Purpose

This course arose out of our work in the care of women in labour, their babies, and their families. Our single overriding objective is to improve the outcome and the process of intrapartum care. One way to achieve that objective is through our continuing education. The ALARM course is one means of that education. The course is maintained and taught by family physicians, nurses, midwives and obstetricians. It has the administrative support and backing of the Society of Obstetricians and Gynaecologists of Canada. It is based on the best current evidence we have about what works to improve care, and incorporates Canadian practice guidelines.

The information and recommendations in this syllabus reflect the emerging clinical and scientific advances as of the date of issue and are subject to change without notice. The information should not be construed as dictating an exclusive course of treatment or procedure to be followed. Correct drug dosages should be verified before administration.
Recognition

This 26th edition of the ALARM Course Manual was revised under the direction of the Obstetrical Content Review (OCR) committee and the ALARM committee.

**Obstetrical Content Review Committee members – 26th Edition**
- P. James Ruiter (Co-Chair), MD, London, ON
- Suzanne Toni Wong (Co-Chair), MD, Toronto, ON
- Amanda Ashe, RM, Newcastle, ON
- Christine Bloch, MD, Stratford, ON
- Sharon Dore, RN, PhD, Burlington, ON
- Wesley Edwards, MD, Ottawa, ON (CAS* representative)
- William Ehman, MD, Nanaimo, BC
- Ronald George, MD, Halifax, NS (CAS* representative)
- Robert Gratton, MD, London, ON
- Jonathon Hey, MD, Saskatoon, SK
- Daniel Kiely, MD, Montreal, QC
- Andrew Kotaska, MD, Yellowknife, NT
- W. Kim MacDonald, MD, Garibaldi Highlands, BC
- Diane Sawchuck, RN, PhD, Vancouver, BC
- Karine Vallee-Pouliot, RM, Montreal, QC
- Megan Williams, MD, Ottawa, ON

*Canadian Anesthesiologists’ Society

**ALARM Committee members – 26th Edition**
- Catherine Cowal (Co-Chair), MD, Burlington, ON
- Marie-Jocelyne Martel (Co-Chair), MD, Saskatoon, SK
- Gisela Becker, RM, St. John’s, NL
- Fran Berard, MD, Winnipeg, MB
- Anne Biringer, MD, Toronto, ON
- Hayley Bos, MD, Victoria, BC
- Krista Cassell, MD, Charlottetown, PE
- Amy Gausvik, MD, Calgary, AB
- Narinder Kainth, RN, Markham, ON
- Stephanie Morel, MD, Montreal, QC
- Suzanne Roberge, MD, Baie-Comeau, QC
- Suzanne Roberts, MD, Saint John, NB
Disclaimer

SOGC has done its best effort to provide a product that is useful in terms of providing educational information based on an evaluation of scientific literature and medical experience. The educational content attempts to describe principles of practice generally applicable in most circumstances.

This document reflects emerging clinical and scientific advances on the date issued and is subject to change. The information should not be construed as dictating an exclusive course of treatment or procedure to be followed. Local institutions can dictate amendments to these opinions. They should be well documented if modified at the local level. None of these contents may be reproduced in any form without prior written permission of the publisher.

All people have the right and responsibility to make informed decisions about their care in partnership with their health care providers. In order to facilitate informed choice, patients should be provided with information and support that is evidence-based, culturally appropriate and tailored to their needs.

This document was written using language that places women at the centre of care. That said, the SOGC is committed to respecting the rights of all people – including transgender, gender non-binary, and intersex people – for whom the guideline may apply. We encourage healthcare providers to engage in respectful conversation with patients regarding their gender identity as a critical part of providing safe and appropriate care. The values, beliefs and individual needs of each patient and their family should be sought and the final decision about the care and treatment options chosen by the patient should be respected.
Suggested Reading

A variety of sources including standard textbooks and articles from the usual journals have been used in the development of the ALARM manual. These sources include but are not limited to:

7. Additionally, we have used two video tapes.
   a. *The Safe and Appropriate Use of Forceps in Modern Obstetrics* (available from Janssen-Ortho)
   b. *Term Breech Patient Selection and Intrapartum Management* (available from Wyeth Pharmaceuticals)
# Table of Contents

Chapter 1 Communication ................................................................. 9  
Chapter 2 Evidence-Based Obstetrics ........................................ 34  
Chapter 3 Patient Safety ............................................................... 45  
Chapter 4 Management of Labour ............................................... 64  
Chapter 5 Support and Pain Management in Labour ..................... 96  
Chapter 6 Induction of Labour ....................................................... 110  
Chapter 7 Umbilical Cord Prolapse ............................................. 145  
Chapter 8 Fetal Well-Being During Labour .............................. 151  
Chapter 9 Vaginal Birth ............................................................... 219  
Chapter 10 Assisted Vaginal Birth .............................................. 228  
Chapter 11 Delivery of Twins ....................................................... 260  
Chapter 12 Trial of Labour After Caesarean ............................. 276  
Chapter 13 Shoulder Dystocia ..................................................... 289  
Chapter 14 Breech Presentation and Delivery ............................ 309  
Chapter 15 Postpartum Hemorrhage .......................................... 333  
Chapter 16 Hypertensive Disorders of Pregnancy ....................... 375  
Chapter 17 Preterm Labour and Preterm Birth ......................... 412  
Chapter 18 Prelabour Rupture of Membranes (PROM) ............... 438  
Chapter 19 Antepartum and Intrapartum Hemorrhage ............... 454  
Chapter 20 Prevention of Early-Onset Neonatal Group B Streptococcal Disease .................................................. 481  
Chapter 21 Venous Thromboembolism and Amniotic Fluid Embolus ............................................................... 502
# Table of Contents

Chapter 1 Communication .................................................................................................................. 9  
  Strategies for Effective Communication ......................................................................................... 9  
  Strategies to foster Interprofessional Care and Planning ............................................................... 10  
  Use of Social Media ....................................................................................................................... 11  
  Special communication situations ................................................................................................ 12  
    1. Informed Consent ................................................................................................................... 12  
    2. Communication in the Consultation Process ........................................................................ 13  
    3. Communicating About Adverse Events ............................................................................... 14  
  Effect of Adverse Events on Parents, Families and Caregivers ..................................................... 15  
    Response and Coping Mechanisms ......................................................................................... 16  
    Follow-Up ................................................................................................................................. 18  
    Coping Mechanisms: Care Providers ...................................................................................... 19  
  Adverse Events and Litigation ..................................................................................................... 19  
  Disclosure .................................................................................................................................... 20  
    Barriers to Disclosure ............................................................................................................... 22  
    What do patients expect? .......................................................................................................... 22  
    Benefits of Disclosure .............................................................................................................. 23  
    How to Disclose ....................................................................................................................... 23  
    Event Reporting ....................................................................................................................... 24  
    Documentation ......................................................................................................................... 25  
    Electronic Medical Record (EMR) ........................................................................................... 26
Chapter 1
Communication

All levels of communication – intra and inter professional, with learners, with support staff, and with women and families are essential for team work, and effective team work is essential for patient safety.¹ A Cochrane review on interventions to promote collaboration between nurses and doctors showed that increasing collaboration improved outcomes of importance to patients and health care managers.² Ineffective communication was cited by The Joint Commission on Accreditation of Healthcare Organizations as the most common root cause of adverse events leading to perinatal death and injury between 1995 and 2014.³ An adverse event is defined as an unexpected occurrence that either causes harm or has the potential to cause harm.⁴ The 2018 review of HIROC and CMPA data showed breakdown in team communication occurred in 20% of cases and communication issues with patients and their families occurred in 4% of cases.⁵

Strategies for Effective Communication

- Mutual respect
- Language that is clear and precise – use defined and agreed upon words and abbreviations. eg:
  - When calling a professional colleague regarding a fetal heart rate tracing don’t say “I have an ugly tracing with some dippy parts” Say “I have an abnormal tracing with late decelerations occurring with 80% of contractions, a baseline of 160, minimal variability”.
  - Avoid making partial statements and assuming others know what you mean. This is called cognitive under specification, defined as “a communication style that leads to a gap in knowledge.”¹⁴ An example cited in an ICU was a nurse telling a physician that a patient had low potassium. The response was “let’s give him a run of 10 × 4.” The nurse entered the order into the computer for potassium chloride 10 meq. IV Q1 hr × 4 doses. This was what the resident intended, despite the lack of a stated drug name, complete dose, route, or schedule. If either the physician or the nurse had been new to the unit, a serious knowledge gap—and possibly an adverse event—could have occurred. It’s often difficult to recognize these gaps when they occur in our own domains.
  - When speaking to parents and families use terminology that everyone understands and use plain language rather than jargon. Patients and their families may not understand the medical terminology that makes communication with other team members clear and efficient.
- If messages or orders are hand written, they must be neat and legible
- Caution should be taken in individuals who speak a different primary language from the caregiver. In our multicultural societies, there is a need for capable interpreters. While families can be used for translation, caregivers need to be aware of potential conflict of interest or values between family members. Use of official translators or a telephone language line may be considered.
• Timely flow and transfer of relevant information; Use of acronyms to ensure timely, logical and complete information has been adopted by many health care facilities. Providing a structure for communication is effective in conveying a clear message. This format can be used in verbal communication as well as documentation. e.g.: SBAR.
  – Situation Take 5-10 Seconds to Explain
  – Background Provide Context and Data
  – Assessment Describe the Specific Problem/Situation
  – Recommendation Explain What You Want to Do About it and When

• Take the time the time needed to communicate in a clear, patient and respectful manner. This is particularly important if there has been an adverse event.
• Clear delineation of the roles of the communicators
• Respect for confidentiality
• Conducive environment:
  – Ambiance, emotional tone, privacy, and distractions can enhance or impede the quality of communication.
• Active listening is an important part of effective communication. It entails using eyes, ears, and brain, and seeking to understand before being understood. Effective listening demonstrates respect.
• Non-verbal messages are generally considered to be five times more influential than verbal messages. Eye contact is considered part of non-verbal communication. Non-verbal communication is often used unconsciously and may more accurately indicate a person’s meaning than the words being spoken. The tone of the words and inflections of speech are also major elements of communication. Many people lack insight into the way they use non-verbal communication, and reviewing videotaped simulations can help them increase their awareness.
• Include everyone the information affects

Professionalism in communication requires that caregivers understand and acknowledge the perspective of other professionals and the perspective of learners within each profession. The hierarchy that has traditionally existed in health care is not useful in a system that requires professionals from multiple professions to work as a team. No profession can function in isolation; members of each must call upon the expertise, skills, perspectives, and information of others to provide comprehensive, coordinated, and safe care.

Because learning to work within an interprofessional model of care is not part of basic training, many health care providers find it challenging; however, focussing on the patient and on quality of care makes the transition easier.

**Strategies to foster Interprofessional Care and Planning**

• Simulations for emergency situations involving all caregivers
  – Rehearsing responses to emergencies as a team using evidence-based, unit-specific protocols facilitates efficiency, performance, and effectiveness in real emergencies
  – Drills also provide an opportunity to practise effective communication between caregivers
• Committee structure that involves all professions
• Care planning that incorporates input from a variety of professions
Debriefing unusual events or normal practices as a team
• Non-punitive interprofessional case reviews
• Development of interprofessional procedures
• Investigating the impact of poor quality relationships as a barrier to improvements in communication and performance

Use of Social Media

Social media is increasingly becoming part of communication for health professionals, parents and families. Engagement with social media, for health-related and educational purposes, can be personal, professional, or both. However, it can raise questions related to confidentiality, ethics, privacy, and control to which we do not have clear answers. Health care values differ from social media values of sharing, openness and informality.

Social Media guidelines for health professionals have been created by professional organization. One example from oncology and vascular specialities suggest:

| Content credibility | • Only Share information from credible sources  
<table>
<thead>
<tr>
<th></th>
<th>• Refute inaccurate information</th>
</tr>
</thead>
</table>
| Legalities          | • Content you write may be used in court  
|                     | • Comply with privacy laws  
|                     | • Respect copyright laws |
| Networking          | • Do not contact patients with a request to join your network  
|                     | • Redirect patient who want to join your personal network to your professional site |
| Patient care        | • Avoid providing advice to non-patients  
|                     | • Make appropriate disclosures and disclaimers regarding the accuracy, timeliness, and privacy of electronic communications. |
| Patient privacy     | • Avoid writing about specific patients.  
|                     | • Make sure you are in compliance privacy laws.  
|                     | • Obtain patient consent when required.  
|                     | • Protect patient information through “de-identification.”  
|                     | • Use a respectful tone when discussing patients. |
| Personal privacy    | • Separate personal and professional profiles  
|                     | • Make sure that your credentials are correctly stated.  
|                     | • Specify whether or not you are representing an employer. |

Social media can be used for public or professional education and support. The Mayo Clinic has a Center for Social Media devoted offering patients a vast library of podcasts and blog posts written from health professionals ([http://socialmedia.mayoclinic.org/](http://socialmedia.mayoclinic.org/)). Facebook groups have been used by universities to provide a stress reduction intervention for medical students. The Canadian Association of Perinatal and Women’s Health Nurses (CAPWHN) has a discussion forum for
members to share information with colleagues across the country. Family Practice has a Family Physician Maternity & Newborn Care listserv (FPMNC).

When used appropriately, social media sites have the ability to promote health, as well as lay and professional education and understanding. However, when used carelessly, there are potential to pose negative professional and legal dangers consequences. Guidelines issued by health care and professional organizations and professional provide sound and useful principles that health care professionals should follow to avoid pitfalls.

Special communication situations

1. Informed Consent

Informed consent must be obtained to protect the patient's right to make decisions about treatment. The Canadian Medical Protective Association states that for consent to be considered valid:

- It must be voluntary: patients must not be coerced and must be free to consent to or refuse the proposed treatment or investigation
- Patients must have the capacity to consent: they must be able to understand the nature of the proposed treatment or investigation and its anticipated effect it will have, and the likely consequences of refusing the treatment or investigation

Patients must be properly informed: they should be informed of the diagnosis, and the proposed investigations and treatments and their risks and chances of success. They must also be informed about the consequences of refusing the proposed interventions.

Consent must be obtained by a caregiver who has knowledge of the procedure, possible side effects, and consequences. Alternatives to the treatment, if they exist, need to be presented and discussed.

For patients to provide consent, they must have the mental capacity or competency to provide consent, consent must be given voluntarily, must be informed, and must apply to a specific act or set of acts. The ultimate responsibility for ensuring informed choice and the mental capacity of the patient to provide consent, and for documenting a valid consent rests with the caregiver proposing and providing the intervention.

The patient is the primary decision-maker in the consent process. If she has decreased capacity for comprehension, the decision for intervention is based on prior informed consent if the patient's wishes are known. If her wishes are not known, the decision can be based on what is judged to be in her best interests. If time permits, discussion and decision making should be undertaken collaboratively by the family and health professionals. Care providers should be aware of the facility's algorithm for determining the substitute decision-maker if the patient is unable to give consent.

If there is refusal of consent or lack of timely agreement, care providers must ensure complete documentation of the discussion and woman's stated rationale for refusal. They must also maintain open communication and show courtesy and respect while continuing to offer appropriate care alternatives and treatments. The CMPA's Good Practices Guide provides further information and a review of key concepts.
2. Communication in the Consultation Process

Consultation is a crucial element of teamwork, and within the consultative process, all participants share responsibility for communication.¹⁹

<table>
<thead>
<tr>
<th>PARENT</th>
<th>REFERRING CAREGIVER</th>
<th>CONSULTANT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Demonstrate courtesy and respect</td>
<td>Demonstrate courtesy and respect</td>
<td>Demonstrate courtesy and respect</td>
</tr>
<tr>
<td>Participate in decision making</td>
<td>Assess patient before referral</td>
<td>Provide reasonable access to services</td>
</tr>
<tr>
<td>Ask questions if unsure of purpose of consultation, investigations, diagnosis, and risks and benefits of proposed treatment options</td>
<td>Communicate reason for consultation and level of consultation requested (opinion, opinion and shared care, or transfer of care)</td>
<td>Report hospital admissions and discharges Return patient care to referring caregiver when appropriate</td>
</tr>
<tr>
<td>Read relevant patient education material</td>
<td>Provide relevant documentation</td>
<td>Provide relevant documentation</td>
</tr>
<tr>
<td>Know which caregiver is responsible for care</td>
<td>Discuss and confirm with the whole team (which includes the patient) who will be the most responsible provider for current and ongoing care</td>
<td>Discuss and confirm with the whole team (which includes the patient) who will be the most responsible provider for current and ongoing care</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ensure the primary care provider is informed if unrelated problems are discovered so referral can be made to an appropriate specialist or sub-specialist.</td>
</tr>
</tbody>
</table>

A consultation can consist of (1) consultation only, (2) consultation with concurrent/shared care, or (3) consultation with transfer of care.

In a 2012 study, Kessler et al.²⁰ described a standardized approach, using the five Cs of consultation that increased the effectiveness of communication:

<table>
<thead>
<tr>
<th>Contact</th>
<th>Communicate</th>
<th>Core Question</th>
<th>Collaborate</th>
<th>Close the Loop</th>
</tr>
</thead>
<tbody>
<tr>
<td>Introduce consulting and consultant physicians. Build relationship.</td>
<td>Give a concise story and ask focused questions.</td>
<td>Have a specific question for or request of the consultant. Decide on reasonable timeframe for consultation.</td>
<td>Discuss with the referring care provider and the consultant any alteration of management or testing of patient’s status.</td>
<td>Ensure that both parties have the same understanding of the plan for the patient and that they maintain proper communication about any changes in the patient’s status.</td>
</tr>
</tbody>
</table>
The need for consultation may be dictated by professional standards or may be established by organizational or community resources. Consultations may involve social workers, dietitians, nurses, midwives, physicians, lactation consultants, community nurses, and many others. Professional bodies describe scope of practice for nurses, midwives, and physicians. The most appropriate course of treatment and referrals should be agreed upon, documented in the chart, and discussed with and understood by the patient. All parties should know which option has been selected and should be prepared to fulfill their responsibilities. The patient, her family, and the health care team must all know who is most responsible for the woman’s care, and this information must be recorded on the patient’s chart. Appropriate communication and fulfillment of responsibilities will improve patient care and satisfaction, care provider satisfaction, clarity of care-planning, quality of care, and patient safety.  

3. Communicating About Adverse Events

Care providers often have to break bad news to patients or families, frequently without having had time to prepare. They must communicate effectively and clearly, but also compassionately. The information they are conveying is life-changing for patients and families, who often remember for years what is said in such situations.

Obstetrical health care providers can find it difficult to talk to patients about adverse events such as miscarriage or termination, fetal anomalies, perinatal death, stillbirth, and the compromised infant. Patients and families are often dissatisfied with the type and amount of information they receive from their health care providers, and many health care providers do not feel confident in their ability to break bad news, but this skill, like other clinical skills, can be learned. Although there are no evidence-based guidelines for relaying bad news, qualitative and descriptive research indicates that appropriate disclosure is an effective risk management strategy.

Improved communication of bad news can reduce distress, enhance coping, and reduce the risk of unrealistic expectations, and inappropriate denial for the patient and her family, and can minimize stress for care providers.

Strategies for the Communication of Bad News

Robert Buckman and Walter Baile have suggested mnemonics for communicating bad news to parents and families:

<table>
<thead>
<tr>
<th>SPIKES: for breaking bad news</th>
<th>CONES: for discussing a medical error or sudden deterioration or death</th>
</tr>
</thead>
<tbody>
<tr>
<td>• S setting up the conversation = environment, planning what you will say, key people present</td>
<td>• C context = quiet area, patient closest to you, maintain eye contact</td>
</tr>
<tr>
<td>• P perception = assessing the patient’s understanding of their condition</td>
<td>• O opening shot = alert patient/family of important news</td>
</tr>
<tr>
<td>• I invitation = get permission to have the discussion; ask before you tell</td>
<td>• N narrative = explain the chronological sequence of events</td>
</tr>
<tr>
<td>• K knowledge = explain the facts, avoid medical jargon</td>
<td>• E emotions = address emotions with empathetic response</td>
</tr>
<tr>
<td>• E emotions = be supportive</td>
<td>• S strategy and summary = summarize discussion and make a plan; let them know the situation is a priority</td>
</tr>
<tr>
<td>• S strategy and summary = summarize the conversation and agree on a plan</td>
<td></td>
</tr>
</tbody>
</table>
Some examples of phrases that could be useful include:

<table>
<thead>
<tr>
<th>USEFUL TO SAY</th>
<th>NOT USEFUL TO SAY</th>
</tr>
</thead>
<tbody>
<tr>
<td>I’m sorry for your loss.</td>
<td>This happened for the best.</td>
</tr>
<tr>
<td>I wish things could have been different.</td>
<td>Time will heal.</td>
</tr>
<tr>
<td>I can sit here with you for (specified amount of time) if you’d like company.</td>
<td>Now you have an angel in heaven.</td>
</tr>
<tr>
<td>If you’re too overwhelmed to talk now, we can talk again later.</td>
<td>Mother Nature knows best.</td>
</tr>
<tr>
<td>If you want to talk about this, I can listen. If you have questions, I’ll try to answer them.</td>
<td>It was a blessing. He would never have been normal anyway.</td>
</tr>
<tr>
<td>Is there someone I can call for you?</td>
<td>You’re young. You can have others.</td>
</tr>
<tr>
<td>Would you like to talk to a counsellor? I can arrange that for (this afternoon/tomorrow/other specified time).</td>
<td>Better for this to have happened now, before you knew the baby.</td>
</tr>
<tr>
<td>Are there things we can find for you? (This could be something as pragmatic as forgotten reading glasses or as poignant as an item made for the dead baby.)</td>
<td>He was born dead. You didn’t have a chance to get attached to him.</td>
</tr>
<tr>
<td>Would you like to get out into the fresh air? We have a quiet garden nearby.</td>
<td>At least you have one already.</td>
</tr>
<tr>
<td>Is there anything you need?</td>
<td>You can have another baby right away.</td>
</tr>
<tr>
<td></td>
<td>Just thank God for your healthy children at home.</td>
</tr>
<tr>
<td></td>
<td>You have to get on with your life.</td>
</tr>
<tr>
<td></td>
<td>Better luck next time.</td>
</tr>
<tr>
<td></td>
<td>Practice makes perfect.</td>
</tr>
<tr>
<td></td>
<td>It’s God’s will.</td>
</tr>
</tbody>
</table>

**Effect of Adverse Events on Parents, Families and Caregivers**

Events occurring in early pregnancy can have a similar effect to those occurring later, although caregivers have historically considered later pregnancy events more significant. Maternal–fetal attachment was originally thought to begin with maternal perception of fetal movements; however, the literature indicates that attachment for many women begins as early as pregnancy symptoms appear and increases when the fetus is seen on ultrasound examination.
Partners also felt significantly enhanced attachment when they watched ultrasound images of the fetus. A woman who has had a negative outcome in a previous pregnancy may delay her attachment until later in the pregnancy.

Miscarriage is usually considered a distressing experience, regardless of the gestational age at which it occurs. The response of the patient and her family may be different when a termination is carried out because of fetal anomalies, and health care providers must be sensitive to this.

A survey of women who had experienced an early loss indicated that nearly three quarters experienced it as the death of their baby, believed they had caused the death of their baby, and felt emotionally and/or physically out of control. Many had nightmares, and most said they felt that part of them had died.

In a 2004 qualitative study, women reported characteristic symptoms of PTSD, such as flashbacks, persistent avoidance of stimuli associated with the trauma, anxiety, emotional detachment, and fear of future pregnancies.

When a miscarriage occurs or a termination is performed there are no legal requirements with respect to disposal of the products of conception, which are usually dealt with in accordance with institutional guidelines for surgical tissue/medical waste. For many women, the option of burial may provide some degree of closure. Care providers should also be sensitive to cultural differences, which may require other approaches to the management of loss.

Although some adverse events are clear (infant is stillborn), others are less so (infant is compromised), and when the extent of the problem or the long-term effects are not known, it is more difficult for clinicians to explain to the patients and more difficult for the patients to understand.

Interpretation of the Event

Personal, cultural, and social context influence how patients and families interpret events. For instance, if an adolescent with significant medical comorbidity delivers a stillborn infant at 24 weeks’ gestation, it’s likely that both the adolescent and her mother will experience mixed emotions. In addition to feeling loss, the mother, as her daughter’s support person, may also feel relief because of concerns about coping with a new baby and the potential harm to her daughter’s health.

Previous personal experience also affects interpretation. A successful outcome with a previous preterm birth will likely lead to optimism in the event of a second preterm birth, whereas a previous neonatal death may mean ongoing anxiety with the subsequent pregnancy.

Men and women may not experience grief in the same way and may have discordant coping mechanisms. Women generally have higher levels of grief and grieve longer. Perceived societal and cultural expectations regarding gender-specific expressions of grief may influence these differences.

Response and Coping Mechanisms

The individual and family response varies according to the nature of the event, interpretation of the event, and coping resources available. The way people cope with adverse events is influenced by their cultural traditions and expectations, their religious affiliations and beliefs, and their family structure. Health care providers should be aware that each
The patient will have her own definition of “family”, which may include colleagues and friends. They should also be aware of their own assumptions, and of the variety of responses to perinatal loss and grief they may encounter within the cultural and religious groups within their geographic area of practice. The ability to cope will also be affected by other events happening at the same time.

Elisabeth Kubler-Ross proposed 5 stages of grief and loss, denial, anger, bargaining, depression, and acceptance, but noted that these stages were not necessarily experienced in the same order or for the same duration. Grief may present in many ways and time frames. Responses may include mood disorders, social withdrawal, impaired memory or concentration, appetite changes, and sleep changes. It is also variable based on culture, on the nature of the event, coping resources, and interpretation of the event.

Up to 20% of parents will suffer psychological symptoms for years following the death of a baby. Potential markers for impaired psychological resolution include not seeing or holding the baby, unsupportive partner or family, and subsequent pregnancy.

Parents often are very anxious and angry and may blame health care providers. They often experience a loss of self-esteem and a sense of failure. They may have difficulty in coping with the immediate tasks such as funeral arrangements and death registration.

Siblings may be the forgotten mourners. It is often left to the parturient partner to share information with other children—their age, awareness of the pregnancy, and personal previous experiences affect their understanding and interpretation.

How Health Care Providers Can Help Patients to Cope

When a baby is sick or dying, it is important for care providers to acknowledge the problem and, in collaboration with neonatal care providers, explain the immediate care plan. If the baby is to be transported to another centre, care providers should explain the process to the parents and determine whether the mother can also be moved to be near the baby. The parents should be involved in important decisions (e.g., withdrawing life support), and care providers should visit both parents and newborn frequently. Care providers may also:

- Encourage parents to have as much contact with the baby as possible
- Allow parents to take the dying baby in their arms
- Respect the need for privacy
- Arrange ongoing care for the parents
- Make referrals that will minimize other potential stresses such as the need for accommodation, transportation, childcare, parental leave

Following a pregnancy loss or the loss of a newborn, parents will often benefit from having tangible mementos such as the following:

- Footprints and handprints (if no ink is available, iodine-based cleanser will work)
- Name band
- Crib card
- Lock of hair
• Bereavement outfits/receiving
• Written notes or poems to or for the baby
• Certificate of life/baptismal certificate
• Photographs:
  – Photographs of the baby might be important if mother and baby are to be separated
  – If the baby is dead, parents may not be able to view pictures of the baby immediately, but might later appreciate having been provided with them.
  – Parents’ expectations of the baby’s appearance may be worse than the reality; appropriate bundling and wrapping of the baby can be helpful to disguise anomalies. (It may be important for health care providers to demonstrate that they are comfortable with the baby.)
  – Photographers specializing in bereavement photography have developed special water-immersion techniques to enhance the quality of images provided to families following their loss.
• Planting trees or flowers
• Ornaments or decorations
• Scrapbooks of cards from friends and family

Health care providers should verify the parents’ cultural or religious preferences before cutting hair, taking photographs, or making other similar interventions.

There is no one right way to grieve, and while institutional protocols for bereavement can be helpful, loss is a complex human experience. Support for a family experiencing loss requires the same kind of flexibility and responsiveness on the part of care providers as other aspects of family-centred maternity care. Listen to the parents’ perceptions and invite their involvement in exploring what ways of coping will work best for them.

Referrals to hospital services such as social work or pastoral care or to community-based support groups can be helpful to families. Many hospitals hold group memorial services for families who have suffered a perinatal loss. Both caregivers and families have found these memorials helpful. Resources for the development of a bereavement program can be found in the Family Centred Maternity Care guideline.

Follow-Up

Let the patient and/or family know who will be available to answer their questions in future and whether you will continue to be available. It is valuable for the primary health care provider to make at least one return visit within 24 hours to allow further discussion and repetition of information. Your hospital’s disclosure policy may be helpful in determining who should be involved and what to say. Lack of communication is one of the most common complaints by women and their families.

It is recommended that obstetrical units have a protocol for the investigation and documentation of perinatal losses. This information gathered in an investigation will be useful in explaining the present event and in planning for future pregnancies. Documentation should include prenatal records, ultrasound reports, genetic testing (antenatal or post mortem), and clinical notes of the circumstances of loss, autopsy results, and any follow-up arrangements made. When the event is a perinatal loss, families should be informed that autopsy results might take several weeks or
even months to obtain. Counselling for future pregnancies is important but may need to be delayed until complete information is available.

**Coping Mechanisms: Care Providers**

The impact of adverse effects on care providers is also highly significant. Health care providers who deal with adverse events may experience feelings of sadness or guilt, depletion of emotional stamina, loss of professional self-esteem or prestige, or fear of litigation. They may not recognize their need for help and support, or they may be unwilling to request assistance. Care providers should talk to colleagues who have dealt with adverse events if they appear distressed. After dealing with adverse events, health care providers may psychologically distance themselves from patients, which can lead to burnout for the care provider and/or ineffective care for patients. Care providers in this situation should be offered the opportunity to debrief and be offered counselling, support, and acknowledgment of their feelings.

The term “second victim,” has been defined as a clinician who “feels personally responsible for the unexpected patient outcomes and feels as though they have failed their patient, second guessing their clinical skills and knowledge base.” According to Higgins, “Clinicians can cope by focusing either on the problem or on their emotions. Focusing on the problem involves learning from a mistake, seeking information, determining what transpired, and dealing with the problem. It is constructive to accept responsibility and to change personal practice.”

A 2011 observational study of nine intrapartum nurses who had participated in a traumatic delivery revealed that “the impact of an unexpected event can be emblazoned on one’s memory for many years, with an immediate response of secondary traumatic stress disorder symptoms.” This observation, while predictable and frequently documented in the women who experience traumatic birth, had not been previously documented in care providers.

Caring for ourselves is an essential and normal part of the process. It is acceptable to show emotions in the presence of death but our emotions should not take precedence over the emotions of the family involved. Feelings of guilt may result even when the management has been appropriate. When management has been less than ideal, it is important to participate honestly in a process of self- and peer-evaluation. The most effective form of risk management is caring for your patient and her family. Seeking out supportive colleagues with whom to acknowledge these feelings can be an effective way to deal with obstetrical crises.

**Adverse Events and Litigation**

The Canadian Medical Protective Association (CMPA) and the Healthcare Insurance Reciprocal of Canada (HIROC) collaborated with Accreditation Canada and Salus Global Corporation to profile the safety and quality of obstetrics services in Canada and also identifies opportunities for improvement. “The goal was to uncover contributing factors to patient safety incidents and identify additional mitigation strategies for providers and health care organizations. Ten years of HIROC data showed obstetrics represented 45% of liability costs and 46% of compensation payments.”
The Canadian Medical Protective Association (CMPA) provides written resource material and individual confidential counselling to physicians related to a concerning event even when no litigation has been enacted. Physicians may call CMPA to report their involvement with a concerning event.

The Canadian Nurse Protective Association (CNPA) offers legal advice, risk-management services, legal assistance and professional liability protection related to nursing practice in Canada. Nurses may be members of CNPA based on registration in their professional association Alberta, British Columbia, Manitoba, New Brunswick, Newfoundland and Labrador, Nova Scotia, Prince Edward Island, Northwest Territories, Nunavut, Saskatchewan or Yukon. Nurses in Ontario, Quebec or Manitoba have to join individually.

The Health Insurance Reciprocal of Canada (HIROC) is the insurer for Midwives and not-for-profit healthcare organizations. HIROC provides risk management services and educational events.

In CMPA data, 25% of cases were associated with intrapartum labour and 45% with intrapartum delivery; HIROC data showed 33% and 34% respectively. Key factors were:

- individual provider decision making and
- individual and team situational awareness and communication
- system issues such as hospital protocols, reduced capacity to respond to obstetrical emergencies

 Disclosure

According to a patient safety Dictionary, disclosure should be understood as the imparting, by health-care workers to patients or their significant others, of information pertaining to any health-care event affecting (or liable to affect) the patient’s interests. The obligation to disclose is proportional to the degree of actual harm to the patient (or the realistic threat of such) arising from an untoward event.61

Disclosure must be made in accordance with organizational policy and local legislation.

Errors that result in adverse events are inevitable, so it is prudent to have policies, protocols, and an organizational plan within which the health care providers will be trained to conduct disclosures.62 The plan must be specific to the circumstances of each individual organization; however, according to the American College of Obstetricians and Gynecologists, it should address the who, what, when, where, and how:

- **Who:** In general, disclosure should be made by the most-responsible care provider. The nature of the adverse event will likely determine what other members of the health care team should be present.
- **What:** Only factual information must be communicated to the patient. Patients must be reassured that as additional, reliable information is obtained, they will be notified promptly.
- **When:** Even if all details of the incident are not known, disclosure must be timely. Disclosure should occur as soon as reasonably possible, while emphasizing to patients that it is an ongoing process of communication.
- **Where:** Disclosure should occur in a quiet and confidential setting that will be most comfortable to the patient.
- **How:** Patient dignity must always be respected. A disclosure conversation should include empathy for what patients and their families have experienced.
Communication of any negative health outcome to a patient or family is difficult for caregivers. Disclosing an adverse event resulting from error is even more difficult. The normal response of a patient or family to injury (whether or not the harm was due to a mistake) includes a mix of fear, anxiety, depression, anger, isolation, humiliation, devaluation, and betrayal.

Lucien Leape has described serious preventable injury as a medical emergency that has two victims: the patient and the care provider. The patient suffers a double wound: the actual physical injury and an emotional wound, i.e., a sense of betrayal and loss of trust. The caregiver can experience profound shame, guilt, and fear resulting in an impaired ability to practise. The organization must be prepared to treat both emergencies in a timely fashion. The keys to treatment are honesty, openness, and apology.

A health care provider’s busy practice and unit may be limited by the inability to keep up and the honest desire for “things to be all right”. This may be misunderstood by the family as minimizing their concern by the team.

Health care professionals and the organizations within which they work have an ethical, fiduciary, professional, regulatory, and legal duty to honestly disclose adverse events to their patients and/or families. Most patients want and expect to be informed of adverse events but care providers are not necessarily in favour of disclosure

- 98% of patients want to be informed of even a minor error; the greater the severity of the outcome, the more they want information
- 92% of patients believe they should always be told about complications, while 68% of physicians believe patients should always be told
- 81% of patients believe they should be advised of possible future adverse outcomes of the complications, while only 33% of physicians believe that patients should be told about possible future outcomes.

A study by Gallagher et al., which explored attitudes and experiences of physicians regarding disclosure, showed wide variation in how they would disclose error to patients. The study suggested that disclosure standards and training are necessary to meet public expectations and promote professional responsibility following errors.

According to the Canadian Medical Protective Association, 8 Canadian provinces and one territory (British Columbia, Alberta, Saskatchewan, Manitoba, Ontario, Nova Scotia, Prince Edward Island, Newfoundland and Labrador, and Nunavut) have adopted “apology legislation.” This approach to disclosure is designed to create greater accountability and transparency in the health care system. Apology legislation:

- Allows individuals and organizations, such as hospitals and other public institutions, to apologize for an accident or wrongdoing, without the apology being used as evidence of liability in a civil legal proceeding under provincial law;
- Promotes accountability, transparency, and patient safety by allowing open and frank discussions between patients and health care providers;
- Reduces the burden on the justice system by encouraging non-litigious resolution of civil disputes.
Barriers to Disclosure

Caregivers may have personal beliefs, or may experience fears, that prevent their disclosing adverse events. They may believe that:

- Disclosure is unnecessary
- It is in the best interests of patients not to be informed
- The outcome would potentially have occurred without the error or intervention (e.g. the patient was terminally ill anyway)
- They lack the experience, skill, and training to communicate difficult information

They may fear:

- Retribution from the recipient of the news
- Loss of the trust and respect of patients
- Legal action
- Censure, loss of respect, and prestige among colleagues
- Loss of job and/or income
- Loss of self-esteem and a loss of self-confidence as a care provider
- Dealing with the emotions of the patient and/or family as well as their own

What do patients expect?

When an adverse event occurs, patients and/or their families are often aware that something has gone wrong. If the event is not disclosed, they may feel devalued and disrespected and may lose trust in the caregivers, the health care organization, or the system. If they feel that information is being purposely withheld or covered up, they may believe the only way they can obtain accurate, complete information is by launching a lawsuit. According to a 2004 report from the Canadian Institute for Health Information, 76% of respondents believed the threat of litigation was important to ensuring that doctors act in the best interests of their patients.

Patients or their families are most likely to consider legal action not because of an event but because of their subsequent interaction with people in the health care system. When the harm becomes apparent despite of the lack of disclosure, or when the adverse event is finally admitted by the caregiver and/or organization, patients and/or their families often experience profound anger. This compounds the trauma already being experienced and can lead to a desire to punish the caregivers or to seek revenge against the organization. Conversely, if patients and/or their families have been involved from the very beginning in all aspects of care and have worked in partnership with the caregivers, disclosure of adverse events, although still difficult, occurs within an established relationship based on open, honest, and transparent communication.

Specifically, patients need to know:

- How do we manage this for my family?
- What you are going to do to help me?
- What specific steps are you taking to ensure this doesn't happened to another family?
- What can we do to help you achieve those system changes?
Benefits of Disclosure

For patients and/or families:

- Begin to recover from the effect of the unanticipated outcome and deal directly with the pain so that they can begin to heal
- Regain trust and work out their feelings of distrust with the people inside the institution, rather than look for help from those outside it
- Understand and obtain the care that may be needed to address the effects of the adverse outcome in the future
- Receive the information needed to make decisions about the next steps, including the possibility of seeking appropriate compensation

For caregivers:

- Address the error, and make the changes necessary to prevent future occurrences
- Express regret, assuage guilt, and begin to heal
- Regain self-esteem and continue to practice

For health care organizations:

- Learn from events and improve faulty systems to protect patients, their families, and health care staff in the future
- Potentially lessen the frequency and severity of litigation through the proper management and control of a disclosure process
- Share with other organizations to prevent the recurrence of the event elsewhere

How to Disclose

Preparation

- Have an organizational plan that will guide the process and ensure all potential issues are addressed
- Review the facts; be sure you know clearly what happened
- Balance the need to have all the information with the need to disclose the information in a timely manner. It might be better to say “we do not know yet but we will tell you when we do” rather than wait and potentially alienate and antagonize the patient and her family
- Determine who needs to be told
- Determine each person’s role
- Assess the readiness (medically stable, awareness level, ability to comprehend, availability of support) of the patient and/or her family to hear what you have to say
- Choose an appropriate time and setting, keeping in mind the basic principles of effective communication and timely follow-up
- Ensure that the medical team are not seated on one side of the table and the family on the other, which may suggest an adversarial relationship.
Meeting with the patient and/or family

- Simply describe what happened in plain, commonly understood language; use a neutral tone
- Describe what is known; give factual, objective information
- Describe the next steps of the process for the patient and/or family; ensure any necessary medical care is identified and made available; offer to transfer care to another caregiver
- Acknowledge the patient’s suffering and express your sympathy
- Apologize for the harm caused to the patient and for your role in it
- Focus on the needs of the patient and/or family
- Describe the next steps in the investigative process
- Explain what is being done to prevent a recurrence
- Seek and respond honestly to the questions and concerns of the patient and/or family; allow ample time for questions
- Establish, with the patient and/or family, a plan for follow-up (detailing who, what, where, when)
- Provide a contact person, who will be available indefinitely, and the support of other available resources (clergy, social services, etc.)

Tasks

- Establish rapport
- Listen actively and empathetically
- Try not to be defensive: this is the time to listen, not to defend your actions
- Recognize and manage your own feelings
- Be open and willing to accept whatever reaction occurs
- Anticipate and be prepared to respond calmly to emotional reactions and behaviours such as crying, yelling, anger, threats, verbal abuse, walking out

Event Reporting

An adverse event is defined as an unexpected occurrence that either caused harm or had the potential to cause harm.

Disclosure of an adverse event also involves timely and accurate reporting of the event (whether harm was caused or not) to the facility in which the care provider works, as well as to their insurers organizations.

Confusion about whether or not a “complication” is an event is one reason for failure to report or delay in doing so, which can have serious consequences. Waters et al. list situations in which an event may or may not be reported. They note that “Nurses exercise considerable judgment in deciding whether or not to formally report an incident, with estimated rates ranging from 10% to 55% of incidents.” They also note that culture plays an important role in reporting rates and that informal reporting occurs where “decisions to report informally or formally were influenced by the knowledge and experience of nurses; relationships with colleagues, physicians, and managers; types of errors; and workload.” Finally, they state that “lack of feedback from administrators about an incident reduces reporting. Time-consuming incident report processes and the inability to report anonymously also reduce incident reporting. Negative relationships amongst
health care providers, within and between disciplines, decrease incident reporting.” These observations are important as the majority of incidents are reported by the nursing profession.

**Documentation**

The principle “if is not documented it is not done” applies to all health care professionals. Thus documentation is essential for maintaining continuity of care and can form part of legal proceeding. The latter point is particularly significant as many legal cases occur years after the event when memories have faded.

The health profession needs to assess their own documentation to ensure key principles are adhered to.

To be effective, documentation must be:

- **Clear**: concise, precise, and legible
  - Legibility is a key factor in medication errors and understanding the plan of care. There is no harm in printing if cursive writing is not easily interpreted.
- **Complete**
  - Using a standardized template. One example is SOAP
    › S=subjective information (i.e. what the parent says)
    › O=Objective information (test results, physical exam)
    › A= assessment (analysis of the situation, diagnosis)
    › P=Plan of care (includes what was said to the parent /family, advice, planned actions)
  - Always include a time, date and signature on all documentation
- **Contemporaneous**
  - Contemporaneous documentation is challenging in emergencies. Having a designated recorder and a template form for urgent situations promotes quality documentation. Just as there is a flow sheet for Code Blue documentation, development of flow sheets for other critical situations such as PPH facilitates complete and timely documentation.
- **Consistent**
  - With other multi-disciplinary notes and records
- **Compliant** with institutional and professional standards

Clark et al. reported that in 54% of legal cases involving shoulder dystocia, the failure to clearly document the management of the dystocia was the primary reason for payment of damages. Often the right things were done but were not documented adequately. A quick, effective system to document procedures and events and excellent verbal communication would likely minimize litigation.

The MORE® Case Management Guides provide suitable templates. The Vacuum Case Management Guide is appended to this chapter as an example. When this approach is used by an interprofessional team (e.g., physician and nurse) after a procedure or delivery, it leads to an effective debriefing of the interaction fostering debriefing as a routine part of practice.
Electronic Medical Record (EMR)

Facilities have increasingly implemented a variety of electronic medical records for either partial or full documentation. The implementation requires time, money, and consistency in care practice as changes in EMR may be difficult. EMR has been variously embraced and challenged by different specialities and different generations of health professionals. The lack of use of a consistent EMR within a region or city poses additional challenges.

<table>
<thead>
<tr>
<th>STRENGTHS</th>
<th>LIMITATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased legibility</td>
<td>Health professionals require education on system use and potentially typing skills</td>
</tr>
<tr>
<td>A time is always included</td>
<td>Repetitive, less individualized, comments with drop down boxes</td>
</tr>
<tr>
<td>Easily retained and printed out</td>
<td>Comments may be in multiple locations- comments may be inconsistently placed</td>
</tr>
<tr>
<td>Easily accessible at multiple locations if the same EMR is used</td>
<td>When working more than 1 facility with different EMR systems, it is challenging to remember the different EMR systems and rules</td>
</tr>
<tr>
<td>Research date more easily obtained</td>
<td>Passwords can expire if not frequently using a given facility EMR</td>
</tr>
<tr>
<td>Positive environmental impact</td>
<td>Expensive</td>
</tr>
<tr>
<td></td>
<td>Security</td>
</tr>
</tbody>
</table>
References


76. Thompson J. Apology Act passes third reading at Queen's Park [news release]. Toronto: HIROC; 2009.


Table of Contents

Chapter 2 Evidence-Based Obstetrics ........................................................................................................... 34
  Types of Evidence: Qualitative or Quantitative ......................................................................................... 34
    Meta-Analysis ........................................................................................................................................... 35
    Caution ...................................................................................................................................................... 35
    Statistics .................................................................................................................................................. 35
    Definitions ............................................................................................................................................... 36
    Statistics in Meta-Analysis ....................................................................................................................... 37
    Confidence intervals ................................................................................................................................. 38
    Results of Meta-Analysis ......................................................................................................................... 39
  The Grade Approach .................................................................................................................................... 41
    Strength of Recommendations .................................................................................................................. 42
Chapter 2
Evidence-Based Obstetrics

In providing maternity care, we endeavour to produce the most desirable results for our patients with minimum risks and costs. Decision-making is severely hampered when information is non-existent, incomplete, or unavailable. When information exists but is not used properly or consistently, the care provided may not be appropriate. The goal of the ALARM course is to promote care based on the best available evidence while also encouraging participants to develop skills in obtaining and evaluating evidence, and incorporating into daily clinical practice.

Types of Evidence: Qualitative or Quantitative

Qualitative evidence is “the organization and interpretation of non-numerical information for the purpose of discovering important underlying dimensions and patterns of relationships” (Polit & Hungler, 1995, p. 630). There are various types of qualitative evidence such as phenomenology, case study, and grounded theory. Qualitative evidence, derived from empirical research, case studies, and grounded theory, indicates patterns or trends and can influence ways of thinking about the issues studied. Although qualitative evidence has a role in achieving clinical understanding, evidence presented in the ALARM course is primarily quantitative.

Quantitative evidence uses numerical data, analyzed through statistical procedures. Quantitative research trials, which include randomized controlled trials (RCTs), cohort studies, case-control studies and surveys, generally provide stronger evidence than qualitative studies, although not all are scientifically rigorous.

Evidence collected from prospective research trials is more powerful than evidence collected from retrospective analyses of outcomes. In a prospective study, a hypothesis is proposed, and then data are collected and analyzed to test it. Variables can be anticipated, and the study designed to control for them. A retrospective study is not as powerful since it looks back in time at events that have occurred (e.g., chart review). The researcher cannot control for variables and must often rely upon charts that are incomplete.

The strongest quantitative evidence comes from randomized controlled trials (RCTs), although even RCTs are variable in quality. In an RCT, participants investigators do not determine which participants are assigned to an intervention or a control group. Based on the group results, the average of selected outcomes can be determined by an RCT.

Thus, a prospective RCT, which controls for known and unknown variables between those receiving and those not receiving a given intervention, is the most powerful way to discover whether the intervention has a significant impact.
Meta-Analysis

A meta-analysis is a statistical evaluation of a collection of studies that are similar in design, study populations, and outcomes examined. If there are differences between intervention and control groups, they are more likely to be evident when the numbers studied are large and when the difference in outcome is great.

In obstetrics, most serious outcomes are rare enough that there have been few randomized controlled trials of sufficient power to demonstrate a difference between an intervention and a control group. Meta-analysis may allow useful information to be obtained from these studies.

The primary benefit, however, of meta-analysis is that published data are systematically reviewed and the information synthesized and made more readily available.

Different meta-analyses of the same outcome may make different conclusions. This is due in part to different inclusion and exclusion criteria. It is therefore important to evaluate the methodology of the meta-analysis to determine its quality.

Caution

The techniques of meta-analysis are such that separate meta-analyses of the same subject may result in different findings. These generally result from the inclusion of different trials. There are 2 main reasons for this:

- Negative results in a randomized controlled trial are less likely to be published, and therefore cannot therefore appear in a meta-analysis. This often occurs when several initial small trials show promising results that are not substantiated in a subsequent well-designed large trial. Failure to include trials in a meta-analysis may also occur because of an incomplete systematic review of the literature.
- The problem of data "excess": the inclusion of multiple publications by different authors that are based on the SAME clinical trial.

Meta-analysis is useful in the absence of a large definitive trial. If a well-designed and well-executed trial exists, the importance of meta-analysis is lessened.

It must always be remembered that much of what is done is not supported by “good” evidence simply because trials have not been done.

Whenever possible, information from sound systematic reviews should be used to guide the appropriate and compassionate practice of medicine.

Statistics

Statistical tests are used to ascertain whether research results are valid and reliable. Following are definitions for some key terms used in the ALARM course.
Definitions

- **Sensitivity** is the likelihood that the diagnostic test will indicate the presence of disease when the disease is actually present. (True positive rate.) \[\frac{a}{a+c}\]
- **Specificity** is the likelihood that the diagnostic test will indicate the absence of disease when the disease is actually absent. (True negative rate.) \[\frac{d}{b+d}\]
- **Positive predictive value** is the likelihood that a positive test result actually means that the disease is present. \[\frac{a}{a+b}\]
- **Negative predictive value** is the likelihood that a negative test result actually means that the disease is absent. \[\frac{d}{c+d}\]

Bayes’ theorem: the predictive value of a test will depend on the prevalence of the disease. With high prevalence, the positive predictive value will increase and vice versa (a positive test for a low prevalence disease is likely to be a false positive).

![Disease-Test Table]

For example, a culture screening for group B streptococcus (GBS):

- Sensitivity of the test (+ve culture) is the chance that if the woman had GBS it will be detected by the test
- Specificity is the chance that the test will indicate no GBS (−ve culture) when in fact the woman does not have it
- Positive predictive value is the chance that a +ve culture represents GBS colonization
- Negative predictive value is the chance that a −ve culture actually rules out GBS
Statistics in Meta-Analysis

Odds ratio

When grouping studies together, the odds ratio (OR) compares the likelihood (relative odds) of the outcome being studied occurring in the group receiving the intervention (the “experimental” or “exposed” group) with the group not receiving the intervention (the “control” or “unexposed” group).

\[
OR = \frac{\text{Odds of observed outcome in experimental group}}{\text{Odds of observed outcome in control group}} = \frac{a \times d}{b \times c}
\]

Relative risk

The relative risk (RR, sometimes called the risk ratio) compares the risk or probability of the outcome in each group rather than the odds.

\[
RR = \frac{\text{Risk of observed outcome in experimental group}}{\text{Risk of observed outcome in control group}} = \frac{a / (a + d)}{c / (b + c)}
\]

The odds ratio serves as a surrogate for the relative risk, which is more difficult to manipulate using the statistics performed in meta-analysis. When the outcome studied is rare, the odds ratio closely approximates the relative risk.

Graphically, the odds ratio and relative risk is presented as a point on a horizontal, logarithmic scale. A vertical line drawn at 1 indicates no difference in the outcome between the 2 groups. Ratios < 1 will be represented to the left of the vertical
line and those > 1 will be represented on the right side of the vertical line. The data presentation is usually constructed so that the results < 1 are an improvement in outcome.

**Confidence intervals**

The confidence interval is a measure of statistical significance, generally calculated as the least and greatest results within which the reported outcome of the experiment would fall 95% of the time. It is displayed graphically as a horizontal line through the outcome point where the left end represents the lowest and the right end represents the highest point. The 95% confidence interval is equivalent to the probability statistic \( P < 0.05 \).

Measurements of confidence do not eliminate the possibility that the results of an experiment are due to chance, they just indicate how likely it is that such a result is due to chance. The judgement of the clinician is required to interpret whether or not a result is clinically significant, regardless of the statistical expression of probability used.

For a single trial, the outcomes of interest are shown with their individual confidence intervals.
Results of Meta-Analysis

In a meta-analysis, the result is plotted below the individual studies used in the analysis. The meta-analysis result is calculated when all the trial results are evaluated, weighted and then pooled for a single given outcome of interest. The associated confidence interval will be narrower than the confidence intervals associated with the individual trials because the number of participants represented is greater.

Effect of Intervention on Outcome of Interest

<table>
<thead>
<tr>
<th>Trial</th>
<th>n</th>
<th>Outcome less likely</th>
<th>Outcome more likely</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trial A</td>
<td>220</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trial B</td>
<td>145</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trial C</td>
<td>550</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trial D</td>
<td>205</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trial E</td>
<td>325</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The confidence intervals displayed for each study will be broader than the confidence interval of the meta-analysis and vary according to study size. In the example above, Trial C has the narrowest confidence interval because of its larger size. The meta-analysis odds ratio is the narrowest of all as it mathematically incorporates all of the trials’ subjects. A trial’s failure to demonstrate a difference may be due to a lack of power (i.e., not enough subjects given the effect size), which is the case in Trial D in the above example. Again, confidence intervals that cross the vertical line at 1 mean that \( P > 0.05 \) for the outcome in that trial. Although this result is therefore not statistically significant, if all the studies lie to one side of the vertical axis, it indicates a trend in the same direction. Trial B is an outlier and requires an explanation (cheating? different population? misdiagnosis?). The trials are then said to be homogeneous. This suggests that a difference may truly exist and may become apparent once the typical odds ratio is calculated or more studies are added to the analysis.
The results displayed in a meta-analysis fall into 1 of 3 categories:

1. The result **lies to the left** of the vertical axis (1) and the confidence interval does not cross 1. This indicates that the outcome is less likely to occur in the treatment group than in the control group and the result is statistically significant.

2. The result is **at or near 1**, and the confidence interval line crosses 1. This indicates that there is no statistically significant difference in outcomes between the groups.

3. The result **lies to the right** of the vertical axis, and the confidence interval line is also completely to the right of 1. This indicates that the outcome is more likely to occur in the treated group than in the control group.

A meta-analysis of studies that failed to achieve statistical significance individually, because of insufficient numbers or small effect size, may show that most of these studies demonstrate the same trend, indicating that the difference does exist.

For example, several studies were conducted regarding the use of antepartum glucocorticoids on fetal lung maturity and the occurrence of neonatal respiratory distress syndrome. Shown are the lead author, year of study publication and number of subjects in each trial.
The Grade Approach

The Grading of Recommendations, Assessment, Development and Evaluation (GRADE) is a system for rating the quality of evidence in systematic reviews and other evidence syntheses, such as health technology assessments and clinical practice guidelines. GRADE offers a transparent and structured process for developing and presenting evidence summaries and for carrying out the steps involved in developing recommendations.

The GRADE approach uses 4 grades:

<table>
<thead>
<tr>
<th>QUALITY OF EVIDENCE GRADES</th>
</tr>
</thead>
<tbody>
<tr>
<td>GRADE</td>
</tr>
<tr>
<td>-----------------</td>
</tr>
<tr>
<td>High</td>
</tr>
<tr>
<td>Moderate</td>
</tr>
<tr>
<td>Low</td>
</tr>
<tr>
<td>Very Low</td>
</tr>
</tbody>
</table>

The GRADE approach to rating the quality of evidence begins with the study design (trials or observational studies) and then addresses 5 possible reasons for downgrading the quality of evidence and 3 for upgrading the quality of evidence.

<table>
<thead>
<tr>
<th>FACTORS THAT CAN REDUCE THE QUALITY OF THE EVIDENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>FACTOR</td>
</tr>
<tr>
<td>Limitations in study design or execution (risk of bias)</td>
</tr>
<tr>
<td>Inconsistency of results</td>
</tr>
<tr>
<td>Indirectness of evidence</td>
</tr>
<tr>
<td>Imprecision</td>
</tr>
<tr>
<td>Publication bias</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>FACTORS THAT CAN INCREASE THE QUALITY OF THE EVIDENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>FACTOR</td>
</tr>
<tr>
<td>Large magnitude of effect</td>
</tr>
<tr>
<td>All plausible confounding would reduce the demonstrated effect or increase the effect if no effect was observed</td>
</tr>
<tr>
<td>Dose-response gradient</td>
</tr>
</tbody>
</table>
Strength of Recommendations

The strength of a recommendation reflects the extent to which a guideline panel is confident that desirable effects of an intervention outweigh undesirable effects, or vice versa, across the range of patients for whom the recommendation is intended.

GRADE specifies 2 categories of the strength of a recommendation. While GRADE suggests using the terms strong and weak recommendations, those making recommendations may choose different wording to characterize the 2 categories of strength.

### IMPLICATIONS OF STRONG AND WEAK RECOMMENDATIONS

<table>
<thead>
<tr>
<th></th>
<th>STRONG RECOMMENDATION</th>
<th>WEAK RECOMMENDATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>For patients</td>
<td>Most patients in this situation would want the recommended course of action, and only a small proportion would not.</td>
<td>The majority of patients in this situation would want the suggested course of action, but many would not.</td>
</tr>
<tr>
<td>For clinicians</td>
<td>Most patients should receive the recommended course of action. Adherence to this recommendation according to the guideline could be used as a quality criterion or performance indicator. Formal decision aids are not likely to be needed to help individuals make decisions consistent with their values and preferences.</td>
<td>Recognize that different choices will be appropriate for different patients, and that you must help each patient arrive at a management decision consistent with her or his values and preferences. Decision aids may well be useful helping individuals making decisions consistent with their values and preferences. Clinicians should expect to spend more time with patients when working towards a decision.</td>
</tr>
<tr>
<td>For policy makers</td>
<td>The recommendation can be adapted as policy in most situations including for the use as performance indicators. The recommendation can be adopted as policy in most situations and can be used as the basis of performance indicators.</td>
<td>Policy-making will require substantial debates and involvement of many stakeholders. Policies are also more likely to vary between regions. Performance indicators would have to focus on the fact that adequate deliberation about the management options has taken place.</td>
</tr>
</tbody>
</table>
Suggested Reading


# Table of Contents

Chapter 3 Patient Safety .......................................................................................................................... 45  
  Introduction ........................................................................................................................................... 45  
  Definitions ............................................................................................................................................. 45  
  Prevalence ............................................................................................................................................ 45  
  Understanding Error in Health Care ....................................................................................................... 47  
  Understanding why we fail ...................................................................................................................... 48  
  Defences against Error ............................................................................................................................ 49  
  Understanding why we succeed (Safety II Production safety) ................................................................. 52  
  The Process of Risk Management ........................................................................................................ 53  
  High Reliability Organizations ............................................................................................................. 55
Chapter 3
Patient Safety

Introduction
The complexity of healthcare systems exposes patients and care providers to risks that may result in significant adverse outcomes. The development of quality (which must, but do not always include a sharp focus on patient safety) and risk management programs in each unit will lead to improved patient care, reduced medico-legal risks, and lower costs. Systems and processes that balance an understanding of why we fail, as well as why we succeed, increase the likelihood of success and minimize the likelihood of errors, and are essential to ensuring patient safety.

Definitions
Risk management: The development and implementation of strategies to prevent or limit injury, damage, harm, discomfort, disability, or distress to the patient.¹

Patient safety: Has been defined as: The absence of harm that results from the provision of care and the reduction and mitigation of unsafe acts, through the use of best practices.²⁻³ In this context, safety is hard to quantify as it is a non-event.

Safety can also be described as the occurrence of successful outcomes. In this way, safety is easier to quantify: it is a dynamic event.⁴

Resilience: The ability of a system to sustain required operations in both expected and unexpected conditions.⁵

Prevalence
A substantial body of evidence points to medical errors as a leading cause of preventable death and injury:⁶

- Adverse outcome data reported by the Institute of Medicine (IOM) revealed health care provider error resulted in 44,000 to 98,000 patient deaths per year. This was higher than the number of deaths from traffic accidents, breast cancer, and HIV infection, making patient death from clinical error the fourth leading cause of death in the United States.⁶ Even using the lower number, death due to clinical error ranked ninth, ahead of motor vehicle accidents, chronic liver disease, alcohol- and drug-induced causes, and various cancers.⁶ The overall societal cost of clinical error approached $38 billion annually. Approximately half of this ($17 billion) was associated with preventable errors.⁶
- In 2004, a report by HealthGrades (Colorado) suggested that the number of deaths due to medical error had been underreported in the IOM study. Data from their review estimated that approximately 195,000 deaths per year were due to preventable hospital errors.  

- In 2000, in the United Kingdom, the National Health Services (NHS) reported that adverse events occurred in association with 10% of all hospital admissions. This resulted in 850,000 reported events at a cost of 2 billion pounds to the NHS annually.  

- The Canadian Adverse Events Study reported a 7.5% incidence of adverse events for all admissions to Canadian hospitals. This study included data from a random sample of charts for non-obstetric, non-psychiatric adult patients in acute care hospitals in five provinces (British Columbia, Alberta, Ontario, Quebec, and Nova Scotia) for the fiscal year 2000. It was estimated that of the nearly 2.5 million admissions to hospitals, 185,000 were associated with an adverse event. Almost 70,000 of these events were potentially preventable. The authors also indicated that there were 9250 to 23,750 preventable deaths from adverse events during this period.  

- An article in 2011 suggested that the “Global Trigger tool” shows that adverse events in hospitals may be ten times greater than previously measured.  

- In 2013, John James wrote in the Journal of Patient Safety the following conclusion to his study: “Given limitations in the search capability of the Global Trigger Tool and the incompleteness of medical records on which the Tool depends, the true number of premature deaths associated with preventable harm to patients was estimated at more than 400,000 per year. Serious harm seems to be 10- to 20-fold more common than lethal harm.”  

- His conclusion is particularly poignant: “In a sense, it does not matter whether the deaths of 100,000, 200,000 or 400,000 Americans each year are associated with PAEs (Preventable Adverse Events) in hospitals.”  

- He continues by saying, “Any of the estimates demands assertive action on the part of providers, legislators, and people who will one day become patients. Yet, the action and progress on patient safety is frustratingly slow; however, one must hope that the present, evidence-based estimate of 400,000+ deaths per year will foster an outcry for overdue changes and increased vigilance in medical care to address the problem of harm to patients who come to a hospital seeking only to be healed.”
Understanding Error in Health Care

Why does the high rate of clinical error in hospitals persist in the face of a plethora of published clinical practice guidelines, policies, recommended procedures and standards? The availability of excellent continuing education courses at the local, regional, and national levels that address evidence-based approaches to patient management has never been greater.

A few facts:

- Humans are fallible. It is impossible to attain human perfection despite extensive training.
- You cannot eliminate error through education or explanation alone.

We work in a complex world with the support of flawed systems.

“A plethora of Guidelines, policies, recommended procedures….”

Perhaps here may lie some of the problem.

Healthcare has been described as a complex adaptive system. If this is so, then many of our efforts in Safety, which have revolved around restricting and controlling humans in an effort to mitigate against their fallibility, may have been misguided. Could these approaches have only considered half of the problem? In so doing, we may have gone overboard in the production of restrictive tools to “guide” the care we give. We need to ask: Could there be so many tools in our tool kit, that abiding to them all would make completing our work impossible?

Furthermore, do all these tools really add to safety of our care? “A worker following a safety rule can create a condition to enable safety to emerge. Too many safety rules can overwhelm, and frustrate a worker, enabling danger to emerge.”

According to Braithwaite et al. over 600 guidelines policies, and procedures guide our work in one single day. These tools constitute what they refer to as work-as-imagined by well-intended managers and policy makers. According to the authors, work-as-done does not resemble work-as-imagined, because the latter cannot account for “how circumstances vary, the diversity of patients, how goal-conflicts abound, how expected resources may be missing” or quite simply the unpredictable nature of both our patients and environment. In this reality, front-line teams must balance efficiency against thoroughness every moment of every day. By this we mean that policies, procedures and other tools to guide the care we give simply cannot be followed the way leaders believe they are. Doing so would lead to the organization not being able to meet demand. To be successful, organizations favour efficiency in this trade-off principle until, it is recognized in hindsight, that they should have been more thorough in that particular case.

How did we come to be here?

A 2015 report from the National Patient Safety Institute examining the slow progress in safety, cited the observation by Schultz. He states that knowledge moves in three phases and notes how those phases apply to risk reduction in health care.
1. **Superficial simplicity**: For example, the belief in the early days of safety that simply adopting the structure and tools of High Reliability Organizations – such as the airline industry (a complicated system, and not a complex one) – would be sufficient.

2. **Confusing complexity**: According to the report, this is the current situation in health care, with an overwhelming number of tools, guidelines, policies, and procedures (work-as-imagined), which are in place to improve safety but which in practical terms can become a form of barrier to safety.

3. **Profound simplicity**: According to the report, “Improved culture is not the means to an end but an end itself. For example, we know that superficial implementation of a surgical checklist changes little. However, a major teamwork and culture intervention that also included checklists (rather than the other way around) reduced mortality by 50% more than secular trends. Thus, according to this checklist example, a core focus on culture (with checklists as a tool) can improve outcomes. This example helps explain why the panel felt leadership and culture are crucial to accelerating progress in patient safety.”

The above realization coupled with the principles of resilience may allow us to reach beyond the low-hanging fruit we have achieved to date. It is important to understand that as complexity and uncertainty rise in our workplace, the usual restrictive tools no longer work well. In short, Healthcare is a complex adaptive system, but it is run as a complicated system, as a result, progress in safety has stalled.

Be that as it may for safety to occur, it is not about reinventing the wheel. However, a balance must exist between reducing the likelihood of things going wrong – the traditional approach: using restrictive tools, or protective safety - and increasing the likelihood that things go right (see Safety II – below). As such, an understanding of the following sections is important.

Errors should be recognized as consequences rather than causes.

**Understanding why we fail**

**Figure 1. The relationship between hazards, defences, and losses**

Defences against Error

Like other organizational structures, the health care system has developed defenses and safeguards. When these defences are breached by hazards, losses may occur.

Classification of Defences

<table>
<thead>
<tr>
<th>HUMAN</th>
<th>SYSTEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Training</td>
<td>Credentialing</td>
</tr>
<tr>
<td>Knowledge</td>
<td>Peer review</td>
</tr>
<tr>
<td>Judgement</td>
<td>Protocols, pathways, and policies</td>
</tr>
<tr>
<td>Manual dexterity</td>
<td>Special teams</td>
</tr>
<tr>
<td>Vigilance</td>
<td>Continuing medical education</td>
</tr>
<tr>
<td></td>
<td>Risk management and quality management</td>
</tr>
</tbody>
</table>

Classification of Failures

Figure 2. Unsafe acts
In Reason’s view, our current medical system functions like a complex technological system. Professor James Reason has described how human beings contribute to the breakdown of such systems. Unsafe acts can have a direct impact on the safety of the system with immediate adverse effects. He calls these active failures. These unsafe acts are the consequences of system failures rather than the causes of the failures.

He also states that “people working in complex systems make errors or violate procedures for reasons that generally go beyond the scope of individual psychology.” He calls these reasons latent failures (conditions). In the healthcare system, this concept can be visualized as a pyramid (Figure 2). At the sharp end are patients and healthcare providers. The major factors that determine safety at the sharp end are related to latent conditions located at the blunt end and at various levels throughout the pyramid. It is at the sharp end of the care system, where we are in a precarious balance where all of the factors that determine safety come together creating “safe” or “unsafe” conditions for both patients and caregivers.

Expanding on these two types of failures:

1. **Active failures**: These occur at the front-line (individual level). They have an immediate effect, affect the patient directly, and have traditionally resulted in some form of punishment and/or retraining of the individual involved. This action discourages reporting, and usually doesn’t prevent similar events from occurring in the future. It is not educational for the team and/or other colleagues, and allows latent failures to remain in the system.

   Active failures include the following:
   
   - Mental Slip (often due to fatigue): e.g. administering the wrong drug or the wrong dose. In this case, care providers know what the right drug or dose is. Retraining would not help prevent this error in the future.
   - Mental Lapse: e.g. forgetting to catheterize the bladder before a procedure that requires one. In this case, care providers forget one of a list of items. Retraining would not help. A checklist might mitigate this risk.
   - Mistake: e.g. misinterpreting an electronic fetal monitor pattern.
   - Procedural violation: e.g. applying obstetrical forceps before full dilatation of the cervix.

2. **Latent failures** are failures that are distant from direct front-line control, may involve poor design or maintenance decisions, and have little direct effect but instead have a cumulative effect. Latent failures do not usually have predictable effects (unlike active failures). They are the greatest threats to patient safety, and they are indicative of a problem with the system rather than the individual.

   Latent failures may be attributable to:
   
   - Human factors: fatigue from a long shift, stress, multi-tasking, multiple different care providers for one patient
   - Local workplace factors: inadequate tools/equipment, poor communication and teamwork, unworkable or ambiguous procedures/protocols, leadership shortcomings, insufficient staff training, staff shortages
Organizational factors: strategies and top-level decisions made by governments (e.g., budget cut to hospital), regulatory bodies (e.g., change in scope of practice), manufacturers (e.g., different drugs in similar appearing containers), or hospital administrators (e.g., staff reductions in face of increasing patient load).

How Breaches Lead to Harm

In an ideal world, each defensive layer would be intact. In reality, however, they are more like slices of Swiss cheese, having many holes—though unlike in the cheese, these holes are continually opening, Shutting, and shifting their location. They are thus in many ways unpredictable. The presence of holes in any one "slice" does not normally cause a bad outcome. Usually, a bad outcome can only happen when the holes in many layers momentarily line up to permit a trajectory of accident opportunity—bringing hazards into damaging contact with victims.17

Figure 3. Reason’s Swiss Cheese Theory.

BMJ 2000;320(7237):768-70. Adapted and reproduced with permission from the BMJ Publishing Group.

The above model (Figure 3) assists us in understanding the concept of a sequence of events, whose individual components will often not lead to harm, but when placed together create the context of an event. We can also understand from this model that the stopping of an arrow is often the act of a human—usually a wonderful, well-intentioned member of our front line team who will often not report his or her achievement. Our culture traditionally would not look favorably on an individual having a ‘near miss’. What would we say to someone who came up to us and announced: "Look at me! I nearly gave the wrong drug!"? Yet, for us to advance in our efforts in patient safety, we owe it to ourselves, and the patients we serve, to report these near misses and study them. We need to understand that a sequence of events exists that led us to the
point where “we nearly gave the wrong drug”. That sequence, if left in place, will affect our colleagues (maybe as early as) next week, next month, or next year. Through pure bad luck, one day that arrow will not be stopped and a patient will be harmed. But the same sequence will have been repeated over and over again (stopped at varying layers of Swiss cheese) for us to learn through that fateful harm event. This is why it is said that errors lie “dormant” in the system. Only by understanding the failures in existing systems can we repair them. A keen understanding of systems, event review, and the ability to create, and implement, recommendations by the individuals at the front line is key to developing a local ownership to problems and create a culture of patient safety. This is the foundation principle of CUSP programs (Comprehensive Unit-based Safety Programs) such as the successful and effective MORE\textsuperscript{20} Program.\textsuperscript{21, 22}

A final thought on the Swiss cheese model. Traditional Healthcare will review harm events and develop recommendations and policies and/or protocols to mitigate new risk - this is a necessary action. These can be seen just as additional layers of Swiss cheese. These tend to be added to the existing ‘layers of cheese.’ The more of these layers exist, the harder it can be for a healthcare worker to navigate and the longer before he or she “starts treating” the patient. Thus, the very presence of all these ‘safety’ layers, over time as they are added to, invite the human to bypass them. The layers – in place for safety - can in and of themselves become a barrier to safe patient care.

