Alok Sharma
Editor

Labour Room Emergencies

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Editor
Alok Sharma
Civil Hospital
Mandi
Himachal Pradesh
India

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"To dear God, whose eternal blessing and divine presence helps us to fulfil all our goals"

This textbook is dedicated to my family and my patients.

Preface

I have welcomed, with enthusiasm, the opportunity to edit this very first edition of the text "Labor Room Emergencies". I have tried to provide a text to help undergraduates, postgraduates, and practitioners to clarify their doubts and to organize some of the intricacies regarding the subject.

I have assembled a group of contributors, nationally and internationally, with an exceptionally broad range of backgrounds and similar interests. All the contributors have emphasized the latest clinical management approaches with deep understanding. They have all contributed to the same goal; that is, to create a source of information that will help in the care of women in the last phase of pregnancy, during labor, and even after delivery.

This text deals with the medical, surgical, and gynecological complications that occur in a patient in the labor room. No two births are the same and no two mothers have the same changes during pregnancy, labor, and puerperium; therefore, it is important to have a text that deals with planning and anticipating any possible complications during these phases of a woman's life.

I have tried my level best—employing a multi-disciplinary approach to the treatment of patients with various disorders—to cover all the relevant aspects pertaining to these phases where obstetricians may need guidance in decision making.

I hope that the aggregate of my efforts has resulted in a text that will be a worthy resource for those who want to care for pregnant women.

Mandi, Himachal Pradesh, India — Alok Sharma

Acknowledgments

Giving thanks is a pleasant job, but it is, nonetheless, difficult when one sincerely tries to put such ideas into words. The following humble words of expression and gratitude cannot really convey the deep feelings of my heart.

In any attempt to create and produce a textbook of obstetrics and gynecology, one must be fortunate enough to have the assistance and support of many talented professionals, both within and outside the obstetrics and gynecology department. To begin with, I am indebted to all my authors for their generous contributions to this book; all of them, despite their busy schedules, provided recent, up-to-date, and evidence-based chapters on various aspects of labor room emergencies. I have selected contributors from different parts of India and abroad, and from various specialities and organizations. I am especially thankful to Dr Naren Aggrawal for his inspiration; he always provided continuous support for the conception of this book. It again has been a pleasure to work with the dedicated professionals from Springer. This publisher has been gracious in offering support without any interference whatsoever, and the team has also ensured that the quality of work is superb.

I am deeply indebted to Dr Eti Dinesh, Senior Editor, for her unconditional support; she has brought her considerable intelligence, energetic work ethic, and creativity to this edition. Her dedication to creating the best textbook possible equalled my efforts to produce an appropriate style for the textbook.

This textbook would have not seen the light of the day without the untiring efforts of Mr Kumar Athiappan, Editorial Co-ordinator, who skilfully kept my project on track through an array of potential hurdles.

I acknowledge my respected parents, Smt. Dhanwanti Devi Sharma and Hans Raj Sharma, who have laid the foundation stone of literacy in me and given me the courage to face the challenges of the world by inculcating good attributes in me.

I thank my wife, Dr Pratibha Sharma, for her tolerance and understanding of the time I spent away from her during my own career and in editing this textbook. Her constant encouragement, moral support, and love has been a source of inspiration to me for completing this work.

I thank my brother, Dr Amit Sharma, for guiding me at every step of the way in shaping this book.

Finally, but certainly not last, I thank my lovely daughter, Hiranya, for her immense patience.

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About the Editor

Alok Sharma was awarded an **MBBS** by Indira Gandhi Medical College (IGMC), Shimla, in 1997. He received a **postgraduate degree in Obstetrics and Gynaecology** in 2010 from Kamla Nehru State Hospital for Mother and Child, IGMC, Shimla, Himachal Pradesh, India, where he currently works as a **registrar**. He is the **founder secretary of the Indian Menopause Society, Shimla Chapter, and also of the Indian Fertility Society, Himachal Pradesh Chapter**. He is **national coordinator** of the **Federation of Obstetric and Gynaecological Societies of India (FOGSI) Sexual Medicine Committee** and **North Zone coordinator** of the **Perinatology Committee, Safe Motherhood Committee, MTP Committee and Public Awareness Committee of FOGSI.** He is an active member of various academic and professional bodies. He has **edited two books on obstetrics** for postgraduates in obstetrics and gynaecology and has **published several articles** in various national and international journals. He is the author of numerous book chapters and has participated in a number of national and international conferences and workshops. He is not only active clinically but also in promoting women's role in society. He is the recipient of **BEST CITIZENS OF INDIA AWARD, 2015**, as well as the **Shimla Gaurav Award**, for his contribution to medical science.

Part I

General Principles

Labor Ward: An Introduction

1

Divya Yadav Sharma, Alok Sharma,
and Spoorthi Prakash

1.1 Introduction

Reducing maternal and infant mortality is an integral part of national development. Globally, 830 women die every day in childbirth [1], most being from poor and rural backgrounds in developing countries. A woman's lifetime risk of maternal death is 1 in 4900 in developed countries and 1 in 180 in developing countries, and this difference is mainly due to inequities in access to health facilities. The global maternal mortality rate is 212/100,000 and in India the rate is 174/100,000 [1]. The infant mortality rate is 32/1000 live births globally and in India the rate is 40.5/1000 live births [2]. With the global implementation of the World Health Organization millennium development goals, and with the implementation of various programs by the Indian National Government, the emphasis in childbirth is on institutional deliveries. In India, in rural areas and even in a few urban areas, traditional birth attendants still conduct deliveries at home, and the lack of sterile instruments and clean surroundings, as well as improper handling techniques, leads to sepsis, jeopardizing the health of both the mother and the baby. As health schemes have been emphasizing the need for 100% institutional deliveries, it is important to know about the place where all these deliveries occur, i.e., the labor room. Not all rooms can be considered as labor rooms, as certain guidelines need to be followed for a labor room, e.g., for its layout, sterilization techniques, necessary equipment and drugs, and waste management. These guidelines are discussed in this chapter.

1.1.1 The Labor Room

The labor room is a place equipped for conducting deliveries. It should be spacious, with cross ventilation. There are two concepts of a labor room, the preferable choice being a labor delivery recovery room, allowing the patient to stay from the beginning of labor to delivery and for 4 h postdelivery for maternal recovery. But in developing countries, where there are large numbers of patients and few beds available for total admission, a conventional labor room concept is adopted, where the patient is placed on a delivery table on full dilatation of the cervix and moved back to the ward after delivery. Adequate lighting, a continuous power supply, a hand-washing station, an examination table, and a baby resuscitation area are essential requirements for such a labor room (Fig. 1.1).

Facility of neonatal intensive care unit with pediatrician availability is ideal for labor room.

D. Y. Sharma (✉) · S. Prakash
S N Medical College, Agra, India

A. Sharma
Kamla Nehru State Hospital for Mother and Child, Indira Gandhi Medical College, Shimla, HP, India

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b

Labor room

General
- Stethoscope
- Fetoscope/handheld fetal Doppler
- Protein urea test kit
- Glucometer
- Digital BP instrument
- Refrigerator

Delivery related
- Delivery table
- Headlight
- Autoclaved delivery tray
- disposable sterile gloves
- Disposable utility gloves
- Mops with stand
- Autoclave
- Sterilized swabs
- Universal precaution kit

Consumables like syringes, dripsets, thread needles

For baby
- Radiant warmer
- Pediatric resuscitator bag with masks
- Baby weighing scale
- Mucus extractor
- Plastic cord clamp

Fig. 1.1 (**a**) Labor room. (**b**) Equipment and accessories required for a labor room

The essential set of labor room (Fig. 1.1a, b) equipment for initial examination includes a stethoscope (Fig. 1.2), a sphygmomanometer (Fig. 1.3), a wall clock, a thermometer, and a measuring tape. For examining the fetal heart, a fetoscope or handheld Doppler device can be used. For high-risk patients in labor, a cardiotocography machine (Fig. 1.4) is used for initial or continuous fetal heart rate monitoring. A partogram should be maintained. Urine dipsticks (Fig. 1.5) are used for initial diagnosis in pre-eclamptic and eclamptic patients. A glucometer (Fig. 1.6) is required for testing maternal glucose levels (Figs. 1.7 and 1.8).

1.1.2 Neonatal Resuscitation

The following algorithm for neonatal resuscitation shows new guidelines for the resuscitation

Fig. 1.2 Cardiotocography machine

Fig. 1.3 Sphygmomanometer

of, primarily, newborn infants transitioning from intrauterine to extrauterine life. The recommendations are also applicable to neonates who have completed newborn transition and require resuscitation during the first weeks after birth. Most of the neonatal resuscitation guidelines remain unchanged from 2010, but there is increasing focus on umbilical cord management, maintain-

Fig. 1.4 Stethoscope

Fig. 1.5 Urine dipstick with matching code on the box

Fig. 1.6 Glucometer

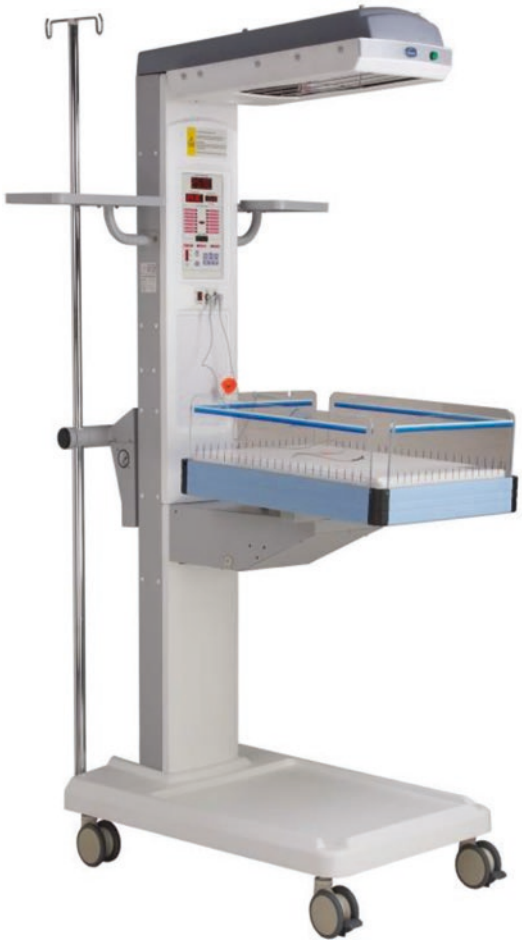
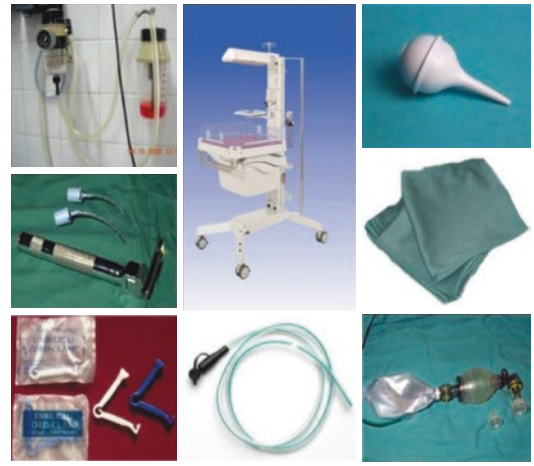


Fig. 1.7 Radiant warmer: Even area for resuscitation

ing a normal temperature after birth, accurate determination of heart rate, and optimizing oxygen use during resuscitation, while routine suctioning for meconium in non-vigorous newborns is de-emphasized. The etiology of neonatal arrest is almost always asphyxia, and therefore the establishment of effective ventilation remains the most critical step [3, 4]. This should be displayed in the labor room for the education of all the staff.



- Overhead warmer with Light source
- Mucus extractor
- Laryngoscope with Endotracheal (ET) tubes
- Suction catheter
- Infant feeding tube
- Cord clamp
- Baby Sheet
- Ambubag

Fig. 1.8 Instruments for infant? Resuscitation

1.2 Equipment for Delivery

A comfortable bed with a headlight should be available with mackintosh to calculate blood loss (Fig. 1.9b). The delivery tray should be autoclaved and should contain a Sims speculum, episiotomy scissors along with 1% xylocaine solution, sponge holding forceps for umbilical cord clamping, and sterile sponges. A tray with clean towels should be available for receiving the baby (Fig. 1.10).

It is essential for the labor room to be equipped with emergency drugs for the management of high-risk cases, as well as for the management of obstetrical emergencies (see Fig. 1.11).

Fig. 1.9 (**a**) Episiotomy instrument. (**b**) Mackintosh usage for calculation of blood loss

Fig. 1.10 Labor table with headlight

Fig. 1.11 Drugs used in the labor room

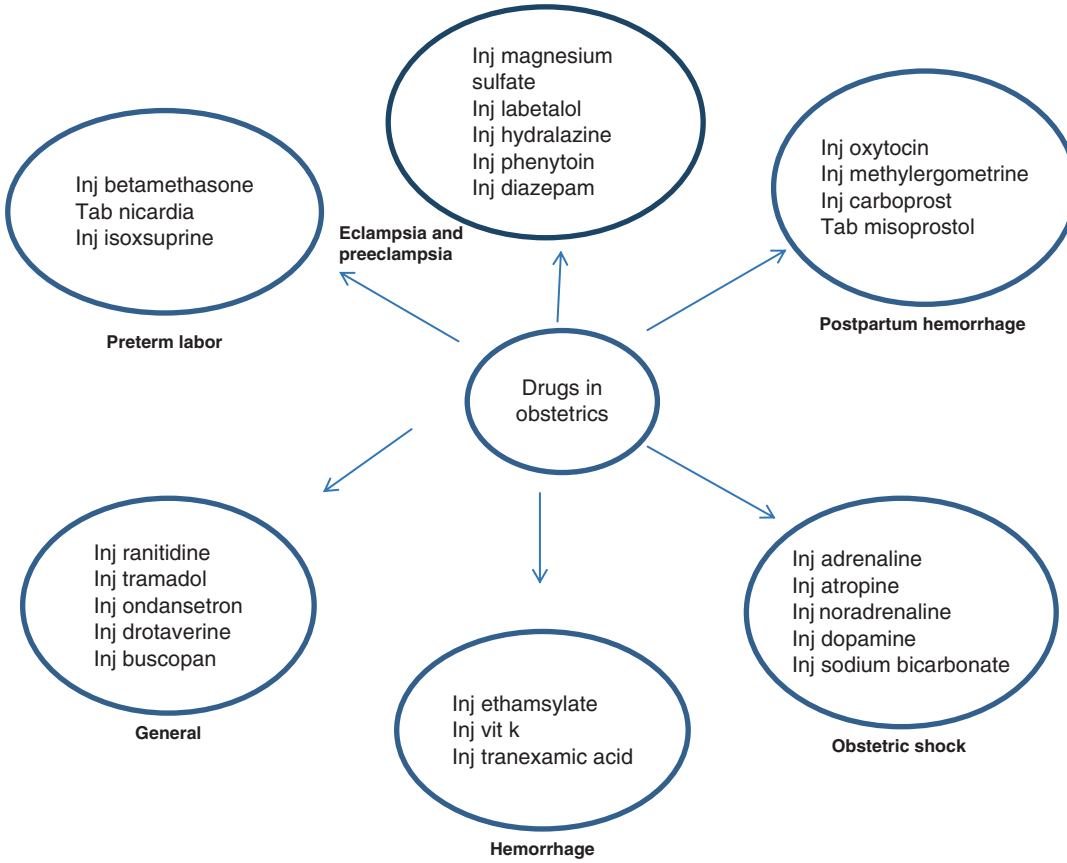


Fig. 1.11 (continued)

1.3 Labor Room Staff, Ethics, and Record Maintenance

The labor room staff should consist of an obstetric consultant for the management of high-risk cases, junior doctors for monitoring, staff nurses trained in JSSK and as skilled birth assistants for assisting deliveries, and workers for cleaning and maintenance of hygiene. The number of staff required depends on the number of beds and the facilities available. Ethics should be maintained in the labor room, including obtaining informed consent when necessary from the patient and her relatives; good behavior by staff and their encouragement of the patient are vital.

1.4 Record-Keeping

Admission records, birth records, and death records should be maintained.

1.5 Family Planning

Each patient should be counseled for birth spacing and advised according to what has been termed the cafeteria method (i.e., anything that works). If the patient is willing, facilities for the insertion of a post-placental intrauterine contraceptive device (IUCD) should be available; IUCD insertion records should be maintained.

1.6 Maintenance of Sanitation

Floors should be vacuum cleaned or washed with disinfectant; delivery instruments should be autoclaved; furniture bed stands should be cleaned with detergent, phenol, or 2% lysol solution; and mattresses and pillows should be covered with water-impermeable covers and should be washed with detergent or disinfected with phenol when necessary. Trolley tops should be wiped with

Fig. 1.12 Hand-washing

Fig. 1.13 Color-coded bins

warm water and detergent to remove dust. Bowls should be autoclaved. The labor room should be cleaned after each delivery.

Hand-washing (Fig. 1.12) is very important as it pertains to getting rid of microorganisms and reduces the chances of sepsis.

Color-coded dustbins (Fig. 1.13) aid in waste management [5]. Human anatomical waste and soiled wastes, expired or discarded medicines, and chemical wastes have to be disposed of in yellow dustbins. Recyclable wastes generated from bottles, iv tubes and iv catheters, and gloves have to be disposed of in red dustbins; waste sharps are to be included in white translucent bins; and broken glass, including medicine vials and ampoules, should be discarded in blue bins.

1.7 Conclusion

For a healthy baby and a healthy mother, it is essential to have a healthy, clean, and well-equipped labor room with skilled and well-behaved staff.

References

1. www.who.int/mediacentre/factsheets/fs348/en/.
2. https://www.unicef.org/media/media_pr_infantmortality.html.
3. nrhm.gov.in/nrhm-components/rmnch-a/maternal-health/guidelines.html labor room guidelines.
4. Australian Resuscitation Council, Guideline 13.1; Introduction to Resuscitation of the Newborn Infant, February 2006.
5. unicef.in/Story/1176/Transforming-Labor-Rooms.

2 Infection Prevention and Waste Disposal

Anuradha Sood, Tarun Sharma, and Aradhna Sharma

2.1 Introduction

Nosocomial infections (hospital-acquired infections, healthcare-associated infections) are a significant cause of morbidity and mortality. They are defined as those infections acquired by a person in a healthcare unit which are secondary to the patient's original condition. These infections also include infections acquired by the patient but appearing after discharge. The symptoms usually appear **after 48 h** of admission. It is estimated that **5–10%** of patients admitted for acute care and emergency procedures acquire healthcare-associated infections (HAI). Of more concern is that more than 70% of infections are due to antibiotic-resistant organisms.

Pregnant females and their foetuses are at an increased risk of acquiring HAIs. All labour rooms, obstetric emergency evaluation areas and operation theatres should have facilities for infection control as they can get puerperal sepsis, neonatal sepsis and other infections acquired during delivery. HAIs also include occupational injuries and infections which a healthcare worker (HCW) can get by virtue of his/her occupation. Needlestick injuries and injuries from other sharps results in approximately 400,000 cases each year. So, infection control measures should be of top most priority for all HCW (doctors, nurses, midwifes, safai karamcharis, etc.).

2.2 Importance of Infection Control in Obstetrics

India accounts for about **1/5 of all maternal deaths and 1/3 (approximately 30%) of all neonatal deaths**. Puerperal sepsis is a fairly common entity especially in the rural and backward areas of India. Most maternal and neonatal deaths occur in the first 7 days after delivery. It accounts for about 19.2% deaths, and it is the second leading cause of death in mothers after haemorrhage and anaemia. In the mother sepsis may lead to blood stream infections, endotoxic shock, peritonitis and abscess formation.

Infections in neonates include neonatal septicaemia, decreased Apgar score and pneumonia. Increased susceptibility of sepsis is more marked in low birth weight neonates (<2500 g) and very low birth neonates (<1500 g). This is due to their poor immune defences and weak cellular and humoral immunity. Vascular or urinary catheters, other indwelling lines or contact with care givers who have bacterial colonization are additional factors contributing to neonatal septicaemia.

A. Sood (✉)
Department of Microbiology, DRPGMC, Kangra at Tanda, Kangra, Himachal Pradesh, India

T. Sharma
Department of Medicine, DRPGMC, Kangra at Tanda, Kangra, Himachal Pradesh, India

A. Sharma
Department of Pharmacology, SLBSGMCH Mandi at Ner Chowk, Mandi, Himachal Pradesh, India

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Table 2.1 Differentiating features of EOS and LOS

Feature	EOS	LOS
Time of appearance	Infection **within 72 h** of birth	Infection **after 72 h** of birth
Mode of transmission	**Vertically** from mother to infant before or at time of birth (i.e. HBV,HIV, TORCH)	At time of birth or **during hospital stay**
Microorganisms	Includes organisms **harboured in genital tract** or which can cross the placenta (e.g. group B streptococcus, *E.coli*, CONS, *H. influenzae*, *Listeria monocytogenes*)	Includes mainly organisms acquired **from the environment** (e.g. *Staphylococcus aureus*, CONS, *E.coli*, *Klebsiella* spp., *Pseudomonas* spp., *Candida* spp., *Acinetobacter* spp.)

HBV Hepatitis B virus, *HIV* human immunodeficiency virus, *E. coli Escherichia coli*, *CONS* coagulase-negative staphylococcus, *H. influenzae Haemophilus influenzae*, *TORCH Toxoplasma gondii*, *Rubella virus*, *Cytomegalovirus*, Herpes virus

Neonatal sepsis is broadly divided into two main categories: early-onset septicaemia (EOS) and late-onset septicaemia (LOS). The main features of the two are tabulated in Table 2.1.

2.3 General Measures to Prevent Spread of Infections in Emergency Obstretic Care

To reduce the spread of infections in both baby and the mother, the following have to be kept in mind:

1. Infection control measures during admissions.
2. Availability of clean environment, clean equipment and other supplies.
3. Availability of trained and skilled staff.
4. Hand washing.
5. Biomedical waste management.
6. Safety from sharps.
7. Safe blood transfusion.
8. Measures to prevent tetanus.
9. Neonatal resuscitation facilities.

2.4 Infection Control Measures During Admissions

1. Proper initial assessment of every patient should be done very carefully. If any patient is assessed to be suffering from contagious illness (measles, rubella, chicken pox, etc.), separate room for delivery should be made available. The staff of nursery/NICU (neonatal intensive care unit) should be notified simultaneously so that adequate neonatal facilities are arranged well in time.
2. Isolation facilities are also necessary for both mothers and neonates suffering from puerperal sepsis, gastrointestinal infections, breast abscesses and skin sepsis.
3. All the items which have been supplied/bought by the patient should be marked carefully.
4. Avoid overcrowding in labour rooms and other procedural rooms to reduce cross infection. Limit the visitors inside these areas.

2.5 Availability of Clean Environment, Equipment and Other Supplies

1. Clean environment to be ensured by:
   (a) Operation theatres and labour rooms should ideally be cleaned after each operating session. Routine cleaning and mopping with water and detergent is required. A disinfectant should be used after known contamination of floors with material from infected patients.
   (b) An interval of at least 10 min should be there between two patients.
   (c) It is the duty of every staff nurse in labour room to clean thoroughly the furniture

and other articles in labour room with a hospital-approved disinfectant.

(d) For cleaning of all contaminated surfaces (labour table, procedure table, trolley surface, Kelly's pad or plastic sheet), use of 0.5% chlorine solution after every procedure should be undertaken.

(e) Maintain a clean sterile field around the delivery/surgical site by placing sterile towels or drapes around the surgical/procedure site. The sterile field includes the PPE (personnel protective equipment) worn and that remains above the level of waist. The back of the gown and shoulders and also the area below the waist are not considered sterile. The sterile operative field includes all sterile drapes above the level of operating table. To maintain a sterile field, only allow sterile items and personnel within the field. Hold the drapes by edges or from underneath surface for placing sterile drapes.

(f) For putting instruments use either a sterile instrument container or sterile drapes.

(g) Do not mix sterile items with contaminated items.

2. Adequate availability of the equipment and the following supplies:

(a) Soap, antiseptics, alcoholic scrubs and plenty of running water for proper hand hygiene should be available in emergency care area.

(b) Hand washing basins should be placed in labour rooms, maternity wards and nurseries.

(c) Sterile delivery packs, episiotomy sets, dressings, drapes and sterile sanitary pads should be freely available.

(d) Equipment, containers and teats for preparing special feeds should be sterilized by central sterile supply department (CSSD). In case of equipment (e.g. tubings, resuscitation apparatus) which do not withstand sterilization, high-level disinfection should be considered.

(e) Sterile disposable aprons, gloves, caps, face masks and shoe covers collectively called as personal protective equipment (PPE) should be available in plenty.

(f) Availability of following drugs to be insured.

(i) Antibiotics to prevent puerperal sepsis and neonatal sepsis.

(ii) Anticonvulsants for treatment of preeclampsia and eclampsia.

(iii) Uterotonic drugs for postpartum haemorrhage.

2.6 Availability of Skilled Staff

Highly skilled and adequate doctors, nurses and birth attendants should be there who are capable of dealing with any kind of emergency especially manual removal of placenta, removal of retained products following miscarriage or abortion, assisted vaginal delivery and basic neonatal resuscitation care. Also they should have the capability of performing caesarean section and blood transfusion. All staff should be screened for MRSA (methicillin-resistant *Staphylococcus aureus*), herpes and candida paronychia initially before induction and from time to time.

2.7 Hand Washing

It is the single most important procedure which can help in reducing the spread of infection in healthcare settings. There are mainly three types of hand washing:

(a) Simple hand washing.
(b) Hygienic hand washing.
(c) Surgical hand washing.

These have been summarized in Table 2.2.

2.7.1 When Is Hand Washing Recommended?

World Health Organization (WHO) has advocated use of hand washing in certain circumstances and these are known as "5 moments of

Table 2.2 Types of hand washing

Type of hand washing	Indication	Agents used for hand washing	Time	Areas to be included
Simple	Routine procedures	Soap and water	10 s	Only hands
Hygienic	Handling of infectious patients, nurseries, outbreak	60–70% alcoholic rub, 5–7.5% povidone iodine, 2–4% chlorhexidine	30 s	Hands and wrists
Surgical	Before minor and major surgery	Same as hygienic hand washing	3–5 min	Up to elbows

Simple hand washing is usually adequate in dealing with obstetric patients; use hygienic hand washing when dealing with premature and low birth babies or during outbreaks and surgical hand washing before any surgical procedure is done. In case of emergency stabilization procedures, a rapid scrubbing technique lasting for 1–2 min focusing on fingertips and hands is acceptable

Table 2.3 Management of HBV exposure

Vaccine status	Source HBV positive	Source HBV negative	Source unknown
Unvaccinated	HBIG 0.06 mL/im vaccine for HBV initiation	Vaccine initiation	Vaccine initiation
Previously vaccinated (written documentation of three or more doses)	Check for anti-HBs antibody titre. If above 10 IU/mL, no vaccine/booster is required; otherwise give HBIG 0.06 mL/im vaccine for HBV initiation	No treatment	No treatment

HBIG hepatitis B immunoglobulin
Infants borne to HBV-infected mothers should receive hepatitis B vaccine and hepatitis B immunoglobulin within 12 h of birth

Hand Hygiene". It is mandatory that hand hygiene is performed in *all patients*:

1. Before undertaking any aseptic procedure in the patient.
2. Before touching any patient.
3. After examining the patient.
4. After contact with patient surrounding.
5. After body fluid exposure.

In case of emergency procedures in an obstetric patient, if there is no time for hand washing, gloves should be worn in all circumstances.

2.8 Management of Needle-Stick Injuries (NSI)

1. Advice to staff or attendant with NSI regardless of the source of injection should be taken seriously with the hospital providing access to advice 24 h.
2. First aid involves immediate washing of the injury site with plenty of soap and water.
3. Both the patient from whom NSI has been got and the healthcare workers should undergo baseline tests, namely, for HBV, HCV and HIV.
4. In case of suspected HIV infection, postexposure prophylaxis (PEP) should be started immediately and not later than 72 h. Monitor for drug side effects. HIV antibody testing should be done at baseline, 6 weeks, 3 months and 6 months.

   There are two regimes for PEP: Basic two-drug and expanded three-drug regime depending upon the severity of exposure.
5. For management of HBV exposure, the following is to be done:

   Wounds and skin sites which have come in contact with blood or body fluids should be washed with soap and water. Mucus membranes should be flushed with water (Table 2.3).
6. For management of hepatitis C virus follow these things:

   No PEP is available.

   Management includes early identification and treatment.

If source is HCV positive, baseline testing for anti-HCV, liver function tests and subsequently follow-up at 4–6 months by ELISA/PCR for HCV RNA are done.

7. Refrain all individuals of NSI from positive sources of HIV, HBV and HCV from donating blood, organ donation, tissue donation and semen donation.

2.9 Blood Transfusion

2.9.1 Remember: Right Patient, Right Blood, Right Time, Right Place

Blood transfusion is essential in the management of life-threatening blood loss due to PPH and anaemia.

1. Avoid unnecessary and inappropriate blood transfusion. Rational use of blood transfusion and other blood products to be established by clinical and laboratory assessment. The obstetrician and anaesthetist should be careful enough to decide which patient needs transfusion.
2. Both the donors of blood and the blood of the patient should be tested for blood type (ABO group) and Rh type (+ or −) followed by cross-matching of the two. The identity check between blood of patient and donated blood is the crucial final step necessary to avoid fatal mistransfusions.
3. All donated blood to be screened for at least HIV, HBV, HCV, *Treponema pallidum* and *Plasmodium*. A safe supply of blood and blood products should be ensured by the blood bank concerned.
4. All transfusion should be completed within 4 hours of receiving the blood unit from blood banks ambient temperature for storage.
5. Monitor the patient for pulse, blood pressure, temperature and respiratory rate at start of blood transfusion, after 15 min and not more than 60 min, after transfusion is complete.

2.10 Safety from Sharps

1. Whenever possible use safer needle devices with built-in safety control or needleless devices.
2. Never recap/bend or remove contaminated needles and other sharps.
3. Never shear or break sharps which are contaminated.
4. Always make needle cutters/containers available near areas where needles may be found.
5. Discard contaminated sharps immediately into appropriate containers.
6. Use puncture-resistant and leakproof containers for sharp disposal. Dispose the containers when 3/4 full.
7. Whenever razors have to be used, use disposable razors.

2.11 Infection Control Measures to Prevent Tetanus

1. All mothers should have received two doses of tetanus toxoid (TT) 0.5 mL im at least 2 weeks apart.
2. In cases where proper sterilization and disinfection facilities are not available, a booster dose (0.5 mL) im to the mother (who has already received two doses) should be given. If the mother has got no previous dose of vaccine, give anti-tetanus serum 1500 U im during delivery, and another shot of tetanus toxoid 0.5 mL im after 4 weeks is required.

2.12 Appropriate Use of Antibiotics

Use prophylactic broad-spectrum antibiotics especially before or during the procedure.

Do not indiscriminately use antibiotics in every patient. Use prophylactic antibiotics in the following circumstances:

In all immunocompromised patients.

After any invasive procedure is undertaken.

After any intrauterine procedures where contamination by vaginal flora is unavoidable like manual removal of placenta, bimanual compression of uterus caesarean section, etc.

2.13 Management of Spills

Spills by blood and body fluids should be done immediately. Wear gloves, masks, gowns and shoe covers. Put sodium hypochlorite (5–6.5%) 1 in 100 dilution for 15–20 min, and cover with an absorbent cloth/cotton/paper. Finally mop the floor.

2.14 Biomedical Waste Management Rules, 2016

Definition: "Biomedical waste" means any waste, which is generated during the diagnosis, treatment or immunization of human beings or animals or research activities pertaining thereto or in the production or testing of biological or in health camps.

These rules shall apply to all persons who generate, collect, receive, store, transport, treat, dispose or handle biomedical waste in any form.

These rules shall not apply to:

(a) Radioactive wastes.
(b) Hazardous chemicals.
(c) Solid wastes covered under the municipal solid waste.
(d) The lead acid batteries.
(e) Hazardous wastes covered under the hazardous.
(f) E-waste,
(g) Genetically engineered microorganisms.

The salient features of these rules are:

1. No untreated biomedical waste shall be mixed with other wastes.
2. The biomedical waste shall be segregated into containers or bags at the point of generation.
3. The containers or bags shall be labelled.
4. Bar code and global positioning system shall be added by the occupier and common biomedical waste treatment facility in 1 year time.
5. Untreated human anatomical waste, animal anatomical waste, soiled waste and biotechnology waste shall not be stored beyond a period of 40–80 h.
6. Microbiology waste and all other clinical laboratory waste shall be pretreated by sterilization.

Schedule I

Colour and type of bag or container	Category/type of waste	Treatment and disposal options
Yellow-coloured non-chlorinated plastic bags or containers	**1. Human anatomical waste** **2. Animal anatomical waste** **3. Soiled waste** **4. Expired or discarded medicines** **5. Chemical waste** **6. Chemical liquid waste** **7. Discarded contaminated linen, mattresses and beddings** **8. Microbiology, biotechnology and other clinical laboratory wastes**	Incineration or plasma pyrolysis or deep burial[a] In the absence of above facilities, autoclaving or microwaving Expired cytotoxic drugs and items contaminated with cytotoxic drugs to be returned back to the manufacturer or supplier for incineration Bags containing residual or discarded blood and blood components to be hydroclaving followed by shredding or mutilation or combination of sterilization and shredding
Red-coloured non-chlorinated plastic bags or containers	**Contaminated waste (recyclable)** (a) Wastes generated from disposable items such as tubing bottles, intravenous tubes and sets, catheters, urine bags, syringes (without needles and *fixed needle syringes*) and vacutainers with their needles cut) and gloves	Autoclaving or microwaving/hydroclaving followed by shredding or mutilation or combination of sterilization and shredding
White (translucent) puncture-proof, leakproof, tamper-proof containers	**Waste sharps including metals:** Needles, syringes with fixed needles, needles from needle tip cutter or burner, scalpels, blades or any other contaminated sharp object that may cause puncture and cuts	Autoclaving or dry heat sterilization followed by shredding or mutilation or encapsulation in metal container or cement concrete; combination of shredding cum autoclaving; and sent for final disposal to iron foundries or sanitary landfill or designated concrete waste sharp pit
Blue cardboard boxes with blue-coloured marking cardboard boxes with blue-coloured marking	**(a) Glassware:** Broken or discarded and contaminated glass including medicine vials and ampoules except those contaminated with cytotoxic wastes. **(b) Metallic body implants**	Disinfection (by soaking the washed glass waste after cleaning with detergent and sodium hypochlorite treatment) or through autoclaving or microwaving or hydroclaving and then sent for recycling

[a]Disposal by deep burial is permitted only in rural or remote areas where there is no access to common biomedical waste treatment facility

3 Delays Recognized in Maternal Mortality

Lubna Hassan and Lauren Woodbury

3.1 Introduction

> If you want to judge a country, see how it treats women—Abdul Ghaffar Khan.

A version of this quote has been attributed to many sources; regardless of the source, it is very relevant because it strikes at the heart of the issue of maternal mortality. The status of women in a given country is inextricably linked to their health, and the reason women die giving birth is no mystery. We know why over 800 women, primarily from the developing world, die every day. They die because they have no or limited access to quality health care. The majority of maternal deaths occur due to direct obstetric causes—hemorrhage, preeclampsia, eclampsia, obstructed labor, sepsis, and unsafe/incomplete abortions or miscarriages. Importantly, deaths from these complications are preventable as treatments are well known and relatively inexpensive [1]. If all women had access to appropriate care, it is estimated that 80–74% of maternal deaths could be averted [2].

This is a crucial moment in the global effort to reduce maternal mortality since it is a time of transition from the Millennium Development Goals (MDGs) to the Sustainable Development Goals (SDGs). Whether countries can implement lessons learned from the MDGs will determine if they succeed in saving women's lives. Achieving the goals laid out in SDG 3.1 and the WHO's elimination of preventable maternal mortality (EPMM) initiative will require national level commitments and the combined efforts of all stakeholders including governments, the relevant UN agencies, national and international NGOs, and the OB/GYN community [3]. The OB/GYN community in South Asia, led by the South Asian Federation of Obstetrics and Gynecology (SAFOG), has tremendous power to be in the vanguard by directly addressing shortfalls in the strategies to eliminate preventable maternal deaths and by helping to overcome the "three delays" in accessing care. **The first delay: delay in seeking care** results from the low status of women, lack of awareness of complications and risk factors, and/or financial limitations. **The second delay: delay in reaching care** is usually due to issues around accessibility including distance from a health facility, prohibitive cost of health care, lack of transportation, and poor infrastructure. **The third delay: delay in receiving care** results from quality of care issues including poor facilities or lack of supplies/equipment, poorly trained personnel, or an inadequate referral system for complicated cases [4].

In the short term, medical causes must be addressed, primarily through emphasizing quality

L. Hassan (✉)
Women's Health Intervention and Development Initiative (WHI-DI), The Woman's Hospital, and SAFOG MNH Committee, Peshawar, Pakistan

L. Woodbury
WHI-DI, Peshawar, Pakistan

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throughout the continuum of care. In the longer term, the contributing social, cultural, and economic factors must also be addressed. Importantly, one must understand that a high rate of maternal death is not an isolated phenomenon. It extends from a lifetime of marginalization experienced by women and girls including poor health and malnutrition in childhood, which continues through adolescence and pregnancy resulting in anemia and other complications. Frequent and inadequately spaced pregnancies and socioeconomic factors like poverty, illiteracy, and lack of empowerment all contribute to high maternal mortality rates.

3.2 Background: The Global Response

The story of the neglected tragedy of maternal mortality is well documented. After the publication of the now famous article "Where is the M in MCH?" [5] and the introduction of the *Safe Motherhood Initiative* in 1987, maternal mortality reduction has gradually gained worldwide attention. A global consensus on what must be done and an increased commitment in many countries rose from this enhanced awareness. In 1999 a joint statement from the WHO, UNFPA, UNICEF, and the World Bank called on countries to "ensure that all women and newborns have skilled care during pregnancy, childbirth and the immediate postnatal period" [6].

In 2000, the UN adopted the UN Millennium Declaration. This historic agreement included eight critical goals—the MDGs—for combating poverty and accelerating human development to be achieved by 2015. MDG 5 (*Improve Maternal Health*) had two targets:

(a) All countries reduce maternal mortality by 75% from the 1990 maternal mortality ratio.
(b) Achieve universal access to reproductive health.[1]

[1]This goal was added in 2007.

Thanks to efforts toward MDG 5 globally, a 44% reduction in the maternal mortality rate (MMR) occurred from 385 per 100,000 live births in 1990 to an estimated 216 in 2015. The annual number of maternal deaths decreased by 43% from 532,000 in 1990 to an estimated 303,000 in 2015. The approximate global lifetime risk of maternal death fell from 1 in 73 to 1 in 180. These figures indicate a substantial reduction, but it is far short of the goal of 75% [7, 8]. Between 1990 and 2015, estimated MMR declined across all MDG regions. However, there is considerable variation in the amount of the reduction between regions and income groups. The estimated lifetime risk of maternal mortality in low-income countries is 1 in 41 which is vastly worse than high-income countries (1 in 3300). Today developing regions accounted for approximately 99% of the global maternal deaths. The worst performing regions are sub-Saharan Africa which alone accounts for roughly 66% of deaths followed by South Asia [9, 10].

3.2.1 The Sustainable Development Goals

Building on the experience gained from efforts to achieve MDG 5, the SDGs establish an enhanced and more comprehensive agenda for maternal health. The aim of SDG 3.1 is to reduce the global MMR to less than 70 per 100,000 live births by 2030 and to have no country with an MMR above 140. This is significantly below the current global MMR of 216. Achieving the SDG target will require reducing global MMR by an average of 7.5% every year. **This is more than three times the annual rate of reduction (2.3%) from 1990 to 2015** [10].

The SDGs provides a more holistic framework that emphasizes the need for universal health coverage [11] and a focus on equity. MNCH has been expanded to Reproductive, Maternal, Neonatal, Child and Adolescent Health (RMNCAH). The UN Secretary-General released the Global Strategy for Women's, Children's and Adolescents' Health 2016–2030 at the same time as the adoption of the SDG declaration.

It provides a broad multi-stakeholder framework for the implementation, follow-up, and review of progress toward the goals. The UN Secretary-General's strategy is complimented by the WHO's elimination of preventable maternal mortality initiative [3].

3.2.2 Emphasizing Quality of Care

Importantly, new global efforts have refocused from simply increasing coverage to highlighting the importance of **quality of care**. This came about due to the realization that merely increasing the *quantity* of interventions has not been enough to reduce maternal mortality to target levels. A likely explanation for this discrepancy is the poor quality of care in many health facilities [12]. To address this issue, a core set of indicators for improving and reporting on quality of care in facilities providing maternal, newborn, and child health services has been developed by the WHO and the Partnership for Maternal, Newborn and Child Health [13]. The emphasis is on the following areas:

1. Routine care during childbirth, including monitoring of labor and newborn care at birth and during the first week.
2. Management of preeclampsia, eclampsia, and its complications.
3. Management of difficult labor with safe, appropriate medical techniques.
4. Management of postpartum hemorrhage.
5. Newborn resuscitation.
6. Management of preterm labor and birth and appropriate care for preterm and small babies.
7. Management of maternal and newborn infections [14].

3.2.3 Importance of Data

Measuring maternal mortality remains an immense challenge, and new global efforts highlight the importance of accurate data. Measuring progress requires robust, internationally comparable civil registration systems. However, the very countries that have a high MMR also have poor data collection systems. Globally 60% of countries do not have accurate data on maternal deaths [15]. Instead, they rely on estimates, and under-reporting continues to be a major issue. Those countries that have reduced their maternal mortality now need to focus on comprehensive maternal death registration to ensure no cases are missed. Importantly, data must also be disaggregated below the national level. If it is not, the average decrease at country level masks pockets of high mortality within countries. Among those countries with low overall maternal mortality, the next challenge is measuring and rectifying inequities among their populations.

Importantly while accurate data is useful for tracking progress toward national goals, alone it does not provide information on the causes of and circumstances that lead or contribute to maternal deaths. This type of detailed information must also be collected as it is crucial for designing effective interventions. Detailed information can only be obtained through maternal death reviews (MDR) or audits [16] which ideally would be conducted as part of a complete, national **Maternal Death Surveillance and Response** (MDSR) system. MDSR is a system that measures and investigates maternal deaths in real time to help stakeholders including the government, donor agencies, NGOs, and health-care workers at all levels understand the underlying factors contributing to the deaths. The primary goal is to eliminate preventable maternal mortality by obtaining and strategically using information to guide public health actions and monitor their impact. Emphasizing *response* is what makes MDSR more effective than other approaches which gather information but don't necessarily lead to action [17].

Performance of MDR (a key component of MDSR) is one of the WHO's core indicators on a country's progress in reducing maternal mortality. Use of these indicators in monitoring quality of care along with the implementation of the MDSR approach will contribute to greater accountability for maternal health and more efficacious coverage of lifesaving interventions, thereby contributing to the end of preventable maternal mortality [17].

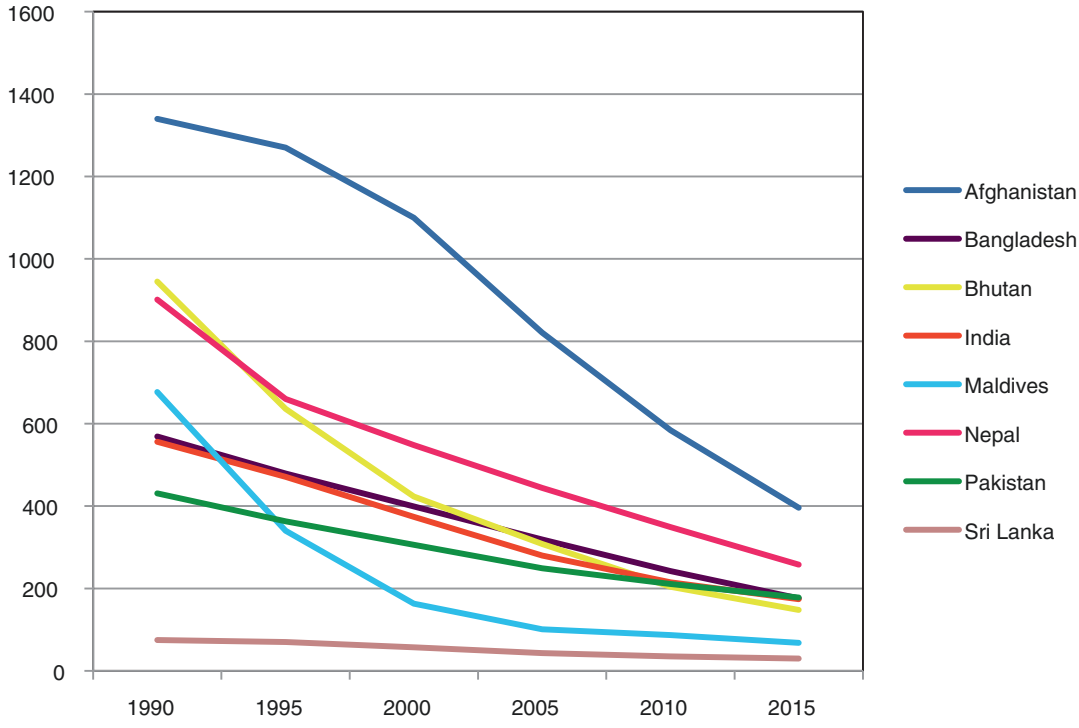


Fig. 3.1 Trends in MMR among SAR countries, 1990–2015

3.3 South Asia Current Trends and Remaining Challenges

South Asia is home to 1.6 billion people across eight countries: Afghanistan, Bangladesh, Bhutan, India, Maldives, Nepal, Pakistan, and Sri Lanka. The reduction in the MMR in the region has been substantial from 550 in 1990 to 190 in 2013. This is a reduction of 65%, the highest rate globally. However, even with this reduction, South Asia still has the second highest rate of maternal deaths, accounting for roughly 24% of global maternal deaths [9]. The table above displays the trends in maternal mortality of South Asian countries from 1990 to 2015 (Fig. 3.1)[2].

There are similarities as well as disparities between and within the South Asian countries. At one end of the spectrum is Sri Lanka, which has health statistics that is nearly comparable to developed countries. The graph shows Sri Lanka's extremely low level of MMR compared to the rest of the region. However, Sri Lanka did not technically achieve MDG 5 because it was unable to reduce its maternal mortality by 75%. This paradox illustrates a gap in the MDGs which dealt in averages and wanted a fixed decrease regardless of the starting point. This shortcoming has been resolved in the SDGs [18]. Like most South Asian countries, Sri Lanka has limited resources and has had its share of disturbances including a civil war. Nonetheless, it still managed to develop its social sector. Successful implementation of MDR and a commitment to universal access are credited with Sri Lanka's low MMR [16]. This is a lesson for other countries in terms of what can be achieved if priorities are set correctly.

Bhutan and the Maldives have also done exceptionally well and were the two countries in the region that achieved MDG 5 with declines of 84 and 90%, respectively. Afghanistan and

[2]Graph derived from data in WHO 2015. Trends in maternal mortality 1990 to 2015. Estimates by WHO, UNICEF, UNFPA, World Bank Group, and the United Nations Population Division.

Nepal also made significant progress falling just short of MDG 5 with declines of 70 and 71%. Nevertheless, Afghanistan's progress notwithstanding it still remains one of the world's "high-MMR" countries at over 400 per 100,000 live births. The three most populace countries, Bangladesh, India, and Pakistan, all made progress but failed to achieve MDG 5 [10]. However, given that their populations combine to account for roughly half of all South Asia, the progress they did make contributed the most to the overall region-wide decline in the total number of maternal deaths: India (74.1%), Bangladesh (12%), and Pakistan (7.6%).Within India, three states, Uttar Pradesh/Uttaranchal, Bihar/Jharkhand, and Rajasthan, contributed more than 50% of the total of India's decline in maternal deaths, while other states lagged behind [9]. Notably, Pakistan started out in 1990 with the second lowest MMR in the region after Sri Lanka, but given its very slow rate of progress, it now has a higher MMR than most countries in the region. In 2015 Pakistan's estimated MMR stood at 178 which is higher than Bangladesh (176), India (174), Bhutan (148), Maldives (68), and Sri Lanka (30) [10].

The gender inequality that persists in every domain of South Asian societies underpins the factors hindering further reductions in MMR. These include high fertility rates, high rates of early marriage and pregnancy, lack of access to family planning, inequity in access to maternal health services, and malnutrition (a frequent underlying cause of maternal deaths) [19].

Moreover, with a few notable exceptions, national and provincial/state governments have failed to follow through on stated commitments. Conspicuously absent in most cases is the necessary budget allocations. The result is short-term or ad hoc interventions rather than comprehensive health system improvements. In addition to an overall funding gap for maternal health at the national level, there are large disparities in the targeting of donor funding and country needs. Some very poor countries with high MMRs have gotten comparatively little funding, while some wealthier countries have gotten more funds [20]. Similarly, progress is undermined by inadequate oversight and coordination of projects leading to wasted resources, duplication of effort, and corruption. Lack of laws or insufficient enforcement of laws to protect women and girls including a failure to prevent child marriage have also hindered efforts to reduce maternal mortality.

Finally, as mentioned earlier, a lack of complete data hampers the ability to design effective interventions and to track progress. With the exception of Sri Lanka, South Asian countries rely on estimates derived from limited surveys and extrapolations of piecemeal facility-based data. In most countries representative community-based data on maternal deaths is almost totally lacking. The WHO estimates that the MMR is *underestimated* by 30% globally and by up to 70% in some countries [21]. The figure below displays the range in in maternal mortality estimates for South Asian countries (Fig. 3.2)[3].

3.4 The Health-Care Needs of Pregnant Women and Newborns

To ensure optimum outcomes for themselves and their newborns, women need a **continuum of care** throughout their entire reproductive lives. The continuum starts at home and includes self-care and prevention through things like proper diet. The next step in the continuum is antenatal care which is the key preventive measure. Approximately 15% of pregnant women will get complications [22]; however, which individuals will experience complications cannot be reliably predicted. All women, therefore, must receive antenatal care from qualified skilled birth attendants (SBAs)[4] in or near a health center with

[3]Graph derived from data in WHO 2015. Trends in maternal mortality 1990 to 2015. Estimates by WHO, UNICEF, UNFPA, World Bank Group, and the United Nations Population Division.

[4]"Skilled care" refers to an appropriately trained, accredited, and competent health-care provider. Since skilled care can be provided by a range of health professionals, whose titles may vary in different countries, it has been agreed to refer to this health-care provider as the "skilled birth attendant" (SBA), to avoid confusion over titles.

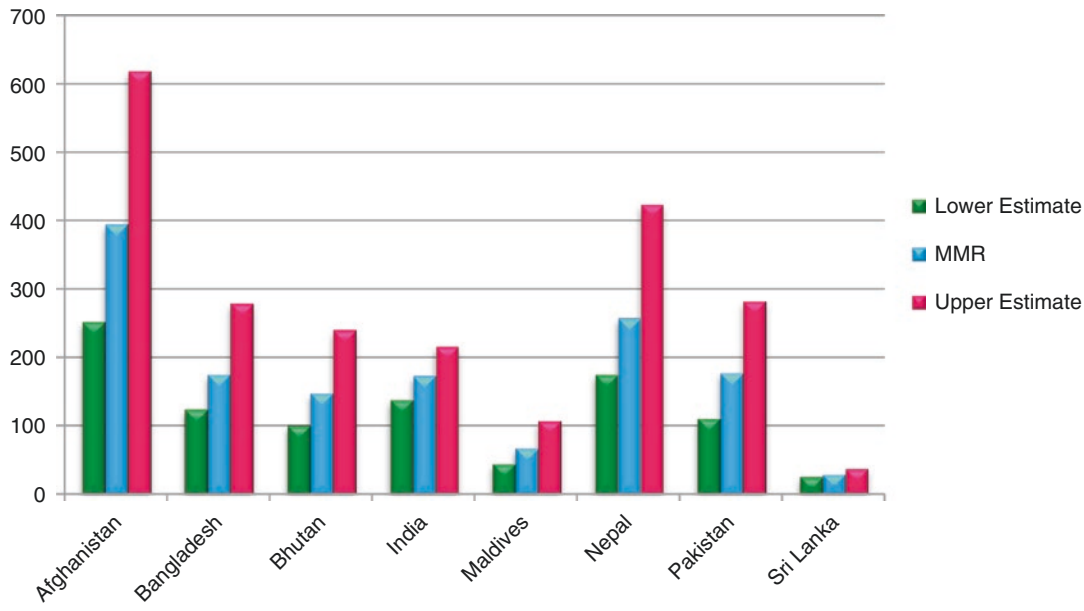


Fig. 3.2 Range in MMR estimates 2015

adequate facilities for EmOC. Antenatal care (ANC) involves a birth plan during a woman's pregnancy including place of delivery. The place of delivery may be the first level where the provision of high-quality midwifery is ensured. In cases that remain free of complications, care can continue at this level. In case of complications, referral systems must be in place for a seamless transfer to a secondary or tertiary level facility, depending on the severity of the complication.

Within the continuum ANC visits provide a window of opportunity for important preventative measures including screening for and diagnosis of complications and diseases. Timely and evidence-based ANC cannot only save lives, but antenatal visits also provide the chance to educate and support women, families, and communities. The WHO explains that "these communication and support functions of ANC are key, not only to saving lives, but to improving lives, health-care utilization, and quality of care. Women's positive experiences during ANC and childbirth can create the foundations for healthy motherhood" [23].

To facilitate the continuum of care from the home or the basic level to a higher level, it is imperative to have a fully functioning health system including adequate transportation and continuous, effective collaboration between those responsible for providing care at every level. The "SBA is the 'lynch pin' in this entire process." However, the mere presence of a skilled provider alone is not enough to protect women. The SBA must have the necessary equipment and must have formal linkages with higher levels of care [6]. Unfortunately there is a gap at this level in most countries, and the disconnection between the specialist obstetrician and the community SBA sometimes causes fatal delays in the provision of care [24].

3.5 The Three Delays Model as a Framework for Eliminating Preventable Maternal Deaths

Factors leading to death may be examined along the Three Delays Model which examines the interrelated set of delays that prevent a pregnant woman from accessing the health care she needs. In 1994, Sereen Thaddeus and Deborah Maine linked causes of maternal mortality to "three

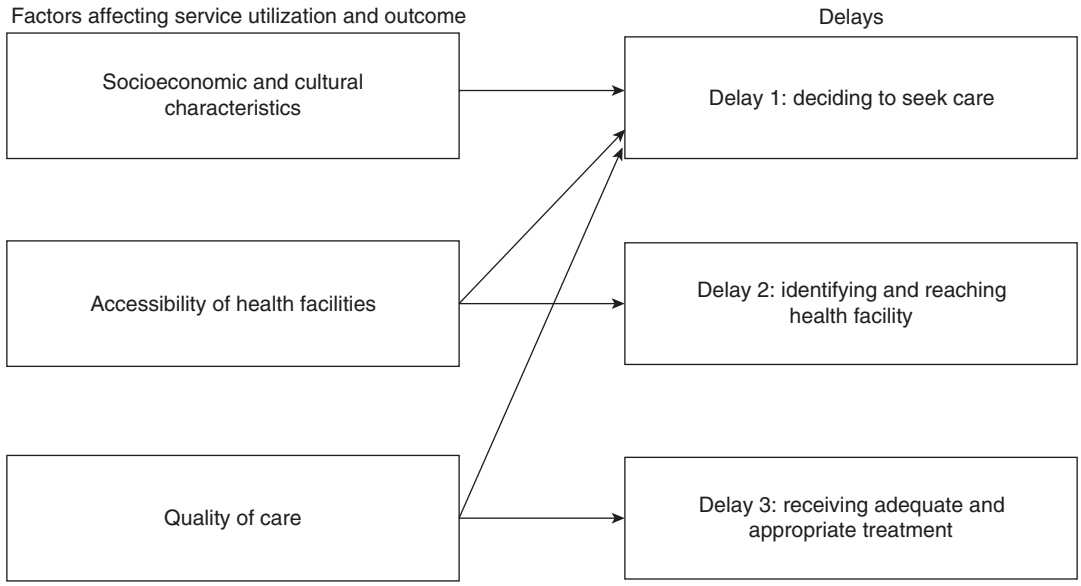


Fig. 3.3 Contributing factors and the three delays

delays." They are defined chronologically, as delays in:

1. The decision to access care.
2. The identification of or transport to a medical facility.
3. The receipt of adequate and appropriate treatment [25].

Socioeconomic and cultural factors, accessibility of facilities, and quality of care may affect the lengths of these delays [26]. Figure 3.3 below illustrates how these factors lead to the delays.

3.5.1 The First Delay: Recognizing the Need and Making the Decision to Seek Care

The first step is for a woman or her family to recognize the need to seek care and then make the decision to do so. Why would a woman or her family not access health care during pregnancy? The following factors often coalesce to delay the decision to seek care [25]:

- Poor education/low literacy (especially of women).
- Lack of access to information and gender inequality.
- Lack of access to health information and education.
- Lack of access to affordable and physically accessible health care.
- The low status of women.
- Previous poor experience of health care.
- Acceptance of maternal death.

These constraints are often compounded by risk factors including age (either too young or too old is a concern), frequent pregnancies, or pre-existing health concerns including anemia, malaria, HIV/AIDs, diabetes, hypertension, and obesity. The way the first delay functions was explored in-depth in a seminal study in Haiti with a sample of 12 maternal deaths that occurred in a longitudinal cohort of pregnant women. In 8 of the 12 cases, researchers found a delayed decision to see medical care. Interviews with family and friends suggested that **a lack of confidence in available medical options was a crucial**

factor in a delayed or never made decisions to seek care [4]. Notably, this finding is prevalent in other high-MMR countries as well.

3.5.2 The Second Delay: Delay in Arrival at a Health Facility

Socioeconomic divisions within countries often mean unequal access to health facilities. The WHO reports "delivery care… is strongly associated with one's income, whether they live in a rural or urban area, and their level of education" [9, 10].

Those living in rural areas face the major obstacle of distance from health-care facilities. In developing nations weak infrastructure often means poor roads and slow transportation methods which can further delay a woman's arrival at a health facility [25].

The delay in reaching an appropriate facility is attributed to the following factors:

- Distance to health centers and hospitals.
- Lack of availability of and cost of transportation.
- Poor roads and infrastructure.
- Geography, e.g., mountainous terrain and rivers.
- The lack of a robust referral network among community providers, such as SBAs/midwives, and health facilities at the basic, secondary, and tertiary levels can also cause or contribute to this delay.

3.5.3 The Third Delay: Delay in Receiving Adequate Health Care

The third delay derives primarily from quality of care issues, and this is where the OB/GYN community has direct power to have an immediate impact. As countries make progress on the first two delays and more deliveries occur in facilities, the third delay will assume greater importance. If facilities prove unable or unwilling to accommodate higher demand, the reduction in maternal deaths will stall. This has been shown in South India where earlier delays have largely been addressed, but facilities are struggling to meet increased demand and thus have not been able to get the MMR below 80 per 100,000 live births.

Delay in receiving adequate care when a facility is reached results from [25]:

- Lack of 24/7 EmOC services, especially in peripheral hospitals.
- Attitude of the staff: culture of blame and shame. It is common for patients/women to be shamed for coming late and/or for SBAs to be blamed for the complications.
- Shortages in staff.
- Insufficient or inappropriate training.
- Facility is ill equipped (most commonly lack of electricity, water, or medical supplies).
- VIP culture (especially prevalent in South Asia) exacerbates this delay since poor (and thus usually higher risk) patients are often treated poorly or ignored by medical staff in favor of more affluent patients.

Because maternal deaths occur both in the facilities and communities, delays one and two can lead to a woman never reaching a facility or arriving in critical condition. Facility data from across South Asia shows that many deaths occur shortly after arrival at hospitals that do provide appropriate EmOC. These deaths are often associated with a late referral. Figure 3.4 below displays background factors contributing to the three delays.

3.6 Solutions Moving Forward

The elimination of preventable maternal mortality in developing countries is a long-term undertaking that will require commitment from stakeholders at all levels. As the preeminent experts, OB/GYNs can lead advocacy efforts with their governments on which policies are needed to improve availability and quality of maternal care. The OB/GYN community has an essential role to play that begins with ensuring adequate and equitable care is provided in their facilities as well as pointing out where shortfalls exist. Crucially, the OB/GYN community has the

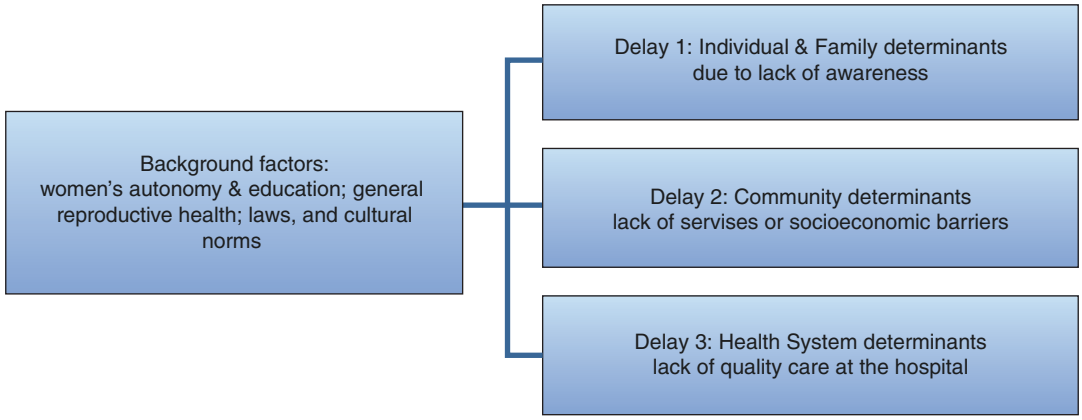


Fig. 3.4 Background factors contributing to delays

ability to have an immediate and direct impact in addressing quality of care issues contributing to the third delay even without substantial government or donor support. Facilities must be prepared for the increase in demand, in terms of properly trained human resource, ICU facilities, emergency teams, blood and components, antibiotics, etc., and in short adequately trained staff with appropriate equipment and supplies.

It is also paramount that facilities are accessible to the poor and vulnerable. Even the best equipped facility will not by itself save lives. The OB/GYN community can begin advocating with the government to ensure the first and second delays are addressed. Similarly, recognizing the crucial role of the community SBA in the OB/GYN bodies of each country, individually and through SAFOG, is central to positively changing the attitudes of its members toward SBAs.

Overall what are needed are specific and implementable actions based on proven strategies and lessons learned. What follows are the key activities identified by the World Bank's Reproductive Health Action Plan (RHAP),[5] lessons learned from MDG 5, and the author's recommendations. The OB/GYN community ideally must be directly involved in designing and/or implementing many of these to maximize efficacy [27–31].

- **Better data:** One of the most important lessons learned from MDG 5 was the gap in vital statistics. Registering each maternal death and determining not just the medical cause but the associated socioeconomic-cultural reasons through an MDSR program will lead to a more robust understanding of the household decision-making. This ensures stakeholders can implement the right policies and programs to change attitudes and behavior.
  - All South Asian countries should move to implement an MDSR program using a representative sample of facility and community-based reviews to ensure a complete understanding of the on-ground reality.
  - Similarly, efforts to build the capacity of health staff to implement MDRs and to respond to findings are needed.
- **Address misconceptions about reproductive health:** Educating women and relatives on reproductive health is essential to combating harmful perceptions and to ensuring obstetric complications/risk factors are recognized in a timely manner.
  - The more efficient strategy of preventing pregnancy complications through regular

[5]RHAP emphasizes 57 priority countries including with high maternal mortality and/or high fertility and moderate to high levels of sexually transmitted infections. Reproductive health profiles were developed for all priority countries. Five South Asian countries (India, Nepal, Pakistan, Bangladesh, and Afghanistan) are identified as priorities, and the actions specified above are common to each country.

ANC must take precedence over simply focusing on treating complications once they occur.
  - This will require a combination of behavior change communication (BCC) programs via mass media and community outreach as well as deploying properly trained SBAs.
- **Promote institutional delivery:** Through education and outreach and provider incentives to generate demand for the service.
  - During ANC visits educate pregnant women about the importance of delivery with skilled health personnel and getting postnatal checks. Encourage and promote community participation in the care for pregnant women and their children, and address the issue of women wanting someone to accompany them to the health facility.
  - Provide vouchers to poor women to cover cost of delivery services.
- **Transport arrangements:** Make transport arrangements, or provide transport vouchers to women in hard-to-reach areas. Emphasize transportation initiatives where it is most needed in rural/remote areas.
- **Emphasize reaching poor women in hard-to-reach rural areas** in the provision of basic and comprehensive emergency obstetric care.
  - Renovate and equip health facilities that serve these areas.
- **Address the inadequate human resources for health** by training more skilled birth attendants and deploying them to the poorest or hard-to-reach areas.
  - Multiskilling of health professionals at all levels including training of general physicians in anesthesia and cesarean sections for providing comprehensive EmOC.
- **Strengthen the referral system:** Simple changes in practice and attitude can save many lives.
  - Ensure the continuum of care throughout pregnancy and postnatally from home to a referral hospital.
  - Institute emergency transport systems training health personnel in appropriate referral procedures (referral protocols and recording of transfers).
  - Establish a culture of referral as part of the birth plan. The crucial link between the community and the hospital and between the SBA and the obstetrician must be acknowledged and strengthened through the institutionalization of the referral system and in continued medical education imparted as a team.
- **Improve quality of services** (basic and comprehensive obstetric care) by introducing and using evidence-based treatment protocols and guidelines (best practices) to be used by skilled attendants at birth at all levels.
  - All women must be attended by a skilled birth attendant with a birth plan which includes such information as: who will accompany the woman, transport, where she will be referred, and baseline emergency treatment including iv fluids and a shock garment.
- **Community workers:** Utilize services of newly introduced village level workers for improving access to quality antenatal services and nutrition and management of anemia among pregnant women and improving access to home level postnatal and neonatal care.
- **Public/private partnerships:** Build capacity in public sector and engage with the private sector to fill gaps and provide quality services, especially to poor women.
- **Address emerging challenges and indirect maternal deaths:** The increasing importance of the infectious and chronic noncommunicable diseases (NCD) that contribute directly and indirectly to maternal mortality is a matter for concern. As countries reduce the MMR, there is a need to strengthen the recognition and management of indirect causes of maternal death and coordinate with other relevant sectors and health providers to address care for NCD. There is also a need to develop innovative education, screening, and management approaches for these conditions, as well as appropriate clinical guidelines and protocols [3].

3.7 Conclusion

It is tragically fitting that one of the world's most famous sites, arguably the most famous in South Asia, is a monument to a woman who died in childbirth. The Taj Mahal was commissioned by Shah Jahan in 1631 in the memory of his wife Mumtaz Mahal who died giving birth to their 14th child [32, 33]. Today tourists flock to it to marvel at its beauty and reflect on the grief of a powerful king who was helpless to save the woman he loved. Around the time the Taj was being built in India, schools for midwives were being established across Europe in response to the high numbers of women dying in childbirth there. Of course, at the time how to save a woman dying from childbirth was largely beyond medical knowledge, but nevertheless in Europe the seeds of modern midwifery were sowed. Today the vast majority of maternal deaths are preventable since the knowledge and technology to save mothers is well known. Yet the women of South Asia continue to pay the price of a lack of understanding, focus, or commitment of their leaders. It is an outrage that in 2017 every 2 min a young woman loses her life while bringing a new life into the world. The fact that so many women are dying of preventable causes goes beyond a simple tragedy. It is one of the biggest public health scandals of our time.

I have been privileged for 34 years to work in the department of OB/GYN in government (public sector) hospitals in the city of Peshawar, Pakistan. The women at these hospitals were usually poor, illiterate, and repressed. I witnessed personally the inequity of the care they received, but I also saw how resilient they were. I realized over my long association with these women that their marginalization is in itself a serious health hazard and that health of women is often compromised not because of lack of medical knowledge or even services but because of infringements on their most basic rights. The world must recognize that repeated, often unwanted pregnancies and high rates of maternal death from preventable causes are not just a public health issue; they are violation of women's human rights.

Fortunately, the OB/GYN community collectively can achieve a great deal. As we develop our specialty, build state of the art hospitals, and provide cutting edge in scientific interventions, we must equally be in the vanguard of eradicating preventable maternal deaths. We must increase our advocacy armed with evidence of effective interventions. We must pursue culturally sensitive approaches that respect the women we serve. We must not get side tracked by empty slogans or competing priorities. With the inclusion of Afghanistan last year, SAFOG now has all eight South Asian countries as members. It can therefore serve as a powerful, unified platform from which to advocate for women's rights. As shown by the successes of some countries in our region and globally, with proper commitment and leadership, especially from the OB/GYN community, even in resource poor countries, preventable maternal deaths can be eliminated.

References

1. Say L, Chou D, Gemmill A, Tunçalp O, Moller AB, Daniels J, et al. Global causes of maternal death: a WHO systematic analysis. Lancet. 2014;2(6):e323–33.
2. United Nations Human Rights Office of the High Commissioner. Preventable maternal mortality and morbidity and human rights. Retrieved from http://www.ohchr.org/Documents/Issues/Women/WRGS/Health/ReportMaternalMortality.pdf.
3. WHO. Strategies toward ending preventable maternal mortality (EPMM); 2015. http://www.everywomaneverychild.org/images/EPMM_final_report_2015.pdf. Accessed 7 July 2016.
4. Barnes-Josiah D, Myntti C, Augustin A. The "three delays" as a framework for examining maternal mortality in Haiti. Soc Sci Med. 1998;46(8):981–93.
5. Rosenfield A, Maine D. Maternal mortality – a neglected tragedy. Where is the M in MCH? Lancet. 1985;2:83–5.
6. WHO. Making pregnancy safer: the critical role of the skilled attendant. A joint statement by WHO, ICM and FIGO; 2004. http://www.who.int/maternal_child_adolescent/documents/9241591692/en/ Accessed 9 July 2016.
7. UNICEF. UNICEF Data: Monitoring the situation of children and women; 2016. http://data.unicef.org/maternal-health/maternal-mortality.html. Accessed 6 July 2016.
8. WHO. Global Health Observatory Data. State of inequality: reproductive, maternal, newborn and child

health. Geneva: World Health Organization; 2015. http://www.who.int/gho/health_equity/report_2015/en Accessed 8 July 2016
9. WHO. Trends in maternal mortality: 1990 to 2013 Estimates by UNICEF, UNFPA, The World Bank, and the United Nations Population Division; 2014. http://apps.who.int/iris/bitstream/10665/112682/2/9789241507226_eng.pdf. Accessed 7 July 2016.
10. WHO. Trends in maternal mortality: 1990 to 2015, Estimates by WHO, UNICEF, UNFPA, World Bank Group and the United Nations Population Fund; 2015. http://apps.who.int/iris/bitstream/10665/194254/1/9789241565141_eng.pdf. Accessed 7 July 2016.
11. Lancet. The Lancet. Women's, children's, and adolescents' health needs universal health coverage. http://www.thelancet.com/journals/lancet/article/PIIS0140-6736(15)01176-9/abstract.
12. Paily VP, Ambujam K, Rajasekharan Nair V, Thomas B. Confidential review of maternal deaths in kerala: a country case study. BJOG 2014;121(Suppl. 4):61–66.
13. WHO. Consultation on improving measurement of the quality of maternal, newborn and child care in health facilities; 2013. http://apps.who.int/iris/bitstream/10665/128206/1/9789241507417_eng.pdf. Accessed 7 July 2016.
14. WHO. 2016. Standards for improving quality of maternal and newborn care in health facilities. http://www.who.int/maternal_child_adolescent/documents/improving-maternal-newborn-care-quality/en/p.6.
15. WHO. 2016. Time to respond: a report on the global implementation of maternal death surveillance and response. http://apps.who.int/iris/bitstream/10665/249524/1/9789241511230-eng.pdf. Accessed 30 Mar 2017.
16. Mathur A, Awin N, Adisasmita A, Jayaratne K, Francis S, Sharma S, et al.Maternal death review in selected countries of South East Asia Region. BJOG 2014;121(Suppl. 4):67–67.
17. WHO. Maternal Death Surveillance and Response. http://www.who.int/maternal_child_adolescent/epidemiology/maternal-death-surveillance/en/. Accessed 7 July 2016.
18. Matsubayashi, T. et al. Analysis of cross-country changes in health services, Chapter 5. In: Peters D, et al. Improving health services in developing countries: from evidence to action; 2009.
19. El-Saharty, Sameh and Naoko Ohno. South Asia's quest for reduced maternal mortality: what the data show. World Bank Group; 2015. http://blogs.worldbank.org/health/south-asia-s-quest-reduced-maternal-mortality-what-data-show.
20. The Partnership for Maternal, Newborn & Child Health; 2014. http://www.who.int/pmnch/knowledge/publications/2014_pmnch_report/en/. Accessed 30 July 2016.
21. WHO. Time to respond: a report on the global implementation of maternal death surveillance and response; 2016. http://apps.who.int/iris/bitstream/10665/249524/1/9789241511230-eng.pdf. Accessed 30 Mar 2017.
22. WHO. The world health report 2005—make every mother and child count; 2005.
23. WHO. WHO Recommendations on antenatal care for a positive pregnancy experience; 2016. http://www.who.int/reproductivehealth/publications/maternal_perinatal_health/anc-positive-pregnancy-experience/en/. Accessed 26 Mar 2016.
24. WHO. Reducing maternal mortality. A joint statement by WHO/UNFPA/UNICEF/World Bank. Geneva; 1999. http://apps.who.int/iris/bitstream/10665/42191/1/9241561955_eng.pdf. Accessed 15 July 2016.
25. Thaddeus S, Maine D. Too far to walk: maternal mortality in context. Soc Sci Med. 1994;38(8):1091–110. https://doi.org/10.1016/0277-9536(94)90226-7. Accessed 10 July 2016
26. D. Maine. Safe motherhood programs: options and issues, Columbia University; 1991.
27. World Bank. Bangladesh - Reproductive health at a glance. Reproductive health at a glance; Bangladesh. Washington, DC: World Bank; 2011. http://documents.worldbank.org/curated/en/307361468212071971/Bangladesh-Reproductive-health-at-a-glance. Accessed 10 July 2016.
28. World Bank. Afghanistan - Reproductive health at a glance. Reproductive health at a glance; Afghanistan. Washington, DC: World Bank; 2011. http://documents.worldbank.org/curated/en/262581467996765917/Afghanistan-Reproductive-health-at-a-glance. Accessed 10 July 2016.
29. World Bank. Pakistan - Reproductive health at a glance. Reproductive health at a glance; Pakistan. Washington, DC: World Bank; 2011. http://documents.worldbank.org/curated/en/512941468325459864/Pakistan-Reproductive-health-at-a-glance. Accessed 10 July 2016.
30. World Bank. India - Reproductive health at a glance. Reproductive health at a glance; India. Washington, DC: World Bank; 2011. http://documents.worldbank.org/curated/en/743521468050934458/India-Reproductive-health-at-a-glance. Accessed 10 July 2016.
31. World Bank. Nepal - Reproductive health at a glance. Reproductive health at a glance; Nepal. Washington, DC: World Bank; 2011. http://documents.worldbank.org/curated/en/357801468289200282/Nepal-Reproductive-health-at-a-glance. Accessed 10 July 2016.
32. Sarkar J. Studies in Mughal India; 1919. https://archive.org/details/studiesinmughali00sarkuoft. Accessed 6 July 2016.
33. Chaghtai M.A. Le Tadj Mahal D'Agra (Hindi). Brussels; 1938.
34. World Health Organization. Maternal mortality Fact Sheet No. 348. Updated November 2015.

Part II

Problems in Pregnancy

Preterm Labor

4

Poonam Varma Shivkumar, Preeti Priyadarshani, and Namit Choksi

4.1 Definition

Preterm labor is defined as regular rhythmic uterine contractions resulting in cervical changes that start before 37 weeks of a viable pregnancy. Changes in the cervix include effacement (the cervix thins out) and dilation (the cervix opens so that the fetus can enter the birth canal) [1].

4.2 Introduction

Preterm birth is a global concern. Every year, an estimated 15 million babies are born preterm, and this number is rising [2]. In the year 2010, almost 24% of the total world's preterm babies were born in India [3].

Preterm birth is the leading cause of neonatal deaths and the second leading cause of death after pneumonia in children under 5 years [2].

The prognosis for individual preterm infants depends primarily on gestational age at birth. Mortality rates vary from about 2% for newborns born at or after 32 weeks to more than 90% for those born at 23 weeks. Many survivors face a lifetime of handicap, including learning disabilities and visual and hearing problems.

4.3 Classification of Preterm Births

Preterm births are classified into three subgroups according to gestational age at birth:

- Extremely preterm (<28 weeks).
- Very preterm (28–<32 weeks).
- Moderate to late preterm (32–<37 weeks).

The problems of preterm babies vary according to the degree of prematurity, with the maximum burden of difficulty being faced by the extremely preterm ones.

4.4 Etiology of Preterm Births

Preterm labor is a heterogeneous process. Multiple variables may simultaneously be responsible for the onset of preterm labor, hence the observed relatively low predictive value of any given variable. Most preterm births happen spontaneously, but some are iatrogenic, whether for medical or non-medical reasons. Genetics may also play a role in the etiology. Comprehending the causes and mechanisms associated with preterm births will help in the development of solutions to prevent preterm birth.

P. V. Shivkumar (✉) · P. Priyadarshani
Department of OBGY, MGIMS Sevagram, Wardha, Maharashtra, India

N. Choksi
Harvard University, Cambridge, MA, USA
e-mail: namit.choksi@mail.harvard.edu

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_4

4.4.1 Risk Factors for Preterm Birth

Factors that increase the risk of preterm birth include the following:

- Socioeconomic factors: Stressful pregnancy for various reasons such as low socioeconomic status, unwed mothers or lack of family support, low pre-pregnancy weight, smoking, alcohol, and substance abuse during pregnancy.
- Infections: Intrauterine infection either as ascending genital tract infection, including bacterial vaginosis, or as a part of systemic infection leading to chorioamnionitis.
- Anatomic abnormalities of the uterus and cervix, either congenital (unicornuate, bicornuate, arcuate, or septate uterus) or acquired (following cervical conization or LEEP or DES exposure in utero).
- History of previous preterm births or recurrent abortions.
- Multiple pregnancies.
- Trauma.
- Iatrogenic: Antepartum hemorrhage, diabetes mellitus, hypertensive disorders of pregnancy, renal diseases, and immunological disorders.
- Idiopathic: Accounts for about 30% of preterm births.

The two strongest risk factors for idiopathic preterm labor are low socioeconomic status and previous preterm delivery. However, a history of prior preterm birth is not useful in the nulliparous patients who make up nearly one half of all patients experiencing preterm birth.

Around 25% of preterm deliveries are elective due to either maternal factors such as preeclampsia or fetal factors such as extreme growth restriction.

4.4.2 Infection

Intrauterine infection is a chronic process accounting for 25–40% of preterm births. Preterm labor is triggered by the activation of the innate immune system. However, the role of infections in causing preterm births decreases as the gestational age advances.

Decidual colonization by microorganisms can occur by several ways: ascending, hematogeneous, iatrogenic by procedures such as amniocentesis, or retrograde through the Fallopian tubes. From the decidua, infection may reach the space between the amnion and chorion, the amniotic fluid, and the fetus.

Asymptomatic colonization of the decidua occurs in up to 70% of women at term, only few of these experience preterm labor pains. *Gardnerella vaginalis*, *Fusobacterium*, *Mycoplasma hominis*, *Ureaplasma urealyticum*, *Mycoplasma genitalium*, and severe untreated *Candida* infection are associated with increased risk of preterm birth.

Endotoxins released by microorganisms and cytokines stimulate decidual responses including the release of prostaglandins which stimulate uterine contractions. The decidua can also release matrix-degrading enzymes that weaken fetal membranes leading to premature rupture. Bacterial vaginosis before or during pregnancy can aggravate the decidual inflammatory responses, and prophylactic antibiotic therapy is associated with decreased preterm birth incidence and complications.

Chorioamnionitis can lead to maternal and fetal sepsis and significant long-term fetal sequelae including cerebral palsy.

4.5 Diagnosis

Initial symptoms of preterm labor can be confused with the discomfort associated with gravidity. Hence, many women experiencing preterm labor present only in the late stages, while many healthy women may have multiple prenatal visits with similar complaints. A confirmatory diagnosis of preterm labor is made only in the presence of cervical changes, either on clinical examination or ultrasonography.

4.5.1 Symptoms and Signs of Preterm Labor

- Increase in the amount of vaginal discharge.
- Change in type of vaginal discharge (watery, mucus, or bloody).

- Pelvic or lower abdominal pressure.
- Constant low, dull backache.
- Mild abdominal cramps, with or without diarrhea.
- Regular or frequent uterine contractions, often painless.
- Ruptured membranes (amniotic membrane breaks with a gush or a trickle of fluid comes out).
- Vaginal spotting or bleeding.

Essential History

- Length of time since onset of symptoms.
- Interval between contractions and duration of each contraction.
- Bleeding or leaking per vaginum.
- Previous history of preterm deliveries.
- History of infections, bleeding, pain, and single or multiple fetuses in the current pregnancy.
- History of smoking.

Examination

- General examination may reveal pallor, high BP, edema feet as per the case.
- Systemic examination findings may be as per the case or may be normal.
- Abdominal examination reveals uterine contractions.
- Speculum examination may reveal dilatation or effacement of the cervix and/or amniotic fluid leak through the cervix.
- Pelvic examination:
  - This should be deferred if a history of vaginal leak is present, to avoid risk of ascending infections.
  - If the membranes have not ruptured, however, cervical assessment should be performed for assessing the onset of premature labor.

4.5.2 Investigations Specific for Preterm Labor

Transvaginal ultrasound examination may be done to measure the length of the cervix and its dilatation. A cervical length of less than 25 mm at or before 24 weeks of gestational age is used as a predictor of preterm labor. The risk of preterm birth increases as the cervical length decreases.

High vaginal swab and cervical swab should be taken in all women with possible premature labor, for Gram staining and culture, as this will detect the infection and allow appropriate antibiotic therapy to be given.

Fetal fibronectin (fFN), an important biomarker, is measured in the vaginal discharge by taking a cervicovaginal swab prior to a vaginal examination. The presence of this glycoprotein in the cervical or vaginal secretions indicates a disruption of the choreo-decidual interface. A positive test (>50 ng/mL) indicates an increased risk of preterm birth, and a negative test has a high predictive value.

Maturity amniocentesis is a prenatal screening procedure involving sampling of amniotic fluid to determine lung maturity. Lecithin-sphingomyelin ratio of 2.4 or more confirms lung maturity. The technique can also be used to detect an infection in the amniotic fluid.

Placental alpha microglobulin-1 (PAMG-1), commercially known as the PartoSure test, is an excellent predictor of imminent spontaneous delivery within 7 days in a woman with preterm labor, with a better positive predictive value than either fFN or cervical length.

4.6 Prevention of Preterm Birth

Primary prevention aimed at reducing or eliminating the risk factors in the general population is a limited strategy which involves public education, improving socioeconomic status of family, smoking cessation, early and regular prenatal visits, and avoidance of late preterm births. Also, due diligence is required when using assisted reproductive technology (ART) as risk of preterm labor increases with multiple pregnancies.

Secondary prevention focuses on women with a prior history of preterm birth which is the most recognizable risk factor. Preventive strategies include cervical cerclage, progesterone, and dedicated clinics [4].

4.6.1 Progesterone

Progesterone, given in the form of 17-hydroxyprogesterone caproate 500 mg intramuscularly weekly or oral dydrogesterone 10–20 mg daily or oral or vaginal natural micronized progesterone 200 mg daily, relaxes the myometrium, maintains cervical length, and has anti-inflammatory properties. Its usefulness has been demonstrated in women with history of recurrent preterm births, as also in women discovered to have a short cervical length. However, the same may not be true of multiple gestations.

4.6.2 Cervical Cerclage

In women with documented cervical incompetence, the cervix is stitched closed with strong sutures. The sutures are removed at term, or earlier if the woman goes in labor. Cervical cerclage is recommended if woman is less than 24 weeks pregnant and either has a history of early premature birth or an ultrasound shows cervical length less than 25 millimeters.

4.7 Management

Defining an ideal management protocol for preterm labor has been an elusive goal. It is managed based on the fact that delay in delivery will give a better chance for the fetus to survive. Certain medications may be given to delay the delivery and improve neonatal outcome. These medications include corticosteroids, magnesium sulfate, and tocolytics.

4.7.1 Corticosteroids

Corticosteroids given to the mother cross the placenta and help in faster maturity of the fetal lungs, brain, and digestive organs. Maximum benefit is observed when they are given between 24 weeks of pregnancy and 34 weeks of pregnancy. As per GOI guidelines, injection dexamethasone is given 6 mg intramuscular 12 hourly four doses for lung maturation [5]. This therapy also gives protection from necrotizing enterocolitis and intraventricular hemorrhage. Injection betamethasone 12 mg intramuscular two doses 24 hours apart may also be given as an alternative. Repeated doses or rescue therapy is not recommended at present. Steroids are contraindicated in chorioamnionitis, maternal tuberculosis, porphyria, and maternal or fetal infections. Maternal diabetes is a relative contraindication.

4.7.2 Magnesium Sulfate

Magnesium sulfate is recommended in women going to deliver very preterm to extremely preterm infant within the next 24 hours [6]. A loading dose of 4 g is given as a single slow intravenous bolus followed by an infusion of 1 g/h over 24 hours or until delivery, whichever is sooner [4]. Monitoring is required for signs of maternal magnesium toxicity. If used as described, magnesium sulfate has a limited tocolytic as well as neuroprotective role by decreasing the incidence of cerebral palsy in the preterm neonates.

4.7.3 Tocolytics

Tocolytics are used to decrease uterine activity for a short time (up to 48 hours) in women with uncomplicated pregnancies. This gives time for action of corticosteroids or magnesium sulfate or for arranging transfer of women to institutions with specialty care centers for preterm infants.

Nifedipine: It is a calcium channel blocker that inhibits the entry of calcium through channels in the cell membrane and causes smooth muscle relaxation. It is the tocolytic of choice; however, concomitant administration of nifedipine and magnesium sulfate should be avoided.

Atosiban: Atosiban is an oxytocin analog that acts as a competitive antagonist of

oxytocin-induced contractions. Atosiban is offered to women in whom nifedipine is contraindicated.

Beta adrenergic receptor agonists: These drugs react with β-adrenergic receptors to reduce intracellular ionized calcium levels and prevent activation of myometrial contractile proteins. However, they are no longer recommended for this purpose due to the associated side effects such as pulmonary edema. Examples are terbutaline and ritodrine.

Indomethacin: This drug inhibits the synthesis of prostaglandin by blocking the enzyme prostaglandin synthase. Though it is effective in delaying preterm birth, associated side effects like oligohydramnios, patent ductus arteriosus, necrotizing enterocolitis, and intraventricular hemorrhage limit its use.

Using multiple tocolytic drugs appears to be associated with a higher risk of adverse effects and so should be avoided.

4.7.4 Antibiotics

Studies examining the use of antibiotics have provided mixed results [7]. Antibiotics may be useful in preventing preterm labor in women with bacterial vaginosis. Erythromycin is recommended in women with preterm prelabor rupture of membranes [8]. However, once chorioamnionitis sets in, antibiotics cannot ameliorate the need for early delivery, and expediting delivery of the baby is of utmost importance.

4.7.5 Emergency Cervical Cerclage

Emergency or "rescue" cervical cerclage is offered to women between 16 and 34 weeks with cervical changes and exposed unruptured fetal membranes. It is not recommended in women with signs of infection, bleeding, or true uterine contractions.

The role of bed rest, hydration, and sedation of the patient is debatable in the present scenario.

4.8 Delivery

- Most preterm babies with cephalic presentation are safely delivered vaginally, with Caesarean section being done for other obstetric indications.
- Elective Caesarean section to avoid the risk of intracranial hemorrhage is not routinely indicated.
- Caesarean section is the preferred mode of delivery in preterm infants with breech presentations.
- Prophylactic forceps delivery to protect the soft preterm head during delivery is not indicated.
- Fetal heart rate monitoringduring labor is recommended and can be done with either a stethoscope or ultrasound Doppler. Continuous monitoring has not proven superior to intermittent auscultations.
- Invasive methods of fetal monitoring such as fetal scalp electrodes and fetal blood sampling are not recommended below 34 weeks of gestation.

4.9 Neonatal Care

After delivery, general principles of neonatal resuscitation are applicable. Plastic wraps or warm blankets are useful during transport of babies to prevent hypothermia. Where facilities are available, preterm infants are cared for in a NICU (Neonatal Intensive Care Unit) on SNCU (Sick Newborn Care Unit), with special emphasis on preventing infection and hypothermia by using radiant warmers and incubators. Management also includes parenteral nutrition and ventilator support if required. Surfactant therapy may be given if required.

In resource-poor settings, simple cost-effective measures such as exclusive breastfeeding, kangaroo mother care (KMC), and precautions for infection prevention such as frequent handwashing go a long way in decreasing preterm morbidity and mortality. Survival rates of infants differ widely depending on the center providing postnatal care.

4.9.1 Complications

One or more organ systems of the preterm infants may be affected, including but not limited to the complications mentioned below:

- CNS: hypoxic ischemic encephalopathy (HIE), cerebral palsy, and intraventricular hemorrhage.
- Respiratory system: respiratory distress syndrome (RDS), bronchopulmonary dysplasia, and pneumonia.
- Cerebrovascular system: patent ductus arteriosus (PDA).
- Ophthalmological: retinopathy of prematurity (ROP).
- Gastrointestinal system: necrotizing enterocolitis, feeding difficulties, and inguinal hernia.
- Metabolic: neonatal hypoglycemia, hypocalcemia, and rickets of prematurity.
- Hematological system: anemia of prematurity, sepsis, and hyperbilirubinemia which can lead to kernicterus.

References

1. American College of Obstetrics and Gynecology: Preterm labour and birth. http://www.acog.org/Patients/FAQs/Preterm-Premature-Labor-and-Birth
2. Born Too Soon: The Global Action Report on Preterm Birth –WHO-2012.
3. Delivered Too Soon –Action Report on Preterm Births in India – 2013.
4. National Institute for Health and Care excellence (NICE Guideline: Preterm labour and birth). nice.org.uk/guidance/ng25.
5. Use of Antenatal Corticosteroids in Preterm Labour: Operational Guidelines; 2014. http://www.tngmssh.tn.gov.in/news/guidelines/Preterm_Labour.pdf.
6. Royal College of Obstetricians and Gynaecologists. Magnesium Sulphate to Prevent Cerebral Palsy following Preterm Birth Scientific Impact Paper No. 29; 2011. https://www.rcog.org.uk/globalassets/documents/guidelines/scientific-impact-papers/sip_29.pdf.
7. Lamont RF. Advances in the prevention of infection-related preterm birth. Front Immunol. 2015;6:566.
8. Kenyon SL, Taylor DJ, Tarnow-Mordi W. Broad spectrum antibiotics for preterm prelabour rupture of membranes. The ORACLE 1 randomised trial. Lancet. 2001;357:979–88.

Prelabor Rupture of Membranes

5

Parneet Kaur and Surbhi Saini

5.1 Introduction

Prelabor rupture of membranes (PROM) remains a subject of great clinical relevance and a problem encountered by each and every obstetrician in day-to-day practice. Preterm prelabor rupture of membranes (PPROM) is rupture of membranes prior to 37 weeks of gestation, but before the onset of labor. PPROM is far more difficult to manage than PROM at term. Several issues need to be considered in formulating a plan of management. While intra-amniotic infection and its sequelae are the primary maternal risks, prematurity is the principal risk to the fetus which can lead to increased neonatal morbidity and mortality.

5.2 Definition [1, 2]

5.2.1 Prelabor Rupture of Membranes (PROM)

Prelabor rupture of membranes is defined as rupture of membranes before the onset of labor.

5.2.2 Term Prelabor Rupture of Membranes (TPROM)

When the rupture of membranes occurs beyond 37 weeks but before the onset of labor, it is called term prelabor rupture of membranes.

5.2.3 Preterm Prelabor Rupture of Membranes (PPROM)

When the rupture of membranes occurs before 37 completed weeks but before the onset of labor, it is called preterm prelabor rupture of membranes.

5.2.4 Prolonged Rupture of Membranes

Rupture of membranes for >24 h before delivery is called prolonged rupture of membranes.

5.3 Incidence [3–5]

PROM occurs in approximately 10% of all pregnancies, and in 70% of these cases, it occurs in pregnancies at term.

PPROM occurs in 3% of all pregnancies and is responsible for approximately 30% of all preterm deliveries.

P. Kaur (✉) · S. Saini
Department of Obstetrics & Gynaecology,
Government Medical College, Patiala, Punjab, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_5

5.4 Etiology [5–7]

Multiple factors predispose patients to PPROM like:

- Subclinical intrauterine infection is a major predisposing factor, especially at early gestation.
- Women with PPROM in previous pregnancy are at increased risk for recurrence in next pregnancy.
- Prior preterm labor in the current pregnancy.
- Low socioeconomic status.
- Body mass index ≤19.8.
- Nutritional deficiencies.
- Smoking.
- Sexually transmitted infections (STIs) in female.
- Cervical conization in the past.
- Uterine distention (e.g., multiple pregnancy, hydramnios).
- Cervical cerclage done for cervical incompetence.
- Vaginal bleeding during pregnancy.
- Presence of a short cervix (< 25 mm by TVS).
- Invasive procedures during pregnancy (e.g., amniocentesis, chorionic villi sampling).

Though there are so many of these known risk factors, still none could be identified in most cases of preterm labor.

The underlying pathophysiology in PROM is considered to be collagen degradation in the fetal membranes caused by increased metalloproteinases. Also in pregnancies complicated by PPROM, amnion also exhibits a higher degree of cell death and apoptosis regulated by bacterial endotoxin, interleukin-1, and tumor necrosis factor-α. These all result in a weakened amnion [6].

5.5 Result of PROM [4]

If managed expectantly, half of the women with TPROM deliver within 5 h and 95% within 28 h. Also, whatsoever may be the clinical presentation or obstetric management, delivery occurs within 1 week of rupture of membranes in 50% of cases with PPROM.

5.6 Diagnosis

Diagnosis of rupture of membranes is important for three reasons:

- To decide for gestational age-specific obstetric management.
- There is increased possibility of cord prolapse and cord compression if the presenting part is not fixed.
- If delivery is delayed after rupture of membranes, intrauterine infection is more likely as time interval increases.

Diagnosis is made by:

- History.
- Sterile speculum examination.
- Ultrasonography.

5.6.1 History

Patient gives typical history of sudden gush of liquor. At times patient gives history of intermittent leaking, continuous leaking of small amount of fluid, or just a feeling of wetness. Thus detailed history of amount, color, and smell of liquor should be elicited. History of time of rupture of membranes should be taken. History of perception of fetal movements should also be taken.

5.6.2 Sterile Speculum Examination

The vulva is cleansed with sterile saline. Antiseptic should not be used as it may interfere with bacteriological assessment. On per speculum examination:

- There will be presence of pool of amniotic fluid in posterior fornix also known as amniorrhexis.
- Clear fluid may be seen coming out from cervical canal.
- If no fluid is seen, we can ask the patient to cough as this may cause liquor to drain.

By doing per speculum examination, we can also estimate cervical dilatation, exclude cord prolapse, and take cultures [4].

In doubtful cases with a strong history of leaking, patient may be asked to wear a sterile sanitary pad for 30–60 min and observed.

If in doubt from history and clinical examination, the following tests can be performed [6, 8, 9]:

1. *Nitrazine test*: The pH of vaginal secretions is acidic and normally ranges from 4.5 to 5.5, whereas the amniotic fluid pH is 7.0–7.5. The use of nitrazine indicator is simple and fairly reliable method. The test papers are impregnated with dye and are used to test the vaginal fluid while doing per speculum examination. The color of the reaction between these paper strips and vaginal fluids is seen and interpreted by comparing with a standard color chart. A pH above 6.5 indicates ruptured membranes. The presence of blood, semen, or bacterial vaginosis may give false-positive test. Also false-negative test may be obtained when amniotic fluid is less.
2. *Ferning pattern*: Microscopic examination of vaginal fluid shows characteristic arborization or ferning. Amniotic fluid when dried on a glass slide crystallizes to form a fern-like pattern due to the presence of sodium chloride, protein, and carbohydrate.

   The above two tests have shown the best results with a sensitivity of 90% [8].
3. *Lanugo hair*: The presence of fetal lanugo hair in vaginal fluid when examined microscopically was considered indisputable evidence of membrane rupture. However, because of limited amounts of fetal lanugo hair in amniotic fluid and also because of the fact that such hairs were present in amniotic fluid only in later weeks of pregnancy, this method never became popular.
4. *Cytologic diagnosis*: These tests were based on cytologic inspection for fetal squamous cells in the vagina, using various stains including Masson stain, Sudan III stain, Papanicolaou stain, Pinacyanole stain, acridine orange stain, and the most popular Nile blue sulfate stain. Although fetal cell-staining techniques were considered rapid, simple, and durable, concerns about their accuracy emerged. They were also time-consuming, needed trained cytologists, were not effective before 32 weeks of gestation, and did not provide certain diagnosis of membrane rupture.

Digital examination (P/V) should not be done unless the woman is in active labor. There are two reasons for this:

- We may transfer the microorganisms from the vagina into the cervix while examining, and this may lead to intrauterine infection, release of prostaglandins, and preterm labor.
- Also digital examination adds little to the information available with speculum examination [2, 8].

5.6.3 Ultrasonography

Ultrasonographic evaluation of amniotic fluid volume does help in documenting oligohydramnios, but is not diagnostic for PROM. If after proper evaluation the diagnosis still remains unclear, then we can instill indigo carmine dye (1 mL in 9 mL of sterile normal saline) transabdominally under the guidance of ultrasound. We can then observe the passage of blue fluid into the vagina which can be confirmed by a stained vulval pad [2, 5]**.** We should not use methylene blue dye for this test because it has been associated with hyperbilirubinemia and hemolytic anemia in infants. Though ultrasonography is not necessary to confirm PROM, it does help in determining the gestational age and position of the fetus, localization of the placenta, residual amniotic fluid volume, estimated fetal weight, and presence of any abnormalities in the fetus [10]**.** A low initial amniotic fluid index has been shown to be associated with shorter latency period and an increased risk of chorioamnionitis. However, amniotic fluid volume assessment does not accurately predict the time of delivery, and thus should not be used alone to decide regarding conservative management.

5.6.4 Newer Biochemical Tests

- IGFBP-1 (Actim PROM) test: It is a rapid test that specifically detects insulin-like growth factor-binding protein-1 (IGFBP-1) in the vaginal fluid. It is a simple bedside test and can be used as a complimentary test to confirm the clinical diagnosis of PROM [11, 12].
- PAMG-1 (AmniSure®) test: It is a rapid immunoassay test which detects placental alpha-microglobulin-1 (PAMG-1) and is found to be reliable in the diagnosis of PROM. The test has a sensitivity and specificity of 98.9 and 100%, respectively [7, 8, 11, 13].
  - It is FDA approved.
  - Simple and easy to perform.
  - Does not require a speculum examination.
  - Has no gestational age limitation.

5.6.5 Role of Amniocentesis

Intrauterine infection can be diagnosed by positive amniotic fluid cultures in 36% of women with PPROM. Most infections are subclinical with no obvious signs and symptoms of chorioamnionitis. Current evidence also tells us that infection is a cause of rupture of membranes rather than its result. Although by going for amniocentesis we can detect subclinical infection before clinical picture of chorioamnionitis develops and also before the onset of fetal infection, the evidence at present is not sufficient to recommend amniocentesis for the diagnosis of intrauterine infection [8].

If we want a favorable obstetric outcome, a timely and accurate diagnosis of prelabor rupture of membranes is critical. A false-positive diagnosis of PROM especially preterm PROM may lead to unnecessary obstetric interventions and delivery of a preterm baby thus increasing perinatal morbidity and mortality. Thus every effort should be made to reach at a correct diagnosis.

5.7 Complications

The fetal membranes act as a barrier to ascending infection. Once the membranes rupture, both the mother and fetus are at risk of infection and other complications.

5.7.1 Neonatal Complications [2, 7]

These are related mainly to prematurity. PPROM is associated with increase in perinatal mortality and neonatal morbidity.

- Respiratory distress syndrome (RDS) occurs in 10–40% cases of PPROM and causes 40–70% of neonatal deaths.
  Despite initial suggestions, subsequent observations do not support association of accelerated pulmonary maturation with PPROM [14].
- Neonatal infections.
- Intraventricular hemorrhage (IVH).
- Necrotizing enterocolitis (NEC).
- Increased risk of neurodevelopmental impairment.
- Fetal pulmonary hypoplasia occurs in 26% of PPROM before 22 weeks.
- Skeletal deformities.
- Severe oligohydramnios may lead to an increased incidence of cord compression and abnormal fetal heart pattern in labor.
- Infections and cord accident contribute to 1–2% risk of antenatal fetal demise.

5.7.2 Maternal Complications [4, 7]

- Clinically evident intra-amniotic infection occurs in 15–25% and is seen more frequently with prolonged PPROM, severe oligohydramnios, and repeated vaginal examinations.
- Postpartum infections occur in 15–20%, with incidence higher at early gestation.
- Abruptio placentae in 2–5%.
- As more fetuses with PPROM present with malpresentation, thus there is increased risk of

cesarean delivery with its attendant complications as compared with term deliveries.

5.8 Management

5.8.1 Confirmation of the Diagnosis of PROM and Its Importance

Early and correct diagnosis of PPROM would allow the obstetrician to plan for management according to gestational age for a better perinatal outcome and minimal complications. On the other hand, a false-positive diagnosis of PPROM may lead to unnecessary obstetric interventions [7].

We should do the following in all cases of PROM [4]:

- Confirm gestational age and fetal presentation, and assess fetal well-being by subjecting all patients to electronic fetal monitoring (EFM) during the initial period of observation to assess any abnormality of fetal heart rate (FHR) and uterine activity.
- If FHR tracings are abnormal or there is evidence of chorioamnionitis, one should proceed for delivery.
- Vaginal bleeding should raise suspicion of placental abruption, and delivery should be considered.

5.9 Gestational Age-Specific Management

5.9.1 Term Prelabor Rupture of Membranes (TPROM)

In these cases delivery should be planned and intrapartum group B streptococcal (GBS) prophylaxis given if indicated [4].

The three main things to consider are:

- Timing of delivery.
- Method of induction.
- Role of antibiotics.

5.9.1.1 Timing of Delivery

A Cochrane review [15] found that induction of labor (IOL) reduced induction delivery interval and incidence of chorioamnionitis. There was no increase in rates of cesarean sections, operative vaginal delivery, or NICU admission. A large trial also found that women accepted IOL more positively than expectant management [16]. ACOG also recommends that when patient presents with PROM at ≥ 37 weeks of gestation, induction of labor should be done if there are no contraindications [4], whereas according to NICE guidelines, women with TPROM should be offered a choice of IOL or expectant management, and IOL is appropriate after 24 h [17]. Since planned and expectant management may not be very different, women should be properly counseled so that they can make informed choices.

5.9.1.2 Method of Induction

Oxytocin should be used for IOL as prostaglandins and mechanical methods of induction (e.g., Foley catheter) are associated with higher rates of intrauterine infection [4].

When IOL is done with oxytocin, we should allow a sufficient period of adequate contractions (at least 12 h) for the latent phase of labor to progress before diagnosing failure of induction and deciding in favor of operative delivery [18, 19].

5.9.1.3 Role of Antibiotics

There are studies which show that use of antibiotics resulted in a statistically significant reduction in chorioamnionitis and neonatal infection [20–22], but according to ACOG, evidence is not sufficient to justify the routine use of prophylactic antibiotics with TPROM [4].

5.9.2 Preterm Prelabor Rupture of Membranes (PPROM)

The time period from rupture of membranes to delivery is called "latency." [5, 23] Following rupture of membranes, delivery is recommended when the risk of ascending infection exceeds the

risk of prematurity. During evaluation of a case, contraindications to expectant management should be ruled out. Absolute contraindications are [10]:

- Intra-amniotic infection, i.e., chorioamnionitis.
- Non-reassuring fetal testing.
- Placental abruption.
- Active labor.

The diagnosis of *chorioamnionitis* [6, 8, 10] is mainly clinical by observing:

- Maternal pyrexia (≥100.4 °F).
- Fetal tachycardia.
- Maternal tachycardia.
- Uterine tenderness.
- Offensive vaginal discharge.

(Maternal temperature, pulse, and FHR auscultation should be checked between 4 and 8 hourly interval.)

Other tests recommended are maternal full blood count, C-reactive protein, and high vaginal swab, but the sensitivity of these tests in detection of intrauterine infection is less. Cardiotocography is useful, and fetal tachycardia is also used in the definition of clinical chorioamnionitis [8]. Clinical chorioamnionitis is present on admission in 1–2% of women who present with PPROM and subsequently develops in 3–8% [23].

At $34^{0/7}$ weeks of gestation or greater, delivery is recommended for all women with rupture of membranes. If expectant management is continued beyond $34^{0/7}$ weeks of gestation, the benefit and risk should be carefully considered and discussed with the patient (ACOG, RCOG) [4, 8].

At $37^{0/7}$ weeks of gestation or greater, expectant management has no role, and delivery should be done in all cases of PROM.

Before $34^{0/7}$ weeks of gestation, expectant management should be done after ruling out contraindications (ACOG) [4].

Between 34 and 37 weeks of gestation, management of cases of PPROM remains a controversial issue. Those favoring delivery at 34 weeks argue that because of the lack of significant neonatal benefit with prolongation of the pregnancy until 37 weeks, early delivery is justified to reduce the risk of chorioamnionitis. A Cochrane review of planned early birth versus expectant management for women with PPROM before 37 weeks of gestation concluded that there was insufficient evidence to guide clinical practice on the benefits and harms of immediate delivery compared with expectant management [24]. The results of recent PPROMT trial says that women with PPROM between 34 and $36^{6/7}$ weeks, in the absence of overt signs of infection or fetal compromise, be managed expectantly with appropriate surveillance (Lancet 2016) [25]. Also after the recently published Antenatal Late Preterm Steroids (ALPS) [26] trial demonstrated that administration of betamethasone to women at risk for late preterm delivery significantly reduced the rate of neonatal respiratory complications, ACOG issued practice advisory [27] that administration of betamethasone may be considered in women with a singleton pregnancy between $34^{0/7}$ and $36^{6/7}$ weeks of gestation at imminent risk of preterm birth. *ACOG now recommends a single course of corticosteroids for pregnant women between $34^{0/7}$ weeks and $36^{6/7}$ weeks of gestation at risk of preterm birth who have not received a previous course of antenatal corticosteroids* [28, 29].

Expectant management is recommended for pregnancies **between $24^{0/7}$ and $33^{6/7}$ weeks of gestation** and generally consists of hospital admission and complete pelvic rest. Patients should be assessed at regular intervals for evidence of infection, abruptio placentae, umbilical cord compression, fetal well-being, and initiation of labor [2]. There is no consensus on the methods [30] and optimal frequency of fetal assessment, but an acceptable strategy would include periodic ultrasonographic monitoring of fetal growth and FHR [4]. The two most common testing modalities are nonstress test and the biophysical profile [23].

Any deterioration in health of the mother or fetus is an indication for delivery and abandoning of expectant management.

Women presenting with **previable PROM (<24 weeks)** should be counseled regarding the risk and benefit of expectant management. They should be offered immediate delivery [4] because

of high perinatal mortality (50%) and serious morbidity (40%) in those who survive [31]. In case the patient decides in favor of expectant management and is clinically stable with no evidence of infection, she can be managed on outpatient basis and should be admitted to the hospital once the pregnancy reaches the period of viability.

5.9.3 Management of Prelabor Rupture of Membranes (ACOG) [4]

5.9.3.1 Early Term and Term ($37^{0/7}$ Weeks or More)

- Deliver.
- GBS prophylaxis if indicated.

5.9.3.2 Late Preterm ($34^{0/7}$–$36^{6/7}$ Weeks)

- Same as for early term and term.

5.9.3.3 Preterm ($24^{0/7}$–$33^{6/7}$ Weeks)

- Expectant management is recommended.
- Antibiotics recommended to prolong latency if there are no contraindications.
- Single-course corticosteroids.
- GBS prophylaxis if indicated.
- Magnesium sulfate for neuroprotection should be administered in active labor.

5.9.3.4 Less than 24 Weeks of Gestation

- Meticulous patient counseling.
- Expectant management or induction of labor.
- Antibiotics may be given as early as $20^{0/7}$ weeks.
- GBS prophylaxis is not recommended.
- Corticosteroids are not recommended.
- Tocolysis is not recommended.
- Magnesium sulfate for neuroprotection is not recommended.

5.10 Expectant Management

5.10.1 Antibiotics

The use of antibiotics following PPROM increases the latency and is associated with a statistically significant reduction in chorioamnionitis and neonatal morbidity and should be given to women with PPROM before $34^{0/7}$ weeks (ACOG, NICE, SOGC) [4, 8, 32].

The two most well-studied regimens [33, 34] used in the largest PPROM randomized controlled trials are:

1. Inj Ampicillin 2 G 6 hourly intravenously and Inj Erythromycin 250 mg 6 hourly intravenously X 48 hours – followed by Tab Amoxycillin 250 mg TDS orally and Tab Erythromycin 333 mg TDS orally X 5 days.
2. Tab Erythromycin 250 mg orally QID X 10 days.

Amoxicillin/clavulanic acid should not be used because they have been reported to increase the risk of necrotizing enterocolitis in neonates [32, 34].

Women presenting with PPROM should be screened for urinary tract infections, sexually transmitted infections, and Group B streptococcus carriage and treated with appropriate antibiotics if positive [32].

Latency antibiotics should be continued till the onset of labor or until the completion of therapy. Studies show that women treated with antibiotics are more likely to remain pregnant up to 3 weeks after randomization, suggesting that the therapy successfully treated subclinical infection rather than just suppressing it [5].

5.10.2 Antenatal Corticosteroids [4, 35–37]

Antenatal corticosteroid administration after PPROM has been evaluated in many clinical trials and has been shown to reduce RDS (34%), IVH (46%), NEC (54%), and neonatal mortality (31%).

They should be administered between 24 and 34 weeks of gestation.

- A single course is recommended.
- Current evidence suggests corticosteroids when administered in antenatal period are not associated with increased risks of maternal or neonatal infection.

- The maximum effect occurs if the fetus is delivered 24 h after the last dose and up to 7 days thereafter.
- Antenatal corticosteroid therapy should be started even when the completion of a full course before preterm birth is uncertain. Partial effect is evident and is worth it.

5.10.2.1 Dose and Route of Administration

- Betamethasone 12 mg intramuscularly in two doses 24 h apart.
- Dexamethasone 6 mg intramuscularly in four doses 12 h apart.

Corticosteroids may be considered for pregnant women as early as $23^{0/7}$ weeks of gestation who are at risk of preterm delivery [28]. Weekly administration of corticosteroids is associated with a reduction in birth weight and head circumference of neonates and so not recommended. Also ACOG does not recommend administration of a rescue course of corticosteroids in patients with PROM [4].

Government of India (GoI) [34] recommends single course of dexamethasone injection to be administered between 24 and 34 weeks of gestation at all levels of health facilities.

Why Is Dexamethasone Injection Recommended by GoI?

Dexamethasone sodium phosphate and betamethasone acetate + phosphate are the only two efficacious and safe corticosteroids to be used during antenatal period. Both these drugs are identical in biologic activity and readily cross the placenta.

Dexamethasone: It is listed in the WHO essential medicines list, inexpensive, and widely available in facilities for multiple indications.

Betamethasone: The salt betamethasone acetate + phosphate, which requires only two doses, is not available in India. Here the salt available is betamethasone phosphate which is short acting and requires more frequent administration. Hence, the dosage schedule of betamethasone phosphate is similar to that of the dexamethasone and has no added advantage over dexamethasone. Betamethasone is also more costly and less stable than dexamethasone at high temperatures. Thus GoI recommends that, in individual cases where dexamethasone injection is not available, the service provider may use betamethasone phosphate injection to give the advantage of corticosteroids to the newborn.

Dexamethasone is thus a more appropriate option and recommended by the Government of India.

5.10.3 Group B Streptococcus (GBS) Chemoprophylaxis

Group B streptococcus (*Streptococcus agalactiae*) is an important cause of maternal and neonatal morbidity and mortality. Asymptomatic colonization of the vagina and rectum with GBS is common in pregnancy. Vertical transmission of this organism from the mother to fetus during labor or delivery may result in invasive infection in the newborn during the first week of life, known as early-onset Group B streptococcal infection. Intrapartum use of antibiotics in these women has led to a decrease in the rate of early-onset but not late-onset GBS disease. Identification of women with GBS is the key factor in the prevention of perinatal GBS disease. In India, the mortality and morbidity associated with the GBS disease remains an under-recognized problem, and there are no guidelines at present for its management, [38] whereas CDC [39] in collaboration with ACOG [40] have formulated national guidelines that are revised from time to time. Approximately 10–30% of pregnant women are colonized with GBS in the vagina or rectum in the USA. Intrapartum GBS chemoprophylaxis has been shown to significantly decrease the incidence of early onset neonatal GBS infection and mortality. The new guidelines provide updated algorithm for screening for GBS and intrapartum antibiotic prophylaxis for women with PROM (Figs. 5.1 and 5.2).

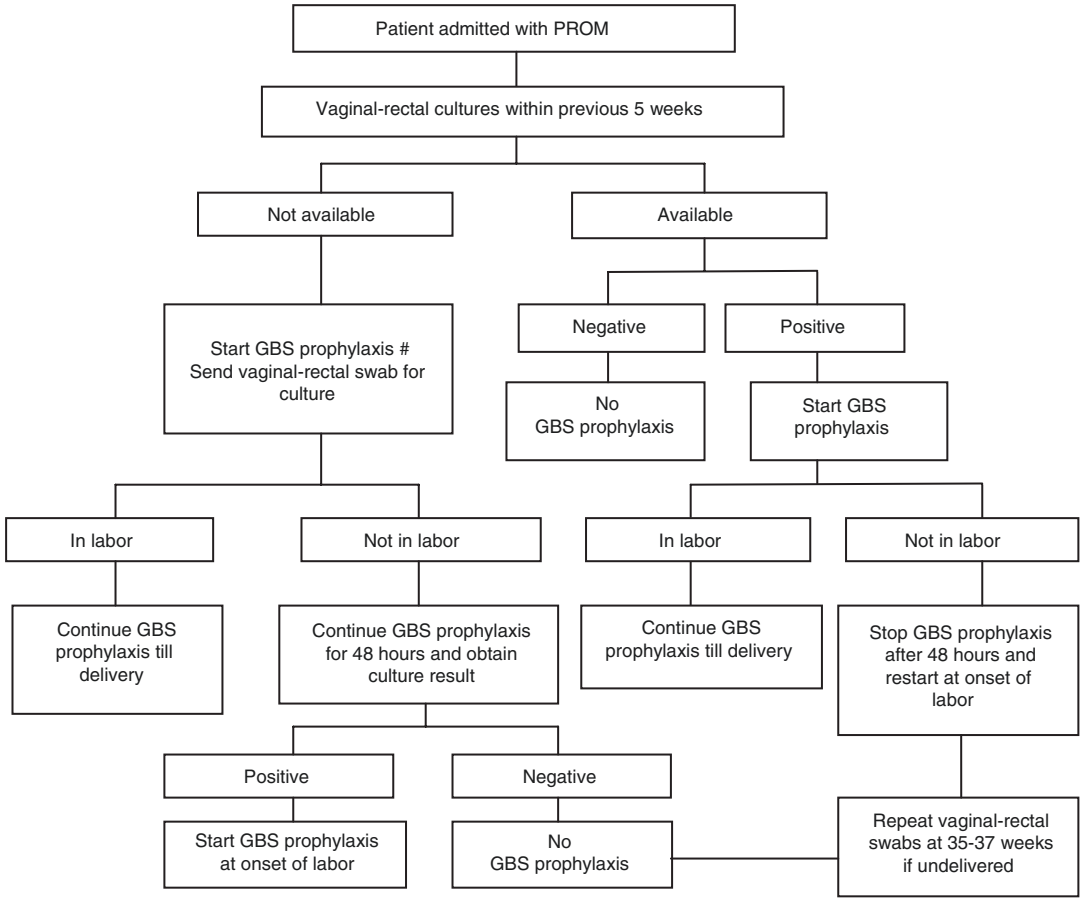


Fig. 5.1 Algorithm for screening for GBS [40]

- Penicillin G, 5 million units IV as initial dose, then 2.5–3 million units every 4 hours until delivery
  or Ampicillin, 2 g IV as initial dose, then 1 g IV every 4 hours until delivery
- In Penicillin-allergic patients give: Cefazolin, 2 g IV as initial dose, then 1 g IV every 8 hours until delivery
  or Vancomycin, 1 g IV every 12 hours until delivery
  or Clindamycin 900 mg IV every 8 hours until delivery
- Antibiotics given for latency in the setting of PPROM that include ampicillin are adequate for GBS prophylaxis. If other regimens are used, GBS prophylaxis should be initiated in addition
- Erythromycin should not be given.
- No intramuscular or oral regime has been shown to be effective.
- A negative GBS screen result is considered valid for 5 weeks. If a patient with history of preterm labor is readmitted with signs and symptoms of preterm labor and had a negative screen result more than 5 weeks prior, she should be rescreened and managed accordingly

Fig. 5.2 Intrapartum intravenous antibiotic prophylaxis for GBS [40]

5.10.4 Magnesium Sulfate for Neuroprotection

Women with PPROM before $32^{0/7}$ weeks who are thought to be at risk of imminent delivery should be considered candidates for fetal neuroprotective treatment with magnesium sulfate (ACOG) [4]. Use of magnesium sulfate for fetal neuroprotection when birth is anticipated has shown to reduce the risk of cerebral palsy in surviving infants [41, 42]. Physicians electing to use magnesium sulfate for fetal neuroprotection should develop specific guidelines [41].

A recommended dose of magnesium sulfate according to NICE [11] is 4 g intravenous bolus over 15 min, followed by an intravenous infusion of 1 g/h until birth or for 24 h (whichever is sooner).

5.10.5 Tocolysis

There are no recommendations for starting tocolytic therapy in women with PPROM [8, 43]. When women with PPROM go into labor, therapeutic tocolysis is not recommended [4, 8].

5.11 Indications to Stop Expectant Management

The following are the indications to stop conservative management:

- If gestational age reaches 34 weeks.
- There is evidence of chorioamnionitis.
- Pulmonary maturity is achieved.

5.11.1 Transport to Tertiary Care Referral Facility

To achieve better perinatal outcomes, delivery of preterm infants should occur at facilities capable of providing the appropriate level of neonatal resuscitative and supportive care according to gestational age**.** Thus if a good NICU is not available, the mother should be transferred to higher center after initiating treatment.

5.11.2 Reseal of Ruptured Membranes

If amniocentesis results in PPROM, the membranes usually reseal with restoration of normal amniotic fluid volume, whereas in women with spontaneous PPROM, resealing of the membranes and restoration of normal amniotic fluid volume occur rarely (estimated at 2.8–13%) [7].

5.11.3 Home Care

ACOG does not recommend home care in cases of PPROM [4]. The women should be admitted to the hospital and under constant supervision for promptly diagnosing evidence of infection and for fetal well-being.

5.11.4 Cervical Cerclage in Cases of PPROM [4]

There are no recommendations whether a cervical cerclage stitch if already applied should be removed after PPROM and decision should be individualized.

5.11.5 Amnioinfusion

Amnioinfusion is neither recommended in women with PROM during labor nor in very preterm PROM as a method to prevent pulmonary hypoplasia [8].

5.11.6 Human Immunodeficiency Virus (HIV) Infection and PPROM

In all cases of HIV-positive patients and PPROM, standard antepartum and intrapartum treatment

guidelines should be followed. Earlier it was said that the more the duration of membrane rupture in labor, the higher the risk of transmission to the newborn. The recent evidence suggests that the duration of membrane rupture is not correlated with risk of vertical transmission in patients who are on highly active antiretroviral therapy as they have a low viral load. The management should be decided on individual basis after full discussion with the patient depending on gestational age, viral load, and duration since the patient is on antiretroviral therapy [4].

5.11.7 Role of Fibrin Glue

There is no recommendation regarding the use of fibrin sealants for second-trimester oligohydramnios caused by PPROM.

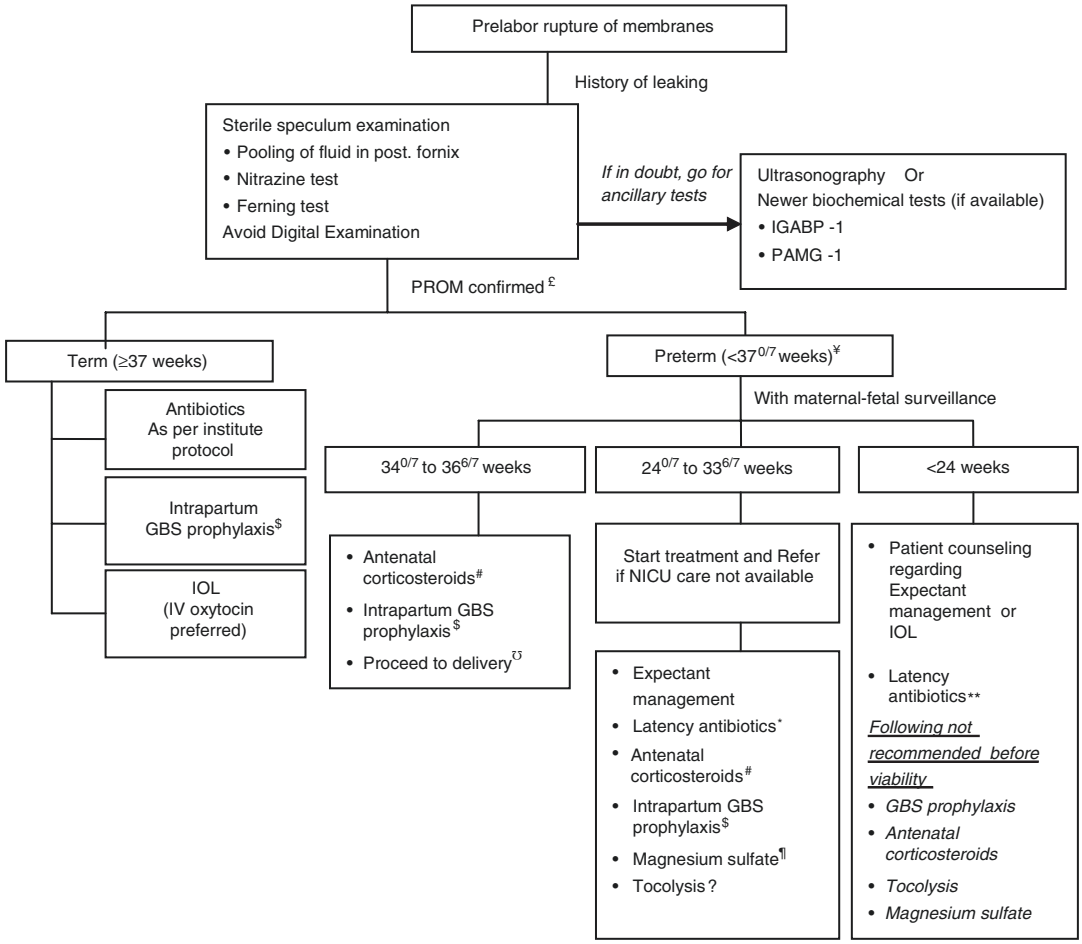
5.11.8 Future Pregnancy (ACOG) [4]

Women with a single gestation and history of prior spontaneous preterm birth due to PPROM should be offered progesterone supplementation starting at 16 weeks to 24 weeks.

5.12 Conclusion

PPROM is a major cause of perinatal morbidity and mortality. Prognosis mainly depends on gestational age at presentation. A timely and accurate diagnosis of PROM along with a good NICU is very important for a favorable pregnancy outcome. Once the diagnosis is confirmed, management includes admission to hospital, administration of antenatal corticosteroids, and latency antibiotics. Utmost aseptic precautions should be taken for the sake of the mother and fetus. Per vaginum examination should be done only when indicated. Careful observation should be made for any clinical evidence of developing chorioamnionitis and other complications and termination of pregnancy decided accordingly. Otherwise delivery should be accomplished once a favorable gestational age of 34 weeks is reached.

LABUOR ROOM PROTOCOL FOR MANAGEMENT OF PROM BASED ON VARIOUS RECOMMENDATIONS



£ *Deliver if evidence of intra-amniotic infection, non reassuring fetal heart tracing, significant abruption, cord prolapse or active labor*

¥ *Terminate pregnancy if signs of chorioamnionitis appear or fetal lung maturity achieved*

* *IV Ampicillin 2g x 6hrly + IV Erythromycin 250mg x 6hrly for 48 hr followed by Oral Amoxycillin 250mg x 8hrly + Erythromycin 333mg x 8hrly for 5 days OR Oral Erythromycin 250mg x 6hrly for 10 days*

** *may be considered as early as 20 0/7 weeks*

$ *Refer Fig. 5.2*

Inj Dexamethasone 6mg I/M x 4 doses, 12 hrs apart Inj Betamethasone 12mg I/M x 2 doses, 24 hrs apart

ʊ *May be managed expectantly PPROMT trial[25]*

¶ *If imminent delivery before 32 weeks*

References

1. DC Dutta's Textbook of Obstetrics, 8th ed. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd; 2015.
2. ACOG Practice Bulletin No.80: Premature Rupture of Membranes. Clinical management guidelines for obstetrician-gynecologists. ACOG Committee on Practice Bulletins-Obstetrics. Obstet Gynecol. 2007 Apr;109(4):1007–19.
3. Arias, Daftary, Bhide. Practical guide to high- risk pregnancy & delivery a South Asian perspective, 3/e. New Delhi: Elsevier; 2011.
4. ACOG Practice Bulletin No.160: Premature Rupture of Membranes. Clinical management guidelines for obstetrician-gynecologists. ACOG Committee on Practice Bulletins. Obstet Gynecol. 2016 Jan;127(1):e39–51.
5. Mercer BM. Preterm premature rupture of the membranes. Obstet Gynecol. 2003;101(1):178–93.
6. Cunningham FG, Leveno KJ, Bloom SL, Spong CY, Dashe JS, Hoffman BL, Casey BM, Sheffield JS, editors. Williams obstetrics. 24th ed. USA: The McGraw-Hill Education; 2014.
7. Caughey AB, Robinson JN, Norwitz ER. Contemporary diagnosis and management of preterm premature rupture of membranes. Rev Obstet Gynecol. 2008;1(1):11–22.
8. Royal College of Obstetricians & Gynaecologists. Preterm Prelabour Rupture of Membranes. Green-top Guideline No.44; 2010.
9. El-Messidi A, Cameron A. Diagnosis of premature rupture of membranes: inspiration from the past and insights for the future. J Obstet Gynaecol Can. 2010;32(6):561–9.
10. Medina TM, Hill DA. Preterm premature rupture of membranes; diagnosis and management. Am Fam Physician. 2006;73(4):659–64.
11. NICE guideline. Preterm labour and birth; 2015. ng25.
12. Abdelazim IA. Insulin like growth factor binding protein1 (Actim PROM test) for detection of premature rupture of fetal membranes. J Obstet Gynaecol Res. 2014;40(4):961–7.
13. Cousins LM, Smok DP, Lovett SM, Poeltler DM. Amnisure placental a microglobulin-1 rapid immunoassay versus standard diagnostic methods for detection of rupture of membranes. Am J Perinatol. 2005;22(6):317–20.
14. Hallak M, Bottoms SF. Accelerated pulmonary maturation from preterm premature rupture of membranes: a myth. Am J Obstet Gynecol. 1993;169(4):1045–9.
15. Dare MR, Middleton P, Crowther CA, Flenady VJ, Varatharaju B. Planned early birth versus expectant management (waiting) for prelabour rupture of membranes at term (37 weeks or more). Cochrane Database Syst Rev. 2006;25(1):CD005302.
16. Hannah ME, Ohlsson A, Farine D, Hewson SA, Hodnett ED, Myhr TL, Wang EE, Weston JA, Willan AR. Induction of labour compared with expectant management for prelabour rupture of the membranes at term. TERMPROM Study Group. N Engl J Med. 1996;334(16):1005–10.
17. NICE clinical guideline: induction of labour; 2008.
18. Rouse DJ, Weiner SJ, Bloom SL, Varner MW, Spong CY, Ramin SM, et al. Failed labor induction: toward an objective diagnosis. Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD) Maternal-Fetal Medicine Units Network (MFMU). Obstet Gynecol. 2011;117:267–72.
19. Spong CY, Berghella V, Wenstrom KD, Mercer BM, Saade GR. Preventing the First Cesarean Delivery: Summary of a Joint Eunice Kennedy Shriver National Institute of Child Health and Human Development, Society for Maternal Fetal Medicine, and American College of Obstetricians and Gynecologists Workshop. Obstet Gynecol. 2012;120(5):1181–93.
20. Flenady V, King JF. Antibiotics for prelabour rupture of membranes at or near term. Cochrane Database Syst Rev. 2002;3:CD001807. https://doi.org/10.1002/14651858.CD001807.
21. Passos F, Cardoso K, Coelho AM, Graça A, Clode N, Mendes da Graça L. Antibiotic prophylaxis in premature rupture of membranes at term: a randomized controlled trial. Obstet Gynecol. 2012;120(5):1045–51. https://doi.org/10.1097/AOG.0b013e31826e46bc.
22. Kenyon S, Boulvain M, Neilson J. Antibiotics for preterm rupture of membranes. Cochrane Database Syst Rev. 2003;2:CD001058.
23. Simhan HN, Canavan TP. Preterm premature rupture of membranes: diagnosis, evaluation and management strategies. BJOG. 2005;112(Suppl 1):32–7.
24. Buchanan SL, Crowther CA, Levett KM, Middleton P, Morris J. Planned early birth versus expectant management for women with preterm prelabour rupture of membranes prior to 37 weeks' gestation for improving pregnancy outcome. Cochrane Database Syst Rev. 2010;3:CD004735.
25. Morris JM, Roberts CL, Bowen JR, Patterson JA, Bond DM, Algert CS, Thornton JG, Crowther CA, PPROMT Collaboration. Immediate delivery compared with expectant management after preterm pre-labour rupture of the membranes close to term (PPROMT trial): a randomised controlled trial. Lancet. 2016;387(10017):444–52. https://doi.org/10.1016/S0140-6736(15)00724-2.
26. Gyamfi-Bannerman C, Thom EA, Blackwell SC, Tita AT, Reddy UM, Saade GR, et al. Antenatal betamethasone for women at risk for late preterm delivery. NICHD maternal–fetal medicine units network. N Engl J Med. 2016;374(14):1311–20. https://doi.org/10.1056/NEJMoa1516783.
27. American College of Obstetricians and Gynecologists. Practice advisory: antenatal corticosteroid administration in the late preterm period; 2016.
28. ACOG Practice Bulletin No. 188: Prelabor Rupture of Membranes. Clinical Management Guidelines for Obstetrician-Gynecologists. Obstet Gynecol. 2018 Jan;131(1):e1-e14. https://doi.org/10.1097/AOG.0000000000002455.

29. American College of Obstetricians and Gynecologists Committee on Obstetric Practice. ACOG Committee Opinion No. 677: Antenatal corticosteroid therapy for fetal maturation; 2016.
30. Sharp GC, Stock SJ, Norman JE. Fetal assessment methods for improving neonatal and maternal outcomes in preterm prelabour rupture of membranes. Cochrane Database Syst Rev. 2014;10:CD010209. https://doi.org/10.1002/14651858.CD010209.
31. van der Heyden JL, van der Ham DP, van Kuijk S, Notten KJ, Janssen T, Nijhuis JG, et al. Outcome of pregnancies with preterm prelabor rupture of membranes before 27 weeks' gestation: a retrospective cohort study. Eur J Obstet Gynecol Reprod Biol. 2013;170(1):12530. https://doi.org/10.1016/j.ejogrb.2013.06.012.
32. Clinical Practice Guideline SOGC. Antibiotic therapy in preterm premature rupture of the membranes. J Obstet Gynaecol Can. 2009;31(9):863–7.
33. Mercer BM, Miodovnik M, Thurnau GR, Goldenburg RL, Das AF, Ramsey RD, et al. Antibiotic therapy for reduction of infant morbidity after preterm premature rupture of the membranes. A randomized controlled trial. JAMA. 1997;278:989–95.
34. Kenyon SL, Taylor DJ, Tarnow-Mordi W, ORACLE Collaborative Group. Broad-spectrum antibiotics for preterm, prelabour rupture of fetal membranes: the ORACLE I randomised trial. Lancet. 2001;357:979–88.
35. Royal College of Obstetricians and Gynaecologists. Greentop Guideline No. 7: antenatal corticosteroids to reduce neonatal morbidity and mortality. London: RCOG; 2010.
36. Use of Antenatal Corticosteroids in Preterm Labour. Operational guidelines. GOI Child Health Division mohfw; 2014.
37. WHO Recommendations on Interventions to Improve Preterm Birth Outcomes. WHO Library Cataloguing-in-Publication Data; 2015.
38. Narava S, Rajaram G, Ramadevi A, Prakash GV, Mackenzie S. Prevention of perinatal group B streptococcal infections: a review with an Indian perspective. Indian J Med Microbiol. 2014;32(1):6–12. https://doi.org/10.4103/0255-0857.124286.
39. Verani JR, McGee L, Schrag SJ. Prevention of perinatal group B streptococcal disease—revised guidelines from CDC, 2010. Division of Bacterial Diseases, National Center for Immunization and Respiratory Diseases, Centers for Disease Control and Prevention (CDC). MMWR Recomm Rep. 2010;59(RR–10):1–36.
40. American College of Obstetricians and Gynecologists Committee on Obstetric Practice. ACOG committee opinion no. 485: prevention of early-onset group B streptococcal disease in newborns. Obstet Gynecol. 2011;117(4):1019–27.
41. Magnesium Sulfate before Anticipated Preterm Birth for Neuroprotection. Committee opinion no. 455. American College of Obstetricians and Gynecologists. Obstet Gynecol. 2010;115:669–71.
42. Magnesium Sulfate Use in Obstetrics. Committee Opinion No. 652. The American College of Obstetricians and Gynecologists Committee on Obstetric Practice Society for Maternal-Fetal Medicine; 2016.
43. Mackeen AD, Seibel-Seamon J, Muhammad J, Baxter JK, Berghella V. Tocolytics for preterm premature rupture of membranes. Cochrane Database Syst Rev. 2014;2:CD007062. https://doi.org/10.1002/14651858.CD007062.

6 Foetal Growth Restriction as IUGR is Obsolete

Manju Puri and Anuradha Singh

6.1 Introduction

Foetal growth restriction (FGR) refers to a pathological condition in which the foetus is unable to grow to its genetically determined biological potential and is below the tenth percentile of the estimated foetal weight for that gestational age. The term FGR needs to be differentiated from the term small for gestational age (SGA) for a foetus in utero. Small for gestational age foetus refers to a foetus with an estimated weight less than tenth percentile on ultrasound. These foetuses may or may not be growth restricted. Majority of SGA foetuses (50–70%) are not growth restricted but are constitutionally small but are healthy and appropriate for the maternal height, weight, ethnic origin and foetal sex [1]. Small mothers give birth to small babies. Of the remaining, 10–15% are small due to inherent defect in the foetus, and the remaining 20–35% are due to chronic placental insufficiency. Physiological SGA babies or constitutionally small babies are healthy and have perinatal morbidity and mortality comparable to appropriate for gestational age babies, whereas FGR babies have higher perinatal mortality and morbidity [2]. Foetal growth restriction is more common in neonates with severe SGA that have a weight below the third percentile [3]. However, average for gestational age AGA foetuses may be growth restricted sometimes. Inspite of being above tenth percentile these foetuses have evident signs of chronic placental insufficiency and foetal compromise such as reduced abdominal circumference, oligohydramnios and abnormal Doppler studies in utero that often result in intrapartum stillbirth. The placenta in these cases is smaller than normal, calcified and meconium stained. These are often missed during antenatal examination.

The challenge to the obstetrician managing foetal growth restriction is to differentiate growth restricted foetuses from constitutionally small healthy foetuses and to monitor and optimally time the delivery of growth restricted foetuses and at the same time avoid inadvertent harm to the healthy foetuses by premature intervention. The aim is to minimize adverse sequelae in growth restricted foetuses due to suboptimal intrauterine environment and balance it against the risks of prematurity.

6.2 Aetiopathogenesis

Foetal growth restriction can be placental mediated or non-placental mediated. The placental-mediated growth restriction is secondary to conditions like hypertensive disorders of pregnancy, antiphospholipid syndrome and diabetes mellitus with vasculopathy. Non-placental-mediated growth restriction is due to intrinsic

M. Puri (✉) · A. Singh
Department of Obstetrics & Gynaecology,
Lady Hardinge Medical College, New Delhi, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_6

Table 6.1 Causes of foetal growth restriction

Maternal	Placental	Foetal
• Smoking • Substance abuse: tobacco, alcohol cocaine, narcotics • Protein calorie malnutrition • Uterine malformation • Hypertensive disorders of pregnancy • Renal disease • Pre-gestational diabetes mellitus with vasculopathy • Cyanotic heart disease • Autoimmune disorders: SLE • Thrombophilia • Haemoglobinopathies	• Placental abnormalities: circumvallate placenta, chorioangioma • Placenta previa • Chronic placental abruption • Abnormal cord insertion: marginal or velamentous	• Multiple pregnancy: twin-to-twin transfusion syndrome, discordant twins • Structural anomalies: gastroschisis • Chromosomal/genetic anomalies: trisomy 13, 18 • Intrauterine infections: *Cytomegalovirus*, toxoplasma, rubella, malaria, syphilis • Inborn errors of metabolism • Exposure to teratogens like warfarin, valproic acid

defects in the foetus like chromosomal abnormalities, structural anomalies, metabolic disorders or intrauterine foetal infections like cytomegalovirus, toxoplasma, malaria, syphilis, etc.

In placental-mediated causes, there is chronic placental insufficiency due to poor placentation and/or thrombosis compromising the blood flow and supply of nutrients and oxygen to the foetus. Conditions affecting the oxygen-carrying capacity of blood in the mother like haemoglobinopathies, cyanotic heart disease, high altitude, smoking, etc. can also result in foetal growth restriction.

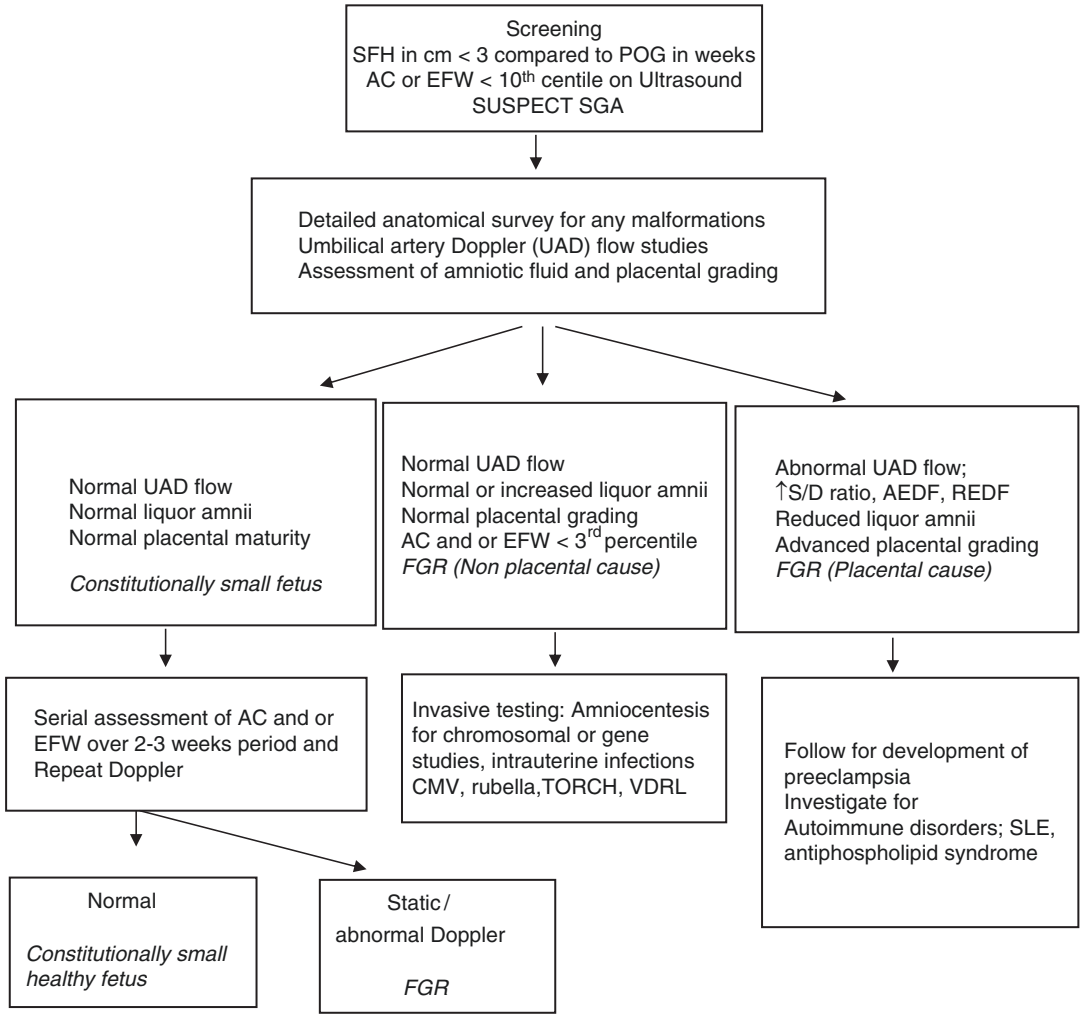
The various causes of foetal growth restriction can be divided into maternal, placental and foetal (Table 6.1).

6.3 Diagnosis and Differential Diagnosis (Flow Chart 1)

The condition is suspected on clinical or ultrasound screening. On clinical examination the symphysiofundal height in centimetres corresponds to the period of gestation from 24 to 36 weeks, and a disparity or lag of more than three between measurement of symphysiofundal height (SFH) in cm and period of gestation in weeks justifies an evaluation for FGR [4]. SFH should be recorded on customized charts. A single measurement of symphysiofundal height below the tenth centile or a static growth across all centiles is an indication for ultrasound evaluation for foetal biometry and foetal weight. However, in women who are obese or have multiple pregnancies, hydramnios or fibroids SFH measurement is not a reliable tool for suspecting FGR or assessing foetal growth [5].

FGR is also suspected on ultrasonographic examination by a lag in foetal biometry that is BPD, HC, AC, FL and estimated foetal weight for that gestational age. Of these parameters, foetal abdominal circumference (AC) or EFW of less than tenth percentile is the most reliable for diagnosis of SGA foetus [5]. Foetal abdominal circumference is the first to get affected due to depletion of liver glycogen and loss of subcutaneous fat in a foetus deprived of nourishment. However, it is important to have an accurate assessment of gestational age preferably by crown-rump length (CRL) in a first trimester ultrasound. Serial assessment of AC and/or EFW for growth velocity should be done at least 3 weeks apart to differentiate normally growing constitutionally small foetuses from growth restricted foetuses with reduced foetal growth velocity [5]. A detailed anatomical survey of the foetus to rule out any structural anomaly should be done in all foetuses with growth below the tenth centile.

The presence of advanced placental maturity or grading and reduced liquor suggests chronic placental insufficiency and foetal growth restriction [6]. Doppler flow studies are effective in diagnosing placental cause of foetal growth



Flow chart 1 Approach to a woman suspected with SGA foetus

restriction. Umbilical artery Doppler flow study is done initially and is followed by middle cerebral artery and venous Doppler studies if required. As the pregnancy advances, the diastolic flow in umbilical artery increases with resultant decrease in the systolic to diastolic (S/D) flow ratio. Normally S/D ratio is less than three after 30 weeks of gestation. In patients with placental cause of FGR, this increase in flow is compromised and is reflected progressively as an increased S/D ratio, absent end-diastolic flow and reversed end-diastolic flow.

In women with evidence of chronic placental insufficiency, further workup for the cause should be initiated. The woman should be followed up for development of preeclampsia, subjected to investigations for the diagnosis of antiphospholipid syndrome, namely, antiphospholipid antibodies, anticardiolipin antibodies, lupus anticoagulant and anti-beta-2 glycoprotein 1 antibodies. The woman is screened for autoimmune disorders like systemic lupus erythematosus by testing for antinuclear antibodies or any infections caused by VDRL and TORCH profile.

In foetuses with normal Doppler flow studies and normal liquor amnii, serial ultrasound examinations are carried out to see the growth velocity, and if found normal, the foetus is likely

to be constitutionally small. If the EFW or AC is less than third centile, it is suggestive of severe SGA and is likely to be FGR. Inherent foetal defects and chromosomal or genetic or intrauterine infections should be suspected in these women. Invasive testing in the form of amniocentesis for karyotyping or gene analysis may be offered in women with severe and/or early FGR, presence of normal or excessive liquor, structural anomalies and normal Doppler flow. It is not indicated in women with FGR due to chronic placental insufficiency.

6.4 Complications

Growth restricted foetuses are at an increased risk of stillbirth and neonatal mortality and morbidity. The risk is directly proportional to the severity of growth restriction and prematurity. The neonate is at an increased risk of hypoglycaemia, hypothermia, hyperbilirubinemia, respiratory distress syndrome, intraventricular haemorrhage, necrotizing enterocolitis, sepsis, seizures, NICU admission and neonatal death. These babies are also predisposed to long-term morbidity in the form of cognitive defects and diseases like type 2 diabetes mellitus, obesity, coronary heart disease, etc.

Table 6.2 Risk factors for FGR

History
Maternal age > 35 yrs
Smoker
Substance use
Hypertensive
Diabetic with vasculopathy
Renal disease
Autoimmune disorder SLE, APS
Cyanotic heart disease
Past history of preeclampsia, still birth, FGR
History of dryg intake like valproic acid, warfarin
History of either parent been born SGA
New factors detected or developing during course of pregnancy
Multiple pregnancies
Threatened abortion
Unexplained antepartum haemorrhage
Poor weight gain
Hypertensive disorders of pregnancy: preeclampsia, severe hypertension
Oligohydramnios
Postdated pregnancy
Examination
BMI< 18 or >30
Malnutrition
SFH less than POG
Reduced liquor
Multiple pregnancy
Investigations
First trimester PAPP A <0.4 MOM
Abnormal Doppler flow studies of uterine artery 20–24 weeks
Echogenic bowel
Lag in abdominal circumference on ultrasound
Oligohydramnios

6.5 Management

6.5.1 Antepartum

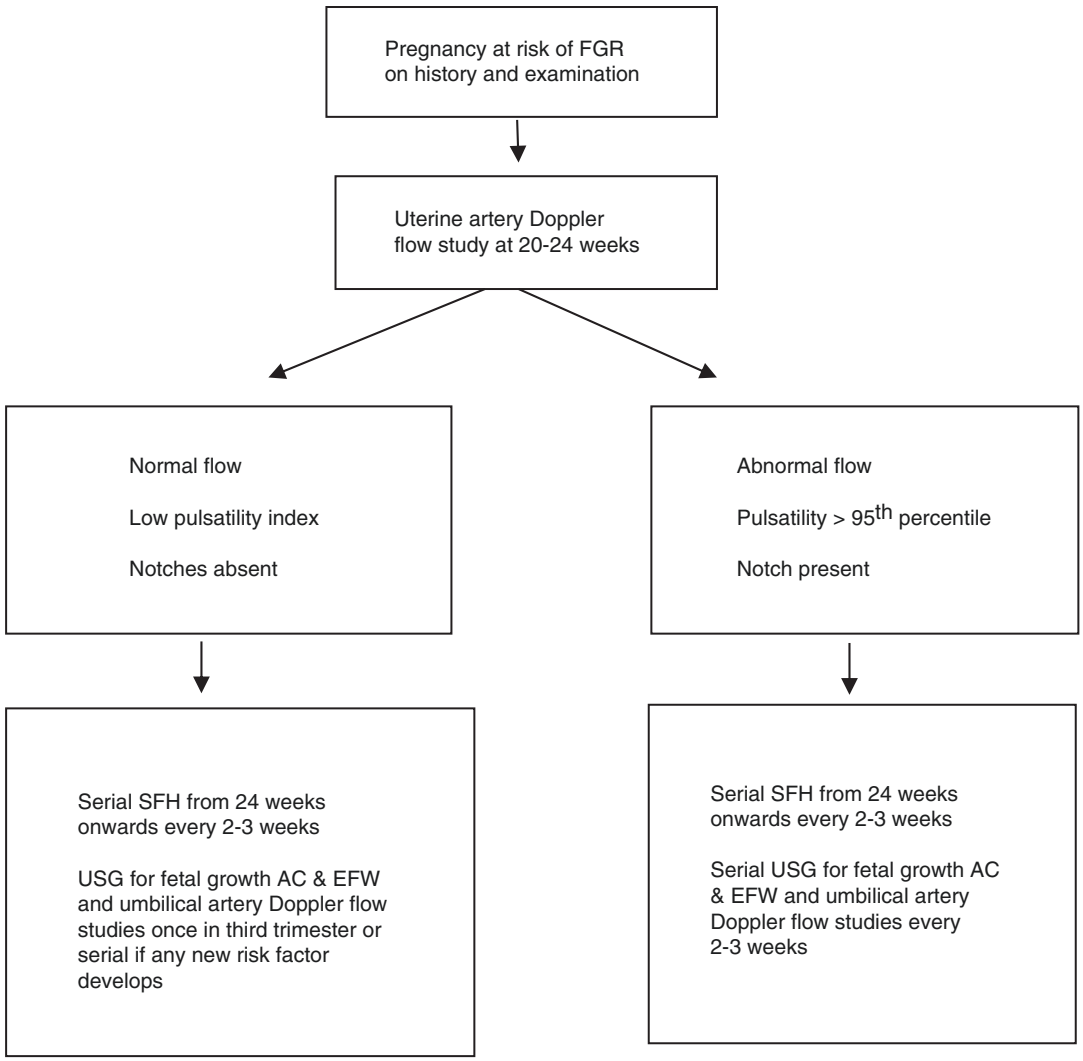
6.5.1.1 Risk Assessment for Foetal Growth Restriction (Table 6.2)

All women should be screened by a detailed history and examination to identify those at high risk of FGR at the time of booking. The various risk factors to be elicited on history are maternal age >35 years, smoking, substance use, maternal diseases like diabetes mellitus with vascular involvement, hypertension, renal disease, autoimmune diseases such as antiphospholipid syndrome, systemic lupus erythematosus and cyanotic heart disease. Past history of preeclampsia, stillbirth or FGR baby is obtained. A detailed examination identifies risk factors like low BMI <18 or high BMI >30 and poor nutrition. Certain conditions may be detected or develop during the course of pregnancy like PAPP A <0.4 MOM on first trimester screening, threatened abortion, echogenic bowel on ultrasound, unexplained antepartum haemorrhage, hypertensive disorder of pregnancy, abruptio placentae, poor weight gain, oligohydramnios, postdated pregnancy, etc. which may increase the risk of FGR in the foetus [4].

6.5.1.2 Antepartum Monitoring and Management of At-Risk Pregnancies (Flow Chart 2)

Women with previous history of chronic placental insufficiency syndromes like preeclampsia or APS can be started on low-dose aspirin 60–100 mg daily from 12 to 16 weeks of pregnancy till 36 weeks of gestation [7]. At-risk women are encouraged to stop smoking and substance use. Malnourished women are advised to improve their diet.

All women at risk of growth restricted foetus should be offered screening with uterine artery Doppler flow study at 20–24 weeks of gestation. In normal pregnancies, the blood flow in uterine arteries increases with disappearance of notch due to decrease in resistance consequent to trophoblastic invasion of myometrial uterine spiral arteries. However, if the flow is abnormal, that is, pulsatility index is more than 95th percentile, and/or notching persists, the woman should be offered serial ultrasound assessment for foetal biometry, estimated foetal weight and umbilical artery Doppler flow studies from 26 to 28 weeks onwards. If it is normal, then she is offered one scan in the third trimester for foetal biometry and



Flow chart 2 Monitoring of a pregnancy at risk of FGR

umbilical artery Doppler flow. If she develops any new high risk factor like preeclampsia during the course of pregnancy, serial ultrasonographic examinations for early detection of growth restriction are advised. Careful abdominal palpation of fundal height and measurement of symphysiofundal height (SFH) are recommended at each antenatal visit from 24 weeks onwards for early detection of any lag in growth.

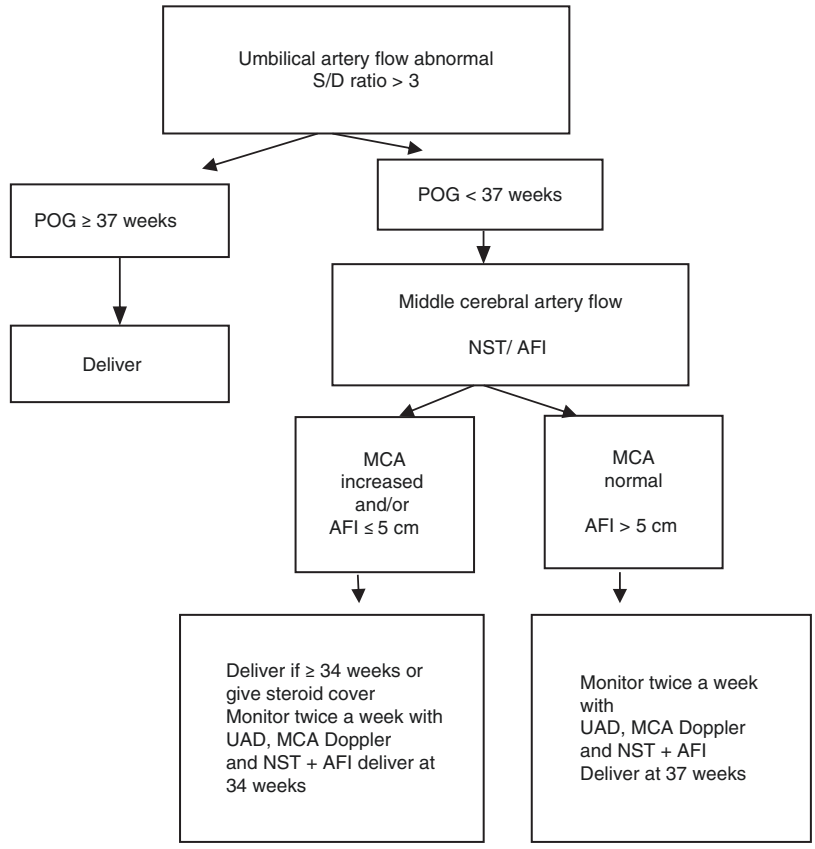
6.5.1.3 Antepartum Management of Pregnancies Diagnosed with Foetal Growth Restriction

Management of pregnancies diagnosed with foetal growth restriction depends on the cause of FGR whether placental or non-placental, period of gestation and the severity of umbilical artery Doppler flow abnormality. Umbilical artery Doppler is the primary tool for surveillance, and if normal, it is repeated every 2 weeks, but if abnormal, the management depends on the severity of abnormality. Nonstress test and venous Doppler compliment the umbilical artery Doppler in monitoring and often help in deciding the time to trigger the delivery in preterm foetuses with increasing severity of umbilical artery Doppler changes.

End-Diastolic Flow Present (Raised SD Ratio): Flow Chart 3.

These women are usually hospitalized but can be monitored on outpatient basis. If the period of gestation is ≥37 weeks, consider delivery of the baby. If it is <37 weeks, the woman is subjected to twice-weekly umbilical artery and middle cerebral artery Doppler and modified biophysical profile (nonstress test and assessment of amniotic fluid volume) till 37 weeks. Presence of beat-to-beat variability is the most important finding on nonstress test, and assessment of amniotic fluid is best based on the single deepest vertical pocket. Single course of antenatal corticosteroids is administered if abnormality is detected at

Flow chart 3
Management of an FGR pregnancy with end-diastolic flow present

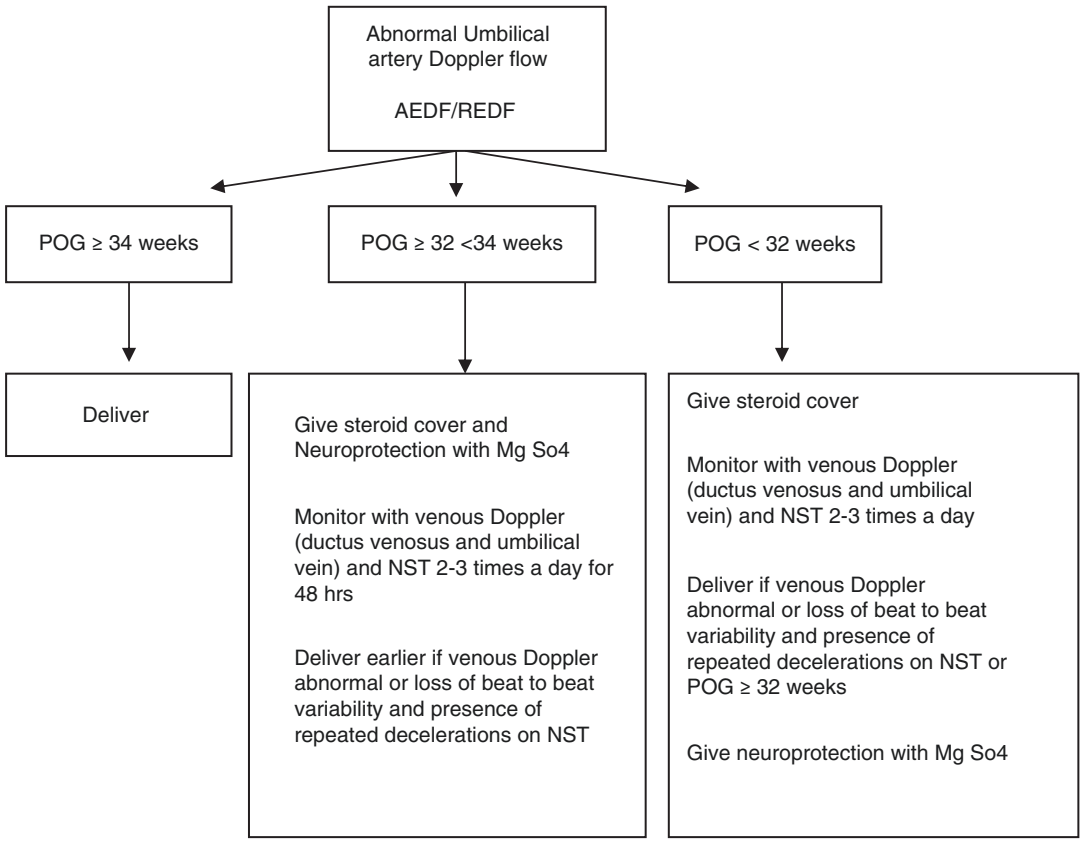


<34 weeks of gestation. The woman is asked to keep daily foetal kick count and report if the foetal movements are <10/12 h. Weekly symphysiofundal height and abdominal girth charting is done along with foetal biometry on ultrasound to assess for the growth of the foetus. Delivery is considered if there is no growth or it plateaus.

Absent or Reversed End-Diastolic Flow (AEDF or REDF) (Flow Chart 4).

These women are hospitalized. If the period of gestation is ≥34 weeks, consider delivery of the baby. If <34 weeks but ≥32 weeks, give antenatal steroid cover and deliver. Careful and close monitoring of the foetus is desirable for 48 h while waiting for the steroid cover to be effective. The woman is subjected to venous Doppler (ductus venosus and umbilical vein) and NST once or twice a day, and earlier intervention is indicated if ductus venous Doppler becomes abnormal, that is, there is intermittent absent or reversed flow velocity or there is loss of beat-to-beat variability or presence of decelerations on NST. The aim is to deliver the hypoxic foetus before it becomes acidotic. In women with period of gestation of <32 weeks, the woman is closely observed after administering steroid cover till changes in venous Doppler or NST are indicative of delivery. The expectant management can extend up to 2 weeks in some cases. The aim is to gain maturity to improve the survival of the neonate. Magnesium sulphate is considered for neuroprotection during labour if the delivery is envisaged before 32 weeks [8].

Pregnancies with AEDF or REDF in umbilical artery Doppler are preferably terminated by caesarean section; however, those with end-diastolic flow present that is raised S/D ratio can be induced.



Flow chart 4 Management of FGR pregnancies with AEDF or REDF

6.6 Intrapartum Management

The delivery should be conducted at a place where facilities for doing an emergency caesarean section and advanced neonatal care are available; otherwise in utero transfer of the foetus to a higher centre is offered. Continuous foetal heart rate monitoring is desirable in the intrapartum period. If it is not available, early ARM and close monitoring of foetal heart rate by intermittent auscultation post contraction every 15–30 min in the first stage and after every contraction in the second stage is indicated. The rate of emergency caesarean section is high. Foetuses with a reactive NST, good beat-to-beat variability and a DVP of >1 cm prior to induction of labour do better.

6.7 Care of the Newborn

By virtue of chronic placental insufficiency and resultant malnutrition and preferential redistribution of blood to the brain, growth restricted neonates are predisposed to complications like hypothermia, hypoglycaemia, hyperbilirubinemia, polycythaemia, hyperviscosity, necrotizing enterocolitis and sepsis. Prematurity further adds to the magnitude of these complications and predisposes them to respiratory distress syndrome and intraventricular haemorrhage. Care has to be taken to prevent these complications by maintaining temperature, normoglycaemia, asepsis and slow initiation of feeds.

These babies need long-term follow-up, as suboptimal intrauterine environment and growth restriction have been associated with cognitive and behavioural problems like attention deficit hyperactivity disorder and short-term memory difficulties and metabolic syndrome later in life [9].

6.8 Summary

Foetal growth restriction is associated with increased morbidity and mortality of the foetus either due to inherent structural, chromosomal or metabolic defect in the foetus or due to suboptimal intrauterine environment consequent to defective placentation or due to infections, drugs or toxins. Growth restricted foetuses need to be differentiated from healthy constitutionally small SGA foetuses as these foetuses are not at increased risk of morbidity and mortality. Abdominal circumference and estimated foetal weight of less than tenth percentile on ultrasound are reliable parameters for the diagnosis of FGR. However, serial growth scans help in differentiating healthy from growth restricted foetuses. Umbilical artery Doppler flow is a primary surveillance tool to diagnose and monitor the severity of placental insufficiency. Term growth restricted foetuses can be delivered, but in preterm growth restricted foetuses, there is a need to balance the adverse effects of growth restriction with the risks of prematurity. Loss of beat-to-beat variability in NST and ductus venous Doppler studies help in deciding the trigger to deliver a premature growth restricted foetus. These deliveries should be carried out at facilities with advanced neonatal intensive care units and trained manpower to manage growth restricted babies. The mode of delivery is decided based on the cause, severity, Bishop's score and other patient characteristics. Due to long-term morbidities in these babies, a long-term follow-up is required.

References

1. Alberry M, Soothill P. Management of fetal growth restriction. Arch Dic Child Fetal Neonatal Ed. 2007;92:62–7.
2. Resnik R. Intrauterine growth restriction. Obstet Gynecol. 2002;99:490–6.
3. Chang TC, Robson SC, Boys RJ, et al. Prediction of small for gestational age infant: which ultrasonic measurement is best? Obstet Gynecol. 1992;80:1030–8.
4. Jelks A, Cifuentes R, Ross MG. Clinician bias in fundal height measurement. Obstet Gynecol. 2007;110(4):892–9.
5. Green top Guidelines No. 31 The investigations and management of the small for gestational age fetus RCOG 2013 London.
6. Chamberlain PF, Manning FA, Morrison I, et al. Ultrasound evaluation of amniotic fluid volume.

The relationship of marginal and decreased amniotic fluid volumes to perinatal outcome. Am J Obstet Gynecol. 1984;150(3):245–9.
7. guidelines No SOGC. 295 Intrauterine growth restriction: screening, diagnosis and management. J Obstet Gynaecol Can. 2013;35(8):741–8.
8. Practice bulletin No. 134. Fetal growth restriction. Obstet Gynecol. 2013;121(5):1122–30.
9. Barker DJ, Gluckman PD, Godfrey KM, et al. Fetal nutrition and cardiovascular disease in adult life. Lancet. 1993;341:938–41.

7 Hypertensive Disorders: Delivery Management

Girija Wagh and Rohan Wagh

7.1 Introduction

Hypertensive disorders in pregnancy (HDPs) are on the rise and significantly seen to contribute to maternal morbidity and mortality [1, 2]. Delivering these patients is an additional challenge due to the dynamic nature of the delivery process, compromised maternal fetal unit, and sensitive and hyperresponsive nature of the maternal vascular responses which may suddenly cause an apparently mild HDP mother go into a tumultuous course. Pain, fluids, and alterations in the hemodynamic parameters are significant contributors to such a course. Additionally one has to try and fathom the systemic involvements due to widespread endothelial dysfunction, assess the coagulation system, and remember that every mother with hypertension has a potential risk of developing complications such as placental abruption, postpartum hemorrhage, disseminated intravascular coagulation (DIC), eclampsia, acute renal failure, respiratory distress, cardiomyopathy, and sometimes death. In addition one has to remember that every apparently normotensive woman has a risk of developing hypertension during labor and entails vigilance during the processes of delivery. Preeclampsia is a misnomer and should ideally be called as *Gestosis* which means disordered pregnancy associated with multisystem involvement [3].

7.2 Background of HDP Context Delivery

It is important to understand some basic physiological changes leading to clinical situation in a hypertensive mother to better understand the management during delivery. Blood pressure measurement that equals 150/100 mm of Hg or more should be considered as severe hypertension in Indian context. The reason being it has been observed that low-risk mothers typically record blood pressure reading of 110/70 mm of Hg when assessed antenatally. Therefore wisdom lies in to starting antihypertensive medications at 150/100 mm of Hg. Rise of blood pressure is the sign which appears at the end of the pathogenesis of the disease process. This therefore mandates investigations and evaluation of the mother carefully. Also sometimes women may be normotensive but still have all the features of HDP, and these can be atypical presentations many times resulting in grave consequences.

G. Wagh (✉)
Obstetrics & Gynecology, Bharati Vidyapeeth University Medical College, Pune, Maharashtra, India

Girija Hospital & Fertility Center, Pune, Maharashtra, India

R. Wagh
BVDUMC, Pune, Maharashtra, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_7

7.2.1 Classification

HDPs are clinically classified as chronic hypertension (CHT) which means that the mother has evidence of hypertension recorded before completion of 20 weeks of gestation or has hypertension after 6 weeks of delivery. CHT is not associated with proteinuria. Women of CHT diagnosed antenatally are at risk of developing cerebrovascular accidents and myocardial infarctions and can convert into proteinuric hypertension eventually, during intranatal and postnatal period. Such an occurrence of proteinuria in already hypertensive mother is called as superimposed preeclampsia. Gestational hypertension is raised blood pressure which initiates after 20 weeks duration of pregnancy without proteinuria, and this reverts back to normal after delivery usually immediately or within a month. Preeclampsia is proteinuric hypertension presenting after 20 weeks of gestation and usually normalized by delivery within a week. Eclampsia is presence of seizures along with proteinuric hypertension. Preeclampsia can rarely occur earlier to 20 weeks of gestation in conditions such as gestational trophoblastic disease in both partial as well as complete mole [4] and multiple gestations. Medical conditions especially certain autoimmune disorders if preexisting can worsen or be unveiled during pregnancy, viz., Hughes's syndrome (APS), thrombocytopenic purpura, systemic lupus erythematosus (SLE), and hemolytic uremic syndrome (HUS), or disorders characterized by microangiopathic thrombosis or hemolysis are known to be associated with proteinuric hypertension before 20 weeks of gestation [5] (Table 7.1).

Categorizing HDP thus is important to help in predicting complications and therapy, but increasingly it has been identified to be one disease process. The fundamental difference between gestational hypertension and preeclampsia is proteinuria, and this in some way seems to cause more delirious effects. The above classification is adapted from the National High Blood Pressure Education Program Working Group classification and has better clinical utility. Clinical classification of preeclampsia is useful in predicting complications and thus planning appropriate clinical approach. Proteinuric hypertension before 34 weeks of gestation is called as early-onset preeclampsia (EPE) and is disease of feto-placental origin and associated with severe complications while late-onset preeclampsia (LPE) is disease which is a result of underlying maternal disease or abnormalities in maternal adaptation to pregnancy.

7.2.2 Pathology and Pathogenesis

The basic pathological features involve endothelial dysfunction, vasoconstriction, and platelet aggregation. These three elementary pathologies are responsible for all the widespread multisystemic involvements giving rise to various complications pertaining to each system (Table 7.2).

Clinical and laboratory assessments should focus on identifying these abnormalities.

Table 7.1 Classification of Hypertension in Pregnancy (ISHDP)

Gestational hypertension	Hypertension that develops after completion of 20 weeks pregnancy, returns to normal within or by 6 weeks, and is not associated with any other features of preeclampsia	6–7% of all pregnancies
Preeclampsia/eclampsia	Hypertension occurs after 20 weeks of pregnancy with significant urinary proteins When associated with fits, it is called eclampsia	5–7% of all pregnancies
Chronic hypertension	Blood pressure reading of ≤140/90 mm Hg present before pregnancy or before 20th week of pregnancy or recorded during 6 weeks postpartum	1–5% of all pregnancies
Preeclampsia superimposed on chronic hypertension	Occurrence of urinary proteins or any other features of preeclampsia in mothers with CHT	20–25% of all CHTs

Table 7.2 Systemic involvements associated with preeclampsia

System	Affliction
Cardiovascular	Cardiac failure, hypertension, thrombocytopenia coagulation failure
Renal	Oliguria, renal failure, tubular, and/or cortical necrosis
Respiratory	Acute respiratory distress syndrome (ARDS), pulmonary edema
CNS	PRES (posterior reversible encephalopathy syndrome, seizures, encephalopathy retinal detachment, cerebral edema, infarction, hemorrhage, cortical blindness
Liver	HELLP, failure or dysfunction, subcapsular hematoma, hepatic rupture

7.2.3 Hemodynamic Changes

The underlying hemodynamic changes of pregnancy and the ones contributed by the abovementioned complications need to be understood especially in the context of dynamism of delivery. Vasoconstriction is a result of raised arterial tone and stiffness and is a well-known characteristic of preeclampsia. Vasoconstriction leads to arterial resistance. Abnormal uterine artery notching and increased pulsatility index on color Doppler assessment are due to uterine artery resistance a reflection of vasoconstriction [6]. In addition the cardiac adaptation of mothers with preeclampsia is identified to be different from the mothers with uncomplicated pregnancy [7–10]. Venous hemodynamic dysfunction has been identified in women with preeclampsia [11]. This venous dysfunction is present for a longer period and much precedes the disease in early-onset preeclampsia (EPE) and is not seen preceding late-onset preeclampsia. The raised blood pressure is a composite result of raised cardiac output as well as raised peripheral resistance due to vasoconstriction with some studies attributing this more to raised cardiac output. Due to endothelial dysfunction, it is observed that the resultant intravascular volume of the mother is reduced considerably as the plasma is pushed the third compartments such as interstitial spaces, peritoneal cavity, etc. Vasoconstriction also is responsible for generalized hypoxia and poor tissue perfusion. In addition the vascular system is highly sensitive to medications, and therefore antihypertensive medications have to be carefully administered. Cerebral vasculature autoregulation system is altered, and there can be hyperresponsiveness due to micro hemorrhages and possibilities of vessel aneurysms in case of mothers with chronic long-standing hypertension.

During delivery there are major alterations in the cardiovascular and hemodynamic systems, and these have to be correlated with the background changes in women with hypertension. Uterine contractions cause considerable rise in left ventricular stroke volume and cardiac output. Birthing pain causes phenomenal increase in heart rate and cardiac output. All these contribute to nearly 20% rise in blood pressure, and this may be further augmented during the end of second stage when baby is being expelled especially due to the Valsalva maneuver. In addition there is increased oxygen consumption during delivery processes and may lead to acidosis in severe preeclampsia and eclampsia which are conditions characterized by hypoxia.

Placental perfusion is deficient in hypertensive pregnancies leading to growth restriction. Delivery may be induced in severe early-onset preeclampsia before term and the fetus is premature. Growth restricted babies and preterm babies have a higher possibility of suffering birth asphyxia, intrapartum death, and need of neonatal intensive care. Close fetal monitoring usually during active labor is mandatory and correct interpretation of CTG (cardiotocography) is important as these neonates may not withstand the placental perfusion alterations during uterine contraction, placental abruption, or seizure.

7.3 Clinical Assessment

The abovementioned underlying pathophysiological mechanisms need thorough clinical evaluation and laboratory assessment. The only obvious clinical sign many a times could be raised blood pressure. Important historical points need to be noted carefully whenever HDP patients

present to the labor room. Careful documentation of their past obstetric history is vital and important. Previous obstetric outcome associated with hypertensive disorder should be carefully inquired into with respect to its time of occurrence, complications, and outcome. Gestational diabetes and obesity, prepregnancy hypertension, and autoimmune diseases such as SLE, APLA, nephropathy, etc. should be investigated. Mothers presenting with seizures in pregnancy should always be primarily considered to have eclampsia, and a differential of cerebral malaria, epilepsy, and other causes should be kept in mind after stabilization with loading dose of magnesium sulfate. Causes of seizures are mentioned in the table below (Table 7.3).

Correct establishment of the gestational age and records of antenatal investigations, scans, and clinical examinations should be done. Sudden or excessive weight gain, gross edema, treatment with antihypertensive medications, blood pressure records, hematocrits, drop in platelet counts, growth restriction, and any pregnancy hemorrhage all should be carefully documented as are associated with HDP. Preeclampsia many a times has nonspecific symptoms, but as a clinical ritual, specific symptoms such as feeling sick, nausea, vomiting, visual disturbances, headaches, epigastric or any abdominal pain, and suddenly risen edema should be asked for.

Table 7.3 Causes of seizures in pregnancy and during delivery

Eclampsia
Cerebral vein thrombosis
Thrombotic thrombocytopenic purpura
Cerebral infarction
Drug and alcohol withdrawal
Hypoglycemia
Infection
Antiphospholipid syndrome

Clinical examination includes quick assessment of blood pressure preferably with a mercury sphygmomanometer in a sitting position or if patient is admitted then in left lateral position is important as the gravid uterus pressing onto the vena cava can cause wrong recording. Muffling of the Korotkoff's sound can be taken as diastolic blood pressure in case the sounds don't disappear due to hyperdynamic circulation. All international guidelines accept systolic blood pressure (SBP) of 140 mm of Hg and diastolic blood pressure (DBP) of 90 mm of Hg as a cutoff to identify hypertension in pregnancy. Severity classification varies in different guidelines, but the FOGSI Gestosis expert group has accepted 150 mm of Hg systolic and 100 mm of Hg diastolic as severe hypertension and when associated with proteinuria to be considered as severe preeclampsia. SBP and DBP values during labor are higher than those observed in the antepartum period. An SBP equal to or higher than 150 mmHg or DBP equal to or higher than 90 mmHg is associated with an increased risk of early postpartum preeclampsia (Table 7.4). The ACOG defines severe preeclampsia as blood pressure of ≥160 mmHg systolic and/or ≥110 mmHg diastolic (on two occasions at least 4 h apart in a rested mother and with proteinuria, fetal growth restriction, and evidence of systemic involvements as mentioned in Table 7.2.

All these parameters should be assessed and looked for in mothers presenting with hypertension. Complications and seizures can occur even in the absence of high blood pressure which now is identified as atypical preeclampsia. Therefore

Table 7.4 ACOG severity classification based on blood pressure along with signs and systemic involvement

Mild to moderate	SBP ≥140–159 and/or DBP ≥90–109 mm of Hg
Severe (any two if present)	SBP ≥160 and/or DBP ≥110 mm of Hg rechecked in a rested patient Proteinuria ≥2+ on dipstick (can be done on 2 samples 4 h apart) or ≥5 g in a 24 h sample Oliguria <500 mL/24 h Headache, scotoma, or other vision issues Pulmonary edema or reduced oxygen saturation, breathlessness Abdominal pain especially in the epigastrium or retrosternal Elevated liver enzymes, evidence of subcapsular hematoma, infarctions reduced platelets

presence of any of the other parameters mentioned above in the absence of hypertension should be identified as preeclampsia. Hyperreflexia should be assessed by brisk knee jerks or ankle clonus and is suggestive of cerebral irritability and needs prophylactic magnesium sulfate to be given. Quick obstetric assessment should be done to assess the size of the uterus, presence of uterine contractions, tenderness with hardened uterus typically a sign of abruption, fetal cardiac activity, and preferably admission CTG which can help in identifying fetal well-being. Cervical dilatation, fetal presentation should also be assessed. Systemic assessment for cardiovascular abnormalities such as valvular heart diseases and respiratory abnormalities such as crepitations and reduced air entry should be assessed.

Oxygen saturation by pulse oximetry is an important risk assessment tool at admission which can help in immediate identification of mothers at risk of complications, and if found to be less than 97%, the mother should be taken care of in obstetric ICU (intensive care unit) or HDU (high dependency unit). Careful evaluation of the cardiovascular system to rule out any preexisting cardiac disease should be done; also the respiratory assessment of the rate and presence of basal crepitations should be done in all mothers presenting to labor wards.

Fundoscopic examination reveals severe arteriolar spasm resulting in corkscrew appearance of the retinal vessels or a beaded pearl necklace like appearance. Other fundoscopic (Table 7.5) features include arteriovenous anomalies, exudates, hemorrhages, and edema [12, 13].

7.4 Laboratory Assessment

All the complications are essentially identified by laboratory assessment as they reflect systemic involvements. The mandatory investigations to be sent are the complete blood count which reveals anemia if hemoglobin is <11 g/dL, hemoconcentration if PCV is more than 40, and thrombocytopenia if platelets are less than 1,00,000/cmm. Additionally liver enzymes and renal function tests are to be done (Table 7.5). In case of a clinical suspected coagulopathy or presence of thrombocytopenia, coagulation profile needs to be sent. Proteinuria by dipstick is reliable enough and more than 1+ is taken as significant.

The American Task Force of 2013 has proposed certain modifications in diagnosis of hypertension taking into account the syndromic nature of preeclampsia. For this they have decided to eliminate presence of proteinuria as essential parameter for diagnosis and have suggested that presence of thrombocytopenia, elevated liver enzymes, pulmonary edema, visual disturbances, and renal insufficiency along with hypertension has to be considered as preeclampsia.

7.5 Fetal Assessment

Fetal assessment clinically should be done by assessing the heart rate of the fetus by stethoscope or electronic handheld Doppler, and the admission CTG assesses oxygenation status of the fetus. CTG documents the fetal heart rate and presence of variability and responses to altered placental perfusion due to uterine contraction. It helps in deciding the route of delivery suitable for better outcome. While interpreting the CTG, it should be remembered that mothers on alpha-methyldopa and/or magnesium sulphate therapy may have reduced baseline variability on CTG and in preterm babies between 32 and 37 weeks the baseline heart rate may be in the range of 150–160 bpm. Presence of accelerations in response to movements and uterine contractions are reassuring. Decelerations and occurrence of sudden bradycardia can be ominous signs and may reflect fetal jeopardy and are to be interpreted in context of labor stage and maternal condition. Bradycardia with sinusoidal pattern and uterine contractions with seesaw pattern are signs of placental abruption which can occur intrapartum in HDP (Fig. 7.2). Sonography helps in correctly estimating the fetal weight and well-being by assessing the amniotic fluid index. Placental location and placental bed assessment for abruption are other valuable information. Color Doppler helps assessment of

Table 7.5 Clinical and laboratory assessment interpretation

Investigation for diagnosis	Values	Significance/inference
Proteinuria	Dipstick: ≥1+ on dipstick on a 24 h urine sample: ≥0.3 g/d protein creatinine ratio: ≥30 mg/mmol	Preeclampsia: proteinuria is due to glomerular endotheliosis
Oxygen saturation: pulse oximetry	SpO_2 < 97%	Immediate risk categorization as a critical patient with possibilities of complications
CBC and blood smear PBS WBCs Platelets	Hemoglobin <11 g/dL is anemia Red cell fragmentation More than 12,000/cmm < 150,000–400,000/(μL) Less than 1,00,000/μL	This can be misleading anemia and can also be due to hemolysis. PCV more than 40 is a sign of hemoconcentration Hemolysis Inflammatory process Thrombocytopenia /HELLP Do coagulation tests
Liver tests	SGOT/AST > 45 IU/L SGPT/ALT >45 IU/L SAP >17–88 (first trim), 25–126 (second trim), 38–229 IU/L (third trim) LDH > 600 U/L Bilirubin>0.1–1.0 mg/dL Albumin <3.5–5 g/dL	Liver cells are damaged or dying, ALT and AST leak into the bloodstream Levels are high due to increased placental production in third trimester Hemolysis and liver dysfunction Liver dysfunction and red cell destruction Albumin is produced in the liver. Other causes also may exist for deficiency
Renal functional tests	Uric acid >6.5 mg/dL Creatinine 0.57–1.10 mg/dL BUN >13 mg/dL	Renal parenchymal disease and placental apoptosis Renal failure Reduced glomerular filtration
Coagulation tests	APTT >9.5–13.8 s PT INR > 9.5–13.8 s Fibrinogen<244–510 (first trim) 291–538 (second trim) 373–619 (third trim) FDP (nonspecific) D-dimer: >1500 μg/L	DIC Liver dysfunction leading to deficient production of clotting factors Exaggerated inflammatory response and endothelial Increased intravascular coagulation increased in fibrinolytic activity DIC
Funduscopic examination 3 stages	Spastic stage: spasm of retinal arterioles Stage of sclerosis: superimposed changes in the vessels Stage of retinopathy: cotton wool spots, microaneurysms, flame shaped and splinter hemorrhages, hard exudates, disc edema, etc.	Vasoconstriction Chronic hypertension Severe hypertension

placental perfusion, and feto-maternal circulation helps in identifying fetal hypoxia. End-diastolic flow in the umbilical artery is examined and if absent (AEDF) or reversed (REDF) is a sign suggesting quick delivery in the fetal interest (Table 7.6).

7.6 Decision to Deliver

HDP patients are either electively chosen for delivery or may present spontaneously. All severe preeclampsia patients are delivered by 34 weeks of gestation or whenever diagnosed after 34 weeks. Mild preeclampsia can be taken till 37 weeks of gestation while gestational hypertension up to 40 weeks but not beyond that duration. Any association of fetal or maternal complication necessitates delivery.

Induction of labor can be considered after stabilizing the maternal condition and assessment of fetal well-being. Dinoprostone gel instillation can be considered in absence of favorable cervix. Remote from term pregnancy, misoprostol intravaginal 25 μg every 3 h can be given with close monitoring and till achieve-

Table 7.6 Indication of delivery in pregnancy with hypertension

Maternal	Fetal
Uncontrolled hypertension	AEDF/REDF on color Doppler assessment
Eclampsia	Variable decelerations
Labor/PROM	Reduced fetal movements and BPP <4
Oliguria	Severe FGR <fifth percentile
Abnormal renal/hepatic tests	AFI <5
Thrombocytopenia	GA ≥34 weeks and severe preeclampsia
Neurological complications	GA ≥37 weeks mild preeclampsia
Pulmonary edema	GA ≥40 weeks in gestational hypertension
Abruption	Intrauterine fetal demise
HELLP	
DIC	

Fig. 7.1 Infusion pump to monitor the delivery of fluids, magnesium sulfate, labetalol, and oxytocics. Also it is convenient to administer loading dose of magnesium sulfate in 100 mL NS safely especially in a prophylactic regimen in severe preeclampsia

ment of cervical change. The uterine contraction is further augmented by concentrated drips of oxytocin (infusion pump (Fig. 7.1) or diluted in 100 mL NS) to avoid fluid overload. Concurrent mechanical dilatation by Foley's catheter or special dilators has been used successfully and helps in reducing the misoprostol dose required for induction of labor. Induction should be aggressive achieving delivery within 24 h. Magnesium sulfate should be started pre-induction and continued 24 h post-delivery. Safe interval of at least 6 h should be maintained before using oxytocin after the last PG (prostaglandin) dose to avoid hyperstimulation and its serious consequences.

7.7 Management of Hypertensive Mother During Labor

7.7.1 Determination of Level of Care

The patients of preeclampsia should be delivered in a multidisciplinary setting. This is necessary to be able to offer immediate help if needed in situations such as respiratory distress, postpartum hemorrhage, seizures, assisted vaginal delivery, and neonatal resuscitation. Hemodynamics change in response to uterine contractions during labor. These changes can further get aggravated by

Table 7.7 Checklist before induction of labor and when a mother of severe hypertension is in labor

• Maternal and fetal condition reassessed (appropriate lab reports, radiology, and CTG/NST)
• Patient and relatives counselled indication/ complications/need for NICU/ICU admission/CS/ blood component therapy
• Informed consent obtained
• Blood grouped cross matched and blood group noted: crossmatch 2 PCVs and keep 4 FFP ready
• Ensure the eclampsia kit and contents
• Equipment: Pulse oximeter/multipara monitor/ suction/crash trolley/emergency tray/EFM
• Ensure PPH kit/fluids (RL, Isolyte E, no 5% dextrose)
• Vaginal delivery/CS delivery/instrumental delivery preparedness
• Neonatal resuscitation preparation
• Inform anesthesiologist/neonatologist/operation theater/blood bank

maternal response to pain. This can lead to increase blood pressure, pulse rate, and increased oxygen consumption. Its good practice to follow a preinduction checklist to ensure optimum care and safety (Table 7.7).

Care offered can be classified in four levels as mentioned below:

- Level 0: Can be managed in a regular obstetric ward. Mild preeclampsia or gestational hypertension with mild FGR and no systematic involvement with normal laboratory parameters.
- Level 1: Mothers at risk of deterioration whose needs can be met on an emergency or critical care team available to help. These are mothers who need additional monitoring after delivery or continuance of magnesium sulfate therapy or ongoing laboratory evaluation but do not need a dependency unit.
- Level 2: Mothers needing close monitoring with a single system failure such as HELLP, thrombocytopenia, or seizures. This can be undertaken in an HDU.
- Level 3: Mothers needing advanced support such as inotropes, respiratory assistance, or have two or more systems involved. This needs a well-equipped critical care unit.

7.7.2 Transferring a Sick Mother

All mothers with hypertension are better delivered in a tertiary care center with well-equipped critical care and neonatal care facility; expert obstetrician, anesthesiologist, and nursing team; and an access to blood bank or component storage facility. All delivery rooms should be well armed to manage immediate obstetric complications such as hemorrhage, seizures, and neonatal resuscitation facility to deliver mothers with hypertension. In case of a severely ill mother, it is better to transfer her to a well-equipped facility. Before transferring, magnesium sulfate loading dose, capsule nifedipine or labetalol, and steroids in case of HELLP syndrome and prematurity should be administered. It is a good practice to refer the patient with all the necessary clinical and medication details and inform the center in advance for prompt and better care.

7.7.3 First Stage of Labor

During first stage of labor when uterine contractions are gradually increasing in intensity and duration, careful vigilance to monitor the mother and the fetus is essential. Partograph gives an instant comparable overview of progress of delivery and should be documented every half an hour. Continuous fetal monitoring is a good practice point, but intermittent frequent monitoring with Doppler/stethoscope too is sufficient. Maternal monitoring during labor includes half-hourly assessment of pulse, and temperature and respiratory rate should be monitored and documented. Tachycardia can imply abruption of placenta and should be looked for during the first stage of labor (Fig. 7.2).

7.7.4 Vigilance for Placental Abruption

Contractions of longer duration and higher intensity with tonic hard uterus or sudden gush of bleeding per-vaginal or sudden tachycardia, bradycardia, or decelerations on the CTG trace are important signs of placental abruption [2]. Amniotomy (artificial rupture of membranes,

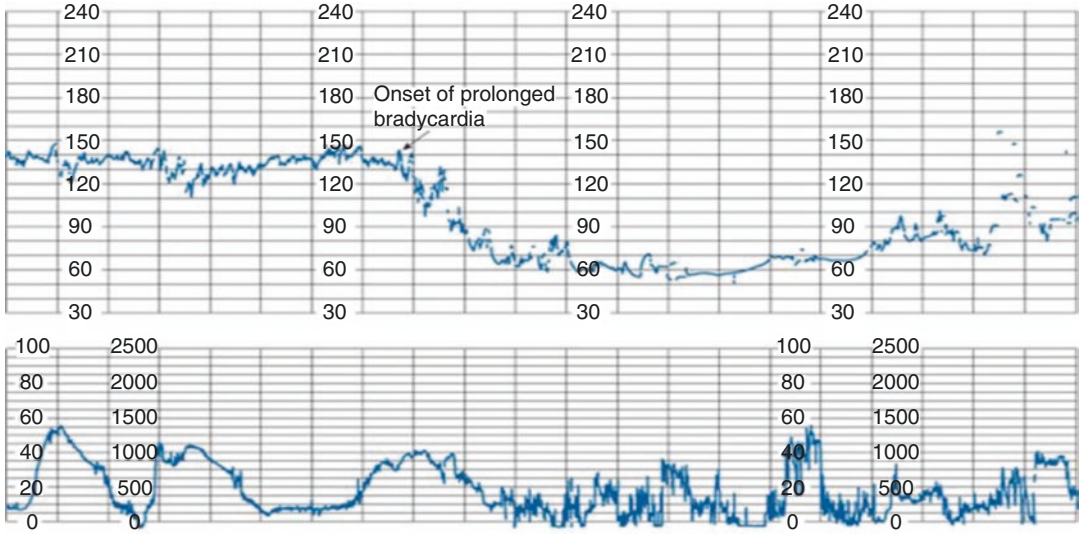


Fig. 7.2 CTG demonstrating reduced baseline variability and onset of prolonged bradycardia due to abruption in a 36 weeks 5 days gestation gravida 2 para 0 mother with severe preeclampsia

ARM) should be done immediately to reduce the intrauterine pressure which aids in reducing the stretch on the retroplacental space and release of thromboplastin in circulation which may initiate the cascade of DIC. In addition it may reduce the possibility or severity of couvelaire uterus which occurs due to dissipating blood causing myometrial dissection which can result in atonic uterus after delivery or necessitate obstetric hysterectomy for intractable bleeding. Quick assessment of this condition prophylactic fluids (but rationally to avoid pulmonary edema), fresh frozen plasma, and delivery within 6–8 h of occurrence of placental abruption can prevent morbidity and mortality.

7.7.5 Seizure Control and Blood Pressure Stabilization

Seizure prevention and control and hypertension stabilization are important measures to be undertaken. During labor blood pressure is known to rise in apparently normotensive mothers. This can further be exaggerated in hypertensive subjects. This blood pressure rise has been attributed to increased cardiac output due to increased blood pushed through the uterine sinuses with increasing contractions and labor pains. In normotensive women, rise in blood pressure during labor is considered as a strong predictor of severe postpartum preeclampsia especially if it is recorded to be more than 150/100 mm of Hg [14]. Close blood pressure monitoring and stepping up of dosages may be needed. Nifedipine and labetalol are found to be useful antihypertensive medications with established safety, efficacy, and fast duration of action. Hypertensive emergency is a hallmark of HDP intranatally as well as postnatally and is characterized by sudden onset of severe hypertension of 160/110 mm Hg or more which lasts or heightens for more than 15 min. Risk of intracerebral injury in the form of hemorrhage or infarct is associated. Severity of SBP is a predictor of such an occurrence than the DBP. Martin et al. have studied patients suffering from stroke and observed that 54% of these patients had SBP of more than 160 mm Hg while 13% had a rise in DBP equal or more than 110 mm Hg [15]. Nonpregnant women are also at risk of stroke with SBP of 160 mmHg or more. Thus 160 mmHg SBP is the threshold of the cerebral circulation autoregulation and when failed results in hemorrhage or seizures. Blood pressure restoration should be in the range of 140–150 mm Hg SBP and 90–100 mm Hg DBP in order to maintain the placental, cerebrovascular, cardiac, and renal perfusion and can help reverse part of the pathology initiated. But the risk of stroke may still prevail after the BP is lowered.

The blood pressure should be lowered promptly but gradually as sudden drop may adversely affect the mother as well as the baby.

7.7.5.1 Hypertensive Crisis

In a crisis situation, the blood pressure should be lowered gradually by 20–30% primarily and then gradually reached to the goal of 140/90 mmHg. Nifedipine is a calcium channel blocker and has fast onset of action and should always be given orally and never sublingually. Sublingual administration leads to erratic absorption and sudden drop in blood pressure which is harmful both to the mother and the fetus. Oral administration is possible only in women who are conscious, and this is its limitation. Onset of action is 15–20 min, and the dose has to be monitored with continuous titration of blood pressure record every 15mins till the requisite control is achieved. Starting with 10 mg nifedipine, further 5–10 mg can be administered based on the response. Up to 40–60 mg can be given in 24 h and 40 mg within the first 1 h. Once stabilized the nifedipine can be administered 8–12 h, and it is better to use the slow release formulation for the same. Nifedipine is contraindicated in the presence of tachycardia and has been identified as a useful antihypertensive even in the settings of severe hypertension.

Mothers, who are unconscious, have tachycardia and are refractory to nifedipine; parenteral labetalol should be used. Labetalol can also be a primary drug of choice in hypertensive crisis. It is a beta-adrenergic antagonist is cardioprotective due to its anti-alpha activity and therefore is a good choice. It is better than pure beta-blockers due to its effects on systemic vascular resistance (decreased leading to vasodilation), afterload (reduced), cardiac contractility (reduced), and heart rate (reduced), and it optimally maintains cardiac output. It is contraindicated in congestive cardiac failure, asthma, diabetes mellitus, and bradycardia (both maternal and fetal). Labetalol is administered intravenously either as small aliquots. The dose is 10–20 mg intravenously, followed by 20–80 mg every 20–30 min with a maximum of 300 mg (infusion pump administration is ideal) (Fig. 7.1) which delivers 1–2 mg/min dose. Duration of action is 3–6 h. Advantage of this drug is that it is safe, easy to administer, and fast acting and does not cause sudden hypotension. Hydralazine is currently available in India and is also used by many workers effectively. Hydralazine is a rapid-acting antihypertensive that can be given in low-dose increments. It acts by achieving smooth muscle relaxation in the arterioles leading to peripheral vasodilation without reducing uteroplacental perfusion.

7.7.6 Fluid Management

Fluid infusion in preeclampsia requires intricate balance in maintaining perfusion and preventing shock and fluid overload. Endothelial dysfunction leads to increased capillary permeability and increased capillary wedge pressure which is typically understood as “capillary leaky syndrome.” Excessive fluids can lead to pulmonary edema and maternal death. Fluids should be restricted and administered at the rate of 80 mL/h or 1 ml/kg/h [16]. This has to be titrated against urinary output, and 700 mL can be added in 24 h for nonsensical losses. Fluid expansion should not be done, and crystalloids are better with Ringer’s lactate as the first choice.

7.7.7 Oxytocics for Augmentation

Labor may have to be augmented to expedite the delivery or to overcome uterine inertia due to generalized hypoxia and magnesium sulfate. Oxytocin only and no prostaglandin should be used for augmentation. Prostaglandin should be stopped once cervical changes occur. Concentrated drips of oxytocin and delivery through an infusion pump (Fig. 7.1) are good practice points to avoid fluid overload and optimum delivery of oxytocin. An interval of at least 6 h should be maintained between the last dose of prostaglandin and the initiation of oxytocin.

7.7.8 Second Stage of Labor

Continuous maternal and fetal monitoring is necessary during the second stage of labor. Reassess the maternal condition for hypertension and complications of hypertension such as pulmonary edema,

altered sensorium, and evidence of abruption of the placenta. Oliguria may be temporary and should not provoke increased intravenous infusion. Controlled intravenous fluids, antihypertensive medications, and magnesium sulfate if necessary should be administered. Increase intrauterine pressure and preload on the heart can precipitate a seizure or intracranial accident due to sudden rise in blood pressure. Recheck the availability of instrumental delivery equipment and neonatal resuscitation facilities. Continuous electronic fetal monitoring is desired if possible. Fetal heart assessment after every uterine contraction is equally validated and should be done. Assisted vaginal delivery may be essential to shorten the second stage. Possibilities of hematomas should be kept in mind in cases of compromised coagulation profile.

7.7.9 Third Stage of Labor

Active management of third stage of labor to be practiced with the use of oxytocin 10 IU administered intramuscularly after the delivery of the baby. In case of thrombocytopenia, intravenous oxytocin diluted can be administered. Injectable prostaglandin can be used, but intramuscular injection can cause hematoma in low platelets. Prophylactic misoprostol 600 μg can be administered by the rectal route effectively. Women with hypertension have less intravascular fluid volume and therefore may have hemodynamic jeopardy even with a smaller amount of blood loss, and therefore close monitoring is essential. Thrombocytopenia can contribute to further blood loss. Careful examination for genital tract injuries, hematomas, and uterine retraction should be made and the mother carefully monitored in the labor room for at least 2 h post-delivery. Injection methylergometrine is contraindicated as it causes sudden hypertension leading to intracranial accidents, myocardial infarctions, and cardiac arrhythmias.

7.8 Blood and Blood Components

It's a good practice to reserve two packed cell volumes and notify the blood bank for need of blood products in case of hemorrhage or DIC. In case of thrombocytopenia, prophylactic fresh frozen plasma may be administered to avoid bleeding and DIC. Blood loss if any to be promptly replaced by packed cell volumes. Platelet transfusion is to be used if platelet counts are less than 20,000 per mm. Coagulation assessment whenever necessary must be undertaken and component therapy planned in accordance.

7.9 Labor Analgesia and Positioning

Epidural analgesia has many benefits to offer along with pain relief, improved placental perfusion, and ease of conversion to anesthesia if cesarean section, assisted vaginal delivery, or exploration of the genital tract becomes necessary. Narcotics can be administered for the same but may alter the mother's sensorium. Left lateral or propped-up position is preferred to avoid the gravid uterus compressing on the inferior vena cava.

7.10 Fourth Stage of Labor

Monitoring of the vital parameters continue after delivery as there is always a risk of postpartum eclampsia, postpartum hemorrhage, or fear of sudden collapse. Vigilance continues, and if necessary antihypertensive medications are administered. Look for adequate urinary output, control of per-vaginal bleeding, and retracted uterus. Continue the magnesium sulfate regimen if already started for 24 h post-delivery or initiate anew if rise in blood pressure is observed.

