Principles of Critical Care in Obstetrics

Volume I

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Part I

Introduction to Critical Care

Epidemiology of Critical Illness in Obstetrics

1

Shikha Singh and Narendra Malhotra

Critical illness in pregnancy as a morbidity outcome is difficult to define and therefore difficult to measure and study precisely. As stated by Harmer, "Death represents the tip of the morbidity iceberg, the size of which is unknown" [1]. The stage at which any condition becomes severe enough to be classified as a critical illness has not been clearly defined. However, it may be helpful to consider critical illness as impending, developing, or established significant organ dysfunction, which may lead to long-term morbidity or death. This allows some flexibility in the characterization of disease severity since it recognizes condition that can deteriorate rather quickly in pregnancy.

It has been suggested that most women suffering a critical illness in pregnancy are likely to be in an intensive care unit. These cases have been described by some as "near-miss" mortality cases. There are many conditions in pregnancy that occur frequently and require special medical care, but do not actually become critical illness. Most women with these complications have relatively uneventful pregnancies that result in good outcome. Nevertheless, each of these conditions can be associated with significant complications that have the potential for serious morbidity, disability, and mortality.

The successful epidemiologic evaluation of any particular disease or condition has several prerequisites. Two of the most important prerequisites are that the condition should be accurately defined and that there should be measurable outcomes of interest. Another requirement is that these must be some systematic way of data collection or surveillance that will allow the measurement of the outcomes of interest and associated risk factors.

Historically, surveillance of pregnancy-related critical illness has focused on the well-defined outcome of maternal mortality in order to identify illnesses or conditions that might have led to maternal death. Maternal mortality data collection is well established in many places, but specific surveillance systems that track severe complications of pregnancy not associated with maternal mortality are rare. Examination of complicating conditions associated with maternal hospitalization can provide information on the types of conditions requiring hospitalized case.

ICU Admissions and Maternal Mortality

Evaluation of obstetric admissions to intensive care units (ICUs) may be one of the best ways to approach surveillance of critical illness in pregnancy. Unfortunately, there is no publicly available

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population-based database for obstetric admissions to ICU that provides sufficiently detailed information to allow in-depth study of these conditions.

The prevalence of obstetric patients requiring critical care ranges from 100 to 900 per 100,000 gestations [2–4]. The maternal mortality due to critical illness is 12–20 % but varies significantly between developing and developed countries (440/10,000 deliveries in India vs. 12/100,000 deliveries in the USA) [5].

A review of 33 studies between 1990 and 2006 by Ananth and Smulian [6], involving 19, 55, and 111 deliveries, found an overall obstetric admission rate to ICU of 0.07–0.89 %.

According to the study, reported maternal mortality for critically ill obstetric patients admitted to an ICU is approximately 8.4 % with range of 0–33 % in different setups. These reports are from developed countries and less developed countries have much higher mortality rates. In a study on obstetric admissions to ICU of King Edward Memorial Hospital (KEMH), Mumbai, by Munnur et al. [5], the maternal mortality was as high as 25 % in Indian patients. Factors leading to adverse outcomes in Indian subjects were lack of antenatal care, delayed presentation, higher severity of illness at presentation, and lack of an aggressive obstetric approach. Organization of health care services and social customs also contributed to low antenatal care and lack of aggressive obstetric approach. Panchal et al. [7], in a retrospective analysis of 1,023 ICU admissions, showed that age, race, hospital type, volume of deliveries, and source of admission were all associated with risk of admission to the ICU in obstetrics.

Illnesses Responsible for Obstetric ICU Admissions

Data pooled by Munnur et al. [5] provides sufficient detail about the primary indication for the obstetrics ICU admission (Table 1.1). It is no surprise that hypertensive disease and obstetric hemorrhage were responsible for over 50 % of the primary admitting diagnoses. Specific organ system dysfunction was responsible for the majority of remaining admissions. Of those, pulmonary, cardiac, and infectious complications had the

Table 1.1 Medical disorders requiring intensive care unit (ICU) admission [5]

Medical disorders	King Edward Memorial Hospital ($n=754$)	Ben Taub General Hospital ($n=174$)
Community-acquired pneumonia	23 (3.1 %)	5 (2.9 %)
Urinary tract infection	2 (0.3 %)	18 (10.3 %)
Malaria	75 (10.0 %)	0
Hematological disorder	12 (1.6 %)	1 (0.6 %)
Congenital heart disease	2 (0.3 %)	2 (1.2 %)
Rheumatic heart disease	16 (2.1 %)	2 (1.2 %)
Aspiration pneumonia	23 (3.1 %)	6 (3.5 %)
Diabetes mellitus	16 (2.1 %)	4 (2.3 %)
Chronic renal failure	4 (0.5 %)	1 (0.6 %)
Trauma	0	1 (0.6 %)
Drug abuse	0	5 (2.9 %)
Rheumatological disorders	2 (0.3 %)	2 (1.2 %)
Anaphylaxis	0	2 (1.2 %)
Asthma	1 (0.1 %)	5 (2.9 %)
DVT/pulmonary embolism	5 (0.7 %)	2 (1.2 %)
Malignancy	1 (0.1 %)	6 (3.5 %)
Acute abdomen	6 (0.8 %)	10 (5.7 %)
CNS infection	6 (0.8 %)	0
Viral hepatitis	47 (6.2 %)	0
Bacteremia	13 (1.7 %)	8 (4.6 %)
Attempted suicide (poisoning/drug overdose)	13 (1.7 %)	1 (0.6 %)
Transfusion reaction	2 (0.3 %)	1 (0.6 %)
Cardiac arrest prior to ICU admission	21 (2.8 %)	1 (0.6 %)
Endocrine	8 (1.1 %)	1 (0.6 %)
Arterial disease	1 (0.1 %)	1 (0.6 %)
Intracranial hemorrhage	9 (1.2 %)	1 (0.6 %)
Cerebral venous thrombosis	26 (3.5 %)	0
Tetanus	2 (0.3 %)	0
Typhoid	1 (0.1 %)	0
Leptospirosis	2 (0.3 %)	0
Cerebral infarction	2 (0.3 %)	0

Table 1.2 Obstetric conditions requiring intensive care unit (ICU) admission [5]

Medical disorders	King Edward Memorial Hospital ($n=754$)	Ben Taub General Hospital ($n=174$)
Preeclampsia/eclampsia	343 (45.5 %)	74 (42.5 %)
Postpartum hemorrhage	115 (15.3 %)	32 (18.4 %)
IUFD	94 (12.5 %)	8 (4.6 %)
Postabortal/puerperal sepsis	49 (6.5 %)	26 (14.9 %)
HELLP syndrome	42 (5.6 %)	31 (17.8 %)
Abruptio placentae	43 (5.7 %)	15 (8.6 %)
Acute fatty liver of pregnancy	33 (4.4 %)	3 (1.7 %)
Antepartum hemorrhage	27 (3.6 %)	4 (2.3 %)
Chorioamnionitis	7 (0.9 %)	22 (12.6 %)
Abortions	18 (2.4 %)	6 (3.5 %)
Abnormal adherence of placenta	8 (1.1 %)	9 (5.2 %)
Peripartum cardiomyopathy	4 (0.5 %)	10 (5.8 %)
Uterine rupture	6 (0.8 %)	3 (1.7 %)
Amniotic fluid embolism	4 (0.5 %)	1 (0.5 %)

greatest frequency. It was also clear from these reports that both obstetric and medical complications of pregnancy were responsible for the obstetric ICU admissions (Tables 1.1 and 1.2).

Causes of Mortality in Obstetric ICU Admissions

When specific causes of mortality for the obstetric ICU admissions were reviewed by Ananth et al., 26 studies gave sufficient data to assign a primary etiology for maternal death (Table 1.3) [8].

Of a total of 138 maternal deaths, over 57 % were related to complications of hypertensive diseases, pulmonary illnesses, and cardiac diseases. Other deaths were commonly related to complications of hemorrhage, bleeding into the central nervous system (CNS), malignancy, and infection. More importantly, despite identified primary etiology for the maternal deaths, nearly all cases were associated with multiple organ dysfunction score (MODS), which again emphasizes the complex condition of these critically ill women.

In a retrospective analysis by Munnur et al. of 10-year data (1992–2001) pertaining to 928 critically ill obstetric patients from King Edward Memorial Hospital (KEMH), Mumbai, being compared to a similar patient population at Houston County Hospital, the mean age of Indian patients was 25.4 ± 4.6 years, of which only 26 % had received prenatal care (at least two prenatal visits) as compared to 86 % of Western patients; only 60 % of Indian patients went for admission within 24 h of onset of illness (vs. 90 % for Western patients), with mean APACHE II score of 16 on Day 1 (vs. ten for Western patients), with altered mental status (50 %), bleeding (40 %), seizures (30 %), fever (27 %), dyspnea (23 %), and jaundice (21 %) being the most common manifestations in this subset (vs. fever 55 %, bleeding 53 %, dyspnea 44 % in Western population).

In both ICUs, 70 % of critically ill pregnant patients were admitted with obstetric disorders. The incidence of preeclampsia/eclampsia (45 %), PPH (15 %), abruptio placentae (6 %), acute fatty liver of pregnancy (4 %), and APH (4 %) in Indian patients was similar to their Western counterparts. Medical disorders were responsible for only 30 % of ICU admissions.

The incidence of organ dysfunction in Indian subjects in the abovementioned study was reported as follows: neurological (63 %), hematologic (58 %), renal (50 %), respiratory (46 %), cardiovascular (38 %), and hepatic (36 %). DIC was seen in 23 % of subjects, while the maximum MODS score was 5 [3–7]. The major causes of CNS dysfunction in Indian subjects were

Table 1.3 Identified primary causes of mortality in obstetric admissions to ICUs [8]

Identified etiology	Number	Percentage
Hypertensive diseases		
Hypertensive crisis with renal failure	36	26.1
HELLP syndrome complications		
Eclampsia complications		
Other hypertensive disease complications		
Pulmonary	27	19.6
Pneumonia complications		
Amniotic fluid embolus		
Adult respiratory distress syndrome		
Pulmonary embolus		
Cardiac	16	11.6
Eisenmenger's complex		
Myocardial infarction		
Arrhythmia cardiomyopathy		
Unspecified		
Hemorrhage	14	10.1
Central nervous system hemorrhage	10	7.2
Arteriovenous malformation		
Brain stem hemorrhage		
Intracranial hemorrhage		
Infection	11	8.0
Sepsis		
Tuberculosis meningitis		
Malignancy	8	5.8
Hematologic	2	1.5
Thrombotic thrombocytopenic purpura		
Gastrointestinal	1	0.7
Acute fatty liver of pregnancy		
Poisoning/overdose	2	1.5
Anesthesia complication	1	0.7
Trauma	1	0.7
Unspecified	9	6.5
Total	138	100 %

eclampsia, cerebral malaria, CNS infections, hepatic coma, and cerebral venous thrombosis. Important causes of renal failure were preeclampsia, DIC, PPH, hemorrhagic shock, severe malaria, leptospirosis, and acute fatty liver of pregnancy. Hematological failure was predominantly due to bacterial sepsis and DIC. Respiratory failure was due to community-acquired pneumonia, acute asthma, and ARDS due to abdominal sepsis. Cardiovascular failure was due to obstetric shock and rheumatic heart disease. Hepatic dysfunction was predominantly due to acute viral hepatitis in Indian subjects and due to HELLP syndrome in Western subjects.

To conclude, understanding the nature of critical illness in pregnancy is an important and evolving process. However, our currently available tools and databases for examining these patients still need improvement [9]. As our understanding of critical illnesses continues to mature, we will hopefully gain greater insight into the specific nature of these conditions that will lead to improved prevention strategies and better therapies for the diseases when they occur. These data will improve our ability to plan and allocate the necessary resources to adequately care for these often complex and severe illnesses. A multidisciplinary approach to manage these patients is required, and it can also be well guided by epidemiology statistics.

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Pregnancy-Induced Alterations in Physiology and Laboratory Reports

2

C.N. Purandare, Madhuri Patel, and Surekha Tayade

Introduction

Pregnancy in the human female is a unique state in which virtually all maternal systems are dramatically altered to permit the sustenance and growth of the intrauterine conceptus. Major physiological changes include cardiovascular, hematologic, metabolic, renal, and respiratory changes, most of which begin soon after conception and continue throughout pregnancy till until late gestation. These changes affect various patient laboratory test results. The body can generally compensate for these changes [1]. However, in the presence of conditions such as anemia, clotting disorders, bleeding during pregnancy, preeclampsia, and trauma caused by motor vehicle accident, the body may not be able to compensate for the changes. At this point, laboratory values can become significantly skewed from the values normally noted during pregnancy. In caring for pregnant women and their unborn infants, it is important for the healthcare provider to understand the normal physiologic changes that occur during pregnancy. The provider can utilize various laboratory tests and diagnostic tools to assess the magnitude of these changes and to identify abnormal changes. It is imperative that they should be aware of both the normal and abnormal laboratory values to be able to make decisions about clinical management of the woman. However, very few laboratories provide clinicians with normal reference ranges during pregnancy. This chapter makes an attempt to discuss the physiological changes and alterations in the laboratory values that occur during pregnancy.

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Changes in Hematological System

Maternal blood volume increases during pregnancy, and this involves an increase in plasma volume as well as in red cell and white cell volumes [2]. The plasma volume increases by

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40–50 %, whereas the red cell volume goes up by only 15–20 %, which causes a situation that is described as "physiological anemia of pregnancy" (normal hemoglobin, 12 g/dL; hematocrit, 35 %) [3]. Because of this apparent hemodilution, blood viscosity decreases by approximately 20 %. The exact mechanism of this increase in plasma volume is unknown. However, several hormones such as renin-angiotensin-aldosterone, atrial natriuretic peptide, estrogen, and progesterone may be involved in this interesting phenomenon. Two current hypotheses attribute the increase to:

1. An underfill state caused by initial vasodilatation, which stimulates hormones such as renin, angiotensin, and aldosterone.
2. An overfill state characterized by an early increase in sodium retention (due to an increase in mineralcorticoids) that retains fluid, causing an increase in blood volume. Levels of clotting factors I, VII, VIII, IX, X, and XII and the fibrinogen count are elevated during pregnancy as well. At present, the majority of observers report a statistically significant fall in platelet count as pregnancy progresses [4]. A recent study that observed an increase in thrombopoietin with the advancement of the gestational age also confirmed this finding [4]. Systemic fibrinolysis also may increase slightly.

White blood cell (*WBC*) *counts*, especially neutrophils, increase naturally during pregnancy. During active labor, there may be another normal increase, even in the absence of infection. In nonpregnant patients, a normal WBC count is somewhere between 5 and 10 (5000–10,000 cells/mm^3), but for pregnancy, those normal values can be between 6 and 16 in the third trimester and may reach 20–30 in labor and early postpartum. When evaluating for infection, therefore, you need to look for other clinical indicators, such as increased temperature, bacteriuria, WBC in urine, uterine tenderness, and fetal tachycardia, and document them [5, 6].

Normal hematologic values		
	Nonpregnant	Pregnant
Hemoglobin (HGB)	12–16 g/dl	11.5–15 g/day
Hematocrit (HCT)	36–48 %	32–36.5 %
Red blood cells (RBC)	4–5.3 × 10^6/cu mm	2.81–4.49 × 10^6/cu mm
White blood cells (WBC)	4–10.6 × 10^3/cu mm	6–20 × 10^3/cu mm

To evaluate the genesis of anemia, the following laboratory values are taken into consideration:

If anemia is from *low iron*, you will see the following results: [1]

- Microcytic/hypochromic red blood cells (smaller/paler than normal)
- Serum ferritin <11 ng/ml (mg/L)
- Transferrin saturation level <16 %
- Serum iron <30 mcg/dl
- Mean corpuscular hemoglobin concentration (MCHC) <30 g/dl
- Iron-binding capacity increased (>400 mcg/dl)

Pregnancy is typically considered a *hypercoagulable state*—meaning that most pregnant women clot more readily than normal and are predisposed to deep-vein thrombosis or other clot-related conditions. During pregnancy, there is an increase in certain factors in the clotting cascade due to normal adaptation (see table). Platelets are usually unchanged in pregnancy, and increased levels of platelets are rare. Normal levels should be 140,000–300,000 per mm^3.

Normal levels for clotting factors		
	Nonpregnant (%)	Pregnant
Factor V	50–147	Increased
Protein S	54–160	30–70 %
Antithrombin	80–130	Should remain stable (a decrease indicates increased thrombosis risk)

Clinical Implications

The increased blood volume serves several important functions: (1) It takes care of the increased circulatory need of the enlarging uterus as well as

the needs of the fetoplacental unit. (2) It fills the ever-increasing venous reservoir. (3) It protects the parturient from the bleeding at the time of delivery. (4) Parturients become hypercoaguable as the gestation progresses. It takes about 6 weeks after delivery for the blood volume to return to normal.

Values for disseminated intravascular coagulation

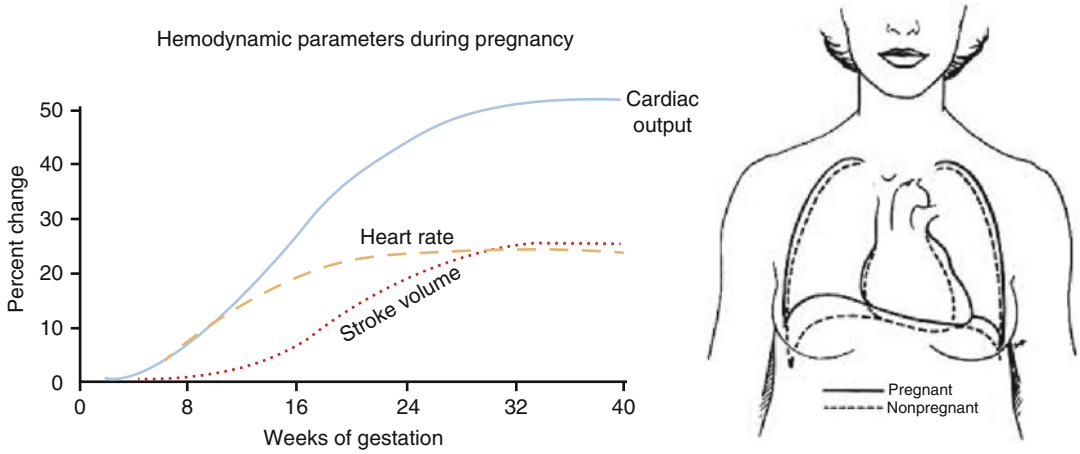
	Normal	DIC
Fibrinogen (factor I)	170–470 mg/dl	↓
Platelets	150,000–400,000 per mm^3	↓
Fibrin split products[a]	<10 mcg/ml	↑
D-dimer[b]	0–0.5 mcg/ml	↑

[a]Also called fibrin degradation products (FSP or FDP) when clots are broken down
[b]D-dimer is made when clots are broken down

Changes in the Cardiovascular System

An increase in cardiac output is one of the most important changes of pregnancy. *Cardiac output increases by 30–40 % during pregnancy, and the maximum increase is attained around 30 weeks' gestation*. The increase in heart rate lags behind the increase in cardiac output initially and then ultimately rises by 10–15 beats per minute by 28–32 weeks' gestation. The increase in cardiac output initially depends mainly on the rise in stroke volume, and later the increase in heart rate also becomes an important factor. With Doppler and M-mode echocardiography technique, increases in end-diastolic chamber size and total left ventricular wall thickness have been observed in recent years. Cardiac output can vary depending on the uterine size as well as on the maternal position at the time of measurement. The enlarged gravid uterus can cause aortocaval compression while the pregnant woman is in the supine position, and this will lead to reduced venous return and ultimately maternal hypotension. This effect will be exaggerated in parturients with polyhydramnios or multiple gestations.

Cardiac output increases further during labor and may show values 50 % higher than prelabor values. In the immediate postpartum period, cardiac output increases maximally and can rise 80 % above prelabor values and approximately 100 % above nonpregnant measurements. The increase in stroke volume as well as in heart rate maintains the increased cardiac output [7].



Clinical Implications

An increased cardiac output might not be well tolerated by pregnant women with valvular heart disease (e.g., aortic or mitral stenosis) or coronary arterial disease.

Changes in the Respiratory System

Changes in the respiratory parameters start as early as the 4th week of gestation. Minute ventilation is increased at term by about 50 % above nonpregnant values. The increase in minute ventilation is mainly due to an increase in tidal volume (40 %)

and, to a lesser extent, to an increase in the respiratory rate (15 %) [8]. Alveolar ventilation is greatly increased as the tidal volume increases without any change in the anatomic dead space. At term the PCO_2 value is decreased (32–35 mmHg). Increased progesterone concentrations during pregnancy decrease the threshold of the medullary respiratory center to carbon dioxide [9, 10].

Clinical Implications

A decreased functional residual capacity as well as increased oxygen consumption can cause a rapid development of maternal hypoxemia.

Changes in the Renal System

The glomerular filtration rate is increased during pregnancy because of increased renal plasma flow [11]. A rise in the filtration rate decreases plasma blood urea nitrogen (BUN) and creatinine concentrations by about 40–50 %. Tubular reabsorption of sodium is increased. However, glucose and amino acids might not be absorbed as efficiently; hence, glycosuria and aminoaciduria may develop in normal gestation [12, 13]. The renal pelvis and ureters are dilated, and peristalsis is decreased.

Normal values for renal function

	Nonpregnant	Pregnant
Serum creatinine	0.6–1.4 mg/dl	0.53–0.9 mg/dl decrease
Serum BUN	7–31 mg/dl	8–10 mg/dl decrease
Serum uric acid	2.4–8.2 mg/dl	2–5.8 mg/dl
Urine Cr clearance	90–130 mL/min	150–200 mL/min
Urine uric acid	150–990 mg/24 h	Increases
Urine glucose	60–115 mg/dl	Increases

Clinical Implications

Normal parturients' BUN (8–9 mg/dl) and creatinine (0.4 mg/dl) values are 40 % less than in nonpregnant women. So nonpregnant values in parturients will suggest abnormal kidney function. Physiological diuresis during the postpartum period occurs between the 2nd and 5th days. The glomerular filtration rate and BUN concentration slowly return to nonpregnant values by the 6th postpartum week [13].

Changes in the Gastrointestinal System

Gastrointestinal motility, food absorption, and lower esophageal sphincter pressure are decreased during pregnancy, probably due to an increased level of plasma progesterone [14]. Lower esophageal sphincter pressure is decreased during pregnancy; on the other hand, intragastric pressure is increased during the last trimester. Heartburn during pregnancy is the result of reduced barrier pressure [15]. The gastric emptying time of solid as well as liquid material is not changed during pregnancy. Because of decreased plasma gastrin concentration during pregnancy, there is reduction in the total acid content of the stomach. Gastric emptying time is significantly slower during labor, and hence, gastric volume is increased.

In addition to increased production of lipids and certain clotting factors, some enzymes found within the liver are also increased without indicating pathology. It is important to distinguish a normal rise in these levels from a pathologic change caused by organ damage or destruction arising, for example, from preeclampsia or hepatitis. In preeclampsia, microclots in the liver and capsular edema are danger signs, and if clotting factors become affected, the patient is at a high risk for disseminated intravascular coagulation (DIC). Diagnoses are not based upon a single abnormal value [16].

Normal hepatic values

Liver enzymes	Nonpregnant	Pregnant
Alanine transaminase (ALT)	14–67 U/L	Unchanged
Aspartate aminotransferase (AST)	6–58 U/L	Unchanged
Alkaline phosphatase (ALP)	38–150 lMU/ml	> up to 2–4 times
Lactate dihydrogenase (LDH)	117–224 U/L	Upper end of normal to 700 U/L

Changes in the Musculoskeletal System

The hormone relaxin is responsible for both the generalized ligamentous relaxation and the softening of collagenous tissues.

Clinical Implications

Relaxation of ligaments and collagen tissue of the vertebral column is the main cause of lordosis during pregnancy.

Changes in the Dermatological System

Hyperpigmentation of certain parts of the body such as the face, neck, and midline of the abdomen is not uncommon during pregnancy. Melanocyte-stimulating hormone is responsible for this change. Enlargement of the breasts is an integral part of the physiological changes of pregnancy [17].

Changes in the Ocular System

Intraocular pressure has been shown to decrease during pregnancy; this is related to (1) increased progesterone levels, (2) the presence of relaxin, and (3) decreased production of aqueous humor due to increased secretion of human chorionic gonadotropin [18].

Maternal Physiological Changes [11]

Enlarged breasts, especially in parturients with short necks, may make intubation extremely difficult. A short-handled laryngoscope as described by Datta and Briwa may be helpful in such cases [19]. Changes in intraocular pressure in parturients may produce visual disturbances as well as contact lens intolerance.

Normally, pregnant women require calories additional to the normal daily requirement. These recommendations, varying from country to country, also suggest the addition of protein, iron, and other mineral and vitamin supplements to provide the necessary materials for fetal *and* maternal welfare throughout the pregnancy. However, it is understood that appropriate nutrition is important for maximizing the possibility of healthy offspring. Hytten and Leitch [12] and others [14] have pointed out that it is difficult to focus on nutrition alone as a factor in the growth and development of normal babies. Women who are appropriately nourished during the course of pregnancy are economically better and educated and have greater access to the medical antepartum care that seems to be associated with improved pregnancy outcomes. Antepartum nutrition, however, continues to be an area of great interest because the balanced intake of food in pregnant women is a simple intervention that may have a significant impact on the outcome of reproduction. The current recommendations proposed by the Food and Nutrition Board of the US National Science Foundation are listed in Table 2.1.

Skin Changes

A lot of changes in the skin are observed during pregnancy. Hyperpigmentation is seen in the areolae, the perineal skin, the anal region, the inner thighs, and the linea nigra, which appears on the abdominal wall. Melasma or chloasma is a blotchy, sharply marginated hyperpigmentation that occurs on the face of dark-haired and dark-complexioned women. It is most often centrally distributed on the face.

By the third trimester of pregnancy, vascular "spiders" due to circulating estrogens occur in

Table 2.1 Recommended dietary allowances (Revised in 2005)

Nutrient	For nonpregnant woman	For pregnant woman
Protein	45 g/day	+30 g/day
Calories	2100	+300
Calcium	1000 mg/day	+1000 mg/day
Iron	18 mg/day	+9 mg/day
Folic acid	400 µg/day	+200 µg/day
Ascorbic acid	75 mg/day	+10 mg/day

about 67 % of white patients and 11 % of black patients. These lesions occur on the neck, throat, face, and arms.

Striae are common among women in late pregnancy. There seems to be a familial tendency in the occurrence of these lesions. When they occur, they first appear during the 6th and 7th months of gestation on the abdominal skin; they then occur on the breasts, upper arms, lower back, buttocks, and thighs. They have been related to a combination of stretching of the skin and increased levels of corticosteroids and estrogen in pregnancy.

Summary

The myriad changes that occur during the pregnant state have to be well understood by the health provider to analyze the condition of the patient. An understanding of some of the major mechanisms that produce these changes is helpful in the analysis of symptoms and problems that arise during the course of a normal gestation. When associated disease is present, understanding of these alterations becomes more important in that they must be distinguished from pathophysiologic changes brought by the disease process. The interaction between disease and gestational physiology may make the appropriate diagnosis and management of the pregnant woman difficult. When a pregnant woman requires medical or surgical therapy, the consultative services of an obstetrician or clinician trained in the complexities of maternal physiology are absolutely critical to the proper management of clinical problems.

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Ethics in the Setting Up of Obstetric HDU and ICU

3

K. Muhunthan and Sabaratnam Arulkumaran

Introduction

Childbirth is a major life event for women and their families. However, in a small proportion, severe and sometimes life-threatening complications occur during pregnancy. Such critically ill women should receive the same standard of care for both their pregnancy-related and critical care needs, delivered by professionals with the same level of competences irrespective of whether these are provided in a maternity or general critical care setting [1].

Maternal critical care is an area which is less discussed than other parts of obstetric care. However, there has been a growing need to address this area from a national and international point of view: to collate, to standardise, to share and to learn. Maternal morbidity and mortality has been analysed by different methods in majority of countries. What has become apparent is that there is still a significant number of morbidity and mortality associated with suboptimal care [2].

Critical care in pregnancy poses a major challenge to clinicians as it requires consideration of the physiological changes associated with pregnancy and the need to reassure the well-being of the foetus [3].

In order to safeguard the right of the woman to live, to have good health and to minimise unacceptable outcome of obstetric morbidity and mortality, it is important to address essential and ethical aspects in planning and setting up an obstetric HDU and ICU.

Implementing a Standardised System on Recognising the Level of Care Needed

It is imperative that all carers understand the terminology used in setting up and organising HDU and ICU to provide care for critically ill patients in the peripartum period.

Maternal critical care, high dependency care and high-risk maternity care are not interchangeable, the term critical care having a more precise definition. It is also recommended that the terms ‘high dependency’ and ‘intensive care’ be replaced by the term ‘critical care’ [4].

It is important to define the level of critical care required by the mother depending on the number of organs requiring support and the type of support required. Such accepted definitions will provide a platform for the woman to receive the needed treatment. Prioritisation of patients based on the needed care is an important key for proper communication and timely admission. Often these facilities are in high demand, and

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ethical practice demands only those who need care in these facilities are admitted.

It is ethical to term facilities as HDU and ICU only if the service provided to mothers meets with the expected level of care [5].

Worldwide several definitions are adopted, and the four levels of critical care as defined by the Intensive Care Society are as follows [6]:

Level 0: Patients whose needs can be met through normal ward care

Level 1: Patients at risk of their condition deteriorating and needing a higher level of observation or those recently relocated from higher levels of care

Level 2: Patients requiring invasive monitoring/intervention that include support for a single failing organ system (excluding advanced respiratory support)

Level 3: Patients requiring advanced respiratory support (mechanical ventilation) alone or basic respiratory support along with support of at least one additional organ

Thus, maternal critical care can be distinguished from 'high-risk' obstetrics because the foetal issues are excluded. The maternal risk factors or obstetric complications that require closer observations or intervention, but, not support of an organ system, are outside the levels 2 and 3 of critical care.

Predicting the Population Requirement for Obstetric Critical Care Beds

Provision of critical care through HDU and ICU to women in the peripartum period needs to meet the requirement of a country or region.

Birth rates are measured in various ways in various countries, and using these figures and the maternal mortality and morbidity data, the health care provider must be able to estimate scientifically the numbers of adult critical care beds required [7]. Another approach of projecting the needed number of facilities is to audit pregnant women and those who were recently pregnant and were admitted to adult general critical care units during the previous years.

A substantial portion of critical care may have been and could be provided through high dependency, rather than intensive care set-up. In order to plan the setting up of such facilities, it is important to differentiate the level of critical care required by any population.

Workforce Development and Staff Competences

Lead professionals in maternity services have a responsibility to ensure staffs are competent in the early recognition of acutely ill and deteriorating patients and are able to perform the initial resuscitation of such patients. This can be achieved by regular certified courses at acute illness management.

Whichever the training modality is practised, assessment of competences is essential. Multidisciplinary scenario-based training in the form of skills and drills has been found to be valuable, particularly when developing team drills for life-threatening clinical situations.

In addition it is imperative that the staffs who are involved in the care of acutely ill patients in the hospital are competent with regard to knowledge, skills and attitudes required for safe and effective treatment and care along a chain of response. Figure 3.1 and the Table 3.1 give the example of safe and effective treatment and care along the chain of response [8].

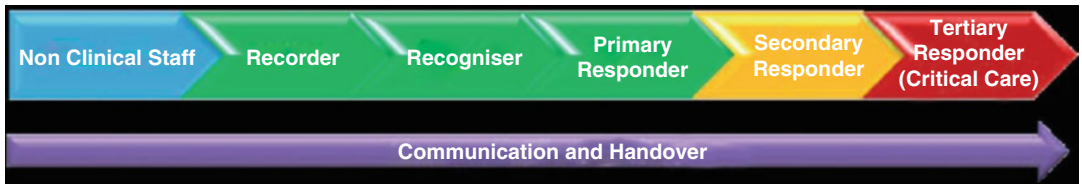

Fig. 3.1 Safe and effective treatment and care along the chain of response

Table 3.1 Safe and effective treatment of care along the chain of response

Non-clinical supporter who may also be the 'alerter' and may include the woman or visitor
The recorder who takes designated measurements and records observations and information. In maternity services this could be a maternity support worker, health care assistant or midwife
The recogniser who monitors the patient's condition; interprets designated measurements, observations and information and adjusts the frequency of observations and level of monitoring. In the maternity setting this could be a midwife, recovery or other nurse working within the unit or foundation doctor
The primary responder who goes beyond recording and further observation by interpreting the measurements and initiating a clinical management plan, e.g. commencing oxygen therapy, insertion of airway adjuncts and selection and administration of a bolus of intravenous fluids. This would be a junior doctor or specialist trainee or foundation doctor with appropriate competencies
The secondary responder who is likely to be called to attend when the patient fails to respond to the primary intervention or continues to 'trigger' or 'retrigger' a response. This individual will assess the clinical effect of the primary intervention, formulate a diagnosis, refine the management plan, initiate a secondary response and have the knowledge to recognise when referral to critical care is indicated. This would be an obstetric or anaesthetic specialist trainee
Tertiary responder: This role encompasses the acute care competencies, such as advanced airway management, resuscitation, clinical assessment and interpretation of acutely ill obstetric patients. In the maternity unit, this role is routinely provided by consultant anaesthetists with certified training in obstetric anaesthesia. The acute care competencies required focus primarily on the clinical and technical aspects of care and the delivery of effective patient management. They assume the possession and application at every level of complementary generic competencies such as record-keeping, team working, interpersonal skills and clinical decision-making. Of particular note in this context is the ability to rapidly access hospital information systems and retrieve patient information, such as blood results and x-rays

Care of the Critically Ill Obstetric Patient in Regular Settings

Before a decision is taken that a woman would need critical care in HDU or ICU, it is obvious that they may have been cared in a less intense set-up like a regular obstetric ward. Hence it is ethical that consultant-led obstetric services should have adequate facilities, expertise, capacity and back-up for timely and comprehensive obstetric emergency care, including the possibility of transfer to intensive care.

It is therefore important that maternity and critical care services design pathways at a local level which ensure that a critically ill parturient accesses equitable care when needed. Such pathways should facilitate mother and baby remaining together unless precluded by a clinical reason. Such arrangements should detail defined escalation arrangements for bringing critical care, midwifery and obstetric competences into the maternity or critical care unit. These arrangements need to take into account local configuration, size and complexity of maternity and critical care services.

Transfer to Critical Care Area from a Maternity Ward

Women may require transfer to a critical care area for a higher level of care (both level 2 and level 3) either pre-delivery or postpartum. Such transfers need to satisfy an accepted standard similar to the ICS Standards' 'Guidelines for the transport of the critically ill adult' and need to be accompanied by an additional plan addressing the maternal, foetal and postnatal needs of the patient [9]. The plan should also indicate whether or not pre-delivery shared care between obstetrics and critical care is essential.

All maternity units must have the facilities and staff to resuscitate, stabilise and transfer critical care patients [10]. The transfer should take place with an appropriately trained practitioner. Although this is generally an anaesthetist, it can be a specific 'transfer' clinician or an intensivist. Positioning of the pregnant patient poses additional risks in the avoidance of aortocaval compression.

Transfer to Maternity Ward from Critical Care Area

After the decision to transfer a patient from a critical care area to the maternity ward has been made, she should be transferred as early as possible during the day.

Transfer from critical care areas to the maternity ward between 22.00 and 07.00 should be avoided whenever possible [9]. Both the critical care and receiving maternity ward teams should take shared responsibility for the care of the patient being transferred [11].

They should jointly ensure that:

- There is continuity of care through a formal structured handover from critical care staff to ward staff.
- There should be a supported written care plan with instructions to medical and nursing staff.
- The receiving staff, with support from critical care staff if required, should deliver the agreed plan.
- The formal structured handover of care should include:
  I. A summary of critical care stay, including diagnosis, treatment and outstanding investigations
  II. A monitoring plan detailing the frequency of observations
  III. A plan for ongoing treatment including drugs and therapies, nutrition plan, infection status and any agreed limitations of treatment
  IV. Physical and rehabilitation needs
  V. Psychological and emotional needs
  VI. Specific communication or language needs

The Maternity and General Critical Care Area Interface

Wherever a pregnant woman is receiving critical care in an HDU or ICU set-up, there must be a fundamental principle that her pregnancy care is continued and integrated into overall care plans and that this continues through to the postnatal period.

The multiple caregivers have to ensure that the needs of the critical care do not overshadow the needs of the woman and her family in regard to midwifery or obstetric care.

The pregnant woman being cared for in a general critical care area requires daily review by a multidisciplinary team including a named obstetric consultant and named senior midwife.

The individualised patient management plan should include care during the antepartum, intrapartum and postpartum periods with significant midwifery input for normal midwifery care.

The role of the maternity team includes discussing any specific obstetric conditions with the critical care team, for example pre-eclampsia, which may be obscured by the woman's current medical emergency. A neonatologist may also be required to advise on management of prematurity if a preterm delivery is a possibility.

As these women are critically ill, there should be regular communication between midwives, obstetricians and neonatologists as more complex aspects of obstetric care are considered.

Whilst the general critical care staff are experienced in communicating and updating family members, it has to be understood that there are different needs and information that the family requires from the midwife, e.g. emotional and social support, potential preparation for premature delivery, a baby in special care, etc.

Intensive care may be physically, emotionally, mentally and financially very taxing to the woman and her family. Awareness and monitoring of mental health is important as these women are more vulnerable due to the impact of increased risk for adverse outcome. The health care team should have good education to provide the needed support to the woman and her family due to the longer recovery period.

Conclusion

It is ethical for us to respect basic human rights, i.e. to preserve an individual's life and health and to provide the care with dignity, self-respect and confidentiality. It is also important that the patient and her relative get all the information and are involved in decision-making. The women should be provided the best of care available. These aspects of care based on basic ethical principles are tested to the extreme when critical care is

delivered in the intensive care setting. In many parts of the world, such care is available to those who could pay as such care is available only in paying centres and not in government hospitals. Pregnant mothers are young and are in their prime of life. Their health and life is compromised due to an obstetric or pre-existing medical complication. If the transient severe illness is overcome by providing critical care, then these mothers will continue to serve as the nucleus of the family and their society. Hence the health community should try and establish critical care for these young women with equal access despite their socio-economic standards and capabilities rather than stretching the health budget to less significant issues.

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Organisation and Role of Critical Care Units: Obstetric HDU/ICU

4

Alpesh Gandhi

Introduction

'*Women are not dying because of a disease we cannot treat. They are dying because societies have yet to make the decision that their lives are worth saving*'. This was declared by the then President, FIGO Dr. Muhammad Fathullah during the World Congress in 1997 at Copenhagen.

MMR in developed countries is <30. MMR in India is 178 (2010–2012, GOI), and in some of the states in India like Assam, it is even >300. MMR in India is even worse than its neighbouring countries like Sri Lanka and Bangladesh. Every year 500,000 pregnant women die in the world. If quality obstetric health care is provided in time, then we could save nearly 80 % of these women, i.e. 400,000 pregnant women in the world.

Maternal mortality is 'just the tip of the iceberg'. There is a vast base to this iceberg – which is maternal morbidity (near miss) – which remains largely undescribed.

There is no published survey or official data of MMR in institutional deliveries in India, but in unpublished data, the rate of maternal mortality at the hospital may be between 60 and 90 in different states which is nearly three times more than the MMR in developed world. There is a big gap in MMR between developing countries and western world in institutional deliveries.

An article was published in the May 2013 publication in the Lancet on implementation of essential obstetric health services and outcome. It showed that the immediate interventions with prophylactic uterotonics and treatment with oxytocics were given in low- and high-resourced countries to the same magnitude in teaching institutions. The same applied for the use of magnesium sulphate. However the maternal mortality was high in countries which have already high mortality, and it was low in countries which have already low mortality. Several explanations were given to this observation such as late interventions, but the most likely explanation is the lack of appropriate level of care, lack of teamwork and the absence of critical care after the life-threatening incident has happened.

Maternal near-miss case means a woman (in pregnancy/labour/puerperium) who almost died due to any life-threatening complications but survived. For every maternal death that occurs, between 11 and 223, women experience a 'near-miss' event in pregnancy. Recent WHO systematic review, global prevalence of SAMM (defined as severe life-threatening obstetric complication necessitating an urgent medical intervention in order to prevent likely death of mother), varies from 0.01 % to 8.23 %. The case fatality ratio is 0.02–37 %.

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The majority of women during their pregnancy, labour and postnatal period require care that can be met through routine obstetric care. A small but significant number, however, require critical care related to the pregnancy itself, aggravation of a pre-existing illness or complications of the delivery. Any pregnant woman can develop life-threatening complications with little or no advance warning which can lead to physical, social, economic and psychological consequences of complications. All such women need access to quality maternal health services that can detect and manage life-threatening complications.

In Europe and the USA, about 0.1–0.9 % of women during pregnancy or labour require intensive care. In India this data is not available. In India, the rate of high risk pregnancy varies from 8 % to 16 %.

Quality maternal health care means facility for invasive monitoring, skill-based services, skilled and experienced persons and 24 h monitoring. All these can be best accomplished in an obstetric ICU set-up where services from expert and trained medical, nursing and technical staff are available. They use sophisticated state-of-the-art equipment, technology for intensive monitoring and immediate life-saving interventions and organ support that may be necessary.

There is an agreement in the developed world on the need for intensive care facilities for the obstetric patient. This level of care may not be attainable for the pregnant in the developing world due to the lack of access to quality health facilities and its cost. It is not viable to have a separate ICU only for obstetrics purpose in smaller hospitals and smaller towns. This is the genesis for the concept of HIGH DEPENDENCY UNIT (HDU). It is interesting to know that in developed countries nearly 30 % of big institutes are having facility of obstetric HDUs, whereas in developing countries like us in India, it is still a dream.

What Is an ICU?

An *intensive care unit* (*ICU*) is a special department of a hospital or health-care facility that provides intensive care medicine. ICU is highly specified and sophisticated area of a hospital which is specifically designed, staffed, located, furnished and equipped, dedicated to the management of critically sick patient, injuries or complications. It is a department with dedicated medical, nursing and allied staffs (ICU Planning and Designing in India – Guidelines 2010, Guidelines Committee) (ISCCM).

What Is an Obstetric ICU?

It is an ICU which is dedicated to manage only obstetric patients having critical obstetrical or medical or surgical complications, managed by staff oriented for obstetric physiology and pathology.

What Is a High Dependency Care Unit (HDU)?

A high dependency unit is an area in a hospital, usually located closely to the intensive care unit, where patients can be cared for more extensively than on a normal ward, but not to the point of intensive care. So it is known as intermediate care units. Patients may be admitted to an HDU because they are at risk of requiring intensive care admission (step up), or at the same time, patients in the intensive care unit who have had an improvement in their condition require a stay in the high dependency unit (HDU) before admission to a general ward (step down). The HDU is similar to ICU except that patients admitted to the HDU are usually less ill or beginning to recover from their operation. It is called step-down, step-up, progressive and intermediate care units. HDU would not normally accept patients requiring mechanical ventilation, but could manage those receiving close monitoring. Patients with multi-organ failure cannot be kept in the HUD. HDU will suffice when organ support is not vital. HDUs are wards for people who need more intensive observation, treatment and nursing care than is possible in a routine ward but slightly less than that given in intensive care. The ratio of nurses to patients is 2:1 which is slightly lower than in intensive care but higher

than in general wards. It has some of its own limitations and advantages. It can be established in most obstetric unit in a room which is equipped for it. HDU is an option in terms of efficacy and fulfils the need of tertiary care centre. It reduces the need of the ICU with continuity of care. The risk of getting hospital-acquired infection is less in the HDU compared to the ICU. HDU care is cheaper as compared to ICU care. Psychologically patient will be more comfortable as her relatives can attend her, and in many cases her baby can be kept with her. It allows continuity of antenatal, intrapartum and postnatal care provided by the same team at the same place. A dedicated obstetric HDU with the knowledge, familiarity and expertise of an obstetrician and a specialist team would be the best place to monitor and treat the critically ill obstetric patients who do not require admission in the ICU.

Why Dedicated Obstetric ICU/HDU Is Required?

In developing countries like in India, the incidence of high-risk pregnancy is approximately 7–15 %.

Care of critically ill patients is a unique challenge in obstetrics. When things go wrong in obstetrics, they go wrong very fast, they fall off a cliff. Any pregnancy can develop life-threatening complications at any time with or without any warning.

Medical conditions might present risk to the pregnancy. Pregnancy may modify the disease state. Drug therapy may be affected by altered pharmacokinetics. Drug therapy may have impact on the foetus.

All such high-risk pregnancies can be saved by quality maternal health services and by means of 24 hours vigilant monitoring with immediate life-saving interventions and organ support. This can be possible with the facility of skill-based services in a dedicated critical care set-up with sophisticated state-of-the-art equipment and technology. Obstetric patients are generally young and healthy and they do recover rapidly. However, the potential for catastrophic complications is real.

Critically Ill Obstetric Patient Is Safer in the Obstetric HDU/ICU than MICU

Usually the obstetric HDU/ICU is a part of the obstetric department, near to the labour room and operation theatre, and so whenever is required, patient can be shifted easily and promptly to LR or operation theatre. This is not routinely possible with MICU. Shifting of patient is very difficult particularly when patient is obstetric. In MICU, ICU beds do not have the facility to be converted into labour table.

Usually NICU is also nearer to the obstetric department, and so neonates can be taken cared easily and promptly which may not be there with MICU. Obstetric HDU and obstetric ICU are nearer to each other in the obstetric department, and so step-up and step-down facilities can be used easily, which is not easily possible with MICU.

In the obstetric HDU/ICU, the in charge of the unit is a trained or experienced dedicated full-time obstetrician, whereas in the case of MICU, it may be a physician or an anaesthetist or a critical care specialist. In the MICU, 98 % patients are having medical or surgical problems, and so ICU staff is not acquainted with obstetrical complications/emergencies, whereas in obstetric HDU/ICU, staff is dedicated and trained in obstetric complications/emergencies.

In obstetric there are two patients, the mother and foetus. Foetal monitoring is only possible in the obstetric HDU/ICU, which is not possible in the MICU.

We have the MICU for medically ill patients, NICU for neonates and even at many places SICU for surgical patients. Then why not to have a dedicated obstetric HDU/ICU to save the high-risk or critically ill mothers?

Studies: A study was conducted on the role of high dependency unit (HDU) in critical care

obstetrics at Santokba Durlabhji Memorial Hospital, Jaipur. Women admitted in the HDU/ICU were studied over 3 years (2009–2012). HDU admissions included high-risk pregnancies (APH, PPH, PIH), sepsis, acute fatty liver of pregnancy, pulmonary thromboembolism and DVT, complications of pre-existing medical disorders, peripartum cardiomyopathy, post-caesarean complications, anaesthesia complications, uterine rupture and other genital tract injuries. Women requiring respiratory or inotropic support were shifted to the ICU. The outcomes of the study included the final outcome of women, burden status in the ICU, women shifted to the ICU from HDU, monetary expenditure to the hospital and cost of treatment to the patient.

Result: Out of 594 high-risk women, 427 (72 %) were managed in the HDU and 167 (28 %) required the ICU. Out of 167 women in the ICU, 109 (65 %) were shifted from the HDU and 58 (35 %) were admitted directly. Out of 427 women in the HDU, 400 (93.6 %) discharged in good condition, none expired and 27 (6 %) LAMA. Out of 167 in the ICU, 106 discharged in good condition, 35 (21 %) expired and 26 (15.6 %) LAMA. The burden in the ICU reduced to one-third in comparison to the last 5 years. Expenditure of the hospital in the maintenance of the ICU decreased by 41 %. Total treatment cost to the patient decreased by 38 %.

In another study, the objective was to establish the utilisation of high dependency care in a tertiary referral obstetric unit. Data of pregnant or recently pregnant women admitted to the obstetric high dependency unit from 1984 to 2007 were included to evaluate the admission rate. Four-year information of an ongoing prospective audit was collated to identify the indications for admission, maternal monitoring, transfers to intensive care unit and location of the baby.

The result was the overall HDU admission rate is 2.67 %, but increased to 5.01 % in the most recent 4 years. Massive obstetric haemorrhage is now the most common reason for admission. Invasive monitoring was undertaken in 30 % of women. Two-thirds of neonates (66.3 %) stayed with their critically ill mothers in the HDU. Transfer to the intensive care unit was needed in 1.4 per 1,000 deliveries conducted. The study concluded that obstetric high dependency care provides holistic care from midwives, obstetricians and anaesthetists while retaining the opportunity of early bonding with babies for critically ill mothers.

The study was also conducted at IPGME & R, Kolkata – one of the biggest tertiary care and referral institutes in Eastern India. Retrospective cohort study was conducted from May 2007 to May 2011. Relevant data regarding obstetric events, indications for HDU transfer/admission, interventions required, length of stay and eventual outcomes were collected, reviewed, tabulated and analysed.

Antenatal mothers were monitored clinically along with CTG, biophysical profile and Doppler study. All patients were individualised and managed with invasive or non-invasive measures as and when required. During the 4-year study period, 5,052 mothers delivered and 57 patients required HDU admission. Thus obstetric admission in the HDU was 11.2 per 1,000 deliveries. This data from Eastern India differed from the other parts. HDU utilisation by obstetric patients was 10.2 in 4 years in Dublin (Ireland) and 26.7 in 23 years in the UK. Unfortunately, data from other developing nations is lacking. Limited accessibility and higher mortality prior to receiving medical attention may help explain this low utilisation of HDU services in Eastern India. This incidence of HDU utilisation reveals just the tip of the iceberg. The indications leading to HDU admission were analysed among the patients. Sepsis accounted for the majority (35.08 %) of admissions, followed by PPH (29.82 %) and severe hypertensive disorders (BP 160/100 mm of Hg) of pregnancy (21.05 %).

Another study was conducted in the Rotunda Hospital, Dublin, with 121 beds. It incorporates a two-bedded HDU, established in June 1996. Patients were also subdivided into those transferred to the ICU of the period January 1994 to June 1996 (before on-site HDU facilities were

available) and those transferred to the ICU between June 1996 and June 1998 (after the HDU was established).

The total number of deliveries was 14,096 before the establishment of the HDU and 12,070 after the establishment of the HDU. One hundred twenty-three patients were admitted to the HDU during the study period June 1996 to June 1998, representing 1.02 % of all deliveries. This includes three patients who were admitted to the HDU from other obstetric centres for further management, but excludes five patients transferred to the ICU and then admitted back to the HDU for 'step-down' care. Eighteen patients were admitted before delivery: nine with antepartum haemorrhage, nine with pre-eclampsia, two with epilepsy, two with appendicitis, two with pulmonary embolism, one with ischaemic heart disease and one with renal impairment. Mean age was 30 years and gestation was 34.8 weeks. Median length of stay in the HDU was 3 days. Seventeen patients were transferred to a general ICU, 12 before the HDU was established (representing 0.08 % of all deliveries) and 5 after (0.04 %). Before the HDU was established, the length of stay in the ICU was 3 days and 2.0 days after the HDU. Prior to the development of onsite HDU facilities at the hospital, ICU utilisation rate was 0.08 % which decreased to 0.04 % following the establishment of this facility. Although not statistically significant, there is an apparent trend towards decreased ICU admission rates following the establishment of the HDU. Transfer to the ICU in the group before the HDU was necessitated predominantly by obstetric complications, with haemodynamic instability as a result of haemorrhage being the commonest ICU admission diagnosis. Following the advent of the HDU, the need for mechanical ventilation became the major indication for maternal ICU admission with an increasing number of patients with haemodynamic instability being managed within the HDU. The duration of ICU stay was short in both groups; interestingly, although not statistically significant, there is a trend towards reduced duration of ICU stay perhaps reflecting the availability of HDU care on discharge to the referring centre.

Wheatley et al. suggested that early intervention and treatment of the critically ill obstetric patient might prevent serious complications and avoid the need for the ICU. In their study, almost 60 % of the patients admitted to the ICU could have been managed appropriately within the HDU setting. The study concluded that a population of critically ill obstetric patients can be managed successfully in an obstetric HDU with the advantage of concurrent expert obstetric and critical care management.

A study was conducted at Liverpool Women's NHS Foundation Trust, Liverpool, UK. In England, the Critical Care Minimum Data Set (CCMDS) was used to assess critical care activity. This uses the number of organs supported to define level of care. One organ is level 2 (high dependency) and two or more level 3 (intensive care). Admissions over a 7-month period were studied to determine rates of admissions and level of care as defined by the CCMDS. Four thousand six hundred eight women were delivered and 239 (5.18 %) were admitted to the HDU. Average length of stay was 1.97 days. 137 (57 %) were CCMDS level 2 and 52 (22 %) level 3. An admission rate of 1–2 % quoted in the literature. This predates the publication of the MAGPIE study. Magnesium sulphate treatment constitutes a significant proportion of their HDU admissions, which may account for this increase.

Obstetric HDU: Obstetric ICU and Triaging Policy

Safe maternity is viewed as a basic human right worldwide. There is an agreement about the need for intensive care facilities for high-risk and critically ill obstetric patients. In the UK, nearly 30 % of hospitals are having HDU facility. All the referral hospitals should have obstetric HDU, and all the district referral hospitals and medical colleges should have both obstetric HDU and obstetric ICU, if prerequisite is fulfilled for its

establishment. It is recommended that all pregnancies with complications may be managed in the obstetric HDU/obstetric ICU after initial examination in the triage area. Patients may be transferred directly to the obstetric HDU/ICU from an emergency department if required, or from a ward if they rapidly deteriorate, or immediately after surgery if the surgery is very invasive and the patient is at high risk of complications.

Admission and Shift In and Shift out Criteria

Obstetric disorders constitute two-thirds of admissions and one-third is due to pregnancy with medical disorders.

Antenatal admissions are more common with medical complications and hypertensive disease of pregnancy, while postpartum patients are admitted more with haemodynamic instability mostly from obstetric haemorrhage, infection and postoperative complications.

Obstetrician will take a decision depending on the clinical condition and severity of illness, whom to admit in the obstetric HDU/ICU or who will require routine care/delivery. To optimally utilise the skills of manpower particularly specialists, normal deliveries or obstetric conditions can be sent to the labour room or ward where SBA-trained nurse/MO can take care and Obstetrician can be called in case of emergency. On the other hand, the high-risk and complicated cases should be admitted to the obstetric HDU/ICU for constant care under overall supervision of an experienced obstetrician. However for round-the-clock monitoring, resident doctors/EmOC-trained doctors can be posted. During examination, a quick initial assessment is required to decide if she is an emergency or complicated case.

Obstetric patient with following conditions/diagnosis may require admission in the obstetric HDU:

(a) Haemodynamic instability
(b) Respiratory dysfunction
(c) Neurologic complications
(d) Acute kidney injury
(e) Haematological complications

Patient with following parameters requires admission in the obstetric HDU/ICU

Obstetric HDU	Obstetric ICU
Systolic blood pressure <90 or >160 mmHg	Systolic BP <80 mmHg or 30 mmHg below patient's usual BP
Diastolic blood pressure <50 or >110 mmHg	
Mean arterial blood pressure <60 mmHg	Heart rate <50 or >140 beats/min
Heart rate <60 or >110 per min	RR <8 or >35 per min
Respiratory rate: >25 per min	U/O <400 ml in 24 h or <160 ml in 8 h and unresponsive to simple routine measures
Urine <0.5 ml/Kg/h (<30 ml per h)	GCS <8 in the context of non-traumatic coma
Any organ dysfunction	Any unarousable patient
	Serum sodium <110 or >160 mmol L
	Serum potassium <2.0 or > 7.0 mmol L
	pH <7.1 or >7.7
	PaO2 <6.6 kPa and/or PaCO2 >8.0 kPa
	SaO2 <90 % on supplemental oxygen
	Need for advanced respiratory support
	Inotropic support
	DIC
	Multi-organ failure
	ARDS

Scope of the HDU: The following conditions may require admission in the obstetric HDU/ICU

Obstetric complications	Pregnancy with medical complications
Pregnancy/labour pain with severe anaemia (<7 g %) and its complications	Pregnancy with gestational diabetes
Accidental haemorrhage – placental abruption, couvelaire uterus	Pregnancy with diabetic ketoacidosis
Postpartum haemorrhage	Pregnancy with cardiac diseases
Placenta previa	Pregnancy with jaundice
Adherent placenta and other placental abnormalities	Pregnancy with thyrotoxicosis
Obstetric hysterectomy	Pregnancy with thyroid storm
Severe pre-eclampsia/hypertensive crisis	Pregnancy with phaeochromocytoma
Eclampsia	Pregnancy with other endocrinal crisis like Addison's disease etc.
Broad ligament haematoma	Postoperative ARF and other renal problems
HELLP syndrome	Leukaemia and other haemolytic disorders
Pregnancy with DIC	Pregnancy with dengue
Sepsis and systemic inflammatory response syndrome (SIRS)	Pregnancy with complications of malaria
Pregnancy with thrombophilias	Pregnancy with asthma and other respiratory problems
Multiple gestation with complications	PPCM – peripartum cardiomyopathy
Pregnancy with complications due to uterine anomaly and pathologies	Pregnancy with appendicectomy or any other surgical emergency
Hydatidiform mole	Pregnancy with OHSS (ovarian hyperstimulation syndrome)
Ruptured ectopic	Pregnancy with acute pancreatitis
Burns during pregnancy	Pregnancy with trauma
Perforation during abortion	Pregnancy with poisoning
Postoperative patients requiring haemodynamic monitoring or intensive nursing care	Pregnancy with cancer
Pulmonary oedema due to perioperative fluid overload, CCF, complication of severe pre-eclampsia or tocolytic therapy with β-agonists, etc.	

There are many other such conditions when obstetrician decides that it is appropriate to admit the patient in the obstetric HDU/ICU.

Isolation: Pregnancy with H1N1, pyometra, HIV and infectious diseases should be admitted in the isolation room of the obstetric HDU/ICU.

Shifting of a Patient from the Ward to the Obstetric HDU/ICU

A. Key steps to be taken are

1. Measure and mention clinical criteria as explained above.
2. Inform the decision to and take consent from the patient and her relatives.
3. Prepare and send case sheets containing H/O, examination, investigations and management.
4. Obstetric HDU/ICU staff should be informed.
5. Patient should be escorted by the doctor/staff with all existing treatment including patent IV line continued while shifting to the obstetric HDU/ICU.
6. Keep monitoring the vitals of the patient.
7. Prevent supine hypotension (give left lateral tilt 15–20°) if required.
8. Ensure patent airway and give oxygen, if required.
9. The baby should be shifted with the mother if she has delivered.
10. Adequately follow up the doctor under which initial treatment was given.

B. Referrals from the outside to the HDU/ICU
Key steps to be taken are

1. The need for referral should be explained to the patient and relatives by the referring facility.
2. All the protocols explained at 'A' above need to be ensured.
3. Proper referral slip should be sent.
4. Emergency medications should be available while transferring.

Planning an obstetric HDU: It includes cost of initial capital expenditure, purchase of new technology, recruitment of staff and rolling annual cost and other indirect costs like training, consumables, IT facility, etc. Early proper referrals can make it a viable unit.

Setting up of the HDU: It should be a special part of the obstetric unit. Location, space, equipments, personnel, protocols, audit, education and training are important issues to be taken cared of. Its set-up requires following specific areas, facilities and protocols.

Location: It should be near the ICU or OT or both. There should be at least one fully equipped obstetric theatre within the delivery suite. Where this is not possible, a lift, for the rapid transfer of women to the theatre, must be available. It should have nearby facilities like the blood bank, fully equipped laboratory, radiology department and NICU care. There should be single entry/exit point to the obstetric HDU/ICU. There should be provision for emergency exit point in case of disasters.

A. *Space*:
- Space should be 120–130 sq ft per bed in the obstetric HDU.
- Space should be 130–140 sq ft per bed in the obstetric ICU.
- Bed space area should have 100–125 % extra space to accommodate nursing station/storage/patient movement area/equipment area and patient toilets and to manoeuvre equipments, beds and trolleys.
- Beds should be 2 ft from the back wall to give caregivers an easy access to the head in case of an emergency.
- There should have bedside movable lockers with facility of trolleys/drawers for keeping medicines, consumables and personal belongings of the patient.

B. *Privacy*:
- There should be a partition between rooms for privacy of a patient.
- Each bed can be separated by fixed partitions or else can be separated by curtains.
- Curtain fabric should be fireproof, waterproof but washable, clean, white or off-white in colour, inherent stain resistance and non-allergic.
- The curtain height is determined by the floor-to-ceiling height and curtains usually finish approximately 8–10″ above finished floor.
- Curtains should have mesh at the top which will allow both light and ventilation throughout the patient room.
- Curtains should be hanging from overhanging rails.

C. *Flooring*:
- Large vitrified non-slippery stainproof tiles.
- The best option is with seamless joints which are easy to clean and move on.
- Colour should be either white or off-white.

D. *Walls*:
- Durable and glassed tiles, ability to clean and visual appeal.
- Stain resistance.
- Flame retardant.
- It has been very useful to have a height up to 6–7 ft finished with similar tiles as of the floor.
- Colours should be chosen carefully to avoid an adverse impact on the skin colours of patients and neonates preferably white or off-white.

E. *Ceiling*:
- Ceiling should be soiling and break-proof due to leaks and condensation.
- It is recommended that no lines or wires be kept or run over the ceiling or underground, it should be easily exportable because damages do occur at any time and, therefore, it should be easy to do repairs.

F. *Nursing station*:
- It should have sitting capacity for dedicated nurse, dedicated obstetrician and two EmOC/MOs and for visiting doctors.

- Adequate space for central monitoring and computers.
- Scrub area and wall clock behind the nursing station.
- Facility for record-keepings and emergency medicines.

G. *HMIS*:
- The facility of HMIS should be provided for data collection, data transmission, data storage, data processing, data analysis and data presentation for decision taking in the hospital management and for the better health-care services.

H. *Storeroom – equipment*:
- A separate room should be made to keep sonography machine, portable X-ray machine, transport ventilator, nebulizer, radiant warmer, blood warmer, crash cart, BIPAP/CPAP, etc.

I. *Storeroom – general*:
- A general store room should be made to keep linens, disposables, consumables, cap and mask, sleepers, etc.

J. *Toilets*:
- Two toilets are required for eight-bedded set-up.

K. *Buffer zone*:
- A protective clean zone to be made available before the entry in the obstetric HDU/ICU which includes area for shoe change, patient's changing room with lockers, counselling room, janitor's closet, two changing/overnight stay rooms for doctors (one each for male and female with lockers, beds, book shelf), dining area, space to keep wheel chair and trolley, changing room for staff and toilets – one each for male and female with urinal in male toilet with flush facility.

L. *Autoclaving and sterilised supply* from the Central Sterilised Supply Department (CSSD)

M. *Waiting area*:
- Waiting area for patient attendants with facility of sitting capacity of three relatives per patient.
- Facilities of drinking water, a large TV with LCD, toilets, newspapers, educational materials, etc.
- Waiting area can be shared with the waiting area for LR and OT.

N. *Other facilities*:

(a) All the cots in the obstetric HDU/ICU must be with birthing bed that is of sufficient size and electrically/manually operated. It should have easy-to-operate different positions like Trendelenburg, reverse Trendelenburg and both side tilt. It is ideal to have good-quality castors with brake system for optimum mobility/stability.

(b) Each bed will require at least two oxygen, one air and two suction outlets and at least ten central voltage stabilised power points (six power points of 5 amp and four of 15 amp) preferably five on each side of the bed. Adapters should be discouraged as they tend to become loose.

(c) Heating, ventilation and air-conditioning (HVAC) system along with ceiling fans and power backup exclusive for the HDU/ICU for uninterrupted power supply. Voltage stabilisation is mandatory. Suitable and safe air quality must be maintained at all times. Temperature should be adjustable within each cubicle/room as per patient comfort and choice.

(d) *General lighting*: Access to outside natural light is recommended by regulatory authority in the USA, which may improve the morale of staff and patients. It can also decrease power consumption. The colourless concealed LED lights with sufficient high illumination can be provided. It should be bright enough to ensure adequate vision without eye strain.

(e) *Hand hygiene and infection prevention control policy*:
- Every bed should have alcohol-based antimicrobial instant hand-wash solution source.
- Sink should be of operation room style with elbow-operated water supply.
- All entrants should don mask and cap and ideally an apron which should be replaced daily.

(f) *Waste disposal and pollution control*: This is mandatory and a huge safety issue both for the patient and staff and doctors of the hospital and society at large. It is important that all government regulations (State Pollution Control Board) should strictly be complied with.

(g) Laboratory backup facility for 24 hours.
(h) In-house or nearby blood bank.
(i) In-house or nearby NICU.
(j) *Lactation support*:
- Facilities for breast-feeding (or use of breast pump) should be available to all postnatal mothers in the obstetric HDU.
- Babies are usually not allowed in the obstetric ICU, but breast pump facility should be made available.

(k) *Fire safety*:
Provision for fire safety preferably automatic water sprinkling system or normal fire extinguishers
(l) For uninterrupted power supply, noiseless generator or inverter with instant switchover, search protector and voltage stabiliser. Earthing pit for proper earthing to be ensured with monitoring once in 2 years.
(m) Nurse calling system with central display with an audiovisual alarm.
(n) Facility for keeping records and registers with nursing station.
(o) Provision for birth companion and visitors. It is very important to value family members and take care of their needs.
(p) *Ambulance:* One hi-tech ambulance with all advanced life support measures for critically ill mother and with transport incubator with ventilator support for critical newborn.

Furniture and equipments: Along with all routine equipments required in routine ward, the following furniture and equipments are necessary for the setting up of an HDU: maternity cot electronically manoeuvred in all positions; glucometer; infusion pump; syringe pump; ultrasound machine with colour Doppler and echo facility; CTG machine; cardiac monitor with CVP monitor; intubation kit; baby resuscitation kit/cart; crash cart fully loaded with BCLS medications; stock of all emergency drugs; if possible O-ve blood, CNS tray with torch, hammer, etc.; central oxygen supply; wall mount suction; pulse oximeter; anaesthesia apparatus; cautery machine; defibrillator; ventilator (it is ideal for the obstetric ICU); BIPAP; refrigerator with deep-freeze facility; X-ray view box; separate eclampsia box; partogram; input/output chart; generator or inverter; intercom and emergency bell facility with a phone list of help lines and all other required facilities; trays for procedures for putting central lines; ICD; catheters; etc. are required for the setting up of an HDU.

Personnel Staffing

Obstetrician is the head of the obstetric HDU/ICU. He/she will decide when to call and whom to call from the list of multidisciplinary team whenever is required (wherever available), for the management of the obstetric patient.

Obstetric HDU working team consists of incharge experienced/trained full-time obstetrician, EmoC/medical officers round the clock and obstetric nursing staff (24×7); nursing staff to patient ratio should be 1:2 (one extra for lay-off or covering leave/day-off).

Obstetric anaesthetists, physician, neonatologists, surgeon and radiologist's services should be made as an assured services. It is ideal to have backup support of these specialists and super specialists on call, if and when required (wherever possible). If the particular specialist is not available, he/she can be called from the outside.

Support staff like *the* pharmacist, dietician, counsellor, housekeeping and cleaning, security, data entry operator, electrical technician, biomedical engineer, etc. should be available.

Obstetric ICU working team consists of incharge experienced/trained full-time obstetricians, obstetric anaesthetist, EmoC/medical officers round the clock and obstetric support staff (24×7). Nursing staff to patient ratio should be 1:1 (there should be one extra for lay-off or covering leave/day-off). Support staff experienced with HDU/ICU nursing care provides continuous observation – including accurate recording of fluid intake, urine output, blood pressure (via arterial line in some cases) and central venous pressure monitoring, pulse oximetry etc. – whenever required.

Services of critical care specialist or physician, neonatologist, surgeon and radiologist should be assured on call. There should be backup support of other specialists and super specialists on call, if

and when required like the haematologist, cardiologist, nephrologists, neurologist, endocrinologist, pulmonologist, vascular surgeon, etc. If the particular specialist is not available in the hospital, he/she can be called from the outside.

Support staff like *the* pharmacist, dietician, counsellor, housekeeping and cleaning, security, data entry operator, electrical technician and biomedical engineer should be available on hand. All staff should be adequately trained in recovery care and cardiopulmonary resuscitation.

Monitoring and Management at the HDU

History – record the date, time and reason for requesting this level of care, name of clinician contacted, a summary of the current problems, review of the patient's observations and finding on clinical examination and a plan for ongoing care. Future review should be completely documented.

Immediate initial assessment and resuscitation should be done.

Maternal observation: The following parameters should be recorded at least hourly in the acute phase of the illness.

(a) Temperature
(b) Blood pressure
(c) Heart rate
(d) Respiratory rate
(e) Transcutaneous oxygen saturation
(f) Hourly urine output

The following Management Is Done as Per Case Requirement

- *Management*: It includes initial assessment of the condition and resuscitation of the patient whenever required. Maternal organ function monitoring of cardiovascular, renal, pulmonary, hepatic and cerebral is done. Baseline and specific investigations as indicated are advised. Primary conditions like severe pre-eclampsia, haemorrhage, sepsis, etc. are treated. Anticonvulsant therapy is given, whenever is required. Injectable magnesium sulphate (MgSO4) is given as per protocol for eclampsia patient. Fluid balance and electrolyte correction is taken cared. Foetal condition is checked by CTG. Fluid therapy in the form of crystalloid/colloid/blood is given. Uteroplacental oxygen delivery is maintained. Left lateral position and oxygen via face mask is given, if required, and non-invasive and invasive monitoring like BP, RR, HR, pulse, SPO2, ABP, CVP, ABGs, hourly UOP, lung functions and others are done. Broad-spectrum antibiotics are given for sepsis if required to cover Gram-negatives and anaerobes after discussion with the microbiologist. Proper care for nutrition is taken. Enteral and parenteral nutrition is given. If required, inotropes are given. Pain management is done. Appropriate clinicians from relevant specialties are involved. Final management is individualised and depends on the underlying clinical condition.
- Watch for:
  - Pulmonary oedema/ARDS/SIRS
  - Multi-organ failure
  - Poor cardiac output despite fluid resuscitation
  - Septic shock
  - DIC
  - Other parameters mentioned earlier for admission in the ICU
- Approximately 2–3 % of patients admitted to the obstetric HDU will require transfer to the ICU.
- Portable monitoring with facility for invasive monitoring must be available to facilitate transfer of obstetric patients to the ICU.
- ↓
  - Transfer to the obstetric ICU.

Care for Foetus in the Obstetric HDU/ ICU

- Generally, foetal morbidity and mortality reflect maternal condition closely. Simple measures such as avoidance of supine hypotension by a 15° left lateral tilt and oxygen via face mask can improve uteroplacental oxygen delivery. Foetal condition should be observed

by continuous electronic foetal monitoring. Corticosteroids should be given if <34 weeks. Labour and delivery is planned as per maternal indication or foetal indication. When required, consider delivery. Route of delivery is decided depending on obstetric indication.

Patient's case record must contain the following:

- Proper history – It should be clear, legible and accurate.
- Probable or confirmed diagnosis
- Laboratory and imaging findings
- Operative note
- Operative findings
- Management summary
- Must include: date, time and signature
- Explaining the conditions of the patients to the birth attendants/relative and getting it documented into the case sheets
- Getting the consent of the patient for any procedure and for blood transfusions and getting it documented into the case sheets

Guidelines and Protocols to Be Followed

As HDU care involves management of critically ill obstetric patients, guidelines and protocols should be in place to encourage appropriate responses to these critical situations and justify actions that are sufficient and efficient, neither excessive nor deficient.

SOPs for all these protocols should be clearly laid down in consultation with experts:

- Admission and discharge criteria to/from the HDU
- Resuscitation of the pregnant patient
- Management of major haemorrhage
- Management of pre-eclampsia and eclampsia
- Severe hypotension/hypertension/DM/DKA/sepsis
- Management of failed/difficult intubation
- Protocol for regional/general anaesthesia and regional block for analgesia
- Protocol for the management of postdural puncture headache
- Guideline for the use of invasive monitoring
- Protocol for the management of postoperative pain
- Protocol for thrombo-prophylaxis
- Management of patients on thrombo-prophylaxis

Note: *In addition*, *protocols for specific obstetric conditions not enumerated above need to be followed as per its standard treatment protocol*.

Discharge from the Obstetric HDU to Ward

The decision of discharging a patient from the obstetric HDU is taken when a patient's physiologic status has been stabilised, patient is haemodynamically stable and the need for intensive monitoring is no longer necessary. When there is no active bleeding, no further continuous intravenous medication or frequent blood tests required, no invasive monitoring is required, no supplementary oxygen is required and patient is ambulatory, then the patient is discharged from the obstetric HDU/ICU and transferred to a ward. When transferring a woman from the HDU to the ward, a personal and detailed handover of care should be given. Average time in the HDU is usually 24–72 hours. Patient should be discharged with full written document.

Indications of transfer from the HDU to the ICU: When patient needs advanced respiratory support, further inotropic support or when patient develops DIC, multi-organ failure or adult respiratory distress syndrome, she is transferred to the ICU for further care and support.

Transfer to the ICU: When RR is outside the range 7–35 breaths/min, pulse is outside the range 40–140 beats/min, BP <80 mmHg or 30 mmHg below patient's usual BP, U/O <400 ml in 24 hours or <160 ml in 8 hours and unresponsive to simple measures, GCS <8 in the context of non-traumatic coma, patient is unarousable, serum sodium outside the range 110–160 mmol/L, serum potassium outside the range 2.0–7.0 mmol/L, pH outside the range 7.1–7.7, PaO2 <6.6 kPa and/or PaCO2 more than 8.0 kPa and SaO2 <90 % on supplemental oxygen

Comparison between obstetric HDU, obstetric ICU and MICU.

Criteria	Obstetric HDU	Obstetric ICU	Medical ICU
No. of units required	Many	Less	Many
Patient	Pregnant women (ante-, intra- or postnatal)	Pregnant women (ante-, intra- or postnatal)	Any patient (male or female)
Admission criteria	Obstetrical or medical complication in a pregnant woman requiring close and continuous monitoring	Obstetrical or medical complication in a pregnant woman requiring ventilatory support and dialysis, multi-organ failure, DIC, etc.	Medical conditions, surgical conditions with life-threatening requiring ventilatory support, complications, multi-organ failure, etc.
Bed requirement	7–10 % of total obstetric patients	0.5–0.9 % of total obstetric patients	Requirements differs from place to place
Space per bed	120–130 sq ft per bed	130–150 sq ft per bed	120–140 sq ft per bed
Facilities required	Basic cardiac and respiratory and metabolic monitors, blood component therapy, foetal monitoring, sonography with colour Doppler/echo, transport ventilator, etc.	HDU + invasive monitoring, ventilators, bedside dialysis, plasmapheresis, pacemakers, bronchoscopy, endoscopy, tracheostomy, own or outsourced CT scan and EMRI	HDU+ invasive monitoring ventilator, bedside dialysis, plasmapheresis, pacemakers, bronchoscopy, endoscopy, tracheostomy, own or outsourced CT scan and EMRI
In charge	Obstetrician	Obstetrician. Critical care specialist or anaesthetist who will help him/her	Critical care specialist/anaesthetists/physician
Baby accompanied	Yes	No	No
Human resources	Nurse/patient – 1:2	Nurse/patient – 1:1	Nurse/patient – 1:1
Team composition	Obstetrician, anaesthetists, neonatologists,	Obstetrician, anaesthetists, neonatologists, intensivists/physician	Anaesthetists, intensivists/physician
	Medical officer	Medical officer	On call – specialists, super specialists for 24 h
	On call – intensivists/physician and multispecialists (wherever possible) for 24 h	On call – specialists, super specialists for 24 h	Trained dedicated nurse and biomedical engineer, support staff
	Trained dedicated obstetric nurse and support staff	Trained dedicated obstetric nurse, biomedical engineer and support staff	
Continuity of care by the same team	Allows continuity of antenatal, intrapartum and postnatal care by the same team	Allows continuity of antenatal, intrapartum and postnatal care by the same team	Does not allow continuity of antenatal, intrapartum and postnatal care by the same team
Hospital-acquired infections	Possibility is less	Possibility is more	Possibility is more
Capacity building	Specially trained in obstetric conditions, its complications and in critical care	Specially trained in obstetric conditions, its complications and in critical care	Specially trained in medical and surgical complications and also in critical care
Financial burden	Treatment is cheaper	Treatment is costly	Treatment is costly
Environment	Patient friendly. Psychologically patient will be more comfortable as relatives can attend her and baby is with mother	Not so patient friendly in comparison to obstetric HDU	Not so patient friendly in comparison to obstetric HDU/ICU

Conclusion

The majority of women during their pregnancy, labour and postnatal period require care that can be met through routine obstetric care. A small but significant number, however, require critical care related to the pregnancy itself, aggravation of a pre-existing illness or complications of the delivery. Any pregnant woman can develop life-threatening complications with little or no advance warning which can lead to physical, social, economic and psychological consequences of complications. All such women need access to quality maternal health services that can detect and manage life-threatening complications. Every year 500,000 pregnant women die in the world. If quality obstetric health care is provided in time, then we could save nearly 80 % of these women, i.e. 400,000 pregnant women in the world.

HDU provides a level of care in between general ward and ICU set-up. Women not requiring ventilator support can be managed in the HDU, reducing the burden of ICUs. Treatment cost reduces, and above all it requires less expenditure to establish and manage the HDU. A dedicated obstetric HDU with the knowledge, familiarity, experience and expertise of an obstetrician and a specialist team would be the best place to monitor and treat the critically ill obstetric patients. It allows continuity of antenatal, intrapartum and postnatal care be provided by the same team. Delivery of the baby takes place in a more familiar and better-equipped environment with minimal disruption of mother-to-baby bonding. Care in an obstetric HDU may avoid exposure of the critically ill pregnant mother to a potentially hazardous ICU environment with the risk of hospital-acquired infection. Patient satisfaction may be increased since it has more liberal family visitation policies. Care in an ICU sometimes becomes focused on the machines, rather than on the patient, whereas in the HDU, humanising aspects of critical care can be addressed in caring for a patient and her family.

When patient needs advanced respiratory support, further inotropic support or when patient develops DIC, multi-organ failure or adult respiratory distress syndrome, she is transferred to the obstetric ICU for further care and support. Patients with high-risk pregnancy and critically ill patients can be managed better in the obstetric HDU/ICU which is not possible in the routine ward which ultimately reduces MMR and morbidity.

Further Reading

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Cardiopulmonary Resuscitation in the Pregnant Woman

5

Amita Gandhi and Alpesh Gandhi

Introduction

Maternal collapse is a rare but life-threatening event with a wide range of aetiology. The outcome for the mother and also for the foetus depends on prompt and effective resuscitation. Maternal collapse is defined as an acute event involving the cardiorespiratory system and/or brain resulting in a reduced or absent conscious level (and potentially death) at any stage in pregnancy and up to 6 weeks after delivery. The incidence of maternal collapse or severe maternal morbidity is unknown as morbidity data are not routinely collected. The data showed a severe maternal morbidity rate of 6/1000 (600/100,000) maternities in Scotland, but not all cases of severe maternal morbidity involved maternal collapse (although all cases of collapse were included in the figures). A recent publication from Dublin showed a severe maternal morbidity rate of 3.2/1000 (320/100,000) births. In the last triennium in the UK, the maternal mortality rate was 14/100,000 births, but again not all maternal deaths are preceded by maternal collapse. Thus, the true rate of maternal collapse lies somewhere between 0.14 and 6/1000 (14 and 600/100,000) births [1]. Cardiac arrest in pregnancy is rare encounter, considered to occur in 1:30,000 births [2].

Cardiopulmonary arrest in the pregnant woman triggers highly emotional reactions and can put the parturient at risk. This chapter reviews the physiological changes secondary to pregnancy in brief and the current knowledge and recommendations regarding the management of a pregnant woman in cardiac arrest.

Following their analysis, of questionnaire survey among obstetricians, anaesthetists and midwives, Einav et al. [3] concluded that clinicians who treat pregnant women in the hospital on a daily basis possess a limited knowledge of the recommendations for treating maternal cardiac arrest.

Clinical Issues

There are many causes of sudden obstetric collapse, which may be pregnancy related or result from conditions not related to pregnancy and possibly existing before pregnancy. Inclusion of all causes of the obstetric collapse is beyond the scope of the chapter, but the common causes of maternal collapse are discussed below.

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Haemorrhage

This is the most common cause of maternal collapse and was responsible for maximum number of maternal deaths. Causes of major obstetrics haemorrhage are postpartum haemorrhage, ante partum haemorrhage from placenta praevia/accrete, placental abruption, uterine rupture and ectopic pregnancy. In most cases of massive haemorrhage leading to collapse, the cause is obvious, but the concealed haemorrhage should not be forgotten, including following caesarean section and ruptured ectopic pregnancy; other rare causes of concealed haemorrhage include splenic rupture and hepatic rupture. Acute inversion of uterus may be a cause due to improper conduct of third stage. Resuscitation includes establishment of respiration and restoration of deficit blood volume and arrest of source of haemorrhage.

Eclampsia

As the cause of maternal collapse is usually obvious in the patient settings, often the diagnosis of pre-eclampsia had already been made and the seizure witnessed. Epilepsy should always be considered in cases of maternal collapse associated with seizure activity. Resuscitation includes correction of airway, control of seizures, blood pressure and delivery.

Amniotic Fluid Embolism (AFE)

It presents as collapse during labour or delivery or within 30 min of delivery in the form of acute hypotension, respiratory distress and acute hypoxia. Seizures and cardiac arrest may occur. Pulmonary hypertension may develop secondary to vascular occlusion either by debris or by vasoconstriction followed by left ventricular failure and if the mother survives, often giving rise to massive PPH. If AFE occurs prior to delivery, profound foetal distress develops acutely. Speedy resuscitation with oxygen administration, positive pressure ventilation, intravenous fluids, vasopressors and, if the foetus is undelivered, immediate delivery should be undertaken. In case of haemorrhage, replacement with red cells, platelets, FFP, cryoprecipitate or fibrinogen is done according to the need.

Pulmonary Thromboembolism

Woman with clinical suspicion of DVT may lead to release of thrombus. PE occurs due to a thrombus blocking a pulmonary artery, which causes sudden onset of dyspnoea, chest pain and features of collapse. Prompt resuscitation and antithrombotic therapy must be started immediately.

Sepsis

Sepsis has been recognised for centuries as a significant cause of maternal morbidity and mortality. Bacteraemia, which may be present in the absence of pyrexia or a raised white cell count, can progress rapidly to severe sepsis shock, leading to collapse. Management includes resuscitation, broad-spectrum antibiotic coverage and removable of septic foci.

Cardiac Disease

The majority of deaths secondary to cardiac causes occur in woman with no previous history. The main causes of death are myocardial infarction, aortic dissection and cardiomyopathy.

Intracranial Haemorrhage

ICH is a significant complication of uncontrolled systolic hypertension with severe headache followed by maternal collapse.

Anaphylaxis

Anaphylaxis is a severely, life-threatening, generalised or systemic reaction resulting in respiratory, cutaneous and circulatory changes and possibly gastrointestinal disturbances and

collapse. Prompt resuscitation with arrest of anaphylaxis is a key of management.

Drug Toxicity and Overdoses

Drug toxicity and overdoses should be considered in all cases of obstetric collapse, and illicit drug overdose should be remembered potential cause of collapse in the hospital like magnesium sulphate in the presence of renal impairment or local anaesthetic agents injected intravenously by accident.

Sudden Obstetric Collapse: D/D

It is imperative to identify reversible causes of cardiac arrest. The age of pregnancy should be quickly established in order to decide about foetal viability. Abdominal ultrasound examination can be performed for this purpose, but it should not delay the resuscitation procedures. The aetiology can also be classified into anaesthesia-related causes and/or non-anaesthesia-related causes [4].

Do spot probable diagnosis: Is it APH or PPH or inversion of uterus?

If not, then think for non-haemorrhagic causes.

H/O and probable diagnosis

H/O severe HT and convulsions – *eclampsia*

H/O grand multipara or previous uterine scar or instrumental delivery – *rupture of the uterus*

H/O mismanaged third stage of labour, short cord or MRP – *inversion of uterus*

H/O SA in higher position, difficult SA during surgery, C/O heaviness in the chest, gabhraman, breathlessness within few min of SA – *high spinal anaesthesia*

H/O vomiting under anaesthesia and problem starts within few hours – *Mendelson's syndrome*

H/O fall in the B.P. within few minutes after SA – *supine spinal shock*

H/O previous cardiac problems, c/o acute left-sided chest pain, gabhraman, hypotension – *maternal cardiac problems*, mainly *myocardial infarction*

H/O vehicular accidents or domiciliary violence – *trauma*

H/O collapse after administration of drugs, S/S allergic reactions – *drug reaction or overdose*

H/O painful stimuli, injections, etc. – *anaphylactic reaction*

H/O collapse immediately after delivery, mainly in multipara or in precipitate labour and no obvious cause or in any case always think for possibility of amniotic fluid embolism – *AFE*

H/O sudden onset of unexplained dyspnoea and tachypnoea, especially in western countries because of venous stasis and hypercoagulability of blood – *pulmonary thromboembolism*

Mortality related to airway problems during extubation of the trachea has increased as spinal anaesthesia-related mortality [5, 6]. Twenty-seven percent of all maternal deaths due to anaesthesia occurred among obese women, whereas 24 % occurred among overweight women.

Physiological Changes in Pregnancy Affect Resuscitation

There are a number of reasons why the processes of cardiopulmonary resuscitation are more difficult to perform and may be less effective in the pregnant than in the non-pregnant population. When these changes occur is not precise, but gradually the presence of increasing mass in the abdomen compromises resuscitative efforts. This may be the case from 20 weeks onwards but will be more marked as the mother approaches term.

Cardiac Output

Cardiac output increases by as much as 50 % by 32 weeks' gestation. At 20 weeks, significant aortocaval compression compromises venous return, and at 30 weeks of gestation, the woman has a significant drop in blood pressure when lying supine [1].

Vena Caval Occlusion

After 20 weeks' gestation, the pregnant woman's uterus can press down against the inferior vena cava

and the aorta, impeding venous return, cardiac output and uterine perfusion. Caval compression limits the effectiveness of chest compressions. The vena cava is completely occluded in 90 % of term pregnant woman lying supine and the stroke volume may be only 30 % of that of a non-pregnant woman. In late pregnancy, hence cardiac output can be increased by as much as 25–30 % simply by moving the patient in a left lateral decubitus position [7]. During cardiac arrest, in order to minimise the effects of the gravid uterus on venous return and cardiac output, a maternal pelvic tilt to the left of greater than 15° is recommended. The tilt needs to be less than 30° for effective closed chest compression to take place. Delivery of the foetus during cardiac arrest will reduce the oxygen demands on the mother and also increase the venous return to the heart making it more possible that resuscitation will be successful.

Changes in Lung Function

The pregnant uterus and increased breast size lead to a 20 % decrease in functional residual capacity and 45 % decrease in chest compliance. With such limited reserve and 20 % increased oxygen consumption, there can be a rapid decline in oxygen saturation following hypoventilation.

Effectiveness of Ventilation

The presence of mucosal oedema and friability, increased secretions and weight gain all contribute to a more difficult airway intubation. There is also a greater risk of aspiration due to the relaxation of the oesophageal sphincter. Passive regurgitation of stomach contents is a very real concern as it is greater in volume and more acidic during pregnancy and so more likely leads to damaging acid aspiration into the lungs. It is imperative that experienced staff provide a protected airway and adequate ventilation via an ET tube as quickly as possible following cardiac arrest.

The following modifications are necessary for managing cardiac arrest in pregnancy:

Management In the UK, resuscitation is conducted according to the UK Resuscitation Council Guidelines: basic life support (BLS), adult advanced life support (ALS) and automated external defibrillation (AED) algorithms and recommendations. These guidelines were updated in 2010 by the International Liaison Committee on Resuscitation and are used in the resuscitation of the pregnant woman worldwide. It is recognised that the divisions into basic and advanced life support are somewhat arbitrary in the hospital setting. In the community setting, basic life support should be administered and rapid transfer arranged, unless appropriate personnel and equipment are available.

Immediately CPR should be started. CPR done is the same whether she is pregnant or not, though with few modifications, if she is pregnant.

The following modifications are necessary for managing cardiac arrest in pregnancy:

1. Earlier definitive airway control.
2. Left lateral displacement of the uterus if GA is more than 20 weeks.
3. Chest compression is done slightly above than in a case of non-pregnant patient.
4. Consideration of an early caesarean delivery.
5. The use of adhesive pads for defibrillation and the removal of the foetal monitor to avoid electrical arcing.

The following instructions may be carried out almost simultaneously by multiple helpers as described in an appropriate order for one person.

Basic Life Support and Adult Advanced Life Support for Obstetric Patient

As per latest recommendations, emergency management is CBA (not ABC).

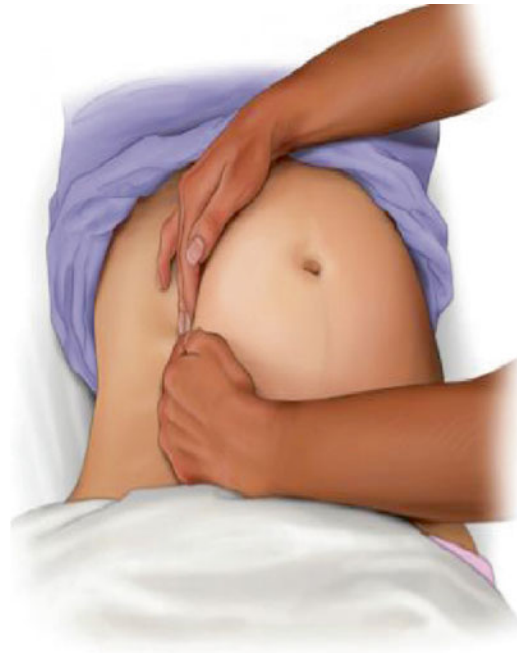
If defibrillation is available immediately then this takes precedence over BLS [8]. Details about defibrillation is on page 41.

Make sure the victim, any bystanders and you are safe:

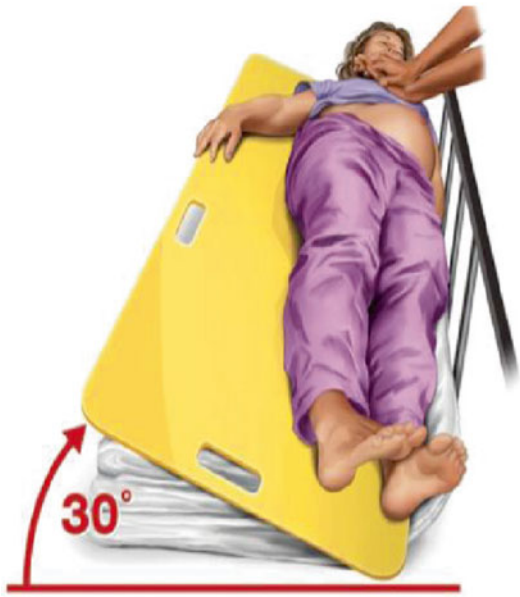
1. Don't waste precious time in starting resuscitation and don't wait for help to arrive.
2. Check responsiveness (shake and shout) – to confirm whether she is responsive or not.

3. Turn the patient onto her back with left lateral tilt.

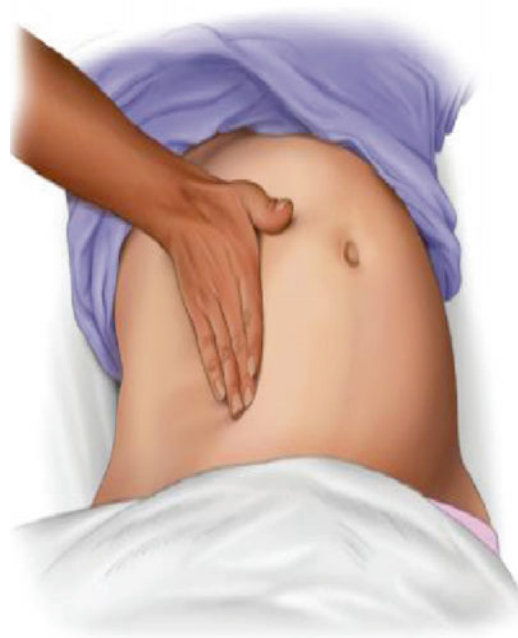
   Good positioning is critical. One person is dedicated to manually displacing the uterus upwards and to the left to relieve the aortocaval compression. A left lateral tilt of 15° on a firm surface will relieve aortocaval compression in the majority of pregnant women. A pillow can be placed under the right buttock. Using soft surfaces such as pillows or blankets is not nearly as effective and compromises effective chest compressions, but is better than leaving the woman supine. Devices such as the Cardiff resuscitation wedge (a wooden frame inclined at a 20–30° angle is specially designed for performing CPR during pregnancy) may be used.
   - Left uterine displacement: If rescuer is on patient's left side, then use the two-handed technique.
   - If rescuer is on patient's right side, use the one-handed technique, depending on the positioning of the resuscitation team.



LUD with two-handed technique [9].



Patient in 30° left lateral tilt using a firm wedge to support pelvis and thorax [9].



LUD using one-handed technique [9].

4. Check breathing (look, listen and feel).

   Assess breathing for no more than 10 s by looking for chest movements, listening for breath sound and feeling for the movement of air.

   Absence of breathing in the presence of a clear airway is now used as a sign of absence of circulation.

   Agonal gasps are present in up to 40 % of cardiac arrest victims. It should be emphasised during training that agonal gasps occur commonly in the first few minutes after sudden cardiac arrest; they are an indication for starting CPR immediately and should not be confused with normal breathing.

   If breathing is present, recovery position is given, manage for help and ambulance, shift her for further advanced care and teamwork, etc.

   If there is no breathing, give two effective breaths to her. One should use barrier devices if available to provide ventilation like pocket mask, face shield and bag valve mask. Pinch the nose, seal your mouth over victim's mouth and deliver two effective breaths that can make the chest rise.
5. *Check circulation*: Assess for circulation for 10 s only (for signs of circulation – look for carotid, not included in latest guidelines). One can assess at the same time while assessing breathing.
6. *If circulation is present*: Continue rescue breathing, and check circulation every minute.
7. *Open the airways* (head tilt – chin lift, modified jaw thrust).

   Use suction if required or remove foreign body with care.

   In pregnancy, the airway is more vulnerable as there is increased risk of regurgitation and aspiration. Check the mouth for foreign body or any material.

   The airway is opened by triple manoeuvre which includes (1) neck extension by backward pressure on the forehead, (2) a jaw thrust by placing fingers behind the angle of the jaw and moving the jaw anteriorly (3) and depressing the chin downwards with both thumbs on tip of chin. This will displace tongue from the pharynx.

   Intubation should then be performed as soon as possible. Apply continuous cricoid pressure during ventilation and intubation due to risk of regurgitation. Early intubation is mandatory, with attention to the use of a smaller endotracheal tube (ETT) 0.5–1 mm smaller in internal diameter than which is used for a non-pregnant woman because the airway may be narrowed from oedema. Its correct position should be confirmed with capnography as per ACLS. Avoid nasal intubation because of the increased mucosal friability during pregnancy.

   A laryngoscope with a shorter handle is useful as the presence of large breasts may interfere with access.
8. If no circulation (or you are not sure for circulation):

   (A) Immediately start chest compression.
      1. One hundred chest compressions per minute.
      2. 30:2 ratio for compression/breath (one cycle)
      3. Gravid uterus >20 weeks limits the effectiveness of chest compressions. It may be shifted away from the IVC and aorta by placing in LUD or by pulling the uterus to the side. This may be accomplished manually or by placement of a rolled blanket, pillow or other object under the right hip and lumbar area. Give left lateral tilt of minimum 15°.
      4. Place the heel of one hand just above the centre of the sternum, with the other hand on top of the first.
      5. Interlock the fingers of both hands and lift the fingers to ensure that pressure is not applied over the patient's ribs.
      6. Position yourself above the patient's chest and with your arms straight.
      7. Do not bend your elbow when doing chest compressions; doing so

will deliver a weak, ineffective chest compression.

8. Perform chest compressions higher, slightly above the centre of the sternum as there is an elevation of the diaphragm and abdominal contents because of pregnancy.
9. Do not apply any pressure over the top of the abdomen or bottom tip of the sternum.
10. "Push hard and push fast". Press down on the sternum to depress it 3–4 cm at a rate of 100 beats per minute.
11. Change the person doing chest compression about every 2 min to maintain efficiency but avoid any delays in the changeover.

(B) *Ventilation breaths*: Keep an airway open and provide ventilation with appropriate adjuncts. This might be a pocket mask or self-inflating bag with mask (AMBU bag). Bag and mask ventilation should be undertaken until intubation can be achieved.

Each ventilatory breath should last about 1 s and should make the chest rise as if a normal breath. Oxygen in high flow should be added as soon as possible.

Tracheal intubation is the most effective way of providing adequate ventilation and should be performed as soon as a trained member of staff is available.

(C) *Intubated ventilation*: Once the patient is intubated, ventilation should continue at ten breaths per minute but does not need to be synchronised with chest compressions. It should then be uninterrupted. During pregnancy, there is increased oxygen requirements and rapid onset of hypoxia, and so it is important to ensure optimal oxygen delivery by high-flow 100 % oxygen to whatever method of ventilation is being employed.

(D) Mouth to mouth breathing (not usually required)

There have been few incidents of rescuers suffering adverse effects from undertaking CPR, with only isolated reports of infections such as tuberculosis (TB) and severe acute respiratory distress syndrome (SARS). Transmission of HIV during CPR has never been reported.

Ensuring head tilt and chin lift.

Close the soft part of the patient's nose with your thumb and index finger.

Open her mouth a little but maintain chin lift.

Take a breath and place your lips around her mouth, making sure that you have good seal.

Blow steadily into her mouth over 1 s watching for her chest to rise.

Maintaining head tilt and chin lift, take your mouth away from the patient and watch for her chest to fall as the air comes out.

Take another breath and repeat the sequence to give another effective breath.

Return to chest compressions quickly.

If circulation is present but no breathing (respiratory arrest), continue rescue breathing and bag and mask ventilation at a rate of ten breaths/minute and a tidal volume large enough to raise the chest. It must be noted that hyperventilation is harmful and should be avoided [4].

Recheck the circulation every ten breaths, taking no more than 10 s each time.

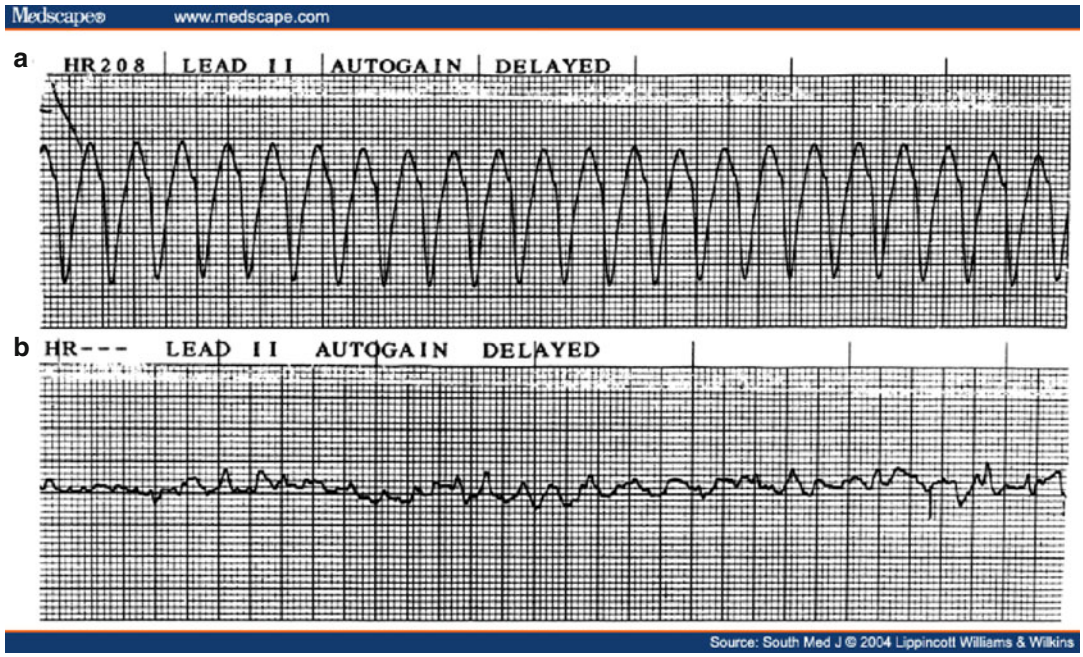
If the patient starts to breathe on her own but remains unconscious, turn her into the recovery position and apply oxygen at the rate of 15 l/min.

Check her condition and be ready to turn her back to start rescue breathing if she stops breathing.

9. Continue CPR till help arrives or patient is shifted in ICU or pt is revived.
10. Change the rescuer after five [5] such cycles to avoid fatigue of rescuer; otherwise, it may lead to ineffectiveness.
11. Manage for immediate ECG, if possible.

12. *Defibrillation*: In majority of these types of cases, ventricular fibrillation or flutter is dying rhythm of ECG. In this case defibrillation with defibrillator is the definitive treatment. Defibrillate the victim using standard ACLS defibrillation doses. If automated external defibrillator (AED) is available, attach it, analyse rhythm and defibrillate. The most frequent initial rhythm in the context of sudden collapse (i.e. not preceded by gradual deterioration or illness) is ventricular fibrillation (VF). The AED allows for early defibrillation by lesser-trained personnel as it performs rhythm analysis and gives information by voice or visual display, and the delivery of the shock is then delivered automatically.
13. There is no evidence that shocks from a direct current defibrillator have adverse effects on the heart of the foetus [10]. If foetal or uterine monitors are in place, remove them before delivering shocks.

If AED is not available, then differentiate the shockable and not shockable rhythm by ECG and plan defibrillation as follows:



Shockable Rhythms

Shockable rhythms are treated with a single shock.

It is followed by immediate continuation of CPR without stopping for a rhythm or pulse check.

Every 2 min the rhythm should be assessed and if necessary a further shock is delivered.

On the shockable side of the algorithm, adrenaline (epinephrine) 1 mg IV is given immediately before the third and every subsequent alternate shock, i.e. approximately every 4 min. Amiodarone 300 mg IV is given before the fourth shock.

Defibrillator Safety Management

The patient should not be near inflammable fluids, fumes or chemicals that could ignite.

If paddles are used rather than the pads:

Keep paddles pressed firmly on the patient's chest – failure to do so may result in a flash arc.

The defibrillation paddles should be placed back in the appropriate containers as soon as the shocks have ended.

Do not discharge the paddles in the air [11].

The operator's hands should be dry.

Prior to applying a shock, the operator must check that nobody has direct or indirect contact with the patient. Advise all bystanders and personnel to "stand clear".

Non-shockable Rhythms

In case of the non-shockable rhythm, i.e. pulseless electrical activity or asystole, adrenaline (epinephrine) 1 mg should be given intravenously immediately.

Atropine 3 mg IV may be given once for asystole or slow rate, i.e. <60 bpm. This will minimise any vagal tone if present.

Along with CPR, correct reversible causes of cardiac arrest which are as follows:

Four Hs

Hypoxia

Hypovolaemia (haemorrhage or sepsis)

Hyperkalaemia and other metabolic disorders

Hypothermia

Hypoxia

Effective ventilation and supplementation is ensured with 100 % oxygen delivery as soon as possible.

Regular checks should be done to ensure adequate airway and ventilation.

Hypovolaemia

Fluid replacement should be given if hypovolaemia is suspected [12].

Large-bore cannula is inserted to allow rapid infusion of fluids [12].

Hyper/Hypokalaemia, Hypocalcaemia and Metabolic Disorders

Electrolyte imbalance may lead to cardiac arrest – it should be confirmed or ruled out with arterial blood gases and serum electrolytes.

ECG monitoring and baseline blood tests should be done for urea and electrolytes at regular interval.

Hypothermia

Hypothermia is defined when the core temperature is below 35 °C.

Record the patient's temperature as soon as possible after a cardiac arrest. In case of hypothermia, consider active warming with warm blankets.

Four Ts

Thromboembolism

Toxicity (drugs associated)

Tension pneumothorax

Cardiac tamponade

14. *Perimortem CS*: It is important to remember that both mother and infant may die if the provider cannot restore blood flow to the mother's heart. When GA >24 weeks and if CPR is not effective, consider the need for an ER caesarean delivery which requires to begin the delivery about 3–4 min after cardiac arrest. It will also facilitate effectiveness of CPR.

    Peri-mortem caesarean section should be performed where resuscitation is taking place. Peri-mortem caesarean section packs are available on resuscitation trolleys in all areas where maternal collapse may occur [8].

    The principle of successful CS delivery is "rapid incision, rapid delivery and rapid closure".

    It is best obtained with large midline vertical abdominal incision and large uterine incision and closure with large running sutures in a single layer.

    After delivering of a baby, immediately placenta is removed manually.

    Immediately hand over the baby to neonatologist.

    There is no need to scrub the abdomen.

    There is no need to administer anaesthesia, as patient is not responsive.

    CS will be relatively bloodless, since there is no circulation and cardiac output.

    Chest compressions and ventilation should be continued.

If mother is resuscitated and pulse returns, then immediately shift her to ICU.

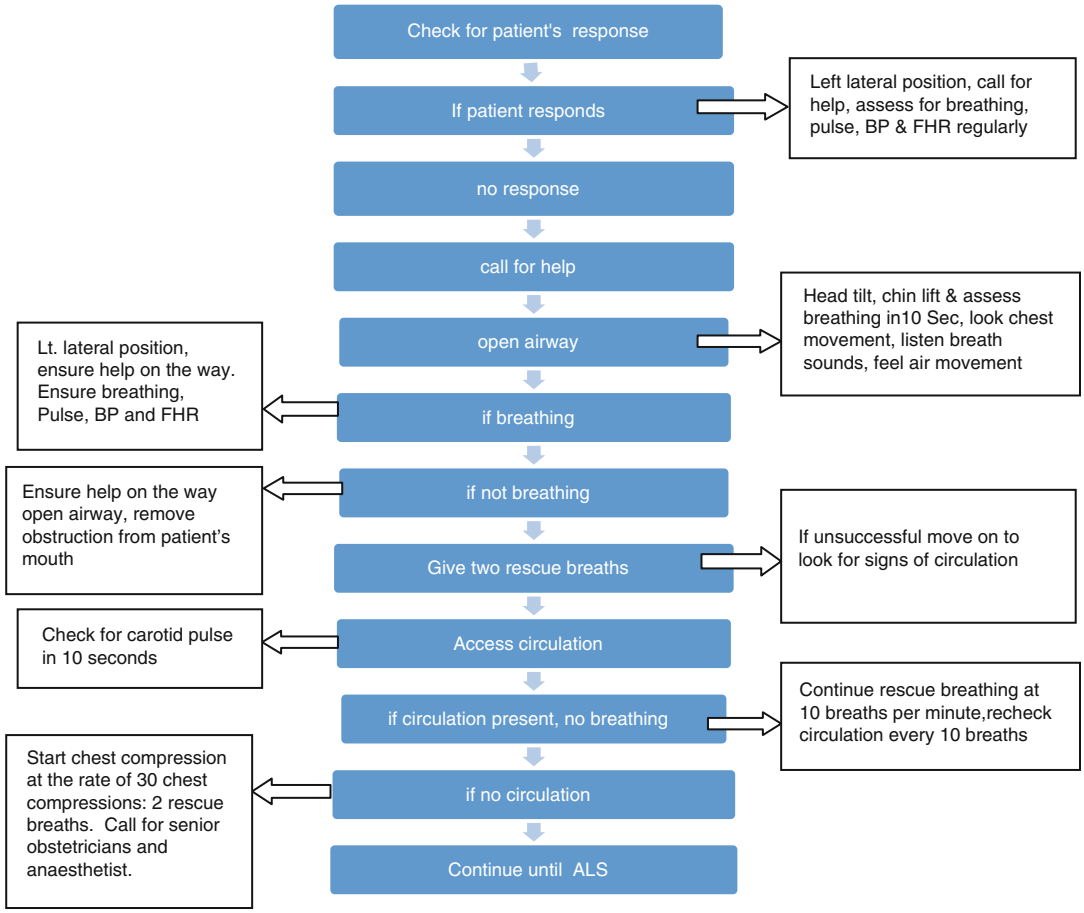
Broad-spectrum antibiotics are started.

Best survival rate for infants >28 weeks occurs when delivery of infant occurs in <5 min after the mother's heart stops beating.

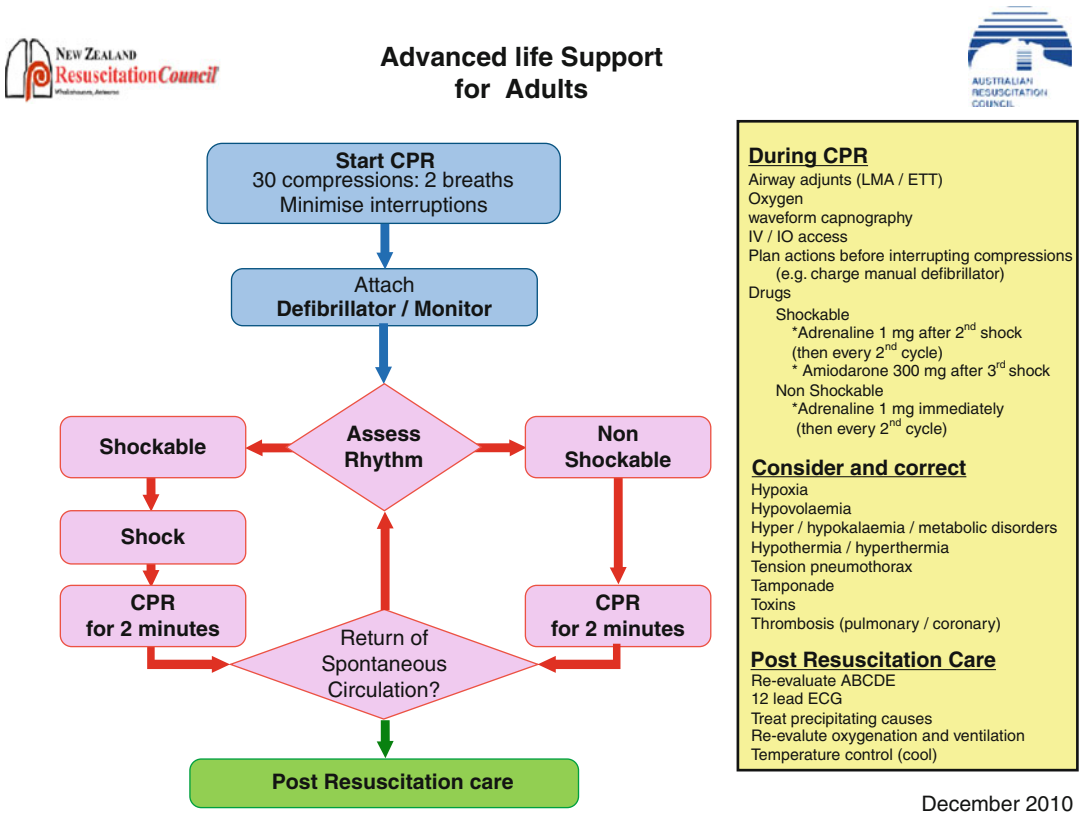
A study was conducted Katz v, Balderston K and DeFreest M of cases from 1985 until 2004. There were 38 cases of perimortem cesarean delivery identified; 34 infants survived (3 sets of twins, 1 set of triplets. Of the 34 infants (25–42 weeks' gestation), time of delivery after maternal cardiac arrest was available for 25. Eleven infants were delivered within 5 minutes, 4 were delivered from 6 to 10 minutes, 2 were delivered from 11 to 15 minutes, and 7 were delivered more than 15 minutes. Of 20 perimortem cesarean deliveries with potentially resuscitatable causes, 13 mothers were resuscitated and discharged from the hospital in good condition. One other mother was successfully resuscitated after the delivery, but died within 24 hours from complications related to AFE. In 12 of 18 reports that documented hemodynamic status, cesarean delivery preceded return of maternal pulse and blood pressure, often in a dramatic fashion. Eight other cases noted improvement in maternal status. Importantly, in no case was there deterioration of the maternal condition with the CS delivery [13].

CS might be necessary to accomplish a successful resuscitation even if the foetus has died [4].

15. Document the procedure in the patient's record.



Algorithm 5.1 Basic life support



Algorithm 5.2 Advanced life support [8]

Drugs Used in Adult Cardiac Arrest

Adrenaline Adrenaline causes vasoconstriction and increases perfusion to the myocardium and the cerebrum [3].

Usually prescribed *dosage*: IV adrenaline 1 mg (1 mL of 1:1000 or 10 mL of 1:10,000).

Ventricular fibrillation/pulseless ventricular tachycardia after initial counter shocks have failed (after second shock and then after every second cycle).

Asystole and electromechanical dissociation (pulseless electrical activity) in initial cycle (and then every second cycle).

Do not interrupt CPR to give medications.

Adverse effects include tachyarrhythmias and hypertension after the person is resuscitated.

Amiodarone

Amiodarone is an antiarrhythmic drug. It is given for VF/pulseless VT (between the third and fourth shock when refractory to defibrillator shocks and a vasopressor).

If three unsuccessful defibrillation shocks for VF/VT, the adult bolus dose of 300 mg (5 mg/kg) of amiodarone IV should be given. An additional bolus of 150 mg could be considered. This may be followed by an infusion of 15 mg/kg over 24 h.

Major side effects include bradycardia, heart block and hypotension.

Lignocaine and amiodarone should not be given together [14, 15].

Sodium bicarbonate 50 mmol IV should only be given to patients if the arrest is associated with hyperkalaemia. Otherwise, it should be given in response to the clinical condition of the patient, e.g. with severe acidosis pH less than 7.1, base excess greater than 10. Usually in cases of septicaemia and diabetic ketoacidosis, it is required. Bicarbonate should be used with caution, because rapid correction of maternal acidosis can reduce the compensatory hyperventilation [16].

Magnesium sulphate 8 mmol (4 mL of 50 % solution) may be used for refractory VF. Other use may be possible in hypomagnesaemia, torsade de pointes (a persistent VF) or digoxin toxicity. These are unlikely in pregnancy.

Calcium 10 mL 10 % calcium chloride (6.8 mmol Ca2+) IV can be used if it is thought that pulseless electrical activity (PEA) is caused by hyperkalemia, hypocalcaemia, overdose of calcium channel blocking drugs or overdose of magnesium (for treatment of pre-eclampsia). Calcium can be given as bolus if the patient has no output, but not in the same line as sodium bicarbonate as this will precipitate.