The focus must be on narrowing the holes, not necessarily adding new layers.

We know how to provide a patient with the best care. What we cannot do, it seems, is provide that same level of care to each and every patient consistently – this can change as we look to High Reliability and Resilience Engineering approaches and create and embed patient safety into our Teams’ DNA.

Reason’s Swiss cheese model is excellent as a primer, but it needs to be understood that it is a little too simple to explain all errors in the complex dynamic system of Healthcare.

**Understanding why we succeed (Safety II Production safety)**

Safety-II offers a complimentary approach to the concept of patient safety.

While understanding the need to improve processes that leave the patient vulnerable to human error (the traditional approach to safety, also known as Safety I – or protection safety – discussed above), Safety II focuses on the important realization that it is remarkable that so many patients have positive outcomes despite important challenges to Care Teams (also known as production safety). These challenges may include an increased patient load, staff calling in sick, equipment not working, units being on red-alert, etc.

Safety II then, looks at the work as done - which allowed care operations to be maintained despite these challenges – as a way to improve existing systems (the existing systems are known as: work as prescribed – or the plethora of guidelines and policies (mentioned above) that we are surrounded by).

The biggest difference between Safety I (the traditional approach to safety) and Safety II is the way in which the human is viewed: a liability versus a resource. Thereby, an important step to sustainable safety is to understand WHY something goes right despite so many challenges to its success. Understanding why we succeed allows us to recreate it and improve the robustness of our processes.
It is important to note that there is no competition between Safety I and Safety II, but a balance between the two. We still need *protection safety*, but we also need *production safety*.

The MORE Programs have identified that for sustainable safety to occur within a unit, the necessary balance between Safety I and II for that unit needs to be identified and nurtured. The MORE approach seeks to identify that *balance* - between Safety I and Safety II - needed for each unit to succeed.

It is clear to many that the traditional approach to safety – Safety I – has managed to fix the proverbial "low hanging fruit." But to make real, substantive, and sustainable change in safety will require an understanding and application of Safety II.  

### The Process of Risk Management

As inferred above, risk cannot be completely or constantly avoided. The intention in risk management is to anticipate risk and prevent or limit harm. Risk identification must concern itself with all aspects that may threaten and/or jeopardize the patient, the care providers, and the facility.

Risk management has five steps:  

1. **The Identification of Risk**
   - This is an essential first step because risk management is a proactive strategy
   - Patients and providers are best served by informed anticipation of problems
   - Risk is identified with reference to history. Has this event, or something like it, happened before?
   - Risk can also be identified when practitioners are informed about particular risks inherent in their group or profession

2. **Risk Assessment**
   - An evaluation exercise
   - Answers the questions of how many or how often (frequency of risk), how much (cost of risk), and under what circumstances (likelihood of risk)
   - Answers to these questions are essential to designing appropriate preventive programs
3. **Action to Manage Risk**

<table>
<thead>
<tr>
<th>AVOIDANCE OF RISK (RESULTS IN NO LOSS)</th>
<th>Discontinue procedures associated with high degree or incidence of risk (e.g., high forceps deliveries).</th>
</tr>
</thead>
<tbody>
<tr>
<td>PREVENTION OF RISK</td>
<td>Hospitals and maternity care providers can take steps to prevent or significantly lower the possibility of mishap. By establishing risk management programs, hospitals and maternity care providers can take steps to prevent or significantly lower the possibility of mishap and adverse outcomes.</td>
</tr>
<tr>
<td>TRANSFER OF RISK</td>
<td>A high-risk case (e.g., preterm labour) can be transferred to a higher level of expertise for care.</td>
</tr>
<tr>
<td>REDUCTION OF RISK (RESULTS IN SOME LOSS)</td>
<td>Prevention and reduction may be confused. It is important to note that reduction is the strategy that is called into play after the damaging event has occurred. One of the most important risk reduction strategies is the immediate care of and attention to persons threatened or injured. Losses attended to immediately can often be mitigated. Losses can be reduced after an event through early investigation and documentation and the provision of full and honest disclosure.</td>
</tr>
<tr>
<td>SEGREGATION OF RISK</td>
<td>Segregation is usually more applicable to facilities (e.g., Level I, II, and III hospitals) than to individual care providers. At the individual level, segregation of risk would occur when consultations with other specialists or colleagues are obtained during the management of a difficult situation (e.g., consulting a hematologist to assist in the management of a woman with a coagulation disorder secondary to preeclampsia).</td>
</tr>
<tr>
<td>FINANCING OF RISK</td>
<td>Premiums are paid by institutions, individuals, and governments to insurance agencies or defence organizations, which assume the responsibility for the financial compensation awarded by the courts.</td>
</tr>
</tbody>
</table>

4. **Implementation of Risk Management Programs**

When considering risk management strategies, it is necessary to be aware of current practice and of alternatives that are both practical and compatible with the objectives of care. It might be attractive from a risk management perspective to stop some high-risk procedures (risk avoidance). However, the nature of practice may make such an alternative unacceptable. An example of an unacceptable approach would be to abandon all forceps deliveries. Another important strategy to manage risk is the development of a process enabling health care providers to learn from no-harm and harm events (near misses and adverse events). Reviews of these occurrences must take place in an objective and non-punitive environment. It is imperative that system-based failure rather than human error is emphasized during reviews of unexpected clinical processes and/or outcomes.

5. **Evaluation of risk management strategies**

It is essential to review the effect of a new process or strategy introduced to reduce or eliminate risk to determine whether it achieved the expected results. Reviews should be as frequent as required to cover all aspects of risk assessment and management. A committee may be appointed to monitor activities. Reports on the success of initial attempts at implementation are integrated into the management plan.
Risk management is a continuous process. It starts with identification and analysis of risk, proceeds to the implementation of strategies to manage risk, and ends with the evaluation of risk-management activities and their results.

**High Reliability Organizations**

**What is Reliability?**

Reliability in Healthcare can be defined as: a process doing what it was intended to do, in the place it was designed to be performed, with the regular staff complement expected.24,27

Reliability of a process is measured by its success:

- If 90 percent of patients receive their pre-op antibiotics at the appropriate time, the Process is reliable 90% of the time.
- The failure rate of a process then is the inverse of its success. So in the above example the failure rate is measured by the 10% of patients who did not get their pre-op antibiotics in time, or 1 in 10, or $10^{-1}$.

Research tells us that a failure rate of $10^{-1}$ or less is characterized as coming from strategies that include Training, Reminders and the Reliance on the good will, attentiveness and hard work of Healthcare Staff.24-28 This would suggest that those ways of doing things hold us to never improving our outcomes.

To improve our system's reliability, we need to change the Status Quo. Where possible, we must move towards a system that understands human fallibility and is designed to take it into account. Systems using quality and safety tools, evidence based procedures, using Human Factors engineering and an understanding of human psychology.24-28

An HRO is defined by its ability to operate technologically complicated systems without error over long periods of time.24-28

To help distinguish which organizations could be considered high reliability organizations, ask the following question. “How many times could the organization's systems have failed with potential catastrophic consequences but did not?” If the answer to your question is many, many times, then it is a high reliability organization.

Several organizational structures have been studied by behavioral scientists and designated as high reliability organizations. They are:

- air traffic control
- nuclear-powered aircraft carriers
- nuclear power plants
- the technical side of banking (e.g., ATMs)

These organizations have three defining characteristics:

- They are complicated, internally dynamic and intermittently, intensely interactive.
- They perform exacting tasks under considerable time pressure.
They carry out these demanding activities with low incident rates and an almost complete absence of catastrophic failures over long periods of time.

Hierarchy is an interesting facet of an HRO. When an HRO is running in a routine mode, it is governed by the conventional hierarchical structure. In an emergency situation, that governance shifts and safety takes precedence i.e. any individual, regardless of position in the organization, takes command of the situation until it is either resolved or someone else with more expertise takes over the management of the crisis. When the emergency has ended, the organization returns seamlessly to its routine control mode.19

A strong feature of HROs is front line ownership of operations and their ability to be aware and adaptive. It has not escaped the attention of most that the front line frequently has the solution to a problem. It is in this way that HRO and Resilience Engineering are in many ways complementary. However, due to the way hospitals function, it appears that all too frequently, no one seems to be listening to the front line. This contributes to an unengaged workforce.

A significant risk in an unengaged workforce is that it may be that they are 'just coming to work'. This is what is meant by the term 'it's just a job'; we might just be content in doing what we are told and finish the day. The only goal we have is that of: going home. This is a result of 'buy-in' (as opposed to Ownership) as described by Zimmerman et al. and is a warning sign of an organization in trouble. It is a workforce that is content to just follow orders – a disengaged, complacent workforce that comes to work, completes their shift and goes home.

What we want in our units is the opposite: an engaged workforce that identifies a problem and owns it to its resolution. Due to the mutual accountability within such a team, the solution has a much likelier chance of success and sustainability. A unit with such front line ownership has an aware and adaptive culture – a necessary step to an HRO. HRO’s understand that surprises should never be; they are anticipated. HRO’s are preoccupied with failure.26,30

“HROs are aware and adaptive enough to intercept or mitigate threatening events and circumstances.” 26

The HRO and normalization of deviance

Paradoxically, HROs view successful operations as potentially dangerous. Success leads to system simplification, short cuts, and “the normalization of deviance.”31-32 Imperceptibly, as time goes on, technical and professional standards degrade, i.e., these short cuts, and non-standard activities become “normal” for that individual or unit.

All group cultures become less safe over time. Risky operational systems and clinical practices continue, because those involved think “they get away with it” most of the time.32 A unit is at its most dangerous when nothing bad has happened for some time.

HROs “actively and continually question assumptions, promote orderly challenge of operating practices and solicit outside views of the routine”32 to address normalization of deviance.
Perinatal and obstetrical units and HROs

“Understanding updated and refined concepts of high reliability in the delivery of obstetric care requires an understanding of what high reliability is not (i.e., a quality improvement method focused on efficiency and productivity. Rather, high reliability is a creation of a culture and processes that radically reduce system failures and effectively respond when failures do occur.”

Perinatal units have many of the characteristics of HROs. They are complex, technical environments with a variety of professional disciplines that have diverse roles and responsibilities. Patients and their families and friends expect obstetrical units to function without error over long periods of time. Normalization of deviance is a characteristic of obstetrical/perinatal units as exemplified in the following:

- Application of forceps or vacuum when the cervix is not quite fully dilated
- Chronic under-staffing
- Fundal pushing with shoulder dystocia
- Use of a combination of prostaglandins and oxytocin for induction of labour
- Artificial rupture of membranes when the head is too high

HRO principles in health care:

- Safety is the priority and is everyone’s responsibility
- Operations are a team effort
- Hierarchy disappears in an emergency. Decisions about safety can be made at any level of the organization
- Communication is highly valued
- Emergencies are rehearsed
- There is interprofessional review of routine functions, near misses, and unexpected events

When obstetrical units successfully adapt HRO principles, they have:

1. A clearly stated goal and purpose: patient safety.
2. Clear language that defines patient safety.
3. Established clear and agreed-upon markers of well-being.
4. Clearly defined organizational teamwork:
   - Minimized hierarchy: high reliability perinatal units thrive on teamwork
   - Everyone is respected and recognized as competent
   - Everyone knows what everyone else is thinking and seeks opportunities to help other team members
   - Everyone is focused on patient safety
   - Planning, data collection, and quality management are interprofessional exercises.
5. Patient care records that are not divided by profession. The principle is to use one patient care record that is maintained, completed, and quality managed by the interprofessional team. The record is the narrative of the patient’s care. Electronic health records have brought a new dimension of challenges in this area.
6. Policies that are unit-wide rather than separate for each profession (nursing, medicine, midwifery, etc.):
– high reliability units have a minimum number of policies
– there is an inverse relationship between the number of written policies and existing reliable functions
– one policy exists in all safe units: a physician, midwife, or nurse will come when requested and no one is reprimanded, in any way, for a false alarm, since the call was made with only one thought in mind, the unit’s goal: patient safety.

7. Two clear operating principles:
   – adherence to professional guidelines
   – minimal intervention but with the ability to intervene, when necessary, in a timely manner.

8. An ability to communicate honestly and openly in perceived hierarchical situations. Establishing good communication in a team environment will break down the barriers of an authority gradient. This enables optimal patient care in environments dealing with critical events.

9. Ensure patient safety will always override hierarchy.

The Effectiveness of High Reliability Units

The MORE\textsuperscript{OB} program, created in 2001 by the Patient Safety Division of the SOGC, is a patient safety program based on the HRO and resilience engineering principles and centered on team function and culture change.

Several studies suggest the potential of the HRO approach. One from Alberta, reported that the MORE\textsuperscript{OB} program, which had been implemented province-wide, had a positive effect on health service delivery as well as newborn and maternal outcomes.\textsuperscript{35} Another, designed to address two HRO principles (“operations are a team effort” and “emergencies are rehearsed”) as they pertain to the management of umbilical cord prolapse found an important improvement in management, most notably a significant reduction in the diagnosis to delivery time interval.\textsuperscript{36} And finally, a review of mandatory reportable harm events in 34 hospitals revealed significant improvement.\textsuperscript{22}

Quality and risk management: health care provider responsibilities\textsuperscript{37}:

- Identify high-risk procedures
- Recognize errors before they result in adverse outcomes
- Ensure that health care providers are aware of their limits and practice within these limits
- Review near misses, accidents, injuries, and adverse events as system failures rather than individual failures
- Establish an environment for clinical error management
- Eliminate the “blame” and “punitive” culture associated with adverse events
- Be aware of local, regional, and national clinical guidelines and policy statements
- Remember the three A’s of quality care and risk management: ability, accessibility, availability
- Promote a culture in which communication and team-building occur.
- Collaborate at a local, regional, and national, level to develop performance indicators and benchmarks for clinical activities
Care Provider Participation

Interprofessional committees are needed to promote teamwork and patient safety. Without the full participation of all of its maternity care providers and middle and senior administrators, a facility cannot have an effective risk management system. Health care providers will accept and support risk management activities if they are aware of the benefits of these activities for their patients and themselves.

All who work in the unit are orientated to confirming fetal and maternal well-being. Variations are reduced by standardizing policies and procedures where possible. Teamwork is the hallmark of the unit. Interprofessional review of adverse outcomes and near misses are practiced in a non-punitive and educational environment.
References


Table of Contents

Chapter 4 Management of Labour ................................................................................................................. 64
  Introduction ...................................................................................................................................................... 64
  Factors that affect Labour ................................................................................................................................. 64
  Assessing Labour ........................................................................................................................................... 65
  Care During the First Stage of Labour ............................................................................................................. 67
    Latent Phase of the First Stage of Labour ......................................................................................................... 67
    Active Phase of the First Stage of Labour ......................................................................................................... 68
  Care During the Second Stage of Labour ......................................................................................................... 70
  Management of The Third Stage of Labour ...................................................................................................... 74
    Dystocia ......................................................................................................................................................... 75
    Avoiding a Primary Caesarean Section .......................................................................................................... 80
  Summary ......................................................................................................................................................... 83
Chapter 4
Management of Labour

Introduction
Labour is a natural physiological process with an expected outcome of vaginal birth. Caregivers should strive to create a positive family experience while maintaining the maternal and newborn wellbeing, preventing complications and responding to emergencies. As 60% of women enter labour without obstetrical or pre-existing health concerns, minimal interventions would be expected to achieve this outcome.

Interventions are considered when the reasons are clearly documented; the benefits outweigh the risks they support a safer outcome, the mother is informed of the risks and benefits, and the intervention avoids unintended consequences, such as, Caesarean section. Avoiding a primary CS provides reduced morbidity and mortality in the index pregnancy and better outcomes for subsequent pregnancies.

Factors that affect Labour
Personal and environmental factors can affect the course of labour and birth and should be considered by the caregiver. Some of these include:

- Support for women during active labour and birth may increase a family's satisfaction with the birth experience, reduce the use of medications and interventions and enhances the positive attitude women need to care for their babies. Ongoing communication should be clear and timely so that the woman is engaged in the process.
- Preparation for labour and birth can be obtained from health care providers, prenatal classes, peers/family, media, internet sites and mobile apps however non English/French speaking and minority population concerns are not well addressed in the available courses.
- Ethnocultural diversity in obstetrical families requires caregivers to develop cultural competence and sensitivity regarding individual practices and the uniqueness of each family.
- Maternal age at birth is increasing across Canada potentially increasing the incidence of medical comorbidity and assisted reproductive technology. In Ontario 4.2% of births occur in individuals >40 years and 18.9% occurred in individuals between 35-39 years. As with all women, the care of the older parturient needs to be individualized and her care planned based on evidence and her needs.
- Location of birth may include hospitals, birthing centres and home. For out of hospital births care is similar to births in the hospital setting but consideration must be given availability and timing of transport, emergency services and attendance of a second caregiver.
Assessing Labour

1. Verify Expected Date of Delivery

Naegele’s rule, developed in 1812, calculates the expected date of delivery using the last menstrual period (LMP): first day of LMP – 3 months + 7 days. Limitations include inaccurate reporting of the date of the LMP, bleeding unrelated to menses, and delayed ovulation. A 1990 retrospective study of uncomplicated pregnancies in Boston compared the length of pregnancy calculated using Naegele’s rule with the actual date of spontaneous labour. In non-parous women, labour occurred 8 days later than predicted by Naegele’s rule (41\(^1\) weeks), and in parous women labour was, on average, 3 days later than predicted by Naegele’s rule (40\(^3\) weeks). A 2015 Cochrane review (N = 25,516, 8 studies) of the routine use of early ultrasound for dating demonstrated a modest reduction in induction of labour for post-term pregnancy from 3.8% to 2.2%. Routine first trimester ultrasound also detects twin gestations at an age when chorionicity can be reliably determined.

Ultrasound measurement of gestational age at 7 to 23 weeks is more accurate than a “certain” menstrual date. Therefore, estimated delivery date based on LMP is best confirmed with ultrasound examination.

Sonographic estimation of gestational age is most accurate early in gestation. Between 7 and 16 weeks’ gestation, ultrasound crown–rump length provides a more accurate estimate of gestational age than certain LMP and should be used to date pregnancy when available. Between 13 and 16 weeks, biparietal diameter is more accurate. Beyond 16 weeks, normal variation in fetal growth and standard error in ultrasound measurement make sonographic estimation of gestational age increasingly less precise.

If first trimester ultrasound measurement is not available, gestational age based on certain LMP in a woman with regular cycles appears to be at least as accurate as ultrasound examination performed between 16 and 23 weeks’ gestation; however, if the ultrasound estimation differs from the LMP estimation by more than 10 days, then the sonographic estimate should be used.

When LMP is uncertain, cycles are irregular, or clinical assessment of uterine size differs from gestational age based on LMP, then first trimester ultrasound examination is indicated, even in settings where routine first trimester ultrasound is not available. If first trimester ultrasound is not performed in these scenarios, then dating should be based on the earliest second trimester ultrasound estimation. As the fertilization date is known in pregnancies resulting from assisted reproductive technologies such as in vitro fertilization, the expected date of delivery should be based on the known conception date.

2. Use a Triage Area and Triage Assessment Scale

The use of a triage area to assess women presenting to hospital, versus a labour room, has the potential to reduce the number of women receiving oxytocin for augmentation, the rate of epidural analgesia for pain relief, and the duration of the active and second stages of labour, and to improve women’s evaluations of their labour and birth experiences.
Use of a specific screening tool in obstetrical triage or in the home, similar to what has been used in emergency units, can facilitate priority setting, responsiveness and flow through the triage unit. The Obstetrical Triage Screening Scale (OTAS), developed in Canada, is a standardized instrument to assess the need for urgency of assessment and for admission. The percentage of patients admitted to the antenatal or birthing unit decreased from 80% (OTAS 1) to 12% (OTAS 5). OTAS has demonstrated interrater reliability in both tertiary and community hospitals.

3. Confirm the Woman is in Labour

Labour onset can be difficult to diagnose. Women are anxious for labour to begin and thus frequently assume symptoms of latent labour indicate a need to go to hospital. Health education for women and families about when they should go to hospital and what symptoms require urgent action are important components of care during pregnancy.

The diagnosis of active labour requires assessment of uterine activity and cervical status. Description of cervical status should include dilatation, effacement, station, consistency, and position as summarized in a Bishop score (see induction of labour chapter for an example). In the USA women are considered in active labour only when they reach 6 cm dilation. In contrast, SOGC identifies active labour when the women reaches 4 cm. Using the SOGC definitions of labour will help guide the diagnosis but it is important to always look at the total clinical picture to determine need for admission.

<table>
<thead>
<tr>
<th>Labour: first stage</th>
<th>Regular uterine contractions accompanied by cervical dilatation and effacement. The first stage of labour includes the latent and active phases.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Latent phase</td>
<td>Presence of uterine activity resulting in progressive effacement and dilatation of the cervix proceeding to active phase. It is complete when a nulliparous woman reaches 4 cm dilatation and a parous woman reaches 4 to 5 cm. Cervical length is generally less than 1 cm.</td>
</tr>
<tr>
<td>Active phase</td>
<td>Presence of a pattern of contractions leading to cervical effacement and dilatation at 4 cm or greater in a nulliparous woman or 4 to 5 cm dilatation in a parous woman.</td>
</tr>
<tr>
<td>Labour: second stage</td>
<td>Full dilatation to delivery of the baby.</td>
</tr>
<tr>
<td>Passive second stage</td>
<td>Full dilatation but without active pushing.</td>
</tr>
<tr>
<td>Active second stage</td>
<td>Full dilatation with active pushing.</td>
</tr>
<tr>
<td>Labour: third stage</td>
<td>Immediately after delivery of the baby to delivery of the placenta.</td>
</tr>
<tr>
<td>Labour: fourth stage</td>
<td>Immediately after delivery of the placenta to 1 hour postpartum.</td>
</tr>
<tr>
<td>Dystocia</td>
<td>Delayed or arrested progress in labour, irrespective of cause.</td>
</tr>
<tr>
<td>In active first stage</td>
<td>Greater than 4 hours of &lt; 0.5 cm/hour dilatation or no dilatation over 2 hours.</td>
</tr>
<tr>
<td>In active second stage</td>
<td>Greater than 1 hour of active pushing without descent of the presenting part.</td>
</tr>
<tr>
<td>Obstructed labour</td>
<td>No cervical dilatation or descent over 2 hours despite evidence of strong contractions (caput, molding, or measured using an IUPC).</td>
</tr>
</tbody>
</table>

Reproduced from SOGC Clinical Practice Guideline no. 336, with permission of The Society of Obstetricians and Gynaecologists Canada.

4. Determine any Contraindications to Vaginal Delivery

Clinical situations that preclude vaginal delivery or that elevate the risk of labour above the maternal or fetal risk of Caesarean section include such conditions as placenta previa, vasa previa, cord presentation; abnormal fetal lie; prior classical CS or significant uterine surgery; active genital herpes, invasive cervical carcinoma.
Care During the First Stage of Labour

Latent Phase of the First Stage of Labour

Studies have shown that “women presenting to the hospital in the latent phase of labour experience increased obstetrical interventions”, including electronic fetal monitoring, epidural analgesia, oxytocin, and Caesarean section, than those who are admitted in active labour. However, it is unclear whether this is because these women spend longer in the hospital or because women who present earlier have underlying problems. It is important to remember that dystocia cannot be diagnosed before the onset of labour or during the latent phase of labour.

Women may be assessed at home to determine if they are in labour. This supports the woman and enables her to await labour at home.

Women who present to hospital in early labour may be asked to walk around or rest in a prelabour lounge or return for reassessment. Alternately women may be sent home with a plan to return to hospital if their status changes. These changes may include increased pain, rupture of membranes, increased frequency or strength of contractions or if the woman is concerned. It can be beneficial to provide the return to hospital or when to contact care providers instructions in writing. Observation, rest, and therapeutic analgesia are preferable to a more active approach of amniotomy and oxytocin induction.

Time Frames for Stages of Labour

The mean and longest acceptable duration of each phase of labour were established by Friedman in the early 1950s and were based on women who had undergone spontaneous or induced labour and delivery and whose fetuses were in vertex and in breech presentations. Mounting evidence has suggested that the Friedman curve may no longer be appropriate for current labour care. The SOGC CPG on MOL stated the active phase is longer and dilation slower than Friedman’s data. The rate of cervical change ranged from 0.5 to 0.7 cm/hour for nulliparas and 0.5 to 1.3/hour for parous women.

Length of spontaneous labour in women with good perinatal outcomes (n >200,000) varied widely. Starting from 4 cm dilation median length of labour varied from 3.7-5.9h (95th percentiles: 14.5-16.7h). The Consortium on Safe Labour looked at 62,415 women in 19 hospitals in the USA. Data showed labour progress can be slow up to 6 cm and after 6cm multiparas progressed faster than nulliparas. Second stage length varied with and without epidurals with the 2nd stage in nulliparas lasting 3.6 and 2.8 hours, respectively.

Enema

A 2007 Cochrane review showed that enemas did not have a significant effect on women’s satisfaction or on infection rates (e.g., for perineal wound infection or other neonatal infections) and concludes that routine use of enemas during labour should be discouraged.
Active Phase of the First Stage of Labour

Partogram (also called Partograph)

A partogram is a graphic labour curve used to document cervical change and fetal descent over time, some partograms include 2 and 4 hour action lines. A 2018 Cochrane review of 11 trials (n=9475) found no benefit of partogram use related to CS rate, oxytocin augmentation or Apgar <7 at 5 minutes. Although there is little demonstrated value in clinical outcomes, the partogram is useful as a communication tool, for handover, for teaching, and to provide a snapshot of labour progress.

Ambulation and Position

Women’s position for labour may be influenced by mother’s choice, caregiver preference, and place of birth, monitoring or medical interventions. Encouragement of movement, position change and upright positions may reduce the length of the first stage of labour and reduce pain.

A 2017 Cochrane review of women without epidural anaesthesia compared women labouring in an upright position (walking or upright non-walking, e.g., sitting, standing, kneeling, squatting, and all fours) versus in recumbent positions (supine, semi-recumbent, and lateral). Authors’ state results should be viewed with caution due low quality of the research. Researchers found women in an upright position had no difference in CS, serious perinatal tears, had first stages that were approximately one hour shorter, and were less likely to have epidural analgesia. However, there was no difference in length of the second stage, mode of delivery, or other outcomes related to the well-being of mothers and babies. For women who had epidural analgesia, there were no differences between those randomized to upright positions and those in recumbent positions for any of the outcomes examined in the review.

Several authors proposed hands and knees position for correction of a posterior presentation. A randomized trial assessed this approach (n=439) for 10 minutes in first stage demonstrated no benefit in conversion to an anterior occiput position but a slight increase in comfort.

Having a comfortable chair in the labour room, not just a bed, encourages flexibility of positions and movement and removes the assumption that the woman has to be in bed.

Eating and Drinking During the First Stage of Labour

A 2013 Cochrane review on restricting oral fluid and food intake during labour (5 studies, n = 3130) looked at women in active labour and at low risk of requiring general anaesthesia. The studies compared restricting women to nothing except ice chips / sips of water or providing women with carbohydrate drinks, specific fluids and foods, or the freedom to eat and drink at will. No significant differences were found in the rate of Caesarean section or assisted vaginal deliveries, or in Apgar scores. The pooled data were insufficient to assess the incidence of the rare outcome Mendelson’s syndrome (aspiration associated with general anaesthesia). The authors concluded that there was no evidence of benefits or harms associated with restricting access to fluid or food for women at low-risk of requiring general anaesthesia.
Women at low risk of requiring general anaesthesia should have the choice to eat or drink as desired or as tolerated in labour. There have been no studies assessing women with risk factors, so there is no evidence with respect to restrictions in this group.

Different solutions and volumes of intravenous fluids do not influence the length of labour in nulliparas. There was no significant difference in the time from enrolment to delivery, second stage duration, and cord artery pH and glucose level.

Amniotomy/Artificial Rupture of Membranes (AROM)

Rupture of membranes appears to affect uterine contractility and cervical dilation through prostaglandin then endogenous Oxytocin synthesis. However, a 2013 Cochrane review of 15 studies with 5583 women showed no clear statistically significant difference between women with amniotomy alone versus intention to preserve the membranes re length of the first stage of labour, caesarean section, maternal satisfaction with birth or Apgar score less than seven at five minutes.

A 2012 Cochrane review of 14 RCTs (n = 8033 women) compared early amniotomy and oxytocin for the prevention or treatment of dystocia. A modest reduction in the duration of labour and CS rate was found, with no difference in other measures of neonatal and maternal morbidity. A smaller RCT (n = 752) comparing early versus late amniotomy without oxytocin found a higher frequency of severe variable fetal heart rate decelerations and CS for abnormal fetal heart rate, yet no difference in overall CS rate or neonatal outcome.

Although the protocols and settings are heterogeneous, routine amniotomy in normally progressing labour is of questionable benefit and may result in more frequent variable fetal heart rate decelerations, and potentially increased CS rate. Amniotomy does appear indicated to accelerate labour that is not progressing adequately.

If amniotomy is performed, it is necessary to ensure that the fetal head is well-applied to the cervix (not ballot table) to minimize the risk of cord prolapse. If the head is well applied following amniotomy, a woman may ambulate. Amniotomy with oxytocin augmentation, and other measures, should be considered if the diagnosis of dystocia has been made.

Active Management of the First Stage of Labour

The active management of labour is a complex process that originally included concrete criteria for the diagnosis of labour (regular contractions with spontaneous rupture of membranes or complete cervical effacement), routine amniotomy in labour, close attention to progress in labour, and liberal use of high-dose oxytocin if cervical dilation was less than 1 cm per hour. The maximum allowable duration of the first stage of labour was 10 hours, and of the second stage, 2 hours. Observational studies by the initial advocates of active management showed lower CS rates, a lower incidence of prolonged labour, better neonatal outcomes, and improved maternal satisfaction. Follow-up observational studies supported these results. A 2013 Cochrane review (7 trials of 5390 low risk women) showed active management was associated with small reductions in the CS rate when compared with various regimes of routine care.
Active management requires more interventions and creates a more medicalized birth process. It is possible that some components of the active management package are more effective than others. Of note, RCTs demonstrating no increase in CS rate with epidural analgesia compared with parenteral opioids routinely used all components of active management of labour protocols.

Care During the Second Stage of Labour

Position

With epidural: A 2018 Cochrane compared women using a recumbent (left, right or semi recumbent – not on their back or legs raised fin stirrups) position with women in an upright position (8 studies n=4464) showed variations in findings. When only the high quality studies (3 trials n=3609), primarily from a 2017 UK trial BUMPES (n=3236), were reviewed they reported increased operative and CS births with an upright position. Women with a recumbent (left, right or semi recumbent) had lower risk of operative birth (CS or AVB). The UK data specifically showed 41.1% SVD in the recumbent position and 35.2% in the upright position. There was no difference in perineal trauma, or significant haemorrhage, In Canada, the epidural rate for vaginal births is 49.7%.

Without epidural: A 2017 Cochrane review (30 studies, n= 9015) compared women using an upright position versus supine positions. The authors caution that much of the evidence was low quality. The upright position demonstrated:

<table>
<thead>
<tr>
<th>INCREASED</th>
<th>DECREASED</th>
<th>NO DIFFERENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Estimated blood loss</td>
<td>• Length of second stage however significant variation between studies, mainly decreased in primigravid group</td>
<td>• Rate of CS, 3rd or 4th degree perineal tears, Babies admitted to NICU</td>
</tr>
<tr>
<td>• AVD, moderate</td>
<td>• Abnormal FHR patterns</td>
<td></td>
</tr>
</tbody>
</table>

A 2015 study of 7832 vaginal deliveries found that squatting or kneeling was associated with an elevated risk of anal sphincter trauma and that delivering in water was not associated with a greater risk of AST than delivering in bed.

There is no perfect position for labour for everyone. Enabling the woman to select her position of comfort, assuming it is clinically safe, often provides the greatest benefit.

When to Push

Each labour is unique thus the timing of pushing and woman’s feeling of an urge to push will differ for everyone. Pushing, generally, may commence when the cervix is fully dilated, the presenting part is engaged and the woman feels an urge to push. Passive descent from full dilatation to the perception of an urge to push, delayed pushing, may be less exhausting and more effective, particularly for women with epidural analgesia. Delayed pushing is preferred when the
woman has no urge to push; particularly if the presenting part is above station +2 or in non-occiput anterior position, the fetal surveillance is normal and the woman’s status is satisfactory.\(^{21}\)

However, a 2018 study compared delayed versus immediate pushing in 2414 nulliparous women with epidurals and term pregnancies.\(^{56}\) There was no significant difference in SVD (85.9% to 86.5%), neonatal morbidity, or perineal lacerations. The immediate pushing group had mean difference of 9 minutes longer pushing time, lower rate of chorioamnionitis and PPH.

### How to Push

Spontaneous pushing encourages women to follow their body’s urges and generally push 3-5 times during a contraction. In Valsalva or directed pushing the mother is asked to take a deep breath, hold the breath (closed glottis), and push downward when uterine contraction starts.

It was suggested that spontaneous pushing allows a slower and controlled descent of the fetus.\(^{57}\) Spontaneous pushing compared to direct pushing showed no difference in overall length of 2nd stage, 3rd or 4th degree laceration/episiotomy, spontaneous vaginal delivery or neonatal outcomes (cord gases, resuscitations).\(^{58}\) Valsalva pushing negatively affected urodynamic factors measured at 3 months post partum.\(^{59}\)

A 2017 Cochrane review summarized their finding with the comment “for the type of pushing, with or without epidural, there is no conclusive evidence to support or refute any specific style as part of routine clinical practice, and in the absence of strong evidence supporting a specific method or timing of pushing, the woman’s preference and comfort and clinical context should guide decisions”.\(^{58}\)

### Length of second stage\(^{43}\)

Limited evidence and multiple conflicting variables making setting a specific time limit for the second stage difficult. ACOG and SMFM in United States published an Obstetric Care Consensus extending time frames in labour and second stage with a view to prevention of Caesarean Section.\(^{60}\) They indicated that a specific absolute maximum length of time spent in the second stage of labour beyond which all women should undergo operative delivery has not been identified.

It is important to distinguish dystocia from obstructed labour when managing the second stage.

Provided there is ongoing progress and fetal status is normal, arbitrary time limits for the second stage are not well founded.\(^{61}\) Thus it may be appropriate to avoid early intervention with operative delivery if fetal surveillance is normal. There is no evidence that a prolonged passive second stage with poor contractions is harmful to the fetus, so time could be taken to achieve adequate contractions using oxytocin, if necessary. Time limits should be initiated when contractions are adequate and active pushing begins. However, continued strong contractions without progress in the second stage indicates obstructed labour and place the fetus and mother at risk.\(^{62}\)

Studies report increased asphyxia related complications and low Apgar scores at 5 minutes when women pushed ≥ 4 hours compared to women who pushed ≤ 1hour however the overall risk of concerns was low.\(^{63}\)\(^{64}\)
Table 4. Recommended Practices in Second Stage by Parity and Use of Epidural Analgesia
(after full dilatation and when power is adequate)

<table>
<thead>
<tr>
<th>NULLIPAROUS</th>
<th>PAROUS</th>
</tr>
</thead>
<tbody>
<tr>
<td>No epidural</td>
<td>No epidural</td>
</tr>
<tr>
<td>Epidural</td>
<td>Epidural</td>
</tr>
<tr>
<td>Total duration¹</td>
<td>3 hours</td>
</tr>
<tr>
<td>Passive 2nd stage²</td>
<td>May wait up to 2 hours before pushing particularly when the presenting part is above +2 station or in a non-occiput anterior (OA) position, and the urge to push is absent. Encourage waiting to allow passive descent.</td>
</tr>
<tr>
<td>Commence pushing</td>
<td>When urge to push present and not able to allow passive descent OR after 2 completed hours of passive second stage</td>
</tr>
<tr>
<td>Assessment</td>
<td>Hourly for descent and position. Reassess the need for assisted birth after 2 hours of active pushing.</td>
</tr>
</tbody>
</table>

¹Continuing beyond the following time limits should be carefully evaluated and consideration given to expedite delivery. Extending these time limits may be appropriate in the presence of continued descent of the head, maternal status and fetal surveillance is normal, and spontaneous vaginal birth is imminent.

²Waiting for up to the time period indicated prior to the onset of pushing is appropriate in the presence of continued descent of the head and fetal and maternal condition are satisfactory.

Episiotomy and Perineal Management

Routine episiotomy does NOT reduce perineal / vaginal trauma. A 2017 Cochrane examined selective vs routine episiotomy with 12 studies (n=6177 women) from a variety of countries including Canada. In situations when unassisted vaginal birth was anticipated, selective episiotomy may result in 30% fewer women experiencing severe / perineal trauma. There may be little or no difference in dyspareunia and urinary incontinence at 6 months with selective vs routine episiotomy. Further research is suggested for women when instrumental delivery is intended.

Strategies to reduce perineal trauma could include:

- warm compresses: A 2011 Cochrane review (n = 1525) of warm perineal compresses in labour versus no intervention showed a reduction in third and fourth degree perineal tears from 5% to 2.5%. A 2013 Cochrane review (n = 2500) of antenatal perineal massage showed modest reductions in episiotomy rate, perineal trauma, and pain.
- Episiotomy angle: The angle at which a mediolateral episiotomy is cut is important. An observational study showed that the angle of an episiotomy from midline performed when the perineum is distended decreased by 20 degrees on average after delivery. An episiotomy performed at 45 degrees leaves an incision after delivery at 25 degrees from midline–dangerously close to the anal sphincter. To avoid causing OASI, an episiotomy should be performed more than 60 degrees from midline when the head is crowning (less than 30 degrees from horizontal). An episiotomy cut at 60 degrees results in a post birth episiotomy angle of...
45 degrees. Midline episiotomy increases the risk of anal sphincter injury, and should not be performed (Figure 3). 

Perineal infiltration is used for repair of lacerations and episiotomy repair. Generous and widespread infiltration should be used. Use of an agent with epinephrine is helpful. Care should be taken not to inject intravascularly or to exceed the toxic dose.

There was insufficient evidence to assess the clinical benefits or harms of routine antibiotic prophylaxis for episiotomy repair after normal birth. 

Figure 3. Episiotomy

![Episiotomy Diagram](image-url)
Risk of obstetrical anal sphincter injury (OASI)

Manual perineal protection was effective in nulliparous women in reducing the risk of OASI by two thirds. A retrospective Australian study found the only statistically significant increase in OASI over time was with non-assisted vaginal births without episiotomy (linear trend $P < 0.001$) and forceps with episiotomy (linear trend $P = 0.004$). Authors of a population study of 113,279 Swedish women (singleton pregnancy, SVD without episiotomy) determined birth position affected rate of OASI. Regardless of parity, the lowest OASI rates were found among women giving birth in standing position and the highest rates among women birthing in the lithotomy position. Birth seat and squatting position involved an increased risk of OASI among parous women.

Although routine episiotomy has been shown to cause more harm than good, selective mediolateral episiotomy should be considered in women considered at increased risk of obstetrical anal sphincter injury such as nulliparous women requiring assisted vaginal birth or those with a history of prior OASI. Randomized evidence is limited but corroborates observational evidence that more liberal (but not routine) use of mediolateral episiotomy prevents OASI at assisted vaginal births, particularly with forceps.

Quality improvement efforts in large regions of Norway and Denmark over the past decade have succeeded in reducing the incidence of OASI from 4% to less than 2%. The key components have been controlled slow delivery of the crowning fetal head and selective use of mediolateral episiotomy (overall rates between 10% and 20%).

Perineal Repair

- A 2010 Cochrane review involving 10,171 women showed decreased pain and need for resuturing with synthetic versus catgut suture material for episiotomy and perineal tear repairs.
- Rapidly absorbable synthetic suture needed to be removed less often than regular synthetic suture. There were no differences in long-term pain or dyspareunia.
- A 2012 Cochrane review involving 8,184 women showed decreased analgesia use, less pain for up to 10 days, and reduced need for suture removal with continuous versus interrupted perineal repair technique. There was no difference in long-term pain or need for re-suturing.
- A randomized trial of 147 women with third or fourth degree tears found a single 1 g IV dose of an anti-anaerobic cephalosporin (cefotetan or cefoxitin) given during repair reduced the incidence of perineal complications from 24% to 8% ($P = 0.037$).

Management of The Third Stage of Labour

Active management of the third stage of labour (AMTSL) involves interventions by the care provider to assist in the expulsion of the placenta to prevent or decrease blood loss. Personnel with the training and skills to actively manage the third stage should be in attendance. The most important component of the active management of the third stage of labour is routine administration of oxytocin.

Further information on third stage management can be found in the Postpartum Hemorrhage chapter.
Dystocia

Dystocia is one of the most common labour problems. Dystocia cannot be diagnosed prior to the onset of the active phase of the first stage of labour or before the cervix is at least 4 cm dilated.

Dystocia is defined as delayed or arrested progress in labour, irrespective of cause. Using the 5th percentile of the population as a cut off, dystocia is identified when any of the following are seen:

- Dilation of less than 0.5 cm/hour over 4 hours or
- No cervical change over 2 hours in the active first stage of labour or
- Greater than 1 hour of active pushing and no descent of the presenting part in the second stage of labour.

Etiology of Dystocia

Successful labour and vaginal delivery depend upon the dynamic relationship between the fetus, the maternal pelvis, and uterine and maternal power. Dystocia can be related to difficulty with any of the 4 Ps:

- power, passenger, passage, and psyche.

**Power**

Contractions: hypotonic, incoordinate or infrequent
Maternal expulsive efforts: ineffective or contraindicated

During labour, palpation of the uterus is recommended to assess uterine contraction strength. However, palpation and external tocometry provide only subjective, qualitative estimates of intensity. Indirect clinical evidence of adequate contraction strength may be obtained by assessing caput and moulding. An intrauterine pressure catheter (IUPC) is the only accurate way of assessing the intensity (in mm Hg) of contractions. Its use is not indicated routinely but may be helpful in certain situations such as

- augmentation during a trial of labour after previous Caesarean section or in the case of grand multiparity (> 5).86
- when it is not possible to palpate contractions because of high BMI or body habitus

When using an IUPC, contraction strength is measured with a goal of an increase of at least 50 to 60 mm Hg above baseline or > 200 Montevideo units (uterine contraction pressure above baseline times number of contractions in 10 minutes). A standard institutional protocol for the augmentation of labour is recommended if the strength of uterine contractions is inadequate.

Epidural analgesia can interfere with uterine contractions and lead to dystocia requiring oxytocin augmentation.87 Early detection and treatment with active management of labour protocols and oxytocin augmentation can reduce the likelihood of CS and operative delivery.44 87

**Passenger**

Fetal-position, attitude, size, abnormalities (e.g., hydrocephalus)

The fetus should be assessed for size and malposition.88 Adequate power in labour will often correct malposition, whereas inadequate power may result in persistent malposition. A normal-sized fetus may also present an increased diameter to the pelvis because the head is not flexed or is asyntotic. Adequate uterine power may overcome this
problem. Use of hands-and-knees position for 10 minutes twice daily was not found to correct an occiput posterior fetal position in late pregnancy. 89

The diagnosis of true or absolute cephalopelvic disproportion should be limited to the uncommon instances of real disproportion, i.e., inability of the well-flexed head (sub-occipito bregmatic presentation) to pass through the bony pelvis despite evidence of adequate uterine power (caput and moulding present and/or IUPC demonstrates adequate contractions). Malposition, on the other hand, may lead to relative disproportion. Accurate assessment and description will guide management, and if a CS is performed for dystocia, helps determine suitability for a future VBAC.

**Passage** Pelvic bony structure

Soft tissue factors (tumours, full bladder, full rectum, vaginal septum, high BMI)

Clinical examination of the passage may reveal prominent spines or sacrum, a narrow pubic arch or a space-occupying mass in the pelvis, such as fibroids. Neither antenatal X-ray pelvimetry nor clinical pelvimetry has been shown to predict the outcome of labour. 90 It is important to ensure the bladder is empty, particularly in the second stage. Soft tissue obstruction may occur in patients with a high BMI.

**Psyche** Pain, anxiety

“Hormones released in response to stress can also bring about dystocia. Sources of stress vary for each woman, but the pain and the absence of a support person are the 2 accepted factors. Confinement to bed and the restriction of maternal movement can also be a source of psychological stress. Anxiety may inhibit normal cervical dilatation, resulting in prolonged labour and increased pain perception. Anxiety also causes an increase in the levels of stress-related hormones (ß-endorphin, adrenocorticotropic hormone, cortisol, and epinephrine). These hormones act on the smooth muscle of the uterus; increased levels can lead to dystocia by reducing uterine contractions.” 91

**Prevention of Dystocia**

Strategies to prevent dystocia include

- Labour preparation
- Continuous support during labour
- Careful labour assessment (consider a partogram)
- Use of appropriate analgesia
- For women with epidural analgesia, if inadequate labour progress is suspected, early artificial rupture of membranes and oxytocin for uterine inertia

**Management of Dystocia**

If a dystocia is suspected in the first stage of labour, management may include

- Formulating, discussing, and documenting a plan
- Amniotomy
Ensuring adequate maternal hydration
Considering therapeutic rest with analgesia, if exhausted
Oxytocin augmentation
If clinical assessment of contraction strength inadequate, considering IUPC
CS (if other interventions have failed)

**Dystocia versus obstructed labour**

It is important to distinguish dystocia from obstructed labour

- **Dystocia** is a lack of adequate progress in labour due to any of 4 causes: power, passenger, passage, and psyche.
- **Obstructed labour**, a form of dystocia, is lack of cervical dilation or descent of the presenting part despite adequate power, i.e., evidence of adequate uterine contractions and/or maternal effort.

When dystocia is diagnosed, a common cause is inadequate uterine contractions and oxytocin augmentation is often indicated. Once strong contractions have been present for 2 to 3 hours without further cervical dilation in the first stage or for 1 to 2 hours without descent in the second stage, significant caput and moulding are usually present.

- If caput and moulding are absent, consider verifying contraction strength using an intrauterine pressure catheter (see management of dystocia below).
- If caput and molding are present, labour is obstructed.

A 2016 Canadian study used mathematical modelling of both dilation and descent to better predict the need for CS intervention.

**Dystocia mandates a search for a cause, then treatment.**

**Obstructed labour is an indication for Caesarean section or assisted vaginal birth, if safely feasible.**

In the event of dystocia due to inadequate power despite appropriate analgesia, hydration, rest, and amniotomy, oxytocin is indicated to achieve adequate contractions before operative delivery is considered. Principal concerns about the use of oxytocin are fetal compromise and uterine rupture. It must be remembered that it is not oxytocin that causes the problem but rather excessive contractions. The correct use of oxytocin achieves adequate contractions while avoiding excess uterine activity that could compromise the fetus or lead to uterine rupture.

**Even with close titration of oxytocin, tachysystole / excess uterine activity can occur. All labour and delivery units must be prepared to manage excess uterine activity whether it is associated with oxytocin use or not. This is outlined in the chapter on induction of labour and fetal health surveillance.**
Complications of oxytocin

<table>
<thead>
<tr>
<th>POSSIBLE COMPLICATION</th>
<th>MECHANISM</th>
<th>PREVENTATIVE MANAGEMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fetal compromise</td>
<td>Tachysystole</td>
<td>Correct dose</td>
</tr>
<tr>
<td>Uterine Rupture</td>
<td>Tachysystole</td>
<td>Correct dose</td>
</tr>
<tr>
<td>Water intoxication</td>
<td>ADH effect</td>
<td>Use crystalloid IV solution with concentrated oxytocin to limit amount of water infused</td>
</tr>
</tbody>
</table>

Sensitivity to oxytocin

Each woman’s uterus varies in its sensitivity to oxytocin. Even in the same uterus, the sensitivity may change during the course of labour. The dose used must be sufficient to achieve adequate contractions. Protocols or guidelines for the administration of oxytocin vary but many suggest starting with a low dose and small increments at intervals of 30 minutes. Starting incremental dosages for augmentation may be less than those for induction. Every obstetrical unit must have an identified and accessible protocol that will include a starting dose, increment interval, and maximum dose allowed before reassessment. Electronic fetal monitoring is initiated or continued when oxytocin is started.

Intravenous oxytocin can be administered by low- or high-dose protocol. The choice of protocol should be based on the relative risks of uterine rupture, sensitivity to oxytocin, and likelihood of placental compromise. The oxytocin protocol must be based on the uterine response and fetal capacity to tolerate the increase uterine activity generated an early rapid uterine response is noted with a high-dose protocol or there are concerns about placental function, then a low-dose protocol should be used. Using a consistent augmentation/induction protocol (whether low or high dose) within a facility reduced the overall Caesarean section rate in nulliparous women from 35.5% to 24.5% OR −0.58.

A 2010 meta-analysis of RCTs including 5400 women concluded that high-dose oxytocin protocols were associated with a lower CS rate than low-dose protocols. Excess uterine activity was more common with high-dose protocols, but there was no difference in maternal or neonatal morbidity. Low dose was defined as 1 to 2 milliunit (mU)/min increments and high dose was defined as ≥ 4 mU/min increments, increased every 30 min. This meta-analysis was dominated by studies that included only nulliparous women. Evidence specific to parous women or women with a prior Caesarean section scar is lacking. Therefore, it is prudent to use a low-dose protocol for labouring women with a prior Caesarean section or other uterine surgery because of a potential increase in the risk of uterine rupture. It is also prudent to use a low-dose protocol in women with grand multiparity because of their increased sensitivity to oxytocin. Women with suspected fetal growth restriction should also receive low-dose oxytocin because of increased risk of placental insufficiency with strong uterine contractions.

Nulliparous women and women with epidural analgesia in labour are particularly likely to benefit from high- rather than low-dose oxytocin augmentation. A small Canadian pilot RCT of accelerated Oxytocin titration protocol (AOT or high dose 4mU/min) vs gradual Oxytocin Titration (GOT or low dose 2mu/min) demonstrated a trend to lower CS in the AOT group.
In nulliparas with epidural analgesia, to reduce the likelihood of Caesarean sections, the clinical goal of augmentation is 4 to 5 contractions in 10 minutes. If IUPC is used, then the goal is 200 to 250 Montevideo units (e.g. 4 contractions of 50 to 60 mmHg in 10 minutes). 44

Example of Low and High Dose Protocol

<table>
<thead>
<tr>
<th></th>
<th>LOW DOSE PROTOCOL</th>
<th>HIGH DOSE PROTOCOL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial dose of oxytocin</td>
<td>1 to 2 mU/min</td>
<td>2 to 4 mU/min</td>
</tr>
<tr>
<td>Increase interval</td>
<td>30 minutes</td>
<td></td>
</tr>
<tr>
<td>Dosage increment</td>
<td>1 to 2 mU</td>
<td>4 mU/min</td>
</tr>
<tr>
<td>Usual dose for good labour</td>
<td>8 to 12 mU/minute</td>
<td></td>
</tr>
<tr>
<td>Maximum dose before reassessment</td>
<td>20 to 30 mU/min</td>
<td>49, 100</td>
</tr>
</tbody>
</table>

CLINICAL CONSIDERATIONS

<table>
<thead>
<tr>
<th>Prior Caesarean section</th>
<th>low dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fetal growth restriction</td>
<td>low dose</td>
</tr>
<tr>
<td>Parity &gt; 4</td>
<td>low dose</td>
</tr>
<tr>
<td>Parous augmentation, no epidural</td>
<td>low dose</td>
</tr>
<tr>
<td>Parous induction</td>
<td>low or high dose</td>
</tr>
<tr>
<td>Parous augmentation with epidural</td>
<td>low or high dose</td>
</tr>
<tr>
<td>Nullipara augmentation, no epidural</td>
<td>low dose</td>
</tr>
<tr>
<td>Nullipara induction</td>
<td>low or high dose</td>
</tr>
<tr>
<td>Nullipara with epidural</td>
<td>consider high dose</td>
</tr>
</tbody>
</table>

Most cases of uterine inertia respond to oxytocin doses under 10 mU/min, but individual needs vary greatly. Many women receiving high-dose oxytocin will never require 10 mU/min and may need finer titration by 1 to 2 mU/min when they approach adequate uterine activity. Clinical judgement is required. There is no evidence suggesting a maximum safe dose; however, reassessment is recommended when the dose reaches 20 to 30 mU/min, and very close monitoring should be employed for doses > 30 mU/min, including IUPC if necessary. 99
Second Stage Labour Dystocia

Close attention to contraction strength and progress in the second stage is necessary. Inadequate or infrequent contractions should prompt oxytocin augmentation, similar to the first stage of labour. It is uncommon for a parous woman to require oxytocin augmentation and caution is advised. Fetal well-being must be assured.

It continues to be important to differentiate dystocia and obstructed labour during second stage.

There are 3 common causes: uterine inertia, fetal malposition, and relative or absolute cephalopelvic disproportion. Differentiating between these causes is essential to assessing fetal risk and determining appropriate management in a prolonged second stage.

If no urge to push occurs after 1 hour of second stage, assess fetal position, caput and moulding, and uterine contraction strength. Consider initiating or increasing oxytocin if contractions are felt to be inadequate. There is no evidence that a prolonged passive second stage with poor contractions is harmful to the fetus. Time limits should be initiated when contractions are adequate and active pushing commences. Oxytocin is often required when epidural analgesia is present. Recommendations allowing a longer second stage in women with epidural analgesia take into account the epidural’s inhibitory effect on uterine contraction strength and maternal motor function.

Adequate uterine power will also often correct fetal malposition. Placement of an intrauterine pressure catheter should be considered if contractions are difficult to assess, although insertion can be challenging at full dilatation.

In the active second stage, if contractions are clearly adequate (with or without oxytocin), significant caput and moulding are present, and there is no progress over 2 hours for a nullipara or 1 hour for a multiparous woman, then labour is obstructed. Assisted vaginal delivery or Caesarean section should be considered. A prolonged second stage in obstructed labour with adequate contractions can be harmful. A population-based, cohort analysis of 120 000 women found an increase in the risk of maternal obstetric trauma, postpartum hemorrhage, puerperal febrile morbidity, composite maternal morbidity, low 5-minute Apgar score, birth depression, admission to the neonatal intensive care unit, and composite perinatal morbidity as the duration of the second stage surpassed 3 hours in nulliparous women and 2 hours in parous women.

A 2015 meta-analysis of 12 studies showed that the risks of uterine incision extension, infection, mean blood loss, and operative time were significantly higher with the push technique than with the reverse breech extraction.

Avoiding a Primary Caesarean Section

The increasing worldwide Caesarean Section (CS) rate is concerning with its associated maternal morbidity and mortality, health care costs, and potential future pregnancy concerns. Prevention of the primary CS as well as encouragement of Trial of Labour after Caesarean (TOLAC), in appropriately screened cases, has been the focus for rate reduction. There are many clinical situations that require a CS for maternal and/or fetal well being however there are many occasions when a CS is done for less than ideal reasons.
Women with first delivery by CS have an increased risk of severe complications in the second pregnancy. A Nordic study (N=213,518), comparing women whose first delivery was vaginal or CS, found that abnormally invasive placenta, uterine rupture and severe postpartum haemorrhage were more common in women with a first CS.

Thus focus of labour management should be to provide a safe labour and birth and be attentive to avoiding the primary CS. Optimal support of normal labour processes, appropriate management of dystocia, and consistency of clinical guidelines and encouragement of TOLAC could lead to a reduction in the CS rate.

**Caesarean Section Rates**

The overall Canadian CS rate (primary and repeat) 2016/2017 was 28.2% of births with the highest rate in British Columbia. The rate of CS in the USA for the same period was 32% with highest numbers in the southern states i.e. Mississippi 37.8%, Louisiana 37.5%, Florida 37.2%. The global CS rate has moved from 12% in 2000 to 21% in 2015.

**Comparison of CS rates by Province**
Causes of Caesarean Sections

Many caregivers rationalize their CS rate based on perceived specific characteristics of their population (e.g.: % of high risk, higher BMI) and not care practices. The 10-Group Classification System, developed by Robson, can be used to analyze CS deliveries in a facility or area and compare rates in similar situations. This classification system is being used increasingly around the world to focus attempts to control CS rate in Group 1 (nulliparous women in spontaneous labour at term) and Group 5 (women with a prior CS at term). Conducting regular reviews/audits of indicators for CS have provided better understanding of reasons for CS and the potential reduce the rate of CS.

The following table outlines the Modified Robson criteria developed by SOGC committee opinion.

<table>
<thead>
<tr>
<th>DESCRIPTION</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Nullipara, singleton cephalic, ≥ 37 weeks, spontaneous labour</td>
</tr>
</tbody>
</table>
| 2 | Nullipara, singleton cephalic, ≥ 37 weeks  
A: Induced  
B: Caesarean section before labour |
| 3 | Multipara, singleton cephalic, ≥ 37 weeks, spontaneous labour |
| 4 | Multipara, singleton cephalic, ≥ 37 weeks  
A: Induced labour  
B: Caesarean Section before labour |
| 5 | Previous CS, singleton Cephalic, ≥ 37 weeks  
A: spontaneous labour  
B: Induced labour  
C: CS before labour |
| 6 | All nulliparous Breeches  
A: spontaneous labour  
B: Induced labour  
C: CS before labour |
| 7 | All multiparous breeches (including previous CS)  
A: spontaneous labour  
B: Induced labour  
C: CS before labour |
| 8 | All multiple pregnancies (including previous CS)  
A: spontaneous labour  
B: Induced labour  
C: CS before labour |
Summary

Labour is a physiological process. Normal labour is enhanced by appropriate latent phase management (e.g., maternal fetal assessment, information and a plan), delayed admission until active labour, continuous support in labour, comfort measures and appropriate pain management, and intervention only when necessary.

Dystocia can be diagnosed only when active labour is established. Dystocia mandates a search for a cause, then treatment. Management may include adequate analgesia, hydration and rest, amniotomy, augmentation, and assisted vaginal birth.

- Obstructed labour is an indication for CS or assisted vaginal birth, if they can be safely preformed.
- For women having epidural analgesia, uterine inertia and inadequate labour progress should be detected early and augmented with ARM and oxytocin.
- Operative intervention should be undertaken when necessary.

Best care in labour takes into account the wishes of the woman, her individual clinical situation, and evidence informed practice. Labour management should support the physiological process of birth while identifying potential concerns and supporting interventions that will increase the likelihood of a vaginal birth.
**Labor Dystocia Flowchart**

**Active Labor?**
- Regular Contractions
- Cervix > 90% effaced
- Cervix ≥ 4 cm dilated
  - **Yes:** Assess Progress
  - **No:** Comfort measures +/- sedation

**Assess Progress**
- < 2 cm in 4 hrs or
  - No cervical dilation in 2 hours, or
  - No descent over 1 hour in 2nd stage
  - **Poor Labor Progress**

**Adequate Power?**
- 4-5 contractions in 10 minutes?
  - Palpate moderate to strong?
  - Caput and molding?
  - **Yes:** Good Progress
  - **No:** Delivery

**Uncertain:**
- Obesity?
- Prior c/s?
- Epidural?
- Tachysystole?
- Difficult Torch pick-up

**No Progress**
- Consider another 60 min to be sure

**Obstructed Labor**
- Consider IUPC

**Operative Delivery**
- >200 Montevideo units (e.g., 40 x 10 mmHg contr in 10 minutes) x 2 hrs without progress
  - 1st stage: 1 hr without progress 2nd stage

**Progress?**
- **Yes:** Delivery
- **No:**

---

**Management of Labour**

---

84
References


109. Health Indicators Interactive Tool.


Local infiltration of the pudendal nerve. Transvaginal technique showing the needle extended beyond the needle guard and passing through the sacrospinous ligament (S) to reach the pudendal nerve (N).

# Table of Contents

Chapter 5 Support and Pain Management in Labour ................................................................. 96

- Prenatal Education .................................................................................................................. 97
- Continuous Support ............................................................................................................... 97
- Non-Pharmacological Pain Relief .......................................................................................... 97
- Pharmacological Methods .................................................................................................... 99
  - Opioids ................................................................................................................................ 100
- Care and Management in the Second Stage of Labour ......................................................... 104
  - Epidural Analgesia .............................................................................................................. 104
Chapter 5
Support and Pain Management in Labour

Women experience a wide range of pain during labour. The severity and tolerance of pain is unique to each labouring woman and cannot be predicted reliably prior to its occurrence. Two themes emerged from a qualitative meta-analysis of 10 studies regarding women's ability to cope with pain:¹ – the importance of individualised, continuous support and an acceptance of pain during childbirth.

As pain and a woman’s response to it are individual, women need to be aware of a variety of strategies to assist with their management of pain. Using a pain scale to assess pain in labour helps to determine the need for offering interventions and the effectiveness of those interventions. It has been suggested that in addition to a pain scale, a coping scale be used. This differentiates those women able to cope with significant pain from those requiring intervention.²

Some women in labour reach the limit of their pain tolerance. Women experiencing excessive pain or anxiety have high endogenous catecholamines.³,⁴ This produces a direct inhibitory effect on uterine contractility and establishes a vicious circle of poor uterine progress leading to increased anxiety leading to increased catecholamines leading to further impairment of progress. The relief of pain by effective support and analgesia may allow release of the uterus from the constraints of the endogenous catecholamines and allow progress in labour. High endogenous catecholamine levels may also adversely affect uterine blood flow and therefore fetal oxygenation.⁵

The management of pain during labour involves more than the simple and timely administration of the best analgesic agent available. Successful control of pain in labour requires active management of the entire process. This should begin with prenatal education and counselling. Measures to enhance comfort and reduce apprehension are required for the care of all women in labour. If appropriate measures are used early in the process of labour, analgesic needs decrease. Those people who care for women in labour need to be aware of all the available options for the prevention of pain as well as for its management. When the health care team understands the indications, possible variations, and potential side effects, the woman and her family are able to make choices in a less stressful environment.

Satisfaction in childbirth is not contingent upon the absence of pain. Many women are willing to experience some pain in childbirth, but they do not want the pain to overwhelm them. For women whose goals for childbirth include the use of measures to manage pain with minimal or no drug use, and for those who have little or no access to pharmacological methods of pain relief, the non-pharmacological methods of analgesia in childbirth, in combination with continuous support, are integral to labour planning. These measures cannot match epidural analgesia for analgesic effectiveness, but they do help women and are not likely to have harmful side effects.
Prenatal Education

Women and their partners seek prenatal education to help them understand the process of birth and the potential options related to labour, pain relief, infant care, and feeding. A 2007 Cochrane review of nine randomized trials involving 2284 women concluded the effects of prenatal education are unknown and that such education for women who have previously had a CS has had little effect on rates of VBAC. No data were reported concerning anxiety, breastfeeding success, or general social support.

In addition to group and individual classes, many women are now accessing Internet information, reality television programs on pregnancy and birth, and textbook information. The effect of these sources of information has not been fully evaluated.

Continuous Support

Continuous labour support (CLS) is associated with lower use of pharmacological analgesia. Supportive care during labour includes:

- Emotional support - continuous presence, reassurance, and praise
- Information - labour progress and advice regarding coping techniques
- Comfort measures - touch, massage, warm baths/showers, promoting adequate fluid intake
- Advocacy
- Supporting partners in their role as a coach

A 2013 Cochrane review of RCTs involving over 15 000 women in both low- and middle-income settings showed that continuous support in labour increased the likelihood of vaginal delivery, reduced CS rate and the use of epidural analgesia, and improved APGAR scores and maternal satisfaction. “Subgroup analyses suggested that continuous support was most effective when the provider was neither part of the hospital staff nor the woman’s social network.”

Doulas, paraprofessionals employed by some women to provide continuous labour support, were the provider in most of the studies in the review.

Nurses and midwives provide the majority of professional continuous labour support. Nurses identified organizational barriers to CLS that included increased patient acuity and patient: nurse ratio. Studies involving traditional birth attendants suggest improved birth outcomes but are of poor quality, not randomized, and uncertain with respect to cost-effectiveness.

Non-Pharmacological Pain Relief

Smith et al. published a 2006 Cochrane review of complementary and alternative therapies for labour pain management. Evidence of benefit in reducing pain exists for acupuncture (RR 0.70, CI 0.49 to 1.00, 2 trials, n=288), and self-hypnosis (RR 0.53, CI 0.36 to 0.79, 5 trials, n=749). The efficacy of acupressure, aromatherapy, audio analgesia, and massage has not been established. The requirement for more one-to-one care in these situations may have influenced
outcomes. A 2018 Cochrane Review on relaxation techniques for pain management in the active phase of labour found that relaxation techniques, yoga, and music have little efficacy in reducing pain and increasing maternal satisfaction, with low or very low quality of evidence. There was insufficient evidence for mindfulness and audio-analgesia techniques.

Non-Pharmacological Techniques:

- Techniques that reduce painful stimuli:
  - Maternal movement and position change:
    - For women who do not have epidural analgesia, movement, position change, or upright positions may reduce the length of the first stage of labour and reduce pain.
    - Women with epidural analgesia who adopt recumbent or supine positions during the second stage of labour may have little or no difference in operative birth rates.
  - Counter-pressure

- Techniques that activate peripheral sensory receptors:
  - Superficial heat and cold
  - Immersion in water during labour
    - A 2009 Cochrane review identified ten RCTs comparing water immersion during the first stage of labour versus no immersion. There was a reduction in the use of epidural/spinal/paracervical analgesia among women allocated to water immersion compared with controls (OR 0.82, 95% CI 0.70 to 0.98, six trials; n=2499). The authors stated that it was not possible to conclude whether it was the water immersion itself or the other associated care practices such as support from caregivers that was responsible for the apparent benefit. There was no difference in assisted vaginal deliveries, Caesarean sections, perineal trauma, or maternal infection. There were no differences in Apgar score < 7 at five minutes, neonatal unit admissions, or neonatal infection rates.
    - Regarding water immersion in the second stage of labour, there was inadequate evidence to support or not to support a woman’s decision to give birth in water.
  - Hypnosis
    - A 2016 Cochrane analysis (9 studies, n=2954) found only poor quality data related to hypnosis. Insufficient evidence exists regarding satisfaction with pain relief or sense of coping with labour.
  - Acupuncture and acupressure
    - There is mixed and limited evidence on the effectiveness of acupuncture or acupressure. An unblinded RCT (2009) of 607 women compared analgesic use in women treated with acupuncture, transcutaneous electrical nerve simulations (tens), or usual care (including sterile water papules, nitrous oxide, warm tub bath, meperidine, and epidural analgesia). Acupuncture was found to reduce the use of nitrous oxide and sterile water papule injections but did not affect epidural analgesia use or opioid consumption or pain scores.
    - Transcutaneous electrical nerve stimulations
      - The analgesic effect of tens was found to be weak. A 2009 Cochrane review reported that although women receiving tens to acupuncture points were less likely to report severe pain (RR 0.41, 95% CI 0.32 to 0.55) and most would use it again (RR 1.54, 95% CI 1.31 to 1.80), there was no difference in pain scores, mode of delivery, duration of labour, use of other analgesia, or augmentation of labour.
    - Intradermal injection of sterile water
A 2009 systematic review (n=828) found that women randomized to intradermal sterile water injections had significantly reduced visual analogue pain scores for up to two hours and a reduced caesarean section rate (4.6%) compared to a saline comparison group (9.9%) (RR 0.51, 95% CI 0.30 to 0.87). The meta-analysis included some small and unblinded studies; therefore the authors recommended a large RCT to validate their findings. A 2012 Cochrane review found the intradermal sterile water injection was not effective for low back or other labour pain.

- Aromatherapy
  - Moderate evidence of some pain relief with inhalation of lavender essence; this may be restricted in hospitals with scent restrictions

- Other techniques that enhance descending inhibitory pathways:
  - Attention focusing and distraction
  - Music and audio-analgesia
  - Biofeedback

Pharmacological Methods

Providing effective pain relief in labour is a primary responsibility of the health care team. Having a thorough knowledge of the pharmacology of the drugs used during labour is necessary in order to promote appropriate and satisfying care and to limit side effects. It is important to understand that:

- Sedative and hypnotic drugs do not provide pain relief and may increase respiratory depression when given with opioids.
- No pharmacological method is devoid of maternal or fetal side effects. With almost all pharmaceutical therapies, some amount of the drug gets access to the fetus. Specifically with intravenous opioids, this may be important to the success of breastfeeding.
- Each opioid has unique pharmacodynamic characteristics that may result in different therapeutic effects and side effects.
- Anticholinergic side effects may occur with opioids and care should be directed at recognizing and addressing these discomforts.
- Women can receive opioids just before birth without significant respiratory depression in the newborn.
- Action should be taken to prevent toxicity with all local anesthetics.
- Epidural analgesia provides the most efficacious pharmacological analgesia, with limited side effects to the mother and fetus.

When the health care team understands the indications, limitations, and side effects of these drugs, adverse maternal and fetal outcomes can be minimized. Ideally, the care provider should discuss options regarding medications, possible variations, and potential side effects during the antenatal period. This allows the woman and her family the opportunity to make choices in a less stressful environment. When labour occurs, appropriate and individually desired non-pharmacologic or pharmacologic agents may be used.
Opioids

Opioids are used routinely in many centres. A variety of options are available. A number of studies have reported on the negative effects of opioids with a longer half-life (e.g., meperidine). The negative effects of meperidine are based on an active metabolite half-life in the newborn that may reach 90 hours and may include respiratory depression and newborn behaviour effects including lack of responsiveness and impaired suckling reflex. The use of meperidine is discouraged and opioids with a shorter half-life (e.g., fentanyl) are preferred. Opioids may be given intramuscularly (IM), subcutaneously, or by repetitive intravenous (IV) boluses, either using a patient-controlled pump or administered by staff. The IV route has the advantage of a rapid effect when needed. Opioids may be usefully combined with an antiemetic, as opioids increase the risk of nausea and vomiting compared to epidural analgesia.

Remifentanil patient controlled analgesia (PCA) may offer some benefits over traditional intramuscular (IM) opioids for labour analgesia. Compared with IM meperidine, labouring women using remifentanil were less likely to choose epidural analgesia, and had a reduced instrumental delivery rate. The narrow therapeutic window, as well as the increased risk of respiratory complications requiring constant nursing presence and oxygen saturation monitoring, have limited the wider use of remifentanil for labour analgesia.

When pharmacologic agents are used for pain control in labour, guidelines regarding their safe and effective use should be available for all staff. These guidelines should include the method of action, average and maximum dose, possible maternal and fetal side effects, precautions, and resuscitative measures for each drug. When any opioid is used, opioid antagonists (e.g., naloxone) and resuscitation capabilities should be available. Before a woman is sent home following opioid administration, she needs to be assessed to determine the effects and side effects of the medication.