7.11 Eclampsia Kit

Always update the eclampsia kit in the delivery rooms mentioned under. This is sourced from the FOGSI ICOG National Eclampsia Registry (Fig. 7.3).

The National Eclampsia Registry: FOGSI—ICOG—The Eclampsia Kit

Sr.no	Item	Specifications	No	Additional/tick
1.	Mouth gag	Disposable if possible	1	
2.	IV set	Infusion pump (Fig. 7.1) preferred	2	
3.	Intracath	No 20 /21	2	
4.	Scalp	No 18/20	2	
5.	Three-way	Preferred	2	
6.	Syringes	20 cc/10 cc/5 cc	2/5/5	
7.	Uribag	Urometer preferred	1	
8.	Inj MgSO4	50/25%	20/20	
9.	Cap nifedipine	5 mg/10 mg/20 mg/20R	5/5	
10.	Inj labetalol		2	
11	Hammer		1	
12.	Torch	–	1	
13.	Foley's catheter	No 16/18		
14.	Uristicks	Protein estimation	2	
15	Needles	20 G: 2 in./11/2in.	10	
16	Ryle's tube	14	1	
17	Bulbs	Plain/Hemogram/platelet/prothrombin/fluoride bulbs/EDTA bulb	1 each	
18	Inj calcium gluconate	10 mL 10%	2	
19	Inj distilled water	5 mL/10 mL/20 mL	10/10/10	
20	Intravenous fluids	RL/Isolyte E /DNS	2/2/2	
21	Misoprostol tablets	25 mcg /50 mcg/100/200	4/4/4/4	
22	Oxytocin ampoules	5 IU	25	
23	Inj PGF2α	250mcg	2	
24	Inj Phenergan	50	2	
25	Eclampsia management protocol	1 copy displayed		
26	Emergency drugs tray	Labeled and updated		

Fig. 7.3 Well-equipped eclampsia room, newborn corner, and the always ready loading dose magnesium sulfate

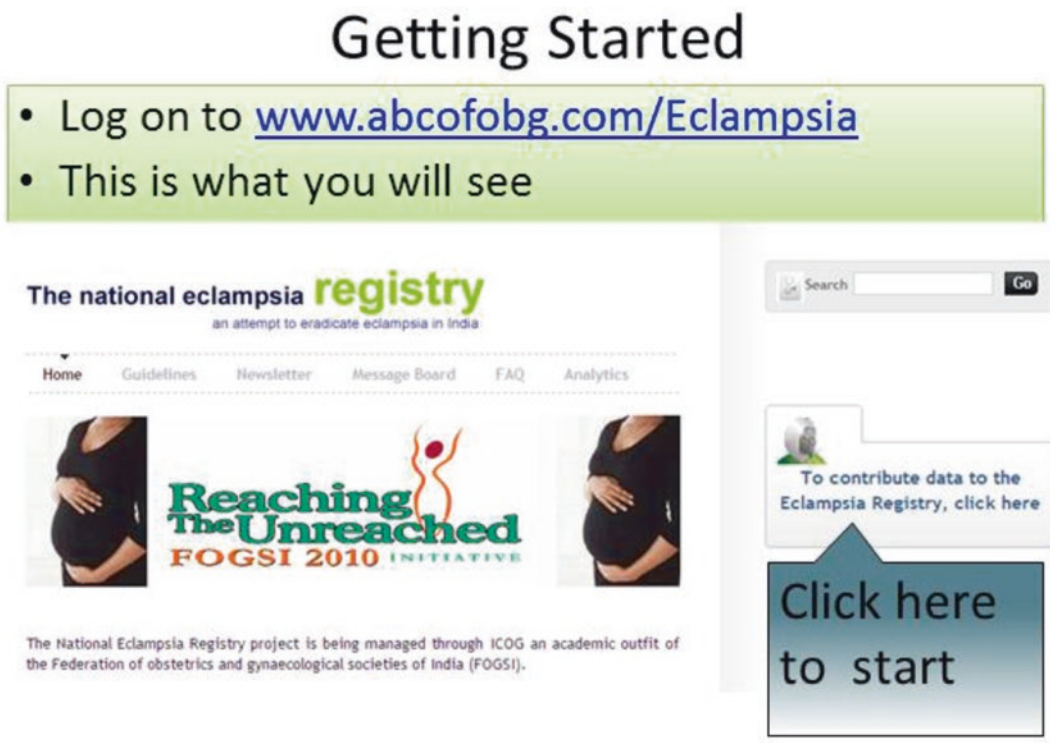


Fig. 7.4 NER FOGSI ICOG

7.12 Conclusions

Delivery of hypertensive mothers need multidisciplinary approach and tertiary level care. Vigilance and proper decision-making are essential. Dynamics of labor may precipitate complications of preeclampsia, and these should be prevented, detected early, and treated promptly. Zero tolerance for eclampsia (enroll as reporter at the National Eclampsia Registry: Fig. 7.4) should be the dictum, and therefore magnesium sulfate should be used liberally.

References

1. Report of the National High Blood Pressure Education Program Working Group. Report on high blood pressure in pregnancy. Am J Obstet Gynecol. 2000;183:S1–22.
2. Sibai BM. Diagnosis and management of gestational hypertension and preeclampsia. Obstet Gynecol. 2003;102:181–92.
3. Cretti A. EPH gestosis or hypertension induced by pregnancy? Ginekol Pol. 1992 Jun;63(6):308–11.
4. Sibai BM. Diagnosis, differential diagnosis and management of eclampsia. Obstet Gynecol. 2005;105:402–10.
5. Hazra S, Waugh J, Bosio P. "Pure" preeclampsia before 20 weeks of gestation: unique entity. BJOG. 2003;110:1034–531.
6. Cnossen JS, Morris RK, ter Riet G, Mol BW, van der Post JA, Coomarasamy A, Zwinderman AH, Robson SC, Bindels PJ, Kleijnen J, Khan KS. Use of uterine artery Doppler ultrasonography to predict pre-eclampsia and intrauterine growth restriction: a systematic review and bivariable meta-analysis. CMAJ. 2008;178(6):701–11.
7. Melchiorre K, Sharma R, Thilaganathan B. Cardiac structure and function in normal pregnancy. Curr Opin Obstet Gynecol. 2012;24(6):413–21.
8. Solanki R, Maitra N. Echocardiographic assessment of cardiovascular hemodynamics in preeclampsia. J Obstet Gynaecol India. 2011;61(5):519–22.
9. Melchiorre K, Sutherland GR, Baltabaeva A, Liberati M, Thilaganathan B. Maternal cardiac dysfunction and remodeling in women with preeclampsia at term. Hypertension. 2011;57(1):85–93.
10. Vasapollo B, Novelli GP, Valensise H. Total vascular resistance and left ventricular morphology as screening tools for complications in pregnancy. Hypertension. 2008;51(4):1020–6.
11. Gyselaers W, Mesens T, Tomsin K, Molenberghs G, Peeters L. Maternal renal interlobar vein impedance index is higher in early- than in late-

onset pre-eclampsia. Ultrasound Obstet Gynecol. 2010;36(1):69–75.
12. Saito Y, Tano Y. Retinal pigment epithelial lesions associated with choroidal ischemia in preeclampsia. Retina. 1998;18:103–8.
13. Ryan SJ, Sunness JP. Pregnancy and retinal disease. In: Ryan SJ, editor. Retina. vol. 2; 1994. p. 1393–403.
14. Cohen J, Vaiman D, Sibai B, Haddad B. Blood pressure changes during the first stage of labor and for the prediction of early postpartum preeclampsia: a prospective study. Eur Obstetr Gynecol Reprod Biol. 2015;184:103–7.
15. Martin JN Jr, Thigpen BD, Moore RC, et al. Stroke and severe preeclampsia and eclampsia: A paradigm shift focusing on systolic blood pressure. Obstet Gynecol. 2005;105:246.
16. National Collaborating Centre for Women's and Children's Health (UK). Hypertension in pregnancy: the management of hypertensive disorders during pregnancy. London: RCOG Press; 2010. NICE Clinical Guidelines, No. 107

Heart Disease

8

Vanita Suri and Pooja Sikka

8.1 Introduction

Cardiac disease complicating pregnancy is seen in about 1–4% women. Due to effective treatment during childhood, the incidence of women with heart disease presenting during pregnancy is increasing. In developing countries like India, rheumatic heart disease still predominates, accounting for 60–80% of cardiac lesions in pregnancy. Congenital heart diseases are by far the commonest conditions in the west forming over 75% of heart diseases seen in pregnancy [1]. Pregnancy poses additional problem in women with underlying cardiac disease. A normal heart can adapt well to the hemodynamic alterations of pregnancy, whereas a diseased heart may not be able to do so and decompensate leading to heart failure. Ideally counseling and management of women of childbearing age with cardiac disease should start before pregnancy occurs. High-risk patients should be managed in specialized centers where multidisciplinary care is available. Diagnostic procedures and interventions should ideally be performed prior to pregnancy. Unfortunately in India, many women still are diagnosed to be having cardiac disease for the first time during pregnancy. This increases the morbidity and mortality associated with these disorders.

V. Suri (✉) · P. Sikka
Department of Obstetrics and Gynecology, PGIMER, Chandigarh, India

8.2 Hemodynamic Alterations During Pregnancy

Pregnancy induces many changes in the cardiovascular system of the woman [1]. It is important to understand physiological changes in the cardiovascular system which occur during pregnancy and puerperium in order to be able to manage the patient during pregnancy and labor.

This includes increase in blood volume and cardiac output (CO) and reductions in systemic vascular resistance and blood pressure (BP). A 30–50% increase in CO occurs in normal pregnancy. It has been shown in various studies that the cardiac output rises early in first trimester and there is further rise during second trimester. In the third trimester, the cardiac output may rise, fall, or plateau [2]. In the postpartum period, there is rapid fall of cardiac output, and the maximum decrease is in the first 2 weeks after delivery [3].

Heart rate starts rising at 20 weeks and peaks at 32 weeks, there after it plateaus and falls to normal within 1 week of delivery. Plasma volume reaches a maximum of 40% above baseline at 24 weeks gestation. Blood pressure falls during first and second trimester due to vasodilatation. The diastolic blood pressure may rise in third trimester and normalizes during early postpartum period.

Pregnancy leads to increase in concentration of fibrinogen, platelet adhesiveness, and other coagulation factors, thus increasing the risk of

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thromboembolism in pregnancy. These physiological adaptations influence the evaluation and interpretation of cardiac function and clinical status.

There are additional changes in the hemodynamics during labor and postpartum period due to positioning of patient, pain, anxiety, and other complications of labor. Choice of analgesia and anesthesia is limited by these changes. SBP and DBP also increase, during uterine contractions. CO increases by 15% in early labor, by 25% during stage 1, and by 50% during expulsive effort [4]**.** These changes become more rapid and abrupt during labor and delivery. Major threat to the pregnant lady is during labor and delivery. Labor produces rapid and severe hemodynamic changes. Thus labor may become very complex, so these women should preferably be delivered in an institute where multidisciplinary care is available. The team should comprise of obstetrician trained in managing these cases, anesthetist, cardiologist, and neonatologist.

8.3 Diagnosis of Heart Disease

History of dyspnea is important in identifying the clinical status. As a general rule, asymptomatic patients will have a good pregnancy outcome. Many of the normal symptoms of pregnancy, such as dyspnea on exertion, orthopnea, palpitations, giddiness, and ankle edema, are also symptoms of cardiac decompensation. However, chest pain, dyspnea at rest, and paroxysmal nocturnal dyspnea are not commonly seen with pregnancy, and patient should be evaluated for heart disease [5]. However relatively asymptomatic patients with conditions like primary pulmonary hypertension, Eisenmenger's syndrome, or stenotic lesions of valves may have an acute deterioration also in the form of varied symptoms. On examination, patient may have jugular venous distension, prominent apical impulses, and presence of third heart sound and murmurs. Diastolic murmurs are rare in normal pregnancy [6]. Detailed physical and cardiovascular examination is needed in case of systolic murmurs of more than 2/6 intensity or continuous murmurs. Presence of heart failure or cyanosis suggests a high-risk pregnancy. ECG and chest x-ray complement the diagnosis and can diagnose complications like heart blocks and heart failure. Echocardiography forms the mainstay of diagnosis and should be carried out in all pregnant women with suspected heart disease. Cardiac catheterization is usually avoided in pregnant women due to the risks associated with radiation exposure. However it may be carried out in patients with suspected coronary artery disease and as part of therapeutic procedures like balloon mitral valvotomy.

8.4 Prepregnancy Counseling

Prepregnancy counseling should be carried out by a joint obstetrics and cardiology team. The maternal risk depends on the underlying condition. According to the Task Force recommendations, maternal risk assessment should be carried out according to the modified World Health Organization (WHO) risk classification [7]. This risk classification is based on the underlying heart disease and any other comorbidity. It also includes contraindications for pregnancy where medical termination of pregnancy is advised. CARPREG and ZAHARA risk scores/predictors are also used by many physicians for predicting maternal cardiovascular complications during pregnancy and neonatal outcome [8, 9]. Conditions that are very high risk include primary pulmonary hypertension; Eisenmenger's syndrome; dilated cardiomyopathy with left ventricular ejection fraction <40%; symptomatic obstructive lesions like aortic stenosis, mitral stenosis, pulmonary stenosis, and coarctation of the aorta; Marfan syndrome with aortic root diameter >40 mm; cyanotic lesions; and women with mechanical prosthetic valves [1]. In contrast, patients with regurgitant lesions like mitral and aortic regurgitation who have normal left ventricular functions, left to right shunt lesions like atrial and ventricular septal defects, hypertrophic cardiomyopathy, surgically corrected congenital heart disease, and mild stenotic lesions have relatively uncomplicated pregnancies [1, 10–15]. Diseases affecting the aorta like Takayasu arteritis

are usually well tolerated unless there is severe aortic obstruction, coronary involvement, or aortic regurgitation [16]. Patients with heart block may need pacemaker implantation. In patients who already have an implanted pacemaker, interrogation of pacemaker should be carried out by the cardiologist to confirm the parameters [17]. Girls with congenital heart disease should be referred to a joint cardiac/obstetric/gynecological clinic for advice about contraception and need for preconception counseling once they decide to plan pregnancy [18].

8.5 Risk Classification in Pregnancy and Heart Disease

Various criteria for risk stratification have been described. Most commonly used is the modified World Health Organization (WHO) risk classification [7, 19]. This risk classification includes all maternal cardiovascular lesions. According to this, very low-risk patients fall in WHO class I and need only 1–2 cardiology visits during pregnancy. Low- and moderate-risk patients fall in WHO class II and cardiology consultation should be carried out in each trimester. High-risk women fall in WHO class III. Monthly or bimonthly cardiology visits are recommended. Women in WHO class IV are at very high risk and should be advised against pregnancy. In case they present early in pregnancy, medical termination of pregnancy should be advised.

The conditions which are considered high risk for the mother and fetus are mitral stenosis with NYHA class II, III, or IV symptoms; mitral regurgitation with NYHA class III or IV symptoms; severe aortic stenosis with or without symptoms; aortic valve disease, mitral valve disease, or both resulting in pulmonary hypertension; aortic valve disease, mitral valve disease, or both with left ventricular ejection fraction less than 40%; Eisenmenger's syndrome; Marfan syndrome; primary pulmonary hypertension and maternal cyanosis.

Asymptomatic aortic stenosis; aortic regurgitation with NYHA class I or II symptoms and normal left ventricular systolic function; mitral regurgitation with NYHA class I or II symptoms and normal left ventricular systolic function; mitral valve prolapsed, with no regurgitation or with mild-to-moderate regurgitation and normal left ventricular systolic function; mild-to-moderate mitral stenosis; and mild-to-moderate pulmonary valve stenosis are better tolerated during pregnancy and are considered low risk [1].

8.6 Antenatal Care

Antenatal care should involve a multidisciplinary team including senior obstetrician, cardiologist, and anesthetist. High-risk patients should be identified by using different risk scores (WHO score is recommended) and all aspects of their management including optimization of drug therapy, anticoagulation, timing and mode of delivery, and use of analgesia during labor should be decided before she goes into labor. Every antenatal checkup should include a detailed maternal obstetric, cardiovascular, and fetal assessment. A plan of management for mode of delivery, timing of delivery, and intrapartum care including need for invasive monitoring, cutting short second stage of labor, and need of oxytocics should be made at 34–36 weeks of pregnancy. The plan should also include postpartum care including need for thrombosis prophylaxis wherever required [18].

8.7 Timing and Mode of Delivery

Labor may precipitate decompensation in any type of heart lesion. There is a standard cardiac care which must be provided to all patients. Generally the mode of delivery is based on obstetrical indications only. However there are a few indications for elective cesarean in women with heart disease. These are aortic root diameter >4.5 cm, severe aortic stenosis, aortic dissection, and recent myocardial infarction [1]. Timing of delivery depends upon primary heart lesion, associated comorbid conditions, and other complications of pregnancy. Induction of labor is not

contraindicated. Use of PGE2, mechanical dilators, and PGE1 is not contraindicated but should be used with caution. We prefer to plan the delivery at around 39 weeks in uncomplicated pregnancies in order to ensure optimum perinatal and maternal outcome.

8.8 Care During Labor

Maintenance of hemodynamic stability should be the main aim. Each cardiac condition needs specific considerations to achieve this aim.

General principles: Propped-up position and oxygen supplementation help in women who are breathless. Fluid intake should be restricted and should not be more than 70 mL/h. Pain relief during labor is essential to prevent tachycardia, and drugs like morphine and tramadol can be administered. Epidural analgesia can be provided with special care to prevent hypotension.

Artificial rupture of membranes should be avoided to augment labor. Infective endocarditis prophylaxis is not recommended for any genitourinary procedure anymore. However, when endocarditis occurs during pregnancy, maternal and fetal mortality rates are 22 and 25%, and variable incidence of bacteremia has been reported by various authors. The incidence can vary from 5 to 19% [20–22].

The ACC/AHA guidelines recommend against prophylaxis in cesarean and vaginal deliveries, but due to paucity of data from India and as such high incidence of infection, individualized decision should be taken by the consultant in charge after assessing the need for it. However it is recommended during vaginal delivery/cesarean in women with prosthetic heart valves and cyanotic heart disease and in those with previous history of infective endocarditis [23, 24]. The cost of treatment, morbidity, and mortality of infective endocarditis to the patient is so high that routinely giving prophylaxis to every woman in labor is not that unjustified especially in our settings. Prophylaxis consists of antibiotics using the AHA guidelines of ampicillin 2.0 g IM or IV plus gentamicin 1.5 mg/kg (not to exceed 120 mg) given at initiation of labor or within 30 min of a cesarean delivery, followed by ampicillin 1 g IM or IV or amoxicillin 1 g orally 6 h later. Vancomycin 1.0 g IV over 1–2 h is recommended for penicillin-sensitive patients.

Second stage of labor puts additional stress on mother's heart, so bearing down efforts should be avoided, and instrumental delivery to cut down second stage of labor is recommended. Active management of third stage of labor should be done. Ergotamine is contraindicated as it produces severe peripheral vasoconstriction. Oxytocin 10 U IM or 25 U in 500 mL of normal saline can be given. Blood pressure, pulse rate, and oxygen saturation should be continuously monitored. Chest auscultation for crepitations should be frequently carried out. This gives a quick idea of deterioration of cardiac status, blood loss, and overzealous use of diuretic and oxytocin.

8.9 Postpartum Care

Low-dose oxytocin infusion (10 U in 500 mL of normal saline) that avoids hypotension should be administered after placental delivery to prevent hemorrhage. Prostaglandin F analogues are useful to treat postpartum hemorrhage, unless an increase in pulmonary artery pressure is undesirable. Early ambulation and elastic stockings reduce the risk of deep vein thrombosis. Heart failure can develop in the first day after delivery due to rapid fluid shifts and hemodynamic stresses. Close monitoring should be continued for at least 24 h after delivery.

8.10 Special Situations

Mitral stenosis: Fluid overload should be prevented in mitral stenosis. Regular monitoring of respiratory rate, auscultation of chest, use of concentrated, titrated doses of Pitocin, and maintaining an input-output record are essential. Tachycardia can precipitate pulmonary edema and atrial fibrillation. Diuretic administration after delivery of the baby reduces the excess preload to the left atrium which it cannot handle in presence of mitral stenosis [10, 15]. In symptomatic patients with severe mitral stenosis, balloon mitral valvotomy or closed mitral valvotomy provides

immediate relief [25]. It is however associated with risk of precipitating preterm labor.

Aortic and mitral regurgitant lesions: They are generally well tolerated and may not require aggressive monitoring unless there is left ventricular dysfunction or pulmonary hypertension.

Congenital heart disease: Patients with severe aortic stenosis, primary pulmonary hypertension, Eisenmenger's syndrome, cyanotic congenital heart diseases like tetralogy of Fallot, and Ebstein's anomaly are at high risk and should be closely monitored [26]. The main aim during labor is to prevent hypotension. Maternal and perinatal outcome is better in patients who have been successfully operated in childhood [13].**Prosthetic heart valves**: Patients with mechanical prosthetic heart valves are at risk for complications like valve thrombosis, thromboembolism, and bleeding due to anticoagulation. Bioprosthetic valves on the other hand are associated with the risk of valve failure during pregnancy. Both types of valves lead to increased risk of endocarditis. The major issue with prosthetic valves in pregnant women is the risk of thrombosis as pregnancy is a hypercoagulable state. Therefore pregnant women with prosthetic heart valves need careful planning and counseling about anticoagulant usage. The preferred treatment for adequate anticoagulation is in the form of vitamin K antagonists (e.g., warfarin) which are associated with risk of warfarin embryopathy when used in the first trimester. A reasonable option is to use unfractionated heparin/low molecular weight heparin in the first trimester and then switch over to warfarin till the 36th week [1, 27–29]. Warfarin should be switched back again to unfractionated heparin from the 36th week since the anticoagulant effect of heparin can be rapidly reversed. Unfractionated heparin should be discontinued 4–6 h before planned delivery and restarted 4–6 h after delivery if there are no bleeding complications. If urgent delivery is needed for a patient on unfractionated heparin, protamine may be used to reverse the anticoagulant effect. If urgent delivery is needed in a patient who is on warfarin; cesarean delivery is preferred to reduce risk of intracranial hemorrhage in an anticoagulated fetus. Fresh frozen plasma may be used prior to cesarean delivery to achieve a target INR of $\leq$2.4. The guidelines recommend use of low molecular weight heparin also instead of unfractionated heparin. With the use of low molecular weight heparin, it is mandatory to measure anti-factor Xa activity [1]. If this investigation is not available in our setting, low molecular weight heparin should not be used. In patients who develop stuck valve doe to thrombus formation, thrombolysis is a reasonable alternative to redo valve surgery [30].

Cardiomyopathies: Cardiomyopathies, though rare disorders, commonly affect young people and are thus encountered in pregnancy. Of these dilated, peripartum and restrictive cardiomyopathies may cause severe complications in pregnancy, while hypertrophic cardiomyopathy is usually well tolerated even in the presence of left ventricular outflow tract obstruction [14, 31, 32]. Peripartum cardiomyopathy is a unique cardiomyopathy that usually occurs in the last month of pregnancy or the early postpartum period. The mainstay of treatment of dilated and peripartum cardiomyopathies is drug therapy with beta-blockers, angiotensin-converting enzyme inhibitors, aldosterone antagonists, and loop diuretics. Out of these angiotensin-converting enzyme inhibitors and aldosterone antagonists are contraindicated during pregnancy and can only be started postpartum. In case of acute deterioration, these patients have to be managed on lines of acute heart failure with propped-up position, oxygen, loop diuretics, digoxin, inotropes in case of hypotension or low cardiac output, and in severe cases mechanical supportive therapy [15].

8.11 Contraception

Care of women with heart disease is incomplete without providing adequate contraception advice. Risks of contraceptive use should be weighed against the risk of pregnancy. Barrier contraceptive is the safest for the woman but is associated with high risk of failure. A copper-containing intrauterine device can be inserted either postplacentally or after 6 weeks. Under aseptic precautions, the risk of infective endocarditis is very low [33]. Risks of excessive bleeding during menstruation should be explained especially to women on anticoagulants [1].

Levonorgestrel releasing intrauterine device is the safest and most effective contraceptive in women with complex heart lesions including cyanotic congenital heart disease and pulmonary hypertension. Low-dose oral contraceptives containing 20 mg of ethinyl estradiol are safe in women with a low thrombotic risk, but not in women at high risk for thrombotic complications, and generally they should be avoided. Monthly injectables containing medroxyprogesterone acetate should not be used in women with heart failure. This is due to fluid retention that they may cause. Tubal ligation is usually safe, even in relatively high-risk women. Vasectomy should be discussed with patients who have completed their family [34].

8.12 Conclusion

Pregnant women with heart disease pose challenges in cardiac and maternal-fetal management. Successful pregnancies can be achieved with good prenatal counseling, adequate antenatal care, and intensive monitoring during labor by cardio-obstetric team. Close collaboration between the obstetrician and cardiologist is required for optimal management of women with heart disease.

8.13 Points to Ponder

Box 1: Care During Pregnancy

- Refer the patient to an institute where cardiology services are available.
- Supervision by obstetrician and cardiologist jointly as a team.
- Early decision for MTP (if indicated).
- Change of drugs to safer alternatives.
- Regular at least two weekly visits in antenatal clinic (medical surgical unit).
- Admission in case of any cardiac indication or pregnancy complication.
- Low threshold for admission.
- Make a plan in third trimester for timing and mode of delivery.
- Anesthesia consultation for labor analgesia and anesthesia in case of cesarean section.

Box 2: Care During Labor

- Planned delivery in high-risk cases.
- Nursing in propped-up position.
- Oxygen inhalation (if indicated).
- Fluid restriction.
- Infective endocarditis prophylaxis (where indicated).
- Liberal analgesia.
- Monitoring of pulse, BP, and RR.
- Cut short second stage with outlet forceps/ventouse.
- Avoid ergometrine.
- Active management of third stage.
- Diuretics after delivery of the placenta.
- Frequent monitoring in the postpartum period.

References

1. European Society of Gynecology (ESG); Association for European Paediatric Cardiology (AEPC); German Society for Gender Medicine (DGesGM), Regitz-Zagrosek V, Blomstrom Lundqvist C, Borghi C, Cifkova R, Ferreira R, Foidart JM, Gibbs JS, Gohlke-Baerwolf C, Gorenek B, Iung B, Kirby M, Maas AH, Morais J, Nihoyannopoulos P, Pieper PG, Presbitero P, Roos-Hesselink JW, Schaufelberger M, Seeland U, Torracca L, ESC Committee for Practice Guidelines. ESC guidelines on the management of cardiovascular diseases during pregnancy: the task force on the Management of Cardiovascular Diseases during pregnancy of the European Society of Cardiology (ESC). Eur Heart J. 2011;32:3147–97.
2. van Oppen AC, Stigter RH, Bruinse HW. Cardiac output in normal pregnancy: a critical review. Obstet Gynecol. 1996;87(2):310–8.
3. Robson SC, Hunter S, Moore M, et al. Hemodynamic changes during the puerperium: a Doppler and M-mode echocardiographic study. Br J Obstet Gynaecol. 1987;94(11):1028–39.
4. Robson SC, Dunlop W, Moore M, Hunter S. Combined Doppler and echocardiographic measurement of cardiac output: theory and application in pregnancy. Br J Obstet Gynaecol. 1987;94:1014–27.
5. Stout KK, Otto CM. Pregnancy in women with valvular heart disease. Heart. 2007;93(5):552–8.
6. Elkayam U. Pregnancy and cardiovascular disease. In: Zipes DP, Libby P, Bonow RO, Braunwald E, editors. Braunwald's heart disease: a textbook of cardiovascular medicine. 7th ed. Philadelphia, PA: WB Saunders; 2005. p. 1965–81.
7. Thorne S, MacGregor A, Nelson-Piercy C. Risks of contraception and pregnancy in heart disease. Heart. 2006;92:1520–5.

8. Siu SC, Sermer M, Colman JM, Alvarez AN, Mercier LA, Morton BC, Kells CM, Bergin ML, Kiess MC, Marcotte F, Taylor DA, Gordon EP, Spears JC, Tam JW, Amankwah KS, Smallhorn JF, Farine D, Sorensen S. Prospective multicenter study of pregnancy outcomes in women with heart disease. Circulation. 2001;104:515–21.
9. Drenthen W, Boersma E, Balci A, Moons P, Roos-Hesselink JW, Mulder BJ, Vliegen HW, van Dijk AP, Voors AA, Yap SC, van Veldhuisen DJ, Pieper PG. Predictors of pregnancy complications in women with congenital heart disease. Eur Heart J. 2010;31:2124–32.
10. Sawhney H, Aggarwal N, Suri V, Vasishta K, Sharma Y, Grover A. Maternal and perinatal outcome in rheumatic heart disease. Int J Gynaecol Obstet. 2003;80:9–14.
11. Sawhney H, Suri V, Vasishta K, Gupta N, Devi K, Grover A. Pregnancy and congenital heart disease--maternal and fetal outcome. Aust N Z J Obstet Gynaecol. 1998;38:266–71.
12. Aggarwal N, Suri V, Kaur H, Chopra S, Rohila M, Vijayvergiya R. Retrospective analysis of outcome of pregnancy in women with congenital heart disease: single-Centre experience from North India. Aust N Z J Obstet Gynaecol. 2009;49:376–81.
13. Kaur H, Suri V, Aggarwal N, Chopra S, Vijayvergiya R, Talwar KK. Pregnancy in patients with tetralogy of fallot: outcome and management. World J Pediatr Congenit Heart Surg. 2010;1:170–4.
14. Sikka P, Suri V, Aggarwal N, Chopra S, Bahl A, Vijayverghia R. Are we missing hypertrophic cardiomyopathy in pregnancy? Experience of a tertiary care hospital. J Clin Diagn Res. 2014:13–5.
15. Suri V, Aggarwal N, Kalpdev A, Chopra S, Sikka P, Vijayvergia R. Pregnancy with dilated and peripartum cardiomyopathy: maternal and fetal outcome. Arch Gynecol Obstet. 2013;287:195–9.
16. Suri V, Aggarwal N, Keepanasseril A, Chopra S, Vijayvergiya R, Jain S. Pregnancy and Takayasu arteritis: a single Centre experience from North India. J Obstet Gynaecol Res. 2010;36:519–24.
17. Suri V, Keepanasseril A, Aggarwal N, Vijayvergiya R, Chopra S, Rohilla M. Maternal complete heart block in pregnancy: analysis of four cases and review of management. J Obstet Gynaecol Res. 2009;35:434–7.
18. https://www.rcog.org.uk/globalassets/documents/guidelines/goodpractice13cardiacdiseaseandpregnancy.pdf.
19. van Hagen IM, Boersma E, Johnson MR, Thorne SA, Parsonage WA, Escribano Subías P, Leśniak-Sobelga A, Irtyuga O, Sorour KA, Taha N, Maggioni AP, Hall R, Roos-Hesselink JW, ROPAC investigators and EORP team. Global cardiac risk assessment in the registry of pregnancy and cardiac disease: results of a registry from the European Society of Cardiology. Eur J Heart Fail. 2016;18:523–33.
20. Sugrue D, Blake S, Troy P, et al. Antibiotic prophylaxis against infective endocarditis after normal delivery--is it necessary? Br Heart J. 1980;44(5):499–502.
21. McFaul PB, Dornan JC, Lamki H, et al. Pregnancy complicated by maternal heart disease. A review of 519 women. Br J Obstet Gynaecol. 1988 Sep.;95(9):861–7.
22. Petanovic M, Zagar Z. The significance of asymptomatic bacteremia for the newborn. Acta Obstet Gynecol Scand. 2001;80(9):813–7.
23. Nishimura RA, Otto CM, Bonow RO, Carrabillo BA, Erwin JP, et al. Practice guideline. J Am Cardiol. 2014;63:e 57–185.
24. Habib G, Lancellotti P, Antunes MJ, Bongiorni MG, Casalta JP, Del Zotti F, Dulgheru R, et al. ESC guidelines for the management of infective endocarditis: the task force for the Management of Infective Endocarditis of the European Society of Cardiology (ESC). Endorsed by: European Association for Cardio-Thoracic Surgery (EACTS), the European Association of Nuclear Medicine (EANM). Eur Heart J. 2015;36:3075–128.
25. Aggarwal N, Suri V, Goyal A, Malhotra S, Manoj R, Dhaliwal RS. Closed mitral valvotomy in pregnancy and labor. Int J Gynaecol Obstet. 2005;88:118–21.
26. Chopra S, Suri V, Aggarwal N, Rohilla M, Vijayvergiya R, Keepanasseril A. Ebstein's anomaly in pregnancy: maternal and neonatal outcomes. J Obstet Gynaecol Res. 2010;36:278–83.
27. Suri V, Keepanasseril A, Aggarwal N, Chopra S, Bagga R, Sikka P, Vijayvergiya R. Mechanical valve prosthesis and anticoagulation regimens in pregnancy: a tertiary centre experience. Eur J Obstet Gynecol Reprod Biol. 2011;159:320–3.
28. Suri V, Sawhney H, Vasishta K, Renuka T, Grover A. Pregnancy following cardiac valve replacement surgery. Int J Gynaecol Obstet. 1999;64:239–46.
29. Nishimura RA, Warnes CA. Anticoagulation during pregnancy in women with prosthetic valves: evidence, guidelines and unanswered questions. Heart. 2015;101:430–5.
30. Saha PK, Joshi B, Suri V, Vijayvergiya R, Sikka P, Aggarwal N, Chopra S. Mitral valve thrombosis in pregnancy: successful restoration with thrombolysis. Am J Emerg Med. 2015;33:1325.e1–2.
31. Sliwa K, Hilfiker-Kleiner D, Petrie MC, Mebazaa A, Pieske B, Buchmann E, Regitz-Zagrosek V, et al. Heart Failure Association of the European Society of Cardiology Working Group on Peripartum Cardiomyopathy. Current state of knowledge on aetiology, diagnosis, management, and therapy of peripartum cardiomyopathy: a position statement from the Heart Failure Association of the European Society of Cardiology Working Group on peripartum cardiomyopathy. Eur J Heart Fail. 2010;12:767–78.
32. Elliott PM, Anastasakis A, Borger MA, Borggrefe M, Cecchi F, Charron P, Hagege AA, et al. Guidelines on diagnosis and management of hypertrophic cardiomyopathy: the Task Force for the Diagnosis and Management of Hypertrophic Cardiomyopathy of the European Society of Cardiology (ESC). Eur Heart J. 2014;35:2733–79.
33. Suri V, Aggarwal N, Kaur R, Chaudhary N, Ray P, Grover A. Safety of intrauterine contraceptive device (copper T 200 B) in women with cardiac disease. Contraception. 2008 Oct;78(4):315–8.
34. World Health Organization. Medical eligibility criteria for contraceptive use. 5th ed; 2015.

Anaemia in Pregnancy

9

Reeti Mehra and Jyotsna Rani

Anaemia in pregnancy is one of the most common medical conditions worldwide. The WHO has defined anaemia in pregnancy if haemoglobin (Hb) is <11 g/dL at any stage of antenatal period [1]. According to NICE guideline and CDC recommendation, the haemoglobin cut-off to define anaemia in pregnancy is <10.5 g/dL in the second and third trimesters and <10 g/dL in the postpartum period [2, 3].

9.1 Incidence and Prevalence of Anaemia in Pregnancy

Anaemia in pregnancy is one of the commonest health problems worldwide especially in developing countries. Despite all efforts of national health programmes to provide optimum antenatal care to all pregnant women, and recommendation of screening for anaemia on their first antenatal visit, more than half of the pregnant women in the world are suffering from this condition [4]. In developed countries, incidence of anaemia in pregnant women is only 15%, whereas relatively higher prevalence (33–75%) has been reported in developing countries [5, 6]. Anaemia in pregnancy has a major impact on nation's health and its economy. It is a major contributing factor in maternal mortality and morbidity and also affects foetal outcome by causing preterm delivery, low birth weight and lower infant Apgar score, thereby adding to the economic burden of healthcare as well [7, 8].

9.2 Grading of Severity of Anaemia in Pregnancy by the WHO

Mild	10.0–10.9 g/dL
Moderate	7.0– 9.9 g/dL
Severe	<7.0 g/dL

9.3 Classification of Anaemia and Their Causes Based on Absolute Reticulocyte Count, RBC Indices and Morphology

9.3.1 Low or Normal Reticulocyte Count (<75,000/cmm), i.e. Hypoproliferative Anaemia

9.3.1.1 Microcytic, Hypochromic (MCV < 80 fL)

- Iron deficiency anaemia
- Thalassemia syndromes
- Sideroblastic anaemia
- Transferrin deficiency

R. Mehra (✉) · J. Rani
Government Medical College and Hospital, Chandigarh, India

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9.3.1.2 Macrocytic (MCV > 100 fL)

- Megaloblastic anaemias
- (Folic acid/vitamin B_{12} deficiency)
- Liver disease
- Reticulocytosis
- Bone marrow failure syndromes
- Drugs (zidovudine, trimethoprim sulphate)

9.3.1.3 Normocytic (MCV 80–100 fL) with Normal Morphology

- Anaemia of renal disease
- Aplastic anaemia
- Infections (malaria, tuberculosis)
- Chronic disease

9.3.2 Increased Reticulocyte count (>100,000/cmm), i.e. Hyperproliferative Anaemia Because of Excessive Haemolysis

9.3.2.1 Normocytic (MCV 80–100 fL), Abnormal Morphology

- Hemoglobinopathies (SS, SC, CC)
- Hereditary spherocytosis
- Autoimmune haemolytic anaemia
- Some enzymatic deficiencies

9.3.2.2 Acute Haemorrhage

Effect of pregnancy on anaemia: The maternal haematological system undergoes dramatic changes during pregnancy. There is an expansion of maternal plasma volume by approximately 40–50%, with increase in red blood cell (RBC) mass also but by 30% only, leading to hemodilution [9]. This may lead to fall in maternal haemoglobin and haematocrit value. Henceforth women who are already anaemic in pre-pregnant state, their Hb falls, and anaemia worsens progressively as the period of gestation advances, if not started on iron therapy. Conversely non-anaemic women in pre-pregnant state achieve normal Hb till the end of 6 weeks of puerperium, if no history of significant obstetric haemorrhage is there.

Approach towards anaemia in pregnancy: Proper history taking includes inquiry about supplementation of iron and folic acid during pregnancy, history of menorrhagia prior to conception, any episode of obstetric haemorrhage, history of pica, worm infestation, hematemesis, melena, hematuria, chronic illness, easy bruising, or family history suggestive of haemoglobinopathies etc. On physical examination, one should look for pallor, icterus, oedema, koilonychia, increased jugular venous pressure, hepatomegaly and splenomegaly. In severe anaemia, there may be presence of soft systolic murmur in the heart (may be physiological) and sometimes basal crepitations denoting lung congestion. Severe anaemia may present with features of congestive cardiac failure.

9.4 Laboratory Investigation

9.4.1 Haemoglobin Concentration

Reduction in Hb value less than 11 g/dL in first trimester and <10.5 g/dL in second and third trimester is the simplest method of diagnosing anaemia. Though decrease in Hb is preceded by depletion of iron store first in cases of iron deficiency anaemia followed by defective erythropoiesis and finally anaemia becomes evident.

9.4.2 Peripheral Blood Picture (PBF)

In iron deficiency anaemia, there is microcytic, hypochromic picture in peripheral blood film along with anisocytosis and poikilocytosis. In mild anaemia or concomitant vitamin B12 or folate deficiency, normocytic picture may be present in lieu of microcytic picture. Macrocytic picture is a characteristic feature of vitamin B12 or folate deficiency. PBF is also looked for WBC differential count, platelet count and their morphology, evidence of haemolysis and malarial parasite.

9.4.3 Red Blood Cell Indices

Impaired haemoglobin synthesis may affect all or any value of MCV, MCH or MCHC. RBC indices help in typing of anaemia. Normal range of MCV is 80–100 fL, of MCH is 27–31 pg/cell and of

MCHC is 32–36 g/dL. Red blood cell distribution width (RDW) along with MCV may help in determination of possible cause of anaemia.

9.4.4 Reticulocyte Count

Increased or decreased reticulocyte count seen in peripheral blood film indicates hypercellular or hypocellular bone marrow, respectively, and cause of anaemia can be predicted with support of other investigations.

9.4.5 Electrophoresis

Electrophoresis is an important test to detect the qualitative or quantitative abnormality of haemoglobin and help in diagnosis of haemoglobinopathies like thalassaemia and sickle cell anaemia.

9.4.6 ESR

Although ESR is raised in anaemia, this is a nonspecific indicator.

Other investigations that may help to establish the cause of anaemia are serum bilirubin; serum LDH; renal function test; urinalysis (routine examination including urinary protein, bilirubin and urobilinogen and microscopy); stool test for occult blood, ova and cyst; X-ray chest PA view; and USG whole abdomen.

Iron deficiency anaemia: Iron deficiency anaemia is the most common cause of anaemia in pregnancy and responsible for about 70–95% of cases [10].

9.5 Physiology and Iron Metabolism

The total iron requirement over the course of pregnancy is approximately 900 mg (range 700–1400 mg), 450 mg of which is consumed in red blood cell expansion [11]. Around 350 mg of iron is used in foetus and placenta. The blood loss in delivery causes loss of 190 mg of iron, and the same amount of iron is consumed in lactational period (1 mg/day). At the same time, amenorrhoea during pregnancy saves approximately 256 mg of iron. Henceforth, at the end of pregnancy followed by postpartum, the woman is left with total iron deficit of approximately 580 mg. Therefore even in iron-replete woman, there is increased iron demand as 4–6 mg/day and 6–8 mg in second and third trimester, respectively [12].

The increased requirement of iron during pregnancy cannot be met by diet alone but is derived partly from maternal reserve. In a well-nourished woman, about half of the total iron requirement get fulfilled from iron store. When the iron reserve already is low due to malnutrition and/or frequent pregnancy, iron deficiency anaemia results.

Foetal iron metabolism: During pregnancy, following absorption of iron from maternal gastrointestinal tract, its transportation occurs to foetal circulation from maternal circulation against the gradient, in contrast to non-pregnant state where it goes to the bone marrow. The transferrin-bound iron from maternal circulation reaches to placenta where several transferrin receptor 1 (TfR1) are present on apical membrane of syncytiotrophoblast. The bivalent iron transferrin complex is internalized after binding with the TfR1 by process of endocytosis, and change in pH leads to release of iron in cytoplasm [13, 14]. Foetal heptacidin has important role in foetal iron metabolism. The overall mechanism is aimed to supply maximum iron to the foetus despite maternal anaemia.

Clinical signs and symptoms: Iron deficiency anaemia in pregnancy can be asymptomatic, and its detection occurs first time during routine antenatal screening. The signs and symptoms are often nonspecific with fatigueness being the commonest complaint. She may complain of weakness, exhaustion, giddiness, palpitations, dyspnoea, hair loss and reduced work performance and swelling of the lower limb or anasarca. Pregnancy with severe anaemia is at risk to have recurrent infection and also associated with increased risk of pre-eclampsia [15, 16]. Few patients with severe anaemia may present with

heart failure during pregnancy (mostly at 32 weeks) or following delivery because of cardiac overload. Anaemic mothers are also at risk to have PPH following delivery. Literature reports high maternal mortality rate in developing countries (varying from 27 to 194 per 100,000 live birth) due to severe anaemia [17]. They may develop lactation failure and postpartum depression in puerperium.

Maternal anaemia causes placentomegaly and affects foetus leading to preterm delivery, low birth weight and its consequences and henceforth increases foetal and neonatal morbidities [18–20]. Literature suggests increased risk of preterm premature rupture of membranes if anaemia develops early in pregnancy, while anaemia later in pregnancy is associated with preterm labor [21]. Maternal anaemia is also associated with risk of birth asphyxia and poor infant Apgar score. Positive correlation has been found between maternal iron supplementation and improvement of Apgar score [22]. Subsequent to preterm labour and low birth weight, associated short-term and long-term outcomes are cerebral palsy, blindness, deafness and hypertension. Mild to moderate anaemia during pregnancy is also a risk factor for iron deficiency anaemia in infant, especially between 6 and 12 months of age [23].

Laboratory diagnosis of iron deficiency: Besides routine test for anaemia, few specific tests are recommended to establish the definite diagnosis of iron deficiency anaemia.

Red blood cell indices: MCV < 75 fl, MCH <25 pg and MCHC<30 g/dL are typical of iron deficiency anaemia. Any or all values of MCV, MCH and MCHC are reduced in iron deficiency anaemia.

Reticulocyte count: Reticulocyte count is decreased in iron deficiency anaemia because of impaired erythropoiesis. On commencing iron therapy, the first sign of improvement seen is increase in reticulocyte count appearing after 7–10 days.

Peripheral blood picture: In iron deficiency anaemia, there is microcytic, hypochromic picture characterized by plenty of pale staining cells different in sizes (anisocytosis) and shape (poikilocytosis) present in peripheral blood film. In mild anaemia or concomitant vitamin B12 or folate deficiency, normocytic picture may be present in lieu of microcytic picture.

Serum ferritin: Serum ferritin is a stable, high molecular weight glycoprotein, and its normal level in serum varies from 50 to 150 ng/mL. This provides an accurate estimation of iron stores. Its serum level can be assayed by ELISA, and the value is not affected by recent ingestion of iron except inflammatory states. Decreased serum ferritin (<30 ng/mL) is the first abnormal sign of iron deficiency anaemia before decline in Hb concentration.

Serum transferrin receptor: This is a newer and reliable method for assessing tissue iron status, and its value increases > threefold in response of iron deficiency anaemia (normal 4–9 mg/L). It provides an early and accurate estimation of the iron deficit between the point of iron store depletion and appearance of iron deficiency anaemia before the MCV gets affected.

Serum iron studies: This includes estimation of serum iron and total iron binding capacity (TIBC); both can help in estimating serum transferrin saturation. Serum iron, transferrin saturation and soluble transferrin receptor are decreased, and TIBC is raised in iron deficiency anaemia, while in chronic disease, serum iron and TIBC both are decreased with normal value of soluble transferrin receptor.

Erythrocyte Zn protoporphyrin concentration: This is a nonspecific indicator because of ineffective erythropoiesis in iron deficiency anaemia. The value is raised >40 mumol/mol haem.

Bone marrow examination: The absence of stainable iron (hemosiderin) in normoblast present in bone marrow is diagnostic of iron deficiency anaemia. This is an invasive test and reserved for cases of severe anaemia where the cause is not established or they are not responding to hematinic.

Prevention and management of iron deficiency anaemia: The WHO recommends 60 mg/day of oral iron supplementation to all antenatal mothers throughout the pregnancy and continued till 3 months of postpartum [24, 25]. Apart from supplementary iron therapy, easily accessible protein- and iron-rich diet should be advised. Common causative agents responsible for anae-

mia like malaria, hookworm or urinary tract infections are also get treated simultaneously. After delivery, women should be advised contraception to avoid early and frequent pregnancy because even non-anaemic woman takes a minimum of 2 years to replenish her iron store which is exhausted in pregnancy and delivery.

Management of iron deficiency anaemia includes correction of the cause of anaemia along with iron therapy. Iron can be administered by oral or parenteral route; decision depends upon the period of gestation at which anaemia is diagnosed and the remaining time available for anticipated delivery.

Oral iron therapy: Oral iron therapy is of choice and recommended ideally when anaemia is not severe and gestation-delivery interval is at least 10 weeks. Oral iron formulations are safe, cheap and effective measures to correct the haemoglobin and replenish the iron store. The current guidelines for iron deficiency anaemia recommend a dose of 100–200 mg of elemental iron per day for a minimum duration of 3 months [27]. Iron preparations are ideally taken on an empty stomach or 1 h before food, as dietary factors may interfere with its absorption or may cause iron chelation making it less bioavailable. Tea and antacids interfere with its absorption, while vitamin C and vitamin A facilitate its absorption. Oral iron preparations are notorious to cause gastric irritation. Henceforth low elemental dose of iron to start with or taking it even in conjunction with meals is advisable for patient having iron intolerance to improve its compliance. Later on, dose is titrated gradually till the recommended dose with growing compliance of the patient. Shifting to different iron formulations or iron syrups may be of help in such group of patients. As iron syrups may cause staining of teeth, taking such preparations with straw is advisable.

Several oral iron preparations are available as capsule, tablet or syrup form, containing varied strengths of elemental iron (Table 9.1). Chiefly bivalent iron formulations like ferrous sulphate, ferrous fumarate and ferrous gluconate are in use. They are different in terms of their absorption and bioavailability as well as cost. Enteric-coated iron formulations are also available but should be avoided [26]. Few trivalent iron compounds such as iron protein succinylate and iron polymaltose complex are also available, and these formulations score over the bivalent preparations in being more tolerant and friendly for the gastrointestinal tract. However trivalent iron preparations have less bioavailability, they need multiple dosing and are expensive.

Side effects of oral iron preparations are nausea, epigastric discomfort and constipation, and few patients may have gastrointestinal intolerance for the same.

When oral iron therapy has been started for established anaemia in pregnancy, response is assessed by getting repeat haemoglobin 2 weeks after commencement of the treatment. The expected increment in haemoglobin is 1 g/dL in 2 weeks. The first sign of improvement in response to iron therapy is increase in reticulocyte count in peripheral blood film, which appears 7–10 days after starting of treatment.

During antenatal visit pregnant women who are not responding to oral iron dosing should be inquired about the colour of stool, thus checking her for compliance. Women, compliant for taking iron, give history of passage of black coloured stool. Other responsible factors are also to look for nonresponders to oral iron therapy.

Table 9.1 Oral iron preparations available over the counter

Iron preparation	Available elemental iron (mg)	Total strength of tablet (mg)	Prophylactic dose (tablets/day)	Therapeutic dose (tablets/day)
Ferrous sulphate	60	300	1	2–3
Ferrous sulphate dried	65	200	1	2–3
Ferrous fumarate	65	200	1	2–3
Ferrous gluconate	35	300	2	3–5

Iron stores need to be replenished by continuing the iron for 3 months and minimum of 6 weeks in postpartum mothers in the dose as being given after attaining the normal serum haemoglobin concentration.

Parenteral iron therapy: Routine antenatal screening for anaemia and regular and continued supplementation of oral iron formulations throughout pregnancy make parenteral iron less commonly to be advised and used in antenatal mothers. Women who have iron intolerance or are nonresponders to oral hematinic because of malabsorption or noncompliance are candidates for parenteral iron therapy. It scores its advantage over oral therapy in its certainty of administration. It increases the Hb at faster rate than oral iron, thus saving the critical time if severe anaemia is detected late in pregnancy, i.e. within 8 weeks of expected delivery. IV iron increases the Hb and replenishes the iron store at faster rate [27]. Parenteral iron can be administered by intramuscular route or by intravenous route, either as injection or infusion. Iron-carbohydrate complexes like iron dextran or iron sucrose and iron gluconate are formulations commonly available in the market. Parenteral iron formulations are depicted in detail in Table 9.2. Before administration of the parenteral iron, serum ferritin estimation is to be done to assess the iron reserve, thus avoiding risk of iron overload.

The total requirement of iron to correct Hb as well to replenish iron store is calculated as follows:

$$\text{Total dose}(\text{mg})\text{of Iron} = \text{Hbdeficit}(\text{g/dL}) \times \text{lean body weight}(\text{lb}) + 1000$$

The Hb deficit for woman is estimated as 12-blood Hb concentration.

Parenteral iron needs administration under medical supervision, preferably in hospital settings because the risk of anaphylactoid reaction is associated with it. Hence, a test dose of 0.5 mL is given to the recipient prior to the administration of recommended dose. The emergency drugs like injection adrenaline, antihistamines, corticosteroids, etc. should be available and ready to combat anaphylactoid reactions.

Intramuscular route: Iron formulations most commonly used for intramuscular route are iron dextran and iron sorbitol complex. Iron dextran is ferric hydroxide with dextran containing iron 50 mg/mL, which can be given intramuscularly or intravenously.

Iron sorbitol complex are smaller molecule and used for intramuscular route only. Its absorption is rapid from injection site within 10 days in contrast to iron dextran which remains there for 3–4 weeks. Iron injection should be given deep in upper and outer quadrant of gluteal muscle, with 20–22 gauze needle using Z technique on daily basis or with injection folinic acid on alternate day.

Disadvantages of intramuscular route: Injections are painful; may cause discoloration of the skin, abscess formation or sarcomatous reaction at injection site; and rarely produce anaphylactoid reaction.

Table 9.2 Detail of parenteral iron preparation

Route of administration	Iron formulation	Concentration of iron (mg/mL)	Test dose required	Maximum dose (mg) permissible in single setting
IM iron	Iron dextran	50	Yes	100 mg
	Iron sorbitol complex	50	Yes	100 mg
IV iron	Iron dextran	50	Yes	100 mg
	Iron sucrose	20	Yes	200 mg
	Iron gluconate	12.5	No	125 mg
	Ferumoxytol	30	No	510 mg
	Iron isomaltoside	100	No	20 mg/kg
	Iron carboxymaltose	50	No	1000 mg

Intravenous route: Low-molecular iron dextran and iron sucrose are safe and common iron preparations used intravenously during pregnancy. Newer formulations like ferumoxytol, iron carboxymaltose and ferric isomaltoside are also available now. They can be administered as single-dose infusion and can be infused fast, and no test dose is required. Experience with these newer preparations are chiefly in patients of chronic kidney disease. These iron preparations should be used in second and third trimester only. Except few studies, their safety and recommendation in obstetric patients in antepartum period need large trial to be conducted in future [28, 29].

Disadvantage of intravenous iron: Local phlebitis, sensitivity reaction, not suitable in patients presenting late in pregnancy the appearance of response seen as rise of Hb requires minimum 4–9 weeks.

Erythropoietin: Recombinant human erythropoietin is chiefly indicated in chronic kidney patients, but it has find its use in resistant cases of severe anaemia in pregnancy during antepartum as well as postpartum states including anaemia due to haematological disorder like thalassaemia and sickle cell anaemia. It may be administered in conjunction with parenteral iron. It acts by stimulating maternal erythropoiesis and many a times may obviate the need of blood transfusion. Recombinant human erythropoietin is a high molecular weight protein, cannot cross the placental barrier and henceforth is safe in pregnancy.

Management of Labour and Delivery: Early screening for anaemia is recommended at first antenatal visit and recheck of haemoglobin concentration at 28 weeks to be done. Daily oral iron supplementation in antenatal period effectively corrects the anaemia, and its occurrence in late pregnancy is usually avoided...If severe anaemia presents in late pregnancy, it indicates the lacunae in implementation of recommendation and provision of national health-care policy and scheme to provide antenatal care to all pregnant women.

Specific recommendations regarding management of iron deficiency anaemia in labour are lacking in literature. Delivery should be conducted in obstetrician-led team. IV access, cross match of blood and screen on admission are the necessary steps along with all the measures taken to minimize the obstetric haemorrhage as less as possible.

9.6 Indications of Blood Transfusion

Women with Hb less than 6 g/dL, presenting in labour or in late pregnancy after 36 weeks, should be managed with adequate blood transfusions with the aim to raise their haemoglobin level to at least 8 g/dL [30].

In labouring women with Hb 7 g/dL, plan of management has to be individualized based on their symptoms, medical history and clinical assessment, accordingly decision to transfuse the blood to be taken. Indications of transfusion with Hb of 7 g/dL are cases of antepartum haemorrhage with continued bleeding or at risk of further episode of significant haemorrhage or those presenting with cardiac decompensation. Packed cell should be transfused under cover of intravenous furosemide to avoid the cardiac overload. Severe anaemia in congestive failure may need a venae section or central line for cardiac monitoring. Prophylactic blood transfusion in asymptomatic patients carries risk of alloimmunization, increases number of hospitalizations and adds to the economic burden. Henceforth the decision to transfuse must be individualized and carefully assessed based on patient's clinical presentation, associated complication in conjunction with the Hb level.

The role of exchange transfusion is sparsely documented in literature and that too only in decompensated severe anaemia.

Along with blood transfusion, other measures must be taken to support her maintaining vitals and oxygen saturation (like propped up position, oxygen supplementation by venturi mask) and restricted use of intravenous fluid. Cutting short of second stage of labour using vacuum or forceps is an individualized decision based on clinical situation to reduce the stress over the heart due to bearing down effort in second stage of labour, although no firm recommendation is there in support of the same.

Restricted use of episiotomy and early and meticulous closure of the same, active management of third stage of labour may reduce obstetric haemorrhage. Judicious use of oxytocin and misoprostol is advocated. Ergometrine is avoided in severe anaemia and in those with cardiac failure.

Role of antibiotics: Women with severe anaemia are prone to develop superimposed infections in antennal period as well as in postnatal period. Henceforth they should have adequate coverage of broad-spectrum antibiotics while in labour and also in immediate postpartum period.

9.7 Hemoglobinopathies

Hemoglobinopathies are genetically inherited disorder owing to either the qualitative or quantitative abnormality of the globin chain of haemoglobin. This includes thalassaemia syndrome which is a quantitative disorder and sickle cell disease, a qualitative disorder of haemoglobin. Sickle cell disease and thalassaemia have different predisposition to occurrence based on regional and ethnic distribution. At-risk population along with their partners need screening by electrophoresis, identification of carrier in couple and affected neonates and preconceptional counselling. The care and management demands multidisciplinary approach involving obstetrician and haematologist.

Sickle cell syndromes: The maternal and foetal risk in sickle cell syndromes are due to increased blood viscosity and occlusion of microcirculation by sickled red blood cell resulting in tissue hypoxia. The patients are prone to develop anaemic crisis and vaso-occlusive crisis causing painful crisis or organ damage. Increased risk of pre-eclampsia, recurrent urinary tract infection, pyelonephritis and thromboembolism is seen with it. Foetal complications include increased risk of miscarriage, intrauterine growth retardation and preterm delivery.

Sickle cell syndromes demand combined multidisciplinary approach including obstetrician, haematologist, anaesthetist and paediatrician and need close supervision in labour. The key points in management of such patients include labour analgesia, adequate hydration and oxygenation to avoid hypoxia and acidosis as a result of stress of labour and continuous intrapartum foetal monitoring. The timing and mode of delivery are affected by obstetric factors. The role of blood transfusion if Hb is <8 g/dL is documented in literature, and the need of thromboprophylaxis should be assessed individually. Following the delivery of the foetus, cord blood is to be sent for haemoglobinopathy screening.

Thalassaemia: Thalassaemia is characterized by low concentration of normal HbA with compensatory rise in HbF or HbA_2. It can be thalassaemia minor if it involves single locus of globin chain, while in thalassaemia major, synthesis of both globin chains is affected resulting in severe anaemia due to chronic haemolysis and subsequently iron accumulation and multiorgan damage.

Thalassaemia minor resembles with iron deficiency anaemia though serum iron studies may be the differentiating feature. These patients may be given oral iron supplementation only if serum ferritin level is reduced. Parenteral iron therapy is contraindicated.

Pregnancy with thalassaemia major needs high level of care and close supervision involving multidisciplinary approach since preconception. The oral medication with desferrioxamine in prepregnancy period is discontinued once the pregnancy is confirmed. These patients develop severe anaemia and need multiple blood transfusions and are at high risk to develop cardiac failure. Obstetric complications are like increased association with pre-eclampsia and intrauterine growth retardation and have higher rate of caesarean section.

Management of thalassaemia major during labour is similar as that of woman with severe anaemia in labour.

9.8 Megaloblastic Anaemia

Folate deficiency is the most common cause of megaloblastic anaemia during pregnancy, while vitamin B12 deficiency is rarely responsible for

it. Poor supply in diet, hyperemesis gravidarum, tropical sprue causing impaired folate absorption, multiple pregnancy or frequent pregnancy, alcohol consumption and smoking, etc. are the predisposing factors responsible for causing folate deficiency.

Women with folate deficiency are at high risk to have complications like miscarriage, preterm labour and abruption placentae. There is increased risk of congenital malformations like cleft lip, cleft palate and neural tube defect. There is also its association seen with hyperhomocysteinaemia. Full-blown megaloblastic anaemia is at greater risk for maternal mortality than iron deficiency anaemia.

Women with megaloblastic anaemia do not respond to iron therapy alone. Clinical features may vary like nausea, vomiting, atrophic glossitis and purpuric spots. Vitamin B12 deficiency may be responsible for neurological symptoms. Severe cases of megaloblastic anaemia may present with congestive cardiac failure.

9.9 Specific Laboratory Investigation

Peripheral blood picture shows macrocytes, hypersegmented neutrophils, Howell-Jolly bodies and pancytopenia. The diagnosis can be made by reduced fasting serum folate level <3 ng/mL and the erythrocyte folate level <20 ng/mL.

Management: Oral folic acid supplementation in dose of 5 mg/day is recommended since preconception till several weeks postpartum if diagnosis is made prenatally. Vitamin B12 deficiency is treated with cyanocobalamine or hydroxocobalamin given by parenteral route as megaloblastic changes in intestine impair its absorption. The dose is 1 mg of hydroxocobalamin or cyanocobalamine on alternate day for 2 weeks.

There is no specific recommendation for management of such women in labour. Indication of blood transfusion is severe anaemia, the associated complication of antepartum haemorrhage and hemodynamic instability.

References

1. WHO. Haemoglobin concentrations for the diagnosis of anaemia and assessment of severity. In: vol. WHO/NMH/NHD/MNM/11.1. Geneva: World Health Organisation; 2011.
2. National Institute for Health and Care Excellence (NICE). Clinical Guideline 62, Antenatal Care: routine care for the healthy pregnant woman.National Collaborating Centre for Women's and Children's Health (UK). London: RCOG Press; 2008. ISBN-13: 978-1-904752-46-2.
3. CDC. Recommendations to prevent and control iron deficiency in the United States. Centers for Disease Control and Prevention. MMWR Recomm Rep. 1998;47(RR-3):1–29.
4. World Health Organization (WHO). Prevention and management of severe anaemia in pregnancy: report of a technical working group. Geneva, Switzerland: WHO; 1993. WHO/FNE/MSM/93.5
5. Massawe SN, En U, Nystrom L, Lindmark G. Effectiveness of primary level care in decreasing anemia at term in Tanzania. Acta Obstet Gynecol Scand. 1999;78:573–9.
6. Nyuke RB, Letsky EA. Etiology of anaemia in pregnancy in South Malawi. Am J Clin Nutr. 2000;72:247–56.
7. Mahomed K. Iron and folate supplementation in pregnancy. Cochrane Database Syst Rev. 2000;2:CD001135. World Health Organization Reproductive Health Library CD-ROM. 2004;7
8. Rousia U, Madan N, Agarwal N, Sikka M, Sood S. Effect of maternal iron deficiency anaemia on fetal outcome. Indian J Patholo Microbiol. 1995;38:273–9.
9. Koller O. The clinical significance of hemodilution during pregnancy. Obstet Gynecol Surv. 1982;37:649.
10. Looker AC, Dallman PR, Carroll MD, Gunter EW, Johnson CL. Prevalence of iron deficiency in the United States. J Am Med Assoc. 1997;277(12):973–6.
11. McFee JG. Iron metabolism and iron deficiency during pregnancy. Clin Obstet Gynecol Ind. 1997;22:799–808.
12. Turmen T, Abouzahr C. Safe motherhood. Int J Gynecol Obstet. 1994;46:145–53.
13. McArdle HJ, Morgan EH. Transferrin and iron movements in the rat conceptus during gestation. J Reprod Fertil. 1982;66(2):529–36.
14. McArdle HJ, Douglas AJ, Morgan EH. Transferrin binding by microvillar vesicles isolated from rat placenta. Placenta. 1984;5(2):131–8.
15. Ekiz E, Agaoglu L, Karakas Z, Gurel N, Yalcin I. The effect of iron deficiency anemia on the function of the immune system. Hematol J. 2005;5:579–83.
16. Rohilla M, Raveendran A, Dhaliwal LK, Chopra S. Severe anaemia in pregnancy: a tertiary hospital experience from northern India. J Obstet Gynaecol. 2010;30(7):694–6.
17. Brabin BJ, Hakimi M, Pelletier D. An analysis of anemia and pregnancy-related maternal mortality. J Nutr. 2001;131(2S-2):604S–14S; discussion 614S–5S.

18. Hemminki E, Rimpela U. Iron supplementation, maternal packed cell volume, and fetal growth. Arch Dis Child. 1991;66:422–5.
19. Agarwal KN, Agarwal DK, Mishra KP. Impact of anaemia prophylaxis in pregnancy on maternal hemoglobin, serum ferritin and birth weight. Indian J Med Res. 1991;94:277–80.
20. Singla PN, Tyagi M, Kumar A, Dash D, Shankar R. Fetal growth in maternal anemia. J Trop Pediatr. 1997;43:89–92.
21. Zhang Q, Ananth CV, Li Z, Smulian JC. Maternal anaemia and preterm birth: a prospective cohort study. Int J Epidemiol. 2009;38(5):1380–9.
22. Goldenberg RL, Culhane JF. Low birth weight in the United States. Am J Clin Nutr. 2007; 85(2):584–90.
23. Colomer J, Colomer C, Gutierrez D, Jubert A, Nolasco A, Donat J, Fernandez-Delgado R, Donat F, Alvarez-Dardet C. Anaemia during pregnancy as a risk factor for infant iron deficiency: report from the Valencia Infant Anaemia Cohort (VIAC) study. Paediatr Perinat Epidemiol. 1990;4(2):196–204.
24. UN Children's Fund, U, WHO. Iron deficiency anaemia. Assessment prevention, and control. A guide for programme managers. Geneva (Switzerland): World Health Organization; 2001.
25. Reveiz L, Gyte GM, Cuervo LG. Treatments for iron deficiency anaemia in pregnancy. Cochrane Database Syst Rev. 2007;2:CD003094.
26. Pavord S. UK guidelines on the management of iron deficiency in pregnancy. Br J Haematol. 2012;56(5):588–600.
27. Komolafe JO, Kuti O, Ijadunola KT, Ogunniyi SO. A comparative study between intramuscular iron dextran and oral ferrous sulphate in the treatment of iron deficiency anaemia in pregnancy. J Obstet Gynaecol. 2003;23(6):628–31.
28. Froessler B, Collingwood J, Hodyl NA, Dekker G. Intravenous ferric carboxymaltose for anaemia in pregnancy. BMC Pregnancy Childbirth. 2014;14:115.
29. Christoph P, Schuller C, Studer H, Irion O, De Tejada BM, Surbek D. Intravenous iron treatment in pregnancy: comparison of high-dose ferric carboxymaltose vs. iron sucrose. J Perinat Med. 2012;40(5):469–74.
30. ACOG Practice Bulletin No. 95: anemia in pregnancy. Obstet Gynecol. 2008;112(1):201–7.

Diabetes

10

K. Aparna Sharma and Gunjan Rai

10.1 Definition

Historically, gestational diabetes mellitus (GDM) is defined as any degree of glucose intolerance with onset or first recognition during pregnancy [1].

Increasingly, it has been seen that diabetes in pregnancy may either be a manifestation of pregnancy-induced resistance to insulin (GDM) or may result from the unmasking of the previously undiagnosed diabetes (overt diabetes or diabetes mellitus). It is vital to distinguish between the two entities as their effects on the maternal and fetal outcomes vary considerably. Table 10.1 shows the summary of the definitions currently being used to differentiate them.

10.2 Intrapartum Issues in Women with Diabetes

Good glycemic control remains important in the intrapartum period because maternal hyperglycemia during labor increases the risk of fetal acidemia and neonatal hypoglycemia. Intrapartum

Table 10.1 Defining diabetes in pregnancy

	Fasting mg/dL	1 h mg/dL	2 h (75-g OGTT) mg/dL	HbA1C	Random
ADA [2, 3]					
DM/overt diabetes (any one criteria)	>126		200	>6.5%	200 mg/dL with symptoms of hyperglycemia
IADPSG (GDM)					
	92 mg/dL	180	153 mg/dL		
WHO/IADPSG [4, 5]					
DM	≥126		≥200	≥6.5	
Impaired fasting	110–126			6.0–6.4	
Impaired glucose tolerance			≥140	6.0–6.4	

ADA American Diabetes Association, *DM* diabetes mellitus, *IADPSG* International Association of Diabetes in Pregnancy Study Group, *WHO* World Health Organization, *OGTT* oral glucose tolerance test 75 g glucose load

K. A. Sharma (✉)
Department of Obstetrics and Gynecology, All India Institute of Medical Sciences, New Delhi, India

G. Rai
Army Hospital (R&R), New Delhi, India

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maternal normoglycemia will not reduce the risk of neonatal hypoglycemia in women with poor antepartum glycemic control, since fetal pancreatic hyperplasia and excessive in utero insulin secretion have been established in response to prolonged exposure to hyperglycemia.

10.2.1 Key Targets

During labor and delivery, the goal is to maintain normoglycemia, that is, blood glucose level between 70 and 126 mg/dL to prevent neonatal hypoglycemia [6, 7]. Intrapartum euglycemia is also important for preventing fetal hyperglycemia. If intrapartum hyperglycemia occurs on a background of chronically poor maternal metabolic control (high glycated hemoglobin [A1C]), this is an increased risk of fetal hypoxemia and acidosis.

Table 10.2 Frequency of intrapartum blood sugar monitoring

Gestational diabetes on diet/medical therapy	At admission; 4–6 h	
Type 1 diabetes Type 2 diabetes	Latent phase 2–4 h	Active phase 1–2 h 1 h if insulin infusion

10.2.2 Glucose Monitoring

The frequency of intrapartum glucose monitoring would depend on the type of diabetes and the antenatal control of blood sugar. Table 10.2 gives the overview of glucose monitoring.

10.2.3 Intrapartum Insulin Administration

Insulin can be administered as either subcutaneous intermittent rapid-acting insulin dosages or as intravenous infusion as shown in Table 10.3.

10.2.4 Emergencies

Three emergency situations encountered during pregnancy in a diabetic mother are:

- Diabetic ketoacidosis.
- Hypoglycemia.
- Hyperosmolar hyperglycemic state.

10.2.4.1 Diabetic Ketoacidosis (DKA)

Diabetic ketoacidosis (DKA) occurs in about 0.5–3% of diabetic pregnant women [8]. Physiological changes in pregnancy predispose

Table 10.3 Intrapartum insulin administration protocols

Maternal plasma glucose mg/dL	Subcutaneous insulin (units)	Insulin infusion (units/h)	Rotating fluids protocol (only in women with GDM and not type 1 or type 2 diabetes)	
			mg/dL	Fluid
			<100	5%DNS
<120	0	0	100–140	RL/NS
121–140	1	1		
141–160	2	2	>140	RL/NS with short or rapidacting insulin infusion to achieve a blood glucose of 100 mg/dL
161–180	3	3		
181–200	4	4		
>200	4 plus IV regular insulin	4 plus IV regular insulin		
Glucose monitoring	2 hourly	1 hourly		

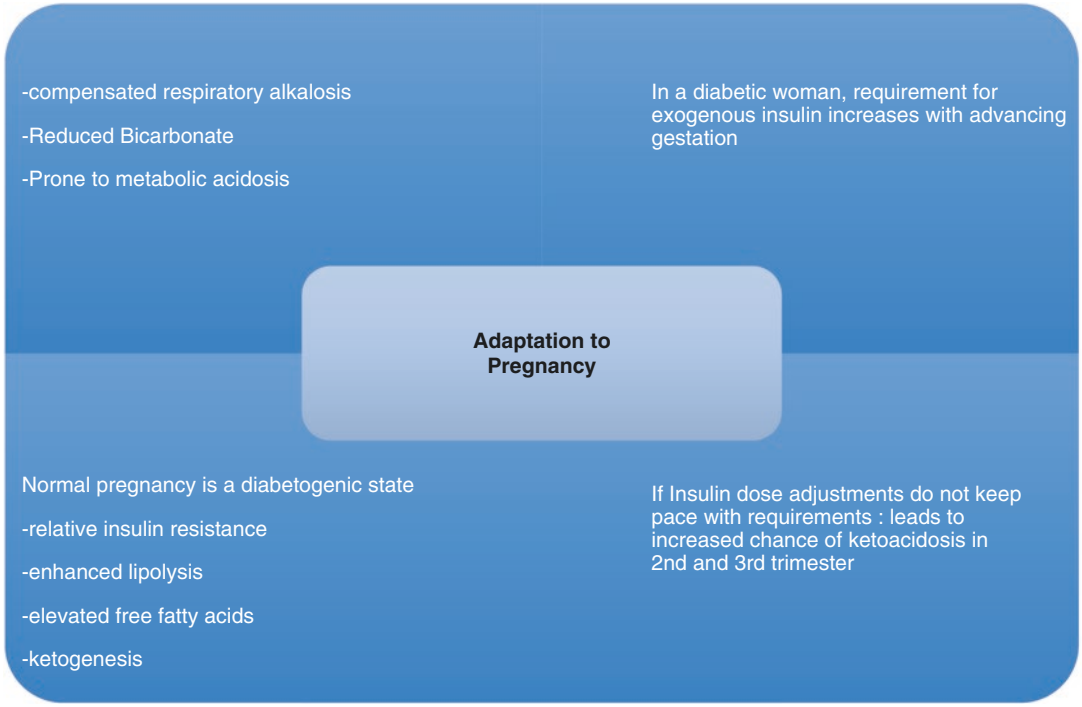


Fig. 10.1 Physiological adaptations in pregnancy predisposing to diabetic ketoacidosis

to the development of DKA in pregnancy as shown in Fig. 10.1. DKA occurs more commonly in women with new onset type 1 diabetes and less frequently with type 2 diabetes and rarely with gestational diabetes. Fetal mortality [9, 10] in DKA is related to the maternal decompensation and is now reported to be 10%.

Factors Predisposing to DKA

The factors predisposing to DKA include:

- Infection
- Vomiting
- Steroids
- Drugs, e.g., betamimetics

Physiological Adaptation in Pregnancy

Figure 10.1 shows the various changes that predispose to DKA in a diabetic pregnant woman.

Pathophysiology of DKA

DKA results in hyperglycemia with metabolic acidosis resulting from decreased effective insulin and increased insulin counter-regulatory hormones as shown in Fig. 10.2.

Diagnosis

In a diabetic pregnant woman on insulin, DKA should be suspected if there is a complaint of nausea and vomiting with persistent moderate hyperglycemia. The classic triad has been often described as:

- D—Dehydration
- K—Ketosis
- A—Acidosis

Patients can present with tachypnea (Kussmaul breathing), tachycardia, abdominal pain, "fruity" acetone odor, and mild neurologic signs/symptoms like drowsiness, lethargy, and even coma. The diagnosis can be confirmed by laboratory findings in Table 10.4.

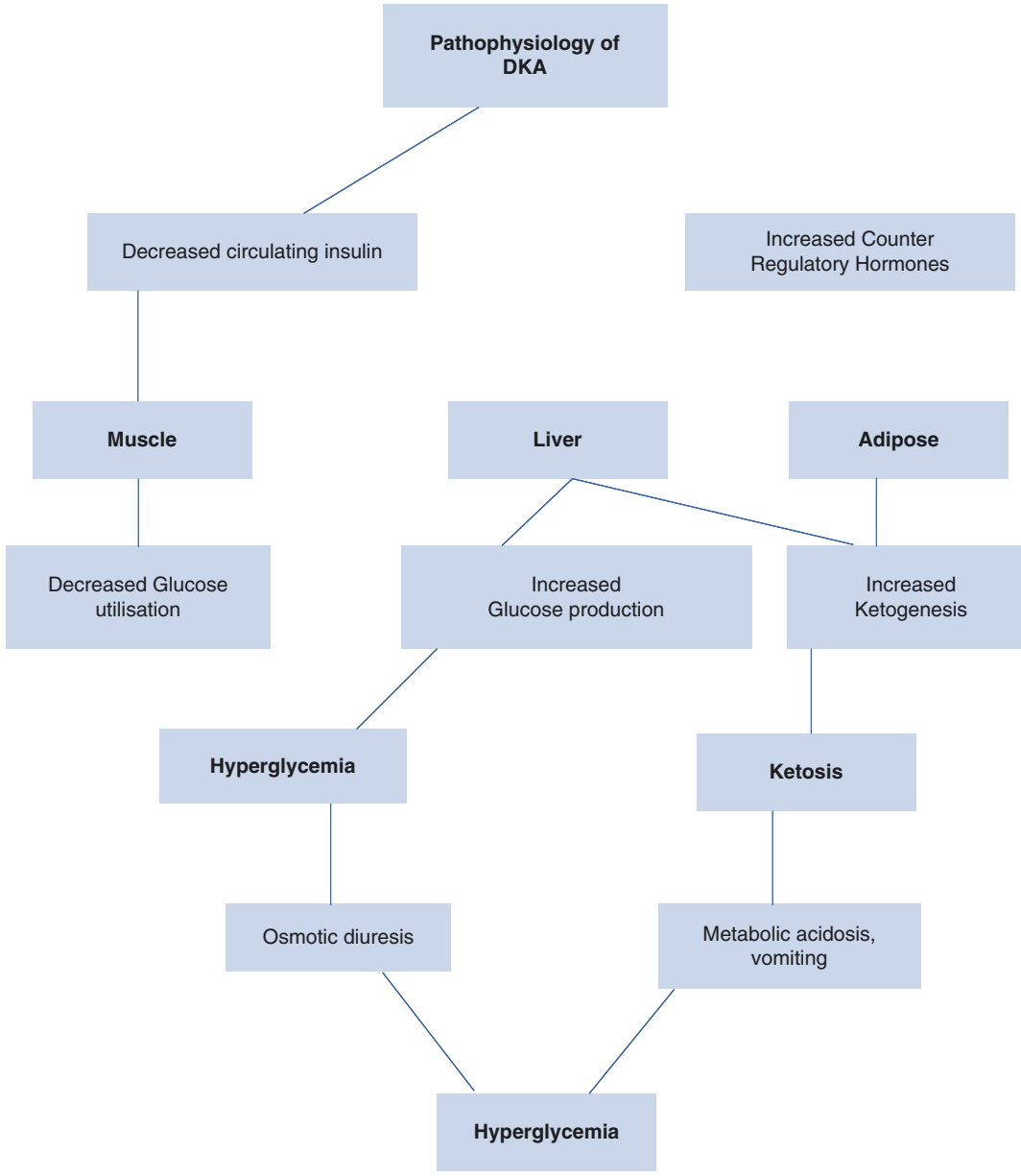


Fig. 10.2 Pathophysiology of DKA

Treatment

Diabetic ketoacidosis is an acute emergency. Its management requires intensive treatment by a multidisciplinary team of obstetricians and endocrinologists. The principles of management are judicious fluid replacement to correct dehydration, insulin replacement, and correction of dyselectrolytemia.

Table 10.5 summarizes the management of DKA.

10.2.4.2 Hyperglycemic Hyperosmolar State (HHS)

This acute emergency is far less commonly encountered as compared to DKA. This is characterized by hyperglycemia which leads to

profound osmotic diuresis, dehydration, and hyperosmolality. Coma can occur if osmolality exceeds 320–330 mOsm/kg. The laboratory changes as compared to DKA have been shown in Table 10.4.

HHS is also precipitated by infection, therapy with glucocorticoids or recent operation. As with DKA the management principles are correcting acidosis and dehydration.

Table 10.4 Laboratory diagnosis of DKA and HHS

		DKA	HHS
1	Hyperglycemia	>250 mg/dL	>600 mg/dL
2	Acidosis	< pH 7.3	>7.3
3	Anion gap	>12 mEq/L	Variable
4	Bicarbonate	<15 mEq/L	>18 mEq/L
5	Ketonemia	>1:2 dilution	Small
6	Serum osmolality (mOsm/kg)	Variable	>320

10.2.4.3 Hypoglycemia

Definition

Hypoglycemia can be defined as "any abnormally low plasma glucose concentration that exposes the subject to potential harm" with a proposed threshold plasma glucose value <70 mg/dL [11].

Signs and Symptoms

The manifestations of hypoglycemia can either be autonomic or neuroglycopenic.

Autonomous Symptoms

Sweating, tremor, anxiety, pallor, palpitations, tachycardia

Neuroglycopenic Symptoms

Confusion, drowsiness, inappropriate behavior, perioral and peripheral tingling, diplopia, slurred

Table 10.5 Management of DKA

Management principles			
Acidosis	Treat dehydration	Monitor fetus	Treat underlying cause
Monitor ABG and anion gap 2 hours monitor S. Glucose and ketones every 2 h	Monitor output Renal functions	• Fetal monitoring. • Transient fetal heart rate abnormalities can occur • Mother should be stabilized before intervention for fetal indication	• Screen for infections • Antibiotics as appropriate
1. **Insulin therapy** – Initiate therapy with 0.1 U/kg bolus followed by 0.1 U/kg/h till acidosis resolves and Ketones clear – Mix 50 U regular insulin in 500 mL NS (10 mL = 1 unit) – Serum glucose should decreased at the rate of 60–75 mg/dL/h – At a blood glucose levels of 250 mg/dL, dextrose should be added to prevent hypoglycemia	**Fluid therapy** Fluid deficit of 100 mL/kg Correct 75% of estimated fluid deficit over first 24 h Initial 24 h: Use isotonic saline (0.9% NS) First hour: 1 L NS Second hour: 0.5–1 L NS Third hour: 0.5 L NS For 24 h: 0.25 L/h 0.45% NS until 75% deficit corrected Continue hydration for 24–48 h till acidosis resolves and ketones clear		
2. **Electrolyte replacement** Monitor S. Electrolytes every 2 hours 2.1 Potassium Anticipate deficit of 5–10 mEq/kg. With maintain urine output (0.5 mL/kg/h). Maintain serum K level at 4–5 mEq/L >5 mEq/L: No treatment 4–5 mEq/L: 20 mEq/L replacement 3–4 mEq/L: 30–40 mEq/L replacement 3 mEq/L: 40–60 mEq/L replacement 2.2 Phosphate: Not usually required 2.3 Bicarbonate: Not usually required			

Table 10.6 Severity of hypoglycemia

Mild	70 mg/dL	– Autonomic symptoms – Able to self-treat
Moderate	55 mg/dL	– autonomic and neuroglycopenic symptoms – Able to self-treat
Severe	40 mg/dL	– Unconsciousness – Require assistance for treatment

speech, headache, unsteady gait, aggressive behavior, convulsions coma.

Severity

The classification of severity of hypoglycemia is as follows (Table 10.6):

Causes

Hypoglycemia results from an imbalance of the delicate glucose and insulin homeostasis. The common causes are

- Skipped meals
- Vigorous exercise
- Excess insulin
- Stress
- Oscillating blood glucose levels
- Impaired hypoglycemia awareness [12]

Consequences

Hypoglycemia has deleterious effects on both the mother and the fetus. It can cause sudden cardiac death and also have long-term consequences on the intellectual function. It can also result in sudden intrauterine fetal death

Treatment of Hypoglycemia

Asymptomatic hypoglycemia or mild hypoglycemia can be treated usually by women. However severe hypoglycemia requires urgent management with intravenous glucose as shown in Fig. 10.3.

10.2.5 Timing of Delivery

Women with gestational diabetes controlled on diet can continue pregnancy up to term (40 weeks) unless there are other indications for an early delivery like preeclampsia. In women who have type 1 or type 2 diabetes or those on insulin or hypoglycemics orally, delivery should be planned after 38 weeks.

10.2.6 Special Situations

10.2.6.1 Scheduled Caesarean Delivery

In women on insulin, the caesarean delivery should be planned early in the morning. The pregnant woman should maintain her usual nighttime dose of intermediateacting insulin, short or rapidacting insulin, and oral antidiabetic medication until admission to the hospital. However, if a longacting insulin is used at night, the dose is decreased by 50% or switched to NPH insulin, and onethird of the longacting nightly dose is given.

The morning dose of insulin or oral antidiabetic agent is held, and the patient is kept nil orally. If the caesarean is delayed, basal insulin (about onethird of the morning dose of intermediate or longacting insulin) is given with a 5% dextrose infusion to avoid ketosis. Glucose levels should be monitored frequently, every 1–3 h.

10.2.6.2 Induction of Labor

Ideally, induction is scheduled for early morning. The patient should maintain her usual nighttime dose of intermediateacting insulin, short or rapidacting insulin, oral antihyperglycemic medication. If she uses a longacting insulin at night, the dose needs to be decreased by 50% or switched to intermediate acting at onethird of the longacting nightly dose.

The woman should eat a light breakfast (half of her usual breakfast intake) and reduce her insulin dose (intermediate- and short-acting insulin) by 50%. Continued oral intake (at 50% of daily intake, 1000–1200 kcal) is permitted during cervical ripening/latent phase when this period is anticipated to exceed 8–12 h.

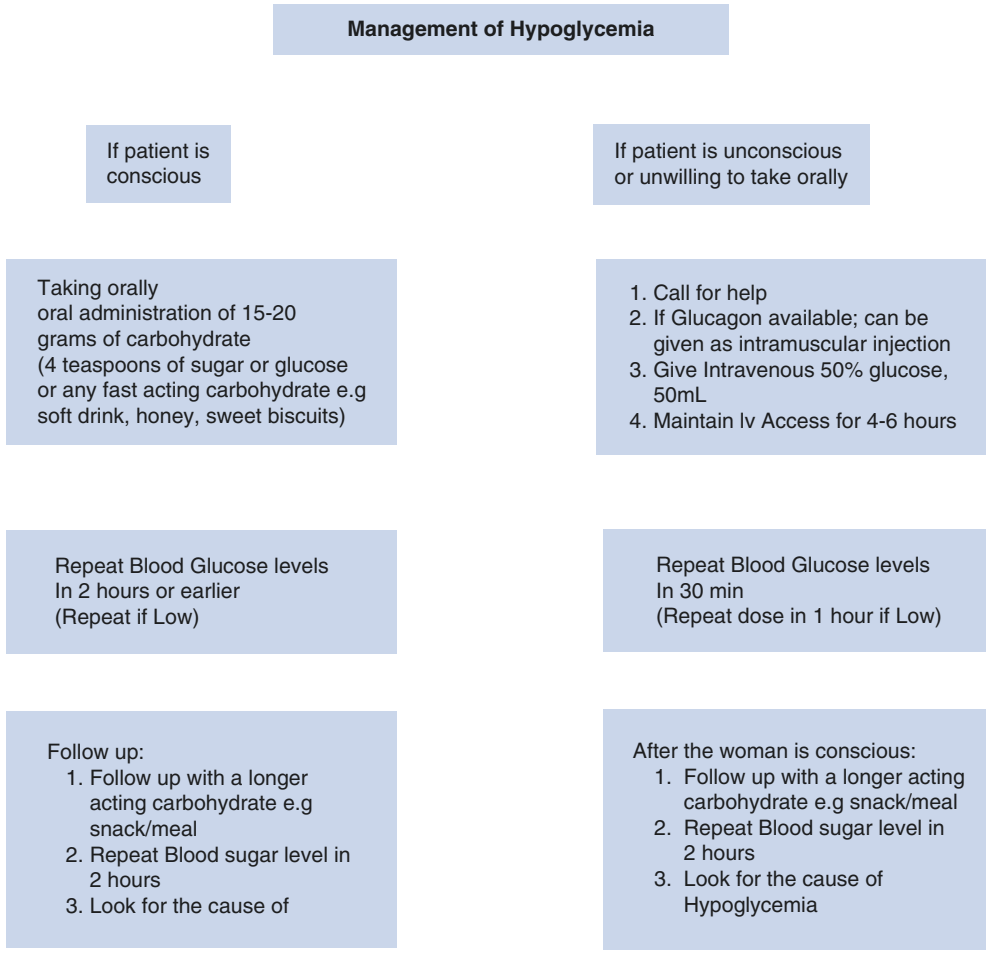


Fig. 10.3 Management of hypoglycemia

References

1. Proceedings of the 4th international workshop-conference on gestational diabetes mellitus. Chicago, Illinois, USA. 14–16 March 1997. Diabetes Care. 1998;21(Suppl 2):B1.
2. American Diabetes Association. Standards of medical care in diabetes--2010. Diabetes Care. 2010;33(Suppl 1):S11–61.
3. American Diabetes Association. Standards of medical care in diabetes—2013. Diabetes Care. 2013;36(Suppl 1):S11–66.
4. World Health Organization. Definition and diagnosis of diabetes mellitus and intermediate hyperglycemia: report of a WHO/IDF consultation (PDF). Geneva: World Health Organization; 2006. p. 21. ISBN 978-92-4-159493-6.
5. Reece EA, Holford T, Tuck S, Bargar M, O'Connor T, Hobbins JC. Screening for gestational diabetes: one-hour carbohydrate tolerance test performed by a virtually tasteless polymer of glucose. Am J Obstet Gynecol. 1987;156(1):132–4.
6. ACOG Committee on Practice Bulletins. ACOG Practice Bulletin. Clinical Management Guidelines for ObstetricianGynecologists. Number 60, March 2005. Pregestational diabetes mellitus. Obstet Gynecol. 2005;105:675.
7. Blumer I, Hadar E, Hadden DR, et al. Diabetes and pregnancy: an endocrine society clinical practice guideline. J Clin Endocrinol Metab. 2013;98:4227.

8. Sibai BM, Viteri OA. Diabetic ketoacidosis in pregnancy. Obstet Gynecol. 2014;123:167.
9. Parker JA, Conway DL. Diabetic ketoacidosis in pregnancy. Obstet Gynecol Clin N Am. 2007;34:533–43.
10. Kamalakannan D, Baskar V, Barton DM, et al. Diabetic ketoacidosis in pregnancy. Postgrad Med J. 2003;79:454–7.
11. American Diabetes Association Workgroup on Hypoglycemia. Defining and reporting hypoglycemia in diabetes. Diabetes Care. 2005;28:1245–9.
12. Gama R, Teale JD, Marks V. Best practice No 173: clinical and laboratory investigation of adult spontaneous hypoglycaemia. J Clin Pathol. 2003;56(9):641–6.

Bronchial Asthma in Pregnancy

11

Priti Kumar, Sanjay Kumar,
and Malavika Chaturvadi

Asthma is reportedly the most common potentially serious yet treatable medical problem to complicate pregnancy occurring in about 3–8% of pregnant women.

The control of maternal asthma is directly proportional to better outcomes in both maternal and fetal.

Large recent studies seem to point to us that asthma follows **a rule of thirds** during pregnancy—1/third of the pregnant women get better, 1/third of the pregnant women remain the same, and in 1/third of the pregnant women, the symptoms worsen.

It has also been noted that in most pregnant asthmatic women, the same disease course during the first pregnancy is followed during the latest pregnancies.

In a large study, most acute exacerbations of asthma occur between 24th and 36th weeks of gestation, and such acute flare-ups are rare during the last month of gestation.

Ninety percent of pregnant women with adequately controlled asthma have no symptoms during labor, delivery, and puerperium, and most of them will go back to their prepregnancy status within 12 weeks.

P. Kumar (✉)
King Georges Medical College, Lucknow, India

Department of Obgyn, Career Institute of Medical Sciences, Lucknow, India

S. Kumar · M. Chaturvadi
Sunflower Medical Centre, Lucknow, India

11.1 Pregnancy-Induced Physiological Changes

Alterations of the cardiorespiratory physiology that occur during pregnancy contribute to the exacerbation of many respiratory problems in women during their pregnancy.

11.1.1 Respiratory System Changes

11.1.1.1 Anatomical Changes

1. Changes in chest wall conformation.
2. Elevation of the diaphragm.
3. Respiratory center stimulation due to the effect of progesterone.

11.1.1.2 Physiological Changes

1. Increase in tidal volume and minute volume.
2. Lung volumes and capacities:
   (a) Inspiratory capacity and tidal volume increased (due to progesterone).
   (b) FRC, ERV, and RV decrease (due to elevation of diaphragm).

$$\begin{bmatrix} \text{FRC, functional residual capacity} \\ \text{ERV, expiratory reserve volume} \\ \text{RV, residual volume} \end{bmatrix}$$

   (c) TLC (total lung capacity)—mild decrease or unchanged.

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_11

In summary, though pulmonary functions are altered in pregnancy, it only induces stress on respiratory function of the pregnant women in the presence of any respiratory disease like asthma.

11.1.1.3 ABG

Because of increased minute ventilation, alveolar ventilation is increased leading to respiratory alkalosis. This gets partially compensated by a metabolic acidosis generated by the kidney.

As a result of this, the gravida's *normal* **ABG** may appear abnormal, i.e., show **features s/o respiratory alkalosis**. Therefore we must keep in mind that for a pregnant woman:

Normal pH: 7.4–7.47.
Normal pCO_2: 25 mm of Hg–32 mm of Hg.

11.1.2 Cardiovascular System Changes

1. Increased blood volume (20–100% above preconception levels) due to increased plasma volume and increased red cell mass.
2. Cardiac output rises by 30–60%.
3. Heart rate rises by 10–20 bpm.
4. Decreased vascular resistance (progesterone-induced relaxation of smooth muscles) leads to a minor fall in both diastolic blood pressure and systolic blood pressure.

11.2 Effects of Asthma on Pregnancy

Poorly controlled maternal asthma raises the risk of:

1. Preterm birth.
2. Small for gestational age infants.
3. Intrauterine growth restriction.
4. Stillbirth.
5. Congenital malformations (e.g., spina bifida, VSD and ASD (ventricular/atrial septal defects).
6. Chorioamnionitis.
7. Gestational diabetes.
8. Low APGAR scores.

Fetal hypoxia as a result of poor asthmatic control of the gravida can even lead to neonatal respiratory difficulties, fetal brain ischemia, and cerebral palsy. In contrast if asthma is well controlled throughout pregnancy, there is little or no increased risk of adverse maternal and fetal outcomes.

Therefore pregnancy should call for optimizing therapy and maximizing lung function in order to decrease the possibility of acute exacerbation.

11.3 Effects of Pregnancy on Asthma

- Worsening of asthma during pregnancy may be due to multiple contributory factors—allergen exposure; upper respiratory tract infection, especially rhinitis; gastroesophageal reflux poor compliance of medication, continue smoking illicit drug use; etc.
- High-risk patients are those who have history of severe preconception asthma and those whose asthma has worsened in earlier pregnancies (Table 11.1).

Table 11.1 Risk factors and triggers involved in asthma

Endogenous factors	Environmental factors
Genetic predisposition	Indoor allergens
Atopy	Outdoor allergens
Airway hyperresponsiveness	Occupational sensitizers
Gender	Passive smoking
Ethnicity	Respiratory infections
Obesity	Diet
Early viral infections	Acetaminophen (paracetamol)
Triggers	
Allergens	
Upper respiratory tract viral infections	
Exercise and hyperventilation	
Cold air	
Sulfur dioxide and irritant gases	
Drugs (β blockers, aspirin) stress	
Irritants (household sprays, paint fumes)	

Apart from these factors, in pregnancy GERD (gastroesophageal reflux disease) and allergic rhinitis are common triggers.

11.4 Differential Diagnosis for Acute Dyspnea in Pregnancy

- Physiological dyspnea of pregnancy which is a benign symptom.
- Pulmonary edema.
- Pulmonary embolism.
- Pneumothorax.
- Pneumonia.
- Worsening asthma.
- Severe asthma.
- Pregnant patients can also suffer from various hematological and cardiac diseases which can produce anemia and lead to dyspnea.
- Airway obstruction.
- Amniotic fluid embolism.
- Acute congestive heart failure (CHF).

11.4.1 Management of the Asthmatic Gravida

The goals of successful asthma management are:

1. Prevention of chronic day and night symptoms.
2. Maintenance of optimal pulmonary function and normal activities using therapies with minimal or no adverse side effects.
3. Maintain fetal oxygenation by preventing episodes of maternal hypoxia (Table 11.2).

The best way to achieve these goals is by a multidisciplinary approach that incorporates regular monitoring of clinical symptoms, self-management education, and the correct use of pharmacotherapies.

The British Thoracic Society (BTS) and Global Initiative for Asthma (GINA) recommend continuing pregnant women on the same asthma therapy used prior to the pregnancy, if their asthma is well controlled.

11.5 Clinical Presentation

Patients, both pregnant and nonpregnant, can present with the following symptoms:

- Cough.
- Shortness of breath.
- Tightness in the chest.
- Noisy, sometimes shallow, breathing.
- Nocturnal awakenings.
- Recurrent episodes of symptom complex.
- Exacerbations possibly provoked by nonspecific stimuli.
- Personal or family history of other atopic disease (like hay fever, eczema).

General physical examination findings may include the following:

- Tachypnea.
- Retraction of the accessory muscles of respiration (sternomastoid, abdominal, pectoralis muscles).
- Agitation—usually a sign of hypoxia or respiratory distress.
- Pulsus paradoxus (>20 mm Hg).

Table 11.2 Asthma control

Characteristic	Controlled (all of the following)	Partly controlled	Uncontrolled
Daytime symptoms	None (≤2/week)	>2/week	Three or more features of partly controlled
Limitation of activities	None	Any	
Nocturnal symptoms/awakening	None	Any	
Need for reliever/rescue treatment	None (≤2/week)	>2/week	
Lung function (PEF or FEV_1)	Normal	80% Predicted	

Abbreviations: *FEV_1* forced expiratory volume in 1 s, *PEF* peak expiratory flow

Table 11.3 Initial clinical assessment of severity in acute asthma

Findings	Mild	Moderate	Severe
Speaking in …	Sentences	Phrases	Words
Heart rate	<100 beats/min	100–120 beats/min	>120 beats/min
Peak Flow/FEV_1 (% predicted)	>75%	50%–75%	<50%
Pulse oximetry	>95%	92%–95%	<92%

Pulmonary findings are as follows:

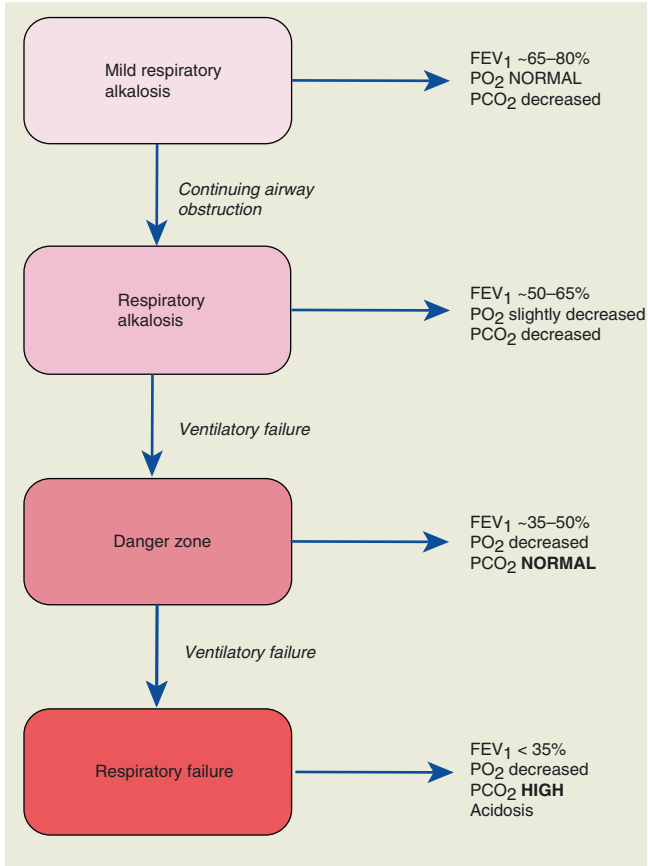
- Diffuse wheezes—Long, high-pitched sounds on expiration and, occasionally, on inspiration.
- Diffuse rhonchi—Short, high- or low-pitched squeaks or gurgles on inspiration and/or expiration.
- Bronchovesicular sounds.
- Expiratory phase of respiration equal to or more prominent than inspiratory phase (Table 11.3).

Signs of fatigue and near-respiratory arrest are as follows:

- Altered levels of consciousness, such as lethargy, which is a sign of respiratory acidosis and fatigue.
- Abdominal breathing.
- Inability to speak in complete sentences.

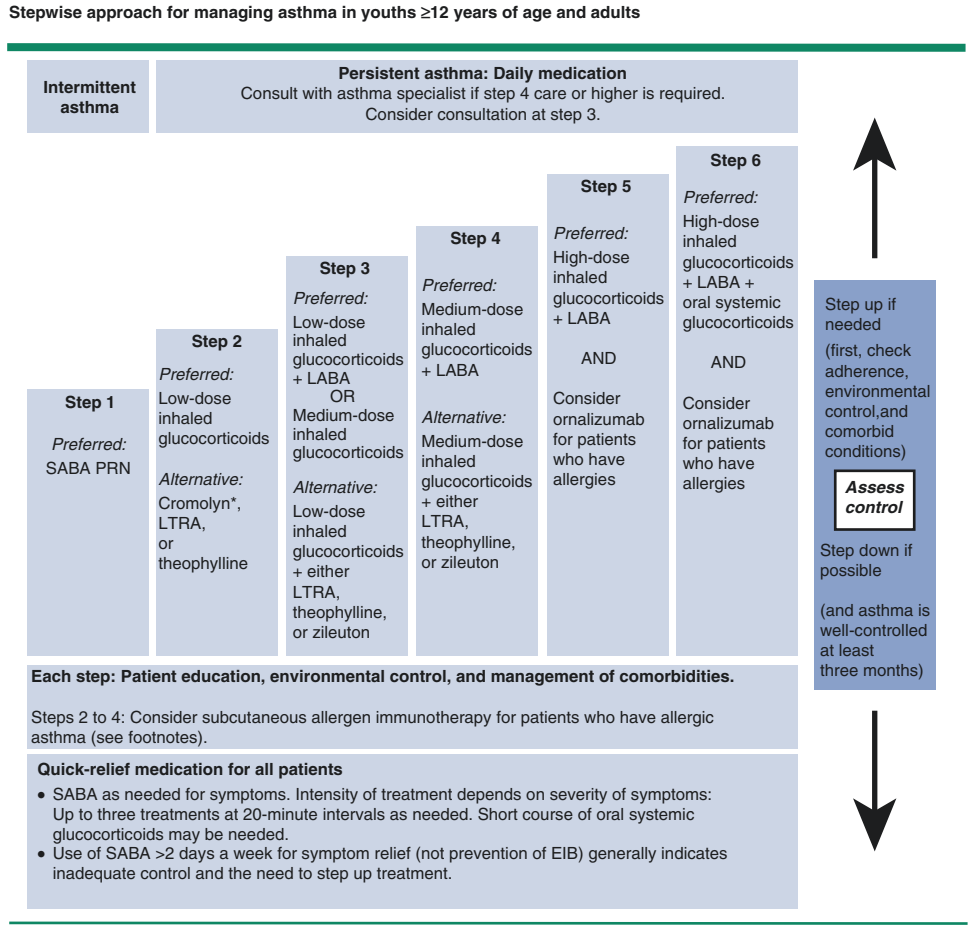
Signs of complicated asthma are as follows:

- Equality of breath sounds: Check for equality of breath sounds (pneumonia, mucous plugs, barotrauma). The amount of wheezing does not always correlate with the severity of the attack. A silent chest in someone in distress is more worrisome.
- Jugular venous distention from increased intrathoracic pressure (from a coexistent pneumothorax).
- Hypotension and tachycardia (think tension pneumothorax).
- Fever, a sign of upper or lower respiratory infections.



11.6 Adjustments to Pharmacologic Therapy in Pregnancy

The general principles of pharmacologic therapy for asthma during pregnancy are similar to those in nonpregnant patients and involve a stepwise approach to achieve and maintain asthma control, as recommended by national and international guidelines.



Preferred pharmacologic step therapy of asthma during pregnancy: NAEPP update 2004

Category	Step therapy
Mild intermittent	Inhaled short-acting beta2 agonist[a] as needed (for all categories)
Mild persistent	Low dose inhaled glucocorticoid[b]
Moderate persistent	Medium dose inhaled glucocorticoid[b]
	OR
	Low dose inhaled glucocorticoid[b] plus long-acting beta agonist[c]
	OR
	Medium dose inhaled glucocorticoid[b] plus long-acting beta agonist[c], if needed
Severe persistent	High dose inhaled glucocorticoid[b] plus long-acting beta agonist[c]
	Prednisone if needed

Based on data from: Quick Reference. NAEPP Expert Panel Report Managing Asthma During Pregnancy: Recommendations for Pharmacologic Treatment-Update 2004. US Dept of Health and Human Services, Bethesda, MD. NIH Publication No. 04-5246, March, 2004

[a]Albuterol is preferred inhaled short acting beta2 agonist during pregnancy

[b]Budesonide is preferred inhaled corticosteroid during pregnancy

[c]Salmeterol is preferred long-acting beta agonist during pregnancy

Long-acting bronchodilators: Implications for pregnancy and lactation

Agent	Human data	Adverse events in animal studies	Crosses placenta	Excreted in human milk
Long-acting beta agonists[a]				
Salmeterol	Limited data suggest safety	Observed in some studies		Unknown
Formoterol	Limited data suggest safety	Observed in some studies		Unknown
Indacaterol[b]		No		Unknown
Olodaterol[b]		Yes		Unknown, but likely
Vilanterol[c]		No		Unknown
Long-acting anticholinergic agents				
Aclidinium[b]		Yes	Not known	Probable
Glycopyrrolate[b]		Yes	Small amounts	Yes
Tiotropium		Yes	Not known	Not known
Umeclidinium[b]			Negligible systemic absorption following oral inhalation	Not known

Current guidelines emphasize the following points:

- Albuterol is recommended as the shortacting beta-agonist of choice.
- For patients with mild persistent or more severe asthma, inhaled glucocorticoids reduce exacerbations during pregnancy, and cessation of inhaled glucocorticoids during pregnancy increases the risk of an exacerbation. Budesonide is the preferred inhaled glucocorticoid for use during pregnancy, as more published gestational human data are available for that medication. However, other inhaled glucocorticoids could be continued if the patient was wellcontrolled on one of these medications prior to pregnancy, and data for fluticasone have been reassuring regarding low birth weight (<2500 grams), small for gestational age (<10% of expected for gestational age), preterm birth (<37 weeks), and major congenital malformations.
- Salmeterol has been recommended as the inhaled longacting beta-agonist of choice in the United States due to the longer duration of clinical experience with this agent compared with formoterol. However, retrospective cohort studies provide reassuring data for both salmeterol and formoterol.
- Montelukast or zafirlukast could be considered as alternative but *not* preferred therapy for mild persistent asthma or as addon therapy to inhaled glucocorticoids, especially for patients who have shown a uniquely favorable response prior to pregnancy. More pregnancy data are available for montelukast than zafirlukast.

11.7 Safety of Specific Medications

Experience with many of the medications used to treat asthma suggests minimal or no known adverse effects for their use during pregnancy. Most drugs used in the treatment of asthma fall into categories B or C.

Drug ratings in pregnancy (US Food and Drug Administration)

Category	Interpretation
A	Controlled human studies show no risk Controlled studies in pregnant women fail to demonstrate a risk to the fetus in the first trimester with no evidence of risk in later trimesters. The possibility of fetal harm appears remote.
B	No evidence of risk in studies Either animal reproduction studies have not demonstrated a fetal risk but there are no controlled studies in pregnant women or animal reproduction studies have shown an adverse effect (other than a decrease in fertility) that was not confirmed in controlled studies in women in the first trimester and there is no evidence of a risk in later trimesters.
C	Risk cannot be ruled out Either studies in animals have revealed adverse effects on the fetus (teratogenic or embryocidal effects or other) and there are no controlled studies in women or studies in women and animals are not available. Drugs should be given only if the potential benefits justify the potential risk to the fetus.
D	Positive evidence of risk There is positive evidence of human fetal risk, but the benefits from use in pregnant women may be acceptable despite the risk (eg, if the drug is needed in a life-threatening situation or for a serious disease for which safer drugs cannot be used or are ineffective).
X	Contraindicated in pregnancy Studies in animals or human beings have demonstrated fetal abnormalities or there is evidence of fetal risk based on human experience, or both, and the risk of the use of the drug in pregnant women clearly outweighs any possible benefit. The drug is contraindicated in women who are or may become pregnant.

- *Shortacting betaadrenergic agonists*: The shortacting, selective beta2 adrenergic bronchodilators (**SABAs**) are used to provide quick relief of asthma symptoms and appear to be relatively safe during pregnancy. However, some casecontrol studies have suggested a slight increase in risk of certain infant abnormalities, viz., gastroschisis, cleft palate, cardiac defects, and autism.

  One problem with assessing the consequences of bronchodilator use in pregnancy is confounding introduced by indication; SABA use is a marker for poorly controlled asthma and more frequent exacerbations, which may independently contribute to the development of congenital anomalies. Furthermore, some studies only have access to data about prescriptions filled and not the frequency of actual use. Even if the statistical associations for relative risk are valid, the anomalies mentioned above are infrequent. Therefore, the absolute increase in risk is very small and, as noted earlier, less than the risk of poorly controlled maternal asthma.
- *Longacting betaadrenergic agents*: Clinical experience with inhalation of the longacting, selective beta2 adrenergic bronchodilators (**LABAs**) during pregnancy is less extensive than with the SABAs. Salmeterol is not expected to increase the risk of congenital anomalies, based on data from animal studies and limited human experience. Animal studies are also reassuring for formoterol, although data from human pregnancies are limited.

  Human safety data for newer LABAs, such as indacaterol, olodaterol, and vilanterol are lacking.

  When comparing a combination LABA plus inhaled glucocorticoid versus monotherapy with a higher dose of the inhaled glucocorticoid, the risk of congenital malformations appears similar. In a study of 1302 pregnant women with asthma, the odds ratio for a major congenital malformation was not increased (OR 1.1, 95% CI 0.6–1.9) when a LABA plus low-dose inhaled glucocorticoid was compared with a medium-dose inhaled glucocorticoid or when a LABA plus mediumdose inhaled glucocorticoid was compared with a highdose inhaled glucocorticoid (OR 1.2, 95% CI 0.5–2.7).
- *Oral/systemic glucocorticoids*: Systemic glucocorticoids have been used fairly extensively during pregnancy to treat asthma exacerbations and rarely for control of severe asthma. For each pregnant woman, the potential risks of gestational oral glucocorticoids must be

balanced against the risks to the mother or infant of inadequately treated asthma. As the risks of severe uncontrolled asthma include maternal or fetal mortality, these risks are considered to be greater than the potential risk of systemic glucocorticoids. Thus, oral glucocorticoids should be used during pregnancy when indicated for the management of severe asthma.

The potential areas of concern that have been raised with systemic glucocorticoids are congenital malformations (primarily cleft palate), preeclampsia, gestational diabetes, low birth weight, a slightly increased risk of prematurity, and neonatal adrenal insufficiency.

Congenital malformations: Data from animal studies in several species suggest that high-dose systemic glucocorticoids may lead to cleft palate. Palatal closure is usually complete by the 12th week of pregnancy, so potential risk would be limited to administration during the first trimester. Human studies are less concerning, but a possible effect cannot be dismissed.

Neonatal adrenal insufficiency following maternal administration of glucocorticoids is distinctly unusual, probably because the nonhalogenated glucocorticoids are largely metabolized to inactive metabolites by the placenta.

- *Inhaled glucocorticoids*: In contrast to oral/systemic glucocorticoids, the safety data on inhaled glucocorticoids are reassuring including many population-based studies.
- *Anticholinergic agents*: (also known as antimuscarinic agents), such as ipratropium, glycopyrrolate, and tiotropium, are not generally used as a primary form of therapy for asthma. However, questions may arise about their safety during pregnancy.

  Fetal tachycardia can occur with the systemic administration of atropine to the mother; however, the minimal chronotropic effect of inhaled ipratropium in the mother suggests that the inhaled preparation should have negligible chronotropic effects on the fetus. Gestational animal studies are also reassuring for ipratropium. Consequently, inhaled ipratropium, the most commonly used drug in this category, is felt to be safe during pregnancy.

  The safety of inhaled tiotropium, aclidinium, glycopyrrolate, and umeclidinium during pregnancy is uncertain as adverse effects were reported in animal studies, and human fetal outcomes have not been reported.
- *Leukotriene modifiers*: Zafirlukast and montelukast (leukotriene receptor antagonists) and zileuton (a 5lipoxygenase inhibitor) are agents that affect leukotriene synthesis or action. We suggest use of montelukast or zafirlukast, in preference to zileuton, and would reserve these agents for addon therapy to inhaled glucocorticoids, especially in patients who had a good response to this medication prior to pregnancy.

  Accumulating evidence for montelukast and zafirlukast is reassuring, although limited.
- *Antiimmunoglobulin E*: Omalizumab is a humanized, recombinant IgG1, monoclonal antiimmunoglobulin E antibody approved for addon therapy in patients with moderate to severe asthma that is inadequately controlled despite appropriate use of inhaled glucocorticoids. Studies of the safety of omalizumab in pregnancy are limited, although available data are reassuring. Immunoglobulin G molecules, such as omalizumab, are known to cross the placenta.

  The initiation of omalizumab during pregnancy is not recommended, although if a woman becomes pregnant while receiving omalizumab, it is suggested that therapy can be continued if the benefits are estimated to outweigh the potential harms.
- *Rarely used medications*: Methylxanthines and cromoglycates are rarely used in the management of asthma due to the availability of alternative agents with greater effectiveness and ease of use.

  Methylxanthines: The clinical use of methylxanthines (theophylline, aminophylline) during pregnancy is limited because of the potential for altered metabolism during pregnancy, the need for drug level monitoring, and

the potential for fetal tachycardia and irritability at the time of delivery. Moreover, inhaled glucocorticoids have been shown to be more effective than theophylline for persistent asthma in nonpregnant patients and at least as effective as theophylline with fewer side effects. Extensive clinical experience suggests that theophylline does not increase the risk of fetal anomalies.

Methylxanthine binding to albumin and hepatic clearance are altered during pregnancy, necessitating careful assessment of serum levels and adjustments to dosing over the course of pregnancy.

Methylxanthines are transferred across the placenta, leading to theophylline concentrations in neonatal and cord blood that are similar to those in maternal blood. Transient tachycardia and irritability have been reported in some neonates of mothers receiving methylxanthines.

Cromoglycates: The availability of the cromolyn sodium and nedocromil is limited and varies from one country to another.

Animal and limited human data on use during pregnancy have not demonstrated an increase in fetal malformations or other adverse effects with cromolyn sodium.

Nonpharmacologic Treatments: The main nonpharmacologic interventions to maintain asthma control during pregnancy are patient education, avoidance of irritant (e.g., cigarette smoke), and control of allergenic triggers of asthma.

11.8 Acute Exacerbations

Acute asthma exacerbations are common during pregnancy and increase the risk of preeclampsia, gestational diabetes, placental abruption, and placenta previa. The recommended pharmacotherapy of acute asthma during pregnancy does not differ substantially from the management in nonpregnant patients. Intensive monitoring of both mother and fetus is essential.

- Maternal monitoring:
  - Continuous measurement of oxygen saturation by pulse oximetry (SpO_2) is prudent, aiming for a $SpO_2 \geq 95\%$.
  - Measurement of expiratory airflow with a peak flow meter (or spirometer) is the best method for objective assessment of the severity of an asthma attack.
  - ($PaCO_2$) >35 mmHg or an arterial oxygen tension (PaO_2) <70 mmHg associated with acute asthma represents more severe compromise during pregnancy than in the nongravid state.
- Fetal monitoring: Fetal heart rate monitoring is the best available method for determining whether the fetus is adequately oxygenated.
- Maternal positioning: In general, pregnant patients with acute asthma should rest in a seated or lateral position, rather than supine, particularly in the third trimester, to avoid aortocaval compression by the gravid uterus.
- Hydration: Intravenous fluids are not necessary unless the patient is unable to maintain oral hydration.
- Supplemental oxygen: Supplemental oxygen (initially 3–4 L/min by nasal cannula) should be administered, adjusting the fraction of inspired oxygen (FiO_2) to maintain a PaO_2 of at least 70 mmHg and/or oxygen saturation by pulse oximetry of 95% or greater.
- Respiratory infections: Most respiratory infections that trigger an exacerbation of asthma are viral rather than bacterial and do not require antibiotic therapy (Table 11.4).

11.9 Medications for Management of Acute Asthma Exacerbations

The recommended agents for management of acute asthma exacerbations in pregnant patients are the same as for asthma exacerbations in nonpregnant adults and adolescents. These agents include inhaled shortacting beta-agonists, inhaled anticholinergic agents, oral or intravenous glucocorticoids, and, if appropriate, intravenous magnesium sulfate.

Table 11.4 Therapeutic options in acute asthma

Acute asthma medications	
Oxygen	Initially 100%, titrate to pulse oximetry > 93%
Beta-agonists	Albuterol (inhaled) Adults—0.5 mg every 20 min × 3 Children—0.15 mg/kg every 20 min × 3 (Consider continuous nebulization if severe)
Corticosteroids	Prednisone Adults—60 mg orally Children—1 mg/kg orally (Consider 2mg/kg méthylprednisolone IV if severe)
Anticholinergics	Ipratropium Adults—0.5 mg every 30 min × 3 Children—0.25 mg every 30 min × 3
Additional therapeutic options for severe exacerbations	
Epinephrine	Adults—0.3 to 0.5 mg SQ every 20mm × 3 Children—0.01 mg/kg SQ every 20 min × 3 (Consider 0.1 mg IV every 30 min in near-arrest states)
Magnesium sulfate	Adults—2 g IV Children—40 mg/kg IV
Xanthines	Aminophylline Adults—Loading dose 6 mg/kg IV, followed by infusion of 0.9 mg/kg/h Children—Loading dose of 7.0 mg/kg IV, followed by 0.5–0.8 mg/kg/h
Heliox	80% helium/20% oxygen, titrate up to 50% helium/50% oxygen to maintain pulse oximetry > 93%.
Anesthetics	Ketamine Adults/children—Loading dose 0.2 mg/kg IV, followed by 0.5 mg/kg/h × 2 h

- *Systemic glucocorticoids*: The indications for systemic glucocorticoids are the same for pregnant patients experiencing an asthma exacerbation, as for nonpregnant patients. The benefits of oral glucocorticoids in preventing exacerbations from becoming lifethreatening asthma outweigh any risk to the mother or fetus.
- *Ipratropium*: It is often used to treat severe acute asthma exacerbations as inhaled ipratropium is felt to be safe during pregnancy.
- *Intravenous magnesium sulfate*: Intravenous magnesium sulfate may be beneficial in acute severe asthma as an adjunct to inhaled beta-agonists and intravenous glucocorticoids.
- *Parenteral betaagonists*: Parenteral beta-agonists are rarely needed for asthma exacerbations. Due to theoretic concerns that the alpha-adrenergic effects of epinephrine might cause vasoconstriction in the uteroplacental circulation, the Working Group on Pregnancy and Asthma recommended that epinephrine generally be avoided during pregnancy except in the setting of anaphylaxis. For the rare patient who requires use of a systemic betaagonist to treat asthma, subcutaneous administration of terbutaline is a reasonable choice.
- *Intravenous aminophylline/theophylline* is *not* generally recommended for use in the emergency management of acute gestational asthma because aminophylline/theophylline provides no additional benefit to optimal inhaled beta-agonist and intravenous glucocorticoid therapy. In addition, when used in combination with intensive inhaled beta-agonist therapy, intravenous aminophylline causes increased adverse side effects.

Pharmacologie management of acute asthma exacerbations during pregnancy

1. $Beta_2$-agonist bronchodilator (nebulized or metered-dose inhaler)
Albuterol by MDI 4–8 puffs every 20 min up to 1 h, then every 1–4 h, as needed
Albuterol by nebulizer 0.083% (2.5 mg/3 mL), 2.5–5 mg every 20 min for 3 doses and then 2.5–5 mg every 14 h, as needed
Albuterol by continuous nebulization, administering 10–15 mg/h
2. Ipratropium
By nebulizer, 500 mg every 20 min for 3 doses, then as needed. Can be given simultaneously with $beta_2$-agonist.
By MDI, 4–8 inhalations every 20 min for 3 doses, then as needed
3. Systemic glucocorticoids (for those with a poor response to treatment after one hour, or with initial therapy for patients on chronic oral glucocorticoids)
For patients who can be managed at home: prednisone 40–60 mg/day in a single or divided dose

	For patients who require hospitalization: prednisone 40–80 mg daily in a single or divided dose (or the equivalent dose of methylprednisolone* intravenously) until peak flow reaches 70% of predicted or personal best, and then taperas patient improves
	For patients who have a life-threatening exacerbation, a higher initial dose of methylprednisolone*, 60–80 mg every 6–12 hours, may be given intravenously, and then tapered as the patient improves, as above
4.	For patients not responding to above therapies, consider adjunct therapies
	Intravenous magnesium sulfate 2 g infused over 20 min, in absence of renal insufficiency^
	Subcutaneous terbutaline 0.25 mg every 20 min for up to 3 doses

- *A follow-up appointment 2–4 days following the emergency room visit is recommended.*
- *Consider referral to an asthma specialist; in addition, involvement of a multidisciplinary team that includes a pulmonologist, neonatologist, obstetrician, and possibly an allergologist should be considered in the follow-up of a pregnant asthma woman.*

11.10 Care in Labor

- Oxytocin is the drug of choice for induction of labor and control of postpartum hemorrhage.
- Analogs of prostaglandin F2alpha can cause bronchoconstriction and should not be used for termination of pregnancy, cervical ripening, induction of labor, or control of uterine hemorrhage.
- Prostaglandin E2 (in gel or suppository form) and prostaglandin E1 (misoprostol) have not been reported to cause bronchoconstriction and are safer analogs if prostaglandin treatment is required.
- For peripartum pain control, butorphanol or fentanyl may be appropriate alternatives as morphine and meperidine should be avoided, if possible.
- Epidural anesthesia is preferred for the asthmatic patient who opts for pain control during labor because it reduces oxygen consumption and minute ventilation in the first and second stage of labor and usually can provide adequate anesthesia if cesarean delivery becomes necessary.
- In the absence of acute severe asthma, reserve cesarean section for the usual obstetric indications.
- If anesthesia is required, regional blockade is preferable to general anesthesia in women with asthma.
- If general anesthesia is required, ketamine and halogenated anesthetics are preferred, because they may have a bronchodilatory effect.
- Use of ergot derivatives for postpartum bleeding or headache should be avoided because of their potential to cause bronchoconstriction.
- If high doses of SABA have been given during labor and delivery, blood glucose levels should be monitored in the baby (especially if preterm) for the first 24 h.
- Women receiving steroid tablets at a dose exceeding prednisolone 7.5 mg/day for more than 2 weeks prior to delivery should receive parenteral hydrocortisone 100 mg 6–8 h during labor.

Fever in Pregnancy

12

Reena Wani and Rashmi Jalvee

12.1 Introduction and Definitions

Fever in pregnancy refers to elevated temperature above the normal variation, often associated with other symptoms.

RCOG defines *maternal pyrexia* as temperature of 38.0 °C once or 37.5 °C on two occasions 2 h apart [1].

Pyrexia is both a symptom and sign which should be taken seriously as it can have dire maternal and fetal consequences. Maternal fever in pregnancy or labor is primarily a cause for concern as we need to deal with two patients, mother and fetus [2]. The duration of fever, the time of occurrence during gestation, and the maximum temperature reached determine the fetal effects irrespective of the cause of fever.

Sepsis may be defined as infection plus systemic manifestations of infection.

Septic shock is defined as the persistence of hypoperfusion despite adequate fluid replacement therapy [3].

R. Wani (✉)
Dept of Obstetrics & Gynecology, HBT Medical College & Dr R N Cooper Municipal Hospital, Mumbai, India

R. Jalvee
HBT Medical College & Dr R N Cooper Hospital, Mumbai, India

12.2 Causes of Fever

Type of infection	Causes
Systemic infection	
Viral	Influenza, rubella, CMV, herpes
Bacterial	GAS, *E. coli*, *Streptococcus*, *Staphylococcus*, toxic shock syndrome, *Pseudomonas* species
Protozoal	Malaria, toxoplasmosis, amoebiasis
Organ-specific infection	
UTI	Cystitis, pyelonephritis
Respiratory	Pneumonia, bronchitis, TB, influenza
Uterine	Chorioamnionitis, STDs
Gastrointestinal	Hepatitis, pancreatitis, enteritis, appendicitis
Cardiac	Subacute bacterial endocarditis
Neurological	Meningitis, malaria, amoebiasis
Noninfectious causes	
Connective tissue disorders	Rheumatoid arthritis
Autoimmune disorders	SLE, inflammatory bowel disease
Drugs	Procainamide, alpha-methyldopa, isoniazid
Sickle cell crisis	Bone crisis, acute chest syndrome, abdominal crisis, joint crisis
Endocrine/ metabolic causes	Diabetic ketoacidosis, pheochromocytoma Hyperthyroidism
Thrombosis	DVT, pulmonary embolism
Malignancy	Leukemia, lymphoma
Pyrexia of unknown origin	

(continued)

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Type of infection	Causes
Postpartum causes	
Genital tract infection	Endometritis, pelvic abscess
UTI	Cystitis, pyelonephritis
Breast	Engorgement, acute mastitis, breast abscess
Wound infection	Caesarean section, episiotomy, perineal tear

Any acute or chronic infectious disease may be contracted during pregnancy, and conception may occur in women already suffering from infection.

12.3 Clinical Features

Fever may be accompanied by any of the following symptoms which warrant further investigations as it may indicate an important disease which could leave an impact on both mother and fetus:

- Skin rash.
- Difficulty in breathing or shortness of breath.
- Persistent cough.
- Persistent diarrhea or vomiting.
- Jaundice.
- Bruising or unusual bleeding.
- Decreased consciousness.

Risk factors for maternal sepsis in pregnancy are [4]:

- Obesity.
- Impaired glucose tolerance/diabetes.
- Impaired immunity/immunosuppressant medication.
- Anemia.
- Vaginal discharge.
- History of pelvic infection.
- History of group B streptococcal infection.
- Amniocentesis and other invasive procedures.
- Cervical cerclage.
- Prolonged spontaneous rupture of membranes.
- GAS infection in close contacts/family members.
- Of black or other minority ethnic group origin.

12.4 Clinical Approach

The clinical approach to examination of pregnant women with fever should be directed toward:

- Assessing maternal condition and stability.
- Ascertaining fetal viability and well-being.
- Directing investigations based on clinical findings.

12.4.1 Assessing Maternal Condition and Stability

- **Detailed history**: about the degree of fever, pattern of fever, and progression of symptoms should be noted. The pattern of fever is typical in certain etiologies (e.g., quotidian, tertian, or quartan fever in malaria, evening rise of fever in tuberculosis, etc.) However, the typical patterns may not always hold true if antipyretic or antibiotic use has been initiated before presentation to the doctor.
- *Clinical signs*:
  - General well-being, sensorium, and higher functions.
  - Evidence of dehydration.
  - Pallor.
  - Oxygen saturation.
  - Pulse rate.
  - Blood pressure.
  - Respiratory rate.
  - Chest auscultation findings.
  - Abdominal findings: presence of tenderness, organomegaly.
  - Presence of rash.
- *Clinical signs suggestive of sepsis* include pyrexia, hypothermia, tachycardia, tachypnea, hypoxia, hypotension, oliguria, impaired consciousness, and failure to respond to treatment. These signs, including pyrexia, may not always be present and are not necessarily related to the severity of sepsis.
- *Need for inpatient admission and intensive care* should be based on the general condition and vital parameters, on the possible diagnosis, and on the basis of investigations.

The following signs and symptoms should prompt indoor admission [4]:
  - Woman appears seriously unwell.
  - Pyrexia more than 38°.
  - Sustained tachycardia more than 90 beats/min.
  - Breathlessness (respiratory rate more than 20 breaths/min).
  - Abdominal or chest pain.
  - Diarrhea and/or vomiting.
  - Uterine or renal angle pain and tenderness.
- *Determine if the patient has gone into labor* based on history and assess for presence of uterine contractions, fetal heart rate, per vaginal findings of cervical dilatation, descent, membranes, liquor, and meconium.
- Need for consultation with a physician, surgeon, or other specialists depending on the symptomatology and vital parameters.

12.4.2 Fetal Viability and Well-Being

Assessment of fetal well-being should be done as per the gestational age.

For patients in the third trimester presenting in the labor room, this includes:

- Assessment of uterine size, fundal height, lie and presentation, and presence of uterine activity.
- Auscultation of fetal heartbeat and electronic fetal monitoring.
- Ultrasound or confirming viability, fetal weight, liquor, and biophysical profile.
  In most cases, the fetal status is not affected. However in vector-borne diseases like malaria and dengue, we have found sudden unexplained intrauterine demise occurring which could be related to hypoglycemia or metabolic disturbances. Hence for pregnancies at term, it is a challenge to determine whether to wait or to deliver the patient.

12.4.3 Investigations

Types of investigations to be performed are based on the history and clinical examination.

The aims of performing investigations are:

- To confirm the diagnosis.
- To monitor the response to treatment.

The basic investigations advised are:

- Complete blood count with differential count.
- Peripheral smear.
- RFT, LFT, serum electrolytes.
- C-reactive protein.
- Urine routine
  These can often give us clues to the diagnosis, for example:
  - Urine pus cells and protein in UTI.
  - Falling platelet count in dengue fever.
  - Differential count suggesting neutrophilic leukocytosis (? bacterial) or lymphocytosis (? Kochs) and leucopenia in viral infections.

Depending upon the symptoms, signs, and the organ system involved, other investigations indicated are the following:

- Swabs—high vaginal swab, wound, placenta (depending on the history).
- Tests for infections: malaria, dengue, hepatitis, etc.
- Other relevant samples—sputum, throat swab, stool.
- Blood cultures if temperature >38 C (chorioamnionitis, septicemia).
- Appropriate cultures should be obtained before initiating antibiotic therapy but should not prevent prompt administration of antimicrobial therapy.
- Chest X-ray (abdominal shield prior to delivery—high index of suspicion needed in India).
- Sputum for microscopy and culture.
- Urine for ketones.
- Blood gases (diabetic ketoacidosis, septicemia).
- Abdominal ultrasound—pelvic abscess, pyometra.
- Doppler—DVT (deep vein thrombosis not common but a specific treatable cause).
- Lumber puncture—meningitis, encephalitis.

12.5 Management [1]

Management of fever depends on the cause.

- To control temperature:
  - Fanning, sponging.
  - Antipyretics like paracetamol 1 g orally or intravenously may be used although the possibility of masking underlying sepsis should not be forgotten.
  - If temperature remains ≥38 C for 30 min, antibiotics should be started.
- Intravenous rehydration should be given if there is evidence of dehydration. Look for skin turgor, urine output, and presence of urine ketones.
- Isolation of patients in the presence of fever with rash (maculopapular rashes caused by rubella, parvovirus, and measles and vesicular rash caused by chicken pox), swine flu. Referral to infectious disease unit, steroids, or ICU care may be needed. Delivery is usually to be delayed till rash subsides or crusts over such that maternal IgG antibodies have come into circulation which are protective to the fetus.
- To treat infections:
  - Intravenous broad-spectrum antibiotics—to be commenced as soon as possible.
  - Inj Co-amoxiclav 1.2 g/Inj cefuroxime 1.5 g and Inj metronidazole 500 mg 8 h.
    In cases of allergy to penicillin and cephalosporins, inj clindamycin 1.5 g 8 h/inj clarithromycin 500 mg 12 h until delivery, and inj gentamicin 3–5 mg/kg daily in divided doses.
  - Blood culture and other sample for culture and sensitivity should be taken before starting antibiotics.
  - Intravenous antibiotics must be continued during labor and in the immediate postpartum period.
  - After intravenous antibiotics, a maximum of 5 days of oral antibiotics is usually sufficient.
- Other infections should be managed based on the etiology (antimalarials, etc.)
- Delivery
  - Induction of labor can be considered in certain cases if fetal well-being is a concern; however maternal health and parameters are to be considered carefully.
  - Labor should be augmented when patient presents with established pains as prolonged labor and high maternal temperature have detrimental effects on the baby. Caesarean section is reserved for obstetric causes.
  - Continuous EFM is important to assess fetal well-being.
  - Pediatrician should be available to attend at delivery.
- Surgical treatment may be necessary in certain conditions as listed below. Senior consultant needs to be involved in decision-making, and appropriate multidisciplinary team involvement may be necessary [1]
  - Retained placenta.
  - Adnexal accidents.
  - Pelvic abscess.
  - Breast abscess.
  - Acute appendicitis.
- Placenta swab for C & S and placenta for histology should be sent to obtain the definitive diagnosis.

12.6 Summary

Common etiologies and clinical approach to diagnosis of fever in pregnancy [5]

Fever with obstetric features (amniotic fluid leak, long-standing fetal death, uterine pain, or tenderness) present	Fever with site-specific features predominating the clinical picture	Fever with systemic features predominating, general condition is affected	Fever in typical patterns (not commonly seen when treatments are initiated early, antipyretics are used)	Fever with rash
Chorioamnionitis	Respiratory infections	Viral fever	Malaria—tertian or quotidian	Measles
Puerperal sepsis	Urinary infections	Malaria	TB—evening rise of temperature	Rubella
	Gastroenteritis	Dengue	Typhoid—step ladder pattern	Chicken pox
	Mastitis or breast abscess	Hepatitis	Puerperal sepsis, iliac vein thrombophlebitis—hectic temperature patterns	Herpes simplex
	Meningitis or cerebral malaria	Leptospirosis		Parvovirus
	Hepatitis			

12.6.1 Principles of Management

- Attempt to find specific etiology and treat if possible.
  Avoid hypoglycemia.
- Be vigilant for sudden changes in maternal status which can affect fetal well-being.
- Control of pyrexia.
- Deliver in the best way at the best time keeping in mind maternal and fetal factors.

References

1. Pyrexia in Pregnancy, Emergencies in obstetrics and gynaecology, S. Arulkumaran; 2006.
2. Editorial: fever in pregnancy: too hot to handle? In: Reena W (ed). Handbook for practicing obstetricians. CBS Publication; 2015.
3. Dellinger RP, Levy MM, Carlet JM, Bion J, Parker MM, Jaeschke R, et al. Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock. Crit Care Med. 2008;36:296–327.
4. Centre for Maternal and Child Enquiries (CMACE). Saving Mother's Lives: reviewing maternal deaths to make motherhood safer: 2006-2008. BJOG. 2011;118(suppl. 1):1–203.
5. Clinical Clues: fever with and without rash. Tank P D. In: Wani R. (ed) Fever in pregnancy: handbook for obstetricians. CBS Publication; 2015.

Epilepsy

13

J. B. Sharma and Monica Gupta

13.1 Introduction

Epilepsy is one of the most common neurological complications of pregnancy with a prevalence of 0.5–1% [1]. Epilepsy is a chronic disorder and is defined as one or more recurrent unprovoked seizures. Pregnant women with known epilepsy are advised to continue anti-epileptic medication to avoid maternal and foetal complication due to seizure. The goal of treatment is optimal control of seizure and minimal exposure of foetus to anti-epileptic medication. Various physiological, endocrine and psychological changes contribute to increase in seizure frequency during pregnancy. Most crucial to management is determining exact aetiology. Prompt and stepwise management of these patients in a multidisciplinary team involving obstetrician, gynaecologist and neurologist can prevent fatal complications to mother and foetus.

Seizure for the first time during pregnancy can have multiple differentials listed in Table 13.1. Management of a pregnant women presenting with epileptic fit involves a detailed history, physical examination, appropriate investigation and prompt treatment for seizure control. History taking is the most important tool in diagnosing seizure and should focus on symptoms during and after the seizure episode. Other associated factors to be taken into account include previous history of seizure, brain tumour or trauma, stroke, precipitating events (alcohol or medications) and past obstetrical history. Symptoms during seizure are usually described by an eyewitness and include presence of aura, changes in respiration, altered consciousness, bowel/bladder dysfunction and generalised or focal tonic-clonic movements. Seizure episode is usually followed by amnesia, weakness, headache, body aches and drowsiness. Physical examination includes pulse and blood pressure measurement and thorough

Table 13.1 Differential of first seizure during pregnancy

First trimester
Metabolic alterations (hypoglycaemia, hyponatraemia and hypocalcaemia)
Drug overdose or withdrawal
Second trimester
Pregnancy-related syncope (peripheral vasodilatation, fall in blood pressure)
Third trimester
Eclampsia
Posterior reversible encephalopathy syndrome
Stroke
All trimester
Mass lesion
Infection
Vascular malformation

J. B. Sharma (✉) · M. Gupta
Department of Obstetrics & Gynaecology, All India Institute of Medical Sciences, New Delhi, India

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neurological examination including optic fundi. Ancillary investigations include electroencephalography (EEG), imaging of the brain and laboratory studies. EEG is safe during pregnancy and is the recommended initial neurodiagnostic investigation. It is found to be normal in 50% of cases, and abnormality indicates risk of seizure recurrence and helps guide therapy. The American Academy of Neurology and the American Society of Epilepsy recommend computed tomography (CT) and magnetic resonance imaging (MRI) of the brain as initial neurodiagnostic tests to help determine underlying aetiology [2]. CT of the brain delivers <1 rad to foetus, and exposure below 5 rad is not associated with increased risk of foetal anomalies or pregnancy loss [3]. MRI has no radiation exposure and is more sensitive than CT but is costly and not readily available. Laboratory studies include full blood count, serum urea and electrolytes, blood sugars and urine protein estimation and toxicology screen.

13.2 Classification

The International Classification of Epileptic Seizures categorises seizures into two broad groups (International League against Epilepsy [4]):

1. Partial seizures are due to initial activation of neurons in one hemisphere. They can be further subdivided into simple or complex:
   (a) Simple partial seizure: Consciousness maintained during ictal phase.
   (b) Complex partial seizure: Impaired consciousness during seizure episode.
2. Generalised seizures: Arise due to activation of neurons in both hemispheres:
   (a) Convulsive: Presence of motor movements and impaired consciousness and can be myoclonic, clonic, tonic and tonic-clonic type.
   (b) Non-convulsive: Absence of motor concomitants.

13.3 Maternal and Foetal Complications

Epilepsy in pregnancy is a serious medical condition associated with both maternal and foetal complications enlisted in Table 13.2. Seizures especially status epilepticus during pregnancy and labour could be fatal for both foetus and mother. Pregnant women with epilepsy are advised to continue anti-epileptic medication to avoid serious maternal and foetal complications. The most important predictor of seizure during pregnancy is the occurrence of seizure prior to pregnancy. Women who had seizure in the month prior to pregnancy had a 15 times greater likelihood of having seizure during pregnancy. If the woman is seizure free for 9 months to 1 year prior to conception, 92% will not have seizure in pregnancy [5]. Polytherapy also increases risk of seizure during pregnancy [6]. Planned pregnancies have a lower likelihood of seizure during pregnancy as most of the women in this group are on monotherapy and not on valproate [7]. Various anti-epileptic drugs for particular seizure type are outlined in Table 13.3. Folic acid supplementation should be given to all women in reproductive age group to reduce risk of neural tube defects. Sodium valproate should be avoided in women of

Table 13.2 Maternal and foetal complications in women with epilepsy

Maternal complications
Recurrent seizures
Status epilepticus
Seizures during labour
Gestational hypertension
Preeclampsia
Maternal injury
Maternal death
Foetal and neonatal complications
Congenital malformation (2–3 times normal)
Miscarriage (2 times normal)
Hypoxia
Low birth weight
Small for gestational age
Foetal injury

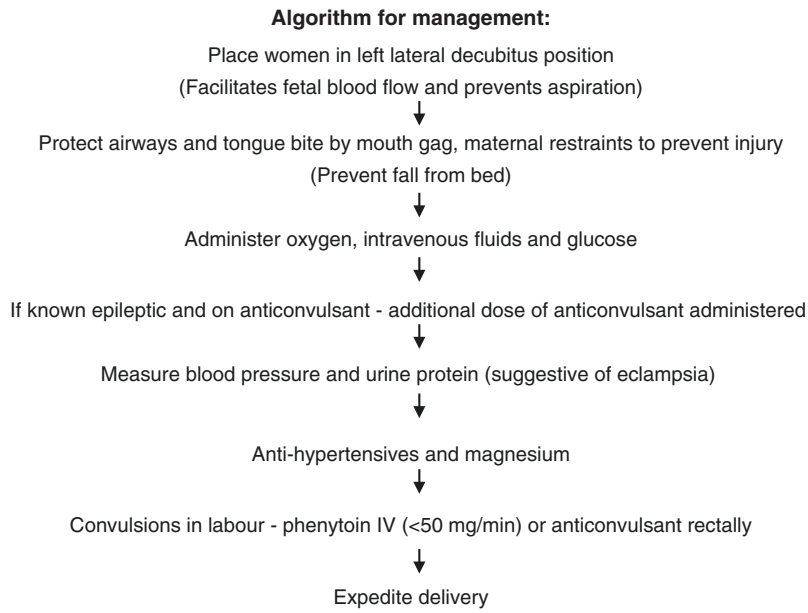
Table 13.3 Various anti-epileptic drugs for seizure type

Seizure type	First-line anti-epileptic drugs
Generalised tonic-clonic	Carbamazepine Oxcarbazepine Lamotrigine Sodium valproate
Tonic or atonic	Sodium valproate
Absence seizures	Ethosuximide Sodium valproate Lamotrigine
Focal	Carbamazepine Levetiracetam Lamotrigine Oxcarbazepine Sodium valproate
Convulsive status epilepticus	Intravenous lorazepam Intravenous diazepam Buccal midazolam
Refractory status epilepticus	Intravenous midazolam Propofol Thiopental sodium

reproductive age as it is associated with highest risk of congenital malformation up to 11% when given in pregnancy [8]. If other anti-epileptics have failed and seizure can be controlled only on valproate, lowest dose preferably less than 700 mg should be used. Lamotrigine and levetiracetam are preferred over others because of least teratogenicity. Physiological changes in pregnancy can alter seizure frequency during pregnancy. Moreover hyperventilation of pregnancy, sleep deprivation and labour pains can lower seizure threshold in pregnancy.

13.4 Management of Pregnant Women Presenting with Seizure

In the antenatal period, women should be regularly assessed at each visit for triggers of seizure including fasting, sleep deprivation and stress. Prompt treatment of seizure if it occurs during pregnancy is essential to avoid maternal and foetal risks. Neurologist should be consulted if there is deterioration in seizure control and dose adjustments made accordingly.



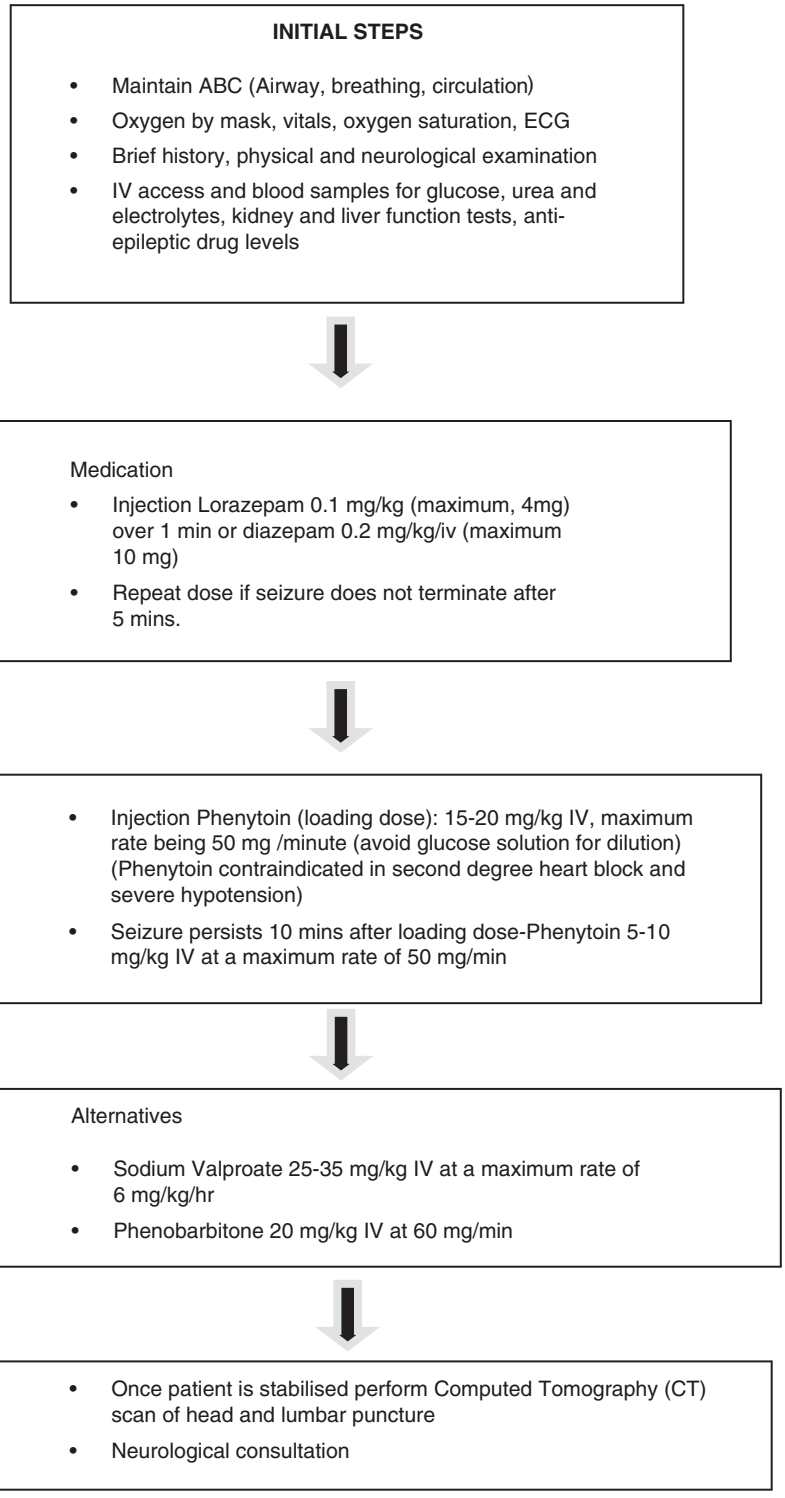
13.5 Management of Seizure in Labour

Convulsive status epilepticus during labour is rare and affects around 1% of women with epilepsy [9]. It is a life-threatening condition; hence precipitating factors should be avoided. If patient is on anti-epileptics, doses should not be missed during labour and delivery. Parenteral alternatives should be considered in case of excessive vomiting. One-to-one support of labouring patient minimises stress of labour and ensures safety in the event of a seizure attack. Adequate hydration and pain relief with epidural will minimise risks of seizures in labour. Left lateral tilt with oxygenation and airway should be maintained at all times. Treatment should be initiated as soon as possible to terminate seizure and prevent maternal and foetal hypoxia and foetal acidosis. Benzodiazepines are the drug of choice for convulsive status with lorazepam being the preferred drug in doses as described below [10]. Tocolytics are administered for persistent uterine hypertonus. Continuous electronic foetal monitoring is commenced once the mother is stabilised. If foetal heart rate deceleration persists beyond 5 min or seizures are refractory, then delivery should be expedited and caesarean performed if vaginal delivery is not imminent.

13.6 Status Epilepticus

Status epilepticus (SE) is the most serious labour room emergency in a patient with epilepsy. In this condition there is failure of normal mechanisms that serve to terminate seizures. It can be of the following types:

(a) Convulsive status epilepticus (CSE): Characterised by continuous convulsive seizures lasting for more than 5 min or two or more seizures with loss of consciousness in between.
(b) Non-convulsive status epilepticus (NCSE): Altered mental status from baseline lasting for at least 30 min and associated with EEG changes.
(c) Refractory status epilepticus: Seizure activity that persists despite first- and second-line anti-epileptics.


INITIAL STEPS
• Maintain ABC (Airway, breathing, circulation)
• Oxygen by mask, vitals, oxygen saturation, ECG
• Brief history, physical and neurological examination
• IV access and blood samples for glucose, urea and electrolytes, kidney and liver function tests, anti-epileptic drug levels
Medication
• Injection Lorazepam 0.1 mg/kg (maximum, 4mg) over 1 min or diazepam 0.2 mg/kg/iv (maximum 10 mg)
• Repeat dose if seizure does not terminate after 5 mins.
• Injection Phenytoin (loading dose): 15-20 mg/kg IV, maximum rate being 50 mg /minute (avoid glucose solution for dilution) (Phenytoin contraindicated in second degree heart block and severe hypotension)
• Seizure persists 10 mins after loading dose-Phenytoin 5-10 mg/kg IV at a maximum rate of 50 mg/min
Alternatives
• Sodium Valproate 25-35 mg/kg IV at a maximum rate of 6 mg/kg/hr
• Phenobarbitone 20 mg/kg IV at 60 mg/min
• Once patient is stabilised perform Computed Tomography (CT) scan of head and lumbar puncture
• Neurological consultation

Refractory Status Epilepticus (Lasting more than 60 min)

- Admit patient to intensive care unit.
- EEG monitoring.
- Central venous access.
- Mechanical ventilation.
- Anaesthetic agent: Midazolam 0.2 mg/kg IV (maximum 10 mg) bolus over 2 min followed by 0.1–0.4 mg/kg/h continuous IV infusion. Alternatively propofol 2–5 mg/kg IV bolus followed by 5–10 mg/kg/h IV infusion can be administered.
- Pharmacologic treatment is continued for 12 h after last seizure to maintain comatose state with EEG monitoring.
- Anaesthetic infusion is reduced every 3 h with EEG monitoring during weaning phase.

13.6.1 Treatment After Control of Seizure

If patient is known case of epilepsy, anti-epileptic drugs are continued and dose adjusted according to serum levels. In patients presenting for the first time with seizures, phenytoin or valproate started to control status epilepticus should be continued.

13.7 Conclusion

The management of pregnant women with epilepsy is a challenge for clinicians. The goal of treatment is adequate seizure control and minimal exposure of foetus to anti-epileptic drugs. Most pregnant females with epilepsy will have a successful outcome of pregnancy, but pre-pregnancy planning is necessary for optimum control of epilepsy. Increased risk periods during pregnancy for seizure attack are intrapartum and postpartum. Prompt treatment and termination of seizure are paramount for maternal and foetal well-being. Multidisciplinary approach is required for successful outcome of pregnancy.

References

1. Edey S, Moran N, Nashef L. SUDEP and epilepsy-related mortality in pregnancy. Epilepsia. 2014;55:e72–4.
2. Krumholz A, Wiebe S, Gronseth G, et al. Practice parameter: evaluating an apparent unprovoked first seizure in adults (an evidence-based review): report of the quality standards sub- committee of the American Academy of Neurology and the American Epilepsy Society. Neurology. 2007;69(21):1996–2007.
3. Practice ACoO. ACOG Committee Opinion. Number 299, September 2004 (replaces No.158, September 1995). Guidelines for diagnostic imaging during pregnancy. Obstet Gynecol. 2004;104(3):647–51.
4. Proposal for revised clinical and electroencephalographic classification of epileptic seizures. From the commission on classification and terminology of the international league against epilepsy. Epilepsia. 1981;22(4):489–501.
5. Harden CL, Hopp J, Ting TY, et al. Practice parameter update: management issues for women with epilepsy-focus on pregnancy (an evidence-based review): obstetrical complications and change in seizure frequency. Neurology. 2009;73:126–32.
6. Thomas SV, Syam U, Devi SJ. Predictors of seizures during pregnancy in women with epilepsy. Epilepsia. 2012;53(5):e85–8.
7. Abe K, Hamada H, Yamada T, Obato-Yasuoka M, Minakami H, Yoshikawa H. Impact of planning of pregnancy in women with epilepsy on seizure control during pregnancy and on maternal and neonatal outcomes. Seizure. 2014;23:112–6.
8. Cunnington MC, Weil JG, Messenheimer JA, et al. Final results from 18 years of the international lamotrigine pregnancy registry. Neurology. 2011;76:1817–23.
9. EURAP Study Group. Seizure control and treatment in pregnancy: observations from the EURAP 1129 epilepsy pregnancy registry. Neurology. 2006;66:354–60.
10. Alldredge BK, Gelb AM, Isaacs SM, Corry MD, Allen F, Ulrich S, et al. A comparison of lorazepam, 1134 diazepam, and placebo for the treatment of out-of-hospital status epilepticus. N Engl J Med. 2001;345:631–7.

Swine Flu and Pregnancy

14

Bindiya Gupta

14.1 H1N1/Swine Flu

The worldwide pandemic of swine flu (H1N1) was identified in Mexico in April 2009 and then spread to other parts of world [1]. The pandemic started in India in August 2009, and the index case was reported from Pune. It is referred to as the *novel H1N1 influenza A infection*, a term that reflects the unique genetic makeup of the virus. The new virus that emerged spread among people who hadn't been near pigs. It was as a result of reassortment (genetic shift) of several swine strains, a human strain, and an avian strain limiting the ability of the immune system to recognize and destroy the new virus giving rise to a pandemic-like situation [2].

It is like seasonal flu, but it can cause more serious health problems for some people like children, elderly, pregnant women, and immunocompromised individuals. Mode of spread is through droplet like any other flu. Patients are infective since 1 day before they have any symptoms till as many as 7 days after they get symptoms. Children can be contagious for as long as 10 days.

The signs and symptoms range from mild infection, afebrile illness to severe complicated pneumonia. They include fever, headache, cough, body aches, sore throat, nasal stuffiness, and gastrointestinal symptoms like vomiting and diarrhea [1]. Signs of severe infection include tachypnea (RR > 30), hypoxia (SpO_2 < 92%), chest pain on breathing, tachycardia (HR > 100), rigors, shock, dehydration and shock, purulent or blood-stained sputum, altered consciousness, or fever [3]. Severe infection warrants hospitalization and sometimes intensive care unit admission [3].

14.2 Pregnancy and Swine Flu

Pregnancy does not predispose women of acquiring influenza infection, but epidemiological studies show that the pregnant women have increased mortality and morbidity to influenza infection. Pregnant women have higher rate of hospital admission, requirement of mechanical ventilation, and higher mortality rate (up to seven times) especially in third trimester [4–7]. In a report by CDC, among 347 severely ill pregnant women, 75 died from 2009 H1N1, and 272 were admitted to an intensive care unit (ICU) and survived. Most of the women who died (62%) had an underlying medical condition like asthma, gestational diabetes, obesity, immune suppression, chronic lung, autoimmune diseases, etc. [8]

Increased fetal morbidity and mortality is also reported during both seasonal and pandemic influenza outbreaks. According to the CDC Pregnancy Flu Line surveillance data 2009, of the 168 pregnancy outcomes, 148 (88%) were live

B. Gupta (✉)
Department of Obstetrics and Gynecology, UCMS and GTB Hospital, New Delhi, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_14

births, 11 (7%) were spontaneous abortions, 7 (4%) were fetal deaths, 1 was an ectopic pregnancy, 1 was a 15-week elective abortion secondary to intrauterine growth restriction live births, 63.6% were born preterm or very preterm, 4.1% were small for gestational age, 43.8% had low birth weight, 69.4% were admitted to the neonatal intensive care unit, and 29.2% had a low 5-minute Apgar score [8]. Changes in the immune, cardiac, and respiratory systems during pregnancy are responsible for increased severity of influenza infection [9].

Pregnancy-related complications of novel H1N1 infection are related to high-grade fever. These include nonreassuring fetal heart rate, fetal tachycardia, febrile morbidity, spontaneous abortions, premature rupture of membranes, neonatal seizures, and intrauterine death [10].

14.3 Diagnosis

Samples for testing include throat and deep nose swabs, nasopharyngeal aspirates, tracheal aspirates, bronchoalveolar lavage (BAL), and sputum. A rapid influenza antigen test is used, but confirmation is done once the reverse transcription polymerase chain reaction (RT-PCR) or a culture is positive [11]. However for suspected patients (probable case), treatment should not be delayed pending the reports.

14.4 General Preventive Precautions

Hygiene is the key to prevent flu in pregnancy. Pregnant women should not travel to places endemic to influenza and should avoid crowded places. The general steps recommended to prevent infection are handwashing; avoiding contact with infected person; cough etiquette and hand hygiene; avoid touching the eyes, nose, and mouth; and carry alcohol-based hand rub [12]. Education of the pregnant woman and staff during the influenza season is recommended. The women should be made aware of the early signs and symptoms and importance of early access to medical care.

Symptomatic patients should be placed on droplet precautions (including gowns, gloves, and N95 respirators). Fever should be treated immediately, and the drug of choice is acetaminophen.

Staff should be trained to isolate individuals with potential influenza infection, and during periods of increased community influenza activity, facilities should consider setting up triage stations that facilitate rapid screening of patients for symptoms of influenza and separation from other patients [13]. No special precautions are needed in disposal of waste or linen.

14.5 Antiviral Drugs in Influenza

Early institution of prevention and treatment with antiviral agents is associated with improved outcomes for pregnant women. Oseltamivir (Tamiflu) is the most common antiviral drug used for prophylaxis and treatment of influenza in pregnancy. The mechanism of action is the competitive inhibition of the neuraminidase enzyme of the virus that acts on the sialic acid residues of the host cells [14]. The treatment should be initiated as soon as possible ideally within 48 h. Decisions to start antiviral treatment should not wait for laboratory confirmation of influenza because it delays treatment, and a negative rapid influenza diagnostic test result does not rule out influenza. Antiviral medications are approximately 70–90% effective in preventing influenza and are useful adjuncts to influenza vaccination. The recommended dose for prevention is 75 mg daily for 7 days, and in exposed individuals it reduces the rate of infection by 70–90% [15]. The recommended dose for treatment is 75 mg twice daily for 5 days. In a study, the percentage of pregnant women with severe illness increased significantly from 3% when the drug was given within 48 h to 44% once the drug was given more than 5 days after symptom onset [16]. Hospitalized patients with severe infections (such as those with prolonged infection or who require

Table 14.1 Antiviral medication for H1N1 in pregnancy

	Chemoprophylaxis	Treatment	Remarks
Oseltamivir (Tamiflu®)	75 mg once daily × 7 days	75 mg twice daily × 5 days	Contraindicated in case of hypersensitivity
Zanamivir (Relenza®)	10 mg (two 5-mg inhalations) once daily × 7 days	10 mg (two 5-mg inhalations) twice daily × 5 days	Respiratory complications that may be associated

intensive care unit admission) might require longer treatment courses. Some experts have even advocated the use of increased (doubled) doses of oseltamivir for some severely ill patients, but limited data suggest that higher dosing may not provide additional clinical benefit.

Another drug zanamivir is an inhalational drug and has lesser effect on fetus as it does not cross the placenta. Antiviral drugs are not a cure, but can shorten the illness and reduce the risk of complications, hasten recovery, and minimize chances of severe illness and hospitalization [17, 18].

Drugs safe for treatment of influenza in pregnancy are summarized in Table 14.1 [19].

14.6 Vaccination

The World Health Organization, Centers for Disease Control and Prevention (CDC) Advisory Committee on Immunization Practices (ACIP) since 2005 recommends the inactivated influenza vaccine for immunization of pregnant women especially during the influenza season irrespective of the period of gestation and that it should be administered as soon as it is available [20–22]. Both the trivalent (H1N1 + H3N2 + influenza B) and the quadrivalent (two strains each of influenza A and B) formulations are safe for use in pregnancy according to ACIP [22, 23]. Vaccination with influenza vaccine not only protects pregnant women but also infants up to 6 months of age due to transplacental transfer of antibodies [24].

The only contraindication to vaccination is severe protein or egg allergy or allergy to a previous dose. Thiomersal, the preservative in the multidose vials, is also considered safe for use in pregnancy [22].

The vaccine is safe, and long-term longitudinal studies have shown that maternal influenza immunization did not increase the number of stillbirths, congenital malformations, malignancies, or neurocognitive disabilities [25–28]. The results of a randomized trial in Bangladesh showed a decrease of 63% of laboratory-confirmed influenza, 29% reduction in febrile respiratory illness in infants, and 36% reduction in febrile respiratory illness in mothers [24]. It also showed that babies of vaccinated cohort weighed an average of 200 g more than babies born to unvaccinated mothers.

According to CDC persistent influenza vaccine efficacy rates of around 60–70% [29]. In a prospective, controlled, blinded trial, vaccine effectiveness of maternal influenza vaccine in their infants was 63% until at least 6 months of age. There was a 29 and 36% reduction in the rates of febrile respiratory illnesses in infants and mothers, respectively [24].

14.7 Newborn Care and Breastfeeding

Breastfeeding should continue as it provides protection from many infections through immunity from maternal antibodies. Breastfeeding is advisable if the woman has received flu vaccination or if she is on antiviral medication. Formula milk preparations should be discouraged, and expressed milk should be given if the mother is too sick to feed the baby or if she is on treatment for less than 48 h [30].

The newborn need not be isolated from the mother unless she is very sick or if the infant is premature with many comorbidities. All precautions such as hand hygiene, the use of masks, cough etiquette, etc. must be used in order to minimize spread of infection.

To conclude, obstetric providers need to be prepared to provide the care necessary to address

the increased morbidity, mortality, and pregnancy-related complications during H1N1 infection in pregnancy.

Key Points

- Patients with novel H1N1 typically present with mild symptoms such as fever, cough, sore throat, and rhinorrhea, but pregnant women are at high risk of complications and severe disease.
- There is no need of confirmation by laboratory testing, and treatment must be offered to all irrespective of the test.
- Oseltamivir is safe in pregnancy, and treatment must be initiated within 48 h of symptoms.
- Influenza vaccine is recommended for all pregnant women irrespective of gestation.
- Breastfeeding is recommended as it helps in strengthening the neonatal immune response.

References

1. Dawood FS, Jain S, Finelli L, Shaw MW, Lindstrom S, Garten RJ, Gubareva LV, Xu X, Bridges CB, Uyeki TM. Emergence of a novel swine-origin influenza A (H1N1) virus in humans. N Engl J Med. 2009;360:2605–15.
2. Neumann G, Noda T, Kawaoka Y. Emergence and pandemic potential of swine-origin H1N1 influenza virus. Nature. 2009;459:931–9.
3. Royal College of Obstetricians and Gynaecologists (RCOG). Pandemic H1N1 2009 influenza: Clinical management guidelines for pregnancy. London: RCOG; 2009.
4. Mullooly JP, Barker WH, Nolan TF. Risk of acute respiratory disease among pregnant women during influenza A epidemics. Public Health Rep. 1986:205–11.
5. Neuzil KM, Reed GW, Mitchel EF, Simonsen L, Griffin MR. Impact of Influenza on Acute Cardiopulmonary Hospitalizations in Pregnant Women. Am J Epidemiol. 1998;148:1094–102.
6. Cox S, Posner SF, McPheeters M, Jamieson DJ, Kourtis AP, Meikle S. Hospitalizations with respiratory illness among pregnant women during influenza season. J Obstet Gynaecol. 2006;107:1315–20.
7. Dodds L, McNeil SA, Fell DB, Allen VM, Coombs A, Scott J, et al. Impact of influenza exposure on rates of hospital admissions and physician visits because of respiratory illness among pregnant women. Can Med Assoc J. 2007;176:463–8.
8. Centers for Disease Control and Prevention (CDC). Maternal and Infant Outcomes Among Severely Ill Pregnant and Postpartum Women with 2009 Pandemic Influenza A (H1N1) --- United States, April 2009--August 2010. MMWR Morb Mortal Wkly Rep. 2011;60(35):1193–6.
9. Goodnight WH, Soper DE. Pneumonia in pregnancy. Crit Care Med. 2005;33(10)
10. Rasmussen SA, Jamieson DJ, Macfarlane K, Cragan JD, Williams J, Henderson Z, Pandemic Influenza and Pregnancy Working Group. Pandemic influenza and pregnant women: summary of a meeting of experts. Am J Public Health. 2009;99(Suppl 2):S248–54.
11. Carlson A, Thung SF, Norwitz ER. H1N1 influenza in pregnancy: what all obstetric care providers ought to know. Rev Obstet Gynecol. 2009;2(3):139–45.
12. Community strategy for pandemic influenza mitigation. http://pandemicflu.gov/professional/community/commitigation.html. Accessed 22 June 2016.
13. Centre of disease control and prevention. Prevention Strategies for Seasonal Influenza in Healthcare Settings. http://www.cdc.gov/flu/professionals/infectioncontrol/healthcaresettings.htm. Accessed 02 July 2016.
14. Beigi RH, Venkataramanan R, Caritis SN. Oseltamivir for influenza in pregnancy. Semin Perinatol. 2014;38(8):503–7.
15. CDC. 2011-2012influenza antiviral medications: summary for clinicians. http://www.cdc.gov/flu/professionals/antivirals/summary-clinicians.htm; Accessed 20 June 2016.
16. Creanga AA, Johnson TF, Graitcer SB, et al. Severity of 2009 pandemic influenza A(H1N1) virus infection in pregnant women. Obstet Gynecol. 2010;115(4):717–26.
17. Jefferson T, Demicheli V, Deeks J, Rivetti D. Neuraminidase inhibitors for preventing and treating influenza in healthy adults. Cochrane Database Syst Rev. 2000;(2):CD001265.
18. Hsu J, Santesso N, Mustafa R, et al. Antivirals for treatment of influenza: a systematic review and meta-analysis of observational studies. Ann Intern Med. 2012;156(7):512–24.
19. CDC. Recommendations for Obstetric Health Care Providers Related to Use of Antiviral Medications in the Treatment and Prevention of Influenza. http://www.cdc.gov/flu/professionals/antivirals/avrec_ob.htm. Accessed 28 June 2016.
20. WHO position paper. Vaccines against influenza. Wkly Epidemiol Rep. 2012;87:461–76.
21. Centre of disease control and prevention. Flu vaccine safety and pregnancy. http://www.cdc.gov/flu/protect/vaccine/pregnant.htm. Accessed 03 July 2016.
22. Committee on Obstetric practice and immunization expert work group. Influenza vaccination during pregnancy. http://www.acog.org/-/media/Committee-Opinions/Committee-on-Obstetric-Practice/co608.pdf. Accessed 04 July 2016.

23. Prevention and control of seasonal influenza with vaccines. Recommendations of the Advisory Committee on Immunization Practices—United States, 2013–2014. Centers for Disease Control and Prevention (CDC) [published erratum appears in MMWR Morb Mortal Wkly Rep 2013;62:906]. MMWR Recomm Rep. 2013;62(RR-7):1–43.
24. Zaman K, Roy E, Arifeen SE, Rahman M, Raqib R, Wilson E, et al. Effectiveness of maternal influenza immunization in mothers and infants [published erratum appears in N Engl J Med 2009;360:648]. N Engl J Med. 2008;359:1555–64.
25. Tamma PD, Ault KA, del Rio C, Steinhoff MC, Halsey NA, Omer SB. Safety of influenza vaccination during pregnancy. Am J Obstet Gynecol. 2009;201:547–52.
26. Carcione D, Blyth CC, Richmond PC, Mak DB, Effler PV. Safety surveillance of influenza vaccine in pregnant women. Aust N Z J Obstet Gynaecol. 2013;53:98–9.
27. Moro PL, Broder K, Zheteyeva Y, Walton K, Rohan P, Sutherland A, et al. Adverse events in pregnant women following administration of trivalent inactivated influenza vaccine and live attenuated influenza vaccine in the Vaccine Adverse Event Reporting System, 1990–2009. Am J Obstet Gynecol. 2011;204:146.e1–7.
28. Bednarczyk RA, Adjaye-Gbewonyo D, Omer SB. Safety of influenza immunization during pregnancy for the fetus and the neonate. Am J Obstet Gynecol. 2012;207:S38–46.
29. Centers for Disease Control and Prevention (CDC). Seasonal Influenza Vaccine Effectiveness, 2005–2016. http://www.cdc.gov/flu/professionals/vaccination/effectiveness-studies.htm. Accessed 03 July 2016.
30. CDC Guidance on Influenza and Infant Feeding. https://www.cdc.gov/breastfeeding/disease/influenza.htm. Accessed 04 July 2016.

Thromboprophylaxis

15

Rashmi Bagga and Rimpi Singla

15.1 Introduction

Pregnancy and the puerperium are known to increase the risk of deep vein thrombosis (DVT) and venous thromboembolism (VTE) [1–3]. The incidence DVT is highest during the puerperium; hence all postpartum women should be instructed about the signs and symptoms and should be examined carefully for any evidence of VTE. Thromboprophylaxis is offered to reduce the risk of VTE and may be in the form of ***mechanical methods*** (compression stockings or pneumatic compression devices) or ***pharmacologic agents*** (anti-coagulating agents). Screening for and identification of such women who require thromboprophylaxis early during pregnancy or in the preconceptional period reduce the risk of subsequent DVT and VTE [4]. These are women at high risk of VTE and women with prosthetic heart valves, atrial fibrillation, left ventricular dysfunction, cortical venous thrombosis and foetal loss due to anti-phospholipid syndrome. Some factors which add to the risk of VTE are history of VTE in the past, hospitalization and bed rest, caesarean section (CS) and inherited thrombophilia [3–5]. It is possible that some situations develop during the course of pregnancy and puerperium which require thromboprophylaxis. Caesarean section (CS), especially emergency CS, is associated with a higher risk of VTE [4, 6]. Early ambulation and mechanical thromboprophylaxis are usually sufficient to reduce the risk of VTE following CS, and pharmacologic prophylaxis is required only if there are additional risk factors for VTE. Women already on anticoagulant therapy (e.g. for a prosthetic valve) which is to be continued during pregnancy should be switched from oral anticoagulants to a heparin-based regimen as soon as pregnancy is diagnosed in order to avoid potential teratogenic effects of oral anticoagulants. Change from warfarin to LMWH can be done during attempted conception or can be done once pregnancy is confirmed, as long as this switchover is feasible before 6 weeks of pregnancy.

15.2 Indications for Thromboprophylaxis

Women who require pharmacologic anticoagulation during pregnancy and puerperium include those with prosthetic heart valves, atrial fibrillation, left ventricular dysfunction, foetal loss due to anti-phospholipid syndrome, and cortical venous thrombosis and women at high risk for VTE. Most of these indications are described in separate sections, and in the present chapter, the main focus is on thromboprophylaxis to prevent VTE. Pregnant women who have had a prior VTE which was related to a high oestrogen state (e.g. prior pregnancy or oestrogen-related VTE) are candidates

R. Bagga (✉) · R. Singla
Department of Obstetrics & Gynaecology, PGIMER, Chandigarh, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_15

for *pharmacologic* thromboprophylaxis because these risk factors may result in recurrent VTE during pregnancy. In contrast, women who had a transient risk factor for prior VTE (like trauma, bed rest, surgery) have less likelihood of a recurrent VTE and require only surveillance, general measures to reduce the risk and *mechanical* thromboprophylaxis. Duration of thromboprophylaxis in postpartum period is usually 6 weeks and up to 3 months in those at a higher risk. However when thromboprophylaxis is administered during hospitalization due to an acute illness, it is usually continued until the patient is ambulatory. The American College of Chest Physicians [7] and the American College of Obstetricians and Gynecologists [8] guidelines for the indications for thromboprophylaxis during pregnancy and puerperium to reduce the risk of VTE are summarized below [4, 7, 8]:

1. Indications for pharmacologic thromboprophylaxis during the puerperium only are:
   (a) No thrombophilia with prior one episode of VTE associated with a transient risk factor that is no longer present (surveillance alone and no anticoagulation is acceptable).
   (b) Lower-risk thrombophilia with no previous VTE (heterozygous factor V Leiden (fVL) mutation; protein S or protein C deficiency; prothrombin (PT) *G20210A* heterozygous) with risks factors like obesity/prolonged immobilization or family history of VTE in first-degree relative before age of 50 years.
   (c) Low-risk thrombophilia with one prior episode of VTE.
   (d) High-risk thrombophilia (antithrombin deficiency, homozygous fVL or prothrombin *G20210A* homozygous, double heterozygous for prothrombin *G20210A* and fVL).
   (e) No thrombophilia and prior one episode of VTE related to pregnancy or use of oestrogens.
   (f) No thrombophilia and prior one episode of VTE that is not associated with any risk factor (i.e. idiopathic).

   In (c–f), during pregnancy, surveillance alone or prophylactic anticoagulation with low molecular weight heparin/unfractionated heparin (LMWH/UFH) are both acceptable.
2. Indications for pharmacologic thromboprophylaxis during pregnancy and puerperium are:
   (a) High-risk thrombophilia and previous one episode of VTE or family history of VTE in first-degree relative.
   (b) Previous two or more episodes of VTE with or without known thrombophilia and not receiving long-term anticoagulation.
   (c) Previous two or more episodes of VTE with or without known thrombophilia and receiving long-term anticoagulation.
   (d) Hospitalized women at risk for VTE (sepsis, pneumonia, trauma, fracture), prolonged bed rest (>3 days) and having other risk factors (obesity, i.e. BMI > 30 kg/m^2; older age, i.e. >35 years; multiparity; critical illness; malignancy; ovarian hyperstimulation).

15.3 Types of Thromboprophylaxis

General measures to prevent VTE should be advised in all women. These are early ambulation, adequate hydration, active and passive leg movements and frequent left-lateral decubitus position during late pregnancy [9].

1. ***Mechanical methods*** are the use of graduated compression stockings and pneumatic compression devices. Graduated compression stockings or anti-embolism stockings (AES) provide graduated compression with a calf pressure of 14–15 mmHg. These are recommended in pregnancy and the puerperium in the following [4, 10]:
   (a) Women who need pharmacologic thromboprophylaxis but have a contraindication to agents due to high risk of haemorrhage, ongoing haemorrhage or coagulopathy.
   (b) In combination with pharmacologic agents in women who are hospitalized

post CS and those who are at high risk of VTE.

Fitting Precautions for AES

(a) Measure girth of legs and use correct stocking size.
(b) Demonstrate how to use AES, and encourage women to wear them throughout day and night during hospital stay until they do not have significantly reduced mobility.
(c) If oedema or post-operative swelling develops, change size of AES.
(d) Remove AES daily for hygiene and to check skin condition.
(e) Discontinue use if there is skin marking or discolouration.

Contraindications to AES [9]

(a) Known allergy to AES material.
(b) Suspected or proven peripheral arterial disease or peripheral arterial bypass grafting.
(c) Peripheral neuropathy.
(d) Local condition where AES may cause damage (e.g. dermatitis, gangrene, pressure ulcers).
(e) Severe leg oedema.
(f) Unusual leg size or shape or leg deformity preventing correct fit.

Foot Impulse and Intermittent Pneumatic Compression Devices (IPC)

All women undergoing CS may have pneumatic compression device placed if they are not receiving anticoagulants [1]. Patient with these devices should be encouraged to use them as much as practically possible. Contraindications to their use are acute DVT or PE and those listed for AES.

2. Pharmacological methods [11]:
   (a) Heparins: Low molecular weight heparin (LMWH) and unfractionated heparin (UFH).
   (b) Fondaparinux.
   (c) Direct thrombin inhibitor.
   (d) Vitamin K antagonists.

(a) *Heparins*: Heparin depends on antithrombin (AT) for its action and clotting factor inhibition. Heparin does not lyse the existing thrombi. Heparin binds and activates AT and induces conformational change. This accelerates AT binding to and inactivation of XIIa, IXa, XIa, Xa and thrombin [12, 13]. UFH can be used as intravenous infusion or subcutaneous injections [13]. Intravenous administration attains therapeutic concentrations in plasma rapidly that can be monitored and modified. Subcutaneous therapeutic dose is large due to low bioavailability. UFH is the best option for patients requiring high doses of anticoagulation and for patients who are at risk of bleeding (like impending delivery) due to its short half-life and reversal capability. It binds to endothelial cells and macrophages and is depolymerised for clearance initially. The second phase of clearance is renal-mediated and is slower and non-saturable. Hence, with the increased or prolonged use of UFH, there is an increase in intensity and duration of its effect [12, 13]. LMWHs are the anticoagulants of choice for thromboprophylaxis during antenatal and postnatal periods. As compared to UFH, LMWHs have higher bioavailability and longer half-life and cause fewer adverse events [14]. It has dose-independent renal clearance. Hence, UFH is preferred over LMWH in severe renal insufficiency (creatinine clearance <30 mL/min) or fluctuating renal function because LMWH metabolism is exclusively renal.

Contraindications/Cautions

- Known bleeding disorder (acquired coagulopathy, haemophilia or von Willebrand's disease).
- Thrombocytopenia.
- Severe liver disease (increased prothrombin time or known to have varices).
- Severe renal disease.
- Women at increased risk of major haemorrhage (e.g. placenta praevia).
- Active antepartum or postpartum haemorrhage.
- Acute haemorrhagic or ischaemic stroke in preceding 4 weeks.
- Uncontrolled hypertension (blood pressure > 200 mmHg systolic or > 120 mmHg diastolic).

Dose

A higher dose is necessary during pregnancy as compared to non-pregnant women due to alterations in metabolism, plasma volume and renal clearance. Prophylactic dose is used for prevention of DVT in women with indications listed above. Therapeutic dose is used for treatment of VTE and for thromboprophylaxis if lesser doses seem insufficient for thromboprophylaxis, e.g. patients with a very high risk of VTE or mechanical heart valves.

UFH: Prophylactic dose: Administered subcutaneously (SC) every 12 h. Monitoring of aPTT is not needed.

- 5000–7500 units in first trimester
- 7500–10,000 units in second trimester
- 10,000 units in third trimester
- 5000–7500 units in puerperium.

UFH: Therapeutic Dose [11]

- 80 U/kg bolus followed by 18 U/kg/h infusion adjusted to maintain activated partial thromboplastin time (aPTT) of 2–2.5 times the control
- 10,000 units or more subcutaneously 12 hourly; dose adjusted to maintain aPTT in therapeutic target range measured 6 h after injection.

Monitor aPTT daily until the target level is attained and then every one to two weekly thereafter.

Dose of LMWH [11, 15]:

LMWH: Prophylactic Dose

- Enoxaparin: 40 mg SC every 24 h.
- Dalteparin: 5000 units SC every 24 h.

LMWH: Intermediate Dose

- Alteration of prophylactic dose according to change in weight during pregnancy (e.g. enoxaparin 40 mg SC twice daily).

LMWH: Therapeutic Dose

- Enoxaparin: 1 mg/kg SC every 12 h or 1.5 mg/kg SC every 24 h; if creatinine clearance is <30 mL/min, then 1 mg/kg SC every 24 h.
- Dalteparin: 100 units/kg SC every 12 h.

For LMWH used in therapeutic dose for treatment, the anti-factor Xa levels are to be maintained between 0.6 units/mL and 1.0 units/mL measured 4–6 h after each dose. However, only few women need increase in the dose of LMWH when it is administered based on the weight. Peak anti-Xa activity occurs after 3–5 h of injection. Due to their predictable dose-response relationship, laboratory monitoring is not required generally [4] except in some high-risk patients (renal insufficiency, obesity, pregnancy) in whom dose adjustments are needed [16]. Prophylactic dose does not need monitoring unless the levels are suspected to be outside the recommended range.

(b) *Fondaparinux*: This is a synthetic analog of the pentasaccharide found in heparin that selectively binds to AT. This results in neutralization of factor Xa and, hence, inhibition of thrombin formation [12, 13]. It has rapid and complete absorption after SC administration and has a long half-life. Fondaparinux is excreted primarily unchanged through kidneys and, hence, contraindicated if there is severe renal impairment [12]. Fondaparinux is as safe and effective as LMWH and UFH for treatment of VTE [17]. Fondaparinux is a good option for thromboprophylaxis in patients with heparin-induced thrombocytopenia (HIT) [18]. Fondaparinux should not be used in patients with weight less than 50 kg. The ACCP 2012 Guidelines suggest that fondaparinux should only be used in women with HIT and not to be used by breast-feeding women.

- *Dose of fondaparinux for VTE* [11, 15].
- <50 kg: 5 mg SC daily
- 50–100 kg: 7.5 mg SC daily
- >100 kg: 10 mg SC daily.
- *Prophylactic dose of fondaparinux*:
- 25 mg SC daily.

(c) *Direct Thrombin Inhibitors (DTI) [19]*: They selectively bind to the active site of thrombin leading to inhibition of reactions catalysed by thrombin, i.e. activation of factors V, VIII and

XIII and protein C, fibrin formation and platelet aggregation. At present, four parenteral DTIs are approved by USFDA: lepirudin, desirudin, bivalirudin and argatroban. DTIs are monitored by aPTT. Desirudin does not need monitoring. At present, data about their safety in pregnancy is limited to case reports.

(d) *Vitamin K Antagonists*: Warfarin is an inhibitor of vitamin K. Vitamin K-dependent factors in the coagulation cascade (II, VII, IX and X and proteins C and S) are altered by vitamin K-dependent reactions before getting released into the circulation. Warfarin blocks the carboxylation system and reduces their binding capacity to phospholipid for activation [20]. Warfarin is used in pregnancy only in those situations where heparin is not suitable, e.g. mechanical prosthetic valves. In the postpartum period, women who require continuous anticoagulation should be given UFH or LMWH. They are converted to warfarin in postpartum period as the risk of bleeding is reduced, i.e. usually after 5 days of delivery [3]. During this switchover phase, heparins should be continued with warfarin for 5 days and additional 1–2 days after achieving target INR. Dose is titrated according to INR, target range 1.5–2.5 times the control. UFH or LMWH can be discontinued after attaining target INR. The duration of postpartum therapy depends upon the indication and should be given for at least 6 weeks postpartum or for 3 months.

Target-Specific Oral Anticoagulants: They include dabigatran, rivaroxaban and apixaban. These are novel anticoagulants having rapid onset and predictable action. Dabigatran reversibly binds to the active site on thrombin. It also has anti-platelet effect [21]. It is used for prevention of stroke and embolism in patients with non-valvular AF. Rivaroxaban is highly selective competitive inhibitor of factor Xa, preventing thrombin generation [22]. Indications are stroke prevention in non-valvular AF and for treatment and secondary prevention of DVT and PE. Apixaban is approved for stroke prevention in non-valvular AF. Currently, no antidote is available to reverse these drugs, and there is little data and clinical experience for their use in pregnancy.

15.4 Adverse Effects and Complications of Pharmacological Thromboprophylaxis

1. Heparin-induced thrombocytopenia (HIT) and HIT with thrombosis (HITT) occur due to antibodies formed against heparin–platelet factor IV complex in some patients exposed to heparins for 5–7 days [23]. A baseline platelet count should be obtained while initiating therapeutic doses and then monitored every other day for initial 4–10 days of treatment. A 50% decrease in platelet count or formation of new thrombus while anticoagulated may be suggestive of HIT. Heparins should be discontinued and alternative anticoagulant be started (danaparoid or fondaparinux). Sodium danaparoid consists of heparan sulphate (84%), dermatan sulphate (12%) and chondroitin sulphate (4%). It is different from UFH and LMWH. It inactivates factor Xa and, to a lesser extent, factor IIa and reacts with only 0–20% of antibodies in HIT. Danaparoid does not impair the formation of haemostatic plug of platelets and, hence, does not cause increased blood loss during delivery. Danaparoid has been used successfully in pregnant patients with HIT who are in need of thromboprophylaxis or treatment for thrombosis, but limited literature is available about their effects on foetus. During pregnancy, danaparoid is preferred; lepirudin and fondaparinux are used only if danaparoid is not available.
2. Haemorrhage: This is the major complication of heparin therapy [24]. The risk of major bleeding is 0–7% and that of fatal bleeding is 0–3%. Risk of haemorrhagic complications is directly related to the intensity of anticoagulation and concurrent use of glycoprotein IIB/IIIA inhibitors [24]. Measure to reduce the risk of haemorrhage are [1, 4]:
   (a) LMWH should be switched to UFH in last month of pregnancy or earlier prior to anticipated delivery as UFH has shorter duration of action and is easier to reverse.

(b) Therapeutic anticoagulation with LMWH is stopped 24 h before induction of labour.
(c) If labour develops in a patient receiving UFH, discontinue it. Protamine is best avoided during antenatal period if haemorrhage can be controlled with routine measures.
(d) Neuraxial block is withheld up to 24 h of last therapeutic dose and 12 h of last prophylactic dose of LMWH.
(e) Anticoagulation should be resumed only 4–6 h after vaginal delivery, 6–12 h after CS or 12 h after neuraxial catheter removal. It may be delayed in case of continued bleeding. Avoid removal of neuraxial catheter within 12 h of last injection.

Reversal of UFH is usually not required if there is minor bleeding. If bleeding persists, anticoagulation may be withheld until bleeding stops. If bleeding is severe, anti-heparin therapy (protamine sulphate) is used [11, 25]. The dose of protamine depends up on the timing of the last dose of UFH. For immediate reversal, dose of protamine is 1 mg for every 100 U of UFH (maximum 50 mg), and aPTT can be used to evaluate the response to reversal. When given as IV infusion, UFH administered during last 2–2.5 h is used to calculate the dose of protamine. Protamine does not reverse the effect of LMWH completely but can neutralize the AT effect. Use 1 mg protamine per 1 mg enoxaparin or per 100 units dalteparin or tinzaparin given in previous 8 h. In fondaparinux-related haemorrhage, recombinant activated factor VII can normalize the coagulation. Warfarin effect is reversed by vitamin K administration (1–2 mg oral or 5–10 mg IV in urgent situations).

3. Osteoporosis [26]: Risk for osteoporosis and vertebral fractures is increased in patients receiving UFH for over a month (approximate incidence 2%).
4. Preservatives: Preservatives like benzyl alcohol in multi-dose vials of LMWH and UFH can have adverse foetal effects. Single-dose delivery syringes generally do not contain preservatives. This however should be confirmed from the product label.
5. Heparin resistance: Antithrombotic action of heparin depends on antithrombin III. Hence, deficiency in plasma antithrombin III (e.g. in DIC, massive VTE, liver failure) results in resistance to heparin [27]. Increased amount of heparin or substitution by oral anticoagulants is required to counteract heparin resistance.
6. Warfarin is associated with teratogenicity and warfarin embryopathy [28]. Hence, patients on warfarin are switched over to heparin at least in the first trimester and also for the rest of pregnancy unless the risk of thrombosis is high as in prosthetic valves.

15.5 Postpartum Use of Anticoagulation

Anticoagulation is reinstituted in the postpartum period in most patients who were receiving anticoagulants during antenatal period. As mentioned earlier, prophylactic anticoagulation is indicated in some patients only during postpartum period. The duration of postpartum anticoagulation depends upon the indication for anticoagulation and is given for at least 6 weeks postpartum or for 3 months. Both heparins and warfarin can be given to breast-feeding women [1, 29].

15.6 Diagnosis and Management of Acute VTE During Pregnancy

Any woman with symptoms or signs of VTE should be hospitalized, and treatment with LMWH/UFH should be started without delay based on clinical suspicion unless absolutely contraindicated [30]. It may be stopped once diagnosis is excluded by objective testing. Compression duplex ultrasound is performed immediately [1, 30, 31]. If this confirms the presence of DVT, treatment for VTE should continue. No further investigation is required. If this is negative, anticoagulants may be discontinued. In case high level of clinical suspicion exists, ultrasound should be repeated after 3–7 days [30].

Electrocardiogram and a chest X-ray are also done. In the event of suspected PE but without clinical features of DVT and/or negative compression ultrasound, ventilation/perfusion scan or CTPA should be performed. If chest X-ray is abnormal, CTPA is preferred over V/Q scan. As compared to CTPA, V/Q scanning may be associated with a higher risk of childhood cancer but a lower risk of maternal breast cancer [30, 32]. However, the absolute risk of either is small. Repeat testing should be done if these investigations are normal, but there is high index of clinical suspicion. Anticoagulants should be continued until diagnosis is definitely excluded.

Initial management of DVT comprises limb elevation and application of graduated compression stocking. Mobilization with stockings should be encouraged. Baseline investigations like complete blood count, renal function tests and liver function tests are done. UFH or LMWH are the anticoagulants of choice for treatment of acute thrombosis [7, 30]. Dose of LMWH should be titrated according to the woman's early pregnancy weight. Monitoring of peak anti-Xa activity is not recommended except in women with renal impairment or recurrent VTE or with very low (less than 50 kg) or very high body weight (more than 90 kg) [30]. An IVC filter may be considered in the patients with iliac vein thrombosis or in patients who have DVT with recurrent PE despite anticoagulation or in patients with contraindications to anticoagulation.

Massive VTE may present as collapse or shock. Such women should be assessed by a team of obstetrician, anaesthetist and haematologist. An urgent portable echocardiogram or CTPA is done. If massive PE is confirmed, immediate thrombolysis is considered. Treatment plan regarding heparin, thrombolytic therapy or surgical embolectomy should be individualized [33]. IV UHF is preferred for initial treatment of massive PE with cardiovascular compromise.

Therapeutic dose is continued for the rest of the pregnancy and for at least 6 weeks postpartum and until at least a total of 3 months [30]. Outpatient follow-up includes clinical assessment and checking platelet count and peak anti-Xa levels (as appropriate). Warfarin should not be used for antenatal VTE treatment and should also be avoided in initial postpartum period until fifth day or as long as woman is at increased risk of postpartum haemorrhage.

References

1. James A, Committee on Practice Bulletins—Obstetrics. Practice bulletin; no. 123: thromboembolism in pregnancy. Obstet Gynecol. 2011;118:718–29. Washington (DC): American College of Obstetricians and Gynecologists (ACOG).
2. Dresang LT, Fontaine P, Leeman L, King VJ. Venous thromboembolism during pregnancy. Am Fam Physician. 2008;77(12):1709–16.
3. Danilenko-Dixon DR, Heit JA, Silverstein MD, Yawn BP, Petterson TM, Lohse CM, Melton LJ III. Risk factors for deep vein thrombosis and pulmonary embolism during pregnancy or post partum: a population based, case-control study. Am J Obstet Gynecol. 2001;184:104–10.
4. Royal College of Obstetricians and Gynaecologist. Reducing the risk of venous thromboembolism during pregnancy and the puerperium. Green-top Guideline No. 37a. 2015. http://www.rcog.org.uk/files/rcog-corp/GTG37aReducingRiskThrombosis.pdf.
5. Zotz RB, Gerhardt A, Scharf RE. Prediction, prevention and treatment of venous thromboembolic disease in pregnancy. Semin Thromb Hemost. 2003;29(2):143–54.
6. Deneux-Tharaux C, Carmona E, Bouvier-Colle MH, Breart G. Postpartum maternal mortality and cesarean delivery. Obstet Gynecol. 2006;108(3 pt 1):541–54.
7. Bates SM, Greer IA, Middeldorp S, Veenstra DL, Prabulos AM, Vandvik PO, American College of Chest Physicians. VTE, thrombophilia, antithrombotic therapy, and pregnancy: antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians evidence-based clinical practice guidelines. Chest. 2012;141:e691S.
8. American College of Obstetricians and Gynecologists Women's Health Care Physicians. ACOG practice bulletin no. 138: Inherited thrombophilias in pregnancy. Obstet Gynecol. 2013;122:706.
9. National Health and Medical Research Council. Clinical practice guideline for the prevention of venous thromboembolism in patients admitted to Australian hospitals. Melbourne: NHMRC; 2009. https://www.nhmrc.gov.au/_files_nhmrc/file/nics/programs/vtp/guideline_prevention_venous_thromboembolism.pdf
10. Sachdeva A, Dalton M, Amaragiri SV, Lees T. Elastic compression stockings for prevention of deep vein thrombosis. Cochrane Database Syst Rev. 2010;(7):CD001484.

11. Alquwaizani M, Buckley L, Adams C, Fanikos J. Anticoagulants: a review of the pharmacology, dosing, and complications. Curr Emerg Hosp Med Rep. 2013;1:83–97.
12. Garcia DA, Baglin TP, Weitz JI, Samama MM. Parenteral anticoagulants: antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians evidence based clinical practice guidelines. Chest. 2012;141:24S–43S.
13. Weitz DS, Weitz JI. Update on heparin: what do we need to know? J Thromb Thrombolysis. 2010;29:199–207.
14. Greer IA, Nelson-Piercy C. Low-molecular-weight heparins for thromboprophylaxis and treatment of venous thromboembolism in pregnancy: a systematic review of safety and efficacy. Blood. 2005;106:401–7.
15. Institute for Safe Medication Practices. QuarterWatch; monitoring FDA MedWatch reports. http://www.ismp.org/quarterwatch/pdfs/2012Q1.pdf.
16. Nutescu EA, Spinler SA, Wittkowsky A, Dager WE. Low molecular-weight heparin in renal impairment and obesity: available evidence and clinical practice recommendations across medical and surgical settings. Ann Pharmacother. 2009;43:1064–83.
17. The Matisse Investigators. Subcutaneous fondaparinux versus intravenous unfractionated heparin in the initial treatment of pulmonary embolism. N Engl J Med. 2003;349:1695–702.
18. Dager WE, Dougherty JA, Nguyen PH, et al. Heparin-induced thrombocytopenia: treatment options and special considerations. Pharmacotherapy. 2007;27:564–87.
19. Di Nisio M, Middeldorp A, Buller HR. Direct thrombin inhibitors. N Engl J Med. 2005;353:1028–40.
20. Ageno W, Gallus AS, Wittkowsky A, et al. Oral anticoagulant therapy: antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians evidence-based clinical practice guidelines. Chest. 2012;141:44S–88S.
21. Spinler BE, Baetz SA. Dabigatran etexilate: an oral direct thrombin inhibitor for prophylaxis and treatment of thromboembolic diseases. Pharmacotherapy. 2008;28:1354–73.
22. Gulseth MP, Michaud J, Nutescu EA. Rivaroxaban: an oral direct inhibitor of factor Xa. Am J Health Syst Pharm. 2008;65:1520–9.
23. Martel N, Lee J, Wells PS. Risk for heparin-induced thrombocytopenia with unfractionated and low-molecular-weight heparin thromboprophylaxis: a meta-analysis. Blood. 2005;106:2710–5.
24. Schulman S, Beth RJ, Kearon C, Levine MN. Hemorrhagic complications of anticoagulant and thrombolytic treatment: American College of Chest Physicians evidence-based clinical practice guidelines (8th Edition). Chest. 2008; 133:257S–98S.
25. McEvoy GK. Protamine sulfate. In: AHFS drug information 2008. Bethesda: American Society of Health-System Pharmacists; 2008. p. 1595–7.
26. Casele H, Haney EI, James A, Rosene-Montella K, Carson M. Bone density changes in women who receive thromboprophylaxis in pregnancy. Am J Obstet Gynecol. 2006;195:1109–13.
27. Wessler S, Gitel SN. Pharmacology of heparin and warfarin. J Am Coli Cardiol. 1986;8:108–208.
28. Chan WS, Anand S, Ginsberg JS. Anticoagulation of pregnant women with mechanical heart valves: a systematic review of the literature. Arch Intern Med. 2000;160:191–6.
29. Richter C, Sitzmann J, Lang P, Weitzel H, Huch A, Huch R. Excretion of low molecular weight heparin in human milk. Br J Clin Pharmacol. 2001;52(6):708–10.
30. Royal College of Obstetricians and Gynaecologists. Thromboembolic disease in pregnancy and the puerperium: acute management. Green-top Guideline No. 37b. 2015.
31. Bates SM, Ginsberg JS. How we manage venous thromboembolism during pregnancy. Blood. 2002;100:3470–8.
32. Schafer AI, Levine MN, Konkle BA, Kearon C. Thrombotic disorders: diagnosis and treatment. Hematology. 2003:520–39.
33. Pillny M, Sandmann W, Luther B, et al. Deep venous thrombosis during pregnancy and after delivery: indications for and results of thrombectomy. J Vasc Surg. 2003;37(3):528–32.

Anaphylaxis

16

Shailesh Kore, Humaira Ali, and Pradnya Supe

16.1 Introduction

Anaphylaxis is a potentially life-threatening hypersensitivity reaction involving multiple organs and requires immediate management [1, 2]. The word "anaphylaxis" is derived from the Greek words ana (meaning backward) and phylax (meaning to protect or guard) implying that the agent administered for its protective effect has on the contrary proved harmful. True definition of anaphylaxis refers only to IgE-mediated hypersensitivity reaction (Type 1), while non IgE-mediated reactions are termed as "anaphylactoid reactions." However it is difficult to distinguish between two conditions clinically. Also the initial management of both these conditions is also identical. The World Allergy Organization has now classified anaphylaxis into immunologic, immunoglobulin E (IgE)-mediated, and non-immunologic reactions [3]**.** Incidence of anaphylaxis during pregnancy is estimated to be around 1 in 30,000 pregnancies [4].

Pregnancy involves several hospital visits with administration or ingestion of medications which can sometimes cause anaphylaxis. Also the immunological changes that occur during the pregnancy can predispose women to anaphylaxis [5].

Anaphylaxis during pregnancy can be devastating and can endanger the life of both the mother and the fetus by causing hypoxic-ischemic encephalopathy and permanent central nervous system damage [6].

16.2 Pathophysiology [7–9]

Anaphylactic and anaphylactoid reactions occur because of release of mediators from mast cells and basophils following their degranulation due to antigen-antibody reaction. These mediators include histamine, tryptase, heparin, chymase, and cytokines and arachidonic acid metabolites (e.g., prostaglandins and leukotrienes), which are stored in the granules of mast cells and basophils.

Classical anaphylactic reaction occurs following reexposure to an antigen to which the individual has already been sensitized due to previous exposure in the past. During the primary exposure to antigen, IgE antibody is produced, and the substance which induces this response is called an "antigen," and the individual is now said to be sensitized to that particular antigen. These IgE antibodies bind to the high-affinity IgE receptor on the surface of mast cells and basophils. Subsequent reexposure to the same antigen results in antigen-antibody reaction, and the antigen-bound IgE antibodies cause degranulation of mast

S. Kore (✉)
L.T.M. Medical College, Mumbai, India

Genetic & Fetal Medicine Committee, FOGSI, Mumbai, India

FOGSI Website Committee, Mumbai, India

H. Ali · P. Supe
L.T.M. Medical College, Mumbai, India

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cells and basophils and release of histamine which is the primary mediator of anaphylaxis along with prostaglandins and leukotrienes. Binding of histamine to its H1 and H2 receptors primarily as well as prostaglandins and leukotrienes to their respective receptors is responsible for the widespread systemic effects seen in anaphylaxis. Other pathways active during anaphylaxis are the complement system, the kallikrein-kinin system, the clotting cascade, and the fibrinolytic system.

Activation of a specific subset of lymphocyte lineage (CD4 + Th2 T cells) is believed to be responsible for IgE production. CD4 + T cells have been classified as Th1 type or Th2 type. Th1 is responsible for cellular immunity and production of interferon gamma, while Th2 plays an important role in humoral immunity as well as perpetuation of allergic response. Multiple factors including genetic and environmental factors along with type and quantity of immune trigger determine what type of response a particular individual would develop. Activation of Th2 cells rather than the Th1 lineage is believed to be responsible for the development and perpetuation of allergic responses.

16.3 Etiology

The etiological causes of anaphylaxis during pregnancy are the same as with the nonpregnant population [5].