Inotropic agent *Dopamine*: Initial intravenous infusion of 2–5 μg/kg/min and increase up to 5–10 μg/kg/min as needed

Dobutamine: Intravenous infusion of 2–40 μg/kg/min

Noradrenaline: 2–4 μg/kg/min dose depending upon blood pressure

Post Successful Resuscitation Care

1. Consider to order the following investigations after successful resuscitation:

   Serum electrolytes
   Arterial blood gases
   Electrocardiogram (ECG)
   Blood glucose levels [17]

2. If perimortem caesarean performed outside the O.T., then transfer to theatre for wound closure.
3. Arrange transfer of the patient to the intensive care unit if appropriate following consultation with senior obstetrician and senior anaesthetist.
4. Re-evaluate for oxygenation and ventilation.
5. Manage for temperature control.
6. Arrange a consultation for friends and relatives with medical personnel to discuss the event (debrief).
7. The patient should be examined for resuscitation-related injuries, e.g. rib fractures [17].
8. Maternal treatment includes maintenance of fetal well-being. The interpretation by sonography or external electronic monitoring may be of utmost importance [7].

Communication and Teamwork

Wherever possible, call senior personnel from the obstetric, anaesthetic and midwifery professions for communication. Ensure that the family is looked after and kept informed about patient's conditions and management done. Document all interventions accurately and with timings.

Conclusion

CPR in the patient with cardiac arrest should be performed with consideration of the physiological changes associated with pregnancy. Standard algorithm should be applied according to basic and advanced life support protocols, with few exceptions. Attention to lateral displacement of the uterus, more aggressive airway management and early consideration of emergency caesarean delivery are major modifications in the management of maternal arrest. Caesarean delivery should be performed as soon as possible. Immediate caesarean delivery not only improves survival of infant but also facilitates maternal resuscitation. All obstetric units that provide care to pregnant patients must be ready to tackle the unpredictable

catastrophic events. They should regularly perform emergency obstetric drills including CPR and should train their staff for it. Everyone should know about how to perform CPR.

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Part II

Procedures and Monitoring in the HDU/ICU Unit

Role of Imaging in Noninvasive Monitoring in Obstetric Intensive Care Unit

6

Santosh Singhal, Rishabh Bora,
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The critically ill obstetric patient presents a unique clinical challenge to the intensivist because of:

- Maternal physiological adaptations to pregnancy
- Pregnancy-specific conditions requiring critical care
- Presence of foetus, whose wellbeing is linked to mother

So a multidisciplinary approach is required from the intensivist, obstetrician, anaesthetist, neonatologist, pathologist and radiologist.

The reasons for ICU admission are:

- Conditions related to pregnancy – Eclampsia, severe pre-eclampsia, antepartum and postpartum haemorrhage, amniotic fluid embolism, pulmonary embolism, acute fatty liver of pregnancy, peripartum cardiomyopathy, aspiration syndrome, uterine perforation, uterine rupture, inversion of uterus, ruptured ectopic pregnancy, septic shock and infections
- Medical diseases aggravated during pregnancy – Congenital heart disease, rheumatic and nonrheumatic valvular disease, pulmonary hypertension, anaemia, renal failure, etc.
- Obstetric predisposition to particular medical condition – Cerebral venous sinus thrombosis, hepatitis E infection and subarachnoid haemorrhage
- Conditions not related to pregnancy – Trauma, asthma, diabetes, autoimmune disease, etc.

In all app. 0.9 % mothers require admission in ICU, and the mortality ranges from 5 to 20 %.

Imaging is a very good noninvasive tool to confirm any diagnosis suspected on the basis of history, signs and symptoms and lab investigations. The imaging modalities to be used may be:

- Ultrasound, one of the most utilized, safe, can be used bedside, inexpensive and is extremely informative
- Colour Doppler, for added value at times
- X-ray, chest X-ray (CXR) is the one used frequently
- MRI, for brain, abdomen and pelvis
- CAT scan, for head, chest, abdomen and pelvis, used less for fear of radiation to foetus
- Echocardiography, very useful in cardiac conditions
- Nuclear medicine scans

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Table showing amount of radiation to foetus in different procedures

Procedure	Foetal dose (millirads)
Chest X-ray	<1
Cervical spine plain film	<1
CT thorax	13–1300 (mean 600)
CT abdomen	250
CT head	<1000
Helical CT pulmonary angiogram (CTPA)	<50
V/Q scan	<100
Barium enema	700–1600

The danger of radiation exposure to foetus has been unnecessarily overestimated. Any exposure less than 5 rad is safe. No single exposure is anywhere near that.

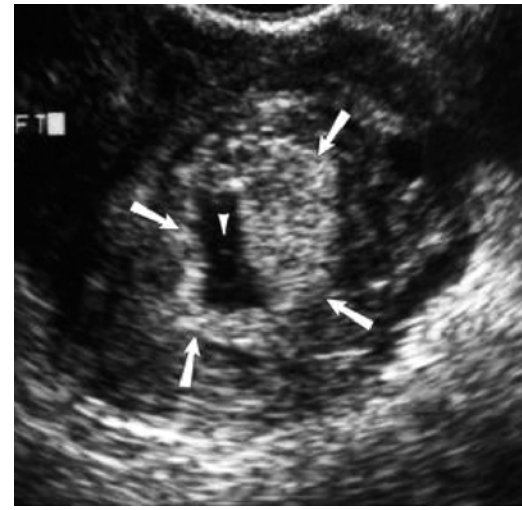

Fig. 6.1 A 36-year-old woman with tubal ectopic pregnancy after artificial insemination. Endovaginal sonogram of left adnexa shows echogenic tubal ring (*arrows*) containing yolk sac (*arrowhead*)

Ultrasonography

*S*onography is essential in evaluating pelvic pain and vaginal bleeding in women of childbearing age because many causes of these two presentations have suggestive or definitive sonographic findings. U/S should be done in all pregnant patients as primary investigation to know location of pregnancy, gestational age, viability, number of gestations, placental location and foetal wellbeing.

Ectopic Pregnancy

Common sonographic findings include one or a combination of the following: cystic or solid adnexal mass, dilated and thick-walled fallopian tube (adnexal ring), free echogenic or sonolucent intraperitoneal fluid, hematosalpinx and an extrauterine gestational sac containing a yolk sac with or without an embryo [1] (Figs. 6.1 and 6.2). Colour-flow Doppler imaging may be helpful in diagnosis of ectopic pregnancy when a trophoblastic Doppler flow signal is present. Taylor et al. [2] reported trophoblastic flow signals (high-velocity, low-impedance flow) in 54 % of ectopic pregnancies in their series. In a patient with a positive pregnancy test, the isolated finding of

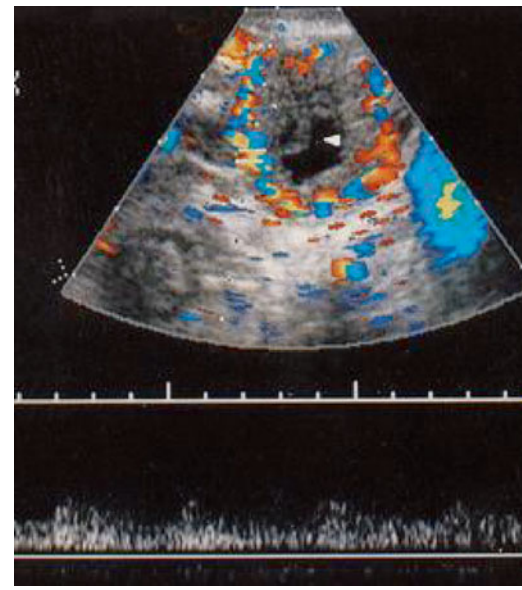

Fig. 6.2 A 26-year-old woman with live tubal ectopic pregnancy. Endovaginal sonogram shows live right-sided tubal ectopic pregnancy with "ring of fire" sign of trophoblastic flow as indicated by Doppler tracing waveform. *Arrowhead* points to embryo with positive cardiac activity at real time

free intraperitoneal fluid in the presence of an empty uterus has been shown to carry a 69 % specificity and a 63 % sensitivity for the diagnosis of an extrauterine gestation [1]. In particular, echogenic fluid has a positive predictive value of

93 % for the presence of a bleeding or ruptured ectopic pregnancy.

Sonography is valuable in characterizing extratubal ectopic gestations such as intraabdominal (Fig. 6.3a, b), cervical and cornual pregnancy. Cornual, or interstitial, gestations account for as many as 3 % of all ectopic pregnancies and carry a high mortality rate as a result of delayed rupture with extensive haemorrhage. Original sonographic descriptions include an eccentric intrauterine location and thinning of the surrounding myometrial mantle to less than 5 mm. The interstitial line (a thin echogenic line extending from the endometrial canal to the cornual sac or hemorrhagic mass) has been described as a highly specific and sensitive sign of interstitial pregnancy [3]. Ackerman et al. [3] saw this interstitial line in 92 % of interstitial ectopic pregnancies in a retrospective 7-year study. Asymmetrically increased low-resistance flow in a uterine cornu may also be a secondary sign of an interstitial pregnancy [3]. Care must be exercised to avoid misinterpreting a normal intrauterine pregnancy in an anomalous uterus – such as a septate or bicornuate uterus – as an interstitial pregnancy. Cervical pregnancies have a worse prognosis than tubal pregnancies because of the potential for uncontrollable haemorrhage. Once a gestational sac is identified in the cervix, a crucial part of diagnosis is to differentiate a cervical pregnancy from an abortion in progress. Features characteristic of a cervical pregnancy include a round or oval noncrenated sac, the presence of foetal cardiac activity, a closed internal os and constant sac shape and location on close follow-up sonograms.

Placenta previa

U/S studies done earlier in this pregnancy are invaluable. A midline longitudinal scan is used before and after evacuating a full bladder in order to minimize distortion of lower uterine segment. Translabial or TVS may be used if technical difficulties are there [4].

Placenta accreta

Placenta accreta can be diagnosed by U/S, mostly by the absence of retroplacental hypoechogenic zone of the decidua/myometrium [5].

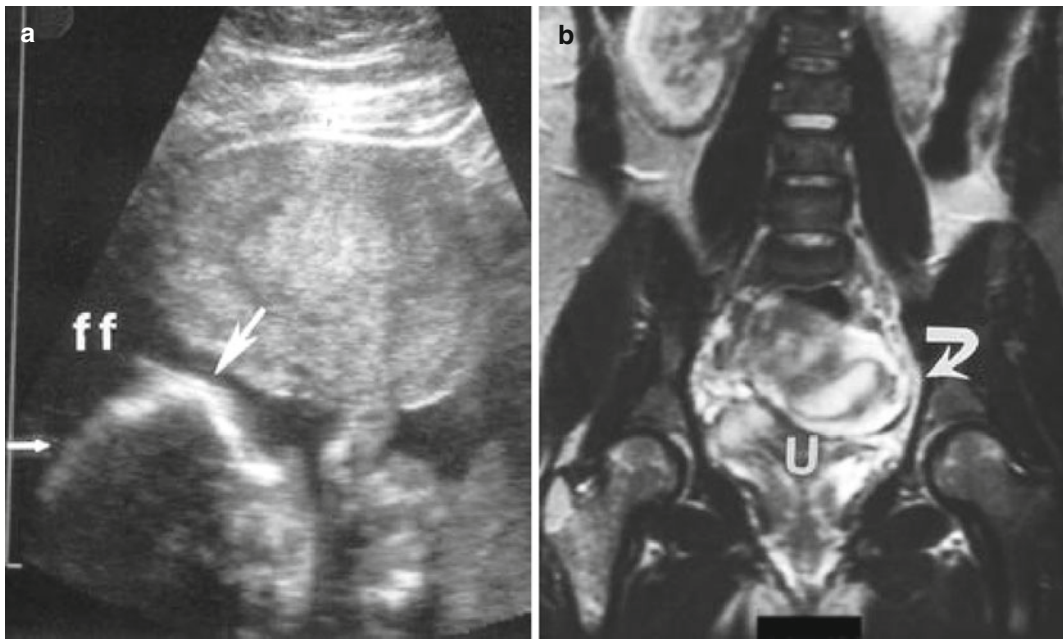

Fig. 6.3 (**a**) A 32-year-old woman with intraabdominal pregnancy. Transverse transabdominal sonogram shows empty uterus with thickened endometrium. Free intraperitoneal fluid (*ff*) and extrauterine pregnancy is seen. *Arrows* point to foetal skull. (**b**) A 32-year-old woman with intraabdominal pregnancy. Coronal T2-weighted MR image reveals presence of empty uterus (*U*) and intraabdominal pregnancy (*arrow*)

Abruptio Placentae and Retroplacental Hematoma

Placental abruption presents with painful vaginal bleeding, consumptive coagulopathy, acute renal failure resulting from acute tubular or cortical necrosis and foetal distress. Sensitivity of U/S is poor (40 %) in diagnosing this. Sometimes U/S can show retroplacental clot but may be difficult to distinguish from normal placental venous lakes. MRI is a better modality for it (Fig. 6.4).

Uterine Dehiscence and Rupture

Reported sonographic signs of uterine rupture include the identification of the protruding portion of the amniotic sac, an endometrial or myometrial defect, an extrauterine hematoma and hemoperitoneum (Figs. 6.5 and 6.6).

Puerperal Ovarian Vein Thrombosis

Ovarian vein thrombosis is an uncommon, but potentially fatal, postpartum complication. The pathogenesis is postulated to be retrograde propagation of thrombosed myometrial veins draining an infected placenta. The diagnosis is critical to

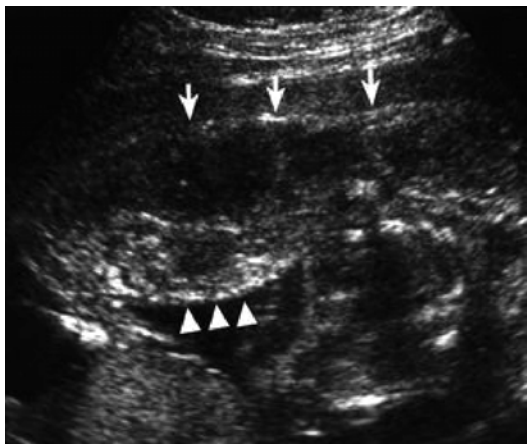

Fig. 6.4 A 32-year-old woman in second trimester with placental abruption presenting with vaginal bleeding. Sagittal transabdominal sonogram shows thickened anterior placenta with extremely heterogeneous echo texture. Portion of placenta closest to maternal surface (*arrowheads*) is much more hypoechoic than remainder of placenta (*arrows*), which is more suggestive of retroplacental abruption. After miscarriage, histopathologic examination revealed placental abruption with infarct involving at least 50 % of placenta

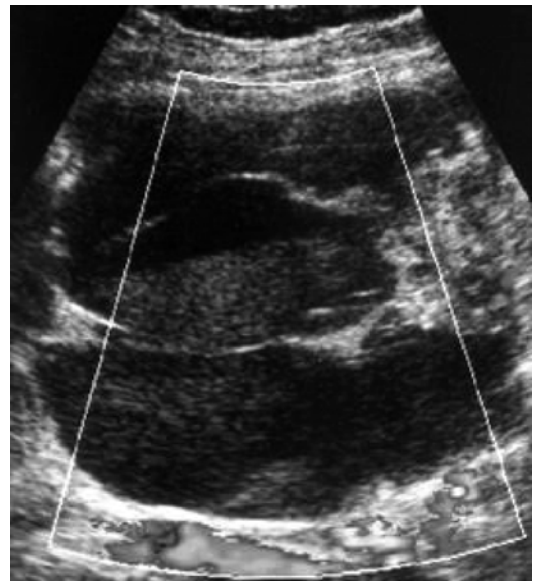

Fig. 6.5 A 36-year-old woman with uterine rupture after prolonged induction of vaginal delivery. Transverse image of pelvis shows complex hematoma. Complex fluid collection and clinical symptoms suggested diagnosis. Uterine rupture was confirmed at surgery

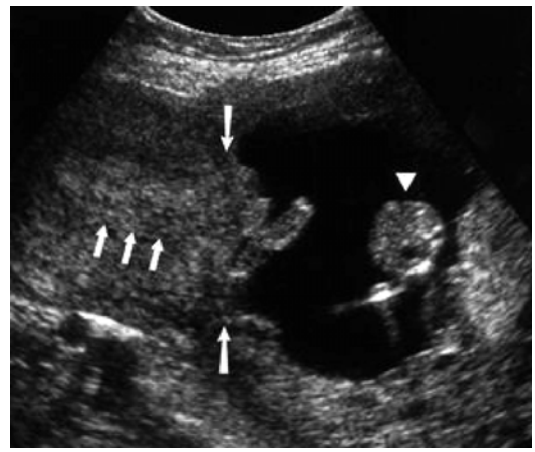

Fig. 6.6 A 28-year-old woman with surgically proven intraabdominal pregnancy resulting from uterine dehiscence because anterior placenta percreta grew anteriorly through previous caesarean scar. Sagittal endovaginal sonogram shows empty uterus, myometrial defect anteriorly and inferiorly (*long arrows*) and extrauterine pregnancy. *Arrowhead* points to foetal abdomen. *Short arrows* indicate endometrium

make in postpartum patients because of the risk of pulmonary embolism.

The sonographic diagnosis of ovarian vein thrombosis is mainly based on identifying the dilated, noncompressible ovarian vein extending into the inferior vena cava in a postpartum woman with signs of infection. Confirming the ovarian vein drainage into the inferior vena cava is important to avoid mistaking the dilated vein for a dilated ureter, dilated fallopian tube or retrocecal appendicitis. The lateral displacement of the ovarian vein by the gravid uterus and its tortuous course during pregnancy may further mimic the sonographic appearance of bowel with thickened wall or appendicitis. The venous thrombosis may extend in a retrograde fashion into the iliofemoral veins, potentially mimicking lower extremity deep venous thrombosis.

PPH and Retained Products of Conception

Retained products of conception after abortion or full-term pregnancy may cause secondary postpartum haemorrhage or may serve as a nidus for uterine infection. Predisposing factors include the presence of a succenturiate lobe or placenta accreta, increta or percreta, preventing complete placental delivery. Sonographic findings include endometrial expansion of heterogeneous echogenic material and focal areas of hyperechogenicity that may represent retained placental calcifications. Retained trophoblastic tissue exhibits low-resistance arterial flow, which is uncommonly seen with endometritis.

Adnexal torsion in pregnancy

Significant ovarian enlargement with absence or reduction of arterial or venous flow on colour Doppler [6].

Uterine perforation

In underdeveloped countries abortion by unskilled attendants may cause uterine perforation. Under U/S guidance a dilator can be passed.

Acute fatty liver of pregnancy

Presents in third trimester or early puerperium with nausea, vomiting and anorexia. Up to 70 % may have jaundice and fever. U/S may show fat deposition in the liver but the hallmark microvesicular steatosis may not be seen on U/S. Rarely rupture or necrosis of the liver may be identified on U/S.

Compression Ultrasonography

Deep vein thrombosis

Compression ultrasound [7] is a highly sensitive and specific modality for the detection of lower extremity DVT, without the need for radiation or contrast exposure. This is a modified 2-point compression technique that focuses on the highest probability areas, decreases study time to 5 min and provides same sensitivity and specificity. Lack of compressibility of vein rules in DVT. Thrombus in pelvic veins will not be detected by this.

Ultrasound and Doppler of eyes

Retinal vascular changes are present in 30–100 % of pre-eclampsia patients; the most frequent finding is vasoconstriction of retinal arterioles. The evaluation of the ophthalmic arterial flow by eco Doppler is helpful. In severe forms the increase in the impedance of orbital vessels is noted.

X-Rays

Chest X-ray is the one needed most frequently.

Amniotic fluid embolism

Though rare (1:8000–1:80,000) it is one of the most dreadful events in obstetrics. In the first phase there is profound respiratory failure with deep cyanosis and cardiovascular shock followed by convulsions and profound coma in second phase.

Chest X-ray may show an enlarged right atrium and ventricle and prominent proximal pulmonary artery and pulmonary oedema.

Pulmonary embolism

Nonspecific CXR findings, such as atelectasis, unilateral pleural effusion, areas of consolidation or an elevation of hemidiaphragm. Chest X-ray with lateral views can rule out pneumonia or pulmonary oedema.

Peripartum cardiomyopathy

Affecting in late pregnancy or early puerperium. Signs and symptoms are similar to heart failure [8]. CXR shows cardiomegaly and pulmonary oedema.

Acute respiratory distress syndrome (ARDS)

Presents as dyspnoea, tachypnoea and tachycardia. Occurs with septic abortion and occasionally in context of a uterine perforation. CXR may be normal immediately after the precipitating event but B/L diffuse infiltrates typically occur within 4–24 h [9].

Aspiration pneumonia

Fever and cough during labour or immediate postpartum period. CXR shows a radiographic infiltrate in a dependent location (basilar segments of the lower lobes or posterior segments of the upper lobes).

Magnetic Resonance Imaging (MRI)

MRI provides superior contrast resolution compared with CT, uses nonionizing radiation and permits imaging in more than one plane.

MRI abdomen

Is helpful in placental location and associated pathology like placenta accreta, increta and percreta, placental abruption, hydronephrosis, adnexal masses and pelvimetry. Multiplanar MR imaging offers a comprehensive assessment of the uterine wall and the peritoneal cavity.

It may show hepatic rupture, subcapsular haematoma or infarction in HELLP syndrome.

MRI brain

MRI is superior to CT for evaluation of cerebral infarction, tumour or infection. In eclampsia cerebral oedema and cerebral haemorrhage may be seen. MRI demonstrates B/L hyperintense lesions on T2-weighted images and hypointense lesions on T1-weighted images without diffusion restriction. MRI abnormalities are commonly located in occipital and parietal lobes.

Cerebral imaging in eclampsia is not always necessary unless there are focal changes, recurrent seizures, deterioration of patient's condition or the need to exclude other aetiologies like epilepsy, uncontrolled hypertension, lupus, intracranial haemorrhage, brain tumours, aneurysm, ITP, metabolic disorders, cerebral vasculitis, cavernous venous thrombosis, CVA and inadvertent vascular injection of injection used for epidural.

MR venography

It is a noninvasive and non-contrast requiring tool for diagnosing cerebral vein thrombosis.

CT Scan

CT head – Invaluable in cerebral haemorrhage.

CT abdomen – May show hepatic complications like rupture, subcapsular haematoma or hepatic infarction in HELLP syndrome.

CT pelvis – It is very useful for diagnosing ovarian vein thrombosis in puerperium (Fig. 6.7).

Helical CT Pulmonary Angiogram (CTPA)

It is superior to V/Q scan for diagnosis of pulmonary embolism, but now due to concern about increased susceptibility of maternal breast tissue to carcinogenic effects of ionizing radiation in pregnancy, it is not used as initial investigation [10].

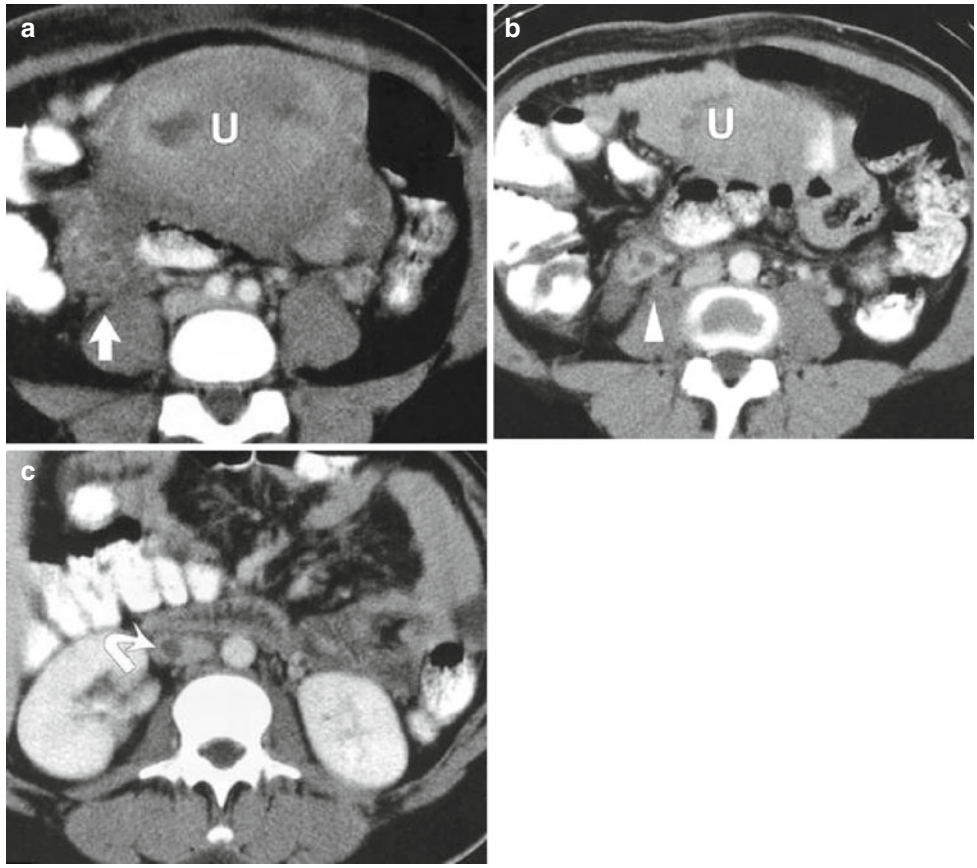

Fig. 6.7 (**a**) A 28-year-old woman with puerperal ovarian vein thrombosis presenting as persistent fever 3 days after uncomplicated vaginal delivery. Sequential axial enhanced CT images show postpartum uterus (*U*, **a** and **b**), enlarged heterogeneous ovary (*arrow*, **a**), dilated tortuous right ovarian vein with partial thrombosis (*arrowhead*, **b**) and partial thrombosis in pararenal inferior vena cava (*arrow*, **c**)

V/Q Scan or Radionuclide Lung Imaging

It is the primary screening technique used in the evaluation of pulmonary embolism in pregnancy. It demonstrates some areas of decreased radioactivity in lung field.

Positron Emission Tomography (PET)

When used for detection of pulmonary embolism, either total dose is reduced or ventilation phase is omitted in pregnancy. In lactating mothers breastfeeding is refrained for 24 h.

Echocardiography

Peripartum cardiomyopathy

There is severe left ventricular systolic dysfunction, ejection fraction <45 % [8]. Other nonspecific finding may be left atrial enlargement, mitral regurgitation and small pericardial effusion.

Amniotic fluid embolism

There is acute left and right heart failure or severe pulmonary hypertension.

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Basic Hemodynamic and Cardiac Monitoring in Obstetrics

7

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The cardiovascular system undergoes a series of changes during pregnancy, puerperium, and labor. These changes are beneficial for the mother and the fetus and ensure optimum fetal growth and development as well as protect the mother during the normal bleeding in the postpartum period. An understanding of these changes is imperative as the physiologic and anatomic adaptations to pregnancy influence the interpretation and evaluation of the pregnant woman's cardiac evaluation and hemodynamic monitoring.

These changes are briefly described in the table below (Table 7.1, Figs. 7.1 and 7.2).

The incidence of intensive care admission for pregnant and postpartum women ranges from 0.7 to 13.5 per 1,000 deliveries (Pollock et al. 2010). The most common indications for ICU admission are postpartum hemorrhage, sepsis, and the hypertensive disorders. Hemodynamic compromise is a common indication for intensive care unit (ICU) admission in the obstetric population manifesting as hypotension, hypertension, or occasionally as cardiac dysrhythmia (Table 7.2).

Hypertension in the obstetric patient is most commonly due to preeclampsia, which can lead to serious complications like pulmonary edema and renal failure.

Since pregnancy is a proarrhythmic state (increased estrogens and physiological changes in the cardiovascular system), the incidence of paroxysmal supraventricular tachycardia is increased in pregnancy. Nonetheless, cardiac dysrhythmias are a rare cause of hemodynamic compromise in the obstetric population.

Hemodynamic monitoring is the measurement and interpretation of biological system that describes the performance of cardiovascular system. It provides quantitative information about vascular capacity, blood volume, pump effectiveness, and tissue perfusion. A patient with hemodynamic compromise should be approached stepwise:

A. Clinical assessment
B. Basic cardiovascular monitoring
C. Preload and fluid responsiveness
D. Cardiac output monitoring
E. Assessment of cardiac contractility
F. Assessment of tissue perfusion

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Table 7.1 Physiological and anatomical changes in the cardiovascular system in pregnancy

Parameter	Change	Antenatal period	Labor	Postpartum
Plasma volume	Increase 50 %	Increases from 6 weeks, peaks 30–32 weeks	Remains same	There is fall due to blood loss
Stroke volume	Increase 20 %	Increases from early pregnancy, peaks at 20 weeks, gradually falls	During straining in 2nd stage (Valsalva), there is a fall	Normal in 6 weeks
Heart rate	Increase 15–20 bpm	Rise more in 2nd half	Further rise seen	Prelabor level by 1 h; normal in 6–12 weeks
Arterial blood pressure[a]	Decrease 10–15 %	Declines by 5–10 mmHg in 2nd T, normal values in 3rd T	Rise by 10–25 % during contractions	Normal values
Cardiac output[b]	Increase 40 %	Increases from 8 weeks, peaks at 32 weeks	Further increase by 25–50 %	Immediate postpartum rise by 80 %[b]; prelabor level in 1 h; normal by 6–12 weeks
Uterine blood flow	~10 % of cardiac output at term	–	–	–
Cardiac anatomy	Heart rotated cephalad and to the left	–	–	–
	Increase chamber size, particularly the left atrium			

[a]The reduction in blood pressure in pregnancy is predominantly secondary to a decrease in the diastolic component which in turn is due to reduction in systemic vascular resistance because of progesterone, the development of the placenta, and a low-resistance vascular bed

[b]The increased cardiac output that develops in pregnancy is further augmented during the third stage of labor as a result of autotransfusion of blood from the uteroplacental to maternal circulation as the uterus contracts

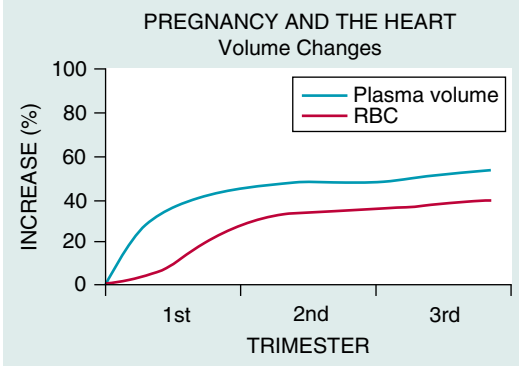

Fig. 7.1 Volume change in pregnancy

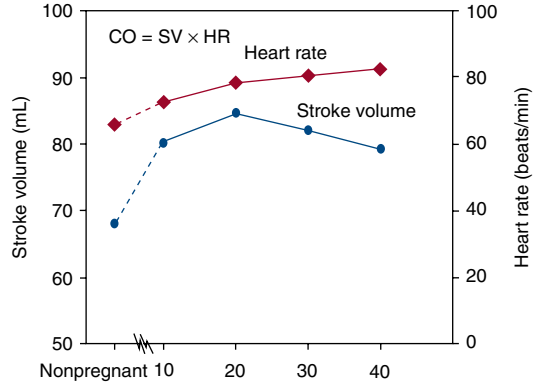

Fig. 7.2 Stroke volume (*SV*) and heart rate (*HR*) (CO = SV × HR) changes in pregnancy

Table 7.2 Common causes of maternal hypotension

Hypovolemic shock	Ruptured ectopic pregnancy, placenta previa, postpartum hemorrhage, trauma, uterine rupture
Septic shock	Puerperal sepsis, septic abortion
Cardiogenic shock	Preexisting valvular heart diseases, peripartum cardiomyopathy
Miscellaneous	Pulmonary thromboembolism, amniotic fluid embolism, uterine inversion

Clinical Assessment

On the background of a focused history, clinical examination is the fastest and least invasive hemodynamic monitoring available. A thorough medical and obstetric history in the antenatal and postnatal period should be elicited. A patient with inadequate global perfusion often presents with one or several of the following features:

- Weak distal pulses
- Agitation
- Confusion
- Poor capillary refill
- Cool extremities
- Diaphoresis
- Tachypnea
- Tachycardia[1]
- Hypotension
- Decreased pulse pressure
- Decreased urine output[2]

Basic Cardiovascular Monitoring

All critically ill patients should have electrocardiograph (ECG), arterial blood pressure, pulse oximetry monitoring along with arterial blood gas (ABG) monitoring and serum lactate measurement.

[1] Occasionally, bradycardia may be an underlying cause of low cardiac output.

[2] Decreased urine output in a patient with previously normal renal function and urine output is an important warning that renal perfusion is inadequate.

ECG Monitoring

A 12-lead ECG of the patient provides information about the heart rate, rhythm, ST-segment, and T waves. Heart rate is an important determinant of cardiac output (cardiac output= stroke volume × heart rate). Alterations in the ST-segment morphology may provide an early recognition of myocardial ischemia. ECG is also useful for the detection of arrhythmias.

Blood Pressure (BP) Monitoring

(a) Noninvasive

The systolic blood pressure represents the highest pressure occurring as a result of left ventricular contraction, whereas the diastolic blood pressure is a measure of the continuous forward flow during the period of cardiac filling and rest. The definition of low blood pressure is patient specific and interpreted in the context of the patient's usual blood pressure. Mean arterial pressure (MAP) is derived to represent the proportion of time in systole and diastole and is an approximation of oxygen perfusion pressure. When there is systemic vasoconstriction, the MAP may be normal or high even though the cardiac output is low. Conversely, if there is peripheral vasodilatation, as in sepsis, the MAP will be low although the CO is high. Hence, maintenance of MAP as a therapeutic target for vasoactive medications is a mainstay in the care of the patients with sepsis. Elevated blood pressure, especially if acute, is associated with tissue malperfusion, e.g., hypertensive encephalopathy.

Blood pressure is measured most commonly by manual or automatic noninvasive sphygmomanometry.

Limitations of Noninvasive BP

- An inappropriately small cuff may render a falsely high blood pressure reading and vice versa.

- Obesity and measurements taken with arm lower than the phlebostatic axis may contribute to falsely high reading.
- Similarly, falsely low reading can be obtained from too large a cuff or from the arm measured too high above the phlebostatic axis.
- Additionally, a number of artifacts and environment may contribute to false readings.

(b) Invasive measurement of blood pressure is a component of advanced hemodynamic monitoring but is included in this section for continuity.

Blood pressure can be measured invasively using an indwelling catheter in an artery. It gives a more accurate measure of BP as compared to noninvasive method. Indications for invasive blood pressure monitoring are:

- Unstable blood pressure
- Severe hypotension or severe hypertension
- Use of rapidly active vasoactive drugs: vasodilators, vasopressors, and inotropes
- Frequent sampling of arterial blood
- Pressure of an intra-aortic balloon pump
- Patients in whom noninvasive blood pressure is inaccurate

Blood pressure is not a sensitive indicator in patients with volume loss because of sympathetic responses as a compensatory mechanism. This is further compounded in pregnant patients because of the physiological increase in blood volume.

Pulse Oximetry

Oxygen saturation (SO2) measures the percent of hemoglobin which is fully combined with oxygen. Pulse oximetry, sometimes called the "fifth vital sign," is a noninvasive method of measuring oxygen saturation of a patient's hemoglobin saturation, (SpO2, i.e., oxygen saturation derived from a pulse oximeter) by using a light signal transmitted through tissue. Assessing oxygen saturation by SpO2 can aid in the assessment of risk in women with preeclampsia and may be a valuable early warning of pulmonary edema in women with preeclampsia if there is a downward trend in oxygen saturation. SpO2 will also be low in hypoxemic conditions such as acute blood loss (APH and PPH) and sepsis.

Normally, oxygen saturation on room air is in excess of 95 %. With deep or rapid breathing, this can be increased to 98–99 %. While breathing oxygen-enriched air (40–100 %), the oxygen saturation can be pushed to 100 %. Inspired oxygen levels are increased, such as breathing from a 100 % oxygen source. A low SpO2 can provide warning of hypoxemia before other signs such as cyanosis or a change in heart rate are observed.

The sigmoid shape of the oxyhemoglobin dissociation curve limits the degree of desaturation that can be tolerated – between 90 % and 100 % saturation, the partial pressure of oxygen in arterial blood (PaO2) will be 60 mmHg or above. Below 90 % saturation, the curve becomes steeper, and small drop in saturation corresponds to large drop in oxygen partial pressure. Taking into account the shape of the oxygen dissociation curve, SpO2 should be maintained > 92 % in most critically ill patients.

Limitations of Pulse Oximetry

SpO2 reading may be unreadable or unavailable if there is loss or diminution of the peripheral pulse as in:

- Proximal blood pressure cuff inflation/external pressure hypotension
- Hypothermia
- Low cardiac output
- Hypovolemia
- Peripheral vascular disease
- Valsalva maneuver such as seen in laboring patients
- Infusion of vasoactive drugs

Arterial Blood Gases

Arterial blood gas analysis typically measures:

- pH (acidity)
- PaCO2 (partial pressure of carbon dioxide)

- PaO2 (partial pressure of oxygen)
- CO2 (carbon dioxide content)
- Base excess (the loss of buffer base to neutralize acid)

These measurements are often used to evaluate oxygenation of the tissues and pulmonary function.

Normal Arterial Blood Gas in Pregnancy

- Mild chronic compensated respiratory alkalosis
- pH ~7.44
- PaCO2 28–32 mmHg
- PaO2 >100 mmHg
- HCO3- 18–22 mEq/L

PaO2, the partial pressure of oxygen in the plasma phase of arterial blood, determines the oxygen saturation of hemoglobin (SO2). The oxygen saturation and the concentration of hemoglobin determine the total amount of oxygen in the blood (CaO2).

$$\begin{bmatrix} CaO_2\left(mLO_2/dL\right) \\ =\left(1.34\times \text{hemoglobin concentration}\times SaO_2\right) \\ +\left(0.0031\times PaO_2\right) \end{bmatrix}$$

Increasing the PaO2 above 100 mmHg has its major effect on increasing the volume of oxygen dissolved in the plasma. Decreasing the PaO2, on the other hand, leads mainly to a reduction in the volume of oxygen carried by hemoglobin. Normal value for PaO2 while breathing room air is 97–100 mmHg.

Changes in PaCO2 occur as a result of change in alveolar ventilation either directly due to respiratory disorder or consequent to adaption of body to a metabolic process.

Serum Lactate

The normal serum lactate level in resting human is approximately 1 mmol/L (0.7–2.0). Elevated serum levels may represent poor tissue perfusion. The association of increased lactate level with circulatory failure, anaerobic metabolism, and the presence of tissue hypoxia has led to its utility as a monitor of tissue perfusion in critically ill patients.

Preload and Fluid Responsiveness

Starling's law states that "the mechanical energy set free in the passage from resting to the active state is a function of length of the fibre." For cardiac contraction, the preload is usually considered to be the end-diastolic pressure when the ventricle has become filled or greater than the cardiac inflow (end-diastolic blood volume) and greater is the stroke output of the ventricle.

An important step, in the presence of hypotension, therefore, is the assessment of preload and fluid responsiveness.

Generally, techniques for the assessment of cardiac preload attempt to measure, either directly or indirectly, the end-diastolic volume of the right or left ventricle or both in combination. These may be static measurements, such as the jugular venous pressure (JVP) and central venous pressure (CVP) (right ventricle), the pulmonary artery occlusion pressure (PAOP) (left ventricle), assessment of right ventricular end-diastolic volume (RVEDV) or global end-diastolic volume (GEDV) via thermodilution; or echocardiographic measurement of left ventricle end-diastolic area (EVEDA). At the bedside, the most readily available static preload assessment method is CVP, but static measurements, in general, have failed as a meaningful endpoint for fluid resuscitation.

Target Values of Central Venous Pressure

In contrast to static measures, dynamic indices of preload rely on the changing physiology of heart-lung interactions to determine whether a patient will benefit from increased preload. Dynamic indices apply a controlled and reversible preload variation and measure the hemodynamic

Normal	0–5 mmHg
Target (for volume resuscitation)	6–10 mmHg
Normal (if ventilated)	5–10 mmHg
Target of vol. resuscitation (if ventilated)	10–15 mmHg

response. This can be done by observing the cardiovascular response to positive-pressure ventilation, or to reversible preload-increasing maneuvers, such as passive leg raising. These indices include:

- Pulse pressure variation (PPV)
- Systolic pressure variation (SPV)
- Stroke volume variation (SVV)
- Inferior vena cava (IVC)/superior vena cava (SVC) collapsibility

Even though dynamic indices have been shown to be superior to static measures for determining preload responsiveness, currently, there are no data on whether managing patients using dynamic indices affects outcomes.

Central venous oxygen saturation (ScvO2) reflects saturation of superior vena cava blood returning to right atrium. This is obtained from a modified central venous catheter using fiber-optic technology. ScvO2 is a global indicator of tissue oxygenation and reflects the balance between oxygen delivery and consumption. It provides a continuous parameter that evaluates tissue oxygen status. This has been shown to be useful in guiding resuscitation in the early stages of septic shock.

Normal oxygen extraction is 25–30 % corresponding to a ScvO2 >65 %.

<65 % = impaired tissue oxygenation.

Cardiac Output (CO) Monitoring

Direct measurement of CO should be considered when a patient remains hypotensive despite adequate fluid resuscitation or when there is ongoing evidence of global tissue hypoperfusion. An adequate CO is essential to deliver oxygen to peripheral tissues. Low CO reflects hypovolemia or ventricular failure.

There are many CO-monitoring devices available today. The invasive techniques are based on Fick principle and on thermodilution principle of pulmonary artery catheter and remain the gold standard. However being invasive they are associated with some complications.

The noninvasive methods which are available are based on echocardiographic evaluation of left ventricular dimensions during systole. Combination of cross-sectional ECHO and Doppler measurement of flow velocity in the heart and great vessels allows determination of volumetric flow. Patient status dictates the type of CO monitoring required.

Assessment of Cardiac Contractility

Assessing cardiac contractility is important in establishing the etiology of shock and in guiding further therapy.

Cardiac performance may be rapidly assessed at bedside using transthoracic echocardiography (TTE). A visual assessment of left ventricle (LV) function will often reveal any significant abnormality. Estimation of LV contractility can be performed by measuring ejection fraction (EF) (normal >55 %).

Echocardiography is complementary to other monitors of hemodynamics. Positive aspects of echocardiography include noninvasiveness, rapid result, and the potential for comprehensive cardiac assessment. Furthermore, dynamic signs of preload responsiveness, such as IVC collapse, are easily measured and are more informative than filling pressures.

Assessment of Tissue Perfusion

Assessing the adequacy of tissue perfusion has traditionally focused on global parameter of perfusion.

In sepsis, however, tissue hypoperfusion may result from a reduction in perfusion pressure due

to both hypotension and abnormal distribution of flow to the tissues. Regional flow to tissues is regulated by the "microcirculation."

There is now strong evidence that failure of microcirculation plays an important role in end-organ dysfunction and in the pathogenesis of sepsis and septic shock.

The microcirculation can be directly visualized using orthogonal polarization spectral (OPS) imaging and sidestream dark field (SDF) imaging devices. These devices use the principle that polarized green light illuminates tissues to a maximal depth of 3 mm and that scattered light is absorbed by hemoglobin of red cells, contained in superficial vessels. This enables visualization of microcirculation and specifically red blood cell (RBC) transit.

Laser Doppler flowmetry (LDF) is a noninvasive diagnostic method that uses the principle of Doppler flow to detect frequency shifts in laser light illuminating RBCs to measure microvascular blood flow.

Near-infrared spectroscopy (NIRS) uses the principle that different chromophores present in skeletal muscle (such as oxyhemoglobin, deoxyhemoglobin, and myoglobin) have differing absorption properties of light, thus allowing tissue oxygen saturation (StO2) to be derived.

These devices are not currently used in routine clinical practices.

Besides the maternal hemodynamic monitoring, critically ill pregnant women should also have fetal monitoring depending on the gestational age and the clinical scenario.

Special Considerations in Obstetrics

Hemodynamic Monitoring in Severe Preeclampsia

Preeclampsia-eclampsia syndrome is a pregnancy-specific multisystem disorder with certain hemodynamic features which are not seen in normal pregnant women. The blood volume in these cases does not increase as much as in normal pregnancy making them poorly tolerant to even normal blood loss in the postpartum period. These patients do not have the expected decrease in the systemic vascular resistance. Besides, the preeclamptic women have a decreased oxygen delivery and oxygen extraction ratio.

Hemodynamic monitoring may be needed in three subsets of patients with preeclampsia: those with refractory oliguria, pulmonary edema, and refractory hypertension. The hemodynamics are very important to guide the correct management in these cases as discussed below.

(a) *Refractory oliguria*

There are three different hemodynamic subsets in cases of preeclampsia with persistent oliguria.

1. Due to intravascular volume depletion, the PAWP (pulmonary artery wedge pressure) is low and SVR (systemic vascular resistance) is increased with hyperdynamic left ventricular function. The treatment is volume resuscitation.
2. This category has renal arteriospasm causing oliguria. PAWP is increased, SVR is normal, and cardiac output is normal. Treatment in this category is focused on reducing renal vasospasm by low-dose dopamine.
3. In this category, the hemodynamics are consistent with systemic vasoconstriction. There is increase in PAWP and SVR. The cardiac output is decreased. These patients respond to a decrease in afterload and diuresis to improve ventricular function.

(b) *Pulmonary edema*

The preeclamptic patients are at an increased risk of pulmonary edema because of decreased colloid oncotic pressure (COP). The risk of pulmonary edema is highest in the postpartum period when further decrease in the COP occurs. The problem can be compounded if large volumes of crystalloids have been given for resuscitation. Other causes are iatrogenic overload and alterations in pulmonary capillary permeability. Preload measurement should guide the fluid management in such high-risk cases.

(c) *Refractory hypertension*

Pulmonary artery catheter (PAC) placement is useful in the management of patients with refractory hypertension (not responding to hydralazine or labetalol). This will help in determining the cause of increased BP – increase in SVR/CO – and help in guiding the management. CVP monitoring is not useful in monitoring fluid management in severe preeclampsia with left ventricular diastolic dysfunction because left-sided changes are not reflected by the right ventricular filling pressure (measured by CVP). Hence, a bolus of fluid may raise the pulmonary capillary wedge pressure without a change in CVP. Thus, PAC remains a standard ICU monitoring method in such cases.

Cardiovascular Diseases in Pregnancy

Use of a pulmonary catheter is indicated when accurate knowledge of the pressure and cardiac output of the left side is required to guide therapy in patients with complicated mitral or aortic stenosis and pulmonary hypertension and patients with poor functional status. Pulmonary catheter is not usually considered in right outflow obstruction, low pulmonary artery pressure, or right-to-left shunt.

Understanding of physiological changes is very important for management of pregnant women with heart disease. Increased intravascular volume is poorly tolerated in women with mitral regurgitation. In sharp contrast, this physiological alteration which leads to increased preload is beneficial to women with aortic stenosis and helps them to maintain a normal stroke volume and cardiac output. The pulmonary artery wedge pressure (PAWP) in such cases should be kept at a higher than normal value to maintain cardiac output.

The increase in heart rate in pregnancy can compromise the diastolic filling in mitral stenosis and lead to pulmonary congestion. Control of the heart rate with beta-blockers is the first line of management. The PAWP can guide the degree of diuresis in these cases.

The decreased vascular resistance is useful in regurgitant lesions – mitral and aortic regurgitation. In such cases, postpartum period is critical when the vascular resistance increases. However, the decreased vascular resistance will worsen the hemodynamic effects in aortic stenosis and right-to-left shunts.

Hemodynamic Monitoring in Pregnancy

Thus, it is seen that hemodynamic and cardiac monitoring is a very important component of critical care obstetrics and is required to manage obstetric emergencies optimally. Knowledge of physiological changes in pregnancy and the different aspects of hemodynamic monitoring is very important for all obstetricians as any pregnancy can become high risk during the antenatal, peripartum, or the postpartum period.

Basic parameter	Normal range	Critical value	Comments
ECG			Diagnosis of arrhythmias, myocardial ischemia, dyselectrolytemia, embolism
BP	SBP: 100–140 mmHg	>160/110 or <90/60 or a fall in SBP >20 mmHg from baseline	A low BP can compromise tissue perfusion. BP >160/110 mmHg can result in all complications of preeclampsia
	DBP: 60–90 mmHg		
MAP {DP+1/3(SP-DP)}	70–100 mmHg	<60 mmHg	MAP <60 mmHg can compromise oxygen perfusion in vital organs
Oxygen saturation (SO2)	>95 % (on room air)	<90 %	Low SPO2 seen in hypoxemic conditions – APH, PPH, sepsis – is a warning sign of pulmonary edema in preeclampsia
ABG	pH: 7.40–7.46	PaO2<60 mmHg	Low PaO2 reflects hypoxemia as given above. Acidosis in septicemia, embolism, and hypovolemic shock
	PaCO2: 28–32 mmHg	PaCO2>45 mmHg	
	PaO2 > 100 mmHg		
	HCO3: 18–22 meq/L		

Basic parameter	Normal range	Critical value	Comments
Advanced parameter			
CVP (right atrial pressure)	2–6 mmHg		Guides the fluid management in shock
PCWP (pulmonary capillary wedge pressure)	4–12 mmHg		PCWP reflects left atrial and ventricular filling pressure and is more useful for fluid monitoring in MI, valvular disease, sepsis, and severe preeclampsia
CO (cardiac output)	5.5–6 L/min		Measurement is helpful when patient does not respond to fluid resuscitation
Cardiac contractility	EF>55 %		Assessed by transthoracic echocardiography (TTE) for establishing the etiology of shock
Tissue perfusion	Direct visualization of microcirculation in tissues		Compromised in hypovolemia, acute hypertension, and sepsis
			Not used in routine clinical practice at present

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Respiratory Monitoring and Blood Gas Physiology

8

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Respiratory Physiology in Pregnancy

Although pregnancy is described as a normal state, a number of physiologic adaptations occur during pregnancy that optimize fetal growth and take care of the increased maternal requirements. These adaptations include cardiovascular, hematologic, metabolic, renal, and respiratory changes. The levels of progesterone and estrogen rise continually throughout pregnancy and support fetoplacental growth.

Respiratory adaptations are a result of the hormonal and mechanical changes occurring in pregnancy.

The hormonal changes lead to capillary engorgement and swelling of the lining in the nose, oropharynx, larynx, and trachea. This can be exacerbated by fluid overload or edema associated with pregnancy-induced hypertension (PIH) or preeclampsia. Changes in the airway are of special importance as any manipulation of the airway can result in bleeding. Endotracheal intubation is therefore difficult and only a smaller than usual endotracheal tube can be passed through the larynx. Airway resistance is reduced, probably due to the progesterone-mediated relaxation of the bronchial musculature.

Mechanical changes result in decreased lung volumes. Upward displacement by the uterus causes up to 4 cm elevation of the diaphragm. However total lung capacity decreases only slightly because of progesterone-induced relaxation of ligaments causing compensatory increases in the diameters of the chest as well as flaring of the ribs. From the middle of the second trimester, expiratory reserve volume, residual volume, and functional residual volume are progressively decreased, by approximately 20 % at term.

Oxygen consumption increases gradually in response to the needs of the growing fetus, culminating in a rise of at least 20 % at term. During labor, oxygen consumption is further increased (up to and over 60 %) as a result of the exaggerated cardiac and respiratory workload (Table 8.1).

Respiratory Monitoring

Monitoring plays an important role in the management of any patients with respiratory failure. Monitoring is required to detect physiological perturbations early, to understand the underlying pathophysiology of disease, to decide the neces-

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Table 8.1 Respiratory physiological changes in pregnancy

Chest wall/lung mechanics	Physiological changes
Chest wall compliance	Decreased
Thoracic diameter	Increased
Diaphragm	Elevated
Lung compliance	Unchanged
Lung volumes	
Total lung capacity	Unchanged or slightly decreased
Vital capacity	Unchanged or slightly increased
Inspiratory capacity	Slightly increased
Functional residual capacity	Decreased
Residual volume	Slightly decreased
Expiratory reserve volume	Decreased
Spirometry	
FEV1	Unchanged
FVC	Unchanged
FEV1/FVC	Unchanged
Gas exchange	
DLCO	Unchanged or slightly decreased
Ventilation	
Minute ventilation	Increased
Tidal volume	Increased
Respiratory rate	Unchanged
Blood gas	
pH	Normal (7.39–7.42)
PaO2	Slightly elevated (100–105 mmHg)
PaCO2	Slightly decreased (32–34 mmHg)
Bicarbonate	Slightly decreased (15–20 meq/L)

sity and priority of therapeutic interventions, and to evaluate their effectiveness or their complications. Monitoring may be clinical monitoring at the bedside or may involve use of sophisticated monitors. Advanced monitoring techniques cannot substitute human vigilance or clinical judgment.

Clinical Monitoring

At the bedside, the subjective feeling of dyspnea and an increased respiratory rate are clues to a potential respiratory problem. Baseline effort tolerance and the ability to speak a complete sentence without pausing reflect respiratory reserve. The use of accessory muscles like the sternomastoid, flaring of alae nasae, and intercostal and substernal retraction along with signs of sympathetic stimulation like tachycardia and hypertension point to an increased work of breathing. Hypoxia causes cyanosis and agitation while hypercapnia causes drowsiness and asterixis. Bradycardia and hypotension indicate failure of compensation and are late manifestations of respiratory failure (Table 8.2).

Table 8.2 Clinical monitoring

Dyspnea
Inability to speak a complete sentence without a pause
Tachypnea
Use of accessory muscles
Hypertension and tachycardia
Cyanosis and agitation (hypoxia)
Drowsiness and asterixis (hypercapnia)
Bradycardia and hypotension (late manifestations)

Measurement of Gas Exchange

Pulse Oximetry

Clinical presentation of hypoxemia in the form of cyanosis is often late and nonspecific. Pulse oximeters measure the saturation of hemoglobin in the tissue real time and with around 95 % sensitivity.

Mechanism

Pulse oximeters distinguish between oxyhemoglobin and reduced hemoglobin on the basis of differential absorption of light at 660 and 940 nm wavelengths. Oxyhemoglobin absorbs much less red (±660 nm) and slightly more infrared (±910 to 940 nm) light than nonoxygenated hemoglobin. Ratio of oxyhemoglobin to nonoxygenated hemoglobin thereby determines the ratio of red to infrared absorption. When red and infrared light are directed from light-emitting diodes

(LEDs) to a photodetector across a pulsatile tissue bed, the absorption of each wavelength by the tissue bed varies cyclically with pulse. During diastole, absorption is due to the nonvascular tissue components (e.g., bone, muscle, and interstitium) and venous blood. During systole, absorption is determined by all of these components and arterialized blood. Thus, the difference between absorption in systole and diastole is in theory due to the presence of arterialized blood. The changes in ratio of absorption between systole and diastole can then be used to calculate an estimate of arterial oxygen saturation. Most oximeters under ideal pulse circumstances measure the saturation indicated by the pulse oximeter (SpO_2) to within 2 % of arterial oxygen saturation.

Cooximetry is performed on whole blood sample obtained from an artery or a vein. It measures absorbance at multiple wavelengths and computes the percentage of oxyhemoglobin, deoxyhemoglobin, methemoglobin, and carboxyhemoglobin (COHb) in total hemoglobin based on different absorption spectra. Cooximetry is considered as the gold standard method of assessing saturation.

Indications for Pulse Oximetry

The Society of Critical Care Medicine considers pulse oximetry (or transcutaneous oxygen measurement) essential monitoring for all ICU patients with an aim of identifying hypoxemia early in critically ill patients and also to titrate supplemental oxygen. Pulse oximeter monitoring is also important during peri-anesthetic period and also during all airway interventions.

Problems with Pulse Oximetry

Accuracy of pulse oximetry will be affected in cases of severe hypoxemia, dyshemoglobinemia, low perfusion states, skin pigmentation, and hyperbilirubinemia. Majority of problems are due to factors that interfere with the absorption of light between the LEDs and photodetector.

Technical Problem Related to Calibration

Pulse oximeters are calibrated for normal volunteers. Hence, most oximeters will give less precise reading in the saturation range of less than 70 %. Thus, oximeters should be considered unreliable when SpO_2 is less than 70 %.

Measurement Sites

Usual sites are finger and toe probes. High ambient light, motion, and perfusion artifacts are likely to affect finger or toe measurements. The earlobe is believed to be the site least affected by vasoconstriction. Long fingernails may prevent correct positioning of the finger pulp over the LEDs used in inflexible probes and therefore produce inaccurate SpO_2 reading without affecting the pulse rate. Adhesive tapes in disposable probes placed over both sides of a finger did not affect measured SpO_2. Since pulse oximetry depends fundamentally on color, artificial nails and nail polish may falsely lower SpO_2.

Motion Artifact

Shivering and other movements result in artifact. The display of a plethysmographic waveform will be distorted affecting SpO_2 reading. Discrepancies in pulse rate as compared to ECG will give a clue.

Ambient Light

Ambient light that affects absorption in the 660 or 910 nm wavelengths, or both, may affect calculation of saturation and pulse. Surgical lights and fiber optic light sources will interfere with saturation. Interference from surrounding lights should be suspected by the presence of pulse values discordant from the palpable pulse or ECG, or changes in the pulse-saturation display when the probe is transiently shielded from ambient light with an opaque object. Newer generations

of pulse oximeters however have little or no effect of ambient light.

Hypoperfusion

Poor peripheral perfusion and cold extremities affect reliability of SpO_2 readings.

Hyperbilirubinemia

Standard pulse oximeters are not affected by hyperbilirubinemia.

Dyshemoglobinemias

Standard pulse oximeters with two wavelength diode systems cannot detect the presence of methemoglobin, COHb, or fetal hemoglobin. Fetal hemoglobin may confound readings in neonates but is rarely a problem in adult. Acquired methemoglobinemia in adults is seen with drugs like topical anesthetics, dapsone. Because methemoglobin absorbs more light at 660 nm than at 940 nm, it affects pulse oximetry reading. Higher levels of methemoglobin will have fixed SpO_2 reading toward 84–90 %. Carboxyhemoglobin (COHb) is seen with smoke inhalation or potential carbon monoxide poisoning. COHb will also have a fixed reading by two-diode oximeter as 90 % oxyhemoglobin and 10 % reduced hemoglobin resulting in false elevation of SpO_2. A gap between pulse oximetry and Po_2 or cooximetrically measured oxygen saturation may suggest dyshemoglobinemia.

Lipids

Patients with elevated chylomicrons and those receiving lipid infusions may have falsely low SpO_2 because of interference in absorption by the lipid.

Hypothermia

Hypothermia-induced vasoconstriction may affect quality of signal. Look for discordance in pulse values from SpO_2 from pulse oximeter with pulse values from ECG monitor.

Practical Implications

Oxygen saturation is one of the important determinants of oxygen delivery to the tissues along with hemoglobin content and the cardiac output. Importantly it is SpO_2 and not the PaO_2 that is the main determinant of the oxygen content of blood and hence oxygen delivery to tissues.

The relationship between SpO_2 and PaO_2 is described by the oxyhemoglobin dissociation curve which is not linear. A saturation of 90 % is a critical threshold because below this level even a small fall in PaO_2 produces a sharp fall in SpO_2.

Also, common misperception with regard to pulse oximetry is that normal SpO_2 rules out respiratory problem. In contrast, a patient with obstructed airway can maintain SpO_2 especially with supplemental oxygen. While on supplemental oxygen, even a small amount of ventilation will maintain oxygenation, but the CO_2 will continue to rise to potentially dangerous levels.

Always watch for an acceptable plethysmographic waveform. Only in the presence of proper waveform that SpO_2 data can be interpreted. Also, the reliability of pulse oximetry is reduced in states of severe hypoxia. Pulse oximetry tends to be unreliable with SpO_2 below 70 %.

Capnography

Capnography measures and displays expired PCO_2 concentration as an absolute value and waveforms. The factors that will affect expired CO_2 readings can be classified as follows:

Factors that can affect CO_2 production include substrate metabolism, drug therapy, and core temperature.

Factors affecting CO_2 transport include cardiac output and pulmonary perfusion.

Factors which can affect ventilation include obstructive and restrictive diseases, respiratory rate, and ventilation-perfusion ratio.

Mechanism

Expired PCO_2 concentration is usually determined by infrared absorbance or mass spectrometry. Conventional bedside capnographs use the infrared technique. Carbon dioxide has a characteristic absorbance of infrared light with maximal absorbance seen near a wavelength of 4.28 mm. A heated wire with optical filters is used to generate an infrared light of appropriate wavelength. When carbon dioxide passes between a focused beam of light and semiconductor photodetector, an electronic signal can be generated that, when calibrated, accurately reflects the PCO_2 of the tested gas.

The other technique is the mass spectrometer which can detect the partial pressure of several gases simultaneously. It is often used in operating rooms for evaluation of other inhalational gases as well.

Gases can be sampled by mainstream or sidestream technique. Mainstream sampling involves placing the capnometer directly in line in the patient's respiratory circuit. All air leaving the patient passes through the capnometer. The sidestream sampling techniques pump expired air through thin tubing to an adjacent analyzing chamber.

The mainstream sampling offers instantaneous analysis of sampled air, but it increases the patient's dead space and also adds weight to the endotracheal tube and breathing circuit. Sidestream sampling removes air from the expiratory circuit, altering measurement of tidal volume. Sampling errors will affect CO_2 estimations.

Indications

Capnography allows for the continuous verification of endotracheal tube placement, which is essential in all intubated patients especially during transport as accidental extubation is an ongoing risk in such patients.

Continuous capnography during cardiac arrest is useful to monitor intubation, ventilation, and also return of spontaneous circulation. A rapid increase in end-tidal CO_2 ($ETCO_2$) concentration during CPR often represents return of spontaneous circulation (ROSC) and can be a useful guide in determining timing of rhythm and pulse checks.

Continuous noninvasive $ETCO_2$ monitoring can be useful in the monitoring of patients with tenuous respiratory status, such as those with severe reactive airway disease or congestive heart failure (Table 8.3).

Practical Implications

Under normal physiologic conditions, the difference between arterial PCO2 (from ABG) and alveolar PCO2 (ETCO2 from capnograph) is 2–5 mmHg. This difference is termed the PaCO2-PETCO2 gradient or the a-ADCO2 and can be increased in the following conditions:

- COPD (causing incomplete alveolar emptying)
- ARDS (causing V/Q mismatch)
- A leak in the sampling system or around the ET tube

Table 8.3 Conditions with altered end-tidal CO2 concentrations

Increased ETCO2	Decreased ETCO2
Increased muscular activity (shivering)	Decreased muscular activity (muscle relaxants)
Malignant hyperthermia	Hypothermia
Increased cardiac output (during resuscitation)	Decreased cardiac output
Bicarbonate infusion	Pulmonary embolism
Tourniquet release	Bronchospasm
Effective drug therapy for bronchospasm	Increased minute ventilation
Decreased minute ventilation	

Thus while ETCO2 may reflect PaCO2 in hemodynamically stable healthy volunteers, in patients who are unstable or who have abnormal pulmonary function, it is unreliable. For example, monitoring of the decompensated COPD patient for hypercapnia by ETCO2 alone may lead to overlooking of significant clinical deterioration (i.e., an ETCO2 of 30 mmHg may actually reflect a PaCO2 of 95 mmHg due to significant pulmonary shunting).

Acid-Base Physiology and Common Acid-Base Disorders

Acid is being continually produced in the body through metabolic processes. Despite this large addition of acids to the body, the pH is maintained within a narrow range {7.35–7.45}. This is necessary because protons are so extraordinarily reactive that even minute changes can drastically alter physiologic processes. Immediate defense of pH is provided by buffers which include HCO3, phosphates, and protein buffers. Regulation of pH ultimately depends on lungs and the kidneys. The role of the lungs is to eliminate CO2 which is the principal end product of acid metabolism in response to changes in blood pH. The role of the kidneys is to retain HCO3 and eliminate protons of nonvolatile acids.

Basic Concepts

The table below depicts an arterial blood gas (ABG) as seen most often in the medical record with the parameters most often evaluated with their normal values (Table 8.4).

Table 8.4 Commonly evaluated ABG parameters

Parameter	Explanation	Value (range)
pH	Arterial blood pH	7.40 (7.35–7.45)
PaCO2	Arterial CO2 pressure, mmHg	40 (35–45)
PaO2	Arterial O2 pressure, mmHg	98 (80–100)
HCO3	Serum bicarbonate concentration, mEq/L	24 (22–26)
O2 sat. %	Arterial oxygen saturation	(95–100 %)

Hypoxia and Hypoxemia

Hypoxia refers to reduced oxygen pressure in the alveolus. Hypoxemia refers to low arterial PaO2. At or near sea level, the following equation will estimate the average value for arterial PaO2:

$$\mathrm{PaO2} = 104.2 - (0.27 \times \mathrm{age})$$

In the alveolus, a reciprocal relationship exists between oxygen and carbon dioxide. Carbon dioxide accumulation as a result of inadequate ventilation displaces oxygen. Also, a normal gradient of about 10 mmHg exists between alveolar oxygen pressures (PAO2) and the arterial oxygen pressure (PaO2). Generally, there is no gradient between the alveolar carbon dioxide pressure (PACO2) and the arterial carbon dioxide pressure (PaCO2). The PaCO2 and PaO2 values will reflect these relationships. The reciprocal relationship between the PAO2 and PACO2 is illustrated by the alveolar gas equation shown below:

$$\begin{aligned}\mathrm{PAO2} &= \mathrm{FiO2}(\mathrm{Atm.\ Pr.} \quad \mathrm{water\ vapor\ pr.}) \\ &\quad -(\mathrm{PaCO2}/0.8) \\ \mathrm{PAO2} &= 0.21(760 \quad 47) - (\mathrm{PaCO2} \times 1.25) \\ \mathrm{PAO2} &= 150 \quad 1.25 \times \mathrm{PaCO2}\end{aligned}$$

FiO2 – fraction of inspired oxygen which is 0.21 for ambient air.

The correction factor for PCO2 is derived by dividing by *R* (respiratory quotient) which is estimated to be 0.8.

Atmospheric pressure – atmospheric pressure which is 760 mmHg at sea level.

Water vapor pressure – water vapor pressure which is 47 mmHg.

The PACO2 is equivalent to PaCO2 because there is no gradient. The PAO2 to PaO2 gradient is normally close to 10 mmHg (up to 21 in older individuals) and is about

$$P(A-a)\mathrm{O2} = 2.5 + (0.21 \times \mathrm{Age})\mathrm{mmHg}$$

An elevated gradient signifies hypoxia due to a problem with gas exchange or ventilation-perfusion mismatch. A normal gradient usually indicates hypoxia due to a decreased PAO2 due to decreased atmospheric O2 due to high altitude. A normal gradient with hypoxia may also be seen in cases of hypercarbia or elevated PaCO2, due to impaired ventilation. Impaired ventilation results in respiratory acidosis that is discussed in greater detail in the next section. The following table illustrates the effect of increased PACO2 in a patient with a normal $P\ (A-a)$ O2 gradient of 10 mmHg (Table 8.5).

PaO2/FiO2 Ratio

In hospitalized patients PaO2 values need to be interpreted in concert with the FiO2 the patient is receiving. The Berlin definition of acute respiratory distress syndrome (ARDS) classifies patients as mild, moderate, or severe based primarily on the PaO2/FiO2 ratio. PaO2/FiO2 ratio more than 300 is considered as normal (Table 8.6).

Relationship between PaO2 and O2 Saturation %

The relationship between PaO2 and O2 saturation is governed by the oxyhemoglobin dissociation curve which is typically an S-shaped curve. The table below depicts the PaO2 and O2 saturation relationship in normal and hypoxic patients (Table 8.7).

Table 8.5 Effect of PACO2 on PaO2

PACO2 (= PaCO2)	PAO2	PaO2
40	97	87
64	67	57
80	47	37

Table 8.6 ARDS severity according to PaO2/FiO2 ratio

	PaO2/FiO2 ratio
Normal	>300
Mild ARDS	200–300
Moderate ARDS	100–199
Severe ARDS	<100

Table 8.7 Relationship between PaO2 and O2 saturation %

	PaO2	O2 saturation
Normal	80–100 mmHg	95–100 %
Mild hypoxia	60–79 mmHg	90–95 %
Moderate hypoxia	40–59 mmHg	75–89 %
Severe hypoxia	<40 mmHg	<75 %

O2 Saturation Gap

The O2 saturation measured by the pulse oximeter is directly obtained from the finger or ear probe, whereas the O2 saturation on the ABG report is usually calculated from the PaO2 of the arterial blood gas sample. A discrepancy in these two values may result if inadvertently a venous ABG sample has been collected. In such a case, the pulse oximeter reading will be normal while the ABG sample will reflect venous oxygen saturation. In case the pulse oximeter reading is low with a normal peripheral circulation and PaO2 and O2 saturation on ABG are low, one should suspect possible circulating abnormal hemoglobin like methemoglobin or carboxyhemoglobin. Performance of cooximetry in such cases usually identifies the problem and also the circulating abnormal hemoglobin.

Acidemia and Alkalemia

Acidemia and alkalemia refer to the alterations in the blood pH. Both respiratory and metabolic disorders can contribute to alterations in pH and are referred to as a respiratory acidosis or respiratory alkalosis and a metabolic acidosis or metabolic alkalosis (see Table 8.8). A single disorder may account for the observed acidemia or alkalemia, but often more than one disorder occur concurrently. These are referred to as mixed or complex acid-base disorders. For example, an alkalemic ABG may exhibit a mixed respiratory acidosis and a metabolic alkalosis. Identifying the simple

Table 8.8 Identifying primary disorder

Parameter	Metabolic acidosis	Metabolic alkalosis	Resp. acidosis	Resp. alkalosis
pH	Decreased	Increased	Decreased	Increased
pCO2	Decreased	Increased	Increased	Decreased
HCO3	Decreased	Increased	Increased	Decreased

as well as the complex acid-base disorders will be possible by applying the stepwise approach outlined in the next section.

Stepwise Approach to Diagnosing Acid-Base Disorders

The following is a seven-step logical approach to analyzing acid-base disorders utilizing the ABG and serum electrolyte data. It was originally proposed by Narins and Emmett and further refined by Morganroth.

Step 1: *Acidemic or alkalemic*?

The pH of the arterial blood gas measurement identifies the disorder as alkalemic or acidemic.

Normal arterial blood pH = 7.40 ± 0.05

Acidemic: pH < 7.35

Alkalemic: pH > 7.45

Step 2: *Is the primary disturbance respiratory or metabolic*?

This step requires one to determine whether the disturbance affects primarily the arterial PaCO2 or the serum HCO3.

- A respiratory disturbance alters the arterial PaCO2 (normal value 40, range 35–45). Go to step 3.
- A metabolic disturbance alters the serum HCO3– (normal value 24, range 20–28).
- If HCO3 <20, metabolic acidosis is present. Go to step 5.
- If HCO3– >28, metabolic alkalosis is present, is respiratory compensation adequate? Go to step 5.

The Henderson-Hasselbalch equation provides the basis for the relationship between the blood pH and PaCO2, HCO3–, and it is shown below. The calculation, however, has no practical value:

$$\mathrm{pH} = \mathrm{pK} + \log\left[\mathrm{HCO3-/PaCO2}\right] \times \mathrm{K}, \text{ or } \left[\mathrm{H+}\right] = 24 \times \mathrm{PaCO2/HCO3-}$$

Step 3: *For a respiratory disturbance*, *determine whether it is acute or chronic*.

A respiratory acidosis results from accumulation of PaCO2 and a respiratory alkalosis results from hyperventilation or a low PaCO2 (specific causes of respiratory acidosis and alkalosis are listed subsequently). For acute disturbances a PaCO2 variation from normal by 10 mmHg is accompanied by a pH shift of approx. 0.08 units. A chronic disturbance reflects renal mediated HCO3– shifts. Renal compensation requires several hours to develop and is maximal after 4 days. Therefore during chronic disturbances, a PaCO2 variation from normal of 10 is accompanied by a smaller pH shift of only 0.03 units. Also, the renal correction brings the pH back toward normal but not completely. These relationships are spelled out in the following equations:

Acute respiratory acidosis :

$$\text{pH decrease} = 0.08 \times \left(\mathrm{PaCO2} - 40\right)/10$$

Chronic respiratory acidosis :

$$\text{pH decrease} = 0.03 \times \left(\mathrm{PaCO2} - 40\right)/10$$

Acute respiratory alkalosis :

$$\text{pH increase} = 0.08 \times \left(40 - \mathrm{PaCO2}\right)/10$$

Chronic respiratory alkalosis

$$\text{pH increase} = 0.03 \times \left(40 - \mathrm{PaCO2}\right)/10$$

Step 4: *For a respiratory disorder*, *is compensation okay*?

Knowledge of the empirical limits of compensation is essential to evaluate if the change in HCO3– is appropriate for the change in PCO2. The limits of compensation are dictated not

only by the nature of the disorder (respiratory acidosis vs. alkalosis) but also by the duration of the disorder (acute vs. chronic). Thus the upper limits for compensation are

$$\text{Respiratory acidosis}:$$
$$< 24\,\text{h} : \Delta[HCO_3] = 1/10\ \Delta\ PCO_2$$
$$> 24\,\text{h} : \Delta[HCO_3] = 3/10\ \Delta\ PCO_2$$

$$\text{Respiratory alkalosis:}$$
$$1-2\,\text{h} : \Delta[HCO_3] = 2/10\ \Delta\ PCO_2$$
$$> 2\ \text{days} : \Delta[HCO_3] = 6/10\ \Delta\ PCO_2$$

Step 5: *Assess the normal compensation by the respiratory system for a metabolic disturbance*.

The respiratory system responds quickly to a metabolic disturbance and most predictably to a metabolic acidosis. The change in PaCO2 exhibits a linear correlation with the change in HCO3–. The equation that predicts the respiratory response to a metabolic acidosis is called Winter's formula:

$$\text{Expected PaCO2} = (1.5 \times \text{HCO3}-) + (8 \pm 2)$$

In the setting of a simple metabolic acidosis, the measured PaCO2 will fall within the range predicted by Winter's formula. If a respiratory disturbance is occurring concurrently with the metabolic acidosis, it would be defined by the direction the PaCO2 varies outside the range predicted by Winter's formula, not by the PaCO2 variation from the normal value of 40.

Working through the following example illustrates how to utilize Winter's formula to assess the respiratory response to metabolic acidosis. If the serum HCO3– is 10 mEq/L, the PaCO2 should be between 21 and 25 according to Winter's formula. If the measured PaCO2 falls outside of this range, then an additional respiratory disturbance must be occurring concurrently. If the measured PaCO2 is less than 21, then the additional disturbance is a respiratory alkalosis. If the measured PaCO2 is greater than 25, then the additional disturbance is a respiratory acidosis.

Winter's formula does not predict the respiratory response to a metabolic alkalosis. The magnitude of respiratory response to metabolic alkalosis is not easily predictable. When present, the respiratory response to metabolic alkalosis is hypoventilation, but the degree of PaCO2 increase does not exhibit a linear relationship with the HCO3–. Two general rules hold up for the respiratory response to a metabolic alkalosis:

- A patient will increase PaCO2 above 40 but not greater than 50–55 to compensate for a metabolic alkalosis.
- A patient will be alkalemic (pH >7.42) if the PaCO2 is elevated to compensate for a metabolic alkalosis. (If the patient is acidemic, pH <7.38, then an additional respiratory acidosis is present.)

Step 6: *For a metabolic acidosis*, *determine whether an anion gap is present*.

The anion gap calculation simplifies the diagnosis of the cause for a metabolic acidosis. What is the anion gap? The normal anion gap is 12 mEq/L. The anion gap is the calculated difference between negatively charged (anion) and positively charged (cation) electrolytes, which are measured in routine serum assays. The total of measured cations represented by sodium (Na+) is greater than the total measured anions, HCO3– and chloride (Cl-). Turned around, that difference or gap also can be viewed as the unmeasured anion concentration. The unmeasured anion concentration dominates the balance between the unmeasured serum anions and cations as illustrated in Table 8.9.

Thus the balance favors the unmeasured anions by 12 mEq/L, which is the normal anion gap. The unmeasured anions rarely change enough to effect anion gap interpretation. Knowledge

Table 8.9 Anion gap reflects the unmeasured anions and cations

Unmeasured anions	Unmeasured cations
Proteins, mostly albumin 15 mEq/L	Calcium 5 mEq/L
Organic acids 5 mEq/L	Potassium 4.5 mEq/L
Phosphates 2 mEq/L	Magnesium 1.5 mEq/L
Sulfates 1 mEq/L	
Total: 23 mEq/L	11 mEq/L

of the unmeasured anions is not essential to the calculation of the anion gap. However, one needs to understand the concept in order to recognize the rare instances when the anion gap is not 12 for reasons other than a metabolic acidosis. These exceptions are listed at the end of this section.

The causes of an anion gap acidosis differ from those of a normal or non-anion gap acidosis. The anion gap determination is an excellent tool for narrowing the list of potential causes of a metabolic acidosis. The simple calculation is shown below.

The anion gap calculation requires values for the serum Na+, Cl-, and HCO3–:

$$\text{Anion gap} = \text{Na}+ - \left(\text{Cl}- + \text{HCO3}-\right),$$

$$\text{Anion gap metabolic acidosis, anion gap} > 12$$

$$\text{Normal or non-anion gap acidosis, anion gap} < 12$$

The calculation of the anion gap provides reliable data with the following rare exceptions. Patients with a low serum albumin (e.g., cirrhosis, nephrotic syndrome, malnutrition) have an anion gap acidosis, but the measured anion gap is normal or <12. The reason is that albumin has many negative charges on its surface and accounts for a significant proportion of the unmeasured anions. Severe hypoalbuminemia may exhibit a normal anion gap as low as four. Therefore in severe hypoalbuminemia if the anion gap increases and approaches 12, one must suspect an additional metabolic cause for the increased anion gap.

In *alkalemic patients* with pH >7.5, the anion gap may be elevated due to metabolic alkalosis and not because of additional metabolic acidosis. This is probably due to unmeasured anion accumulation. Specifically, the negative charges on the surface of albumin become more negative in alkalemic conditions which would increase the unmeasured anions and the anion gap. The distinction between whether an anion gap is due to alkalemia or an underlying acidosis in an alkalemic patient needs to be considered in some clinical situations.

Step 7: *Determine whether other metabolic disturbances coexist with an anion gap acidosis*.

A non-anion gap acidosis or a metabolic alkalosis may exist concurrently with an anion gap acidosis. This determination requires one to account for the increase in the anion gap and determine whether additional variation in HCO3– exists. If no other metabolic disturbance exists, then the following calculation would result in 24:

$$\text{Corrected HCO3}- = \text{measured HCO3} - + \left(\text{anion gap} - 12\right)$$

If the corrected HCO3– varies significantly above or below 24, then a mixed or more complex metabolic disturbance exists. To be more specific, if the corrected HCO3– is greater than 24, a metabolic alkalosis coexists. If the corrected HCO3– is less than 24, then a non-gap acidosis coexists.

The following examples help one understand how this step works. A patient with an anion gap metabolic acidosis has a HCO3– of 10 mEq/L and an anion gap of 26. By calculating the corrected HCO3–, one finds the result to be 24 and can conclude that no other metabolic disturbance coexists. If this patient had a HCO3– of 15 and an anion gap of 26, then the corrected HCO3– would be calculated to 29, a value significantly greater than 24. One would then conclude that metabolic alkalosis coexists with the gap acidosis.

Specific Acid-Base Disorders

Respiratory Acidosis

Respiratory acidosis results from hypoventilation which is manifested by the accumulation of CO2 in the blood and a drop in blood pH. Examples of specific causes can be categorized as follows:

Central nervous system depression (sedatives, CNS disease, obesity)

Hypoventilation syndrome

Pleural disease (pneumothorax)

Lung disease (COPD, pneumonia)
Musculoskeletal disorders (kyphoscoliosis, Guillain-Barre syndrome, myasthenia gravis, polio)

Respiratory Alkalosis

Respiratory alkalosis results from hyperventilation which is manifested by excess elimination of CO2 from the blood and a rise in the blood pH. Examples of specific causes are listed below:

Catastrophic CNS event (CNS hemorrhage)
Drugs (salicylates, progesterone)
Pregnancy (especially the third trimester)
Decreased lung compliance (interstitial lung disease)
Liver cirrhosis
Anxiety

Anion Gap Metabolic Acidosis

Anion gap acidosis results from accumulation of acidic metabolites and is manifested by a low HCO3– and an anion gap >12 (anion gap calculation discussed in step 6). Examples of specific causes:

Lactic acidosis (sepsis, left ventricular failure)
Ketoacidosis (diabetic hyperglycemia, EtOH withdrawal)
Uremia
Alcohol poisons or drug intoxication (methanol, ethylene glycol, paraldehyde, salicylates)

One may use a mnemonic device to remember these items. MULEPAK is a mnemonic commonly used (methanol, uremia, lactic acidosis, ethylene glycol intoxication, paraldehyde intoxication, aspirin, ketoacidosis) (Also KUSMAL and MUDPIES).

Non-anion Gap Metabolic Acidosis

Non-anion gap acidosis results from loss of bicarbonate or external acid infusion and is manifested by a low HCO3–, but the anion gap is <12 (anion gap calculation is discussed in step 6). Examples of specific causes:

GI loss of HCO3– (diarrhea)
Renal loss of HCO3–
Compensation for respiratory alkalosis
Carbonic anhydrase inhibitor (Diamox)
Renal tubular acidosis
Ureteral diversion
Other causes: HCl or NH4Cl infusion, Cl gas inhalation, hyperalimentation

A mnemonic device may be used to remember this list of causes. The commonly used mnemonic is ACCRUED (acid infusion, compensation for respiratory alkalosis, carbonic anhydrase inhibitor, renal tubular acidosis, ureteral diversion, "extra" alimentation or hyperalimentation, diarrhea).

Metabolic Alkalosis

Metabolic alkalosis results from elevation of serum bicarbonate. Examples of specific causes:

Volume contraction (vomiting, overdiuresis, ascites)
Hypokalemia
Alkali ingestion (bicarbonate)
Excess gluco- or mineralocorticoids
Bartter's syndrome

Is the Traditional Approach to ABG Analysis Adequate?

The traditional approach at the descriptive and practical level allows us to diagnose and treat most acid-base disorders, but some disorders like saline acidosis are difficult to explain using the traditional approach described above. The identification of pathophysiological mechanisms requires calculation of anion gap and correction of anion gap for albumin and phosphate or calculation of standard base excess and correction of standard base excess for albumin and phosphate. An alternative approach called the Stewart approach has been described that is based on

quantitative acid-base chemistry, the law of electrical neutrality, and the law of conservation of mass. The Stewart approach is elegant, explains acid-base physiology in a lucid manner, and allows identification of mechanisms directly. It is however more cumbersome and involves a lot of calculations. A detailed description is beyond the scope of the present chapter and can be obtained from the reference below.

Suggested Reading

Dr. Kellum's and Dr. Elber's site on Stewart Approach. www.acid-base.org.

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Obstetric Monitoring in Critically Ill Pregnant Women

9

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Admission of the pregnant or postpartum women to the intensive care unit is uncommon but may require specialized knowledge for successful management [1].

Assessment and Management may be affected by

1. Physiological change associated with pregnancy
2. Pregnancy-specific conditions
3. Presence of a fetus
4. Clinicians' lack of familiarity

Pregnancy-specific conditions

- Preeclampsia
- Eclampsia
- Tocolytic pulmonary edema
- Peripartum cardiomyopathy
- Amniotic fluid embolism
- Obstetric hemorrhage

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Assessment of Fetus [2]

- NST
- Biophysical profile
- USS
- Doppler
- Fetal pH

NST

- NST is primarily a test of fetal condition. It describes fetal heart rate acceleration in response to fetal movement perceived by the mother.
- NST is the most widely used primary testing method for assessment of fetal well-being and has also been incorporated into the biophysical profile testing system.
- The NICHD fetal monitoring workshop (1997) has defined acceleration based on gestational age.

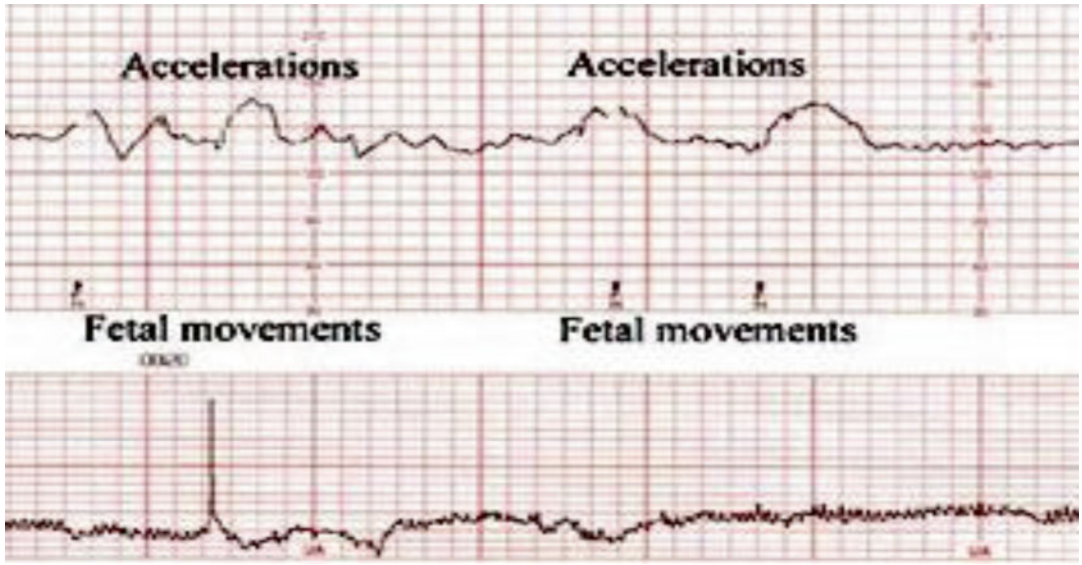
Accelerations >32 weeks

- ≥15 bpm above baseline for ≥15 s, but less than 2 min, and all occurring within 20 min of beginning the test.

Accelerations <32 weeks

- ≥10 bpm above baseline for ≥10 s, lasting less than 2 min.
- The tracing of a premature fetus, especially before 28 weeks, typically will have a higher fetal heart rate baseline and exhibit less variability and lower-amplitude accelerations than a fetus over 32 weeks.

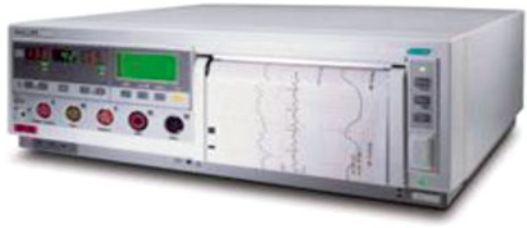
A. Gandhi et al. (eds.), *Principles of Critical Care in Obstetrics: Volume I*,
DOI 10.1007/978-81-322-2692-5_9



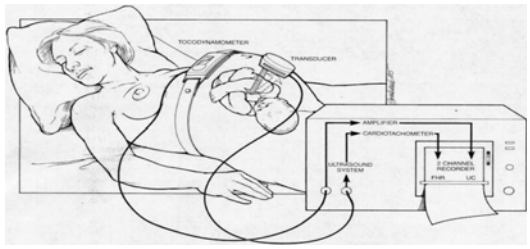
Abnormal NST

- Baseline oscillation of less than 5 bpm
- Absent accelerations as well as beat-to-beat variability
- Late decelerations with spontaneous uterine contractions

Electronic Fetal Monitor



External Fetal Monitoring



Acoustic stimulation test

- An artificial larynx is positioned on the maternal abdomen and a stimulus of 1–2 s is applied. This may be repeated up to three times for up to 3 s.
- Response to vibroacoustic stimulation is considered normal if FHR acceleration of at least 15 bpm for at least 15 s occurs within 15 s after the stimulation with prolonged fetal movements.

Biophysical Profile

- It is a more accurate means of assessing fetal health by real-time ultrasound device. It requires 30–60 min.

Components and Score for BPP

Component	Score 2	Score 0
Nonstress test	2 accel of 15 beats/min for 15 s in 20–40 min	0 or 1 accel in 20–40 min
Fetal breathing	1 episode of rhythmic breathing lasting 30 s within 30 min	<30 s of breath in 30 min

Component	Score 2	Score 0
Fetal movement	3 discrete body or limb movements within 30 min	2 mov in 30 min
Fetal tone	1 episode of extension of a fetal extremity with return to flexion, or opening or closing of hand	No movement or no ext/ flexion
Amnionic fluid volume	Single vertical pocket >2 cm	Largest single vertical pocket = 2 cm

BPS	Interpretation	Management
10	Normal	No intervention; repeat test weekly except in diabetic and post term preg
8/10 normal fluid	Normal	No intervention; repeat testing per protocol
8/10 decreased	Chronic fetal	Deliver
	Asphyxia suspected	
6	Possible fetal asphyxia	If amnionic fluid volume is abnormal, deliver
		If normal fluid is at > 36 weeks with favorable cervix, deliver
If repeat test ≤6, deliver		
If repeat test >6, observe		
Repeat as protocol		
4	Probable fetal asphyxia	Repeat testing same day; if biophysical profile score ≤6, deliver
0–2	Almost certain	Deliver fetal asphyxia

AFI by USG

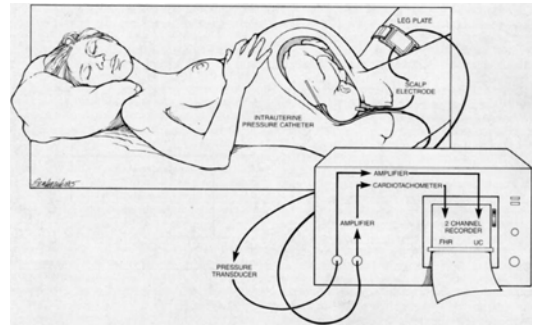
- Assessment of amnionic fluid has become an integral component in the antepartum assessment of pregnancies at risk for fetal death.
- Based on the rationale that decreased uteroplacental perfusion may lead to decreased fetal renal blood flow, decreased micturition, and, ultimately, oligohydramnios.
- AFI of 5 cm or less was associated with significantly increased perinatal morbidity and mortality.

Doppler

- Noninvasive technique to assess blood flow by characterizing downstream impedance.
- The umbilical artery S/D ratio, the most commonly used index, is considered abnormal if it is elevated above the 95th percentile for gestational age or if diastolic flow is either absent or reversed.

Absent or reversed end-diastolic flow signifies increased impedance, and it is uniquely associated with fetal growth restriction.

Internal Fetal Monitoring



- There are two types of internal monitors – a fetal scalp electrode and an intrauterine pressure catheter (IUPC). They are only used once the patient's "water has broken" and her cervix has dilated enough.
- A fetal scalp electrode is a device that monitors the baby's heart rate. It consists of a small clip that is placed on the baby's scalp.

Intrauterine pressure catheter (*IUPC*) is a small catheter that is placed up into the patient's uterus. It goes between the uterine wall and the baby. It directly measures the strength of her

contractions and resting tone in millimeters of Hg. It provides more accurate information as to the strength of contractions than an external monitor (tocodynamometer).

Interpreting FHR

- Identify fetal heart rate baseline.
- Identify existence and degree of variability (absent, minimal, moderate, or marked).
- Determine whether there are accelerations or decelerations.
- Pattern of uterine contractions.
- Correlate accelerations and decelerations with uterine contractions and identify the pattern.
- Determine whether the FHR recording is reassuring, nonreassuring, or ominous.

Fetal scalp blood sampling

- Measurements of the pH in capillary scalp blood help.
- An illuminated endoscope is inserted through the dilated cervix after ruptured membrane. The skin is wiped clean with a cotton swab and coated with a silicone gel to cause the blood to accumulate as discrete globules. An incision is made through the skin to a 2 mm depth with a special blade on a long handle.
- pH> 7.25 labor is observed.
- pH is between 7.20 and 7.25 pH repeated within 30 min.
- pH< 7.20, another scalp blood sample, is collected immediately and the mother is taken for surgery.

Fetal pulse oximetry

- Allow assessment of fetal oxyhemoglobin saturation once the membrane ruptured.
- A unique padlike sensor is inserted through the cervix and positioned against the fetal face where it is held in place by the uterine wall.
- Fetal oxygen saturation normally varies between 30 and 70 % throughout labor.
- Saturation values <30 %, however, when persistent for 2 min or longer; were associated with fetal compromise.

Fetal ECG

- As fetal hypoxia worsens, there are changes in the ST segment and PR interval of the fetal ECG.

Decision making around delivery in the critically ill

- Decision making will be influenced by viability of fetus.
- Is there maternal benefit to delivery?
- What are the risks of delivery to mother and fetus?
- What method of delivery?
- How to manage delivery, IOL in presence of coagulopathy.
- When to deliver the fetus?

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Fetal Surveillance in Critically Ill Obstetric Patient

10

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Introduction

Assessment of fetal well-being is important not only for high-risk patients but also for other pregnant women who might develop unexpected complications in the course of otherwise normal pregnancies.

A wide range of tests of fetal well-being have been introduced during the last 30 years.

Both biochemical tests (which monitor the endocrine function of the placenta or the fetoplacental unit) and biophysical methods of monitoring (which provide different information about fetal growth and physiological function) have the theoretical ability to detect changes in fetal well-being that may occur over hours, days, or weeks.

There is no ideal antepartum fetal surveillance test for high-risk fetus.

The predictive accuracy of each test depends on the underlying pathophysiology of high conditions.

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The primary purpose of antepartum fetal surveillance has been to avoid fetal death and neonatal morbidity due to fetal hypoxemia and acidosis.

The know pathophysiology process leading to fetal death or in utero neurological injury are decreased uteroplacenatal blood flow, decreased gas exchange, metabolic causes, fetal sepsis, fetal anemia, injections, and cord accidents.

Antenatal monitoring is clinically useful due to two reasons:

- *Firstly*: These methods can detect or predict fetal compromise.
- *Secondly*: With appropriate interpretation and action, they can reduce the frequency or severity of adverse perinatal events and needless interventions.

One of the most challenging part of being an obstetrician is when our pregnant patient lands up being critically ill and we face the decision of whose safety to consider first – fetus or the mother. Most of us along with a critical care doctor rely on standard intensive care principles altering them logically considering the physiological changes associated with pregnancy and the need to take care of the fetus.

The importance of obstetric ICU is highlighted by the fact that most of these patients are young and disease-free initially despite which the mortality still remains higher in comparison to

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non-obstetric patients. It is important for the ICU team to realize that in a critically ill obstetric patient, the safety of the mother is of prime importance, in case one has to choose between the mother and the unborn fetus. Successful maternal and neonatal outcomes for patients admitted to critical care units are largely dependent on a multidisciplinary approach to management requiring input from critical care personnel, obstetricians, anesthetists, and neonatologists [1].

Most patients admitted to an ICU require intensive hemodynamic monitoring, and the same is true for obstetric patients. These patients would also need fetal heart monitoring which may not be available as a routine in an ICU. It is advisable to institute invasive monitoring in all hemodynamically unstable obstetric patients [2].

Groups which can be identified in an obstetric patient getting admitted into the ICU:

1. Conditions related to pregnancy – eclampsia, severe preeclampsia, hemorrhage, amniotic fluid embolus, acute fatty liver, peripartum cardiomyopathy, amniotic fluid embolism, aspiration syndromes, infections, etc.
2. Medical diseases that may be aggravated during pregnancy – congenital heart diseases, rheumatic and nonrheumatic valvular diseases, pulmonary hypertension, anemia, renal failure, etc.
3. Conditions that are not related to pregnancy – trauma, asthma, diabetes, autoimmune diseases, etc.

Our main aim once a pregnant mother is admitted in the ICU should be:

1. Understand the normal hemodynamic changes associated with pregnancy.
2. Review hemodynamic monitoring in pregnancy.
3. Examine the airway changes associated with pregnancy that affect airway intubation.
4. Examine the use of vasopressors in pregnancy.
5. Assess mortality prediction scores in pregnancy.

Hemodynamics

Indications for hemodynamic monitoring in obstetrics include severe preeclampsia with refractory hypertension, oliguric renal failure, and unclear intravascular volume status. Other indications in obstetrics include structural heart disease that may decompensate during labor and delivery, or cardiovascular collapse, such as in the setting of an acute amniotic fluid embolism. Indications for hemodynamic monitoring in ARDS or septic shock are unchanged in pregnancy compared with the nonpregnant population [3].

Normal Pregnancy Hemodynamics

Pregnancy is associated with profound hemodynamic changes that prepare women for the blood loss that occurs with delivery (Table 10.1). Blood volume increases by 20–52 % by the end of gestation [1, 2]. This increase begins as early as 6 weeks of gestation, peaks at 28–32 weeks, and then plateaus. Total blood volume in pregnancy varies but is generally 1,200–1,600 mL greater than in a nonpregnant individual [4]. The rise in blood volume results from an expansion of erythrocytes and an even greater increase in plasma volume. The disproportionate increase in erythrocytes to plasma volume results in the physiologic anemia of pregnancy [3].

In late gestation, the supine position can dramatically affect cardiovascular hemodynamics as well as respiratory function. Compression of the inferior vena cava by the gravid uterus leads to a decrease in venous return and preload, resulting in a reduction in CO of as much as 25 %. The left lateral position will relieve inferior vena cava compression and improve organ perfusion. Plasma colloid oncotic pressure falls during normal pregnancy, from 23.2 mm Hg in the first trimester to 21.1 mm Hg at term [5] and to 16 mm after delivery; that fall is even more pronounced in preeclampsia [6]. This drop in colloid oncotic pressure not only contributes to the development of pulmonary edema even in

Table 10.1 Physiological changes in pregnancy

	Direction of chance	Percentage change or normal range in pregnancy
Blood volume	↑	30–40 % increase
Heart rate	↑	Increased by 10–20 beats/min
Cardiac output	↑	30–60 % increase
Systemic vascular resistance	↓	25–30 % decrease
Blood pressure	↓	10–15 11 Hg decrease in first two trimesters
Colloid oncotic pressure	↓	10–15 % decrease
Total lung capacity	↓	4–5 % decrease
Functional residual capacity	↓	20 % decrease
Diffusion capacity	↔	No change
Tidal volume	↑	Increased
Respiratory rate	↔	No change
Minute ventilation	↑	50 % increase
PaO_2	↑	Average 100–105 mm Hg
$PaCO_2$	↓	Average 28–32 mm Hg
pH	↑	Mild respiratory alkalosis
Alveolar-arterial gradient	↑	Increase in late gestation to approximately 20 mm Hg

the absence of elevated hydrostatic pressures but also contributes to its higher incidence in the postpartum period compared with the antenatal period.

Fetal Considerations

A. Emergencies, trauma in obstetric patients:
   1. Primary survey, resuscitation
      Identify life-threatening problems by priority. Adequate resuscitation improves fetal outcome.
   2. Fetal well-being – decide if delivery is indicated.
   3. Secondary survey, comprehensive assessment.
   4. Definitive care.

B. Other conditions:

First: fetal evaluation by ultrasound

Presence, rate, and location of fetal heart; number and position of fetus(es); placental location; hematoma clot; amniotic fluid volume

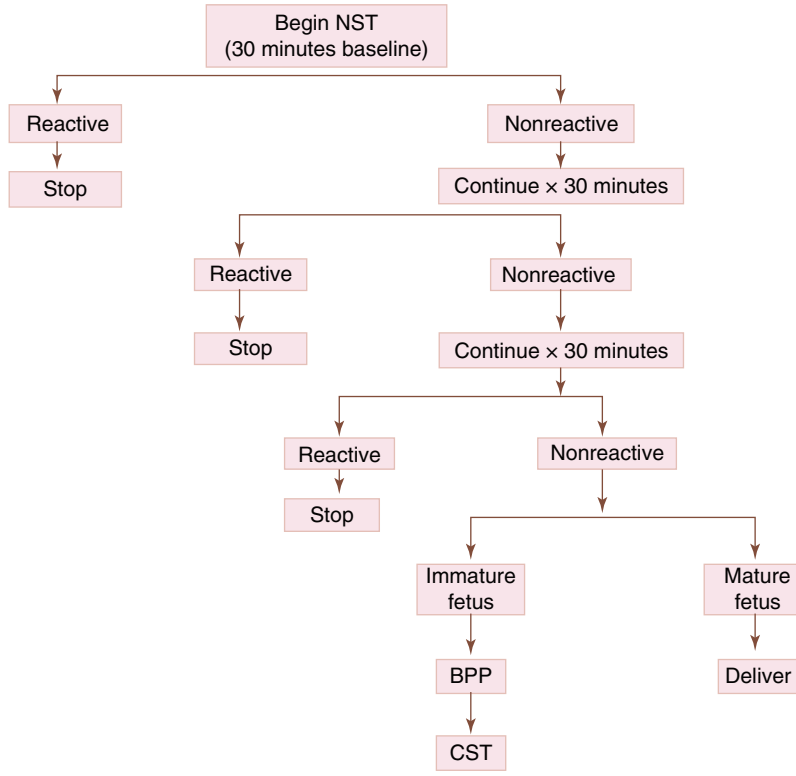
Location of fetus – inside/outside the uterus, intraperitoneal free fluid, damage to other structures

Second: fetal and uterine evaluation by CTG
heart rate – tachycardia, bradycardia variability; normal, abnormal decelerations

The evaluation of fetal heart rate patterns, without the added stress of induced contractions, has been widely incorporated into antenatal care for both screening and diagnosis. It is an assessment of immediate fetal condition.

The rationale underlying this is that the presence of spontaneous fetal heart rate accelerations associated with fetal movements (fetal reactivity) is an indicator of fetal well-being and vice versa.

NST method of antepartum fetal surveillance since three decades. NST testing originated with the early work of Hammache which linked the occurrence of fetal heart accelerations with fetal well-being. CTG is based on principle of gradually developing uteroplacental insufficiency. A stage of fetal compromise can be identified by exposing the fetus to graded stress.



Uterus – size, shape, tone, contractions, tenderness

Decision

1. Fetus – alive, dead. FHR – normal, compromised
2. Placenta – normal, abnormal
3. Amniotic fluid – normal, abnormal
4. Uterus – normal, ruptured

Correlating Fetal Heart Tracing

- Normal CTG. Check maternal pulse: different. The fetus is stable (Fig. 10.1).
- Decreased variability (Fig. 10.2) – compromised fetus. Evaluate further by color Doppler.
  Causes of decreased baseline variability
  - Sleep/quiet phase
  - Hypoxia
  - Prematureness
  - Tachycardia
  - Drugs (sedatives, antihypertensives, anesthetics)
  - Congenital malformations
  - Cardiac arrhythmias
- Decelerations – compromised fetus, but may live for 30 min. Consider delivery.
- The below figure shows FIGO classification of decelerations.

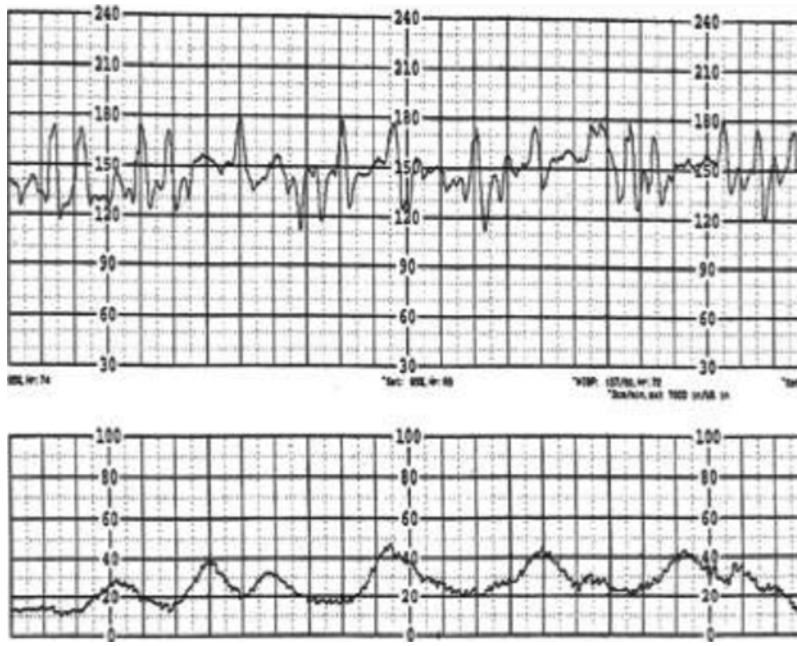


Fig. 10.1 Shows normal fetal heart tracing

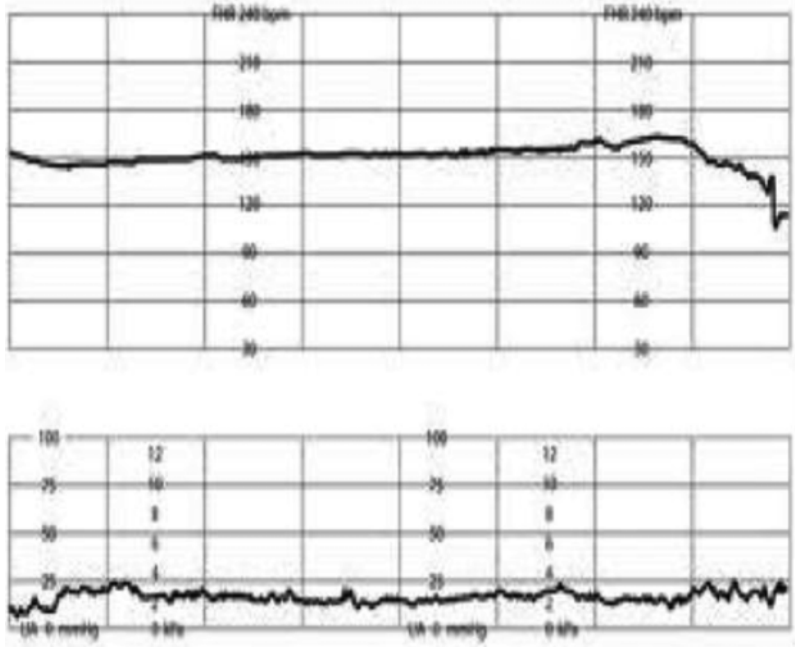
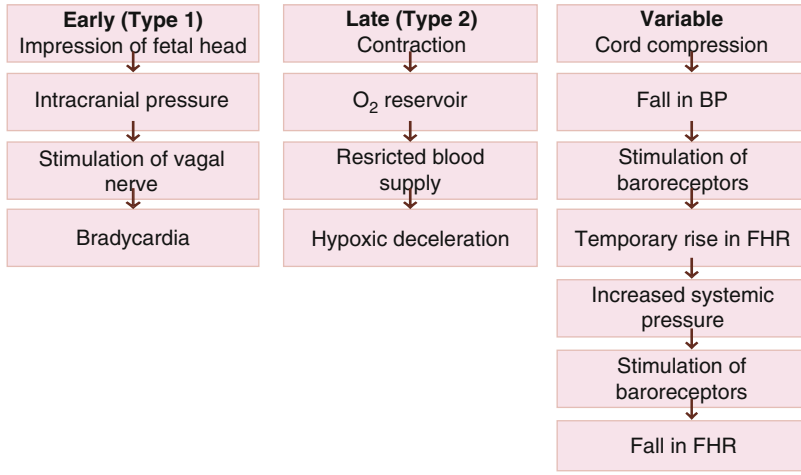


Fig. 10.2 Shows decreased variability on fetal heart tracing

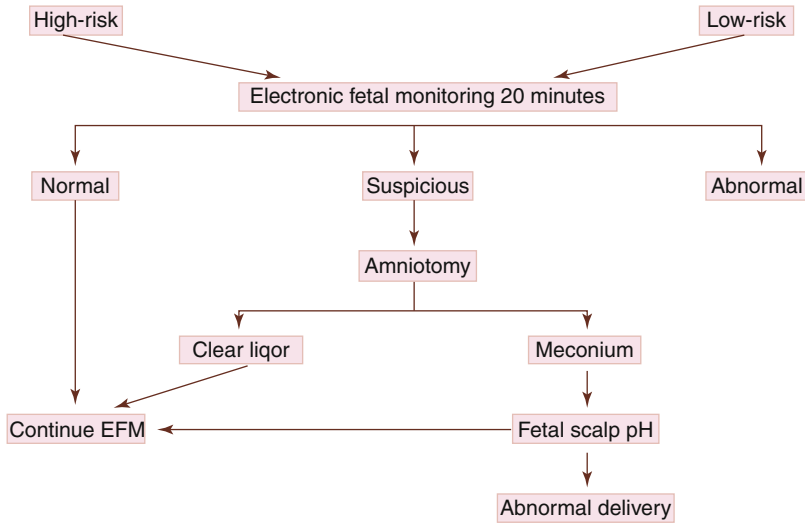


- Tachycardia over 180 – fetus compromised, continue monitoring (Fig. 10.3)
  Causes of tachycardia
  – Maternal hypovolemia
  – Anemia
  – Fever
- Bradycardia below 60 – fetus is dying – risk of brain damage/death increases with every minute: stat delivery
  Causes of bradycardia
  – Cardiac arrhythmias
  – Cord compression
- Sinusoidal (Fig. 10.4) – severe anemia (<5 g Hb), dying fetus, stat delivery
- Normal FHR (Fig. 10.1) – frequent contractions, continuous monitoring for 4 h in a high-risk/intensive care setting, 1:1 nursing

A *reactive test* is present when two or more FHR accelerations are clearly recorded during a 20-min period, each acceleration with 15 or more beats per minute and lasting 15 or more seconds, usually occurring simultaneously with episodes of fetal activity.

Variability (the minor fluctuations of the baseline rate assessed over a minute) depends on the interaction of the fetal sympathetic and parasympathetic systems and is influenced by gestational age, maternal medications, fetal congenital anomalies, fetal acidosis, and fetal tachycardia. The normal baseline variability is more than five beats.

Decision-making on Electronic Fetal Monitoring



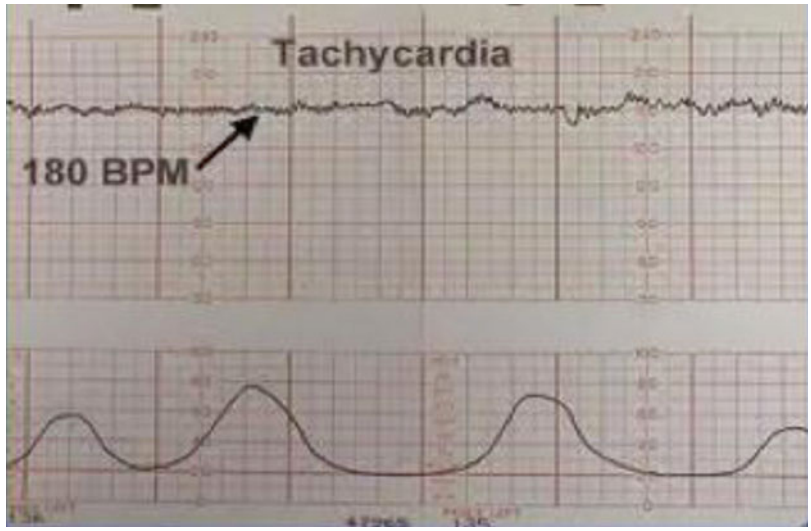


Fig. 10.3 Shows fetal tachycardia

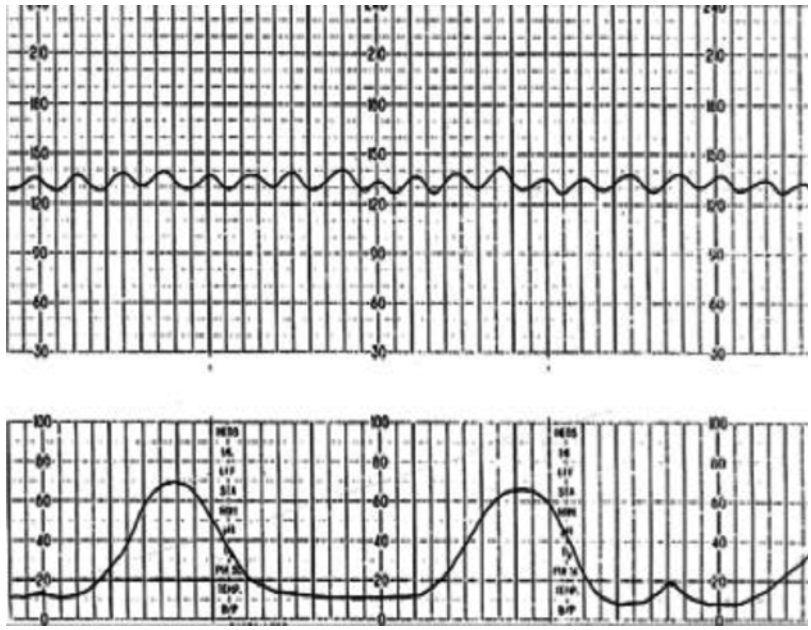
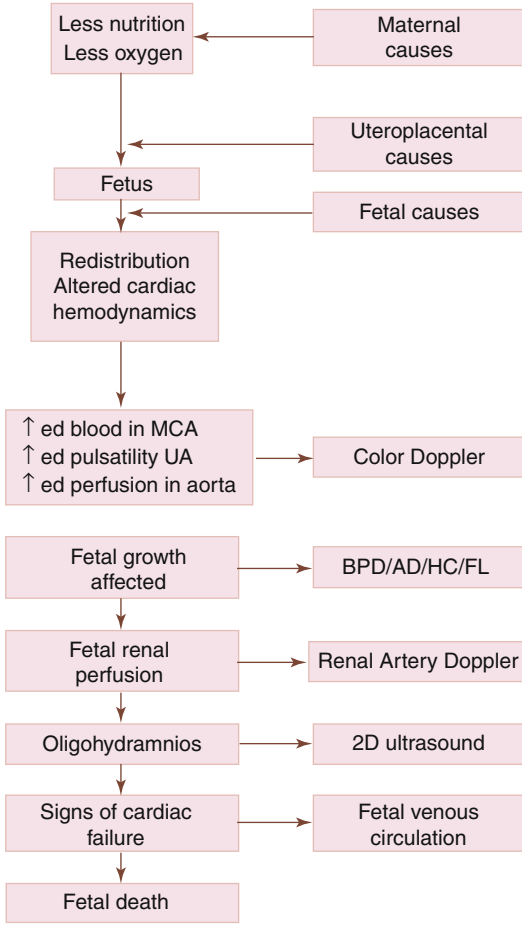


Fig. 10.4 Shows sinusoidal pattern

Addition of color Doppler gives information about fetal oxygenation, hypoxia, and acidosis, and 3D/4D ultrasound picks up subtle soft tissue markers for growth restriction. Fetal compromise is defined as a hypoxemic, hypoxic, or acidotic fetus; this is a pathophysiological deterioration of many of the FGR fetuses.

Fetal compromise is diagnosed by Doppler flow velocimetry of uterine, placental, and fetal circulation. Doppler picks up compromise earlier than biophysical profile or nonstress testing.

Pathophysiology Leading to Fetal Death and Role of Doppler in Each Step



Fetal and maternal Doppler examination gives an early and reliable warning of the fetal perfusion status and hypoxemia and acidosis can be diagnosed by Color Doppler examination is different. It is a screening kept to distinguish women at risk to develop PIH, and IUGR in low-risk cases amounts to preventive obstetrics. Doppler ultrasound is a noninvasive technique used in obstetrics to improve perinatal outcome in high-risk situations. There are accepted tools in diagnosis and management of FGR fetuses that help in reducing perinatal mortality.