**Suggested Opioid use in labour:**

<table>
<thead>
<tr>
<th>STAGE OF LABOUR</th>
<th>NULLIPAROUS</th>
<th>PAROUS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Latent Stage:</td>
<td>IM/SC Morphine</td>
<td>IM/SC Morphine</td>
</tr>
<tr>
<td>Early Active Stage:</td>
<td>IM/SC or IV Morphine</td>
<td>IV Morphine or Fentanyl</td>
</tr>
<tr>
<td>Late Active Stage:</td>
<td>IV Morphine or Fentanyl</td>
<td>IV Fentanyl</td>
</tr>
<tr>
<td>Second Stage:</td>
<td>IV Fentanyl</td>
<td>IV Fentanyl</td>
</tr>
</tbody>
</table>

**Nitrous Oxide**

Entonox is a mixture of 50% nitrous oxide, 50% oxygen that provides little analgesia, but high patient satisfaction. It must be self-administered for safety reasons via a demand-valve; and used with scavenging in a well-ventilated room for workplace safety. Deep inhalation should begin as soon as the woman is aware of the onset of a contraction to allow for maximal benefit. It is often useful for the woman who has coped well until transition and then requires some form of analgesia for a short time. Shorter use will decrease environmental exposure, as nitrous oxide is destructive to the ozone layer and has global warming. It may also be used as an adjunct during other procedures such as the placement of a pudendal block or perineal repair. It has been demonstrated that the majority of women who opt to use nitrous oxide...
will convert to neuraxial analgesia, and factors associated with conversion are labour induction (RR 2.0, 95% CI 1.2 to 3.3) and labour augmentation (RR 1.7, 95% CI 1.0 to 2.9).  

### Peripheral Nerve Blocks

A pudendal block can be used for analgesia of the perineum in the second stage of labour. This form of analgesia can be very useful for the sacral nerves and should be considered when other regional analgesia is not available or provides insufficient sacral spread. A diagram depicting pudendal block administration is included as an appendix. Ten (10) mL of 1% plain lidocaine or equivalent is injected at two locations through or just inferior to the sacrospinal ligament, just medial to the ischial spine on each side. The effect is usually felt within three to four minutes.

### Neuraxial Analgesia

The BORN Ontario database indicated that in 2009, 63% of nulliparous women and 39% of multiparous women received neuraxial analgesia. Neuraxial analgesia for labour with a catheter in the epidural space (i.e. labour epidural analgesia (LEA)) can provide effective pain relief throughout all stages of labour and delivery. The hormonal response to pain includes a rise in endogenous catecholamines. The effective relief of pain lowers epinephrine concentrations and may result in improved uterine contractions and possibly improved placental perfusion. A particular benefit of epidural analgesia exists for women with dystocia who require augmentation. Effective pain relief may increase the acceptance of augmentation and the likelihood of subsequent vaginal delivery.

### Onset of Neuraxial Analgesia

Neuraxial techniques have evolved significantly and modern techniques allow labour neuraxial analgesia to be administered effectively and safely for both mother and unborn child. Labour epidural analgesia (LEA) can be initiated with administration of medication (local anesthesia and opioid) into the epidural space or intrathecal space (combined spinal epidural (CSE)). The advantage of CSE is that it provides faster onset of analgesia compared to LEA initiated through the epidural catheter. However, CSE may be associated with increased maternal side effects including hypotension and opioid-induced pruritus. The concern that the epidural catheter placed as part of a CSE technique is “untested” is not evidence based, as CSE is associated with lower failure rates for labour or CS anesthesia compared to standard epidural. A dural-puncture epidural (DPE) is a new technique similar to a CSE, however unlike CSE no intrathecal medications are administered after the dura is punctured. Although the role of DPE has not yet been sufficiently studied, a RCT published in 2017 has demonstrated that DPE provides better sacral spread and a lower incidence of asymmetrical block compared to LEA without dural puncture, and less maternal pruritus and hypotension when compared to CSE.

### Maintenance of Neuraxial Analgesia

Much has changed in the way local anesthetics are administered to give epidural analgesia. Continuous epidural infusion (CEI) with patient controlled analgesia (PCEA) where a patient can give herself a bolus of local anesthetic with
the push of a button is a standard in most large obstetric centres. PCEA has been shown to produce greater maternal satisfaction and excellent analgesia despite using low-dose/very low-dose epidural solutions as compared to traditional CEI. A 2002 meta-analysis of nine studies (n=640) showed lower anesthetic dose and less motor block with PCEA compared to CEI. 35,36

An alternative to CEI is programmed intermittent epidural bolus (PIEB). PIEB allows the pump to give a bolus dose of local anesthetic at specific intervals (e.g. 8 ml every 45 mins rather than 10 ml/hour). This permits greater spread of local anesthetics in the epidural space and better analgesia. PIEB can be used in conjunction with PCEA. A 2013 meta-analysis of RCTs showed PIEB reduces overall local anesthetic dose and improves maternal satisfaction. 38 There is a potential that PIEB may improve instrumental delivery rates and the need for anesthesia interventions. More evidence is required to investigate its effect on obstetric outcomes.

**Obstetric Outcomes**

A 2018 Cochrane review involving 11,000 women showed that compared with opiate or no analgesia, epidural analgesia (low and high doses), provides superior pain relief in labour, higher maternal satisfaction with pain relief, a small decrease in neonatal acidosis, and less need for naloxone. 27 Epidural analgesia was associated with:

- Increased risk of assisted vaginal birth (RR 1.44)
  - However a post hoc subgroup analysis of trials conducted after 2005, which include low dose epidural analgesia, showed that this effect is negated when trials before 2005 are excluded from this analysis (RR 1.19, 95% CI 0.97 to 1.46)
- Maternal hypotension (RR 11.3)
- Motor-blockade (RR 31.7)
- Maternal fever (RR 2.51)
- Urinary retention (RR 14.2)
- Longer second stage of labour (mean difference 15.4 minutes)
- Less nausea and vomiting than opioid analgesia (RR 0.62)

There was no evidence of a significant difference in:

- Overall risk of CS (RR 1.07)
- Risk of CS for abnormal FHR tracing (RR 1.32)
- Dystocia (RR 0.93)
- Long-term backache (RR 1.00)
- Oxytocin administration (RR 1.19, 95% CI 1.00 to 1.26, $I^2 = 80\%$
- Apgar score less than seven at five minutes (RR 0.73) 27

Modern epidural techniques use lower concentrations of local anesthetic than previously. In a meta-analysis (n=2000), low-dose epidurals were as effective as high-dose epidurals at relieving pain, but were less likely to cause motor block, urinary retention, or to interfere with ambulation. 40 A meta-analysis in 2017 evaluated the obstetric outcomes of epidural analgesia with low concentrations of local anesthetics (e.g. less than 0.1% bupivacaine) compared with no epidural analgesia. Low concentrations of local anesthetics for epidural analgesia do not prolong labour, and do not increase
the instrumentation rate\textsuperscript{41}. Early detection and treatment with active management of labour protocols and oxytocin augmentation can reduce the likelihood of CS and operative birth\textsuperscript{42, 43}.

A 2014 meta-analysis of 21 randomized controlled trials (n = 2859 women) compared epidural labour analgesic solutions of local anesthetics, with or without the addition of opioids, on neonatal outcomes\textsuperscript{44}. There was no difference seen in 1 and 5 minute Apgar scores, cord gases or neonatal neurological status at 2 and 24hrs between the two groups. These results demonstrate that the addition of opioids to the epidural local anesthetic solution is safe for the neonate in the first 24hrs of life.

As the role of epidural analgesia and maternal fever has become clearer a separate 2012 meta-analysis of RCTs involving 4667 women showed increased neonatal septic workup (RR 2.58) and antibiotic treatment (RR 2.76) in women receiving epidural analgesia\textsuperscript{45}.

Epidural analgesia can be safely used in women choosing vaginal birth after caesarean (VBAC). A large single-centre retrospective analysis of 7149 women choosing VBAC compared women who received and did not receive epidural analgesia\textsuperscript{47}. It was found that maternal and neonatal morbidity did not differ between the two groups. The presence of epidural analgesia had no impact on the incidence of uterine rupture, PPH, prolonged maternal hospitalization, neonatal Apgar scores or NICU admission.

**Timing of epidural analgesia**

A 2009 randomized trial of 12,793 nulliparous Chinese women reported that epidural initiation early in the first stage of spontaneous labour (cervical dilation of at least 1.0 cm) does not seem to prolong labour or increase CS rate compared to later initiation at a cervical dilation 4.0 cm or greater\textsuperscript{49}. Similarly a RCT of 750 American nulliparous women comparing systemic analgesia to early LEA showed no significant difference in CS rate\textsuperscript{50}. Early LEA compared to systemic analgesia significantly lowered pain scores and the time between intervention till vaginal delivery\textsuperscript{50}. A 2014 Cochrane review of 9 studies (n=15,499 women) comparing early vs late initiation of epidural (< or > 4.5 cm dilatation) showed no difference in AVB, length of second stage, Apgar score less than 7, or umbilical artery pH\textsuperscript{51}.

**Breastfeeding**

Neuraxial analgesia with fentanyl doses as high as 2 mcg/ml do not hinder the onset or success at 6 weeks post-partum of breastfeeding the newborn\textsuperscript{52, 53}.
Care and Management in the Second Stage of Labour

Epidural Analgesia$^{54,55}$

**Continue epidural analgesia.** Discontinuing epidural analgesia in the second stage to allow “effective” pushing, results in the sudden return of pain that may be worse than if there hadn’t been relief provided at all. The woman may become so distracted and distressed by the pain that she cannot push effectively. Maintaining epidural analgesia does not increase the incidence of assisted vaginal birth.$^{56}$ A secondary analysis of early versus late pushing in a RCT revealed an increased incidence of Caesarean birth, mid-pelvic procedures, and third- and fourth-degree tears for women who reported sub-optimal pain control during the second stage of labour.$^{57}$

- Extend the traditional time limits for second stage as long as progress is being made.
- Avoid early intervention with operative delivery if fetal surveillance is normal.
- Use oxytocin, if needed, for lack of progress.
References


## Table of Contents

Chapter 6 Induction of Labour ................................................................. 110

Introduction ......................................................................................... 110

Definitions ......................................................................................... 110

Morbidity and Mortality ..................................................................... 111

Indications .......................................................................................... 114

  High Priority .................................................................................. 114

  Special Considerations .................................................................... 115

  Contraindications to Induction ....................................................... 117

Pre-Induction Assessment .................................................................. 118

  Predictors of successful induction: .................................................. 118

Prevention of Induction/Averting Induction ....................................... 119

  Prevention Strategies to reduce induction for post-term ................. 119

Options for Cervical Ripening: Unfavourable Cervix ................. 121

  Mechanical options ........................................................................ 121

  Pharmacologic Options .................................................................... 122

Options for IOL with a Favourable Cervix ...................................... 128

  1. Castor Oil .................................................................................... 128

  2. Amniotomy .................................................................................. 128

  3. Oxytocin ..................................................................................... 128

  4. Vaginal PGE₂ ............................................................................. 130

Follow-Up ............................................................................................ 131

  Postpartum Considerations ............................................................ 131

Summary .............................................................................................. 132

Post-Term Pregnancy .......................................................................... 143

  Clinical Management Algorithm ................................................. 143
Chapter 6
Induction of Labour

Introduction

Definitions

Induction of Labour (IOL) or induced labour: The initiation of contractions in a pregnant woman who is not in labour to help her achieve a vaginal birth within 24 to 48 hours.

Successful Induction of Labour: A vaginal delivery within 24 to 48 hours of IOL. (See Management of Labour chapter for further information on this topic.)

Elective Induction: The IOL in the absence of acceptable fetal or maternal indications.

Cervical Ripening: The use of pharmacologic or other means to soften, efface, or dilate the cervix prior to IOL to increase the likelihood of a vaginal delivery.

Tachysystole:
- > 5 contractions in 10 minutes, averaged over 30 minutes, and/or
- Inadequate resting tone: uterine resting period between contractions of < 30 seconds OR the uterus does not return to resting tone between contractions, and/or
- Prolonged contraction: lasting > 90 seconds.

NOTE:
- Tachysystole may occur in presence of a normal, atypical or abnormal fetal heart tracing
- The terms "hyperstimulation" and "hypercontractility" should not be used.

Incidence

The frequency of IOL varies by location and institution, but the overall rate in Canada has remained constant since 1995: 20.7% in 1995–1996; 20.25% in 2004–2005; and 21.2% in 2013–2014 (see Figure 1). In British Columbia, the rate of induction has remained fairly constant at approximately 21% between 2000–2001 and 2014–2015. In 2007–2008, the rate of induction in BC was 27.1% for nulliparous women and 16% for parous women (n = 43,499 total women).
Morbidity and Mortality

When undertaken for appropriate reasons and by appropriate methods, induction benefits both mothers and neonates.\textsuperscript{1} If done inappropriately, it may expose both mother and neonate to unnecessary risks.

For nulliparous women, IOL is associated with an increased risk of operative delivery and Caesarean section (CS)\textsuperscript{4,5} (Figures 2 and 3).\textsuperscript{6}

Figure 2. Caesarean Section Rate in Nulliparous Women by Spontaneous Versus Induced Labour, British Columbia, April 1, 2000 to March 31, 2011
Figure 3. Caesarean Section Rate in Parous Women by Spontaneous Versus Induced Labour, British Columbia, April 1, 2000 to March 31, 2011

Source: BC Perinatal Database Registry
Note: Women with late terminations are excluded from the dataset

Figure 4: Induction Rate by LHIN among Low Risk Women

Definition of low risk women: hospital birth, nulliparous, full term (between 37 and 42 weeks of gestational age), singleton, live birth, cephalic presentation, without or minor complications of pregnancy, without or minor pre-existing maternal health conditions, no diabetes in pregnancy and no hypertension disorder in pregnancy and age at 35 years old or under.

Source: BORN Ontario Database
The literature examining mode of delivery for induced versus spontaneous labour is controversial. Observational research suggests a higher risk of operative delivery with IOL. Levine et al. conducted a retrospective cohort study (n = 836) examining term IOL and risk of CS stratified by parity (n = 605); nulliparas undergoing induction had an increased CS rate compared with those who had spontaneous labour (27% vs. 11%, odds ratio [OR] 3.13, 95% confidence interval [CI] 1.76 to 5.57) as did multiparas (13% vs. 3%, OR 4.04, 95% CI 1.36 to 11.94). However, this was a secondary analysis that was not powered to evaluate all the indications for induction. Differences in indication may therefore not have been accounted for. In a 2014 meta-analysis of 31 trials (n = 12,166), Wood et al. showed that in randomly assigned patients, a policy of indicated IOL was associated with a lower risk of CS compared with expectant management (OR 0.83, 95% CI 0.76 to 0.92). However, a 2016 cross-sectional analysis of 42,950 nulliparous women with uncomplicated pregnancies at term found an increased risk of emergency CS with IOL at 37 to 40 weeks (OR 2.54; 95% CI 2.4, 2.7), with no difference in perinatal mortality. A 2016 cohort study of women > 41 weeks had similar findings. A 2017 retrospective cohort study (n=402,960 singletons) in Austria found IOL was associated with an increased risk for CS (OR 1.53; 95% CI 1.45-1.60) and for operative vaginal delivery (OR 1.21; 95% CI 1.15-1.27). In summary, several randomized controlled trials have shown no increase in likelihood of CS with IOL; however, epidemiological and cohort data from many jurisdictions consistently show higher rates of CS in women who undergo IOL, and particularly in nulliparous women.

To prevent the risks of IOL, including a possible increase in CS rate, IOL should be performed only with medical indication, with the exception of logistical considerations.

**Risks associated with IOL include:**

- Uterine tachysystole with or without fetal heart rate changes
- Failure to achieve labour
- Uterine rupture (scarred or unscarred uterus)
- Chorioamnionitis
- Cord prolapse with artificial rupture of membranes (ARM)
- Inadvertent delivery of preterm infant (with inadequate dating)
- Operative vaginal delivery, especially when epidural analgesia used during labour
- Caesarean section (CS)
- Postpartum hemorrhage
- Adverse neonatal outcomes in induction without medical indications at 37 weeks
- Current evidence does not identify a causal relationship between labour induction generally, or oxytocin induction specifically, and autism spectrum disorder.

Every effort should be made to ensure cervical ripening before initiation of induction. If the attempted induction does not achieve labour, then the need, urgency, and method should be re-evaluated in light of the original indication and observed fetal response.
Indications

Induction is indicated when the risk of continuing the pregnancy, for the mother or fetus, exceeds the risk associated with induced labour and delivery. The indication must be convincing, compelling, consented to, and documented. The most common indication is post-dates.\textsuperscript{15,16}

Physician or patient convenience are not acceptable indications. However, in rural communities, the need for a woman to travel to a larger centre for labour and delivery (especially if adverse weather conditions may impede travel) may be an acceptable indication.

The observed increase in stillbirth in women >40 years of age over 40 weeks justifies offering induction of labour at 39\textsuperscript{0}-40\textsuperscript{0} weeks. \textsuperscript{17-19,21-22}

The 2018 ARRIVE trial randomized 3000 low-risk, nulliparous women to induction of labour at 39\textsuperscript{0} to 39\textsuperscript{4} weeks, or expectant management. No difference was found in the primary outcomes of perinatal death or severe neonatal complications. The frequency of Cesarean delivery was lower in the induction group (18.6\% vs. 22.2\%; relative risk, 0.84). Change in practice is not recommended based on this study alone. Research methodology and generalizability of the study, along with patient preference and resources should be considered.\textsuperscript{23}

For all inductions, the patient, her family, and the primary care provider must have a clear understanding of the potential risks and benefits involved. Discussion should include reason for induction, method of induction, and risks of induction, including failure to achieve labour and CS.\textsuperscript{24}

Care providers should determine whether or not labour should be induced according to the urgency of the clinical situation and the availability of resources. The following are examples of situations in which induction may be considered:

High Priority

- Severe preeclampsia or eclampsia at any gestational age (GA), or preeclampsia ≥ 37 weeks
- Significant maternal disease
- Significant but stable antepartum hemorrhage
- Chorioamnionitis
- Term prelabour rupture of membranes with maternal group B streptococcus colonization
- Suspected fetal compromise

Other Indications

- Pregnancies ≥ 41\textsuperscript{0} weeks (see below)
- Uncomplicated twin pregnancy ≥ 38 weeks
- Diabetes mellitus (glucose control may dictate urgency)
- Alloimmune disease at or near term
- Oligohydramnios\textsuperscript{25}
• Gestational hypertension (≥ 38 weeks)
• Intrauterine fetal death
• Premature rupture of membranes at or near term, group B streptococcus negative
• Logistical considerations (rapid labour, distance to hospital)
• Intrauterine death in a prior pregnancy (IOL may be performed to alleviate parental anxiety but there is no known medical or outcome advantage for mother or baby)
• Post-term pregnancy

Special Considerations

Pregnancies ≥ 41\textdegree{} weeks

Prevention of post-term pregnancy is the leading indication for induction and deserves special discussion and consideration. Post-term is defined as a gestation ≥ 42\textdegree{} weeks (294 days from the first day of the last menstrual period) and occurs in approximately 6 \% of births. Marquette et al. calculated cumulative, day-specific probability for onset of spontaneous labour (n = 15,253) among pregnancies between 41\textdegree{} and 42\textdegree{} weeks. The likelihood of beginning spontaneous labour within 24 hours was 14.1 \%, and the likelihood of beginning labour within 7 days was 67.6 \%.\textsuperscript{26} Post-term pregnancies have been shown to have an associated increase in perinatal mortality, morbidity, and operative delivery.\textsuperscript{27-29} The most frequent cause of an apparently prolonged gestation is an error in determining accurate dating. Accurate dating based on ultrasonography performed in early pregnancy can reduce the incidence of pregnancies diagnosed as post-term (relative risk [RR] 0.59; 95\% CI 0.42 to 0.83) and subsequently minimize unnecessary intervention such as IOL.\textsuperscript{30}

Several trials have examined the policy of inducing labour at ≥ 41 weeks’ gestation in an attempt to avert adverse outcomes associated with post-term pregnancy. The following meta-analysis\textsuperscript{31} of 19 trials (7,984 women) concluded that a “policy of labour induction at 41 completed weeks or beyond was associated with fewer (all-cause) perinatal deaths.” (2006)

- The relative risk for perinatal death in the trials where IOL was initiated after 41 weeks was 0.25 with 95\% CI of 0.05 to 1.18 (10 trials, 0/2,835 vs. 6/2,808). For trials where inductions were initiated after 42 weeks, the RR for perinatal death was 0.41 with CI of 0.06 to 2.73 (2 trials, 1/151 vs. 3/145). When the 41-week and 42-week trials were analyzed together, the RR reached significance at 0.30; CI 0.09 to 0.99.
- In trials where IOL occurred after 41 weeks, there was a reduced risk of meconium aspiration syndrome (RR 0.29; 95\% CI 0.12 to 0.68, 4 trials, 1,325 women).
- There was no difference in the risk of Caesarean section (10 trials at 41 weeks, n = 5,755, RR 0.92; 95\% CI 0.76 to 1.12; and 5 trials at 42 weeks, n = 810, RR 0.97; 95\% CI 0.72 to 1.31).
- There was no difference in the risk of Apgar scores of < 7 at 5 minutes.

A 2018 Cochrane review of 30 trials reporting on 12,479 women found that compared to a policy of expectant management, a policy of elective IOL for pregnancies at or beyond 41 weeks is associated with:

- fewer (all cause) perinatal deaths (RR 0.33; 95\% CI 0.14 to 0.78)
- fewer stillbirths (RR 0.33; 95\% CI 0.11 to 0.96)
- fewer CS (RR 0.92; 95\% CI 0.85 to 0.99)
• slightly higher operative vaginal births (RR 1.07; 95% CI 0.99 to 1.16)
• lower rates of NICU admission (RR 0.88; 95% CI 0.77 to 1.01)
• fewer infants with Apgar scores < 7 at 5 minutes (RR 0.70; 95% CI 0.50 to 0.98)

There was no difference between the groups for:
• perineal trauma (RR 1.09; 95% CI 0.65 to 1.83)
• postpartum hemorrhage (RR 1.09; 95% CI 0.92 to 1.30)
• neonatal trauma (RR 1.18; 95% CI 0.68 to 2.05)

Therefore, a policy of induction at ≥ 41 weeks’ gestation is recommended to avert the risks associated with post-term pregnancy (see Appendix). If, following discussion with the patient, induction is not chosen, then twice-weekly fetal surveillance is strongly recommended. At a minimum, serial surveillance should consist of daily fetal movement counts and, for the 41- to 42-week pregnancy, at least twice-weekly non-stress testing and ultrasound assessment of amniotic fluid volume (AFV).

**Term Prelabour Rupture of Membranes**

Intravenous oxytocin is the preferred agent for term prelabour rupture of membranes. However, oral misoprostol is a promising agent because it has both cervical ripening and uterotonic effect and does not require vaginal examination with the attendant risk of infection. (See Prelabour Rupture of Membranes Chapter.)

**Group B Streptococcus + and Rupture of Membranes**

Induction should be started as early as possible after rupture of membranes to establish labour within 24 hours. (See Prevention of Early-Onset Neonatal Group B Streptococcus Disease Chapter.)

**Trial of Labour after Caesarean (TOLAC)**

IOL remains an option for women choosing trial of labour after Caesarean (TOLAC). However, the potential increased risk of uterine rupture associated with any induction, and the potential decreased likelihood of achieving VBAC (especially in absence of a previous vaginal delivery) should be discussed.

IOL that requires cervical ripening is associated with a lower rate of success and an increased risk of uterine rupture, mainly in women who have not experienced a previous vaginal delivery.

IOL in presence of a scarred uterus requires caution:
• Mechanical cervical ripening with a Foley catheter can be safely used
• Prostaglandins have been associated with increased risk of uterine rupture and should not be used.
• Oxytocin using a Low-dose protocol can be used with careful maternal-fetal monitoring and consideration of the maximum dose to be administered. The timely availability of human and physical resources to respond to an emergency is required
Suspected Fetal Macrosomia

A 2016 Cochrane review of 4 trials (n = 1190) compared expectant management with IOL for the indication of suspected fetal macrosomia (birth weight > 4000 g). It found:

- Mean birthweight was lower in the induction group (mean difference −178.03 g, 95% CI −315.26 to −40.81)
- No differences in the risk of CS or instrumental delivery
- Increased third and fourth degree tears in the induction group
- Fewer fractures (of any kind) and fewer cases/instances of shoulder dystocia in the induction group
- No differences in risk of brachial plexus injury, measures of neonatal asphyxia, 5-minute Apgar score < 7, or low arterial cord blood pH
- Increased use of neonatal phototherapy.

The authors conclude that to prevent 1 fracture, it would be necessary to induce labour in 60 women.

The American College of Obstetricians and Gynecologists 2016 Bulletin on fetal macrosomia states induction is NOT supported because of the inaccuracy of ultrasound estimation of fetal weights and no improvement in maternal or fetal outcomes. Ultrasound estimation of fetal weight can be used to eliminate macrosomia to reduce intervention. Expert opinion indicated consideration of CS in the case of estimated fetal weight > 4500 g in women with diabetes mellitus and > 5000 g in women who did not have diabetes mellitus. Women with suspected fetal macrosomia should be informed about the risks and benefits of vaginal delivery and Caesarean section depending on the severity of macrosomia. A meta-analysis of RCTs for induction of labour for suspected macrosomia at term in non-diabetic women vs. expectant management (4 studies, n=1190) showed no difference in outcomes for those induced vs. those managed expectantly (CS, operative vaginal delivery, shoulder dystocia, intracranial hemorrhage, brachial plexus palsy, Apgar <7 at 5 min, cord blood pH<7, and mean birth weight). The induction group had significantly lower time to delivery, lower rate of birth weight >4000 and >4500 g, and lower incidence of fetal fractures. They conclude that induction of labour > 38 weeks for suspected fetal macrosomia in non-diabetic women can be considered a reasonable option.

Contraindications to Induction

IOL should not be undertaken if there are any contraindications to labour or vaginal delivery, including:

- Placenta or vasa previa or cord presentation
- Abnormal fetal lie or presentation (e.g., transverse lie or footling breech)
- Prior classical or inverted T uterine incision
- Significant prior uterine surgery (e.g., full thickness myomectomy); the operative report information should be obtained
- Active genital herpes
- Pelvic structural deformities
- Invasive cervical carcinoma
- Previous uterine rupture
Pre-Induction Assessment

Before induction, there are several clinical elements that need to be considered to estimate success and minimize the risk of CS.

Predictors of successful induction\(^{44, 45}\)

- Bishop score > 6
- Prior vaginal delivery

The less compelling the indication for induction, the more attention should be paid to adequate cervical ripening.

The condition of the cervix at the start of induction is the most important predictor of success. The Bishop score was developed in 1964 as a predictor of success for an elective induction. The most important element of the Bishop score is dilatation followed by effacement, station, and position, with the least useful element being cervical consistency.\(^{46, 47}\) Xenakis et al. clearly demonstrated that women who had a Bishop score at entry of ≤ 3 had significantly higher rates of failed induction (9.4% vs. 0.7%, \(P < 0.01\)) and of Caesarean section (29% vs. 15.4%, \(P < 0.01\)) than those with a Bishop score > 3.\(^{48}\) Nova Scotia’s Atlee database also shows that the risk of CS in low risk, nulliparous women is highest in those undergoing labour induction when compared with those entering spontaneous labour.\(^{49}\)

Figure 5. Likelihood of successful vaginal delivery

Reproduced with permission from Salus.
The cervix is considered unfavourable if the Bishop score is $\leq 6$ and favourable if it has a Bishop score of $> 6$. Many studies have used other Bishop score values to separate the data.

### Table 1. Modified Bishop Scoring System

<table>
<thead>
<tr>
<th>Factor</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dialation</td>
<td>0</td>
</tr>
<tr>
<td>Effacement (%)</td>
<td>0–30</td>
</tr>
<tr>
<td>Length (cm)</td>
<td>$&gt; 3$</td>
</tr>
<tr>
<td>Consistency</td>
<td>Firm</td>
</tr>
<tr>
<td>Position</td>
<td>Posterior</td>
</tr>
<tr>
<td>Station</td>
<td>Sp –3 or above</td>
</tr>
</tbody>
</table>

### Prevention of Induction/Averting Induction

Quality improvement programs have been shown to reduce the number of elective inductions and unplanned CS. Several studies have shown a significant reduction in the number of elective inductions after the implementation of an induction committee. The role of the committee was to review each request and enforce the use of proper indications for induction.

Institutional factors may play a role in the CS rate of induced labours. Brennan et al. compared CS rates in 10 different groups defined by the Robson criteria. In the group of low-risk women induced at term, the low induction centres had a lower overall CS rate than the higher induction centres (17.7% vs. 27.8%, $P < 0.008$).

### Prevention Strategies to reduce induction for post-term

#### Ultrasound

Every effort should be used to determine dates accurately. Early (8-12 weeks) ultrasound (US) examination provides the most accurate estimated date of delivery and has been demonstrated to decrease the diagnosis of post-term pregnancy.
The 2014 SOGC clinical practice guideline on determination of gestational age by ultrasound states:

“When performed with quality and precision, ultrasound alone is more accurate than a “certain” menstrual date for determining gestational age in the first and second trimesters (≤ 23 weeks) in spontaneous conceptions, and it is the best method for estimating the delivery date.”

If there is more than 1 first-trimester scan with a mean sac diameter or crown-rump length measurement, the earliest ultrasound with a crown-rump length equivalent to at least 7 weeks (or 10 mm) should be used to determine the gestational age.

A 2010 Cochrane review by Whitworth et al. reported reduced rates of IOL for post-term pregnancy (RR 0.59; 95% CI 0.42 to 0.83) with routine early pregnancy ultrasound imaging.

**Figure 6. Routine/Revealed vs Selective/Concealed Ultrasound in Early Pregnancy. Outcome-Induction for “post-term” Pregnancy**

### Sweeping (Stripping) of Membranes at Term

There is evidence that sweeping of membranes (3 circumferential passes within the cervix or cervical massage for 15 to 30 seconds when unable to pass the external cervical os) promotes the onset of labour by increasing local production of prostaglandins. Meta-analyses have found that when sweeping is performed in term women at 38 weeks and beyond, it is associated with a reduced duration of pregnancy and reduced rate of pregnancy continuing beyond 41 weeks. A 2014 RCT (n = 134) found pregnancy duration decreased by 3 days ($P = 0.001$). Sweeping of membranes
must be performed in 8 women to prevent 1 formal IOL. Discomfort during vaginal examination and other adverse effects (e.g., bleeding, irregular contractions) were more frequent in women who had the procedure performed. The reviewers concluded that when membrane sweeping is used, the subsequent reduction in the need for more formal induction methods needs to be balanced against women’s discomfort and other adverse effects.

Options for Cervical Ripening: Unfavourable Cervix

It is important that cervical ripening be considered before IOL in women with an unfavourable cervix. Many methods for cervical ripening are effective. Amniotomy and oxytocin are not effective cervical ripening methods.

Mechanical options

The use of balloon catheters and laminaria or synthetic laminaria tents may affect cervical softening, effacement, and dilatation with fewer or no systemic effects. The probable mechanism of action is local prostaglandin (PG) production. Balloon methods have the advantage of cost effectiveness and a lower risk of uterine tachysystole.

A 2011 systematic review by Fox et al. comparing Foley catheter versus intravaginal misoprostol (9 studies, n = 1603) found no difference in time to delivery, rate of Caesarean section, or rate of chorioamnionitis. However, the vaginal misoprostol group had a higher rate of tachysystole.

Mechanical methods are not likely to be effective in inducing labour on their own. In a 2011 World Health Organization evidence summary of the comparison of balloon catheter plus oxytocin versus misoprostol alone, the combination approach was associated with more vaginal deliveries achieved within 24 hours (1 trial, 158 participants).

However, a 2008 systematic review found there may be an increased risk of both maternal and newborn infection associated with specific mechanical methods of cervical ripening. This review found that for women who underwent cervical ripening with a Foley catheter alone versus a pharmacological agent, there was an increase in maternal infection (defined as pyrexia of 38°C, chorioamnionitis, peripartum infection, or chorioamnionitis and/or endomyometritis) (OR 1.5; 95% CI 1.07 to 2.09) and chorioamnionitis (OR 2.05; 95% CI 1.22 to 3.44). However, with the use of a Foley catheter, there was no increase in neonatal infection (OR 1.2; 95% CI 0.48 to 2.97). Double balloon catheters have not been shown to be more beneficial.

Evidence suggests that for women > 34 weeks with rupture of membranes, use of transcervical Foley catheter in addition to oxytocin does not shorten the time to delivery compared to oxytocin alone, and may increase the incidence of intraamniotic infection.

More recent RCTs and meta-analyses have demonstrated similar safety and effectiveness for Foley catheter as for vaginal/oral misoprostol and vaginal prostaglandin E2 inserts. Using a Foley catheter is the better option when there are concerns about fetal well-being or placental function, because there is less risk of tachysystole. Use of a Foley catheter is also cheaper than use of prostaglandin E2 and when used in an outpatient setting, cheaper than ORAL PGE.

Reviews of Foley balloon catheter for cervical ripening concluded that its use is relatively safe, even as an option for outpatient cervical ripening.
Balloon devices: Foley catheter (for protocol details see the SOGC guideline\textsuperscript{24} at technique for Foley):

- no. 14 to 18 Foley with a 30 mL balloon
- pre-test Foley balloon before insertion
- sterile technique, insert past internal os
- inflate with 30 to 60 mL water\textsuperscript{86, 87}
- Tension is not necessary\textsuperscript{88} Catheter should be removed within 24 hours if it has not been spontaneously expelled
- Insertion may be facilitated by a stylet
- contraindications: low-lying placenta
- relative contraindications: rupture of membranes\textsuperscript{89}, genital tract infection

Pharmacologic Options

Prostaglandins

Agents include:

- vaginal PGE\(_2\) gel (Prostin)
- posterior fornix slow release PGE\(_2\) (Cervidil)
- intra-cervical PGE\(_2\) gel (Prepidil)
- oral PGE\(_1\) (Oral misoprostol)

Considerations for Prostaglandin Use

When considering prostaglandin use, patient acceptance and preference of the various options are important. Cost considerations also may play a role in the choice of prostaglandin. Proprietary intravaginal PGE\(_2\) formulations cost between $40 and $80 per dose. Misoprostol is produced generically and costs approximately $0.05 per tablet. In women with term or late-preterm ruptured membranes, oral misoprostol provides cervical ripening without the need for vaginal examinations or laminaria (cf. Cervidil). Prostaglandin gels are uterine stimulants whose effects may not easily be reversed.

Certain precautions must be taken with prostaglandins:

- Electronic fetal surveillance for a minimum of 30 minutes before PGE\(_2\) application and for 1 hour post application. If tracing atypical or abnormal, do not administer prostaglandin.
- Vaginal gel PGE\(_2\) (Prostin) should NOT be placed in the cervical canal.
- Prostaglandins should NOT be used as augmentation agents.
- Uterine activity should be carefully assessed before repeating the dose
- Prostaglandins should NOT be used in patients with previous CS because of the increased risk of uterine rupture.\textsuperscript{90, 91}
- Oral PGE\(_1\) (oral misoprostol)\textsuperscript{38} or vaginal PGE\(_2\) (Prostin)\textsuperscript{1} may be considered with ruptured membranes at term.
Further research is needed on the use of slow-release PGE$_2$ (Cervidil) in women with ruptured membranes. The manufacturer suggests that it should be used with caution in these women and that uterine activity and fetal status should be carefully monitored.\(^2\)

**Adverse reactions may occur:**
- Tachysystole with or without fetal heart rate changes (incidence is similar to use of oxytocin)
- Gastrointestinal side effects
- Vaginal irritation

**Prostaglandin E$_2$**

Also known as dinoprostone, PGE$_2$ is available as an intravaginal or an intracervical gel. Intravaginal gels or preparations are easier to employ, cause less patient discomfort, and are preferred because they result in more timely vaginal delivery than mechanical methods.\(^3\)

---

**CAUTION**

*It is important to ensure that vaginal agents (Prostin, Cervidil) are not inserted into the cervical canal because they have a much higher dosage than intracervical preparations (Prepidil).*

---

PGE$_2$ is a bronchodilator and is not contraindicated in women who have asthma.\(^4\) Adverse cardiovascular events are rare, idiopathic, and usually occur almost immediately after the gel or preparation has been inserted.

**Route and Dose**

**Vaginal**
- PGE$_2$ (Prostin) 1 to 2 mg into posterior fornix
- PGE$_2$ (Cervidil) 10 mg into posterior fornix. Cervidil released at 0.3 mg/hr. Should be removed when the woman is in active labour or at 12-24 hours post insertion. If a favourable cervix is not achieved a second dose may be used if there are no contraindications.

**Intracervical**
- PGE$_2$ (Prepidil) 0.5 mg intracervical
- Intracervical preparations should not be used in women with PROM.
- Any formulation may be used for cervical ripening
- Initial application may be followed by repeat PGE$_2$ or oxytocin, as per the manufacturer’s recommendation

Protocols have been developed to increase the chance of a successful outcome and enhance safety:
- Patients should be seen by experienced staff in a controlled setting where resuscitation and delivery can be performed
- PGE$_2$ applied by a knowledgeable caregiver
Prostaglandin Advantages
- Patient acceptance
- Lower operative delivery rate than oxytocin
- Less need for oxytocin induction

Prostaglandin Disadvantages
- Increased risk of uterine rupture with previous CS
- Side effects may include: nausea, vomiting and/or diarrhea
- Gel preparations are difficult to remove in instances of tachysystole

Outpatient Management

Outpatient management may be considered with use of vaginal PGE$_2$ gel (Prostin), slow release PGE$_2$ (Cervidil) or intra-cervical PGE$_2$ gel (Prepidil) when IOL is done for maternal indication, and after post-administration electronic fetal assessment is complete. Outpatient management should not be considered when oral PGE$_1$ (oral misoprostol) is used.

Although some centres use prostaglandins on an outpatient basis, there is limited evidence demonstrating safety, so fetal well-being and the absence of significant uterine activity must be assured before discharge. Women should be carefully instructed to return promptly for assessment if uterine activity increases. Outpatient management is NOT appropriate when IOL is undertaken because of suspected fetal compromise (e.g., intrauterine growth restriction, poor biophysical profile).

Prostaglandin E$_1$

Misoprostol is a synthetic prostaglandin E$_1$ analogue manufactured for the prevention and treatment of gastric ulcers associated with the use of non-steroidal anti-inflammatory drugs.

It is not licensed for use in obstetrics and gynaecology, although it is used off-label worldwide for three main applications: postpartum hemorrhage (PPH), incomplete miscarriage, and IOL. The American College of Obstetricians and Gynecologists and FDA have endorsed its off-label use for IOL.

Like other prostaglandins, misoprostol causes both cervical ripening and uterine contractions in a dose-dependent fashion. It is available only in oral form (100 mcg and 200 mcg tablets), but is readily absorbed trans-mucosally (sublingually, bucally, vaginally, or rectally). Following oral or sublingual administration, levels peak at 30 minutes; however, because of first-pass liver metabolism, the peak maternal serum concentration and the duration and area under the curve are twice as great for sublingual as for oral dosing. Fifty micrograms orally is approximately equivalent to 25 mcg sublingual. When it is given vaginally, peak levels are delayed and the area under the curve is lower than with sublingual administration.

Route and Dose

Over 200 randomized trials have been published comparing various doses and routes of misoprostol with oxytocin and other vaginal prostaglandins. The higher the dose, the greater the success with IOL and the greater the risk of uterine...
tachysystole. The safety and effectiveness of low-dose misoprostol for IOL is well established. Two Cochrane reviews, published in 2010 and 2014 and including more than 100 trials and 17,000 women, compared both vaginal and oral misoprostol with PGF₂α, oxytocin, and each other. They concluded:

- "Oral misoprostol as an induction agent is effective at achieving vaginal birth. It is more effective than placebo, as effective as vaginal misoprostol and results in fewer caesarean sections than vaginal dinoprostone or oxytocin."\(^{103}\)
- "The vaginal route should not be researched further as another Cochrane review has shown that the oral route of administration is preferable to the vaginal route."\(^{104}\)

The accepted, effective, safe oral dose is 50 mcg every 4 hours as needed. There is not evidence for a safe maximum number of doses; however, if there is no effect after 4 doses, reassessment and consideration of an alternate method of cervical ripening are recommended.

The corresponding sublingual dose is 25 mcg; however, this dose is difficult to prepare accurately from commercially available tablets and may be associated with greater risk of uterine tachysystole with FHR changes than the 50 mcg oral dose.

Protocols using more frequent, smaller doses of a misoprostol solution prepared from dissolving a 200 mcg tablet in water have also been studied. Outcomes were similar to those achieved by administration of 50 mcg every 4 hours.\(^{103}\) Preparation and administration of the oral solution are more time consuming, however, and there are concerns regarding poor tablet solubility.

**CAUTION**

When inducing labour at term, institutions must take precautions to avoid accidental use of the 200 mcg tablets, commonly used for PPH and for treatment of first and second trimester miscarriage.

**Dose Preparation**

The risk of accidental overdose can be minimized by having a hospital pharmacy pre-cut 100 mcg tablets into 50 mcg doses which can be individually foil-packaged. The 50 mcg doses are then kept in a separate location from the larger 200 mcg tablets commonly kept in labour rooms for the treatment of postpartum hemorrhage and miscarriage management.

When misoprostol is given orally, it is important that the tablet or solution be swallowed and not held in the mouth sublingually or buccally, which would cause higher blood levels and greater risk of uterine tachysystole. Repeat doses may be given every 4 hours until regular or painful uterine contractions.
Side Effects of Prostaglandins

Potential maternal side effects include nausea, vomiting, diarrhea, abdominal pain, shivering, and chills and fever; however, these side effects are dose-dependent and are rare with a dose of 50 mcg. Misoprostol does not cause bronchospasm and is safe for use by patients who have asthma. The most important significant side effect of any prostaglandin given for induction is uterine tachysystole with or without fetal heart rate changes.

The risk of uterine “hyperstimulation” with fetal heart rate changes with a 50 mcg oral dose of misoprostol is the same as with vaginal PGE2, at approximately 1% to 2%.[105] Prostaglandin-related prolonged contractions can rarely cause placental abruption or uterine rupture. Prostaglandin use is therefore not generally recommended in women with a history of prior Caesarean section or significant uterine scar; and prostaglandins should be used carefully in grandmultiparous women. Oxytocin should not be used within 4 hours of the last oral misoprostol dose.[2]

Eligibility

- Clear indication for IOL
- Greater than 35 weeks’ gestation

Use with Caution

- Grand multipara (>6 prior vaginal deliveries)
- Fetal growth restriction or oligohydramnios

Exclusion criteria

- < 35 weeks’ gestation
- Previous Caesarean section or other significant uterine surgery
- Abnormal fetal heart tracing
- Regular or painful uterine contractions

Initial Patient Evaluation

- Routine initial assessment including vital signs
- History, physical, and admission by physician
- A normal NST should be documented

Monitoring

Continuous electronic fetal monitoring is recommended for 30 minutes after misoprostol administration. It is recommended for 1 hour whenever there is increased uterine activity within 4 hours of a misoprostol dose. Patients should remain in hospital but may ambulate after normal fetal assessment with stable uterine activity. If the woman is not in active labour and contractions are mild 4 hours after the last misoprostol dose, she may return home for sleep as needed, and return in the morning to continue induction.
Vigilance in identifying uterine tachysystole is critical for the safe use of PGE₂ and PGE₁.

**Management of tachysystole depends on whether FHR changes are present. A treatment protocol for excess uterine activity is recommended for every labour unit along with a Tachysystole tray.**

**Tachysystole Tray suggested contents:**

- 1 normal saline 1000 ml
- 1 small sterile bowl
- 8 sterile 4 x 4
- 1 nitroglycerine sublingual spray
- 2 sponge forceps
- 2 60 cc syringe Toomey cath tip
- 2 normal saline 100 ml
- # 18 Foley catheter
- Gloves size 6-8, one of each
- Water based lubricant
- IV Nitroglycerine Kit (Nitroglycerine 50 mg/ 10 ml; syringes, needles, alcohol swabs, medication labels)

If there is tachysystole with normal fetal heart tracing:

- Remove prostaglandin if possible (Cervidil or recently applied vaginal gel)
- Decrease the rate of oxytocin infusion rate (if applicable)
- Maintain close continuous EFM

If there is tachysystole with atypical or abnormal fetal heart tracing:

- Undertake immediate assessment (attending physician or midwife)
- Stop oxytocin, if running
- Remove prostaglandin if possible (Cervidil or recently applied vaginal gel)
- Conduct pelvic examination to assess cervical dilatation and rule out prolapsed cord
- Initiate intraterine resuscitation
- Apply scalp electrode if any question about external FHR pick-up or interpretable tracing
- Consider acute tocolysis with nitroglycerin (50 mcg IV doses every 90 seconds to 3 minutes, to maximum of 200 mcg over 15 minutes). To date, the evidence for safety and efficacy remains inconclusive. Necessary monitoring includes maternal BP, maternal SaO₂ and continuous EFM. Sublingual nitro does not work and will give the woman a headache.
- Make immediate preparation for delivery if these measures do not rapidly lead to resolution of the fetal heart rate abnormality

If tachysystole resolves and the cervix remains unfavourable, indications and fetal well-being should be re-evaluated before further intervention. Foley catheter cervical ripening may be preferred as it does not carry a risk of tachysystole.
Options for IOL with a Favourable Cervix

1. Castor Oil

Several studies have reported effectiveness of oral ingestion of castor oil for inducing labour within 24 hours of ingestion, with no maternal or fetal complications, in multiparous women, and reduces need for other medications.\textsuperscript{109,111} It may be tried safely post-dates on an outpatient basis.

2. Amniotomy

Amniotomy creates commitment to delivery, and is effective with a favourable cervix. Trials indicate it should be used in conjunction with oxytocin in most instances. A 2009 RCT demonstrated that women who receive oxytocin immediately following amniotomy (compared with those who receive it 4 hours later) are more likely to be in labour within 4 hours, to have a shorter amniotomy to delivery time and achieve delivery within 12 hours, and to experience greater maternal satisfaction.\textsuperscript{112} A 2011 systematic review comparing amniotomy and IV oxytocin to vaginal prostaglandins found that with IV oxytocin and amniotomy, there were higher rates of PPH and maternal dissatisfaction.\textsuperscript{113}

Care must be taken in cases of high presenting part due to increased risk of cord prolapse. After amniotomy, note the amount, colour, and consistency of the fluid and assess fetal well-being.

3. Oxytocin

First synthesized in 1955, oxytocin is a hormone produced in the hypothalamus, stored in the posterior pituitary, and secreted in a pulsatile manner. IV infusion of oxytocin has been the most common method of induction and remains valuable for properly selected women.

Induction of labour with oxytocin is considered a high-risk area of practice by both the Canadian Medical Protective Association (CMPA) and the Healthcare Insurance Reciprocal of Canada (HIROC). The two most common issues in obstetrical incidents involving oxytocin induction (or augmentation) (2004-2013) included failure to reduce or discontinue oxytocin infusion and delay in notifying or consulting with the physician for unresolved uterine tachysystole, signs of uterine rupture, or abnormal fetal heart rate patterns.\textsuperscript{114}

All units offering oxytocin induction of labour must have oxytocin induction policies, protocols and safe staffing levels available when induction of labour with oxytocin is undertaken.

Oxytocin can be used:

- 30 minutes after dinoprostone insert (Cervidil) removal
- 4 hours after oral misoprostol administration
- 6 hours after vaginal prostaglandin E\textsubscript{2} gel administration
- Early after amniotomy\textsuperscript{24}
- There is no contraindication to giving oxytocin over several days in the event onset of labour is not achieved and membranes intact.
Physiology

Oxytocin receptors are found in the myoepithelial cells of the breast, the myometrium, and the deciduas.

- Myometrial smooth muscle
  - rhythmic contraction at low dose
  - increased sensitivity as term approaches (insensitive at < 20 weeks)
  - infusions of 6 milliunits per minute (mU/min) give the same oxytocin levels that are found in spontaneous labour and most women will have a clinical response at 8 to 10 mU/min at term

- Cervix
  - no direct effect

- Vasoactive
  - very minimal vasopressor response
  - hypotension possible with bolus IV administration

- Antidiuretic activity - water intoxication possible with high-dose oxytocin (> 40 mU/min)

Protocol

- Cervix should ideally be favourable (Bishop Score > 6)
- Experienced care providers and adequate resources should be available to manage dystocia and other emergencies
- When uterine activity cannot be adequately evaluated by external monitoring and palpation, consider use of IUPC to monitor uterine activity
- Administration:
  - Given by infusion pump into a mainline IV and titrate to uterine response
  - Describe dosage as milliunits per minute (mU/min)
  - Concentrations vary but avoid large free water load (dextrose 5% in water [D5W] should not be given)
  - Institutional protocols should be used

The goal of oxytocin is to achieve effective uterine contractions and cervical changes with the minimally effective dose. Once this state is achieved, the oxytocin dose can be maintained.

Oxytocin may be administered using either low- or high-dose protocols. See MOL chapter.

There are no randomized clinical trials comparing different timing of the use of oxytocin after prostaglandin gel. Many studies have used a 6-hour interval.
**Tachysystole with Oxytocin Administration**

Discontinue oxytocin and institute tachysystole protocol.

Restarting oxytocin:

- Remember that the half-life of oxytocin is approximately 6 to 8 minutes. If oxytocin is restarted, consider beginning at a lower dose.

Discontinuation of oxytocin when active phase of induced labour is established

A 2018 Cochrane Review (10 trials, 1,888 women) compared oxytocin discontinuation after the active phase of induced labour was established vs. oxytocin continuation.\(^{115}\) The analysis by 'intention-to-treat' found that, compared with oxytocin continuation, discontinuation of oxytocin in the active phase of labour:

- reduced caesarean delivery rate (RR 0.69; 95% CI 0.56 to 0.86) (9 trials, 1784 women, low level certainty*)
- reduced tachysystole combined with abnormal fetal heart rate (RR 0.15; 95% CI 0.05 to 0.46)
- reduced suspicious/pathological fetal heart findings (RR 0.65; 95% CI 0.51 to 0.83).

The authors conclude that discontinuation of oxytocin in active labour had little or no impact on use of epidural analgesia, Apgar < 7 at 5 minutes and umbical artery pH <7.10.

*Most trials included in the review had "risk of bias" concerns so results should be interpreted with caution.

NOTE: Oxytocin appropriately titrated to the maternal uterine and fetal heart rate response is considered safest clinical practice.

**4. Vaginal PGE\(_2\)**

**Comparison of Pharmacologic Methods**

A 2009 Cochrane review compared efficacy of oxytocin with vaginal PGE\(_2\) and intracervical PGE\(_2\) for third trimester cervical ripening and IOL.\(^{116}\) The odds ratios for a persistently unfavourable cervix after 12 to 24 hours and failure of vaginal delivery in 24 hours favour the use of vaginal prostaglandins.
### Follow-Up

#### Postpartum Considerations

If oxytocin has been used during the labour, anticipate postpartum hemorrhage and take appropriate preventive action. (For third stage of labour management and prevention of PPH, see the PPH chapter.)

---

**Figure 7. Oxytocin Alone vs Vaginal PGE$_2$**

<table>
<thead>
<tr>
<th>OUTCOME</th>
<th>OR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cx unfavourable</td>
<td>2.42 (1.43, 4.09)</td>
</tr>
<tr>
<td>No vaginal delivery 24 hrs</td>
<td>1.77 (1.31, 2.38)</td>
</tr>
<tr>
<td>Hyperstimulation (NR FHR)</td>
<td>0.35 (0.04, 3.28)</td>
</tr>
<tr>
<td>Chorioamnionitis</td>
<td>0.66 (0.47, 0.92)</td>
</tr>
<tr>
<td>Caesarean section</td>
<td>1.11 (0.94, 1.30)</td>
</tr>
<tr>
<td>5 min. Apgar &lt; 7</td>
<td>0.62 (0.36, 1.05)</td>
</tr>
<tr>
<td>Neonatal infection</td>
<td>0.68 (0.42, 1.09)</td>
</tr>
<tr>
<td>Serious neonatal morbidity</td>
<td>3.00 (0.31, 28.82)</td>
</tr>
</tbody>
</table>

Odds Ratio

(95% confidence interval)
Summary

- The reasons for induction must be compelling, convincing, consented, and documented
- The method should match the situation: the degree of urgency of the indication and the status of the cervix should be considered.
- Ideally, the cervix should be favourable (Bishop score > 6) before amniotomy and before initiation of oxytocin.
- Oxytocin should always be used with caution and only where policies, protocols and safe staffing levels are in place at the time of the induction. Oxytocin must be titrated to maternal uterine and fetal heart rate response.
- Patient preference must be considered.
- Comparison summary**113, 117**
  - membrane sweeping reduces post-term gestations.
  - mechanical methods (e.g., intracervical balloon) are more likely than PGE$_2$ or misoprostol to reduce the occurrence of tachysystole.
  - PGE$_2$ and vaginal misoprostol are more effective than oxytocin in bringing about vaginal delivery within 24 hours but are associated with more tachysystole.**118**
  - oxytocin plus amniotomy is more effective than amniotomy alone in achieving vaginal delivery within 24 hours.**119**
  - vaginal misoprostol PGE$_1$ is more likely than PGE$_2$ or oxytocin to result in vaginal delivery within 24 hours but is associated with increased tachysystole.
  - vaginal misoprostol may reduce the likelihood of CS compared with intravenous oxytocin in the case of an unfavourable cervix.
  - oral misoprostol is associated with a greater reduction in the rate/incidence of CS than vaginal PGE$_2$ or placebo.
  - oral misoprostol is associated with a lower rate of tachysystole but there is more need for oxytocin augmentation than with vaginal misoprostol.
  - sample sizes in RCTs of induction are too small to exclude differences in rare adverse outcomes such as uterine rupture, amniotic fluid embolism, or perinatal asphyxia.
References


Appendix

Post-Term Pregnancy

Clinical Management Algorithm

(Adapted from BCPHP Guideline: Post-term Pregnancy and SOGC: Guidelines for the Management of Pregnancy at 41\textsuperscript{0} to 42\textsuperscript{0} Weeks)

GESTATIONAL AGE DETERMINATION

- Use biometry from US done at 7 to 16 weeks, if available
- Use sure LMP if no US < 16 wks and 16- to 23-week US not differ by ≥ 10 days
- Use 16- to 23-week US biometry if differs from sure LMP by ≥ 10d
- If unsure LPM or irreg. cycles, use earliest 1st or 2nd TM U/S

Healthy Pregnancy

- Offer membrane sweeping between 38 and 41 weeks
- Elective induction, NST and/or AFV assessment are NOT recommended

Maternal risk factors or evidence of fetal compromise

- Inform woman of risks and benefits of elective induction vs. expectant management.
- Offer induction of labour

To 40+6 weeks

At 41 weeks

Elective Induction

- Explain procedure & book induction
- Establish Bishop score
- Cervical-ripening before induction if necessary

Expectant Management

- Daily fetal movement counts
- NSTs twice weekly
- US for AFV twice weekly

Induce

If AFV assessment not available OR if NST or AFV abnormal

To 40+6 weeks

At 41 weeks

Induction of Labour
Chapter 7 Umbilical Cord Prolapse ................................................................. 145
  Background .................................................................................................. 145
    Incidence .................................................................................................. 145
    Morbidity and Mortality .......................................................................... 146
    Risk Factors and Risk Reduction ......................................................... 146
  Diagnosis .................................................................................................. 147
  Management .............................................................................................. 147
    Recommended actions ............................................................................. 147
  Summary .................................................................................................... 148
Chapter 7

Umbilical Cord Prolapse

Background
In overt umbilical cord prolapse, the cord lies below the presenting part of the fetus and protrudes through the cervix after rupture of membranes. In occult umbilical cord prolapse, the cord is alongside the presenting part of the fetus, and membranes may or may not be ruptured. Cord presentation is the presence of the umbilical cord between the fetal presenting part and the cervix, with or without membrane rupture.

Incidence
In retrospective reviews of large samples, the incidence of cord prolapse has been reported to be from 0.1% to 0.6%. The incidence of overt cord prolapse varies with the fetal presentation. The lowest rate occurs in cephalic presentations and the highest in transverse lie presentations.
Morbidity and Mortality

There is significant morbidity associated with umbilical cord prolapse, even with appropriate treatment. Markers of possible morbidity include low Apgar scores and low cord pH. These become progressively worse with increasing decision-to-delivery times. Other markers of morbidity are not significantly increased. Perinatal mortality ranges from 0.02% to 12.6%. In Canada the number of deaths due to cord prolapse remains very low, ranging from 1 to 15 deaths per year during the years 2009 to 2013.

Risk Factors and Risk Reduction

Factors Associated With Increased Incidence

<table>
<thead>
<tr>
<th>MATERNAL AND FETAL CHARACTERISTICS</th>
<th>IATROGENIC FACTORS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unstable lie (transverse, oblique)</td>
<td>Amniotomy</td>
</tr>
<tr>
<td>Malpresentation</td>
<td>Scalp electrode application</td>
</tr>
<tr>
<td>Polyhydramnios</td>
<td>Intrauterine pressure catheter insertion</td>
</tr>
<tr>
<td>Preterm labour</td>
<td>Attempted external cephalic version</td>
</tr>
<tr>
<td>Grand multiparity (i.e., parity of ≥ 5)</td>
<td>Expectant management of preterm prelabour rupture of membranes</td>
</tr>
<tr>
<td>Male fetal sex</td>
<td>Manual rotation of fetal head</td>
</tr>
<tr>
<td>Pelvic tumours</td>
<td>Amnioreduction</td>
</tr>
<tr>
<td>Placenta previa and low-lying placenta</td>
<td></td>
</tr>
<tr>
<td>Cephalopelvic disproportion</td>
<td></td>
</tr>
<tr>
<td>Multiple gestations</td>
<td></td>
</tr>
<tr>
<td>Preterm rupture of membranes</td>
<td></td>
</tr>
<tr>
<td>Fetal congenital anomalies</td>
<td></td>
</tr>
<tr>
<td>Birth weight &lt; 2500 g</td>
<td></td>
</tr>
<tr>
<td>Unengaged presenting part</td>
<td></td>
</tr>
</tbody>
</table>

Risk Reduction

Fifty percent of obstetric interventions are responsible for cord prolapse, therefore, interventions such as amniotomy should be carefully timed, and thoughtful consideration should be given to the indications and the risks and benefits of...
the intervention. Care should be taken to ensure good application of the presenting part to the cervix before artificial rupture of membranes.

There should be an evaluation of the risks of prolapse and thus the need for fetal surveillance as soon as possible after membrane rupture.

**Diagnosis**

Overt umbilical cord prolapse is most commonly diagnosed by visualizing the cord through the introitus or by palpation of the cord in the vagina.

A sudden deceleration in fetal heart rate in women with rupture of membranes is often the first indication of cord prolapse. This should prompt vaginal examination and intrauterine resuscitation.

Occult cord prolapse must be suspected in all patients with persistent or significant decelerations on fetal heart monitoring. Variable decelerations with contractions associated with a prompt return to baseline is often seen with occult cord prolapse.

Cord presentation is diagnosed either by palpation of the cord through the membranes or as an incidental finding on ultrasound.

**Management**

Overt prolapse is an emergency situation requiring immediate and life-saving interventions.

**Recommended actions**

- Call for assistance and ensure the availability of staff capable of resuscitating a potentially depressed infant
- Perform a pelvic examination to determine
  - cervical effacement and dilatation
  - station of the presenting part
  - presence of pulsations within the cord vessels
- Initiate intrauterine resuscitation
- Elevate the presenting part, leaving the examining hand in place, and maintain the elevation until delivery. This may require insertion of the entire hand into the vagina
- Place the woman in the knee-chest or Trendelenburg position. With beds that restrict the application of Trendelenburg, it may be acceptable to elevate the woman’s hips instead of placing the bed in Trendelenburg
- Monitor the fetal heart rate
- Do not attempt to replace the cord. Keep the cord warm (with saline-soaked cloth) if it is outside the vagina, and manipulate it as little as possible.
- Prepare for immediate Caesarean section. If vaginal delivery is imminent and immediately feasible, then it is acceptable to proceed with vaginal delivery while a CS is being organized.
- If there will be a prolonged time to CS or there is a need to transport the woman to another centre, consider the following:
  - Place Foley catheter, fill bladder with 500 to 700 mL normal saline, clamp the Foley (this must be drained before Caesarean section). This is to elevate the presenting part and to suppress uterine contractions.
  - Administer tocolytic therapy.
  - Expediously perform a Caesarean section.

Health care providers should communicate with the woman and her partner/support person regarding any management processes throughout the labour and delivery.

Health care providers should ensure that women at risk are aware of:

- The potential for prolapse.
- The need for fetal surveillance as soon as possible after membrane rupture.
- Positions that might be helpful to relieve pressure on the cord while awaiting transfer to hospital.
- Interventions that will occur in hospital in the event of a cord prolapse.

It is suggested that the time from diagnosis to delivery is not the only important predictor of fetal outcomes. An expedited vaginal birth is an acceptable option, if feasible, when CS is not available or cannot be performed in a timely manner.

Cord presentation diagnosed in labour is managed by Caesarean section before rupture of membranes when there is a viable fetus.

Practising emergency drills has demonstrated improved outcomes. A 2009 retrospective study shows the impact of training on cord prolapse outcomes at a maternity unit with 5000–6000 births per annum. In 2000, the hospital introduced a multi-professional obstetric emergency training course that looked at the key interventions needed to reduce the decision-delivery interval and improve newborn outcomes. The study reviewed case records for two 7-year periods (one pre-training and one post-training) and found that decision-to-delivery interval decreased from a pre-training average of 25 minutes to a post-training average of 14.5 minutes ($P < 0.001$).

**Summary**

Umbilical cord prolapse is a true obstetrical emergency with potentially severe consequences. All obstetrical care providers and units should be intimately familiar with the diagnosis and management of this life-threatening condition. Protocols should be developed and drills practised routinely to ensure that rapid and accurate care can be provided to mitigate the effects and improve outcomes.
References


11. Deaths, by cause, chapter XVI: Certain conditions originating in the perinatal period (P00 to P96), age group and sex, Canada. [CANSIM database]. Ottawa, Ontario: 2017.


# Table of Contents

Chapter 8 Fetal Well-Being During Labour...................................................................................................................... 151

Introduction ................................................................................................................................................................. 151

Part One: The Physiologic Basis of Intrapartum Surveillance .......................................................................................... 151
  Definitions ................................................................................................................................................................. 151
  Physiology ................................................................................................................................................................. 152

Morbidity and Mortality .................................................................................................................................................. 155
  Neonatal Encephalopathy ....................................................................................................................................... 156
  Cerebral Palsy .......................................................................................................................................................... 157
  Fetal Acid-Base Balance ........................................................................................................................................ 160
  Acid-Base Assessment in Labour ............................................................................................................................. 160
  Umbilical cord blood analysis .................................................................................................................................. 162
  Interpretation of results ............................................................................................................................................. 166

Part Two: Fetal Health Surveillance in Labour ............................................................................................................... 167
  Normal labour contraction pattern ............................................................................................................................. 169
  Intermittent Auscultation .......................................................................................................................................... 171
  The Process of Systematic Interpretation ................................................................................................................... 182
  Scalp stimulation ....................................................................................................................................................... 199
  Intrauterine resuscitation ......................................................................................................................................... 200
  General considerations and recommendations ......................................................................................................... 202
  Reducing Unnecessary Interventions as a Result of Fetal Surveillance ...................................................................... 203

Maintaining Standards in Fetal Surveillance .................................................................................................................. 204

Other Technologies ......................................................................................................................................................... 206
Chapter 8
Fetal Well-Being During Labour

Introduction
The goal of intrapartum FHS is to detect potential fetal decompensation and to allow timely and effective interventions to prevent perinatal/neonatal morbidity or mortality. Intermittent Auscultation (IA) and Electronic Fetal Monitoring (EFM) are screening tests for intrapartum fetal well-being; decisions regarding which method to use are based on maternal/fetal risk of adverse events and informed decision making by the patient. Caregivers respond to FHS with actions to maintain/improve fetal oxygenation or expedite delivery. Responses to FHS should correlate with the clinical picture. Clear communication between members of the interdisciplinary team promotes effective care.¹

- It is important to recognize that intrapartum fetal surveillance is only a screening test for fetal compromise, and that no screening test is perfect. When the IA or EFM tracing is classified this refers only to the surveillance and does not necessarily reflect the status of the fetus.

Inadequate fetal monitoring is frequently cited as a component of substandard care when compensation is awarded after litigation for birth asphyxia.² A collaborative report from Accreditation Canada, Healthcare Insurance Reciprocal of Canada and the Canadian Medical Protective Association found in the ten years ending in 2015, “Failure to interpret/respond to abnormal fetal status” represented, by far, the highest risk as a proportion of maternal/newborn claims (42%).³

Part One: The Physiologic Basis of Intrapartum Surveillance

Definitions
Accurate, non-subjective terms should be used when discussing fetal health.

- Hypoxemia – decreased oxygen content in blood
- Hypoxia – decreased oxygen content in tissues
- Acidemia – increased hydrogen content in blood
- Acidosis – increased hydrogen content in tissues
- Asphyxia (hypoxic acidemia) – hypoxemia, hypercapnia and metabolic acidosis
Incidence

Atypical or abnormal EFM tracing patterns may occur in up to 80% of all labours, indicating that in most cases, fetuses with episodes of EFM tracings classified as atypical or abnormal are not actually experiencing hypoxia or asphyxia.

Physiology

Fetal Oxygenation

All human cells require oxygen and glucose to maintain aerobic metabolism, their main source of energy production. Glucose can usually be stored and mobilized when needed, but total lack of oxygen supply for just a few minutes is enough to place the cells at risk. During fetal life, oxygen supply is entirely dependent on maternal respiration and circulation, placental perfusion, gas exchange across the placenta, and umbilical and fetal circulations. Complications occurring at any of these levels may result in decreased oxygen concentration in fetal arterial blood (hypoxemia) and ultimately in the tissues (hypoxia). Some degree of hypoxemia occurs in almost all fetuses during labor, but it is the intensity, duration, and repetitive nature of the event, together with the individual variation in the capacity of each fetus to cope with the situation, that will determine the severity of the resulting hypoxia.

Difficulties in carbon dioxide (CO₂) elimination across the placenta will result in elevated CO₂ concentrations, and this gas will combine with water to increase carbonic acid (H₂CO₃) concentration, a phenomenon called respiratory acidemia. The process is quickly reversible with re-establishment of placental gas exchange, as CO₂ diffuses rapidly across the placenta. There is no evidence of injury from isolated respiratory acidemia.

When hypoxia occurs, cellular energy production can still be maintained for a limited time by anaerobic metabolism, but this process produces 19 times less energy and results in the accumulation of lactic acid inside the cell, and its dispersion to the extracellular fluid and fetal circulation. The increased concentration of hydrogen ions of intracellular origin in the fetal circulation is called metabolic acidemia, but it closely parallels hydrogen ion concentration in the tissues, so the term metabolic acidosis is frequently used as a synonym. The hydrogen ions of lactic acid are transferred very slowly across the placenta, but they are buffered by circulating bases, comprised mainly of bicarbonate, hemoglobin, and plasma proteins. The depletion of these buffering agents (increasing base deficit, or base excess in negative numbers) indicates the growing inability to neutralize hydrogen ions, and their continued production will ultimately lead to the disruption of cellular enzyme systems and to tissue injury.

Although placental permeability to oxygen is high, fetal oxygen concentration (pO₂) is markedly low compared with maternal oxygen concentration (40 mm Hg in umbilical vein vs. 95 mm Hg in maternal artery). However, oxygen saturation and content in the umbilical vein are almost identical to those in maternal arterial blood. This is because of a higher hemoglobin concentration in fetal blood and its higher affinity for oxygen. The fetal oxygen dissociation curve is shifted to the left and is steeper than the maternal curve. This allows the fetus to have a higher oxygen saturation and content at a low pO₂ value and produces a larger fall in oxygen saturation (releases oxygen to the tissues). Another important compensatory mechanism for the low fetal pO₂ is increased tissue oxygen extraction and a high organ blood flow secondary to high fetal cardiac output.
The fetus depends on the transfer of oxygen from the maternal lungs to the maternal blood, delivery of that oxygen to the uterus and placenta, diffusion of the oxygen across the placenta to fetal blood, and finally the distribution of fetal blood to fetal tissues through fetal cardiovascular activity. A problem with any of these processes will reduce the availability of oxygen to the fetal tissues.

During the contractions of normal labour there is a decrease in uteroplacental blood flow. The reduction in blood flow results in diminished oxygen delivery to the fetus. This causes an increase in pCO\(_2\), a decrease in pO\(_2\), and a decreased pH. These changes do not fall outside the normal range. The healthy fetus compensates and recovers during the resumption of normal placental perfusion that occurs between contractions. As a result, the healthy fetus usually does not display any changes in heart rate. When there has been chronically compromised uteroplacental function, the increase in pCO\(_2\) and the decrease in pO\(_2\) and pH, may exceed critical thresholds, and there may be changes in the fetal heart rate. A long labour or excessive uterine contractions may challenge even a fetus with normal uteroplacental function.

### a) Maternal factors affecting fetal oxygenation

Decreased maternal arterial oxygen tension:

- Respiratory disease
- Hypoventilation, seizure, trauma
- Smoking

---

*Fetal Well-Being During Labour*
Decreased maternal oxygen carrying capability:

- Significant anemia (e.g., iron deficiency, hemoglobinopathies)
- Carboxyhemoglobin (e.g. smoking: tobacco, cannabis)

Decreased uterine blood flow:

- Hypotension (e.g., blood loss, sepsis)
- Regional anaesthesia
- Maternal positioning

Maternal conditions:

- Vasculopathies (e.g., systemic lupus erythematosus, chronic hypertension)
- Diabetes mellitus (type 1 and 2)
- Antiphospholipid syndrome
- Medical disease (e.g. Cyanotic heart disease, Chronic obstructive pulmonary disease, hyperthyroidism, renal)
- Cholestasis of pregnancy
- Pre-pregnancy BMI > 35

b) Uteroplacental factors affecting fetal oxygenation

Excessive uterine activity:

- Tachysystole secondary to oxytocin, prostaglandins or normal labour
- Placental abruption

Uteroplacental dysfunction:

- Placental abruption
- Placental infarction-dysfunction marked by intrauterine growth restriction, oligohydramnios, or abnormal Doppler studies
- Chorioamnionitis
- Uterine rupture

C) Fetal factors affecting fetal oxygenation

- Preterm (< 37 weeks) or Postterm (>42 weeks) fetus in labour
- Malpresentation (e.g., breech)
- Polyhydramnios
- Oligohydramnios
- Cord compression, prolapse, or entanglement (e.g., 3 or more nuchal loops or umbilical cord knots)
- Velamentous cord insertion
A 2018 study of 53 singletons with velamentous cord insertions (matched with 103 controls) found associations with pH < 7.20, 5 min. Apgar < 7 and cord avulsion requiring manual placental extraction. There was also trends for increased surgical delivery for EFM changes, Bwt < 10%, abruption and fetal and neonatal death.