Most common causes of this condition during pregnancy and labor are:

1. Food allergy—Peanut allergy is the most common food allergy. Other foods commonly associated with anaphylaxis include cow's milk, eggs, shellfish, soy, wheat, and tree nuts.
2. Medications—The most common drugs which cause anaphylaxis are the group of beta-lactam antibiotics (penicillin) particularly when given by parenteral route. These drugs are often used to prevent maternal infection when given prior to/during cesarean delivery or neonatal group B streptococcal infection. Other drugs include cephalosporins, sulfonamides, NSAIDs, aspirin, insulin, general anesthetics, insulin, progesterone, blood products, biological agents, radiocontrast materials, and immunotherapy. Also iron compounds like iron dextran and iron sorbitol injections which are commonly used to treat anemia during pregnancy are known to cause occasional anaphylactic reactions. Fortunately newer iron preparations like iron sucrose are less known to cause such reactions. Various B complex injections (B1, B6, B12) have also reported to cause such reactions.
3. Latex—Sensitization and subsequent anaphylactic reaction to latex can occur during various gynecological and obstetric procedures.
4. Hymenoptera venoms—Insect bite from honeybees, wasps, fire ants, etc. can also elicit IgE-mediated anaphylaxis.
5. IgG and immune complex-mediated reactions can occur to blood and blood products commonly used in transfusion. Partially unbound iron released from iron sucrose can increase the oxidative stress and cause adverse reactions.

Most common cause of anaphylaxis during labor and delivery is prophylactic injection of a penicillin or cephalosporin to prevent neonatal group B streptococcal (GBS) infection or to prevent maternal infection after cesarean delivery [10–12].

16.4 Diagnosis

Anaphylaxis is diagnosed when there is rapid development of symptoms and signs following history of exposure to a likely or known allergen and its subsequent evolution over period of minutes to hours [13]. The diagnosis of anaphylaxis is primarily clinical. The diagnosis can be made in the presence of clinical features even if laboratory tests are negative. Clinical diagnosis is made based on detailed history and development of characteristic features (symptoms and signs) which are often sudden in onset and rapidly progressing. The characteristic symptoms typically occur within a few minutes to hours after exposure to known or likely antigen. Symptoms often involve more than one system, most common being skin/cutaneous (80–90%), respiratory (70%), cardiovascular, and/or gastrointestinal (45–50%) cases.

Features of anaphylaxis in pregnancy include intense itching in the vulvar and vaginal areas, uterine cramps, low back pain, preterm labor, and fetal distress. There is mostly history of exposure to known or likely antigen. The severity of symptoms and signs may vary depending on type and quantum of allergen. Hypotension and shock may not occur in all cases although more than one organ system is typically involved.

16.4.1 Diagnostic Criteria

The National Institute of Health (NIH) in 2006 has set the diagnostic criteria for anaphylaxis based on three clinical scenarios [1]. The World Allergy Organization published similar guidelines which can help in prompt diagnosis and management. These guidelines enlist clinical criteria, in which, if present, diagnosis of anaphylaxis during pregnancy is most likely.

1. First, in women with no prior history of anaphylaxis or in the absence of any know allergen, anaphylaxis is diagnosed by a rapid onset (minutes to hours) of a reaction that involves the skin, mucosal tissue, or both (generalized urticaria, itching or flushing, swollen lips, tongue, and/or vulva), alongside at least one of the following symptoms:
   - Respiratory compromise—dyspnea, features of bronchospasm, stridor, reduced peak expiratory flow, and hypoxemia.
   - Reduced blood pressure or features suggestive of end-organ dysfunction like hypotonia, syncope, incontinence, or collapse.
2. Second, when after exposure to a likely allergen, occurrence of two or more of the following is within minutes to hours:
   - Involvement of the skin or mucosal tissue (generalized urticaria, itching or flushing, swollen lips, tongue, and/or vulva).
   - Respiratory symptoms, dyspnea, features of bronchospasm, stridor, and hypoxemia.
   - Decreased blood pressure features suggestive of end-organ dysfunction like hypotonia, syncope, incontinence, or collapse.
   - Persistent features suggestive of gastrointestinal involvement like pain, abdominal cramps, vomiting, etc.
3. Third, on exposure to a known allergen, reduced blood pressure alone is sufficient for the diagnosis of anaphylaxis. Reduced blood pressure is defined as systolic blood pressure of less than 90 mmHg or 30% decrease from woman's baseline blood pressure.

   General and systemic examination findings may vary depending on the severity and the organs involved and may show the following:

- General appearance: patient may be anxious, restless, and disoriented.
- Respiratory findings: angioedema of the tongue and lips, stridor or wheezing, loss of voice, hoarseness, and tachypnea.
- Dermatologic: urticaria (i.e., hives) is the classical skin manifestation which can occur anywhere on the body; angioedema or soft-tissue swelling or erythema can also occur.
- Cardiovascular: tachycardia, hypotension, and shock may occur immediately, without any other findings.
- Neurological: patient may be disoriented or may be agitated and restless.

The tryptase test is a useful indicator of mast cell activation. Mast cells are large tissue cells present in highest amounts in the skin. They release tryptase and other substances as part of the body's normal response to injury but also may release them as part of an allergic response. Though diagnosis of anaphylaxis is mainly clinical, tryptase test may be done to confirm a diagnosis of anaphylaxis, as the cause of someone's acute symptoms, particularly when done with a histamine test [14]. This is especially true if the person has recurrent episodes and/or if the diagnosis is uncertain.

Other tests done in anaphylaxis are those which are done to diagnose organ damage, like renal function tests, liver function tests, etc.

16.5 Differential Diagnosis

Since the most commonly involved systems in anaphylaxis are the skin, respiratory system, cardiovascular, and GIT, differential diagnosis of

anaphylaxis is the conditions related to these systems. Most of the differential diagnoses of anaphylaxis during antenatal period of pregnancy are not different from general population. History and clinical features would help to differentiate these conditions from below-mentioned conditions. However, during labor and delivery, conditions like pulmonary embolism and cardiac conditions may pose a challenge to differentiate from anaphylaxis.

First three trimesters before labor and delivery	Acute asthma, acute generalized urticaria, acute angioedema, syncope/fainting, panic attack, acute anxiety attack, postprandial syndromes, upper airway obstruction such as nonallergic angioedema, hereditary angioedema types 1, 2, and 3, shock Nonorganic diseases such as vocal cord dysfunction, hyperventilation, psychosomatic episode, Munchausen stridor
Labor and delivery	Pulmonary edema, pulmonary embolism, acquired and congenital cardiac conditions, hypotension caused by acute blood loss such as abruption placentae or uterine rupture, amniotic fluid embolism, cerebrovascular accidents, preeclampsia-eclampsia-associated symptoms such as laryngopathia gravidarum and seizures

16.6 Maternal and Fetal Outcomes in Anaphylaxis

Anaphylaxis during pregnancy, labor, and delivery can be devastating for the mother and the fetus. Maternal hypoxia, uterine hypoperfusion, umbilical vessel vasoconstriction, and peripheral fetal vasodilatation induced by histamine could lead to impairment of fetal regulation of cerebral flow and induce severe neurological damage.

Following hypoxia, there is a compensatory redistribution of blood with more blood supply preferentially reaching the vital organs like the brain, heart, placenta, and adrenal glands. There is also an increased oxygen uptake and tissue oxygen extraction from the fetal circulation along with decreased body movements. Once the compensatory mechanisms are overwhelmed by prolonged hypoxia; there can be hypoxic ischemic encephalopathy and permanent CNS damage in newborn despite survival in a case of anaphylactic shock [15].

Maternal cardiac arrest necessitates immediate cesarean delivery as fetal hypoxia can occur rapidly [16]. Chances of neonatal morbidity are high if anaphylaxis occurs during labor. Chances of neonatal survival are better if event occurs during cesarean section as fetal extraction can be performed concurrently along with maternal resuscitation [17].

16.7 Management of Anaphylaxis during Pregnancy

Anaphylaxis is a life-threatening medical emergency, and immediate diagnosis and prompt intervention are of paramount importance. The management guidelines for tackling anaphylaxis in pregnancy is similar to that of general population as mentioned below:

Diagnosis based on clinical features.

Immediately stop trigger factors/causative agents.

Ask for help. Call anesthetists/neonatologist if in the third trimester.

Administer adrenaline/epinephrine (0.5/1 mg/0.5–1 mL of 1:1000) intramuscularly every 10 min till pulse and BP improve. In severe cases with cardiovascular collapse, it may be given in IV-diluted solution slowly titrated against BP.

Monitor patient pulse oximeter and do ECG.

Maintain patent airway > supply O_2 (15 /min) by face mask. Consider intubation in severe cases.

Secure two large-bore cannula IV access.

Patients who have hypotension should be kept in head low or Trendelenburg position. If woman is in third trimester, keep left lateral position to avoid aorto-veno compression.

Crystalloids and colloids to treat hypotension.

Try to maintain BP above 90 mmHg and $SaO_2 > 90\%$.

Secondary therapy in the form of antihistaminics (chlorpheniramine 10–20 mg slow IV) and/or steroids (inj hydrocortisone 100–300 mg IV).

In the case of persistent hypotension, noradrenaline drip 4 mg of noradrenaline in 500 mL of dextrose at dose of 0.05–0.1 mg/kg/min.

Monitor ABG. In the case of acidosis, consider sodium bicarbonate 8.4% solution (0.5–1 mmol/kg) IV.

Bronchospasm to be treated with bronchodilators either aminophylline 250 mg IV over 20 min or salbutamol 250 mg IV slowly. Alternatively salbutamol can also be given by nebulizer.

If woman is in the third trimester, fetal heart rate monitoring by fetal monitor is to be done. Emergency delivery/CS may be required in some cases showing fetal distress.

There should be proper documentation of all events with timings and monitoring.

Once the patient is out of acute attack, when trigger factor is not known, further investigations in detail may be done to find out cause of anaphylaxis. In case delivery has not been contemplated, sonography and regular monitoring of the fetus are warranted. All possible trigger factors/drugs should be strictly avoided, and note of such orders should be mentioned in bold letters on all her outdoor and indoor papers. Also patient should be told to avoid these items/drugs to avoid similar event.

	Cardiovascular reaction/ hypotension	Bronchospasm/laryngoedema	Cutaneous reaction/ angioedema
Primary Rx	Epinephrine 1:1000 solution IV at 1 µg/min increased gradually to 2–10 µg/min IV fluid: 1 L of normal saline every 30 min or as per response	Oxygen by mask, maintain $SaO_2 > 90\%$ Albuterol 0.5 mL of 0.5% solution in 2.5 mL of isotonic saline by nebulizer or two puffs by metered dose inhaler every 15 min up to 3 doses	Diphenhydramine 1–2 mg/kg or 25–50 mg parenterally
Refractory cases/ recurrence	Norepinephrine 4 mg in 1 L 5% dextrose in water at 2–12 µg/min Glucagon 1 mg in 1 L 5% dextrose n water at 5–15 µg/min	Epinephrine 0.3–0.5 mL of 1:1000 solution IM, every 10 min as needed Methyl prednisolone 125 mg IV every 6 h	Hydrocortisone Inj

16.8 Medications and Dosage as per Clinical Features

Patient management as well as monitoring depends on the severity of the initial reaction and the treatment response.

Important and additional measures to be taken in pregnant females are as follows [16, 18–21]:

1. Emergency resuscitation team should include anesthesiologists, obstetrician, and a neonatologist along with the physician.
2. In the case of respiratory distress or vomiting, the woman should be placed on her left side, and lower extremities should be elevated to increase the venous return. The uterus may need to be displaced manually to the left. Supine position may lead to compression on the inferior vena cava, and hypotension and shock may get exacerbated leading to fatality.
3. Continuous electronic monitoring of the maternal blood pressure, heart rate, respiration, and oxygen saturation along with continuous electronic fetal monitoring is desirable. If electronic monitoring is not available, the maternal vital parameters as well as the fetal heart rate should be monitored as frequently as every 5 min.
4. The resuscitation team should be prepared for emergency cesarean section for anaphylaxis refractory to medical management or when there is fetal distress.

Biphasic or late phase reactions can occur several hours after primary episode of anaphylaxis. Though almost 90% of these reactions occur within the first 4 h, observation for 24 h has been advocated [9, 22].

16.9 Prevention

Avoiding the known allergen is the most important in prevention of further attacks of anaphylaxis. Patients must also be made aware of

cross-reacting allergens, particularly nuts and drugs. Patients with known allergies who are susceptible to anaphylaxis should be advised to wear Medic Alert bracelets. They should be advised to carry auto-injector kits of epinephrine (e.g., EpiPen) and should also be taught how to use the same by their physician in case the patient develops symptoms suggestive of anaphylaxis [23].

16.10 Immunotherapy

The 2011 Allergen Immunotherapy Practice Parameter has laid down certain guidelines for the potential benefits versus the risk involved in immunotherapy in pregnant patients [24]. Allergen immunotherapy is avoided during pregnancy because of the potential systemic reactions as well as the resultant effect on the fetus. The therapy can be discontinued if the dosage is below the therapeutic range once the pregnancy is diagnosed, but the maintenance dose can be continued during pregnancy.

16.11 Summary

Anaphylaxis although rare in pregnancy can have a potential catastrophic effect on the maternal as well as fetal health. The diagnosis is primarily clinical. Guidelines may be helpful in prompt diagnosis. A high index of suspicion, multidisciplinary approach by team of specialty doctors, as well as fetal monitoring during and after anaphylactic episode may play an important role in managing anaphylactic reaction in pregnancy with optimum outcome.

References

1. Sampson HA, Muñoz-Furlong A, Campbell RL, et al. Second symposium on the definition and management of anaphylaxis: summary report-Second National Institute of Allergy and Infectious Disease/Food Allergy and Anaphylaxis Network symposium. J Allergy Clin Immunol. 2006;117(2):391–7.
2. Muraro A, Roberts G, Worm M, et al. Anaphylaxis: guidelines from the European Academy of Allergy and Clinical Immunology. Allergy. 2014;69(8):1026–45.
3. Johansson SG, Bieber T, Dahl R, et al. Revised nomenclature for allergy for global use: report of the Nomenclature Review Committee of the World Allergy Organization, October 2003. J Allergy Clin Immunol. 2004;113(5):832–6.
4. Mulla ZD, Ebrahim MS, Gonzalez JL. Anaphylaxis in the obstetric patient: analysis of a state wide hospital discharge database. Ann Allergy Asthma Immunol. 2010;104:55–9.
5. Simons FE, Schatz M. Anaphylaxis during pregnancy. J Allergy Clin Immunol. 2012;130(3):597–606.
6. Chaudhuri K, Gonzales J, Jesurun CA, Ambat MT, Mandal-Chaudhuri S. Anaphylactic shock in pregnancy: a case study and review of the literature. Int J Obstet Anesth. 2008;17:350–7.
7. Kemp SF, Lockey RF. Anaphylaxis: a review of cause and mechanism. J Allergy Clin Immunol. 2002;110:341–8.
8. Dykewicz MS. Anaphylaxis and inflammation. Clin Allergy Immunol. 2002;16:401–9.
9. Kemp SF. Current concepts in pathophysiology, diagnosis and management of anaphylaxis. Immunol Allergy Clin N Am. 2001;21:611–34.
10. Philipson EH, Lang DM, Gordon SJ, et al. Management of group B streptococcus in pregnant women with penicillin allergy. J Reprod Med. 2007;52:480–4.
11. Sengupta A, Kohli JK. Antibiotic prophylaxis in cesarean section causing anaphylaxis and intrauterine fetal death. J Obstet Gynaecol Res. 2008;34:252–4.
12. Khan R, Anastasakis E, Kadir RA. Anaphylactic reaction to ceftriaxone in labour. An emerging complication. J Obstet Gynaecol. 2008;28:751–3.
13. Simons FER, Ardusso LRF, Bilo MB, et al. World allergy organization guidelines for the assessment and management of anaphylaxis. J Allergy Clin Immunol. 2011;127:593. e1–22.
14. Schwartz LB. Diagnostic value of tryptase in anaphylaxis and mastocytosis. Immunol Allergy Clin N Am. 2006;26(3):451–63.
15. Schatz M, Simons E, Dombrowski M. Anaphylaxis in pregnant and breastfeeding women. In: Basow DS, editor. UpToDate. Waltham, MA: UpToDate; 2012.
16. Suresh MS, LaToya Mason C, Munnur U. Cardiopulmonary resuscitation and the parturient. Best Pract Res Clin Obstet Gynaecol. 2010;24:383–400.
17. Di Chiara L. Vasopressin in the treatment of anaphylactic shock in paediatric congenital heart surgery. Eur Cardiol. 2008;4(1):111–2.
18. Heinly TL, Lieberman P. Anaphylaxis in pregnancy. Immunol Allergy Clin N Am. 2000;20:83–8.
19. Powrie RO. Anaphylactic shock in pregnancy. In: Belfort M, Saade G, Foley M, Phelan J, Dildy G, editors. Critical care obstetrics. 5th ed. Oxford (UK): Wiley-Blackwell; 2010. p. 596–604.
20. Kaneko K, Maruta H. Severe anaphylactoid reaction to ranitidine in a parturient with subsequent fetal distress. J Anesth. 2003;17:199–200.
21. Soar J, Perkins GD, Abbas G, Alfonzo A, Barelli A, Bierens JJLM, et al. European resuscitation council

guidelines for resuscitation 2010 section 8. Cardiac arrest in special circumstances: electrolyte abnormalities, poisoning, drowning, accidental hypothermia, hyperthermia, asthma, anaphylaxis, cardiac surgery, trauma, pregnancy, electrocution. Resuscitation. 2010;81:1400–33.
22. Kemp SF, Lockey RF. Anaphylaxis: a review of causes and mechanism. J Allergy Clin Immunol. 2002;110:341–8.
23. Huang S. A survey of Epi-PEN use in patients who have experienced anaphylaxis: a 3-year survey. Mayo Clin Procedure. 1994;69:16–23.
24. Cox L, Nelson H, Lockey R, Calabria C, Chacko T, Finegold I, et al. Allergen immunotherapy: a practice parameter third update. J Allergy Clin Immunol. 2011;127(suppl):S1–55.

Rhesus-Negative Mother

17

Richa Aggarwal and Amita Suneja

17.1 Introduction

Hemolytic disease of the newborn (HDN) secondary to rhesus alloimmunization was once a major contributor to perinatal morbidity and mortality. However, with the advent of anti-D immunoprophylaxis, the disease prevalence and the associated morbidity and mortality are markedly reduced with approximately 1–6 cases occurring every 1000 live births [1]. However, the disease is still a major problem in developing countries, including India with an estimated 56,700 cases of Rh-HDN occurring annually [2].

The incidence of Rh incompatibility varies by race and ethnicity. Approximately 15% of whites are Rh negative, compared to only 5–8% of African Americans and 1–2% of Asians [3]. Around 5% of the Indian population is Rh negative.

17.2 Pathophysiology

The RhD polypeptide is an integral membrane protein exclusively expressed on erythrocytes and identified as early as 38 days after conception [4]. With advancing gestational age, the volume and frequency of fetomaternal hemorrhage (FMH) increase with quoted rates being 3%, 12%, and 46% of women in each of the three successive trimesters [5]. The volume required to cause alloimmunization depends upon the immunogenicity of the Rh-positive erythrocytes and the immune responsiveness of the mother.

After fetomaternal hemorrhage in a Rh-negative mother, the initial response to D antigen exposure is slow and produces IgM anti-D. On subsequent antigenic exposure, there is rapid production of IgG anti-D antibodies which cross the placenta and coat D-positive fetal erythrocytes. Anti-D IgG is a non-agglutinating antibody which does not bind complement; hence, there is no intravascular hemolysis, but sequestration and subsequent destruction of antibody-coated red cells in the fetal liver and spleen is the mechanism of fetal anemia. Mild-to-moderate hemolysis is manifested as increased amniotic fluid indirect bilirubin levels. Severe hemolysis leads to increased red blood cell production by the spleen and liver of the fetus leading to portal hypertension and placental edema and eventually fetal ascites. Hepatomegaly, increased placental thickness, and polyhydramnios usually precede the development of fetal heart failure. Further liver damage decreases albumin production leading to the development of hydrops fetalis [1]. Coexistent ABO incompatibility causes rapid clearance of incompatible red cells and, hence, reduces the overall exposure to D antigen and, therefore, decreased risk of alloimunization.

R. Aggarwal · A. Suneja (✉)
Department of Obstetrics and Gynaecology, UCMS and GTB Hospital, Delhi, India

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17.3 Tests for Fetomaternal Hemorrhage

Kleihauer-Betke acid-elution test is the most widely used test for quantifying FMH [6]. Fetal RBCs mainly contain fetal hemoglobin (HbF), which is resistant to acid elution, while adult hemoglobin is acid-sensitive. The test is performed on the mother's blood; the blood undergoes acid elution and staining. The acid-resistant fetal cell stains red; maternal cells stain pink. However, its limitations include (1) cases of maternal hemoglobinopathies where maternal red cells carry excess fetal hemoglobin and (2) cases nearing term, when fetus has already started to produce hemoglobin A [7].

Flow cytometry is an alternative technique for quantifying the size of FMH [8]. Its results are more accurate and more reproducible compared to Kleihauer test, and since it detects RhD-positive cells, it is particularly useful in patients with high HbF levels. It is used when a Kleihauer screening test indicates a large FMH which requires accurate quantification and follow-up. The rosetting technique is another serological method used to quantify FMH of RhD-positive red cells.

A 300 μg intramuscular dose of anti-D immunoglobulin is sufficient for 15 mL of fetal red cells or 30 mL of fetal whole blood. The additional dose of anti-D should be calculated on the basis of extra 20 μg for each mL of fetal cells present. A maternal sample should be collected 72 h after the intramuscular administration of anti-D (48 h of intravenous anti-D) to assess the removal of fetal cells following a large fetomaternal hemorrhage. More anti-D may be necessary if fetal cells remain.

No more than five units of rhesus immune globulin should be administered by the intramuscular route in one 24-h period. If using an intravenous preparation, two ampoules (600 μg) may be given every 8 h [1].

17.4 Management of Sensitized Antenatal Mother

A titer of more than 1:4 is considered sensitized. Once Rh antibodies are detected, the next step is to determine the concentration of antibodies and the likelihood of HDFN. It is important to find out if the antibodies are immune or passive, and history of any prior anti-D injection within last 12 weeks should be elicited.

17.4.1 Distinguishing Between Passive and Immune Anti-D

The concentration of passive anti-D Ig in maternal samples post-prophylaxis rarely exceeds 1:4 following a dose of 300 μg. Passive anti-D Ig can be detected in the blood within minutes after intramuscular injection, reaches peak concentration in 3–7 days, and can be detected for more than 12 weeks by an indirect antiglobulin test (IAT). Immune anti-D on the other hand becomes detectable only after 4 weeks of exposure to D-positive cells and reaches a peak concentration after 6–8 weeks, if there is no further exposure [9].

On serial monitoring, if the anti-D level is falling, it is probably passive, whereas if it is steady or rising, it is probably immune. Prophylactic anti-D should continue unless it is established that the anti-D is immune.

A critical titer is defined as the titer significantly associated with development of fetal hydrops. This titer varies with institutions based on clinical correlation with hemolytic disease of the newborn and is between 8 and 32 in most centers. In Europe and the United Kingdom, the concentration of anti-D is measured in IU/mL. A threshold value of 15 IU/mL has been recommended for invasive testing as values below this are usually associated with only mild hemolytic disease of the newborn [10].

Figure 17.1 summarizes the antenatal management of a Rh-negative pregnant woman with positive antibody screen to Rh antibodies.

17.5 Timing of Delivery

In the case of a Rh-negative non-sensitized woman, it is reasonable to continue pregnancy up to 40 weeks' gestation. However, if the pregnancy continues beyond 40 weeks of gestation, a second dose of Rh Ig should be considered [11].

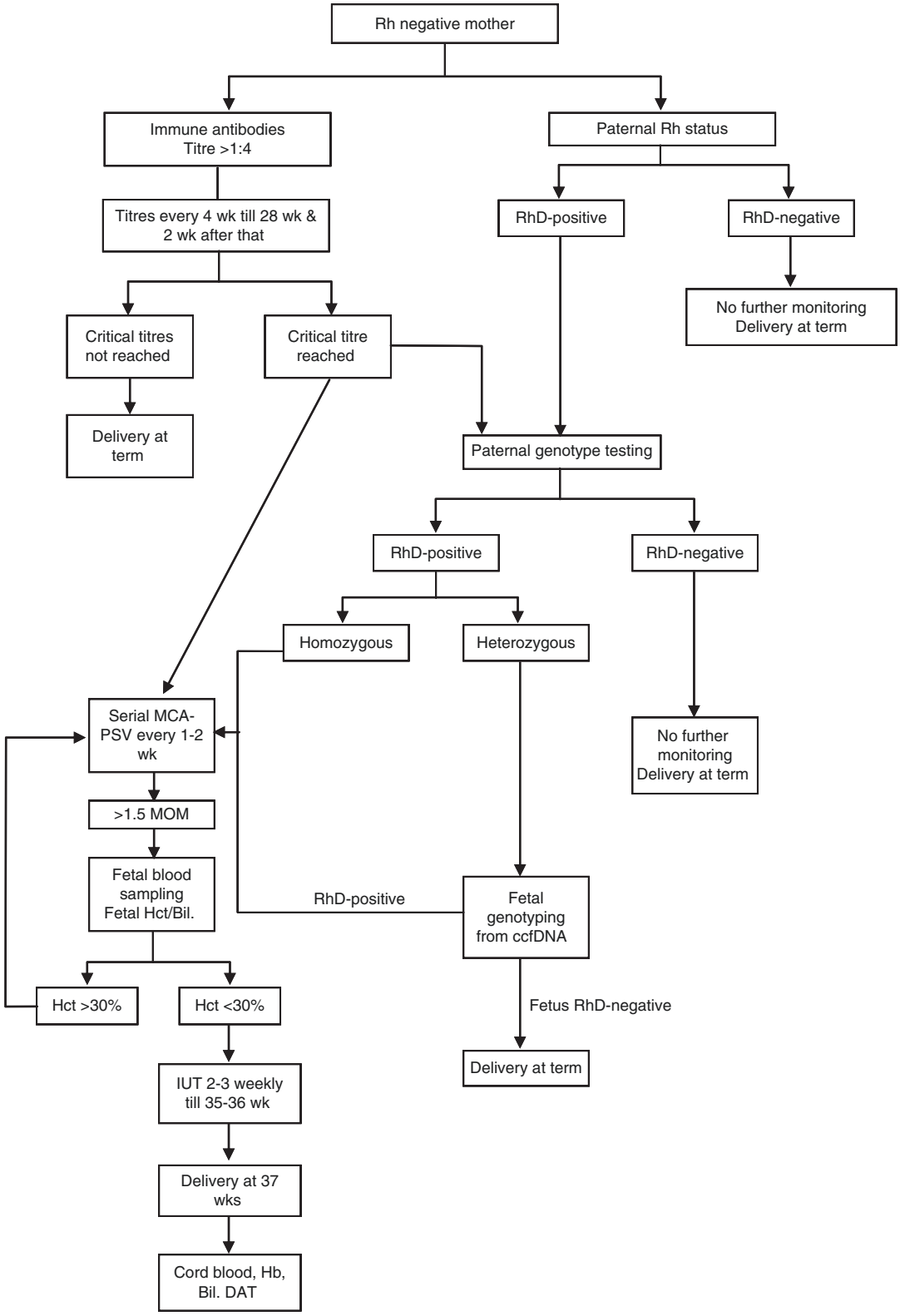

Fig. 17.1 Flow chart depicting the antenatal management of isoimmunized Rh-negative mother

With mild fetal hemolysis, pregnancy may be continued till 37–38 weeks of gestation and then delivery planned. Induction may be considered earlier if fetal lung maturity is documented by amniocentesis [3].

Fluorescence depolarization techniques (TDx-FLM) should be avoided for conforming fetal lung maturity, as values may be falsely elevated due to excess bilirubin; the lamellar body count or lecithin-sphingomyelin ratio is not affected by increased bilirubin and is preferable [1].

With severely sensitized pregnancies requiring multiple invasive procedures, the risks of repeated cord blood sampling and transfusions must be weighed against neonatal risks associated with prematurity. Since the neonatal survival rate after 32 weeks of gestation is more than 95% in most neonatal units, transfusion may be performed at 30–32 weeks of gestation, with delivery planned at 32–34 weeks of gestation after administering maternal steroid. However, many clinicians recommend intrauterine transfusions up to 36 weeks of gestation if feasible, in order to limit neonatal morbidity, and delivery be planned between 37 and 38 weeks of gestation [12].

17.6 Management in the Delivery Room

17.6.1 Rh-Negative Mother Non-isoimmunized

First stage of labor—sweeping and stretching of the membranes should be avoided as it increases the amount of fetomaternal hemorrhage. The maternal and fetal monitoring remains the same as per the hospital protocol for low-risk pregnancy.

17.6.1.1 Active Management of Third Stage

During the active management, fetomaternal transfusion is increased. However, considering the poor availability of the Rh-negative blood and the potential for postpartum hemorrhage, active management of the third stage is not discouraged.

17.6.1.2 Cord Clamping

Though still controversial, and no recommendations in place regarding timing of cord clamping in Rh-negative pregnancy till date, studies have shown that delayed cord clamping is associated with decreased postnatal exchange transfusion needs, an improvement in the hemoglobin level at birth, and longer delay between birth and first transfusion with no significant differences in maximum bilirubin levels and intensive phototherapy rates or total phototherapy treatment duration as compared to neonates with immediate cord clamping [13].

17.6.1.3 Intrapartum Fetal Sampling

The cord blood sample should be collected to determine the baby's D group to find out if the mother requires post-delivery prophylactic anti-D Ig. Direct antiglobulin test (DAT) on the cord samples of D-positive babies born to non-isoimmunized D-negative women is not recommended routinely because following routine antenatal anti-D prophylaxis (RAADP), anti-D Ig can cross the placenta and bind to fetal D antigen sites, and therefore, 3–6% false-positive DAT have been reported leading to unnecessary additional investigations [14]. The baby should be monitored for hyperbilirubinemia.

17.6.1.4 Postpartum Prophylaxis

The risk of sensitization if an Rh-negative mother does not receive postpartum anti-D IgG prophylaxis after an Rh-positive baby is 12–16%, which is reduced to 1.6–1.9% following postpartum prophylaxis. In 99.67% of cases, the volume of fetomaternal hemorrhage occurring at the time of delivery is less than 15 mL [11].

Certain risk factors like traumatic deliveries including cesarean section, manual removal of the placenta, stillbirths and intrauterine deaths, abdominal trauma during the third trimester, and twin pregnancies are associated with large FMH, but more than 50% of the cases of large FMH occur in women without identified risk factor.

Postpartum prophylaxis includes administration of 300 μg of anti-D to all D-negative with D-positive babies which is sufficient to protect against a FMH of 30 mL of fetal whole blood or

15 mL of fetal red cells. If a full dose of anti-D has been given within 21 days before delivery, there is no need to repeat it after birth if excess FMH has been excluded [15].

17.6.1.5 Quantification of Fetomaternal Hemorrhage

About 0.3% of women have an FMH greater than 15 mL which will not be covered by 300 μg of anti-D Ig and, hence, would be inadequately protected [11]. Routine screening of all women at the time of delivery for excessive fetomaternal hemorrhage should therefore be undertaken. A Kleihauer screening test should be performed within 2 h of delivery to identify RhD-negative women with a large FMH who require additional anti-D Ig [6]. The maternal sample should be collected after 30–45 min to give time for any FMH to be dispersed in the maternal circulation [16].

Where there is no facility to perform Kleihauer testing to quantify the FMH at delivery, it is reasonable to administer a standard postnatal dose of 300 μg anti-D Ig [6]. For successful immunoprophylaxis, anti-D Ig should be given as soon as possible after the potentially sensitizing event but always within 72 h. If it is not given before 72 h, it should still be given as there may be some protection when anti-D is given up to 13 days or even 28 days after a potentially sensitizing event [11]. Ideally, anti-D Ig should be given into the deltoid muscle. Women with bleeding disorders should receive anti-D Ig subcutaneously or by intravenous route. In cases of very large FMH, i.e., in excess of 80 mL, intravenous anti-D should be considered. Consent should be obtained and recorded in the case notes.

If the pregnancy is nonviable and no sample can be obtained from the baby, *prophylactic anti-D should be administered to the woman*, *if she is D-negative* [1].

The administration of rhesus immune globulin after a postpartum tubal ligation is controversial. Keeping in mind the possibility of a new partner in conjunction with the availability of in vitro fertilization, it seems prudent to administer rhesus immune globulin in these women. In addition, RhD sensitization would limit the availability of blood products if the patient later requires a transfusion [1].

17.6.2 Rh-Negative Mother Isoimmunized

The mode of delivery is dependent on standard obstetric grounds. Prior intrauterine therapy is not an indication for an elective cesarean section [17].

17.6.2.1 Intrapartum Fetal Sampling

If the mother is positive for immune RhD antibodies, cord blood should be tested for RhD antigen. A DAT along with hemoglobin and bilirubin levels should be performed on cord blood sample, and baby should be observed for clinical signs of jaundice [6].

Babies who have received in utero transfusion of D-negative blood for the prevention of HDFN may type as D-negative on testing for several months after birth. In these cases, when D-positive cells are released into the circulation, there may be further red cell destruction, and therefore, the baby's hemoglobin and bilirubin concentrations should be regularly monitored, and hence, early discharge is not advisable [16].

17.6.2.2 Postpartum Prophylaxis

Women who are already sensitized to RhD should not be given anti-D Ig.

17.6.2.3 Neonatal Follow-Up

Overall survival is reported to be 84%. Survival of nonhydropic fetuses (92%) is significantly better compared with hydropic fetuses (70%) [1]. Suppression of erythropoiesis is common after several intravascular transfusions. These infants are born with extremely low reticulocytes despite a low packed cell volume and normal erythropoietin values. Elevated levels of passively acquired maternal antibodies in the neonatal circulation along with suppression of fetal bone marrow production of red cells result in need for several top-up transfusions in the initial 1–3 months. Weekly neonatal hematocrit and reticulocyte counts should therefore be assessed until recovery of hematopoietic

function is documented. Threshold hematocrit values of less than 30% in the symptomatic infant or less than 20% in the asymptomatic infant have been suggested as indication for transfusion [1].

17.6.2.4 Long-Term Follow-Up

Cerebral palsy and developmental delay are relatively common in fetuses with hemolytic disease of the newborn when compared with unaffected infants, although a normal outcome is seen in more than 90% of cases [18]. Sensorineural hearing loss is more common in infants affected by HDFN because of prolonged exposure to elevated levels of bilirubin and its toxic effect on the developing eighth cranial nerve. Therefore, newborn screening for hearing loss is warranted with follow-up screening at 1 and 2 years of age considered [2].

17.6.3 Patient Coming in Labor with Positive Indirect Antiglobulin Test

A patient may present for the first time in labor with a positive ICT report without titers done and no further workup. The maternal sample need not be sent for quantification at this time, but the baby should be monitored for signs of HDFN as treatment decisions will be based on the severity of neonatal anemia and/or jaundice. The results of quantification are unlikely to have a major influence on clinical decisions at this stage and are not available as quickly as measures of hemoglobin and bilirubin [14].

References

1. Moise KJ. Management of rhesus alloimmunization in pregnancy. Obstet Gynecol. 2002;100:600–11.
2. Mathai SS, Venkatnarayan K. Jaundice in the newborn. In: Gupta P, Menon PSN, Ramji S, Lodha R, editors. PG textbook of pediatrics. 1st ed. New Delhi: Jaypee Brothers Medical Publishers(P) Ltd; 2015. p. 435–45.
3. American College of Obstetricians and Gynecologists. Management of alloimmunization during pregnancy. In: ACOG practice bulletin no. 75. Washington, DC: American College of Obstetricians and Gynecologists; 2006.
4. Recommendations for the use of anti-D immunoglobulin. Prescribers J. 1991;1:137–45.
5. Bowman JM, Pollock JM, Penston LE. Fetomaternal transplacental hemorrhage during pregnancy and after delivery. Vox Sang. 1986;51:117–21.
6. Royal College of Obstetricians and Gynaecologists (RCOG). The management of women with red cell antibodies during pregnancy. Green-top Guidelines no. 65 May 2014.
7. Cunningham FG, Leveno KJ, Bloom SL, Spong CY, Dashe JS, Hoffman BL, Casey BM, Sheffield JS. Fetal disorders. In: Williams obstetrics. 24th ed. New York: McGraw Hill Education; 2014. p. 306–20.
8. Johnson PR, Tait RC, Austin EB, Shwe KH, Lee D. Flow cytometry in diagnosis and management of large fetomaternal haemorrhage. J Clin Pathol. 1995;48:1005–8.
9. Mollinson PL, Engelfreit CP, Contreras M. Development of immune anti-D Blood transfusion in clinical medicine. 10th ed. Oxford: Blackwell; 1997. p. 82.
10. Nicolaides KH, Rodeck CH. Maternal serum anti-D antibody concentration and assessment of rhesus isoimmunisation. Br Med J. 1992;304:1155–6.
11. Fung Kee Fung K, Eason E, Crane J, et al. Prevention of Rh alloimmunization. J Obstet Gynaecol Can. 2003;25(9):765–73.
12. Boggs TR Jr. Survival rates in Rh sensitizations: 140 interrupted versus 141 uninterrupted pregnancies. Pediatrics. 1964;33:758–62.
13. Garabedian C, Rakza T, Drumez E, et al. Benefits of delayed cord clamping in red blood cell alloimmunization. Pediatrics. 2016;137(3):e20153236.
14. British Committee for Standards in Haematology (BCSH), White J, Qureshi H, Massey E, Needs M, Byrne G, Daniels G, Allard S. Guideline for blood grouping and red cell antibody testing in pregnancy. Transfus Med. 2016;26(4):246–63. https://doi.org/10.1111/tme.12299.
15. Hartwell E. Use of Rh immune globulin. ASCP Practice Parameter. Am J Clin Pathol. 1998;110:281–302.
16. Qureshi H, Massey E, Kirwan D, Davies T, Robson S, White J, Jones J, Allard S, British Committee for Standards in Haematology (BCSH). Guideline for the use of anti-D immunoglobulin for the prevention of haemolytic disease of the fetus and newborn. Transfus Med. 2014;24(1):8–20.
17. Kumar S, Regan F. Management of pregnancies with RhD alloimmunisation. BMJ. 2005;330(7502):1255–8.
18. Moise KJ, Whitecar PW. Antenatal therapy for haemolytic disease of the fetus and newborn. In: Hadley A, Soothill P, editors. Alloimmune disorders in pregnancy. Anaemia, thrombocytopenia and neutropenia in the fetus and newborn. Cambridge, UK: Cambridge University Press; 2002. p. 141–63.

18 Vaginal Bleeding in Early Pregnancy

Kiran Aggarwal

18.1 Introduction

Vaginal bleeding is a common complication in early pregnancy and is seen in 20–25% of pregnancies in the first trimester. Fifty percent of pregnancies which have bleeding in early gestation ultimately abort [1]. It is a commonly encountered presentation in early pregnancy units. Any woman in reproductive age group presenting with bleeding per vaginum should be suspected to have pregnancy or pregnancy-related complications. The presentation may vary from a stable patient to a life-threatening condition as may happen in ectopic pregnancy, incomplete abortion, septic abortion, or trauma. Careful history taking and examination accompanied with ultrasound and beta hCG values help in appropriate management and optimal outcomes.

In this chapter we present the causes of bleeding in early pregnancy, relevant history, and examination to be done followed by investigations and diagnosis. Overall management of the cases would be discussed together last.

K. Aggarwal (✉)
Lady Hardinge Medical College, New Delhi, India

18.2 Causes of Bleeding in Early Pregnancy

The bleeding in early pregnancy may be because of pregnancy-related causes like abortion, ectopic, etc. or those incidentally associated with normal pregnancy like traumatic tears, polyps, infections, etc. Miscarriages are the most common cause of bleeding in early pregnancy (Table 18.1).

18.2.1 History

Like for any medical disease, first the condition of the patient should be assessed. If she is hemodynamically compromised, resuscitative measures like starting IV fluids, oxygen, and arranging blood should be done. Then a brief quick relevant history should be taken.

History of amenorrhea with acute abdominal pain/collapse and adnexal tenderness may indicate a ruptured ectopic. Similarly fever, abdominal pain, foul smelling discharge, hypotension with pallor, abdominal tenderness, and peritonitis may indicate a septic abortion with or without septic shock.

If patient is stable, then relevant history should be taken to arrive at a diagnosis.

History of amenorrhea is important. Last menstrual period should be documented and gestational age should be calculated. It is important to note that patient may present with ectopic pregnancy without any amenorrhea. Duration and flow of last

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Table 18.1 Differential diagnosis of bleeding in early pregnancy

Causes related to pregnancy and uterus	Associated causes with normal pregnancy (Cervix, vaginal, and vulval causes)
• Abortion: Threatened, missed, and incomplete abortion • Septic abortion • Sub chorionic bleeding • Ectopic pregnancy • Molar pregnancy • Idiopathic bleeding in pregnancy	• Vulval, vaginal, and cervical infections and ulcerations • Polyps • Malignancy with pregnancy like cervical/vaginal malignancy • Postcoital tear/iatrogenic trauma • **Bleeding which may not be from genital tract:** • Hemorrhoidal (piles) • Urinary tract infections/ bladder stone

menstrual period is important to note whether it was a normal period or an episode of bleeding only. It can also be implantation bleeding.

Cramping pain may signify process of expulsion of products/clots as in miscarriage.

The amount, color, and duration of bleeding with history of passage of clots should be asked to estimate blood loss. Fresh blood may signify threatened or incomplete abortion. Vaginal, cervical lesions and trauma will also cause fresh red-colored bleeding. Old brownish bleeding may be seen in missed abortion, molar pregnancy, or subchorionic bleeding.

History of passage of any products is important. It may signify products or may be a decidual cast in ectopic pregnancy rarely. Passage of grapelike products denote vesicular mole.

Any other systemic complaints should be evaluated like burning micturition, bowel symptoms like constipation, inability to pass flatus, vomiting, decreased urine output, etc. This helps in diagnosing septicemia, subacute intestinal obstruction, etc.

Associated history of interference, instrumentation, should be taken.

History of drug intake like abortifacients, nonsteroidal anti-inflammatory drugs, and hormones is important. History of assisted reproduction methods may point toward heterotopic pregnancy.

Past history of ectopic, abdominal pain, pelvic inflammatory disease, and abortions should be taken as they are risk factors for repeat disease in this pregnancy.

18.2.2 Signs and Symptoms

One should be very considerate and gentle in examining the patient. Patient's general condition, pallor, tachycardia, blood pressure, chest, and cardiovascular system should be evaluated quickly.

Abdomen should be palpated for uterine size if palpable which is normally felt per abdomen after 12 weeks. Any associated mass-like fibroids or ovarian should be noted. Tenderness, guarding, doughy abdomen, and rigidity if present may signify peritonitis, or hemoperitoneum. Shifting dullness should be seen.

Local examination should be done. Vulva is seen externally for any disease and the amount of bleeding. Per speculum examination may reveal any vaginal or cervical lesions like cervicitis, vaginitis, polyps, cervical cancer, etc. Presence of postcoital tear should be looked for. Dilated cervical os with blood and/or products coming from it may signify incomplete or inevitable abortion.

Any products lying in the vagina or being extruded from the cervix are noted. They should be gently removed with ovum forceps and examined for chorionic villi and send for histopathological examination.

Bimanual pelvic examination would reveal uterine size, softness, mobility of the uterus, adnexal masses, and cervical motion tenderness. It should be assessed whether size of the uterus corresponds with the period of gestation or not. It may be smaller in complete abortion, incomplete abortion, or missed abortion. Enlarged uterus with closed os with bleeding will indicate threatened abortion, or if the uterus is soft and normal size, it may indicate complete abortion. Bulky uterus with os open or closed signifies inevitable or incomplete abortion. Normal size uterus with tender adnexal mass would point toward ectopic.

The uterus may be larger in vesicular mole or multiple pregnancy or if associated with fibroids.

Features of peritonitis, intestinal obstruction with a toxic patient with infected discharge from the os, or products lying in the cavity may suggest septic abortion or intestinal injury following perforation. If handheld Doppler is available after 11–12 weeks of gestation, fetal cardiac activity can be confirmed.

Pouch of Douglas should be palpated for any collection fullness, nodules. Any foreign body in the vagina or cervical canal should be also noted for any attempted induced abortion.

18.2.3 Laboratory Investigations

Hemoglobin is done to assess the blood loss and anemia. Blood group and Rh should be known. Increased total leukocyte count and differential count with increased poly morphs will indicate sepsis.

Kidney function tests may be deranged in sepsis and blood loss. Coagulation profile should be done in massive hemorrhage and sepsis.

Urine for pregnancy test to confirm pregnancy should be done. Urine microscopic examination should be done to find pus cells and bacteria for urinary tract infection.

If clinical examination and history is not conclusive, then ultrasound and beta hCG may be done.

All Rh-negative women with bleeding should receive $Rh_O(D)$ immune globulin, to protect against development of Rh alloimmunization.

Ultrasound examination: Transvaginal sonography is a valuable tool to assess location and viability of early pregnancy. For a normal pregnancy by 5 weeks of period of gestation, a 5 mm intrauterine sac should be visible. It is important to distinguish it from pseudogestational sac of ectopic pregnancy [1]. Yolk sac is visible at 6 weeks when gestational sac is greater than 10 mm in diameter. Cardiac activity should be present when the embryo exceeds 5 mm in length [1]. If these discriminatory criterions are not met, it may indicate pregnancy failure, and evaluation should be done again after 1 week to confirm findings of a loss.

Patients with positive urine pregnancy test with bleeding per vaginum, transvaginal ultrasound tells us about intrauterine pregnancy and its viability. It also reveals any retained products of conception in incomplete abortion.

Empty uterus on ultrasound with adnexal mass with positive urine pregnancy test is almost diagnostic of ectopic pregnancy (Fig. 18.1). Hemoperitoneum can be easily picked by ultrasound examination. Molar pregnancy, subchorionic hematoma, multiple gestations, and demise of one of the twins are other important findings in work up of an early pregnancy bleeding which can be confirmed by ultrasound.

If urine pregnancy test is positive and the uterus is empty with no adnexal mass, then the term pregnancy of unknown location is used. In this situation differential can be complete abortion, emerging normal pregnancy, or ectopic pregnancy.

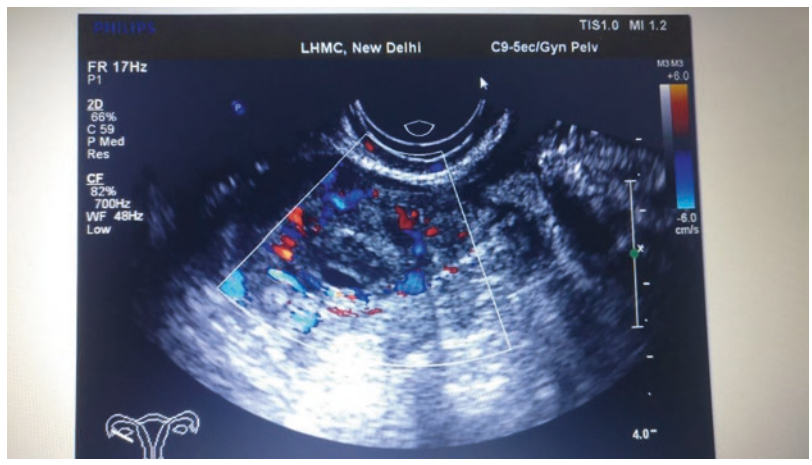


Fig. 18.1 Ectopic pregnancy

18.2.4 Measurement of Beta Unit of Human Chorionic Gonadotropin

The beta hCG can be detected in the blood around 22–23 days of last menstrual period. The routinely available urinary pregnancy test kits detect beta hCG of 25 miu/ml [2]. This test is positive in most of the ectopic pregnancies.

The level of beta hCG beyond which an intrauterine sac should certainly be seen on ultrasound scan is called discriminatory level. This varies in different reports. A level of 1500–200 IU/l is generally taken at which pregnancy should be visible on transvaginal scan.

If the pregnancy location is not seen, then diagnosis of ectopic is almost certain. Complete abortion, failing intrauterine pregnancy, and very early twin pregnancy can be the other differentials.

If the values are below the discriminatory levels, then pregnancy won't be visible on ultrasonography and may be called a pregnancy of unknown location. Serial beta hCG levels may be needed to arrive at a diagnosis. For a normal developing intrauterine pregnancy, beta hCG rise is seen to be at least 50% in 48 h in almost 99% of normal pregnancies [3]. Twenty percent of ectopics also may show beta hCG rise of 50% in 48 h [4].

Slowly rising titers may indicate ectopic pregnancy or failing intrauterine pregnancy. Falling beta hCG may indicate nonviable intrauterine pregnancy or resolving ectopic pregnancy. The rate of decrease for a spontaneous abortion is dependent on the initial β-hCG level. The average rate of decrease in the first 48 h is typically greater than 70%. A rate of decrease less than 21–35% (depending on initial level) is inappropriate, and an ectopic pregnancy should be suspected [4]. One should think of gestational trophoblastic neoplasia if the titers are very high.

18.2.5 Diagnosis and Management

18.2.5.1 Ectopic Pregnancy

Ectopic pregnancies constitute 1–2% of all first trimester pregnancies. Risk factors for ectopic pregnancies are prior tubal pregnancy, surgeries on fallopian tube like recanalization, sexually transmitted diseases, pelvic inflammatory disease and other infections like appendicitis, and in our country tuberculosis. Infertility and ART also increase the risk of ectopic pregnancies.

At a value of 1500–2000 mIU/mL of beta hCG, intrauterine gestational sac is always seen [1]. If at this level sac is not seen on the ultrasound, a diagnosis of ectopic is almost confirmed. Very early multiple gestations may have higher beta hCG values early on before pregnancy is visible. Heterotopic pregnancy should also be thought of [5].

Adnexal mass, cervical movement tenderness, free fluid in the pelvis with empty uterus, and a positive urine pregnancy test are almost diagnostic of ectopic pregnancy. Clinical history and examination should always be correlated.

Management of ectopic pregnancy can be medical or surgical or expectant.

18.2.6 Medical Management

Methotrexate is used for medical management of ectopic pregnancy and can be used when following situations exist (Table 18.2). Contraindications of the drug also should be known before using it.

Single-dose methotrexate treatment [6].

Table 18.1 Prerequisites for medical management of ectopic pregnancy

Prerequisites for medical management	Contraindications to use of methotrexate
Patient is hemodynamically stable with few or no complaints	Deranged liver enzymes, complete blood count, and platelet count
No free fluid in the abdomen and pelvis	Any known allergy to the drug
Gestational sac less than 3.5 cm	Breast feeding and renal disease
Absent embryonic cardiac activity	
Beta hCG levels less than 5000 IU/L	

Complete blood count, kidney function test, and liver function are done.

Day 1 beta hcg levels methotrexate 50 mg/m^2.

Day 4 and 7 beta hcg levels.

There should be a fall of 15% between day 4 and 7. Follow the levels weekly till normal.

After methotrexate administration, the β-hCG level should decrease by at least 15% from day 4 to day 7. If the β-hCG levels do not decrease by at least 15% from day 4 to day 7 or if it plateaus or increases after the first week following injection, it is taken as treatment failure. In this case, additional methotrexate administration or surgical intervention is required [7].

18.2.7 Surgical Management

This should be done when patient is not stable, there is intraperitoneal bleed, cardiac activity is present, sac is big, or any contraindication to medical treatment exists.

Laparoscopy is the procedure of choice unless patient is hemodynamically compromised. Salpingostomy is done when tube is conserved, and salpingectomy is a radical procedure when the affected tube is removed.

18.2.8 Expectant Management

Expectant management can be done when beta hCG levels are less than 1000 IU/L and on decreasing trend. Sac is very small or not seen. There should not be any symptoms and no free fluid in the abdomen. Patient should be counseled that in spite of the fall, she may need surgical management.

18.3 Early Pregnancy Loss

This occurs in 10% of clinically determined pregnancies. Most common cause of first trimester abortions is chromosomal abnormalities. Previous history of abortions and advancing maternal age are important risk factors [8, 9].

Detailed history, clinical examination, and correlation with ultrasound are important. When needed beta hCG levels should be done to make a diagnosis of pregnancy loss. If the patient wishes is stable and wants to wait for final decision, her opinion should be considered.

Threatened abortion: Uterine bleeding with viable intrauterine early pregnancy before 20 weeks with closed cervical os is diagnostic of threatened miscarriage. Treatment is expectant and watchful observation. Ninety to ninety-six percent viable pregnancies with bleeding continue [10].

Inevitable miscarriage: If cervix is ballooned out or dilated or the products can be felt through the cervical canal with increasing bleeding and pain, then it is inevitable that the pregnancy is going to aborted. Management is expectant, or medical or surgical intervention may be taken depending on the amount of bleeding and the condition of the patient.

18.3.1 Complete Miscarriage

If the conceptus is expelled out totally, then patient would complain of less bleeding and pain. On examination os will be generally closed and uterus would be just bulky or normal size. Ultrasound reveals empty uterus with no adnexal mass. This would lead to a diagnosis of complete abortion.

It may be confused with an ectopic pregnancy or pregnancy of unknown location. Demonstration of chorionic villi in the tissue that was passed out, falling beta hCG levels and improvement of symptoms, indicates complete abortion. No treatment is needed; if there is a doubt, then serial beta hcg levels should be followed till they are negative.

Incomplete abortion: os is open; products of conception are inside the uterine cavity. Uterine size may be smaller than expected according to the period of gestation. Severe bleeding may occur as the uterus is not well contracted and patient may present in shock. Generally if bleeding is there, surgical evacuation should be there. With minimal bleeding medical management can be done.

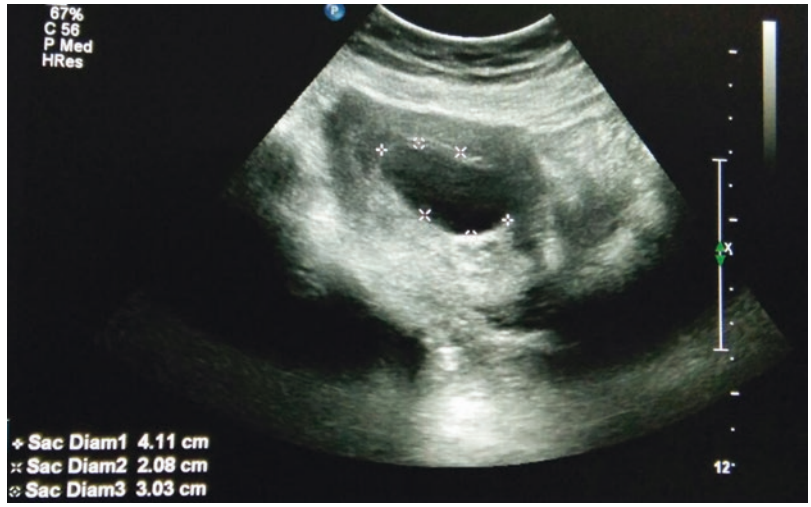


Fig. 18.2 Missed abortion

Missed abortion. It is the demise of the embryo or fetus in utero before 20 the week of pregnancy and retention of products of conception for a long time. Patient may complain loss of symptoms of pregnancy. Brownish blood discharge may be seen. Ultrasound reveals an embryonic gestational sac or fetal node without cardiac activity (Fig. 18.2). Expectant or a medical or surgical treatment may be given.

18.4 Expectant Management for Abortion

This constitutes waiting without any intervention for the products to be expelled. This seems to be more successful in symptomatic patient and those with history of passage of products rather than missed abortion. If the patient is stable and there is no fever, anemia, infection, or significant bleeding, expectant management may be tried. Expectant management is successful in achieving complete expulsion in approximately 80% of women [11].

18.5 Medical Management

Misoprostol a prostaglandin E1 analogue can be used as an alternative to surgical evacuation or watchful expectancy.

18.5.1 Misoprostol for Early Pregnancy Loss

Eight hundred micrograms per vaginally is used. Dose can be repeated if needed. After 7–14 days, complete expulsion may be evaluated by ultrasound.

Women who are Rh(D) negative and unsensitized should receive Rh(D)-immune globulin within 72 h of the first misoprostol administration [12].

18.5.2 Surgical Evacuation

If the patient is bleeding or has infection, then surgical evacuation may be done. Suction evacuation is the procedure of choice. Manual vacuum aspirator may be used [13, 14]. Contraception after early pregnancy loss: patient may start hormonal contraception immediately after early pregnancy loss. Intrauterine copper device may be inserted after surgical evacuation.

18.6 Other Causes

Other causes like vaginitis, trauma, malignancies, polyps, etc. can be seen on visual inspection and treatment may be done accordingly.

Ectropion: this is eversion of columnar epithelium and may be bleeding on coitus or pelvic examination or per speculum examination. Per say no treatment is needed.

Implantation bleeding: this may occur at the time of the expected menses after fertilization and is usually spotting and may be because of fertilized egg implanting in the deciduas.

18.7 Conclusion

Usually early pregnancy bleeding is associated with adverse pregnancy outcomes like miscarriage IUGR, preterm birth, etc. With light spotting or light bleeding, prognosis is usually good, especially in the early gestation like before 6–8 weeks.

Threatened abortions with documented viable pregnancy usually have a good prognosis. When clinical diagnosis is difficult, transvaginal scan and beta hCg values should be seen. Medical management of ectopic is effective only if patient is ready for follow-up. Surgical evacuation is a time-tested quick procedure to empty the uterus. Psychological support to the patient is utmost needed.

References

1. Paspulati RM, Bhatt S, Nour SG. Sonographic evaluation of first-trimester bleeding [published correction appears in Radiol Clin North Am. 2008;46(2):437]. Radiol Clin N Am. 2004;42(2):297–314. http://www.sciencedirect.com/science/journal/00338389
2. ACOG Committee on Gynecologic Practice. Avoiding inappropriate clinical decisions based on false-positive human chorionic gonadotropin test results. Obstet Gynecol. 2002;100(5 pt 1):1057–9.
3. Barnhart KT, Sammel MD, Rinaudo PF, Zhou L, Hummel AC, Guo W. Symptomatic patients with an early viable intrauterine pregnancy: HCG curves redefined. Obstet Gynecol. 2004;104(1):50–5.
4. Barnhart KT. Clinical practice. Ectopic pregnancy. N Engl J Med. 2009;361(4):379–87.
5. Barnhart K, Mennuti MT, Benjamin I, et al. Prompt diagnosis of ectopic pregnancy in an emergency department setting. Obstet Gynecol. 1994;84:1010–5.
6. Stovall TG, Ling FW, Gray LA. Single-dose methotrexate for treatment of ectopic pregnancy. Obstet Gynecol. 1991;77:754–7.
7. Lozeau AM, Potter B. Diagnosis and management of ectopic pregnancy [published correction appears in Am Fam Physician. 2007;75(3):312]. Am Fam Physician. 2005;72(9):1707–14.
8. Barnhart KT, Gosman G, Ashby R, Sammel M. The medical management of ectopic pregnancy: a meta-analysis comparing "single dose" and "multidose" regimens. Obstet Gynecol. 2003;101(4):778–84.
9. Nybo Andersen AM, Wohlfahrt J, Christens P, Olsen J, Melbye M. Maternal age and fetal loss: population based register linkage study. BMJ. 2000;320:1708–12.
10. Tongsong T, Srisomboon J, Wanapirak C, et al. Pregnancy outcome of threatened abortion with demonstrable fetal cardiac activity: a cohort study. J Obstet Gynecol (Tokyo 1995). 1995;21:331.
11. Luise C, Jermy K, May C, Costello G, Collins WP, Bourne TH. Outcome of expectant management of spontaneous first trimester miscarriage: observational study. BMJ. 2002;324:873–5.
12. Zhang J, Gilles JM, Barnhart K, Creinin MD, Westhoff C, Frederick MM. A comparison of medical management with misoprostol and surgical management for early pregnancy failure. National Institute of Child Health Human Development (NICHD) Management of Early Pregnancy Failure Trial. N Engl J Med. 2005;353:761–9.
13. Goldberg AB, Dean G, Kang MS, Youssof S, Darney PD. Manual versus electric vacuum aspiration for early first-trimester abortion: a controlled study of complication rates. Obstet Gynecol. 2004;103:101–7.
14. Dalton VK, Harris L, Weisman CS, Guire K, Castleman L, Lebovic D. Patient preferences, satisfaction, and resource use in office evacuation of early pregnancy failure. Obstet Gynecol. 2006;108:103–10.

Vaginal Bleeding in Late Trimester

19

Kiran Guleria, Bhanu Priya, and Archana Chaudhary

19.1 Introduction

Vaginal bleeding in late pregnancy or antepartum hemorrhage (APH) poses life-threatening morbidity to mother and compromises fetus either due to uteroplacental insufficiency or preterm birth. Antepartum hemorrhage is defined as bleeding from or in the genital tract after 24 weeks of pregnancy and before birth of baby. It accounts for 3–5% of pregnancy-related complications [1]. Placenta previa, placental abruption, and vas previa are most important causes for vaginal bleeding in late trimester. Optimal management of these complications depends on timely detection and well-planned intervention with the multidisciplinary approach. Table 19.1 enumerates the causes of vaginal bleeding in late trimester, and this chapter will discuss the major three causes of vaginal bleeding.

There are no consistent definitions of the severity of APH. It is recognized that the amount of blood lost is often underestimated and that the amount of blood coming from the introitus may not represent the total blood lost (e.g., in a concealed placental abruption). It is important, therefore, when estimating the blood loss, to assess for signs of clinical shock. The presence of fetal compromise or fetal demise is an important indicator of volume depletion.

K. Guleria (✉) · B. Priya · A. Chaudhary
Department of Obstetrics & Gynecology, University College of medical Sciences & GTB Hospital, Delhi, India

According to RCOG guidelines 2011, the hemorrhage is considered to be:

Minor hemorrhage—blood loss less than 50 mL that has settled.

Major hemorrhage—blood loss of 50–1000 mL, with no signs of clinical shock.

Massive hemorrhage—blood loss greater than 1000 mL and/or signs of clinical shock.

Regardless of the site of bleeding, women presenting with an APH may be broadly divided into two groups:

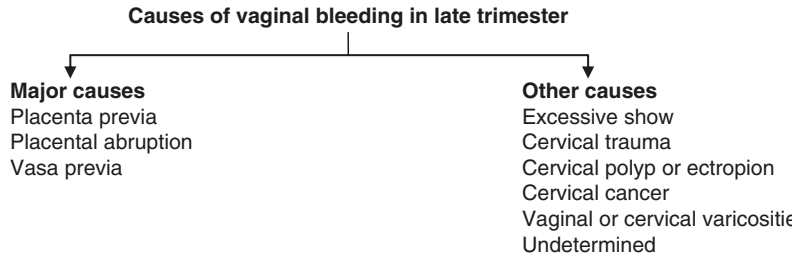
- Those with a major hemorrhage.
- Those with an APH where immediate resuscitative measures are not required.

19.2 Major APH: Emergency Management

- **Observation—**General maternal condition, pulse, BP, respiration, and oxygen saturation.
- **History—**LMP, pregnancy history, recent trauma, amount of blood loss, and pain.
- **Call for help**—Additional staff.
- **Basic life support**—Airway, breathing, and circulation.
- **IV access and fluid replacement**—Via large bore cannula. Crystalloid (up to 2 L of ringer lactate/Hartmann's solution) or colloid (up to 1 L) depending upon the severity of bleeding.
- **Blood and blood products**—RCC to be transfused according to the patient's condition.

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_19

Table 19.1 Causes of vaginal bleeding in late trimester



Four units of FFP and ten units of cryoprecipitate (two packs) can be transfused if coagulopathy is suspected even before blood investigations arrive.

- **Investigations**—CBC, coagulation profile, KFT, electrolytes, Kleihauer-Betke test, ABG in severe cases.
- **Obstetric examination**—Uterine size, fetal presentation, and lie. Assess uterine activity, pain, and tenderness.
- **CTG and USG**—To assess fetal Well-being and placental localization.
- **Speculum examination**—To observe for amount and source of bleeding.
- **Consider delivery**—To improve maternal hemodynamics.
- **Medication**—If time permits corticosteroids for fetal lung maturation, consider $MgSO_4$ for fetal neuroprotection if <30 weeks of gestation and imminent delivery is likely. Anti-D if she is Rh−ve.
- **Documentation.**
- **Communication—**With the woman and her family should be clear and unambiguous.

19.3 APH Where Immediate Resuscitative Measures Are Not Required

- **History**
  - Timing and amount of blood loss.
  - Associated features—e.g., trauma or sexual intercourse.
  - Fetal movements since the bleeding has started.
  - Previous episodes of bleeding in current pregnancy.
  - Review of any USG performed earlier in the pregnancy, particularly for placental site.
  - Past obstetric, gynecological, medical, and surgical history.
- **Examination**
  - General condition—PR, BP, RR, temp, pallor, edema.
  - Obstetric examination—Fundal height, fetal size and presentation, uterine tenderness.
  - Vaginal examination—With speculum only, to assess the site of bleeding.
- **Blood investigations**
  - CBC.
  - Blood group and cross match (at least two units).
  - Coagulation profile.
  - Kleihauer-Betke test.
  - KFT, electrolytes.
- **Fetal well-being assessment**
  - CTG.
  - USG.
- **Ultrasound scan**
  - For placental location.
  - An ultrasound scan is not the investigation of choice to diagnose a placental abruption.
- **Medication**
  - Corticosteroids if <34 weeks.
  - Anti-D if Rh-ve.
  - If birth is imminent at a gestation less than 30 weeks, consider $MgSO_4$ infusion for fetal neuroprotection.
  - Analgesia if required.
- **Documentation and communication**

While it has been a common clinical practice to routinely admit women who have experienced an APH, there is no high level evidence to support this practice.

19.4 Placenta Previa

19.4.1 Definition

Placenta previa is defined as implantation of placenta in lower segment of uterus (LUS) overlying or within 2 cm of internal os [2]. It is classified ultrasonographically as *major* when the placenta either partly or completely covers the internal os and *minor* degree when the leading edge of the placenta is within 2 cm of internal os but not covering it [3].

19.4.2 Incidence and Epidemiology

Placenta previa occurs in approximately 0.5% of pregnancies reaching third trimester. The diagnosis of low-lying placenta is often identified at 16–20 week ultrasound; however, 90% will not have abnormal placentation after 30 weeks of gestation. Therefore, a transvaginal scan (TVS) at 20 weeks can reclassify 26–60% of cases where transabdominal scan (TAS) showed a diagnosis of low-lying placenta.

19.4.3 Risk Factors

- Multiple pregnancy.
- Advanced age > 35 years.
- High parity >6 pregnancies.
- Smoking.
- Deficient endometrium as in scarred uterus due to previous cesarean section or myomectomy or any uterine surgery.
- Manual removal of placenta.
- Endometritis.
- Uterine curettage.

19.4.4 Pathophysiology

The pathophysiology of placenta previa is not fully understood. Preferentially placenta grows in fundal region of the uterus as it has more blood supply than LUS. Abnormal placentation can be due to the above mentioned risk factors or failed placental apparent migration that occurs due to differential growth of LUS.

Table 19.2 Relationship between previous cesarean section (CS) and risk of placenta previa and accreta

No. of CS	Placenta previa incidence (%)	Placenta accrete incidence (%)
0	0.26	3
1	0.65	11
2	1.5	40
3	2.2	61
4	10	67
5	10	67

A previous cesarean section can influence this apparent migration of placenta and chances of persistent placenta previa, and further progression to abnormal invasion (placenta increta and percreta) may occur (Table 19.2) [4].

19.4.5 Clinical Presentation

- Painless vaginal bleeding, recurrent episode.
- Uterine tenderness and irritability is unusual, but sometimes may be there if associated with abruption placentae.
- Fatal malpresentation or unusually high and mobile presenting part.
- Asymptomatic, incidental USG finding.
- Excessive vaginal bleeding in labor.

19.4.6 Complications of Placenta Previa

19.4.6.1 Maternal

- Hemorrhagic shock.
- Preterm labor and prelabor rupture of membranes.
- Increased operative interference, cesarean section.
- Placenta accreta, increta, percreta.
- PPH.
- Amniotic fluid embolism.
- Maternal morbidity and mortality.

19.4.6.2 Fetal

- Prematurity.
- Fetal growth restriction.

- Malpresentation.
- Hypoxia.
- Perinatal morbidity and mortality.

19.4.7 Diagnosis of Placenta Previa

The diagnosis is confirmed by USG localization of placenta. Anterior placental edge is easily visualized with partially full bladder and then empty bladder. Posterior placenta poses problem with shadowing by the presenting part which is overcome by oblique visualization with transducer placement lateral to midline.

19.4.8 Management

19.4.8.1 Principles

- All women should report to their antenatal care provider.
- Clinical assessment for expectant versus urgent intervention to manage maternal and fetal compromise.
- If no maternal compromise full history and examination. No PV examination should be done.
- Corticosteroids for fetal lung maturity between 24 and 34 weeks.
- No role of tocolytics (RCOG 2011).
- A speculum examination should be done 72 h after bleeding has stopped.

19.4.8.2 Expectant Management

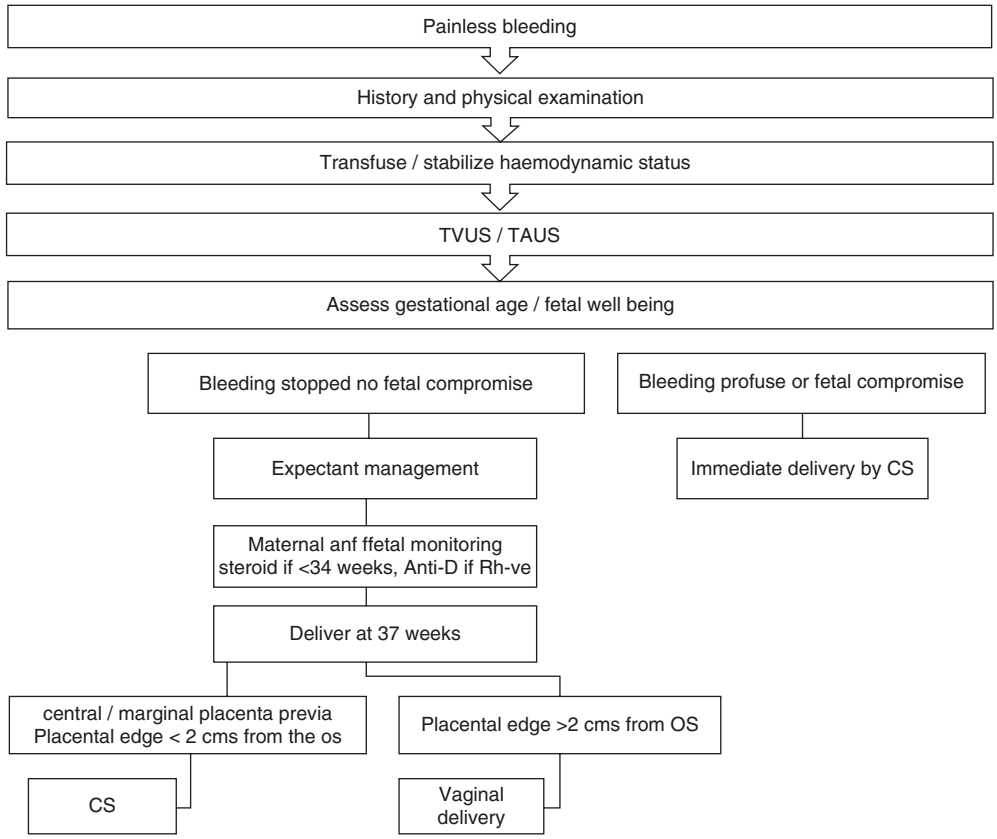
It aims to prolong pregnancy and achieve fetal maturity while minimizing maternal and fetal risks. It targets those patients who had sentinel bleed, and maternal health is not impaired. A policy of expectant management, pioneered by MacAfee, continues to be the standard [5]. The focus on bed rest and restricted physical activity has been shown to reduce preterm birth and perinatal mortality. Corticosteroid coverage is given according to gestational age. Any antenatal bleeding should be treated with full dose of Anti- D in Rh-negative mothers [6, 7]. Hospital admission with bed rest is an option, but carefully selected women with readily available access to intervention can be managed as outpatients especially in minor anterior placenta previa. Blood should be arranged and anemia should be corrected. Elective CS/pregnancy termination should be planned at 37 weeks if patient remains stable.

19.4.8.3 Emergent Management

It aims at maternal resuscitation with initially with fluid and then blood. Continuous electric fetal monitoring should be assessed to detect any sign of fetal compromise. Complete blood count, PT (prothrombin time), INR (international normalized ratio), APTT (activated prothrombin time), and D-Dimer should be sent. At least four units of blood should be cross matched. Ultrasound should be done to confirm placental localization if not done prior. Women with maternal or fetal compromise are required to be delivered immediately by CS. A double setup examination is occasionally appropriate, when the clinician has suspicion of minor placenta previa. The examination should be done in operation theater beginning with the placental edge, with immediate availability of anesthesia and blood to take up women for immediate caesarian section if required. Cesarean section for placenta previa may be associated with the following difficulties and complications:

- Fetal malpresentation may make extraction of fetus difficult.
- Poorly developed and vascular lower uterine segment may lead to an extension of the incision and hemorrhage.
- Difficulty may be encountered in uterine entry in an anterior placenta. There may be a need to cut through the placenta (not preferred) or preferably separate it partially.
- Placenta accreta/percreta may necessitate peripartum hysterectomy.
- Postpartum hemorrhage may occur due to inability of the lower uterine segment to contract efficiently. Hemostatics compression sutures, uterine or internal iliac artery ligation, or hysterectomy may be required.

19.4.9 Summary of the Management of Placenta Previa



19.5 Abruptio Placentae

19.5.1 Definition

Placental abruption denotes premature separation of normally located placenta either completely or partially. The degree of separation of placenta determines the maternal and fetal compromise.

19.5.2 Incidence and Epidemiology

It affects 1–2% of pregnancies. The incidence increases in proportion to the number of previous pregnancy with abruptio placentae.

19.5.3 Risk Factors

- Idiopathic.
- Maternal hypertension.
- Previous h/o abruption.
- Smoking.
- Substance abuse.
- Short umbilical cord.
- Rapid uterine decompression (premature rupture of membrane, delivery of first twin).
- Thrombophilia.
- Blunt trauma.

19.5.4 Pathophysiology

The pathophysiology is multifactorial. With blunt trauma over the abdomen, placental separation and retro-placental hemorrhage can occur. The risk of prematurity, stillbirth, and growth restriction are more common with abruption placentae. Bleeding from abruption may result in external hemorrhage or bloody liquor amnii or may retain as retroplacental clot or both. Bleeding in the

myometrium may cause couvelaire uterus and can result in postpartum hemorrhage. Separation of placenta releases thromboplastic substances and results in consumption of clotting factors and can progress to disseminated intravascular coagulation.

Grades of abruption [8].

Grade I: mild abruption, often diagnosed at time of birth, when retroplacental clot is identified, explaining undiagnosed bleeding.

Grade II: symptomatic women with live fetus.

Grade III: severe abruption with dead fetus

- III A: Grade III abruption without coagulopathy.
- III B: Grade III abruption with coagulopathy.

19.5.5 Clinical Features

- Fifty percent of women are in established labor.
- Diagnosis by clinical presentation is usually possible.
- Vaginal bleeding (mild/heavy).
- Abdominal pain and uterine tenderness are common features.
- Uterus may be overdistended due to concealed hemorrhage.
- Signs of hemorrhagic shock may be there, like hypotension, tachycardia decreased urine output.
- Signs of fetal compromise or IUFD.

19.5.6 Complications of Abruption

Maternal	Fetal
Hypovolemic shock	Fetal hypoxia
Acute renal failure	Prematurity
DIC	FGR
Preterm labor/preterm rupture of membranes	Fetal death
CS/instrumental delivery	
Sheehan syndrome	
Maternal death	
PPH	
Complication of blood transfusion	

19.5.7 Diagnosis

The diagnosis of abruption is mainly clinical. Ultrasound can also be used for diagnosis for abruption but its sensitivity is low (24%) [9]. Thus, it fails to detect three fourth of cases. Laboratory evaluation includes CBC, PT/INR/APTT, D-dimer, fibrinogen levels, and thrombin time.

19.5.8 Management

Expectant management has limited place.

Only in selected cases of very preterm small abruptions in the absence of fetal compromise, it is practiced. The basic principles of expectant management remain same as that of placenta previa.

- Administer maternal corticosteroids.
- Observe for further bleeding.
- Maintain maternal hemoglobin levels.
- Maternal and fetal observations as indicated (intensive electronic fetal monitoring, regular umbilical artery Doppler).
- Monitor for FGR.
- Short-term tocolytics to allow administration of corticosteroids are only recommended till 34 weeks in mild abruption [9].
- Anti-D if Rh negative.

19.5.8.1 Emergent Management

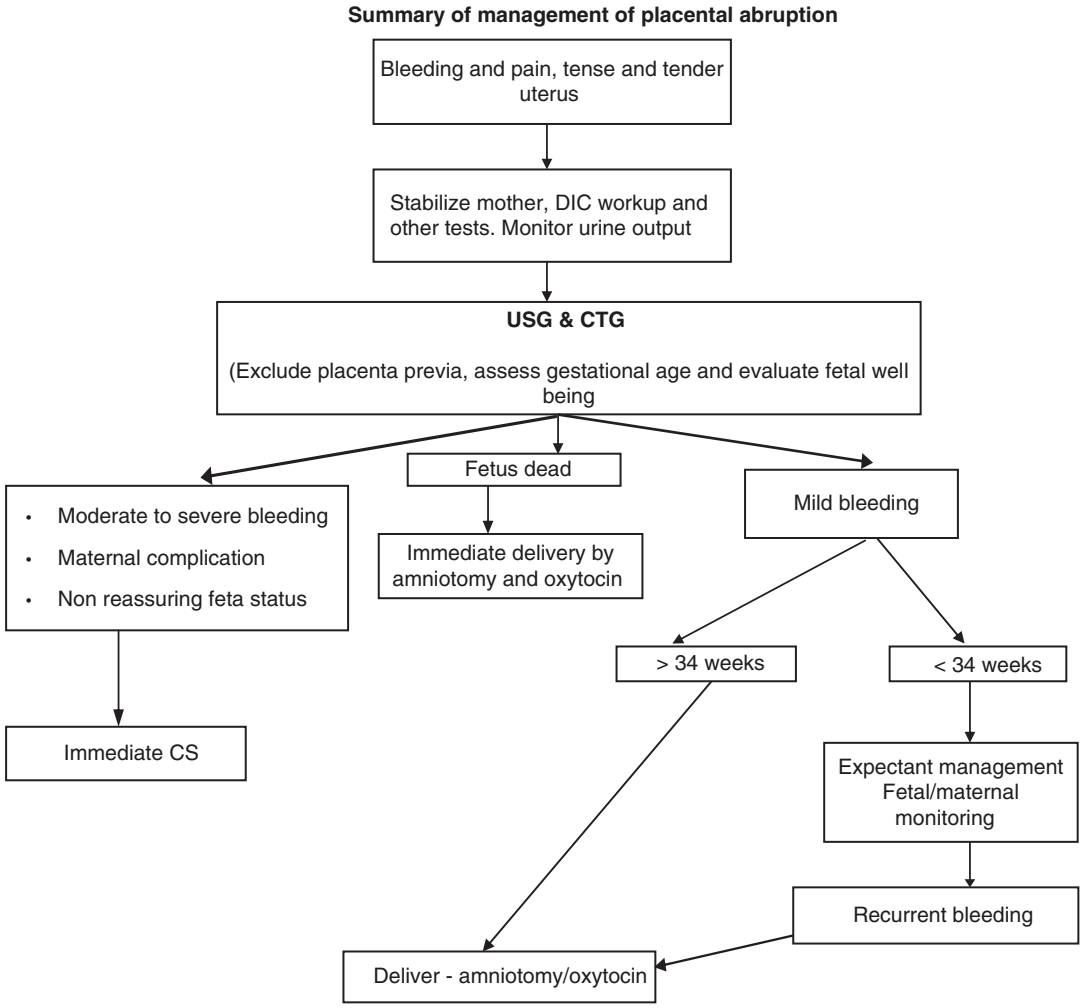
The first step involves the rapid stabilization of maternal cardiopulmonary status and fetal well-being assessment. Adequate fluid and blood resuscitation are warranted. Laboratory investigations are sent. Nonreassuring fetal heart necessitates early CS. When fetal death has occurred secondary to abruption, vaginal delivery should be the goal. In women who have suffered major blood loss or major abruption, the development of DIC should be considered and should be corrected. Up to four units of FFP and ten units of cryoprecipitate may be given while awaiting the results of the coagulation studies [10]. There is a risk of postpartum hemorrhage in women with abruption. Placental

examination should be done for any area of abruption or calcification.

19.5.9 Mode of Delivery in Abruption

- Cesarean section if
  - Abruption is severe and bleeding persistent.
  - Nonreassuring fetal status.
- Vaginal delivery if
  - Fetus is alive, fetal heart rate pattern normal.
  - Fetus is dead and maternal condition is stable.

19.5.10 Summary of Management of Placental Abruption



19.6 Vasa Previa

Vasa previa is the velamentous insertion of the umbilical cord into the membranes in the lower uterine segment resulting in the presence of fetal vessels between the cervix and presenting part.

19.6.1 Incidence and Epidemiology

It is a rare entity with a reported incidence of 1:2000 to 1:6000 [11, 12]. It is of two types.

Vasa previa type 1: Secondary to a velamentous cord insertion in a single or bilobed placenta.

Vasa previa type 2: Arises from fetal vessels running between lobes of a placenta with one or more accessory lobes (RCOG 2011).

19.6.2 Risk Factors

- Bilobed placenta.
- In vitro fertilization.
- Low-lying placenta.
- Multiple pregnancy.
- Succenturiate lobe.
- Velamentous insertion of the cord.

19.6.3 Pathophysiology

Vasa previa is of no major maternal risk but carries a high risk of fetal mortality from exsanguination (33% to 100%), particularly at the time of membrane rupture (fetal blood volume at term is approximately 250 mL) [13]. Even in the absence of bleeding, vessel compression may result in compromise of the fetal circulation [14].

19.6.4 Clinical Features

Vasa previa typically manifests as onset of bleeding at the time of amniotomy or spontaneous rupture of membrane. Rarely, vessels are palpated in the presenting membranes, prohibiting amniotomy.

19.6.5 Diagnosis

Advances in imaging techniques (color Doppler, transvaginal ultrasound) have improved antenatal detection rates. Vasa previa may occur where a low-lying or even placenta previa "migrates" to be out of the lower segment, but some fetal vessels continue to traverse the cervix or lower segment. The diagnosis is confirmed when umbilical arterial waveforms are documented at the same rate as the fetal heart rate.

There are also two quick biochemical tests that can be done to detect fetal blood, but, delivery should not be deferred for confirmation of fetal blood in women with severe hemorrhage or when fetal heart tones are nonreassuring. The blood sample is taken from the vaginal vault to check for fetal blood cells or fetal hemoglobin. The Apt test is most commonly used; it is based on the resistance of fetal hemoglobin to denaturation by alkaline agents and can be performed in the labor and delivery unit. Second is Wright stain in which collected blood is evaluated for presence of nucleated red blood cells. This test can be performed without delay, assuming a normal fetal heart rate.

19.6.6 Management

If the diagnosis of vasa previa is strongly suspected in the presence of fetal compromise in labor, or if hemorrhage is significant, emergency cesarean section (category I) is required (RCOG 2011), early notification of neonatal team, followed by neonatal resuscitation including volume replacement with O negative blood.

A color Doppler ultrasound late in the third trimester should be repeated, in case vasa previa was identified in the second trimester. In cases of confirmed vasa previa in the third trimester, antenatal admission from 28 to 32 weeks in a unit with appropriate neonatal facilities will facilitate quicker intervention in the event of bleeding or labor.

The summary of management of vaginal bleeding in late trimester is depicted in (Fig. 19.1).

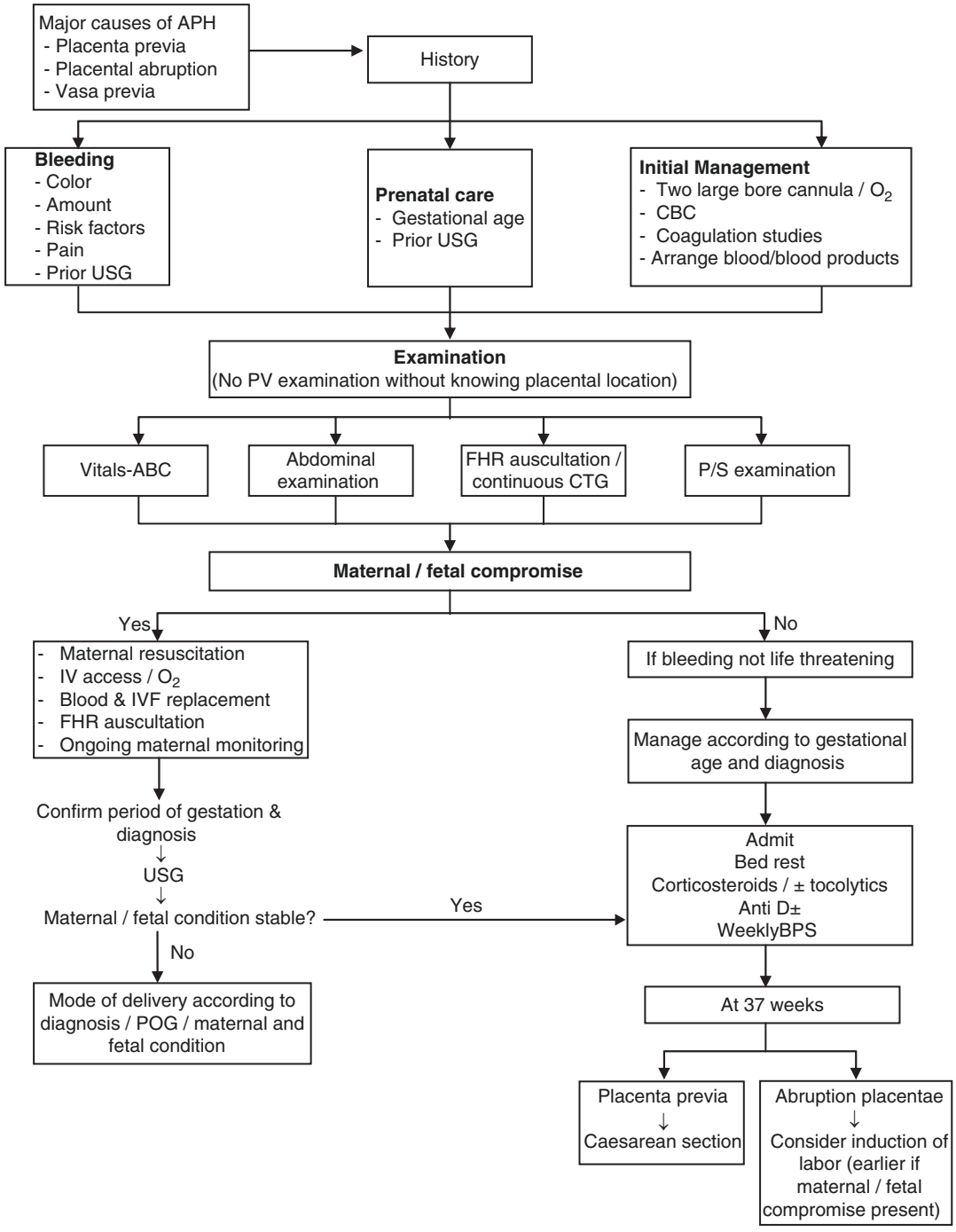


Fig. 19.1 Algorithm of management of vaginal bleeding in late trimester

References

1. Calleja-Agius J, Custo R, Brincat MP, Calleja N. Placental abruption and placenta praevia. Eur Clin Obstet Gynaecol. 2006;2:121–7.
2. Bhide A, Prefumo F, Moore J, Hollis B, Thilaganathan B. Placental edge to internal os distance in the late third trimester and mode of delivery in placenta praevia. BJOG. 2003;110:860–4.
3. Royal college of Obstetricians and Gynaecologists. Green Top Guidelines No 27: Placenta previa, Placenta previa accrete and Vasa previa: Diagnosis and Management. London: RCOG; 2011.
4. Silver RM, Landon MB, Rouse DJ, Leveno KJ, Spong CY, Thom EA, et al. National institute of child health and human development maternal-fetal medicine units network. Maternal morbidity associated with multiple repeat cesarean deliveries. Obstet Gynecol. 2006;107:1226–32.
5. Macafee CHG. Placenta praevia—a study of 174 cases. J Obstet Gynaecol Br Emp. 1945;52:313–24.
6. Royal College of Obstetricians and Gynaecologists. Green top guidelines no 22: Rhesus D Prophylaxis, the use of anti-D Immunoglobulin for. London: RCOG; 2011.
7. Qureshi H, Massey E, Kirwan D, Davies T, Robson S, et al. BCSH guideline for the use of anti-D immunoglobulin for the prevention of haemolytic disease of the fetus and newborn. Transfus Med. 2014;24(1):8–20.
8. Sher G, Statland BE. Abruptio placentae with coagulopathy; a rational basis for management. Clin Obstet Gynecol. 1985;28(1):15–23.
9. Towers CV, Pircon RA, Heppard M. Is tocolysis safe in the management of third-trimester bleeding? Am J Obstet Gynecol. 1999;180(6 pt 1):1572–8.
10. Royal College of Obstetricians and Gynaecologists. Prevention and management of postpartum haemorrhage. In: Green-top Guideline No. 52. London: RCOG; 2009.
11. Oleyese KO, Turner M, Lees C, Campbell S. Vasa previa: an avoidable obstetric tragedy. Obstet Gynecol Surv. 1999;54:138–45.
12. Stafford IP, Neumann DE, Jarrell H. Abnormal placental structure and vasa previa: confirmation of the relationship. J Ultrasound Med. 2004;23:1521–2.
13. Oyalese KO, Turner M, Lees C, Campbell S. Vasa previa: an avoidable obstetric tragedy. Obstet Gynecol Surv. 1999;54:138–45.
14. Zeltzer JS. Vaginal bleeding in late pregnancy. Chapter 254. In: Rakel RE, Bope ET, editors. Conn's current therapy. 60th ed. Philadelphia: Saunders; 2008.

Postterm Pregnancy

20

Meenakshi B. Chauhan and Roopa Malik

20.1 Introduction

Duration of human pregnancy is known to be 280 days. In clinical practice expected date of delivery is forecasted by adding 280 days to date of last menstrual period. But a pregnancy is said to be "term" between 37 weeks and 42 weeks of gestation. There are several changes during this 5-week-long interval which determine the neonatal outcome especially the respiratory morbidity. Many observational studies have shown increased risks to both the fetus and the mother in continuing pregnancy beyond expected date of delivery [1, 2]. A large retrospective study evaluated fetal and neonatal mortality rates in 181,524 late-term and postterm pregnancies and found a significant increase in fetal mortality after 41 weeks of gestation compared with 40 weeks (odds ratio, 1.5, 1.8 and 2.9 at 41 weeks, 42 weeks and 43 weeks of gestation, respectively) [3]. A study by Caughey and Musci found an increase in the rate of meconium and intrauterine death for every week after 37 weeks' gestation and a large increase beyond 41 weeks [4].

The American College of Obstetrician and Gynecologists (ACOG) has now endorsed a recommendation that the label "term" be replaced by designations early term (37-0/7 weeks through 38-6/7 weeks), full term (39-0/7 weeks through 40-6/7 weeks), late-term (41 0/7 weeks-through 41 6/7 weeks) and post term (42-0/7 weeks and beyond) [5]. Moreover, very often in clinical practice, there is imprecision in dating of pregnancy. This difficulty arises when there are mistaken dates or pregnancy occurs in lactational amenorrhoea or soon after withdrawal of oral contraceptive pills or when bleeding occurs in early part of pregnancy. Therefore, it is difficult to decide at what gestation the women should be delivered for good maternal and fetal outcomes.

In clinical practice the terms postterm, postdates, prolonged and postmature are often used interchangeably. *Postmature* term is used for the clinical syndrome in the infant showing features of pathologically prolonged pregnancy. The term *postdates* should not to be used as it is often not easy to date pregnancy in all the women accurately. Postterm or prolonged pregnancy should be the preferred term used to describe pregnancy extended beyond expected dates.

ACOG and the World Health Organization (WHO) have defined postterm pregnancy as that lasts 42 weeks (294 days) or more from the first day of the last menstrual period [6, 7].

20.2 Incidence and Risk Factors

The incidence of postterm pregnancy ranges from 4% to 19% [8]. The incidence varies with the method chosen to estimate period of gestation. Blondel et al. studied the incidence of

M. B. Chauhan (✉) · R. Malik
Department of Obstetrics and Gynaecology,
Pt. B. D. Sharma, PGIMS, Rohtak, Haryana, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_20

postterm pregnancy based on the last menstrual period or sonographic evaluation at 16–18 weeks and found 6.4% incidence when gestation was calculated based on the last menstrual period, while it decreased to 1.9% when sonographic fetal biometry was used as a parameter for estimating gestation [9]. The time of sonographic evaluation also alters the incidence, e.g. Caughey et al. reported 2.7% incidence compared with 3.7% when sonographic assessment was done in first as compared to second trimester of pregnancy [10]. Therefore, using the menstrual dates alone to label an index pregnancy as postterm is often inaccurate. Both last menstrual dates and sonography should be used to label a patient as postterm.

The incidence also varies according to ethnicity of the mother. African American and Asian women are less likely to reach 41–42 weeks of gestation [11]. Primigravidas are at more risk of having a postterm pregnancy as compared to multigravidas. Recurrence of having a postterm pregnancy is also seen which points to genetic inheritance. Similarly, the daughters of women with history of giving birth to postterm babies are at a higher risk of having a postterm pregnancy. Laursen et al. found that maternal, but not paternal, genes influenced prolonged pregnancy [12]. Obesity (BMI > 25 kg/m^2) is also an important risk factor for having a postterm pregnancy [13]. Other risk factors include prior postterm pregnancy and carrying a male fetus. Fetal disorders have also been associated with postterm pregnancies, such as fetal adrenal hypoplasia and anencephaly, since there is impaired hypothalamic-pituatary-adrenal axis in these fetuses leading to decreased corticotrophin releasing hormone (CRH) secretion which in turn delays onset of labour. Rare cause may be placental sulfatase deficiency which is of X-linked inheritance [14].

20.3 Diagnosis

The diagnosis of a postterm pregnancy should be based on both date of last menstrual period and an early sonographic estimation of gestation using crown rump length (CRL). A Cochrane database systemic review has shown that early sonographic evaluation leads to reduction in need for induction of labour in postterm pregnancy [15].

In case pregnancy is the result of in vitro fertilisation (IVF) or intracytoplasmic sperm injection (ICSI), the date of embryo transfer can be used for estimation of gestation. Other less reliable methods include date of positive serum/urinary human chorionic gonadotrophin (hCG), clinical uterine size measurement in early pregnancy, fundal height, perception of quickening or detection of fetal heart sound on Doppler.

20.4 Pathophysiology

Pathogenesis of prolonged pregnancy is not clearly understood. It appears that the mechanism responsible for parturition fails to be triggered. Corticotropin releasing hormone (CRH) produced by placenta has been related to the length of gestation [16]. In women who deliver postterm, the rate of rise of CRH is found to be slower [17].

As the gestation progresses beyond 37 weeks, a number of changes take place which play an important role in outcome of these women.

20.4.1 Placental Changes

With advancing gestation changes take place in placenta as well. Morphological changes that occur with placental senescence can be seen on ultrasound.

- Grade 0—placenta is usually homogeneous in appearance, without echogenic densities, limited by a smooth chorionic plate. It is usually seen in early pregnancy.
- Grade 1—the chorionic plate begins to acquire subtle undulations, and echogenic densities appear to be randomly dispensed throughout the placenta sparing basal layer.
- Grade II—indentations in chorionic plate become more marked, echogenic densities

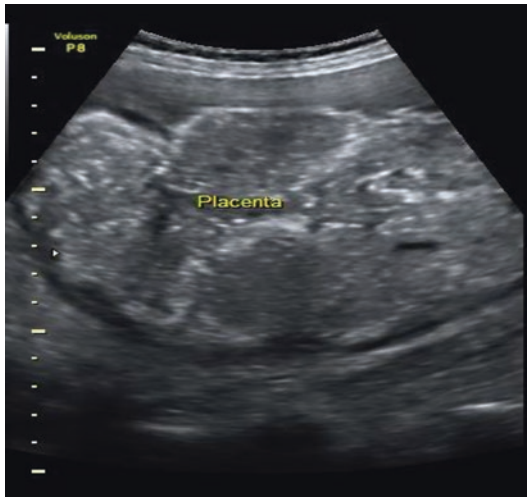


Fig. 20.1 Placenta grade III in sonography in postterm pregnancy

appear in basal layer and comma-like densities seem to extend from chorionic plate into placental substance.

- Grade III—the indentations in chorionic plate become more marked, giving appearance of cotyledons. There is increase in confluence of comma-shaped densities that become intercotyledon septations. Also, the central portion of cotyledons become echo-free (fall out areas), and large irregular densities in the form of acoustic shadows appear (Fig. 20.1).

Although grade 0 and I placentas are not seen in postterm pregnancies, both grade II and II are seen with equal frequency in postterm pregnancies. Therefore, placental grading alone is a poor marker for predicting postterm pregnancy. Grossly, the incidence of placental calcifications and infarction is increased as pregnancy advances beyond 38 weeks.

20.4.2 Amniotic Fluid Changes

The volume of amniotic fluid reaches its peak at around 38 weeks of gestation. Following this there is a gradual decline in amount as well as an increase in the density of amniotic fluid. The decline is at a rate of 125 mL/week till it reaches around 800 mL at 40 weeks. This decline is maximum after 42 weeks of gestation. The decrease in amount is attributed to redistribution of fetal circulation and reduction in renal perfusion. The density increases due to increased vernix caseosa.

Although the amount of liquor can be clinically estimated, it can be quantified using ultrasonography by either amniotic fluid index (AFI) or single deepest vertical pocket (SDP). The AFI is calculated as the sum of largest fluid pockets in four quadrants of uterus excluding any fetal part or umbilical cord. It is expressed in centimetres. An AFI of 8–20 cm is considered to be normal, while AFI <5 cm is considered to be oligohydramnios. SDP is calculated by vertical measurement of largest fluid pocket in uterus. A value of >2 cm is considered to be normal.

Regardless of the method used for diagnosis of oligohydramnios, it is associated with fetal distress and increased neonatal morbidity. There is increased risk of cord compression during labour. At the same time as there is passage of meconium in already reduced amount of amniotic fluid, an increased risk of *meconium aspiration syndrome* prevails due to thick viscous meconium.

20.5 Fetal Complications

20.5.1 Postmaturity Syndrome

Shime et al. reported incidence of postmaturity syndrome in nearly 10% of pregnancies between 41 weeks and 43 weeks of gestation [18]. Postmaturity syndrome is an entity in which the fetus has unique appearance with wrinkled, patchy and peeling skin and thin long extremities classically described as “old man” looks (Fig. 20.2). Wrinkling is maximum in palms and soles. These features are suggestive of wasting and loss of subcutaneous fat. The infant is usually alert with open eyes and appears worried. The nails are long. This picture is not seen in all cases of postmaturity. The likelihood increases with associated oligohydramnios.

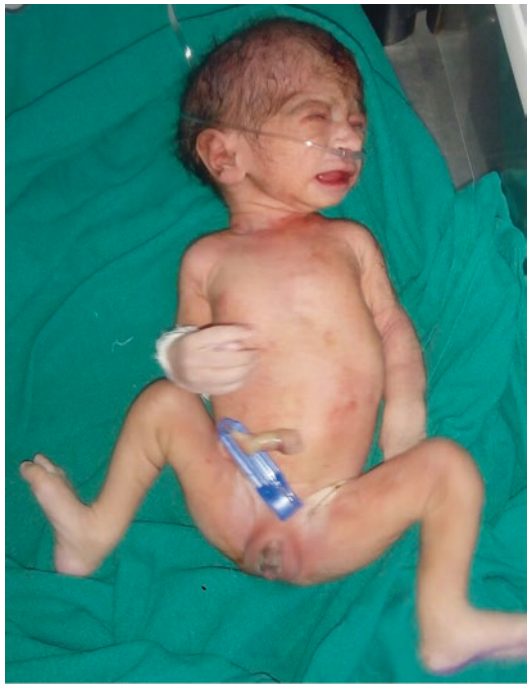


Fig. 20.2 Postmature neonate showing "old man look"

20.5.2 Fetal Macrosomia

Fetal growth continues to occur beyond 37 weeks although at a slower pace. It suggests that although there is placental senescence, placental function is not severely compromised in some of the pregnancies resulting in a macrosomic infant. The chances of birth trauma increase with fetal macrosomia due to higher incidence of operative vaginal deliveries resulting in cephalhematoma, facial nerve injuries and increased risks of shoulder dystocia, leading to birth asphyxia, clavicle fracture or humerus fracture and brachial plexus injuries.

The American College of Obstetricians and Gynecologists (ACOG) (2013b) has recommended that current evidence does not support early induction in a woman at term with suspected fetal macrosomia in order to mitigate both maternal and fetal morbidities. Moreover, the college concluded that in absence of diabetes, vaginal delivery is not contraindicated for women with estimated fetal weight up to 5000 g. Caesarean section should be done if there is prolonged second stage or arrest of descent in babies with estimated weight is >4500 g [19].

20.5.3 Fetal Asphyxia

Postterm pregnancy is often associated with oligohydramnios. This can lead to cord compression. This along with aging of placenta resulting in uteroplacental insufficiency can be responsible for birth asphyxia.

20.5.4 Meconium Aspiration

Due to decreased liquor, meconium becomes viscous leading to meconium aspiration syndrome. Its complications include requirement of assisted ventilation, pneumonia and pulmonary hypertension.

20.6 Maternal Complications

Postterm pregnancy is also associated with significant risk to the mother. There is an increased risk of the following complications:

1. Labour dystocia (9–12% vs. 27% at term) [20].
2. Perineal lacerations due to macrosomia (3.3% vs. 2.6% at term) [21].
3. Operative vaginal delivery [22].
4. Increased caesarean rate and its complications [23].
   A retrospective study including 119,254 low-risk pregnancies demonstrated significant increase in rate of maternal complication beyond 40 weeks' gestation [24].

20.7 Prevention

Accurate gestational age determination decreases the incidence of postterm pregnancy. As described, both date of last menstrual period and early ultrasonography should be used for accurate diagnosis and appropriate management of postterm pregnancy. Several studies have indicated that when ultrasonography is used to confirm menstrual dating, the incidence of postterm pregnancies is reduced, as is the need for obstetric

intervention. For example, in one study, the rates of postterm pregnancies reduced from 9.5% to 1.5% when ultrasonography was used to confirm LMP dating [10].

There are various complementary options that might be useful to stimulate labour, although evidence is limited to support most of these including date fruit [25], castor oil [26], sexual intercourse [27], breast stimulation [28], acupuncture [29] and homoeopathy [30].

In a recent Cochrane review, *membrane sweeping* at *38–40 weeks*, which involves digital separation of membranes from the lower uterine segment through internal cervical os triggering prostaglandin release, has shown to decrease the incidence of pregnancies progressing beyond 41 weeks of gestation [31].

Women with late-term or postterm pregnancies should be counselled that the procedure may not be successful and may be painful. It may be associated with uncomplicated bleeding and irregular uterine contractions without labour. Contraindications of membrane sweeping include placenta previa and other obstetric contraindications.

20.8 Management

Antepartum care includes accurate diagnosis, fetal surveillance and option of induction of labour or expectant management.

20.8.1 Fetal Surveillance

There is insufficient evidence to recommend management strategy between 40 and 42 completed weeks. Thus not considered mandatory, initiation of fetal surveillance at 41 weeks is a reasonable option. There are several options for fetal surveillance, including non-stress test (NST), biophysical profile (BPP) and modified BBP (NST and amniotic fluid assessment). There is not enough data to define the optimal type and frequency of testing. Though a small number of studies demonstrate twice-weekly surveillance to be superior to once a week surveillance, precise recommendations cannot be established [32]. A Cochrane review of five randomised and quasi-randomised trials of fetal surveillance in 2974 high-risk pregnancies that included postterm pregnancies found no difference in perinatal death between BPP and NST groups (RR, 1.35; 95% CI, 0.6–2.98) [33].

Although limited by retrospective design, current evidence suggests that an ultrasonographic assessment of amniotic fluid volume to detect oligohydramnios is warranted. Available data from RCTs support the use of single deepest vertical pocket (SDP) of amniotic fluid in place of amniotic fluid index (AFI) to diagnose oligohydramnios as it was found to be associated with reduction in unnecessary interventions without an increase in adverse perinatal outcomes [34].

When oligohydramnios was observed in postterm pregnancies, there were statistically significant increased rates of meconium-stained amniotic fluid, fetal growth restriction, fetal heart abnormalities, caesarean delivery and fetal demise [35, 36]. If oligohydramnios is detected at 41 0/7 weeks of gestation or beyond, delivery is indicated.

Diminution of fetal movements appears to be associated with increased risk of perinatal morbidity. Though there is limited data to show that maintaining the fetal kick chart yields any benefit nonetheless, professional consensus is that women should report their obstetrician when they perceive reduced fetal movements.

20.8.2 Induction of Labour

ACOG recommends that induction of labour can be considered for women between 41 0/7 weeks and 42 0/7 weeks of gestation. Given the evidence of increase in perinatal morbidity and mortality, induction of labour should be done between 42 0/7 and 42 6/7 weeks of gestation [37].

On the other hand RCOG recommends offering induction of labour between 41 and 42 weeks of gestation and increased surveillance from 42 weeks in women who decline induction of labour [38].

Once a decision of induction has been made, the obstetrician needs to assess the Bishop score. It is based on dilatation, consistency, position, effacement of cervix and station of head. Success

of induction depends on condition of cervix and station of head. If the score is unfavourable, then methods of cervical ripening are used to increase the chances of vaginal delivery. Ripening can be done by membrane stripping, Foley's catheter, oxytocin, mifepristone, nitric oxide donors and most effectively by prostaglandins.

20.8.3 Intrapartum Care

Women with postterm pregnancy are at high risk of fetal asphyxia during labour; therefore they should immediately report to hospital on commencement of labour and should be carefully monitored. Electronic monitoring is recommended during labour.

Decision for amniotomy is particularly tricky as further reduction in amniotic fluid volume can enhance chances of cord compression, but it helps identification of thick meconium which may be dangerous to the fetus. Also, after amniotomy, intrauterine fetal monitoring can be commenced for more precise monitoring.

Identification of thick meconium at amniotomy is particularly worrisome for the fear of meconium aspiration syndrome. In a large RCT, amnioinfusion had not shown to decrease risk of meconium aspiration syndrome or perinatal death [39]. According to ACOG 2012, amnioinfusion did not reduce meconium aspiration syndrome; however, it remains a reasonable treatment approach for repetitive variable decelerations [40].

A caesarean delivery should be done for a woman in early labour with thick viscid meconium stained amniotic fluid. Early oropharyngeal suctioning at the delivery of head is no longer recommended [41].

20.9 Management of Newborn

If the amniotic fluid is meconium stained, pharyngeal aspiration before delivery of shoulders is not recommended. The staff managing the postterm neonate must be skilled in intubation and endotracheal aspiration though routine endotracheal intubation of a vigorous neonate is not recommended. A protocol should be established for transfer of neonate to the neonatal intensive care unit (NICU).

20.10 Role of Vaginal Birth After Caesarean Delivery in Management of Postterm Pregnancy

A successful vaginal birth after caesarean delivery is associated with lesser maternal as well as fetal morbidity. Therefore, trial of labour after caesarean delivery (TOLAC) remains a reasonable option for women with uncomplicated postterm pregnancies with previous caesarean section. However, as with pregnancies without previous caesarean section, the TOLAC failure rate increases with advancing gestational age, from 22.2% before 40 weeks of gestation to 35.4% after 41 weeks of gestation [42].

Labour induction further increases the risk of uterine rupture as opposed to spontaneous labour in these women. Thus, TOLAC remains an option for women with postterm pregnancy with previous caesarean delivery, but they should be counselled regarding their individualised risks such as failure of TOLAC and scar rupture.

20.11 Asian Perspective

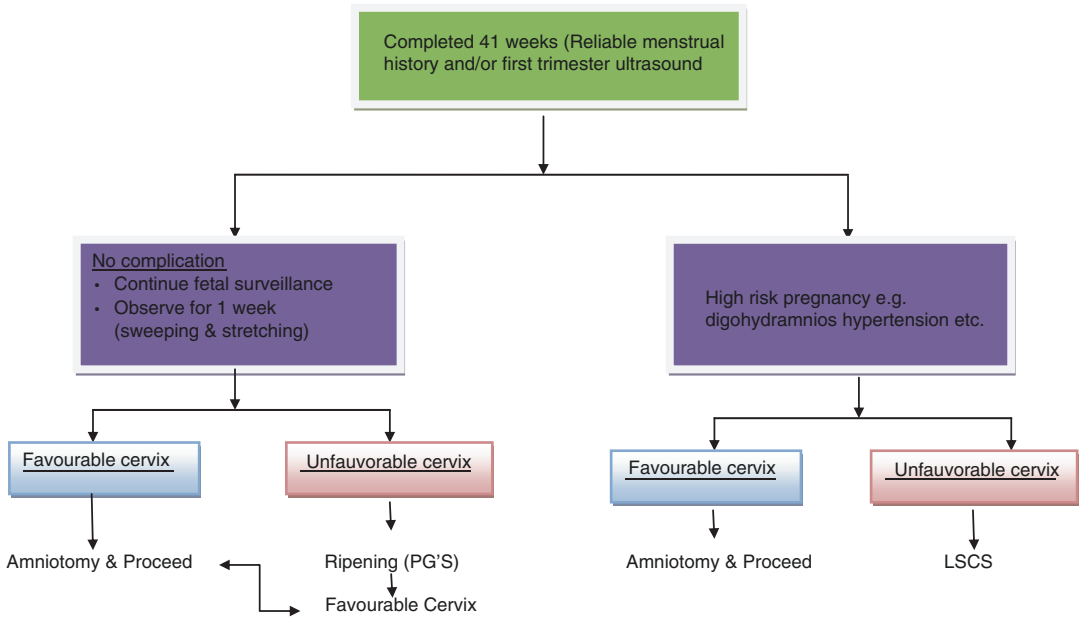
Studies have shown Asian and African American women have a shorter duration of gestation [11]. Mathai et al. found that the mean gestation at delivery following spontaneous labour was 39 weeks in Indian women [43]. An Indian study demonstrated higher rate of meconium-stained amniotic fluid and meconium aspiration beyond 41 weeks of gestation than those who delivered between 40 and 41 weeks of gestation [44]. In another Indian study on 2010 among women whose pregnancies were carried beyond 40 weeks, intrapartum complications were meconium-stained liquor in 18.6%, adverse fetal heart rate patterns in 8.4% and shoulder dystocia in

2%. The study found that induction of labour reduced perinatal mortality and reduced caesarean section for fetal distress [45].

Although there is paucity of large studies to make any recommendations, obstetricians should be aware of this racial discrepancy before any decision-making.

20.12 Summary

1. Postterm pregnancy is associated with increased fetal, neonatal and maternal risks.
2. Accurate determination of gestational age should be carried out using the last menstrual date and first trimester ultrasound.
3. Induction of labour should be considered between 41 and 42 weeks of gestation and definitely recommended beyond 42 weeks of gestation considering increase in perinatal morbidity and mortality after 41 weeks.
4. Antepartum surveillance should be done if decision of conservative management is taken between 41 and 42 weeks of gestation and beyond.



Flow chart for management of postterm pregnancy

References

1. Mac Dorman MF, Kirmeyer S. Fetal and perinatal mortality, United States, 2005. Natl Vital Stat Rep. 2009;57(8):1.
2. Smith GC. Estimating risks of perinatal death. Am J Obstet Gynecol. 2005;192:17–22.
3. Divon MY, Haglund B, Nisell H, Otterblad PO, Westgren M. Fetal and neonatal mortality in postetrm pregnancy: the impact of gestational age and fetal growth restriction. Am J Obstet Gynecol. 1998;178:726–31.
4. Caughey AB, Musci TJ. Complications of term pregnancies beyond 37 weeks of gestation. Obstet Gynaecol. 2004;103:57–62.
5. American College of Obstetricians and Gynecologists. Definition of term pregnancy. Committee Opinion No. 579. Nov 2013.
6. American College of Obstetricians and Gynaecologists Practice Bulletin: Management of postterm pregnancy, September 2004.
7. World Health Organization (WHO): recommended definition terminology and format for statistical tables related to perinatal period and rise of a new certification for cause of perinatal deaths. Modifications recommended by FIGO as amended, October 14, 1976. Acta Obstet Gynaecol Scand. 1977;56:347.
8. Divon MY, Feldman-Leidner N. Postdates and antenatal testing. Semin Perinatol. 2008;32(4):295.
9. Blondel B, Morin I, Platt RW, et al. Alogrithms for combining menstural and ultrasound estimates of

gestational age: consequences for rates of preterm and postterm birth. Br J Obstet Gynaecol. 2002;109:718.
10. Caughey AB, Nicholson JM, Washington AE. First – vs second-trimester ultrasound: effect on pregnancy dating and perinatal outcomes. Am J Obstet Gynecol. 2008;198(6):703.el.
11. Caughey AB, Stotland NE, Washington AE, et al. Who is at risk for prolonged and postterm pregnancy? Am J Obstet Gynecol. 2009;200:683.
12. Laursen M, Bille C, Olesen AW, et al. Genetic influence on prolonged gestation a population-based Danish twin study. Am J Obstet Gynecol. 2004;190:489.
13. Halloran DR, Cheng YW, Wall TC, et al. Effect of maternal weight on postterm delivery. J Perinatol. 2012;32:85–90.
14. Mac Donald PC, Siiteri PK. Origin of estrogen in women pregnant with an anencephalic fetus. J Clin Invest. 1965;44:465.
15. Whiworth M, Bricker L, Neilson JP, et al. Ultrasound for fetal assessment in early pregnancy. Cochrane Database Syst Rev. 2010;4:CD007058. https://doi.org/10.1002/14651858.CD007058.pub2. http://onlinelibrary.wiley.com/doi/10.1002/14651858.CD007058.pub2/full
16. McLean M, Bisits A, Davies J, Woods R, Lowry P, Smith R. A placental clock controlling the length of human pregnancy. Nat Med. 1995;1:460–3.
17. Ellis MJ, Livesey JH, Inder WJ, Inder WJ, Prickett TC, Reid R. Plasma corticotrophin-releasing hormone and unconjugated estriol in human pregnancy: gestational patterns and ability to predict preterm delivery. Am J Obstet Gynecol. 2002;186:94–9.
18. Shime J, Gare DJ, Andrews J, et al. Prolonged pregnancy: surveillance of the fetus and the neonate and the course of labor and delivery. Am J Obstet Gynecol. 1984;148:547.
19. American College of Obstetricians and Gynaecologists: Fetal macrosomia. Practice Bulletin No. 22, November 2000, Reaffirmed 2013b.
20. Rand L, Robinson JN, Economy KE, Norwitz ER. Post-term induction of labor revisited. Obstet Gynecol. 2000;96:779–83.
21. Campbell MK, Ostbye T, Irgens LM. Post-term birth: risk factors and outcomes in a 10-year cohort of Norwegian births. Obstet Gynecol. 1997;89:543–8.
22. Alexander JM, McIntire DD, Leveno KJ. Forty weeks and beyond: pregnancy outcomes by week of gestation. Obstet Gynecol. 2000;96:291–4.
23. Treger M, Hallak M, Silberstein T, et al. Post-term pregnancy: should induction of labor be considered before 42 weeks? J Matern Fetal Neonatal Med. 2002;11:50–3.
24. Caughey AB, Stotland NE, Washington AE, et al. Maternal obstetric complications of pregnancy are associated with increasing gestational age at term. Am J Obstet Gynecol. 2007;196:155.e1–6.
25. Al-Kuran O, Al-Mehaisen L, Bawadi H, et al. The effect of late pregnancy consumption of date fruit on labour and delivery. J Obstet Gynaecol. 2011;31(1):29–31.
26. Kelly AJ, Kavanagh J, Thomas J. Castor oil, bath and/or enema for cervical priming and induction of labour. Cochrane Database Syst Rev. 2013;(7):CD003099.
27. Kavanagh J, Kelly AJ, Thomas J. Sexual intercourse for cervical ripening and induction of labour. Cochrane Database Syst Rev. 2001;(3):CD 003093.
28. Kavanagh J, Kelly AJ, Thomas J. Breast stimulation for cervical ripening and induction of labour. Cochrane Database Syst Rev. 2005;(3):CD 003392.
29. Smith CA, Crowther CA, Grant SJ. Acupuncture for induction of labour. Cochrane Database Syst Rev. 2013;(8):CD002962.
30. Smith CA. Homeopathy for induction of labour. Cochrane Database Syst Rev. 2008;(4):CD003399.
31. Boulvain M, Stan CM, Irion O. Membrane sweeping for induction of labour. Cochrane Database Syst Rev. 2005;(1):CD000451. https://doi.org/10.1002/14651858.CD000451PUB2.
32. Boehm FH, Salyer S, Shah DM, Vaughn WK. Improved outcome of twice weekly nonstress testing. Obstet Gynecol. 1986;67:566–8.
33. Lalor JG, Fawole B, Alfirevic SA, Devane D. Biophysical profile for fetal assessment in high risk pregnancies. Cochrane Database Syst Rev. 2008;(1):CD000038. https://doi.org/10.1002/14651858.CD000038.pub2.
34. Nabhan AF, Abdelmoula YA. Amniotic fluid index versus single deepest vertical pocket as a screening test for preventing adverse pregnancy outcome. Cochrane Database Syst Rev. 2008;(3):CD006593. https://doi.org/10.1002/14651858.CD006593.pub2.
35. Chamberlain PF, Manning FA, Morrison I, Harman CR, Lange IR. Ultrasound evaluation of amniotic fluid volume. I. The relationship of marginal and decreased amniotic fluid volumes to perinatal outcome. Am J Obstet Gynecol. 1984;150:245–9.
36. Bochner CJ, Medearis AL, Davis J, Oakes GK, Hobel CJ, Wade ME. Antepartum predictors of fetal distress in post-term pregnancy. Am J Obstet Gynecol. 1987;157:353–8.
37. American College of Obstetricians and Gynecologists. Management of Late-Term and Postterm pregnancies. Practice Bulletin No 146, August 2014.
38. National Collaborating Centre for Women's and Children's Health/National Institute for Health and Clinical Excellence (NCCWCH/NICE). Induction of labour. London (UK): National Institute for Health and Clinical Excellence (NICE); 2008. p. 32. (Clinical guideline; no. 70).
39. Fraser WD, Hofmeyr J, Lede R, et al. Amnioinfusion for the prevention of the meconium aspiration syndrome. N Engl J Med. 2005;353:909.
40. American College of Obstetricians and Gynecologists. Amnioinfusion does not prevent meconium aspiration

syndrome. Committee Opinion No 346,October 2006. Reaffirmed 2012.
41. American College of Obstetricians and Gynecologists. Management of delivery of a newborn with meconium-stained amniotic fluid. Committee Opinion No. 379, September 2007, Reaffirmed 2013c.
42. Coassolo KM, Stamilio DM, Pare E, Peipert JF, Stevens E, Nelson DB, et al. Safety and efficacy of vaginal birth after cesarean attempts at or beyond 40 weeks of gestation. Obstet Gynecol. 2005;106:700–6.
43. Mathai M, Thomas S, Peedicayil A, et al. Growth pattern of the Indian fetus. Int J Gynaecol Obstet. 1995;48:21–4.
44. Asseja V, Sangwan K, Puri M. Should induction of labour be considered earlier than 42 weeks in post dated pregnancy? Int J Gynae Plastic Surg. 2012;4:22–6.
45. Chhabra S, Dargan R, Nasare M. Induction of labour in postdated pregnant women. J Coll Physicians Surg Pak. 2012;22:644–7.

Part III

Labour

Diagnosis of Labour

21

Fawzia Hossain

21.1 Introduction

In this chapter, we shall deal with the mechanism of normal labour, how to diagnose labour and the influence the power (uterine activity), passage (maternal pelvis) and passenger (fetus) have on this process. What are the signs of true labour? What do the guidelines say regarding false labour pains, causes of onset of labour, normal physiology, stages of labour, mechanism of labour clinical course of stages of labour and its management?

We shall build on this knowledge and discuss abnormal patterns of labour, the causes and management options for the treatment of dysfunctional labour.

21.2 Normal Labour

Normal labour is characterised by the onset of regular contractions associated with cervical effacement and dilatation with progressive descent of the presenting part.

A baby presenting by the vertex will generally enter the pelvis with the occiput in the lateral position owing to the fact that the transverse diameter is the largest pelvic inlet diameter. As the presenting part descends through the pelvis, it rotates so that the occiput moves into the anterior position. The fetal spine is connected to the back of the skull, and therefore pressure directed along the spine causes flexion of the fetal head as descent and rotation occur. On passing through the outlet, delivery of the head is achieved by extension. Restitution occurs when the head realigns with the shoulders. External rotation occurs as the shoulders rotate from the transverse position at the inlet to the A/P position at the outlet.

Normal labour begins spontaneously at 37–42 weeks, progresses at an acceptable rate and results in the spontaneous vaginal delivery of a live undistressed neonate in the occipitoanterior position [1–3] (Fig. 21.1).

21.3 Dysfunctional Labour

Dysfunctional labour refers to abnormal labour patterns. The first stage of labour is considered abnormal when cervical dilatation rate is less than 1 cm per hour during active labour. The second stage is considered dysfunctional if there is failure to deliver the fetus within 1 h of commencement of active pushing [1–3].

Dysfunctional labour may be due to problems with the fetus (passenger), problems with maternal or soft tissues (passage) or disorders of the uterine activity (power). Each of these will be addressed later in this chapter.

F. Hossain (✉)
Department of Obstetrics and Gynaecology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh

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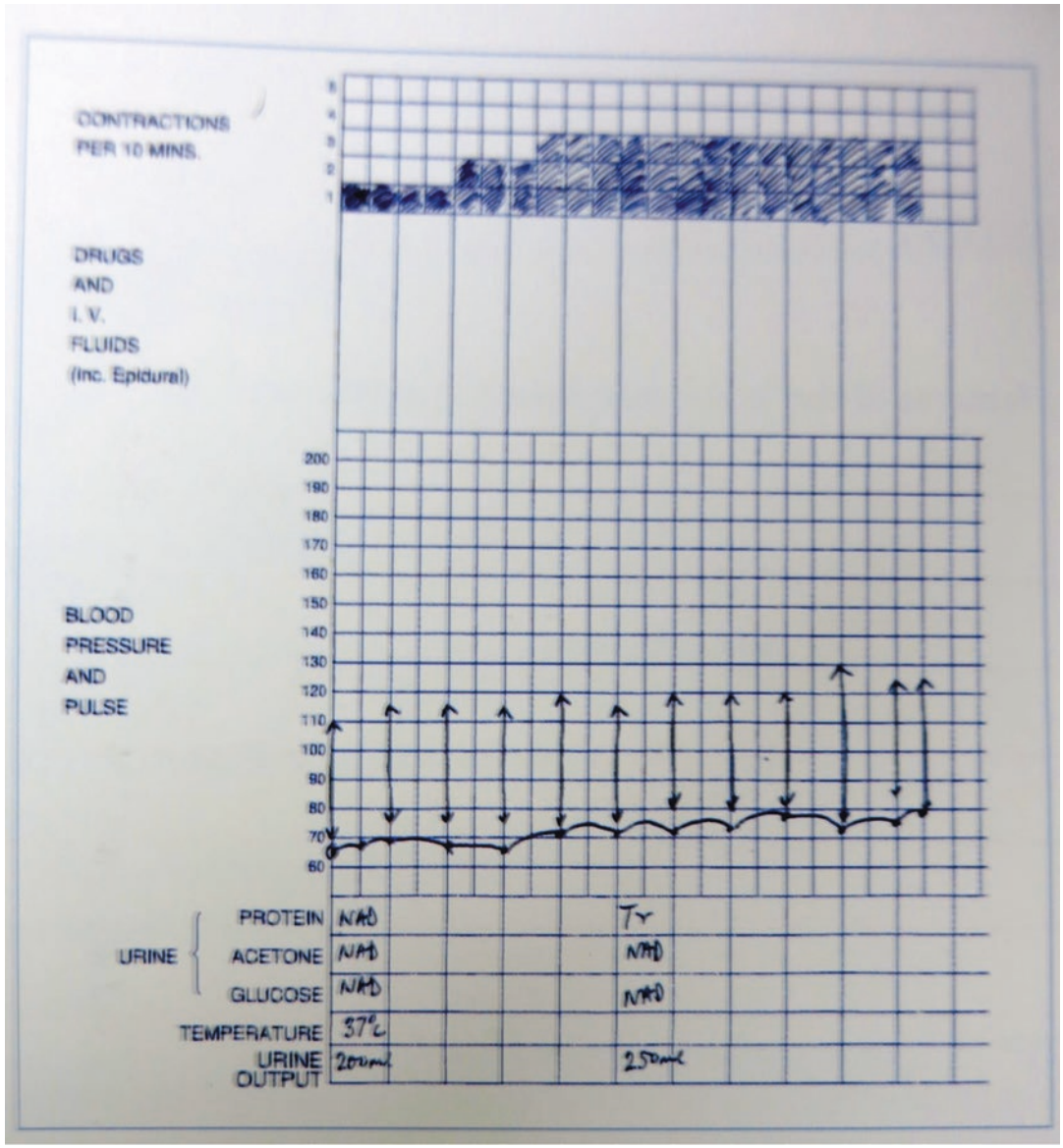


Fig. 21.1 The normal partogram

Labour wards may not be the appropriate environment for women in the latent phase.Women value home assessment in the latent phase, and this can reduce the number of visits to hospitals [4].

The duration of the latent phase is particularly difficult to measure, as women experience the onset of labour in a variety of different ways [5].

A long latent phase can often be a discouraging and exhausting experience, and women need good consistent psychological support.

The following definitions of stages of labour are recommended [6]:

Latent phase

- It is a period of time, when there are painful contractions, with cervical effacement and dilatation up to 4 cm or less.

Active phase

- There are regular painful contractions, and there is progressive cervical dilatation from 4 cm [7].

21.3.1 First Stage

Latent phase
- Cervix less than 4 cm dilated.

Active phase
- Cervix between 4 cm and 10 cm dilated.
- Rate of cervical dilatation at least 1 cm/h.
- Effacement is usually complete.
- Fetal descent through birth canal begins.

21.3.2 Second Stage

- Early phase (non-expulsive).
- Cervix fully dilated (10 cm).
- Fetal descent continues.
- No urge to push.

Late phase (Expulsive)
- Fetal presenting part usually reaches the pelvic floor, and the woman has the urge to push.
- It typically lasts <1 h in primigravida and <30 min in multigravida.
- Carry out vaginal examinations at least once every 4 h in the first stage of labour, and plot the findings on the partograph.
- The partograph is very helpful in monitoring the progress of labour and in the early detection of abnormal labour patterns [8].

21.3.3 Third Stage

This is from delivery of the baby to delivery of the placenta. The uterus contracts shearing the placenta from the uterine wall. This separation is often indicated by a small spillage of dark blood and a lengthening of the cord. The placenta can then be delivered by controlled cord traction.

21.4 Common Definitions [1–3]

Lie—this refers to the relationship between the longitudinal axis of the uterus and the longitudinal axis of the fetus. This is generally longitudinal but may be transverse or oblique.

Presenting part—this is the portion of the fetus felt on vaginal examination.

Position—this is the relationship between a defined area of the presenting part (known as the dominator) and the mother's pelvis.

Station—this refers to the level of the presenting part in relation to the ischial spines.

Attitude—this refers to the relationship of the fetal head and limbs to the fetal trunk. The attitude is generally one of flexion.

Vertex—this is the area bounded by the anterior fontanelle (bregma), posterior fontanelle and the biparietal eminences.

Occiput—this is the area below the posterior fontanelle.

Sinciput—this is the area in front of the anterior fontanelle. This is divided into the brow (area between the anterior fontanelle and the root of the nose) and the face (area below the root of the nose).

21.5 Anatomical and Physiological Considerations of the Pelvis and Fetus

21.5.1 The Passenger

The cranium is made up of five bones: two parietal bones, two frontal bones and the occiput. Suture lines separate the skull bones. The sagittal suture separates the two parietal bones, and the frontal suture separates the two frontal bones, while the lambdoid suture separates the occipital bone and the parietal bones (Figs. 21.2 and 21.3).

The anterior fontanelle is formed by the junction of the sagittal, frontal and two coronal sutures and is diamond-shaped. The posterior fontanelle is formed by the junction of the sagittal and two lambdoid sutures and is Y-shaped.

The fetal skull has been divided into areas to facilitate the description of the presenting part.

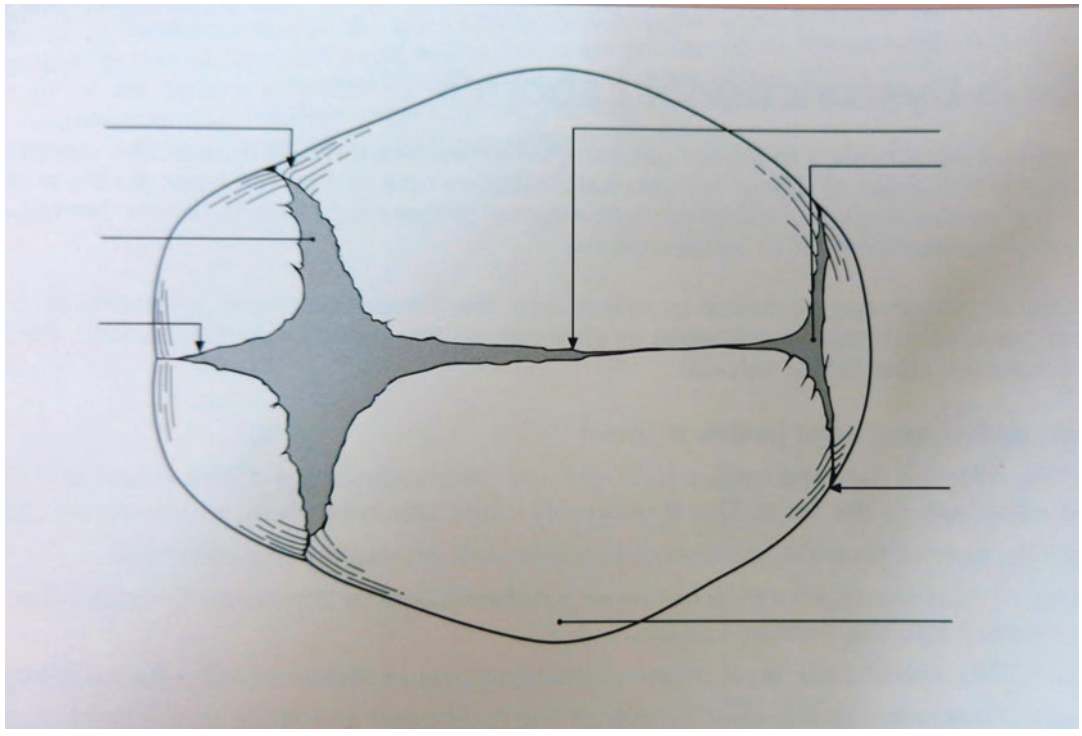


Fig. 21.2 The suture lines and fontanelle of the cranial bones

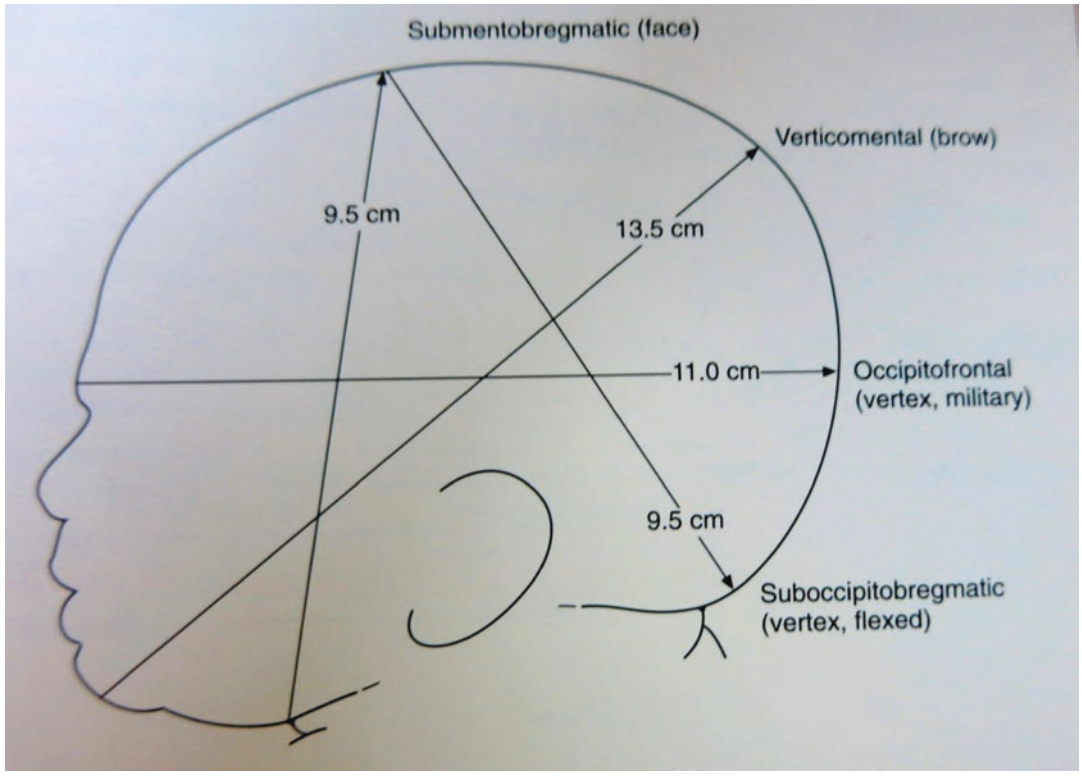


Fig. 21.3 The four sagittal diameters

21.6 What Factors Influence the Diameter of the Presenting Part?

- Presentation and position.
- Malformations.
- Overall fetal size.

The portion of the fetal skull that presents in labour is dependent on the degree of flexion of the fetal head.

During the course of normal labour, the vertex presents, and due to the insertion of the spine posteriorly, flexion of the fetal head results. The widest transverse diameter in this position is the biparietal diameter (9.5 cm). The sagittal diameter tends to be the suboccipitobregmatic diameter (9.5 cm, below the occiput to the centre of the anterior fontanelle).

If the head fails to flex, the resulting diameter is the occipitofrontal (11–12 cm, occiput to the root of the nose).

Further head extension results in the brow presentation and the mentovertical diameter (14 cm, chin to the centre of the sagittal suture).

Continued extension results in a face presentation and submentobregmatic diameter (9.5 cm, angle between the neck, chin and the centre of the anterior fontanelles).

Malformations may also influence the size of the presenting part. Examples of this will include anomalies such as hydrocephalus or large space occupying lesions such as teratomas.

21.6.1 The Passages

The female pelvis consists of a pair of innominate bones (pubis, ischium and ilium) joined anteriorly by the pubic symphysis while articulating with the sacrum and coccyx posteriorly.

The brim of the pelvis is comprised of the upper border of the pubic symphysis, iliopectineal line, ala of the sacrum and sacral promontory. The brim of the normal gynaecoid pelvis is almost round although the sacrum intrudes posteriorly. The transverse diameter of the brim generally measures 13.5 cm and the AP diameter 11 cm.

The midpelvis is almost circular in outline and is bordered by the apex of the pubic symphysis, the ischial spines, the sacrospinous ligament and the tip of the sacrum. Contained within the midcavity is the plane of least dimension which measures 10.5 cm in its transverse plane at the level of the ischial spines. It is at this level that arrest of labour commonly occurs.

The outlet is diamond shaped and is bounded by the subpubic arch, ischial tuberosities, sacrotuberous ligaments and the coccyx.

It is said that the plane and the brim makes an angle of 55° with the horizontal in the erect position [1–3] (Figs. 21.4 and 21.5).

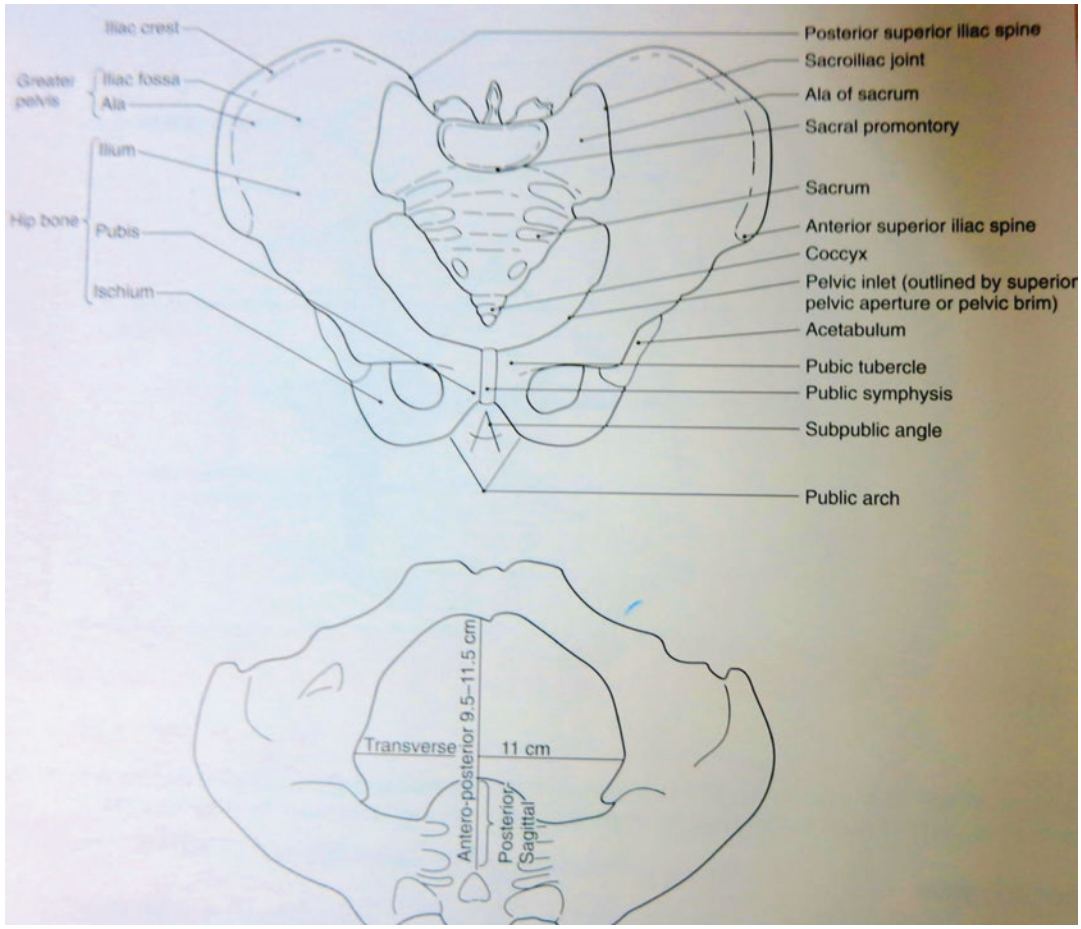
21.6.2 False Labour Pain

Irregular and unpredictable contractions may come at intervals between 10 min, 8 min, 6 min and even 2 min.

- There is no progress of labour.
- Contractions are felt as non-specific abdominal tightening.
- Change in activity or posture causes contractions to slow down or stop.
- There is no evidence of show.
- Membranes remain unruptured.

True labour develops into a regular pattern, with contractions growing closer in a regular pattern. With false labour, contractions remain irregular. True labour contractions last more than 30 s at the onset and progress up to 60 s, whereas false labour contractions vary in length and intensity. The contractions in true labour continue regardless of the activity or change in posture of the mother and can grow even stronger. False labour contractions can often cease spontaneously irrespective of the mother's activity.

With true labour, the pain tends to begin high in the abdomen, radiating throughout the entire abdomen and lower back or vice versa. In the case of false labour, the contractions are often concentrated in the lower abdomen and groin.



Type	Inlet	Midcavity	Outlet
Android male-like	Heart-shaped	Intraspinous diameter	Pubic arch narrow reduced
Anthropoid ape-like	Ovoid AP > transverse	Adequate	Adequate
Gynaecoid			
Normal female	Oval transverse > AP	Adequate	Pubic arch > 90 diameter degrees
Platypelloid	Transverse > AP	Wide intraspinous	Wide pubic arch diameter

Fig. 21.4 The female pelvis

21.7 How Can Dysfunctional Labour Be Diagnosed?

Dysfunctional labour can be diagnosed by careful and repeated assessment of the power, passage and the passenger. The partogram is a means of graphically displaying this intrapartum information in a clear and focused way and facilitates effective transfer of information to other caregivers. The partogram has been in use for over 20 years [9]. Use of the partogram has been shown to be associated with a reduction in prolonged labour, reduction in the augmentation of labour and reduction in sepsis [8].

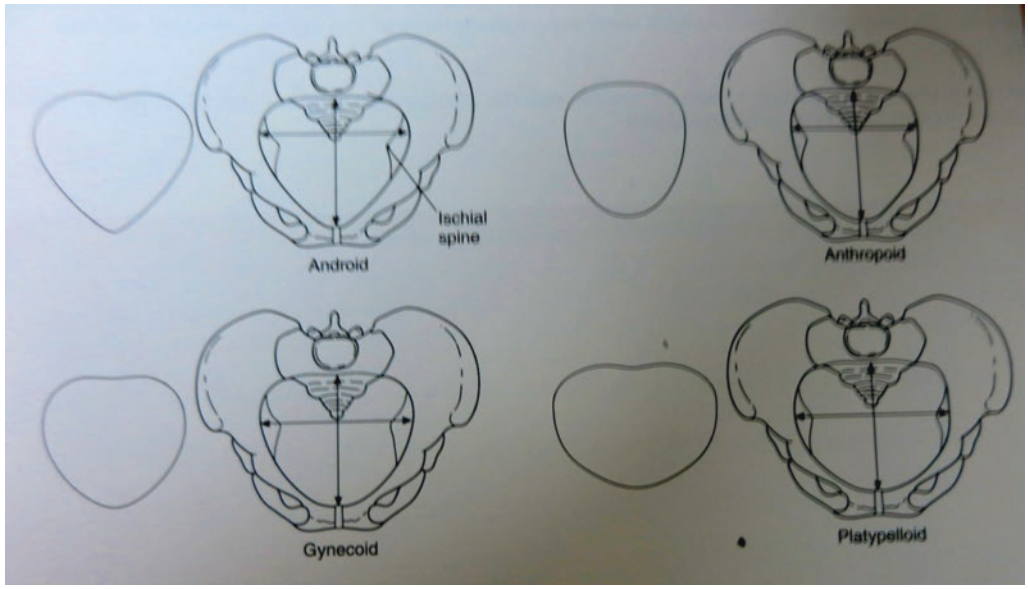


Fig. 21.5 Four pelvis shapes

21.8 What Information Is Contained on the Partogram? [10]

21.8.1 Details of the Power

Frequency of contractions. The number of contractions occurring over a 10 min period is recorded and plotted.

Duration of contractions. Effective uterine activity is generally sustained for a period greater than 40 s.

Amplitude. This is an assessment of the perceived strength of contractions. It must be stressed that this is a very subjective assessment. The intensity of uterine contractions can only be accurately defined by the placement of an intrauterine pressure catheter. The value of formal intrauterine pressure monitoring is limited.

21.8.2 Details of the Passenger

Fetal heart rate recording. Listening to the fetal heart following a contraction every 15 min for a period of 1 min during the first stage. The fetal heart rate should be recorded after each expulsive contraction during the second stage.

21.8.3 Station

Position. Count the number of sutures—three around the posterior fontanelles and four around the anterior fontanelles.

Moulding. This refers to change in relationship between skull bones. Three degrees of moulding are noted:

- 1+ Suture lines touch.
- 2+ Suture lines overlap and are reducible.
- 3+ Suture lines overlap and are irreducible.

Ideally, the degree of moulding should be assessed across two separate suture lines, and scores are added producing a score out of 6.

Application of the cervix.

Caput formation.

Details of the passages—cervix:

- Effacement.
- Dilatation.

Pelvic anatomy. Clinical pelvimetry is a means of assessing pelvic capacity through a vaginal examination. Unfavourable factors, which may be detected, include prominence of the ischial spines (interspinous width) or

prominence of the ischial tuberosity (intra-tuberous width), narrow subpubic angle (if this is <90°, the diameter of the outlet may be reduced), and proximity of the sacrum to the pubis symphysis (can you reach the sacrum with ease?). Clinical pelvimetry is subjective and results must be interpreted with caution (Fig. 21.6).

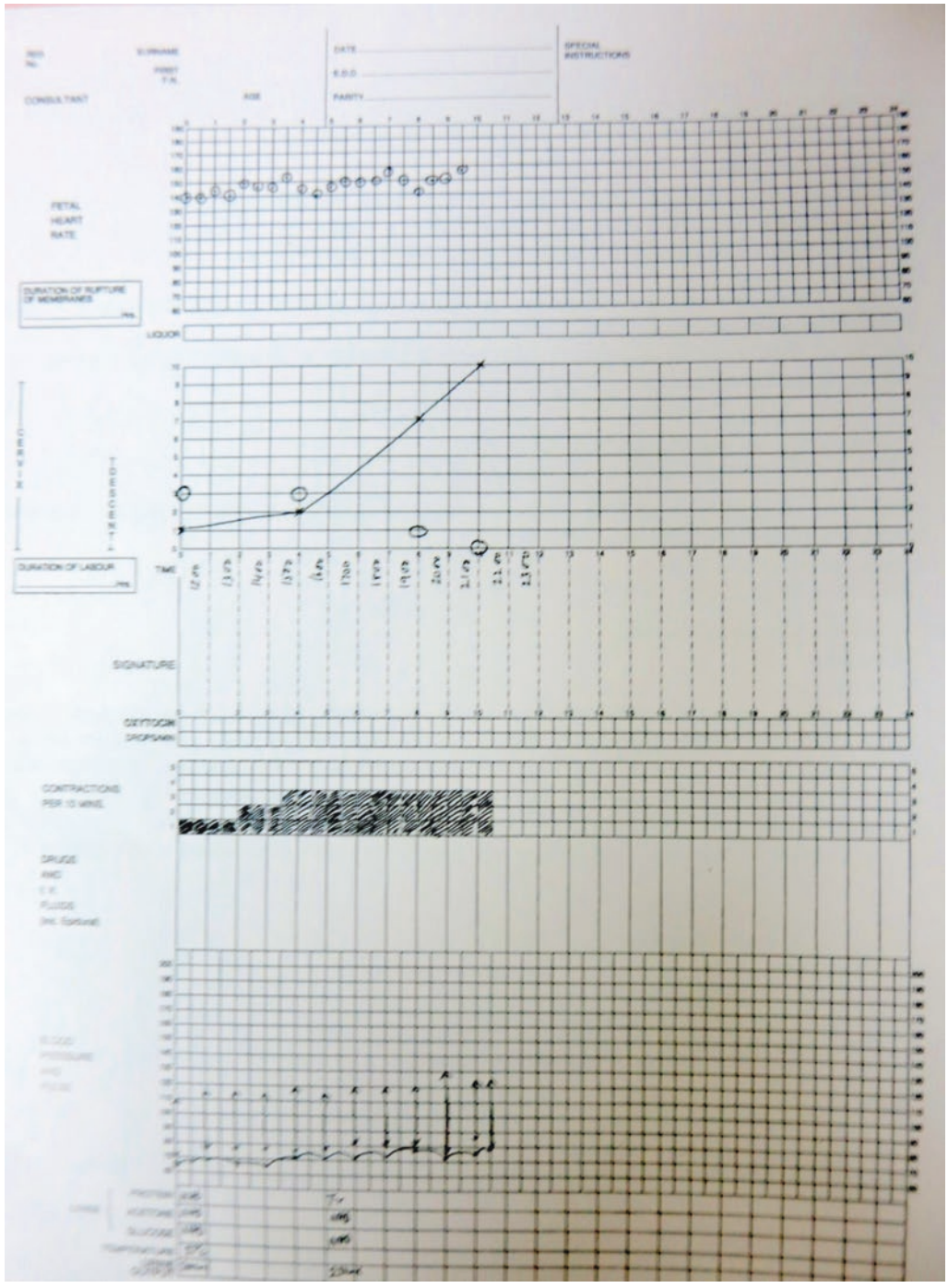


Fig. 21.6 Partogram showing normal progress of labour

With the advent of the partogram, three abnormal first stage patterns of prolonged labour have been described [11]:

1. Prolonged latent phase.
   The latent phase of labour begins with the onset of painful, regular uterine contractions and ends at the start of the accelerative phase of labour when progressive cervical dilatation is evident. Cervical contractions have been demonstrated during early labour particularly in the uneffaced cervix. Cervical contractions are not seen when the cervix dilates beyond 3–4 cm. This may explain the latent phase of labour. The latent phase should not exceed 6 h in a primiparous patient and 4 h in a multip.
2. Primary dysfunctional labour.
   This is defined as progressive cervical dilatation at a rate of less than 1 cm/h during the active phase of labour.
3. Secondary arrest.
   This refers to arrest of cervical dilatation after a period of normal active phase dilatation.

21.9 Management of Labour

21.9.1 Progress in the First Stage of Labour

Progress in the first stage of labour is characterised by changes in the cervix and descent of the presenting part. The cervical changes include progressive dilatation and effacement and movement from a posterior to an anterior position simultaneously. For example, a change in the cervix from a posterior to an anterior position may be regarded as progress even though there may be little change in cervical dilatation. If over a period of 3–4 h there has been little or no progress as defined above, then consideration should be given to the following possibilities:

- The woman is not in labour.
- Progress is not occurring because of inadequate uterine activity, and augmentation with intravenous oxytocin is required.
- Progress is not occurring because of a malposition, e.g. occipitoposterior or malpresentation, e.g. brow, or there is relative disproportion between size of the baby and size of the maternal pelvis.

21.10 Action to Be Taken when Progress in Labour Is Slow

1. If the woman has not established labour and there are no other problems, she may be offered the option of returning home or being transferred to an antenatal ward to await the onset of labour.
2. If labour is established but progress is slow, the midwife may discuss management with the midwife coordinator in the first instance. Intravenous oxytocin may be required.

The senior obstetrician must be informed about any woman given intravenous oxytocin to augment labour. Great care must be exercised if intravenous oxytocin is administered to a multiparous woman as there is a risk of uterine rupture. This decision must be taken by a doctor of at least registrar level.

21.11 Oxytocin Regime

The initial dose is 5 I.U. in 250 mL of N. Saline at a rate of 6 ml per hour via an ALARIS pump. The dose is then titrated against response by the doctor/midwife in attendance.

Serial increases in dose should be made at a 30-min interval doubling the rate of infusion:

2 mL U/min	6 mL/h
4 mL U/min	12 mL/h
8 mL U/min	24 mL/h
16 mL U/min	48 mL/h

Any further increases should only occur if the case has been discussed with senior medical staff.

For all women given intravenous oxytocin, a vaginal examination should be should be performed 2–3 h after starting the infusion to ensure a satisfactory response in labour progress (after

2 h for a multiparous woman, after 3 h for a nulliparous woman).

Senior medical staff must be informed if: There is failure to progress in the first stage of labour despite the use of intravenous oxytocin.

You wish to administer a second bag of intravenous oxytocin.

21.12 Guidelines for the Management of the Second Stage of Labour

The second stage of labour should be characterised by **progressive** descent of the presenting part. For satisfactory progress to be made, there must be strong contractions, occurring approximately 3 in 10 min with 1–2-min relaxation between contractions.

There is no good evidence to limit the second stage of labour. As long as maternal and fetal conditions are satisfactory and there is clear progress with descent of the presenting part, there is no reason to intervene [12]. However, maternal morbidity increases with second stages in excess of 3 h [13].

Epidural analgesia is associated with a prolongation of the second stage. Though progressive descent of the presenting part should occur automatically.

A plan for the management of the second stage of labour is made for each woman. It is essential that the woman's past obstetric history is considered, e.g. there is a history of previous caesarean section, difficult instrumental delivery, shoulder dystocia and preterm delivery.

21.13 General Principles

1. Position—the upright position should be chosen (Cochrane database). Some women may prefer the left lateral position, and if this is adopted, a wedge should be used to prevent supine hypotension. If a woman chooses an alternative position, this option should be facilitated.
2. If the mother has strong urge to push at full cervical dilatation, she should be allowed to push. Progress should be reassessed after 30 min if the presenting part is not visible.
3. In the absence of an urge to push, do not start active maternal pushing until presenting part is at least 2 cm past the spines. This is important when the woman has epidural analgesia and does not always feel an urge to push.
4. In the presence of epidural analgesia, if the presenting part is not well past the spines, give an epidural top up and allow the presenting part to descend. Reassess progress by vaginal examination after 1 h.
5. The influence of epidural analgesia on progress in second stage and mode of delivery is controversial (Howell CJ). The epidural should not be allowed to wear off without the prior consent of the woman.
6. Nulliparous women with an epidural should all be offered oxytocin during second stage (Fig. 21.7).

21.14 Guidelines for the Administration of Oxytocin in the Second Stage of Labour for Nulliparous Women with Epidural Analgesia

21.14.1 Suggested Management

Once full cervical dilatation is diagnosed in a nulliparous woman with an effective epidural and the fetus is presenting by the vertex, then commence intravenous oxytocin (5 units in 250 mL N. Saline) at 2 mU per minute. Double the dosage every 15 min to a maximum of 16 mU per minute or until contractions are 3 or 4 strong in 10 min [12–14].

If after 1 h of oxytocin the fetal head has not descended sufficiently to commence maternal pushing efforts, then alert medical staff to review [15].

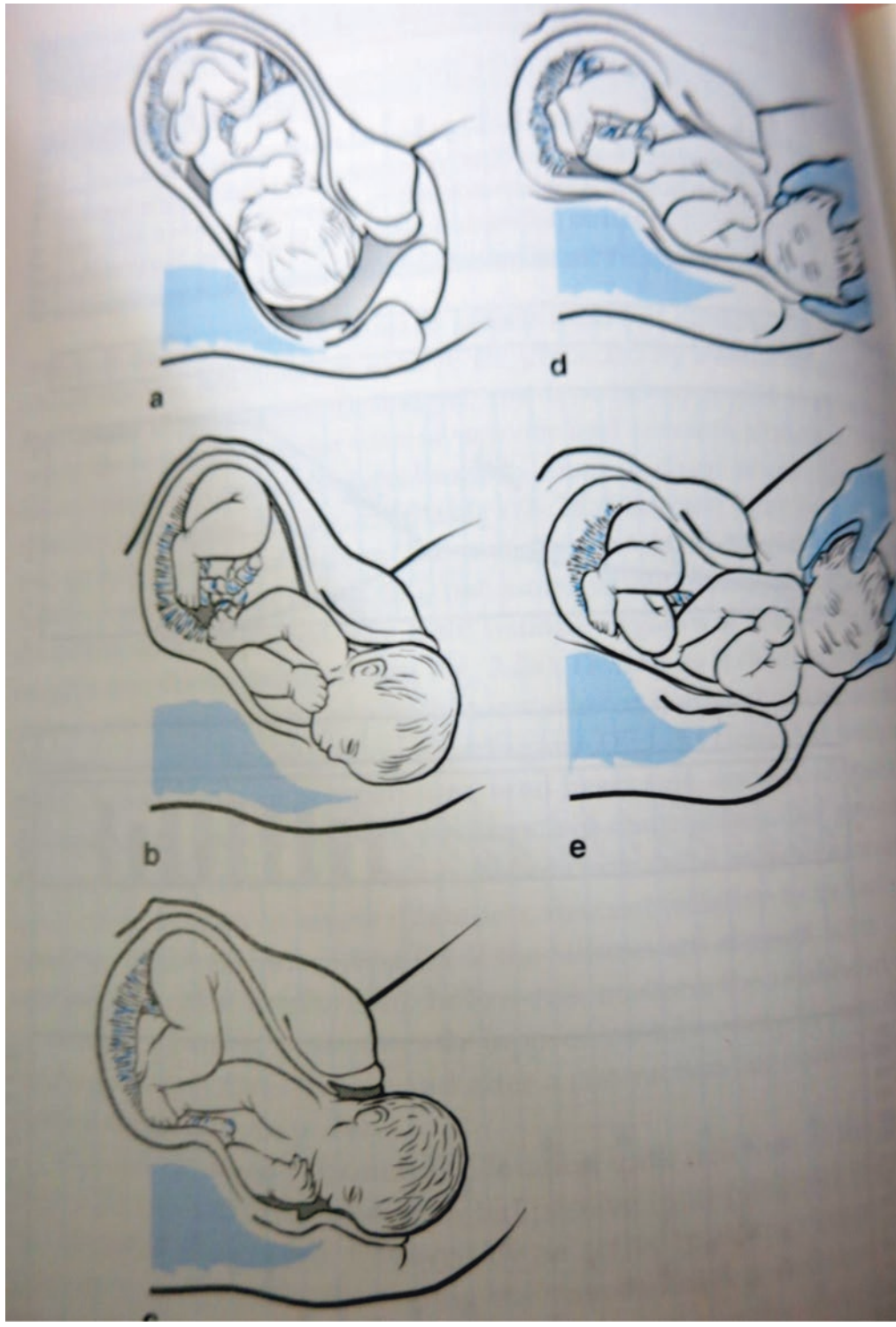


Fig. 21.7 (**a**) Full dilatation. (**b**) Descent of the head with extension and rotation of the head. (**c**) The shoulders remain behind the symphysis pubis. (**d**) External rotation of the head and delivery of the anterior shoulder. (**e**) Delivery of the posterior shoulder

21.15 Failure to Progress in the Second Stage

21.15.1 Guidelines for Management of Nulliparous Women Without Epidural Analgesia and All Multiparous Women

The second stage of labour should be characterised by progressive descent of the presenting part. If no progress has occurred after 1 h, then consider the following:

(a) There is an undiagnosed malposition or malpresentation—if in doubt, please ask senior medical/midwifery staff to re-examine the woman.
(b) Augmenting uterine activity with oxytocin.

Great care must be exercised if a multiparous woman is given oxytocin in the second stage due to the risk of uterine rupture. Oxytocin must not be given to any multiparous woman without discussion with senior resident obstetrician.

21.16 Management of Third Stage of Labour

21.16.1 Active Management

This method is associated with less blood loss than a physiological third stage and requires the administration of either Syntometrine 1 mL (contains ergometrine 0.5 mg and Syntocinon 5 units) or Syntocinon 10 units. Both Syntometrine and oxytocin promote uterine contraction and retraction and are given with delivery of the anterior shoulder. Women without contraindication (see below) should be **offered the choice of either Syntometrine or oxytocin if they wish active management of the third stage. Women should be given the following information to enable them to make an informed choice:** Syntometrine is associated with a significantly higher incidence of nausea and vomiting compared with oxytocin. Syntometrine is associated with a small reduction in the incidence of postpartum haemorrhage when compared with oxytocin. However there is no difference in the incidence of major postpartum haemorrhage (greater than 1000 mL) comparing the two drugs [16].

Syntometrine should be given IM at delivery to a grand multiparous patient (>para 5).

Ergometrine or Syntometrine should not be used in hypertensive disease; cardiac disease, following beta-agonist infusion, i.e. salbutamol or ritodrine; severe asthmatics; and women with Raynaud's disease.

In these situations intramuscular Syntocinon 10 units should be administered.

Aim to complete third stage in less than 30 min from delivery of the baby.

21.16.2 Physiological Management: Refer to Midwifery-Led Guidelines

This is only appropriate for use when the woman has had a normal, physiological labour and is at low risk of a postpartum haemorrhage. In physiological management, there should be:

- No prophylactic oxytocic drug.
- The cord should not be cut or clamped until the placenta is delivered.
- No cord traction but use of maternal effort or assisted by putting the baby to the breast. If the placenta is retained after 1 h, then one further attempt at delivery using gentle fundal pressure should be made. If this is unsuccessful, the labour ward coordinator and medical staff should be informed.

21.16.3 Examination of the Placenta and Membranes

- It is the responsibility of the person who delivers the placenta to check that it is complete (i.e. after an operative delivery, this should be the doctor).
- The examination should be performed as soon as possible after the delivery, and if there is any suspicion that it is incomplete, a senior doctor should be informed.

- If there is a missing cotyledon, exploration of the uterus should be arranged.
- If the membranes appear to be incomplete, a speculum examination should be performed to ascertain if they are visible within the os and can be removed with sponge forceps.

21.16.4 Manual Removal of the Placenta

- If the placenta remains undelivered 30 min after an active third stage or 1 h following physiological management, the registrar should be informed and arrangements made to remove the placenta manually. The registrar should be informed earlier if there is heavy blood loss or the patient's condition is not stable.
- **A partially adherent placenta can result in a very heavy blood loss and should be treated as an emergency.**
  - The procedure may be performed in a labour ward if regional analgesia is used the patient must be transferred to theatre if general anaesthesia is required.
    - Site an intravenous infusion, draw blood for FBC, and cross match 2 units.
    - Adequate analgesia must be ensured and the anaesthetist contacted to top up an external epidural or site a spinal.
    - When it is certain that removal is complete, an infusion of 20 units oxytocin in 500 ml saline should be given over 4 h, during which time the woman must be kept under observation on the labour ward or HDU. An indwelling catheter should be inserted and left until the woman is fit for transfer to the ward.
    - Prophylactic antibiotics (a cephalosporin and metronidazole) should be given after manual removal of the placenta [17] Grade A recommendation RCOG 38th Study Group-Placenta: Basic Science and Clinical Practice).

How Can Dysfunctional Labour Be Corrected?

Correct diagnosis of labour is critical to the management of dysfunctional labour. To diagnose labour there must be painful regular contractions, with associated cervical dilatation and effacement. The diagnosis of labour prior to these criteria being met will lead to inappropriate intervention dystocia of a woman not yet in labour [18].

The active management policy proposed by O'Driscoll in 1983 [3] and supported by recent trials has been shown to depend on a whole package of care for the detection and management of dysfunctional labour, which included help and personal support in labour.

Robson demonstrated a reduction in caesarean section rates in primipara women at term from 7.5% to 2.4% using the following package of care:

Careful attention to the accurate diagnosis of labour.

Good personal support in labour.

Artificial rupture of the membranes at 2 h if the rate of cervical dilatation was less than 1 cm per hour. (Recent studies have suggested that amniotomy alone is ineffective at correcting dystocia.)

Oxytocin to augment labour after a further 2 h if progress remained unsatisfactory.

Two hourly examinations if normal progress was occurring without syntocinon, 4 hourly vaginal examination after oxytocin was commenced unless indicated earlier.

Pushing in the second stage only if the head was low and there was an urge to push.

Oxytocin in the second stage after 1 h if there was no descent of the head.

Oxytocin for the second stage if there was no descent after 30 min of pushing.

Women are encouraged to adopt whatever position they feel most comfortable, avoiding the supine position.

Syntocinon.

This will increase the amplitude, duration and frequency of contractions and should be given by a low-volume

infusion pump. Regimens should commence at a low rate (1 mU/min) and increased at intervals of 30 min with the dose titrated against contractions aiming to achieve 3–4 contractions in 10 min. A maximum rate of 12 mU/min has been suggested. Using this regime reduces the incidence of uterine hypertonicity, reduces syntocinon doses and reduces caesarean section rates for fetal heart rate anomalies [19].

Hyperstimulation is associated with an increase in contraction frequency with a rise in uterine baseline tone with resultant reduced placental perfusion.

21.17 Posture

The woman should be in a posture she feels most comfortable in. Supine position should be discouraged to avoid caval compression. Gravity and movement improve fetal descent. The lateral and upright positions are beneficial since they reduce the second stage of labour, instrumental delivery rates, episiotomy and perineal tear rates and fetal heart rate anomalies [20].

21.18 Pain Relief

Pain suppresses uterine activity via the autonomic nervous system. Anxiety and pain stimulate the sympathetic nervous system. Adrenaline is also a potent inhibitor of uterine activity. Fear increases the perception of pain.

21.19 Support

It reduces analgesic intake, improves Apgar score, may shorten the duration of labour and improves patient satisfaction rate [17, 21].

21.20 Empathy

Ensure that mothers are informed and psychologically prepared for labour.

Ensure the correct diagnosis of labour.

Provide emotional and physical support to the mother in labour.

Regularly assess progress in labour and record your findings on the partogram.

In cases of slow progress, attempt to ascertain the cause (passenger, passage and power).

21.21 Be Cautious

Using syntocinon in multiparous patients or where a uterine scar is present.

When performing instrumental deliveries when the first stage of labour has been dysfunctional especially if the dilation interval from 7–10 cm exceeds 3 h.

Not to confuse caput for descent of the fetal head when performing second stage Caesarean sections.

21.22 Conclusion

One should be comfortable, understand and diagnose, normal labour at the end of this chapter. Recognize dysfunctional labour, when reviewing partograms, recognise the potential causes of dysfunctional labour and be able to appropriately manage and counsel in labour.

References

1. Effective procedures. In: Maternity care suitable for audit. London: RCOG; 1997.
2. Recommendations arising from the 26th RCOG study group: intrapartum fetal surveillance, In: Spencer JAD, Ward RHT, editors. Intrapartum fetal surveillance. London: RCOG press; 1993. 1969; 2(655).
3. O'Driscoll K, Jackson RJ, Gallagher JT. Prevention of prolonged labour. Br Med J. 1969;2(5655):477–80.
4. Spiby et al. 2008; Jansssen et al. 2006.
5. Gross et al. 2006; Albers 2001; Enkin et al. 2000.
6. Simkin and Ancheta 2000.NICE 2007.
7. Evidence Based Guidelines for Midwifery-Led Care in Labour ©The Royal College of Midwives 2012. WHO/EHT/CPR 2004 reformatted. 2007.
8. WHO/EHT/CPR 2004 reformatted. 2007. WHO Surgical Care at the District Hospital 2003.
9. Friedman EA. Evolution of graphic analysis of labour. Am J Obstet Gynaecol. 1978;132(7):824–8275.

10. World Health Organisation. Safe Motherhood Programme. WHO partograph in management of labour. Lancet. 1994;343(8910):300–1404.
11. Gee H, Brown JS. Cervical contraction: the response of the cervix to oxytocic stimulation in the latent phase of labour. Br J Obstet Gynaecol. 1993;100:635–40.
12. Paterson CM, et al. The characteristics of the second stage of labour singleton deliveries in the North West Thames Health Region, 1988. Br J Obstet Gynaecol. 1992;99:377–80.
13. Saunders NS, et al. Neonatal and maternal morbidity in relation to the second stage of labour. Br J Obstet Gynaecol. 1992;99:381–5.
14. Howell CJ. Epidural versus non-epidural analgesia for pain relief in labour. Cochrane Database Syst Rev. 2000;(2):CD000331.
15. Saunders NJ, et al. Oxytocin infusion during second stage of labour in primiparous women using epidural analgesia: a randomised double blind placebo controlled trial. BMJ. 1989;299:1423–6.
16. McDonalds S, Prendiville WJ, Elbourne D. Prophylatic Syntometrine versus oxytocin for delivery of the placenta. Cochrane Database Syst Rev. 2000;(2):CD000201.
17. Hodnett ED. Caregiver support for women during childbirth. Cochrane Database Syst Rev. 2000;(3):CD000199.
18. Robson A, Sudamore JW, Walsh SM. Using the audit cycle to reduce caesarean section rates. Am J Obstet Gynaecol. 1996;174:199–205.
19. Induction of labour RCOG Greentop Guideline March 2000.
20. Gupta JK, Nikodem VC. Woman's position during second stage of labour. Cochrane Database Syst Rev. 2000;(2):CD002006.
21. Kennell J, Klaus M, McGrath S, Robertson S, Hinkley C. Continuous emotional support during labour in a US Hospital: a randomised control trial. JAMA. 1991;265:2197.

Induction of Labor

22

Kanan Yelikar and Sonali Deshpande

22.1 Introduction

Induction of labor is one of the most commonly performed obstetrical procedures all over the world. Induction of labor refers to techniques for stimulating uterine contractions to accomplish delivery prior to the onset of spontaneous labor [1]. Augmentation of labor refers to increasing the frequency and improving the intensity of existing uterine contractions in a patient who is in labor and not progressing adequately, in order to accomplish vaginal delivery [1].

22.2 Indications for Delivery Before Onset of Labor

22.2.1 Obstetrical and Medical Indications

Delivery before the onset of labor is indicated when the maternal/fetal risks associated with continuing the pregnancy are thought to be greater than the maternal/fetal risks associated with early delivery [2]. Induction is generally preferred when there are no contraindications to labor and vaginal birth. The relative risk of delivery versus continuation of pregnancy is influenced by factors such as gestational age, presence/absence of fetal lung maturity, severity of the clinical condition, cervical status, and maternal demographic factors. However, the magnitude of maternal/fetal risk of early delivery can rarely be determined with precision.

Examples of conditions where induction is often indicated include, but are not limited to [2]:

- Prolonged pregnancy.
- Prelabor (premature) rupture of membranes.
- Preeclampsia, eclampsia, HELLP syndrome (hemolysis, elevated liver enzymes, low platelets).
- Fetal demise.
- Maternal diabetes.
- Fetal growth restriction.
- Chorioamnionitis.
- Abruptio placentae.
- Oligohydramnios.
- Cholestasis of pregnancy.
- Alloimmunization with fetal effects.

Elective or Marginally Indicated Induction at Term Induction of labor without medical indication is termed as elective induction of labor. Sometimes this is done for reasons like previous history of precipitate labor, distance from the hospital, or social reasons. However,

K. Yelikar (✉)
Department of OB/GYN, Govt. Medical College & Hospital, Aurangabad, India

S. Deshpande
Govt. Medical College & Hospital, Aurangabad, India

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the potential advantages are reduction in stillbirth, macrosomia and meconium passage. It is also done to ensure that the delivery occurs at the time when hospital has optimum number of staff. In addition, the risk of delivery en route to the hospital is reduced among patients with a history of rapid labor or who live far from the hospital. However there is insufficient evidence to support this practice as routine. The major concerns associated with elective ("social") or marginally indicated induction at term are the potential for cesarean delivery in latent phase, the increased length of labor, neonatal morbidity if delivery is before 39 weeks of gestation [3], and cost [4–6].

There is expert consensus that elective induction should not be performed before 39 weeks of gestation because of the increased risk of neonatal morbidity and mortality. In particular, the following clinical scenarios are not indications for early term induction (i.e., at 37–38 weeks): maternal anxiety or discomfort related to normal pregnancy; a previous pregnancy with labor abnormalities, shoulder dystocia, or rapid labor; suspected macrosomia; or the distance the mother lives from the hospital [7].

22.3 Contraindications to Induction

In each of the following settings, there is general consensus that the maternal/fetal risks associated with labor and vaginal delivery and induction of labor are usually contraindicated:

- Prior classical or other high-risk cesarean incisions.
- Prior uterine rupture.
- Prior transmural uterine incision entering the uterine cavity.
- Active genital herpes infection.
- Placenta previa or vasa previa.
- Umbilical cord prolapse or persistent funic presentation.
- Transverse fetal lie.
- Invasive cervical cancer.
- Category III fetal heart rate tracing.
- Severe fetal growth restriction with signs of fetal compromise on Doppler.

22.4 Predicting a Successful Induction

The probability of successful labor induction varies widely depending upon several factors, including:

- Characteristics of the population being induced (e.g., nulliparous or multiparous, intact or ruptured membranes, baseline cervical status, placental insufficiency present or absent, gestational age, previous vaginal delivery, mean newborn size, maternal height and body mass index) [8, 9].
- Choice of endpoints (e.g., delivery within 24 h, delivery within 48 h, dose/duration of oxytocin, interval from preinduction cervical ripening to delivery versus time from induction to delivery, route of delivery, maternal and neonatal morbidity).
- Cervical status is another important factor for predicting the likelihood of successful induction. The bishop score appears to be the best available tool for assessing cervical status and predicting the likelihood that induction will result in vaginal delivery. Systematic reviews of controlled studies concluded the bishop score is as, or more, predictive of the outcome of labor induction than fetalfibronectin [8] or sonographic measurement of cervical length [8, 10, 11] and that dilation is the most important element of the bishop score [8]. Other cervical scoring systems like fields system, Burnett, Caldor, and Friedman modifications of the bishop system are also available for this purpose [12]. The time of day when induction is started does not appear to be a factor in success [13].

Modified Bishop's Score. The modified Bishop score is the cervical assessment system most commonly used in clinical practice [14]. This system tabulates a score based upon the station of the presenting part and four characteris-

tics of the cervix: dilatation, effacement, consistency, and position.

Modified Bishop Scoring System

	0	1	2	3
Dilation, cm	Closed	1–2	3–4	5–6
Effacement, percent	0–30	40–50	60–70	≥80
Station*	−3	−2	−1,0	+1, +2
Cervical consistency	Firm	Medium	Soft	
Position of the cervix	Posterior	Midposition	Anterior	

A Bishop score ≥ 8 suggests the changes of having a vaginal delivery are good and the cervix is considered favorable or ripe for induction. If the Bishop score is ≤ 6, the chances of having a vaginal delivery are low and the cervix is considered unfavorable or unripe for induction. A simplified Bishop score can be calculated using only dilation, station, and effacement. Using these three variables, a simplified Bishop score ≥ 5 has a similar predictive value for vaginal delivery as a classic Bishop score ≥ 8
*Based on a −3 to +3 scale

22.4.1 Pre-Induction Cervical Ripening

Cervical ripening is the term used to denote the process of cervical softening, thinning, and dilatation with the goal of reducing the rate of failed induction and induction delivery interval. Unfavorable Bishop's score is an indication for cervical ripening.

- **Sweeping of fetal membrane is** also called as stripping of membranes and a simple outpatient technique which strips amniotic membranes of lower uterine segment. It is recommended for reducing formal induction of labor in nulliparous woman at 40 and 41 weeks and parous woman at 41 weeks (NICE guidelines 2008). Sweeping of membranes results in increase local production of prostaglandins from the decidua and adjacent membranes and results in initiation of labor.
- **Prostaglandin** administration causes dissolution of collagen bundles and an increase in the submucosal water content of the cervix. These changes lead to a cervical state that is associated with greater success when labor is induced with oxytocin [15]. Prostaglandins also cause the uterus to contract and may initiate labor. Prostaglandins are preferred method of cervical ripening in women with an unscarred uterus. Although excessive uterine activity is a disadvantage of prostaglandin use, these agents have a good safety profile in women with unscarred uteruses: The neonatal complications like low Apgar score, neonatal intensive care unit admission, meconium staining, and cesarean delivery are equivalent to the rates associated with use of balloon catheters for cervical ripening [16].

Uterine activity and fetal heart rate monitoring are indicated for at least 30 min after administration of prostaglandins for cervical ripening and should be maintained as long as regular uterine activity is present [17].

If labor does not ensue or is not progressing adequately after administering prostaglandins, repeated doses can be given. Alternatively, oxytocin may be initiated, or, if technically possible, amniotomy can be performed and appears to be more effective than repeated dosing with prostaglandin [5].

Prostaglandin E1 (Misoprostol). Misoprostol, a synthetic PGE1 analogue, is an effective drug for induction of labor. It is cheap and stable at room temperature and very useful in developing countries. The American College of Obstetricians and Gynecologists (ACOG) has indicated that use of misoprostol appears safe and efficacious when used as a cervical ripening and/or labor induction agent when utilized judiciously. Though 50 mcg dose is more effective than 25 mcg (e.g., higher rate of vaginal delivery and lower rate of oxytocin use), 25 mcg dose is safer (lower rate of tachysystole, hyperstimulation, cesarean deliveries for nonreassuring fetal heart rate, NICU admissions, and meconium passage) [18]. Therefore, lower doses, such as 25 mcg, should be used initially, with redosing intervals of 4–6 h [19–21]. The World Health Organization (WHO) suggests 25 mcg every 6 h [22] maximum up to 150 μg in 24 h. The

time interval between the final dose and initiation of oxytocin should be around 4 h because of the potential possibility for uterine tachysystole with concurrent oxytocin and misoprostol administration.

Routes of Administration of Misopristol. Vaginal route is the most preferred route. Oral route is convenient and effective [23]. The concentration of orally administered misoprostol peaks sooner and declines more rapidly than with vaginal administration [24]. Buccal or sublingual administration is a novel approach which avoid first pass hepatic metabolism associated with oral ingestion and may therefore increase bioavailability similar to that achieved with vaginal administration.

Contraindications to Misoprostol. Prostaglandins are not used for cervical ripening or labor induction in term pregnancies with a prior cesarean birth or other prior major uterine surgeries (e.g., extensive myomectomies and hysterotomies) because of the increased risk for uterine rupture [6]. Pre-existing uterine activity is a relative contraindication to use of prostaglandins. Addition of an exogenous uterotonic agent, in the presence of any degree of antecedent uterine activity, will cause excessive uterine activity. For patients having frequent, low-amplitude, painless contractions or ≥2 painful contractions/10 min as baseline uterine activity or who have received one dose of prostaglandin, second dose should be delayed or avoided, as there appears to be cumulative uterotonic effect.

Side effects of misoprostol include tachysystole, hyperstimulation, fever, chills, vomiting, and diarrhea. The frequency of these side effects depends on the type of prostaglandin, dose, and route of administration. Uterine contractile abnormalities are encountered in up to 30% of cases depending on the route of administration; other systemic effects occur in up to 5% of cases.

Prostaglandin E2. Prostaglandin E2 preparation is available for cervical ripening. Intracervical gel contains 0.5 mg of Dinoprostone gel for endocervical administration. The dose can be repeated in 6–12 h if cervical change is inadequate and uterine activity is minimal following the first dose. The manufacturer recommends that the maximum cumulative dose of Dinoprostone not exceed 1.5 mg (i.e., three doses) within a 24-hour period. The time interval between the final dose and initiation of oxytocin should be around 6 h because of the potential for uterine tachysystole with concurrent oxytocin and prostaglandin administration. PGE2 is contraindicated in bronchial asthma and heart disease.

Mechanical Methods. Mechanical methods are among the oldest approaches used to promote cervical ripening. Advantages of these techniques compared with pharmacologic methods include their low cost compared with some drugs, low risk of tachysystole, few systemic side effects, and convenient storage requirements (no refrigeration or expiration, which are issues for some drugs). Disadvantages include a possible small increase in the risk of maternal and neonatal infection from introduction of a foreign body [11], the potential for disruption of a low-lying placenta, some maternal discomfort upon manipulation of the cervix, and frequent need for oxytocin induction of labor.

The most common mechanical method is insertion of a balloon catheter.

These methods likely work, at least in part, by causing the release of prostaglandin F2-alpha from the decidua and adjacent membranes or prostaglandin E2 from the cervix. In addition, insertion of a catheter or dilator directly dilates the cervix.

Under strict aseptic precaution, a 24 F Foley catheter with 30–50 mL bulb is inserted into the cervix and passed into the internal os and into the extraamniotic space. The bulb is then filled with 30–50 mL saline, and the Foley is pulled back gently, so that the bulb rests against the internal os. The catheter is then strapped to the patient's thigh. The catheter is left in place until it is extruded or for up to 6–12 h.

Mifepristone (RU486) is a progesterone receptor antagonist. The rationale behind its use for induction of labor is that in spontaneous labor, a fall in progesterone is one of the main factors leading to onset of labor. When compared to placebo, it is more effective in increasing the chances of spontaneous labor and reducing the need for

prostaglandins. Usually after 48 h, the ripening of the cervix occurs. Dose: tablet 200 mg orally.

22.4.2 Labor Induction

Once the cervix becomes favorable, labor is induced, usually with oxytocin, with or without amniotomy. Prostaglandins used for ripening the cervix may also stimulate uterine contraction, and labor may follow. Mechanical methods of cervical ripening are usually followed by oxytocin to induce labor. Prostaglandins are very effective in ripening, effacing, and dilating the cervix to the extent required for amniotomy. Amniotomy in itself may be ineffective without use of oxytocin, and in any case, it cannot be done unless the cervix is dilated enough to allow the passage of instrument. Oxytocin is most effective when the cervix is already favorable or when membranes have ruptured.

Oxytocin. Oxytocin is a polypeptide hormone produced in the hypothalamus and secreted from the posterior lobe of the pituitary gland in a pulsatile fashion. It is identical to its synthetic analogue, which is among the most potent uterotonic agents.

Synthetic oxytocin administration is a proven method of labor induction [25]. Exogenous oxytocin administration produces periodic uterine contractions first demonstrable at approximately 20 weeks of gestation. Myometrial responsiveness increases with advancing gestational age until 34 weeks, at which time it levels off until spontaneous labor begins, when it increases rapidly [26]. Increases in myometrial sensitivity are due primarily to increases in myometrial oxytocin binding sites [27]; progress during spontaneous labor is not related to increasing oxytocin concentration, uterine contractions are not associated with changes in plasma oxytocin concentration, and hypocontractile labor does not appear to be the result of a deficit of oxytocin [28].

Regimen. Oxytocin is most commonly given intravenously. It cannot be administered orally because the polypeptide is degraded into small, inactive forms by gastrointestinal enzymes. The plasma half-life is short, estimated at 3–6 min [29]. Steady-state concentrations are reached within 40 min of initiation or dose change [30].

Infusion pumps are used to allow continuous, precise control of the dose administered. A common contemporary practice is to make a solution of 5 units of oxytocin in 500 mL crystalloid (10 mU in 1 mL). In the absence of infusion pumps, the drops per minute have to be titrated and monitored accurately.

When uterotonic drugs are administered, continuous monitoring of uterine activity and fetal heart rate is important so that the dose can be adjusted up or down if uterine activity is inadequate or excessive. The maximum dose of oxytocin has not been established and the end point is usually the achievement of uterine contractions of at least 3 in 10 min each lasting for 40 s.

Some examples of oxytocin regimens are described below:

- **Low dose**—Low-dose protocols are based on the pharmacokinetics of oxytocin [17]. The dose of oxytocin is initiated at 0.5–1 mU/min and increased by 1 mU/min at 30–40-min intervals. This interval is based upon studies showing approximately 40 min are required for any particular dose of oxytocin to reach a steady-state concentration and maximal uterine contractile response [30].
- Slightly higher doses (begin at 1–2 mU/min and increase by 1–2 mU/min) and shorter incremental time intervals (15–30 min) have also been recommended [31, 32].
- **High dose—**Active management of labor regimens, and others, use a high-dose oxytocin infusion with short incremental time intervals [33, 34]. A maximum oxytocin dose has not been established; however, most labor and delivery units do not go above 40 mU/min. The most common complication of high-dose regimens is uterine tachysystole.

Regimen	Starting dose (mU/min)	Incremental dose	Dosage interval
Low dose	0.5–2	1–2 mU/min	15–40 min
High dose	6	3–6 mU/min	15–40 min

- **Pulsatile**—Pulsatile administration of intravenous oxytocin at 6–10-minute intervals is

effective and may better simulate normal labor than continuous oxytocin administration [35, 36]. Advantages of pulsatile administration include significantly lower total doses of oxytocin and less hyperstimulation. However, pulsatile administration does not reduce the rate of failed vaginal birth. The time from the start of the infusion to delivery is not shortened and may be longer than with continuous oxytocin administration. Pulsatile oxytocin administration requires special equipment and is rarely used in contemporary obstetric practice.

Amniotomy. Amniotomy alone may be an effective method of labor induction but can only be performed in women with partially dilated and effaced cervices [37]. To reduce the risk of cord prolapse, the clinician should ensure that the fetal vertex is well applied to the cervix and the umbilical cord or other fetal part is not presenting. The fetal heart rate before and after the procedure should be documented, and also note the color of the amniotic fluid.

The combination of amniotomy plus intravenous oxytocin administration was more effective for labor induction than amniotomy alone [38].

22.5 Complications and Side Effects

All methods of labor induction carry risks and are associated with side effects.

22.5.1 Oxytocin or Prostaglandins

Abnormal uterine contraction. Abnormal or excessive uterine contractions can occur with the use of prostaglandin compounds or oxytocin.

Tachysystole is defined as more than five contractions in 10 min, averaged over a 30-minute window [39].

Uterine hypersystole/hypertonus—a contraction lasting at least 2 min with a normal fetal heart rate.

Uterine hyperstimulation—tachysystole or hypertonus associated with non-reassuring fetal heart rate pattern.

Since uterine activity causes intermittent interruption of blood flow to the intervillous space, excessive uterine activity that exceeds the critical level for an individual fetus will result in fetal hypoxemia. This, in turn, can lead to category II or III fetal heart rate patterns and fetal acidosis [40–44].

The various prostaglandin preparations have up to a 5% rate of uterine tachysystole, which is usually well tolerated and not associated with an adverse outcome. The reported risk of tachysystole with oxytocin varies widely. Tachysystole occurs more frequently when higher doses of oxytocin or prostaglandins are used [45–48].

Concurrent administration of oxytocin and a prostaglandin is believed to increase the risk of tachysystole since both drugs carry a risk of this complication.

Management of tachysystole/hyperstimulation—In case of tachysystole/hyperstimulation:

Turn the patient in left lateral position.

Administer oxygen by face mask (10 L/min).

Give intravenous fluids (e.g., fluid bolus of 500 mL of lactated Ringer's solution or more).

Discontinue the intravenous oxytocin infusion.

Cervical/vaginal lavage is not helpful in case of prostaglandin gel.

Remnants of a misoprostol tablet can be removed from the vagina if necessary.

Administer a tocolytic if hypertonus does not respond to discontinuation of drug, i.e., inj. Terbutaline 250 mcg subcutaneous or intravenous.

Proceed with cesarean section if abnormal FHR does not improve with these measures.

Uterine rupture—uterine rupture is an unusual event, during induction of labor, particularly in the unscarred uterus. The condition is associated with very high maternal and fetal morbidity and mortality. It is more common in previously scarred uterus (cesarean scar, myomectomy, or uterine curettage with an undiagnosed perforation). It can also occur in multigravidas with previous vaginal deliveries and rarely in primigravida.

Amniotic fluid embolism—it is a rare but dreadful complication of induction of labor.

Side effects of oxytocin—adverse maternal effects from oxytocin administration include cardiovascular instability (hypotension, tachycardia, myocardial ischemia, arrhythmias), nausea, vomiting, headache, and flushing [49]. Rarely, large doses have caused water retention, leading to hyponatremia.

Hyponatremia—oxytocin has a similar structure to vasopressin (antidiuretic hormone) and can cross-react with the renal vasopressin receptor. If high doses (e.g., 40 mU/min) of oxytocin are administered in large quantities (e.g., over 3 L) of hypotonic solutions (e.g., 5% dextrose in water [D5W]) for prolonged periods of time (≥7 h [50–52]), excessive water retention can occur and result in severe, symptomatic hyponatremia, similar to the syndrome of inappropriate antidiuretic hormone secretion [53, 54].

Symptoms of severe acute hyponatremia include headache, anorexia, nausea, vomiting, abdominal pain, lethargy, drowsiness, unconsciousness, grand mal type seizures, and potentially irreversible neurologic injury.

If water intoxication occurs, oxytocin and any hypotonic solutions should be stopped. Correction of hyponatremia must be performed carefully and consists of restricting water intake and careful administration of hypertonic saline if the patient is symptomatic. **Hypotension**—hypotension can result from rapid intravenous injection of oxytocin. Although this is most commonly observed at cesarean delivery when large oxytocin boluses (>5 units) are given to prevent postpartum hemorrhage [55–58], it is prudent to administer oxytocin for induction by infusion pump or slow drip to avoid adverse cardiovascular effects, as well as tachysystole.

Neonatal hyperbilirubinemia—use of oxytocin has been associated with neonatal hyperbilirubinemia in some studies, but not others. Hyperbilirubinemia may be more related to factors related to induction (e.g., preterm pregnancy complications) than to a direct effect of oxytocin.

Side effects of prostaglandins—side effects of prostaglandins include fever, chills, vomiting, and diarrhea. The frequency of these side effects depends on the type of prostaglandin, dose, and route of administration.

Amniotomy—complications of amniotomy include removal of barrier to infection, disruption of an occult placenta previa, rupture of a vasa previa, and umbilical cord prolapse. Many of these same risks, and inadvertent amniotomy, also apply to membrane stripping.

Failed induction—induction of labor usually culminates in vaginal delivery; this occurs less often than when women enter labor spontaneously. A low Bishop score, before or after attempts at cervical ripening, is a poor prognostic factor for successful induction.

There is no universal standard for what constitutes a failed induction. The key principle is to allow adequate time for cervical ripening and development of an active labor pattern before determining that an induction has failed. This minimizes the number of cesarean deliveries performed for failed induction in patients who are progressing slowly because they are still in the latent phase of labor [59–61]. Criteria for failed induction are not uniform but can be defined as failure to generate regular contractions approximately every 3 min and cervical change after at least two doses of prostaglandins and 24 h of oxytocin administration [62]. Membranes should be artificially ruptured, if safe and feasible. After rupture of membranes, the induction may be considered a failure if regular contractions and cervical change do not occur after at least 12 h of oxytocin administration. If induction fails, the following options should be discussed with the woman:

1. A further attempt to induce labor.
2. Cesarean section.

22.6 Induction of Labor in Special Situations

Prolonged pregnancy—an ultrasound to confirm gestation should be offered before 20 weeks of gestation, as this reduces the need for induction for perceived post-term pregnancy. Women

with uncomplicated pregnancies should be offered induction of labor beyond 41 weeks. From 42 weeks, woman who declines induction of labor should be offered increased antenatal monitoring consisting of a twice-weekly CTG and ultrasound estimation of maximum amniotic pool depth.

Preterm prelabor rupture of membranes— induction of labor should not be carried out before 34 weeks unless there are additional obstetric indications (infection or fetal compromise); after 34 weeks, decision should be made depending upon risk to the mother and fetus and local availability of NICU.

The choice of prostaglandins is the same as that with intact membranes. Both intracervical PGE2 and misoprostol can be used if the cervix is unfavorable. Vaginal prostaglandins do not increase the risk of infection with ruptured membranes.

Prelabor rupture of membranes at term— induction of labor is recommended for women with prelabor rupture of membranes at term within 24 h. With induction, there was no increase in cesarean section rate but a significant reduction in the risk of chorioamnionitis or endometritis and a reduced number of neonates requiring admission to the neonatal intensive care unit. The beneficial effect was more evident with oxytocin. Oxytocin is regarded as the first option, though prostaglandins should be considered if the Bishops score is low. ACOG guidelines support 25 μg intravaginal misoprostol every 3–6 h or 50 μg per vaginal every 6 h. At this time IV oxytocin is preferred method for induction for PROM.

Intrauterine fetal demise in late second or third trimester—in the event of an intrauterine fetal death, if the woman is physically well, her membranes are intact, and there is no evidence of infection, and bleeding choice of expectant management can be offered, but if any of the above condition is present, then immediate induction of labor is the preferred option. Oral mifepristone followed by intracervical PGE2 or misoprostol is the preferred method of induction of labor at all gestational ages.

FIGO recommendation for doses of misoprostol in intrauterine fetal death

IUFD in third trimester	25 mcg vaginally six hourly or 25 mcg orally two hourly
IUFD in second trimester	13–17 weeks 200 mcg vaginally six hourly (max four doses)
	18–26 weeks 100 mcg vaginally six hourly (max four doses)

In patients having unfavorable cervix, away from term. Extraamniotic ethacridine lactate with intracervical catheter along with high concentration oxytocin drip is more effective. Intravaginal misoprostol (high dose) is superior to oxytocin before 24 weeks. Aggressive induction for pressing indication (as eclampsia with IUFD) away from term, extra amniotic ethacridine lactate with foleys catheter in situ along with intracervical PGE2 gel. After 6 h tab. Misoprostol every 4 h till the desired effect.

Fetal macrosomia—there is no evidence that induction of labor is beneficial in suspected fetal macrosomia. As accurate estimation of birth weight is difficult even with use of ultrasound, routine induction of labor is not recommended in suspected fetal macrosomias.

Fetal growth restriction—if there is severe fetal growth restriction with confirmed fetal compromise, induction of labor is not recommended. Fetal growth restriction with severe oligohydramnios and compromised fetus, IOL should not be offered as these babies are at a higher risk of perinatal death due to uteroplacental insufficiency, lower metabolic reserves due to intrauterine malnutrition or pre-existing hypoxia and an umbilical cord more prone to compression due to a reduction in amniotic fluid volume. Continuous intrapartum CTG is advocated in IOL in complicated and high-risk pregnancies.

Breech presentation—induction is not routinely recommended if fetus is in breech presentation in view of maternal fetal morbidity, but if external cephalic version is unsuccessful and woman declines the cesarean section, then induction of labor with vaginal prostaglandins may be offered after discussing the risk. The perinatal outcome is definitely better with elective LSCS, hence the preferred mode of delivery.

Previous cesarean section—in case of previous scar if vaginal delivery is indicated, then induction with intracervical PGE2 gel may be done. But risk of emergency cesarean section and uterine rupture should be discussed with the woman. Misoprostol is contraindicated for induction at term with a previous scar. Labor induction in previous LSCS needs clinical precautions, specific consent, and sound judgment. Judgment comes from experience! Experience comes from poor judgment! Vaginal birth after C-section is possible only with a single previous vaginal delivery, average birth weight, normal progress of labor, and ripe cervix. Risks of maternal morbidity with failed VBAC is 14.1%. With planned repeat C. Section it is 3.6%, and with successful VBAC it is 2.4%.

It clearly means that VBAC has to be chosen very very carefully and moreover the decision to induce labor in previous LSCS is controversial and definitely needs a well-equipped hospital and trained personnel. The management plan includes a gradual and step-up methodology. After stripping of membranes, wait for spontaneous onset of labor with fetal monitoring, if Bishops score >8 do amniotomy & oxytocin. In unfavorable cervix do Foley's catheter or PGE2 gel.

Induction of labor for maternal request—induction should not be offered on maternal request alone. Under exceptional circumstances, induction may be considered at 39–40 weeks.

22.7 Conclusion

Induction of labor in special situations is challenging to the obstetrician. Individualization of the case is a must and much depends on the skill and will of both the clinician and the patient. When induction of labor takes place in women with recognized risk factors, it should take place in a well-equipped hospital with facilities for monitoring and emergency C-section and availability of skilled personnel.

22.8 Summary and Recommendations

- Induction of labor is one of the most commonly performed procedures in obstetrical medicine.
- Induction of labor is indicated when continuing the pregnancy is thought to be associated with greater maternal or fetal risk than intervention to deliver the pregnancy, and there is no contraindication to vaginal birth.
- The bishop score is the best available tool for predicting the likelihood that induction will result in vaginal delivery. A score ≥ 8 out of 13 is associated with a probability of vaginal delivery.
- Avoid elective induction of labor at term as it has got the potential for iatrogenic prematurity, long labor, and higher healthcare costs, without proven medical/obstetric benefits.
- The modified bishop score is the best available tool for predicting the likelihood that induction will result in vaginal delivery. A score ≥ 8 out of 13 is associated with a probability of vaginal delivery.
- Cervical ripening methods fall into two main categories: Pharmacologic and mechanical. Because the state of the cervix plays such a crucial role in labor induction, a cervical examination is essential before determining which method to use for labor induction.
- The most commonly used approach includes use of prostaglandins such as Dinoprostone and misoprostol as well as intracervical Foley balloon placement.
- Membrane sweeping is effective in reducing the interval to spontaneous onset of labor and the overall duration of pregnancy when performed at term.
- For women with favorable cervices undergoing induction, administration of oxytocin with early amniotomy should be practiced. Implementation of a standardized oxytocin induction protocol can minimize errors in oxytocin administration, as well as complications from oxytocin administration.
- If tachysystole with fetal heart rate changes occurs, uterotonic drugs should be stopped. If there is no prompt response to discontinuation of the uterotonic and supportive measures (e.g., left lateral position, intravenous fluid bolus), administer a tocolytic, such as terbutaline 250 mcg subcutaneously or intravenously or atosiban 6.75 mg intravenously over 1 min for fetal resuscitation. Nitroglycerin

60–90 mcg intravenously is usually successful in recalcitrant cases.

- Because oxytocin induction is less successful when used in women with a low bishop score (≤6 out of 13), a ripening process is generally indicated prior to administering oxytocin in these cases.
- There is no universal standard for defining a failed induction. It is important to allow adequate time for cervical ripening and development of an active labor pattern before determining that an induction has failed. Failed induction is defined as failure or inability to generate regular contractions every 3 min and cervical change after at least 24 h of oxytocin administration. Membranes should be artificially ruptured when safe and feasible. After rupture of membranes, a failed induction is defined by absence of regular contractions and cervical change after at least 12 h of oxytocin administration.
- For women with favorable cervices undergoing induction, administration of oxytocin with early amniotomy is more useful than amniotomy alone. Use of oxytocin alone without amniotomy is also reasonable.
- High-dose oxytocin regimens decrease the time from admission to vaginal delivery, but do not appear to decrease the incidence of cesarean delivery compared to low-dose therapy. High-dose regimens are associated with a higher rate of tachysystole than low-dose regimes, but no significant difference in neonatal outcomes.
- High doses of oxytocin should not be administered in hypotonic solutions, as this can lead to excessive water retention and dilutional hyponatremia.