It is important to remember that obstetric management should not be solely based on abnormal Doppler. All possible clinical, biochemical, and Doppler information should be put together along with the "art" of obstetrics to get the best possible outcome.

Important Points

Specific Points in Antepartum Care [1]

- Maternal position: A left lateral tilt of 15° on a firm surface will relieve aortocaval compression.
- Thromboprophylaxis: (following RCOG guidelines) requirement of prophylactic low molecular weight heparin (LMWH) [7, 8].
- Increased risk of urinary tract infection (UTI): Regular midstream specimen (MSU) should be taken.
- Use of drugs: vigilant for hemodilution; in the presence of epidural/spinal increased peripheral vascular resistance can result in an increase in the volume of drug distribution.
- Fluid balance: Anemia/heart disease/PIH/eclampsia – fluid overload can get a poor outcome. Follow the NICE guideline on management of hypertension in pregnancy in the critical care setting [9].
- Access to a operation theater in case of urgent delivery: obstetric trolley.
- Emergency drugs (always to be ready): hydralazine, $MgSO_4$, oxytocin, ergometrine, carboprost, labetolol, eclampsia pack, postpartum hemorrhage (PPH) pack.
- Antenatal steroids: RCOG Green-top guidelines [10].
- Multidisciplinary approach: daily communication and combined ward rounds.
- Fetal monitoring surveillance plan.
- Regular fetal growth ultrasound.
- Daily fetal heart rate monitoring if no maternal perception of fetal movements greater than 28 weeks gestation.

Specific Points for Postpartum Care

- Breastfeeding
- No *tilt* required
- Thromboprophylaxis: once clotting normalized for LMWH
- Debriefing and follow-up during ICU admission
- OT/PT consults following ICU
- To have normal checks: anti-D, midwife to take care of perineum, breastfeeding, bonding as per NICE postnatal care guideline [11]
- Clinical pharmacists: drug safety, pregnancy, and breastfeeding

Conclusion

Wherever a pregnant woman is receiving care, it should be a must that her pregnancy care is continued through to the postnatal period. The multiple caregivers have to ensure that the needs of the critical care do not overshadow the needs of the woman and her obstetric care.

Successful maternal and neonatal outcomes for patients admitted to a critical care facility are largely dependent on a multidisciplinary approach from ICU physician, obstetricians, anesthetists, neonatologists, and nursing staff.

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Infection Prevention and Control Policy in Obstetric HDU and ICU

11

Jayam Kannan

Introduction

Healthcare-associated infections (HAI) remain a major cause of morbidity, mortality and excess healthcare costs. HAI contribute a considerable cost to the healthcare system, as well as to patients and their families with prolonged hospital stays, readmissions and additional diagnostic tests and treatment. Obstetric units are not an exception. Most infections are caused by the microorganisms of the mothers' vaginal flora. In India, the scenario of high-risk delivery unit (HDU) development is fast catching up and after initiatives, promotion, education and training programmes. There has been stupendous growth in this area but much needs to be done in area of infrastructure, human resource development, protocol, guidelines formation and research which are relevant to Indian circumstances. An acceptable and logistically feasible zero compromise can be made on qu ality healthcare delivery, yet acceptable guidelines can be adopted for making HDU designing. Guidelines which may be good for both rural and urban areas as also for smaller and tertiary centres which may include teaching and non-teaching institutes are required. The major obstetric indications for admission in tertiary units were septicaemia (35.08 %) [1].

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There are many complex reasons for difficulties in these developments in India, due to:

- Increasing population, rising poverty, crowded living conditions, poor sanitation and malnutrition – all of which foster the spread of disease.
- Environmental degradation and widespread air, soil and water pollution also increase the spread of infection.

Infection control in delivery care units in Gujarat state, India, has been done in 2011 [2] wherein it is found, 70 % of respondents said that standard infection control procedures were followed, but a written procedure was only available in 5 % of facilities. Alcohol rubs were not used for hand cleaning, and surgical gloves were reused in over 70 % of facilities, especially for vaginal examinations in the labour room. Most types of equipment and supplies were available, but a third of facilities did not have wash basins with 'hands-free' taps. Only 15 % of facilities reported that wiping of surfaces was done immediately after each delivery in labour rooms. Blood culture services were available in 25 % of facilities, and antibiotics are widely given to women after normal delivery. A few facilities had data on infections and reported rates of 3–5 %.

Genital tract sepsis has become a leading cause of maternal death in the UK, and this is of particular relevance to the maternity high

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dependency unit (MHDU) where it is likely that not only women with a diagnosis of sepsis may be cared for but also women who are at risk of maternal sepsis. It was commented upon in the last confidential enquiry that the advent of antibiotics and aseptic precautions had led to a dramatic reduction in the number of deaths from sepsis in the early years of the confidential enquiry and that this in turn had removed the anxiety of maternal sepsis from our 'collective memory'. The report recommended action to raise awareness of the recognition and management of maternal sepsis in all healthcare professionals who may care for the obstetric patient and also that maternal early warning scoring systems be implemented [3].

Obstetric sepsis may be specific to pregnancy but may also occur as a result of any of the common community-acquired pathogens (e.g. pneumococcal pneumonia). The most recent (eighth) Report of the Confidential Enquiries into Maternal Deaths in the UK revealed an increase in genital tract sepsis-related mortality from 0.85 per 100,000 deliveries in the 2003–2005 triennium to 1.13 per 100,000 for the 2006–2008 period. Substandard evaluation and management of the septic parturient have contributed significantly to this increased mortality rate [1].

Special Features of Pregnancy Adaptation for Sepsis

In the non-pregnant population, there are well-recognised definitions for bacteraemia, infection, sepsis, systemic inflammatory response syndrome, severe sepsis and septic shock. These definitions are applicable to the obstetric population, but the physiological adaptations to pregnancy make interpretation of physiological derangements less clear-cut. Physiological adaptations to pregnancy predispose parturients to infection as well as limit their physiological reserve in the event of sepsis. The markedly increased cardiac output, blood volume and associated reduction in systemic vascular resistance at term leaves an already-stressed cardiovascular system at risk of precipitous decompensation in the event of sepsis-induced vasodilation and myocardial depression. The relative hypoalbuminemia of pregnancy puts parturients at greater risk of developing pulmonary oedema in the event of sepsis and its associated leaky capillaries. The mild respiratory alkalosis (and associated compensatory mild metabolic acidosis) that develops as a result of the increased minute ventilation makes parturients less capable of compensating for the metabolic acidosis associated with sepsis/septic shock.

What Is Sepsis?

Obstetrician must be clear about definitions in sepsis. The systemic inflammatory response syndrome (SIRS) is tachycardia, tachypnoea, pyrexia or hypothermia, or a WBC greater than 12,000 mm^3 or less than 4,000 mm^3 whilst sepsis is a confirmed infectious process. The sepsis syndrome (severe sepsis) is SIRS with a confirmed infectious process and organ dysfunction.

It is imperative in any unstable patient to consider sepsis within the differential diagnosis. Early symptoms and signs are usually non-specific (tachypnoea can be an early significant sign), and it is not uncommon for a routine blood test abnormality (white cell count, rising CRP) to be found which will alert the medical staff to occult sepsis.

A patient can have dual pathologies, i.e. two obstetric conditions at the same time, and it is important to remember this. If a patient has severe pre-eclampsia, she may have symptoms relating to this primary disease but may also have sepsis. The signs of pre-eclampsia can cloud and compound the symptoms, signs and investigations of sepsis. Most infections of obstetric origin are polymicrobial involving aerobic and anaerobic organisms indigenous to the genital tract and commonly include streptococci, *E. coli*, *Klebsiella* and *Bacteroides* species.

Predisposing factors for puerperal sepsis include:

Caesarean section
Prolonged labour and membrane rupture

Traumatic vaginal delivery
Forceps
Episiotomy
Retained placenta
Repeated vaginal examinations
Intrauterine manipulation
Low socio-economic groups

The first assessment is a *clinical* review of the patient: a detailed history and a full examination are necessary. Normally, puerperal sepsis presents with a temperature, lower abdominal pain and uterine tenderness. If untreated, it will lead onto malaise, anorexia, foul lochia and severe abdominal pain. A pelvic abscess may develop when a spiking temperature, vomiting, lower abdominal pain, ileus and a tender abdominal mass may be palpated in the pelvis. In the general examination, two phases of sepsis may be distinguished.

1. Hyperdynamic phase – warm pink patient with bounding pulse
2. Hypovolaemic phase – patient with pallor, sweating with a thready pulse

Sources of Obstetric Infection

Urinary tract infection/pyelonephritis, postpartum endometritis (Caesarean/vaginal delivery), chorioamnionitis and septic abortion are some of the specific causes.

We do see cases of pneumonia, typhoid and malaria as seen in the general population.

Pyelonephritis is a leading cause of maternal sepsis. The genitourinary physiological adaptations to pregnancy which put mothers at risk include the reduced renal concentrating ability and urinary stasis secondary to ureteric dilation and bladder flaccidity. The relatively acidotic vaginal wall epithelium along with the presence of greater glycogen stores predisposes the parturient to chorioamnionitis and septic abortion. The gravid uterus, increased intra-gastric pressure as well as reduced gastro-oesophageal sphincter tone put parturients at increased risk of aspiration and subsequent pneumonitis.

The Most Important Microorganisms Are Group B *Streptococcus* and *E. Coli*

- Caesarean section is associated with higher endometritis (10–20 %) than vaginal delivery.

Infection Prevention and Control Policy in Obstetric HDU and ICU

This starts with the initial planning for HDU [4]. Designing the bed layout and providing optimum space for each bed according to various types of space available, choose between free hanging power columns or head end panel facilities. Patient area should not be less than 100 sq ft per patient (>125 sq ft will be ideal). Designing and planning of location, space, facilities for nursing staff, support staff, like cleaning staff, guards and Class IV. Equipmentation includes ETO sterilisation and pollution control buckets, one set for each bed.

Environment Protection

Effective steps and planning to control nosocomial infections include flooring, walls, pillars and ceilings, lighting, surrounding heating/AC/ventilation, waste disposal and pollution control, as well as protocol about allowing visitors, shoes, etc., inside HDU.

Negative-pressure isolation rooms (isolation of patients infected/suspected to be infected with organism spread via airborne droplet nuclei <5 μm in diameter). In these rooms, the windows do not open. They have greater exhaust than supply air volume. They have pressure differential of 2.5 Pa. In clean to dirty airflow, i.e. direction of the airflow is from the outside adjacent space (i.e. corridor, anteroom) into the room, air from room is preferably exhausted to the outside, but may be re-circulated provided it is through HEPA filter NB: re-circulating air taken from areas intended to isolate a patient with TB is a risk not worth taking and is not recommended.

Positive-pressure isolation rooms (to provide protective environment for patients at highest

risk of infection, e.g. neutropenia, post-transplant) may be required in tertiary setup.

Waste Disposal and Pollution Control

This is mandatory and a huge safety issue both for the patient and staff/doctors of the hospital and society at large. It is important that all government regulations (State Pollution Control Board in this particular case) should strictly be complied with. It is mandatory to have four covered plans (yellow, blue, red, black) provided for each patient or may be one set between two patients to save space and funds. This is needed to dispose of different grades of wastes.

Hand Hygiene and Prevention of Infection

Use of an alcohol-based handrub is the preferred hand hygiene method (NICE CG 74) when hands are not visibly soiled. Because cleansing hands with an alcohol handrub is quicker and simpler than handwashing with soap and running water, it may improve hand hygiene among healthcare workers. If an alcohol-based handrub is unavailable, wash hands with soap and running water.

Appropriate times for Staff to Use Alcohol-Based Handrub

Cleaning hands with an alcohol-based handrub kills or inhibits microorganisms, but this does *not* remove microorganisms or soil [5]. Alcohol-based handrub is used only if hands are not visibly dirty. Because using alcohol alone tends to dry the skin, it is best to use a commercially available product. If none are available, make an alcohol handrub solution by adding together 2 mL of glycerin, propylene glycol or sorbitol and 100 mL of 60–80 % alcohol. Always clean and disinfect reusable bottles used to store alcohol handrub solutions before refilling them.

- Immediately when arriving at work
- Before and after examining each client
- After touching anything that might be contaminated
- After contact with body fluids or excretions, mucous membranes, nonintact skin or wound dressings
- After handling specimens
- Before putting on gloves for clinical procedures
- After removing any type of glove
- Before handling an invasive device or doing an invasive procedure (inserting a central venous or indwelling catheter, spinal tap, etc.)
- Before leaving work

Kinds of Gloves Required

1. Sterile gloves

   These are used when there will be contact with the bloodstream or with tissues under the skin (e.g. surgical procedures, pelvic examination for women in labour, etc.). Such gloves should be discarded after one use. Never use the same pair of gloves to care for more than one person.
2. Single-use exam gloves

   These gloves, which are clean but are not sterile, are used when there will be contact with intact mucous membranes or where the primary purpose of gloving is to reduce the provider's risk of exposure. These gloves should be discarded after one use. Never use the same pair of gloves to care for more than one person.
3. Utility or heavy-duty household gloves

   These are used for handling contaminated items, handling medical or chemical waste and performing housekeeping activities.

Gloving Tips

- Always wash utility gloves before you take the gloves off your hands.
- Always cleanse hands with an alcohol-based handrub after removing any type of gloves, or wash hands with soap and running water if they are visibly contaminated.

- Always discard sterile and single-use gloves after one use. Never process or reuse these gloves (regardless of past practices at your facility), as this has been associated with the transmission of infections.

Every bed should have attached alcohol-based anti-microbial instant handwash solution source, which is used before caregiver (doctor/nurse/relative/paramedical) handles the patient. Water basin at all bedsides has not proven popular and successful because of poor compliance by one and all and also for reasons of space constraints and maintenance issues.

An operation room style sink with elbow- or foot-operated water supply system with running hot and cold water supply with antiseptic soap solution source should be there at a point easily accessible and unavoidable, where two people can wash hands at a time.

This sink should have an immaculate drainage system, which usually may become a point of great irritation and nuisance in later years or months. All entrants (irrespective of doctors or nurses) should don mask and cap in ICU and ideally an apron which should be replaced daily).

No dirty/soiled linen/material should be allowed to stay in ICU for long times for fear of spread of bad odour, infection and should be disposed off as fast as possible. Dirty linen should be replaced regularly at fixed intervals.

Surgical Hand Preparation Tips

- If you routinely perform surgical procedures, you should keep your fingernails short.
- Always keep your hands above your elbows during and after scrubbing with alcohol-based handrubs or antiseptic solutions.
- Always follow the manufacturer's recommendations for the use of alcohol-based handrubs or antiseptic solutions.
- Warm water makes antiseptics work more effectively. Avoid using hot water, which removes protective oils from the skin.

Double Gloving

The WHO recommends that double gloving (i.e. wearing two pairs of sterile gloves) be used in countries with a high prevalence of hepatitis B, hepatitis C and HIV for surgical procedures anticipated to last longer than 30 min, for procedures where contact with large amounts of blood or other body fluids is likely (e.g. vaginal deliveries) and for high-risk orthopaedic procedures. A review of research studies showed that wearing a second pair of sterile gloves significantly reduced perforations to the inner gloves (and thus would – in theory – lessen the likelihood that an infection will be transmitted to the healthcare worker), without apparently affecting surgical performance. A single pair of gloves generally provides appropriate protection to healthcare workers during nonsurgical client care that may involve contact with blood or other body fluids; in these cases, double gloving is not recommended, because it unnecessarily wastes resources.

Management of Septic Shock Cases

This should be managed in accordance with the Surviving Sepsis Campaign guidelines and requires a multidisciplinary team approach. Bacteraemia can progress rapidly to severe sepsis and septic shock leading to collapse. The most common organisms implicated in obstetrics are the streptococcal groups A, B and D, pneumococcus and *E. coli*.

Recognition of Sepsis

Each hour of delay in achieving administration of effective antibiotics is associated with an increase in mortality. Regular completion of vital signs on a MEOWS chart and documentation of results will assist in the early recognition of sepsis and the critically ill woman.

Sites for Sepsis

- Endometrium
- Urinary tract
- Episiotomy
- Abdominal incision
- Lung infection
- Breast
- Legs – phlebitis
- Epidural site
- Systemic concomitant disease – e.g. influenza

Blood Transfusion Reactions

Environmental – warm fluids, warming blankets
Drugs – atropine overdose, drug interactions, neuroleptics
Endocrine – hyperthyroidism, phaeochromocytoma
Hypothalamic injury – cerebral hypoxia or oedema

Investigations in Sepsis

CBC – WBCs up or down, platelets normal or down
CRP – elevated with a rising trend
Coagulation screen – disseminated intravascular coagulation
Urea and electrolytes – renal impairment
*LFT*s – liver dysfunction, hypoalbuminaemia
Electrocardiograph – dysrhythmias or ischaemia
Blood gases – hypoxaemia, acidosis
Blood cultures – aerobic and anaerobic
Other cultures – high vaginal, sputum, urine, drains, wound, catheters
Specific – chest X-ray, ultrasound, CT scan, laparotomy

Diagnostic Criteria

Include the following signs of systemic infection:

- Fever (core temp >38.8 °C) OR hypothermia (core temp <36 °C)
- Tachycardia (>90 beats min −1)
- Tachypnoea (>30 bpm)
- Hypotension (SAP <90 mmHg)
- Hypoxia
- Decreased capillary refill
- Mottling of skin
- Altered mental status (anxiety)
- Oliguria
- Significant fluid balance
- WCC <4 or >12
- C-reactive protein >2 SD above normal
- Creatinine increase
- Hyperglycaemia (>7.7 mmol litre-1) in non-diabetic patients
- Coagulopathy (INR >1.5, APPT >60 s, platelets <100)
- Lactate >3 mmol/l

Criteria 1 for Considering if a Patient Has Sepsis [5]

Known or strongly suspected infection and any two of the following:

Fever or hypothermia (temperature −38 °C or −36 °C)
Tachycardia: heart rate −100/min
Tachypnoea: respiratory rate −20/min or spontaneous
PaCO2 −4.3 kPa
WBC −12,000/mm^3 or −4,000/mm^3 *or a clinical suspicion*

Criteria 2 Indicating Hypoperfusion or Organ Failure

Any ONE of these:

Systolic BP, 90 mmHg or mean arterial pressure (MAP)
Pressure for more than 1-h urine output, 0.5 ml/kg/h for more than 1 h
Deteriorating level of consciousness (not due to sedation or known CNS disease)
Metabolic acidosis: pH, 7.30 + base deficit. 5 mmol/l or lactate.4.0 mmol/l

Treatment

If criteria 1 and 2 are fulfilled, the diagnosis of sepsis syndrome or severe sepsis is made. The

time of diagnosis should be documented. This is a medical emergency and the patient's consultant should be informed.

The following ward and HDU actions should be taken in the *first hour*:

100% oxygen via a rebreathing oxygen mask
Early fluid resuscitation (as much as 20 ml/kg immediately)
Blood cultures (as well as genital tract, sputum, urine and line swabs)
Broad-spectrum antibiotics should be prescribed (with microbiologist's advice)
All blood specimens taken including CRP
Urinary catheterisation
Lactate sample taken
Check whether all these steps have been done after 1 h

It is important to plan for ongoing care of the patient. Identify the source and ensure that continuous antibiotics are being given. Take cultures before giving antibiotics and swab all likely sites. It is important to remember that 75 % of cultures are positive from the primary source and about 20 % of blood cultures are positive in severe infections.

Acute Management Plan for Septic Patient

Oxygen – up to 100 %
IV fluid resuscitation
Monitoring – clinical and technical
Recording – HDU chart noting trends
Physiotherapy
Drugs
DVT prevention
Therapeutic antibiotics
Co-morbid conditions
Management of concomitant obstetric conditions

The Surviving Sepsis Campaign [6] has updated the management of sepsis and septic shock and the following 'care bundle' should be applied immediately or within 6 h, and it has been shown to significantly improve survival rates.

1. Measure serum lactate.
2. Obtain blood cultures/culture swabs prior to antibiotic administration.
3. Administer broad-spectrum antibiotic(s) within the first hour of recognition of severe sepsis and septic shock according to local protocol.
4. In the event of hypotension and/or lactate >4 mmol/l: normal include
   (a) Deliver an initial minimum of 20 ml/kg of crystalloid/colloid.
   (b) Once adequate volume replacement has been achieved, a vasopressor (norepinephrine, epinephrine) and/or an inotrope (e.g. dobutamine) may be used to maintain mean arterial pressure over 65 mmHg. Further management consists of level 3 critical care and referral to the intensive care team for continuation of care bundle.
5. In the event of hypotension despite fluid resuscitation (septic shock) and/or lactate over 4 mmol/l:
   (a) Achieve a central venous pressure of at least 8 mmHg (or over 12 mmHg if the woman is mechanically ventilated) with aggressive fluid replacement.
   (b) Consider steroids.
6. Maintain oxygen saturation with facial oxygen. Consider transfusion if haemoglobin is below 7 g/dl. Continuing management involves continued supportive therapy, removing the septic focus, administration of blood products if required and thromboprophylaxis (The management and early recognition of the severely ill obstetric women/August 2012/ review August 2015).

It is important to acknowledge some of the maternal deaths from sepsis were associated with fluid overload and pulmonary oedema. Pregnant women may be particularly susceptible to lung damage because of alterations in fluid dynamics. There is a fine balance on management between

maintaining the circulation and risking lung damage.

MRSA Screening

Hull and East Yorkshire Hospitals has a comprehensive policy which details all aspects of MRSA screening; however, fertility or obstetric admissions are excluded unless they have high-risk criteria.

High-risk criteria are:

- Patients known to have or previously have had MRSA.
- Patients who are themselves healthcare workers.
- Patients who are frequent hospital attenders. This is defined for MRSA screening purposes as: 'An inpatient stay within the last year or Three or more attendances at out-patient clinics in the last year'.
- All 'long-stay patients'. All patients who have been inpatients for over 30 days must have a full screen if they have not been screened for other reasons. This should be repeated every 30 days whilst they remain inpatients.
- When patients are admitted directly toward a screen will be taken within 24 h of admission.

Use of Prophylactic Antibiotics

1. All women undergoing elective or emergency Caesarean section [7] should receive antibiotic prophylaxis. (I-A)
2. The choice of antibiotic for Caesarean section should be a single dose of a first-generation cephalosporin. If the patient has a penicillin allergy, clindamycin or erythromycin can be used. (I-A)
3. The timing of prophylactic antibiotics for Caesarean section should be 15–60 min prior to skin incision. No additional doses are recommended. (I-A)
4. If an open abdominal procedure is lengthy (>3 h) or estimated blood loss is greater than 1,500 mL, an additional dose of the prophylactic antibiotic may be given 3–4 h after the initial dose. (III-L)
5. Prophylactic antibiotics may be considered for the reduction of infectious morbidity associated with repair of third- and fourth-degree perineal injury. (I-B)
6. In patients with morbid obesity (BMI >35), doubling the antibiotic dose may be considered. (III-B) 7. Antibiotics should not be administered [7, 8] solely to prevent endocarditis for patients who undergo an obstetrical procedure of any kind. (III-E)

Conclusion

Early screening of high-risk mothers, vigilant antenatal care and proper maintenance of asepsis during delivery and post-partum period can reduce HDU utilisation rate and can result in healthier outcome. The variety of grades of recommendations hamper across-guideline comparison. Nevertheless, infection rates before compliance with NICE guidance have ranged from 5.7 to 9.0 %. After introducing the guidelines, rates of SSI were reduced by 3.3 and 3.8 %, respectively, in many centres. Local health policy decisions on whether to include or refrain from including screening measures in preventive care programmes can be facilitated by the comparison of recommendations from international evidence-based guidelines. Beyond the availability of evidence, our country's health policy makers will have to make a judgement on the value of the test for a population-wide screening.

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12 Transfusion of Blood Components and Derivatives in the Obstetric Patients

Lakhbir Dhaliwal and Rakhi Rai

Introduction

Blood transfusion in obstetrics is of paramount importance as the maternal mortality due to obstetric haemorrhage is 25–30 % and anaemia is 15 % and in the antenatal period foetus is also at stake due to incorrect transfusion or due to anaemia. The inappropriate transfusion rate is around 15–45 %, either due to transfusion in unindicated cases or due to too late or too little transfusion in indicated cases. The risk of transfusion reaction and transmission of infections with restricted availability of blood has made us to limit the transfusion to indicated cases only. Transfusion of blood and blood products requires expertise and training of different cadre of workers – doctors, blood bank staff and nurses – to ensure the blood safety. The major risk of transfusion is that of incorrect blood/ blood product replacement. Hence strict guidelines need to be followed to have correct sampling, crossmatching and administrative processes.

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Discussion

Appropriate use of blood products is defined as the transfusion of safe blood products to treat a condition leading to significant morbidity or mortality that can't be prevented or managed effectively by other means.

- Blood transfusion may be a life-saving procedure but it is not without risk.
- Always weigh the risk of transfusion against no transfusion.
- The decision to transfuse blood should not be based on haemoglobin levels alone, but also on the patient's clinical needs.
- Transfusion does not treat the cause of anaemia.
- Transfusion does not correct the non-haematological effects of iron deficiency.

How to Prevent the Need for Blood Transfusion during Pregnancy or at the Time of Delivery

- Anaemia should be treated in pregnancy, preferably by oral haematinics. Parenteral iron is indicated if patient is not able to tolerate oral iron or if patient compliance is poor.
- Women who are at risk of haemorrhage should be advised to have a hospital delivery.
- Blood loss at the time of delivery should be minimised.

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- Active management of third stage of labour is recommended to decrease the blood loss.

Blood and Blood Components

Blood from a single donor can be divided into different blood components and can be used according to patient's needs:

Blood product – Any therapeutic substance prepared from human blood

Whole blood – Unseparated blood collected into an approved container containing an anticoagulant-preservative solution

Blood component

1. A constituent of blood, separated from whole blood, such as:
   - Red cell concentrate
   - Red cell suspension
   - Plasma
   - Platelet concentrates
2. Plasma or platelets collected by aphaeresis*
3. Cryoprecipitate, prepared from fresh frozen plasma, which is rich in factor VIII and fibrinogen

Plasma derivative such as:

- Albumin
- Coagulation factor concentrates
- Immunoglobulins

Blood Safety

The following factors affect the risks associated with blood transfusion:

- Incidence and prevalence of infections in blood donors transmitted by blood transfusion
- Procedures for selection and screening of donors
- Quality of screening of donated blood for infections
- Efficacy of blood grouping, preparation of blood components, storage and transportation of blood and blood products
- Prescription of transfusion of blood and blood products when there is no other alternative
- Reliability of system ensuring the receipt of compatible blood by the patients

Key Points for Transfusion Procedures

- There should be standard operating procedures for transfusion processes.
- There should be a clear communication between blood bank and clinical staff to ensure the issue of appropriate and safe blood.
- Blood should not be released if the blood request form is incomplete and sample label was not appropriate.
- Issued blood should be properly stored before transfusion or during transport to prevent the contamination of blood or loss of function.
- The patient's identify must be cross-checked with blood before transfusion. The patient should be properly monitored during transfusion and after completion of transfusion.

The following steps need to be in place in each hospital for ensuring safe blood transfusion:

- Standard blood request form.
- Schedule for ordering blood for common surgical procedures.
- Clear cut indications for use of blood and blood products.
- Standard operating procedures for transfusion process at each stage must be followed as:
  - Ordering of blood and blood products for emergency conditions.
  - Ordering of blood and blood products for elective surgeries.
  - Blood request form must be sent complete.
  - Taking and proper labelling of pretransfusion blood sample for crossmatching.
  - Collection of blood products from blood bank.
  - Proper transport and storage of blood products after being issued from blood bank.

  - Transfusion of blood products after cross-checking the patient identity.
  - Maintenance of record of transfusion in patient record sheets.
  - Monitoring of patient during and after completion of transfusion.
  - Investigation, management and recording of any transfusion reaction if it occurs.
- The staff should be well trained in following the standard operating procedures.

Ordering of Blood Products

Procedure for ordering of blood products depends on:

- Urgent need for blood
- Definite need for blood
- Possible need for blood

Informing the Patient

Patient and the relatives should be informed about the need for transfusion and risks associated with it and possible alternatives in the language they understand the best. Written consent should be taken after providing all the information. For the unconscious patients, next of the kin should sign the consent.

- Assess the patient's need for blood.
- Inform patient and relatives about the transfusion and record it in patient record form also.
- Note the indication of transfusion in patient's file.
- Type of blood product and quantity required to be noted.
- Complete blood request form accurately, and also mention the reason for transfusion so that suitable product can be selected for compatibility testing.
- Correctly label the blood sample.
- Record in patient's file about:
  - Type and amount of blood and blood products transfused
  - Donation no. of each unit transfused
  - Blood group of each unit transfused
  - Time of start of transfusion
  - Signature of person who checked and transfused the blood products

Urgent Request for Blood

Blood bank should also understand the pressure of clinicians under which they are treating a patient who requires immediate transfusion. Blood bank should use laboratory procedures which are appropriate to the urgency of the situation.

There should be standardisation on the language to be used between blood bank staff and clinical staff to avoid any misinterpretation.

- Extremely urgent – Within 10–15 min
- Very urgent – Within 1 h
- Urgent – Within 3 h
- Same day
- Or date and time required

Information on Blood Request Form

- Date of request
- Date and time blood is needed
- Where blood is required (ward)
- Patient name with sirname
- Age
- Patient gender
- Hospital registration number
- Provisional diagnosis
- Reason of transfusion
- No. of units of blood/blood products required
- Urgency of request
- Patient blood group if known
- Presence of antibodies (any)
- History of any previous transfusion
- History of any previous transfusion reaction
- No. of previous pregnancies and maternal infant compatibility
- Relevant medical history/condition
- Name and signature of person requesting blood

- If another blood request is sent to blood bank for same patient within short-time framework, same identification pointers should be used on request form so that even the blood bank staff should know that they are dealing with same patient.
- If patient needs repeat blood transfusion, new blood sample must be sent for crossmatching especially if transfusion was given more than 24 h back as antibodies may form after being immunologically stimulated by transfused red cells in order to avoid incompatible blood transfusion. Also patient's blood sample sent for crossmatching should not be more than 7 days old as red cell alloimmunisation is most likely to occur in the third trimester.
- Only Kell-negative blood should be transfused in women of child-bearing age due to risk of alloimmunisation.
- Pre-autologous deposit is not recommended in pregnancy.

Storage of Blood and Blood Products

Red Blood Cells/Whole Blood

- Red blood cells/whole blood can be stored at 2–6 °C as below 2 °C, red cells which freeze get haemolysed and above 6 °C, there *is a risk* of bacterial contamination.
- They should be transfused within 30 min of removal from refrigerator.
- If red blood cells/whole blood has been kept out of refrigerator for more than 30 min, they should be discarded because of the risk of bacterial contamination and loss of cell function.
- If in domestic fridge, store in middle shelf below chiller tray, but never in the freezer.

Platelet Concentrates

- Stored at temperature of 20–24 °C as platelets lose their capacity of blood coagulation/clotting at lower temperature.
- Storage life is restricted to 3–5 days.
- They should be transfused as soon as possible and should never be kept in the refrigerator.

Fresh Frozen Plasma

- Stored at −25 °C.
- Factor V and VIII levels fall rapidly over 24 h if FFPs are not stored at −25 °C/colder.
- Should be transfused within 30 min of thawing.

Tips for Storage of Blood and Blood Products

- Never open the door of the refrigerator repeatedly.
- Blood/blood products should not be so tightly packed that *cold air* is not able to circulate in between the packs.
- Never keep anything else other than blood/blood products in the refrigerator.
- Platelets should not be stored in the refrigerator.
- Never store blood in the door of domestic refrigerator/near freezer compartment.

Blood and Various Blood Products and Their Characteristics

Parameters	Whole blood	PRBC (CPDA-1)	PRBC-SAGM
Description	Collected from blood donor in CPDA-1 solution	Red blood cell concentrate from which most of plasma has been removed	Red cell concentrate from which major part of the plasma and the buffy coat layer has been removed with addition of a nutrient solution (SAGM)
Volume	399 ml (350 blood + 49 ml CPDA-1) and 513 ml (450 blood + 63 ml CPDA-1)	200–300 ml	250–350 ml
Haemoglobin	12 g/100 ml	20 g/100 ml (≥45 g/bag)	≥45 g/bag
Haematocrit	30–40 %	65–75 %	55–65 %
Storage condition	+2 to +6 °C	+2 to +6 °C	+2 to +6 °C
Shelf life	35 days	35 days	42 days
Indications	Red cell replacement in acute blood loss	Anaemic patients	Anaemic patients
	Exchange transfusion	In acute blood loss along with crystalloids and colloids	Previous history of febrile reactions to red cell transfusion
	Where PRBCs are not available		
Administration	Should be transfused within 30 min of removal from refrigerator	Should be transfused within 30 min of removal from refrigerator	Should be transfused within 30 min of removal from refrigerator
	Transfusion to be completed within 4 h of starting blood	Transfusion to be completed within 4 h of starting blood	Transfusion to be completed within 4 h of starting blood

Platelet Products

Parameters	Platelet concentrate (PC)	Apheresis platelets (SDAP)
Description	Prepared from either 350 ml or 450 ml whole blood	Platelet concentrate derived from blood donor using an apheresis
	Also known as random-donor platelets (RDPs)	Also known as single-donor apheresis platelets (SDAP)
Volume	50–90 ml	200–300 ml
Platelet content	$3.5–4.5 \times 10^{10}$/unit	$3–7 \times 10^{11}$/unit
Dosage	1 unit of platelet concentrate/10 kg body weight	One pack is usually equivalent to one therapeutic dose
Storage and shelf life	3–5 days at 20–24 °C	5 days at 20–24 °C
Indications	Thrombocytopenia	Same as for RDPs
	Platelet function defects	Patients experiencing frequent febrile reactions with platelet concentrate
	Prevention of bleeding due to thrombocytopenia, such as in bone marrow failure	
Administration	Initiate transfusion slowly for first 15 min unless massive blood loss	Same as random-donor platelet
		ABO compatibility is more important

Plasma Products

Parameters	Fresh frozen plasma (FFP)	Cryoprecipitate
Description	Plasma separated from one whole blood within 6 h of collection and then rapidly frozen to −25 °C colder	Prepared from fresh frozen plasma by collecting the precipitate formed by controlled thawing at +4 °C
Volume	150–220 ml	15–20 ml
Content	Contains normal plasma levels of stable clotting factors, albumin and immunoglobulins. Factor VIII level at least 70 % of normal fresh plasma levels	Factor VIII : 80–100 IU/bag
		Fibrinogen : 150–300 mg/bag
Dosage	Initial dose of 15 ml/kg	1 bag/10 kg body weight
Storage and shelf life	At −25 °C or colder for up to 1 year	At −25 °C or colder for up to 1 year
Indications	Multiple coagulation factor deficiencies:	As an alternative to factor VIII concentrate in the treatment of inherited deficiencies of:
	Liver disease	Von Willebrand factor (von Willebrand disease)
	Warfarin overdose	Factor VIII (haemophilia A)
	Depletion of coagulation factors in patient receiving large-volume transfusions	Factor XIII
	Disseminated intravascular coagulation (DIC)	As a source of fibrinogen in acquired coagulopathies: disseminated intravascular coagulation (DIC)
	Thrombotic thrombocytopenic purpura (TTP)	
Administration	Must normally be ABO compatible to avoid risk of haemolysis in recipient	Can be transfused across ABO barrier
	Infuse as soon as possible after thawing	After thawing, infuse as soon as possible
	Labile coagulation factors rapidly degrade; use within 6 h of thawing	Must be infused within 6 h of thawing

Collection of Blood Products Prior to Transfusion

Patient identification slip showing patient name, husband name, age, hospital registration no., patient's ward and blood group duly filled by the clinician should be shown in the blood bank for issuance of blood.

Time limits for infusion		
	Start infusion	Complete infusion
Whole blood/ red cells	Within 30 min of removing pack from refrigerator	Within 4 h (or less in high ambient temperature)
Platelet concentrates	Immediately	Within 20 min
Fresh frozen plasma	Within 30 min	Within 20 min

The blood bag which has been kept for >4 h at room temperature or pack that has been opened or shows any sign of deterioration should be discarded.

Cannulae for Transfusion

- Sterile
- Flexible plastic cannulae (safe and preserve veins)

Blood Transfusion Set

- Should have 170–200 μm filter (which removes clots and small clumps of debris that may form during collection and storage).
- Should be changed every 12 hours during blood transfusion or after administration of two to four units of red blood cells.

- Rapid and under pressure transfusion through a small IV access can lead to haemolysis.
- Never use an administration set used for red cells, subsequently for platelet transfusion since platelets will adhere to fibrin captured in the filter.
- Don't add any medication to any blood component prior to its transfusion.
- Blood products must not come in contact with incompatible solutions like 5 % dextrose, ringer lactate, etc.
- In case of transfusions through central line, medications or solutions should be given through separate lumen without damaging the blood product,
- Don't warm blood components in hot water, in a microwave or on a radiator.

Need for Warming of Blood before Transfusion

- In elective transfusions, there is no need to warm blood as blood is usually transfused over 2–4 h.
- Warming of blood is required in following situations:
  - Large-volume rapid transfusions are required because cold components can lead to hypothermia and, hence, cardiac complications and morbidity and mortality.
  - If clinically significant cold agglutinins are present.
- Blood should be warmed in only blood warmers which have visible thermometer and audible warning alarm.

Criteria for Transfusion of Red Blood Cells: In Chronic Anaemia

A. *Duration of pregnancy less than 36 weeks*
   1. Hb 5.0 g/dl or below, even without clinical signs of cardiac failure or hypoxia
   2. Hb between 5.0 and 7.0 g/dl and in the presence of the following conditions:
      - Established or incipient cardiac failure or clinical evidence of hypoxia
      - Pneumonia or any other serious bacterial infection
      - Malaria
      - Pre-existing heart disease

B. *Duration of pregnancy 36 weeks or more*
   1. Hb 6.0 g/dl or below
   2. Hb between 6.0 g/dl and 8.0 g/dl and in the presence of the following conditions:
      - Established or incipient cardiac failure or clinical evidence of hypoxia
      - Pneumonia or any other serious bacterial infection
      - Malaria
      - Pre-existing heart disease

If the Hb is 7–8 g/dl in postnatal period, where there is no continuing or threat of bleeding, the decision to transfuse should be made on an informed individual basis. In fit, healthy, asymptomatic patients, there is little evidence of the benefit of blood transfusion.

Indications of Blood Transfusion in Acute Blood Loss

(a) Estimated or anticipated blood loss >15 % of total blood volume
(b) Diastolic blood pressure <60 mmHg
(c) Systolic blood pressure decrease >30 mmHg
(d) Oliguria/anuria
(e) Tachycardia (>100 beats/min)
(f) Mental status changes
(g) Shortness of breath, light headedness or dizziness with mild exertion
   - Blood loss of greater than 30 % of blood volume causes significant clinical symptoms, but resuscitation with crystalloid alone is usually successful in young healthy patients with blood loss of up to 40 % of blood volume.
   - Start volume replacement with up to 2 l of crystalloid. Follow plasma expanders until the blood is available.

Criteria for Transfusion of Platelets

1. Recent platelet count <10,000/L (for prophylaxis in stable, non-febrile patient)
2. <20,000/L for prophylaxis with fever or instability

3. Recent platelet count <50,000/L: Risk of haemorrhage or rapidly falling platelet count
4. Platelet dysfunction (prolonged bleeding time >1.5× the upper limit of normal) with:
   (a) Petechiae
   (b) Purpura
   (c) Bleeding
   (d) Invasive or surgical procedure

Criteria for the Transfusion of Fresh Frozen Plasma

- PT and/or APTT > the upper limits of normal
- Those patients with a suspected coagulation deficiency (PT/PTT pending) who are bleeding or at risk of bleeding from an invasive procedure
- Massive transfusion (>1 blood volume in 24 h)
- Disseminated intravascular coagulation
- Warfarin therapy
- Vitamin K deficiency
- Liver disease

Before Transfusion

- Prior to the administration of blood or blood components, the indications, risks and benefits of a blood transfusion and possible alternatives must be discussed with the patient and documented in the medical record.
- Take informed consent.
- Check the identification of the patient.
- Name, registration no, blood group and unit number on the reaction form and the labels on the bags should tally in addition to records in patient's file.
- Check the expiry date of blood component (written on the bag whole blood/packed RBCs).
- Pretransfusion vitals should be recorded in reaction form and in the patient's file.
- Transfuse within 30 min of the issue from blood bank.
- In patients with an elevated temperature, give an antipyretic and give time to have an effect; postpone the transfusion as long as possible.

Blood Bag Should Be Checked

- At time of release from blood bank
- On reaching the ward/operation theatre
- At time of start of transfusion

Blood Should Not Be Transfused If

- There is leakage from blood bag.
- Plasma appears pink *indicating* haemolysis.
- RBCs appear purple/black *indicative* of contamination.
- Clots are present in the blood bag which indicates that anticoagulant was not *mixed* properly.
- There are signs of *haemolysis* at *line* of red blood cells and plasma.

Final identity check was made at patient's bedside.

During Transfusion

Monitoring the transfused patient
1. For each unit of blood transfused, monitor the patient at the following stages:
Before starting the transfusion
As soon as the transfusion is started
15 min after starting transfusion
At least every hour during transfusion
On completion of the transfusion
4 h after completing the transfusion
2. At each of these stages, record the following information on the patient's chart:
Patient's general appearance
Temperature
Pulse
Blood pressure
Respiratory rate
Fluid balance
Oral and IV fluid intake
Urinary output
3. Record:
Time the transfusion is started
Time the transfusion is completed
Volume and type of all products transfused
Unique donation number of all products transfused
Any adverse effects
4. Monitor the patient carefully particularly during the first 15 min of the transfusion to detect early signs and symptoms of adverse effects

Instructions should be given to patient when possible regarding the possible symptoms of reaction, and be told to inform if any of symptom occurs:

- Hives or itching
- Fever with or without chills or rigors
- Back pain
- Pain at infusion site
- Difficulty in breathing
- Palpitations

Key Points for Transfusion of FFPs, Platelets and Cryoprecipitates

- Should not be given only on the basis of clinical suspicion unless there is delay in obtaining results of blood counts and coagulogram.
- FFPs and cryoprecipitates should ideally be of same blood group as that of recipient. But if unavailable, FFPs of different blood group can be given provided the unit does not have high anti-A or anti-B activity.
- Anti-D prophylaxis is not required if Rh D-negative women receive Rh D-positive FFPs or cryoprecipitates.
- Platelet should be of compatible group.
- Anti-D should be given to Rh-negative woman who receives Rh-positive platelets.
- rFVIIa is an option after discussing with the haematologist in case of intractable haemorrhage.

Transfusion Reactions

All suspected transfusion reactions should be reported to the blood bank.

- Transfusion reactions may be acute or delayed reactions.
- Acute reactions occur within 24 h of start of transfusion and delayed reactions occur days, months after transfusion.
- Acute reactions may occur in 1–2 % of transfused patients.

Investigation of Acute Transfusion Reactions

- Report all the transfusion reactions immediately to blood bank.
- Call for help – Anaesthetist, duty doctors' team, seniors and blood bank officials.
- Record in the patient's file about:
  - Type of transfusion reaction
  - Time of reaction after start of transfusion
  - Type and amount of blood/ blood product transfused
  - Details of the blood bag transfused like unique donation no., bag no., blood group and expiry date
- Verify the patient's identity with compatibility label on the blood bag and compatibility report.
- Take samples immediately and send for testing. One 'EDTA' and one clotted blood sample to be taken from the vein opposite from the site of transfusion. Send samples for:
  - Haemogram
  - Coagulogram
  - RFTs – Urea, creatinine
  - DCT
  - Electrolytes
  - Plasma haemoglobin, bilirubin
  - Urine sample for haemoglobinuria
  - Blood culture of patient and blood bag to be sent
  - Patient's 24 h urine sample to look for haemolysis

Similar samples are to be sent 12 and 24 h after reaction.

- Remaining blood/blood products along with transfusion set should not be discarded and sent to the blood bank for testing.

Complete Transfusion Reaction Form

- Complete the transfusion reaction form and send form along with the blood bag to the blood bank.
- Record results of all investigations in patient's record form and also in discharge for future reference.

Type of Transfusion Reactions and Their Management

Category 1: Mild

- *Caused* by release of histamine to proteins in donor plasma.
- *Symptoms* – Pruritis.
- *Signs* – Urticaria, rash.
- *Management*
  1. Slow the transfusion.
  2. Administer antihistaminic IM (chlorpheniramine 0.1 mg/kg or equivalent).
  3. Continue the transfusion at normal rate if there is no progression of symptoms after 30 min.
  4. If no clinical improvement within 30 min or if signs and symptoms worsen, treat as Category 2.
- *Prevention* – Antihistamine chlorpheniramine 0.1 mg/kg IM or IV can be given 30 min prior to the start of transfusion if there is a past history of allergic reactions.

Category 2: Moderately Severe

Category 2 transfusion reactions *are caused by cytokines* released from leucocytes in stored blood components or by infused white cells with antibodies in patient's plasma resulting in the release of pyrogens.

- *Symptoms* – Anxiety, pruritis, difficulty in breathing, palpitations and headache.
- *Signs* – Flushing, urticaria, rigors, fever, restlessness and tachycardia.
- *Management*:
  1. Stop the transfusion. Replace the infusion set and start normal saline.
  2. Notify the blood bank immediately.
  3. Send blood unit with infusion set, freshly collected urine and new blood samples (one clotted and one anticoagulated) from the vein opposite to the infusion site with appropriate request form to the blood bank and laboratory for investigations.
  4. Administer antihistamine IM (e.g. chlorpheniramine 0.1 mg/kg or equivalent) and antipyretic.
  5. Give IV corticosteroids and bronchodilators if there are anaphylactoid features (e.g. bronchospasm, stridor).
  6. Collect 24 h urine for evidence of haemolysis.
  7. If clinical improvement, restart transfusion slowly with new blood unit and observe carefully.
  8. If no clinical improvement within 15 min or if signs and symptoms worsen, treat as Category 3.
- *Prevention of Category 2*:
  - In patients who are regular transfusion recipients or who have past history of febrile non-haemolytic reactions.
  - Give antipyretics 1 h before starting transfusion and repeat after 3 h after starting transfusion.
  - Transfuse blood/blood products slowly, i.e. whole blood/red blood cells over 3–4 h per unit and platelet concentrates up to 2 h per concentrate.
  - Keep patient warm.
  - If still, febrile reaction is not controlled, use filtered red cells/platelet concentrates to remove the leucocytes.

Category 3: Life-Threatening

It includes various types of serious life-threatening reactions:

- Acute intravascular haemolysis
- Bacterial contamination and septic shock
- Fluid overload
- Anaphylactic reactions
- Transfusion-associated lung injury

Symptoms – Anxiety, chest pain, loin pain, shortness of breath, pain near infusion site and headache.

Signs – Fever, rigors, tachycardia, tachypnoea, restlessness, hypotension, haemoglobinuria and features of DIC.

Management:

1. Stop the transfusion. Replace the infusion set and keep IV line open with normal saline.

2. Infuse normal saline (initially 20–30 ml/kg) to maintain systolic BP. If hypotensive, give over 5 min and elevate patient's legs.
3. Maintain airway and give high-flow oxygen by mask.
4. Give adrenaline (as 1:1,000 solution) 0.01 mg/kg body weight by slow intramuscular injection.
5. Give IV corticosteroids and bronchodilators if there are anaphylactoid features (e.g. bronchospasm, stridor).
6. Give diuretic: furosemide 1 mg/kg IV.
7. Send blood unit with infusion set, fresh urine sample and new blood samples (one clotted and one anticoagulated) from the vein opposite to the infusion site with appropriate request form to the blood bank and laboratory for investigations.
8. Assess for bleeding from puncture sites or wounds. If there is clinical or laboratory evidence of DIC, give blood products.
9. Start inotropes if required.
10. If urine output is decreasing or laboratory evidence of acute renal failure is present (rising K+, urea, creatinine):
    - Maintain fluid balance accurately.
    - Give further furosemide.
    - Consider dopamine infusion, if available.
    - Seek expert help: The patient may need dialysis.
11. If bacteraemia is suspected (rigors, fever, collapse), start broad spectrum IV antibiotics, to cover pseudomonas and gram-positives.

Severe Transfusion Reactions in Detail

Acute Intravascular Haemolysis

Acute intravascular haemolysis is mainly due to incompatible blood transfusion usually being ABO incompatibility, other being rare. Antibodies present in the patient's plasma can haemolyse the incompatible transfused red cells.

The major reasons behind incompatible blood transfusion are:

- Incorrect filling of blood request form
- Wrong blood sample, i.e. sample from wrong patient was sent for compatibility testing
- Wrong labelling of sample vial
- Inadequate checking of blood/blood products prior to transfusion

Symptoms
- Pain in the limb where cannula was inserted
- Loin pain
- Apprehension
- Nausea, vomiting
- Cola-coloured urine

Signs – Fever, rigors, tachycardia, hypotension, breathlessness, haemoglobinuria, oliguria and features of DIC

Treatment – Same as Category 3

Prevention
- Complete and correctly filled blood request forms should be sent to the blood bank.
- Labelling of vial should be correctly done.
- Identity check is a must before transfusion.

Bacterial Contamination and Septic Shock

- Affects 0.4 % of red cells
- 1–2 % of platelet concentrates

Causes

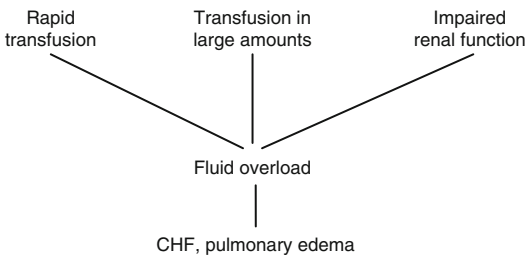
Contamination of blood by any of the following:

- Bacteria from donor's skin during collection of blood
- If donor is having bacteraemia at time of donation
- During processing of the blood
- Damaged blood bag
- During thawing of FFPs/cryoprecipitates

Risk of bacterial growth increases with duration the blood units remain out of refrigerator.

- The main organisms involved are pseudomonas and staphylococcus. *Pseudomonas* can grow at 2–6 °C and *Staphylococcus* grows at 20–24 °C in platelet concentrates.
- Symptoms and signs – Fever, rigors, hypotension, nausea, vomiting, diarrhoea and dyspnoea
- *Treatment*
  Supportive
  IV antibiotics

Fluid Overload



Occurs especially in patients with chronic severe anaemia and heart disease

Treatment
  Supportive
  Diuretics

Anaphylaxis

- Rare but serious transfusion reaction
- Occurs secondary to cytokines in plasma/Ig A deficiency
- Occurs within minutes of starting transfusion
- Presents as pruritis, urticaria, flushing, angioedema, hoarseness, stridor, wheezing, chest tightness, cyanosis, nausea, vomiting and diarrhoea
- It may manifests as cardiovascular collapse, respiratory failure but no fever
- Treated as Category 3

Transfusion-Related Acute Lung Injury (TRALI)

Caused by antibodies in the donor plasma against patient leucocytes.

- Occurs within 1–4 h of starting transfusion.
- Manifests as respiratory distress, tachycardia, fever, hypotension.
- Opacity is seen on chest X-ray.
- There is no specific treatment.
- Intensive respiratory and general supportive measures are required.

Delayed Complications of Transfusion

Can occur days, months or years after transfusion

Transfusion-transmitted infections
- HIV-1 and HIV-2
- Hepatitis B and C
- HTLV-I and HTLV-II
- Chagas disease
- Malaria, syphilis
- Cytomegalovirus
- Other rare infections: human parvovirus B19 and hepatitis A

Other delayed complications

- Delayed haemolytic reaction
- Posttransfusion purpura
- Graft-vs.-host disease
- Iron overload (in patients who receive repeated transfusions)

Table showing delayed transfusion reactions

Complication	Presentation	Treatment
Delayed haemolytic reactions	5–10 days posttransfusion:	Usually no treatment
	Fever	If hypotension and oliguria occurs, treat as acute intravascular haemolysis
	Anaemia	
	Jaundice	
Posttransfusion purpura	5–10 days posttransfusion:	High-dose steroids
	Increased bleeding tendency	High-dose intravenous immunoglobulin
	Thrombocytopenia	Plasma exchange
Graft-VS-host disease	10–12 days posttransfusion:	Supportive care
	Fever	No specific treatment
	Skin rash and desquamation	
	Diarrhoea	
	Hepatitis	
	Pancytopenia	
Iron overload	Cardiac and liver failure in transfusion-dependent patients	Prevent with iron-binding agents: desferrioxamine

Suggested adult flow rate				
Component	First 15 min	After 15 min	Special considerations	ABO compatibility
Red blood cells (RBCs)	1–2 ml/min (60–120 ml/h)	As rapidly as tolerated approximately 4 ml/min 240 ml/h	Infusion should not exceed 4 h	Whole blood: ABO identical
			For patients at risk of fluid overload, adjust flow rate to 1 ml/kg/h	RBCs: ABO compatible with recipient's donor
				Crossmatch required
Platelets	2–5 ml/min (120–130 ml/h) during the first 5 min	300 ml/h or as tolerated (after the first 5 min)	Generally given over 1 h	Crossmatch not required
				ABO/Rh compatibility preferable but not required
				May be HLA matched
Plasma	2–5 ml/min (120–130 ml/h) during the first 5 min	As rapidly as tolerated (after the first 5 min): approximately 300 ml/h	Thaw time may be needed before issue	Crossmatch not required
				ABO compatibility with recipient red cells

Documentation of Transfusion

Documentation of transfusion is a must to safeguard yourself from the medicolegal issues.

The following points should be noted in the patient record sheet:

- It is a must to note that patient and her relative have been informed about the transfusion in their own language.
- Reason for transfusion.
- Signature of the clinician who advised the transfusion.
- Pretransfusion check of:
  - Patient's identity
  - Blood pack
  - Compatibility label
  - Name and signature of person performing the pretransfusion check
- About the transfusion:
  - Type and volume of each unit transfused
  - Unique donation no. of each unit transfused
  - Blood group of each unit transfused
  - Time of start of transfusion of each unit
  - Monitoring records before, during and after transfusion of each unit
  - Signature of person who transfused the unit
- Any transfusion reaction.

Haemovigilance

The National Haemovigilance Programme of India was launched in December 2012 by National Institute of Biologicals, Noida, Ministry of Health and Family Welfare, Government of India, with the aim of tracking the adverse events related to transfusion and helps to identify the trends and to recommend the interventions required to improve the blood safety in the country.

Conclusion

Blood transfusion is a multistep process requiring expertise at different levels; hence a close working relationship is needed between clinicians and blood bank staff to ensure the blood safety. Proper documentation along with identity checks at different levels is required not only for patient safety but also for safeguarding yourself from the medicolegal issues. Hence ensure the right patient is getting right product in the right amount at the right rate at the right time.

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Fluid and Electrolyte Balance in Critically Ill Obstetric Patient

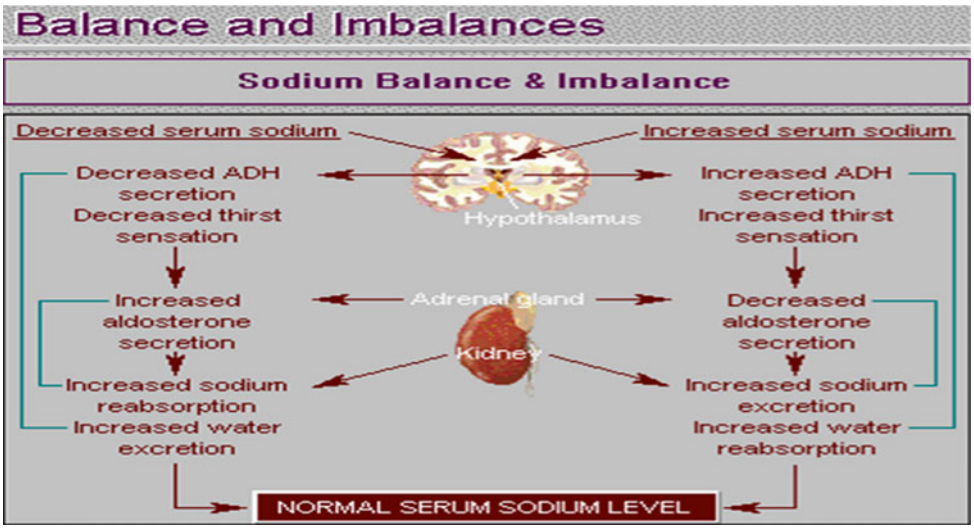
13

Ruchika Garg and Rekha Rani

Introduction

A variety of physiologic cardiovascular changes occur during pregnancy, and these must be considered when managing volume status in complicated obstetric patients. Beginning in early pregnancy, total body water slowly increases by 6–8 l due to retention of an additional 500–900 mEq of sodium [1–3]. This leads to a steady rise in plasma volume throughout the first two trimesters and into the early third, with a plateau at approximately 32 weeks [4]. In a singleton pregnancy at term, the plasma volume is nearly 50 % greater than that seen in the nonpregnant individual [5]. A smaller but parallel increase occurs in red cell mass, with a resultant physiologic fall in haemoglobin concentration [4].

Balance and Imbalance



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Maternal cardiac output starts to increase at approximately 10 weeks and plateaus near the end of the second trimester at levels 30–50 % higher than nonpregnant values [6–10]. This results from an increase in both stroke volume and heart rate. In the third trimester, heart rate reaches a peak of 10–15 beats/min over baseline [10]. Systolic and diastolic blood pressures fall throughout the first two trimesters, reaching a nadir at 24–28 weeks before increasing to nonpregnant levels at term [11].

The systolic pressure decreases an average of 5–10 mmHg, while diastolic pressure falls 10–15 mmHg [12]. Blood pressure and cardiac output may be further affected by maternal posture. Late in pregnancy, the gravid uterus can mechanically obstruct the aorta and vena cava while supine [13, 14]. In addition, the changes in cardiac output and blood pressure result in an initial decrease in systemic vascular resistance (SVR), followed by a rise towards nonpregnant values near term [11].

Colloid oncotic pressure (COP) is another important variable affected by pregnancy. Both plasma and interstitial COP fall throughout gestation, the latter decreasing to a greater extent [15]. There is an accompanying increase in capillary hydrostatic pressure [16]. An increase in hydrostatic pressure or decrease in plasma COP may overcome the delicate balance and favour oedema formation in late pregnancy. After delivery, there is a further fall in plasma COP, reaching a nadir between 6 and 16 h and returning towards intrapartum levels after 24 h [17, 18].

The described physiologic changes seen in pregnancy affect both the assessment of a pregnant patient's volume status as well as subsequent treatment.

Parameter Change Impact on Resuscitative Care

Plasma volume ↑ 50 % dilutional anaemia

- ↓ O2-carrying capacity
- Enhanced physiological reserve against haemorrhage
- Heart rate ↑ 15–20 bpm
- Chest compressions during CPR likely to be less effective as demand is higher
- Cardiac output ↑ 40 %
- Arterial blood pressure ↓ 10–15 % ↓ physiological reserve
- Uterine blood flow accounts for 10 % of cardiac output at term
- Potential for massive blood loss
- Cardiac anatomy; heart rotated cephalad and to the left
- ↑ Chamber size, particularly the left atrium

Predisposition to cardiac dysrhythmias, especially supraventricular tachycardia

Haemorrhage

Haemodynamic Compromise

Haemodynamic compromise is a common indication for ICU admission in the obstetric population manifesting as hypotension, hypertension or more rarely as a cardiac dysrhythmia.

Causes of maternal hypotension include:

- Obstetric haemorrhage (particularly postpartum)
- Sepsis
- Peripartum cardiomyopathy
- Amniotic fluid embolism
- Pulmonary embolism
- Uterine rupture
- Epidural/spinal anaesthetic

The reduction in blood pressure in pregnancy is predominantly secondary to a decrease in the diastolic component which is reflective of the progesterone-stimulated reduction in systemic vascular resistance and the development of the placenta, a low-resistance vascular bed. The increased cardiac output that develops in pregnancy is further augmented during the third stage of labour (delivery of the placenta) as a result of autotransfusion of blood from the utero-placental to maternal circulation as the uterus contracts. Relief of aorto-caval

compression also increases preload. The utero-placental vascular bed under normal circumstances is maximally dilated.

Resuscitation of the Haemodynamically Compromised Patient

The priority in resuscitation is to optimise and maintain maternal cardiac output and preserve adequate tissue and placental perfusion, establish large-bore intravenous access and send blood for:

- Complete blood count
- Urea, creatinine and electrolytes
- Liver function tests
- Acid-base analysis
- Coagulation screen
- Group/type and crossmatch

Fluid therapy in combination with vasoactive and/or inotropic agents is central to the initial correction of haemodynamic compromise in the critically ill obstetric patient. The colloid oncotic pressure in the obstetric patient is reduced approximately 14 % from the nonpregnant state. Overzealous fluid loading in obstetric patients, particularly those with pre-eclampsia, can precipitate pulmonary oedema due to leaky capillaries. The best fluid for resuscitation will depend on the cause of haemodynamic instability. Major haemorrhage generally requires replacement with blood products, while other causes of shock will require judicious use of either a crystalloid or colloid solution or a combination of both. In general, critically ill obstetric patients are probably better off with a slightly negative volume status given the potential deleterious effects of fluid overload and noncardiogenic pulmonary oedema. *They may tolerate a negative volume status better given their lack of significant comorbidities*. Vasopressors are commonly used in obstetrics particularly following spinal or epidural anaesthesia for caesarean section. Ephedrine and phenylephrine are the most commonly used agents to offset the effects of sympathetic blockade. Phenylephrine causes less foetal acidosis than ephedrine. If vasopressors are required in a critical care setting, the choice of agent should be determined by maternal mean arterial pressure, systemic vascular resistance and cardiac output and should follow appropriate fluid loading. All vasopressor agents may have deleterious effects on utero-placental perfusion. As in all patients with haemodynamic compromise, maternal response to therapy and clinical status in terms of tissue perfusion should be continuously re-evaluated.

Basic indicators of tissue perfusion include:

- Level of consciousness (Glasgow coma score)
- Vital signs
- Urine output
- Acid-base status and lactate concentration

Other monitors which may be considered include:

- Minimally invasive devices (e.g. trans-oesophageal echocardiography, oesophageal Doppler or pulse contour analysis).
- Invasive monitoring (e.g. central or mixed venous oxygen saturation).
- Noninvasive assessments (e.g. transthoracic echocardiography which is not appropriate for continuous monitoring but valuable for the assessment of global cardiac function). Assessment of the utero-placental-foetal unit (foetal well-being) is an important guide to adequacy of tissue perfusion and resuscitation.

Haemorrhage commonly complicates childbirth and remains a major cause of maternal mortality [19]. Blood transfusion secondary to hemorrhage during gestation is relatively common, occurring in 1 % to 2 % of pregnancies. Although uterine atony is the leading cause of significant bleeding requiring transfusion, retained placenta, trauma, placenta previa and abruptio placentae are also important aetiologies [20]. In cases of postpartum haemorrhage, visual estimations can be in error by as much as 50 % when compared to quantitative measures [21–23]. This results in a large underestimation of the prevalence of postpartum haemorrhage [24]. Additionally, normal vital signs do not necessarily preclude the presence of significant blood loss [25]. The above factors may lead to a delay in diagnosis with an associated delay in therapy.

The previously described normal physiologic changes that occur during gestation allow most pregnant patients to tolerate the inevitable blood

loss associated with delivery. In early haemorrhage, vascular tone increases, as do heart rate and myocardial contractility, to improve oxygen delivery. Cardiac output is redistributed, selectively maintaining perfusion to the adrenal glands, brain and heart at the expense of other organs, including the uterus.

Before delivery, this shunting may lead to foetal hypoxia and distress. With continued blood loss and delayed or inadequate resuscitation, secondary changes occur in the microcirculation. Initially, interstitial fluid enters capillary beds. Later, capillary endothelial damage occurs, resulting in an increased permeability and leakage of fluid back into the interstitial space. Finally, organ ischemia and cell death result [26].

The primary goal of therapy is to restore and maintain tissue oxygen delivery. This begins with aggressive replacement of intravascular volume. At the same time, supplemental oxygen should be added.

Lactated Ringer's and 0.9 % sodium chloride (normal saline) are the two most common crystalloid solutions. They distribute primarily throughout the extracellular space, expanding both the intravascular and interstitial compartments. With the infusion of 1 l of lactated Ringer's solution, 200 ml will remain in the vasculature while 700 ml enters the interstitium [17].

Roughly 3 Litres of Crystalloid Are Required for Each Litre of Blood Loss

Lactated Ringer's has the advantage of containing small quantities of additional electrolytes and lactate.

The lactate is converted to bicarbonate by the liver. In theory, the bicarbonate may buffer some of the lactic acidosis produced from poor perfusion and offset expansion acidosis resulting from dilution of existing buffers (Table 13.1).

Lactated Ringer's is recommended by the American College of Surgeons as the initial fluid for resuscitation. Normal saline is a secondary choice because of the potential for hyperchloraemic acidosis [27].

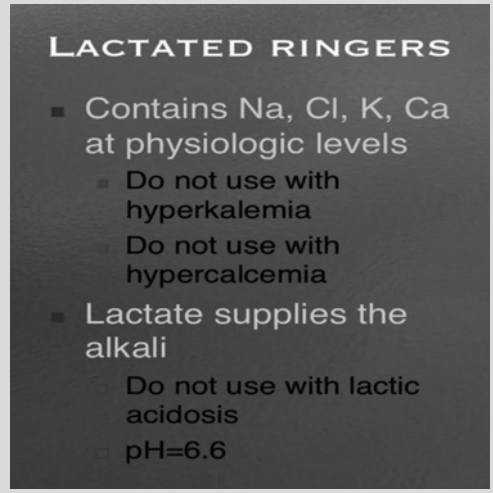

Table 13.1 Composition and properties of crystalloid solutions

Solution	pH	Na^+(mEq)	Cl^-(mEq)	mOsmL
5 % dextrose	5			253
Normal (0.9 %) saline	6.1	154	154	308
Lactated Ringer's[a]	6.7	130	109	274
3 % sodium chloride	5.8	513	513	1026
7.5 % sodium chloride	5.7	1283	1283	2567

[a]Also contains K^+ (4 mEq); Ca^{2+} (3 mEq), and lactate (28 mEq), which is converted to HCO_3^-

Colloid solutions may be used as an alternative or an adjunct to crystalloids. Colloids are solutions containing large-molecular-weight substances. Their distribution is limited primarily to the intravascular space; thus, intravascular volume is expanded with little increase in the interstitial volume. All of these solutions have been associated with *anaphylaxis*, but the incidence is low (less than 0.04 %).

Table 13.2 Characteristics of colloid solutions

Solution	pH	mOsmL	Duration of action (h)
5 % albumin	6.9	300	2
6 % hetastarch	5.5	310	12–24
Dextran 40	4.5	300	<3
Dextran 70	4.5	300	6
5 % plasma protein fraction	7	300	2

Albumin in a 5 % solution is the colloid most commonly used for volume expansion. Adverse effects and decrease in free calcium levels may be due in part to citrate binding. There is also a decrease in platelet aggregation as well as dilution of clotting factors, which may lead to prolongation of the prothrombin time (PT) and partial thromboplastin time (PTT) (Table 13.2).

Preparation of the 5 % albumin kills HIV and hepatitis B and C viruses, thus eliminating the risk of infection with these agents. Plasma protein fraction consists primarily of albumin with lesser amounts of alpha-and beta-globulins. Hetastarch is derived from cornstarch and contains molecules of varying sizes. The

plasma-expanding effects are long-lasting. Its use may be associated with a rise in amylase levels during the first 24 h. An increased bleeding time may be seen as a result of increased fibrinolysis, decreased platelet adhesion and decreased factor VIII activity.

Dextrans consist of high- and low-molecular-weight preparations, dextran 70 and dextran 40, respectively. These solutions have been associated with bleeding due to decreased platelet adhesion and dilution of clotting factors [19].

Considerable controversy exists as to whether crystalloids or colloids are the optimal fluid for initial resuscitation in hypovolaemic shock. Crystalloids require considerably larger infused volume than colloids to obtain a comparable level of volume expansion.

Fluid and Blood Component Therapy in Obstetric Patients

Fluid prescriptions are very important. Prescribing the wrong type or amount of fluid can do serious harm. Assessment of fluid requirements needs care and attention, with adjustment for the individual patient.

The volume infused should be titrated against clinical parameters like:

1. Blood pressure
2. Peripheral capillary filling
3. Urinary output
4. Central venous pressure

In the intensive units, other indices like oxygen delivery and consumption indices and mixed oxygen saturation levels may be used to optimise fluid management.

- *Urine output is a poor guide to fluid requirements in sick patients, and oliguria does not always require fluid therapy (full assessment is required)* [28].
- *Maintenance requirement: 30 ml/kg/24 h of 'water'.*
- *It is vital that sick patients receive the right amount of the right fluid at the right time.*
- *Euvolaemic: veins are well filled, extremities are warm, and blood pressure and heart rate are normal.*
- *Hypovolaemic: Patient may have cool peripheries, respiratory rate>20, systolic BP<100 mmHg, FEWs ≥5, HR>90 bpm, postural hypotension, oliguria and confusion. History of fluid loss or low intake.*
- *May respond to 45° passive leg raise.*
- *Catheterise.*
- *Replacement of losses, either previous or current. If losses are predicted, it is best to replace these later rather than give extra fluid in anticipation of losses which may not occur. This fluid is in addition to maintenance fluid.*
- *Check blood gases.*
- *Resuscitation:*
  The patient is hypovolaemic as a result of dehydration, blood loss or sepsis and requires urgent correction of intravascular depletion to correct the deficit. Obtain weight (estimate if required). Maintenance fluid requirement approximately 30 ml/kg/24 h.
- *Review recent U&Es, other electrolytes and Hb.*

Maintenance Fluid

IV fluid should be given via volumetric pump if a patient is on fluids for over 6 h or if the fluid contains potassium.

Always prescribe as ml/hr not 'x hourly' bags.

Never give maintenance fluids at more than 100 ml/h.

Do not 'speed up' bags; rather give replacement for losses.

Weight kg	Fluid requirement	ml/24 h	Rate ml/h
35–44	1200	(500 ml '10 hourly')	50
45–54	1500	('8 hourly')	65
55–64	1800	('7 hourly')	75
65–74	2100	('6 hourly')	85
≥75 (100 ml per hour max)	2400	('5 hourly')	100

Preferred maintenance fluids: 0.18 %NaCl/4 % glucose with or without added potassium (20 mmol) in 500 ml. One litre bags are available. This fluid *if given at the correct rate* provides all water and Na+/K+ requirements until the patient can eat and drink or be fed. Excess volumes of this fluid (or any fluid) may cause hyponatraemia.

If serum sodium is ≤132 mmol/l, use PL148 for maintenance.

Electrolyte Requirements

- Sodium 1 mmol/kg/24 h (approx. 1 × 500 ml 0.9 %NaCl)
- Potassium 1 mmol/kg/24 h (give 20 mmol/500 ml bag)

A sodium of <125 mmol/l is dangerous (0.9 %NaCl or fluid).

- Restriction is the first-line treatment and frequent U&Es are required.

Potassium Maintenance and Replacement

A normal potassium level does not mean that there is no total body potassium deficit. Give potassium in maintenance fluid. Only in critical care areas give up to 40 mmol in 100 ml bags via a central line at 25–50 ml/h. Ensure IV cannulae are patent and clean.

Potassium-containing fluids *must* be given via a pump.

Give K orally if possible.

Estimate replacement fluid/electrolyte requirements by adding up all the losses over the previous 24 h and give this volume as PlasmaLyte 148 (PL148). Use 0.9 %NaCl with KCl for upper GI or bile loss (high NaCl content). Otherwise, avoid it as it causes fluid retention. Diarrhoea may lead to potassium loss.

- Fluid

Fluid content/l	Na	K	CI	Mg	Ca	mmol/l	Osm
0.9 %NaCI	154	0	154	0	0	0	308
0.18 %NaCl	30	0	30	0	0	Gluc 40 g/l	284
4 % glucose ±K							
0.45 %NaCl	77	0	77	0	0	Gluc 50 g/l	406
5 % glucose							
Hartmann's	131	q5	111	0	2	Lactate 29	274
PlasmaLyte	140	5	98	1.5	0	Acetate 27	297
148 (PL148) gluconate 23							
5 % glucose	0	0	0	0	0	Gluc 50 g/l	278

Plasma osmolality is approximately 285–295 mosm/l
Osm osmolality

Resuscitation Fluid

For severe dehydration, sepsis or haemorrhage leading to hypovolaemia and hypotension.

For urgent resuscitation use PlasmaLyte 148 (PL148) or colloid (Gelaspan/Albumin). PL148 is a balanced electrolyte solution and is better handled by the body than 0.9 %NaCl.

Give albumin only in severe sepsis.

Use major haemorrhage protocol. Treat sepsis.

Call for help.

For severe *blood loss*, initially use colloid or PL148 until blood/clotting factors arrive. Use O negative blood for torrential bleeding. Severely *septic* patients with circulatory collapse may need inotropic support in a critical care area. Their blood pressure may not respond to large volumes of fluid; excessive volumes (many litres) may be detrimental.

In summary: assess why, how much, which fluid:

- Take time and consult senior if you are unsure.
- Patients on IV fluids need regular blood tests.
- Patients should be allowed food and drink ASAP.

Consider critical care referral if:

- GCS ≤8 or falling from higher levels
- O2 saturation<90 % on 60 % O2 or higher
- PaCO2 >7 kPa unresponsive to NIV
- Persistent hypotension and/or oliguria unresponsive to 2000 ml fluid/or concern over cardiac function
- Metabolic acidosis: base deficit -8 or worse, bicarbonate <18 mmol/l, lactate >3 mmol/l and not improving in 2 h
- Aggressive/agitated patients whose treatment (e.g. oxygen/IV therapy) is compromised due to agitation

Referral is not always appropriate – consult senior doctor.

Fluid challenge algorithm hypovolaemia: low BP, tachycardia, low CVP/JVP, oliguria, reduced skin turgor, poor tissue perfusion, capillary refill time >4 s.

Patients with epidurals may need vasoconstriction rather than fluid but must be assessed for other causes of hypotension. Give 250–500 ml IV fluid challenge with PL148 or Gelaspan over 5–15 min adequate response.

NO
YES
YES

References: Southampton Fluid Guidance 2009

Theoretically, this excess volume distributed within the interstitial space may impair oxygen transport to cells. In addition, the duration of the effect is also shorter with crystalloid solutions.

Renal function is also a management concern in patients being treated for hypovolaemic shock. Both animal and human data suggest that crystalloids are better at preserving renal function than colloids.

Blood and Blood Components in Haemorrhage

In severe haemorrhage, the goal of restoring oxygen delivery to the tissues is only partially achieved by initial intravascular volume replacement and improved perfusion. Significant blood loss resulting in symptomatic decreased oxygen-carrying capacity requires blood transfusion. Blood component therapy has largely eliminated the use of whole blood in hypovolaemic shock.

Guidelines for Use of Blood Components

Component	Description	Indications	Dosage effect	Shelf life	Storage conditions
Packed red blood cells	300–350 ml of RBSs in CPD-A or CPD with additive solution collected from 450 ml of whole blood	Symptomatic anaemia acute or chronic for restoration of O_2-carrying capacity	One unit RBC increases Hct by 3 %; increases, Hb by 1 g% (approx.)	42 days	2–6 °C
Fresh frozen plasma (FFP)	200–250 ml, prepared within 6 h of whole blood collection, contains all clotting factors including labile factors (V and VIII)	Multiple coagulation factor deficiency, massive transfusion, liver disease, DIC, TTP	10–15 ml/kg increases factor levels by 20–30 %	Frozen for 1 year	–30 °C or lower Transfuse preferably within 6 h after thawing at 37 °C
Cryoprecipitate	10–25 ml	Haemophilia A, von Willebrand's disease, Hypofibrinogenaemia, F XIII deficiency	Factor VIII – 1 unit/kg increases level by 2 %	Frozen – 1 year	–30 °C or lower. Use preferably within 6 h after thawing at 37 °C

Component	Description	Indications	Dosage effect	Shelf life	Storage conditions
Random-donor platelet (RDP)	$>5 \times 10^{10}$ platelets/unit from 450 ml of blood 45–65 ml	Bleeding from thrombocytopenia, functional defect of platelets	Increases platelet count by 5000–10,000 per unit	5 days	20–24 °C with continuous agitation

Packed Red Blood Cells

Packed red blood cells are the component of choice for the treatment of hypovolaemic shock due to haemorrhage. Each unit can be expected to increase the haemoglobin by approximately 1 g/dl. Packed red blood cells should be given when oxygen-carrying capacity is inadequate.

Fresh Frozen Plasma (FFP)

It is the plasma portion of whole blood frozen within 8 h of collection. FFP is preserved for those patients with bleeding and prolongation of the PT and PTT. FFP should not be used for volume expansion. In case of massive blood transfusion, regular haemostatic monitoring has to be done. Many authorities recommend the addition of one unit of FFP for every five units of PRBC for the patients who require continuous transfusion.

Cryoprecipitate

Cryoprecipitate is the insoluble portion of FFP obtained when thawing between 1° and 6°. It contains factors VIII and XIII, von Willebrand's factor and fibrinogen because cryoprecipitate contains concentrated fibrinogen. It also may be beneficial in cases of consumptive coagulopathy. For isolated hypofibrinogenaemia, cryoprecipitate should be used rather than FFP.

Platelets

In the bleeding patients, transfusion is indicated when the platelet count falls below 50,000/mm cube. Platelets should not be given prophylactically with massive blood transfusion. The patient's clinical condition and serial platelet counts should guide for platelet therapy. Platelet counts can be expected to rise approximately by 5000–10,000/mm cube with each unit of platelet concentrate transfused.

Thrombocytopenia is likely after 1.5–2 times the blood volume has been replaced.

Transfusion-Related Morbidity

Transfusion-related morbidity can be significant, and it is one of the main reasons behind efforts to promote the effective use of blood products in the country.

Much of the remaining risk is due to the 'window period' between infection and development of detectable antibodies.

Currently, although risk of transfusion – transmitted infection – is low, it is not zero. Others include acute or delayed haemolytic reactions, alloimmunisation and allergic and febrile reactions.

Per Unit Risk of Transfusion-Related Infections and 'Window Period' Enter an Early Infection May Go Undetected

Infection	Per unit risk	Window (d)
HIV	1/450,000–660,000	20–45
HCV	1/3300	28
HBV	1/200,000	14–120
HTLV	1/50,000-70,000	Unknown

HBV hepatitis B virus, *HCV* hepatitis C virus, *HIV* human immunodeficiency virus, *HTLV* human T-cell lymphotropic virus

Management of Fluid Balance in Pre-eclampsia

Fluid management for the patient with pregnancy-induced hypertension (PIH) presents a challenge for the obstetrician. This is especially true for the subset of patients with pre-eclampsia.

In general, PIH is characterised by a failure to achieve the normal pregnancy-associated plasma volume expansion. Later in gestation, this is combined with manifestations of excess interstitial fluid. *The paradox of intravascular volume depletion combined with increased extracellular volume leads to divergent opinions regarding optimal fluid management. Volume replacement has been advocated by some because of the intravascular hypovolaemia, whereas others have recommended volume restriction and even diuretics because of the extracellular fluid excess*.

We suggest that management plans be tailored to the clinical situation. In uncomplicated PIH, a crystalloid infusion limited to between 75 and 125 ml/h throughout the intrapartum and early postpartum period (12–24 h) is appropriate. Clinical resolution will begin in most patients during this time with a brisk, spontaneous diuresis.

In more complicated cases, or when other therapeutic interventions are planned, the patient's intravascular volume status becomes an important management consideration.

Oliguria, pulmonary oedema, antihypertensive therapy and regional analgesia or anaesthesia are clinical settings where meticulous fluid management is important.

Oliguria-urine output <30 ml/h over a 2-h period. An initial fluid challenge of 500–1000 ml of crystalloid is indicated. If adequate urine output does not result, further therapy should be guided by careful assessment of oxygenation status and, if compromised, by invasive haemodynamic monitoring.

Complications such as pulmonary, cerebral or laryngeal oedema can result from overzealous volume infusion.

Oliguria due to selective renal arteriospasm. Improvement is seen with vasodilator therapy. In this clinical scenario, consideration could be given to low-dose dopamine, which causes selective renal artery vasodilation.

Intravascular fluid overload can occur due to decreased left ventricular function and vasospasm. Treatment consisted of fluid restriction and vasodilator therapy.

Haemodynamic Subsets of Oliguric Pre-eclamptic Patients

Group	Cardiac output	PCWP	SVR	Treatment
I	Increased	Low	Increased	Intravenous fluid
II	Normal/increased	Normal/increased	Normal	Afterload/preload reduction; consider dopamine
III	Decreased	Elevated	Increased	Afterload reduction and fluid restriction; consider diuretics

PCWP pulmonary capillary wedge pressure, *SVR* systemic vascular resistance

In patients requiring volume expansion, colloids would seem to be an ideal choice, considering the known early postpartum decrease in colloid osmotic pressure and the further decrease noted with the use of intrapartum intravenous fluids.

Pulmonary oedema associated with PIH occurs after delivery in 70–80 % of cases.

Postpartum mobilisation of interstitial fluids contributes to the occurrence of pulmonary oedema by increasing PCWP.

The associated increase in hydrostatic pressures, decrease in COP and increase in vascular permeability resulting from endothelial damage all combine to increase the risk of pulmonary oedema. Management consists of supplemental oxygen, fluid restriction, diuretics and possibly vasodilators to facilitate afterload reduction. Use of the pulmonary artery catheter in extremely complicated cases to aid therapy should be encouraged.

Fluid management in the pregnant patient with PIH is complex and should be tailored to the specific clinical situation. Careful fluid therapy in which intake and output are balanced is adequate for most uncomplicated cases. When complications arise or therapeutic interventions that lower

blood pressure are contemplated, volume expansion may be desirable. In particularly difficult cases, pulmonary artery catheterisation may help guide fluid therapy.

- Timely delivery.
- Strict fluid balance including hourly urometry.
- Avoid sharp falls in blood pressure (sublingual nifedipine and bolus IV oxytocin).
- Avoid simultaneous pharmacological interventions.
- Avoid β-mimetic tocolysis.
- Aviod ergometrine.
- Replace sudden blood losses promptly but carefully.
- Avoid nonsterodial anti-inflammatory agents.

NSAIDS should be avoided as these can induce sudden anuria in susceptible patients. Women with pre-eclampsia who lack the normal hypervolaemia of pregnancy are much less tolerant of blood loss at delivery. Acute tubular necrosis may be precipitated by acute hypovolaemia on the background of intravascular depletion, renal artery vasospasm and microangiopathy. It is hard to define what constitutes a significant obstetric haemorrhage in this context, but a loss of greater that 500 ml and/or that which causes tachycardia should be taken seriously.

Although prompt treatment of severe hypertension is thought to be essential to reduce the chance of intracerebral haemorrhage, overly rapid or vigorous treatment of hypertension, particularly with vasodilators, can lead to collapse requiring fluid resuscitation. This fluid can be later displaced into interstitial tissues as vasodilatation reverses and blood pressure rises. Sublingual nifedipine and bolus doses of hydralazine, in particular, are known to provoke sharp falls in blood pressure. This also applies to intravenous bolus doses of oxytocin (10 IU or more) which promote vasodilatation; oxytocin infusions are preferred if large doses are needed. Tocolysis with β-adrenergic agents is also contraindicated because of the risk of precipitating noncardiogenic pulmonary oedema.

Intravenous Fluid Therapy

Crystalloid or Colloid?

Because serum oncotic protein levels are low, many advocate the use of colloid infusions (synthetic products or human albumin solutions.) These do initially increase the colloid osmotic pressure. In contrast, crystalloid solutions dilute the oncotic proteins and reduce COP_P.

Use of albumin for fluid resuscitation in critically ill patients has been debated. The recent SAFE trial comparing use of human albumin versus saline solutions in intensive care found that there were similar outcomes in both groups. A systematic Cochrane review went further and stated that mortality was increased with use of albumin in critical care situations. There was insufficient evidence to determine the effects.

The value of albumin in the peripartum management of pre-eclampsia has not been tasted. In the absence of any proven benefit, with reason to believe it might be harmful, and because its cost is much greater, we advise that colloid should be avoided in favour of crystalloid solutions.

Hartmann's Solution or Normal Saline?

Plasma volume expansion is greater and more sustained with normal saline than with Hartmann's solution. Subjects infused with Hartmann's solution developed their diuresis more quickly and to a greater degree (1000 ml over 6 h compared with 450 ml).

Hartmann's solution might be more suitable for women with pre-eclampsia but specific research would be necessary.

The precise nature of a conservative fluid regimen varies from unit to unit. Some employ a simple formula such as a maximum of 2.51 in 24 h or 1 ml/kg/h, whereas others prefer to calculate the rate as previous hour's urinary output plus 40 ml or 1 ml/kg/h. Once a patient is able to tolerate oral intake, it is sensible to allow free oral fluids and decrease intravenous fluids steadily in line with this.

Management of High-Risk Cases

For these women who develop severe oliguria/anuria in the presence of HELLP, sepsis or haemorrhage, the risk of renal failure is much greater. Treatment with either continuous furosemide or renal dose dopamine infusion will promote a dieresis. But all-cause morbidity and mortality rates have not been reported as yet.

Management of Pulmonary Oedema

The patient should be sat upright and given oxygen by face mask. Furosemide should be given intravenously where there is evidence of fluid overloaded, but care must be taken to monitor for cardiovascular collapse if the intravascular compartment is depleted.

The action of furosemide is complex and includes reduction of pulmonary artery pressure as well as dieresis. In severe cases, continuous positive airway pressure (CPAP) ventilation may be beneficial as found in other causes of pulmonary oedema.

Septic Shock

Infections complicating pregnancy and the puerperium are common events. The incidence of bacteraemia associated with these infections is low and reported at less than 1 %. Septic shock may occur in up to 5 % of these bacteraemic patients. Mortality rates of 20–50 % are seen with septic shock. Nevertheless, prompt empiric initiation of antibiotics and appropriate fluid therapy are cornerstones in management of the pregnant patient, as they are in the nonpregnant population.

Infections Leading to Septic Shock and Associated Frequency in the Obstetric Patient

Site	Frequency of septic shock (%)
Postpartum endometritis (caesarean delivery)	85
Postpartum endometritis (vaginal delivery)	1–4
Urinary tract infection	1–4
Septic abortion	1–2
Chorioamnionitis	0.5–1
Necrotizing fasciitis	<1

The immediate therapeutic goal is to improve oxygen delivery and maintain perfusion to vital organs. Aggressive fluid replacement is the mainstay of early treatment and is aimed at eliminating hypovolaemia, improving hypotension and maintaining or increasing cardiac output. Often, large volumes of fluid are required.

Hypotension and cardiac function may not completely respond to fluid alone. It then becomes important to determine volume status objectively in order to prevent fluid overload, as well as to evaluate the need for vasopressor and inotropic agents.

Characteristics of Common Vasoactive Drugs

Drug[a]	Dosage	Blood pressure	Systemic vascular resistance	Cardiac output	Renal perfusion
Isoproterenol	1–5 μg/min	No change/decrease	Decrease	Increase	Increase
Dobutamine	2–20 μg/kg/min	Increase	Decrease	Increase	No change
Dopamine					
Low dose	1–10 μg/kg/min	No change	Decrease	Increase	Increase
High dose	>20 μg/kg/min	Increase	Increase	Increase	Decrease
Epinephrine	1–8 μg/min	Increase/decrease	Decrease	Increase	Decrease

Drug[a]	Dosage	Blood pressure	Systemic vascular resistance	Cardiac output	Renal perfusion
Norepinephrine	2–8 μg/min	Increase	Increase	No change/ increase	Decrease
Phenylephrine	20–200 μg/min	Increase	Increase	Decrease	Decrease

[a]Listed in order of increasing vasopressor activity and decreasing inotropic activity

Pulmonary Artery Catheter

Fluid management in most obstetric conditions consists of a peripheral venous catheter with monitoring of vital signs and fluid balance. In the complicated obstetric patient, however, the flow-directed pulmonary artery catheter can be a valuable clinical aid in the assessment and management of volume status. In the early 1970s, the Swan-Ganz catheter moved from the research laboratory into medical and surgical intensive care units to facilitate monitoring of the critically ill patient.

Now, use of the pulmonary artery catheter increased in the critically ill obstetric patient.

Indications for the Use of the Pulmonary Artery Catheter in Obstetric Patients

Indication

- Massive blood loss complicated by:
  - Respiratory compromise
  - Oliguria after clinically appropriate fluid replacement
- Pregnancy-induced hypertension complicated by:
  - Persistent oliguria unresponsive to fluid challenge
  - Pulmonary oedema of uncertain aetiology or unresponsive to conventional therapy
- Septic shock
  - Adult respiratory distress syndrome
  - Cardiac disease intrapartum or intraoperatively

Normal Values for Cardiorespiratory Parameters of Term-Pregnant and Nonpregnant Patients

Cardiovascular parameter	Nonpregnant	Pregnant
Heart rate (beats/min)	71 ± 10	17 % increase
Mean arterial pressure (mmHg)	86 ± 7.5	No change
Central venous pressure (mmHg)	3.7 ± 2.6	No change
Pulmonary capillary wedge pressure (mmHg)	6.3 ± 2.1	No change
Cardiac output (l/min)	4.3 ± 0.9	43 % increase
Systemic vascular resistance (dynes • cm • s^{-5})	1530 ± 520	21 % decrease
Pulmonary vascular resistance (dynes • cm • s^{-5})	119 ± 47	34 % decrease
Colloid oncotic pressure	20.8 ± 1	14 % decrease
Colloid oncotic pressure – pulmonary capillary wedge pressure gradient	14.5 ± 2.5	28 % decrease

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Mechanical Ventilation in Critically Ill Obstetric Patient

14

Mohammed Azam Danish

Introduction

Ventilating a critically ill obstetric patient is an art. There are many ventilators, and more importantly many modes are incorporated into the mechanical ventilator to confuse us more. It's reasonably important for us to have a basic understanding of the few basic and newer modes of ventilation.

Goals of Mechanical Ventilation [1]

- Reversal of apnea
- Reversal of respiratory distress
- Reversal of severe hypoxemia
- Reversal of severe hypercapnia

Indications of Mechanical Ventilation [2]

1. Eclampsia
2. Acute respiratory distress syndrome (ARDS; due to aspiration, amniotic fluid embolism, etc.)
3. Pulmonary edema
4. Severe cardiac failure
5. Exacerbation of respiratory disease like bronchial asthma
6. Major trauma

Terms Related to Mechanical Ventilation

Mechanical ventilation can be classified into positive-pressure ventilation and negative-pressure ventilation (Fig. 14.1):

- Noninvasive ventilatory strategies have not been extensively studied in the obstetric population, and there is a potentially greater risk of gastric aspiration [2].
- For patients with a reduced level of consciousness, a lack of respiratory drive, or a severe acidosis, noninvasive ventilation is unsuitable.
- Invasive mechanical ventilation is required for optimal management of critically ill obstetric patients.
- Ventilatory strategies in the obstetric population are similar to the non-obstetric population [2].

Classification of Ventilators Based on Delivered Breaths

Cycling Mechanism

- Cycling mechanism is nothing but the way the ventilator terminates the breath (Fig. 14.2).

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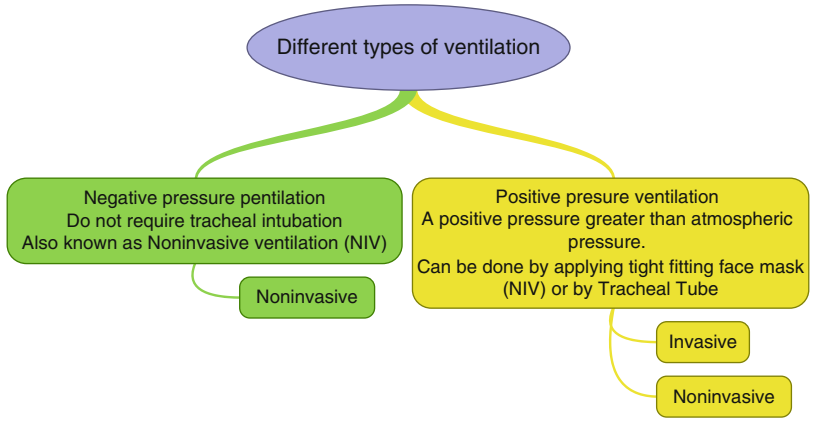

Fig. 14.1 Types of Ventilation

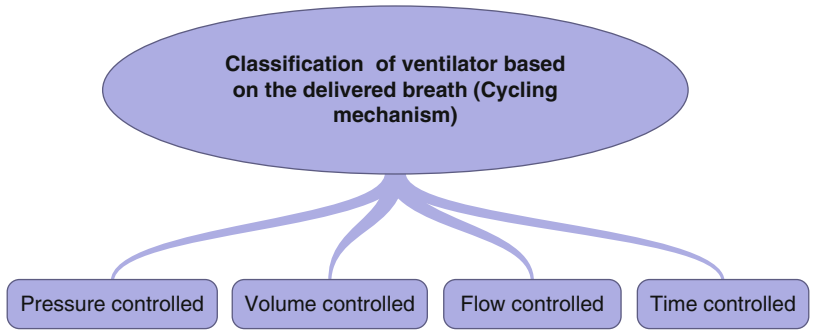

Fig. 14.2 Classification of Ventilation based on cycling mechanism

Pressure-Controlled or Pressure-Targeted Ventilation

- When the clinician uses set ventilating pressure as target, the ventilator delivers breath, till the preset pressure is achieved.
- During pressure control, volume cannot be set (volumes delivered are variable), hence risk of volume trauma.

Volume-Controlled or Volume-Targeted Ventilation

- When the clinician uses set tidal volume as target, the ventilator delivers breath, till the preset volume is achieved, and expiration follows.
- During volume control, pressure cannot be set (volume delivered are at variable pressures), hence risk of barotrauma.

Flow-Controlled or Flow-Targeted Ventilation

- When the clinician uses set flow as target, the ventilator delivers breath, till the preset flow is achieved, and expiration follows.

Time-Controlled or Time-Targeted Ventilation

- When the clinician uses set time as target, the ventilator delivers breath, till the preset fixed time intervals have been reached.

Modes of Ventilation

- Mode is defined as the method of providing inspiratory support or predefined way of interaction between the patient and the ventilator.

They can also be defined as the instruction given to the ventilator to deliver type or types of breath to the patient.

- Modes can be simple modes like volume ventilation and pressure ventilation or special or advance modes like dual modes (combined volume and pressure ventilation) (Fig. 14.3).

Volume Ventilation

During volume ventilation the clinician can set few parameters – Target tidal volume, respiratory rate, and inspiratory flow rates can be set, so that the ventilator can deliver the type of breath to patient's and clinician's need. Inspiratory flow rates can be set by I:E ratio or Ti.

Pressure Ventilation

During pressure ventilation the clinician can set few parameters – Target set pressure (<30 cm of water) respiratory rate, and inspiratory flow rates can be set, so that the ventilator can deliver the type of breath to patient's and clinician's need. Inspiratory flow rates can be set by I:E ratio, Ti, or Ti%.

Dual Modes

This mode helps the clinician to set targeted pressure and at the same time the desired tidal volume is assured. In this mode the desired tidal volume is delivered at lowest inspiratory pressures.

Initial Setting Up of the Ventilator

Once the patient meets the criteria of mechanical ventilation, the initial setting up of ventilator follows (Fig. 14.4):

Tidal Volume

- The minute ventilation increases by 40 % due to progesterone mediated increased sensitivity of respiratory center to carbon dioxide [3].
- It is calculated based on the ideal body weight (IBW).
- IBW = 105 + 5 (height in inches – 60); we get weight in pounds; hence to convert it into kilograms then, the pounds must be divided by 2.2.
- Tidal volume is set based on the pulmonary mechanics – Like in conditions such as drug overdose, organophosphorus compound poisoning, and neuromuscular disorder tidal volume = 6–8 ml/kg IBW (ideal body weight). One must take care not to exceed plateau pressures of 30 cm of water.
- In obstructive disease, tidal volume must be 8–10 ml/kg.
- In restrictive disease especially ARDS/ALI, tidal volume must be 4–8 ml/kg. Studies have shown that increased tidal volumes are associated with increased mortality.

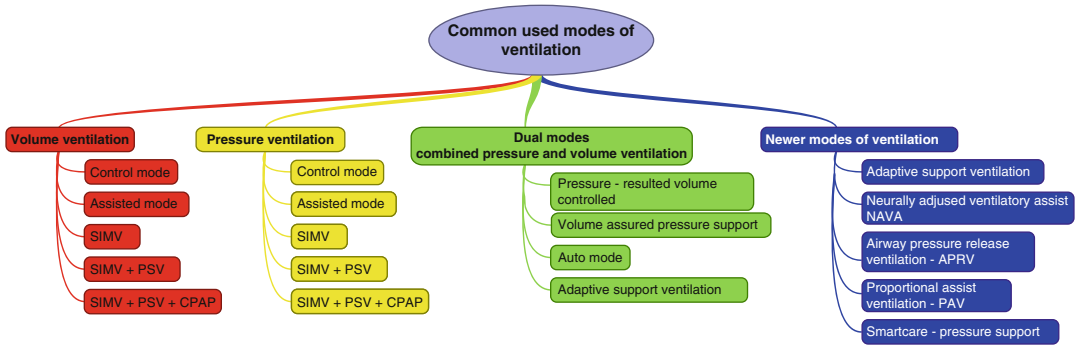

Fig. 14.3 *SIMV* synchronized intermittent mandatory ventilation, *PSV* pressure support ventilation, *CPAP* continuous positive airway pressure

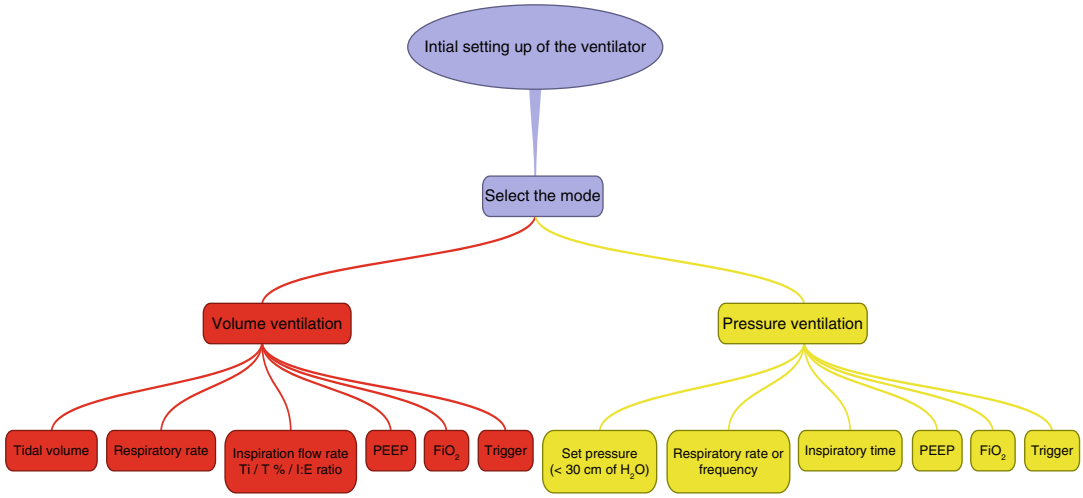

Fig. 14.4 Initial Settings of Ventilator

Respiratory Frequency or Rate

- It is set to suffice the minute ventilation (tidal volume X respiratory rate).
- The rate can be adjusted to patient's ABG. Mild respiratory alkalosis is acceptable during pregnancy ($PaCO_2$ = 32–35 mmHg).
- Normal respiratory rate is set between 8 and 12/min, so as to avoid hypercarbia.
- In obstructive disease, respiratory rate is 8–12/min [4].
- In restrictive disease especially ARDS/ALI, respiratory rate is 15–25/min [4].
- Targeted $PaCO_2$ should be in lower ranges than normal patients so that fetus is not subjected to acidosis due to hypercarbia (mild respiratory alkalosis is acceptable) [2].
- Persistent hypocapnia with respiratory alkalosis (pH > 7.48) may result in uterine artery vasoconstriction and decreased fetal perfusion.
- Permissive hypercarbia should be avoided in obstetric patients [2].
- Studies have documented a level up to $PaCO_2$ 60 mmHg without any fetal compromise [5].
- ABG sampling should be done in a sitting or semirecumbent or 45° head-up position if possible [3,6].
- Lower $PaCO_2$ levels are shown if ABG is done in supine position [2].

Inspiratory Flow Patterns and Flow Settings

- The flow pattern can be square, descending pattern.
- Square flow pattern is used when initiating mechanical ventilator.
- Decelerating or descending flow pattern is always associated with pressure ventilation, and the clinician cannot change this setting.
- In normal lungs, both flow patterns are acceptable.
- In restrictive disorder especially in ALI/ARDS, descending flow pattern is acceptable as it maintains low peak pressure and improves gas distribution and exchange and high airway pressure.
- In obstructive disease descending flow pattern is acceptable as it improves gas distribution.
- In normal lungs, high flows are acceptable (50–60 L/min).
- In ARDS there is slow inspiratory time (Ti), in COPD, there are faster flows, so that the inspiratory time is short and the expiratory time is prolonged, and hence carbon dioxide can be eliminated [4] (Figs. 14.5 and 14.6).

Inspiratory flow setting [4]	
High flow rates	Slow flow rates
High peak inspiratory pressure	Low peak inspiratory pressure
Poor gas distribution	Improves gas distribution and exchange
Short inspiratory time and I:E ratio	Increased inspiratory time and I:E ratio
	Increase in mean airway pressure
	Decrease in expiratory time and leads to air trapping or Auto PEEP

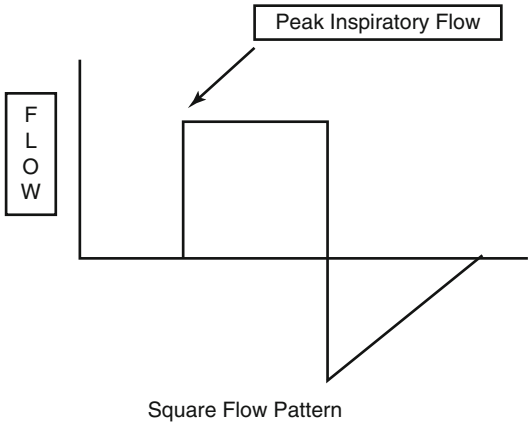

Fig. 14.5 Ventilator graphic – Flow pattern (Square)

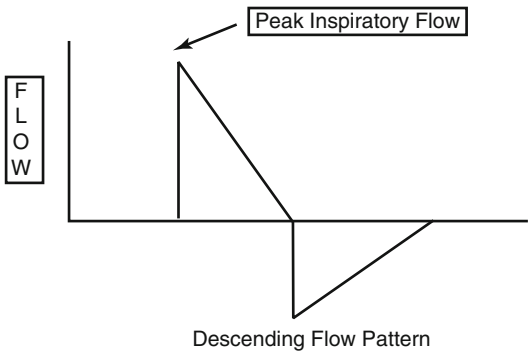

Fig. 14.6 Ventilator graphic – Flow pattern (descending flow)

FiO_2 and PEEP

- These patients also need higher PO_2 values to generate a gradient across the placenta for adequate fetal oxygenation.
- The initial FiO_2 is usually set at 100 % or 1.0 to avoid V/Q mismatch (V/Q ventilation–perfusion) and to prevent hypoxemia. The FiO2 needs to be reduced at the earliest, to prevent oxygen toxicity, atelectasis, etc. [4].
- PEEP – positive end-expiratory pressure is nothing but positive pressure maintained at the end of expiration to prevent alveoli collapse; this is done to maintain the functional residual capacity (FRC) and hence improves oxygenation [4].
- Avoid high levels of positive end-expiratory pressure in the parturient greater than 20 weeks gestation as it may further impair venous return and cardiac output.
- A normal physiological PEEP of 3–5 cm of water must be routinely used in all ventilated patients. This physiological PEEP do not affect the hemodynamics.
- Target maternal PaO_2 >67.5 mmHg to avoid fetal hypoxia.
- Lower PaO_2/FiO_2 limits are not acceptable and not advisable in obstetric patient with ARDS.
- Maternal hypercarbia must be avoided due to the potential for fetal acidosis and hypoxia.

Patient Monitoring During Ventilation [4]

- Ventilator pressure
- Lung pressure
- Ventilator graphics

Ventilator Pressure and Lung Pressure

Peak Inspiratory Pressure (PIP)

Peak inspiratory pressure = transairway pressure + plateau pressure.

- The highest pressure seen on the ventilator – Generally set 5–10 cm of H_2O above the PIP level.
- If PIP is increased, it means that airway pressure is high.

Plateau Pressure (Plateau)

- It's measured at the end of inspiration.
- It can be measured by inspiratory pause or hold.
- It reflects lung parenchyma or alveolar pressure.
- It's a direct measure of static compliance.
- Decreased lung compliance means increased plateau pressure; if plateau pressure is increased, the patient is at risk of barotrauma.

Compliance

- Stretch ability of the lung is called compliance.
- Two types include dynamic and static compliance.
- It is the change in volume per unit change in pressure.
  - Compliance = $\Delta V/\Delta P$.
- ΔV = volume change (lung expansion).
- ΔP = pressure change (work of breathing).
- It is due to reduced transalveolar pressures during pregnancy as a result of reduced total lung compliance (mainly reduced chest wall compliance).
- Hence higher plateau pressures may be required slightly more than 30 cm of H_2O.
- This must be avoided in patients with ARDS.
- This is done to achieve acceptable PaO_2 and $PaCO_2$ levels.

Cuff Pressure Monitoring

- Endotracheal cuff pressure should be maintained less than 25 mmHg (<25 mmHg) [4].
- This is done to avoid tracheal ischemia and necrosis [4].
- Minimal leak test (MLT) – Techniques to be used so that it's enough to touch the wall of the trachea [4].

Ventilator Graphics

- The graphics are used to interpret interactions between the patient and ventilator.
- It helps the clinician to make changes appropriately based on the graphs.
- Two types of graphic representation are available – scalar and loops.
- Scalars – Any variable (single) displayed against time is called as scalars.(e.g., volume vs. time or flow vs. time).
- Loops – Display of two scalar values (e.g., volume vs. pressure or flow vs. volume) is known as loops.
- Graphics help the clinician to identify Auto PEEP, air leaks, peak inspiratory pressures, active exhalation, inadequate inspiratory flows, and compliance (Figs. 14.7, 14.8, and 14.9).

Hemodynamic Monitoring in Critically Ill Obstetric Patients

Routine Monitoring

- Electrocardiogram – ECG
- Noninvasive blood pressure – NIBP
- Pulse oximetry – SpO_2
- Urine Output

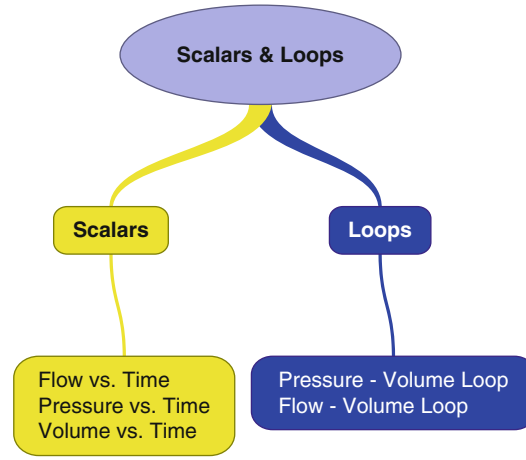

Fig. 14.7 Types of Ventilator Graphics

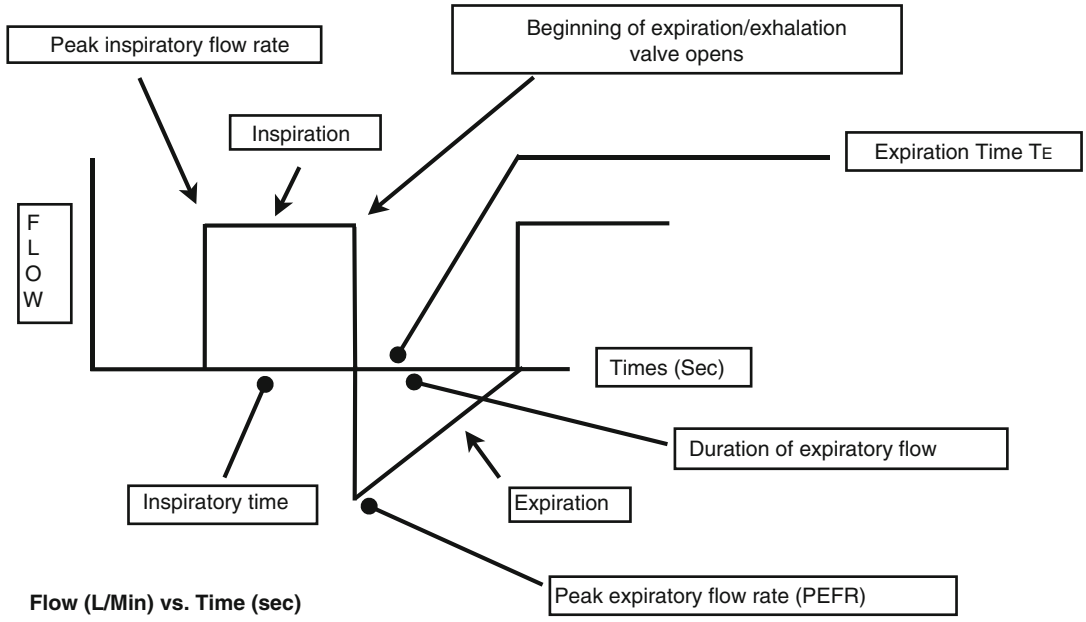

Fig. 14.8 Flow Scalars

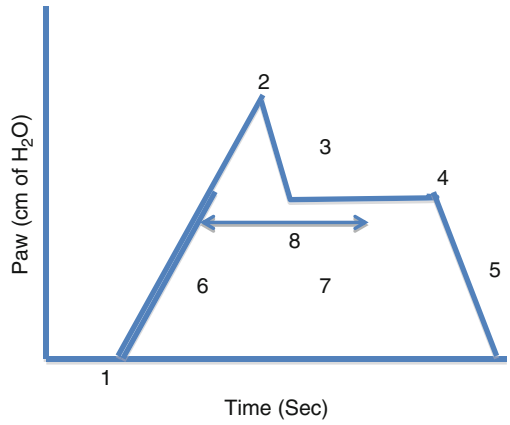

Fig. 14.9 Components of inflation pressure. *1* starts, *2* peak inspiratory pressures, *3* plateau/alveolar pressure, *4* begin expiration (exhalation valve opens), *5* expiration, *6* airway resistance, *7* distending pressure, *8* inspiratory pause

Special Monitoring

- Invasive blood pressure – IBP
- Central venous pressure monitoring (controversial)
- Acid Base Gas (ABG) – To monitor oxygenation and ventilation
- End-tidal carbon dioxide (EtCO2)
- Fetal heart rate monitoring
- Plateau pressures
- Compliance

Short Forms

- PIP – Peak Inspiratory pressure
- WOB – Work of breathing

Positioning [7]

- Aortocaval compression occurs as early as 18–20 weeks. Hence patient should always have lateral or tilted position which leads to left uterine displacement.

Weaning from Mechanical Ventilator

- Weaning is defined as gradual withdrawal of mechanical support from the patient and encouraging the patient to breath spontaneously, or weaning is the process of withdrawing mechanical ventilatory support and transferring the WOB from the ventilator to the patient [4] (Figs. 14.10).

Conditions that may interfere a successful weaning outcome [8].

Conditions	Examples
Patient/ pathophysiological	Fever, infection, renal failure, sepsis, sleep deprivation
Cardiac/circulatory	Arrhythmias, BP (high or low), CO (high or low), fluid imbalance
Dietary/acid base/ electrolytes	Acid-base imbalance, electrolyte disturbances, anemia

Common weaning criteria

Category	Examples	Values
Ventilatory criteria	$PaCO_2$	<50 mmHg with normal pH
	Vital capacity	>10–15 ml/kg
	Spontaneous V_T	>5–8 ml/kg
	Spontaneous RR	<30/min
	MV	<10 L
Oxygenation criteria	PaO_2 with PEEP	>100 mmHg with FiO_2 up to 0.4
	PaO_2 without PEEP	>60 mmHg with FiO_2 0.4
	SaO_2	>90 % with FiO_2 up to 0.4
	Q_S/Q_T (shunt)	<20 %
	P (A-a) O_2	<350 mmHg with FiO_2 of 1
	PaO_2/FiO_2	>200 mmHg
Pulmonary reserve	Max. ventilation	2 × MV with FiO_2 up to 0.4
	MIP (maximum inspiratory pressure)	>20–30 cm H_2O in 20 s
Pulmonary measurements	*Static compliance*	*>30 ml/cm H_2O*
	V_D/V_T	*<60 %*

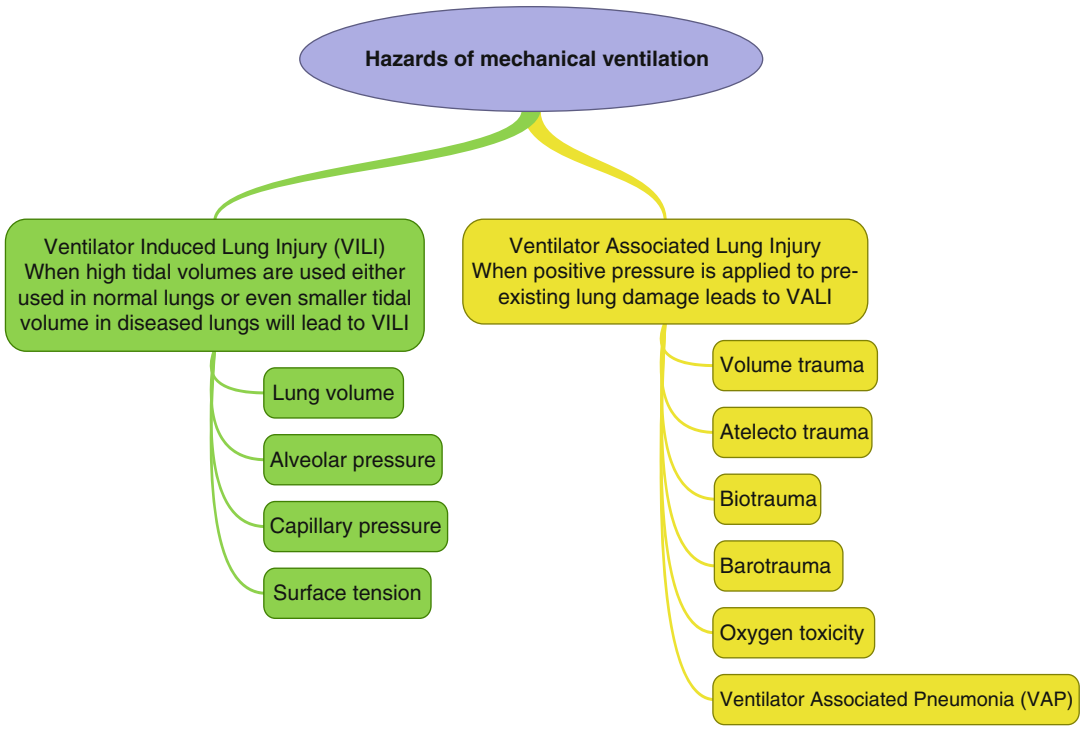

Fig. 14.10 Hazards of Mechanical Ventilation

Rapid Shallow Breathing Index (RSBI) [8]

- RSBI = respiratory rate or frequency (F)/tidal volume (VT).
- Normal RSBI = <100–105.
- It predicts spontaneous breathing trial (SBT).
- It has 97 % sensitivity and 65 % specificity.

