance of hemodynamics [10].

Anesthetic implications

There is up to 30% decrease in minimum alveolar concentration of volatile anesthetic agents. Pregnant females are physiologically more sensitive to intravenous induction and sedative agents. There is a 25–40% decrease in spinal dose of local anesthetics (LA) since the end of first trimester implying that changes in epidural space anatomy is not the sole reason. It has been found that progesterone increases the sensitivity of neuronal membranes to LA. Pregnant females are more prone to hypotension and haemodynamic instability following sympathetic blockade caused by neuraxial anesthesia [11].

Renal System

Renal blood flow and glomerular filtration rate (GFR) are increased but no change is observed in histology or number of nephrons [12]. Due to progesterone and mechanical compression of ureters, renal pelvis and calyces are dilated. Increase in GFR causes decrease in serum creatinine (normal range: 0.4–0.8 mg/dl) and blood urea nitrogen (normal range: 8–10 mg/dl). Reduced vascular responsiveness to vasopressors (angiotensin II, norepinephrine and antidiuretic hormone) is observed due to an altered vascular receptor expression. Nitric oxide synthesis is increased during pregnancy resulting in systemic and renal vasodilation [13].

Anesthetic implications

Since pregnant females have a lower normal range of serum creatinine, a small rise in values reflects a larger reduction in renal function. Low albumin levels lead to increased free levels of highly protein-bound drugs such as digoxin, midazolam, thiopentone sodium and phenytoin [14].

Endocrine System

Thyroid gland is enlarged due to both follicular hyperplasia and increased vascularity. Due to the increase in thyroid-binding globulin caused by oestrogen, total T3 and T4 levels are increased by 50% but free T3 and T4 levels do not change. Thyroid stimulating hormone levels fall during first trimester but recover during rest of the pregnancy. Both subclinical hypo- and hyperthyroidism occur and are not associated with adverse outcomes [15]. Placental lactogen and dopamine cause hyperprolactinaemia during pregnancy. There is a 30% increase in oxytocin stores in the pituitary, which is released during labour and just after delivery. The oxytocin response to stress is diminished during pregnancy to prevent preterm labour [16].

Anesthetic implications

Pathological states arising due to iodine-deficient hypothyroidism or hyperthyroidism have much relevance to the practice of anesthesia and should be managed keeping in mind the physiological changes of pregnancy. GA can mask the signs and symptoms of hypoglycemia while neuraxial anesthesia can lead to exaggerated hemodynamic instability in patients with autonomic dysfunction related to diabetes mellitus or diabetic ketoacidosis [17].

Transplacental Transfer of Drugs and Safety

Transplacental transfer of drugs has been studied in animals (pregnant ewes, guinea pigs) and in vitro human placental models but application of these data to clinical practice is questionable. Due to inaccessibility of placenta in vivo, human studies are impractical and majority of studies provide data from single measurement of maternal and umbilical cord drug concentrations from samples obtained at delivery [18].

The Anesthetic Impact and Clinical Implications During Different Trimestes:

The anesthetic components of surgery take on major proportions depending on the procedure. Any elective general surgical operation should be avoided if at all possible during pregnancy. Emergency surgery can be cardiac or noncardiac, obstetric or non-obstetric, and has a variety of consequences. During the three trimesters, the anesthetic effect and clinical implications are slightly variable [19].

The First Trimester

During the first trimester, anesthetic management aims to avoid factors that may interfere with embryonic development while maintaining adequate maternal oxygenation, normovolemia, and stable hemodynamics [20]. Pregnancy-induced physiological adaptations affect the cardiovascular, respiratory, renal, hepatic, and neurological systems, increasing oxygen consumption and reducing functional residual capacity, thereby increasing the risk of hypoxemia. Hyperventilation leads to physiological hypocarbia, reduced minimum alveolar concentration, and more rapid inhalational induction. Excessive ventilation during general anesthesia should be avoided, as marked hypocapnia may reduce maternal cardiac output and uterine blood flow through vasoconstriction [21].

Second Trimester

To lessen the risk of spontaneous abortion and premature labor, it has been standard practice to postpone surgery until the second trimester. By this time, the majority of physiological changes have reached a plateau, making anesthetic management safer than in the first or third trimesters. Although the second trimester is believed to be a safer phase for emergency surgery than the first and third trimesters, it is nevertheless connected with several substantial physiologic changes that can endanger both the mother's and the fetus' lives [22].