References

1. Wing DA, Frinelli CK. Abnormal labour induction of labour. In: Gabbe SG, Niebyl JR, Simpson JL, London MB, Galan HL, Jauniaux ERM, Driscoll DA, editors. Obstetrics, normal and problem pregnancies. 6th ed. Philadelphia: Saunders Elsevier Publication; 2013. p. 286–310.
2. Ezebialu IU, Eke AC, Eleje GU, Nwachukwu CE. Methods for assessing pre-induction cervical ripening. Cochrane Database Syst Rev. 2015;(6):CD010762.
3. Prins RP, Neilson DR Jr, Bolton RN, et al. Preinduction cervical ripening with sequential use of prostaglandin E2 gel. Am J Obstet Gynecol. 1986;154:1275.
4. Boulvain M, Kelly A, Irion O. Intracervical prostaglandins for induction of labour. Cochrane Database Syst Rev. 2008;(1):CD006971.
5. Beckmann M, Kumar S, Flenady V, Harker E. Prostaglandin vaginal gel induction of labor comparing amniotomy with repeat prostaglandin gel. Am J Obstet Gynecol. 2015;213:859.e1.
6. Lydon-Rochelle M, Holt VL, Easterling TR, Martin DP. Risk of uterine rupture during labor among women with a prior cesarean delivery. N Engl J Med. 2001;345:3.
7. Motaze NV, Mbuagbaw L, Young T. Prostaglandins before caesarean section for preventing neonatal respiratory distress. Cochrane Database Syst Rev. 2013;(11):CD010087.
8. Shetty A, Danielian P, Templeton A. Sublingual misoprostol for the induction of labor at term. Am J Obstet Gynecol. 2002;186:72.
9. Souza AS, Amorim MM, Feitosa FE. Comparison of sublingual versus vaginal misoprostol for the induction of labour: a systematic review. BJOG. 2008;115:1340.
10. Witter FR, Rocco LE, Johnson TR. A randomized trial of prostaglandin E2 in a controlled-release vaginal pessary for cervical ripening at term. Am J Obstet Gynecol. 1992;166:830.
11. Heinemann J, Gillen G, Sanchez-Ramos L, Kaunitz AM. Do mechanical methods of cervical ripening increase infectious morbidity? A systematic review. Am J Obstet Gynecol. 2008;199:177.
12. Pennell CE, Henderson JJ, O'Neill MJ, et al. Induction of labour in nulliparous women with an unfavourable cervix: a randomised controlled trial comparing double and single balloon catheters and PGE2 gel. BJOG. 2009;116:1443.
13. Salim R, Zafran N, Nachum Z, et al. Single-balloon compared with double-balloon catheters for induction of labor: a randomized controlled trial. Obstet Gynecol. 2011;118:79.
14. Levy R, Kanengiser B, Furman B, et al. A randomized trial comparing a 30-mL and an 80-mL Foley catheter balloon for preinduction cervical ripening. Am J Obstet Gynecol. 2004;191:1632.
15. Keirse MJ. Natural prostaglandins for induction of labor and preinduction cervical ripening. Clin Obstet Gynecol. 2006;49:609.
16. Vaknin Z, Kurzweil Y, Sherman D. Foley catheter balloon vs locally applied prostaglandins for cervical ripening and labor induction: a systematic review and metaanalysis. Am J Obstet Gynecol. 2010;203:418.
17. ACOG Committee on Practice Bulletins—Obstetrics. ACOG practice bulletin no. 107: induction of labor. Obstet Gynecol. 2009;114:386.

18. McMaster K, Sanchez-Ramos L, Kaunitz AM. Balancing the efficacy and safety of misoprostol: a meta-analysis comparing 25 versus 50 micrograms of intravaginal misoprostol for the induction of labour. BJOG. 2015;122:468.
19. Hofmeyr GJ, Gülmezoglu AM, Pileggi C. Vaginal misoprostol for cervical ripening and induction of labour. Cochrane Database Syst Rev. 2010;(10):CD000941.
20. Calder AA, Loughney AD, Weir CJ, Barber JW. Induction of labour in nulliparous and multiparous women: a UK, multicentre, open-label study of intravaginal misoprostol in comparison with dinoprostone. BJOG. 2008;115:1279.
21. Tan TC, Yan SY, Chua TM, et al. A randomised controlled trial of low-dose misoprostol and dinoprostone vaginal pessaries for cervical priming. BJOG. 2010;117:1270.
22. Tang J, Kapp N, Dragoman M, de Souza JP. WHO recommendations for misoprostol use for obstetric and gynecologic indications. Int J Gynaecol Obstet. 2013;121:186.
23. Alfirevic Z, Aflaifel N, Weeks A. Oral misoprostol for induction of labour. Cochrane Database Syst Rev. 2014;(6):CD001338.
24. Khan RU, El-Refaey H, Sharma S, et al. Oral, rectal, and vaginal pharmacokinetics of misoprostol. Obstet Gynecol. 2004;103:866.
25. Alfirevic Z, Kelly AJ, Dowswell T. Intravenous oxytocin alone for cervical ripening and induction of labour. Cochrane Database Syst Rev. 2009;(4):CD003246.
26. Calderyro-Barcia R, Sereno JA. In: Calderyro-Barcia R, Heller H, editors. The response of human uterus to oxytocin throughout pregnancy. London: Pergamon Press; 1959.
27. Fuchs AR, Fuchs F, Husslein P, Soloff MS. Oxytocin receptors in the human uterus during pregnancy and parturition. Am J Obstet Gynecol. 1984;150:734.
28. Thornton S, Davison JM, Baylis PH. Plasma oxytocin during the first and second stages of spontaneous human labour. Acta Endocrinol. 1992;126:425.
29. Rydén G, Sjöholm I. The metabolism of oxytocin in pregnant and non-pregnant women. Acta Obstet Gynecol Scand Suppl. 1971;9(Suppl 9):37.
30. Seitchik J, Amico J, Robinson AG, Castillo M. Oxytocin augmentation of dysfunctional labor. IV. Oxytocin pharmacokinetics. Am J Obstet Gynecol. 1984;150:225.
31. Hauth JC, Hankins GD, Gilstrap LC 3rd, et al. Uterine contraction pressures with oxytocin induction/augmentation. Obstet Gynecol. 1986;68:305.
32. Blakemore KJ, Qin NG, Petrie RH, Paine LL. A prospective comparison of hourly and quarter-hourly oxytocin dose increase intervals for the induction of labor at term. Obstet Gynecol. 1990;75:757.
33. O'Driscoll K, Foley M, MacDonald D. Active management of labor as an alternative to cesarean section for dystocia. Obstet Gynecol. 1984;63:485.
34. Thorp JA, Boylan PC, Parisi VM, Heslin EP. Effects of high-dose oxytocin augmentation on umbilical cord blood gas values in primigravid women. Am J Obstet Gynecol. 1988;159:670.
35. Cummiskey KC, Dawood MY. Induction of labor with pulsatile oxytocin. Am J Obstet Gynecol. 1990;163:1868.
36. Tribe RM, Crawshaw SE, Seed P, et al. Pulsatile versus continuous administration of oxytocin for induction and augmentation of labor: two randomized controlled trials. Am J Obstet Gynecol. 2012;206:230.e1.
37. Bricker L, Luckas M. Amniotomy alone for induction of labour. Cochrane Database Syst Rev. 2000;(4):CD002862.
38. Howarth GR, Botha DJ. Amniotomy plus intravenous oxytocin for induction of labour. Cochrane Database Syst Rev. 2001;(3):CD003250.
39. American College of Obstetricians and Gynecologists. ACOG practice bulletin no. 106: intrapartum fetal heart rate monitoring: nomenclature, interpretation, and general management principles. Obstet Gynecol. 2009;114:192.
40. Bakker PC, Kurver PH, Kuik DJ, Van Geijn HP. Elevated uterine activity increases the risk of fetal acidosis at birth. Am J Obstet Gynecol. 2007;196:313.e1.
41. Jonsson M, Nordén-Lindeberg S, Ostlund I, Hanson U. Acidemia at birth, related to obstetric characteristics and to oxytocin use, during the last two hours of labor. Acta Obstet Gynecol Scand. 2008;87:745.
42. Simpson KR, James DC. Effects of oxytocin-induced uterine hyperstimulation during labor on fetal oxygen status and fetal heart rate patterns. Am J Obstet Gynecol. 2008;199:34.e1.
43. Peebles DM, Spencer JA, Edwards AD, et al. Relation between frequency of uterine contractions and human fetal cerebral oxygen saturation studied during labour by near infrared spectroscopy. Br J Obstet Gynaecol. 1994;101:44.
44. Heuser CC, Knight S, Esplin MS, et al. Tachysystole in term labor: incidence, risk factors, outcomes, and effect on fetal heart tracings. Am J Obstet Gynecol. 2013;209:32.e1.
45. Flannelly GM, Turner MJ, Rassmussen MJ, Strong JM. Rupture of the uterus in Dublin: an update. J Obstet Gynaecol. 1993;13:440.
46. Smith JG, Merrill DC. Oxytocin for induction of labor. Clin Obstet Gynecol. 2006;49:594.
47. Wing DA, Ortiz-Omphroy G, Paul RH. A comparison of intermittent vaginal administration of misoprostol with continuous dinoprostone for cervical ripening and labor induction. Am J Obstet Gynecol. 1997;177:612.
48. Wing DA, Miller H, Parker L, et al. Misoprostol vaginal insert for successful labor induction: a randomized controlled trial. Obstet Gynecol. 2011;117:533.
49. Dyer RA, Butwick AJ, Carvalho B. Oxytocin for labour and caesarean delivery: implications for the anaesthesiologist. Curr Opin Anaesthesiol. 2011;24:255.

50. Lilien AA. Oxytocin-induced water intoxication. A report of a maternal death. Obstet Gynecol. 1968;32:171.
51. Bilek W, Dorr P. Water intoxication and grand mal seizure due to oxytocin. Can Med Assoc J. 1970;103:379.
52. Moen V, Brudin L, Rundgren M, Irestedt L. Hyponatremia complicating labour-rare or unrecognised? A prospective observational study. BJOG. 2009;116:552.
53. Whalley PJ, Pritchard JA. Oxytocin and water intoxication. JAMA. 1963;186:601.
54. Feeney JG. Water intoxication and oxytocin. Br Med J (Clin Res Ed). 1982;285:243.
55. Thomas JS, Koh SH, Cooper GM. Haemodynamic effects of oxytocin given as i.v. bolus or infusion on women undergoing caesarean section. Br J Anaesth. 2007;98:116.
56. Svanström MC, Biber B, Hanes M, et al. Signs of myocardial ischaemia after injection of oxytocin: a randomized double-blind comparison of oxytocin and methylergometrine during caesarean section. Br J Anaesth. 2008;100:683.
57. Bhattacharya S, Ghosh S, Ray D, et al. Oxytocin administration during cesarean delivery: randomized controlled trial to compare intravenous bolus with intravenous infusion regimen. J Anaesthesiol Clin Pharmacol. 2013;29:32.
58. In JH, Choi JW, Jung HS, et al. Severe hypotension and water intoxication developed after an accidental oxytocin overdose in a morbidly obese patient undergoing cesarean section -a case report. Korean J Anesthesiol. 2011;60:290.
59. Xenakis EM, Piper JM, Conway DL, Langer O. Induction of labor in the nineties: conquering the unfavorable cervix. Obstet Gynecol. 1997;90:235.
60. Rouse DJ, Owen J, Hauth JC. Criteria for failed labor induction: prospective evaluation of a standardized protocol. Obstet Gynecol. 2000;96:671.
61. Simon CE, Grobman WA. When has an induction failed? Obstet Gynecol. 2005;105:705.
62. Spong CY, Berghella V, Wenstrom KD, et al. Preventing the first cesarean delivery: summary of a joint Eunice Kennedy Shriver National Institute of Child Health and Human Development, Society for Maternal-Fetal Medicine, and American College of Obstetricians and Gynecologists workshop. Obstet Gynecol. 2012;120:1181.

23 Augmentation and Management of Labor

Sudhir R. Shah

23.1 Introduction

Management of labor is the term used for all the procedures done to help the pregnant woman, once she starts labor pains and till she delivers the newborn. Labor can be progressed in its natural way during all the three stages of it without much interference from attending obstetricians and staff members. An average time period from the initiation of labor pains to the delivery of the newborn (I-D interval) ranges from 12 to 18 h in primigravida and 8 to 10 h in multigravida. It has been proved that the longer the duration of this I-D interval, the more are the chances of infection, dehydration, fatigue, chest pain, body pain and the psychological effect on the mother to be. It can cause disturbances in the vital data like irregular and slow foetal heart rates, brain damage and lung infection in the newborn. These can be described as maternal and foetal morbidities.

O'driscoll and Friedman have studied such labors and have concluded that labor should be conducted actively rather than its own natural way. No labor period in primigravida (I-D interval) should be more than 12 h. Close supervision, timely intervention, tender loving care and constant encouragement to the woman in labor should be the KEY factors in managing labor patients. Primary aim should be to deliver the newborn within the time period of 10–12 h in primis.

23.2 Management of Labor

A woman in labor is extremely apprehensive and emotional for her course of labor and its outcome. The hours she passes in labor pains, the mechanical process of labor and delivering the foetus through narrow vaginal canal are most disturbing, distressing and painful.

Tender care, soothing words, soft touch and constant encouragement boost up the spirit and stamina of the pregnant woman. It leads to smooth course of labor by making the uterine contractions more regular, effective and painless. Fear-tension-pain and dystocia make a vicious cycle, which must be broken.

23.3 O'Driscoll's (Professor Emeritus National Maternity Hospital, Dublin) and Others' Views

It is a good idea to be aware of ACTIVE MANAGEMENT OF LABOR, which originated in Dublin at the National Maternity Hospital (NMH).

S. R. Shah (✉)
ObGyn Department, Satnam Hospital, Rajkot, Gujarat, India

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The principal points of active management are as follows:

- A precise 'beginning' of labor.
- The labor not lasting longer than 12 h.
- Augmentation of contractions with oxytocin is given if dilatation does not increase at the precise rate.
- The progress of the labor is charted on a graph called a partogram.

Every clinic should use the normal monogram of labor progress and should develop alert line-alert zone-action line and action zone of their own clinic and patients should be put on it for knowing progress and should listen the alarming bells at the earliest. The aim is that a happy mother delivers a happy and healthy newborn and both of them make obstetricians happier and cheerful.

23.4 Normal Labor: The Basics

The posture of the foetus at term is called as **Attitude,** and it indicates the relation of the foetal parts with each other. The normal attitude is such which corresponds to the inner shape and volume of the uterine cavity. Generally foetus is bent forward with back makes the convexity. The foetal head is so flexed that the chin touches the chest. The thighs are flexed over the abdomen. The arms are usually crossed over.

The **lie** is the relation of the long axis of the foetus to that of the uterus. The lie is usually longitudinal at term. The presenting part is the portion of the foetus at the level of the brim of the pelvis. Normally it is called cephalic **presentation** when the head lies at the brim of the pelvis. **Position** refers to the relation of the occiput, in case of cephalic presentation, to the brim of the pelvis. Most of the time, it is occipito anterior—left or right side. Accordingly it is known as left occipito anterior (LOA) or right occipito anterior (ROA). Left occipito lateral (LOL) is common.

23.5 Onset of Labor

Labor can be defined as series of events by which the mature foetus is expelled out from the mother through cervico-vaginal route. Normal labor conveniently refers to the expulsion of the foetus in cephalic presentation—more commonly in left or right occipito anterior position. The word 'delivery' refers to the actual birth of the baby. For the circumstances responsible for the commencement of the labor pains, there are different views and theories. The common dictum 'when the fruit is ripe—it will fall' applies also. It appears that labor is initiated and maintained not by a single reason but more than many theories are acting at a time to start labor pains. At term progesterone drop, excessive sensitization of myometrium to present oxytocin in blood and prostaglandins effectivity on myometrium are the reasonable theories for the onset of labor. The real fact is uterine contraction and retractions in the last few days of pregnancy do help in engagement of the foetal head in to the brim of the pelvis. The labor commences when these uterine contractions and retractions become more regular—coming at shorter intervals and more intensive and effective.

Labor commences by effacement of the cervix first.

The cervix first becomes shorter in length and then starts dilating.

Labor is divided into three stages:

1. The first stage of labor begins with the first true labor pains and ends with the full dilatation of the cervix.
2. The second stage of labor begins with the full dilatation of the cervix and ends with the birth of the newborn.
3. The third stage of labor begins with the birth of the newborn and ends with the delivery of the placenta.

One hour after the placental expulsion is considered as fourth stage of labor by many schools of thoughts, as chances of post-partum haemorrhage are maximum during this 1 h.

The causes of labor pains may be due to hypoxia of the contracted muscle cells, compression of the nerves in the cervix and lower segment of the uterus and stretching of the cervix due to dilatation.

The interval between the onset of contractions diminishes gradually from 10 min to 2 min in the second stage of the labor. Relaxation of the uterus between the two contractions is necessary for the foetus to get maternal blood and oxygen to survive.

The duration of each contraction ranges from 20 to 40 s. Each contraction has three phases—increment, acme and decrement.

Under the influence of contraction and retraction in labor, the uterus is differentiated in to two portions. The upper segment is actively contracting and thicker. The lower segment is passively dilating and thinner. As labor progresses, the cervix merges into the lower segment of the uterus. In a nutshell, the upper segment contracts, retracts and expels the foetus, while the lower segment and cervix dilate in response to the activity of upper segment. Thus an expanded muscular tube is formed through which the baby can pass. Retraction is the temporary shortening of upper segment which will not go back to its original position after the true contraction of the uterus. This retraction helps the foetus to descent in to the pelvic cavity. The relaxation of the lower segment is just opposite to the retraction of the upper segment.

When upper segment contracts judiciously, the cervix gives the resistance. Because of this cervical resistance, force of the uterine contractions increases to the level that a time comes when it overcomes the resistance of the cervix. The result is the cervix first becomes shorter and then becomes dilated.

In short, labor may be regarded as contest between the FORCE of expulsion and the POWER of resistance provided by the cervix and pelvic muscles. Effacement or taken up of the cervix is shortening of cervical canal from 2 cm to almost nil. The taken up cervix ultimately merges with the lower uterine segment. Condition of external Os of the cervix remains the same. The cervix becomes thin and papery when it is fully effaced, but the cervical os remains little dilated of 1 cm only (tip of the finger). It starts dilating soon after to reach to full dilatation of the cervix—that is, 10 cm dilatation.

23.6 Pelvic Canal

The pelvic bones, ligaments and some important muscles make the bony birth canal—known as passage of the passenger, the foetus. Because of the irregular shape of the pelvic canal and the relatively large dimensions of the foetal head at different levels, it is evident that not all the diameters of the head can pass through the diameters of the pelvis. For that suitable accommodation of different foetal segments is required to the completion of the child birth.

By the end of the first stage of labor the uterine contractions causes two parts of the uterus.Upper—the active and Lower-the passive. Once the cervix starts dilating, presenting part starts descending slowly and steadily in the pelvic canal—in case of primis. During the course of labor, spontaneous rupture of amniotic membrane usually occurs. The total effect of all the above forces and adaptation leads to descent of the foetus in the pelvic canal and ultimately the delivery of the newborn through the vagina by adopting different positions turn by turn.

The cardinal movements of the foetus in the mechanism of labor are:

1. Engagement.
2. Descent.
3. Flexion.
4. Internal rotation.
5. Extension.
6. External rotation.
7. Expulsion.

Though the above movements have been mentioned separately, it usually occurs with one another in the process of delivery.

23.7 The Clinical Course

The sinking of the foetal head into the pelvic cavity reduces the bulk of the uterus and abdominal enlargement of the pregnant mother. This is called 'lightening'. After lightening, the patient is at ease. False labor pains occur during the last few days of confinement**. T**hey are irregular, mostly confined to the lower segment of the uterus and not associated with SHOW or dilatation of the cervix. False pains are relieved by simple analgesics.

Show is the blood-stained discharge from the vagina coming out from effaced cervical canal, and it is the cervical mucus. It mixes with the blood coming out due to separation of the membranes and some dilatation of the cervix. This blood-stained mucus—SHOW—is the first sign of labor pains even before the patient starts feeling pains of true labor contractions of the uterus. The average duration of the first stage of labor in primigravida is about 12–16 h—with marked individual variation.

In the second stage of labor, uterine contractions are long-lasting, usually 50–80 s, and come at 2- to 3-min intervals. During the second stage, the muscles of the abdomen are brought into play. In the second stage of labor, the head descends still further and bulges, and the perineum becomes thin, tense and stretched as the scalp of the foetal head is seen in the gap—which may or may not go back inside in between the contractions. If it remains there,making diameter of at least 3 inch by the scalp of the head, it is called CROWNING of head. This is the right moment of applying episiotomy cut to facilitate the delivery of the newborn. Now comes the attending obstetrician in active role to help deliver the baby without any problems. The next chapter is on normal conduction of labor.

After the successful delivery of the newborn, the uterus will tonically contract and retract. The size will reduce to a greater extent. Because of the disparity of the sizes of the uterine wall and placenta, the placenta will start separating from the uterine wall. There will be a sudden gush of bleeding, and the placenta will deliver with active help of the attending obstetrician. Once the placenta comes out, the uterus contracts to a hard cricket ball. The muscles of the uterus have a tendency to relax in between contraction, especially when she is on oxytocin drip. In such cases, drip should be continued to prevent post-partum haemorrhage.

23.8 Normal Labor: Standard Conduction

The fundamentals in conducting normal labor or delivery in the hospital set-up are very clear, and one must observe that the standards and precautions should always be maintained. Summarizing these facts will enable us to understand what more and what better can be done keeping the originality intact.

All knows about the old age fear of LABOR PAINS in the minds of primigravidas.

Doctor-patient relationship plays a great role in smooth conduction of labor. Unnecessary fear about labor pains, overheard talk from problematic neighbours and oversentimental reactions from friends and family members create fearful image of delivery in the blank mind of a pregnant woman. In a hospital practice of a maternity department, a special impression should be initiated from the first visit of a woman to the hospital and should be maintained seriously by the doctors and all the staff members till she delivers and even after that. Antenatal education and knowledge about labor plays a major role in eliminating wrong ideas about pregnancy and labor. Antenatal exercise should be taught to all pregnant patients. It promotes mind and muscle relaxation and breathing control and develops confidence in themselves.

It is taken for granted that in today's hospital practice,through records of all the visits are kept in a standardized antenatal form. All pathological investigations and ultrasound examinations should be advised at regular intervals. The patient and her husband should be thoroughly instructed for the importance of regular check-ups, filing and keeping all the records and emergency numbers with them. The patient and her husband or near relatives should be taken to the place where she has to be admitted when labor pains start and introduced to the staff members at the registration counter.

They should also take the virtual visit to intensive labor care room and understand the names of common procedures of conduction of labor. If possible short film of labor conduction and child birth should be shown to all pregnant women to eliminate the fears and understanding of the facts.

23.9 Management of the First Stage of Labor

The first stage of labor starts from the first true labor pains and ends with full dilatation of the cervix. The average time period of the first stage of labor is 12–18 h in primigravida. Patients admit to the labor room at the different time of their first stage. It goes from just onset of labor to 3 cm dilatation of the cervix to any point of the first stage of labor. After routine protocol of admission, thorough general and physical examination including pulse, BP, CVS, and RS examination should be done and recorded. Abdominal examination is similar to the one carried out during each antenatal visit.

Abdominal examination will give the idea of:

- Uterine contraction details—intensity and frequency.
- Lie, presentation and position of the foetus—longitudinal/cephalic/vertex/LOA.
- Engagement of the head of the foetus.
- Auscultation with stethoscope or fetoscope or Doppler gives the idea about foetal heart condition and wellbeing. The normal range of FHR is 120–160/min.

23.9.1 Internal Examination

Vaginal examination is very essential on admission and then should be done every two hourly to understand the progress of labor. It gives the following informations:

- Dilatation of the cervix (like 3–5 cm; full dilatation, 10 cm).
- Effacement of the cervix (like 25%, 50%, etc.—it depends on length of the cervix, 1 cm 50% effaced cervix).
- Station of presenting part (0, at the level of the ischial spine, +1 = 1 cm below the ischial spine).
- Presence or absence of amniotic fluid membrane.
- Palpate two ischial spines to determine the mid-cavity of the pelvis.

Generally the foetal head descents with the progress of labor. Vital signs of progressive labor in correct direction are:

- Progressive dilatation of the cervix.
- Progressive effacement of the cervix.
- Progressive descent of the foetal head.

Frequent examination of the patient in the form of maternal pulse-uterine contractions, their intensity and frequency and FHR is very necessary to know the condition of the mother and the foetus.

- The tempo and emotion of the woman in labor must be maintained by the attending staff members and obstetricians.
- Pain is the major factor of first stage of labor and should be dealt with proper medication.
- Liquid diet which should be allowed in the beginning and then juice only and then nil orally should be the diet protocol.

Tender touch, soft-spoken words, emotional support, positive attitude, pleasant surroundings, perfect analgesia, comfortable dress and smiling faces of the helping staff make the so-called long journey of 12 h enjoyable and result oriented for the woman in labor and for the newcomer too.

23.10 Management of Second Stage of Labor

There are certain signs or events which enable the doctor to know that the patient is now in the second and most important stage of labor. Second stage of labor starts with the full dilatation of the cervix, and it ends with the delivery of the newborn. The average duration in primigravida is about 45–70 min.

The events include:

- Full dilatation of the cervix.
- Complete effacement of the cervix.
- Descent and full flexion of the foetal head.
- Repetitive and long-lasting uterine contractions with bearing down from the patient.
- Head on the perineum.
- Crowning of the head.

The doctors help to deliver the newborn by:

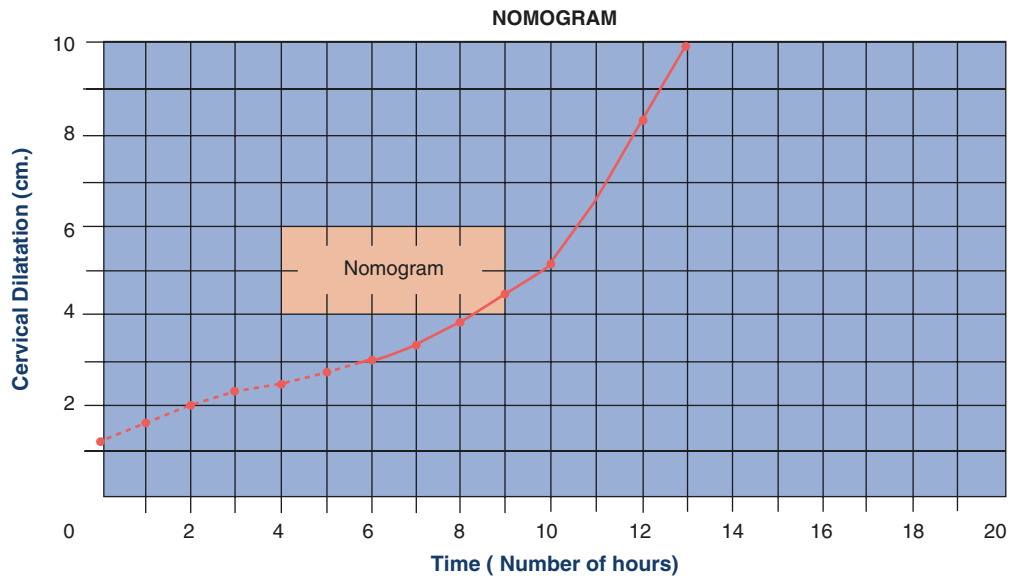
- Good lithotomy position of the mother.
- Giving good-sized episiotomy on left side of the perineum.
- Supporting the perineum with the right hand.
- Controlling the occiput of the foetal head by the thumb and index finger of the left hand to prevent sudden extension of the head at the height of uterine contraction.
- Pushing the head low to deliver the anterior shoulder of the baby first.
- Pulling the head up to deliver the posterior shoulder of the baby.
- Pulling the head towards the doctor conducting the delivery and delivering the body and legs of the baby.
- All aseptic and antiseptic precautions must be taken while delivering a newborn foetus. Sister's trolly should be well prepared. First cry of the newborn is the best vocal song for the mother and for the obstetricians too. The baby should be separated from the mother by cutting the umbilical cord at the right place and in between the two artery forceps. It is better to hand over the baby to the neonatal doctor in charge for the further management of the newborn baby. This ends the vital second stage of labor.

23.11 Management of Third Stage of Labor

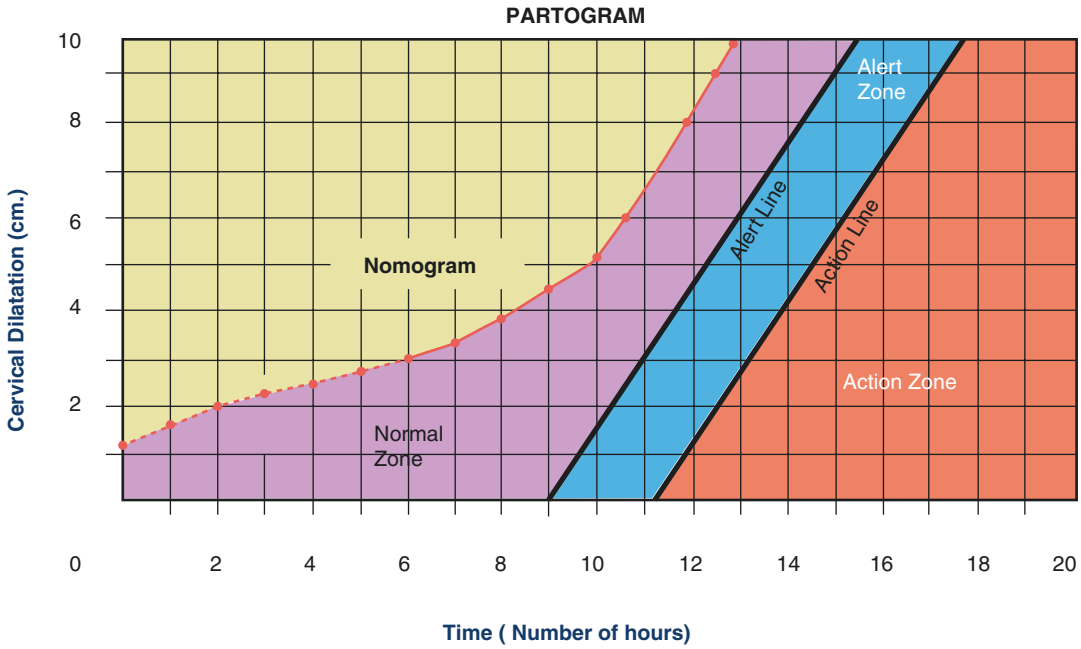
Third stage of labor begins with complete delivery of the newborn and ends with complete delivery of the placenta. The average duration of the third stage of labor is about 15 min and ranges from 10 to 30 min in the primigravida.

23.12 Nomogram

Nomogram is an average standard graphical evaluation between the progressive cervical dilatation in comparison with the time elapsed in numbers of hours. Nomogram differs from primigravidas to multigravidas. It also differs from continent to continent and country to country.



Friedmann (1955) studied the course of normal labor in thousands of primigravidas. He studied two points: the relation of cervical dilatation to the number of hours in the first stage of labor. He made a graphical record keeping these two points in mind and made a curve. He gave the name NOMOGRAM to that curve. Philpott introduced ALERT LINE to this nomogram and named the zone right to the line as ALERT ZONE. Any particular partogram that enters the alert zone means something is alarming, and close watch should be kept on the progress of labor henceforth. O'Driscoll (1970) gave the name ACTIVE MANAGEMENT OF LABOR by suggesting some modification in this nomogram with alert line. He gave the concept of action line on the nomogram, and the zone right to action line was named as action zone. If any patient's partogram enters the action zone, delivery of the baby should be within 2 h to reduce the maternal and foetal morbidity and mortality.



To understand the perfect progress of labor in parturient woman and to diagnose any deviation from the normal progress to prolong one at earliest time, alert line is useful.

Alert line is the parallel line drawn 2 h away to the maximum slope of nomogram on the right side. Zone on the right side of the alert line is called alert zone. The value of alert line is understandable. Whenever the partogram of a particular parturient woman crosses the alert line, it means labor is a little slow and time has come to accelerate the labor. Some steps must be taken to keep the partogram line on the left side of the alert line. No place again to 'doing nothing'.

The next important line and zone are action line and action zone. Any progressive graph in the alert zone that moves towards the action line, touches it, crosses it and enters the action zone in spite of some measures taken to regularize the labor needs active steps. To regulate the progress, more aggressive steps should be necessary with the aim that newborn should be delivered soon. The upper limit is 120 min, to save the life of the baby and to decrease the morbidity of mother and the foetus. This is the real ACTION needed in action zone. This concept was given by O'Driscoll after long study and was further more advocated by S.N. Daftary and colleagues S'Sudhir and

N'Raman of India to bring the modifications in conduction of labor actively in India.

Many obstetricians have followed this concept, and the result published by them was an eye-opener. It clearly concludes that maternal and foetal morbidity and mortality decrease by adopting this method. It also reduces instrumental vaginal delivery and incidence of caesarean section (C. section) rates. To top it all, the patient enjoys her delivery as she is under constant observation and knowing that all surrounding medicos are assembled to help her and her precious child to give the best possible result.

Thus active management of labor is more methodical and scientific approach to the conduction of normal labor with the help of partogram documentation. The aim is to have a happy mother, happy and healthy baby and the happiest doctor. It includes complete management of the patients in labor including her pain relief problems, acceleration of labor by ARM-oxytocin drip, outlet forceps to vacuum extraction and LSCS.

Every maternity ward set-up should make their own labor conduction programme in a methodical way. Complete documentation and record keeping should be done to avoid any medicolegal problems.

23.13 Active Management of Labor

Watchful expectancy in conducting a normal labor in primigravida is like an old wives' tale now. Active management of labor is the talk of the field today. No sooner the primigravida patient is admitted to the intensive labor care room (ILCR), and everyone from the staff nurses to the obstetricians should become ACTIVE.

Every institution should decide their own criteria for taking the patient on active management.

Most acceptable criteria for primigravidas are as follows:

1. Term size (>37 weeks) primigravida.
2. No medical complications present (heart disease, diabetes, etc.).
3. No obstetric complications present (APH, PIH, etc.).
4. Positive willingness of the patient to join this way of conducting labor.
5. Patients should be in true and active labor.
   (a) Cervix more than 3 cm dilated.
   (b) Cervical effacement more than 25%.
   (c) Effective uterine contractions more than 3 in 1 min.
   (d) Cephalic presentation with LOA-LOL-ROA-ROL position.
   (e) Station of the head at least 0 or +1.
   (f) Good if membranes are intact—if not, fluid should be clear.
6. Consent of the patient and one near relative for any active interference in labor at any time—for instrumental delivery like vacuum/forceps or LSCS if needed.

Every above thing should be written clearly in English and in local language for the patient's understanding. They should be explained thoroughly before taking consent.

For regular antenatal patients, this procedure should be done in advance.

A small booklet written in local language about the rules and regulations of the hospital, information regarding conduction of labor, charges, etc. is better and must be given to all registered pregnant patients.

23.13.1 Clinic's Criteria

- Availability of staff—round the clock.
- Availability of neonatologist—on call.
- Availability of one attending obstetrician—round the clock who is trained for this type of management of labor.
- Availability of anesthesiologist—on call.

23.13.2 Programme

1. Proper documentation should be done.
   History—antenatal history—is vital data.
2. Abdominal findings to be written on admission—position and engagement of the head.

FHR should be documented with rate per minute.
3. Vaginal examination done to know the dilatation and effacement of the cervix, station of the foetal head, status of membrane and status of the ischial spines.
4. Instruction of proper diet plan given like only fluids—only water or Nil orally.
5. Enema—if required—given.
6. Patients should be registered into AML sheet with correct name, age and address.

23.13.3 Active Management of First Stage of Labor

Dilatation of the cervix in centimetres is documented at a respective point on a standard nomogram with alert and action line. Standard nomogram is a normal graph on which every new patient's labor progress is documented in graphical manner.

To explain- if the dilatation of cervix on admission of the patient is 3 C M, a point is noted on a corresponding point of a standard nomogram.

This means the onward progress of the labor documented will make a personal curve of that patient. This curve if mimics the nomogram the said patient is progressing normally. Any deviation in the patient's curve can be recognized at the earliest time.

- Intravenous Jelco is inserted and fixed on the forearm. 500 mL 5D or Ringer's lactate plain drip started with 10 drops per minute.
- No analgesia is given if there is no remarkable pain. Otherwise some injectable analgesic drug should be given.
- Patient should be in labor table in ILCR.
- She should be asked to lie down in left lateral position, and deep breathing is suggested in between the two uterine contractions.
- Inform her that she is normal—all here are for the purpose to help her. Everything is going fine. Your progress will be excellent, and the newcomer inside says hello to the mom. These few kind words will make a magic in progress of labor, so always do that.

Repeat per vaginal examination and FHS listening should be done after 2 h of admission time. This examination will provide maximum important information.

- If the progress of labor is good then dilatation of the cervix can be around 5 cm. Contractions are regular and FHS Normal, Effacement and station is progressing.
- Dilatation of the cervix is noted on the patient's partogram. In such condition, graph will just follow the nomogram and hence will be parallel to it.
- If dilatation of the cervix is 4 cm or less, then that line joining the first admission point to this current point will be on the right side of the curve and towards the alert line. This means progress of labor is slow. Patient may still be in normal zone, and if vital data (maternal pulse and foetal heart sound) are within normal limits, nothing active should be done. Contraction may become normal, and labor may progress well in the next examination findings.
- If dilatation is slow after next 2-hour findings, the current point may go left to the alert line, and the patient's partogram may cross the alert line and enter the alert zone in time to come.
- This situation is alarming or alerting to the obstetrician. Membranes can be ruptured, and 5 units of oxytocin are added to the drip of 500 mL of 5D—new bottle. This will accelerate the uterine contractions—if they are primarily hypotonic. If the cervix may not be dilating inspite of effective uterine contractions primiprost tablet one can be put at the mid cervical region.Cerviprime gel also can be used for this purpose.
- Evaluate the progress after 2 h. If the drugs and ARM (artificial rupture of membrane) have acted well, the dilatation and effacement of the cervix will increase, and the partographic curve will again follow the nomogram and will go parallel to alert line and hence will be in alert zone only.
- If the patient complains of PAIN due to strong contractions of uterus after starting the oxytocin drip—the pain should be relieved.

If the dilatation of the cervix is still slow on next examination after ARM and oxytocin drip/gel application, the partogram will go away from nomogram and may travel through the alert zone, touch the action line and enter the ACTION ZONE.

- It needs re-evaluation of foetal position and descent of the foetal head. If the vital datas are normal—the Ox drip should be made faster—15–18 drops per minute or till you palpate strong uterine contractions without disturbing the HR much, and that dose—drops per minute-should be fixed.If the cervical dilatation is more than 7 cm with 75% effacement—good descent and good FHS—wait for 2 h. The cervix may become fully dilated within this time, and patient may deliver in the action zone normally or by assisted vaginal delivery.
- If the cervical dilatation is less than 7 cm dilatation with 50% effacement—good descent and good FHS—wait for 2 h. The cervix may become fully dilated within 3–4 h, but a VERY CLOSE WATCH on all vital data should be kept. If needed Ox drip may be fastened.
- If cervix is NOT progressively dilating in spite of all above measures and the patient is in ACTION ZONE, caesarean section is suggested to get the best result instead of waiting a few hours more in expectation to have vaginal delivery. It is just a wastage of time and will increase the foetal morbidity and not the chances of vaginal delivery.

23.13.4 Active Management of Second Stage of Labor

The second stage of labor begins with the full dilatation of the cervix and ends with the birth of the newborn. The time period is 55 min on an average and ranges from 40 to 80 min.

Active management of second stage works on the principle that there should not be any delay in the second stage of labor due to:

- Undescent of the foetal head.
- Poor and non-effective uterine contractions.
- Outlet resistance, delayed rotation of foetal head and perineal resistance.

All these factors should be managed actively to reduce its wrong effect on the mother and the foetus. Unnecessary pressure of the pelvic muscles and perineum causes brain damage of the foetus. Once the foetal head is on the perineum, the mother should not suffer from excessive pain. It causes maternal distress and increases maternal morbidity.

If the outlet of the pelvis is normal and uterine contractions are good, the head will descend on its own without much bearing down by the mother. Once the crowning of the head is expected and not occurring for any reason, obstetrician's interference is necessary and required at once.

The situation becomes like this:

- Painful contractions of uterine muscles.
- Delayed internal rotation of the foetal head.
- Delayed extension of foetal head.
- Maternal pulse rate increase.
- FHR increase.

Here comes the AML in the picture.

One unit of oxytocin injection is diluted in 9 mL of water and given slowly—taking 1 min—by IV route of the already continuous drip. This will increase the intensity of uterine contractions. Strong tonic contraction will come in 1 min and crowing will occur. This is the time to give episiotomy to remove the perineal resistance. No sooner episiotomy is given, the head will deliver, so it is better to give support to the head from below and upwards. Delivery of the child will be in 2–3 min after the completion of stat Ox injection of 1 unit. This method is also described as MEDICAL FORCEP for vaginal delivery. Tonic contraction of the uterus is of high intensity, and child may suffer of cut down of maternal blood flow. If this situation remains for more than 2 min, there are chances that foetal heart sound drops. So if the delivery is not imminent, then perineal or outlet forceps must be kept ready and applied to deliver the child.

23.13.5 Active Management of Third Stage of Labor

Signs of placental separations are the uterus becoming globular and contracted and being felt above the brim of the pelvis, sudden gush of blood from the vagina and fundus of the uterus going a little upwards as the separated placenta bulges the lower segment.

Delivering the placenta needs real skill and art, no hasty movements and pulling up the cord. Allow the placenta to separate on its own, and watch out for the above signs to appear. Pull the umbilical cord slowly and keep one palm on the fundus. The placenta should be taken in one tray to examine it fully. All attached membranes should also be given a chance to deliver fully or pulled with the artery forceps. Massage the uterus for 5 min to contract it tonically. The tonicity of the uterus will prevent PPH. The uterus has a habit of relaxing in between and especially when the patient is on oxytocin drip. So in such cases, a 10-unit drip should be continued for 1 h after the delivery to prevent PPH. This is also called the fourth stage of labor.

23.14 Conclusion

In conducting labor in primi- or any gravid, active watch is needed once the patient is in true labor. The stress in primigravida is more emotional than physical. Progress of labor should be closely watched and documented in terms of dilatation and effacement of the cervix, regularity of effective uterine contraction and conditions of the foetus and mother. Active interference in terms of ARM-oxytocin drip should be done as and when required. The aim of the obstetrician should be having a healthy mother to have a healthy baby in decided time. The mother and obstetrician should be happy and joyful at the end of the whole event.

Further Reading

Boylan P. RCOG conference on modern management of labour. Lecture given in Birmingham, March 1995.

Flanagan A, Fitzpatrick K. Jubilee Hospital Belfast. N. Ireland.

Goodlin RC. Low risk obstetrics for low-risk mothers. Lancet. 1980;1:1017–9.

Jeffcoate TNA. Prolonged labour. Lancet. 1961;278:61–7.

Keirse MJNC, van Oppen ACC. Preparing the cervix for induction of labour (chapter 61) and Comparison of prostaglandins and oxytocin for inducing labour (Chapter 63). In: Effective care in pregnancy and childbirth. Oxford: Oxford University Press; 1989; see also Keirse, MJNC, A final comment. managing the uterus, the woman or whom?, (roundtable debate: active management part 2) Birth, 1993; 20(3): 150-61; Turnbull, AC, Uterine contractions in normal and abnormal labour, J Ob Gyn Br Empire, 1957; 64: 321-32.

Kierse MJNC. Augmentation of labour (chapter 58). In: Effective care in pregnancy and childbirth. Oxford: Oxford University Press; 1989.

Klein M. Active management of labour: whose agenda? (roundtable debate: active management part 1). Birth. 1993;20(2)

O'Driscoll K, Meagher D. Active management of labour. 2nd ed. London: Balliere Tindall; 1986.

O'Driscoll K, et al. Prevention of prolonged labour. BMJ. 1969;2(655):477–80; see also O'Driscoll, K, Active management of labour, BMJ, 1973; 3(872) 135-7; O'Driscol, K, et al Active management of labour: care of the fetus, BMJ, 1977; 2: 1451-3; O'Driscoll, K, et al, Active management of labour as an alternative to caesarean section for dystocia, Ob Gyn, 1984; 63(4): 485-90.

O'Driscoll K, Meagher D, Boylan P. Active management of labour. third ed. London: Mosby Year Book Europe Ltd; 1993.

Rothman BK. The active management of physicians, (roundtable debate: active management part 2). Birth. 1993;20(3):150–61.

Sha SR, et al. Active management of labour step by step. New Delhi: Jaypee Bros; 1979.

Fetal Surveillance in Labour

24

Praveena Pai and Taswin Kaur

24.1 Introduction

Labour is a culmination of events which starts with fertilization. It is an arduous process in which the fetus is subjected to strong uterine contractions with a potential for hypoxic insult. Surveillance of fetus in labour is hence crucial but remains challenging.

24.2 The Need for Intrapartum Surveillance

Despite advances in antepartum and intrapartum care, the annual number of stillbirths worldwide remains very high—2.7 million third trimester stillbirths, of which 1.3 million occur in labour [1]. Labour surveillance figures in one of the five priority actions are recommended by these authors to 'change the curve' of stillbirths.

More important is the perinatal morbidity in the form of hypoxic ischaemic encephalopathy (HIE) and cerebral palsy (CP) that has been linked to inadequate oxygenation of the fetus during labour. HIE is the short-term neurological dysfunction caused by insufficient fetal oxygenation in labour and cerebral palsy (spastic quadriplegic or dyskinetic type) is the long-term neurological dysfunction associated with it. It is important to note that only 10–20% of cerebral palsy cases are caused by intrapartum hypoxia [2]. The majority are caused by antepartum problems such as infections, metabolic diseases and others. Nevertheless, there is no doubt that labour increases the exposure of the fetus to hypoxic conditions as explained below and fetal surveillance is crucial. The aim of fetal surveillance in labour is early identification of fetuses at risk of hypoxic insult and timely intervention to prevent the same.

24.3 Physiology of Fetal Oxygenation

The fetal oxygen concentration (PO2) is considerably lower than the mother's (40 mmHg in umbilical vein vs. 95 mmHg in maternal artery). However, there is a higher haemoglobin concentration in fetal blood, and the fetal haemoglobin has a higher affinity for oxygen. The fetal oxygen dissociation curve is shifted to the left and is much steeper compared to the maternal curve. This allows fetus to have higher oxygen content and saturation despite low PO2 and release of more oxygen at tissue level [3].

Fetus gets its energy by aerobic metabolism which depends on a constant supply of glucose and oxygen. Any obstacles in the oxygen sup-

P. Pai (✉)
Manipal Fertility, Mangalore, Karnataka, India

Kanachur Institute of Medical Sciences, Mangalore, Karnataka, India

T. Kaur
Chandra Superspeciality Hospital, Anantapur, Andhra Pradesh, India

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ply pathway can lead to fetal hypoxaemia (decreased arterial oxygen concentration) and eventually hypoxia (decreased tissue oxygen concentration). In the absence of oxygen, the fetus tries to produce energy through anaerobic metabolism, but this process is much less efficient and results in production of lactic acid plus accumulation of hydrogen ions in extracellular fluid and within the cells [2]. This eventually leads to cell death.

24.4 Fetal Response to Hypoxia

As the fetus moves from normal oxygenation to asphyxia, various compensatory mechanisms kick in:

(a) Fetal heart rate increases to increase fetal cardiac output.
(b) There is decrease in breathing, movement and tone—To decrease the oxygen consumption.
(c) Redistribution of fetal circulation—Preferential flow to the brain, heart and adrenals and decreased to kidneys, gut, lungs and liver.

It hence follows that any hypoxic injury that causes fetal brain damage will be associated with injury to other organ systems reflected as metabolic acidosis. Based on this, essential criteria have been put forth by various societies to define an acute intrapartum event as sufficient to cause cerebral palsy [3]. All the essential criteria need to be met.

The American College of Obstetricians and Gynecologists Criteria:

- Evidence of a metabolic acidosis in fetal umbilical cord arterial blood obtained at delivery (pH <7.0 and base deficit −12 mmol/L).
- Early onset of severe or moderate neonatal encephalopathy in infants born at 34 or more weeks of gestation.
- Cerebral palsy of the spastic quadriplegic or dyskinetic type.
- Exclusion of other identifiable aetiologies such as trauma, coagulation disorders, infectious conditions or genetic disorders.

24.5 Methods of Intrapartum Fetal Surveillance in Current Usage

In the early twentieth century, auscultation of the fetal heart was an acceptable standard of care. By the 1970s, fetal heart rate tracing by electronic fetal monitoring was thought to be superior to intermittent data through auscultation. Here, we discuss the two most prevalent methods of fetal surveillance—intermittent auscultation (IA) and electronic fetal monitoring (EFM).

24.5.1 Intermittent Auscultation

Fetal heart can be done by various methods—stethoscope, handheld Doppler, intermittent use of an external transducer of the cardiotocography unit or ultrasound machine. IA is deemed to be suitable for low-risk women, provided there is a 1:1 care available to the labouring woman. In this baseline heart rate is assessed by listening and counting the number of beats for 1 min, preferably after a contraction. Maternal pulse should be checked during auscultation to avoid falsely recording the maternal pulse. IA has its advantages as it is less costly, enables a woman to remain mobile (though the same can now be achieved with telemetry in EFM) and requires minimal training. The presence of a healthcare provider continuously by the labouring woman to carry out IA has definite merits. However it has several disadvantages. IA is helpful in assessing the baseline heart rate, rhythm and the presence/absence of accelerations and decelerations. It cannot, however, be used for assessing variability or describing types of decelerations. Subtle changes in the fetal heart rate may be missed. It is difficult to perform in obese women. With rising medicolegal cases involving women in labour, IA provides no objective evidence about the events as there are no paper tracings or recordings available.

24.5.2 Electronic Fetal Monitoring (EFM)

The most common method of intrapartum fetal surveillance worldwide is the use of EFM. Although there are two types of EFM, internal and external, EFM is often synonymous with external fetal monitoring. Changes in Fetal heart rate (FHR) are recorded by an ultrasound transducer attached to the maternal abdomen. Transducers are connected to a CTG machine where it records the fetal heart rate changes and their relationship to the uterine contractions. Continuous EFM during labour has been associated with reduction in neonatal seizures but with no significant difference in cerebral palsy, infant mortality or other standard measures of neonatal wellbeing [4]. In fact a higher incidence of caesarean sections and instrumental deliveries was noted in the EFM group. Consequently, it is strongly recommended that EFM should be resorted to only in high-risk women. These could be antenatal factors such as hypertension, diabetes, fetal indications such as fetal growth restriction or intrapartum indication like induced labour to name a few [3].

24.5.3 Method of EFM

This method uses a cardiotocograph (CTG) to record fetal heart rate (FHR) and hence assess fetal wellbeing. In this, an ultrasound transducer is placed on maternal abdomen after localizing the FHR and simultaneously noting the maternal pulse rate (to avoid false FHR interpretation). Doppler shifts are detected and sent to the computer system that interprets the impulses. Uterine contractions are monitored using a tocodynamometer attached to the uterine fundus which evaluates the increased myometrial tension measured through the abdominal wall. This method provides accurate information on the frequency and the average duration of contractions; but it cannot comment on the intensity or strength of contractions.

24.6 Technical Aspects

24.6.1 Maternal Position During Monitoring

Before delving into the details of CTG, it is important to pay attention to a few technical aspects. Supine position of the mother is known to affect placental perfusion due to the aorto-caval compression, and hence monitoring should be done in sitting, semi-recumbent or left lateral position. Extra care should be taken in women with epidural analgesia while giving top-ups. Those women who are interested in remaining ambulant should be given the option of telemetry, where available.

24.6.2 Paper Speed

The horizontal speed for CTG paper in most countries is 1 cm/min. However some countries such as North America and Japan use 3 cm/min, and a few others such as the Netherlands use 2 cm/min [5]. The healthcare providers need to be familiar with their paper speed to avoid misinterpretation of the CTG trace. A not so uncommon error is when paper speed is erroneously set at 3 cm/min. Here the variability can appear much reduced, to those used to the 1 cm/min setting and unnecessary interventions can occur.

24.6.3 Internal FHR Monitoring

External FHR monitoring uses Doppler ultrasound transducer to detect the movement of cardiac structures. It is reasonably accurate in most situations but is more prone to signal losses particularly in the obese, signal artefacts such ‘double counting’ of FHR during decelerations, especially second stage and inadvertent counting of maternal heart rate. In such situations internal FHR monitoring should be resorted to. Internal FHR monitoring is also advisable where fetal cardiac arrhythmias are suspected. In internal FHR monitoring, a disposable fetal electrode is clipped

on to the presenting part (head or breech). This electrode measures the ventricular depolarization cycles by evaluating the time intervals between successive R waves. It is more accurate but more expensive. The membranes should have ruptured before the electrode can be applied. Moreover it is contraindicated in situations with increased risk of transmissible infections such as active herpes lesions; seropositive hepatitis B, C, D and E; or human immunodeficiency virus (HIV). It is also contraindicated where the fetus is likely to be suffering from blood disorders and is relatively contraindicated in preterm infants <32 weeks.

24.6.4 Monitoring of Maternal Heart Rate

Simultaneous documentation of the maternal pulse is a good practice every time the CTG is assessed by a health practitioner. This is inbuilt in some modern-day machines but, where not available, should be done manually. This is particularly essential when the fetus has a low normal baseline (around 100 beats per minute) or when there is maternal tachycardia or in second stage where CTG interpretation becomes difficult (e.g. where fetal accelerations are seen to coincide with contractions).

24.6.5 Monitoring of Twins or Higher Order Gestations

Dual-channel monitors allow simultaneous monitoring of both FHRs in twins. Machines are also available with three channels. If there is difficulty, the presenting twin can be monitored by internal FHR monitoring provided there are no contraindications as highlighted above. Some machines have an inbuilt ability of increasing or decreasing one of the FHRs by 20 beats so as to ensure easy interpretation of the CTG.

24.6.6 Storage of CTG Traces

CTG traces form an important part of the patient records and are important medicolegal documents. At the start of a CTG, the paper speed should be checked. Patient name and hospital number should be documented. Every time a CTG is assessed, date and time, any interventions (e.g. per vaginal examination, epidural top-up), overall impression of the CTG (explained below) and signature and name of the assessor should be documented. These should be stored carefully and where available should be archived in digital files.

24.7 Components of CTG

It is well known that CTG has a 60% false-positive rate and wide interobserver variability [6]. CTG analysis involves evaluation of five basic features—baseline, variability, accelerations, decelerations and contractions.

24.7.1 Baseline

This is the mean FHR after excluding the accelerations and decelerations analysed over a period of 10 min and expressed as beats per minute (bpm) (Fig. 24.1). According to FIGO 2015 [6], the normal baseline varies between 110 and 160 bpm, but according to NICE 2014 [7] guideline, the normal baseline varies between 100 and 160 bpm. It reflects maturity of the sympathetic and parasympathetic nervous systems, the former responsible for an increase and the latter responsible for a decrease in the baseline. Hence it is not unusual to see a higher baseline in preterm fetuses as their parasympathetic system is less well developed and to find a baseline of 90–110 in post-term fetuses. A rise in baseline above 160 bpm for >10 min is called *tachycardia*. This may be due to maternal pyrexia (infection), dehydration, hyperthyroidism, initial stages of non-acute fetal hypoxia, administration of drugs (B2 agonists such as salbutamol, terbutaline, ritodrine; parasympathetic blockers such as atropine) or fetal cardiac arrhythmias. A drop in baseline of below 100 bpm for >10 min is known as *bradycardia*. This may be caused by a maternal hypotension, hypothermia, drugs such as B2 blockers,

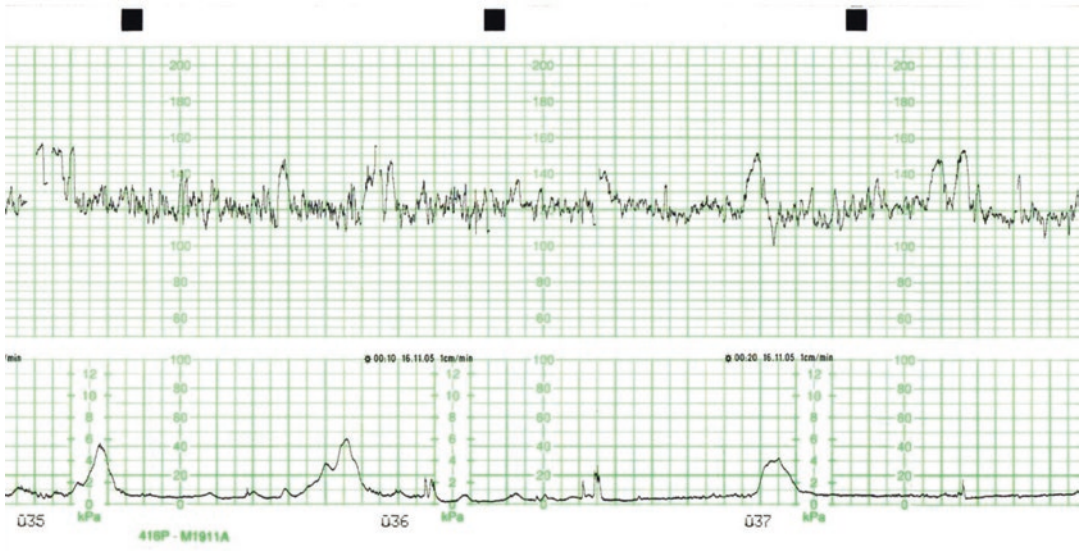

Fig. 24.1 CTG showing baseline of 130 bpm, variability of>10, accelerations, no decelerations, contractions 1 in 10 min

fetal heart block, acute hypoxia or late sign of non-acute asphyxia.

24.7.2 Variability

It is the bandwidth variation of baseline that is noted after excluding the accelerations and decelerations over a period of 1 min. It is maintained by the interplay between the sympathetic and parasympathetic nervous systems. The normal range is 5–25 beats. It is classified as *reduced* when <5 beats and *salutatory* if >25 beats. Normal variability is an indicator of a robust autonomic system. Reduced variability may signal hypoxia especially when associated with decelerations (Fig. 24.2). It may also be seen due to drugs such as CNS (central nervous system) depressants or alternating with normal variability during fetal sleep (known as *cycling pattern*). *Saltatory pattern* is usually seen with rapidly evolving hypoxia during active maternal pushing, with the use of oxytocin, use of ephedrine or even cervical examination. Though poorly understood, it is believed to reflect the attempt of autonomic system to stabilize the baseline in the face of rapidly evolving hypoxia and should be taken seriously in the presence of decelerations [6].

24.7.3 Accelerations

These are transient increases in baseline of >15 beats lasting for 15 s or more. They occur in response to fetal movements and thus reflect a healthy somatic nervous system. Presence of two or more accelerations in a 20 min trace is a reassuring feature. Their absence may signal fetal sleep, effect of drugs such as pethidine or more sinister causes like infection or intrauterine brain haemorrhage. It is important to note that accelerations do not coincide with uterine contractions and if this occurrence is noted, it may be due to erroneous monitoring of maternal heart instead of FHR.

24.7.4 Decelerations

A transient drop in FHR to >15 bpm below baseline for 15 s or more is defined as a deceleration. These are described as early, variable and late. *Early* decelerations (Fig. 24.3) occur due to compression of the head by uterine contractions and are hence seen in late first or second stage. Altered cerebral blood flow elicits a vagal slowing of the FHR, and these are usually benign. They start with the uterine contractions, nadir

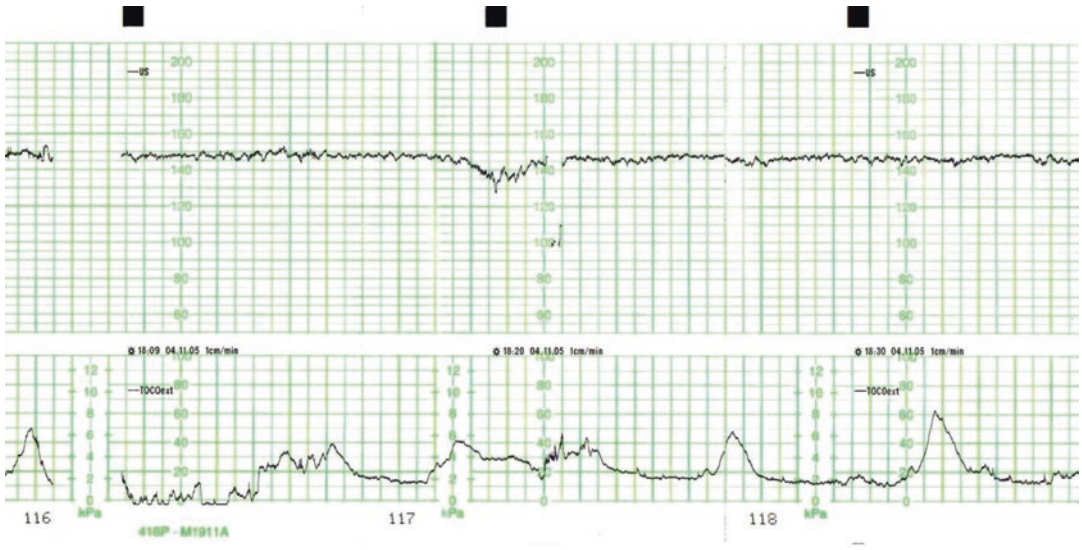

Fig. 24.2 Reduced variability (3–5 bpm)

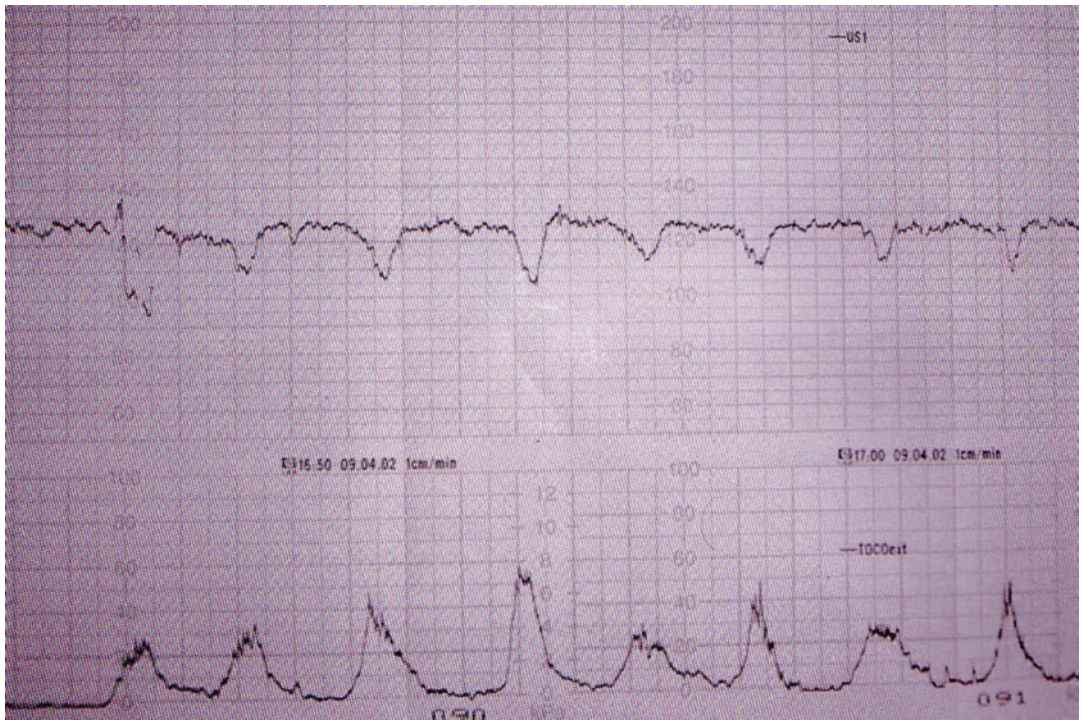

Fig. 24.3 Early decelerations

coincides with the peak of contraction, and they reach the baseline by the end of contraction. *Late decelerations* (Fig. 24.4) begin a bit later than the contractions, reach a nadir later than the peak of contraction and return to baseline after the contraction has worn off. These are mediated by stimulation of central and peripheral chemoreceptors by the hypoxia and hypercarbia. *Variable*

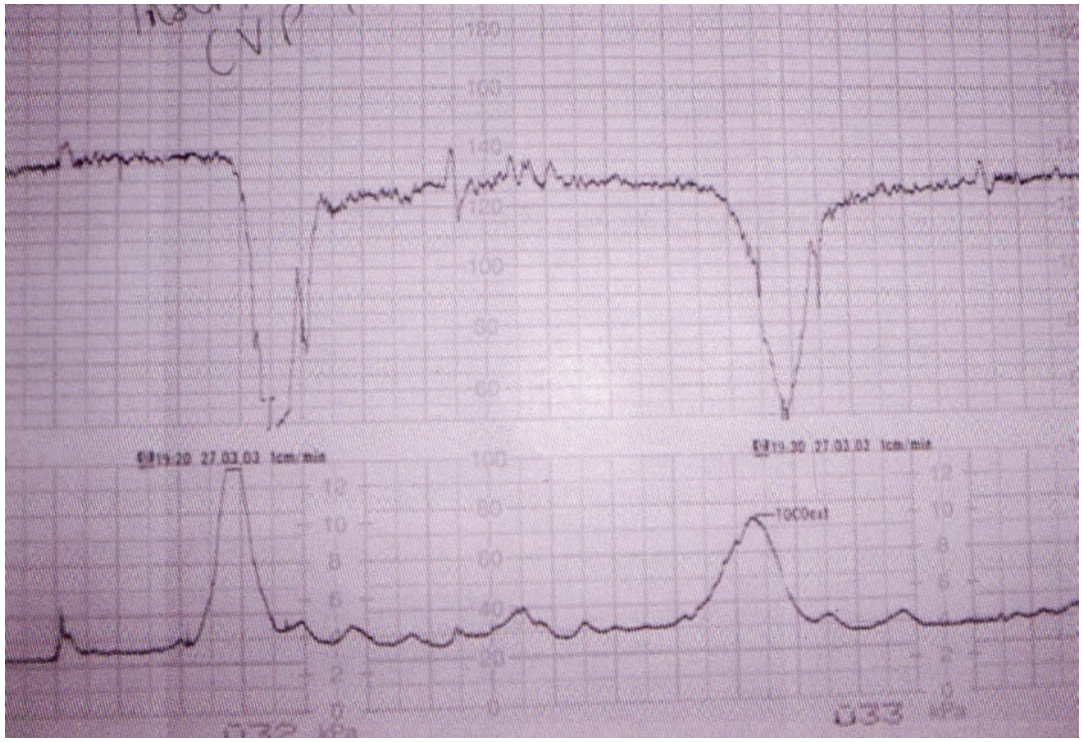

Fig. 24.4 Late decelerations

decelerations (Fig. 24.5) vary in size, shape and their relation with the uterine contractions. These are the commonest type of decelerations seen in labour and are due to umbilical cord compression. These are of two types. *Typical variable decelerations* are those which have an initial rise from baseline (shouldering), drop of <60 bpm followed by a rapid rise above baseline (second shouldering) and final drop to baseline—altogether not lasting >60 s. The initial shouldering is due to the compression of the thin-walled umbilical vein, resulting in fetal hypovolemia and compensatory increase in FHR. This is followed by the compression of umbilical artery too causing systemic hypertension and a protective drop in FHR to avoid fetal stroke, and finally there is recovery to baseline as the compression ceases [6]. These are baroreceptor mediated. *Atypical* ones do not show shouldering, the drop in baseline is >60 bpm, and the final recovery to baseline may be delayed. These are mediated by a combination of baro- and chemoreceptors. *Prolonged deceleration* is one which lasts for >3 min.

Sinusoidal pattern is an FHR pattern which is regular, smooth undulation resembling a sine wave, with an amplitude of 5–15 bpm, frequency of 3–5 cycles/min, is associated with absence of accelerations and lasts longer than 30 min [5]. It is associated with fetal anaemia that may result due to Rh isoimmunisation, twin-twin transfusion, ruptured vasa praevia, etc. *Pseudo-sinusoidal* pattern has a more benign aetiology such as fetal sucking or analgesic administration to mother. It has a jagged, saw-toothed appearance rather than a smooth sine wave appearance and rarely lasts longer than 30 min.

Contractions are necessary for progress of labour. *Tachysystole* is a condition where the frequency of contractions exceeds 5 in 10 min. If it persists without amelioration of cause, FHR starts showing decelerations, and this condition is known as *hyperstimulation syndrome*.

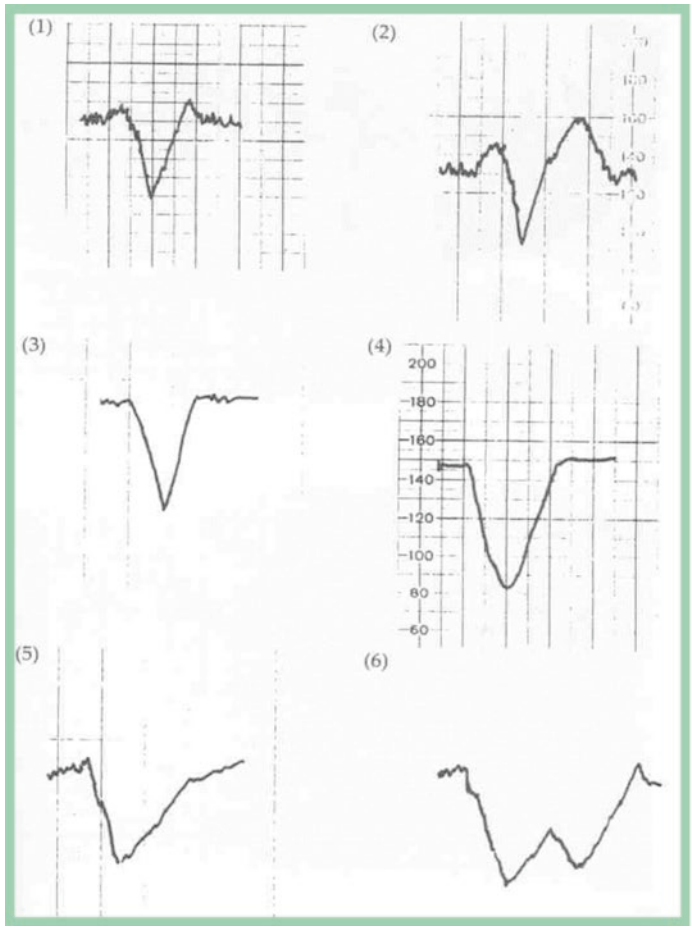

Fig. 24.5 Types of variable decelerations. (**a**) Normal shouldering. (**b**) Overshoot shouldering. (**c**) Loss of shouldering. (**d**) Smoothening of trough. (**e**) Late recovery. (**f**) Biphasic deceleration

24.7.5 CTG Analysis

Various authorities have suggested slightly different methods of evaluating CTG (FIGO, NICE, ACOG), two of which are illustrated below. It is imperative that clinicians looking after women in labour understand the fetal physiology and do not rely on 'pattern recognition' alone to interpret CTGs [6]. A popular acronym to assess CTGs completely is '*DR C BRAVADO*', which means—**d**efine **r**isk, assess **c**ontractions, **b**aseline **r**ate, **a**ccelerations, **va**riability, **d**ecelerations and **o**verall analysis.

Adapted from NICE 2014 guidelines: intrapartum care for healthy women and babies [7]

	Features		
Description	Baseline (beats per minute)	Variability (beats per minute)	Decelerations
Normal/ reassuring	100–160	>5	None or early

Description	Features		
	Baseline (beats per minute)	Variability (beats per minute)	Decelerations
Non-reassuring	161–180	<5 for 30–90 min	*Variable decelerations* – drop from baseline by 60 bpm or less and taking 60 s or less to recover, present for >90 min, occurring with over 50% of contractions *Or* – drop from baseline by more than 60 bpm or taking more than 60 s to recover, present for >30 min, occurring with over 50% of contractions *Or* *Late decelerations*—Present for up to 30 min occurring with over 50% of contractions
Abnormal	>180 or <100	<5 for more than 90 min	Non-reassuring variable decelerations still observed 30 min after starting conservative measures occurring with >50% of contractions *Or* Late decelerations—Present for >30 min, do not improve with conservative measures occurring with >50% of contractions *Or* Bradycardia or a single prolonged deceleration lasting ≥3 min

FIGO classification—with permission from Diogo Ayres-de-Campos [5]
Cardiotocography classification criteria, interpretation and the recommended management

	Normal	Suspicious (one non-reassuring feature)	Pathological (one abnormal feature or two non-reassuring features)
Baseline	110–160 bpm	Lacking at least one character of normality but with no pathological features	<100 bpm
Variability	5–25 bpm		– Reduced variability – Increased variability – Sinusoidal pattern
Decelerations	No repetitive decelerations		Repetitive late or prolonged decelerations >30 min or 20 min if reduced variability, or one prolonged deceleration with >5 min
Interpretation	Fetus with no hypoxia/acidosis	Fetus with low probability of having hypoxia/acidosis	Fetus with high probability of having hypoxia/acidosis
Management	**No intervention necessary to improve fetal oxygenation state**	**Action to correct reversible causes if identified, close monitoring or additional methods to evaluate fetal oxygenation**	**Immediate action to correct reversible causes, additional methods to evaluate fetal oxygenation or if this is not possible to expedite delivery. In acute situations (cord prolapse, uterine rupture, placental abruption) immediate delivery should be done**

24.7.6 Adjuncts to CTG

As mentioned earlier, CTG shows a high inter- and intraobserver variability. Large but very early studies showed a limited benefit of CTG when applied to all women—50% reduction in neonatal seizures and no difference in perinatal mortality and cerebral palsy cases [4]. On the contrary there was a 63% increase in caesarean and 15% increase in instrumental deliveries. To reduce the incidence of false positives and unnecessary medical interventions, various adjunctive tests have been recommended. These include *fetal blood sampling* for pH and lactate levels, *fetal*

stimulation, *fetal pulse oximetry* and *ST wave form analysis*. Fetal pulse oximetry went out of use when the commercialization of the electrodes was stopped. Fetal scalp stimulation digitally or vibroacoustic stimulation on mothers' abdomen was essentially used to distinguish the reduced variability due to fetal sleep from that due to hypoxia. Its role in other situations is limited.

24.7.7 Fetal Blood Sampling (FBS)

Broadly speaking, FBS is used in cases of suspicious CTG or pathological CTG, unless of course the pathological CTG is secondary to an acute hypoxic event such as cord prolapse, in which event, rapid delivery is essential. Contraindications to internal FHR monitoring apply to FBS too. Ruptured membranes and cervical dilatation of at least 3 cm are prerequisites. The same sample can be used for both pH and lactate analysis. The failure to obtain a result is lower with lactate estimation compared to pH as the amount required for lactate estimation is only 5 μL as against 50 μL for pH [8]. The interpretation and management are explained in the table below.

FIGO classification—with permission from Diogo Ayres-de-Campos [5]

Classification of fetal blood sample results and management			
pH	Lactate (mmol/L)	Interpretation	Management
≥7.25	≤4.1	Normal	– Monitor CTG – Repeat FBS within 1 h if CTG abnormality persists
7.21–7.24	4.2–4.8	Borderline	– Improve fetal oxygenation – Repeat FBS in 20–30 min – Consider delivery if persisting borderline report
≤7.20	≥4.9	Abnormal	– Rapid delivery

FBS use is mainly seen in Britain and parts of Europe. It has not become universally popular as it takes a time (median 18 min from decision to result) and often needs to be repeated due to the evolving hypoxia [9]. It cannot be done in early labour. There is a small risk of fetal bleed and infection. Cochrane review in 2013 found a small decrease in caesarean rate when CTG+ FBS was used though the instrumental deliveries increased [4]. A systematic review of studies analysing CTG + FBS effect in labour concluded that it does provide additional information on fetal wellbeing and is associated with decreased operative deliveries [10]. A high-quality RCT in this regard is still lacking.

24.7.8 Combined CTG and Electrocardiographic Monitoring

ST segment changes precede failing cardiovascular changes, and this forms the basis for using ST segment changes in combination with CTG for fetal surveillance in labour. This is also known as STAN (ST Analysis, Neoventa Sweden). A fetal electrode is used to measure both the CTG and ST signals. Information is obtained about the T wave in relation to QRS complex and shape of ST segment. On detecting relevant changes, the monitor gives warnings in the form of 'ST events' which need to be interpreted according to specific guidelines taking into account the CTG changes too [11]. A normal CTG or a normal FBS needs to be ensured before commencing STAN. This method has been in use since 2000 and has been the subject of six meta-analyses. The conclusion is that STAN reduces operative deliveries and FBS, but effect on neonatal metabolic acidosis is still debatable [12].

24.7.9 Computer Analysis of CTG

This is a recent attempt at reducing the intra- and interobserver variations seen in CTG interpretation. Here, there is a computer analysis of the CTG traces, and an alert is issued (visual and audible) so that the clinician can take remedial steps. A few studies have analysed its ability to predict neonatal academia and found promising results [13, 14].

24.8 Conclusion

Labour is a taxing period for the fetus, and healthcare workers have to look out for signs of evolving hypoxia. Good knowledge of the pathophysiology of fetal oxygenation, thorough understanding of CTG and its limitations and application of adjunctive tests where feasible are needed to ensure satisfactory outcomes and prevent operative interventions.

Learning Points

- At the beginning of labour, women should be categorized into low and high risk based on their antenatal history. Intermittent auscultation may be sufficient for the former, but situation can change during labour.
- CTG interpretation should follow DR C BRAVADO—For complete assessment of situation.
- During second stage, the progression of hypoxia is even faster—Urgent action to relieve the situation or expediting delivery is essential.
- Regularly updating oneself in CTG interpretation is necessary for all professionals taking care of labouring women.

References

1. de Bernis L, Kinney M, Stones W, ten Hoope-Bender P, Vivio D, Leisher S, et al. Stillbirths: ending preventable deaths by 2030. Lancet. 2016;387(10019):703–16.
2. Ayres-de-Campos D, Arulkumaran S, FIGO Intrapartum Fetal Monitoring Expert Consensus Panel. Physiology of fetal oxygenation and the main goal of intrapartum fetal monitoring. Int J Gynaecol Obstet. 2015;131(1):5–8.
3. Fetal Health Surveillance in Labour. Chapter 11. 4th Edition of The ALARM International Program.
4. Alfirevic Z, Devane D, Gyte GM. Continuous cardiotocography (CTG) as a form of electronic fetal monitoring (EFM) for fetal assessment during labour. Cochrane Database Syst Rev. 2013;(5):CD006066.
5. Ayres-de-Campos D, Spong CY, Chandraharan E, FIGO Intrapartum Fetal Monitoring Expert Consensus Panel. FIGO consensus guidelines on intrapartum fetal monitoring: cardiotocography. Int J Gynaecol Obstet. 2015;131(1):13–24.
6. Pinas A, Chandraharan E. Continuous cardiotocography during labour: analysis, classification and management. Best Pract Res Clin Obstet Gynaecol. 2016;30:33–47.
7. National Institute of Clinical Excellence. Intrapartum care: care of healthy women and their babies during childbirth. NICE clinical guideline CG190. December 2014.
8. Ramanah R, Martin A, Clement MC, Maillet R, Riethmuller D. Fetal scalp lactate microsampling for non-reassuring fetal status during labor: a prospective observational study. Fetal Diagn Ther. 2010;27(1):14–9.
9. Tuffnell D, Haw WL, Wilkinson K. How long does a fetal scalp blood sample take? BJOG. 2006;113(3):332–4.
10. Jorgensen JS, Weber T. Fetal scalp blood sampling in labor–a review. Acta Obstet Gynecol Scand. 2014;93(6):548–55.
11. Visser GH, Ayres-de-Campos D, FIGO Intrapartum Fetal Monitoring Expert Consensus Panel. FIGO consensus guidelines on intrapartum fetal monitoring: adjunctive technologies. Int J Gynaecol Obstet. 2015;131(1):25–9.
12. Amer-Wahlin I, Kwee A. Combined cardiotocographic and ST event analysis: a review. Best Pract Res Clin Obstet Gynaecol. 2016;30:48–61.
13. Costa A, Ayres-de-Campos D, Costa F, Santos C, Bernardes J. Prediction of neonatal acidemia by computer analysis of fetal heart rate and ST event signals. Am J Obstet Gynecol. 2009;201(5):464.e1–6.
14. Schiermeier S, Pildner Von Steinburg S, Thieme A, Reinhard J, Daumer M, Scholz M, et al. Sensitivity and specificity of intrapartum computerised FIGO criteria for cardiotocography and fetal scalp pH during labour: multicentre, observational study. BJOG. 2008;115(12):1557–63.

Partograph

25

Geetika Gupta Syal

25.1 Introduction

In developing countries, prolonged and obstructed labour is one of the leading causes of maternal and neonatal morbidity and mortality. The causes may lie in the passage, the passenger or the power. It is therefore important not only to identify the cause of prolonged labour but to take timely steps to prevent and manage prolonged labour.

Partograph or partogram is a simple graphical record showing progress of labour and maternal and fetal conditions during labour. It helps to identify any delay in the progress of labour and signs of maternal or fetal distress easily so that early interventions can be taken.

25.1.1 History

Partograph was originally designed by Friedman in 1954. It was called the Friedman's curve (Fig. 25.1) [1]. It had a sigmoid shape and consisted of latent, acceleration and deceleration phases. Later Philpott and Castle improvised it by including the alert and action lines in it [2–4]. Since then various partographs have been introduced to monitor the progress of labour. The WHO partograph which was modified in 2000 is currently used in most of the centres.

25.2 Components of Partograph (WHO-Modified Partograph)

The modified WHO partograph (WHO removed the latent phase in the now recommended "modified partograph") begins at the active phase of labour where the dilatation of the cervix is 4 cm or more (Figs. 25.2 and 25.3).

At the top part of the partograph, there is an identification part where the name of the parturient, her gravid and para status, the hospital registration number, the date and time of admission of the labouring woman and time of rupture of membranes are to be recorded. The three main components of the graph are:

1. Fetal record.
2. Record of progress of labour.
3. Maternal record.

1. **Fetal record**

   This is the part just below the patient identification data. It consists of fetal heart rate record, condition of liquor and degree of moulding, one below the other.

   (a) Fetal heart rate (FHR): the range of the graph is from 80 beats per minute to 200 beats per minute. Each square signifies half an hour. The FHR is recorded every

G. G. Syal (✉)
Obstetrics & Gynaecology, Kamla Nehru State Hospital for Mother & Child, Indira Gandhi Medical College, Shimla, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_25

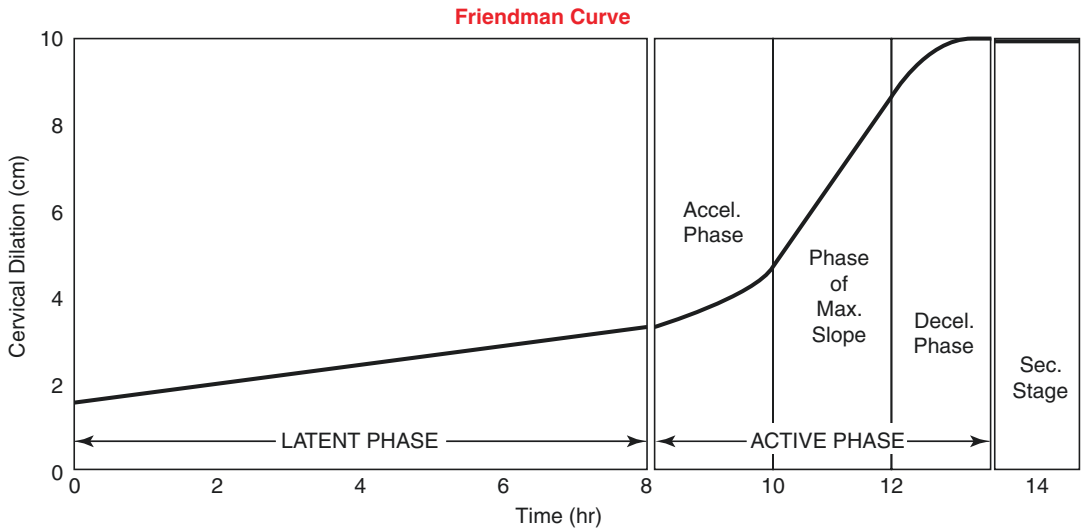

Fig. 25.1 Friedman curve

15 min to half an hour in the first stage of labour and every 5 min in the second phase. It should be auscultated with stethoscope for full 1 min after the contraction, or continuous electronic fetal heart rate monitoring can be used. The normal FHR is between 110 and 160 beats per minute [5]. If abnormalities are noted, immediate action is to be taken.

(b) Liquor: the colour of the liquor is to be noted in the squares. It can give an indication of fetal distress. If the amniotic membrane is intact, it is indicated with the letter "I". If the membranes have ruptured, then the letters "A", "C", "B" and "M" are used to denote absent liquor, clear liquor, blood-stained liquor and meconium-stained liquor, respectively.

(c) Moulding: this is an important indicator of the adequacy of the passage (pelvis) for the passenger (fetus). The degrees of moulding are to be noted in the squares after every vaginal examination.

- 0 The sutures can be easily felt as the skull bones are separated.
- 1+ The bones are just touching each other.
- 2+ The bones are overlapping but reducible, i.e. can be easily separated with pressure of the finger.
- 3+ The bones are overlapping and irreducible.

2. **Progress of labour**

This is the middle portion of the partograph and the most important. It is also called cervicograph. In this portion we plot the dilatation of the cervix and descent of fetal head. Below the cervicograph is the section to record uterine contractions.

(a) Cervical dilatation: as described by Friedman, cervical dilatation follows a sigmoid curve. The active phase has acceleration phase, phase of maximum slope and phase of deceleration.

The y-axis of cervicograph has markings from 0 to 10 and represents cervical dilatation in cm. The x-axis shows time in hours. It has markings from 0 to 24. However, in the modified WHO partograph, there is no latent phase. The dilatation of the cervix is marked with "x" after estimating the same by vaginal examination.

There are two lines printed on the cervicograph which are "alert" and "action" line. The alert line is plotted at 4 cm dilatation which corresponds to the active phase of labour. It is plotted up to 10 cm of dilatation; after 4 cm, the cervix should be expected to dilate at the rate of 1 cm per hour. Four hours beyond the alert line,

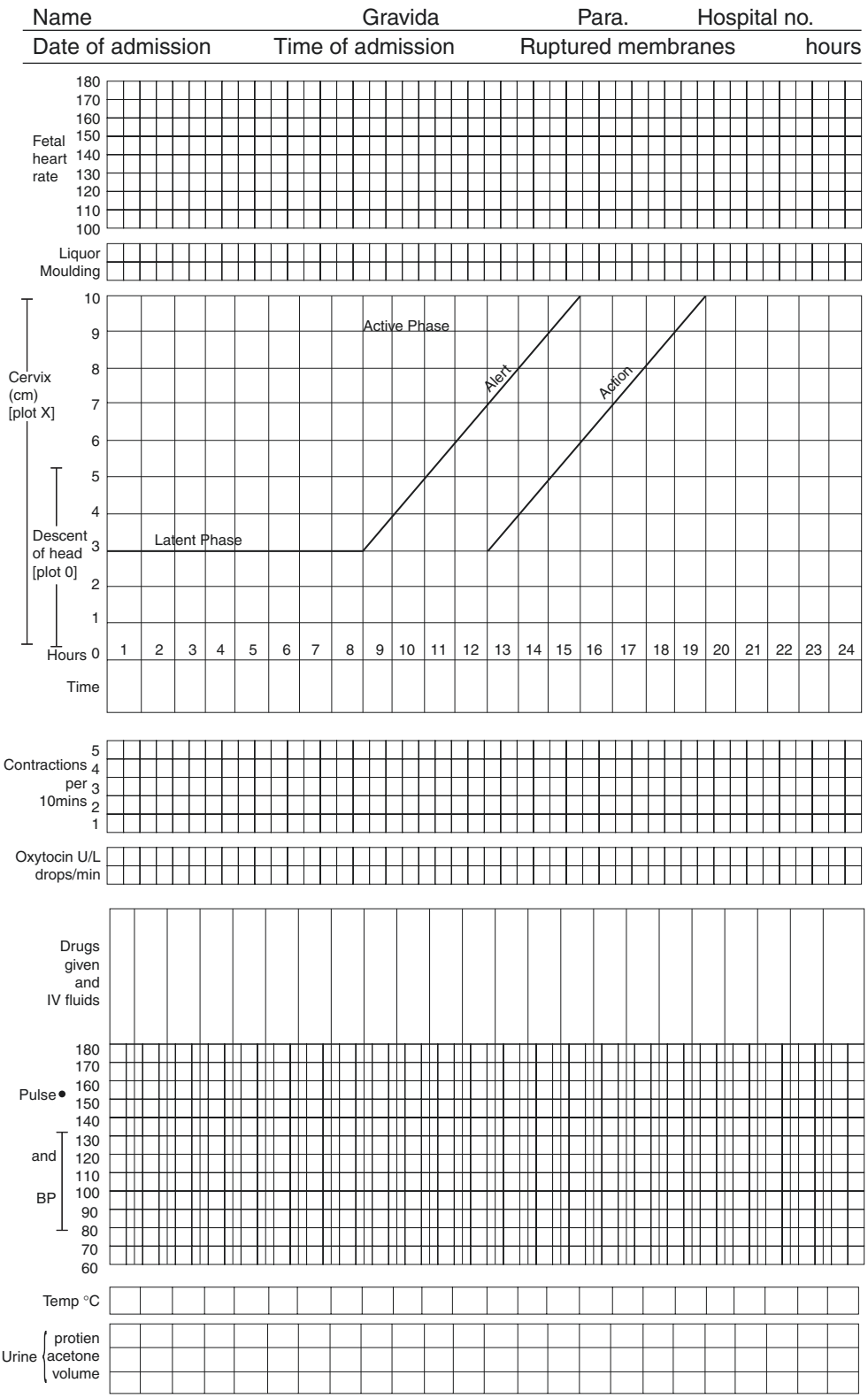

Fig. 25.2 WHO partograph

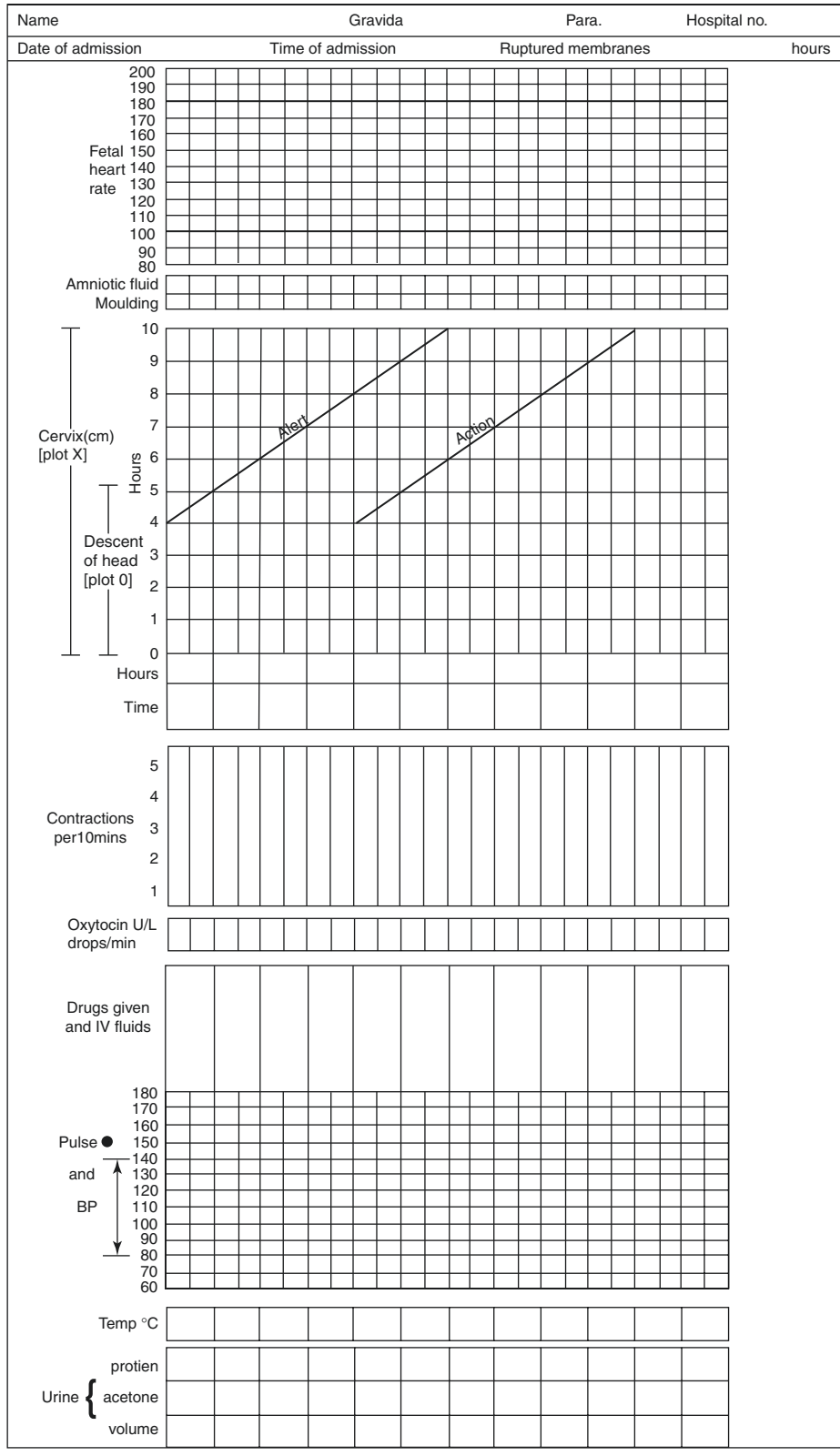

Fig. 25.3 WHO-modified partograph

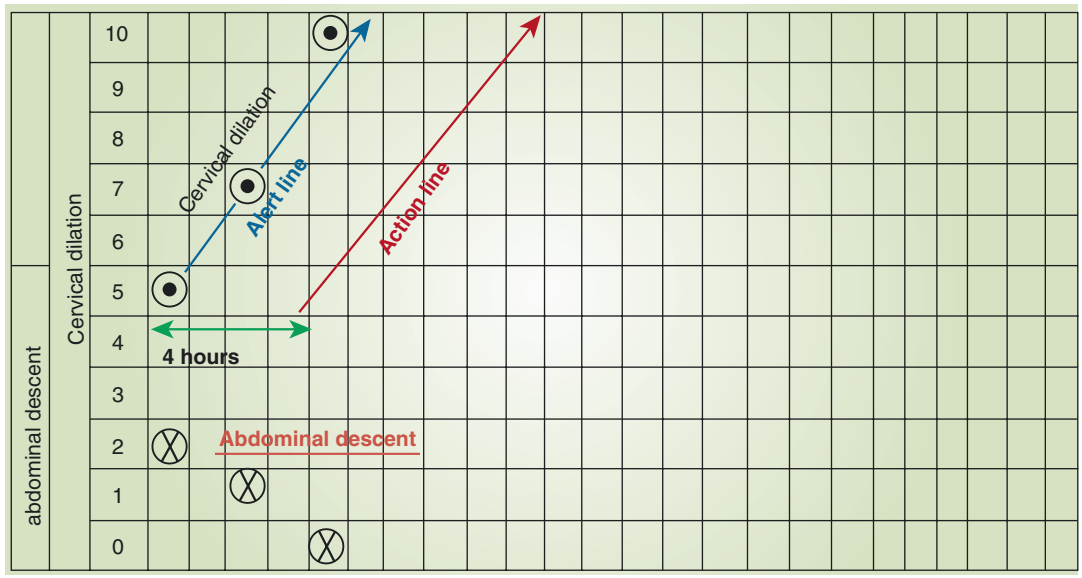

Fig. 25.4 Cervicograph

i.e. 4 h to the right of alert line, is plotted the action line, and both are parallel to each other. If there is delay in labour, the plot will approach the action line, which signifies that some action is to be taken (Fig. 25.4).

(b) Descent of fetal head: this part also a measure of progress of labour is marked on the cervicograph with "o" (Fig. 25.4). The descent of fetal head can be measured by abdominal method and by vaginal examination. In the abdominal method, it is measured in terms of fifths above the pelvic brim. The width of five fingers is used to find the fifths above the pelvic brim. A head which is above the pelvic brim accommodates all five fingers. With progressive descent of the fetal head, the portion of the head above the brim is represented by the respective number of fingers.

In the vaginal examination, the station of fetal head is assessed in relation to the ischial spines. The head at the level of the ischial spine is 0 station. The head above the ischial spines is represented in negative numbers and below as positives. A head that is 5 cm above ischial spines is noted as −5 and that at 4, 3,2 and 1 cm above the spines are −4, −3, −2 and −1, respectively.

To plot the descent on the partograph, the numbers 0–5 on the y-axis of the cervicograph are used. However, for vaginal examination values, the following conversions are to be used.

Station of fetal head	Mark on cervicograph
−4 or −3	5
−2 or −1	4
0	3
+1	2
+2	1
+3	0

The last two rows of this portion are to write the number of hours since the beginning of monitoring and the time on the clock.

(c) Uterine contractions: adequate uterine contractions are required for good progress of labour. Half-hourly contractions are assessed and recorded on the partograph. Frequency of contractions is measured as number of contractions in 10 min. One contraction is represented by one square. The number of squares is shaded according to the number of contractions.

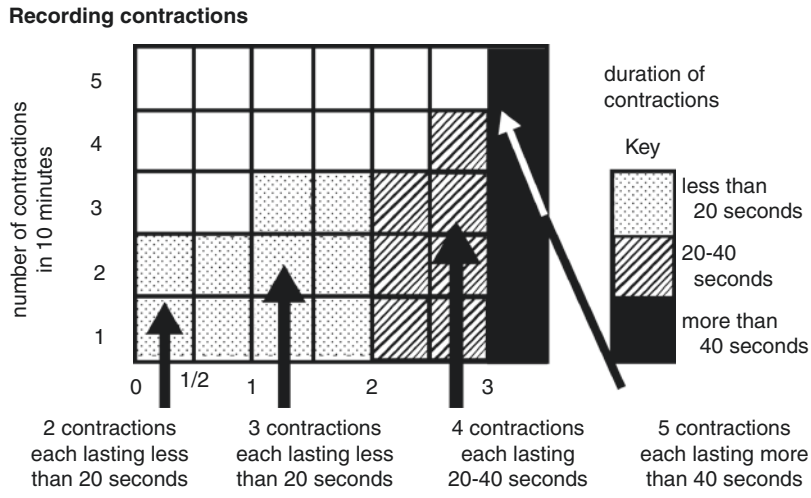

Fig. 25.5 Partographic representation of uterine contractions

The intensity of contractions is assessed by the duration of contractions. The shading patterns used are "dotted" for contractions lasting less than 20 s, "diagonal lines" indicating contractions between 20 and 40 s and "solid shading" for contractions more than 40 s (Fig. 25.5).

3. **Maternal record**: the next section of the partograph is the maternal record, where the vitals of the parturient and the drugs and fluids given to her are recorded. There are two rows for recording the administration of oxytocin being given to the patient during the course of labour. In the next part, the drugs and iv fluids given to the labouring woman are noted. Just below is the area to record the vitals of the patient. The chart is labelled from 60 to 180 where the pulse is recorded half-hourly and the blood pressure is noted every 4 hourly. Below that the temperature of the patient is noted in degree centigrade. At the bottom the characteristics of maternal urine are noted. Volume of the urine and presence of proteins and acetone are charted.

Advantages:

1. Pictorial representation on a single page and hence easy to grasp.
2. Recording labour events is easy and quantifiable.
3. Easy and early recognition of abnormal labour and thus effective in preventing prolonged labour and its sequel. In a WHO multicentre trial, improvements in maternal and fetal mortality and morbidity took place among both nulliparous and multiparous women after the use of a partograph [6].
4. Inexpensive.

Disadvantages:

1. Risk factors present before the start of labour are not identified.
2. Vaginal examination done by separate individuals increases the range of error and unnecessary intervention.

25.3 Paperless Partograph

It was first used and demonstrated by Dr. A K Debdas of Jharkhand India [7]. It is a simple, low-skill method for predicting the expected time of delivery (ETD), hence preventing prolonged labour. In this, the care provider records two timings, the alert ETD and the action ETD.

25.3.1 Calculation of ETD

It uses the principle of Friedman's formula where labour progress is determined by the rate of cervical dilatation at the rate of 1 cm/h, in the active phase. Thus the original concept used in WHO partograph is kept intact. The alert ETD is calculated

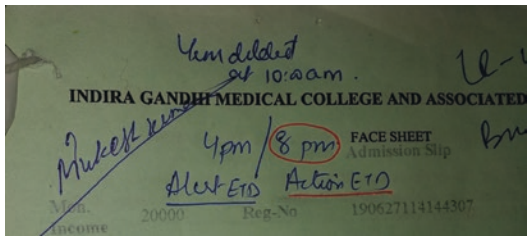

Fig. 25.6 Paperless partogram

by simply adding 6 h to the time when the cervix of the parturient is 4 cm dilated. The action ETD is calculated by adding another 4 h to alert ETD.

Both the ETDs are mentally calculated after determining the cervical dilatation and noted in big and bold letters on the case sheet. They are encircled, the action ETD in red.

25.3.2 Significance of ETDs

If the patient has not delivered by the time of alert ETD, one has to get alert, and reassessment of the case in view of the "passage", "passenger" and "power" is to be done. In case the patient fails to deliver in the next 4 h, i.e. by the action ETD, action is taken to deliver her soon by suitable interventions (Fig. 25.6).

Advantages

- Easy to use as it requires a simple two-step calculation.
- Can be used by health workers in low-resource settings and midwives.
- Not time-consuming.

25.4 Conclusion

Partograph is a valuable tool to assess the progress of labour and it is designed for the early detection of the protracted and arrest disorders and of fetal and maternal distress, thus reducing the maternal and neonatal mortality and morbidity. The use of partograph should be promoted to improve the management of labour.

References

1. Friedman EA. Primigravid labor; a graphicostatistical analysis. Obstet Gynecol. 1955;6:567–89.
2. Philpott RH. Graphic records in labour. BMJ. 1972;4:163.
3. Philpott RH, Castle WM. Cervicographs in management of labour in primigravidae I. The alert line for detecting abnormal labour. J Obstet Gynaecol Br Commonw. 1972;79:592–8.
4. Philpott RH, Castle WM. Cervicographs in the management of labour in primigravidae II. The action line and treatment of abnormal labour. J Obstet Gynaecol Br Commonw. 1972;79:599–602.
5. ACOG: management of fetal heart rate tracings. Practice bulletin no. 116, Nov 2010, reaffirmed 2013b.
6. World Health Organization. World Health Organization partograph in management of labour. Lancet. 1994;343:1399–404.
7. Debdas AK. Paperless partogram. 41st Annual Scientific Sessions 2008: Sri Lanka College of Obstetrics and Gynaecologists. SLJOG vol. 30. 2008;1:124.

Pain Relief in Labor

26

Ajay Sood and Nishi Sood

> The delivery of the infant into the arms of a conscious and pain-free mother is one of the most exciting and rewarding moments in medicine—Moir

Birth of a child is one of the most dangerous moments in the life of a woman. It is most of the time surrounded by misconceptions. Severe pain as a result of labor and delivery is the most feared event in pregnant women. However, the perception of pain is highly variable and unpredictable. Most women report intense pain from their first contraction, while some may not experience pain till the second stage of labor. Labor pain caused by uterine contractions and cervical dilatation in the first stage is transmitted through visceral afferent sympathetic nerves from T_{10} to L_1. In the second stage, painful stimuli due to perineal stretching are carried by pudendal nerve and sacral nerves S_2 to S_4 (Fig. 26.1). The exact mechanism of this difference in pain perception is not completely understood but may be genetically related. A study by Debiec J et al. found that Asian women experienced greater pain in labor than women of other races [1]. The association of pain has also been found with a single nucleotide polymorphism in the β2-adrenergic gene [2]. Other factors affecting pain include shape and size of the pelvis, presentation of fetus, and augmentation of contractions.

The McGill Pain Questionnaire ranks labor pain toward the upper end on the pain scale between that of cancer pain and amputation of a digit. The intensity of labor pain is variable with nulliparous women experiencing greater pain (Fig. 26.2), hence the importance of providing effective and safe analgesia during labor though till date it has remained an ongoing challenge. Throughout history several methods for labor analgesia have been advocated. Labor analgesia did not start till the middle of the nineteenth century. John Snow in 1853 gave chloroform analgesia to Britain's Queen Victoria while giving birth to her eighth child.

Currently there is a shift in obstetrical anesthesia practice with emphasis on overall management of patient care including quality of analgesia rather than simple focus on pain relief only [3]. Better understanding of physiology as well as pharmacology along with the coming up of obstetric anesthesia as a subspecialty has led to an absolute advancement in the field of labor analgesia. Regional analgesia for labor has today become a part of standard obstetric care.

A. Sood
Department of Anaesthesia, IGMC, Shimla, India

N. Sood (✉)
Department of Obg, IGMC, Shimla, India

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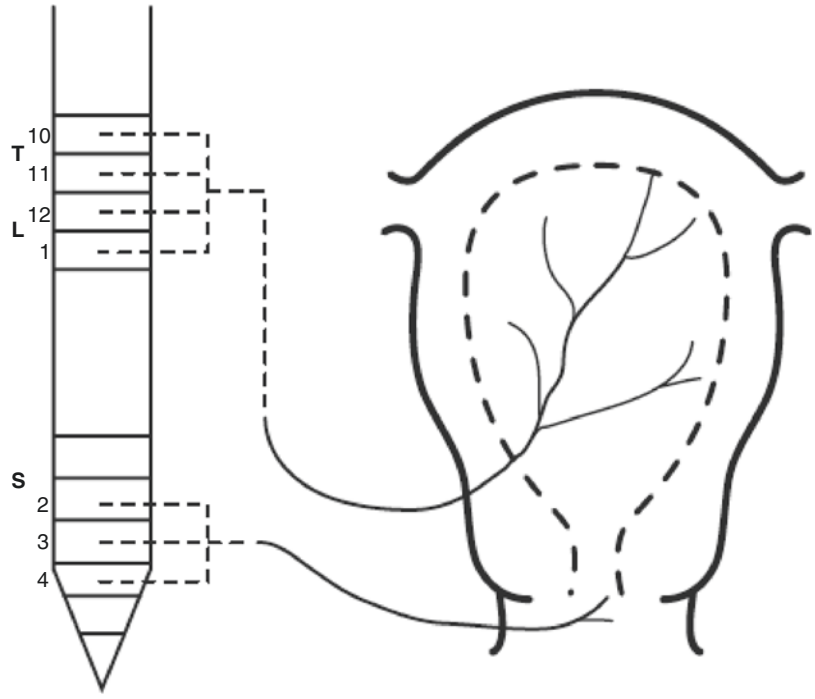

Fig. 26.1 Sources of pain during labor

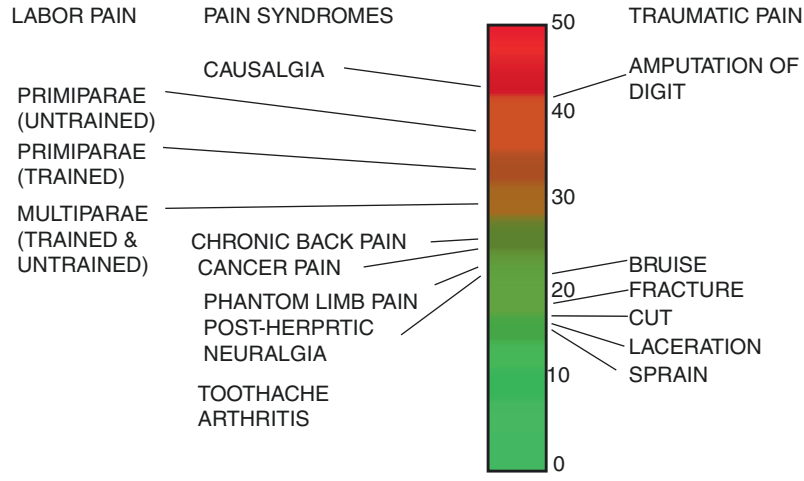

Fig. 26.2 Severity of labor pain

26.1 Effects of Labor Pain

Painful contractions result in maternal hyperventilation, causing respiratory alkalosis with a leftward shift of the oxyhemoglobin dissociation curve and increased maternal hemoglobin affinity for oxygen. Hypocarbia also leads to hypoventilation in between contractions, which may further decrease the maternal PaO_2. Increased oxygen consumption which occurs during labor along with hypocarbic uteroplacental vasoconstriction is a potential cause of fetal hypoxemia. Increases in cardiac output and vascular resistance may increase maternal blood pressure. The response of sympathetic stimulation to pain also leads to increase in levels of catecholamines which can depress the uterine activity and uteroplacental circulation. Effective labor analgesia

benefits most the high-risk parturient, i.e., those with preeclampsia or cardiac disease and those with marginal uteroplacental circulation and function by attenuating these responses.

26.2 Methods of Pain Relief in Labor

26.2.1 Nonpharmacologic Methods

Expectation of the patient concerning labor influences the childbirth experience.

The concept of natural childbirth was given by Eappen and Robbins in 1940 which consisted of teaching relaxation techniques and the elimination of fear to prevent labor pain [4]. Fernand Lamaze introduced psychoprophylaxis involving focusing on objects or breathing exercises to distract from pain, along with the use of conventional analgesic drugs [5]. Although taught extensively, there is a lack of scientific validation of the efficacy of these methods. Patients have been found to use coping techniques in the early first stage of labor but less so as labor progresses. Prepared childbirth by most modern instructors provides information about the elementary physiology of pregnancy and delivery with the aim to reduce fear and teach relaxation and breathing exercises.

Nonpharmacologic methods are preferred by some patients for pain management during labor. Acupuncture has been found to be useful in treating postoperative analgesia after cesarean section but shown poor results in controlling pain during labor [6]. In a meta-analysis to observe the efficacy of acupuncture, it was found that acupuncture had a small but statistically significant effect on labor pain, but the effect lasted for only 30 min [7].

A Cochrane review on massage for labor pain relief found that though pain decreased marginally in the massage group in the first stage of labor however no difference was seen in the second and third stages of labor [8].

Hypnosis as a relaxation technique has also been used for pain relief in labor. However no conclusive evidence is reported in literature. Other nonpharmacologic techniques such as transcutaneous nerve stimulation (TENS), hydrotherapy, presence of a support person, intradermal water injections, and even biofeedback have all been used for pain relief during labor.

26.2.2 Pharmacological Treatment of Pain in Labor

26.2.2.1 Inhalational Analgesia

Inhalational agents were the earliest to be used for labor analgesia in modern times. The technique involves the inhalation of subanesthetic concentrations of an agent while the mother remains awake with intact protective laryngeal reflexes. It can be used alone or in combination with other methods of analgesia. Increasing usage of volatile anesthetics in childbirth resulted in rising incidence of side effects especially neonatal depression and maternal gastric aspiration [9, 10]. This leads to the origin of fasting measures, and the guidelines recommended by Mendelson became cornerstones of obstetric anesthesia practice.

Nowadays volatile anesthetics are not very popular for pain relief in labor although nitrous oxide is still being used in few less developed countries. It is mixed with O_2 in a 50:50 ratio or slightly more for patient inhalation via a demand valve through a low-resistance breathing system. It takes 45 s for the analgesic effect to be achieved; hence the parturient should start breathing Entonox at the start of a contraction to achieve desired brain concentration at the height of contraction. Deep, slow breathing and abstaining from Entonox between contractions is encouraged. Studies have demonstrated different response to inhaled N_2O varying from moderate analgesia [11, 12] to no difference in visual analogue pain scores [13]. The analgesic effect of N_2O may be confounded by sedative and relaxing effects that benefit the laboring woman. Use of N_2O in O_2 is safe and does not cause hypoxia, unconsciousness, or loss of protective airway reflexes [14] which is commonly seen with opioids. Also there is no effect on uterine contractility and neonatal depression irrespective of duration of use.

The efficacy of small concentrations of halothane, enflurane, isoflurane, desflurane, and sevoflurane in labor, alone and in combination with nitrous oxide, has also been demonstrated [15–17]. As sevoflurane has early onset and short duration of action, it is the most preferred inhalational agent for labor analgesia and can be given as patient-controlled inhalation analgesia. But with the use of inhalational agents, environmental pollution is always a concern as well as risk of loss of consciousness and compromised airway reflexes leading to aspiration [18].

26.2.2.2 Parenteral Agents

Many agents that activate the μ-opioid receptors provide good pain relief during labor. Opioids are widely available and relatively cheap, hence commonly used. However as there is placental transfer of opioids, their use in labor is a compromise between effective analgesia and unwanted side effects.

Meperidine is the most commonly used long-acting opioid in obstetric practice worldwide [19]. Maternal $T^{1/2}$ of meperidine is 2–3 h but is 13–23 h in fetus and newborn due to an active metabolite normeperidine which crosses the placenta and causes respiratory depression. Newborn is at greater risk with increased dose and shorter dosing intervals [20].

Morphine is no longer used for labor analgesia as it causes excessive maternal sedation and significant neonatal respiratory depression with loss of fetal beat-to-beat variability.

Butorphanol and nalbuphine are mixed agonist/antagonist opioids which are very popular, especially in the USA, for the relief of labor pain. These drugs do cause respiratory depression but exhibit a ceiling effect with increasing doses. The dose of butorphanol is 2–4 mg intramuscularly but is associated with 75% incidence of transient sinusoidal fetal heart rate pattern. For this reason nalbuphine 10 mg i.v. has become the drug of choice in many institutions though it causes some maternal sedation.

Nowadays, shorter-acting fentanyl and ultrashort-acting remifentanil are gaining popularity for pain relief. Fentanyl is a highly lipid-soluble synthetic opioid with 100 times analgesic potency in comparison to morphine. Its onset of action after intravenous administration is within 2–3 min with a short duration of action and no major metabolites thus making it a good drug for labor analgesia. In small doses it does not cause any significant difference in neonatal Apgar scores and respiratory depression [21]. Because of its favorable pharmacokinetics and pharmacodynamics, patient-controlled intravenous analgesia (PCA) is also a suitable mode for fentanyl administration.

Remifentanil has emerged as an alternative to regional analgesia for patients with contraindications and for those unwilling for regional anesthesia. It is an ultrashort-acting opioid and has a favorable safety profile. As it is metabolized by placental esterase, hence fetal blood concentration is less. Moreover esterase enzymes are mature in the fetus so the metabolism of remifentanil is unaffected [22]. The recommended dose of remifentanil is 20 μg intravenously. During remifentanil infusion, it is important to monitor for maternal hypoventilation as episodes of oxygen desaturation are observed.

Ketamine, a phencyclidine derivative, in doses of 10–20 mg intravenously, also produces good analgesia in 2–5 min without loss of consciousness. Doses up to 1 mg kg^{-1} do not cause fetal depression and have no effect on uterine tone. Although its short duration of action makes ketamine unsuitable for first-stage analgesia, it may be effective just prior to vaginal delivery or as an adjuvant to regional anesthesia. The potential for unpleasant psychomimetic effects however must be borne in mind though the incidence is minimal with low doses.

In case of parenteral analgesics, intravenous administration is superior to intramuscular injection as there is less variability in peak plasma concentrations and faster onset of analgesia. Patient-controlled analgesia (PCA) is now widely used in pain relief. Suggested advantages of PCA are better analgesia at lower doses, resulting in less maternal respiratory depression, less placental transfer, less emesis, and higher patient satisfaction. Meperidine, fentanyl, and the mixed agonist/antagonist nalbuphine are the most frequently used opioids for PCA administration.

26.3 Neuraxial Analgesia

26.3.1 Epidural Analgesia

Epidural analgesia for labor was started by Stoeckel in 1909 when he gave caudal epidural anesthesia in 141 cases. Since then, lumbar epidural analgesia has been frequently used for labor and has become the gold standard for pain management in obstetrics [23]. Neuraxial analgesia is the most effective method of intrapartum pain relief with least depressant effect on the parturient and fetus in current practice [24]. Epidural analgesia also offers versatility in the level and duration of effect. Moreover segmental lumbar analgesia can be titrated for the first stage and extended into the sacral segments for the second stage of labor. By providing effective pain relief, epidural analgesia attenuates the adverse physiological responses to pain. In addition, effective epidural analgesia reduces maternal catecholamine levels, increasing intervillous blood flow and resulting in improved uteroplacental perfusion [25]. A much larger beneficial effect is seen in patients having preeclampsia [26]. Local anesthetics, at clinically used concentrations for neuraxial blocks, do not have a direct effect on uterine activity. Neonatal effects may occur, indirectly, from reduced uteroplacental perfusion due to hypotension. The duration and severity of maternal hypotension is important factor in determining fetal hypoxia and consequent neonatal neurobehavioral changes.

Preprocedural assessment for risk factors for regional analgesia and general anesthesia is recommended. Although any laboring woman has the potential to require cesarean section and theoretically should be kept fasting, labor as such takes many hours so adequate nutrition and hydration to the parturient has to be ensured. While balancing these two considerations, the American Society of Anesthesiologists (ASA) has recommended that clear fluids be allowed during the administration of regional analgesia and labor and a period of absence from solids is not required before the placement of regional analgesia.

26.3.1.1 Indications

Current ASA guidelines as well as ACOG committee recommend that maternal request for pain relief during labor is sufficient indication for any sort of intervention and the decision should not left on any degree of cervical dilation [27]. However, there are some categories of patients who will obtain specific benefits. Epidural analgesia decreases the hemodynamic effects of contractions as well as those due to pain response, which is desirable for patients with hypertensive disorders, asthma, diabetes, and cardiac and intracranial neurovascular disease. Epidural analgesia is specifically indicated when general anesthesia involving intubation is suspected to be difficult, as the block can be extended should operative intervention become necessary. Obstetric indications include prolonged labor, oxytocin augmentation of labor, and any factors that place the parturient at high risk for cesarean section. Fetal indications include prematurity, breech presentation, and multiple gestation, as greater control of delivery is possible and the depressant effects of systemic opioids are avoided.

Epidural has frequently been blamed by the obstetricians for increased rate of operative delivery. However the Cochrane Database trials have clearly stressed that epidural analgesia does not significantly impact the risk of cesarean section. In two meta-analyses of randomized trials, patients who received or did not receive epidural analgesia were compared, and no relationship of epidural and increased cesarean section was found [28]. However regional analgesia does increase the duration of labor by an average of about 1 h, and also there is increased incidence of occipitoposterior position as well as increased chance for augmentation of labor with oxytocin. However the low-concentration mixtures which are used nowadays have resulted in decreased incidence of these undesirable effects [29].

26.3.1.2 Contraindications

Contraindications to regional anesthesia in obstetric patients are same as those which apply to the general population. Absolute contraindications are infection over the site of placement, frank coagulopathy, hypovolemic shock, and

Table 26.1 Absolute contraindications to neuraxial analgesia

Refractory maternal hypotension
Maternal coagulopathy
Thrombocytopenia
Low molecular weight heparin within 12 h
Septicemia
Skin infection over needle site
Increased intracranial pressure

patient's refusal or inability to cooperate (Table 26.1). The American College of Gynecologists has concluded that women with platelet count of >50,000/μL can however be a candidate for regional analgesia [30]. Relative contraindication in the parturient in performing neuraxial blockade is bacteremic patients with risk of subsequent development of meningitis and epidural abscess. There is consensus that regional anesthesia is safe in parturients with recurrent genital herpes infection, in the absence of systemic symptoms.

Women receiving anticoagulation therapy are at a higher risk of developing spinal cord hematoma and subsequent compressive symptoms. The ACOG [30] has reaffirmed that:

1. Women receiving unfractionated heparin therapy but having normal activated partial thromboplastin time (aPTT) should be able to receive regional analgesia.
2. Women who are only on low-dose heparin or getting prophylactic dose of unfractionated heparin are not at increased risk and can be considered for neuraxial analgesia.
3. For women on once-daily dose of LMWH, wait of 12 h after last injection is warranted before placement of regional analgesia.
4. After the epidural catheter removal, low molecular weight heparin should be started only after at least 2 h.

26.3.1.3 Conduct of Epidural Analgesia

Epidural anesthesia in the laboring patient is performed similarly as in the general surgical patient. Resuscitation equipment trolley should be kept ready for immediate use. After obtaining informed consent, a good intravenous access is established. Vital signs are to be continuously monitored. At the time of epidural placement, the fetal heart rate should also be monitored. The sitting position for introduction of epidural improves identification of the midline especially in obese patients and also reduces the incidence of aortocaval compression, whereas the lateral position apart from being more comfortable position for the laboring patients offers advantage of less incidence of orthostatic hypotension. Ultrasound has come up in recent times for imaging of the spine helping in easy identification of epidural space and predicting difficult spine score.

The most common sites for epidural catheter placement are into the space between $L_{2–3}$ and $L_{3–4}$. Analgesia should be initiated preferably after a test dose to prevent inadvertent intrathecal or intravascular placement [31]. The inclusion of epinephrine in test dose is controversial in pregnancy.

26.3.1.4 Maintenance of Epidural Analgesia

A bilateral sensory level of analgesia is established at initiation of the epidural block, which is subsequently maintained by continuous infusion, intermittent bolus injection, or patient-controlled epidural analgesia (PCEA) of a dilute mixture of local anesthetic, combined with a narcotic. An infusion of a dilute mixture offers the advantages of a uniform level of analgesia, increased maternal hemodynamic stability, less chance of local anesthetic toxicity, and a slower ascent of the level of anesthesia should intravascular or subarachnoid migration of the catheter occur in comparison to intermittent injections. The use of dilute concentrations also avoids loss of motor function. The intermittent technique needs reinforcement every 1–2 h though continuous techniques do not necessarily abolish the need for top-ups. Neonatal outcome is the same with both techniques. The superiority of either technique is controversial though patient satisfaction is reportedly higher with continuous techniques.

PCEA (patient-controlled epidural analgesia) consists of intermittent demand dosing controlled

by the patient, with or without a background continuous infusion. It is a method of the drug delivery system offering many advantages [32]. As there is a wide range of dose requirements among patients, the ability of the PCEA technique to titrate dose to effect is a big advantage. Another potential benefit is increased patient satisfaction from a greater degree of autonomy.

Whichever modality is used for the maintenance of epidural analgesia, the patient must be assessed regularly for maternal hemodynamics, fetal heart rate, sensory level, and degree of motor blockade. It is strongly recommended that oxygen saturation be monitored with epidural opioid use.

26.3.1.5 Complications

Hypotension and inadequate analgesia are the most common complications [33]. Uncorrected hypotension leads to decreased uteroplacental perfusion, causing fetal hypoxemia and acidosis, and must be treated urgently. Hypotension can be prevented by intravenous prehydration as well as by avoiding aortocaval compression. Inadequate epidural analgesia occurs in 1.5–5% of laboring patients, even in experienced hands. When the parturient complains of discomfort, the sensory level of analgesia should be assessed and the cervical dilation, stage of labor, uterine contraction, and fetal heart rate tracing noted. Other factors to be ruled out in case of inadequate analgesia include equipment problems (pump malfunction/disconnected tubing/empty reservoir bag) and a full bladder. The other potential risks associated with the epidural technique include subarachnoid catheter migration and opioid-related side effects. Owing to the dilute infusions used, intravascular catheter migration is more likely to result in diminishing analgesia than systemic toxicity. A progressive increase in the sensory level of analgesia or significant motor blockade may indicate subarachnoid migration. In both situations, the catheter should be examined and replaced as necessary. Intrathecal opioids as with parenteral administration are known to result in dose-dependent side effects which increase if the patient is also getting systemic opioids. In doses used clinically in epidural, opioids have no significant effect on neonatal Apgar scores though reports of fetal heart rate decelerations have been found.

Patient discomfort may also be an indication of progression to the second stage of labor as sacral nerves are large-diameter, myelinated fibers that require a large volume of more concentrated local anesthetic solution for blockade. An occipitoposterior fetal head is associated with more severe pain, often felt in the back. An increased concentration and infusion rate have to be used. Opioid boluses (50 μg fentanyl in saline) or PCEA may be considered. Severe, unrelenting pain despite an adequate sensory level and present between contractions may indicate placental abruption or uterine rupture known as Crawford's sieve [34].

Accidental subarachnoid placement/injection: The incidence of dural puncture varies approximately between 0.2% and 2%. The subsequent incidence of postdural puncture headache (PDPH), resulting from a 16 to 18G needle puncture, may reach 76–87% [35]. If inadvertent dural puncture occurs during epidural needle placement, the catheter may be threaded and a continuous spinal technique employed, or the epidural may be re-sited at another interspace.

Accidental intravenous placement/injection: Vessel puncture can take place on initial insertion of the epidural needle or catheter or later due to migration of the catheter tip. The typical frequency appears to be in the range between 7% and 8.5% in pregnant patients. The incidence of intravascular catheter placement can be reduced by injecting fluid through the epidural needle before catheter placement [35]. This emphasizes the need for vigilance when administering large drug doses through an indwelling epidural catheter for cesarean delivery. Accidental slow intravenous injection of local anesthetics results in subjective maternal symptoms of light headedness, apprehension, tinnitus, perioral paresthesia, and a metallic taste. Close attention to these prodromal symptoms will provide early warning of intravascular injection of local anesthetics and avoid their more dangerous toxic effects, such as seizures, ventricular dysrhythmias, and cardiac arrest.

26.3.1.6 Other Complications

Back pain after childbirth is common irrespective of whether regional analgesia has been used or not [36].

Infection is uncommon with practice of strict aseptic technique while giving spinal and epidural anesthesia [37, 38]. Another problem encountered during regional analgesia is because of the increased vascularity of the epidural space in pregnancy resulting in increased incidence of vessel injury. However, with normal platelets and coagulation factors, formation of epidural hematoma is extremely uncommon [39]. Hence all pregnant patients who have received epidural analgesia should be monitored till complete resolution of the epidural blockade. Direct nerve damage is exceedingly rare in laboring [40, 41].

26.3.2 Spinal Analgesia

In most cases, a single-shot spinal injection is not suitable for the first stage of labor because of its finite duration. Occasionally, it may be employed to provide analgesia in multiparous women presenting at an advanced stage of cervical dilation, when only one injection is anticipated. Continuous spinal analgesia can be provided via an intrathecal catheter. This technique has been used for patients in whom placement of an epidural catheter is problematic (morbid obesity, abnormal vertebral anatomy). Initial injection of 2.5 mg of bupivacaine with 25 μg of fentanyl is followed by infusion of the standard local anesthetic/opioid mixture but at lower rates of 1–2 mL h^{-1}. All continuous spinal catheters must be labeled appropriately, and the sensory level and intensity of motor block must be monitored closely to avoid high spinal anesthesia. The use of spinal microcatheters is not advocated by the Food and Drug Administration (FDA) because of cauda equina syndrome and more technical difficulties [42].

26.3.3 Combined Spinal-Epidural Analgesia

The combined spinal-epidural (CSE) technique offers the advantage of the rapid and reliable onset of profound analgesia combined with the flexibility and longer duration of epidural technique to provide satisfactory and safe analgesia [43].

There are several commercially available combined needle devices. A spinal needle is placed through the epidural needle, and after ensuring CSF flow, medication is given intrathecally. Subsequently an epidural catheter is placed through the epidural needle. This is the needle-through-needle technique. For spinal medication, a small dose of local anesthetic like 2.5 mg of bupivacaine and short-acting, lipid-soluble opioids such as fentanyl 10–25 μg or sufentanil 5–15 μg are used which give sensory analgesia without any motor block for a duration of 90–120 min. The low concentrations of local anesthetics and opioid used in epidural also allow the patient to ambulate. The usual practice is to give drugs intrathecally, followed by continuous epidural infusion of 0.125% bupivacaine with 2 μg mL^{-1} of fentanyl at 10 mL h^{-1} [44].

A CSE technique allows for a smooth change between the epidural and spinal portions of analgesia. Addition of adrenaline to the opioid/local anesthetic combination may prolong the analgesia. Different studies however have found no difference in maternal satisfaction, ability to ambulate, and mode of delivery between CSEA and epidural techniques [45].

26.3.3.1 Ambulation and Neuraxial Labor Analgesia

The interest in ambulation came from the belief that the upright posture along with mobility of the patient may have positive effect on the outcome of labor. Initial concerns with ambulation were for maternal safety (hypotension, risk of injury) and fetal well-being. However it is clear that ambulation does not improve labor outcome [46]. Uncomplicated and less painful labor permits ambulation, rather than vice versa. A typical walking epidural consists of continuous epidural infusion of a dilute local anesthetic/opioid mixture, e.g., 0.0625–0.1% bupivacaine with fentanyl 2 μg mL^{-1}, which retains sensory and motor function adequate to ambulate [47]. Prior to allowing ambulation, the clinician should assess for orthostatic blood pressure changes, lack of motor block (e.g., a deep-knee bend), and retention of proprio-

ceptive abilities (e.g., toe-pointing). The fetal heart rate is to be monitored on an intermittent basis or via a telemetry unit, and patients should not walk unaccompanied [48].

26.3.3.2 Neuraxial Analgesic Medications

Intrathecal lidocaine and 2-chloroprocaine have been extensively used but have been found to be associated with transient neurologic symptoms and even delayed cauda equina syndrome [49, 50]. Most of these cases were associated with high-concentration preparations with varying preservatives. Hence nowadays preservative-free local anesthetics are recommended to be used in an epidural catheter. Bupivacaine (0.0625–0.125%) and ropivacaine (0.1–0.2%) are the most commonly used drugs for obstetric analgesia because sensory blockade is greater than motor blockade with these drugs in comparison to lidocaine or 2-chloroprocaine. Transient neurologic symptoms and cauda equina syndrome however have also been reported with intrathecal bupivacaine [51]. Ropivacaine and levobupivacaine are less cardiotoxic even after accidental intravenous injection and hence safe. There are different dosing regimens for these drugs, some of which are highlighted in (Table 26.2).

Lipid-soluble opioids like fentanyl l–3 μg/mL or sufentanil 0.1–0.5 μg/mL added to local anesthetics allow the reduction of dose along with decreased motor blockade, preserved analgesia, and enhanced maternal satisfaction [52]. Another common adjuvant used is α2-agonist clonidine which can be given either epidurally or in spinal [53] though not recommended by the US Food and Drug Administration (FDA) because of the risk of hemodynamic instability. A more selective α2-adrenergic receptor agonist dexmedetomidine has also been found to be efficacious when combined with epidural bupivacaine [54]. Cholinesterase inhibitor neostigmine has similar effects to that of epinephrine and has also been used as adjuvant [55]. Epinephrine a nonselective adrenergic agonist acting on α1-, α2-, β1-, and β2-adrenergic receptors causes vasoconstriction in the epidural space delaying the vascular uptake of local anesthetic and opioid and prolonging the duration of analgesia [56]. Adrenaline itself also provides analgesia by activating α2-adrenergic receptors [57].

Table 26.2 Commonly used continuous and patient-controlled infusion regimens

Technique	Drug	Concentration and dosage
Epidural continuous infusion	Bupivacaine	0.0625–0.25% at 8–15 mL/h
Epidural continuous infusion	Ropivacaine	0.1–0.2% at 6–12 mL/h
Patient-controlled epidural analgesia (PCEA)	Bupivacaine	0.125%,4 mL basal infusion, bolus dose 4 mL, lockout 20 min, maximum 16 mL/h
PCEA	Bupivacaine + fentanyl 2 μg/mL^{-1}	0.125%,6 mL basal infusion, bolus dose 3 mL, lockout 10 min, maximum 24 mL/h
PCEA	Ropivacaine	0.1%,6 mL basal infusion, bolus dose 4 mL, lockout interval 10 min, maximum 30 mL/h
PCA	Fentanyl	50 μg loading dose i.v., PCA bolus 20 μg, 3 min lockout
Continuous spinal analgesia	Bupivacaine	Initial dose (bupivacaine 0.25%,1 mL + 25 μg fentanyl) followed by 1–2 mL/h (bupivacaine 0.125% + fentanyl 2 μg/mL)
PCA	Fentanyl	25 μg fentanyl continuous infusion, PCA bolus 25 μg, 12 min lockout, 4-h maximum dose 600 μg
PCA	Nalbuphine	5 mg loading dose i.v., PCA bolus 1 mg, 6 min lockout, discontinue when cervix completely dilated

26.4 Regional Nerve Blocks

Nerve blocks have been used frequently by obstetricians to relieve labor pain.

Paracervical block (Fig. 26.3) is performed to inhibit nerve conduction from the body of the uterus and cervix by injecting local anesthetic submucosally, in lateral vagina fornices at 4 o'clock and 10 o'clock. It provides pain relief because of cervical dilation but has no effect on pain arising from contractions of the uterine body. It provides better analgesia in comparison to intramuscular meperidine [58], while it is comparable to PCA with i.v. fentanyl [59]. Rarely this block can result in accidental injection into the presenting part, which can have grave consequences [60]. The close proximity of the injection site to the uterine artery also results in uterine arterial vasoconstriction and high fetal blood levels of local anesthetic which can cause fetal bradycardia. The needle guide technique of paracervical block is more safe.

Pudendal nerve block: The pudendal nerve can be blocked using either a transvaginal or transperineal approach in second stage of labor or for operative vaginal delivery. Though useful, it however inhibits the urge to push in the second stage of labor [61]. The other drawbacks of this block include a high rate of block failure, hematoma formation, and accidental fetal injection of anesthetic drug.

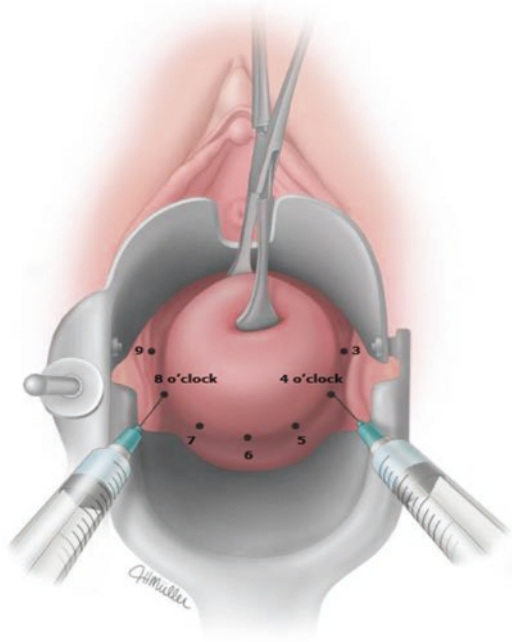

Fig. 26.3 Paracervical block

26.5 Conclusion

There is no single method of treating labor pain which can be used in any situation and which can satisfy every woman. Individual tailoring of anesthetic management of labor pain is required taking all the factors into account and finally providing safe and effective labor analgesia. Regional techniques have become the gold standard for intrapartum pain relief. Different trials have shown lower maternal pain scores and greater maternal satisfaction with regional analgesia in comparison to systemic opioids, use of nitrous oxide, or other methods. Early regional labor analgesia improves maternal satisfaction and has no negative impact on the mode of delivery. Recent recommendations are regarding the usage of large doses of less concentrated solutions of bupivacaine-opioid mixtures both at the start and for further maintenance of labor analgesia using PCEA. In the future, technologically superior pumps utilizing algorithm-based CI-PCEA programs might be more useful as they result in even spread of the infusate and better pain management. The use of microcatheters for continuous spinal analgesia and ultrasound guide for space identification can help to overcome difficulties in the placement of epidural catheters especially in difficult cases.

References

1. Debiec J, Conell-Price J, Evansmith J, et al. Mathematical modelling of the pain and progress of the first stage of nulliparous labor. Anesthesiology. 2009;111:1093–110.
2. Reitman E, Conell-Price J, Evansmith J, et al. Beta2-adrenergic receptor genotype and other variables that contribute to labor pain and progress. Anesthesiology. 2011;114:927–39.
3. Wong CA. Advances in labor analgesia. Int J Women's Health. 2009;1:139–54.

4. Eappen S, Robbins D. Nonpharmacological means of pain relief for labor and delivery. Int Anesthesiol Clin. 2002;40(4):103–14.
5. Lamaze F: Painless childbirth. Celestin LR (trans.) London: Burke, 1958.
6. Wu HC, Liu YC, Ou KL, et al. Effects of acupuncture on post cesarean section pain. Chin Med J. 2009;122:1743–8.
7. Cho SH, Lee H, Ernst E. Acupuncture for pain relief in labour: a systematic review and meta-analysis. BJOG. 2010;117:907–20.
8. Smith CA, Levett KM, Collins CT, et al. Massage, reflexology and other manual methods for pain management in labour. Cochrane Database Syst Rev. 2012;(2):CD009290.
9. Mendelson CL. The aspiration of stomach contents into the lungs during obstetric anesthesia. Am J Obstet Gynecol. 1946;52:191–205.
10. Moya F. Use of a chloroform inhaler in obstetrics. N Y State J Med. 1961;61:421–9.
11. Klomp T, Van Poppel M, Jones L, et al. Inhaled analgesia for pain management in labour. Cochrane Database Syst Rev. 2012;9:CD009351.
12. Westling F, Milsom I, Zetterström H, et al. Effects of nitrous oxide/oxygen inhalation on the maternal circulation during vaginal delivery. Acta Anaesthesiol Scand. 1992;36:175–81.
13. Carstoniu J, Lewtam S, Norman P, et al. Nitrous oxide in early labor: safety and analgesic efficacy assessed by a double-blind, placebo-controlled study. Anesthesiology. 1994;80:30–5.
14. Yentis MY, Cohen SE. Inhalational analgesia and anesthesia for labor and vaginal delivery. In: Shnider and Levinson's anesthesia for obstetrics. Philadelphia: Lippincott Williams & Wilkins; 2002. p. 189–97.
15. Abboud TK, Shnider SM, Wright RG, et al. Enflurane analgesia in obstetrics. Anesth Analg. 1981;60:133–7.
16. Abboud TK, Gangolly J, Mosaad P, Crowell D. Isoflurane in obstetrics. Anesth Analg. 1989;68:388–91.
17. Swart F, Abboud T, Zhu J. Desflurane analgesia in obstetrics: maternal and neonatal effects. Anesthesiology. 1991;75:A844.
18. Yeo ST, Holdcroft A, Yentis SM, Stewart A, Bassett P. Analgesia with sevoflurane in labour. II. Sevoflurane compared with Entonox for labour analgesia. Br J Anaesth. 2007;98:110–5.
19. Olofsson C, Irestedt L. Traditional analgesic agents: are parenteral narcotics passe and do inhalational agents still have a place in labour? Baillieres Clin Obstet Gynaecol. 1998;12:409–21.
20. Nissen E, Widstrom AM, Lilja G. Effects of routinely given pethidine during labour on infants' developing breastfeeding behaviour: effects of dose-delivery time interval and various concentrations of pethidine/norpethidine in cord plasma. Acta Paediatr. 1997;86:201–8.
21. Rayburn W, Rathke A, Leushcen P, et al. Fentanyl citrate analgesia during labor. Am J Obstet Gynecol. 1989;161:202–6.
22. Kan RE, Hughes SC, Rosen MA, et al. Intravenous remifentanil: placental transfer, maternal and neonatal effects. Anesthesiology. 1998;88:1467–74.
23. Hawkins JL. Epidural analgesia for labour and delivery. N Engl J Med. 2010;362:1503–10.
24. Robinson JO, Rosen M, Evans JM, et al. Maternal opinion about analgesia for labour. A controlled trial between epidural block and intramuscular pethidine combined with inhalation. Anaesthesia. 1980;35:1173–81.
25. Hollmen AI, Jouppila R, Jouppila P, et al. Effect of extradural analgesia using bupivacaine and 2-chloroprocaine on intervillous blood flow during normal labour. Br J Anaesth. 1982;54:837–42.
26. Jouppila P, Jouppila R Hollmen A, Koivula A. Lumbar epidural analgesia to improve intervillous blood flow during labor in severe preeclampsia. Obstet Gynecol. 1982;59:158–61.
27. American Society of Anesthesiologists Task Force on Obstetric Anesthesia. Practice guidelines for obstetric anesthesia: an updated report. Anesthesiology. 2007;106:843–63.
28. Leighton BL, Halpern SH. The effect of epidural analgesia on labor, maternal and neonatal outcomes: a systematic review. Am J Obstet Gynecol. 2002;186:S69–77.
29. Comparative Obstetric Mobile Epidural Trial (COMET) Study Group UK. Effect of low-dose mobile versus traditional epidural techniques on mode of delivery: a randomised controlled trial. Lancet. 2001;19–23(2001):358.
30. American College of Obstetricians and Gynaecologists. Obstetric analgesia and anaesthesia. Practice bulletin 36, July 2002, Reaffirmed 2013b.
31. Gaiser RR. The epidural test dose in obstetric anesthesia: it is not obsolete. J Clin Anesth. 2003;15:474–7.
32. Gambling DR, Yu P, Cole C, et al. A comparative study of patient controlled epidural analgesia (PCEA) and continuous infusion epidural analgesia (CIEA) during labour. Can J Anaesth. 1988;35(3 Pt 1):249–54.
33. Scott DB, Hibbard BM. Serious non-fatal complications associated with extradural block in obstetric practice. Br J Anaesth. 1990;64:537–41.
34. Crawford JS. The epidural sieve and MBC (minimal blocking concentration): a hypothesis. Anaesthesia. 1976;31:1277–80.
35. Lussos S, Datta S. Anesthesia for cesarean delivery. Int J Obstet Anesth. 1992;1:208–21.
36. MacArthur AJ, Macarthur C, Weeks SK. Is epidural anesthesia in labor associated with chronic low back pain? A prospective cohort study. Anesth Analg. 1997;85:1066–70.
37. McKenzie AG, Darragh K. A national survey of prevention of infection in obstetric central neuraxial blockade in the UK. Anaesthesia. 2011;66:497–502.
38. Green LK, Paech MJ. Obstetric epidural catheter-related infections at a major teaching hospital: a retrospective case series. Int J Obstet Anesth. 2010;19:38–43.

39. Bateman BT, Myhre JM, Leffert J, et al. The risk and outcomes of epidural hematomas after perioperative and obstetric epidural catheterization: a report from the multicenter perioperative outcomes group research consortium. Anesth Analg. 2013;116:1380–5.
40. Reynolds F. Neurological infections after neuraxial anesthesia. Anesthesiol Clin. 2008;26:23–52.
41. Cook TM, Counsell D, Wildsmith JA. Major complications of central neuraxial block: report on the third National Audit Project of the Royal College of Anaesthetists. Br J Anaesth. 2009;102:179–90.
42. Arkoosh VA, Palmer CM, Yun EM, Sharma SK, Bates JN, Wissler RN, et al. A randomized, double-masked, multicenter comparison of the safety of continuous intrathecal labor analgesia using a 28-gauge catheter versus continuous epidural labor analgesia. Anesthesiology. 2008;108:286–98.
43. Macarthur AJ. Gerard W. Ostheimer: "What's new in obstetric anesthesia" lecture. Anesthesiology. 2008;108:777–85.
44. Norris MC, Fogel ST, Conway-Long C. Combined spinal-epidural versus epidural labor analgesia. Anesthesiology. 2001;95:913–20.
45. Simmons SW, Cyna AM, Dennis AT, Hughes D. Combined spinal-epidural versus epidural analgesia in labour. Cochrane Database Syst Rev. 2007;(3):CD003401.
46. Nageotte MP, Larson O, Rumney PJ. Epidural analgesia compared with combined spinal-epidural analgesia during labor in nulliparous women. N Engl J Med. 1997;337:1715–9.
47. Sia AT, Chongj L, Tay DH, et al. Intrathecal sufentanil as the sole agent in combined spinal-epidural analgesia for the ambulatory parturient. Can J Anesth. 1998;45:620–5.
48. Parry MG, Fernando R, Bawa GP, Poulton BB. Dorsal column function after epidural and spinal blockade: implications for the safety of walking following low-dose regional analgesia for labour. Anaesthesia. 1998;53:382–7.
49. Beardsley D, Holman S, Gantt RM, et al. Transient neurologic deficit after spinal anesthesia: local anesthetic maldistribution with pencil point needles? Anesth Analg. 1995;81:314–20.
50. Ong B, Baker C. Temporary back and leg pain after bupivacaine and morphine spinal anaesthesia. Can J Anaesth. 1995;42:805–7.
51. Chabbouh T, Lentschener C, Zuber M, et al. Persistent cauda equina syndrome with no identifiable facilitating condition after an uneventful single spinal administration of 0.5% hyperbaric bupivacaine. Anesth Analg. 2005;101:1847–8.
52. Collis RE, Davies DW, Aveling W. Randomised comparison of combined spinal-epidural and standard epidural analgesia in labour. Lancet. 1995;345:1413–6.
53. O'Meara ME, Gin T. Comparison of 0.125% bupivacaine with 0.125% bupivacaine and clonidine as extradural analgesia in the first stage of labour. Br J Anaesth. 1993;71:651–6.
54. Sabbe MB, Penning JP, Ozaki GT, et al. Spinal and systemic action of the alpha 2 receptor agonist dexmedetomidine in dogs: antinociception and carbon dioxide response. Anesthesiology. 1994;80:1057–72.
55. Van de Velde M, Berends N, Kumar A, Devroe S, Devlieger R, Vandermeersch E, et al. Effects of epidural clonidine and neostigmine following intrathecal labour analgesia: a randomised, double-blind, placebo-controlled trial. Int J Obstet Anesth. 2009;18:207–14.
56. Niemi G, Breivik H. Adrenaline markedly improves thoracic epidural analgesia produced by a low-dose infusion of bupivacaine, fentanyl and adrenaline after major surgery: a randomised, double-blind, cross-over study with and without adrenaline. Acta Anaesthesiol Scand. 1998;42:897–909.
57. Meert TF, Noorduin H, Van Craenendonck H, et al. Effects of adrenaline, an alpha 2-adrenoceptor agonist, the volume of injection, and the global pain state of the animal on the activity of epidural sufentanil. Acta Anaesthesiol Belg. 1989;40:247–61.
58. Jensen F, Qvist I, Brocks V, et al. Submucous paracervical blockade compared with intramuscular meperidine as analgesia during labor: a double-blind study. Obstet Gynecol. 1984;64:724–7.
59. Nikkola EM, Jahnukainen TJ, Ekblad UU, et al. Neonatal monitoring after maternal fentanyl analgesia in labor. J Clin Monit Comput. 2000;16:597–608.
60. Morishima HO, Covino BG, Yeh MN, et al. Bradycardia in the fetal baboon following paracervical block anesthesia. Am J Obstet Gynecol. 1981;140:775–80.
61. Pace MC, Aurilio C, Bulletti C, et al. Subarachnoid analgesia in advanced labor: a comparison of subarachnoid analgesia and pudendal block in advanced labor: analgesic quality and obstetric outcome. Ann N Y Acad Sci. 2004;1034:356–63.

Nutrition in Labor

27

Priya Kannan

The experience of childbirth is unique to all cultures. This is a unifying factor for all cultures. Each woman's experience and her need during labor are individualistic. Traditionally, special foods have been offered during labor, and some specific food has been discouraged as well. The general belief and practice was to give food during labor to enhance energy of the woman. This was also acknowledged by doctors of that era [1]. After the landmark publication by Dr. Curtis Mendelson in [2], which implied aspiration as a cause of maternal death, intake during labor was completely restricted. The practice of fasting during labor has been under scrutiny in recent times, primarily due to the potential detrimental effects of fasting. Primarily owing to modern-day anesthesiology, it is time to compare the disadvantages of fasting to risk of aspiration during anesthesia.

Mendelson in the 1940s in his landmark publication reported that "there was an increased risk of the stomach contents entering the lungs during general anesthesia." He also explained in the same publication that as the food particles from the stomach are acidic in nature, when aspirated, it can lead to lung damage and possibly death. This influenced protocols in birth settings. Based on his extensive work, Dr. Mendelson proposed the following interventions during labor:

1. To avoid oral intake during labor.
2. To provide energy alternatively through intravenous route.
3. Wherever possible to use local anesthesia over general anesthesia.
4. Anesthesia should be administered by doctors trained specially in the particular field of specialization. (in those days, it was not mandatory that only anesthetists administer anesthesia.)

Since then birth settings have restricted oral intake during labor. The assumption was that gastric volume could be reduced by fasting. This view has been contended as there are no recent evidences to support this belief [3]. To compensate for the energy source during labor, intravenous glucose (or dextrose) has been suggested to be given [4–8]. The practice of intravenous glucose has been seen to have side effects in the mother such as low sodium, fluid overload, and increased blood glucose concentration. And moreover, there is very less evidence on the effectiveness of this approach [9]. On the contrary, it has been shown that intravenous fluids during labor may cause rebound low blood glucose in the baby, increased jaundice, and acidosis. These effects were observed more in compromised babies [10].

P. Kannan (✉)
Garbba Rakshambigai Fertility Centre, Chennai, India

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27.1 Physiology of Energy Utilization During Labor

During early days and for centuries before that, women were encouraged to eat during labor to avoid general weakness, delayed labor, and serious postpartum hemorrhage, as listed by DeLee. He revised his statement after Dr. Mendelson's publication. The uterus uses glucose as its primary source of energy. Fat is another source of energy for the uterus. There is increased need for oxygen during labor [11], and hence there is increased necessity for glucose by women in labor [12]. During labor, to compensate for the increased demand, the liver increases glucose production. Metabolic processes are readjusted to suit the increased need for energy during pregnancy and labor. There is a dearth of research articles about specific nutritional needs of women in labor. It is suggested that the energy requirement during labor could be similar to that of moderate cardiac exercise.

Elevated ketones are seen during exercise and starvation. The same may happen as part of the physiological response during labor, especially while the mother is fasting. Much significance was not attached to this until associations were established between elevated ketone levels, longer labor, and maternal psychological stress [13]. It is however difficult to determine whether the longer labor hours is due to ketone production or vice versa. During long labor, presence of ketonuria should be taken as a sign of metabolic imbalance.

Due to the increased fetal energy requirements resulting in increased fat metabolism along with the pregnancy-induced hormone changes, pregnant women are prone to ketosis [14]. Kubli et al. [15] concluded that ketone synthesis is increased in women in labor who are subjected to prolonged fasting especially ß-hydroxybutyrate and acetoacetic acid. In the 1960s and 1970s, to overcome ketosis, women in labor were administered intravenous fluids with glucose or dextrose. When given in large doses aggressively, intravenous dextrose was shown to cause fetal lactic acidosis, newborn jaundice, and hypoglycemia. Hence this protocol was discontinued [16]. Presently there are no guidelines to manage ketosis of labor.

27.2 Effect of Oral Intake during Labor

27.2.1 Light Diet

Scrutton et al. [17] compared the effect of light diet with water and water only in regard to ketosis. The light diet consisted of food such as cereal, bread, butter/jam, low-fat cheese, fruit juice, and beverages with milk such as coffee, tea, and hot chocolate along with water. Ketosis was effectively prevented in the light diet group, and the mothers had significantly increased gastric volume. The end points measured included duration of labor, oxytocin requirement, Apgar scores of the newborn, and mode of birth. There was no statistical difference in any of the parameters; both arms of the study have no significant difference. The drawback of the study was that it wasn't sufficiently powered enough.

27.2.2 Sports Drink

Kubli et al. in their study in 2002, which aimed to study metabolic effects during labor, compared isotonic "sports drinks" to only water during labor. The women in the study group were given 925 mL of sports drink that provided 28 kcal/dL (64 g/L or 6% carbohydrate solution), which also provided small amounts of plasma beta-hydroxybutyrate. The nonesterified fatty acids were measured at two points: (1) early labor and (2) end of the first stage of labor. The residual gastric volume was assessed using ultrasound 45 min after delivery. The study found that at the end of the first stage of labor, in the water-only group, plasma beta-hydroxybutyrate and nonesterified fatty acids had increased, and plasma glucose ($P = 0.007$) had decreased significantly. The other parameters studied, namely, maternal and neonatal outcome, gastric volume, incidence of vomiting, volume of vomit during labor or within an hour of delivery, dura-

tion of labor, rate of augmentation, mode of birth, Apgar scores, and umbilical artery and vein pH, were also similar in both groups. Hence the researchers concluded that there is a reduction in maternal ketosis without increase in gastric volume when isotonic drinks were given to women in labor.

27.2.3 Carbohydrate Solution

201 consecutive nulliparous women, with singleton in cephalic presentation in early labor (2–4 cm of cervical dilatation), were included in a randomized, double-blind, placebo-controlled trial by Scheepers et al. in [18]. The women in the study arm received a solution to drink which had 12.6% carbohydrate. The intake in the placebo group was on an average 300 mL, while the median intake in the study group was 400 mL. The carbohydrate solution was well tolerated. While the rate of abdominal delivery was significantly higher in the study group, 7% in the placebo group, and 21% in the study group (RR 2.9, 95% CI 1.29–6.54), there were no statistically significant differences in other parameters such as duration of labor or use of forceps or vacuum to assist vaginal delivery.

The same group [19] continued to study the effects of oral carbohydrate on maternal metabolism, fetal metabolism, and clinical outcomes. This study involved 202 women in advanced labor with 8–10 cm of cervical dilatation. The women in labor were given either a carbohydrate drink or placebo. Metabolic parameters were measured, in a subgroup of 28 women. In the carbohydrate group, instrumental delivery was not reduced (RR 1.1, 95% CI 0.9–1.30), though there was a marginal reduction in the cesarean section rate which however was not statistically significant (1% vs. 7%, RR 0.2, 95% CI 0.02–1.2). In the study group, there was an increase in maternal lactate and a reduction in maternal free fatty acids and a positive venous-arterial lactate difference in the umbilical cord. Though the placebo group had negative venous-arterial lactate difference in the umbilical cord, the differences in pH were comparable.

In an interesting parallel prospective randomized controlled trial, 190 singleton cephalic pregnancy in labor with 3–4 cm dilatation were assigned to either carbohydrate (intervention $N = 87$) or control ($N = 90$) group. The women in the intervention group were asked to have three dates with 110 mL water or three dates with 110 mL light tea without sugar or 110 mL orange juice drink. The primary outcome was to measure the duration of the active phase of labor. It was seen that there was a reduction in the active stage of labor in the intervention group ($P < 0.05$), but there were no significant differences in other maternal and neonatal outcomes. Hence the authors concluded that oral intake of carbohydrate could be an effective way of shortening the duration of second stage of labor in low-risk women.

27.2.4 Low-Fat Diet

In a prospective randomized controlled trial by O'Sullivan et al. in [20], 2426 nulliparous, nondiabetic women in labor with less than 6 cm cervical dilatation were included. One arm were given low-fat diets during labor and others water only. In this study, no significant difference was noted in terms of spontaneous vaginal delivery rate (44%), duration of labor (597 min vs. 612 min), cesarean delivery rate (30% vs. 30%), or incidence of vomiting (35% vs. 34%) between the study and control groups. Hence, the authors concluded that a light diet during labor did not alter obstetric, neonatal outcomes nor did it increase the incidence of vomiting.

27.2.5 Energy Drink

In another study by Kardel et al. [21] involving 213 healthy nulliparous women at gestational age of >36 weeks, extra energy was given to women in labor along with the self-regulated diet. The aim of the study was to know the effect of the extra energy on the duration of labor. The result showed that the median time to delivery in the intervention group was 9 h (61%) and the median

time to delivery in placebo group was 58 h (58%). The difference in duration seen was not statistically significant. Hence, it was concluded that energy drink beyond self-regulated diet during labor was not essential.

27.3 Does Oral Intake Influence the Following?

27.3.1 Length of Labor

Two randomized controlled trials ([17, 18]) have found that intake during labor did not have any significant effect on the length of labor, as also seen in the studies mentioned above.

27.3.2 Nausea and Vomiting

One randomized controlled trial by Scrutton et al. in [17] observed an increase in the incidence of nausea and vomiting in women who had oral intake during labor. In this RCT, twice as many women who had light diet vomited compared to women who had water only (38% vs. 19%). Three other trials [15, 22, 23] did not find any difference in the incidence of nausea and/or vomiting in women who were allowed to have isotonic drinks during labor. In the sports drink group, mean plasma glucose remained unchanged, while it was less in the water-only group, and this difference was statistically significant.

27.3.3 Operative Birth

A study by Scheepers et al. in [18] found cesarean section rate to be higher in women who had carbohydrate drinks in labor as opposed to those drinking only water (21/102 = 21% vs. 9/99 = 9%), while in another trial by Kubli et al. in [15], the group did not see any difference in cesarean section rate between the carbohydrate and water-only groups (carbohydrate drink group, 6/30 = 20%, vs. water-only group, 8/30 = 27%).

27.4 Physiology of Gastric Emptying in Pregnant Women

Pregnancy is marked by the following features—reduced esophageal sphincter tone and reduced GI tract motility. The gastrin levels are found to be high during the late third trimester, labor, and also during the immediate postpartum period. The increase in size of the uterus as pregnancy progresses leads to increase in the intragastric pressure, especially in the third trimester [24], which makes the pregnant women more prone to regurgitation and aspiration. Any other factor that augments these physiologic changes such as GERD or COPD increases the risk of pulmonary aspiration, especially when general anesthesia is administered during operative birth [25]. In another study by Smith et al. in [26], contrary to the prevalent theory and practice, they concluded that there was no correlation between the volume of gastric content and the probability of pulmonary aspiration.

It has been shown in many studies that the stomach is never completely empty [27], but still, it is regular practice to insist on at least 6 h of fasting prior to surgical intervention to have an empty stomach. It has been suggested that contrary to the existing belief, fasting would result in accumulation of acidic secretion in the stomach thus increasing the gastric volume, which also may contribute to increasing the risk of aspiration [28]. A gastric pH less than 2.5 and/or a volume greater than 0.4 mL/kg would cause lung injury if aspirated [29]. In spite of this basic physiological information and multiple publications that have demonstrated that fasting does not reduce maternal morbidity by pulmonary aspiration, the practice of withholding food and fluids to women in labor continues to be in practice till now.

27.5 Aspiration as a Contributor of Pregnancy-Related Mortality

To know the extent of aspiration as a cause of maternal death is very pertinent at this juncture. Hawkins et al. analyzed data from CDC during the

period 1979–2002 which reported 5946 pregnancy-related deaths [30]. Out of the 5946 cases of maternal mortality, 56 were attributed to administration of anesthesia. It was observed that there was a decreasing trend in the case fatality rate for general anesthesia. In the period 1991–1996, the incidence of anesthesia-related mortality was 16.8 for every 1 million general anesthesia administered, while it had reduced to 6.5 for every 1 million general anesthesia in the period 1997–2002. The decrease in the fatality rate has been suggested due to increased standards of care and improvements in anesthesia.

In a detailed analysis of maternal mortality in the USA between the period 1998 and 2005, Berg et al. [31] found 14.5 deaths per 100,000 live births (12.0 per 100,000 live births in 1998 to 16.8 per 100,000 live births in 2005). This was higher than the previous 20 years of the Pregnancy Mortality Surveillance System. It was also noted that there was a change in the trend. While a decline in maternal mortality due to hemorrhage and hypertensive disorders was seen, an increase was noted in maternal mortality due to medical conditions, particularly cardiovascular. The following causes each contributed to 10–13% of maternal deaths—hemorrhage, infection, thrombotic pulmonary embolism, hypertensive disorders, cardiomyopathy, cardiovascular conditions, and non-cardiovascular medical conditions. During the period 2003–2005, specific check boxes were introduced to mark the cause of maternal mortality. The study proposed that the increase in maternal mortality reported could be due to improved methods to identify pregnancy-related deaths and determine the cause of mortality. The interesting fact noted in this population study was that anesthesia-related maternal mortality was the lowest—1.2%. A recent Cochrane review by Paranjothy et al. [32], concluded that medications to increase gastric pH and gastric emptying have been found to be effective in women undergoing general anesthesia for cesareans.

Various studies that have been published in this area have identified the following as risk factors of aspiration in cases of anesthesia-related deaths, namely, obesity, poor preoperative general condition, emergency procedures, hypertension, embolism, and hemorrhage [26, 27, 33]. These studies can help to make an algorithm to identify patients at risk and provide individualized management.

27.6 Safety

27.6.1 For the Mother

27.6.1.1 Mortality

Obstetrical anesthesia has advanced considerably since the 1940s. Though there have been marked improvements in drugs and monitoring in general anesthesia, in obstetric scenario, there has been definitive shift from general anesthesia to regional anesthesia. The present scenario is different to the era of Mendelson when (a) women were encouraged to eat heavy meals during labor, (b) anesthetic techniques were also different from present-day modern anesthesiology, and (c) anesthesia was administered by untrained doctors.

Confidential inquiry into maternal mortality in the UK, in which every maternal death from the 1950s was investigated on individual basis, has not shown any link between maternal mortality and women eating and drinking in labor. In another study, the majority of maternal deaths were said to be associated with older anesthetic techniques and inexperienced practitioners between the 1960s and the 1990s [34].

27.6.1.2 Excessive Fluids

While solid food restriction has been in vogue for women in labor, water-only diet has been allowed. From the studies that have already been quoted in this chapter, it has been shown that excessive water (around 7–8 L) intake in women in labor has adverse effects on the mother and baby; some newer case reports have even reported hyponatremia in babies [35]. Obstetricians, midwives, and all involved in antenatal care of women need to be aware of this risk and make parents aware of it.

27.6.2 For the Baby

No difference in Apgar scores or NICU admissions in babies was noted between women who were eat-

ing and drinking in labor and women who were on water-only or carbohydrate drinks. Hence, there is insufficient data to conclude on the impact of maternal intake on the outcomes of the baby [18].

27.7 Current Practice

Hawkins et al. in [36] surveyed 740 US hospitals regarding the hospitals' policy on oral intake during labor. They found that in most hospitals, clear fluids were given during latent phase of labor, but 6% of hospitals had nil oral policies. During the active phase, women were restricted to only sips of fluid or ice chips, but 18% of hospitals had nil oral policies. Only 8% of hospitals had policies that permitted women in labor to have food during the latent phase. Almost all hospitals were found to have nil oral policies during active phase of labor.

Britain, Australia, and the Netherlands were found to have more liberal policies in regard to food intake during labor. A survey of the prevalent policies in England and Wales found that 96% of maternity units allowed oral intake. 67.2% of hospitals allowed fluids only, and 32.8% gave fluids and food [37]. While in New South Wales, Australia, 60.5% of women in labor were allowed food and fluid [38]. In the Netherlands, 67% of midwives and 73% of obstetricians allowed women in labor to choose food or fluid of their choice during labor. Survey of all births and cesarean births between the period 1983 and 1992 in the Netherlands found that the rate of aspiration which led to maternal mortality was as low as 0.018 per 1000 cesarean births and 0.001 per 1000 among all births. Interestingly, the rate of fatal aspiration was the same in the more liberal Netherlands as compared to the more restrictive USA and Great Britain [39].

27.8 What Women Want?

During early labor, there are elevated levels of prolactin which stimulates maternal appetite, but, with progression of labor, increase in oxytocin secretion reduces the desire to eat [40]. This would comprise the normal physiological changes related to appetite in labor.

In the context of woman-centered care, it would be important to know how women feel about intake restriction during labor. What is the pattern of eating and drinking women would prefer while undergoing one of the most important stages of their life? There are many surveys and trials which have attempted to address these questions. Safety of liberalized policies has been the core of such surveys and studies. Frye in 1994 suggested that eating in labor makes the woman feel normal and healthy, by helping her to be energetic, thus avoiding exhaustion. The psychosocial aspect of fasting should definitely be considered. Recent surveys have suggested that women preferred the process of labor to be comfortable and were not happy by the restriction of food and drink [41].

There have been winds of change. Surveys in the early 1980s in the UK and the USA revealed that not many consultants were averse to allowing women in labor to have food and fluids. But few surveys in 1990 and later have shown that some sort of food and fluids, usually water, was allowed during labor [9]. There is no strong evidence to show that liberalization of food intake policy during labor has compromised the safety of women in labor in the UK.

27.9 Current Recommendation

The American Society of Anesthesiologists currently does not recommend fasting in low-risk patients. They have stated that as parturient aspiration rates are nearly extinct due to advancements in anesthesia practice, identification of high-risk populations becomes essential. Early epidural placement in patients with preeclampsia and eclampsia and in obese patients is suggested [42]. ASA has suggested that restrictions should be on "case-to-case basis" depending on each patient's at-risk factors for aspiration and in women who are more likely to have operative birth. The American Society of Anesthesiologists recommends complete restriction of solid foods in women in labor.

The statement of the American College of Obstetricians and Gynecologists was published in 2009–2015. It states that those patients who are undergoing cesarean delivery but without any complications may have some amount of clear

liquids up to 2 h before induction of anesthesia. However, AJOG recommended against particulate-containing fluids. The recommendation of AJOG is similar to ASA in determining intake restriction in women at risk. It also stated that since there was insufficient evidence on the safe period of fasting after solid intake, any patient posted for elective cesarean delivery or elective postpartum tubal ligation, a minimum preoperative fasting for 6–8 h was recommended. As it is difficult to predict non-elective procedures such as cesarean section, it was suggested to avoid solid foods in laboring patients [43]. Another recent study also recommended that women in labor with low risk of aspiration or operative birth may be given solid food basically due to advancements in anesthesia [44].

The Cochrane review on the same subject in 2013 included five studies involving 3130 women. There were four comparisons with associated data and 41 meta-analyses. The conclusion after analysis of the included studies was thus:

- Restriction of food and fluids during labor does not have any benefit nor does it increase risk in low-risk women.
- Differences in rates of abdominal delivery, operative vaginal birth, and 5-minute Apgar score were not statistically significant between various studies or placebo groups.
- No case of regurgitation was reported in women in whom general anesthesia was administered for operative birth, in any of the studies included in the Cochrane review.
- Most studies did not study maternal experience, expectation, and hypoglycemia.

27.10 Conclusion

It can be stressful for women in labor to have restriction on their oral intake. Apart from feeling tired during the process, fasting during labor also has a psychological impact on the mothers-to-be. Moreover, an intervention such as restriction of food and fluids during labor should have adequate evidence, more so in this era of modern anesthesiology. On the other hand, to have liberal policies that allow food and fluid during labor also should have foolproof evidence in regard to its safety to the mother and newborn. Safety of the mother-to-be has always has been and should always remain the top priority.

It would be best to evaluate women in labor on a case-to-case basis to assess those at increased risk for cesarean birth and for factors that increase their risk of aspiration or difficult intubation. Now is probably the best time to revisit the controversy based on evidence available and establish guidelines on restriction of intake during different phases of labor.

References

1. DeLee J. Obstetrics for nurses. 5th ed. Philadelphia, PA: W. B. Saunders; 1918.
2. Mendelson CL. The aspiration of stomach contents into the lungs during obstetric anesthesia. Am J Obstet Gynecol. 1946;52:191–205.
3. Micklewright A, Champion P. Labouring over food: the dietician's view. In: Champion P, McCormick C, editors. Eating and drinking in labour. 1st ed. Oxford: Books for Midwives; 2002. p. 29–45.
4. Pengelley L, Gyte G. Eating and drinking in labour (V). An update of the NCT briefing paper. Pract Midwife. 1998a;1(12):26–9.
5. Pengelley L, Gyte G. Eating and drinking in labour (I). A summary of medical research to facilitate informed choice about the care of mother and baby. Pract Midwife. 1998b;1(7–8):34–7.
6. Pengelley L, Gyte G. Eating and drinking in labour (II). A summary of medical research to facilitate informed choice about the care of mother and baby. Pract Midwife. 1998c;1(9):27–9.
7. Pengelley L, Gyte G. Eating and drinking in labour (III). A summary of medical research to facilitate informed choice about the care of mother and baby. Pract Midwife. 1998d;1(10):19–21.
8. Pengelley L, Gyte G. Eating and drinking in labour (IV). A summary of medical research to facilitate informed choice about the care of mother and child. Pract Midwife. 1998e;1(11):24–6.
9. Hart D. Eating and drinking during labour. In: Hall Moran V, Dykes F, editors. Maternal and infant nutrition and nurture: controversies and challenges. London: Quay Books; 2006. p. 102–27.
10. O'Sullivan G, Hart D, Shennan A. A rational approach to aspiration prophylaxis. In: Halpern S, Douglas J, editors. Evidence-based obstetric anaesthesia. Oxford: BMJ Books; 2005. p. 178–91.
11. Eliasson AH, Phillips YY, Stajduhar KC, Carome MA, Cowsar JD Jr. Oxygen consumption and ventilation during normal labor. Chest. 1992;102(2):467–71.
12. Maheux PC, Bonin B, Dizazo A, Guimond P, et al. Glucose homeostasis during spontaneous labor in

normal human pregnancy. J Clin Endocrinol Metab. 1996;81(1):209–15.
13. Chang S. The psychological and physiological effects of ketonuria during labour [thesis]. Michigan: University of Michigan; 1993.
14. Dumoulin JG, Foulkes JE. Ketonuria during labour. Br J Obstet Gynaecol. 1984;91(2):97–8.
15. Kubli M, Scrutton MJ, Seed PT, et al. An evaluation of isotonic 'sport drinks' during labor. Anesth Analg. 2002;94(2):404–8.
16. Toohill J, Soong B, Flenady V. Interventions for ketosis during labour. Cochrane Database Syst Rev. 2008;(3):CD004230.
17. Scrutton MJ, Metcalfe GA, Lowy C, et al. Eating in labour: a randomised controlled trial assessing the risks and benefits. Anaesthesia. 1999;54(4):329–34.
18. Scheepers HC, Thans MC, de Jong PA, et al. A double-blind, randomised, placebo controlled study on the influence of carbohydrate solution intake during labour. BJOG Int J Obstet Gynaecol. 2002;109(2):178–81.
19. Scheepers HC, de Jong PA, Essed GG, Kanhai HH. Carbohydrate solution intake during labour just before the start of the second stage: a double-blind study on metabolic effects and clinical outcomes. BJOG. 2004;111(12):1382–7.
20. O'Sullivan G, Liu B, Hart D, et al. Effect of food intake during labour on obstetric outcome: randomised controlled trial. Br Med J. 2009;338:b784.
21. Kardel KR, Henriksen T, Iversen PO. No effect of energy supply during childbirth on delivery outcomes in nulliparous women: a randomised, double-blind, placebo-controlled trial. J Obstet Gynaecol. 2010;30(3):248–52.
22. Parsons M, Bidewell J, Nagy S. Natural eating behavior in latent labor and its effect on outcomes in active labor. J Midwifery Womens Health. 2006;51(1):e1–6.
23. Tranmer JE, Hodnett ED, Hannah ME, et al. The effect of unrestricted oral carbohydrate intake on labor progress. J Obstet Gynecol Neonatal Nurs. 2005;34(3):319–28.
24. Blackburn S. Maternal, fetal, and neonatal physiology: a clinical perspective. 3rd ed. Philadelphia, PA: Saunders; 2007.
25. Providing oral nutrition to women in labor. J Midwifery Womens Health. 2016;61(4):528–34. https://doi.org/10.1111/jmwh.12515.
26. Smith I, Kranke P, Murat I, et al. Perioperative fasting in adults and children: guidelines from the European Society of Anaesthesiology. Eur J Anaesthesiol. 2011;28(8):556–69.
27. American Society of Anesthesiologists Committee. Practice guidelines for preoperative fasting and the use of pharmacologic agents to reduce the risk of pulmonary aspiration: application to healthy patients undergoing elective procedures: an updated report by the American Society of Anesthesiologists Committee on standards and practice parameters. Anesthesiology. 2011;114(3):495–511.
28. Singata M, Tranmer J, Gyte GM. Restricting oral fluid and food intake during labour. Cochrane Database Syst Rev. 2013;(8):CD003930.
29. Ng A, Smith G. Gastroesophageal reflux and aspiration of gastric contents in anesthetic practice. Anesth Analg. 2001;93(2):494–513.
30. Hawkins JL, Chang J, Palmer SK, Gibbs CP, Callaghan WM. Anesthesia-related maternal mortality in the United States: 1979–2002. Obstet Gynecol. 2011;117(1):69–74.
31. Berg CJ, Callaghan WM, Henderson Z, Syverson C. Pregnancy-related mortality in the United States, 1998 to 2005. Obstet Gynecol. 2010;116(6):1302–9.
32. Paranjothy S, Griffiths JD, Broughton HK, Gyte GM, Brown HC, Thomas J. Interventions at caesarean section for reducing the risk of aspiration pneumonitis. Cochrane Database Syst Rev. 2014;(2):CD004943.
33. Mhyre JM, Riesner MN, Polley LS, Naughton NN. A series of anesthesia-related maternal deaths in Michigan, 1985–2003. Anesthesiology. 2007;106(6):1096–104.
34. Lewis G, Drife J. Why mothers die 2000–2002: the sixth report of the confidential enquiries into maternal deaths in the United Kingdom. London: RCOG Press; 2004. www.cemach.org.uk/publications/WMD2000_2002/content.htm
35. Johansson S, Lindow S, Kapadia H, et al. Perinatal water intoxication due to excessive oral intake during labour. Acta Paediatr. 2002;91(7):811–4.
36. Hawkins JL, Gibbs CP, Martin-Salvaj G, Orleans M, Beaty B. Oral intake policies on labor and delivery: a national survey. J Clin Anesth. 1998;10(6):449–51.
37. Michael S, Reilly CS, Caunt JA. Policies for oral intake during labour. A survey of maternity units in England and Wales. Anaesthesia. 1991;46(12):1071–3.
38. Parsons M. Policy or tradition: oral intake in labour. Aust J Midwifery. 2001;14(3):6–12.
39. Scheepers H, Essed G, Brouns F. Aspects of food and fluid intake during labour. Policies of midwives and obstetricians in the Netherlands. Eur J Obstet Gynecol Reprod Biol. 1998;78(1):37–40.
40. McNabb M. Changes in maternal food appetite and metabolism in labour and the shift from fetal to neonatal metabolism. In: Champion P, McCormick C, editors. Eating and drinking in labour. Oxford: Books for Midwives; 2002. p. 46–110.
41. Iravani M, Zarean E, Janghorbani M, Bahrami M. Women's needs and expectations during normal labor and delivery. J Educ Health Promot. 2015;4:6.
42. American Society of Anesthesiologists. Practice guidelines for obstetric anesthesia: an updated report by the American society of anesthesiologists task force on obstetric anesthesia and the society for obstetric anesthesia and perinatology. Anesthesiology. 2016;124(2):270–300.
43. American College of Obstetricians and Gynecologists. Oral intake during labor ACOG Committee opinion no. 441. Obstet Gynecol. 2009;114:714.
44. Sperling JD, Dahlke JD, Sibai BM. Restriction of oral intake during labor: whither are we bound? Am J Obstet Gynecol. 2016;214:592–6.

Meconium

28

Yogita Dogra

28.1 Introduction

Meconium is the first intestinal discharge from newborns. It is a viscous, dark-green material composed of intestinal epithelial cells, mucus, lanugo and intestinal secretions (e.g. bile). The characteristic colour results from bile pigments, especially biliverdin. It also contains undigested debris from swallowed amniotic fluid. Meconium is sterile which differentiates it from stool.

Obstetrical teaching conventionally viewed meconium passage as a potential warning of fetal asphyxia. Moreover obstetricians have also long realized the prognostic dilemma of meconium. It occurs mostly in term and post-term pregnancies. It may be associated with fetal compromise but is also common in normal labours.

The meconium staining has been graded as:

(a) Thick—viscous, tenacious containing large amount of particulate material.
(b) Thin—fluid is normal except for greenish colour.
(c) Moderate—if it is thicker and darker in colour.

Y. Dogra (✉)
Department of Obstetrics and Gynecology,
All India Institute of Medical Sciences (AIIMS),
New Delhi, India

Meconium-stained liquor (MSL) has also been classified by visual examination after spontaneous or artificial rupture of membranes as:

(a) Grade I—MSL is translucent, light yellow-green in colour.
(b) Grade II—MSL is opalescent with deep green and light yellow in colour.
(c) Grade III—MSL is opaque and deep green in colour.

Thick MSL but not thin is associated with poor perinatal outcome [1, 2].

28.2 Incidence

Fetal passage of meconium before or during labour is common with incidence ranging from 12 to 20%. The incidence during labour increases with gestational age also—30% at 40 weeks and 50% at 42 weeks [3]. Presence of meconium below vocal cord is known as meconium aspiration. It occurs in 20–30% of all infants with meconium with approximately 12% mortality [4].

28.3 Pathophysiology

Three theories have been proposed to explain meconium passage by fetus:

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1. Pathological explanation proposes that fetus pass meconium when hypoxia stimulates arginine vasopressin (AVP) release from fetal pituitary gland. AVP stimulates colonic smooth muscle to contract, resulting in intraamniotic defecation.
2. Physiological explanation—meconium passage represents normal gastrointestinal tract maturation under neural control.
3. Final theory suggests that meconium passage follows vagal stimulation from common but transient umbilical cord compression with resultant increased bowel peristalsis.

The effects of meconium in amniotic fluid are well reported [5]. Meconium decreases the antibacterial activity of amniotic fluid by altering levels of zinc which subsequently increases the risk of perinatal bacterial infection. Then, meconium acts as irritant to fetal skin and thus increases the incidence of erythema toxicum. Aspiration of meconium is the most severe complication before, during and after birth. It induces hypoxia via four major pulmonary effects: airway obstruction, pulmonary hypertension, chemical pneumonitis and surfactant dysfunction.

28.4 Causes

Risk factors promoting the passage of meconium in utero include the following:

Maternal risk factors:

- Preeclampsia and eclampsia.
- Placental insufficiency.
- Gestational diabetes mellitus.
- Post-term pregnancy.
- Maternal chronic respiratory or cardiovascular diseases.
- Drug abuse, especially tobacco and cocaine.
- Chorioamnionitis/maternal infection.

Fetal risk factors:

- Oligohydramnios.
- Intrauterine growth restriction (IUGR).
- Poor biophysical profile.

28.5 Complications

Moderate and thick meconium is associated with meconium aspiration syndrome, increased risk of birth asphyxia, increased operative interference, low Apgar scores, decreased umbilical cord pH and overall increased perinatal mortality. However, thin MSL is associated with low risk of perinatal complications.

MSL has also been associated with increased rate of admission to neonatal intensive care unit (NICU), cerebral palsy, neonatal sepsis and seizures [6–8]. Moreover, it is cited that the presence of MSL is linked with intrapartum chorioamnionitis [9] and postpartum endometritis [10].

Children with meconium aspiration syndrome may develop chronic lung disease from intense pulmonary intervention.

Infants with meconium aspiration syndrome have a slightly increased incidence of respiratory tract infections in the first year of life because the lungs are still in recovery phase.

28.6 Identification and Management of MSL

Antenatal identification of women at risk for meconium passage in utero is important so that intrapartum surveillance can be improved. Once meconium is identified during labour, close monitoring of the fetus clinically or with CTG becomes obligatory. The woman should be informed of the significance of MSL. A risk assessment should be done to include the stage of labour (per vaginal examination), parity, whether the meconium staining is significant or light and current fetal wellbeing. Presence of meconium in absence of fetal heart rate abnormalities is not always indicative of fetal compromise [11]. The woman is nursed in left lateral position with oxygen inhalation. Hydration is maintained. After the initial hypoxic insult initiating the passage of meconium, subsequent repetitive episodes due to prolonged labour or abnormal uterine activity may cause severe asphyxia [12]. Such repetitive episodes can be avoided by vigilant fetal monitoring, active management of labour and optimal

care after birth. This would prevent unnecessary caesarean sections in all cases of meconium-stained liquor in the absence of a definitive indication.

Thin MSL—If no fetal heart rate abnormalities, active management of labour is done. Caesarean section is indicated if abnormal CTG and other obstetric reasons, if any.

Thick MSL—Suggest prompt intervention, need for presence of skilled paediatrician at the time of delivery and need for intensive care in the neonatal period to give a positive outcome.

Pre-labour rupture of membranes—Any woman reporting to labour room with spontaneous rupture of membranes with meconium staining should be advised admission for assessment. If MSL is confirmed, continuous electronic fetal monitoring (CEFM) should be commenced, and a plan is made for mode of delivery according to department protocols.

Low-risk intrapartum woman in the community setting—If during labour, MSL becomes evident, a risk assessment should be undertaken including transfer time. If transfer to a unit with neonatal facilities can be achieved before delivery, the woman should be advised to transfer, by ambulance. If birth is expected before transfer can be facilitated, preparations should be made for resuscitation of the newborn and ambulance for transfer of the baby, following birth.

28.7 Prevention of Meconium Aspiration Syndrome (MAS)

Prevention of MAS is paramount. Fetal status should be closely monitored in an attempt to identify fetal distress.

28.7.1 Role of Amnioinfusion

Amnioinfusion is theoretically beneficial to dilute meconium and thus reduce the risk and severity of meconium aspiration. Warm saline or Ringer's lactate is infused transcervically through a catheter or infant feeding tube into the uterine cavity or transabdominally through a spinal needle when membranes are intact. Thick meconium suggests oligohydramnios, as meconium passed into a normal volume of amniotic fluid will usually appear thin. Amnioinfusion may therefore correct oligohydramnios, relieving umbilical cord compression.

Nonetheless, current evidence does not support routine amnioinfusion to prevent meconium aspiration syndrome [13–15].

28.7.2 Role of Antibiotics

There is currently no evidence to support the routine administration of antibiotics during labour to women with MSL.

28.8 Management of Baby Born Through MSL

- Resuscitation equipment should be checked prior.
- Paediatrician should be called for delivery.
- Current recommendations no longer advise routine intrapartum suctioning for infants born to mothers with MSL [16, 17].
- When meconium aspiration occurs, intubation and immediate airway suctioning can remove much of the aspirated meconium.
- There are no clinical trials to justify suctioning of airway based on the consistency of meconium.
- Do *not* perform the following in an attempt to prevent aspiration:
  - Squeezing the chest of the baby.
  - Inserting a finger into the baby's mouth.
- The American Academy of Pediatrics Neonatal Resuscitation Program Steering Committee and the American Heart Association have published the guidelines for management of the baby exposed to meconium. The guidelines are as follows [18]:
  - If the baby is not vigorous (defined as depressed respiratory effort, poor muscle tone and/or heart rate <100 beats/min): Use direct laryngoscopy, intubate, and suction the trachea immediately after delivery.

Suction for no longer than 5 s. If meconium is not retrieved, do not repeat intubation and suction. If meconium is retrieved and no bradycardia is present, reintubate and suction. If the heart rate is low, administer positive pressure ventilation and consider suctioning again later.
  - If the baby is vigorous (defined as normal respiratory effort, normal muscle tone and heart rate >100 beats/min): Do not electively intubate. Clear secretions and meconium from the mouth and nose with a bulb syringe or a large-bore suction catheter. Injury to the vocal cords is more likely to occur when an attempt is made to intubate a vigorous newborn.
  - In both cases, the rest of the initial resuscitation steps should ensue, including drying, stimulating, repositioning and administering oxygen as necessary.
- All observations must be documented in a timely manner.
- If the baby's condition causes concern at any time, a review by the neonatal team should be requested and baby shifted to NICU.

28.9 Conclusion

Meconium-stained liquor by itself is not associated with an adverse neonatal outcome. Most of the babies remain asymptomatic and need only routine care. Association of MSL with abnormal CTG is associated with poor outcome, increased caesarean section rate and increased neonatal complications.

References

1. Mahomed K, Nyoni R, Masona D. Meconium-staining of the liquor in a low-risk population. Paediatr Perinat Epidemiol. 1994;8:292–300.
2. Ziadeh SM, Sunna E. Obstetric and perinatal outcomes of pregnancies with term labour and meconium-stained amniotic fluid. Arch Gynecol Obstet. 2000;264:84–7.
3. Steer PJ, et al. Fetal distress in labour, high risk pregnancy management options. 3rd ed. Philadelphia, PA: Elsevier INC; 2006. p. 1450–72.
4. Khatun MHA, et al. Fetal outcome in deliveries with MSL–Bangladesh. J Child Health. 2009;33(2):41–50.
5. Singh BS, Clark RH, Powers RJ, Spitzer AR. Meconium aspiration syndrome remains a significant problem in the NICU: outcomes and treatment patterns in term neonates admitted for intensive care during a ten-year period. J Perinatol. 2009;29(7):497–503.
6. Berkus MD, Langer O, Samueloff A. Meconium-stained amniotic fluid: increased risk for adverse neonatal outcome. Obstet Gynecol. 1994;84:115–0.
7. Katz VL, Bowes WA. Meconium aspiration syndrome: reflection on a musky subject. Am J Obstet Gynecol. 1992;166:171–83.
8. Nathan L, Leveno KJ, Carmody TJ, Kelly MA, Sherman ML. Meconium: a 1990s perspective on an old obstetric hazard. Obstet Gynecol. 1994;83:329–32.
9. Romero R, Hanaoka S, Moshe M, et al. Meconium-stained amniotic fluid: a risk factor for microbial invasion of the amniotic cavity. Am J Obstet Gynecol. 1991;164:859–62.
10. Josephson A. An epidemiologic study of postcesarean infection. Am J Infect Control. 1984;12:19–25.
11. Miller FC. Meconium staining of amniotic fluid. Clin Obstet Gynaecol. 1975;121:45–50.
12. Fujikura T, et al. The significance of meconium staining. Am J Obstet Gynaecol. 1975;121:45–50.
13. ACOG Committee Obstetric Practice. ACOG Committee no. 346: amnioinfusion does not prevent meconium aspiration syndrome. Obstet Gynecol. 2006;108(4):1053–5.
14. Velaphi S, Vidyasagar D. Intrapartum and postdelivery management of infants born to mothers with meconium-stained amniotic fluid: evidence-based recommendations. Clin Perinatol. 2006;33(1):29–42.
15. Hofmeyr GJ, Xu H. Amnioinfusion for meconium-stained liquor in labour. Cochrane Database Syst Rev. 2010;(1):CD000014.
16. Committee on Obstetric Practice, American College of Obstetricians and Gynecologists. ACOG committee opinion no. 379: management of delivery of a newborn with meconium-stained amniotic fluid. Obstet Gynecol. 2007;110(3):739.
17. Vain NE, Szyld EG, Prudent LM, Wiswell TE, Aguilar AM, Vivas NI. Oropharyngeal and nasopharyngeal suctioning of meconium-stained neonates before delivery of their shoulders: multicentre, randomised controlled trial. Lancet. 2004;364(9434):597–602.
18. Kattwinkel J, Perlman JM, Aziz K, Colby C, Fairchild K, Gallagher J, Hazinski MF, Halamek LP, Kumar P, Little G, McGowan JE, Nightengale B, Ramirez MM, Ringer S, Simon WM, Weiner GM, Wyckoff M, Zaichkin J. Neonatal resuscitation: 2010 American heart association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation. 2010;122:S909–19.

Part IV

Delivery

Episiotomy

29

Manishi Mittal

29.1 Introduction

Episiotomy is a surgical incision given at the perineum during the second stage of labour, in order to enlarge the vaginal orifice and facilitate delivery of the baby. The earliest description of episiotomy can be dated back to 1741, when Ould described "an incision made towards the anus with a pair of crooked probe-scissors introducing one blade between the head and the vagina, as far as shall be thought necessary" [1].

Large disparity has been reported between rates of episiotomy used throughout the world varying from 9.7% (Sweden) to 30% (Europe) [2] to 100% (Taiwan) [3]. In the USA, episiotomy use has decreased from 62.50% in 1983 [4] to 30–35% in 2003 [5]. Few studies describing rates of episiotomy in institutions or otherwise are available from India. In a cross-sectional study performed in Karnataka, including 3595 women, rate of episiotomy was observed to be 23.5% [6]. In another study conducted at Chennai, episiotomy rate was found to be 67%. The investigators observed that doctors had more predilection to conduct episiotomy (77.4%) as compared to nurses (53.1%) or trained birth attendants (5%). Moreover, it was higher in tertiary care centres. Instrumental deliveries and primiparae were seen to be high risk factors for episiotomy [7].

Evidence promotes selective episiotomy over routine. Many authors recommend that rate of episiotomy should not be more than 30% of vaginal deliveries [8].

29.2 Technique

During the second stage of labour, if there is insufficient space for the head, or the perineum is rigid, perineal tear may occur. To prevent this, an episiotomy is given.

Reasons for popularity of episiotomy:

- Smooth and easier to repair surgical incision, instead of laceration.
- Postoperative pain was thought to be less, with early healing. This belief, however, was found to be incorrect [9].
- Preservation of muscle tone with maintained sexual function and a decreased risk of prolapse and faecal/urinary incontinence [10].
- Lesser chance of third-degree tears.
- Shortened second stage of labour—less fetal asphyxia and cranial trauma.
- More space for instrumental deliveries or if rotation manoeuvres are required, e.g. in shoulder dystocia.

M. Mittal (✉)
Mittal Maternity and Superspeciality Hospital, Yamunanagar, Haryana, India

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29.2.1 Timing

Too early incision causes excessive bleeding from site of cut, while delayed one will be unable to prevent any lacerations. Usually, the episiotomy is performed just before crowning during contraction, when the fetal head is visible up to a diameter of 3–4 cm. Incision is given when the perineum is stretched at the height of contraction. If performed with forceps delivery, incision is usually given after application of blades.

29.2.2 Analgesia

The latest National Institute of Health and Care Excellence (NICE) guidelines recommend that appropriate analgesia should be given before performing episiotomy, though it may be deferred in case of fetal distress [11]. Local lignocaine (10 mL of 1% solution) application in the subcutaneous tissue before incision is the most commonly used method (Fig. 29.1).

Pudendal nerve block: It has the advantage of minimal blood loss and no fetal depression. It is given transvaginally by injection of 10 mL of 1% lignocaine on each side into the pudendal nerve, where it nears the ischial spine, by going through the sacrospinous ligament. Aspiration of the syringe to check for inadvertent entry into the pudendal artery should be done before injecting.

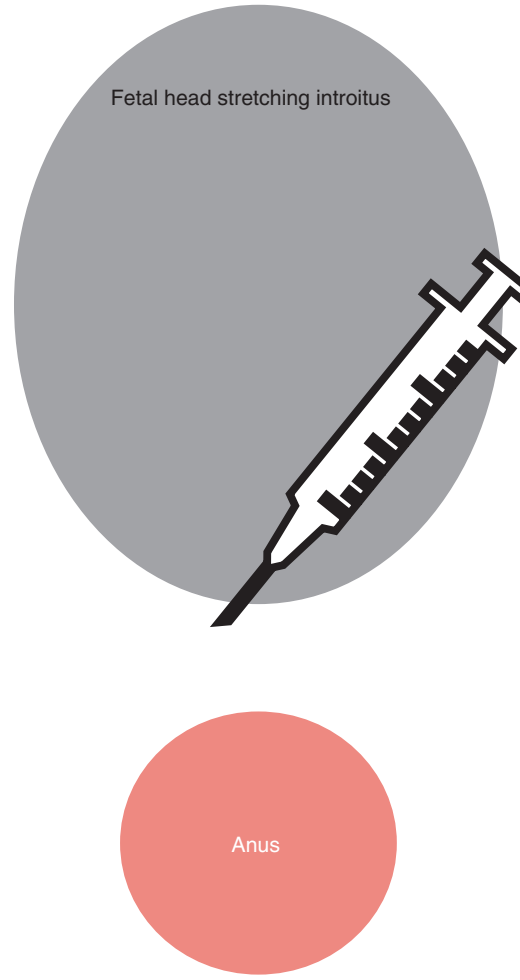

Fig. 29.1 Two fingers are introduced between the fetal head and vaginal wall. Lignocaine solution is introduced into the subcutaneous tissue starting from the posterior fourchette, after aspiration to check for infiltration into blood vessels

29.2.3 Types of Episiotomy (Fig. 29.2):

1. Median (midline or medial) episiotomy

   This type starts from the posterior fourchette and extends along the midline posteriorly, covering around half of the length of the perineum. The angle with the midline remains between 0° and 25°.
2. Mediolateral episiotomy

   This term most commonly identifies the incision originating from the posterior fourchette (within 3 mm of the midline) and extending laterally and downwards away from the rectum at an angle of 45–60° from the midline. However, the mediolateral episiotomy is defined in a wide variety of ways in different obstetric textbooks.
3. Lateral episiotomy

   This episiotomy originates 1 or 2 cm lateral to the midline in the introitus, going towards the ischial tuberosity. It is usually longer than other episiotomies. Lateral episiotomy is not preferred, because of many adverse effects like injury to Bartholin's duct. However, it is said to be more common than documented as often inaccurately given mediolateral episiotomy becomes lateral.

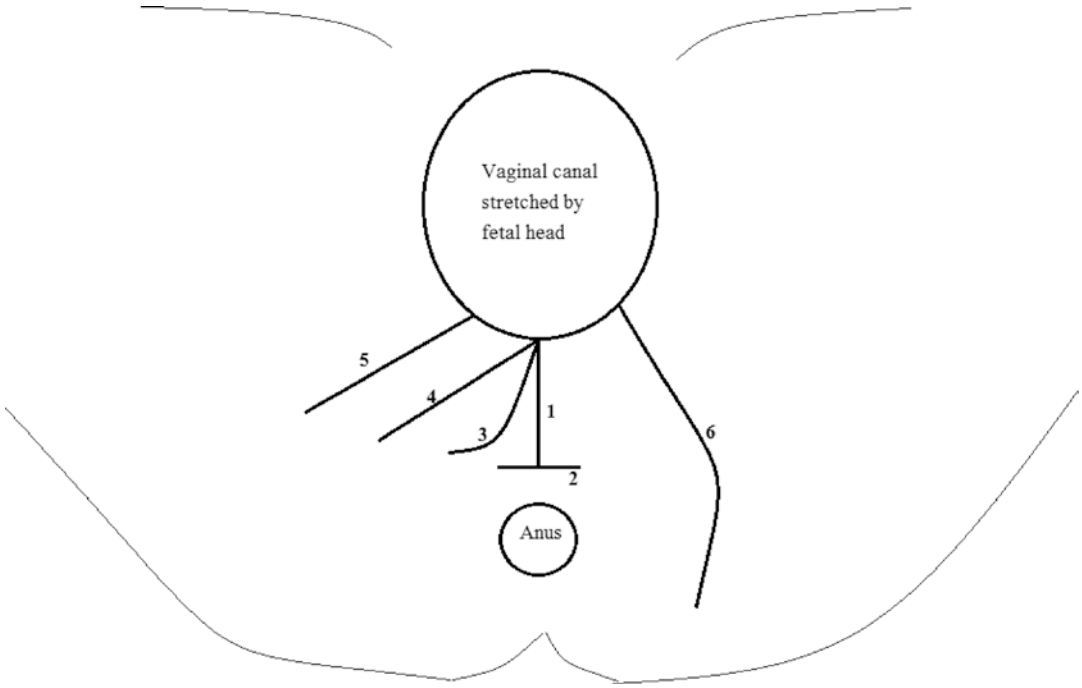

Fig. 29.2 Types of episiotomy. (1) Median episiotomy, (2) modified median episiotomy, (3) "J"-shaped episiotomy, (4) mediolateral episiotomy, (5) lateral episiotomy, (6) radical lateral (Schuchardt incision)

4. J-shaped episiotomy

   This episiotomy starts as a midline incision, then curving laterally 2–5 cm away from the anus towards the ischial tuberosity. Curved scissors are used for this procedure.
5. Modified median episiotomy

   It is a modification of the midline episiotomy. A transverse incision is added to each side of the midline episiotomy (total measuring 2–5 cm) just anterior to the expected location of the anal sphincter. This technique was said to increase the outlet diameter by 83% [12]. However, it is not a popular technique.
6. Radical lateral episiotomy (Schuchardt incision)

   This procedure is not common in obstetrics. The incision goes deep into a vaginal sulcus and then curves downwards and laterally around the rectum. It provides access to the parametrium in radical vaginal hysterectomy or trachelectomy and allows removal of a neglected vaginal pessary. Rarely, it may be used in difficult labour (large head, difficult breech, or shoulder dystocia) [13].
7. Anterior episiotomy

   For women with a history of infibulation (closure of the vaginal vestibule by fusion of the labia majora, done in some cultures to prevent intercourse), anterior episiotomy or deinfibulation is required during delivery. The obstetrician's finger is inserted through the introitus and directed towards the pubis. The scar is corrected by incising the fused labia till the external urethral meatus is visible. Clitoral remnants should not be incised. Additionally, mediolateral episiotomy may be necessary during delivery.

Despite the many advantages of median episiotomy, mediolateral episiotomy is preferred due to the important complication of the extension of episiotomy into the anal canal (Table 29.1). In a Dutch study involving more than 43,000 deliveries, a fourfold fall in severe perineal lacerations was found with mediolateral technique [15]. When performed, mediolateral episiotomy is given on a stretched perineum starting from the posterior fourchette. The

Table 29.1 Comparison of commonly used techniques [14]

Complications	Median episiotomy	Mediolateral episiotomy
Surgical repair	Easy	Difficult
Healing	Good	Occasionally faulty
Postoperative pain	Less	More
Restoration of anatomy	Good	May be inaccurate
Blood loss	Less	More
Sexual dysfunction	Rare	Occasional
Extensions into third- or fourth-degree tears	Common	Uncommon
Voluntary extension of incision	Not possible	Possible
Incision of muscles	Negligible	Muscles are cut
Injury to anal sphincter complex	More	Less

incision is preferably given at an angle of 45–60° to the right of the midline [11, 16]. A 60° incision angle is seen to be associated with lesser anal sphincter trauma, anal incontinence and perineal pain [17]. In another study, it was estimated that there is a 50% relative decrease in the occurrence of third-degree tears for every 6° angle away from the midline [18].

Structures cut during episiotomy:

1. Posterior vaginal wall
2. Muscles: Superficial and deep transverse perineal muscles, bulbospongiosus, part of levator ani
3. Transverse perineal branches of pudendal nerve and vessels
4. Subcutaneous tissue, fascia and skin

29.2.4 Episiotomy Repair

It is mostly performed after complete expulsion of the placenta, spontaneous or assisted. Complete aseptic precautions should be taken. Repair is performed with patient in lithotomy position. Most commonly used suture material is 1-0 or 2-0 chromic catgut. Other suture materials have been recommended like polyglycolic acid derivatives, e.g. vicryl and vicryl rapide. Polyglycolic acid sutures have higher tensile strength, with smooth passage through tissue,

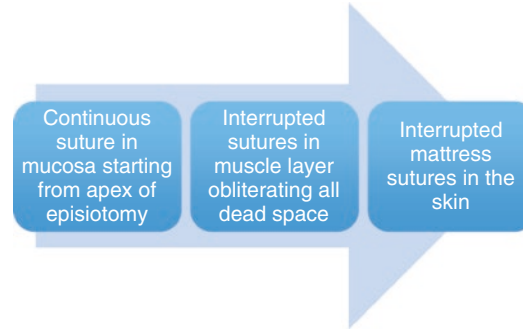

Fig. 29.3 Episiotomy is sutured in three layers

and are easy to handle with excellent knotting ability and secure knots. A review of 18 randomized controlled trials (RCTs) conducted on suture materials for episiotomy reported that synthetic absorbable sutures, when compared with catgut, had lesser postoperative pain, decreased use of analgesia, reduced wound breakdown and decreased requirement for re-suturing compared to catgut [19]. However, standard synthetic sutures (Vicryl) had to be removed more often than catgut or rapidly absorbed synthetic sutures. Use of catgut has been discontinued in most European countries, but, being comparatively cheaper, it is still being used in India. But when compared on a large scale, use of synthetic sutures appears to be less expensive in the long run rather than catgut with its associated morbidities.