- *Single umbilical artery (due to less Wharton's jelly cushioning and umbilical cord coiling)*\(^{23}\).
- In a 2014 study of 34 196 pregnancies Ashwal et al. reported that the 162 fetuses with single umbilical artery were associated with:
  - a higher rate of Caesarean section (CS) due to non-reassuring fetal heart rate (5.5% vs. 1%, \(P = 0.02\))
  - small for gestational age (14.3% vs 4.9%, \(P = 0.009\))
  - lower birth weight
  - higher rate of composite adverse outcome (CS or operative delivery because of non-reassuring fetal heart rate, prolonged neonatal admission, 5-minute Apgar score < 7 and umbilical artery pH < 7.2) (20.9% vs. 8.8%, \(P = 0.005\))\(^{24}\)
- In a 2015 study of 27 752 pregnancies with 127 fetuses with single umbilical artery, Naveiro-Fuentes et al. recommended monitoring fetal growth closely and monitoring intrapartum fetal surveillance because of its association with a lower weight for gestational age, higher risk of low umbilical cord blood pH and increased Caesarean section for abnormalities in fetal surveillance.

- Decreased fetal oxygen carrying capacity
- Significant anemia (e.g., isoimmunization, fetal-maternal bleed, ruptured vasa previa)
- Carboxyhemoglobin (maternal smoking)

### d) Fetal response to hypoxia/asphyxia

Reduction in oxygen delivery to the fetus produces cardiovascular, metabolic, and behavioral responses, including:

- **Redistribution of fetal blood flow**
  - increased flow to the brain, heart, and adrenal glands
  - decreased flow to the kidneys, lungs, gut, liver, and peripheral tissues
  - increase in blood pressure
- Decreased movement, tone, and breathing action (changes in biophysical profile)
- Fetal tachycardia (this may be preceded by transient bradycardia)
- Anaerobic metabolism (decreased pH)

### Morbidity and Mortality

The central nervous system of the fetus remains vulnerable to damage throughout gestation, but this is particularly significant between 28 and 32 weeks’ gestation.

Because of the fetal physiologic response to hypoxia (redistribution of blood flow to vital organs including the brain), any injury to the fetal brain as a result of intrapartum hypoxia must be associated with injury to other organ systems since the other systems will have been deprived of oxygen first.
1. Neonatal Encephalopathy

Neonatal encephalopathy (NE) is a clinically defined syndrome of disturbed neurologic function in the earliest days of life in an infant born at or beyond 35 weeks of gestation, manifested by a subnormal level of consciousness or seizures, and often accompanied by difficulty with initiating and maintaining respiration and depression of tone and reflexes. NE and its subset of hypoxic-ischemic encephalopathy are conditions defined for term infants (> 37 completed weeks of gestation) and near-term infants (> 34 completed weeks of gestation). The incidence of hypoxic-ischemic encephalopathy is reported as 1.9 per 1000 term births and the incidence of NE is 3.8 per 1000 term births.

The fetus lives in a relatively hypoxic environment but normally exists with a surplus of oxygen to meet its metabolic needs. In response to impairments in blood gas exchange, adaptive mechanisms usually maintain fetal oxygenation. This process is known as compensation. Hypoxia can occur in degrees. In the simplest form, hypoxia will be brief and the fetus will easily cope with the physiologic changes that occur (e.g., intermittent cord compression). When hypoxia continues over time, the fetus begins trying to meet its metabolic needs in a less than optimum oxygen environment. This can lead to metabolic acidosis. Hypoxia plus metabolic acidosis results in asphyxia. The severity and duration of asphyxia will affect the outcome. The aim of fetal surveillance in labour is to detect signs of a decompensating fetus before damage. Sustained hypoxia almost always results in hypotension and ischemia. Regardless of the mechanism, cerebral ischemia is the final common pathway leading to brain injury. Other mechanisms include diminished systemic perfusion, emboli (largely cardiac in origin), thrombosis (often from tissue wall damage), or hypercoagulable states. There is no evidence to suggest that systemic hypoxia alone can produce irreversible brain damage. The severity and duration of asphyxia will affect the outcome.

Neonatal encephalopathy results from many conditions. Seventy percent of cases are secondary to events that occur before labour. These events include prenatal stroke, infection, cerebral malformation, genetic disorders, and others. Only 19% of cases of NE meet the criteria for intrapartum hypoxia, while another 10% experience a significant intrapartum event that may be associated with intrapartum hypoxia. The pathway from intrapartum asphyxia to subsequent cerebral palsy must progress through NE. The incidence of NE that can be attributed to intrapartum hypoxia, in the absence of any other pre-conceptional or antepartum abnormalities, is approximately 1.6 per 10 000.

Ultimately, sustained hypoxia leading to severe metabolic acidosis and cardiovascular decompensation with systemic hypotension may lead to cerebral ischemia and brain damage. Asphyxia is the most common pathogenic mechanism underlying hypoxic-ischemic encephalopathy. Asphyxia may occur at any point in the infant’s antepartum, intrapartum, or postpartum life. Isolated hypoxia does not cause cerebral damage unless it is prolonged, severe, and associated with a vulnerable fetus.

The type of cerebral injury caused by hypoxic-ischemia depends on the nature of the insult, the maturation of the brain and its vascular development.

- At term, the injury is predominately to the subcortical white matter and cerebral cortex. The areas between the end branches of the major cerebral vessels are the areas of the brain at highest risk. The damage is usually to the motor cortex that controls the proximal and upper extremities. Spastic quadriplegia is by far the most common outcome. A severe hypoxic/hypotensive insult may affect deeper brain tissues.
Hypoxic-ischemia in the preterm fetus is more likely to cause damage to the periventricular white matter. The resulting lesion is called periventricular leukomalacia. Moderate injury generally affects the lower limbs while severe lesions frequently involve both extremities. The long-term manifestations include spastic diplegia, spastic quadriplegia, and other visual and cognitive deficits.

In humans, no threshold of hypoxia has been determined that reliably predicts biologic injury. Severe metabolic or mixed metabolic acidosis, indicating decompensation with damage to target organs such as lungs, heart, and kidneys, is required to diagnose fetal asphyxia.

### 2. Cerebral Palsy

Cerebral palsy (CP) is considered a neurological disorder caused by a non-progressive brain injury or malformation that occurs while the child’s brain is under development. CP is characterized by the early onset of abnormal movements or postures. "Research supports that spastic quadriplegia, especially with associated movement disorders, is the only type of CP associated with acute interruption of blood supply. Purely dyskinetic or ataxic CP, especially when there is an associated learning difficulty, commonly has a genetic origin and is not caused by intrapartum or peripartum asphyxia.”

**Incidence**

The incidence of CP in term infants is 2 to 3/1000 live births and has remained stable for the past 30 to 40 years.

Advances in neonatal care have increased the survival of extremely premature neonates. The result has been an increase in the incidence of CP in these very low birth weight babies. However, the small number of these small babies, relative to the overall population, has had no significant effect on the total incidence of CP.
Factors Associated with CP

- Maternal medical conditions (e.g., hypertension)
- Multiple gestation
- Preterm infants
- Intrauterine growth restriction
- Autoimmune conditions
- Trauma
- Asphyxia (antepartum, intrapartum, or neonatal)
- Neonatal respiratory complications
- Infection (clinical chorioamnionitis and severe—not mild—histological chorioamnionitis)
- Central nervous system anomalies
- Metabolic abnormalities
- Developmental abnormalities
- Substance abuse and/or smoking
- Placental abnormalities

Term and near-term infants make up at least 50% of all cases of CP even though they are at relatively low risk compared with very preterm infants. Infants weighing < 1500 g at birth make up approximately 25% of all cases of CP.

Is cerebral palsy the result of intrapartum events?

Many studies reveal that CP is rarely the result of adverse intrapartum events. Even if there has been intrapartum hypoxia and/or acidosis, CP rarely occurs. Only 10% to 20% of children with CP had demonstrated intrapartum asphyxia.

Criteria to define an Acute Intrapartum Hypoxic Event as Sufficient to Cause Cerebral Palsy

In 2014, the Report of the American College of Obstetricians and Gynecologists’ Task Force on Neonatal Encephalopathy emphasized that there are multiple causal pathways that lead to cerebral palsy in term infants and that knowledge gaps still preclude a definitive test or set of markers that will identify an infant in whom neonatal encephalopathy is attributable to an acute intrapartum event. To determine the likelihood that an acute intrapartum hypoxic-ischemia event contributed to neonatal encephalopathy, a comprehensive assessment is necessary of neonatal status and all potential contributing factors, including maternal medical history, obstetric antecedents, intrapartum factors (including fetal surveillance and issues relating to the delivery itself), and placental pathology.

When more of the following elements are met, it becomes more likely that an acute peripartum or intrapartum hypoxia-ischemia event played a role in the pathogenesis.

1. Neonatal signs:
   a. Apgar score < 5 at 5 and 10 min.
   b. Fetal umbilical artery pH < 7.0, and/or base deficit ≥ 12 mmol/L
   c. Neuroimaging evidence of acute brain injury seen on brain MRI or MR spectroscopy
d. Presence of multisystem organ failure consistent with hypoxic-ischemic encephalopathy (e.g., renal or hepatic injury, hematologic or metabolic abnormalities, cardiac dysfunction, gastrointestinal injury)

2. Type and timing of contributing factors:
   a. A sentinel hypoxic or ischemic event occurring immediately before or during labour and delivery (e.g., ruptured uterus, placental abruption, umbilical cord prolapse)
   b. Fetal heart rate (FHR) patterns consistent with an acute peripartum or intrapartum event
   c. Timing and type of brain injury patterns based on imaging studies consistent with an etiology of an acute peripartum or intrapartum event
   d. No evidence of other proximal or distal factors that could be contributing factors (e.g., abnormal fetal growth, maternal infection, fetal and/or maternal hemorrhage, neonatal sepsis, and chronic placental lesions)

3. Developmental outcome is spastic quadriplegia or dyskinetic cerebral palsy (other subtypes of cerebral palsy are less likely to be associated with acute intrapartum hypoxic-ischemic events).
   - There is a continuum of increasing risk of NE with worsening acidemia, but even in the presence of significant acidemia, most newborns will be neurologically normal. The presence of metabolic acidemia does not define the timing of the onset of a hypoxic-ischemic event. MRI is the modality that best defines the nature and extent of cerebral injury in NE. Distinct patterns of abnormalities are recognized in hypoxic-ischemic cerebral injury and have prognostic value for predicting later neurodevelopmental impairments. If no injury is noted on MRI after 24 hours of life then it is unlikely that significant peripartum or intrapartum hypoxic-ischemic brain injury was a significant factor in NE. The full extent of injury may not be evident on MRI until after the first week of life.26

A 2012 cohort study of 51,519 neonates who had validated umbilical cord arterial pH values found that the risk of adverse neurological outcome was significantly increased below 7.10 (0.36%) and more so below 7.00 (2.95%). Seventy-five percent of the neonates with abnormal neurological outcomes had pH levels above 7.10.36

Precise and effective communication and documentation are essential.

Do not
- Use the term “fetal distress”
- Overstate the significance of meconium
- Use the term “asphyxia” without hard evidence including abnormal umbilical cord blood gas results
- Use qualifiers such as “significant” or “severe”

Instead use
- Normal or abnormal IA or normal, atypical or abnormal EFM tracing
- “Asphyxia” only with biochemical evidence (e.g. scalp pH, cord blood gases)

Summary
- Despite improved technology and neonatal care, rates of CP are still 2 to 3/1000 live births
- Most documented asphyxia does not result in CP
- Most infants diagnosed with CP had uncomplicated term deliveries
- Available tests of fetal well-being are not highly predictive of adverse central nervous system outcomes
Fetal Acid-Base Balance

Normal metabolic processes in the fetus result in a continuous production of hydrogen ions that are buffered by various mechanisms to maintain a stable pH level.

Definitions of Terms

**pH:** pH is a scale used to express the degree of acidity or alkalinity.

**Buffer:** Substances that interact with acids in the body to minimize changes in pH. The 2 main buffers are hemoglobin and plasma bicarbonate. Bicarbonate is the buffer result most often reported when a blood gas analysis is performed.

**Base deficit/excess:** Base deficit (positive number) refers to the number of units of base required to neutralize the amount of acidosis occurring. Base excess (negative number) refers to the number of units of base that would need to be reduced to reach neutrality.

3. Acid-Base Assessment in Labour

**Fetal scalp blood sampling**

Fetal scalp blood sampling can reduce the increased operative intervention rates associated with electronic fetal monitoring.

Placental perfusion is reduced with every uterine contraction. Most term fetuses enter labour with normal placental function and tolerate labour well. However, when placental function is not adequate, as in women with hypertension in pregnancy or fetuses with intrauterine growth restriction, exposure to uterine contractions may lead to the rapid development of fetal respiratory and metabolic acidosis. Glucose metabolism in the absence of oxygen results in an increase in lactic acid. Fetal scalp blood gas or lactate sampling can provide valuable objective clinical information to help guide decision-making about the preferred timing and method of delivery.

**Indications**

- Gestational age > 34 weeks’ gestation when delivery is not imminent
- Resources available to perform the analysis and respond to the results in a timely manner
- Membranes ruptured, cervix at least 2 to 3 cm dilated
- Women with atypical and/or abnormal EFM tracings

**Contraindications:**

- Gestational age ≤ 34 weeks
- Face presentation
- Known or suspected fetal bleeding disorder (hemophilia, thrombocytopenia)
- Family history of a bleeding disorder (hemophilia, von Willebrand)
- Active maternal infection (HIV, genital herpes, hepatitis, known or suspected intrauterine sepsis)
Fetal scalp blood gas testing:

- typically requires a 30 to 50mcL blood sample, and sampling failure rates as high as 21% have been reported. This may be due to inadequate volume or contamination with air or amniotic fluid. pH alone is a less accurate predictor of hypoxia than full blood gas assessment, which includes base deficit. BD is helpful in quantifying metabolic acidosis.

- A 2017 retrospective study of 343 deliveries where the EFM tracing was not normal (would include atypical and abnormal tracings) and fetal blood gas assessment (FBGA) performed, found the PPV was only 50% but the NPV was 91% for predicting postpartum acidosis (pH ≤ 7.15). The authors concluded that FBGA can be used to rule out but not rule in neonatal acidosis. Effectively, they found that it can avoid unnecessary interventions such as CS or AVB in up to 90% of cases but not reliably detect fetal acidosis.

- recommended actions related to pH level:
  - pH ≥ 7.25: Scalp sampling should be repeated within 30 minutes only if the FHR abnormality persists.
  - pH 7.21 to 7.24: Scalp sampling should be repeated within 30 minutes or delivery should be considered if there has been a rapid fall since the last sample.
  - pH ≤ 7.20: Delivery is indicated.

Fetal scalp blood lactate testing:

- This point of care test requires as little as 0.6 mcL, which reduces sampling failures.

- A 2015 Cochrane review compared intrapartum fetal lactate testing with pH estimation and found that lactate testing was 99% successful in predicting hypoxia compared with only 79% for pH testing. Fewer incisions were needed to draw blood for lactate testing than for pH testing, and time from sampling to result was shorter. Despite these potential advantages of lactate testing, there was no difference in mode of delivery, neonatal outcomes, Apgar scores, encephalopathy, or admission to the neonatal intensive care unit. However, there was no available evidence comparing the effect of fetal scalp blood lactate estimation with no sampling on clinical outcomes.

- Although there appears to be an increase in mean umbilical cord blood lactate level from 370 to 420 weeks’ gestation, no studies have addressed whether the gestational age should be considered in determining the critical level of scalp lactate at which clinical action must be taken. Fetal scalp blood samples taken for lactate measurement within 60 minutes of birth correlate well with umbilical arterial and venous lactate measurements following delivery. Lactate levels correlate well with both fetal scalp and cord blood pH and BD.

- Predictive lactate values and levels for intervention vary significantly depending on the lactate analyzer used and the manufacturers’ recommendation; therefore, decision-making criteria should be adjusted according to the device used.

- It may be that the value of scalp lactate testing lies in its strong negative predictive value for fetal acidemia at birth.

Interpretation of results

The results of fetal scalp sampling should be interpreted with the total clinical picture in mind. This includes the clinical features of the mother and baby, the existence of any prenatal risk factors, gestational age, the duration of labour,
progress in labour, the presence of meconium, maternal fever, and the severity of the atypical and/or abnormal FHR characteristics.

The trend of the fetal scalp sampling values should be determined as it will indicate whether the fetal condition is improving or deteriorating and therefore what, if any, intervention is warranted. Although there is little evidence with respect to the frequency of fetal scalp sampling. The Society of Obstetricians and Gynaecologists of Canada (SOGC) had recommends that if the clinical picture does not improve or delivery is not imminent, a repeat sample should be considered within 30 to 40 minutes.

Limitations of fetal scalp blood sampling

- It provides only instantaneous and not continuous information. Repeat sampling may be necessary.
- There are technical limitations including equipment availability, operator experience and skill level.
- The procedure may be uncomfortable for the woman.

4. Umbilical cord blood analysis

Dr Virginia Apgar developed the Apgar score as a rapid tool to assess the immediate status of the newborn and the need for resuscitation. It was not developed as a method of assessing the degree of asphyxia. The Apgar score alone cannot link birth events to neurological sequelae. A low Apgar score can be associated with various maternal-fetal conditions including fetal malformation, infection, meconium aspiration, vigorous manipulation of the upper airway, and immaturity.

Umbilical cord blood gas analysis provides an objective method to evaluate the fetal condition at delivery. This facilitates effective newborn care and quality assurance and/or improvement initiatives.

Recommendations for cord blood analysis from professional organizations:

- The SOGC strongly recommends measuring both umbilical arterial and venous cord gases after all births, as they may help in providing appropriate care to the newborn and in planning subsequent management. They also assist quality assurance / improvement initiatives.¹

In hospitals where blood gas analysis is not immediately available, alternatives include the following:

- Cord blood samples in pre-heparinized syringes are most accurate and stable at room temperature for 60 minutes.⁴⁹
- If not analyzed within 60 minutes the sample should be stored at 4-8°C and the time of analysis documented.
- A clamped, 20 cm segment of cord for delayed analysis. Umbilical artery blood is stable for pH analysis for up to 60 minutes at room temperature.⁵⁰
- A 2006 study which sampled umbilical arterial and venous lactate, base excess, pH and PCO₂ at 0, 20, 40 and 60 minutes from a doubly clamped segment and a segment still attached to the placenta (unclamped) found the pH was stable in the clamped vessels over the 60 minutes, however, there was a steady decrease in the pH of the unclamped vessels. The base excess was significantly lower by 20 min. in the unclamped cord and
by 40 min. in the clamped cord. Lactate levels were significantly higher by 20 min. in both the unclamped and clamped segments. The study concluded:

- Delayed sampling from unclamped cords is unreliable for pH, base deficit or lactate by 20 min.
- For clamped cords, the pH is stable for 60 min. but base excess is unreliable by 40 min and lactate is unreliable by 20 min.

Rationale for routine umbilical cord blood gas analysis

The objective of measuring cord pH and acid-base status is to quantify the degree of perinatal asphyxia. Blood gas measurements reflect fetal and placental oxygenation at birth and assist in the provision of appropriate care to the newborn and in planning ongoing management. A complete blood gas analysis is necessary (pH, BD, pCO₂, HCO₃, pO₂, O₂ saturation) since the use of pH alone will not differentiate between respiratory and metabolic acidosis.

The availability of cord blood gas analysis may reduce the incidence of successful litigation in the event of a poor outcome, particularly when the outcome is delayed (i.e., not apparent in the neonate).

A 2016 study of 26,669 term singletons whose 5-minute Apgar scores were all ≥ 7 found that 0.5% of these had an umbilical cord arterial pH of ≤ 7.0 and 1.4% had a base excess ≤ −12. These infants were found to be at increased risk of NICU admission because of neonatal complications such as respiratory distress syndrome and sepsis. The authors concluded that universal rather than selective umbilical cord gas analysis would enable better stratification of neonatal risk levels and care.

An observational study of approximately 20,000 births in Australia from 2003 to 2006 during which universal umbilical (arterial and venous) cord blood gas and (arterial) lactate analysis was performed found a progressive improvement in these values during the study. The authors concluded that the improvement was independent of obstetric interventions and suggested that it was due to the provision of fetal acid-base biochemical data at delivery, which progressively influenced care.

Normal Umbilical Cord Blood Gas Values

Reference Ranges of 3522 Term* Vaginal Deliveries

<table>
<thead>
<tr>
<th>ARTERIAL</th>
<th>MEAN</th>
<th>SD</th>
<th>RANGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.27</td>
<td>0.07</td>
<td>7.2 to 7.34</td>
</tr>
<tr>
<td>pCO₂ (mmHg)</td>
<td>50.3</td>
<td>11.1</td>
<td>39.2 to 61.4</td>
</tr>
<tr>
<td>pO₂ (mmHg)</td>
<td>18.4</td>
<td>8.2</td>
<td>10.2 to 26.6</td>
</tr>
<tr>
<td>HCO₃ (mEq/L)</td>
<td>22</td>
<td>3.6</td>
<td>18.4 to 25.6</td>
</tr>
<tr>
<td>Base excess (mEq/L)</td>
<td>−2.7</td>
<td>2.8</td>
<td>−5.5 to 0.1</td>
</tr>
<tr>
<td>O₂ saturation (%)</td>
<td>23.3</td>
<td>16.2</td>
<td>7.1 to 39.5</td>
</tr>
</tbody>
</table>
Fetal Well-Being During Labour

<table>
<thead>
<tr>
<th>VENOUS</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.34</td>
<td>0.06</td>
<td>7.28 to 7.40</td>
</tr>
<tr>
<td>pCO₂ (mmHg)</td>
<td>40.7</td>
<td>7.9</td>
<td>32.8 to 48.6</td>
</tr>
<tr>
<td>pO₂ (mmHg)</td>
<td>28.5</td>
<td>7.7</td>
<td>20.8 to 36.2</td>
</tr>
<tr>
<td>HCO₃ (mEq/L)</td>
<td>21.4</td>
<td>2.5</td>
<td>18.9 to 23.9</td>
</tr>
<tr>
<td>Base excess (mEq/L)</td>
<td>−2.4</td>
<td>2.0</td>
<td>−4.4 to 0.4</td>
</tr>
<tr>
<td>O₂ saturation (%)</td>
<td>49.4</td>
<td>16.9</td>
<td>32.5 to 66.3</td>
</tr>
</tbody>
</table>

*Reference ranges of preterm neonatal umbilical cord gas values are similar to those at term.

Considerations regarding umbilical cord blood gas analysis include:

1. A 2009 Cochrane review suggests that delayed cord clamping may confer some benefit in preterm infants not requiring resuscitation. Evidence suggests that delayed clamping of the cord for 30 to 120 seconds after birth in preterm infants reduces the rate of intraventricular hemorrhage, anemia, and the need for transfusions. In term infants, delays of up to 180 seconds after birth have not been associated with adverse outcomes and have resulted in increased iron stores in these infants at 6 months of age although they may increase the need for phototherapy. There is no evidence to support early cord clamping as part of active third stage management in preventing postpartum hemorrhage. A delay in cord clamping of at least 1 minute provides sufficient placental fetal transfusion. The Canadian Paediatric Society, in its 2017 Practice Point, Update for Canadian NRP providers: A case based review stated: For vigorous term and preterm infants, delayed cord clamping for 30-60 s is recommended.

There is evidence delayed umbilical cord clamping may have a minor influence arterial blood gas values:

- Lievaart et al., in 1984, determined that when arterial cord blood samples taken within seconds of birth were compared with samples taken at 60 seconds, there was a mean decrease in pH of 0.043 (range 0.008 to 0.076) and an increase in the BD of 1.3 (range 0.2 to 3.0). These changes were not observed in the venous samples.

- Wiberg et al., in 2008, sampled cord arterial and venous blood immediately and at 45 and 90 seconds. Compared with immediate sampling, the mean arterial pH declined from 7.24 to 7.21 and the BD increased from 4.85 to 6.14. Corresponding venous samples had a much smaller mean pH decrease from 7.32 to 7.31 and BD increase from 4.93 to 5.19.

It is therefore important to document when the cord was clamped and when blood was drawn.

2. Sampling both the umbilical artery and umbilical vein is recommended:

- The umbilical artery values correlate better with fetal status than venous values, which correlate better with the placenta.

- The distended umbilical vein stabilizes the arteries making it easier to sample the artery first.
− In order to ensure enough filling of the umbilical arteries, the cord can be milked from the placenta to the first clamp before the second is applied; this may be helpful when delayed cord clamping is performed.
− If the cord samples are inadequate, samples may be obtained from the fetal (chorionic) side of the placenta (arteries pass over top of the veins).
− An umbilical vein sample is required for quality control. Without an umbilical vein sample there is no way to identify sample error. Up to 25% of samples assumed to be arterial are, in fact, venous. Collecting both arterial and venous samples will confirm that the source is arterial or venous. This is especially valuable in situations where risk factors for fetal compromise are present. The suggested normal range of difference between vessels is 0.03 in pH and 8 mm Hg in pCO₂.

3. To identify the type and severity of fetal acidosis. The following example of 2 cases with similar arterial but different venous values had dissimilar neonatal outcomes. The infant in case A required resuscitation at birth and assisted ventilation for 48 hours, and developed CP at 1 year of age. The infant in case B had a 5-minute Apgar score of 8 with no neonatal problems.

<table>
<thead>
<tr>
<th></th>
<th>CASE A</th>
<th></th>
<th>CASE B</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Artery</td>
<td>Vein</td>
<td>Artery</td>
</tr>
<tr>
<td>pH</td>
<td>7.03</td>
<td>7.10</td>
<td>7.04</td>
</tr>
<tr>
<td>pCO₂ (mmHg)</td>
<td>63</td>
<td>50</td>
<td>67</td>
</tr>
<tr>
<td>pO₂ (mmHg)</td>
<td>6.8</td>
<td>20</td>
<td>13.5</td>
</tr>
<tr>
<td>BE (mmol/L)</td>
<td>−12.5</td>
<td>−12.6</td>
<td>−11.2</td>
</tr>
</tbody>
</table>

4. Normal blood gas values change during labour: pH, bicarbonate, and pO₂ decrease, and pCO₂ and BD increase.

5. Blood gas analyzers assess pH and pCO₂ directly, but BD is calculated and will vary depending on whether the calculation uses blood or extracellular fluid. Generally BD values calculated using blood are greater than those using extra cellular fluid.

6. In the presence of a high pCO₂, metabolic acidosis may be erroneously reported when BD is calculated using blood. In the perinatal period, calculating BD using extracellular fluid will prevent the influence of pCO₂ on reporting of metabolic acidosis. Care providers should be aware that most hospitals do not recognize this fact and analyze the acid-base status using blood.

7. A low umbilical artery pH by itself does not define asphyxia. All of the criteria, as noted in the CP and fetal asphyxia sections, should be present to correlate birth hypoxia with adverse neurological outcome.

8. Arterial-venous pH differences may add information about the cause of the acidemia at birth. Restriction of umbilical flow increases the difference between umbilical arterial and umbilical venous pH values, while impairment of maternal perfusion of the placenta may be associated with small differences.
Neonatal morbidity and mortality according to pH cut-off

<table>
<thead>
<tr>
<th>pH</th>
<th>NEONATAL DEATHS</th>
<th>SEIZURES</th>
<th>BOTH</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.15 to 7.19 (n = 2236)</td>
<td>3 (0.1%)</td>
<td>2 (0.1%)</td>
<td>1 (0.05%)</td>
</tr>
<tr>
<td>7.10 to 7.14 (n = 798)</td>
<td>3 (0.4%)</td>
<td>1 (0.1%)</td>
<td>0</td>
</tr>
<tr>
<td>7.05 to 7.09 (n = 290)</td>
<td>0</td>
<td>0</td>
<td>1 (1.1%)</td>
</tr>
<tr>
<td>7.00 to 7.04 (n = 95)</td>
<td>1 (1.1%)</td>
<td>1 (1.1%)</td>
<td>1 (1.1%)</td>
</tr>
<tr>
<td>&lt; 7.00 (n = 87)</td>
<td>7 (8%)</td>
<td>8 (9.2%)</td>
<td>2 (2.3%)</td>
</tr>
</tbody>
</table>

**Interpretation of results**

The pathogenesis and clinical significance of respiratory acidosis are different from those of metabolic acidosis. Respiratory acidosis is considered a part of normal birth. It develops rapidly and disappears rapidly following the first neonatal breaths when the newborn is able to eliminate CO$_2$ through respiration. Respiratory acidosis occurs in the blood vessels and develops when interruption of blood flow occurs, for example with cord compression, causing a decrease in CO$_2$ transport from the fetus to the placenta. Carbon dioxide accumulates and after reacting with water produces hydrogen ions and bicarbonate. When the hydrogen ions exceed the buffer capacity of the blood, they accumulate in the vessel causing a decrease in pH.

Metabolic acidosis, on the other hand, develops as a result of fetal hypoxia that causes the fetus to shift to anaerobic metabolism (metabolism in a less than optimum oxygen environment) to maintain a positive energy balance. Lactic acid is produced in the tissue and is dissociated to lactate and hydrogen ions. Some of the latter find their way to blood vessels, reducing the pH value. Metabolic acidosis is generated in hypoxic tissues, takes longer to develop and to disappear, and may be associated with significant fetal damage.

<table>
<thead>
<tr>
<th>TYPES OF ACIDOSIS (DECREASED pH)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory</td>
</tr>
<tr>
<td>Metabolic</td>
</tr>
<tr>
<td>Mixed</td>
</tr>
</tbody>
</table>
Part Two: Fetal Health Surveillance in Labour

The goal of intrapartum fetal health surveillance is to perform a screening test to detect potential fetal decompensation and to intervene early enough to prevent fetal injury or death. The fetal brain, which is the primary organ of interest, cannot currently be evaluated antenatally. Characteristic FHR changes often precede brain injury. The most valuable non-invasive method of intrapartum evaluation that is currently available is FHR assessment. A consistent and standard approach to FHR monitoring may provide an opportunity for early intervention.

- The data obtained from intermittent auscultation and the electronic fetal monitor should always be interpreted in conjunction with the total clinical picture. Interpretation of the fetal heart pattern and the management plan are dependent on the fetal health before labour, the maternal clinical condition, the stage of labour, and the uterine activity.
- A systematic method for interpretation, documentation, and communication will help to prevent missed steps in the assessment process and the omission of important elements of documentation and communication.
- All care providers need to have a formal process for communication about the status of the fetus during labour that uses standardized terminology.
- Supportive care in labour is an extremely important adjunct to all fetal surveillance techniques and should be the basis of intrapartum care.

Benefits of Continuous Support During Childbirth

<table>
<thead>
<tr>
<th>Outcome</th>
<th>RR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any analgesia</td>
<td>0.90 (0.84–0.97)</td>
</tr>
<tr>
<td>Regional analgesia</td>
<td>0.93 (0.88–0.99)</td>
</tr>
<tr>
<td>Artificial oxytocin</td>
<td>0.97 (0.90–1.04)</td>
</tr>
<tr>
<td>Operative vaginal delivery</td>
<td>0.90 (0.84–0.96)</td>
</tr>
<tr>
<td>Caesarean section</td>
<td>0.79 (0.67–0.92)</td>
</tr>
<tr>
<td>Low 5-min Apgar score</td>
<td>0.70 (0.50–0.96)</td>
</tr>
<tr>
<td>Dissatisfaction</td>
<td>0.69 (0.59–0.79)</td>
</tr>
</tbody>
</table>

Favours support                  Favours usual care
Principles of Intrapartum Surveillance:

Classify IA or EFM Tracing

Interpret clinically (in light of total situation)

Respond (communication and teamwork)

Choice of Method of Surveillance in Labour

Comparing Continuous EFM with IA in Labour

<table>
<thead>
<tr>
<th>Outcome</th>
<th>RR</th>
<th>(95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Caesarean section</td>
<td>1.63</td>
<td>(1.29–2.07)</td>
</tr>
<tr>
<td>Operative vaginal delivery</td>
<td>1.15</td>
<td>(1.01–1.33)</td>
</tr>
<tr>
<td>NICU admission</td>
<td>1.01</td>
<td>(0.93–1.10)</td>
</tr>
<tr>
<td>Perinatal death</td>
<td>0.86</td>
<td>(0.59–1.23)</td>
</tr>
<tr>
<td><strong>Neonatal seizures</strong></td>
<td><strong>0.50</strong></td>
<td><strong>(0.31–0.80)</strong></td>
</tr>
<tr>
<td>Cerebral palsy</td>
<td>1.75</td>
<td>(0.84–3.63)</td>
</tr>
</tbody>
</table>

When comparing continuous EFM with IA during labour, a 2013 Cochrane review (including both low- and high-risk patients) found that:

- Continuous EFM is associated with a higher rate of CS and instrumental (operative) vaginal deliveries than IA.
- There was no significant difference in perinatal mortality.
- There was no difference in the incidence of CP.
- The only clinical benefit of EFM was a 50% reduction in neonatal seizures.

Using a number-needed-to-treat calculation, the authors estimated that 667 women would have to be continuously monitored to prevent one neonatal seizure. However, as many as 61 extra caesarean sections would be associated with preventing one seizure.
A normal EFM tracing is indicative of fetal well-being. However, an atypical or abnormal tracing has low predictive value for poor neonatal outcomes.

The woman and her partner must be informed about the various methods of fetal surveillance and be involved in decisions about their use in labour and birth.

1. **Assessment of Uterine Activity**

   The fetal heart rate is assessed in relation to the uterine activity pattern. Uterine activity is evaluated to:
   
   - Identify abnormal contraction patterns that might adversely affect oxygen delivery to the fetus.
   - Correctly classify the FHR patterns with EFM.

   **FHR patterns detected by intermittent auscultation are not classified using the same terminology used for EFM.**

### Normal labour contraction pattern

#### Methods of assessment

- Palpation by hand. The frequency and duration of contractions, and an estimate of their intensity, as well as the resting tone can be determined by palpation
- External, electronic, fetal tocodynamometer: only the relative frequency and duration of contractions can be determined using an external tocodynamometer. The external transducer does not measure intensity or resting tone. Palpation should be used to determine these characteristics
- An internal intrauterine pressure catheter (IUPC) is the most accurate method. It is not used frequently because of its invasive nature and the restrictions it places on the woman’s mobility
- A combination of the above techniques

The woman’s perception of her contractions should always be considered in conjunction with the above methods.

#### Characteristics of normal contractions and their assessment:

- **Frequency:** Uterine contractions are quantified as the number of contractions in a 10-minute window, averaged over 30 minutes. Normal is ≤ 5 contractions in 10 minutes. Tachysystole is > 5 contractions in 10 minutes (averaged over 30 min).
- **Duration:** Measure from the beginning to the end of the contraction and record in seconds. Normal is < 90 seconds.
- **Intensity:** Assess how strong the contractions feel on palpation and what type of pain the woman states she is feeling. The intensity of contractions **cannot** be measured accurately with an external tocodynamometer. Intensity evaluation may be estimated by palpation (described as mild, moderate, or strong; with a strong contraction the uterus cannot be indented). Objective, accurate measurement is obtained only when using
an intrauterine pressure catheter. Normal intensity is > 25 mm Hg and < 75 mm Hg above the baseline (except in the second stage of labour). 74

- **Resting tone:** The uterine tone is described as soft or firm between contractions by palpation. The uterus is soft between contractions for a minimum of 30 seconds to allow adequate placental perfusion. Normal resting tone with an IUPC is < 7 to 25 mm Hg. 72 Resting tone cannot be accurately measured using an external tocodynamometer.

Contractions are rarely of a fixed frequency or duration. Therefore, frequency and duration may be expressed in a range. For example, 2 to 3 contractions in 10 minutes, each lasting 50 to 80 seconds.

**Tachysystole:**

Excessive uterine activity can be endogenous (spontaneous) or exogenous (overstimulation of the uterus during labour induction or augmentation). Most commonly, excessive uterine activity is secondary to the use of oxytocin or another uterotonic agent. If a uterotonic medication is not being used, consider the possibility of a placental abruption.

Tachysystole is the term used to describe all forms of excessive uterine activity:

- > 5 contractions per 10-minute period averaged over 30 minutes and/or
- Inadequate resting tone: uterine resting period between contractions of < 30 seconds OR the uterus does not return to resting tone between contractions, and/or
- Prolonged contraction: lasting > 90 seconds

Note: If EFM tracing is atypical or abnormal in the first 10 minutes of tachysystole a response is required without waiting 30 minutes

The terms hypercontractility and hyperstimulation (previously used to describe excessive uterine activity with atypical or abnormal FHR tracings) should be abandoned. 72

**Tachysystole – Normal Tracing**

![Tachysystole Tracing](image)
1. Intermittent Auscultation

Intermittent auscultation is the preferred technique for intrapartum fetal surveillance in low-risk pregnancies if the following criteria are met:

- The presence of practitioners experienced in the technique of auscultation, palpation of contractions, and the recognition of abnormal FHR patterns on auscultation
- The existence of a delivery unit protocol / guideline addressing the technique and frequency of assessment (for an example go to URL: http://sogc.org/wp-content/uploads/2013/01/gui197CPG0709r.pdf).
- The presence of a delivery unit guideline outlining the clinical interventions to be used when abnormal FHR findings are present (for an example go to URL: http://sogc.org/wp-content/uploads/2013/01/gui197CPG0709r.pdf).
- The presence of an educational program to provide fundamental knowledge and to regularly update all staff regarding the delivery unit IA protocol / guideline and the steps in clinical intervention for abnormal FHR changes detected on IA.
- The ability to provide skilled nursing support on a 1:1 basis once auscultation is required every 15 minutes or less.

Benefits of Intermittent Auscultation:

- Less costly than continuous EFM
- Less restrictive for the woman (permits increased freedom of movement)
- Adaptable to varied labour positions and practices (e.g., water immersion)
- Lower intervention rates, compared with EFM, without compromising neonatal outcome

Limitations of Intermittent Auscultation:

- May be difficult to auscultate fetal heart sounds in obese women
- Some women find it intrusive because of the frequency of auscultation
What is assessed with IA?

- Contraction pattern
- Maternal heart rate (MHR) to differentiate from FHR
- Baseline FHR (the heart rate counted in bpm for 1 minute between contractions)
  - Assessing the FHR is an indirect method for evaluating fetal oxygenation and well-being. Regulation of the FHR is under the influence of intrinsic (autonomic nervous system) and extrinsic factors.
  - The average fetal heart rate decreases as gestational age increases. It is determined by a pacemaker in the sino-atrial node in the heart.
  - FHR changes are primarily controlled by a balance between the sympathetic and the parasympathetic nervous systems. Other factors affecting FHR include
    › Drugs in maternal / fetal circulation
    › Congenital fetal cardiac defects
    › Fetal rest and activity cycles
    › Hypoxemia / hypoxia / acidemia / acidosis
    › Maternal hemodynamics.
- Rhythm (regular or irregular)
- Accelerations and decelerations (abrupt or gradual increases or decreases from the baseline heart rate)

What cannot be assessed with IA?

- Baseline variability
- Classification / type of deceleration heard. There is no research to indicate that a practitioner can distinguish the type of deceleration on auscultation. Therefore, decelerations cannot be classified as they can be when using EFM

Auscultation technique:

- Palpate the maternal abdomen to identify fetal presentation and position (Leopold's manoeuvres)
- Assess the uterine contraction pattern by hand; frequency, duration, intensity and resting tone can be adequately assessed using a hand (or hands) and a watch
- Assess the MHR to differentiate from the FHR
- Place the stethoscope or Doppler monitor over the area of maximum intensity of fetal heart sounds (usually over the fetal back or shoulder)
- Listen to hear the FHR and place a finger on mother’s radial pulse to differentiate maternal from fetal heart rate
- Establish a baseline heart rate by listening and counting between uterine contractions for a **full minute (60 seconds)**
- After the FHR baseline is established, regular assessments **immediately after contractions** for 30 to 60 seconds will determine whether the FHR is within the same range. In active labour, a 30-second auscultation period may be more feasible while a 60-second sampling period will improve accuracy.
Table 11 Recommended Frequency Of Intermittent Auscultation During Labour

Frequency of assessments, response and documentation should always consider maternal fetal status and will need to occur more frequently in the presence of abnormal FHS or other changes in the maternal/fetal condition.

<table>
<thead>
<tr>
<th>1st STAGE: LATENT PHASE</th>
<th>1st STAGE: ACTIVE PHASE</th>
<th>2nd STAGE: PASSIVE PHASE</th>
<th>2nd STAGE: ACTIVE PHASE</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Initial assessment</td>
<td>Every 15 to 30 minutes</td>
<td>At least every 5 minutes or immediately following each contraction</td>
<td></td>
</tr>
<tr>
<td>• At least every 1 hour if admitted to hospital</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Individualized based on maternal fetal status if in triage or midwifery care at home (not admitted to hospital)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Recommendations for the use of intermittent auscultation

a. IA is preferred for healthy women ≥ 37\textsuperscript{0} weeks’ gestation (up to 41\textsuperscript{3} weeks’ gestation), in spontaneous labour, and in the absence of risk factors for an adverse perinatal outcome.

b. From 41\textsuperscript{3} weeks until 42 weeks’ gestation IA is preferred, provided that a non-stress test and an amniotic fluid volume assessment are normal. Post-term pregnancy (> 42 weeks’ gestation) is associated with an increased risk of adverse fetal outcome and EFM is the preferred method of fetal surveillance.

c. Below 37\textsuperscript{0} weeks’ gestation EFM is recommended because the incidence of other pathologies and adverse outcomes is increased.

d. Intermittent auscultation with epidural analgesia or combined spinal-epidural analgesia
   - Maternal hypotension and fetal heart rate changes are often seen in the first 60 minutes after the initiation of regional anaesthesia.
   › Combined spinal-epidural analgesia is associated with higher risk of FHR changes than epidural alone.
   › The cause of the FHR changes may be related to changes in maternal blood pressure and uterine and fetal perfusion pressures. The FHR abnormalities are often seen without associated maternal hypotension and other factors play a role.
   - In general, epidural and spinal anaesthesia, in the absence of maternal hypotension or uterine hypertonus, cause minimal changes in the FHR.
   - The use of IA is appropriate after initiation of regional analgesia.
   - It is recommended that the frequency of auscultation be increased to every 5 minutes for 30 minutes after the initial dose of an epidural and following any epidural bolus injection (top-up), as long as maternal vital signs are normal.
   - Patient-controlled epidural analgesia (PCEA) uses a dilute local anaesthetic and opioid solution rather than the bolus of concentrated local anesthetic agents used in spinal and traditional continuous epidurals. It has been proven to be safe for ambulation in labour and hypotension does not occur after a self-administered bolus. Therefore, IA is acceptable when PCEA is used, and the FHR does not need to be monitored after each PCEA self-administered dose. IA should be done according to the...
usual obstetrical protocols. The use of EFM in this circumstance should be based solely on obstetrical considerations.\(^1\)

If maternal hypotension is a persistent problem, continuous EFM should be initiated.

e. For women attempting vaginal birth after Caesarean section, continuous EFM is recommended.

f. **IA assessment:**

Assess FHR before:

- Initiation of labour-enhancing procedures (e.g., amniotomy)
- Administration of medications
- Administration or initiation of analgesia / anaesthesia
- Patient transfer

Assess FHR after:

- Admission of woman
- Artificial or spontaneous rupture of membranes
- Vaginal examinations
- Tachysystole (if persistent convert to EFM)
- Any abnormal event during labour (e.g., maternal hypotension)

**IA Interpretation**

1. **Normal**

   - Normal contraction pattern
   - Normal baseline rate (110 to 160 bpm)
   - Presence of accelerations
     - accelerations suggest the presence of fetal well-being. However, since auscultation is done intermittently, the absence of accelerations on its own is not necessarily concerning and does not make the auscultation findings “abnormal.” When considering the significance of the absence of accelerations and whether other actions to determine fetal well-being are indicated, it is important to consider the auscultation findings in light of the total clinical picture, including the general activity of the fetus, the stage of labour, and other risk factors.
   - Regular rhythm

2. **Abnormal**

   - Tachysystole\(^{81}\)
   - Abnormal baseline rate
     - tachycardia (FHR >160 bpm for 10 minutes)
     - bradycardia (FHR < 110 bpm for 10 minutes)
     - Changing FHR baseline (increasing or decreasing over time)\(^2\)
• Presence of decelerations
• Arrhythmia: An irregular heart rate not associated with uterine activity. An arrhythmia requires further assessment and EFM

In the presence of abnormal FHR characteristics detected by intermittent auscultation that are unresponsive to maternal position change, increased surveillance by continuous EFM and consideration of fetal scalp sampling or delivery considered.

If EFM is initiated in response to abnormal IA, EFM does not need to remain in place for the remainder of the labour. Once a normal EFM pattern has been confirmed, usually for a minimum of 20 minutes, the EFM may be removed and IA resumed.
3. Electronic Fetal Monitoring

Terminology

<table>
<thead>
<tr>
<th>Tachysystole</th>
<th>Describes all forms of excessive uterine activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Periodic patterns</td>
<td>Those associated with uterine contractions</td>
</tr>
<tr>
<td>Episodic patterns</td>
<td>Those not associated with uterine contractions.</td>
</tr>
<tr>
<td>Repetitive decelerations</td>
<td>3 or more decelerations in a row</td>
</tr>
<tr>
<td>Recurrent decelerations</td>
<td>Occur with ≥ 50% of contractions in a 20 min. window</td>
</tr>
<tr>
<td>Intermittent decelerations</td>
<td>Occur with &lt; 50% of contractions in a 20 min. window</td>
</tr>
<tr>
<td>Episodic gradual deceleration</td>
<td>Gradual deceleration not associated with a contraction</td>
</tr>
<tr>
<td>Interpretable EFM tracing</td>
<td>EFM tracing that has a continuous display of the UA and FHR with minimal gaps</td>
</tr>
</tbody>
</table>

Admission EFM Tracing (cardiotocography or CTG) Assessment

A 2012 Cochrane review compared the effects of admission CTG with IA on maternal and infant outcomes for women without risk factors (n = 13000).83

- **Main results:**
  - although the difference is not statistically significant if the strict $P < 0.05$ criterion is used, women allocated to admission EFM had a higher probability of Caesarean section than women assessed with IA (risk ratio [RR] 1.20, 95% confidence interval [CI] 1.00 to 1.44, 4 trials, 11 338 women)
  - women having admission EFM had significantly increased use of continuous EFM during labour (RR 1.30, 95% CI 1.14 to 1.48, 3 trials, 10 753 women) and fetal blood sampling (RR 1.28, 95% CI 1.13 to 1.45, trials, 10 757 women)
  - no difference was found in the rate of instrumental vaginal birth (RR 1.10, 95% CI 0.95 to 1.27, 4 trials, 11 338 women), fetal and neonatal deaths (RR 1.01, 95% CI 0.30 to 3.47, 4 trials, 11 339 infants) or other secondary outcomes

- **For women at low risk:**
  - contrary to its present use, there is no evidence of benefit for the use of admission CTG, and it should not be used
  - admission EFM probably increases the CS rate by approximately 20%
  - women should be informed that admission CTG is likely associated with an increase in the incidence of Caesarean section without evidence of benefit83, 84

- These recommendations are supported by a 2018 multicentre randomized trial of 3045 low risk healthy women presenting with signs and symptoms of possible labour compared admission assessment with IA vs. EFM. The study found no statistical difference in caesarean section (however, low adherence to the study protocol made study quality questionable) but did find a statistically significant increased use of continuous
EFM during labour in the EFM group, therefore supporting the use of IA when assessing low risk women with signs and symptoms of possible labour.

**Recommendations:**

- Admission EFM tracing assessments **are not** recommended for healthy women at term in labour in the absence of risk factors for adverse perinatal outcome as they may lead to unnecessary interventions, and there is no evident benefit.
- Admission EFM tracing assessments **are** recommended for women with risk factors for adverse perinatal outcome.

**Methods of electronic fetal monitoring:**

- **External:** An ultrasound transducer (heart sound detection) and tocodynamometer (uterine pressure measurement) are applied to the woman’s abdomen and held in place by external belts or adhesive strips.
  
  **Advantages:**
  - Non-invasive
  - Does not require a dilated cervix
  - Does not require ruptured membranes

  **Disadvantages:**
  - Need for re-adjustment with maternal or fetal movement
  - Possibility of false or misleading recording of heart rate
  - Inability to record intensity of contractions
  - The ultrasound transducer may record the maternal pulse and may not obtain a clear tracing in obese women or women with polyhydramnios. Also, artefact may be recorded, and there may be doubling or halving of the FHR when it is outside the normal range
  - The tocodynamometer indicates approximately when the uterine contractions start and end but does not measure the intensity of the contractions

- **Internal:** A spiral electrode to record the fetal heart rate is attached to the fetal scalp through the maternal vagina and cervix. This should be considered at any time EFM is indicated when external monitor is not possible or effective. Eg. Obesity, during epidural placement. When using a spiral electrode it is essential to concurrently verify the maternal pulse to differentiate it from the presumed fetal heart rate.
  - Membranes must be ruptured. Uterine activity may be assessed concurrently using an external tocodynamometer or an IUPC.

**Contraindications to the use of a spiral electrode:**

- Placenta previa
- Face presentation
- Unknown presentation
- HIV seropositivity
- Active genital herpes
• Maternal hepatitis B or C
• Intrauterine infection

An IUPC is placed into the uterine cavity through the open cervix and transmits pressure changes in the uterus. An IUPC accurately measures the intrauterine resting tone and the intensity, duration, and frequency of contractions.

An IUPC:

• Provides an accurate measure of intrauterine pressure
• Provides a more accurate assessment than external monitoring of the relationship between contractions and FHR pattern changes
• Is useful in cases of dysfunctional labour
• Is useful in obese women when external monitoring is unsatisfactory
• Allows women greater mobility than external pressure monitoring
• An IUPC may be useful when:
  • Contraction strength is difficult to assess clinically (e.g., obesity)
  • Oxytocin doses above 30 mU/min are required
  • Augmenting labour in women with a prior CS scar
  • Amnioinfusion is required to treat variable decelerations due to cord compression.

The relative risks and benefits of the use of IUPC in the case of undiagnosed vaginal bleeding or intrauterine infection must be considered, as must the contraindications listed above for the spiral electrode.

Studies addressing fetal and maternal risks of internal monitoring report differing conclusions:

• A 2003 Alberta retrospective review of 90 cases of early-onset GBS disease (between 1993 and 1997), at a time before the recommendation for universal GBS maternal screening, identified intrauterine monitoring as an independent risk factor for early-onset GBS disease (odds ratio [OR] 2.24; 95% CI 1.22 to 4.13)\(^9\)
• A case–control study (covering the years 2000 to 2011) of 40 cases of early-onset neonatal sepsis made up of 8 GBS, 11 *Escherichia coli*, 12 coagulase-negative staphylococci, 4 viridans group streptococci, 1 *Enterococcus faecalis*, and 4 other non-specified organisms versus 80 controls, did not demonstrate a significant relationship between fetal scalp electrode (FSE) use and early-onset neonatal sepsis\(^9\)
• A 2013 retrospective cohort study by Harper et al. compared women in labour with (n = 3944) and without (n = 2501) internal monitors (FSE, IUPC, or both)\(^9\):  
  • the use of a fetal scalp electrode alone was not associated with adverse neonatal outcome (a composite of 5-minute Apgar score < 3, cord pH < 7.1, cord base excess < −2, or admission to level 3 nursery) or with maternal fever
  • the use of an IUPC alone (adjusted OR, 2.4; 95% CI, 1.8 to 3.2) or the combined FSE and IUPC (adjusted OR, 2.0; 95% CI, 1.6 to 2.5) was associated with an increased risk of maternal fever but no increase in adverse fetal outcome. These outcomes were corrected for time from rupture to delivery ≥ 12 hours, primiparity, group B streptococcus status, and regional anaesthesia. The authors recommended that an IUPC be used only when external monitoring is inadequate.\(^9\)
• A 2013 Cochrane review of 3 studies (n = 1945) comparing the ROUTINE USE of internal versus external tocodynamometry during induced or augmented labour found no differences for any of the outcomes
**studied.** These included uterine rupture, hyperstimulation, Apgar score < 7 at 5 minutes, umbilical artery pH, admission to NICU, mode of delivery or instrumental deliveries, maternal infection, neonatal sepsis or morbidity, and hospital costs.92

**Indications for continuous EFM**

EFM is recommended for women at risk for adverse perinatal outcome. Pregnancy complications such as hypertension, placental abruption, fetal growth restriction, multiple pregnancy, prematurity (< 37^0 weeks), post-term (≥ 42^0 weeks), and chorioamnionitis have been associated with an increase in FHR abnormalities and the development of neonatal encephalopathy, CP, and perinatal death.90,91 Although there is insufficient evidence to indicate the conditions in which better outcomes are expected with EFM than IA, it seems reasonable to recommend the use of EFM in the following situations, as recommended by the Royal College of Obstetricians and Gynaecologists.40

Ambulation remains an important component of care during labour when EFM is being used. When possible, support maternal upright positions, ambulation and hydrotherapy such as with the patient standing beside the bed, squatting and kneeling. Where available, use wireless and waterproof technology that allows position changes, hydrotherapy and movement during labour and pushing.1

<table>
<thead>
<tr>
<th>Antenatal &amp; intrapartum conditions associated with an increased risk of adverse fetal outcome* where intrapartum electronic fetal surveillance may be beneficial</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Maternal</strong></td>
</tr>
<tr>
<td>• Hypertensive disorders of pregnancy</td>
</tr>
<tr>
<td>• Diabetes: Pre-existing and gestational</td>
</tr>
<tr>
<td>• Antepartum hemorrhage</td>
</tr>
<tr>
<td>• Maternal medical disease (cardiac, anemia, hyperthyroidism, vascular disease, renal disease, smoking)</td>
</tr>
<tr>
<td>• Motor vehicle collision / trauma (EFM recommended for a minimum period of 4–6 hrs)94,95</td>
</tr>
<tr>
<td>• Pre-pregnant BMI &gt; 35 kg/m² (see below)11</td>
</tr>
<tr>
<td>• Maternal perception of reduced or absent fetal movement96</td>
</tr>
<tr>
<td><strong>Antenatal</strong></td>
</tr>
<tr>
<td>• Intrauterine growth restriction</td>
</tr>
<tr>
<td>• Prematurity (&lt; 37^0 weeks)</td>
</tr>
<tr>
<td>• Oligohydramnios</td>
</tr>
<tr>
<td>• Polyhydramnios14</td>
</tr>
<tr>
<td>• Abnormal umbilical artery Doppler velocimetry</td>
</tr>
<tr>
<td>• Abnormal BBP or NST</td>
</tr>
<tr>
<td>• Isoimmunization</td>
</tr>
<tr>
<td>• Multiple pregnancy</td>
</tr>
<tr>
<td>• Breech presentation13</td>
</tr>
<tr>
<td>• Significant congenital anomaly (compatible with life)12</td>
</tr>
<tr>
<td>• Single umbilical artery24</td>
</tr>
<tr>
<td>• 3 or more nuchal loops19</td>
</tr>
<tr>
<td>• Velamentous cord insertion22,98</td>
</tr>
</tbody>
</table>
### Antenatal & intrapartum conditions associated with an increased risk of adverse fetal outcome* where intrapartum electronic fetal surveillance may be beneficial

<table>
<thead>
<tr>
<th>Intrapartum</th>
<th>Maternal</th>
<th>Fetal</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Vaginal bleeding in labour</td>
<td>Meconium staining of the amniotic fluid</td>
</tr>
<tr>
<td></td>
<td>Intrauterine infection / chorioamnionitis</td>
<td>Abnormal FHR on auscultation</td>
</tr>
<tr>
<td></td>
<td>Previous CS/Trial of labour after CS</td>
<td>Breech presentation</td>
</tr>
<tr>
<td></td>
<td>Prolonged rupture of membranes (&gt; 24 hours at term)</td>
<td>FHR dysrhythmia</td>
</tr>
<tr>
<td></td>
<td>Combined spinal-epidural analgesia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Oxytocin induction or augmentation</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hypertonic uterus</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Preterm labour (&lt; 37th weeks)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Post-term pregnancy (&gt; 42 weeks)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tachysystole</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Labour dystocia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Limitations in reliably determining UA and FHR with IA (e.g. maternal position or obese body habitus)</td>
<td></td>
</tr>
</tbody>
</table>


### BMI

Because of the adverse perinatal outcomes sometimes associated with maternal pre-pregnancy BMI >35, intrapartum EFM could be considered. Due to the limitations in reliably determining uterine activity and FHR with IA or external EFM in the presence of obese maternal body habitus, the use of a FSE and/or an IUPC should be considered.

### Epidural analgesia and intermittent auscultation

Epidural analgesia may cause maternal hypotension, which will decrease uteroplacental perfusion and result in intrapartum fetal heart rate abnormalities. These abnormalities usually occur in the first 30 to 60 minutes after initiation of the epidural. Although some authorities include epidural analgesia as an indication for continuous EFM, there is little research to suggest best practice. The SOGC Intrapartum Fetal Surveillance guideline states that intermittent auscultation may be used to monitor the fetus when epidural analgesia is used during labour, provided that a protocol is in place for frequent IA assessment (e.g., every 5 minutes for 30 minutes after epidural initiation and after bolus top-ups as long as maternal vital signs are normal).

Following epidural anaesthesia during labour, morbidly obese women (BMI ≥ 40 kg/m²) have more frequent hypotension and fetal heart rate abnormalities than normal weight women (BMI < 25 kg/m²). This supports the recommendation that these women receive continuous EFM.
PCEA is different from an intermittent bolus technique with concentrated local anaesthetic agents. It is used with dilute local anaesthetic and opioid solution (≤ 0.125% bupivacaine or equivalent). PCEA has been proven to be safe for ambulation in labour, and there is no evidence supporting the need for maternal vital signs to be taken after a self-administered bolus, as hypotension does not occur. Since maternal hemodynamics are stable with PCEA, there is no need to monitor the FHR after each self-administered PCEA top-up, which means that IA is acceptable and should be carried out according to usual obstetrical protocols, and that use of EFM should be based upon obstetrical considerations.

Systematic Interpretation of EFM Tracings

Principles:

A consistent and systematic analysis and interpretation of EFM tracings must be used by all care providers involved in a labouring woman’s care. Analysis refers to defining and measuring the characteristics of the tracing, and interpretation refers to the clinical meaning attributed to these measurements. Consistency is the key to achieving effective communication with other health care providers and patients, to accurate documentation, and to ensuring steps are not missed in the assessment process.

The definitions and explanations in this section are based in part on the recommendations from the 2008 NICHD Workshop on Electronic Fetal Monitoring. This NICHD workshop was convened to revisit nomenclature, interpretation, and research recommendations for EFM. An adequate tracing of the FHR and uterine contractions is required. EFM tracings are dependent on fetal gestational age and fetal and maternal physiologic status, which should therefore be factors in the assessment and evaluation of EFM. It is essential that the EFM tracing effectively demonstrates both the uterine activity and the FHR. FHR patterns are categorized as baseline, periodic, or episodic. Periodic patterns are those associated with uterine contractions, and episodic patterns are those not associated with uterine contractions. A full description of EFM requires an assessment of maternal risk factors and a qualitative and quantitative description of:

- Uterine activity characteristics (frequency, duration, intensity of contractions, and resting tone)
- Maternal heart rate (MHR)
- Baseline FHR
- Baseline FHR variability
- Presence of accelerations
- Presence of decelerations
- Changes or trends in FHR tracings over time
- Classification of the tracing (normal, atypical, or abnormal)
• Overall interpretation of the surveillance
• Response (communication and teamwork)

Electronic fetal surveillance should be reviewed and documented with the same frequency as intermittent auscultation.

The Process of Systematic Interpretation

1. Is the tracing interpretable?
   - Is the uterine activity reliably recorded?
   - Is there FHR artefact interfering with the accurate interpretation? Maternal heart rate (MHR) artefact has been found to be present in as many as 55% of EFM tracings. The MHR tracing can look quite similar to a normal FHR tracing, particularly in the active second stage when the mother may be tachycardic and displaying increases in heart rate with pushing. Consider simultaneous maternal (e.g., using the finger O2 saturation monitor) and fetal heart rate monitoring in labour particularly during the second stage.106 It is important to note that in the case of fetal demise a spiral electrode may detect the maternal HR.
   - Some external fetal monitors provide the option of monitoring the MHR and FHR (one or multiple) simultaneously. When the MHR or FHR(s) are similar, these monitors may emit "Coincidence Alarms" indicating that that supposed FHR may be maternal or the FHR from one fetus is actually derived from another. Providers need to be knowledgeable about the monitoring system and how to communicate and respond to these alarms, including: repositioning the transducers, alternative methods of MHR assessment (e.g., palpation, SpO2 monitor) and using FECG or direct visualization FHR with US.
   - Is there continuous recording, or are there spaces that make interpretation difficult or impossible?
   - Is the tracing of a sufficient time period to permit an accurate interpretation?
   - Does the quality of the uterine activity and FHR pattern allow for accurate interpretation?

2. What is the paper speed and graph range?
   - Paper speed should be standardized within each region and institution.
   - Appearance of FHR patterns is dramatically different with different paper speeds.
   - The 2019 SOGC guideline on intrapartum fetal surveillance recommends that in Canada institutions should standardize to a national paper speed of 3 cm/min\(^1\). For this reason, only 3 cm/min tracings are included.

3. What is the mode of monitoring (external or internal)?

4. What is the uterine activity pattern?
   - Contraction frequency (number present in 10 minutes averaged over 30 minutes)
   - Contraction duration (in seconds)
   - Contraction intensity (mild, moderate, or strong by palpation if using an external tocodynamometer, or in mmHg with an IUPC)
   - Uterine resting tone (soft or firm by palpation if using an external tocodynamometer, or in mmHg with an IUPC)
5. **What is the baseline FHR?**

- First, assess maternal heart rate to confirm the EFM detected HR is fetal and not maternal.
- Baseline FHR is the mean FHR rounded to increments of 5 bpm during a 10-minute segment of the tracing, excluding accelerations and decelerations and periods of marked FHR variability (segments of the baseline that differ by > 25 bpm). There must be 2 minutes of identifiable baseline (not necessarily contiguous) in any 10-minute window or the baseline is indeterminate. It may be necessary to assess a previous 10-minute period to determine baseline. The normal baseline rate is 110 bpm to 160 bpm. If the baseline FHR is less than 110 bpm, it is termed bradycardia. If the baseline FHR is greater than 160 bpm, it is termed tachycardia. The presence of either of these findings requires further assessment.
- Identify deviations from normal:

  **Tachycardia**

![Tachycardia graph]

  **Bradycardia**

![Bradycardia graph]
6. **What is the baseline variability?**

- Variability is a normal, physiologic characteristic of the FHR. Variability is largely controlled by the effect of the vagus nerve on the heart. Persistent hypoxia causing acidosis affects the autonomic nervous system early and results in a decrease in FHR variability.
- Variability refers to the fluctuations in the baseline FHR that are irregular in amplitude and frequency. It is determined in a 10-minute segment of baseline, excluding accelerations or decelerations. The difference between the lowest and highest rate is the range and/or amplitude of variability. The terms **absent, minimal, moderate, or marked** rather than “good” or “poor” are used to classify baseline variability. Because of the subjectivity of the visual evaluation of variability, careful re-evaluation is recommended in borderline assessments.

- Other conditions that can lead to decreased or absent variability include:
  - Fetal sleep (most common). Decreased variability associated with a fetal sleep state in a healthy term fetus is usually less than 40 minutes. It may extend to 90 minutes or more in some cases. Reduced variability longer than 40 minutes requires confirmation of fetal well-being.
  - Medications: narcotics, sedatives, β-blockers
    - magnesium sulphate infusion is associated with a transient decrease in variability during the bolus and a clinically insignificant decrease in FHR baseline (average 2.4 bpm) without any other significant change in FHR patterns. FHR variability returns to normal during maintenance infusion.
    - betamethasone and dexamethasone may affect variability and fetal movements for 3 days after administration and will return to normal. Studies report a decrease in fetal heart rate baseline during day 1 and then an increase during days 2 to 3, whereas variability is increased during day 1 and then decreased during days 2 to 3. All values return to baseline during day 4.
  - Preterm fetus: variability is usually moderate by 32 weeks’ gestation
  - Fetal tachycardia
  - Congenital anomalies
  - While smoking

- Moderate variability reliably predicts the absence of fetal metabolic acidemia at the time it is observed. Such variability can be measured only with EFM.

- **Marked variability** (sometimes referred to as a saltatory pattern) if present for >10 minutes is a characteristic of an abnormal FHR tracing

<table>
<thead>
<tr>
<th>CLASSIFICATION</th>
<th>RANGE OF AMPLITUDE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent</td>
<td>Undetectable</td>
</tr>
<tr>
<td>Minimal</td>
<td>≤ 5 bpm</td>
</tr>
<tr>
<td>Moderate</td>
<td>6 to 25 bpm</td>
</tr>
<tr>
<td>Marked</td>
<td>&gt;25 bpm</td>
</tr>
</tbody>
</table>
- Absent variability (amplitude range undetectable)

- Minimal variability (amplitude range ≤ 5 bpm)

- Moderate variability (amplitude range 6 to 25 bpm)
Marked variability (amplitude range > 25 bpm)

A sinusoidal pattern is a smooth, sine wave-like undulating pattern in the FHR baseline with a cycle frequency of 3 to 5 per minute that persists for ≥ 20 minutes. The amplitude of the oscillations is usually 5 to 15 bpm. This differs from and should not be confused with FHR variability. The sinusoidal pattern is abnormal and is most frequently associated with fetal anemia and/or hypoxia.
7. **Are there periodic (with contractions) or non-periodic (not associated with contractions) changes in the FHR?**

**Accelerations**

An acceleration is an **abrupt** increase (onset to peak in < 30 seconds) in FHR that is ≥ 15 bpm **above the baseline** for ≥ 15 seconds (10 bpm for 10 seconds for gestations < 32\(^0\) weeks) from the onset to the return to baseline. A prolonged acceleration is an increase ≥ 2 minutes. If the acceleration is sustained for ≥ 10 minutes it is considered a change in baseline rate. The presence of accelerations is a normal finding but not necessary during labour to define a tracing as being normal. Accelerations, in the presence of an abnormal EFM tracing, do not change the classification but may be considered as part of the total clinical situation.

**Decelerations**

A deceleration is a decrease in the FHR.

- Repetitive decelerations are defined as ≥ 3 in a row.\(^1\)
- Decelerations are defined as “recurrent” if they occur with ≥ 50% of uterine contractions in any 20-minute segment. Decelerations occurring with < 50% of uterine contractions in any 20-minute segment are defined as “intermittent”.\(^2\)
- Decelerations are distinguished on the basis of their waveform, being either “abrupt” or “gradual” onset:
  1. Abrupt (onset of the decrease to nadir <30s) = **Variable deceleration**
  2. Gradual, usually symmetrical, decrease & return (onset to nadir ≥ 30s)
     - **Early deceleration:** Onset, nadir & recovery **coincident** with onset, peak & end of contraction
     - **Late deceleration:** Onset, nadir & recovery **after** onset, peak & end of contraction
   - Note regarding gradual decelerations:
     - Visually apparent without a depth criteria
     - When not associated with an apparent contraction it is termed an **episodic gradual deceleration**. These are usually related to undetected contractions which indicates a reassessment of uterine activity.

**Early**

- A **gradual** decrease in the FHR (onset to nadir ≥ 30 seconds) and return to baseline associated with a contraction. Usually symmetrical, the nadir occurs at the same time as the peak of the contraction. In most cases, the onset, nadir, and recovery of the deceleration are **coincident with** the beginning, peak, and ending of the contraction, respectively.
• Secondary to fetal head compression
• Considered to be benign
• Not associated with fetal acidemia

Late

• a **gradual**, usually symmetrical, decrease and return to baseline FHR in association with a uterine contraction. The onset, nadir, and recovery of the deceleration occur *after* the beginning, peak, and end of the contraction. The onset to the nadir of the deceleration is ≥ 30 seconds. The nadir of the deceleration occurs after the peak of the contraction.
• late decelerations are associated with uteroplacental insufficiency and may imply a degree of hypoxia.\textsuperscript{114}
Variable

- an abrupt decrease in the FHR. The onset of the deceleration to the nadir is < 30 seconds. The FHR decreases to at least 15 bpm below the baseline and the deceleration lasts for ≥ 15 seconds but < 2 minutes.
- may be episodic (not associated with contractions) or periodic (associated with uterine contractions) (if periodic, their onset, depth, and duration commonly vary with successive contractions).
- the most common decelerations seen in labour
- uncomplicated variable deceleration
  - often have “shoulders”, i.e., an initial acceleration followed by a rapid deceleration to the nadir, a rapid return to the baseline and a secondary acceleration.\(^{115}\)
  - the physiology is thought to be a baroreceptor response to cord compression in labour. During a contraction there may be a sequential compression of the umbilical vein first (resulting an initial period of hypovolemia/hypotension inducing a baroreceptor triggered brief increase in FHR: "shoulder") followed by the compression of the umbilical arteries (resulting in an increase in fetal BP triggering a baroreceptor induced rapid decrease in HR) and finally as the contraction fades the artery is released but the vein remains compressed (resulting in brief hypovolemia and a secondary "shoulder") until the contraction ends and the FHR returns to the baseline.
  - not consistently associated with a poor neonatal outcome.\(^{40}\)

Complicated variable deceleration (may be indicative of fetal hypoxia)\(^{115,116}\)

- failure to return to baseline by end of contraction.
- deceleration lasting ≥ 60 seconds AND down to ≤ 60 bpm OR decrease by ≥ 60 bpm below baseline \(^{117,118}\)
- prolonged secondary acceleration (post-deceleration smooth overshoot) of 20 bpm and/or lasting > 20 seconds
- variable deceleration in the presence of:
  - absent or minimal baseline variability
  - fetal bradycardia or tachycardia

In a 2018 prospective study by Cahill et al, of 8580 women laboring at ≥ 37\(^{th}\) weeks gestation with a singleton cephalic fetus, the total deceleration area, especially in combination with fetal tachycardia was most predictive of acidemia. This emphasizes the significance of complicated variable decelerations that meet the criteria by their depth and duration.\(^{117}\)
1. Failure to return to baseline by end of contraction

Note: Importance of the timing of the periodic variable deceleration’s recovery

- It is very important to recognize when the recovery is delayed after the end of contraction. This delayed recovery can be abrupt or gradual. Physiologically this type of complicated variable is considered to have the same significance as a late deceleration and if repetitive define the tracing as abnormal.