Third Trimester

In the third trimester, decision-making gets a little easier because a caesarean section can be performed before the major surgery. Due to increased airway oedema caused by hormone interactions, all additional challenges to airway control during the first two trimesters are amplified. An emergency operation after 28 weeks can be performed with the introduction of steroids, which give critical cover for fetal lung maturation and prevent the neonate from developing acute respiratory distress syndrome [23].

Hemodynamic monitoring

Hemodynamic monitoring refers to the continuous assessment of cardiovascular status primarily blood pressure (BP), cardiac output, and vascular tone to evaluate tissue perfusion and cardiovascular stability during anesthesia and surgery. Traditional monitoring (e.g., non-invasive blood pressure, heart rate) provides intermittent or indirect measures of perfusion but cannot reliably predict sudden drops in blood pressure, especially in dynamic physiological states such as pregnancy and anesthetic induction. Advanced non-invasive indices aim to fill this predictive gap by reflecting peripheral perfusion or intravascular volume status in real time [24].

Predictors of hypotension

Perfusion Index (PI)

The Perfusion Index (PI) is a non-invasive parameter derived from pulse oximetry photoplethysmography and represents the ratio of pulsatile to non-pulsatile blood flow, reflecting changes in peripheral blood volume with each heartbeat [25]. PI is easy to measure, provides continuous bedside monitoring, and serves as an indirect indicator of peripheral microcirculatory perfusion. Increasing evidence suggests that normalization of macrocirculatory parameters does not necessarily indicate adequate microcirculatory perfusion, making PI a valuable adjunct in hemodynamic assessment. It has demonstrated clinical utility in shock resuscitation, fluid management, vasopressor therapy, outcome prediction, risk stratification, and pain assessment [26].

Measurement and Reference Range of Perfusion Index (PI)

The Perfusion Index (PI) is measured using a pulse oximetry probe that detects transmitted infrared light and separates the photoplethysmographic signal into pulsatile (arterial) and non-pulsatile (venous and tissue) components. PI is calculated as the ratio of pulsatile to non-pulsatile light absorption, providing an indirect measure of peripheral perfusion. Accurate measurement requires minimizing factors such as poor probe placement, motion artifacts, nail polish, and ambient light, with the middle finger being the preferred monitoring site [26].

Applications of Perfusion Index (PI) in Hemodynamic Management

The Perfusion Index (PI) is a useful non-invasive marker of both macrocirculatory and microcirculatory status, with several applications in hemodynamic monitoring. Assessment of fluid responsiveness: Increases in PI following fluid administration or passive leg raising (PLR) indicate fluid responsiveness. In septic shock, a 33% increase in PI after crystalloid infusion or a >9% increase during the PLR test reliably predicts responsiveness to fluid therapy [27].

Guiding fluid resuscitation: PI can assist in determining when to initiate or discontinue fluid resuscitation. Low PI suggests inadequate tissue perfusion and may indicate the need for further fluids, whereas normalization of PI without fluid responsiveness supports stopping fluid administration and considering fluid removal. PI-guided resuscitation has been associated with reduced fluid administration, shorter hospital stay, and lower organ failure scores compared with lactate-guided strategies [27].

Assessment of vascular tone: PI is inversely related to vascular tone and can reflect vasodilation or vasoconstriction. It has been used to assess the success of regional nerve blocks and to predict hypotension after anesthesia. In obstetric patients, a baseline PI >3.5 before cesarean section has been associated with an increased risk of hypotension following spinal anesthesia [27].

Inferior Vena Cava Collapsibility Index (IVCCI)

Definition & Mechanism: IVCCI is a dynamic ultrasound-derived index of respiratory variations in the diameter of the inferior vena cava (IVC). History, physical findings, and laboratory tests have limited sensitivity and specificity to assess relative intravascular volume or volume responsiveness [28] .

Procedures and Technical Steps

Subcostal View

We begin by visualizing the heart using the subcostal approach in most supine patients, with a phased-array (cardiac) or curvilinear (abdominal) probe. The probe is then rotated vertically, with the orientation marker pointed cranially, and moved 1 to 2 cm to the right of the patient's midline, while maintaining visualization of the right atrium (RA), to view the inferior venal cava (IVC) in its long axis [29].