Hazards of Mechanical Ventilation [9] (Fig. 14.10)

FAST HUG [10]

Give your patient FAST HUG everyday:

- F = Feeding
- A = Analgesia
- S = Sedation
- T = Thrombosis prophylaxis
- H = Head end of the bed to be elevated
- U = Ulcer prophylaxis
- G = Glycemic control

Summary

The most important point to be remembered is that, there is a need to care for two lives. The clinician must be aware of physiological changes that occur during pregnancy. The raising levels of progesterone that may lead to vasodilated state must be borne in mind. A team approach with active involvement of the obstetrician is a must [2]. Special attention must be paid for acid aspiration and compression of the inferior vena cava and aorta. Estrogen levels may lead to edema of the upper airway and hence warrants the use of smaller endotracheal tube, which may further lead to increased resistance to passive expiration [11]. The list of drugs that causes teratogenicity must be avoided during pregnancy.

Intense monitoring of the patient must be done along with fetal heart rate monitoring by the obstetric nurse at least 4–8 h in patients who are critically ill and more frequent monitoring if condition deteriorates [2]. Little evidence exists regarding the management of ARDS specifically in pregnancy. A multidisciplinary approach involving maternal-fetal medicine, neonatology, anesthesiology, and intensivist clinicians is essential to optimizing maternal and fetal outcomes [12].

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Nutrition in the Critically Ill Obstetric Patient

15

Kamini A. Rao and Smitha Avula

Introduction

> Let food be thy medicine and medicine be thy food. —— Hippocrates

Nutrition in a critically ill patient is a challenge in itself; altered metabolism in pregnancy with the superadded stress of critical illness makes the scenario more complex, vis-a-vis catering to adequate fetal nutrition for optimal growth of the fetus. Altered physiology in pregnancy and increased metabolic demand of critical illness pose diagnostic and therapeutic dilemmas in the management of critically ill pregnant patient (CIPP). Vulnerability to infection due to immunosuppression and thrombosis due to hypercoagulable state render the woman to succumb to the perils of thromboembolism and sepsis. Most common causes of admission to an ICU for obstetric patients are severe preeclampsia and eclampsia, hemorrhage and its complications, septic abortions, heart disease with cardiac failure, severe anemia, and non-obstetric causes like trauma and sepsis.

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In India, one woman dies every 5 min from a pregnancy-related cause [1]. It is estimated that 15 % of the deaths of women in reproductive age in India are maternal deaths. India has an MMR of 190 deaths per 100,000 live births [2]. According to District Level Health Survey II (DLHS-II) in 2002–2004, in urban areas, more than three-quarter (76 %) of the deliveries conducted were safe as against less than two-fifths (37 %) in rural areas. Extending appropriate antenatal care to the rural areas with prompt referral to tertiary care can minimize the mortalities. Multidisciplinary approach involving maternofetal medicine, intensivists, and nutritionists is warranted in the care of a critically ill pregnant patient (CIPP).

Altered Metabolism in Pregnancy

Maternal metabolism changes dramatically during pregnancy. Early gestation is an anabolic state in the mother with an increase in maternal fat stores and increase in insulin sensitivity. Nutrients are stored in early pregnancy to meet the fetoplacental and maternal demands of late gestation and lactation. In contrast, late pregnancy is better characterized as a catabolic state with increased insulin resistance which results in increase in maternal glucose and free fatty acid concentrations, allowing for greater substrate availability for fetal growth [3].

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Table 15.1 Physiological changes in pregnancy and their effect in resuscitation of a critically ill pregnant patient [4]

	Changes in pregnancy	Impact on resuscitation
Cardiovascular		
Plasma volume	↑ 50 %	Dilutional anemia
		↓ O_2 carrying capacity
Heart rate	↑ 15–20 bpm	↑ CPR circulation demands
Cardiac output	↑ 40 %	
Uterine blood flow	10 % of CO at term	Potential for rapid massive hemorrhage
Systemic vascular resistance	↓	Sequesters blood during CPR
Arterial BP	↓ 10–15 mmHg	↓ reserve
Respiratory changes		
Respiratory rate	↑	↓ buffering capacity, acidosis more likely
O_2 consumption	↑ 20 %	Hypoxia develops more quickly
Residual capacity	↓ 25 %	↓ buffering capacity, acidosis more likely
Arterial pCO_2	↓	
Laryngeal edema		Difficult intubation
Others		
Gastric motility	↓	Increased risk of aspiration
Lower esophageal sphincter	Relaxed	Increased risk of aspiration
Uterus	Enlarged	Aortocaval compression causes supine hypotension, reduces venous return, and significantly impairs CPR
Weight	Increases	Makes ventilation difficult

A thorough understanding and sound knowledge of the hemodynamic and other physiological changes of pregnancy is required to meet the increased metabolic demands in a CIPP (Table 15.1).

Green Top Guideline No. 56 Jan 2011

Critical Illness Worsening the Metabolic Scenario: Fuel to the Fire

Claude Bernard put forth "the principle of homeostasis" or controlled stability of the internal milieu of cells and tissues [5]. He proposed that "the fixity of the internal environment is the condition for free life," which is relevant to the critically ill whose homeostasis must be restored as rapidly as possible to survive the injury.

Critically ill patients are hypermetabolic and have increased nutrient requirements. Catabolic state and the development of resistance to anabolic signals takes place in order to reset the hierarchy of the delivery of the energy substrates preferring the brain over the brawn i.e. to prioritize vital tissues over the insulin-dependent organs, namely, fat and muscle [6, 7]. The magnitude of insulin resistance has been correlated with the severity of the condition [8]. The loss of lean tissue which occurs in catabolic patients suggests that depletion to a critical level may occur after 14 days.

The actual energy expenditure is extremely difficult to predict during critical illness [9]. It is influenced not only by intrinsic physiological factors like hyperthermia, tachycardia, and shivering but also by extrinsic therapeutic interventions like sedatives, use of β-blockers, and active cooling [10–13].

Estimating Energy Expenditure

For critically ill patients, indirect calorimetry seems the most feasible method. Four independent quantities required to be measured for this are daily intake of fat, carbohydrate, and protein, daily nitrogen excretion, daily oxygen consumption, and daily CO_2 production. For bedridden patients who are completely at rest during the entire 24 h, three to five measurements of resting energy expenditure (REE) may be extrapolated to obtain a reasonably good estimate of total energy expenditure (TEE) for the 24 h period. Van Lanschot et al. investigated the Harris-Benedict equation vs the accurate measurement of TEE by continuous indirect calorimetry. There is an overestimation of TEE when caloric needs were estimated by a formula albeit the difficulties associated with continuous indirect calorimetry make it less feasible in the practical scenario. The Harris-Benedict equation is an empirically derived equation with variables that reflect the

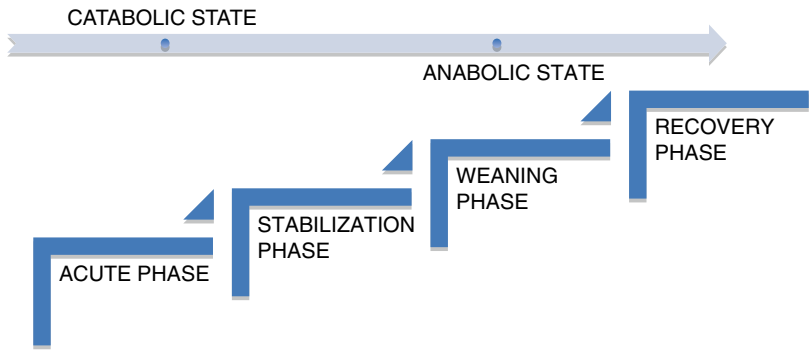

Fig. 15.1 Phases of illness paradigm criteria for critical illness
PHASE 1– Acute (6–24 h)
PHASE 2 – Stabilization (2–4 h)
PHASE 3 – Stable/weaning (24–72 h)
PHASE 4 – Recovery (indefinite)

relative contributions to overall heat production per square meter body surface area of activity, age, sex, and body size [7] (Fig. 15.1):

$$\text{BEE (Kcal/Day)} = 655 + (9.6 \times \text{Wt in kg}) + (1.7 \times \text{Ht in cm}) - (4.7 \times \text{age})$$

$$\text{BEE} \times 1.25^{*} + 300 \text{ Kcals for singleton pregnancy} + 500 \text{ Kcal for twin gestation}$$

*1.25 is the stress factor for pregnancy

BEE equals energy expenditure in kilocalories per day.

There are specific goal priorities for each phase. The acute phase is hypermetabolic and warrants effective organ support. Recovery phase involves recommencement of anabolism and restoration of organ function [14].

Nutritional Support

Nutritional support does not merely refer to provision of calories but also proteins, fluid, electrolytes, minerals, micronutrients, and fiber. Nutritional therapy is aimed at preserving lean body mass, maintaining immunological function, and averting metabolic complications. Nutritional therapy needs to be more focused in specifically attempting to attenuate the metabolic response to stress, to prevent oxidative cellular injury, and to favorably modulate the immune response.

Table 15.2 Indications of nutritional support in the malnourished and those at risk of malnutrition

Nutritional support	In malnourished	In patients at risk of malnourishment
	BMI <18.5	Eaten nothing/little for >5 days and likely to eat nothing/little for 5 days or longer
	Weight loss >10 % in 3–6 months	Poor absorptive capacity and/high nutrient losses and/increased nutritional needs from causes such as catabolism
	BMI <20 Kg/m^2 and an unintentional weight loss of >5 % in 3–6 months	

Indications for Nutritional Suppport

Nice Guideline 32 Feb 2006
(Table 15.2)

Goals of Nutrition in Critically Ill

- 25–35 Kcal/Kg/day total energy – the target of total calories must be set keeping in mind the use of inotropes may increase caloric requirement by 2.5 times [15], with each 1 °C fever the caloric need increases by 10 % [16], and sepsis may increase basal need by 1.9 times [17, 18].

- 0.8–1.5 g protein/Kg/day – an additional 20 % is required in second and third trimesters.
- 30–35 ml fluid/Kg with additional allowance for extra losses.
- Lipids are important in placental and fetal development. Both soybean and safflower emulsions carry the FDA Pregnancy Category of C; however, case studies support safe use during pregnancy. It is recommended that lipid infusions be limited to 30 % or less of the total calories.
- The use of omega 3 fatty acids in diet in obstetric population was advocated to decrease the incidence of preterm labor and preeclampsia, although the results of trials are showing no added benefits in this regard [19, 20]. Docosahexaenoic acid – a type of omega 3 fatty acid – has shown some promise in terms of better outcomes in neonatal visual and neural testing [21].
- Adequate electrolytes, minerals, micronutrients, and fiber

Goals Specific to CIPP

- Adequate B6, B12, and folate supplements.
- Maintain blood glucose concentration between 80 and 120 mg% to prevent hyperglycemia and fetal macrosomia.
- Weight gain 0.5–1 lb/week in second and third trimester.
- Ketosis should be prevented and suggest inadequate carbohydrate intake.
- Monitor fetal growth and well-being with periodic ultrasound examinations.
- Hypercoagulability of pregnancy and prolonged immobilization may warrant thromboprophylaxis.
- Levels of iron, serum ferritin, B12, and folate need to be monitored.
- Prophylactic vitamin K supplementation.
- Adequate fiber to prevent constipation.
- Adiponectin/leptin ratio could be useful to evaluate the metabolic state in critical obstetric patients, due to the exclusive secretion of leptin by adipose tissue [22].
- Serum protein markers (albumin, prealbumin, transferrin, C-reactive protein) are not validated for determining adequacy of protein provision and should not be used in the critical care setting in this manner.

Modes of Nutritional Support

1. *Enteral nutrition*
2. *Total parentral nutrition*

Enteral Nutrition (EN)

Delivering early nutrition support therapy, primarily using the enteral route, is seen as a proactive therapeutic strategy that reduces disease severity, diminishes complications, decreases length of stay in the ICU, and favorably impacts patient outcome.

The specific reasons for providing early EN are to maintain gut integrity, modulate stress and the systemic immune response, and attenuate disease severity. Additional endpoints of EN therapy include use of the gut as a conduit for the delivery of immune-modulating agents and use of enteral formulations as an effective means for stress ulcer prophylaxis.

Although enteral nutrition is the preferred route of nutrient delivery in pregnancy, it is often difficult to achieve in pregnant women. Decreased gastroesophageal sphincteric tone and delayed gastric emptying increase the risk of gastric reflux with nasogastric tube. Hence, naso-enteric feeding tube placement is less invasive than and better tolerated than the other modalities. Post-pyloric feeding is recommended over nasogastric feeds due to increased risk of aspiration. There are only few limited case reports on the use of percutaneous endoscopic gastrostomy in pregnant women. It does not require radiologic confirmation for placement and causes relatively less gastroesophageal reflux, esophagitis, and aspiration pneumonia when compared to naso-enteric access, though higher expertise is required for the same.

Nutritional support should be introduced cautiously in the critically ill pregnant patient. It should be started at no more than 50 % of the

Table 15.3 Indications of EN and TPN

Indication	EN	PN
Medical	Inflammatory bowel disease	Nonfunctional/leaking GI tract
	Respiratory/renal/hepatic failure	
Neurological	Cerebrovascular accident, brain injury/tumor	
Surgical	Fistula/burn/sepsis, GI cancer	
Miscellaneous	Hyperemesis gravidarum, anorexia nervosa, transition from PN	

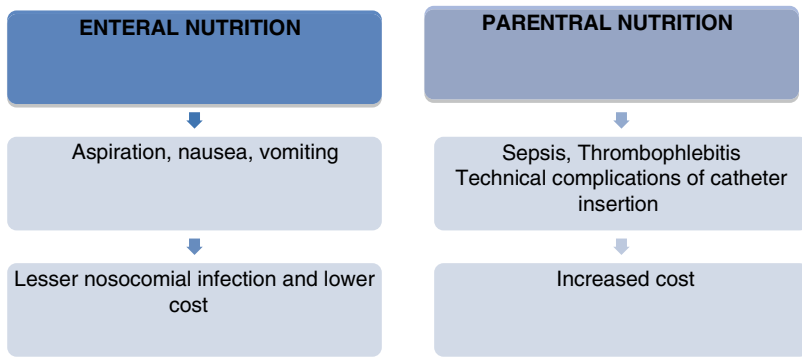

Table 15.4 Enteral vs parentral nutrition

estimated target energy and protein requirement. It must be built up to meet full needs over the first 24–48 h according to metabolic and GI tolerance (Tables 15.3 and 15.4).

Complications

Catheter-Related Complications

Immunocompromised state of pregnancy favors infection and thrombosis. Russo-Stieglitz et al. reported a 34 % incidence of complications related to central catheters in pregnant patients, with an incidence of 50 % in subclavian vs only 9 % in peripherally inserted catheters. Infection in the peripherally inserted central catheter lines can predispose to preterm labor and sepsis.

Refeeding Syndrome

Refeeding syndrome was first described in Far East prisoners of war after the Second World War. Starting to eat again after a period of prolonged starvation seemed to precipitate cardiac failure. It can occur in both enteral and parentral nutrition (Fig. 15.2).

At-Risk Group

Patient has one or more of the following (Table 15.5):

Patient has two or more of the following (Table 15.6).

Nice Guidelines 2006

Refeeding syndrome can be prevented in these risk categories by specialized and appropriate care.

- Starting nutrition support at a maximum of 10 Kcal/Kg/day and increasing levels slowly to meet full needs by 4–7 days.
- Using only 5 Kcal/Kg/day in extreme cases.

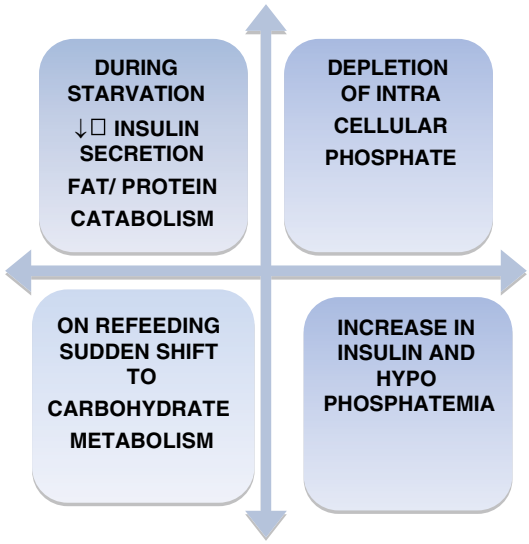

Fig. 15.2 Refeeding syndrome: shift of metabolism from a catabolic to an anabolic state

Table 15.5 Identifying patients at risk of refeeding syndrome: one or more of the following

BMI <16 Kg/m^2
Unintentional weight loss >15 % in the last 3–6 months
Little or no nutritional intake for the past 10 days
Low levels of potassium, magnesium, and phosphate prior to feeding

Table 15.6 Identifying patients at risk of refeeding syndrome: two or more of the following

BMI <18.5 Kg/m^2
Unintentional weight loss >10 % in the last 3–6 months
Little or no nutrition for more than 5 days
Drugs including insulin, antacids, diuretics, and chemotherapy

- Closely monitoring fluid balance and restoration of circulatory volume.
- Providing before and during the first 10 days oral thiamine 200–300 mg/day and a balance multivitamin/trace element supplement.
- Providing oral/enteral/intravenous supplement of potassium 2–4 mmol/kg/day, phosphate likely requirement 0.3–0.6 mmol/kg/day, and magnesium 0.2 mmol/kg/day (IV) and 0.4 mmol/kg/day oral. Pre-feeding correction of plasma levels is unnecessary.

Special Scenarios

1. *In Acute Renal Failure*

   Is associated with major fluid, electrolyte, and acid-base equilibrium derangements such as hypo/hypernatremia, hyperkalemia, hyperphosphatemia, and metabolic acidosis. Restriction of potassium, magnesium, and phosphate is not required in patients who are on daily renal replacement therapy (RRT). Serum electrolyte levels largely depend on electrolyte composition of the dialysate and the intensity of RRT. Hypophosphatemia and hypomagnesemia can frequently be observed in chronic RRT and should be anticipated. Macronutrients required are influenced by the type and severity of the underlying disease, type and intensity of the extracorporeal RRT, nutritional status, and associated complications, rather than by the ARF itself. Fluid restriction and electrolyte-free formulae are required. Enhanced requirement for micronutrients induced by RRT should be met. Patient should be monitored for vitamin A toxicity. Vitamin C inappropriate supplementation can lead to secondary oxalosis, and dose should not exceed 30–50 mg/day. Thiamine and selenium deficiency has been noted despite supplementation [23].

   From metabolic point of view, patients with chronic kidney disease (CKD) or chronic heart disease who develop a superimposed acute illness should be considered to be similar to patients in ARF.

2. *In Chronic Kidney Disease*

   Energy intake >30–35 kcal/kg/day is associated with better nitrogen balance and is recommended in stable CKD patients. The end points of management are prevention and treatment of protein energy wasting leading to cachexia; ensuring optimal levels of energy, essential nutrients, and trace elements; and attenuation of disease progression through protein/phosphate restriction.

3. *In Inflammatory Bowel Disease*

   Prevalence of IBD-associated malnutrition is high due to inadequate oral intake, malabsorption, increased intestinal loss, and increased caloric needs. Nutritional deficiencies are

constantly present in IBD, but their type and incidence varies with the type of disease [24].

Anemia and hyposideremia are more common in ulcerative colitis, whereas patients with Crohn's disease develop severe nutritional deficiencies including hypoalbuminemia. Enteral nutrition in Crohn's disease favors remission and alleviates inflammation. TPN in Crohn's disease is an exception of the rule. In contrast, inflammatory response in UC is not altered by nutritional support.

TPN is restricted to certain cases involving efforts to close enterocutaneous or other complicated fistulas in patients with fistulizing CD, the treatment of short bowel syndrome following extensive resections for CD, or when EN is impractical for other reasons. There are no advantages of TPN therapy over EN therapy regarding fistula healing. Immunotherapy with glutamine and short-chain and omega-3 fatty acids is still under trial for their use in IBD [25].

Conclusion

- Nutrition plays a key role in the management of CIPP.
- Nutritional support extends beyond the paradigm of mere provision of calories to proteins, lipids, micronutrients, and minerals and is extending its scope into immunonutrition as well.
- Pregnancy per se warrants an overall increase in nutritional support involving both macronutrients and micronutrients especially thiamine, B12, folate, iron, and fiber.
- Different phases of critical illness, representing varying metabolic states from high fuel demand catabolic state to a more steady plateau phase and into a prolonged anabolic state, should be borne in mind while planning nutritional support.
- Customized nutritional plans have to be catered to in specific overlapping scenarios with pregnancy as in acute renal failure and inflammatory bowel disease.
- Judicial use of enteral vs parentral nutrition can optimize nutrition and minimize complications.
- The implications of nutritional support in CIPP extend beyond the horizon of mere provision of calories but maintain the metabolic milieu of pregnancy catering to the needs of both the mother and her unborn fetus and combat the critical illness.

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Part III

Clinical Shock Syndromes

16 Post-partum Haemorrhage: Prevention, Medical and Mechanical Methods of Management

Ruchika Garg

Changing Practices

Definitions

PPH is defined as any blood loss that causes a major physiological change (e.g. a fall in blood pressure), as the risk of dying from PPH depends not only on the amount and rate of blood loss but also on the health of the woman.

Pathophysiology

Healthy women can compensate for considerable blood loss over lengthy periods without demonstrating any cardiovascular changes. Mild shock occurs when about 20 % of the blood volume is lost, which results in decreased perfusion of non-vital organs and tissues (skin, fat, skeletal muscle and bone), with pale, cool skin and a feeling of increasing coldness. When 20-40 % of blood volume is lost there is moderate shock with decreased perfusion of vital organs (liver, gut, kidney), oligomers and or Andrea, drop in blood pressure and mottling of skin of extremities especially legs. When 40 % or more of the blood volume is lost, severe shock occurs resulting in decreased perfusion to the heart and brain, restlessness, agitation, coma, electrocardiographic and electroencephalographic abnormalities and possibly cardiac arrest [1].

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Prevention

Women with identified risk factors should be transferred to centres with transfusion facilities and an intensive care unit (ICU) for delivery if these are not available locally. The Royal College of Obstetricians and Gynaecologists urges for the prevention and management of PPH in high-risk cases and recommends the introduction of strategies for the management of unpredicted PPH [2].

It should include antenatal risk assessment and management that assures that anaemia or other health problem are treated and women are sufficiently healthy to withstand PPH.

Active management of third stage includes administration of uterotonic agents, controlled cord traction and uterine massage after delivery of the placenta, as appropriate. This approach reduces the risk of PPH, post-partum anaemia, blood transfusion requirements, prolonged third stage of labour and use of therapeutic drugs for PPH [1].

Oxytocin is a first-line agent because it is effective 2–3 min after injection, and, as it has minimal secondary effects, it can be used in all women. If oxytocin is not available, other uterotonics can be used, such as ergometrine maleate 500 μg i.m., ergometrine with oxytocin, 5 IU/ml or misoprostol.

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Misoprostol, a prostaglandin E_1 analogue, is more stable than oxytocin and has been administered by oral, sublingual and rectal routes [3]. The main side effects are nausea, vomiting and diarrhoea. Shivering and elevated body temperature have also been reported. Moreover, rectal misoprostol causes less pyrexia and shivering than oral misoprostol. Oral administration of misoprostol should, therefore, be considered in low-resource settings where safe administration and/or appropriate storage conditions for injectable oxytocin and ergot alkaloids are not possible [4]. A recent Cochrane review on the use of prostaglandins for the prevention of PPH concluded that neither intramuscular prostaglandins nor misoprostol is preferable to conventional injectable uterotonics as part of the management of the third stage of labour especially for low-risk women [5].

Advantage of intramuscular carbetocin over intramuscular oxytocin is its longer duration of action in comparison to oxytocin, in terms of both amplitude and frequency of contractions, but there is insufficient evidence that intravenous carbetocin is as effective as oxytocin to prevent PPH. Nevertheless, carbetocin is associated with reduced need for additional uterotonic agents and uterine massage, and there are no significant differences in adverse effects between carbetocin and oxytocin [6].

There is very little evidence to suggest that the timing of cord clamping has an impact on the incidence of PPH. One study reported no significant difference in PPH associated with timing of cord clamping [7]. However, immediate cord clamping may reduce the quantity of red blood cells a newborn receives, whereas delayed cord clamping is associated with less anaemia, intraventricular haemorrhage and late-onset sepsis especially in preterm infants [8, 9]. For these reasons, the collaborative ICM/FIGO group decided *not to include early cord clamping in the active management protocol*. The cord may be clamped at the time the baby is dried and wrapped and passed to the mother to breast-feed. The placenta usually separates by that time and it is appropriate to apply cord traction. *Early clamping* may be indicated in case *of foetal distress* when immediate resuscitation is required.

To avoid maternal overexertion, they should not begin cord traction before the uterus has contracted and the expulsion of the placenta has begun, thus allowing the mother to expel the placenta without interference [10].

Active management of the third stage of labour should be offered by skilled attendants, as potential risks such as uterine inversion, may result from inappropriate cord traction.

Treatment

Haemostasis Algorithm

General medical management
H-Ask for help
A-Asses (vital parameters, blood loss) and resuscitate
E-Establish aetiology (4Ts-tone, tissue, trauma, thrombin), Ecbolics – (Syntometrine, ergometrine, bolus syntocinon), Ensure availability of blood and blood products
M-Massage the uterus
O-Oxytocin infusion, prostaglandins (intravenous, rectal intramuscular, intramyometrial)

Specific surgical management
S-Shift to operation theatre, bimanual compression, antishock garment, especially if transfer is required
T-Tissue and trauma to be excluded and proceed to tamponade balloon and uterine packing
A-Apply compression sutures
S-Systematic pelvic devascularisation (uterine, ovarian, quadruple, internal iliac)
I-Interventional radiology, uterine artery embolisation
S-Subtotal or total abdominal hysterectomy

Medical Management

Assess (Vital Parameters, Blood Loss) and Resuscitate

Meticulous estimation of the blood loss forewarns of impending haemorrhagic shock. Different methods of estimation have been evaluated [11], and guidelines to improve accuracy of the visual estimation of blood loss have been suggested

[12]. Two large-bore cannulae should be inserted and blood samples taken for full blood count, group and save or cross-match (depending on the severity of haemorrhage), coagulation screen and renal and liver profile.

Fluid resuscitation in PPH is often overly conservative because of underestimation of volume and rapidity of blood loss. Symptoms of hypovolaemia are often delayed due to pulmonary oedema, or failure may be misleading. *A loss of 1 l of blood requires replacement with 4–5 l of crystalloid (0.9 % normal saline or lactated Ringer's solution) or colloids until cross-matched blood is available, as most of the infused fluid shifts from the intravascular to the interstitial space* [11].

The Golden Hour

If the estimated blood loss is more than one third of the woman's blood volume (blood volume (ml) = weight (kg) ×80) or more than 1,000 ml or a change in haemodynamic status. As more time elapses between the onset of severe shock and resuscitation, the chances of survival decrease because metabolic acidosis sets in. The 'golden hour' is the time at which resuscitation must be commenced to ensure the best chance of survival. The probability of survival decreases sharply after the first hour if the patient is not effectively resuscitated [10].

For the general acute management of PPH, a '*rule of 30*' has been proposed. If the patient's systolic blood pressure (SBP) falls by 30 mmHg, heart rate (HR) rises by 30 beats/min, respiratory rate (RR) increases to >30 breaths/min and haemoglobin (Hb) or haematocrit (Hct) drops by 30 %, and/or if her urinary output is <30 ml/h, then the patient is most likely to have lost at least 30 % of her blood volume and is in moderate shock leading to severe shock [13].

Shock index (SI) may also be valuable in the monitoring and general management of women with PPH. It refers to heart rate (HR) divided by the systolic blood pressure (SBP). The normal value is 0.5–0.7; however, with significant haemorrhage, it increases to 0.9–1.1 [13]. The change in SI of an individual patient appears to be a better correlate in identifying early acute blood loss than the HR, SBP or DBP used in isolation [14].

O-Oxytocin Infusion, Prostaglandins

Ergometrine is an ergot alkaloid. It has potential development of severe hypertension and myocardial ischaemia.

It is a second-line agent for uterine atony (0.25 mg repeated every 15 min to a maximum dose of 2 mg). This is 80–90 % effective in stopping PPH in cases that are refractory to oxytocin and ergometrine. It has bronchoconstrictive properties and is, therefore, contraindicated in asthma. Side effects include diarrhoea, vomiting, fever, headache and flushing.

*Recombinant activated factor VII (rFVIIa, NovoSeven) w*as originally used in treating haemorrhage in patients with haemophilia with inhibitors, acquired haemophilia or other inherited bleeding disorders. In recent years, it has also been used in non-haemophilic haemorrhage including life-threatening obstetric haemorrhage. A number of case reports of empirical 'off-label' use of rFVIIa show that it may be an alternative haemostatic agent when the standard treatment is ineffective [15].

Surgical Management

Uterine tamponade can be effective in decreasing haemorrhage secondary to uterine atony and that procedures such as uterine artery ligation or B-Lynch suture may be used to obviate the need for hysterectomy. In patients with stable vital signs and persistent bleeding, arterial embolisation may be suitable, especially if the rate of loss is not excessive [16].

S-Shift to Operating Theatre (Antishock Garment, Especially if Transfer Is Required and Bimanual Compression)

In low-resource settings (home births, midwifery-led units or remote areas), transfer to centre with

facilities is indicated at this stage. A new type of non-pneumatic antishock garment (NASG) can reverse the effect of shock on the body's blood distribution by applying external counter pressure to the legs and abdomen and returning blood to the vital organs, thus stabilising women until a hospital can be reached. The use of this device could be critical to decrease maternal mortality in low-resource settings where reaching a health facility is time consuming.

T-Tissue and Trauma to Be Excluded and to Proceed to Tamponade with Balloon or Uterine Packing

Uterine packing has long been considered safe, quick and effective for controlling PPH [17].

Successful use of uterine balloon tamponade has been reported using a number of devices, *including the Foley catheter, a condom, the Sengstaken-Blakemore oesophageal catheter (SBOC), the Rusch urological hydrostatic balloon and the Bakri balloon. The SBOC has been the most frequently reported device*. Overall, the reported success rates vary between 70 and 100 % [18]. Uterine tamponade with the SBOC has been described as a prognostic test in obstetric haemorrhage by Condus et al. [19]. In their study, the 'tamponade test' had a positive result of >87 % for successful management of PPH. Furthermore, the use of balloon tamponade in the successful management of PPH secondary to extensive vaginal lacerations has recently been reported [20].

The 'tamponade test' can arrest bleeding in the majority of women with severe PPH and allow the obstetrician to identify which women will require laparotomy. This method has the advantage that (i) insertion is easy and rapid with minimal anaesthesia; (ii) it can be performed by relatively inexperienced personnel; (iii) removal is painless and (iv) failed case can be identified rapidly. The early use of balloon tamponade may be expected to result in reduced total blood loss and haemorrhage-related sepsis or long-term complications (such as menstrual problems or problems with conception) have been reported in women who have undergone uterine tamponade.

Uterine Balloon Tamponade

How to Do It

- Exclude local trauma or retained tissue in the uterus under spinal, epidural or general anaesthesia.
- Secure the anterior lip of the cervix with a sponge forceps.
- When the Sengstaken-Blakemore catheter is used, cut and remove the distal tube to facilitate insertion and retention in the uterine cavity.
- Hold the balloon catheter with another sponge forceps and insert it into the uterine cavity.
- Fill the balloon with warm sterile water or a warm saline solution until it becomes visible in the cervical canal. When the pressure exceeds that of the patient's blood pressure, no additional fluid needs to be added and the bleeding should stop.
- If there is no bleeding through the cervix or through the drainage channel of the balloon catheter, the 'tamponade test' result is pronounced successful and no further solution is added.
- If the bleeding does not stop, the result is unsuccessful and laparotomy is indicated.
- The uterine fundus is palpated abdominally and a mark is made with a pen as a reference line from which any uterine enlargement or distension would be noted.
- Administer oxytocin infusion (40 IU in a litre of normal saline solution) to keep the uterus contracted.
- Keep the patient under constant surveillance after insertion of the tamponade balloon catheter. *Monitor pulse, blood pressure, uterine fundal height and signs of any vaginal bleeding or bleeding through the lumen of the catheter every 30 min.* Check temperature every 2 h and urinary output hourly via an indwelling Foley catheter with a urometer.
- Give intravenous broad-spectrum antibiotics at the time of insertion and for up to 3 days.

Removal of the Balloon

- After 6–8 h, if the uterine fundus remains at the same level and there is no active bleeding through the cervix or the central lumen of the catheter, it is safe to remove the balloon provided that the woman is stable and adequate blood replacement has been provided.
- Keep the patient fasting for 2 h after the balloon is removed in case surgery is needed under anaesthesia.
- Deflate the balloon slowly, but do not remove it for 30 min.
- Continue the oxytocin infusion even if there is no bleeding.
- If there is still no bleeding after 30 min, discontinue the oxytocin infusion and remove the balloon catheter.
- If bleeding starts when the balloon is deflated, inflate the balloon again.

A-Apply Compression Sutures

If the patient is stable and bimanual compression of the uterus successfully achieves haemostasis, then compression sutures may be of value. Various modifications have been reported to the original B-Lynch [21] suture technique [22, 23]. The obvious disadvantage is the need for laparotomy and, usually, hysterotomy (although some modified types have avoided this step of the procedure). Recognised complications include erosion through the uterine wall, pyometra and uterine necrosis [18].

S-Systematic Pelvic Devascularisation

This requires laparotomy and progressive, stepwise devascularisation, whereby the uterine, ovarian and finally iliac arteries are ligated. Vaginal ligation of the uterine arteries has also been described.[25] Internal iliac artery ligation is usually effective in arresting bleeding from all sources within the genital tract, but is can be time consuming, is technically challenging and carries risks of injury to other structures. Prerequisites include a haemodynamically stable patient, substantial surgical expertise and a desire to preserve fertility. The reported success rates are between 40 % and 100 % [18].

The Stepwise Pelvic Devascularisation Technique

1. Absorbable sutures should be used for all ligatures.
2. Bilateral uterine artery ligation at the level of the uterine border beside the upper part of the lower uterine segment is usually the first step.
3. If bleeding continues and is likely to originate from the lower uterine segment:
   - The bladder should be reflected inferiorly.
   - A second lower bilateral uterine vessel ligation should be performed at the lower part of the uterine segment, 3–5 cm below the upper ligatures. At this level, the uterine artery is ligated bilaterally at the reflection of the cervicovaginal branch. This ligature should obliterate most of the branches of the uterine artery to the lower uterine segment and a branch that extends to the upper portion of the cervix.
   - The ligatures should include a significant amount of myometrium to avoid damage to the uterine vessels and to obliterate some of the arterial branches.
4. Bilateral ovarian artery ligation. A suture is passed through the avascular area in the infundibulopelvic ligament to include the ovarian vessels.
5. Bilateral internal iliac artery ligation. This step should be performed by surgeons with expertise in the anatomy of the pelvis.
6. Concomitant blood and blood product transfusion (and resuscitation measures) should be provided according to the patient's haemodynamic status.

The choice of any measure may depend on the availability of facilities and a number of rapidly changing parameters, such as the degree of ongoing bleeding, the estimated blood loss and the haemodynamic status of the woman.

S-Subtotal or Total Abdominal Hysterectomy

Subtotal or total abdominal hysterectomy is usually the final option in the management of PPH and should not be delayed if the conservative measures have failed. A gravid uterus is vascular and urinary tract injuries are more common due to the anatomical changes in pregnancy. However, subtotal hysterectomy may not be effective when the source of the bleeding is in the lower segment, cervix or vaginal fornices.

Conclusions

Prompt resuscitation of the patient with restoration of circulating blood volume and identification of the cause of bleeding should be performed by a multidisciplinary team approach.

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Conservative and Nonconservative Surgical Management of Postpartum Hemorrhage

17

V.P. Paily and Vasanthi Jayaraj

Introduction

Hemorrhage still remains the leading cause of maternal mortality and morbidity in the developing countries. In the developed countries also, postpartum hemorrhage (PPH) is important in the obstetric practice, but it contributes to maternal mortality and morbidity less often because of prophylactic steps and prompt attention. In both settings, a surgical approach will be called upon when medical management fails or along with it.

The division into conservative and nonconservative surgical procedures is artificial but serves to categorize them in a practical sequence of increasing importance and level of skills. Several factors should be considered in deciding on the appropriate steps in a given situation. They include the cause of the bleeding, the condition of the patient, and the facilities and expertise available. In some conditions like atonic PPH, medical management should be the first one to be considered, whereas in genital tract trauma surgical management takes priority. In any setting, general measures to stop the bleeding (e.g., oxytocics) and resuscitation of the patient (e.g., intravenous fluids, blood, and blood products) are relevant.

Procedures like brace stitches, stepwise devascularization, and internal iliac artery ligation fall under conservative surgical steps. Hysterectomy is the only condition that falls under the nonconservative surgical procedures and usually is considered as the last step when all conservative measures have been exhausted. This should not be the case. Ideally hysterectomy should follow the medical measures and conservative management in rapid sequence and depending on the degree of bleeding and the general condition of the patient.

In any case of PPH, medical methods and various types of tamponade are the first steps to be considered. These are covered in other chapters of this book. Only the surgical steps, conservative and nonconservative, will be dealt with here. These include clamping the uterine arteries vaginally as a first aid, tackling the lower genital tract lacerations, procedures like various tamponade stitches (e.g., B-Lynch and Hayman's), and ligation of major vessels supplying the uterus, cervix, and vagina. If none of these work, we have to proceed to hysterectomy. However, unless properly selected, even hysterectomy may not stop the PPH.

We feel that the importance of early venous access with wide-bore cannula needs to be highlighted. This is a top priority so that fluid resuscitation can start. When the veins are collapsed, even ultrasound guidance does not help to get access to the veins. Rather than blindly

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trying on all possible sites and creating hematomas from counter-punctured vessels, one should try the open method to access the veins (cut down). Every labor room should have a set for this readily available. It should contain two mosquito forceps, an iris scissors, a small towel 18″ × 12″ with a central hole, and a couple of gauze pieces and cotton balls. Along with this should be a surgical blade (11F), intravenous cannulas (14 and 16F), and a foil of 2-0 plain catgut or 3-0 delayed absorbable suture. Together these constitute a cut down set. Open access to veins should be attempted in the antecubital fossa or just above the ankle. The cannula should be threaded into the vein after removing the stilette.

The procedures that will be covered in this chapter are:

1. Transvaginal uterine artery clamp
2. Lower genital tract lacerations
3. Brace stitches of different types
4. Major vessel ligation including internal iliac artery ligation
5. Temporary occlusion (with clamps) of common iliac arteries or lower end of the aorta
6. Obstetric hysterectomy
7. Which procedures to choose in a given situation

Transvaginal Uterine Artery Clamping

About 85 % of blood reaching the gravid uterus is through the uterine arteries. These branches of internal iliac arteries join the uterus on its sides at the level of the isthmus. Anatomically, this point of entry of the uterine artery is only about 1 cm above the lateral fornix of the vagina. Hence it is possible to reach the uterine arteries through the lateral fornix in a postpartum situation when the tissues will be soft.

Based on the above principle, the senior author has developed a clamp which can be applied transvaginally to occlude the uterine arteries at the point where it approaches the uterus on its sides (Fig. 17.1). These are long atraumatic clamps, 12 in. long, of which 4 in. is the distance from the box joint to the tip. The tip itself is bent at right angles and is shaped like that of a sponge-holding forceps. Even when tightened to the maximum, the blades will be at a distance of about 3 mm between them so that the soft tissue of the cervix which will be between them will not get unduly compressed. One forceps is applied at 3 o'clock and the other at 9 o'clock position of the cervix. One blade goes inside the cervical canal and the other on the lateral fornix, and just

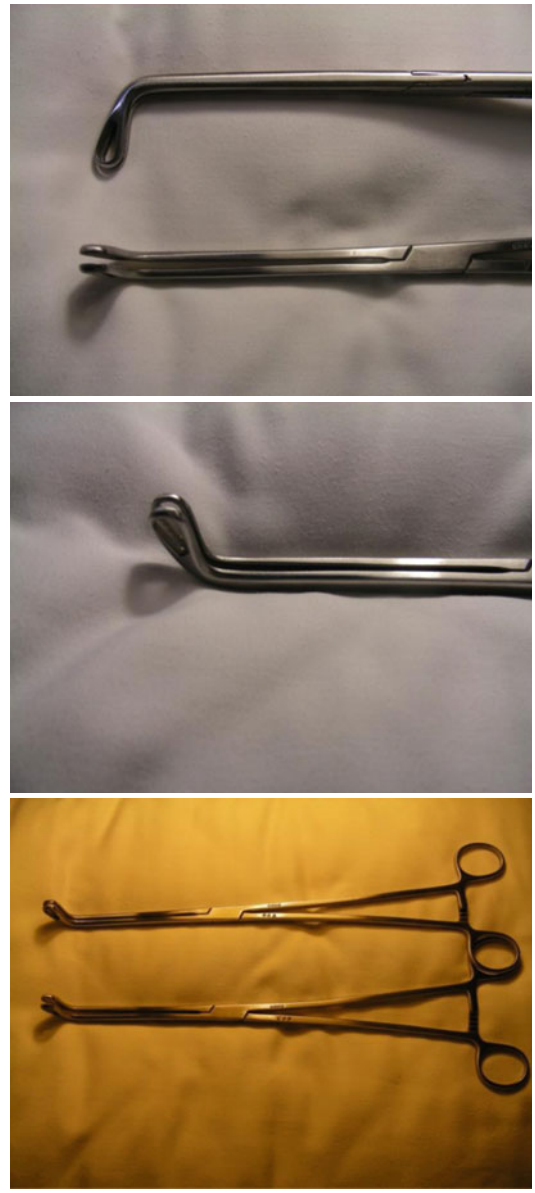

Fig. 17.1 Transvaginal uterine artery clamp

before tightening the blades, the tissue is pushed up so that it will reach up to include the uterine artery of that side.

The concern is that it may occlude the ureters as well. The ureters cross the uterine arteries in the parametrium just lateral to the uterine border. So it is possible that ureters may get caught in the clamp. Since it is only a blunt compression for a few minutes, one does not have to be concerned too much about this ureteric occlusion.

The method is recommended only as a first aid while other definitive measures to stop the bleeding are being assembled. In other words this is only a more effective and easy substitute for bimanual compression of the uterus or aorta.

We have tried this so far in four cases of atonic PPH; in all of them the bleeding immediately stopped. As is to be expected, the uterus will be flabby when the clamp is on, because there is no blood reaching the uterus. Oxytocics may be given intravenously but even that will not act on the uterus until the clamps are removed. While the clamp is on, one should start intravenous lines, prepare other resuscitative measures, and if necessary even shift the patient to the theater. The colleagues like anesthesiologists can be summoned, so that in case conservative measures do not work, one can proceed with surgical procedures without delay. In all four cases mentioned above, further surgical procedures like laparotomy were not required.

The procedure is undertaken on the delivery table. The patient is on dorsal or lithotomy position, in which she has given birth. The additional equipment required is the cervical inspection set consisting of three large bladed Sim's speculums and three large sponge-holding forceps (Fig. 17.2). Anesthesia is not required as the vagina in the immediate postpartum period is quite roomy. Two of the long special clamps for uterine arteries described above are required, one for each side.

With the Sim's speculum the upper vagina is exposed, and the anterior and posterior lips of the cervix are held with the sponge-holding forceps provided in the cervical inspection set. The uterine artery clamp is then inserted with one blade in the cervical canal and the other in the lateral fornix. With gentle upward pressure

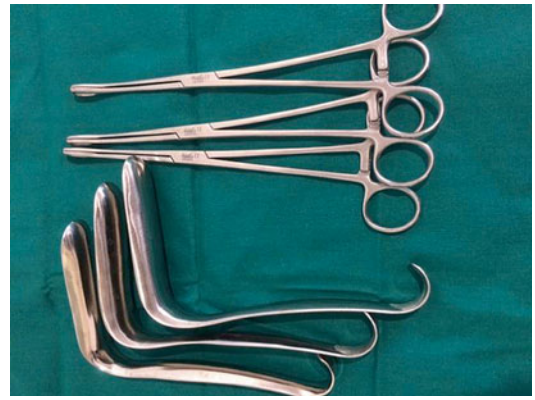

Fig. 17.2 Cervical inspection set

the lateral fornix is pushed up so that the right-angled parts of the blades reach the level of the uterine arteries. The forceps is then clamped. The gap of about 3 mm between the blades right from the box joint to the tip ensures that the soft tissue of the vaginal portion of the cervix will not prevent the fenestrated blades near the tip from occluding the vessels. At the same time, the tissue will not be compressed too much. The tissues being soft and edematous, too tight a compression can break through tissue. The flat, serrated surface and fenestration of the blades and the gap between them even on maximum tightening of the blades are built-in safeguards against the forceps cutting into the tissue.

Lower Genital Tract Lacerations

Lower genital tract trauma is a frequent cause of PPH. There is concern that it may be on the increase due to widespread use of prostaglandins for ripening of cervix and induction of labor. Prostaglandins make the local tissues more friable. This affects the perineum, vagina, cervix, and lower segment. Tears can follow spontaneous vaginal delivery or instrumental deliveries. Usually bleeding from such lacerations is easy to tackle because of the easy accessibility. However, at times it can be quite taxing when the lacerations are extensive and deep or associated with tears involving the lower segment of the uterus. See vignette in box.

Box 1. Vignette: A Case of Extensive Vaginal Lacerations Following Forceps Delivery

A 35-year-old, para 2, was referred to the higher center for severe bleeding following forceps delivery. She had induction of labor with prostaglandin E1, reached full dilatation, and had forceps delivery for delayed second stage. Severe bleeding followed delivery.

There were extensive vaginal lacerations. Suturing was attempted first in the labor room and later in the operation theater. Since the bleeding persisted, she had laparotomy and hysterectomy. Still she continued to bleed. In spite of four units of whole-blood transfusion, she developed hypotension. At that point the vagina was packed and she was sent to the higher center.

At the higher center, after initial resuscitation she was examined in the theater. On removal of the pack, the vagina was found to have extensive lacerations, with multiple sutures, but still bleeding (Fig. 17.3). Attempts at visualization and repair were difficult because of the bleeding. Immediately the abdomen was reopened and bilateral internal iliac artery ligation was done. Following this the vaginal bleeding reduced. The sutures were undone and the vagina was reconstructed. She made an uneventful recovery.

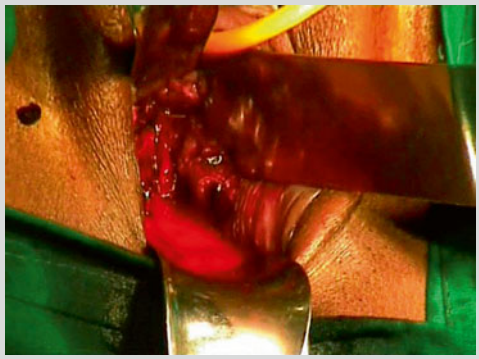

Fig. 17.3 Extensive vaginal lacerations

There are few general principles to be kept in mind, while tackling lower genital tract trauma:

(a) The bleeding will be more in a postpartum woman than in a gynecological setting. This is because of the increased vascularity to genital organs during pregnancy.
(b) The vaginal artery and descending branch of the uterine artery are the main sources of blood supply to the vagina and perineum. In addition there will be blood reaching the lower end through the pudendal arteries, which are branches of the posterior division of the internal iliac arteries. The plexus of vessels around the vagina will have free anastomosis with the branches of the vesical artery supplying the bladder. Since most of the arteries supplying the vagina are branches of the internal iliac artery, bilateral internal iliac artery ligation will help in controlling the bleeding. This will help to get a dry field to suture the lacerations.
(c) While suturing the lacerations the proximity of the rectum behind and urinary bladder in front should be in one's mind. Needles should not be passed too deep anteriorly and posteriorly; otherwise these viscera may be encroached, leading to fistula formation later. Deeper bites on the sidewalls of the vagina have less of this risk.
(d) Restoration of anatomy should be the guiding principle. A layered closure to restore the planes should be attempted. For example, while closing a perineal laceration, the edges of the rectal mucosa, external and internal sphincters, vaginal mucosa, perineal muscles, and finally perineal skin should be approximated. In case of vaginal lacerations, the superficial vaginal epithelium should be kept on the surface as far as possible, lest it can lead to sequestration and inclusion dermoid formation later.
(e) Local application of pressure in the form of packing is an effective way of temporarily arresting the bleeding from vaginal and cervical lacerations. But this should be considered only as a first aid. Anatomical approximation

of tissues should be achieved as early as possible.

A brief description of the various types of lower genital tract trauma is given below from the angle of controlling the bleeding.

Perineal Lacerations and Extended Episiotomy

Bleeding in cases of perineal lacerations is from the branches of the vaginal and pudendal arteries. They may anastomose with branches of the inferior mesenteric artery. If there are spurting vessels, they have to be tied individually or cauterized. However, on many occasions identification of the bleeding points may not be easy. It may be enough to tie the tissue in bulk.

The direction and length of extension of episiotomy are usually unpredictable. Often there is tendency to extend medially involving the anorectum and the sphincters. Careful approximation of tissues and hemostasis is important to prevent hematoma formation and later breakdown leading to fistula and incontinence. An upward extension of episiotomy can involve the rectum. A hematoma can work its way by the side of the rectum going up to the hollow of the sacrum. Evacuation of such hematomas is essential. Usually the bleeders are only at the lower level even when the hematoma has extended toward the sacrum. Evacuation of the hematoma and packing the vagina will ordinarily control the bleeding. However, if the bleeding continues from a higher level, laparotomy may be required.

Hematoma can extend to the vulva from the episiotomy or vaginal lacerations. Our own policy is to evacuate the hematoma, even though many recommend to leave alone smaller ones (less than 2 cm) which are not increasing in size.

Lateral vaginal wall tears may involve the clitoral vessels which are branches of the pudendal artery and ascend up close to the ischiopubic ramus. If the artery is torn, the bleeding can be significant. Identifying and ligating these vessels is important; otherwise it will quickly lead to hematoma formation.

Cervical Lacerations

Cervical lacerations usually occur at 3 o'clock and 9 o'clock positions. If they extend high, the descending cervical branch of the uterine artery will be involved and bleeding can be quite brisk. Tears at other positions also can occur. A particularly significant tear is annular tear, which may even be full circle with the rim of the cervix detaching from the rest. In all tears, repair or reattachment is advisable. From the angle of PPH, small tears (less than 2 cm) can be left alone, but we recommend repair of any tear of the cervix more than 0.5–1 cm in length. This will make the cervix involute and get back to normal shape, rather than remain everted and hypertrophied.

A good cervical inspection set should be available in every labor room. Such a set will contain at least two single bladed speculums and three long broad sponge-holding forceps. These help to expose the cervix. We recommend putting interrupted full-thickness stitches for approximating the edges of the tear. Catgut or delayed absorbable synthetic materials like polyglycolic acid or polyglactin can be used for this.

If the tear has extended deeper than the fornix, it is possible that it has become a ruptured uterus, and laparotomy will be required (see the chapter on rupture of the uterus).

Brace Stitches

B-Lynch published in 1997 his experience with anteroposterior compression of the uterine walls with a single strand of no. 2 catgut [1]. The principle was to compress the uterine walls about 3 cm medial to the lateral border of the uterus on either side and across at the level of the isthmus. This results in compression of the arcuate vessels running through the anterior and posterior uterine walls. The suture running across at the level of the isthmus will compress the vessels running upward from the level of the cervix. These vessels are supplied by the blood vessels joining the uterus from the rectum through the uterosacral ligaments, vagina, and bladder.

Our clinical experience and reports in the literature bring in some problems with the B-Lynch

procedure. First of all, if the suture is really tight, the central segment of the uterus bound by the vertical stitches on either side and the transverse stitch at the isthmus can become ischemic and result in necrosis [2]. There have been case reports of this happening. Another practical problem noted is that the single-strand suture material which has to go through the uterine wall a few times will not tighten with uniform tension in the entire length. In addition, as per original recommendations of B-Lynch, one has to incise the lower segment transversely even if it is not a case of cesarean section, to see if the uterus is empty and whether there is ongoing bleeding even after the stitch. Because of these reasons we usually perform a modification of the B-Lynch brace stitch recommended by Hayman et al. [3].

The Hayman stitch is relatively simple (Fig. 17.4). Vertical brace stitches are taken on either side in the same location as in B-Lynch stitch but are tied separately. The horizontal part at the isthmus is omitted. Chromic catgut (2F) on a straight needle is the ideal suture material, but a curved needle (40 or 50 mm) with no. 1 polyglactin or polyglycolic acid also can be used. The disadvantage of these delayed absorbable materials is that the braided suture may not pass through the tissues as easily as catgut. However it has the advantage of being stronger and less likely to break.

Because of the absence of horizontal compression at the isthmus, any blood collected in the cavity can flow freely to the vagina and be revealed. The minimum number of transcavitary passages reduces the risk of synechiae formation.

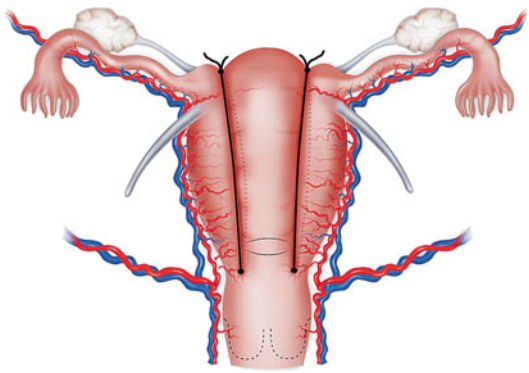

Fig. 17.4 Brace stitch (Hayman's type)

In case the placenta had encroached the lower segment and there are bleeding sinuses, compression can be achieved with square stitches as advocated by Cho [4].

Some have recommended additional anchoring stitches at the fundus to prevent the vertical stitch from falling off from the uterus when it involutes allowing strangulation of the bowel loops which may get trapped inside it. We do not find it necessary. A tight stitch with braided material causes abrasions on the serosa and keeps the suture from falling loose leading to entanglement of bowel loops.

Other Types of Brace Stitches

There have been many modifications of B-Lynch stitches other than Hayman's. The late Dr. S. Gunasheela from Bangalore devised a technique of circular stitches starting from the lower segment up to the fundus compressing the uterus circumferentially. Pereira et al. described a combination of vertical and transverse stitches to compress the uterus [5]. The Hackethal technique is to take the U-shaped stitch compressing the anterior and posterior walls of the uterus [6]. This is popular in France and some European countries.

Failure and Complications

In about 25 percent of cases in large series, the stitch may not control the bleeding even though smaller case reports indicate much higher success rates [7]. There have been reports of uterine necrosis and synechiae formation following brace stitches [8]. Recently Zhang et al. have described a technique of removable stitch application for postpartum hemorrhage [9]. This is with a view to reduce the delayed complications of the brace stitch described above.

Major Vessel Ligation

O'Leary and O'Leary reported in 1966 the technique of mass closure of the uterine vessels in cases of intractable postpartum hemorrhage [10].

However it was Abd Rabbo who came out with the suggestion of stepwise devascularization as treatment for PPH, sequentially ligating the various vessels [11]. He described the technique of ligating the vessels in the serial order of unilateral uterine artery ligation, bilateral uterine artery ligation, low uterine vessel ligation, unilateral ligation of anastomosing branch between uterine and ovarian arteries, and finally bilateral ligation of this vessel. In that report, hysterectomy could be avoided and the patient could be saved in all the 103 cases.

We have been following the technique with minor modifications and have found that the procedure is easy to do and helps in averting hysterectomy in the majority of cases.

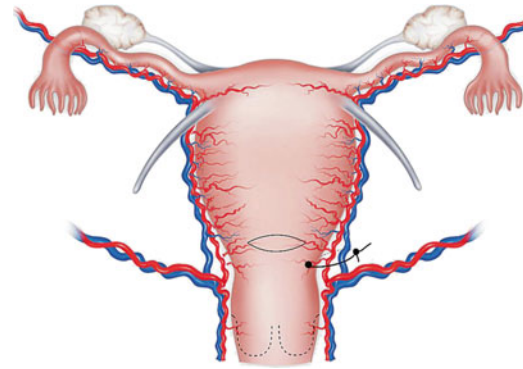

Fig. 17.5 Uterine artery ligation. Uterine artery ligation at the isthmus of the uterus. The stitch should include the vessels, with part of adjacent uterine wall, at the level close to where the uterine artery joins the uterus. The bladder has to be pushed down

Steps of Uterine Artery Ligation

It helps to exteriorize the uterus first and push the bladder down after incising the uterovesical fold of the peritoneum. This displaces the ureter downward and laterally, reducing the risk of injury to it. At the level of the isthmus, about 2–3 cm below the level of the uterine incision for cesarean section, the suture is taken from front to back on the lateral aspect of the myometrium. One has to make sure that there is no bowel or other viscera behind, that may get injured from the needle tip. Another practical point is that if a curved needle on the needle holder is used, the tip of the needle emerging on the posterior surface of the uterus should be held with a clamp before the needle holder is released. Otherwise, there is the risk of the needle receding into the myometrium and the whole process may have to be repeated. Once the needle is pulled through, it is brought back to the anterior aspect by puncturing the broad ligament lateral to the vessels on the side of the uterus but medial to the round ligament. An avascular area to pass the needle can be easily identified by transillumination. I t is important to avoid a vessel puncture because that can lead to a hematoma formation. The lateral myometrium with the bunch of vessels, artery and vein, can then be tied (Fig. 17.5).

The level at which the vessels are to be tied can be modified depending on the situation. If it is a lateral extension of a cesarean wound tearing the uterine vessels, the ligature does not have to be taken very low. However, in that situation the upper part of the torn vessels also will have to be tied above the level of the tear.

On the other hand, if the vessel ligation is to control the bleeding from an atonic uterus, the ligature has to be put lower down close to the point where the uterine artery approaches the side of the uterus. The branch anastomosing with the ovarian artery has to be tied close to the cornual region (described below). Some authors have described putting stitches at different levels on the side of the uterus. We have not found this necessary.

If the uterine artery ligation is done for atonic bleeding, bilateral ligation is essential. On the other hand, if it is for trauma like extension of cesarean section, unilateral ligation will be enough.

Ligation of Anastomosing Vessel between Uterine and Ovarian Arteries

The ovarian arteries, direct branches from the aorta, supply the ovaries and tubes and extend further to supply the cornual region of the uterus (Fig. 17.6). The share of blood supplied to the uterus between the uterine and ovarian arteries will vary depending on the location of

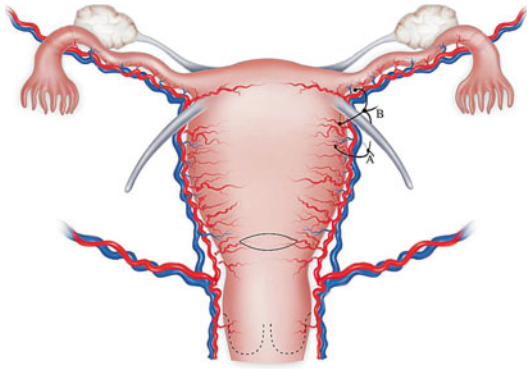

Fig. 17.6 Ligation of anastomosing branch between uterine and ovarian arteries. Tying of the anastomosing branches of uterine and ovarian arteries at the cornual region. (**a**) The usually seen illustration of stitch for the anastomosing branch. Blood flow to the fundus of the uterus will continue unimpeded. (**b**) The recommended technique. The arcade of vessels on the mesosalpinx, from just under the tube to the side of the uterus, is included so that the branches supplying the fundal region are occluded

the placenta. A fundal or cornual placenta will attract more blood from the ovarian arteries.

Ligation of this anastomosing branch at the level of the round ligament as is shown in many textbooks will not achieve the full benefit. At the cornual region just below the insertion of the tube, the arcuate artery supplying the cornual and fundal regions will take off. So it is important to occlude the branches of the ovarian artery close to the cornual region and the ascending trunk of the uterine artery. These can be achieved in one ligature, provided care is taken to avoid injury to the vessels with the needle tip. This is possible if one transilluminates the area and takes the first bite close to the tube between the tube and the vessel, and then the needle is made to pass through the cornual myometrium before tying the knot (see picture).

When done on both sides, ligation of the uterine and ovarian branches should take care of most of the blood supply to the uterus. Still the uterus will not undergo ischemic necrosis because of the blood reaching it at the level of the cervix through uterosacral ligaments, vaginal vessels, and bladder vessels.

Ligation of the Descending Cervical Branch of the Uterine Arteries

The uterine artery after reaching the side of the uterus sends off a branch downward to supply the cervix; this branch anastomoses with the vaginal artery. In case there is a tear of the cervix or upper vagina, ligation of this branch will help to reduce the bleeding.

Access to this vessel is a little more tricky. The bladder will have to be pushed well down. There is the risk of bleeding from the plexus of vessels in the parametrium and chance of injury to the ureter is greater. So ligation of this vessel is not recommended unless there is trauma as described above. To ligate this vessel, push the bladder well down, take a part of the cervical myometrium, and include the vessel with the surrounding parametrium, making sure that the ureter is not injured.

Internal Iliac Artery Ligation

Almost all the vessels supplying pelvic organs arise from the internal iliac artery. Hence, ligation of the internal iliac artery, bilateral or unilateral, depending on the indication, became a standard step in any heavy intractable pelvic bleeding. The clinical scenario for this procedure has however changed recently. In our own experience, with the adoption of brace stitches and stepwise devascularization of the uterus and temporary clamping of the aorta or common iliac artery described in this chapter, the incidence of internal iliac artery ligation has come down dramatically. We now use it for traumatic bleeding from the vagina or cervix, rather than atonic PPH. Another occasion to use it is as a backup to prevent reactionary hemorrhage after obstetric hysterectomy for placenta previa accreta.

Anatomical Considerations

Anatomy of the internal iliac artery and its relation to nearby structures vary between individuals. Soon after its origin the internal iliac artery gives off the posterior division to supply the gluteal region. The ventral division continues

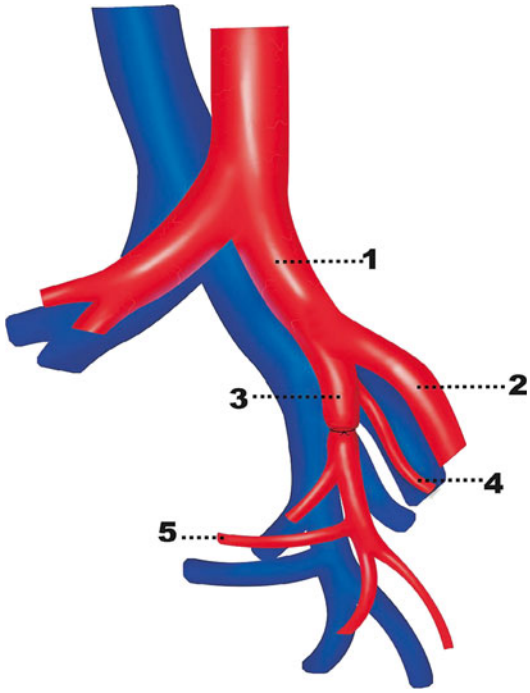

Fig. 17.7 Anatomy of the internal iliac artery. The internal iliac artery and its branches: (*1*) common iliac artery, (*2*) external iliac artery, (*3*) internal iliac artery (anterior division), (*4*) internal iliac artery (posterior division), and (*5*) uterine artery

downward and medially to give off its branches that supply the uterus, urinary bladder, and vagina (Fig. 17.7). The most common complication in attempting to tie the internal iliac artery, especially by the novices, is injury to the adjacent veins. Internal and external iliac veins run close to this artery. While dissecting one has to keep this in mind to avoid injury to the veins. In a patient in shock, which may be the scenario when internal artery ligation is contemplated, the veins may be collapsed, and unless one keeps these anatomical facts in mind, it is easy to injure the veins.

On the left side the presence of the mesosigmoid adds another problem especially while trying to approach the internal iliac artery directly. If the vessel is identified by palpation of the pulse and approached directly taking care of the vessels in the mesosigmoid, access should not be a problem. Moreover if one follows the broad ligament approach described below, accessing the internal iliac artery on the left side should not present much of a problem.

Often it is emphasized to ligate the internal iliac artery after the posterior division of the artery takes off. This is to avoid ischemia to the gluteal muscles in case the posterior division is occluded. However, this should be a rare complication as the collaterals on the back with branches of the lumbar vessels should prevent such ischemia. Still, it will be better to tie the vessels about 2–3 cm below the origin of the internal iliac so that the risk of occluding the posterior division is reduced.

Procedure

There are two ways to approach the internal iliac arteries – direct approach and the broad ligament or indirect approach. The author favors the broad ligament approach whenever it is possible. After hysterectomy and in nonpregnant women, direct approach is required.

In the broad ligament approach, the uterosacral fold is incised and extended laterally toward the round ligament. Palpation of the external iliac pulsation and psoas muscle will help to identify the direction of dissection. Blunt dissection, separating the leaves of the broad ligament, will take one to the external iliac arteries. Similar dissection cranially following the external iliac artery will take us to the common iliac artery after which the internal iliac artery can be identified. The ureter usually crosses at the bifurcation of the common iliac artery into external and internal. We prefer to keep the ureter shifted medially. Once the internal iliac is identified, retraction of the intact peritoneal leaves of the broad ligament will help to develop the space without the bowel and omentum coming in the way.

In the direct approach, the internal iliac artery is identified by locating the common iliac artery tracing it down from the aorta and then down to the internal iliac artery. The peritoneum over the internal iliac artery is then held with an Allis forceps and incised vertically for a distance of about 3–4 cm. The vessel is then exposed and location for ligation is identified.

Once the location for ligation on the internal iliac artery is identified (about 2–3 cm below its

origin), the areolar tissue on its surface and on the sides are gently pushed aside with the tip of a long artery clamp. The tip of a right-angled clamp (Mixter clamp) is then negotiated under the artery from one side till its tip is seen on the other side. The clamp is used to gently lift the artery from its bed while it is negotiated. The tip of the suture material is fed to the open Mixter clamp and then pulled out from under the artery (Fig. 17.8). Some have recommended the use of a Babcock forceps to lift the internal iliac artery to facilitate the passage of the clamp underneath. We have found that as a useful step, especially in the deep pelvis.

There is divergence of opinion regarding suture material. Our recommendation is to use non-braided absorbable suture material. In practical terms no. 1 catgut will be the usual choice. But even a braided suture made of polyglycolic acid or polyglactin can be used. We will recommend against suture materials like silk, prolene, etc.

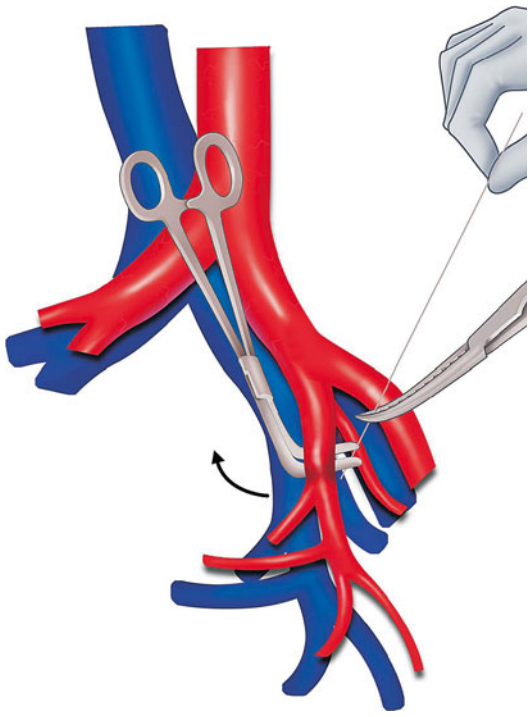

Fig. 17.8 Ligation of the internal iliac artery. Taking sutures under the internal iliac artery with right-angled clamp. The Mixter clamp is negotiated under the internal iliac artery without injuring the veins. The tip of the thread is fed to the open clamp with the help of a long forceps

Complications

The most dreaded complication is injury to the adjacent vessels, especially external iliac veins. If it happens it has to be repaired as otherwise it can lead to exsanguination, shock, and cardiac arrest. Other structures that are reported to be injured include the ureter, external iliac artery, nerves on the pelvic sidewall, and vessels of the mesosigmoid.

There have been rare reports of ischemic pain or necrosis of gluteal muscle. This happens when the posterior division of the internal iliac artery is tied off and there is no effective anastomosis from the lumbar vessels. Ligating the internal iliac about 2–3 cm below its origin will reduce the chance of this complication.

Temporary Occlusion of Lower End of Aorta or Common Iliac Arteries with Special Atraumatic Clamp

This proposal is a total deviation from the traditional recommendations for managing PPH. In case one is faced with a patient who is in shock or is bleeding so profusely that shock is anticipated any moment, the first response should be to stop that bleeding. If it is in an institution and the bleeding is from an atonic uterus, the effective first aid that can be implemented fastest is to apply transvaginal uterine artery clamps (see description above). But if that stage is over and the decision is to proceed with laparotomy, we have to stop the bleeding immediately. The recommendation is that the caregiver should apply aortic compression. This is difficult especially when the patient is obese. The aim should be to compress the aorta against the lumbar vertebra. It causes discomfort for the patient and is tiring for the operator. If the need arises during a cesarean section, it is easier as the patient is under anesthesia and the open abdomen helps to apply the pressure directly to the aorta. However this cannot be continued effectively for long. The atraumatic clamp developed by us

primarily to occlude the aorta or common iliac arteries in tackling placenta previa accreta (see description below and Fig. 17.9) can be used temporarily to occlude the aorta while the patient is being resuscitated.

As soon as the anesthesia and arrangements are ready, the abdomen should be opened through a midline incision which may go above the umbilicus if necessary. The aim is to exteriorize the uterus and pack the bowels to expose the lower end of the aorta. Alternatively the common iliac arteries also can be targeted but the former may be better and easier. The aim is to directly apply an atraumatic clamp to occlude the lower end of the aorta (just above the division into common iliac arteries). By palpation this segment of the aorta can be easily identified. A Babcock forceps can be applied directly to the aorta to gently lift it up. The special atraumatic clamp can then be applied to include the aorta so that the distal tips go beyond the posterior wall of the aorta. Applying the clamp like this does not need dissection to isolate the aorta, a difficult and dangerous task under the circumstances. The clamp is provided with long ratchet which is tightened only to the extend the pulsation in the common iliac arteries stops thus avoiding crush injury of the aortic walls. As an additional precaution the forceps is designed to leave a gap between the blades even when the clamp is maximally tightened. This will prevent ischemic damage to the vessel walls.

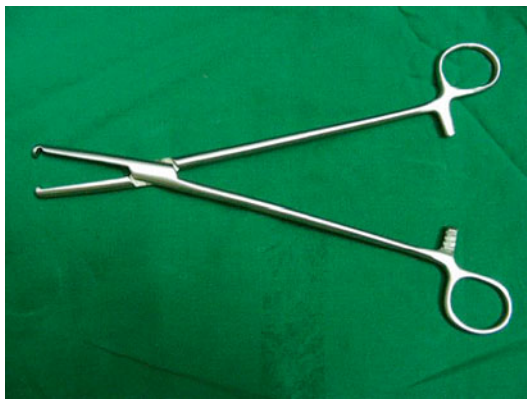

Fig. 17.9 Atraumatic common iliac/aorta clamp designed and patented by the first author

In addition to clamping the aorta or common iliac arteries, the blood supply to the uterus coming through the ovarian vessels directly from the aorta also has to be controlled. The same type of clamp can be used to occlude the infundibulopelvic ligament, thereby occluding the ovarian vessels. It is better to include the ipsilateral round ligament also in the clamp to cushion the vessels and avoid injury to the ovarian veins.

Clamping the aorta as described above cuts off the blood supply to the pelvis and lower limbs. This is time sensitive and we should try to reestablish circulation as early as possible and in any case within about 90 min. The surgeon should be reminded of the time elapsed every 5 min. When the tissue is deprived of blood supply, anaerobic metabolism will continue and metabolic acidosis will ensue. When the clamps are released, then metabolites may get back to circulation with possibility of systemic acidosis. The anesthesiologist should be watchful for this. Another concern is whether there will be increased risk of thrombosis. In any case, such morbid postpartum phase will increase the risk of thrombosis. We recommend thromboprophylaxis in the postpartum period once the acute bleeding phase is over and patient is stable.

Clinical Scenarios where These Clamps Can Be Used

These clamps were developed to tackle placenta previa accreta in previous cesarean sections which will be described later under obstetric hysterectomy. The other clinical situation in which this strategy is recommended is in patients who are detected late and are in shock or on the verge of it. Similar situations can arise during cesarean section or while tackling complications like ruptured uterus or inversion of uterus. The aim is to arrest the rapid deterioration and allow the anesthesiologist time to resuscitate the patient. At the same time the obstetrician also gets time to arrange for blood, summon additional manpower, and plan definitive steps.

Obstetric Hysterectomy

This is the nonconservative management of PPH. All types of PPH, primary or secondary, as well as atonic and traumatic, can lead to this. However, the two most common clinical settings ending in obstetric hysterectomy at present are the placenta previa accreta and uncontrolled or late-recognized atonic PPH with patient in shock or coagulation failure. There are other reasons for obstetric hysterectomy unrelated to PPH like fibroid complicating pregnancy or rarely carcinoma cervix which do not come under consideration here.

By definition obstetric hysterectomy refers to removal of the uterus during pregnancy or within 6 weeks (puerperium) postpartum. The procedure will have to be modified depending on the general condition of the patient and the indications. For example, a patient already in shock demands a different approach compared with somebody who is still stable and with a proper resuscitation team around. In most occasions time will be the critical element; there will be the need for finishing off the procedure quickly. A policy of clamp, cut, and drop is followed till the uterine arteries are clamped. However, if it is not such an emergent situation, one can proceed the same way as in abdominal hysterectomy for gynecological indications. But since the stumps are thicker and edematous, double ligation of stumps is recommended to prevent slippage of the knot or reactionary hemorrhage.

Removal of the cervix should be based on the indication. Even a few extra minutes required to push the bladder down to the level of the vagina and remove the cervix can be critical in a collapsed patient. More importantly, one is opening up the vascular bed with potential for primary and reactionary hemorrhage when the plexus of vessels under the bladder communicating with the vaginal vessels is considered. Another additional reason to retain the cervix is the ease with which suturing is possible when the thicker and firmer cervix is available compared with the thinned out and friable vagina. So our policy is to routinely do only subtotal hysterectomy except when there is a cervical cause of bleeding like tears of the cervix. Even in placenta previa accreta done electively, this is the policy (see below).

A typical case scenario is one of PPH where the patient has gone in for shock and DIC and a relatively late decision is taken to do hysterectomy. Time taken for completing the procedure is critical here. General anesthesia should be the obvious choice. The surgeon should opt for a vertical midline incision, usually below the umbilicus. The uterus is quickly exteriorized. We would immediately put atraumatic hemostatic clamps in the lower end of the aorta and infundibulopelvic ligaments. Clamps are sequentially applied on either side of the uterus for the upper pedicles. After incising and pushing down the uterovesical fold of the peritoneum with the bladder, clamps are applied on both sides for the uterine arteries. The blood supply to the uterus is now cut off except for the few anastomotic vessels with the bladder, vagina, and rectum.

We would strongly recommend subtotal hysterectomy leaving a small part of the lower end of the cervix intact in all cases of atonic PPH. This policy is followed unless the cause of bleeding is in the cervix. In addition, since the bladder dissection is less, the bleeding from base of the bladder will also be less. Our practice is to take horizontal mattress stitches for the cervical stumps. The pedicles are usually double ligated. We leave a wide-bore drain to warn us about any reactionary hemorrhage.

Hysterectomy for Abnormally Invading Placenta Previa (Placenta Previa Accreta)

Tackling placenta previa accreta is a nightmare, whether you work in a resource-poor or resource-rich setting. The basic issue is the uncontrollable bleeding that floods the operative field during surgery. This torrential bleeding is due to a variety of reasons:

1. There is extra blood supply to the lower segment through new arteries.
2. These new blood vessels lack the inherent ability to contract and stop bleeding as they are devoid of muscular wall.

3. The lower segment tissues are more friable.
4. The placenta will be abnormally implanted, penetrating deeper into the uterine wall or even beyond so that it is difficult to separate it from the uterus.
5. The additional blood flow comes through vessels which are not controlled by procedures like brace stitch and internal iliac artery ligation.
6. The traditional hemostatic steps like oxytocics are not as effective in this setting of lower segment bleeding.

The usual clinical scenario is like this – the surgeon tries to separate the bladder from the lower segment; it starts the bleeding which is usually so torrential that it overwhelms the ability to clear the field with suction or mops. This leads to panic reaction. Further attempts to deliver the fetus through the operative field flooded with blood result in injury to the bladder, irregular extension of the uterine incision, etc. Attempts to reach the baby are hindered by the placenta which occupies part of the space. Before one realizes it, the patient would have lost so much of the blood volume that she goes into vascular collapse and cardiac arrest. The shock and metabolic acidosis lead to disseminated intravascular coagulation, perpetuating the bleeding, and consequent deterioration and death.

The key to avoid this course of events is to prevent the uncontrollable bleeding in the first place. The second strategy is to replace the blood rapidly so that she does not go into hemodynamic collapse and its consequences. If facilities like cell salvage are available, the lost blood is retrieved and reused.

The Standard Practice Followed in Resource-Rich Settings Is Somewhat like This

Definite protocol is available to manage these cases. They are tackled only in centers where there are adequate resources and manpower. Typically these designated centers will have the services of senior obstetricians, anesthesiologists, vascular surgeons, oncosurgeons, urologists, interventional radiologists, transfusion specialists, and technicians to handle cell salvage and other machinery required.

Large amounts of blood and blood products will be readily available. Critical care setting will be ensured for follow-up care. Multiple wide-bore IV lines and central lines for monitoring are put prior to surgery.

Usual sequence of events is to plan the surgery as an elective procedure between 34 and 38 weeks. All the abovementioned specialists will be available. The ultrasound scan or MRI would have shown the location of the placenta and how deep the invasion is especially in relation to the bladder.

In many centers the interventional radiologists put in balloons into the internal iliac vessels or aorta and keep it ready for inflation as soon as the fetus is delivered.

A vertical abdominal incision followed by incision on the uterus above the level of the placenta is used to deliver the fetus. The intravascular balloon is then inflated. The decision is made whether to proceed with hysterectomy or local resection of the abnormally invading cotyledon and retain the uterus for future childbearing. If the decision is to do hysterectomy, sequential clamps are applied until the uterine arteries are reached. The bladder is then separated from the lower segment and hysterectomy is completed. If on the other hand the decision is to retain the uterus, the placenta is separated manually. When it comes to the adherent cotyledons, a part of the uterine wall also may have to be removed, and gap in the uterine wall is closed with sutures. Additional hemostatic procedures like brace stitches may be used to prevent subsequent bleeding.

In a resource-poor setting such preparations may not be possible. Hence we have developed the following strategy to deal with placenta previa accreta.

It is impossible to assemble so many specialists, equipments, blood, and blood products in a resource-poor setting. The outcome of management is usually grave and often fatal. But the need to prevent the uncontrollable bleeding is

everywhere. We reasoned that if only the common iliac arteries or the lower end of the aorta is occluded temporarily after delivery of the fetus, dissection to separate the placenta can be undertaken in a relatively clear field.

But such vascular clamps that can be used by an obstetrician to occlude the aorta or common iliac arteries were not available. So we designed and produced such a clamp (Fig. 17.9) and piloted its use. These were atraumatic clamps which could be applied by the obstetrician after pulling up the concerned vessel with a Babcock forceps. This is applied after the fetus is delivered and can be kept in place for about 60–90 min.

The usual procedure is reproduced here with permission, from the book "Why Mothers Die, Kerala" (2006–2009 second edition) [12].

Stepwise Approach to Placenta Previa Accreta

1. Establish the diagnosis and extent of placental invasion.
2. Counsel the patients and relatives about the seriousness of the situation without frightening them.
3. Plan surgery (cesarean) as an elective procedure.
4. Arrange enough blood and blood products.
5. Ensure presence of experienced obstetrician and urologist (if obstetrician is not confident enough to tackle a potential urological injury).
6. Insert bilateral ureteric catheters and a Foley catheter in the bladder.
7. Use regional anesthesia or regional with general anesthesia for the surgery.
8. Use vertical incision on the abdomen extending above the umbilicus and classical incision on the uterus above the level of the placenta.
9. If accreta placenta is confirmed on seeing the very vascular bulge of the lower segment, decide whether to do hysterectomy without disturbing the placenta or leave the placenta and uterus in situ. Do not try manual removal.
10. If decision is to leave the placenta behind, tie the cord close to the placenta and remove the excess length of cord, close the uterus, and come out leaving the placenta in situ.
11. If the decision is for hysterectomy, apply tourniquet to both infundibulopelvic ligaments and occlude the blood flow through common iliac arteries (lower end of the aorta) with specially developed clamps. Proceed with hysterectomy, up to the level of uterine arteries.
12. By sharp dissection separate the bladder from the uterus and do a subtotal hysterectomy leaving behind part of the cervix below the level of the placenta. Avoid unnecessary separation of the bladder from the vagina.
13. Double ligate all the pedicles.
14. Remove occluding clamps and tourniquets and ensure that circulation to lower limbs is reestablished by palpating the femoral pulse.
15. Close the abdomen after leaving a wide-bore drain. Time elapsed from the time of common iliac clamps should be announced every 5 min. Try to complete the procedure in about 30–40 min. Keep the patient under close monitoring in a high dependency unit.

Since the publication of the above procedure, we have used this method in more than 50 cases with a dramatic reduction of blood transfusion requirements and bladder injury. There were no maternal deaths in the cases where the above strategy was followed.

It is possible to do resection of the abnormally invading cotyledons and save the uterus for future childbearing as reported by Palacios J et al. [13], but we have not had an occasion to do so till now.

Which Procedure to Choose

Faced with acute hemorrhage, the obstetrician often is in a dilemma regarding what course to take or which surgical step to choose. The first step should be to arrest the bleeding as quickly as possible. This often will involve steps of first aid like transvaginal uterine artery clamp or pack or tamponade with balloons or condom catheter. The definitive surgical steps will depend on the clinical situation. Perineal, vaginal, or cervical lacerations may be tackled in the labor room

itself. If the lacerations are complex or extending deeper, taking the patient to the theater and tackling under anesthesia in the operation theater may be better.

If bleeding is severe and persistent, taking her to the theater becomes mandatory. Laparotomy with a vertical midline incision is advisable. Our own practice is to choose between brace stitches and stepwise devascularization depending on the case scenario. If the uterus is atonic, we immediately go for brace stitch of the Hayman's type, whereas if the uterus has a tendency to contract and relax, stepwise devascularization is chosen. Often the two steps may be combined.

Internal iliac artery ligation is usually employed for traumatic PPH. In cases with DIC already set in, internal iliac ligation should be undertaken only after weighing the pros and cons – the advantages of reducing bleeding from the already opened vascular channels and the disadvantage of opening up new tissue planes with inherent risk of additional bleeding.

In atonic PPH which has already resulted in DIC, the decision to do a quick subtotal hysterectomy versus replacement of coagulation factors and correcting the coagulopathy to save the uterus with additional conservative surgeries can be a challenging one. The age of the patient and whether the family is complete are factors that will influence the decision. However top priority should be given to arrest the bleeding which can be lifesaving. As is said, it is better to live without the uterus than to die with the uterus.

Experience related to postpartum hemorrhage from Mother Hospital, Thrissur, Kerala 2005–2014

Total number of deliveries	20,850
PPH	215
Medical management	148
Conservative surgical management	50
Hysterectomy	17 (for atonic 5, placenta previa accreta 12)

Conclusions

Making a choice of conservative and nonconservative surgeries to arrest PPH can be challenging even to the most experienced obstetrician. Those in training should keep the skill updated by practicing on manikins. New developments like transvaginal uterine artery clamps and atraumatic common iliac/aortic clamps are lifesaving additions to the obstetrician's armamentarium. It is essential that the surgical skills are maintained and passed down to the junior colleagues who are more likely to be on the scene when the PPH arises. Timely intervention with conservative steps will help to save the uterus and even the life of the parturient.

Summary

The various surgical steps that are available to arrest postpartum hemorrhage are described in this chapter. Two new innovations – transvaginal uterine artery clamp and atraumatic common iliac artery/aortic clamps – are presented. Conservative surgical steps like brace stitches, devascularization of the uterus, and internal iliac artery ligation are described. Obstetric hysterectomy especially for placenta previa accreta with the use of atraumatic common iliac artery/aorta clamp is presented in detail. What is crucial is the timely use of these steps before the patient slips to shock and multiorgan failure.

Acknowledgment We are grateful to the management and colleagues at Mother Hospital, Thrissur, especially Dr. Philo Akkarapatty and Mrs. Suseela, head nurse in charge of labor room. This report is based on the experience gained from that hospital in managing postpartum hemorrhage. The initial trials of the transvaginal uterine artery clamps as well as the common iliac/aortic clamps were done in the same hospital.

Figures 17.4, 17.5, 17.6, 17.7, and 17.8 are reproduced from the book "A Comprehensive Textbook of Postpartum Haemorrhage, 2nd Edition," edited by Arulkumaran S, Karoshi M, Keith LG, Lalonde AB, and B-Lynch C, Chapter 53 "Initial Interventions to Combat Hemorrhage During Cesarean Section and Internal Iliac Artery Ligation" (by V P Paily). I am grateful to Sapiens Publishing who have given free permission for reproduction of the contents for educational purposes.

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18 The Lower Segment of Uterus – A Critical Area in Childbirth and Resulting Trauma

Ajit C. Rawal

Introduction

Contraction/retraction are considered essential prerequisites for both placental detachment and postpartum hemostasis, but this physiological hemostasis is not well established at the lower segment of the uterus, and it is worst in the case of lower segment cesarean section (LSCS) because of surgical scarring. Thus a new, clinically important class of postpartum hemorrhage (PPH) is "lower segment PPH" (atonic/traumatic). However, one clinical observation is that hysterectomy is not necessary in all patients with central placenta previa. The only possible conclusion is that there may be qualitative and quantitative differences in the musculature of the lower segment in different patients.

We Obstetricians Never Thought of Certain Aspects of the Lower Segment

1. Length of the lower segment
2. Broadness (width) of the lower segment in the upper and lower parts and during different trimesters of pregnancy
3. Thickness of the lower segment
4. Vascularity of the lower segment
5. Relation of the lateral vascular bundle to the lower segment
6. Relation of the bladder and peritoneum to the lower segment
7. How it heals and behaves after LSCS
8. Involution of the lower segment

A recent literature search on this topic confirms that the nature and origin of these differences have never been investigated.

Controversy Was Centered on the Subject of the Origin of the Lower Segment

There are three main views on the origin of the lower uterine segment in pregnancy:

1. The isthmus (first described by anatomist Aschoff in 1906) [1], is that part of the cervix lying between the anatomical internal os above and the histological internal os below. Stieve has shown that after the second month the isthmus is opened up and expanded so as to become part of the corpus [2].
2. The second view is that the lower segment is formed from the lower part of the corpus only, the cervix not contributing any part of it (Barbour, Von Franque) [3, 4]. Its upper limit is the firm attachment of peritoneum on the

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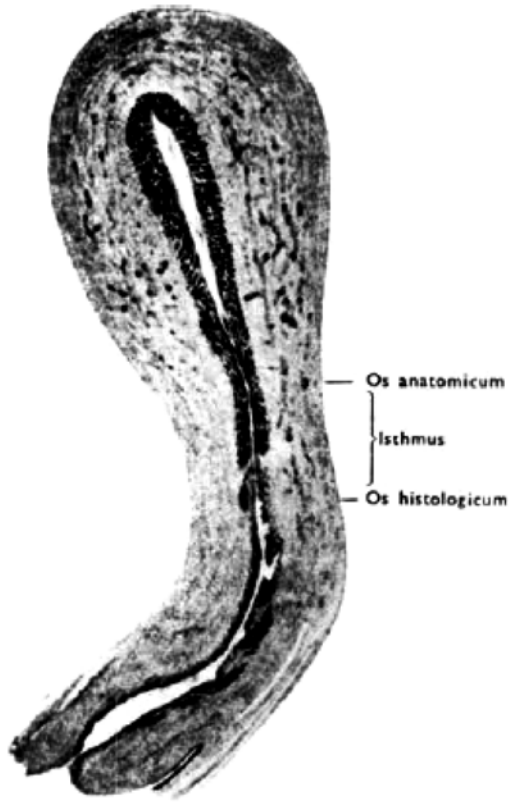
front of the uterus which is 9–12 mm above the internal os.

3. The third possible view is that the lower segment is formed both from the isthmus and from the lower part of the corpus, the part that is corresponding to the area of loose attachment of peritoneum.



Drawing which shows the site of the isthmus together with its upper and lower boundaries – the os internum anatomicum and the os intenum histologicum (Aschoff).

Aschoff (1906)

The isthmus of the uterus is located between the anatomical internal os and the histological internal os of Aschoff. From the end of the second month of pregnancy onward, unfolding occurs and the lower segment becomes part of the corpus.

The histological internal os becomes the internal os of the full-term pregnant uterus. According to Stieve [2], it is not mere stretching but actual growth, the histological internal os being the point at which the lining of the lumen changes from the endometrial to the endocervical columnar epithelium. Both these landmarks are luminal and have no identifiable analogs on the serosal surface of the uterus except for the attachment of the loose peritoneum.

The lower segment would be formed from the lower part of the body of the uterus, corresponding to the area of loose attachment of the peritoneum (Barbour, 1907) [5].

The Lower Segment

The lower segment is formed from the isthmus and the part of the body corresponding to the area of loose attachment of peritoneum. The formation starts taking place between the 70th and 100th day after the LMP. The important point is that if a lower segment hysterotomy is attempted on the 70th day an incision made below the line of firm attachment of the peritoneum to the uterus enters the cervical canal slightly lower at the vault of the vagina, but on the 100th day a similar incision is found to enter the lower part of the uterine cavity.

Dan Forth Described

Regarding the lower uterine segment, the uterus has two parts, the corpus and the cervix, and the fibromuscular junction is situated between the muscular corpus and the predominantly fibrous cervix [6]. This junction lies at the internal os of the nonpregnant uterus and migrates up the wall of the uterus during pregnancy. This concept more logically explains why so many low-lying placentas are nothing of the sort, and certainly not previa by the end of pregnancy.

Nowadays, all obstetricians accept the concept of a "lower uterine segment" because it is so firmly established in obstetric teaching. The difficulties that we face regarding this subject are of defining either clinically or by imaging the upper boundary of the "lower uterine segment" (Munro Kerr's Operative Obstetrics) [7].

The thing to understand is that at term the upper part of the lower segment is about 10 cm above the internal os. The area of loose attach-

ment of the peritoneum on the front of the uterus and upper limit is marked by the firm attachment of the peritoneum and corresponds to the site of the retraction ring of Barbour. The lower segment is thinner than the upper segment and the muscles fibers are arranged more or less transversely.

In labor, the lower segment is passive and takes no part in the contractions of the upper segment, but it does stretch and become part of the birth canal. This stretching is facilitated by the fact that the cervix is more or less fixed by its attachments (Munro Kerr), a phenomenon called receptive relaxation.

Retraction Ring

This was first described by Bandl in 1875; thus, it is known as "Bandl's ring." The fact that it was due to excessive retraction of uterus was first recognized by Barbour; thus it is also referred to as the retraction ring of Barbour. It is a ring-shaped furrow formed at the junction of the contracting and retracting upper segment and the passive lower segment.

It is also formed in normal labor and lies about two fingers above the upper part of pubic hair but is not palpable. However, in obstructed labor due to excessive retraction and contraction (intermittent) the lower segment becomes stretched and the fetus is pushed into the lower segment. It a state of imminent rupture of the lower segment and Bandl's ring can frequently be palpated at or near the navel.

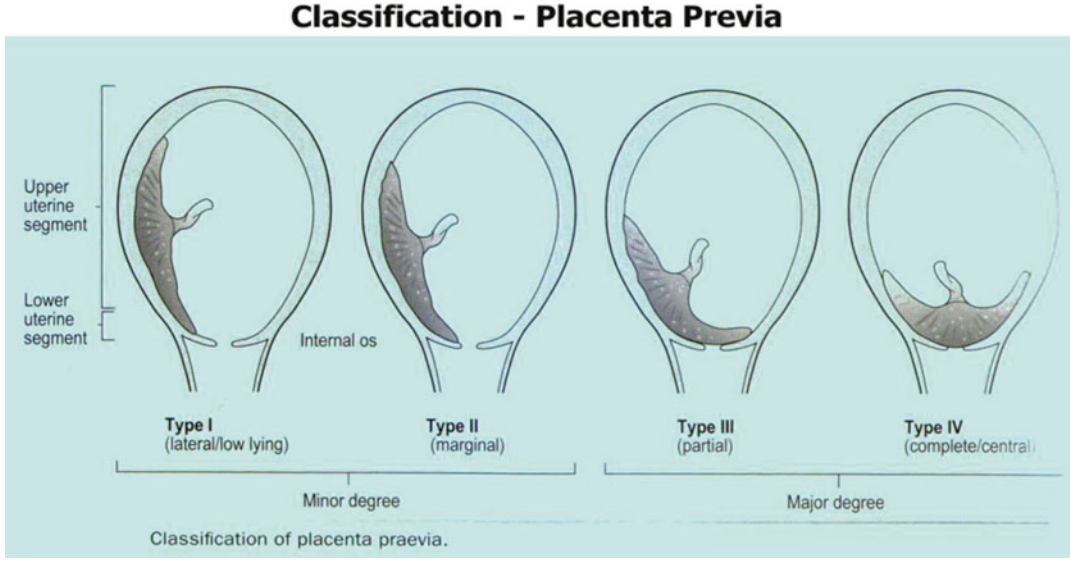
Placenta Previa

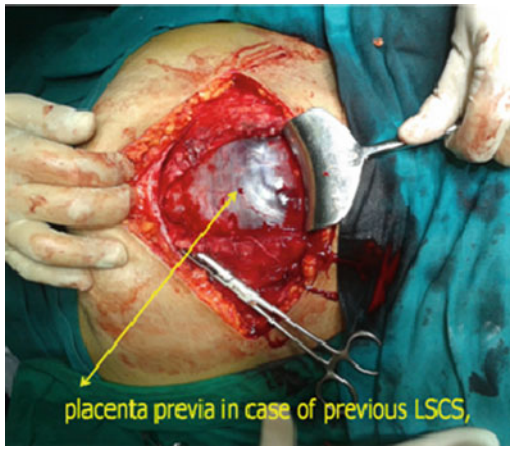
This defined as part or all of the placenta that is implanted in the lower uterine segment.

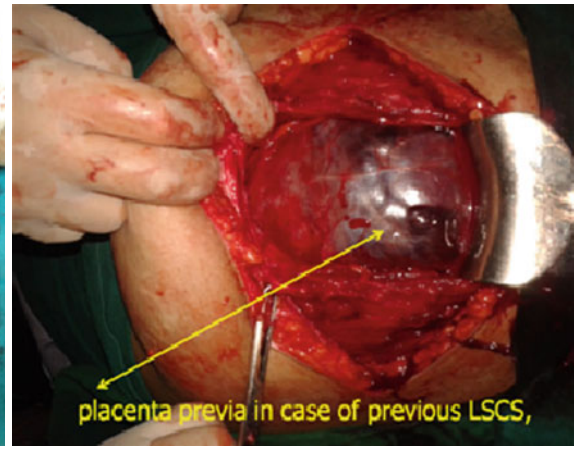
As the lower segment develops, the placenta attached to the upper uterine segment appears to move up and away from the internal os, which is called placental migration. Thus by the first trimester in 20%–30% cases, the placenta remains low-lying as a true placenta previa.

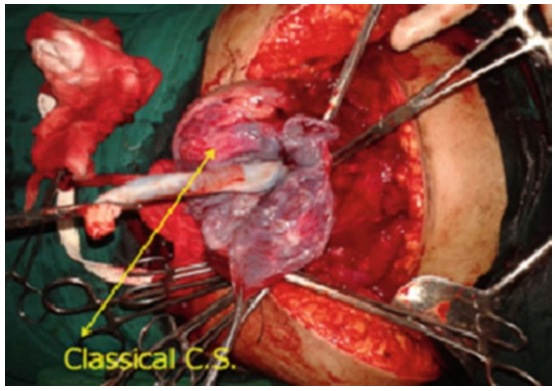
About 80 % of all women with a placenta previa bleed before the onset of labor. Major cases of placenta previa tend to bleed earlier in gestation, more frequently and more heavily than minor cases, but sometimes, in complete placenta previa, the woman may not bleed until the onset of labor. Bleeding from the lower segment is painless and inevitable. The first occurrence may not be serious and is mostly at night.

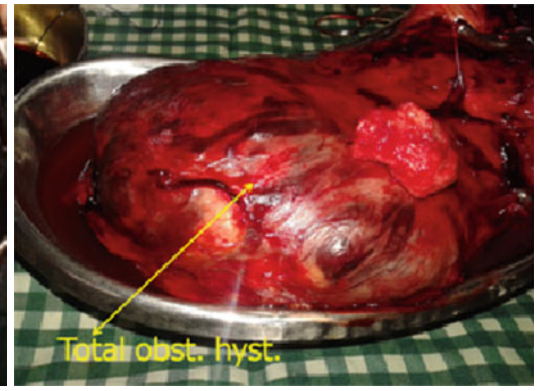
The biggest problem is that in case of placenta previa with previous LSCS, there is 10% chance of the placenta being adherent to the uterine musculature (accrete, increta and percreta) and may be one of the three varieties, accreta, increta or percreta. Ultrasound, color Doppler, and MRI definitely aid diagnosis, and management planning can be done. The incidence rises related to the increased number of women delivered by cesarean section.

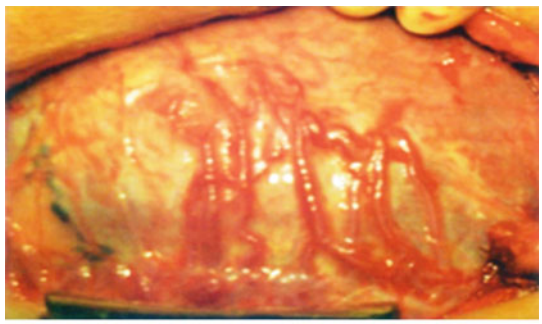




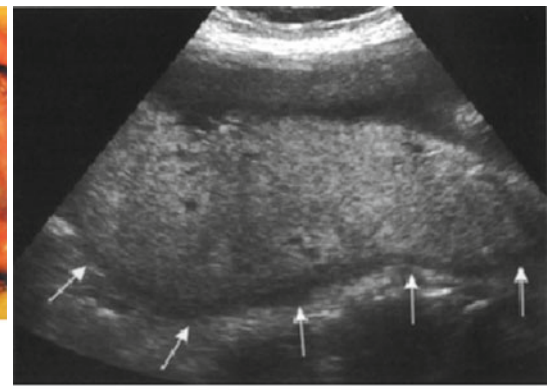




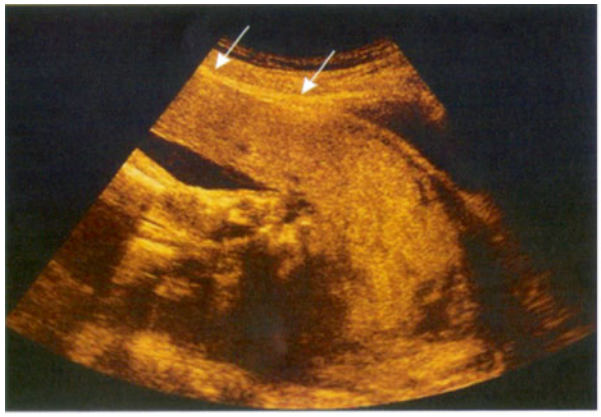




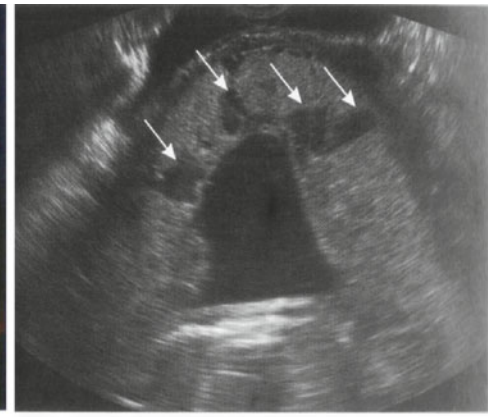
Intraoperative image of placenta percreta.



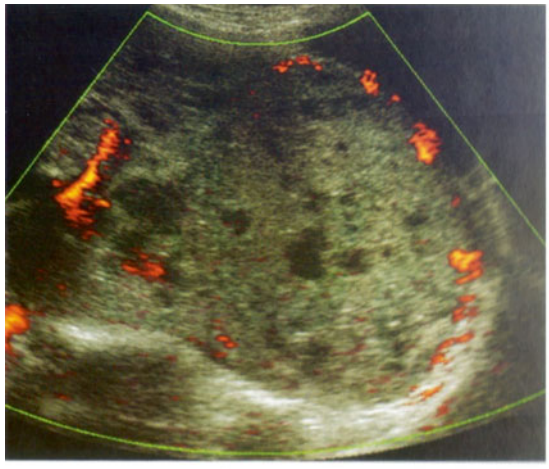
Retroplacental hypoechoic zone in normal placenta.



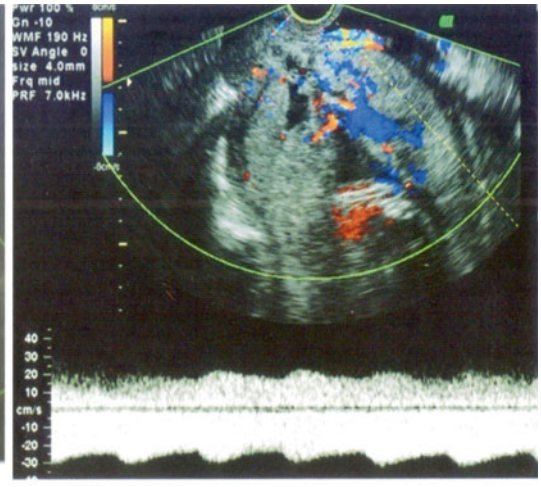
Absent retroplacental zone in adherent placenta.



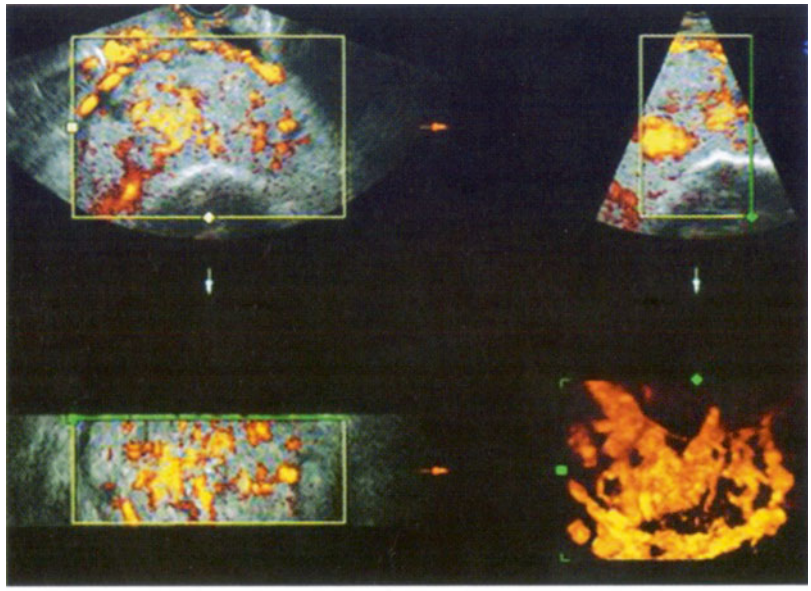
Intraplacental lacunar spaces.



Periplacental vascularity and interruption of bladder uterine interface.



Vascular lakes with high velocity low resistance flow.



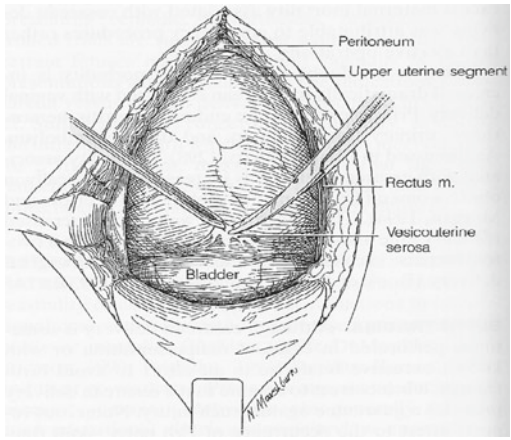
Chaotic branching on power angio.

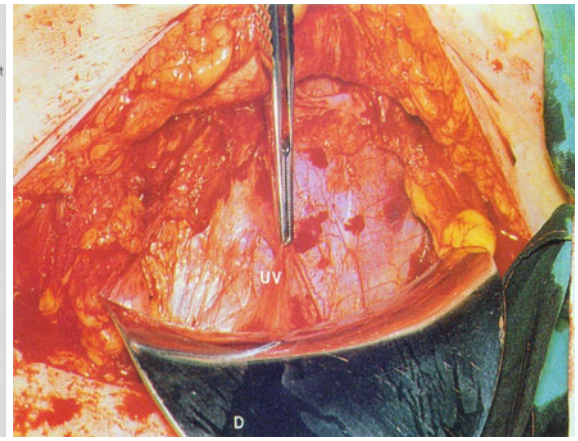
Patients with placenta previa should be managed by senior and experienced obstetricians. The hemorrhage at the time of cesarean section can be rapid, massive, and unrelenting. In most cases a total hysterectomy is required, as LS and the cervix itself may be involved. The pathological condition should be diagnosed before delivery, a classical cesarean section carried out, the infant delivered, the cord clamped, and a total obstetric hysterectomy performed. Counseling of transfusions and the major surgery of obstetric hysterectomy and its complications must be done.

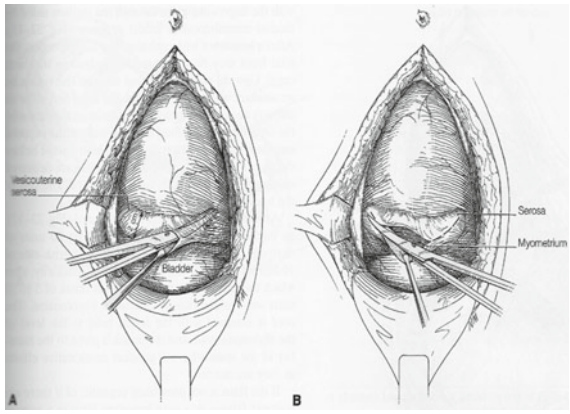
Provided that the placenta is not separated, no bleeding and further child bearing are paramount. The cesarean section is completed and the degeneration and sloughing of the placenta are awaited. This conservative treatment is fraught with the risk of potential sepsis and hemorrhage and this may result in re-surgery and hysterectomy. There are those who advocate methotrexate. The worst situation is placenta previa percreta, with developing invasion of the bladder wall and the broad ligament. Another potential preemptive maneuver in these cases is the preoperative placement of a vascular catheter in the internal iliac artery by the interventional radiologist to use for embolization immediately postdelivery.

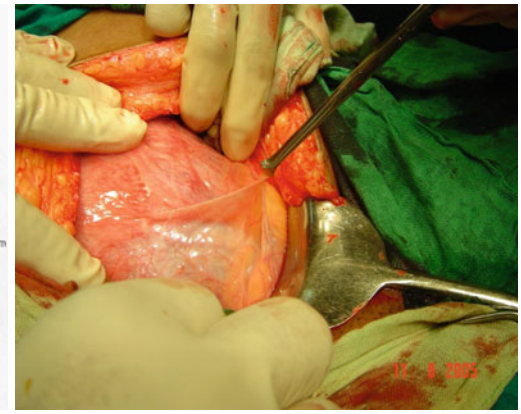
Lower Segment Caesarean Section (Munro Kerr)

The orientation of the lower segment- the loose uterovesical peritoneum is identified and adheres to the surface of the uterus, i.e., the upper margin of the lower segment. The uterovesical peritoneum is grasped using Allis forceps, elevated and incised, the lower flap is elevated and dissected lower to expose the lower segment. The fascia over the lower segment should not be dissected.

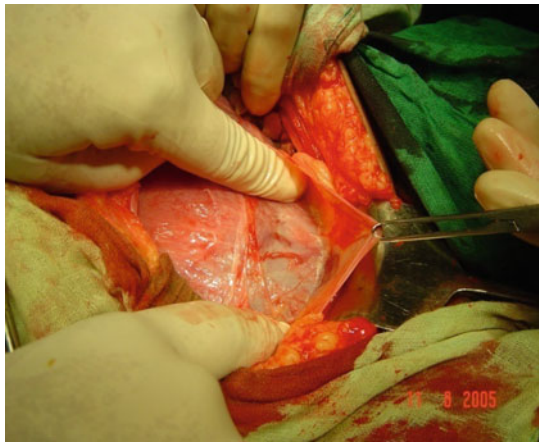


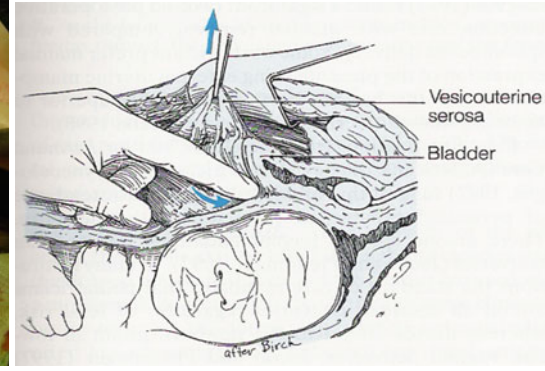






Peritoneum is grasped, elevated and incised





Dissection of peritoneum

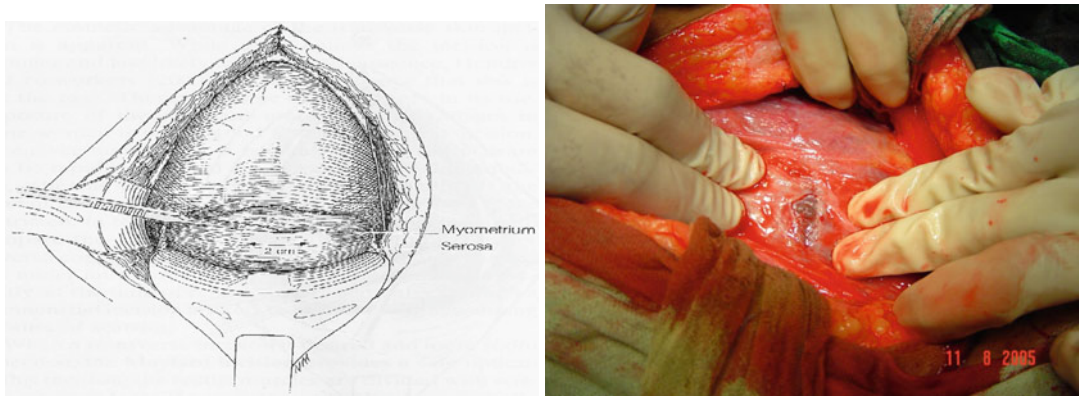
Opening of the Uterus

- Avoid undue haste and enthusiasm.
- Use a knife.
- Level of incision – the upper part of the lower segment –to the widest diameter practicable.

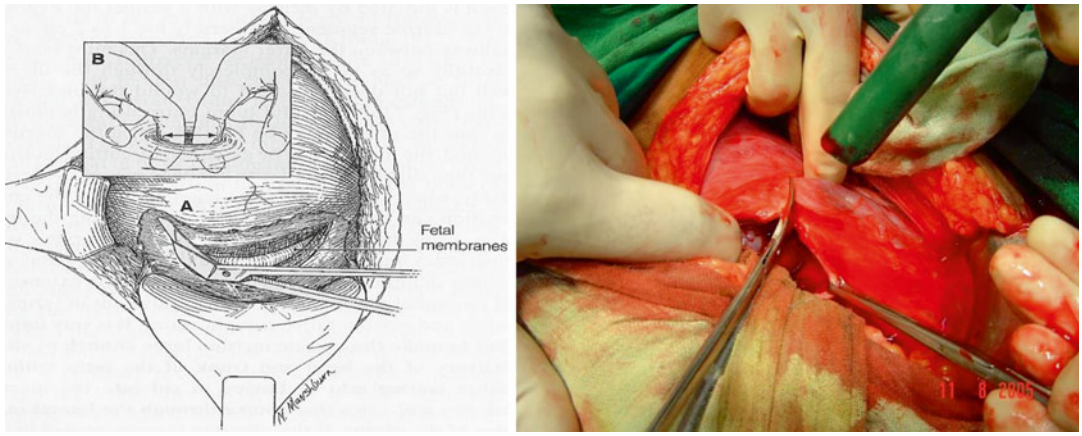
It is very important in the case of prolonged/obstructed labor and preterm LSCS. An incision should be made at the upper part of the lower segment – a low entry – with a risk of extension of the incision to the broad ligament and damage to the vessels with the risk of entry into the vagina (laparoelytrotomy).

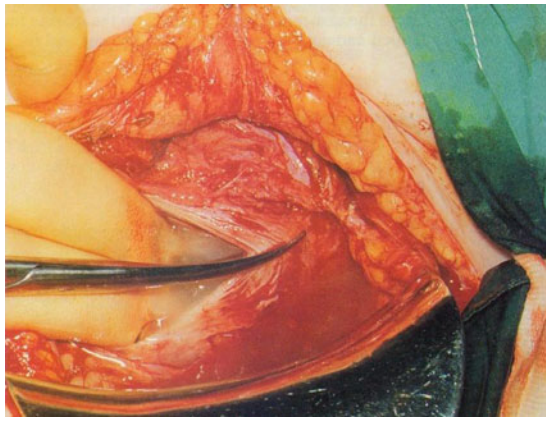
Using the knife, the LS should be exposed for 1–2 cm in the mid line transversely, taking care to avoid injuring the fetus.