2. Deceleration lasting ≥ 60 seconds AND down to ≤ 60 bpm or decrease by ≥ 60 bpm below baseline (“rule of 60’s”) \(^{118,119}\)
3. Overshoot (20 bpm increase for 20 seconds)

4. Absent or minimal baseline variability

5. Baseline tachycardia or bradycardia
**Decrease in FHR**

- **Abrupt onset (<30 sec)**
  - <15 bpm or <15 sec
  - **Variability**
  - Variable Deceleration
    - Failure to return to baseline by end of contraction
    - <70 bpm & > 60 sec
    - Loss of baseline variability
    - Biphasic
    - Overshoot
    - Tachycardia/bradycardia

- **Gradual onset (≥30 sec) and return (no depth criteria)**
  - ≥15 bpm & ≥15 sec
  - With contraction
  - After contraction
    - Early Deceleration
    - Late Deceleration

- **Uncomplicated Variable**
- **Complicated Variable**
Prolonged Deceleration

A deceleration of $\geq 15$ bpm below the baseline and lasting $> 2$ minutes but $< 10$ minutes from onset to return to baseline. (A deceleration lasting $> 10$ minutes is a change in baseline heart rate.)

In a 2013 retrospective cohort study of 5388 singleton, non-anomalous gestations that had reached full dilation, Cahill et al. addressed the significance of prolonged decelerations that precede and are unresolved before delivery (termed "terminal deceleration"). The last portion of EFM before delivery was analyzed ($\geq 10$ minutes and up to 30 minutes, if available). Overall, decelerations greater than 2 minutes (median duration was 3.3 min) occurred in 17.7% of the women but of these only 1.3% were found to have a pH of $\leq 7.10$. For every additional 120 seconds of duration beyond 2 minutes, a decrease in umbilical artery pH of 0.042 was noted. Decelerations of $\geq 10$ minutes (termed "terminal bradycardia") were associated with a 12.9% overall incidence of pH of $\leq 7.10$. \(^{119}\)

8. Classification, (Normal, Atypical or Abnormal tracing), Interpretation and Response

- Normal: Characteristics are within normal parameters.
- Atypical: Physiological response. Further vigilant assessment is required, especially when combined features are present. This may involve the correction of a reversible cause for compromise, intrauterine fetal resuscitation, and/or further fetal evaluation (scalp stimulation and/or scalp blood sampling if $> 34$ weeks, ultrasound)
- Abnormal: Possible fetal compromise. Action is required. The overall clinical situation should be reviewed, and intrauterine resuscitation and prompt operative delivery (vaginal or Caesarean section) is indicated unless delivery is imminent or there is evidence of normal oxygenation by scalp pH or lactate assessment.

SOGC Classification of intrapartum EFM tracings \(^1\)

*Note: It requires the presence of only one feature in the table under Atypical and Abnormal to classify the tracing as atypical or abnormal
<table>
<thead>
<tr>
<th></th>
<th>NORMAL TRACING</th>
<th>ATYPICAL TRACING</th>
<th>ABNORMAL TRACING</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Uterine activity</strong></td>
<td>• Normal contraction pattern</td>
<td>• Tachysystole may be present with normal, atypical or abnormal tracings; monitor closely for concerning FHR characteristics</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Baseline</strong></td>
<td>• 110–160 bpm</td>
<td>• 100–110 bpm</td>
<td>• &lt; 100 bpm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• &gt; 160 bpm for 30–80 min</td>
<td>• &gt; 160 bpm for &gt; 80 min</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Rising baseline</td>
<td>• Erratic baseline</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Arrhythmia (irregular rhythm)</td>
<td></td>
</tr>
<tr>
<td><strong>Variability</strong></td>
<td>• 6–25 bpm</td>
<td>• ≤ 5 bpm for 40–80 min</td>
<td>• ≤ 5 bpm for &gt; 80 min</td>
</tr>
<tr>
<td></td>
<td>• ≤5 bpm for &lt; 40 min</td>
<td></td>
<td>• ≥ 25 bpm for &gt; 10 min</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Sinusoidal</td>
</tr>
<tr>
<td><strong>Accelerations</strong></td>
<td>• Spontaneous accelerations present (but not required)[145]</td>
<td>• Absence of acceleration with scalp stimulation</td>
<td>• Usually absent (presence of accelerations does not change the tracing classification)</td>
</tr>
<tr>
<td></td>
<td>• Accelerations with fetal scalp stimulation</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Decelerations</strong></td>
<td>• None</td>
<td>• Repetitive (≥3) uncomplicated variable decelerations</td>
<td>• Repetitive (≥3) complicated variable decelerations</td>
</tr>
<tr>
<td></td>
<td>• Non-repetitive uncomplicated variable decelerations</td>
<td>• Non-repetitive complicated variable decelerations</td>
<td>• Recurrent late decelerations (with ≥ 50% of contractions)</td>
</tr>
<tr>
<td></td>
<td>• Early decelerations</td>
<td>• Intermittent late decelerations (occurring with &lt;50% of contractions)</td>
<td>• Single prolonged deceleration &gt; 3 min</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Single prolonged deceleration &gt; 2 min but &lt; 3 min</td>
<td></td>
</tr>
<tr>
<td><strong>Interpret Clinically</strong></td>
<td><strong>No evidence of fetal compromise</strong></td>
<td><strong>Physiologic response</strong></td>
<td><strong>Possible fetal compromise</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Determine the significance/cause of abnormal tracing:</td>
<td>• Determine the significance/cause of abnormal tracing:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Evaluate total clinical picture: stage of labor, gestational age, risk factors</td>
<td>• Evaluate total clinical picture: stage of labor, gestational age, risk factors</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Determine duration of effect and reserve tolerance of the fetus</td>
<td>• Determine duration of effect and reserve tolerance of the fetus</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Response</strong></td>
<td>EFM may be interrupted for periods of up to 30 min. if maternal-fetal condition stable and/or oxytocin infusion rate stable</td>
<td>VIGILENCE</td>
<td>ACTION REQUIRED</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Institute intrauterine resuscitation</td>
<td>• Institute intrauterine resuscitation and plan for delivery</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Communicate the tracing classification to the team and continue with ongoing fetal monitoring</td>
<td>• If clinically appropriate, obtain fetal scalp blood sampling (&gt;34 weeks, prolonged deceleration &lt; 3 min)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Perform fetal scalp stimulation and consider fetal blood sampling (&gt;34 weeks)</td>
<td>• Undertake transfer / operative delivery promptly UNLESS:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Consider transfer / delivery if tracing persists or deteriorates</td>
<td>• Tracing improves</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Fetal scalp sampling is normal</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Spontaneous delivery is imminent</td>
</tr>
</tbody>
</table>
Considerations:

- Does the classified tracing and its interpretation correlate with the clinical picture including the gestational age, pregnancy history, presence of risk factors, labour pattern, fetal behavioural state, or other extrinsic factors likely to influence the EFM tracing?
- Is further assessment and/or action necessary?
- The more atypical or abnormal features of EFM tracings that occur concurrently, the greater is the possibility of fetal compromise
- It is essential that all EFM tracings are considered in relation to previous EFM tracings. As each tracing is interpreted, an appropriate clinical action can be undertaken to lessen the impact on the fetus or to remove the effect entirely

**NOTE:** if an abnormal or atypical tracing improves with resuscitative maneuvers the response will return to that applied to the improved tracing. Ongoing clear distinction of MHR and FHR is essential.

### ELECTRONIC FHR, POTENTIAL CAUSES, ASSOCIATIONS, AND CLINICAL ACTIONS TO BE CONSIDERED

<table>
<thead>
<tr>
<th>PATTERN DEFINITION</th>
<th>ASSOCIATIONS OR POTENTIAL CAUSES</th>
<th>ADDITIONAL CLINICAL ACTIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Baseline:</strong> 110–160 bpm</td>
<td>• Normal physiologic response</td>
<td>No action necessary. EFM may be interrupted for periods up to 30 minutes if all other surveillance elements are normal, maternal-fetal condition is stable and, if oxytocin is being administered, the infusion rate is not increased.</td>
</tr>
</tbody>
</table>
| **Bradycardia:** 100–110 bpm | **Maternal:**  
  • Hypotension  
  • Drug response  
  • Maternal position  
  • Connective tissue diseases with congenital heart block (e.g., systemic lupus erythematosus).  
  **Fetal:**  
  • Umbilical cord occlusion  
  • Fetal hypoxia / acidosis  
  • Vagal stimulation such as with chronic head compression or with vertex presentation, occipital posterior or transverse position  
  • Fetal cardiac conduction or structural defect | 1. Measure maternal pulse and differentiate fetal from maternal heart rate  
2. Vaginal examination (elevate presenting part if umbilical cord prolapse)  
3. **Intraterine resuscitation** (see below)  
4. Discontinue oxytocin  
5. Decrease uterine activity (tocolysis) if indicated  
6. If cause is not obvious or correctable, consider intrapartum U/S to evaluate dysrhythmia  
7. If persistently <100 bpm, and/or associated with other borderline patterns of concern:  
  a. obtain scalp sample if clinically appropriate  
  b. expedite delivery |
| **Bradycardia:** <100 bpm | | |
### ELECTRONIC FHR, POTENTIAL CAUSES, ASSOCIATIONS, AND CLINICAL ACTIONS TO BE CONSIDERED

<table>
<thead>
<tr>
<th>PATTERN DEFINITION</th>
<th>ASSOCIATIONS OR POTENTIAL CAUSES</th>
<th>ADDITIONAL CLINICAL ACTIONS</th>
</tr>
</thead>
</table>
| **Tachycardia:** > 160 bpm for 30–80 minutes or rising baseline | Maternal:  
- Fever  
- Infection  
- Dehydration  
- Hyperthyroidism  
- Endogenous adrenaline or anxiety  
- Medication or drug response  
- Anemia | 1. Assess maternal temperature  
2. Decrease maternal temperature (if elevated)  
3. Review maternal medications  
4. Discontinue oxytocin  
5. Consider: the duration of rupture of membranes (ROM), positive vaginal culture, especially group B streptococcus (GBS)  
6. Intrauterine resuscitation  
7. If cause is not obvious or correctable, consider intrapartum U/S to evaluate arrhythmia  
8. If persistent (> 80 min):  
   a. obtain scalp sample if clinically appropriate  
   b. expedite delivery |
| **Tachycardia:** > 160 bpm for > 80 minutes or erratic baseline | Fetal:  
- Infection  
- Prolonged fetal activity or stimulation  
- Chronic hypoxemia  
- Cardiac abnormalities  
- Congenital anomalies  
- Anemia | 1. Assess maternal temperature  
2. Decrease maternal temperature (if elevated)  
3. Review maternal medications  
4. Discontinue oxytocin  
5. Consider: the duration of rupture of membranes (ROM), positive vaginal culture, especially group B streptococcus (GBS)  
6. Intrauterine resuscitation  
7. If cause is not obvious or correctable, consider intrapartum U/S to evaluate arrhythmia  
8. If persistent (> 80 min):  
   a. obtain scalp sample if clinically appropriate  
   b. expedite delivery |
| **Irregular FHR** |  
- Possible fetal arrhythmia | Initiate EFM and further investigations as indicated |
| **Moderate Variability:**  
6–25 bpm  
≤ 5 bpm for < 40 min. |  
- Interaction between the fetal sympathetic and parasympathetic nervous system (autonomic nervous system) | No action (normal response) |
| **Minimal variability:**  
≤ 5 bpm for 40–80 min |  
- Fetal sleep  
- Prematurity  
- Medication (analgesics, sedatives)  
- Hypoxic acidemia | 1. Review history for predisposing factors (prematurity, medications, etc.)  
2. Attach fetal scalp electrode if possible  
3. Obtain fetal scalp sample if clinically appropriate  
4. Prepare for delivery |
| **Absent (undetectable) variability:**  
undetectable or minimal (≤ 5 bpm) for >80 min |  
- Fetal sleep  
- Prematurity  
- Medication (analgesics, sedatives)  
- Hypoxic acidemia  
- Mild hypoxia  
- Fetal gasping  
- Unknown | 1. Attach fetal scalp electrode if possible  
2. Obtain fetal scalp sample if clinically appropriate  
3. Prepare for delivery |
| **Marked variability:** ≥ 25 bpm for >10 minutes |  
- Fetal sleep  
- Prematurity  
- Medication (analgesics, sedatives)  
- Hypoxic acidemia  
- Mild hypoxia  
- Fetal gasping  
- Unknown | 1. Attach fetal scalp electrode if possible  
2. Obtain fetal scalp sample if clinically appropriate  
3. Prepare for delivery |
### ELECTRONIC FHR, POTENTIAL CAUSES, ASSOCIATIONS, AND CLINICAL ACTIONS TO BE CONSIDERED

<table>
<thead>
<tr>
<th>PATTERN DEFINITION</th>
<th>ASSOCIATIONS OR POTENTIAL CAUSES</th>
<th>ADDITIONAL CLINICAL ACTIONS</th>
</tr>
</thead>
</table>
| **Sinusoidal pattern**                                  | • Severe fetal anemia (Hb <70)  
• Tissue hypoxia in fetal brain stem  
• May be transiently present with a healthy fetus  
Note: Pseudosinusoidal patterns may be similar but are usually transient and retain moderate variability and associated with good fetal outcomes. May be associated with medications including narcotic analgesia. | 1. Consider clinical picture  
2. Attach fetal scalp electrode if possible  
3. Consider fetal scalp stimulation  
4. Consider APT test or Kleihauer Betke; middle cerebral artery Doppler if available  
5. Prepare for delivery |
| **Accelerations:**                                       |                                                                                                 |                                                                                             |
| Spontaneous                                            | • Normal FHR response to increased fetal activity  
• Direct sympathetic stimulation of the fetus  
• Occlusion of umbilical vein only | No action (normal response)  
May be due to occlusion of umbilical vein only, as in association with variable decelerations (see text) |
| Periodic                                                |                                                                                                 |                                                                                             |
| In the presence of atypical or abnormal tracing absent acceleration with fetal scalp stimulation | • Hypoxic acidemia  
• Possible fetal abnormality | 1. Attach fetal scalp electrode if possible  
2. Obtain fetal scalp sample if clinically appropriate  
3. Prepare for delivery |
| **Early decelerations:**                                | • Associated with head compression  
• Not normally associated with fetal acidemia. | No action (normal response)                                                                 |
| Gradual decrease (onset to nadir ≥30 seconds) and return to baseline  
Onset, nadir, and recovery of deceleration coincident with the beginning, peak, and ending of contraction |                                                                                                 |                                                                                             |
| **Occasional (<3) uncomplicated variable decelerations:** | • Not usually associated with poor neonatal outcome | 1. No action necessary (normal response)  
2. Very common in labour  
3. Occurs in more than half of second stages |
| Initial acceleration  
Rapid deceleration to the nadir  
Rapid return to baseline  
Secondary acceleration |                                                                                                 |                                                                                             |
| **Repetitive (≥ 3) uncomplicated variable decelerations** | Due to cord compression | 1. Observe in early first stage of labour  
2. Watch for development of combined patterns or complicated variables |
## ELECTRONIC FHR, POTENTIAL CAUSES, ASSOCIATIONS, AND CLINICAL ACTIONS TO BE CONSIDERED

<table>
<thead>
<tr>
<th>PATTERN DEFINITION</th>
<th>ASSOCIATIONS OR POTENTIAL CAUSES</th>
<th>ADDITIONAL CLINICAL ACTIONS</th>
</tr>
</thead>
</table>
| **Repetitive (≥ 3) complicated variable decelerations:** | May be associated with fetal acidemia | 1. Intrauterine resuscitation (see below)  
2. Amnioinfusion may ameliorate  
3. Confirm fetal well-being, directly or indirectly (fetal scalp stimulation, and fetal scalp blood sampling if clinically appropriate  
4. Prepare for delivery |
| • Failure to return to baseline by end of contraction  
• Deceleration lasting ≥ 60 seconds AND down to ≤ 60 bpm or decrease by ≥ 60 bpm below baseline  
• Prolonged acceleration after deceleration (smooth overshoot) of >20 bpm and/or lasting > 20 sec  
• Loss of baseline variability  
• Presence of tachycardia or bradycardia | | |
| **Occasional late decelerations**  
**Single prolonged deceleration >2 min but <3 min** | • May be a response to uteroplacental function during labour (e.g., reduced uterine blood flow associated with maternal position)  
• Fetal chemoreceptor / vagal response  
• May be associated with transient fetal acidemia | 1. Mother in left lateral position  
2. Check maternal vital signs  
3. Continue to observe |
| **Late decelerations: >50% of contractions**  
A gradual decrease and return to baseline FHR in association with a uterine contraction. The onset, nadir and recovery of the deceleration occur after the beginning, peak, and end of the contraction. The onset to the nadir of the deceleration is usually > 30 seconds and the nadir is beyond the peak of the contraction. | • Fetal chemoreceptor / vagal response due to decreased pO₂  
• Altered maternal blood flow to the placenta (e.g., maternal hypotension)  
• Reduced maternal arterial oxygen saturation  
• Placental changes altering maternal-fetal gas exchange (e.g., placental insufficiency, uterine hypertonus or tachysystole)  
• May be associated with fetal acidemia | When persistent and repetitive, it is mandatory to act upon this pattern  
1. Intrauterine resuscitation  
2. Obtain fetal scalp sample if clinically appropriate.  
3. Prepare for delivery |
| **Single prolonged deceleration: >15 bpm for >3 min** | • Fetal baroreceptor and chemoreceptor response to profound changes due to:  
• uterine tachysystole  
• severe umbilical cord compression  
• maternal hypotension  
• maternal seizure  
• rapid fetal descent | 1. Vaginal examination to rule out cord prolapse  
2. Prepare for delivery |
### ELECTRONIC FHR, POTENTIAL CAUSES, ASSOCIATIONS, AND CLINICAL ACTIONS TO BE CONSIDERED

<table>
<thead>
<tr>
<th>PATTERN DEFINITION</th>
<th>ASSOCIATIONS OR POTENTIAL CAUSES</th>
<th>ADDITIONAL CLINICAL ACTIONS</th>
</tr>
</thead>
</table>
| Inadequate tracing for interpretation | | 1. Ensure that equipment is working properly  
2. If external monitor is in use, reposition to obtain a clear continuous signal  
3. Anticipate need for internal monitoring, if unable to maintain a technically adequate tracing despite interventions  
4. Confirm uterine activity pattern and uterine resting tone by abdominal palpation  
5. Assess and document the maternal heart rate concurrently with the fetal heart rate to differentiate maternal from fetal heart rate  
6. If using internal EFM, confirm the presence of fetal heart sounds by auscultation |

| EFM monitor HR Coincidence Alarms | In singleton gestation, suggest that the supposed “fetal” HR signal may be the MHR.  
In multiple gestation, may indicate the same or that the supposed FHR signal from one fetus is actually derived from another fetus. | 1. Reposition transducers  
2. Consider alternative methods of monitoring the MHR including digital palpation, SpO₂ monitor  
3. Consider fetal scalp electrode and/or bedside US to directly visualize FHR |

---

### Scalp stimulation

Digital fetal scalp stimulation provides an indirect assessment of acid-base status. It elicits a sympathetic nervous system response. The fetal scalp is stroked lightly for 15 seconds during a vaginal examination. Gentle digital scalp stimulation is recommended. Applying substantial pressure may produce a vagal response in the fetus and result in bradycardia. An FHR acceleration secondary to this stimulus suggests a normoxic fetus. Digital fetal scalp stimulation should not be used as a resuscitative intervention.

A 2018 Irish prospective study of 298 fetal scalp stimulations (FSS) followed by fetal blood sampling (FBS) done in response to abnormal EFM tracings found that FSS resulted in an acceleration or normal variability in over 80% of those with scalp pH ≥ 7.25, providing reassurance of fetal wellbeing. Reassuringly, all neonates classified as normal by FSS, but abnormal by FBS (pH ≤ 7.20) had normal Apgar scores and cord pH results. The study suggested that FSS has the potential to be a reliable and effective alternative to fetal blood sampling in the context of an abnormal EFM tracing.

- Digital scalp stimulation **should not be performed** during an FHR deceleration. Decelerations are secondary to a vagal response that prevents a sympathetic nerve response (acceleration) during scalp stimulation.
• An acceleration of 15 bpm amplitude with duration of 15 seconds has a very high negative predictive value and a very high sensitivity with regard to the absence of fetal acidosis.\textsuperscript{121, 122}
• An acceleratory response is associated with a scalp pH of > 7.20.
• Although an acceleratory response suggests fetal well-being, the absence of an acceleration does not necessarily predict fetal compromise.
• When acceleration does not occur in response to scalp stimulation, direct assessment with fetal scalp blood sampling should be considered.
• If fetal scalp blood sampling is not available or possible, consider prompt delivery depending on the overall clinical situation.

Intrauterine resuscitation\textsuperscript{1}

The goal of intrauterine resuscitation is to improve uterine blood flow, umbilical circulation, and fetal–maternal oxygen saturation.

NOTE: if an abnormal or atypical tracing improves with resuscitative maneuvers the response should return to that applied to the improved tracing.

When intrauterine resuscitation is undertaken

- Stop or decrease oxytocin\textsuperscript{126}/remove vaginal PGE2
- Change maternal position (to left or right lateral)
- Check maternal vital signs, including differentiation of MHR from FHR
- Modify or pause pushing efforts if in the second stage of labour
- Improve maternal hydration, with an intravenous fluid bolus, only if indicated eg. maternal hypovolemia
- Evaluate effects of maternal analgesia modality eg. epidural
- Perform a vaginal examination to rule out cord prolapse, relieve pressure from the presenting part on the cord and assess progress
- Consider tocolysis (e.g., with IV nitroglycerin) in the presence of tachysystole\textsuperscript{127}
- Provide supportive care to reduce maternal anxiety (this reduces catecholamine effects)
- Consider amnioinfusion if variable decelerations are present.
  - A 2012 Cochrane review of amnioinfusion for suspected umbilical cord compression in labour found that when fetal blood sampling was not used to confirm fetal compromise\textsuperscript{128} transcervical amnioinfusion was associated with the following reductions: Caesarean section overall; FHR decelerations; Apgar score < 7 at 5 minutes; meconium below the vocal cords; postpartum endometritis; maternal hospital stay > 3 days; mean cord umbilical artery (Ua) pH was higher with amnioinfusion group.
  - Perform transcervical amnioinfusion:
    › Instillation of fluid into the amniotic cavity through the cervix via an inserted tube, usually an IUPC with amnioinfusion capacity (occasionally a 12 to 14 FR pediatric nasogastric feeding tube or Foley catheter is used).
Purpose: used to augment the amniotic fluid volume to decrease the size and frequency of repetitive and/or complicated variable decelerations associated with low fluid.

Prerequisites: ruptured membranes

Contraindications: chorioamnionitis, low-lying placenta (on ultrasound near term)

Complications are rare but include fever and chorioamnionitis

Insertion technique: the IUPC is inserted (after membranes ruptured) into the amniotic cavity beside the fetal presenting part into the uterine cavity. Crystalloid (Ringer's lactate or normal saline) is then infused by infusion pump or gravity through the IUPC. There is no benefit in giving prophylactic antibiotics. There is no evidence that warming the fluid above room temperature is beneficial; however, if it is warmed, a blood warmer is recommended.

Infusion protocol: various options are used: e.g., bolus (50 to 1000 mL) followed by constant infusion; serial boluses (200 to 1000 mL) given every 20 minutes to 4 hours) or constant infusion. One documented protocol includes an initial infusion at 10 to 15 mL/minute continued until decelerations improve when the rate is reduced to 100 to 200 mL/hour.

Monitoring: EFM to assess fetal status; intraperitoneal pressure should be monitored to ensure satisfactory relaxation between contractions

A 2014 Cochrane review assessing amnioinfusion for meconium-stained liquor, in settings with standard peripartum surveillance, found it to be ineffective in reducing meconium aspiration syndrome, perinatal death or severe morbidity.

Consider administration of oxygen by mask when maternal hypoxia or hypovolemia is suspected or confirmed.

There is little evidence to evaluate its effectiveness when used in the management of suspected fetal compromise. Prophylactic maternal oxygen administration during the second stage of labour has been associated with abnormal cord blood gas levels at birth. A 2012 Cochrane review found that cord blood pH values < 7.2 were more frequent when women received prophylactic oxygen (RR 3.51; 95% CI 1.34 to 9.19).

in 2014, Hamel et al. reviewed the limited evidence available. When supplemental oxygen is given when suspected fetal hypoxia is not the result of maternal hypoxia:

- it may correct the fetal hypoxia but will not correct acidosis
- it can lead to decreased umbilical cord pH, increased need for neonatal resuscitation, and increased markers of free radical activity
- it will not reduce the CS rate in the presence of fetal compromise

Therefore, maternal O₂ supplementation should be reserved for the treatment of maternal hypoxia or hypovolemia and not used for management of atypical or abnormal fetal heart rate tracings.

Maternal Heart Rate (MHR)

Assess and document the maternal heart rate concurrently with the fetal heart rate to differentiate maternal from fetal heart rate:

- At initial assessment when confirming fetal life and determining baseline FHR
- At any time when there is uncertainty between the MHR and FHR.
c. Frequency of assessment and documentation will vary with the stage of labour:
   − In the active first stage and passive second stage of labour every 4 hours with intact membranes;
   − In the active first stage and passive second stage of labour every 2 hours with ruptured membranes;
   − In the active second stage of labour every 15-30 minutes

The incidences of MHR artifact can be a significant and frequent confounder when attempting to interpret the FHR. The MHR should be verified to differentiate it from the FHR; frequency of assessments will vary with the stage of labour and FHS. The MHR can be determined using palpation of the radial pulse, oxygen saturation probe or tocodynamometer. When using the tocodynamometer it is important to initially verify the maternal heart rate on the tracing by concurrent comparison with the maternal pulse. The use of simultaneous maternal and FHR monitoring for women undergoing EFM could be considered, especially during the second stage of labour when the work of labour increases the maternal heart rate. Providers need to be knowledgeable of the monitoring system in their institution, and institutions should have a clearly communicated process for responding to coincidence alarms and/or cross-channel verifications between maternal and fetal heart rates.

**General considerations and recommendations**

- When a normal tracing is identified, it may be appropriate to interrupt EFM for up to 30 minutes to facilitate periods of ambulation, bathing, or position change, provided that the maternal and fetal condition is stable, and, if oxytocin is being administered, the infusion rate is not increased.

- In the case of an atypical intrapartum EFM tracing, any action taken must consider the potential causes, the duration of the effect, and the reserve (tolerance) of the fetus. Any reversible cause of compromise should be identified and modified (correction of maternal hypotension, treatment of excessive uterine contractility). Further fetal evaluation by means of scalp stimulation is recommended, and fetal scalp blood testing (if > 34 weeks) may be considered. Other obstetrical parameters (e.g., gestational age, estimated fetal weight, presence or absence of meconium, and the phase and stage of the labour) will affect decision-making. Ongoing fetal evaluation is required, and delivery should be considered if the situation persists over time or if the tracing deteriorates.

- Significance of some EFM Tracing Patterns
  - A 2015 prospective study of 1070 women who had fetal scalp blood lactate analysis performed for EFM patterns that were not normal found:
    › isolated reduced variability (0 to 4) in most cases was not a sign of lactic acidemia
    › "severe" variable decelerations (defined as abrupt and lasting > 60 seconds) and late decelerations increased the likelihood of acidemia to the same extent
    › the combination of tachycardia and "severe" variable or late decelerations was associated with the highest rate of academia
    › fetal scalp lactate was normal in 97.5% of cases with a normal tracing.
Reducing Unnecessary Interventions as a Result of Fetal Surveillance

- The classification system of EFM tracings is intended to be as sensitive as possible to detect fetal acidemia without a significant false positive rate. A 2012 study reported that the SOGC classification system had an 88% sensitivity but only 37% specificity for detecting an umbilical arterial pH of ≤ 7.15. Although different pH cut-off points might have yielded different results, it is important to recognize that EFM interpretation is more sensitive than specific and to consider efforts that may reduce interventions based on false positive EFM interpretation.

Efforts to reduce intervention due to false positive EFM should include:

- Promotion of the use of IA in low-risk pregnancies because of the low incidence of true fetal compromise and the evidence of increased interventions with EFM for women with low-risk pregnancies.
- Not using “admission tracings” for women without risk factors for fetal compromise
- Assessment of the total clinical picture before making any decision to use EFM
- Fetal scalp blood sampling > 34 weeks’ gestation, if available and possible, to clarify any abnormal FHR tracing and reduce interventions
- Consideration of intrapartum fetal scalp stimulation. FHR acceleration in response to stimulation suggests the absence of fetal acidosis, although its absence does not predict fetal acidosi
- Consideration of maternal heart rate artefact as the cause
- Attention to all aspects of the EFM record, including baseline variability. Consideration of the presence of moderate FHR variability and/or the presence of accelerations to correlate with a high negative predictive value for severe metabolic acidosis and neonatal respiratory morbidity. Intrauterine resuscitation in all instances of atypical and abnormal tracings (including the reduction or discontinuation of oxytocin and modifying/pausing pushing in the second stage)
- Regular interprofessional quality assurance and educational fetal health surveillance programs
- Recognition that EFM tracing interpretation is subjective and consequently subject to individual interpretation

- Hindsight bias affects the EFM interpretation and management recommendations when neonatal outcome is known. Reif et al (2016) reported that 123 health care professionals interpreted 42 tracings without knowledge of fetal outcome and then again 2 months later with knowledge of the outcome: When the Ua pH was ≤ 7.0, normal classifications decreased by 76% and “pathologic” interpretations increased by 51%
- When the Ua pH was ≥ 7.20 the “pathologic” interpretations decreased by 40%
- In a 2015 study by Sabiani et al., 22 obstetricians classified the same 30 term intrapartum EFM tracings twice (3 months apart). Their agreement with their own previous interpretations was mediocre and the agreement with other specialists was low (kappa 0.11 to 0.18).

4. Documentation of Fetal Health Surveillance Assessments (IA and EFM)

It is essential to document the fetal surveillance throughout both the first and second stages of labour. Documentation should include the MHR, contraction pattern, FHR patterns, and the classification of IA (normal or abnormal) or the EFM tracing (normal, atypical or abnormal). Any clinical action taken on the basis of the classification should be documented, as should the maternal and fetal response to them.
• Accurate and timely documentation of all fetal health assessments and clinical actions taken is essential
• Standardization of documentation tools for both the first and second stages of labour
• Standardization of terminology and acronyms and abbreviations
• Standardization of EFM paper speed

Whatever documentation system is used, the following should be recorded:

**Uterine activity characteristics obtained by palpation or electronically:**
- Frequency
- Duration
- Intensity
- Relaxation between contractions

**MHR**

**FHR data:**
- Indication: if data derived from EFM
- Numerical baseline rate (in bpm)
- Rhythm: if auscultation (regular or irregular)
- Variability: if data derived from EFM
- Nature of changes from the baseline, i.e., acceleration or deceleration (type of deceleration if data derived from EFM)

**The classification, interpretation and response:**
- Normal or abnormal IA or normal, atypical, or abnormal EFM tracing
- Specific actions taken when changes in FHR occur
- Maternal and fetal responses to interventions
- Subsequent return to normal findings
- Other maternal observations and assessments

Fetal surveillance should be classified and documented:
- Active first and passive second stages of labour: every 15 to 30 minutes if IA and every 15 min. with EFM.
- Active second stage of labour: every 5 minutes if IA and every 15 min. if tracing and caregiver continuously present.

**Maintaining Standards in Fetal Surveillance**

Although there is no best evidence to indicate how often practitioners should update their fetal surveillance knowledge and skills, periodic review is required. Reviews should be interprofessional to ensure common terminology and shared understanding and to make it clear that fetal surveillance is a team responsibility.  

A 2011 systematic review evaluating cardiotocography (CTG or EFM) training programs found that these programs improved participant reaction, learning, behaviour change, and impact. There was evidence that knowledge was maintained for 6 months but clinical skills decreased over that time, suggesting that skills reinforcement training is important. Recognizing
that failure to act and delay in responding to EFM abnormalities are responsible for most cases of suboptimal care, this review also suggested that training programs include teamwork, communication, and emergency response.\textsuperscript{138}

A 2016 retrospective study evaluated the impact on term neonates of a national multidisciplinary fetal surveillance education program implemented in Australia and New Zealand in 2004. The outcomes included a significant improvement in intrapartum hypoxic death, Apgar scores < 5 and rates of hypoxic-ischemic encephalopathy without an increase in emergency CS in labour.\textsuperscript{139}

The SOGC Board passed the following recommendations in Dec. 2017:

To enhance perinatal safety in Canada, and to improve the quality of intrapartum care provided to labouring mothers:

1. all providers of intrapartum Obstetrical care commit to formal education in fetal surveillance in labour, and maintain up-to-date status with their education.
2. all providers of intrapartum Obstetrical care participate in regularly scheduled multidisciplinary Obstetrical skills drills at their hospital.
3. all hospitals that are engaged in Obstetrical care support intrapartum fetal surveillance education for their physicians, nurses, and midwives, and also assist in the development of regularly scheduled Obstetrical skills drills for all practitioners.

2018 Canadian Association of Perinatal and Women’s Health Nurses (CAPWHN) Board Position Statement and Recommendations on FHS education:

- All nurses providing pregnancy and intrapartum assessment using fetal health surveillance:
  - Participate in FHS education every 2 years.
  - Maintain documentation of their continuing education.
- FHS education programs should incorporate defined expected outcomes consisting of theory review/update and face-to-face interdisciplinary case-based education and discussion.
- All health care facilities providing antenatal and intrapartum fetal health surveillance should:
  - Provide opportunities for nurses to attend FHS education.
  - Fund opportunities to enhance FHS knowledge by way of workshops, paid work days, or conference attendance.
  - Assist in the development and implementation of regularly scheduled ‘obstetrical skills drills’ for all practitioners (nurses, midwives, physicians and support staff).

Therefore, the following is recommended:\textsuperscript{1}:

All providers of intrapartum obstetrical care (Physicians, Nurses, Midwives) should be required to commit to formal education in fetal health surveillance (FHS) and maintain up to date competence with formal education review every 2 years.

Each facility should provide opportunities for all intrapartum care providers (Physicians, Nurses, Midwives) to regularly attend an interdisciplinary educational discussion of FHS clinical situations, including both IA and EFM, to ensure common terminology, shared understanding and to foster the concept of team responsibility.
Other Technologies

Fetal pulse oximetry - Not recommended

This technology is an adjunct to EFM and is intended to continuously monitor intrapartum fetal O₂ saturation when an atypical or abnormal tracing is present. The cervix must be ≥ 2 cm dilated, the fetus must be in a vertex presentation, and membranes must be ruptured. Sensors may lie against the fetal cheek, temple, or along the fetal back, or attach to the fetal head by suction or clip.

A 2014 Cochrane review (7 trials, n = 8013)¹¹⁹ compared fetal pulse oximetry and EFM with EFM alone.

- No difference was found in CS (RR 0.99; CI 0.86 to 1.13), CS for dystocia, or in other maternal outcomes (chorioamnionitis, endometritis, uterine rupture, length of stay or maternal satisfaction) or fetal outcomes (Apgar scores, Uₐ pH, NICU admission, skin trauma, death)
- In 2 trials there was a decrease in operative birth (CS, forceps or vacuum) for atypical/abnormal fetal status but only one small study found any difference in overall operative delivery rates
- The authors concluded that the addition of fetal pulse oximetry does not reduce CS rates. Although there is limited support for its use when the fetal surveillance is not normal, its use was not recommended.¹⁴⁰

This review and others have failed to produce convincing evidence (improved neonatal outcome or reduced operative delivery rates) for fetal pulse oximetry to be recommended as an adjunct to EFM or as an independent fetal surveillance technique, and it is not recommended for routine use.¹

Fetal electrocardiogram: ST waveform analysis – Not recommended

- This technology is used in combination with standard EFM. Fetal electrocardiogram (ECG) requires a specialized monitor and proprietary software. It measures the FHR, fetal ECG, and the uterine activity.
- Physiologically, changes occur in the fetal QRS complex and T wave relative to the metabolic state of the fetal heart. Through analyzing the ST segment and the T/QRS ratio in conjunction with FHR patterns, it is proposed that decision-making about intervention can be more precise. Care providers need both initial and continuing training to achieve and maintain expertise in this technique.
- Currently this method uses a fetal scalp clip requiring adequate cervical dilation and ruptured membranes. A non-invasive abdominal fetal ECG monitor has been developed that uses cutaneous electrodes applied to the maternal abdomen to detect the fetal ECG. A 2013 study using this method has demonstrated that the maternal and the fetal heart rate can be simultaneously recorded, which could reduce the chance of misinterpreting the maternal heart rate as the FHR.¹⁴¹
- A 2015 Cochrane systematic review¹⁴² of 6 RCTs, involving 26 446 women, comparing the use of fetal ST waveform analysis to EFM alone (evidence graded moderate to high), demonstrated
  - No obvious difference in primary outcomes
    - Caesarean section deliveries
    - severe metabolic acidosis (Uₐ pH < 7.05 and BD > 12 mmol/L)
    - neonatal encephalopathy
However, there were fewer fetal scalp samples during labour (RR 0.61; 95% CI 0.41 to 0.91) and marginally fewer operative vaginal deliveries (RR 0.92; 95% CI 0.86 to 0.99).

- There was no statistically significant difference in low Apgar scores at 5 minutes, babies requiring neonatal intubation or admission to special care unit.

- The authors concluded that the modest benefit of fewer fetal scalp samplings and instrumental vaginal births have to be considered against the disadvantages of needing ruptured membranes and the use of an internal scalp electrode. They found little strong evidence that ST waveform analysis had an effect on the primary outcome measures.

Blix et al. and Saconne et al. published systematic reviews and meta-analyses of the 6 studies included in the 2015 Cochrane review by Neilson. Although Blix found a slight reduction in of neonatal metabolic acidosis (OR 0.64; 95% CI 0.46 to 0.88; NNT 401 to prevent 1 case of metabolic acidosis), this finding was not shared by Saconne et al. (0.5% vs 0.7%; RR 0.74; 95% CI 0.54 to 1.02). The authors of both studies concluded that the addition of ST analysis does not significantly improve perinatal outcomes.

The use of ST waveform analysis for the intrapartum assessment of the compromised fetus is not recommended for routine use.

**Intrapartum cardiotocography with computer analysis – Not recommended**

- A 2017 trial from the UK and Ireland randomized 47,062 women, with risk factors indicating EFM, to a computerized interpretation and decision support group or a no computerized-decision-support group. The authors found no difference in the incidence of poor neonatal outcome between the groups.

- A 2018 Systematic review with meta-analysis of 3 RCTs (54,492 participants) found that intrapartum fetal monitoring with computer analysis compared with visual analysis did not decrease the incidence of newborn metabolic acidosis or obstetric intervention.
References


60. Philip AGS, Saigal S. When should we clamp the umbilical cord? NeoReviews. 2004;5:e142.


63. Wiberg N, Kallen K, Olofsson P. Delayed umbilical cord clamping at birth has effects on arterial and venous blood gases and lactate concentrations. BJOG. 2008;115:697-703.


Available from: [http://fn.bmj.com/content/80/3/F246.full.pdf+html](http://fn.bmj.com/content/80/3/F246.full.pdf+html).


# Table of Contents

Chapter 9 Vaginal Birth ................................................................................................................................. 219  
  Introduction .................................................................................................................................................. 219  
  Assessment when birth is imminent ........................................................................................................... 219  
    Things to ask if possible ................................................................................................................................. 219  
    Physical Assessment .................................................................................................................................. 219  
    Consider ..................................................................................................................................................... 220  
    Signs of imminent birth include .................................................................................................................. 220  
  Delivery .......................................................................................................................................................... 220  
    1. Principles ................................................................................................................................................ 220  
    2. Position Mother ....................................................................................................................................... 220  
    3. Equipment .............................................................................................................................................. 221  
    4. Delivery Technique ................................................................................................................................. 221  
    5. Ongoing Care .......................................................................................................................................... 224  
    6. Communication and Documentation ..................................................................................................... 224
Chapter 9
Vaginal Birth

Introduction
The goal of this chapter is to review care of a patient having an imminent birth related to a rapid labour or presenting late in labour. This information would be useful to any care providers in labour and delivery units. Emergency rooms as well as paramedics. The care provided will depend on the location, the care providers available, and the status of the pregnancy and the fetus.

Individual facilities need to adapt the general principles discussed in this chapter according to their location, resources, and personnel available.

Assessment when birth is imminent
Obstetrical care providers should obtain baseline information by asking clear, concise questions during initial assessment or when moving to a safe location for birth.

Things to ask if possible
1. Do you have your pregnancy records with you?
2. When is your baby due? / How many weeks are you?
3. Have your membranes ruptured? If so, what colour is the fluid?
4. How many babies have you delivered and were they vaginal or Caesarean section deliveries?
5. Any problems in pregnancy—you or baby?
6. Are you taking any drugs—prescribed by your doctor or recreational or from the health food store?

Physical Assessment
1. Cervical dilatation and fetal position and presentation
2. Fetal Health Surveillance (Contraction pattern and fetal heart assessment by intermittent auscultation or electronic fetal monitoring)
3. Maternal vital signs
Consider

1. Establish IV access and blood work (CBC, X & Type due to PPH risk)
2. Ask additional questions as time permits
   a. Results of GBS swab?
   b. Has the fetus been moving?
   c. Who has been your primary care provider during pregnancy? If it is an obstetrician specializing in high-risk pregnancies, ensure risk status has been clarified.
3. Obtain antenatal records and ultrasound reports if not available from the patient.

Signs of imminent birth include

- Increased show
- Separation of the labia, bulging perineum and rectum
- Woman says “baby is coming”¹
- Uncontrollable urge to push/bear down
- Sensation of need to have a bowel movement
- Crowning of the presenting part
- Maternal passage of stool

Delivery

1. Principles

   The goal is to support a slow and gentle birth in a calm, supportive and safe environment.

   - Remain calm—take a deep breath
   - Identify yourself to the family
   - Use body fluid precautions; use of sterile equipment whenever time and situation allow²
   - If delivery is imminent, it is safer to deliver wherever the patient is rather than to attempt transportation to a birthing unit.
   - Transfer site if patient or team at risk
   - Try to provide privacy and warmth if in an unplanned location
   - Call for equipment and personnel. (e.g.: person skilled in delivery, additional nurses, staff with NRP).
   - Remain with the woman; establish rapport and cooperation, provide support and reassurance.

2. Position Mother

   Ideally, the parturient's position should allow good visibility of the perineum and access to permit monitoring of the fetal heart. It is best to deliver the baby onto a solid surface to increase safety for the baby.
3. Equipment

Open equipment for delivery and newborn resuscitation.

An emergency delivery kit should be available for use in non-birthing areas such as the emergency department. A kit stored in a hospital-grade backpack allows rapid response and staff can carry equipment safely with their hands-free.

If a kit is preset for transport, medications may be stored separately in a medications area near the backpack. Expiry dates of medications should be regularly reviewed.

Suggestions for a kit

<table>
<thead>
<tr>
<th>FOR DELIVERY</th>
<th>FOR THE INFANT</th>
<th>MEDICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Gloves, eye protection</td>
<td>• Cord clamp</td>
<td>• Oxytocin, syringe &amp; needle</td>
</tr>
<tr>
<td>• 4 Kelly clamps</td>
<td>• Blankets</td>
<td>• Misoprostol</td>
</tr>
<tr>
<td>• 1 pair scissors</td>
<td>• Cord blood syringes and tube or blood gas syringes</td>
<td></td>
</tr>
<tr>
<td>• Towels</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Kidney basin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Sponges</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Documentation forms</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

4. Delivery Technique

Principles

At the time of delivery, the parturient should be encouraged to adopt whatever positions are comfortable. Encourage a position that allows visibility and manoeuvres in the event of a concern (e.g., shoulder dystocia).

The obstetrical care provider supporting the birth should

- NEVER take his or her eyes off the perineum once delivery is imminent
- Await restitution of the shoulders and external rotation of the fetal head
- Obtain arterial and venous umbilical cord gases or clamp segment of the cord (pH maintained up to one hour at room temp)
- Obtain cord blood if the parturient is Rh negative (or if her Rh status is unknown)

Delivery of head

- The woman should be encouraged to push only when she has the urge to do so and to refrain from prolonged breath-holding (Valsalva) pushing.¹
- Use one hand to protect the perineum with gentle pressure using a sponge or cloth. The other hand may be used to prevent rapid delivery of the head with fingers or hand “lightly placed on the advancing head to monitor descent and prevent very rapid crowning and extension”.²
• Good visualization of the perineum and manual perineal protection has been shown to reduce the risk of anal sphincter tear.\textsuperscript{5,6}

• Studies have compared a “hands-on” delivery technique (hands are used to put pressure on the baby’s head in the belief that flexion will be increased, and to support (“guard”) the perineum, and to use lateral flexion to facilitate the delivery of the shoulders) with a “hands poised” technique (hands poised, prepared to put light pressure on the baby’s head in case of rapid expulsion but not to touch the head or perineum otherwise and to allow spontaneous delivery of the shoulders).\textsuperscript{7} It appears that both hands-on and hands poised techniques are reasonable and that the decision to use one or the other rests with the individual care provider.\textsuperscript{2} This recommendation is also supported in the 2014 NICE Guideline on Intrapartum Care.\textsuperscript{10}

• There is evidence that warm, moist packs applied to the perineum in the late second stage may relieve perineal pain and increase comfort.\textsuperscript{11} A 2011 Cochrane review (\(N = 1525\)) of warm perineal compresses in labour versus no intervention showed a reduction in third and fourth-degree perineal tears from 5\% to 2.5\% (absolute risk reduction 2.5\%; number needed to treat = 40 to prevent one anal sphincter injury); however, no significant difference was found for the outcome of intact perineum.\textsuperscript{12}

• Use a sponge, towel, or bedsheet to cover the anus to prevent contamination of the clean field by maternal feces.

• The parturient should be coached to pant (not push) with crowning.

• The head should, if possible, be delivered between or at the end of a contraction.

Check for nuchal cord

• \textbf{DO NOT CUT THE CORD until the shoulders are delivered}

• If a nuchal cord is found, consider the following:
  – attempting to slide the cord gently over the infant’s head if the cord is very loose
  – pushing the cord back over the shoulder, allowing the shoulder to slip under it as the infant delivers
  \hspace{1cm} keeping the newborn close to the perineum and having the rest of the body deliver or “somersault” out.\textsuperscript{13} This somersault keeps head and torso close to the perineum preventing traction of the cord

Shoulders

• The parturient should be assisted to pant while awaiting restitution, external rotation of the head, and WAIT until the next contraction to deliver the shoulders.

• In normal birth there is usually a pause between delivery of the head and body. DO NOT RUSH. During this pause, the uterus relaxes and the fetus restitutes. The drop in the cord pH during this pause is 0.011 per minute.\textsuperscript{13} As long as the woman is not pushing and the uterus is relaxed, venous return from the fetal head to thorax is maintained and therefore so is perfusion of the fetal brain. If fetal heart rate is normal before delivery, the pause does not alter fetal acidosis and may assist with maternal expulsive efforts.\textsuperscript{14-16}

• If the head retracts very close to the perineum after delivery of the head, hyperflex the parturient’s lower limbs at the hips and lower her head (McRobert’s manoeuvre) while awaiting the next contraction.

• Care providers should NOT PULL ON THE FETAL HEAD or apply downward lateral pressure.

• If the anterior shoulder does not deliver with a contraction, maternal effort, and gentle traction, this indicates shoulder dystocia. Call for help and begin a planned approach to shoulder dystocia (see Shoulder Dystocia chapter).
Infant

- The infant should be guided in the direction of an upward motion to complete the delivery
- Placing the infant skin-to-skin on the mother’s abdomen to reduce temperate loss (or a safe surface if the mother’s abdomen is not an option).
- Dry and cover with warm blankets.
- Verify that there is no twin.

Cord

- Delay cord clamping by at least 60 seconds (and up to 120 seconds) in newborns not requiring resuscitation. This delay has been shown to benefit preterm\textsuperscript{14, 15} and term\textsuperscript{16-19} newborns. Apply the first clamp 3 cm to 5 cm from the newborn.
- Apply a second clamp at least 10 cm away from the first clamp to allow newborn venous and arterial samples to be collected. It may be helpful to milk the cord (from the placenta toward the baby) to fill vessels to allow easier collection of cord gases.
- Apply a third clamp near the first clamp, and cut the cord.
- Collect cord blood for gases and Rh status.

Placenta

- Signs of placenta separation include gush of blood, cord lengthening, uterine fundus rising up in the abdomen, and uterus becoming firmer.
- Active management of the third stage, including
  - Administration of prophylactic oxytocin (10 IU IM) after delivery of the anterior shoulder is indicated to prevent postpartum hemorrhage\textsuperscript{18, 20}. This may be administered by a nurse when an MD or MW is not present if a medical directive or standing order is in place. The woman/parturient should be informed of the medication that is being administered and why.
  - When oxytocin is not available, misoprostol 400 mcg may be given sublingually after the birth of the baby. The parturient should be informed that fever and shivering are possible side effects\textsuperscript{21}.
  - If medications are not available / declined, encourage breastfeeding.
  - A skilled care provider may use controlled cord traction to assist in delivery of the placenta. Gentle traction is applied in the axis of the pelvis (45 degrees from the horizontal in a supine parturient) during a uterine contraction to dislodge the placenta from the uterine cavity to the vagina. Use external counter-traction (one hand supporting the uterus just above the pubic bone); this reduces the duration of the third stage. Care must be taken that excessive traction does not cause tearing of the umbilical cord or placenta.
  - In situations where there are no skilled care providers present, controlled cord traction is not recommended. Wait for signs of placental separation (cord lengthening, uterus firm and globular on palpation at the umbilicus) after which encourage the woman to bear down with contractions until there is spontaneous placental delivery.
  - Ensure that all the membranes are delivered using gentle traction and ring forceps, if needed.
Assess the fundus and ensure that it is well contracted and that there is no significant bleeding; uterine massage can be performed after delivery of the placenta, as needed.

Inspect the placenta for completeness

Keep the placenta until verified by an experienced provider

Precipitous delivery is associated with an increased risk of postpartum hemorrhage.

- Uterine atony: Prolonged or very rapid labour patterns are associated with less effective clamping down of the uterine muscle postpartum. If bleeding is excessive, management would include additional uterotonics and uterine massage. Uncontrollable bleeding is best controlled by bimanual compression of the uterus until help arrives. IM or IV anesthesia would be nice.
- Cervical, vaginal, or perineal tears: rapid delivery of the baby may contribute to increased risk of such tears. Explore and identify perineal or other trauma and observe/manage appropriately.

Newborn care

- Place the baby skin-to-skin on the mother
- Perform the initial steps of neonatal resuscitation, if required, and continue as indicated.
- Record Apgar scores at 1 and 5 minutes.
- Perform assessment of the newborn for injuries (e.g., fractures of the clavicle and/or humerus). A rapid delivery of the baby may also increase likelihood of newborn trauma.

5. Ongoing Care

- Monitor postpartum vital signs, bleeding, fundal height and tone, perineum, and urinary output.
- If the family has no prenatal records and/or there has been no prenatal care, it may be helpful to involve your facility social worker to determine if support is needed or there are child protection issues.
- Consult with other support services (e.g., chaplain) as needed for the family.
- Explore with women and their support persons factors such as parity, distance to hospital and access to care that might have contributed to her presenting when birth was imminent or to her having given birth unexpectedly.
- If the parturient does not have a family physician, determine a strategy for post-hospital newborn care.

6. Communication and Documentation

- Complete the delivery summary.
- A narrative summary may be required for additional details such as place of birth, arrival of health care personnel, delivery circumstances, newborn resuscitation.
- Determine if any patient safety or learning issues warrant a quality care review.
- Ensure there is notification of the primary care provider.
References


Table of Contents

Chapter 10 Assisted Vaginal Birth ................................................................................................................................... 228
  Introduction ........................................................................................................................................................................... 228
    Definitions ........................................................................................................................................................................ 228
    Classifications ................................................................................................................................................................. 228
    Incidence ........................................................................................................................................................................... 229
  Techniques .......................................................................................................................................................................... 231
    Vacuum ............................................................................................................................................................................. 233
    Forceps ............................................................................................................................................................................ 235
    Comparison of Vacuum and Forceps for AVB .................................................................................................................. 236
    Caution: sequential use of vacuum and forceps .............................................................................................................. 238
  Management ...................................................................................................................................................................... 240
    Vacuum Extraction .......................................................................................................................................................... 240
    Forceps Application ........................................................................................................................................................ 242
    Techniques for Use of Vacuum and Forceps .................................................................................................................. 243
  Follow-Up .......................................................................................................................................................................... 249
    Documentation ................................................................................................................................................................. 249
    Suggested format for a chart note .................................................................................................................................. 250
  Summary .......................................................................................................................................................................... 250
Chapter 10
Assisted Vaginal Birth

Introduction

Assisted vaginal birth (AVB), or operative vaginal birth, refers to the use of vacuum or forceps to achieve a vaginal delivery in the second stage of labour. Both methods are safe and reliable for assisting childbirth provided that the operator has adequate training and experience and appropriate attention is paid to the indications and contraindications. In every case, consideration must be given to the maternal and fetal risks associated with using either instrument and to the risks of the alternative choice of Caesarean section (CS). The procedure should be undertaken only when there is a reasonable chance of success and a backup plan is in place.

The choice of instrument should suit both the clinical circumstances and the preference of the health care provider and parturient. It is essential that the parturient provide informed consent.

Definitions

Station: The level of the leading edge of the fetal skull is expressed as the number of centimeters around a reference line of zero drawn at the ischial spines. When the skull is above the spines, it is represented as a negative number, reaching a value of -5 at the pelvic inlet. When below it is expressed as a positive number reaching +5 at the perineum. Careful distinction between skull and caput is important.

Engagement: When the biparietal diameter of the head enters the plane of the pelvic inlet. Engagement is usually complete when the leading edge of the skull is at or below the ischial spines (station 0); however, extensive moulding and caput can bring the leading edge of the scalp and skull to the level of the spines before the biparietal diameter is engaged in the pelvic inlet.

Caput: Subcutaneous fetal scalp swelling secondary to pressure of labour.

Chignon: Exaggerated caput secondary to suction force from a vacuum.

Classifications

The classification of operative vaginal deliveries is based on the station of the head within the pelvis and the degree of rotation from an occiput anterior (OA) position.

Outlet
- Scalp is visible at a point between the introitus without separating the labia and the perineum
- Fetal skull has reached the pelvic floor
- The sagittal suture is in anterior-posterior diameter, right, or left OA or occiput posterior (OP) position however rotation does not exceed 45°.
Low
- Leading point of fetal skull is at station ≥ +2 and above the pelvic floor
- Two sub-divisions: rotation is £ 45°; rotation is > 45°

Mid
- Head is engaged and leading point of skull is between station 0 and +2

Incidence

Rates of AVB, particularly forceps use continues to decline nationally and internationally. Reasons postulated for the decline are the perceptions that Caesarean birth is a safer alternative and that vacuum-assisted delivery is easier. Other reasons cited are the decline in opportunities for residents to learn to perform forceps-assisted delivery and potential medical legal implications.

In the United States from 1990 to 2010, the overall AVB rate declined from 11.7% to 5.5%. The rate of forceps deliveries declined steeply from 6.5% to 1% and the rate of vacuum deliveries has remained roughly the same, around 5%. Despite the overall recent trend downward, Portugal recently experienced an increase in both vacuum- and forceps-assisted births as part of a successful national effort to reduce the overall Caesarean section rate.

Figure 1. Use of Vacuum and Forceps in Vaginal Deliveries: United States, 1990–2010
In Canada, the decline in AVB rates has been more modest. In 2010–2011, the rates of all AVB, vacuum-assisted deliveries, and forceps-assisted deliveries in Canada were 13.5%, 9.6%, and 3.2% respectively. There is considerable provincial variation within Canada (Table 1) as well as variation between selected western countries (Figure 2). The rate of AVB is higher in nulliparous than in parous women.

Table 1. Assisted Deliveries in Canada

<table>
<thead>
<tr>
<th>Province</th>
<th>VACUUM, %</th>
<th>FORCEPS, %</th>
<th>TOTAL AVB, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>British Columbia</td>
<td>10</td>
<td>4</td>
<td>14</td>
</tr>
<tr>
<td>Alberta</td>
<td>12.5</td>
<td>4</td>
<td>16.5</td>
</tr>
<tr>
<td>Saskatchewan</td>
<td>13</td>
<td>3</td>
<td>16</td>
</tr>
<tr>
<td>Manitoba</td>
<td>6.5</td>
<td>3.2</td>
<td>9.7</td>
</tr>
<tr>
<td>Ontario</td>
<td>10</td>
<td>3.8</td>
<td>13.8</td>
</tr>
<tr>
<td>Quebec</td>
<td>9</td>
<td>3</td>
<td>12</td>
</tr>
<tr>
<td>New Brunswick</td>
<td>9</td>
<td>3.8</td>
<td>12.8</td>
</tr>
<tr>
<td>Nova Scotia</td>
<td>8</td>
<td>4.5</td>
<td>12.5</td>
</tr>
<tr>
<td>Prince Edward Island</td>
<td>4</td>
<td>2.5</td>
<td>6.5</td>
</tr>
<tr>
<td>Newfoundland &amp; Labrador</td>
<td>10</td>
<td>6</td>
<td>16</td>
</tr>
<tr>
<td>Yukon</td>
<td>7</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td>Northwest Territories</td>
<td>5.5</td>
<td>1</td>
<td>6.5</td>
</tr>
<tr>
<td>Nunavut</td>
<td>1.5</td>
<td>0</td>
<td>1.5</td>
</tr>
</tbody>
</table>

Adapted from Highlights of 2008–2009 selected indicators describing the birthing process in Canada [Bulletin]. Ottawa: Canadian Institute of Health Information; 2009 Oct.

Figure 2. Assisted Deliveries by Country
Techniques

Careful clinical assessment is a prerequisite to AVB. Pelvic adequacy, fetal station, fetal position, caput and molding should be assessed. Abnormalities of the position or attitude of the vertex can result in relative cephalopelvic disproportion (CPD). The fetal station should be assessed abdominally in fifths above the pelvic brim as well as by pelvic examination (Figure 3). Extensive moulding or caput may confuse the vaginal assessment of descent and may indicate the possibility of relative CPD. If more than one fifth of the fetal head is palpable above the pelvic inlet, AVB is inadvisable. A 2013 prospective trial study demonstrated that a strategy of manual rotation for fetuses in posterior or transverse positions at full dilatation is associated with a reduction in the rate of operative delivery.13

Figure 3. Abdominal Examination of Fifths Above the Pelvic Brim

Used with permission of Salus Global Corporation14
Manual rotation

Successful rotation after the onset of the second stage of labour is more likely to be successful if it is performed before arrest occurs. Manual rotation can convert 90% of OP or transverse arrest situations to OA.

Manual rotation is more successful in multiparous women and young women.

Rotation is important if there is a need for a fast delivery and/or if there is minimal or slow descent after a trial of pushing.

First, empty the bladder.

There are two methods for rotating the fetus.

1. A hand is inserted into the vagina with the palm upward. Digital rotation is performed by placing the tips of the index and middle fingers in the anterior segment of the lambdoid suture near the posterior fontanelle (see Figure 2.6.E.4).

The fingers are used to flex and slightly dislodge the vertex, rotating the fetal head to the OA position by rotation of the operator’s hand and forearm. The thumb may also be used with gentle downward pressure more anteriorly on the parietal bone to aid this rotation. The fetal head should be held in place for a few contractions to prevent rotation back towards the posterior position.

2. The operator’s four fingers are placed behind the posterior parietal bone with the palm up and the thumb over the anterior parietal bone. The right hand is used for the left OP position, and the left hand is used for the right OP position. The head is grasped with the tips of the fingers and thumb. During a contraction, the patient is encouraged to push and the operator attempts to flex and rotate the fetal head anteriorly. Upward pressure may help to slightly elevate the head and facilitate rotation (see Figure 2.6.E.5).

As part of informed consent, patients should be informed of the potential risks and benefits of the selected instrument (vacuum or forceps) before application.

The risk of perineal trauma and the small risk to the with vacuum or forceps must be weighed against the maternal risks of Caesarean section (at advanced station). If the fetal heart rate is abnormal, the risk to the fetus of any delay required to deliver by Caesarean section must also be taken into consideration.
Vacuum

Vacuum extractor use in modern obstetrics began with metal mushroom-shaped cups designed by Malmstrom and Bird. These inspired rigid plastic cups. In an effort to reduce scalp trauma, soft cups of silicone and rubber were later developed.

Soft cups are less likely to cause cephalohematoma and scalp injury than forceps, but are more likely to lead to an unsuccessful vaginal delivery. There are no differences in neonatal or maternal outcomes between the two methods.\textsuperscript{15}
Maternal risks can be minimized by ensuring both the non-traumatic insertion of the device and avoiding soft tissue entrapment as well as controlling the rate of descent and delivery over the perineum.

Application of the vacuum device to the fetal scalp invariably causes exaggeration of subcutaneous caput and formation of a chignon, which resolves within hours of birth. Bruising is also common and is benign. Large caput and bruising can easily be confused with a cephalohematoma, which is a deeper collection of blood between a skull bone and periosteum that occurs in approximately 5% of vacuum deliveries. Because the periosteum is tightly adherent to each individual cranial bone the swelling does not cross suture lines, is self-limiting and forms a tense swelling immediately adjacent to bone that persists after the caput and chignon have subsided. The swelling does not cross sutures. It is benign and resolves over weeks to months; however, resorption of hemoglobin can result in modest hyperbilirubinemia. Swelling that resolves within hours or days is not a cephalohematoma.

A more serious but rare complication of vacuum delivery is subgaleal (subaponeurotic) hemorrhage (SGH) or bleeding into the potential space between the skull periosteum and the scalp aponeurosis. With SGH, the suture lines of the skull do not limit hemorrhage into the subgaleal space as they do in cephalohematoma. As a result, subgaleal hemorrhages can extend over the entire calvaria from the brow to the nuchal ridge and from ear to ear. The volume of blood that accumulates can be sufficient to cause severe or fatal hypovolemic shock. However, early detection and prompt transfusion of blood and plasma will usually be life saving. In modern obstetrical practice, subgaleal hemorrhage is rare, reported in 1/1000 rigid Kiwi OmniCup deliveries.

Figure 4. Subgaleal Hemorrhage Versus Cephalhematoma

Successful vacuum delivery with minimal fetal scalp trauma can best be achieved by minimizing traction forces and likelihood of pop-off through correct application of the cup over the flexion point of the fetal scalp and avoiding excessive, incorrect, or prolonged (> 10 minutes) traction. Fetal scalp trauma (hemorrhage and laceration) can best be prevented by avoiding excessive, incorrect, or prolonged (> 10 minutes) traction, and by not applying any rotational force to the vacuum cup. To minimize traction forces and likelihood of pop-off, the vacuum cup must be applied to the...
flexion point. Vacuum traction should be coordinated to the maternal expulsive effort, and the angle of traction must follow the pelvic curve. The vacuum should not be used to apply rotational force, which can cause scalp trauma. Vacuum should be avoided if fetal coagulopathy is known or suspected.¹⁸ ²²

After every successful or attempted vacuum delivery, the condition of the neonate should be monitored closely for SGH. Enlargement of the chignon, increasing head circumference, hypovolemia, or tachycardia should prompt careful pediatric assessment for SGH. ²³ ²⁴ Early detection and treatment can be lifesaving. A suggested surveillance protocol is found later in the chapter.

Forceps

Forceps vary by their shank, pelvic curve and cephalic curve, characteristics that may be best suited to the clinical situation. Simpson forceps are commonly used for delivery of a moulded fetal head, often seen in nulliparous women. Tucker-McLane forceps have a more rounded cephalic curve, suitable for the unmoulded fetal head seen in multiparous women. Kjelland forceps are designed for rotation of the fetal head and lack a pelvic curve.

The risk of maternal soft tissue injury is greater with forceps than with vacuum.¹ A 2013 study from the United Kingdom demonstrated an adjusted odds ratio of obstetrical anal sphincter injury (OASI) with forceps delivery of 4.43, P < 0.005. ²⁶ Hehir et al. also demonstrated increased risk of OASI with forceps with a rate of 8.6% compared to a spontaneous vaginal delivery rate of 1.3%. ²⁷ An education program in Norway that included good communication with the woman, adequate perineal support, visualization of the perineum during delivery, and mediolateral episiotomy for most forceps-assisted deliveries reduced the forceps-associated OASI rate by half. The more liberal but not routine use of episiotomy was felt to be responsible in part for the reduction in OASI.²⁸

The fetal risks with forceps delivery include the risk of facial laceration and cephalohematoma of 1% each. The risk of symptomatic intracranial hemorrhage is 0.1%. ¹ External ocular injuries and facial nerve palsies are more common with forceps than with vacuum. ²⁵ A 2009 Canadian-authored study supports the view that facial nerve palsies associated with operative vaginal delivery have a very good prognosis. ²⁶

Neonatal skull fracture has been reported after both spontaneous and forceps deliveries. The incidence is greater after forceps deliveries.²² Forceps should generally not be used if fetal coagulopathy is known or suspected.

There remains a role for mid-forceps deliveries. The risk of a mid-forceps delivery must be compared with that of its alternatives: vacuum-assisted birth or intrapartum second stage CS. Mid-cavity and rotational forceps delivery should be performed only by experienced operators and should be generally conducted as a “double set-up” in an operating room with immediate ability to perform Caesarean section in case vaginal delivery is not accomplished.

Rotational forceps deliveries have been the subject of several studies in the United Kingdom. Bradley et al. showed there was no difference in trauma arising as a result of manual or forceps rotation and that there was less trauma if the baby was delivered in the occiput anterior instead of occiput posterior position.²⁸
Comparison of Vacuum and Forceps for AVB

The choice of vacuum or forceps for AVB should be based on the total clinical picture including the expertise of the practitioner and the availability of CS. A 2010 Cochrane meta-analysis of 32 studies showed forceps and the metal vacuum cup (versus soft cup) were most successful in completing a vaginal birth; however, use of forceps was associated with higher rates of maternal complications, including perineal trauma, tears, more use of general and regional anaesthesia, and more incontinence. The metal cup was associated with more neonatal scalp trauma. The urgency for delivery needs to be balanced against potential risks to the mother and baby. The following graphs from the meta-analysis of controlled trials compare forceps delivery and vacuum extraction.

Maternal trauma from AVB is primarily from pelvic floor injury. Arguably, the best long-term data on symptomatic pelvic floor injury from vacuum delivery is found in a 20-year follow-up of 4000 primiparous Swedish women who underwent either one spontaneous vaginal birth, one vacuum assisted vaginal birth, or one caesarean section. Overall, the incidence of urinary incontinence, fecal incontinence, and pelvic organ prolapse was almost identical 20 years after spontaneous or vacuum-assisted birth; however, the rate of OASI was almost three times greater after vacuum delivery (6.3%) than after spontaneous delivery (2.4%). Women who sustained OASI had almost double the incidence of urinary incontinence (60% vs 40%) and fecal incontinence (30% vs 15%) compared with women without OASI, whether they had a vacuum delivery or not. The incidence of pelvic organ prolapse did not differ. It appears that the risk of urinary and fecal incontinence is associated with OASI rather than vacuum delivery itself. The incidence of OASI and severe perineal trauma is at least twice as high with forceps compared with vacuum. A small randomized trial indicated higher risk of fecal incontinence after forceps than after vacuum. If a practitioner believes that delivery can successfully be accomplished with either instrument and has appropriate training and skill using both, choosing a vacuum extractor will minimize long-term maternal incontinence compared with forceps.

Figure 5. Forceps Delivery Versus Vacuum

![Risk Ratio Graph](image-url)
In one study, although retinal hemorrhage was found in neonates after both vacuum and forceps delivery, vision at 5 years of age was normal. Clinically significant intracranial hemorrhage is a rare complication of vacuum and forceps delivery; however, it should be noted that asymptomatic subdural hemorrhage also occurs during spontaneous birth. A series of 111 asymptomatic neonates screened with postnatal magnetic resonance imaging found subdural hemorrhage in 6% of spontaneous deliveries, 8% of vacuum assisted deliveries, and 28% of deliveries accomplished with the use of vacuum and forceps. All infants remained asymptomatic, and all hemorrhages resolved on follow-up magnetic resonance imaging.

California surveillance data demonstrated that the lowest risk of fetal intracranial injury occurred with spontaneous vaginal delivery and CS without labour. An intermediate risk occurred in those infants who had vacuum- or forceps-assisted deliveries or an intrapartum CS. The highest risk was reported in infants delivered with a combination of vacuum and forceps or who had a CS following unsuccessful assisted vaginal delivery. The relative clinical significance of these hemorrhages was not reported. Long-term follow-up of children delivered by vacuum or forceps has not revealed differences in neurological abnormalities or cognitive development when compared with children who delivered spontaneously.

<table>
<thead>
<tr>
<th>Neonatal</th>
<th>Forceps (n/N)</th>
<th>Ventouse (n/N)</th>
<th>Risk Ratio (RR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apgar &lt; 7 at 1 min.</td>
<td>77/701</td>
<td>78/722</td>
<td>1.08 [0.83, 1.41]</td>
</tr>
<tr>
<td>Apgar &lt; 7 at 5 min.</td>
<td>17/702</td>
<td>21/723</td>
<td>0.82 [0.44, 1.54]</td>
</tr>
<tr>
<td>Low pH Umbil. Artery</td>
<td>22/132</td>
<td>24/132</td>
<td>0.92 [0.54, 1.55]</td>
</tr>
<tr>
<td>Scalp Injury</td>
<td>25/848</td>
<td>18/840</td>
<td>1.36 [0.75, 2.48]</td>
</tr>
<tr>
<td>Facial Injury</td>
<td>10/596</td>
<td>1/580</td>
<td>5.10 [1.12, 23.25]</td>
</tr>
<tr>
<td>Intracranial Injury</td>
<td>1/115</td>
<td>0/143</td>
<td>4.83 [0.20, 115.98]</td>
</tr>
<tr>
<td>Cephalohematoma</td>
<td>66/1273</td>
<td>120/1283</td>
<td>0.64 [0.37, 1.11]</td>
</tr>
<tr>
<td>Fracture</td>
<td>1/315</td>
<td>0/322</td>
<td>3.07 [0.13, 74.96]</td>
</tr>
<tr>
<td>Retinal Hemorrhage</td>
<td>30/584</td>
<td>42/546</td>
<td>0.68 [0.43, 1.06]</td>
</tr>
</tbody>
</table>

Table 2. Effect of Mode of Delivery in Nulliparous Women on Neonatal Intracranial Injury
Singleton Nulliparous Deliveries, California 1992–1994

<table>
<thead>
<tr>
<th>DELIVERY METHOD</th>
<th>NUMBER</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>SVD</td>
<td>387 799</td>
<td>66.5</td>
</tr>
<tr>
<td>Vacuum</td>
<td>59 354</td>
<td>10.2</td>
</tr>
<tr>
<td>Forceps</td>
<td>15 945</td>
<td>2.7</td>
</tr>
<tr>
<td>Vacuum &amp; forceps delivery</td>
<td>2817</td>
<td>0.5</td>
</tr>
<tr>
<td>Caesarean section</td>
<td>117 425</td>
<td>20.1</td>
</tr>
<tr>
<td>Total</td>
<td>583 340</td>
<td>100</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>DELIVERY METHOD</th>
<th>INTRACRANIAL HEMORRHAGE RATE</th>
<th>RATE, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>CS without labour</td>
<td>1 per 2750</td>
<td>0.04</td>
</tr>
<tr>
<td>Spontaneous vaginal delivery</td>
<td>1 per 1900</td>
<td>0.05</td>
</tr>
<tr>
<td>Intrapartum CS</td>
<td>1 per 907</td>
<td>0.11</td>
</tr>
<tr>
<td>Vacuum assist</td>
<td>1 per 860</td>
<td>0.12</td>
</tr>
<tr>
<td>Forceps assist</td>
<td>1 per 664</td>
<td>0.15</td>
</tr>
<tr>
<td>CS after attempted AVB</td>
<td>1 per 334</td>
<td>0.33</td>
</tr>
<tr>
<td>Vacuum &amp; forceps</td>
<td>1 per 256</td>
<td>0.39</td>
</tr>
</tbody>
</table>

In a Canadian cohort study of 288 women who experienced a failed vacuum delivery, 81.5% had a successful forceps delivery and 5.9% had a CS following failed vacuum and forceps. There were no subgaleal, intracranial, subdural, or intraventricular hemorrhages, and no skull fractures. Other small studies have concluded that sequential use of instruments for AVB can increase neonatal injury.