Basic principles of episiotomy repair are correct reapproximation, proper haemostasis and suturing without tension. The episiotomy is usually stitched in three layers—the mucosa, muscle layer and skin (Fig. 29.3). Suturing of the mucosa is started 1 cm above the apex of the incision, wherein any retracted vessels are ligated. This step is very important to prevent the formation of haematoma. Continuous sutures are then applied till the hymenal ring, approximating the mucosa and submucosal tissue. After this, the underlying muscle layer is sutured in either interrupted or running manner. Care should be taken not to leave any dead space. After proper reapproximation of these layers, the perineal skin is sutured. This is done with subcuticular or interrupted mattress sutures. Some authors recommend complete incision to be closed in continuous fashion. An RCT by Kettle et al. in

1542 women showed that continuous suturing was associated with less perineal pain [20].

29.3 Postpartum Perineal Care

Application of cold compresses over the perineum, immediately after delivery, helps to reduce discomfort and oedema. Afterwards, the area of repair should be regularly cleaned with plain soap at least once or twice a day and after urination or defecation. Pain relief can be given with NSAIDs like diclofenac or ibuprofen. Cold sitz baths may provide additional pain relief by reducing excitability of nerve endings and decreased nerve conduction, along with local vasoconstriction. Local lignocaine ointment application was not found to be effective in reducing pain or discomfort [21]. Patient should be discharged only after proper inspection of the wound. Patient should not sit cross-legged.

If the woman complains of persistent or excessive pain, a complete examination should be done to identify a haematoma, infection or serious complications like angioedema, necrotizing fasciitis or perineal cellulitis. Haematoma usually becomes obvious few hours after delivery, while infection appears after 3 or 4 days.

Resumption of sexual intercourse early after delivery may be problematic due to incomplete healing. Moreover, breast-feeding can suppress oestrogen for long periods causing vaginal atrophy and dryness. No definite time period has been recommended, though 2 weeks is considered all right depending on the woman's desire and comfort. Routinely, the episiotomy wound is healed and almost asymptomatic by 3 weeks after delivery.

29.4 Complications of Episiotomy (Table 29.2)

29.4.1 Perineal Tears (Fig. 29.4)

Perineal tears are commonly seen in spontaneous vaginal deliveries and even when episiotomy is given. Risk for third- and fourth-degree lacerations is increased in cases of nulliparity, prolonged second stage of labour, persistent occipito-posterior position, instrumental delivery, use of local anaesthesia and Asian race [22]. However, for women living in Asia, no relation has been found between ethnicity and severe perineal trauma. The vast diversity of the term "Asian" is a confounding factor [23]. More local studies are necessary to estimate the risk in Indian women.

Though episiotomy was introduced as a method to prevent perineal tears, evidence suggests that this is not always true. The association between episiotomy and severe perineal tears has been found to be significant even in accepted indications such as macrosomia, instrumental deliveries, non-reassuring fetal heart rate patterns, occipito-posterior position and shoulder dystocia [24]. However, there is no proof of causality. Both episiotomies and severe perineal tears might be dependent on a third factor (such as parity or estimated fetal weight) rather than each other.

As assessed in a recent Cochrane review, restrictive use of episiotomy resulted in less severe perineal trauma (RR 0.67, 95% CI 0.49–0.91), less suturing (RR 0.71, 95% CI 0.61–0.81) and fewer healing complications (RR 0.69, 95% CI 0.56–0.85) as opposed to routine use. On the other hand, restrictive episiotomy caused more anterior perineal trauma (RR 1.84, 95% CI 1.61–2.10) [25].

First- and second-degree tears are easy to repair with suturing done in layers similar to episiotomy, after securing the apex. In third-degree perineal tears, the ends of the sphincter are identified by a dimple on the anal skin a little anterior to the anus. These ends are approximated, after securing with Allis forceps, with a figure-of-eight suture or suturing the fibrous sheath in interrupted fashion. In fourth-degree laceration, the mucosa should be repaired separately. The most common procedure is approximation of the

Table 29.2 Complications of episiotomy

Immediate	Delayed
Perineal tears	Dyspareunia
Vulval haematoma/haemorrhage	Scar endometriosis
Infection	Fistula
Wound dehiscence	
Obstetric anal sphincter injuries	
Necrotizing fasciitis	

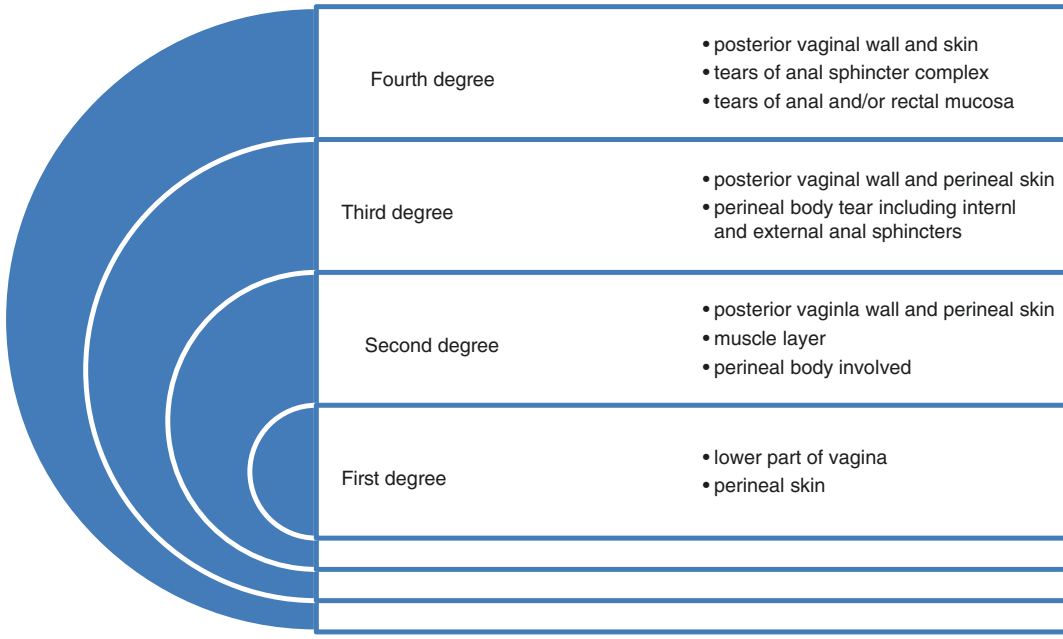

Fig. 29.4 Degrees of perineal lacerations

mucosal edges with chromic or polyglycolic sutures, followed by an overlapping second layer. The remaining tissues are repaired similar to a third-degree laceration. Careful and timely repair prevents fistula formation.

29.4.2 Haemorrhage

Postpartum haemorrhage (PPH) is an important cause of maternal morbidity and mortality. Around 20% of PPH is attributed to excessive bleeding from episiotomy or lacerations of the uterus, cervix, vagina or vulva [26].

If the episiotomy involves arteries or large varicosities, the bleeding is excessive. In addition, PPH may occur if the episiotomy is too large, or the time difference between incision and delivery, or between delivery and episiotomy repair, is too long. Suspicion of PPH from lacerations or episiotomy is kept if fresh haemorrhage is occurring even with a firm retracted uterus.

If blood vessels in the subcutaneous tissue are not properly ligated, concealed haemorrhage may lead to formation of a haematoma. As bleeding from the haematoma is concealed, it might not be recognized early, and the patient may present in shock after few hours.

PPH can be prevented by timely and adequate repair of the episiotomy, taking care that the apex is correctly identified and ligated. The whole birth canal should be examined to identify any other lacerations or bleeding points. After episiotomy repair, patient should be kept under observation with regular monitoring of vitals, bleeding per vaginum and perineal swelling for at least 2–3 h, for early recognition of PPH and haematoma.

In case of vaginal haematomas or large lacerations extending superiorly, the repair should be done in the operation theatre with adequate anaesthesia and fluid management. The haematoma should be completely evacuated and exploration done to identify the bleeding point, which is then securely ligated. Drainage of blood should be allowed by leaving the cavity open.

29.4.3 Infection

Infection of the episiotomy wound is not very common (0.5–3%) [26] in spite of the high rate of con-

tamination during delivery. This may be due to the excellent blood supply to the perineum. Infection is more common in large wounds as more tissue is devitalized. In addition, patients with pre-existing infection in the anogenital area are at higher risk. Women with third- or fourth-degree lacerations are also at higher risk for infection.

Episiotomy infection generally presents with pain and discomfort. Mass is not commonly formed as drainage occurs spontaneously. When examined, gaping of the wound is seen. Proper rectovaginal examination should be performed to assess the anal sphincter and presence of any fistula.

The infection is usually mixed in type with both aerobic and anaerobic organisms. Treatment includes systemic antibiotics with local heat and irrigation. Occasionally, surgical debridement with removal of sutures is required for adequate drainage. Perineorrhaphy for secondary closure should be done only after granulation tissue has appeared. Early repair of episiotomy wound dehiscence is preferred nowadays.

Extension of infection to the parametrium may cause lymphangitis. Deep vaginal or cervical lacerations can extend to the base of the broad ligament, and infections may cause lymphangitis, parametritis and bacteremia.

29.4.4 Wound Dehiscence

Wound dehiscence is commonly associated with infection. There is no relation of dehiscence to technique of repair. Other predisposing factors are coagulation disorders, smoking and human papillomavirus infection. The patients present with pain, fever, purulent discharge and dysuria, with or without urinary retention. The whole vulva might become oedematous, ulcerated and covered with exudates in severe cases.

Treatment includes early repair of the wound under cover of systemic broad-spectrum antibiotics. Before starting repair, the wound should be cleaned and free of infection. Pink granulation tissue should be visible before starting procedure. The internal and external anal sphincters must be identified and adequately repaired. Secondary repair is done in layers similar to primary episiotomy closure.

Postoperative care includes regular cleaning of wound, low-residue diet, stool softeners and abstinence. No per rectal or per vaginum medication should be given till healing is complete.

29.4.5 Peroneal Neuropathy

It results in foot-drop and weakness on dorsiflexion of the foot, occasionally with paresthesia in the foot and second toes. This problem usually becomes obvious 1–2 days after delivery. It may occur due to prolonged episiotomy repair causing pressure on the nerve from knee stirrups. Patients at risk are small women with relatively large babies and those with prolonged labour (compression of the L4–L5 lumbosacral nerve trunk). The problem is managed conservatively with good prognosis. Sometimes, a short leg brace may be required.

29.4.6 Necrotizing Fasciitis

It is found more in immunocompromised patients like diabetics, obese and hypertensive women. It is an acute, fulminant and rapidly spreading polymicrobial infection of the superficial and subcutaneous fascia, which can be fatal. Clinical features are excessive pain with tenderness and induration at wound site, with central necrosis and surrounding purplish erythema.

29.4.7 Obstetric Anal Sphincter Injuries (OASI)

This is a serious complication of episiotomy and is dependent on factors such as accuracy of the angle of incision, the dimensions of the cut and the distance of the incision point of the episiotomy from the midline [27].

However, evidence is contradictory regarding whether episiotomy increases or decreases the risk of OASI. A recent meta-analysis found lower risk of OASI when mediolateral episiotomy was given. Therefore, the authors recommended that episiotomy should not be withheld, especially in primigravidae [28].

One study has quantified the risk of OASI depending on characteristics of episiotomy. They found that risk of OASI is decreased by 70% with every 5.5 mm increase in depth of the episiotomy. Moreover, the risk is reduced by 56% when the incision point of episiotomy moves further from the posterior fourchette by 4.5 mm. Lengthening of episiotomy by 5.5 mm decreases the risk of OASI by 75%. The chances of OASI also depend on the angle of incision to the midline, increasing with an angle of either <15° or >60°. Therefore, episiotomies that are near the posterior fourchette, short and shallow episiotomies and those with angles smaller than 15° or larger than 60° have higher chances of third-degree and fourth-degree perineal lacerations [27]. However, this is a small study, and more research is needed before protective effect of episiotomy can be established.

29.4.8 Endometriosis

Scar endometriosis occurring at episiotomy site is a very rare complication, and incidence is even lesser than in caesarean section scar. Pathogenesis is said to be due to implantation of endometrial cells at the episiotomy site during delivery. Scar endometriosis usually presents as a slow-growing subcutaneous nodule at the site of episiotomy or surrounding area. The nodule is often painful with episodes of pain and swelling coinciding with the menstrual cycle. Additional diagnostic modalities are rarely required. Wide local excision is preferred, being both diagnostic and therapeutic. The margins of incision should be wide and clear to prevent recurrence or malignant transformation.

29.4.9 Fistula

Though rare, fistula is a serious complication seen in cases of obstructed labour, inappropriate repair of fourth-degree perineal lacerations or breakdown of episiotomy repair. Rectovaginal fistula is the one usually associated with obstetric injuries. Symptoms are involuntary release of flatus and/or faeces into the vagina. The site and size are confirmed by proper rectovaginal examination (under anaesthesia if required).

Fistula is best prevented by adequate and careful repair of lacerations and episiotomy. Repair of the fistula is done transvaginally with around 90% closure rate. If lower down, it is converted into complete perineal tear and then repaired accordingly. If higher up, flap method is required. Larger or recurrent fistulas may require diverting colostomy during healing period.

29.4.10 Dyspareunia

Dyspareunia is a not uncommon complication of episiotomy due to formation of tender scar.
OASI is related to more perineal pain than other perineal trauma. Episiotomy has been seen to cause more dyspareunia and vaginal dryness than spontaneous second-degree tears [25].

29.4.11 Pelvic Floor Disorders (Stress Incontinence, Overactive Bladder, Anal Incontinence and Prolapse)

Though initially it was thought that episiotomy improves the pelvic floor strength, it has now been suggested that after healing of episiotomy, the pelvic floor becomes scarred with weakening of the tissues, predisposing to pelvic organ prolapse and other disorders [29]. Prolapse, mostly rectocele, becomes obvious only in old age due to further weakening of pelvic musculofascial supporting tissues.

A 2005 systematic review concluded that the relation of episiotomy to pelvic floor disorders is still not established. They found that the relative odds for pelvic floor disorders were similar whether prior episiotomy was given or not. On the other hand, the relative odds of prolapse were doubled for women with multiple spontaneous lacerations, when compared to one laceration or none [30].

29.5 To Cut or Not To Cut?

Even though episiotomy is the most common obstetric procedure performed worldwide, still the controversy remains about when and how fre-

quently it should be used. The World Health Organization does not recommend routine use of episiotomy because there is no evidence for any beneficial effect of routine use [31]. Other national and international guidelines have similar recommendations.

The latest NICE guidelines recommend the following [11]:

- Routine episiotomy should be avoided in spontaneous vaginal deliveries.
- Episiotomy should be performed if clinically necessary, e.g. during operative delivery or fetal distress.
- Multiparae with a history of third- or fourth-degree trauma are not at higher risk of repeat injury; hence, episiotomy should not be offered as a routine.

Avoidance of routine episiotomy was also the suggestion by the authors of a recent Cochrane review that included 8 randomized controlled trials studying over 5000 deliveries comparing routine episiotomy (75.15%) versus restrictive episiotomy (28.4%) [25]. Their main results were:

Advantages of restrictive use:

- Less posterior perineal trauma (episiotomy may involuntarily extend into the anal sphincter or rectum).
- Less suturing.
- Lesser healing complications.

Advantages of routine use:

- Lesser anterior perineal trauma.

There was no difference in severe vaginal or perineal trauma, dyspareunia or several pain measures.

Findings in other studies show that episiotomy increases the risk of third- and fourth-degree tears. The incidence of faecal and flatus incontinence was increased four- to sixfold in women with an episiotomy compared to those with intact perineum; risk was tripled when compared to spontaneous lacerations [32].

On the other hand, few studies have found more chances of short-term perineal pain with restrictive episiotomy [33]. Episiotomy, according to few researchers, has a protective effect on quality of life and pelvic floor disorders after 1 year follow-up [34].

Selective indications for episiotomy:

1. Shoulder dystocia and breech delivery
2. Forceps or vacuum extraction
3. Delivery in occipito-posterior position
4. Impending perineal rupture if episiotomy not performed
5. Elastic rigid perineum
6. Operative vaginal delivery
7. Previous perineal surgery—pelvic floor repair and perineal reconstructive surgery
8. Fetal distress

In case of instrumental vaginal delivery, the opinions are varied. The American College of Obstetricians and Gynecologists (ACOG) opposes episiotomy during operative delivery, due to more perineal pain and dyspareunia [35]. The RCOG, however, supports use of restricted episiotomy depending on the operator's judgement. Still, there is no conclusive evidence. There is data to support that mediolateral episiotomy is safer than midline in operative delivery [36]. Sphincter injures are higher with vacuum extraction; therefore, episiotomy can be used in such a situation.

If episiotomy is not to be routinely used, then what method should be used to reduce perineal trauma during delivery? Not many studies answer this question. Antenatal perineal massage, warm compresses, use of birth pools and avoidance of the upright position have been suggested as preventive measures. A recent Cochrane review concluded that warm compresses during the second stage of labour significantly decreased third- and fourth-degree tears [37]. Various devices have been tried to reduce lacerations, e.g. the EpiNo device (causes stretching of pelvic floor muscles), but none has become popular.

To conclude, each case is individual, and attending obstetrician is best suited to decide whether episiotomy is to be given or not.

References

1. Baskett TF, Calder AA, Arulkumaran S. Munro Kerr's operative obstetrics. 11th ed. London: Elsevier; 2007.
2. Carroli G, Belizan J. Episiotomy for vaginal birth. Cochrane Database Syst Rev. 2000;(2):CD000081.
3. Graham ID, Carroli G, Davies C, Medves JM. Episiotomy rates around the world: an update. Birth. 2005;32(3):219–23.
4. Thacker S, Banta D. Benefits and risks of episiotomy: an interpretive review of the English language literature, 1860–1980. Obstet Gynecol Surv. 1983;38:322–38.
5. American College of Obstetricians-Gynecologists. ACOG Practice Bulletin. Episiotomy. Clinical management guidelines for obstetrician-gynecologists. Number 71, April 2006. Obstet Gynecol. 2006;107(4):957–62.
6. Bhatia JC, Cleland J. Determinants of maternal care in a region of South India. Health Transit Rev. 1995;5:127–42.
7. Sathiyasekaran BWC, Palani G, Iyer RH, Edward S, Dharmappal CD, Rani A, et al. Population based study of episiotomy. Sri Ramachandra J Med. 2007;1:9–14.
8. Borges BB, Serrano F, Pereira F. [Episiotomy—routine versus selective use]. Acta Med Port. 2003;16(6):447–54.
9. Larsson PG, Platz-Christensen JJ, Bergman B, Wallstersson G. Advantage or disadvantage of episiotomy compared with spontaneous perineal laceration. Gynecol Obstet Investig. 1991;31(4):213–6.
10. Gainey HL. Postpartum observation of pelvic tissue damage: further studies. Am J Obstet Gynecol. 1955;70(4):800–7.
11. Delgado Nunes V, Gholitabar M, Sims JM, Bewley S, Guideline Development Group. Intrapartum care of healthy women and their babies: summary of updated NICE guidance. BMJ. 2014;349:g6886.
12. May JL. Modified median episiotomy minimizes the risk of third degree tears. Obstet Gynecol. 1994;83:156–7.
13. Kalis V, Laine K, de Leeuw JW, Ismail KM, Tincello DG. Classification of episiotomy: towards a standardisation of terminology. BJOG. 2012;119(5):522–6.
14. Corton MM, Leveno KJ, Bloom SL, Spong CY, Dashe JS. Williams obstetrics. 24th ed. New York: McGraw-Hill; 2014.
15. Anthony S, Buitendijk SE, Zondervan KT, van Rijssel EJ, Verkerk PH. Episiotomies and the occurrence of severe perineal lacerations. Br J Obstet Gynaecol. 1994;101(12):1064–7.
16. Royal College of Obstetricians and Gynaecologists. The management of third and fourth degree perineal tears. Green top guideline no. 29. London: RCOG; 2015.
17. Kalis V, Landsmanova J, Bednarova B, Karbanova J, Laine K, Rokyta Z. Evaluation of the incision angle of mediolateral episiotomy at 60 degrees. Int J Gynecol Obstet. 2011;112(3):220–4.
18. Tincello DG, Williams A, Fowler GE, Adams EJ, Richmond DH, Alfirevic Z. Differences in episiotomy technique between midwives and doctors. BJOG. 2003;110(12):1041–4.
19. Kettle C, Dowswell T, Ismail KM. Absorbable suture materials for primary repair of episiotomy and second degree tears. Cochrane Database Syst Rev. 2010;(6):CD000006. https://doi.org/10.1002/14651858.CD000006.pub2. Review
20. Kettle C, Hills RK, Jones P, Darby L, Gray R, Johanson R. Continuous versus interrupted perineal repair with standard or rapidly absorbed sutures after spontaneous vaginal birth: a randomised controlled trial. Lancet. 2002;359(9325):2217–23.
21. Minassian VA, Jazayeri A, Prien SD, Timmons RL, Stumbo K. Randomized trial of lidocaine ointment versus placebo for the treatment of postpartum perineal pain. Obstet Gynecol. 2002;100(6):1239–43.
22. Combs CA, Robertson PA, Laros RK Jr. Risk factors for third-degree and fourth-degree perineal lacerations in forceps and vacuum deliveries. Am J Obstet Gynecol. 1990;163(1 Pt 1):100–4.
23. Wheeler J, Davis D, Fry M, Brodie P, Homer CS. Is Asian ethnicity an independent risk factor for severe perineal trauma in childbirth? A systematic review of the literature. Women Birth. 2012;25(3):107–13.
24. Steiner N, Weintraub AY, Wiznitzer A, Sergienko R, Sheiner E. Episiotomy: the final cut? Arch Gynecol Obstet. 2012;286:1369–137.
25. Carroli G, Mignini L. Episiotomy for vaginal birth. Cochrane Database Syst Rev. 2009;(1):CD000081.
26. Decherney AH, Nathan L, Goodwin TM, Laufer N, Roman AS. Current diagnosis & treatment obstetrics & gynecology. 11th ed. New York: McGraw-Hill; 2012.
27. Stedenfeldt M, Pirhonen J, Blix E, Wilsgaard T, Vonen B, Oian P. Episiotomy characteristics and risks for obstetric anal sphincter injuries: a case-control study. BJOG. 2012;119(6):724–30.
28. Verghese TS, Champaneria R, Kapoor DS, Latthe PM. Obstetric anal sphincter injuries after episiotomy: systematic review and meta-analysis. Int Urogynecol J. 2016;27:1459–67.
29. Casey BM, Schaffer JI, Bloom SL, Heartwell SF, McIntire DD, Leveno KJ. Obstetric antecedents for postpartum pelvic floor dysfunction. Am J Obstet Gynecol. 2005;192(5):1655–62.
30. Handa VL, Blomquist JL, McDermott KC, Friedman S, Muñoz A. Pelvic floor disorders after vaginal birth: effect of episiotomy, perineal laceration, and operative birth. Obstet Gynecol. 2012;119(2 Pt 1):233–9.
31. World Health Organization. Care during the second stage of labour. In: Care in normal birth: a practical guide. Geneva: World Health Organization; 1996. http://www.who.int/reproductive-health/publications/MSM 96 24/MSM 96 24 chapter4.en.html.
32. Signorello LB, Harlow BL, Chekos AK, Repke JT. Midline episiotomy and anal incontinence: retrospective cohort study. BMJ. 2000;320(7227):86–90.

33. Macleod M, Goyder K, Howarth L, Bahl R, Strachan B, Murphy DJ. Morbidity experienced by women before and after operative vaginal delivery: prospective cohort study nested within a two-centre randomised controlled trial of restrictive versus routine use of episiotomy. BJOG. 2013;120(8):1020–7.
34. Bertozzi S, Londero AP, Fruscalzo A, Driul L, Delneri C, Calcagno A, Di Benedetto P, Marchesoni D. Impact of episiotomy on pelvic floor disorders and their influence on women's wellness after the sixth month postpartum: a retrospective study. BMC Womens Health. 2011;11:12.
35. Committee on Practice Bulletins—Obstetrics. ACOG practice bulletin no. 154: operative vaginal delivery. Obstet Gynecol. 2015;126(5):e56–65.
36. Royal College of Obstetricians and Gynaecologists. Operative vaginal delivery. Green top guideline no. 26. London: RCOG; 2011.
37. Aasheim V, Nilsen AB, Lukasse M, Reinar LM. Perineal techniques during the second stage of labour for reducing perineal trauma. Cochrane Database Syst Rev. 2011;(12):CD006672.

Instrumental Delivery

30

Parul Kotdwala and Munjal Pandya

30.1 Introduction

There has been mention of forceps in Sanskrit (1500 BC), Egyptian, Greek, Roman, and Persian writings and pictures, but thought to be used for the extraction of dead fetus. Around 1600 AD, Peter Chamberlen (England) used forceps on live fetus (Fig. 30.1). Levert (1747) introduced the pelvic curve. Sir James young Simpson (1845) developed a forceps appropriately fitting both cephalic and pelvic curvatures. James Haig Ferguson (1862–1934) modified Simpson's forceps by shortening the handle and placing slots to allow the application of traction tapes increasing fetal head flexion. Joseph DeLee (1920) advocated prophylactic forceps delivery. Christian Kielland (1915) invented rotational forceps. William Smellie (1975) was the first one to describe "cephalic application" rather than previously performed "pelvic application." He also designed English lock. Arthur Wrigley (1902–1983) invented Wrigley's forceps useful for outlet forceps application. KN Das modified application of forceps at midcavity level (Fig. 30.2). Moolgaonkar (1962) developed his forceps, which is an improvement upon the Kjelland instrument (Fig. 30.3). He devised this specifically for narrower pelves of

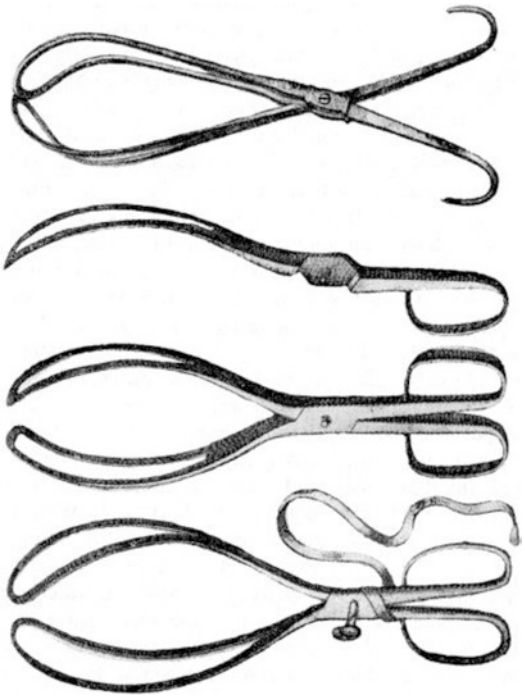

Fig. 30.1 Chamberlen's forceps

P. Kotdwala (✉)
Department of Obstetrics and Gynecology,
V.S. Hospital & N.H.L. Municipal Medical College,
Ahmedabad, Gujarat, India

Zydus Hospital, Ahmedabad, Gujarat, India

Kotdawala Women's Clinic,
Ahmedabad, Gujarat, India

M. Pandya
AMC MET Medical College, Sheth L.G. Hospital,
Ahmedabad, Gujarat, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_30

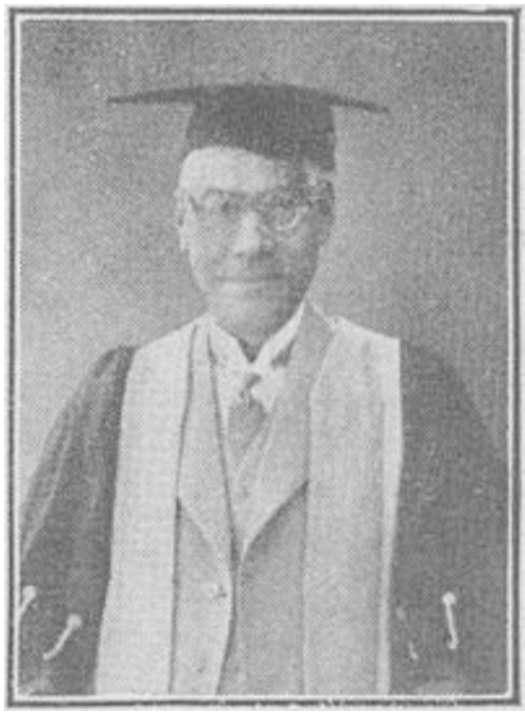

Fig. 30.2 Kedarnath Das (1867–1936)

Fig. 30.3 Das's modified forceps

Indian women, though later this instrument has gained popularity in Britain also. The use of instrumental delivery is mostly to reduce the duration of the second stage of labor, but in modern practice, with the advent of fetal surveillance systems, the length of the second stage is not an absolute indication. Studies suggest that morbidity increases significantly only after 3–4 h in the second stage [1]. Instrumental deliveries are increased when epidural analgesia is used due to lack of Ferguson's reflex [2].

> The obstetric forceps is a unique instrument. A simple and honest implement, compared to many of man's inventions. Its history is nevertheless complicated with confusion and irony and its inventions shrouded in secrecy. It probably has saved more lives than any instrument ever devised and yet it did not appear until countless generations of men struggled into the world without it, or failed to arrive. A real and solid thing, easily fabricated, it is yet capable of such subtle variations that its evolution has never stopped. It is designed specifically to rescue life, and yet it descended from an instrument of death! [3]

30.2 Indications for Operative Vaginal Delivery [4]

1. Fetal indications:
   - Fetal distress, based on abnormal heart rate pattern of fall in fetal scalp pH.
2. Maternal indications:
   - To shorten the second stage of labor and reduce the effects of the second stage of labor on medical conditions (e.g., cardiac disease class III or IV, hypertensive crisis, myasthenia gravis, spinal cord injury patients at risk of autonomic dysreflexia, proliferative retinopathy).
3. Inadequate progress:
   - Nulliparous women—lack of continuing progress for 3 h (total of active and passive second-stage labor) with regional anesthesia, or 2 h without regional anesthesia.
   - Multiparous women—lack of continuing progress for 2 h (total of active and passive second-stage labor) with regional anesthesia, or 1 h without regional anesthesia.
   - Maternal fatigue/exhaustion.

30.2.1 Contraindication to Operative Vaginal Delivery [4]

- *Relative contraindication*: unfavorable attitude of fetal head, rotation >45° from occipitoanterior or occipitoposterior, midpelvic station
- *Absolute contraindication*: Nonvertex or brow presentation, no engaged head. Fetal coagu-

lopathy, incomplete cervical dilation, cephalopelvic disproportion

30.3 Parts of Forceps (Figs. 30.4, 30.5, and 30.6)

Forceps are composed of two blades, each one having four parts:

- Blades: The blades are the parts which grasp the fetal head. Each blade has two curves: A cephalic curve, which fits in the shape of fetal head and a pelvic curve which corresponds to birth canal axis. Some forceps have blades which are fenestrated. The blade which will come in contact with the left maternal pelvic wall is called the left blade, and the one which will come in contact with the right pelvic wall is the right blade.
- Shank: It is the part connecting handle with the blade. The shanks are parallel in the straight forceps, or crossing in some forceps.
- Lock: There are many types of locks for articulation between shanks. English lock is more common where socket at the junction of the handle fits into the opposite shank.
- Handles: These are the parts to hold the device and to give traction to the fetal head.

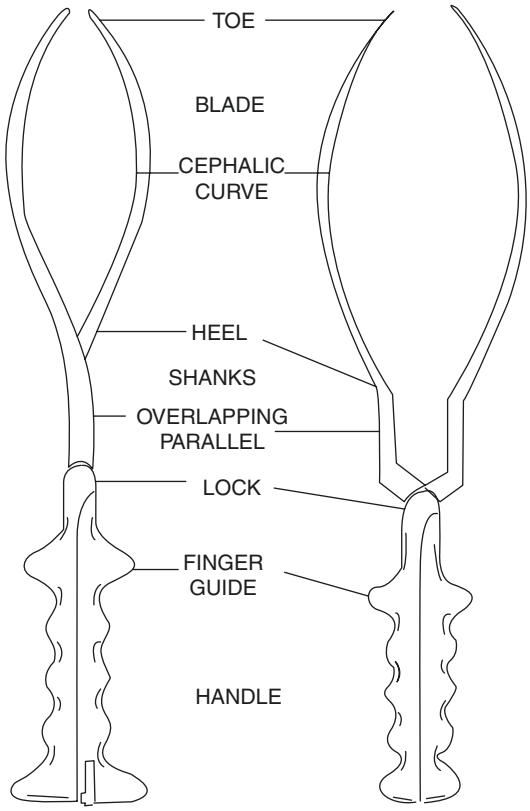

Fig. 30.4 Parts of forceps

30.4 Types of Forceps

1. Simpson's forceps (Fig. 30.7): It is a type of midcavity/low forceps. It is used when the sagittal suture is in direct anteroposterior position or within 45° from the midline.
2. Piper's forceps (Fig. 30.8): Used for aftercoming head of breech.
3. Wrigley's forceps (Fig. 30.9): It is a type of outlet forceps, the most commonly used forceps.
4. Kielland's forceps (Fig. 30.10): It is a type of rotational forceps when the occiput is in transverse or posterior position, requiring considerable experience and skill. It has minimal pelvic curve, longer shank, and a sliding lock.

Fig. 30.6 Tucker-McLane forceps

Fig. 30.5 Anatomy of forceps

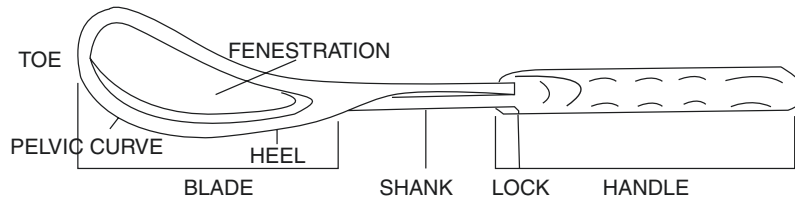

Fig. 30.7 Simpson's forceps

Fig. 30.8 Piper's forceps

Shank has two knobs on the same side to identify the progressive rotation.

5. Axis traction forceps: Axis traction was devised by Tarnier in 1877 (Fig. 30.11). When straight forceps are applied in midcavity, traction becomes difficult. The traction rods enable traction to be applied in the axis of pelvic cavity. Neville-Barnes and Haig-Ferguson's forceps belong to this category (Fig. 30.12).
6. Laufe's forceps (Fig. 30.13): Specially designed to limit fetal cranial compression with divergent or parallel blades.

30.4.1 Functions of Obstetric Forceps

1. Traction
2. Compression
3. Rotation
4. Other functions:
   - Vectis
   - As a protective cage, for preterm head
   - Inducing a uterine contraction

30.5 ACOG Criteria for Types of Forceps Delivery [5]

1. Outlet forceps:
   - The scalp is visible at introitus, without separating labia.
   - The fetal skull has reached the pelvic floor.
   - The sagittal suture is in anteroposterior diameter, right or left occiput anterior or posterior position (i.e., the fetal head is at or on perineum and rotation does not exceed 45°).
2. Low forceps: The leading point of the fetal skull is at a station greater than or equal to +2 cm and is not on the pelvic floor; any degree of rotation may be present.

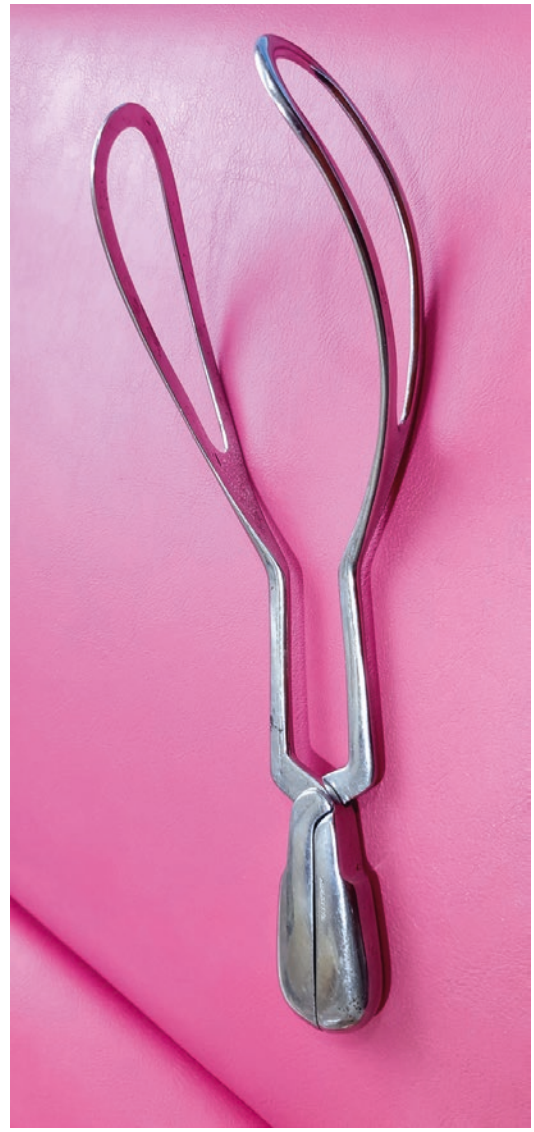

Fig. 30.9 Wrigley's forceps

Fig. 30.10 Kjelland's forceps

3. Mid forceps: The station is above +2 cm, but the head is engaged.

Fig. 30.11 Tarnier forceps

Fig. 30.12 Haig Ferguson's forceps

4. High forceps: this is not included in the classification. Previous systems classified high forceps deliveries as procedures performed when the head is not engaged. High forceps deliveries are not recommended.

30.5.1 Selection of Instruments

The choice of a forceps for delivery should be made on the basis of the particular circumstances of that delivery.

1. Low (Outlet) forceps: The saggital suture is in midline, and the occiput is either anterior or posterior. Traction power required is much less. Asynclitism is not an issue. Hence, an instrument with short shanks with or without fenestra is adequate.

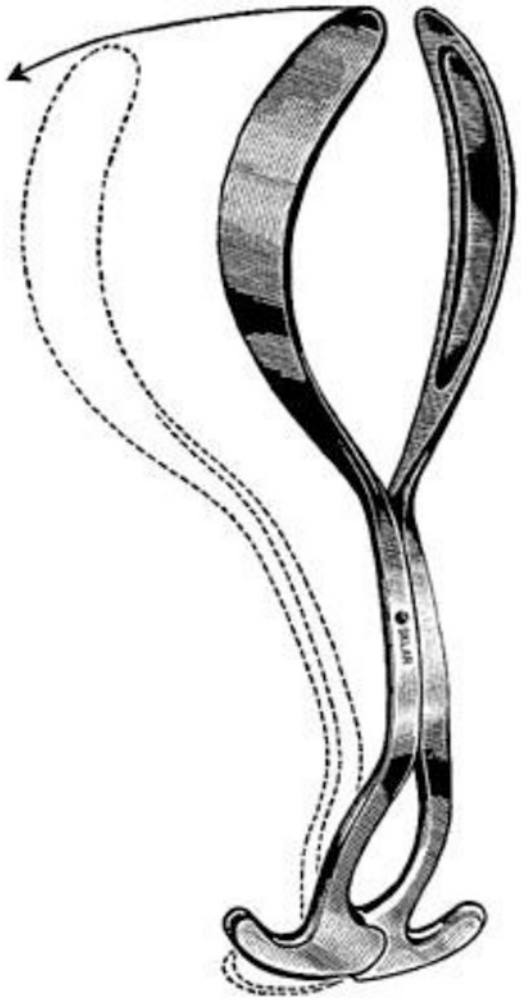

Fig. 30.13 Laufe's forceps

2. Mid forceps: The head is higher. Direction of traction is complicated. Better grasp is required as traction force required is more. Asynclitism may be present. Rotation may be required. So forceps with long shanks and with good axis traction attachment are required; forceps for rotation and sliding locks may be required.
3. High forceps: This procedure is now obsolete due to its difficulties and hazards. It is of historical interest only.
4. Face presentation: Good grasp of head is needed. To maintain extension of the head, proper downward traction is necessary. Sometimes rotation is required. So forceps with fenestrated blades (for better grasp) and axis traction device are the preferred ones.
5. Brow: As it is a transitory presentation towards the face, the choice of instrument is the same as for face presentation. Strong traction necessitates axis traction.
6. Breech: On after-coming head Piper's forceps with long shanks and reverse pelvic curve to underlie the baby are helpful. The offset pelvic curve keeps the head in sustained flexion.
7. Cesarean section: The idea is to assist the delivery of the head, especially in a floating head. A short shank would make extraction easy, and any of the conventional instruments suffices. In a deeply engaged head, a single member of the forceps pair can be used as a vectis, with the pubic symphysis acting as a fulcrum. Murless extractor, with a folding blade, may be a preferred one for this purpose.

30.5.2 Prerequisites

Before application of forceps/vacuum, the following prerequisites should be fulfilled.

- Explanation of procedure with its consequences should be a routine along with a documented consent.
- Abdominal examination should confirm only 1/5th of palpable head.
- Vaginal examination should confirm the station +1, with head descent during uterine contractions. One should carefully rule out a deflexed head and/or asynclitism. Pelvic assessment for size adequacy is vital. Excessive caput succedaneum and molding needs to be evaluated for the possibility of a cephalo-pelvic disproportion.
- Cervix has to be fully dilated.
- Membranes should be ruptured.
- Position of fetal head must be known.
- Bladder should be empty and may need catheterization for this.

- Episiotomy is generally employed, but may depend upon the operator's personal judgment [6].

30.5.3 Analgesia

- *Perineal infiltration*: 10–20 mL of 1% lignocaine is injected fanwise from a point in the midline at fourchette.
- *Pudendal block*: can be given by two methods. In the external method, the point of injection is upon the perineum midway between ischial tuberosity and introitus. A 12.5 cm long spinal needle is used, which is advanced up to the ischial spines under guidance of a vaginal finger. At a point below and beyond the ischial spine, the sacrospinous ligament is pierced and 10 cc of 1% xylocaine is injected after checking that the needle point is not in a blood vessel. The same procedure is repeated on the opposite side. In the internal method, the needle pierces the vaginal mucosa overlying the ischial spines under guidance of a finger trans-vaginally and the injection is given in the same manner. Internal method has less chance of infection. Generally, a pudendal block requires additional local perineal infiltration as few nerves may escape the block effect, namely—the branches of ilio-inguinal, genital branch of genitofemoral and posterior cutaneous nerve of the thigh escape the block.
- When a trial of forceps is contemplated and proceeding to cesarean section is a possibility, an epidural anesthesia is the preferred mode.
- *Epidural or spinal analgesia*: Generally reserved for a Kielland's rotational forceps.

30.6 Technique

An outlet forceps is used more in modern practice. Midcavity forceps are not routinely practiced nowadays due to the possibility of increased morbidity. A lithotomy position with legs in stirrups is preferred. Perineum is washed with copious antiseptic lavage by Savlon. Bladder evacuation is confirmed. The perineal infiltration and pudendal block are carried out as needed. The blades of forceps are assembled to make sure that the pair is correct. The uterus must be contracting well, and oxytocin infusion is started if necessary.

30.6.1 Low Forceps: Outlet Forceps

When the head is crowning (it distends the vulva, and does not recede in between contractions) also known as +3 station (when the BPD has crossed the ischial spines, i.e., only the soft tissues are holding it back), and when the sagittal suture is in midline, that forceps delivery is known as "outlet forceps operation" or "low forceps operation."

There are two instruments available for this operation:

1. Wrigley's forceps with short handles and pelvic curve
2. Short Simpson's forceps with straight blades

Technique: The left blade should be inserted first to facilitate articulation. The insertion of left blade is begun by creating a space between the fetal head and pelvic wall by the pre-lubricated middle and index fingers of the right hand inserted into the vagina along the left side. This will confirm the full retraction of the cervix and will provide a shield to the maternal soft tissues.

The handle is held upright in the left hand slightly towards the right maternal groin as if it were a pen. The right thumb is placed at the heel of the blade. By allowing the shanks to slide over the thumb, the downward descent of the branch is controlled. The handle is now gently lowered in a sweeping arc which begins towards the operator's left and ends in the middle when the shank and handle reach a horizontal position. During this maneuver, the blade is guided upwards into the pelvis along its pelvic curve. The right branch is inserted in a likewise manner, though the hands are reversed.

In a good application of forceps the locking should be accomplished in an almost effortless manner. If resistance is encountered their application and relationship must be rechecked and adjusted. Easy locking is frequently facilitated by equal depression of the handles.

Correct application is noted generally by Denman's criteria: three landmarks are checked for the diagnosis of a proper forceps application: (1) the posterior fontanelle, (2) the sagittal suture, and (3) the fenestration.

The posterior fontanelle should be one finger breadth above (or below in case of face to pubis) the plane of the shanks. If this is improper, the pivot point of the head is off center, and traction may cause either extension or overflexion of the head. This may result in larger diameter passing through the canal and may increase the chances of trauma to vagina.

The sagittal suture should be perpendicular to the plane of the shanks. If this is not appropriate there is a risk of a brow-mastoid application, and the risk of trauma to facial nerve or eye of the fetus.

The posterior fenestration of the blade should admit no more than a fingertip. If there is too much room at the bottom of the blades, the tip may not be anchored at or below the malar eminences, and traction may cause slippage and trauma to the infant's face.

30.6.2 Traction

After checking the application, traction should be applied, preferably in sitting position with operator at comfortable level—his flexed forearms slightly beneath the edge of the table. After grasping the forceps from beneath, traction should derive from flexed, unaided forearms during contraction of the uterus in a plane continuous with the curve of the pelvis.

The traction should mimic and coincide with uterine contractions. The pull should begin gradually, reach an acme of intensity, be sustained for a short but definite interval, and then gradually subside. The first pull is treated as a "trial traction," which judges the existent resistance and required efforts. The fetal status is also checked by listening to heart sounds during the pull. The forceps blades should be unlocked in between the contractions to release the compression over fetal head.

Once the occiput has passed beneath the subpubic angle the direction of pull is gradually changed by elevating the handles to about 40° above horizontal plane. Once the biparietal diameter has passed the vulval ring the forceps can be removed in a reversed order of their application.

30.6.3 Occipitoposterior (Face to Pubis)

If the occiput is posterior, the technique of insertion and application of forceps blades remain the same as in occiput anterior. Only the application check is reversed. Since the occiput is posterior, the traction directions will change. First the pull is horizontal, once the occiput is visible—the forceps is moved anterior to increase flexion of the head. After the occiput is delivered at fourchette, the forceps are depressed towards posterior to let the face glide out under the pubic rami. A wide episiotomy is in order, as wider occiput may distend the posterior vagina more.

30.6.4 Face Presentation

1. Mentum anterior: Kjelland's forceps are particularly well suited because of the lack of a pelvic curve. This straightness in the blades permits delivery with maintenance of extension of the head, which is the key to a satisfactory forceps delivery of the face. The direction of pull is nearly horizontal which alone maintains proper extension and permits the chin to traverse the inner surface of the symphysis. When this happens, then and only then flexion is performed, the handles are lifted upwards, and delivery of the head is completed.
2. Mentum posterior: A vaginal delivery is avoided nowadays due to significant risk of trauma to the baby.

30.6.5 After-coming Head in Breech Presentation

Piper's forceps is the most expedient and useful tool for the delivery of an after-coming head, as it prevents cerebral trauma and decreases morbidity by twofold.

Before applying the forceps and after delivery of the shoulders, it is necessary for an assistant to hold the baby in a swayback position with the arms out of the way. The operator then briefly holds the locked forceps in position below the baby and facing the introitus. A single kneeling position is the best. The handle of the left blade is grasped by the left hand while the right hand is introduced alongside the head in the birth canal. The blade is carried upwards diagonally across the introitus and introduced in a direct manner to apply along an axis near to or on to occipitomental line. The application of the right blade is carried out in the same manner except that the hands are reversed. One has to be careful to prevent extension of the head in introducing the second branch.

It is important to ascertain that the blades have been placed at sufficient depth, so that they overlie the ears, the tips extending a bit beyond, to obtain a good grasp of the head and to prevent fetal trauma. The forceps are then articulated. Traction is made in an obviously downward curve. The direction of pull is not varied. There is only a limited time available once compression of the cord occurs by the delivered parts of the baby. Hence the pull and the delivery of the head are achieved straightaway, unlike intermittent pulls in cephalic forceps operations. There is a springing in the shanks of the forceps to prevent the compression of the head.

30.7 Rotational Forceps

Whenever the head is unrotated, meaning the posterior fontanelle is deviated by more than 15° from midline, we may need to rotate the fetal head to bring the sagittal suture in the midline for a safe delivery. This can be achieved by either the Scanzoni maneuver or by using specially designed instruments like Kjelland's forceps.

30.7.1 Scanzoni Maneuver [7]

Friedrich Wilhelm Scanzoni, a German obstetrician, described this technique in 1849. It is used for occipito-posterior as well as occipito-transverse presentations.

In case of occipito-posterior presentations, the blades are applied with reference to the pelvis as if it is an occipito-anterior position. The head is then rotated by moving the handles in a wide arc outside, to eliminate wider movement of the blades inside the pelvis, to avoid trauma to the maternal soft tissues. Once the head is rotated to anterior position, the blades which are now upside down are removed and reapplied in regular way. The application criteria are rechecked, and the traction is applied for the delivery.

In case of occipito-transverse position, the posterior blade is applied first, with pelvic curve towards the occiput of the fetus. The anterior blade is then inserted similarly. The blades are articulated. After flexion of the neck by forceps, the handles are rotated anterior from lateral position along with uterine contractions. Once rotated, the application is rechecked and traction is applied for delivery (Fig. 30.14).

30.7.2 Kielland's Forceps

This is one of the most versatile forceps ever designed addressing all aspects of rotational delivery. It has a negated pelvic curve for easy rotation, narrower blades to minimize trauma during application and rotation, curved inner surface of blades for better grip of fetal head, flat outer surface of blades to minimize soft tissue entrapment trauma, and sliding locks to correct asynclitism. There are knobs on the handle, which should be towards occiput, when the blades are applied. The forceps is articulated before application in front of the patient, and the anterior blade is selected for first insertion. It can be inserted by three methods: direct, wandering, and classical. In the direct method the anterior blade is guided to the front of the fetal head behind the pubic bones. In the wandering method, the anterior blade is passed posteriorly along the curve of the sacrum, and then is rotated over the face of the

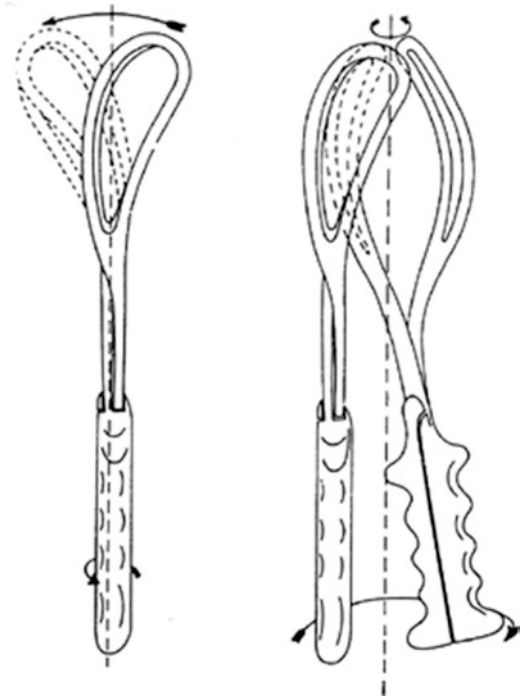

Fig. 30.14 Scanzoni maneuver

fetus to the anterior plane. The classical method, meant for high forceps operations, is abandoned now. Once the anterior blade is in position, the posterior blade is inserted directly in the hollow of sacrum. The blades are then articulated. In case of an asynclitism, the handles will not be in the same plane. This is corrected by sliding the shanks over each other. The head is then rotated in "key in lock" manner. After rotation is complete, the application is rechecked, and then the traction is applied for the delivery.

30.8 Prophylactic Forceps

DeLee (1920) proposed a radical thought of conducting all vaginal deliveries with forceps assistance. He propounded that by using forceps we are reducing the duration of the second stage as well as prolonged distension of pelvic viscera. This according to him protects the fetal brain by minimizing the prolonged effects of compression. DeLee has published his data showing higher satisfaction and positive memory of labor event by his patients who were offered prophylactic forceps delivery (Fig. 30.15).

30.9 Trial Forceps

In cases of borderline cephalopelvic disproportion a trial of forceps is attempted, a term coined by Douglas and Kaltrieder (1953) and Jeffcoate (1953), means a tentative application of instrument under operating theater conditions with all preparations ready for a resort to immediate cesarean section if the forceps delivery is not progressing smoothly.

30.10 Failed Forceps

Inadvertent failure to deliver baby after attempting forceps application is known as failed forceps. Usually this happens in improper selection of the case, or when the prerequisites are not met. A failed forceps is fraught with many complications and traumas to both the mother and the infant.

30.11 Complications

> Cautiously and tenderly must this iron instrument be used! We must recollect that no sensations can be imparted to the operator's hand of any injury that may be done to women, and we must remember that one injudicious thrust, one forcible attempt at introduction, one violent effort in extraction may bruise, may lacerate, may destroy! Bear in mind that the metallic blades have no feeling and can not communicate to our perceptions a knowledge of any mischief we may inflict. (Dr. John Ramsbotham, 1844)

There are maternal and fetal complications which happen due to faulty technique and improper use of the instrument.

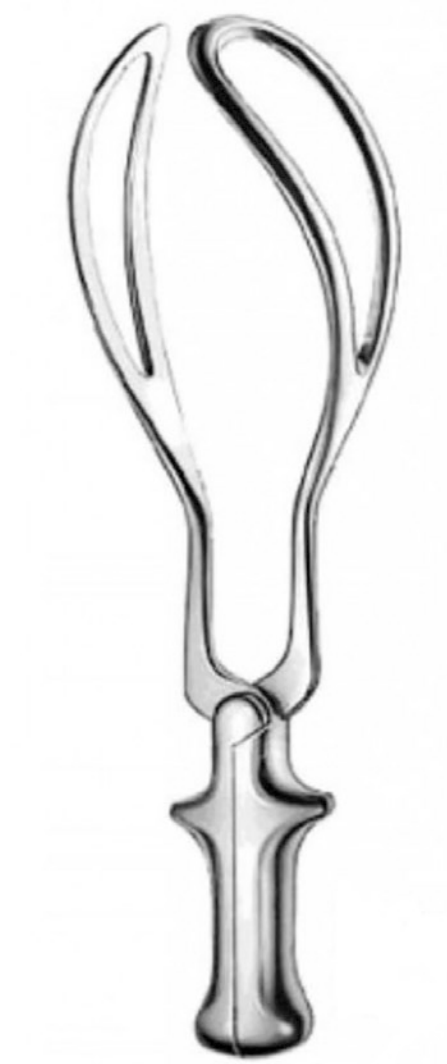

Fig. 30.15 DeLee's forceps

Maternal complications: Episiotomy wound extension, perineal tears, vaginal lacerations, paraurethral tears, bladder injury, rectal injury, cervical tear, lower uterine segment tears, puerperal sepsis, traumatic hemorrhage, bladder atony, obstetric shock.

Fetal complications: Intracranial hemorrhage, skull fractures, cephalhematoma, injuries to eyes, hearing defects, fractures of malar bones and atlanto-axial injuries, facial nerve palsy, Erb's palsy, spinal cord injuries.

A proper selection of cases, adhering to the safety protocols, adequate training and supervision, readiness to abandon the procedure in case of poor progress, and regular audit will reduce the complications, which can be lethal in some instances.

30.12 Ventouse Delivery

James Young Simpson (1849), often considered the father of anesthesia, is also credited with designing the first "Air Tractor" consisting of metal syringe and an attached soft rubber cup for a vacuum-assisted delivery. This did not gain popularity and almost after a century, Tage Malmstrom—a Swedish professor—developed the ventouse or Malmstrom extractor in the 1950s. This was originally made of a metal cup, with the pulling chain incorporated in the tube through which the vacuum suction was also created. Bird (1969) altered this by separating the traction chain and the suction point. Newer materials like plastic and silicon rubber in place of the metal have gained popularity and these vacuum cups are now used more frequently than a forceps for traction at delivery.

The main difference between a vacuum traction and a forceps traction is that the forceps are applied to grip the skull bones and so the pull and compression is primarily on the skull bones, whereas the vacuum cup is applied to the scalp tissue overlying the crown of the fetal head, and the traction force is on the skin overlying the scalp! The vacuum hence has a poorer grip, and may get detached frequently upon a pull applied to it, whereas a forceps has a better grip. But the same difference can translate into more compression trauma with forceps. Vacuum creation takes time, and hence a forceps is preferred in presence of fetal distress, where a quicker delivery is planned. Because of lesser potential for trauma and less training required, vacuum is being used more frequently than a forceps.

30.12.1 Types

There are two types of vacuum cups: The cups are made up of metal, plastic, and soft silastic. The vacuum apparatus consists of vacuum bottle, gauge, and connecting tubing (Fig. 30.16, 30.17, and 30.18).

30.12.2 Prerequisite

These are by and large the same as for a forceps delivery. Vacuum has a benefit over forceps as it can be applied even when the full dilatation of cervix has not happened, or when the rotation of head to midline is yet not complete. It may not be used when there is significant fetal distress, prematurity, if the fetus has a hemorrhagic condition. The chignon artificial caput takes a bit of time to develop, and in case of fetal distress, if time is of essence, a forceps is preferred. In a preterm fetus and when the fetus has a hemorrhagic condition, there is higher risk of intracranial hemorrhage and hence a vacuum is better avoided. Vacuum should not be applied before 34 weeks of gestation, and the safety of vacuum extraction between 34 and 36 weeks is uncertain and should be used with caution [6]. Vacuum extractors are contraindicated in a face presentation.

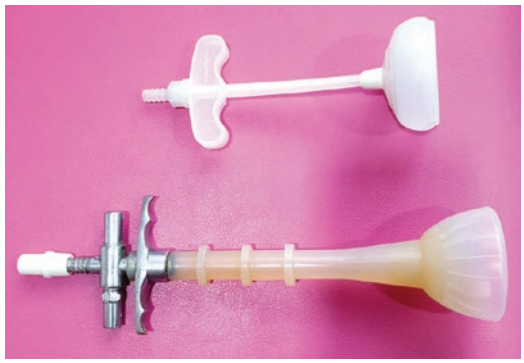

Fig 30.16 Vacuum cups

30.12.3 Analgesia

Usually, like in a forceps delivery, a perineal infiltration and/or pudendal block suffice for a vacuum delivery.

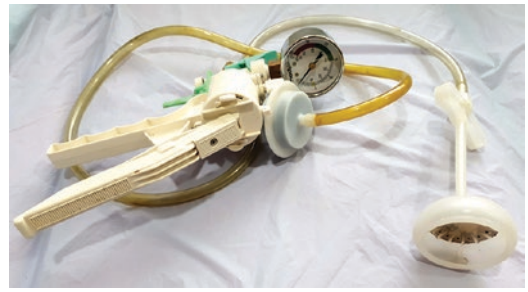

Fig 30.17 Handheld vacuum apparatus

Fig 30.18 Kiwi Omni Cup + Malmstrom

30.12.4 Technique

Central to the application of vacuum cup is the concept of "flexion point." It is a point on sagittal suture, 3 cm towards the crown from posterior fontanelle. An application of cup with its midpoint over the flexion point allows the most favorable diameter of fetal head to pass upon further traction. A de-flexion of the head occurs if the cup is applied anterior to this point, and similarly if it is applied to far posterior, it may lead to hyper-flexion of the head. If the cup goes on any one side of sagittal suture, it will produce asynclitism leading to larger diameter to be presented at the time of delivery. After application of the vacuum cup, fingers inserted to free any part of the cervix or vaginal wall which might get trapped inside the cup. A negative vacuum pressure is then created (150 mm of Hg), and is increased up to 500–600 mm of Hg. Once this pressure is achieved, the application is rechecked, and traction is applied. Traction should coincide with uterine contractions and maternal bearing down efforts to enhance descent of the fetus. The pull is at right angle to the cup. The direction depends upon the station of the head. Higher the station, downward is the pull. Once the head is crowning, the pull is in horizontal direction, gradually moving upwards. Once the head is delivered, the vacuum cup is detached from the suction device and is removed. Subsequent delivery of the baby is like any vaginal delivery after the delivery of the head.

If the cup comes off while applying traction, it can be reattached and the delivery process is continued. Sometimes, if the vacuum has come off a couple of times, but by this time the head has rotated, or has come low down in pelvis, subsequent delivery may be accomplished by a forceps. Generally a vacuum delivery is abandoned if the vacuum cup has come off for more than three times. In a condition of a failed vacuum delivery, a decision is made to either switch to a forceps delivery or a cesarean section.

Chignon: This is an elevated soft tissue swelling that occurs over fetal scalp due to the vacuum application. It gets settled within next 2–3 days. The parturient lady and her kin may be counseled about it to allay their fears of something being wrong! A cord blood sample may be taken for pH analysis [6]. Postpartum care is in the form of analgesics, bladder and bowel care, and thrombo-prophylaxis in case of high-risk women.

30.12.5 Complications

- Neonatal injuries like scalp abrasions, retinal hemorrhages, facial nerve palsy, hyperbilirubinemia, and neonatal jaundice are reported. These are generally self-contained and may be observed for spontaneous resolution most of the times.
- Hematoma confined to the skull bone and subgaleal hematoma are rare but may cause high morbidity and mortality in neonates.

Higher failure rates of operative vaginal delivery are associated with [6]

- Maternal BMI > 30
- Estimated fetal weight 4000+ g or clinically big baby
- Occipito-posterior position
- Higher station (mid-cavity) or when 1/5th+ of fetal head is palpable on abdominal examination

30.12.6 Vacuum Versus Forceps

- Vacuum needs less pain relief and leads to less perineal trauma including third degree tears [8].
- Less pelvic space is required for vacuum.
- Vacuum-increased risk of failure, more cephalhematoma, more retinal hemorrhages, more intracranial hemorrhages.
- Vacuum is more likely to fail delivery as compared to forceps.

References

1. Cheung YW, Hopkins LM, Caughey AB. How long is too long: does a prolonged second stage of labor in nulliparous women affect maternal and neonatal morbidity? Am J Obstet Gynecol. 2004;191:933–8.
2. Ferguson JKW. A study of the motility of the intact uterus at term. Surg Gynecol Obstet. 1941;73:359–66.
3. Laufe LE. Obstetric Forceps. New York: Harper & Row; 1968.
4. British Columbia Reproductive care program. Obstetric Guideline 14: Assisted vaginal birth: the use of forceps or vacuum extractor. Vancouver: BCRCP; 2001.
5. ACOG Practice Bulletin. Clinical management guidelines for obstetrician-gynecologists; Number 17, June 2000
6. RCOG Green-top Guideline No. 26
7. Cigna S, Gaba ND, Larsen JW. Get a handle on the scanzoni maneuver. Contemporary OB/GYN. May 26, 2016.
8. Johanson RB, Menon BKV. Vacuum extraction versus forceps for assisted vaginal delivery (Cochrane review). In: The Cochrane Library. Oxford: Update software; 2001. p. 2.

Caesarean Delivery

31

Niranjan Chavan

31.1 Introduction

Caesarean section is delivery of the baby through the abdominal route by making an incision on the uterine wall. Many papers have been published stating that the ideal caesarean rate should be between 10% and 15%. Despite this, both developing and developed countries are witnessing a tremendous increase in caesarean rate. The increase in caesarean rate can be attributed to the reluctance of obstetricians towards VBAC and giving operative vaginal delivery a try. However, there is no denying that caesarean sections when indicated significantly reduce maternal and foetal morbidity and mortality. Caesarean section does expose a woman to additional surgical and anaesthesia risks as compared to the physiological normal delivery. So, every caesarean section should be indicated, and the risks and benefits should be carefully evaluated.

31.2 History

Caesarean section has been reported in both western and non-western history. Greeks claim that Asclepius was born from his mother's abdomen directly by Apollo. This mythological reference was followed by the mention in Mauryan empire history from Indian. Chanakya was a wise advisor of King Chandragupta. When the king's wife was accidently poisoned while she was carrying Bindusara, Chandragupta delivered the prince by opening the abdomen. A more recent and documented reference of caesarean section comes from Siegershausen, Switzerland, in the 1580s. However, the origin of word 'caesarean' is unclear. It is likely that the term comes from the *Lex Regia* or royal law legislated by one of the early kings of Rome Numa Pompilius in 715 BC. This law proclaimed that women who die before delivering their infant had to have the infant delivered through the abdomen before burial. This law continued under the ruling of Caesars when it was called *Lex Caesarea* (Fig. 31.1).

Fig. 31.1 The extraction of Asclepius from the abdomen of his mother

N. Chavan (✉)
LTMMC and Sion Hospital,
Mumbai, Maharashtra, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_31

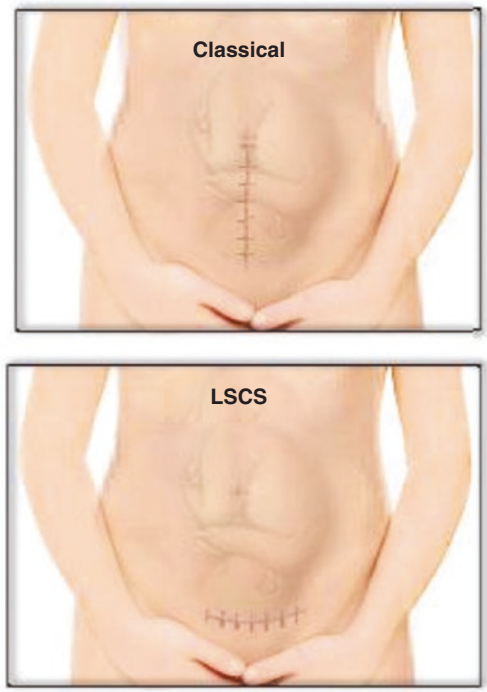

Fig. 31.2 Types of caesarean section

31.3 Types of Caesarean Section

Traditional caesarean section: Midline vertical incision is taken over the abdomen. Once the skin is incised, the uterus is also incised vertically, and the baby is delivered.

Lower uterine segment caesarean section (LSCS): As the name suggests, a transverse incision is taken on the abdomen followed by a transverse incision on the lower uterine segment. It minimises the risk of haemorrhage and incisional hernia and gives a cosmetic scar (Fig. 31.2).

31.4 Preoperative Considerations

- *Consent*: Obtaining informed consent is a process and not merely a medical record document. Indication, procedure and its complications and effect of caesarean delivery on future pregnancies must be discussed with the patient.
- Nil by mouth for at least 8 h before the planned procedure.
- *Investigations*: Complete blood count, blood grouping and crossmatching and antibody screening [1].
- Preoperative shaving of the incision site is not required. If the pubic hair over the proposed incision site is thick, it can be clipped short, rather than shaved.
- Antacid prophylaxis.
- *Antibiotic prophylaxis*: Single dose of a first-generation cephalosporin or ampicillin is given for both elective and emergency caesarean sections intravenously. ACOG states that prophylaxis has to be administered within 60 min prior to the start of planned caesarean delivery or after baby is delivered and the cord is clamped [2].
- The woman should be placed in a 15° left lateral tilt to avoid aorto-caval compression.
- American College of Obstetricians and Gynecologists (2010) do not recommend continuous foetal heart monitoring before a scheduled caesarean section. That said, foetal heart sounds should be documented in the operating room prior to surgery.

31.5 Planned Caesarean Section

31.5.1 Planned Vaginal Delivery vs. Planned Caesarean Section

If we compare a planned caesarean section with a vaginal delivery in a woman with uncomplicated pregnancy, it can be stated that:

Planned caesarean section may reduce the risk of the following in women:

- Perineal injury
- Injury to the vagina
- Early postpartum haemorrhage
- Obstetric shock

Planned caesarean section may increase the risk of the following in babies:

- Neonatal intensive care unit admission

Planned caesarean section may increase the risk of the following in women:

- Longer hospital stays
- Risk of anaesthesia
- Postoperative pain

31.5.2 Planning of Delivery

- Risk and benefits should be discussed with the patient.
- Informed consent should be taken after taking into consideration the clinical diagnosis, personal preferences and ethical issues.
- Refusal is patient's right and should be respected even when CS is clinically indicated.
- Record of all the clinical factors and patient counselling should be properly taken.

31.5.3 Indications of Planned Caesarean Section [3]

1. Breech presentation and transverse lie (Fig. 31.3)
   (a) Singleton breech or transverse lie of more than 38 weeks
   (b) Singleton breech or transverse lie with failed external cephalic version
   (c) Term breech presentation or transverse lie for whom external cephalic version is contraindicated
2. Multiple pregnancy [4]
   (a) If the first baby of the twin or triplet pregnancy is not cephalic, it is advisable to take for caesarean section
3. Placenta praevia
   (a) Minor or major placenta praevia
4. Morbidly adherent placenta

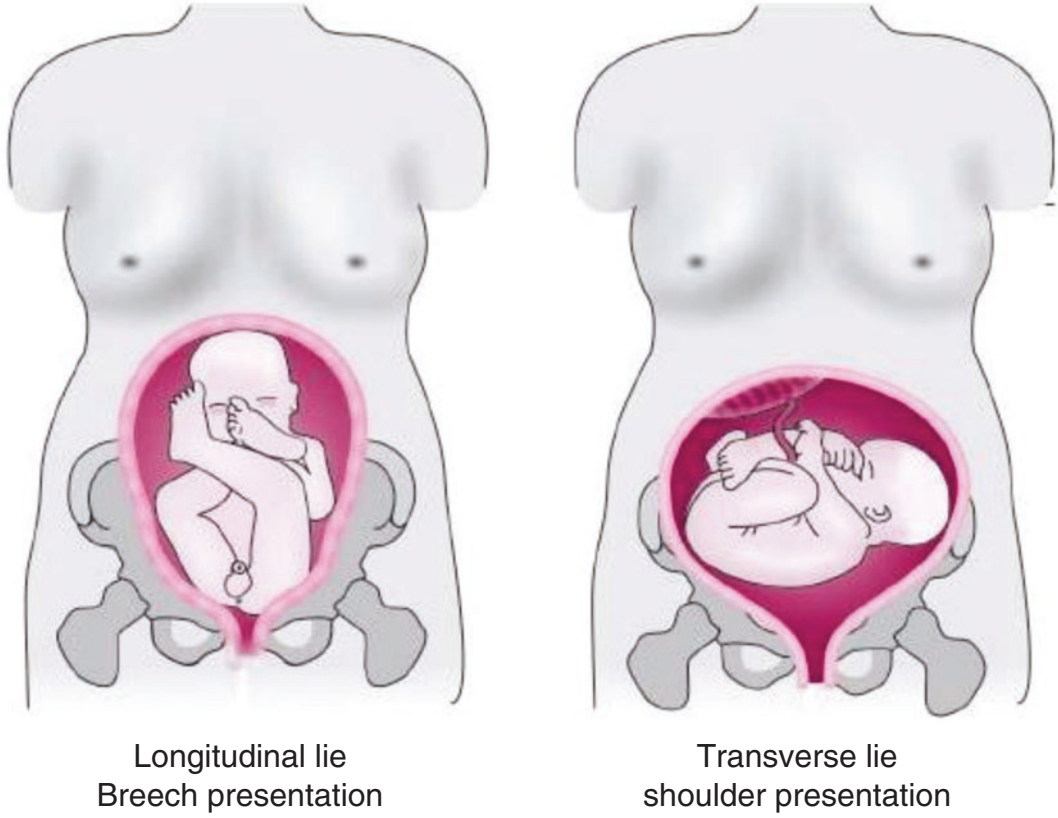

Fig. 31.3 Breech presentation and transverse lie common indications for planned LSCS

Urgency	Definition	Category
Maternal or fetal compromise	Immediate threat to life of woman or fetus	1
	No immediate threat to life of woman or fetus	2
	Requires early delivery	3
No maternal or fetal compromise	At a time to suit the woman and maternity services	4

Fig. 31.4 A classification relating the degree of urgency to the presence or absence of maternal or foetal compromise

(a) Every low-lying placenta should be evaluated for morbidly adherent placenta at 32–34 weeks

5. HIV and hepatitis
   (a) Caesarean section does not prevent vertical transmission
   (b) HIV positive with a viral load more than 400/mL
   (c) However, LSCS is indicated if there is a coinfection with hepatitis C to prevent vertical transmission
6. Herpes simplex virus
   (a) LSCS is indicated to prevent neonatal herpetic infections
7. Maternal request for caesarean section

Planned caesarean section should not be offered for the following indications [5]:

1. Preterm labour
2. Small for gestation age
3. Predictive CS for cephalic pelvic disproportion
4. Body mass index >50

31.5.4 Timing for Elective CS

Any baby delivered before term has a considerable risk for neonatal lung hypoplasia. So, elective LSCS should be postponed till 38 weeks until otherwise indicated.

31.6 Emergency Caesarean Section

31.6.1 Absolute Indication

1. Contracted pelvis or cephalopelvic disproportion
2. Pelvic mass causing obstruction for, e.g. cervical or broad ligament fibroid
3. Advanced carcinoma cervix
4. Vaginal obstruction (Fig. 31.4)

31.6.2 Relative Indication

1. Non-assuring foetal heart rate
2. Cord prolapse
3. Relative cephalopelvic disproportion
4. Antepartum haemorrhage
5. Previous 2 CS
6. Previous classical CS
7. Features of scar dehiscence
8. Dystocia leading to nonprogress of labour
9. Failed induction
10. Failed instrumental delivery
11. Bad obstetric history (Fig. 31.5)

31.6.3 Timing of Unplanned Caesarean Section

- 30 min for CS category 1
- CS category 2 within 75 min of making the decision
- Category 3 or 4: timing depending upon the maternal and foetal conditions

31.7 Intraoperative Management

31.7.1 Anaesthesia

- Spinal anaesthesia is preferred over general anaesthesia as it results in lesser maternal morbidity except few cases like ecalampsia where there are chance of convulsions on

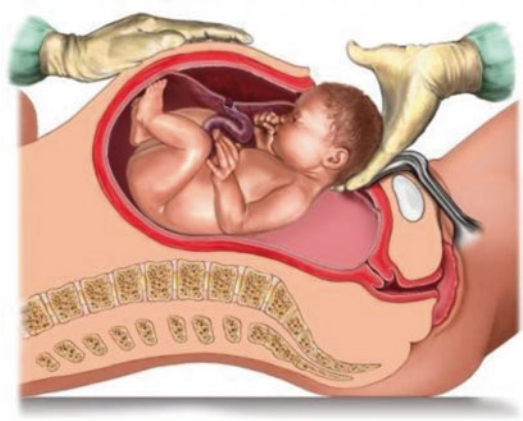

Fig. 31.5 Delivering the baby in LSCS

table and dropping fetal heart rates where every second counts to save the fetus.

- To counter the hypotension occurring during spinal anaesthesia, the patient should be properly hydrated, and the use of ephedrine is also advised.
- Antiemetics and antacids should be given as premedications.
- General anaesthesia should be preceded by preoxygenation, cricoid pressure and rapid induction.

31.7.2 Position

Supine position with a lateral tilt of 15°.

31.7.3 Surgical Techniques

- Prophylactic antibiotics should be administered before skin incision. Co-amoxiclav is the antibiotic of choice if not otherwise indicated.
- Transverse abdominal incision is preferred because of its cosmetic effect and less postoperative pain.
- Joel Cohen incision (a straight skin incision, 3 cm above the symphysis pubis) is the advised transverse incision.
- If the lower uterine segment is well formed, blunt extension of the incision in a smiley fashion is better than sharp extension as it might reduce the chance of haemorrhage.
- Oxytocin 5 IU by slow intravenous injection should be given after the delivery of the baby (Fig. 31.6).
- At CS, Manual removal of placenta is not advised unless indicated as it is associated with higher incidence of endometritis.
- Exteriorising the uterus for closure causes more pain. Intraperitoneal closure of the uterus should be done as it gives a better chance for the uterus to contract and minimises chances of infection [6].
- Uterus incision should be sutured in two layers.
- Both visceral and parietal peritoneum closures are not advisable.
- Subcutaneous tissue should only be closed if the fat layer is more than 2 cm (Fig. 31.7).

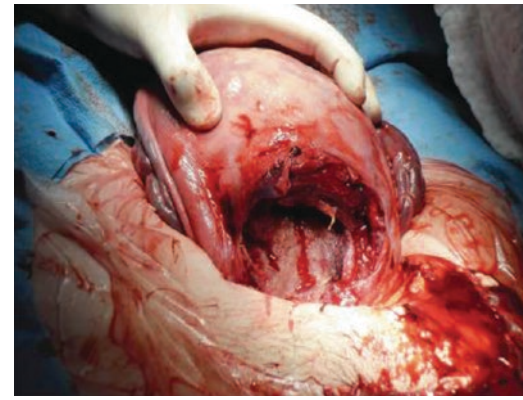

Fig. 31.6 Uterus after placenta removal with minimal bleeding

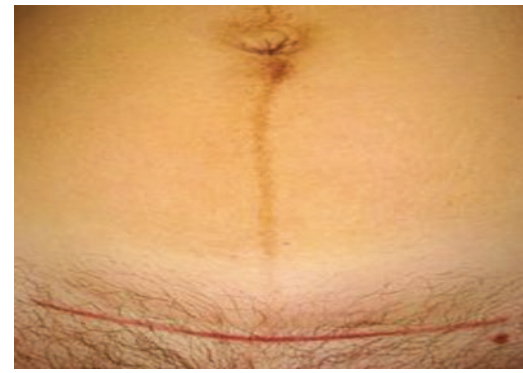

Fig. 31.7 A 7-week-old caesarean section scar and linea nigra visible on a 31-year-old mother

31.8 Postoperative Management

- After caesarean section the patient should be monitored half hourly for initial 2 h and one hourly for the next 4 h. General condition, pulse, BP, temperature, abdominal girth, uterine tone, per vaginal bleed and urine output should be diligently charted.
- Proper pain management is important.

31.9 Peripartum Management

- *Intravenous fluids*: Patients should be well hydrated. If the patient was kept NBM before the elective surgery, at least three pints of IV fluids should be transfused. As spinal anaesthesia causes sudden hypotension, it is advisable to transfuse at least 2–3 L during the surgery. Any crystalloid can be used typically; at least 2–3 L is infused during surgery. Postoperatively, 3 L of fluid should prove adequate during the first 24 h after surgery. Urine output should be maintained to a minimum of 30 cc/h.
- Close monitoring of the amount of vaginal bleeding is necessary for at least an hour in the immediate postoperative period.
- Abdominal examination should be done to check the tone of the uterus postoperatively.
- Criteria for transfer to the postpartum ward include minimal bleeding, stable vital signs and adequate urine output.
- In postnatal ward, monitoring should be continued and four hourly documentation of vitals, abdominal girth, uterine tone, PV bleed and urine output is necessary.
- The haematocrit is routinely measured the morning after surgery.
- The Foley catheter can be removed after 12 h of surgery if patient's urine output was satisfactory.
- In uncomplicated cases, solid food may be offered within 8 h of surgery.
- Early ambulation lowers the risk of venous thromboembolism.
- The incision is inspected on the fourth postoperative day.
- The patient can be discharged on day 4 of surgery if there is no puerperal complication, and breastfeeding is properly initiated.

31.10 Complications

31.10.1 Intraoperative Complications

1. Anaesthesia related:
   (a) Aspiration syndrome
   (b) Hypotension
   (c) Spinal headache
2. Haemorrhage:
   (a) Uterine vessel damage
   (b) Uterine atony
   (c) Lacerations: uterine, vertical laceration into vagina, broad ligament
3. Injury to the bladder especially common in previous LSCS, in low vertical incision and in long obstructed labour (Fig. 31.8)

31.10.2 Postoperative Complications

- Urinary tract infection
- Stress incontinence (occurs in about 4% of women after CS)
- Uterovesical and vescicovaginal fistula

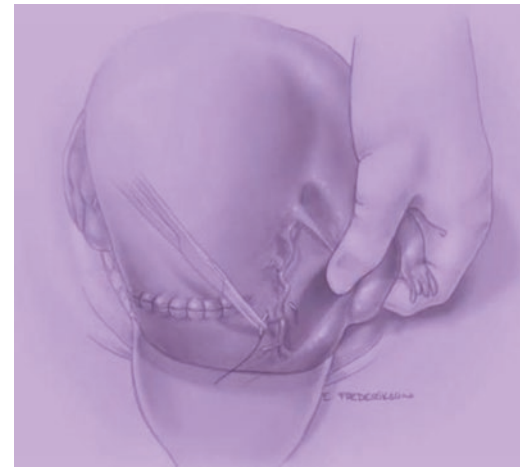

Fig. 31.8 Uterine artery ligation to control PPH

- Endometritis
- Irregular vaginal bleeding
- Thromboembolic disease

References

1. Caesarean section. NICE guidelines, Published 23 Nov 2011. nice.org.uk/guidance/cg132.
2. Safe Prevention of the Primary Cesarean Delivery. American Congress of Obstetricians and Gynecologists and the Society for Maternal-Fetal Medicine. Mar 2014. Retrieved 20 Feb 2014.
3. Lavender T, Hofmeyr GJ, Neilson JP, Kingdon C, Gyte GM. Caesarean section for non-medical reasons at term. Cochrane Database Syst Rev. 2012;3:CD004660. https://doi.org/10.1002/14651858.CD004660.pub3.
4. Biswas A, Su LL, Mattar C. Caesarean section for preterm birth and, breech presentation and twin pregnancies. Best Pract Res Clin Obstet Gynaecol. 2013;27(2):209–19. https://doi.org/10.1016/j.bpobgyn.2012.09.002.
5. http://www.acog.org/Resources-And-Publications/Committee-Opinions/Committee-on-Ethics/Elective-Surgery-and-Patient-Choice.
6. Bamigboye AA, Hofmeyr GJ. Closure versus non-closure of the peritoneum at caesarean section: short- and long-term outcomes. Cochrane Database Syst Rev. 2014;8:CD000163. https://doi.org/10.1002/14651858.CD000163.pub2.

Breech in Labor

32

Geetha Balsarkar and Nirmal Nitin Gujarathi

32.1 Overview and Purpose

Breech presentation is defined as a fetus in a longitudinal lie with the buttocks or feet closest to the cervix. This occurs in 3–4% of all deliveries. The percentage of breech presentation decreases with advancing gestational age from 22–25% of births prior to 28 weeks' gestation to 7–15% of births at 32 weeks' gestation to 3–4% of births at term.

32.2 Etiology for Breech Presentation

Maternal:

- Uterine anomalies like septate uterus and bicornuate uterus
- Fibroids
- Previous breech deliveries
- Nulliparity

Placental:

- Placenta previa
- Cornu-fundal placentation

Fetal:

- Prematurity
- CNS malformation
- Neck masses
- Aneuploidy
- Oligo-/polyhydramnios

Fetal abnormalities are observed in 17% of preterm breech deliveries, and at term it's 9%. Perinatal mortality is 2–4 times higher and mostly associated with malformations and prematurity, irrespective of mode of delivery.

32.3 Types of Breech Presentation

- Frank breech (50–70%): Hips flexed, knees extended
- Complete breech (5–10%): Hips flexed, knees flexed
- Footling or incomplete (10–30%): One or both hips extended, foot presenting

G. Balsarkar (✉) · N. N. Gujarathi
Department of Obstetrics and Gynecology,
Nowrosjee Wadia Maternity Hospital, Seth
G.S. Medical College, Mumbai, Maharashtra, India

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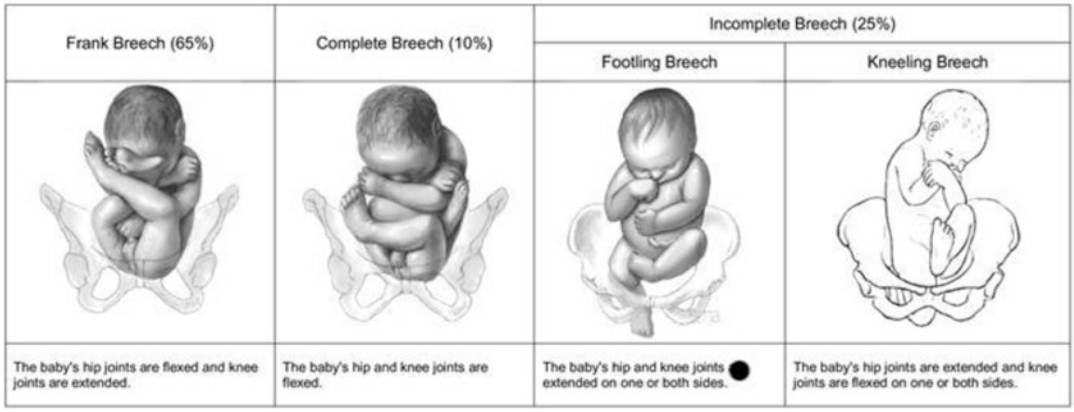


32.3.1 Clinical Presentation

Clinical diagnosis of breech presentation may be difficult by palpation alone. Features suggestive of breech are:

- History of subcostal discomfort with solid, non-ballotable, fetal pole palpable at the uterine fundus.
- Fetal heart sound auscultation above or around the umbilicus.
- Palpation of fetal ischial tuberosities, sacrum, and anus on vaginal examination.
- Such observations are imprecise, and it is estimated that 30% of breech presentations are not diagnosed until onset of labor. As abdominal palpation has a sensitivity of 28% and specificity of 94%, ultrasound examination remains the gold standard.

32.3.2 Mode of Delivery

Dilemma starts when patient comes to an obstetrician with breech presentation. There was a dictum "Once a breech, always cesarean delivery." Till (1959), breech vaginal delivery was the rule. After **Wright** study showed reduction in perinatal morbidity and mortality with cesarean delivery for breech presentation, cesarean delivery was proposed instead of vaginal breech delivery [1].

Multiple factors are to be considered prior to deciding the route of delivery for breech fetuses. Factors include fetal characteristics, pelvic dimensions, coexistent pregnancy complications, operator experience, patient preference, and hospital capabilities.

32.4 Term and Preterm Breech Fetuses

Preterm babies have different set of risk factors compared to their term counterparts. So it becomes mandatory separate and discuss.

32.4.1 Term Breech Fetus

Overall data regarding the superior perinatal outcome with respect to planned cesarean delivery are conflicting. Term breech trial collaborative group (Hannah 2000) [2] has influenced our thinking pattern regarding vaginal breech delivery. It showed that planned cesarean delivery was associated with lower risk of perinatal mortality compared to planned vaginal delivery—3 per 1000 versus 13 per 1000. Cesarean delivery was associated with a lower serious neonatal morbidity—1.4% versus 3.8%. Critics have demonstrated fallacies in term breech trial, so ACOG has to modify its stance on breech presentation, and now it is

recommending that "the decision regarding the mode of delivery should depend on the [2] experience of the health care provider" and "that planned vaginal delivery of a term singleton breech fetus may be reasonable under hospital-specific protocol guidelines."

In contrast, the presentation et Mode d'Accouchement (PREMODA) study showed no difference in corrected neonatal mortality rates and neonatal outcomes according to delivery mode. In spite of evidences on both sides of debate, rates of planned vaginal delivery attempts continue to decline [3].

32.4.2 Preterm Breech Fetuses

There are no randomized studies regarding preterm breech deliveries; planned cesarean delivery appears to confer a survival advantage. Reddy and associates reported data from the National Institutes of Health, retrospective multicenter cohort study (2012), for deliveries between 24 and 32 weeks of gestation. Fetuses within these gestational ages showed low completion rate, and those who completed were associated with higher neonatal mortality rates compared with planned cesarean delivery. The Maternal Fetal Medicine Committee of the Society of Obstetricians and Gynaecologists of Canada recommends that vaginal breech delivery is reasonable when the estimated fetal weight is >2500 g [4].

32.4.3 Delivery Complications

1. Maternal Morbidity and Mortality: Increased rate of maternal morbidity and perinatal morbidity is anticipated with breech delivery, either vaginal or cesarean. In cesarean delivery, hysterotomy incision gets extended while delivering fetal head, irrespective if manual or with forceps. In vaginal delivery, vaginal wall or cervical lacerations and tears are common. Manipulation during vaginal delivery may cause extended lacerations and episiotomy extensions. Uterine atony and postpartum hemorrhage can occur.
2. Perinatal Morbidity and Mortality: Preterm delivery and breech presentation are common associations. In addition, birth trauma can contribute to mortality. There is no difference between routes of delivery. Fractures of the humerus, clavicle, and femur are more common. In some cases, traction may separate epiphyses. Some rare injuries like upper extremity paralysis, skull fractures, spinal cord injuries, and abdominal visceral injuries are noted.

32.4.4 Imaging Techniques

Unlike cephalic presentation, aftercoming head in breech delivery doesn't undergo molding. To avoid entrapment of aftercoming head, pelvimetry becomes important. In addition to this fetal size, the type of breech and degree of neck flexion or extension become important:

1. Sonography: Usually performed as part of prenatal care. If not done gross fetal anomalies like hydrocephalus or anencephaly can be ruled out prior to planned vaginal breech delivery. Head flexion can also be determined by sonography. Extension of the head is contraindication for vaginal breech delivery. If sonographic imaging is uncertain, then two-view radiography of the abdomen is useful for head inclination. Biparietal diameter of >90–100 mm is considered as exclusion criteria for vaginal breech delivery as per Roman et al. [5].
2. Pelvimetry: Nowadays, bony pelvis assessment can be done by one-view computed tomography, MRI, or plain film radiography. As per Azria (2012), inlet diameters of the pelvis should be as follows: inlet anteroposterior >105 mm, inlet transverse diameter >120 mm, and midpelvic interspinous

diameter >100 mm. Others use maternal-fetal biometry correlation. Michel et al. defined values as follows: the sum of the inlet obstetrical conjugate minus the fetal BPD is >15 mm, the inlet transverse diameter minus the BPD is >25 mm, and the midpelvis interspinous diameter minus BPD is >0 mm [6].

32.4.5 Decision-Making

As per the ACOG guidelines, risk versus benefits should be discussed with the patient and relatives prior to making any decision regarding the route of delivery.

Factors favoring cesarean delivery for breech presentation are:

1. Lack of operator experience
2. Patient request for cesarean delivery
3. Large fetus: >3800–4000 g
4. Viable healthy preterm fetus
5. Severe FGR
6. Fetal anomaly incompatible with vaginal delivery
7. Prior perinatal or neonatal death/trauma
8. Incomplete or footling breech
9. Hyperextended head
10. Unfavorable pelvis
11. Prior cesarean

32.5 Management of Labor and Delivery

32.5.1 Vaginal Breech Delivery

There are three types of vaginal breech deliveries:

1. Spontaneous breech delivery: This occurs in preterm deliveries and abortions. No traction or manipulation of infant is required.
2. Assisted breech delivery: The most common type of vaginal breech delivery. In this type, the fetus is allowed to be delivered up to umbilicus, spontaneously, and then maneuvers are initiated to assist in the delivery for the body, arms, and head.
3. Total breech extraction: In this type, fetal feet are grasped and the entire fetus is extracted. Nowadays, this is used only for delivering the second non-cephalic twin. In non-dilated cervix with singleton pregnancy, delivering fetal head becomes problematic. So, it can't be used in such cases. This is associated with 25% of birth injury and 10% of mortality rate.

32.5.2 Labor Induction and Augmentation

Induction of labor is controversial in breech presentation. Marzouk and associates [7] found similar perinatal outcome with spontaneous labor or with cervical preparation and augmentation of labor. As per Kotaska, some protocols agree with augmentation and some avoid it. At our institution, we prefer cesarean delivery for healthy, viable preterm/term fetuses [7].

32.5.3 Management of Labor

Rapid assessment in receiving room/Obs emergency room should be made with regard to membranes, labor, and fetal condition. Surveillance in view of fetal heart rate and uterine contractions should be done on admission. Arrangement for senior or expert obstetrician, anesthetist, and neonatologist should be done.

Intravenous lines should be accessed and crystalloid infusions should be started. Cervical dilatation and effacement should be assessed, as it will help in decision-making, with regard to the route of delivery. In early labor, pelvimetry and sonographic assessment should be done, as it will be complementary in decision-making.

Fetal heart rate should be monitored by electronic fetal heart monitoring. If not available, then every 15 min intermittent auscultation is required.

Cord prolapse risk should be always kept in mind, especially when it is a frank breech case. Therefore, ARM should be gradual, and post-ARM fetal heart tracing and monitoring for the first 5–10 min is mandatory.

Scoring system was developed by Zatuchni and Andros for prediction of successful vaginal breech delivery.

According to ZA scoring, if score is less than 4, then cesarean delivery is recommended [8].

Zatuchni-Andros Breech Scoring

	Add 0 Points	Add 1 Point	Add 2 Points
Parity	0	1	2
Gestational age, wk	39+	38	<37
EFW, lb	8	7–8	<7
Previous breech	0	1	2
Dilation	2	3	4
Station	–3	–2	–1

If the score is 0–4, cesarean delivery is recommended.

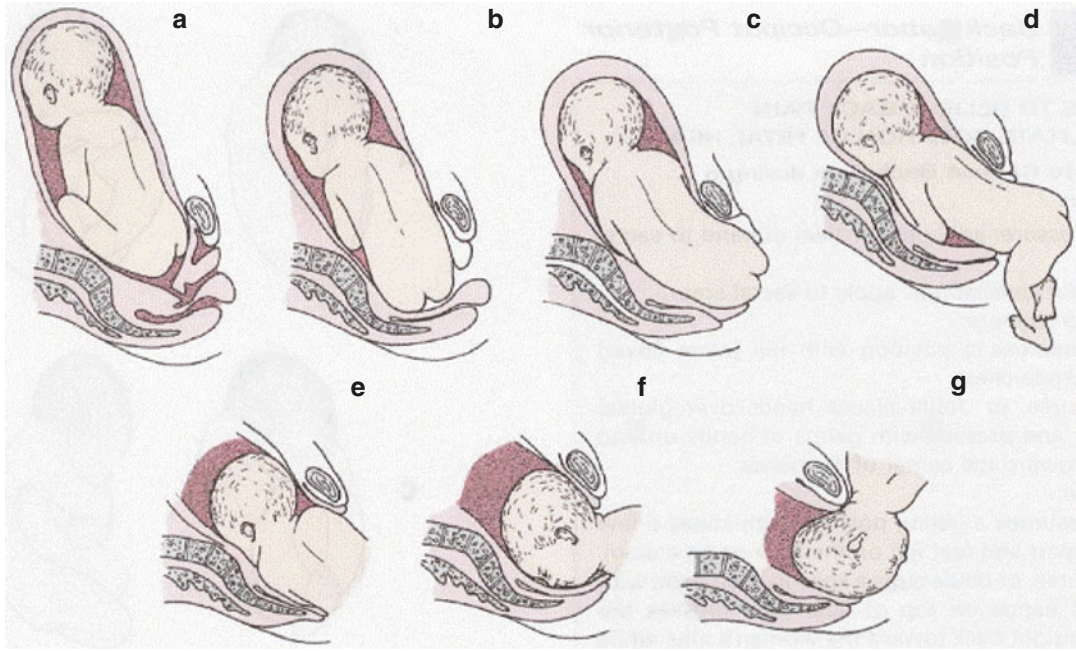
32.5.4 Cardinal Movements with Breech Delivery

Engagement and descent occur in one of the oblique pelvic diameters with bitrochanteric diameter. Anterior hip descends rapidly than the posterior hip. When it meets the pelvic floor, internal rotation of 45° occurs, which allows bitrochanteric diameter to be in anteroposterior diameter of pelvic outlet.

After rotation, descent continues until the perineum is distended by advancing breech and the anterior hip appears at the vulva, called as "climbing of the perineum". By lateral flexion of the fetal body, posterior hip is then forced over the perineum, allowing the infant to straighten out when the anterior hip is born. Legs and feet follow breech and can be born spontaneously or with assistance.

After breech birth, minimal external rotation occurs, which helps back to turn anteriorly and then shoulders get engaged in one of the oblique diameters of the pelvis. Shoulders descend rapidly and undergo internal rotation, with bisacromial diameter occupying anteroposterior plane.

Following shoulders, the head gets engaged in one of the oblique diameters and then rotates to bring the nape of the neck under the pubic symphysis. The head is delivered by flexion.



32.5.5 Partial Breech Extraction

Ideally, episiotomy should be given whenever partial breech extraction or assisted breech delivery is to be done. Breech is allowed to be delivered till the umbilicus. It helps in reducing morbidity, due to undue manipulations. Delivery of breech draws the umbilical cord which gets compressed in the vagina. Therefore, once breech crosses the introitus, the abdomen, thorax, arms, and head must be delivered promptly.

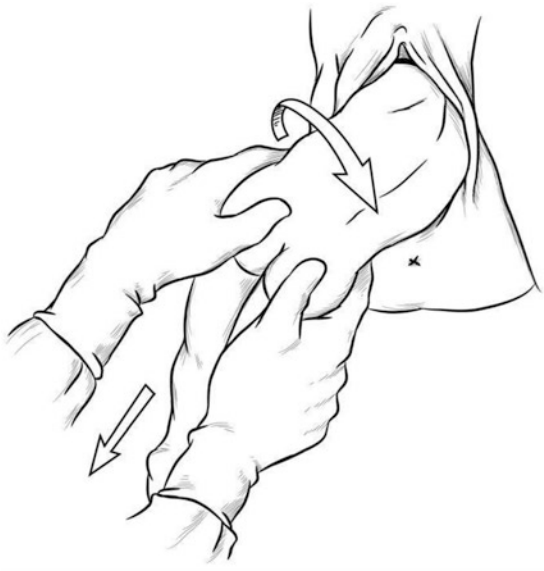
In order, the posterior hip delivers first, followed by the anterior hip, and then external rotation makes the fetus in a sacrum anterior position.

The legs of the fetus are delivered by splinting the medial aspect of the femur and by exerting pressure laterally to sweep each leg away from the midline.

Following leg delivery, the fetus is grasped with hand and thumbs at anterior superior iliac crests and sacrum, respectively, in a fashion to avoid abdominal soft tissue trauma. Wet towel helps in grasping the fetus. Maternal pushing efforts and extraction should be coordinated.

32.5.6 Delivery of the Arms

The rule of thumb of a successful breech delivery is steady, gentle, downward traction until the lower edges of the scapulas are delivered spontaneously. Unless one axilla is visible, no attempt should be made to deliver the shoulder and arm. Once the axilla is visible, shoulder and arm delivery can be done. There are two methods in the delivery of arms, depending on which shoulder delivers first. In the first method, the trunk is rotated in such a way that the anterior shoulder and arm appear at the vulva and are easily released. Then, the body of the fetus is rotated in 180°, and the posterior shoulder and arm are released. This is called as Lovset's maneuver.



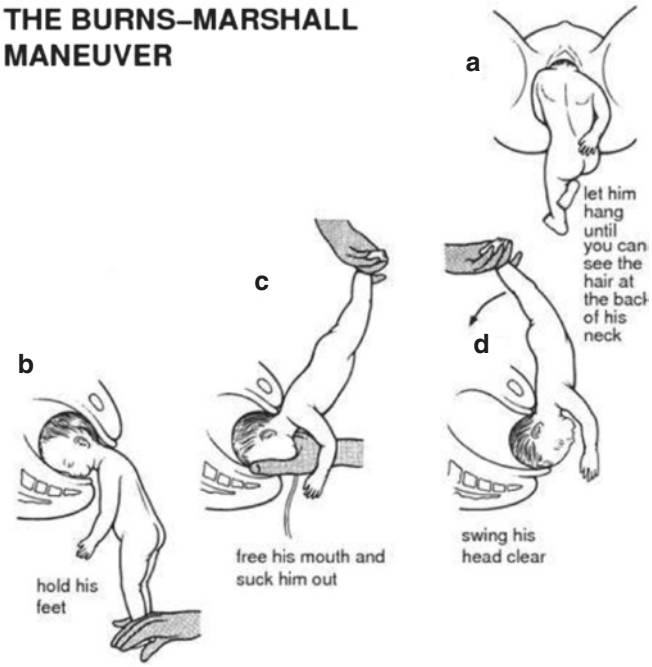
In the second method, as breech extraction continues, an upward traction is employed with the fetus drawn to the mother's left thigh, which helps in delivering the posterior shoulder and arm. The fetal body is depressed and the delivery of anterior shoulder follows.

Delivery of the arms is not always easy, so in difficult cases, the posterior arm should be freed first, as more space is available on the posterior aspect of the pelvis.

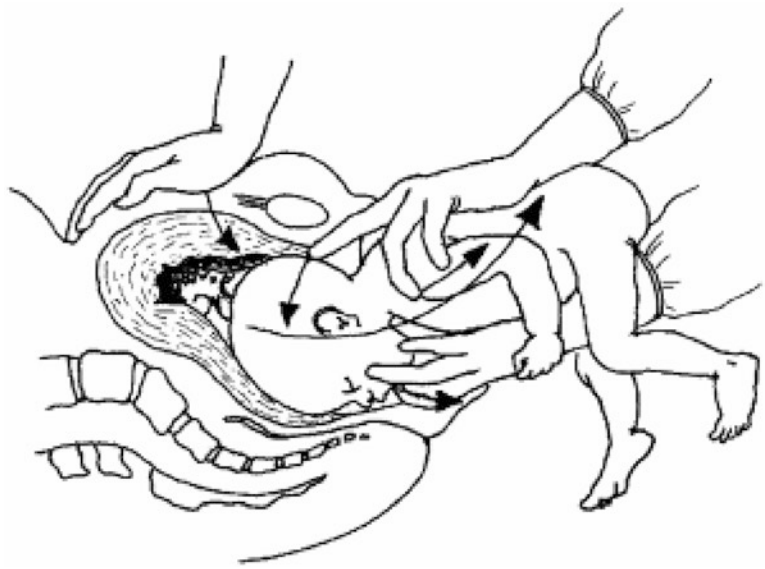
Nuchal arms—One or both fetal arms occasionally may be found around the back of the neck and impacted in pelvic inlet, called as nuchal arm. In such cases the fetus should be rotated by half circle in direction to release the nuchal arm. If this is not successful, then extraction can be done by hooking the arm with fingers and forcing the arm over the shoulder and down the ventral surface for the delivery of the arm. Fracture of the humerus or clavicle can occur during this maneuver.

32.5.7 Delivery of Aftercoming Head

- *Burn-Marshall Maneuver*: In this technique, the leg, the trunk, and the shoulders are delivered, remaining the head. The head is allowed to hand until the nape of the neck. The feet are grasped, and with gentle traction, it is swept over the mother's abdomen. This leads to fetal head delivery.

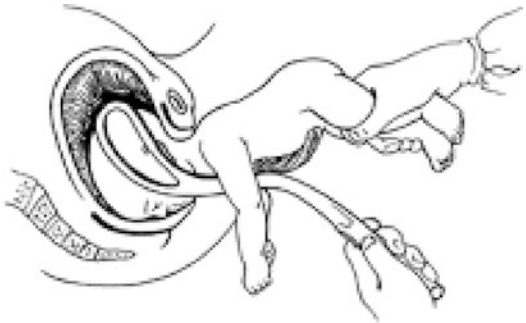


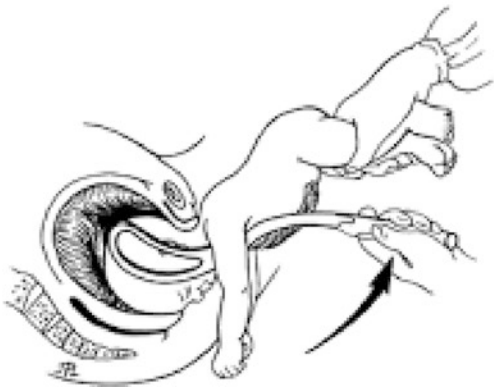
- *Mauriceau Maneuver*: The index and middle finger of one hand are applied over the maxilla, to flex the head, while the fetal body rests on the palm of the hand and forearm. Two fingers of the other hand are hooked over the fetal neck, and grasping shoulders, downward traction is applied till the suboccipital region comes below the pubic symphysis. The assistant should give gentle suprapubic pressure to maintain head flexion; the fetus is then elevated toward the maternal abdomen, and the mouth, nose, brow, and occiput are delivered, respectively, over the perineum.



32.5.8 Forceps for Aftercoming Head

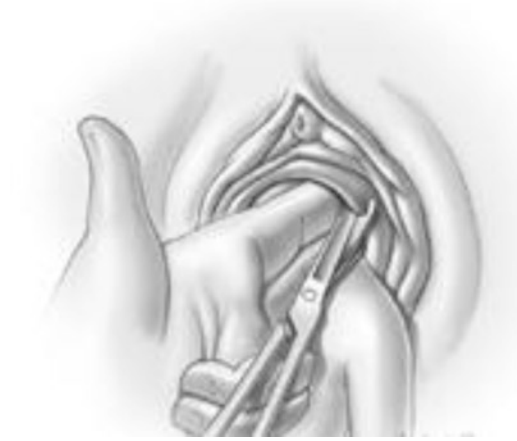
Piper forceps or divergent Laufe forceps may be used electively or when Mauriceau maneuver cannot be accomplished. Blades should be applied directly to engage the head while keeping it flexed with suprapubic pressure. The assistant has to hold the body of the fetus and arms wrapped in towel, so that it does not come in way of forceps application or traction. Piper forceps are used directly in one-knee kneeling position. Once applied, the head is delivered by gently pulling outward and raising handle simultaneously. This helps in delivering the face over the perineum. The body and head should move in unison to minimize trauma.





32.5.9 Entrapment of Aftercoming Head

In small preterm fetuses, incomplete cervical dilatation causes entrapment of aftercoming head. In this situation, manually, the cervix should be pushed over the occiput. If this fails, then Duhrssen's incision should be taken on the cervix, and the head should be delivered. Other options are IV nitroglycerine for cervical relaxation. General anesthesia with halogenated agents can be helpful. Zavanelli maneuver can be useful in some cases. Symphysiotomy can also be used as an aid for entrapped aftercoming head.



32.5.10 Total Breech Extraction

Total extraction may be required sometimes, in cases of complete or incomplete breech. A hand has to be introduced, the feet have to be grasped, and the ankles are held with the second finger lying in between. The feet are brought out through the introitus with gentle traction.

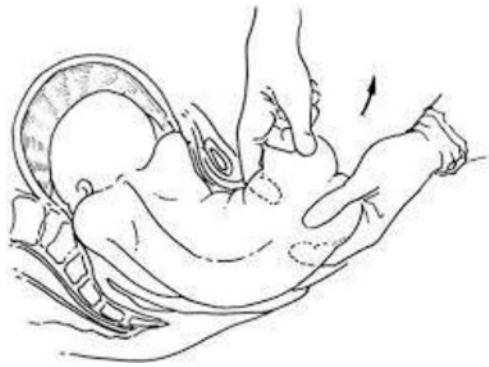
As legs begin to emerge, downward traction continues. As the buttocks appear at the introitus, traction is applied till the hips are delivered. The fetal back rotates to anterior. Breech extraction is completed in a similar way as it is done in partial breech extraction.

In cesarean delivery all these maneuvers are used through hysterotomy incision.

32.5.11 Frank Breech

Moderate traction is exerted in each groin by a finger and is aided by episiotomy. Once breech is pulled out, steps of partial breech extraction are followed. If this doesn't work, then breech

decomposition has to be used, which converts frank breech into footling breech within the birth canal. It may be difficult in cases of PROM where amniotic fluid is less or in cases of oligohydramnios. Uterine relaxants like general anesthesia, magnesium sulfate, or betamimetic agents can be used. Pinard maneuver helps in breech decomposition. In Pinard maneuver, two fingers are inserted along one extremity to the knee, which is then pushed away from the midline after spontaneous flexion. With traction the foot is delivered.



32.6 Version

Changing fetal presentation by physical manipulation, either by substituting one pole by the other, in longitudinal presentation, or converting transverse or oblique lie into longitudinal presentation. In external version, manipulations are done through the abdominal wall, and in internal version, it is done inside the uterine cavity.

32.6.1 External Cephalic Version (ECV)

As per the American College of Obstetricians and Gynecologists (2012), versions should be offered to all near-term breech presentations. Average success rate for version is 58% with range of 35–86%. Success rate is higher for transverse lie fetuses.

Indications: Breech presentation at or around 36 weeks. If done prior to it, allows a breech to return, and at 36 weeks, if any iatrogenic complication occurs, immediate delivery can be done.

Contraindication: Conditions where vaginal delivery is contraindicated, e.g. placenta previa or non-reassuring fetal status, uterine malformations, multiple gestation, uterine bleeding, and rupture of membranes. Prior uterine surgeries come under relative contraindication.

Factors favoring successful outcome include multiparity, adequate amniotic fluid, fetal weight between 2500 and 3000 g, unengaged presenting part, and nonobese patient [9].

Scoring system was developed to predict successful outcome of ECV.

Scores to Predict External Cephalic Version Success

	Add 0 Points	Add 1 Points	Add 2 Points
Parity	0	1	>2
Dilation	>3 cm	1–2 cm	0 cm
EFW, g	<2500	2500–3500	>3500
Placenta	Anterior	Posterior	Lateral/fundal
Station	>–1	–2	<–3

32.6.1.1 Complications

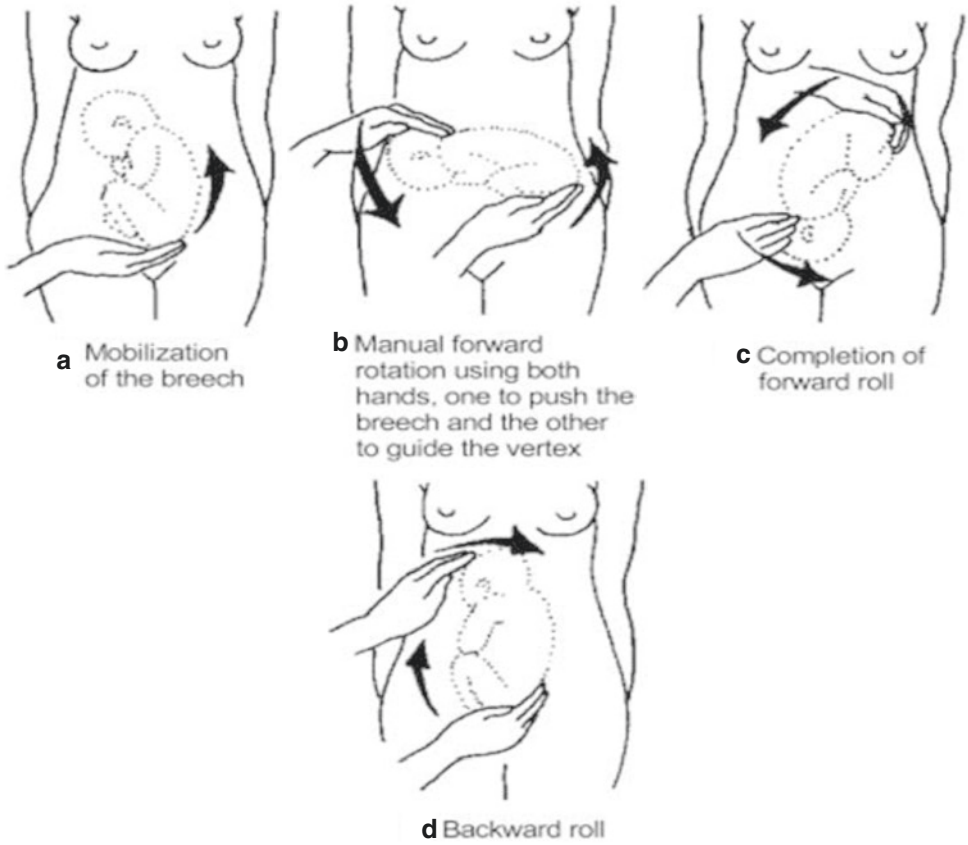
Prior counseling to the patient and relatives regarding success rates, conversion back to breech, and procedural risks should be done. Procedural risks include abruptio placentae, uterine rupture, fetomaternal hemorrhage, preterm labor, fetal compromise, amniotic fluid embolism, and rarely maternal death. Fetal death is a very rare complication.

32.6.1.2 Technique

ECV should be done in setting where emergency cesarean delivery setup is available. Prior to procedure sonographic examination should be performed to assess presentation, amniotic fluid index, fetal anomalies, and placental locations. Rh-D-negative women should receive anti-D immunoglobulin.

Forward roll: Each pole of the fetus is held with one hand; the breech is dislodged from the maternal pelvis and gently displaced laterally, in clockwise direction toward the fundus; and fetal head is directed toward the pelvis.

Backward flip: If forward roll is unsuccessful, then backward flip is attempted, which is the opposite of forward roll.



Procedure should be done, preferably, in the operation theater. Version attempts should not be continued if patient complains of excessive discomfort and abnormal fetal heart rate.

After version nonstress test should be done immediately, to check for fetal distress.

32.6.1.3 Tocolysis

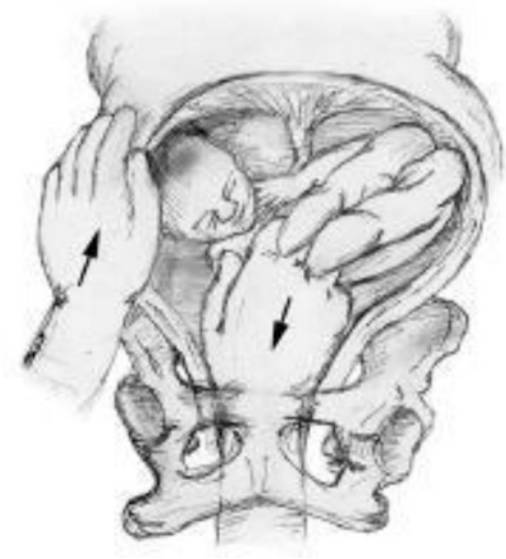
The American College of Obstetricians and Gynecologists suggests the use of tocolytic agents during the ECV procedure (2012). Betamimetics like terbutaline, ritodrine, or salbutamol, calcium channel blockers like nifedipine, and nitric oxide

donors like nitroglycerine can be used. As per Fernandez et al., terbutaline is better than any other drug. As terbutaline is not available, at our institution we use nifedipine or isoxsuprine [10].

ACOG (2012) says there is not enough evidence to recommend conduction analgesia routinely for ECV.

32.6.2 Internal Podalic Version (ICV)

In modern obstetrics, this maneuver is used only for delivering second twin. It should be done preferably with intact membranes. A hand is inserted inside the uterine cavity. Accoucher holds one or both feet and gently pulls them through completely dilated cervix. The other hand should be used to push the fetus transabdominally. This is followed by breech extraction.



References

1. Wright RC. Reduction of perinatal mortality and morbidity in breech delivery through routine use of cesarean section. Obstet Gynecol. 1959;14:758–63.
2. Hannah ME, Hannah WJ, Hewson SA, et al. Planned caesarean section versus planned vaginal birth for breech presentation at term: a randomised multicentre trial. Lancet. 2000;356:1375–83.
3. Goffinet F, Carayol M, Foidart J-M, et al. Is planned vaginal delivery for breech presentation at term still an option? Results of an observational prospective survey in France and Belgium. Am J Obstet Gynecol. 2006;194:1002–11.
4. Reddy UM, Zhang J, Sun L, et al. Neonatal mortality by attempted route of delivery in early preterm birth. Am J Obstet Gynecol. 2012;207:117.e1–8.
5. Roman H, Carayol M, Watier L, et al. Planned vaginal delivery of fetuses in breech presentation at term: prenatal determinants predictive of elevated risk of cesarean delivery during labor. Eur J Obstet Gynecol Reprod Biol. 2008;138:14–22.
6. Azria E, Le Meaux J-P, Khoshnood B, et al. Factors associated with adverse perinatal outcomes for term breech fetuses with planned vaginal delivery. Am J Obstet Gynecol. 2012;207:285.e1–9.
7. Marzouk P, Arnaud E, Oury J-F, Sibony O. [Induction of labour and breech presentation: experience of a French maternity ward]. J Gynecol Obstet Biol Reprod. 2011;40:668–74
8. Zatuchni GI, Andros GJ. Prognostic index for vaginal delivery in breech presentation at term. Am J Obstet Gynecol. 1967;98:854–7.
9. Buhimschi CS, Buhimschi IA, Wehrum MJ, et al. Ultrasonographic evaluation of myometrial thickness and prediction of a successful external cephalic version. Obstet Gynecol. 2011;118:913–20.
10. Fernandez CO, Bloom SL, Smulian JC, et al. A randomized placebo-controlled evaluation of terbutaline for external cephalic version. Obstet Gynecol. 1997;90:775–9.

Cord Prolapse and Transverse Lie

33

Aswath Kumar and Neetha George

33.1 Cord Prolapse

33.1.1 Definition

- Umbilical cord (funic) presentation—when the cord felt alongside the presenting part or below it in labor and membranes not ruptured
- Occult cord prolapse—when the membrane ruptured and the cord felt alongside the presenting part
- Overt cord prolapse—when the cord felt below the presenting part with membranes ruptured; the cord may be in the vagina or outside the introitus (Fig. 33.1)

33.1.2 Incidence

0.6% of all deliveries

33.1.3 Etiology

The occurrence of cord prolapse is most commonly associated with:

1. Breech, transverse, oblique, unstable lie, face, brow
2. A high head at the onset of labor
3. Multiple gestation—second of the twins
4. Grand multiparity
5. Abnormal placentation
6. Preterm labor, rupture of membranes
7. Polyhydramnios
8. Obstetric manipulations such as ARM, ECV, IPV, manual rotation of fetal head, forceps delivery, application of scalp electrode, and IUPC
9. CPD, contracted pelvis
10. Long umbilical cord
11. Fetal anomalies
12. Male fetus

33.1.4 Pathophysiology

Cord compression or spasm of the cord (handling cord, atmospheric temperature) leads to hypoxia which is manifested as prolonged abrupt bradycardia or repetitive variable decelerations in CTG and meconium passage, low APGAR, and birth asphyxia leading to sudden IUD, neonatal death, or morbidity.

33.1.5 Diagnosis

The diagnosis is commonly made during a vaginal examination; the examiner feels a soft, usually

A. Kumar (✉) · N. George
Jubilee Mission Medical College,
Thrissur, Kerala, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_33

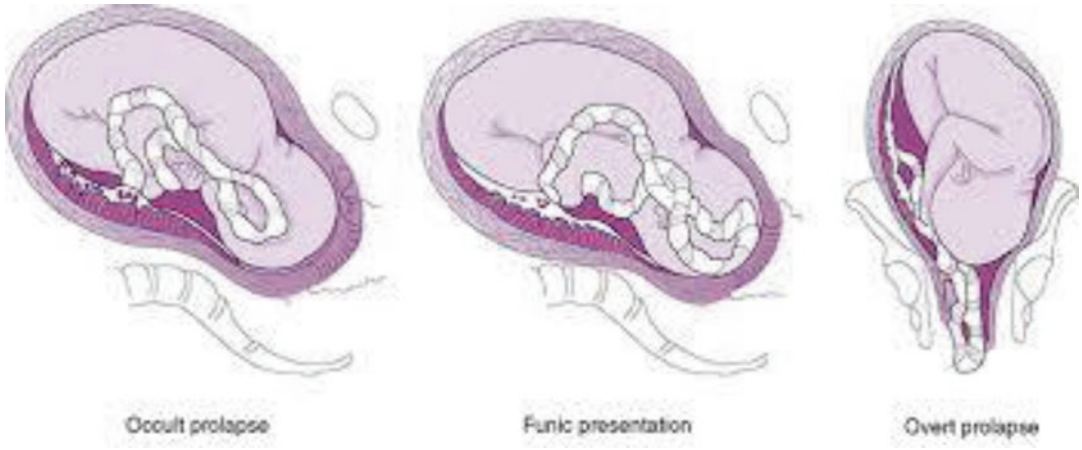


Fig. 33.1 Types of cord prolapse

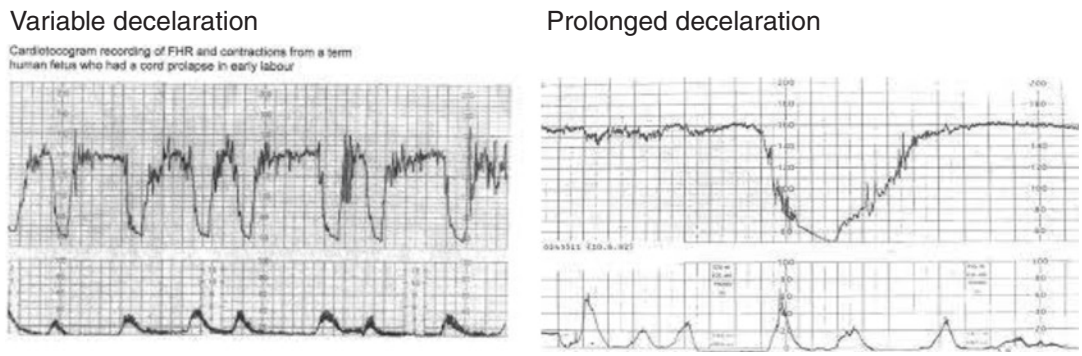


Fig. 33.2 CTG in cord prolapse

pulsatile structure through the membranes or with membranes ruptured. The absence of pulsations and meconium passage are ominous signs.

A speculum examination should never be omitted in PROM and PPROM. The only hint usually is variable deceleration or prolonged bradycardia following rupture of membranes (Fig. 33.2).

33.1.6 Prevention

1. ARM should be avoided when the presenting part is high or mobile. A stabilizing induction should be done in polyhydramnios with high head. A controlled ARM with a spinal needle can be done in cases of high head where contractions have been established and fixity of the head is yet to occur. Facilities for a crash CS should be available in every labor room.
2. Transverse, oblique, or unstable lie patients should be offered ECV after 34 weeks after informed consent. Oblique or transverse lie patients persisting after 37 weeks can be offered repeat ECV if no contradiction for the same.
3. PROM/PPROM patients with mobile head should be restricted till the head is fixed.
4. Upward pressure on the presenting part should be kept to minimum during per vaginum and ARM.
5. If the cord presentation persists after the established labor, CS is indicated.
6. Beware of cord prolapse, during amnioinfusion.

33.1.7 Management

Retrofilling the bladder (Fig. 33.3a, b).

33.1.8 Fetal Complications

Among fetal complications prematurity and congenital malformations are the major adverse outcomes associated with cord prolapse in our hospital settings, but one cannot forget birth asphyxia which is also associated with cord prolapse. Asphyxia further results in hypoxic–ischemic encephalopathy and cerebral palsy. The cause of asphyxia is cord compression, and umbilical arterial vasospasm prevents venous and arterial blood flow to and from the fetus.

33.1.9 Summary and Recommendations

- The first sign of cord prolapse is usually severe, prolonged fetal bradycardia or moderate to severe variable decelerations after a previously normal tracing. The prolapse may be overt or occult.
- Standard obstetrical management of cord prolapse is prompt cesarean delivery to avoid fetal compromise or death from compression of the cord. However, vaginal delivery may be a reasonable option in select cases when delivery is imminent and can be safely assisted.
- Intrauterine resuscitation using maneuvers such as elevation of the presenting part manually or by retrofilling the bladder, placing the patient in a Trendelenburg or knee-chest position, and administering a tocolytic may reduce pressure on the cord while preparations are being made for delivery.
- Reported perinatal mortality related to cord prolapse varies widely, from 0% to 3% for events occurring among patients monitored on a labor and delivery unit. Asphyxia and complications related to prematurity and congenital anomalies are the major causes of poor outcome. The degree of cord compression, the interval between cord prolapse and delivery, and the successful use of intrauterine resuscitation maneuvers all impact the risk of asphyxia.
- Awareness of patients at high risk of prolapse may help facilitate prompt diagnosis and delivery when prolapse occurs.
- Transverse, oblique, or unstable lie patient should present to the hospital urgently when they begin labor or rupture membranes.
- In high-risk patients, the risk of cord prolapse may be reduced by minimizing the use of obstetric interventions that may disengage the presenting part and by performing controlled amniotomy.
- We do not routinely perform ultrasound examinations to assess umbilical cord position near term, but would order an ultrasound examination to confirm cord position when there is a clinical suspicion of funic presentation.
- For patients with a funic presentation that has not resolved before labor at term, management depends on the risk of cord prolapse.
- Patients with a floating vertex, cervical dilation >2 cm, and persistent funic presentation at ≥39 weeks are at very high risk of cord prolapse with rupture of membranes and unlikely to benefit from expectant management, especially if polyhydramnios is present. The authors offer these women induction of labor by “needling” the membranes in a controlled environment, with the anesthesia team ready for an emergency cesarean delivery in the event of cord prolapse.
- Patients in whom the vertex has descended, the cervix is not significantly dilated, and amniotic fluid volume is normal are at an increased risk of cord prolapse, but not the highest risk group. Advise these women to come to labor and delivery as soon as labor begins so that the cord position can be evaluated. The patient can continue to labor with a goal of vaginal delivery if the funic presentation has resolved. If the funic presentation is confirmed, we monitor the patient/fetal heart rate continuously and perform a controlled amniotomy, with the anesthesia team ready for an emergency cesarean delivery in the

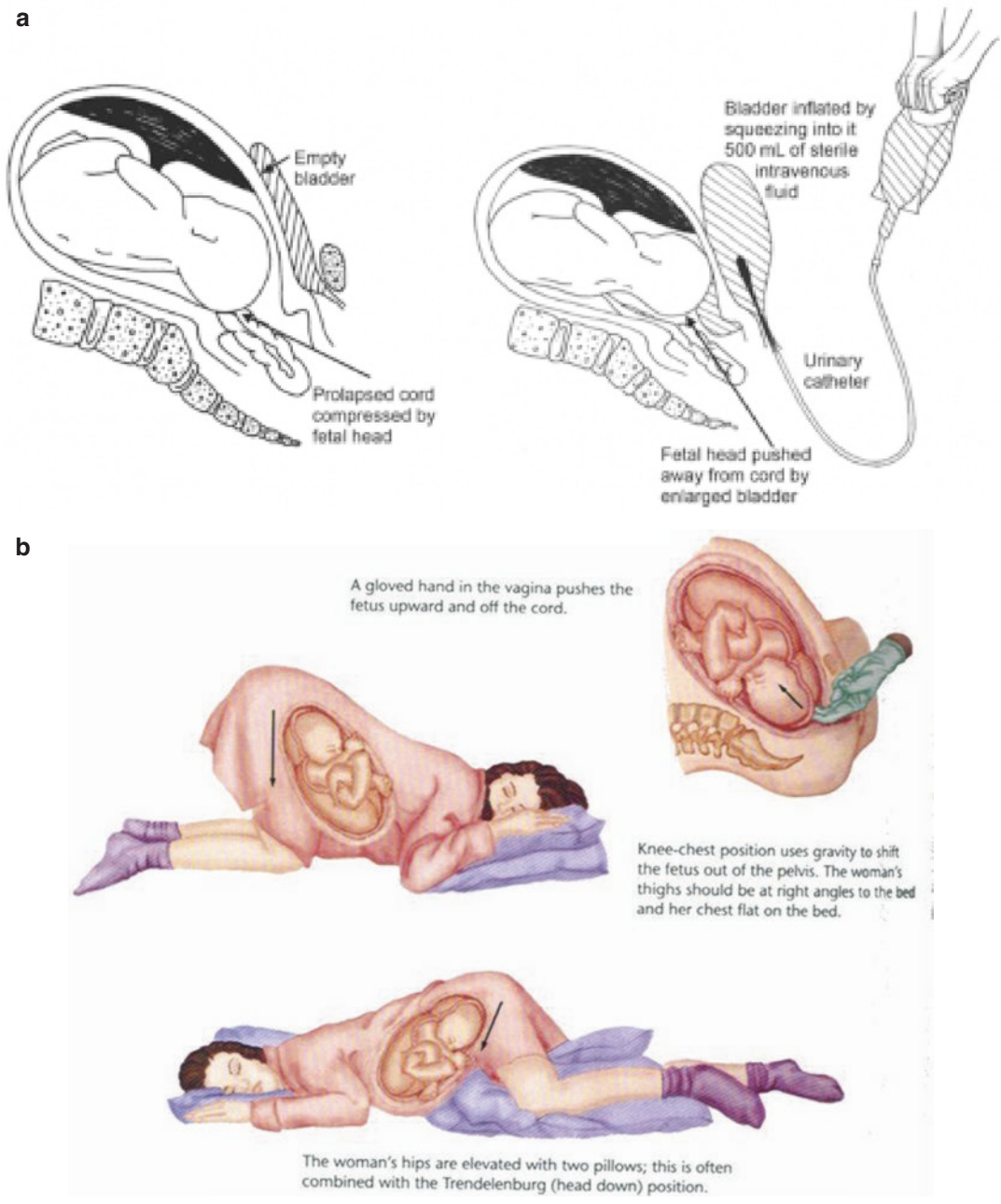


Fig. 33.3 (**a**) Retrofilling the bladder (**b**) Position for prolapse cord

event of cord prolapse. Cesarean delivery without a trial of labor is also reasonable.

33.2 Transverse Lie

33.2.1 Definition

When the long axes of mother and fetus are at right angles to one another, a transverse lie is present. Because the shoulder is placed so frequently in the brim of the inlet, this malposition is often referred to as the shoulder presentation. The denominator is the scapula (Sc); the situation of the head determines whether the position is left or right, and that of the back indicates whether it is anterior or posterior. Thus, LScP means that the lie is transverse, the head is on the mother's left side, and the baby's back is posterior. The part that actually lies over the pelvic brim may be the shoulder, back, abdomen, ribs, or flank. This is a serious malposition whose management should not be left to nature.

33.2.2 Incidence

The incidence of transverse lie is around 1:500. The incidence is higher before term (as high as 1 in 50 at 32 weeks' gestation).

33.2.3 Etiology

This abnormality is more common in multiparas than primigravidas because of the laxness of the uterine and abdominal muscles. Similar conditions in which there is relatively excess space for the fetus are polyhydramnios and prematurity. Other causes include anything that prevents engagement of the head or the breech, such as placenta previa; an obstructing neoplasm; multiple pregnancies; fetal anomalies; fetopelvic disproportion; contracted pelvis; and uterine abnormalities such as uterus subseptus, uterus arcuatus, and uterus bicornis. In many instances, no etiologic factor can be determined, and we assume that the malposition is accidental. The head happens to be out of the lower uterine segment when labor starts, and the shoulder is pushed into the pelvic brim.

33.2.4 Diagnosis of Position: Transverse Lie

33.2.4.1 Abdominal Examination

The appearance of the abdomen is asymmetrical.

The long axis of the fetus is across the mother's abdomen.

The uterine fundus is lower than expected for the period of gestation. It has been described as a squat uterus. Its upper limit is near the umbilicus, and it is wider than usual.

Palpation of the upper and lower poles of the uterus reveals neither the head nor the breech. The head can be felt in one maternal flank. The buttocks are on the other side.

The abdomen is wide, whereas the uterine fundus extends to only slightly above the umbilicus. When the back is anterior, a hard resistance plane extends across the front of the abdomen. When it is posterior, irregular small parts are felt through the abdominal wall.

33.2.4.2 Vaginal Examination

The "gridiron" feel of the ribs and the palpation of the scapula and the clavicle on opposite sides of the thorax are distinguishing features. The position of the axilla indicates the side of the mother toward which the shoulder is directed.

33.2.5 Fetal Heart

Fetal heart is heard best at or just below the umbilicus.

33.2.6 Active Management

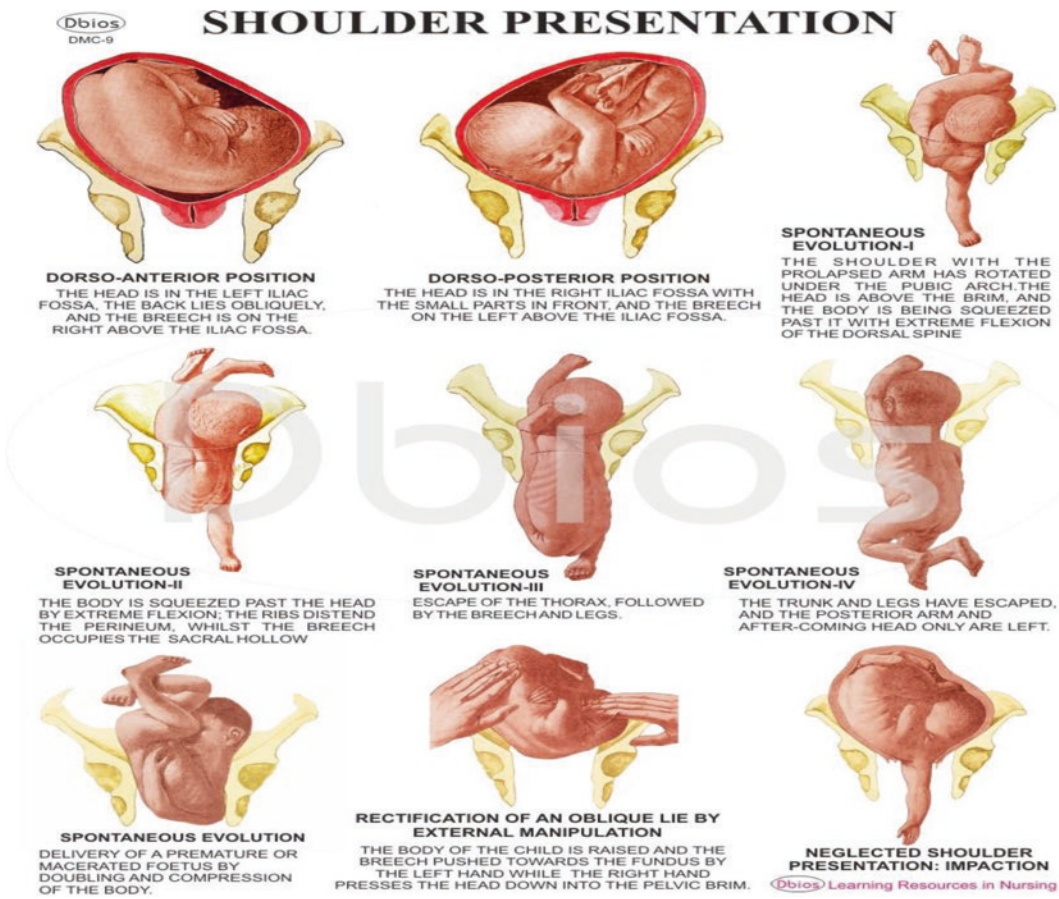
Admission is often advised from 37 to 39 weeks' gestation, which provides the opportunity for (1)

daily observations of fetal lie and presentation to be made, (2) active treatment to correct the lie if necessary, (3) calling for immediate assistance upon membrane rupture or the onset of labor, and (4) urgent delivery if the lie is not longitudinal, fetal distress occurs, or the cord is presenting or has prolapsed. Evidence to support this approach is provided by one small study of expectant management for unstable lie after 37 weeks' gestation that reported that 17% presented in labor with a transverse lie and 6% had a prolapsed cord resulting in neonatal death in 1%. 44 If spontaneous resolution to a longitudinal lie occurs and a cephalic or breech presentation is maintained for 48 h, women may be discharged home to await labor. Some currently discourage labor with a breech presentation. If spontaneous resolution of an abnormal lie does not occur, an active approach to management may be adopted. External cephalic version can be attempted if facilities permit. Immediate delivery is mandatory in the event of placental abruption, membrane rupture, cord prolapse, or acute fetal distress for any reason. Rhesus immunoprophylaxis should be given to at-risk women neither before nor soon after the version attempt and an estimate of the volume of any fetomaternal hemorrhage made about 20 min after the attempt at version, using the Kleihauer-Betke method or flow cytometry to determine whether additional prophylaxis is necessary. If a longitudinal lie is not maintained, the version can be repeated as often as necessary, and if unsuccessful, women should be kept in the hospital. The success of version for unstable lie is unclear, but it is probably greater than for breech presentation, which is usually quoted around 40–65%.

Tocolysis can be used, including infusions of ritodrine 50 μg/min for 15 min or terbutaline sulfate 250 μg intravenously over 1–2 min or GTN spray, but is often unnecessary with a transverse or oblique lie. In the event the lie remains unstable, a stabilizing induction may be performed usually at 38–39 weeks' gestation. Following transfer to the labor suite, an external cephalic version is performed if necessary to convert the fetal lie to longitudinal. Once the fetus is in position, regular abdominal palpations are performed to confirm the lie is maintained and a titrated intravenous infusion of oxytocin commenced to stimulate uterine contractility. Although contractions can also be stimulated with local (vaginal) or oral prostaglandins, this is probably less advisable because the response to prostaglandins can be unpredictable and occasionally hyperstimulation occurs, which would be especially concerning if the lie reverts to oblique or transverse, when tocolysis and/or emergency cesarean section is required. As soon as contractions are occurring at 10-min intervals or more frequently, a low amniotomy ideally needling is performed, having ensured at vaginal examination that the lie is still longitudinal, the presentation is not compound, and in particular, the cord is not presenting. If the cord presents, an emergency cesarean section is necessary. Once low amniotomy is performed, a reasonable volume of amniotic fluid should be released, followed by confirmation that the cord has not prolapsed and the presenting part is fixed in the pelvic brim. Thereafter, once labor is established, management continues as for uncomplicated cases, being ever mindful that cord prolapse might still occur. Finally, a decision to deliver by elective antepartum cesarean section at 38–40 weeks' gestation is a legitimate management option. Ideally, an attempt should be made to convert the lie to longitudinal immediately before the laparotomy or after opening the peritoneum but before incising the uterus. If successful, a lower segment incision in the uterus can be used for the delivery.

Planned cesarean section is particularly appropriate if antenatal external cephalic version is contraindicated, previous attempts at version fail, or there is a mechanical obstruction to vaginal delivery.

ECV can be tried at any time before labor with intact membranes after 32 weeks and ideally after 34 weeks. In difficult fetal extraction especially with no liquor with established labor, a low vertical incision of the lower segment may sometimes be required. If labor continues with transverse lie, the shoulder gets impacted in the upper part of the pelvis, and a retraction ring forms. With time, the retraction ring rises high, and the uterus will rupture especially in a multi with neglected shoulder



presentation. In primi, obstructed labor and Bandl's ring are formed before rupture. Only if the fetus is small especially less than 800 g, spontaneous delivery as a corpore conduplicate is possible.

33.3 Intrapartum

When the fetal lie is transverse or oblique and the membranes rupture or labor starts, the options for management depend on (1) whether there is a cord presentation or prolapse, (2) the stage of labor whether before full cervical dilation or during the second stage, and (3) whether the membranes are intact or ruptured.

33.3.1 First Stage of Labor

Management depends on whether the fetal membranes are intact or ruptured. With intact membranes, an external version can be attempted. This is performed between contractions with or without tocolysis to relax the uterus before the attempt. Tocolytic agents that can be used in this situation include β-agonists such as terbutaline infused at 5–20 μg/min, salbutamol (albuterol) 2.5–4.5 μg/min, ritodrine 100–350 μg/min, 60 or the oxytocin antagonist atosiban, given a single intravenous bolus dose of 6.75 mg.

If the version is successful, a repeat vaginal examination should be performed to exclude cord presentation or prolapse.

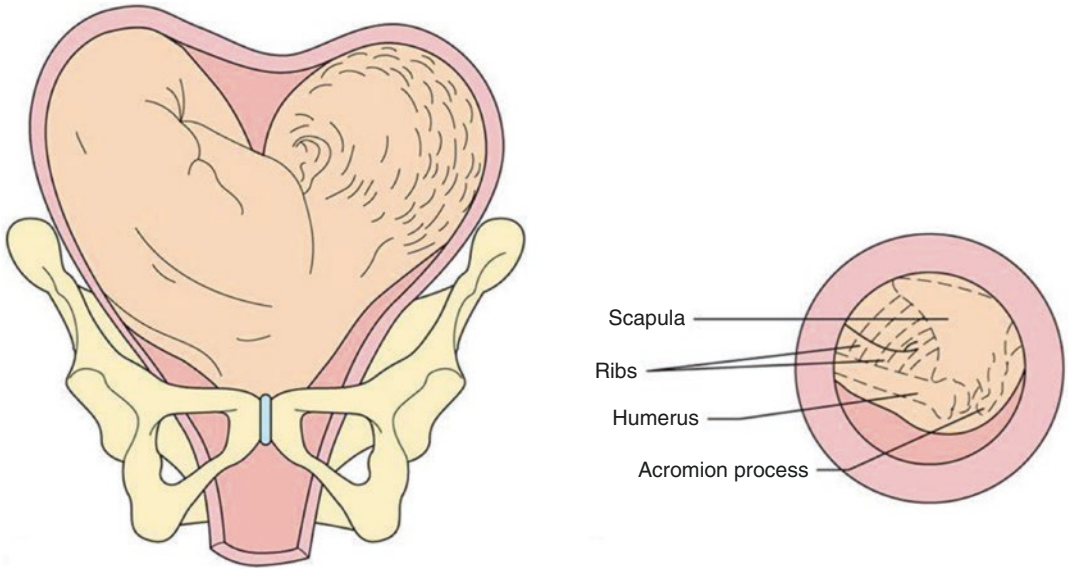


Fig. 33.4 Correlating abdominal and vaginal examination findings

Once this has been confirmed, labor can be allowed to establish, ensuring the lie remains longitudinal until the presenting part engages in the pelvis, with progress augmented with oxytocin if necessary. If the version has been unsuccessful, delivery by cesarean section should be performed. With ruptured membranes, delivery by cesarean section is generally the only option unless the woman is in very early labor and an attempt at external cephalic version is successful. A shoulder presentation, which may include an arm prolapsed into the vagina, is a serious situation. Clamping of the uterine wall around the fetus by a retraction or Bandl's ring may have contributed. Delivery must be by cesarean section. Administration of a uterine relaxant such as halothane administered by the anesthetist can facilitate the delivery, which is most safely achieved through a J-shaped, inverted T, or classic uterine incision. This incision allows the fetus to be withdrawn through the fundus of the uterus, whereas a Pfannenstiel incision makes it nearly impossible to manipulate the fetus into a position that will allow delivery without damage to the fetus and the uterus. This approach to delivery is still indicated even if the fetus has already died, to avoid uterine rupture and other serious uterine trauma at the time of the delivery (Fig. 33.4).

33.3.2 Second Stage of Labor with Intact Membranes

Only for the second baby of the twins.

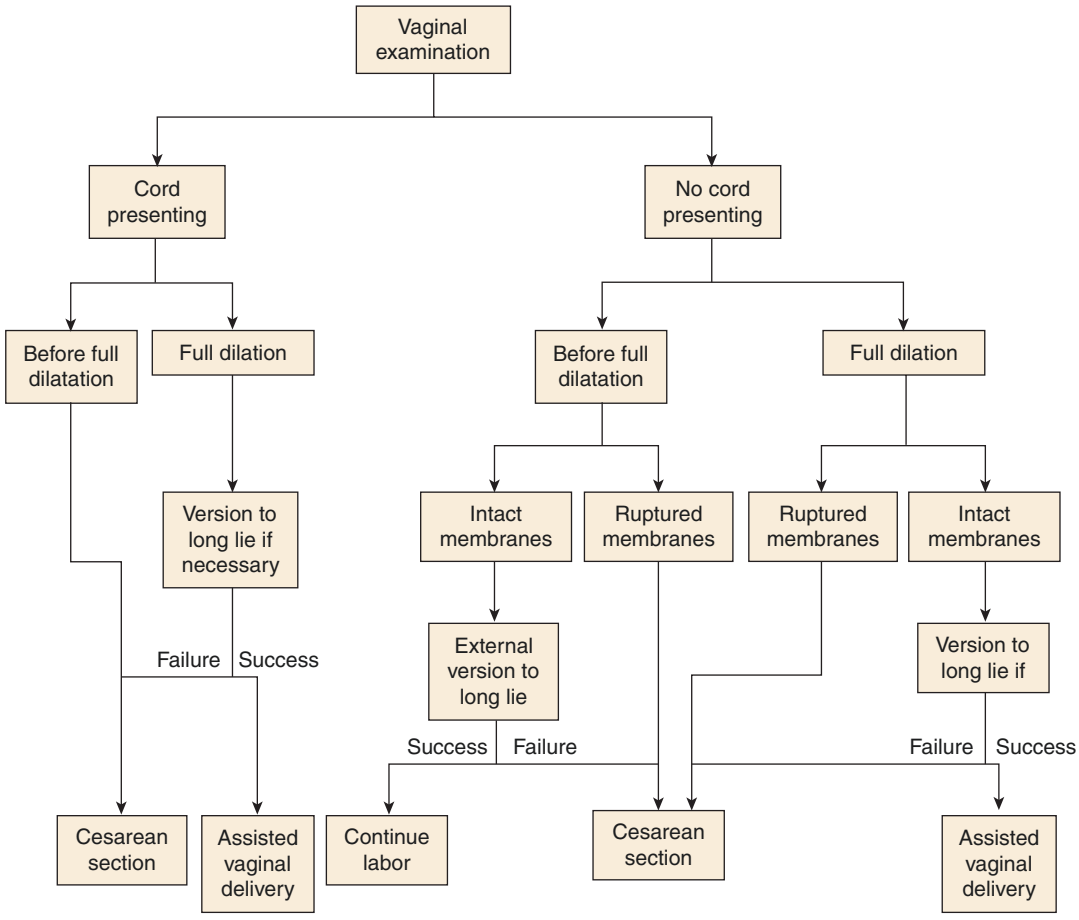
If fetopelvic disproportion has been excluded, correction to a longitudinal lie by external cephalic or internal podalic version followed by an assisted vaginal cephalic or breech delivery should be anticipated. If internal podalic version is attempted, this requires adequate anesthesia (either general or fully effective regional block). Using appropriate aseptic techniques, the obstetrician introduces a hand into the uterus, confirms the foot of the anteriorly positioned leg by identifying the heel, and applies traction on the foot, withdrawing the foot and leg and subsequently the breech through the vagina. It is ideal if both feet can be grasped and pulled down, as this avoids the "splits," with one leg down and one leg up, which can splint the baby and lead to a difficult delivery. The management is then as described for breech extraction. Great care must be taken to avoid both fetal bony injury and lac-

eration of the uterus. Tocolysis may be helpful for this procedure. If the version is not successful, delivery should be achieved by cesarean section; attempts at version should be performed only when immediate resort to section is available. The obstetrician should be aware of the possibility of atonic primary postpartum hemorrhage when uterine relaxants have been administered to assist the delivery.

33.3.3 Second Stage of Labor with Ruptured Membranes

If there is a compound presentation involving a hand and the fetal head is engaged in the pelvis, it may be possible with vaginal manipulation to encourage the hand to withdraw from the pelvis so that an assisted vaginal delivery can continue. This should be done only in the second of twins. In all other cases, cesarean section is required. If the fetus is in a transverse or oblique lie or there is a significant compound presentation, delivery should be performed as urgently as possible using a U-shaped, J-shaped, inverted T, or classic cesarean section incision unless an attempt at external version can be successfully made on opening the abdominal wall immediately before the uterus is incised. Struggling to deliver the fetus through a lower segment incision can cause serious trauma to the fetus, uterus, or both. Incision extensions may result in compromised healing and a vulnerable area of scar integrity, which may predispose to uterine rupture in future pregnancies or labors (Fig. 33.5).

33.3.4 Management of Unstable Lie



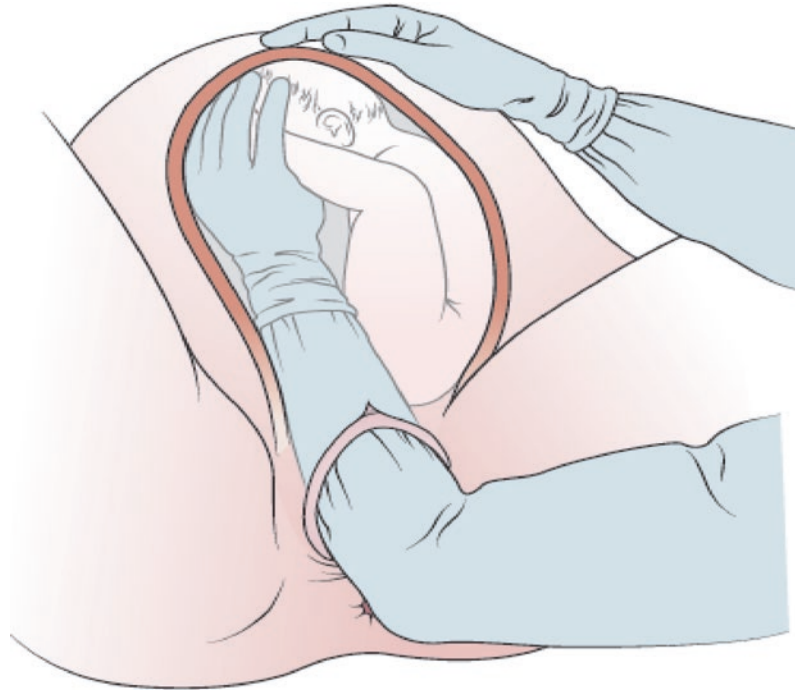


Fig. 33.5 Internal podalic version

Overdistended Uterus

34

Saswati Sanyal Choudhury

34.1 Introduction

Overdistended uterus is said to be present when the height of the fundus is greater than the gestational age. First consideration of this condition is wrong dates of last menstrual period. Other possible conditions are multiple pregnancy, polyhydramnios, foetal macrosomia, foetal hydrops and presence of big uterine myomas. All these obstetric complications have increased risk to the mother and foetus leading to increased maternal and perinatal mortality and morbidity. Each obstetric condition needs to be diagnosed for management. Emergency ultrasound in cases where the condition is not diagnosed before plays a vital role for proper diagnosis.

34.2 Diagnosis of Overdistended Uterus

34.2.1 Clinical Management

Symphyseal-fundal height is a screening tool for abnormal growth of foetus and uterine distension. Foetomaternal factors that may increase SFH are amount of amniotic fluid, amount of abdominal fat, presence of large fibroids and multifoetal pregnancy. Thorough clinical history of the patients to find out the duration of symptoms, confirmation of gestational weeks and whether she was already diagnosed to have one of these conditions are important.

Acute onset of sudden enlargement of the uterus with pain can be a presenting symptom of acute polyhydramnios.

If patient presents in shock and severe pallor, possibility of concealed accidental haemorrhage has also to be kept in mind.

Family history of diabetes mellitus or patient already diagnosed with DM usually means foetal macrosomia.

Patients with Rh-negative blood group have a possibility of foetal hydrops. Careful clinical examination can diagnose the causes of overdistension.

Associated firm localised swelling on uterine surface confirms uterine leiomyoma. She may also give history that she is a diagnosed case of fibromyoma.

Confirmation of accuracy of gestational age is very important to rule out wrong dates.

If only one foetus is felt on abdominal examination, then consider wrong dates, macrosomia or polyhydramnios.

If multiple foetal poles and multiple parts are felt, then multiple pregnancy is confirmed by ultrasound.

S. S. Choudhury (✉)
Fakhruddin Ali Ahmed Medical College,
Barpeta, Assam, India

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34.2.2 General Management

Patient needs rest and propped-up position in severe cases especially with acute polyhydramnios and multiple pregnancy. Preterm labour is a known complication of overdistension for any causes. If there is shortening of the cervix on examination or there is uterine contraction, steroid for foetal lung maturity must be given in preterm multiple pregnancy and hydramnios.

During labour close monitoring is done and delivery should occur in tertiary centres. Wide bore intravenous cannula should be kept in place in advance as postpartum haemorrhage is common in overdistension. Active management of the third stage is routinely done although it does not prevent PPH in all cases. Prostaglandin injections for controlling haemorrhage should be readily available. Blood should be kept crossmatched.

34.3 Management for Specific Conditions

34.3.1 Polyhydramnios

Clinically polyhydramnios is said to be present when AFI is more than 95th centile for gestational age or maximum vertical pool length of more than or equal to 8 cm and AFI more than or equal to 25 cm. Clinically, detectable polyhydramnios occurs in 0.5–1% of pregnancies [1–7].

Patients usually present with respiratory embarrassment. Abdominal pain, preterm labour and PPROM are common presentations. There may be palpitation, leg oedema, vulval oedema and abdominal oedema. Patient may also have complains of aggravated piles and varicosities in lower extremities. Maternal mirror syndrome in which maternal condition mimics the foetus with signs of preeclampsia may be the presenting feature in foetal hydrops. Abdominal skin is stretched, shiny, oedematous and tense. Fluid thrill is found along with difficulty in palpation of foetal parts with presence of external ballottement and difficulty in locating foetal heart sound. Malpresentation is often associated.

34.3.2 Causes of Polyhydramnios

1. Idiopathic
2. Maternal causes:
   (a) Maternal diabetes
   (b) Maternal alloimmunisation
   (c) Cardiac or renal disease due to excessive transudation
   (d) Maternal substance abuse
3. Foetal causes:
   (a) Parvovirus infection
   (b) Congenital syphilis
   (c) Foetal anomalies:
   - CNS anomaly: anencephaly, spina bifida, meningocele
   - Oesophageal atresia, trachea-oesophageal fistula
   - High gut obstruction: duodenal atresia
   - Thoracic tumours
   - Facial dysmorphism, congenital goitre, cystic hygroma.
   - Congenital diaphragmatic hernia
   - Foetal Bartter syndrome
   - Myotonic dystrophy
   - Foetal sacrococcygeal teratoma
   - Foetal vein of Galen syndrome
   - TTTS in multiple pregnancy
4. Placental causes: placental tumours

All these conditions need to be diagnosed as each of them has specific management. Detailed history of drug exposure, maternal diabetes, prior prenatal screening for aneuploidy, family history of myotonic dystrophy or past history of skeletal dysplasia or arthrogryposis and red cell alloimmunisation are very important.

34.3.3 Investigations for Polyhydramnios

34.3.3.1 Maternal

Glucose tolerance test, blood grouping and antibody titre if not done already.

34.3.3.2 USG

1. Degree of polyhydramnios.
2. Presence of foetal anomalies especially those which impede foetal swallowing should be

looked for. Possible USG findings are markers of chromosomal defects, absence of stomach bubble or double bubble sign, diaphragmatic hernia, abnormal posture or absent movement which may suggest neuromuscular disorder and abnormal long bones which suggest skeletal dysplasia and are associated with polyhydramnios.
3. Placental localisation, evidence of abruption and looking for any placental mass.

34.3.3.3 Doppler

Middle cerebral Doppler study for evidence of foetal anaemia, foetal cardiac scan and presence of tachyarrhythmia and evidence of TTTS are also essential for aetiological diagnosis.

34.3.3.4 Karyotyping

For anomalous foetus karyotyping also may be necessary.

34.3.3.5 Evidence of Infection

Serology for parvovirus and maternal screening for toxoplasmosis and cytomegalovirus can be linked up with foetal hydrops.

34.3.3.6 Maternal Complications of Polyhydramnios

There is increased risk of preterm delivery and perinatal mortality. Abnormal presentation leading to increased incidence of operative delivery is another maternal risk associated with polyhydramnios. Placental abruption due to rapid decompression is a grave complication. Other risks are dependent on aetiology. Cord prolapse, dysfunctional labour, retained placenta and postpartum haemorrhage are also common complications of gross polyhydramnios. Subinvolution of the uterus and puerperal sepsis are possible postpartum maternal risks.

34.3.3.7 Foetal Complications

There is increased perinatal mortality.

34.3.3.8 Treatment Options for Polyhydramnios

Counselling forms an important part of management. Even an USG is not sufficient to diagnose oesophageal atresia and tracheobronchial fistula, and these conditions are confirmed only after the baby is born so a guarded reassurance is necessary with apparently idiopathic cases of polyhydramnios. For mild cases expectant management is done. Indomethacin in a dose of 25 mg every 6 h till 32–34 weeks is also used by some authors, but it has a variety of foetal side effects including closure of ductus arteriosus, oligohydramnios and foetal renal damage although not all reports support these associations. Indomethacin reduces foetal urine production through direct renal effect.

Amnioreduction is done in severe cases if patient is symptomatic and prolongation of pregnancy is indicated. It is done with an 18–20 g needle under USG guidance to avoid the placenta and with a large syringe or with vacuum bottles till AFI is 10–20 cm. The uterus should be monitored for any contraction for 12–24 h, and follow-up at 1–3 weeks is indicated depending on severity. The procedure can be repeated if condition recurs [8].

Delivery is to be done in tertiary care, and involvement of neonatologist is necessary to manage newborns with prematurity and other high-risk neonates associated with polyhydramnios. AMTSL is routinely done for all cases, and close observation for PPH for 2 h following delivery may save maternal life.

34.3.4 Multiple Pregnancy

Use of assisted reproductive techniques has greatly increased the incidence of multiple pregnancy. Chorionicity of twins should be determined, and it is possible as early as first trimester. Routine first trimester USG is to be done in all patients with ART, family history of multiple pregnancy and elderly mothers as well as mothers having previous multiple pregnancy.

34.3.4.1 Maternofoetal Outcome in Multiple Pregnancy

Most common complications of multiple pregnancy are preterm labour and PPROM. Mean age of gestation in twins is 37 weeks and 31 weeks for triplets. Twins are usually smaller at any given gestational age than singletons. Twin-to-twin transfusion syndrome occurs in monozygotic

twins due to vascular sharing of the placenta which leads to growth retardation of one foetus and cardiac failure of the other. Anaemia, pre-eclampsia and placenta praevia are common complications of multiple pregnancy and add to the mortality and morbidity of both mother and foetus. Higher-order multiple pregnancy has much worse outcome than twin.

34.3.4.2 Management of Multiple Pregnancy in Labour Room

Ultrasonography is used to ascertain the chorionicity, foetal growth and signs of TTTS. FGR is managed with regular monitoring and steroids for lung maturity. For severe TTTS, foetoscopic laser surgery can be done if available to divide vascular anastomoses. The presence of short cervix of less than 20 mm is a significantly higher risk of preterm labour and can be treated with steroid and progesterone. Tocolytics for short period can be used so that steroid works for lung maturity.

34.3.4.3 Delivery

If the patient is already in labour, IV cannula is fixed. Blood should be sent for crossmatching. If presentation of the first twin is cephalic, then vaginal delivery is attempted. For all other cases of non-vertex first twins, caesarean is a safer procedure.

The main risk of twin delivery is hypoxia for the second twin during protracted second stage and premature placental separation. Immediately after delivery of the first twin, continuous foetal monitoring of the second twin is to be done. After ascertaining the longitudinal lie, ARM is done and intravenous oxytocin is started. If the second twin is transverse, external cephalic version is done followed by ARM. If membrane is ruptured in transverse lie, internal podalic version in an expert hand can save the baby especially with cord prolapsed. In case of any signs of foetal distress with vertex presentation, vacuum extraction or breech extraction can be performed. Ideally, the second twin should be delivered within 30 min. The third stage of labour should be actively managed [9]. In case of higher-order multiple gestation, selective foetal reduction with injection of potassium chloride in such cases as early in pregnancy as possible can reduce its complication rates.

34.3.5 Foetal Macrosomia

Assessment of foetal weight by ultrasound is very important to know the possibility of vaginal delivery. Associated diabetes mellitus must be kept in mind, and adequate control during labour is necessary. Anticipation of prolonged labour and obstruction should be there, and partographic management is vital. The most dangerous complication is shoulder dystocia. By definition it is a delivery that requires additional obstetric manoeuvres to release the shoulders after gentle downward traction has failed [10]. It is usually unpredictable. Identification of risk factors and antenatal care plan that aims to modify them may reduce its incidence. RCOG guideline recommends elective CS in macrosomia (foetal weight >4.5 kg) with diabetes [11]. Previous history of shoulder dystocia also needs elective caesarean section. Management aims prevention of birth asphyxia while avoiding injury to foetus and mother.

First-line manoeuvres are McRoberts manoeuvre where woman is kept in supine position with hips acutely flexed and the knees closed to the chest which straightens lumbosacral angle allowing descent of the posterior shoulder. It has a success rate of 90%.

Directed suprapubic pressure continuously on the posterior aspect of anterior shoulder may facilitate its rotation to an oblique position, adduction of shoulders and reduction in bisacromial diameter. Second-line manoeuvres are delivery of posterior arm, Rubin's manoeuvre, Wood's screw and reverse Wood's screw where the aim is to rotate the shoulders to 180° and enable delivery by bringing down anterior shoulder posteriorly.

Third-line manoeuvres are Zavanelli manoeuvre and symphysiotomy.

34.3.6 Fibroid in Pregnancy

Uterine leiomyomas are found in 4% of all pregnancies. Out of these 10–30% of women develop complications during pregnancy [12]. A detailed history should be taken to find out whether she is a known case of fibroid. In addition to large for date uterus, palpable lumps on the uterine sur-

face may be felt which can be multiple. Spontaneous abortion rate i higher in pregnant women with fibroids compared with control subjects without fibroids (14% vs. 7.6%, respectively) [13]. If the placenta is situated near fibroid, chances of early pregnancy haemorrhage are also high [14]. Chances of having placenta praevia are also twofold higher in some studies [15, 16]. Large fibroids causing distortion of uterine cavities can cause foetal compression deformities like torticollis and dolichocephaly [17, 18]. Foetal malpresentation may also occur with large fibroids, multiple fibroids and fibroids in lower segment [16, 19] like breech, transverse or oblique lies. Incidence is reported to be 13.4% vs. 4.5% in controls [15, 20]. Tenderness over fibroids can be elicited in fibroid with so-called red degeneration which can lead to preterm labour or abortion.

USG is the mainstay of diagnosis, and this is also important for its location like subserous, interstitial or submucous or corporeal or cervical fibroid.

34.3.6.1 Management of Complications of Fibroid in Pregnancy

Management is basically conservative with reassurance in antenatal period. Special management is necessary if it is painful, previous myomectomy and abnormal presentation. In case of pain, symptomatic treatment is required with paracetamol in moderate pain, and opioids may be necessary in severe cases. Majority of pain is self-limiting and pregnancy is unaffected.

Vaginal delivery even in the presence of large uterine fibroids (>5 cm) should not be regarded as a contraindication, and a trial of labour should be allowed [16, 20].

Incidence of postpartum haemorrhage in fibroid is conflicting. Pooled data suggests a slight rise of its incidence (2.5% vs. 1.4%) [15]. Distortion of cavity and effect on myometrial contraction by fibroids are thought to be the causes of postpartum haemorrhage and may land up in peripartum hysterectomy. Active management of the third stage should be offered to all women with fibroid as a preventive measure. Incidence of retained placenta is also little higher (1.4% vs. 0.6%) [15].

In a systematic review, women with fibroids were at a 3.7-fold increased risk of caesarean delivery (48.8% vs. 13.3%, respectively) [15]. Elective CS is done in abnormal presentation with fibroid. Transabdominal USG and TVS is done to know the precise relationship of fibroids and presenting part and exclude other cases of malpresentation like placenta praevia. An experienced surgeon should do it in a planned way. Transverse incision is generally preferred, but midline may be required if fibroids are large and multiple. If the lower segment is occupied by a big fibroid, then options are myomectomy first and foetal extraction or an upper segment CS depending on the experience of the surgeon. The general rule is avoidance of fibroids wherever possible, and incision should be away from fibroid as it can warrant bleeding requiring blood transfusion, uterine artery ligation and/or puerperal hysterectomy [21, 22]. Myomectomy at the time of caesarean delivery should only be performed if there is difficulty in extraction of the baby and closure of the wound. Alternative ways of delivery of foetus should be kept in mind, like use of vacuum extraction of vertex or delivery as breech. Increased blood loss is anticipated and crossmatched blood should be ready. Risk of hysterectomy is very low but should be counselled beforehand.

Previous myomectomy may pose difficulty due to adhesion and intraoperative haemorrhage.

34.3.6.2 Myomectomy in Pregnancy

Some literatures have reported that antepartum myomectomy can be safely performed in the first and second trimester of pregnancy [23, 24]. Indications are severe pain from a degenerating subserosal or pedunculated fibroid, a large or rapidly growing fibroid or any large fibroid (>5 cm) located in the lower uterine segment.

Uterine artery ligation or uterine artery embolisation (UAE) may be done in selected cases of fibroids immediately after caesarean delivery which may reduce incidence of blood loss and chance of hysterectomy [25, 26].

Postpartum care includes monitoring of them as some intramural fibroid may become submucous polyp and may cause bleeding in late postpartum period and will need hysteroscopic removal in the near future.

34.4 Conclusion

Overdistended uterus poses problems to both mother and child. Management depends on aetiological factor. Anticipation of complications well in advance and preparedness to combat the complications by experienced obstetricians will save invaluable lives of mothers.

References

1. Ioannou C, Papageorghiou A. Polyhydramnios. In: Arulkumaran S, Regan L, Papageorghiou AT, Monga A, Farquaharson DIM, editors. Oxford desk reference: obstetrics & gynaecology. Oxford: Oxford University Press; 2011. p. 164–5.
2. Beall MH, Belooseky R, Ross MG. Abnormalities of amniotic fluid volume. In: James D, Steer PJ, Weiner CP, Gonik B, Crowther C, Robson S, editors. High risk pregnancy: management options. 4th ed. St. Louis, MO: Saunders/Elsevier; 2011. p. 203–5.
3. Hill LM, Breckle R, Thomas ML, Fries JK. Polyhydramnios: ultrasonically detected prevalence and neonatal outcome. Obstet Gynecol. 1987;69:21.
4. Dashe JS, McIntire DD, Ramus RM, et al. Hydramnios: anomaly prevalence and sonographic detection. Obstet Gynecol. 2002;100:134.
5. Thompson O, Brown R, Gunnarson G, Harrington K. Prevalence of polyhydramnios in the third trimester in a population screened by first and second trimester ultrasonography. J Perinat Med. 1998;26:371.
6. Biggio JR Jr, Wenstrom KD, Dubard MB, Cliver SP. Hydramnios prediction of adverse perinatal outcome. Obstet Gynecol. 1999;94:773.
7. Pri-Paz S, Khalek N, Fuchs KM, Simpson LL. Maximal amniotic fluid index as a prognostic factor in pregnancies complicated by polyhydramnios. Ultrasound Obstet Gynecol. 2012;39:648.
8. Piantelli G, Bedochi L, Cavicchioni O, et al. Amnioreduction for treatment of severe polyhydramnios. Acta Biomed. 2004;75(Suppl 1):56–8.
9. Thilaganathan B. Multiple pregnancy. In: Arulkumaran S, Papageorghiou AT, Monga A, Farquharson DIM, editors. Oxford desk reference: obstetrics & gynaecology. Oxford: Oxford University Press; 2011. p. 158–9.
10. American College of Obstetrician and Gynaecologists. Shoulder dystocia. ACOG practice bulletin clinical management guidelines for obstetrician-gynacologists. No. 40. Obstet Gynaecol. 2002;100:1045–50.
11. Royal College of Obstetrician & Gynaecologists (RCOG). Shoulder dystocia guideline no. 42. Green top guidelines. London: RCOG; 2005.
12. Katz VL, Dotters DJ, Droegemueller W. Complications of uterine leiomyomas in pregnancy. Obstet Gynecol. 1989;73:593–6.
13. Benson CB, Chow JS, Chang-Lee W, et al. Outcome of pregnancies in women with uterine leiomyomas identified by sonography in the first trimester. J Clin Ultrasound. 2001;29:261–4.
14. Winer-Muram HT, Muram D, Gillieson MS. Uterine myomas in pregnancy. J Can Assoc Radiol. 1984;35:168–70.
15. Klatsky PC, Tran ND, Caughey AB, Fujimoto VY. Fibroids and reproductive outcomes: a systematic literature review from conception to delivery. Am J Obstet Gynecol. 2008;198:357–66.
16. Vergani P, Locatelli A, Ghidini A, et al. Large uterine leiomyomata and risk of cesarean delivery. Obstet Gynecol. 2007;109:410–4.
17. Chuang J, Tsai HW, Hwang JL. Fetal compression syndrome caused by myoma in pregnancy: a case report. Acta Obstet Gynecol Scand. 2001;80:472–3.
18. Romero R, Chervenak FA, DeVore G, et al. Fetal head deformation and congenital torticollis associated with a uterine tumor. Am J Obstet Gynecol. 1981;141:839–40.
19. Phelan JP. Myomas and pregnancy. Obstet Gynecol Clin N Am. 1995;22:801–5.
20. Coronado GD, Marshall LM, Schwartz SM. Complications in pregnancy, labor, and delivery with uterine leiomyomas: a population-based study. Obstet Gynecol. 2000;95:764–9.
21. Ehigiegba AE, Ande AB, Ojobo SI. Myomectomy during cesarean section. Int J Gynaecol Obstet. 2001;75:21–5.
22. Buttram VC Jr, Reiter RC. Uterine leiomyomata: etiology, symptomatology, and management. Fertil Steril. 1981;36:433–45.
23. Celik C, Acar A, Çiçek N, et al. Can myomectomy be performed during pregnancy? Gynecol Obstet Investig. 2002;53:79–83.
24. Wittich AC, Salminen ER, Yancey MK, Markenson GR. Myomectomy during early pregnancy. Mil Med. 2000;165:162–4.
25. Liu WM, Wang PH, Tang WL, et al. Uterine artery ligation for treatment of pregnant women with uterine leiomyomas who are undergoing cesarean section. Fertil Steril. 2006;86:423–8.
26. Pron G, Mocarski E, Bennett J, et al. Pregnancy after uterine artery embolization for leiomyomata: the Ontario multicenter trial. Obstet Gynecol. 2005;105:67–76.

Shoulder Dystocia

35

Vandana Rani Bhuria

35.1 Introduction

Shoulder dystocia is an unexpected, uncertain, and unpreventable obstetric emergency. It is the nightmare of obstetricians, nurses, midwives, and other healthcare providers.

Shoulder dystocia (SD) is defined as a vaginal cephalic delivery that requires additional obstetric maneuvers to deliver the fetus after the head has delivered and gentle downward traction has failed [1]. Though subjective in nature, both the American College of Obstetricians and Gynecologists Practice Bulletin [2] and the Royal College of Obstetricians and Gynaecologists Green Top Guidelines [3] are in agreement with this definition of shoulder dystocia. Shoulder dystocia (Fig. 35.1) occurs when either the anterior or, less commonly, the posterior fetal shoulder is impacted on the maternal pubic symphysis or sacral promontory [3]. Typically shoulder dystocia is diagnosed by the classic "turtle sign" (once the fetal head is delivered, it retracts back tightly against the maternal perineum) [4].

Spong and colleagues defined shoulder dystocia as a "prolonged head-to-body delivery time (e.g., more than 60 s) and/or the necessitated use of ancillary obstetric maneuvers." The 60-s interval was selected as, in their study, it was approximately two standard deviations above the mean value for head-to-body time for uncomplicated deliveries [5].

35.1.1 Incidence

Incidence of shoulder dystocia varies worldwide from 0.6% to 3% among vaginal deliveries of fetuses in vertex presentation [6]. Differences in reported incidence may be present because of clinical variations in defining shoulder dystocia and the population studied and because of overdiagnosing or underdiagnosing milder forms of shoulder dystocia [2]. Studies involving the largest number of vaginal deliveries (34,800–267,228) report incidences between 0.58% and 0.70% [7–12].

35.1.2 Pathophysiology

Of the three diameters of the pelvic brim, the anteroposterior is the narrowest, the oblique is larger, and the transverse diameter is the widest diameter. The fetal bisacromial diameter (12 cm) enters the pelvis at an oblique angle, with the posterior shoulder ahead of anterior one. It then rotates to the anteroposterior position at the pelvic outlet with the external rotation of the fetal head. The anterior shoulder then slides under the pubic symphysis for delivery.

During descent, if the fetal shoulders remain in an anteroposterior position or descend

V. R. Bhuria (✉)
Pt B. D. S. PGIMS, Rohtak, Haryana, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_35

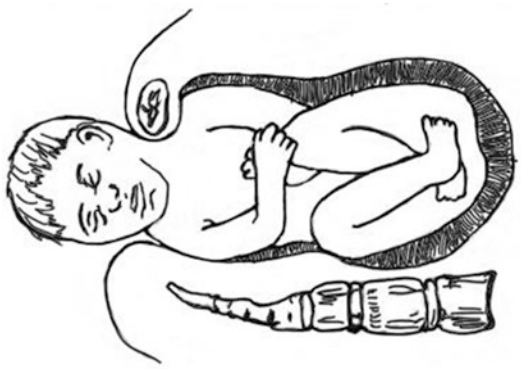


Fig. 35.1 Shoulder dystocia

simultaneously rather than sequentially into the pelvic inlet, then the conditions are set for shoulder dystocia. In these cases, it is the anterior shoulder that becomes impacted behind the pubic symphysis and the posterior shoulder almost always descends below the sacral promontory. In extremely rare cases, both the shoulders may remain above the pelvic brim, resulting in bilateral shoulder dystocia.

If the anterior or posterior shoulders remain impacted, the descent of fetal head continues; this may result in the stretching of the nerves in the brachial plexus which may cause nerve injury of fetus, further leading to neonatal brachial plexus palsy.

Compression of the umbilical cord or compression of the vessels in the fetal neck by a tight nuchal cord or by combination of both may result in fetal acidemia. With the delivery of the head, the volume of uterine contents is reduced, and the uterus contracts down which diminishes or stops the blood flow to the intervillous space. Since the fetal chest is compressed, the respiratory effort of fetus and oxygenation are impeded.

35.2 Risk Factors

Various antepartum and intrapartum factors have been reported to be associated with shoulder dystocia (Table 35.1) [3, 13, 14], but these risk factors were found to have a low positive predictive value, both singly and in combination [15, 16].

35.2.1 Shoulder Dystocia and Macrosomia

A definite relationship exists between fetal size and shoulder dystocia [17], but it is not a good predictor: partly because it is difficult to predict fetal size accurately but also because the majority of infants with birth weight of equal to more than 4500 g do not develop shoulder dystocia [18]. Equally important, 48% of the births complicated by shoulder dystocia occurs with infants weighing less than 4000 g [8]. The overall incidence of shoulder dystocia varies based on fetal weight, occurring in 0.6–1.4% of all infants with a birth weight of 2500–4000 g, increasing to a rate of 5–9% among the fetuses weighing 4000–4500 g born in mothers without diabetes [14].

The diagnosis of fetal macrosomia is precise. Several ultrasound measurements to predict macrosomia and an alert for shoulder dystocia have been proposed like abdominal circumference (AC > 350 mm) [19], newborn shoulder width [20], and 3D ultrasound weight estimation [21]. However, ACOG supports the use of the 4500 g cutoff for diagnosis of macrosomia as it is; at this rate, sharp increase is seen in risk of morbidity for infants and mothers [22].

The prediction of macrosomia by ultrasound is limited by the fact that fetal weight prediction is less accurate at higher birth weights. Moreover, the third trimester ultrasound scans have a sensitivity of just 60% for macrosomia (over 4.5 kg) [23].

35.2.2 Shoulder Dystocia and Diabetes

Infants of diabetic mothers tend to have a two- to fourfold increased risk of shoulder dystocia as compared to infants of the same birth weight born to nondiabetic mothers [15, 17]. The shoulder girth of fetus is composed of tissues that are insulin sensitive and respond to hyperglycemia and hyperinsulinism, while the head circumference and brain growth are less affected. As a result, higher shoulder-to-head circumference ratio is observed in infants of diabetic mothers

Table 35.1 Risk factors for shoulder dystocia

Sr. no.	Prepregnancy	Antepartum	Intrapartum
1	Previous shoulder dystocia	Diabetes mellitus	Oxytocin augmentation
2	Prior macrosomia	Excessive maternal weight gain	Prolonged active phase of first stage of labor
3	Pre-existing diabetes	Suspected macrosomia	Prolonged second stage of labor
4	Maternal obesity (BMI >30 kg/m^2)	Short stature	Secondary arrest in second stage
5	Prior gestational diabetes	Post-term induction	Protracted or failure of descent of head
6	Advanced maternal age		Operative or assisted vaginal delivery (forceps/vacuum)
7			Inappropriate maneuvers (fundal pressure)
8			Epidural anesthesia

further causing increased rates of shoulder dystocia.

Cohen reported that shoulder dystocia was predicted in 100% of diabetic pregnancies when the differences between the ultrasonic measurements of biparietal diameter minus the abdominal circumference exceeded 2.5 cm [24].

35.2.3 Obesity and Shoulder Dystocia

WHO defined obesity as a body mass index (BMI) of 30 or greater. Worldwide, a staggering rise has been observed in the prevalence of obesity. Etiology of obesity is multifactorial, mainly attributed to urbanization, changing dietary habits and sedentary lifestyles. A number of complications are attributed to obesity including increased risk of pregnancy loss, congenital malformations, gestational hypertension, and gestational diabetes mellitus. Intrapartum complications include increased risk of cesarean section, labor dystocia, instrumental delivery, and postpartum hemorrhage. Postpartum complications include increased rates of infection, thromboembolism, and increased hospital stay. Obese women tend to have increased risk of macrosomic infants as compared to non-obese women. Weiss reported that the incidences of macrosomia, defined as the birth weight of over 4000 g, was 8.3% in nonobese group, 13.3% in an obese group, and 14.6% among morbidly obese women [25]. The relationship between obesity and shoulder dystocia may be largely related to the increase in incidence of macrosomia seen in this group, rather than to obesity alone [26]. To support this notion, Robinson reported no increase in shoulder dystocia among obese women with infants of normal weight [27]. Thus, to conclude, obese women with macrosomic baby pose as a risk factor for shoulder dystocia, but obesity alone cannot be taken as independent risk factor for shoulder dystocia.

35.2.4 Recurrent Shoulder Dystocia

A recent study showed that recurrent shoulder dystocia was higher than the general population (3.7% vs. 0.7%, OR 7.36, 95% CI 3.68–14.23, $P < 0.01$) [28]. However, the rate of recurrence appears to be lower than previously estimated by Bingham et al. [29] in 2010. According to their study, about 12% of parturients with a history of shoulder dystocia have a recurrent dystocia in subsequent pregnancy with a risk of about 1 in 8 (OR 8.25). Brachial plexus injury occurs in 19/1000 vaginal births during the first episode of shoulder dystocia and in 45/1000 vaginal births after recurrent shoulder dystocia [29]. However, the true incidence of recurrent shoulder dystocia remains unknown because obstetrician and patients often do not choose to attempt a trial of labor when there was a history of complicated delivery or an injured infant with brachial palsy [2]. However, in cases of previous shoulder dystocia and brachial plexus injury or other complication, cesarean section could be justified in next pregnancy. The woman and her attendants

Table 35.2 Complications of shoulder dystocia

Fetal complications	Maternal complications
Brachial plexus injury (most common)	Postpartum hemorrhage (both atonic and traumatic)
Fetal distress with or without permanent neurological damage	Complete perineal tears
Fetal death	Extension of episiotomy, vulval hematoma
Clavicular fractures	Bladder atony/rupture
Fractures of humerus	Symphyseal separation or diathesis with or without transient femoral neuropathy
	Rectovaginal fistula
	Uterine rupture

should always be explained about the risk of recurrence and should be given option of either the cesarean section or vaginal delivery. The factors such as the severity of any previous neonatal injury, any serious maternal intrapartum event, predicted fetal weight, and choice of mother and family should all be considered and discussed with the woman and her family while making plans for route of delivery.

35.3 Maternal and Neonatal Consequences

Shoulder dystocia results in a lot of maternal and fetal complications (Table 35.2). In general, shoulder dystocia poses a greater risk to the fetus than the mother. The number of maneuvers employed to relieve shoulder dystocia is directly proportional to the increase in fetal and neonatal morbidity [30].

35.3.1 Fetal Complications

1. Neonatal Brachial Plexus Palsy (NBPP)
   Brachial plexus injury is the most common and serious complication, occurring in 5–15% of neonates born after shoulder dystocia. The most common injuries can result in Erb's or Erb-Duchenne palsy (injury to upper brachial plexus nerve roots, C5–C6) or Klumpke's palsy (injury to the lower nerve roots, C8–T1). Rarely, the whole brachial plexus will be injured leading to flail arm.

   A stretching force is transmitted to the tissues that connect the fetal trunk and the fetal head-neck, under which lies the brachial plexus, leading to neonatal brachial plexus palsy (NBPP). Conjunction of events generating stretching and compression forces leading to NBPP are enumerated as follows:

   (a) Impaction of either the anterior or posterior shoulder behind the pubic symphysis or sacral promontory, respectively, while the long axis of the fetus is pushed down the birth canal with each uterine contraction.
   (b) When the fetal neck is compressed against the maternal pubic symphysis or sacral promontory.
   (c) Any amount of traction applied by the birth attendant.

   Neonatal Response

   Neonatal response to injury in form of stretching and compression forces in cases of shoulder dystocia varies and depends on many variables like tensile strength of fetal tissues, the degree of protective tone of muscles around fetal shoulders/neck, and the metabolic condition of fetus (measured by acid-base status).

   In 2014, the ACOG Task Force on neonatal brachial plexus palsy (NBPP) reviewed the current literature, including consensus opinion as well as multiple published cases of peer-reviewed literature related to brachial plexus injury. This report concludes that NBPP occur in 1.5 of 1000 births. Only 50% of NBPP cases are

preceded by shoulder impaction. Greater than 80% cases of NBPP occur in women without any risk factors. In fact, NBPP has not only been associated with routine vaginal deliveries not complicated by shoulder impaction but even with routine cesarean deliveries in 4% of cases. NBPP is associated with 4–40% of clinically apparent cases of shoulder impaction. Most injuries resolve, however, the incidence of persistent NBPP at 1 year of life ranges from 0.5% to 1.6% [31, 32].

Although shoulder dystocia and disimpaction maneuvers historically have been attributed for the etiology of these palsies, brachial plexus injury may occur in utero [33]. Probable mechanisms of intrauterine insult include the endogenous propulsive forces of labor, in utero position of the fetus, failure of the shoulders to rotate, and abnormal intrauterine pressures arising from uterine anomalies (such as fibroids, intrauterine septum, bicornuate uterus); all these injuries may also contribute to brachial plexus injury [13, 14].

The timing of brachial plexus injury can be determined by electromyelography within 24–48 h of delivery. Electromyelographic evidence of muscular denervation normally requires 10–14 days to develop. Its presence in the early neonatal period strongly suggests an insult predating delivery [13].

2. Fetal Asphyxia
   In shoulder dystocia, the combination of hypoxia, obstructed cerebral venous return, and trauma during delivery renders the fetal brain vulnerable to damage. The umbilical artery pH is estimated to theoretically fall 0.04 pH units per minute in the interval between delivery of the fetal head and trunk (mean umbilical artery pH at term is 7.27). In general, to deliver a previously well-oxygenated fetus with normal acid-base status, the operator has 4–5 min to deliver the fetus before the possibility of permanent hypoxic damage.

   Thus, there is time for the logical and systemic application of maneuvers to safely deliver the fetus before the permanent hypoxic damage. On the other hand, if the fetus is already hypoxic when shoulder dystocia occurs, the time to permanent hypoxic damage may be much shorter.

   Severe cases of shoulder dystocia may even end in hypoxic ischemic encephalopathy and fetal death.
3. Fractures
   Fractures of clavicle and less commonly humerus are also reported in infants after shoulder dystocia. These fractures typically heal well without any deformities. Fracture or dislocation of the cervical spine is extremely infrequent but can be associated with desperate and ill-advised twisting maneuvers of the fetal head.

35.4 Maternal Complications

1. Genital Tract Lacerations
   Since extra space is required for the maneuvers to overcome shoulder dystocia; thus, extension of episiotomy and third and fourth degree perineal tears are more common with shoulder dystocia. With more sphincter lacerations, the potential exists for a higher incidence of long-term flatal and fecal incontinence.
2. Postpartum Hemorrhage
   Both traumatic (bleeding from lacerations) and atonic postpartum hemorrhage is seen after shoulder dystocia.

35.5 Prediction and Prevention

Shoulder dystocia is considered as a fatal, unpredictable, and unpreventable obstetrical event. Even the identification of risk factors like suspected fetal macrosomia, a history of prior shoulder impaction, excessive weight gain in pregnancy, prolonged pregnancy, advanced maternal age, male fetal gender, multiparity, oxytocin augmentation, and prolonged second stage

and epidural anesthesia, the occurrence of shoulder dystocia cannot be accurately predicted. The predictive value of any one or combination of risk factors for shoulder dystocia is not high enough to be useful for prediction and prevention of shoulder dystocia. Therefore, accurate prediction of shoulder dystocia is not possible, and thus, attempts to allow a practical prevention strategy have been unsuccessful till now.

35.5.1 Prevention

Identification of the risk factors predisposing to shoulder dystocia and anticipation of the problem may prove beneficial for the prevention of shoulder dystocia. The relationship between fetal macrosomia and shoulder dystocia is well recognized. The concept of elective induction or elective cesarean section for prevention of shoulder dystocia in macrosomic babies needs a second thought and is discussed.

ACOG states that "Elective induction of labor or elective cesarean delivery for all women suspected of carrying a macrosomic fetus is not appropriate" due to the fact that ultrasound is not considered as an accurate predictor of macrosomia [2].

The RCOG also affirms that elective cesarean section is not recommended for pregnancies complicated by suspected fetal macrosomia without maternal diabetes mellitus [3].

It has been evaluated that to prevent one permanent neonatal brachial plexus injury, an additional 2345 cesarean sections would be required at a cost of 4.9 million dollars per year, if cesarean section would be done for all fetuses suspected of weighing 4000 g or more [34].

Although the diagnosis of fetal macrosomia is imprecise, planned cesarean delivery may be considered for suspected fetal macrosomia with estimated fetal weight exceeding 5000 g in women without diabetes and 4500 g in women with diabetes (level C recommendation, ACOG) [2].

RCOG does not support induction of labor in women without diabetes at term where the fetus is thought to be macrosomic (grade A recommendation, RCOG) [3]. A meta-analysis of randomized controlled trials of the effect of treatment in women with gestational diabetes concluded that early induction of labor reduces the incidence of shoulder dystocia [35]. The NICE diabetes guidelines recommend that diabetic pregnant women who have a normally grown fetus should be offered elective birth through induction of labor or by elective cesarean section if indicated, after 38 completed weeks [36].

35.5.2 Diagnosis

Shoulder dystocia should be suspected when the underlying signs are present:

1. Slow crowning of the fetal head.
2. Difficulty with the delivery of face or chin.
3. The fetal head retracts into the perineum (turtle sign) after expulsion due to reverse traction from shoulders being impacted at the pelvic inlet.
4. Failure of restitution of the fetal head.
5. Failure of descent of shoulders.

Diagnosis is made when:

- Gentle downward traction of the fetal head fails to accomplish delivery of the anterior shoulder.

35.6 Management

The key to management of shoulder dystocia is anticipation and planning. The important principle to remember is that the problem with the shoulder is at the level of the pelvic brim. Thus, traction or twisting movements involving the head are illogical, ineffective, and potentially traumatic.

If the shoulder dystocia is diagnosed, avoid the P's.

1. Panic.
2. Pulling on the head. Never put strong downward pressure on the fetal head as it may cause injury to brachial plexus.
3. Pushing (on the fundus). Fundal pressure is illogical and can only compound the problem by forcing the anterior shoulder against the unyielding symphysis.

4. Pivoting (sharply angulating the head using coccyx as a fulcrum).

Thus, excessive force to fetal head or neck and fundal pressure should be avoided, as they are unlikely to free the impaction and may further impact the shoulders and cause uterine rupture or other injury.

Timely management of shoulder dystocia requires prompt recognition and action. The health attendant should be instructed to observe for the signs of shoulder dystocia and to be vigilant for timely action. At the point when shoulder dystocia is diagnosed, one of the major concerns is the risk of fetal hypoxia, further leading to hypoxic ischemic encephalopathy and further leading to infant death. Fetal hypoxic insult results from a number of reasons which include umbilical cord compression between fetal body and maternal pelvis and compression of neck and central venous congestion. This further reduces placental intervillous flow from prolonged increased intrauterine pressure and causes secondary fetal distress. The fifth CESDI report on shoulder dystocia identified that 47% of the babies died within 5 min of the head being delivered [37]. It is, therefore, important to manage the problem efficiently and carefully, efficiently as to avoid fetal hypoxic acidosis, and carefully so as to avoid unnecessary trauma (RCOG level III evidence) [3].

For the above mentioned reasons, shoulder dystocia should be managed systematically. The Advanced Life Support in Obstetrics has offered a structural framework for managing shoulder dystocia in a systematic manner, the "HELPERR" mnemonic [38].

H—Call for help.
E—Evaluate for episiotomy.
L—Legs (McRobert's maneuver).
P—Suprapubic pressure.
E—Enter maneuvers (internal rotation).
R—Remove the post arm.
R—Roll the patient (all-fours positions).

The HELPERR mnemonic is designed to do one of the three things:

1. Increase the functional size of the bony pelvis.
2. Application of suprapubic pressure leads to decrease in bisacromial diameter of the fetus.
3. Internal rotation maneuvers change the relationship of the bisacromial diameter within the bony pelvis.

35.6.1 H—Call for Help

Once shoulder dystocia is diagnosed, immediately call for extra help including an expert obstetrician, a pediatric resuscitation, anesthetist, and midwives.

The woman should be made to bring the buttocks to the end of the bed.

- Provide a simple and clear explanation to the woman about the actions you are about to undertake.
- Ask for cooperation from the woman.
- Once diagnosed, the presence of additional assistance in the delivery room is critical. One person is designated for recording of events, obtaining required equipments, and notifying the clinician of time intervals.
- Documentation of maneuver used and the duration of each maneuver may be valuable to prompt the clinician to move on to other maneuvers, rather than persisting in one that is not working.
- The appropriate amount of time to be spent on each maneuver is 30 to 60 s.

The orders of steps need not to be the same as mnemonic suggests; it is more important that the maneuvers be employed efficiently and appropriately.

35.6.2 E—Evaluate for Episiotomy

Since shoulder dystocia is a bony impaction, so performing an episiotomy will not release the shoulders. Episiotomy is only required to make space for the obstetrician's hand in the vagina for performing internal maneuvers, if required. Since maximum cases of shoulder dystocia are relieved by McRobert's maneuver and suprapubic pressure, episiotomy is not necessary in all the

cases of shoulder dystocia, unless internal maneuvers are required to release the shoulder (RCOG grade B recommendation) [3].

However, episiotomy may be more difficult to perform when the fetal head is tight against the perineum. It will be beneficial to perform episiotomy, if shoulder dystocia is strongly anticipated.

35.6.3 L—Legs (McRobert's Maneuver)

Mechanism: This maneuver rotates the symphysis superiorly and straightens the lumbosacral angle. These motions push the posterior shoulder over the sacral promontory, allowing it to fall into the hollow of the sacrum and rotate the symphysis over the impacted shoulder.

In addition, it reduces the angle of inclination of the pelvis so that plane of pelvic inlet is brought perpendicular to the expulsive forces. The combination of these changes in the fetopelvic relationship reduces the propulsion-extraction forces necessary to deliver the shoulders.

Technique: With the woman in the recumbent position, the maternal hips are slightly abducted and acutely flexed by bringing the knees toward the chest, i.e., hyperflexion of maternal thighs against the abdomen (Fig. 35.2). Routine traction (as applied during a normal delivery) in an axial direction should then be applied to the fetal head to assess whether shoulders have been released.

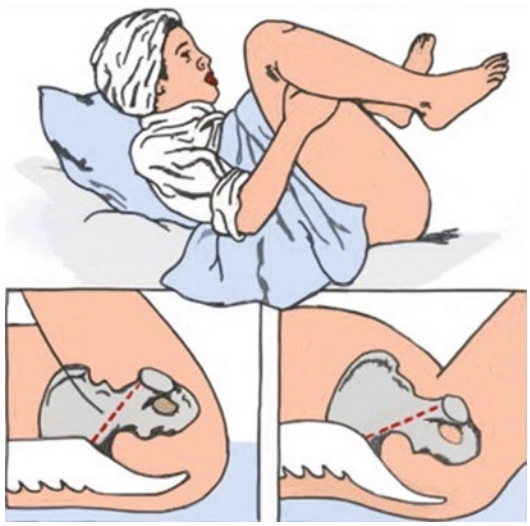


Fig. 35.2 McRobert's maneuver

McRobert's maneuver is considered to be simple and most effective intervention, making it an ideal first step in management. The success of McRobert's maneuver in resolving shoulder dystocia (used either alone or in combination with suprapubic pressure) is reported between 42% and 90% [3].

McRobert's maneuver has a low rate of complication; therefore, its performance is a reasonable initial approach (level C recommendation, ACOG) [2].

However, overly continued and aggressive hyperflexion and abduction of the maternal thighs onto the abdomen should be avoided because this is often associated with increased traction further leading to increased risk of brachial plexus injury.

35.6.4 P—Suprapubic Pressure (Rubin's I Maneuver)

Mechanism: The suprapubic pressure pushes the posterior aspect of anterior shoulder toward the fetal chest further reducing the bisacromial diameter. Actually, pushing behind the scapula tends to adduct the shoulders which are a narrower diameter than the abducted shoulders.

Technique: External manual suprapubic pressure is applied for approximately 30–60 s while the delivering personnel continues gentle traction.

The suprapubic hand should be placed over the fetus' anterior shoulder applying pressure in a "CPR" style in such a way that the shoulder will adduct or collapse anteriorly and pass under the symphysis. Simultaneously while the woman is placed in McRobert's position, place both the hands over the posterior aspect of fetal shoulder, and apply continuous pressure in a downward and lateral motion (Fig. 35.3). Initially continuous pressure is applied; but if it fails, a rocking motion is recommended to dislodge the shoulder from behind the pubic symphysis. Simultaneously, gentle traction is applied to deliver the shoulder.

If this maneuver fails, the next maneuver should be immediately attempted.

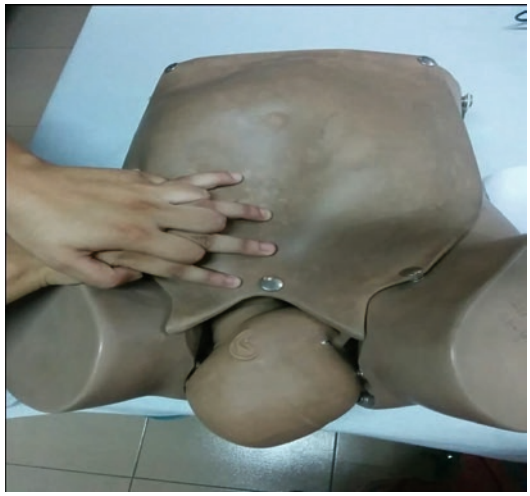


Fig. 35.3 Suprapubic pressure

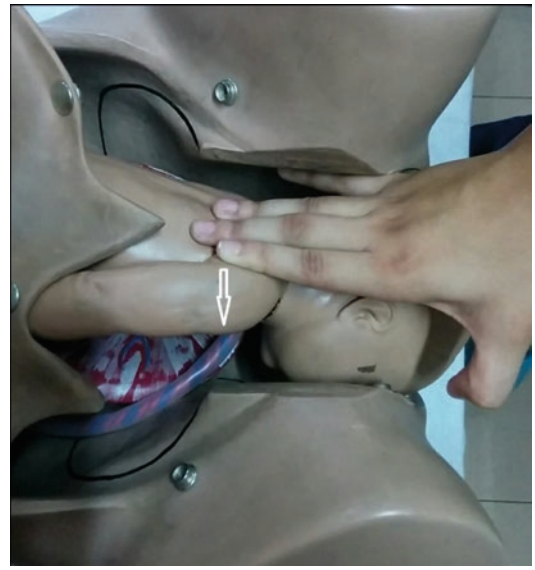


Fig. 35.4 Rubin's II maneuver

If these simple maneuvers fail, then, the obstetrician should make a choice between the all-fours position and internal manipulation. The clinical judgment, individual circumstances, and experience of the delivery personnel should guide the obstetrician to decide their order. In the HELPERR mnemonic, the following order is suggested:

35.6.5 E—Enter Internal Maneuvers

35.6.5.1 Rubin's II Maneuver

The narrowest diameter of the pelvic brim is the anteroposterior diameter; so, it is logical to rotate the fetal shoulders to wider oblique diameter. Under no circumstances should one twist the head in an attempt to rotate the shoulders. It will not work and will cause trauma to the brachial plexus and cervical spine.

In Rubin's II maneuver, digital pressure is applied to the posterior aspect of anterior shoulder with two fingers of the hand inserted inside the vagina pushing it toward the fetal chest. This rotates the shoulders forward into wider oblique diameter (Fig. 35.4). McRobert's maneuver can still be simultaneously applied during this maneuver which may help facilitate its success.

If the Rubin's II maneuver is unsuccessful, then the obstetrician should proceed to the Woods corkscrew maneuver without any delay.

35.6.5.2 Woods Corkscrew Maneuver

Principle: Woods (1943) described the relationship between the fetal shoulders and the pelvic bony landmarks which they must traverse: the symphysis pubis, sacral promontory, and coccyx. Using wooden models, he described that the relationship between the fetal shoulders and these bony landmarks was similar to the threads of a screw. It is not possible to push or pull the shoulders through, but they can be "corkscrewed" through the pelvis by rotating the shoulders 180° [39].

While maintaining the pressure of Rubin's II maneuver, the obstetrician introduces the second hand and two fingers are placed on the anterior aspect of the fetal posterior shoulder. The fetal posterior shoulder is pushed off the midline to rotate the fetus 180° (Fig. 35.5). Because the fetal posterior shoulder is below the pelvic brim, as it rotates, it remains at that level and once in the anterior position is accessible for delivery. During this 180° rotation, the former anterior shoulder which started above the pelvic brim rotates to become the posterior shoulder and, in doing so, rotates below the level of the pelvic brim. Woods screw maneuver correctly defines the relationship between the fetal shoulders and the pelvic brim and provides a logical solution, in the same

manner that Lovset's maneuver does for delivery extended arms in breech presentation.

35.6.5.3 Reverse Woods Screw Maneuver

If Rubin's II maneuver or Woods screw maneuver fails, then we should move to reverse Woods screw maneuver.

In this maneuver, the fingers are placed on the back of the posterior shoulder of the fetus and attempt to rotate it through 180° in a direction opposite to that of Woods screw maneuver (Fig. 35.6). This maneuver adducts the fetal posterior shoulder, thus rotating the shoulders into an oblique plane for delivery.