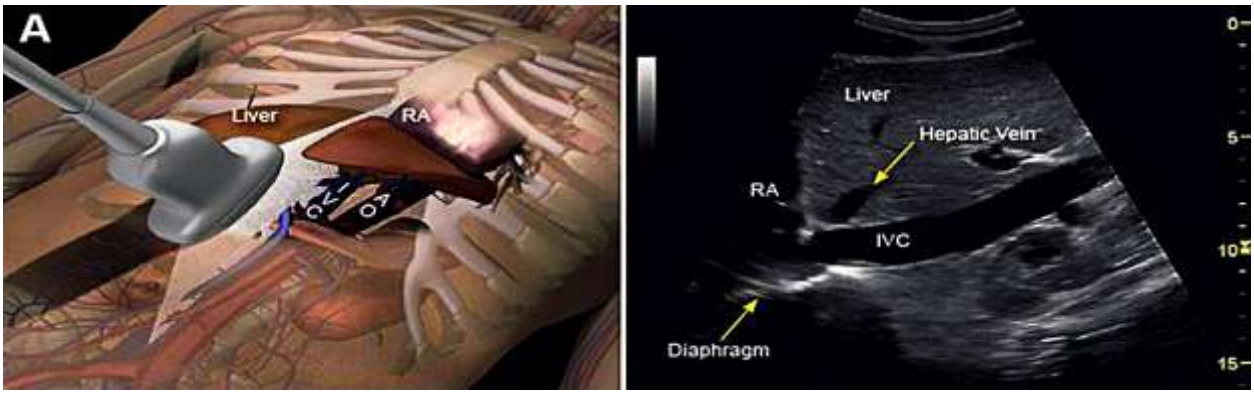

Figure 3. (A) Subcostal IVC window. [29]

B-Mode Vs M-Mode

Measuring IVC maximum and minimum diameters using M mode has not been shown to be superior to choosing maximum and minimum diameters viewed frame by frame in B-Mode. [30]

Calculation of Collapsibility Index vs Distensibility Index

The physiology of spontaneous breathing is different from that of mechanical ventilation. During spontaneous inspiration, negative intrathoracic pressure increases venous flow to the heart and reduces the IVC diameter. At end expiration, the intrathoracic pressure increases to zero, decreasing the venous return and maximizing the IVC diameter. This has been defined as collapsibility index (CI). CI = (IVC max-IVC min)/IVC max. [30]

Assessment of Relative Intravascular Volume

The major clinical value of IVC ultrasound findings is to potentially eliminate either the possibility of overt relative intravascular hypervolemia or hypovolemia in a given patient. A patient with a small IVC maximum diameter or large IVC CI may be “euvolemic”, but is unlikely to have elevated cardiac filling pressures, and a patient with a large IVC maximum diameter and small IVC CI may be “euvolemic”, but is unlikely to have reduced cardiac filling pressures. [31]

Hypovolemic Shock

Definition

Shock is best defined as a life-threatening, generalized form of acute circulatory failure associated with inadequate oxygen utilization by the cells. It is a state in which the circulation is unable to deliver sufficient oxygen to meet the demands of the tissues, resulting in cellular dysfunction. The result is cellular dysoxia, i.e. the loss of the physiological independence between oxygen delivery and oxygen consumption, associated with increased lactate levels. Some clinical symptoms suggest an impaired microcirculation, including mottled skin, acrocyanosis, slow capillary refill time and an increased central-to-toe temperature gradient [32].

Pathophysiology and Features of Shock

Shock is a state of acute circulatory failure resulting in inadequate tissue perfusion and oxygen delivery. It can arise from four main mechanisms: hypovolemia due to fluid or blood loss, cardiogenic failure caused by impaired cardiac contractility or arrhythmias, obstructive shock resulting from conditions such as pulmonary embolism, tension pneumothorax, or cardiac tamponade, and distributive shock due to loss of vascular tone, as seen in sepsis,

anaphylaxis, or spinal injury. These mechanisms may coexist or evolve during the course of illness; for example, patients with hemorrhagic or cardiogenic shock may subsequently develop septic shock [33].

Epidemiology

Up to one-third of patients admitted to the ICU are in circulatory shock, and early recognition of the condition is vital if subsequent tissue injuries are to be avoided. Shock can be categorized according to the underlying cause, including septic shock, cardiogenic shock, anaphylactic shock and shock associated with burns, trauma and hemorrhage. In the 1,679 ICU patients in the European Sepsis Occurrence in Acutely Ill Patients II (SOAP II) trial, septic shock was the most frequent cause of shock, accounting for 62 % of cases, followed by cardiogenic shock (17 %) and hypovolemia (16 %) [33]

Diagnosis of Shock

The diagnosis of shock is based on a combination of clinical, hemodynamic, and biochemical findings rather than hypotension alone. Clinical evidence of impaired tissue perfusion includes abnormalities in the peripheral circulation (cold, clammy, pale, or cyanotic skin), renal function (urine output <0.5 mL/kg/h), and neurological status (confusion, disorientation, or reduced consciousness). Importantly, normal blood pressure does not exclude shock, as compensatory vasoconstriction may maintain arterial pressure despite significant tissue hypoperfusion [32].