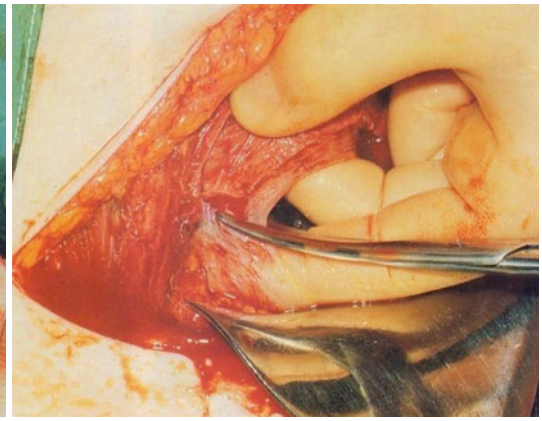
Extension of the lower segment incision on either side with a slight curve upward should be made using curved mayo or bandage scissors. In earlier gestation when the width of the lower segment is smaller, the angles of the uterine incision can be directed upward, which will produce an enlarged trap door effect. Using the fingers to pull the incision may extend downward owing to the arrangement of the lower segment muscle fibers and the lateral vessels may be damaged. An inverted "T" incision should never be made at the lower segment.

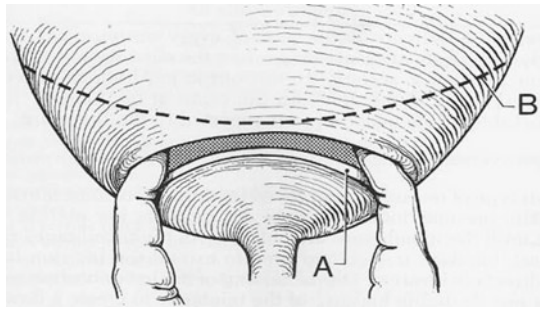


Finger compression at LS at the time of Incision





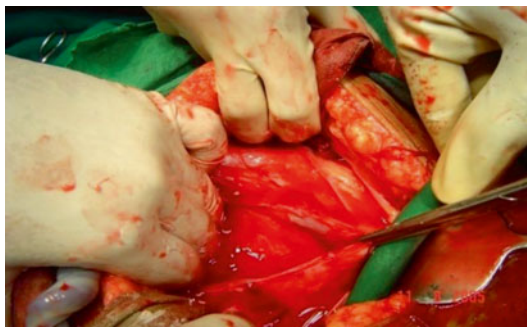




Extension of LS Incision

How to Catch the Edges of the Uterine Incision

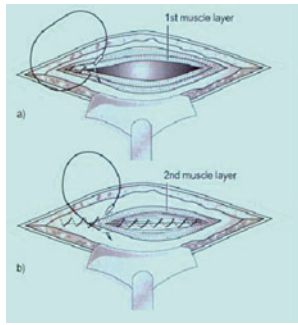
They should be caught before the placenta is delivered. Lift the upper segment with your left hand, which allows blood to go into the pelvis and the lower edge of the lower segment becomes visible to catch. If there is bleeding from the lateral angle, insert two fingers behind the broad ligament and lift the angles.

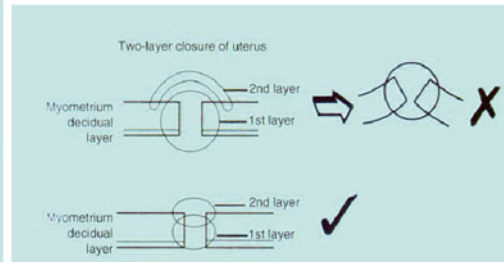


Closure of the Lower Uterine Segment, Traditionally in Two Layers

An increasing number of obstetricians have moved to a single-layer closure continuous suture, either running or locked, which saves about 5 min of operating time. There is as yet conflicting and inadequate data on the outcome of single- versus double-layer closure. In one large cohort study there was a fourfold increase in uterine rupture in subsequent pregnancy for single- versus double-layer closure. At least one large trial (CAESAR) is under way to investigate this.

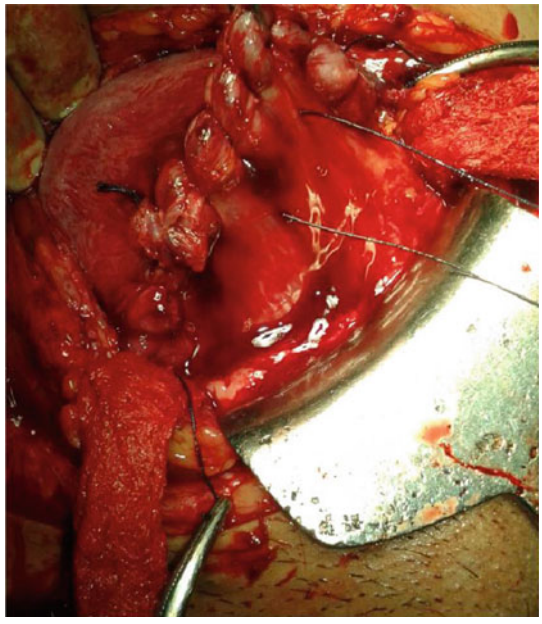
One observation – it is advisable to see the thickness and vascularity of the lower segment muscles before making any decisions. A very thin lower segment (because of previous LSCS) has often interrupted suturing with a thinner Vicryl 2–0 (Potter's technique). The first layer should include the cut edges of muscles but avoid the decidua if possible, usually with a running suture, which avoids the bunching and elevation of tissues seen with a continuous locking suture. It is also haemostatic.



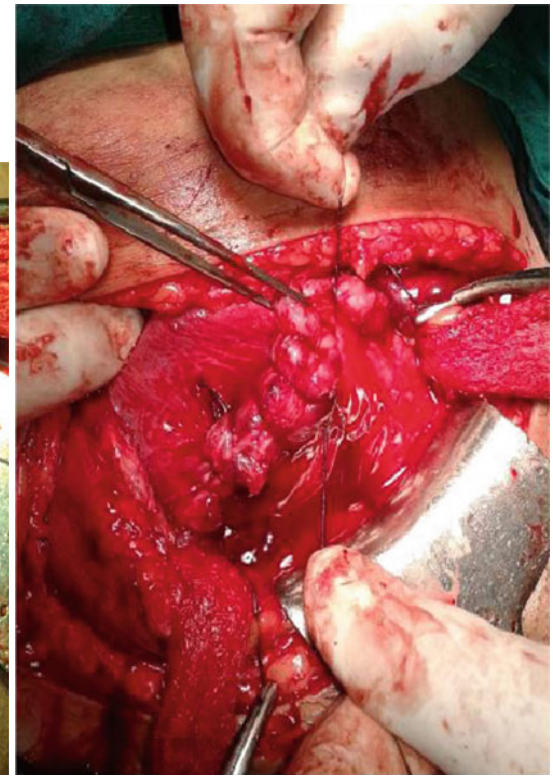


The second layer of sutures, either running or locking, approximates a fold of muscle to cover the first layer. The most commonly used sutures are Vicryl no. 0 or no. 1.

Another change is to omit closure of the visceral peritoneum associated with a marginally shorter operating time and less postoperative pain. Lower segment bleeding, in spite of adequate suturing, requires transverse (horizontal) mattress sutures.



transverse (Horizantal) mattress sutures



transverse (Horizantal) mattress sutures

Michael Stark

A single-layer closure with Vicryl – continuous nonlocking or locking – may require extra mattress sutures if bleeding occurs from a sutured lower segment [8]. It does not affect the integrity of the uterine scar (Chapman 1997) [9].

Great care should be taken with angle suturing. Ideally, the angles should be sutured separately. Modification of the Potter's method of closure consists of interrupted sutures with Vicryl 1–0 to the full thickness of myometrium.

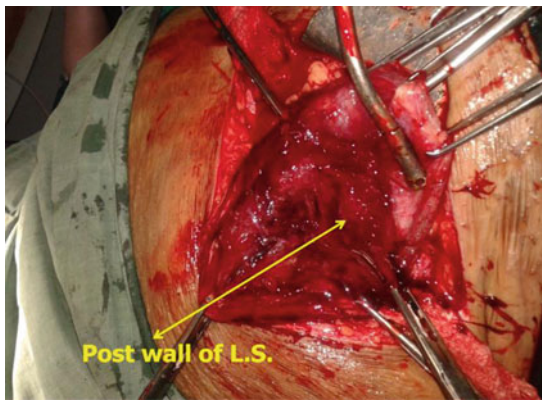
Difficulties While Suturing

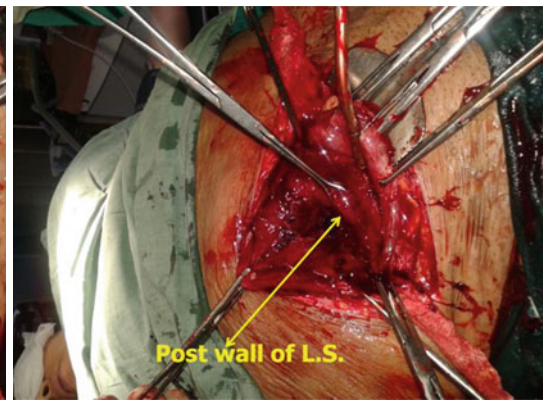
The lower edge of the lower segment is longer and thinner than the upper edge of the lower segment. Care should be taken with regard to the bladder, especially when there has been a previous LSCS. A serious error would be suturing of the upper border of the incision to a transverse ridge of the posterior wall.

A simplified variation of Munro Kerr's technique of LSCS described by Michael Stark at Misgav Ladach Hospital in Jerusalem is where the lower segment is identified. A transverse incision is put by knife on the free peritoneum 1-1.5 cm above the bladder fold. The lower segment is cut with knife in the middle 1-2". The edges of the incision are pulled apart by fingers to enlarge it to 10-12 cm. The baby delivery and the placental delivery are similar to conventional Munro Kerr method.

After delivery of the fetus and placenta the lower segment is closed with one layer using Vicryl no. 1 running locking sutures.

Experience with this technique suggests that it might result in a shorter operating time, less blood loss, and fewer postoperative analgesic requirements. The technique of Cohen's incision is used to open the abdomen. One should know when to use what and what precautions should to be taken.





Lower Segment Vertical Caesarean Section, Rarely Used, Can Be Used in Earlier Gestational Age When the Width of the Lower Segment Is Reduced

Certain Pathological Conditions to Be Understood at the Lower Segment

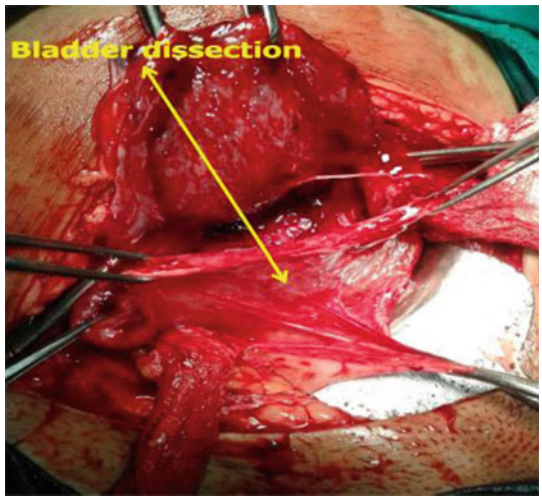
1. Lower segment in the case of previous LSCS (one, two, or more).
2. Adherent bladder at the lower segment in the case of previous LSCS.
3. Preterm pregnancy.
4. Lower segment fibroids and LSCS.
5. Lower segment adherent to the lower abdomen wall because of previous surgery.
6. What about the posterior uterine wall in the lower segment of the uterus? Particularly after placenta previa.
7. Incomplete rupture
   Benign type:
   (a) Dehiscence of a previous lower segment scar
   (b) Found incidentally at the time of repeat LSCS
   (c) After some time it may extend laterally and bleed into the broad ligament, with formation of a broad ligament hematoma.
8. Complete lower segment rupture:
   (a) Neglected obstructed labor
      Redouble efforts to overcome obstruction in multiparous patients (Bandl's ring).
   (b) Obstetric manipulation: its only acceptable role in modern obstetrics is in the delivery of the second twin.
      Neglected shoulder presentation – internal version and breech extraction.
   (c) Longitudinal lateral lower segment rupture can occur.
   (d) High forceps and rotation forceps delivery play an unacceptable role in modern obstetrics.
      If the lower segment, cervix, and vaginal vault (colporrhexis) are collectively ruptured, this is the worst emergency to handle.
   (e) Previous lower segment scar rupture at the trial of labor.
   (f) As a rule, vaginal examination should be carried performed for the palpation of the lower segment after vaginal birth in the case of previous LSCS to rule out lower segment rupture.
9. Lower segment subepithelial (decidua) trauma and lower segment PPH.
   – Induced labor.
   – Use of mesoprost.
   – Precipitated labor.
10. Abnormally vascular lower segment
11. Lower segment tear during LSC:.
   (a) Particularly in the case of prolonged, obstructed labor.
   (b) Previous LSCS: some tears are so bad that hepatic artery ligation (HAL) is needed to reduce bleeding before suturing.

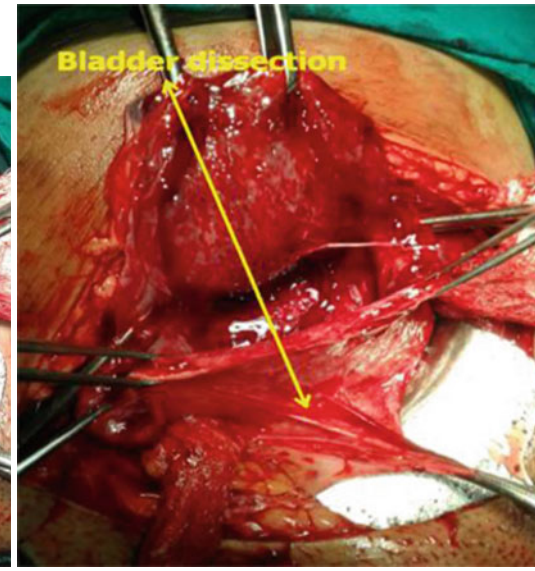
Lower segment cesarean section and injury to the bladder/ureter

Risk factors – obstetric, surgical, anatomical.

Bladder injury usually occurs at the dome of the bladder.

- Bladder dissection in the case of previous LSCS
- In the case of prolonged/obstructed labor
- Lower segment tear/rupture
- Abnormal placentation in the case of previous LSCS
- Injury during suturing the lower segment
- Obstetric hysterectomy.
- Lower segment vesical fistula – abdominal route of repair with 100 % result.





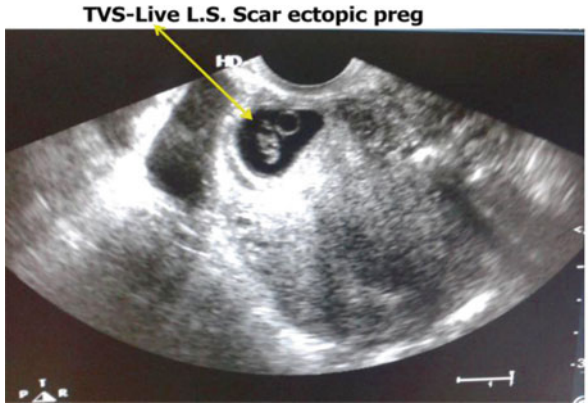
Ureteric Injury

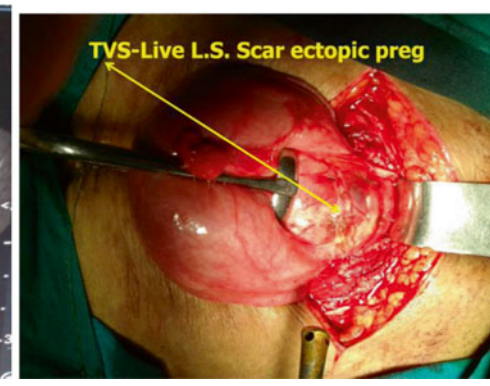
Extension of a lower segment tear laterally into the broad ligament or down to the cervix, when suturing or controlling hemorrhage.

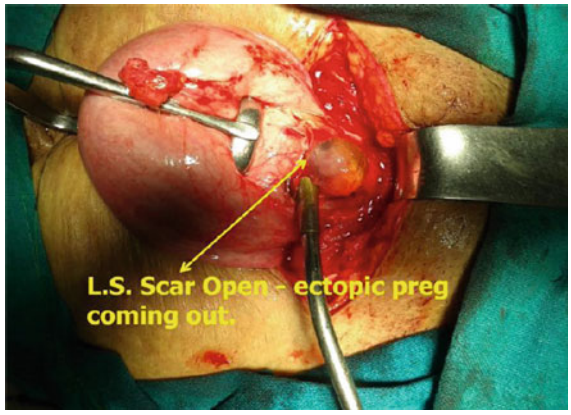
Secondary Hemorrhage from LSCS

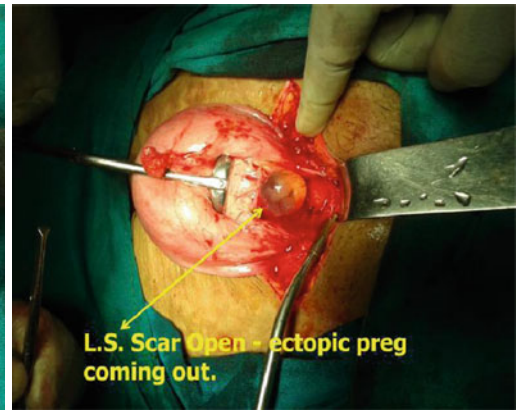
This is believed to be due to local infection at the site of lower segment closure, causing erosion of the blood vessels or arteriovenous anastomosis. It may lead to recurrent bleeding, sometime massive, 2–3 weeks after LSCS and may need subtotal obstetric hysterectomy at some time.

1. Lower segment in unicornuate pregnant uterus
2. Constriction ring dystocia.
3. Ectopic pregnancy in previous LSCS scar - rare – systemic MTX, 100% success with β HCG less than 5000. If there is bleeding needs exploration.
4. Mid-trimester lower segment rupture or hematoma with termination of pregnancy with the use of misoprost.
5. Lower segment abscess and dehiscence due to infection.
6. Suturing and controlling hemorrhage at the lower segment after subtotal obstetric hysterectomy for atonic P.P.H.






L.S. Scar Open - ectopic preg
coming out.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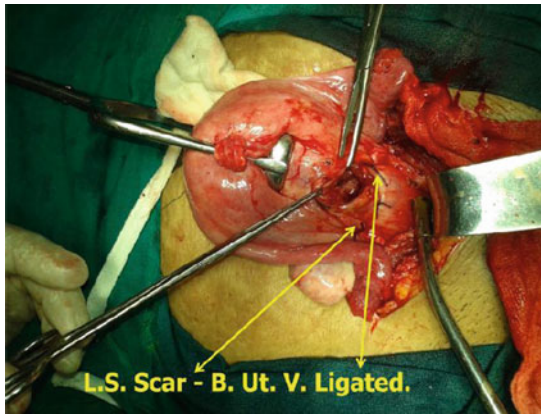
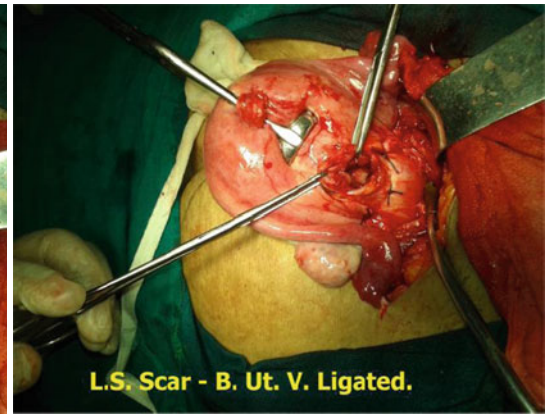
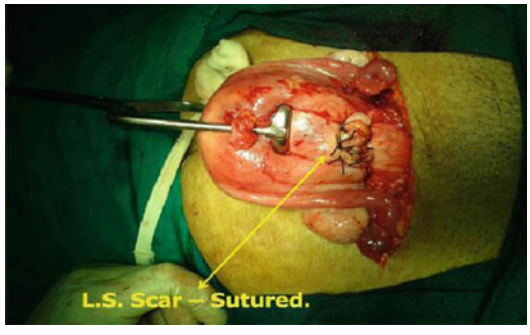
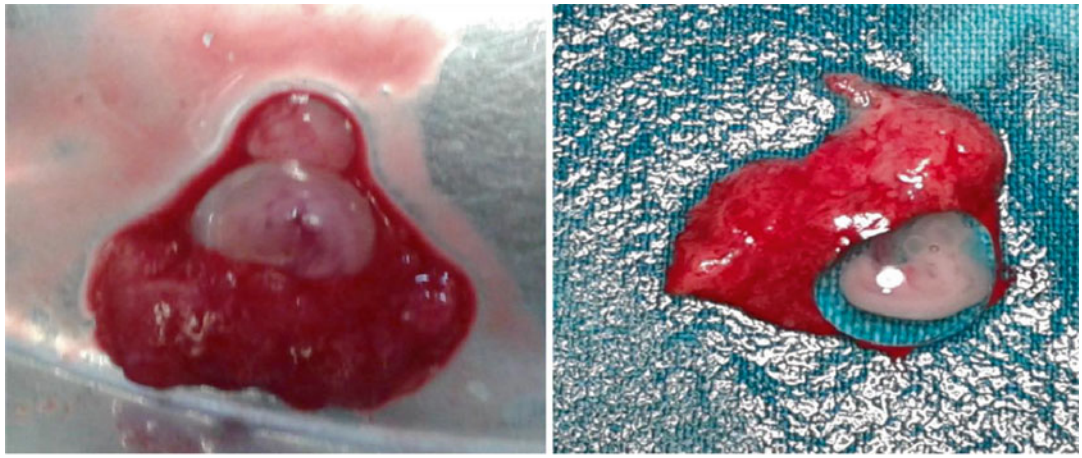

L.S. Scar - B. Ut. V. Ligated.


L.S. Scar - B. Ut. V. Ligated.


L.S. Scar – Sutured.



Healing of the Lower Segment Incision (Williams 1921)

The lower uterine segment heals by regeneration of the muscular fibers and not by the development of scar tissues unless not adequately sutured.

Observation at repeat LSCS with an unopened lower segment, there is certainly no trace of the former incision or at the most an invisible linear scar (Mcintyre 1924, Wilson 1951) [10, 11]. The uterus is removed and fixed in formalin. There is no visible scar, only a shallow furrow.

Postgraduate Obstetrics/Gynecology (J.C. Mcclure Browne)

For lower segment healing wound sepsis or incomplete hemostasis is unfavorable and scar formation between the apposed wound surfaces is the rule. According to Wilson (1951) regeneration of plain muscles occurs [11].

Schwarz and Coworkers (1938) Concluded

Healing occurs mainly by fibroblast proliferation, but the scar shrinks and connective tissue proliferation becomes less obvious; this all depends on how the uterine muscles are opposed [12].

Good apposition without the decidua in between, without hematoma and infection, the proliferation of connective tissue is minimal and the normal relation of smooth muscles to connective tissue is gradually reestablished.

LSCS for Placenta Previa

Careful assessment of ultrasound images helps to delineate the lower segment covered by the placenta and guides the site of uterine incision and direction of the hand so that the shortest route to the placental edge is achieved before rupture of the membrane and delivery of the fetus. At least two PCV (Packed Cell Volume) must be in the O.R. (Operating Room). Perfect counseling of the seriousness regarding the condition and even the need for obstetric hysterectomy should be done. In most cases, the lower segment is sufficiently developed to allow standard LSCS. Rarely, huge vessels over the lower segment are worth ligating. It is best not to cut through the placenta but to use the hand to separate the placenta to the nearest edge, rupture the membrane, deliver the baby, and promptly clamp the cord. This problem may not arise in after placenta previa.

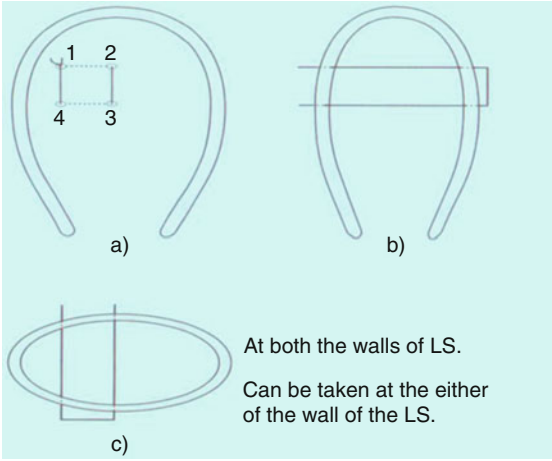
Now there is the problem of lower segment PPH as it is not as contractile as the upper segment. Lower segment edges can be caught with Allis's forceps and the "entire bowl of the lower segment" should be tightly packed and pressure applied for 4 min. When the packing is removed, discrete points of bleeding can be ligated. Uterine artery ligation is carried out with the lower segment open (ACR) [13, 14].

Full-thickness compression sutures (CHO) making sure to leave a portal in the middle to allow the efflux of lochia. Adequate suturing of the lower segment should also be ensured. Check vaginally for bleeding before closing the abdomen.

As a last resort total obstetric hysterectomy should be carried out using HAL or embolization. A balloon device can be inserted and run down through the cervix inflated and if it works the lower segment should be sutured over the balloon.

Uterine Artery Ligation at Lower Segment

Bilateral ascending uterine artery ligation has been reported to be a safe and easy method of controlling bleeding during LSCS in women with the presence or increased risk of preoperative or postoperative blood loss. In 30 years of experience, only 10 failures in 265 patients have been reported.



Postage Stamp Sutures (CHO)

With the use of straight needle

Concept of Therapeutic and Prophylactic Ligation of Uterine Vessels

Before the lower segment is sutured.

PPH

If the amount of blood loss is more than expected by the obstetrician during the procedure it may be atonic or traumatic. Average blood loss at cesarean section is 700 ml (Pritchard). Most of it is from the placental site. However, anticipation is the most effective strategy.

Factors Associated with Blood Loss of More than 1–1.5 l at Cesarean Section

Vascular and bleeding from the lower segment in 20 %. Damage to vessels at an angle – 60–70 %. Lower segment tear in prolonged/obstructed labor and previous cesarean section. Placenta previa, accidental hemorrhage (APH).

Induced/augmented labor. Preeclampsia, elderly primipara, multiparity. Preterm LSCS, History previous PPH. Amnionitis. Broad ligament hematoma. Over-distended uterus – twin pregnancy, post-term pregnancy, large baby.

Uterine Artery Ligation

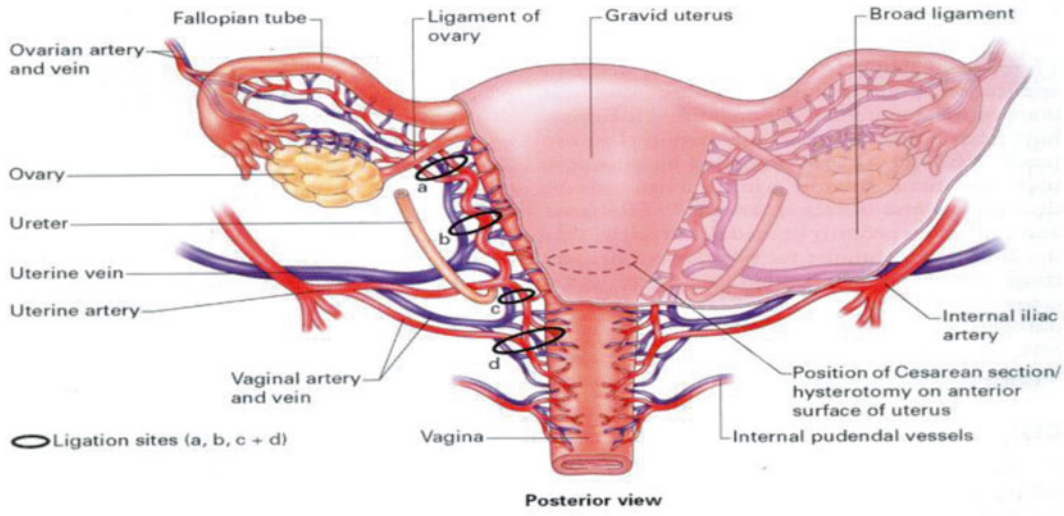
One of the most effective approaches to a more difficult situation. The technique of bilateral ligation of uterine vessels has been described by O'Leary and O'Leary. Water presented the first data on uterine artery ligation for controlling PPH in 1952 [16].

This is a logical step because 90 % of the uterine blood flow is derived from the uterine arteries, with the remainder coming from the ovarian, cervical, and vaginal arteries.

The uterine vessels are ligated by using Vicryl no.1 on an atraumatic needle passing through the lower segment wall to the uterine vessels from the anterior to the posterior wall, a hand should be placed behind the uterus to prevent injury to the sigmoid colon on the left side and the small bowel on the right side. The needle is guided and brought forward through the vascular area in the broad ligament lateral to the uterine vessels and the suture is tied to control the bleeding. The suture is placed below the site of the uterine incision (the lower part of the lower segment). The same thing is done at the upper part of the lower segment to control the bleeding from anastomosis of the ovarian vessels.

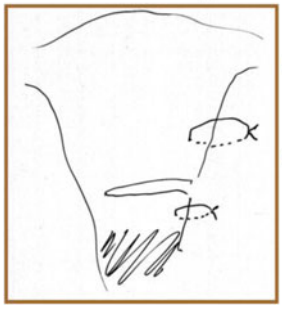
This technique is performed when the lower segment is not sutured or sutured and there is bleeding from the angle of the lower segment. Care should be taken with regard to the bowel behind and the bundle of vessels should not be punctured. Unilateral ligation is enough to control the bleeding as lower segment trauma is only on one side. Bladder mobilization is not necessary and the ureter is not at risk.

The vessels are not divided and recanalization occur. The resumption of normal menses and normal pregnancy after ligation has been documented.

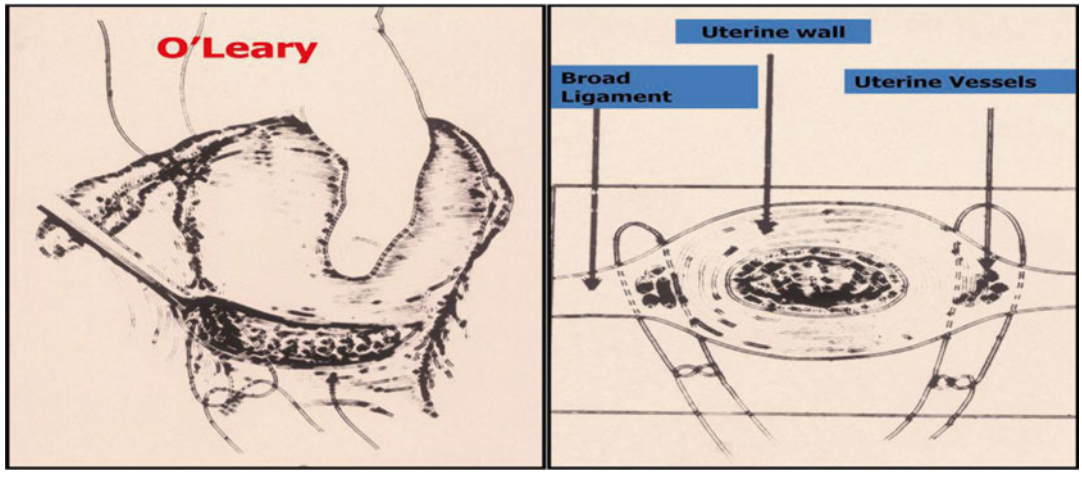


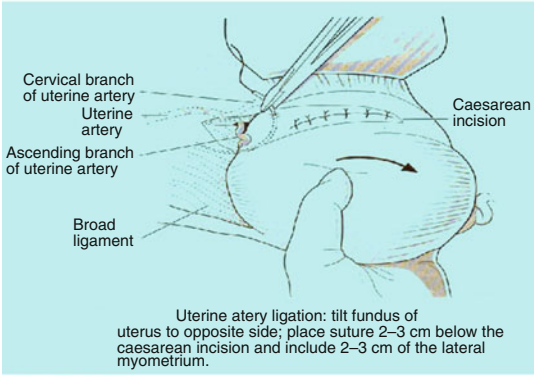
Placement of ligatures in the process of stepwise devascularization, including ligature of the descending uterine and vaginal arteries

Uterine Art. Ligation O'Leary 1995

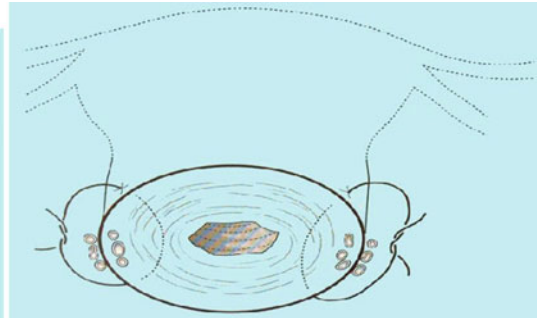


- Take a firm bite through lat. Ut. Wall
- Bring back the needle through avascular area of broad ligament

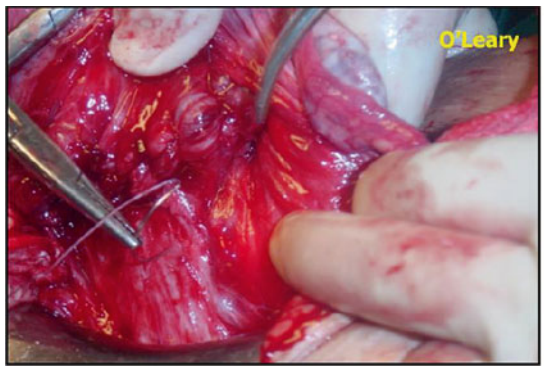


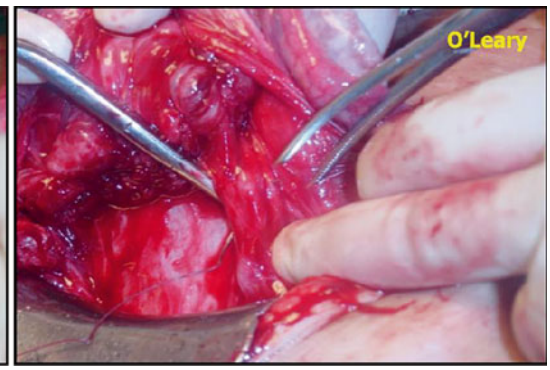


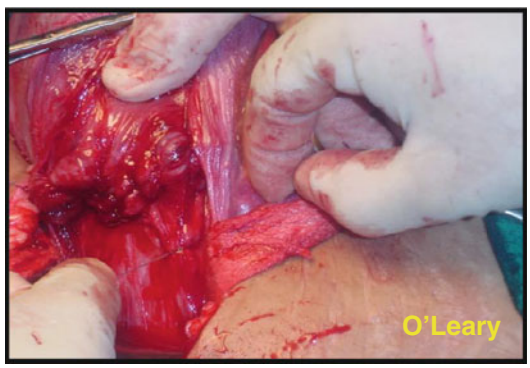
Uterine atery ligation: tilt fundus of uterus to opposite side; place suture 2–3 cm below the caesarean incision and include 2–3 cm of the lateral myometrium.



Uterine artery ligation: coronal view of lower uterine segment and placement of suture to include adjacent myometrium and all branches of the uterine vessels.

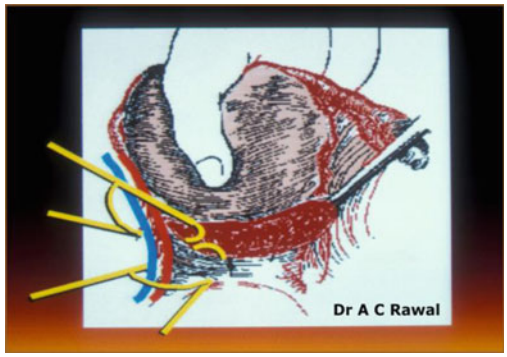


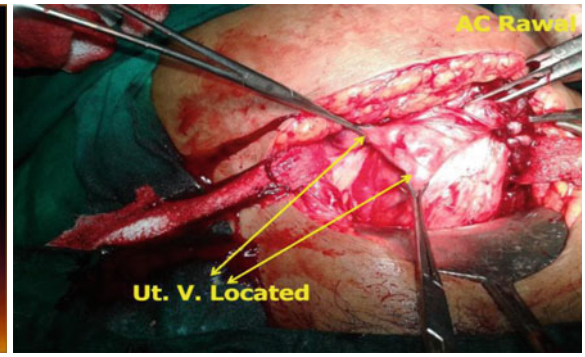


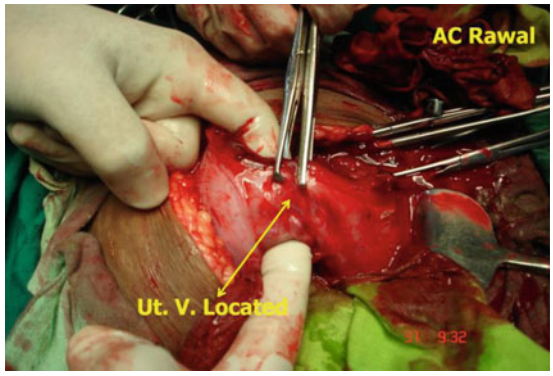


A modification of this technique (uterine artery ligation) is worth mentioning and is performed by the author (ACR techniques). It can be done at trauma (laceration) or if there are tears of the lower segment or damage to the lateral angle vessels. It is an open technique where the lower segment is not sutured in LSCS.

An Allis forceps applied to angle of the open lower segment and pulled medially and to form a 'U' shape with the respective uterine vessels. Then a Vicryl No. 1 suture is passed from the lateral aspect of the uterine vessels into the uterine cavity, i.e.,from the outside in, and then the needle is passed inside out at lower edge of the lower segment coming out medially to the vessels and tied to control the bleeding from uterine vessels. The same thing can be done to the upper part of the lower segment incision to stop the bleeding from the anastomosis. There is no need to place a hand behind the uterus, as there is no chance of bowel injury, as with the previous technique.


Dr A C Rawal


AC Rawal
Ut. V. Located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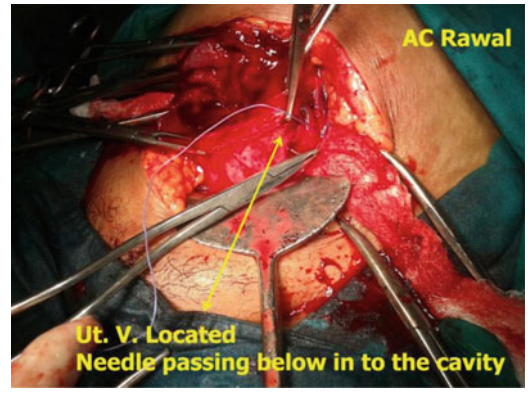
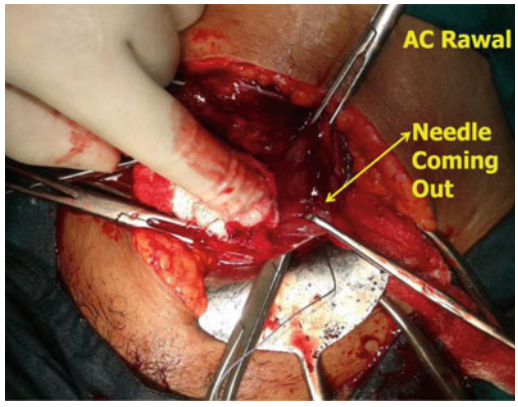
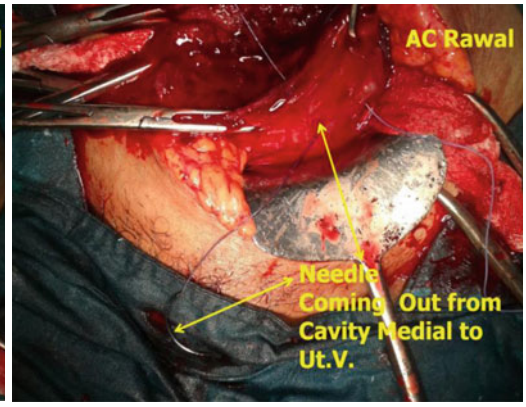
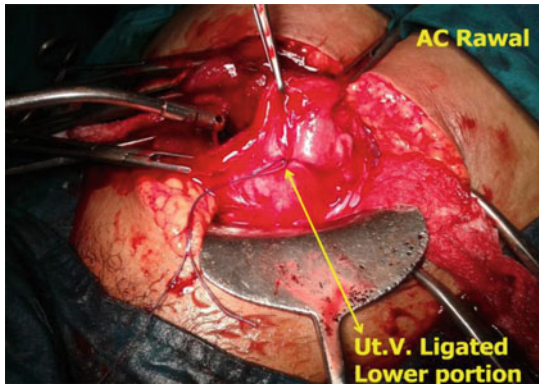
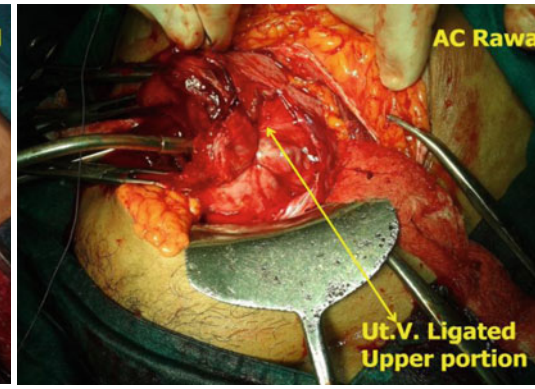
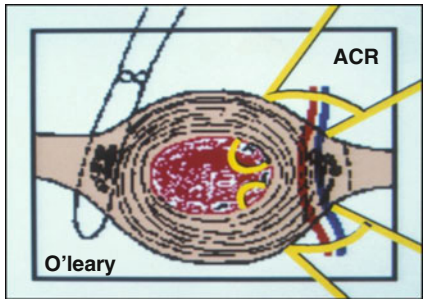

AC Rawal
Ut. V. Located
Needle passing below in to the cavity


AC Rawal
Needle
Coming
Out


AC Rawal
Needle
Coming Out from
Cavity Medial to
Ut.V.







Difference Between the two technics.

Instead of struggling with the lower segment angle bleeding because of laceration or tears, select a specific uterine artery ligation to reduce the bleeding and suturing of tear and lower segment can be carried out.

Bilateral uterine artery ligation is a very good procedure to control the bleeding in cases of placenta previa.

Bilateral ascending uterine artery ligation has been reported to be a safe and easy method of controlling bleeding during cesarean section in women with the presence or increased risk of perioperative blood loss and traumatic injury of the lower segment [16].

In the largest retrospective series covering 30 years of experience only 10 failures in 265 patients were reported. Sometimes when the laceration or tearing of the lower segment is so bad and involves the vault of the vagina, particularly in the case of obstructed labor or when uterine artery ligation has failed, bleeding is diffuse, and hematoma formation has obscured the source of bleeding, another approach is needed. Bleeding is temporarily controlled by applying pressure to the suspected site of vascular injury and hypogastric artery ligation should be performed, which reduces the pulse pressure. The trauma site at the parametrial structures and at the vault of the vagina can be seen there by facilitating suturing, avoiding injury to the ureter and bladder.

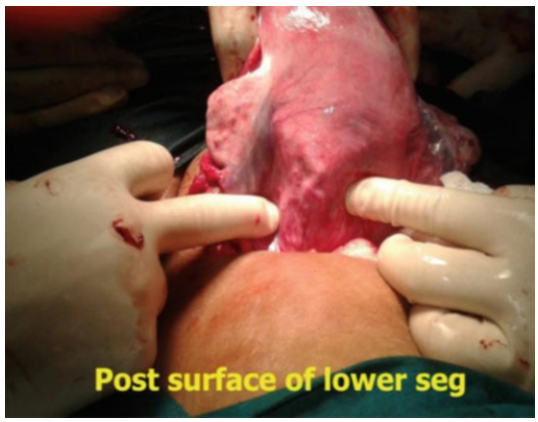
The use of the techniques described above controls bleeding in most situations and very rarely does the surgeon resort to obstetric hysterectomy.

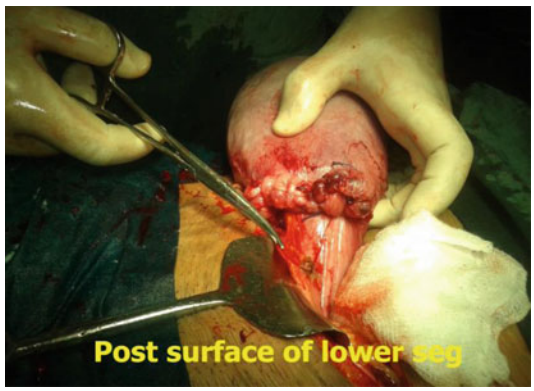
Maulick's Maneuver

At the lower segment of the uterus, swab holders are applied on both sides at the level of the lower segment as far down as possible, with a waiting period of 15–30 min.

Posterior Surface of the Posterior Wall of the Lower Segment

Adhesion separation occurs during delivery of the uterus for suturing of the lower segment or for examination leads to hemorrhage because of the separation of the endometriotic or inflammatory adhesions. Rarely, there is a hematoma at the posterior surface.





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Ruptured Ectopic Pregnancy

19

Abdul Vahab and P. Mumtaz

There is a sixfold increase in the incidence of ectopic pregnancy over the last decade due to increasing popularity of ART procedures and tubal recanalization surgeries and increasing incidence of pelvic inflammatory diseases [1]. The most common clinical presentation of ectopic pregnancy is first-trimester vaginal bleeding and/or abdominal pain [2].

Tubal rupture is one of the inevitable courses of a pregnancy implanted in the tube. It causes considerable intraperitoneal hemorrhage, leading to shock.

Ruptured ectopic pregnancy is the major cause of pregnancy-related maternal mortality in the first trimester [3]. Most of these deaths occur prior to hospitalization or proximate to the woman's arrival in the emergency department.

In the developing countries it is estimated that 10 % of women admitted with a diagnosis of ectopic pregnancy ultimately die from the condition [4].

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Pathophysiology of Hemorrhagic Shock

Acute hemorrhage causes a decreased cardiac output and decreased pulse pressure. These changes are sensed by baroreceptors in the aortic arch and atrium. With a decrease in the circulating volume, neural reflexes cause an increased sympathetic outflow to the heart and other organs. The response is an increase in heart rate, vasoconstriction, and redistribution of blood flow away from certain nonvital organs, such as the skin, gastrointestinal tract, and kidneys.

The brain has remarkable autoregulation that keeps cerebral blood flow constant over a wide range of systemic mean arterial blood pressures. The kidneys can tolerate a 90 % decrease in total blood flow for short periods of time.

The hallmark clinical indicators of shock have generally been the presence of abnormal vital signs, such as hypotension, tachycardia, decreased urine output, and altered mental status. These findings represent secondary effects of circulatory failure, not the primary etiologic event.

The general appearance of a patient in shock can be very dramatic. The skin may have a pale, ashen color, usually with diaphoresis. The patient may appear confused or agitated and may become obtunded. The pulse first becomes rapid and then becomes dampened as the pulse pressure diminishes. Systolic blood pressure may be in the

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normal range during compensated shock. The conjunctivae are inspected for paleness.

The abdominal examination searches for signs of intra-abdominal bleeding, such as distention, pain with palpation, and dullness to percussion. Patients with a history of vaginal bleeding undergo a full pelvic examination. In all women of reproductive age group, a pregnancy test has to be done to rule out ectopic pregnancy.

Clinical Presentation in Ruptured Ectopic Pregnancy

In a population-based registry of ectopic pregnancy from France, the incidence of rupture was 18 % [5].

If the ectopic pregnancy is situated in the isthmus, rupture occurs around 6–8 weeks; if in ampulla it occurs later and interstitial pregnancy much later toward the end of the first trimester. Massive bleeding can cause hypovolemic shock and even death. A self-limiting bleeding can later on lead to formation of chronic ectopic pregnancy.

The clinical presentation largely depends on the amount of internal hemorrhage.

Patients with small hemorrhage not amounting to shock will present with lower abdominal pain with or without bleeding per vagina and increasing tiredness gradually progressing to shock. Not diagnosing the condition in the early phase leads to loss of valuable time and pushes the patient to inevitable hypovolemic shock.

The classical presentation is that of the acute abdomen. Usually there is a sharp pain from the pelvis followed by syncopal attack. The patient may be brought in with shock. Tubal rupture can result in life-threatening hemorrhage.

Symptoms include severe or persistent abdominal pain or symptoms suggestive of ongoing blood loss (e.g., feeling faint or loss of consciousness). Sometimes unexplained fainting, shoulder tip pain, and unusual bowel symptoms are the features.

There may or may not be history of amenorrhea and vaginal bleeding.

Abdominal pain—There is no pain pattern that is pathognomonic for ectopic pregnancy. The pain associated with ectopic pregnancy is usually located in the pelvis. It may be diffused or localized to one side. As the blood gets collected in the peritoneal cavity, pain may be felt in the middle or upper abdomen. Massive intra-abdominal bleeding can cause diaphragmatic irritation, and there may be referred pain that is felt in the shoulder. Patient may complain about constant urge to defecate which is not relieved even after defecation. This points toward pooling of blood in the pouch of Douglas.

The timing, character, and severity of abdominal pain vary, and the onset of the pain may be abrupt or slow, and the pain may be continuous or intermittent. The pain may be dull or sharp; it is generally not crampy. Tubal rupture may be associated with an abrupt onset of severe pain, but rupture may also present with mild or intermittent pain.

Unfortunately, atypical presentation is also relatively common in cases of tubal rupture with hemodynamic compensation. The condition may be mistaken for other gynecological disorders and gastrointestinal or urinary tract disease, including appendicitis, acute PID, ruptured corpus luteum or follicular cysts, threatened or inevitable spontaneous abortion, ovarian torsion, and urinary tract infection.

The 1997–1999 and 2003–2005 Confidential Enquiries into Maternal Deaths reports pointed out that most of the women who died from ectopic pregnancy were misdiagnosed in the primary care or in casualty [6, 7]. It was therefore recommended that all clinicians should be made aware of the atypical clinical presentations of ectopic pregnancy. In the 2006–2008 Centre for Maternal and Child Enquiries (CMACE) report, four of the six women who died from early ectopic pregnancy complained of diarrhea, dizziness, or vomiting as early symptoms without triggering any suspicion of ectopic pregnancy [8].

A slow trickle from the site may proceed to an ongoing bleeding which may last for a few days before getting adequate medical attention and hence can even cost one's life.

In any woman of reproductive age who presents with abdominal pain, the possibility of ectopic pregnancy should be kept in mind and should be ruled out.

Clinical Examination

In an emergency, where the patient has collapsed and there is high clinical suspicion of tubal rupture, extensive clinical examination is inappropriate and immediate surgical intervention is indicated.

In a patient with atypical presentation or mild symptoms, detailed clinical examination is warranted.

Physical examination—Initial assessment of general condition including severity of discomfort, orientation in time and space, and level of hydration should be checked. Vital signs should be measured and hemodynamic stability assessed. Postural hypotension may be the only and initial sign of young healthy patients with blood loss. Vital signs, including postural changes, may be normal early in the course of significant bleeding due to compensatory mechanisms [9]. As ectopic pregnancy affects young women, so remarkable hemodynamic compensation is often seen. Tachycardia could be the initial sign followed by hypotension and both worsening as the patient goes in for decompensation.

The next step is abdominal examination; it may reveal lower abdominal tenderness. If rupture with significant bleeding has occurred, the abdomen may be distended, and diffused or localized tenderness to palpation and/or rebound tenderness may be found on examination. There can be shifting dullness due to hemoperitoneum.

It is important to do a meticulous pelvic examination. It has two components, speculum examination and bimanual examination. The speculum examination is used to rule out cervical or vaginal lesion and to assess the volume of bleeding by noting the quantity of blood in the vagina and presence or absence of active bleeding from the cervix.

The bimanual pelvic examination is often unremarkable in a woman with a small, unruptured ectopic pregnancy. But in a patient with hemoperitoneum, there can be signs like cervical motion pain, mass, and/or tenderness in the adnexa.

The uterus may be somewhat enlarged but will likely be smaller than appropriate for gestational age. Uterine enlargement in women with ectopic pregnancy may be due to endocrine changes of pregnancy, rare cases of heterotropic pregnancy, or incidental uterine pathology (most commonly, uterine fibroids).

Palpation of the adnexa should be performed with only a small degree of pressure, since excessive pressure may rupture an ectopic pregnancy. Findings on examination may include cervical motion and adnexal and/or abdominal tenderness. An adnexal mass is noted in some women. Great caution is required during bimanual examination as this may exacerbate bleeding.

Investigations

Ultrasound plays a key role in the diagnosis of ectopic pregnancy along with β-hCG estimation. Key point is exclusion of intrauterine pregnancy be it viable or nonviable.

Diagnosis can be straightforward when a transvaginal ultrasound scan (TVS) positively identifies an IUP or ectopic pregnancy [10]. If TVS fails to identify the location of a pregnancy, such women are currently diagnosed as having a "pregnancy of unknown location" (PUL) [11, 12].

The 2006–2008 CMACE report drew attention to a maternal death secondary to ruptured ectopic pregnancy where a diagnosis of PUL had been made. Although most patients with a PUL will subsequently be diagnosed with either a failed IUP (a spontaneous abortion) or viable IUP, the report highlights that 7–20 % will be diagnosed with an ectopic pregnancy. It is therefore very important that a diagnosis of PUL should trigger further diagnostic tests and follow-up until the final outcome of the pregnancy is known.

Transvaginal Ultrasonography [TVUS]

Transvaginal ultrasound (TVUS) is the most useful tool for determining the location of a pregnancy. High-definition ultrasonography, particularly using

the transvaginal route, has revolutionized the assessment of patients with early pregnancy problems, allowing visualization of both normal and abnormal gestations [13]. TVUS should be performed as part of the initial evaluation and may need to be repeated, depending upon the hCG level or a suspicion of rupture.

TVUS alone (without measurement of hCG) can exclude or diagnose an ectopic pregnancy *only* if one of the following findings is present:

- Findings diagnostic of an intrauterine pregnancy (IUP, gestational sac with a yolk sac or embryo)
- Findings diagnostic of a pregnancy at an ectopic site (gestational sac with a yolk sac or embryo)

An adnexal mass is the most common ultrasound finding in ectopic pregnancy and is present in 89 % or more of cases [14–16].

In a healthy IUP, a TVS should identify the intrauterine gestational sac with almost 100 % accuracy at a gestational age of 5.5 weeks [17]. Ultrasound visualization of a yolk sac or embryo in addition to a gestational sac is needed for the confirmation of intrauterine pregnancy [17]. This is because an ectopic pregnancy can be accompanied by a "pseudosac," a collection of fluid within the endometrial cavity that may be the result of localized breakdown of the decidualized endometrium. It can be differentiated from gestational sac by its central location and lack of hyperechoic decidual reaction around them [18]. In addition, pseudosacs are transient, rather than consistent.

Suspicion of an ectopic pregnancy increases if free fluid (representing blood) is visualized, either surrounding the uterus or in the pouch of Douglas [19]. A small amount of free fluid in the pouch of Douglas is common in early pregnancy, due to increased vascular permeability.

The ultrasound examination is also used to evaluate whether rupture of the tube or other structures has occurred. A finding of echogenic fluid (consistent with blood) in the pelvic cul-de-sac and/or abdomen is consistent with rupture. A small amount of fluid is present in many women, and a small amount of blood may be present in other conditions like spontaneous abortion, ruptured ovarian cyst, tubal abortion, and initial stage of tubal rupture.

It is important to assess the amount of hemoperitoneum; it may be a small or large amount of blood.

The identification of an IUP can rule out ectopic pregnancy in most settings unless a heterotropic pregnancy is suspected, where an ectopic pregnancy coexists with an IUP [20]. They are rare (1 in 40,000), although more common after assisted conception, and difficult to diagnose.

The positive identification of a non-cystic adnexal mass with an empty uterus has a sensitivity of 84–90 % and a specificity of 94–99 % for the diagnosis of an ectopic gestation [21].

False positives can, however, occur if other structures such as the corpus luteum, the bowel, a paratubal cyst, a hydrosalpinx, or an endometrioma are mistaken for an ectopic pregnancy.

False negatives can occur if the ectopic pregnancy is small or if it is concealed by bowel or uterine anomalies such as fibroids. It is therefore possible for an ectopic pregnancy to go unnoticed on an ultrasound scan, especially if the patient is asymptomatic.

Either ultrasound or other abdominal imaging modalities are used for evaluation in the rare cases of abdominal pregnancy [22]. Magnetic resonance imaging is useful if ultrasound is not able to elucidate the location of a pregnancy. Examples of this include differentiating an intrauterine pregnancy (IUP) from a cervical or interstitial pregnancy or elucidating the anatomic relationships of an abdominal pregnancy.

Computed tomography generally has no role in the evaluation of pregnancy of unknown location, due both to its limited resolution of tissue planes and use of radiation.

Management of Ruptured Ectopic Pregnancy

This has two components: general supportive measures to combat effects of blood loss and surgical measures to arrest bleeding.

General Measures

This includes laboratory studies and medical care including fluid and blood replacement strategies.

Laboratory Studies

(a) Hemoglobin and hematocrit values remain unchanged from baseline immediately after acute blood loss. During the course of resuscitation, the hematocrit may fall secondary to crystalloid infusion and re-equilibration of extracellular fluid into the intravascular space.

No absolute hematocrit or hemoglobin level threshold that should prompt transfusion exists. A hemoglobin concentration of less than 7 g/dL in the acute setting in a patient that was otherwise healthy is concerning only because the value most likely will drop considerably after re-equilibration.

(b) If the patient is in severe shock, arterial blood gas may be the most important laboratory value.

Acidosis is the best indicator in early shock of ongoing oxygen imbalance at the tissue level. A blood gas with a pH of 7.30–7.35 is abnormal but tolerable in the acute setting.

The mild acidosis helps unload oxygen at the peripheral tissues and does not interfere with hemodynamics.

A pH below 7.25 may begin to interfere with catecholamine action and cause hypotension unresponsive to inotropics. Although this is a time-honored concept, recent data do not find evidence of this phenomenon.

Metabolic acidosis is a sign of underlying lack of adequate oxygen delivery or consumption and should be treated with more aggressive resuscitation, not exogenous bicarbonate. Life-threatening acidemia (pH < 7.2) initially may be buffered by the administration of sodium bicarbonate to improve the pH. However, be aware that no survival benefit to this practice has been documented.

(c) Coagulation studies generally produce normal results in the majority of patients with severe hemorrhage early in the course.

The prothrombin time (PT) and the activated partial thromboplastin time (aPTT) will identify major problems with secondary hemostasis.

The best test for platelet function is the bleeding time. This test is difficult to perform in the patient with acute hemorrhage.

Medical Care

(a) *Fluid management*

The primary treatment of hemorrhagic shock is to control the source of bleeding as soon as possible and to replace fluid.

As soon as the patient is received, two large-bore cannulas should be inserted and blood should be taken for investigations, grouping, and cross matching, and fluid replacement therapy should be initiated.

Crystalloid is the first fluid of choice for resuscitation. Administer 2 L of isotonic sodium chloride solution or lactated Ringer's solution in response to shock from blood loss. Fluid administration should continue until the patient's hemodynamics become stabilized. Because crystalloids quickly leak from the vascular space, each liter of fluid expands the blood volume by 20–30 %; therefore, 3 L of fluid needs to be administered to raise the intravascular volume by 1 L.

Alternatively, colloids restore volume in a 1:1 ratio. Currently available colloids include human albumin, hydroxyethyl starch products (mixed in either 0.9 % isotonic sodium chloride solution or lactated Ringer's solution), or hypertonic saline-dextran combinations. The product that is usually avoided in large-volume (>1,500 mL/d) restoration is the hydroxyethyl starch product mixed in 0.9 % isotonic sodium chloride solution because it has been associated with the induction of coagulopathy. The other products have not been so implicated.

(b) *Blood replacement*

Packed red blood cells should be transfused at the earliest. It doesn't have the risk of fluid overload as with whole blood, especially when multiple transfusions are required. If possible, blood and crystalloid

infusions should be delivered through a fluid warmer. Start type-specific blood when available. FFP generally is infused when the patient shows signs of coagulopathy, usually after 6–8 U of PRBCs. Platelets become depleted with large blood transfusions. Platelet transfusion is also recommended when a coagulopathy develops.

Surgical Management

The management of ruptured ectopic pregnancy is surgical, which may be laparoscopic or open approach.

Laparoscopy or Laparotomy?

Type of surgical approach is decided by the hemodynamic condition of the patient, size and site of ectopic mass, and surgeon's expertise. A ruptured ectopic pregnancy is not necessarily an indication for laparotomy, but if the patient is hemodynamically unstable and quick clamping of the bleeding vessels is indicated in order to prevent further deterioration of the patient, laparotomy may be the better option. Cornual and interstitial pregnancies were traditionally managed by laparotomy, but laparoscopy is also described by those who have expertise in laparoscopic surgeries.

Laparoscopy has some advantages over laparotomy. Laparoscopic approach is preferred to open approach in a hemodynamically stable patient. Advantages of laparoscopic approach are shorter operative times, less intraoperative blood loss, lower requirements for analgesia, and shorter hospital stay [23–25]. Patients treated by laparotomy had more adhesions compared to those treated by laparoscopy. But tubal patency after salpingostomy was similar in both laparoscopy and laparotomy.

Cochrane review has confirmed that laparoscopic salpingostomy was associated with decreased cost, operative time, blood loss, and hospital stay compared to salpingostomy done through laparotomy [26].

Minilaparotomy may be another option when laparoscopy is not feasible or available. Postoperative pain, recovery time, and complication rates were found to be less when compared to laparotomy [27].

Salpingectomy or Salpingostomy?

Be it laparoscopy or laparotomy, the surgery mostly performed is salpingectomy, where the entire fallopian tube is removed.

Tubal surgery may be conservative when the tube is salvaged, as in salpingotomy, salpingostomy, and fimbrial expression of ectopic pregnancy. Salpingectomy is considered as radical surgery. In situations where the contralateral tube is unhealthy, salpingostomy may be done. Studies have shown that chances for subsequent IUP are not increased after salpingostomy compared with salpingectomy. Linear salpingostomy is considered in unruptured ectopic pregnancy and when the rest of the affected tube appears to be normal. If the contralateral tube appears damaged, salpingostomy has to be considered in a woman who wants to retain fertility. Conservative surgical methods also expose the woman to the risk of reactionary hemorrhage in addition to the possibility of ectopic pregnancy in that tube in the future. Salpingectomy is the mainstay in the management [28].

Surgical Techniques

Salpingostomy: A linear incision is made on the antimesenteric border of the tube, and products of conception are removed. The incision may be made by scalpel or needle tip cautery. This may be done through laparoscopy or laparotomy. Contraindications for salpingostomy are ruptured tube, severely damaged tube, and previous history of ectopic pregnancy on the same side. If too much of cauterization is required to achieve hemostasis, that tube is better removed.

There are several methods for laparoscopic salpingectomy. One approach is to push a pretied surgical loop around the tube using a grasping forceps. The knot is tightened; the tube is then resected and removed. A second loop can be placed on the excised stump.

Alternatively, electrosurgery can be used to fulgurate vessels in the mesosalpinx, followed by resection of the tube with scissors. It is important

to elevate the tube and apply the coagulation very close to the tube to avoid inadvertently damaging the ovarian vessels. The cornual portion of the tube is excised close to the uterus.

Salpingectomy: If laparotomy is performed, total salpingectomy is the usual procedure. It is done by placing a clamp across the mesosalpinx and then placing a second clamp across the proximal portion of the fallopian tube as close as possible to the cornua. The tips of the clamps should touch to completely occlude the vessels in the mesosalpinx. The tube is then excised and the pedicles ligated using a 2-0 or 3-0 synthetic absorbable suture.

Hajenius and associates conducted a Cochrane Database review in 2007.

Their findings showed:

1. No significant increase in overall tubal pregnancy following salpingostomy.
2. Both methods were followed by a similar number of intrauterine pregnancies.
3. Fewer subsequent ectopic pregnancy following laparoscopy, though this was not statistically significant.
4. Laparoscopy resulted in shorter operative time, less blood loss, less analgesic requirements, and shorter hospital stay.

Another main concern about salpingostomy is the possibility of persistent trophoblastic disease.

Persistent Trophoblast

It complicates 5–20 % of salpingostomies, which can be diagnosed by a failure of serum β-hCG levels to fall. β-hCG values fall quickly and are at 10 % of preoperative values by day 12 [29].

If the serum β-hCG on the first postoperative day is less than 50 % of the preoperative value, persistent trophoblast rarely occurs [30].

Factors that increase the risk of persistent trophoblast are [31]:

(a) Serum β-hCG concentrations >3,000 IU/l
(b) Pregnancy <2 cm in size
(c) Implantation medial to salpingostomy site
(d) Early therapy that is before 6 weeks

Women should be followed up with serial β-hCG measurements, and systemic methotrexate treatment may be required if the levels fail to fall as expected.

So in all patients planning for salpingostomy, a preoperative β-hCG estimation and postoperative follow-up are mandatory.

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Cardiogenic Shock in Pregnancy

20

Sourya Acharya

Introduction

Cardiogenic shock (CS) is characterised by systolic arterial hypotension (<90 mmHg) due to severe depression of the cardiac index [<2.2 (L/min)/m^2] and an elevated pulmonary capillary wedge pressure (PCWP) >18 mmHg.

Systolic and diastolic myocardial dysfunction leads to severe reduction in cardiac output which causes progressive peripheral tissue and cardiac ischemia, and the back pressure/elevated filling pressure leads to pulmonary congestion.

Initially a number of compensatory mechanisms are activated in order to support the circulation, but later on, these compensatory mechanisms may become maladaptive and produce a worsening of haemodynamics. A vicious spiral of progressive myocardial dysfunction occurs that ultimately results in death.

Systemic Compensatory Mechanisms in Cardiogenic Shock

1. Increased sympathetic activation
2. Activation of renin-aldosterone axis
3. Increase in ADH secretion
4. Secretion of atrial natriuretic peptide (ANP)

In cardiogenic shock, the left ventricle is not able to pump sufficient blood to meet the metabolic demands of the tissues. The compensatory response is tachycardia, but, eventually, hypervolemia, pulmonary venous congestion and generalised oedema occur. Inadequate oxygen delivery leads to cellular damage, multiorgan failure and ultimately death. The clinical signs of cardiogenic shock are distended neck veins, dyspnoea, tachypnoea, the presence of a third heart sound, systolic or diastolic murmurs and generalised oedema.

As shown in Table 20.1, changes in cardiovascular and other systemic physiology pose significant challenges in acute resuscitative management.

Changes in Blood Volume

1. Maternal blood volume increases from 25 % to 52 % by late pregnancy [1].
2. The plasma volume increases by 45–50 % causing haemodilution or anaemia of pregnancy, which is at its maximum by 32 weeks of gestation.
3. Red blood cell mass increases by 20 % causing secondary erythropoiesis.
4. Elevated levels of oestrogen and progesterone increase plasma aldosterone level and renin

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activity, which in turn promotes sodium retention and increases total body water and hypervolemia.
5. During pregnancy, blood volume increases 1–1.5 L, and the volume of total body water is 6–8 L, 4 L of which is extracellular. All these are necessary for maintaining adequate uteroplacental haemodynamics.

Changes in Blood Pressure

1. Both the systolic and diastolic blood pressure decrease until mid-pregnancy.
2. With gradual recovery to nonpregnant values by late gestation [2], the blood pressure decrease likely occurs because of decreased vascular resistance.

The measurement of brachial artery pressure is not indicative of the uterine arterial blood pressure because the uterine arterial pressure can be extremely low, even when the blood pressure measurement of the arm is normal.

Changes in Heart Rate

The maternal heart rate becomes elevated by 12 weeks' gestation; this reaches and stays at 120 % of the baseline by 32 weeks of pregnancy [3].

The maternal tachycardia may be secondary to cardiac adaptation to volume overload and elevated free thyroxine serum levels.

Changes in Cardiac Output and Stroke Volume

Maternal cardiac output increases 30–50 % during pregnancy [4, 5]. This increase occurs by 10 weeks' gestation and peaks at the end of the second trimester.

The increase in cardiac output during gestation is the result of an increase in heart rate and stroke volume. In late pregnancy, the cardiac output is increased due to the tachycardia rate [5].

Systemic Vascular Resistance

Systemic vascular resistance decreases and reaches a nadir by the 24th week of pregnancy, with a progressive rise towards the baseline value at term. The two important factors that reduce systemic vascular resistance are the dilatation of peripheral blood vessels and the presence of the placental circulation.

The placental vascular bed is a low-resistance vascular system perfused with a large portion of the maternal cardiac output.

Uterine veins increase enormously in size and number during gestation, and uterine vascular resistance is greatly decreased during pregnancy.

Intrapartum Haemodynamics

Cardiac output increases during various phases of labour. The uterine contractions augment cardiac output. Each uterine contraction expresses from 300 to 500 mL of blood [6]. The infusion of blood back into the maternal circulation increases venous return and augments stroke volume.

Common Causes of Cardiogenic Shock in Pregnancy

1. Severe valvular heart diseases (mitral and aortic)
2. Peripartum cardiomyopathy
3. Pulmonary embolism
4. Amniotic fluid

Table 20.1 summarises the physiological changes in cardiorespiratory systems and its effect on resuscitation [7].

Basic Management of Cardiogenic Shock

These cases should be managed by an appropriately skilled and experienced multidisciplinary team, usually in referral centres, in obstetric ICU/HDU.

Table 20.1 Physiological and physical changes in pregnancy

Cardiovascular system
1. Reduced oxygen-carrying capacity – plasma volume increased by up to 50 % dilutional anaemia
2. Heart rate increased by 15–20 bpm – increased CPR circulation demands
3. Cardiac output increased by 40 %/venous return significantly reduced by pressure of gravid uterus on IVC – increased CPR circulation demands
4. Uterine blood flow 10 % of cardiac output at term potential for rapid massive haemorrhage
5. Systemic vascular resistance decreased sequesters blood during CPR
6. Arterial blood pressure decreased by 10–15 mmHg decreased reserve
7. Venous return decreased by pressure of gravid uterus on IVC increased CPR circulation demands. Decreased reserve
Respiratory system
1. Respiratory rate increased, decreased buffering capacity, acidosis more likely
2. Oxygen consumption increased by 20 %; hypoxia develops more quickly
3. Residual capacity decreased by 25 %, decreased buffering capacity, acidosis more likely
4. Arterial PCO_2 decreased buffering capacity, acidosis more likely
5. Laryngeal oedema, increased difficult intubation
Other changes
1. Gastric motility decreased, increased risk of aspiration
2. Lower oesophageal sphincter relaxed, increased risk of aspiration
3. Uterus enlarged; diaphragmatic splinting reduces residual capacity
4. And makes ventilation more difficult
5. Aortocaval compression causes supine hypotension
6. Reduces venous return and significantly impairs CPR
7. Weight increases; large breasts may interfere with intubation; makes ventilation more difficult

Physical Examination

Important general physical signs in cardiogenic shock are:

1. Anxious look.
2. Diaphoresis.
3. Pulse: tachycardia, low volume, thready, narrow pulse pressure and pulsus alternans; irregularity indicates arrhythmias like atrial fibrillation in valvular heart diseases, and absence of a pulse may indicate thromboembolic phenomena.
4. BP: <90 mm systolic.
5. Respiratory rate is increased, accessory muscles of respiration are in action and orthopnoea may be present.
6. Pallor may be present.
7. Cyanosis (central and peripheral depending on the aetiology).
8. JVP will be raised.

CVS examination: Will vary with the underlying condition. Shock with predominant LVF may reveal an S3 gallop.

Respiratory system: Bibasal inspiratory crepitations suggest pulmonary oedema.

Treatment of Cardiogenic Shock

Oxygen therapy: Because of the increased oxygen requirements and rapid onset of hypoxia in pregnancy, it is important to ensure optimal oxygen delivery by adding high-flow 100 % oxygen to whatever method of ventilation is being employed.

Circulation and Pharmacologic Therapy

Circulation

In the absence of breathing despite a clear airway, *chest compressions should be commenced immediately*.

Chest compressions should not be delayed by palpating for a pulse but should be commenced immediately in the absence of breathing and continued until the cardiac rhythm can be checked and cardiac output confirmed. Compressions may be made difficult because of obesity and the tilted position. Hand position should be over the centre of the chest, and it is important to ensure that the direction of compression is perpendicular to the

chest wall; thus, the angle of tilt must be taken into account. Compressions should be performed at a ratio of 30:2 ventilations unless the woman is intubated, in which case chest compressions and ventilations may be desynchronised, with compressions being performed at a rate of 100/min and ventilations at a rate of 10/min.

Defibrillation energy levels should be used same as in the nonpregnant patient.

If defibrillation is required, the same settings should be used as in the nonpregnant patient as there is no change in thoracic impedance.

Two wide-bore cannulae should be inserted as soon as possible.

- Morphine 2–4 mg IV and diuretics (furosemide IV 0.5–1 mg/kg) for reduction of preload which specially helps in improvement of acute pulmonary oedema.
- Nitroglycerine (NTG) can be given IV to reduce preload, if BP is above 100 mmHg systolic. Dose is 10 mcg/min.
- Inotropic and/or vasopressor drug therapy to maintain mean arterial pressure (MAP) of 60 or 65 mmHg.

Ionotropic Therapy

- Dopamine is the drug of choice to improve cardiac contractility in patients with hypotension. Dose: 5–10 mcg/kg/min IV infusion. The infusion rate is adjusted according to the blood pressure and other haemodynamic parameters. The patient may require doses as high as 20 mcg/kg/min.

Features of dobutamine are as follows:

- Dobutamine may be preferable to dopamine if the systolic blood pressure is higher than 80 mmHg. It is initiated with a dose of 7.5–10 mcg/kg/min.
- Compared with dopamine, dobutamine has lesser effect on myocardial oxygen demand.

If the patient remains hypotensive despite moderate doses of dopamine, then norepinephrine may be administered.

Norepinephrine is started at a dose of 0.5 mcg/kg/min and titrated to maintain an MAP of 60 mmHg. The dose of norepinephrine may vary from 0.2 to 1.5 mcg/kg/min. Doses as high as 3.3 mcg/kg/min have been used.

After successful resuscitation, cardiogenic shock cases should be managed by an expert cardiology team. After initial resuscitation, the continuing management of cardiac disease is similar to that in the nonpregnant state, although in many cases delivery will be necessary to facilitate this [8].

Cardiogenic Shock/Acute Pulmonary Oedema in Special Situations

Mitral Stenosis

The most common cause of mitral stenosis is rheumatic valvular disease, which is often first diagnosed during pregnancy. Cardiac decompensation and pulmonary oedema may occur in pregnant women with overt or silent mitral valve stenosis during the second or third trimester. The risk of maternal death is greatest during labour and during the immediate postpartum period. Usually immediately after delivery the preload increases because of autotransfusion from the uterus back into the maternal circulation leading to increase in preload causing pulmonary oedema. This process continues for further 1–3 days postpartum extending the risk of pulmonary oedema [9].

Management

The treatment of mitral stenosis in pregnancy is:

1. Bed rest.
2. Oxygen therapy.
3. Diuretics.
4. Beta-adrenergic receptor blockers like atenolol and metoprolol decrease the incidence of maternal pulmonary oedema without adverse effects on the fetus or neonate [10].

In cases of MS complicated with atrial fibrillation, rate control should be tried with beta-blockers and digoxin. Anticoagulation should be added to prevent systemic embolisation. Cardioversion

should be performed if pharmacologic therapy fails to control the ventricular response.

Anticoagulation in Atrial Fibrillation

Due to the high incidence of embryopathy during the first trimester and bleeding during parturition, warfarin should be used during 12–36 weeks of pregnancy only. Standard therapy during pregnancy would be:

- SC/IV heparin for up to 12 weeks antepartum (aPTT 1.5–2.5 times of normal).
- Warfarin from 12 to 36 weeks (maintain INR 2.5–3.0).
- SC/IV heparin after 36 weeks.
- Low molecular weight heparin (LMWH) may also be used instead of unfractionated heparin.

Surgical management If mitral stenosis is diagnosed before pregnancy, mitral commissurotomy is preferred during pregnancy; the second trimester is the preferred period for any invasive procedure. Percutaneous valvuloplasty using the Inoue balloon technique has become the accepted treatment for patients with severe symptomatic mitral stenosis.

Peripartum Cardiomyopathy

Peripartum cardiomyopathy is an idiopathic disorder that occurs during the last month of pregnancy and up to 6 months postpartum.

The incidence of this disease is one case in 1500–4000 deliveries. The high risk factors associated with this condition are as shown in Table 20.2.

Table 20.2 Peripartum cardiomyopathy

Risk factors
1. Old age
2. Multiparity
3. Twin gestation
4. Pre-eclampsia
5. Viral myocarditis
6. Abnormal immune response to pregnancy
7. Increased haemodynamic burden of pregnancy
8. Hormonal abnormalities
9. Malnutrition

Clinical Features

Physical examination often reveals tachycardia and tachypnoea, elevated or reduced blood pressure, increased jugular venous pressure, displaced apical impulse, right ventricular heave, murmurs of mitral and tricuspid regurgitation, third heart sound, pulmonary rales and peripheral oedema [10].

The treatment consists of diuretics, vasodilators for afterload reduction, digoxin and careful follow-up. Inflammatory myocarditis may respond to immunosuppressive therapy.

Amniotic Fluid Embolism

Amniotic fluid embolism (AFE) is a dangerous peripartum syndrome characterised by a sudden onset of severe dyspnoea, hypoxemia, haemodynamic collapse, coagulopathy and seizures. AFE is a rare disorder, occurring in one case in 20,000–30,000 pregnancies, but it accounts for 10 % of all maternal deaths [11].

The fetal substance may initiate an anaphylactoid reaction, resulting in endogenous mediator release and causing hypotension, tachycardia, hypoxemia and seizures. This may lead to pulmonary arterial vasospasm and transient pulmonary hypertension, followed by acute cardiogenic shock and pulmonary oedema.

The diagnosis of AFE is based on a characteristic clinical picture. Treatment consists of oxygenation and haemodynamic support. The mortality rate for AFE is high: 86 % of patients may succumb to this disorder. Approximately 40 % of cases have fetal death at the time of presentation, and placental abruption occurs in 50 % of the cases.

Pulmonary Embolism

The risk of developing deep venous thrombosis (DVT) and pulmonary embolism (PE) increases markedly during the latter stages of pregnancy and is greatest during postpartum. Rates of maternal mortality from PE have been reported at 2.6 cases per 100,000.

The signs and symptoms of PE can easily be masked by the physiologic dyspnoea and

tachypnoea of pregnancy. In nonpregnant patients, tachypnoea, dyspnoea, pleuritic chest pain, fear of doom, anxiety and crackles are present in only 50 % or more of patients [12].

Objective Diagnostic Testing

1. Though the quantitative plasma D-dimer enzyme-linked immunosorbent assay (ELISA) rises in the presence of DVT or PE, it has got little diagnostic value in pregnancy because it is normally elevated during second and third trimester.
2. Electrocardiogram

   Various ECG findings are encountered in PE:

1. Sinus tachycardia.
2. S1Q3T3 sign: an S wave in lead I, Q wave in lead III and inverted T wave in lead III. This finding is relatively specific but insensitive.
3. Perhaps the most frequent abnormality is T-wave inversion in leads V_1–V_4.
4. Venous Doppler of the lower limbs to detect DVT.
5. Chest CT: Computed tomography of the chest with intravenous contrast is the principal imaging test for the diagnosis of PE in patients with PE; RV enlargement on chest CT indicates an increased likelihood of death within the next 30 days compared with PE patients who have normal RV size on chest CT. When imaging is continued below the chest to the knee, pelvic and proximal leg DVT also can be diagnosed by CT scanning.
6. Lung scanning (V/Q scan) is used mostly for patients who cannot tolerate intravenous contrast. Small particulate aggregates of albumin labelled with a gamma-emitting radionuclide are injected intravenously and are trapped in the pulmonary capillary bed. The perfusion scan defect indicates absent or decreased blood flow, possibly due to PE. Ventilation scans, obtained with a radiolabelled inhaled gas such as xenon or krypton, improve the specificity of the perfusion scan. A high-probability scan for PE is defined as one that indicates two or more segmental perfusion defects in the presence of normal ventilation.
7. Echocardiography is not a reliable diagnostic imaging tool for acute PE because most patients with PE have normal echocardiograms. Transthoracic echocardiography rarely images thrombus directly. The best-known indirect sign of PE on transthoracic echocardiography is McConnell's sign: hypokinesis of the RV free wall with normal motion of the RV apex.
8. Pulmonary angiography: Chest CT with contrast has virtually replaced invasive pulmonary angiography as a diagnostic test. Invasive catheter-based diagnostic testing is reserved for patients with technically unsatisfactory chest CTs and those in whom an interventional procedure such as catheter-directed thrombolysis or embolectomy is planned. A definitive diagnosis of PE depends on visualisation of an intraluminal filling defect in more than one projection.

Management

PE is a medical emergency. Treatment is with intravenous unfractionated heparin, unless the patient has a high risk or contraindication to the use of any anticoagulants. The initial loading dose should be 5000–10,000 U. Following loading, an infusion of 18 U/kg is started. Monitor and keep the activated partial thromboplastin time in the therapeutic range, which is 1.5–2 times the baseline value. Heparin is usually given throughout pregnancy. Warfarin should be avoided throughout pregnancy because it can cause embryopathy. After delivery warfarin can be initiated and continued for at least 6 months with INR of 2.5–3 as target [13].

Although data are relatively modest, low molecular weight heparin (LMWH), which does not cross the placenta, can be administered once a day and does not require monitoring.

Vena cava filters are positioned within the infrarenal inferior vena cava to trap thrombi arising from the lower extremities. Patients with documented venous thromboembolic disease who have contraindications to anticoagulation therapy or in whom conventional therapy has failed are candidates for inferior vena cava filter placement.

Thrombolysis in Pregnancy

Considering that rt-PA does not cross the placenta and taking into account that the complication rates do not exceed those of large randomised controlled trials, thrombolytic therapy should not be withheld in pregnant patients in case of life-threatening or potentially debilitating thromboembolic disease [14].

Obstetric Management in Case of Cardiogenic Shock

Aims of obstetric management should focus on left lateral tilt to relieve vena caval pressure, CTG monitoring of fetal heart rate, O_2 therapy and judicious use of tocolytics if there is fetal distress and to maintain perfusion to uterus and planned delivery.

In worst situation if the general condition of patient worsens and there is no response to correctly performed CPR within 4 min of acute cardiac emergency following cardiogenic shock, time should not be wasted by moving the woman to an operating theatre; a perimortem caesarean section can be performed anywhere, with a scalpel being the only essential equipment required. With no circulation, blood loss is minimal and no anaesthetic is required.

To ensure there are no delays in executing a perimortem caesarean section when indicated, the equipment necessary should be immediately available on the resuscitation trolley. The operator should use the incision that will facilitate the most rapid access. In terms of the best incision to use, a midline abdominal incision and a classic uterine incision will give the most rapid access, but many will be unfamiliar with this approach, and as delivery can be achieved rapidly with a transverse approach, the operators should use the approach they are most comfortable with [15].

Summary

The multidisciplinary team approach should manage the emergency with the following aims.

Aims of Management of Cardiogenic Shock

(a) For mother
   1. Search for underlying cause.
   2. Decrease preload: fluid restriction, diuretics and spironolactone.
   3. Decrease afterload: GTN, hydralazine, SNP, IABP and AC-I's contraindicated.
   4. Optimise contractility: milrinone, dobutamine (but reduces placental blood flow in animal models), adrenaline and VAD.
   5. Increase coronary oxygenation + perfusion: O_2, Hb, stents, CABG and IABP.
   6. Decrease myocardial work: IABP, VAD, VA ECMO and beta-blockers once stable.

(b) For fetus
   1. CTG monitoring, oxygen therapy, tocolytics if distressed and planned or urgent delivery depending upon fetomaternal general condition

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The Recognition and Management of Maternal Sepsis

21

Karen Orr, Damien Hughes, Claire Jamison, and Paul Fogarty

Introduction

In 1990, the United Nations set out to reduce maternal mortality by 75 % by the year 2015 [67]. Whilst there has been a notable reduction from approximately 523,000 deaths worldwide in 1990 to 289,000 in 2013, further improvements will have to occur to reach the desired target. 99 % of these deaths occur in developing nations, and a subsection is invariably preventable [71].

Worldwide, between the years 2003 and 2009, an estimated 261,000 (10.7 %) of deaths occurred as the result of maternal sepsis. In developing nations, 10.7 % of maternal deaths were due to sepsis compared to 4.7 % in developed countries [57]. Worldwide the incidence of maternal sepsis is increasing as a 2006 analysis identified the percentage of maternal deaths occurring as the result of sepsis as 2.1 % in developed countries and between 7.7 % and 11.6 % in developing nations [37].

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The incidence of maternal sepsis not resulting in death is harder to estimate but is thought to occur in between 0.1 and 0.6 per 1000 deliveries in developed nations [5]. Whilst still relatively uncommon in comparison to other obstetric emergencies, the mortality rate is significant and can approach to 10 % [66]. In developing nations, the incidence varies between 0.03 % and 0.7 % with a mortality rate of 33.3 % [36].

A recent UK study examining severe maternal sepsis morbidity showed that for every 1 woman who died, 14 suffer life-threatening septic shock. The incidence of severe sepsis was 47 cases per 100,000 and septic shock 9.1 per 100,000 maternities. Patients with Group A Streptococcal infections were more likely to progress to septic shock [3].

Within the UK, whilst the overall maternal mortality rate has fallen from 13.95 per 100,000 maternities in the years 2003–2005 to 11.39 per 100,000 maternities in the 2006–2008 trienniums, the incidence of sepsis-induced deaths increased from 0.85 to 1.13 per 100,000 pregnancies in the same time periods and, in that report, was the leading cause of direct maternal deaths. Substandard care was identified in 69 % of deaths from sepsis in the 2006–2008 trienniums, comparing to 61 % across all cause fatalities [13].

The recently published report into UK maternal deaths from the years 2009 to 2012 has shown a statistically significant reduction in overall maternal deaths to 10.1 per 100,000 pregnancies.

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Almost 25 % of women who died had severe sepsis, and although the most recent rates of fatal genital tract sepsis fell to 0.5 per 100,000 maternities, genital tract sepsis represented less than a quarter of sepsis deaths, with the remainder being caused by influenza and other infections [38].

The significant reduction in the rate of genital tract sepsis is thought to be as the result of increased public and professional awareness of the recognition and management of sepsis through various publications and initiatives [17, 54, 63]. It should be noted with caution, however, that scarlet fever and the resulting increased risk of Group A maternal Streptococcal infection tend to be cyclical in nature, with peaks occurring approximately every 4 years [38]. Ongoing vigilance is therefore imperative.

Sepsis can present at any stage of pregnancy and, given its frequently insidious course, should never be underestimated. Pregnant or recently pregnant women can rapidly descend into fulminant sepsis with resultant multi-organ failure unless early identification and prompt treatment are instituted. Frequently the combination of a young population alongside the physiological changes of pregnancy masks the development and progression of simple infections into life-threatening situations. This chapter will focus on the key messages concerned with the diagnosis and management of maternal sepsis.

Definitions

The Surviving Sepsis Campaign defines sepsis as "the presence (probable or documented) of infection, together with systemic manifestations of infection" and provides a useful diagnostic classification as detailed in Table 21.1 [17]. Severe sepsis is defined as "sepsis-induced tissue hypoperfusion or organ dysfunction", and the features are described in Table 21.2 [17].

Whilst the criteria outlined in Table 21.1 are highly sensitive for sepsis, they have low specificity, and it is vital that they are applied in the setting intended, i.e. suspected or confirmed infection. Little work has been done to validate these criteria in the setting of maternal sepsis, but small studies have shown a sensitivity of 100 % but a specificity of 17 % with a positive predictive value of only 1.7 % in accurately identifying sepsis in pregnancy [42]. Further difficulty in applying these criteria will occur at particular stages of pregnancy, e.g. active labour with additional confounding factors in operation.

In the absence of fully validated criteria for maternal sepsis, it is reasonable to have a high index of suspicion for the presence of sepsis should a mother display these features and, conversely, relevant variables should be actively and regularly sought in women with confirmed or suspected infection in order to determine the current severity and risk of further deterioration from the infection.

Table 21.1 Diagnostic criteria for sepsis [17]

Infection, documented or suspected, and some of the following:
General variables:
Fever (>38.3 °C)
Hypothermia (core temperature <36 °C)
Heart rate >90/min
Tachypnea (>20 breaths/min)
Altered mental status
Significant oedema or positive fluid balance (>20 mL/kg over 24 h)
Hyperglycaemia (plasma glucose >140 mg/dL or 7.7 mmol/L) in the absence of diabetes
Inflammatory variables
Leucocytosis (WBC count >12,000/μL)
Leukopenia (WBC count <4000/μL)
Normal WBC count with greater than 10 % immature forms
Plasma C-reactive protein more than two sd above the normal value
Plasma procalcitonin more than two sd above the normal value
Haemodynamic variables
Arterial hypotension (SBP <90 mmHg, MAP <70 mmHg or an SBP decrease >40 mmHg)
Organ dysfunction variables
Arterial hypoxaemia (PaO_2/Fio_2 <300)
Acute oliguria (urine output < 0.5 mL/kg/h for at least 2 h despite adequate fluid resuscitation)
Creatinine increase >0.5 mg/dL or 44.2 μmol/L
Coagulation abnormalities (INR >1.5 or APTT >60 s)
Ileus (absent bowel sounds)
Thrombocytopenia (platelet count <100,000 μL–1)
Hyperbilirubinaemia (plasma total bilirubin >4 mg/dL or 70 μmol/L)
Tissue perfusion variables
Hyperlactataemia (>1 mmol/L)
Decreased capillary refill or mottling

Table 21.2 Features of severe sepsis (sepsis-induced tissue hypoperfusion or organ dysfunction) [17]

Sepsis-induced hypotension
Lactate >2 mmol/L
Urine output <0.5 mL/kg/h for more than 2 h despite adequate fluid resuscitation
Acute lung injury with PaO_2/FIO_2 <250 in the absence of pneumonia as infection source
Acute lung injury with PaO_2/FIO_2 <200 in the presence of pneumonia as infection source
Creatinine >2.0 mg/dL (176.8 μmol/L)
Bilirubin >2 mg/dL (34.2 μmol/L)
Platelet count <100,000 μL
Coagulopathy (international normalised ratio >1.5)

The 2011 Centre for Maternal and Child Enquiries (CMACE) report into maternal deaths in the UK suggested that any of the following "red flag" signs and symptoms should prompt urgent assessment for underlying sepsis:

- Pyrexia >38 °C or unexplained hypothermia.
- Sustained tachycardia >100/min.
- Breathlessness, particularly a respiratory rate >20/min.
- Abdominal or chest pain.
- Diarrhoea and/or vomiting.
- Reduced or absent foetal movements or absent foetal heart.
- Spontaneous rupture of membranes or significant vaginal discharge.
- Uterine or renal angle pain and tenderness.
- The woman is generally unwell or seems unduly anxious, distressed or panicky.
- Persistent vaginal bleeding and abdominal pain post-delivery.

Whilst these symptoms are not unique to infection, sepsis should be considered in the differential diagnosis and actively ruled out or treated [13]. All pregnant or recently pregnant women who are unwell should have appropriate observations recorded and acted upon [38].

Risk Factors for the Development of Maternal Sepsis

Whilst any pregnant or recently pregnant woman has the potential to develop sepsis, certain women present a higher risk, which should alert the clinician to the need for a high index of suspicion (Tables 21.3 and 21.4).

Table 21.3 Risk factors for maternal death from sepsis (developed countries) [62]

Caesarean section (emergency)
Prolonged rupture of membranes
Retained products of conception
Premature labour or miscarriage
History of pelvic or other infection
Interventions, e.g. cerclage, multiple vaginal examinations
Low income
Obesity
Diabetes
Anaemia
Recent sore throat or upper respiratory tract infection in family
Winter months
Migrants from developing countries

Table 21.4 Risk factors for maternal death from sepsis (developing countries) [62]

Poverty
Young age
First pregnancy
Anaemia
Home delivery without trained birth assistant
Specified traditional birth assistant practices
Failure to recognise severity
Distance from healthcare facilities
Lack of medical resources

As population obesity levels rise, associated maternal complications also increase, including the risk of life-threatening sepsis. In the mid-2000s, the prevalence of maternal obesity in the UK was approximately 20 % [28]. One population-based study identified an odds ratio of 2.12 for obese women developing sepsis in pregnancy after controlling for mode of delivery [1], and another study showed a non-significantly increased odds ratio of 1.6 in overweight woman with a body mass index of 25–30 [40]. This heightened risk has been attributed to the increased risk of wound, genitourinary and uterine infection [1].

In the 2011 CMACE report, 8/29 deaths from sepsis occurred before 24-week gestation. Infection should be considered in all cases of termination of pregnancy or miscarriage where

persistent abdominal pain, pyrexia or ongoing bleeding occurs [13].

Diabetes appears to be a risk factor not only for the development of uncomplicated maternal sepsis but also progression to life-threatening sepsis with a 47 % increase in the risk of developing severe sepsis compared with nondiabetic women [2].

Caesarean section is a well-recognised factor in the development of maternal sepsis through wound, genital tract and intra-abdominal infections but additionally respiratory and urinary tract sources. A population study of 1.6 million mothers identified an adjusted odds ratio of 1.99 for the development of sepsis following Caesarean section compared with vaginal delivery [2].

Risk appears to be cumulative with a 25 % increase in the risk of uncomplicated sepsis and a 57 % rise in the progression to severe sepsis for each additional risk factor encountered [2].

Management Priorities

The priorities in management are outlined in Table 21.5 and will be discussed in further detail below. Appendix 1 describes an algorithm for management of maternal sepsis.

Early Recognition

Early recognition of the mother who is either displaying signs of sepsis or indeed who is at risk of progressive deterioration is vital and requires careful attention to routine observations as well as a high index of suspicion, particularly in high-risk mothers, as discussed previously.

All healthcare workers dealing with pregnant women should have training in the features of and risk factors for maternal sepsis [54].

Table 21.5 Management priorities in maternal sepsis

Early recognition
Aggressive resuscitation and treatment, including early antibiotics
Source control
Early review by senior doctors and midwives

Regular recording of vital signs should occur and include parameters such as temperature, pulse rate, blood pressure and respiratory rate. These should be recorded at all hospital or general practitioner attendances and repeated at an appropriate frequency in those with or at high risk of developing sepsis. Charts should be filed in patient notes and referred to during subsequent attendances [59].

These observations should be recorded on a Modified Early Obstetric Warning Score (MEOWS) chart [54] with an appropriate algorithm for escalation of frequency of observations and alerting relevant clinicians. It is widely known that physiological abnormalities frequently precede critical illness [15] and therefore allow timely intervention by clinical teams from various specialities including midwifery, anaesthetics, obstetrics, critical care and microbiology amongst others.

There are a number of MEOWS charts in use, with one validated and endorsed following the seventh CEMACH report [14]. This relies on a "track and trigger" system, where, if a parameter falls outside a defined threshold, a response is triggered. As a minimum, 12 hourly recording of temperature, blood pressure, respiratory rate, oxygen saturation, conscious level (using the *A*wake, *V*oice, *P*ain, *U*nresponsive scale) and pain scores should be undertaken. A trigger is defined as a single red parameter or 2, less severe, yellow triggers as shown in Table 21.6. The appropriate

Table 21.6 Limits of trigger thresholds for MEOWS parameters [59]

	Red trigger	Yellow trigger
Temperature, °C	<35 or >38	35–36
Systolic BP, mmHg	<90 or >160	150–160 or 90–100
Diastolic BP, mmHg	>100	90–100
Respiratory rate, breaths/min	<10 or >30	21–30
Heart rate, beats/min	<40 or >120	100–120 or 40–50
Oxygen saturations, %	<95	–
Pain score	–	2–3
Neurological response	Unresponsive, pain	Voice

Table 21.7 Response algorithm for MEOWS triggers (1 "Red" or 2 "Yellow" parameters) [59]

Midwife action	Give oxygen 10 L/min
	If pregnant, left lateral tilt
	Record observations every 30 min
	Call obstetric and anaesthetic teams
	Review observation and prescription chart
Medical response	Respond within 10 min
	Confirm observations
	History and examination
	Develop and implement management plan
	Decide on appropriate clinical area to manage patient
	Consider escalation/referral
	Plan for review
	Document in notes

actions to be undertaken are outlined in Table 21.7 and include increased frequency of observations and urgent clinical review. A sample MEOWS chart is provided in Appendix 2 [59].

The overall sensitivity of MEOWS triggers in predicting maternal morbidity was 89 % with a specificity of 79 %. The positive predictive value was slightly lower at 39 %, but the negative predictive value was extremely high at 98 %, suggesting, if used accurately, that most patients with abnormal physiological parameters, as the result of a condition such as sepsis, will be identified [59].

Timely recognition of maternal sepsis has been highlighted through a number of clinical examples as being key in preventing further deterioration and possible death [3].

A thorough clinical history and examination should complement the measured observations with a number of signs and symptoms being particularly important:

- Otitis media or sinusitis precipitating CNS infections
- Rectal or vaginal pain or discharge suggestive of genital tract infections
- Abdominal pain requiring opioid analgesia following vaginal delivery
- Upper tract or respiratory symptoms alerting the clinician to the possibility of *Group A Streptococcus* or influenza [38]

It is vital that even after sepsis has been identified and treated that ongoing recording of observations at appropriate time intervals is continued.

Aggressive Resuscitation and Treatment

The Surviving Sepsis Campaign (SSC) was founded in 2002 in order to increase awareness of sepsis and septic shock and develop guidelines to aid the evidence-based management of the condition with a view to improving morbidity and mortality. The most recent guidelines developed in 2012 [17] provide an update on the assessment and management of patients with sepsis and septic shock.

A major review of 29,000 patients with sepsis showed a significant difference ($p<0.001$) in mortality between sites displaying high compliance with the SSC resuscitation bundle (38.6 %) and low compliance sites (29.0 %) [44].

The Royal College of Gynaecologists endorses the SSC approach to the management of maternal sepsis and has developed a modified resuscitation bundle to be implemented within the first 6 h of the diagnosis of sepsis, "The Sepsis 6" (Table 21.8) [54].

Table 21.8 Resuscitation bundle: tasks to be carried out within the first 6 h of diagnosis

Obtain blood cultures prior to antibiotic administration
Administer broad-spectrum antibiotic within 1 h of recognition of severe sepsis
Measure serum lactate
In the event of hypotension and/or a serum lactate >4 mmol/L, deliver an initial minimum 20 mL/kg of crystalloid or an equivalent to target a mean arterial pressure of >65 mmHg
Apply facial oxygen to maintain oxygen saturation
In the event of persistent hypotension despite fluid resuscitation (septic shock) and/or lactate >4 mmol/L
(a) Achieve a central venous pressure (CVP) of ≥8 mmHg
(b) Achieve a central venous oxygen saturation (SvO2) ≥70 %

Obtain Blood Cultures Prior to Antibiotic Administration

The rationale for obtaining blood cultures prior to antibiotic administration lies in the fact that although between 30 % and 50 % of patients with severe sepsis have a bacteraemia at presentation [17], the yield can be reduced by as much as 50 % if antibiotics precede the culture sample [45]. The caveat to this recommendation is that unnecessarily delaying antibiotic therapy in order to obtain a blood sample for culture is potentially harmful and should not occur. An opportune time to obtain blood for culture is at the time of establishing intravenous access in order to administer antibiotics. The process should be undertaken using aseptic precautions in order to avoid sample contamination.

If the patient has other indwelling lines, particularly central venous lines, additional samples should be drawn from these devices and thought given to their removal or replacement if they represent a likely source of infection. Alternative sources of infection should be considered and relevant samples obtained if possible, for example, urine, sputum and vaginal and wound swabs. As before, antibiotic administration should not be delayed unnecessarily in order to obtain these samples.

In addition to obtaining blood for culture, other relevant samples according to clinical history should be analysed for the presence of infective organisms and include sputum, urine, breast milk, vaginal, throat or wound swabs, cerebrospinal fluid and stool samples. Whilst these are not as time critical as obtaining blood for culture, they should be obtained as early as possible.

Administer Broad-Spectrum Antibiotic within 1 h of Recognition of Severe Sepsis

Whilst obtaining relevant culture samples is vital, initial antibiotic choice will invariably be empirical, based on the likely source, patient characteristics and, most importantly, local guidelines. Culture results will allow rationalisation of antimicrobial cover but frequently are not available for a number of hours or even days and therefore are often not useful in the early management of maternal sepsis.

Studies have demonstrated a mortality benefit when antibiotics are administered early in patients with severe sepsis [43], and in fact some studies have demonstrated a measurable increase in mortality for each hour delay in antibiotic administration that occurs [17].

The most common organisms present in women dying from sepsis are *Lancefield Group A beta-haemolytic Streptococcus*, *Streptococcus pneumoniae* and *E. coli* [13, 38]. Mixed infections are possible, and the likely causative organisms in prolonged rupture of membranes, urinary tract infections and cerclage are C*oliform* spp. Severe skin infections may be caused by S*taphylococcal* spp. with the less common but highly lethal *Clostridium perfringens*, the cause of gas gangrene also prevalent. Other anaerobic genital tract infections include *Bacteroides* spp. [54].

Any pre-existing allergies should be sought and antimicrobials adjusted accordingly, and in addition, caution should be exercised in pregnant or breastfeeding mothers. Local microbiology services should be consulted for advice in challenging circumstances.

Suggested empirical antimicrobials are listed in Table 21.9 [13].

Patients at risk of hospital-acquired or multidrug-resistant pathogens include those who are immunocompromised, residents in long-term healthcare facilities and frequent or prolonged hospital admissions. Advice should be sought from hospital microbiology services with consideration given to local guidelines if these pathogens are suspected.

The antimicrobial regime should be reassessed after 48–72 h using microbiological and clinical data, with an attempt made to rationalise the drugs to target both the severity of the infection and the causative organisms if identified. The total duration of antimicrobials will typically be around 7–10 days [17].

Measure Serum Lactate

Elevated serum lactate is indicative of inadequate tissue oxygenation, and a level of >4 mmol/L,

Table 21.9 Suggested empirical antimicrobials for maternal sepsis [13]

Unknown organism and the woman is not critically ill	*Co-amoxiclav* (1.2 g 8 hourly) plus *Metronidazole* (500 mg 8 hourly)
	or
	Cefuroxime (1.5 g 8 hourly) plus *Metronidazole* (500 mg 8 hourly)
	or
	Cefotaxime (1–2 g 6- to 12 hourly) plus *metronidazole* (500 mg 8 hourly)
In cases of allergy to penicillin and cephalosporins	*Clarithromycin* (500 mg 12 hourly)
	or
	Clindamycin (600 mg to 1.2 g by intravenous infusion three or four times daily)
	plus
	Gentamicin (3–5 mg/kg daily in divided doses every 8 h by slow intravenous injection)
Severe sepsis or septic shock	*Piperacillin–tazobactam* (4.5 g 8 hourly)
	or
	Ciprofloxacin (600 mg 12 hourly)
	plus
	Gentamicin
	or
	Meropenem (500 mg to 1 g 8 hourly by intravenous injection over 5 min) plus *gentamicin*
Suspected Group A Streptococcal infection	*Clindamycin* (600 mg to 1.2 g by intravenous infusion three or four times daily) is more effective than penicillin as it inhibits exotoxin production

even in the absence of hypotension, is correlated with poorer outcomes because of either increased severity of illness or inadequate treatment [60]. Goal-directed treatment targeted to a reduction in serum lactate within the first 6 h of diagnosis has been shown to significantly reduce 60-day mortality from sepsis [31]. Serum lactate levels are not altered significantly from normal ranges in healthy pregnant women [5], although may transiently rise immediately following delivery [38].

In addition to the measurement of serum lactate, blood should be sent for full blood picture, urea and electrolytes, liver function tests, coagulation screen, arterial blood gas, C reactive protein and major cations such as magnesium and calcium. These should be repeated at appropriate intervals if abnormalities are detected or if there is a significant change in clinical parameters.

In the event of hypotension and/or a serum lactate >4 mmol/L, deliver an initial minimum 20 mL/kg of crystalloid or an equivalent to target a mean arterial pressure (MAP) of >65 mmHg.

Fluid resuscitation using boluses of 20 mL/kg should begin as early as possible with some patients requiring repeated fluid boluses using a MAP of 65 mmHg as a target [17]. Whilst persistent hypotension may be as the result of intravascular depletion, other possible causes such as loss of vasomotor tone and myocardial depression as the result of septic mediators should be considered. Other alternative causes include haemorrhage, oxytocic drugs and renal failure [13].

Whilst MAP >65 mmHg is an achievable and appropriate target, other suggested endpoints to fluid resuscitation include lactate levels, skin perfusion, mental status and, importantly, urine output [17].

The optimal MAP target in pregnant women with sepsis has not been widely studied, and given that the maternal population is often younger with fewer co-morbidities than general patients with sepsis, it is possible that lower MAP values may be well tolerated. In the absence of robust clinical data, it is reasonable to continue to target a MAP of 65 mmHg [5].

The choice of resuscitation fluid is the subject of ongoing debate, particularly with respect to colloids versus crystalloids. Recent evidence showed a significant increase in mortality from 43 % to 51 % when starch-based colloids were used for resuscitation in severe sepsis compared with crystalloids. There was also an increased need for the use of renal replacement therapy (22 % vs. 16 %) in patients given starch-based colloids [51]. Other published evidence has led to the SSC recommendation that starch-based colloids should be avoided in sepsis with crystalloid fluids used in preference [7, 27, 48].

Albumin may be considered as a resuscitation fluid, particularly when large volumes of crystalloids have been administered [17]. Albumin was

shown to be as safe and effective as 0.9 % saline when used in septic patients in a randomised controlled trial [23], with a non-significant trend towards reduced mortality (OR 0.82) with albumin compared to other fluids in a large *meta*-analysis. Subgroup analysis comparing albumin to crystalloid resuscitation showed a significant reduction in mortality with albumin (OR 0.78) [16]. A large randomised trial published after the most recent SSC update did not identify a survival benefit when albumin was used in addition to crystalloids [8]. This may influence future SSC recommendations.

Accurate fluid balance is essential with all administered fluids both enteral and parenteral being recorded along with fluid output such as blood and gastrointestinal loss in addition to regular, preferably hourly urine output [13].

Whilst women with severe sepsis or septic shock will frequently require large volume fluid resuscitation, fluid overload may lead to fatal pulmonary or cerebral oedema. Features suggestive of fluid overload can be difficult to separate from the features of sepsis but may include a sustained elevation in respiratory rate or persistently low oxygen saturations despite high-flow oxygen. If fluid overload is suspected and arterial hypotension persists, involvement of the critical care team may be necessary, with the use of vasoactive drugs to maintain blood pressure considered [13].

Vasopressors may be required in the face of life-threatening hypotension even when circulating volume has not yet been adequately restored, but are most commonly used when hypotension persists, despite adequate fluid resuscitation. Noradrenaline administered via a central line as an infusion is recommended to aid perfusion pressure, targeting a MAP of 65 mmHg, and will invariably require the involvement of critical care specialists. Second-line agents include adrenaline and vasopressin, although again, these should only be used in specialist critical care areas. Dopamine is not considered for routine use but may be used as an alternative agent in selected patients such as those with bradycardic side effects from noradrenaline. In patients with impaired cardiac output resulting from myocardial dysfunction, dobutamine may be beneficial to aid with inotropy, although again, this should occur in a specialist area [17].

Apply Facial Oxygen to Maintain Oxygen Saturation

Initially, high-flow oxygen via a facemask should be applied, and this can be down titrated according to arterial blood gas results and oxygen saturations. Consideration should be given to the use of humidified oxygen to aid comfort and sputum clearance, particularly when oxygen therapy is required for a number of hours or days.

In the Event of Persistent Hypotension Despite Fluid Resuscitation (Septic Shock) and/or Lactate >4 mmol/L

(a) *Achieve a central venous pressure (CVP) of ≥8 mmHg*
(b) *Achieve a central venous oxygen saturation ≥70 % (SvO_2)*

If hypotension with MAP <65 mmHg persists despite adequate fluid resuscitation and vasopressor use, additional resuscitation endpoints should be considered. Patients in this category will invariably have had input from critical care specialists and will be monitored in a high dependency area.

Severe sepsis can be associated with either an abnormally high or low SvO_2, either because of inadequate oxygen delivery due to reduced global cardiac output or altered microcirculatory oxygen delivery or as the result of reduced tissue oxygen extraction owing again to impaired tissue perfusion or direct septic mediator effects.

The SSC campaign is based around the data published by Rivers in 2001 identifying measurable benefit with the use of early goal-directed therapy (GDT). When early GDT was implemented, mortality was reduced from 46.5 % to 30.5 % ($p = 0.009$) and targets such as SvO_2 >70 % and CVP >8 mmHg were resuscitation endpoints in this study. Patients in the early GDT group received more fluid therapy (5 vs. 3.5 L, $p < 0.001$), suggesting the relationship between early aggressive fluid resuscitation and survival [53].

Two recent large randomised controlled trials have, however, failed to confirm the benefit seen with early GDT. The ProCESS trial compared the use of early GDT, protocol-based standard care with usual care in patients with septic shock and demonstrated no difference in either 90-day- or 1-year mortality or the need for organ support [74]. The ARISE trial confirmed these findings and again compared early GDT with usual care in patients with septic shock, with no difference found in 90-day mortality [64].

Following these publications, the SSC have made the following recommendations:

- Monitoring and targeting CVP and SvO_2 does not necessarily confer survival benefit in patients with sepsis and is no longer evidence based.
- No harm was identified in using these parameters.
- No changes have been made to the current SSC guidelines, but modifications may occur with future updates [17].

Source Control

Source control is the process of definitively managing the focus of infection, frequently utilising surgical intervention. Potential foci of infection are outlined in Table 21.10 [5].

Table 21.10 Potential foci of infection in maternal sepsis [5]

Source	Response
Chorioamnionitis	Delivery of baby
Retained products of conception	Evacuation
Retained placenta (despite medical management)	Manual evacuation
Uterine or bowel injury (usually postpartum)	Laparotomy
Wound infection	Debridement
Incomplete miscarriage	Medical or surgical evacuation
Severe mastitis/breast abscess	Consider incision and drainage
Perineal abscess	Incision and drainage
Genital tract trauma with infection/abscess	Debridement and/or drainage

Review by Senior Doctors/Midwives

It goes without saying that it is inappropriate for junior staff to manage high-risk women without the support of more experienced colleagues as well as outside referral to additional teams such as critical care, microbiology and general medicine. This was highlighted as a key area of management in the most recent MBRRACE report and consultant-to-consultant referral considered appropriate when specialist advice is needed [38].

Supportive Therapy

There are a number of additional areas of focus that should be attended to when managing a woman with severe sepsis or septic shock, most of which are common to the care of any patient with a critical illness. These are outlined in Table 21.11.

Glucose Control

Within the general population with severe sepsis or septic shock, the recommendation is to maintain blood glucose levels less than 180 mg/dL, having commenced protocolised insulin therapy when two consecutive readings are greater than 180 mg/dL. Blood glucose levels should be monitored every 1–2 h following commencement of insulin therapy until stabilised; following which, 4-hourly monitoring should be undertaken [17]. The choice of insulin therapy should conform to local hospital protocols but consideration given to the use of continuous intravenous insulin infusions alongside intravenous fluids containing potassium. Careful monitoring of electrolytes particularly serum potassium and sodium levels as well as fluid balance should be undertaken.

Table 21.11 Supportive therapy in severe sepsis [17]

Glucose control
Venous thromboembolism prophylaxis
Stress ulcer prophylaxis
Avoiding anaemia

The target of 180 mg/dL is based on evidence from the NICE-SUGAR trial, which identified an increased mortality when blood glucose levels of less than 110 mg/dL ("intensive insulin therapy") were targeted in comparison to the more modest 180 mg/dL. The increased mortality was felt to be secondary to the significantly higher incidence of inadvertent hypoglycaemic episodes seen when lower blood glucose targets were used [65]. Whilst subsequent *meta*-analyses did not confirm this higher level of mortality, no reduction in mortality and therefore patient benefit was seen when intensive insulin therapy was instituted [26, 35].

Whilst the use of capillary samples to obtain blood for glucose analysis is convenient, caution should be exercised in patients with severe sepsis in whom peripheral circulation is compromised, leading to falsely elevated or lowered capillary glucose levels. In these patients, serum glucose levels should be monitored [17].

Pregnant women, particularly obese women, are at risk of the development of impaired glucose tolerance even in the absence of a formal diagnosis of diabetes mellitus, and in physiological states, blood glucose levels may be higher than the non-pregnant population. The recommendations described here are validated in the non-pregnant population, but, in the absence of large-scale evidence, can be extrapolated to the pregnant population with severe sepsis.

Venous Thromboembolism Prophylaxis

Pregnant women with severe sepsis are at a significantly increased risk of venous thromboembolism on account of the combined effects of pregnancy and critical illness. Whilst a full discussion on the use of venous thromboembolism prophylaxis is beyond the scope of this chapter, it is important to briefly discuss its role.

The choice of pharmacological agent will depend on local protocols, but head-to-head studies in the non-pregnant acutely ill population suggested a reduction in the incidence of pulmonary embolism (hazard ratio 0.51) when low molecular weight heparin was used in comparison to unfractionated heparin, although no significant difference in the incidence of deep venous thrombosis was seen [50, 52]. Choice of both agent and dose may need to be adjusted in the presence of kidney injury.

The timing and nature of delivery, including the use of neuraxial blockade, will need to be considered when selecting the drug, dose and frequency.

The use of non-pharmacological methods such as sequential compression devices and graduated compression stockings is recommended in addition to pharmacological agents [50] and is of particular importance when these agents are contraindicated, e.g. major haemorrhage, thrombocytopenia, etc. [17, 34].

Stress Ulcer Prophylaxis

Again, this topic will not be discussed in detail in this chapter, but consideration should be given to the use of pharmacological agents to prevent the development of stress-induced gastric ulceration. Risk factors include coagulopathy, mechanical ventilation and hypotension as well as pre-existing peptic ulceration, and should a woman display these risk factors, consideration should be given to the use of H2-receptor antagonists for prophylaxis [17].

Avoiding Anaemia

It is widely accepted that in the non-bleeding pregnant woman, red cell transfusion should not be considered unless haemoglobin levels are less than 70 g/L [33]. A large multicentre trial comparing transfusion thresholds of 70 and 90 g/L in patients with septic shock found no difference in mortality, ischaemic events and use of life support, suggesting a threshold of 70 g/L is safe in the non-bleeding septic pregnant woman [29].