Caution: sequential use of vacuum and forceps

The Society of Obstetricians and Gynaecologists of Canada, The Royal College of Obstetricians and Gynaecologists, and the American College of Obstetricians and Gynecologists suggest caution with sequential use of vacuum and forceps because of the potential for fetal injury. If descent is not achieved with traction on a correctly applied vacuum cup, cephalopelvic disproportion must be suspected, and an attempt at forceps-assisted delivery is not routinely advisable. However, if a vacuum or forceps cannot be optimally applied, switching instruments before traction may be appropriate. If an initial vacuum traction effort fails for a technical reason, correcting the problem or switching instruments may also be appropriate. Occasionally, vacuum traction results in adequate descent, but excessive chignon develops, filling the...
cup and removing the surface area required to generate traction. This is a technical failure of the vacuum rather than CPD, and completion of the delivery with forceps from a low or outlet station is unlikely to compound fetal risk. The Royal College of Obstetricians and Gynaecologists notes that the risks of forceps versus a Caesarean section at advanced station should be considered and that judicious use of outlet forceps after failed vacuum may avert a potentially difficult Caesarean section.38

Table 3. Criteria for Use of Vacuum and Forceps

<table>
<thead>
<tr>
<th>VACUUM</th>
<th>FORCEPS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Indications</strong></td>
<td><strong>Indications</strong></td>
</tr>
<tr>
<td>• Atypical or abnormal fetal heart rate pattern</td>
<td>• Atypical or abnormal fetal heart rate pattern</td>
</tr>
<tr>
<td>• Medical indications to avoid Valsalva manoeuvre (e.g., cerebral vascular disease, cardiac conditions)</td>
<td>• Medical indications to avoid Valsalva manoeuvre (e.g., cerebral vascular disease, cardiac conditions)</td>
</tr>
<tr>
<td>• Inadequate progress of labour</td>
<td>• Inadequate progress of labour</td>
</tr>
<tr>
<td>• Lack of effective maternal expulsive effort1</td>
<td>• Lack of effective maternal expulsive effort1</td>
</tr>
<tr>
<td>• Autorotation of fetal malposition possible</td>
<td>• Suboptimal attitude or position of the fetal head may be corrected</td>
</tr>
<tr>
<td>• Assistance at CS if required</td>
<td>• Assistance at CS if required</td>
</tr>
<tr>
<td><strong>Contraindications</strong></td>
<td><strong>Contraindications</strong></td>
</tr>
<tr>
<td>• Non-cephalic, face, or brow presentation</td>
<td>• Non-cephalic or brow presentation (except Piper forceps for after coming head of a breech)</td>
</tr>
<tr>
<td>• Lack of knowledge of fetal position</td>
<td>• Lack of knowledge of fetal position</td>
</tr>
<tr>
<td>• Fetal conditions (e.g., bleeding disorder, demineralization disorder)</td>
<td>• Fetal conditions (e.g., bleeding disorder, demineralization disorder)</td>
</tr>
<tr>
<td>• Any contraindications to vaginal delivery</td>
<td>• Any contraindication to vaginal delivery</td>
</tr>
<tr>
<td>• Less than 34 weeks’ gestation2</td>
<td>• Less than 34 weeks’ gestation2</td>
</tr>
<tr>
<td><strong>Prerequisites</strong></td>
<td><strong>Prerequisites</strong></td>
</tr>
<tr>
<td>A mnemonic delineating these prerequisites is included in this chapter and may be copied for use in labour and delivery suites.</td>
<td>A mnemonic delineating these prerequisites is included in this chapter and may be copied for use in labour and delivery suites.</td>
</tr>
<tr>
<td>• Informed consent</td>
<td>• Informed consent</td>
</tr>
<tr>
<td>• Appropriate anaesthesia</td>
<td>• Appropriate anaesthesia</td>
</tr>
<tr>
<td>• Maternal bladder empty</td>
<td>• Maternal bladder empty</td>
</tr>
<tr>
<td>• Vertex engaged</td>
<td>• Vertex engaged</td>
</tr>
<tr>
<td>• Cervix fully dilated</td>
<td>• Cervix fully dilated</td>
</tr>
<tr>
<td>• Adequate uterine contractions</td>
<td>• Adequate uterine contractions</td>
</tr>
<tr>
<td>• Membranes ruptured</td>
<td>• Membranes ruptured</td>
</tr>
<tr>
<td>• Position of the head must be known</td>
<td>• Position of the head must be known</td>
</tr>
<tr>
<td>• Experienced operator, adequate facilities, and resources available</td>
<td>• Experienced operator, adequate facilities, and resources available</td>
</tr>
<tr>
<td>• Operator knowledge of the instruments, their use, and the complications that can arise</td>
<td>• Operator knowledge of the instruments, their use, and the complications that can arise</td>
</tr>
<tr>
<td>• Reasonable chance of success (no evidence of CPD)</td>
<td>• Reasonable chance of success (no evidence of CPD)</td>
</tr>
<tr>
<td>• Backup plan if the procedure is unsuccessful</td>
<td>• Backup plan if the procedure is unsuccessful</td>
</tr>
<tr>
<td>• Ongoing fetal and maternal assessment</td>
<td>• Ongoing fetal and maternal assessment</td>
</tr>
<tr>
<td>• Appropriately skilled personnel for neonatal resuscitation</td>
<td>• Appropriately skilled personnel for neonatal resuscitation</td>
</tr>
</tbody>
</table>
Management

Vacuum Extraction

The vacuum extractor is designed to apply traction upon the fetal scalp to assist maternal expulsive effort and it is unlikely to succeed in the absence of maternal expulsive effort. It can be used for rotational deliveries; however, it must not be used to apply rotational forces. The vacuum may be used cautiously to correct attitude (deflexion) if it is correctly applied and if the direction of traction follows the pelvic curve.

Different types of vacuum extraction devices are available, including hard cups, soft silastic cups, and the Kiwi OmniCup. Although several comparisons of different cups have been published, the success and complication rates of each device are difficult to compare because operator training and experience are essential to optimize outcomes. In some reports, the Kiwi OmniCup is associated with a higher failure rate than the soft cup vacuum, whereas in others (with caregivers trained and mentored in the use of the Kiwi OmniCup), failure rates are lower. In a 2010 Cochrane review, the anterior soft cup was compared with a metal cup. The metal cup had a lower rate of failed delivery (8 studies with 1076 women; RR 1.63, 95% CI 1.17 to 2.28). Scalp injury and cephalohematoma were less likely with the soft cup (RR 0.67 and 0.61 respectively). There was no significant difference in other neonatal or maternal outcomes including Caesarean section, episiotomy, and perineal and vulvar trauma.

Success with vacuum-assisted birth requires application of the cup to the flexion point on the fetal scalp. The flexion point is just anterior to the posterior fontanelle. In a flexing application, the cup is centered over the flexion point with the posterior edge over the posterior fontanelle and the anterior edge well back from the anterior fontanelle. This minimizes the fetal head diameter that must transit the maternal pelvis, reducing the force required, the likelihood of pop-offs, and the risk of fetal and maternal trauma.

Optimal vacuum success is achieved with a tailored approach that matches an instrument appropriate to the clinical situation. If the fetus is in an anterior position at a low station, any vacuum cup can be successfully applied in a flexing application, and re-usable or disposable soft cups can be used to minimize minor scalp trauma. For mid-pelvic anterior deliveries, larger cups such as a Kobayashi, Silc, or 6 cm anterior Bird cup can generate more traction and are more likely to succeed than smaller cups. For deliveries with the fetus in an occiput transverse or posterior position, the Kiwi OmniCup and the 5 cm Bird posterior cups have pivoting traction handles and side-port suction that allow placement high enough in the pelvis to achieve a flexing application. This allows autorotation to occur with traction in the axis of the pelvis. For occiput transverse and posterior deliveries, a vacuum extractor with a handle that is perpendicular to the plane of the cup should not be used, as the flexion point cannot be reached.
Figure 6. Correct Cup Application for Occiput-Posterior and -Transverse Positions

[Diagram showing incorrect and correct cup application]

Used with permission of Salus Global Corporation
CLINICAL SITUATION | SUGGESTED VACUUM DEVICE
--- | ---
Station 0 to +2; occiput anterior | Bird 6 cm metal; Kobayashi or Silc silicone cup
Any station > 0; occiput transverse or posterior | Kiwi 5 cm OmniCup; Bird 5 cm posterior cup
Station ≥ +3; occiput anterior | Kobayashi, Silc, Kiwi OmniCup, Mityvac, or Bird 6 cm anterior cup

The caregiver should have proper training and experience with the specific instrument used, recognizing that skills may be different for each instrument. Vacuum pop-offs are not normal in a vacuum-assisted delivery. Causes include:

- Poor seal causing vacuum leak
- Impingement of maternal soft tissue
- Deflexing application
- Cephalopelvic disproportion (CPD)
- Uncontrolled traction technique (no counter-traction)
- Traction that is too rapid
- Improper angle of traction

**Forceps Application**

Debate about the indications for and safety of forceps operations has continued for over 200 years, and has mostly been concerned with mid-forceps deliveries. In most countries, rates of CS have risen as operative vaginal delivery rates have fallen. This trend has not been shown to confer benefit to the mother or baby.
The forceps application is checked in three ways. The following mnemonic assists in this endeavour. Position For Safety, Posterior fontanelle, Fenestration, Sagittal suture.

If forceps are applied so that the posterior fontanelle is more than a fingerbreadth above the plane of the shanks, traction will result in deflexion of the fetal head, increasing the diameter of the presenting part for delivery. If greater than a fingerbreadth of fenestration is still palpable after application of the blades, then the blades may slip, increasing the likelihood of fetal injury. When the sagittal suture is not perpendicular to the plane of the shanks throughout its length, the application of the forceps is asymmetrical, increasing the risk of fetal injury.

Obstetrical forceps are applied to the fetal head to perform the following functions:

- Traction
- Rotation (if required)
- Flexion
- Extension

When one or more of these functions is attempted, there is simultaneous fetal head compression, which is the primary undesirable effect associated with the use of forceps. Proper technique, including accurate application and correct traction, can minimize compressive forces.

Forceps should never be applied through a cervix that is not fully dilated or with an unengaged presenting part.

Techniques for Use of Vacuum and Forceps

In all cases of assisted vaginal delivery, the obstetrical care provider should consider

- Fetal status before and throughout the attempted delivery
- The time required to initiate a Caesarean section if the procedure fails.
- Choice of instrument, based on clinical circumstances and operator experience.
- Location of the delivery, which should be determined in discussion with the team:
  - For delivery from a low or outlet station when the operator is very confident of success, delivery in a hospital delivery room is appropriate.
  - For delivery from the mid-pelvis (above station +2), when malposition is present (OT or OP), or if the operator is not confident of success, delivery should be performed in an operating room with immediate access to Caesarean section.
  - However, if the fetal heart rate is abnormal, it may be appropriate to attempt delivery in a delivery room while preparations for emergency Caesarean section are underway.
Figure 8. Assisted Vaginal Birth Decision Tree

Assisted Vaginal Birth Decision Tree

Alternatives to AVB have been considered: Time?, Oxytocin?, Analgesia?, C-Section?

Yes

AVB indicated

Yes

Prerequisites achieved? Contraindications absent? (including that of a vaginal birth)

No Other Plan

Yes

Fetal Status

FHR Abnormal

AVB in labor room and Call for back-up: OB, OR, Peds, Anesthetist as necessary

No

FHR Normal

Determine best location for AVB: Rotation <45°? Spines +2 or more? High likelihood of success?

Yes

Low/Outlet AVB in labor room Call for back-up as necessary

No

Trial of AVB in OR with double set up Call for back-up

Used with permission of Salus Global Corporation
### Table 4. Assisted Vaginal Birth Mnemonic

<table>
<thead>
<tr>
<th>VACUUM</th>
<th>FORCEPS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A</strong></td>
<td><strong>ADDRESS</strong>&lt;br&gt;<strong>ANAESTHESIA</strong>&lt;br&gt;<strong>ASSISTANCE</strong>&lt;br&gt;<strong>ABSENCE</strong></td>
</tr>
<tr>
<td>• Address and obtain consent. The indication(s) and plan must be clear, well understood by the birthing woman and fully documented</td>
<td></td>
</tr>
<tr>
<td>• Adequate pain relief is available</td>
<td></td>
</tr>
<tr>
<td>• Assistance for the neonate is available</td>
<td></td>
</tr>
<tr>
<td>• Absence of contraindication to the procedure</td>
<td></td>
</tr>
<tr>
<td><strong>B</strong></td>
<td><strong>BLADDER</strong></td>
</tr>
<tr>
<td>• Bladder catheterized</td>
<td></td>
</tr>
<tr>
<td>• Backup plan discussed with birthing woman and team, documented; resources available</td>
<td></td>
</tr>
<tr>
<td><strong>C</strong></td>
<td><strong>CERVIX</strong>&lt;br&gt;<strong>CONTRACTIONS</strong></td>
</tr>
<tr>
<td>• Cervix fully dilated, membranes ruptured</td>
<td></td>
</tr>
<tr>
<td>• Adequate contractions and maternal expulsive efforts</td>
<td></td>
</tr>
<tr>
<td><strong>D</strong></td>
<td><strong>DETERMINE</strong>&lt;br&gt;<strong>DYSTOCIA</strong></td>
</tr>
<tr>
<td>• Determine fetal position, station, and pelvic adequacy</td>
<td></td>
</tr>
<tr>
<td>• Determine location of delivery (in delivery room or trial in OR)</td>
<td></td>
</tr>
<tr>
<td>• Prepare for possible shoulder dystocia</td>
<td></td>
</tr>
<tr>
<td><strong>E</strong></td>
<td><strong>EQUIPMENT</strong>&lt;br&gt;<strong>EFM</strong></td>
</tr>
<tr>
<td>• Inspect vacuum cup, pump, tubing, and check pressure</td>
<td></td>
</tr>
<tr>
<td>• EFM indicated: keep scalp clip applied, re-apply clip beside vacuum, or ensure adequate transabdominal fetal monitoring</td>
<td></td>
</tr>
<tr>
<td>• Select forceps according to clinical requirements.</td>
<td></td>
</tr>
<tr>
<td>• Phantom application</td>
<td></td>
</tr>
<tr>
<td>• EFM indicated: keep scalp clip applied, or ensure adequate transabdominal fetal monitoring</td>
<td></td>
</tr>
<tr>
<td><strong>F</strong></td>
<td><strong>FONTANELLE</strong>&lt;br&gt;<strong>FLEXION POINT</strong>&lt;br&gt;<strong>FOR VACUUM</strong></td>
</tr>
<tr>
<td>• Introduce the cup into the posterior aspect of the vagina while protecting the maternal tissues and making space with the opposite hand</td>
<td></td>
</tr>
<tr>
<td>• Soft cups are inserted by compressing the cup in an AP diameter</td>
<td></td>
</tr>
<tr>
<td>• Hard cups are slid in sideways and flipped onto the fetal skull</td>
<td></td>
</tr>
<tr>
<td>• The cup is centred over the flexion point (the point where traction will facilitate the smallest diameter of the fetal skull passing through the pelvis). The flexion point is over the sagittal, suture just anterior to the posterior fontanelle on the fetal skull.</td>
<td></td>
</tr>
<tr>
<td>• In the correct position the edge of the cup reaches onto the posterior fontanelle.</td>
<td></td>
</tr>
<tr>
<td>• When the fetus is OP, the flexion point is usually more posterior than first appreciated. With correct application, the 11-cm mark on the traction cable of a Kiwi OmniCup should be at the fourchette.</td>
<td></td>
</tr>
<tr>
<td>• Ensure that no maternal tissue is between the fetal head and the vacuum cup by sweeping finger around cup to clear maternal tissue. Reconfirm this after any loss of contact during traction. Note: It is often not possible to reach to the far edge of a Kiwi OmniCup or 5 cm Bird cup correctly applied to the flexion point of an OT or OP fetus.</td>
<td></td>
</tr>
<tr>
<td>• Initially increase vacuum pressure to resting pressure 100–200 mm Hg (0.1–0.3 kg/cm²) then, for traction, increase to 500–600 mm Hg (0.6–0.8 kg/cm²)</td>
<td></td>
</tr>
<tr>
<td>• Forceps are applied between contractions. Traction may be performed with or without a contraction</td>
<td></td>
</tr>
<tr>
<td>• Left hand, maternal left side, pencil grip and vertical insertion, with right thumb directing blade</td>
<td></td>
</tr>
<tr>
<td>• Right blade, right hand, maternal right side, pencil grip and vertical insertion with left thumb directing blade</td>
<td></td>
</tr>
<tr>
<td>• Fingers should be inserted into the vagina only to guide the blades and not to apply pressure on or to displace the fetal head</td>
<td></td>
</tr>
<tr>
<td>• Lock blade and support; should lock without pressure</td>
<td></td>
</tr>
<tr>
<td>• Check application PFS:</td>
<td></td>
</tr>
<tr>
<td>− Posterior fontanelle located midway between the blades, and 1 fingerbreadth above the plane of the shanks with the lambdoid sutures equal distance from the forceps blades</td>
<td></td>
</tr>
<tr>
<td>− Fenestration of blades barely felt. Equal amount of fenestration felt on each side (with a solid blade, it should be possible to insert no more than a fingertip between the blade and the fetal head)</td>
<td></td>
</tr>
<tr>
<td>− Sagittal suture perpendicular to plane or shanks with occipital sutures 1 cm above respective blades</td>
<td></td>
</tr>
</tbody>
</table>
### Assisted Vaginal Birth

<table>
<thead>
<tr>
<th>VACUUM</th>
<th>FORCEPS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>G</strong></td>
<td><strong>H</strong></td>
</tr>
<tr>
<td><strong>GENTLE</strong></td>
<td><strong>HALT</strong></td>
</tr>
<tr>
<td><strong>TRACTION</strong></td>
<td><strong>TRACTION</strong></td>
</tr>
<tr>
<td>• The vacuum pressure may be released between contractions to rest pressure or maintained at traction pressure.(^{55})</td>
<td>• Traction in axis of birth canal</td>
</tr>
<tr>
<td>• No rotational force is applied but the fetal head may rotate on its own with descent.</td>
<td>• Handle elevated (do not elevate handle too early)</td>
</tr>
<tr>
<td>• Traction is applied in the direction of the pelvic curve—initially downward and finally upward.</td>
<td></td>
</tr>
<tr>
<td>• Non-dominant thumb on cup to provide counter-traction and finger monitoring edge contact with fetal scalp to control descent and detect imminent pop-off.</td>
<td></td>
</tr>
<tr>
<td>• As contraction begins:</td>
<td></td>
</tr>
<tr>
<td>– ensure adequate vacuum pressure</td>
<td></td>
</tr>
<tr>
<td>– pull in the axis of the birth canal with contraction and maternal expulsive efforts</td>
<td></td>
</tr>
</tbody>
</table>

The decision to pause, continue, call for assistance, or move to a backup plan rests with the clinician and is based on clinical circumstance (e.g., fetal well being, progress, likelihood of success). Guidelines are provided as to when to reconsider but the decision is made by the clinician performing the procedure based on his/her expertise and assessment of the clinical situation.

**Caesarean section advised if**

- No progress after two pulls with a properly positioned cup and good traction
- Delivery is not imminent after four contractions, reassess the method of delivery
- 3 pop-offs, without obvious cause
- 20 minutes elapsed time and delivery is still not imminent\(^{55, 56}\)

The longer the cup is on, the more chance there is of fetal trauma.\(^{55, 57}\) It is imperative that some descent is observed with each pull. If these limits are reached and delivery is not imminent or there is evidence of scalp trauma, the procedure should be abandoned.\(^{55}\)

**Caesarean section advised if**

- Difficulty or failure of proper application
- Failure of rotation, if attempted and required
- No descent with initial traction
- If delivery is not imminent after 3 traction attempts\(^{57}\)
<table>
<thead>
<tr>
<th>I INCISION</th>
<th>VACUUM</th>
<th>FORCEPS</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Routine episiotomy is not required for every assisted vaginal birth.² ⁵⁸</td>
<td>• Selective mediolateral episiotomy has been shown to reduce OASI in assisted vaginal birth.⁵⁵ The number of episiotomies needed to prevent one OASI appears to be between 8 and 20.⁴⁹</td>
<td>• An episiotomy should be performed when the perineum is well distended by the fetal head. If performed too early, the episiotomy is likely to extend and may involve the anal sphincter.</td>
</tr>
</tbody>
</table>
| • Midline episiotomies increase the risk of third and fourth degree tears and should be avoided.² ⁵⁸ ⁶¹ | • “Mediolateral” episiotomies are commonly cut too close to the midline.⁶² ⁶³ To avoid the anal sphincter, the angle should be 60–70 degrees from midline (20–30 degrees from horizontal) when the perineum is distended. After delivery, this angle decreases by approximately 20 degrees.⁴⁴ Initiating the episiotomy 1 cm lateral to the midline on the fourchette may also be protective.⁶³ | • Certain factors increase the risk of OASI and should lower the threshold for performing an episiotomy:  
  − when forceps are used  
  − in nulliparous women  
  − when abnormal FHR requires rapid delivery without time for the perineum to stretch  
  − in women with a prior history of OASI |
| J JAW | • Remove vacuum when jaw is reachable or delivery assured | • Forceps are removed as the fetal head delivers through the perineum. |
Figure 9. Correct Episiotomy Placement

Used with permission of Salus Global Corporation
Follow-Up

Maternal and neonatal care should include

- Active third stage management
- Umbilical arterial and venous blood gas analysis
- Examination for maternal trauma, including vaginal and/or cervical lacerations and OASI; if OASI occurs, prophylactic IV antibiotics, stool softeners, and pelvic physiotherapy should be provided
- Examination for neonatal trauma, including scalp trauma, signs of cerebral irritation (poor suckling, listless), cephalohematoma, or SGH

Many centres have established observation protocols for newborns who have had forceps or vacuum applied during their delivery. Evidence for a particular subgaleal surveillance protocol is lacking; however, the following protocol is suggested: perform newborn assessments at 1h, 2h, 4h, then q4h x 24 h involving measurement of newborn head circumference and heart rate. Notify physician of an increase in head circumference of 1 cm or more or heart rate greater than 170 beats per minute. Further aspects of assessment may include bogginess or ballotable scalp, lethargy, and colour (modified McMaster protocol).

Neonatal trauma occurs more commonly in primigravidas, but the overall rate after vacuum delivery is low. In a Canadian study of 1000 deliveries using the Kiwi OmniCup, neonatal trauma rates were abrasions/blister 11.4%, hematoma 14.7%, intracranial hemorrhage 0.4%, and 1 subgaleal hemorrhage (0.1%) that did not require transfusion.

The delivery should be reviewed with the parturient woman and her partner. Reassurance should be given regarding chignon, caput, cephalohematoma, or facial marks.

Documentation

The indication and method of operative technique employed must be clearly and completely documented in all operative deliveries. The position and station of the fetal head at the commencement of the intervention must be stated. A contemporaneous written note and a dictated operative record are recommended.

The need for the intervention must be convincing, compelling, and documented.

Discussion and informed consent are essential before the intervention; however, in cases of abnormal fetal heart rate, the consent process may necessarily be brief. After assisted vaginal delivery, the parturient women should be given a clear explanation describing the procedure and its outcome. All questions should be addressed and contemporaneous documentation can then be completed.
Suggested format for a chart note

- Date/time
- Physician
- Indication
- Record of discussion with the parturient woman of the risks, benefits, and options
- Position and station of the fetal head, (vaginally and abdominally)
- Amount of moulding and caput present
- Assessment of maternal pelvis
- Assessment of fetal heart rate and contractions
- Type of vacuum or forceps used
- Number of attempts and ease of application of vacuum or forceps
- Duration of traction for forceps and duration of application for vacuum (start and stop time noted), and force used
- Any rotation applied with forceps or autorotation that occurs with vacuum
- For vacuum, number of pop-offs
- Position of chignon on fetal scalp (vacuum): flexing versus deflexing; median versus paramedian (see Figure 7 above)
- Description of any maternal and neonatal injuries
- Initiation of monitoring for subgaleal hemorrhage (vacuum)

This may also serve as a template to dictate a delivery summary.

Summary

Assisted vaginal birth by vacuum or forceps is an appropriate and effective obstetrical intervention in certain clinical situations. Clinical indications, contraindications, and expected maternal and fetal/neonatal outcomes should be carefully assessed before the instrument is applied.
References


## Appendix A.

### Table of Vacuum Conversions\(^9\)

<table>
<thead>
<tr>
<th>mm Hg</th>
<th>inches Hg</th>
<th>lb/in(^2)</th>
<th>KG/CM(^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>100</td>
<td>3.9</td>
<td>1.9</td>
<td>0.13</td>
</tr>
<tr>
<td>200</td>
<td>7.9</td>
<td>3.9</td>
<td>0.27</td>
</tr>
<tr>
<td>300</td>
<td>11.8</td>
<td>5.8</td>
<td>0.41</td>
</tr>
<tr>
<td>400</td>
<td>15.7</td>
<td>7.7</td>
<td>0.54</td>
</tr>
<tr>
<td>500</td>
<td>19.7</td>
<td>9.7</td>
<td>0.68</td>
</tr>
<tr>
<td>600</td>
<td>23.6</td>
<td>11.6</td>
<td>0.82</td>
</tr>
</tbody>
</table>
# Appendix B: Vacuum Mnemonic

| A       | ADDRESS                      | • consent                      |
|         | ANAESTHESIA                  | • adequate pain relief         |
|         | ASSISTANCE                   | • neonatal support             |
|         | ABSENCE                      | • of contraindication          |
| B       | BLADDER                      | • bladder empty                |
| C       | CERVIX CONTRACTIONS          | • fully dilated, membranes ruptured |
|         |                               | • adequate                     |
| D       | DETERMINE                    | • position, station and pelvic adequacy |
|         |                               | • think possible shoulder dystocia |
| E       | EQUIPMENT                    | • inspect vacuum cup, pump, tubing and check pressure |
| F       | FONTANELLE                   | • position the cup just anterior to or over the posterior fontanelle |
|         |                               | • sweep finger around cup to clear maternal tissue |
|         |                               | • 100 mm Hg initially          |
| G       | GENTLE TRACTION              | • as contraction begins:        |
|         |                               |   - increase pressure to APPROX. 600 mm Hg (follow manufacturer’s range) |
|         |                               |   - prompt mother for good expulsive effort |
|         |                               |   - pull with contractions only |
|         |                               | • traction in axis of birth canal |
|         |                               | • pressure may be maintained in between contractions, but no traction is to be exerted. |
| H       | HALT                         | if:                           |
|         |                               | • no progress after two pulls  |
|         |                               | • no imminent delivery after 4 contractions |
|         |                               | • no progress after 3 pop-offs |
|         |                               | • no progress after 20 minutes |
| I       | INCISION                     | • consider episiotomy (not routinely required) |
| J       | JAW                          | • remove vacuum when jaw is reachable or delivery assured |

Adapted from Bachman J. J Fam Pract 1989.
## Appendix C: Forceps Mnemonic

<table>
<thead>
<tr>
<th></th>
<th>Description</th>
</tr>
</thead>
</table>
| A | ADDRESS ANAESTHESIA  
ASSISTANCE  
ABSENCE                      |
|   | • consent  
• adequate pain relief  
• neonatal support  
• of contraindication         |
| B | BLADDER                                                                                       |
|   | • bladder empty                                                                               |
| C | CERVIX  
CONTRACTIONS                                                                                   |
|   | • fully dilated, membranes ruptured  
• adequate                                            |
| D | DETERMINE                                                                                     |
|   | • position, station and pelvic adequacy  
• think possible shoulder dystocia                    |
| E | EQUIPMENT                                                                                     |
|   | • check the equipment                                                                          |
| F | FORCEPS                                                                                       |
|   | • phantom application  
• left blade, left hand, maternal left side, pencil grip and vertical insertion, with right thumb directing blade  
• right blade, right hand, maternal right side, pencil grip and vertical insertion with left thumb directing blade  
• lock blade and support – check application  
• posterior fontanelle 1 cm above plane of shanks  
• fenestration not > 1 fingerbreadth between it and scalp  
• sagittal suture perpendicular to plane or shanks with occipital sutures 1 cm above respective blades |
| G | GENTLE TRACTION                                                                               |
|   | • applied with contraction/expulsive effort                                                     |
| H | HANDLE ELEVATED                                                                               |
|   | • traction in axis of birth canal  
• do not elevate handle too early                  |
| I | INCISION                                                                                      |
|   | • consider episiotomy (not routinely required)                                                 |
| J | JAW                                                                                           |
|   | • remove forceps when jaw is reachable or delivery assured                                    |

Adapted from Bachman J. J Fam Pract 1989.
# Table of Contents

Chapter 11 Delivery of Twins ................................................................. 260

  Introduction ........................................................................................................... 260
  Definitions ............................................................................................................. 260
  Incidence ................................................................................................................... 262
  Morbidity and Mortality ............................................................................................ 262
  Reducing Iatrogenic Twin Pregnancies .................................................................... 263
  Predisposing Factors ................................................................................................. 263
  Etiology .................................................................................................................... 263
  Diagnosis .................................................................................................................. 263
    Chorionicity ........................................................................................................... 263
  Management .............................................................................................................. 264
    Antenatal Considerations ......................................................................................... 264
    Delivery Location ..................................................................................................... 265
    Transport Considerations ......................................................................................... 265
    Method of Delivery .................................................................................................. 265
    Timing of Delivery ................................................................................................... 266
  Types of Presentations ............................................................................................... 267
  Management of Labour .............................................................................................. 269
    Spontaneous Labour ............................................................................................... 269
    Preterm Labour ...................................................................................................... 269
    Induction ................................................................................................................ 269
    Fetal Surveillance .................................................................................................. 269
    Analgesia and/or Anaesthesia for Multiple Gestation ............................................. 270
    Augmentation of Labour ......................................................................................... 270
    Cord Clamping and Blood Sampling ....................................................................... 270
    Third Stage and Postpartum Management .............................................................. 270
  Summary ................................................................................................................... 271
Chapter 11
Delivery of Twins

Introduction

Definitions

- **Multiple gestation/pregnancy**: more than one fetus in the same pregnancy.
- **Twin gestation**: two fetuses in the same pregnancy.

Twins may be:

- **Monozygotic**: developed from one ovum (egg).
- **Dizygotic**: developed from two ova (eggs).
- **Monochorionic**: developed with a single placenta (always monozygotic).
- **Dichorionic**: developed with two placenta (always diamniotic).
- **Monoamniotic**: fetuses in a single amniotic sac (always monozygotic and monochorionic).
- **Diamniotic**: fetuses in two, separate amniotic sacs.
- **Conjoined**: bodies of two fetuses are connected (always monozygotic).
Figure 1. Twin Chorionicity and Amnionicity

MONOCHORIONIC
MONOAMNIOTIC

MONOCHORIONIC
DIAMNIOTIC

DICHORIONIC DIAMNIOTIC
FUSED PLACENTAE

DICHORIONIC DIAMNIOTIC
SEPARATE PLACENTAE

Amnion
Amnion
Chorion
Chorion

Diamnionic, Monochorionic Twin Placenta
Diamnionic, Dichorionic Twin Placenta

Used with permission of Salus Global Corporation
Incidence

Spontaneous twins occur in approximately 1 in 90 pregnancies. As the use of ovulation induction and assisted reproductive technologies (ART) increased, the rate of multiple gestations also increased, reaching 3.1% in Canada by 2005. The rate has remained relatively stable since: in 2014, it was 3.3%. This is more than 12 000 births annually in Canada. The rate of monozygotic twins (3 to 5 per 1000 birth) is only increased by ART, specifically IVF, whereas the rate of dizygotic twins fluctuates with heredity, race, maternal age, and parity. Twins after ART are mostly dizygotic because they arise from multi-embryo transfer.

Morbidity and Mortality

Perinatal mortality rate:
- Monoamniotic twins (50% to 60%)
- Monochorionic diamniotic twins (4.4%)
- Dichorionic twins (1.2%)

Antenatal complications:
- Preeclampsia (10% to 20%)
- Prematurity (40% to 50%)
- Discordant growth and intrauterine growth restriction (15% to 25%)
- Twin-to-twin transfusion syndrome (5% to 10%)
- Congenital anomalies
- Death of one fetus (2% to 5%)
- Neurodevelopmental deficit in surviving twin after death of the other twin in a monochorionic twin pregnancy (25%)
- Cerebral palsy
- PROM

Complications related to delivery:
- Cord accidents
- Malpresentation
- Uterine atony
- Placental abruption
- Placenta Previa
- Vasa previa secondary to a velamentous insertion of the cord

Postpartum complications:
- Hemorrhage
- Postpartum depression
Reducing Iatrogenic Twin Pregnancies

A 2012 cohort study showed that compared with spontaneous twin pregnancies, those conceived using ART had higher rates of prematurity, Caesarean section, and obstetric morbidity (postpartum hemorrhage (PPH), premature rupture of membranes, and cervical insufficiency). It is therefore incumbent on care providers to be judicious in the application of ART. Strategies such as single IVF embryo transfer and embryo reduction are recommended, and in some jurisdictions are controlled by legislation.

Predisposing Factors

The largest influence on the rate of twins is the use of ART, which increases the rate of both monozygotic and dizygotic twins. The rate of dizygotic twins is also increased with:

- increasing maternal age and parity
- a history of twins on the maternal side
- women who are Black
- better nutritional status
- elevated pituitary gonadotropins, which is also a possible common link for the other predisposing factors (above)

Etiology

About two thirds of twins arise from the fertilization of 2 ova. They are called dizygotic or fraternal twins. About one third of twins arise from the fertilization of a single ovum that subsequently divides into 2 embryos. They are called monozygotic, or identical twins. Monozygotic embryos that fail to separate completely become conjoined twins.

Either or both processes can also result in more than 2 fetuses. For example, quadruplets may arise from a single ovum or as many as 4 ova.

Diagnosis

Twins should be considered in any woman with predisposing factors, and particularly in women who have undergone assisted reproduction. Routine first trimester dating ultrasound has the advantage of detecting twins early, when the chorionicity can most reliably be determined.

Chorionicity

- Chorionicity refers to the number of chorionic membranes present. Simply, monochorionic twins share one placenta and dichorionic twins each have their own placenta.
• Chorionicity should not be confused with zygosity. About 25% of monozygotic twins are dichorionic. All dizygotic twins are dichorionic.
• Monochorionic twins have one placenta with shared vascular circulation
• Dichorionic twins have 2 placenta with separate vascular circulation.
• The incidence of perinatal mortality is higher in monochorionic twins than in dichorionic twins. This is primarily due to twin-to-twin transfusion syndrome in diamniotic twins and cord entanglement in monoamniotic twins.
• Determining the chorionicity is important in the management of twin gestation. This is best done by an early ultrasound examination at 7 to 14 weeks.
• Establishing chorionicity by ultrasound can also be aided by examining the placenta, seeking the lambda sign in the dividing membrane, and determining the sex of the fetuses.

When ultrasound has not been obtained, a twin pregnancy should be suspected. Whenever the symphysis–fundal height is larger than expected. For instance, the late second trimester symphysis–fundal height exceeds the gestational age by an average of 5 centimetres in a twin gestation. The auscultation of a second fetal heart tone at any gestational age should also prompt the clinician to consider the diagnosis of twin pregnancy. Abdominal palpation alone is an unreliable method of detecting twins. In rare cases, twins may become apparent only at delivery.

The differential diagnosis of a large-for-gestational-age-uterus includes:
• Multiple pregnancy
• Molar pregnancy (hydatidiform mole)
• Inaccurate dates
• Polyhydramnios
• Macrosomia
• Uterine Fibroids
• Adnexal and/or abdominal masses

Management

Antenatal Considerations

Early diagnosis of twins allows for appropriate antenatal, intrapartum and post-partum planning.

Antenatal care focuses on screening for complications specific to twin pregnancies, in addition to care routine in all pregnancies. Complications specific to twin pregnancies include premature birth, growth discordance, intrauterine growth restriction, gestational hypertension, and twin-to-twin transfusion syndrome.
Prenatal Genetic Testing

- At 11 to 14 weeks, nuchal translucency in combination with maternal age ± serum analytes can be used to assess the risk of aneuploidy.
- Alternatively, non-invasive prenatal genetic testing is now widely available and valid for twins.

Ultrasound Surveillance

- Depending on resources and geography, ultrasound assessments are recommended in monochorionic diamniotic twins every 2 weeks from 16 weeks gestation to monitor for twin-to-twin transfusion syndrome (early enough to allow intervention) and selective IUGR.¹²
- For dichorionic twins, a growth ultrasound is recommended every 4 weeks, starting at 20 weeks to assess for growth restriction. In contrast to growth in singletons, growth in normal dichorionic twins falls off slightly at approximately 30 to 32 weeks. Normal monochorionic/diamniotic twin growth falls off at 28 weeks.¹³ ¹⁴

There is no evidence that routine hospitalization or bed rest prevents preterm birth for women with twins.¹⁵

Delivery Location

Delivery location should be discussed and agreed upon by the woman, her family, and her care providers. The 2000 SOGC consensus statement¹⁰ recommends that for a planned twin delivery, anaesthetic, obstetrical, neonatal, and nursing staff trained in twin delivery be present in hospital. A primary care physician or midwife may provide hands-on care under the supervision of a consultant physician/obstetrician who has expertise in the delivery of twins. Each of the twins should have an assigned care provider, skilled in neonatal resuscitation, present at the delivery. Caesarean section should be available.

Transport Considerations

Antenatal transport of the woman to another centre should be considered when local staffing or resources are insufficient, particularly when twins are preterm. Careful assessment of the safety of transport and communication between the sending and the receiving centres are essential, as described in the 2005 SOGC Maternal Transport Policy.¹⁸ Local, regional, and provincial guidelines should also be considered.

Method of Delivery

Vaginal delivery should be the goal unless there are contraindications. A 2013 trial that randomized 2804 women to planned Caesarean section or planned vaginal delivery (with CS if medically indicated) showed no benefit of planned CS. In this study, twins were ≥ 32 weeks gestation, twin A was vertex, and an obstetrician skilled in vaginal twin delivery was present.¹⁹ Three-month follow-up showed no difference in maternal outcomes including breastfeeding and problematic incontinence.²⁰ Two-year pediatric follow-up showed no difference in death or neurodevelopmental delay in children.
born by planned CS or planned vaginal birth. A French case series of 1009 women demonstrated similar safety and intrapartum CS rate for dichorionic and monochorionic/diamniotic twins with planned vaginal delivery.

**Timing of Delivery**

The timing of delivery of twins is a controversial topic. The 2011 NICE guidelines recommend a decision tree to help determine the optimal timing of delivery. This includes preparing the woman and her family for the possibility of an early delivery and its sequelae, even in an uncomplicated pregnancy. According to the NICE guidelines, approximately "60% of twin pregnancies result in spontaneous birth before 37 weeks. . . . Continuing uncomplicated twin pregnancies beyond 38 weeks increases the risk of fetal death".

In determining the optimal timing of delivery, the risk of stillbirth with continuing pregnancy must be weighed against the risk of adverse outcomes related to prematurity. Elective or spontaneous preterm birth increases the risk of neonatal admission to the NICU. As a result, the NICE guidelines suggest offering elective delivery at 36 weeks for monochorionic diamniotic twin pregnancies and at 37 weeks for dichorionic twin pregnancies. However, further studies have suggested that elective delivery before 37 weeks is of no benefit and may increase the rate of perinatal morbidity. Elective delivery, particularly by CS, at 36 to 37 weeks carries an increased risk of neonatal respiratory morbidity. Induction of labour with twins also carries maternal risks including an increased likelihood of CS if the cervix is unripe.

Further studies are being conducted to determine the optimum time of delivery. Currently, it is reasonable to offer elective delivery for monochorionic twins at between 36 and 37 weeks' gestation, and for dichorionic twins at between 37 and 38 weeks' gestation. If a woman declines elective delivery, weekly appointments with a specialist obstetrician are recommended with weekly antenatal fetal surveillance and biweekly growth ultrasound assessment. Delivery by 39 to 40 weeks is recommended to reduce the risk of stillbirth.

For monoamniotic twins, care in a tertiary centre should be considered from 24 weeks' gestation due to universal cord entanglement. Close surveillance and planned CS between 32 and 33 weeks is often recommended, although there is some evidence that later delivery may be appropriate.
Types of Presentations

Figure 2. Types of Presentation in Twin Gestation

Used with permission of Salus Global Corporation
Both Twins Cephalic

- Vaginal delivery should be expected.
- After the delivery of the first twin, the presentation of the second twin should be determined and cord presentation ruled out. If necessary, presentation should be confirmed using ultrasound.
- When uterine contractions are inadequate, augmentation of labour is recommended to assist in the descent of the head of the second twin.
- Artificial rupture of the membranes should be attempted when the head of the second twin has descended to station 0 or −1. Care should be taken to identify cases with cord presentation.

First Twin Cephalic, Second Twin Breech or Transverse

- First twin delivered vaginally.
- Vaginal delivery of the second twin is suggested as long as the estimated fetal weight is between 1500 and 4000 grams and the obstetrician is trained in vaginal breech delivery.\(^{31}\)
- Immediately after the delivery of the first twin, the presentation of the second twin must be confirmed. If there is any doubt about the presentation, ultrasound examination should be used to clarify. The decision to manage the second twin actively or expectantly is left to the individual obstetrician. If expectant management is chosen, ongoing fetal surveillance is required.
- If the second twin is in a footling breech presentation or in a transverse lie and fetal monitoring is atypical or abnormal, there are two options: (1) breech extraction (with internal podalic version if needed), and (2) CS.
- If the second twin is in a transverse lie and fetal monitoring is reassuring, there are three options: (1) external version to vertex, (2) internal podalic version and breech extraction, or (3) CS.
- **Breech extraction should not be performed if the second twin is significantly larger than the first.** If breech extraction is performed, it is preferable to leave the amniotic sac intact until one foot or both feet are identified and secured with the operating hand.

First Twin Breech

When the first twin is in frank or complete breech presentation, the same issues exist as for vaginal delivery of a singleton breech. The mother must be informed of all pertinent risks, including the possibility of expulsive delay due to trapped fetal parts resulting in adverse fetal outcome. This risk can be avoided by Caesarean section, which is generally recommended; however, several case series have shown reasonable safety and potential reduced maternal morbidity with a trial of labour in women with first twin breech.\(^{32,35}\)

First Twin Non-Longitudinal

If the first twin is not in a longitudinal lie, Caesarean section is indicated.
Management of Labour

Spontaneous Labour

Ideally, planned delivery locations should have been previously agreed upon and the woman (re-)located near her delivery hospital. Should she enter labour in a setting without prerequisite operative and specialist capabilities, the safety of transport to a larger centre must be weighed against the risks of local delivery with available resources.

Preterm Labour

Preterm labour is a frequent complication of multiple gestations and the primary cause of the increase in perinatal morbidity and mortality in multiple gestations. Women with multiple gestations should be taught the early warning signs and symptoms of preterm labour and advised to report promptly for evaluation. When preterm labour is diagnosed in a smaller hospital, consideration must be given to tocolytic therapy, administration of glucocorticoids, and transport of the woman to a regional referral centre. A 2012 Cochrane review does not support the routine use of betamimetics to suppress preterm labour in twin pregnancies.

Induction

There are no adequate clinical trials to date comparing elective induction of labour versus expectant management for uncomplicated twin pregnancies. The indications and contraindications for induction of labour with multiple gestations include all of the factors that would apply to a singleton gestation. Growth restriction of one or both twins, sometimes manifest as a significant disparity in estimated fetal weights, is a sufficient indication for induction.

The methods used for labour induction should be the same as those used for a singleton pregnancy. The safety of induction in the presence of a previous CS in a multiple pregnancy is unknown.

Fetal Surveillance

All fetuses must have assessment of their well-being in labour. Twin pregnancy carries higher risk of perinatal morbidity and mortality than singleton pregnancy. This is related to a number of factors including umbilical cord problems, placental dysfunction, and/or twin-to-twin transfusion. After delivery of the first twin, the second twin is at additional risk because of potential cord compression and intrapartum placental abruption.

Continuous, simultaneous electronic fetal monitoring is recommended for twin pregnancies during labour. Some current electronic monitoring machines have the capacity to use two separate Doppler ultrasound transducers and to externally monitor each twin successfully. Electronic monitoring may be more successful at producing interpretable tracings if the leading twin is monitored with a scalp electrode and the second twin with Doppler ultrasound. The scalp electrode should be applied as soon as labour is well-established.
Following delivery of the first twin, monitoring of the second twin should be continued with an external transducer. When the second twin is in a longitudinal lie and membranes can be safely ruptured, monitoring with a fetal scalp electrode can be commenced.

During the second stage of labour with twins, ultrasound is helpful to determine presentation and location of the fetal heart of twin B if necessary.

**Analgesia and/or Anaesthesia for Multiple Gestation**

The risks, benefits, and limitations of analgesia and anaesthesia should be discussed with the woman and her family. Epidural analgesia is used widely during labour with twin gestations. It provides effective labour analgesia and adequate anaesthesia for intrauterine fetal manipulation or CS, if required.

**Augmentation of Labour**

If labour is dysfunctional, augmentation is an option. The same indications and methods are used as in a singleton pregnancy. Augmentation of labour after delivery of the first twin may be appropriate.

**Cord Clamping and Blood Sampling**

There is no evidence to guide the practice of delayed cord clamping after delivery of the first twin. Delayed cord clamping of 60 seconds for each dichorionic twin would seem appropriate. For monochorionic twins, since there may be vascular communications between the placentas, delayed cord clamping of twin A may result in transfusion from twin B to twin A. A delay of perhaps 30 seconds might be more appropriate after delivery of a monochorionic twin A. Delayed cord clamping after delivery of monochorionic twin B is appropriate.

Blood may be obtained for cord gases from an isolated clamped segment of cord at the time of delivery of each twin; however, since placental vascular connections commonly exist in monochorionic twins, blood sampling or drainage of the intact cord must be delayed until after the birth of the second twin.

**Third Stage and Postpartum Management**

After delivery of the second twin, active management of the third stage of labour is indicated. There is an increased risk of PPH; therefore, an oxytocin infusion should be continued for 2 to 3 hours following delivery of the placenta to ensure that the uterus stays well-contracted. Sublingual or rectal misoprostol may be used as adjunct prophylaxis against delayed PPH as its effect lasts for several hours.

Whenever possible, the woman and her babies should be kept together postpartum. Breastfeeding twins can be challenging. Early initiation and expert assistance may benefit both mother and babies. The increased risk of postpartum depression following multiple births should be kept in mind, and renewed contact should be made with a multiple pregnancy support group or other previously identified support providers.
Summary

All complications of pregnancy are more likely to occur in a twin pregnancy, and a high level of vigilance and diligence
is required to minimize the inherent risks and optimize the outcomes. It is important to establish chorionicity early in a
twin pregnancy.

If possible, twins should be delivered in a centre with facilities for continuous electronic fetal surveillance, intrapartum
ultrasound, and emergency CS. Staff skilled in the care of twins should also be available.
References


2. Live births and fetal deaths (stillbirths), by type (single or multiple), Canada, provinces and territories. Table 102-4515. [CANSIM database]. Ottawa: 2013.


# Table of Contents

Chapter 12 Trial of Labour After Caesarean................................................................. 276

  Introduction ................................................................................................................. 276
  Definitions ..................................................................................................................... 276
  Incidence ....................................................................................................................... 276
  Factors to Consider ........................................................................................................ 277

Benefits and Risks of TOLAC .................................................................................... 278

  Maternal Outcomes Associated With TOLAC Versus ERCS ...................................... 278
  Uterine Rupture ............................................................................................................ 279
  Considerations for Future Pregnancies ......................................................................... 280

Management .................................................................................................................. 281

  Requirements for a TOLAC ......................................................................................... 281
  Selection of Candidates for a TOLAC ........................................................................... 282
  Special Considerations ................................................................................................. 282

Management of Labour ................................................................................................. 283

  Fetal Heart Rate Patterns and Uterine Rupture ............................................................ 283
  Induction and Augmentation of Labour ....................................................................... 284
  Signs and Symptoms of Uterine Scar Rupture .............................................................. 284
  Management of Uterine Rupture ................................................................................. 285

Summary ......................................................................................................................... 285
Chapter 12

Trial of Labour After Caesarean

Introduction

The primary indication for Caesarean section (CS) in Canada is a previous CS, accounting for over 30% of the total number of CS. Every year, over 30 000 women in Canada undergo either trial of labour or repeat CS. Professional associations, including the Society of Obstetricians and Gynaecologists of Canada, the Royal College of Obstetricians and Gynaecologists, and the American Congress of Obstetricians and Gynecologists, recommend that eligible women be offered a trial of labour after CS.\(^2\)

If a woman has requested a trial of labour after CS and is a good candidate for a planned trial of labour, she and her health care providers must understand and accept the risks and benefits. Not all women (or care providers) will find the same balance of risks and benefits acceptable.\(^2\) Discussions about delivery options in future pregnancies may begin as early as the postpartum period following CS.

Definitions

<table>
<thead>
<tr>
<th>Term</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vaginal birth after Caesarean (VBAC)</td>
<td>Vaginal delivery after a CS in a previous pregnancy.</td>
</tr>
<tr>
<td>Trial of labour after Caesarean (TOLAC)</td>
<td>A plan to attempt labour and vaginal delivery in a woman who has had a previous CS.</td>
</tr>
<tr>
<td>Elective repeat Caesarean section (ERCS)</td>
<td>A CS performed before the onset of labour in a woman who has had a CS in a previous pregnancy.</td>
</tr>
<tr>
<td>Uterine rupture</td>
<td>The complete separation of the myometrium with or without extrusion of the fetal parts into the maternal peritoneal cavity.</td>
</tr>
<tr>
<td>Uterine dehiscence</td>
<td>A clinically occult and incomplete disruption: the fetal membranes are not ruptured and the fetus is not outside the uterus, and the peritoneum over the defect is usually intact. Not all studies are consistent in this definition</td>
</tr>
</tbody>
</table>

Incidence

The Canadian Institute for Health Information reported the CS rate in Canada increased to 28.2 % and the repeat CS rate was 81.2 % in 2016-2017. The primary indication for CS in Canada is a previous CS, accounting for over 30% of the total. In British Columbia, the proportion of women with a previous cesarean delivery who were deemed eligible for vaginal birth after cesarean delivery increased from 75% in 2010 to 80% in 2014\(^4\)
In Canada, the TOLAC rate for women with only one previous pregnancy resulting in a CS was 32.8% in 2003–2004, decreased to 28.2% in 2007–2008 and then increased to 31.4% in 2013–2014.

A 2011 review found that the overall TOLAC rate among studies in the United States was 58%, with a range of 28% to 70%. In studies initiated after 1996, 44% of women had a TOLAC compared with 62% of women in studies initiated before 1996. The success rate among women who underwent TOLAC is approximately 74% in the United States.

Factors to Consider

In general, VBAC success is approximately 70-75% and is referenced as such in the Canadian, American, British and Australian/New Zealand guidelines. A prospective study of 14,529 women who underwent a TOLAC between 1999 and 2002 identified factors predictive of outcome. The following should be discussed with women considering the choice of a TOLAC versus repeat CS.

**Factors that increase the likelihood of a successful VBAC include:**
- Previous successful VBAC
- Previous vaginal delivery
- Favourable cervix
- BMI <30
- Birth weight <4000 g
- Spontaneous onset of labour
- Indication for previous CS (e.g., breech presentation) not present in current pregnancy
- Maternal age < 40 years

**Factors that decrease the likelihood of a successful VBAC include:**
- Previous CS performed for dystocia
- Need for induction of labour requiring cervical ripening
- Need for augmentation of labour
- Gestational age > 40 weeks
- Estimated birth weight > 4000 grams
- Maternal body mass index > 30 kg/m²
- Maternal hypertension

A prediction model for TOLAC success was developed retrospectively from 9,616 women who underwent a TOLAC. The successful VBAC rate was 75%. It has been shown to be valid in a Canadian population. This model predicts the probability of VBAC success and may be used in practice as a primary method to refine counselling during antepartum visits for women who have undergone a prior CS. The strongest predictor of a successful VBAC (85-90%) is a previous vaginal delivery. It is also associated with a decreased risk of uterine rupture. Adolescents are more likely than adult women to attempt VBAC, and they are as likely to be successful. Adolescents should be encouraged to attempt a trial of labour after prior CS when appropriate to lower the risks of lifelong maternal morbidity from numerous repeat CS.
Benefits and Risks of TOLAC

Successful VBAC avoids major abdominal surgery, has a lower risk of hemorrhage, thromboembolism, infection and a shorter recovery period than ERCS. In addition for those anticipating future pregnancies, VBAC reduced the risks of multiple CS including surgical risks and abnormal placentation. However, TOLAC is associated with maternal and neonatal risks. Maternal morbidity includes uterine rupture, hemorrhage, thromboembolism, and infection, hysterectomy and death. Most of the maternal morbidity from TOLAC occurs with failed TOLAC. Successful VBAC has fewer complications that ERCS but failed TOLAC and consequent repeat CS has a higher risk than ERCS. The key point is that the risk of maternal morbidity is primarily related to the likelihood of a successful VBAC. Stratification of patient based on prediction models and/or factors associated with success and failure of TOLAC is paramount.

Maternal Outcomes Associated With TOLAC Versus ERCS

There are currently no randomized controlled trials comparing maternal or neonatal outcomes in women undertaking TOLAC and those undergoing an elective repeat caesarean section (ERCS). Much of the evidence about the safety of a TOLAC versus an ERCS is based on observational data. Recommendations and decisions about a TOLAC should therefore be made cautiously.

A 2011 analysis found the following short-term maternal outcomes with TOLAC versus ERCS:

<table>
<thead>
<tr>
<th>POTENTIAL HARM</th>
<th>TOLAC</th>
<th>ERCS</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Maternal death</strong></td>
<td>3.8 per 100 000 TOLAC</td>
<td>13.4 per 100 000</td>
<td>Significantly higher for ERCS</td>
</tr>
<tr>
<td>(95% confidence interval [CI] 0.9 to 15.5)</td>
<td>(95% CI 4.3 to 41.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Uterine rupture</strong></td>
<td>4.7/1000 (0.47%)</td>
<td>0.3/1000 (0.026%)</td>
<td>Significantly higher for TOLAC</td>
</tr>
<tr>
<td>(95% CI 0.28 to 0.77)</td>
<td>(95% CI 0.009 to 0.082)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Length of stay</strong></td>
<td>2.55 days</td>
<td>3.92 days</td>
<td>Length of stay is higher for ERCS</td>
</tr>
<tr>
<td>(95% CI 2.34 to 2.76)</td>
<td>(95% CI 3.56 to 4.29)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Hemorrhage</strong></td>
<td>6.6 per 1000</td>
<td>4.6 per 1000</td>
<td>Not statistically significantly different</td>
</tr>
<tr>
<td>(95% CI 2.0 to 22.1)</td>
<td>(95% CI 1.6 to 13.2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Hysterectomy</strong></td>
<td>0.17%</td>
<td>0.28%</td>
<td>Not statistically significantly different</td>
</tr>
<tr>
<td>(95% CI 0.12 to 0.26)</td>
<td>(95% CI 0.12 to 0.67)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Uterine Rupture

Within the “Evidence Report/Technology Assessment Report Vaginal Birth After Cesarean: New Insights” it was noted that “While numerous studies have been published relating to uterine rupture and/or dehiscence (393 articles), only 8 cohort studies were good or fair quality, included the population of interest, and used the anatomic definition for uterine rupture contained in this report.”

The risk of uterine rupture for all women with a prior CS regardless of route of delivery is 0.3 percent (95% CI 0.2 to 0.4). The risk of uterine rupture for women undergoing a TOLAC is significantly elevated at 0.47 percent (95% CI 0.28 to 0.77) compared with the risk for women undergoing an ERCS (0.026; 95% CI 0.009 to 0.082). There were no maternal deaths due to uterine rupture in any of the 8 studies reviewed. The risk of hysterectomy due to uterine rupture ranged from 14% to 33%.

#### Risk factors for uterine rupture

Induction of labour increases the risk of uterine rupture. The risk of uterine rupture among women who had induction of labour was lowest with oxytocin (1.1%), followed by prostaglandin E₂ (2%), and highest with misoprostol (6%). However, these risk estimations may be imprecise given the inconsistency in study design and methodology; the results should be interpreted with caution.

Women with a prior classical (vertical) uterine incision are at increased risk of uterine dehiscence or rupture. Compared with women with prior low transverse CS, women with prior low vertical CS are not at a significantly increased risk of uterine dehiscence or rupture.

The following also increase the risk of uterine rupture (and all should be documented):

- Short interval from previous CS (< 18 months)
- More than 2 previous CS
- Previous CS for dystocia in the second stage of labour
- Locked single-layer closure of the previous uterine incision (single-layer unlocked or 2-layer closure acceptable)

### Potential Harm

<table>
<thead>
<tr>
<th>Potential Harm</th>
<th>TOLAC</th>
<th>ERCS</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infection rate</td>
<td>46 per 1000</td>
<td>32 per 1000</td>
<td>Not statistically significantly different</td>
</tr>
<tr>
<td></td>
<td>(95% CI 15 to 135)</td>
<td>(95% CI 13 to 73)</td>
<td>A trend towards increased endometritis was seen with ERCS compared with TOLAC; in contrast, chorioamnionitis was increased in TOLAC compared with ERCS. Increasing body mass index was associated with increased fever in patients undergoing TOLAC</td>
</tr>
</tbody>
</table>
Ultrasound assessment of lower uterine segment (LUS) thickness has studied to try to predict an increased risk of uterine rupture. A meta-analysis of LUS thickness showed a thickness of 2.1-4.0 mm had a strong negative predictive value and thickness of 0.6-2.0 mm a strong positive predictive value. The study could not determine a LUS thickness cut off value usable in clinical practice. This is a research area to watch. Some are advocating for its use.

Neonatal Mortality and Morbidity

A large meta-analysis found the rate of perinatal death to be significantly higher with TOLAC than with ERCS (1.3 per 1000; 95% CI 0.59 to 3.04 versus 0.5 per 1000; 95% CI 0.07 to 3.82). The overall risk of perinatal death due to uterine rupture was found to be 6.2%. Overall, the literature relating to response time between premonitory signs of uterine rupture and perinatal mortality is insufficient. However, there is suggestion that fetal bradycardia is an ominous sign for fetal extrusion, which is associated with poor perinatal outcomes.

The same meta-analysis found no differences in Apgar scores or in rates of sepsis or NICU admission between neonates whose mothers underwent TOLAC and those who underwent ERCS. There was insufficient evidence to determine if rates of respiratory distress, neonatal trauma, or asphyxia/hypoxic-ischemic encephalopathy varied between TOLAC and ERCS. No studies were found that explored the effect of a TOLAC versus an ERCS on breastfeeding initiation or continuation.

Considerations for Future Pregnancies

Placenta Previa

A 2010 review of 203 studies found that women who had undergone a prior CS had a statistically significant increased risk of placenta previa compared with women who had not undergone prior CS at a rate of 12 per 1000 (95% CI 8 to 15 per 1000). The incidence increased with increasing number of prior CS. Prior CS was a significant risk factor for maternal morbidity in women with placenta previa. Compared with women who had placenta previa but no prior CS, women with one prior CS and placenta previa had a statistically significant increased risk of blood transfusion (15% vs. 32.2%), hysterectomy (0.7% to 4% vs. 10%), and composite maternal morbidity (15% vs. 23% to 30%). For women with 3 or more prior CS and placenta previa, the risk of hysterectomy and composite maternal morbidity rose significantly (0.7% to 4% vs. 50% to 67%, and 15% vs. 83%, respectively).

Placenta Accreta

The incidence of placenta accreta rose with increasing number of prior CS. The results were statistically significant for women with 2 or more prior CS (odds ratio 8.6 to 29.8).

Women with placenta previa were at increased risk for placenta accreta, and the risk increased with increasing number of prior CS. Women with more than 3 prior CS and placenta previa had a 50% to 67% incidence of placenta accreta.
Management

Requirements for a TOLAC

Obstetrical guidelines recommend that a TOLAC should be attempted only in hospitals that can provide emergency CS, blood products, and neonatal resuscitation. A TOLAC is always associated with a risk of uterine rupture, however small, and a good outcome is not guaranteed under any circumstances. There is little evidence to provide guidance about how quickly a CS would need to be done to prevent serious neonatal morbidity and mortality. The ACOG and Green-top guidelines indicate the ability to perform an immediate CS. The ACOG guideline states that in settings where the resources are not immediately available, there should be a clear protocol to perform an emergency CS in a timely fashion. The SOGC guideline discusses a “timely” response to an emergency CS and recognizes some facilities may take up to 30 minutes. Timely emergency Cesarean sections (initiated within 30 minutes) although reasonable, may not prevent serious neonatal morbidity and mortality.

The 2019 SOGC TOLAC guideline states “that a TOLAC is optimal in an institution that has in-house obstetric, anesthesia, and surgical staff to reduce the decision-to-delivery interval in cases of suspected uterine rupture. The SOGC acknowledges that this may not be possible in birthing units across Canada.” Hospitals and care providers offering a TOLAC should have emergency protocols that include situations in which the specialists who are needed to perform CS may not be immediately available. Women should be aware of the resources to effect an urgent CS. Care providers should be able to recognize the signs and symptoms of uterine scar rupture and have a management plan in place should this occur. Regular emergency drills or other simulation exercises may be useful for team preparedness in these rare emergencies.

The SOGC recommends that a TOLAC be offered to women with one previous transverse low-segment CS following appropriate discussion and documentation of maternal and perinatal risks and benefits as part of obtaining informed consent.

Access to the previous operative report is desirable, and the opinion of the previous surgeon may be helpful. If the operative report is not available, TOLAC is permissible if the clinical circumstances of prior CS suggest there was an uncomplicated lower segment incision. There must be no contraindications to vaginal birth.

The following are contraindications to a TOLAC:

• Any contraindications to labour
• Previous or suspected classical uterine incision for CS
• Previous inverted T uterine incision
• Previous uterine rupture
• Previous major uterine reconstruction (e.g., full thickness repair for myomectomy, repair of müllerian anomaly, cornual resection)
• Inability to perform an emergency CS
• Patient refusal
Selection of Candidates for a TOLAC

Consent Discussion and Documentation

Informed consent for TOLAC should include a documented discussion of the risks and benefits of elective CS versus TOLAC. The likelihood of successful VBAC is fundamental to the discussion. Women must understand the evidence if they are to make informed decisions about planning a TOLAC or a repeat Caesarean section. A consent form specific for TOLAC/VBAC may be useful for hospitals offering TOLAC to reflect local resources and availability to perform an emergency CS.

Health care providers should be aware of the factors that influence women’s choices. These include:

- Care provider’s recommendation
- Recovery time and the need to return to previous activities, including caring for other children
- Cultural traditions and beliefs
- Safety for parturient and neonate

Patient counselling during the decision-making process (this should be documented):

- Discuss the risks and benefits of TOLAC and ERCS, including possible effects on future pregnancies
- Discuss the likelihood of successful VBAC, prediction models may be helpful.
- Review the risks associated with each of the available induction options if induction of labour is planned
- Offer written information (e.g., published guidelines from professional organizations, decision aids)
- Encourage the woman and her partner to participate in decision-making; respect patient autonomy
- Recommend resources that provide additional information when applicable
- Document the counselling and informed choice process, including the woman’s decision and a plan of care

Special Considerations

Number of previous Caesarean sections

Women with more than one previous CS delivery may still be candidates for TOLAC. A systematic review of 17 studies reported a rate of uterine rupture after one CS was 0.72 % and after 2 CS was 1.59%. Two large studies provide conflicting results. One showed no difference in rate of rupture \(^\text{15}\) while the other showed an increase from 0.9% to 1.8%. \(^\text{20}\)

The selection of candidates for a TOLAC depends on the clinical situation and should be re-evaluated throughout the pregnancy

Type of previous incision

Guise et al. undertook a comprehensive review of studies comparing the risks and outcomes of VBAC versus Caesarean section published between 1980 and 2004. \(^\text{21}\) In their analysis, they combined uterine rupture and dehiscence rates (7 studies from 1983 to 1999). They concluded there was little difference in the incidence of uterine rupture or dehiscence in women with vertical lower uterine segment incisions and women with transverse low segment incisions.
Type of closure of previous uterine incision

The risk of uterine rupture after an unlocked single-layer closure of the myometrium seems to be comparable with that after a double-layer closure. However, locked single-layer continuous suturing as opposed to a double-layer closure of the hysterotomy site may increase the risk of uterine rupture in women undergoing trial of labour in a future pregnancy.16

Interdelivery interval

A 2010 study by Bujold et al. showed an interdelivery interval shorter than 18 months should be considered a risk factor for uterine rupture. The population in this study consisted of women with singleton pregnancies and one previous CS who underwent a TOLAC at term.22

Hypertensive disorders of pregnancy

Data from a retrospective cohort study (n = 25 500) showed that women with gestational hypertension were less likely than normotensive women to choose a TOLAC and were also less likely to be successful. Women with gestational hypertension who underwent a TOLAC were no more likely to have uterine rupture than those who were normotensive.10

Twin pregnancy

Outcomes of TOLAC in women with a twin pregnancy are similar to those in women with a singleton pregnancy.2,5

Management of Labour

Studies have reported that women admitted with a more favourable cervical status in spontaneous labour have a 2-fold increase in the likelihood of VBAC compared with those with an unfavourable cervix.5,18

Antepartum consultation with an obstetrician may be advisable, depending on the clinical situation and local practice. The management of a TOLAC2,23 includes careful observation of labour progress—lack of progress with adequate contractions for 2 to 3 hours warrants reassessment of the mode of delivery—fetal well-being, and maternal well-being. Epidural or other analgesia may be used for usual indications.

Electronic fetal monitoring (EFM) should be used during active labour, because EFM tracing is the best marker of uterine rupture. There is no need to restrict activity (telemetry can facilitate mobility while allowing continuous monitoring).

Fetal Heart Rate Patterns and Uterine Rupture

Fetal heart rate (FHR) tracing abnormalities, especially complicated variables or late decelerations are the most commonly observed signs of uterine rupture.24,25

Leung et al. analyzed the FHR and uterine contraction pattern immediately prior to 78 cases of uterine rupture. Prolonged deceleration (alone or preceded by either severe late or variable decelerations) occurred in 71% of the cases of uterine rupture. In addition, prolonged deceleration occurred in 100% of the FHR tracings in which total fetal extrusion occurred.26 The appearance of recurrent late decelerations may be an early sign of impending uterine rupture.27
In some cases, it appears that there may be very little warning prior to the bradycardia in terms of fetal heart rate changes. However, a multicenter case–control study analysed FHR tracings in the two hours preceding uterine rupture during TOLAC and found that in the hour preceding uterine rupture, there are often significant FHR abnormalities. This leads us to consider the possibility of an earlier C-section when faced with an atypical or abnormal FHR tracing before the onset of terminal bradycardia jeopardizing maternal and fetal prognosis.

Induction and Augmentation of Labour

Induction of labour for maternal or fetal indications remains an option for women undergoing TOLAC. However, the potential increased risk of uterine rupture associated with any induction and the potential decreased possibility of achieving VBAC should be made clear to the parturient.

Induction of labour that requires cervical ripening is associated with a lower rate of successful VBAC and an increased risk of uterine rupture. This occurs mainly in women who have not had a prior vaginal delivery. Induction and augmentation of labour in women undergoing a TOLAC remains controversial and requires caution.

Recommendations regarding the induction or augmentation of labour during a TOLAC:

- Mechanical cervical ripening with a Foley catheter has been safely used before induction of labour in this clinical situation.
- The use of oxytocin is acceptable but careful surveillance is recommended as is consideration of the maximum dose to be administered. If oxytocin is used, then a low-dose protocol is recommended.
- Prostaglandins have been associated with increased risk of rupture and should not be used
- The timely availability of the human and physical resources to respond to an emergency is required.

All of these issues should be carefully considered and discussed with the woman before a management plan is finalized. Informed consent is essential before induction commences.

Signs and Symptoms of Uterine Scar Rupture

The signs and symptoms of uterine rupture include fetal heart rate abnormalities, vaginal bleeding, and acute onset of scar pain or tenderness (seldom masked by an epidural; this sign is neither sensitive nor specific).

The following may also occur:

- Hematuria
- Maternal tachycardia, hypotension, or hypovolemic shock
- Easier abdominal palpation of fetal parts
- Unexpected elevation of the presenting part
- Chest pain, shoulder tip pain, and/or sudden shortness of breath

A change in uterine activity (decrease or increase) is an uncommon and unreliable sign. A 2014 study of 97,028 births identified 52 uterine ruptures (0.05%): 25 complete and 27 partial. Most (89%) occurred in women with a previous Caesarean section. In complete ruptures, fetal heart rate abnormalities were the most frequent sign (82%), while the
complete triad of fetal heart rate abnormalities, pain, and vaginal bleeding was present in only 9%. The signs and symptoms of partial ruptures were very different; these were asymptomatic in 48% of the cases.  