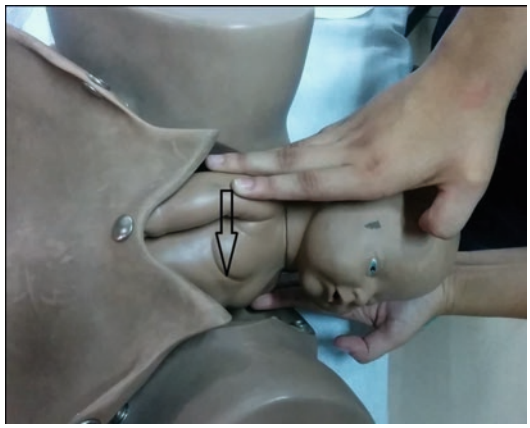


Fig. 35.5 Woods screw maneuver

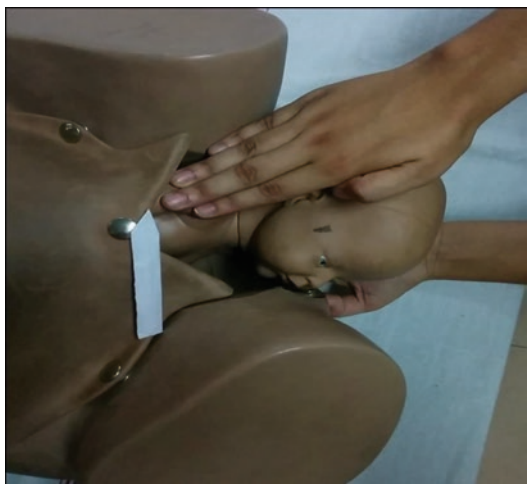


Fig. 35.6 Reverse Woods screw maneuver

At times, these maneuvers are very difficult to perform, and it becomes necessary to push the fetus into the pelvis slightly to accomplish the maneuvers, especially in cases when the anterior shoulder is partially wedged under the symphysis.

35.6.6 R—Remove the Posterior Arm (Jacquemier's Maneuver)

Unless it is the case of bilateral shoulder dystocia, in all other cases, the posterior shoulder is accessible as it has descended beneath the sacral promontory into the sacral bay.

In order to effect this maneuver, the obstetrician must insert his hand far into the vagina and attempt to locate the posterior arm. Once forearm is located, the elbow is flexed so that arm can be delivered by a sweeping motion over the chest wall of the fetus. In practice, however, the fetus is so tightly lodged in the pelvis that this may not be successful. If so, one just has to reach further along the fetal forearm until one can grasp it and pull the arm across the fetal chest (Fig. 35.7). If done in correct manner, first the posterior hand followed by the arm and finally shoulder will be reduced, facilitating delivery of the infant. As the arm is removed, the fetus rotates in a corkscrew manner.

If delivery is not accomplished, then, support the posterior shoulder and trunk of the fetus, and rotate them 180°, which will bring the anterior shoulder to become the posterior shoulder, below the level of the pelvic brim, and allow its delivery.

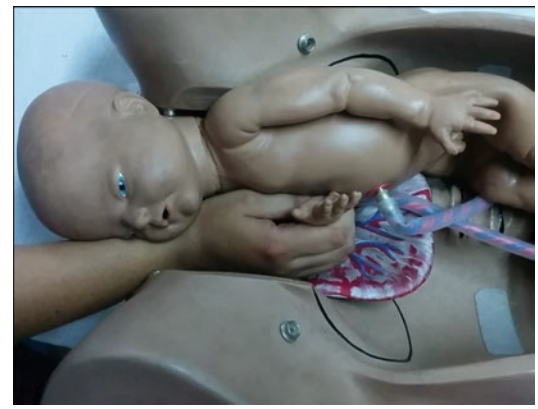


Fig. 35.7 Remove the posterior arm (Jacquemier's Maneuver)

Jacquemier's maneuver is one of the maneuvers most likely to cause fractures of the clavicle and humerus. However, it is almost always successful in delivering the fetus, and these fractures heal well without long-term sequelae, whereas the alternatives of fetal hypoxia, brachial plexus palsy, and cervical spine injury may not.

35.6.7 R—Roll the Patient (All-Fours Maneuver or Gaskin Maneuver)

Gaskin maneuver is a safe, effective, and rapid technique, named after midwife, Ina May Gaskin. In this maneuver, the woman is guided to the all-fours position on her hands and knees. This maneuver exploits the effect of gravity, thereby, increasing space in the hollow of sacrum to facilitate delivery of the posterior shoulder and arm (Fig. 35.8).

Theoretically, the flexibility of sacroiliac joint may allow a 1–2 cm increase in the anteroposterior diameter of the pelvic brim. As patient is turned from a supine to all-fours position, the sagittal measurements of the pelvic outlet increase up to 20 mm, and the true obstetrical conjugate increases by as much as 10 mm. After rotating to all-fours position, the fetal shoulder dislodges, and with the gentle traction, the posterior shoulder is delivered first.

Fig. 35.8 All-fours maneuver or Gaskin maneuver

Disadvantages:

1. To assist woman into all-fours position, it may require a number of personnel, and it is difficult for a woman who is fatigued by process of labor or restricted by intravenous lines, fetal monitors, or epidural anesthesia to stay in this position.
2. Clinicians who are unfamiliar attending delivery in all-fours position may find difficulty in conducting delivery in this position. To overcome this problem, one should go with the gravity first and, thus, provide gentle downward traction to deliver the shoulder closest to the ceiling first.

35.6.8 "Last Resort" Maneuvers

"Last resort" or third-line maneuvers have been described, and clinician should proceed to these maneuvers in cases of failure of maneuvers dictated in the "HELPERR" mnemonic. These include:

1. Cleidotomy
2. Muscle relaxation
3. Zavanelli maneuver
4. Abdominal rescue
5. Symphysiotomy

35.6.8.1 Cleidotomy

Cleidotomy means deliberate fracture of the fetal clavicle. It reduces the fetal bisacromial diameter, thereby, making the shoulders more flexible. Due to high complication rate, it is usually reserved for a dead fetus or an anomaly in the fetus incompatible with life (anencephaly).

Technique: Direct pressure is applied on the midportion of the fetal clavicle which will cause fracture of the clavicle, thereby reducing the bisacromial diameter. Cleidotomy can be performed with a strong straight embryotomy scissors. With the help of scissors, first cut the skin over the clavicle, and push the scissors round the bone. Considerable strength is often required for performing cleidotomy.

Complications: Underlying subclavian vessels and brachial plexus are vulnerable to trauma.

35.6.8.2 Muscle Relaxation

Agents like halothane and sublingual nitroglycerine can be used to achieve musculoskeletal or uterine relaxation.

"Heroic" Measures

If the several attempts of the above measures are also unsuccessful, the following techniques have been described for cases of catastrophic shoulder impaction.

35.6.8.3 Zavanelli Maneuver (Cephalic Replacement)

Rare cases of shoulder dystocia, especially the bilateral shoulder dystocia, which are unresponsive to traditional maneuvers, cephalic replacement may prove as most appropriate. If the other more traditional maneuvers fail, it is important not to resort to trauma but to consider cephalic replacement.

The normal mechanism of delivery of the head of fetus is reversed by grasping the head, rotating it to the occipito-anterior position and flexing the head, which allows replacement in the vagina (Fig. 35.9).

In some cases, presumably those with bilateral shoulder dystocia, the head will return to the vagina with remarkable ease. However, in other cases, tocolysis will be required to assist this maneuver. Uterine relaxation can be achieved with intravenous nitroglycerine or terbutaline, and this can also help restore the uteroplacental circulation and fetal oxygenation. Once the fetal head has been reposited to the vagina and the fetal heart is normal, then, the baby is delivered by cesarean section.

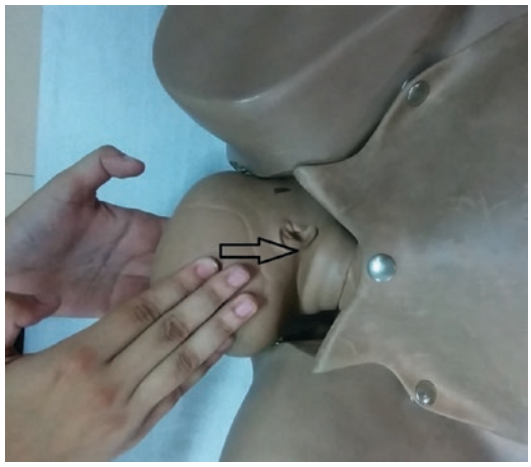


Fig. 35.9 Zavanelli technique

Before proceeding to Zavanelli maneuver, an operating team with expert obstetrician, anesthetist, and a neonatologist must be present. In cases where nuchal cord has already been clamped and cut, we should never attempt cephalic replacement.

While this technique is often successful, there can be associated maternal morbidity with uterine rupture in up to 5% of cases and blood transfusion required in some 10% [40, 41]. Perinatal death and morbidity from asphyxia and trauma are also encountered. These may have been due to other methods tried and failed before cephalic replacement.

35.6.8.4 Abdominal Rescue

This is another rare alternative approach for cases in which both the traditional maneuvers and cephalic replacement have failed. Abdominal rescue was described by O'Shaughnessy where a lower uterine segment incision facilitates vaginal delivery of impacted shoulders [42].

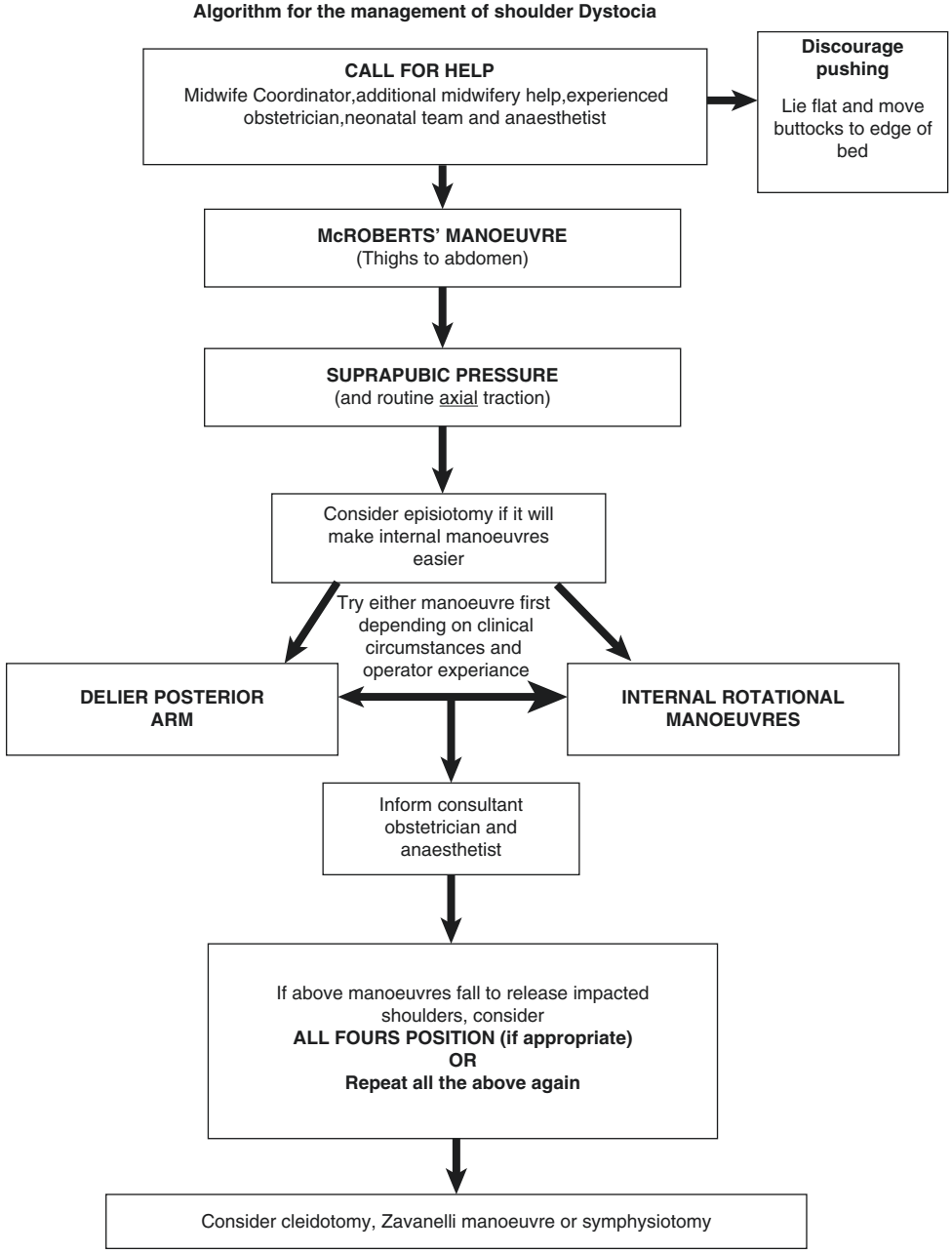
After confirming that the fetus is still alive, a lower segment cesarean section is done. The surgeon will then rotate the shoulder to the oblique position, facilitating descent of the posterior shoulder beneath the promontory where it can then be delivered directly. This is then followed by rotation of fetal body and rotation of former anterior shoulder beneath the pelvic brim to allow its delivery also.

35.6.8.5 Symphysiotomy

Symphysiotomy is a procedure in which the fibrous cartilage of pubic symphysis is divided under local anesthesia. However, none of the published series favors its successful use for relieving shoulder dystocia. Symphysiotomy is also associated with serious maternal morbidity and poor neonatal outcome (level III evidence, RCOG) [3].

Since there is potential low chance of fetal survival after significantly prolonged entrapment, this should be taken into account before performance of these heroic maneuvers. Additionally, the associated significant maternal morbidity and mortality must be weighed against the likelihood of intact fetal survival.

The management protocol for shoulder dystocia is presented in algorithm [43] below:



Baby to be reviewed by neonatalogist after birth and referred for Consultant Heonatal review if any concerns
DOCUMENT ALL ACTIONS ON PROFORMA AND COMPLETE CLINICAL INCIDENT REPORTING FORM.

35.7 The Role of Fundal Pressure

Fundal pressure in case of shoulder dystocia is illogical as it can only compound the problem by forcing the anterior shoulder against the unyielding symphysis. Fundal pressure is associated with increased neonatal complications and may even result in uterine rupture [18].

35.7.1 Posterior Axilla Sling Traction (PAST) for Intractable Shoulder Dystocia

Cluver and Hofmeyr [44] had recorded 19 cases of shoulder dystocia where PAST had been used, and it was successful in 18 and partially successful in 1 case. They had confirmed that when all other maneuvers for shoulder dystocia prove to be unsuccessful, PAST (posterior axillary sling traction) can be lifesaving resort. In this technique, a sling in form of a suction catheter or a firm urinary catheter is placed around the posterior axilla. The suction catheter is folded over operator's index finger forming a loop which is then fed posteriorly behind the posterior shoulder. The loop is pulled through creating a sling around the posterior shoulder. Traction is then applied to the sling to deliver the posterior shoulder. After that, the posterior arm is swept out easily as space is created after delivery of the posterior shoulder. If the abovementioned technique fails, shoulders can be rotated with help of sling. The sling traction is directed laterally toward the side of baby's back and then anteriorly, while digital pressure is applied behind the anterior shoulder to assist rotation. This is still a new technique, and until more safety data are available, it should be kept reserved for cases in which other commonly used techniques have failed [44].

35.7.2 Postpartum Management

Maternal complications associated with shoulder dystocia are postpartum hemorrhage and complete perineal tears. Cervicovaginal exploration should always be done after delivery of the baby to rule out cervical tears and vaginal lacerations. Other reported complications like bladder rupture, uterine rupture, symphyseal separation, sacroiliac joint dislocation, and lateral femoral cutaneous neuropathy should always be kept in mind and ruled out to avoid the long-term complications.

The neonatal complications include brachial plexus injury, clavicle and humerus fractures, fetal asphyxia and sequelae, neurological damage, and fetal demise. The care of newborn after shoulder dystocia should involve a multidisciplinary approach including pediatrics, pediatric neurology, physical therapy, and possible reference to a brachial plexus injury center. Clear communication with the parents regarding care plan should be done.

35.7.3 Risk Management

35.7.3.1 Training

Annual "skill drills" including shoulder dystocia, to improve awareness and for training of birth attendants, are recommended jointly by both the Royal College of Midwives and the RCOG [45].

35.8 Medical Simulation

Medical simulation is a relatively new field to provide training for rare, potentially catastrophic events that requires coordinated team work. The aim of medical simulation is to improve procedural competency and quality of patient care and to reduce maternal and neonatal morbidity and mortality in obstetrical emergencies. Shoulder dystocia, being highly unexpected and unpredictable, poses a huge challenge for the healthcare workers and so is well suited for simulation training. Trained individuals are allowed to recognize the problem and to effectively and rapidly apply the maneuvers so that the fetal shoulder is disimpacted and the neonate is handed over to pediatrician in a timely fashion [46].

Simulation training with repeated skill practice provides an opportunity to the healthcare

workers to experience the emergency scenario of shoulder dystocia without any risk to the actual patients and the neonates.

In the Unites States, shoulder dystocia training is now recommended by the Joint Commission on Accreditation of Healthcare Organizations. However, ACOG concludes—despite studies from Draycott and colleagues and others [47, 48]—that, owing to "limited data," till date "there is no evidence that introduction of simulation training can reduce the frequency of persistent NBPP."

35.9 Documentation

After a shoulder dystocia, the event should be properly documented in the medical record. Always document the truth that you were aware of potential complications and you refrained from panicking, responded appropriately, and performed the appropriate maneuvers. The key points that should be mentioned:

- Risk factors for shoulder dystocia, if any
- Estimated fetal weight and pelvimetry
- Documentation of any labor abnormality
- Names of staff persons present at delivery, their role, and the time they arrived
- Timing of delivery of head
- Timing of delivery of shoulders
- Position of fetal head at time of delivery
- Which shoulder was the anterior shoulder
- Maneuvers performed, their timing and sequence
- Whether episiotomy was performed and lacerations, if any
- Whether fetal injuries were identified
- The estimated blood loss
- APGAR score
- The results of cord blood gas analysis

Poor inadequate documentation can be associated with serious medicolegal consequences. The use of structured proforma has been suggested to improve accurate record keeping in the clinical setting [7]. ACOG and RCOG [43] both have standardized reporting forms.

35.10 Conclusion

Despite its rarity, all healthcare professionals attending deliveries must be aware with the risk factors, management, and documentation of shoulder dystocia. A team oriented approach is imperative for the management of shoulder dystocia with each team member familiar and trained in the manoeuvres used for relieving shoulder dystocia. RCOG has recommended annual skill drills for shoulder dystocia. Key points in successful management of shoulder dystocia include anticipation, constant preparedness, team approach, and appropriate documentation.

35.11 Future Perspective

Future directions include:

- Further research on accurate prediction of risk factors of shoulder dystocia
- Improvement in diagnostic imaging for a more accurate prediction of fetal macrosomia
- Additional studies for more information about how shoulder dystocia related brachial plexus injury occur
- Prospective evaluation of periodic skill drills

References

1. Resnick R. Management of shoulder dystocia girdle. Clin Obstet Gynecol. 1980;23:559–64.
2. American College of Obstetricians and Gynecologists. Shoulder dystocia. ACOG practice bulletin clinical management guidelines for obstetrician-gynecologists. Number 40, November 2002. Obstet Gynecol. 2002;100:1045–50.
3. Royal College of Obstetricians and Gynaecologists. RCOG guideline no. 42. Dec 2005.
4. Gherman RB. Shoulder dystocia: prevention and management. Obstet Gynecol Clin N Am. 2005;32:297–305.
5. Spong CY, Beall M, Rodrigues D, et al. An objective definition of shoulder dystocia: prolonged head-to-body delivery intervals and/or the use of ancillary obstetric maneuvers. Obstet Gynecol. 1995;86:433–6.

6. Gherman RB. Shoulder dystocia: an evidence-based evaluation of the obstetric nightmare. Clin Obstet Gynecol. 2002;45:345–62.
7. McFarland M, Hod M, Piper JM, Xenakis EM, Langer O. Are labor abnormalities more common in shoulder dystocia? Am J Obstet Gynecol. 1995;173:1211–4.
8. Baskett TF, Allen AC. Perinatal implication of shoulder dystocia. Obstet Gynecol. 1995;86:14–7.
9. Gherman RB, Ouzounian JG, Goodwin TM. Obstetric maneuvers for shoulder dystocia and associated fetal morbidity. Am J Obstet Gynecol. 1998;178:1126–30.
10. McFarland MB, Langer O, Piper JM, Berkus MD. Perinatal outcome and the type and number of maneuvers in shoulder dystocia. Int J Gynaecol Obstet. 1996;55:219–24.
11. Ouzounian JG, Gherman RB. Shoulder dystocia: are historic risk factors reliable predictors? Am J Obstet Gynecol. 2005;192:1933–5; discussion 1935–8.
12. Smith RB, Lane C, Pearson JF. Shoulder dystocia; what happens at the next delivery? Br J Obstet Gynecol. 1994;101:713–5.
13. Gottlirb AG, Galan HL. Shoulder dystocia: an update. Obstet Gynecol Clin N Am. 2007;34:501–31.
14. Baxely EG, Gobbo RW. Shoulder dystocia. Am Fam Physician. 2004;69(7):1707–14.
15. Nesbitt TS, Gilbert WM, Herrchen B. Shoulder dystocia and associated risk factors with macrosomic infants born in California. Am J Obstet Gynecol. 1998;179:476–80.
16. Bahar AM. Risk factors and fetal outcome in cases of shoulder dystocia compared with normal deliveries of a similar birth weight. Br J Obstet Gynaecol. 1996;103:868–72.
17. Acker DB, Sachs BP, Friedman EA. Risk factor for shoulder dystocia. Obstet Gynecol. 1985;66:762–8.
18. Gross TL, Sokol RJ, Williams T, Thompson K. Shoulder dystocia: a fetal-physician risk. Am J Obstet Gynecol. 1987;156:1408–18.
19. Maticot-Baptista D, ACollian A, Martin R, Maillet DR. Prevention of shoulder dystocia by an ultrasound selection at the beginning of labour of foetuses with large abdominal circumference. J Gynecol Obstet Biol Reprod (Paris). 2007;36(1):42–9.
20. Verspyck E, Goffinet F, Hellot M-F, Milliez J, Marpeau L. Newborn shoulder width: determinant factors and predictive value for shoulder dystocia. J Gynecol Obstet Biol Reprod. 2000;29:192–6.
21. Schild RL, Fimmers R, Hansmann M. Fetal weight estimation by three-dimensional ultrasound. Ultrasound Obstet Gynecol. 2000;16(5):445–52.
22. American College of Obstetricians and Gynecologists. Fetal macrosomia. ACOG practice bulletin clinical management guidelines for obstetrician-gynecologists. Number 22. Washington, DC: American College of Obstetricians and Gynecologists; 2000.
23. Rouse DJ, Owen J. Prophylactic caesarean delivery for fetal macrosomia diagnosed by means of ultrasonography—a Faustian bargain? Am J Obstet Gynecol. 1999;181:332–8.
24. Cohen B, Penning S, Major C, Ansley D, Porto M, Garite T. Sonographic prediction of shoulder dystocia in infants of diabetic, others. Obstet Gynecol. 1996;88(1):10–3.
25. Weiss JL, Malone FD, Emig D, et al. Obesity, obstetric complications and cesarean delivery rate-a population-based screening study. Am J Obstet Gynecol. 2004;190(4):1091–7.
26. Usha Kiran TS, Hemmadi S, Bethel J, Evans J. Outcome on pregnancy in a women with an increased body mass index. BJOG. 2005;112(6):768–72.
27. Robinson H, Tkatch S, Mayes DC, Bott N, Okun N. Is maternal obesity a predictor of shoulder dystocia? Obstet Gynecol. 2003;101(1):24–7.
28. Ouzounian JG, Gherman RB, Chauhan SP. Recurrent shoulder dystocia: analysis of incidence and risk factors. Am J Perinatol. 2012;29(7):515–8.
29. Bingham J, Chauhan SP, Hayes E, Gherman R, Lewis D. Recurrent shoulder dystocia: a review. Obstet Gynecol Surv. 2010;65(3):183–8.
30. McFarland MB, Langer O, Piper JM, et al. Perinatal outcome and the type and number of maneuvers in shoulder dystocia. Int J Gynaecol Obstet. 1996;55:219–24.
31. American College of Obstetricians and Gynecologists. Neonatal brachial plexus palsy, American College of Obstetricians and Gynecologists, Task force on neonatal brachial plexus palsy. 2014.
32. Torki M, et al. Severe brachial plexus palsy in women without shoulder dystocia. Obstet Gynecol. 2012;120:539–41.
33. Doumouchtsis SK, Arulkumaran S. All brachial plexus injuries caused by shoulder dystocia? Obstet Gynecol Surv. 2009;64(9):615–23.
34. Benjamin K. Part 1. Injuries to brachial plexus: mechanism of injury and identification of risk factors. Adv Neonatal Care. 2005;5(4):181–9.
35. Horvath K, Koch K, Jeitler K, Matyas E, Bender R, Bastian H, et al. Effects of treatment in women with gestational diabetes mellitus: systematic review and meta-analysis. BMJ. 2010;340:c1395.
36. National Institute for Health and Clinical Excellence. Diabetes in pregnancy. Management of diabetes and its complications from pre-conception to the postnatal period. Clinical guideline 63. London: NICE; 2008.
37. Focus Group Shoulder Dystocia. Confidential enquiries into stillbirths and deaths in infancy. Fifth annual report. London: Maternal and Child Health Research Consortium; 1998. p. 73–9.
38. Gobbo R, Baxley EG. Shoulder dystocia. In: ALSO: advanced life support in obstetrics provider course syllabus. Leawood, KS: American Academy of Family Physicians; 2000.
39. Woods CE. A principle of physics as applicable to shoulder delivery. Am J Obstet Gynecol. 1943;45:796–805.
40. Sandberg EC. Zavanelli maneuver: twelve years of recorded experience. Obstet Gynecol. 1999;93:312–7.

41. Vaithilingam N, Davies D. Cephalic replacement for shoulder dystocia: three cases. Br J Obstet Gynecol. 2005;112:674–5.
42. O'Shaughnessy MJ. Hysterotomy facilitation of the vaginal delivery of the posterior arm in a case of severe shoulder dystocia. Obstet Gynecol. 1998;92:693–5.
43. Royal College of Obstetricians and Gynaecologists. RCOG guideline no. 42. 2nd ed. March 2012.
44. Cluver CA, Hofmeyr GJ. Posterior axilla sling traction for shoulder dystocia: case review and a new method of shoulder rotation with the sling. Am J Obstet Gynecol. 2015;212:784.e1–7.
45. Royal College of Obstetricians and Gynaecologists, Royal College of Midwives. Towards safer childbirth. Minimum standards for the organisation of labor wards: report of a joint working party. London: RCOG Press; 1999.
46. Fahey JO, Mighty HE. Shoulder dystocia: using simulation to train providers and teams. J Perinat Neonatal Nurs. 2008;22(2):114–22; quiz 123–4.
47. Deering S, Poggi S, Macedonia C, Gherman R, Satin AJ. Improving resident competency in the management of shoulder dystocia with simulation training. Obstet Gynecol. 2004;103(6):1224–8.
48. Draycott TJ, Crofts JF, Ash JP, et al. Improving neonatal outcome through practical dystocia training. Obstet Gynecol. 2008;112(1):14–20.

Postpartum Hemorrhage

36

Sheela V. Mane, Vijay Kumar Koravi,
Priyanka Dilip Kumar, and Meenakshi Kandoria

36.1 Introduction

> An error doesn't become a mistake until you refuse to learn from it. —Orlando Battista

Hemorrhage, even to this day, is the major killer of pregnant women both in developing and developed countries. Postpartum hemorrhage (PPH) is a dreaded complication of labor and delivery and presents itself not only in women at high risk for the complication but also sometimes in apparently low-risk gravida in the most innocuous and unexpected situations. *Global statistics for causes of maternal mortality state that maternal anemia directly or indirectly is responsible in 19.3% of cases*, hemorrhage in 23.7%, pregnancy-induced hypertension (PIH) and eclampsia in 13.1%, unsafe abortions in 12.6%, sepsis in 10.6%, obstructed labor in 6.4%, and others in 14.2%. It is obvious that hemorrhage remains the major killer, and healthcare workers need to be alerted and trained to handle this emergency [1].

It is important to understand "why bleeding occurs." The rule of four Ts (tone, tissue, trauma, thrombin) will quickly take us to the cause. Atony of the uterus is responsible for 75–90% of the cases of PPH. Trauma to the uterus, cervix, vagina and perineum, and tissue (retained placenta and membranes) and coagulopathy will account for the rest.

Though contribution of PPH as a cause of maternal death is 25% globally. In India, PPH contributes to 38% of all maternal deaths, which is considerably high given the Indian Maternal Mortality Ratio (*MMR*) of *212/100,000*, and not merely suboptimal obstetric care was responsible. The newer trend in obstetric morbidity due to rise in cesarean section and comorbidities like obesity, diabetes, human immunodeficiency virus (HIV) infections, and drug abuse is adding to the problems [2].

There is evidence showing that comprehensive emergency obstetric care is responsible for 33% reduction of maternal death.

Research by the Prevention of Maternal Mortality Project has indicated attention to three delays in the Emergency Obstetric Care (EmOC).

S. V. Mane (✉) · P. D. Kumar
Department of OBG, Anugraha Nursing Home, Bangalore, Karnataka, India

V. K. Koravi
Department of OBG, Dr. B.R. Ambedkar Medical College, Bangalore, Karnataka, India

M. Kandoria
Department of OBG, Kamala Nehru State Hospital for Mother and Child, Indira Gandhi Medical College, Shimla, Himachal Pradesh, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_36

Delay in:

- Decision to seek EmOC
- Reaching healthcare facility

- Obtaining treatment at healthcare facility

The *third delay is detrimental and not uncommon.* Hard fact about mortality from PPH is that it is different from other major obstetric causes of death. If not managed, PPH can cause death in 8–12 h, obstructed labor in 2 days, and sepsis in 6 days, but with severe PPH, if not promptly managed, a woman can die in just 2 h after delivery [2].

36.2 Classification and Definitions

PPH can be primary or secondary, with primary hemorrhage occurring within the first 24 h of delivery and secondary between 24 h and 6–12 weeks postpartum. Primary PPH occurs in 4–6% of pregnancies and is caused by uterine atony in greater than 80% cases.

The *classical definition* of PPH is blood loss more than 500 mL within 24 h after vaginal delivery or more than 1000 mL after cesarean section. Significant blood loss can be well tolerated by most young healthy women, and an uncomplicated delivery often results in blood loss more than 500 mL without any compromise to the mother's condition [3].

A decline in hematocrit of greater than 10% has also been used to define PPH. This hematocrit value provides an objective laboratory measure. However, this may not reflect the current hematologic status in acute situations since it can take hours for losses to create laboratory value changes. Hypotension, dizziness, pallor, and oliguria occur when blood loss is substantial.

Any bleeding that results in hemodynamic instability, if left untreated, should be considered PPH and managed accordingly.

In severe PPH if there is a blood loss of more than 1500 mL, decline in hemoglobin of more than 4 g/dL, acute transfusion of at least 4 units of red blood cells (RBCs), or surgical/nonsurgical hemostatic intervention (angiographic embolization, surgical arterial ligation, or hysterectomy), will need to be decided soon.

Definition of Major PPH:

- Blood loss of greater than 150 mL/min (within 20 min causing loss of >50% of blood volume).
- Sudden blood loss of greater than 1500–2000 mL (loss of 25–35% of blood volume).
- In major PPH if there is blood loss of more than 2500 mL, transfusion of more than 5 units of packed red blood cells (PRBC) or treatment for ensuing coagulopathy will have to be done without delay [4].

36.2.1 Massive Blood Loss

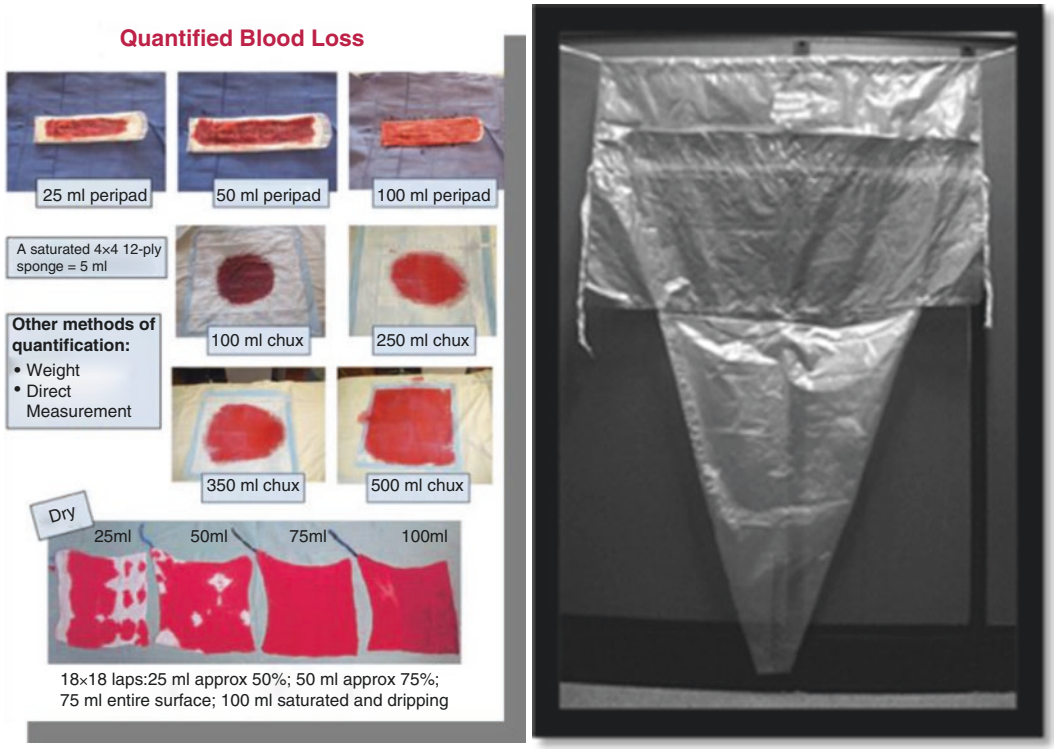
This is defined as the loss of one blood volume in a 24-h period or transfusion of more than ten units of blood within a 24-h period. The rate of blood loss is an important factor. A practical definition is the loss of 50% of blood volume within a 3-h period or loss at a rate of 150 mL/min.

Circulating volume at term is approximately 5 L. For an average built woman between 50 and 70 kg, massive hemorrhage has occurred when the loss is estimated at 1.5–2.0 L.

This situation could be seen within 10–15 min of delivery; appropriate action (according to protocol) must be initiated to restore circulating blood volume and tissue oxygenation while awaiting blood products and laboratory test results to guide the replacement therapy [5].

36.2.1.1 Methods to Estimate Blood Loss

Underestimation of blood loss following delivery can be avoided if all shed blood is measured and sponges, wraps, swabs, etc. are carefully weighed.



- Visual estimation: This common method underestimates PPH by 30–50%. This inaccuracy increases as blood loss increases and delays diagnosis and timely action.
- Use of the blood collection drape: Funnel-shaped plastic bag hangs from the edge of the delivery table and is calibrated with two lines, alert line of yellow color at the 350 mL mark and a red action line at the 500 mL mark.
- Collection of blood in a kidney tray or in a calibrated container.
- Clotted blood volume represents half of the blood volume required to form the clots.

36.2.1.2 Initial Response Management

The *golden hour* refers to the first 60 min from the time of recognition of PPH. It is the time in which resuscitation must begin to achieve maximum survival. As more delay is there to start the resuscitation, the percentage of surviving patients decreases. Too little, too late is the cause of mortality: too little IV fluids, blood, and clotting factors and too late resuscitation, blood replacement, and surgical measures to arrest bleeding [6].

The approach should be through a staged process:

- Call for and mobilize help.
- Communicate the problem to the woman, family members present, and multidisciplinary team members with timely escalation to senior obstetric and midwifery staff and involvement of hematologist, blood bank, and anesthetists. The lead clinician should delegate tasks during maternity emergency.
- Significant blood loss due to any cause requires standard maternal resuscitation measures.

1. Airway, breathing, circulation.
2. Two large-bore intravenous (IV) access—Gray (No. 16) and green (No. 18); IV set, blood set, and three-way stopcock.
3. Oxygen by face mask at the rate of 6–8 L/min.
4. Infusion of crystalloids (normal saline (NS)/Ringer's lactate (RL)) three times in volume to the blood lost.
5. Reserve/get two units of PRBC, depending on the extent of hemorrhage [7, 8].

36.2.2 Stage 1

Stage 1 is initiated when the estimated blood loss (EBL) is more than 500 mL after vaginal delivery or more than 1000 mL after cesarean section, or vital signs show more than 15% change from baseline (heart rate >100 bpm, blood pressure 85/45 mmHg, O_2 saturation <95%).

Successful management of PPH requires that both components should be simultaneously and systematically addressed. Resuscitative measures, diagnosis, and treatment of the underlying cause must occur quickly before sequelae of severe hypovolemia develop.

36.2.2.1 Resuscitation

The goal of initial resuscitation is to achieve sufficient circulating blood volume to enable transfer of the patient to a site where effective treatment can occur. The degree of initial volume resuscitation will depend on the level of care that can be offered at the facility. If necessary, the patient must be accompanied by an experienced member of staff to a higher level of care.

Initial resuscitation is based on the airway, breathing, circulation (ABC) approach with advanced resuscitation guided by the clinical situation.

36.2.2.2 Assessment

Monitor the conscious state and vital signs at regular intervals, oxygen saturation (by pulse oximeter), uterine tone, and urine output. Maintain record of EBL. It is important to recognize the clinical signs of varying degrees of blood loss.

Emergency response measures should be initiated and steps taken to assure fluid resuscitation and core perfusion maintained via lower extremity elevation and in some cases anti-shock compression wrap.

A designated "PPH box" is a good risk management approach as all necessary equipment is quickly available in it.

Administer oxygen via mask at 10 L/min; keep saturation more than 95%. If patient is not breathing, use assisted ventilation. Intubate the deeply unconscious.

- If no pulse, start cardiopulmonary resuscitation (CPR).
- Position the woman to maximize venous return. Lower head of bed. Raising the legs improves venous return and is consistent with the positioning used to diagnose and treat the underlying causes of bleeding.
- Intravenous access—insert at least two (14 or 16 gauge) cannulae.
- Full blood count (FBC), clotting screen [fibrinogen, activated partial thromboplastin time (APTT), prothrombin time (PT), D-dimer], collect blood for crossmatch.
- Insert indwelling urinary catheter—urine output monitored at 15-min intervals.
- Avoid hypotension by adequate fluid replacement in relation to ongoing measured blood loss.
- Avoid hypothermia, as this increases the risk of disseminated intravascular coagulation (DIC). Prewarm resuscitation fluids and use warm air blankets.
- Commence bimanual massage. Bimanual massage results in a decrease in bleeding even if the uterus remains relatively atonic, thus allowing resuscitation a chance to begin to catch up with blood loss.
- Documentation: Scribe assessments and response to management on the observation chart.

If the bleeding is rapid or woman is hemodynamically unstable:

- Delegate two persons to continue with resuscitative measures.
- If unsuccessful, perform aortocaval compression.

36.2.2.3 Identify Cause and Stop the Bleeding

- Continue uterine massage to stimulate a contraction and expel any clots present.
- If bleeding continues despite a well-contracted uterus, look for other causes (e.g., incomplete placenta, cervicovaginal tears, and hematomas). Use the four Ts mnemonic:
  - Tone (uterine), 70%
  - Trauma (uterine rupture/cervical or vaginal lacerations), 20%
  - Tissue (retained placental tissue), 10%
  - Thrombin (bleeding disorder), 1%

Bedside clotting test: Take 2 mL of venous blood into a plain glass test tube. Hold the tube in closed fist to keep it warm (+37 °C). After 4 min, tip the tube slowly to see if a clot is forming. Tip it again every minute until the blood clots and the tube can be turned upside down. If a clot does not form after 7 min or a soft clot forms that breaks down easily, it is suggestive of a clotting disorder [9].

36.2.2.4 Fluid Resuscitation

Aggressively restore circulating fluid volume and thereby perfusion to vital structures. Volume replacement is guided by the patient's response to initial therapy, not by the initial classification category.

- Perform initial resuscitation with large volumes of crystalloid solution: normal saline (NS) 0.9%, compound sodium lactate solution (Hartmann's solution), or lactated Ringer's solution as rapid fluid bolus, 2 L over 10 min:
  - To resuscitate more quickly, administer IV fluids using a pressure infusion device/blood pressure (BP) cuff.
  - When using crystalloid, the ratio of resuscitative IV fluid required to blood lost is 3:1 because most of the infused fluid shifts from intravascular space to interstitial space. This shift, along with oxytocin use, may result in peripheral edema in the days following PPH.
  - If large amounts (>10 L) of crystalloids are being infused, Ringer's lactate (RL) is preferred over NS.
  - Dextrose-containing solutions (5% dextrose in water or dextrose normal saline [DNS]) have no role in the management of PPH.
  - PPH of up to 1500 mL in a healthy woman can usually be managed by crystalloid infusion alone if the cause of bleeding is arrested. Blood loss in excess of this usually requires the addition of a PRBC transfusion.
- Colloids (Haemaccel, Gelofusine, albumin, dextran, hydroxyethyl starch) are largely retained within the intravascular space. Large volumes of colloids (>1000–1500 mL/days) can have an adverse effect on hemostasis. No colloid solution has been demonstrated to be superior to NS. Colloids and blood can replace volume lost on one-to-one basis, whereas 3 volumes of crystalloids are required to replace 1 volume of blood lost [10].
- Administer uterotonic agents; a stepwise progression of medical therapy with uterotonics is undertaken.
- Commence vigorous massage and therapeutic oxytocin as a 5 U IV bolus, as 20 U in 1 L of NS IV runs as fast as possible, or as 10 U intramyometrially with a spinal needle if no immediate IV access is available. Prepare and commence an oxytocin infusion, 40 U oxytocin in 1000 mL Hartmann's solution or NS 0.9%.
- Repeat intramuscular (IM) bolus oxytocin 10 U. Alternatively, repeat bolus of ergometrine 250 mcg IM. The traditional second-line agent has been ergometrine.
- If there is no response to oxytocin infusion and no contraindication to ergometrine use, repeat ergometrine 25–50 mcg IV or 250 mcg IM after 2–3 min.
- Many use IM carboprost as a second-line agent. It is 80–90% effective in stopping PPH in cases refractory to oxytocin and ergometrine.
- Consider misoprostol 800 mcg per rectum. It remains the third-line agent.

Carbetocin is a long-acting synthetic oxytocin agonist that binds to smooth muscle receptors of the uterus and causes rhythmic contractions and increases the frequency of contractions and uterine tone. It should not be used as a first-line agent. The toxicity spectrum is similar to that of oxytocin. Dosage is 100 mcg slow IV injection [11–13].

Prostaglandins enhance uterine contractility and cause vasoconstriction. They control PPH in 86% when used alone and in 95% when combined with oxytocin. In cases where it is not effective, chorioamnionitis or other risk factors for hemorrhage are often present. Prostaglandins given orally, sublingually, or per rectum are less effective than oxytocin or ergometrine given parenterally.

The use of any uterotonic agent has precautions and contraindications especially when combining these agents; the risks of their use must be weighed against the risks of intractable or uncontrolled hemorrhage.

Reassess at 10 min. For bleeding unresponsive to oxytocics, check for unrecognized lacerations and uterine rupture.

- If systolic blood pressure (SBP) is less than 100 mmHg and pulse is more than 110, give colloid 500 mL, repeat twice if necessary, or 1.5 L crystalloid if no colloid available.
- If SBP is less than 100, pulse is more than 110, and 3.5 L of clear fluids have been given, notify blood bank, and start blood transfusion.
- Concerns of concealed hemorrhage and uterine overdistension have made the use of uterine packing very rare. Consider using balloon tamponade with a Bakri balloon, Foley catheter, or Sengstaken-Blakemore tube and condom tamponade which is useful in uterine atony and placenta accreta. After 12 h, the balloon can be gradually deflated, and the catheter may be removed in 24 h if there is no bleeding.
- Explore the uterine cavity manually for retained placental fragments or lacerations (ideally under anesthesia). Uterine rupture needs repair of laceration/hysterectomy.
- Refractory uterine atony may need hysterectomy or internal iliac artery ligation.
- Angiographic embolization for intractable puerperal hematomas can be used primarily when hemostasis is not obtained by surgical methods [14].

36.2.2.5 Blood Transfusion

Blood and blood product transfusion may be required if:

- Blood loss is continuing.
- Blood volume lost is more than 30%, add rule of 30.
- Patient's clinical status reflects developing shock despite aggressive resuscitation.

Be aware of the capabilities of blood bank regarding timing, type, and amount of blood products available. Good communication with the blood transfusion service is essential. PRBCs are initially used with other blood components given if indicated. Uncrossmatched group—specific blood or O-negative blood—may be used until fully crossmatched blood becomes available.

- For catastrophic bleeding, rapidly transfuse 2–4 U of whole blood to replace lost oxygen carrying capacity and to restore circulating volume. If using PRBCs which are very viscous, the reduced infusion rate can be overcome by adding 100 mL of NS to each unit. Do not use RL because the calcium contained in the solution may cause clotting:
  - Two units of O-negative blood should be transfused if crossmatched blood is unavailable.
  - Crossmatched or type-specific blood should be used as soon as it is available.
- Consider thawing two units fresh frozen plasma (FFP). One liter of FFP should be given (15 mL/kg) with every 6 U of blood transfused.
- Cryoprecipitate (more concentrated form of fibrinogen and other clotting factors (VIII, XIII, von Willebrand factor)) is used if there is DIC or if fibrinogen is less than 1 g/L.
- Use a blood warmer if the infusion rate (>100 mL/min) or the total volume infused is high.
- Check laboratory every 30–60 min when transfusion is initiated.
- Thrombocytopenia is likely after 1.5–2 times the blood volume has been replaced. Use platelet transfusion to keep the platelet count more than 50×10^9/L or more than $80–100 \times 10^9$/L if surgical intervention is necessary:

  - Each unit of platelets increases the count by 10×10^9/L. These are usually given in packs of 5–6 U.
  - If bleeding is continuing and platelet count is less than 50×10^9/L, administer 10–12 U initially.
  - Platelet preparations contain some RBCs; administration of anti-D immunoglobulin is recommended for Rh-negative women once the crisis has passed [15].
- Monitor electrolyte and acid-base status:
  - Hypocalcemia due to citrate intoxication is rarely seen.
  - Hyperkalemia and acidosis due to the use of stored blood are theoretical risks and unimportant if perfusion of vital organs is maintained.
- How urgent is the blood chart?
- Other risks of transfusion include infection, transfusion reaction, and development of atypical antibodies.
- Intraoperative blood salvage use has been limited by theoretical concerns about amniotic fluid embolism and infection. It may be life-saving in remote regions with limited blood banking services.
- Patients may refuse transfusion of blood products based on religious/other grounds, though refusal may not extend to all related products. Autotransfusion can be considered. Human erythropoietin and activated factor VIIIa are usually acceptable.

36.2.2.6 Coagulopathy

If coagulation test results are abnormal from the onset of PPH, strongly consider and treat an underlying cause (e.g., abruptio placentae, HELLP syndrome, fatty liver of pregnancy, intrauterine fetal demise, amniotic fluid embolus, septicemia, and pre-existing disorder). Seek the advice of a hematologist in case of massive transfusion or coagulopathy.

Dilutional coagulopathy occurs when 80% of the original blood volume has been replaced. Infuse 4 U FFP followed by additional units to normalize the coagulation findings. Many recommend addition of 1 U of FFP for every 5 U of PRBCs.

DIC may develop if shock has led to marked hypoperfusion of tissues, causing damage and release of tissue thromboplastins. Here, D-dimer levels are raised and fibrinogen levels are very low with a prolonged PT.

- Cryoprecipitate 6–12 U along with FFP is useful because of the grossly reduced fibrinogen levels. Cryoprecipitate provides fibrinogen and is faster to prepare in the blood bank. It is helpful immediately before any surgical intervention in patients with abnormal coagulation test results.
- Recombinant activated factor (rFVIIa) has been useful in severe PPH complicated by DIC [16, 17].
- The use of heparin and antifibrinolytic therapy is not recommended in women with DIC of obstetric origin.

36.2.2.7 Other Hemostatic Agents

- Tranexamic acid (antifibrinolytic), 1 g, slow IV significantly decreases bleeding, but is not effective in major hemorrhage [18].
- Desmopressin (DDAVP): Its activity is based on increasing the level of coagulation factors VIII and von Willebrand and direct activation of platelets.
- Recombinant activated factor VIIa. Its use has the potential to increase thrombotic events and is only recommended:
  - After all conventional therapies have failed (10–12 U of PRBCs, 6–9 U of FFP, 2–3 U of platelets).
  - In the setting of DIC.
  - In patients refusing blood transfusion.
  - Dose: 30–90 mcg/kg.
    - Repeated 20–30 min if less than 90 mcg/kg was used, and there is no clinical response.
    - If there is a response noted but with continued coagulopathy, additional dose is given in 2–3 h.
  - Platelet count should be greater than 50,000, as well as normalizing any acidosis and hypothermia.
  - Conventional modalities should be continued along with this treatment [19].

36.2.2.8 Endpoints of Initial Resuscitation

If blood loss has stopped, the endpoints are to normalize BP and to reduce heart rate to less than 110 beats/min. At the same time, recovery to conscious level, breathing, and peripheral perfusion should also occur. These goals represent equivalent replacement of the initial volume lost and should have been achieved within half to 1 h.

- Although urine output is a fairly useful guide to assessment of severity of shock, it should not be used as an endpoint to initial resuscitation as this may lead to excessive volume administration and pulmonary edema. Renal function is often slow to return, and the initial insult may have caused acute renal failure.
- Hemoglobin and hematocrit are of little/no value in the initial resuscitation of acute hemorrhage.
- Central venous cannulation is of little value in the initial stages unless peripheral venous access proves impossible.
- Oxygen therapy should be continued even if the patient is breathing normally, and saturation should be monitored continuously (pulse oximetry).
- Active management of body temperature should be maintained throughout. Use warm fluids and a forced air warmer or warmed blankets.

Failure to achieve a response to resuscitation implies continued bleeding, which must be identified and treated while resuscitation continues. The status of the patient, severity of bleeding, and response to initial management steps determine if and when the protocol for massive obstetric hemorrhage is instituted. Consultation with appropriate specialists and placement in an intensive care setting are preferred.

36.2.3 Stage 2

If there is ongoing massive blood loss, bleeding reaches more than 1500 mL or unstable vital signs:

- Notify blood bank and request thawing of FFP, platelets, and cryoprecipitate. Ensure massive transfusion protocol is initiated. Utilize fluid warmer and rapidly transfuse:
  - 4 U PRBCs.
  - 4 U FFP.
  - 1 U platelets.
  - Blood is administered as component therapy, with a ratio of 1 PRBC:1 FFP/freeze-dried plasma (FDP) after the first 2 PRBCs.
  - If initial dose of transfusions do not correct the deficit or laboratory values, double the products until corrected. Ask blood bank to stay 4 U ahead during this event.
  - Up to 1 L of FFP and 10 units of cryoprecipitate (2 packs) may be given empirically while awaiting the results of coagulation studies.
- Notify the Intensive Care Unit (ICU) of potential admission for close surveillance. Pay close attention to the level of consciousness, pulse, BP, and urine output. Pulse oximetry is useful for evaluating tissue perfusion and oxygen saturation. Arterial line placement aids in monitoring BP and allows easy access for blood work. Central venous pressure (CVP) line placement is helpful for patients in critical condition with ongoing bleeding. As this is a poor indicator of blood volume, it should never be used alone to guide volume replacement. Frequent auscultation of the lung fields helps detect pulmonary edema or the development of adult respiratory distress syndrome.
- Closely monitor the complete blood count (CBC), coagulation (PT, APTT, fibrinogen, D-dimer), arterial blood gas (ABG), and acid-base status every 15–30 min:
  - Hemoglobin (Hb) is invariably overestimated during resuscitation of ongoing hemorrhage. Maintain Hb greater than 8 g/dL.
  - ABG: Acidosis is associated with inadequate perfusion. Rising lactate level indicates inadequate tissue perfusion.
  - Platelet count and coagulation parameter abnormalities are common. If blood loss

continues following surgical correction, give platelet concentrate if count is less than 50×10^9/L and correct abnormal coagulation (APTT and thrombin time, PT).
  - Urea and electrolytes: Electrolyte studies are not usually helpful during acute resuscitation. Administration of large volumes of blood can cause hyperkalemia or hypocalcemia that need correction. Acute renal failure is best monitored using urine output (>30 mL/h indicates adequate renal perfusion). Urea and creatinine do not increase significantly until later.
- Consent patient for possible laparotomy/hysterectomy/interventional radiology.
- Ensure the availability of an operating room. Initiate B-Lynch suture, uterine artery ligation, and intrauterine packing/balloon if not attempted yet. Consider hysterectomy if options exhausted or fertility is not desired [20–22].
- Alert interventional radiology (IR) to prepare for possible embolization.
- Inotropes: If patient remains shocked despite adequate volume resuscitation (no further bleeding, CVP >10 cm, mean arterial pressure <65 mmHg), then inotropic support is appropriate).
- Terminate the protocol once vital signs and labs are stable.
- Continue transfusions until patient's vitals are stable and labs reach minimal values.

The following is a plan for managing massive obstetric hemorrhage adapted from Bonner. The word "order" is a useful mnemonic.

- Organization
  - Call experienced staff.
  - Alert the blood bank, ICU, IR and operating theater.
  - Record vital signs, urine output, and fluids, and drugs administered.
- Resuscitation
  - Administer oxygen by mask.
  - Place two large-bore (14-guage) IV lines.
  - Take blood for crossmatch of 6 U PRBCs, CBC, coagulation screen, urea, creatinine, and electrolytes.
  - Begin immediate rapid fluid replacement with NS/RL.
  - Transfuse PRBCs.
- Defective blood coagulation
  - Order coagulation screen (APTT) if fibrinogen, PT, blood film, and D-dimer are abnormal.
  - Give FFP if coagulation tests are abnormal and sites are oozing.
  - Give cryoprecipitate if abnormal coagulation tests are not corrected with FFP and bleeding continues.
  - Give platelet concentrates if the platelet count is less than 50×10^9/L and bleeding continues.
  - Before surgical intervention, use cryoprecipitate and platelet concentrates.
- Evaluation of response
  - Monitor pulse, BP, ABG, and acid-base status, and consider monitoring CVP.
  - Measure urine output using an indwelling catheter.
  - Order regular CBC and coagulation tests to guide blood component therapy.
- Remedy the cause of bleeding
  - If postpartum, use oxytocin, prostaglandin, or ergonovine.
  - Explore and empty the uterine cavity and consider uterine packing [23].
  - Examine the cervix and vagina, ligate any bleeding vessels, and repair trauma.
  - Ligate the uterine vessels (uterine and/or internal iliac arteries) [24–27].
  - Consider arterial embolization/hysterectomy [28–31].

36.2.3.1 Transporting a Woman Who Is Bleeding

- Prepare for transfer when blood loss exceeds more than 350 mL in the first hour or more than 500 mL within 2 h of delivery.
- Send a skilled provider with the woman to ensure an open airway, to deliver first aid if the woman goes into shock, and to explain the care provided.

- Elevate legs to improve blood supply to vital organs.
- Keep the woman warm.
- Continue uterine massage during transport.
- Provide bimanual uterine compression (external if possible and internal if necessary).
- Ensure that referral facility knows what uterotonics the woman has been given and when.

36.2.3.2 The Non-pneumatic Anti-shock Garment (NASG)

NASG is used as a lifesaving first-aid device in uncontrollable PPH. It controls bleeding, reverses shock, and stabilizes the patient's condition to enable safe transport to a center, which is equipped with comprehensive obstetric care facilities [32, 33].

How Does NASG Work?

In hemorrhagic shock due to PPH, the brain, heart, and lungs are deprived of oxygen because the blood accumulates in the lower abdomen and legs as a result of blood loss.

Correct application of NASG delivers a pressure of 20–40 mmHg to the lower body. The skin tight suit squeezes the blood back into circulation in order to supply oxygenated blood to the heart, brain, and lungs. The patient remains conscious, vital signs are stabilized, and organ damage is avoided so that she can be safely transported. Direct pressure on the fundus of the uterus reduces blood supply to the uterus, thereby reducing bleeding. In short, NASG acts like "autotransfusion."

Advantages of NASG

The experts who pioneered the device are of the opinion that it can reduce death at childbirth by over 50%. According to Prof. Miller, it is a very potent first-aid device, which could have a huge impact on reducing the maternal morbidity and mortality [22].

The WHO recommends NASG in low resource settings, especially because it is cost-effective and reusable. It has been used successfully in over 6000 women in 6 countries. In the UK and the USA, it is being used in remote rural areas and in Jehovah's Witnesses women who decline blood transfusion. In African countries such as Zambia and Zimbabwe, it is being used in urban centers as well. The Tamil Nadu Government has made NASG available at all health centers and also in ambulances.

The method of application of NASG, cleaning, storing, and its use in special situations are demonstrated in the YouTube video titled, "Saving Mothers' Lives: An NASG Training Video."

References

1. Koblinsky M, Campbell O, Heichelheim J. Organising delivery care. What works for safe motherhood. Bull World Health Organ. 1999;77:399–406.
2. Combs CA, Murphy EL, Laros RK Jr. Factors associated with postpartum hemorrhage with vaginal birth. Obstet Gynecol. 1991;77:69–76.
3. Bonnar J. Massive obstetric haemorrhage. Bailliers Best Pract Res Clin Obstet Gynaecol. 2000;14:1–18.
4. Yiadom MY, Carusi D. Postpartum haemorrhage in emergency medicine [online]. Available from: http://emedicine.medscape.com/article/796785-overview#showall. Accessed Mar 2013.
5. Waterstone M, Bewley S, Wolfe C. Incidence and predictors of severe obstetric morbidity: case control study. Br Med J. 2001;322:1089–94.
6. Charbit B, Mandelbrot L, Samain E, et al. The decrease of fibrinogen is an early predictor of the severity of post partum haemorrhage. J Thromb Haemost. 2007;5:266–73.
7. Sobieszczyk S, Bręborowicz GH. Management recommendations for post partum hemorrhage. Arch Perinat Med. 2004;m10:53–6.
8. NHS Quality Improvement Scotland. Scottish confidential audit of severe maternal morbidity. 5th Annual report 2007 [online]. Available from: http://s3.amazonaws.com/zanran_storage/www.nhsquality.org/Contentpages/654122215.pdf. Accessed Mar 2013.
9. Arulkumaran S. Massive and intractable PPH, lessons from confidential inquiries. Available from: http://www.sicog2013.com/Portals/Management.
10. Prevention and management of postpartum hemorrhage, RCOG green-top guideline no. 52, May 2009.
11. Lynch CB, Keith LG, Lalond AB, Karoshi M, editors. A textbook of postpartum hemorrhage: a comprehensive guide to evaluation, management and surgical intervention. Dumfriesshire: Sapiens Publishing; 2006.
12. Boucher M, Nimrod CA, Tawagi GF, Meeker TA, Rennicks White RE, Varin J. Comparison of carbetocin and oxytocin for the prevention of postpartum hemorrhage following vaginal delivery: a double-blind randomized trial. J Obstet Gynaecol Can. 2004;26:481–8.
13. Su LL, Chong YS, Samuel M. Oxytocin agonists for preventing postpartum haemorrhage. Cochrane Database Syst Rev. 2007;3:CD005457.
14. Oleen MA, Mariano JP. Controlling refractory atonic postpartum haemorrhage with Hemabate sterile solution. Am J Obstet Gynecol. 1990;162:205–8.
15. Ramanathan G, Arulkumaran S. Postpartum hemorrhage. J Obstet Gynaecol Can. 2006;28:967–73.
16. Atoyebi W, Mundy N, Croxton T, Littlewood TJ, Murphy MF. Is it necessary to administer anti-D to prevent RhD immunization after the transfusion of RhD-positive platelet concentrates? Br J Haematol. 2000;111:980–3.
17. Hewitt PE, Machin SJ. Massive blood transfusion. In: ABC of transfusion. London: BMJ Publishing; 1998. p. 49–52.
18. Franchini M, Manzato F, Salvagno GL, Lippi G. Potential role of recombinant activated factor VII for the treatment of severe bleeding associated with disseminated intravascular coagulation: a systematic review. Blood Coagul Fibrinolysis. 2007;18:589–93.
19. As KA, Hagen P, Webb JB. Tranexamic acid in the management of postpartum haemorrhage. Br J Obstet Gynaecol. 1996;103:1250–1.
20. Boehlen F, Morales MA, Fontana P, Ricou B, Irion O, de Moerloose P. Prolonged treatment of massive postpartum haemorrhage with recombinant factor VIIa: case report and review of the literature. BJOG. 2004;111:284–7.
21. B-Lynch C, Coker A, Lawal AH, Abu J, Cowen MJ. The B-Lynch surgical technique for the control of massive postpartum haemorrhage: an alternative to hysterectomy? Five cases reported. Br J Obstet Gynaecol. 1997;104(3):372–5.
22. Soumunkiran A, Ozdemir I, Demiraran Y, Yucel O. B-Lynch suture after the failure of hypogastric artery ligation to control post-partum hemorrhage due to placenta increta in a patient with the factor V Leiden mutation. J Obstet Gynaecol Res. 2007;33(4):557.
23. El-Hamamy E, B-Lynch C. A worldwide review of the uses of the uterine compression suture techniques as alternative to hysterectomy in the management of severe post-partum haemorrhage. J Obstet Gynaecol. 2005;25(2):143–9.
24. B-Lynch C. Conservative surgical management. In: B-Lynch C, Lalonde AB, editors. A textbook of postpartum haemorrhage. Kolkata: Jaypee; 2006. p. 287–98.
25. Tamizian O, Arulkumaran S. The surgical management of post-partum haemorrhage. Best Pract Res Clin Obstet Gynaecol. 2002;16(1):81–98.
26. Gilstrap LC, Ramin SM. Postpartum Hemorrhage. Clin Obstet Gynecol. 1994;37(4):824–30.
27. AbdRabbo SA. Stepwise uterine devascularization: a novel technique for management of uncontrolled postpartum haemorrhage with preservation of the uterus. Am J Obstet Gynecol. 1994;171(3):694–700.
28. Vegas G, Illescas T, Munoz M, Perez-Pinar A. Selective pelvic arterial embolization in the management of obstetric hemorrhage. Eur J Obstet Gynecol Reprod Biol. 2006;127:68–72.
29. Moore M, Morales JP, Sabharwal T, Oteng-Ntim E, O'Sullivan G. Selective arterial embolisation: a first

line measure for obstetric haemorrhage. Int J Obstet Anesth. 2008;17:70–3.
30. Oei PL, Chua S, Tan L, Ratnam SS, Arulkumaran S. Arterial embolization for bleeding following hysterectomy for intractable post-partum hemorrhage. Int J Gynecol Obstet. 1998;62:83–6.
31. Yamashita Y, Harada M, Yamamoto H, Miyazaki T, Takahashi M, Miyazaki K, Okamura H. Transcatheter arterial embolization of obstetric & gynaecological bleeding: efficacy & clinical outcome. Br J Radiol. 1994;67(798):530–4.
32. Morris J, Meyer C, Fathalla MF, Youssif MM, Al-Hussaini TK, Camlin C, Miller S. Treating uterine atony with the NASG in Egypt. Afr J Midwifery Womens Health. 2011;5(1):37–42.
33. Ojengbede O, Galadanci H, Morhason-Bello IO, Nsima D, Camlin C, Morris J, Butrick E, Meyer C, Mohammed AI, Miller S. The non-pneumatic anti-shock garment for postpartum haemorrhage in Nigeria. Afr J Midwifery Womens Health. 2011;5(3):135–9.

The Retained Placenta

37

Bipin Pandit and Pooja Bandekar

Birth is a mystery. Words are not enough
Marie O'Connor

37.1 Background

The United Nations in 2000 have set Millennium Development Goals, one of which is to bring maternal mortality down by three quarters. To achieve the goal, postpartum haemorrhage (PPH)-related maternal deaths have to go down. Health workers of the low-resource countries need to have adequate relevant medication and to have training in first aid procedures. Most important, there is a need of evidence-based guidelines on the various interventions across the world. This will help to lay the foundation for the strategies, policies and programme development.

37.2 Introduction

Postpartum haemorrhage is an extremely important causative factor of maternal mortality in the developing world. Postpartum haemorrhage associated with retained placenta amounts to almost 0.6–3.3% of normal deliveries [1–3]. Mortality is very low if timely hospital care and blood banks are available. In PPH, the overall case fatality rate is high. The commonest cause of death is haemorrhage, especially when timely manual removal is not available or when approach to hospital is difficult. Hence an immediate and effective medical method of management has a major role in reducing the maternal mortality.

37.3 What Is PPH?

Postpartum haemorrhage is defined as blood loss *more than or equal to* 500 mL within 24 h of birth; severe PPH is blood loss greater than or equal to 1000 mL within 24 h. Postpartum haemorrhage is the commonest cause of maternal mortality. Majority of complications due to PPH occur in the first 24 h postdelivery (primary PPH), whereas any abnormal or excessive bleeding from the birth canal occurring between 24 h and 12 weeks postpartum is regarded as secondary postpartum haemorrhage.

37.4 What Is Retained Placenta?

The placenta retained in utero, with its commonest complication, PPH, is one of the nightmares of delivery events, which no ante-natal care can predict or even prevent [4, 5]. There are conditions, first, in which the placenta remains in the uterus, retained placenta, or second, an adherent placenta.

B. Pandit
R N Cooper Hospital, Mumbai, Maharashtra, India

P. Bandekar (✉)
Seth G.S. Medical College & Nowrosjee Wadia Maternity Hospital, Mumbai, Maharashtra, India

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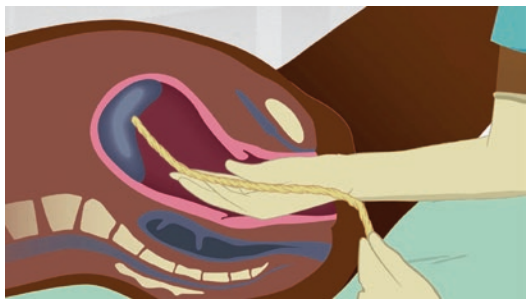
In retained placenta, the placenta may separate partially or completely, but due to an associated secondary complication (usually mechanical), it fails to expel from the uterus, the most common cause being uterine inertia.



37.4.1 Pathophysiology

The key factor is the retro-placental myometrium.

Brandt in 1993 explained the role of a uterine contraction in the process of detachment of the placenta from the decidual bed [6]. Herman first used ultrasonography to show that a retroplacental myometrial contraction is necessary to generate shearing forces between the placenta and myometrium which finally leads to its separation. He divided the third stage into four phases according to the ultrasound appearances [7].

In the latent phase, immediately after expulsion of the baby, the whole of the myometrium contracts apart from the retro-placental part. In the contraction phase, the retro-placental myometrium contracts leading to the separation of the placenta from the decidua. In the next phase, uterine contraction leads to placental expulsion. Contractions occurring prior to delivery are insufficient to cause placental separation as the foetus prevents the myometrium from achieving the necessary strain for detachment [8]; some of the authors hence omit the contraction stage from the classification [9]. These authors have elaborated the events using Doppler. They say that the blood flow through the arcuate and radial arteries is decreased during the latent phase and completely cuts off at the onset of the contraction phase [9]. This reduced blood flow is due to the myometrial 'physiological ligature'. Hence maternal blood flow to the placenta stops prior to 'placental detachment and only blood from the intervillous spaces is lost. USG studies also help to understand the causes of the retained placenta. The duration of the 3rd stage of labour depends on the duration of the latent phase and a prolonged 3rd stage is due to failure of contraction in the retro-placental area'. In the retained placenta, there is an overall failure of retro-placental contraction. Doppler studies confirm that the blood flow through the myometrium to the placenta continues irrespective of placenta accreta or prolonged latent phase [9], hence more chances of haemorrhage during manual removal of placenta. This also explains why partial or forced separation of the placenta prior to onset of the contraction phase is associated with high rates of haemorrhage. If the pro-contractile stimuli are strong enough, then labour can be successful despite of persisting, localised placental inhibition. But there will be more chance of retained placenta because of the strong persistent placental inhibition of retro-placental myometrial contractility [10–13].

Many local inhibitors have been identified. Progesterone is an important inhibitor of myometrial contractility in animals; its role in women is under evaluation. The anti-progesterone mifepristone sensitises myometrium to given prostaglandins and hence is used for induction of labour in all trimesters of pregnancy in humans [14–16]. Recent evidence, however, suggests reduction in progesterone metabolites as its mechanism of action [17, 18]. Nitric oxide, a powerful smooth muscle relaxant, is produced in large amount by nitric oxide synthase (NOS) in the placenta [19]. It is rapidly oxidised following its production. Exogenous NO seems to relax myometrium. A

prolonged latent phase is an important cause of retained placenta. This could be either maintained by eliminating the inhibitor (e.g. by treatment with an anti-progesterone) or by stimulation with oxytocics. Umbilical vein oxytocin is a localised stimulus to the retro-placental myometrium [20–22].

37.5 Management of Retained Placenta

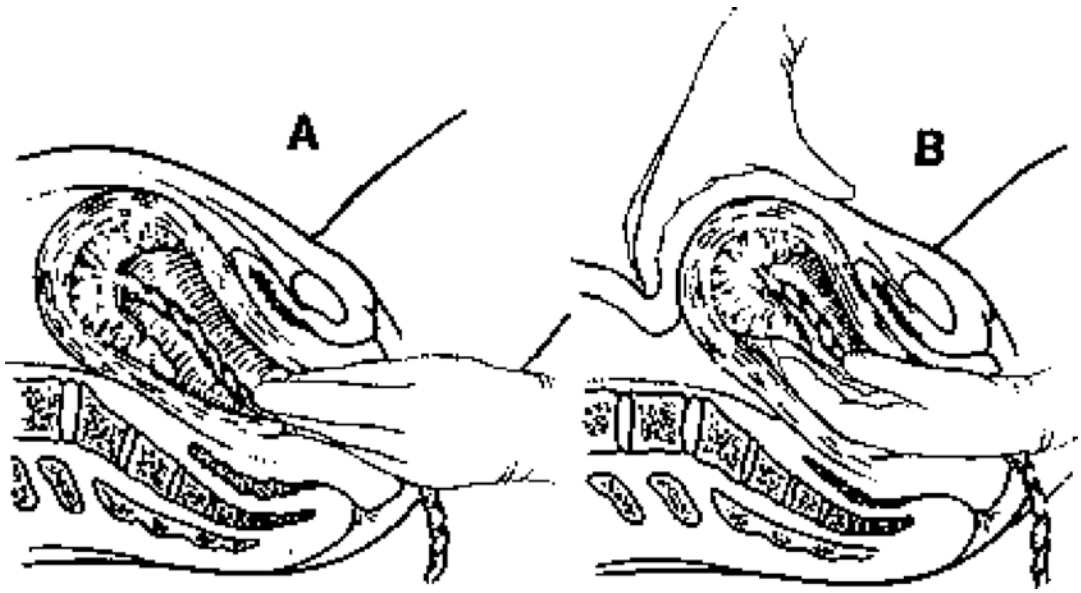
37.5.1 Manual Removal

Presently commonest mode of treatment for a retained placenta is its manual removal under anaesthesia though the female is exposed to risks of anaesthesia as well as infections. Both risk factors are higher in the developing world. The time that is allowed before manual removal may vary; recommended is a delay of ½–1 h in the absence of haemorrhage. There is no increase in haemorrhage till 30 min postpartum [1]; also between 30 and 60 min almost 40% more of placentae will be spontaneously expelled with the loss of an average of just about 0.3 L of blood [23].

There are multiple management options in cases of placenta accreta. Partial manual removal followed by curettage is used to remove maximum possible placenta. The remaining bits of trophoblast is usually reabsorbed spontaneously, but the B-HCG take longer to return to normal [24]. In the placenta percreta, blood continues to flow via the area of invasion even after the maximum part of the placenta is removed due to the lack of the myometrial physiological ligature which would normally cease the flow [9]. In a LSCS, the haemostasis may be achieved through myometrial bed ligatures or through uterine or internal iliac artery ligation [25]. At times, a hysterectomy may be required. If placenta percreta is diagnosed antenatally using ultrasound [9], then it is advisable to opt for conservative treatment. This involves leaving the placenta in situ after delivery. The levels of B-HCG are followed serially and manual removal and curettage performed if indicated [26]. Methotrexate may be beneficial [27].

37.5.1.1 WHO Recommendation

Stat dose of antibiotics (ampicillin or first-generation cephalosporin) post manual removal of the placenta (quality of evidence, very low; strength of recommendation, strong).



37.5.2 Systemic Oxytocics

Their role is controversial. Due to prophylactic oxytocin, most placental expulsion occur at 20 and 40 min, irrespective of the number of placentae that eventually need manual removal [28].

A continuous infusion of 5 IU/h of oxytocin stimulates strong phasic contractions. Ergometrine produces a long continuous contraction for up to 90 min and is the less frequently used drug. However, it is often used in rural areas to buy time, due to its easy availability and also as there is no need of intravenous infusion. E1 analogue, misoprostol, an orally active prostaglandin, has a similar effect for around 90 min [29]. It is cost-effective, resistant to heat and has a good oral availability and hence an excellent drug for rural use.

37.5.2.1 WHO Recommendation

10 IU of oxytocin IM in combination with controlled cord traction can be offered (no formal scientific evidence of benefit or harm; strength of recommendation, weak). Ergometrine is not preferred drug, as it may cause tetanic uterine contractions, which may delay delivery of the placenta (quality of evidence, very low; strength of recommendation, weak). The use of prostaglandin E2 (dinoprostone) is not recommended (quality of evidence, very low; strength of recommendation, strong) [30–32].

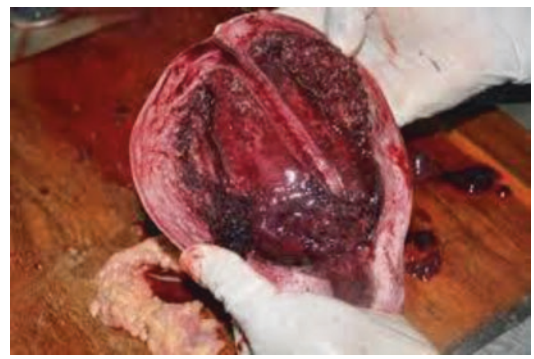
37.5.3 Umbilical Vein Oxytocin Injection

Direct delivery of oxytocin in the retro-placental myometrium is achieved by injecting it into the placental bed through the umbilical vein. The treatment is directed at the area with the contractile failure. Results have been varied. A recent Cochrane review 34 concludes that the use of umbilical infusion of oxytocin is effective in the management of retained placenta, despite the fact that their meta-analysis showed the decrease in retained placenta rates are not significantly different than that obtained with expectant management.

The inconclusive results could be due to insufficient delivery of the oxytocin to the retro-placental myometrium or an inconsistency concerning the dose of oxytocin. There are no comparative studies to assess efficacy of different doses of oxytocin. Hence the choice of dosage of drug is largely empirical. Trials mainly use 10–20 IU oxytocin, although doses of up to 100 IU have been reported [33]. The trials that have used higher doses have found more success rates. A dose of as little as 5 IU when given to the mother IV can produce significant changes in maternal BP [34]. Although oxytocin can clearly pass via the placenta, the data is unclear as to how quickly this occurs or whether it is complete [35]. A full randomised trial of the appropriate dosages is then required to review the efficacy of the technique.

37.5.3.1 WHO Recommendation

Intraumbilical vein injection of oxytocin with saline can be offered for the management of retained placenta (quality of evidence, moderate; strength of recommendation, weak). However manual removal of placenta always remains the definitive treatment. (No formal assessment of quality of evidence. Strength of recommendation: strong) [36–38].

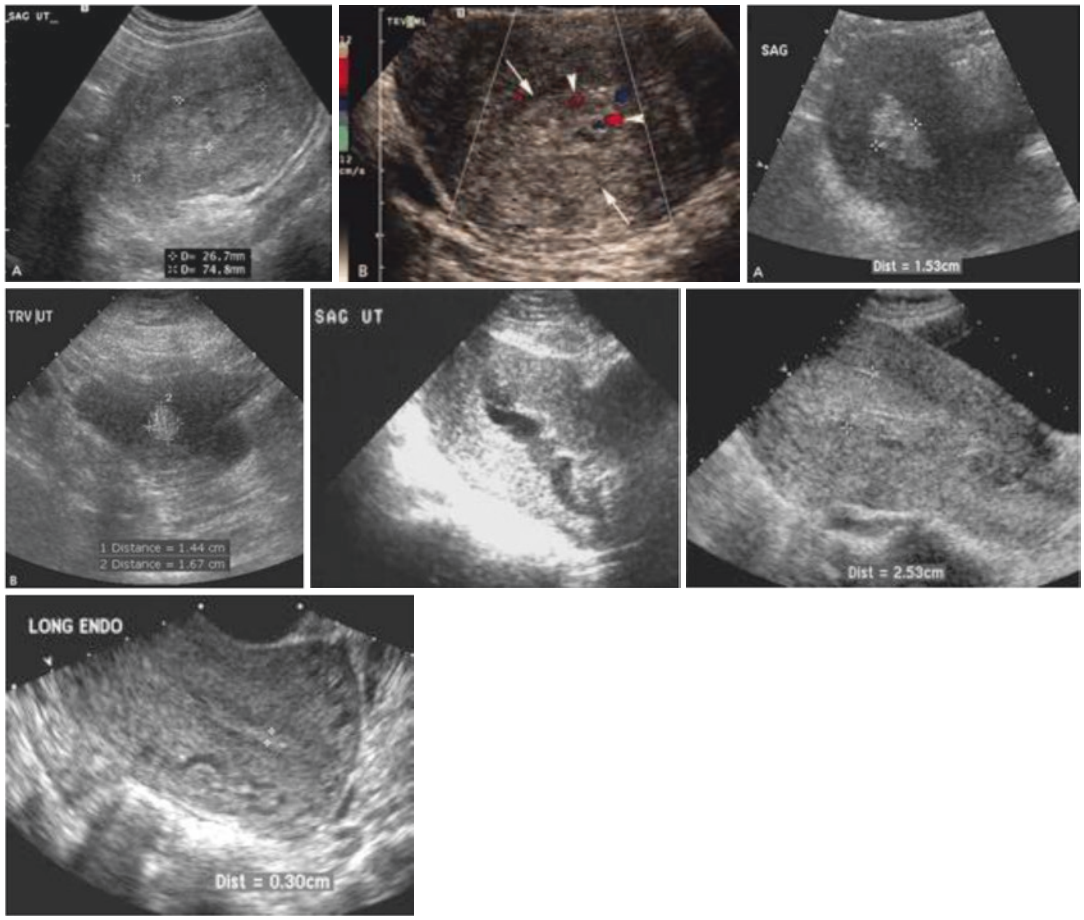


37.6 Conclusion

The retro-placental myometrium is the key in placental separation in the third stage of labour. Retained placentae are related with a localised

retro-placental contractile failure. This belt fails to contract throughout labour in many female with dysfunctional labours. The use of umbilical vein injections of oxytocin helps retain placentae to be treated medically. Medical management of this pathology of retained placenta will become the treatment of choice if delivery of oxytocics to retro-placental myometrium is improved. This could have important public health implications in rural areas where facilities for manual removal are not available.

> It makes sense that the placenta almost looks like a tree with many branches - a tree of life.
>
> —Ricki Lake



USG and colour Doppler images of retained placenta

References

1. Combs CA, Laros RK. Prolonged third stage of labour: morbidity and risk factors. Obstet Gynecol. 1991;77:863–7.
2. Gordon JE, Gideon H, Wyon JB. Midwifery practices in rural Punjab, India. Am J Obstet Gynecol. 1965;93:734–7.
3. Tandberg A, Albrechtsen S, Iverson DE. Manual removal of placenta. Acta Obstet Gynecol Scand. 1999;78:33–6.
4. Confidential Enquiry into Maternal Deaths. Why mothers die. Report of the confidential enquiry into maternal deaths 1994–1996. London: The Stationary Office; 1998.
5. Harrison KA. Childbearing, health and social priorities: survey of 22,774 consecutive hospital births

in Zaria, Northern Nigeria. Br J Obstet Gynaecol. 1985;92(Suppl 5):100–15.
6. Brandt ML. The mechanism and management of the third stage of labour. Am J Obstet Gynecol. 1933;25:662–7.
7. Herman A, Weinraub Z, Bukovsky I, et al. Dynamic ultrasonographic imaging of the third stage of labor: new perspectives into third stage mechanism. Am J Obstet Gynecol. 1993;168:1496–9.
8. Deyer TW, Ashton-Miller JA, Van Baren PM, Pearlman MD. Myometrial contractile strain at uteroplacental separation during parturition. Am J Obstet Gynecol. 2000;183:156–9.
9. Krapp M, Baschat AA, Hankeln M, Gembruch U. Gray scale and color Doppler sonography in the third stage of labor for early detection of failed placental separation. Ultrasound Obstet Gynecol. 2000;15:138–42.
10. Enkin MW, Wilkinson C. Manual removal of placenta at caesarean section (Cochrane review). In: The Cochrane Library, Issue 3. Oxford: Update Software; 1999.
11. Weeks AD. The role of the placenta in dysfunctional labour. Trans N Engl Obstet Gynaecol Sac.
12. Romero R, Hsu YC, Athanassiadis AP, et al. Preterm delivery: a risk factor for retained placenta. Am J Obstet Gynecol. 1990;163:823–5.
13. Lye SJ, Ou C-W, Teoh T-G, et al. The molecular basis of labour and tocolysis. Fetal Matern Med Rev. 1998;10:121–36.
14. Weeks AD, Stewart P. The use of low dose mifepristone and vaginal misoprostol for first trimester termination of pregnancy. Br J Fam Plan. 1995;21:85–6.
15. Weeks AD, Stewart P. The use of mifepristone in combi-nation with misoprostol for second trimester termination of pregnancy. Br J Fam Plan. 1995;21:43–4.
16. Hofmeyr GJ, Gulmezoglu AM. Vaginal misoprostol for cervical ripening and labour induction in late pregnancy (Cochrane review). In: The Cochrane Library, Issue 1. Oxford: Update Software; 2000.
17. Schellenberg J-C, Liggins GC. Initiation of labour: uterine and cervical changes, endocrine changes. In: Chard T, Grudzinskas JG, editors. The uterus. Cambridge: Cambridge University Press; 1994.
18. Thornton S, Terzidou V, Clark A, Blanks A. Progesterone metabolite and spontaneous myometrial contractions in vitro. Lancet. 1999;353:1327–9.
19. Ledingham M-A, Thompson AJ, Greer IA, Norman JE. Nitric oxide in parturition. Br J Obstet Gynaecol. 2000;107:581–93.
20. Ramsay B, Sooranna SR, Johnson MR. Nitric oxide synthase activities in human myometrium and villous trophoblast throughout pregnancy. Obstet Gynecol. 1996;87:249–53.
21. Thompson A, Telfer JF, Kohnen G, et al. Nitric oxide synthase activity and localisation do not change in uterus and placenta during human parturition. Hum Reprod. 1997;12:2546–52.
22. Sladek SM, Magness RR, Conrad KP. Nitric oxide and pregnancy. Am J Physiol. 1997;272:R441–63.
23. Carroli G, Belizan JM, Grant A, Gonzalez L, Campodonico L, Bergel E, Grupo Argentino de Estudio de Placenta Retenida. Intra-umbilical vein injection and retained placenta: evidence from a collaborative large randomised controlled trial. Br J Obstet Gynaecol. 1998;105:179–85.
24. Reyes FI, WInter JSD, Falman C. Postpartum disappearance of chorionic gonadotrophin from the maternal and neonatal circulations. Am J Obstet Gynecol. 1985;153:486–9.
25. Johanson RB. Mechanism and management of placental non-separation. In: Kingdom J, Jauniaux E, O'Brien S, editors. The placenta: basic science and clinical practice. London: RCOG Press; 2000.
26. Dunstone SJ, Leibowitz CB. Conservative management of placenta praevia with a high risk of placenta accreta. Aus N Z J Obstet Gynaecol. 1998;38:429–33.
27. Arulkumaran S, Ng CSA, Ingemarsson I, Ratnam SS. Medical treatment of placenta accreta with methotrexate. Acta Obstet Gynecol Scand. 1986;65:285–6.
28. Prendivlle WJ, Elbourne D, McDonald S. Active versus expectant management of the third stage of labour (Cochrane review). In: The Cochrane Library, Issue 3. Oxford: Update Software; 1998.
29. Danlelsson KG, Marions L, Rodriguez A, Spur BW, Wong PYK, Bygdeman M. Comparison between oral and vaginal administration of misoprostol on uterine contractility. Obstet Gynecol. 1999;93:275–80.
30. Bagley CM. A comparison of 'active' and 'physiological' management of the third stage of labour. Midwifery. 1990;6:3–17.
31. Donald I. Postpartum haemorrhage. In: Donald I, editor. Practical obstetric problems. London: Lloyd-Luke Ltd.; 1976.
32. McDonald S. Physiology and management of the third stage of labour. In: Bennett VR, Brown LK, editors. Myles textbook for midwives. 13th ed. Edinburgh: Churchill Livingstone; 1999.
33. Ng PS, Chan ASM, Sin WK, Tang LCH, Cheung KB. Yeun PM. A multlcentre randomized controlled trial of oral misoprostol and i.m. syntometrine In the management of the third stage of labour. Hum Reprod. 2001;16:31–5.
34. Carroll G, Bergel E. Umbilical vein injection for management of retained placenta (Cochrane review). In: The Cochrane Library, Issue 4. Oxford: Update Software; 2000.
35. Pipingas A, Hofmeyr GJ, Sese IKR. Umbilical vessel oxytocin administration for retained placenta: in-vitro study of various Infusion techniques. Am J Obstet Gynecol. 1993;168:793–5.
36. Wilken-Jensen C, Strom V, Nielsen MD, Rosenkilde-Gram B. Removing placenta by oxytocin—a controlled study. Am J Obstet Gynecol. 1989;161:155–6.
37. Hendricks CH, Brenner WE. Cardiovascular effects of oxytocic drugs used post partum. Am J Obstet Gynecol. 1970;108:751.
38. Patient C, Davison JM, Charlton L, Baylis PH, Thornton S. The effect of labour and maternal oxytocin Infusion on fetal plasma oxytocin concentration. Br J Obstet Gynecol. 1999;106:1311–3.

Further Reading for WHO Recommendation

Carroli G, Bergel E. Umbilical vein injection for management of retained placenta. Cochrane Database Syst Rev. 2001;(4):CD001337.

Chongsomchai C, Lumbiganon P, Laopaiboon M. Prophylactic antibiotics for manual removal of retained placenta in vaginal birth. Cochrane Database Syst Rev. 2006;(2):CD004904.

Criscuolo JL et al. [The value of antibiotic prophylaxis during intrauterine procedures during vaginal delivery. A comparative study of 500 patients]. J Gynecol Obstet Biol Reprod. 1990;19(7):909–18.

Smaill F, Hofmeyr GJ. Antibiotic prophylaxis for cesarean section. Cochrane Database Syst Rev. 2002;3:CD000933.

The Release Trial: a randomised trial of umbilical vein oxytocin versus placebo for the treatment of retained placenta. http://isrctn.org/ISRCTN13204258.

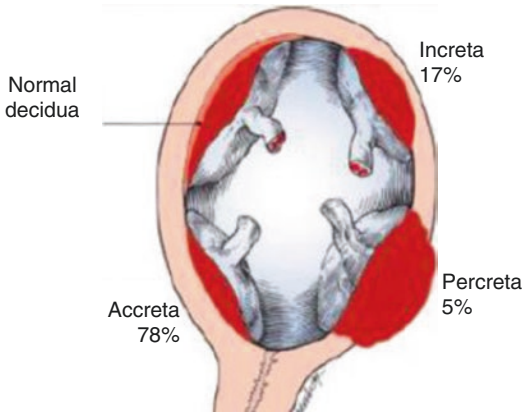
Van Beekhuizen HJ, et al. Sulprostone reduces the need for the manual removal of the placenta in patients with retained placenta: a randomized controlled trial. Am J Obstet Gynecol. 2006;194(2):446–50.

Placental Adhesive Disorders

38

Uday Thanawala and Saloni Suchak

Described by Irving and Hertig in 1937 as "the abnormal adherence, either in whole or in part, of the afterbirth to the underlying uterine wall", this term is used to describe also percreta and increta. It occurs as a consequence of partial or complete absence of the decidua basalis and defective formation of Nitabuch's layer.



Depending on the degree of invasion, the adherent placentas can be:

- Accreta: placental villi attached to the myometrium
- Increta: invasion of the placental villi into the myometrium
- Percreta: placental villi completely penetrating the myometrium, including breaching the serosa and invading the surrounding structures, such as the bladder, broad ligament or sigmoid colon

Based on the number of lobules involved, the anomalous attachment can be described as:

- Total placenta accreta: involves all lobules
- Partial placenta accreta: involves at least two but not all of the lobules
- Focal placenta accreta: involves only a single lobule, either a portion or the entire lobule

38.1 A Dreadful Condition

An adherent placenta is an obstetrician's nightmare. Clinically it can lead to:

- Massive obstetric haemorrhage, leading to disseminated intravascular coagulopathy.
- Hysterectomy.
- Risk of surgical injury to the ureters, bladder, bowel, or neurovascular structures.
- ICU complications: adult respiratory distress syndrome, acute transfusion reaction, electrolyte imbalance and renal failure.
- Blood loss: The average blood loss at delivery in women with placenta accreta is 3000–5000 mL [1].

U. Thanawala (✉)
Thanawala Maternity Home,
Navi Mumbai, Maharashtra, India

S. Suchak
Suchak Hospital, Mumbai, Maharashtra, India

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As many as 90% of patients with placenta accreta require blood transfusion, and 40% require more than 10 units of packed red blood cells.

Maternal mortality with placenta accreta has been reported to be as high as 7% [2]. Maternal death may occur despite optimal planning, transfusion management and surgical care.

38.2 Incidence Is Increasing!

The reported incidence of placenta accreta has increased from approximately 0.8 per 1000 deliveries in the 1980s to 3 per 1000 deliveries in the past decade.

- 1 in 4027 pregnancies in the 1970s
- 1 in 2510 pregnancies in the 1980s [3]
- 1 in 533 pregnancies for the period of 1982–2002 [4]

Why Is This Happening?

It has been noticed that the rising incidence of placenta accreta parallels rising caesarean section rate [5]. It is possibly the incision on the uterus which causes abnormal vascularization and tissue oxygenation of the scar area resulting in defective decidualization and excessive trophoblastic invasion.

38.2.1 Risk Factors

Accordingly women at the highest risk for placenta accreta are the ones who have had myometrial damage from a previous caesarean delivery followed by implantation of the placenta over previous uterine scar. The risk for placenta accreta in a patient with placenta praevia and prior caesarean delivery increases with the number of previous caesareans. Silver and colleagues reported the risk for the first, second, third, fourth and fifth or greater caesarean delivery to be 3.3%, 11%, 40%, 61% and 67% [6]. Additional risk factors include uterine surgery such as myomectomy, endometrial ablation and dilatation and curettage. The reported incidence of placenta accreta has increased from approximately 0.8 per 1000 deliveries in the 1980s to 3 per 1000 deliveries in the past decade [7] (Table 38.1).

95% of women have identifiable risk factors, and one should be always aware of this in the history. *If a patient has a placenta praevia or low-lying placenta, especially when she has had a previous LSCS*, *look for it!* This is important since only a high degree of suspicion is the key to have a prenatal diagnosis of this condition—in which undiagnosed leads to definite morbidity and even mortality.

38.2.2 Diagnosis: Ultrasound?/ Colour Doppler?/MRI?

Prenatal diagnosis and knowledge of the extent of placental invasion are instrumental in optimizing patient outcomes in placenta accreta. Foreknowledge allows for referral to a tertiary care centre, multidisciplinary approach when necessary and meticulous planning.

All antenatal patients need to have a routine ultrasound scanning at 20 weeks of gestation that should include placental localization [9].

Table 38.1 Categorizing risk factors

Most frequent	Frequent	Infrequent	Non-specific
• Previous caesarean [8] • Placenta praevia • Multiple and abrasive D and C	• Anterior placenta and previous iterative caesarean sections • Placental insertion in area of previous uterine surgery • Endometrial infection after abortion	• Endometrial thermablation • Radiation • Placenta praevia and assisted conception techniques	• Tabaquism (TOBACCO ADDICTION) • Age over 35 years

Table 38.2 Ultrasonographic findings

Ultrasonographic findings suggestive of placenta accreta are as follows
1. Loss of normal hypoechoic retroplacental zone
2. Multiple vascular lacunae (irregular vascular spaces) with placenta giving "Swiss cheese" appearance [12]
3. Blood vessels or placental tissue bridging uterine-placental margin, myometrial-bladder interface or crossing uterine serosa
4. Retroplacental myometrial thickness <1 mm
5. Numerous coherent vessels visualized with three-dimensional power Doppler in basal view [13]

38.2.2.1 Ultrasound

- In general, greyscale predicts abnormal placentation with a sensitivity of 77–86%, specificity 96–98%, positive predictive value 63–88% and a negative predictive value 95–98%. Suspected diagnosis of placenta praevia at 20 weeks of gestation by abdominal scan should be confirmed by transvaginal scan [9]. TVS will reclassify 26–60% of cases where the abdominal scan diagnosed a low-lying placenta, meaning fewer women will need follow-up. Numerous prospective observational trials have used TVS to diagnose placenta praevia, and none has experienced any haemorrhagic complications, thus confirming the safety of this technique. So, do not hesitate to do a TVS in case of a placenta praevia [10, 11]. With three-dimensional power Doppler, visualizing numerous coherent vessels on the basal view has been reported to have a sensitivity of 97% and a specificity of 92% (Table 38.2).

38.2.2.2 Colour Doppler

- Diffuse or focal lacunar flow
- Vascular lakes with turbulent flow (peak systolic velocity over 15 cm/s)
- Hyper-vascularity of serosa-bladder interface
- Markedly dilated vessels over peripheral subplacental zone
- The additional use of colour Doppler does not significantly improve diagnostic sensitivity over grey-scale ultrasonography alone.

38.2.2.3 Magnetic Resonance Imaging

The most valuable features of placenta accreta on MRI are:

- Dark intraplacental bands on T2-weighted sequences
- Uterine bulging
- Heterogeneous signal intensity within the placenta
- The American College of Radiology recommends that MRI be used in pregnancy only if the diagnostic information cannot be obtained with ultrasonography [14]. They further specify that MRI contrast agents should not be used, and the use is only justified with "overwhelming potential benefit to the patient or fetus". The benefits are proper planning and optimizing the maternal outcome. Antenatal ultrasound can be complemented by magnetic resonance imaging in equivocal cases to distinguish those women at special risk of placenta accreta [9].

As of today, the mainstay of prenatal diagnosis for abnormal placentation remains ultrasound with MRI being used only as an adjunct in intermediate cases. An ultrasound or MRI diagnosis of placenta accreta predicts the need for hysterectomy with a sensitivity of 78%, 67% and a specificity of 67%, 50%, respectively [15].

Follow-up scan at 32 weeks: Placenta praevia or suspected placenta accreta, imaging should be performed at around 32 weeks of gestation to clarify the diagnosis and allow planning for third-trimester management, further imaging and delivery [9].

- 90% of major praevias at this gestation will persist [16].

38.3 Antenatal Management

If women are managed at home, they must have safety precautions in place and have ready access to the hospital. They must report any bleeding, contractions or pain (including vague suprapubic period-like aches). Anaemia corrected by IV iron in the antenatal period/packed cell transfusion where near to term. A haematocrit greater than 30 is a reasonable goal [17]. Most patients who undergo an obstetric hysterectomy will need a blood transfusion, hence ensuring that the patient does not have any rare alloantibodies before delivery is also important.

All these patients have to receive antenatal steroids at 26 weeks of gestation (2 doses of 12 mg betamethasone 24 h apart or 4 doses of 8 mg dexamethasone 12 h apart).

Prolonged inpatient care can be associated with an increased risk of thromboembolism; therefore, mobility should be encouraged together with the use of thromboembolic deterrent stockings and adequate hydration. Limiting anticoagulant thromboprophylaxis to those at high risk of thromboembolism seems reasonable [18].

38.4 Delivery Preparation (Care Bundle)

Referral to a tertiary centre and a multidisciplinary approach is of the utmost importance.

The six elements considered to be reflective of good care were:

1. Consultant obstetrician planned and directly supervising delivery.
2. Consultant anaesthetist planned and directly supervising anaesthetic at delivery.
3. Blood and blood products available.
4. Multidisciplinary involvement in pre-op planning.
5. Discussion and consent include possible interventions (such as hysterectomy, leaving the placenta in place, cell salvage and intervention radiology).
6. Local availability of a level 2 critical care bed.

38.5 Timing of Delivery

The ACOG in July 2012 were of the opinion that the delivery timing for placenta accreta must be individualized. Patient must be counselled for high potential of hysterectomy, profuse haemorrhage, probable blood transfusion, increased complications and possible maternal death. Compared to a peripartum hysterectomy, a planned obstetric hysterectomy has been shown to have decreased intraoperative blood loss, less intraoperative hypotension and decreased need of blood transfusion. The benefit of a planned delivery before the onset of labour must be weighed against the risk of prematurity for the neonate. Patients with placenta praevia and a cervical length less than 30 mm at 32 weeks have been found to have an increased risk of haemorrhage, uterine activity and preterm birth [19].

An expert opinion in 2010 recommended the delivery for an uncomplicated praevia at 36–37 weeks and at 34–35 weeks for a suspected placental invasion [20]. According to a recent survey of 508 members of the Society for Maternal Fetal Medicine, many maternal-fetal medicine practitioners perform amniocentesis for fetal lung maturity before delivery (46.8%) which they most commonly schedule at 36 weeks (48.4%). **In the Indian context, doing an amniocentesis for lung maturity is not practical, and that is the reason that all these cases should receive antenatal steroids at 26 weeks**. A contingency plan for an emergency hysterectomy must be outlined including protocols for massive transfusions and the multidisciplinary team contact in the afterhours.

In summary, each patient must be evaluated, and an individualized delivery plan must be formed based on placental location, extent of invasion, cervical length, clinical course in pregnancy and the capabilities of the delivering facility and care team.

- Consent: The different risks and treatment options should have been discussed and a plan agreed, which should be reflected clearly in the consent form.

- This should include the anticipated skin and uterine incisions and whether conservative management of the placenta or proceeding straight to hysterectomy is preferred in the situation where accreta is confirmed at surgery.

Additional possible interventions in the case of massive haemorrhage should also be discussed, including cell salvage and interventional radiology when available.

38.6 Preoperative Planning

Pneumatic compression stockings should be placed preoperatively and maintained until the patient is fully ambulatory.

Prophylactic Antibiotics

Preoperative cystoscopy with placement of ureteral stents may help prevent inadvertent urinary tract injury.

Packed red blood cells and thawed fresh frozen plasma should be available in the operating room.

Blood bank should be placed on alert for a potential massive haemorrhage. Current recommendations for blood replacement in trauma situations suggest a 1:1 ratio of packed cells to fresh frozen plasma.

Surgical Plan

- Scan to map the placenta to guide about skin incision.
- Attempting placental separation risks hysterectomy in up to 100% of cases and therefore is illogical.
- Consider opening the uterus at a site distant from the placenta and delivering the baby without disturbing the placenta.
- Conservative management of placenta accreta when the woman is already bleeding is unlikely to be successful and risks wasting valuable time [21].

38.6.1 Anaesthesia

Neuraxial anaesthesia has become more common in deliveries involving placenta accreta. Continuous epidural and combined spinal-epidural anaesthesia are both viable options with a reported rate of conversion to general anaesthesia of about 28–29% when regional anaesthesia was first used [22]. General anaesthesia is recommended in a difficult airway, extensive dissection, prolonged operating time and massive haemorrhage where anticipated.

38.6.2 Interventional Radiology

Therapies in conjunction with interventional radiology, such as internal iliac balloon catheterization or postpartum arterial embolization, have been described with mixed results. Usually preoperative placement of bilateral endovascular internal iliac artery balloons occurs followed by inflation and subsequent occlusion of the bilateral internal iliac arteries after delivery of the infant. Decreased mean blood loss, mean blood volume transfused and duration of surgery have been reported in some studies but with no difference in mean haemoglobin change or intensive care unit admission.

38.7 Surgical Technique

Position: Consideration should be given in placing the patient on the operating table in specialized stirrups in a modified dorsal lithotomy position with left lateral tilt to allow for direct assessment of vaginal bleeding, provide access for placement of a vaginal pack and allow additional space for a surgical assistant.

Incision: Most caesarean deliveries are now performed via a Pfannenstiel incision; in the presence of a probable accreta, a median or paramedian skin incision may be preferable for the advantages of improved visibility and superior

access to the fundus or even posterior uterine wall for possible alternative hysterotomy sites as well as for hysterectomy. If the surgery is begun with a Pfannenstiel incision and exposure is not adequate, the best approach is to perform a modified Cherney incision.

Abnormal placentation can create aberrant vasculature and dilated vessels over the area of the placental insertion. In cases of placental accreta, the areas of placental invasion outside the uterus may also be affected by the abnormal blood supply. Care should be taken not to compromise the parasitic vasculature when entering the abdomen and exposing the uterus. With extensive placental invasion, profuse haemorrhage immediately develops with attempted placental delivery or if the placenta is incised during the hysterotomy. With uteroplacental blood flow at 700–900 mL/min near term, every minute of haemorrhage avoided is significant. If the placenta is in the lower uterine segment and especially in the setting of a percreta, fully developing the bladder flap and dissecting it around the placental invasion before hysterotomy can aid in prompt haemorrhage control and hysterectomy if necessary. In addition to preparing the bladder flap and avoiding damage to dilated vasculature, making the hysterotomy well away from the placenta will further avoid massive haemorrhage. A trans-fundal or even posterior uterine wall incision may be required depending on placental location [23, 24].

With suspected placenta accreta, the most conservative approach to avoid life-threatening haemorrhage is proceeding to planned hysterectomy with no attempt at placental delivery and the placenta left in situ. After the delivery, the cord is ligated and cut close to the placenta, the placenta is not delivered, and the edges of the vertical incision are quickly re-approximated, with either three or four towel clips or with a mass running suture, for haemostasis. Ensure adequate uterine tone with pitocin or other uterotonics as needed. Quick haemostasis can be gained by the delayed ligation technique where round ligaments, utero-ovarian ligaments and tubes, and uterine vessels are doubly clamped and cut from their attachments before any ties are placed. Delayed bleeding once the engorged vessels normalize can be avoided by using two Heaney clamps for the vascular uterine and adnexal pedicles with a simple suture replacing the proximal clamp and a Heaney-transfixing suture replacing the distal. When placental bladder invasion is suspected, the extent of invasion can be evaluated by cystotomy once the uterine blood supply is interrupted. If the trigone is not involved in the placental invasion, the involved portion of the bladder can be resected or left adherent to the uterus (Table 38.3).

Table 38.3 Surgical Technique

Surgical Technique
• Dorsal lithotomy position (advisable)
• Vertical midline skin incision
• Dissect bladder flap before delivery
• Classical uterine incision away from the placenta
• No attempt at placenta removal
• Placenta left in situ
• Hysterectomy

With imaging modalities having only a 63–88% positive predictive value, when future fertility is desired and index of suspicion for placenta accreta is low, an initial attempt to deliver the placenta is reasonable. If the placenta separates or the diagnosis of accreta was not anticipated, a focal area of invasion or bleeding from the placental bed may be discovered after placental removal. If oversewing the placental bed is not adequate, successful treatment with uterine tamponade via a balloon or packing has been described [25]. A haemostatic square suturing technique to approximate posterior and anterior uterine walls, especially in areas with heavy bleeding, has also been reported. Of the 23 cases where the square suturing technique was described, all resumed normal menstrual function, and six had confirmation of normal postpartum uterine cavities [26].

38.7.1 Persistent Haemorrhage

Massive haemorrhage can lead to coagulopathy, and tissue oedema/friability can lead to bleeding not fully controlled after removal of the uterus. Uterine artery and hypogastric artery ligation are

often mentioned in conjunction with severe postpartum haemorrhage. The technique requires surgical experience in the retroperitoneum, and visualization may be difficult with severe haemorrhage. Careful manipulation of the hypogastric artery must be performed lateral to medial, and absorbable suture should be placed 3 cm distal to the bifurcation of the common iliac artery to avoid the posterior branch of the internal iliac artery. Possible complications include limb and/or tissue ischemia as well as haemorrhage if the iliac vein is compromised during dissection [27].

38.7.2 Resuscitation

When a large blood loss is anticipated, several techniques can be used to decrease the chance of allogenic transfusion.

- Autologous blood donation/transfusion
- Acute normovolaemic haemodilution
- Intraoperative cell salvage
- Haemostatic resuscitation

Autologous transfusions can be done after 30 weeks but require a haematocrit of 34 and at least 2 weeks recovery before surgery. Acute normovolaemic haemodilution (ANH) is begun in the operating room before the start of surgery where 2–3 units of whole blood are collected from the patient and replaced by colloid/crystalloid to maintain normovolaemia. The patient must have an initial haemoglobin of at least 10 g/dL, no history of cardiac disease and a predicted blood loss of at least 20% of the patient blood volume. The blood can be stored at room temperature for at least 6 h. In the late 1990s, patients with massive haemorrhage were resuscitated with large volumes of crystalloid and packed red blood cells (PRBCs). Other products such as fresh frozen plasma (FFP), platelets and cryoprecipitate were used only as indicated by abnormal haematologic parameters such as fibrinogen <100 mg/dL, platelets <50,000/mm^3 or abnormal coagulations studies. This approach prevented coagulopathies in massive haemorrhage, and also the liberal use of crystalloid/PRBCs alone creates a dilution of clotting factors or "dilutional coagulopathy". Hypothermia and acidosis can further aggravate the patient coagulation dysfunction.

The use of whole blood in obstetric haemorrhage has been shown to address possible coagulopathy, decrease the rate of acute tubular necrosis and reduce the donor exposures. Aggressive crystalloid resuscitation is avoided not only to prevent haemodilution but also to circumvent clots breaking free after volume expansion and increasing blood pressure. In addition, keeping the systolic blood pressure between 80 and 100 mmHg may be optimal to limit continuing blood loss.

Recombinant factor VIIa has been shown to limit the amount of blood products transfused in haemorrhage but has not been shown to have a survival benefit. Recombinant factor VIIa binds to tissue factor and activates the clotting cascade so fibrinogen and clotting factors must be present for it to be effective. There are also valid concerns about thromboembolism associated with its use.

These theories have led to haemostatic resuscitation, which has three main concepts:

- Permissive hypotension
- 1:1:1 ratio transfusion of PRBC/FFP/platelets
- Early use of recombinant factor VIIa

Although these concepts were developed for trauma patients, some evidence exists that increased fibrinolytic activity and similar processes also occur in obstetric haemorrhage, and this modern resuscitation technique is often used in massive obstetric haemorrhage.

38.8 Conservative Surgery: Leaving the Placenta In Situ

Alternative approaches to the management of placenta accreta have been reported whereby the placenta is left in situ and a hysterectomy is not performed. Most involve ligation of the umbilical cord close to the insertion site, either avoiding attempts at placental separation completely or minimizing the placental size with resection and retention of only the adherent placental segments,

and closure of the hysterotomy without hysterectomy. Additional treatments used with alternative approaches included arterial ligation (such as uterine or bilateral hypogastric artery ligation), embolization with interventional radiology, administration of uterotonics and/or methotrexate therapy. The placenta is either reabsorbed or removed at a later date with curettage and/or hysteroscopic resection [28].

Risks with this approach

- Outcome unpredictable.
- Increased risk of significant complications, like infection and haemorrhage, as well as the need for later hysterectomy.
- Reported cases of subsequent successful pregnancy in patients treated with this approach are rare.

In a paper by Bretelle, of the 26 patients treated with this approach, 21 (80.7%) successfully avoided hysterectomy, whereas 5 (19.3%) eventually required it. However, the majority of the 21 patients who avoided hysterectomy did require additional treatment, including hypogastric artery ligation, arterial embolization, methotrexate, blood product transfusion, antibiotics or curettage. Except in specific cases, hysterectomy remains the treatment of choice for patients with placenta accreta [29].

38.9 Methotrexate

- As methotrexate is a folate antagonist and decreases trophoblast activity, it has been suggested that administration will decrease placental vascularity and lead to placental necrosis and absorption. But here the trophoblasts are no longer dividing, thereby rendering methotrexate ineffective. Small studies have reported mixed results. No convincing data for the use of methotrexate for postpartum management of placenta accreta [30].

The dosing of methotrexate that has been used is highly variable, ranging from one 50-mg intramuscular postpartum dose to a 50-mg dose injected into the umbilical vein at the time of caesarean section with a 50-mg intramuscular dose postoperatively within 4 days followed by weekly 50-mg intramuscular doses for up to 4 weeks [31].

Common toxicities are nausea, vomiting, diarrhoea and mucosal ulcers, but numerous other side effects exist including leukopenia, anaemia, gastrointestinal ulcerations, dose-related hepatotoxicity and a rare hypersensitivity-like lung reaction. Many patients treated conservatively and studied retrospectively did not receive methotrexate, and placental absorption still occurred.

38.10 Postoperative Care

Patients undergoing peripartum hysterectomy are in danger of complications related to intraoperative hypotension, anaemia, continued coagulopathy, possible recurrent haemorrhage and prolonged operative time. Respiratory, cardiac, renal, endocrine and other organ system dysfunctions are common. Pulmonary oedema, acute respiratory distress syndrome, renal failure requiring dialysis, acute tubular necrosis and, with large transfusions, transfusion-related lung injury have all been reported [32].

Patients should have close monitoring of their vital signs, strict inputs and outputs recorded with urine output being measured via an indwelling urinary catheter, centralized monitoring with assessment of peripheral oxygenation by pulse oximetry and initial trending of electrolytes, complete blood counts and coagulation studies to include fibrinogen. Correction of persistent severe anaemia or coagulopathies with further blood products and treatment of electrolyte abnormalities is important for continued stabilization of the patient. Appropriate thromboprophylaxis based on the patient's individual risk factors and postpartum hypercoagulability state should also be initiated. The surgeon should have a low threshold for re-exploration and haemorrhage control when bleeding is suspected [33].

38.11 Case Report

The author Dr. Saloni Suchak has managed a case conservatively.

36-year-old gravida 2, para 1 living 1 previous LSCS with placenta percreta and placenta covering that is diagnosed at 20+ weeks. At 38 weeks, planned for elective LSCS with consent taken for an obstetric hysterectomy. Blood and blood products kept ready and managed in a hospital set-up with a good ICU backup. Abdomen opened via a midline vertical incision and on opening placenta noted extending laterally on both sides till the lateral pelvic wall with no place to place any clamps, placenta over the lower segment. Classical C section done avoiding incision over placenta. Baby delivered by breech extraction, cord cut and placenta put back into the uterine cavity with no attempt at separation. Uterus closed and procedure completed with adequate haemostasis. Post-op patient was given inj methotrexate on day 0, 4 and 7 and then repeated on day 14. Beta HCG levels were monitored, and patient was also given inj leucovorin as per the protocol. RFTs and LFTs monitored closely. Patient did not require any transfusions. The placental bulk slowly reduced with time and vascularity decreased over the next 3 months. Patient had a bout of bleeding around a month post-op where she passed some placental bits. Six months later the retained bits were completely resorbed. Good antibiotic cover and close monitoring helped in the successful outcome of this case.

38.12 Summary

- Management of placenta accreta at a tertiary care centre and a multidisciplinary team approach improve patient outcomes.
- Greyscale ultrasound has 77–86% sensitivity and 96–98% specificity for diagnosis of placenta accreta.
- MRI should only be used in ambiguous cases, and gadolinium contrast should be avoided.
- Delivery timing should be individualized but generally around 34–35 weeks estimated gestational age.
- Acute normovolaemic haemodilution, intraoperative cell salvage, whole blood use and haemostatic resuscitation can all be tools to aid in treatment of predicted increased blood loss.
- Interventional radiology techniques, such as hypogastric or aortic occlusion, may be used but likely do not alter overall major clinical outcomes.
- Planned caesarean hysterectomy with no attempt at placental delivery is the treatment of choice.
- Alternative approaches, such as leaving the placenta in situ without hysterectomy, have increased risks and should be reserved for individualized patients.
- Patients should be monitored closely postoperatively and may require intensive care unit admission.

References

1. Hudon L, Belfort MA, Broome DR. Diagnosis and management of placenta percreta: a review. Obstet Gynecol Surv. 1998;53:509–17.
2. O'Brien JM, Barton JR, Donaldson ES. The management of placenta percreta: conservative and operative strategies. Am J Obstet Gynecol. 1996;175:1632–8.
3. Wu S, Kocherginsky M, Hibbard JU. Abnormal placentation: twenty-year analysis. Am J Obstet Gynecol. 2005;192:1458–61.
4. Read JA, Cotton DB, Miller FC. Placenta accreta: changing clinical aspects and outcome. Obstet Gynecol. 1980;56:31–4.
5. Wortman AC, Alexander JM. Placenta accrete, increta and percreta. Obstet Gynecol Clin North Am. 2013;40(1):137–54.
6. Silver RM, Landon MB, Rouse DJ, et al., National Institute of Child Health and Human Development Maternal-Fetal Medicine Units Network. Maternal morbidity associated with multiple repeat cesarean deliveries. Obstet Gynecol. 2006;107:1226–32.
7. Flood KM, Said S, Geary M, Robson M, Fitzpatrick C, Malone FD. Changing trends in peripartum hysterectomy over the last 4 decades. Am J Obstet Gynecol. 2009;200:632.e1–632.e6 (Multiple time series level II-3).
8. Clark SL, Koonings PP, Phelan JP. Placenta previa/accreta and prior cesarean section. Obstet Gynecol. 1985;66:89–92.
9. Placenta praevia, placenta praevia accreta and vasa praevia: diagnosis and management. Green–top guideline no. 27, Jan 2011.

10. Smith RS, Lauria MR, Comstock CH, Treadwell MC, Kirk JS, Lee W, et al. Transvaginal ultrasonography for all placentas that appear to be low-lying or over the internal cervical os. Ultrasound Obstet Gynecol. 1997;9:22–4.
11. Lauria MR, Smith RS, Treadwell MC, Comstock CH, Kirk JS, Lee W, et al. The use of second-trimester transvaginalsonography to predict placenta praevia. Ultrasound Obstet Gynecol. 1996;8:337–40.
12. Lim PS, Greenberg M, Edelson MI, et al. Utility of ultrasound and MRI in prenatal diagnosis of placenta accreta: a pilot study. AJR Am J Roentgenol. 2011;197(6):1506–13.
13. Hull AD, Salerno CC, Saenz CC, Pretorius DH. Three-dimensional ultrasonography and diagnosis of placenta percreta with bladder involvement. J Ultrasound Med. 1999;18:853–6.
14. Shih JC, Palacios Jaraquemada JM, Su YN, et al. Role of three-dimensional power Doppler in the antenatal diagnosis of placenta accreta: comparison with gray-scale and color Doppler techniques. Ultrasound Obstet Gynecol. 2009;33:193–203.
15. Warshak CR, Eskander R, Hull AD, et al. Accuracy of ultrasonography and magnetic resonance imaging in the diagnosis of placenta accreta. Obstet Gynecol. 2006;108:573–81.
16. Dashe JS, McIntire DD, Ramus RM, Santos-Ramos R, Twickler DM. Persistence of placenta previa according to gestational age at ultrasound detection. Obstet Gynecol. 2002;99:692–7.
17. Imdad A, Bhutta ZA. Routine iron/folate supplementation during pregnancy: effect on maternal anaemia and birth outcomes. Paediatr Perinat Epidemiol. 2012;26(Suppl 1):168–77.
18. Royal College of Obstetricians and Gynaecologists. Green-top guideline no. 37: reducing the risk of thrombosis and embolism during pregnancy and the puerperium. London: RCOG; 2009.
19. Stafford IA, Dashe JS, Shivvers SA, et al. Ultrasonographic cervical length and risk of hemorrhage in pregnancies with placenta previa. Obstet Gynecol. 2010;116(3):595–600.
20. Spong CY, Mercer BM, et al. Timing of indicated late-preterm and early-term birth. Obstet Gynecol. 2011;118(2 Pt 1):323–33.
21. Eller AG, Porter TF, Soisson P, Silver RM. Optimal management strategies for placenta accreta. BJOG. 2009;116:648–54.
22. Lilker SJ, Meyer RA, Downey KN, et al. Anesthetic considerations for placenta accreta. Int J Obstet Anesth. 2011;20(4):288–92.
23. Catling S. Blood conservation techniques in obstetrics: a UK perspective. Int J Obstet Anesth. 2007;16(3):241–9.
24. Belfort MA. Placenta accreta. Am J Obstet Gynecol. 2010;203(5):430–9.
25. Vrachnis N, Iavazzo C, Salakos N, et al. Uterine tamponade balloon for the management of massive hemorrhage during cesarean section due to placentaprevia/increta. Clin Exp Obstet Gynecol. 2012;39(2):255–7.
26. Cho JH, Jun HS, Lee CN. Hemostatic suturing technique for uterine bleeding during cesarean delivery. Obstet Gynecol. 2000;96(1):129–31.
27. Porreco RP, Stettler RW. Surgical remedies for postpartum hemorrhage. Clin Obstet Gynecol. 2010;53(1):182–95.
28. Sentilhes L, Ambroselli C, Kayem G, et al. Maternal outcome after conservative treatment of placenta accreta. Obstet Gynecol. 2010;115(3):526–34.
29. Bretelle F, Courbiere B, Mazouni C, Agostini A, Cravello L, Boubli L, et al. Management of placenta accreta: morbidity and outcome. Eur J Obstet Gynecol Reprod Biol. 2007;133:34–9.
30. Mussalli GM, Shah J, Berck DJ, Elimian A, Tejani N, Manning FA. Placenta accreta and methotrexate therapy: three case reports. J Perinatol. 2000;20:331–4.
31. Mussalli GM, Shah J, Berck DJ, et al. Placenta accreta and methotrexate therapy: three case reports. J Perinatol. 2000;20(5):331–4.
32. Alexander JM, Sarode R, McIntire DD, et al. Whole blood in the management of hypovolemia due to obstetric hemorrhage. Obstet Gynecol. 2009;113(6):1320–6.
33. American College of Obstetricians, Gynecologists. ACOG practice bulletin no. 100: critical care in pregnancy. Obstet Gynecol. 2009;113(2 Pt 1):443–50.

Inversion Uterus

39

Pratima Mittal and Jyotsna Suri

39.1 Introduction

Inversion of the puerperal uterus, which is the passage of the uterine fundus through the endometrial cavity and the cervix, is the turning of the uterus inside out (Fig. 39.1). It is a rare but catastrophic complication of the third stage of labor, which is associated with a very high maternal morbidity and mortality, and hence it is very important for all obstetricians to be familiar with the emergency management of this condition.

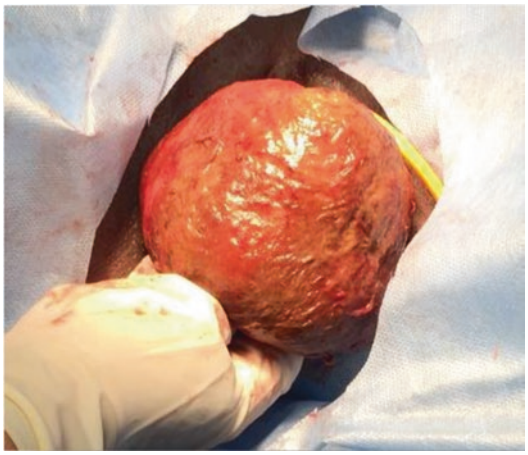

Fig. 39.1 A case of acute third-degree puerperal inversion of the uterus

P. Mittal (✉) · J. Suri
Vardhman Mahavir Medical College & Safdarjung Hospital, New Delhi, Delhi, India

The incidence of uterine inversion has been variably reported from 1 in 1200–57,000 deliveries [1]. A retrospective review over a 24-year period estimated an incidence of 1 in 3737 after vaginal delivery and 1 in 1860 after cesarean section [2]. After the institution of active management of the third stage of labor in 1988, the incidence of inversion following vaginal delivery has fallen 4.4-fold [2].

39.2 Classification

Inversion of the uterus can be categorized into four degrees, according to the extent of inversion of the fundus [3] (Fig. 39.2):

- First degree: the fundus dips into the uterine cavity; also known as incomplete inversion.
- Second degree: the fundus traverses the uterine cavity through the cervix; also known as complete inversion.
- Third degree: the fundus protrudes up to or beyond the introitus; also called the prolapsed inversion.
- Fourth degree: the uterus and vagina invert completely and come out of the introitus; referred to as total inversion. This condition is most often seen in the non-puerperal state.

It has been observed that most of the cases (90%) present as second- or third-degree acute inversion to the emergency room [1].

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_39

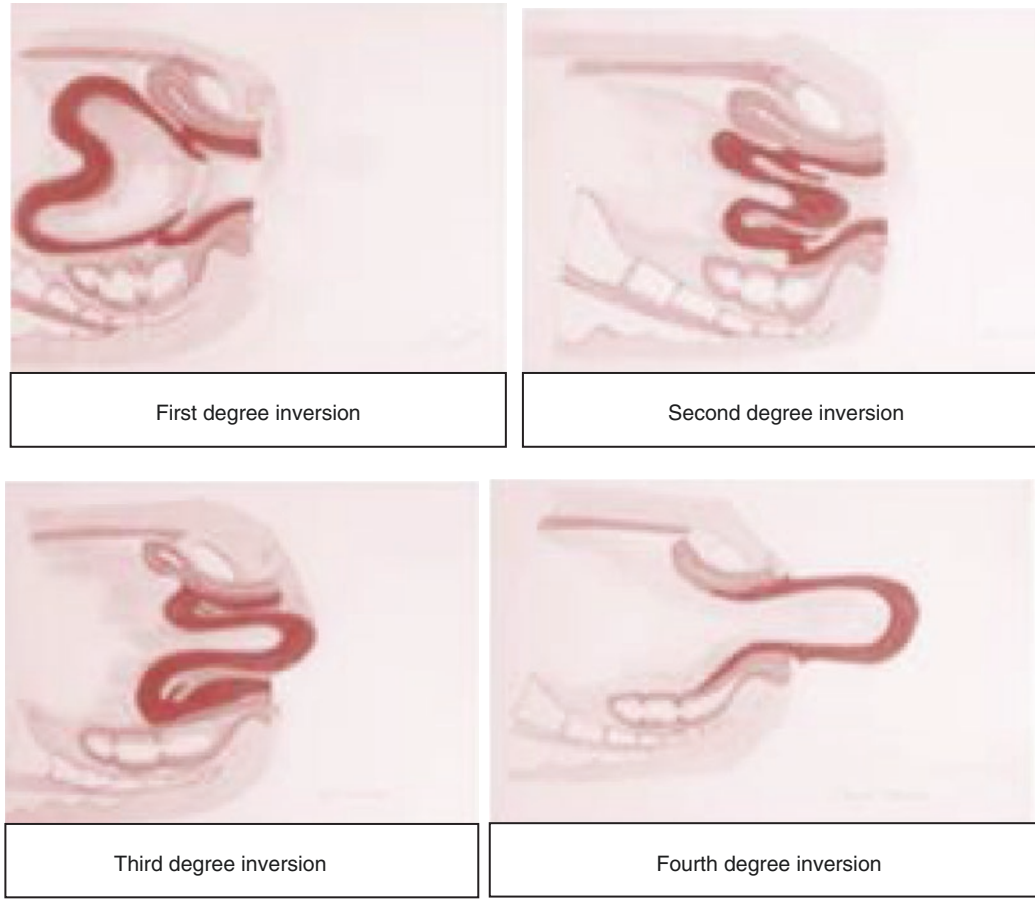

Fig. 39.2 Degrees of uterine inversion

The classification according to timing since delivery is as follows [4]:

- Acute inversion—occurs within 24 h of delivery and before contraction of the cervical ring
- Subacute inversion—presents from 24 h to 4 weeks after delivery and after contraction of the cervical ring
- Chronic inversion—presents after 4 weeks of delivery

Acute presentation is the most common, whereas chronic inversion is the rarest, making it a diagnostic dilemma [4].

39.3 Risk Factors and Pathogenesis

The pathogenesis of inversion of the puerperal uterus is not clearly known. However the factors which have been associated with this condition are as follows [5, 6].

39.3.1 Factors Related to Labor Management

Mismanaged third stage of labor has been considered historically as a very important pathogenetic mechanism in the causation of inversion

uterus. However a recently published RCT has shown inconsistent results between third stage management and inversion of the uterus [7]. The practices which have been associated with inversion of uterus are:

- Excessive cord traction especially on a fundal placenta
- Fundal pressure (Credé's maneuver) during the third stage of labor
- Rapid labor and delivery
- Use of tocolytic-relaxed uterus and lower segment

39.3.2 Placental Factors [5, 6]

- Fundal attachment of placenta
- Retained placenta
- Placenta accreta (especially if fundal)
- Short cord

39.3.3 Maternal Factors

- Anomalies of uterus
- Uterine fibroids
- Nulliparity

39.3.4 Fetal factors

- Macrosomia

39.4 Diagnosis

Clinical presentation is usually diagnostic, especially in the case of complete inversion. The postpartum patient usually presents with a smooth round mass protruding through the introitus and postpartum hemorrhage, which may be accompanied by shock. Since the shock is usually out of proportion to the blood loss, it is considered to be of neurogenic origin, chiefly due stretching of the parasympathetic nerves in the pelvis and increased vagal tone. However it is possible that the blood loss is underestimated as is often the case, and the pathogenesis of shock is hypovolaemia [8]. Other presenting features can be abdominal pain and sometimes retention of urine. Per abdomen examination reveals loss of the fundus in the periumbilical area. Vaginal examination confirms the presence of a firm globular mass protruding from the cervix. In case of prolapsed inversion, the inverted uterus will be seen lying outside the introitus.

The diagnosis of incomplete inversion is more challenging, as the bleeding and pain may be milder. The abdominal examination shows absence of the normal globular fundus of the uterus; instead the fundus may appear cupped. Examination through the dilated cervix reveals the fundus within the uterine cavity.

39.4.1 Role of Imaging Modalities

Imaging is not required normally for the diagnosis. In some cases where the differential diagnosis of a prolapsed fibroid is considered, an ultrasound scan may be useful. A homogenous mass in the uterine cavity and absence of fundus confirm the diagnosis. MRI is rarely required.

39.5 Management

The patient should be managed with great alacrity as delay has shown to increase the maternal morbidity and mortality.

39.5.1 Principles of Management

- Reposit the prolapsed uterus—with or without anesthesia
- Treat the shock/PPH
- Prevent reinversion

Repositioning the prolapsed uterus is to be done as soon as the diagnosis is made; delay may lead to formation of the cervical constriction ring, which makes the repositioning more difficult and may result in need for surgical intervention.

39.5.2 Manual Replacement

The steps of management of an acute case where manual replacement is possible:

- Call for help and mobilize the OT staff, anesthetist, and an experienced obstetrician.
- Rusticate the patient using the ABC approach. Insert two wide bore canulae, and draw blood for blood grouping, cross matching, hemogram, and coagulation study. Give boluses of crystalloid fluid till the patient stabilizes. Blood products are to be transfused if inversion is accompanied with PPH, which is often the case.
- Stop administration of any uterotonic drug as this will make repositioning more difficult.
- Do not try to remove the placenta if it is still attached to the uterus. This can lead to torrential bleeding and collapse of the patient [5].
- In case of bradycardia due to vagal stimulation, injection of atropine, 0.5 mg I/V, can be given.
- **Manual repositioning** should be attempted without any delay. The right hand of the obstetrician should be inserted along the axis of the vagina, and the fundus should be gently pushed in a direction toward the umbilicus (Fig. 39.3). This procedure is known as the **Johnson maneuver**.
- In case a cervical ring is felt, give pressure on the part of the uterus closest to the cervical ring, and then proceed upward, i.e., the portion which prolapsed last is reposited first and the part which prolapsed first (the fundus) is reposited in the end.
- If manual reposition fails due to constriction ring, uterine relaxants can be given and another attempt made. Any of the following relaxants can be given—nitroglycerine 50 μg IV, terbutaline 0.25 mg I/V or S/C and magnesium sulfate 4–6 g I/V over 20 min. Inhalational agents like isoflurane and halothane can also be given. These can only be administered in the operating room after securing the airway of the patient.
- An alternative to manual replacement is the **hydrostatic method** popularized by **O'Sullivan** and published in British Medical Journal in 1945. A pressure of 3–5 L of warm water held about a meter above the patient is allowed to flow into the vagina via tubing which is placed into the posterior fornix. The pressure of fluid is maintained with the palm of the surgeon and by the labial apposition around the surgeons palm by the assistant [9]. A modification of the procedure uses a vacuum cup to which the tubing is attached. The vacuum cup is placed in the vagina, and the seal between the cup and vagina prevents leakage of the fluid [10, 11] (Fig. 39.4).

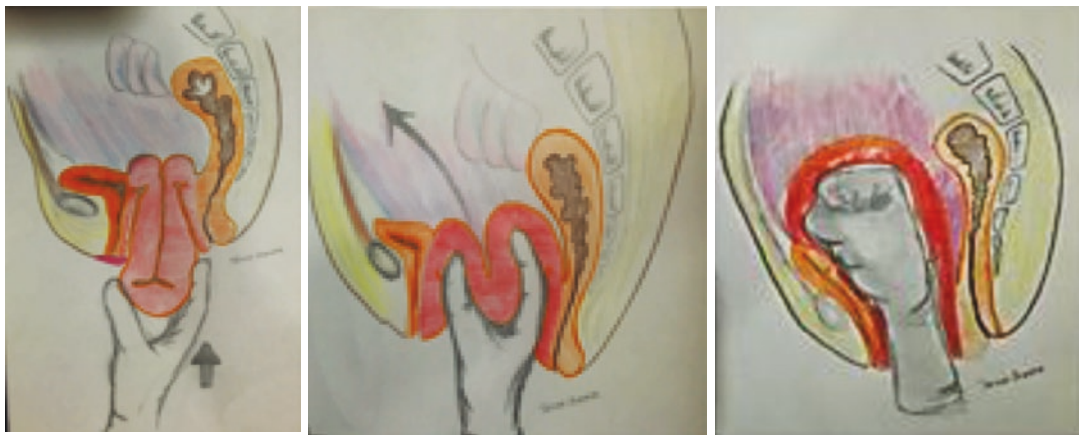

Fig. 39.3 The direction in which the inverted uterus is reposited

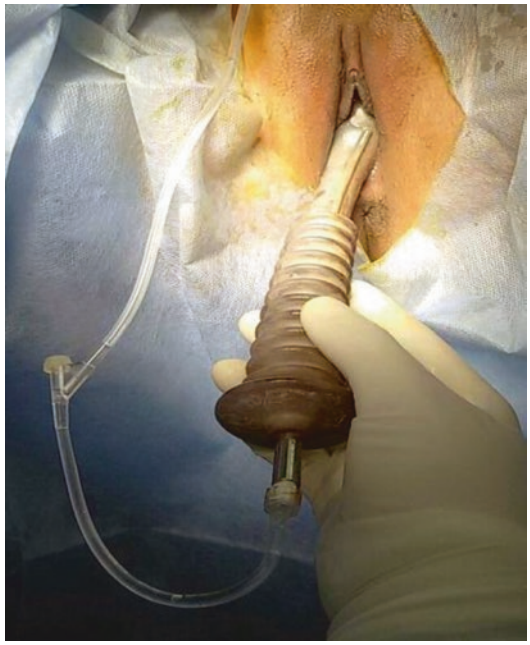

Fig. 39.4 Modification of the O'Sullivan hydrostatic method. Here a vacuum cup is to create a seal after placing the inverted uterus into the vagina. Note the fluid which is flowing under pressure through the vacuum cup

39.5.3 Surgical Methods

Surgical means are resorted to when manual reposition is not successful due to a tight constriction ring. The surgery may be conducted by the abdominal or the vaginal route.
Abdominal approach:

1. Huntington procedure—during laparotomy, the fundal structures are identified inside the cup of inversion. Babcock clamps are used to hold both the round ligaments. Progressive upward traction and reclamping are performed till the inversion is corrected. The whole process can be facilitated by pushing the fundus of the uterus vaginally by an assistant.
2. Haultain procedure—this procedure involves the bisection of the posterior aspect of the cervical constriction ring. Once the ring is released, a manual reposition or the Huntington procedure can be performed to bring the uterus back to its anatomical position followed by repair of the incision.

Vaginal approach:
Spinelli procedure—this approach is rarely used. The cervical constriction ring is incised anteriorly, and the manual replacement of the inverted uterus is done, followed by a repair of the incision. The risk of bladder injury limits the practice of this approach.

39.6 Interventions Post Correction

- If the placenta is still attached to the uterus, we can wait for its spontaneous separation. Alternatively manual removal can be done in the operation theater so that any complication can be handled immediately.
- It is important that the uterus should remain contracted after it is reposited back, to prevent PPH and reinversion. The fundus should be stabilized by the surgeon till it is firmly contracted and in position.
- 20–40 units of oxytocin in 1 L of crystalloid should be infused at the rate of 125 mL/h. Other oxytocics which can be given are 250 μg carboprost I/M, repeated 6 hourly for 24 h (contraindicated in respiratory disorders); misoprostol, 800 μg inserted in the vagina or rectum; and methylergonovine 200 μg I/M, 6 hourly for 24 h (contraindicated in uncontrolled hypertension).
- Broad-spectrum antibiotics should be administered to prevent endometritis and puerperal sepsis.

39.7 Recurrence of Inversion in Next Pregnancy

The risk of recurrence of inversion in the next pregnancy has not been studied extensively. The available literature does not indicate an increased risk for inversion in the subsequent pregnancies [2].

39.8 Summary and Key Points

- Inversion of the uterus is a catastrophic event which entails a high maternal mortality and morbidity due to massive postpartum hemorrhage and shock. It is therefore important to recognize and treat it with great alacrity.
- The uterine inversion can be classified according to the degree of inversion and the timing of presentation in relation to the delivery.
- The most commonly seen presentation is complete or prolapsed inversion of acute onset.
- Mismanaged third stage of labor and placental, maternal, and fetal factors are associated with the pathogenesis of uterine inversion.
- The principles of treatment include prompt repositioning of the uterus, management of shock and PPH, and prevention of reinversion.
- No attempt should be made to deliver the placenta before repositioning an inverted uterus as it can lead to torrential hemorrhage.
- Repositioning is best achieved by the Johnson maneuver if the cervical constriction ring has not formed. Uterine relaxants can be used as adjuvants for the procedure.
- Hydrostatic method and its modification using the vacuum cup also have good results and should be attempted before a surgical intervention.
- The surgical method which is preferred is the Huntington method as it does not involve giving an incision over the uterus. In case it is not successful, the Haultain method may be used. Vaginal surgical approach is rarely advocated.
- Post reposition of the uterus of the patient should be given uterotonics for at least 24 h to prevent reinversion.

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References

1. Mornini A, Angelini R, Giardini G. [Acute puerperal uterine inversion: a report of 3 cases and an analysis of 358 cases in the literature]. Minerva Ginecol. 1994;46:115.
2. Baskett TF. Acute uterine inversion: a review of 40 cases. J Obstet Gynaecol Can. 2002;24:953.
3. Pauleta JR, Rodrigues R, Melo MA, Graça LM. Ultrasonographic diagnosis of incomplete uterine inversion. Ultrasound Obstet Gynecol. 2010;36:260.
4. Livingston SL, Booker C, Kramer P, Dodson WC. Chronic uterine inversion at 14 weeks postpartum. Obstet Gynecol. 2007;109:555.
5. Witteveen T, van Stralen G, Zwart J, van Roosmalen J. Puerperal uterine inversion in the Netherlands: a nationwide cohort study. Acta Obstet Gynecol Scand. 2013;92:334.
6. Adesiyun AG. Septic postpartum uterine inversion. Singapore Med J. 2007;48:943.
7. Deneux-Tharaux C, Sentilhes L, Maillard F, et al. Effect of routine controlled cord traction as part of the active management of the third stage of labour on postpartum haemorrhage: multicentre randomised controlled trial (TRACOR). BMJ. 2013;346:f1541.
8. Beringer RM, Patteril M. Puerperal uterine inversion and shock. Br J Anaesth. 2004;92:439.
9. Momani AW, Hassan A. Treatment of puerperal uterine inversion by the hydrostatic method; reports of five cases. Eur J Obstet Gynecol Reprod Biol. 1989;32:281.
10. Ogueh O, Ayida G. Acute uterine inversion: a new technique of hydrostatic replacement. Br J Obstet Gynaecol. 1997;104:951.
11. Tan KH, Luddin NS. Hydrostatic reduction of acute uterine inversion. Int J Gynaecol Obstet. 2005;91:63.

Rupture Uterus

40

Rujuta Fuke

40.1 Introduction

Rupture uterus is an acute catastrophic event occurring during pregnancy and labor resulting in grave complications to mother and the baby constituting obstetric emergency. High degree of suspicion and clinical skills are required to tackle this complication; failure to do so causes high rate of stillbirths and maternal morbidity and even mortality. The signs and symptoms associated with this acute obstetric emergency are usually nonspecific, causing delay in diagnosis and initiation of definitive management. The short time for instituting definitive therapeutic action makes uterine rupture in pregnancy a much feared event for medical practitioners.

40.2 Definition

Rupture uterus is defined as disruption of all the layers of uterus including visceral peritoneum (serosa) after 28 weeks of gestation with or without the baby lying in the peritoneal cavity. At times it may cause massive hemorrhage from edges of the disruption leading to hemoperitoneum and shock.

R. Fuke (✉)
Department of Obstetrics and Gynecology, Government Medical College and Hospital, Nagpur, Maharashtra, India

40.3 Incidence

The incidence of uterine rupture differs in scarred and unscarred uterus, common in former than latter. The incidence of uterine rupture is approximately 1 in 1536 pregnancies (0.07%). In modern industrialized countries, the uterine rupture rate during pregnancy for a woman with a normal, unscarred uterus is 1 in 8434 pregnancies (0.012%) [1]. The incidence of scar rupture as found out in a retrospective analysis of 7 years study in a tertiary hospital in India was 1 in 1633 deliveries (0.061%) [2]. The prevalence more or less has remained constant over a period of time. Due to improved obstetric care in case of complicated obstructed labor and institutional deliveries, the rate of rupture uterus has decreased, but at the same time, there is increased incidence of uterine rupture in previous cesarean section cases.

40.4 Risk Factors

Following are the risk factors associated with rupture uterus:

1. Scarred uterus—Scarred uterus of previous cesarean section is more prone for rupture as uterus is actively contracting in postpartum period, resulting in weakening of the scar [3, 4]. The occurrence of scar rupture is determined by number of previous cesarean section, previous

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myomectomy, previous metroplasty, inter conceptional period and type of uterine incision like low transverse, low vertical, classical in upper segment, high transverse, T-shaped. Likewise, vaginal delivery after previous cesarean section, post operative period like postpartum sepsis or wound infection, or resuturing and obstetric complication in present pregnancy like grand multipara, elderly, big baby, abnormal presentation, multiple pregnancy, contracted pelvis, dystocia, abnormal placentation and accidental hemorrhage, use of oxytocics to augment or induce labor all determine the occurrence of the scar rupture.

2. Multiparity—There is thinning of uterine wall due to repeated childbirths, resulting in increased chances of spontaneous rupture [5]. If the pregnancy is complicated by previous scar, the chances of rupture uterus are doubled.
3. Maternal age—Advanced maternal age is directly proportional to the incidence of rupture uterus as often the baby is macrosomic causing uterine dystocia [6].
4. Placentation—Abnormal placentation like accrete, percreta, increta, previa, and abruption all weaken the uterine wall musculature and cause abnormal uterine activity and increased chances of uterine rupture.
5. Injudicious use of oxytocics—Oxytocics like oxytocin and prostaglandins when used inadvertently can lead to uterine rupture especially in grand multipara and previous cesarean section patients [7–10].
6. Multiple gestations—Overdistension due to multiple gestations or polyhydramnios in unscarred uterus is rarely responsible for uterine rupture, but when it is complicated by previous cesarean scar or pregnancy, complication mentioned earlier leads to increased chances of scar rupture. The ACOG 2010 guidelines for VBAC recommend that women with one previous cesarean delivery with a low transverse incision, who are otherwise appropriate candidates for twin vaginal delivery, may be considered candidates for TOLAC [11].
7. Uterine anomalies like bicornuate uterus (Fig. 40.1) [12].

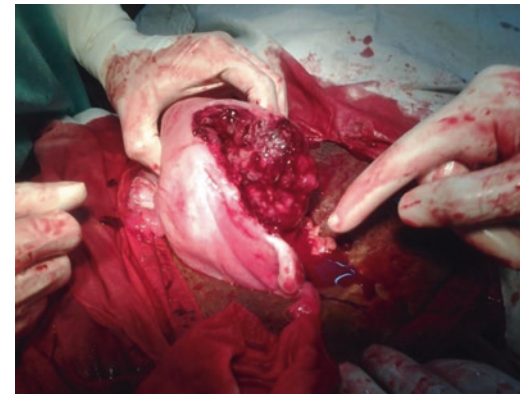


Fig. 40.1 Rupture of the uterus at fundus in bicornuate uterus. The fetus was lying in the peritoneal cavity with hemoperitoneum. Uterine repair was done to conserve future childbearing (Image is from personal collection of the author)

8. Dystocia and uncoordinated uterine action and hyperstimulation of uterus leading to obstructed labor can result in uterine rupture.
9. Trophoblastic invasion of the myometrium by weakening the uterine musculature.
10. Instrumentation like attempted forceps delivery through incompletely dilated cervix can cause uterine rupture.
11. Uterine manipulation like external cephalic version, internal podalic version, and manual removal of placenta all cause increased chances of uterine rupture in scarred and unscarred uterus.
12. Trauma to the uterus in the form of either direct blow to the abdomen or fall or accidents can result in traumatic rupture of the uterus.
13. Couvelaire uterus as in abruption of the placenta—The blood from concealed hemorrhage is accumulated in the uterine walls causing weakening of the musculature with abnormal uncoordinated uterine action.

40.4.1 Rupture of the Unscarred Uterus

Rupture of unscarred uterus also known as spontaneous rupture is usually an uncommon occurrence. A healthy uterus is unlikely to give way in labor

and less so in pregnancy. The causes are previous damage to the uterine wall due to previous dilatation and curettage or manual removal of the placenta, grand multipara due to thin uterine walls, congenital malformation of the uterus like bicornuate uterus, and weakening of walls in couvelaire changes of uterus in abruptio placenta. During labor, however, the causes of rupture of unscarred uterus include obstructed labor and grand multipara more so with the inadvertent use of oxytocics.

The rupture of the uterus is usually complete and involves the upper segment and occurs later in pregnancy. The rupture due to obstructed labor involves the thinned-out lower segment and usually extends from one lateral side of the uterus to the upper segment, whereas the nonobstructive rupture involves fundal region and usually complete.

40.4.2 Rupture of the Scarred Uterus

The rate of primary cesarean section is alarmingly increasing since the last two decades posing a major threat in rising incidence of scar rupture as the main reason of increased uterine rupture. The scar of myomectomy and metroplasty rarely gives way as these procedures are done in nonpregnant state and scar heals well due to uterine quiescence, and when they rupture, they give way in late third trimester or early in labor [13]. On the contrary, the scar of previous cesarean section and hysterotomy is of more concern especially on the latter.

The scar of classical cesarean section is likely to rupture in later months of pregnancy, whereas scar of lower-segment cesarean section gives way during labor predominantly. However, the scar of classical cesarean section, hysterotomy, and myomectomy is more at risk of rupture during labor. Rupture in previous low transverse uterine incision is increased by a subsequent trial of labor, subsequent augmentation of labor, subsequent induction of labor, the use of prostaglandins, inter-delivery interval (cesarean delivery and subsequent conception if it is <6 months), one layer versus two layer closure, more than one prior delivery, maternal age, fetal macrosomia, multiple gestations, and gestation beyond 40 weeks [14]. Previous successful vaginal delivery and subsequent successful VBAC have a protective association. Current ACOG guidelines discourage the use of prostaglandins to induce labor in most women with a previous cesarean delivery.

The incidence of scar rupture is 1–2% in lower-segment scar, whereas it is five to ten times more in classical cesarean section.

Other causes of uterine rupture are direct trauma to the uterus as in accidents of external blow or due to iatrogenic procedures like internal podalic version, instrumental deliveries, breech extraction through incompletely dilated cervix, manual removal of the placenta, injudicious administration of oxytocics, and the use of prostaglandins in previously scarred uterus (Fig. 40.1).

40.5 Pathology of Uterine Rupture

40.5.1 Types

1. **Incomplete rupture:** Incomplete rupture usually results from rupture of previous lower-segment uterine scar and may extend up to the cervix and fornix causing colporrhexis. It may result from upward extension of cervical tear during difficult instrumental delivery with or without formation of broad ligament hematoma. In incomplete rupture, the peritoneal layer is intact, and the placenta and fetus remain inside the uterine cavity, or part of the fetus may lie between the layers of broad ligament.
2. **Complete rupture:** Complete rupture occurs when the scar of classical cesarean section gives way in upper segment or in spontaneous rupture of obstructive or nonobstructive variety. In complete rupture, the peritoneal coat is not intact resulting in fetus with or without placenta escaping out of uterus and lying in peritoneal cavity. The uterus remains contracted without much of blood loss unless a major vessel is affected.
3. **Scar dehiscence:** Scar dehiscence occurs when part of the previous scar and not the entire length is disrupted. The fetal mem-

branes remain intact with minimal or no bleeding.

4. **Scar rupture:** Scar rupture occurs when the entire length of the scar along with membranes is disrupted. There is a varying amount of bleeding from the margins or its extensions.
   Although a uterine scar is a well-known risk factor for uterine rupture (most of which arise from prior cesarean delivery), the majority of events involving the disruption of uterine scars result in uterine scar dehiscence rather than uterine rupture. These two entities must be clearly distinguished, as the options for clinical management and the resulting clinical outcomes differ significantly.

40.5.2 Sites

The upper segment and fundus are involved in spontaneous nonobstructive rupture, whereas in obstructive type, the anterior lower segment is involved. The rupture occurs transversely extending into lateral surface of the uterus damaging major blood vessels. The margins are ragged and necrosed. Sometimes, in obstructive variety posterior wall rupture also occurs due to compression of the presenting part against sacral promontory for prolonged duration. This can extend downward to involve the cervix, vagina, and bladder. In case of rupture of previous scar, the rupture occurs at the site of the scar. The margins are cleaned and fibrosed with minimal bleeding. The rent at the lower segment can involve the lateral sides and cause damage to blood vessels resulting hemoperitoneum and hemorrhagic shock. In case of traumatic rupture because of destructive operations and instrumental deliveries, the rupture of uterus is same as that occurs in spontaneous obstructive variety but with much damage to surrounding structures and at times difficult to define anatomy for repair.

40.5.3 Clinical Manifestations of Uterine Rupture

There are no universal criteria to diagnose uterine rupture as clinical manifestations depend on the timing, site, and extent of uterine rupture. Familiarity with this entity and high index of suspicion are required to diagnose the condition. However, the following are some of the symptoms and signs that may lead one to suspect the diagnosis.

40.5.4 Symptoms

1. Abdominal pain—Patient usually complains of dull abdominal pain. It is difficult to notice as patient is already in labor pain. However, it is constant dull aching unlike episodic uterine contractions and confined to suprapubic region in previous cesarean section scars. It is accompanied by difficulty in passing urine and pain during micturition. The pain may become severe with the feeling of something giving way.
2. Vaginal bleeding—The pain is accompanied by slight vaginal bleeding.
3. Feeling of something giving way with acute pain—This usually marks the complete rupture. The onset may not be dramatic in case of previous lower-segment cesarean section scarred uterus and sometimes referred to as "silent rupture."
4. Fainting attack—Patient may have fainting attack and collapse because of hemorrhage and shock or vagal stimulation due to intense pain.
5. In case of traumatic rupture, event leading to rupture is usually evident like history of trauma, accident or operative vaginal delivery, destructive procedures, uterine manipulation, manual removal of placenta, etc.

40.5.5 Signs

1. Tachycardia—It is usually the first sign of impending scar rupture.
2. Hypotension—It occurs secondary to hypovolemic shock due to intraperitoneal bleeding.
3. Suprapubic tenderness—It is palpable over previous scar site and is the sign of scar dehiscence.

4. Hematuria—It may be present especially in obstructed labor or rupture involving the urinary bladder.
5. Signs of obstructed labor—Exhaustion, dehydration, tachycardia, increased temperature, distended tender lower segment, Bandl's ring, dry edematous vagina, fetal distress, or absent fetal heart sounds.
6. Non-reassuring fetal heart rate pattern—Fetal heart rate variability and persistent, prolonged fetal bradycardia are pathognomonic of scar dehiscence and scar rupture [15].
7. Intrauterine fetal death—If definitive surgical intervention is not undertaken in crucial period of 35 min, it can result in fetal demise.
8. Palpation of fetal parts superficially—Fetal parts are palpable superficially in case of complete rupture.
9. Cessation of uterine contractions—With complete rupture, uterine contractions cease and pain diminishes.
10. Loss of uterine contour—It may be accompanied by loss of ovoid uterine contour.
11. Shock and signs of intraperitoneal bleeding—It occurs depending on the site, type, and extent of uterine rupture.
12. Recession of fetal parts on per vaginal examination—Loss of station of presenting part should raise a suspicion of uterine rupture because of the withdrawing of fetal parts in the abdominal cavity.
13. In case of rupture following instrumental delivery, exploration of uterus to feel the rent confirms the diagnosis. Shortening of the cord immediately following a difficult vaginal delivery is also one indication of uterine rupture.

40.5.6 Consequences of Uterine Rupture [16]

1. Fetal: fetal hypoxia/anoxia, fetal acidosis, admission to neonatal intensive care unit, and fetal/neonatal death.
2. Maternal: severe maternal blood loss and anemia, hypovolemic shock, maternal bladder injury, obstetric hysterectomy, and maternal death.

40.6 Management of Ruptured Uterus

The most critical aspects of treatment in the case of uterine rupture are establishing a timely diagnosis and minimizing the time from the onset of signs and symptoms until the start of definitive surgical therapy. Once a diagnosis of uterine rupture is established, the immediate stabilization of the mother and the delivery of the fetus are imperative.

As a rule, the time available for successful intervention after frank uterine rupture and before the onset of major fetal morbidity is only 10–37 min [17]. Therefore, once the diagnosis of uterine rupture is considered, all available resources must quickly and effectively be mobilized to successfully institute a timely surgical treatment that results in favorable outcomes for both the newborn and the mother. The treatment is based on two principles, resuscitation followed by laparotomy and sometimes both simultaneously.

1. Call for help—The juniors working in labor room (LR) should immediately notify the senior about the incidence and ask for help from rest of the LR staff.
2. Quick history and examination with monitoring of vitals.
3. Secure an intravenous line—This is utmost important before the patient goes in hypovolemic shock with collapsed veins preferably with wide bore cannula.
4. Oxygen by ventimask—To increase the oxygen saturation of the mother in hemorrhagic shock and prevent fetal anoxia.
5. Blood for grouping and cross matching—The blood should be withdrawn from the same wide gauge cannula for IV line, and adequate blood should be arranged for transfusion.
6. Treatment of hypovolemic shock—By means of IV fluids and plasma volume, expanders should be initiated.

Definitive surgical management: The definitive management is surgical. Urgent laparotomy is needed. It is better to call a general surgeon if need arises or if we are suspecting trauma to the blad-

der. The fetus may be in the uterus in partial rupture or may be in the peritoneal cavity along with the placenta. In that case, it is more likely to be dead. The fetus is extracted by entering the abdomen quickly by midline infraumbilical incision.

After the fetus is successfully delivered, the type of surgical treatment for the mother depends on the factors like type and extent of uterine rupture, amount of bleeding, general condition of the mother, and desire for future childbearing.

Uterine bleeding is typically most profuse when the uterine tear is longitudinal rather than transverse.

Conservative surgical management involving uterine repair should be reserved for women who have the following findings:

1. Desire for future childbearing.
2. Low transverse uterine rupture.
3. No extension of the tear to the broad ligament, cervix, or paracolpos.
4. Easily controllable uterine hemorrhage.
5. Good general condition.
6. No clinical or laboratory evidence of coagulopathy.

Repair is done usually in previously scarred uterus when the margins of the defect are clean and regular and bleeding less. It is done by excising the fibrous tissue at the margins. Sometimes, rupture in unscarred uterus has to be repaired in view of future childbearing, but in that case chances of peritonitis, septicemia, and rupture uterus in next pregnancy are very common. Repair if done is usually combined with sterilization operation in patients with completed childbearing.

Hysterectomy should be considered the treatment of choice when intractable uterine bleeding occurs or when the uterine rupture sites are multiple, longitudinal, or low lying. Usually because of poor general condition of patient, a quick subtotal hysterectomy is done, thus minimizing operative time and injury to the bladder and ureters. However if condition permits then a total hysterectomy may be done. In case of broad ligament hematoma, the anterior leaf of broad ligament is opened, hematoma is drained, bleeding points are secured, and anterior leaf is sutured. In case of failure to secure bleeding points, anterior division of anterior iliac artery is ligated. Care is taken not to injure ureters at any point of time.

Because of the short time available for successful intervention, the following two premises should always be kept firmly in mind: (1) maintain a suitably high level of suspicion regarding a potential diagnosis of uterine rupture, especially in high-risk patients, and (2) when in doubt, act quickly and definitively.

Prevention: The following measures are to be adopted to prevent catastrophic uterine rupture in pregnancy and labor.

1. Identification of high-risk cases like history of previous cesarean section, myomectomy, hysterotomy, abnormal presentation, contracted pelvis, big baby, and grand multipara to provide quality antenatal care and education to them.
2. Proper documentation of operative procedures like hysterotomy, myomectomy, or classical or t-shaped uterine scar in the discharge card so that in next pregnancy these patients are identified and labeled as high risk.
3. Encouraging institutional deliveries of all patients, high risk in particular.
4. Indoor admission at 36 weeks in patients with history of previous myomectomy, classical cesarean section, or hysterotomy.
5. Strict vigilance during labor in these high-risk patients.
6. No undue force to be used in external cephalic version, and there is no role of internal podalic version in modern obstetrics.
7. Proper selection of cases for vaginal birth after cesarean section (VBAC).
8. Judicious use of oxytocics.
9. Prompt measures to tackle prolonged and obstructed labor.
10. Attempted forceps delivery and breech extraction through incompletely dilated cervix are to be avoided.
11. The cervix and uterus are to be explored after instrumental delivery and destructive procedures.
12. Manual removal of the placenta in morbidly adherent placenta should be attempted by senior experienced obstetricians.

13. Labor room should be well equipped to tackle any obstetric emergency preferably with attached operation theater.
14. Acute obstetric emergency drills should be practiced by labor room team to face such emergencies.

40.6.1 Prognosis

Prognosis depends on many factors like etiology, site of rupture, preceding events, maternal general condition, early diagnosis, and quick intervention. Lower scar rupture has better prognosis than spontaneous obstructed variety with high maternal and fetal death rate.

40.7 Conclusion

Rupture uterus constitutes one of the many obstetric emergencies that are frightening to the labor room residents. Rupture of the uterus can be spontaneous, scar rupture, or iatrogenic. Rupture of the uterus may occur either during pregnancy or during labor. The diagnosis of rupture uterus is difficult. Strict vigilance on the signs and symptoms and high index of suspicion are necessary to diagnose the entity. An abnormal CTG should not be overlooked especially in high-risk patients for scar rupture. Management is centered on prompt measures involving senior or experienced consultants. It involves immediate resuscitation and laparotomy often resulting in obstetric hysterectomy. Repair may be done in cases where margins are clean. Repair and permanent sterilization is done when the woman has completed her family to avoid the future catastrophes. Obstetric emergency drill training should be regularly practiced by the labor room team to face such emergencies.

References

1. Gardeil F, Daly S, Turner MJ. Uterine rupture in pregnancy reviewed. Eur J Obstet Gynecol Reprod Biol. 1994;56(2):107–10.
2. Sinha M, Gupta R, Gupta P, Rani R, Kaur R, Singh R. Uterine rupture: a seven year review at a Tertiary Care Hospital in New Delhi, India. Indian J Community Med. 2016;41(1):45–9. https://doi.org/10.4103/0970-0218.170966.
3. Mozurkewich EL, Hutton EK. Elective repeat cesarean delivery versus trial of labor: a meta-analysis of the literature from 1989 to 1999. Am J Obstet Gynecol. 2000;183(5):1187–97.
4. Rosen MG, Dickinson JC, Westhoff CL. Vaginal birth after cesarean: a meta-analysis of morbidity and mortality. Obstet Gynecol. 1991;77(3):465–70.
5. Golan A, Sandbank O, Rubin A. Rupture of the pregnant uterus. Obstet Gynecol. 1980;56(5):549–54.
6. Shipp TD, Zelop C, Repke JT, et al. The association of maternal age and symptomatic uterine rupture during a trial of labor after prior cesarean delivery. Obstet Gynecol. 2002;99(4):585–8.
7. Mokgokong ET, Marivate M. Treatment of the ruptured uterus. S Afr Med J. 1976;50(41):1621–4.
8. Rahman J, Al-Sibai MH, Rahman MS. Rupture of the uterus in labor. A review of 96 cases. Acta Obstet Gynecol Scand. 1985;64(4):311–5.
9. Ravasia DJ, Wood SL, Pollard JK. Uterine rupture during induced trial of labor among women with previous cesarean delivery. Am J Obstet Gynecol. 2000;183(5):1176–9.
10. Blanchette H, Blanchette M, McCabe J, Vincent S. Is vaginal birth after cesarean safe? Experience at a community hospital. Am J Obstet Gynecol. 2001;184(7):1478–84; discussion 1484–7.
11. Practice bulletin no ACOG. 115: Vaginal birth after previous cesarean delivery. Obstet Gynecol. 2010;116(2 Pt 1):450–63.
12. Nahum GG. Uterine anomalies, induction of labor, and uterine rupture. Obstet Gynecol. 2005;106(5):1150–2.
13. Sizzi O, Rossetti A, Malzoni M, Minelli L, La Grotta F, Soranna L. Italian multicenter study on complications of laparoscopic myomectomy. J Minim Invasive Gynecol. 2007;14(4):453–62.
14. Leung AS, Farmer RM, Leung EK, Medearis AL, Paul RH. Risk factors associated with uterine rupture during trial of labor after cesarean delivery: a case-control study. Am J Obstet Gynecol. 1993;168(5):1358–63.
15. Andersen MM, Thisted DL, Amer-Wåhlin I, Krebs L, Danish CTG Monitoring During VBAC Study Group. Can intrapartum cardiotocography predict uterine rupture among women with prior caesarean delivery? A population based case-control study. PLoS One. 2016;11(2):e0146347. https://doi.org/10.1371/journal.pone.0146347. eCollection 2016.
16. Leung AS, Leung EK, Paul RH. Uterine rupture after previous cesarean delivery: maternal and fetal consequences. Am J Obstet Gynecol. 1993;169(4):945–50.
17. Bujold E, Gauthier RJ. Neonatal morbidity associated with uterine rupture: what are the risk factors? Am J Obstet Gynecol. 2002;186(2):311–4.

Broad Ligament Haematoma

41

Vaishali Korde-Nayak and Parag Biniwale

41.1 Introduction

During pregnancy the uterus, vagina and vulva have rich vascular supplies. Any significant trauma during birth process may result in formation of a haematoma. Puerperal genital haematomas are relatively uncommon causes of PPH but can lead to serious morbidity and even maternal death [1]. The reported incidence of significant postpartum haematoma is around 1 in 500–700 deliveries [2].

Supralevator haematomas also known as broad ligament haematomas are rare, with widely varying incidence of between 1:500 and 1:20,000 deliveries [3]. As the symptoms are nonspecific and size dependent and bleeding is often concealed, most of them are difficult to diagnose.

Whitridge Williams was the first person to report broad ligament haematoma in 1904 [4]. He reported a series of cases in a monograph on subperitoneal haematoma. He studied 33 cases of spontaneous broad ligament haematomas and ascribed them to capillary bleeding [4] (Fig. 41.1).

V. Korde-Nayak (✉)
MIMER Medical College, Pune, Maharashtra, India

P. Biniwale
Biniwale Clinic, Pune, Maharashtra, India

41.2 Types of Puerperal Genital Haematomas

1. **Infralevator Haematomas**
   (a) Location—Below the levator ani muscle
   (b) Include the vulva, perineum and lower vagina, episiotomy site
   (c) More common than supralevator variety
   (d) Occur following vaginal birth/instrumental deliveries/big baby/any other obstetric trauma
2. **Supralevator Haematomas**
   (a) Location—Above the levator ani muscle, in the leaves of broad ligament
   (b) Mostly due to an extension of cervical tear, forniceal tear or uterine incision and uterine rupture
   (c) Less common than infralevator haematomas
   (d) Can be associated with spontaneous vaginal birth, but commonly occur following instrumental vaginal deliveries, difficult caesarean section or vaginal birth after caesarean (VBAC), etc.

Broad ligament haematoma occurs secondary to lacerations/tear in the upper vagina, cervix or uterus that extends into uterine or vaginal vessels or vessels of the broad ligament. The engorged vessels of pregnancy bleed profusely in the space between the leaves of broad ligament accommodating significant blood collection.

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_41

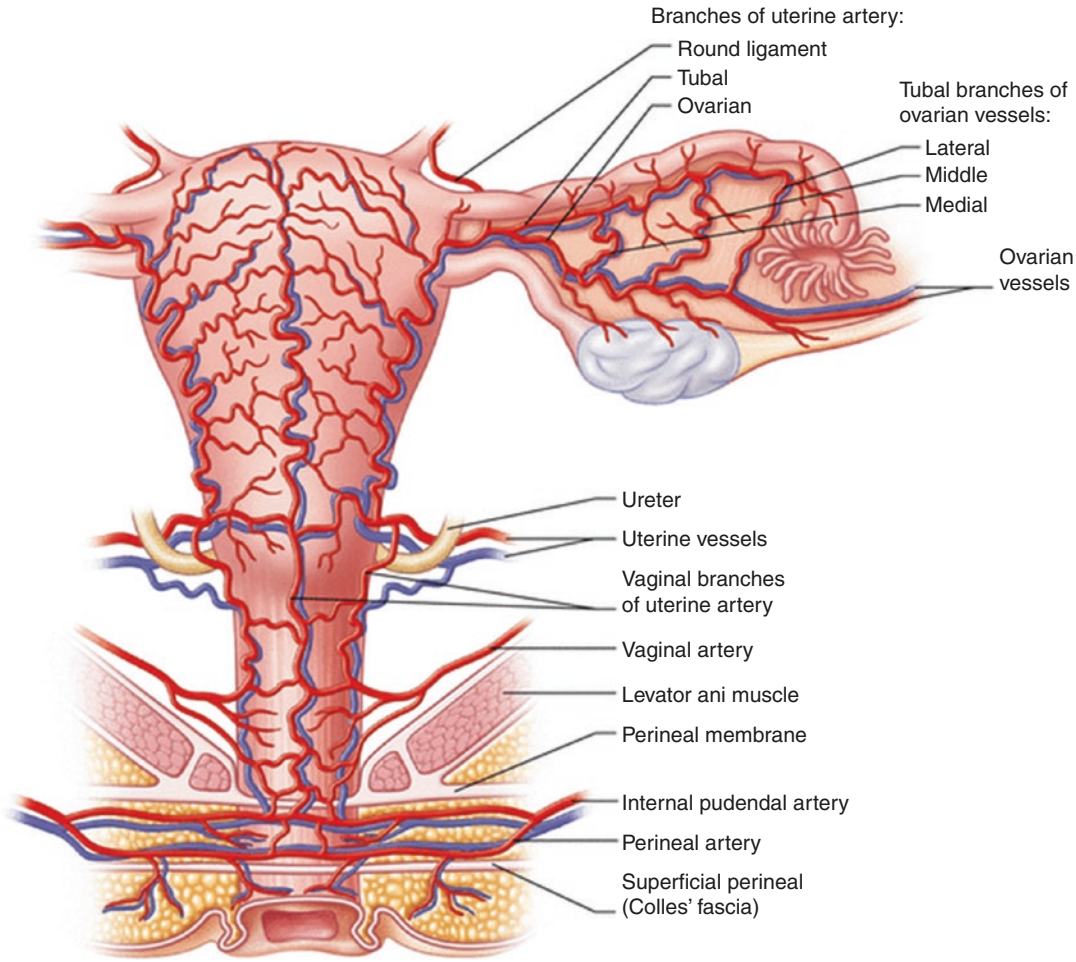


Fig. 41.1 Blood supply of the female genital tract [5]

This is mainly seen in:

- Extension of cervical tear or primary colporrhexis (vault rupture)
- LSCS scar rupture
- Spontaneous rupture of paravaginal venous plexus adjacent to the vault

The diagnosis is usually delayed, as there are no obvious symptoms like pain or vaginal bleeding**.** Sometimes unexplained shock with features suggestive of internal haemorrhage immediately following delivery raises the suspicion. Maternal mortality chances are high if the diagnosis is delayed. So strong suspicion, good clinical examination and prompt treatment are the key words in the management.

Broad ligament haematoma can occur with the following obstetric incidences

- Spontaneous vaginal birth
- Episiotomy
- Caesarean section
- Precipitate labour
- Instrumental vaginal delivery (commonly forceps)

- Assisted breech delivery
- Twins
- Prolonged second stage of labour
- Big baby
- Vaginal birth after caesarean (VBAC)
- Hereditary clotting deficiencies
- Vulvar varicosities
- Pre-eclampsia [6, 7]

Fifty percent of these haematomas are diagnosed immediately, while the other 50% are discovered within 24 h [7]. The arterial origin haematomas are rapidly expanding and appear bright or dark red, while the slow expanding haematomas are of venous origin and appear dark red or bluish in colour [7]. It may be concealed with no obvious vaginal bleeding.

41.2.1 Symptomatology

Symptoms can be quite vague and usually develop within a few hours of delivery. They depend upon the size of the haematoma (amount of blood loss), the rate at which its formation occurs and the type of haematoma.

Haematomas are usually diagnosed when patient presents with haemorrhagic shock or even death, due to its insidious nature [7]. The speed of diagnosis depends on the extent of the bleeding, its associated consequences and the level of awareness and suspicion of the medical staff.

41.2.2 Clinical Presentation

- Features of hypotensive shock.
- Disproportionate pallor to the visible blood loss.
- Persistent throbbing pelvic pain/back pain may be there.
- Inability to pass urine.
- Pressure in the recto-anal area/fullness.
- An urge to push within the first few hours after delivery.
- Headache, dizziness, restlessness.

A broad ligament haematoma can present with abdominal pain, but often it first presents with signs of hypovolaemia, including cardiovascular collapse.

General Exam—signs of shock, e.g. pallor, sweaty, cold, clammy, dizzy, elevated pulse, decreased blood pressure.

P/A Exam

- A swelling/mass may be felt just above the inguinal ligament.
- The uterus gets pushed upward/laterally to the opposite side of the broad ligament haematoma.

P/V Exam

- Occlusion of the vaginal canal by a bulge
- A boggy mass/swelling felt through the fornix

P/R Exam—Presence of the soft boggy mass.

41.2.3 Differential Diagnosis

- Intraperitoneal haemorrhage
- Rectus sheath haematoma
- Ovarian mass/pelvic mass
- An abscess

41.3 Investigations

41.3.1 Blood Tests

Sudden fall in haematocrit levels is suggestive of internal haemorrhage. A full blood count and coagulation screen are most important to determine baseline values. Crossmatching and replacing adequate blood units as per the clinical picture is crucial.

41.3.2 Imaging

- USG
- CT scan
- MRI

Radiological investigations are not only useful for diagnosis but also to assess the volume of blood loss and any extension into the pelvis. They also help in monitoring progress or resolution of the haematoma.

MRI is the most precise investigation to define the exact location, size and extent of a haematoma. It can also help to differentiate between a pelvic mass, such as an abscess or endometrioma. But USG is the most commonly available, non-invasive investigation with good sensitivity. In emergency, bedside USG is the boon.

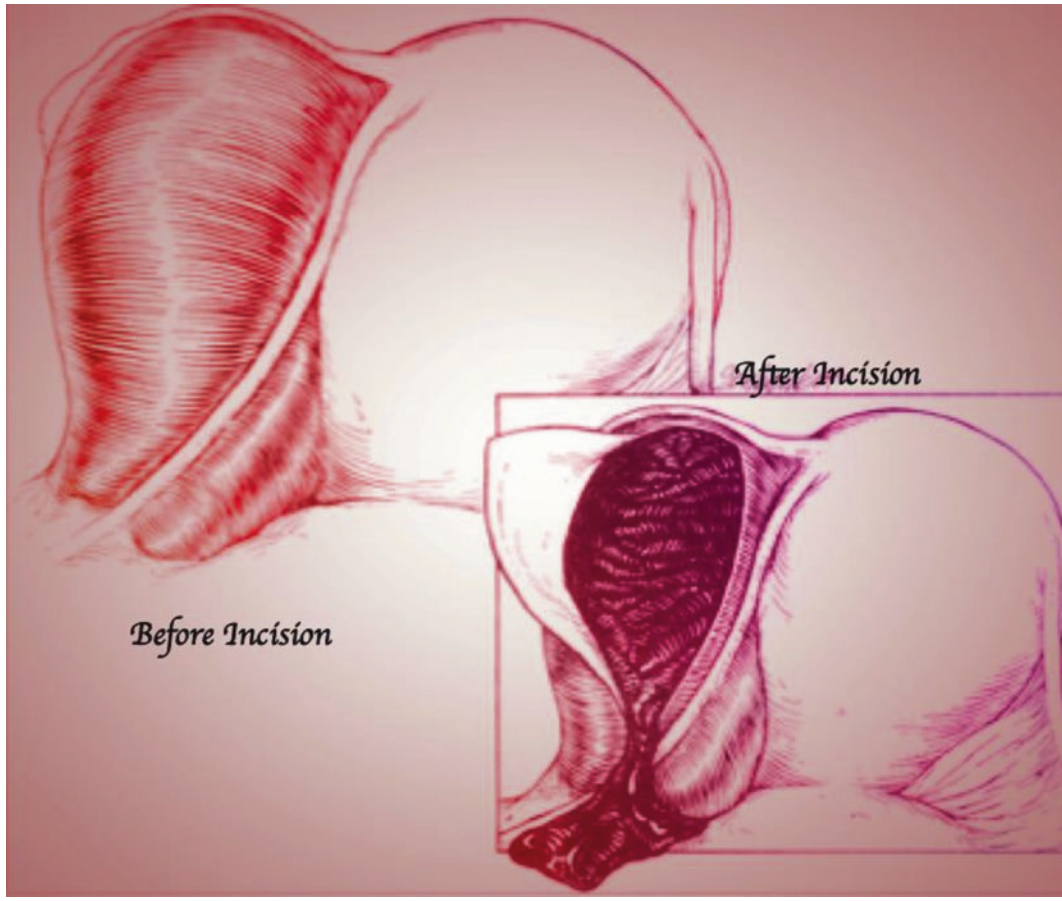
41.4 Management

41.4.1 Resuscitation

- Assess approximate blood loss.
- In case of hypovolemic shock, immediate resuscitative measures are the first line of treatment, like wide bore IV cannula insertion bilaterally and aggressive IV fluid replacement with crystalloids/colloids (e.g. Hartmann's, sodium chloride 0.9%).
- Blood samples for haematocrit, coagulation profile and crossmatch. Replace adequate blood units.
- Indwelling urinary catheter to monitor urinary output, to maintain fluid balance and also to tackle urinary retention resulting from pain and/or the pressure of haematoma.
- The aim of treatment is to stop further blood loss, minimize tissue damage and relieve pain.
- Broad-spectrum IV antibiotics cover to reduce the risk of infection of the haematoma.
- In case of conservative management, continuous monitoring is required to ensure that the haematoma is not increasing in size further.

41.4.2 Definitive Treatment

- Definitive treatment varies depending on size of hematoma, its location and haemodynamic stability of patient.
- Small non-spreading broad ligament haematomas are self-limiting and can be managed conservatively. Radiological confirmation is required of resolving haematoma.
- Larger and expanding subperitoneal haematomas require emergency exploration. The anterior leaf of the broad ligament (peritoneum) is to be incised, and the blood clots are to be scooped out.
- The bleeding points, if visible, are to be secured and ligated.
- Random blind sutures should not be placed to prevent ureteric damage.
- If the oozing continues, bilateral internal iliac artery ligation should be considered. Packing can also be considered.
- The presence of associated scar dehiscence/rupture uterus may modify the treatment.
- Abdominal drain is useful to assess the post-operative loss.
- As recurrence is common, strict vigilance after primary repair/packing is required.



Broad ligament haematoma [8].

41.4.3 Recurrence

Recurrence is known for haematomas; so continuous monitoring of the vitals is very crucial postoperatively. Haematomas can recur after surgical management. Continued monitoring for signs of blood loss is essential. Replacement of lost blood units is equally important for good haemostasis. If it recurs, further surgical exploration along with bilateral internal iliac ligation or more radical surgery like hysterectomy may be necessary [8].

41.4.4 Newer Modalities

41.4.4.1 Pelvic Arteriography and Arterial Embolization

Recently under radiological guidance, pelvic arteriography and arterial embolization have been found effective to control postpartum haemorrhage and haematomas [9–11]. The success rate in controlling bleeding from haematomas is over 90% in the recent studies published [9–11].

Through the femoral artery, pelvic vasculature is accessed; bleeding vessel-showing extravasation of dye is identified and selectively embolized. Different embolic agents—temporary like absorbable gelfoam or permanent like metal coils—are used [8]. Temporary embolic agents have less risk of the ischemic problems [12]. The duration of occlusion is usually 2–3 weeks, with good revascularization later on.

Embolization is a good conservative option to preserve fertility and menstrual function [12]. Brown et al. in 1979 reported the first successful case of embolization for an intractable haematoma [13]. The patient continued to bleed despite abdominal hysterectomy and bilateral internal artery ligation [13]. Even though not many studies have been published since then, all have reported effective control of the bleeding and acceptable short-term results [14–16].

Previous studies suggest that selective embolization should be used as the first-line treatment for haematomas and persistent PPH. Bienstman-Pailleux et al. [17] have suggested an algorithm by which we should first consider pelvic arterial embolization and then the conventional surgeries.

41.4.4.2 Advantages of Selective Embolization

- It avoids the risk of exploratory laparotomy (and hysterectomy).
- The option of surgery can be considered only in case the interventional procedure fails.
- Attempting embolization, after surgical internal iliac ligation, increases the difficulty of the procedure.

41.4.4.3 Complications

- Accidental embolization of peripheral vessels leading to serious ischemic complications (usually collateral circulation protects).
- Vessel perforation [12].
- Allergy to contrast.
- Low-grade fever [12, 18].
- Site specific/groin haematoma [12].
- Post embolization pain/pain in buttock (ischemic) [12].
- Pelvic infections [12, 18].
- Ovarian failure, though rare, has been reported [18].

41.4.4.4 Limitations of Selective Embolization

- Non-availability of facilities for interventional radiology universally.
- Non-availability of expertise, especially during emergency and in vicinity of the labour room [19, 20].
- Patient needs to be haemodynamically stable [19, 20].
- The time consumed during procedure is around 1–2 h.

41.4.5 Postpartum Care

- Monitor vitals and vigilance for recurrence of haematoma.
- Effective analgesia with paracetamol/NSAID (NSAID are contraindicated in PPH, severe preeclampsia, renal disorders).
- No rectal administration of analgesics.
- Indwelling catheter until haemodynamically stable.
- Continue broad-spectrum antibiotic cover.

41.5 Conclusion

Broad ligament haematomas are less common but can be associated with serious maternal morbidity and mortality.

The common presenting symptom may be hypovolemic shock in absence of significant pain in abdomen or vaginal bleeding. As enormous amount of blood can get accumulated in broad ligament without distending the sensitive labia and perineum, so they may remain painless, while making the patient haemodynamically unstable.

The primary aims of treatment are to prevent further blood loss and correct hypovolemia. The optimal management of genital haematoma is still debatable. The treatment is variable depending on the size, location of haematoma and

haemodynamic stability of patient. In situations where facilities for selective arterial embolization are not available, conventional exploration including incision, evacuation and drainage of haematoma, ligation of bleeding vessels whenever possible and vaginal packing remains the gold standard. Replacement of adequate blood volume and coagulation factors is crucial for complete haemostasis.

Ligation is not always effective in the treatment of pelvic haemorrhage [21].

Thus selective embolization can act as both a diagnostic and therapeutic tool. Bleeding sites can be identified and embolization can be done using a gelfoam material. Usually the occlusion remains for 2–3 weeks, with good revascularization later on [21].

Selective angiographic embolization, performed by an experienced and well-trained radiology team, carries minimal risks. So, whenever available, it should be used as the first line of treatment. A multicentre randomized controlled trial is warranted to compare and clarify its role to conventional and conservative therapy [22].

41.5.1 Relative Ethical Issues

- Dilemma while choosing the most appropriate treatment when research on the condition is scarce?
- Would interventional radiology be cost-effective in treating PPH and haematomas?

Ref.: Puerperal genital haematomas, South Australian Perinatal Practice Guidelines [23].

Overview of Management

- Assess the haemodynamic condition.
- Prompt examination of the vulva, perineum and vagina followed by abdominal and rectal examination to identify site of haematoma.
- Estimate blood loss, and monitor ongoing blood loss.
- Resuscitation measures—intravenous fluid replacement with crystalloids/colloids (e.g. Hartmann's, sodium chloride 0.9%, ±blood transfusion).
- Urgent haematocrit, coagulation profile.
- Indwelling catheter to avoid possible urinary retention and fluid balance.
- Blood and components transfusion as per requirement.
- Offer analgesia (oral or intramuscular opioid).
- USG to confirm the diagnosis, assess volume and determine whether it is still expanding.
- In huge/expanding hematomas, consider surgical evacuation of clot to prevent pressure necrosis, septicaemia and haemorrhage and packing the cavity for 24 h.
- Consider internal iliac artery ligation if there is intractable bleeding.
- Consult an interventional radiologist, if available, to consider occlusion of the internal iliac artery/arteries by balloon catheter or embolization as an alternative to laparotomy for internal iliac artery ligation.

References

1. Fliegner JR. Postpartum broad ligament haematomas. J Obstet Gynaecol Br Commonw. 1971;78:184–9.
2. Hankins G, Zahn C. Puerperal haematomas and lower genital tract lacerations. In: Hankins G, et al., editors. Operative obstetrics. Norwalk, CT: Appleton and Lange; 1995. p. 57–72.
3. Cheung TH, Chang A. Puerperal haematomas. Asia-Oceania J Obstet Gynaecol. 1991;17:119–23.
4. Williams JW. Subperitoneal hematoma following labor not associated with lesions of the uterus. Trans Am Gynecol Soc. 1904;29:186.
5. Addo V, Kokroe FA, Reindorf RL. Broad ligament haematoma following a Snake Bite. Ghana Med J. 2009;43:181–2.
6. Mirza FG, Gaddipati S. Obstetric emergencies. Semin Perinatol. 2009;33:97–103.

7. Edmonds K. Dewhurst's textbook of obstetrics and gynecology. 6th ed. London: Blackwell Science; 1999.
8. Mawhinney S, Holman R. Practice points puerperal genital haematoma: a commonly missed diagnosis. Obstet Gynaecol. 2007;9:195–200.
9. Bloom AI, Verstandig A, Gielchinsky Y, Nadiari M, Elchalal U. Arterial embolisation for persistent primary postpartum haemorrhage: before or after hysterectomy? BJOG. 2004;111:880.
10. Badawy SZA, Etman A, Singh M, Murphy K, Mayelli T, Philadelphia M. Uterine artery embolization: the role in obstetrics and gynecology. Clin Imaging. 2001;25:288–95.
11. Dildy GA 3rd. Postpartum haemorrhage: new management options. Clin Obstet Gynecol. 2002;45:330–44.
12. Salomon LJ, deTayrac R, Castaigne-Meary V, Audibert F, Musset D, Ciorascu R, et al. Fertility and pregnancy outcome following pelvic arterial embolization for severe post-partum haemorrhage. A cohort study. Hum Reprod. 2003;18:849–52.
13. Brown BJ, Heaston D, Poulson AM. Uncontrollable postpartum bleeding: a new approach to haemostasis through angiographic arterial embolization. Obstet Gynecol. 1979;54(3):361–5.
14. Heffner LJ, Mennuti MT, Rudoff JC, McLean GK. Primary management of postpartum vulvovaginal haematomas by angiographic embolization. Am J Perinatol. 1985;2(3):204–7.
15. Chin HG, Scott DR, Resnik R, Davis GB, Lurie AL. Angiographic embolization of intractable puerperal haematomas. Am J Obstet Gynecol. 1989;160(2):434–8.
16. Villella J, Garry D, Levine G, Glanz S, Figueroa R, Maulik D. Postpartum angiographic embolization for vulvovaginal haematoma: a report of two cases. J Reprod Med Obstet. 2001;46:65–7.
17. Bienstman-Pailleux J, Huissoud C, Dubernard G, Rudigoz RC. Management of puerperal haematomas. J Gynecol Obstet Biol Reprod (Paris). 2009;38:203–8.
18. Goodwin SC, Walker WJ. Uterine artery embolization for the treatment of uterine fibroids. Curr Opin Obstet Gynecol. 1998;10(4):315–20.
19. Doumouchtsis SK, Papageorghiou AT, Arulkumaran S. Systematic review of conservative management of postpartum haemorrhage: what to do when medical treatment fails. Obstet Gynecol Surv. 2007;62:540–7.
20. Sentilhes L, Gromez A, Clavier E, Resch B, Verspyck E, Marpeau L. Predictors of failed pelvic arterial embolization for severe postpartum haemorrhage. Obstet Gynecol. 2009;113:992–9.
21. Dahdouh EM, Balayla J, Dube J. Angiographic embolization of a postpartum vulvovaginal haematoma in a patient with situs inversus totalis: an effective second line treatment. Case Rep Obstet Gynecol. 2013;2013:323781.
22. Benrubi G, Neuman C, Nuss RC, Thompson RJ. Vulvar and vaginal haematomas: a retrospective study of conservative versus operative management. South Med J. 1987;80(8):991–4.
23. South Australian Perinatal Practice Guidelines. Puerperal genital haematomas. Adelaide: Dept of Health, Govt of South Australia; 2012.

Amniotic Fluid Embolism

42

Kiran Pandey and Amrita Singh

42.1 Introduction

Amniotic fluid embolism is an obstetric emergency in which amniotic fluid, fetal cells, hair, or other debris enters mother's bloodstream through placental bed and thereby initiates allergic reaction resulting in cardiopulmonary collapse, respiratory compromise, and coagulopathy leading to massive hemorrhage.

Amniotic fluid embolism (AFE) was first described in 1926 by J.R. Meyer, but its real significance as a killer disease was probably recognized in 1941 when **Steiner and Lushbaugh**, who published an autopsy series of eight pregnant women who died due to sudden shock during labor, believed that AFE was the commonest cause of death in the first 10 h after delivery.

The term AFE is a misnomer now also known as "sudden obstetric collapse syndrome" and "anaphylactoid syndrome of pregnancy".

AFE is classically characterized by a triad of hypoxia, hypotension, and consumptive coagulopathy; however many a times nonclassical presentation may also dominate (Fig. 42.1).

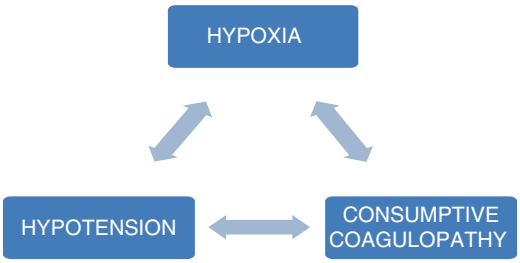


Fig. 42.1 Classical triad of AFE

42.2 Incidence

AFE is more likely underreported in many medical communities as it is a diagnosis of exclusion with no specific diagnostic tests.

AFI is a rare condition ranging from **1 in 8000 to 1 in 80,000** deliveries as per the study done by Gilbert and Danielsen [1]. Reported incidence varies from 1.9/100,000 to 6/100,000 according to some studies.

42.2.1 Maternal Mortality

The case fatality of all the cases ranges from 11% to 43% (Fig. 42.2).

AFE is the fifth most common cause of mortality [2]. Traditionally AFE was associated with an 80% mortality rate. More recent reports would suggest the mortality is between 20% and 40%, with some as low as 13%.

K. Pandey (✉) · A. Singh
Department of OBGYN, GSVM Medical College, Kanpur, Uttar Pradesh, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_42

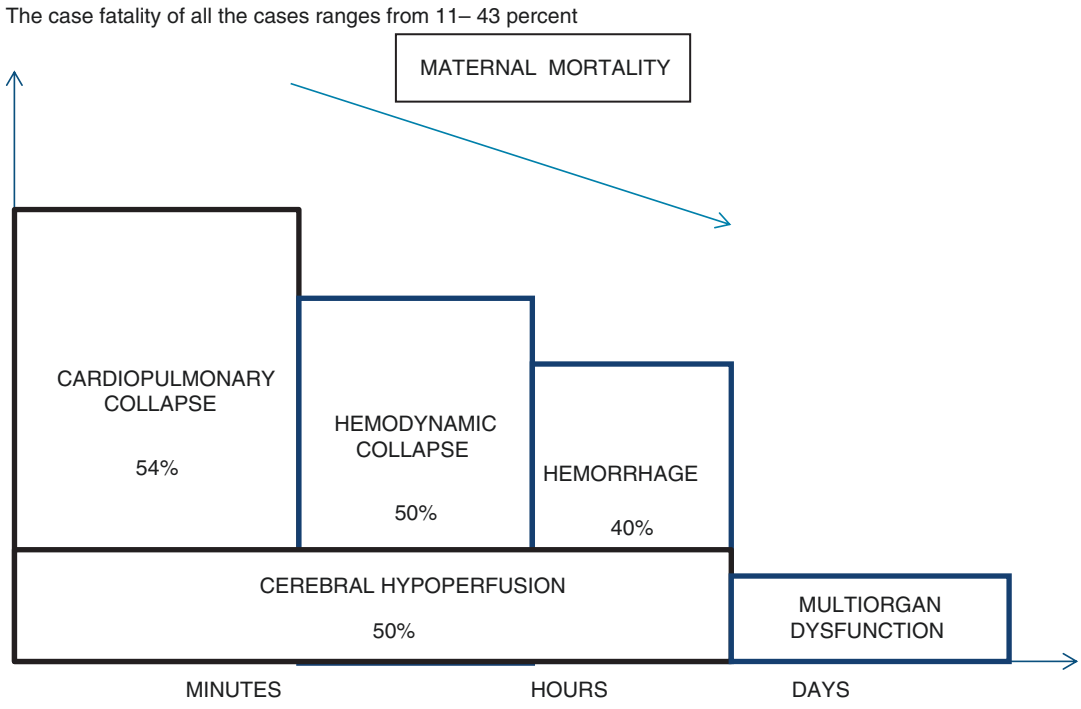


Fig. 42.2 Mortality rate according to antecedent cause on time scale

Neonatal outcome is poor, if AFE develop antenatally and **survivors however have long-term neurological impairments.**

42.2.2 Recurrence

According to a review by Agustin Conde et al published in American Journal of Obstetrics & Gynaecology, 2009, a total of 9 cases of successfull pregnancy following AFE have been reported.

On the basis of available limited experience and evidences, AFE is found to be nonrecurrent [3].

42.3 Predisposing Causes

In order for amniotic fluid to enter circulation, there are three prerequisites:

1. Ruptured membranes
2. Ruptured uterine or cervical vein
3. Pressure gradient from the uterus to vein

Various risk factors include [4]:

- Maternal age >35 years
- Placenta previa and abruption
- Cesarean sections or assisted delivery procedures (forceps, vacuum)
- Eclampsia
- Fetal distress
- Induction/augmentation of labor
- Meconium-stained amniotic fluid
- Tears in uterine or other large pelvic veins

Association of **uterine hypertonus appears to be an effect rather than cause of AFI. It is likely because uterine blood flow ceases** when intrauterine pressure exceeds 35–40 mmHg [5].

42.4 Pathogenesis

The pathogenesis of AFE is complicated and not clear; its development envisages **mechanical obstruction** of the pulmonary vessels by amniotic fluid components leading to release of **humoral and immunological factors.**

42.4.1 Pathophysiology [6]

Entrance of amniotic fluid to maternal circulation:

- Endocervical veins
- Placental insertion site
- Site of uterine trauma (Figs. 42.3 and 42.4)

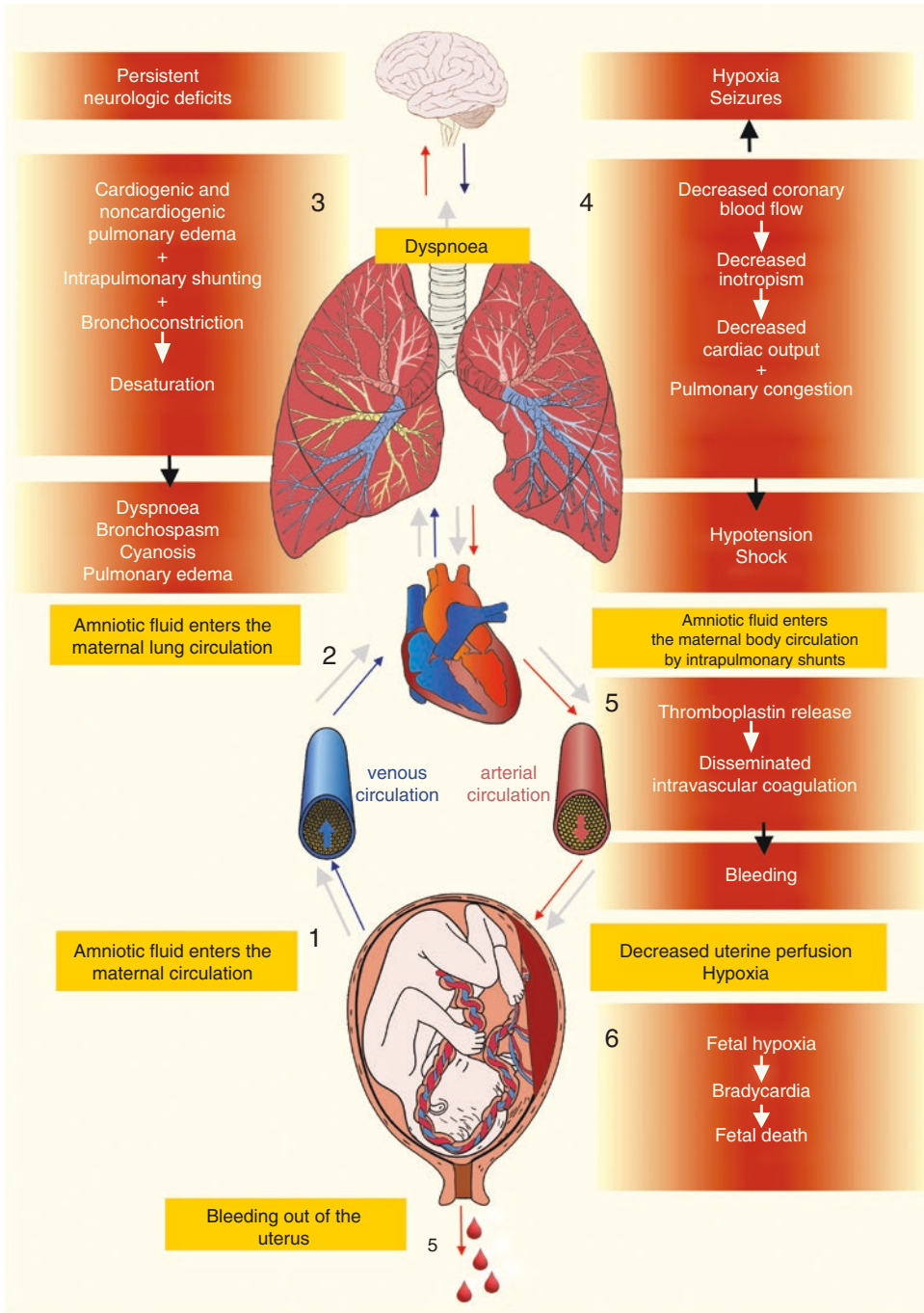
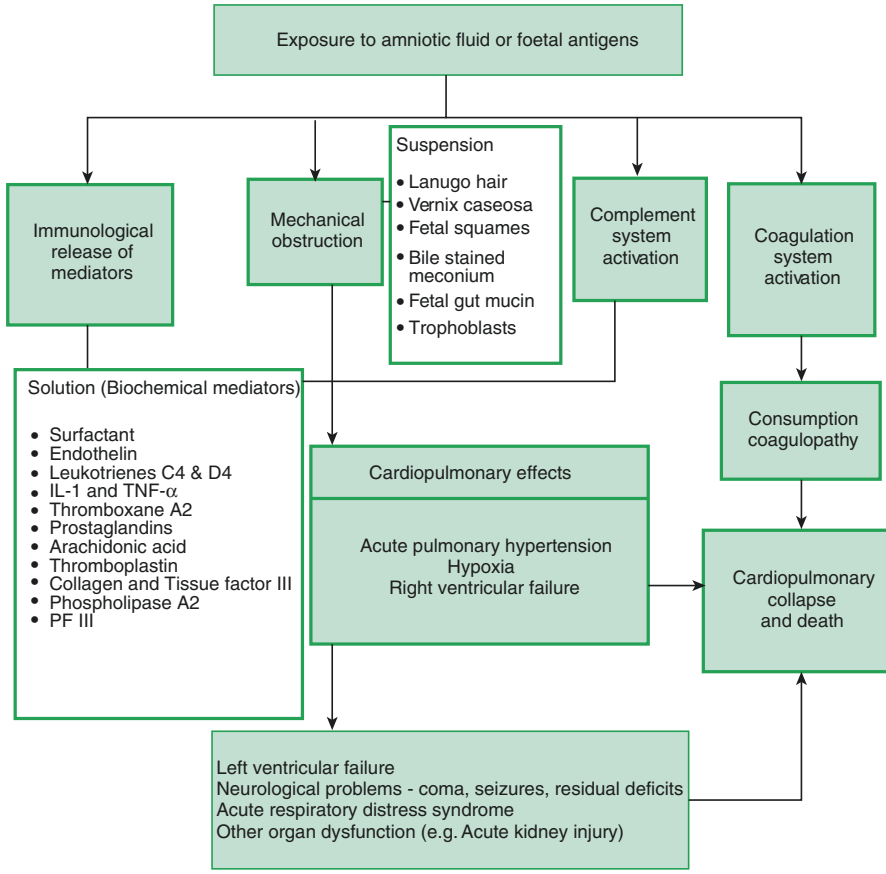


Fig. 42.3 Pathophysiology of amniotic fluid embolism



1. Mechanical obstruction of pulmonary veins by amniotic fluid components
2. Anaphylactoid reaction: numerous vasoactive substances (bradykinin, histamine, others) and procoagulant substances in amniotic fluid can lead to endothelial activation and massive inflammatory reaction similar to anaphylaxis.
Due to presence of
• Lag period
• Amniotic fluid debris in non AFES mother
3. Complement activation: the reason behind why some women tolerate transfer of amniotic fluid component with no problem or clinical symptoms and others do not is complement activation does not occur in some women.

Fig. 42.4 Proposed hypothesis and mechanisms

42.4.2 Clinical Presentation [7] (Table 42.1)

Warning Signs

- Respiratory distress
- Chest pain
- Light-headedness
- Restlessness
- Panic
- Tingling in fingers
- Nausea, vomiting

Table 42.1 Symptoms of AFE in diagnosed cases

Hypotension	60%	Cyanosis	90%
Fetal distress	90%	Coagulopathy	50%
Pulmonary edema or ARDS	45%	Dyspnea	75%
Cardiopulmonary arrest	65%	Seizures	15%

First phase:

In the first phase, the patient experiences acute shortness of breath due to pulmonary hypertension. This rapidly progresses to *cardiac failure* because of pressure overload leading to a reduction of perfusion to the heart, lungs, and finally brain. Not

long after this stage, the patient will lose consciousness due to *circulatory collapse* leading to death of mother unless managed on war footing.

Second phase:

About 40% of the initial survivors will pass onto the hemorrhagic phase after left heart failure. The blood loses its ability to clot and there is excessive bleeding. Collapse of the cardiovascular system leads to fetal distress and death unless the child is delivered swiftly (Fig. 42.5).