Adjunctive Therapies

Additional adjunctive therapies are found in Table 21.12.

Table 21.12 Adjunctive therapies in maternal sepsis [17, 54]

Steroid therapy
Intravenous immunoglobulin

Steroid Therapy

The use of steroid therapy in sepsis remains controversial. Initial studies suggested a mortality benefit when steroid therapy was instituted in septic shock unresponsive to vasopressor therapy [4], but this benefit was not confirmed in a large multicentre trial, although this trial included all patients with septic shock rather than just those with vasopressor-resistant shock. Whilst the time to resolution of normotension was faster, there was an increased incidence of superinfection with additional microorganisms with a relative risk of 1.27 [61].

Given that only a small minority of women with maternal septic shock fall into the category of vasopressor-resistant shock, the use of steroid therapy will not be applicable to the vast majority of patients. Consideration of the effects of steroid therapy on maternal blood glucose as well as foetal effects should be made.

Currently the surviving sepsis campaign recommends the use of low-dose steroid therapy in critically unwell patients if septic shock persists despite adequate fluid resuscitation and the use of vasopressor infusions [17].

Intravenous Immunoglobulin

Certain bacteria relevant to maternal sepsis modulate their effects through the production of exotoxins, in particular *Staphylococcus* spp. and *Streptococcus* spp. Immunoglobulin therapy acts through immunomodulation, inhibition of production of tumour necrosis factor and interleukins as well as neutralisation of the superantigen effect of exotoxins [54]. Both the Department of Health [19] and the RCOG [54] recommend the use of intravenous immunoglobulin in severe invasive staphylococcal or streptococcal infection if other therapies have failed. There is no evidence for its use in other, particularly Gram-negative infections, and it is contraindicated in congenital deficiency of immunoglobulin A.

Patient Location and Monitoring

The decision of where to manage the patient will depend on several factors including:

- The severity of the patient's condition including the presence of one or more organ failures
- The stage of pregnancy or labour
- Local arrangements and provision including staffing numbers and skill mix

Whilst not exhaustive, some indications for transfer to the critical care unit are outlined in Table 21.13.

The minimum level of monitoring that should be undertaken is outlined in Table 21.14 but should be adapted according to the needs of the situation.

Infection Control Issues

Infection control measures such as hand washing and equipment sterility are common to the management of all patients with maternal infections. In

Table 21.13 Indications for transfer to critical care in maternal sepsis [54]

System	Indication
Cardiovascular	Persistent hypotension or lactaemia despite adequate fluid resuscitation suggesting the need for vasopressor or inotropic therapy
Respiratory	Pulmonary oedema
	Mechanical ventilation
	Airway protection
Renal	Severe acute kidney injury
	Dialysis
Neurological	Decreased conscious level
Miscellaneous	Multi-organ failure
	Uncorrected acidosis
	Persistent hypothermia

Table 21.14 Minimum monitoring standards in severe maternal sepsis

Observation	Frequency
Regular observations including conscious level, respiratory rate, oxygen saturations, heart rate, blood pressure, temperature	Hourly (or more often if unstable)
Urine output	Hourly
Blood tests including renal function, electrolytes, full blood picture, arterial blood gas, coagulation screen, lactate	Twice daily (more often if abnormal)

any case of suspected or confirmed maternal sepsis, the neonatal team should be informed to ensure optimum management of the baby. Some general infection control measures are found in Table 21.15.

Invasive Group A Streptococcal infection is a notifiable disease in the UK, and advice should be sought from local infection control and microbiology teams. As a minimum, the affected woman should be managed in a single room should facilities exist for her safe management, with scrupulous attention to hand hygiene. Healthcare workers should wear fluid repellent surgical masks with visors at delivery. The neonatal team should be informed to enable prophylaxis for the baby, and close personal and healthcare worker contacts should be monitored for symptom development to enable early treatment if necessary [54].

Specific Causes of Maternal Sepsis

The various aetiologies of maternal sepsis are outlined in Table 21.16 and discussed further below. Some of the specific presenting features will be described, but it is important to recognise the often non-specific presentation of sepsis and to actively seek the features of sepsis described previously. Regardless of the cause, the role of early antimicrobials and other treatments as already discussed is vital.

Of note, delay in surgical intervention in genital tract sepsis was noted as a contributing factor in the deaths of several women in the recent MBRRACE report [38] and, as a result, should be considered early in the management of women with chorioamnionitis, endometritis, retained products of conception and surgical wound infections.

Table 21.15 General infection control measures to reduce maternal sepsis [30]

Purpose	Infection control measure
Avoidance of infection	Identification and reduction of risk factors for maternal sepsis
	Hand hygiene
	Surgical asepsis
	Environmental improvements
	Clean equipment
	Antibiotic prophylaxis
	Training of traditional birth attendants
Early detection of infection	Clinical monitoring
	Screening and treatment for *Group B Streptococci* colonisation
	Screening and treatment for bacterial vaginosis
	Treatment of chorioamnionitis before and during labour
Reduction of complications	Barrier nursing of infected individuals
Behavioural and organisational change	Issue of guidelines
	Training
	Audit/quality improvement

Table 21.16 Specific aetiology of maternal sepsis

Pregnancy related	Chorioamnionitis
	Endometritis
	Retained products of conception including septic abortion
	Wound infection
	Mastitis
Non-pregnancy related	Influenza
	Pneumonia
	Pharyngitis
	Appendicitis
	Cholecystitis
	Pyelonephritis
	Meningitis
Low-income countries	TB
	HIV
	Malaria

Chorioamnionitis

The intrauterine infection, chorioamnionitis, usually results from ascending polymicrobial infection in the setting of ruptured membranes; however, it can occur with intact membranes following invasive procedures or haematogenous spread [46]. The prevalence is thought be around 4 % of all maternities, but complicates up to 10 % of preterm deliveries [21].

Risk factors include prolonged rupture of membranes, *Group B Streptococcus* colonisation, young age, prolonged labour, nulliparity, multiple vaginal examinations, meconium-stained amniotic fluid and bacterial vaginosis. Causative organisms include *Group A and B streptococcus*, anaerobes such as *Bacteroides*, *Mycoplasma* and *E. coli* [46].

Presentation can be non-specific but may include pyrexia, abdominal tenderness, foul smelling liquor and foetal tachycardia or distress. The sequelae of chorioamnionitis involve both maternal and foetal effects, with increased Caesarean section delivery (two to three times more likely), wound infection, pelvic abscess, haemorrhage and maternal or foetal bacteraemia [46].

Women remain at risk following Caesarean delivery as the uterine repair creates an anaerobic environment in which pathogens can thrive. If antibiotic therapy fails, consideration of abscess formation or indeed distant infection should be considered with surgical intervention as appropriate [6].

Endometritis

Endometritis refers to infections of the *endo*-, *myo*- and *peri*-metrium. It frequently presents post-delivery following ascension of bacteria from the genital tract during labour, with colonisation of the decidua and amniotic fluid [46].

The infection is most commonly mixed with both anaerobes (*Peptostreptococcus*, *Bacteroides* and *Clostridium* spp.) and aerobes (*Group B streptococcus*, *Group A streptococcus*, *Enterococcus* and *E. coli*) contributing. Severe infections complicate haematoma formation or the presence of devitalised tissue and can include *Streptococcus pyogenes* and *Staphylococcus aureus*.

Again a high index of suspicion is required for diagnosis, but features may include tachycardia, uterine tenderness and purulent vaginal discharge. It should be noted that uterine tenderness may be absent in severe cases of *Group A Streptococcal* infection, due to denervation of the uterus [6].

Of women developing pyrexia after delivery, only 20 % of patients following a vaginal delivery are diagnosed with endometritis compared to 70 % of those following Caesarean section delivery [46]. Patients with endometritis following Caesarean section are particularly at risk of pelvic abscess formation and peritonitis. Ninety percent of women respond to antibiotic therapy, but if there is no, or an inadequate, response to appropriate anti-microbial therapy after 48–72 h, consideration for further imaging such as a CT scan should be undertaken [46].

Retained Products of Conception including Septic Abortion

The infection of septic abortion is mediated through metritis and can follow incomplete miscarriage or intentional abortion. Patients may present 2–7 days following abortion or miscarriage with non-specific symptoms such as abdominal pain, nausea and fever, and although often present, the patient may not volunteer other symptoms such as vaginal discharge [46].

Both legal and illegal abortions carry huge emotional consequence, and patients may try to conceal the nature of original procedure, requiring careful questioning, management and support.

Source control is of particular importance in this condition with urgent evacuation of retained products of conception being life-saving. Hysterectomy may be required if gas-forming infection is suspected, and this is indicated by a dusky, devitalised uterus with crepitus in the surrounding tissues [46].

Regulation of abortion services is vital in reducing the risks associated with this procedure. In the USA, patients are screened and treated for gonorrhoea and chlamydia when presenting for medical abortion, with a proven reduction in infection rates following the introduction of this policy [24].

Wound Infection

Although the use of prophylactic antibiotics during Caesarean section has reduced the risk of postoperative wound infections, they still occur in up to 6 % of deliveries, most commonly in urgent Caesarean sections in women with ruptured membranes [22]. Other risk factors include increased blood loss, longer operating times, obesity, diabetes mellitus, immunosuppression, smoking, anaemia and low socioeconomic class [46].

Causative organisms are often derived from the endogenous vaginal tract flora and include both Gram- positive and Gram-negative organisms as well as facultative anaerobes [46]. Recurrent abscess formation is a feature of *Panton-Valentine leukocidin-producing Staphylococcal* infection and should be considered in unusual or severe cases [54].

Antibiotic therapy will be sufficient in the majority of cases with consideration given to abscess formation if symptoms persist or worsen after the fourth postoperative day. Imaging or incision and drainage may be required [46].

Necrotising fasciitis is amongst the most feared complication of wound infection and occurs due to rapidly spreading infection of the tissues down to the deep fascia. Typical but not universal signs include extreme pain out of keeping with clinical signs, purple skin discolouration, tissue crepitus and bullae formation. Polymicrobial infections are often involved, but the most common organisms are *Group A Streptococcus* species, *Staphylococcus aureus*, and *Clostridium perfringens* [6]. Urgent, often extensive, surgical debridement can be life-saving, with input from local microbiology services vital. In addition to appropriate antibiotics, immunoglobulin therapy may be indicated.

Although not technically "wounds", intravenous cannula, drains and other invasive devices are a potential source of infection and should be regularly inspected for signs of erythema, pain and discharge. Removal is key in controlling infection, but antibiotics may be needed in addition.

Mastitis

Frequently mastitis is overlooked as a cause of severe sepsis but in the 2011 CMACE report identified two women who died as the result of mastitis-related sepsis, one from *Group A Streptococcus* and the other *S. aureus* [13]. The RCOG recommends that all women with severe mastitis displaying systemic symptoms or those not responding within 48 h to oral antibiotics be referred to hospital for assessment [54].

It often is unilateral and usually presents 1-week postpartum. Clinical features include thickening and hardening of the affected breast, erythema and severe pain and are often preceded by engorgement. *Staphylococcus aureus* is the most common pathogen, but consideration should be given to the involvement of *methicillin-resistant Staphylococcus aureus*, particularly if the woman or neonate had a prolonged hospital admission [46].

Breast milk should be sent for culture and sensitivity along with skin swabs and intravenous antibiotics commenced if the woman has been admitted to hospital. Breast pumping may be beneficial and consideration given to incision and drainage if abscess formation has occurred [46].

Influenza

Between 2009 and 2012, 36 women in the UK died as the result of influenza during the pandemic, and these deaths accounted for 43 % of all deaths from sepsis. Of the women that died, 33 had suspected or confirmed influenza A and 3 had influenza B. Influenza is highly infectious and tends to follow a seasonal pattern, with highest prevalence during winter months [38].

Risk factors identified as causing a more severe clinical picture included pregnancy, obesity, asthma and patients with heart disease. Pregnancy in particular resulted in more severe disease with a four times higher rate of hospital admission and seven times higher rate of intensive care admission in pregnant women with influenza [38].

In 94 % of the women who died in the recent pandemic, influenza was not considered as a cause at initial presentation, leading to delays in diagnosis and treatment [38]. A high index of suspicion should be maintained when women present with respiratory symptoms during times when the community prevalence of influenza is high, with appropriate investigations undertaken. Presenting symptoms can include shortness of breath, fever, myalgia, dry cough and headache, possibly with recent infective contacts.

Influenza vaccination is recommended in pregnancy with evidence to suggest a reduction in maternal morbidity and mortality and improved foetal outcomes including reduced likelihood of premature birth, low birth weight and influenza infection as a neonate [47, 49]. None of the women who died from influenza in the MBRRACE report were vaccinated [38], and as 62 % of deaths occurred after the vaccination programme started, some of these deaths may have been preventable.

The use of neuraminidase inhibitors, such as oseltamivir and zanamivir, in the management of influenza has remained controversial, and pregnant women were excluded from most trials evaluating their use. Observational evidence suggests benefit with the early use of neuraminidase inhibitors [75], and both the Department of Health and RCOG recommend their use in pregnant women with signs of influenza, preferably within 48 h of the onset of symptoms, even in the absence of confirmed infection [18]. Zanamivir is the recommended drug of choice in pregnancy, with oseltamivir suggested for women with asthma, chronic obstructive pulmonary disease or severe complicated H1N1 influenza [18]. These drugs appear safe for both mother and baby when taken in pregnancy based on current evidence [38].

Pneumonia

A number of physiological changes of pregnancy render a woman at greater risk of respiratory infections compared to the non-pregnant women and include increased respiratory demand, reduced chest excursion, reduced functional residual capacity and reduced respiratory reserve. Not only do they increase the risk of infection, they can exacerbate the clinical course of the condition. It is estimated to complicate up to 1.5 per 1000 pregnancies in the USA [32].

All common pathogens involved in the development of pneumonia are causative organisms in pregnancy and include *Streptococcus pneumonia,* which tends to cause pyrexia and a cough productive of rust-coloured sputum, and the atypical pathogens including *Mycoplasma*, which tends to result in a non-productive cough, rash and myalgia [6]. Rarely PVL-associated staphylococcal necrotising pneumonia may occur, carrying a mortality of 70 % in otherwise healthy individuals [54].

A chest x-ray should be considered alongside involvement of respiratory physicians and chest physiotherapists to ensure optimum treatment. Sputum for culture alongside urine for antigen testing, if available, should be sent.

Viral pneumonia can also occur in pregnancy, although albeit uncommonly. *Varicella zoster* pneumonia in pregnancy requiring mechanical ventilation can have a mortality of up to 14 % despite optimal treatment [41]. Chest x-ray may show widespread infiltrates in comparison to the often-localised consolidation of early bacterial pneumonia.

Pharyngitis

Although most throat infections in pregnancy are of viral original and non-severe, approximately 10 % are caused by *Group A Streptococcus*, which can result in genitourinary and systemic infections [54]. If three of the four Centor criteria are present (fever, tonsillar exudate, no cough, tender anterior cervical lymphadenopathy), appropriate antibiotics should be commenced, most commonly a penicillin [12].

Appendicitis

Appendicitis complicates approximately 1 in 1500 pregnancies and can be difficult to diagnose as the appendix is deflected by the expanding fundus, leading to unusual sites of pain and tenderness. The appendix is more likely to rupture during pregnancy (up to 20 %) as the displaced omentum is less able to contain an inflamed appendix [46].

Clinical features include abdominal pain and tenderness, nausea, fever and leucocytosis. Diagnosis is usually made clinically although the risks and benefits of CT imaging must be weighed against the potential for an unnecessary operation if the diagnosis is in doubt [46].

Cholecystitis

Gallstone disease can occur in up to 10 % of pregnancies [68], and acute cholecystitis occurs when there is obstruction of the cystic duct with bacterial infection complicating in up to 85 % of these cases. Clinical features include right upper quadrant pain, fever, nausea and leucocytosis. Diagnosis can be confirmed with non-invasive ultrasound scanning. Conservative management with antibiotics and intravenous fluids may be sufficient, but surgical cholecystectomy may be required in non-resolving cases or in complications such as pancreatitis. Endoscopic retrograde cholangiopancreatography may be beneficial in common bile duct obstruction [46].

Pyelonephritis

The incidence of pyelonephritis in pregnancy is estimated to be 2 % [70]. Early signs and symptoms include dysuria and flank pain and tenderness alongside the more non-specific symptoms of nausea, chills and rigours. Patients may present at any stage of pregnancy, including post partum, although 90 % occur prior to delivery [70]. Specific investigations will include urinalysis with urine microscopy and culture alongside ultrasound scanning of the renal tracts to exclude structural abnormalities or the presence of renal calculi. Screening for and treating asymptomatic bacteriuria in pregnancy reduces the risk of developing pyelonephritis from 20–35 % to 1–4 % [20].

The most common causative organisms include Gram-negative bacilli such as *Escherichia coli* or *Klebsiella* species although other organisms such as *Group B Streptococci* can contribute [70]. The presence of resistant organisms such as extended-spectrum beta lactamase (ESBL) organisms should be considered in women with long-term urinary catheter insertion, prolonged or repeated hospital admissions or long-term residence in a healthcare facility. Such women should be managed with the input of specialist microbiology services and may require carbapenem therapy [54].

Risk factors for the development of pyelonephritis include multiparity, diabetes mellitus, urinary tract stones or malformations and low socioeconomic status [70].

Women with pyelonephritis are more likely to develop anaemia (OR 2.6), septicaemia (OR 56.5), acute kidney injury (OR 16.5), preterm birth (OR 1.3), low birth weight birth (OR 1.3), chorioamnionitis (OR 1.3) and Caesarean delivery (OR 1.2) compared to women without [70].

Tuberculosis

Tuberculosis, whilst still relatively uncommon in the developed world, has an increasing incidence and already represents a huge burden of disease within the developing world [39].

Screening (tuberculin skin test or interferon-gamma release assay test) should be considered in the following situations:

- Close contact with a patient with active TB.
- Concomitant HIV infection.
- Immunocompromised patients.
- Symptoms of TB (weight loss, night sweats, fever and cough).
- Illegal drug users.
- Patients from endemic areas should be managed with a high degree of suspicion (Latin America, Caribbean Africa, Asia, Eastern Europe, Russia) [46].

It should be noted that TB might present differently in pregnancy, with up to half presenting

with non-specific symptoms and extra pulmonary disease [39].

Severe sepsis is uncommon in TB but may occur in those who are immunocompromised such as patients with HIV, and treatment for latent TB is recommended in these patients as the rate of conversion to active TB is approximately 8 % [11]. Involvement of the infectious disease team is vital in these situations and in particular when extra pulmonary disease is suspected.

HIV

HIV is the leading cause of death amongst women of childbearing age in sub-Saharan Africa, and this region also displays the highest rates of maternal mortality [72]. The rate of maternal mortality amongst women with HIV is eight times higher than non-infected women, and this may be as the result of pregnancy accelerating the progression of HIV or HIV increasing the risk of obstetric complications generally [9].

Whilst maternal mortality in women with HIV is difficult to quantify due to a paucity of data, one review identified that 12 % of pregnancy-related deaths occurred as the result of HIV in a region with an overall HIV incidence of 2 %. When the HIV incidence rose to 15 %, the mortality attributable to HIV was 50 %. The authors concluded, based on the estimated incidence of HIV worldwide, that 5 % of pregnancy-related deaths worldwide and 25 % in sub-Saharan Africa are attributable to HIV infection [10].

Looking specifically at sepsis in HIV-infected pregnant women, those who had a vaginal delivery had a rate of infection three times higher than the non-HIV pregnant women, with the increased risk elevated to six times should a Caesarean delivery be undertaken. Women with HIV had an increased risk of wound infection (OR 1.75) and endometritis (OR 1.86) [9].

Benefits have been seen when HIV-infected women are given prophylactic antibiotics during labour [58]; however, the most important modifiable factor in reducing both sepsis and all-cause mortality is ensuring access to antiretroviral therapy for affected individuals [73]. Consultation with an infectious disease specialist is advised with local protocols on the management of pregnant women with HIV infection developed in order to improve outcomes, particularly in regions with high rates of infection.

Malaria

Pregnant women infected with the malarial parasite *Plasmodium falciparum* are at an increased risk of maternal anaemia, low birth weight, intrauterine growth restriction and preterm birth, and many of the effects are thought to occur as the result of placental sequestration. Pregnant women have a three times increased risk of contracting severe malaria compared to the non-pregnant population [56].

Diagnosis of malaria in pregnancy can be challenging as parasites may sequester in the placenta but be undetectable in peripheral blood smears. Placental sampling may be required to confirm diagnosis [25].

The mortality of infection with P. falciparum can approach 50 % in pregnancy. Clinical features are often non-specific and include fatigue, headache and fever with progression to seizures, pulmonary oedema, renal failure and jaundice if untreated [69]. History of travel to an endemic area should be actively sought.

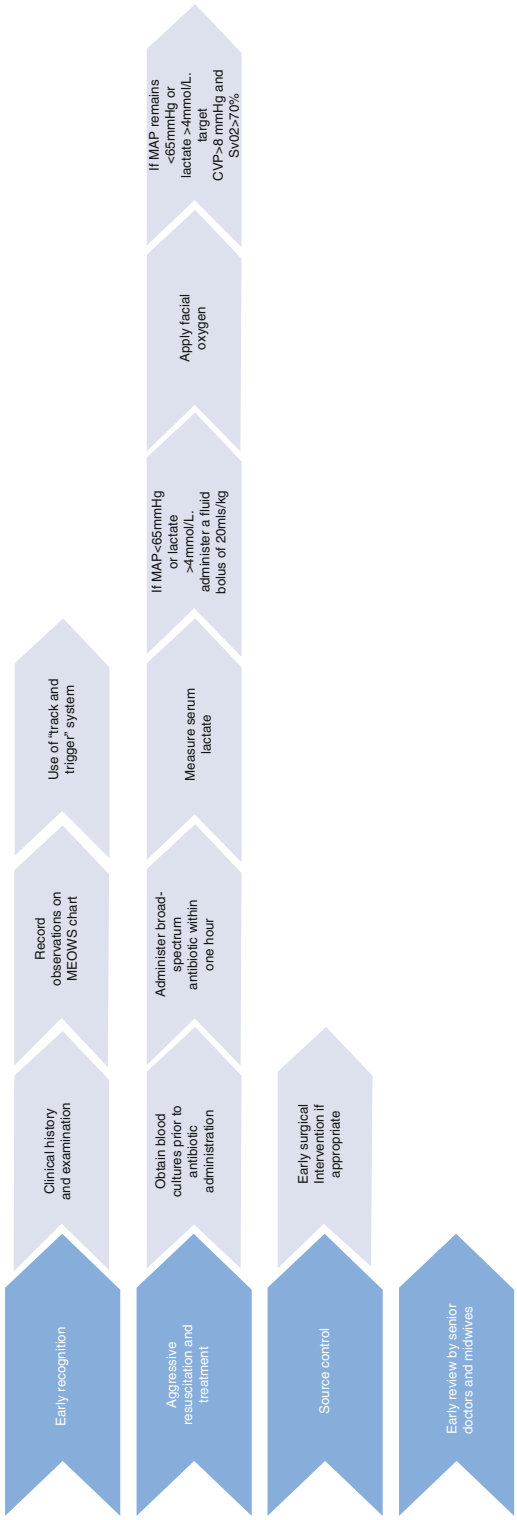
For severe, falciparum malaria, intravenous artesunate is the treatment of choice, with quinine used if artesunate is unavailable. Quinine and clindamycin should be used in uncomplicated falciparum or mixed malaria and chloroquine for other malarial parasites. Primaquine is contraindicated in pregnancy and advice from local infectious disease specialists sought early [55].

Complications of malaria that should be identified early and treated if possible include hypoglycaemia, pulmonary oedema, anaemia, hyperpyrexia, seizures, metabolic acidosis, coagulopathy, renal failure and secondary bacterial infections [55].

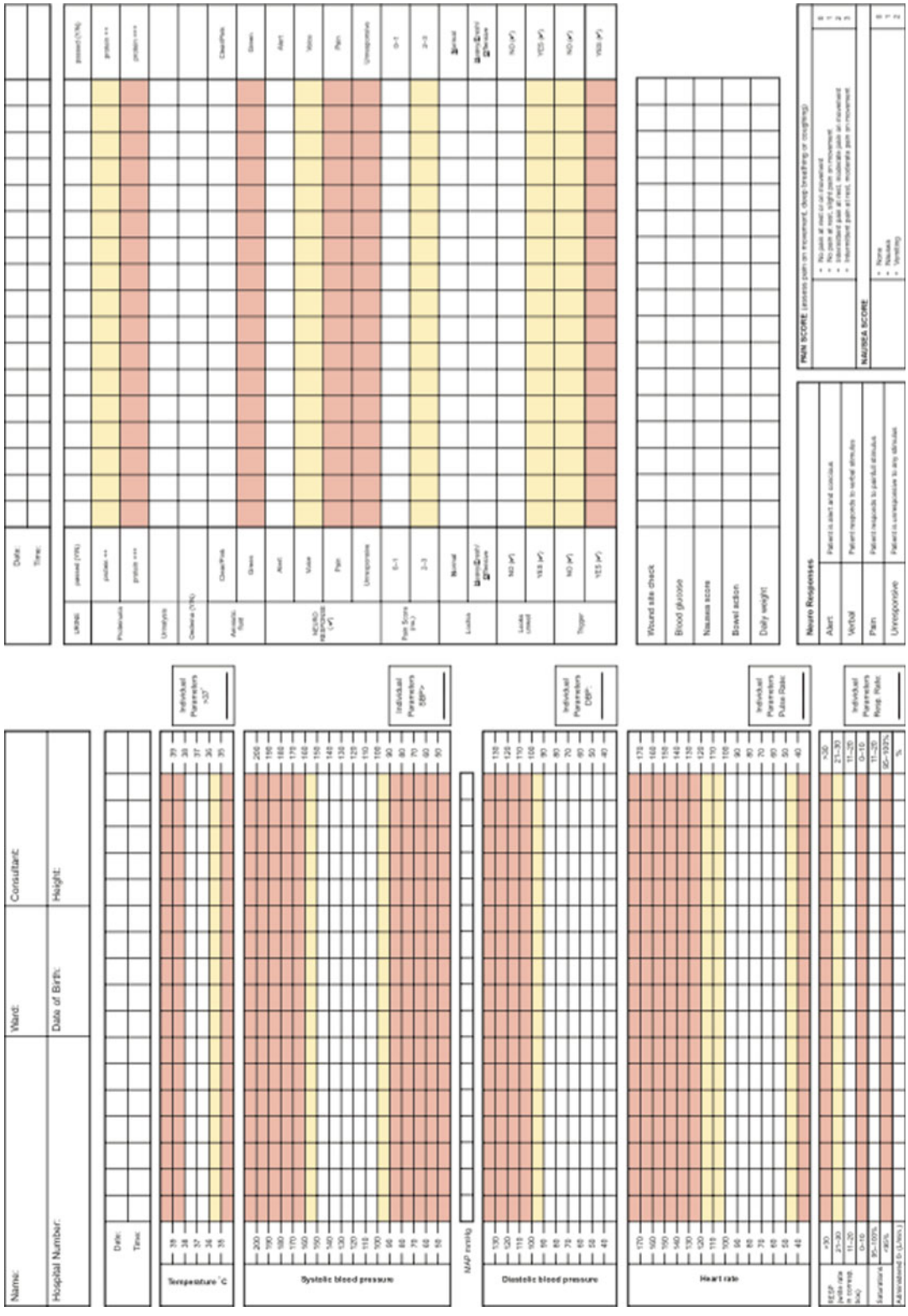
Conclusion

Maternal sepsis remains a significant contributor to maternal mortality, but its impact can be lessened with early recognition, aggressive resuscitation and treatment, source control and input from senior medical, nursing and midwifery staff.

Appendix 1: Algorithm for Management of Maternal Sepsis



Appendix 2: Sample MEOWS Chart [59]



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Anaphylactic Shock in a Pregnant Woman

22

Veena Agrawal

Definition and Prevalence

Anaphylactic shock in a pregnant women is a rare but potentially fatal to mother as well as fetus; most common systems involved are - cutaneous, respiratory, cardiovascular, and gastrointestinal (GI). Anaphylaxis was first termed by Portier and Richet in 1902 when they found that injections of the fluid from the nematocysts of Physalia, the Portuguese man of war, and the tentacles of the sea anemone Actinia induce a violent reaction in dogs that had survived an earlier injection without distress. The term comes from the Ancient Greek ana "against" and phylaxis "protection." Richet won the Nobel Prize in medicine or physiology in 1913 for that [1].

Anaphylaxis during pregnancy can be catastrophic for both mother and infant. The exact incidence of anaphylaxis is unknown; worldwide it is between 0.05 % and 2 % of the population but appears to be increasing [2]. Data regarding the prevalence among pregnant women is limited. Current estimates of incidence suggest that maternal anaphylaxis occurs in approximately 1 in 30,000 pregnancies, although this is based on limited evidence [3].

Classification [4–8]

1. Immunological reaction mediated by IgE antibodies or non-IgE mediated (e.g., IgG and immune complex complement mediated).
2. Non-immunologically mediated reactions, but involve different pathophysiological processes, with similar symptomatology previously called "anaphylactoid" or "pseudo-allergic" – especially by Paul Kallos – and are now called "nonimmune anaphylaxis" according to a consensus of the World Allergy Organization (WAO). This cannot be detected by skin test or in vitro allergy diagnostic procedures.
3. Idiopathic.

Etiology

Medications are the leading cause of anaphylaxis-related deaths [9]. An alteration in immunological status due to increased progesterone level during pregnancy may predispose pregnant women to anaphylaxis even though the high levels of placental histaminase may act as a protective mechanism for the fetus [10].

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1. During pregnancy etiology is same as non-pregnant population [11, 12]:
   (a) Immunological IgE-mediated reactions:
      (i) Foods – certain foods are more likely than others including peanuts, tree nuts, cow's milk, eggs, wheat, soy, fish, and shellfish.
      (ii) Drugs:
         1. Most commonly:
            (a) Penicillin – serious reactions occur about twice as frequently following intramuscular or intravenous administration versus oral administration, but oral administration may also induce anaphylaxis. Neither atopy nor a genetic history of allergic rhinitis, asthma, or eczema is a risk factor for the development of penicillin allergy.
            (b) Other beta-lactam antibiotics. Cephalosporins in penicillin-allergic patients (older agents such as cephalothin, cephalexin, cefadroxil, and cefazolin are more than newer agents such as cefprozil, cefuroxime, ceftazidime, or ceftriaxone due to greater antigenic similarity of the side chain).
            (c) Sulfonamide.
         2. Less frequently:
            (a) Many other antibiotics
            (b) Thiamine, protamine, gamma globulin, formaldehyde, and ethylene oxide
            (c) Muscle relaxants, for example, suxamethonium, alcuronium, vecuronium, pancuronium, and atracurium, used during anesthesia
            (d) Chlorhexidine, insulin, and seminal proteins
      (iii) Latex – sensitization to latex seems to occur more frequently in women than in men and a higher incidence of anaphylactic reactions to latex during obstetric and gynecological surgical procedures [13, 14].
      (iv) Hymenoptera venoms (bee, wasp, yellow jacket, hornet, fire ant) contain phospholipases and hyaluronidases and other proteins which can elicit an IgE-mediated anaphylaxis.
   (b) Non-IgE mediated (e.g., IgG and immune complex complement mediated):
      (i) Whole blood, serum, plasma, serum products, and immunoglobulins cause type II hypersensitivity. These reactions are either by formation of antigen-antibody reactions on the red blood cell surface or from immune complexes resulting in complement activation (anaphylatoxins C3a, C4a, and C5a) or directly by induction of vascular permeability and contraction of smooth muscle. This reaction causes agglutination and lysis of red blood cells and perturbation of mast cells resulting in anaphylaxis [6, 8].
      (ii) Iron sucrose – immune mechanisms and iron agent releasing bioactive, partially unbound iron into the circulation, resulting in oxidative stress, appear to cause severe adverse reactions [15].
   (c) Non-immunological reactions:
      (i) Opioids, dextrans, protamine, and vancomycin involve specific receptors, or non-receptor-mediated mast cell activation can activate several inflammatory pathways, including complement, coagulation, and vasoactive (kallikrein-kinin) systems.
      (ii) Intravenous administration of radiocontrast media.
      (iii) Sulfating agents – sodium and potassium sulfites, bisulfites, metabisulfites, and gaseous sulfur dioxides are added to foods and drinks.

2. During labor and delivery:
   (a) Drugs:
      (i) Most common is prophylactic injection of penicillin or cephalosporin to prevent neonatal group B streptococcal (GBS) infection or to prevent maternal infection after cesarean delivery.
      (ii) Other antibiotics.
      (iii) Labor induction agents – oxytocin and misoprostol [16].
      (iv) In perioperative settings:
         1. Neuromuscular blockers, decreasing order of importance: suxamethonium, vecuronium, atracurium, pancuronium, rocuronium, mivacurium, and cisatracurium.
         2. Epiduralmedications.
         3. General anesthetics.
         4. NRL.
         5. Chlorhexidine [17].
         6. Colloid solution – none of the colloids in clinical use (plasma protein solution, gelatine, hydroxyethyl starch, and dextran) is free from the risk of anaphylactic reactions. Even though the incidence of anaphylactic reactions is low (0.03 %), lethal outcome might be encountered. Pathomechanisms is not known [18].

Augmentation or Summation Anaphylaxis (Risk Factors)

Intensity of the reaction is influenced by the genetic variables, degree of sensitization, age, simultaneous exposure to other allergens, underlying infection, physical exercise or psychological stress, or concomitant medication (e.g., beta-blockers, NSAIDs, α-blockers, β-blockers, angiotensin-converting enzyme [ACE] inhibitors); this phenomenon has been called augmentation or summation anaphylaxis 5.

Pathophysiology [19]

Whether the mechanism is triggered via an immunological or non-immunological pathway or whether through an IgE- or non-IgE-mediated mechanism, the final pathway involves mast cells and basophils, resulting in a similar severe allergic-type response. Repeated exposure to allergens initiates immune response that generates IgE isotype. These IgE antibodies then bind to the high-affinity IgE receptor (FcεRI) on the surface of mast cells and basophils. These mast cells and basophils coated by IgE are "sensitized." On reexposure the allergen may cross-link the mast cell or basophil surface-bound allergen-specific IgE to activate the signal with tyrosine phosphorylation, Ca++ influx, degranulation, and release of pharmacologically active mediators such as histamine, leukotriene, proteases, chemotactic factors, prostaglandins, and cytokines that play an important role in the pathophysiology of anaphylaxis and have an effect on a number of organ systems. The principal effects of these products are vasodilation and smooth muscle contraction. Primary mediators are preformed mediators in granules like histamine, cytokines (TNF-α, IL-1, IL-6), chemoattractants for neutrophils and eosinophils, and enzymes like tryptase, chymase, and cathepsin. Secondary mediators formed after activation are leukotrienes, prostaglandins, and Th2 cytokines – IL-4, IL-5, IL-13, and GM-CSF. Nonimmune anaphylactic reactions may be due to complement activation, coagulation/fibrinolysis system activation, or the direct pharmacological release of mediators. Physiologic responses to mediators include cause vasodilation, fluid extravasation, smooth muscle contraction, increased mucosal secretions, and stimulation of sensory nerve endings (Table 22.1).

Symptoms and Signs

Anaphylaxis is characteristically a disease of fit patients and is rarely seen in critically ill or shocked patients, other than asthmatics. The speed of onset relates to the mechanism of

exposure and the severity of the reaction. Parenteral antigen exposure may cause life-threatening anaphylaxis within minutes, whereas symptoms can be delayed for some hours following oral or topical exposure. Cardiovascular and cutaneous symptoms dominate in adults [20] (Table 22.2).

Cardiovascular disturbances are the hallmark of anaphylaxis in obstetrics [21]. The mechanisms of vasodilation and catecholamine resistance are activation of the adenosine-5′-triphosphate (ATP)-sensitive potassium channels (K_{ATP} channels), acti-

Table 22.1 Physiologic responses to mediators

Histamine	
Binding to H_1 receptors	Coronary artery vasoconstriction, tachycardia, vascular permeability, pruritus, bronchospasm, and rhinorrhea
Binding to both H_1 and H_2 receptors	Headache, flushing, and hypotension
Binding to H_2 receptors	Increase atrial and ventricular contractility, atrial chronotropy, and coronary artery vasodilation
Prostaglandins, principally prostaglandin D_2 (PGD_2)	Bronchospasm and vascular dilatation and mucous secretion act as chemotactic signals for eosinophils and neutrophils
Cytokines – interleukin (IL)-4 and IL-13	Initiate generation of antibody and inflammatory cell responses
Leukotriene B4, LTC4 is converted into LTD4 and LTE4 and platelet-activating factor (PAF)	Hypotension, bronchospasm, and mucous secretion acting as chemotactic signals for eosinophils, neutrophils, and platelet
Proteases, tryptase, and chymase; proteoglycans such as heparin and chondroitin sulfate; and chemokines and cytokines	Activate the kallikrein-kinin contact system, the complement cascade, and coagulation pathways
Eosinophils	May be inflammatory (e.g., release cytotoxic granule-associated proteins) or anti-inflammatory (metabolize vasoactive mediators)

Table 22.2 Symptoms and signs

Cardiovascular signs and symptoms	Signs of shock such as faintness, pallor, or clammy skin
	Tachycardia >100 bpm
	Systolic BP <90 mmHg
	Decreasing level of consciousness
	Myocardial ischemia/angina
	Cardiac arrest
Airway problem	Laryngeal or pharyngeal or/and tongue edema
	Difficulty in breathing and swallowing
	Sensation that the throat is "closing up"
	Hoarse voice
	Stridor
Breathing problem	Shortness of breath and raised respiratory rate
	Wheeze
	Decreased oxygen saturations
	Confusion secondary to hypoxia
	Cyanosis
	Respiratory exhaustion or respiratory arrest
Central nervous system	Disability caused by airway, breathing, or circulation problem
	Sense of impending doom
	Anxiety, panic
	Dizziness
	Confusion
	Headache
	Decreased conscious level
	Incontinence
Gastrointestinal manifestations	Nausea, vomiting
	Abdominal cramps
	Diarrhea
Mucocutaneous symptoms	Exposure – look for skin changes; skin, mucosal, or both skin and mucosal changes often the first feature. Present in over 87 % of anaphylactic reactions
	Erythema – a patchy, or generalized, red rash
	Urticaria (also called hives, nettle rash, weals, or welts) anywhere on the body
	Angioedema – e.g., eyelids and lips, sometimes in the mouth and throat

vation of the inducible form of nitric oxide (NO), and deficiency of the vasopressin hormone. Aortocaval compression by pregnant uterus reduces venous return and thereby reducing cardiac output which is worsened by the peripheral vasodilatation and increased vascular permeability, thus causing intravascular volume depletion. Low outflow and metabolic acidosis with hyperlactatemia induced by anaphylaxis leads to persistent hypotension, cardiovascular collapse, or cardiac arrest. Cardiac abnormalities seen during anaphylactic shock are usually due to an underlying cardiac disease or the side effects of catecholamines administered rather than the anaphylaxis itself. Mucocutaneous signs and symptoms are masked by peripheral vasoconstrictive effect due to compensatory mechanism and appear after the normalization of the arterial blood pressure [22].

Diagnosis

Anaphylaxis is a clinical diagnosis. Clinical presentation is variable. Anaphylactic shock is a serious, rapid-onset, allergic reaction that may cause death. Symptoms and signs include sudden onset and rapid progression of life-threatening airway problem, breathing problem, circulatory problem, loss of consciousness, and/or skin and/or mucosal changes [23, 24] (Table 22.3).

Table 22.3 Clinical criteria for diagnosing anaphylaxis according to the World Allergy Organization

Anaphylaxis is highly likely when any one of the following three criteria is fulfilled:
1. Acute onset of an illness (minutes to several hours) with involvement of the skin, mucosal tissue, or both (e.g., generalized urticaria, itching or flushing, swollen lips-tongue-uvula) *AND AT LEAST ONE OF THE FOLLOWING*
A. Respiratory compromise (e.g., dyspnea, wheezing, bronchospasm, stridor, reduced PEF, hypoxemia)
B. Reduced blood pressure or associated symptoms of end-organ dysfunction (e.g., hypotonia [collapse], syncope, incontinence) OR.
2. Two or more of the following that occur rapidly after exposure to a likely allergen for that patient (minutes to several hours):
A. skin-mucosal tissue symptoms (e.g., generalized urticaria, itch, flush, swollen lips, tongue, or uvula)
B. Respiratory symptoms (e.g., dyspnea, wheezing, bronchospasm, stridor, reduced PEF, hypoxemia)
C. Reduced blood pressure or associated symptoms (e.g., hypotonia [collapse], syncope, incontinence)
D. Persistent gastrointestinal symptoms (e.g., crampy abdominal pain, vomiting) OR
3. Reduced blood pressure of less than 90 mmHg or greater than 30 % decrease from that person's baseline after exposure to known allergen for that patient (minutes to several hours). Normal values for vital signs are different in late pregnancy. Respiratory rate increases by 10 % and heart rate increases by 15 %. Systolic blood pressure does not change but diastolic blood pressure decreases by 15 %. Supine hypotension syndrome occurs in more than 10 % of pregnant women

Clinical Grading System

No validated global clinical grading systems of anaphylactic reactions exist that link the clinical features with severity, urgency, treatment, or outcome. The WAO grading system for SR has been endorsed by the following WAO Regional and National Member Societies: AAAAI, Latin American Society of Allergy and Immunology (SLAAI), Asia Pacific Association of Allergy, Asthma and Clinical Immunology (APAAACI), and the ACAAI. These grades correlated well with epinephrine usage [25]. In grade I reactions, spontaneous improvement can occur without any specific treatment. In more severe cases, the anaphylactic reaction should be treated with appropriate doses of epinephrine and fluid infusion based on its severity and on clinical response (Table 22.4).

Fetal and Maternal Outcomes

Anaphylaxis during pregnancy, labor, and delivery can be catastrophic for the mother and, especially, the infant.

Fetal Outcomes

During pregnancy Common changes in hematological and biochemical parameters occur with pregnancy that impact on maternal/fetal resusci-

Table 22.4 World Allergy Organization grading system

Grade 1	Grade 2	Grade 3	Grade 4	Grade 5
Symptom(s)/sign(s) of one organ system present: *Cutaneous* Generalized pruritus, urticaria, flushing, or sensation of heat or warmth or Angioedema (not laryngeal, tongue, or uvular) or *Upper respiratory* Rhinitis (e.g., sneezing, rhinorrhea, nasal pruritus, and/or nasal congestion) or Throat clearing (itchy throat) or Cough perceived to come from the upper airway, not the lung, larynx, or trachea or *Conjunctival* Conjunctival erythema, pruritus, or tearing *Other* Nausea, metallic taste, or headache	Symptom(s)/sign(s) of more than one organ system present: Measurable but not life-threatening symptoms or *Lower respiratory* Asthma: cough, wheezing, shortness of breath (e.g., less than 40 % PEF or FEV1 drop, responding to an inhaled bronchodilator) or *Gastrointestinal* Abdominal cramps, vomiting, or diarrhea or *Other* Uterine cramps	Life-threatening symptoms: *Lower respiratory* Asthma (e.g., 40 % PEF or FEV1 drop, NOT responding to an inhaled bronchodilator) or *Upper respiratory* Laryngeal, uvula, or tongue edema with or without stridor	*Lower or upper respiratory* Respiratory failure with or without loss of consciousness *Cardiovascular* Hypotension with or without loss of consciousness	Death

Patients may also have a feeling of impending doom, especially in grades 2, 3, or 4

Table 22.5 Biochemical changes in pregnancy

Parameter	Nonpregnant	Term	Pregnancy impact on resuscitative care
PaO_2 (kPa/mmHg)	13.3/100	13.7/103	A rightward shift of the maternal oxyhemoglobin dissociation curve (P50 = 4 kPa/30 mmHg) that represents a compensatory mechanism to improve fetal oxygenation
$PaCO_2$ (kPa/mmHg)	5.3/40	4/30	Maintenance of maternal/fetal CO_2 gradient is important for ongoing fetal CO_2 excretion
HCO_3^- (mmol/L/mEq/L)	24	20	↓ Buffering capacity, acidosis more likely

tation (Adapted from the Royal College of Obstetricians and Gynecology Green-top Guideline No. 56, http://www.rcog.org.uk/files/rcog-corp/GTG56.pdf) (Table 22.5).

Umbilical vein PaO_2 is approximately 35–40 mmHg because it is mixed with deoxygenated blood which is sufficient to saturate fetal hemoglobin to 80–85 % because of the left shift in the hemoglobin oxygen dissociation curve. The oxygen consumption of the healthy fetus is approximately 6.0–5.5 mL/kg/min at term, 5.4 mL/kg/min <28 wks [26, 27]. The oxygen reserve is approximately 42 mL; oxygen is consumed by the fetus at a rate of 20 ml/min, which means that the fetus has an oxygen reserve for 2 min only. However, the fetal oxidative metabolism can be sustained despite reduction in fetal O2 delivery of 40–50 % by redistributing blood flow to the vital organs if fetal hypoxemia is present for brief periods, but further reduction produces anaerobic metabolism, brain damage, and fetal death [28, 29]. The level and duration of maternal hypotension probably determine the extent of injury, while

fetal maturity, antenatal factors which affects the fetal oxygen reserve, possibly dictates the extent and site of injury. Maternal hypoxia, uterine hypoperfusion, umbilical vessel vasoconstriction, and peripheral fetal vasodilation induced by histamine could lead to the impairment of fetal regulation of cerebral flow and induce severe neurological damage. The primary sites affected in full-term neonates are often the basal ganglia and thalamus and in preterm are the deep gray matter, brainstem, and cerebellum. The fetus is at risk of hypoxic-ischemic encephalopathy and permanent central nervous system damage, despite maternal survival in anaphylactic shock [10, 30, 31]. Neonatal neurological abnormalities, including rigidity of the extremities, seizure-like movements, brain damage, hypoxic encephalopathy, and neonatal death, were reported in 46 % of these cases after an ineffective resuscitation.

During labor Neonatal morbidity was reported if anaphylaxis occurred during labor, but no neonatal neurological abnormalities or death were reported when maternal anaphylaxis occurred during cesarean delivery because fetal extraction was concurrently performed during maternal resuscitation [22].

Maternal Outcomes [11]

Maternal signs and symptoms include intense vulvar and vaginal itching, low back pain, uterine cramps, fetal distress, and preterm labor. The delayed use of epinephrine is a risk factor for a poor outcome; nevertheless, some patients still die despite receiving epinephrine. Poor outcomes can occur regardless of the antigenic trigger, and death can occur even in idiopathic anaphylaxis.

Differential Diagnosis [6, 12, 13]

During the first three trimesters, differential diagnosis is similar to the differential diagnosis of anaphylaxis in nonpregnant patients. During labor and delivery, the differential diagnosis also includes all other causes of maternal respiratory distress or cardiovascular compromise, such as pulmonary embolism, pulmonary edema, cardiomyopathy, acute coronary syndrome, mitral stenosis, hypotension, cerebrovascular accident, and AFE.

A. First three trimesters, before labor and delivery:
   1. Common diagnostic dilemmas, such as acute asthma, acute generalized urticaria, acute angioedema, syncope/fainting, panic attack, and acute anxiety attack.
   2. Postprandial syndromes, such as scombroidosis, pollen-food allergy syndrome (oral allergy syndrome), monosodium glutamate reaction, sulfite reaction, and food poisoning.
   3. Upper airway obstruction (other forms), such as nonallergic angioedema, includes hereditary angioedema types I, II, and III.
   4. Shock (other forms), such as hypovolemic, septic, and cardiogenic.
   5. Pulmonary embolism.
   6. Pheochromocytoma.
   7. Other: excess endogenous histamine, such as mastocytosis/clonal mast cell disorder; flush syndromes, such as carcinoid syndrome; certain tumors; and system.
   8. Capillary leak syndrome.

B. Labor and delivery:
   1. Pulmonary embolism (thrombotic) and pulmonary edema
   2. Amniotic fluid embolism
   3. Preeclampsia/eclampsia-associated symptoms, such as laryngopathia gravidarum and seizures
   4. Hypotension caused by spinal block, local anesthetic, or hemorrhage
   5. Cardiac conditions (acquired and congenital)
   6. Cerebrovascular accident
   7. Other

Treatment (Fig. 22.1)

The treatment of anaphylactic shock in pregnant women differs in two respects from that of nonpregnant women. First, normal physiologic

changes occur during pregnancy, and secondly, the mother and the fetus, two patients, have to be managed. Prompt management is critically important. In case of suspicion of anaphylaxis in a pregnant woman, four aspects should be handled: the severity of the anaphylaxis, individual complications regarding a pregnant woman, unfavorable effects of the regularly used treatment during that specific gestation, and the need of fetal extraction based of gestational age.

International consensus on (*ICON*) *anaphylaxis* provides a unique perspective on the principal evidence-based anaphylaxis guidelines developed and published independently from 2010 to 2014 by four allergy/immunology organizations [32]. The primary goal is cardiopulmonary support and reversal of the effects of anaphylaxis with epinephrine (adrenaline). Patients having anaphylaxis should be treated using the airway, breathing, circulation, disability, and exposure (ABCDE) approach. Cardiac collapse and respiratory compromise cause the most urgent concern as they can be fatal and require admission to the ICU. Patients presenting with milder symptoms can rapidly deteriorate and should be closely monitored.

Basic Treatment First-Line Therapy

Take short history; rapidly assess the patient's airway, breathing, and circulation (ABC), mental status, and skin; estimate the body weight (mass) with special consideration of the gestational age of the fetus; and remove exposure to the trigger, if possible (e.g., discontinue an intravenously administered diagnostic or therapeutic agent). Simultaneously, call for help and manage the case by resuscitation team (including an anesthesiologist, obstetrician, and neonatologist). Inject epinephrine, and place the patient in left lateral tilt of at least 15° to prevent aortocaval compression with the lower extremities elevated and do not allow the patient to sit or stand suddenly because this can lead to cardiac arrest caused by the empty inferior vena cava/empty ventricle syndrome. Administer oxygen by face mask or by oropharyngeal airway at a flow rate of 6–8 L/min as obstetric patients have increased oxygen requirements and are more prone to rapid acute oxygen desaturation. Pregnant women are more prone to regurgitation and aspiration so intubate early if deemed necessary. Insert an intravenous catheter and give intravenous fluid resuscitation, and initiate cardiopulmonary resuscitation with continuous chest compressions. At frequent and regular intervals, monitor the patient's blood pressure, cardiac rate and function, respiratory status, and oxygenation and obtain electrocardiograms; start continuous noninvasive fetal monitoring. Delivery will most likely improve maternal ventilation and may improve neonatal survival depending on gestational age. Delivery may be carried out under regional anesthesia thereby avoiding the need for invasive airway management.

Epinephrine (Adrenaline)

According to the World Health Organization (2011), World Allergy Association (2010), Joint Task Force anaphylaxis update, and 2010 NIAID Guidelines, epinephrine (adrenaline) is a first-line therapy for the reversal of anaphylaxis symptoms and should be administered as soon as the diagnosis is suspected along with cardiorespiratory resuscitation. Adrenaline has an effect on both α and β adrenoceptors. Stimulation of α_1 adrenoreceptor has vasoconstrictor effects on the small arterioles, and precapillary sphincters in most body organ systems increase peripheral vascular resistance thus improving blood pressure and coronary perfusion and decreasing angioedema. Stimulation of β_1 adrenoceptors has both positive inotropic and chronotropic cardiac effects. Stimulation of β_2 receptors by triggering the rise in intracellular cyclic AMP causes bronchodilation and decreases release of histamine, tryptase, and other mediators of inflammation from mast cells and basophils thereby attenuating the severity of the reaction when given early [33–35].

The early use of epinephrine in vitro inhibits the release of platelet-activating factor (PAF) in a time-dependent manner, giving support to the use

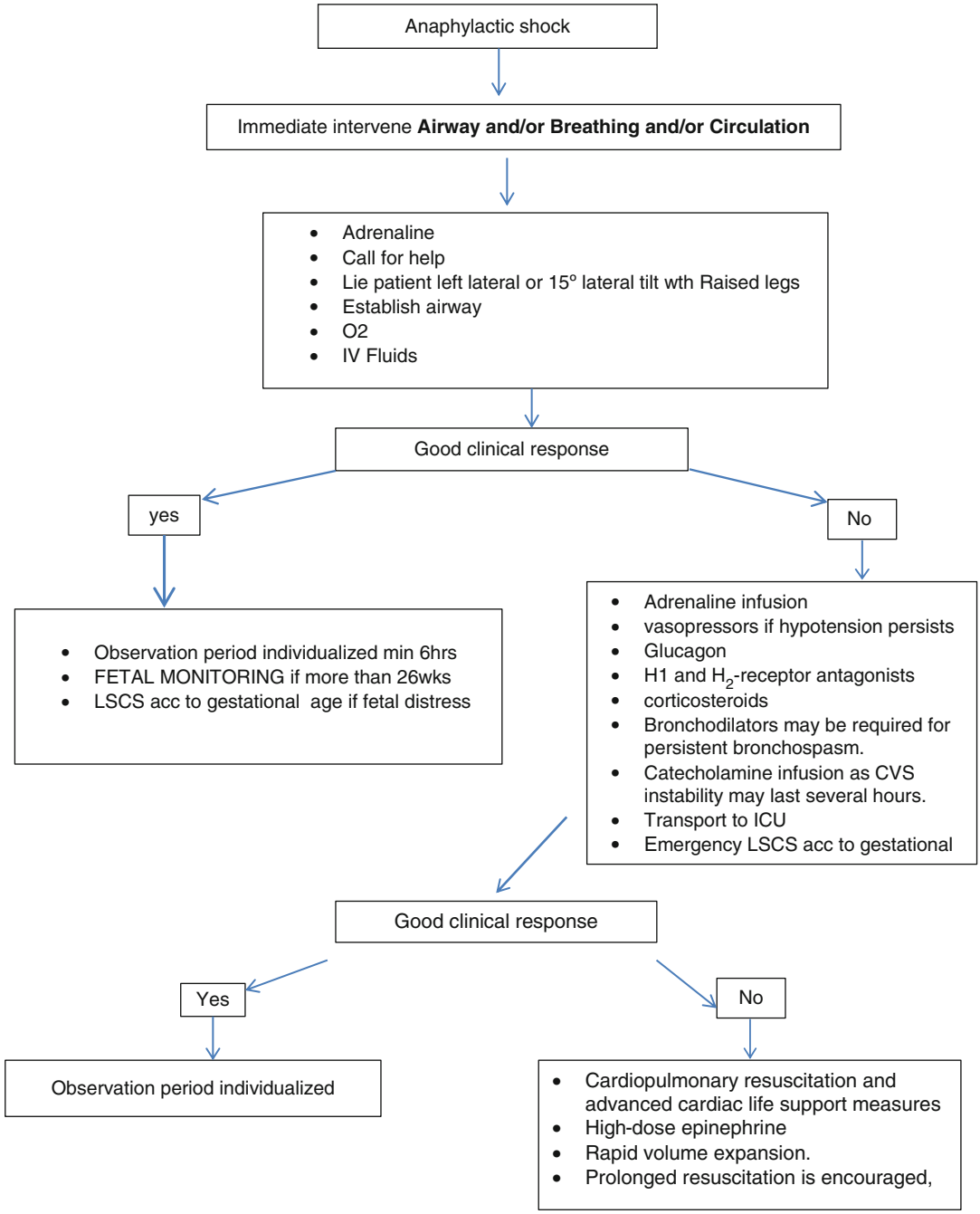

Fig. 22.1

of this medication with the first signs and symptoms of anaphylaxis.

Although epinephrine increases uterine vascular resistance through its α-adrenergic-mediated blood vessel vasoconstriction, the best hope for fetal survival is maternal survival. Therefore, epinephrine should be the treatment of choice for anaphylaxis during pregnancy, as recognized by the latest French guidelines [4] and the Eighth Report of the Confidential Enquiries into

Maternal Deaths in the United Kingdom. There is no absolute contraindication to the use of epinephrine. The lack of, or delay in, epinephrine administration accounts for the majority of deaths caused by anaphylaxis [36].

Dose and route of epinephrine Recommendations for the maximum initial dose of epinephrine or the route of injection differ from one guideline to another. The World Health Organization and the World Allergy Organization guidelines recommend that epinephrine should be administered at a dose of 0.01 mg/kg of a 1:1,000 solution (to a maximum of 0.5 mg) into the intramuscular (IM) space of the mid-anterolateral thigh due to rapid absorption as compare to IM or subcutaneous administration at other sites, because epinephrine has a vasodilator effect in skeletal muscle, which facilitates rapid absorption from IM injection. The time to highest blood concentration (Cmax) is 8 min when given IM in the lateral thigh than when given either SC (34 min) or IM in the deltoid muscle of the arm. In contrast, the powerful vasoconstrictor effect of epinephrine injection into subcutaneous tissue delays its absorption; injection can be repeated every 5–15 min as needed. Adrenaline may be injected through clothing in emergency, and if available, EpiPen can be used instead.

Racemic epinephrine via a nebulizer application is effective in the case of laryngeal edema and also in bronchospasm, but it does not replace IM administration of epinephrine. 2 ml of 1 mg/ml of adrenaline is given with oxygen through a nebulizer and respiratory mask.

For patients who do not respond to two doses of IM epinephrine and/or are experiencing profound hypotension or shock, or in whom cardiorespiratory arrest is imminent, intravenous adrenaline should be administered slowly with extreme care to avoid potentially lethal complications such as cardiac arrhythmias, myocardial ischemia, and cerebrovascular accident. Continuous infusion is safer rather than intermittent boluses. An epinephrine infusion should be started at 1 μg/min and increased 1 μg/min every 5–10 min to a maximum of 10 μg/min, or 0.1 mg of a 1:10,000 solution can be administered IV every 5 min [37]. Continue the infusion up to 60 min after the resolution of all symptoms and signs of anaphylaxis, and then wean over the next 30 min and stop, watching closely for any recurrence.

Fluid therapy: Because of increased permeability and vasodilation, there is dramatic shift of intravascular volume causing hypotension in anaphylaxis; the treatment after epinephrine is aggressive IV fluid administration. Give a rapid IV fluid challenge; 1 L of 0.9 % saline or Ringer's lactates is infused over 15 min; monitor the response; give further doses as necessary. Large volumes (exceeding 5 L) of crystalloid may be required. The exact amount should be individualized and based on blood pressure and urine output. Both crystalloid and colloid can be used, but use of crystalloid versus colloid is controversial. French guidelines recommend that colloids be used after the crystalloid dose exceeds 30 mL/kg. But use of colloids can themselves cause histamine release and can worsen any reaction. All hyperosmolar solutions can release histamine directly. The risk is greatest with gelatin solutions [37]. There is no evidence from randomized controlled trials that resuscitation with colloids reduces the risk of death, compared to resuscitation with crystalloids. As colloids are not associated with an improvement in survival and are considerably unacceptably high rate of renal side effects, homeostatic and anaphylactoid effects of colloids on coagulation and on anaphylaxis may increase the risk of death, and more expensive than crystalloids, use of colloids is not justified [38]. A Cochrane review of randomized controlled trials of crystalloids versus colloids in thousands of surgical patients requiring volume replacement found that colloid administration did not correlate with increased survival [37].

A few authors have advocated even using a negative fluid balance in obstetric patients – the rationale being the fact that mortality associated with pulmonary edema has been found to be much higher due to acute hypovolemic renal injury in critically ill pregnant patients.

Oxygenation Supplement the highest concentration of oxygen with high-flow oxygen (usually greater than 10 l/min) using a mask with an oxygen reservoir as soon as possible to maintain

adequate mentation and an oxygen saturation of at least 91 % as determined by pulse oximetry. Management of the airway with advanced airway devices may initially present a challenge because of bronchospasm, increased bronchial secretions, and swollen or inflamed respiratory anatomy.

Other Drugs

Other Vasoactive Substances

If adrenaline and volume substitution are insufficient to control symptoms, other vasoactive substances and inotropes such as noradrenaline, vasopressin, methylene blue, or glucagon are used. According to the Cochrane Database Syst Rev. 2011, no clear superiority of dopamine, dobutamine, norepinephrine, phenylephrine, or vasopressin (either added to epinephrine alone or compared with one another) has been demonstrated in clinical trials [39].

Arginine vasopressin Vasopressin, a nonapeptide, is synthesized as a large prohormone in the paraventricular and supraoptic nuclei of the hypothalamus. Vasopressin acts on V1, V2, V3, and oxytocin-type receptors (OTR). This agent is a direct systemic vasoconstrictor. It induces vasoconstriction by activating vasopressin 1 receptors, decreasing the synthesis of nitric oxide (NO) synthetase, blocking the target enzyme of the NO pathway, blunting the increase in cGPM, and directly inactivating KATP channels in vascular smooth muscle. It is synergistic with other vasopressors and stimulates cortisol secretion by increasing adrenocorticotropic hormone production and release. It is important for osmoregulation and maintenance of normovolemia. In severe anaphylactic shock, refractory to epinephrine and fluid, vasopressin adding arginine vasopressors resulted in prompt hemodynamic stabilization. Dose of vasopressin is between 0.0003 and 0.008 U/kg/min [22, 40–42].

Glucagon IV is reported to have been successful for refractory hypotension and may reverse refractory bronchospasm and hypotension in patients on beta-blockers by activating adenyl cyclase directly and bypassing the beta-adrenergic receptor. It has direct inotropic and chronotropic effects and might restore hemodynamic stability. It is given as 1–5 mg intravenously, followed by an infusion at 5–15 μg/min titrated to response. Protection of the airway is important since glucagon may cause emesis/aspiration in drowsy or obtunded patients.

Dopamine Dopamine is a potent α and β adrenoreceptors agonist and has a short half-life. Dopamine in low doses via vascular D1 dopaminergic receptors leads to renal, mesenteric, and coronary vasodilatation. Dopamine (400 mg in 500 mL of 5 % dextrose) can be infused at 2–20 mg/kg/min and titrated to maintain systolic blood pressure of greater than 90 mmHg.

Norepinephrine Noradrenaline is a potent α and β1 adrenoreceptor agonist and weak β2 adrenoreceptor agonist; hence, the bronchodilatory effect is less. Therefore, there is little effect on the lung. The usual dose is 0.02–0.15 μg/kg/min.

Methylene blue Histamine is the major anaphylaxis mediator. Inducing nitric oxide (NO) production, it consequently increases guanylate cyclase, which promotes arteriolar vasodilation by increasing cyclic GMP. Methylene blue blocks accumulation of cyclic GMP by competitively inhibiting the enzyme guanylate cyclase. This results in reduced responsiveness of the vasculature to cyclic guanosine monophosphate-mediated vasodilators, such as nitric oxide. Cases have been reported that intravenous methylene blue is safe and the most effective treatment to prevent cerebral ischemia and could be used in anaphylactic shock refractory to epinephrine [43–45].

Additional Therapy

H1 and/or H2 receptor antagonists: Antihistamines do not inhibit histamine release but compete with histamine at the receptor sites. Serum histamine levels peak early in anaphylaxis and quickly return to baseline despite the persistence of severe physi-

cal compromise. Antihistamines have a slower onset of action and hence cannot block events that occur after acute histamine receptor binding and hence should never be given in lieu of epinephrine. H1 and H2 antihistamines in combination are more effective in palliating the cutaneous manifestations [46]. Diphenhydramine (1–2 mg/kg per dose IV; slow infusion; maximum dose 50 mg) and ranitidine (1–2 mg/kg per dose IV, IM; maximum dose 75–150 mg; if given IV, infuse over 10–15 min) are an appropriate combination. Resuscitation Council UK Guideline still recommends chlorphenamine 10–20 mg intramuscularly or slowly intravenously after initial resuscitation to counter histamine-mediated vasodilation and bronchoconstriction.

Corticosteroids

Steroids are unlikely to be helpful in the treatment of acute anaphylaxis as they have a delayed onset of action (4–6 h), but it may decrease airway swelling and prevent protracted anaphylaxis and they have not been shown to decrease the incidence of biphasic symptoms. A Cochrane review does not recommend the use of glucocorticoids in the treatment of anaphylaxis [47]. Corticosteroids decrease arachidonic acid metabolites by inducing synthesis of nuclear regulatory proteins to inhibit phospholipid membrane breakdown and alter the activation and migration of other inflammatory cells (i.e., polymorphonuclear leukocytes). After initial resuscitation when refractory bronchospasm or refractory shocks persist, corticosteroids should be administered. Although the exact dose and preparation are unclear, inject hydrocortisone slowly intravenously or intramuscularly, taking care to avoid inducing further hypotension. NICE Guidelines and the World Allergy Organization recommend 1.5–3 mg/kg IV (200 mg IM or IV slowly) of hydrocortisone in IgE-mediated reactions. Alternately, 1–2 g of methylprednisolone (30–35 mg/kg) may be useful in catastrophic pulmonary vasoconstriction.

Bronchodilators Bronchodilator therapy with salbutamol (inhaled or IV), ipratropium (inhaled), and aminophylline (IV) may be administered in patients with persistent respiratory distress or wheezing (NICE Guidelines).

Atropine (0.02 mg/kg) can also be used in the unstable patient with hypotension and bradycardia.

During cardiac arrest Key interventions are basic life support/advanced cardiovascular life support therapy. Death from anaphylaxis may be associated with profound vasodilation, intravascular collapse, tissue hypoxia, and asystole. Difficulties in achieving adequate volume replacement and ventilation are frequent. Because of angioedema and upper or lower airway obstruction, bag-mask ventilation and tracheal intubation may fail. Consider fiber-optic tracheal intubation and cricothyrotomy. Support of circulation requires rapid volume resuscitation (typically 2–4 L of isotonic crystalloid), epinephrine IV, and vasopressors. Steroid is of value in the postresuscitation period. Administer atropine in the asystole. Effective CPR may maintain sufficient oxygen delivery until the catastrophic effects of the anaphylactic reaction resolve.

Obstetrical Management

Controversies exist regarding the best timing and mode of delivery following anaphylaxis during pregnancy. Fetal distress should be treated by prompt aggressive maternal medical management. If fetal distress is not relieved or there is inadequate maternal resuscitation, an emergency cesarean delivery should be done for better neonate outcome. There are potential risks of surgery in a gravida with hypoxemia, hypotension, or both, and the potential risk of neonatal morbidity and mortality caused by prematurity, especially if gestational age, is less than 32 weeks [48, 49].

Monitoring

Assess airway, breathing, and circulation (ABC) initially and reassess regularly. Early warning system (EWS) itself is a simple and easy to use

tool at the bedside, which may be of help in recognizing patients with potential for acute deterioration. Different MEWS systems are in use worldwide [50] (Table 22.6).

During pregnancy in addition to frequent or continuous monitoring of maternal oxygenation, blood pressure, and cardiac rate and function, regular fetal heart monitoring (continuous electronic monitoring, if possible) is recommended for patients who are more than 24 weeks pregnant. Fetal distress should be relieved by correcting maternal hypoxia and/or hypotension with appropriate medical management; however, if the distress persists, emergency cesarean section should be considered.

Institute the following:

1. Use bag-mask ventilation; if difficult or impossible, consider early tracheal intubation (if equipment and expertise are available). If the patient is intubated, give high-concentration oxygen with a self-inflating bag. Occasionally emergency tracheotomy is required, especially if there is inspiratory stridor.
2. Pulse oximetry – SpO_2; maintain the PaO_2 as close to normal as possible (approximately 13 kPa or 100 mmHg) or 94–98 %. Minimum above 8 kPa (60 mmHg) or 90–92 % oxygen saturation.
3. Capnograph – $EtCO_2$; a normal SpO_2 on oxygen does not necessarily mean ventilation is adequate (because the pulse oximeter detects oxygenation and not hypercapnia). The patient may be breathing inadequately (with a high $PaCO_2$).
4. ECG – see findings of rate, rhythm, ischemia, conduction, etc.
5. BP (intra-arterial) – measure accurate real-time BP.
6. CVP – CVP is performed, if required, to guide fluid therapy; administer inotropes.
7. Noninvasive or minimally invasive techniques (transesophageal echocardiography, Doppler ultrasound, transthoracic bioimpedance, and arterial pulse waveform analysis) for cardiac output monitoring.
8. Nasogastric tube.
9. Urinary catheter to monitor hourly output.

Duration of Monitoring

Because no standard exists, current guidelines recommend that the duration of monitoring be individualized. According to NICE Guidelines, patients who have had an airway, breathing, or circulation (ABC) problem should be observed for at least 6 h. Patients with a good response to initial treatment should be warned of the possibility of an early recurrence of symptoms and in some circumstances should be kept under observation for up to 24 h.

Investigations

Anaphylaxis remains a largely clinical diagnosis and is valid even if the results of laboratory tests are within normal limit.

General Investigations

General investigations are carried out, appropriate for a medical emergency, e.g., 12-lead ECG, chest X-ray, urea and electrolytes, arterial blood gases, etc.

Specific Investigations

Laboratory studies are not usually required: rarely helpful and must not delay initial resuscitation. There are more than 100 biomarkers of mast cell and basophil activation; histamine and mast cell tryptase are the only ones measured.

Mast cell tryptase assessment Serum mast cell tryptase is the principal protein content of mast cell granules and is released, together with histamine and other amines, in anaphylactic and anaphylactoid reactions. Its levels peak 60–90 min and may persist for as long as 5 h. This is an insensitive biomarker for anaphylaxis, but serial measurements (e.g., on arrival, 1 h later and before discharge) may improve sensitivity and specificity. A negative test does not completely exclude anaphylaxis [51].

Plasma histamine assessment Histamine level rises within 10 min of onset but fall again within 30 min. Histamine and its metabolite, N-methylhistamine, can also be measured in a 24 h urine but is not generally available.

Other recent biomarkers It includes platelet-activating factor (PAF), bradykinin, chymase, mast cell carboxypeptidase A3, dipeptidyl peptidase I, IL-33, and other cytokines, leukotrienes, and prostaglandins. Low levels of the PAF acetylhydrolase have been reported in fatal anaphylaxis, and failure of this enzyme to inactivate PAF may help identify individuals at risk of severe or even fatal anaphylaxis [12].

Postmortem findings In many cases of fatal anaphylaxis, no specific macroscopic findings are present at postmortem examination. Signs may show an "empty heart" attributed to reduced venous return from vasodilation; laryngeal edema; eosinophilia in the lungs, heart, and tissues; and evidence of myocardial hypoperfusion Laboratory findings could detect increased levels of serum tryptase and increased total and specific IgE serum levels. In the presence of a typical clinical history, absence of postmortem findings does not exclude the diagnosis of anaphylaxis.

Summary: Anaphylactic Shock – No Time to Think

1. Anaphylaxis is a rare event in pregnancy.
2. Anaphylaxis is a serious, rapid-onset, allergic reaction that may cause death. Severe anaphylaxis is characterized by life-threatening hypotension, upper airway obstruction, and/or bronchospasm.
3. Diagnosis can be difficult, with skin features being absent in up to 20 % of people. Anaphylaxis must be considered as a differential diagnosis for any acute-onset respiratory distress, bronchospasm, hypotension, or cardiac arrest.
4. High degree of suspicion is essential for prompt diagnosis and treatment.
5. Immediate management:
    1. Immediately call for help.
    2. Position the patient in left lateral or 15° lateral tilt to relieve aortocaval compression with leg elevated.
    3. Establish and maintain an open airway.
    4. Aggressive recognition and prompt treatment with epinephrine, volume replacement, and oxygen remain critical to ensuring good patient outcome.
    5. Planning for a cesarean delivery is necessary when the diagnosis of anaphylaxis has been made, as it may lead to cardiac arrest.
6. Subsequent management:
    1. If after two doses of IM adrenaline and initial management hemodynamic instability persists, a continuous drip, IV adrenaline is started.
    2. Diphenhydramine and ranitidine are second-line therapy to epinephrine.
    3. Inhaled beta-agonist is given for bronchospasm.
    4. Vasopressors and glucagon can be given if hypotension persists.
    5. Cardiopulmonary resuscitation and advanced cardiac life support is necessary for cardiopulmonary arrest.
    6. Atropineis is given if it is asystole.

Table 22.6 Early warning systems (EWS)

Score	3	2	1	0	1	2	3
Systolic BP	<45 %	30 %	15 % down	Normal for patient	15 % up	30 %	>45 %
Heart rate (BPM)	–	<40	41–50	51–100	101–110	111–129	>130
Respiratory rate (RPM)	–	<9	–	9–14	15–20	21–29	>30
Temperature (°C)	–	<35	–	35.0–38.4	–	>38.5	–
AVPU	–	–	–	Alert	Voice	Pain	Unresponsive

7. Check ABGs for acidosis and consider bicarbonate 0.5–1.0 mmol/kg (8.4 % solution = 1 mmol ml).
8. Patient is admitted in the intensive care unit for prolonged monitoring.
9. Follow-up is individualized.

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Sudden Obstetric Collapse

23

Lisa M. Nathan and Asha Rijhsinghani

Introduction

In 1990, there were 523,000 maternal deaths worldwide. This decreased to 289,000 in 2013 [1]. Fifty-eight percent of the maternal deaths occurred in ten countries – India, Nigeria, Pakistan, Afghanistan, Ethiopia, Democratic Republic of the Congo, United Republic of Tanzania, Kenya, China, and Uganda. Among these ten countries, India and Nigeria account for one-third of all global maternal deaths, with India having the highest number of maternal deaths. Every year in India, about 50,000 maternal deaths occur which translates into almost 137 maternal deaths per day. Maternal illness also leads to an increase in preterm delivery and perinatal mortality of 25 %.

In 2000, the United Nations (UN) held the Millennium Summit, where eight goals were set to address the world's main development challenges. In view of the fact that maternal mortality is both a health and development issue, included in these goals was the Millennium Development Goal (MDG) 5, to decrease the maternal mortality ratio by three-quarters by 2015. Due to efforts to achieve MDG5, India has seen a decline in maternal mortality at a yearly rate of 4.0 %. The decrease is attributed to the increase in skilled birth attendance [2]. However, with the increasing number of women choosing institutional delivery, there is fear of overcrowding of the healthcare facilities. Concerns have been raised that care is compromised and may increase rates of maternal near misses [3].

As recently as 2007, a study of anesthesiologists, obstetricians, and emergency room physicians revealed that the physicians most likely to care for the sick pregnant women lacked adequate basic knowledge of resuscitation of the pregnant patient [4]. This has been confirmed in subsequent studies [5]. Early recognition of deteriorating maternal status and knowledge of advanced cardiac life support (ACLS) will prevent maternal deaths, reducing maternal mortality rates.

Near Miss/SAMM

The near miss is defined as a patient with an acute organ dysfunction which if untreated in a timely fashion could lead to maternal death. It has also been described as severe acute maternal morbidity (SAMM) which is described as "a very ill

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pregnant or recently delivered woman who would have died had it not been but luck and good care on her side" [6]. In 2009, the WHO defined SAMM as "a woman who nearly died but survived a complication that occurred during pregnancy, childbirth or within 42 days of termination of pregnancy" [7]. The classification aimed to standardize the quality of maternal care, to help identify the causes of the near-miss cases, to make reliable comparisons between countries, and to help develop individualized national programs based on the shortfalls identified in the countries.

The rate of near-miss cases is said to be five times that of maternal mortality [8, 9]. However, in India, the maternal death rate is higher. This difference has been attributed to poor management of obstetrical emergencies at the referring hospitals as well as ineffective and inefficient referral mechanisms [10]. In a 2000–2002 report from the UK, substandard care was suspected to have been the reason for 46 % of maternal deaths from preeclampsia and eclampsia that may have been avoidable [11]. Increased maternal mortality rates in patients with severe preeclampsia have been attributed to delays in treatment of the severe hypertension and delays in diagnosis and management of pulmonary edema. Other causes of possible unavoidable deaths are undiagnosed cardiac defects such as pulmonary hypertension that has a high maternal mortality rate, especially in the postpartum period, venous thromboembolism during pregnancy and especially in the postpartum period, and unrecognized pulmonary embolism with a delay in initiating anticoagulants [12].

In a retrospective study of peripartum women admitted to an intensive care unit (ICU) in Mumbai, the maternal mortality rate was reported to be high at 21.6 %. In addition to lack of prenatal care, the high maternal mortality was attributed to a delay in referring the patient to the ICU [13]. A delay of over 24 h between onset of the illness and transfer of the patient to a higher level of care significantly correlated with maternal mortality [13]. Pregnant patients that experience sudden collapse would then fall into this category.

A healthy mother may develop acute conditions and quickly deteriorate to major organ system failure and subsequent maternal death. The progression of the disease results in systemic inflammatory response syndrome (SIRS) with subsequent organ involvement, organ failure, and ultimately death. When patients in such a life-threatening state receive timely interventions that ultimately lead to survival, it is defined as near miss. The number of organ systems involved also impacts the maternal mortality, with the mortality rising above 70 % in patients when three organ systems are affected and >80 % when four organ systems are affected. Early detection of the maternal condition and prompt referral to a tertiary care center is recommended to reduce maternal mortality [13].

Organs Involved in Maternal Collapse

Involvement of the respiratory system, the cardiovascular system (CVS), and the central nervous system (CNS) has the strongest association with poor maternal and fetal/neonatal outcome. Furthermore, the higher the number of organs involved, the greater the maternal and fetal risks. Viral hepatitis and disseminated intravascular coagulation (DIC) are independently associated with higher maternal mortality.

Conditions Associated with Near Miss

In a pregnant patient, unexplained tachypnea or tachycardia may be the first signs of impending collapse, and these may easily be overlooked. Conditions associated with near miss vary by country. Hemorrhage is the condition most common associated with near misses worldwide and it accounts for a third of cases in Asian countries alone [14]. In developing countries as a whole, the conditions are many, including hemorrhage, anemia, sepsis, obstructed labor, hypertensive deaths, abortion, embolism, ectopic pregnancy, and other direct and indirect causes of

deaths. Other less common causes include amniotic fluid embolism and cardiac arrest.

Specific conditions associated with hemorrhage include uterine atony, massive postpartum hemorrhage (PPH), placenta accreta/percreta, and subsequent oliguria. In these cases, maternal deaths are attributed to lack of early recognition of the amount of blood lost, lack of hemorrhage protocols, delay in active management, unjustified administration of diuretics to women with oliguria secondary to PPH, delay in performing hysterectomy in cases of massive PPH, and lack of recognition of placenta accreta/percreta. Inadequate and delayed management of massive hemorrhage in the postpartum period has been reported to cause myocardial damage in up to 50 % of patients, even in the young parturient [15].

Physiology and Causes of Maternal Collapse and Cardiac Arrest

Physiology

Understanding the physiologic changes during pregnancy is essential in evaluating the critically ill patient. Early in pregnancy increases occur in maternal blood volume, cardiac output, respiratory rate, and oxygen consumption. These changes are essential in maintaining proper perfusion and functioning of the maternal and fetal organs. As the gestation advances, there is a decrease in the residual lung capacity, venous return, and laryngeal space. These changes reduce the organ systems' reserve, increasing the risks of acute and rapid decompensation in the event of systemic challenges. The CNS, CVS, and respiratory systems are most commonly affected.

Physiologic factor	Change
Blood volume	Increased
Cardiac output	Increased
Respiratory rate	Increased
Oxygen consumption	Increased
Blood pressure	Decreased
Residual lung capacity	Decreased
Laryngeal space	Decreased
Venous return	Decreased

Causes of Maternal Collapse

The more common conditions that start the cascade of generalized decompensation, maternal collapse, and cardiac arrest include acute massive hemorrhage that may or may not be recognized immediately, severe sepsis, and pregnancy-induced hypertension. These complications may occur in the antepartum or postpartum period. Other less common causes include pulmonary embolism, amniotic fluid embolism, intracranial hemorrhage, cardiac arrest, magnesium toxicity, trauma, and anesthetic complications. Even when the serum levels are low, magnesium toxicity can occur and the effects can range from AV nodal conduction blocks to bradycardia and cardiac arrest.

Hemorrhage

Antepartum hemorrhage can result from placenta previa (with or without an accreta/percreta), abruptio placenta, and uterine rupture. In addition, incomplete abortion or spontaneous abortion in the second trimester with subsequent retained placenta can cause significant hemorrhage. In the postpartum period, the most common cause is uterine atony. Patients may present with acute anemia and hypotension requiring immediate blood transfusion, or in some cases an emergency hysterectomy may be indicated.

Common Signs/Symptoms of Hemorrhage

1. Fast, weak pulse
2. Low blood pressure
3. Pallor
4. Sweatiness, cold and clammy skin
5. Low urine output
6. Rapid breathing
7. Anxiousness, confusion

Sepsis

Sepsis is a major cause of maternal collapse, and it may occur in the antepartum or the postpartum

Common Findings/Characteristics of Sepsis in Pregnancy

1. Temperature instability (fever, but may be afebrile)
2. Hypotension
3. Tachycardic
4. Tachypneic
5. Altered mental status
6. Oliguria
7. Low WBCs
8. Rapid progression
9. Common microorganisms
   (a) Streptococcus A, B, and D
   (b) Pneumococci
   (c) *E. coli*

Septic Shock Management

1. Get blood cultures.
2. Place two large-bore IVs, one in each arm.
3. Start broad-spectrum antibiotics (gentamicin, clindamycin, and ampicillin (or penicillin)).
4. Provide oxygen supplementation.
5. Check arterial blood gas.
6. Check serum lactate level.
7. Check CBC and transfuse if hemoglobin <7 g/dL.
8. Monitor urine output.
9. Fluid resuscitation with IV crystalloids.
10. Pressors if systolic BP <65 mmHg.
11. Evaluate for cause and treat if possible.

period. Patients with severe sepsis during the antepartum period may present with acute hypotension due to shock and DIC [16].

Pregnancy-Induced Hypertension

Hypertensive disorders in pregnancy are very common. An important early distinction to be made is whether the hypertension preceded the pregnancy or not. Preeclampsia, eclampsia, and HELLP are pregnancy-specific syndromes that can be deadly. In cases of severe HTN, preeclampsia, and/or HELLP, eclampsia, intracranial hemorrhage, and liver rupture may lead to maternal collapse related to either brain involvement or acute blood loss and hypotension. Pulmonary edema is another cause of maternal mortality in this setting and is due to alveolar-capillary compromise. Early initiation of oxygen and IV furosemide can be life-saving. Diagnosing and treating the primary condition, which may be cardiac, overhydration, or pregnancy-induced hypertension, is of primary importance.

Anesthetic complications Local anesthetic agents can cause systemic toxicity which is termed LAST. Though rare, injection of local anesthetic agents can lead to CNS or cardiac toxicity that can result in death. When these occur, it is often due to inadvertent intravascular injection of the anesthetic agent [17].

Other obstetric and non-obstetric causes of maternal collapse should also be considered. These include: stroke which is often arterial. Cerebral venous thrombosis due to: dehydration, sepsis, trauma, sickle cell disease, thrombophilia, and metastatic choriocarcinoma.

Non-obstetric Causes of Maternal Collapse

1. Medications: MgSO4
2. Illicit drugs and toxins
3. Hypoglycemia
4. Vasovagal reaction – low pulse, low BP

Very Rare Causes of Maternal Collapse

1. Marphan syndrome: aortic aneurysm rupture
2. Hemoperitoneum due to: spleen rupture, liver rupture

Basics of Cardiopulmonary Resuscitation (CPR)

Since the basic knowledge of providing cardiopulmonary resuscitation to the pregnant patient is lacking among the obstetricians, anesthesiologists, and the emergency room physicians, training every doctor in ACLS will reduce maternal mortality by up to 50 %.

In 2010 the American Heart Association published the guidelines for return of spontaneous circulation (ROSC) and ACLS based on 440 publications [18]. The guidelines included specific instructions for resuscitating the pregnant patient. The entire details of CPR are beyond the scope of this chapter and can be found in reference [18].

The guidelines include but are not limited to:

In cardiac arrest cases, immediately shout for help. In an antepartum patient, it is imperative to displace the uterus laterally either manually with two-hand technique or by placing the patient in a left lateral tilt by 30° and placing a firm wedge to support the pelvis and the thorax [19].

Start CPR immediately with chest compressions and ventilation. The chest compressions will be more cephalad in the antepartum patient. Without the presence of advanced airway, the ratio of chest compressions to rescue breaths is 30:2, with the rate of the chest compressions being 100/min. The chest compressions should have a depth of at least 2 in., allowing complete chest recoil. In presence of advanced respiratory airway such as an endotracheal tube, the chest compressions should be continuous at a rate of 100/min and the ventilation should be 8–10/min. Tidal volume recommended is about 600 mL, and chest rise will be seen for a second. Routine use of cricoid pressure is not recommended since it can impede ventilation. In cases of ventricular fibrillation or ventricular tachycardia, the initial CPR should be followed by early defibrillation. Epinephrine may be used if initial 2 min of CPR does not result in return of normal cardiac rate and rhythm. In cases of VF/VT that does not respond to CPR or epinephrine, amiodarone may be used.

In the third trimester, due to aortocaval compression by the gravid uterus, CPR may fail. Data on such cases is very limited. Based on the limited data that may include biases, in such cases a perimortem cesarean delivery initiated within 4 min of arrest has been associated with higher maternal survival due to more effective CPR with decompression of the uterus and return in blood pressure and pulse [20]. In addition, it has been found that maternal brain injury will occur within 6 min after cessation of blood flow to the brain.

Emergency Management CBA (Not ABC)

Call for HELP. Tilt the patient left lateral 30° for better resuscitation.

1. Check the circulation.
   Carotid pulse and blood pressure (capillary filling)
2. Check for breathing.
   Check for cyanosis.
3. Check airway.
   Response to verbal command and stimulation

Carotid Pulse +, But No Breathing

1. O2, mask, intubation

No Carotid Pulse, Start Resuscitation

1. Chest compressions/breaths (30/2)
2. Pulse O2, blood gas
3. EKG ASAP: arrhythmias or asystole

Conclusion

Every unit that cares for pregnant patients must prepare for the unpredictable catastrophic events by conducting mock procedures and drills. Armed with the knowledge of conditions associated with increased maternal morbidity and mortality, timely identification of these conditions and prompt care of the patient should help reduce maternal loss. It is important to consider the following, in every unit that cares for pregnant patients.

1. Organization of the rapid response team.
2. Anesthesiology consult in the antenatal period, for all cases deemed as high risk.
3. Provide members of the rapid response team with preassigned duties: OB doctors, anesthesiologist, ICU staff, blood bank, nurses, OT staff, ambulance, radiologist, secretary, pharmacy, and pediatrician.
4. Establish protocols.
5. Establish clear communication channels.
6. Anticipation – recognize risk factors.
7. Early recognition of the critical clinical situation.
8. Stabilization.
9. Immediate transfer after stabilization.
10. Timely ICU admission, within <24 h of admission.
11. Drills, drills, drills.

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24 Disseminated Intravascular Coagulation (DIC) and Thrombocytopenia in Pregnancy

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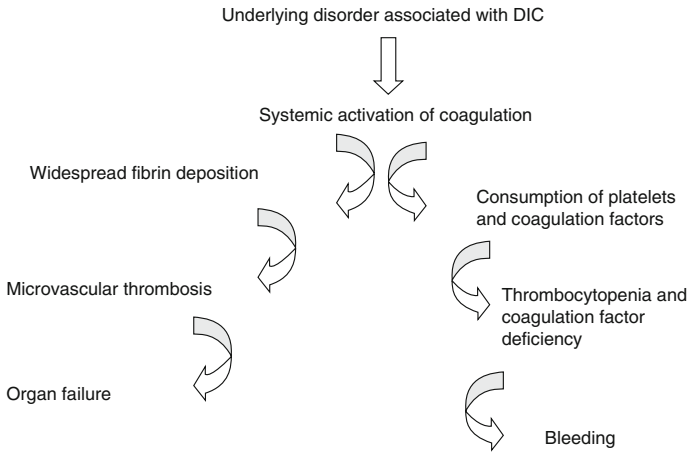
DIC Definition [1]

Systemic thrombohemorrhagic disorder seen in association with well-defined clinical situations and laboratory evidence of:

- Procoagulant activation
- Fibrinolytic activation
- Inhibitor consumption
- Biochemical evidence of end-organ damage or failure

Processes in DIC [2]



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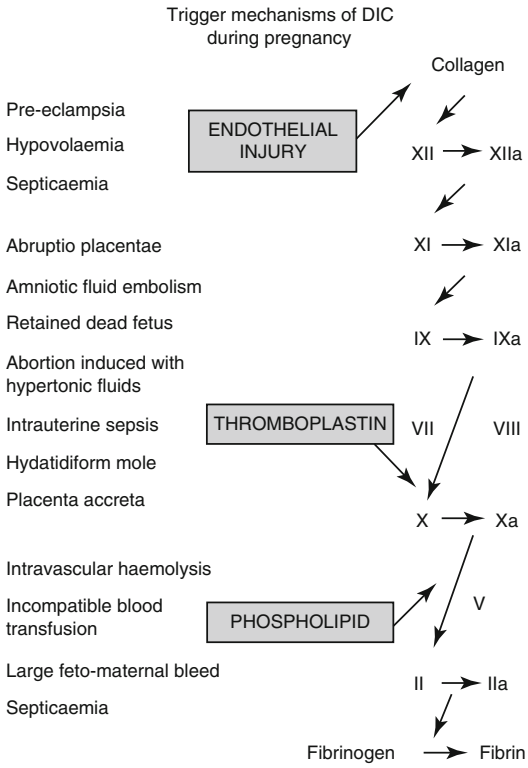
Pathogenesis of DIC in Pregnancy [3]



Three main triggers:
- Endothelial injury
- Thromboplastin release
- Phospholipid exposure

End result = generation of thrombin with ↑fibrin deposition

Causes of DIC in Pregnancy

- Abruptio placentae
- PPH
- Preeclampsia
- AFLP
- Sepsis
- Amniotic fluid embolism
- Saline-induced therapeutic abortion
- Retained dead fetus or products of conception

Diagnosis of Obstetrical DIC [4]

DIC is suspected in patients with unexplained bleeding or venous thromboembolism, especially if a predisposing condition exists. If DIC is suspected, platelet count, PT, PTT, plasma fibrinogen level, and plasma D -dimer level (an indication of in vivo fibrin deposition and degradation) are obtained.

Slowly evolving DIC produces mild thrombocytopenia, a normal to minimally prolonged PT (results are typically reported as INR) and PTT, a normal or moderately reduced fibrinogen level, and an increased plasma D -dimer level. Because various disorders stimulate increased synthesis of fibrinogen as an acute-phase reactant, a declining fibrinogen level on two consecutive measurements can help make the diagnosis of DIC. Initial PTT values in slowly evolving DIC may actually be shorter than normal, probably because of the presence of activated coagulation factors in the plasma.

Severe, rapidly evolving DIC results in more severe thrombocytopenia, more prolonged PT and PTT, a rapidly declining plasma fibrinogen level, and a high plasma D -dimer level.

A factor VIII level can sometimes be helpful if severe, acute DIC must be differentiated from massive hepatic necrosis, which can cause similar abnormalities in coagulation studies. The factor VIII level is elevated in hepatic necrosis because factor VIII is made in hepatocytes and released as they are destroyed; factor VIII is reduced in DIC because of the thrombin-induced generation of activated protein C, which proteolyses the activated form of factor VIII.

Spectrum of DIC in Obstetrics [3]

Severity of DIC	In vitro findings	Obstetric conditions commonly associated
Stage 1: low-grade compensated	↑FDPs	Preeclampsia and related syndromes
	↓Platelets	
Stage 2: uncompensated but no hemostatic failure	As above plus: ↓↓Platelets	Small abruptio
	Fibrinogen	Severe preeclampsia
	Factors V and VIII	
Stage 3: rampant with hemostatic failure	As above plus:	Abruptio placentae
	↓↓Platelets	Amniotic fluid embolism
	Gross depletion of coagulation factors (particularly fibrinogen)	Eclampsia
	↑↑FDPs	

Rapid progression may occur if underlying cause not treated

Treatment

- Treatment of cause
- Possibly replacement therapy (e.g., platelets, cryoprecipitate, fresh frozen plasma, natural anticoagulants)
- Sometimes heparin
- Treat acidosis, hypothermia, and hypocalcemia

Immediate correction of the cause is the priority (e.g., broad-spectrum antibiotic treatment of suspected gram-negative sepsis, evacuation of the uterus in abruptio placentae). If treatment is effective, DIC should subside quickly. If bleeding is severe, adjunctive replacement therapy is indicated, consisting of platelet concentrates to correct thrombocytopenia, cryoprecipitate to replace fibrinogen and factor VIII, and fresh frozen plasma to increase levels of other clotting factors and natural anticoagulants (antithrombin, proteins C, S, and Z). The effectiveness of infusion of concentrates of antithrombin in severe, rapidly evolving DIC is unresolved.

Heparin is useful in the treatment of slowly evolving DIC with venous thrombosis or pulmonary embolism [5]. Heparin usually is not indicated in rapidly evolving DIC with bleeding or bleeding risk, except in women with a retained dead fetus and evolving DIC with a progressive decrease in platelets, fibrinogen, and coagulation factors. In these patients, Heparin is administered for several days to control DIC, increase fibrinogen and platelet levels, and decrease excessive coagulation factor consumption. Heparin is then stopped and the uterus evacuated.

Key Points

- In DIC, the coagulation cascade is activated when blood is exposed to tissue factor.
- DIC usually begins rapidly and causes bleeding and microvascular occlusion, leading to organ failure.
- DIC sometimes begins slowly and causes thromboembolic phenomena rather than bleeding.
- Severe, rapid-onset DIC causes severe thrombocytopenia, very prolonged PT and PTT, a rapidly declining plasma fibrinogen level, and a high plasma D-dimer level.
- Immediate correction of the cause is the priority; severe bleeding may also require replacement therapy with platelet concentrate, cryoprecipitate, and fresh frozen plasma.
- Heparin is useful in slow-onset DIC but rarely in rapid onset (mainly in women with a retained dead fetus).

Thrombocytopenia in Pregnancy

Definition

The normal range of platelets in nonpregnant women is 150,000–400,000/μL

Average platelet count in pregnancy is decreased (213,000/μL vs 250,000/μL).

Change in platelet count is due to hemodilution, increased platelet consumption, and increased platelet aggregation driven by increased levels of thomboxane A_2.

Thrombocytopenia can be defined as platelet count less than 150,000/μL or platelet count below the 2.5th percentile for pregnant patients (116,000/μL).

Classification

Classification of thrombocytopenia in pregnancy is arbitrary and not necessarily clinically relevant.

Mild thrombocytopenia is 100,000–150,000/μL. Moderate thrombocytopenia is 50,000–100,000/μL. Severe thrombocytopenia is <50,000/μL.

In normal pregnancies, 7.6 % of women present with mild thrombocytopenia during pregnancy, and 65 % of them will not be associated with any pathology.

Any pregnant patient with a platelet count of less than 100,000/μL should undergo further clinical and laboratory assessment.

Manifestations

Clinical assessment is the most important factor for the evaluation of a pregnant patient with thrombocytopenia.

Medical history may include the following:

- Current or previous bleeding problems
- Family history of bleeding
- Alcohol or substance abuse history
- Past obstetrical history
- Transfusion history

Examination findings suggestive of thrombocytopenia include the following:

- Petechiae, ecchymoses, and nose and gum bleeding
- Rare – hematuria, gastrointestinal bleeding, intracranial bleeding

Bleeding associated with surgery is uncommon unless the platelet counts are lower than 50,000/μL.

Clinically significant spontaneous bleeding is rare unless counts fall below 10,000/μL.

Etiologic Classification

The etiologic classification for thrombocytopenia can be divided into three broad categories:

1. Increased destruction
2. Decreased production
3. Sequestration

Platelet destruction is more common in the obstetric practice.

Classification of Thrombocytopenia in Pregnancy

Increased platelet destruction involves gestational thrombocytopenia and immunologic-related thrombocytopenia, including the following:

- Immune thrombocytopenic purpura (ITP)
- Systemic lupus erythematosus (SLE)
- Antiphospholipid syndrome
- Connective tissue disorders
- Drug induced
- HIV related
- Viral infections (e.g., Epstein-Barr virus)
- Lymphoma

Nonimmunologic-related thrombocytopenia may include the following:

- Preeclampsia/eclampsia
- HELLP syndrome

- Thrombotic thrombocytopenic purpura
- Hemolytic uremic syndrome
- Acute fatty liver of pregnancy
- Heparin-induced thrombocytopenia
- Vascular malformations
- Hypersplenism

Decreased platelet production may also be noted and includes vitamin B12 and folate deficiency, as well as bone marrow suppression that can be caused by the following:

- Drug induced
- Aplastic anemia
- Paroxysmal nocturnal hemoglobinuria
- Infection
- Bone marrow infiltration (hematologic malignancy, nonhematologic malignancy)

Splenic sequestration may be caused by the following:

- Portal hypertension
- Liver disease
- Portal or hepatic vein thrombosis
- Myeloproliferative disorders
- Lymphoproliferative disorders
- Storage disease (e.g., Gaucher disease)
- Infection (e.g., malaria)

The most common causes of thrombocytopenia in pregnancy are as follows:

- Gestational thrombocytopenia (70 %)
- Preeclampsia (21 %)
- Immune thrombocytopenic purpura (3 %)
- Others (6 %)

Gestational Thrombocytopenia

Incidence

The incidence of gestational thrombocytopenia is 8 % of all pregnancies and accounts for more than 70 % of cases of thrombocytopenia in pregnancy.

Pathophysiology

The pathophysiology of gestational thrombocytopenia is unknown, but two main factors are associated with GT.

1. Accelerated platelet activation is suspected to occur at placental circulation.
2. Accelerated consumption of platelets is due to the reduced lifespan of platelets during pregnancy.

Diagnosis

The following may be noted:

- Asymptomatic patient with no history of abnormal bleeding
- Mild thrombocytopenia (counts >70,000/μL)
- Usually detected incidentally on routine prenatal screening
- No specific diagnostic tests to definitively distinguish gestational thrombocytopenia from mild ITP
- Usually develops in the third trimester

Clinical Manifestations

These include the following:

- No prepregnancy history of low platelets or abnormal bleeding is noted.
- Platelet counts normalize within 2–12 weeks following delivery.
- Burrows reported that all women with GT had normal or normalizing platelet counts by the seventh postpartum day [6].

Fetal/Neonatal Risks

No pathological significance for the mother or fetus is noted. No risk for fetal hemorrhage or bleeding complications is observed [7].

Management Considerations

Preconceptional

Gestational thrombocytopenia can recur. Risk of recurrence is unknown.

Antepartum

Monitor platelet count periodically. No treatment is necessary for gestational thrombocytopenia. Invasive approaches to fetal monitoring (fetal blood sampling) are not indicated.

Labor and Delivery

Mode of delivery is determined by obstetric/maternal indications. Epidural anesthesia is considered safe when platelet count is >50,000/μL. Antepartum anesthesia consultation should be obtained to discuss availability of regional analgesia. Document return of maternal platelet count to normal levels after delivery.

Regional Anesthesia Considerations

The presence of a coagulopathy is cited as a specific contraindication to the use of regional anesthesia due to concern for an epidural hematoma, which can result in serious neurologic complications. Only two cases of epidural hematoma have been reported in gravidas receiving epidurals in labor (one patient had gestational hypertension and the lupus anticoagulant, and the other patient had an ependymoma). All other cases of nonpregnant epidural hematomas occurred in women receiving anticoagulants. Historically, anesthesia recommendations were that epidurals should be withheld if platelet counts were <100,000/μL.

Three series have been published of gravidas undergoing regional analgesia (epidural or spinal) with unexplained, or initially unrecognized, thrombocytopenia at the time of the procedure [8–10]. The combined total was 105 women with platelet counts <150,000/μL; of these, 51 had platelet counts <100,000/μL. No anesthesia complications were reported in these series. Nevertheless, some authors are still reluctant to advise epidurals for platelet counts <100,000/μL due to the small sample sizes in these studies.

Some anesthesiologists recommend a bleeding time prior to placing an epidural in a thrombocytopenic parturient. Bleeding time is influenced by various factors, has large interobserver variation, and cannot predict bleeding or transfusion requirements. This is not useful in assessing platelet function with ITP or GT, and its use should be discouraged.

Preeclampsia/Eclampsia

Preeclampsia accounts for 21 % of cases of maternal thrombocytopenia. Thrombocytopenia is usually moderate and platelet count rarely decreases to <20,000/μL. Thrombocytopenia in patients with preeclampsia always correlates with the severity of the disease. It is considered a sign of worsening disease and is an indication for delivery.

Pathophysiology

Vascular endothelial damage increases platelet activation.

Clinical Manifestations

The most severe spectrum of preeclampsia is established when the vascular endothelial damage produces microangiopathic hemolytic anemia, elevating liver enzymes along with thrombocytopenia and establishing a syndrome known as HELLP (hemolysis, elevated liver enzymes, low platelets).

HELLP syndrome accounts for 21 % of maternal thrombocytopenia in pregnancy.

HELLP syndrome is a variant of severe preeclampsia, first described by Dr. Louis Weinstein in 1982.

Incidence

Hypertensive disorders occur in 7–10 % of all pregnancies; HELLP complicates 10 % of all women with preeclampsia.

Diagnosis and Classification of HELLP Syndrome

Hemolysis is associated with the following:

- Abnormal results on peripheral smear (presence of schistocytes)
- Total bilirubin >1.2 mg/dL
- Lactic dehydrogenase (LDH) >600 U/L

Elevated liver enzymes (three standard deviations above the mean) are as follows:

- Aspartase aminotransferase (AST) >70 U/L
- LDH >600 U/L
- A platelet count <100,000/μL may be noted.

Approximately 50 % of patients have complete HELLP (all components present), and 50 % have incomplete HELLP (at least one components present: EL, HEL, ELLP, LP).

Some physicians subclassify HELLP based on the severity of thrombocytopenia as follows:

- Class 1 – platelet count >50,000/μL
- Class 2 – platelet count 50,000–100,000/μL
- Class 3 – platelet count 100,000–150,000/μL

Clinical Manifestations

Clinical manifestations often are nonspecific (nausea/vomiting, headache in 50 %, epigastric or right upper quadrant pain in 50–67 %).

Early HELLP syndrome is often misdiagnosed as heartburn, with many women prescribed antacids prior to the correct recognition of this potentially life-threatening condition. Having a high index of suspicion for HELLP syndrome in the second half of pregnancy is important.

Not all patients with HELLP syndrome meet the strict criteria for preeclampsia. Approximately 15 % have diastolic blood pressure (BP) >90 mmHg; 15 % have minimal or no proteinuria. Major complications can still occur despite normal blood pressure and proteinuria.

The maternal mortality rate with HELLP is 1 %, resulting from ruptured subcapsular hematomas, hemorrhage, and stroke.

Thrombocytopenia is usually moderate, with counts rarely >20,000/μL.

Major hemorrhage is uncommon, but incisional site oozing or subcutaneous hematomas may occur.

Maternal thrombocytopenia reaches a nadir at 24–48 h postpartum.

Fetal/Neonatal Risks

The perinatal mortality rate is 11 %. Perinatal deaths may occur from placental abruption, asphyxia, and extreme prematurity.

Fetal growth restriction is common, sometimes occurring before maternal manifestations of HELLP.

Neonates may be at increased risk for thrombocytopenia.

Treatment

Delivery is the ultimate cure. Delivery may be delayed for 24–48 h prior to 32–34 weeks' gestation to administer corticosteroids if the patient is asymptomatic and the fetus has reassuring testing.

Magnesium sulfate ($MgSO_4$) should be administered intrapartum and postpartum, regardless of blood pressure levels, to prevent seizures (eclampsia).

HELLP syndrome with thrombocytopenia does not by itself require a cesarean delivery, although cesarean delivery may be acceptable prior to 32 weeks' gestation with an unfavorable cervix due to an anticipated long induction time in a clinically deteriorating gravida.

Maintain platelet counts >20,000/μL for vaginal delivery and 50,000/μL for cesarean delivery. If platelets fall below 50,000/μL prior to cesarean delivery, be prepared to administer platelets just prior to surgery and/or intraoperatively. A range of six to ten units of platelets is usually administered at the time of skin incision, and an additional six units are administered if oozing is noted during the surgery.

If thrombocytopenia is severe, regional anesthesia and pudendal blocks may be contraindicated. In this situation, intravenous narcotics can still be administered for analgesia during labor.

Pfannenstiel incision with primary closure is acceptable for cesarean delivery. Thrombocytopenia and elevated liver function tests commonly worsen postpartum. Platelets should start normalizing by the third postpartum day.

Immune Thrombocytopenic Purpura

ITP is also known as idiopathic thrombocytopenic purpura or autoimmune thrombocytopenic purpura (ATP).

Incidence

Incidence is 1 per 1000–10,000 pregnancies, and it accounts for 3 % of all thrombocytopenic gravidas.

Pathophysiology

Immunoglobulin G (IgG) antiplatelet antibodies recognize membrane glycoproteins and coat the platelets, which then are destroyed by the reticuloendothelial system, predominantly in the spleen.

Antiplatelet antibodies may cross the placenta and cause significant fetal thrombocytopenia (<50,000/μL), which could result in bleeding complications in the neonate.

Minor bleeding complications include purpura, ecchymoses, and melena.

Major bleeding complications include intracranial hemorrhage leading to neurologic impairment or death.

Diagnosis

ITP is a diagnosis of exclusion. The following may be noted:

- Persistent thrombocytopenia (<100,000/μL), increased number of megakaryocytes in the bone marrow, exclusion of systemic disorders or medications/drugs, absence of splenomegaly
- Approximately 80 % of cases are associated with antiplatelet antibodies, although these are not required for the diagnosis.

Clinical Manifestations

The following may be noted:

- Easy bruising, petechiae, epistaxis, and gingival bleeding, although some women are asymptomatic.
- Significant hemorrhage is rare, even when counts fall to less than 20,000/μL.

Maternal Treatment for ITP

No treatment is necessary if platelet counts remain above 50,000/μL and the patient is asymptomatic. However, many physicians will treat for asymptomatic platelet counts of less than 50,000/μL, abnormal bleeding, or prior to invasive procedures such as cesarean delivery or regional anesthesia.

Below are recommended treatments for maternal thrombopenia due to ITP. While they all improve maternal platelet counts, none have been shown to adequately prevent or treat fetal/neonatal thrombocytopenia.

With *steroids* (e.g., prednisone), the following is noted:

- Response time is 3–7 days; maximum effect occurs by 2–3 weeks.

- Approximately 70 % of patients will respond, and 25 % will enter complete remission.
- Risks include hyperglycemia, fluid retention, and bone calcium loss.

With *intravenous immune globulin* (IVIG), the following is noted:

- IVIG works by binding to platelets, blocking the attachment of antiplatelet antibodies.
- IVIG is ideal when time is inadequate for steroids to take effect (prior to surgery or low platelet counts with bleeding).
- Response time is 6–72 h.
- Approximately 70 % of patients will return to pretreatment levels within 30 days.
- This treatment is very expensive.

With *anti-D immunoglobulin* in Rh-positive, nonsplenectomized women, the following is noted:

- Anti-D immunoglobulin binds to maternal red blood cells and results in Fc receptor blockade. The spleen directs its phagocytotic activity to the coated red cells rather than to antibody-coated platelets.
- It is not useful in Rh-negative or splenectomized women.
- Response time of anti-D immunoglobulin is 1–2 days, peak effect in 7–14 days, average duration 30 days.
- Little data are available on the use of anti-D immunoglobulin in pregnant women; risk-benefit ratios need to be considered prior to its usage.

With *splenectomy*, the following is noted:

- Splenectomy removes the organ responsible for the destruction of IgG-coated platelets.
- In nonpregnant women, splenectomy is used for patients who are unresponsive to IVIG.
- Splenectomy usually is avoided during pregnancy for technical reasons, although it remains an option in the first and second trimesters when ITP is severe (counts <10,000/μL) and the patient does not respond to steroids or IVIG.
- Complete remission occurs in two thirds of cases.
- Splenectomy does not have an impact on circulating antibodies that may still cross the placenta and cause neonatal thrombocytopenia.

With *platelet transfusion*, the following is noted:

- This is a temporary measure, which should be administered for life-threatening hemorrhage and should be available prior to surgery for patients with severe thrombocytopenia.
- Six to ten units of platelets are usually administered at one time.
- Platelet counts normally rise by 10,000/μL for each unit of platelets transfused, but in ITP the rise is less pronounced due to destruction of donor platelets.

Key Points

- Gestational thrombocytopenia is the most common cause of thrombocytopenia during pregnancy (70 %), but other underlying causes must be considered as well.
- A thorough history and physical examination is important to rule out most other causes.
- Look at the remainder of CBC and smear to rule out pancytopenia and platelet clumping associated with pseudothrombocytopenia.
- If no antecedent history of thrombocytopenia is present and platelet counts are >70,000/μL, the condition is more likely to be GT.
- If platelet counts fall to <50,000/μL or if a preexisting history of thrombocytopenia is present, the condition is more likely to be ITP.
- Direct or circulating antiplatelet antibodies have no value in the workup of thrombocytopenia in pregnancy because they usually are nonspecific and will not distinguish GT from ITP.
- Cesarean deliveries for ITP or GT should be reserved for obstetrical indications only because vaginal delivery

itself has not been demonstrated to be a cause for intracranial hemorrhage.

- Invasive procedures to determine fetal platelet counts (scalp sampling, PUBS) are no longer considered necessary for ITP, because an infant who is thrombocytopenic may be delivered vaginally. However, PUBS may still be of value in alloimmune thrombocytopenia to assess the severity of the condition and therapeutic response.
- With ITP, obtain cord blood at delivery (if possible) for platelet count and notify the pediatricians to assess neonatal platelet counts due to the risk for continued quantitative platelet decline and postnatal hemorrhage.
- For GT, document normalization of maternal platelet counts after delivery.

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Part IV

HDP and It's Problems Requiring Critical Care

Hypertensive Crisis in Pregnancy

25

Girija Wagh

Introduction

Hypertension affects 10 % of pregnancies, many with underlying chronic hypertension, and approximately 1–2 % will undergo a hypertensive crisis at some point during their lives [1]. Hypertension in pregnancy that becomes severe enough to qualify as a hypertensive crisis is particularly challenging as it is associated with severe risk to the maternal and fetal well-being. This risk may evolve over days or hours and may present as worsening blood pressure culminating in hypertensive crisis resulting in cerebrovascular accidents, renal injury, and seizures. Fetal affliction and death due to placental abruption and acute fetal distress are many a times directly linked to the maternal risks of hypertensive encephalopathy and cerebrovascular accident.

Definition

Hypertensive crises or emergencies are defined as a sudden increase in systolic and diastolic blood pressures associated with "acute end-organ damage" (i.e., cardiovascular, renal, central nervous system) that requires immediate management. Acute blood pressure rise in the absence of end-organ damage is called as "hypertensive urgency" [2–7]. In obstetrics eclampsia and encephalopathy, acute left ventricular failure with pulmonary edema, acute myocardial ischemia, peripartum cardiomyopathy, acute renal failure, and microangiopathic hemolytic anemia (HELLP) can be categorized as hypertensive crises. In fact severe preeclampsia is hypertensive crisis with its characteristics as described in Table 25.1.

Postoperative period in case of cesarean sections or obstetric laparotomies and labor can be associated with high elevated blood pressures. If in the absence of end-organ damage, these can be categorized as hypertensive urgency. Postoperative hypertension is defined so when pressure greater than 190 mmHg and/or diastolic blood pressure greater than 100 mmHg on two consecutive readings is recorded [8, 9]. This however is a subjective definition. This can be as a result of active physiological changes during the early postoperative period. It may result in adverse sequel in obstetric patients who have cardiac diseases, hypertensive diseases, and severe anemia. Even if transient, it may be life-threatening and requires a special mention. Transient high blood pressure can also be seen immediately before, during, and after parturition. Any blood pressure greater than 169 mm of Hg systolic and 109 mm of Hg diastolic in a pregnant and a parturient is taken as hypertensive

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Table 25.1 Severe preeclampsia: new definition

Severe preeclampsia: ACOG task force 2013
SBP/DBP ≥160/110 mmHg, respectively, occurring twice, 4 h apart at rest
Thrombocytopenia (platelets, 100,000 μL)
Impaired liver function defined as either unexplained RUQ unresponsive to medications or hepatic transaminase levels twice normal
Progressive renal insufficiency: creatinine >1.1 mg/dL or twofold increase in creatinine in the absence of underlying renal disease
Pulmonary edema
New onset cerebral or visual disturbances

emergency necessitating antihypertensive medications [10–12].

All obstetricians and birth attendants should be aware of the occurrences of hypertensive crises both emergency and urgency as many times they can be unpredictable and sudden in occurrence. Early and correct diagnosis and prompt and correct therapy have been proved to considerably lower the associated mortality and morbidity [13–16]. Knowledge about the possibility of occurrence with the pathophysiology of the disease and the rational management of the condition is mandatory for obstetricians. This is because improper rapid blood pressure reduction can cause complications. This chapter attempts to explain the pathophysiology, the current concepts, and misconceptions in the context of hypertensive emergencies in association with pregnancy.

Historical Aspects

The syndrome of hypertensive emergency was first described by Volhard and Fahr in 1914 and was characterized by severe accelerated hypertension, associated with evidence of renal disease and by signs of vascular injury to the heart, brain, retina, and kidney. It also was associated with a rapidly fatal course ending in heart attack, renal failure, or stroke [17]. Before the widespread use of antihypertensive agents, the first large study of the natural history of malignant hypertension was published in 1939 [18]. In that pivotal report by Keith and colleagues, untreated malignant hypertension had a 1-year mortality of 79 % and a median survival of 10.5 months.

Epidemiology

Pathophysiology

The factors that lead to the swift rise of blood pressure in patients with existing severe hypertension are not very clear. A possibility of a triggering factor can be considered to cause such a rise on superimposed on pre-existing hypertension. Severity of the underlying hypertension is identified as an important risk of developing crisis or malignant hypertension. The role of mechanical stress on the vessel wall therefore could be critical in its pathogenesis. The stressed vessel wall releases humoral vasoconstrictor substances, and these could be responsible for the initiation and persistence of the hypertensive crisis [19, 20]. This release of vasoconstrictor substances causes rise in blood pressure which causes endothelial damage. Any injury to the vessel lining causes local intravascular activation of the clotting cascade. This further causes fibrinoid necrosis of small blood vessels and release of vasoconstrictor substances and the vicious cycle continues [19, 20]. Thus, the vicious cycle perpetuates causing further vascular injury, tissue ischemia, and release of vasoconstrictor substances [19, 20]. Increased arterial causes pressure natriuresis, i.e., increased excretion of sodium along with water which is a compensatory mechanism to maintain blood pressure within the normal range. The volume depletion that results from pressure natriuresis stimulates the release of vasoconstrictor substances from the kidney. This release of renal vasoconstrictor substances has been postulated to play a central role in the pathophysiology of malignant hypertension [21]. Activation of the renin–angiotensin system has been strongly associated with the initiation and persistence of the vascular injury associated with severe hypertension [22–24]. Vasopressin, endothelin, and catecholamines also are postulated to play important roles in the pathophysiology of hypertensive emergencies [25–28].