Management of Uterine Rupture

This is an obstetrical emergency. Survival of the mother and fetus depends on:

- Prompt identification
- Rapid volume expansion and the use of blood products
- Timely access to a surgical team for surgical intervention
- Uterine repair or hysterectomy
- Prophylactic antibiotic therapy
- The attendance of a neonatal resuscitation team

Summary

Although some women who attempt a trial of labour will be unsuccessful, the overall maternal morbidity and mortality is lower than it is with elective repeat Caesarean section. Best evidence suggests that this is a reasonable and safe choice for the majority of women with previous Caesarean. While the incidence of uterine rupture is low, it is a serious complication with risk to both the parturient woman and the fetus. The increased risk of uterine rupture associated with a TOLAC underlines the need for careful selection of candidates, counselling, and management in labour. It is essential to discuss the risks of TOLAC and those of ERCS, including the effect that the mode of delivery will have on subsequent pregnancies, with women who have had a previous Caesarean section.
References


## Table of Contents

Chapter 13 Shoulder Dystocia ........................................................................................................................................ 289  
  Definition ................................................................................................................................................................ 289  
  Incidence ................................................................................................................................................................ 289  
  Risk Factors .............................................................................................................................................................. 289  
  Morbidity and Mortality ........................................................................................................................................... 290  
    Fetal/Neonatal ................................................................................................................................................. 290  
    Neonatal brachial plexus palsy (NBPP) ........................................................................................................... 290  
    Fractures of the clavicle and humerus ............................................................................................................... 291  
    Hypoxic ischemic encephalopathy and death ................................................................................................. 291  
    Maternal ........................................................................................................................................................... 291  
  Prediction and Prevention ....................................................................................................................................... 291  
  Diagnosis .............................................................................................................................................................. 292  
  Management .......................................................................................................................................................... 292  
  After the Baby is Delivered ................................................................................................................................... 295  
    Clinical ............................................................................................................................................................ 295  
  Risk Management .................................................................................................................................................... 295  
  Summary .............................................................................................................................................................. 296  
  ALARM Mnemonic .................................................................................................................................................. 300
Chapter 13
Shoulder Dystocia

Definition
Shoulder dystocia is the term used for the situation where one or both shoulders are trapped above the pelvic brim after the head has delivered. Almost always it is only one shoulder, the anterior one, that is impacted, against the symphysis pubis. Much rarer, both shoulders are stuck above the pelvic brim, with the posterior shoulder held up by the sacral promontory.

Clinically, shoulder dystocia is defined by the inability of the fetal shoulders to deliver spontaneously with maternal effort alone, or with gentle downward traction on the fetal head, after vaginal cephalic delivery. Additional obstetric maneuvers are needed to deliver the shoulders and body.

Incidence
The overall incidence is approximately 1% of all vaginal births.

The reported incidence was 1.4% in the United States and 0.6% in other countries. The incidence increases with increasing birthweight: approximately 5% for birthweight 4,000 – 4,500 grams; 10% for birthweight 4,500 – 5,000 grams; 20% for birthweight >5,000 grams. The incidence is 0.6% with spontaneous delivery and 2% with assisted vaginal delivery (AVB). Whether assisted vaginal delivery is an independent risk factor unrelated to fetal size, or whether the need for assisted vaginal delivery is a reflection of problems between fetal size and pelvic dimension, is unclear.

The incidence is 2-4 fold higher in women with pre-existing or gestational diabetes. This may reflect the increased incidence of fetal macrosomia, larger shoulders, and higher body fat compared with nondiabetic infants.

Risk Factors
The main risk factor is fetal macrosomia. However, about half of shoulder dystocia cases occur in women with no obvious risk factors, who have neither diabetes nor a baby weighing over 4,000 grams. The possibility of shoulder dystocia is present with every vaginal delivery. Certain factors should raise suspicion for shoulder dystocia.
Most cases of shoulder dystocia cannot be predicted or prevented. Clinical risk factors are not reliable predictors for shoulder dystocia or neonatal brachial plexus injury. The great majority of deliveries in the presence of risk factors do not result in shoulder dystocia.

**Morbidity and Mortality**

**Fetal/Neonatal**

Complications of shoulder dystocia include neonatal brachial plexus palsy (NBPP), fractures of the clavicle and humerus, hypoxic ischemic encephalopathy, and rarely fetal/neonatal death.

**Neonatal brachial plexus palsy (NBPP)**

About 10-20% of shoulder dystocia cases result in a brachial plexus injury. This can be a consequence of the clinician exerting downward lateral traction on the fetal head in an attempt to free the impacted anterior shoulder. This causes stretching of the cervical nerves in the neck; in the worst case the cervical nerves are ruptured or avulsed from the spinal cord. About 20% of babies with brachial plexus injury will have permanent impairment.

It has been traditionally thought that NBPP was invariably due to physician traction. While lateral downward traction during shoulder dystocia may be severe enough to injure the brachial plexus, it is clear that not all cases are due to excess traction by health care professionals. There are multiple reports of NBPP in the absence of shoulder dystocia. About half of brachial plexus injuries occur without a diagnosis of shoulder dystocia. The etiology of neonatal brachial plexus palsy (NBPP) is thought to be multifactorial.

Other factors such as uterine contractions and maternal expulsive efforts during the labour and delivery process and individual susceptibility likely also contributed to the development of the NBPP.
Fractures of the clavicle and humerus

Less serious complications are fractures of the clavicle or humerus, which can occur with forced adduction of the fetal shoulders and removal of the posterior arm respectively. Clavicular and humeral fractures generally heal well.

Hypoxic ischemic encephalopathy and death

The most feared complications for the fetus/baby is death or permanent brain injury as a result of asphyxia and/or hypovolemic shock. The risk increases with an increasing interval of head to body delivery and the number manoeuvres needed to reduce the shoulder. The duration of shoulder dystocia alone is not an accurate predictor of neonatal asphyxia or death. One study showed that the interval was less than 5 minutes in 47% of cases of death. Several factors may contribute to the risk of morbidity or mortality. In the study cited above, twenty five percent of cases had evidence of fetal compromise prior to delivery. Occlusion of the umbilical cord may lead to a drop in arterial pH although studies have shown the rate of decrease in pH may be slower that was traditionally thought. Occlusion of the umbilical vein or compression of the fetal chest may lead to sequestration of the fetal blood in the placenta and resultant hypovolemic shock. Finally it has been suggested that elevated intrauterine compared with atmospheric pressure impairs venous return from the fetal brain and can lead to local ischemia. These factors are likely responsible for the observation that hypoxic ischemic encephalopathy cause by shoulder dystocia often occurs with normal or near normal umbilical arterial cord gases.

Maternal

Perineal tears may be severe and can occur during appropriate management secondary to the manoeuvres required to free the impacted shoulder. There is an increased risk of obstetrical anal sphincter injury (OASI). Uterine atony and postpartum haemorrhage are common occurring in 10 – 20 percent of cases. Maternal symphyseal separation and lateral femoral cutaneous neuropathy have been reported likely secondary to hyperflexion of the hips.

Prediction and Prevention

While there are a number of recognized risk factors (Table 1), studies of these either alone or in combination are poor predictors of shoulder dystocia.

There is no clear strategy to predict or prevent shoulder dystocia. In a review of six studies that reported birth rate, 27% of shoulder dystocia occurred in babies weighting less 4000 gms, 39% of babies weighted 4000-4500 grams and 34% were over 4500 gms. Ultrasound estimates of fetal weight to identify macrosomia are commonly used. Ultrasound estimation of fetal weight is imprecise, but it is better than other methods in predicating fetal macrosomia.

One proposed way to avoid shoulder dystocia is to perform a cesarean section in cases of suspected fetal macrosomia. Depending on the threshold estimated fetal weight, this approach could result in the performance of up to 1,000 elective cesarean sections to prevent one permanent injury to the fetus. The ACOG recommendation is to reserve
elective cesarean section for nondiabetic women with estimated fetal weight more than 5 kilograms and for diabetic women more than 4.5 kilograms.\textsuperscript{1-24}

When there has been previous shoulder dystocia, the recurrence risk if vaginal delivery is allowed is 5 – 10\%.\textsuperscript{4} The estimated fetal weight, gestational age, maternal diabetes status, and the presence of prior neonatal injury should be evaluated before deciding the route of delivery.

Another approach suggested in the literature is to induce labour at 37-38 weeks in women thought clinically and by ultrasound to have a macrosomic fetus or in diabetic women. The rationale is if the baby is smaller, presumably there will be less shoulder dystocia. A randomized trial in nondiabetic women showed that a policy of early induction compared to expectant management led to less macrosomia (of more than 4 kilograms) and less shoulder dystocia (1\% compared to 4\%) but no difference in fetal outcomes.\textsuperscript{25} The cesarean rate was not increased with a policy of induction. A meta-analysis concluded that induction of labour for suspected macrosomia showed similar findings.\textsuperscript{26} Conversely, a meta-analysis of induction of labour in women with macrosomia and gestational diabetes did not show a reduction in shoulder dystocia.\textsuperscript{27} The American College of Obstetrics and Gynecology still discourages induction of labour for macrosomia.\textsuperscript{24}

**Diagnosis**

Shoulder dystocia can be suspected if the fetal chin delivers with difficulty and the head recoils against the perineum (‘turtle sign’). Spontaneous restitution does not occur. The mother fails to deliver the anterior shoulder with her own expulsive efforts during the next contraction.

It is recommended by some that an attempt at gentle downward (axial, longitudinal) traction on the head be made before a diagnosis of shoulder dystocia is made.\textsuperscript{1,2} This initial traction attempt is unnecessary and may itself injure the brachial plexus nerves.\textsuperscript{28} Without intervention after delivery of the head, the shoulders almost always deliver spontaneously without downward traction.\textsuperscript{13} There is no need to deliver the shoulders during the same contraction as the delivered head and most often, spontaneous delivery of the shoulders occurs with the next contraction. Provided the antecedent fetal heart rate has been normal, once the head is delivered, it is safe to wait for two or three minutes until the next contraction occurs and the mother again feels the urge to push.\textsuperscript{11,13,28}

**Management**

Given our inability to predict the occurrence of shoulder dystocia reliably, every delivery should be considered to have the potential for a shoulder dystocia. Therefore, a management protocol must be in place and well-known to all caregivers. In pregnancies where shoulder dystocia has a high likelihood, the woman and her support persons need to be prepared for the manoeuvres that may be used.

Avoid the 4 P’s. DO NOT!

1. Pull, on the head
2. Push, on the fundus
3. Pivot, or rotate, the head
4. Panic
The ALARMER mnemonic has been developed to assist in the appropriate and consistent management of this potential complication. External manoeuvres (McRoberts Manoeuvre and suprapubic pressure) are often attempted first as they are simple, rapid, and effective. If unsuccessful, internal manoeuvres (rotation or delivery of the posterior arm) or “all fours” position should be attempted.

When shoulder dystocia is recognized, it is important to instruct the woman to avoid pushing between contractions while manoeuvres to relieve the obstruction are carried out. This will facilitate manoeuvres and maximize fetal cerebral perfusion. Once a manoeuvre is completed, mother may be asked to push to determine if the manoeuvre resolved the problem.

A Ask for help
L Lift/hyperflex Legs (McRoberts manoeuvre)
A Anterior shoulder disimpaction (suprapubic pressure and/or Rubin manoeuvre)
R Rotation of the posterior shoulder
M Manual removal posterior arm
E Episiotomy
R Roll over onto “all fours”

Ask for help
- Set up unique paging protocols for obstetric emergencies to assure that appropriate equipment and personnel are available consistent with local circumstances. Get an assistant to help with the McRoberts manoeuvre. Summon a neonatal resuscitation team. An anaesthesiologist may help with necessary anaesthesia.
- The initial indication that shoulder dystocia may be present is often a turtle sign. When this occurs, it may be helpful to elevate the legs in McRoberts position and call for additional help while awaiting the next contraction. In many instances of a turtle sign, the infant will deliver spontaneously with the next contraction. If it does not, the team is poised to institute additional measures.

In normal birth there is usually a pause between delivery of the head and body. In a healthy baby, who has had a normal FHR during labour, this pause does not significantly affect fetal acidosis and may facilitate delivery of the shoulders. The drop in cord pH during this pause is minimal. With the following contraction, if the shoulders do not deliver spontaneously, the diagnosis of shoulder dystocia is made and additional manoeuvres should be instituted.

Maternal positioning
- Flatten the head of the bed, drop or remove the bottom of the bed.
- Bring the woman to the end of the bed

McRoberts manoeuvre
- Hyperflex both legs at the hips (McRoberts manoeuvre). The knees should be close to the mother’s shoulders to increase the anterior posterior diameter of the pelvis
- With the next contraction, ask the mother to push. Do not pull on the head
Anterior shoulder disimpaction

- Abdominal approach – apply suprapubic pressure with the heel of clasped hands from the posterior aspect of the anterior shoulder to dislodge it. Apply a steady pressure first and, if unsuccessful, apply a rocking pressure. It is necessary to know the position of the occiput so as to apply pressure from the correct side for greater effectiveness. It is also useful to have a stool in all delivery suites in order to facilitate this manoeuvre in the event of a shorter assistant.

- Vaginal approach – the index or middle finger, or the whole hand is advanced usually laterally and moved to the anterior shoulder to effect adduction of the anterior shoulder of the baby by applying pressure to the posterior aspect of the shoulder (i.e., the shoulder is pushed towards the chest, or pressure is applied to the scapula of the anterior shoulder) (Rubin manoeuvre).

These manoeuvres attempt to position the shoulders to utilize the smallest possible diameter of the shoulders through the largest diameter of the pelvis. Rotation is into the oblique diameter. Ask the mother to push. Do not pull on the head.

Rotation of the posterior shoulder

Woods’ manoeuvre is a screw-like manoeuvre. Pressure is applied with the whole hand in the vagina to the anterior aspect of the posterior shoulder and an attempt is made to rotate the posterior shoulder to an anterior position. Alternatively, one can rotate the posterior shoulder in the opposite direction with pressure on the posterior aspect of the posterior shoulder. Success of this manoeuvre allows easy delivery of the anterior shoulder once it is past the symphysis pubis. In practice, the anterior shoulder disimpaction manoeuvre and Woods’ manoeuvre may be done repetitively to achieve disimpaction of the anterior shoulder.

Manual removal of the posterior arm

The arm is usually flexed at the elbow. If it is not, pressure in the antecubital fossa can assist with flexion. The hand is grasped, swept across the chest and delivered. If the posterior hand cannot be reached, delivery of the posterior shoulder using axillary traction is usually successful. With either technique fracture of baby’s humerus is common; however, fractures heal whereas brachial plexus palsies may be permanent.

Episiotomy

Episiotomy is an option that may facilitate the Woods’ manoeuvre or manual removal of the posterior arm by creating more room for the accoucheur’s hand. However, shoulder dystocia is not caused by obstructing soft tissue. Therefore, performing an episiotomy will not, on its own, relieve a shoulder dystocia.

Roll over to “all fours” position

This manoeuvre may be considered early in the management of shoulder dystocia.

Moving the woman onto “all fours” with the back arched appears to increase the effective pelvic dimensions, allowing the fetal position to shift (Gaskin’s manoeuvre). This may free the impacted shoulder. With gentle downward pressure on the posterior shoulder, the anterior shoulder may become more impacted (with gravity) but will facilitate the freeing up of the posterior shoulder. This position may also allow easier access to the posterior shoulder for rotational manoeuvres or removal of the posterior arm. In one series, as a first line manoeuvre, this manoeuvre was successful in 29% of cases.
The “all fours” position may be used in the presence of epidural analgesia unless the degree of motor block makes rolling over and maintaining the position impossible.

**Further Considerations**

The initial steps of McRoberts manoeuvre and suprapubic pressure will resolve about a quarter of cases. Even if McRoberts manoeuvre is unsuccessful, the hyperflexed position of the legs will often let the posterior shoulder slide deeper in the sacral concavity, making rotation manoeuvres and delivery of the posterior arm easier. McRoberts manoeuvre and suprapubic pressure, even if successful alone, are associated with brachial plexus injury about 10% of the time, \[35, 36\] possibly a result of the initial gentle downward traction or subsequent traction after the legs are hyperflexed. It has been suggested that the first manoeuvre should be delivery of the posterior arm or shoulder. \[32\]

If the above steps have not worked, one can repeat the manoeuvres under general anaesthesia. The above manoeuvres are most likely to fail if neither shoulder is in the pelvic concavity, in other words, the posterior shoulder is also out of the pelvis. In this situation one may have to resort to cephalic replacement. \[37, 38\] or symphysiotomy. \[39, 40\]

Brachial plexus palsy can occur regardless of the procedures used to disimpact the shoulders because all maneuvers can increase the degree of stretch on the brachial plexus. \[1, 2, 35, 36\] There can be significant perinatal morbidity even when shoulder dystocia is managed appropriately. The most important principal in the management of shoulder dystocia is to have a rehearsed, methodical, efficient (not panicked or rushed) approach with a sequence of manoeuvers. These are supported by the extended care team and practiced in simulation

**After the Baby is Delivered**

**Clinical**

**Mother**

Remember the SIGNIFICANT risk of maternal injury (tears) and postpartum hemorrhage. Actively manage the third stage. Inspect for and repair lacerations.

**Newborn**

It is recommended to follow NRP guidelines in appropriate care of the baby. The newborn should be carefully examined to ensure there is no evidence of injury.

**Risk Management**

Both HIROC and the CMPA have identified management of shoulder dystocia as one of the 5 top high risk areas for litigation. (Delivery in focus: Strengthening Obstetrical care in Canada. A 10 year review of CMPA and HIROC data, 2018)
Preparation

Regular training in the management of emergencies (such as shoulder dystocia) results in a sustained improvement in team performance during the actual events. Hands-on training using a mannequin is better than using a video demonstration. Simulation-based training has been shown to reduce the risks associated with shoulder dystocia.

The implementation of a shoulder dystocia protocol has been found to decrease the diagnosis of BPI at delivery and at neonatal discharge.

Discussion, Documentation and Debriefing

1. Document and describe. Using a preformatted documentation sheet or checklist will aid in proper documentation. Include in your documentation:
   a. sequence of all manoeuvres used, including which shoulder was impacted
   b. episiotomy, if done
   c. individuals present in the room
   d. the time of delivery of the head, onset of next contraction and delivery of the body
   e. condition of the newborn
   f. whether cord gases sent

2. Debrief with the mother and family about what occurred and what management steps were taken. Document your discussion.

3. Discuss and debrief with the clinical team.

Summary

1. Don’t panic.
2. Be prepared. Develop and practice a standard management protocol (regular and repeated emergency drills). The ALARMER mnemonic is helpful.
3. Prepare the woman and her partner when the potential for shoulder dystocia appears high.
4. Do not pull on the head, neither to make the diagnosis of shoulder dystocia nor after McRoberts’ manoeuvre and suprapubic pressure are used. There should be no traction on the baby’s head.
5. If the baby is depressed, do not clamp the cord immediately. Drying, evaluation and stimulation of the baby for the initial 30 seconds can be done with the cord intact. If ventilation is required at 30 seconds of age and PPV can be instituted with the cord intact, cord clamping should be delayed another 30 – 60 seconds to optimize placental auto transfusion.
6. Document events and explain to mother and partner.
References


Appendix A

ALARM Mnemonic

A  ASK for help
L  LIFT / hyperflex LEGS

Lift: McRoberts Manoeuvre

- McRoberts manoeuvre
- flexion of thighs on abdomen
- requires assistance

Used with permission of Salus Global Corporation
A **ANTERIOR** shoulder disimpaction

**Anterior disimpaction - 1) Suprapubic pressure**

*Used with permission of Salus Global Corporation*
Directed from side of fetal back
- CPR-type motion
- NO fundal pressure
Anterior disimpaction - 2) Rubin manoeuvre

Used with permission of Salus Global Corporation
R旋转

后肩旋转 - 步骤 1

- 前肩部压力
- 可以与前部脱位操作结合使用
- 无宫体压力

图示：后肩旋转步骤1。

• 前肩部压力
• 可以与前部脱位操作结合使用
• 无宫体压力
Rotation of posterior shoulder - Step 2
Rotation of posterior shoulder – Step 3

- may be repeated if delivery not accomplished by Steps 1 & 2
**M** MANUAL removal posterior arm

**Manual removal of posterior arm**

- splint humerus
- pressure in antecubital fossa to flex arm
- sweep arm over chest
- grasp wrist / forearm
- deliver arm

Used with permission of Salus Global Corporation

**E** EPISIOTOMY

**R** ROLL over onto ‘all fours’
# Table of Contents

Chapter 14 Breech Presentation and Delivery .............................................................................................................. 309

Definitions ........................................................................................................................................................................ 309
Incidence ........................................................................................................................................................................ 310
Morbidity and Mortality ...................................................................................................................................................... 310
Etiology and Risk Factors ................................................................................................................................................... 310
Diagnosis of Non-Cephalic Presentation .......................................................................................................................... 310

Management of Breech ...................................................................................................................................................... 311
External Cephalic Version .................................................................................................................................................... 311
Timing of ECV ....................................................................................................................................................................... 311
Predictors for successful ECV ............................................................................................................................................... 313
ECV Procedure ...................................................................................................................................................................... 313
Procedures that may Facilitate Turning the Breech ........................................................................................................... 314

The Limitations Of The Term Breech Trial .......................................................................................................................... 316
Vaginal Breech Delivery ........................................................................................................................................................ 317
Setting and Consent ............................................................................................................................................................... 323
Risks and Complications ....................................................................................................................................................... 323
Preparation and Labour Management .................................................................................................................................. 324
Delivery Technique ................................................................................................................................................................. 324

Follow-Up ............................................................................................................................................................................ 326
Care After Breech Delivery Should Include .......................................................................................................................... 326
Documentation: Breech Delivery .......................................................................................................................................... 326

Summary .............................................................................................................................................................................. 327
GETHIPPOS Mnemonic ......................................................................................................................................................... 331
Chapter 14
Breech Presentation and Delivery

Definitions
When the buttocks of the fetus enter the maternal pelvis before the head, the presentation is termed a breech.

There are three different types of Breech:

**Complete Breech:** Fetal hips flexed, knees flexed (foot may be adjacent to or just below buttocks) (5% to 10% of breech presentations)

**Footling or Incomplete:** One or both fetal hips extended, foot or knee presenting (10% to 30% of breech presentations)

**Frank Breech:** Fetal hips flexed, knees extended (50% to 70% of breech presentations)

Figure 1. Types of Breech Presentation

Used with permission of Salus Global Corporation
Incidence

Breech presentation occurs in 3% to 4% of all pregnancies reaching term. The earlier the gestation, the higher the percentage of breech fetuses. At 28 weeks’ gestation, approximately 24% of fetuses are in the breech presentation.

Morbidity and Mortality

Breech presentation is associated with an increased frequency of perinatal mortality and morbidity due to prematurity, congenital anomalies (which occur in 6.3% of all breech presentations compared with 2.4% of non-breech presentations), cord prolapse and birth trauma/asphyxia. A 2009 study by Andersen et al. indicated that breech presentation alone puts the fetus at higher risk for cerebral palsy than does cephalic presentation. However, vaginal breech delivery itself was not associated with risk for cerebral palsy.

Etiology and Risk Factors

As term approaches, the fetus is usually accommodated in the uterine cavity in a longitudinal lie with the vertex presenting. Any factor that precludes or makes it more difficult for the fetus to be accommodated in the uterus as a vertex presentation is a risk factor for breech. These factors include:

- Prematurity
- Oligohydramnios
- Uterine anomalies (i.e., septate, bicornuate, or didelphic uterus) or tumours (e.g., large fibroids)
- Placenta implanted low in the uterus or placenta previa
- Fetal anomalies (e.g., anencephaly or hydrocephaly)

Other risk factors for breech include previous breech delivery or idiopathic causes. If either the pregnant woman or the father of her fetus was a breech delivery, then the likelihood that the fetus will be breech is doubled.

Diagnosis of Non-Cephalic Presentation

Performing Leopold's manoeuvres during third trimester prenatal examinations will make the diagnosis in the majority of cases. If there is any doubt, vaginal examination or ultrasound may be performed to confirm the presentation. An abdominal X-ray may be used to confirm the diagnosis if ultrasound is unavailable.
Management of Breech

Term breech management involves 3 options: external cephalic version, Caesarean section, or vaginal breech delivery.

External Cephalic Version

External cephalic version is a procedure whereby a fetus is turned in utero from a non-cephalic to a cephalic presentation by manipulation of the maternal abdomen.

A meta-analysis of 5 RCTs comparing ECV at term with no attempt at ECV showed a significant reduction in non-cephalic births (RR 0.38; 95% CI, 0.18 to 0.80) and CS (RR 0.55; 95% CI 0.33 to 0.91). There was no significant effect on perinatal mortality (RR 0.51; 95% CI 0.05 to 5.54) or other measures of perinatal outcome. It is therefore recommended that all women with breech presentation at or beyond 36 weeks’ gestation, who are appropriate candidates, be offered an ECV. A recent trial compared obstetrical outcomes for women with a cephalic presentation at birth resulting from successful ECV to those resulting from spontaneous cephalic version. It showed that women with a cephalic-presenting fetus at birth as a result of ECV are not at greater risk of obstetrical interventions compared with women whose fetuses turned spontaneously.

Timing of ECV

The ideal time to carry out ECV has been the subject of debate. A large multicentre RCT (n = 1543) compared ECV at between 34 and 36 weeks’ gestation with ECV after 37 weeks’ gestation. When ECV was performed at 34 to 36 weeks:

- Fewer fetuses remained breech at delivery (51% vs. 59%)
- There was
  - A 4% absolute reduction in delivery by CS (52% vs. 56%)
  - A 2% absolute increase in preterm birth < 37 weeks: (6.5% vs. 4.5%)
  - No difference in neonatal morbidity
  - No perinatal deaths related to ECV

Waiting to perform ECV allowed spontaneous version to occur more often (25% vs. 14%), whereas earlier ECV allowed time for repeat attempts if the initial attempt was unsuccessful. Approximately one quarter of repeat attempts in this trial were successful.

ECV should not be attempted before 34 weeks’ gestation as most breech fetuses will turn prior to that gestation, and, if emergency delivery is required for complications, neonatal morbidity is high. After 34 weeks, the timing of ECV can be decided by a woman and her care provider on the basis of a discussion of the risks and benefits above.

Although the success rate of ECV declines as gestational age advances, ECV may be attempted up until the time of labour. ECV may even be attempted in early labour if membranes are intact and the uterus remains relaxed long enough between contractions.
Prerequisites

1. Singleton pregnancy
2. Gestation > 34 weeks
3. No contraindication to labour
4. Fetal well-being established before procedure (i.e., non-stress test or biophysical profile)
5. Amniotic fluid volume adequate
6. Availability of ultrasound
7. Position of fetus known before procedure
8. Facilities and personnel available for immediate Caesarean section

Contraindications

Absolute

1. Any contraindication to labour e.g. placenta previa, or abnormal or atypical fetal heart pattern, compromised fetus, active genital herpes simplex virus infection, previous classical uterine incision, or other uterine surgery that would increase the risk of uterine rupture (hysterotomy, myomectomy, full thickness uterine wall incision, etc.)
2. Antepartum hemorrhage
3. Some major fetal anomalies
4. Multiple gestation (except delivery of second twin)
5. Ruptured membranes

Relative

1. Oligohydramnios
2. Hyperextension of the fetal head
3. Two or more previous Caesarean sections
4. Morbid obesity
5. Active labour
6. Uterine malformation
7. Fetal anomaly

The relative contraindications listed above negatively affect the likelihood of ECV being successful and need to be considered when deciding whether or not to attempt ECV. It has also been shown that ECV is somewhat less likely to be successful with an anterior placenta, although this is not statistically significant.10, 11

ECV appears to be safe in women who have had a Caesarean section with a low transverse uterine incision in a previous pregnancy.12 There are very limited data on the safety of ECV in women who have had 2 or more Caesarean sections.
Risks

1. Placental abruption (0.4% to 1%)\textsuperscript{9,13,14}
2. Rupture of the membranes and subsequent possible cord prolapse
3. Initiation of labour
4. FHR abnormalities, the most common being transient bradycardia (1.1% to 47%). \textsuperscript{13,17} (Note that fetal bradycardia necessitating an emergency Caesarean section is uncommon at 0.5%. \textsuperscript{18})
5. Alloimmunization/maternal-fetal hemorrhage (0% to 5%)\textsuperscript{1}

A 2009 study by Clock et al. did not find an increase in intrapartum risk of delivery by Caesarean section after successful ECV.\textsuperscript{13} Intrauterine death is rare, and evidence suggests that the rate is not increased by this procedure.\textsuperscript{7}

Predictors for successful ECV

Clinical Predictors\textsuperscript{20,21}

- Multiparity
- Lack of engagement
- Relaxed uterus
- Fetal head palpable abdominally
- Low maternal weight

Ultrasound Predictors\textsuperscript{22}

- Posterior placenta
- Complete breech
- Amniotic fluid index >10 cm

ECV Procedure

Obtain informed consent (this should be documented and ideally should include a written signed consent). The patient should be informed that:

- Successful ECV will reduce the chance of a CS (success varies widely from 30% to 80%)\textsuperscript{17,23,26}
- Sedation and tocolysis may be used
- The procedure may be uncomfortable
- There are risks to the procedure (see above)

The procedure must be performed in a facility with the ability to carry out immediate intervention, including a CS, if needed.

A non-stress test or biophysical profile should be carried out and must be normal before the procedure is started.
An ultrasound examination should be performed to confirm the fetal position. Real-time ultrasound examination should also be performed intermittently during the procedure to check progress and monitor the fetal heart rate.

The abdomen may be lubricated with ultrasound gel or powder to make the procedure easier.

In the initial ECV attempt, the direction of rotation should be so that the fetus “follows its nose” (i.e., a forward roll). Proceed as follows:

- Dislodge the fetal buttocks from the maternal pelvis, pushing upwards and then laterally
- Grasp the fetal head and direct it downwards
- Slowly rotate the fetus by pushing upwards and to the side of the fetal back with the hand holding the buttocks, at the same time guiding the head downwards and to the opposite side
- When the fetal head reaches a lower level than the maternal buttocks, manoeuvre the head over the pelvic inlet
- If the forward roll attempt fails, a backward flip (i.e., the opposite direction) may be attempted

An assistant may be helpful to facilitate the ECV.

Stop the procedure if the woman is too uncomfortable or the fetal heart rate is abnormal. Most atypical and abnormal FHR patterns will resolve. If the FHR doesn't recover with intrauterine resuscitation, an emergency CS must be done.

Fetal surveillance (i.e., a non-stress test) is continued for a minimum of 20 minutes after an attempted ECV, whether or not the ECV is successful. If the version was successful, the woman should continue to receive antenatal care and await labour.

If version is not successful, discuss with the woman appropriate arrangements for her ongoing antenatal care and choice of delivery method.

Administer Rh immunoglobulin 300 mcg to unsensitized Rh-negative women. Routine assessment with the Kleihauer-Betke test for the possibility and degree of maternal-fetal bleed is not necessary since it has been shown that only 0.08% of bleeds with ECV will be greater than 30 mL (300 mcg of Rh immunoglobulin will prevent sensitization following a bleed up to a 30 mL bleed). 

Advise the woman to report any abdominal pain, symptoms of labour, bleeding, fluid leakage, fever, or decreased fetal movements.

Procedures that may Facilitate Turning the Breech

Tocolytics

Evidence for the use of tocolytics for improving the success of ECV is limited. A 2012 Cochrane review concluded that beta-mimetic drugs are superior to placebo, nifedipine, or nitroglycerin; however, these are not used in Canada and have considerable adverse maternal effects. The Cochrane authors concluded there was enough evidence on nitroglycerin to recommend against its use.
**Epidural or spinal analgesia**

Several trials have now shown the benefit of spinal or epidural analgesia in improving the success of ECV. A 2011 meta-analysis of 6 RCTs (n = 508) showed improved success with ECV under regional analgesia (60% vs. 35%), and a trend toward fewer Caesarean sections (48% vs. 59%). More recent trials have confirmed the success of this meta-analysis.

**Moxibustion**

Moxibustion is a traditional medicine technique involving the burning of sticks or cones of the herb moxa (*Artemisia vulgaris*) close to the pressure point on the fifth toe in order to induce a warming sensation that in turn has been suggested to promote turning of the baby to cephalic presentation. A Cochrane review concluded that there is insufficient evidence to support its use and larger trials are needed to evaluate it adequately.

**Postural management**

Managing posture (e.g., knee–chest) to promote cephalic version has been assessed in a Cochrane systematic review of RCTs and has not been shown to be effective. All the trials were small, and no effect on the rate of non-cephalic births from postural management was detected between the intervention and control groups (5 RCTs, n = 392, RR 0.95; 95% CI 0.81 to 1.11). Similarly, there were no differences detected for CS (4 RCTs, n = 292, RR 1.07; 95% CI 0.85 to 1.33).

**Controversies in The Management of the Breech Presentation**

The management of the breech presentation continues to provoke controversy. A policy of elective CS for all breech presentations became popular in the 1990s. In a 10-year review of CS trends in Canada, from 1979–1980 to 1988–1989, Caesarean sections performed because of breech presentation increased by 66%. This policy was instituted without appropriate supporting evidence.

In 2000, the Term Breech Trial (TBT) was published. This trial was a multicentre RCT in which women with breech singleton pregnancies at term were randomized to either a planned CS or a planned vaginal birth. The trial was stopped early after the review of an interim analysis showed a large reduction in risk of perinatal or neonatal mortality or serious neonatal morbidity with planned CS. The final results (developed and developing countries) showed the rate of perinatal or neonatal mortality or serious neonatal morbidity to be 1.6% in the planned Caesarean group and 5.0% in the planned vaginal birth group. Perinatal death was also reduced in the planned Caesarean group (0.3% vs. 1.3%; RR 0.23; 95% CI 0.07 to 0.81).

A Cochrane review of planned CS for term breech delivery includes the findings from the Term Breech Trial and 2 prior, much smaller trials, and confirms these findings. In the total sample (worldwide), perinatal or neonatal death (excluding fatal anomalies) was reduced overall (RR 0.29; 95% CI 0.10 to 0.86) with a policy of planned CS.

The reviewed trials indicate that a policy of planned CS compared with planned vaginal delivery was associated with a decrease in perinatal or neonatal death and/or neonatal morbidity. However, among survivors, there was no significant difference in outcomes at age 2 and as the long-term outcome following perinatal morbidity appeared good, the most
relevant outcome is the reduction in perinatal and neonatal death. This reduction was found mostly in developing countries with a baseline perinatal mortality > 20/1000. There was no significant difference in perinatal or neonatal mortality in developed countries with low baseline perinatal mortality rates.

The Limitations Of The Term Breech Trial

The major limitations of the TBT are critical to estimating the true risk of labour for a breech fetus. They can be grouped as follows:

1. **Inadequate case selection and intrapartum management**
   Pre- or early labour ultrasound was not required, which may have allowed fetuses with growth restriction due to placental insufficiency to go undetected. At least 7 of the trial’s 16 perinatal deaths were in growth-restricted fetuses. Continuous electronic fetal monitoring was also not required, and was used with only one third of fetuses. The trial protocol allowed cervical dilatation to be as slow as 0.5 cm/hr in the first stage of labour and allowed the second stage to last for up to 3.5 hours. The findings of the TBT therefore suggest that the following strategies may increase the safety of term breech deliveries: ultrasound estimation of fetal weight to detect abnormal fetal growth, fetal head attitude and type of breech presentation, and close attention to progress of labour.

2. **Maternity units with markedly varying levels of skill within the group**
   Although a practitioner experienced in vaginal birth was expected at every delivery, a licensed obstetrician was not present at 13% of births in the planned vaginal birth group versus 2% in the planned Caesarean section group, and there was a high degree of crossover in the trial: 10% of women randomized to planned CS delivered vaginally.

3. **Short-term morbidity used as a surrogate marker for long-term neurological impairment**
   Despite the large difference in short-term outcome, even with the limitations in the TBT, women had a 97% chance of having a neurologically normal 2-year old, regardless of planned mode of delivery.

In 2006, Goffinet et al. published the PREMODA study, a multicentre descriptive study 4 times larger than the TBT. Prospective data were collected from 8105 women in 174 centres in France and Belgium, using the same short-term combined outcome of perinatal mortality or serious neonatal morbidity as the TBT. Although not strictly comparable, the PREMODA outcomes are in contrast to those of the TBT. There was no difference in perinatal mortality (0.08% vs. 0.15%) or serious neonatal morbidity (1.6% vs. 1.45%) between a trial of labour and planned CS. The only difference in outcome was a 0.16% incidence of 5-minute Apgar score < 4 in the trial of labour group versus 0.02% in the planned CS group. The PREMODA study is 8 times larger than the low-perinatal-mortality subset of the TBT, and therefore provides a robust estimate of the risk of a cautious breech trial of labour in a modern, well-supported obstetrical unit.

In light of these studies, the SOGC published new guidelines for vaginal delivery of breech presentation in June 2009. Vaginal breech birth has been associated with a higher risk of perinatal mortality and short-term neonatal morbidity than elective Caesarean section. However, the short-term neonatal morbidity nearly always resolves, and any increase in perinatal mortality is small. Although perinatal mortality in developed countries was not significantly different between the arms of the Term Breech Trial, 2 delivery-related perinatal deaths occurred in 511 labours versus none in the planned
CS group, a point estimate of 1/250. In the PREMODA study, there were no delivery-related perinatal deaths in 2502 labours. Therefore, careful case selection and labour management in a modern obstetrical setting may achieve a level of safety similar to elective Caesarean section. Planned vaginal delivery is reasonable in selected women with a term singleton breech fetus. The long-term neurological outcomes of infants do not differ by planned mode of delivery even in the presence of serious short-term neonatal morbidity.

Vaginal Breech Delivery

The SOGC guideline makes the following recommendations for women who choose a trial of labour for a vaginal breech delivery:

Labour Selection Criteria

For a woman with suspected breech presentation, pre- or early labour ultrasound examination should be performed to assess type of breech presentation, fetal growth and estimated fetal weight (EFW), and attitude of fetal head. If ultrasound is not available, Caesarean section is recommended.

Contraindications to planned vaginal breech birth

- Cord presentation
- Footling breech presentation (one or both hip(s) extended)
- Hyperextended fetal head
- Clinically inadequate maternal pelvis
- Fetal anomaly incompatible with vaginal delivery
- Fetal macrosomia ( >4000g)
- Fetal growth restriction (EFW <2500g)
  - Fetal metabolic acidosis in labour due to placental factors puts the fetus at elevated risk of asphyxia if delay occurs during delivery. It is very important, therefore, to rule out significant fetal growth restriction before delivery. Significant cord compression (with variable FHR decelerations) leading up to delivery can also cause metabolic acidosis and predisposes the fetus to compromise if there is delay during delivery. Any fetus at elevated risk of metabolic acidosis, or with evidence of acidosis in labour, is more safely delivered by CS.

Vaginal breech delivery can be offered when the EFW is between 2500 g and 4000 g.

Labour Management

- Clinical pelvic examination should be performed to rule out significant pelvic contraction. Radiologic pelvimetry is not necessary for a safe trial of labour; good progress in labour is the best indicator of adequate fetal-pelvic proportions
Continuous electronic fetal heart monitoring is recommended in the first stage and mandatory in the second stage of labour. Membranes should be kept intact as long as possible. However, when membranes rupture, immediate vaginal examination is recommended to rule out prolapsed cord.

- In the absence of adequate progress in labour, Caesarean section is advised.
- Induction of labour is not recommended for breech presentation. Oxytocin augmentation is acceptable in the presence of uterine dystocia during the first and second stage of labour. In the PREMODA study, oxytocin was routinely administered during the second stage of labour to ensure good uterine activity.\(^{37}\)
- A passive second stage without active pushing may last up to 90 minutes, allowing the breech to descend well into the pelvis. Once active pushing commences, if delivery is not imminent after 60 minutes, Caesarean section is recommended.
- The active second stage of labour should take place in or near an operating room with equipment and personnel available to perform a timely Caesarean section if necessary.
- A health care professional skilled in neonatal resuscitation should be in attendance at the time of delivery.

**Delivery Technique**

- The health care provider for a planned vaginal breech delivery needs to possess the requisite skills and experience.
- The requirements for emergency Caesarean section, including availability of the hospital operating room team and the approximate 30-minute timeline to commence a laparotomy, must be in accordance with the recommendations of the SOGC policy statement, “Attendance at Labour and Delivery”\(^{39}\).
- The health care provider should have rehearsed a plan of action and should be prepared to act promptly in the rare circumstance of a trapped after coming head or irreducible nuchal arms: symphysiotomy or emergency abdominal rescue can be lifesaving.
- Total breech extraction, where a hand is reached into the uterus and the fetus delivered, should only be used for the second twin and is inappropriate for term singleton breech delivery.
- Effective maternal pushing efforts are essential to safe delivery and should be encouraged.
- At the time of delivery of the after coming head, an assistant should be present to apply suprapubic pressure to favour flexion and engagement of the fetal head.
- Spontaneous or assisted breech delivery is acceptable. Fetal traction should not be used, and fetal manipulation must be applied only after spontaneous delivery to the level of the umbilicus.
- Nuchal arms may be reduced by the Bickenbach or Løvset’s manoeuvres (Figures 2 & 3).
- The fetal head may deliver spontaneously, with the assistance of suprapubic pressure (Bracht manoeuvre), by Mauriceau-Smellie-Veit manoeuvre (Figure 4), or with the assistance of Piper forceps (Figure 5).
Figure 2. Bickenbach reduction of nuchal arms

**A.** Release of the posterior arm. The legs are grasped at the ankles and raised briskly until the fetal body is near-vertical. A vaginal hand then reaches into the sacral hollow and sweeps the humerus from posterior to anterior across the fetal chest. **B.** Release of the anterior arm. The fetal body is then lowered briskly toward the floor until the axilla appears at the introitus. A vaginal hand reaches behind the pubic symphysis and sweeps the humerus from posterior to anterior across the fetal chest.
Figure 3. Løvset’s manoeuvre to reduce nuchal arms

A. Release of the posterior arm. The fetus is grasped by the bony pelvis with 2 hands and initially raised towards the maternal pubic symphysis. Turning the fetal torso while lowering it allows the fetal humerus to be swept out under the pubic symphysis.

B. Release of the first arm brings the other arm posterior. Rotating the fetal trunk back through a sacrum-anterior position to the other side allows the remaining arm to similarly be swept out under the pubic symphysis.
Note that as the fetal head is being delivered, flexion of the head is maintained by suprapubic pressure provided by an assistant, and simultaneously by pressure on the maxilla (inset) by the operator as traction is applied.\textsuperscript{3}
Figure 5. Piper forceps for delivery of the after coming head

Note the direction of movement shown by the arrows. The fetal body is elevated using a warm towel and the left blade of the forceps is applied to the after coming head. The right blade is applied with the body still elevated. Forceps delivery of the after coming head.3

Used with permission of Salus Global Corporation
Setting and Consent

- In the absence of a contraindication to vaginal delivery, a woman with a breech presentation should be informed of the risks and benefits of a trial of labour and elective Caesarean section, and informed consent should be obtained. A woman’s choice of delivery mode should be respected.
- The consent discussion and chosen plan should be well documented and communicated to labour-room staff.
- Hospitals offering a trial of labour should have a written protocol for eligibility and intrapartum management.
- Women with a contraindication to a trial of labour should be advised to have a Caesarean section. Women choosing to labour despite this recommendation have a right to do so and should not be abandoned. They should be provided the best possible in-hospital care.
- Theoretical and hands-on breech birth training simulation should be part of basic obstetrical skills training programs such as ALARM and MORE to prepare health care providers for unexpected vaginal breech births.

Risks and Complications

- Low 1-minute Apgar scores are commonly due to elevated CO₂ that accumulates during cord compression at delivery. The CO₂ is easily excreted through adequate neonatal ventilation.
- Entrapment of the fetal head by an incompletely dilated cervix occurs uncommonly; however, it occurs more frequently with preterm fetuses (< 32 weeks), since the head is larger than the body. Adequate power from above (maternal pushing efforts and suprapubic pressure) is usually enough to complete delivery of the head, but Dührssen incisions of the cervix are sometimes necessary. A Zavanelli-type manoeuvre to push the fetus back up into the uterus followed by a Caesarean section has been performed successfully in rare circumstances but must be considered a last resort.
- Nuchal arms, defined as one or both arms stuck between the fetal neck and the maternal pubic bone, occur in 0% to 5% of vaginal breech deliveries and in 9% of breech extractions. Complications of nuchal arms include brachial plexus injury and fractured humerus. Most occurrences of nuchal arms can be prevented by refraining from the use of traction on the fetus during delivery. Nuchal arms can be released using the Lovset’s or Bickenbach manoeuvres (Figures 2 and 3).
- A case series published in 1976 reported cervical spine injury in fetuses with a hyperextended head delivered vaginally. Ballas and Toaff reported 20 cases of hyperextended necks, defined as an angle of extension greater than 90° (“star-gazing”), discovered on antepartum radiographs. Of the 11 fetuses delivered vaginally, 8 (73%) sustained complete cervical spinal cord lesions, defined as either transection or nonfunction. This forms the basis of recommendations to rule out hyperextension of the fetal head; however, most of these fetuses were preterm, some may have had existing neurological abnormalities, and the predominant delivery technique at the time involved traction.
- Cord prolapse occurs in approximately 5% of vaginal breech deliveries. Cord prolapse risk depends on the type of breech (frank 1%, complete 5%, or footling 10% to 25%). Cord prolapse is also more common in multiparous women (6%) than in primigravid women (3%). Electronic fetal monitoring in labour and
immediate vaginal examination upon membrane rupture will detect cord prolapse in a timely fashion. In a monitored setting with ready access to CS, the risks of cord prolapse are almost always mitigated.

**Preparation and Labour Management**

A written vaginal breech labour protocol may assist in ensuring all important components are in place.

- If possible, and if time permits, use ultrasound assessment (or recent report) to confirm the lie and presentation, assess the head position, get an EFW, assess amniotic fluid volume, confirm placental location, and rule out major congenital anomalies such as hydrocephalus
- Ensure presence of a physician experienced with breech delivery
- Ensure an anaesthesiologist is available or in house
- Call for an anaesthesiologist
- Ensure presence of clinicians experienced in newborn resuscitation
- Ensure presence of experienced nursing staff
- Employ continuous electronic fetal surveillance. A fetal scalp ECG clip applied to the fetal buttocks can improve FHR detection as the breech descends.
- Monitor labour progress using a partogram. If there is significant delay in the first or second stage, CS is advised.
- Oxytocin augmentation is acceptable for poor uterine contractions; however, if progress is slow despite strong contractions (clinically or by intrauterine pressure catheter), oxytocin is inadvisable.
- Empty maternal bladder just before delivery
- Ensure availability of forceps for the after coming head
- Ensure OR staff are in-house during active second stage for possible emergency CS

**Delivery Technique**

1. Total breech extraction should not be performed to deliver a singleton breech.
2. The necessity of effective pushing in the second stage of labour should be explained to the woman.
3. Analgesia should be adequate; however, dense epidural analgesia will hamper maternal pushing efforts.
4. Spontaneous descent and expulsion to the umbilicus should occur with maternal pushing only: **DO NOT PULL ON THE BREECH!**
5. Rotation to the sacrum anterior position usually occurs spontaneously and is desired. If the fetus appears to be rotating to a sacrum posterior position, grasp the fetal pelvis and gently rotate to sacrum anterior.
6. Episiotomy may be considered once the anterior buttock and anus are “crowning.”
7. Spontaneous delivery of the entire breech fetus is desirable and is common with adequate maternal pushing efforts and suprapubic pressure, if needed. However, assisted breech delivery is acceptable if there is a delay in delivery, and the following manoeuvres may be required:
   a. Pinard’s manoeuvre to deliver the fetal legs may be considered once the popliteal fossae are visible.
Figure 6. Pinard’s manoeuvre

Pinard’s manoeuvre is accomplished by inserting 2 fingers along 1 leg to the knee, which is then pushed away from the midline (abducted) at the same time as flexing the leg at the hip. This causes spontaneous flexion of the knee and delivery of the foot.42 The knees of a frank breech are hyperextended at this point and it is important to correctly identify the popliteal fossae to avoid further hyperextension and damage to the fetal knee.

b. Løvset’s manoeuvre for nuchal arms. Rotate the body to facilitate delivery of the arms by sweeping the anterior humerus across the chest of the fetus (Løvset’s manoeuvre). Rotate the other arm anterior and repeat (Figure 3).

8. Support the baby to maintain the head in a flexed position. Suprapubic pressure may help. Maternal expulsive efforts should be encouraged.
9. The body should be supported in a horizontal position.
10. The Mauriceau-Smellie-Veit manoeuvre can be used to deliver the head in flexion (Figure 4).
11. Use forceps, if needed. Piper’s forceps were specifically designed for this purpose. (Figure 5).
12. Delivery of the breech with the woman in the upright position, as a means of increasing power from above seems to reduce the need for intervention, and is commonly used in some countries. 

Follow-Up

Care After Breech Delivery Should Include

- Active third stage management
- Cord blood gas analysis
- Examination for maternal trauma
- Examination for neonatal trauma: examine the hips with care; repeat the examination before discharge
- Review delivery with the family
- Documentation

Documentation: Breech Delivery

A complete review of risks and benefits for vaginal delivery and consent must be clearly and completely documented in all cases. A contemporaneous written note and a dictated operative record are recommended. It must be documented whether the vaginal delivery was undertaken as an incidental emergency or was planned and consensual.

Suggested format for a chart note (this may also serve as a template to dictate a delivery summary):

- Date and time
- Health care provider(s) present
- Type of breech
- Record of discussion with the woman of the risks, benefits, and options
- Assessment of maternal pelvis
- Fetal heart rate and contractions
- Progress in labour, including time of commencement of active pushing
- Manoeuvres or manipulations required
- Time between crowning and complete delivery of the fetus
- Number of attempts and ease of application of forceps (if used)
- Duration of traction and force used (if forceps used)
- Description of maternal and neonatal injuries (if any)
Summary

Management of a fetus in breech presentation continues to provoke much discussion and controversy despite many studies and trials. An informed discussion and consent process involving the woman presenting with a breech is critical.

Currently the recommended management for the singleton term breech is to offer external cephalic version. If ECV is unsuccessful, declined, or unavailable, and in a setting of appropriate experience, support, and informed consent, a planned vaginal delivery of a frank or complete breech may then be contemplated. However, all care providers involved in managing deliveries must be prepared for the unexpected vaginal breech delivery. Therefore, the technique and manoeuvre for a safe assisted vaginal breech delivery should be practiced and reviewed regularly. GETHIPPOS is a good example of a mnemonic that can assist in preparedness for vaginal breech birth (see Appendix).
References


# Appendix

## GETHIPPOS Mnemonic

<table>
<thead>
<tr>
<th>G</th>
<th>Growth assessment: rule out IUGR</th>
</tr>
</thead>
<tbody>
<tr>
<td>E</td>
<td>EFM recommended</td>
</tr>
<tr>
<td>T</td>
<td>Type of breech (frank or complete)</td>
</tr>
<tr>
<td>H</td>
<td>Help needed (from department of anaesthesia, OR staff, pediatrics, second MD)</td>
</tr>
<tr>
<td>I</td>
<td>IV in place, CBC &amp; group and screen</td>
</tr>
<tr>
<td>P</td>
<td>Progress in labour adequate? (maximum 60-minute active second stage)</td>
</tr>
<tr>
<td>P</td>
<td>Power from above after crowning (Bracht manoeuvre &amp; oxytocin) safer than pulling from below</td>
</tr>
<tr>
<td>O</td>
<td>Oxytocin ready and hanging to ensure strong contractions at delivery</td>
</tr>
<tr>
<td>S</td>
<td>Smellie-Veit manoeuvre for after coming head if needed</td>
</tr>
</tbody>
</table>
# Table of Contents

Chapter 15 Postpartum Hemorrhage

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Introduction</td>
<td>333</td>
</tr>
<tr>
<td>Definitions</td>
<td>333</td>
</tr>
<tr>
<td>Incidence</td>
<td>333</td>
</tr>
<tr>
<td>Identification and Diagnosis</td>
<td>334</td>
</tr>
<tr>
<td>Etiology</td>
<td>334</td>
</tr>
<tr>
<td>Risk Factors</td>
<td>335</td>
</tr>
<tr>
<td>Prevention</td>
<td>336</td>
</tr>
<tr>
<td>Active Management of Third Stage Labour (AMTSL)</td>
<td>336</td>
</tr>
<tr>
<td>Uterotonic Agents</td>
<td>337</td>
</tr>
<tr>
<td>Non-Pharmacologic Measures Influencing Blood Loss at Delivery</td>
<td>343</td>
</tr>
<tr>
<td>Management</td>
<td>346</td>
</tr>
<tr>
<td>Estimating Blood Loss</td>
<td>346</td>
</tr>
<tr>
<td>Assess Uterine TONE by palpating the Fundus</td>
<td>346</td>
</tr>
<tr>
<td>Additional Measures to Stabilize the Patient and Lessen Blood Loss with Uterine Atony</td>
<td>347</td>
</tr>
<tr>
<td>Other Causes of Excessive Bleeding at Delivery</td>
<td>352</td>
</tr>
<tr>
<td>Bleeding and Anticoagulants</td>
<td>354</td>
</tr>
<tr>
<td>Summary</td>
<td>354</td>
</tr>
<tr>
<td>Management of Postpartum Hemorrhage</td>
<td>368</td>
</tr>
<tr>
<td>Contents of Obstetric Hemorrhage Equipment Tray</td>
<td>369</td>
</tr>
<tr>
<td>Injection of the Umbilical Vein for Retained Placenta:</td>
<td>371</td>
</tr>
<tr>
<td>Uterine Compression Sutures</td>
<td>372</td>
</tr>
</tbody>
</table>
Chapter 15

Postpartum Hemorrhage

Introduction

Definitions

Postpartum hemorrhage (PPH) has been defined as blood loss in excess of 500 mL in a vaginal birth and in excess of 1000 mL in an abdominal delivery. Clinical estimates of blood loss are often inaccurate, so for clinical purposes, any blood loss that has the potential to produce hemodynamic instability should be considered a postpartum hemorrhage.

Primary (immediate/early) postpartum hemorrhage occurs within the first 24 hours after delivery. Approximately 70% of immediate PPH cases are due to uterine atony.

Atony of the uterus is the failure of the uterus to contract adequately after delivery of the infant(s).

Secondary (delayed/late) postpartum hemorrhage occurs between 24 hours after delivery of the infant and 6 weeks postpartum. Most late PPH is due to retained products of conception, infection, or both.

Incidence

Worldwide, postpartum hemorrhage occurs in about 10% of all deliveries and is a leading cause of maternal mortality. A 2012 systematic review on regional prevalence of PPH demonstrated the lowest prevalence in Oceania (7.2%) and the highest in Africa (25.7%). The North American and European rates were similar, at 13.1% and 12.7% respectively. These authors noted that the reported prevalence of PPH varied not only by region, but also by the methods used to manage the third stage of labour and to measure blood loss.

The Public Health Agency of Canada reported that between 2010/2011 and 2014/2015, blood transfusion and PPH were the most common source of major maternal morbidity. PPH was the leading cause of direct maternal mortality accounting for 49 maternal deaths (1.4/100 000 deliveries).

Studies in high-resource countries have observed an increase in PPH, predominantly due to atonic PPH. A cohort study analyzed >100,000 deliveries in Montreal between 1978 and 2007 and found that increasing rates of labour induction, augmentation of labour, previous Caesarean section (CS), placenta previa and abnormal placentation were associated with the observed rise in PPH. A 2014 cohort study reviewing over 2 million deliveries in Canada between 2003 and 2010 identified an increase of PPH by 22% (5.1% to 6.2%), mostly attributable to the increase in atonic PPH. Increases were also noted in PPH requiring blood transfusion, PPH with hysterectomy, and use of uterine suturing or embolization/ligation of uterine vessels to control PPH.
Identification and Diagnosis

The amount of blood loss required to cause hemodynamic instability will depend on the pre-existing condition of the woman. Hemodynamic compromise is more likely to occur in association with conditions such as anemia (e.g., iron deficiency, thalassemia) or a volume-contracted state (e.g., dehydration, preeclampsia).

Hypovolemic Shock

<table>
<thead>
<tr>
<th>DEGREE OF SHOCK</th>
<th>BLOOD LOSS</th>
<th>SIGNS AND SYMPTOMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>&lt; 20%</td>
<td>• Diaphoresis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Delayed capillary refill</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Cool extremities</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Anxiety</td>
</tr>
<tr>
<td>Moderate</td>
<td>20% to 40%</td>
<td>The above plus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Tachycardia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Tachypnea</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Postural hypotension</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Oliguria</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt; 40%</td>
<td>The above plus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Hypotension</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Agitation/confusion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Hemodynamic instability</td>
</tr>
</tbody>
</table>

Etiology

It may be helpful to think of the causes of PPH in terms of the four T's:

- **Tone** – uterine atony, distended bladder
- **Trauma** – uterine, cervical, or vaginal injury, perineal tears, episiotomy
- **Tissue** – retained placenta or clots
- **Thrombin** – pre-existing or acquired coagulopathy

The most common cause of PPH is uterine atony. Myometrial blood vessels pass between the muscle cells of the uterus. The primary mechanism of immediate hemostasis following delivery is myometrial contraction causing occlusion of uterine blood vessels, the so-called ‘living ligatures’ of the uterus.

Significant blood loss can occur very quickly. Women can lose up to 500 ml of blood in 1 minute during a PPH. The average woman has approximately 5 litres of blood in her circulation. At this rate of loss, it is possible for a woman to exsanguinate within 10 minutes. Rapid, efficient action must be taken to save the woman’s life and to prevent complications related to significant blood loss.
Risk Factors

<table>
<thead>
<tr>
<th>ETIOLOGIC PROCESS</th>
<th>CLINICAL RISK FACTORS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abnormalities of uterine contraction (Tone)</td>
<td>Over-distended uterus</td>
</tr>
<tr>
<td></td>
<td>• polyhydramnios</td>
</tr>
<tr>
<td></td>
<td>• multiple gestation</td>
</tr>
<tr>
<td></td>
<td>• macrosomia</td>
</tr>
<tr>
<td></td>
<td>• rapid labour</td>
</tr>
<tr>
<td></td>
<td>• prolonged labour</td>
</tr>
<tr>
<td></td>
<td>• high parity</td>
</tr>
<tr>
<td></td>
<td>• oxytocin use</td>
</tr>
<tr>
<td></td>
<td>• induction of labour</td>
</tr>
<tr>
<td>Uterine muscle exhaustion</td>
<td></td>
</tr>
<tr>
<td>Intra-amniotic infection</td>
<td>• fever</td>
</tr>
<tr>
<td></td>
<td>• prolonged ROM</td>
</tr>
<tr>
<td>Functional/anatomic distortion of the uterus</td>
<td></td>
</tr>
<tr>
<td>Uterine-relaxing medications</td>
<td></td>
</tr>
<tr>
<td>Bladder distension, which may prevent uterine contraction</td>
<td></td>
</tr>
<tr>
<td>Retained Products of conception (Tissue)</td>
<td>Retained placenta or membranes</td>
</tr>
<tr>
<td></td>
<td>• abnormal placentation</td>
</tr>
<tr>
<td></td>
<td>• retained cotyledon or succenturiate lobe</td>
</tr>
<tr>
<td></td>
<td>• incomplete placenta at delivery</td>
</tr>
<tr>
<td></td>
<td>• previous uterine surgery</td>
</tr>
<tr>
<td></td>
<td>• high parity</td>
</tr>
<tr>
<td></td>
<td>• abnormal placenta on ultrasound</td>
</tr>
<tr>
<td>Retained blood clots</td>
<td>• atonic uterus</td>
</tr>
<tr>
<td>Genital Tract Trauma (Trauma)</td>
<td>Lacerations of the cervix, vagina, or perineum</td>
</tr>
<tr>
<td></td>
<td>• precipitous delivery</td>
</tr>
<tr>
<td></td>
<td>• operative delivery</td>
</tr>
<tr>
<td>Extensions, lacerations at CS</td>
<td>• malposition</td>
</tr>
<tr>
<td></td>
<td>• deep engagement</td>
</tr>
<tr>
<td>Uterine rupture</td>
<td>• previous uterine surgery</td>
</tr>
<tr>
<td>Uterine inversion</td>
<td>• high parity</td>
</tr>
<tr>
<td></td>
<td>• fundal placenta</td>
</tr>
</tbody>
</table>
### Prevention

#### Active Management of Third Stage Labour (AMTSL)

Postpartum hemorrhage is the leading cause of direct maternal mortality worldwide. Because of this, the guidelines of multiple national societies (SOGC, RCOG, RANZCOG, and CNGOF) and international institutions (FIGO and WHO) support the use of uterotonic as part of the active management of the third stage of labor.

Expectant, or physiologic, management of the third stage allows the placenta to deliver spontaneously and the uterus to contract spontaneously. By contrast, ACTIVE MANAGEMENT involves caregiver intervention to assist in the expulsion of the placenta by increasing uterine contractions, and to avoid PPH by preventing uterine atony. The usual components of AMTSL have included:

- Administration of Uterotonic Agents
- Controlled Cord Traction (CCT)
- Uterine Massage after delivery of the placenta

Of these 3 components, administration of an uterotonic agent (usually oxytocin) has the greatest impact on blood loss. Controlled cord traction reduces the need for manual removal of the placenta (RR 0.69). However, it did not decrease blood loss of >1000ml in a randomized trial of >23,000 women. CCT requires countertraction on the uterine fundus.

---

<table>
<thead>
<tr>
<th>Abnormalities of Coagulation (Thrombin)</th>
<th>ETIOLOGIC PROCESS</th>
<th>CLINICAL RISK FACTORS</th>
</tr>
</thead>
</table>
| Abnormalities of Coagulation (Thrombin) | Pre-existing states  
- Hemophilia A  
- Von Willebrand disease  
Prior invasive treatment of PPH with embolization (39%) and ligation (26%) | history of hereditary coagulopathies  
- history of liver disease  
- history of other episodes of excessive bleeding |
| Acquired in pregnancy  
- idiopathic thrombocytopenic purpura  
- thrombocytopenia with preeclampsia  
- disseminated intravascular coagulation  
- dead fetus in utero  
- severe infection  
- abruptio  
- amniotic fluid embolus | bruising  
- elevated BP  
- fetal demise  
- fever, neutrophilia or neutropenia  
- antepartum bleed hemorrhage  
- sudden collapse |
| Therapeutic anti-coagulation | history of thrombotic disease |
| Other | BMI > 30  
Selective serotonin reuptake inhibitors  
Serotonin–norepinephrine reuptake inhibitors |
by a skilled birth attendant to avoid uterine inversion. Evidence is limited regarding the independent benefit of uterine massage as a preventive measure.  

A joint statement by the International Confederation of Midwives and FIGO and a review by WHO support the recommendation that all deliveries should be attended by a caregiver trained to manage the third stage of labour. 

A 2015 Cochrane meta-analysis of AMTSL determined that compared with expectant management, active management reduced the risk of PPH ≥1000ml (average Risk Ratio 0.34) and that of hemoglobin concentration less than 9 g/dL (average Risk Ratio 0.50), regardless of the woman's bleeding risk profile. Many earlier studies on AMSTL included early cord clamping along with oxytocin and cord traction. Subsequently, it has been shown that delayed cord clamping does not increase PPH. 

### Uterotonic Agents

During the third stage, the muscles of the uterus contract, compressing the blood vessels that pass through the uterine wall to the placental surface and slowing the flow of blood. The contraction also causes the placenta to separate from the uterine wall. The absence of uterine contractions is called atony. Uterotonics promote uterine contractions.

The CLASSES OF uterotonic agents include Oxytocics, Ergot Alkaloids, and Prostaglandins.

### UTEROTONICS for the prevention and treatment of PPH

<table>
<thead>
<tr>
<th>MEDICATION CLASS</th>
<th>CHARACTERISTICS</th>
<th>SIDE EFFECTS/ CONTRAINDICATIONS</th>
<th>USE IN PREVENTION OF PPH</th>
<th>USE IN TREATMENT OF PPH</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Oxytocics</strong></td>
<td>Preferred first line uterotonic.</td>
<td>Rare: Nausea, vomiting, headache, flushing.</td>
<td>10 unit IM</td>
<td>Same as prevention</td>
</tr>
<tr>
<td>Oxytocin</td>
<td>Stimulates muscle of upper uterine segment causing contraction to compress blood vessels.</td>
<td>Hypotension with rapid IV bolus.</td>
<td>20-40 units/litre of RL or NS infused quickly</td>
<td>Run infusion wide open</td>
</tr>
<tr>
<td></td>
<td>IV: Acts immediately</td>
<td>Water intoxication with high doses/prolonged infusion/hypotonic IV solution. Use normal saline or Ringers Lactate.</td>
<td>5 units IV over 1-2 minutes</td>
<td></td>
</tr>
<tr>
<td></td>
<td>IM: 3-5 minutes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carbetocin</td>
<td>Long-acting oxytocin analogue</td>
<td>Nausea, vomiting, flushing, headache.</td>
<td>100 mcg IV over 1 minute, or IM, for scheduled Caesarean Section</td>
<td>Limited data</td>
</tr>
<tr>
<td>Ergot Alkaloids</td>
<td>Stimulates myometrium of the upper AND lower uterine segments</td>
<td>Nausea, vomiting, Hypertension: CONTRAINDICATED in all Hypertensive Disorders of Pregnancy</td>
<td>0.2 to 0.25mg IM or IV</td>
<td>Same as prevention</td>
</tr>
<tr>
<td></td>
<td>IV: Acts&lt; 1 minute</td>
<td></td>
<td>2 hours</td>
<td>Repeat every</td>
</tr>
<tr>
<td></td>
<td>IM: 2-5 minutes</td>
<td></td>
<td></td>
<td>2 hours</td>
</tr>
</tbody>
</table>
### Postpartum Hemorrhage

#### Prostaglandins

<table>
<thead>
<tr>
<th>Drug</th>
<th>Causes vasoconstriction and enhanced contractility of the myometrium</th>
<th>Fever (most common with &gt; 600mcg)</th>
<th>Fastest acting: 400 mcg SL</th>
<th>Alternate: 800mcg rectally</th>
</tr>
</thead>
<tbody>
<tr>
<td>Misoprostol</td>
<td>Prostaglandin E1</td>
<td>400mcg sublingual (achieves highest serum peak level)</td>
<td>250mcg IM or IMM (intramyometrial) every 15 minutes; maximum 8 doses (2mg)</td>
<td></td>
</tr>
<tr>
<td>Carboprost (Hemabate)</td>
<td>Prostaglandin F2α</td>
<td>400mcg sublingual (achieves highest serum peak level)</td>
<td>250mcg IM or IMM (intramyometrial) every 15 minutes; maximum 8 doses (2mg)</td>
<td></td>
</tr>
</tbody>
</table>

#### ADDITIONAL MEDICATION

<table>
<thead>
<tr>
<th>Drug</th>
<th>Inhibits fibrinolysis</th>
<th>Not proven effective</th>
<th>1 gram IV over 10 minutes, within 3h of PPH diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tranexamic Acid</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Oxytocin

- Oxytocin is the drug of choice for prevention and treatment of PPH due to efficacy, low incidence of side effects and low cost.

The Abu Dhabi study included in the Cochrane analysis demonstrated the benefits of active intervention during the third stage with controlled cord traction and 10 units of oxytocin administered intramuscularly (IM) compared with minimal intervention. With active management, there was a lower incidence of PPH (5.8% vs. 11%), reduced incidence of retained placenta at ≥ 30 minutes (1.6% vs. 4.5%), and less need for additional uterotonic agents (2.3% vs. 5.1%).

A 2011 randomized controlled trial (RCT) in high resource countries compared active (oxytocin 10 U IM, early cord clamping, and controlled cord traction) versus expectant management in 1802 women. Blood loss was reduced (535 mL vs. 680 mL) and PPH > 1000 mL was lower (10% vs. 16.8%) in the active versus expectant management groups. Blood loss increased for increased duration of the third stage (40 mL / 5-min duration) and increased placenta weight (44 mL / 100 gm weight).

Higher prophylactic amounts of oxytocin may be required in a woman exposed to oxytocin in labour when delivered by CS. Lavoie et al. measured that woman who had a CS after labour exposed to exogenous oxytocin needed an infusion rate of 28 IU/h higher to prevent bleeding as defined by the surgeon compared to women undergoing an elective CS (44.2 IU/h vs 16.2 IU/h).

Oxytocin may be given IM or by slow IV bolus. The IV route may be more effective: A double-blinded, placebo-controlled RCT of >1000 women comparing 10u oxytocin given IV push over 1 minute with 10u IM showed less PPH >1000mL.
(5% vs 8%) and less need for transfusion (2% vs 4%). The difference in blood loss of 500-999ml did not reach statistical significance (19% vs 23%; P=0.07).

IV Oxytocin must be given by slow bolus (≥1 minute) to minimize the impact of hemodynamic changes. Hypotension and tachycardia have been demonstrated with invasive monitoring during oxytocin bolus. RCOG NICE 2016 guidelines recommend oxytocin 5U IV infused slowly (to mitigate the hemodynamic and cardiac concerns noted above) to prevent PPH in all women undergoing CS.

Additional oxytocin infusion after routine administration of oxytocin bolus in women booked for elective CS reduces blood loss and the need for additional uterotonics.

Oxytocin has an anti-diuretic effect, and must be given in an isotonic IV solution (Normal Saline or Lactated Ringers) to avoid hyponatremia/water intoxication.

**Carbetocin**

**Prevention**
- Reduces the need for additional uterotonics but not PPH for elective CS
- Reduces the need for uterine massage but no other measure of bleeding after vaginal delivery in women with one risk factor for PPH

**Treatment**
- There is limited data on the use of carbetocin for treatment of PPH.

Carbetocin is a long-acting oxytocic. The recommended dose of carbetocin is 100 micrograms IM or IV given slowly over one minute. The pharmacokinetics of both administration routes are almost the same: Whether given IV or IM, a firm uterine contraction occurs within 2 minutes in 90% of women. Durations of action is about 1 hour when given IV and 2 hours when given IM.

Carbetocin compared with oxytocin infusion has been shown to reduce bleeding secondary to uterine atony in elective CS and is associated with less need for additional uterotonics but there was no difference in PPH.

A 2012 Cochrane review concluded that, for vaginal deliveries, there is insufficient evidence that 100 mcg IV carbetocin is as effective as oxytocin in preventing PPH. The need for uterine massage was decreased with carbetocin (RR 0.54 for CS and 0.70 for vaginal delivery). Carbetocin should not be used as a first-line agent before other uterotonic agents.

RCTs comparing carbetocin with oxytocin in women with 1 or 2 risk factors for PPH in term vaginal deliveries did not show any significant difference for bleeding, need for other uterotonic medications, and blood transfusion.

Studies of Carbetocin in the treatment, rather than prevention, of PPH are very limited. A randomized trial of 100 women receiving 100mcg of Carbetocin or 5units of Oxytocin suggested less total blood loss in those suffering a minor PPH (500-999ml), but no difference in major PPH (≥1000ml).