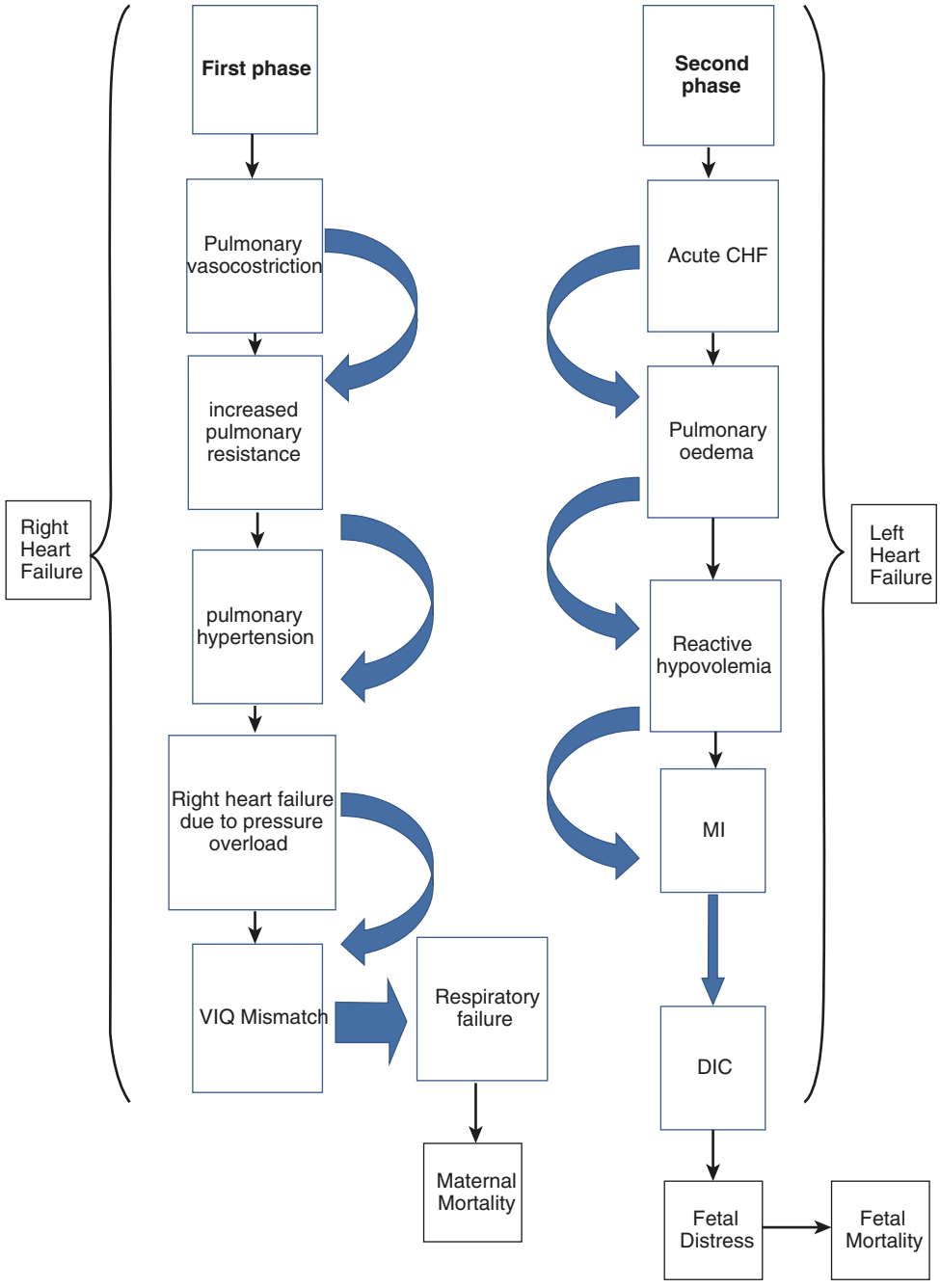


Fig. 42.5 First and second phase of AFE

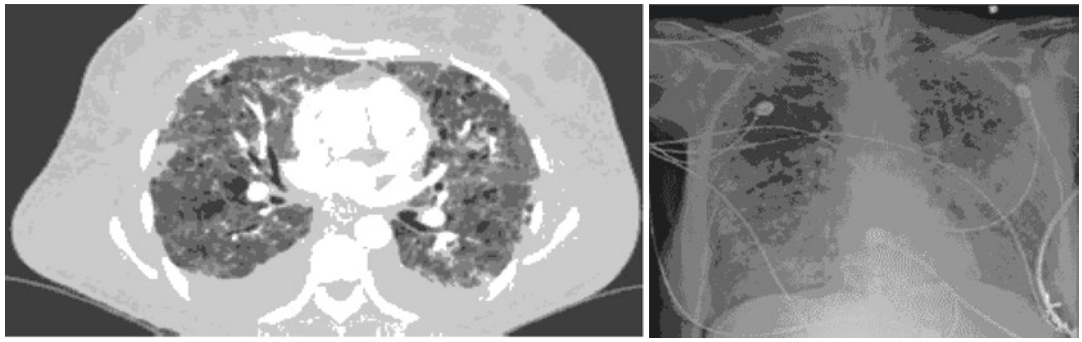


Fig. 42.6 HRCT of the chest showing ground glass infiltrate in AFE

Various systemic changes are as follows [4]:

1. **Cardiovascular system:** severe pulmonary hypertension leading to right heart failure and subsequently left heart failure too.
2. **Hematological changes:**
   Within 4 h there is rise in APTT and PT with fall in fibrinogen level.
3. **Respiratory changes:**
   Hypoxia secondary to:
   (a) Pulmonary vasoconstriction and cardiogenic pulmonary edema because of left heart failure
   (b) Inflammation of pulmonary vasculature leading to capillary leak and noncardiogenic pulmonary edema.
4. **Neurological changes**
   Encephalopathy and seizures due to hypoxia (Fig. 42.6).

42.5 Diagnosis

42.5.1 Diagnostic Criteria for AFI

There is no uniform criteria for amniotic fluid embolism which is a diagnosis of exclusion; currently the most cited ones are those of **UKOSS (UK Obstetric Surveillance System) and Benson**.

In case of sudden postpartum collapse or maternal death of unexplained etiology within 30 min from the onset of birth, AFE should be considered as its etiology as per UK registry.

As per UK Obstetric Surveillance System (UKOSS) 2010 and Benson [8]:

Table 42.2 Symptoms of AFE for the diagnosis according to UKOSS 2010 and Benson [8]

• Hypotension (and/or cardiac arrest)
• Respiratory distress
• Disseminated intravascular coagulation
• Coma and/or seizures
• Acute fetal compromise
• Cardiac arrest
• Cardiac arrhythmia

Pregnant women presenting with cardiovascular collapse within 48 h after birth or during labor and no other clear cause can be elicited, possibility of AFE to be considered with one or more of the following symptoms (Table 42.2).

42.6 Laboratory Diagnosis

There are no specific laboratory tests to diagnose AFE:

- Hemodynamic parameters, ECG, blood gas analysis, chest X-ray, and laboratory tests (including blood count, cardiac enzymes, and coagulation tests) and specific tests such as transesophageal echocardiography (TEE) and ROTEM which measures the viscoelastic properties of the blood clot.
- **Zinc coproporphyrin, sialyl-Tn antigen, tryptase, or C3 and C4 complement and detection of insulin-like growth factor-binding protein have not been established in routine clinical diagnosis** but are promising markers.

- As fetal cells can be detected in 21–100% of pregnant women without AFE, it is not pathognomonic for diagnosis.

Therefore laboratory diagnosis can be divided into two categories for confirming diagnosis and for treatment monitoring.

For treatment optimization and monitoring:

- **Complete blood count.**
- **Coagulation parameters.**
- **Including FDP, fibrinogen levels, derranged coagulation profile with increased APTT and PT and arterial blood gases revealing hypoxemia will help in making the diagnosis of AFE.**
- Spo2, co2, ph, as well as perfusion index is measured.
- **Chest X-ray** may show an enlarged right atrium and ventricle and prominent proximal pulmonary artery and pulmonary edema.
- **V/Q mismatch.**
- **Echocardiogram** may demonstrate acute left heart failure, acute right heart failure, or severe pulmonary hypertension.

Transesophageal echocardiography is beneficial to demonstrate regional wall motion abnormalities and evaluate valve function, ejection fraction, right-sided pressures, chamber size, and visualization of debris. In two case reports during the acute phase of AFE, TEE findings revealed severe pulmonary hypertension and acute right ventricular failure with a leftward deviation of the interatrial and interventricular septa.

42.7 Rotational Thromboelastometry

ROTEM test [9] measures the viscoelastic properties of the blood clot. These properties are presumed to represent the coagulation profile of the patient. ROTEM assess the rotational changes of a system composed of a needle and cup. The blood is placed inside the cup and the needle is inserted into the blood. While the system rotates, the blood is allowed to coagulate. The coagulation process changes the rotational properties of the system and a computer draws a graph of the events. These are less useful for confirming diagnosis but much more so for monitoring and treatment optimization (Table 42.3).

Table 42.3 Required investigations

Treatment optimization and monitoring	For confirmation of diagnosis [10, 11]
Complete blood count Coagulation parameter Arterial blood gases Chest X-ray V/Q mismatch Echocardiogram Transesophageal echocardiography Rotational thromboelastometry ROTEM test	Cervical histology Serum tryptase Serum sialyl-Tn antigen Zinc coproporphyrin PMV analysis (if PA catheter in situ)

42.8 Differential Diagnosis of AFE (Table 42.4 and 42.5)

42.8.1 Treatment

42.8.1.1 Targets of Management

1. **Maintaining cardiovascular and pulmonary equilibrium**
2. **Systolic blood pressure >90 mmHg**
3. **Urine output >25 mL/h**
4. **Arterial pO_2 >60 mmHg**
   (a) **Establishing and maintaining uterine tone**
   (b) **Correct coagulation abnormalities (Figs. 42.7 and 42.8)**

Table 42.4 Differential diagnosis according to the association with pregnancy

Pregnancy-specific diagnosis	Incidental causes associated with pregnancy	Medical disorders associated with pregnancy
Acute hemorrhage Uterine rupture Eclampsia Peripartum cardiomyopathy	High regional block local Anesthetic toxicity Air embolism Anaphylaxis Transfusion reaction	Pulmonary embolism Sepsis Cardiac ischemia Arrhythmia

Table 42.5 Differential diagnoses by clinical symptoms [12]

Symptoms→ Differential diagnosis↓	Chest pain	Dyspnea	Neurological symptoms	Hypotension	Coagulopathy	Fetal distress	Common period of occurrence
Amniotic fluid embolism	+/–	+++	++	+++	++	++	Intrapartum/immediate postpartum
Pulmonary embolism	+++	+++	+	++	–	+	Intrapartum/antepartum
Myocardial infarction	+++	++	+	++	–	+	Peripartum/postpartum
Peripartum cardiomyopathy	++	++	+	+/–	–	+/–	Third trimester to 4 months postpartum

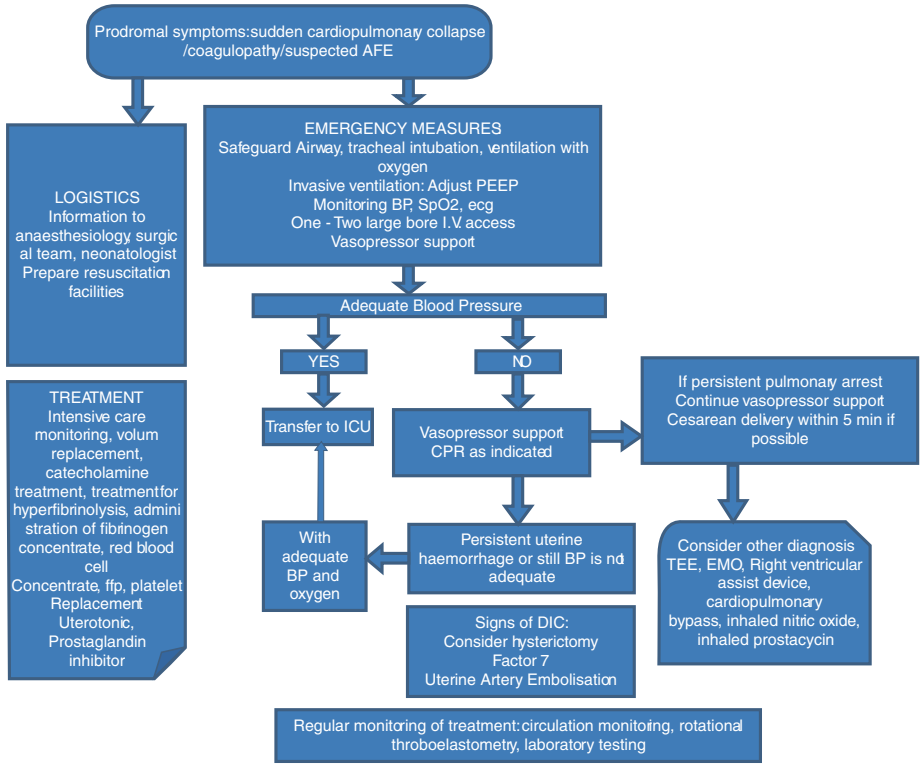


Fig. 42.7 Management protocol

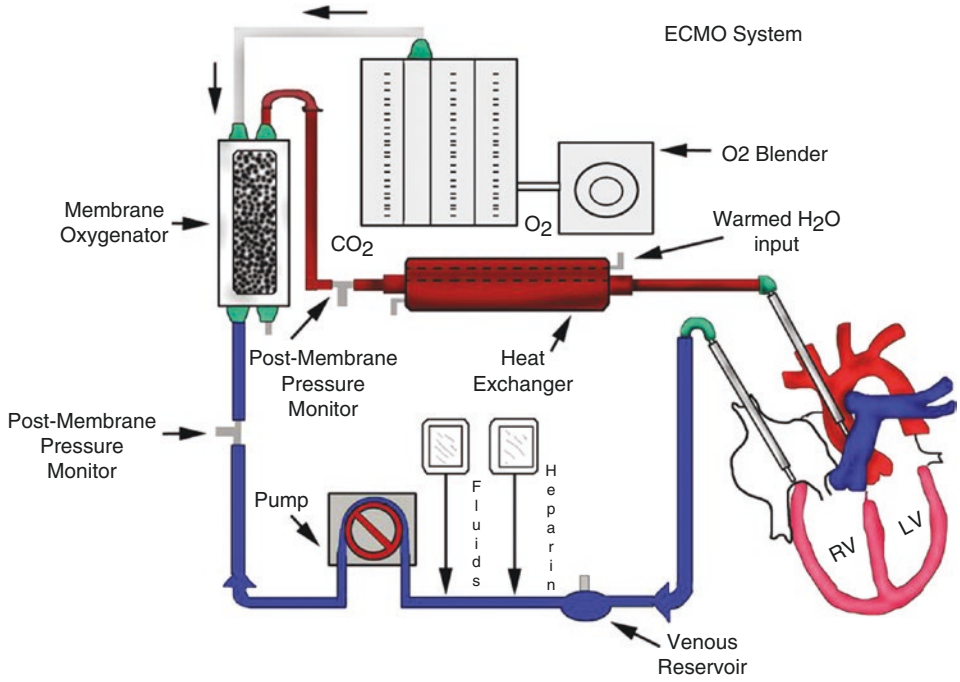


Fig. 42.8 Extracorporeal membrane oxygen system

42.8.2 Modern Approach

42.8.2.1 Modern Strategies for Treatment of AFE

Indication	Strategies
Right sided heart failure and pulmonary hypertension	Right ventricular assist device (RVAD); Consider ECMO
	Inhaled nitric oxide
Pulmonary hypertension and severe hypoxia	Inhaled epoprostenol
Left sided heart failure and severe hypoxia	Intra-aortic balloon pump counter pulsation
	Extracorporeal membrane oxygenation (ECMO)
	Cardiopulmonary bypass
Massive hemorrhage	Uterine artery embolization
	Recombinant factor Vila
Removal of circulating amniotic fluid contents and improve organ perfusion	Continuous hemofiltration
	Plasmapheresis
	Cell salvage combined with blood filtration
Disseminated intravascular coagulation	Serum protease inhibitors
Anaphylaxis	High-dose corticosteroid administration

Cardiopulmonary bypass, nitric oxide, and plasmapheresis are specific interventions which have been described in exceptional circumstances.

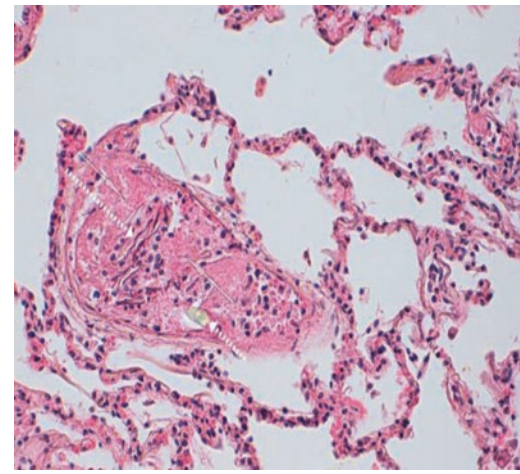
In the event of cardiorespiratory arrest, **three additional considerations should be kept in mind during CPR on a pregnant woman** [4]**:**

1. **Left lateral tilt to reduce the impact of aortocaval compression on venous return during resuscitation.**
2. **Early intubation to secure the airway as there is high maternal oxygen consumption and reduced FRC**.
3. If there is **no response to CPR then after 4 min of traditional resuscitation measures and if the fetus is over 20 weeks gestation, a perimortem cesarean should be performed with the aim of achieving delivery within 5 min.**

42.8.2.2 Forensic Postmortem Evidence of AFE

Unexpected and sudden maternal death during childbirth raises suspicion on diagnosis towards AFE, and [13, 14] in 30–40% of cases of histologically confirmed AFE, hemorrhagic shock has been found as cause of death [15].

1. Detection of formed amniotic fluid component such as lamellar, adjacent epidermal squames, meconium component, or lanugo hairs constitutes histological evidence of AFE [16, 17].
2. Embolic material is found mainly in pulmonary arterioles and capillaries. Fibrin thrombi in connection with amniotic fluid are universal and can be detected even after a survival time of 2 h or more [15].
3. Conventional stains such as **hematoxylin and eosin** as a surveillance and immunohistochemical staining of fetal epithelial cells using **cytokeratin** is now the standard procedure, and representative samples from each lung segment should be taken [17].
4. Morphologically determined severity of AFE doesn't correlate with severity of symptoms [15].
5. **An absence of histological evidence of amniotic fluid components in the lung following clinical manifestation of AFE and maternal death rules out AFE**; in case of anaphylactoid reaction, the transfer of a small histomorphologically undetected amount of amniotic fluid into the maternal circulation may be the cause, but in such cases there would be no histological evidence of DIC [15].
6. If the mother survives longer, it should be borne in mind that as yet there is no reliable information on how long amniotic fluid components remain in the circulation [15].



A blood vessel enclosed by lamellar epithelial squames (long dotted arrow) embedded in a fibrin thrombus (two transparent arrows). The lower part of the picture shows a transparent, cylindrical structure corresponding to a lanugo hair (short dotted arrow). Hematoxylin and eosin staining: 200×. Survival time: 8-h immunohistochemically marked epithelial squames in pulmonary arterioles (arrows). Cytokeratin, 200×.

42.9 Medicolegal Pitfalls

Sudden unexpected death of a pregnant woman of unknown **cause must be classified as unexplained death, and autopsy must be requested; not requesting is a pitfall.**

Failure to respond is a pitfall. AFE is a clinical diagnosis. Steps must be taken to stabilize the patient as soon as symptoms manifest.

Failure to perform perimortem cesarean delivery in a timely fashion is a pitfall.

Failure to consider the diagnosis during legal abortion is a pitfall.

42.10 Summary

- **AFE is a sudden and unexpected rare but life-threatening complication of pregnancy.**
- **It is one of the leading causes of death resulting directly from child birth; case-related mortality is 11–44%.**
- **It has a complex pathogenesis and serious implications for both mother and infant associated with high rates of mortality and morbidity.**
- **Main risk factors are maternal age >35 years, cesarean section, placenta previa, and multiple pregnancy.**
- **Diagnosis of exclusion.**
- **Suspect AFE when confronted with any pregnant patient who has sudden onset of respiratory distress, cardiac collapse, seizures, unexplained fetal distress, and abnormal bleeding. Obstetricians should be alert to the symptoms of AFE and strive for prompt and aggressive treatment.**

References

1. Gilbert WM, Danielsen B. Amniotic fluid embolism: decreased mortality in a population-based study. Obstet Gynecol. 1999;93(6):973–7. https://doi.org/10.1016/s0029-7844(99)00004-6.
2. Ellingsen CL, Eggebø TM, Lexow K. Amniotic fluid embolism after blunt abdominal trauma. Resuscitation. 2007;75(1):180–3. https://doi.org/10.1016/j.resuscitation.2007.02.010.
3. Conde-Agudelo A, Romero R. Amniotic fluid embolism: an evidence-based review. Am J Obstet Gynecol. 2009;201:445.e1–13.
4. Tan A, McDonnell N. Amniotic fluid embolism, Anesthesia tutorial of the week. 2010;197:1
5. Cunnigham FG, Leveno KJ, Bloom SL, Spong CY, Dashe JS, Hoffman BL, Casey BM, Sheffield JS. Williams obstetrics. 24th ed. New York: McGraw-Hill Education; 2014.
6. Gei A, Hankins GDV. Amniotic fluid embolus: an update. Contemp Ob/Gyn. 2000;45:53–66.
7. Moore J, Baldisseri MR. Amniotic fluid embolism. Crit Care Med. 2005;33(10 Suppl):S279–85. https://doi.org/10.1097/01.CCM.0000183158.71311.28.
8. Benson MD. A hypothesis regarding complement activation and amniotic fluid embolism. Med Hypotheses. 2007;68:1019–25.
9. Collins NF, Bloor M, Mc Donnell NJ. Hyperfibrinolysis diagnosed by rotational thromboelastometry in a case of suspected amniotic fluid embolism. Int J Obstet Anest. 2013;22:71–6.
10. Conde-Agudelo A, Romero R. Amniotic fluid embolism: an evidence-based review. AJOG. 2009;201:445.
11. Toy H. Amniotic fluid embolism. Eur J Gen Med. 2009;6:108–15.
12. Feige A, Rath W, Schmidt S, editors. Amniotic fluid embolism, pulmonary embolism. New York: Thieme; 2013. p. 142–9.
13. Franchitto N, Minville V, Dédouit F, Telmon N, Rougé D. Medical responsibility in the operating room: the example of an amniotic fluid embolism. J Forensic Sci. 2012;57:1120–3.
14. Jecmenica D, Baralic I, Alempijevic D, Pavlevic S, Kiurski M, Terzic M. Amniotic fluid embolism—apropos two consecutive cases. J Forensic Sci. 2011;56:247–51.
15. Sinicina I, Pankratz H, Bise K, Matevossian E. Forensic aspects of post-mortem histological detection of amniotic fluid embolism. Int J Legal Med. 2010;124:55–62.
16. Knight M, Tufnell D, Brocklehurst P, Spark P, Kurinczuk J, On Behalf of the UK Obstetric Surveillance System. Incidence and risk factors for amniotic-fluid embolism. Obstet Gynecol. 2010;115:910–7.
17. Sisodia SM, Bendale KA, Khan WAZ. Amniotic fluid embolism: a cause of sudden maternal death and police inquest. Am J Forensic Med Pathol. 2012;33:330–4.

Postpartum Maternal Collapse

43

Smiti Nanda and Deepti Jain

43.1 Introduction

Postpartum maternal collapse is a serious emergency which occurs just after delivery and affects the cardiorespiratory system and/or brain. There is alteration in the conscious level and death may also occur. The incidence of maternal collapse has been found to be between 0.14 and 6/1000 (14 and 600/100,000) births in the United Kingdom, after an analysis of severe maternal morbidity and maternal mortality rates in the United Kingdom [1].

Since postpartum collapse is a rare event, it is important that the staff in labour room is skilled in initial resuscitation techniques, to diagnose the cause of collapse and avert morbidity and mortality.

43.2 Causes of Maternal Collapse

Postpartum collapse may result from obstetrical conditions, i.e. those related directly to pregnancy and childbirth [2]. However medical and surgical conditions associated with pregnancy can also get aggravated during labour and may result in collapse.

S. Nanda (✉)
Department of Obstetrics and Gynecology, Postgraduate Institute of Medical Sciences, Rohtak, Haryana, India

D. Jain
Chhotu Ram Hospital, Rohtak, Haryana, India

Table 43.1 Causes of maternal collapse

S. no	4Hs	4Ts	Specific to pregnancy
1	Hypovolaemia	Thromboembolism	Pre-eclampsia
2	Hypoxia	Toxicity	Eclampsia
3	Hypo/hyperkalemia, other electrolyte disturbances	Tension pneumothorax	
4	Hypothermia	Tamponade	

The Resuscitation Council (UK) employs the well-known 'aide-memoire' for the common reversible causes of collapse, which includes the 4Ts and the 4Hs [1]. Eclampsia and intracranial haemorrhage are two potential causes of collapse in pregnant women and hence are being added to the list of causes of maternal collapse (Table 43.1).

Here in this chapter, we discuss maternal collapse chiefly occurring during labour or just after delivery, i.e. postpartum maternal collapse. The causes of postpartum collapse are now detailed below.

1. Haemorrhage

   In a pregnant woman, the increase in cardiac output by about 30–60% till 32–36 weeks and the hyperdynamic circulation results in excessive blood loss from the uterus after delivery. If the mother is anaemic or there is a coexistent coagulation disorder, the blood loss can be more detrimental for the mother. Concealed haemorrhage often delays action in a woman with collapse

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_43

and needs to be looked for wherever hypovolaemia is suspected.

One quarter of all maternal deaths result from severe obstetric haemorrhage, and hence it is a significant cause of collapse. Postpartum haemorrhage can be caused by uterine atony and trauma (such as in case of uterine rupture, cervical/vaginal lacerations) and also due to coagulopathy. Visible haemorrhage in a case of collapse is detected earlier and thus with a better salvage rate. However in concealed haemorrhage such as in caesarean section, in large perineal hematomas, a high degree of suspicion is required for early detection and management.

2. Thromboembolism

   It was found to be the commonest cause of maternal death in the last CEMACH report. There were 41 deaths from thromboembolism (33 pulmonary embolisms and 8 cerebral venous thromboses) [3].

3. Amniotic fluid embolism has been found to have a frequency of about 7.7 per 100,000 deliveries, with a mortality rate of about 60–80% [4].

   The classic signs of amniotic fluid embolism are:
   - Respiratory distress
   - Altered mental state
   - Profound hypotension
   - Severe hypoxia
   - Coagulopathy and eventually death

   These symptoms occur during labour or within 30 min of delivery.

4. In the CEMACH report, cardiac disease was found to be a significant cause of maternal death, responsible for 48 maternal deaths [3]. The high-risk cardiac group for postpartum collapse includes patients with coronary artery disease, pulmonary hypertension, endocarditis, cardiomyopathy and dysrhythmias.

5. Sepsis

   Sepsis is a leading cause of maternal morbidity and mortality in intensive care units. The obstetric patient in labour can develop sepsis because of associated conditions such as pyelonephritis, chorioamnionitis, endometritis, pancreatitis, etc. Sepsis, severe sepsis and finally septic shock lead to collapse. Streptococci groups A, B and D, pneumococcus and *Escherichia coli* are the common pathogens responsible for sepsis in obstetrics [1].

6. Drug Toxicity/Overdose

   Drugs used in anaesthesia and labour room should be classified and stored in labelled containers to avoid inadvertent faulty administration in a hurry. Magnesium sulphate given in a patient with renal impairment, opioids and local anaesthetic agents given intravenously by mistake are the drugs causing overdose or toxicity.

7. Eclampsia

   The occurrence of seizures in a pregnant woman with a history suggestive of pre-eclampsia can lead to an early diagnosis of eclampsia. In a woman with seizures and collapse, in the absence of hypertension and albuminuria, epilepsy or other neurological causes should be looked for.

8. Intracranial Haemorrhage

   Patients with uncontrolled hypertension, particularly systolic hypertension, may develop intracranial haemorrhage during labour or after delivery. However intracranial haemorrhage may also occur from ruptured aneurysms and arteriovenous malformations.

9. Anaphylaxis

   Anaphylaxis is referred to a severe systemic hypersensitivity reaction with respiratory, cutaneous, circulatory and gastrointestinal disturbance, followed by collapse. The incidence of anaphylaxis is between 3 and 10/1000 and a mortality rate of around 1% [5].
   - A patient with anaphylaxis may develop severe airway and/or breathing and/or circulatory disturbance.
   - The skin and muscle may show generalized flushing, urticaria and angioedema.

10. Acute Inversion of the Uterus

    Acute inversion often occurs when deliveries are conducted at home by untrained helpers. The uterus is pulled inside out when traction is applied on the umbilical cord before placental separation has been achieved. Inversion uterus may result in collapse because of a vasovagal reaction. A fatal haemorrhage may also ensue following uterine inversion leading to collapse.

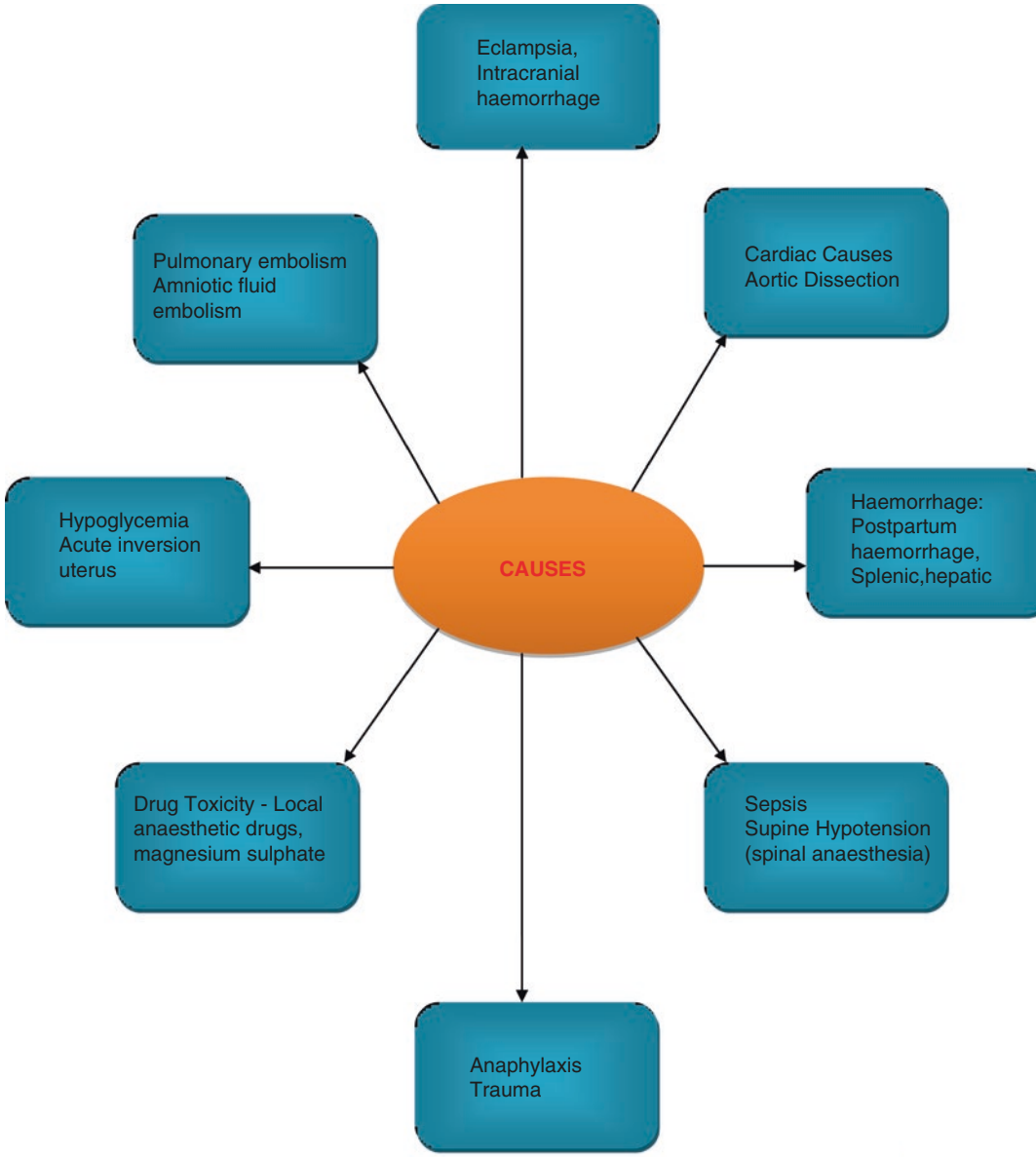


Fig. 43.1 Causes of postpartum collapse

11. Miscellaneous

Maternal collapse following delivery may occur due to hypoglycaemia and other metabolic/electrolyte disturbances. Pregnant women with diabetes or renal insufficiency are especially susceptible to electrolyte disturbances. Airway obstruction because of aspiration or foreign body may also lead to collapse (Fig. 43.1).

43.3 Management of Maternal Collapse

For management of maternal collapse, resuscitation is the initial basic step, followed by ascertaining the cause of collapse, and then appropriate steps are taken to correct the underlying disorder (Fig. 43.2).

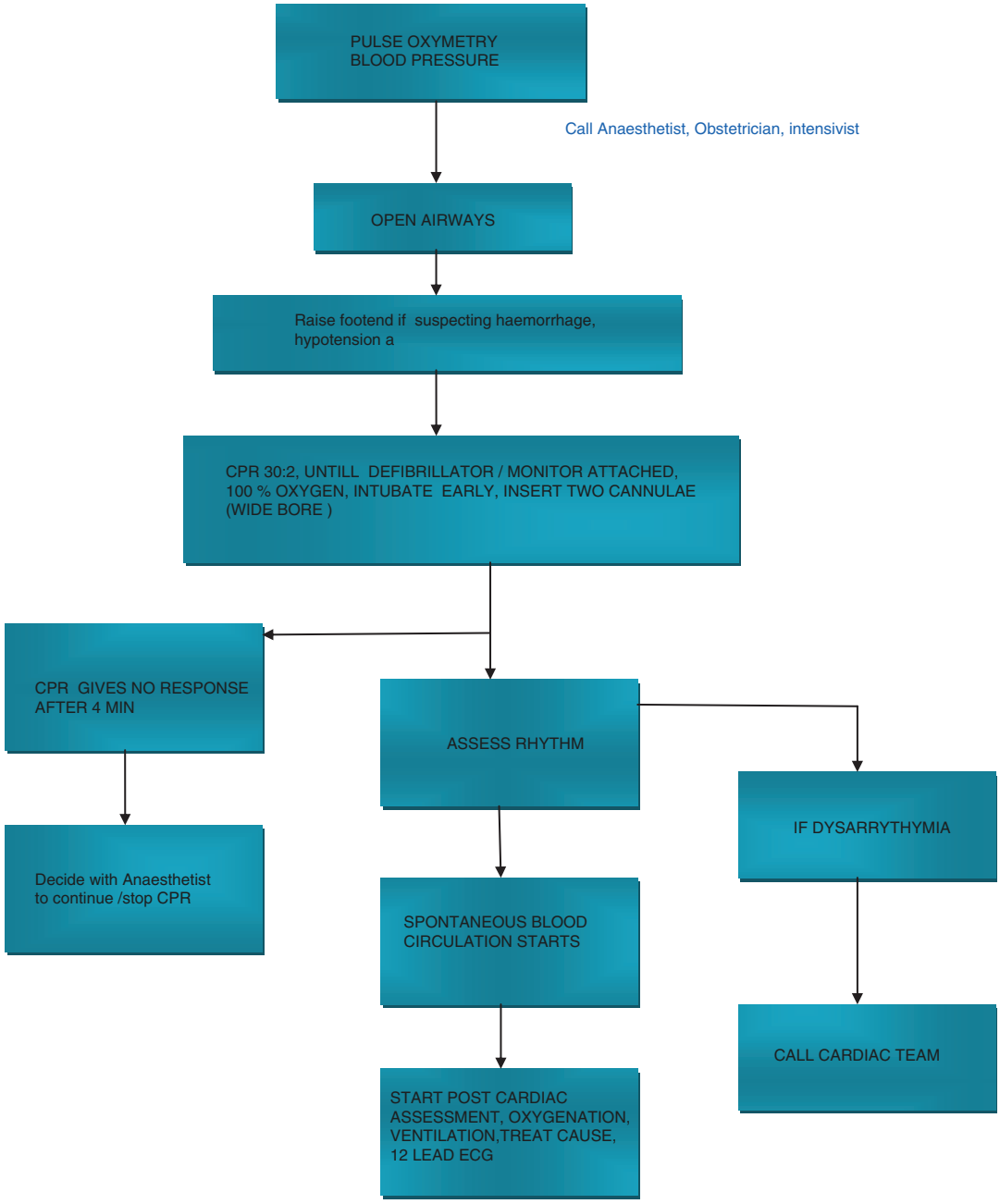


Fig. 43.2 Management of postpartum collapse (algorithm)

43.4 Maternal Resuscitation

In 2010, the UK Resuscitation Council formulated guidelines for resuscitation in a patient with collapse, and these are now recommended for use in pregnant women with collapse [6].

43.4.1 Basic Steps in Resuscitation in Obstetric Collapse

'Call for help' is the basic requirement when collapse occurs in labour room. A consultant obstetrician, anaesthetist, haematologist and intensivist need to be called urgently as soon as the information regarding a collapse case is received from the labour room. The ABCDE approach is followed with.

Step 1. Airway

An endotracheal tube should be inserted as quickly as possible to facilitate good oxygen delivery and effective ventilation.

Step 2. Breathing

100% oxygen should be provided by endotracheal tube. However till the time intubation has not been achieved, oxygen should be given with bag and mask, and chest compressions are begun and performed in a ratio of 30 compressions:2 ventilations.

Step 3. Circulation

(a) At the outset, as a clear airway has been ensured. Chest compressions should be started immediately. Chest compressions are performed vigorously and continued till an adequate cardiac output and cardiac rhythm is restored. The hands should be placed over the Centre of the chest, and the chest is compressed at right angles to the chest wall to a depth of 5 cm but less than 6 cm. Chest compressions and ventilations are performed in a ratio of 100:10 per minute.

(b) Two wide gauge canulae are inserted and intravenous fluids given aggressively, if haemorrhage is suspected. However, in a case of pre-eclampsia or eclampsia, careful volume of fluid should be given to prevent fluid overload.

(c) If cardiac rhythm is not restored, a defibrillator is used in similar settings as for the nonpregnant patient.

Step 4. Drugs

The drugs should be used in the same algorithm and doses as in any other patient with collapse.

A resuscitation emergency tray containing life-saving drugs such as noradrenaline, adrenaline, dobutamine, dopamine, atropine, hydrocortisone, oxytocin, Methergin, midazolam, frusemide, Epsolin and labetalol should always be kept ready for use.

Resuscitation should be continued until a decision to stop the same is taken by the consultant anaesthetist and obstetrician.

Step 5. After the patient has been resuscitated, efforts should now be made to quickly diagnose the cause of collapse which should then be treated accordingly. A quick history is taken which includes illnesses associated with pregnancy like heart disease, diabetes and anaemia. The history of similar occurrences in previous deliveries is elicited as conditions like peripartum cardiomyopathy tend to recur in future deliveries. An inquiry regarding surgeries such as caesarean section, hysterotomy and myomectomy is made, as the scarred uterus may have ruptured during labour. The antenatal chart is studied to learn about events of pregnancy like pre-eclampsia, severe anaemia, hypertension, pyelonephritis or antepartum haemorrhage, which may have contributed to the present collapsed state. The record of labour is checked to know about intrapartum haemorrhage, excessive use of uterotonics like oxytocin and misoprostol and instrumentation in the second stage such as vacuum or

forceps application. Drugs administered during labour like narcotic analgesics, magnesium sulphate and antibiotics are taken note of, which may have caused overdose toxicity, respiratory depression or anaphylaxis. In the history of the third stage of labour, events like undue pulling on the cord before placental separation, third stage haemorrhage, manual removal of placenta, breathlessness and convulsions are also searched for.

The general physical examination of the mother is done recording the rate and volume of pulse, blood pressure, oxygen saturation and level of consciousness. The cardiac and respiratory functions are then assessed. A churning sound in the heart, if heard, is suggestive of air embolism. On auscultation of the chest, rales if present are suggestive of pulmonary oedema. The uterine contour is checked on examination of the abdomen. A firm and rounded, contracted uterus rules out atonic postpartum haemorrhage and inversion uterus. Free fluid may be felt on abdominal examination in case of intraperitoneal haemorrhage. An inspection of the vulva is done to see if haemorrhage is still continuing. After the delivery of placenta, pelvic examination is done to look for cervical tears, vaginal lacerations and incomplete inversion of the uterus.

43.5 Management of the Cause of Collapse

1. Haemorrhage

   Postpartum haemorrhage should first be managed aggressively with blood transfusion. Further treatment is given, depending upon the aetiology whether it is atonic, traumatic or coagulopathy. If haemorrhage is due to an atonic uterus, the management includes bimanual uterine compression, systematic stepwise use of interventions to stop bleeding with uterotonics (oxytocin, 15-methyl prostaglandin F2 alpha, misoprostol, ergometrine), uterine tamponade with balloon, insertion of brace suture), advanced surgical interventions (uterine devascularization by ligation of uterine or internal iliac arteries), interventional radiology and definitive surgery (hysterectomy). In case of traumatic postpartum haemorrhage, bleeding is stopped with suturing of vaginal or cervical tears, evacuation of perineal haematomas and repair of vaginal vault or uterine rupture. In case of suspected coagulation disorders, fresh blood and blood components are utilized to combat haemorrhage.
2. Venous Thromboembolism

   Pulmonary embolism just after delivery presents with shock, refractory hypoxemia and/or right ventricular dysfunction on echocardiogram. Following resuscitation, in the women with pulmonary embolism, intravenous unfractionated heparin is the preferred treatment, because of its quick onset of action and extensive use in this situation.

   In massive life-threatening pulmonary embolism with haemodynamic compromise (or with limb or life-threatening ischaemic complications from extensive iliofemoral vein thrombosis), thrombolytic therapy is considered [7].

   In high-risk obstetric patients such as those with obesity and hereditary or acquired thrombophilias, thromboprophylaxis may help in reducing maternal morbidity and mortality.
3. Eclampsia

   Magnesium sulphate protocol should be started for eclampsia. The blood pressure should be lowered as soon as possible using an intravenous agent such as labetalol. Fluid balance is important, and since there is generalized vasoconstriction, overloading is to be avoided. Further investigations should be considered urgently to establish if other systems are involved in the process such as thrombocytopenia and hepatic or renal dysfunction. Brain imaging is required urgently in case of repeated seizures or focal neuro-

logical deficit, suggesting possible intracranial pathology.

4. Amniotic Fluid Embolism

   The primary goal of management of amniotic fluid embolism includes cardiopulmonary stabilization in order to maintain vascular perfusion and prevent hypoxia. Fresh blood and blood products are arranged pre-emptively, as coagulopathy often follows amniotic fluid embolism.

5. Coagulopathy

   In the patient with evidence of coagulatory dysfunction, fresh frozen plasma should be infused aggressively. Other therapies like steroids, heparin, plasmapheresis and haemofiltration have been tried but clear evidence is still lacking.

6. Cardiac Disease

   After the collapsed mother has been resuscitated, cardiac disease is managed by the hospital cardiology team.

   In a case of myocardial infarction, antithrombin therapy can be given, although with a risk of placental site haemorrhage. If required, percutaneous angioplasty can be performed as a definitive treatment modality [8].

7. Sepsis

   The guidelines given by surviving sepsis campaign for the management of sepsis can be utilized in cases with postpartum collapse also. Survival rates in severe sepsis can be improved by giving appropriate antimicrobial therapy very quickly in the initial hours of sepsis along with rapid resuscitative efforts.

   - Serum lactate is measured.
   - Blood cultures and culture swabs are taken prior to antibiotic therapy.
   - Broad spectrum antibiotics are administered early, preferably within the first hour of diagnosis of severe sepsis and shock.
   - If patient is in hypotension and/or lactate is 7.4 nmol/L or more:
     (a) 20 mL/kg of crystalloid/colloid is transfused.
     (b) When adequate volume or fluid has been transfused, blood pressure is managed with vasopressors (norepinephrine, epinephrine) and or inotropic drugs (e.g. dobutamine) to maintain mean arterial pressure above 65 mmHg.
     (c) In refractory hypotension, steroids can be used.
   - Oxygen saturation is maintained with facial oxygen. If haemoglobin is below 7 g/dL, blood transfusion is given.
   - The focus of sepsis is now removed and thromboprophylaxis may be given [9].

8. Drug Overdose Toxicity

   (a) Magnesium sulphate toxicity is treated with 10 mL of 10% calcium gluconate given slowly intravenously.
   (b) Systemic local anaesthetic agent toxicity is now managed with lipid rescue.

   Cardiac arrest due to local anaesthetic toxicity not responding to standard therapy is now being treated with an intravenous infusion of 20% lipid therapy [10].

9. Intracranial Haemorrhage

   A diagnosis should be established with an expert neuroradiology team and the patient managed depending upon the diagnosis made after imaging.

10. Anaphylaxis

    - Anaphylaxis is treated with 500 μg (0.5 mL) of 1:1000 adrenaline given by intramuscular route.
    - Adrenaline treatment can be repeated after 5 min if there is no response.
    - Chlorpheniramine 10 mg and hydrocortisone 200 mg can be given both intramuscularly and slowly intravenously [11, 12] (Fig. 43.3).
    - All cases of postpartum maternal collapse should be reviewed by a team of experts and new hospital guidelines formulated to insure better salvage rates in the future.

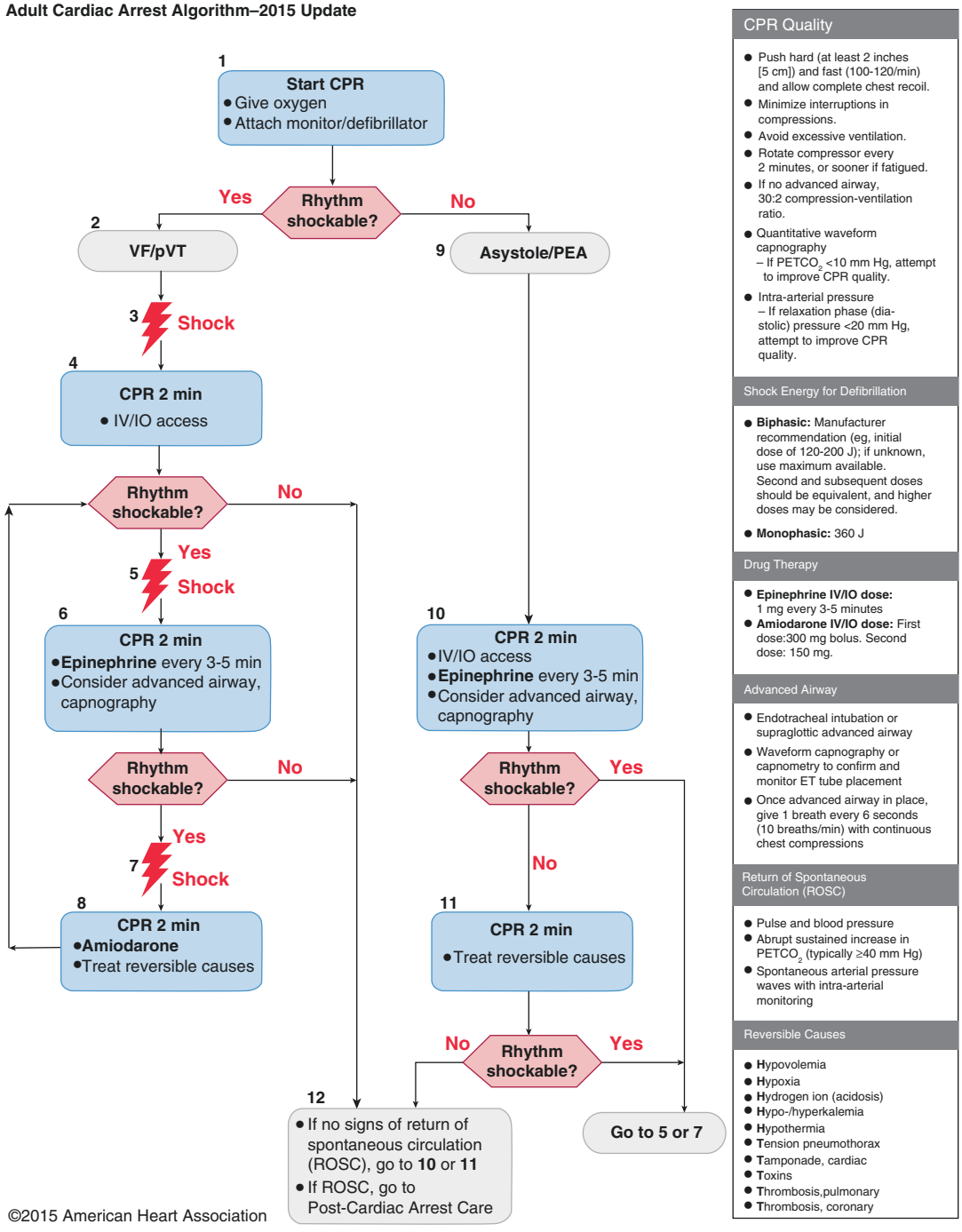


Fig. 43.3 Steps of CPR (AHA guidelines) [14]

43.6 Prevention

Good antenatal care can help identify many risk factors. Identification of risk factors, involvement of specialists from related fields, careful management in labour such as following the partogram, wise decision-making during the second stage of labour regarding operative procedures and close watch for vitals and haemorrhage in the third stage can go a long way in preventing the catastrophic condition of collapse. Lastly, if collapse does occur, aggressive stepwise resuscitation, diagnosis and treatment of the cause leading to collapse can help in preventing maternal mortality and giving us better salvage rates [13].

References

1. Maternal collapse in pregnancy and the puerperium. Royal College of Obstetricians and Gynecologists, Green Top Guideline No. 56, 2011.
2. Barnes J. Post-partum maternal collapse. BMJ. 1955;1(4925):1333–5.
3. Royal College of Obstetricians and Gynaecologists. Saving mothers' lives 2003–2005. Confidential enquiry into maternal and child health. London: RCOG Press; 2007.
4. Abenhaim HA, Azoulay L, Kramer MS, Leduc L. Incidence and risk factors of amniotic fluid embolisms: a population based study on 3 million births in United States. Am J Obstet Gynecol. 2008;199:49–52.
5. Working group of Resuscitation Council (UK Emergency treatment of anaphylactic reactions; Guidelines for healthcare providers. London: Resuscitation Council (UK); 2008. http://www.resus.org.uk/pages/reaction.pdf.
6. Nolan JP, Soar J, Zideman DA, Barrent D, Bossaert LL, Deakin CD, et al., ERC Guidelines Writing Group. European Resuscitation Council Guidelines for Resuscitation 2010 Section 1. Executive summary. Resuscitation. 2010;81:1219–76.
7. Thrombolic disease in pregnancy and the puerperium: acute management, RCOG Green Top Guidelines No. 37 b, April 2015.
8. Steer PJ, Gatzoulis MA, Baker P. Heart disease and pregnancy. London: RCOG Press; 2006.
9. Levy MM, Dellinger RP, Townsend SR, Lind-Zwirble WT, Marshall JC, Bion J, et al. The Surviving Sepsis Campaign: results of an international guideline-based performance improvement program targeting severe sepsis. Crit Care Med. 2010;38(2):367–74.
10. Foxall G, McCahon R, Lamb J, Hardman JG, Bedforth NM. Levobupivacaine-induced seizures and cardiovascular collapse treated with Intralipid. Anaesthesia. 2007;62:516–8.
11. National Patient Safety Agency. Safer practice with epidural injections and infusions. Patient safety alert 21. London:National Patient Safety Agency; 2007.
12. Harper NJ, Dixon T, Dugué P, Edgar DM, Fay A, Gooi HC, et al. Suspected anaphylactic reactions associated with anaesthesia. Anaesthesia. 2009;64:199–211.
13. Nanda S, Peena LK. Postpartum collapse. Curr Obstet Gynaecol. 2009;16(8):221–8.
14. AHA CPR Guidelines—Updated 2014/2015.

Postpartum Sepsis

44

Madhu Nagpal

44.1 Introduction

Maternal sepsis contributes to nearly 10–15% of maternal morbidity and 8–10% of maternal mortality. It is still responsible for poor general health of women in developing countries [1]. The maternal sepsis is seen in 0.1–0.6 per 1000 deliveries in developed countries resulting in high mortality rate of 10% [2]. In developing world, the incidence of sepsis varies from 0.03% to 0.7% contributing to almost one third of all maternal deaths [3]. As per WHO fact sheet 2010 [4], sepsis results in maternal mortality in 8%. In India it is still a direct cause of death in 13% as per report of ICMR [5]. There are many reasons for it, one being late recognition of severe sepsis and late arrival at the referral site, despite intensive planning in policy making and interventions adopted during policy implementation. A decline in number of total maternal mortality by 4.5% annually has been observed due to improved maternal healthcare in the last few years, although proposed decline of 5.5% has not been achieved to meet WHO-defined millennium development goal 5 (MDG-5).

44.2 Definition

Postpartum sepsis is defined as any genital tract infection which occurs within 28 days of miscarriage, induced abortions and childbirth. Any maternal fever more than 38 °C on 2 successive days in the first 10 postpartum days excluding the first 24 h indicates puerperal infection. Although institutional deliveries are being encouraged where aseptic norms for childbirth are observed rigidly now, incidence of sepsis after delivery is significantly high. The number of illegal abortions has also declined due to strict observance of norms of MTP Act and PC-PNDT Act.

44.3 Sepsis

Sepsis is broadly defined as infection leading to systemic inflammatory response syndrome in various intensities from mild to severe. It is mild when there is initial inflammatory response represented by pyrexia and pain. It is called severe sepsis when it has either induced tissue hypoperfusion recognizable as systolic blood pressure being <90 mmHg, MAP <70 mmHg, SBP fall by >40 mmHg or SBP being <2SD below normal for patient's age after excluding other causes of hypotension, increased lactate levels and oliguria. Septic shock is defined as severe hypotension as a result of severe sepsis which becomes refractory to fluid resuscitation, further progressing to multiorgan dysfunction syndrome (MODS).

M. Nagpal (✉)
SGRD IMS&R, Amritsar, Punjab, India

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44.4 Sepsis and Pregnancy Physiology

Pregnancy is a special situation where maternal physiological changes are significantly altered with a little reserve for compensation in case of sepsis. The cardiovascular system undergoes hyperdynamic circulatory changes with increased blood volume, increased cardiac output and reduced systemic vascular resistance. There is decrease in albumen levels predisposing to pulmonary oedema due to hyperpermeability of capillaries and increased minute ventilation leading to fluid shift to third space. The respiratory system changes bring mild respiratory alkalosis with mild metabolic acidosis with little compensatory reserve in sepsis. All the more the interpretation of physiological limits is individually variable and ill defined.

44.5 Sepsis Microbiology

The causative organisms are a spectrum of aerobic Gram-positive as well as Gram-negative bacteria besides anaerobic infections.

The common **aerobic Gram-positive bacteria** are beta-haemolytic streptococci—groups A, B and D, *Streptococcus faecalis*, *Staphylococcus aureus* and enterococci. The **Gram-negative** intestinal bacilli are *Escherichia coli*, *Proteus mirabilis* and *Klebsiella pneumoniae*. The **Gram variable** organism is *Gardnerella vaginalis*. The **anaerobic group** includes commonly *Peptococcus*, *Peptostreptococcus*, anaerobic streptococci, *Bacteroides fragilis*, Bovis and fusobacteria. Some uncommon infections of **Clostridia group** as *Cl. welchii* and *Cl. tetani* and **others** from *Mobiluncus* species and *Prevotella* species may occur. Some **specific organisms** in obstetrical practice are *Chlamydia trachomatis*, *Mycoplasma hominis* and *Neisseria gonorrhoeae*.

44.6 Sepsis Pathology

The normal flora inhabiting the lower genital tract or neighbouring urinary or anal tract acts as an inoculum on the devitalized, traumatized mucosal surface or raw placental site, which promotes bacterial proliferation and spreads into the bloodstream to manifest as clinical infection.

Puerperal infection is usually multimicrobial causing *endometritis/endomyometritis* originating from sloughed out infected decidua, placental site and underlying myometrium. It spreads out via the bloodstream and lymphatics and by contiguity. There occurs inflammation of pelvic cellular tissues as well as exudation and pus formation presenting as infected adnexal masses. The resultant *parametritis* may spread as *pelvic cellulitis* and *peritonitis* and form *pelvic abscess* with a collection in the pouch of Douglas, trying to spread into generalized peritoneal cavity as well as subhepatic region under the diaphragm. The salpingitis whether *perisalpingitis*, *interstitial salpingitis* or sometimes *endosalpingitis* may form *tubo-ovarian abscess* and rupture. The *broad ligament abscess* may point towards inguinal ligament. The severe form of infection may exhibit as *septicaemia and septicaemic endotoxic shock*. The exotoxins of *Staphylococcus aureus* in puerperium may cause *toxic shock syndrome*. At times, *necrotizing fasciitis* may occur due to Group B haemolytic streptococci or due to polymicrobial infection.

44.7 Pathogenesis

It is related to interaction of host factors with infecting organisms and type of procedural interference. The mechanism is a host response initiation leading to hyperinflammation as systemic inflammatory response syndrome (SIRS) stimulating resultant compensatory anti-inflammatory response of complement system and coagulation cascade. This provokes acute phase reaction with leucocyte recruitment and oxidative stress. There occurs leukocyte apoptosis with microcirculatory dysfunction and metabolic alterations of the autonomic nervous system causing neuroendocrinal reaction and multiorgan dysfunction or failure.

The host factors are *local tissue factors* which are related to tissue necrosis or haematoma resulting in local hypoperfusion and resultant loss of

tissue integrity. Various cellular energy processes are disturbed resulting in damage of local endothelial, parenchymal or immune competent cells, thus affecting membrane permeability; cellular electrolyte transfer involving Na, K and calcium; as well as release of neurotransmitters or hormones. This activates various cytokines, lipid peroxidation and release of free radicals and enzymes like phospholipases, proteases and endonucleases resulting in membrane disintegration and DNA damage. This causes apoptosis and necrosis of local endothelial, parenchymal and immune cells. The *systemic factors* are related to severity of tissue insult, haemorrhagic shock as well as insufficient lactate clearance. The *injury factor* depends on site of injury like abdominal or vaginal, so type of mutilation, extent of tears, lacerations and operative interventions are important factors. The *therapeutic factors* include any delay in recognizing injury, delayed institution of antibiotics and delay in doing reparative procedures. The other issues are need of ventilation, prolonged ICU stay, massive blood transfusions or occurrence of resistant nosocomial infections. The individual *patient factors* are also related to genetic predisposition to exaggerated inflammatory response and coagulation cascade due to gene polymorphisms of TNF β2/β2 and heat shock protein 70-2A/A genotypes resulting in increased mortality of septic patients. The patient may be diabetic, may be alcohol and drug user or may be suffering from chronic renal insufficiency or chronic obstructive airway disease besides having immunosuppression.

The infective organisms are usually from the perineum and vagina, and the invasion is due to inadequate levels of asepsis observed or improper tissue handling during surgical procedures where tissue devitalization has occurred due to prolonged operative time or where it has already been a handled case from domiciliary settings.

44.8 Risk Factors

They are many *antenatal factors* like miscarriage, prolonged rupture of membrane, chorioamnionitis, preterm labour, poor economic status, malnutrition, obesity, anaemia, diabetes, upper respiratory tract infection in the subject and her close associates, winter season, previous history of pelvic infection, genital tuberculosis, drug abuse and immunosuppression. Some *intrapartum factors* are prolonged labour with ruptured membranes, repeated per vaginum examinations, difficult instrumental delivery, emergency caesarean section, postpartum haemorrhage, manual removal of placenta, exploration for retained products of conception, unhygienic conditions at birth place and lack of aseptic precautions. Many a times the risk factors are more than one, so risk of developing severe sepsis is compounded, e.g. with diabetes and obesity requiring emergency caesarean—the risk of developing severe sepsis is more than the nondiabetic subject. Some *social and demographic factors* prevalent in developing countries in low-resource settings are traditional birth attendant practices, unclean domiciliary childbirth, delayed recognition of sepsis, poor accessibility to healthcare, lack of awareness and social taboos.

44.9 Clinical Symptomatology

Clinical features reflect the severity of underlying pathology. The subject complains of low-grade fever, pain in the lower abdomen and associated uterine tenderness in endometritis. With endomyometritis, the fever may be high grade with rigors with significant tachycardia where per vaginum examination reveals subinvoluted tender uterus with foul smelling lochia. This with parametrial tenderness may represent parametritis where per vaginum examination reveals posterior fornix tenderness, induration and nodularity explaining uterosacral ligament involvement justifying abdominal and pelvic pain both. The excitation pain on cervical motion is indicative of parametritis and pelvic peritonitis. The toxic look of patient with all of the above symptoms and presence of loose stools may suggest pelvic abscess formation which on P/V and P/R examination is seen as a bulge in posterior fornix due to collection in the pouch of Douglas. The most severe presentation is in situation of surgical

complications due to injury of the bowel or bladder, septicaemic shock with multiple organ failure resulting in renal and hepatic impairment and coagulation failure as terminal end disease.

44.10 Septic Shock

It is due to toxins liberated in the bloodstream leading to septicaemia. The first stage is reversible and has two phases. The initial phase is warm phase due to vasodilatation where hypotension is associated with fever 101–105 °F with rigors, tachycardia, tachypnoea and flushed skin. The patient is usually alert. Oliguria may ensue. Leucopenia exists. The latter phase is that of vasoconstriction where the patient has cold clammy skin, subnormal temperature, bradycardia, cyanosis, occasional jaundice, disseminated intravascular coagulopathy (DIC) and oliguria. Leucocytosis exists.

The second stage is irreversible due to prolonged cellular hypoxia resulting in metabolic acidosis, acute renal failure, cardiovascular failure, pulmonary oedema, adrenal failure exhibited as multiorgan dysfunction syndrome (MODS) and finally death.

This needs differentiation from amniotic fluid embolism, pulmonary embolism, adult respiratory distress syndrome and myocardial infarction.

Grossly sepsis can be graded as:

- Grade I—when sepsis is limited to the uterus only
- Grade II—when it is beyond the uterus but limited to the pelvis
- Grade III—when it is beyond the pelvis presenting as generalized peritonitis, endotoxic shock, acute renal failure and jaundice

44.11 Diagnosis

History of exact chronology of the events leading to sepsis is very important and all details questioned from the subject or the relatives. The type of childbirth process, settings in which it was done and also if any complication occurred for which any added intervention was performed are enquired. The information related to urinary and bowel function is recorded. The associated obstetrical complication with preexisting medical or surgical comorbidity should be considered while planning further management. If the patient has been received from outside, the referral notes should be thoroughly checked to note antibiotics given, record of any drug allergy and blood transfusions in view of existing clinical status.

Examination—Thorough general physical examination is performed and vitals are monitored. The urine output and proper bowel function are ascertained. Per abdomen as well as per vaginum examination is done to know pelvic findings related to the vagina, cervix, uterus and adnexa. Per rectum examination is done for any additional information. The derangement of clinical parameters is proportionate to the severity of sepsis and organ damage.

The additional findings of middle ear infection or sinusitis potentiating CNS involvement, rectal or vaginal pain or discharge indicating genital tract infections, upper respiratory tract infections or respiratory symptoms indicating *group A streptococcus* or influenza are important.

The following "red flag" signs and symptoms should be considered important for early diagnosis and assessment for underlying sepsis as per Centre for Maternal and Child Enquiries (CMACE) UK report 2011 [6]. If the woman looks anxious and sick and presents with persistent vaginal bleeding and pain in the lower abdomen in the postpartum period, sepsis should be ruled out first.

The definitive signs of sepsis are:

- Hyperthermia >38 °C or unexplained hypothermia
- Persistent tachycardia >100/min
- Dyspnoea particularly a respiratory rate >20/min
- Pain abdomen or chest pain
- Vomiting and/or diarrhoea
- Pain and tenderness in renal angle

- Significant vaginal discharge/foul smelling infected lochia
- Uterine tenderness

The observations of an inpatient should be marked on a Modified Early Obstetric Warning Score (MEOWS) chart [7] by the resident staff and appropriately observed more frequently, alerting senior obstetricians so that periodical review is done. If early signs are recognized in time, then critical and advanced problematic situations can be avoided and timely intervention instituted by involving senior multispecialty team members from anaesthesia, obstetrics, critical care and microbiology department.

The MEOWS chart drafted after the seventh CEMACH report [8] is commonly used. It has a "track and trigger" system, where, if a parameter falls outside a defined range, a response is triggered. As for monitoring of sepsis, 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 charted 12 hourly.

A trigger is marked as severe, red parameter, or less severe, yellow trigger. The appropriate actions include more intensive and frequent observations with periodical clinical review. The MEOWS parameters are documented as (see Annexure 1):

	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
• Resp. 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	Nonresponsive to pain stimulus/voice	

The response to this observation necessitates accelerating the action for starting oxygen 10 L/min and recording parameters every half an hour. The specialist obstetric and anaesthetic team members can review observations and revise prescription after repeat history and examination. The MEOWS triggers can predict maternal morbidity with a sensitivity of 89% and specificity of 79%. The positive *p*-value was slightly lower at 39%, but the negative *p*-value was as high as 98%, suggesting its beneficial role in recognizing sepsis early in patients with abnormal physiological parameters [7]. This is useful in reducing maternal death. The decision to refer to a tertiary care and intensive management can be made. The referral document should be properly scrutinized and detailed clinical history and examination findings recorded.

The *diagnostic criteria* for infection are as follows as per Surviving Sepsis Campaign—International guidelines for management of severe sepsis and septic shock [9]: *General variables* are:

- Temperature (>38.0 °C), hypothermia (core temperature <36 °C)
- Pulse rate >90/min
- Resp. rate (>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 are:

- WBC count >12,000/μL, WBC count <4000/μL
- Plasma C-reactive proteins value >2 SD above the normal value
- Plasma procalcitonin value >2 SD above the normal

Haemodynamic variables are:

- Arterial hypotension (SBP <90 mmHg, MAP <70 mmHg or an SBP decrease >40 mmHg)

Organ dysfunction variables are:

- 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)
- Serum creatinine level rise >0.5 mg/dL or 44.2 μmol/L
- Coagulation abnormalities (INR >1.5 or APTT >60 s)
- Thrombocytopenia (platelet count <100,000/μL)
- Hyperbilirubinemia (plasma total bilirubin >4 mg/dL or 70 μmol/L)
- Paralytic ileus (absent bowel sounds)

Tissue perfusion variables are:

- Hyperlactatemia (>1 mmol/L)
- Decreased capillary refill or mottling

Features of *severe sepsis* or sepsis-induced tissue hypoperfusion or organ dysfunction/sepsis-induced hypotension are:

- Lactate levels >2 mmol/L
- Urinary 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
- Acute lung injury with PaO_2/FIO_2 <200 in the presence of pneumonia
- Serum creatinine level >2.0 mg/dL (176.8 μmol/L)
- *Serum bilirubin >2 mg/dL (34.2 μmol/L)*
- Platelet count <100,000/μL
- Coagulopathy as international normalized ratio (INR) >1.5

The organ system function is represented as per below:

Central nervous system	Glasgow coma score
Respiratory system	$PaO_2/F1O$
Cardiovascular system	Blood pressure measure
Haematological system	Platelet count
Hepatic function	Serum bilirubin level
Renal function	Serum creatinine level
Gastrointestinal system	No appropriate parameter available

44.12 Investigations

The requisitioned laboratory tests are directed towards appropriate diagnosis, treatment, prognosis and prevention of organ damage.

Complete blood count—Hb, PCV,TLC, DLC, platelet count, blood sugar, liver function tests, renal function tests, thyroid function tests, C-reactive protein, viral markers, serum electrolytes (Na, K, calcium, coagulation profile), prothrombin index, FDP, procalcitonin, serum lactate levels, Rh ABO, peripheral blood film for malarial parasite. Blood is sent for culture sensitivity testing.

Urine tests—Routine albumin, sugar, microscopic examination for pus cells, bile products, urinary protein quantification, calcium creatinine ratio and urine culture sensitivity is done.

Vaginal endocervical swab for culture sensitivity of aerobic and anaerobic bacteria is sent.

Ultrasonography is done for total abdomen and pelvis to ascertain pathology of genital organs like retained products, adnexal masses and pelvic abscess. Colour Doppler for suspected thrombosis of leg and pelvic veins is a useful investigation.

Chest X-ray—reveals atelectasis, pleural effusion, pneumonia, and pulmonary tuberculosis.

CT scan is done to know the extent of local pelvic pathology as well as for interventional purposes.

The biochemical prognostic markers are:

CRP evaluation is done periodically to know response to therapy and in case of deterioration it suggests impending organ failure.

Procalcitonin (PCT)—the elevated levels from D1 to D5 are strongly indicative of sepsis with multiorgan dysfunction.

Cytokines—TNFα, IL-6, IL-8, IL-10 and IL-18 estimations are available in specified laboratory settings only.

44.13 Management Plan

The management approach is for early recognition, aggressive resuscitation and intensive treatment with early antibiotics, source control and repeated review by senior doctors and midwives.

Early recognition of sepsis is the hallmark of effective treatment policy.

The aggressive resuscitation and treatment is instituted.

The Surviving Sepsis Campaign (SSC) 2002 has considerably increased awareness of sepsis management by developing guidelines for evidence-based management to improve outcome and reduce maternal morbidity and mortality [9]. The updated Guidelines 2012 are followed now for assessment and management of subjects with sepsis and septic shock. The Royal College of Obstetricians and Gynaecologists recommends the SSC approach [10] to manage maternal sepsis which offers modified resuscitation bundle to be initiated in the first 6 h of diagnosis of sepsis as below:

(a) To obtain blood sample for blood cultures prior to antibiotic administration
(b) To administer broad-spectrum antibiotic within the first hour of recognition of severe sepsis
(c) To measure serum lactate immediately
In case of hypotension and/or a serum lactate value >4 mmol/L, infuse an initial minimum 20 mL/kg of crystalloid or an equivalent to target a level of mean arterial pressure (MAP) of >65 mmHg.
Apply facial oxygen mask to maintain oxygen saturation.
If hypotension persists despite fluid resuscitation (septic shock) and/or lactate value >4 mmol/L, then target achieving a central venous pressure (CVP) of ≥8 mmHg and a central venous oxygen saturation (SvO2) ≥70%.

The aim of obtaining blood cultures prior to giving antibiotics is that although almost half of the subjects with severe sepsis have a bacteraemia at the time of presentation, it may be further masked by another half if antibiotics are given prior to obtaining the culture sample. So there should be no unnecessary delay in taking sample for blood culture and starting antibiotic therapy. The best approach is to obtain blood sample for culture at the same time while establishing intravenous cannulation in order to give antibiotics. The aseptic precautions to avoid sample contamination should be observed. If the patient has other indwelling catheters and canulas, particularly central venous lines, additional samples should be drawn from these invasive sites. In case of infection, they can be replaced.

The other relevant samples are urine, sputum and vaginal and wound swabs. Sometimes breast milk, throat swabs, cerebrospinal fluid and stool samples are also taken.

The choice of antibiotic is made with the aim to cover all common infecting aerobic Gram-positive, Gram-negative and anaerobic organisms. Early goal-directed antibiotic therapy is instituted [11].

Where the organism is not specified yet and the subject is still not critically ill, the first choice is:

- Co-amoxiclav (1.2 g 8 hourly) plus *metronidazole* (500 mg 8 hourly)
  Ampicillin (1 g 6 hourly) plus *metronidazole* (500 mg 8 hourly)
  Cefuroxime (1.5 g 8 hourly) plus *metronidazole* (500 mg 8 hourly)
  Cefotaxime (1–2 g 6–12 hourly) plus *metronidazole* (500 mg 8 hourly)
- Clarithromycin (500 mg 12 hourly) with sensitivity to penicillin and cephalosporins
  Clindamycin (600 mg to 1.2 g by intravenous infusion three or four times daily)
- Gentamicin injection (3–5 mg/kg body wt daily in divided doses every 8 hourly slowly)

For severe sepsis or septic shock [12]:

- Piperacillin–tazobactam (4.5 g 6 hourly) Ciprofloxacin (600 mg 12 hourly) *plus gentamycin*
- Meropenem (500 mg to 1 g 8 hourly by IV injection over 5 min) plus *gentamycin*

For suspected group A streptococcal infection, give:

- Clindamycin (600 mg to 1.2 g by intravenous route three or four times daily) is more effective than penicillin as it inhibits exotoxin production.

The antibiotic cover is continued for 2 weeks or till complete cure is achieved under vigilant control of culture sensitivity reports or microbiologist's opinion. The spectrum initially started as broad spectrum can be converted to narrow spectrum for rational use and can be changed if clinical response is not observed.

The fluid resuscitation using boluses of 20 mL/kg should be started as early as possible, with some patients requiring repeated fluid boluses to achieve MAP of 65 mmHg as a target. While intravascular depletion may cause persistent hypotension, other possible causes such as loss of vasomotor tone and myocardial depression as a result of septic mediators should be considered. Other significant alternative reasons include haemorrhage, oxytocic drugs and renal failure.

While achieving MAP >65 mmHg is an ideal aim, other significant targets to fluid resuscitation are serum lactate levels, skin perfusion, mental status and more importantly urinary output. The optimal MAP target in pregnant women with sepsis is not defined, and presuming that the maternity subjects are usually young having fewer comorbidities than other older septic patients, it is possible that lower MAP values may be well tolerated. In the absence of strong clinical evidence to support, it is fairly justifiable to **target a MAP of 65 mmHg.**

The raised serum lactate level indicates inadequate tissue oxygenation, and a level of >4 mmol/L is associated with poor outcome reflecting either severe type of illness or inadequate treatment even in the absence of hypotension.

The vasopressors may be needed in case of emergent life-threatening hypotension even when circulating volume has not yet been adequately restored but are mostly used when hypotension persists, despite adequate fluid resuscitation. **Noradrenaline** administration as an infusion is recommended to aid perfusion pressure, targeting a MAP of 65 mmHg, under vigilance of critical care intensivists. Second-line agents include **adrenaline and vasopressin**, which should only be used in specialist critical care areas. **Dopamine** is not recommended for routine use but may be used as an alternative agent in some patients having bradycardiac side effects of noradrenaline. In patients with impaired cardiac output resulting from myocardial dysfunction, **dobutamine** is beneficial to aid with inotropy.

The source control is the process of specific management of the focus of infection, and it requires surgical intervention or procedure for sepsis reduction:

- Plan early delivery of baby for chorioamnionitis.
- Do evacuation of the uterus if retained products of conception seen.
- Do manual removal of placenta for retained placenta.
- Remove foreign body as early as possible.
- Do laparotomy for uterine or bowel injury (usually postpartum).
- Do debridement for wound infection and antiseptic dressing.
- Do needle aspiration/incision and drainage for severe mastitis/breast abscess.
- Do incision and drainage for perineal/pelvic abscess.
- Do repair of genital tract injury.

Other aspects of supportive therapy in severe sepsis which need attention are:

- Glucose/fluid control
- Electrolyte balance
- Prophylaxis for venous thromboembolism
- Prophylaxis for stress ulcer
- Avoiding or treating anaemia as needed
- Corticoid therapy
- Intravenous immunoglobulin therapy

The patient management area as HDU/ ICU will depend on several factors including severity of sepsis, evidence of one or more organ failures, stage of pregnancy or labour and availability of infrastructure and provision of facilities including skilled medics and paramedics in critical care.

The indications for transfer to critical care in maternal sepsis are as below:

- Cardiovascular system—where persistent hypotension or lactatemia exists despite adequate fluid resuscitation necessitating the need for vasopressor or inotropic therapy
- Respiratory system—with pulmonary oedema, requirement of mechanical ventilation and airway protection
- Renal system—where severe acute kidney injury is recognized and dialysis is warranted
- Neurological system—when conscious level of the subject remains low
- Multiorgan failure, uncorrected acidosis and persistent hypothermia

Preventive obstetrics is a new concept based on awareness and knowledge of all measures of general infection control applicable to site, subject and procedure essential to reduce maternal sepsis:

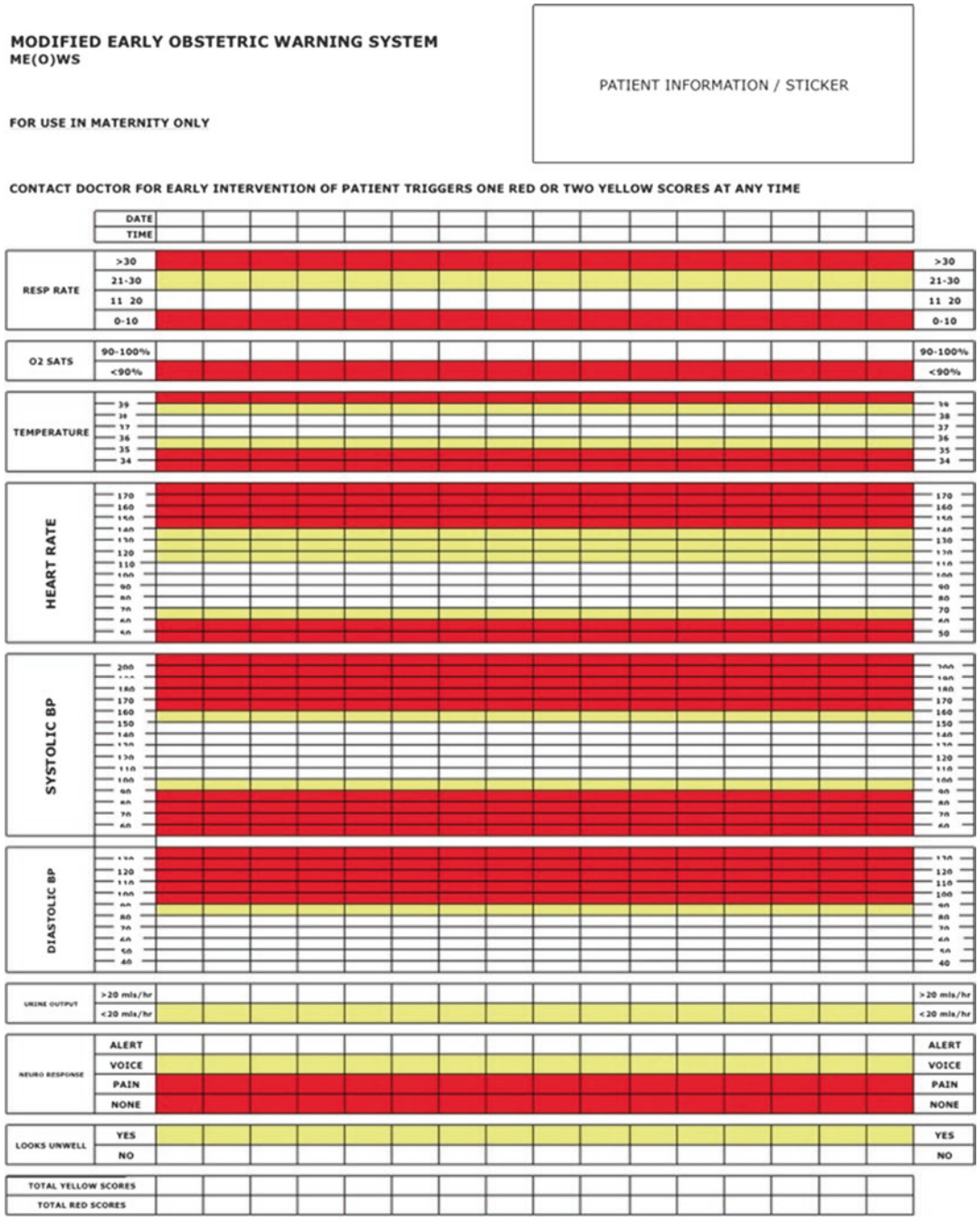
- Good nutritional support and anaemia prevention or treatment in antenatal period
- Hospital infection control measures including environmental cleanliness
- Handwashing/hygiene
- Avoidance/early detection of infection, prevention of nosocomial infections
- Asepsis during procedure, clean equipment, barrier nursing of infected subjects
- Antibiotic prophylaxis before surgical intervention
- Screening and treatment for *group B streptococci* colonization, bacterial vaginosis
- Treatment of chorioamnionitis before and during labour
- Clinical monitoring and management to avoid prolonged labour
- Reduction of obstetrical complications during instrumental deliveries
- Behavioural and organizational change with strong implementation policy
- Issue of hospital guidelines and antibiograms
- Training of medical and paramedical staff
- Periodical audit/quality improvement

Prophylactic antibiotic 1 h prior to caesarean delivery reduces risk of infection by 70–80%. Appropriate management of pregnancy-related conditions like chorioamnionitis, endometritis, septic incomplete abortion, wound infection and mastitis is recommended. Some other nonpregnancy-related comorbidities affecting sepsis severity like influenza, pneumonia, pharyngitis, appendicitis, cholecystitis, pyelonephritis, meningitis, etc. should be treated vigorously.

44.14 Conclusion

Sepsis can be identified early—from its clinical presentation related to its underlying condition. The severity of sepsis is variable depending on many factors. The early recognition and management can prevent serious maternal morbidity and mortality. The preventive community obstetrics has a definitive role in improving general health of women. The need of critical care units and skilled staff is important for survival of severe sepsis subjects in shock or with multiorgan failure.

Annexure 1: The MEOWS Chart



References

1. Arulkumaran N, Singer M. Puerperal sepsis. Best Pract Res Clin Obstet Gynaecol. 2013;27:893–902.
2. Timezguid N, Das V, Hamdi A, et al. Maternal sepsis during pregnancy or the postpartum period requiring intensive care admission. Int J Obstet Anaesth. 2012;21(1):51–5.
3. Kaye DK, Kakaire O, Osinde MO. Systematic review of the magnitude and case fatality ratio for severe maternal morbidity in sub-Saharan Africa between 1995 and 2010. BMC Pregnancy Childbirth. 2011;11:65.
4. WHO Fact sheet: Countdown to 2015 decade report (2000–2010) WHO 2010. www.who.int/mediacentre/factssheets/fs348.
5. Final pilot report-ICMR—Estimates of maternal mortality ratios in India and its states—a pilot study. www.icmr.nic.in/finalpilotreport.pdf. July 2003.
6. Centre for Maternal and Child Enquiries (CMACE). Saving mother's lives: reviewing maternal deaths to make motherhood safer: 2006–08. The eighth report on confidential enquiries into maternal deaths in United Kingdom. BJOG 2011;118 Suppl 1:1–203.
7. Singh S, McGlennan A, England A, Simons R. A validation study of the CEMACH recommended modified early obstetric warning system (MEOWS). Anaesthesia. 2012;67:12–8.
8. Centre for the Enquiry into Maternal and Child Health (CEMACH). Saving mothers' lives: reviewing maternal deaths to make motherhood safer 2003–2005. In: Lewis G, editor. The seventh confidential enquiry into maternal deaths in the United Kingdom. London; 2007.
9. Dellinger RP, Levy MM, Rhodes A, et al. Surviving sepsis campaign: International guidelines for management of severe sepsis and septic shock. Intensive Care Med. 2013;39(2):165–228.
10. Royal College of Gynaecologists. Bacterial sepsis in pregnancy. Green-top guideline No. 64a. 2012.
11. Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med. 2001;345:1368–77.
12. Barton J, Sibai B. Severe sepsis and septic shock in pregnancy. Obstet Gynecol. 2012;120:689–706.

Secondary PPH

45

Kumud Bala Gupta and Anshu Kakkar

45.1 Introduction

Postpartum haemorrhage is the most common cause of maternal mortality worldwide. Most cases of morbidity and mortality due to PPH occur within the first 24 h of delivery. However, the jeopardy of PPH is rising with the secondary form of PPH occurring between 24 h and 12 weeks postpartum, when the woman is already discharged home. As what has been reported by many studies, women presenting with secondary postpartum haemorrhage usually do so during second postpartum week, with the next largest proportion during the third week. In developed countries, secondary postpartum haemorrhage occurs in <1–2% of pregnancies.

45.2 Definition

Postpartum haemorrhage can be divided into two categories. Primary postpartum haemorrhage is defined as blood loss equal to or greater than 500 mL within the first 24 h after birth. Blood loss greater than or equal to 1000 mL is labelled as severe postpartum haemorrhage. Secondary postpartum haemorrhage is defined as any abnormal and excessive bleeding from the birth canal occurring after 24 h and 12 weeks postnatally [1]. It is much less common than primary PPH, occurring in about 1% of deliveries. The majority of cases occur within 3 weeks of delivery. The amount of bleeding is usually less than primary PPH. Definition of secondary PPH does not include volume of blood loss or the condition of women, it may vary from mild inconvenience to fatal.

45.3 Causes

The four main causes of secondary PPH can be summarized as 4 'T's which include tissue (retained placenta, placenta accreta), tone (atonic uterus, subinvolution at placental site), trauma of genital tract (vaginal, cervical laceration, uterine rupture or vulval haematoma) and thrombogenic disorders (Von Willebrand's disease, carrier of haemophilia A or B, factor XI deficiency or use of anticoagulants, e.g. warfarin). Extremely rare causes also have to be considered, including trophoblastic disease, chronic uterine inversion and the development of false aneurysm or arteriovenous fistula at the site of a healing caesarean section scar [2]. Placental site vessel subinvolution is also one of the rare causes of secondary PPH, and this situation is frequently underdiagnosed by clinicians.

K. B. Gupta (✉)
Tenzin Hospital, Shimla, Himachal Pradesh, India

Department of Obstetrics and Gynaecology, Shimla, Himachal Pradesh, India

A. Kakkar
Shri Guru Ram Rai Medical and Health Sciences, Dehradun, Uttarakhand, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_45

45.4 Risk Factors

Risk factors for PPH include grand multiparity, multiple gestation, preeclampsia, IUGR, previous spontaneous miscarriage or retained placenta in previous pregnancies. The conditions associated with abnormal maternal trophoblastic interactions have higher tendency of retained products and secondary PPH [3]. Immediate PPH is a risk factor for secondary PPH. Hence, it is likely that risk factors for primary PPH are also risk factor for secondary PPH.

45.5 Diagnoses

Secondary PPH is a diagnosis of exclusion. It usually presents 7–14 days after delivery and may present as slight to excessive bleeding. Small amount of bleeding may persist for several weeks after delivery; therefore some bleeding defined as secondary PPH may be normal [4]. It is important to exclude normal resumption of menstrual period after childbirth, common side effect of hormonal contraception given during this period.

45.6 Management

The management of patient with secondary PPH includes stabilization of patient and investigation for cause. If the bleeding is mild and settling, the uterus is not tender and is appropriately involuted, there are no other signs of sepsis, initial observation is justified. Ultrasound may help this decision if it suggests that the uterus is empty and without retained placental tissue. In the patients with heavy bleeding or signs of sepsis, the primary treatment includes uterotonics and antibiotics. Surgical intervention is only needed in the patients where bleeding is uncontrolled and should be done after appropriate antibiotic cover for at least 24 h [5].

Detailed history should be taken regarding antenatal high risk factor, obstetric history, labour events, mode of delivery, intrapartum or postpartum complications and postpartum contraception, history of fever, pain in the abdomen and amount of bleeding. Antenatal and delivery records should be checked. It is important to know the place of delivery, especially in low-resource areas as the deliveries conducted at home are more often associated with retained placenta and endometritis.

Examination includes temperature, pulse and blood pressure, clinical assessment of anaemia, abdominal distention, uterine involution, tenderness and tone, amount of bleeding, foul-smelling lochia, healing of episiotomy or perineal tear. The lower genital tract and cervix should be carefully inspected under anaesthesia for any laceration and discharging haematoma. Complete haemogram and high vaginal swab should be sent for investigation. Ultrasound is helpful in diagnosing retained product of conception; however, it may not be accurate, so it should be overruled by clinical consideration. If the bleeding is mild and settling and there are no signs of sepsis, initial observation is justified. The patients with heavy bleeding, subinvoluted uterus and signs of sepsis require intravenous fluid replacement with crystalloid, uterotonic drugs and broad-spectrum antibiotics to cover gram-positive, gram-negative and anaerobic organisms. Patients with anaemia or heavy bleeding may also require blood transfusion.

In case of retained placental tissue (found in 1/3 of cases), uterine exploration under anaesthesia is required after antibiotic cover. Usually the cervix is open enough to admit finger and uterine cavity can be explored. The products can be removed by sponge forceps followed by gentle suction curettage. However, since puerperal uterus is soft, it is prone to perforation. The tissue removed should be sent for culture and sensitivity as well as histopathology to rule out trophoblastic disease [6].

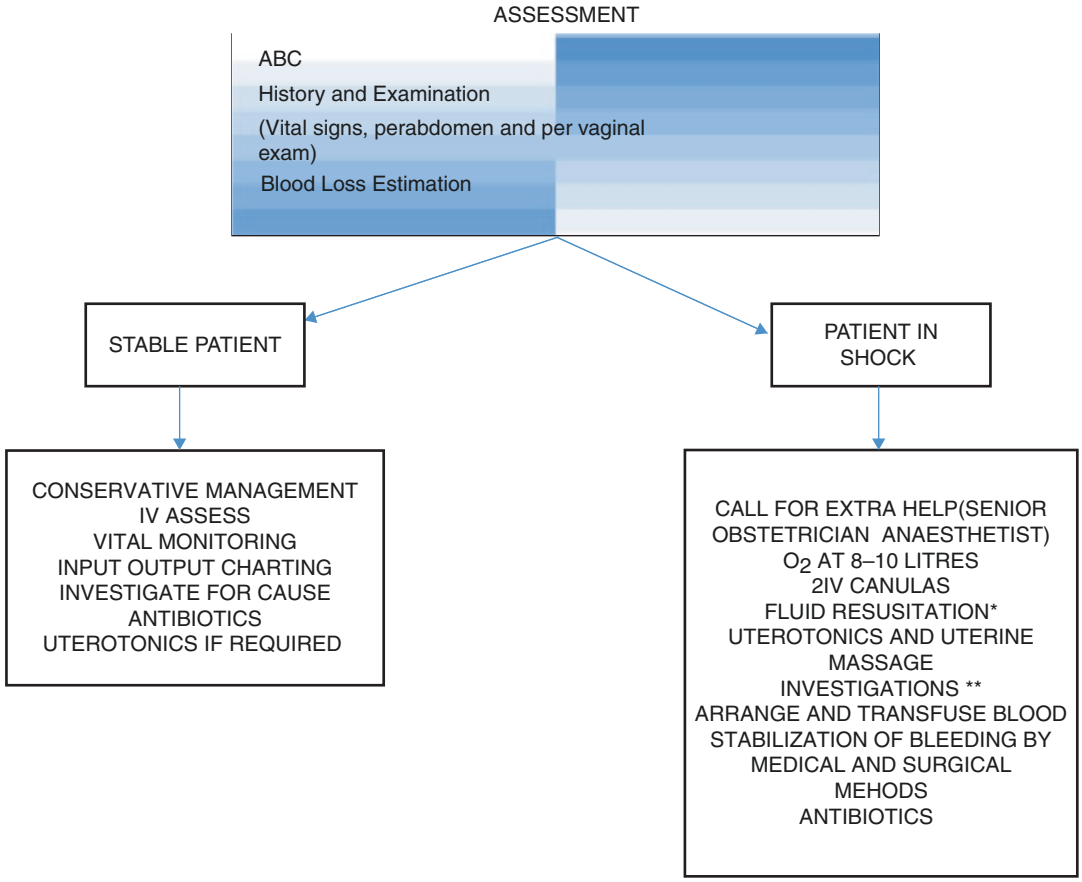
Secondary PPH from dehiscent lower segment caesarean section incision is certainly a rare condition. The possible causes as mentioned in the literature can be defective drainage leading to marked uterine distension followed by dehiscence or infection. Uterine packing is done to

control the haemorrhage; however, it is associated with recurrence of bleeding after removal of pack. Supravaginal hysterectomy is definitive and the safest treatment in such rare cases.

Various studies have shown that conservative medical approach for secondary PPH is superior to surgical treatment as the letter is associated with increased rate of secondary infertility.

45.7 Conclusion

Secondary postpartum haemorrhage occurs in just 1% of women, is associated with primary postpartum haemorrhage and retained placenta and may result in significant maternal morbidity. This problem deserves more attention than it has received in recent years.



Secondary PPH Flow Chart

References

1. Who guidelines in the management of postpartum haemorrhage and retained placenta.
2. South Australian Perinatal Practice Guidelines. Secondary Postpartum haemorrhage.
3. Feigenberg T, Eitan Y, Sela HY, Elchalal U, Ben-Meir A, Rojansky N. Surgical versus medical treatment of secondary postpartum haemorrhage. Acta Obstet Gynecol Scand. 2009;88(8):909–13.
4. Heys RF. Secondary postpartum haemorrhage after caesarean section. Br Med J. 1973;2(5861):308.
5. Edhi MM, Aslam HM, Naqvi Z, Hashmi H. Postpartum haemorrhage causes and management. BMC Res Notes. 2013;6:236.
6. Hoveyda F, Mackenzie IZ. Secondary postpartum haemorrhage: incidence, morbidity, and current management. BJOG. 2001;108:927–30.

46 Blood and Blood Product Transfusion

Suchitra N. Pandit and Deepali P. Kale

46.1 Introduction

Obstetric hemorrhage contributes to major causes of maternal mortality; it may be life-threatening in 1–2% of the deliveries [1, 2].

Delay to recognize the severe hemorrhage or underestimation of the blood loss often leads to inadequate replacement of blood and blood products further leading to development of disseminated coagulopathy, adding risk to maternal life. Also delayed treatment increases the chance of patient to undergo multiple transfusions.

The key measure in the management of major obstetric hemorrhages is transfusion of blood and blood products. However, the decision about time of transfusion and whether to transfuse or not to transfuse the bleeding patient can sometimes be complex. It is obviously not without the risks, so the transfusion has to be justified in all cases.

A better understanding of the risks of transfusion over the last century has given a boost for the developments of more sophisticated donor testing like pre-transfusion infection screening tests, pre-transfusion testing, recipient identification, and improvements in blood component characteristics and quality (e.g., leukoreduction, irradiation, pathogen inactivation).

The transfusion needs can be curtailed by optimizing the hemoglobin levels in the antenatal period thereby reducing the occurrence of anemia, prompt management of third stage of labor, and use of prophylactic measures for minimizing anticipated blood loss in cases of placenta previa.

46.2 Measures to Minimize the Risks of Transfusion

- Screening for anemia is to be offered during the admission and at 28 weeks in singleton gestations and an additional full blood count at 20–24 weeks to be done for women with multiple gestations [3, 4] as recommended by the National Institute for Health and Care Excellence (NICE).
- Hemoglobin level optimization in the antenatal period remains the main strategy for reducing the need for blood transfusion. Anemia in pregnancy is defined as hemoglobin levels of less than 11 g% in the first trimester, 10.5 g%, and less than 10 g% in the postpartum period.
- Oral iron on trial basis for 2 weeks is the initial step in management of normocytic normochromic anemia and warrants further evaluation if no rise in hemoglobin levels in a compliant patient. Antenatal use of oral iron along with or without folic acid has shown

S. N. Pandit (✉)
Kokilaben Dhirubhai Ambani Hospital and Research Hospital, Mumbai, Maharashtra, India

D. P. Kale
Seth G.S. Medical College and Nowrosjee Wadia Maternity Hospital, Mumbai, Maharashtra, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_46

50% reduction in anemia in the third trimester of pregnancy [5].
- If the patient at term does not respond to oral iron, parenteral iron is subsequent line of management. The advantages are shorter duration of therapy and rapid response compared to oral iron therapy [5].
- Anemic patients are at the risk of antepartum and postpartum hemorrhage.
- Blood loss in the third stage of labor can be avoided by active management of third stage.
- Delivery in an equipped hospital with blood bank and resuscitation facilities is recommended for women who are at a high risk of hemorrhage [5].

46.3 General Principles of Blood Transfusion

1. **Consent**
   - Valid consent should be obtained wherever possible before resuming blood transfusion. In case of emergency situation, information about blood transfusion should be given retrospectively as it may not be possible to obtain the consent before the transfusion.
   - The discussion about the need for transfusion and the consent should be documented in the patients' clinical record sheets.
   - In case of Jehovah's witness the patients who decline transfusion of specific blood components need to be documented in case records. The discussions should be conveyed to all the staffs and the clinicians [6, 7].
2. **Requirements for Group and Screen Samples and Cross Matching**
   - Blood group and antibody status determined at the time of antenatal registration and at 28 weeks for all women [8, 9].
   - Samples used in pregnancy for screening and blood group and typing should preferably be less than 3 days old.
   - High-risk women like those having low-lying placenta or morbid adhesions of placenta with no significant alloantibodies should be the candidates for anticipated transfusion.
   - Weekly group and screen samples should be sent to exclude or identify new antibody formation and to ensure the availability of blood if the need arises.
   - Maternal alloantibodies can cause hemolytic disease in newborn and are of importance in selection of blood for transfusion in the mother. Care should be taken to avoid the risk of hemolytic transfusion reactions [10].
   - Transfusion or pregnancy may stimulate an unexpected antibody production against the red cell antigen by mounting primary or secondary immune response [11].
   - A 3-day rule can be formally deviated in pregnant women without clinically significant alloantibodies. In the case of major obstetric emergencies like placenta previa, once-a-week testing can be done to check for alloantibodies.
   - Close liaison with the hospital blood bank is necessary.
3. **Specification of Blood Products in Pregnancy and Puerperium**
   - Patient should receive compatible red cells of ABO, RhD, and K (Kell) types and should be transfused appropriately.
   - Blood components should be screened for CMV (cytomegalovirus).
   - Antenatal women who are Rh-negative should be given Rh-negative blood or blood components to curtail the risk of Rh isoimmunization [11].
   - Immediate issue of O-negative, RhD-negative, and K-negative units should be requested in case of major obstetric hemorrhage, and their provision should be ensured in case of emergency situation till the group-specific blood is available.
   - Development or detection of atypical red cell antibody is done by screening for these antibodies. The relevant antibody or antibodies are detected by specific further testing. A specific red cell unit negative for red cell antigens is then selected for transfusion.

4. **Intraoperative Cell Salvage**
   - Intraoperative cell salvage (IOCS) is reserved for non-obstetric patients where the anticipated blood loss induces anemia or where expected blood loss exceeds 20% of estimated blood volume [12–14].
   - Current evidences recommend the usage of IOCS in obstetrics [15, 16]; however the routine practice of IOCS in obstetrics needs to be supported by more evidence from randomized controlled trials.
   - Cell salvage should be performed by an experienced team.
   - Injection anti-D 1500IU should be administered if cell salvage is used during cesarean section in RhD-negative non-sensitized women and fetal blood group is positive (cord blood).
   - A need for more anti-D is assessed post-transfusion by estimation of FMH >30–40 mL performed on maternal blood sample.
5. **Obstetric Hemorrhage Management with Blood Components**

Measures to Prevent Postpartum Blood Loss

Postpartum blood loss can be reduced by means of mechanical measures like allowing the placenta to get separated and expelled spontaneously.

Uterine atony, bimanual uterine compression, and uterine packing are the primary measures to be taken in case of obstetric hemorrhage along with active management in the third stage of labor.

Meanwhile the pharmacological agents like oxytocic, ergometrine, and prostaglandin analogues should be administered.

Obstetricians should be trained with the surgical methods to prevent postpartum hemorrhage like uterine compression sutures and stepwise devascularization of the uterus.

A set of protocols for the management of major obstetric hemorrhage is displayed in the labor ward, and regular drills should be done as a process of audit.

(a) **RBC Transfusion**
- The clinical and hematological judgements should be the basis of RBC transfusion [17].
- The red cell transfusion is usually advised when hemoglobin levels are below 6 g%.
- The organ ischemia, the rate risk of any potential or actual bleeding, the patient's intravascular volume status, and the risk of complications due to inadequate oxygenation are guide for decision to transfuse [18].
- In case of an acute hemorrhage, a patient may have normal initial hemoglobin to begin with, but the hemoglobin level can drop subsequently to a significant level below the normal. Hence clinical judgment is of utmost importance to avoid losing the vital time of resuscitation.
- O- and RhD-negative red cells should be transfused in situations of severe uncontrollable hemorrhage. The risk of incompatibility may arise due to irregular antibodies where the blood group is not known. The working staff must be aware of all the available blood group types in the blood bank fridge.
- **Components Description**
- Red blood cells are derived as RBC concentrate from whole blood donations. It is prepared by centrifugation or collected by apheresis method. The addition of citrate (anticoagulant) along with one or more preservative solutions gives hematocrit ranging from approximately 50–65% to 65–80%.
- An average of about 50 mL of donor plasma (147–278 mg of iron) mostly in the form of hemoglobin, with additions of preservatives and anticoagulant constitute the RBC concentrate [19, 20].
- **Recommendations**
- The aim to maintain hematocrit is minimally at 21–24%. Majority of protocols

recommend 6 units of packed red blood cells be prepared [21–24].
- In a 70 kg patient, a single unit of PRBCs would raise the hematocrit by approximately 3–4% [25].
- However, due to rise of expanded blood volume during pregnancy, the expected increase in hematocrit may be slightly less.

(b) **Platelets**
- A platelet count below 50×10^9/L in an acutely bleeding patient is an indication for transfusion of platelets which is a critical level of platelets essential for hemostasis.
- Platelet count can be used as a only guide if patients clinical condition warrants further platelet transfusion.
- However platelet transfusion may be decided to be given at count of 75×10^9 to be on a safer margin if there is ongoing hemorrhage [26].
- Platelet availability should be confirmed, and communication with the respective blood bank should be maintained.
- The ABO blood group platelet concentrate is preferably given as same group of recipient. Some studies have demonstrated that transfusion of non-identical platelets has been associated with the poor increments in the platelet counts which is clinically insignificant in terms of hemostatic effectiveness [27].
- When there is limited supply of platelet concentrate or when HLA-matched platelets are required and the best match is not ABO compatible in such scenario, transfusion of ABO non-identical platelets may be acceptable [27].
- To avoid development of anti-D antibodies, RhD-negative platelet concentrate should be given to RhD-negative women [27].
- Anti-D immunoglobulin 250 IU is given if RhD-positive platelets are transfused to RhD-negative women. It is sufficient for covering the possible sensitization by five adult therapeutic doses of platelets. It should be administered within a period of 6 weeks [27].
- **Administration**
- A separate set used for platelet transfusion is recommended [28].
- **Description of Components**
- The standard equivalent of 6 units of whole blood-derived pooled platelets is one platelet pheresis unit. It can increase the platelet count in a 70 kg patient by approximately 40–50,000/μL [25].
- **Dosage**
- To prevent or to treat hemorrhage 4–10 units of RDPs or one SDP are indicated as per the requirements to maintain target platelets.
- **Response**
- The platelet count should be repeated between 10 min and 3 h posttransfusion.
- The conditions like presence of fever, sepsis, splenomegaly, severe bleeding, consumptive coagulopathy, HLA alloimmunization, and treatment with certain drugs (e.g., amphotericin B) affect the response to platelet transfusion adversely [29–31].
- Platelet counts are used only to guide the need for transfusion. It should be interpreted according to patients' clinical condition [32].

(c) **Fresh Frozen Plasma (FFP)**
- During major obstetric hemorrhage, advisable dose for FFP is 12–15mg/kg administered in every 6 units of RBC; the results of coagulation tests are a guide for further transfusions.
- Target for prothrombin time (PT) and activated partial thromboplastin time (APTI) is to maintain them below 1.5× normal [33].
- During a bleeding episode as a line of investigations, regular full blood count and coagulation tests should be performed.
- FFP will take 30 min to defrost.
- Simultaneous resuscitation should be continued with volume-expanding fluids or red cells appropriately.
- **Component Description**
- Fresh frozen plasma contains all the coagulation factors. It can be used up to 24 h

after defrost and up to 5 days if relabeled as "thawed plasma."
- Use of FFP and PRBCs in a contemporary manner is recommended during massive hemorrhage.
- One unit of FFP has a volume of approximately 250 mL.
- FFP should contain the functional quantities of all coagulation factors. It must be frozen at -18C or colder within 6–8 h of collection [20, 34, 35].
- The half-life of a specific factor, the pretransfusion levels, the desired posttransfusion levels, and the expected duration of desired levels determine the dose of specific factor when they are used to correct isolated coagulation factor (e.g., factor V or XI) [36–38] .

(d) **Cryoprecipitate**
- In case of major obstetric hemorrhage, standard dose of 2–5 pools of cryoprecipitate should be administered at early stage.
- Further cryoprecipitate transfusions should be guided by fibrinogen levels for which the aim should be more than 1.5 g/L.
- FFP and cyroprecipitate of similar groups as that of recipient should be used. FFP of different ABO are acceptable in case of unavailability provided that it would have high titres of anti-A or anti-B activity [38].
- Anti-D prophylaxis is required if RhD-negative women receive Rh-positive FFP or cryoprecipitate [38].
- Each unit contains 150 mg of fibrinogen for a total of at least 1500 mg in a pool of 10 units in a total volume of approximately 80–100 cc. Cryoprecipitate release from blood bank is generally in groups of 6–10 units.
- Expected increase in the fibrinogen level of a 70 kg patient is by approximately 75 mg/dL with 10 units of cryoprecipitate. It is worth noting that a 10 unit pool represents ten separate donor exposures.
- In the presence of ongoing bleeding and hyperfibrinogenemia, additional units of cryoprecipitate should be used.

46.4 Pharmacological Measures to Manage Major Obstetric Hemorrhage

- **Recombinant Factor VIIa (rVIIa)**
- Factor VIIa is licensed for treatment of inherited bleeding disorders and plays an important role in initiation of blood coagulation process.
- Trauma and Obstetric cases dosage of 60–90 mcg/kg is recommended in trauma and obstetrical [39, 40].
- No evidences are found to support the prophylactic use of rVIIa in order to reduce blood loss for cesarean sections. In case of intractable obstetric hemorrhage, administration of rVIIa should be done in liaison with hematologist consultation.
- **Role of Antifibrinolytics**
- Tranexamic acid as an antifibrinolytic is used to prevent excessive blood loss [41–43].
- **Obstetric Protocols for Managing the Intrapartum Anemia**
- The set of standard criteria should be used for red cell transfusions in women who are not actively bleeding, if hemoglobin is less than 7 g/dL in labor, or during the immediate postpartum period. In the clinical condition, medical history is used to guide decision of transfusion. In postpartum women if hemoglobin is less than 7 g/dL, decision to transfuse is made on an individual basis.
- Prompt identification of the anemia and treatment impacts the reduction of the need for blood transfusion.
- **Women Who Are Not Willing to Accept Blood Transfusion** (Jehovah's Witness)
- These are certain groups of people who refuse blood transfusion for their religious reasons. Management of their pregnancy can pose a significant challenge [44–46].
- All women refusing blood transfusion need multidisciplinary team management.
- Hemoglobin should be optimized to prevent anemia. Early use of iron replacement is indicated for correction of anemia. Parenteral iron is indicated if no response to oral iron is noted.
- Proper documentation of discussion and consent or the refusal of blood and its components

or other transfusion-sparing techniques should be made in the antenatal records.
- Pharmacological, surgical, and mechanical measures should be sought to prevent blood loss and to minimize the need for blood transfusion and subsequent risks associated with it.
- Intraoperative cell salvage can be an option for the patient who refuses allogenic blood transfusion.
- Women must understand the implications of refusing the blood and also should be aware of the mortality data for Jehovah's witnesses compared to the non-Jehovah's witness.
- Women should bear some identification marks like wrist bands labeled as "no blood" so it is communicated to all the staff and treating team.

46.5 Immediate Steps for All Reactions

The transfusion must be stopped immediately; intravenous access must be kept open with 0.9% sodium chloride (Table 46.1).

Table 46.1 Adverse transfusion reactions

Acute reactions	Delayed reactions
Hemolysis-immunologic (acute hemolytic transfusion reaction)	Immunologic: delayed hemolytic transfusion reaction
Febrile nonhemolytic	Graft-versus-host disease
Anaphylaxis	Posttransfusion purpura
Urticaria	Red cell alloimmunization
Transfusion-related acute Lung injury (TRALI), noncardiogenic Pulmonary Edema	Platelet refractoriness
Congestive heart failure	Immunomodulation
Septic complication	Nonimmunologic; iron overload
Hypothermia, hyperkalemia, hypocalcemia	Infections HIV, hepatitis B, hepatitis C, CMV, protozoal infection HTLV, Parvo B19

All transfusion-related reactions must be notified to the blood bank in a written format along with the transfusion reaction section of the blood bag which should be completely filled and sent.

If transfusion is terminated, freshly collected blood and urine samples should be sent to the blood bank. The blood unit and the administration set should be sent to the blood bank.

PBRCS
- Initial request: 4–6 units of RBCs
- O-negative or type-specific blood initially until cross match units are released

FFP
- RBCs to FFP ratio not to exceed 3:2
- Infuse FFP to maintain INR <1.5

Platelets
- Single donor apheresis platelet pack.
- In the face of ongoing hemorrhage, infuse to maintain platelet count >50,000–100,000/μL.

Cryoprecipitate
- Initial request: 10 units' cryoprecipitate if fibrinogen is less than 100 mg/dL
- Additional units to maintain fibrinogen concentration 100–125 mg/dL

46.6 Recombinant Activated Factor VII (rVII)

If available, it is used when there is continued hemorrhage and all other blood replacement therapies have failed (i.e., after the use of 10–12 units of PRBC, 6–12 units of FFP, and 2–3 units of platelets). *It is expensive and not available in all hospitals*.

Summary of Recommendations

- For massive obstetrical hemorrhage, use a ratio of PRBCs to FFP to platelets that is 6

units of PRBC/4 units of FFP/1 unit of pheresis platelets.
- If hemorrhage is ongoing after initial treatment, consider increasing the amount of FFP to a ratio of 4 units of PRBC/4 units of FFP/1 unit of pheresis platelets.
- If there is no response to initial therapy and the bleeding exceeds the expected volume for routine delivery, request laboratory analysis for the following immediately:
  - CBC with platelets
  - PT/PTT
  - Fibrinogen
- Repeat laboratory investigations 1–3 every 30 min until patient is stable.

46.7 Conclusion

The main aim is to provide adequate blood product replacement and to prevent or rectify DIC.

Identification and correction of anemia in the antenatal period may reduce the need for blood transfusion.

The pharmacological, mechanical, and surgical measures to prevent blood loss should also be adopted for the same purpose of preventing intrapartum and postpartum anemia.

To eliminate transfusion, an integrated strategy for blood safety is necessary.

References

1. Bonnar J. Best practice & research. Clin Obstet Gynaecol. 2000;14:1–18.
2. Santoso J, Saunders B, Grosshart K. Massive blood loss and transfusion in obstetrics and gynecology. Obstet Gynecol Surv. 2005;60(12):827–37.
3. Antenatal care for uncomplicated pregnancies NICE guidelines [CG262] Published date: March 2008.
4. Multiple pregnancy: antenatal care for twin and triplet pregnancies Clinical guideline Published: September 2011.
5. Blood Transfusions in Obstetrics (Green-top Guideline No. 47). Published: May 2015.
6. The Association of Anaesthetists of Great Britain and Ireland. Management of anaesthesia for Jehovah's witnesses. 2nd ed. London: AAGBI; 2005.
7. Rainaldi MP, Tazzari PL, Scagliarini G, Borghi B, Conte R. Blood salvage during caesarean section. Br J Anaesth. 1998;80:195–8.
8. Guidelines for the clinical use of red cell transfusion. Br J Hematol. 113:1(2001);24–31.
9. United Kingdom Blood Services. In: Borghi DBL, editor. Handbook of transfusion medicine. 3rd ed. London: The Stationery Office; 2001.
10. Green-top Guideline No. 65. The Management of Women with Red Cell Antibodies during Pregnancy.
11. Milkins C, et al. Guidelines for pre-transfusion compatibility procedures in blood transfusion laboratories. Transfus Med. 2013;23(1):3–35.
12. National Blood Authority Australia. Guidance for the provision of Intraoperative Cell Salvage. Canberra: National Blood Authority; 2014.
13. Joint United Kingdom (UK) Blood Transfusion and Tissue Transplantation Services Professional Advisory Committee (JPAC). Intraoperative Cell Salvage Education.
14. Goodnough LT, Monk TG, Sicard G, Satterfield SA, Allen B, Anderson CB, et al. Intraoperative salvage in patients undergoing elective abdominal aortic aneurysm repair: an analysis of cost and benefit. J Vasc Surg. 1996;24:213–8.
15. Allam J, Cox M, Yentis SM. Cell salvage in obstetrics. Int J Obstet Anesth. 2008;17:37–45.
16. Geoghegan J, Daniels JP, Moore PA, Thompson PJ, Khan KS, Gülmezoglu AM. Cell salvage at caesarean section: the need for an evidence-based approach. BJOG. 2009;116:743–7.
17. Murphy MF, Wallington TB, Kelsey P, Boulton F, Bruce M, Cohen H, et al. Guidelines for the clinical use of red cell transfusions. Br J Haematol. 2001;113:24–31.
18. Practice guidelines for blood transfusion by American Red Cross, 2nd ed. 2007.
19. Brecher M, editor. Technical manual. 15th ed. Bethesda, MD: AABB Press; 2005.
20. Circular of Information for the Use of Human Blood and Blood Components. Prepared jointly by the AABB, America's Blood Centers and the American Red Cross. July 2002.
21. Bonnar J. Best practice & research. Bailliere's Clin Obstet Gynaecol. 2000;14:1–18.
22. Stanford University. Obstetrical Emergency Hemorrhage Protocol. Not Published.
23. Barbeiri R. Emergency Obstetrical Hemorrhage Protocol. Approved and in use in Department of Obstetrics, Women's and Children's Hospital. *ach the US Labor and Delivery Suite*. OBG Manage. 2007;19(7):8–11.
24. Matot I, et al. A survey of physicians' attitudes toward blood transfusion in patients undergoing cesarean section. Am J Obstet Gynaecol. 2004;190(2):462–7.
25. Puget Sound Blood Center. http://www.psbc.org/therapy/index.htm.
26. Fakhry SM, Sheldon GF. Massive transfusion in the surgical patient. In: Jeffries LC, Brecher ME, editors. Massive transfusion. Bethesda, MD: American Association of Blood Banks; 1994.
27. British Committee for Standards in Haematology, Blood Transfusion Task Force. Guidelines for the use of platelet transfusions. Br J Haematol. 2003;122:10–23.

28. British Committee for Standards in Haematology, Stainsby D, MacLennan S, Thomas D, Isaac J, Hamilton PJ. Guidelines on the management of massive blood loss. Br J Haematol. 2006;135:634–41.
29. Anonymous. Royal College of Physicians of Edinburgh consensus conference on platelet transfusion. Transfus Med. 1998;8:149–51.
30. Sacher RA, Kickler TS, Schiffer CA, et al. Management of patients refractory to platelet transfusion. Arch Pathol Lab Med. 2003;127:409–14. 50.
31. Slichter SJ, Davis K, Enright H, et al. Factors affecting posttransfusion platelet increments, platelet refractoriness, and platelet transfusion intervals in thrombocytopenic patients. Blood. 2005;105:4106–14.
32. Matot I, Einav S, Goodman S, Zeldin A, Weissman C, Elchalal U. A survey of physicians' attitudes toward blood transfusion in patients undergoing cesarean section. Am J Obstet Gynecol. 2004;190(2):462–7.
33. O'Shaughnessy DF, Atterbury C, Bolton Maggs P, Murphy M, Thomas D, Yates S, et al. British Committee for Standards in Haematology, Blood Transfusion Task Force. Guidelines for the use of fresh-frozen plasma, cryoprecipitate and cryosupernatant. Br J Haematol. 2004;126:11–28.
34. Nilsson L, Hedner U, Nilsson IM, Robertson B. Shelf-life of bank blood and stored plasma with special reference to coagulation factors. Transfusion. 1983;23(5):377–81.
35. O'Neill EM, Rowley J, Hansson-Wicher M, et al. Effect of 24-hour wholeblood storage on plasma clotting factors. Transfusion. 1999;39(5):488–91.
36. Anonymous. Practice guidelines for perioperative blood transfusion and adjuvant therapies: an updated report by the American Society of Anesthesiologists Task Force on Perioperative Blood Transfusion and Adjuvant Therapies. Anesthesiology. 2006;105:198–208.
37. Dzik WH. Component therapy before bedside procedures. In: Mintz PD, editor. Transfusion therapy: clinical principles and practice. 2nd ed. Bethesda, MD: AABB Press; 2005.
38. O'Shaughnessy DF, Atterbury C, Maggs PB, et al. Guidelines for the use of fresh-frozen plasma, cryoprecipitate and cryosupernatant. Br J Haematol. 2004;126:11–28.
39. Levi M, Levy JH, Andersen HF, Truloff D. Safety of recombinant activated factor VII in randomized clinical trials. N Engl J Med. 2010;363(19):1791–800.
40. Franchini M, Franchi M, Bergamini V, Montagnana M, Salvagno GL, Targher G, et al. The use of recombinant activated FVII in postpartum hemorrhage. Clin Obstet Gynecol. 2010;53:219–27.
41. Sekhavat L, Tabatabaii A, Dalili M, Farajkhoda T, Tafti AD. Efficacy of tranexamic acid in reducing blood loss after cesarean section. J Matern Fetal Neonatal Med. 2009;22:72–5.
42. Gungorduk K, Yildirim G, Asicioglu O, Gungorduk OC, Sudolmus S, Ark C. Efficacy of intravenous tranexamic acid in reducing blood loss after elective cesarean section: a prospective, randomized, double-blind, placebo-controlled study. Am J Perinatol. 2011;28(3):233–40.
43. Movafegh A, Eslamian L, Dorabadi A. Effect of intravenous tranexamic acid administration on blood loss during and after cesarean delivery. Int J Gynaecol Obstet. 2011;115:224–6.
44. Singla AK, Lapinski RH, Berkowitz RL, Saphier CJ. Are women who are Jehovah's Witnesses at risk of maternal death? Am J Obstet Gynecol. 2001;185:893–5.
45. Massiah N, Athimulam S, Loo C, Okolo S, Yoong W. Obstetric care of Jehovah's Witnesses: a 14-year observational study. Arch Gynecol Obstet. 2007;276:339–43.
46. van Wolfswinkel ME, Zwart JJ, Schutte JM, Duvekot JJ, Pel M, Van Roosmalen J. Maternal mortality and serious maternal morbidity in Jehovah's witnesses in the Netherlands. BJOG. 2009;11:1103–8.

Part V

Fetus and Neonate

Neonatal Resuscitation

47

R. K. Kaushal

47.1 Introduction

To resuscitate means to revive from unconsciousness or apparent death. Neonatal resuscitation is an attempt to facilitate the dynamic transition from foetal to extrauterine physiology. Perinatal hypoxia is one of the leading causes of perinatal mortality in developing countries, and birth asphyxia is an important cause of static developmental, neurologic handicap in later life. Ninety percent newly born babies need little or no assistance to begin breathing at birth. Others will need some assistance, and <1% would require more extensive resuscitative measures [1].

47.2 Anticipation and Preparedness

It is possible to anticipate birth asphyxia in a setting of high-risk deliveries [2] (List 47.1). But some unforeseen events may take place, and a depressed baby at birth may come as a surprise. So each and every delivery must be considered a potential emergency, and at each delivery, there should be at least one trained person whose primary responsibility is to take care of the baby and is capable of resuscitation or to initiate resuscitation and call for more skilled available help if required. In case of multiple births, equal number of such trained personnel should be available. All the equipment essential for neonatal resuscitation, in functional order, should be available at all delivery points [1] (List 47.2).

List 47.1: Predisposing Risk Factors for Birth Asphyxia

1. Maternal Factors
   (a) Antepartum
   - Elderly mothers (>35 years)
   - Short stature
   - Systemic medical illness
   - Bad obstetrical history
   - Diabetes mellitus
   - Hypertension, eclampsia
   - Oligo-/polyhydramnios
   - Post-term/multiple gestation/abnormal presentation
   - Maternal medication/substance abuse
   - Rh isoimmunization
   - Unsupervised pregnancy

   (b) Intrapartum
   - Difficult/traumatic/operative delivery
   - Meconium-stained amniotic fluid (MSAF)
   - Prolonged labour/rupture of membranes (24hrs)
   - Precipitate/premature labour
   - Use of narcotics/GA
   - Significant intrapartum bleeding

R. K. Kaushal (✉)
Sri Ram Hospital,
New Shimla, Himachal Pradesh, India

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2. Foetal Factors
   - Intrauterine growth retardation (IUGR)
   - Foetal distress
   - Foetal hydrops
   - Foetal malformations
   - Macrosomia

List 47.2: Essential Resuscitation Equipment and Material

Resuscitation Platform

Resuscitation trolley with baby mattress, overhead heat source and timer (Fig. 47.1)

Fig. 47.1 Resuscitation trolley

Suction Equipment
- Mechanical suction machine with pressure gauge
- Mucus aspirator (standby)
- Meconium aspirator
- Suction catheters 5, 6, 8, 10, 12 and 14F
- Nasogastric tubes

Positive Pressure Ventilation Equipment
- Self-inflating resuscitation bag 200, 500 and 750 cc
- Oxygen reservoir
- Flow-inflating bag
- T-piece resuscitator
- Oxygen source
- Compressed gas source
- Oxygen blender
- Cushioned rim face masks of different sizes
- Pulse oximeter with neonatal probe
- Laryngoscope with straight blades '0' and '1' sizes
- Endotracheal tubes 2.5, 3.0, 3.5, and 4.0 mm
- Stylet, scissors
- Laryngeal mask airway (LMA)

Umbilical Vein Catheterization Equipment
- Surgical blades
- Umbilical vein catheters 3.5 and 5F
- Three-way stopcock

Medications
- Epinephrine
- Volume expanders—normal saline, Ringer's lactate
- Sterile water
- Sodium bicarbonate, naloxone hydrochloride

Miscellaneous
- Hand sanitizer, antiseptic solution
- Syringes of different sizes

- Gloves, clean warm linen, micropore sticking tape
- Cardiac monitor with electrodes
- Stethoscope
- Transport incubator

Sequence of resuscitation: Steps and sequence of resuscitation are based on cycle of **a**ssessment, **d**ecision and **a**ction.

Assessment Before Birth

Look for any of high-risk conditions predisposing for birth asphyxia as per List 47.1. Specially note gestational age, amniotic fluid clear or meconium-stained amniotic fluid (MSAF) and singleton or multiple gestation [2].

Assessment After Birth

Answer three questions by looking at the baby:
Gestation—Is it full term?
Respiration—Breathing or crying?
Muscle tone/Activity—Is it good?

If the answer to all three questions is 'yes', the baby stays with mother for **routine** care, ongoing evaluation and stabilization.

Routine Care

- Provide warmth—place the baby skin to skin on mother's abdomen covered with warm clean linen.
- Clear airway if indicated.
- Wipe the baby dry with warm clean linen.
- Initiate breast feeding.
- Ongoing observation of breathing, colour and activity.

If the answer to any of the above three questions is 'no', the baby will need resuscitation in one or more of the following categories of intervention in sequence (ABCD):

A. **Airway and stabilization—Initial steps**
B. **Breathing—Ventilation of lungs**
C. **Circulation—Chest compressions**
D. **Drug administration—Epinephrine and volume expanders**

At each category of intervention, three signs, respiration, HR and state of oxygenation (colour or preferably Spo2 with pulse oximetry), are evaluated frequently. HR is the most important of the three.

47.2.1 Initial Steps

First 60 s after birth (FGM) is crucial to stabilize, assess and begin ventilation if required [3]. The sequence of initial steps is as follows:

- Provide warmth.
- Position and clear the secretions to establish the open airway.
- Dry, remove wet linen and reposition.
- Stimulate breathing if needed.

Baby is **born** wet, has to be kept uncovered if resuscitation is required and hence is predisposed to hypothermia. So baby should be kept under heat source to maintain body temperature. At the same time, take precautions to avoid hyperthermia. To keep the airway open, baby is put supine with neck in 'sniffing' position, i.e. slightly extended to align posterior pharynx, larynx and trachea in line to allow unrestricted air entry. It is best achieved by putting a rolled towel under the shoulders with an elevation of 2–2.5 cm off the mattress. Airway should be cleared by suction if there is obvious obstruction or there is need for positive pressure ventilation (PPV).

Clear fluid/secretions can be cleaned by wiping the mouth with clean gauze/cloth or suction with bulb syringe or suction catheter. With mechanical suction, the negative pressure should not exceed 80–100 mmHg. Suction **mouth** before **nose** to ensure that there is nothing in the mouth for the newborn to aspirate if she/he gasps, while the nose is being suctioned first. It can easily be remembered by thinking 'M' comes before 'N' in alphabets. If the baby is born through MSAF or meconium is present on baby's skin or in the upper airway, assess whether the baby is vigorous or nonvigorous by observing breathing, HR and muscle tone. Baby is nonvigorous if breathing is depressed or HR is <100 bpm or muscle tone is

weak. In vigorous baby the airway is cleaned as described above. In a nonvigorous baby in addition to upper airway, clearing of trachea by endotracheal suction is required to prevent complications of meconium aspiration. Endotracheal suction in these babies is still practised even though limited data has not established its significant benefit.

Tracheal suction: Clear the mouth and posterior pharynx to visualize the glottis, intubate with appropriate-size endotracheal tube (ET tube), and apply direct suction to the ET tube for 3–5 s as you withdraw the tube. Repeat the procedure as necessary until little or no meconium is recovered or baby's HR indicates that resuscitation with PPV must proceed without delay.

Once the airway is clear, quickly dry the head and body with clean, warm linen to prevent evaporative heat loss. Discard wet linen; rewrap in dry, warm, clean linen; and reposition head in sniffing position. Clearing the airway and drying will provide stimulation for most babies to initiate breathing. If normal breathing is not established at this stage, additional tactile stimulation may be provided once or twice by flicking the soles or gently rubbing the back, trunk or extremities. If the newborn is still not breathing normally, assume the baby to be in terminal apnoea where no amount of further stimulation will work, and proceed to the next step.

Evaluation After Completion of Initial Steps

On completion of initial steps within about 30 s, evaluate respiration, HR and colour or preferably Spo2.

Respiration: Look for cry or normal breathing or laboured breathing or apnoea.

HR: Palpate and count pulse rate at the base of umbilical cord, or listen to heart beat with stethoscope over precordium; count for 6 s, and multiply it with 10 to give quick estimate of HR per minute.

Skin colour: Generally skin colour is a good visible indicator of state of oxygenation. Central cyanosis is suggestive of low blood oxygenation. However an oximeter should be used to confirm the perception of cyanosis because normal transition of blood oxygen saturation from birth level of 60% takes about 10 min to reach 90% at a state of eventual air breathing in a healthy newborn [4] (Box 47.1). Skin colour is also affected by pigmentation. So baby may appear slightly cyanotic for first few minutes after birth. Hence it is recommended to monitor SPo2 with pulse oximeter instead of skin colour in the following situations [1]:

- To confirm perception of cyanosis
- Presence of central cyanosis
- Administration of oxygen to a newborn
- Anticipated resuscitation beyond initial steps
- PPV required longer than few breaths

Monitoring Spo2: Attach pulse oximeter probe in a preductal site, i.e. right wrist or hand, over hypothenar eminence before attaching to the instrument to achieve signals quickly [1]. It will display SPo2 as well as HR. However attaching pulse oximeter should not affect or delay further resuscitation.

Box 47.1: Targeted Preductal Spo2 After Birth

1 min	60–65%
2 min	65–70%
3 min	70–75%
4 min	75–80%
5 min	80–85%
10 min	85–90%

Decision and Action

1. Normal breathing or crying: Keep with mother for routine care.
2. Laboured breathing or persistent cyanosis with HR >100 bpm: Presence of grunting or intercostal recessions or baby working hard to breathe indicates laboured breathing. Such a baby needs **continuous positive airway pressure (CPAP) or** free-flow oxygen if CPAP is not possible.
3. Apnoea or gasping or HR < 100 bpm: Proceed to ventilation of lungs with PPV (Sect. 47.2.2).

How to deliver CPAP: It is a technique to provide positive pressure throughout the cycle of breathing to the airway of a spontaneously breathing baby, so as to prevent the collapse of alveoli at the end of expiration, thereby improving oxygenation and decreasing work of breathing especially in preterm babies with low surfactant. It can be done with flow-inflating bag (not with self-inflating bag) or T-piece resuscitator and appropriate-size face mask. Bag should be connected to a source of compressed gas, oxygen blender and pressure gauge. Desired amount of CPAP can be adjusted through flow controlling valve. The mask is held tightly over baby's face. Generally positive pressure of 4–6 cm is adequate. If CPAP is required for a little longer, use nasal prongs instead of face mask.

Oxygen: In term babies begin with 21% oxygen, if there is no improvement in HR or SPo2, use higher concentration by graded increase to 100% to attain target SPo2 (Box 47.1). In preterm babies, start with 30–90% oxygen, and then titrate its concentration to achieve target SPo2. Avoid giving 100% oxygen for more than a few minutes to prevent oxygen toxicity [2].

How long to give CPAP: Stop CPAP when baby has normal breathing, no cyanosis or maintaining expected SPo2 in room air and HR is persistently >100 bpm. Persistent cyanosis or subnormal SPo2 despite adequate breathing is suggestive of congenital cyanotic heart disease or persistent pulmonary hypertension of newborn (PPHN).

47.2.2 Positive Pressure Ventilation (PPV)

Indications: Apnoea or Gasping or HR <100 bpm or Failure of CPAP

PPV can be delivered with any of the three devices, viz. self-inflating or flow-inflating bags or T-piece resuscitator attached to appropriate-size cushioned face mask. The latter two devices need compressed gas source. Self-inflating bag (Fig. 47.2) is a simple, easy-to-use, handy device which can be used in room air without source of compressed gas. In fact it should be available at all delivery points as a standby if either of the other two in use at a particular place goes out of

Fig. 47.2 Self-inflating resuscitation bag and cushioned face mask

Fig. 47.3 Closed-end oxygen reservoir

order. When attached to oxygen source without and with oxygen reservoir (Fig. 47.3), it delivers 40% and 90–100% oxygen, respectively.

How to Give PPV with Self-Inflating Bag

Assemble the selected appropriate-size bag and face mask, and attach to oxygen reservoir and oxygen source. Attach pulse oximeter if not already in place. Test all components of the equipment for any cracks/leaks and proper functioning by blocking the mask or patient outlet with the palm of your hand. Set the pop-off valve to release at 30–40 cm of water, so as to avoid barotrauma to the lungs in case of inadvertent excess pressure. Position yourself on baby's side or head end to use the device effectively, and leave the chest and abdomen for visual monitoring and easy access for possible further interventions like chest compressions and umbilical vessel canulation. Ventilate in open airway (sniffing) position at a rate of 40–60 breaths per minute following a sequence of 'squeeze': two–three 'squeeze', two–three 'squeeze', etc. with a pressure of 30–40 cm of water for first breath and 15–20 cm for subsequent breaths. Squeeze represents inspiration and two–three represents expiration (inward movement of chest wall).

Indicators of effective ventilation: Rising HR is the first and most important indicator of good response to ventilation and is associated with rise in SPo2. If the response is poor after 5–10 breaths, check for breath sounds on both sides of chest and chest rise with each breath. Poor breath sounds/chest rise indicates inadequate seal between mask-face interface or blocked airway or low inflation pressure. Take proper steps to overcome these problems. It can be remembered by the acronym 'MR SOPA' and addressed in sequence (Table 47.1).

Evaluation, Decision and Action After 30 s of PPV

1. *Rising HR and SPo2*: Continue PPV, and monitor HR, breath sounds and chest movement to avoid over-/underinflation. When HR is stabilized above 100 bpm, reduce the rate and pressure of PPV; observe for effective spontaneous breathing. Discontinue PPV once the HR is persistently above 100 bpm, sustained effective spontaneous respiration and SPo2 in the target range on room air. Keep baby in special newborn care unit (SNCU) or neonatal intensive care unit (NICU) for post-resuscitation care.

Table 47.1 Corrective steps for effective ventilation

Acronym	Corrective step	Aim
M	Mask adjustment	To ensure proper seal
R	Reposition	Head in 'sniffing' position
S	Suction of mouth, nose and pharynx	Clear secretions if any to maintain open airway
O	Open mouth	Slightly open mouth, and lift the jaw forward to ensure open airway
P	Pressure	Gradually increase the inflation pressure to hear B/L breath sounds and chest rise. Increase oxygen concentration
A	Alternative airway	Consider ET tube placement or laryngeal mask airway

2. *HR >60 bpm to <100 bpm*: Continue PPV while monitoring HR and respiratory effort every 30 s. At the same time, ensure effective ventilation, adjust oxygen concentration to meet target SPo2, and also look for any underlying complications. If PPV with mask is continuing for more than 2–3 min, put in orogastric tube; leave it in place to prevent gastric distension which may hamper ventilation or cause aspiration. ET intubation with appropriate-size ET tube should be done at this stage to improve ventilation if not already done.
3. *HR <60 bpm and deterioration of baby's condition*: Continue PPV with 100% oxygen, and begin coordinated chest compressions.

47.2.3 Chest Compressions or External Cardiac Massage

Indication: HR <60 bpm despite at least 30 s of effective PPV.

Basis of chest compressions: Baby with persistent bradycardia has depressed myocardial contractility and thus ineffective circulation resulting in progressive hypoxemia and acidosis. Chest compressions and coordinated PPV with 100% oxygen improve circulation and oxygenation of vital organs including myocardium.

Site of compressions: Lower third of sternum between xiphoid process and imaginary line between the nipples (Fig. 47.4).

Position of the baby and resuscitators: Position is the same as for PPV but ensure firm support to the back. At least two persons are required at this stage: one to perform PPV and the other for chest compressions. It is important that both position themselves and work in a way that each can do effective job without interfering with the other. Person giving PPV stands on side and the other performing chest compressions on the head end so as to leave abdomen unobstructed for securing umbilical line and drug administration if needed.

Technique:

1. Two thumb technique: Here two thumbs are placed side by side or one over the other at the site mentioned above to provide vertical compressions; the hand and fingers encircle the chest to support the back/spine (Fig. 47.4).
2. Two-finger technique: Here the tips of two fingers (nails trimmed), middle finger with either index or ring finger of one hand, are placed perpendicular at the site described above, and the other hand is placed under the centre of back/spine to provide support (Fig. 47.4).

Depth of compressions: Approximately 1/3 of AP diameter of the chest followed by slightly longer release to allow the heart to refill. Do not lift thumbs or fingers completely off the chest to avoid waste of time in relocating compression area or lose control over the depth of compression.

Rate and rhythm of PPV and compressions: PPV and chest compressions are to be coordinated because if both are given simultaneously, it will decrease the efficacy of each other. Ninety chest compressions are to be coordinated with 30 PPVs, making a total of 120 events per minute or one cycle of 3 compressions and 1 PPV (3:1) in 2 s. The chest compressor counts aloud while providing three compressions 'one and two and three' followed by breath, i.e. squeezing the bag by the person giving PPV, in a sequence of 'one and two and three and breathe' and so on.

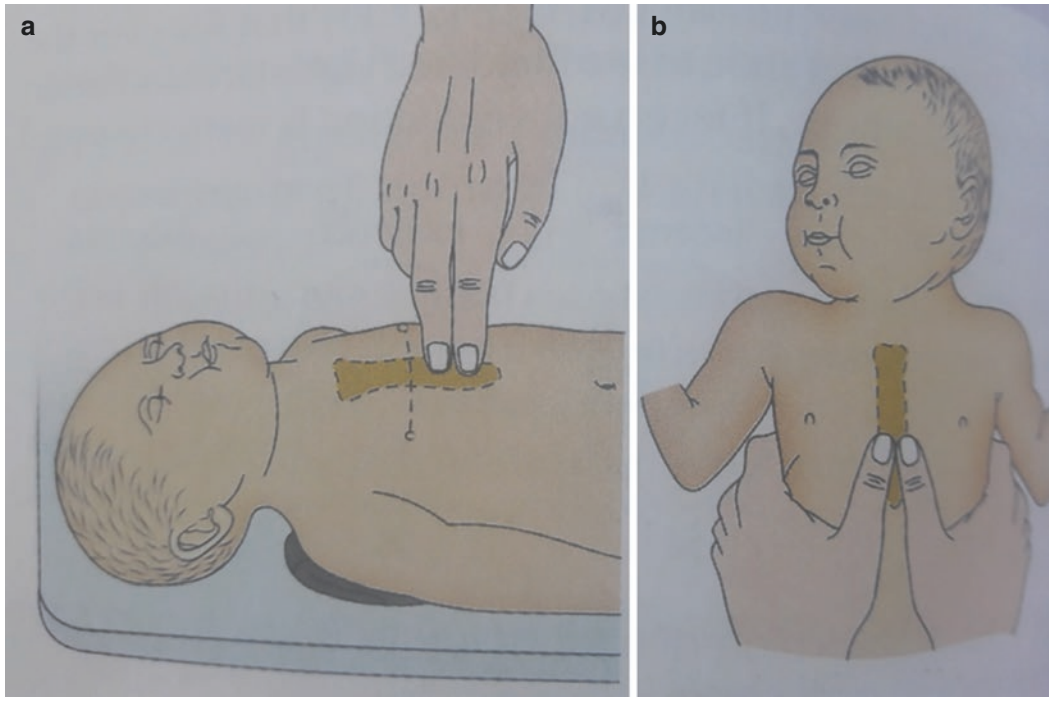


Fig. 47.4 Two-finger (**a**) and two-thumb (**b**) technique of chest compression

Evaluation After 45–60 s of Coordinated Chest Compressions and PPV Assess HR without interrupting chest compressions and PPV.

Decision and Action

1. *HR >100 bpm*: Stop chest compressions and follow as described above under PPV.
2. *HR >60 and <100 bpm*: Stop chest compressions, and continue PPV with 100% oxygen at a rate of 40–60/min, and reassess after 30 s.
3. *HR <60 bpm with no improvement in baby's condition*: Perform chest compression and ventilation corrective steps—**MRSOPA** (Table 47.1); continue coordinated chest compression and PPV with 100% oxygen, and proceed to the next step '**D**', i.e. drug administration. Perform ET intubation if not already done.

Endotracheal intubation: It is relatively a more difficult skill to master and needs frequent practice to maintain success.

Indications for ET Intubation

- Tracheal suction as described earlier.
- PPV over prolonged period.
- PPV with mask not effective despite all corrective steps at any stage of resuscitation.
- Facilitate coordination, and maximize efficacy of ventilation with chest compressions.
- Surfactant administration, suspected diaphragmatic hernia and extreme prematurity.

Selection of laryngoscope, laryngoscope blade and ET tube: Neonatal laryngoscope with straight blade, size '0' for preterm/VLBW and '1' for full-term baby (Fig. 47.5). Size and lip to tip length of ET tube are selected according to gestation and birth weight of the baby (Table 47.2)

ET tube connector should be placed at a distance of 4–5 cm proximal to lips (total length 13–15 cm). CO_2 detector if available may be used to verify the placement of ET tube.

Table 47.2 Size and length of ET tube

Birth weight (g)	Gestation (weeks)	Inner diameter of ET tube (mm)	Lip to tip length (cm)
<1000	28	2.5	6
1000–2000	28–34	3.0	7
2000–3000	34–38	3.5	8–9
>3000	>38	3.5–4	9–10

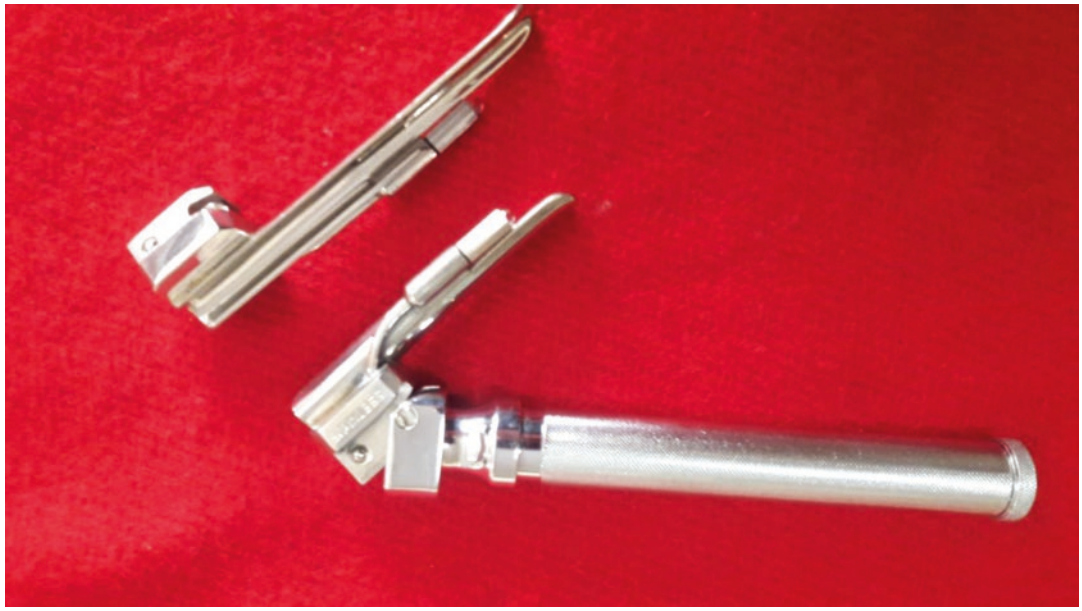


Fig. 47.5 Laryngoscope with neonatal straight blades

47.2.4 Drug Administration (Medication)

Despite good ventilation and augmented cardiac output with chest compressions, a very small number of neonates (<0.1%) will still have HR <60 bpm. It may be due to myocardial dysfunction due to severe asphyxia or shock due to blood loss. If HR and SPo2 are normal on PPV but poor or no respiratory drive, it may be due to hypoxic ischaemic damage to the brain, severe acidosis, structural anomaly of the brain, neuromuscular disorder or drugs received by mother such as narcotics, magnesium sulphate and general anaesthesia.

Types of Medication: Intravenous access should be established by canulating umbilical vein.

1. **Adrenaline**: It is indicated when HR remains <60 bpm after effective ventilation (with ET tube) along with coordinated chest compressions over 45–60 s. Its ionotropic and chronotropic action will increase the HR and myocardial contractility.

   Dose: 0.1–0.3 mL/kg of 1:10,000 solution (prepared by 10 times dilution of 1:1000 adrenaline), i.e. 0.01–0.03 mg/kg, IV, rapidly followed by 0.5–1.0 mL flush with normal saline to ensure that drug has reached the circulation. Adrenaline can also be given through ET tube, while IV access is being obtained, as 0.5–1.0 mL/kg 1:10,000 (0.05–0.01 mg/kg), followed by several PP breaths to distribute the drug throughout the lungs for rapid distribution and absorption.

   Response: While continuing PPV and chest compressions, check HR about 1 min after adrenaline administration.

   (a) **HR >60 bpm**: Discontinue chest compressions; continue PPV and monitoring.
   (b) **HR <60 bpm**: Continue coordinated PPV with chest compressions, and repeat adrenaline every 3–5 min. Assess for hypovolemic shock as evidenced by pallor, delayed capillary refill time (CRT) and/or weak pulse.
2. **Volume expanders**: Indication—hypovolemic shock.

10 mL/kg of normal saline or Ringer's lactate or O-/Rh-negative blood specially if there is setting for blood loss, given IV over 5–10 min.

3. **Naloxone**: Indication: Poor or no respiratory drive with normal HR and SPo2 on PPV with history of maternal narcotic administration within 4 h. Since safety and long-term effects are not yet well established, naloxone is generally not necessary as long as baby can be adequately ventilated till the effect of narcotic weans off [1]. Dose: 0.1 mg/kg IV. Continue PPV till spontaneous breathing is established.
4. **Sodium bicarbonate**: Its use in neonatal resuscitation is controversial because it is hypertonic, irritant and may cause intraventricular haemorrhage (IVH) and worsen intracellular acidosis. In case of severe metabolic acidosis, it may be administered as 2 meqv/kg at 1 meqv/min slowly IV while maintaining adequate ventilation.

Post-resuscitation care: Neonates who have required resuscitation beyond initial steps are at risk of developing problems associated with perinatal compromise even after successful resuscitation. The likelihood of developing post-resuscitation complications is directly proportional to the length and extent of resuscitation required. So most of these resuscitated babies with HR >100 bpm and normal expected SPo2 require only routine care, many will require frequent monitoring of vitals and awareness of potential complications, and few will need continued respiratory support and laboratory studies like haematocrit, blood sugar, electrolytes and blood gas analysis in SNCU/NICU.

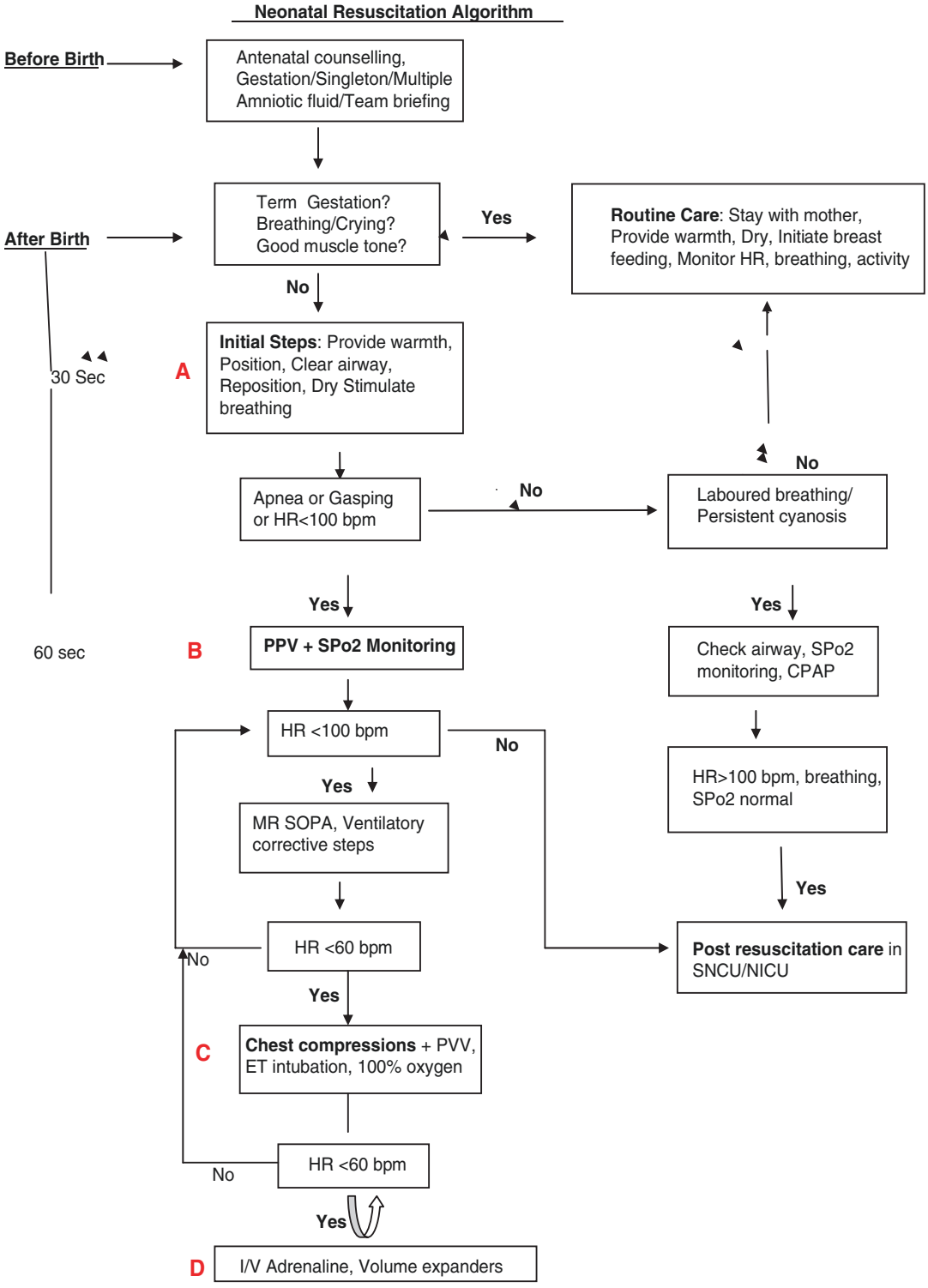
Resuscitation failure: If baby shows poor or no response to resuscitation, suspect conditions like pneumothorax, hydrothorax, PPHN, congenital pneumonia, hypoplastic lung, diaphragmatic hernia and congenital cyanotic heart disease, and take appropriate steps accordingly.

Decision to Abandon Resuscitation [1]

1. Confirm absent heartbeat for at least 10 min: Current data indicate that after 10 min of asystole, survival is either unlikely, or it is with severe disability.
2. Severe bradycardia without improvement in general condition despite adequate resuscitation; it may be discontinued on a case-to-case basis and parental consent based on their threshold of acceptable risk of morbidity.
3. **Do not resuscitate**: There are situations where gestation, birth weight and or congenital malformations are associated with almost certain early death or survival with unacceptably high morbidity. Here again after parental consent, it is appropriate not to initiate resuscitation in the following situations:
   - Confirmed gestation <23 weeks or birth weight <400 g
   - Anencephaly
   - Confirmed lethal genetic disorder or malformation like trisomy 13 and 18

47.3 Conclusion

Three to five percent of neonates develop birth asphyxia, and FGM is of utmost importance for its prevention, timely assessment and effective management. Most of the times, it can be predicted in high-risk settings, but at times it may occur as a surprise. Hence a person skilled in neonatal resuscitation must be available at all delivery points. Anticipation, adequate preparedness in terms of personnel and equipment, timely recognition and quick corrective measures are critical for success of neonatal resuscitation. ABCD of resuscitation consists of maintaining open **a**irway, **b**reathing by effective lung ventilation, **c**irculation by chest compressions and rarely **d**rug administration to augment circulation.


Neonatal Resuscitation Algorithm
Before Birth
Antenatal counselling, Gestation/Singleton/Multiple Amniotic fluid/Team briefing
After Birth
Term Gestation? Breathing/Crying? Good muscle tone?
Yes
Routine Care: Stay with mother, Provide warmth, Dry, Initiate breast feeding, Monitor HR, breathing, activity
No
30 Sec
A
Initial Steps: Provide warmth, Position, Clear airway, Reposition, Dry Stimulate breathing
Apnea or Gasping or HR<100 bpm
No
Laboured breathing/ Persistent cyanosis
No
Yes
Yes
60 sec
B
PPV + SPo2 Monitoring
Check airway, SPo2 monitoring, CPAP
HR <100 bpm
No
HR>100 bpm, breathing, SPo2 normal
Yes
MR SOPA, Ventilatory corrective steps
Yes
HR <60 bpm
No
Post resuscitation care in SNCU/NICU
No
Yes
C
Chest compressions + PVV, ET intubation, 100% oxygen
HR <60 bpm
No
Yes
D
I/V Adrenaline, Volume expanders

References

1. Kattwinkel J. Text book of neonatal resuscitation. 6th ed. New Delhi: Jaypee Brothers Medical Publishers(P) Ltd; 2011.
2. Gupta P. Text book of pediatrics. 1st ed. New Delhi: CBS Publishers and Distributors Pvt Ltd; 2013.
3. Carlo WA. Delivery room emergencies. In: Kliegman RM, Stanton BF, St. Geme III JW, Schor NF, editors. Nelson TB of pediatrics(South Asian edition). New Delhi: Elsevier; 2016. p. 844–8.
4. Gaur A. Neonatal resuscitation. In: Gupte S, editor. Recent advances in pediatrics, perspectives in neonatology(special volume 25), vol. 2014. New Delhi: Jaypee Brothers. p. 61–7.

Neonatal Injuries

48

Piyush Gautam and Nivedita Sharma

Definition: Birth injury may be defined as an impairment of the infant's body or structure due to adverse influences, which occurred at birth. Injury may occur during the antenatal or intrapartum period or even during resuscitation and is often unavoidable [1].

Birth injuries are commonly seen by physicians looking after newborn infants. They may range from the relatively common soft tissue injuries that most often require careful observation to the more severe injuries like intracranial bleeding that may be life-threatening and need immediate intervention. The risk of birth trauma does not decrease after a cesarean section, especially after failed forceps or vacuum extraction [2].

48.1 Risk Factors

In cases where labor is complicated by fetal size, prematurity, or malpresentation, normal intrapartum compressions, contortions, and forces can lead to injury. The following factors may increase the risk of birth injury:

Maternal factors:

1. Primiparity
2. Maternal short stature
3. Maternal pelvic anomalies

Fetal factors:

1. Macrosomia
2. Very low birth weight
3. Extreme prematurity
4. Fetal anomalies
5. Malpresentation (breech, face, shoulder dystocia)

Related to delivery:

1. Prolonged labor
2. Unusually rapid labor
3. Use of forceps or vacuum extraction

48.2 Evaluation

A newborn at risk for birth injury should be thoroughly examined from head to toe, including a detailed neurological examination, as injury may be occult. Things to be looked for are symmetry of structure and function, cranial nerves, range of motion of individual joints, and integrity of the scalp and skin.

Table 48.1 enlists common injuries encountered in a newborn.

P. Gautam (✉) · N. Sharma
Department of Pediatrics, Dr RPGMC Tanda, Kangra, HP, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_48

Table 48.1 Common injuries encountered in a newborn

Type of injury	Example
Soft tissue injuries	Abrasions, bruises, lacerations, subcutaneous fat necrosis
Extracranial bleeding	Cephalhematoma, subgaleal bleed
Intracranial bleeding	Subarachnoid, epidural, subdural, cerebral, cerebellar hemorrhage
Nerve injuries	Facial nerve, brachial plexus, phrenic nerve, recurrent laryngeal nerve
Spinal cord injuries	Epidural hemorrhage of the cervical cord
Fractures/ dislocations	Clavicle, humerus, femur, skull
Torticollis	Due to bleeding in the sternocleidomastoid muscle
Eye injuries	Subconjunctival, retinal, vitreous hemorrhage, orbital fracture
Solid organ injury	Liver, spleen, adrenal gland

48.3 Soft Tissue Injury

This is the most common form of traumatic birth injury and includes petechiae, ecchymosis, and bruising. Most of these injuries result from difficult extractions from the breech position, shoulder dystocia, and use of the vacuum or forceps. Soft tissue injuries, though usually minor, may increase the risk of significant hyperbilirubinemia.

Petechiae are usually present over the head, neck, and upper chest. These are present after birth, do not increase, and are not associated with bleeding from other sites. A platelet count should be obtained if there is bleeding from other sites or the petechiae progress. Breech delivery can lead to severe vaginal or scrotal edema and bruising. This usually resolves spontaneously, though drainage of a testicular hematoma may rarely be required [2].

An electrode placed on the scalp for fetal heart monitoring may cause abrasions or lacerations, which may get secondarily infected. If malpositioned, it may cause facial or ocular trauma.

Subcutaneous fat necrosis is an area of induration due to local ischemia from trauma. It is usually seen late during the first week, has red or purple discoloration, and resolves spontaneously by 6–8 weeks [2].

48.4 Sternocleidomastoid (SCM) Injury

Congenital muscular torticollis is seen in approximately 0.4% of births [4]. One of the mechanisms proposed to cause this condition is manual stretching of the neck causing rupture of the muscle with formation of a hematoma and subsequent fibrosis leading to torticollis. The infant usually presents at 2–3 weeks of age with head tilt to the side of the lesion, with a 1–2 cm palpable mass in SCM region.

Management involves physiotherapy, with stretching exercises done many times in a day, that results in 90% recovery within 3–4 months [4].

48.5 Extracranial Injuries

48.5.1 Caput Succedaneum

A caput succedaneum is a subcutaneous, extraperiosteal fluid collection that extends over the presenting portion of the scalp. It has poorly defined margins and extends across suture lines. It resolves spontaneously over a few days and no treatment is required.

48.5.2 Cephalhematoma

Cephalhematoma results from collection of blood in the subperiosteal space, due to rupture of superficial veins between the skull and periosteum (Fig. 48.1). It may occur in up to 2.5% of live births, the incidence being higher in forceps and vacuum deliveries [2]. It is usually present over the parietal or occipital bone and does not cross suture lines. Hemorrhage is rarely serious enough to necessitate blood transfusion though it may result in significant hyperbilirubinemia.

Fig. 48.1 Neonate with a large cephalhematoma in the left parietal region

Linear skull fractures may be associated in up to 5% cases, but they usually do not require any treatment [2].

Diagnosis is clinical, but if there are neurological signs and symptoms, a computed tomography (CT) scan of the head must be done to rule out intracranial involvement.

Management involves observation only, as most lesions resolve spontaneously over a few weeks. Attempts at aspiration and incision may introduce infection and are contraindicated.

48.5.3 Subgaleal Hemorrhage

Hemorrhage under the aponeurosis of the scalp results in a subgaleal bleed. The overall incidence is 1 in 2000 births but may be as high as 1 in 200, most commonly seen in vacuum or forceps deliveries, especially with multiple attempts [2]. The injury results from rupture of emissary veins between the scalp and intracranial venous sinuses due to traction on the scalp. It is usually seen within the first few hours of birth as a fluctuant swelling that crosses suture lines.

Diagnosis is purely clinical. Blood loss may be significant and may result in shock. The newborn must therefore be monitored closely for signs of hypovolemia like tachycardia, feeble pulses, and prolonged capillary refill. Laboratory investigations include serial hematocrit monitoring and bilirubin levels. A coagulation profile may be considered to rule out a bleeding disorder [3].

Treatment is largely supportive. Significant bleeding may require fluid replacement or even blood transfusion.

48.6 Cranial Injuries

Skull fractures can be linear, usually involving the parietal bone, or depressed, involving the parietal or frontal bones. Depressed fractures often follow the use of forceps (Fig. 48.2). Occipital bone fractures are seen with breech deliveries. Due to the resilient nature of the bone, skull fractures in neonates are usually depressed that results in a "ping-pong" deformity without discontinuity. Treatment is usually not required as majority of infants are asymptomatic, unless there is associated intracranial bleed. A CT scan of the head must be done and a neurosurgical consultation taken if intracranial injury is suspected [2].

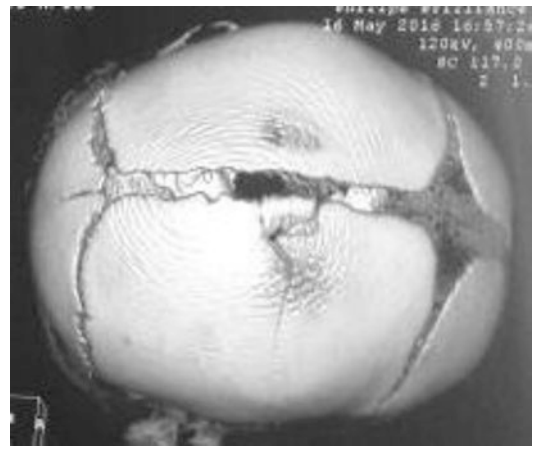


Fig. 48.2 CT scan showing linear undisplaced fracture of the right parietal bone extending into the sagittal suture

48.7 Intracranial Injury

In term infants, symptomatic intracranial hemorrhage occurs in approximately 5.1–5.9 per 10,000 live births [5, 6]. Risk factors are macrosomia, forceps delivery, vacuum extraction, and prolonged second stage of labor. Although the neonate may be asymptomatic after birth, majority will present within 48 h of birth, most commonly with apnea and seizures [7].

Intracranial bleed can be subdural, epidural, subarachnoid, or intraparenchymal.

48.7.1 Subdural Hemorrhage

Subdural hemorrhage is the most common intracranial bleed resulting from birth trauma. The incidence ranges from 2.9 per 10,000 live births in spontaneous vaginal deliveries to 8–10 per 10,000 live births in vacuum and forceps deliveries [5]. Risk factors include large fetal head, rigid pelvis, breech, face, presentation, prolonged labor, difficult instrumental delivery, or rarely a bleeding diathesis. Subdural hemorrhage results from rupture of the draining veins and venous sinuses in the subdural space. The ruptured vessels may range from small superficial veins to large venous sinuses when the symptoms are severe. Symptoms include altered level of consciousness, irritability, seizures, bulging fontanelle, apnea, and focal neurological signs.

A CT scan of the head is the diagnostic procedure of choice. An ultrasound done at the bedside may pick up the bleed in some cases. MRI may sometimes be required to more clearly delineate posterior fossa bleeds. Since subdural hematomas may be associated with coagulation disorders, a coagulation profile must be obtained [8]. Most infants can be managed with supportive care and control of seizures, if any. Surgical evacuation may be needed in cases with a large bleed and signs of brainstem compression or hydrocephalus [8, 9].

48.7.2 Epidural Hemorrhage

Injury to the middle meningeal artery results in epidural hemorrhage, though it is rarely seen. A cephalhematoma or a skull fracture is frequently found in association [10]. The newborn may present with diffuse neurologic symptoms, seizures, and a bulging fontanelle.

A CT scan of the head will show a high-density lentiform lesion in the temporoparietal region. An epidural hematoma will require surgical drainage in most cases.

48.7.3 Subarachnoid Hemorrhage

Symptomatic subarachnoid hemorrhage occurs in 1.3 per 10,000 live births in spontaneous vaginal deliveries to 2 or 3 per 10,000 live births in vacuum and forceps deliveries [11]. The incidence is higher in preterm and asphyxiated newborns. It results from rupture of small leptomeningeal vessels or the bridging veins in the subarachnoid space. Symptoms include irritability, seizures, or depressed sensorium.

CT scan of the head is the diagnostic procedure of choice. Ultrasonography is relatively insensitive.

Management is largely supportive. A post-hemorrhagic hydrocephalus may develop in case of a large bleed; therefore, the infant must be followed up with serial cranial ultrasound and measurement of head size [12] (Fig. 48.3).

48.8 Neurological Injuries

48.8.1 Facial Nerve Palsy

Facial nerve palsy is the most common nerve injury in newborns, occurring in approximately 1% of births [2]. Compression of the nerve by forceps or prolonged pressure against the maternal sacral promontory is the usual cause. The newborn usually presents with a

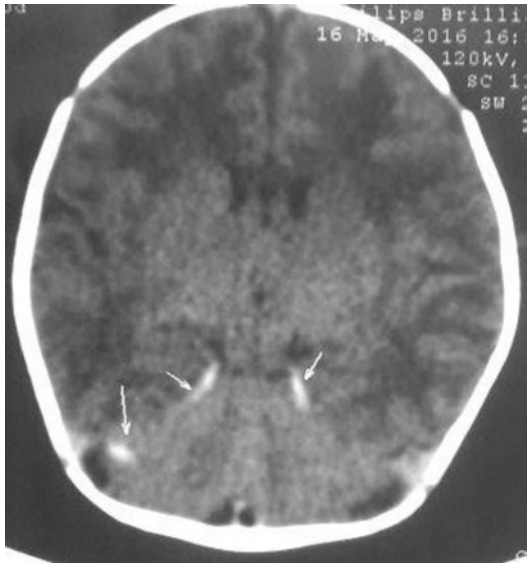


Fig. 48.3 Parenchymal bleed in bilateral cerebellar hemispheres with bilateral tentorial subdural hemorrhage

lower motor neuron neuron facial palsy with flattened nasolabial fold, deviation of the angle of the mouth toward the normal side, and inability to wrinkle the forehead and completely close the affected eye.

Differential diagnosis includes congenital nerve palsy, which commonly has other features of craniofacial dysmorphism or multisystem findings, e.g., Moebius, DiGeorge, and Goldenhar syndromes and trisomy 13 and trisomy 18 syndromes [13]. In congenital hypoplasia of the depressor anguli oris muscle, there is a localized movement abnormality of the corner of the mouth.

Prognosis is excellent with complete recovery occurring by 3 weeks. Management includes prevention of corneal injury by using artificial tears and eye patch [1].

48.8.2 Spinal Cord Injury

Spinal cord injuries at birth are rare, but extremely serious. Risk factors include breech delivery, shoulder dystocia, and hyperextended neck in vaginal delivery. Clinical presentation depends on the site and severity of injury, ranging from subtle neurological signs to paraparesis and respiratory failure. Diagnosis can be confirmed by MRI of the spine. Management is supportive and involves stabilization of the head, neck, and spine. Early neurosurgery consultation must be taken. Prognosis is generally poor, especially if the infant remains ventilator dependent for more than 24 h [14].

48.8.3 Phrenic Nerve Injury

Phrenic nerve injury leads to paralysis of the ipsilateral diaphragm (C3–C5). The nerve is injured where it crosses the brachial plexus, resulting in injury to the brachial plexus in approximately 75% of cases [3].

The injury is usually unilateral and occurs due to excessive traction on the neck and arm, as may occur in breech and difficult forceps deliveries. The infant presents with respiratory distress, with decreased movement of the hemithorax and reduced breath sounds on the affected side. Chest radiographs reveals elevation of the affected diaphragm. The diagnosis can be confirmed by ultrasonography or fluoroscopy that shows paradoxical movement of the diaphragm during inspiration [1].

Management involves respiratory support with continuous positive airway pressure or mechanical ventilation. Recovery occurs in 1–3 months in most cases. Refractory cases may require surgical plication of the diaphragm [3].

48.8.4 Brachial Plexus Injury

Brachial plexus injury occurs in 0.1–0.2% of births, risk factors being shoulder dystocia, macrosomia, breech presentation, and instrumental deliveries [3].

Brachia plexus injury can present as Erb's palsy, Klumpke's palsy, and injury to the entire plexus.

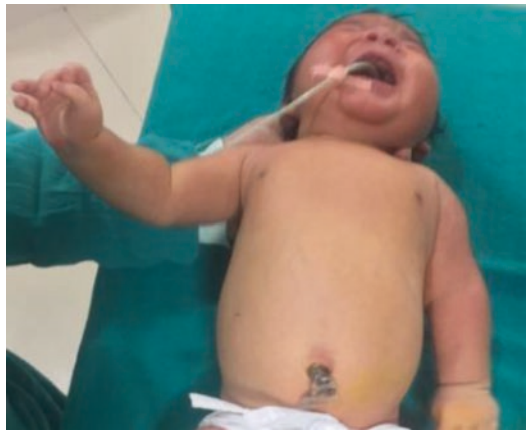


Fig. 48.4 Asymmetric Moro's reflex with porter's tip hand in a macrosomic infant of a diabetic mother with left-sided Erb's palsy

48.8.4.1 Erb's Palsy

Erb's palsy is the most common type, occurring in approximately 90% of cases. It involves injury to the upper trunk of the brachial plexus (C5, C6, and C7). The involved arm is held in the "waiter's tip" position, with adduction and internal rotation of the shoulder, extension of the elbow, pronation of the forearm, and flexion of the wrist and fingers (Fig. 48.4). The Moro reflex is asymmetric and biceps reflex is absent. It is associated with diaphragmatic paralysis in 5% cases.

48.8.4.2 Klumpke's palsy

Klumpke's palsy is rare and occurs in less than 1% cases. Involvement of C8 and T1 nerves results in weakness of the intrinsic muscles of the hand and long flexors of the wrist and fingers. The grasp reflex is absent but the biceps reflex is present.

In up to 10% cases, the entire plexus is involved, resulting in a flaccid extremity with absent reflexes.

A good physical examination is all that is required to make the diagnosis. A radiograph of the shoulder and upper arm must however be done as fractures of the clavicle and humerus may be associated. Absence of neurological signs often rules out brain injury.

Spontaneous recovery occurs in approximately 90% of brachial plexus injuries. The prognosis is poorer in total plexus and lower plexus injuries. Treatment is mainly conservative and involves passive range of motion exercises to prevent contractures. Physical therapy must be started after 7–10 days when post-injury neuritis has resolved [3].

48.8.5 Recurrent Laryngeal Nerve Injury

During breech or forceps delivery, excessive traction on the fetal head can lead to injury of the recurrent laryngeal nerve. The injury is usually unilateral and the infant is often asymptomatic at rest, having hoarseness and inspiratory stridor on crying. Bilateral involvement results in stridor, severe respiratory distress, and cyanosis [1].

Differential Diagnosis: If a newborn presents with the above mentioned symptoms in absence of a history of trauma, one must consider congenital laryngeal and central nervous system malformations and mediastinal masses.

Diagnosis can be made by using direct or fiber-optic laryngoscopy.

Spontaneous recovery usually occurs in unilateral injury by 6 weeks of age. Bilateral paralysis has a variable prognosis and may occasionally require tracheostomy [1].

48.8.6 Facial Injuries

48.8.6.1 Nasal Injuries

Nasal septal dislocation is the most common nasal injury, having an incidence of 0.6–0.9%. The infant presents with features of upper airway obstruction. On examination, the nose is deviated to one side, with asymmetric nares and flattening of the side of the dislocation. On applying pressure on the tip of the nose, there is collapse of the nostrils and the deviated septum becomes more apparent. In the misshapen nose, however, compression does not cause nasal deviation. Since delay in treatment can lead to long-term cosmetic deformity, urgent ENT consultation should be sought. The dislocation is relieved by rhinoscopy and manual reduction [3].

48.8.6.2 Ocular Injuries

Minor ocular injuries, including subconjunctival and retinal hemorrhages, are commonly seen after vaginal delivery. Malpositioned forceps can lead to ocular and periorbital injury including orbital fracture, vitreous hemorrhage, corneal injury, and lacrimal duct injury. Retinal and subconjunctival hemorrhages resolve spontaneously within a few days. In case of significant ocular injury, prompt ophthalmology consultation must be taken [1].

48.8.6.3 Ear Injuries

Ear injury may occur during forceps application resulting in abrasions, lacerations, and hematoma formation. Complications include cauliflower ear, perichondritis, and ossicular disarticulation. If cartilage and temporal bone are involved, ENT consultation must be taken. A hematoma of the pinna must be drained to prevent development of a cauliflower ear [1].

48.9 Bone Injuries

Clavicular fracture is the most common bony injury during delivery, with an incidence of 0.5–1.5% of live births.

Fractures most commonly occur during normal, spontaneous vaginal delivery, but the incidence increases with vigorous manipulations, as may occur in case of shoulder dystocia and breech extractions.

The humerus is the most common long bone to get fractured. The neonate may remain asymptomatic if the fracture is nondisplaced. The first sign may be a callus at 7–10 days of age. A complete fracture may present with a crepitus, a palpable callus, or an obvious deformity in case of humeral fracture. There may be pseudoparalysis of the affected arm. Fractures of the humerus and clavicle may be associated with brachial plexus and radial nerve injuries. Fracture of the femur is most commonly seen after a breech delivery. Examination reveals an obvious deformity of the thigh, decreased limb movement, and tenderness on palpation. An X-ray will confirm the diagnosis [1].

48.9.1 Treatment

Clavicular fracture management involves alleviation of pain with analgesics. Movement can be decreased by pinning the infant's sleeve to the shirt until callus is formed. Humeral fracture needs splinting for 2 weeks. Displaced fracture will need closed reduction and casting. Femoral fracture is managed with traction and suspension of both legs with a spica cast, even if there is unilateral involvement [1].

48.10 Intra-abdominal Injury

Intra-abdominal injuries are uncommon and include rupture or subcapsular hemorrhage into the liver, spleen, or adrenal gland. Liver is the most commonly injured solid organ. Risk factors are macrosomia, breech delivery, asphyxia, and hepatosplenomegaly. There will be signs of hypovolemia including lethargy, pallor, tachypnea, and tachycardia that may progress to shock. An abdominal ultrasound will confirm the diagnosis.

Management includes restoration of blood volume using crystalloids, blood transfusion, and correction of coagulopathy. Adrenal insufficiency may need steroid therapy. Surgical consultation must be taken, as a laparotomy may be required.

48.11 Prevention

The occurrence of birth injuries is often unpredictable and unavoidable. But anticipation of injury in certain high-risk situations can either prevent the injury or help in early diagnosis. Some measures that can help in injury prevention are the following:

During antenatal period:

1. Screen out women with high-risk factors, e.g., those with cephalopelvic disproportion, macrosomia, and malpresentation.

During intranatal period:

1. Perform continuous fetal monitoring.
2. Avoid difficult forceps.

3. Judicious selection of suitable candidates for instrumental delivery.
4. Breech delivery by skilled personnel.

48.12 Summary

With improvement in obstetric care, perinatal morbidity and mortality due to birth injury has decreased over the past 25 years [2]. But clinicians caring for newborns will continue to encounter birth-related injuries. Furthermore, associated clinical problems after birth may mask the signs and symptoms of many injuries; as a result, one may miss the injury. It is therefore important for the attending clinician to anticipate and recognize such injuries so that the morbidity and mortality resulting from birth trauma is reduced.

References

1. Cloherty JP, Eichenwald EC, Stark AR. Manual of neonatal care. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins. 2010.
2. Rosenberg AA. Traumatic birth injury. NeoReviews. 2003;4(10):e271.
3. Uhing MR. Management of birth injuries. Pediatr Clin North Am. 2004;51:1169–86.
4. Jaber MR, Goldsmith AJ. Sternocleidomastoid tumor of infancy: two cases of an interesting entity. Int J Pediatr Otorhinolaryngol. 1999;47(3):269–74.
5. Towner D, Castro MA, Eby-Wilkens E, Gilbert WM. Effect of mode of delivery in nulliparous women on neonatal intracranial injury. N Engl J Med. 1999;341(23):1709–14.
6. Sachs BP, Acker D, Tuomala R, Brown E. The incidence of symptomatic intracranial hemorrhage in term appropriate for gestation age infants. Clin Pediatr (Phila). 1987;26(7):355–8.
7. Pollina J, Dias MS, Li V, Kachurek D, Arbesman M. Cranial birth injuries in term newborn infants. Pediatr Neurosurg. 2001;35(3):113–9.
8. Perrin RG, Rutka JT, Drake JM, Meltzer H, Hellman J, Jay V, et al. Management and outcomes of posterior fossa subdural hematomas in neonates. Neurosurgery. 1997;40(6):1190–9. [discussion:1199–200]
9. Huang CC, Shen EY. Tentorial subdural hemorrhage in term newborns: ultrasonographic diagnosis and clinical correlates. Pediatr Neurol. 1991;7(3):171–7.
10. Negishi H, Lee Y, Itoh K, Suzuki J, Nishino M, Takada S, et al. Nonsurgical management of epidural hematoma in neonates. Pediatr Neurol. 1989;5(4):253–6.
11. Abroms IF, Rosen BA. Neurologic trauma in newborn infants. Semin Neurol. 1993;13(1):100–5.
12. Harpold TL, McComb JG, Levy ML. Neonatal neurosurgical trauma. Neurosurg Clin N Am. 1998;9(1):141–54.
13. Shapiro NL, Cunningham MJ, Parikh SR, Eavey RD, Cheney ML. Congenital unilateral facial paralysis. Pediatrics. 1996;97(2):261–4.
14. MacKinnon JA, Perlman M, Kirpalani H, Rehan V, Sauve R, Kovacs L. Spinal cord injury at birth: diagnostic and prognostic data in twenty-two patients. J Pediatr. 1993;122(3):431–7.

Part VI

Miscellaneous

Postexposure Prophylaxis in HIV

49

Pranav Sood and Shivani Sood

ARV drugs are in use since the last three decades for postexposure prophylaxis following occupational exposure to HIV, but more recently, the same has been extended to accidental exposure like unprotected intercourse, sexual assault, needle prick injuries, IV drug users, etc. Use of ART for postexposure prophylaxis is supported by animal studies [1], a case control study [2], and systematic reviews which have demonstrated reduced risk of acquiring chronic infection and cost-effectiveness in high-risk groups [3, 4]. That ARV drugs are effective in postexposure prophylaxis is further supported by their role in preexposure prophylaxis and in preventing mother-to-child transmission.

Postexposure prophylaxis cannot be considered 100% effective because its effectiveness depends upon a number of factors like timing of initiation, adherence to treatment, completion of course, drug resistance, viral load, etc. Postexposure prophylaxis should form a part of wider strategy to prevent acquiring HIV infection, HBV, HCV, and other blood-borne viruses [5, 6].

Exposures that may warrant postexposure prophylaxis include:

1. Parenteral or mucous membrane exposure (sexual exposure and splashes to the eye, nose, or oral cavity)
2. Body fluids like blood, blood-stained saliva, CSF, breast milk, genital secretions, and pleural, pericardial, synovial, rectal, and peritoneal fluids.
3. With high background prevalence of HIV infection, all exposures can be considered.

Exposures that do not require postexposure prophylaxis include:

1. When the source is known to be HIV negative, etc.
2. When the exposed individual is known to be HIV positive
3. Exposure to body fluids that do not pose a significant risk like tears, sweat, urine, non-blood-stained saliva, etc.

Percutaneous needle stick injury has a risk of 23 in 10,000 exposures to an infected source [7].

Steps to be taken in case of accidental exposure to HIV:

1. HIV testing of exposed person and source if possible.
2. First aid in case of a broken skin or wound.
3. Counseling if postexposure prophylaxis is to be prescribed:
   (a) Risks and benefits
   (b) Side effects
   (c) Risk of HIV
   (d) Consent
4. Postexposure prophylaxis should be started as early as possible preferably within 72 h of exposure.
5. 28-day prescription.

P. Sood (✉) · S. Sood
Kamala Nehru Hospital, Shimla, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_49

6. Assessment of underlying comorbidities and drug-drug interactions.
7. Retesting at 3 months.
8. Preventive measures.

According to ART guidance, postexposure prophylaxis can be dispensed by trained nurses, midwives, and nonphysicians [3].

Recent WHO guidelines recommend triple combination therapy:

1. Simplified prescribing.
2. Availability of
   (a) Better tolerated
   (b) Less toxic drugs
   (c) Difficulty in assessing drug interactions
3. Completion rates are similar compared to two drug regimes.

Doses of ARV drugs for HIV postexposure prophylaxis for adults and adolescents

Tenofovir (TDF)	300 mg once daily
Lamivudine (3TC)	150 mg twice daily or 300 mg once daily
Emtricitabine (FTC)	200 mg once daily
Lopinavir/ritonavir	400 mg/100 mg twice daily or 800mg/(LPV/r)
	200 mg once daily
Atazanavir/ritonavir (ATV/r)	300 mg + 100 mg once daily
Raltegravir (RAL)	400 mg twice daily
Darunavir + ritonavir (DRV/r)	800 mg + 100 mg once daily or 600 mg + 100 mg twice daily
Efavirenz (EFV)	600 mg once daily

Tenofovir and lamivudine are the combination of choice for HIV postexposure prophylaxis (strong recommendation, low-quality evidence).

Lopinavir/ritonavir or atazanavir/ritonavir is the preferred third drug (conditional recommendation, very low-quality evidence). These are widely available in low- and middle-income group countries.

RAL, DRV/r, and EFV can be considered if available, with limited availability owing to higher cost.

Indirect comparisons have been made between zidovudine + lamivudine [8–19] and tenofovir + lamivudine [20–22] in several studies. Completion rates were 78% in the latter group compared to 59% in the former. Discontinuation rate was higher in the former group in comparison to the latter (3.2 vs. 0.3%) due to some adverse event.

Newer drugs, dolutegravir (high potency and tolerability), rilpivirine (high tolerability), and elvitegravir (high tolerability and convenient coformulation), have promise, but there are no current recommendations for their use.

Efavirenz is also recommended as an alternative third-line drug for postexposure prophylaxis. It is well tolerated but has limited acceptability in HIV-negative individuals (nervous system and mental events).

Doses of ARV drugs for HIV postexposure prophylaxis for adults and adolescents

	LPV/r	ATV/r	RAL	DRV/r	EFV	NVP
Discontinuation rate in postexposure prophylaxis	Low (use for preventing mother-to-child transmission of HIV)	No data				Low (use for preventing mother-to-child transmission of HIV)
Daily dosing	Twice daily	One tablet once daily	Twice daily	Once or twice daily	Once daily	Twice daily
Availability of heat-stable age-appropriate formulation	Yes	No	Yes	No	Yes	Yes
Accessibility in country (registration status)	High	Low	Low	Low	High (>3 years)	High (all ages)
Accessibility by health providers	High	High	High	High	High	High
Availability of WHO prequalified generic formulation	Yes	Yes	No	No	Yes	Yes
Age indication	>14 years	>3 months	>2 weeks	>3 years	>3 months	<2 years

Characteristics of third drug options for HIV postexposure prophylaxis for adults and adolescents

	LPV/r	ATV/r	RAL	DRV/r	EFV
Discontinuation rate in postexposure prophylaxis	7%	21%	2%	6%	No data
Daily dosing	Two tablets twice daily	One tablet once daily	One tablet twice daily	One tablet once or twice daily	One tablet once daily
Availability of heat-stable formulation	Yes	Yes	Yes	No	Yes
Accessibility in country (registration status)	High	Low	Low	Low	High
Accessibility by health providers	High	High	High	High	Low
Availability of WHO prequalified generic formulation	Yes	No	No	No	Yes

Side Effects

Tenofovir is associated with low rate of renal toxicity. It should be avoided in the following conditions:

(a) If GFR <50 mL/min
(b) Long-standing diabetes
(c) Uncontrolled hypertension
(d) Renal failure

Tenofovir- and lamivudine-based postexposure chemotherapy is associated with potential risk of hepatic flares especially in people infected with HBV [23, 24].

(a) HBV testing is not a precondition for starting postexposure prophylaxis.
(b) Should be done in centers where testing facilities are available.
(c) People with established infections should be monitored for hepatic flares after discontinuation.

Nevirapine is not recommended for use in adolescents and adults because of life-threatening complications and interactions with other drugs [25].

Postexposure Prophylaxis ARV Regimens: Children (≤10 Years Old)

Zidovudine + lamivudine is the treatment of choice.

Abacavir + lamivudine is alternative.

Tenofovir + lamivudine is another alternative (strong recommendation, low-quality evidence).

Lopinavir/ritonavir is the preferred third drug (conditional recommendation, very low-quality evidence).

Alternative regime includes ATV/r, R AL, DRV, EFV, and NVP.

Zidovudine

(a) Anemia, transient and mild
(b) Seen in infants receiving postnatal prophylaxis
(c) In HIV-positive children receiving zidovudine

Abacavir

(a) Hypersensitivity reactions
(b) Particularly in Caucasian and Asian children

Lopinavir/ritonavir oral liquids should not be used for preterm infants or infants <2 weeks.

Nevirapine is used in these infants.

Toxicity profile of nevirapine remains unclear beyond 2 years, and there are concerns of serious adverse effects which limit its use beyond 2 years.

References

1. Corbett EL, et al. Uptake of workplace HIV counseling and testing: a cluster-randomized trial in Zimbabwe. PLoS Med. 2006;e238:3.
2. Grabbe KL, et al. Increasing access to HIV counseling and testing through mobile services in Kenya: strategies, utilization, and cost-effectiveness. J Acquir Immune Defic Syndr. 2010;54:317–23.

3. Granich R, et al. Achieving universal access for human immunodeficiency virus and tuberculosis: potential prevention impact of an integrated multi-disease prevention campaign in Kenya. AIDS Res Treatment. 2012;2012:412643.
4. Lugada E, et al. Comparison of home and clinic-based HIV testing among household members of persons taking antiretroviral therapy in Uganda: results from a randomized trial. J Acquir Immune Defic Syndr. 2010;55:245–52.
5. Menzies N, et al. The costs and effectiveness of four HIV counseling and testing strategies in Uganda. AIDS. 2009;23:395–401.
6. van Schaik N, et al. Earlier HIV diagnosis – are mobile services the answer? S Afr Med J. 2010;100:671–4.
7. Chirawu P, et al. Acceptability and challenges of implementing voluntary counseling and testing (VCT) in rural Zimbabwe: evidence from the Regai Dzive Shiri Project. AIDS Care. 2010;22:81–8.
8. Sweat M, et al. Community-based intervention to increase HIV testing and case detection in people aged 16-32 years in Tanzania, Zimbabwe, and Thailand (NIMH Project Accept, HPTN 043): a randomized study. Lancet Infect Dis. 2011;11:525–32.
9. Outlaw AY, et al. Using motivational interviewing in HIV field outreach with young African American men who have sex with men: a randomized clinical trial. Am J Public Health. 2010;100(Suppl. 1):S146–51.
10. Prevention and treatment of HIV and other sexually transmitted infections among men who have sex with men and transgender people: recommendations for a public health approach. Geneva: World Health Organization; 2011. http://whqlibdoc.who.int/publications/2011/9789241501750_eng.pdf. Accessed 15 May 2013.
11. Delivering HIV test results and messages for re-testing and counseling in adults. Geneva: World Health Organization; 2010. http://whqlibdoc.who.int/publications/2010/9789241599115_eng.pdf. Accessed 15 May 2013.
12. Planning, implementing and monitoring home-based HIV testing. Geneva: World Health Organization; 2012. http://apps.who.int/iris/bitstream/10665/75366/1/9789241504317_eng.pdf. Accessed 15 May 2013.
13. Baeten JM, et al. Antiretroviral prophylaxis for HIV prevention in heterosexual men and women. N Engl J Med. 2012;367(5):399–410.
14. Grant R, et al. Preexposure chemoprophylaxis for HIV prevention in men who have sex with men. N Engl J Med. 2010;363(27):2587–99.
15. Thigpen MC, et al. Antiretroviral preexposure prophylaxis for heterosexual HIV transmission in Botswana. N Engl J Med. 2012;367(5):423–34.
16. Choopanya K, et al. Antiretroviral prophylaxis for HIV infection in injecting drug users in Bangkok, Thailand (the Bangkok Tenofovir Study): a randomized, double-blind, placebo-controlled phase 3 trial. Lancet. 2013;381(9883):2083–90.
17. Guidance on oral pre-exposure prophylaxis (PrEP) for sero discordant couples, men and transgender women who have sex with men at high risk of HIV: recommendations for use in the context of demonstration projects. Geneva: World Health Organization; 2012. http://apps.who.int/iris/bitstream/10665/75188/1/9789241503884_eng.pdf. Accessed 15 May 2013.
18. Ansara DL, Hindin MJ. Formal and informal help-seeking associated with women's and men's experiences of intimate partner violence in Canada. Soc Sci Med. 2010;70:1011–8.
19. Weller SC, Davis-Beaty K. Condom effectiveness in reducing heterosexual HIV transmission. Cochrane Database Syst Rev. 2009;1:CD003255.
20. French PP, et al. Use-effectiveness of the female versus male condom in preventing sexually transmitted disease in women. Sex Transm Dis. 2003;30:433–9.
21. Frank AP, et al. Anonymous HIV testing using home collection and telemedicine counseling. A multicenter evaluation. Arch Intern Med. 1997;157:309–14.
22. Effectiveness of sterile needle and syringe programming in reducing HIV/AIDS among IDUs. Geneva: World Health Organization; 2004. www.who.int/hiv/pub/idu/e4a-needle/en/index.html. Accessed 15 May 2013.
23. Effectiveness of drug dependence treatment in preventing HIV among injecting drug users. Geneva: World Health Organization; 2003. www.who.int/entity/hiv/pub/idu/drugdependencefinaldraft.pdf. Accessed 15 May 2013.
24. Konopnicki D, et al. Hepatitis B and HIV: prevalence, AIDS progression, response to highly active antiretroviral therapy and increased mortality in the EuroSIDA cohort. AIDS. 2005;19:593–601.
25. Puoti M, et al. Mortality for liver disease in patients with HIV infection: a cohort study. J Acquir Immune Defic Syndr. 2000;2:211–7.

Ultrasonography in Labor

50

Neha Gupta

50.1 Introduction

Digital pelvic examination has been the gold standard of monitoring the progress of labor, even though it is quite uncomfortable for the patient. It is imprecise [1] and subjective in nature. It has high interobserver variations and inaccuracies [2] in the assessment, which is a well-established fact. With the advent of portable machines and availability of ultrasound machines in the labor room, now the concept of *sono-obstetrician* is evolving rather than just the obstetric sonogram.

Over the last 30 years, intrapartum sonography has gone a long way from the assessment of the fetal heart rate in labor or locating the placenta to assessing the progress of labor. There are now extensive studies available which explore the use of this noninvasive, cheap, and highly informative modality in assessing the progress of labor, identifying the cases of arrest of labor, and timely deciding the mode of delivery.

Hence, in this chapter, I outline the requirements for doing intrapartum sonography and its role during labor progression in accordance with the best available evidence and my own experience (Table 50.1).

Table 50.1 Uses of ultrasound in the intrapartum setting [3]

Basic uses
1. Identification of fetal heartbeat
2. Determination of fetal presentation
3. Estimation of fetal weight
4. Site of the placenta
5. Determination of the degree of flexion of the head
6. Assistance in the birth of second twin
7. Postpartum bleeding
Advanced uses
1. Determination of fetal head position, station, and rotation
2. Diagnosis of protracted labor
3. Assessment of intrapartum fetal well-being

50.2 General Setup During Labor

Examiner: The intrapartum ultrasound should be done by an expert who has the knowledge about the use of ultrasound as well its use in labor. As we still know, that use of ultrasound in labor is not a routine in many centers. Still, in our teaching, there is a great gap between knowing the delivery skills and obstetricians having sonography skills.

Ultrasound probes: It is not really required to have really expensive machines to do the intrapartum sonography. I have used convex abdominal as well as the volume probes. I have not used transvaginal probes for doing intrapartum ultrasound, though there are some studies which are using TVS probes for measuring preinduction cervical length.

N. Gupta (✉)
Jaypee Hospital, Noida, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_50

50.3 Ultrasound Machine Settings

The settings of the ultrasound machine to be employed for the adequate visualization of adequate intrapartum fetal head are [4]:

1. Lowest possible angle of insonation
2. Lower output frequency
3. Highest insonation depth
4. Wide volumetric area with low sound volumes

50.4 Types of Ultrasound Exploration Techniques

1. Transabdominal—The abdominal probe (convex or volume) is kept transversally in the suprapubic region of the maternal abdomen. This is mainly for assessing head rotation.
2. Translabial or transperineal—The abdominal probe is placed inside a rubber glove covered with an ultrasound gel and then placed longitudinally in the medial sagittal position between both labia majora and below the symphysis pubis with right side of the probe facing toward the anus (Fig. 50.1).
   Turing the probe transversely will give the cervix and head.

50.5 Ultrasound Prior to Induction of Labor

Induction of labor is a common obstetric intervention, performed in about 20% of pregnancies [6]. However, about 20% of women having induction of labor, need a cesarean section for delivery either because of failed induction, failure to progress in labor, or fetal distress [7].

50.5.1 Preinduction Cervical Length

The measurement of cervical length within 24 h prior to the induction of labor by means of transvaginal ultrasound is an excellent predictor of the likelihood of vaginal delivery.

The Pandis et al [8] suggested 28 mm for the cervical length and 3 as the Bishop score are the

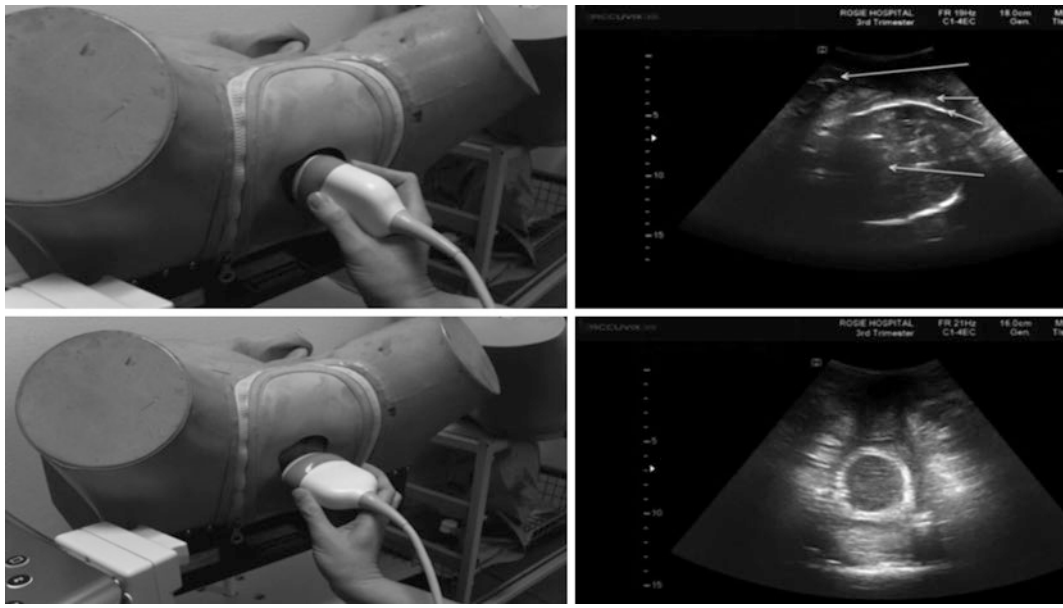


Fig. 50.1 Transperineal sagittal application of the 2D transducer (**a**) gives views of the maternal symphysis pubis and the upper part of the cervix lying just above the upper part of the fetal skull (**b**). Rotation of the 2D transducer by 90° (**c**) provides a view of the cervix (**d**) [5]

best cutoff points for the prediction of successful induction. However, the cervical length appears to be a better predictor than the Bishop score, with a sensitivity of 87% and a specificity of 71% compared to 58% and 77%, respectively [8].

There will be reduction in the need of prostaglandin by 50% for the induction of labor in nullipara at term if 28 mm is the cut off used for pre-induction transvaginal cervical length [9].

Other parameters like angle of progression and elastography score of the cervix have also been used to predict their accuracy, but have not found to be useful [10].

Preinduction transvaginal cervical length of 28 mm or less measurement is a useful predictor for the likelihood of vaginal delivery.

50.6 Ultrasound-Based Prolonged Pregnancy Clinic

The risk of intrauterine and postnatal death is increased from 2.4 per 1000 pregnancies at 40 weeks to 5.8 at 43 weeks. Hence, postdate pregnancy is the most common cause of induction of labor.

Rao et al. [11] proposed an ultrasound-based assessment of the cervix and well-being of the fetus around the 40th week of gestation. Unless there is evidence of a specific medical or obstetric indication, induction of labor is delayed by 7–10 days, thereby increasing the percentage of females going into spontaneous labor by about 80% [11].

Hence, the concept of ultrasound-based prolonged pregnancy clinic has been introduced.

50.7 Ultrasound During Labor

Digital vaginal examination is used to assess the progression of labor. The parameters assessed are the cervical dilatation and effacement and head rotation and station. All these parameters can be assessed by means of ultrasound as well. Just by using a combination of transabdominal and transperineal ultrasound, we can gauge the progress of labor.

The major advantage is that the ultrasound being noninvasive and nonintrusive modality, avoids the discomfort associated with the digital vaginal examination. We are not proposing to do away with the digital vaginal examination but to use ultrasound as an adjunct to it. By using ultrasound, the number of per vaginum examination can be decreased, and the incidence of chorioamnionitis can be decreased from 10% for 13 vaginal examinations [12] to 4% for 2 DVE.

50.7.1 First Stage

The parameters assessed in the first stage are **head rotation, head station, and cervical dilatation**.

50.7.1.1 Head Rotation

Head rotation is the easiest parameter to assess on the scan. As a obstetrician I feel, it is the most difficult parameter to assess on DVE especially in the presence of caput. It has been seen that the assessment of the position of the occiput is inaccurate in majority of the DVE (digital vaginal examinations) [12, 13].

When we do DVE, we assess the head rotation by determining the position of the posterior fontanel and label it according to a 12-hour clock (with 12.00 h representing occiput anterior position and 03.00 h representing left occiput transverse position). Almost similar method was proposed by Akmal et al. [14].

Fetal head rotation was defined with 2D ultrasound by keeping the probe transverse at the suprapubic region. Using the fetal spine or orbits as the landmark and occiput as the denominator, it was expressed according to a 12-hour clock as for the digital VE (Figs. 50.2 and 50.3)

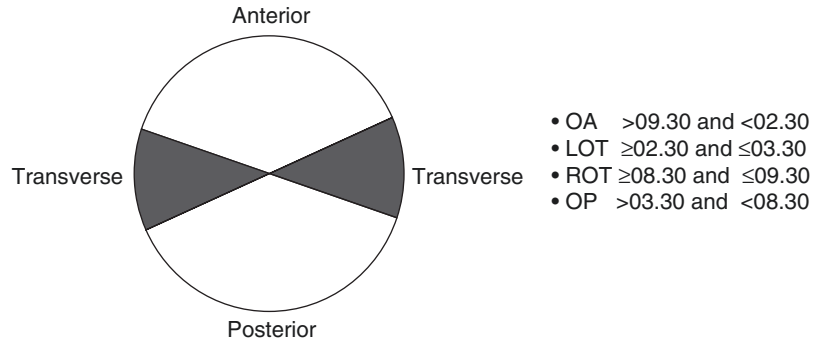


Fig. 50.2 Determination of occipital position

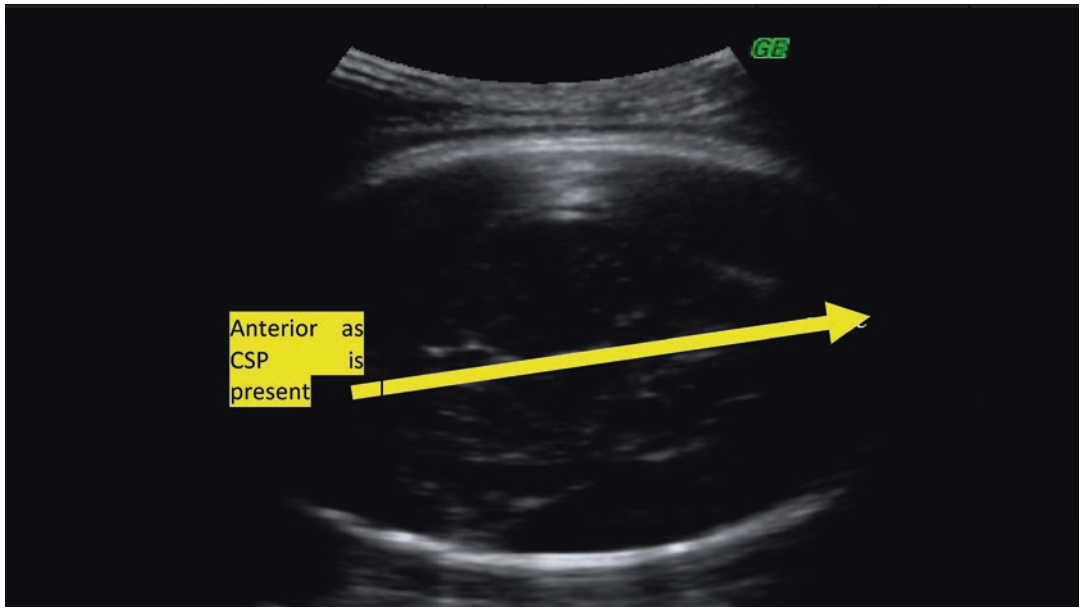


Fig. 50.3 Cavum septum pellucidum is present toward left and occiput toward right (GE denotes right side of the probe). The midline echo is almost horizontal as shown by arrow, hence ROT

The interobserver agreement was nearly 15° in 90% of cases and 30° in all cases for sonographically determined fetal occiput during labor [14].

If you have experience in the obstetric scanning, then determining the fetal position, based on such landmarks as the fetal orbits, cerebellum, midline echo of the brain, and occiput, is easy and highly reproducible (Figs. 50.3, 50.4, and 50.5).

The use of vaginal examination, transabdominal ultrasound, and transvaginal ultrasound to determine fetal head position was compared by Zahalka et al. They found that the transvaginal ultrasound was the most precise method [15].

A high body mass index is the only significant factor that can make transabdominal ultrasound exploration difficult.

When we did a pilot study on about 12 women, we found that head rotation was easily picked up on scan by suprapubic transabdominal scan in 100% of examinations.

50.7.1.2 Head Station

Ultrasound during labor can clearly determine the position of the fetal head. This information provides objective data to pinpoint the labors whose progress is slow.