The global vasoconstriction is contravened in the cerebral circulation. In the initial phases of severe hypertension, the cerebral vessels constrict to maintain cerebral perfusion pressure in the face of increased systemic arterial pressure. Once the

limits of autoregulation are exceeded, reflex cerebral dilatation and resultant overperfusion lead to microvascular damage, exudation, microthrombus formation, and increased intracranial pressure, which in turn result in the encephalopathic picture. In pregnancy, the susceptibility to complications increases due to loss of cerebrovascular autoregulation, resulting in hypertensive encephalopathy once the upper limits of cerebral perfusion pressures are exceeded [29]. Rapid control of blood pressure is needed even more because of the risks of placental abruption and stroke. Stroke is of special concern in the setting of thrombocytopenia or HELLP syndrome. Cerebral edema may be more closely associated with endothelial cell injury than with blood pressure [30]; still control of blood pressure may help minimize the endothelial cell injury.

Clinical Presentations

Any hypertensive disorder during pregnancy or after delivery can be associated with hypertensive emergency, i.e., acute onset of systolic blood pressure equal to or more than 160 mm of Hg, diastolic blood pressure equal to or more than 110 mm of Hg, or both. Such an acute onset of high blood pressure recorded correctly by standardized technique and persistent for or more than 15 min is called as hypertensive emergency. Such an onset of severe hypertension can be seen during pregnancy in the second half in the absence of chronic hypertension and in mothers with superimposed proteinuric hypertension in chronic hypertensives, gestational hypertension, and HELLP. Cerebral injury is associated with severe hypertension especially the systolic blood pressure rise. The degree of systolic blood pressure as opposed to diastolic blood pressure is the most important predictor of cerebral infarct or intracerebral hemorrhage. In a case series by Martin et al of patients suffering from stroke, it was observed that 54 % of these patients had systolic blood pressure of equal to or more than 160 mm of Hg and only 13 % had a rise in diastolic blood pressure equal or more than 110 mm of Hg. Severe systolic blood pressure of 160 mm of Hg or more is also known to be associated with stroke in nonpregnant women, and this is the pressure where probably the autoregulatory mechanism of the intracerebral circulation may be challenged.

The symptoms and signs of hypertensive crises vary from patient to patient. Headache, altered level of consciousness, and/or focal neurologic signs are seen in patients with hypertensive encephalopathy. On physical examination, these patients may have retinopathy with arteriolar changes, hemorrhages, and exudates, as well as papilledema. In other patients, the cardiovascular manifestations of hypertensive crises may predominate, with angina, acute myocardial infarction, or acute left ventricular failure. In some patients, severe injury to the kidneys may lead to acute renal failure with oliguria and/or hematuria. In pregnant patients, the acute elevations in blood pressure may range from a mild to a life-threatening disease process. The clinical features vary but may include visual field defects, severe headaches, seizures, altered mental status, acute cerebrovascular accidents, severe right upper quadrant abdominal pain, congestive heart failure, and oliguria. In the vast majority of cases, this process can only be terminated by delivery.

Management of Hypertensive Crisis in Pregnancy

The three important aspects of management of hypertensive crisis are diagnosis, stabilization, and delivery of the mother. In pregnancy cardiac, renal, and cerebrovascular damages are responsible for the major morbidity and mortality. Fetal morbidity and mortality are associated with the maternal condition. Therefore, the first step is to stabilize the blood pressure. This should be prompt but not sudden and also optimum in order to maintain the placental perfusion.

Blood Pressure Control

Blood pressure restoration should be in the range of 140–150 mmHg systolic and 90–100 mmHg diastolic. This helps in maintaining the placental, cerebrovascular, cardiac, and renal perfusion and helps reverse part of the pathology initiated. This however does not eliminate the likelihood of stroke.

Sudden reduction in blood pressure is detrimental for both the mother and the fetus. Antihypertensive medications are indicated when the blood pressure records ≥160 mm of Hg systolic and ≥110 mm of Hg diastolic. In presence of other associated adverse factors can be commenced at 150/100 mm of Hg reading. In a crisis situation the blood pressure may be lowered by 20–30 % initially, and then gradual reduction may be sought.

The drugs available for rapid blood pressure control are nifedipine orally and labetolol and hydralazine parenterally. The dosages of both these commonly used medications are mentioned in the table below (Table 25.2).

It is important to note that nifedipine is an effective and easy to administer medication but should never be used sublingually. Even during oral administration, it has to be given gradually as oral absorptions too can cause catastrophic reduction in blood pressure which can be hazardous for both the mother and the baby.

Labetolol

It is a beta-adrenergic antagonist with alpha-blocking activity and therefore said to be cardioprotective. It causes decreased systemic vascular resistance (vasodilation), reduces afterload, and reduces cardiac contractility and heart rate but maintains cardiac output unlike pure beta blockers. The drug is metabolized in the liver. It can cause fetal and maternal bradycardia and postural hypotension. Intravenous labetolol is consistently recommended across guidelines for initial therapy in acutely severe hypertension. Labetolol is contraindicated in cardiac abnormalities and asthma. Neonatal bradycardia is associated with labetolol, and fetal heart should be closely monitored for this. In case of severe hypertension, labetolol can also be given orally in case the intravenous access is not possible.

Hydralazine

The ACOG 2011 Committee Opinion on treatment of severe hypertension along with labetolol also recommends hydralazine as a first-line therapy. Hydralazine is associated with more severe side effects precluding its use. Parenteral hydralazine causes maternal hypotension and tachycardia especially in volume-depleted mothers. It is also associated with significant fetal tachycardia (Table 25.3).

Table 25.2 Antihypertensive medications for urgent control

Drug	Dose and route	Concerns	Advantages
Labetolol	10–20 mg IV, then 20–80 mg every 20–30 min, maximum of 300 mg for infusion 1–2 mg/min dose. Duration of action: 3–6 h	Contraindications: congestive cardiac failure, asthma, diabetes mellitus, bradycardia	Less incidence of hypotension
Nifedipine	Tabs only: 10–30 mg per orally, repeat in 45 m if needed till the requisite control is achieved total dose of 40–120 mg in 24 h can be used	Sublingual use can cause drastic decrease in blood pressure	Effective and easy to administer. Can be shifted to sustained release formulations once the preliminary control has been achieved
Hydralazine	Begin with no more than 5 mg slow IV push over 1–2 min. If no response in 15–20 min or if desired diastolic BP not achieved, repeat as ordered, 5–10 mg slow IV push. Maximum cumulative dose in 12 h of 40 mg IVP. Half-life: 3–5 h	Hypotension and angina. Headache, epigastric pain, and nausea	Hydralazine is a rapid-acting antihypertensive that can be given in low-dose increments. It causes smooth muscle relaxation in the arterioles thus causing peripheral vasodilation without reducing uteroplacental blood flow

Table 25.3 Stepwise management of hypertensive crisis in obstetrics

1. BP: >160/110 mm of Hg: PO2: should be >90: PR should be between 70 and 100 beats/min
2. Auscultate the fetal heart: preferably strap the EFM machine
3. Administer nifedipine 10 mg orally with sip of water: do not give sublingually
4. Measure the BP every 10 mins
5. After 30 mins if the BP remains the same, repeat 20 mg of nifedipine
6. If no response of reduction of BP to 150/100 mm of Hg, consider repeat dose of 10 mg of nifedipine or labetolol
7. Give 20 mg of labetolol every 20 mins monitoring the BP every 20 mins till the goal of 150/100 mm of Hg is achieved (maximum dose allowed is 240 mg)
8. Oral labetolol too can be considered if IV access not available. Start with 400 mg orally and repeat after every 30 mins with BP measurement
9. Mg SO_4 full loading dose: 4 g IV with 10 G IM to be administered
Admit and investigate
1. CBC with platelet counts and PBS for hemolysis
2. SGOT, SGPT, LDH
3. Se creatinine/Se uric acid
4. Se bilirubin, se electrolytes if critical and unconscious
5. Coagulation profile if platelets less than 1 lakh/cmm
6. Proteinuria: dipstik/spot test for protein; creatinine ratio or 24 h protein estimation
7. Other maternal and fetal surveillance tests serology, blood group, USG with fetal Doppler
Facility
1. During the acute management phase, mothers should be managed in an ICU, HDU, or a delivery unit capable of critical care
2. Physicians, critical care specialists, neonatologists, and anesthesiologist should be involved in the care
3. Preparedness for blood components and obstetric expertise should be reaffirmed
4. Shifting to a better level facility can be considered but with stabilization, seizure prevention, and proper transportation and information to the receiving center (steroids, magnesium sulfate, nifedipine)
Delivery decision: only after stabilization and proper maternal and fetal evaluation. No mother to be delivered without proper stabilization of hypertension

Conclusion

Hypertensive crisis is not a rare entity, and every obstetric unit should be well equipped with infrastructure, personnel, and protocols to handle it. Severe blood pressure must be attended to promptly and effectively. Both the diastolic and systolic blood pressures need to be tackled. Underlying pathology and end-organ damage need to be investigated and appropriate delivery decisions undertaken.

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Eclampsia

26

Sanjay Gupte

Eclampsia is an acute and severe complication of preeclampsia. In classic eclampsia severe rise in hypertension is followed with acute headache and tonic-clonic convulsions. Eclampsia cases are rarely seen in developed countries (1 in 2000); however, in developing countries, the figures are much higher.

The FOGSI-ICOG National Eclampsia Registry has brought forth some revealing trends. Eclampsia prevalence among registry patients is 1.9 %. National sample surveys in the past have shown prevalence to be 1–5 %. This is out of the 111,725 deliveries analyzed from the cases reported by 175 reporting centers. The number of cases of eclampsia is more than cases of imminent eclampsia. This points to the lost opportunities of prevention. 17 % of preeclampsia patients are actually in the adolescent age group reflecting the very early age at marriage in spite of several awareness programs and legal guidelines. 76.34 % of the patients were between 21 and 30 years of age, thus rendering a very young population morbid and at risk of mortality. It also is a disease of the first-time pregnant woman as 81 % of the patients with preeclampsia are primigravid.

Antenatal care has been identified as the single intervention which could influence the maternal mortality of our country. Many women still seem to be unreached with this basic pregnancy evaluation. Most of the patients reported by the registry were registered for antenatal care either in the second (40.98 %) or the third trimester (46.28 %). Very few (12.54 %) were booked in the first trimester [1].

Pathophysiology of Eclampsia

Pathophysiology of eclampsia has remained enigmatic for a long time. This was because clinical and investigative research was difficult in such an acute and severe condition. New research has identified two different pathways causing the clinical symptoms of convulsions.

Severely raised systemic blood pressure leads to first cerebral vasospasm followed by decreased cerebral perfusion, leading to cerebral anoxia. This leads to ischemic damage and cerebral edema causing eclamptic foci in the brain and convulsion.

Raised cerebral perfusion pressure also causes endothelial damage, barotrauma, and vascular damage, leading to cerebral edema, hemorrhage, and cerebral irritability in some cases.

But there are some atypical cases where systemic hypertension is not high but still they get eclamptic convulsions. This is depicted to be due to abnormal autoregulation in the cerebral circulation.

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The classical sign seen on MRI in eclamptic patient is known as PRES or posterior reversible encephalopathy syndrome. PRES is thought to develop when an increase in blood pressure, in conjunction with endothelial cell dysfunction, exceeds the upper limit of cerebrovascular autoregulation and results in forced vasodilatation of the cerebral resistance vessels. Subsequent hyperperfusion, capillary bed injury, and, eventually, breakthrough of the blood-brain barrier with resulting vasogenic edema are thought to be its pathophysiological hallmarks [2].

PRES can present with eclamptic seizures; additional features could be severe headache, visual deficit to the extent of blindness, confusion, and in severe cases with signs of intracranial hemorrhage. So far this condition was thought to be completely reversible, hence the name.

However recent publications where long-term neuroimaging was done have shown persistent neurological and cognitive problems [3].

Knowing this pathophysiology is important because this allows us to choose ideal agent to prevent eclampsia. This would be a drug causing peripheral vasodilatation and reduction in cerebral perfusion pressure, which maintains normal cerebral flow. Magnesium sulfate comes close as such an agent. It has systemic antihypertensive effect (though not very pronounced). It reduces cerebral perfusion pressure by causing vasodilatation. In addition it also has membrane-stabilizing action which is vital to prevent eclamptic convulsions.

Another good agent acting in a similar fashion is labetalol. It is a selective alpha-1 and nonselective beta-blocker. Its alpha to beta blockade ratio are 1:3 and 1:7 for oral and intravenous route. And at higher doses, it has been shown to be a membrane stabilizer. It has further advantages like dose-dependent decrease in blood pressure without reflex tachycardia. It does not cause fetal distress as it does not decrease uteroplacental perfusion. It also has action on antiplatelet aggregation by thromboxane-reducing effect and helps in fetal lung maturation stimulation.

Management of Eclampsia

Management starts with prevention. Magpie Trial conducted in June 2002, which had a sample size of 10,141 women with preeclampsia in 175 secondary- and tertiary-level hospitals in 33 countries, showed beyond doubt that magnesium sulfate is an ideal agent to prevent eclampsia in severe preeclamptic women [4].

Recently FOGSI (Federation of Obstetric and Gynecological Societies of India) has come out with guidelines for management of eclampsia [5].

The following are the principles of management of eclampsia:

Call for help	
CAvoid tongue bite	Insert airway/mouth gag
Avoid injury	Padded bed rails, restraints
Maintain oxygenation O2	Pulse oximetry
Minimize aspiration	Lateral decubitus position, oral suction
Initiate magnesium sulfate.	
Control blood pressure.	
Delivery (LSCS is preferred for obstetric/fetal indications only).	

Drug of choice is MgSo4. It is safe drug. Pritchard regimen is the preferred regimen.

1. Loading dose: 4 g {four ampoules of 50 % w/v MgSO4 + 12 ml normal saline or sterile water} slow IV at 4 ml/5 min rate using 20 ml syringe is given. 5 g {five ampoules of 50 % w/v MgSO4 + 1 ml 2 % lignocaine} injection is given deep intramuscular in each buttocks using 10 ml syringe.
2. Maintenance dose**:** It is given every 4 hours as 5 g {five ampoules of 50 % w/v MgSO4 + 1 ml 2 % Lignocaine} deep intramuscular injection in alternate buttocks, monitoring the following signs:

   (a) Respiratory rate – >16/min.
   (b) Patellar reflexes are present.
   (c) Urine output – >100 ml in last 4 h (25 ml/h).

(Serum monitoring of magnesium levels are not routinely recommended as it has not been shown to be superior to clinical monitoring). Maintenance dose is given till 24 h past delivery or last convulsion, whichever is late.

3. Administer the full magnesium sulfate loading dose before transfer to a higher-level health care if management of such patients is not possible at that facility**.**
4. Dose for recurrence of convulsion: After loading dose, if convulsions do not stop or/recur, repeat 2 g MgSO4 slow IV, or alternatively IV diazepam or IV thiopentone sodium are given**.**

Alternate regimen is Zuspan IV regimen:

1. Loading dose: 4 g {four ampoules of 50 % w/v MgSO4 + 12 ml normal saline or sterile water} slow IV using 20 ml syringe is given at a rate not to exceed 1 g/min.
2. Maintenance dose: It is given as an infusion of 1 g/h till 24 h past delivery or last convulsion, whichever is late.
3. This regimen requires vigilant monitoring and is recommended in certain situations like thrombocytopenia (<75,000 platelets/mm^3) or DIC where intramuscular injections are avoided.

Vaginal delivery is preferred so induction of labor is recommended immediately after stabilization of patient. Cesarean section is done for obstetric indications.

Continue use of antenatal antihypertensive treatment during labor.

Drugs preferred are labetalol, nifedipine, and hydralazine. The dosage used in this acute emergent situation are (Table 26.1):

- Nifedipine and hydralazine can cause tachycardia. They are not recommended to be used in patients with heart rate above 100/min. Labetalol is the appropriate drug in such patients.
- Labetalol should be avoided in patients with bradycardia (heart rate <60 bpm) and asthma and in those with congestive cardiac failure. Here, nifedipine is the drug of choice.
- MgSO4 is recommended to prevent eclampsia and not as an antihypertensive.
- Nifedipine and MgSO4 can be used concurrently.
- Sublingual nifedipine is not recommended.

Table 26.1 Antihypertensive therapy for severe hypertension

Medication	Onset of action	Dose
Labetalol	10–15 min	20 mg IV, then 40–80 mg every 10 min up to maximum dose of 300 mg or continuous infusion at 1–2 mg/min
Nifedipine	5–10 min	10 mg orally, repeated in 30 min, (20 mg) × 2 doses, then 10–20 mg every 4–6 h up to maximum dose 240 mg/24 h
Hydralazine	10–20 min	5–10 mg IV every 20 min up to maximum dose of 30 mg

BP of ≥160 mmHg systolic or ≥110 mmHg diastolic

New Discoveries about CPP: Cerebral Perfusion Pressure

With the help of newer investigations like MRI, CT scan, transcranial Doppler, as well as positive photon emission studies, eclamptic patients are differentiated in two groups. The first group is of patients with minimal or no impairment of consciousness between two convulsions. This group of patients has better prognosis as their autoregulation is still intact and cerebral perfusion pressure within limits. The second group is of patients with impaired consciousness in between two convulsions. These patients have extremely raised cerebral perfusion pressure and many develop intracranial hemorrhage, but at the same time it is important to lower the blood pressure gradually, otherwise cerebral perfusion pressure may fall

suddenly. Diastolic blood pressure should not to be lowered by more than 30 mmHg to maintain cerebral perfusion. Otherwise it can lead to cerebral ischemia.

Postpartum Eclampsia

Finally it is important to remember that just delivering the patient of severe preeclampsia is not an adequate treatment. Vigilance has to be maintained for postpartum eclampsia. The National Eclampsia Registry showed that 13.72 % patients have postpartum eclampsia. Hence it is important to keep close follow-up of severe preeclampsia cases even after the delivery.

Conclusion

Eclampsia can be a life-threatening condition both for the mother and the fetus. Hence prevention should be the goal. But if eclampsia does occur, proper emergency help should always be kept ready. By following the clinical guidelines mentioned and training the caregiver team with eclampsia drills carried out from time to time, maternal and perinatal mortality as well as morbidity can be reduced to a great extent.

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Antepartum Hemorrhage

27

Nidhi Gupta

Introduction

In addition to unacceptably high perinatal mortality [1], antepartum hemorrhage (APH) is an important contributory cause of maternal mortality and morbidity in the developing countries. Although it is not preventable, an early diagnosis and treatment can improve maternal and perinatal outcome to a large extent.

Definition

Any bleeding to and from the genital tract, during pregnancy, between 20 weeks of pregnancy and delivery of fetus is known as antepartum hemorrhage. Similar bleeding prior to the age of viability is known as threatened abortion.

In the past, 28 weeks was considered the age of viability. But, currently with improved neonatal intensive care unit (NICU) facility, particularly in the industrialized world, fetal survival is possible as early as 22 weeks.

However, a universal agreement is lacking regarding the age of viability and so the definition of APH is therefore variable from country to country (Canada, 20 weeks, and the United Kingdom, 24 weeks) [2]. However, irrespective of the age of viability, any bleeding during the second half of pregnancy should be taken seriously and proper evaluation must be done.

Incidence

As many cases of APH are mild in nature and is not recognizable clinically, the exact incidence of the condition is difficult to determine.

Moreover, confusion about the age of viability in different countries makes the situation more complex. APH complicates about 2–5 % of all pregnancies [3].

Causes

The causes of APH are diverse, varied, and multifactorial (Table 27.1).

The source of bleeding could be either fetoplacental or maternal. From the obstetric view point, the two most important causes of APH are placenta previa and abruptio placentae, constituting more than 50 % of cases of APH.

However, in majority of the cases of APH, the cause remained unknown and classified under the category of indeterminate, unexplained, unclassified, or idiopathic [4].

The other rare but significant causes include vasa previa; ruptured uterus and show prior to onset of labor; local lesions of the cervix, vagina,

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Table 27.1 Causes of antepartum hemorrhage

I.	Placental causes
	Placenta previa 30 %
	Abruptio placentae 35 %
II.	Extraplacental causes
	Cervical erosion
	Cervical cancer
	Cervical polyp
	Trichomonas vaginitis
	Vulval/vaginal varicosities
III.	Others
	Vasa previa
	Marginal vein bleeding
	Excessive show
IV.	Trauma
	Foreign body
	Genital laceration
V.	Systemic disorders
	Von Willebrand disease and other bleeding diathesis
	Leukemia
VI.	Unknown/undetermined

and vulva; and also bleeding from the urinary or gastrointestinal tract.

Management

Principles

The management of APH should be planned as general management (applicable to all cases of APH) with appropriate modification, depending on the type and severity of bleeding and specific management depending on the cause of bleeding, gestational age, and condition of the fetus.

Setup

It is preferable that the management should be undertaken in a setup having facility of round-the-clock blood transfusion, immediate termination of pregnancy (if required) by cesarean section, neonatal resuscitation, and intensive care facilities for both the mother and the neonate.

Transport and Transfer

If the setup lacks those necessary facilities, the patient needs to be transferred by an ambulance to a nearby appropriate center for management. The patient should be resuscitated with intravenous fluids. As the fetus is safer in the uterus, it is better to continue the resuscitation and transfer of the patient simultaneously. Fetal hemoglobin oxygen-binding capacity maintains adequate oxygenation until maternal partial pressures are less than 60.

Many a times, the insignificant bleeding at presentation could herald a subsequent significant bleeding at a later date and those cases also demand equal attention.

Initial management includes eliciting a proper history, physical examination of the patient as regards to fetomaternal condition, and initiation of immediate treatment and investigation (refer to Tables 27.2, 27.3, and 27.4).

Immediate Treatment

Irrespective of the cause, significant vaginal bleeding during pregnancy needs to be managed with rapid assessment of maternal and fetal status, fluid resuscitation, replacement of blood products when necessary, and an appropriately timed delivery [6]. As the bleeding is unpredictable in nature and having the possibility of sudden deterioration in clinical condition, even a minor bleeding may have considerable significance in pregnancy and should receive appropriate attention by the on duty obstetrician.

Table 27.2 Relevant history in cases of antepartum hemorrhage

Initiating factors of bleeding, if any (trauma, coitus, etc.)
Details of bleeding: amount, character, color
Associated abdominal pains or uterine contraction
History of *previous bleeding* in the current pregnancy
History of *ruptured membranes*
Gestational age estimation
Information about *placental site* (by the latest USG)
Perception of *fetal movements*

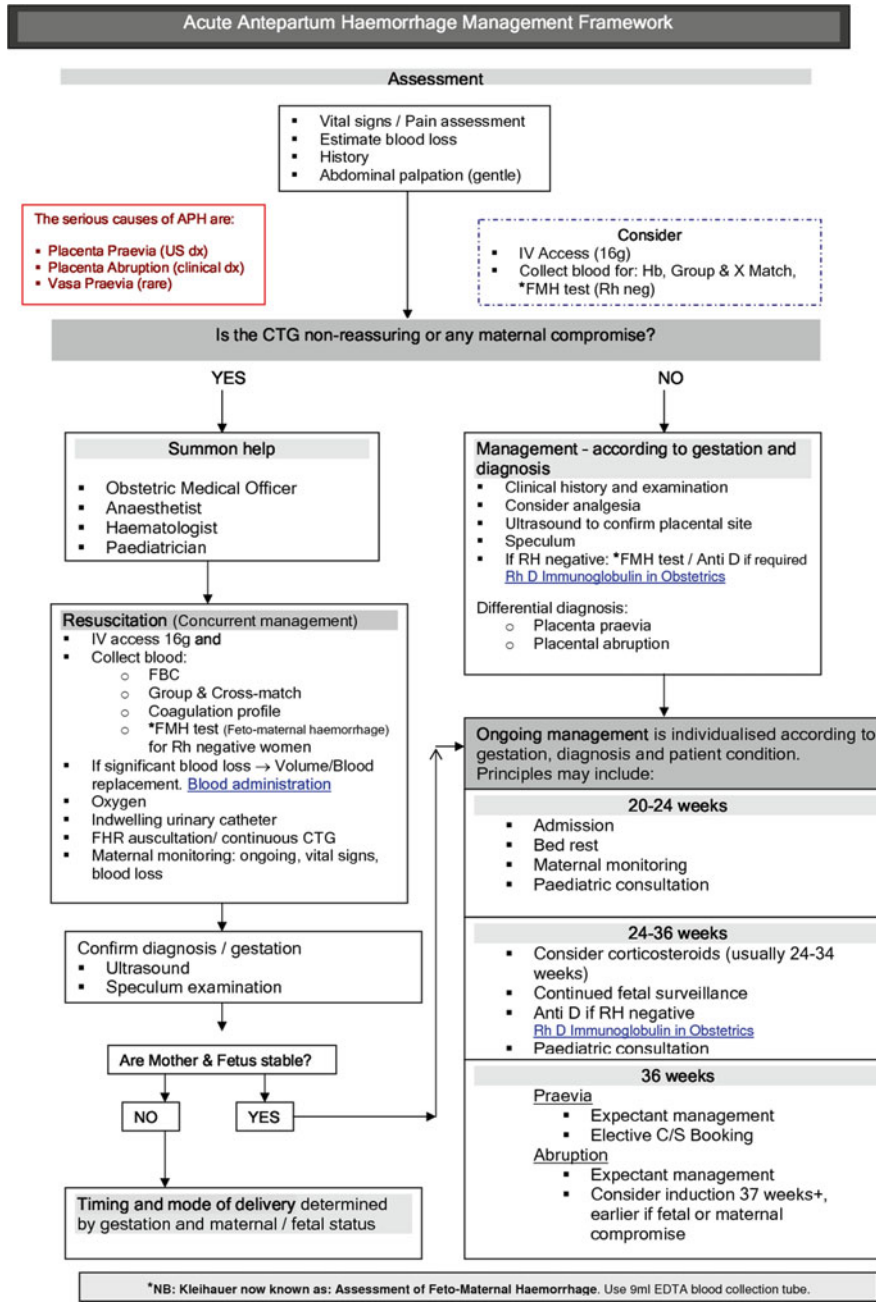
Acute Antepartum Haemorrhage Management Framework
Assessment
Vital signs / Pain assessment
Estimate blood loss
History
Abdominal palpation (gentle)
The serious causes of APH are:
Placenta Praevia (US dx)
Placenta Abruption (clinical dx)
Vasa Praevia (rare)
Consider
IV Access (16g)
Collect blood for: Hb, Group & X Match, *FMH test (Rh neg)
Is the CTG non-reassuring or any maternal compromise?
YES
NO
Summon help
Obstetric Medical Officer
Anaesthetist
Haematologist
Paediatrician
Management - according to gestation and diagnosis
Clinical history and examination
Consider analgesia
Ultrasound to confirm placental site
Speculum
If RH negative: *FMH test / Anti D if required
Rh D Immunoglobulin in Obstetrics
Differential diagnosis:
Placenta praevia
Placental abruption
Resuscitation (Concurrent management)
IV access 16g and
Collect blood:
FBC
Group & Cross-match
Coagulation profile
*FMH test (Feto-maternal haemorrhage) for Rh negative women
If significant blood loss → Volume/Blood replacement. Blood administration
Oxygen
Indwelling urinary catheter
FHR auscultation/ continuous CTG
Maternal monitoring: ongoing, vital signs, blood loss
Ongoing management is individualised according to gestation, diagnosis and patient condition. Principles may include:
20-24 weeks
Admission
Bed rest
Maternal monitoring
Paediatric consultation
24-36 weeks
Consider corticosteroids (usually 24-34 weeks)
Continued fetal surveillance
Anti D if RH negative
Rh D Immunoglobulin in Obstetrics
Paediatric consultation
36 weeks
Praevia
Expectant management
Elective C/S Booking
Abruption
Expectant management
Consider induction 37 weeks+, earlier if fetal or maternal compromise
Confirm diagnosis / gestation
Ultrasound
Speculum examination
Are Mother & Fetus stable?
NO
YES
Timing and mode of delivery determined by gestation and maternal / fetal status
*NB: Kleihauer now known as: Assessment of Feto-Maternal Haemorrhage. Use 9ml EDTA blood collection tube.

Table 27.3 Physical examination of cases with antepartum hemorrhage

Maternal vital signs: pulse, blood pressure (BP), respiration rate (tachycardia and hypotension could be a late feature of shock in a young healthy pregnant lady)
Clinical evidence of shock: restlessness, pallor, and cold clammy extremities
Gentle abdominal examination: to look for whether fundal height is compatible with estimated gestational age, lie and presentation of the fetus, any uterine activity, consistency of uterus, any localized uterine tenderness, number and viability of fetuses, and any fetal compromise by auscultation and/or cardiotocography
Vaginal examination: traditionally, digital examination is not undertaken at the time of presentation and before placenta previa is excluded conclusively. However, vulval inspection is advisable to quickly assess whether bleeding is continuing or not and also to have an estimate of probable blood loss. A sterile speculum examination can be performed safely before placental localization, but a digital examination should be avoided until placenta previa is excluded by ultrasonography [5]

Table 27.4 Immediate treatment and investigation in a case of antepartum hemorrhage

Intravenous (IV) line is established preferably with a wide-bore cannula to facilitate rapid volume replacement
Blood sample is obtained for hemoglobin or hematocrit estimation and determination of grouping and typing. In presence of severe or continuous bleeding, at least four units of blood should be kept ready by cross-matching
Pending arrival of blood, volume replacement should be started with IV fluids, initially with crystalloids and then, if necessary, by colloids
Clinically, if abruptio is suspected, a coagulation profile and measurement of urea, creatinine, and serum electrolytes are also performed
In Rh-negative women, Kleihauer-Betke test is performed on maternal blood. If vasa previa is suspected, an alkali denaturation test (APT) is performed on vaginal blood to identify fetal source of bleeding
Soon after maternal and fetal condition is stable, an ultrasonography (USG) scan is arranged to localize placenta and to confirm or exclude placenta previa or to exclude a major placental abruption. It can also provide additional requisite information about the fetus
Cardiotocography may show evidences of fetal compromise, an indirect evidence of uterine irritability

For management of massive obstetric hemorrhage cases, each obstetric unit must have a protocol of their own, and there should be a good interdepartmental liaison between the labor ward and the blood bank.

Subsequent Management

After initial assessment and institution of treatment, depending on persistence of bleeding and its quantum, gestational age and condition of the fetus, the option is either immediate delivery or expectant management. Subsequent management depends on specific cause.

Placenta Previa (Fig. 27.1)

Definition

Previa means "in front." It is a type of placenta when at least a part of it is implanted in the lower uterine segment and in front of the internal cervical os.

It is one of the leading causes of vaginal bleeding in the second and third trimester of pregnancy and thereby responsible for both fetal and maternal morbidity and mortality.

Classification

I. *Site of implantation* (Fig. 27.2)
Depending on the sites of implantation, placenta previa is typically divided into four grades:
- *Grade I*, *low-lying placenta*: Placenta is implanted into the lower uterine segment, but the lower limit of it does not reach up to the internal os.
- *Grade II*, *marginal placenta previa*: Leading placental edge reaches but does not cover the internal os.
- *Grade III*, *partial placenta previa*: Placenta partially or asymmetrically covers the internal os.

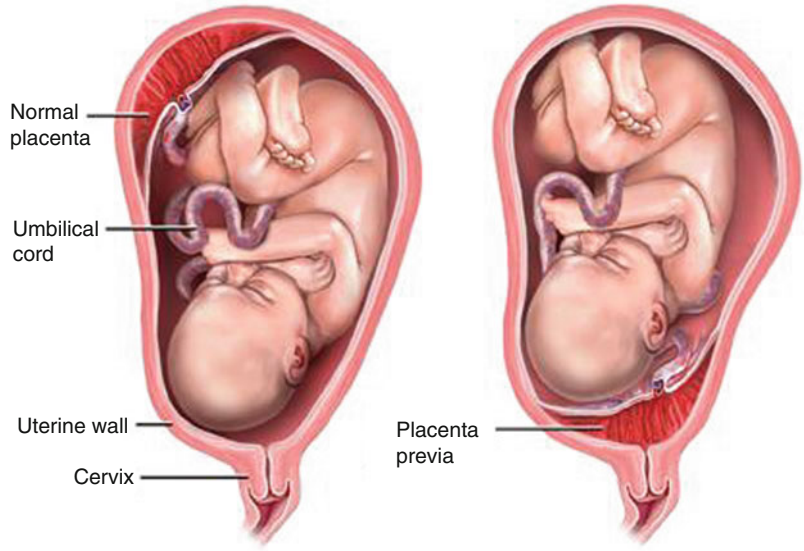

Fig. 27.1 Normal placenta and placenta previa

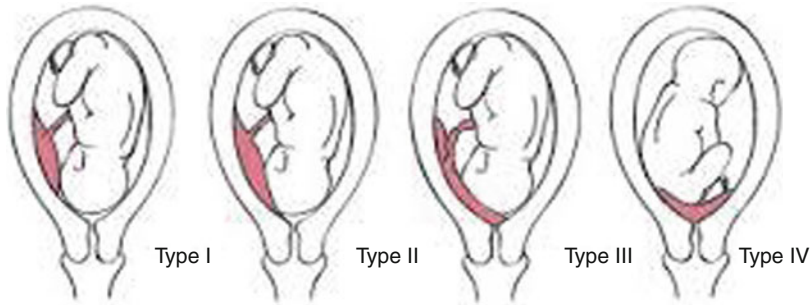

Fig. 27.2 Types of placenta previa

Grade IV, *total or central placenta previa*: Placenta covers the internal os completely.

II. *Anatomical* (*based on TVS*)

Total: Placenta completely covers the internal os.

Partial: Lower placental border does not cover but is within 30 mm of the internal cervical os.

Low lying: Placental margin can be seen with endovaginal probe but is more than 30 mm away from the internal os.

Incidence

At term, the incidence of placenta previa is 0.4–0.8 % of all pregnancies [7].

Placenta previa is a common incidental observation on second-trimester ultrasonography and found in approximately 4 % of cases at 20–24 weeks gestation [8]. At term, the incidence is however reduced to only 0.4 % of pregnancies [9]. This is because of the so-called placental migration, resulting from differential growth of the uterus and placenta.

Etiology

The exact reason for implantation of blastocyst into the lower uterine segment and subsequent development of placenta previa is unknown. Although a chance occurrence cannot be ruled out, a number of well-recognized associations had been noted with placenta previa.

Damage to the endometrium or myometrium due to previous uterine surgery or infection might be a contributory factor for such implantation. Significant association had been noticed between previous CS deliveries and placenta previa [10]. A uterine scar also predisposes to morbid placentation, and the risk of placenta accreta, increta, and percreta increases with the number of cesarean deliveries [11] (Table 27.5).

The incidence increases with age, parity, tobacco use, and previous cesarean deliveries.

Drug abuse (especially cocaine) and history of abortion (spontaneous or induced, particularly their number) are also recognized risk factors for placenta previa.

Increase in number of cesarean deliveries increases the incidence of placenta previa so that it becomes almost ten after four or more cesarean deliveries. Abruptio placentae in previous pregnancy increases the chance of placenta previa in present pregnancy. The risk of recurrence of placenta previa in subsequent pregnancy is increased by eight to ten times [12].

Table 27.5 Risk factors for placenta previa

Advanced age
High parity
Cigarette smoking (*risk increased by three- to sixfold*)
Cocaine use (*risk increased by 2.4-fold*)
Previous CS deliveries – risk increased more with number of previous CS
Previous abortion or curettage (*risk increased by 1.8-fold*)
Previous manual removal of placenta
Deficient endometrium due to presence or history of submucous fibroid or endometritis
Large placenta, e.g., multiple pregnancy

Maternal Complications

The complications are related to bleeding and its consequences. Globally, although, maternal mortality had improved.

The maternal risks are summarized in Table 27.6.

Fetal Complications

Because of conservative management and improved neonatal care facility, perinatal mortality had improved significantly over the years, but compared to the control population, perinatal mortality associated with placenta previa is higher [13] by a factor of 2.9–4.25. Perinatal mortality in the industrialized world is 42–81 per 1,000 births. The causes of increased perinatal mortality and morbidity are shown in Table 27.7.

Presentation

The classic presentation is vaginal bleeding with the following characteristics as outlined in Table 27.8.

Second-trimester scan can identify asymptomatic low-lying placenta. These cases need to be counseled properly mentioning the possible risk of bleeding and also the fact of the so-called placental migration during the course of pregnancy. In addition, nine to ten of cases of placenta previa are associated with placenta accreta.

Diagnosis (Fig. 27.3)

Many cases of placenta previa are diagnosed by routine ultrasound. In other cases, the initial diagnosis is made when the patient comes to the hospital with vaginal bleeding. Placenta previa is a common incidental finding on second-trimester USG and should be confirmed in the third trimester. Placenta previa is a common

Table 27.6 Maternal complications

Antepartum	Intrapartum	Postpartum
Antepartum hemorrhage with varying degree of shock Malpresentation most common is breech Premature labor	Early rupture of the membrane Cord prolapse Intrapartum hemorrhage Increased incidence of operative interference PPH: Imperfect retraction Placenta accrete, increta, and percreta Trauma Retained placenta	Sepsis Subinvolution Embolism Recurrence in subsequent pregnancies (4–8 %)

Table 27.7 Fetal complications

Increased incidence of respiratory distress syndrome (RDS) because of increased frequency of preterm birth (almost 50 %). The *more premature*, *the severe is RDS and the poorer the neonatal prognosis*
Increase incidence of major congenital abnormalities. *The most common malformations are those of the central nervous system* (*CNS*), *cardiovascular system* (*CVS*), *respiratory and gastrointestinal* (*GI*) *system*
Perinatal mortality is also directly related to total amount of blood lost antepartum
Increased incidence of fetal anemia
Increased incidence of intrauterine growth retardation (IUGR) (occurs in 16 %)
Additional fetal risks include cord prolapse and compression and sudden unexplained fetal death due to rupture of vasa previa and severe maternal hypovolemic shock
Fetal malpresentation

Table 27.8 Pattern of bleeding in placenta previa

Bleeding occurs suddenly and is usually unprovoked
Bleeding is usually bright red, painless condition (*absence of pain is considered, as a distinguishing point from abruptio placentae*). However, some degree of uterine irritability is present in about 20 % of the cases either because of associated abruption or because of simultaneous onset of labor
Initial bleeding is not usually profuse. It spontaneously resolves on its own, only to recur later. First episode of bleeding is often referred to as "warning hemorrhage" or "sentinel bleed"
The first episode of bleeding is commonly noted at 27–32 weeks of gestation
In more than 50 % of cases, bleeding occurs prior to 36 weeks

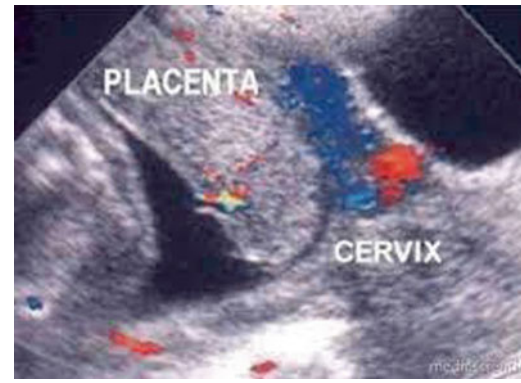

Fig. 27.3 Ultrasound diagnosis of placenta previa

incidental observation on second-trimester USG and found in approximately 4 % of cases at 20–24 weeks gestation. At term, the incidence is however reduced to only 0.4 % of pregnancies. This is because of the so-called placental migration, resulting from differential growth of the upper and lower segments of the uterus with the growth and development of pregnancy. Only 10 % of low-lying placentas identified at the 16–20 weeks USG will remain low at term [14].

Ultrasonography is the mainstay of diagnosis of placenta previa. Transabdominal sonography (TAS) route is commonly employed. It is a simple, safe, and precise method to localize the placenta with an accuracy of 93–98 %. Because of focal

uterine contraction or distension of the bladder, a false-positive result is a distinct possibility. Errors in diagnosis are most likely in cases of posterior placenta previa, because of the difficulties in proper identification of the lower uterine segment.

Transvaginal sonography (TVS) is considered as gold standard for localization of the placenta and diagnosis of placenta previa. When the diagnosis of placenta previa is clinically suspected or remains uncertain or equivocal by TAS, TVS clarifies the situation and confirms the diagnosis [15]. TVS is safe, is more accurate than TAS, does not increase the chance of bleeding, does not require a full bladder, and has better diagnostic accuracy with posterior placenta previa [16]. In addition, TVS changes the diagnosis made by TAS in 26 % of cases. The angle between the transvaginal probe and the cervical canal is such that the probe does not enter the cervical canal. Some advocate insertion of the probe no more than 3 cm for visualization of the placenta. Placenta previa is diagnosed on transvaginal scan when the leading placental edge is less than 3 cm away from the internal os. As an alternative to TVS, especially when instrumentation of the vaginal canal is a concern, experts in USG can utilize the transperineal approach to diagnose placenta previa. This method could also be used as a complimentary to TAS [17].

Magnetic resonance imaging (MRI) has been suggested as a safe, alternative, and most accurate method to diagnose placenta previa. The high cost limits its availability. A large trial determining the efficacy and safety of the use of MRI during pregnancy has not been performed, and further investigation is required. MRI is not widely available or used, and at the moment it is relevant only in cases of inconclusive USG findings. It is suggested that MRI may help in confirming the diagnosis of an invasive placenta and identify organ involvement associated with placenta percreta [18].

Management

The management of placenta previa is guided by the principle of expectancy: to allow the pregnancy to progress to as close to term as possible so that delivery can be undertaken.

Incidental Diagnosis of Placenta Previa

Because of the possibility of migration of the placenta to the upper uterine segment in more than 90 % of cases, expectant management is generally followed. The length by which the placenta overlaps the internal os at 18–23 weeks is highly predictive for the persistence of placenta previa [19, 20]. The likelihood of a previa persisting until term increases if the previa is complete, if it is present at a later gestational age, or if there is a history of cesarean delivery. The length by which the placenta overlaps the internal os at 18–23 weeks is highly predictive for the persistence of placenta previa. If the overlap is less than 1.5 cm (0.6 in.) at 18–23 weeks, placenta previa typically resolves, and if the overlap is 2.5 cm (l in.) or greater at 20–23 weeks, persistence to term is likely. The likelihood of a previa persisting until term increases if the previa is complete, if it is present at a later gestational age, or if there is a history of cesarean delivery [21].

Based on the aforesaid information, a conservative approach is logical and usually followed at home. During the interim period of expectancy, management is based on the following principles:

- Strenuous work is avoided.
- Sexual intercourse is avoided.
- Advised to attend hospital, if there is any bleeding.
- Regular intake of hematinics is ensured and maintained.
- Placental location is again reevaluated by USG.

If placenta previa is still present by repeat USG through transvaginal route, the same precautionary measures are followed. If placenta previa persists beyond 32–34 weeks, migration to the upper segment by term is unlikely. Although rescanning is usually followed in most of the centers, there is no evidence that rescanning "at-risk" patients in the third trimester reduces adverse fetal or maternal outcome from placenta previa. Randomized trials addressing this issue are needed. Rescanning

is definitely indicated in cases of persistent malpresentation and/or vaginal bleeding, and TVS is preferred. Waiting beyond 37 weeks is not likely to benefit the fetus or mother. CS usually is scheduled at a gestational age that will maximize the likelihood of fetal maturity and minimize the risk of hemorrhage that may result from the normal onset of uterine contractions.

Because placenta previa may resolve close to term, it is recommended that no decision on mode of delivery be made until after USG at 36 weeks. Women whose placental edge is 2 cm or more from the internal os at term can expect to deliver vaginally unless heavy bleeding ensues. The vaginal delivery should be attempted in a facility capable of moving the patient rapidly to cesarean delivery if necessary. Although not universally followed, documentation of pulmonary maturity by amniocentesis in women with a nonbleeding placenta previa at 36–37 weeks is performed in some centers prior to scheduled cesarean delivery.

If Presents with Bleeding

Initial management of APH (as discussed in the previous section) is instituted without delay. Depending on the severity or persistence of bleeding, gestational age, and maternal and fetal condition, the options for subsequent management are immediate delivery, expectant management, and termination of pregnancy.

The following clinical situations as shown in Table 27.9 demand immediate delivery by CS.

The mode of delivery depends on the grade of placenta previa and state of the cervix. The options are immediate CS and examination in the operation theater (OT), with or without anesthesia ("double setup"). Cesarean route is the preferred method of delivery in most of the situations (fetal malposition or malpresentation, fetal heart rate (FHR) abnormality, and major degree placenta previa) and the only option in the presence of profuse bleeding. In case of a low-lying or marginal placenta previa, the descending fetal head may "tamponade" the bleeding placental edge (thereby reducing bleeding from the separated placental edge) and permit vaginal delivery.

The "double setup" examination (Table 27.10) is indicated in cases of grade I or grade II anterior placenta previa or when the USG findings for localization of the placenta are inconclusive.

Prerequisites for "double setup" examination include:

- Intravenous (IV) lines started.
- Cross-matched blood is available.
- Examination is performed in the OT.
- An experienced obstetrician should perform the examination.
- Second obstetrician must be scrubbed and ready to operate.
- Anesthetist must be present in the OT.
- Pediatrician must be present in the OT.
- Operation theater nurse scrubbed with tray for CS ready.

Mode of Anesthesia

Majority (85 %) of the anesthetists, however, prefer regional anesthesia, if the examination is performed in cases of minor degree and only a minority (l5 %) in cases of major degree placenta previa. The advantage of general anesthesia (GA)

Table 27.9 Indications for immediate delivery

Deteriorating condition of the mother
Persistent heavy bleeding
Gestational age more than 36 weeks
Estimated fetal weight more than 2,500 g
Fetal distress in a viable fetus
Contractions that do not respond to medication

Table 27.10 Double setup

Sterile speculum examination is done to see the cervix. The cervix is partly dilated and the placental tissue is visible; placenta previa is confirmed and delivery is planned
If the cervical os is not dilated, vaginal fornices are palpated carefully
Spongy tissue felt – placenta previa confirmed
Fetal head felt – placenta previa excluded
If the diagnosis is still in doubt, careful digital examination is done through the cervix
Soft tissue felt within the cervix – placenta previa confirmed
Fetal parts and membranes are felt both centrally and marginally – placenta previa is excluded

is proper relaxation of the patient making examination easier and rapid progression to CS, if necessary. The main disadvantage of GA is to wake the patient from the effects of anesthesia, if vaginal delivery is allowed. A second anesthesia with the associated increased risk is required, if the patient needs CS, during the course of vaginal delivery, or for management of adherent placenta. The disadvantage of epidural anesthesia is hypotension, which may worsen the effects of hypovolemia, if bleeding is severe or persistent or volume replacement is inadequate. A compromised approach is drawing the drugs for induction of GA and preparing the anesthetic machines and instruments ready before the procedure. With such preparation and in experienced hands, the interval between examination and delivery could be short.

Keeping everything ready for CS, a careful examination was then undertaken to determine whether placental tissue could be seen or felt near the cervix, and the method of delivery was decided by the findings. This procedure is of significance in the developing countries, where USG is not readily available or the report is not reliable. The primary reason for performing this examination is to confirm or exclude placenta previa to explore the possibility of vaginal delivery.

If the placenta is felt anteriorly, but the edge does not reach up to the os, the membranes are ruptured in preparation for vaginal delivery. Persistent bright red bleeding following rupture of the membranes or any brisk bleeding anytime during the procedure indicates abandoning the procedure and to proceed for immediate CS. Today, the "double setup" examination has largely been replaced by reliable ultrasound localization of the placenta.

Expectant Management

Provided the initial episode of bleeding resolves and the fetomaternal condition is stable, it is reasonable to delay delivery until fetal maturity is attained or any subsequent bouts of significant bleeding supervene. Although not universally followed, in some centers, expectant treatment is also considered even when the initial bleeding is severe and when the bleeding either stops or slows down. The goal of this expectant approach is to improve perinatal outcome by prolongation of pregnancy, and several groups had reported that. Bed rest is instituted with strict avoidance of any inciting factors of bleeding. Measures, including blood transfusion and administration of hematinics, are taken to raise hemoglobin status to prepare the patient to withstand any further significant rebleeding. The aim is to maintain maternal hemoglobin at least 10 g% or hematocrit 30 %. In addition to bed rest, steroids are usually given to hasten fetal lung maturity [22].

In Rh-negative women, an injection of Rh immune globulin is also administered after performing Kleihauer-Betke test to determine the appropriate dose [23].

In all cases of expectant management, two units of blood must be available at all times. During the expectancy period, some interventional measures were tried and/or adopted to improve maternal and fetal outcome.

Elective Cervical Cerclage

To prolong pregnancy and prevent iatrogenic prematurity due to bleeding, cervical cerclage has been proposed and tried [24]. The Cochrane meta-analysis found that cerclage decreased the risk of premature birth before 34 weeks (relative risk, 0.45; 95% confidence interval, 0.23–0.87); however, it is recommended that additional studies of cerclage be performed before this clinical practice is introduced [25].

To summarize, cervical cerclage may reduce very premature births, although the evidence is not very strong.

Tocolytic Agents

Tocolytic agents may be used safely to prolong gestation if vaginal bleeding occurs with preterm contractions [26]. Although studies using tocolytic agents show a trend toward increased frequency of bleeding episodes, neither is this bleeding significant nor does it increase the requirement of blood transfusion.

Uterine contractions in the presence of placental previa may be due to abruption (associated in 10 % of cases), and due consideration should be given to exclude abruption prior to administration of a tocolytic agent.

At present, there is inadequate evidence to suggest that either inpatient or outpatient care leads to a superior outcome. On the contrary, outpatient management is appropriate for selected patients who do not have active bleeding and who can rapidly access a hospital with operative labor and delivery services. This approach is applicable to only grade I to III placenta previa and asymptomatic grade IV placenta previa [27]. However, in clinical practice, frequently a combination of inpatient and outpatient management is undertaken. For a particular patient, the decision is taken by analyzing the following factors shown in Table 27.11.

Termination of Pregnancy

Cesarean section is the recommended method of delivery in nearly all cases of placenta previa except those cases of grade I placenta previa, where the distance between the leading placental edge and the cervix is at least 20 mm to allow vaginal delivery. CS, as an optional method of delivery, is also contributed by increased incidence of malpresentation and other obstetric factors.

In cases of anterior placenta previa, it is often beneficial to perform a scan immediately prior to CS, to localize the placenta precisely, and to look for any "placenta-free window" so as to plan the incision at the lower uterine segment during CS. In the absence of such "placenta-free window," the options for access to the fetus are either transplacental approach or "classical" incision in the upper uterine segment. The former approach requires speed and may cause significant fetal blood loss. The latter approach may be associated with undue delay in delivery of the fetus and more troublesome bleeding from a partially separated placenta with resultant fetal blood loss and anoxia, along with the potential risk of scar rupture in subsequent pregnancy. However, a lower segment approach is preferred by most obstetricians. Because of inevitable fetal blood loss, cutting or tearing through the placenta is avoided by many. Less retractile nature of the lower uterine segment provokes increased intraoperative bleeding through torn placental sinuses. To achieve hemostasis, the following measures (Table 27.12) can be undertaken with variable degree of success.

Recently, prothrombin complex and recombinant factor VII are being utilized to control massive hemorrhage with placenta previa.

Women with a history of previous cesarean delivery and placenta previa or a placenta located at the site of the previous incision are at a higher risk of morbidly adherent placenta and so should be evaluated accordingly with color flow Doppler imaging. The method has a positive predictive value for detection of placenta accreta of 87.5 % [29]. MRI of the pelvis may help to confirm the diagnosis of an invasive placenta and delineate organ involvement in women with a placenta percreta [30]. Nine to 10 % of cases of placenta previa are associated with placenta accreta, an

Table 27.11 Factors affecting outpatient management of placenta previa cases

Each case is given due importance and individualized approach is adopted
History of bleeding – stopped/continuing
Grade of placenta previa
Patient's social circumstances, distance of residence from the hospital, transport facility, manpower support
Patient's wishes
Obstetrician's preferred practice

Table 27.12 Intracesarean methods to reduce blood loss

Bleeding sinuses oversewn with atraumatic sutures
Direct pressure with warm packs. But if these packs are left in situ during closure of the uterus, bleeding might continue and remain concealed
Administration of oxytocics with intramyometrial injection of prostaglandin F2a
Horizontal and vertical compression sutures were tried with some success [28]
Uncontrollable bleeding is tackled by ligation of the uterine artery and internal iliac artery
Hysterectomy may be required as a last resort to save the woman

abnormally firm attachment of the placenta to the wall of the uterus. Placenta accreta prevents separation of the placenta from the uterine wall at the time of delivery and can induce severe bleeding thereby increasing the need for peripartum hysterectomy (>90 %). This possibility needs to be discussed with the patient, prior to CS. More than 50 % of patients with placenta accreta require blood transfusion. However, hysterectomy can be avoided by employing a conservative approach, wherein the placenta is left in situ followed either by internal iliac artery ligation or uterine artery embolization or by adopting a medical approach with administration of systemic methotrexate.

When possible, the procedure should be performed electively with the following preparations: adequate venous access, ready availability of blood and necessary medications, and discussion about the possible need of hysterectomy in dire situations. An experienced surgeon, preferably a consultant, should perform the operation or at least be readily available. Available data suggests that the risk of bleeding may be more with GA, which is contrary to the commonly held view. Accumulated evidence also indicates that regional anesthesia is a safe alternative to GA in both elective and emergency cesarean deliveries in cases of placenta previa.

Summary: Placenta Previa

- Transvaginal sonography is the most reliable diagnostic method and without any risk too.
- Postcesarean pregnancy increases the risk of placenta previa and also adherent placenta.
- Routine anomaly scan at 20–22 weeks has a high false-positive and 7 % false-negative rate for diagnosis.
- "Double setup" procedure is gradually being replaced by sonographic localization of placenta.
- Management depends on degree of placenta previa, gestational age, and fetal presentation.
- Expectant treatment in preterm pregnancy improves the perinatal outcome.
- Cesarean section is the preferred mode of delivery in major degree placenta previa and to be performed by experienced obstetrician.

Abruptio Placentae (Fig. 27.4)

Definition

Premature separation of a normally situated placenta curing the antenatal or intrapartum period of pregnancy is known as abruptio placentae. Approximately, 20 % of all cases of APH are due to abruption.

Incidence

Abruption has been estimated to occur in 6.5 pregnancies per 1,000 births and up to 1.5 in all pregnancies [31, 32]. Many cases of abruptio remain unrecognized until delivery, and in one study histological examination of the placenta reveals that the incidence is as high as 4.5. Thus, the criteria used for diagnosis is responsible for reported wide variation in incidence (0.49–1.8 %). Approximately, 50 % of placental abruptions occur before 36 weeks gestation, resulting in adverse outcomes secondary to prematurity [33].

Causative Associations

The cause of abruption is unknown in most cases. Within few hours, however, the cause may become obvious. Various risk factors are associated with placental abruption (Table 27.13).

The risk factors include chronic hypertension, preeclampsia, thrombophilias, abdominal trauma and abruption in a previous pregnancy [34], and

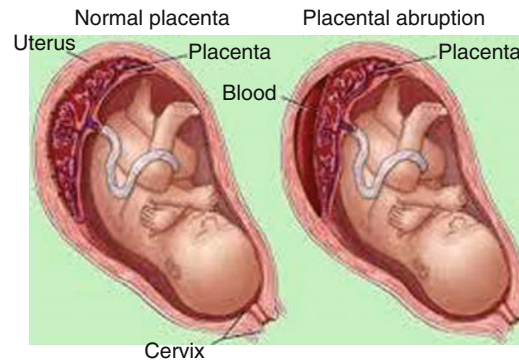

Fig. 27.4 Normal placenta and abruptio placentae

Table 27.13 Risk factors for abruptio placentae

Hypertension – the most common in 44 % of cases
Cigarette smoking
Trauma (road traffic accident (RTA), domestic violence)
Sudden decompression of the uterus
Short umbilical cord
Alcohol consumption
Cocaine abuse
Retroplacental fibroid
Amniotomy or amniocentesis
Severe intrauterine growth retardation (IUGR)
Advanced maternal age
Prolonged preterm premature rupture of membranes (PROM)
Abruptio in previous pregnancy
Thrombophilia
Chorioamnionitis
External cephalic conversion
Hyperhomocysteinemia
Circumvallate placenta
Increased alpha-fetoprotein level

tobacco or cocaine use [35]. Chronic hypertension is associated with a threefold increase in the risk of abruption. Although maternal hypertension is considered a risk factor, there is no consensus as to whether hypertension precedes abruption or vice versa. The other independent associations of placental abruption include severe fetal growth restriction, prolonged rupture of the membranes, chorioamnionitis (infection of placenta and membranes), hypertension (including preeclampsia, nonproteinuric pregnancy-induced hypertension, and preexisting hypertension), cigarette smoking, advanced maternal age, and unmarried status [36].

Abruptio in cases of preterm premature rupture of membranes (PPROM) typically presents with bleeding followed by dribbling. Trauma, notably road traffic accidents and even domestic violence, may also cause abruption. Sudden uterine decompression after rupture of membranes or delivery of a first twin may precipitate placental abruption [37]. Placental abnormalities (especially circumvallate placenta) and increased level of alpha-fetoprotein are also associated with abruption. Placental abruption is twice as common in twin than singleton pregnancies [38]. The risk of placental abruption in subsequent pregnancies is significant varying from 6 % to 16.7 % after one episode and increases to 25 % after two such episodes. In one study, the incidence increased by at least tenfold (incidence 4–5 %) [39].

The association of folic acid deficiency with abruption was not confirmed by any large prospective studies. On the other hand, the associations between abruption and specific thrombophilias (such as factor V Leiden mutation, prothrombin gene mutation, hyperhomocysteinemia, activated protein C resistance, antithrombin III deficiency, and anticardiolipin immunoglobulin G antibodies) have been reported in different literatures, and this risk may be independent of the presence of preeclampsia. The presence of thrombophilias may also influence the severity of abruption. The association between abruption and thrombophilias, however, had not been uniformly documented in all the related studies.

Classification (Fig. 27.5)

Broadly, abruptio is classified into two groups:

1. *Revealed* – where obvious bleeding is noted in the lower genital tract
2. *Concealed* – where entire blood is contained inside the gravid uterus without any manifestation of external bleeding

Abruptio is concealed in 20–35 % and revealed in 60–65 % of cases [40]. The concealed type is more dangerous and associated with more severe maternal and fetal complications.

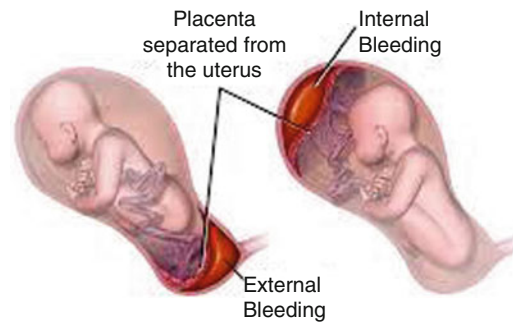

Fig. 27.5 Revealed and concealed hemorrhage in abruptio placentae

A more precise classification of abruption based on the extent of separation (partial vs complete) and location of separation (marginal vs central) is as follows:

Class 0: is asymptomatic. Diagnosis is made retrospectively by finding an organized blood clot or a depressed area on a delivered placenta.

Class 1: is mild and represents approximately 48 % of all cases. Characteristics include the following:
- No vaginal bleeding to mild vaginal bleeding
- Slightly tender uterus
- Normal maternal blood pressure (BP) and heart rate
- No maternal or fetal compromise

Class 2: is moderate and represents approximately 27 % of all cases. Characteristics include the following:
- No vaginal bleeding to moderate vaginal bleeding
- Moderate-to-severe uterine tenderness with possible tetanic contractions
- Maternal tachycardia with orthostatic changes in BP and heart rate
- Fetal compromise or distress
- Hypofibrinogenemia (i.e., 50–250 *mg/dL*)

Class 3: is severe and represents approximately 24 % of all cases (0.2 of all pregnancies). The characteristics are:
- No vaginal bleeding to heavy vaginal bleeding
- Very painful tetanic uterus with stony hard consistency
- Maternal shock
- Hypofibrinogenemia (i.e., <150 mg/dl.) and coagulopathy
- Fetal death

Fetomaternal Complications

The fetomaternal complications are dependent upon severity of hemorrhage, extent of placental separation, health status of mother and fetus, gestational age, and effectiveness of interventions.

Maternal Risks

Maternal mortality: Placental abruption is a recognized cause of maternal death [41], especially in resource-poor settings and developing countries. The maternal mortality rate is approximately 1 % [42].

Hypovolemic shock: As a result of severe maternal blood loss, hypovolemic shock is the major immediate maternal risk. Because of concealed bleeding, blood loss is often underestimated.

Coagulopathy/disseminated intravascular coagulation: There may also be clinical and hematological evidence of disordered blood clotting as thromboplastin are released by placental damage and coagulation factors are consumed in the enlarging retroplacental clot at a rate that is faster than the body's ability to replace them.

Postpartum hemorrhage: It can result from coagulation failure or from couvelaire uterus (a condition where blood sips into the myometrium and impairs its ability to contract).

Renal failure: Acute renal failure may result from either hypovolemia or disseminated intravascular coagulation (DIC) and may be seen later in the forms of either acute tubular or cortical necroses.

Multiple organ failure: Ischemic necrosis of distal organs (e.g., hepatic, adrenal, pituitary) is the result of severe prolonged hypotension.

Fetal Risks

Perinatal mortality rate of abruptio is 119 per 1,000.

It varies from 4.4 % to 67.3 % depending on the severity of the condition and neonatal care facilities. More than 50 % of perinatal deaths are stillbirths. Neonatal death occurs in 10–30 % of cases. Approximately, 50 % of placental abruptions occur before 36 weeks of gestation, resulting in adverse outcomes secondary to prematurity.

The causes of *perinatal deaths* include prematurity, congenital malformation, intrauterine growth restriction (IUGR), and fetal hypoxia. DIC may result from the release of thromboplastin into the maternal circulation with pla-

cental separation. This occurs in about 10 % of abruptions indicating severe abruption and is more common with fetal death [43].

Fetal growth restriction is noted in 80 % of infants born before 36 weeks of gestation.

The rate of *congenital malformation* is increased by twofold to threefold. Significant fetal bleeding may cause *neonatal anemia*.

Clinical Presentation

Placental abruption may present with a variable combination of vaginal bleeding, abdominal pain, uterine contraction, shock, or fetal distress depending on the degree of separation of the placenta (Table 27.14).

Mild placental detachment: It may not be demonstrable on ultrasound as the blood clot is not easily distinguishable from the placenta.

Moderate placental detachment and hemorrhage: At least one-quarter of the placenta has become detached and less than 1,000 ml of blood lost. There may be abdominal pain and a tender uterus, the mother may be in shock, and the fetus may be hypoxic and may show abnormal heart rate patterns.

Severe placental detachment and hemorrhage: At least 1,500 ml of blood lost, shock usual, and uterus firm to hard and very tender. The fetus is almost always dead. Hypotension in one-third of cases, but may be normal in spite of shock. Coagulopathy is common.

Table 27.14 Clinical presentation of abruptio placentae

Vaginal bleeding may or may not be obvious ("revealed" or "concealed" variety)
Variable degree of pain over the uterus is a prominent feature and is continuous in nature
Uterine contractions may start and cause additional, intermittent, pain
Faintness and collapse with or without signs of shock may occur
Typically, the uterus is extremely hard and tender, without any relaxation
Fetal parts are difficult to palpate
Fetal heart sounds may not be audible if death has occurred

The presenting symptoms of abruption are as follows:

- Vaginal bleeding – 70–80 % (characteristically dark and nonclotting).
- Abdominal or back pain and uterine tenderness – 70 %.
- Fetal distress – 60 % (in grade I and II cases).
- Abnormal uterine contractions (e.g., hypertonic, high frequency) – 35 %.
- Uterine contractions are often difficult to differentiate from pain of abruption. Nearly 50 % of the patients with abruption are in established labor.
- Fetal death (in grade III it is inevitable by definition) occurs in 15 % of cases.

Pathogenesis

It is thought that abruption is caused by degenerative changes in the spiral arterioles, leading to decidual necrosis. Following this, the vessels can rupture and bleeding ensues and ultimately forms a retroplacental clot. Placental abruption can be a self-extending process with the accumulating blood clot causing more separation and thus more hemorrhage until the edge of the placenta is reached. After this, blood can escape through the potential space between the chorion and the decidua until it reaches the cervix. Blood can also reach the amniotic cavity (by disrupting the placenta, producing blood-stained amniotic fluid) and the myometrium (causing the bruised, so-called couvelaire uterus). There is usually severe fetal hypoxia associated with sizeable placental separation, and sudden fetal death is common.

Diagnosis

Placental abruption is essentially a clinical diagnosis. Features of moderate-to-severe abruption are suggestive. In mild cases, diagnosis is often suspected and confirmed after delivery by demonstration of a retroplacental clot indenting the maternal surface of the placenta. Placental abruption can cause fetomaternal

hemorrhage, so Kleihauer test could be diagnostic in mild and silent cases.

Ultrasound imaging has a much smaller role in the diagnosis. The sensitivity of USG for detection of abruptio is only 24 % [44]. USG may not be helpful in diagnosing acute severe abruption, as the acoustic characteristics of a fresh retroplacental clot are similar to those of placenta and differentiation is difficult. In less severe cases where the pregnancy is allowed to continue, the clot becomes hyperechogenic within 1 week and sonolucent within 2 weeks and therefore more obvious by USG. Ultrasonography helps in excluding placenta previa and fetal congenital abnormality in addition to detecting fetal viability, number, presentation, estimated fetal weight, and gestational age. The cases managed expectantly can be monitored with USG by determining the size of the hematoma, its location, and change in size over time. This information may help to decide the time of delivery in mild cases managed expectantly.

Management

The management depends on:

- Severity of the condition
- Maternal condition
- Fetal condition
- Gestational age
- Associated complications

The management can be divided into:

I. *General measures*
II. *Specific measures*

General management is already discussed elsewhere.

Specific measures to be followed are:

- Immediate delivery
- Expectant management
- Management of complications

The traditional, *main principles of clinical care* of a woman with placental abruption include:

- Adequate maternal resuscitation
- Assessment of fetal condition
- Adequate analgesia for pain relief
- Early delivery
- Adequate blood transfusion
- Monitoring of maternal condition

Early Delivery

Early delivery is lifesaving for the fetus. In one case series, 30 % of perinatal deaths occurred within 2 h of admission [45]. A decision-to-delivery interval of 20 min or less resulted in improved neonatal outcomes in a case-control study of severe abruption. Acute blood clots and the placenta are hyperechoic on USG and are difficult to distinguish from one another. So, the definitive management should never be delayed for confirmation of diagnosis by USG.

If the fetus is already dead, as is often the case, vaginal delivery should be the goal. It has been suggested that in some cases of severe abruption, high levels of fibrin degradation products might inhibit uterine contractions and make vaginal delivery difficult to achieve. This might contribute to atonic PPH as well. Most women with abruption and DIC deliver vaginally. However, if surgical intervention is required at any point of time, measures should be taken to rapidly correct the coagulation defect by transfusion of fresh blood, fresh frozen plasma (FFP), and cryoprecipitate. To achieve vaginal delivery, amniotomy is usually preferred. It not only accelerates labor, but is also believed to reduce the incidence of coagulopathy by reducing intra-amniotic pressure and thereby minimizing the entry of tissue thromboplastin into the maternal systemic circulation. Augmentation with oxytocin may be necessary in some cases. To identify fetal distress early, continuous fetal monitoring should be per-

formed during vaginal delivery. A nonreassuring fetal heart tracing necessitates rapid, usually cesarean, delivery. As 15.4 % of live born infants of abruption do not survive, choosing a particular method of delivery in cases of live fetus may not always be very easy [46]. The study that shows 52 % perinatal mortality following vaginal delivery as compared to 16 % following CS favors CS as the preferred option [47]. A shearing force produced by uterine contraction, during vaginal delivery, may aggravate the chance of further placental separation. So, even an apparently normal fetus runs the risk of compromised oxygen supply during labor. In fact, it has been recommended that if the baby is alive and the gestation not so preterm as to make fetal survival extremely unlikely, delivery should be by CS [48].

Initial management includes rapid stabilization of maternal cardiopulmonary status and assessment of fetal well-being. Prompt treatment and monitoring of the mother is essential. Maternal vital signs (pulse, BP, respiration rate) along with central venous pressure (CVP), urine output, vaginal bleeding, etc. are monitored continuously. Treatment of hypovolemic shock, if needed, should be initiated early and maintained vigorously with appropriate monitoring. In the absence of fetal heart sound, it is anticipated that a massive-concealed bleeding had already taken place and traditional teaching advises transfusion of at least two units of blood. Coagulation parameters are also monitored in this situation and also when coagulopathy is suspected. As some degree of coagulopathy is expected in about 30 % of severe cases of placental abruption, quick identification and treatment of the condition are important. The best treatment for DIC, as a complication of placental abruption, is immediate delivery along with ancillary measures suggested by the hematologist. Maternal stabilization requires serial evaluation of the hematocrit and coagulation studies to determine whether DIC is present. Fetal condition is also monitored intermittently or continuously, as required. Because the unpredictable nature of abruption does not allow for controlled trials, the management remains empiric. The Cochrane review found no randomized controlled trials assessing interventions for placental abruption that met inclusion criteria.

Expectant Treatment

In selected patients, with an aim to improve fetal maturity, expectant treatment is considered provided the following criteria are satisfied:

- Vaginal bleeding is slight.
- Abdominal pain is mild and localized.
- Uterus relaxed and not irritable.
- Fetal heart rate is normal.
- Maternal condition is hemodynamically stable.

The treatment is continued with appropriate monitoring of fetal condition clinically and by investigations (cardiotocography (CTG), biophysical profile, and Doppler velocimetry) as available.

The timing of delivery was decided by:

- Any further episodes of vaginal bleeding
- Gestational age
- Fetal condition
- Neonatal care facilities

Despite the lack of evidence, induction of labor is often advocated in patients at term because of the possible deterioration in placental function in patients even in absence of any acute or chronic fetal compromise being documented. For expectant treatment, routine admission of these patients lacks any concrete evidence. If any retroplacental clot is detected by USG, serial scan may help to monitor the size and change in echogenicity of the clot.

Any deterioration of fetal condition demands immediate delivery. Some cases of mild abruption may be associated with labor. In such cases, it is difficult to determine what precedes the other. Although tocolysis is generally contraindicated, the only possible role of tocolysis is in cases of mild abruption before 34 weeks of gestation, to allow administration of corticosteroids [49].

Prevention

Randomized controlled trials of sufficient power are required to assess interventions (diet, vitamin supplements, and antithrombotic therapy) to prevent abruption. By reducing cigarette smoking, drug abuse, and domestic violence, it is possible to reduce the incidence of abruptio. Adopting this approach would certainly provide other health benefits. The "Magpie" Trial demonstrated a reduction in the incidence of abruptio with intrapartum treatment of magnesium sulfate in cases of preeclampsia [50].

Summary: Abruptio Placentae

- A variety of associations are noted.
- Diagnosis is essentially clinical.
- Ultrasonography has a small role in diagnosis. Sonography primarily is utilized for excluding placenta previa and monitoring retroplacental clot and fetal conditions during expectant treatment.
- Management depends on grade. Vaginal delivery is performed in dead fetus and CS is performed in live fetus.
- Correction of coagulopathy is essential prior to embarking on delivery.
- Major risk factors include intrauterine device (IUD), maternal DIC, and renal failure.
- Recurrence risk is 6–17 after one episode and almost 25 after two episodes.

Undetermined Antepartum Hemorrhage

It is the most common cause of APH, and the diagnosis is essentially made by exclusion of placenta previa and abruptio placentae and other obvious causes. The patient usually presents with painless bleeding which is usually mild in nature and settles spontaneously [51]. Marginal sinus rupture appears to be the most common cause of bleeding in cases of undetermined APH [52]. The bleeding is usually painless.

Causes

Although the cause remains unknown in majority of the situations, in a proportion of these cases, the cause may become evident later on, and it includes circumvallate placenta, marginal sinus rupture, "show," trauma, cervicitis, genital tract tumors, genital infections, vulval varicosities, and vasa previa. Unrecognized cases of minor placenta previa or mild abruption, diagnosed only after delivery, are also included in the list of causes of undetermined APH.

Risks

The main concern about unclassified vaginal bleeding is their association with an increased risk of preterm delivery and a small increase in the risk of fetal congenital abnormality.

Management

Each case is managed with individualistic approach. Anti-D prophylaxis is offered to all Rh-negative women. Once the diagnosis is reasonably established by exclusion of more important causes (not necessarily more common), the management depends on the following conditions:

Nature of bleeding: Persistent or recurrent
Severity of bleeding: Mild or heavy
Gestational age of the fetus: Term or preterm
Fetal condition: Good or distressed
Fetal congenital abnormality: Present or not
Presumed cause: If possible to ascertain

The management options are either immediate delivery or expectant. The mode of delivery depends on the following conditions:

- State of the fetus
- Fetal lie and presentation
- Cervical condition
- Any other associated high-risk factor

Once the significant causes of APH are excluded, there is no obvious advantage in managing such patients in the hospital. The patient can be monitored with appropriate advice and to attend hospital immediately should any emergency arise. The general tendency is to discharge the patient from hospital once the bleeding stops for at least 24 h.

Vasa Previa

Introduction

Vasa previa is the velamentous insertion of the umbilical cord into the placental membranes overlying the lower uterine segment. As a result, the fetal vessels appear between the cervix and the presenting part. Despite being very rare in incidence (0.03 %), associated very high perinatal mortality made rapid intervention essential for fetal survival [53].

Risk Factors

Risk factors for vasa previa include in vitro fertilization, placenta previa, and bi-lobed and succenturiate-lobed placentas [54].

Presentation

Vasa previa typically manifests as unanticipated bleeding at the time of amniotomy or spontaneous rupture of membranes.

Diagnosis

The condition is considered when fetal vessels are seen or felt during vaginal examination. Rarely, vessels are palpated in the presenting membranes, prohibiting artificial rupture of the membranes and vaginal delivery. Antenatal diagnosis is possible by color Doppler visualization of fetal vessels by endocavity USG. CTG shows characteristic sinusoidal pattern or baseline fetal tachycardia or bradycardia.

The hemorrhage is fetal and so exsanguinations can occur rapidly [55]. Alkali denaturation test confirms the fetal source of bleeding by taking a blood sample from the vaginal vault [56].

Management

Suspicion, diagnosis, and immediate delivery are the crucial steps. Immediate delivery by lower segment cesarean section is the only way to salvage the fetus. Delivery should not be deferred for confirmation of fetal blood in women with severe hemorrhage or when CTG is nonreassuring.

Management guidelines for vasa previa:

- Emergency cesarean section in the presence of bleeding.
- Elective cesarean section prior to the onset of labor.
- Admission to hospital with appropriate neonatal facilities from 28 to 32 weeks of gestation.
- Corticosteroids for fetal lung maturity.
- Laser ablation in utero may be tried in well-equipped centers.

Prevention

There are no strategies for primary prevention of vasa previa. Theoretically, the complications of vasa previa could be avoided by antenatal screening of high-risk women and by performing CS at 37–38 weeks when vasa previa is present. Screening can be carried out with transvaginal color flow Doppler to identify the presence of vessels in the fetal membranes. Although it has been suggested for women at increased risk [57], there is no evidence that screening in a general population changes outcomes, and because the condition is rare (1 diagnosis per 5,215 screenings), this approach is also cost prohibitive [58].

Fetal Risks

Studies demonstrate a 33–100 % rate of perinatal mortality secondary to vasa previa.

Summary: Unclassified Bleeding

- Diagnosis is made by exclusion of other causes.
- Vasa previa must be confirmed or excluded.
- There is an increase in the overall risk of adverse perinatal outcome.

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HELLP Syndrome

28

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Introduction

The HELLP syndrome is a serious complication in pregnancy characterized by *h*aemolysis, *e*levated *l*iver enzymes and *l*ow *p*latelet count occurring in 0.5–0.9 % of all pregnancies and in 10–20 % of cases with severe preeclampsia.

It was first described by Weinstein in 1982. The acronym is for hemolysis (H), elevated liver enzymes (EL) and thrombocytopenia (low platelet count – LP). It is more frequent in older, multiparous Caucasian women. In 70 % of the cases, the disorder is diagnosed in the antepartum period: 10 % before 27 weeks of gestation, 70 % between 27 and 37 weeks of gestation and 20 % after 37 weeks of gestation. In 30 % of cases, it is diagnosed in the postpartum period. The risk of recurrence in a subsequent pregnancy is estimated at 19–27 %.

Diagnosis

A wide range of symptoms none of which are diagnostic are right upper abdominal quadrant or epigastric pain, nausea and vomiting seen in 30–90 % cases. The upper abdominal pain may be fluctuating, colic-like. Many patients report a history of malaise some days before presentation. Up to 30–60 % of women have headache; about 20 % have visual symptoms and 5 % have jaundice. However, women with a HELLP syndrome might also have unspecific symptoms or subtle signs of preeclampsia or nonspecific viral syndrome-like symptoms. Women with partial HELLP syndrome have fewer symptoms and develop less complication than those with the complete form. However, a partial or incomplete HELLP syndrome may develop to a complete form of the disorder [1].

Classification

In the past diagnostic criteria for HELLP syndrome were variable and led to inconsistent diagnosis. Two classifications for the HELLP syndrome are commonly used. The Tennessee System classification proposed by Sibai is based on the assessment of the following parameters: AST>70 UI/L, LDH >600 UI/L and thrombocytes <100,000/μL. Accordingly, there are two forms: *complete* (all elements present) and *partial* HELLP syndrome (one or two elements present) [2].

The Mississippi classification as of 2006:

Class 1, severe thrombocytopenia (platelets ≤50,000/μL), evidence of hepatic dysfunction (AST [aspartate aminotransferase] and/or

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ALT [alanine aminotransferase] ≥70 IU/L) and evidence suggestive of hemolysis (total serum LDH ≥600 IU/L);

Class 2, similar criteria except thrombocytopenia is moderate (>50,000 to ≤100,000/μL); and

Class 3, includes patients with mild thrombocytopenia (platelets >100,000 but ≤150,000/μL), mild hepatic dysfunction (AST and/or ALT ≥40 IU/L), and hemolysis (total serum LDH ≥600 IU/L) [3–5] (Table 28.1).

Diagnostic Criteria for HELLP Syndrome

HELLP syndrome is diagnosed when all three of the following are present:

1. *Hemolysis*
   (i) Increase in LDH
   (ii) Red cell fragmentation leading to schistocytes and Burr cells
   (iii) Haemoglobinuria
   (iv) Decrease in haptoglobin level
   (v) Elevated serum bilirubin (≥1.2 mg/dL)
   (vi) Significant drop in haemoglobin levels

   Haemolysis is due to a microangiopathic haemolytic anaemia (MAHA). Red cell fragmentation caused by high velocity passage through damaged endothelium. Destruction of red blood cells by haemolysis causes increased serum lactate dehydrogenase (LDH) levels and decreased haemoglobin concentrations. Liberated haemoglobin is converted to unconjugated bilirubin in the spleen or may be bound in the plasma by haptoglobin. The haemoglobin-haptoglobin complex is cleared quickly by the liver, leading to low or undetectable haptoglobin levels in the blood, even with moderate haemolysis. Low haptoglobin concentration (<1 to <0.4 g/L) can be used to diagnose haemolysis and is the preferred and more specific marker of haemolysis.
2. *Elevated liver enzymes*
   In 30 % of cases, a moderate increase in gamma glutamyl transferase, alkaline phosphatase and serum bilirubin occurs. Elevation of liver enzymes like asparate aminotransferase (AST) and alanine aminotransferase (ALT) levels are mostly due to liver injury. Plasma glutathione S-transferase-a1 (GST or GST-a1) may provide a more sensitive indicator for acute liver damage than AST and ALT and allow earlier recognition. However, measurement of (GST) is not widely available.
3. *Low platelet count*
   It has to be differentiated from other causes of thrombocytopenia like preeclampsia, gestational thrombocytopenia and immune thrombocytopenic purpura. Decreased platelet count is due to their increased consumption. Platelets are activated and adhere to damaged vascular endothelial cells, resulting in increased platelet turnover with shorter lifespan.

Table 28.1 Diagnostic criteria for HELLP syndrome

HELLP class	Tennessee classification	Mississippi classification
1	Platelets <100,000/μL	Platelets <50,000 μL
	AST> 70 IU/L	AST or ALT >70 IU/L
	LDH >600 IU/L	LDH >600 IU/L
2		Platelets >50,000 and <100,000 μL
		AST or ALT >70 IU/L
		LDH >600 IU/L
		Platelets >100,000 and <150,000 μL
3		AST or ALT >40 IU/L
		LDH >600 IU/L

Pathophysiology

The pathophysiology of HELLP syndrome is ill-defined.

1. Some theorize that, because HELLP is a variant of preeclampsia, the pathophysiology stems from a common source. In preeclampsia, defective placental vascular remodelling during weeks 16–22 of pregnancy with the second wave of trophoblastic invasion into the decidua results in inadequate placental perfusion. The

hypoxic placenta then releases various placental factors such as soluble vascular endothelial growth factor receptor-1 (sVEGFR-1), which then binds vascular endothelial growth factor (VEGF) and placental growth factor (PGF), causing endothelial cell and placental dysfunction by preventing them from binding endothelial cell receptors. The result is hypertension, proteinuria, and increased platelet activation and aggregation [6].

2. Furthermore, activation of the coagulation cascade causes consumption of platelets due to adhesion onto a damaged and activated endothelium, in addition to shearing of erythrocytes as they traverse through capillaries laden with platelet-fibrin deposits. Multiorgan microvascular injury and hepatic necrosis causing liver dysfunction contribute to the development of HELLP. Hepatic ischemia causes hepatic infarction, subcapsular hematomas and intraparenchymatous haemorrhage, which may result in hepatic rupture [7].
3. In HELLP syndrome, high plasma-soluble HLA antigen (sHLA-DR) levels are found. The syndrome can be considered an acute rejection of the fetal allograft [8].
4. The significantly increased levels of tissue plasminogen activator and plasminogen activator inhibitor-1 (PAI-1) in the context of HELLP syndrome compared to normal pregnancy suggest that platelet activation and the alteration of plasminogen activation are involved in the pathogenesis of this syndrome [9, 10].

Symptoms

A wide range of symptoms none of which are diagnostic are right upper abdominal quadrant or epigastric pain, nausea and vomiting seen in 30–90 % cases. The upper abdominal pain may be fluctuating, colic-like. Many patients report a history of malaise some days before presentation. Up to 30–60 % of women have headache; about 20 % have visual symptoms and 5 % have jaundice. However, women with a HELLP syndrome might also have unspecific symptoms or subtle signs of preeclampsia or nonspecific viral syndrome-like symptoms. Women with partial HELLP syndrome have fewer symptoms and develop less complication than those with the complete form. However, a partial or incomplete HELLP syndrome may develop to a complete form of the disorder [1].

Any patient diagnosed with HELLP syndrome should be considered to have severe preeclampsia.

Signs

(i) Mild pallor – sometimes no pallor at all, due to haemoconcentration
(ii) Oedema of feet
(iii) Haematuria – due to thrombocytopenia, deranged coagulation profile
(iv) Petechiae and ecchymotic patches – due to thrombocytopenia, deranged coagulation profile
(v) Proteinuria – due to depressed renal function
(vi) Ascites – due to hypoalbuminaemia caused by proteinuria and increased intravascular hydrostatic pressure
(vii) Intrauterine growth restriction – due to hypertension
(viii) Pappilloedema – due to hypertension
(ix) Basal crepitations – in cases of pulmonary oedema

Investigations

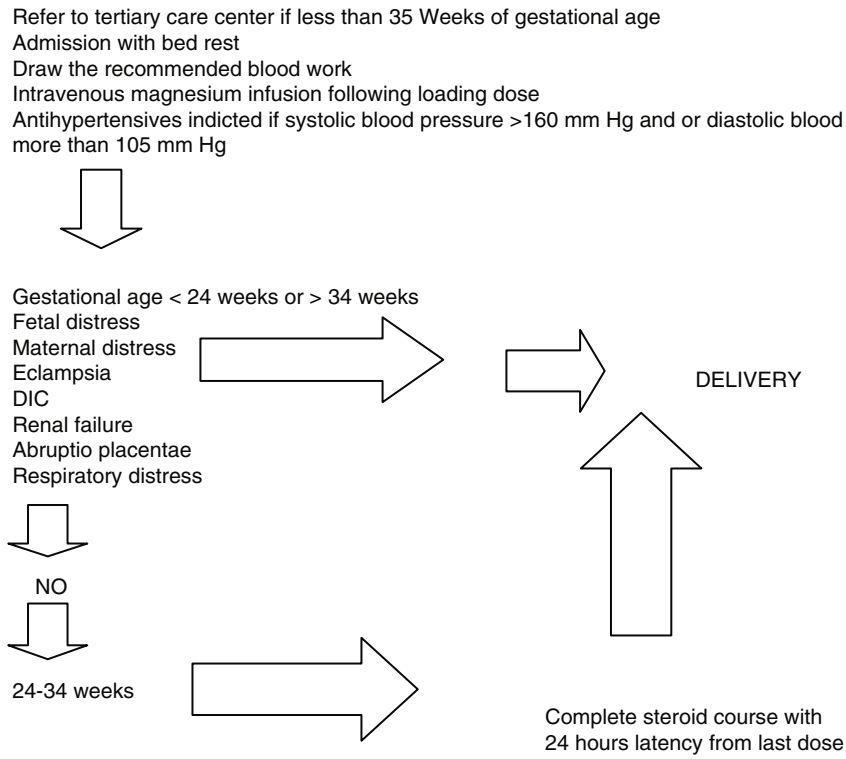
(i) Haemoglobin with complete and differential leucocyte count and platelet count
(ii) Liver function tests (S. bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase, total protein, S. albumin)
(iii) Renal function tests (S. creatinine, BUN, electrolytes)
(iv) Coagulation profile (PT, INR, APTT, d-dimer, FDP, S. fibrinogen)
(v) Urine for routine examination and microscopy
(vi) 24-h urine protein

(vii) Fundoscopy
(viii) Obstetric ultrasound for fetal weight, AFI, and Doppler studies
(ix) Ultrasound of abdomen
(x) Renal ultrasound

Differential Diagnosis

(i) Acute fatty liver of pregnancy (AFLP)
(ii) Thrombotic thrombocytopenic purpura (TTP)
(iii) Haemolytic uraemic syndrome (HUS)
(iv) Immune thrombocytopenic purpura (ITP)
(v) Systemic lupus erythematosus (SLE)
(vi) Antiphospholipid syndrome (APS)
(vii) Cholecystitis
(viii) Fulminant viral hepatitis
(ix) Acute pancreatitis
(x) Disseminated herpes simplex
(xi) Haemorrhagic or septic shock [11] (Table 28.2).

Management



The optimal treatment for maternal safety in confirmed cases of severe preeclampsia or HELLP syndrome is delivery. Unfortunately, at early gestational ages, the neonatal risk of prematurity must be balanced against the maternal and fetal risks of expectant management. Although management of HELLP syndrome is highly controversial, once diagnosed, a decision should be made regarding delivery. Due to the progressive nature of the disease, these patients should always be hospitalized with strict bed rest and care in labour and delivery due to the potential for sudden deterioration of maternal or fetal condition. After assessment and stabilization of maternal status, the fetus is evaluated by fetal heart rate tracing, biophysical profile and/or Doppler studies. The assessment of maternal and fetal status helps determine when delivery is

Table 28.2 Difference between HELLP syndrome, TTP/HUS and AFLP

	HELLP	TTP/HUS	AFLP
Ammonia	Normal	Normal	Increased
Anaemia	Present	Severe	Normal
Antithrombin III	May be decreased	Normal	Decreased
AST	Increased	Normal	Increased
Bilirubin	Increased (indirect)	Increased	Increased (direct)
Creatinine	May or may not be increased	Increased	Increased
Fibrinogen	Normal	Normal	Decreased
Glucose	Normal	Normal	Decreased
Hypertension	Present	May or may not be present	May or may not be present
LDH	Increased	Significantly increased	Increased
Proteinuria	Present	May or may not be present	May or may not be present
Thrombocytopenia	Present	Present	May or may not be present

required or imminent, as delivery is the only true cure for this syndrome. When the mother and fetus are both stable and the gestational age is less than 34 weeks, there is considerable disagreement regarding management. For most, delivery is preferably delayed for 24–48 h for steroid administration. Prompt delivery is indicated when the gestational age is beyond 34 weeks, or earlier in presence of nonreassuring fetal status or if there are associated complications of HELLP syndrome such as multiorgan dysfunction, DIC, abruptio placentae, renal failure, pulmonary oedema, liver infarction, haemorrhage, etc. [1, 12].

There is no consensus regarding the use of high-dose steroids such as dexamethasone (10 mg every 12 h IV) in class 1 and 2 HELLP syndrome or complicated class 3 HELLP syndrome, other than for the indication of aiding fetal lung maturity. HELLP syndrome is not an indication for caesarean delivery. Magnesium sulphate should be administered intrapartum and early postpartum for seizure prophylaxis regardless of blood pressure. As in patients with severe preeclampsia, antihypertensives are used for systolic blood pressures above 160 and or diastolic pressures of more than 105 to avoid intracerebral bleeding [6]. The preferred antihypertensives include hydralazine, labetalol and nifedipine. For pain control in labour, small intermittent doses of narcotics can be given intravenously. When the platelet count is below 75,000, regional anaesthesia and pudendal blocks are both contraindicated to avoid the risk of bleeding and haematoma formation. It should be noted that low platelet count is not a contraindication for local infiltration of anaesthetics for episiotomy or perineal laceration repair. Both maternal and fetal conditions are assessed continuously during the intrapartum period. Platelet count should be maintained at more than 20,000 and 40,000 for vaginal and caesarean delivery, respectively. In patients with platelet count at less than 40,000, four to ten units of platelets are transfused at the time of intubation. Platelet transfusion is also indicated in patients with significant bleeding or platelet count less than 20,000 irrespective of the intended mode of delivery. Hypoglycaemia is seen in patients with HELLP syndrome, and every attempt should be made to keep blood sugar above 60 mg/dl. With good supportive care, a majority of patients recover completely. It is important to continue monitoring fluid balance, laboratory abnormalities and pulse oximetry closely into the immediate postpartum period. Patients who have developed severe complications of HELLP syndrome warrant monitoring for several days. Seizure prophylaxis with magnesium is continued for 24–48 h postpartum, and some also continue the high-dose intravenous steroids for the first 24–48 h after delivery. Maternal serum LDH and platelets are the best markers of disease status [13–15].

Complications Associated with HELLP Syndrome

Maternal

(i) DIC – 15 %
(ii) Abruptio placentae – 10–15 %
(iii) Marked ascites – 10–15 %
(iv) Wound hematoma or infection – 14 %
(v) Pulmonary oedema – 8 %
(vi) Pleural effusions – 6–10 %
(vii) Acute renal failure/acute tubular necrosis – 3 %
(viii) Subcapsular hematoma/infarction/failure – <02 %
(ix) Laryngeal oedema – 1–2 %
(x) Retinal detachment, vitreous haemorrhage and cortical blindness – 1 %
(xi) Death – 1 %
(xii) Others – Adult respiratory distress syndrome, sepsis, stroke, pancreatitis, myocardial infarction and diabetes insipidus, 1 % [4, 16–18]

Fetal

(i) Perinatal mortality ranges from 10 % to 20 % in HELLP syndrome.
(ii) Preterm delivery is up to 70 %.
(iii) High risk of respiratory distress syndrome, bronchopulmonary dysplasia, intracerebral haemorrhage, necrotizing enterocolitis and neonatal thrombocytopenia [19, 20].

Recent Advances

Antithrombin supplementation may correct hypercoagulability, stimulate prostacyclin production, regulate thrombin-induced vasoconstriction and improve fetal status [21].

S-nitrosoglutathione in women with severe preeclampsia lowers maternal mean arterial pressure and reduces platelet activation and uterine artery resistance [21].

Plasmapheresis with fresh frozen plasma could be used to reverse the deteriorating course of some advanced HELLP syndrome [22].

Antenatal *corticosteroids* in some studies show fetal as well as maternal benefit in HELLP syndrome. Steroid leads to disease stabilization and improvement of maternal laboratory indices like platelet count, LDH and liver enzymes [22].

Eculizumab is C-5 complement inhibitor resulted in improvement of clinical and lab parameters and pregnancy prolongation by 17 days [23].

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Part V

Critical Conditions in LR/OT

Amniotic Fluid Embolism and Pulmonary Embolism

29

Nidhi Patel and Ajesh Desai

Abbreviations

ABG	Arterial blood gas analysis
AFE	Amniotic fluid embolism
ARDS	Adult respiratory distress syndrome
CBC	Complete blood count
CTPA	Cardiothoracic pulmonary angiography
CXR	Chest X-ray
DIC	Disseminated intravascular coagulation
FFP	Fresh frozen plasma
HDU	High dependency unit
PE	Pulmonary embolism

Amniotic Fluid Embolism

Background

Amniotic fluid contains various concentrations of fetal squamous epithelial cells, lanugo hair, vernix, mucin, zinc coproporphyrin, prostaglandins, and platelet-activating factors.

Amniotic fluid embolism is a rare but potentially catastrophic condition. The etiology is uncertain but is thought to occur in response to amniotic fluid containing vernix and other solids entering the maternal circulation [1] (Fig. 29.1).

Definition

AFE is a condition in which amniotic fluid and fetal debris enter the systemic maternal circulation, producing an anaphylactic reaction causing sudden cardiovascular collapse, altered mental status, and DIC.

Pathogenesis

It requires two things to produce AFE:

1. A breech in the physical barriers between maternal and fetal environment, mainly at the level of the endocervical veins, uterine trauma sites, and the placental attachment site.
2. A pressure gradient that favors the entry of amniotic fluid from the uterus into the maternal circulation must be present.

Timing

Seventy percent of amniotic embolism may occur during labor, 19 % during caesarean section, and 11 % post delivery [2]. AFE has also been reported during first-trimester surgical termination of pregnancy, second-trimester termination, abdominal trauma, and amniocentesis [3].

Incidence

The incidence is rare. It is approximately 1 in 15,000 deliveries [4].

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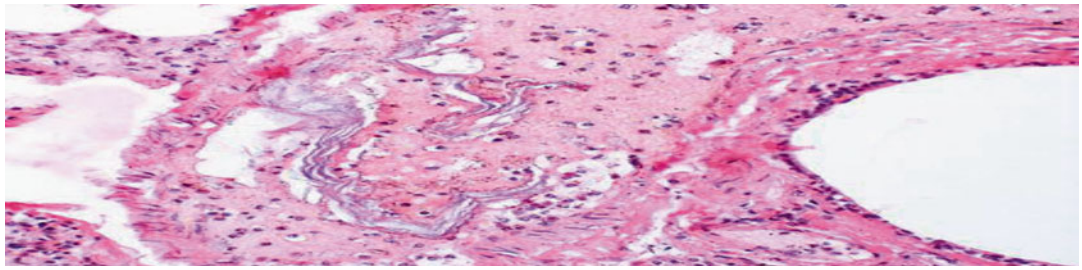


Fig. 29.1 Presence of intravascular squames in amniotic fluid embolism

Table 29.1 Incidence of AFE and case fatality rates in published series

Author	Year published	Incidence (per 100,000 maternities)	Case fatality rate (%)
Knight et al. [5]	2012	1.9–6.1	11–43
Knight et al. [6]	2010	2.0	20
Abenhaim et al. [7]	2008	7.7	21.6
Tuffnell [8]	2005	Not reported	29.5
Gilbert and Danielsen [9]	1999	4.8	26.4
Clark et al. [10]	1996	Not reported	61
Burrows and Khoo [11]	1995	3.4	22
Morgan [12]	1979	Not reported	86

Table 29.1 mentions the incidence of AFE in various studies.

Maternal Neonatal Morbidity/Mortality

Mortality and morbidity are high. It accounts for 14 % of maternal deaths in developed countries and is the second leading cause of maternal deaths in developed countries. Maternal mortality rate has declined from an initial rate of about 80 % to a current level of 22 % with improved recognition and immediate treatment [4] (Table 29.2).

The maternal fatality rate is reported as 35 % [14], within the range of that reported in national registries in the United Kingdom and the United States, respectively, 37 and 61 %. The UK registry reported neurological impairment in 7 % of survivors [15]. Neonatal survival was 79 % in the US registry and 78 % in the UK registry.

Table 29.2 Survival rates

Year	Survival rates (%)	Reason
1979	14	Survival rates improved due to improved resuscitation techniques
2005	30	
2010	80	

RCOG 2011 [13]

Risk Factors

No definite risk factors have been established, and AFE is such a rare event that no risk factor is likely to be of prognostic value. Risk factors are used for retrospective analysis. Risk approach should not be used for altering the clinical management of individual women as baseline risk remains low [7].

However the following risk factors have been identified and mentioned in the literature:

1. Advanced maternal age
2. Placenta previa
3. Placental abruption
4. Operative delivery
5. Induction of labor
6. Polyhydramnios
7. Caesarian delivery
8. Uterine rupture
9. PIH
10. Cervical laceration

Protective Factors

Maternal age <20, dystocia

Response

The body responds in two phases :

- Pulmonary vasospasm causing respiratory and cardiovascular collapse

- The development of coagulopathy (DIC) and hemorrhage (Flow Charts 29.1 and 29.2)

Clinical Presentation

The pregnant women who develop AFE present in any of the phases mentioned below based on place where she has delivered institution or home. Referral cases will be present in phase 1 or phase 2 based on the severity of AFE.

Not all AFE are rapidly progressive and early diagnosis and supportive treatment may result in better outcomes.

First phase: The patient experiences acute shortness of breath and hypotension. This rapidly progresses to cardiac failure leading to a reduction of perfusion to the heart and lungs. This may be preceded by "premonitory symptoms" such as shivering, coughing, restlessness, numbness, agitation, tingling, vomiting, and an unpleasant taste in the mouth. Not long after this stage, the patient will lose consciousness due to circulatory collapse. More than 80 % of women with AFE experience cardiorespiratory arrest within the first few minutes [16].

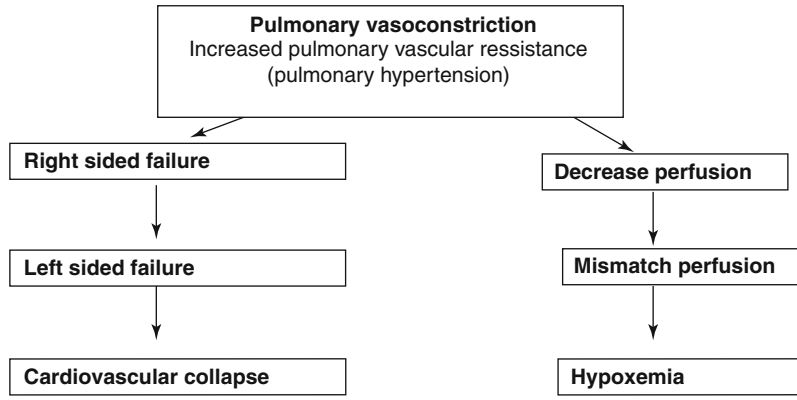
Second phase: Although many women do not survive beyond the first stage, about 40 % of the initial survivors will pass onto the second phase. This is known as the hemorrhagic phase due to excessive bleeding as the blood loses its ability to clot. Collapse of the cardiovascular system leads to fetal distress and death unless the child is delivered swiftly.

Diagnosis

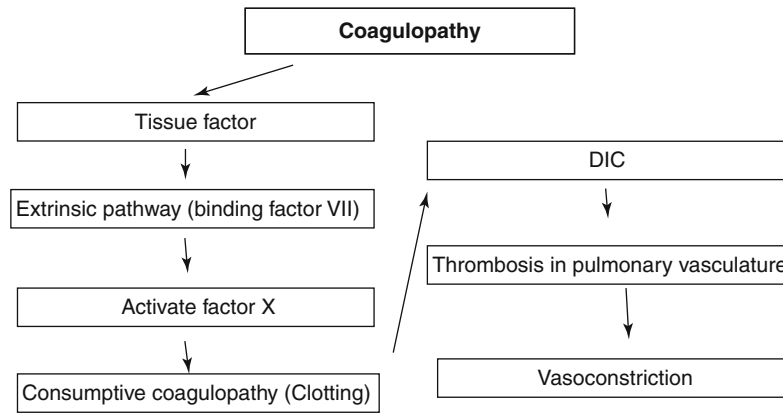
Diagnosis is difficult and may be a matter of exclusion.

Diagnosis is based on suspicion when patient presents with presentation mentioned above.

Currently there is no test available for diagnosis in survivors. Usually autopsy reveals presence of fetal material in pulmonary circulation.



Flow Chart 29.1 Hemodynamic change



Flow Chart 29.2 Mechanism of coagulopathy

Sensitivity of these findings is low as these are also observed in normal women (Table 29.3).

AFE should be suspected in a woman experiencing one or more of the following findings during pregnancy, labor and delivery, or up to 48 h postpartum: Hypotension and/or cardiac arrest, DIC, coma, and seizures.

Tests

When AFE is suspected, the following investigation should be advised:

Laboratory: CBC, coagulation profile, arterial blood gases, cardiac enzymes, and electrolytes

ECG: For tachycardia, ST- and T-wave abnormalities, cardiac arrhythmias, or asystole

Pulse oximetry: To measure oxygen saturation

Chest X-ray: For diffuse bilateral heterogeneous and homogeneous areas of increased opacity

Echocardiography: May reveal severe pulmonary hypertension, acute right ventricular failure with a leftward deviation of the interatrial and interventricular septum, obliterating the left ventricle cavity

Table 29.3 Clinical presentations in AFE [10]

Signs or symptoms	Frequency (%)
Hypotension	100
Fetal distress	100
Pulmonary edema or ARDS	93
Cardiopulmonary arrest	87
Cyanosis	87
Coagulopathy	83
Dyspnea	49
Seizure	48
Uterine atony	23
Bronchospasm	15
Transient hypertension	11
Cough	7
Headache	7
Chest pain	2

Differential Diagnosis

Pulmonary thromboembolism
Air embolism
Cardiac arrhythmia
Anesthetic complications (total spinal/epidural)
Peripartum cardiomyopathy
Aspiration of gastric contents
Postpartum hemorrhage (Table 29.4)

Complications

Complication following the diagnosis of an amniotic fluid embolism includes:

- Disseminated intravascular coagulation
- Multiorgan failure
- Death

Recurrence

Recurrence of AFE has not been reported in the literature.

Table 29.4 Amniotic fluid embolism vs. pulmonary embolism [1]

Clinical features of AFE compared to pulmonary embolism		
	AFE	PE
Timing of onset	Most likely to occur during delivery	Any time
Early symptoms	Dyspnea, restlessness, panic, feeling cold, paresthesia, pain less likely	Dyspnea, cough, hemoptysis
Collapse	Highly likely	May occur
DIC	Highly likely	Absent
ECG	Nonspecific	Nonspecific
CXR	Pulmonary edema, ARDS, right atrial enlargement, prominent pulmonary arc	Segmental collapse, raised hemidiaphragm, unilateral pleural effusion
ABG	Nonspecific	Nonspecific
CTPA	Negative	Positive

Management of Amniotic Fluid Embolism (AFE)

Aim

Aims include the rapid correction of hypoxia, hypovolemia, and coagulopathy for prevention of multiple organ failure.

Treatment

In severe cases, transfer of the patient to HDU may be necessary.

Steps for Management of AFE

1. Lie the patient in the left lateral position.
2. Maintain oxygen saturation at 90 % or higher by administering oxygen in adequate concentrations by face mask, bag valve mask, or endotracheal intubation.
3. Insert two size 16 intravenous cannulas.
   Treatment of hypotension: Optimization of preload, with rapid volume infusion of isotonic crystalloid solutions. Fluid therapy should be based on pulmonary artery catheter or transesophageal echocardiography monitoring. Monitor central venous pressure.
4. Insert a Foley's urinary catheter and monitor and record the patient's hourly urine output.
5. Take bloods for grouping, full blood count (FBC), and clotting and renal and liver function. Send to the laboratory for urgent analysis and crossmatch six units of blood.
6. Monitor vital signs including temperature, blood pressure, pulse, respirations, and oxygen saturation levels and record the observations every 15 min initially.
7. The obstetric registrar/consultant on call should request an urgent ECG and monitor arterial blood gases frequently. Request chest X-ray and a ventilation perfusion scan (VQ) scan if facilities are available.
8. *Cardiac arrest*: Cardiopulmonary resuscitation should be initiated immediately. Uterine evacuation after unsuccessful resuscitation may be therapeutic for the mother, because the weight of the gravid uterus on the inferior vena cava impedes blood return to the heart and decreases systemic blood pressure.
9. *Treatment of refractory hypotension*: Inotropes, such as dobutamine, dopamine, and milrinone, can be added, because *B*-adrenergic effects improve myocardial contractility in addition to the *a*-adrenergic vasoconstrictor effects.
10. Maintain the vital parameters as mentioned in Table 29.5.
11. *Management of coagulopathy and bleeding*: Discuss with consultant hematologist on call for first-line treatment for correcting the coagulopathy and potential severe hemorrhage associated with AFE.

    Packed red blood cells: To maintain oxygen delivery to the tissues.

    Fresh frozen plasma.

    Platelets.

    Cryoprecipitate: Particularly useful to replenish clotting factors in lieu of FFP in volume-restricted patients. It contains fibronectin which could facilitate the removal of cellular and particulate matter from the blood.

    Recombinant activated factor VIIa: For severe DIC, resistant to conventional blood product replacement.

Table 29.5 Maintenance of vital parameters during management

Essential parameters	Minimum value to be maintain
O_2 saturation	>90 %
Blood pressure	>90 mmHg
PO_2	At least 60 mmHg
Urine output	>25 ml/h

The use of heparin to treat consumptive coagulopathy and corticosteroids to induce immunosuppression is very controversial and therefore should not be given.

12. *Management of postpartum hemorrhage*: Look for the usual causes of PPH such as atony, retained product of conception, or cervical or uterine lacerations. If bleeding is profuse and pharmacological intervention is unsuccessful, hysterectomy may be necessary. Consider mechanical and/or surgical techniques to control uterine hemorrhage (Bakri balloon, B-Lynch suture). Bilateral uterine artery embolization has been successful in controlling blood loss in two reported cases [17].
13. *Surgical intervention*
    Perform perimortem caesarean section in women who have suffered a cardiac arrest and who are unresponsive to resuscitation. It may be appropriate to perform a caesarean section for other maternal or fetal indications before cardiac arrest.
14. *Other less common therapeutic approaches*:
    Aprotinin and serine proteinase inhibitor FOY for DIC
    Uterine artery embolization for severe PPH
    Cardiopulmonary bypass
    Pulmonary artery thromboembolectomy
    Thrombolysis with tissue plasminogen activator
    Continuous hemodiafiltration
    Arteriovenous hemofiltration
    Exchange transfusion
    ECMO
    Intra-aortic balloon counterpulsation
    Inhaled nitric oxide
    Prostacyclin
    Sildenafil
15. *Fetal considerations and management*: In most instances, AFE does not occur until after delivery. When AFE occurs before or during delivery, the fetus is in grave danger from the onset because of the maternal cardiopulmonary crisis. The following steps should be taken:
    - Continuous monitoring should remain in progress.
    - Ensure that the patient is positioned in the left lateral position.
    - As soon as the mother's condition is stabilized, delivery of the viable infant should be expedited.
    - Alert the pediatric team if birth is imminent.

If resuscitation of the mother is futile, an immediate grade 1 emergency caesarean delivery may be necessary to save the infant. The sooner the fetus is delivered after maternal cardiopulmonary arrest, the more favorable is the fetal outcome.

Pulmonary Embolism

Background

Pregnancy and the puerperium are well-established risk factors for deep vein thrombosis (DVT) and pulmonary embolism (PE), which are collectively referred to as venous thromboembolic disease (VTE). It is the leading cause of maternal mortality in developing countries [18, 19].

Definition

Pulmonary embolism is a sudden blockage in a lung artery by a blood clot that travels to the lung from a vein in the leg. PE is a serious condition that can:

1. Damage part of your lung because of a lack of blood flow to lung tissue. This damage may

lead to increased pressure in the pulmonary arteries causing pulmonary hypertension.
2. Cause low oxygen levels in your blood.
3. Lack of oxygen can damage other organs of the body.

PE can lead to sudden death if the blood clot is large or there are large no. of small clots.

Pathogenesis

There is five- to sixfold increased risk of pulmonary embolus during pregnancy [20]. This is due to changes in the composition of the blood and changes in blood flow. Changes in the composition of the blood in pregnancy are both increase in clotting factors and a decrease in natural anticoagulation. Changes in blood flow are results of hormonal influence which causes distension of veins and stasis in blood flow in limbs more marked in the left limb.

Incidence

Ten to twenty percent of VTEs are PEs. The incidence of pulmonary embolism in pregnancy varies between 1 per 1000 and 1 per 3000 deliveries [21–23]. The risk of pulmonary embolism is higher in pregnancy beyond 35 years of age.

The risk of thromboembolism during pregnancy and the postpartum period is ten times greater than that for nonpregnant patients.

About 1 per 100,000 pregnant women die from pulmonary embolism [19].

Maternal Neonatal Morbidity/Mortality

Overall mortality rate in PE is 15 %. The risk of maternal mortality remains the same and evenly distributed in antenatal (equal in all trimester) and postnatal period [19, 23, 24]. Postnatal period is the most dangerous time in terms of deaths per week but no period of pregnancy is without risk.

Survival Rates

Survival of the women with PE depends on size of the emboli, percentage of pulmonary vascular occlusion, and health status.

Healthy patients may survive a PE that occludes >50 % of the pulmonary vascular bed. Small emboli may begin to lyse immediately and resolve within hours. It does not produce any physiological effects.

Larger emboli or many small emboli occluding 50 % of pulmonary vasculature will produce right ventricular failure and shock. This situation will lead to death if left untreated.

Sites of Thrombosis in Pregnancy

Superficial venous thrombosis, puerperal ovarian vein thrombosis, and septic pelvic vein thrombosis are the conditions with incidence varying from 0.025 to 15 % associated with PE.

Treatment of this condition decreases the incidence of PE including mortalities associated with PE.

Risk Factors

Risk factors for pulmonary embolus in pregnancy include:

Age: Incidence of pulmonary embolus is doubled in pregnant women aged over 35 years compared to those aged less than 35 [22]; the mortality from pulmonary embolus is nearly 100 times greater in pregnant women aged over 40 years compared to those age 20–25 years [25].

Parity: The risk increasing in those who have had more than three pregnancies independent of age [25], so too is obesity [26].

Operative delivery: Increases the risk of pulmonary embolus between two- and eightfold, the risk varying depending on whether caesarean section was an emergency or elective procedure.

Method of anesthesia: Epidural block reduces the risk compared to general anesthesia [27].

Lactation suppression drugs: Estrogen administration to suppress lactation is another risk factor.

Bed rest: Although it is assumed to be a risk factor for pulmonary embolus, there is little evidence that supports this hypothesis [28].

Thrombophilia: It is an important risk factor for pulmonary embolus in the pregnant as well as

in the nonpregnant state. Hirsch et al. [29] found factor V Leiden heterozygosity in 20 % of cases of thromboembolism in pregnancy, whereas the prevalence in their community was of the order of 5 %. However, it is difficult to estimate the risks for a given thrombophilia with precision, because they vary depending on patient selection.

History of thromboembolism: Women with this history are at higher risk compared to women without this history. Antiphospholipid syndrome, central venous cauterization, immobilization, and obesity are other identified risk factors.

Clinical Presentations in PE

- Tachypnea, dyspnea, pleuritic pain, and cough are the major presenting symptoms in women suffering from PE (Table 29.6).
- Dyspnea or chest pain, either sudden onset or evolving over a period of days to weeks: APE.
- Recognition of the symptoms and signs of VTE may reduce diagnostic delays.
- Signs of pulmonary hypertension (elevated neck veins, loud P_2, right-sided gallop, and right ventricular shift) may be present.
- Signs and symptoms of VTE are highly suggestive for PE but neither sensitive nor specific.
- Massive PE: Sudden onset of near syncope or syncope, hypotension, severe hypoxemia, or cardiac arrest.
- Associated evidence of deep vein thrombosis should be assessed.

Table 29.6 Clinical presentation

Signs or symptoms	Frequency (%)
Tachypnea	89
Dyspnea	81
Pleuritic pain	72
Apprehension	59
Cough	54
Tachycardia	43
Hemoptysis	34
Temperature >37 °C	34

- Pleuritic chest pain, a pleural rub (more peripheral emboli), and hemoptysis will suggest pulmonary infarction. Pulmonary infarction occurs in 10 % of cases as the lungs have doubled the blood supply.

Probability of PE

Clinical management of PE depends on probability of PE (Flow Chart 29.3).

Probability	High	Intermediate	Low
Symptoms (dyspnea and tachypnea)	+	+	+
Identifiable cause for these symptoms	–	–	+
Presence of risk factor	+	–	–

Investigation

Women should be subjected to the following investigation based on the probability.

Take samples of arterial blood for blood gas analysis.