PLACENTA PREVIA MANAGEMENT

Placenta previa is defined as the condition where the placenta overlies the internal cervical os and is interposed between the presenting part of the fetus and the birth canal, thus blocking the natural route of vaginal delivery. Placenta previa poses major risks to both maternal and perinatal health and is an absolute indication for cesarean delivery (CD). The primary concern is the risk of life-threatening antepartum hemorrhage due to placental separation from the lower uterine segment as the cervix dilates, often necessitating massive transfusion and emergent delivery. Additional relevant implications of placenta previa are postpartum hemorrhage and preterm birth [34].

Why Placenta Previa Demands Greater Anesthetic Considerations

In a normal pregnancy, anesthetic care during delivery is often routine and focused on pain management or standard cesarean section protocols. In contrast, placenta previa represents a high-risk obstetric scenario because of the extraordinary risk of sudden, massive maternal hemorrhage. Approximately 45% of patients with symptomatic placenta previa require emergency cesarean delivery for peripartum hemorrhage [35].

Because active bleeding represents an acute obstetric emergency, anesthesiologists must be prepared to manage severe hemodynamic instability, secure rapid-access large-bore intravenous lines, and initiate massive transfusion protocols. Consequently, the anesthesiologist is a core member of a multidisciplinary "placenta team" that must be activated at 30 weeks of gestation to coordinate patient safety, plan operative strategies, and conduct team briefings [36].

- **Associated Complications**

The clinical course of placenta previa is frequently complicated by several major maternal and fetal risks **[37]:**

- **Peripartum and Postpartum Hemorrhage (PPH):** Previa is a leading cause of severe obstetric hemorrhage. This bleeding is often sudden and can quickly lead to hypovolemic shock.
- **Placenta Accreta Spectrum (PAS) Disorders:** Placenta previa is highly correlated with abnormal placental adherence and invasion (PAS), particularly in patients with a history of prior cesarean delivery. PAS dramatically increases the risk of massive, life-threatening hemorrhage and often requires an emergency peripartum hysterectomy.
- **Preterm Birth and Urgent Cesarean Delivery:** Antepartum bleeding often necessitates delivery prior to term. A short cervical length (<3 cm) in asymptomatic previa patients is a key predictor, associated with marked increases in hemorrhage (79% vs. 28%) and urgent cesarean sections (69% vs. 21%).
- **Maternal Morbidity:** Severe complications include the need for massive blood transfusions, intensive care unit (ICU) admission, and long-term psychological impacts such as posttraumatic stress disorder (PTSD) or postpartum depression.

- **Perioperative Anesthetic and Surgical Management**

Managing these patients requires a highly coordinated, phased multidisciplinary approach [38]:

- **Preoperative Planning (30–34 Weeks):** Diagnosis is confirmed via transvaginal ultrasound, and the multidisciplinary placenta team (including anesthesia, urology, interventional radiology, and neonatology) is activated. At 34 weeks, a detailed operative and anesthetic plan is formulated, including preparation for massive hemorrhage.
- **Intraoperative Anesthetic Choices:** Anesthesiologists must decide between general and regional (epidural/spinal) anesthesia. While regional anesthesia can be safely used in stable, elective cases, general anesthesia is often preferred or required during active, uncontrolled hemorrhage, or when there is a high suspicion of invasive PAS that may require a prolonged, complex cesarean hysterectomy. Comparative literature in the paper highlights maternal hemodynamics, blood loss, and neonatal outcomes under general vs. epidural anesthesia for placenta previa totalis.
- **Surgical Hemostatic Coordination:** The anesthesia team must work in close coordination with the surgical team as various hemostatic interventions are deployed, including:
  - Prophylactic internal iliac artery balloon occlusion to temporarily reduce pelvic blood flow.
  - Uterine tourniquet devices to occlude uterine arteries.
  - Bakri balloon tamponade or intrauterine vacuum-induced hemorrhage-control devices to mechanically control lower segment bleeding.
  - Compressive sutures of the lower uterine segment.
  - Administration of prophylactic tranexamic acid (TXA) to mitigate blood loss [39].

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