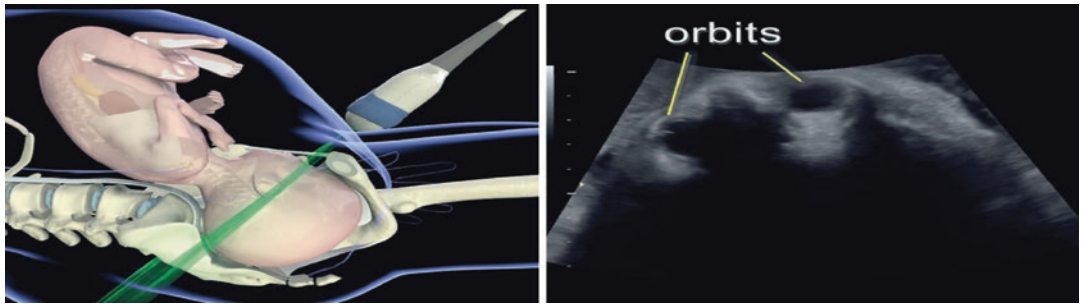


Fig. 50.4 If orbits are seen by suprapubic transabdominal scan—occipitoposterior position [16]

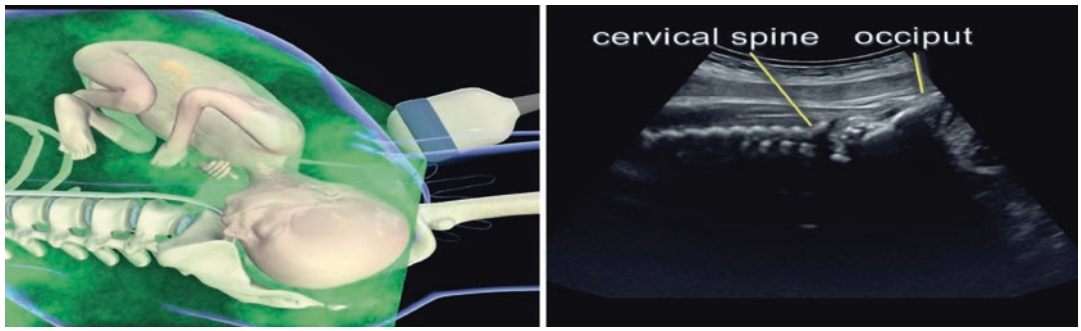


Fig. 50.5 If the spine is seen by suprapubic transabdominal scan—occipitoanterior position [16]

The reliability of DVE in the assessment of fetal head station was demonstrated by Dupuis et al. [17] using a birth simulator.

A fetal head mannequin was placed in the birth simulator and the operators then determined the head position clinically. The head position was incorrectly assessed by 50–88% of residents and in 36–80% of cases for obstetricians. The misdiagnosis of a station as midpelvic rather than high-pelvic accounted for 88% and 67% of the errors made by residents and obstetricians, respectively. This misinterpretation of the head station can have serious implications for the management of patients in labor.

Various parameters have been used to demonstrate head progression during labor. These were done translabially and the technique as described above was followed (Table 50.2):

1. Head–perineum distance (Fig. 50.6)—**The fetal head–perineum distance (HPD) is measured as the shortest distance from the outer bony limit of the fetal skull to the skin surface of the perineum by a transperineal ultrasound examination in a transverse view**. The transducer is moved until the shortest distance to the fetal head is visualized [18]. It is also used as a parameter to assess the head descent to assess progress of labor in SONOPARTOGRAM [19].

Table 50.2 Co-relation between fetal head descent and head–perineum distance [20]

	HPD (mm)
High cavity	50
Mid cavity	38
Low cavity	20

An advantage is that it is a quiet easy parameter to perform [18] which does not require much expertise. We also had used it and believe the same.

However, the measurements may vary with the degree of compression of the soft tissue especially when the head is not engaged.

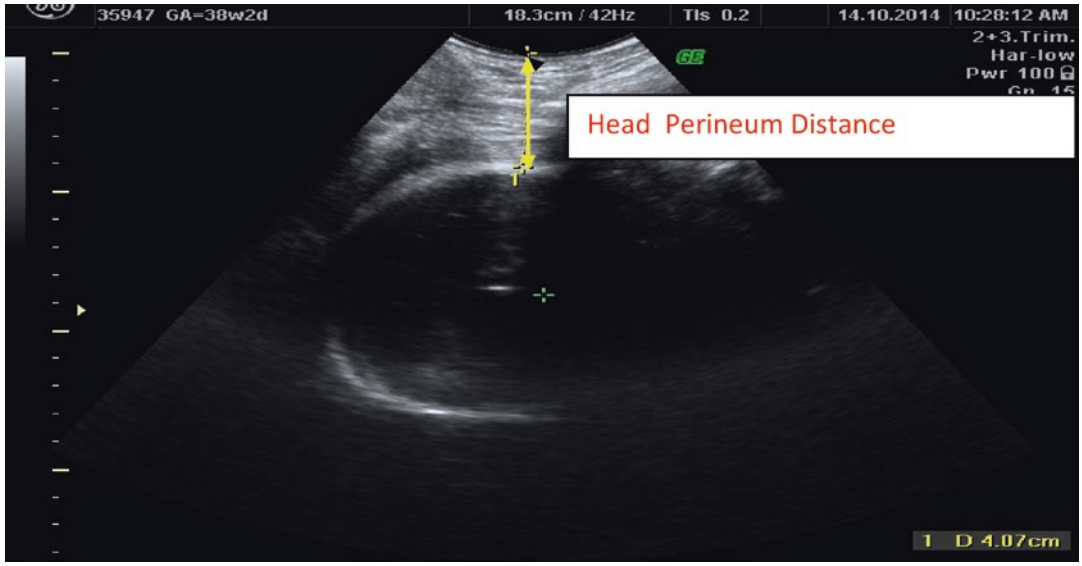


Fig. 50.6 Head–perineum distance—transperineal ultrasound with probe in transverse direction, shortest distance between the skull and skin of the perineum

2. **Angle of progression**—The most important measurement is the angle of progression of the fetal head. It is defined as the angle between a line through the midline of the pubic symphysis and a line from the inferior apex of the symphysis to the leading part of the fetal skull (Fig. 50.7).

   An angle of progression of 120° or greater is an excellent predictor of a successful vaginal delivery. Kalache et al. [21] evaluated this measurement prospectively in women at term with failure to progress in the second stage of labor and concluded that if the angle of progression was ≥120°, the probability of either an easy and successful vacuum extraction or a spontaneous vaginal delivery was 90%. Barbera et al. [22] found a good intra- and interobserver variability for measurements of the angle of progression.

The angle of progression >120° increases the likelihood of vaginal delivery. It has the best inter- and intra-observer reproducibility when studying fetal head descent during labor.

3. **Head direction** [23]—It is the line drawn through the midline of the fetal head and its correlation with the line drawn through the middle of the symphysis pubis (Fig. 50.8). Three types of head directions were determined: head down, horizontal, and head up:
   (a) Head up is when the head points upward.
   (b) Head down is when the leading part of the head is pointing downward in the pelvis.
   (c) All other angles are considered horizontal.

   The head direction, together with the descent in the maternal pelvis, is a good indicator of successful vaginal delivery. An upward direction of the fetal head is a good prognostic sign for vaginal delivery, in contrast with a downward or horizontal head direction.

   Among all parameters, this is the easiest to perform and understand. It does not involve any measurement and just a glance will let us know that:
   – If the head is down, then the head is in the upper 1/3 of the pelvis.
   – When horizontal, the head is in the midpelvis.

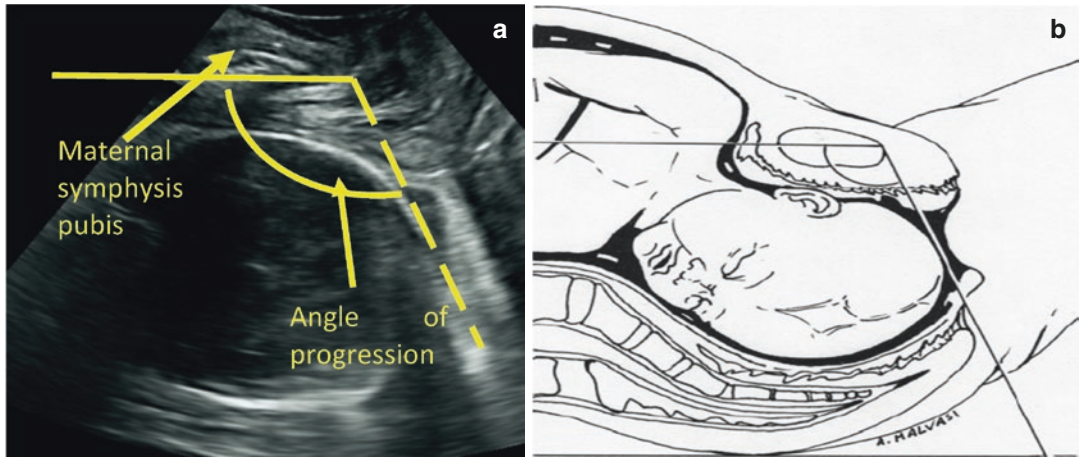


Fig. 50.7 (**a**) Ultrasound image and drawing demonstrating the angle of fetal head progression, described as the angle between a line through the midline of the pubic symphysis (*continuous yellow line*) and a line from the inferior apex of the symphysis to the leading part of the fetal skull (*interrupted yellow line*). (**b**) The image shows the equivalent diagram of the angle of the progression of the fetal head

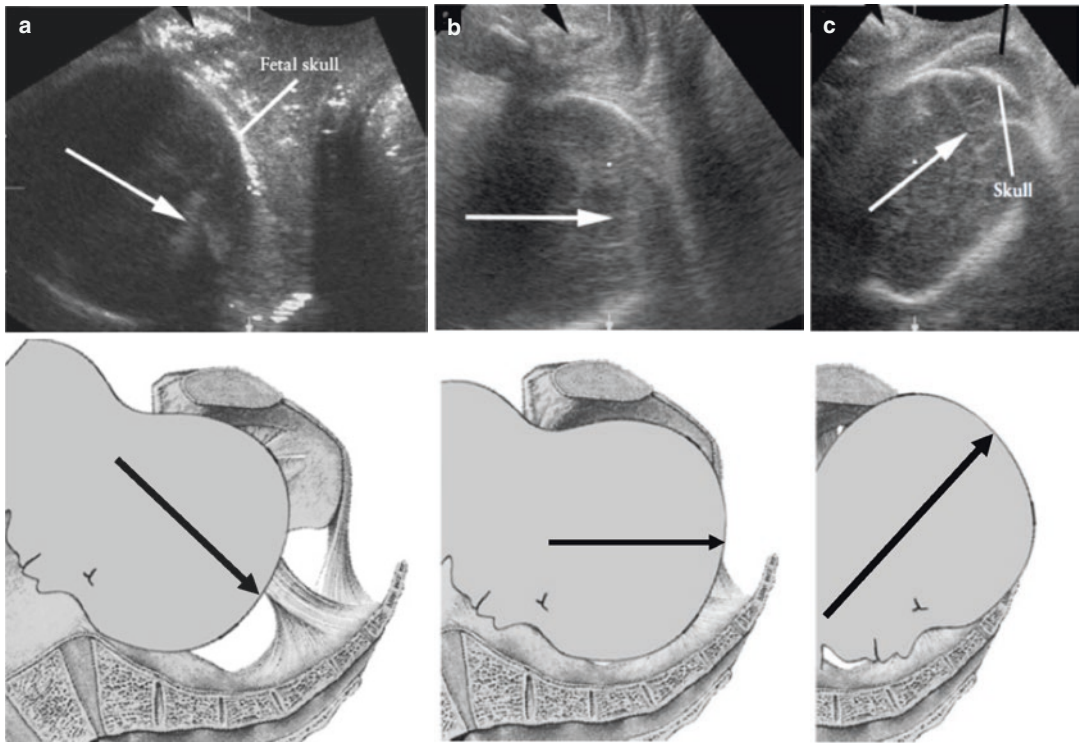


Fig. 50.8 Categorization of fetal head direction (indicated by black arrows) in longitudinal translabial sonograms compared: (**a**) downward direction; (**b**) horizontal direction; (**c**) upward direction [23]

– When pointing upward, then the head is in lower 1/3 of the pelvis [24].

4. **Head–symphysis distance (HSD)**—It is the distance between the lowest edge of the symphysis pubis and the nearest point of the fetal skull along a line passing perpendicular to the long axis of the symphysis pubis [25] (Fig. 50.9). As the head descends and rotates, HSD decreases.
5. **Head progression distance (HPD)**—This is done in transperineal sagittal mode. This was defined by Gilboa et al. [26] in the second stage of labor. The ultrasound transducer, covered with a glove and ultrasound gel, was placed on the perineum in a sagittal position (Fig. 50.10), and measurements were made during maternal pushing**.**

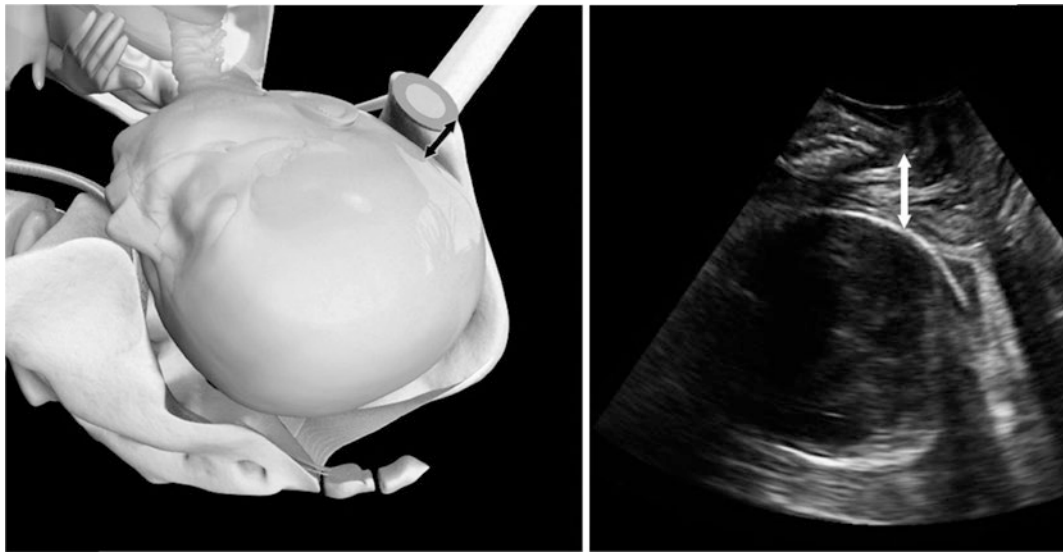


Fig. 50.9 Arrow denotes head–symphysis distance

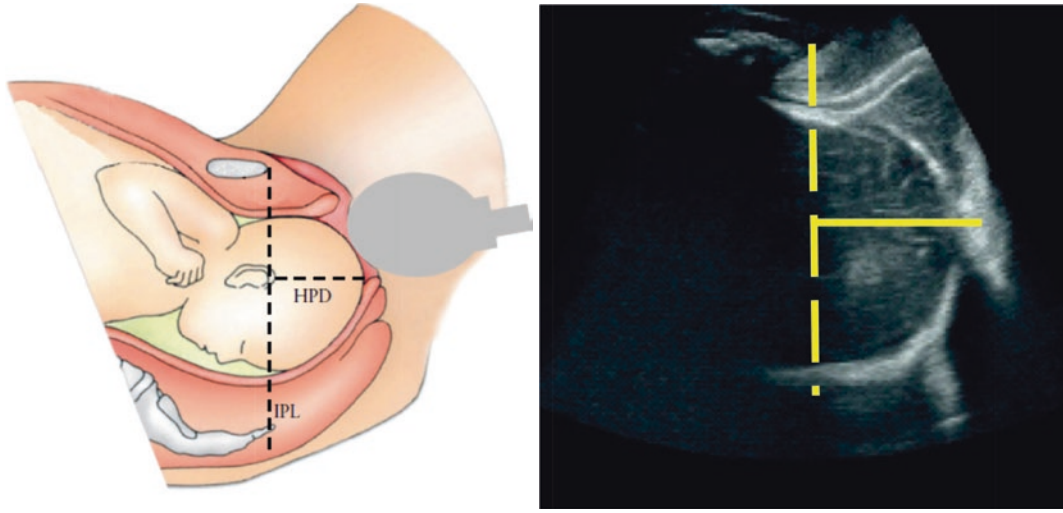


Fig. 50.10 Sonographic assessment of head progression distance (HPD): the transducer position on the perineum in the sagittal plane is tilted to demonstrate the pubic symphysis in a horizontal view. IPL, infrapubic line (Adapted from Kalache et al. [21])

The HPD was measured as the perpendicular distance from the IPL (a line drawn vertically from the inferior edge of the echogenic core of the symphysis pubis) to the lowest part of the fetal bony skull.

50.7.1.3 Cervical Dilatation

The difference of ≥2 cm in the measurement of cervical dilatation between the two examiners has been observed in more than 11% of occasions [1]. Electromechanical devices [27] and 3D ultrasonography [28] have also been used for the assessment of the cervical dilatation.

2D Method

The method of measurement of cervical dilatation by means of two-dimensional gray-scale ultrasound was introduced by Hassan et al. [5].

The transducer is placed on the perineum at the level of the posterior fourchette in a longitudinal position (Fig. 50.11a). By means of slight sideward movements of the probe, the views of the maternal pubic symphysis and fetal skull landmarks can be obtained (Fig. 50.11b).

The anterior part of the cervix was identified above the upper part of the fetal skull in a sagittal view. The transducer is then rotated by 90° (Fig. 50.11c) without losing the upper part of the cervix image. Slight sideward movements of the transducer were performed to obtain clear views of the cervix. The circular aspect of the cervix was obtained (Fig. 50.11d). The cervical dilatation was measured in the anteroposterior plane with the cursors placed on the inner part of the cervical tissue anteriorly and posteriorly (inner–inner) (Fig. 50.12).

According to them, there was positive correlation between 2D ultrasound measurement of

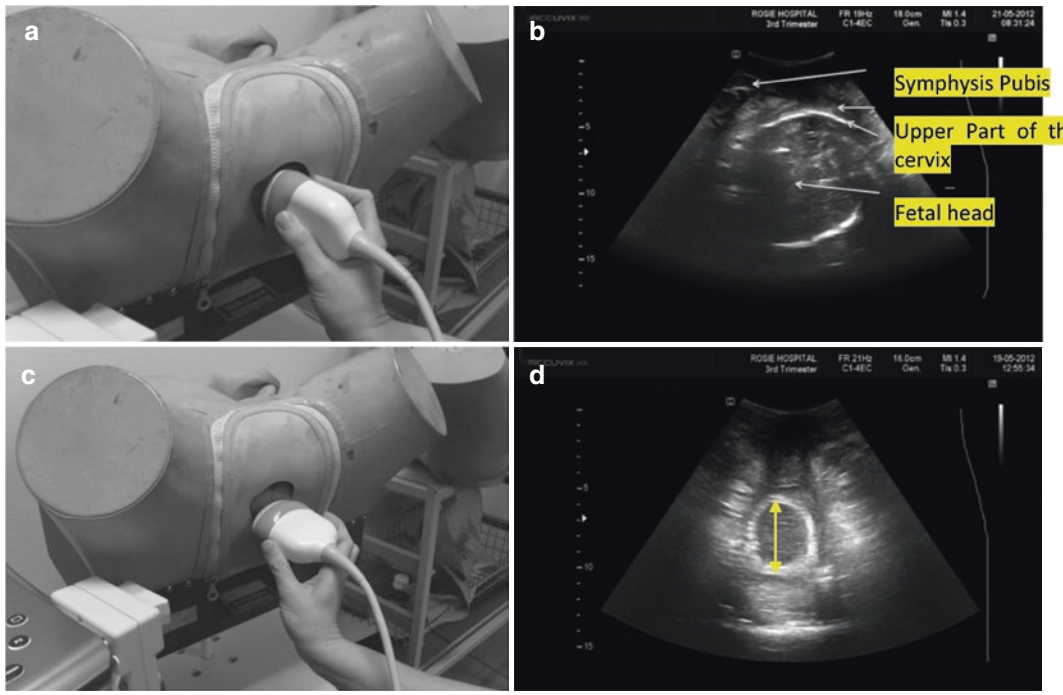


Fig. 50.11 Two-dimensional (2D) ultrasound technique to measure cervical dilatation in labor. Transperineal sagittal application of the 2D transducer (**a**) gives views of the maternal symphysis pubis and the upper part of the cervix lying just above the upper part of the fetal skull (**b**). Rotation of the 2D transducer by 90° (**c**) provides a view of the cervix (**d**)

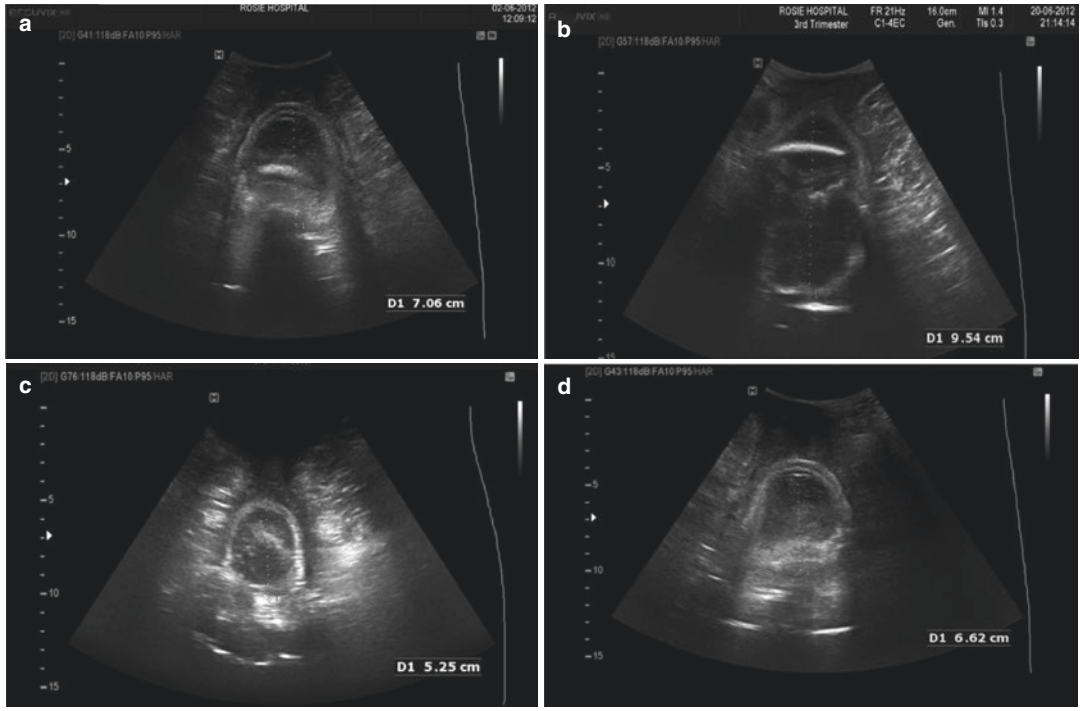


Fig. 50.12 Representative images obtained by two-dimensional (2D) ultrasound at cervical dilatations of (**a**) 7.1 cm, (**b**) 9.5 cm, (**c**) 5.3 cm, and (**d**) 6.6 cm

cervical dilatation and digital vaginal examination ($P < 0.001$).

Assessment of Progression of Labor

Sonopartogram [19] is the concept introduced by Hassan et al. It is a sonographic analysis of the progress of labor. It is a simple and an objective way of monitoring the key parameters of labor. The sonopartogram is based on the conventional partogram, but adapted for recording of ultrasound parameters (Fig. 50.13):

1. Cervical dilatation—based on 2D method described above.
2. Fetal head descent as head–perineum distance.
3. Fetal head rotation on a 12-hour clock.
4. Caput—transperineally in a sagittal view as the maximum distance between the fetal skin and bone of the leading arc of the skull (proximal skull to outer skin).
5. Molding is seen when the skull bones of the vertex overlap.

The acquisition of data regarding the progress of labor was more complete for the sonopartogram than the conventional partogram. The agreement between digital VE and US was good for cervical dilatation and head rotation but less for head descent. US assessment of the progress of labor is feasible in most cases.

When we did the pilot study in our department on 15 women, our experience was that the data could be acquired in all the cases. The head rotation and HPD were the easiest to measure. The cervical dilatation on ultrasound needs little practice. But with this sonopartogram, we could reduce the number of per vaginum examinations. It gave as an insight into stalled labor as occipitoposterior or still in transverse position after the full dilatation.

Caput and molding could easily be assessed.

What I felt was that head–perineum distance was not well co-relating with the head station and hence preferred the use of HEAD DIRECTION to assess the head station, and

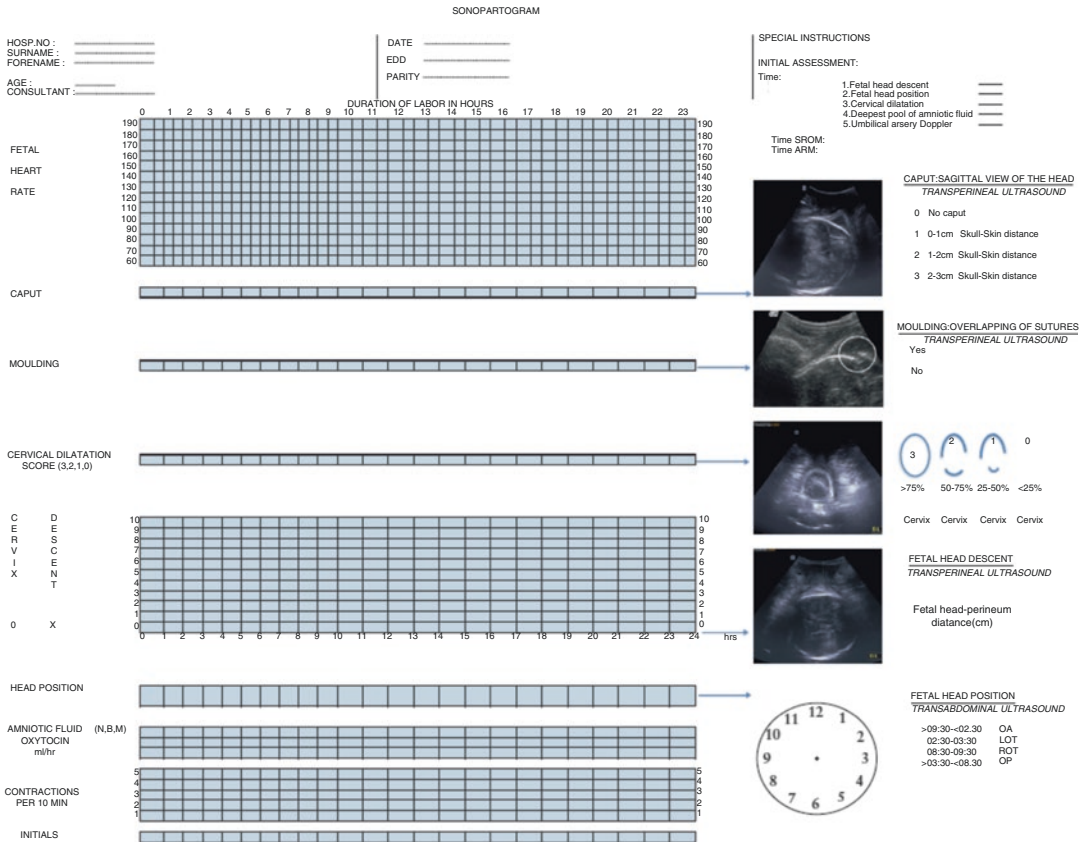


Fig. 50.13 A sample sonopartogram showing fetal head descent, cervical dilatation, and head rotation with explanatory ultrasound images

hence we used a modified version of sonopartogram—MODIFIED SONOPARTOGRAM.

Assessment of Intrapartum Fetal Well-Being (Intrapartum Doppler)

Uterine, umbilical, middle cerebral arteries and ductus have been studied for the assessment of intrapartum detection of acidosis [29]. However, till now none seems promising. The use of these tools is limited to the context of research trials.

50.7.2 Second Stage

The second stage starts after the full dilatation of the cervix:

1. Head progression distance, angle of progression, and head direction—all seen on transperineal sagittal scan that can provide great details about the head descent.
2. The head rotation can be easily assessed transabdominally, sometimes when the head has entered the pelvis and is usually greater than 0 station. Midline angle [30] is a useful parameter in that aspect.

Midline angle—on transperineal axial plane—is the angle between the anteroposterior axis of the maternal pelvis. It is helpful in assessing the angle of rotation of the fetal skull (< or >45) (Fig. 50.14).

Advantages of using intrapartum assessment of labor

1. With the systematic use of these parameters in women for whom no or inadequate progression of labor is suspected, we could

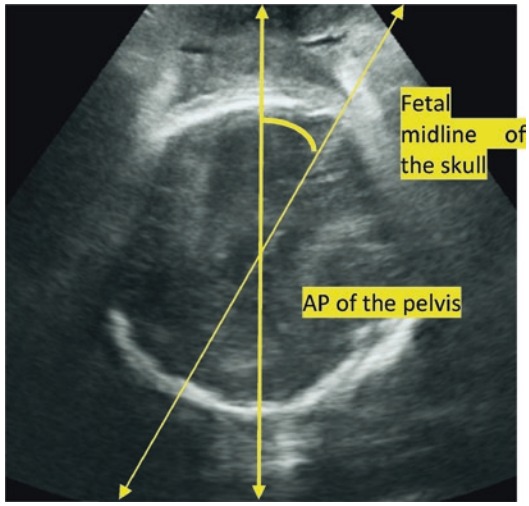


Fig. 50.14 Midline angle

objectively demonstrate the presence of such diagnoses. These parameters would result in more accurate diagnoses and more exact indications for C-sections.

2. Diagnosis of fetal head position in protracted labors especially in the presence of caput and molding—as occipitoposterior position [31] would provide explanation to that.
3. Head station, degree of head rotation, and amount of caput and molding can be assessed sonographically, and it can be utilized for the application of vacuum or forceps [32]. It will minimize the failure associated with the same.

50.7.3 Third Stage of Labor

Third stage of labor is defined as the part of labor from the birth of the baby until the placenta (afterbirth) and fetal membranes are delivered.

Krapp et al. used real-time and color Doppler imaging to characterize the normal and abnormal third stage of labor [33]. Placental separation from the myometrium was defined clinically and correlated with cessation of color Doppler-detected blood flow in basal plate vessels.

Three sonographic phases of separation were the interval between delivery of the fetus and the beginning of placental separation (latent phase), the monophasic or multiphasic shearing off of the placenta (detachment phase), and the interval between completed placental separation and vaginal delivery of the placenta (expulsion phase) [33].

Cessation of blood flow between the basal placenta and myometrium following delivery of the fetus was the sonographic hallmark of normal placental separation. Persistent blood flow demonstrated by color Doppler sonography was suggestive of placenta accreta.

50.8 Conclusion

Availability of portable ultrasound machines has made its way in intrapartum ultrasonography such that now from ultrasonographic obstetrics, we are becoming sono-obstetricians. Increasing utilization is improving our knowledge of dynamics of childbirth. Sonopartogram is a good tool to assess the progress of labor. As an obstetrician and fetal medicine specialist, I believe ultrasonographic examination provides adjunctive information about the progress of labor along with per vaginum examination.

References

1. Buckmann EJ, Libhaber E. Accuracy of cervical assessment in the active phase of labour. BJOG. 2007;114:833–7.
2. Tuffnell DJ, Bryce F, Johnson N, Lilford RJ. Simulation of cervical changes in labour: reproducibility of expert assessment. Lancet. 1989;2:1089–90.
3. Barber M, Gutierrez L, Plasencia W, Valle L, Garcia-Hernandez JA. Role of ultrasound in the labor ward. J Matern Fetal Neonatal Med. 2010;30(3):241–3.
4. Ghi T, Contro E, Farina A, Nobile M, Pilu G. Three-dimensional ultrasound in monitoring progression of labor: a reproducibility study. Ultrasound Obstet Gynecol. 2010;36(4):500–6.
5. Hassan WA, Eggebø TM, Ferguson M, Lees C. Simple two-dimensional ultrasound technique to assess intrapartum cervical dilatation: a pilot study. Ultrasound Obstet Gynecol. 2013;41:413–8.
6. National Institute for Health and Care Excellence. Inducing labour (Clinical Guideline 70). 2008. https://www.nice.org.uk/guidance/CG70. Accessed 13 March 2019.

7. Crowley P. Interventions for preventing or improving the outcome of delivery at or beyond term. Cochrane Database Syst Rev. 2000;2:CD000170.
8. Pandis GK, Papageorghiou AT, Ramanathan VG, Thompson MO, Nicolaides KH. Preinduction sonographic measurement of cervical length in the prediction of successful induction of labour. Ultrasound Obstet Gynecol. 2001;18(6):623–8.
9. Park KH, Kim SN, Lee SY, Jeong EH, Jung HJ, Oh KJ. Comparison between sonographic cervical length and Bishop score in preinduction cervical assessment: a randomized trial. Ultrasound Obstet Gynecol. 2011;38:198–204.
10. Pereira S, Frick AP, Poon LC, Zamprakou A, Nicolaides KH. Successful induction of labor: prediction by preinduction cervical length, angle of progression and cervical elastography. Ultrasound Obstet Gynecol. 2014;44:468–75.
11. Rao A, Celik E, Poggi S, Poon L, Nicolaides K. Cervical length and maternal factors in expectantly managed prolonged pregnancy: prediction of onset of labor and mode of delivery. Ultrasound Obstet Gynecol. 2008;32:646–65.
12. Westover T, Knuppel RA. Modern management of clinical chorioamnionitis. Infect Dis Obstet Gynecol. 1995;3:123–1132.
13. Sherer DM, Miodovnik M, Bradley KS, Langer O. Intrapartum fetal head position I: comparison between transvaginal digital examination and transabdominal ultrasound assessment during the active stage of labor. Ultrasound Obstet Gynecol. 2002;19:258–63.
14. Akmal S, Tsoi E, Kametas N, Howard R, Nicolaides KH. Intrapartum sonography to determine fetal head position. J Matern Fetal Neonatal Med. 2002;12:172–7.
15. Zahalka N, Sadan O, Malinger G, Liberati M, Boaz M, Glezerman M, et al. Comparison of transvaginal sonography with digital examination and transabdominal sonography for the determination of fetal head position in the second stage of labor. Am J Obstet Gynecol. 2005;193(2):381–6.
16. Youssef A, Ghi T, Piluisuog G. How to perform ultrasound in labor: assessment of fetal occiput position. Ultrasound Obstet Gynecol. 2013;41:476–8.
17. Dupuis O, Silveira R, Zentner A, Dittmar A, Gaucherand P, Cucherat M, Redarce T, Rudigoz RC. Birth simulator: reliability of transvaginal assessment of fetal head station as defined by the American College of Obstetricians and Gynecologists classification. Am J Obstet Gynecol. 2005;192:868–74.
18. Eggebø TM, Heien C, Økland I, Gjessing LK, Romundstad P, Salvesen KA. Ultrasound assessment of fetal head-perineum distance before induction of labor. Ultrasound Obstet Gynecol. 2008;32(2):199–204.
19. Hassan WA, Eggebø T, Ferguson M, Gillett A, Studd J, Pasupathy D, Lees CC. The sonopartogram: a novel method for recording progress of labor by ultrasound. Ultrasound Obstet Gynecol. 2014;**43**:189–94.
20. Maticot-Baptista D, Ramanah R, Collin A, Martin A, Maillet R, Riethmuller D. Ultrasound in the diagnosis of fetal head engagement. A preliminary French prospective study. J Gynecol Obstet Biol Reprod. 2009;38(6):474–80.
21. Kalache KD, Duckelmann AM, Michaelis SAM, Lange J, Cichon G. Transperineal ultrasound imaging in prolonged second stage of labor with occipitoanterior presenting fetuses: how well does the 'angle of progression' predict the mode of delivery? Ultrasound Obstet Gynecol. 2009;33:326–30.
22. Barbera AF, Pombar X, Perugino G, Lezotte DC, Hobbins JC. A new method to assess fetal head descent in labor with transperineal ultrasound. Ultrasound Obstet Gynecol. 2009;33(3):313–9.
23. Henrich W, Dudenhausen J, Fuchs I, Kämena A, Tutschek B. Intrapartum translabial ultrasound (ITU): sonographic landmarks and correlation with successful vacuum extraction. Ultrasound Obstet Gynecol. 2006;28(6):753–60.
24. Ghi T, Farina A, Pedrazzi A, Rizzo N, Pelusi G, Pilu G. Diagnosis of station and rotation of the fetal head in the second stage of labor with intrapartum translabial ultrasound. Ultrasound Obstet Gynecol. 2009;**33**:331–6.
25. Youssef A, Maroni E, Ragusa A, De Musso F, Salsi G, Iammarino MT, Paccapelo A, Rizzo N, Pilu G, Ghi T. Fetal head–symphysis distance: a simple and reliable ultrasound index of fetal head station in labor. Ultrasound Obstet Gynecol. 2013;**41**:419–24.
26. Gilboa Y, Kivilevitch Z, Spira M, Kedem A, Katorza E, Moran O, Achiron R. Head progression distance in prolonged second stage of labor: relationship with mode of delivery and fetal head station. Ultrasound Obstet Gynecol. 2013;**41**:436–41.
27. Lucidi RS, Blumenfeld LA, Chez RA. Cervimetry, a review of methods for measuring cervical dilatation during labour. Obstet Gynecol Surv. 2000;**55**:312–20.
28. Zimerman AL, Smolin A, Maymon R, Weinraub Z, Herman A. American Institute of Ultrasound in Medicine. Intrapartum measurement of cervical dilatation using translabial 3-D ultrasonography: correlation with digital examination and interobserver and intraobserver agreement assessment. J Ultrasound Med. 2009;28:1289–96.
29. Stuart IP, Lindow SW, van der Elst CD. Fetal acidosis and Doppler velocimetry of umbilical artery in labour. UOG. 1993;3:256–9.
30. Ghi T, Youssef A, Maroni E, Arcangeli T, De Musso F, et al. Intrapartum transperineal ultrasound assessment of fetal head progression in active second stage of labor and mode of delivery. Ultrasound Obstet Gynecol. 2013;**41**:430–5.
31. Blasi A, D'Amico R, Fenu V, Volpe A, Fuchs I, Henrich W, Mazza V. Sonographic assessment of fetal spine and head position during the first and second stages of labor for the diagnosis of persistent occiput posterior position: a pilot study. Ultrasound Obstet Gynecol. 2010;**35**:210–5.

32. Akmal S, Kametas N, Tsoi E, Hargreaves C, Nicolaides KH. Comparison of transvaginal digital examination with intrapartum sonography to determine fetal head position before instrumental delivery. Ultrasound Obstet Gynecol. 2003;21:437–40.

33. Krapp M, Baschat AA, Hankeln M, Gembruch U. Gray scale and color Doppler sonography in the third stage of labor for early detection of failed placental separation. Ultrasound Obstet Gynecol. 2000;15:138–42.

Cardiopulmonary Resuscitation

51

Gian Chauhan and Kartik Syal

51.1 Introduction

Cardiac arrest in pregnancy is among the most challenging clinical scenarios. Although most features of resuscitating a pregnant woman follows same protocols as of adult resuscitation, but several aspects and considerations are uniquely different. The most obvious differences in pregnant woman resuscitation are that there are two lives involved in it, the mother and the fetus, and physiological changes in pregnancy, which are not there in nonpregnant adult patient. Inpatient sample from the USA nationwide suggests that cardiac arrest occurs in 1:12,000 admissions for delivery [1]. Globally, 800 maternal deaths occur daily [2, 3]. The Centers for Disease Control and Prevention have documented steady increase in maternal mortality trends in the USA, since 1989 to 2009, from 7.2 deaths per 100 000 live births in 1987 to 17.8 deaths per 1,00,000 live births in 2009 [4].

Management of cardiac arrest in pregnancy, labor, and delivery has received very limited attention. Knowledge deficits [5, 6]and poor resuscitation skills [7] could be major contributor to poor outcomes. Despite these problems, recent data show that the rate of survival after maternal cardiac arrest may be as high as 58.9% [1]; though higher than in most other arrest populations, this class of patients has mostly healthy bodies and comes into dangerous situation only due to exaggeration of physiological changes; thus further improvement of survival justifies appropriate training and preparation for such events despite their rarity.

The best outcome for neonatal survival is likely to be achieved only by successful maternal resuscitation. Some of the key steps in resuscitating a pregnant woman are timely initiation of uninterrupted chest compressions, left lateral displacement of uterus, midsternal hand positioning, use of small size endotracheal tube, continuous cricoid pressure, and intravenous access above the gravid uterine level; but there are many roadblocks in proper implementation of these protocols, which needs urgent attention.

First and foremost, since maternal cardiac arrest is such a rare occurrence on labor and delivery floors, obstetric care providers have very infrequent exposure to this catastrophic situation. Current advanced cardiac life support (ACLS) requirements and training are insufficient for sustaining resuscitation skills [8]. Surveys conducted on obstetric anesthesiologists, obstetricians, and emergency care physicians have found the knowledge regarding basic concepts of CPR in pregnant women to be grossly inadequate [5, 9]. They recommend that ACLS and CPR for parturients should be taught in a better manner and repeated at regular intervals to practitioners at all levels.

G. Chauhan (✉) · K. Syal
Department of Anaesthesia, IGMC, Shimla, Himachal Pradesh, India

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Secondly, the single most important step in successful resuscitation [10] is timely initiation of uninterrupted chest compressions along with recognition of cardiac rhythm and timely use of defibrillator. Further, titling the pregnant patient during the cardiac arrest usually takes time, resulting in significant uncalled interruptions in chest compressions. Supine manual leftward displacement (LUD) of the uterus has been found to be as effective as a left lateral tilt [11]. A recent simulation study shows [12] that this position could also be favorable for rescuers to perform high-quality supine chest compressions [13].

Thirdly, recent AHA guidelines emphasize more on initiating cesarean delivery within 4 min of maternal cardiac arrest [14]. Since 30% of the cardiac output is shunted to gravid uterus and unless the fetus is delivered within this time period, cardiac compressions may not be effective in maintaining the maternal cardiac output.

When performed, cesarean delivery should occur at the site of the arrest. Equipments for cesarean delivery should be readily available. Hospital should develop clear protocols to establish first responder's role and have the appropriate number of staff to respond to a pregnant patient in cardiac arrest. Hospital should also have clear and effective system of activation of maternal cardiac arrest team, with roles clearly defined.

All the important factors related to maternal arrest, including maternal physiology, as it relates to resuscitation, causes of arrest, pre-event planning of the critically ill pregnant patient, risk stratification during pregnancy, management of the critically ill pregnant patient, basic life support (BLS), advanced cardiovascular life support (ACLS) in pregnancy, neonatal issues, emergency medical service (EMS) care, immediate postarrest care, medicolegal considerations (emergency doctrine principle works here, implying lack of intervention would have led to patient deterioration), and knowledge translation, training, and education recommendations, all are the hallmark of improving maternal cardiac arrest outcome.

51.2 Causes of Cardiac Arrest in Pregnancy

The most common causes of cardiac arrest are hemorrhage, cardiovascular diseases (myocardial infarction, aortic dissection, and myocarditis), amniotic fluid embolism, sepsis, aspiration pneumonitis, pulmonary embolism, and eclampsia [1, 15]. Important iatrogenic causes of maternal cardiac arrest include hypermagnesemia from magnesium sulfate administration and anesthetic complications.

After hemorrhage, common cardiac cause for cardiac arrest during pregnancy is myocardial infarction which may be due to premature coronary artery/aortic atherosclerosis or dissection. This relates in part to an increase in proportion of pregnancies in women post 35 years of age. The paradoxical, generally unlooked for, age- and gender-related incidence of myocardial infarction in this category of patients places them at a position of risk having previously unrecognized heart disease, making it a particular challenge for prediction and prevention. The second most common cardiovascular cause is aortic dissection/aneurysm, and the third is the category of complex congenital heart diseases. The latter category is at the other extreme of risk, with an extraordinarily high incidence of cardiac arrest and death during pregnancy, labor, and delivery. The most common etiologies of maternal arrest are given in Table 51.1.

51.3 Important Physiological Changes in Pregnancy

Fetal development and maternal maintenance of pregnancy require multiorgan physiological adaptations that are important to understand for the team responding to cardiopulmonary arrest during pregnancy.

51.3.1 Cardiovascular System

Cardiac output rises 30–50% as a result of increased stroke volume and, to a lesser extent,

Table 51.1 Most common etiologies of maternal arrest and mortality

Letter	Cause	Etiology	
A	Anesthetic complications Accident/trauma	High neuraxial block Hypotension Loss of airway Aspiration Respiratory depression Local anesthetic systemic toxicity Trauma Suicide	
B	Bleeding	Coagulopathy Uterine atony Placenta accrete Placenta Abruption Placenta previa Retained product of conception Uterine rupture Surgical Transfusion reaction	
C	Cardiovascular causes	Myocardial infarction Aortic dissection Cardiomyopathy Arrhythmias Valve disease Congenital heart disease	
D	Drugs	Oxytocin Magnesium Drug error Opioids Insulin Anaphylaxis	
E	Embolic causes	Amniotic fluid embolus Pulmonary embolus Cerebrovascular event Venous air embolism	
F	Fever	Sepsis Infection	
G	General	**5Hs**	**5Ts**
		Hypovolemia	**T**ension pneumothorax
		Hypoxia	**T**amponade cardiac
		Hydrogen ion (acidosis)	**T**oxins
		Hypo-/hyperkalemia	**T**hrombosis pulmonary
		Hypothermia	**T**hrombosis coronary
H	Hypertension	Preeclampsia Eclampsia HELLP syndrome Intracranial bleed	

increased maternal heart rate (15–20 bpm) [16, 17]. Systemic vascular resistance decreases as a result of an increase in several endogenous vasodilators, including progesterone, estrogen, and nitric oxide, leading to a decrease in mean arterial pressure, reaching a nadir in the second trimester [18]. The enlarging uterus can produce increased afterload through compression of the aorta and decreased venous return through compression of the inferior vena cava, starting at 12–14 weeks of gestational age [19]. As a result, the supine position, which is most favorable for resuscitation, can lead to hypotension [19, 20]. A magnetic resonance imaging study comparing

the maternal hemodynamic in the left lateral position with those in the supine position was performed [21]. The study found that at 20 weeks of gestational age, there was a significant increase in ejection fraction of 8% and stroke volume of 27% in the left lateral position. At 32 weeks, there was increase in ejection fraction of 11%, in end-diastolic volume of 21%, in stroke volume of 35%, and in cardiac output of 24% in the left lateral position [21].

These changes put the heart in higher-than-normal workload adapted somewhat by hormone-induced decreased SVR which is partially obliterated by increasing gravid uterus. Thus very less margin for necessity to increase cardiac output is left in later half of pregnancy. Also there is significant compression of large vessels by the growing uterus which impedes resuscitation efforts.

Uteroplacental blood flow increases from 50 to close to 1000 mL/min during pregnancy, which is up to a maximum of 20% of maternal cardiac output at term [22]. Expanded intravascular space and a fall in uterine vascular resistance facilitate sufficient placental blood flow. Overall uterine vascular reactivity is altered, which is characterized by decreased tone, enhanced vasodilation, and blunted vasoconstriction, thus *bringing the fetus in greater advantage in view of critically ill scenarios*. Systemic hypotension though can overwhelm the compensatory mechanisms, which attempt to maintain uterine blood flow.

51.3.2 Respiratory System

Functional residual capacity decreases by 10–25% during pregnancy as the uterus enlarges and elevates the diaphragm. Increased ventilation (i.e., increase in tidal volume and minute ventilation) occurs, beginning in first trimester, reaching at a level 20–40% above baseline by term, mediated by the elevated serum progesterone level [23]. This produces a mild respiratory alkalosis with compensatory renal excretion of bicarbonate, resulting in an arterial carbon dioxide pressure of ~28–32 mmHg (3.7–4.3 kPa) and plasma bicarbonate level of 18–21 meq/L [24]. *These changes mask the symptoms of some sinister problems like embolism which may lead to a "near-miss" situation*.

Oxygen consumption increases because of the demand of fetus and maternal metabolic processes, reaching at a level 20–33% above baseline by third trimester [25]. *The reduced functional residual capacity reservoir and increased consumption of oxygen are responsible for the rapid development of hypoxemia* in response to hypoventilation or apnea in the pregnant woman [26].

The oxyhemoglobin dissociation curve is shifted to the right in the mother during pregnancy (P_{50} increases from 27 to 30 mmHg). A higher partial pressure of oxygen is therefore required to achieve the same maternal oxygen saturation. The same curve is shifted to the left in the fetus (P_{50} is 19 mmHg) conferring relative resilience to hypoxic conditions.

51.3.3 Airway

Upper airway edema and friability occur as a result of hormonal effects and may reduce visualization during laryngoscopy and increase the risk of bleeding; *thus increased chances of ventilation and/or intubation failure are there*. This, along with rapid occurrence of hypoxia in pregnancy, gives the obstetric anesthesiologists and obstetricians very less time to save two lives. Thus emergency kits with all working intubation aids should always be present in the labor room.

51.3.4 Renal System

Pregnancy is characterized by glomerular hyperfiltration and increased renal blood flow by 40% to accommodate for fetal detoxification of metabolic by-products too and maintenance of maternal osmoregulation (there is increased circulatory intravascular volume). Altered tubular function prevents wasting of glucose, amino acids, and proteins which are essential for both maternal and fetal metabolisms. On balance, Starling forces favor a narrowing of the oncotic pressure-wedge pressure gradient, *increasing the tendency for pulmonary edema to develop* [27].

51.3.5 Gastrointestinal System

Progesterone relaxes gastrointestinal sphincters and prolongs transit time throughout the intestinal tract during the second and third trimesters [28, 29] *predisposing the patient to aspiration of stomach contents*. Gravid uterus pushing gastroesophageal sphincter into thorax causes anatomical and, thus, physiological changes in it, thus aggravating the condition.

51.4 Gestational Age Estimation

Decisions made during a maternal cardiac arrest may require estimation of gestational age, though it is made unimportant in protocol-based guidelines. Symphysis fundal height is measured from the top of the maternal pubic bone to the top of the uterine fundus. In a singleton pregnancy, with the fetus in a longitudinal lie, this height in centimeters will approximately correspond to the period of gestation in weeks if measured between 16 and 36 weeks of gestation. If a tape measure is not available, finger breadths are usually used as a surrogate for the centimeters. Classically accepted rule-of-thumb landmarks may be used: Gestational age is 12 weeks if the uterus is palpable at above the pubic symphysis, 20 weeks if the uterus is palpable at the level of umbilicus [30], and 36 weeks if the uterus is palpable at the level of the xiphisternum. However fundus can be a poor predictor of the gestational age and may reach the umbilicus between 15 and 19 weeks of gestation [31]. In the last month of pregnancy, after 36 weeks of gestation, there may be diminution of the fundal height from 36 weeks down to ~32 weeks as the fetal head engages into the pelvis. Fundal height may also be skewed by other factors such as abdominal distention [30] and increased body mass index; therefore, fundal height may be a poor predictor of gestational age.

The rule of the thumb in maternal resuscitation is that if the gravid uterus is felt at the umbilicus, it is taken to be as of 20 weeks, the importance being that pregnant patient with uterus below umbilicus is managed with resuscitation protocols similar to adult nonpregnant patients, whereas if the uterus is felt at or above the umbilicus, it is taken to be causing significant aortocaval compression [20]*, and there are unique important changes in resuscitation protocols which are to be followed essentially.*

51.5 The Critically Ill Pregnant Patient

51.5.1 Pre-event Planning

The critically ill pregnant patient may be managed in units not accustomed to managing obstetric patients such as the intensive care unit (ICU), coronary care unit (CCU), or medical or surgical ward. Teams on these units need to prepare for unexpected emergencies in these patients [32] by covering the following four important steps:

1. *Preparation for cardiac arrest*: Educate staff about the management of cardiac arrest in pregnancy.
2. *Prepare for perimortem cesarean delivery (PMCD)*: Identify contacts details or appropriate code calls to mobilize the entire maternal cardiac arrest response team, and ensure the availability of equipment for cesarean delivery and resuscitation of the neonate. In cultures that require consent for a PMCD, even in the event of cardiac arrest, pre-event consent should be obtained.
3. *Preparation for management of obstetric complications*: Stock drugs and equipment commonly available in obstetric units, including oxytocin and prostaglandin $F_2\alpha$. Pre-event planning for power of attorney related to healthcare decisions should be done for the critically ill patient.
4. *Decision involving the resuscitation status of the neonate*: Decision about the fetal viability should be made in collaboration with the obstetrician, neonatologist, and family. The decision depends upon the gestational age and, to a significant degree, the neonatal facilities available. This information should be clearly documented.

51.5.2 Severity of Illness and Early Warning Scores

The British Centre for Maternal and Child Enquiries report of 2011 (2006–2008 triennium) has stated that timely recognition of pregnant women at risk of potentially life-threatening conditions plays an important role in the appropriate institution of treatment [33]. Brief checklists are provided for the identification of a number of conditions, including sepsis, respiratory distress, and neurological complications. The report also highlights the potential value of modified early obstetric warning scores. In a more recent publication, using a large British ICU data set, Carl et al. [34] described the evaluation of several preexisting obstetric early warning scores and the development and validation of a new obstetric score and demonstrated excellent discrimination between survivors and nonsurvivors for this new score. These scores can be used to monitor patients by clinical use of an early warning score chart (Fig. 51.1) and can accurately identify patients at high risk of mortality, although not specifically mortality resulting from cardiac arrest. Therefore, they are of value in patient management and triage.

Date						
Time						
Systolic BP						
<80	3					
80-89	2					
90-139	0					
140-149	1					
150-159	2					
>160	3					
Respiratory Rate						
<10	3					
10-17	0					
18-24	1					
25-30	2					
>30	3					
Heart Rate						
<60	3					
60-110	0					
111-150	2					
>150	3					
Fio_2 to keep sat.>96%						
Room Air	0					
24%-39%	1					
>40%	3					
Temperature °C						
<34.0	3					
34.1-35.0	1					
35.1-37.9	0					
38.0-38.9	1					
>39.0	3					
Conciousness						
Alert (GCS=15)	0					
Not Alert (GCS<15)	3					

Fig. 51.1 Warning score chart (Reference from Carle et al. [34]). A score ≥6 should trigger a call for support from intensive care unit or rapid response team and initiation of continuous monitoring of vital signs. *BP* blood pressure, *FiO_2* fraction of inspired O_2, *GCS* Glasgow Coma Scale Score, *Sat* saturation

51.5.2.1 Management of the Unstable Pregnant Patient

Rapid response to instability in the pregnant patient is essential for the prevention of cardiac arrest. Recommended measures [35] for the management of unstable patients are as follows: (1) Maternal hemodynamic must be optimized by placing patient in full left lateral decubitus position to relieve aortocaval compression (*Class I; Level of Evidence C)*. (2) Intravenous access must be established, preferably above the diaphragm (*Class I; Level of Evidence C)*. (3) Hypoxia must be treated by giving 100% O_2 by facemask (*Class I; Level of Evidence C*). Class of recommendations and level of evidence to clinical strategies, interventions, and treatment are shown in Table 51.2. It is recommended that response teams for the confined pregnant patient on an obstetrical unit consist of four persons minimum, which includes a critical care/emergency physician, obstetrician, anesthesiologist, and neonatologist, in addition to appropriate nursing and technical support.

51.5.3 Cardiac Arrest Management

51.5.3.1 Basic Life Support (BLS)

2015 Guidelines Update for adult CPR recommend that single rescuer should initiate chest compressions before giving rescue breaths (C-A-B rather than A-B-C) [36–38] to reduce

Table 51.2 Applying class of recommendations and level of evidence to clinical strategies, interventions, and treatment

LEVEL OF EVIDENCE (LOE)	CLASS I Benefit >>>Risk	CLASS II a Benefit >>Risk	CLASS II b Benefit ≥ Risk	CLASS III Benefit <<Harm
	Procedure/treatment **SHOULD BE** performed/ administered	**REASONABLE** to perform procedure/treatment	Procedure/treatment **MAY BE CONSIDERED**	**CLASS III—No benefit** **Procedure Test**: Not helpful **Treatment**: No proven benefit **CLASS III—Harm** **Procedure Test**: Excessive cost w/o benefit or harmful **Treatment:** Harmful to patient
	Recommendation			
LEVEL A Multiple population evaluated. Data derived from multiple randomized clinical trials or meta-analysis	Procedure/treatment is **useful/effective** CLASS I LOE A CLASS I LOE A	Favour of procedure/ treatment being **useful/effective** **CLASS IIa LOE A**	Usefulness/efficacy less well established **CLASS IIb LOE A**	Procedure or treatment is not useful/effective and may be harmful **CLASS III LOE A**
LEVEL B Limited population evaluated. Data derived from single randomized trial or nonrandomized studies	Procedure/treatment is useful/effective CLASS I LOE B	Favour of procedure/ treatment being useful/effective **CLASS IIa LOE B**	Usefulness/efficacy less well established **CLASS IIb LOE B**	Procedure or treatment is not useful/effective and may be harmful **CLASS III LOE B**
LEVEL C Very limited population evaluated. Only consensus opinion of experts, case studies or standard of care	Procedure/treatment is useful/effective CLASS I LOE C	Favour of procedure/ treatment being useful/effective **CLASS IIa LOE C**	Usefulness/efficacy less well established **CLASS IIb LOE C**	Procedure or treatment is not useful/effective and may be harmful **CLASS III LOE C**

delay to first compression. The single rescuer should begin CPR with 30 chest compressions followed by 2 breaths [39]. High-quality CPR constitutes five basic components, i.e., compressing the chest at an adequate rate and depth, allowing complete chest recoil after each compression, minimizing interruptions in compressions, and avoiding excessive ventilation. The recommended chest compression rate and depth are between 100 to 120/min and 2″–2.4″ (5–6 cm), respectively.

The *key BLS components for adults, children, and infants* and *BLS healthcare provider, Adult Cardiac Arrest Algorithm—2015 Update* are shown in Table 51.3 and Fig. 51.2.

51.5.4 Basic Life Support in Pregnancy

Chest compressions for the pregnant patient are the same, as the most current recommendations for adult resuscitation, so the resuscitation of a pregnant patient having the uterus below umbilicus level remains the same as in normal adult patient. Resuscitation of a pregnant patient having uterus at or above the level of umbilicus needs certain modifications to improve the circulation, as the maternal physiology is different from normal adult. *The cardiac arrest in pregnancy in-hospital BLS algorithm* should be used as a guide to management (Fig. 51.3)

Table 51.3 Summary of key BLS components for adults, children, and infants

Components	Adult and adolescent	Children (Age 1 year to puberty)	Infants (28 days to 1 year)
Scene safety	Make sure the environment is safe for rescuer and victim		
Recognition of cardiac arrest	• Check for responsiveness • No breathing or no normal breathing (gasping only) • No pulse felt within 10 s • Breathing and pulse check can be performed simultaneously in less than 10 seconds (HCP only)		
Activation of emergency response system	*Single rescuer with no mobile device*: Leave the victim, and activate ERS, and get AED before staring CPR. Otherwise send someone, and start CPR immediately	*Witnessed collapse* • Follow steps for adults and adolescent on the left *Unwitnessed collapse* • Give 2 min of CPR • Leave victim to activate ERS and get AED • Return to the child and infant, and resume CPR; use the AED as soon as available	
Compression ventilation ratio without advanced airway	**1 or 2 rescuers:** 30:2	**1 rescuer :** 30:2 **2 or more rescuers:** 15:2	
Compression ventilation ration with advanced airway	Continuous compressions @ 100–120/min Give 1 breath every 6 s (10 breaths /min)		
Compression rate	100–120/min		
Compression depth	At least 2 in. (5 cm) No >2.4 in. (6 cm)	At least 1/3 AP diameter of chest About 2 in. (5 cm)	At least 1/3 AP diameter about 1 $^{1}/_{2}$ in. (4 cm)
Hand placement	2 hands on lower half of sternum	2 hands or 1 hand (optional for small child) on lower half of the sternum	**1 rescuer** 2 fingers in the center of the chest just below the nipple line **2 or more rescuers** 2 thump-encircling hands in the center of the chest, just below the nipple line
Chest recoil	Allow full chest recoil after each compression; do not lean on the chest after each compression		
Minimizing interruptions	Limit interruptions in chest compressions to less than 10 s		

51.5.4.1 First Responders

Nurses are often first responders in cardiac arrest; however, any hospital staff may witness or discover a patient in arrest and should be able to begin basic emergency care [40]. Rapid mobilization of expert resuscitation teams and BLS performed competently until the arrival of these teams give woman the best chance for return of spontaneous circulation (ROSC). First responder should initiate the usual resuscitation measures simultaneously, including placement of the backboard and provision of chest compressions, and appropriate airway management, defibrillation when appropriate, and manual left uterine displacement (LUD). To accomplish all tasks effectively, a minimum of four BLS responders should be present.

51.5.5 BLS Modifications

51.5.5.1 Adult Chest Compression Science

As with all adult resuscitations, high-quality chest compressions are essential to maximize the patient's chance of survival. For high-quality chest compressions, the patient must be supine on hard surface (*Class I; Level of Evidence C*). Chest compressions must be performed at least 100 per minute but not exceeding 120 per minute,

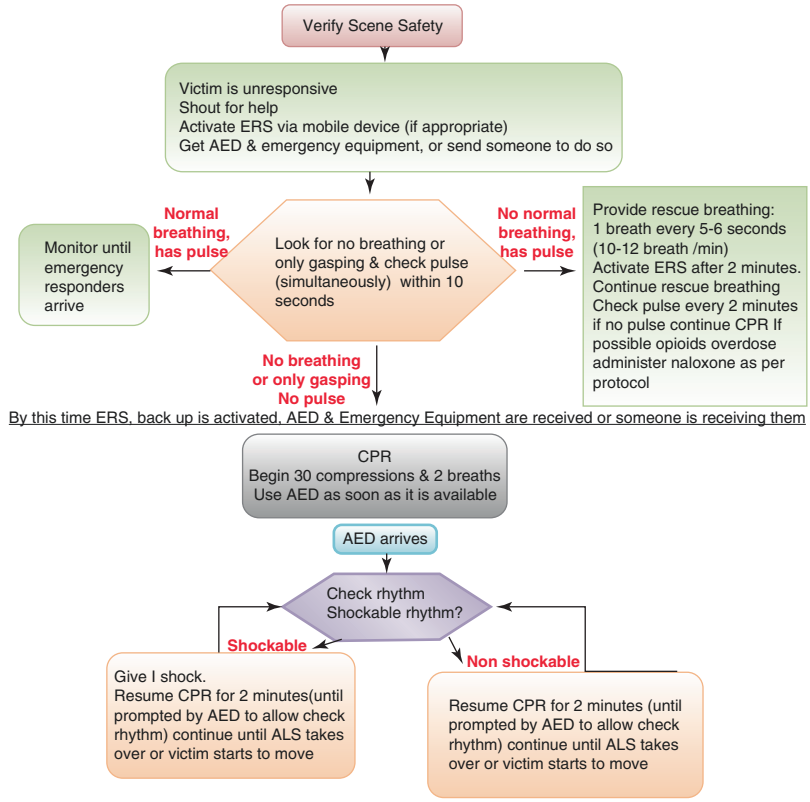


Fig. 51.2 Healthcare provider Adult Cardiac Arrest Algorithm—2015 Update (*Reference from 2015 AHA guidelines update for CPR & ECC*)

at a depth of at least 2 in (5 cm) but not exceeding 2.4 in. (6 cm), allowing full chest recoil before the next chest compression, with minimum interruptions, and at a compression/ventilation ratio of 30:2 [39] (*Class IIa; Level of Evidence C*). Interruptions should be minimized and limited to 10 s only except for specific interventions such as insertion of advanced airways or use of a defibrillator [39] (*Class IIa; Level of Evidence C)* because hospital beds are typically not firm and some of the force intended to compress the chest results in mattress displacement rather than chest compression.

51.5.6 Factors Affecting Chest Compressions in Pregnant Woman

51.5.6.1 Relief of Aortocaval Compression (LUD vs. LLT)

In the pregnant patient, supine positioning will result in aortocaval compression. Relief of aortocaval compression must be maintained continuously during resuscitative efforts and continued throughout postarrest care.

During resuscitation, manual LUD, either with one hand (Fig. 51.3a) or two hands (Fig. 51.3b), should be used to relieve aortocaval compression *(Class I; Level of Evidence C).* Previously, left lateral tilt had been the preferred option to relieve aortocaval compression during resuscitation.

Rees and Willis [41] found certain *disadvantages*, at >30° left lateral tilt: (1) The manikin slid off the inclined plane at >30° left lateral tilt. (2) Chest compression force was reduced as the angle of inclination was increased. (3) Inferior vena cava compression can still occur [42]. (4) In addition, the heart has been shown to shift laterally during tilt as compared with the supine position. Therefore, chest compressions performed with the patient in a tilt could be significantly less effective than those performed with the patient in the usual supine position, and this could have a

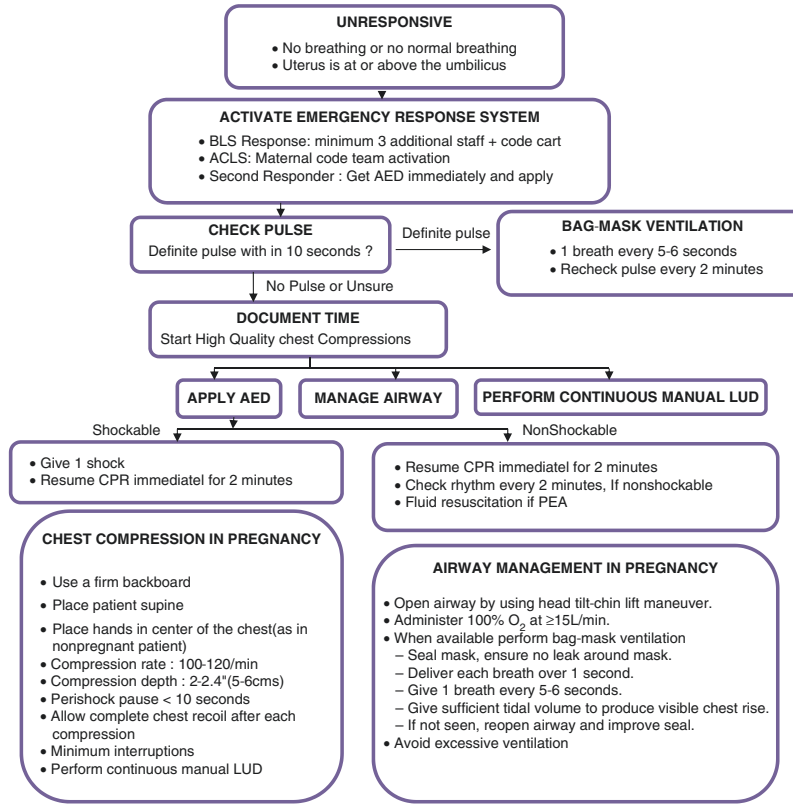


Fig. 51.3 Cardiac arrest in pregnancy in-hospital basic life support (BLS) algorithm: simultaneous C-A-B-U (chest compressions-airway-breathing-uterine displacement). *AED* automated external defibrillator, *CPR* cardiopulmonary resuscitation, *PEA* pulseless electrical activity (Reference from AHA Scientific statement 2015)

major impact on the chance of successful resuscitation [41, 43].

On the other hand, manual LUD [11] has certain definite benefits over LLT. *Benefits of manual LUD* include the following: (1) Easier access for both airway management and defibrillation. (2) While manual LUD is performed, the patient can remain supine and receive usual resuscitative measures, including high-quality chest compressions without hindrance (Fig. 51.4). (3) Manual LUD can be performed from the left of the patient, where the uterus is cupped and lifted up and leftward off the maternal vessels, or from the right of the patient, where the uterus is pushed upward and leftward off the maternal vessels. The rescuer must be careful not to inadvertently push down, which would increase the amount of inferior vena cava compression and negatively affect maternal hemodynamics.

51.5.7 Positioning of Hands During Chest Compressions

The rescuer should place the heel of one hand on the center (middle) of the victim's chest (lower half of the sternum) and the heel of the other hand on top of the first so that the hands overlap and are parallel *(Class IIa; Level of Evidence C)*.

51.6 Perimortem Cesarean Delivery (PMCD)

PMCD may be defined as the delivery of fetus when there is maternal cardiac arrest and is most commonly undertaken during resuscitation. Cesarean delivery is the mode of birth of fetus except in some rare cases where vaginal delivery may also be undertaken. ACLS and PMCD algo-

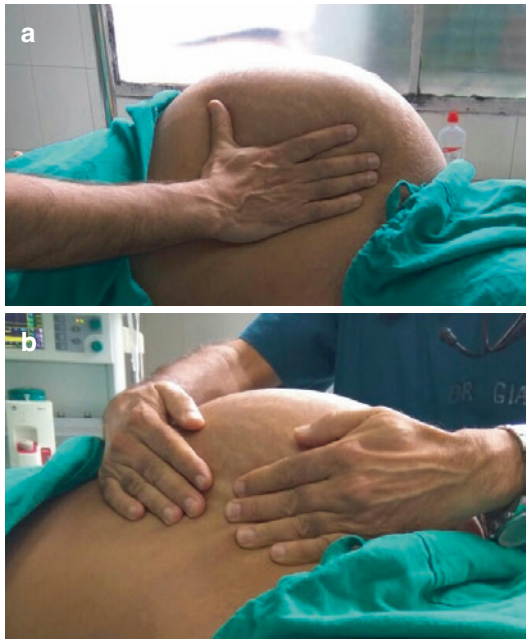


Fig. 51.4 (**a**) Manual left uterine displacement (LUD) by one-handed technique from right of the patient during adult resuscitation. (**b**) Manual left uterine displacement by two-handed technique from left of the patient

rithm for in-hospital cardiac arrest is shown in Fig. 51.5.

51.6.1 Why Perform PMCD in Cardiac Arrest?

A review of all published cases of PMCD up to 2010 showed that PMCD was an intervention which produced survival benefit in 19 of 60 cases (31.7%) of maternal cardiac arrests, and there were no cases in which PMCD may have been deleterious to maternal survival [14]. Several case reports of PMCD during a maternal cardiac arrest resulted in either hemodynamic improvement or even brought about spontaneous circulation, that is, only after the uterus had been emptied [44–54]. In a case series where 38 PMCD cases were studies, 12 out of 20 women for whom maternal outcome was recorded had ROSC immediately after delivery [44]. The critical point to emphasize and always practice is that both mother and infant may die if the pro-

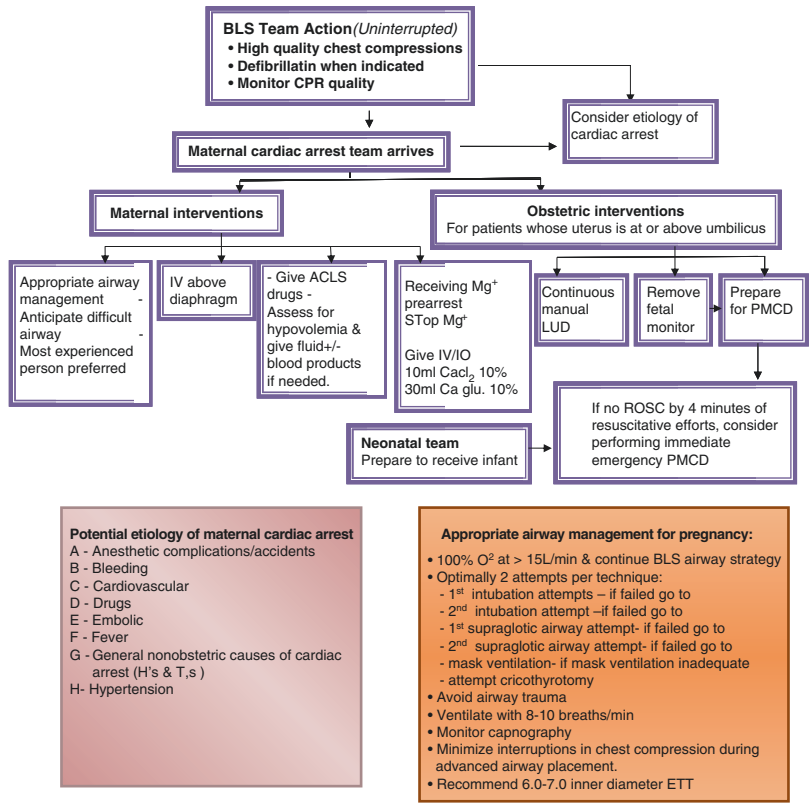


Fig. 51.5 Cardiac arrest in pregnancy in-hospital advanced cardiovascular life support (ACLS) algorithm. *BLS* indicates basic life support, *CPR* cardiopulmonary resuscitation, *ETT* endotracheal tube, *IV* intravenous, *IO* intraosseous, *LUD* left uterine displacement, and *ROSC* return of spontaneous circulation (*PMCD* perimortem cesarean delivery) (Reference from AHA Scientific statement 2015)

vider cannot restore blood flow to the mother's heart. So, the purpose of timely perimortem delivery is twofold. First the facilitation of resuscitation where cardiac output has not yet been established, by relieving aortocaval compression by emptying the gravid uterus significantly improves the resuscitative efforts. Second and of critical importance, early delivery of baby, the second patient, is done with a decreased risk of hypoxic neurological sequelae which may be permanent.

Literature about PMCD cases of cardiac arrest occurring before the third trimester concluded that if the fundus is felt above the level of the umbilicus, aortocaval compression may lead to resuscitation failure, and PMCD should be undertaken regardless of period of gestation [31].

51.6.2 The Importance of Timing with PMCD

The 5-min limit that the resuscitators have to establish that whether cardiac arrest can be reversed by BLS and ACLS was first brought to the medical literature in 1986 and has been regularly added in specialty guidelines [44, 55]. It was recommended that PMCD should begin at 4 min and should ensure delivery at 5 min after failed resuscitative efforts. The time interval was chosen to minimize the risks of neurological damage, which begin to develop after 4–6 min of anoxic cardiac arrest if there is no ROSC [56]. The rescue team is not required to wait 5 min before initiating PMCD, and there are circumstances that support a start even before the 4th min of resuscitation [30]. For example, in an obvious fatal scenario [40] in which maternal prognosis is grave and resuscitative efforts appear almost to be a failure, moving directly to PMCD may be appropriate, especially if the fetus is viable. This time frame is ideal and best, though studies have shown benefits of PMCD even when it is delayed for 10-even 30 min [56].

51.6.3 PMCD Technique

The procedure should be performed at the site of resuscitation, and time should not be wasted for moving the patient to operation theater, as transport increases the time to PMCD [57]. Also time should not be wasted waiting for surgical equipment or abdominal preparation. If available, antiseptic solution should be poured on the maternal abdomen. The only equipment required to start a PMCD is a scalpel, nothing else. Resuscitative efforts should be continued during the procedure, including manual LUD. Physician can decide the technique to perform PMCD, as both vertical incision and the Pfannenstiel incision are acceptable, although vertical incision is easier and faster and also provides better access for direct cardiac compression.

During PMCD, the fetus is given to neonatal team after delivery and placenta is delivered. The uterus should be closed with running locking stitch of absorbable suture, and the abdomen is closed in the regular fashion.

Recommendations for PMCD

1. Resuscitation team leader should activate the protocol for a PMCD as soon as cardiac arrest is identified in pregnant woman *(Class I; Level of Evidence C).*
2. During cardiac arrest, if the pregnant woman (with the uterus felt at or above the umbilicus) has not achieved ROSC with usual resuscitation measures with manual uterine displacement, it is recommended to prepare to evacuate the uterus, while resuscitation continues *(Class I; Level of Evidence C).*
3. Decision on the optimal timing of a PMCD for both mother and infant is complex and requires consideration of factors such as the cause of the arrest, maternal pathology and cardiac function, fetal gestational age, and some technical practical aspects (i.e., may be delayed until qualified staff is available to perform the procedure). Shorter *arrest-to-delivery* time is associated with better outcome (*Class I; Level of Evidence C*).

4. PMCD should be strongly considered for every mother in whom ROSC has not been achieved even after 4 min of resuscitative efforts (*Class IIa; Level of Evidence C*).

When PMCD is performed on in-hospital patient, the following are recommended:

1. Woman should not be transported to an operating room (*Class IIa; Level of Evidence C*).
2. Team should not wait for surgical equipments to begin the procedure; only scalpel is required (*Class IIa; Level of Evidence C*).
3. Team should not spend time on lengthy antiseptic procedures; just pour the antiseptic solution, or eliminate the step entirely (*Class IIa; Level of Evidence C*).
4. Continuous manual LUD should be performed throughout the PMCD until fetus is delivered (*Class IIa; Level of Evidence C*).

51.6.4 Site of PMCD

Simulation of chest compression on manikins has shown that the quality of CPR decreases during transport to the operating room [58]. Because immediate cesarean delivery may be the best way to optimize the condition of the mother and fetus, the operation should ideally be undertaken at the site of the arrest. A pregnant patient with in-hospital cardiac arrest should not be moved to operation theater or elsewhere for cesarean delivery. Management should occur at the site of the arrest (*Class I; Level of Evidence C*).

51.7 Defibrillation During Maternal Cardiac Arrest

Rapid judicious application of defibrillation in the setting of ventricular fibrillation or pulseless ventricular tachycardia is of utmost importance for the survival of the patient. The same currently recommended defibrillation guidelines as of adult resuscitation should be used in the pregnant patient. There is no modification of the recommended energy values of electric shock during pregnancy [35] (*Class I; Level of Evidence C)*. The patient may be defibrillated using biphasic shock energy of 120 to 200J (*Class I; Level of Evidence B*) with subsequent escalation of energy output if first shock is not effective and the device allows the option [59]. For in-hospital setting where staff may have problems in recognizing ECG rhythm or where defibrillation experience is rare, such as in an obstetric unit, automated external defibrillator may be a better option to be used during resuscitation [59] *(Class IIb; Level of Evidence C*). Anterolateral defibrillator pad placement is recommended to be used in a pregnant patient as a reasonable default (*Class IIa; Level of Evidence C*).The lateral pad/paddle should avoid the breast fat and be placed under it; this is an important consideration in the pregnant patient.

51.8 Airway and Breathing

Hypoxemia develops more rapidly in the pregnant compared with the nonpregnant patient; therefore rapid, high-quality, and effective airway and breathing interventions are essential. Early bag-mask ventilation with 100% oxygen is recommended [35]. Airway management is to be considered more difficult in the pregnant patient; therefore appropriate airway algorithm for pregnancy should be instituted. For first responder with minimal airway experience, bag-mask ventilation with 100% oxygen is most rapid noninvasive strategy to initiate ventilation [60]. An oral airway may help relieve obstruction in obesity, sleep apnea, and airway edema where difficulty in bag-mask ventilation is anticipated [61].

51.8.1 Vaginal Delivery During Maternal Cardiac Arrest

Only few published cases describe vaginal delivery during a cardiac arrest in pregnancy [62].

Obstetrician involved in an intrapartum cardiac arrest resuscitation may conduct a vaginal examination, provided that CPR is being efficiently performed by other resuscitators. If the cervix is found to be favorable and dilated fully and the fetal head is quite low, early, quick-assisted vaginal delivery can be considered (*Class IIa; Level of Evidence C*).

51.9 Advanced Cardiovascular Life Support

A quick and protocol-based well-coordinated action to maternal cardiac arrest is important. The ACLS resuscitators will continue BLS tasks on maternal cardiac arrest patients and perform advanced airway management and insert an intravenous cannula above the diaphragm which is used to give the usual ACLS drugs in usual adult doses when indicated. As soon as the obstetric and neonatal teams arrive on the scene, preparation for PMCD should begin. The ACLS algorithm includes PMCD as a treatment option in the maternal cardiac arrest scenario where ROSC is not achieved by 4 minutes after the onset of cardiac arrest; this is in patients with the uterus extending to or above the umbilicus. Along with this cause of the arrest needs to be considered and addressed as necessary. The cardiac arrest in pregnancy in-hospital ACLS algorithm should be used as a guide management (Fig. 51.4).

51.9.1 The Maternal Cardiac Resuscitators

To activate and achieve quick effective code team response is one of the most fundamental tasks to be completed during maternal cardiac arrest [35]. Each hospital must have a specific method to activate the maternal cardiac arrest team. There should be single call method to activate the maternal cardiac arrest team, notifying all members and ensuring that the resuscitators and the equipment are brought to the scene without delay (*Class 1; Level of Evidence C*).

The maternal cardiac arrest team would ideally be composed of the following [32] (*Class 1; Level of Evidence C*): (a) a critical care physician/emergency physician and nurses; (b) obstetrician and obstetric nurse; (c) anesthesia care provider, obstetric anesthesiologist or staff anesthesiologist and anesthesia assistant or certified nurse anesthetist; and (d) neonatology team, physician and nurse.

51.9.2 Special Equipment Required for a Maternal Cardiac Arrest

Special equipment is required for a maternal cardiac arrest. Specialized equipment should include a PMCD tray (Table 51.4) but at a minimum must include a scalpel. In addition, equipment for a difficult airway (Table 51.5) may be required for the mother. Neonatal resuscitation equipment will be required if the fetus is delivered and viable (Table 51.6).

51.9.3 Breathing and Airway Management During Maternal Cardiac Arrest

51.9.3.1 Management of Hypoxia

Hypoxia should always be considered as a cause of cardiac arrest, as oxygen reserves are lower and the metabolic demands are higher in pregnant patient compared with nonpregnant patient; thus, early ventilator support may be necessary (*Class 1; Level of Evidence C*). Furthermore, it should be kept in mind that cardiac arrest due to hypoxia (e.g., severe pneumonia, aspiration,

Table 51.4 Recommended equipment for perimortem cesarean delivery

Equipment contents of the emergency tray
• Scalpel with No. 10 blade
• Lower end of a Balfour retractor
• Pack of sponges
• 2 Kelly clamps
• Needle holder
• Forceps
• Sutures and suture scissors

Table 51.5 Recommended airway and breathing equipment

Equipment to be used first	Equipment to be used by experts
• Oxygen • Bag-valve-mask devices (e.g., Ambu Bag with disk valve as opposed to duckbill valve preferred) • Appropriate size face masks and oral airways • Stethoscope • Pulse oximeter • Qualitative carbon dioxide detector • Suction device	• Laryngoscope and assorted blades • Video laryngoscope • Cuffed tracheal tubes: size 6.0–7.0 mm inner diameter with a semirigid stylet and a range of backup sizes available • Gum elastic bougie • Airway exchange catheter • Supraglottic airways in a range of sizes • Flexible fiber-optic intubation equipment • Equipment suitable for emergency • Invasive airway access (e.g., cricothyrotomy) • Exhaled carbon dioxide detector

amniotic fluid embolism, acute pulmonary distress, opioids, high spinal block) requires early attention to airway and ventilation.

51.9.3.2 Airway Management

It is essential to be familiar with airway management algorithm in maternal cardiac arrest, given the high likelihood of difficult airway in the pregnant patient. Endotracheal intubation should be performed by an experienced laryngoscopist (*Class I; Level of Evidence C*) [63, 64]. Starting with an ETT with a 6.0–7.0 mm inner diameter is recommended (*Class I; Level of Evidence C*). Optimally no more than two laryngoscopy attempts should be made (*Class IIa; Level of Evidence C*) [65, 66]. Supraglotic airway placement is the preferred rescue strategy for failed intubation (*Class I; Level of Evidence C*) [67]. If attempts at airway management fail and bag and mask ventilation is not possible, current guide-

Table 51.6 Neonatal resuscitation equipments and drugs

Airway	Breathing	Circulation/drugs	Miscellaneous
Suction apparatus: Suction bulb Mechanical suction Suction catheters (6F–12F) Meconium aspirator ET intubation supplies: Laryngoscope (blade size: 0 for preterm and 1 for term infants) Extra bulbs and batteries ET tubes (internal diameters: 2.5, 3.0, 3.5, 4.0 mm) Stylets ET-securing device Carbon dioxide detector Tapes and scissors Laryngeal mask airway size 1 (for use when ET intubation is not feasible)	Positive-pressure ventilation: Device: T-piece resuscitator/ flow-inflating bag/self-inflating bag with oxygen reservoir (least preferred) Bag sizes 240–750 mL, fitted with pop-off valve Mask sizes 0 and 1 (cushioned preferred) Sources of compressed air and O2 O2 blender with a flowmeter (capacity up to 10 L/min) Plastic tubing	IV access: Sterile gloves Antiseptic solution Cord tie Scalpel Umbilical venous catheters (3.5F–5.0F) 3-Way stopcock Suture for securing 24-Gauge IV cannulas (for use if umbilical vessel access not feasible) IO needle (rarely needed) Drugs: Epinephrine 1:10 000 (0.1 mg/mL) Normal saline bags 10% dextrose bags 4.2% sodium bicarbonate (rarely indicated) Saline flushes	Temperature regulation: Heat source (radiant warmer preferred) Warm towels Thermometer (servo-controlled preferred) Clean plastic bag (for extremely premature neonates) Monitoring: Neonatal stethoscope Neonatal cardiac leads Pulse oximeter Neonatal/infant oximeter probes Clock (digital preferred) Other: Firm resuscitation surface (preferably height adjustable) Good light source Nasogastric tubes (6F–10 F) Sterile syringes (1, 3, 5, 20 mL) Clean, sterile gloves Sterile gauzes Bottles to collect blood samples suitable for low-volume blood testing

lines for emergency invasive airway access should be undertaken as per resuscitation protocol (i.e., call for assistance, call for equipment). Continuous waveform capnography is now universally recommended as the most reliable method of confirming correct placement of ETT and also to monitor henceforth (*Class I; Level of Evidence C*).

51.9.3.3 Drug Therapy During Cardiac Arrest

Administration of 1 mg epinephrine IV/IO every 3–5 min during adult cardiac arrest should be considered [68]. In view of the effects of vasopressin on the uterus and because both agents are considered equivalent [69, 70], epinephrine should be the preferred agent (*Class IIb; Level of Evidence C*).

All current ACLS drugs at recommended doses should be used in pregnant patient also without modifications (*Class IIb; Level of Evidence C*).

For refractory (shock-resistant) ventricular fibrillation and tachycardia, amiodarone 300 mg rapid infusion should be administered with 150mg doses repeated as needed (*Class IIb; Level of Evidence C*).

No medication should be withheld or reduced because of concerns about fetal teratogenicity [71] (*Class IIb; Level of Evidence C*).

Medication doses do not require alteration to accommodate the physiological changes of pregnancy, although there are changes in volume of distribution and clearance of medication during pregnancy (*Class IIb; Level of Evidence C*).

Use of steroids? A new concept under investigations, whether steroids in combination with vasopressors lead to improved survival in cardiac arrest or not? A randomized study of patients in whom cardiac arrest occurred when they were in hospital demonstrated that the combined vasopressin-epinephrine and methylprednisolone use during CPR and also use of subsequent stress-dose hydrocortisone in postresuscitation period of hypotension led to improved survival to hospital discharge compared with epinephrine alone [72]. Despite the promising results, additional studies are needed before recommendations can be made about combined vasopressors.

51.9.4 Fetal Assessment During Cardiac Arrest

Fetal assessment should not be performed during resuscitation, and fetal monitors should be removed or detached as soon as possible to facilitate PMCD without delay or hindrance (*Class I; Level of Evidence C*).

51.10 Neonatal Resuscitation

51.10.1 Neonatal Resuscitation Team

Team composition optimally should include a neonatologist/pediatrician, neonatal nurses, and respiratory therapists who should be familiar with the local neonatal resuscitation algorithms [73]. At least one member of the team must be skilled in emergency neonatal endotracheal intubation.

The neonatal resuscitation team should be notified of the impending delivery and its circumstances as

early as feasible to allow maximum preparatory time (*Class I; Level of Evidence C*).

The following critical information should be provided to the neonatal resuscitation team leader:

gestational age, number of fetuses, and mode of delivery (*Class I; Level of Evidence C*).

In the event of multiple pregnancies, it is recommended that each fetus be resuscitated by a separate

resuscitation team (*Class I; Level of Evidence C*).

51.10.2 Neonatal Resuscitation Equipment

PMCD may be performed outside the maternity unit and will require the team to perform resuscitation in a relatively unfamiliar environment that

may lack optimal equipment. Each hospital must have prestocked neonatal crash carts available, the locations of which should be clearly marked and known to the neonatal resuscitation team. Alternatively, neonatal resuscitation equipment can be prestocked in easy-to-carry bags that can be taken to the area of need by the resuscitation team on notification of impending delivery (Table 51.6).

51.10.3 Assessment of the Newborn

The majority of neonates delivered by PMCD are likely to require active resuscitation [14]; the severity of perinatal depression and extent of resuscitation may vary. Management of the neonate after PMCD should follow the most current AHA guidelines [73].

51.10.4 Neonatal Resuscitation: Key Issues and Major Changes in 2015 Update

The order of the three assessment questions has changed to (1) term question, (2) good tone, and (3) breathing or crying.

The golden minute (60-s) mark for completing the initial steps, reevaluating, and beginning ventilation for avoiding unnecessary delay in initiation of ventilation.

New recommendation that delayed cord clamping for longer than 30 s is reasonable for both term and preterm infants.

Temperature should be recorded and maintained between 36.5 and 37.5 °C.

Routine intubation for tracheal suction is no longer suggested.

Assessment of heart rate by using 3-lead ECG may be reasonable.

Resuscitation of preterm newborns of less than 35 weeks of gestation should be initiated with low oxygen (21–30%). Initial resuscitation with higher oxygen (65% or greater) is not recommended.

Chest compression technique: (2 thumb-encircling hands) and compression to ventilation ratio (3:1 with 90 compressions and 30 breaths per minute), rescuer may use higher ratio (e.g. 15:2) if arrest is believed to be of cardiac origin. One hundred percent oxygen use during CPR is reasonable.

51.11 Immediate Postarrest Care

It is essential that multidisciplinary team continues care in the postarrest period. As with other postarrest patients, the pregnant patient who is successfully resuscitated will require thorough assessment, monitoring, and treatment as complications arise.

The 2015 AHA recommends the following:

1. If the patient is still pregnant, she should be placed in the full left lateral decubitus position, provided that this does not interfere with additional management issues such as monitoring, airway control, and intravenous access. If the patient is not in full left lateral tilt, manual LUD should be maintained continuously (*Class I; Level of Evidence C*).
2. The patient should be transferred to ICU unless an operation is required (*Class I; Level of Evidence C*).
3. Optimal pre-event planning should be ensured as discussed above (*Class I; Level of Evidence C*).
4. Multidisciplinary care must continue (*Class I; Level of Evidence C*).
5. The cause of the arrest should continue to be considered and treated accordingly (*Class I; Level of Evidence C*).

51.11.1 Postarrest Antiarrhythmic Therapy

Postarrest therapy for recurrent life-threatening arrhythmias includes consideration of an implantable cardioverter-defibrillator (ICD) or medical therapy in the pregnant patient as in the nonpregnant [74] (*Class I; Level of Evidence C*). β-Blockers are often used as first-line therapy for arrhythmias and are generally safe in pregnancy

(metoprolol is a preferred β-blocker used in pregnancy) (*Class IIa; Level of Evidence C*). Amiodarone should be considered first-line therapy in recurrent primary ventricular tachycardia and ventricular fibrillation (*Class IIa; Level of Evidence C*). Evaluation of reversible causes of arrhythmias should be routine. Thyroid dysfunction, adverse drug effects, electrolyte disturbances, cardiac ischemia, and heart failure should be identified and treated (*Class I; Level of Evidence C*).

51.11.2 Fetal Risk of Postresuscitation Intervention

Three major principles guide the decisions made by clinicians at this stage:

1. Maternal well-being is the overriding priority because maternal demise or unfavorable recovery never bodes well for the unborn baby.
2. Embryogenesis is mostly complete by 12 weeks of gestation; hence, even teratogenic drugs (e.g., warfarin, phenytoin, corticosteroids) are unlikely to cause malformations if the event occurs after the first trimester.
3. Drugs may cause fetal toxicity rather than teratogenicity in late pregnancy (e.g., angiotensin-converting enzyme inhibitors, which can cause fetal renal failure and oligohydramnios).

   The risks and benefits of medication use in the postarrest period should be considered on an individual basis.

51.11.3 Medicolegal Considerations

Implementation of a quality incident notification system has shown that such type of program can help identify avoidable adverse outcomes and can be used to improve practices of cardiopulmonary resuscitation [75–77].

All cases of cardiac arrest and maternal near miss should be reviewed by the maternal cardiac arrest committee for the hospital (*Class I; Level of Evidence C*). Identified deficiencies should be corrected (*Class I; Level of Evidence C*).

51.11.4 Knowledge Translation Strategy

Knowledge translation, also referred to as dissemination and implementation, has been defined by the National Center for the Dissemination of Disability Research as "the collaborative and systemic review, assessment and identification, aggregation, and practical application of high quality research by key stakeholders (consumers, researchers, practitioners, policy makers) for the purpose of improving the lives of individuals" [78]. This statement represents an important step in the knowledge translation process: the collaborative filtering of information by experts so that only the most valid and useful knowledge is left [79].

All stakeholders/specialties involved in maternal resuscitation should form maternal cardiac arrest committees within each institution to ensure guidelines implementation, training, and institution of mock code drills (*Class I; Level of Evidence C*).

51.11.5 Maternal Resuscitation Training

An analysis of simulated maternal cardiac arrest involving participants trained in ACLS suggests that performance during an actual event may be suboptimal [80]. The rarity of maternal cardiac arrest [7] implies that participants in CPR courses could benefit from review of obstetric specific interventions. Data from The Joint Commission suggests that communication failures are the root cause of neonatal morbidity and mortality in 70% of cases [81] that occur in obstetric domain. In addition, in an analysis of preventable maternal mortalities, facility factors, including lack of institutional preparedness, contributed significantly to the fatal outcome in 75% of cases [82]. So maternally oriented CPR courses are likely more relevant to the learning needs and goals of obstetric staff; these courses have been developed

by groups in the United Kingdom and the United States [45, 83, 84].

Recommendations:

1. Periodic multidisciplinary drills may help institutions optimize safety systems (*Class IIa; Level of Evidence C*).
2. Specific courses on maternal resuscitation should be available to staff if not available outside local institutions (*Class IIa; Level of Evidence C*).
3. The future goal should be to have national and international programs in maternal resuscitation (*Class I; Level of Evidence C*).

51.12 Conclusion

Maternal cardiac arrest is a complex clinical scenario as it involves multispecialties and complex care decisions. Although maternal cardiac arrest is rare, it appears to be increasing in frequency. The number of high-risk women undergoing pregnancy is on the rise, as is the rate of severe complications related to pregnancy (including cardiac arrest) [4].

The newly developed in-hospital and out-of-hospital BLS and ACLS algorithms should be the backbone of the response plan to a maternal cardiac arrest. Special attention should be paid to manual LUD, the difficult airway, and the appropriate use of PMCD. Lifesaving interventions such as defibrillation and medications should not be withheld in the setting of pregnancy.

Training, mock code drills, and review of cases should become routine in teaching programs of BLS and ACLS in maternal cardiac arrest. A maternal cardiac arrest committee must be formed at every institution, and emergency response plans specific to each institution must be developed and implemented.

References

1. Mhyre JM, Tsen LC, Einav S, Kuklina EV, Leffert LR, Bateman BT. Cardiac arrest during hospitalization for delivery in the United States, 1998–2011. Anesthesiology. 2014;120:810–8. https://doi.org/10.1097/ALN.0000000000000159.
2. Cantwell R, Clutton-Brock T, Cooper G, Dawson A, Drife J, Garrod D, Harper A, Hulbert D, Lucas S, McClure J, Millward-Sadler H, Neilson J, Nelson-Piercy C, Norman J, O'Herlihy C, Oates M, Shakespeare J, de Swiet M, Williamson C, Beale V, Knight M, Lennox C, Miller A, Parmar D, Rogers J, Springett A. Saving mothers' lives: reviewing maternal deaths to make motherhood safer: 2006–2008. The Eighth Report of the Confidential Enquiries into Maternal Deaths in the United Kingdom. BJOG. 2011:118(suppl 1):1–203.
3. Centers for Disease Control and Prevention. Pregnancy mortality surveillance system. 2014. http://www.cdc.gov/reproductivehealth/maternalinfanthealth/pmss.html. Accessed 12 May 2014.
4. Kuklina E, Callaghan W. Chronic heart disease and severe obstetric morbidity among hospitalisations for pregnancy in the USA: 1995–2006. BJOG. 2011;118:345–52. https://doi.org/10.1111/j.1471-0528.2010.02743.x.
5. Cohen SE, Andes LC, Carvalho B. Assessment of knowledge regarding cardiopulmonary resuscitation of pregnant women. Int J Obstet Anesth. 2008;17:20–5. https://doi.org/10.1016/j.ijoa.2007.10.002.
6. Einav S, Matot I, Berkenstadt H, Bromiker R, Weiniger CF. A survey of labour ward clinicians' knowledge of maternal cardiac arrest and resuscitation. Int J Obstet Anesth. 2008;17:238–42. https://doi.org/10.1016/j.ijoa.2008.01.015.
7. Lewis G, editor. The Confidential Enquiry into Maternal and Child Health (CEMACH): saving mothers' lives: reviewing maternal deaths to make motherhood safer 2003–2005: The Seventh Confidential Enquiry Into Maternal Deaths in the United Kingdom. London: CEMACH; 2007.
8. Seethala RR, Esposito EC, Abella BS. Approaches to improving cardiac arrest resuscitation performance. Curr Opin Crit Care. 2010;16:196–202.
9. Lipman SS, Daniels KI, Carvalho B, Arafeh J, Harney K, Puck A, et al. Defi cits in the provision of cardiopulmonary resuscitation during simulated obstetric crises. Am J Obstet Gynecol. 2010;203:179.e1–5.
10. Fisher N, Eisen LA, Bayya JV, Dulu A, Bernstein PS, Merkatz IR, et al. Improved performance of maternal-fetal medicine staff after maternal cardiac arrest simulation-based training. Am J Obstet Gynecol. 2011;205:239.e1–5.
11. Kundra P, Khanna S, Habeebullah S, Ravishankar M. Manual displacement of the uterus during Caesarean section. Anaesthesia. 2007;62:460–5. https://doi.org/10.1111/j.1365-2044.2007.05025.x.
12. Kim S, You JS, Lee HS, Lee JH, Park YS, Chung SP, et al. Quality of chest compressions performed by inexperienced rescuers in simulated cardiac arrest associated with pregnancy. Resuscitation. 2013;84:98–102.

13. Jeejeebhoy FM, Zelop CM, Windrim R, Carvalho JC, Dorian P, Morrison LJ. Management of cardiac arrest in pregnancy: a systematic review. Resuscitation. 2011;82:801–9.
14. Einav S, Kaufman N, Sela HY. Maternal cardiac arrest and perimortem caesarean delivery: evidence or expert-based? Resuscitation. 2012;83:1191–200. https://doi.org/10.1016/j.resuscitation.2012.05.005.
15. Creanga AA, Berg CJ, Ko JY, Farr SL, Tong VT, Bruce FC, Callaghan WM. Maternal mortality and morbidity in the United States: where are we now? J Womens Health (Larchmt). 2014;23:3–9. https://doi.org/10.1089/jwh.2013.4617.
16. Tan EK, Tan EL. Alterations in physiology and anatomy during pregnancy. Best Pract Res Clin Obstet Gynaecol. 2013;27:791–802. https://doi.org/10.1016/j.bpobgyn.2013.08.001.
17. San-Frutos L, Engels V, Zapardiel I, Perez-Medina T, Almagro-Martinez J, Fernandez R, Bajo-Arenas JM. Hemodynamic changes during pregnancy and postpartum: a prospective study using thoracic electrical bioimpedance. J Matern Fetal Neonatal Med. 2011;24:1333–40. https://doi.org/10.3109/14767058.2011.556203.
18. Carbillon L, Uzan M, Uzan S. Pregnancy, vascular tone, and maternal hemodynamics: a crucial adaptation. Obstet Gynecol Surv. 2000;55:574–81.
19. McLennan C, Minn M. Antecubital and femoral venous pressure in normal and toxemic pregnancy. Am J Obstet Gynecol. 1943;45:568–91.
20. Ueland K, Novy MJ, Peterson EN, Metcalfe J. Maternal cardiovascular dynamics, IV: the influence of gestational age on the maternal cardiovascular response to posture and exercise. Am J Obstet Gynecol. 1969;104:856–64.
21. Rossi A, Cornette J, Johnson MR, Karamermer Y, Springeling T, Opic P, Moelker A, Krestin GP, Steegers E, Roos-Hesselink J, van Geuns RJ. Quantitative cardiovascular magnetic resonance in pregnant women: cross-sectional analysis of physiological parameters throughout pregnancy and the impact of the supine position. J Cardiovasc Magn Reson. 2011;13:31. https://doi.org/10.1186/1532-429X-13-31.
22. Palmer SK, Zamudio S, Coffin C, Parker S, Stamm E, Moore LG. Quantitative estimation of human uterine artery blood flow and pelvic blood flow redistribution in pregnancy. Obstet Gynecol. 1992;80:1000–6.
23. Contreras G, Gutiérrez M, Beroíza T, Fantín A, Oddó H, Villarroel L, Cruz E, Lisboa C. Ventilatory drive and respiratory muscle function in pregnancy. Am Rev Respir Dis. 1991;144:837–41. https://doi.org/10.1164/ajrccm/144.4.837.
24. Lucius H, Gahlenbeck H, Kleine HO, Fabel H, Bartels H. Respiratory functions, buffer system, and electrolyte concentrations of blood during human pregnancy. Respir Physiol. 1970;9:311–7.
25. Pernoll ML, Metcalfe J, Schlenker TL, Welch JE, Matsumoto JA. Oxygen consumption at rest and during exercise in pregnancy. Respir Physiol. 1975;25:285–93.
26. Archer GW Jr, Marx GF. Arterial oxygen tension during apnoea in parturient women. Br J Anaesth. 1974;46:358–60.
27. Adutaya A, Hladunewich M. Obstetric nephrology: renal hemodynamic and metabolic physiology in normal pregnancy. Clin J Am Soc Nephrol. 2012;7:2073–80.
28. Lawson M, Kern F Jr, Everson GT. Gastrointestinal transit time in human pregnancy: prolongation in the second and third trimesters followed by postpartum normalization. Gastroenterology. 1985;89:996–9.
29. Chiloiro M, Darconza G, Piccioli E, De Carne M, Clemente C, Riezzo G. Gastric emptying and orocecal transit time in pregnancy. J Gastroenterol. 2001;36:538–43.
30. Stallard TC, Burns B. Emergency delivery and perimortem C-section. Emerg Med Clin North Am. 2003;21:679–93.
31. Svinos H. Towards evidence based emergency medicine: best BETs from the Manchester Royal Infirmary, BET 1: emergency caesarean section in cardiac arrest before the third trimester. Emerg Med J. 2008;25:764–5. https://doi.org/10.1136/emj.2008.066860.
32. Hui D, Morrison LJ, Windrim R, Lausman AY, Hawryluck L, Dorian P, Lapinsky SE, Halpern SH, Campbell DM, Hawkins P, Wax RS, Carvalho JC, Dainty KN, Maxwell C, Jeejeebhoy FM. The American Heart Association 2010 guidelines for the management of cardiac arrest in pregnancy: consensus recommendations on implementation strategies. J Obstet Gynaecol Can. 2011;33:858–63.
33. Cantwell R, Clutton-Brock T, Cooper G, Dawson A, Drife J, Garrod D, Harper A, Hulbert D, Lucas S, McClure J, Millward-Sadler H, Neilson J, Nelson-Piercy C, Norman J, O'Herlihy C, Oates M, Shakespeare J, de Swiet M, Williamson C, Beale V, Knight M, Lennox C, Miller A, Parmar D, Rogers J, Springett A. Saving mothers' lives: reviewing maternal deaths to make motherhood safer: 2006–2008: the Eighth Report of the Confidential Enquiries Into Maternal Deaths in the United Kingdom [published correction appears in *BJOG*. 2014;122:e1]. BJOG. 2011;118(suppl 1):1–203.
34. Carle C, Alexander P, Columb M, Johal J. Design and internal validation of an obstetric early warning score: secondary analysis of the Intensive Care National Audit and Research Centre Case Mix Programme database. Anaesthesia. 2013;68:354–67. https://doi.org/10.1111/anae.12180.
35. Vanden Hoek TL, Morrison LJ, Shuster M, Donnino M, Sinz E, Lavonas EJ, Jeejeebhoy FM, Gabrielli A. Part 12: cardiac arrest in special situations: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care [published correction appears in *Circulation*. 2011;123:e239 and *Circulation*. 2011;124:e405]. Circulation. 2010;122(suppl 3):S829–61. https://doi.org/10.1161/CIRCULATIONAHA.110.971069.
36. Lubrano R, Cecchetti C, Bellelli E, Gentile I, Loayza Levano H, Orsini F, Bertazzoni G, Messi

G, Rugolotto S, Pirozzi N, Elli M. Comparison of times of intervention during pediatric CPR maneuvers using ABC and CAB sequences: a randomized trial. Resuscitation. 2012;83:1473–7. https://doi.org/10.1016/j.resuscitation.2012.04.011.
37. Sekiguchi H, Kondo Y, Kukita I. Verification of changes in the time taken to initiate chest compressions according to modified basic life support guidelines. Am J Emerg Med. 2013;31:1248–50. https://doi.org/10.1016/j.ajem.2013.02.047.
38. Marsch S, Tschan F, Semmer NK, Zobrist R, Hunziker PR, Hunziker S. ABC versus CAB for cardiopulmonary resuscitation: a prospective, randomized simulator-based trial. Swiss Med Wkly. 2013;143:w13856. https://doi.org/10.4414/smw.2013.13856.
39. Berg RA, Hemphill R, Abella BS, Aufderheide TP, Cave DM, Hazinski MF, Lerner EB, Rea TD, Sayre MR, Swor RA. Part 5: adult basic life support: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care [published correction appears in *Circulation*. 2011;124:e402]. Circulation. 2010;122(suppl 3):S685–705. https://doi.org/10.1161/CIRCULATIONAHA.110.970939.
40. Einav S, Shleifer A, Kark JD, Landesberg G, Matot I. Performance of department staff in the window between discovery of collapse to cardiac arrest team arrival. Resuscitation. 2006;69:213–20. https://doi.org/10.1016/j.resuscitation.2005.09.015.
41. Rees GA, Willis BA. Resuscitation in late pregnancy. Anaesthesia. 1988;43:347–9.
42. Archer TL, Suresh P, Shapiro AE. Cardiac output measurement, by means of electrical velocimetry, may be able to determine optimum maternal position during gestation, labour and caesarean delivery, by preventing vena caval compression and maximising cardiac output and placental perfusion pressure. Anaesth Intensive Care. 2011;39:308–11.
43. Yun JG, Lee BK. Spatial relationship of the left ventricle in the supine position and the left lateral tilt position (implication for cardiopulmonary resuscitation in pregnant patients). Fire Sci Eng. 2013;27:75–9.
44. Katz V, Balderston K, DeFreest M. Perimortem cesarean delivery: were our assumptions correct? Am J Obstet Gynecol. 2005;192:1916–20.
45. Dijkman A, Huisman CM, Smit M, Schutte JM, Zwart JJ, van Roosmalen JJ, Oepkes D. Cardiac arrest in pregnancy: increasing use of perimortem caesarean section due to emergency skills training? BJOG. 2010;117:282–7. https://doi.org/10.1111/j.1471-0528.2009.02461.x.
46. Page-Rodriguez A, Gonzalez-Sanchez JA. Perimortem cesarean section of twin pregnancy: case report and review of the literature. Acad Emerg Med. 1999;6:1072–4.
47. Cardosi RJ, Porter KB. Cesarean delivery of twins during maternal cardiopulmonary arrest. Obstet Gynecol. 1998;92(pt 2):695–7.
48. McDonnell NJ. Cardiopulmonary arrest in pregnancy: two case reports of successful outcomes in association with perimortem caesarean delivery. Br J Anaesth. 2009;103:406–9. https://doi.org/10.1093/bja/aep176.
49. Stehr SN, Liebich I, Kamin G, Koch T, Litz RJ. Closing the gap between decision and delivery: amniotic fluid embolism with severe cardiopulmonary and haemostatic complications with a good outcome. Resuscitation. 2007;74:377–81. https://doi.org/10.1016/j.resuscitation.2007.01.007.
50. McCartney CJ, Dark A. Caesarean delivery during cardiac arrest in late pregnancy. Anaesthesia. 1998;53:310–1.
51. Lurie S, Mamet Y. Caesarean delivery during maternal cardiopulmonary resuscitation for status asthmaticus. Emerg Med J. 2003;20:296–7.
52. O'Connor RL, Sevarino FB. Cardiopulmonary arrest in the pregnant patient: a report of a successful resuscitation. J Clin Anesth. 1994;6:66–8.
53. Finegold H, Darwich A, Romeo R, Vallejo M, Ramanathan S. Successful resuscitation after maternal cardiac arrest by immediate cesarean section in the labor room. Anesthesiology. 2002;96:1278.
54. Parker J, Balis N, Chester S, Adey D. Cardiopulmonary arrest in pregnancy: successful resuscitation of mother and infant following immediate caesarean section in labour ward. Aust N Z J Obstet Gynaecol. 1996;36:207–10.
55. Lipman S, Cohen S, Einav S, Jeejeebhoy F, Mhyre JM, Morrison LJ, Katz V, Tsen LC, Daniels K, Halamek LP, Suresh MS, Arafeh J, Gauthier D, Carvalho JC, Druzin M, Carvalho B. Society for obstetric Anesthesia and Perinatology. The Society for Obstetric Anesthesia and Perinatology consensus statement on the management of cardiac arrest in pregnancy. Anesth Analg. 2014;118:1003–16. https://doi.org/10.1213/ANE.0000000000000171.
56. Katz VL, Dotters DJ, Droegemueller W. Perimortem cesarean delivery. Obstet Gynecol. 1986;68:571–6.
57. Lipman S, Daniels K, Cohen SE, Carvalho B. Labor room setting compared with the operating room for simulated perimortem cesarean delivery: a randomized controlled trial. Obstet Gynecol. 2011;118:1090–4. https://doi.org/10.1097/AOG.0b013e3182319a08.
58. Lipman SS, Wong JY, Arafeh J, Cohen SE, Carvalho B. Transport decreases the quality of cardiopulmonary resuscitation during simulated maternal cardiac arrest. Anesth Analg. 2013;116:162–7. https://doi.org/10.1213/ANE.0b013e31826dd889.
59. Link MS, Atkins DL, Passman RS, Halperin HR, Samson RA, White RD, Cudnik MT, Berg MD, Kudenchuk PJ, Kerber RE. Part 6: electrical therapies: automated external defibrillators, defibrillation, cardioversion, and pacing: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care [published correction appears in *Circulation*. 2011;123:e235]. Circulation. 2010;122(suppl 3):S706–19. https://doi.org/10.1161/CIRCULATIONAHA.110.970954.

60. Gruber E, Oberhammer R, Balkenhol K, Strapazzon G, Procter E, Brugger H, Falk M, Paal P. Basic life support trained nurses ventilate more efficiently with laryngeal mask supreme than with facemask or laryngeal tube suction-disposable: a prospective, randomized clinical trial. Resuscitation. 2014;85:499–502. https://doi.org/10.1016/j.resuscitation.2014.01.004.
61. Kheterpal S, Han R, Tremper KK, Shanks A, Tait AR, O'Reilly M, Ludwig TA. Incidence and predictors of difficult and impossible mask ventilation. Anesthesiology. 2006;105:885–91.
62. Baghirzada L, Balki M. Maternal cardiac arrest in a tertiary care centre during 1989–2011: a case series. Can J Anaesth. 2013;60:1077–84. https://doi.org/10.1007/s12630-013-0021-9.
63. Quinn AC, Milne D, Columb M, Gorton H, Knight M. Failed tracheal intubation in obstetric anaesthesia: 2 yr national case-control study in the UK. Br J Anaesth. 2013;110:74–80. https://doi.org/10.1093/bja/aes320.
64. McDonnell NJ, Paech MJ, Clavisi OM, Scott KL. ANZCA Trials Group Difficult and failed intubation in obstetric anaesthesia: an observational study of airway management and complications associated with general anaesthesia for caesarean section. Int J Obstet Anesth. 2008;17:292–7. https://doi.org/10.1016/j.ijoa.2008.01.017.
65. Balki M, Cooke ME, Dunington S, Salman A, Goldszmidt E. Unanticipated difficult airway in obstetric patients: development of a new algorithm for formative assessment in high-fidelity simulation. Anesthesiology. 2012;117:883–97. https://doi.org/10.1097/ALN.0b013e31826903bd.
66. Mhyre JM, Healy D. The unanticipated difficult intubation in obstetrics. Anesth Analg. 2011;112:648–52. https://doi.org/10.1213/ANE.0b013e31820a91a6.
67. Apfelbaum JL, Hagberg CA, Caplan RA, Blitt CD, Connis RT, Nickinovich DG, Hagberg CA, Caplan RA, Benumof JL, Berry FA, Blitt CD, Bode RH, Cheney FW, Connis RT, Guidry OF, Nickinovich DG, Ovassapian A, American Society of Anesthesiologists Task Force on Management of the Difficult Airway. Practice guidelines for management of the difficult airway: an updated report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway. Anesthesiology. 2013;118:251–70. https://doi.org/10.1097/ALN.0b013e31827773b2.
68. Neumar RW, Otto CW, Link MS, Kronick SL, Shuster M, Callaway CW, Kudenchuk PJ, Ornato JP, McNally B, Silvers SM, Passman RS, White RD, Hess EP, Tang W, Davis D, Sinz E, Morrison LJ. Part 8: adult advanced cardiovascular life support: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care [published correction appears in *Circulation*. 2011;123:e236 and *Circulation*. 20113;128:e480]. Circulation. 2010;122(suppl 3):S729–67. https://doi.org/10.1161/CIRCULATIONAHA.110.970988.
69. Aung K, Htay T. Vasopressin for cardiac arrest: a systematic review and meta-analysis. Arch Intern Med. 2005;165:17–24. https://doi.org/10.1001/archinte.165.1.17.
70. Gueugniaud PY, David JS, Chanzy E, Hubert H, Dubien PY, Mauriaucourt P, Bragança C, Billères X, Clotteau-Lambert MP, Fuster P, Thiercelin D, Debaty G, Ricard-Hibon A, Roux P, Espesson C, Querellou E, Ducros L, Ecollan P, Halbout L, Savary D, Guillaumée F, Maupoint R, Capelle P, Bracq C, Dreyfus P, Nouguier P, Gache A, Meurisse C, Boulanger B, Lae C, Metzger J, Raphael V, Beruben A, Wenzel V, Guinhouya C, Vilhelm C, Marret E. Vasopressin and epinephrine vs. epinephrine alone in cardiopulmonary resuscitation. N Engl J Med. 2008;359:21–30. https://doi.org/10.1056/NEJMoa0706873.
71. Pavek P, Ceckova M, Staud F. Variation of drug kinetics in pregnancy. Curr Drug Metab. 2009;10:520–9.
72. Mentzelopoulos SD, Malachias S, Chamos C, Konstantopoulos D, Ntaidou T, Papastylianou A, Kolliantzaki I, Theodoridi M, Ischaki H, Makris D, Zakynthinos E, Zintzaras E, Sourlas S, Aloizos S, Zakynthinos SG. Vasopressin, steroids, and epinephrine and neurologically favorable survival after in-hospital cardiac arrest: a randomized clinical trial. JAMA. 2013;310:270–9. https://doi.org/10.1001/jama.2013.7832.
73. Kattwinkel J, Perlman JM, Aziz K, Colby C, Fairchild K, Gallagher J, Hazinski MF, Halamek LP, Kumar P, Little G, McGowan JE, Nightengale B, Ramirez MM, Ringer S, Simon WM, Weiner GM, Wyckoff M, Zaichkin J. Part 15: neonatal resuscitation: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care [published correction appears in *Circulation*. 2011;124:e406]. Circulation. 2010;122(suppl 3):S909–19. https://doi.org/10.1161/CIRCULATIONAHA.110.971119.
74. Connolly SJ, Dorian P, Roberts RS, Gent M, Bailin S, Fain ES, Thorpe K, Champagne J, Talajic M, Coutu B, Gronefeld GC, Hohnloser SH, Optimal Pharmacological Therapy in Cardioverter Defibrillator Patients (OPTIC) Investigators. Comparison of beta-blockers, amiodarone plus beta-blockers, or sotalol for prevention of shocks from implantable cardioverter defibrillators: the OPTIC Study: a randomized trial. JAMA. 2006;295:165–71. https://doi.org/10.1001/jama.295.2.165.
75. Clark SL, Meyers JA, Frye DK, Perlin JA. Patient safety in obstetrics: the Hospital Corporation of America experience. Am J Obstet Gynecol. 2011;204:283–7. https://doi.org/10.1016/j.ajog.2010.12.034.
76. Florea A, Caughey SS, Westland J, Berckmans M, Kennelly C, Beach C, Dyer A, Forster AJ, Oppenheimer LW. The Ottawa hospital quality incident notification system for capturing adverse events in obstetrics. J Obstet Gynaecol Can. 2010;32:657–62.
77. Grunebaum A, Chervenak F, Skupski D. Effect of a comprehensive obstetric patient safety program on compensation payments and sentinel events. Am J Obstet Gynecol. 2011;204:97–105. https://doi.org/10.1016/j.ajog.2010.11.009.

78. National Center for the Dissemination of Disability Research. What is knowledge translation? FOCUS Technical Brief No. 10. 2005. http://ktdrr.org/ktlibrary/articles_pubs/ncddrwork/focus/focus10/Focus10.pdf. Accessed 5 Sept 2015.
79. Graham ID, Logan J, Harrison MB, Straus SE, Tetroe J, Caswell W, Robinson N. Lost in knowledge translation: time for a map? J Contin Educ Health Prof. 2006;26:13–24. https://doi.org/10.1002/chp.47.
80. Lipman SS, Daniels KI, Carvalho B, Arafeh J, Harney K, Puck A, Cohen SE, Druzin M. Deficits in the provision of cardiopulmonary resuscitation during simulated obstetric crises. Am J Obstet Gynecol. 2010;203:179. e1–179.e5.
81. death P i, delivery i d. Sentinel Event Alert. 2004;30:1–3.
82. California Maternal Quality Care Collaborative. CDPH/CMQCC/PHI. The California Pregnancy-Associated Mortality Review (CA-PAMR): Report from 2002 and 2003 Maternal Death Reviews, Section on Preventable Deaths 2011:47–48. http://www.cmqcc.org/resources/1885. Accessed 5 Sept 2015.
83. Lipman SS, Daniels KI, Arafeh J, Halamek LP. The case for OBLS: a simulation-based obstetric life support program. Semin Perinatol. 2011;35:74–9. https://doi.org/10.1053/j.semperi.2011.01.006.
84. Schimmelpfennig K, Stanfill TJ. Advanced cardiovascular life support for the obstetric population: bridging the gap. J Perinat Neonatal Nurs. 2012;26:136–46. https://doi.org/10.1097/JPN.0b013e318252363e.

52 Intimate Partner Violence During Pregnancy

Anita Pal and Rohini Rao

Globally intimate partner violence (IPV) during pregnancy is rising alarmingly but still is a preventable health problem affecting millions of women worldwide. Intimate partner violence during pregnancy has many serious public health issues and thus results in significant negative health consequences for woman and child's well-being [1–6].

The IPV includes psychological abuse, progressive isolation, deprivation, intimidation, stalking, emotional trauma, controlling behaviour, physical impairment or even sexual assault jeopardising the pregnancy, chronic health problems and eventually death in some cases [7].

Regardless of the age, race, ethnicity, culture, socioeconomic status, religion, educational status and sexual orientation, it is seen worldwide and this burden of violence is borne by women and may have lifelong consequences.

52.1 Global Prevalence

A multi-country study conducted by WHO on women health and domestic violence (Fig. 52.1) found that the prevalence of IPV in pregnancy has a varied range from 28% in Peru province to only 1% in Japan with the average ranging between 4 and 12% in different parts of the world [8].

The data on IPV analysed by International Violence Against Women Survey found that IPV during pregnancy is between 2% in Denmark, Cambodia, Australia and the Philippines and 13.5% in Uganda, and this study also concluded that the majority is between 4% and 9%. Some of the clinical studies worldwide found that IPV during pregnancy is highest in Egypt with 32% followed by India (28%) and Saudi Arabia (21%) [9, 10].

52.2 Causes and Risk Factors for Intimate Partner Violence

A multi-country study conducted by WHO on domestic violence against women and their health clearly reported that the women who had physical abuse during pregnancy also had the history of abuse prior to the pregnancy. But almost 50% of the women according to the WHO study stated that they were abused for the first time in pregnancy [8].

A research done by Jasinski in 2004 clearly states that pregnancy does not stop or prevent the occurrence of intimate partner violence, but there is no strong evidence about whether IPV increase or decreases during pregnancy [11].

A. Pal (✉)
Kamla Nehru State Hospital for Mother and Child, Indira Gandhi Medical College, Shimla, India

Shimla Sanatarium and Hospital, Shimla, India

R. Rao
Kamla Nehru State Hospital, Shimla, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_52

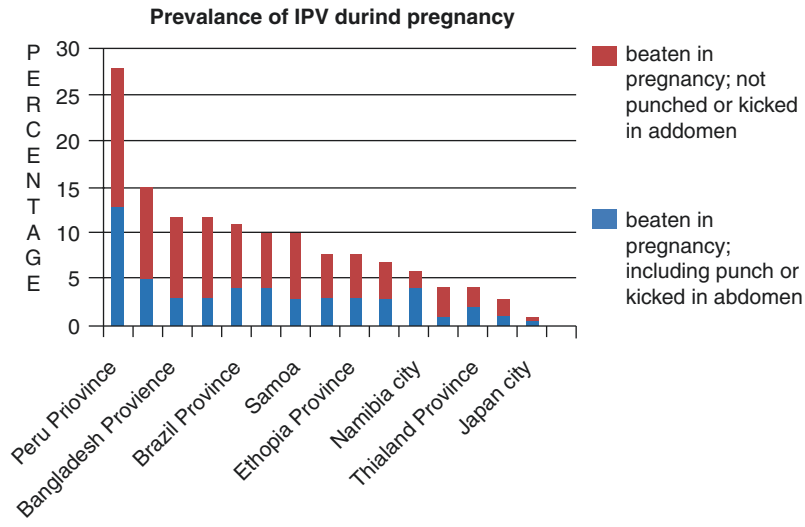


Fig. 52.1 IPV in pregnancy

1. Individual Factors
   (a) Male factors which increase likelihood of abusing or committing violence [12–14]:
   - Witnessing or experiencing abuse or violence as a child
   - Young age
   - Use of alcohol or drugs
   - Low level of education
   - Personality disorders
   - Acceptance of violence (i.e. a man can beat his partner)
   - Past history of IPV

   (b) Women factors which increase likelihood of accepting the abuse or experiencing violence [12, 13, 15]:
   - Low level of education
   - Sexual abuse during childhood
   - Exposure to abuse or violence between parents
   - Acceptance of violence
   - Prior exposure to other forms of violence or abuse
2. Relationship Factors

   Factors associated with increased risk of both perpetration of men and victimisation of women [12, 13, 16]:
   (a) Dissatisfaction or conflict in the relationship
   (b) Economic stress
   (c) Male dominance
   (d) Man having multiple partners
   (e) Difference or the disparity of education, i.e. female partner is more educated than her male counterpart
3. Social and Community Factors [12, 13]
   (a) Inequality regarding genders
   (b) Poverty
   (c) Women with low socioeconomic status
   (d) Lack of civil rights for female partners, including inequitable and restrictive divorce and marriage laws
   (e) For IPV weak community sanctions

There are many beliefs around the globe about gender role and IPV (Table 52.1) [13, 17, 18].

52.3 Health Consequences of Intimate Partner Violence

IPV during pregnancy can affect women's physical and mental health directly as injuries or indirectly as chronic health problems due to exaggerated stress. Whenever there is history of violence, the woman is at risk of many diseases or other serious health conditions which could have fatal or grave to nonfatal consequences on the women health and foetal growth or pregnancy outcomes (Fig. 52.2) [12].

Table 52.1 Gender role in IPV

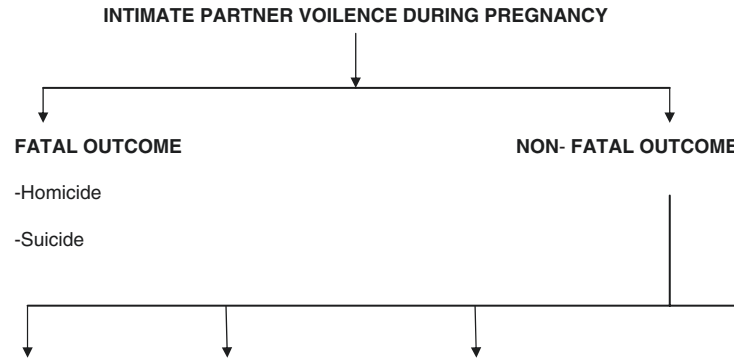
Beliefs that support IPV associated with the gender role	
1.	A man is considered socially superior to a woman and can assert power over a woman as his right
2.	For incorrect behaviour, the man can physically discipline her as his right
3.	For resolving conflicts in a personal relationship, physical violence is accepted at some places
4.	A man has every right in marriage for sexual intercourse
5.	It's the duty of the women to tolerate IPV to keep her family together
6.	There are some situations in which women deserve to be beaten
7.	Any sexual activity is regarded as a marker of masculinity (including rape)
8.	Considering that the girls are solely responsible for sexual urge in males

52.4 Suicide and Homicide

Suicide and homicide are the gravest consequences of IPV in pregnancy. Data from 11 US cities suggest that during pregnancy there is increased risk of becoming a victim and pregnant women are prone to be killed by their partner. The partners who actually abuse the women in pregnancy are more dangerous and are more likely to commit homicide [19].

Sexual, physical and psychological abuse during pregnancy can lead to anxiety, stress, depression as well as suicide attempts [20, 21].

Both suicide and homicide are two preventable causes of maternal mortality. Studies indicate that maternal injuries can be a leading cause of maternal morbidity and mortality [22–24].



SEXUAL & REPRODUCTIVE HEALTH	NEGATIVE BEHAVIOUR AND MENTAL HEALTH	PHYSICAL HEALTH AND INJURIES	SOCIAL RESTRICTIONS
Forced sexual intercourse	Substance abuse & alcohol consumption	Injuries like bruises & lacerations	Isolating the woman from family & friends
Other forms of sex	Smoking	Beating -slapping -hitting -kicking	Monitoring their movements
STIs, HIV, UTIs	Eating disorders	Physical impairment -fractures -broken teeth	Restricting access to financial resources, employment, medical facilities and education
Preterm	Poor self esteem	Sight & hearing damage	
Low birth weight babies	Sleeping disorders	Strangulations	
Miscarriage	Depression	Burns	
Unsafe abortion	Delay in antenatal care		
Obstetric complications	Anxiety & phobias		

Fig. 52.2 Intimate partner violence during pregnancy

According to a study done by Palladino, 54.3% of the pregnancy-associated suicides had history of intimate partner violence which ultimately attributes to suicides, and 45.3% of homicides associated with pregnancy were having evidence of IPV [25].

52.5 Negative Health Behaviour and Mental Health

The female who is abused during pregnancy is associated with negative health behaviour during the pregnancy or the postpartum period which includes substance abuse, smoking and alcohol consumption. Some of these females also develop eating disorders along with depression, stress, anxiety, sleeping disorders and poor self-esteem as compared to the nonabused women. These women develop low self-esteem; that is why to cope with the shame, stress or mental and physical suffering caused by IPV, they start to smoke, drink and indulge in self-medications. The delay in seeking the prenatal care is either due to the abusive partner who prevents her from leaving the house or the woman herself delays the appointments because of the injuries [2, 12, 26].

52.6 Physical Health and Injuries

The physical injuries resulting from IPV during pregnancy include bruises and welts, abrasions and lacerations, fractures and broken teeth, sight and hearing loss, beating and slapping, kicking and hitting on the abdomen and other private parts, head and neck injuries and attempts for strangulations. The direct hitting or blows on pregnant woman's abdomen are concerning as they lead to negative reproductive health outcome [6, 12, 27, 28].

A WHO multi-country study showed that the prevalence of physical injuries among females who were physically abused by their male partner in the last 12 months ranged from 3% in Serbia to 29% in Ethiopia. It also found that the women who were abused were likely to have poor mental and physical health compared to the nonabused women even years after the violence [29].

52.7 Social Restrictions

Sometimes the male partners in IPV try to isolate the women from friends and family; they also control and monitor their movements and can reach up to the extent that they restrict their partner's access to education, employment, financial resources and finally even medical care and help.

52.8 Sexual and Reproductive Health

The nonfatal IPV can lead to a spectrum of events of negative sexual and reproductive health consequences on the life of the female partner and the newborn. It may start with sexually transmitted diseases, pelvic inflammatory infections, urinary tract infection, forced sexual intercourse within marriage and sexual dysfunctions or sometimes makes it difficult for a woman to have access to contraception or condom use by their male partners [2, 30–32].

IPV has been associated with increased risk of unwanted pregnancy, unsafe abortions or miscarriage. The females who have faced abuse during pregnancy have a higher rate of preterm labour and intrauterine growth retardation than those not experiencing the abuse. The pregnancy complications include antepartum haemorrhage and prenatal death and may extend up to unattended pregnancy and delieveries [33–39].

52.9 Factors Not Allowing the Woman to Leave Her Partner

Not all females are ready to stay as passive victims with their violent partners. Some of the females adopt and plan strategies for their safety and child protection. But still a large number of women stay in their violent relationship because of the following factors:

- Lack of economic support
- Concern related to her children
- Fear of retaliation
- Family and friends not supportive
- Fear of divorce and losing custody of children
- Hope that the partner will change

Despite the above-mentioned factors and barriers, most of the abused women eventually leave their violent partner after multiple episodes of violence or years of violence. A WHO multi-country study clearly suggests that 19–51% of women who were ever physically abused by their violent partners have left their home for at least one night and 8–21% of women have left their home 2–5 times [29].

52.10 Roadmap for Prevention and Responding to IPV

Recently a number of international studies have highlighted various approaches for prevention of IPV and dealing with such cases. A multisectoral approach is required to achieve this goal. It needs co-ordinated and collaborated steps from government and social institutions/organisations.

The specific plans which are needed for achieving long-term effectiveness in controlling IPV include the following:

- Reforming the civil and criminal frameworks.
- Raising the awareness about existing laws through media.
- Coalition of institutions including government and civil is to be built.
- Building of evidence-based awareness and advocacy.
- By communication we have to use behavioural change so that society changes.
- Transforming every sector regarding gender perspective and special attention has to be given to IPV against women.
- Empowering the girls and women socially and economically.
- Comprehensive service has to be built and given in response to IPV.
- Starting of school-based programmes for life skills.
- Non-violence and gender equality should be promoted by engaging boys and men in society.
- Early intervention should be provided to a family who is at risk.

References

1. Shah PS, Shah J. Knowledge synthesis group on determinants of preterm/LBW births. Maternal exposure to domestic violence and pregnancy and birth outcomes: a systematic review and meta-analyses. J Womens Health (Larchmt). 2010;19:2017–31.
2. Campbell JC. Health consequences of intimate partner violence. Lancet. 2002;359:1331–6.
3. Murphy CC, Schei B, Myhr TL, Mont D, Abuse J. A risk factor for low birth weight? A systematic review and meta-analysis. CMAJ. 2001;164:1567–72.
4. Kiely M, El-Mohandes AA, Gantz MG, Chowdhury D, Thornberry JS, El-Khorazaty MN. Understanding the association of biomedical, psychosocial and behavioral risks with adverse pregnancy outcomes among African Americans in Washington, DC. Matern Child Health J. 2011;15:S85–95.
5. Silverman JG, Decker MR, Reed E, Raj A. Intimate partner violence around the time of pregnancy: association with breastfeeding behavior. J Womens Health (Larchmt). 2006;15:934–40.
6. El Kady D, Gilbert WM, Xing G, Smith LH. Maternal and neonatal outcomes of assaults during pregnancy. Obstet Gynecol. 2005;105:357–63.
7. Family Violence Prevention Fund. Reproductive health and partner violence guidelines: an integrated response to intimate partner violence and reproductive coercion. San Francisco, CA: FVPF; 2010. http://www.futureswithoutviolence.org/userfiles/file/HealthCare/Repro_Guide.pdf. Accessed 12 Oct 2011.
8. García-Moreno C, Jansen HA, Ellsberg M, Heise L, Watts C. WHO multi-country study on women's health and domestic violence against women: initial results on prevalence, health outcomes and women's responses. Geneva: World Health Organization; 2005.
9. Devries KM, Kishor S, Johnson H, Stöckl H, Bacchus L, Garcia-Moreno C, et al. Intimate partner violence during pregnancy: prevalence data from 19 countries. Reprod Health Matters. 2010;18(36):1–13.
10. Campbell J, Garcia-Moreno C, Sharps P. Abuse during pregnancy in industrialized and developing countries. Violence Against Women. 2004;10(7):770–89.
11. Jasinski JL. Pregnancy and domestic violence: a review of the literature. Trauma Violence Abuse. 2004;5(1):47–64.

12. Heise L, Garcia Moreno C. Violence by intimate partners. In: Krug EG, et al., editors. World report on violence and health. World Health Organization: Geneva; 2002. p. 87–121.
13. WHO/LSHTM. Preventing intimate partner and sexual violence against women: taking action and generating evidence. Geneva/London: World Health Organization/ London School of Hygiene and Tropical Medicine; 2010.
14. Johnson KB, Das MB. Spousal violence in Bangladesh as reported by men: prevalence and risk factors. J Interpers Violence. 2009;24(6):977–95.
15. Abramsky T, et al. What factors are associated with recent intimate partner violence? Findings from the WHO multi-country study on women's health and domestic violence. BioMed Central Public Health. 2011;11:109.
16. Ko LC. Sexual violence against women and children in Chinese societies. Trauma Violence Abuse. 2009;10(1):69–85.
17. Heise L, Ellsberg M, Gottemoeller M. Ending violence against women. Baltimore, MD: Johns Hopkins University School of Public Health, Center for Communications Programs; 1999.
18. Swart LA, et al. Violence in adolescents' romantic relationships: findings from a survey amongst school-going youth in a South African community. J Adolesc. 2002;25(4):385–95.
19. Campbell JC, Webster D, Koziol-McLain J, Block C, Campbell D, Curry MA, et al. Risk factors for femicide in abusive relationships: results from a multisite case control study. Am J Public Health. 2003;93(7):1089–97.
20. Martin SL, Li Y, Casanueva C, Harris-Britt A, Kupper LL, Cloutier S. Intimate partner violence and women's depression before and during pregnancy. Violence Against Women. 2006;12(3):221–39.
21. Zeitlin D, Dhanjal T, Colmsee M. Maternal-foetal bonding: the impact of domestic violence on the bonding process between a mother and child. Arch Womens Ment Health. 1999;2(4):183–9.
22. Krulewitch CJ, Pierre-Louis ML, de Leon-Gomez R, Guy R, Green R. Hidden from view: violent deaths among pregnant women in the district of columbia, 1988–1996. J Midwifery Womens Health. 2001;46:4–10.
23. Nannini A, Weiss J, Goldstein R, Fogerty S. Pregnancy-associated mortality at the end of the twentieth century: Massachusetts, 1990–1999. J Am Med Womens Assoc. 2002;57:140–3.
24. Campbell JC, Glass N, Sharps PW, Laughon K, Bloom T. Intimate partner homicide: review and implications of research and policy. Trauma Violence Abuse. 2007;8:246–69.
25. Palladino CL, Singh V, Campbell J, Flynn H, Gold KJ. Homicide and suicide during the perinatal period: findings from the national violent death reporting system. Obstet Gynecol. 2011;118:1056–63.
26. Parker B, Bullock L, Bohn D, Curry M. Abuse during pregnancy. In: Campbell JC, Humphreys J, editors. Family violence and nursing practice. Philadelphia, PA: Lippincott; 2003. p. 77–94.
27. Thananowan N, Heidrich SM. Intimate partner violence among pregnant Thai women. Violence Against Women. 2008;14(5):509–27.
28. Bacchus L, Bewley S, Mezey G. Domestic violence in pregnancy. Fetal Matern Med Rev. 2001;12(4):249–71.
29. Garcia-Moreno C, et al. WHO multi-country study on women's health and domestic violence against women: initial results on prevalence, health outcomes and women's responses. Geneva: World Health Organization; 2005.
30. Campbell J, Soeken K. Forced sex and intimate partner violence. Violence Against Women. 1999;5(9):1017–35.
31. Champion J, Shain R. The context of sexually transmitted disease: life histories of woman abuse. Issues Ment Health Nurs. 1998;19(5):463–79.
32. Gazmararian JA, et al. The relationship between pregnancy intendedness and physical violence in mothers of newborns. Obstet Gynecol. 1995;85(6):1031–8.
33. Altarac M, Strobino D. Abuse during pregnancy and stress because of abuse during pregnancy and birthweight. J Am Med Womens Assoc. 2002;57(4):208–14.
34. Bullock LF, McFarlane J. The birth-weight/battering connection. Amer J Nurs. 1989;89(9):1153–5.
35. Valladares E, Ellsberg M, Pena R, Hogberg U, Persson LA. Physical partner abuse during pregnancy: a risk factor for low birth weight in Nicaragua. Obstet Gynecol. 2002;100(4):700–5.
36. Fanslow J, Silva M, Whitehead A, Robinson E. Pregnancy outcomes and intimate partner violence in New Zealand. Aust N Z J Obstet Gynaecol. 2008;48(4):391–7.
37. Pallitto CC, Campbell JC, O'Campo P. Is intimate partner violence associated with unintended pregnancy? A review of the literature. Trauma Violence Abuse. 2005;6(3):217–35.
38. Janssen PA, Holt VL, Sugg NK, Emanuel I, Critchlow CM, Henderson AD. Intimate partner violence and adverse pregnancy outcomes: a population-based study. Am J Obstet Gynecol. 2003;188(5):1341–7.
39. Jejeebhoy SJ. Associations between wife-beating and fetal and infant death: impressions from a survey in rural India. Stud Fam Plann. 1998;29(3):300–8.

Examination of Sexual Assault Victim

53

Alka Vijay Kuthe

53.1 Introduction

Sexual abuse is a serious infringement of one's rights to health and protection. It is not only a particular nation's problem but has become global public health issue. Sexual abuse until 1970 was restricted to poor economy class of people only and was rarely found. Now the cases are seen in all socioeconomic groups. Sexual abuse includes genital–genital, genital–rectal, oral–genital, hand–breast, hand–rectal, and hand–genital contact, exposure of sexual anatomy, forced view of sexual anatomy, and using a victim in the production of pornography or showing pornography.

Sexual assault is cognizable, non-bailable offence under the criminal law, and the offender is liable for punishment that includes imprisonment of varying duration, fine, and even death penalty. It is one of the serious crimes in India.

Sexual intercourse or sex-related acts performed in a way which is against the provision of the law of the land [1] (sexual offences) are classified into:

1. *Natural offences:* Sexual offences which are performed in the order of nature, i.e., by penetration of the female organ (vulva) by the male organ (penis), are called as natural offences.

   Examples: (1) Incest, (2) adultery, and (3) rape
2. *Unnatural offences:* Sexual offences where intercourse is performed against the order of nature, i.e., when the act does not involve penetration of a woman's vagina by the penis of a man, are included under unnatural offences.

   Examples: (1) Bestiality, (2) buccal coitus, (3) tribadism, and (4) sodomy
3. *Sexual perversions:* These are the sexually perverted acts such as (1) necrophilia, (2) sadism, (3) masochism, (4) transvestism, (5) fetichism, (6) masturbation, (7) voyeurism, (8) frotteurism, (9) undinism, and (10) exhibitionism.

Rape as said by the *UN*'s human-rights chief is a "*national problem*" and is regarded as one of India's most common crimes against women. A new case is reported every 20 min even in developed countries. Among Indian cities, New Delhi has the highest rate of rape cases. Rape cases in India have doubled between 1990 and 2008. 24,206 rape cases were registered in India in 2011 (National Crime Records Bureau) although experts agree that the number of unreported cases is much higher. Considering the magnitude, seriousness of the offence, and its long-term impact on the life of the victim, it is the obligatory duty of the medical and paramedical personnel to help the judiciary and crime department to convict the

A. V. Kuthe (✉)
Kuthe Accident Hospital and Maternity Nursing Home, Amravati, Maharashtra, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_53

offender by collecting evidence against him by doing proper examination of the sexual assault victim.

Sexual Assault Victim

53.2 Examination of the Alleged Case of Sexual Assault Victim

The comprehensive forensic medicolegal examination of the victim is very important in the full investigation of the case and the building of an effective prosecution in the court. It should be conducted without unnecessary delay so that the findings should not disappear or mislead. For example, spermatozoa, if present in the vagina, should not disintegrate [1]. The alleged victim of sexual assault should be protected from any additional emotional trauma during physical examination. The details of medical examination of the victim are as follows.

Objects of Medical Examination [2]

1. Look for physical signs (injuries) that will corroborate the history given by the victim.
2. Collect and preserve all physical (trace) evidence for laboratory examination.
3. Offer treatment to the victim for injuries and against venereal disease or pregnancy.
4. Prevent or minimize permanent psychological damage.

General Method [2]

The following is an outline of the examination procedure:

1. Authorized person either a magistrate or the officer in charge of a police station should send a requisition for examination of the victim in connection with the alleged incident of rape.
2. To identify the victim before the medical officer, an authorized person should be there about whom there should be mention in the requisition.
3. The woman cannot be forced for medical examination against her will by the court or the police. Therefore prior written consent in the presence of a witness should be obtained before the start of medical examination of the alleged victim if she is 12 years or above and of parents or guardians (about whom there should be mention in the requisition) if her age is below 12 years or if the alleged victim is mentally unsound or intoxicated. So as to avoid subsequent criminal charge of indecent assault against the examining doctor, consent for examination is a must.
4. It is necessary to note down identification marks of the alleged victim.
5. The rape victim should be allowed to *give her own account* of the act without any questions being put to her.
6. The name of the victim and her parent/husband if married, age, height, weight, residence, occupation, date, time and place of examination, and name of the police station

by whom the requisition is given should be accurately recorded.

7. The developmental, behavioral, mental, and emotional status in the case of the child/adolescent victim should also be briefly assessed.
8. Medicolegal examination is one of the star evidences in the judicial proceeding that should not be ignored. The tenderness and swelling of the vulva may disappear after some hours. The possibility of finding spermatozoa from the genital tract also decreases with delay. Therefore it is advisable to examine and collect all relevant and feasible materials from a victim at the earliest after the incident, to achieve the above goal.
9. Record statements of the victim and of others with her separately. They should include:
   (a) Preliminary affairs
   (b) Date, time, and place of the offence alleged
   (c) Parties' exact relative positions
   (d) Evidence of struggle or resistance
   (e) Calls for help
   (f) Whether she experienced any pain then and afterwards
   (g) Was there ejaculation during the act, either within the vagina or outside
   (h) Any associated discharge
   (i) Whether she had any bleeding from the vagina
   (j) Whether there was loss of consciousness anytime during the attack
   (k) Any other events after the alleged assault
   (l) When the first complaint was filed and if there was undue delay, the reason for that.

   All this must be taken down verbatim.
10. It is important to note down previous history with regard to sexual experience, menses, vaginal discharge, venereal disease, pregnancies, pelvic operations, etc. Children who are subjected to sexual assaults may not be able to give proper history. So the examiner must have high degree of suspicion. Many times a sexually assaulted victim might have been brought to a gynecologist, only with the history of trauma and bleeding per vagina or inability to pass the urine or loss of appetite. Hence, a medical practitioner is not only morally but also legally bound to take detailed history from the child, guardian, accompanying person, and police inspector and document it.
11. The victim is examined in the presence of an adult mentally sound female attendant, either hospital nurse or a female relative of the alleged victim. This applies specially to male medical officer.
12. Note down the physical development in order to determine her capacity to struggle and resist.
13. Her general behavior and mental status should be observed, while she narrates her story.
14. Her gait should be observed, and specific questions related to pain while walking, on micturition, or defecation should be asked. We can notice guarded gait due to pain, the victim walking with legs apart and slow steps(women should not be made naked to test the gait).
15. The removal of dress should be done by the victim herself or by the female attendant and not by the male doctor himself. No force to undress her should be made by the attendant.
16. A second examination should be done after stoppage of menstruation if the victim is in menstrual period.

The identity of the victim against whom an offence is alleged to have been committed should not be disclosed and is *not permitted* [3].

Evaluation of Mental Status [1]

The victim should subsequently be referred to a psychiatrist if mental unsoundness is doubted. Note down if she appears to be intoxicated and under the influence of drugs or alcohol, and her blood and urine samples should be preserved after necessary physical examination.

Evaluation of Developmental Status of Secondary Sex Features [1]

The appearances and growth of breasts, axillary, and pubic hair should be noted. The sexual habit of the woman can be guessed from the

appearance of the breasts. The breasts will be hemispherical, firm, and spongy with similar nipples and have pinkish areola in a woman not accustomed to sexual practices. The breasts will be larger, lax, and slightly pendulous with larger raised nipples in women habituated with sexual practices. The areolar color may not change before pregnancy.

Examination Proper [2]

It includes:

1. **Examination of the clothes:**

   Inquiry about the change of clothing and a bath or wash should be made. Attempt should be made to find out whether the clothes of the victim are those worn at the time of sexual assault or changed. Each item of the clothing should be examined for stains (blood, seminal, mud, grass, etc.), soiling, tears and loss of buttons, and the site and type of damage. If the offence has been committed in an open place, corroborations can sometimes be obtained by finding grass, leaves, mud, etc. on the buttocks or on the back. In certain cases stains may be present on pieces of material or handkerchief used by the victim after assault for cleaning purpose. Vulval pads and vaginal tampons whether worn at or after the time of the incident should be preserved. Clothes play very important role in corroborating or contradicting her story. Foreign hair, fibers, etc. found on the clothes or on the skin surface must be preserved and compared with those found on the accused.
2. **General examination:**

   It includes inspection of the whole body for marks of violence resulting from struggle, their appearance, situation, extent, and probable age. They can be found around/on (1) the mouth and throat, produced while preventing her from calling for help, (2) wrists and arms, (3) the inner side of thighs and knees, (4) the back from pressure on hard ground, and (5) the breasts by rough handling. True bite marks and love bites are usually found on the breasts, neck, chest wall, lower abdomen, and upper parts of the thighs. The nature and situation of the general injuries should correspond with the victim's description of the assault. Marks of violence are likely to be found in one-third cases. The absence of general injuries may be due to: (1) Submission of the victim due to fear of injury or death, etc. (2) Insufficient force to produce an injury. (3) After 48 h bruises may not be noticed. (4) Delayed reporting of the incident during which minor injuries will heal.

   General injuries are observed only in 1/5th cases because of (1) alleged sexual act consisting of only rubbing or touching the genitalia, (2) sexually experienced victim, (3) elasticity of genitalia and hymen in a post-pubertal female, and (4) the use of lubricants. The victim usually scratches the assailant during the struggle. Debris under the nails should be removed and examined for epidermal cells, blood, fibers, etc. At the same time, any damaged fingernails should be noted.
3. **Special local examination** [2]:

   After completing the consent formalities depending upon the age of the alleged victim, she is placed on an examination table in good light with her legs drawn up and widely opened (lithotomy or knee-chest position). If possible, a vaginal speculum should be used. Vaginal lining should be examined to see any abrasion, bruises, erosions, or vault tears. Digital examination may show (1) areas of pain and tenderness in the vagina, (2) some laxity of the vaginal orifice (indicating previous penetration), or (3) elongation of the posterior fornix of the vagina (indicating frequent sexual intercourse).The size of the vagina should be noted as admitting one, two, or three fingers as the case may be(Pl refer Supreme Court Judgment quoted later on).

   The police or court has no power of compelling a woman to submit the private part of her person to the examination of a medical practitioner [3].

   The local findings may change depending upon the type of victim as below.

(a) *Rape on a virgin* [2]: The hymen is examined by gently separating the labia. Rupture of the hymen occurs with the first intercourse, which is the main evidence of rape in a virgin. The nature and extent of injury varies in different cases depending upon (1) disproportion between male and female parts, (2) extent of penetration, (3) the nature of the hymen, and (4) amount of force used. Tearing usually occurs posteriorly at the sides in the 4 or 8 o'clock position or in the midline of the hymen. More than one tear may occur. Several hymenal lacerations indicate first intercourse. Tears usually occur in the posterior midline of the hymen because the hymen lies suspended across a potential space, whereas anteriorly the periurethral tissues buttress the hymen. Any object passing through the hymenal orifice which is larger than its original opening will cause a V-shaped cleft or clefts. One deep tear may be seen at 6 o'clock position or a number of tears usually in the posterior half of the membrane. With healing over a period of months, V-shaped tear becomes rounded and appear as U-shaped defects. In pre-pubertal children, the posterior tear of the hymen may involve fourchette producing a deep U-shaped defect. The margins of the torn hymen are sharp and red which bleed on touch; the tissues around about them are swollen and tender soon after the act. The edges of laceration are congested and swollen after 3–4 days of an offence and get healed completely in a week, but they do not unite. Rupture of the hymen due to sudden stretching can be caused by agents other than male genital organ such as fingers, and therefore, evidence of local injury is not proof of penetration. Many a times, in the absence of hymenal tearing, there is abrasion and bruising of the hymen and the vaginal orifice.

For close examination of the hymen, Glaister-Keen glass rod, warmed to body temperature, should be passed through the hymenal orifice. Then it is passed around the posterior surface of the hymen, which is slightly stretched by separation of labia minora. The edges of the hymen become slightly everted by this procedure. By slowly rotating the sphere around the edges, natural notches are easily differentiated from tears, recent or old. This method does not cause any injury or pain. Another method is to pass a finger into the rectum above the perineal body and push the posterior vaginal wall forwards and downwards. This pushes the hymen forwards which is clearly seen entirely, lying against the posterior vaginal wall.

Injury to the labia is not common, but fingernail scratches may be present on the labia, particularly the labia minora. The labia may be red and inflamed with slight edema of the vaginal introitus if it is the first sexual act. Bruising and lacerations of external genitals may be present with redness, swelling, and inflammation if there is disproportion between the male and female genitals. Swelling and congestion of the mucosa at introitus, the clitoris, and the labia minora are caused by genital stimulation, but they may also be caused by digital stimulation or masturbation. These signs usually fade in 1 or 2 h. Swelling and tenderness of the labia minora may indicate sexual activity. The posterior commissure is usually intact in the virgin and often ruptures at first intercourse, especially if there is much disparity in the size of the penis and vagina. The fourchette is fragile and often tears during the first intercourse. Fossa navicularis disappears. Bruising of the vagina is seen as dark-red areas against the overall redness of the vaginal mucosa, and within 24 h, the color becomes deep red or purple. It is more frequently seen on the anterior vaginal wall in the lower third and on the posterior vaginal wall in the upper third. Bruising of this nature is more consistent with penile penetration than with digital penetration. These types of injuries can

occur during consenting sexual intercourse. In rape or digital penetration without consent, where preliminary stimulation has not taken place, initial lubrication will be lacking, due to which more severe local bruising or abrasion can result. In women of childbearing age, frank laceration of the vaginal wall or vault is rare following sexual intercourse, but it can occur in very young children and in the atrophic post-menopausal vagina. Posterior laceration of the vaginal wall occurs with violent intercourse or where there has been considerable disproportion between the penis and the vagina. Severe stretching/tearing of the vagina and labia may occur even with slight entry of the penis. Some degree of local injury may be caused by sudden forcible dilatation of the vagina in majority of adult rapes. But one should keep in mind that even with consenting woman, bruising, laceration, or abrasion are at times seen with forceful intercourse that does not indicate rape. In all cases where there are no marks of fresh injury, one should do vaginal examination to assess (1) the laxity of the vaginal orifice, (2) the length of the vagina into the posterior fornix, (3) the number of fingers that can be introduced through the hymenal orifice, and (4) the areas and the degree of tenderness complained of by the patient. All these should be attempted if the state of the hymen permits. A finger may be inserted into the vagina in most young women although the hymen is intact, which is felt as a constricting ring around the tip of the finger. Vaginal examination helps the examiner to assess the elasticity of the hymen and to determine the degree of penetration which would be possible without its rupture. The possibility of sexual intercourse may be inferred if the vaginal opening easily admits two fingers (as per the present laws of evidence, two-finger test has no special importance). The vagina needs to be inspected for signs of bruising, abrasion, and laceration by introducing speculum.

(b) *Rape on Deflorate Women* [2]:

The hymen is completely destroyed, the vaginal orifice dilated, and the mucus membrane wrinkled and thickened in the case of deflorate women, even without childbirth. Complete penetration can occur in such women without any evidence except semen. The presence of spermatozoa in the vagina is the only proof that penetration has occurred. Therefore, the absence of injuries under certain circumstances does not exclude even complete penetration. In a married woman though, marks of violence to the genitalia are less likely to be found, but they must be looked for because rape is associated with greater violence than sexual intercourse with consent. The vagina may show laceration, bruising, deep injury with effusion of blood, swelling, and inflammation of the vulva even when no marks of violence indicating a struggle may be found externally. Tearing or perforation of the vagina may occur when it is thin or fragile. When older women are raped, senile atrophy and friability of their genitalia result in extensive vaginal lacerations and perineal trauma. In women who have been used to sexual intercourse, injuries from rape usually disappear or become obscure in 3–4 days. When there has been much violence, they may persist for longer duration. The chief evidence of crime may be found on other parts of the body in the form of injury marks.

(c) *Rape on children:*

In India, due to the superstitious belief that gonorrhea and syphilis or other sexual transmitted diseases can be cured by sexual intercourse with a virgin, rape on children is common [3].

The following are steps of systematic examination of the sexual assault on child victim.

First Approach: The first approach a forensic pathologist should employ, during a medicolegal investigation of a sexual assault

in child, is of grave importance. First, to provide a simple and comprehensive overview of the process that will be followed, he should spend enough time to become acquainted with the child. The health provider should start his conversation with interesting topic and not "threatening" to the child. Discussing with the child about special interests (school, sports, or music lessons) decreases the tension and enables the child to relax, which is important for the vaginal and anal examinations. A lab coat or other hospital and medical suits put on by a doctor may be frightening for younger children. The history must be obtained by a highly skilled professional without leading questions. When taking a history, the doctor must use language that is appropriate, supportive, and demonstrative of a friendly and caring attitude. What they state should be recorded in their own words.

Assessment: A full clinical inspection must be undertaken including skeletal radiological survey. Such examinations involve a forensic medical examiner and a pediatrician. However, at times, it may be necessary to involve another medical professional such as genitourinary physician, psychiatrist, or family planning doctor.

Medical History: Medical history is of grave importance. Old injuries should be recorded meticulously and considered with extreme caution.

Examination: It includes complete physical examination with special attention to the mouth, breasts, genitals, perineal region, buttocks, and anus, with careful photographic recording of any trauma away from the genital area. These injuries, though can be serious, can be overlooked when the examiner focuses attention only on the genital area. Restraining force can be severe enough to leave "fingertip" and other bruises on the limbs or strangling marks on the neck. There can be trauma to the breast, inner thigh, or other para-genital areas. If bite marks are found, it is important to measure and photograph them carefully to allow matching or exclusion of the teeth of the alleged offender.

Genital Examination: Detailed examination of the genital region along with examination of the anal area in the appropriate position is necessary to detect positive findings in the child victim. At times the physical examination may reveal normal findings as the child usually is unaware of what is happening and also not capable of resisting. The hymen is deeply situated, and, as the vagina is very small, it is impossible for the adult organ to penetrate. Therefore the penis is placed either in between the thighs or within the vulva. There may be little redness and tenderness of the vulva, and the hymen may remain intact.

During sexual act, the penis compresses the labia both anteriorly and laterally, producing bruising of both labia majora and minora. Further penetration forces the penis backwards and the hymen is torn posteriorly. If the penis further advances in to the vagina, the hymenal tear extends into or through the perineal body and often involves the anterior wall of the anorectal canal. The younger the child, the more widespread are the injuries. Circumferential injuries of the mucosa of the vestibule are common. Full penile penetration can cause bruising and tears of the anterior and posterior vaginal wall. The bladder can get involved in the anterior tears, while posterior tears can involve the anorectal canal. The vaginal vault may rupture and the abdominal viscera may herniate through the vagina. Some scratching or bruising of the labia and vestibule can occur without circumferential tears if the infant vagina is digitally penetrated. There can be linear tear in the hymen in the posterior or posterolateral quadrant, which may extend into the posterior vaginal wall and onto the skin of the perineum. While separating the thighs for examination, the victim may experience severe pain due to local inflammation. There can be difficulty while walking due to pain. In the differential diagnosis of anal sexual abuse, the practitioner must be aware of non-abuse-related conditions, for example, Crohn's disease, child with significant constipation, etc.

The hymen may remain intact after rape, in the following circumstances [1]:

1. If there is incomplete penetration.
2. There may be congestion or bruise of course if the hymen is tough, fleshy, and elastic.
3. Due to deeper placement of the hymen and less capacity of the vagina, in very young children, full penetration with rupture of the hymen may not occur. There may be congestion, bruise, or even tear laceration in the posterior wall of the introitus and vagina which may extend up to the perineum.

4. **Evidence sampling—laboratory examination**:

The next important step is gathering the biological evidence of the alleged sexual abuse that has occurred within the preceding 72 h. Corroborative evidence can be obtained with the help of forensic laboratory and are decided by the forensic expert who decides about cultures and serological tests for sexually transmitted infections according to the special circumstances of the case. The swabs from the mouth, anus, or vagina should be allowed to dry in the atmosphere before they are sealed. Though the documented presence of an ejaculate is the most positive identifying element for the expert, its absence by no means refutes sexual assault. Evidence should be preserved carefully and a written record kept. For the identification of assailant identification, tests for genetic markers in the blood saliva and serum (ABO typing and other blood enzyme systems), apart from exploration of different characteristics of the head and pubic hair, should be performed within 72 h of acute sexual assault or sexual abuse. The identity of a perpetrator can be established with a high degree of certainty DNA fingerprinting. If one suspects that the child has been abused under the influence of drugs, toxicological analysis of blood and urine should also be performed. Sexual abuse is a criminal offence and is investigated by the police. Physicians and care providers need to report all suspected cases of child abuse and neglect. These laws offer protection to them. But if one fails to report suspected child abuse, it may result in a penalty. Pregnancy test should be performed in each case of sexually abused girl in a reproductive age if she misses the next menstrual cycle.

5. **Corroborative Signs of Rape** [2]

Medical examination can reveal some signs which indirectly support the main issue of rape. Rape can be inferred if these signs are present in the alleged victim. They are as follows:

(a) *Seminal fluid*: The thighs, pubic hair, and vagina should be examined. The presence of spermatozoa in the vagina is proof of sexual intercourse but not of rape. Similarly their absence is not proof that intercourse has not taken place, for they might have disappeared due to washing or there might not have been an emission, or the alleged victim might have been aspermic due to vasectomy or naturally. The presence of fresh seminal stains on the person or clothes of a woman provides strong evidence of intercourse, attempted or committed. Samples for seminal stains should be taken from the introitus and perineum before digital examination.

(b) *Sexually transmitted diseases*: The fact of sexual contact can be inferred indirectly from the diagnosis of sexually transmitted disease in the case, as the assailant may transmit the disease to the alleged victim if he is suffering from the disease. It becomes a strong corroborative evidence of rape, if the period of clinical manifestations after the alleged rape matches with the incubation period of the disease especially if the man denies sexual intercourse with the alleged victim. That will also help in treating the disease in the sexually abused victim. In gonorrhea, an inflammation with an abundant mucopurulent discharge will be seen in 2–4 days (occasionally a week), while in syphilis an indurated ulcer on the external genitals

may appear in about 3 weeks. In the case of adults, smears should be taken from the cervix and urethra for gonococci which are kidney shaped, intracellular, Gram-negative diplococcic. In the case of small girls, smears should be taken from the vagina. After ruling out the perinatal transmission, gonorrhea or syphilis infections are diagnostic of sexual abuse. It is extremely unlikely to find herpes type 2, chlamydia, trichomonas, and condyloma infections due to anything but abuse especially in children above the age of 1 year. An initial negative smear may be of value, if a positive smear is obtained within a few days of assault. A blood sample should be taken for serological examination in cases of suspected syphilis. An initial negative report may be of value, if a positive reaction is obtained after 6 weeks.

(c) *Mark of violence or struggle (*scratches, abrasions, and bruises on the body) especially on the forearm, wrist, face, breasts, nipples, cheeks, lips, chest, lower part of the abdomen, inner aspects of the thighs, and back should be specially looked for. The victim may show signs of active resistance in the form of bent or broken fingernails due to scratching the accused. Medical examination may detect debris under the nails in the form of blood, fibers, hair, and skin fragment from the accused.

Swabs from teeth bite area should be taken for the presence of saliva of the accused for investigation.

6. **Findings Related to Time of Assault** [2]:
   (a) *Wounds*: If the age of any wound present is consistent with the alleged time of rape, it will be a valuable piece of evidence.
   (b) *Venereal Disease*: The presence of venereal disease is useful in determining the approximate time of sexual assault. The medical examination of the victim and the accused and the evidence collected may either confirm or contradict the allegations made.
   (c) *Seminal Fluid*: The motility of the spermatozoa deposited in the vagina is lost within 1–6 h. At the end of 6 h, no motile sperms are found. Sperms may be recovered up to 24 h from the vagina.
7. **Specimens to be collected from victim:**
   The objects of collecting specimen are:
   (1) To obtain confirmation of the allegation
   (2) To attempt to establish a link between victim and the scene
   (3) To attempt to establish a link between victim and the assailant

   The following specimens are needed to be collected:
   (1) Avulsed head hairs.
   (2) Pubic hair combings.
   (3) Avulsed pubic hair.
   (4) Matted pubic hair, if present.
   (5) Loose hairs found anywhere on the body.
   (6) Saliva for secretor grouping.
   (7) Swabs from bite marks for saliva.
   (8) Blood (plain) for grouping.
   (9) Blood (anticoagulated) for alcohol and drugs.
   (10) Urine for drug screening.
   (11) Nail scrapping or nail cuttings for blood or tissue from other party.
   (12) Secretions from the introitus, vagina, posterior fornix, and cervical os should be obtained on the swab, transferred to a slide and spread out in the form of a thin film, and fixed and examined microscopically for the presence of sperms or any venereal infection such as gonorrhea or syphilis.
   (13) Swabs from any soiled areas of the skin.

   Sometimes even after sexual intercourse/alleged sexual assault, no sperms could be detected if [3]:
   (1) Condom was used.
   (2) The accused failed to ejaculate or ejaculation occurred outside the vagina.
   (3) The accused is azoospermic/vasectomized person.
   (4) Vaginal douching is done immediately after the act.

Dried blood and seminal stains on the external genitals and thighs should be scraped

and preserved for subsequent examination. The accused can be identified by noting the stains of urine, saliva, hair, and general debris present on the clothing of the victim by comparing them with the known materials from the accused. The finding of venereal disease also gives clue.

Examination Protocol in India [4]

The public anger against the treatment of rape survivors has forced the government to look into the existing anti-rape laws, and that also includes the crucial medical examination to ascertain the incidence of rape generally done by government hospitals and medical centers.

CNN-IBN's medical expert Dr. Shubham Pant takes us through the existing provisions in the Indian law, a protocol whereby there is a checklist that must be followed in the case of medical examination of a rape survivor:

(1) Medical examination can only be done with the prior consent of the rape survivor.
(2) Only allopathic government doctors, registered with the Medical Council of India, are allowed to conduct the examination. There should ideally be a woman gynecologist present during the examination.
(3) Samples are to be collected from the genitalia and under the nails.
(4) At the time of medical examination of a female by a male doctor, female attendant must be present, otherwise the doctor can be charged with indecent assault under the law.

The two-finger test to determine sexual assault and rape has been phased out in certain hospitals though many places still continue the practice. In India, due to the stigma attached to rape and medical examination, the victim has to face a lot of psychological and physical trauma.

Virginity Test [5]

The virginity test determines whether a person, usually a girl or woman, is a *virgin*, i.e., whether she has never engaged in *sexual intercourse*. It checks for the presence of an intact *hymen*, assuming falsely that it can only be torn as a result of sexual intercourse. It is widely considered controversial, both because of its implications for the tested girls and women and since it is viewed as *unethical*. A detailed examination of the hymen may be performed in cases of suspected rape or child sexual abuse, but the condition of the hymen alone is not conclusive.

Two-Finger Test [5]

Previously *two-finger test* was used to determine if she was "habituated to sexual intercourse." However, in the present era the usefulness of these criteria has been questioned by medical authorities and opponents of virginity testing, the reason being that vaginal laxity and the absence of a hymen can both be caused by other factors.

As per the Supreme Court of India, the two-finger test on a rape victim violates her right to privacy, and therefore it asked the Indian government to provide better medical procedures to confirm sexual assault. The test was also strongly criticized by *the Human Rights Watch* as **degrading and unscientific** and an additional assault on traumatized women.

Thus evidence of rape is obtained from:

1. Marks of violence on the body of the victim and the accused
2. The presence of stains of semen or of blood on the clothes and the body of the victim and the accused
3. Marks of violence in the genital region
4. The finding of seminal fluid in the vagina
5. The diagnosis of sexually transmitted infections in both the parties

Rape is a legal definition and not a medical diagnosis. Medical proof of intercourse does not necessarily mean rape. The doctor should just give opinion that there are signs of recent vaginal penetration, recent sexual intercourse, general physical injuries, and/or intoxication, and since the signs are consistent with the history given, rape might have been committed by the alleged accused. He/she should never give confirmed diagnosis of rape. Independent corroboration of the victim's story may be obtained by the statement of eye witness and evidence gathered from the bodies of both the victim and accused and from the crime scene. Many a times as there is least possibility of eye witness, since many rapes occur at the

time and/or areas when the victim is found alone, circumstantial evidence is offered in most of the cases.

8. **Follow-up** [6]**:**

   Follow-up visit helps the victim to recover from the trauma, both physical and mental, and regain dignity and self-respect. Accordingly, follow-up involves (1) tetanus prophylaxis, (2) prevention and termination of pregnancy, (3) treatment of injuries, (4) prevention and treatment of any sexually transmitted disease, and (5) referral to crisis intervention centers for support by social workers and psychiatrists.

9. **Medicolegal aspects:**

   (a) **Death as a result of rape** may occur from [2]:
   - Hemorrhage from injuries to genitals and perineum.
   - Shock due to fright and emotion or by blunt force.
   - If the mouth and nostrils are closed by the hand or cloth to prevent the female from crying for help, leading to suffocation/asphyxia or by strangulation.
   - Septic infection after several days or weeks.
   - Murder to prevent the identification of the accused by the victim. Throttling with the bare hands of the assailant or by strangulation with a ligature from material readily available at hand, e.g., nylon stocking, pantyhose, or the scarf, may cause death of the victim. Victims may commit suicide out of shame and stigma to the family.

   (b) Mental derangements, convulsions, and epileptic fits.

   (c) It may disrupt the victim's physical and sexual life. It can affect her future.

   (d) Due to gross injury of the vagina1, she may suffer from vaginal stricture or recto-vaginal fistula.

   (e) **Rape trauma syndrome** [3]

   A **psychological response in** an adult victim of rape can be described in two stages:
   - An immediate or an acute (disorganization) phase: It is characterized by emotional reactions like shock, loss of strength/courage, and anxiety. It is also associated with the feeling of guilt and humiliation.
   - A long-term (reorganization) phase: As the name suggests, it is the period during which the victim gradually readjusts her life. At times she may complain of nightmares (bad horrible dreams) and various phobias.

   (f) Additional mental trauma is caused by pregnancy resulting out of rape.

   (g) False charges [2].

There are cases of false charges made by the consenting woman when the sexual intercourse act is discovered by parents/husband, to save her position or when she herself discovers that she is pregnant or for purposes of taking revenge or for blackmailing and forcing him to marry her. Adult women may stain their garments with a solution of starch or white of egg to simulate seminal stains and with the blood of an animal. Similarly the parents themselves may injure the child's genital organs by introducing a blunt instrument or a thumb in to vagina or apply irritants such as chillies within vagina with the purpose of substantiating a false allegation of rape against an individual with a view to take revenge or extorting money from him (their child is trained to tell a circumstantial story of rape).

Nature of allegations [7]: False allegations contain common elements , such as the following: (1) Assailant was a total stranger or unknown person, because of which she can describe in vague and nonspecific terms. (2) She cannot describe the assailant because she kept her eyes closed. (3) She was assaulted by more than one person difficult to describe. (4) She offered resistance but was forcibly overcome as the assailant was strong or powerful. (5) And she cannot describe details and sequence of the sexual activities. Because of these complexities in the history taking and further investigation procedure, it has been very aptly said, **"Rape is an allegation, easily made—hard to prove and harder to disprove** [6]."

A female patient may put a false charge of rape against the doctor; therefore a female nurse/attendant should always be present during examination of genitals, during administration of general anesthesia, and during any surgery on female [8].

Evidence: All such false allegations, are finally rejected by medical evidence, inconsistencies in the statement of the victim herself, to uncertainty as to consent, to lack of corroboration, absent definite laboratory findings. Whenever there is undue delay in filing the FIR, always look at it with suspicion since there is a possibility of false allegations.

After having done proper examination of the alleged victim/accused and studying the laboratory reports of the materials collected, one can definitely find relation between the accused, the victim, and offence.

10. **Relationship between the accused, the offence, and the victim** [1]:
    (a) Marks of struggle/resistance, on the body of the victim as well as the accused (may be absent).
    (b) Hymenal rupture in the victim and rupture of frenulum in the accused (may not be found).
    (c) The pubic hair available on the body of the victim if matches with the pubic hair of the accused and vice versa.
    (d) DNA profile and blood group factor detected from the blood/blood stains, seminal/saliva/blood stains on the clothes, and tissue debris from the nails of the victim matches with the DNA profile and blood group of the accused.
    (e) Teeth bite mark pattern on the body of one matches with the other.
    (f) Presence of seminal fluid in the vagina of the victim and presence of vaginal cells and absence of smegma on the glans of the accused.
    (g) Garments of both the victim and the accused stained with soil and mud of the complained place of occurrence.
    (h) Presence of gonorrhea in the accused and the victim.

The co-relationship stresses the importance of meticulous medicolegal examination of both the alleged victim, accused, and above all the role played by forensic laboratory.

Guidelines and protocols for medicolegal care for survivors/victims of sexual assault are published by the Ministry of Health and Family Welfare, Government of India. Its Part II has given the following instructions for doctors:

53.3 One-Page Instructions for Doctors [9]

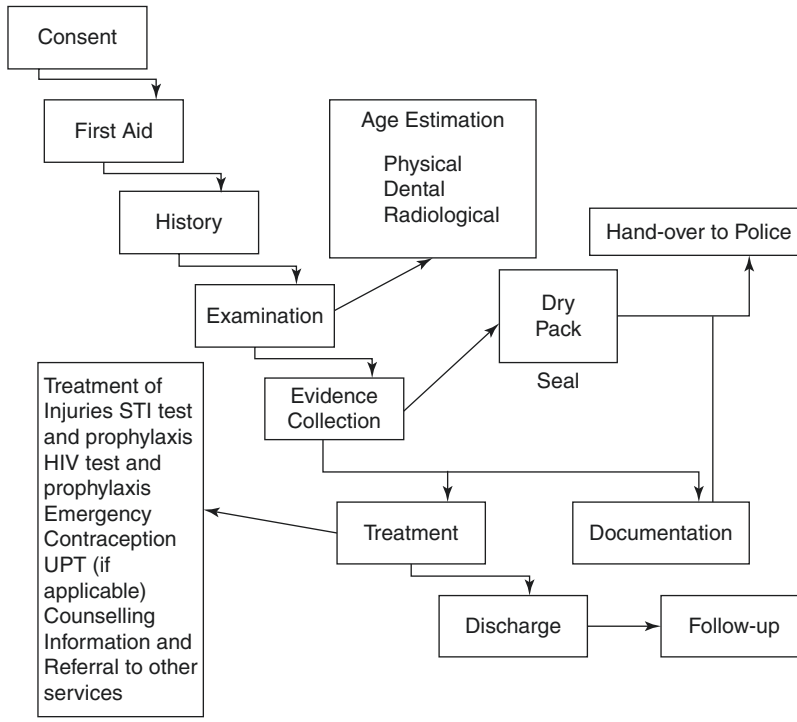
The healthcare provider who has taken history and examined the alleged victim should be well aware of the guidelines issued by the MoHFW and the comprehensive care to be provided to the survivor of the sexual assault.

1. **Informed consent:** Doctors should take informed consent of the alleged victim about the nature and purpose of examination and of the child's parent/guardian/person in whom the child reposes trust. This information should include:
    - The medicolegal examination assists the investigation, arrest, and prosecution of those who committed the sexual offence.
    - Forensic evidence may be collected with the consent of the survivor. This may include removing and isolating clothing, scalp hair, foreign substances from the body, saliva, pubic hair, samples taken from the vagina, anus, rectum, and mouth and collecting a blood sample.
    - The survivor or the parent/guardian/person whom the child reposes trust has the right to refuse either a medicolegal examination or collection of evidence or both. But that should not deny treatment to survivor.
    - Law requires that the hospital/examining doctor should inform the police about the sexual offence. However, one should continue to treat the survivor even if she does not wish to participate in the police investigation. Informed refusal will be documented in such cases.

2. "Two-finger test" must not be conducted for establishing an incident of sexual violence, and no comment on the size, elasticity of the vagina or hymen, or about past sexual experience or habituation to sexual intercourse should be made as it has no bearing on a case of sexual violence. No comment on shape, size, and/or elasticity of the anal opening or about previous sexual experience or habituation to anal intercourse should be made.
3. Examine the body parts for sexual violence-related findings (such as injuries, bleeding, swelling, tenderness, discharge). This includes both micro mucosal injuries and severe injuries which would take longer to heal.
   - Injuries must be recorded with details.
   - If a past history of sexual violence is reported, then record relevant findings. Though sexual violence is largely perpetrated against females, it can also be perpetrated against males, transgender, and intersex persons.
4. The nature of forensic evidence collected will be determined by nature of sexual violence and time lapsed between incident of sexual violence and examination and whether survivor has bathed or washed herself.
5. **Opinion**: The issue of whether an incident of rape/sexual assault occurred is a legal issue and not a medical diagnosis. Consequently, only findings in relation to medical findings should be recorded in the medical report without drawing conclusion.

   - Drafting of provisional opinion should be done immediately after examination of the survivor.
   - It should be always kept in mind that normal examination findings neither refute nor confirm sexual violence. Hence circumstantial/other evidences may be taken into consideration.
   - The injuries may not be evident due to inability of survivor to offer resistance because of intoxication or threats or delay in reporting for examination.

All the above steps of history taking, examination, and evidence collection are shown in the flow chart [9].



53.4 Conclusion

As said earlier, sexual assault is a criminal offence to be investigated by the police. Therefore medical professional must report all suspected cases, and failure to report may result in a penalty. The duty of medical professional is to illicit proper history; collect the best possible specimens in time, in optimum condition; and treat and above all support the victim. Injuries often speak for themselves. Following the investigation protocol along with proper documentation and the gathered evidence will definitely increase the value of medical evaluation of sexual violence. Good compassionate care along with prophylaxis and proper management of pregnancy and infection coupled with effective investigation and prosecution of the culprit will be very much beneficial to the victim.

Examination in the Case of Rape Victim [8]
(Report of the Victim of Rape)
Medicolegal Case No.....
To
The Investigating Officer.....
Ref: Your letter no.....dated..... in relation to
Crime..... {mention Sec. of I.P.C./Cr P C}.
I have the honor to forward herewith the result of my examination of.....
Daughter/wife of .....resident of ..... P.S.
.....Tahsil..... District.....
Brought to hospital on.....at.....
Patient examined on.....at.....
Consent for examination {if patient is less than 12, consent from guardian is taken}.....
Question asked.....
Replies given.....
Signature or left thumb impression of person.....
Identification marks (1).....(2).....
Brought and identified by police constable .....
No. .....Police station.....
The patient was examined and treated on OPD basis or was admitted on .....and discharged on .....
Light arrangement.....
Female nurse/attendant/relative present, her name.....
And her signature.....
History of the Incidence
Ask the victim to narrate the history in her words.

Any history of occupation, educational status, pelvic surgeries, sex life, pregnancy, deliveries and L.M.P., etc. {if she is menstruating, ask her to come again for second examination if necessary}.

Clothings

[Detailed examination of clothes is needed, if the same were worn at the time of the incidence. After examination of the clothes on her body, she should be asked to remove her clothes and then the clothes are again examined].

Look for:

(a) Stains
(b) Foreign body – hair/fiber/grass/mud/dust, etc.
(c) Tears
(d) Loss of buttons or hooks, etc.

General Examination

1. Any foreign body on her body
2. Any stains on the body
3. Evidence of struggle/resistance
4. Injuries on the body [abrasions, contusions, bite marks]
5. Examination of the fingernails [nail clippings to be preserved and sent to FSL]
   (a) Any epithelial cells
   (b) Any stain
   (c) Any damage
   (d) Any foreign body
6. Physical strength—height/weight
7. Chappals/ornamentals and glass bangles, etc.
8. Mental status
9. Intoxication
10. Gait
11. Age
    (a) As claimed by her
    (b) Secondary sexual characters
    (c) General examination
    (d) X-ray examination
    (e) Teeth

Local Examination

(Examine in lithotomy or knee-chest position in good light. If there is pain, local anesthesia should be applied.)

(a) Matting of pubic hair
(b) Local hygiene
(c) Any foreign body
(d) Any status
(e) Any injury to perineum
(f) Labia majora
(g) Labia minora
(h) Vestibule
(i) Hymen – intact/torn/position and age of tear
(j) Vagina
(k) Evidence of STD
(l) Any discharge
(m) Evidence of spermatozoa in the vagina
(n) Evidence of smegma in the vagina

Laboratory Investigations

The following material should be collected and sent to forensic science laboratory [FSL]:

(a) Vaginal swab for evidence of spermatozoa, blood cells, pus, etc.
(b) Vaginal fluid [for acid phosphatase estimation]
(c) Smear from the urethra for gonococci
(d) Any stain/foreign body found on the clothes or body
(e) Nails
(f) Blood for VDRL, blood grouping, and alcohol
(g) Anal swab in the case of sodomy
(h) Buccal swab in the case of buccal coitus
(i) Swab from bite marks

***DNA profiling helps link the victim and the accused**.

Opinion

[Give opinion about sexual intercourse, signs of struggle, and the injuries found. Never comment on rape as it is not a medical diagnosis].

Date: ..... Signature of Dr. ... .

Place: ..... Name of Dr. ....

Time: ..... Designation ....

Seal: .....

References

1. Sexual offences and sex perversions. In: Apurba N, editor. Principles of forensic medicine including toxicology. 3rd ed. Kolkata: New Central Book Agency(P) Ltd; 2010. p. 687–702.
2. Sexual offences. In: Narayan RKS, editor. The synopsis of forensic medicine and toxicology. 18th ed. Hyderabad: K.Suguna Devi; 2004. p. 182–188.
3. Sexual offences. In: Kumar A, editor. Text book of forensic medicine (medical jurisprudence and toxicology. 1st ed. Kala Amb: Avichal Publishing Company; 2014. p. 231–235.
4. http://www.firstpost.com/india/rape-examination-in-india-checklist-and-suggestions-lJan4 2013. Accessed 4 July 2016.
5. https://en.wikipedia.org/wiki/Virginity_test. Accessed 28 July 2016.
6. Natural sexual offences. In: Parikh CK, editor. Section V in Parikhs textbook of medical jurisprudence forensic medicine and toxicology. 6th ed. New Delhi: CBS Publishers & Distributors; 2000. p. 533–543.
7. Sexual offences. In: Narayan RKS, editor. The essentials of forensic medicine and toxicology. 29th ed. Hyderabad: K.Suguna Devi; ; 2010. p. 358–368.
8. Sexual offences. In: Singhal SK, editor. Singhal's forensic medicine & jurisprudence. 4th ed. Mumbai: The National Book Depot; 2010. p. 288–290.
9. Medico-legal care for survivors/victims of sexual violence: Part II: Guidelines and protocols. Ministry of Health and Family Welfare Government of India. Accessed 17 Nov 17

Shock in Obstetrics

54

Rajesh Kumar Verma, Rohini Rao,
and Kunal Kumar Sharma

SHOCK—defined as failure of circulation which is life threatening. It is characterised by low perfusion of vital organs due to low cardiac output.

Types

1. Hypovolaemic—due to body fluid or blood loss
2. Cardiogenic—direct damage to the heart
3. Extracardiac obstructive—obstruction to blood flow
4. Distributive—abnormal distribution of blood flow

Shock in Obstetrics

Haemorrhagic shock (most common):

(a) Haemorrhage in antenatal period during the first trimester
(b) Antepartum haemorrhage
(c) Postpartum haemorrhage

Neurogenic shock:

(a) Ruptured ectopic pregnancy.
(b) Concealed intrauterine haemorrhage.
(c) Forceps delivery or breech delivery in incompletely dilated cervix.
(d) During internal version of foetus.
(e) Crédé's method.
(f) Uterine rupture.
(g) Uterine inversion.
(h) Splanchnic shock—Seen due to accumulation of blood in splanchnic area after sudden emptying of the uterine cavity, e.g. rupture of membranes in a patient of polyhydramnios.

Cardiogenic shock: It is observed in conditions in which cardiac myocytes are unable to generate adequate stroke volume due to lack of efficient contraction, myocardial infarction and cardiac failure.

Endotoxic shock: It occurs due to toxins that precipitate vascular disturbance.

Anaphylactic shock: It is seen in hypersensitivity reactions to drugs.

Other causes:

(a) Embolism: amniotic fluid, air or thrombus
(b) Mendelson's syndrome

Note: A patient can present with shock due to multifactorial aetiologies, e.g. incomplete abortion leading to haemorrhagic and endotoxic shock, whereas ruptured ectopic and uterine rupture eventually lead to haemorrhagic and neurogenic shock.

R. K. Verma (✉)
Department of Anaesthesia, IGMC,
Shimla, Himachal Pradesh, India

R. Rao
Department of Obstetrics & Gynecology, IGMC,
Shimla, Himachal Pradesh, India

K. K. Sharma
Civil Hospital Sunni,
Shimla, Himachal Pradesh, India

A. Sharma (ed.), *Labour Room Emergencies*, https://doi.org/10.1007/978-981-10-4953-8_54

Haemorrhagic shock in obstetrics is due to antepartum or postpartum haemorrhage. Haemorrhagic shock is classified as in Table 54.1:

A more detailed parameter-based ATLS classification of shock is depicted in Table 54.2:

Measurement of surgical blood loss: Anaesthesiologists and obstetricians frequently underestimate blood loss. Massive blood loss leads to errors in judgement of estimation, which is responsible for inadequate replacement of intravascular volume. The young patients exhibit signs of hypotension and tachycardia after significant haemorrhage has already taken place. Haemodynamic management requires continuous assessment of patient status by clinical assessment, by classical monitoring equipment and by microprocessor-enabled monitors like Flotrac Vigileo™. The blood loss measurement can be done by the following methods:

Visual assessment:

Direct measurement of blood collected in calibrated drapes.

Estimation of blood:

Fully soaked 4″ × 4″ sponge contains approximately 10 mL of blood.

Soaked laparotomy pads contain 100–150 mL of blood.

Measurement of blood in suction canister after evacuation of amniotic fluid/dilutional saline (Fig. 54.1).

Laboratory evaluation:

Serial haemoglobin/haematocrit measurements—It basically reflects ratio of blood loss to plasma. Used for cases where the operative procedure is for long duration.

Gravimetric method:

Blood loss is estimated by gain in weight of swabs and towels after soakage with blood.

Table 54.1 Categories of shock

Category	Whole blood volume loss %	Pathophysiology
Mild (compensated)	<20%	Peripheral vasoconstriction to preserve cerebral and coronary blood flow
Moderate	20–40%	Decreased perfusion of kidneys, intestine and pancreas
Severe (uncompensated)	>40%	Decreased coronary and cerebral perfusion

Fig. 54.1 Disposable calibrated drape

Table 54.2 Advanced trauma life support (ATLS) classification of shock

	Class 1	Class 2	Class 3	Class 4
Blood loss (%)	<15	15–30	30–40	>40
Heart rate (beats/min)	<100	>100	>120	>140
Systolic blood pressure (mmHg)	Normal	Normal	Decreased	Decreased
Pulse pressure	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate (breaths/min)	14–20	20–30	30–40	>35
Mental state	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic

However, it underestimates blood loss by 25% because it doesn't take into account the blood loss by evaporation, blood lost in crevices of floor and blood collected in body cavities. Whereas, overestimation can also occur if the swabs also get soaked with pus, urine and irrigational fluids.

Colorimetric method:

Swabs, sponges and towels are mixed thoroughly with known volume of fluid. The change in optical density of water is measured at the isosbestic absorption wavelength of haemoglobin.

Radioactive tracer dilution method [1]: Either patient's RBCs are labelled with Cr^{51} or pooled human albumin labelled with I^{125} is used. The activity of tracer is first measured and then injected intravenously. The activity remaining in the syringe is measured and it is deducted from the amount of isotope injected. After 15 min, sample is drawn from opposite arm, and its activity is measured to estimate the change in blood volume. Repeated measurements over time reflect the changes in blood volume intraoperatively (Table 54.3).

Transfusion trigger is the haemoglobin level below which transfusion of RBCs is indicated. Various guidelines are as follows:

Habibi recommendations—Transfuse blood if:

- Hb ≤ 8 g/dL in all types of patients
- Hb ≤ 10 g/dL in patient of ischaemic heart disease, emphysema
- Hb ≤ 10 g/dL with autologous blood
- Hb ≤ 12 g/dL in ventilator-dependent patient

If blood loss >20% blood volume in adults or ≤100 mL in paediatric patients

Herbert recommendations—Transfuse blood if:

- Hb ≤ 10 g/dL in unstable angina and anterior wall MI
- Hb levels between 10 and 12 g/dL in critically ill patients

$$\text{Blood loss} = \frac{\text{Colorimeter reading} \times \text{volume of solution}}{200 \times \text{patient's Hb}\left(\text{g/dL}\right)}$$

ASA 1996 guidelines:

Transfusion of blood just based on Hb levels without considering other conditions is not indicated. Whenever possible, use acute normovolaemic haemodilution, preoperative autologous blood donation and post-op modifications to reduce blood loss. Transfusion is RARELY required if Hb is >10 g/dL, whereas it is ALWAYS required if Hb is <6 g/dL. If Hb is between 6 and 10 g/dL, the blood transfusion depends on complications of inadequate oxygenation and cardiovascular status.

The American College of Surgeons Trauma Committee's Advanced Trauma Life Support (ATLS) system classifies hypovolaemic shock into four stages based on the volume of haemorrhage. Obstetric patients are often mildly tachycardiac. However, tachycardia of more than 120 beats per minute is exhibited when the patient has had a blood loss amounting to 30–40% of her

Table 54.3 American College of Surgeons Advanced Trauma Life Support Classification of Haemorrhage Severity

Haemorrhage severity	Class 1	Class 2	Class 3	Class 4
Blood loss (mL)	< 750	750–1500	1500–2000	>2000
Pulse rate (per minute)	<100	>100	>120	>140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure (mmHg)	Normal	Decreased	Decreased	Decreased
Respiratory rate (per minute)	14–20	20–30	30–40	>40
Urine output (mL/h)	>30	20–30	5–15	Negligible
Central nervous system (mental status)	Slightly anxious	Mildly anxious	Anxious confused	Lethargic

Table 54.4 Trigger thresholds for MEOWS parameters

	Red trigger	Yellow trigger
Temperature (°C)	<35 or >38	35–36
Systolic blood pressure (mmHg)	<90 or >160	150–160 or 90–100
Diastolic blood pressure (mmHg)	>100	90–100
Respiratory rate (breaths/min)	<10 or >30	21–30
Oxygen saturation (%)	<95	–
Pain score[a]	–	2–3
Neurologic response	Unresponsive, pain	Voice

[a]Pain score: 0 = no pain at rest or movement; 1 = no pain at rest, slight pain on movement; 2 = intermittent pain at rest, moderate pain on movement; 3 = moderate pain at rest, severe pain on movement

total blood volume. Change in blood pressure and neurological status occurs later.

The modified early obstetric warning system (MEOWS) was established in the backdrop of this knowledge, with the primary aim to detect impending adverse events in a patient of shock. MEOWS is not specific to haemorrhage, but it has a positive predictive value of 39% and a negative predictive value of 98% in predicting maternal morbidity [2] (Table 54.4).

The rate of development of coagulopathy in obstetric haemorrhage is another major concern. The pathophysiology involves rapid consumption of coagulation factors, like fibrinogen. Postpartum haemorrhage and placental abruption trigger coagulopathy which is not in proportion to the amount of haemorrhage that has occurred in the patient. Once coagulopathy develops, the requirement for additional resources like blood products increases; therefore, the institutional protocol in such a situation must be clearly defined in advance in order to mobilize personnel and resources. Team practice drills of using this protocol have shown to decrease the severity of haemorrhage and mortality.

The AABB recommends limited use of transfusion after moderate haemorrhage in a haemodynamically stable adult patient. This advice takes into account the risks of anaemia while comparing it with the risks of transfusion.

The haemoglobin concentration determines the oxygen content in the blood and also the oxygen delivery to the tissues. However, compensatory physiologic responses offset the negative effect of anaemia on oxygen transport. There occurs an increase in cardiac output by combined effect of tachycardia and raised stroke volume. The blood viscosity and systemic vascular resistance decrease, thereby augmenting blood flow to tissues. Also, there occurs an increase in tissue oxygen extraction. As haemoglobin concentration falls to 5 g/dL, the systemic vascular resistance decreases, whereas the heart rate, stroke volume and cardiac index increase. The rate of oxygen transport and mixed venous oxyhaemoglobin saturation decrease at a haemoglobin level of 5.0 g/dL.

Risks of blood transfusion include haemolytic reactions, transfusion-associated circulatory overload (TACO), transfusion-related acute lung injury (TRALI), transfusion-related immunomodulation (TRIM) and bacterial and viral infections.

54.1 Management of Haemorrhagic Shock in Obstetric Patient

 # 1 Patient preparation:

- Two large-bore 14–16 G IV canula
- Oxygen at 6–8 L/min via facemask
- CVP and IBP access
- Foley's catheterization
- Correct hypovolemia by crystalloids or colloids
- Arrange blood
- Correct coagulopathy by FFP, cryoprecipitate, platelet concentrate, NovoSeven
- Emergency coagulation profiling of the patient
- Anti-aspiration prophylaxis

#2 Control the bleeding:
Surgical management

- Uterine tamponade by Sengstaken-Blakemore catheter or bimanual compression
- B-Lynch compression sutures

- Radiological embolization with gelatin sponge, polyurethane foam or polyvinyl methyl alcohol particle
- Arterial ligation of uterine artery, ovarian artery or internal iliac artery
- Emergency hysterectomy if above measures fail

Anaesthetic management: General anaesthesia is preferred for patient with uncontrolled haemorrhage, coagulopathy and foetal distress. Rapid sequence induction of anaesthesia with ketamine or etomidate using smaller-sized endotracheal tube is advocated. Maintenance of anaesthesia with TIVA technique using ketamine and propofol achieves optimal results in patients with uncontrolled bleeding. Once bleeding gets controlled, balanced anaesthesia technique using volatile agents can be started. FiO_2 is kept at 100% initially, thereafter as per the maternal tolerance; nitrous oxide or isoflurane 0.5% is started. Pancuronium is preferred for patient with obstetric haemorrhage in shock. Cisatracurium is preferred over atracurium, especially if the patient has shock associated with acute renal failure. Vecuronium is preferred in uncomplicated case presenting only with massive blood loss. Fentanyl 5–10 μg/kg provides adequate analgesia to such patients. In order to provide uterine relaxation during dilatation and curettage, use of volatile anaesthetic agents or intravenous nitroglycerin is advocated.

Uterotonic therapy is given by the following drugs:

Oxytocin: The convention of administering 10–40 IU infused in 1000 mL crystalloid solution at an unspecified rate can lead to catastrophic scenarios in these patients presenting with hypovolaemic haemorrhagic shock. Phenylephrine is a selective α_1 agonist, which when given in intermittent boluses of 25–50 mg mitigates the haemodynamic effects of oxytocin. The initial rate of oxytocin infusion is recommended to be 0.3 IU/min (i.e. ED_{90}). In case of inadequate response, the infusion rate should be increased to 0.6 IU/min (i.e. twice the ED_{90} value).

Methylergonovine: The recommended dose is 0.2 mg intramuscular. Bolus intravenous *administration is not recommended due to high incidence of nausea and vomiting. The duration of uterotonic effect is 2–4 h. Due to the production of tetanic uterine contractions, the use of methylergonovine is restricted in postpartum period. Side effects of this drug include hypertension, coronary vasospasm, myocardial ischaemia and seizures*.

Carboprost [15-methyl prostaglandin $F_2\alpha$]: It is a synthetic prostaglandin analogue which is given in a dose of 0.25 mg (250 μg) intramuscularly. This dose can be repeated every 15–30 min to a maximum cumulative dose of 2 mg. Its efficacy in reducing postpartum haemorrhage is well documented. Adverse effects associated with its use are bronchospasm, dyspnoea and hypoxaemia. These are seen in patients with coexisting disease like asthma. It is also not advocated to use it in patients with acute PID, cardiac disease and renal disease.

Misoprostol [prostaglandin E1 analogue]: The usual dose to treat patients presenting with postpartum haemorrhage is 600–1000 μg per rectum. Other routes are oral, buccal, vaginal and sublingual. This drug belongs to FDA category X for pregnant patients. Adverse effects like fever, chills, nausea, vomiting and diarrhoea can occur with its usage.

Intramyometrial PGE_2 0.5 mg or **$PGF_{2\alpha}$** 0.2 mg

Medical management [3]

- If Hb <9g/dL, start RBC transfusion until Hb levels increase more than 9 g/dL.
- Identify and correct platelet or coagulation abnormalities using Sonoclot or thromboelastogram.
- Measure CVP and keep its target goal to be >8 mmHg.
- Also keep MAP >65 mmHg.
- If MAP <65 mmHg but CVP >8 mmHg, start vasopressors like noradrenaline or dopamine.
- If CVP <8 mmHg, give fluid boluses with 0.9 NaCl or RL at 20 mL/kg.
- Alternatively noninvasive cardiac output monitoring devices like Flotrac shall help to determine whether the patient shall respond to iv fluids or to vasopressor support.

- Tranexamic acid 1 g iv bolus over 5 min can be repeated after 30–60 min only once.
- FFP: PRBC transfusion in the ratio 1:1.4
- Platelet transfusion if platelet count is <50,000/mm [3]
- NovoSeven 60–120 μg/kg iv [4]

Other measures

- Prevention of hypothermia
- Rewarming of fluids administered
- Cell salvaging devices
- Corticosteroids to tide over the stress response
- Meticulous correction of arterial blood gas parameters

Septic shock in obstetric patient

The mortality associated with this type of shock ranges from 30 to 50%. Due to the predominant rural population in India, many cases of gynaecological infections are underdiagnosed at peripheral institutes. Also the practice by quacks at the remote villages under lack of sterile equipment predisposes a large number of females for septic shock. The underlying pathophysiology in sepsis consists of a matrix of events that are associated with immune system and coagulation system.

54.2 Management [5, 6]

The patient presents with end-organ dysfunction along with systolic blood pressure less than 90 mmHg, mean arterial pressure less than 60 mmHg and blood lactate levels more than 4 mmol/L.

- Resuscitate the patient with crystalloids at 20 mL/kg.
- If systolic blood pressure is still less than 90 mmHg and mean arterial pressure is still less than 60 mmHg, insert a CVP line for guidance of fluid management.
- If CVP is <8 mmHg, repeat the fluid bolus of crystalloids at 20 mL/kg. Once the CVP is ≥8 mmHg, measure the mean arterial pressure.
- If mean arterial pressure is <60 mmHg, start vasopressors in the following dosages:
  - Noradrenaline at 0.05–0.5 μg/kg/min
  - Dopamine at 5–20 μg/kg/min
  - Adrenaline at 0.05–2 μg/kg/min
  - Phenylephrine at 2–10 μg/kg/min
  - Vasopressin at 0.04 units/min
- Once the mean arterial pressure becomes more than 60 mmHg, measure the central venous blood oxygen saturation ($ScvO_2$).
- If $ScvO_2$ is <70%, look at haematocrit. If haematocrit is <30, transfuse PRBCs.
- If cardiac index is <3.5 L/min/m^2, start dobutamine at 2.5–10 μg/kg/min.
- Once $ScvO_2$ is >70% but vasopressors are still required, look at the APACHE II score of the patient. If it is ≥25, administer activated Drotrecogin alfa.
- But if patient is in moribund state and with increased risk of bleeding, measure serum cortisol level.
- If serum cortisol is <15 μg/mL, you can consider corticosteroid replacement therapy. However, this therapy is unlikely to benefit if serum cortisol is more than 34 μg/mL.
- Initial empirical antibiotic management is based on the likely clinical presentation of the patient. Usually we start with a broad-spectrum antibiotic that covers most types of bacteria [7]. Other factors to be considered are the healthcare-associated infections and the community prevalent infections in the area. Once culture reports are available, the organism-based antibiotic regimen is started.
- The sooner the management of the patients starts, the more likely is their survival.

References

1. Bassingthwaighte JB, Holloway GA. Estimation of blood flow with radioactive tracers. Semin Nucl Med. 1976;6(2):141–61.
2. Singh S, McGlennan A, England A, Simons R. A validation study of the CEMACH recommended modified early obstetric warning system (MEOWS). Anaesthesia. 2012;67:12–8.

3. Nunez TC, Cotton BA. Transfusion therapy in haemorrhagic shock. Curr Opin Crit Care. 2009;15:536–41.
4. Stein DM, Dutton RP. Use of recombinant factor VII a in trauma. Curr Opin Crit Care. 2009;15:536–41.
5. Dellinger RP, Levy MM, Carlet JM, et al. Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock: 2008. Crit Care Med. 2008;36:296–327.
6. Rivers E, Nguyen B, Havstad S. Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med. 2001;345:1368–77.
7. Micek ST, Welch EC, Khan J. Empiric combination antibiotic therapy is associated with improved outcome against sepsis due to Gram-negative bacteria : a retrospective analysis. Antimicrob Agents Chemother. 2010;54:1742–8.