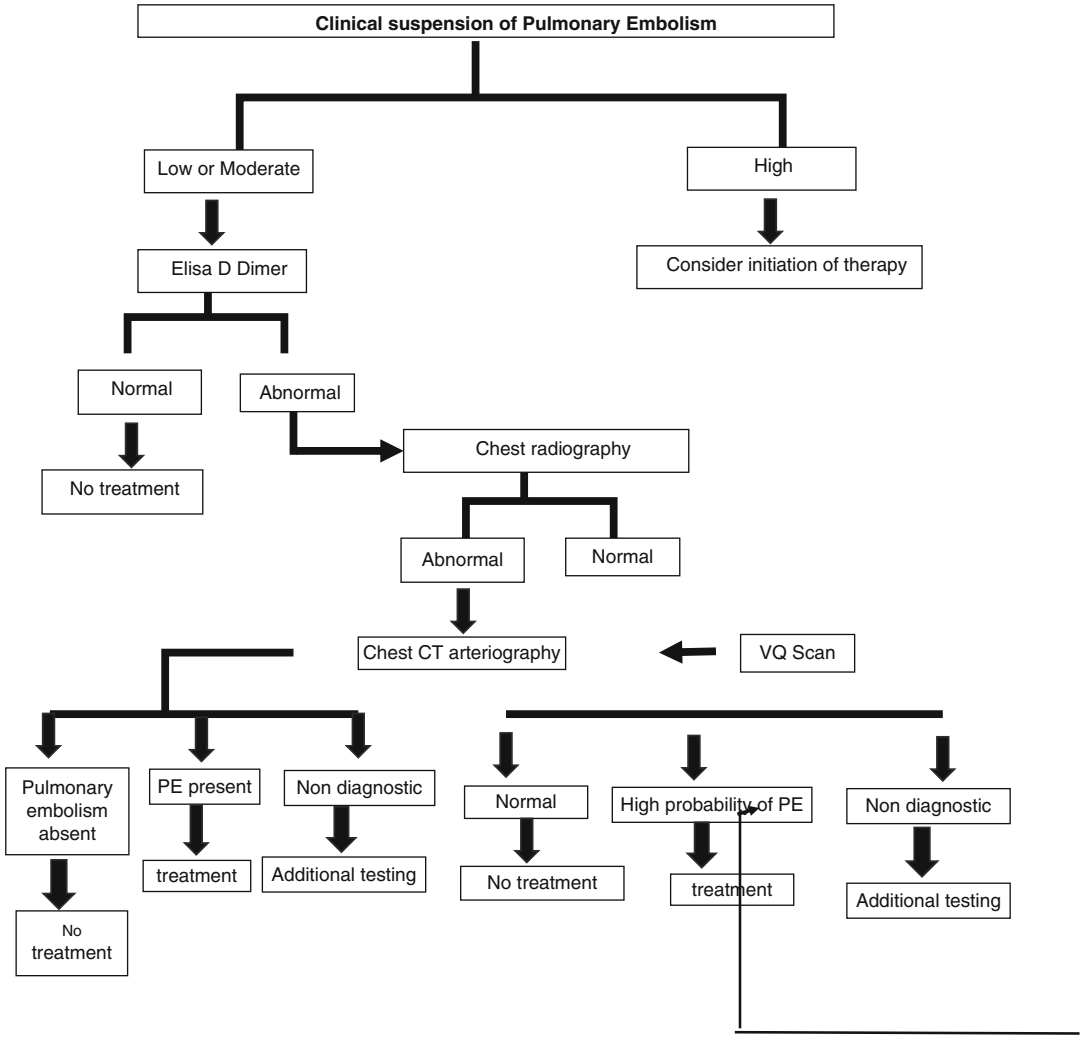
ECG may be taken. The findings are nonspecific. It may show tachycardia, P pulmonale, right axis deviation, and S1Q3 pattern.

Chest X-ray is done but generally nondiagnostic.

Lung scan should be done if available. Radiation dose used is well within permissible limits [30]. Nursing mothers should not breastfeed for 15 h after lung scan [31]. It is argued that ventilation scans add little diagnostic precision [31, 32].

Echocardiography: It is useful in pregnancy as it does not involve radiation. It will show a variety of abnormalities in patients with major central pulmonary embolus [33] as well as exclude other causes of collapse, in particular aortic dissection.

Plasma D-dimer assay: Its role is limited to the case of PE with low probability. In low probable cases if D dimer is negative, no further testing is required [18–52]. The levels are elevated because of the activation of fibrin degradation. If this test is used in pregnancy, it will need different set of cutoff values since fibrin degradation is part of normal pregnancy.



Flow Chart 29.3 Diagnostic

Magnetic resonance imaging: *I*t has recently been shown to be of value in the diagnosis of pulmonary embolus. If preliminary results are confirmed, this could well be the technique of choice for lung imaging in pregnancy because of the lack of radiation.

Pulmonary angiography: Role in pregnancy is limited as it involves radiation beyond permissible levels.

Leg vein imaging: Ultrasound is preferred to detect leg vein thrombosis. This can be done from 20 weeks in left lateral position in high-risk cases.

Avoid false-positive results caused by obstruction to venous flow from the gravid uterus [50].

Thrombophilia testing: As in the nonpregnant state, the presence or absence of thrombophilia has prognostic value relating to recurrence risk and could be used to aid decision making with regard to length of treatment. Thrombophilia status is also important with regard to obstetric management since, in general, women with thrombophilia have a poorer pregnancy outcome. Unfortunately, testing for many thrombophilias is affected both by pregnancy and by anticoagulant treatment.

Antiphospholipid antibodies: Anticardiolipin antibody is not affected and this should be sought in all women who have pulmonary embolus in pregnancy.

Pregnancy-Specific Differential Diagnosis

Amniotic fluid embolus – refer to chapter on amniotic fluid embolism.

Choriocarcinoma should be rule out in cases where PE is suspected.

Management of Pulmonary Embolism

The management of pulmonary embolus in pregnancy is difficult because few internists have much experience of managing pregnant women, and few obstetricians have any experience of pulmonary embolus. In addition there is confusion about the safety of maternal investigations for the fetus.

Initial critical care management in OB-ICU

Heparin, low molecular weight heparin, and warfarin

Heparin is preferred to warfarin for the treatment of pulmonary embolus in pregnancy.

Heparin, both low molecular weight [46] and unfractionated [38], does not cross the placenta or the breast. It is therefore safe for the fetus and for the breast-fed infant.

This is probably because warfarin, in contrast to heparin, does cross the placenta; also the fetus has a relatively immature clotting system so that a dose of warfarin which gives therapeutic anticoagulation in the mother is likely to over-anticoagulate the fetus.

If the mother receives full anticoagulation with warfarin at the time of delivery, there are fetal risks of gross retroplacental and intracerebral bleeding [41].

Dose and Duration

- Unfractionated heparin −10,000 units intravenously twice daily for 10 days (aiming for activated partial thromboplastin time 1.5–2.5 times the control value)
- Low molecular weight heparin such as enoxaparin 40 mg once daily

Laboratory Monitoring

It is accepted that this level of anticoagulation is less than that usually obtained with warfarin maintaining the international normalized ratio at 2.0–3.0. Although it has been suggested that when administered subcutaneously, unfractionated heparin should be given twice daily in doses (presumably in excess of 10,000 units) to maintain the activated partial thromboplastin time in the normal therapeutic range [42, 50], this is very difficult to achieve in practice and usually causes unacceptable bruising at the injection site. There are no comparative studies that indicate that this level of anticoagulation is necessary. Furthermore, although the aim may be to achieve an activated partial thromboplastin time ratio of 1:5, this is not necessarily achieved, leaving the patient at risk from both under- and over-anticoagulation.

In High-Risk Patient (Risk of Further Embolization)

High-dose subcutaneous low molecular weight heparin, e.g., enoxaparin 1 mg/kg every 12 h for a further 6 weeks.

Although high-dose low molecular weight heparin is not currently recommended for the treatment of acute pulmonary embolism in pregnancy, there is no reason why it should not be used in the treatment after the first week granted that lower-intensity anticoagulation has been satisfactory in current clinical practice.

Side Effect

Bone demineralization occurs when anticoagulants are used for more than 2 year. Changes are reversed 1 year after the cessation of therapy and breast-feeding [35]. About 2 % of all patients taking heparin for 3 months or more in pregnancy will have symptoms from bone fracture due to demineralization. The incidence of such symptoms may be less with low molecular weight heparin. Bone densitometry studies will access the level of demineralization.

Thrombocytopenia: It is very uncommon in pregnant patients even if they take heparin long term.

Anaphylaxis: It is associated with intravenous use of heparin. If necessary, use corticosteroid with intravenous heparin to reduce the risk [45].

Intranatal Management

Labor, surgical procedures, and epidural block are safe providing that the thrombin time, activated partial thromboplastin time, and platelet count are normal. The patients do not bleed excessively, and nor is epidural hematoma a problem provided that the thrombin and activated partial thromboplastin times are not prolonged by more than 5 s [44].

The dose of subcutaneous unfractionated heparin may be reduced to 7500 units twice daily in anticipation of the contraction in circulating blood volume and to counter any bleeding risk. This reduction is not required when low molecular weight heparin is used [44].

Treatment in Postnatal Period

The excess risk of thromboembolism associated with pregnancy continues after delivery for an ill-defined period, believed by most clinicians to be no longer than 6 weeks. Therefore, treatment for pulmonary embolus occurring in relation to pregnancy should continue until 6 weeks after delivery or until 3 months after the initial episode whichever is the longer. Since warfarin is not secreted in significant quantities in breast milk, patients may convert to warfarin after delivery even if they are breast-feeding [47]. There is also no problem with either unfractionated heparin or low molecular weight heparin with regard to breast-feeding.

Indication to Give Warfarin

Patients with artificial heart valves, heparin allergy, or very active antiphospholipid syndrome may still require treatment with warfarin in pregnancy despite the fetal and maternal risks.

Thrombolysis

Because of the risk of bleeding, thrombolytic treatment should not be used at the time of delivery unless it appears that the patient is likely to die. Streptokinase was the agent used most frequently. It (and probably other thrombolytic drugs) does not cross the placenta because of its high molecular weight. However in the mother, bleeding is the major side effect, usually from the genital tract and often severe; the overall incidence of bleeding is about 8 % [51].

Surgical Procedure

Procedures such as embolectomy are only occasionally indicated in pregnancy; one obvious situation would be massive pulmonary embolus immediately after delivery when thrombolysis should not be used. However, in most maternity units thrombolytic treatment or even transvenous catheter fragmentation or embolectomy [27] is more likely to be available.

Caval Filters

Interrupted devices should only be placed where recurrent pulmonary embolus has occurred despite adequate anticoagulation or where patients cannot receive conventional anticoagulant treatment [34, 37, 40].

Prophylaxis

In women who have had thromboembolism in the past, the recurrence risk in pregnancy has been estimated to be about 12 % whatever the circumstances of the original clot and independent risk factors such as thrombophilia [53]. The table below shows the prophylaxis in low and high recurrence cases.

Low risk for recurrence	High risk for recurrence [27]
One previous episode of thromboembolism (pulmonary embolism or deep vein thrombosis) no matter what the original circumstances and who have no other high-risk factors	More than one episode or have thrombophilia or a family history of thromboembolism, suggesting that they may have undiagnosed thrombophilia
Low-dose aspirin (75 mg) once daily from as soon as pregnancy has been confirmed until delivery [51, 54]	Subcutaneous heparin in doses as above as soon as pregnancy is confirmed

Low risk for recurrence	High risk for recurrence [27]
At delivery, patients should take subcutaneous heparin [46] either unfractionated heparin 7500 units twice daily or low molecular weight heparin such as enoxaparin 40 mg once daily, until at least the 1st week after delivery, and then either continue heparin for a further 5 weeks or switch to warfarin [49]	Management at delivery and thereafter is as for low-risk patients [44]

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Management of Critical Cord Accidents

30

A.K. Debdas

Introduction

Cord accidents are dire obstetric emergencies and should be dealt with in war footing. As these do not happen every day, it is recommended that all labour room should have a printed protocol readily available for instant use (see at the end of the article).

Anatomical Pathology of Cord Accidents [1]

This can be classified into three types:

I. Cord prolapse – Naked cord (not covered by the bag of membranes) hanging in the vagina ahead of the presenting part. Sometimes it comes out of the introitus. The risk of heavy cord compression and consequent fetal distress and fetal death is highest in these cases (Fig. 30.1).
II. Cord presentation – Here although the cord has come out into the vagina ahead of presenting part, but it is still within the bag of membranes which is intact. In these cases the chance of severe compression is low because the liquor present in the bag acts as a buffer to compression. This however is no cause of complacency because the membrane may rupture at any time inducing severe cord compression (Fig. 30.2).
III. Occult cord prolapse (see Fig. 30.3)

It is a deductive diagnosis and often an intelligent assumption. The situation here is like this: In a patient in labour, occurrence of sudden, recurrent and severe FHR abnormality like severe bradycardia and gross irregularity on simple auscultation by fetoscope or picked up by doptone raises the suspicion of such occurrence. Depiction of 'variable deceleration', i.e. typical 'M'-shaped tracing on CTG [3] (see Figs. 30.4 and 30.5), almost certifies cord compression, specially when on vaginal examination no cord is found to present or prolapse and when no other cause of such severe fetal distress is present. In some of these, the cord lies by the side of the presenting part rather than ahead of it, but *no attempt should be made to check this* by exploring with finger because that may precipitate an actual presentation or prolapse (Fig. 30.3).

Note: No time is to be wasted by doing US scan in these cases to prove or disprove the occult presence of the cord – it is the severe fetal distress which must be managed immediately to relieve the fetus.

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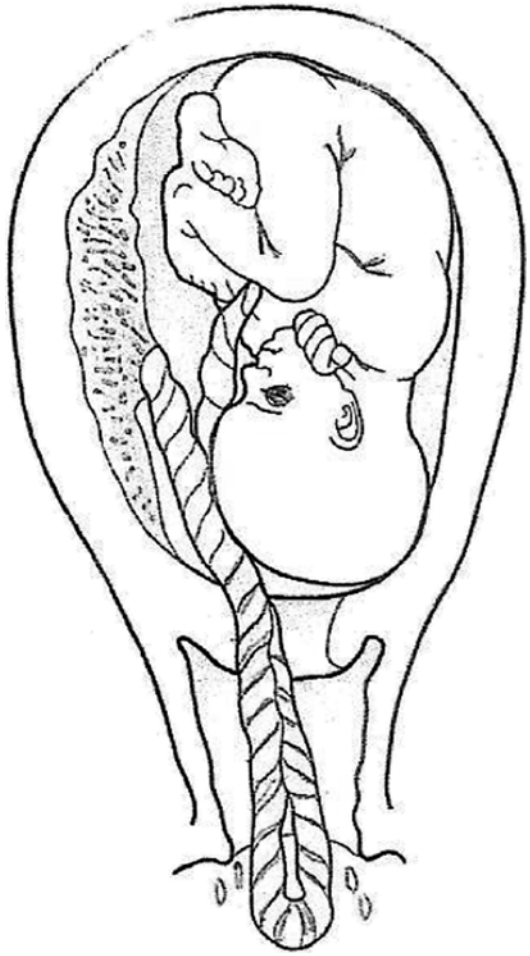

Fig. 30.1 Membranes have ruptured and cord has prolapsed. It is hanging outside the introitus

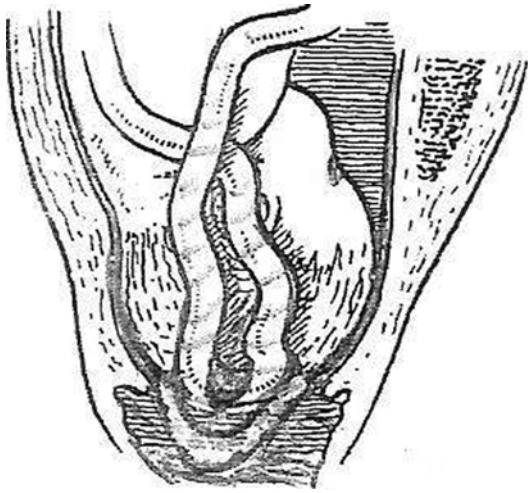

Fig. 30.2 Cord presentation: cord is ahead of the head within the bag of membranes

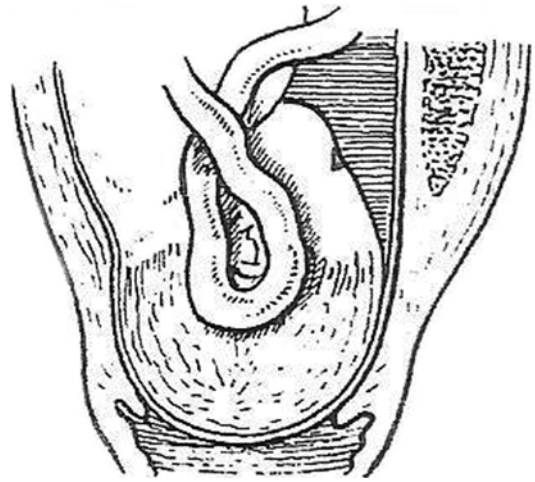

Fig. 30.3 Occult cord prolapse: the cord has not come down ahead of the presenting part. It is lying by the side of the ear of the fetus. So, it is not palpable on vaginal examination (occult). It is suspected. In the cases of unexplained severe fetal distress.CTG shows typical 'M'-shaped tracing

Basic Cause of the Cord Accidents/ Prolapse [1, 2, 4, 5]

Bad fit between presenting part and the cervix and consequent – 'loss of ball valve action'.

Pregnancy factors that can disturb the ball valve action

Malpresentation – Transverse lie, breech, or compound presentation
Multiple pregnancies
Hydramnios
Premature labour (small baby and relatively more liquor)
Low placenta and marginal attachment of cord to placenta, long cord

Obstetric procedures that can disturb the ball valve action (iatrogenic)

During ARM specially if the presenting part is loosely applied to cervix
During manual rotation of the head for occipito-posterior position of the head
During internal podalic version
During scalp electrode application
During amnioreduction for hydramnios
During amnioinfusion

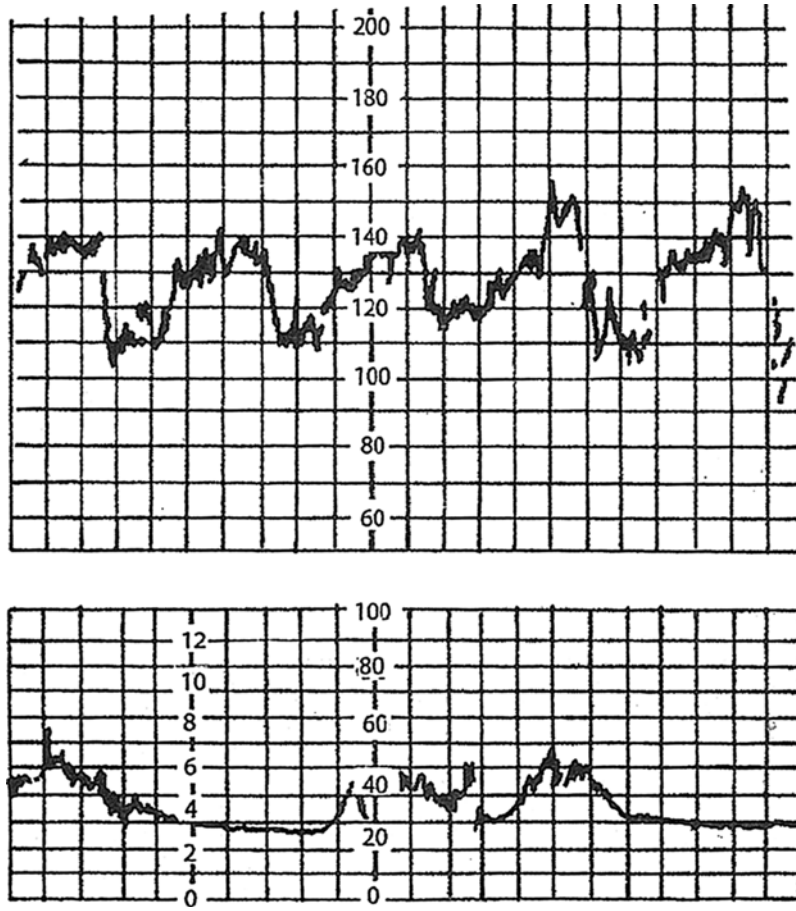

Fig. 30.4 CTG tracing showing variable deceleration. Note the variable occurrence of 'M'-shaped decelerations which is typical of cord compression

Incidence

One in 300, mostly associated with transverse lie, unstable lie and breech, but the incidence is reducing because more and more elective CS or early CS are being done for these cases. It happens more commonly in multigravida [2].

Precautionary Measures for Cord Accidents

- Vaginal examination should be done immediately as soon as membranes rupture to exclude cord prolapse.
- Presence of cord before or at the side of the presenting part should be carefully excluded before, during and after ARM.
- One should be extra careful about the cord at every vaginal examination in the cases having predisposing factor for cord prolapse.

Prediction of Occurrence of Cord Prolapse

To do US scan in cases who are having predisposing factors for cord prolapse to see the location of cord.

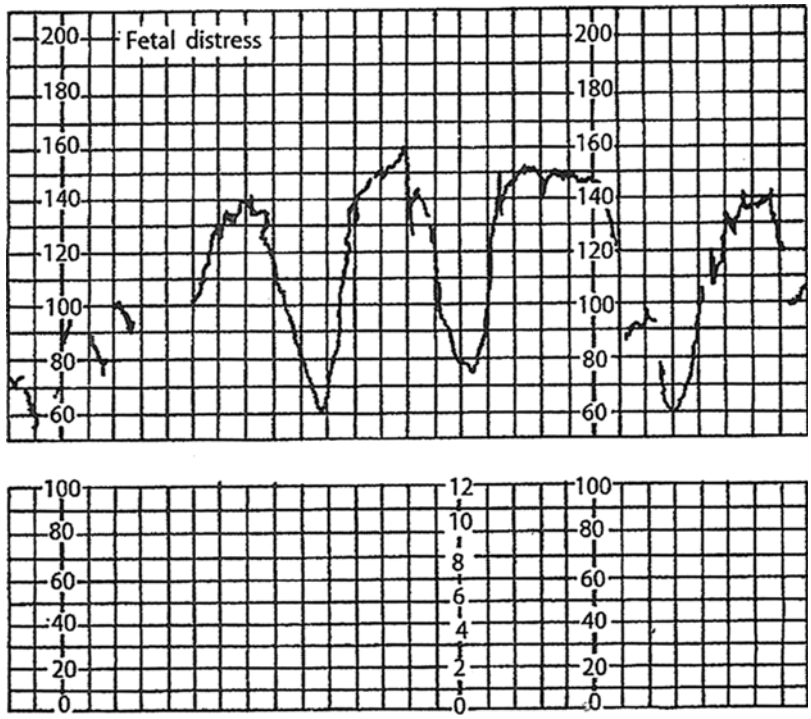

Fig. 30.5 CTG tracing showing variable deceleration in a case of cord prolapse. Note the typical 'M'-shaped decelerations

There is however insufficient evidence to support such practice [2].

Prevention of Cord Prolapse [2]

- Elective admission in hospital of cases of transverse and oblique lies at 37 weeks so that prompt management can be done in case the cord prolapse
- Elective caesarean section on these cases (and also breeches) at term
- Applying fundal pressure while doing ARM specially where the head is not engaged

Diagnosis of Cord Accidents

Diagnostic Points

- *Typical feel* – This has to be specially learned while conducting the third stage of labour (doing controlled cord traction).
- *Presence of pulsation* – This has to be done very gently because manipulation of cord can cause spasm of the blood vessels within. Presence of pulsation additionally certifies that the fetus is alive. Pulsation may not be felt during an uterine contraction so recheck for it after the contraction is over.
- CTG (if available) : It is most objective and does simultaneous documentation. Belting of the transducers are to be done very carefully without giving much pressure on the uterus to prevent rupture of membranes or drainage of liquor.
- By digital Doptone : Its advantage is – the effort of counting FHR which needs some skill is not required. The FHR is brightly visible in the display window.
- By ordinary audio Doptone: With this though the FHS is loudly audible but the FH rate needs manual counting.
- Pinards Fetoscope : This is not recommended because it is not at all objective. Besides it needs lot of pushing and prodding of uterus which may cause rupture of membranes, drainage of liquor and even pressure on the prolapsed cord.

Differential Diagnosis

Cervical lip – specially the external os
Fetal ear

These two structures can be rolled by the examiner's vaginal fingers as if these are umbilical cord.

Management of Cord Accidents

(Some Instant Actions Which Should Be Carried Out)

Oxytocin drip is stopped if it is running.
Oxygen inhalation is started through face mask.
Do tocolysis – by giving 0.25 mg terbutaline subcutaneously [2].
If contractions are fierce and frequent, give fast drip of 500 ml of Ringers Lactate solution as bolus [2].

Actual Management

Three factors on which management basically depends:

- Whether the baby is alive or dead
- Whether the os is dully dilated or not
- Whether the fetus is mature enough to survive postnatally

Whether the baby is alive or dead

WHEN BABY IS DEAD – Vaginal delivery can be allowed.

There are some unsuitable conditions for allowing vaginal delivery like placenta praevia, Transverse lie – specially if it is impacted and the baby is big.

A lot of physical and *moral support* is required for conducting the vaginal delivery.

WHEN BABY IS ALIVE – Arrange to deliver her by suitable means depending on the degree of dilatation of cervical os and the maturity of the fetus (see below).

'Staff Duty Distribution' Protocol for Efficient Management [4] Where the Fetus Is Alive

Principle – Division of responsibility and allocation of definite responsibility.

Duty allocation system

I. *Duties of the person who detected the cord prolapse*
(Her or his finger is still in the vagina)
1a. *In the case where the cord has actually prolapsed* – In this case, she or he should not remove her or his finger from the vagina and should keep pressing upwards the presenting part so that it does not cause pressure on the cord.
1b. *In the case where the cord is only presenting and the membranes are intact* – In this case, the fingers should be taken out of vagina in case accidentally the membranes get ruptured. Sterile vulval pad is to be given, and frequent examination of the pad is to be done for its wetness. Patient also is to be warned to inform immediately in case she feels any leak.
2. She or he should:
   - Loudly announce the diagnosis.
   - Direct others on duty in labour room to swing into action *as par protocol* (see later) which should be easily available in labour room.

II. *Duties of the assistant*, *1*
Her or his duties are mainly *administrative* and may be carried out either by a doctor or a nurse:
1. Communication – To the senior most obstetrician available in the hospital, to OT, to anaesthetist, to neonatologist, to the blood bank and also to one or two colleague to come and help
2. Counselling and consent taking – Patient, her relations are to be counselled and consent to be taken for caesarean section and also for operative vaginal delivery

3. Record keeping:
   In author's unit, it is done in 'real time' and 'electronically' to save time and remove ambiguity. For this, this assistant simply has to put her cell phone in 'audio recording mode' and speak to it loudly each time a job is done by her or by any other staff mentioning the time on the clock at that moment.

III. *Duties of assistant, 2*
It is *all clinical* job.
1. Pre-op preparation for CS – Put up IV drip, draw blood and send for cross-matching and Hb.
2. Positioning of the patient – so as to take the pressure off from the cervix and the cord by arranging one of the following:
   - Raise the foot end of the bed.
   - Put her in the exaggerated left lateral position (see Fig. 30.6).
   - Put her in knee elbow position (See Fig. 30.7) – this is quite strenuous for the mother.
   - Put in a large Foley's catheter and distend its balloon up to 500–700 ml by attaching it to an IV drip and raising the bottle high [2].
   - Inflatable "Fetal Pillow" which is used to elevate low impacted fetal head during caesarean section may also be used for the purpose.

   This and also the Foley's catheter are to be deflated just before making the uterine incision for caesarean.
3. Fetal monitoring – It should be *continuous* by digital doptone, preferably by CTG.

Dilatation-Dependent Decisions

That is whether the cervical os is dully dilated or not

Os not fully dilated – Emergency caesarean section is performed except in case of multigravida with past history of vaginal delivery, with os 8 cm or more dilated. Vacuum extractor may be used because arranging for caesarean section in most units in developing countries like India takes at least 30 min to 1 h.

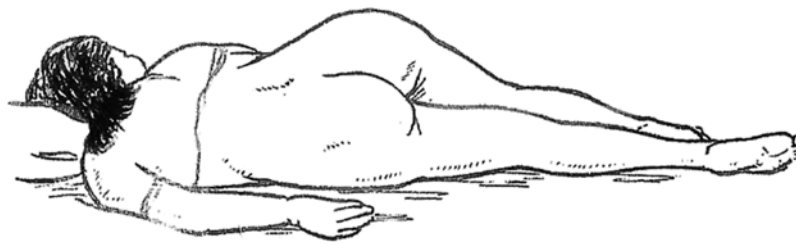

Fig. 30.6 Exaggerated left lateral position. In this position, weight of uterus gravitates upwards off pelvis

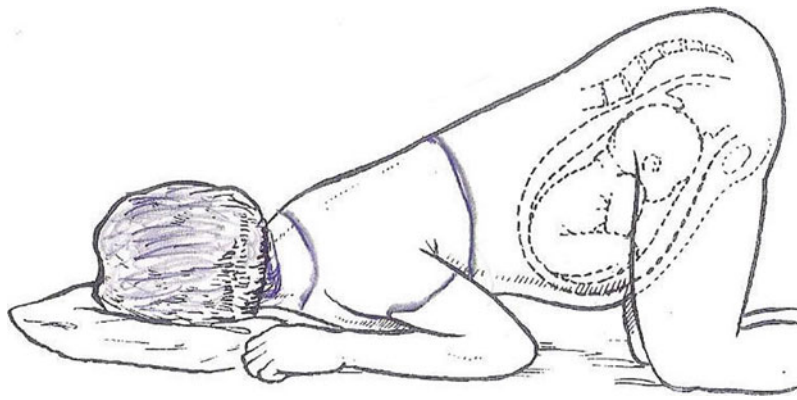

Fig. 30.7 Knee-chest position. Figure shows how the weight of the fetus very effectively comes off the pelvis in this position

Os fully dilated – Explore the possibility of vaginal delivery which may be assisted by:

Forceps (faster method) or ventouse provided other perquisites of application of these methods are fulfilled.
For breech cases, breech extraction is to be done.
For second twin with transverse lie – internal podalic version with breech extraction may be done by a skilled obstetrician.
For all vaginal deliveries, early rather large episiotomy should be given for facilitation to conduct delivery of a baby.

Fetal Maturity-Dependent Decisions

The main question to ponder here is whether the fetus is mature enough to survive postnatally.

For too tiny (<1500 g) premature fetus, emergency CS for cord prolapse may be counterproductive because of very high PNM and also high morbidity in such cases.

Patient' relatives are to be counselled about it.

Such premature CS should be considered only if the unit has good NICU facility.

Practical Points about Caesarean Section for Cord Prolapse [2]

Time target
Fetus with sign of fetal distress is best delivered within 30 min; for others, it can be 60 min.
Pre-incision FHS checking
It is a must and to be done by doptone just before making incision to confirm that baby is still alive.
Anaesthesia for CS
Generally GA is preferred for the speed with which it can be induced.
However, if there is no sign of fetal distress regional, anaesthesia may be chosen.

Management of Cord Prolapse Diagnosed outside Hospital [2]

- Positioning of mother – Put her in exaggerated left lateral position.
- Call an ambulance with special request to come immediately
- Send her to the nearest hospital with caesarean section facility.
- Inform the hospital that such a case is coming to stay prepared to manage the case.

Perinatal Mortality

It has been reported as 36–12/1000 [2].

Instant Management Tips cum Protocol

- Fetus DEAD: Allow vaginal delivery.
- Fetus ALIVE: Follow the following protocol:
  - Prevent compression of cord: By finger pressure on the presenting part and positioning of mother: Knee-chest or exaggerated left lateral position.
  - In cord '*presentation*' cases: Avoid rupturing the membranes.
  - In cord '*prolapse*' cases: Avoid handling the cord, may cause spasm of cord vessels.
  - Call for URGENT help:
    For arranging emergency CS if the os is not fully dilated.
    For Forceps or Ventouse if she is fully dilated

- Do continuous FHR monitoring: By CTG or at least by digital doptone.
- Start oxygen inhalation: Through mask or catheter.

- Stop oxytocin drip: To reduce the risk of compression.
- Do urgent tocolysis: If contractions are frequent and fierce, inject terbutaline 0.25 mg subcutaneously.
- For CS:
  - Try to do within 30–45 min – Use GA for speed if there is fetal distress.
  - Just before making incision – Auscultate FH by doptone to make sure that the baby is still alive.
  - For very premature fetus weighing <1500 g: Counsel guardian about prognosis, immediate and delayed.
- A neonatologist must be in attendance.
- The mother and her relatives are to be counselled from time to time and consent obtained.

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Acute Inversion of the Uterus

31

Gokul Chandra Das and Gitanjali Deka

Introduction

Acute inversion of the uterus is a rare but serious obstetrical complication, seen in the immediate postpartum period. It is defined as 'the turning inside out of the uterine fundus into the uterine cavity'. Uterine inversion is associated with significantly high maternal morbidity and mortality. Women can sink into profound shock. Immediate diagnosis and management can reduce maternal mortality.

Incidence

Incidence of uterine inversion varies according to geographical location and ranges from 1 in 2000 to 1 in 50,000 deliveries [1]. It has also been reported during caesarean section, but in lesser extent. Maternal mortality has been reported to be 15 % [2]. A fourfold decrease in the incidence of acute uterine inversion is noted after the introduction of active management of the third stage of labour [3]. Increased number of institutional deliveries is another cause for this change.

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Classification

According to timing of event, inversion can be classified as acute (within 24 h), subacute (>24 h to 4 weeks) or chronic type (>4 weeks). The relative prevalence of acute inversion is more than subacute and chronic types.

According to severity, it can be incomplete or complete inversion. In incomplete variety, there will be cupping of the fundus (1st degree), or fundus may pass through the cervix but still inside the vagina (2nd degree). In complete type, the uterus, cervix and vagina are completely inverted and visible outside the introitus (3rd degree).

Causes and Predisposing Factors

It is well established that mismanagement of the third stage of labour (premature traction on umbilical cord and fundal pressure before separation of placenta) is the commonest cause of acute uterine inversion. This can happen when delivery is conducted by untrained dais, a situation more likely to occur in developing countries, which explains why the incidence in India is higher than that of the UK or USA [4]. In majority of cases, acute inversion of uterus can be prevented, by skilfully conducting vaginal delivery, specially the third stage of labour with routine active management of third stage of labour.

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Other risk factors are uterine atony, fundal implantation of a morbidly adherent placenta, manual removal of the placenta, precipitate labour, a short umbilical cord, connective tissue disorders (Marfan syndrome, Ehlers-Danlos syndrome), etc. However, in up to 50 % of cases, no risk factors are identified and considered as idiopathic. Therefore, this condition can be unpredictable.

Clinical Presentation

The presentation varies depending on the degree of severity of the inversion. Severe lower abdominal cramping, bearing down sensation with mild tachycardia and fall of blood pressure are found in early stages. But the classical presentation is that of major obstetric haemorrhage and shock, which may occur in 94 % of cases. It should be noted that, initially, shock may be neurogenic due to the parasympathetic effect caused by traction of the ligaments supporting the uterus. Therefore, a high indication of suspicion in cases where shock is out of proportion to blood loss can help in making an early diagnosis and avoid massive haemorrhage.

On abdominal examination, there will be cupping of the uterine fundus in incomplete variety and in complete-type uterus is not at all palpable abdominally. On vaginal examination, a reddish mass is seen in the vagina or outside the introitus.

Usually clinical diagnosis is enough to confirm acute inversion, but if condition permits, ultrasonography should be done.

Management

Once diagnosis is established, management should be started immediately. Resuscitation and repositioning of the uterus must be undertaken simultaneously.

Resuscitation

- Shout for help: Urgently mobilize all available personnel.
- Call for senior obstetrician and anaesthetist
- Monitor vital signs (pulse, B.P., respiratory rate, temperature)
- Start iv line with large bore cannula and infuse IV fluid (NS or RL) at the rate appropriate for the woman's condition.
- Catheterize the bladder.
- Send for blood requisition.
- Administer oxygen at 6–8 L per min by mask or nasal cannula.
- Appropriate antibiotic cover is given to prevent infection.

Steps of Manual Reposition of Uterus

- Do not attempt to remove the placenta from inverted uterus if not already removed.
- With sterile gloves on, grasp the uterus and push it through the cervix towards the direction of umbilicus to its normal position.
- Use the other hand to support the uterus per abdominally.
- After successful reposition of the uterus, remove the placenta manually.
- Keep the hand inside the uterus and ask the assistant to start oxytocin infusion immediately to maintain appropriate uterine contraction.
- Once the uterus is contracted firmly, remove the vaginal hand in cone-shaped manner.

O'Sullivan's Hydrostatic Technique

If manual reposition is unsuccessful, then O'Sullivan's hydrostatic technique [5] is employed. O'Sullivan's hydrostatic technique or manoeuvre first, described by JV O'Sullivan in British Medical Journal in 1945 and still, is a very effective technique. It is a method of reinverting the uterus by infusing warm saline into the vagina, preferable done under sedation. Uterine rupture should be excluded before performing O'Sullivan's technique.

- A woman is in deep Trendelenburg position with head below the level of the perineum or in exaggerated lithotomy position.
- Use 2×1 L bags of warm irrigation fluid (0.9 % sodium chloride) attached to a wide bore infusion set (cystoscopy irrigation set).

- Insert the hand in to the vagina with the open end of the tube near the posterior fornix. Seal the introitus by holding the labia around the forearm or wrist with other hand. An assistant will cover above the surgeon's hand to prevent leakage of fluid.
- Infuse warm saline in to the vagina under gravity. Three to four litters of fluid may be required.
- Gradual distension of the vagina increases the circumference of the cervix and traction on round ligament resulting in correction of the inversion.
- Oxytocin infusion is started immediately to maintain appropriate uterine contraction.
- The placenta should be removed at this stage, after repositioning of the uterus and complete correction of the inversion in order to avoid shock and torrential bleeding.
- A careful manual exploration is done to exclude the possible genital tract trauma.
- Appropriate antibiotic coverage is required to prevent infection.

Ogueh and Ayida Technique

Alternate to O'Sullivan's technique, Ogueh and Ayida technique [6] can be tried. This technique, first described by Dr. O. Ogueh and Ayida in 1997, in London, is also similar to that of the O'Sullivan's technique; the only difference is to maintain an adequate water seal, and they use ventouse cup. They suggested that the open end of the cystoscopy tube or infusion tube is attached to a silastic ventouse cup and place in the lower vagina at the inner aspect of the introitus to create a more effective seal [6]. Any fluid leaking around the ventouse cup is effectively blocked by the surgeon's palm at the introitus. Therefore, adequate hydrostatic pressure is generated to reduce the inversion.

Role of General Anaesthesia

If hydrostatic technique is unsuccessful, the procedure can be repeated under general anaesthesia. If the patient is stable and not bleeding and vital signs are stable, spinal anaesthesia can be given [7]. Success rate of manual reposition or hydrostatic reposition under anaesthesia is very good.

Role of Tocolytics

To facilitate replacement of the uterus, myometrial relaxation is achieved by tocolytics, such as iv magnesium sulphate (4 g), iv terbutaline (0.25 mg) or iv nitroglycerin (50 μg) [8]. Tocolytics are mainly tried in haemodynamically stable patient.

Role of Surgery

Surgery for acute inversion of uterus is rarely needed. Surgery is indicated when hydrostatic method fails or recurrence after initial correction or in delayed referral cases. In these cases, patient is treated conservatively with injectable antibiotics, local douching and antiseptic packing of the vagina. Spontaneous correction of inversion is noted in few cases, otherwise plan for major surgery, electively.

Huntington's Operation [9]

Following laparotomy, the inversion site is exposed. The cup of the inverted uterine fundus is identified. Two Allis tissue forceps are introduced in to the cup on each side 2 cm below the ring. Gentle upward traction is exerted on the forceps; new pair of forceps should be placed 2 cm below the previous one and traction applied to pull out the fundus gently. Repeated clamping and tractions are required to complete the procedure.

Haultain's Operation [7]

In this operation after laparotomy, posterior cervical ring is incised about 5–6 cm longitudinally. Fundus of the uterus is pulled out of the cervical

ring similar to that of the Huntington's operation. After correction, repair the hysterotomy incision with interrupted sutures. Forty units of oxytocin in 500 ml NS is started to maintain the uterine contraction.

Role of Laparoscopy

Vijayaraghvan and Sujatha [10] reported a case where acute inversion of the uterus was managed under laparoscopic guidance. Patient must be haemodynamically stable prior to laparoscopic surgery.

Conclusion

Acute inversion of uterus is a life-threatening obstetric complication. Early diagnosis and immediate management will decrease maternal morbidity and mortality. Institutional delivery and strict reliance on the AMTSAL protocol can prevent inversion of uterus to a great extent.

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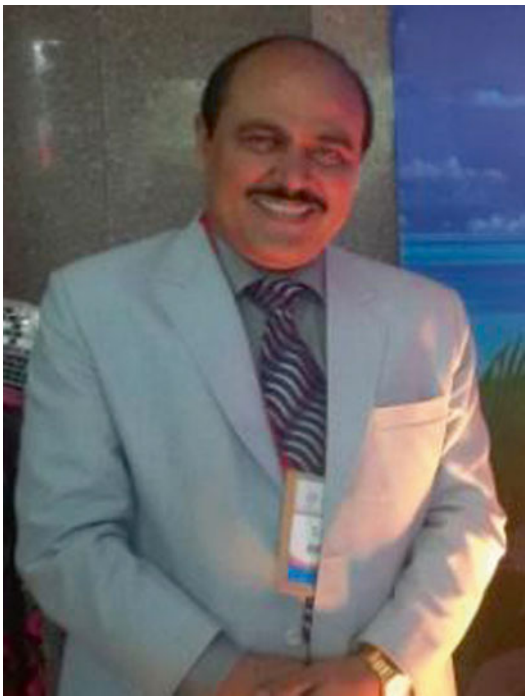
Rupture of the Gravid Uterus

32

Ashis Kumar Mukhopadhyay



A serious and catastrophic obstetric complication, rupture of the pregnant uterus has a high maternal and neonatal mortality and morbidity particularly in developing countries. In developed countries, widespread availability and easy access to good quality ante and intranatal care have significantly reduced the risk of uterine rupture.

Dehiscence is a more common event that seldom results in major maternal or foetal complications. By definition, uterine scar dehiscence constitutes *separation of a pre-existing scar or a weakness in muscle* that *does not disrupt the overlying visceral peritoneum* (*uterine serosa*) and that does not significantly bleed from its edges. The foetus, placenta and umbilical cord must be contained within the uterine cavity, without foetal distress.

By contrast, *uterine rupture* is defined as a *full-thickness separation of the uterine wall and the overlying serosa*, usually associated with clinically significant uterine bleeding; foetal distress; expulsion or protrusion of the foetus, placenta or both into the abdominal cavity; and the need for prompt caesarean delivery, uterine repair or hysterectomy.

Prevalence of Uterine Rupture

Most of the studies are institution based and do not always reflect the true incidence in the community. Recent WHO review summarizes the prevalence rate from few published reports, discussed under the headings below.

From 1976 to 2012, 25 peer-reviewed publications described the incidence of uterine rupture,

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and these reported 2,084 cases among 2,951,297 pregnant women, yielding an overall uterine rupture rate of *1 in 1,146 pregnancies* (*0.07 %*) [1].

From the time of diagnosis to delivery, only 10–37 min are available before clinically significant foetal morbidity becomes inevitable. Foetal morbidity invariably occurs because of catastrophic haemorrhage, foetal anoxia or both.

Rupture of Unscarred Uterus

Report from less and least developed countries show that more than 75 % of the ruptures occur in an intact uterus, whereas it is extremely uncommon in developed countries. Such cases carry a high maternal mortality (1–13 %) and a very high perinatal mortality (up to 92 %).

The normal, *unscarred uterus is least susceptible to rupture*. A 10-year Irish study by Gardeil et al. showed that the overall rate of unscarred uterine rupture during pregnancy was 1 per 30,764 deliveries (0.0033 %). *No cases of uterine rupture occurred among 21,998 primigravidas*, *and only 2* (*0.0051 %*) *occurred among 39,529 multigravidas with no uterine scar* [2].

A meta-analysis of seven large, modern (1976–1998) studies from developed countries revealed 149 uterine ruptures among 1,108,660 deliveries. This finding suggested that the *modern rate of unscarred uterine rupture during pregnancy is 0.013 %* (*1 of 7,440*). This rate of spontaneous uterine rupture *has not changed appreciably over the last 40 years*, *and most of these events occur at term and during labour*. In developed countries, most of the uterine ruptures follow previous caesarean sections (incidence in the range of 1 %), whereas those without previous section, it is extremely rare (1 per 10,000 deliveries).

An *eightfold increased incidence* of uterine rupture of 0.11 % (1 of 920) has been noted *in* developing countries. This increased incidence of uterine rupture has been attributed to a higher-than-average incidence of neglected and obstructed labour due to inadequate access to medical care.

WHO systematic review also found that in less and least developed countries, uterine rupture is more prevalent than in developed countries [3].

Classification of Rupture

1. *Rupture of previous scar*
   - Myomectomy
   - Hysterotomy
   - Caesarean section
2. *Traumatic rupture of unscarred uterus*
   - External cephalic version
   - Blunt trauma
3. *Spontaneous rupture of unscarred uterus with underlying pathology*
   - Uterine anomalies
   - Multiparity
   - Previous manual removal with adherent placenta
   - Curettage with/without perforation
4. *Spontaneous rupture of unscarred normal uterus*

Aetiology

- Rupture of uterine scar
- Obstructed labour
- Uterine hyperstimulation
- Other factors
- Iatrogenic

High-risk group for rupture of uterus: Uterine rupture is more likely to occur in the high-risk group listed below:

A. *Unscarred uterus*
- Grande multigravida
- H/O MTP or septic abortion
- Uterine anomalies
- Trauma
  - Blunt external trauma
  - External cephalic version
- Obstetric history with:
  - H/O prolonged labour or obstructed labour
  - H/O manual removal of placenta

B. *Scarred uterus*
- Myomectomy: open or laparoscopic
- Hysterotomy
- Previous C-section

Contributory Factors of Uterine Rupture

- In developing countries, majority of the ruptures in unscarred uterus follow obstructed labour. Other factors that contribute are:
  - High parity
  - Injudicious use of oxytocics
  - Instrumental deliveries
  - Scar dehiscence

Anticipation of Rupture

- Previous LSCS done for:
  - Obstructed labour
  - Inverted T incision
  - Lateral extension of incision
- Classical scar
- Hysterotomy
- Myomectomy where cavity has been opened
- Obstructed labour in multiparty

Detection of Uterine Rupture

- During pregnancy
- During labour
- After delivery

During Pregnancy

Only classical scar and hysterotomy scars rupture during pregnancy. Any of the features like dull abdominal pain/slight bleeding/rising pulse rate/tenderness on scar and FHS absent may cause concern and suspicion.

Symptoms and Signs

- *Previous post-operative course* – An uneventful post-operative recovery in the primary C-section signifies that the union of the scar is characterized by good formation of bridging tissue which eventually strengthens itself over a period of 18–24 months. On the other hand, a stormy post-operative period in the form of fever, signs of local infection and tenderness hampers successful healing of the scar and may result in a weak scar in the next pregnancy.
- *Scar tenderness* – Palpation of the area of the erstwhile uterine scar in the region of lower segment (assuming that all CSs today are LSCSs) elicits important information regarding integrity of the scar. If the palpation is non-tender, soft and elastic in feel, it suggests a healthy scar for all practical purposes.
- *Premature rupture of membranes* – A sign of incoordinate uterine action causing rupture of membranes before term.
- *Easy palpability of foetal parts* – Almost diagnostic of rupture of the pregnant uterus which loses its contour and in most of the cases foetus translocates out of the uterus.
- *Silent rupture* – More than half of the cases are diagnosed incidentally at the time of repeat section.

Features of Obstructed Labour

- *Something giving way* – This is the sensation the mother experiences in most of the cases of ruptured uterus.
- *Cessation of uterine contractions* – Because of lack of continuity of the uterine wall.
- *Haemorrhagic shock* – Rupture may be associated with massive intraperitoneal haemorrhage.

After Delivery

- Features of shock without obvious bleeding P/V are an indication of exploration for rupture.

Differential Diagnosis

1. *Severe abruptio placentae*
   - Unbearable abdominal pain and uterine tenderness.
   - Disproportion between bleeding volume and degree of anaemia.

- Ultrasound may show retroplacental hematoma, and foetus is intrauterine.
- Usually associated with pregnancy-induced hypertensive diseases or trauma.

2. *Intrauterine infection*
   - Usually seen in premature rupture of membrane, prolonged labour, and multiple vaginal examinations.
   - May have abdominal pain, uterine tenderness, etc.
   - Temperature rise.
   - Abdominal examination: foetus is intrauterine.
   - White blood cell and neutrophil counts rise.

Management Options

Repair of rupture, allowing future childbearing – Feasible and safe, and should be adopted in cases where the rupture is linear, clean cut and relatively hypovascular, especially in cases where future childbearing is an issue for the mother.

- *Repair of rupture with tubectomy* – In cases where the client does not want future children, but she is young and wants preservation of the uterus. All the conditions for repair of the rupture as mentioned above must be fulfilled
- *Subtotal hysterectomy* – Most of the cases of uterine rupture need a quick subtotal hysterectomy as a life-saving measure. This procedure is fast, causes little blood loss and recovery is good.
- *Total hysterectomy* – Considering the theoretical risk of future stump carcinoma, many gynaecologists prefer a total hysterectomy rather than the simpler procedure of subtotal hysterectomy. This is of course an ideal operative management, but the client must be haemodynamically stable and not much of blood loss has taken place primarily.

Previous Uterine Myomectomy and Uterine Rupture

Nearly all uterine ruptures that involved uteri with myomectomy scars have occurred during the third trimester of pregnancy or during labour. In India, only few cases of a spontaneous uterine rupture are reported before 20 weeks of gestation [4].

Previous Laparoscopic Myomectomy

Risk of pregnancy-related uterine rupture attributable to laparoscopic myomectomy is 1 % (95 % confidence interval [CI], 0.5–5 %) [5]. Different authors reported no pregnancy-related uterine ruptures in four studies of 320 pregnancies in women who previously underwent laparoscopic myomectomy. Uterine rupture has been reported to occur as late as 8 years after laparoscopic myomectomy. This finding suggests that additional investigations with long-term follow-up are needed.

Management Proper

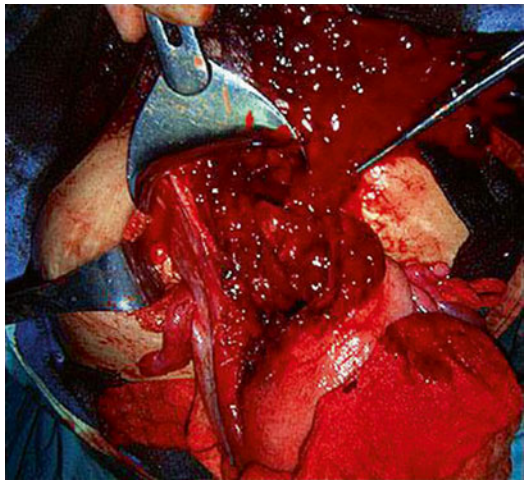
- *General resuscitative measures:*

  Maintenance of haemodynamic status is of utmost importance.

  Early goal-directed therapy (*EGDT*)*:*

  - Rapid crystalloid infusion. MAP should be maintained at 65 mmHg and urine output should be 30 ml/h.
  - Blood transfusion.
  - Insertion of CVC, PAC. CVP is ideally maintained at 8–12 mmHg
  - Administration of ionotropes like dopamine, norepinephrine or dobutamine
  - Tissue oxygenation is maintained by supplemental oxygenation maintaining oxygen saturation at >70 %. Blood transfusion improves the tissue oxygenation especially if haematocrit <30 %. Mechanical ventilation is needed in ARDS anti-inflammatory agents like hydrocortisone or dexamethasone must be used liberally.

- Emergency laparotomy. The perinatal outcome is best if the baby is delivered within 10–15 min of rupture



- A timely diagnosis and minimizing the time from the onset of signs and symptoms until the start of definitive surgical therapy
- Immediate stabilization of the mother
- Surgical treatment for the mother

Surgical Treatment: Determinants

- *Type of uterine rupture* – A ragged rupture with extensive oozing rarely benefits from repair. Hysterectomy is often lifesaving.
- *Extent of uterine rupture* – Extension is more common on the left side. Huge broad ligament haematoma may be quite difficult to control. No specific bleeding point is seen. In these cases, bilateral internal iliac artery ligation is beneficial followed by packing and drainage.
- *Degree of haemorrhage* – Massive intra-peritoneal haemorrhage can cause rapid deterioration of haeamodynamics
- *General condition of the mother* determines anaesthesia, incision and extent of operation.
- *Mother's desire for future childbearing* – This is very important determinant for conservative surgery on uterus and its extent.

Conservative Surgical Management (Uterine Repair)

- Low transverse uterine scar rupture
- No extension of the tear to the broad ligament, cervix or paracolpos
- Relatively smooth margins of the scar
- Easily controllable uterine haemorrhage
- Good general condition
- Desire for future childbearing
- No clinical or laboratory evidence of an evolving coagulopathy
- Any situation where these conditions are not met with needs a more radical surgical approach, usually a quick subtotal hysterectomy. This is particularly so when the mother has lost a substantial amount of blood, and her general condition is very low.
- Most of these situations are emergent and hence there is little scope of any preparations or investigations. But a quick check on Hb%, blood grouping and typing and serology is mandatory. IV fluid (crystalloids) must run rapidly and arrangement should be made for at least three to four bottles of packed cells.
- Counselling plays a very important role in these extreme situations. The relatives must be convinced that the operation is binding and life saving. There must be a fully informed consent in place before anaesthesia is put on.

Repeat CS: Changing Trends

In the United States prior to the 1980s, the dictum was 'once a caesarean section, always a caesarean section'. ACOG in 1988 and 1999 acknowledged that a trial of labour among patients with a previous caesarean section was a reasonable option. This has brought the risk of uterine rupture in trial of labour in developed countries, though the incidence is probably less than 1 %.

Factors Influencing Uterine Rupture After Trial of Labour After Caesarean (TOLAC) [6]

- *Maternal age* – Incidence of rupture is more at advanced maternal age above 35 years.
- *Nature of previous delivery* – An elective C-section is less likely to rupture compared to an emergency C-section.
- *Number of caesarean sections* – Two or more C-sections pose more risk than a single section and therefore unsuitable for TOL.
- *Non-reassuring CTG* on admission.
- *Gestational age* – term size.
- Spontaneous vs *induced labour.*
- Sonographic estimate of *foetal size.*

Vaginal Birth After Previous C.S. (VBAC)

Often associated with serious co-morbidities including damage to urinary tract, infection, need for blood transfusion and prolonged hospital stay, increased neonatal mortality and morbidity.

When a non-reassuring tracing appears during trial after previous caesarean, the obstetrician has less than 20 min to deliver the baby by emergency section to avoid permanent damage or death [7].

How Can Rupture Be Predicted?

- *Non-reassuring CTG tracing* (*55–87 %*) – A *normal* CTG has all four features that are *reassuring*. A CTG is *non-reassuring* if it has one feature which is *non-reassuring*, but the others are *reassuring*. It along with other clinical features acts as a predictor of impending rupture
- *Continuous pain abdomen* – Unlike rhythmic labour pain which is intermittent and typical of uterine contractions.
- *Vaginal bleeding* – Anything which seems to be in excess of show, in association with uterine tenderness or superficial feeling of the foetal parts is a good predictor of rupture.
- *Disturbance of uterine contractions*, otherwise called inordinate uterine action, is suggestive of altered polarity of normal uterine contractions
- *Sudden collapse* – Indicates rapid intraperitoneal bleeding and shock.
- Extreme *thinning* of the scar on ultrasonography – A very strong predictor of impending rupture, either in pregnancy or more likely, in labour.
- Previous history of *classical scar* – If such history is available, it is almost certain that rupture is inevitable, mostly in labour. Classical scars rarely unite with precision and accuracy; therefore, it is most likely to give way, sooner or later.

Strategies to Reduce the Rupture

- Careful vigilance during the antenatal period can predict many ruptures.
  - Detect 'high-risk' cases (see above)
  - Use of partogram in all labours.
  - Early referral policy.
  - Injudicious use of oxytocin and rampant use of prostaglandins should be strongly discouraged.
  - Patient transportation services should be provided as a priority.
  - Donors accompanying the patient moving to a higher centre can be life-saving.
  - Careful selection of VBAC patients.
  - Awareness through media.
  - Contraception and FP services should be more effective.

Prophylaxis

- At-risk mothers should have mandatory hospital delivery – contracted pelvis and previous history of caesarean delivery, hysterotomy and myomectomy. General anaesthesia should be avoided in external version.
- Judicious selection of case with previous history of C.S. for vaginal delivery and induction and augmentation of labour by oxytocin.

- Internal podalic version should never be done in obstructed labour.
- Forceps delivery or breech extraction through incompletely dilated cervix should be avoided.
- Destructive vaginal operation or manual removal in morbid adherent placenta should be done by senior person.

Should We Revert Back to the Dictum of 'Once a C.S., Always a C.S.'?

- It has been calculated that it would take 370 elective repeat caesarean sections to prevent one uterine rupture due to trial of labour [8].
- Elective repeat caesarean section is not guaranteed to prevent uterine rupture.

Summary and Key Points

- Uterine rupture is a rare but serious and catastrophic complication of pregnancy and labour.
- Although there is no reliable method for prediction of rupture, a high degree of suspicion can help in the early detection of these cases.
- Early detection and management have a good prognosis for both mother and her baby.
- Trial of vaginal delivery after caesarean section (VBAC) is a legitimate and rational way of management.
- Whenever possible, repair of the uterus should be attempted. It gives psychological boost to the patient and also preserves the possibility of future childbearing.
- Pregnancy after repair of rupture is generally safe for the mother and baby, but delivery must be by caesarean section.

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Shoulder Dystocia

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Madhu Nagpal

Shoulder dystocia is an intrapartum emergency. It is due to the delay in delivery of trunk after head has delivered out of introitus due to impaction/arrest of shoulders behind the symphysis pubis. The consensus definition as failure of delivery of shoulders spontaneously and/or with gentle normally exerted downwards traction of head, thus requiring maneuvers to deliver one or both shoulders, is widely accepted [5]. The delay in head to trunk delivery by >60 s is also considered diagnostic of dystocia. Shoulder dystocia is neither truly predictable nor accurately preventable but is vulnerable to happen under certain high-risk situations.

Incidence

The incidence in general is 0.2–1.75 %. In many situations, it has been under or over reported as figures are derived from labor room records which may have missed certain difficult cases or may have recorded overenthusiastically certain unreal situations. The incidence has increased definitely with increased size of babies being born nowadays and more attention paid to document its occurrence [7]. The incidence rises from 0.6 % with 3–3.5 kg birth weight to 10.5 % with 4–4.5 kg birth weight [1].

To know its real incidence – standardization of criteria for registry has been recommended.

Criteria for Registry

1. Ultrasonographic estimated fetal weight more than 4 kg
2. Maneuvers if required for delivery of shoulders
3. Delayed head to body delivery time >60 s
4. Turtle sign – retraction of fetal head back into introitus following delivery

Risk Assessment: Antenatal Factors

Maternal obesity and increased BMI : With woman weighing <90 kg, the incidence is 0.6 %, while with body weight more than 113 kg, the risk is 5 % [6].

Macrosomia: With fetal birth weight 4–4.5 kg, the incidence is 8.6 %, and if birth weight is >4.5 kg then 35.7 % [5]. According to another study by Spellacy et al., [9] with birth weight 4–4.5 kg, the incidence is 10.3 % and with birth weight >4.5 kg–14.6 %. The ultrasonographic estimated fetal weight >4 kg is a risk factor with error of ±12 %, more so in diabetics,

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but all large babies do not have dystocia. Macrosomia in diabetics has relative risk of 5.2 % as compared to nondiabetics. The risk is 7 % in GDM with metabolic anthropometric derangement as shoulders and trunk size is more than head due to hyperglycemia/hyperinsulinemia effect on fetal abdominal growth.

Postmaturity is another risk factor. At 40 weeks the incidence is 12 %, and at 42 weeks it is 21 % due to associated macrosomia.

Previous shoulder dystocia is an independent variable factor; however, other cofactors like big size, gestational age, glucose intolerance, and previous neonatal injury contribute.

Sometimes an anencephalic fetus with normal size shoulders causes arrest. But presently seldom anencephalic fetus is left undiagnosed to deliver past mid trimester.

Risk Assessment: Intrapartum Factors

1. Prolonged late first stage at 8 cm dilatation
2. Prolonged second stage with instrumental delivery
3. Arrested descent of head in second stage of labor

Mechanism

Normally when head exits through the pelvic outlet, although bisacromial diameter is bigger than biparietal diameter, shoulders enter the pelvic brim in oblique diameter. The posterior shoulder leads via sacral hollow or sacrosciatic notch, while the anterior shoulder of fetus accommodates well in obturator foramen. When bisacromial diameter is much bigger than biparietal diameter or with altered pelvic brim, if posterior shoulder occupies the sacral hollow and the anterior shoulder is impacted below pubic symphysis, arrest occurs. When both shoulders are arrested above brim, bilateral shoulder dystocia occurs. The compression of umbilical cord in birth canal creates an emergency situation.

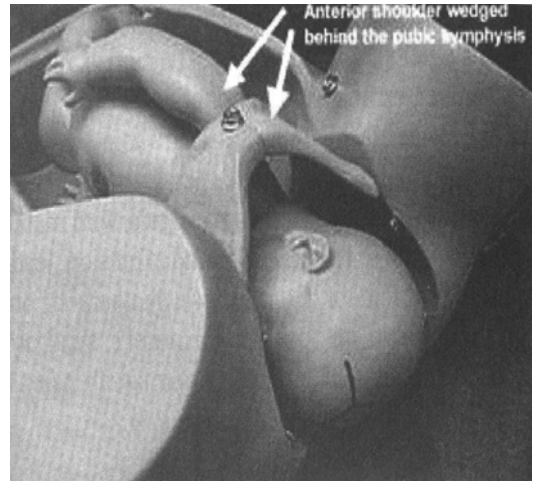

Fig. 33.1 Shoulder dystocia

Diagnostic Suspicion

1. When head delivers but does not undergo spontaneous external rotation, instead tightly recoils back tightly against perineum – Turtle neck sign.
2. When normal amount of downward traction is unable to deliver the shoulder.
3. When on examination posterior shoulder is felt in the sacral hollow and anterior shoulder is stuck behind pubic symphysis, it is unilateral shoulder dystocia.
4. When posterior shoulder is not felt in sacral hollow, then it is bilateral shoulder dystocia (Fig. 33.1).

Management: Aim

1. To reduce head to body delivery time in order to release cord compression early
2. To avoid fetal and maternal injury due to aggressive manipulations

Management: Steps

1. Always suspect in case of delay.
2. Ask for assistant, pediatrician, and anesthesiologist.
3. Initially attempt gentle downward traction on fetal head with moderate suprapubic pressure

by the assistant to disimpact anterior shoulder supported by maternal expulsive efforts. If not successful, then abandon further vigorous traction or twisting of head.
4. Adopt standard obstetrical emergency drill.
5. Assess success of maneuvers used quickly.

Suprapubic Pressure

The abducted diameter of fetal shoulder is more than adducted diameter, so apply suprapubic pressure to back of fetal scapula, pushing it down and laterally toward larger oblique transverse diameter of pelvic inlet (Fig. 33.2).

McRoberts' Maneuver

A Liberal episiotomy is given. The maternal hips are hyperflexed and abducted to position with thighs on abdomen closely which straightens the maternal lordosis and lumbosacral angle and rotates the maternal pelvis cephalad reducing the inclination angle from 25 % to 10 %. The suprapubic pressure is applied downward and laterally by the heel of the hand to push the posterior aspect of anterior shoulder toward fetal chest. This reduces bisacromial diameter, so that anterior shoulder is rotated in oblique pelvic diameter, free to slip behind symphysis pubis from brim with gentle downward traction. This has less complications and is 90 % successful [4] (Figs. 33.3 and 33.4).

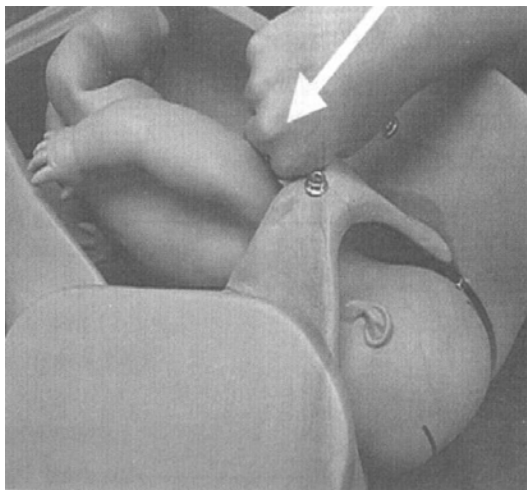

Fig 33.2 Suprapubic pressure maneuver

Post Shoulder Delivery

Pass the hand deep into vagina in sacral hollow, identify the fetal humerus and follow it to the elbow, then flex the elbow, and grasp the forearm and hand, sweeping it across the fetal chest. With traction on the arm and support of the fetal shoulder, deliver by rotating the post shoulder by 180° to anterior and then vice versa (Fig. 33.4).

Wood's Screw Maneuver

With infants back on maternal right, insert two fingers of right hand on anterior aspect of posterior shoulder and exert pressure to rotate the baby by 180° while maintaining adduction of post shoulder, so that this being below in pelvis is screwed around at the level under pubic arch and is delivered from ant position, similar to Lovset in breech.

Or anterior shoulder is rotated by 180° in cork screw manner by applying pressure on post aspect of ant shoulder which is made to disimpact and come in pelvis [10] (Fig. 33.5).

All Four's Maneuver

Patient is repositioned in knee arms position so as to disimpact the shoulder. The flexibility of sacroiliac joints allows 1–2 cm increase in sagittal diameter of the pelvic inlet, and gravity helps to push posterior shoulder anteriorly below the sacral promontory.

Zavanelli's Maneuver

The posterior shoulder is not accessible at the brim, baby being alive, cephalic replacement back in the vagina is done. Uterine relaxant terbutaline is given and cesarean section is done. The success rate is 92 %, but fetal trauma is not ruled out.

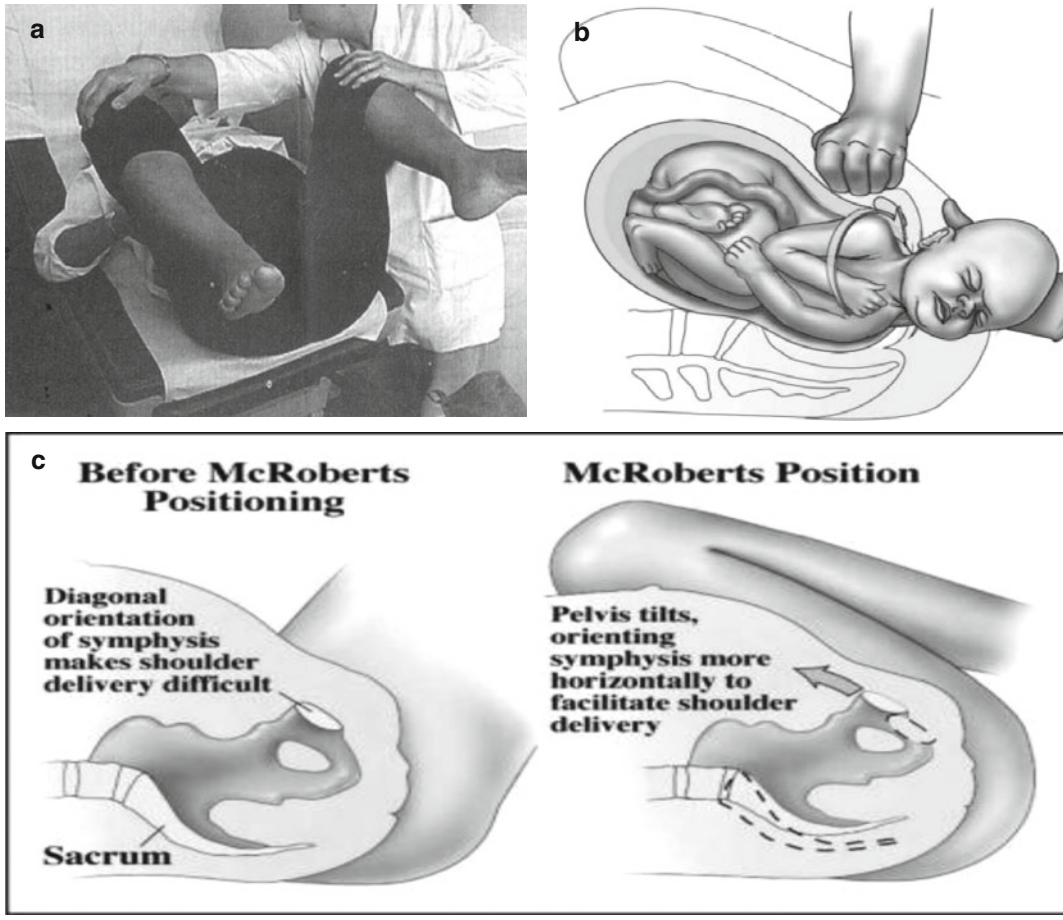

Fig. 33.3 (**a**) McRobert's position. (**b**) Suprapubic pressure during McRobert's maneuver. (**c**) Principle of McRobert's positioning

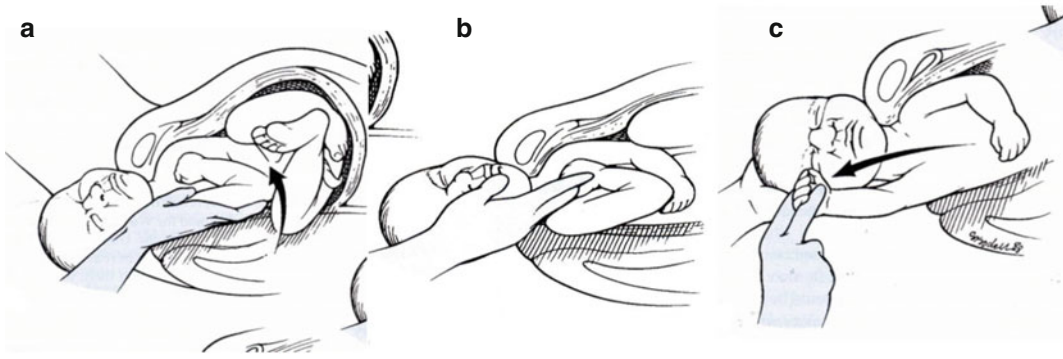

Fig. 33.4 Posterior shoulder delivery. (**a**) Tracking humerus till elbow. (**b**) Flexing elbow. (**c**) Grasping forearm and hand and delivering by sweeping over the chest

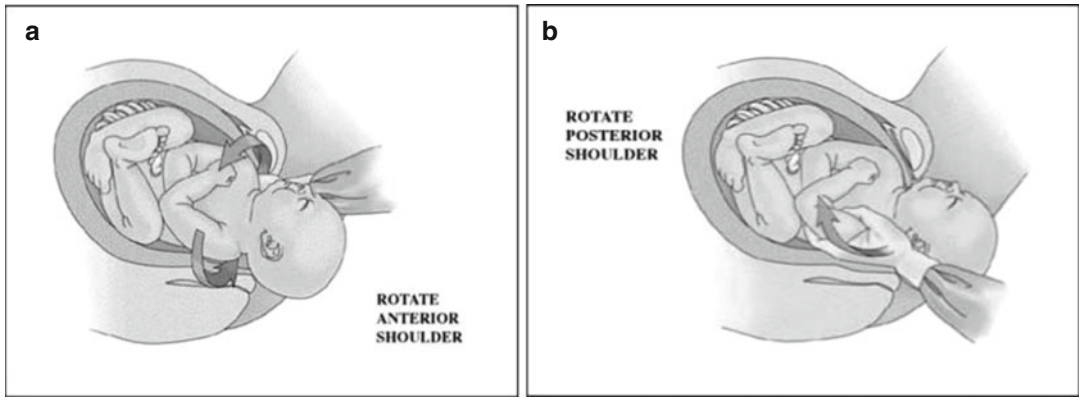

Fig. 33.5 Woodscrew maneuver. (**a**) Rotation of anterior shoulder. (**b**) Rotation of posterior shoulder

Parallel Forceps

Grasp the fetal chest and abdomen thereby rotating the shoulders to the optimum diameter of the pelvic inlet.

Cleidotomy

There is limited clinical experience with this procedure available. Intentional fracture of clavicle to reduce bisacromial diameter is not done. This procedure is done in dead baby preferably. In alive baby, it may be done in superficial portion of clavicle. It heals without any sequelae, but the risk of injury to subclavian vessels remains.

Symphysiotomy

It is a surgery done with expertise. Usually it is done in live baby but still carries a lot of fetal morbidity. The risk of bladder injury exists. Some permanent disability of mother may remain.

Maternal Morbidity

It is due to lacerations of genital tract, extension of episiotomy, and rupture uterus. Postpartum hemorrhage may occur due to uterine atony, prolonged labor, large infant, and increased blood loss from vaginal tears.

Fetal Injuries

Neonatal neuromusculoskeletal injuries cause morbidity and mortality. About 11 % suffer serious neonatal trauma.

Nervous System

Brachial plexus injury occurs in 5–15 % of neonates. The incidence of 17 % injury mostly brachial plexus injury is reported in a study of 205 shoulder dystocias [8]. The range of permanent palsy with brachial plexus injury is 4–32 %. Erb's palsy occurs due to avulsion of C 5 and 6 nerve roots causing paralysis of deltoid, supraspinatus, infraspinatus, biceps, and brachioradialis muscles. It is clinically seen as internal rotation, adduction of shoulder, and extension and pronation of the elbow, possibly weak wrist joint. C4 injury results in phrenic nerve palsy and paralysis of hemidiaphragm. C8–T1 injury causes Klumpke's paralysis with marked weakness of intrinsic muscles of the hand, finger flexion, and extension leading to claw hand. The sensation is impaired. The involvement of cervical sympathetic outflow is associated with ipsilateral Horner's syndrome and eventually diminished

pigmentation of the iris. Skeletal injuries: Clavicle fracture occurs in about 2 %, while humerus fracture is rare. Usually they heal spontaneously. The spine fractures are rare, but dislocations occur due to rotation of the head on spine [2].

Hypoxic cerebral damage after head delivery occurs in 7 % as umbilical pH falls at rate of 0.04 units/ml [11]. So if not already hypoxic, there is probably 4–6 min safe limit for delivery of shoulder without hypoxic damage. Nearly 1.5 % may require cardiac resuscitation or develop hypoxic ischemic encephalopathy. The incidence of depressed neonates rises sharply after delay of 3 min in delivery.

To combat any emergency, skills need to be updated regularly on mannequins. Labor room staff must follow a standard protocol. The progression to next maneuver should be methodical. The urgent need to release shoulder and the potential damage due to undue force for traction need to be balanced.

Standard Shoulder Dystocia Drill

1. Identify the risk factor.
2. Identify the intrapartum delay in delivery of shoulders.
3. Give liberal episiotomy.
4. Apply gentle downward traction first with maternal own efforts to push downward with suprapubic pressure by the assistant.
5. McRoberts' maneuver along with suprapubic pressure be tried next which is successful in mild to moderate cases.
6. If above fails, do Woodscrew maneuver of delivery of posterior arm.
7. If they fail, then do all four's position delivery.
8. Zavanelli's procedure is done for live fetus only.
9. Symphysiotomy is practically not done.
10. Cleidotomy is recommended for dead fetus only.
11. Availability of anesthesiologist, neonatologist, and expert obstetrician ensured.

Conclusions and Recommendations

1. Mostly shoulder dystocia is not accurately predicted or prevented.
2. Elective induction of labor or elective C.S. for all macrosomic fetus is not appropriate.
3. Elective cesarean is indicated for estimated fetal weight 5000 g in nondiabetic (4500 g in Indians) and 4500 g (4000 g in Indians) in diabetes mellitus.
4. No single maneuver is superior to others in releasing impacted shoulder and reducing risk of injury.
5. When diagnosed, shoulder dystocia drill is recommended.
6. McRoberts' maneuver with suprapubic pressure is a preferred initial approach.
7. Counseling and documentation save from the medicolegal problems.

ACOG [3] Document states:

1. Most cases are not accurately predictable or preventable.
2. Estimated fetal weight, gestational age, maternal glucose intolerance, and severity of previous neonatal injury can be used for decision making of route of delivery, where either route may be appropriate.
3. Elective induction of labor or elective cesarean section for all macrosomic babies is not applicable accurately.
4. Planned cesarean section for nondiabetic women with fetus >5 kg and diabetic with fetus >4.5 kg may be considered.
5. Periodic shoulder dystocia drill for residents and staff of labor suites should be conducted.

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Difficulty in the Delivery of a Baby During LSCS

34

Parul J. Kotdawala and Munjal J. Pandya

Introduction

Over the last three decades, there is a steady rise in cesarean sections globally [1]. This has mainly happened due to expanding indications for primary cesarean section. We now perform elective CS in almost all breech pregnancies; preterm labor; various pregnancy situations such as associated medical problems, e.g., diabetes, hypertension, and immune problems; IVF pregnancies; advanced age pregnancies; and morbidly obese mothers. These higher rates of primary cesarean sections have led to very high repeat cesarean section rates! In almost all recent surveys for indications for CS, "previous cesarean section" has become the number one indication, contributing to almost 40–50 % of CS. The US data also shows a rise from 21 % to 32 % in 15 years [2]. These factors like previous cesarean section, morbidly obese woman, and preterm elective cesarean section have brought in their wake peculiar situations for the delivery of the baby during CS. We have tried to discuss various difficulties encountered in delivering the baby during CS and various means to minimize trauma to the baby as well as to the mother. We have also outlined current concepts and have enlisted suggestions to ease the delivery of the baby with the evidence base. Difficult fetal extraction occurs in approximately one in ten cesarean deliveries, more commonly seen with preterm, elective, and late intrapartum cesarean sections.

In a small survey conducted in our hospital (Smt. SCL Hospital, NHL Mun. Medical college 2003–2004), we found the frequency of indications as previous CS 28.0 %, fetal distress 25.3 %, breech 10.7 %, and CPD 10.7 %.

The various difficulties encountered during delivery of the baby can be listed as following:

1. Abdominal wall issues like previous scars, adhesions, and physical disability in the mother.
2. Problems of access to the lower segment like adhesions due to previous surgery, tumors like fibroid in the lower segment, or cancer of the cervix where trauma to the cervix may upstage the cancer. Uterine malformation, torsion of the uterus, and pre-labor CS where the formation of lower segment is incomplete also contribute to the difficulties.

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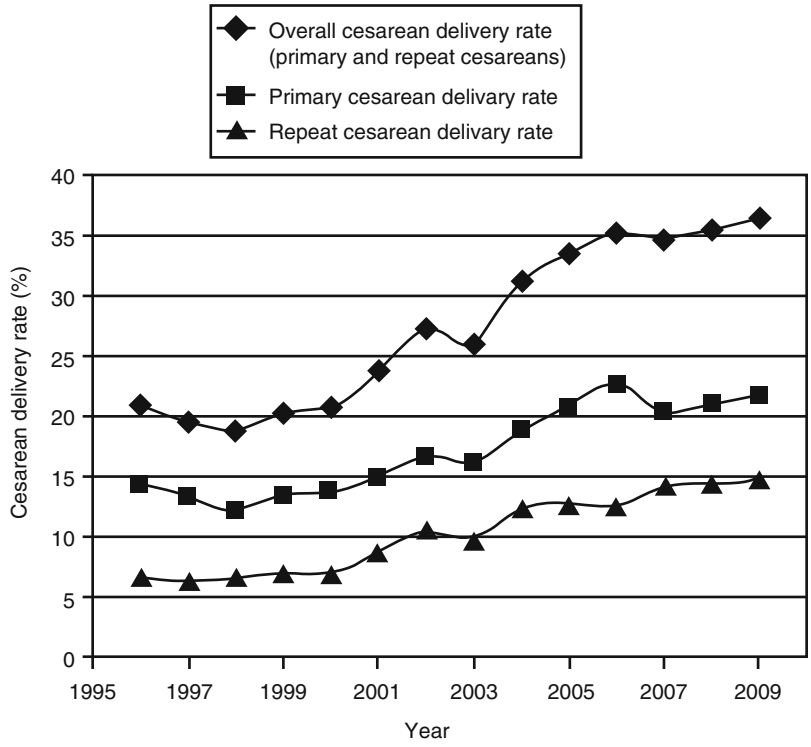

3. A mal-positioned baby, fetus with high floating head or deeply engaged head may also pose problems in smooth delivery. Placenta previa, especially those located anteriorly, would make the delivery of the baby extremely testing!

Abdominal Wall

- Scars over the abdomen: Scars of previous cesarean section/sections or scars of laparotomy may lead to extensive adhesions which can pose problem while entering the abdominal cavity. Inflexible scar tissues may need a slightly bigger incision. Once an adequate sized scar is made, the delivery of the baby is not affected much.
- In general a vertical scar gives easy access to the upper parts of the uterus, but may make access to the extreme lower part of the uterus difficult, especially in an obese woman. A transverse scar gives an easy access to the lower segment, but may not allow access to the upper segment easily. One may employ the incision according to the need of individual case.
- Adhesions: Adhesions due to previous surgeries in the abdomen, due to endometriosis, or due to extensive pelvic inflammatory disease would cause problems to reach the lower uterine segment.

Obesity Recent fact sheet published by WHO has shown alarming trends for global obesity. In 2014, more than 600 million adults, 18 years and older, were obese. In 2014 about 13 % of the world's adult population - 15 % of all women were obese & 40 % of all women were overweight. The worldwide prevalence of obesity more than doubled between 1980 and 2014 [3].

Operative and postoperative complications among obese pregnant women include increased rates of excessive blood loss, operative time greater than 2 h, wound breakdown, infection, and endometritis. Sleep apnea occurring in this group of women may further complicate anesthetic management and postoperative care [4].

For obese women who require cesarean delivery, consideration should be given to using a higher dose of preoperative antibiotics for surgical prophylaxis than a normal-weight woman. Attempts to decrease the incidence of wound breakdowns and infections that have been studied include closure of the subcutaneous layers and the placement of subcutaneous drains. Although suture closure of the subcutaneous layer after cesarean delivery in obese patients may lead to a significant reduction in the incidence of postoperative wound disruption, postoperative placement of subcutaneous draining systems has not shown to be of consistent value in reducing postoperative morbidity. Prophylaxis against venous thromboembolism is vital in obese women due to higher risk, and the use of pneumatic compression, elastic bandages, and medical prophylaxis with unfractionated heparin or low molecular weight (LMW) heparin is indicated. An emergency cesarean delivery should not be delayed to start the medical prophylaxis, but mechanical measures may be employed. Postpartum medical prophylaxis is recommended for patients who are at high risk of venous thromboembolism. As there is higher chance of emergency cesarean delivery and more complications, some resource planning like additional blood products, a large operating table, and extra personnel in the delivery is advisable. The type and placement of skin incision will also vary from routine low transverse incision, and at times one may need to consider placing the incision above the panniculus.

The massively obese group was observed to be at significantly increased risk for delayed delivery and long operative time (emergency cesarean section 32.6 % vs. 9.3 %, prolonged delivery interval 25.6 % vs. 4.6 %, and total operative time 48.8 % vs. 9.3 %, blood loss >1,000 ml 34.9 % vs. 9.3 %, multiple epidural placement failures 14.0 % vs. 0 %, postoperative endometritis 32.6 % vs. 4.9 %, and prolonged hospitalization 34.9 % vs. 2.3 %) [5].

Incision over the abdominal wall beneath the panniculus is avoided so as to prevent wound infection postoperatively. Instead, a supraumbilical approach would give entry to the uterus easily, but cosmetically, it may not look good. The other approach is by lifting the panniculus by a Montgomery strap and putting an incision just above the pubic symphysis, which is cosmetically sound, but it makes access to the uterus difficult [6, 7] (Fig. 34.1).

Conventional wisdom dictated a low transverse incision after pulling up the panniculus by various means and performing the CS and to employ a vertical incision if this was not possible. Both of these had a higher morbidity attached; the low transverse may not be adequate enough for intra-abdominal maneuvers for the delivery of the fetus. It also has a higher chance of post-op infection (due to overlying panniculus reducing aeration

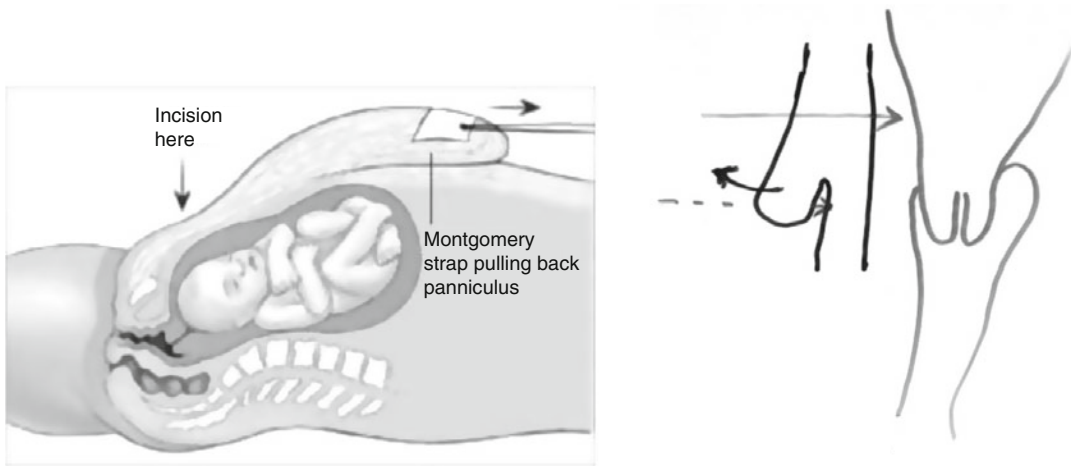

Fig. 34.1 Panniculus

and less drying and irritation due to rubbing). The vertical incision has difficulty in accessing the lower uterine segment, higher rate of disruption, and hernia risk. Current experience has shown that a high transverse incision above the panniculus, after pulling it down as shown in the figure, may be the most appropriate in obese women. When the lower segment has not formed, preterm elective CS, a deliberate transverse incision just below or above the umbilicus, and a fundal delivery of the baby would be the most appropriate. A lower incision and delivery through the lower segment is far more traumatic and risky in comparison to the marginally higher risk (2–3 %) of subsequent rupture. Several recent studies have concluded that if the lower segment seems inaccessible due to large panniculus, it is better to opt for a high transverse incision with fundal delivery for better perinatal outcomes [8–10].

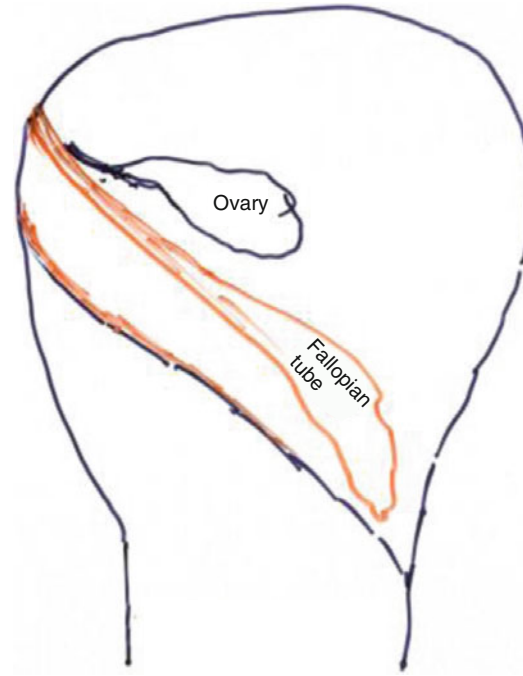

Fig. 34.2 Torsion

Lower Uterine Segment

- *Adhesions*: Adhesions covering the lower uterine segments, omental flaps extending over the fundus of the uterus, and the urinary bladder adhering high up to the upper uterine segment would cause difficulty in putting an incision over the lower uterine segment. A clear delineation of tissue planes is important for a safe delivery. In some cases where very low transverse incision was employed in a previous CS (Pfannenstiel), at times one may find direct contiguous adhesions between the uterus and the abdominal wall. Such adhesions require sharp dissection and may at times damage the bladder.
- *Tumors in the lower segment* (*fibroid, Carcinoma cervix, etc*.): Tumors/fibroid in the wall of the lower uterine segment along with its increased vascularity would prevent an easy entry through it. An incision just above the tumor may work well in accessing the uterine cavity and also for post delivery myomectomy if deemed fit. In a pregnancy with carcinoma of the cervix, one needs to be very gentle in handling lower parts of the uterus to avoid dissemination of the carcinoma as well as to avert significant bleeding in case of direct trauma. A fundal delivery may be the most appropriate route since this woman would never become pregnant again!
- *Torsion*: Generally there is dextrorotation of the uterus. Excessive rotation may lead to torsion of the uterus bringing the uterine vessels anteriorly, as shown in the figure. If an incision is placed without correcting the torsion, inadvertent damage to the uterine vessels may occur. A proper orientation and correction of the torsion are very vital in this rather rare phenomenon (Fig. 34.2).
- *Anterior placenta previa*: An anteriorly placed placenta overlying the lower uterine segment can be a major dilemma. An incision through the placenta is to be avoided, as it leads to fetal blood loss. The fetoplacental unit has a blood volume of just 450 ml, and a minor blood loss of 50 ml may be significant for the fetal health. The aim while dealing with placenta previa should be to cause minimum separation of the

placenta to reach the membranes. Separation of the placenta leads to stoppage of oxygen supply to the fetus, leading to fetal asphyxia. Hence one should either dissect the placenta upwards or downwards, whichever side the placental edge is closer, reach the membranes, and rupture them. A preoperative USG mapping of the placenta is very important to help decide the direction of placental dissection. Generally speaking, if the placenta is covering the internal os, membranes are to be ruptured superiorly (toward upper segment) after putting an incision over the lower uterine segment. If the placenta is in the lower segment, but short of the internal os, the rupture of membranes is performed going down toward the cervix after incision. This has to be a very gentle handling as the lower segment is thin and decidualization makes it prone to tear (Fig. 34.3).

- *Transverse lie*: There will be narrow lower uterine segment in the absence of a presenting part in it. Hence, the incision over this narrow uterine segment would be found inadequate to bring the baby out. An upward extension at the lateral margin of the incision on one side in the form of a "J," or lateral upward extensions on both sides of incision (like flap valve) in the form of a "U" should be made to get adequate opening and to facilitate safe delivery of the fetus. A midline vertical extension of the transverse incision (inverted "T" shape) is tantamount to a faulty judgment and will result in a weak scar and should be avoided (Fig. 34.4).
- *Polio/pelvic trauma*: Polio or accidental pelvic trauma would disrupt the normal pelvic anatomy making it difficult to have access to the baby. One needs to improvise according to the alterations in the pelvis and get safe access to the baby, and no "rule of thumb" plan can be proposed.

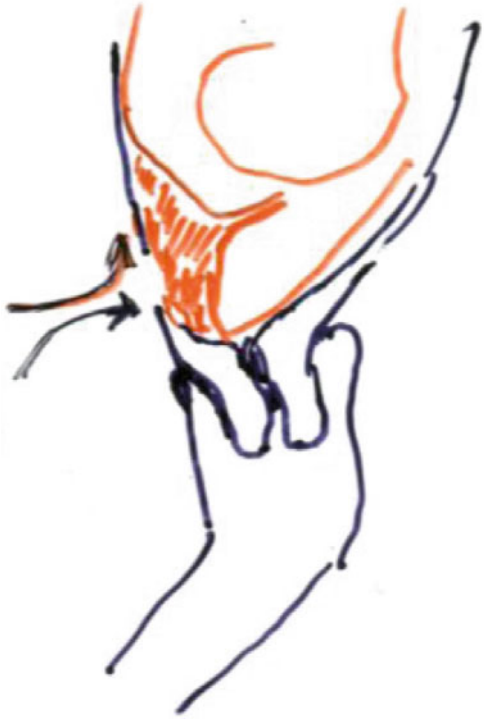

Fig. 34.3 Anterior placenta previa

Difficulties Encountered in Case of Deeply Engaged Head

- *ERR sequence*: Outlined by Andrew Chao [11], ERR sequence is an interesting maneuver for a safe delivery of the engaged head. Although this looks quite complex and a bit too intricate, it is well worth a mention here (Fig. 34.5).

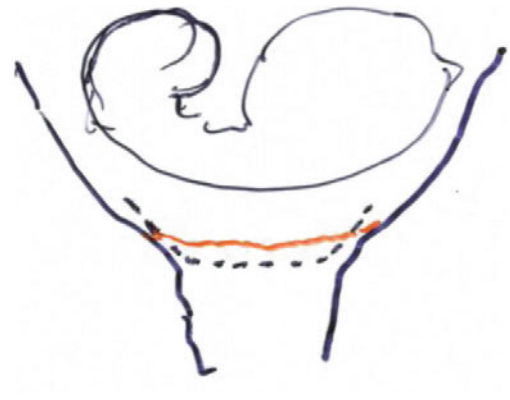

Fig. 34.4 Transverse lie

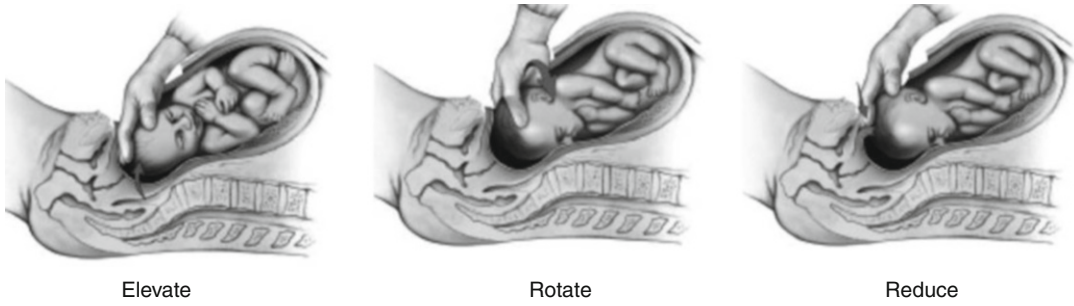

Fig. 34.5 ERR sequence

1. Elevate: Lock the fingers into a quarter-circle around the vertex. Apply traction out of the pelvis with the hand and the entire extended arm.
2. Rotate: Grasp the fetal head between the thumb and fingers and rotate it so the occiput faces the incision.
3. Reduce: Push the lower edge of the uterine incision down until it is posterior to the fetal head.

Too long trial Long trial of labor and failure of vaginal delivery would end up in a deeply engaged head, especially in deep transverse arrest. Baby delivery at cesarean in this situation has difficulty in passing fingers below the head to disimpact and forward pull for delivery. Here a forcible pushing of fingers and hand below the head may be very traumatic with lateral scar extensions and vertical tears toward the bladder. The following options may be employed to deal with this situation:

Push the head up from the vagina In this an assistant remains at the vaginal end between two legs. A Whitmore position is employed to increase the inlet dimensions to facilitate disengagement of a jammed head. As shown in the figure, Whitmore position leads to pressure on the acetabula and opening of the pelvic inlet. This is a modified lithotomy position where thighs are moderately abducted and flexed to approximately 135° relative to the trunk [12] (Fig. 34.6). The moderately abducted thighs would press the bilateral acetabula which results in more opening up of the pelvic cavity which can allow the push from below for delivering deeply engaged head. The vaginal hand pushes the head up out of the pelvis which can then be flexed and delivered by the abdominal hand (Fig. 34.7).

Intravenous nitroglycerin IV nitroglycerin bolus has been tried successfully to relax the uterus temporarily. Once the uterine muscle relaxes a little bit, one may be able to glide fingers below the head and dislodge it for a smooth delivery. An IV bolus of nitroglycerin (0.25–0.5 mg) will relax the uterus for approximately 20 s, long enough to pass fingers below the head. The anesthesiologist needs to be taken in to confidence as a short but steep dip in blood pressure is anticipated. Nitroglycerin does decrease the blood supply to the uterus, but the bolus dose has a transient effect which doesn't cause any fetal hypoxia. Intraoperative nitroglycerin application during cesarean section has no unfavorable effect on the condition of newborns <32 weeks or between 500 and 1,500 g. The incidence of intraoperative maternal blood loss >1,000 ml was not increased. Differences in the interval between nitroglycerin application and cutting of the umbilical cord have no clinically relevant effects on Apgar scores or arterial umbilical pH [13, 14].

Pull from above Patwardhan described two maneuvers for different situations [15] (Fig. 34.8):

1. *Back anterior*: If the back is anterior and the head is deeply engaged, one needs to deliver

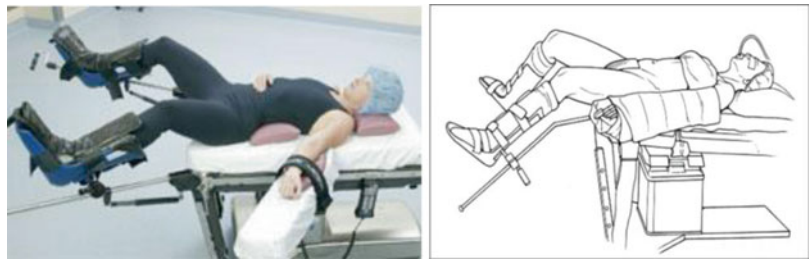

Fig. 34.6 Modified lithotomy position

Fig. 34.7 Push from below

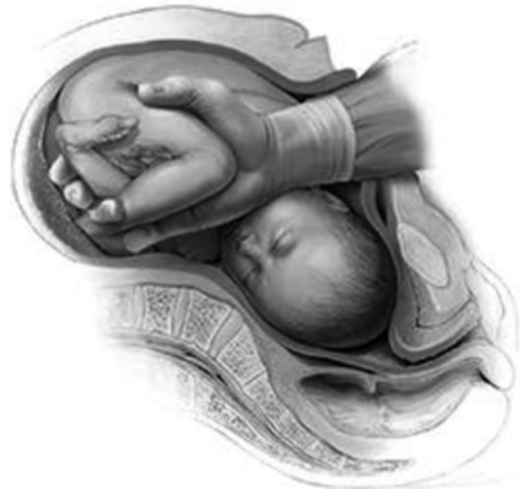

Fig. 34.8 Pull from above

one hand and the shoulder out of incision, to be followed by the second hand and the shoulder. Thereafter, the further pull in the grove of the abdomen will double up the child, and gradually the lower back, buttocks, and the legs will be delivered. Subsequent pull on the baby will bring out the head at last.

2. *Back posterior* (*reverse breech delivery/foot extraction method*): If the back is posterior and the head is deeply engaged, the feet are in the front. Passing the hand up from the abdomen and pulling down the feet is easy, followed by the buttocks, and the head is delivered at the end.

Back lateral In almost a similar way, the operator's hand is passed to the opposite side, and the foot is grasped and pulled down and out. The buttocks, trunk, and the head will follow.

Pull vs. Push In general it has been found that a push from the vagina is more traumatic to the baby as well as to the genital tract [16–20]. A pull from above, by pulling at the foot (reverse breech extraction), is safer for both the newborn and the mother. Several studies have confirmed this.

Short Simpson's Forceps

Vectis Mechanical disengagement of the fetal head has been tried since ages (Fig. 34.9). A thin metal blade of an instrument occupies much lesser space than fingers. A "spoon-shaped device," also called a "Vectis," works

quite well. The blade is passed between the lower segment and the fetal head till the device handle touches the symphysis pubis. Further sliding leads to fulcrum effect at the symphysis and lifts the fetal head up and anteriorly. Simultaneous fundal pressure will lead to delivery of the head. To facilitate easy insertion, a device with a hinge is also available. Vectis with a flat blade avoids tissue locking into the fenestrum. Murless head extractor is also an innovative design of a Vectis [21] (Fig. 34.10). In the absence of a special device, a single short straight blade of Simpson's forceps can also be used effectively.

Head disengaging device In a bid to disengage a deeply impacted fetal head from the vagina, a new device has been developed and tried in the UK. Known as "Fetal Disimpacting System," the device is used vaginally. The deflated device is folded and placed just above the pelvic floor. It is then distended by injecting into it 100 ml saline (range 60–120 ml). A study showed mean elevation of 3 cm with this [22] (Figs. 34.11 and 34.12).

A simple but innovative device (Snorkel) to disengage a deeply engaged head is worth a try (Figs. 34.13 and 34.14). This simple disposable device has a flat pad with multiple holes, which is attached to a tube to blow air into it. The flat pad is inserted vaginally, and is guided posteriorly, between the head and the genital tract. Once in place air is blown into the tube, creating an air pocket below the head, allowing easy passage of fingers below the head during CS [23].

The problem of dealing with deeply engaged head or a CS during stage II of labor can be very taxing. In a recent survey among resident doctors in the UK, it was confirmed that a majority of them were not confident of dealing with this situation. The sentinel audit report published by the RCOG recommended a consultant's presence whenever cesarean section is performed at full dilatation. It also goes on to say "Proper training of resident doctors should be done for delivery of deeply engaged head," underlining the need for special emphasis on this skill development in residency training program [24].

Fig. 34.9 Vectis

Fig. 34.10 Murless head extractor

Floating head Difficulties encountered in case of floating head can be due to an elective pre-labor CS, too large head, preterm fetus, hydramnios, placenta previa, etc.

To ease up the head delivery, the first option is to induce uterine contractions to facilitate descent and expulsion. One should rupture the membranes and let the liquor drain out. The reduction in the volume inside the cavity will bring about uterine contraction. A simultaneous oxytocin infusion will help augment these contractions. A predelivery infusion of dilute oxytocin may achieve the same results, but care must be taken to avoid uterine hyperstimulation and resultant fetal compromise. Since the head is difficult to grasp and pull with a gloved hand, either pulling devices like vacuum extractor or obstetrical forceps may be employed, or the foot

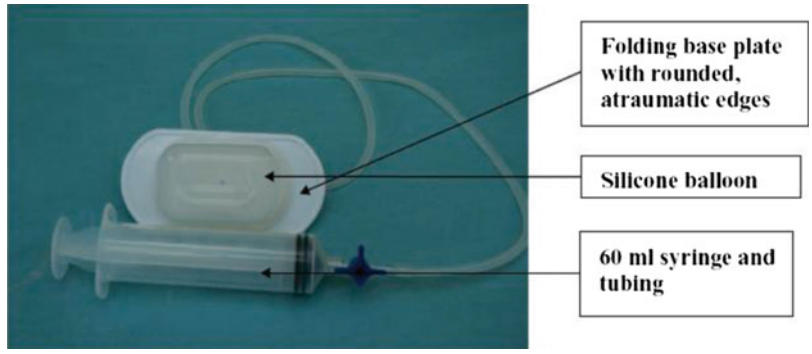

Fig. 34.11 Head disengaging device 1a

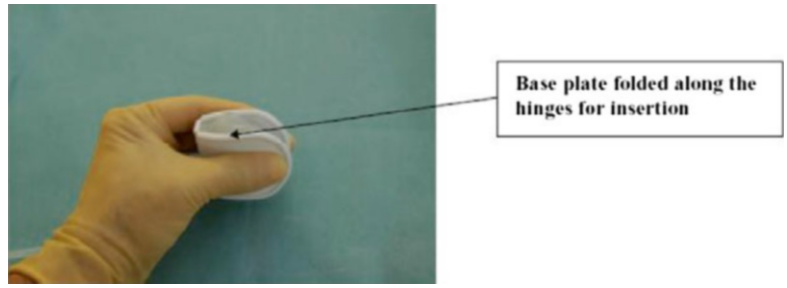

Fig. 34.12 Head disengaging device 1b

Fig. 34.13 Snorkel 1a

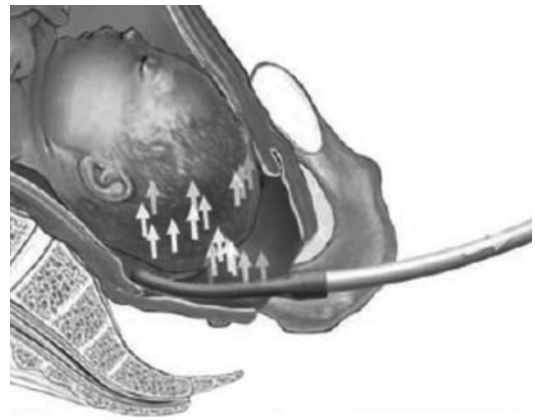

Fig. 34.14 Snorkel 1b

extraction by reverse breech delivery may be employed. In case of foot extraction, one may need to act swiftly, and should not let much drainage of liquor, to allow the fetal somersault during the delivery!

Forceps/vacuum Both forceps and vacuum have been tried for delivery of a floating head.

Forceps: Short Simpson's forceps without a pelvic curve is the best suited instrument for head delivery (Figs. 34.15 and 34.16). Generally the head will be in one of the transverse positions. Hence there will be a posterior and an anterior application. The anterior application can be difficult at times. The Barton's forceps with a hinged anterior blade is being proposed as a great tool to avoid this difficulty of application! The shank angling is also beneficial in easy application than straight shanks of a Simpson's forceps.

After application, one should rotate the face anteriorly (occiput posterior) in a bid to reduce the transverse dimension of the head, and then pull out in a rotational arc toward the chest of the mother. Some colleagues rotate the face first to the anterior by inserting a finger in the mouth of the baby holding firm, and using a direct lateral application of the forceps blades on each side of the head! A direct pull out in transverse is also quite reasonable as in routine CS the head is delivered in a transverse position.

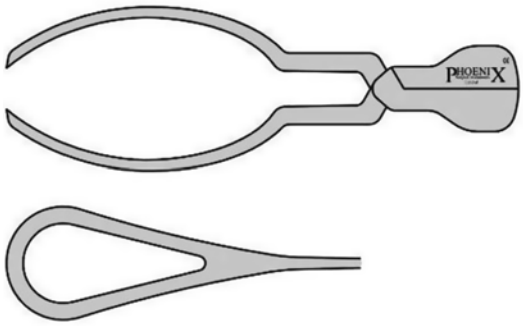

Fig. 34.15 Forceps

Fig. 34.16 Forceps

Barton's forceps An effective aid in cesarean deliveries. The unique qualities of this classic medical instrument make it an effective, ergonomic option for cesarean deliveries involving a high transverse position of the fetal head [25] (Fig. 34.17).

Vacuum Vacuum delivery of the floating head seems very plausible (Figs. 34.18 and 34.19). But the correct application is very vital. Otherwise it may harm the fetus rather than facilitate the delivery. A correct application would be on the flexion point, the point at which the mento-vertical diameter crosses the sagittal suture, promoting flexion of the fetal neck. This will result in lesser traction force required to deliver the baby. A misplaced cup is the cause of majority of the complications. As most of the vacuum cups are designed for vaginal use have the pulling direction perpendicular to the device, their use during CS where the traction angle is almost a tangent, is ineffective. This leads to situations where the cup either slides over the scalp or it comes off due to the angle of the pull. To help an easy and optimally directed pull, special vacuum cups for CS are now designed. The Omni C is one such cup. The figure itself is self explanatory! While applying the vacuum cup to

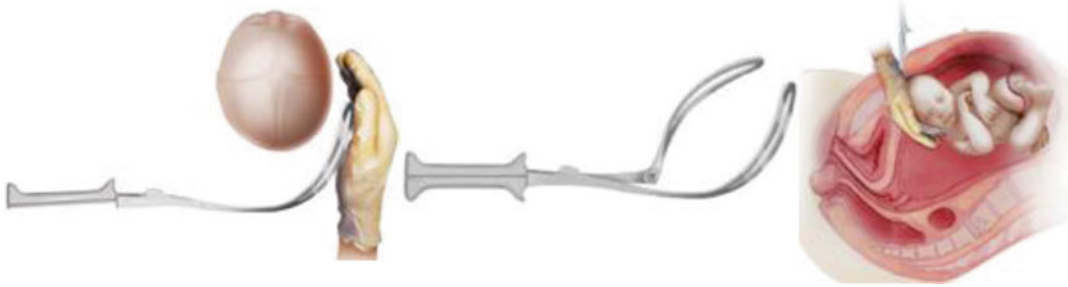

Fig. 34.17 Barton's Forceps

Fig. 34.18 Compare vacuum cup design for vaginal delivery and CS application

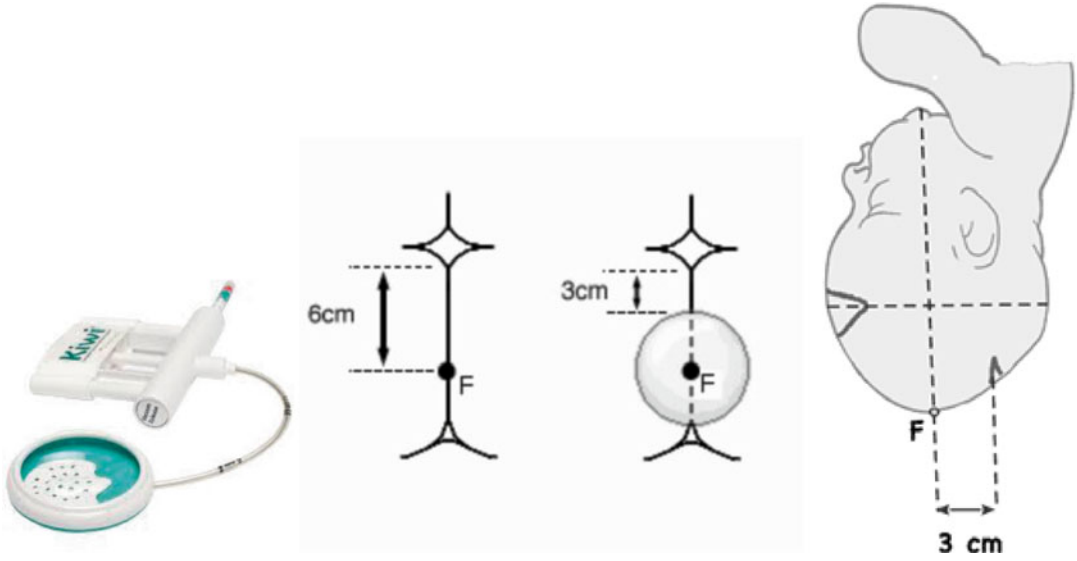

Fig. 34.19 Vacuum cup and flexion point

the scalp of the fetus, a tedious interphase of liquor may test the patience of the operator [26] (Fig. 34.20).

Kiwi Omni C cup is a type of rigid "posterior cup" indicated for cesarean section.

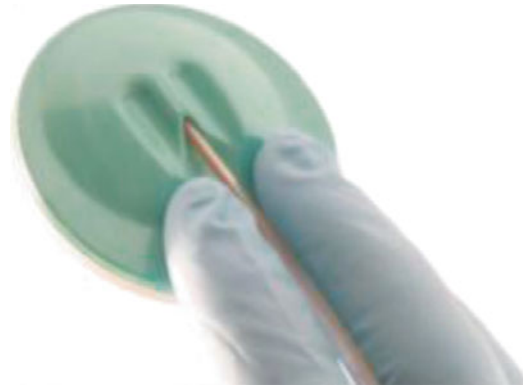

Fig. 34.20 Kiwi Omni C cup

Malpresentations

Transverse lie (1:300 deliveries): There can be a curve of the fetal spine oriented upward (dorso-superior) in which fetal small parts present at the cervix, or the curve of the fetal spine can be oriented downward (dorso-inferior) in which fetal shoulder presents at the cervix. In dorso-superior the delivery is easy as the feet are lying in the vicinity of the cervix. A direct pull on one of the feet and gradual delivery will be easy. But the more difficult situation is dorso-inferior. Here if the surgeon is sure of the lie of the fetus, he

should push the head of the fetus up toward the fundus before putting an incision on the uterus. With an assistant pushing the head up, the buttocks will come closer to the cervix and will help gripping one of the feet or insert a finger in the groove of the breech. Otherwise, the foot principle (reverse breech extraction) is the best option. Sometimes, if the lower segment is too narrow, a lower segment vertical incision on the uterus may be an option.

Breech presentation is one of the easiest presentations to deliver at cesarean section, even easier than a cephalic presentation. One can always track the foot from the buttocks and pull it out and thereby conduct delivery. Breech complicated with 1 ft in the vagina of locked twin would need extra caution so as to avoid any fetal trauma/maternal tears.

Malformed uterus Malformed uterus generally leads to nonvertex presentations. So, very close deliberate examination to confirm not only fetal lie but position of the back and feet becomes vital in conducting delivery. A general rule of thumb of following foot and delivering the baby would be least traumatic to baby as well as to the uterus.

Twins/multiple pregnancy Clear detailed examination to know the lie and position of the first fetus and possibility of locking of the second fetus is to be anticipated. "First breech and second vertex" twins are the most high risk for locking, and this should be anticipated in this combination [27]. Generally, in case of the second fetus, rupture of membranes followed by foot extraction is a preferred mode of delivery because of the time lapsed already in the delivery of the first fetus. The second fetus is always at disadvantage because after the delivery of the first fetus, uterine contractions lead to reduction in placental perfusion and any delay would aggravate compromise in fetal oxygenation, so a prompt delivery of the second fetus is always planned. An oxytocin infusion is started soon after the delivery of the first baby to help quicker delivery as well as to reduce the chance of PPH.

Overstretched Lower Segment (Bandl's Ring)

This rather rare and curious condition can create problems in the delivery of the baby during CS (Fig. 34.21). Due to extended labor and prolonged drainage of liquor amnii, the upper segment of the uterus retracts and thickens, leading to overstretching of the lower segment. This differential thickness is pronounced at the junction known as Bandl's ring. This narrowing leads to holding of fetal parts above it at the time of delivery. A forcible delivery through this ring may produce trauma to the uterus. It is vital that either the ring is reduced by giving uterine relaxants like nitroglycerin, or the ring is deliberately cut at its anterior part for a safe delivery. Hence a lower segment vertical incision on the uterus extending and cutting through the ring is the most appropriate method of delivery during CS.

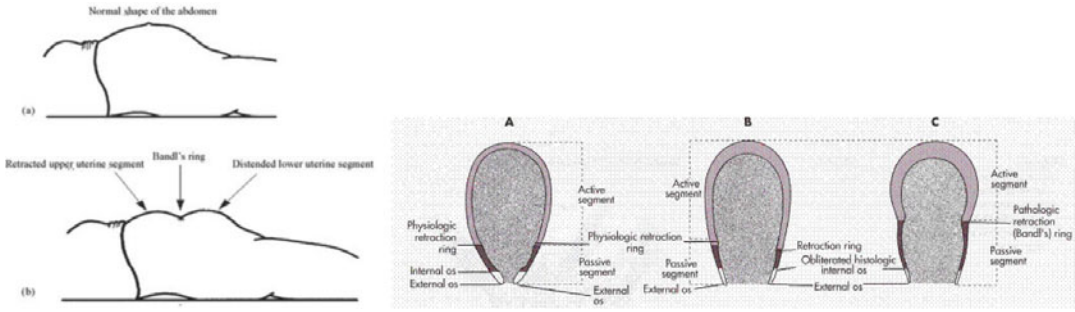

Fig. 34.21 Bandl's Ring

Conclusion/Summary

The rates of cesarean sections are on the rise. The delivery of the fetus at Cesarean section can be testing, and in approximately 10% of cesarean sections, the operator encounters difficulty in the delivery of the fetus. The rising rates of primary cesarean sections have contributed to a large number of repeat cesarean section. These repeat C sections, along with preterm, elective & late intra-partum C sections lead to higher possibility of difficult fetal extraction

- Access to abdominal cavity has to be planned ahead in consultation with the patient.
- An inaccessible lower segment is not an end of the world situation. Upper segment C section can be a valid option & should be seriously considered, if serious harm is anticipated in accessing the lower segment.
- Floating head should be allowed to descend by letting the liquor drain and let the uterus contract by using oxytocin. A delivery by pulling at & delivery of the foot first, or use of vacuum/forceps will help.
- Deeply engaged head: A semi-lithotomy position, reverse breech extraction, use of I/V nitroglycerine, and use of disengaging devices are safer options.
- Obese women: A horizontal incision above the panniculus, at times going supra-umbilical will facilitate the smooth delivery. A fundal delivery of the baby may be considered.
- In cases of anterior placenta previa a detailed mapping of its margins will help decide the side where the membranes may be accessed with minimal separation of placenta & avoid incising the placenta.
- An emphasis on training the resident doctors for intra-partum C sections will increase their confidence and efficiency.

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