There is no efficacy or safety data for repeated doses of Carbetocin.
A new heat-stable form of Carbetocin has been developed. It may be particularly useful in low-resource settings, where cold storage is not available. A multicentre trial randomized almost 30,000 women to receive 100mcg of heat-stable Carbetocin IM or 10units of Oxytocin IM immediately after vaginal birth. The Carbetocin was shown to be non-inferior (as effective) to oxytocin for blood loss of at least 500ml or use of additional uterotonic agents. Non-inferiority was not shown for blood loss of at least 1000ml; however, the study was not powered for this outcome. There was no significant difference in adverse effects or other measure of bleeding (blood transfusion, manual removal of placenta, additional surgical procedures). 64

**Ergonovine**

**Prevention**
- It is as effective as oxytocin but causes more side effects
- Its use is contraindicated in women with hypertension.
- It stimulates contraction of the lower uterine segment as well as the fundus of the uterus

**Treatment**
- It can be repeated IM or IV every 2 hours

Ergonovine stimulates the myometrial alpha-adrenergic receptors of the upper and lower segments of the uterus producing a tetanic contraction. It is an effective agent for the treatment and prevention of PPH, 65 66 68 but it has more adverse effects (nausea, vomiting and hypertension) than oxytocin, making it a less preferred agent. 69 70 It is contraindicated in the presence of hypertension. The dose is 0.2 to 0.25 mg, has a rapid onset of action (< 1 minute for IV and 2 to 5 minutes for IM) and interval dosing is every 2 hours. 71

Compared to placebo, ergonovine reduce the incidence of PPH > 500ml and severe PPH (>1000ml). 72 Studies show inconsistent results regarding retained placenta or need for manual removal. The Dublin trial which used IV administration had a higher incidence of retained placenta, but used a dose of 0.5mg. 73

Compared to oxytocin, ergonovine:
- Is equally effective for the prevention of PPH
- Has a higher incidence of headaches, nausea, and hypertension 70

**Misoprostol**

**Prevention**
- Reduces risk of PPH better than placebo but not as effectively as oxytocin.
- 400 mcg SL is preferred dose and route

**Treatment**
- Misoprostol dose is 400 mcg SL.
- There is a delay of at least 30 minutes between administration and onset of action.
Misoprostol has been extensively studied in obstetrics as an uterotonic agent. It has not been shown to be superior to other uterotonics in AMTSL for the prevention and treatment of PPH. Because of its stability at room temperature, ease of administration, low cost, and uterotonic properties, the World Health Organization includes misoprostol in its essential medication list, for situations where oxytocin is not available or cannot be safely used.\(^7\)

A 2011 study by Elati and Weeks measured the effect of 3 doses of misoprostol (200 mcg, 400 mcg and 600 mcg) and 10 U IM oxytocin on uterine contractions in the immediate postpartum period using an intrauterine pressure catheter.\(^7\) There was no difference between the misoprostol doses. At 10 minutes, the intrauterine pressure (IUP) with oxytocin was much higher than misoprostol. The pressure was the same at 30 minutes while the IUP was much higher for misoprostol than for oxytocin from 50 to 120 minutes. The incidence of high fever (temperature > 39 degrees) was the same for the 200 mcg and 400 mcg doses (8.3%) and much higher for the 600 mcg dose (45.4%).\(^7\) A 2013 Cochrane review by Hofmeyr et al. supported that adverse effects increased at doses greater than 600 mcg.\(^7\)

Reviews of the pharmacokinetics of misoprostol can be summarized as follows:

<table>
<thead>
<tr>
<th>ROUTE</th>
<th>PEAK ONSET (T-MAX)</th>
<th>BIOAVAILABILITY (UAC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sublingual</td>
<td>30 minutes</td>
<td>120 minutes</td>
</tr>
<tr>
<td>Oral</td>
<td>30 minutes</td>
<td>120 minutes</td>
</tr>
<tr>
<td>Vaginal</td>
<td>70 to 80 minutes</td>
<td>6 hours</td>
</tr>
<tr>
<td>Rectal</td>
<td>40 to 60 minutes</td>
<td>6 hours</td>
</tr>
</tbody>
</table>

The sublingual route has a much higher serum peak level than the oral route because it avoids the first pass metabolism by the liver.

There have been many clinical studies involving misoprostol in order to determine its place in the medical prevention and treatment of PPH. Comparison of studies is challenging because of the variety of doses (400 to 800 mcg) and routes of administration.

The studies of misoprostol and prevention of PPH can be summarized as follows:

- Misoprostol is more effective than placebo\(^7\)
- The recommended dose is 400 mcg sublingual\(^7\)
- Misoprostol is less effective than IM uterotonics in prevention of PPH\(^7\)\(9\)\(10\)
- In low resource countries, misoprostol\(^8\) (600 mcg SL) was less effective than oxytocin (10 IU IM) for the incidence of primary PPH > 500 mL within 24 hours (28.6% vs. 17.4%) for uncomplicated, term vaginal deliveries\(^10\)

The misoprostol studies for the treatment of PPH have shown:

- Misoprostol was as effective as IV oxytocin for treatment of PPH in women given prophylactic IM oxytocin\(^9\)
- Misoprostol was less effective than IV oxytocin for treatment of PPH in women not given prophylactic IM oxytocin\(^9\)
• Misoprostol (600 mcg SL) as an adjunct to routine IV oxytocin had a non-significant trend to lower blood loss in vaginal deliveries.\textsuperscript{87}

In women with a retained placenta:

• Injection of misoprostol (800 mcg in normal saline) into the umbilical vein has been associated with a lower rate of manual removal of placenta than oxytocin infusion or placebo.\textsuperscript{88}
• Administration of misoprostol (800 mcg PO) is not associated with a lower rate of manual removal of placenta compared with placebo.\textsuperscript{89}

**Carboprost / Hemabate (15-methyl F\textsubscript{2α} prostaglandin)**

**Prevention**
• Not superior to oxytocin and higher cost

**Treatment**
• Recommended dose is 250 mcg IM / intramyometrially
• Can be used every 15 minutes, total 8 doses

Carboprost is a synthetic injectable prostaglandin similar to misoprostol. A 2012 Cochrane review did not find it superior to oxytocin for prevention of PPH.\textsuperscript{79}

For women with severe PPH unresponsive to other oxytocics, studies in the 1980s first showed that PGF\textsubscript{2α} decreased the need for hysterectomy.\textsuperscript{90} In a retrospective study of 236 women with persistent postpartum hemorrhage, bleeding was successfully controlled with PGF\textsubscript{2α} in 88%.\textsuperscript{91} Takagi showed that injection of PGF\textsubscript{2α} directly into the myometrium produced strong uterine contractions more quickly than when given IM. Side effects were also less common. Doses above 250mcg were no more effective.\textsuperscript{92}

**Tranexamic Acid**

**Prevention:**
1. Reduces blood loss >1000ml in women at risk of bleeding undergoing elective CS but not vaginal birth
2. Used in addition to usual uterotonic in elective CS.

**Treatment:**
1. Second line medical treatment shown to reduce maternal death but not hysterectomy.
2. Reduces the need for laparotomy to control bleeding
3. Dose is 1g IV (100mg/ml at a rate of 1ml/min = 10ml over 10 minutes)
4. Can be repeated if:
   a. Bleeding persists after 30 minutes
   b. Bleeding re-starts within 24 hours

Tranexamic acid (TXA) is an inhibitor of fibrinolysis. It acts by preventing the conversion of plasminogens to plasmin, stabilizing clot formation.
For prevention of PPH, the majority of studies have compared TXA with placebo as adjuncts to usual prophylactic care with oxytocin. No benefit was shown in the TRAAP study randomizing >4000 women with planned vaginal birth to receive prophylactic TXA and oxytocin, compared to oxytocin alone.\textsuperscript{93} In contrast, a review by Novikova\textsuperscript{94} showed TXA decreased blood loss > 1000ml in women who underwent CS (RR 0.23, 4 studies 1534 women). Based on the latter study, RCOG recommends considering IV TXA (0.5 – 1.0 g) in addition to oxytocin for women at increased risk of PPH undergoing CS.\textsuperscript{52}

In a 2014 RCT,\textsuperscript{97} 747 women with a high incidence of anaemia were given tranexamic acid (1 g IV slowly 10 minutes before elective CS). The mean total blood loss was 242 mL in the tranexamic acid group versus 510 mL in the control group. The mean drop in hematocrit and hemoglobin levels was statistically significantly lower in the tranexamic acid group than in the control group.

For treatment of PPH, the largest study was the 2016 WOMAN trial\textsuperscript{99} an RCT of 20060 women diagnosed with active PPH (> 500ml after VD after > 1000ml after CS or sufficient blood loss to cause hemodynamic instability) that compared the addition of TXA to usual care. Most (96%) women received prophylactic uterotonics and almost all women received a secondary uterotonic (99.6% in the TXA study group and 99.5% in the control group). The study showed that women given TXA less than 3 hours after diagnosis of PPH had a reduction in overall death due to bleeding (1.2% vs 1.7%) and need for laparotomy to control bleeding (0.8% vs 1.3%). There was no benefit if TXA was given 3 hours after diagnosis of PPH for death, the need for hysterectomies, or secondary outcomes.\textsuperscript{99} The majority of centres participating in the WOMAN trial were in low resource settings. The lack of difference in hysterectomy rate, even with TXA given early, may be in part due to the move to hysterectomy early in the management of PPH because other medications and intensive care support are limited.

There was no increase in thromboembolic events with TXA.

**NON-PHARMACOLOGIC MEASURES INFLUENCING BLOOD LOSS AT DELIVERY**

**Cord Traction**

The use of cord traction has been shown to reduce the incidence of retained placenta without significant reduction of PPH.

The mainstay of AMTSL is the IM injection of oxytocin.\textsuperscript{35}

A 2015 Cochrane review\textsuperscript{100} as well as a large 2013 randomised controlled trial study (TRACOR)\textsuperscript{34} both concluded that controlled cord traction (CCT) resulted in fewer manual removal of placentas but no difference in PPH when compared to expectant or physiological delivery of the placenta.
Uterine Massage before Placental Delivery

Uterine massage before the delivery of the placenta is of no benefit. It may increase blood loss and should not be done.\(^{101, 102}\)

Two of the trials included in a 2013 Cochrane review found no evidence to show that prophylactic uterine massage to reduce PPH was effective.\(^{103}\)

A multicentre RCT of 2340 vaginal deliveries showed that the addition of uterine massage after routine oxytocin did not reduce blood loss.\(^{104}\)

A secondary analysis of 39 202 births found that uterine massage was associated with increased hemorrhagic risk.\(^{48}\)

Timing of Cord Clamping

As with controlled cord traction, the timing of cord clamping has not been independently studied in relation to the prevention of PPH. However, studies have demonstrated the benefits for the neonate of delayed cord clamping.

Delaying cord clamping by 30 to 120 seconds seems to be associated with less need for transfusion for anaemia and less intraventricular hemorrhage in non-resuscitated premature infants < 37 weeks compared with early clamping.\(^{105, 106}\)

Delayed clamping is supported in term newborns in order to provide an ongoing blood flow from the placenta to the newborn and facilitates early skin-to-skin contact.\(^{107}\)

A 2007 systematic review and meta-analysis by Hutton and Hassan\(^{108}\) comparing early (< 1 minute) with late (> 2 minutes) cord clamping suggests that there is a physiological benefit of delayed clamping to the newborn that extends into infancy. Advantages included prevention of anaemia over the first 3 months of life, and enhanced iron and ferritin stores for up to 6 months. There was no increase in respiratory distress, defined as tachypnea or grunting. There was no statistical difference in bilirubin levels or in number of infants receiving phototherapy in the late versus early group. A 2013 Cochrane review\(^{45}\) included 15 RCTs involving a total of 3911 women and infant pairs that compared early (< 60 seconds) to late (> 60 seconds) cord clamping on maternal and neonatal outcomes. The authors concluded that there was no difference in the rate of postpartum hemorrhage for mothers, and measured the same benefits to the newborns as did Hutton and Hassan. In contrast to the Hutton and Hassan review, there was an increased risk of neonatal jaundice requiring phototherapy.

Timing of Placental Delivery

- The incidence of PPH increases when a placenta is retained for > 15 minutes

An important risk factor for PPH is the failure of the placenta to deliver in a timely manner. Fifty percent of placentas deliver within 5 minutes, and 90% within 15 minutes of the baby’s birth.\(^{109}\) Magann et al. concluded that the risk of PPH increases 3-fold if the placenta is delivered > 15 minutes after the delivery of the infant rather than < 15 minutes after (13.3% vs. 4.4%).\(^{110}\) A retrospective study of 25 016 cases correlating outcomes with the timing of placental delivery determined that the optimal timing for predicting blood transfusion with an undelivered placenta was at 17 minutes.
of the third stage and that the risk of transfusion increased 3-fold at ≥ 30 minutes. IV access should be ensured at 15 minutes of an undelivered placenta.111

A 2012 case-control study identified several risk factors for placental retention, including previous retained placenta, preterm delivery, prolonged oxytocin use, preeclampsia, and ≥ 2 miscarriages or abortions.112

A 2012 RCT by Van Stralen et al.89 studied 99 women with a retained (> 60 minute) placenta after oxytocin prophylaxis (5 U IM or IV). The study showed no difference in manual removal of the placenta or blood loss for women who received misoprostol (800 mcg orally) one hour after childbirth compared with those who received placebo.

**Placental Cord Drainage**

- Reduction in length of third stage and blood loss statistically but not clinically significant
- Studies limited in quality

A 2011 Cochrane review identified 3 studies involving 1257 vaginal deliveries addressing placental cord drainage. There was a reduction in the length of the third stage of 2.85 minutes and blood loss was reduced by 77 ml. A major confounding factor was that only 1 of the 3 studies used an uterotonic (methyl-ergometrine) for prevention. The definition of a prolonged third stage in the studies varied from 30 minutes to 45 minutes. More research is required to determine the efficacy of this intervention.113

A prospective study compared cord drainage (cord not clamped after cutting) with routine clamping before cutting in 485 vaginal deliveries. All women received 5 IU oxytocin prophylaxis.114 The mean estimated blood loss was significantly lower in the cord drainage group than in the control group (207.04 ± 123.3 vs. 277.63 ± 246.9 mL). The third stage of labour was significantly shorter in the cord drainage group than in the control group (3.5 ± 1.9 vs. 7.7 ± 3.4 minutes). No adverse events occurred during the cord drainage period.

**Injection of the Umbilical Vein for Retained Placenta**

- Umbilical cord vein injection of misoprostol but not oxytocin has been shown to avoid manual removal of a retained placenta, although not PPH

Manual removal of a retained placenta may lead to complications such as infection, uterine perforation, hemorrhage, and maternal discomfort. Several interventions to prevent these complications have been studied, using various agents to assist in the detachment of the placenta and avert a manual removal. The timing of the interventions varied between 30 minutes and 45 minutes with a retained placenta.

Intravascular injection of misoprostol may be of benefit but data are limited.88,115-119 Misoprostol can be injected into the vein directly using a syringe, or the Pipingas technique can be used. (See Appendix)

Note: If bleeding is heavy, the placenta needs to be removed quickly to allow the uterus to contract. Manual removal should be done.
Management

Clinicians must keep in mind that the management of PPH requires a team approach including several simultaneous interventions.  

Estimating Blood Loss

Be prepared with a well-established protocol since this is fundamental to safe patient care. In the case of a severe PPH, a readily available obstetric hemorrhage equipment tray (see Appendix for contents) will facilitate the prompt management of hemorrhage.

Estimation of blood loss can be done by weighing blood soaked material or the use of a calibrated under buttock drape in women suspected of PPH.

The shock index (heart rate / systolic blood pressure) has been evaluated in the non-obstetrical population as an objective measurement of hemodynamic instability. In the non-pregnant population, the normal shock index range is 0.5 to 0.7.

A retrospective case-control study of 50 women with massive PPH and 50 women with no PPH was undertaken to establish an obstetrical shock index (OSI) to enable earlier prediction of significant blood loss and the need for blood transfusion. Mean OSI in the control group (i.e., normal delivery) at 10 minutes and 30 minutes was 0.74 (range, 0.4 to 1.1) and 0.76 (range, 0.5 to 1.1), respectively. In the case group, mean OSI at 10 minutes and 30 minutes was 0.91 (range, 0.4 to 1.5) and 0.90 (range, 0.5 to 1.4), respectively, with 64% requiring blood products. In the case group, 89% of women with an OSI of 1.1 or more at 10 minutes required transfusion; 75% with an OSI of 1.1 or more at 30 minutes required transfusion. The study authors recommended that the normal OSI range should be 0.7 to 0.9 for obstetrical patients. “An OSI of more than 1 seems to be a useful adjunct in estimating blood loss in cases of massive PPH and in predicting the need for blood and blood products.”

A 2015 retrospective cohort study of 233 women with PPH > 1500 mL in low-resource settings found that a shock index < 0.9 was reassuring whereas an index ≥ 0.9 was predictive of ICU admission, need for blood transfusion, and invasive surgical procedures.

Assess Uterine TONE by palpating the Fundus

Uterine atony is the cause of 70% of Postpartum Hemorrhages. Clinicians should address this first. If the uterine fundus is firm, but bleeding remains excessive, evaluate for retained TISSUE, genital tract TRAUMA, or coagulopathy (THROMBIN). (See “If the uterus is FIRM” section below.)

If the uterus is boggy/tonic

If the placenta has NOT delivered, it must be removed manually while uterotonic drugs are being given, IV access is considered and the bladder emptied.
If the placenta has delivered, proceed to **external uterine massage and uterotonics**. Oxytocin is first-line treatment and should be given by rapid IV infusion if access is immediately available. Otherwise, IM oxytocin should be given until IV access can be established. Proceed immediately to bimanual massage if the uterus remains boggy and bleeding persists. This tamponade will reduce further bleeding until assistance arrives or the uterine tone improves.

**Bimanual massage/compression technique**: The uterus is compressed between a hand in the vagina against the anterior part of the cervix and a hand on the fundus.

If analgesia allows, the **uterus should be explored** at this stage to rule out retained **TISSUE** (placental fragments, membranes, and clots), uterine inversion, or uterine rupture.

Remove clots in the upper vaginal area or lower segment of the uterus.

**Emptying the bladder** may help with assessment and subsequent manoeuvres in the management of PPH. It may also assist in keeping the uterus contracted. In addition, urine output is a reliable way of monitoring the effectiveness of fluid resuscitation in a woman with ongoing hemorrhage.

If the uterus is still boggy, proceed with further pharmacologic intervention:

- oxytocin (first line): $^{124, 125}$
- 5 units of oxytocin by slow IV bolus over 1 minute
- oxytocin 20 to 40 units in 1 litre of normal saline, initially wide open
- 10 units oxytocin IM if cardiovascular collapse or no IV access

If the uterus is still boggy and has not been fully explored, this must be done now.

If bogginess or hemorrhage continues, consider

- Ergonovine
- Carboprost/Hemabate
- Misoprostol
- Tranexamic Acid

**ADDITIONAL MEASURES TO STABILIZE THE PATIENT AND LESSEN BLOOD LOSS** with uterine atony

- Uterine balloon tamponade
- External Aortic compression
- Uterine Artery embolization
- Laparotomy for vessel ligation, placement of compression sutures, or hysterectomy

A UK study found that for every 10 000 deliveries there were 2.2 cases of PPH requiring a second-line therapy. (This did not include those treated with an intrauterine balloon only.) Recognizing that these cases varied in both cause and complexity, these are the success rates noted for each intervention when it was the initial second-line therapy: Uterine compression sutures 75%, pelvic vessel ligation 36%, interventional radiology techniques 86% and recombinant Factor VIIIa 31%. 26% had a hysterectomy. $^{131}$
Uterine Balloon Tamponade (BT):

- effective method to temporarily stop bleeding
- reduces morbidity (blood loss, transfusion, embolization, hysterectomy, ICU admission)
- safe for transport
- may be used in combination with other interventions (CS + uterine compression sutures)
- more effective when used earlier, particularly in vaginal deliveries; consider early use if bleeding not controlled after 10 minutes bimanual massage

The use of internal uterine compression has proven to be successful in the management of severe PPH. It involves intrauterine compression of the endometrial tissue used in conjunction with oxytocin, thereby causing compression of the uterine surface and decreasing blood flow to the uterine wall. It can be used as a definitive or temporary treatment, thus making it an excellent tool to control bleeding for cases requiring transfer to higher level treatment centres.

Specific devices have been designed for this purpose, including the Bakri Balloon. The Sengstaken-Blakemore esophageal catheter and condom catheters (a condom tied over the end of a straight bladder catheter) have also been shown to be effective.

The insertion of the balloon device is a relatively simple procedure that requires the operator to ensure that the entire balloon is positioned past the cervical canal. Once inserted, the balloon is filled with a sterile solution such as normal saline. Depending on the device, 250ml is infused initially, then further NS more slowly until the bleeding stops, up to 500 mL. The balloon is then left in place. Ultrasound can be used to confirm placement. To prevent expulsion of the balloon, the cervix can be “pinched” with 2 ring forceps on opposite sides to narrow the diameter, or vaginal packing can be placed below the balloon. After successful tamponade, a continued oxytocin infusion may be required to maintain uterine tone. Prophylactic antibiotics should be considered. The balloon can be left in place from 8 hours to 48 hours and then gradually deflated and removed, observing for recurrence of bleeding. This should be done in a centre prepared to provide a higher level of care if needed.

Multiple retrospective studies have reported success rates of 80% to 95% using balloon tamponade to treat PPH when bleeding has not been controlled by massage and uterotonic agents.

Clinicians can consider attempting to remove the balloon before 24 hours has elapsed. A 2016 retrospective cohort study of 274 women comparing BT for PPH for 2-12 hours vs > 12 hours found no significant differences in blood loss post-insertion, RBC transfusion, embolization, hysterectomy and ICU admission, but the > 12 hour group had higher rates of post-partum fever (27% vs 15%) and longer hospital stay (3.7 vs 3.1 days).

Several studies have illuminated the overall success and early use of BT to control PPH after preventative and secondary uterotonic agents, avoiding the need for invasive procedures (uterine artery ligation, radiological intervention, and hysterectomy).

A retrospective study by Howard and Grobman correlating timing of intervention and outcomes, determined that use of tamponade at an earlier estimated blood loss had higher nadir hemoglobin, reduction of blood transfusion, fewer intensive care unit admissions, and fewer hysterectomies when compared with arterial embolization.

Use of BT after vaginal delivery (VD) is overall more successful compared to CS. A 2016 prospective study of 226 women with severe PPH showed BT (after preventative and second line uterotonic agents) to be successful 83% of the time.

Postpartum Hemorrhage
It was more successful after VD than CS (89% vs 66%) and there were more failures (need for invasive procedures and hysterectomy) in the CS group than in the VD group (50% vs 19%). Blood loss was higher in the failure group (1508ml vs 1064ml) despite similar estimated blood loss at the time of diagnosis. Coagulopathy was higher in the failure group (50% vs 17%). Bleeding that stopped or dramatically reduced by 15 minutes was associated with success (98 vs 16%)

**External Aortic Compression**

In women with active hemorrhage, external aortic compression with the non-pneumatic anti-shock garment (NASG) has been shown to reduce blood loss and associated maternal morbidity and mortality without compromising vascular flow to the lower limbs.\(^{148,149}\)
The aorta can be compressed manually to slow bleeding while other measures are undertaken. Standing on the woman's left, the aorta is compressed with the right fist while using the left hand to determine that femoral artery pulsation is no longer palpable, indicating successful compression.

**Uterine Artery Embolization**

Uterine artery embolization has been used as an alternative to surgery for PPH refractory to treatment.\(^{150}\)

Success rates vary from 78% to 96% for emergency and prophylactic embolization.\(^{151-153}\)

Factors predictive of failure were disseminated intravascular coagulation, need for massive transfusion, hemodynamic instability, and hemoglobin level < 95 g/dL.\(^{152,153}\)

Subsequent pregnancies are possible although may involve higher risk to the mother and/or fetus.

A study by Inoue et al.\(^{151}\) found that following uterine artery embolization, 53% of women who desired fertility became pregnant. Of the 40 women who became pregnant, 28 gave birth. Four deliveries were preterm, and five were associated with a hysterectomy due to placenta accreta.

**LAPAROTOMY FOR VESSEL LIGATION, COMPRESSION SUTURES AND HYSTERECTOMY**

**Uterine Artery Ligation (UAL)**

Uterine Artery ligation, in experienced hands, can help control postpartum haemorrhage. A 2012 prospective study showed a 96% success rate with uterine artery ligation in cases of severe PPH unresponsive to other intervention, thus avoiding the need for hysterectomy.\(^{152}\) In patients with central placenta previa and a planned caesarean section, a RCT of 100 patients showed lower blood loss when the uterine arteries were ligated prior to the uterine incision.\(^{157}\)
Uterine Compression Sutures (see Appendix for diagrams)
Various techniques have been described in which sutures placed around and through the uterus at laparotomy are then tied to provide sustained compression of the uterus, decreasing bleeding.\textsuperscript{158-162} The techniques developed by Christopher B-Lynch and JH Cho are included in the Appendix of this chapter.

Numerous studies have demonstrated these techniques to be safe and efficacious for the treatment of severe, atonic PPH.\textsuperscript{124,143,144,152}

There were few uterine synechia when the uterine cavity was evaluated 3 to 6 months later.\textsuperscript{163,164}

A 2014 cohort study of 252 women found that having had PPH and a B-Lynch suture was not associated with an increased risk of future adverse pregnancy outcomes.\textsuperscript{166}

A prolonged delay\textsuperscript{167} of 2 to 6 hours between delivery and uterine compression suture was associated with a 4-fold increase in hysterectomy.

**Emergency hysterectomy.** Although a last resort, this must be considered before the progression of hemorrhage to the point of cardiovascular collapse. The most common risk factors are multiparity, CS in previous or current pregnancy, and abnormal placentation.\textsuperscript{168}

If the uterus is FIRM:
- Explore the lower genital tract for TRAUMA (TEARS)
- Ensure adequate analgesia
- Ensure good lighting and exposure
- Undertake surgical repair of vaginal and cervical lacerations
- Temporise with packing

If the bleeding continues and is originating from a firm uterus:
- evaluate for an acquired coagulopathy\textsuperscript{169} (THROMBIN)
- fibrinogen levels are physiologically elevated in pregnancy. Low normal levels (under 3 g/L) in a setting of severe postpartum hemorrhage are abnormal and replacement should be considered\textsuperscript{169,170}

If coagulation is abnormal
- correct with fresh frozen plasma (FFP), cryoprecipitate, platelets, and packed red blood cells

If the coagulation is normal
- prepare for the OR
- rule out uterine rupture or an inadequately repaired incision
- consider vessel ligation or embolization
- Consider using B-Lynch technique or Cho technique, or performing hysterectomy
- if surgical expertise is unavailable, consider tamponade, stabilization, and transport
OTHER CAUSES OF EXCESSIVE BLEEDING AT DELIVERY

Uterine Inversion

- Occurs in 1/25 000 deliveries
- Often is iatrogenic and more common in grand multiparous (parity > 5) women
- The placenta may appear at the introitus with a mass attached
- The woman may experience bradycardia and shock secondary to increased vagal tone
- Replacement of the uterus should be performed promptly without removing the placenta
- Uterine relaxation may facilitate this manoeuvre
- Replacement order is by "last out, first in" Begin by returning the normally most distal part of the uterus to its original position, followed by the proximal wall, and lastly, the uterine fundus.
- Use exploratory laparotomy for replacement if all else fails

Uterine Rupture

- Most common in women with prior uterine surgery
- Grand multiparous women or those undergoing induction or augmentation are at risk
- Following vaginal delivery, a defect may be palpated on manual exploration
- Vigorous resuscitation and emergency laparotomy are indicated

Placenta Accreta (see also Antepartum Hemorrhage chapter)

- Placenta accreta is the abnormal implantation of the placenta with villus attachment to the myometrium resulting in loss of the normal cleavage plane
- Occurs in 1/2500 deliveries (10-fold increase in the last 50 years due mainly to increase in CS rate and the resulting lower uterine scar).
- 13-fold increased risk for PPH\textsuperscript{171}, massive hemorrhage and peripartum hysterectomy\textsuperscript{172}
- Most common in women with prior uterine surgery, especially with an anterior placenta, and increases with increased number of CS\textsuperscript{173-175}
- Women with placenta previa and grand multiparous women are at risk
- Commonly presents as a retained placenta
- Uterine embolization with placenta in situ has been shown to be successful in controlling PPH and avoiding hysterectomy\textsuperscript{176, 177}
- If the placenta seems adherent at the time of attempted manual removal then consider placenta accreta
- Early recognition, preferably during the antenatal period, and anticipation of this event is preferable in this high-risk emergency situation

Women in whom placenta accreta is diagnosed antenatally have better outcomes, are less likely to need blood transfusions, and are less likely to experience attempts to remove the placenta.\textsuperscript{178}
PPH and the need for blood transfusion are less likely when the placenta is left in place to conserve the uterus or before hysterectomy. Appropriate resuscitation and consultation are indicated as the risk for severe hemorrhage is extremely high. The blood bank should be notified and blood products prepared.

**Management of PPH can progress as follows as the volume of blood loss increases:**

Blood loss > 500 mL to 1000 mL with normal vital signs (placenta has delivered)

1. Help (nearest members, PPH kit)
2. Bimanual external/internal uterine massage to
   a. express intra-uterine blood/clots and
   b. stimulate uterine contraction
3. Foley catheter
4. Additional uterotonics
5. IV access with rapid infusion crystalloid fluids (up to 2L)
6. Type and cross 2U RBC

Blood loss continues (up to 1500 mL, or > 2 uterotonics) with normal vital signs

1. Help (obstetrics, anaesthesia, ER, ICU, blood lab, OR)
2. Measure vital signs and blood loss
3. O2
4. Second IV access for IV crystalloids
5. Intrauterine balloon catheter
6. Uterotonics + TXA
7. STAT lab (CBC, coagulation factors, fibrinogen)
8. Get 2U RBC, thaw 2U FFP
9. Prepare OR transfer

Blood loss continues (>1500ml OR > 2U RBC OR at risk for bleeding / coagulopathy) OR any patient with hemodynamic instability

1. Help (obstetrics, anaesthesia, ER, ICU, blood lab, OR)
2. Move to OR for possible hysterectomy
3. Massive transfusion protocol

**Don’t forget your ABCs**

- Talk to and observe the woman
- Monitor vital signs
- Remember that compensatory responses to blood loss in these women are excellent and may give caregivers a false sense of security
- Commence at least one large bore IV (preferably 16 gauge or larger)
- Run a crystalloid solution drip wide open (e.g., saline is preferred to Ringer’s lactate)
- Obtain a CBC, cross match, and consider coagulation studies (PT, PTT, LFTs, fibrinogen, calcium, lactate)
Get help

- Consider the need for additional personnel to manage the resuscitation
- Notify the laboratory of the potential need for massive transfusion support

**BLEEDING AND ANTICOAGULANTS**

**Low Molecular Weight Heparin**

Low molecular weight heparin (LMWH) is the drug of choice for prevention of thromboembolic disorders, but its impact on the incidence of PPH is unclear. In a systematic review which included 1320 women receiving prophylactic LMWH, the RR of PPH was 1.45, just reaching statistical significance, but with a difference in blood loss of just 33ml and no difference in transfusion. **Roshani et al.** performed a retrospective cohort study of 95 women (524 controls) who received therapeutic doses of LMWH, and who had been advised to stop it at the onset of contractions, rupture of membranes, or the day before planned induction or CS. The incidence of PPH was not significantly increased, with a rate of 18% in users and 22% in non-users. There was no increase in PPH or severe PPH, and the median blood loss in normal vaginal deliveries was actually lower in LMWH users (200 mL) than in non-users (300 mL). These authors note that the difference may have been due to differing strategies to prevent bleeding.

**Summary**

1. Active management of the third stage of labour reduces the incidence and severity of PPH.
2. Postpartum hemorrhage is an emergency that requires a clear understanding of the pathophysiology responsible.
3. A clear management plan that ensures adequate volume replacement and secures hemostasis must be in place.
4. The importance of the assessment and management of the woman’s Circulation, Airway, Breathing (CABs) cannot be overstated.
5. The woman should be resuscitated and additional help summoned.
6. Uterine atony is the causes 70% of PPH.
References


100. Hofmeyr GJ, Mshweshwe NT, Gulmezoglu AM. Controlled cord traction for the third stage of labour. Cochrane Database of Systematic Reviews. 2015;1:CD008020.


120. The American Congress of Obstetricians and Gynecologists A. Obstetric Hemorrhage Checklist. 2015.


138. Bakri® Postpartum Balloon with Rapid Instillation Components (Product Monograph)


Appendix A

Management of Postpartum Hemorrhage

Postpartum Hemorrhage

Used with permission of Salus Global Corporation
Appendix B

Contents of Obstetric Hemorrhage Equipment Tray

Access/exposure
- 3 vaginal retractors
- 2 ring forceps

Eye needles
- Straight, 10cm
- Curved 70-80mm, blunt point

Sutures
- No.1 vicryl
- 0 and 2 chromic catgut with curved needle
- ethiguard curved, blunt point monocryl

Uterine/vaginal tamponade
- Vaginal packs
- Kerlix gauze roll
- Uterine balloon (Sengstaken-Blakemore, Rusch urological balloon, Bakri balloon, surgical glove and catheter)

Diagrams
- Uterine artery and ovarian artery ligation
- Uterine compression techniques; B-Lynch and Cho
Appendix C

MASSIVE TRANSFUSION PROTOCOL

Many institutions will have their own massive transfusion protocols. One example is:

Stanford University School of Medicine\cite{18}
## Appendix D

### Injection of the Umbilical Vein for Retained Placenta:

**Pipingas Technique**

1. Explain the procedure and obtain consent.

2. Prepare a syringe with the medication in 30 mL normal saline. Crush and dissolve 4 × 200 mcg tablets misoprostol in 30 mL normal saline (forms milky solution).

3. Identify the umbilical vein. Recut the cord if necessary.

4. Insert a size 10 nasogastric tube into the umbilical vein. If resistance is felt, retract the catheter by 1 to 2 cm, and then advance further, if possible.

5. The tube has reached the placenta when the majority of the catheter is inserted and resistance is felt. (The lengths of the umbilical cords varied between 30 and 47 cm in the Rogers’ study.\(^{88}\))

6. Retract by 3 to 4 cm to ensure that the tip is in the umbilical vein and not in a placental branch.

7. Attach the syringe, and inject the solution, and then clamp off the cord with the catheter.

8. Note the time of the injection.

9. Wait 10 to 30 minutes for the placenta to deliver.
Appendix E

Uterine Compression Sutures

B-Lynch technique

Cho Technique

# Table of Contents

Chapter 16 Hypertensive Disorders of Pregnancy ................................................................. 375  
   Introduction .................................................................................................................. 375  
      Definitions and Terminology .................................................................................... 375  
      Incidence .................................................................................................................. 376  
   Pathogenesis ............................................................................................................. 377  
   Morbidity and mortality ............................................................................................... 378  
      Morbidity and Mortality with Preeclampsia .............................................................. 380  
      Morbidity and Mortality With Chronic Hypertension .............................................. 380  
   Diagnosis and Evaluation ......................................................................................... 380  
      Clinical Evaluation .................................................................................................. 380  
      Investigations .......................................................................................................... 382  
   Risk Factors and Risk Reduction .............................................................................. 385  
      Predicting Onset of Preeclampsia .......................................................................... 385  
      Predicting Maternal and Perinatal Morbidity and Mortality with Established  
         Gestational Hypertension and Preeclampsia ......................................................... 386  
      Risk Reduction ........................................................................................................ 386  
   Management .............................................................................................................. 387  
      Antihypertensive Therapy ....................................................................................... 388  
      Fluid Management ................................................................................................... 390  
      Symptomatic Support .............................................................................................. 391  
      Seizure Prophylaxis ................................................................................................. 392  
      Management of HELLP Syndrome ........................................................................ 394  
      Transport .................................................................................................................. 394  
      Delivery .................................................................................................................... 394  
      Timing of Delivery ................................................................................................... 395  
      Postpartum Management ......................................................................................... 395  
      Future Cardiovascular Risk ..................................................................................... 396  
      Points to Remember in Management .................................................................... 397  
   Summary ..................................................................................................................... 397
Chapter 16
Hypertensive Disorders of Pregnancy

Introduction

Hypertensive disorders are a leading cause of direct maternal death. Women who have had preeclampsia and gestational hypertension have an increased likelihood of future cardiovascular disease, diabetes mellitus, metabolic syndrome, and related mortality.\(^1\) Preeclampsia is a cause of acute renal failure and increases the risk of chronic kidney disease.\(^2\) Women with severe preeclampsia have poorer health-related quality of life, often related to perinatal death or the admission of their infant to the NICU.\(^3\) Women who develop preeclampsia before 37 weeks’ gestation are at a significant risk in subsequent pregnancies of stillbirth, placental abruption, spontaneous preterm rupture of membranes, and small for gestational age (SGA) babies, even in the absence of preeclampsia in the subsequent pregnancy.\(^4\) Chronic hypertension is associated with increased risk of congenital malformations in the newborn, independent of antihypertensive use.\(^5,\)\(^6\) Evidence suggests that risk of depressive symptoms in adulthood is increased in offspring of women who developed preeclampsia.\(^7\) These offspring may also have increased risk for impaired cognitive functions in childhood and adulthood.\(^8\) They are more likely to be hypertensive by age 21,\(^9\) and to demonstrate an abnormal lipid profile in early adulthood.\(^10\)

While the incidence of eclampsia has dropped in Canada from 15 per 10 000 deliveries in 2004–2005 to 5 per 10 000 deliveries in 2014–2015,\(^11\) the complications resulting from and associated with eclampsia remain significant. Eclampsia is strongly associated with maternal death, need for assisted ventilation, adult respiratory distress syndrome (RDS), acute renal failure, embolism and neonatal death, neonatal RDS and SGA infants.\(^12\)

All obstetrical care givers will eventually manage a woman with hypertension in pregnancy or with the complications arising from this condition. The management options for hypertensive disorders of pregnancy will vary according to resources. Geography, weather, and access to specialists or Level III hospitals may create conditions in which primary caregivers must respond to emergency situations such as stabilizing or treating women with hypertensive disorders of pregnancy.

Definitions and Terminology

Classification of the Hypertensive Disorders of Pregnancy\(^2\):

1. Chronic hypertension
2. Gestational hypertension
3. Preeclampsia–de novo (in a previously normotensive woman) or superimposed on chronic hypertension
4. White coat hypertension
### DEFINITIONS

<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>sBP ≥ 140 or dBP ≥ 90 mmHg (see “Diagnosis” section for measurement technique)</td>
</tr>
<tr>
<td>Severe hypertension</td>
<td>sBP ≥ 160 or dBP ≥ 110 mmHg</td>
</tr>
<tr>
<td>Chronic hypertension</td>
<td>Predates pregnancy or appears before 20 weeks' gestation</td>
</tr>
<tr>
<td>Gestational hypertension</td>
<td>New onset hypertension after 20 weeks' gestation, with no other maternal organ dysfunction</td>
</tr>
<tr>
<td>Preeclampsia</td>
<td>Gestational or chronic hypertension with one or more of the following new onset conditions:</td>
</tr>
<tr>
<td>Early onset = &lt;34 wk; Late onset = ≥34 wk</td>
<td>1. proteinuria</td>
</tr>
<tr>
<td></td>
<td>2. other maternal organ dysfunction (renal, liver, neurologic, hematologic)</td>
</tr>
<tr>
<td></td>
<td>3. uteroplacental dysfunction (fetal growth restriction)</td>
</tr>
<tr>
<td>White coat hypertension</td>
<td>sBP ≥ 140 or dBP ≥ 90 mmHg in office/clinic, but lower with home or ambulatory BP monitoring</td>
</tr>
<tr>
<td>Eclampsia</td>
<td>The occurrence of seizures in a preeclamptic patient that cannot be attributed to other causes</td>
</tr>
</tbody>
</table>

### Incidence

It is estimated that the global incidence of preeclampsia ranges from 1% to 5.6%, while the incidence of eclampsia ranges from 0.1% to 2.9%. The highest rates are in Africa.

In Canada, the incidence of chronic hypertension in pregnancy, gestational hypertension and preeclampsia has remained relatively stable from 2004–2005 to 2010–2011. In the same time period, the incidence of eclampsia dropped from 1.5 to 0.8 per 1000 deliveries, and dropped further in 2014-2015 to 0.5 per 1000 deliveries.

<table>
<thead>
<tr>
<th>CONDITION</th>
<th>INCIDENCE IN PREGNANCY, %</th>
<th>RISK OF DEVELOPING PREECLAMPSIA, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preeclampsia</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Chronic hypertension</td>
<td>121</td>
<td>21</td>
</tr>
<tr>
<td>Gestational hypertension</td>
<td>5</td>
<td>2522</td>
</tr>
<tr>
<td>Gestational hypertension before 34 weeks' gestation</td>
<td>352324</td>
<td></td>
</tr>
<tr>
<td>Eclampsia</td>
<td>0.05</td>
<td></td>
</tr>
</tbody>
</table>
Pathogenesis

The exact etiology of preeclampsia is unknown. A prevalent theory describes a 2-stage process in which Stage 1 is inadequate placental perfusion, and Stage 2 is the maternal syndrome resulting from the materials generated by this insult.²⁹

Debate exists as to whether the underlying mechanism of preeclampsia is failure of the maternal cardiovascular system to adapt to pregnancy with required remodelling of arteries, reduction in arterial stiffness and enhanced endothelial function.³⁰-³² This theory further suggests that the nature of the hemodynamic abnormalities differs between early onset preeclampsia (decreased cardiac output and increased systemic vascular resistance) and late onset (increased cardiac output and relative reduced vascular resistance); these differences would guide the selection of antihypertensive medications.³⁴

Preeclampsia is likely not a single condition, but rather a condition with several subtypes. This would account for the varying clinical presentation and lack of highly useful predictive tests or preventive interventions.³⁰

Figure 1. Possible etiology of preeclampsia

Abnormal placentation or excessive fetal demands

Mismatch between uteroplacental supply and fetal demands

Maternal endothelial cell dysfunction

Maternal and fetal manifestations of preeclampsia
Morbidity and mortality

According to the Canadian Perinatal Surveillance System, the overall maternal mortality rate in Canada was 7.4 per 100 000 deliveries in 2015. It has fluctuated between 5.1 and 11.9 since 1999. Hypertensive disorders of pregnancy are the second most common cause of direct maternal mortality. Eclampsia is associated with need for assisted ventilation, adult RDS, acute renal failure, and embolism.

Table 1. Diagnosis Associated With Maternal Deaths

<table>
<thead>
<tr>
<th>DIAGNOSIS</th>
<th>NUMBER OF MATERNAL DEATHS</th>
<th>MATERNAL DEATHS PER 100 000 HOSPITAL DELIVERIES (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diseases of the circulatory system</td>
<td>89</td>
<td>2.5 (2.0-3.1)</td>
</tr>
<tr>
<td>Other indirect causes</td>
<td>78</td>
<td>2.2 (1.7-2.7)</td>
</tr>
<tr>
<td>Postpartum hemorrhage</td>
<td>49</td>
<td>1.4 (1.0-1.8)</td>
</tr>
<tr>
<td>Hypertension complicating pregnancy, childbirth and the puerperium</td>
<td>42</td>
<td>1.2 (0.8-1.6)</td>
</tr>
<tr>
<td>Obstetric embolism</td>
<td>39</td>
<td>1.1 (0.8-1.5)</td>
</tr>
<tr>
<td>Major puerperal infection</td>
<td>27</td>
<td>0.8 (0.5-1.1)</td>
</tr>
<tr>
<td>Ectopic and molar pregnancy /abortive outcome</td>
<td>26</td>
<td>0.7 (0.5-1.1)</td>
</tr>
<tr>
<td>Antepartum hemorrhage, placental abruption, and placenta previa</td>
<td>21</td>
<td>0.6 (0.3-0.9)</td>
</tr>
</tbody>
</table>

Source: Canadian Institute of Health Information (CIHI), Discharge Abstract Database. Used with permission.

* Quebec does not contribute to the Discharge Abstract Database. Manitoba data, which were incomplete for earlier years, were included from 2004/2005. CI – Confidence interval.
Figure 2. Rate of the most common severe maternal morbidities in Canada

1. Blood transfusion
2. Blood transfusion with comorbidity
3. Postpartum hemorrhage and blood transfusion
4. Cardiac arrest/failure, myocardial infarction or pulmonary edema including ICD-10 O75.4 (other complications of obstetric surgery and procedures)
5. Embolization or ligation of pelvic vessels or suturing of uterus and postpartum hemorrhage
6. Hysterectomy
7. Uterine rupture during labour
8. Puerperal sepsis
9. Repair of bladder, urethra, or intestine
10. Eclampsia
11. Postpartum hemorrhage and hysterectomy
12. Placenta previa with hemorrhage and blood transfusion
13. Cardiac arrest/failure, myocardial infarction or pulmonary edema excluding ICD-10 O75.4 (other complications of obstetric surgery and procedures)

Source: Canadian Institute for Health Information-Discharge Abstract Database (CIHI-DAD).
Data for Quebec were excluded because they do not contribute to CIHI-DAD.
CI = confidence interval

Perinatal health indicators for Canada 2017: a report from the Canadian Perinatal Surveillance System.
Morbidity and Mortality with Preeclampsia

All caregivers must be able to promptly recognize the signs, symptoms, and laboratory findings of preeclampsia. Preeclampsia has potential for multi-organ involvement and carries risks for fetal, perinatal, and maternal morbidity and mortality.

<table>
<thead>
<tr>
<th>MATERNAL</th>
<th>FETAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroke (risk is with sBP ≥ 160 mm Hg)</td>
<td>Oligohydramnios</td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td>Intrauterine growth restriction</td>
</tr>
<tr>
<td>Hepatic failure</td>
<td>NICU admission</td>
</tr>
<tr>
<td>Jaundice</td>
<td>Prematurity due to maternal indication for delivery</td>
</tr>
<tr>
<td>Seizure (eclampsia)</td>
<td>Fetal death (3-fold increase)</td>
</tr>
<tr>
<td>Placental abruption</td>
<td></td>
</tr>
<tr>
<td>Acute renal failure</td>
<td></td>
</tr>
<tr>
<td>Maternal death</td>
<td></td>
</tr>
</tbody>
</table>

Morbidity and Mortality With Chronic Hypertension

Patients with chronic hypertension will develop superimposed preeclampsia in 21% of cases. Those who do NOT develop preeclampsia are still at increased risk of requiring Caesarean section (rate 41%), perinatal mortality (4 per 1000), and having an SGA baby (< 2500 gm; 17%). Neonates are at increased risk of NICU admission (20%), and of congenital anomalies, particularly cardiac, independent of use of antihypertensive medications.

Preconception counselling is valuable for optimizing BP control with medications that are safe to use during pregnancy, and for initiating administration of ASA for risk reduction (see “Management” and “Risk Reduction” sections, below).

Diagnosis and Evaluation

Clinical Evaluation

When blood pressure is measured, ensure that:

- The patient has a minimum rest period of 10 minutes after arrival.
- The patient is in a sitting position with her upper arm at heart level.
- The appropriate size cuff is used (a cuff too small overestimates the BP; a cuff too large underestimates BP). The length of the cuff should be 1.5 times the arm circumference.
- A blood pressure cuff is never placed over clothing.
• A mercury or aneroid sphygmomanometer is used; automated machines may underestimate systolic and diastolic BP by 10 to 5 mm Hg, especially in hypertensive patients. If an automated BP monitor is used, it should be calibrated against a mercury or aneroid sphygmomanometer. An RCT on follow-up using an automated BP machine (Omron) versus a Hg sphygmomanometer, demonstrated that, once the diagnosis of hypertension had been made, there were no differences in the outcomes of severe hypertension, nor in incidence of small for gestational age at delivery, in women who suffer hypertension in pregnancy. A blood pressure device which has been validated specifically for use in pregnancy and preeclampsia is the CRADLE Vital Signs Alert device.

• Korotkoff sound V (disappearance of pulse sounds) is used to define the diastolic pressure.

• If the BP is consistently higher in one arm, the arm with the higher values is used for all BP measurements.

Ambulatory blood pressure monitoring using an automated blood pressure machine is useful in ruling out “white coat” hypertension.

**Blood pressure criteria for a diagnosis of hypertension in pregnancy**

• A systolic BP of ≥ 140 mmHg or a diastolic BP of ≥ 90 mm Hg based on at least 2 measurements taken in the same arm ≥ 15 minutes apart after an initial rest period of > 10 minutes.

• Severe hypertension should be defined as a sBP ≥ 160 mm Hg or a dBP ≥ 110 mm Hg. A sBP over 160 mmHg is associated with increased risk of maternal stroke.

The following are not criteria for a diagnosis of hypertension in pregnancy

• An incremental and/or relative rise ≥ 30/15 mm Hg in systole or diastole is within the normal variation in BP seen throughout pregnancy, and is NOT a criterion for a diagnosis of hypertension.

• Mean arterial pressure is NOT used as a criterion to define hypertension in pregnancy because it is cumbersome to calculate.

There is a variation in dBP and sBP by gestational age. Typically, in nulliparous women, dBP drops to a nadir at 19 weeks while the sBP drops to a nadir at 17 weeks. In parous women, the nadir occurs at 20 weeks for diastole, and at 18 weeks for systole. In a large prospective study, women with high BMI had BP readings that were 4 mmHg higher than those with normal BMI.

Following confirmation of hypertension, assess for symptoms and signs suggesting involvement of other maternal organ systems (Table 2).
Table 2. Signs and symptoms that suggest involvement of other maternal organ systems

| CENTRAL NERVOUS SYSTEM                                      | • Presence of a severe headache  
|                                                          | • Visual disturbance (e.g., blurring, scotomata)  
|                                                          | • Tremulousness, irritability, somnolence  
|                                                          | • Hyperreflexia  
| CARDIORESPIRATORY                                         | • Chest pain  
|                                                          | • Dyspnea: check maternal O₂ saturation  
|                                                          | • Distended neck veins  
| HEMATOLOGIC                                               | • Bleeding  
|                                                          | • Petechiae  
| HEPATIC                                                   | • Right upper quadrant/epigastric pain  
|                                                          | • Severe nausea and vomiting  
| RENAL                                                     | • Reduced urine output (oliguria) < 15 mL/hr, is non-specific, has many causes, and is not diagnostic  
|                                                          | • Edema (including facial and dependent) and weight gain are NOT diagnostic criteria for preeclampsia  
|                                                          | • Proteinuria indicates glomerular dysfunction (See “Evaluation of Proteinuria” below)  

In a woman with preeclampsia, the following symptoms predict eclampsia: right upper quadrant pain, headache, visual disturbances, and vomiting. Epigastric pain predicts HELLP syndrome. In patients with established preeclampsia, epigastric pain and chest pain have been shown to be moderate predictors of adverse maternal and perinatal outcomes.

Investigations

All women with new onset hypertension after 20 weeks’ gestation should have, as a minimum, the following investigations:

- Platelet count
- AST (Aspartate Transaminase)
- Urinalysis, urine dipstick, or spot urine protein: creatinine ratio to screen for proteinuria
- Ultrasound assessment of fetal growth and amniotic fluid volume, plus umbilical artery Doppler studies if those are abnormal

If a patient develops thrombocytopenia (<150 x 10⁹/L) or dropping hemoglobin, testing for DIC and hemolysis is indicated.

Evaluation of Proteinuria

- Urine protein excretion of ≥ 300 mg/day (0.3g/day) on a 24-hour urine collection is the gold standard for a diagnosis of proteinuria.
• Proteinuria of ≥ 2+ on dipstick is highly suggestive of proteinuria in excess of 300 mg/24 hours and reliably establishes presence of proteinuria. Proteinuria of 1+ on dipstick should be confirmed by a 24-hour urine collection because of significant false positive and false negative results. Proteinuria of 1+ on dipstick should be confirmed by a 24-hour urine collection because of significant false positive and false negative results.51, 52
• A urinary protein to creatinine ratio (UPCR) at a level ≥ 30 mg/mmol urinary creatinine in a spot (random) urine sample is suggestive of proteinuria. A UPCR level of < 30 mg/mmol rules out proteinuria.
  – measurement of the UPCR offers a less cumbersome alternative to a 24-hour urine collection.
  – samples that are not first void samples were found to have a sensitivity of 90%, specificity of 100%, a negative LR of 0.1 with an AUC of 1.0.53

NOTE
While the presence of proteinuria has a direct impact on maternal and perinatal outcomes, the degree of proteinuria (high versus low) has no impact on worsening maternal nor perinatal outcomes. Therefore, once proteinuria >300mg/24 hours has been documented, there is no need to repeat this measurement.

Routine urinalysis should not be used to screen for protein at all prenatal visits in asymptomatic, normotensive, low-risk women, as there is no evidence that it provides earlier detection of preeclampsia or improves outcome when compared to blood pressure measurement alone.55, 56

NOTE
Early onset (< 34 weeks) preeclampsia or severe hypertension warrants consideration of further investigations looking for underlying conditions or alternate diagnoses (e.g., antiphospholipid antibody syndrome).57

Appendix A provides the differential diagnoses associated with laboratory abnormalities encountered in women with preeclampsia (Magee et al.36).

Ongoing Evaluation

Some women who develop a hypertensive disorder of pregnancy will require immediate delivery, particularly those with preeclampsia demonstrating severe features:

• Inability to control maternal blood pressure
• Worsening maternal organ dysfunction (hepatic, renal, neurologic symptoms, HELLP*) (*Hemolysis, Elevated Liver Enzymes, Low Platelets)
• Fetal indication for delivery

or, those who are close to term. (See “Timing of Delivery” section)

Others, remote from term, might be observed, with ongoing consultation, recognizing that deterioration can occur quickly. Table 3 provides a guideline for minimum frequency of reassessment.58 Care must be individualized and the
plan of care reassessed at each visit. Women diagnosed with preeclampsia may require hospitalization at the time of diagnosis.

**Table 3. Minimum Frequency of Reassessment**

<table>
<thead>
<tr>
<th></th>
<th>PREECLAMPSIA</th>
<th>GESTATIONAL HYPERTENSION</th>
<th>CHRONIC HYPERTENSION</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Blood pressure</strong></td>
<td>Twice daily</td>
<td>Minimum 2x/week, or home BP monitoring</td>
<td>Each visit</td>
</tr>
<tr>
<td><strong>Proteinuria screen</strong></td>
<td>daily until &gt;300mg/24h, then D/C</td>
<td>1x/week</td>
<td>Each visit</td>
</tr>
<tr>
<td><strong>CBC, liver enzymes</strong></td>
<td>2x/week, or more frequent if unstable</td>
<td>If sharp ↑BP or proteinuria develops</td>
<td>If sharp ↑BP or proteinuria develops</td>
</tr>
<tr>
<td><strong>NST, amniotic fluid volume</strong></td>
<td>NST daily</td>
<td>1x/week</td>
<td>Each visit in 3rd trimester</td>
</tr>
<tr>
<td></td>
<td>AFV 2x/week</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ultrasound growth/AFV</strong></td>
<td>Weekly; more frequent if abnormal</td>
<td>Every 2-4 weeks; more frequent if abnormal</td>
<td>Start of 3rd trimester; repeat if AFV or SF height low</td>
</tr>
<tr>
<td></td>
<td>+/-Doppler</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*urine dipstick, urinalysis or UPCR; begin 24h urine collection if ≥ 2+

Inform women with preeclampsia that they should report any decrease in fetal movement, or any new headache, vision change, right upper quadrant or epigastric pain, or shortness of breath.
Risk Factors and Risk Reduction

Predicting Onset of Preeclampsia

Maternal characteristics and maternal history will identify 30% of women who develop preeclampsia. Risk markers of greater importance are highlighted in grey in Table 4. Those in bold within the shaded areas confer the highest risk.

Table 4. Risk markers for preeclampsia

<table>
<thead>
<tr>
<th>First trimester markers</th>
<th>Second or third trimester markers</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographics</strong></td>
<td><strong>Past history</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Current pregnancy</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Multiple pregnancy</strong></td>
</tr>
<tr>
<td>Maternal age</td>
<td>Obesity (BMI ≥ 35 kg/m²)</td>
</tr>
<tr>
<td>&gt;40 yrs or &lt;18 yrs</td>
<td>Family history of preeclampsia</td>
</tr>
<tr>
<td></td>
<td>(mother or sister)</td>
</tr>
<tr>
<td></td>
<td><strong>First ongoing pregnancy</strong></td>
</tr>
<tr>
<td></td>
<td>• First visit sBP ≥ 130 mm Hg</td>
</tr>
<tr>
<td></td>
<td>• dBP ≥ 80 mm Hg</td>
</tr>
<tr>
<td></td>
<td><strong>Inter-pregnancy interval ≥ 10 yrs</strong></td>
</tr>
<tr>
<td></td>
<td>• New partner (first pregnancy or short duration of exposure)</td>
</tr>
<tr>
<td></td>
<td><strong>Systolic BP &gt; 120 mm Hg</strong></td>
</tr>
<tr>
<td></td>
<td>Abnormal prenatal genetic screen (IPS, FTS or MSS) analytes</td>
</tr>
<tr>
<td></td>
<td>Abnormal uterine artery Doppler velocimetry</td>
</tr>
<tr>
<td></td>
<td>Excessive weight gain in pregnancy</td>
</tr>
<tr>
<td></td>
<td>Cardiac output &gt; 7.4 L/min</td>
</tr>
<tr>
<td>• Ethnicity: Nordic, Black, South Asian, or Pacific Island</td>
<td></td>
</tr>
<tr>
<td>• Lower socioeconomic status</td>
<td>Non-smoking</td>
</tr>
<tr>
<td></td>
<td>Increased pre-pregnancy triglycerides</td>
</tr>
<tr>
<td></td>
<td>Family history of early-onset cardiovascular disease</td>
</tr>
<tr>
<td></td>
<td>Cocaine and/or methamphetamine use</td>
</tr>
<tr>
<td></td>
<td>Inter-pregnancy interval &lt; 2 yrs</td>
</tr>
<tr>
<td></td>
<td>Reproductive technologies to conceive (subfertility)</td>
</tr>
<tr>
<td></td>
<td>New partner (first pregnancy or short duration of exposure)</td>
</tr>
<tr>
<td></td>
<td>Gestational trophoblastic disease</td>
</tr>
<tr>
<td></td>
<td>Infection during pregnancy (e.g., UTI, periodontal disease)</td>
</tr>
<tr>
<td>• Previous preeclampsia</td>
<td>• Inter-pregnancy interval ≥ 10 yrs</td>
</tr>
<tr>
<td>• Anti-phospholipid antibodies</td>
<td>• First visit sBP ≥ 130 mm Hg</td>
</tr>
<tr>
<td>• Pre-existing medical condition(s)</td>
<td>• dBP ≥ 80 mm Hg</td>
</tr>
<tr>
<td>- Hypertension or first visit dBP ≥ 90 mm Hg</td>
<td>• New partner (first pregnancy or short duration of exposure)</td>
</tr>
<tr>
<td>- Renal disease or first visit proteinuria</td>
<td>Gestational trophoblastic disease</td>
</tr>
<tr>
<td>- Diabetes mellitus</td>
<td>Infection during pregnancy (e.g., UTI, periodontal disease)</td>
</tr>
<tr>
<td>- Collagen vascular disease</td>
<td>• Abnormal uterine artery Doppler velocimetry</td>
</tr>
<tr>
<td>- Periodontitis</td>
<td>Excessive weight gain in pregnancy</td>
</tr>
<tr>
<td>• Multiple pregnancy</td>
<td>Cardiac output &gt; 7.4 L/min</td>
</tr>
<tr>
<td>• First ongoing pregnancy</td>
<td>Elevated uric acid</td>
</tr>
</tbody>
</table>
Numerous studies continue to examine the usefulness of trophoblast and angiogenic markers, as well as uterine artery Doppler studies in first or second trimester, to predict the onset of preeclampsia. However, these are not currently recommended as part of regular clinical practice.

Abnormal levels of some of these markers, such as low PAPP-A (pregnancy associated placental protein A), low PlGF (placental growth factor) or high total HCG (human chorionic gonadotropin) may be reported as part of an otherwise normal prenatal genetic screening test. Women with such results, particularly with multiple abnormal biomarkers, warrant increased surveillance for hypertensive disorders in pregnancy.

Placental growth factor (PlGF) should not be ordered as an early screening test for preeclampsia.

**Predicting Maternal and Perinatal Morbidity and Mortality with Established Gestational Hypertension and Preeclampsia**

Earlier gestational age, chest pain or dyspnea, low PaO$_2$, low platelets, elevated creatinine, and elevated AST have been shown to have an excellent predictive value of adverse maternal outcomes occurring within 48 hours and up to 7 days (AUC ROC 0.88). In one multicentre study, an oxygen saturation (SpO$_2$) of less than 93% was found to be highly predictive of poor maternal and neonatal outcome within 48 hours. The PIERS (Preeclampsia Integrated Estimate of Risk) study group created a calculator where the above values can be entered for a given patient (available at: [https://pre-empt.cfri.ca/monitoring/fullpiers](https://pre-empt.cfri.ca/monitoring/fullpiers)), including those with early onset preeclampsia.

The degree of proteinuria has not been found to be an independent predictor of adverse maternal or perinatal outcomes.

Elevated levels of uric acid are not meaningfully associated with adverse maternal or perinatal outcome, so should not determine timing of delivery.

**Risk Reduction**

**Calcium Supplementation**

Calcium supplementation (≥ 1 g/day) or an increase in dietary calcium intake (3 to 4 dairy servings per day) has been shown to decrease the rate of preeclampsia only in low- and middle-income country populations with a low dietary intake of calcium (< 600 mg/day). A large multicentre study in the United States showed no benefit of calcium supplementation in reducing the rate of preeclampsia.

**Low Dose ASA**

Low dose ASA decreases the risk of preeclampsia in high risk populations, particularly those with chronic hypertension, diabetes mellitus or previous preterm preeclampsia. It should be **STARTED** after diagnosis of pregnancy, and **BEFORE 16 WEEKS** whenever possible, to obtain the greatest benefit. It is continued until term. It is taken once daily at bedtime.
The **OPTIMAL DOSE** of ASA has been the subject of much research. 162mg per day appears to decrease the risk of preterm preeclampsia more than the commonly-used 81mg in those who start it before 16 weeks gestation:

- Meta analyses of studies using 50-150mg daily had shown an overall 10-17% decrease in preeclampsia,\(^1\) with a dosage of <75mg per day providing no significant benefit.\(^2\)
- 81 mg/day changes platelet function in just 71% of pregnant women, while a dosage of 162mg/day alters platelet function in 97%.\(^3\)
- On this basis, the 2017 ASPRE trial used 150mg daily, randomizing high risk women at 11-14 weeks gestation. In the treated group, the Odds Ratio for delivery with preeclampsia at <37 weeks gestation was 0.38.\(^4\)

**WHO** should be offered low dose ASA, based on risk factors that can be identified before 16 weeks gestation, is also an area of much research. The following table is one example of a tool which can be used: Women with 1 or more Major Risk Factor, as well as those who have 2 or more Moderate Risk Factors, have a preeclampsia risk of about 3.5%, or a Relative Risk of about 5, compared to those with no risk factors. Evidence is limited as to which women within these groups would benefit and to what degree.

<table>
<thead>
<tr>
<th>MAJOR RISK FACTOR</th>
<th>MODERATE RISK FACTOR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antiphospholipid Antibody syndrome</td>
<td>Prior Placental Abruption</td>
</tr>
<tr>
<td>Chronic Hypertension</td>
<td>Multifetal pregnancy</td>
</tr>
<tr>
<td>Prior Preeclampsia</td>
<td>Chronic Kidney disease</td>
</tr>
<tr>
<td>Pregestational Diabetes</td>
<td>Prior Stillbirth</td>
</tr>
<tr>
<td>Pre-Pregnancy BMI &gt; 30kg/m(^2)</td>
<td>Maternal age &gt; 40 years</td>
</tr>
<tr>
<td>Assisted Reproductive Technology</td>
<td>Nulliparity</td>
</tr>
<tr>
<td></td>
<td>Systemic Lupus Erythematous</td>
</tr>
</tbody>
</table>

(Adapted from Bartsch et al\(^5\) and Viguiliouk et al\(^6\))

In a low risk population, primary prevention of preeclampsia with ASA remains unproven at the present time. It is currently a topic of research.

There is no evidence of adverse effects of low dose ASA on either the mother or newborn.\(^7\)\(^,\)\(^8\)

**Management**

In general, management includes evaluation of the mother and fetus; prevention of severe maternal complications (organ damage, seizure, cerebral vascular accidents, deep vein thrombosis, and death) and severe fetal complications (placental abruption, growth restriction, and stillbirth); symptomatic support; and delivery.
Antihypertensive Therapy

Antihypertensive medication reduces the mother’s risk of developing severe hypertension and its potential sequelae (cerebrovascular accident). It does not necessarily reduce the risk of seizures (eclampsia) or prevent adverse fetal outcomes such as IUGR. While the acute management of a hypertensive crisis to prevent a maternal cerebrovascular accident is critical, too rapid a drop in maternal BP may cause a reduction in utero-placental perfusion resulting in fetal compromise. Antihypertensive therapy should aim to reduce the systolic BP to < 160 mm Hg and the diastolic BP to < 110 mm Hg over a few hours. 21

There is insufficient evidence to determine the ideal BP associated with optimal maternal and perinatal outcomes. A reasonable goal is sBP 130-155mmHg and dBP 80-105mmHg. 36

In the presence of some comorbidities, such as pre-existing renal disease, there may be indications for lower target blood pressures. The medications available can be divided into those used for acute (severe hypertension) and those for maintenance therapy.

**Acute therapy (severe hypertension)**

An obstetric consultation should be obtained to guide therapy when severe hypertension is diagnosed.

Management includes immediate, intensive medical treatment with intravenous access and maternal/fetal monitoring. It is recommended that all perinatal units have a management protocol for the treatment of severe hypertension.

Intravenous labetalol, intravenous hydralazine, and oral nifedipine are commonly used to treat acute, severe hypertension (Table 5). The pharmacokinetics of intravenous labetalol and intravenous hydralazine are very similar. The first choice is either oral nifedipine or IV labetalol because hydralazine has been associated with more adverse outcomes including maternal hypotension, placental abruption, abnormal fetal heart rate patterns, maternal oliguria, and need for Caesarean section. 91

**Table 5. Treatment for acute hypertension**

| AGENT    | DOSE                                      | ROUTE | ONSET  | PEAK   | CAUTION                                                                
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Labetalol</td>
<td>Initial dose 20mg; repeat 20–80 mg every 30 minutes; maximum 300mg</td>
<td>IV</td>
<td>5 minutes</td>
<td>30 minutes</td>
<td>Contraindicated in women with asthma or heart failure; May cause neonatal bradycardia</td>
</tr>
<tr>
<td>Nifedipine</td>
<td>Initial dose 10 mg; repeat 10–20 mg every 45 minutes; maximum 50 mg</td>
<td>Swallowed (not chewed)</td>
<td>30 minutes</td>
<td>45 minutes</td>
<td>Immediate release preparation is used here. (PA no longer available in Canada, and XL not for acute therapy.)</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>Initial dose 5mg; repeat 5–10mg every 30 minutes; maximum 20 mg</td>
<td>IV</td>
<td>5 minutes</td>
<td>30 minutes</td>
<td>May increase the risk of maternal hypotension</td>
</tr>
</tbody>
</table>

References: 36, 47, 58, 92
When the Maximum dose listed above has been given, consider addition of a second antihypertensive medication. For refractory cases, IV infusion of labetalol and/or hydralazine may be considered.

**NOTE**

Nifedipine and magnesium sulphate may be used at the same time.

Although they are both calcium antagonists, concurrent use was NOT shown to cause a potentiation of the hypotensive effect in a large retrospective review.\(^{93}\)

In management of severe hypertension, oral labetalol and oral methyldopa are less effective in lowering BP than Nifedipine. However, they remain reasonable choices when Nifedipine is not available or intravenous access is not feasible.\(^{94}\)

**Maintenance therapy (chronic hypertension)**

There is insufficient evidence to determine the ideal ongoing BP levels associated with optimal maternal and perinatal outcomes for women with chronic hypertension. Blood pressures levels that are too low may compromise placental perfusion, so suggested targets are sBP 130-155 mmHg and dBP 80-105 mmHg. Women entering pregnancy on antihypertensive medications may need the dose lowered or medication(s) stopped completely to remain within this range.\(^{95}\)

A 2018 Cochrane review (including 31 studies) showed that treating hypertension below the severe range (< sBP 160 and < dBP 110) decreases the risk of progression to severe hypertension by one half, but has little or no impact on development of preeclampsia, fetal or neonatal death including miscarriage, small for gestational age infants, or preterm birth below 37 weeks.\(^{96}\)

Women with underlying medical conditions, such as renal disease, may have lower target blood pressures, determined on a case by case basis.

**Maintenance therapy (gestational hypertension)**

Target blood pressures are the same as for chronic hypertension.

**NOTE**

About 25% of women with chronic or gestational hypertension will develop preeclampsia. Lowering blood pressure does not change this, as hypertension is only one (late) manifestation of a complex underlying process (see “Pathogenesis” section, above). Hence, ongoing close follow-up is critical.
Table 6 shows oral antihypertensive medications commonly used in Canada. No differences in maternal or fetal outcomes have been demonstrated. 26

Table 6. Maintenance therapy

<table>
<thead>
<tr>
<th>AGENT</th>
<th>DOSE</th>
<th>CAUTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Labetalol</td>
<td>Initial dose 100 mg twice a day;</td>
<td>Contraindicated in women with asthma.</td>
</tr>
<tr>
<td></td>
<td>Maximum 400 mg 3 times a day (1200mg/day)</td>
<td></td>
</tr>
<tr>
<td>Nifedipine XL</td>
<td>Initial dose 20 mg once daily;</td>
<td>Contraindicated in women with aortic stenosis</td>
</tr>
<tr>
<td></td>
<td>Maximum 60 mg twice a day</td>
<td>Ensure XL Preparation</td>
</tr>
<tr>
<td>Methyldopa</td>
<td>Initial dose 250 mg twice a day;</td>
<td>May cause depression</td>
</tr>
<tr>
<td></td>
<td>Maximum 500 mg 4 times a day (2g/day)</td>
<td></td>
</tr>
</tbody>
</table>

References: 26, 58, 91

Note

Angiotensin converting enzyme inhibitors and angiotensin receptor blockers are contraindicated because of IUGR, prematurity and oligohydramnios. They are not teratogens, but disturb fetal and neonatal renal function. Women who conceive on them should be switched to alternate antihypertensive medications as soon in pregnancy as possible. 101, 102

Atenolol is not recommended because of increased rates of IUGR among infants born to women taking Atenolol at conception or during first trimester. 103

Beta-blockers taken in late pregnancy increase the risk of neonatal hypoglycemia and bradycardia (adjusted Odds Ratio for both <2). 104

There is an increased risk of congenital anomalies overall (OR 1.3), and congenital heart defects specifically (OR 1.6), in women with chronic hypertension, independent of their use of antihypertensive medications. This suggests an underlying association between maternal hypertension and congenital anomalies, independent of the adverse effects of some medications. 105, 106

Fluid Management

Hypertensive women may not tolerate large fluid volume shifts. Iatrogenic pulmonary edema is a concern because of the large amounts of intravenous fluids that may be inadvertently administered intrapartum. Intravenous and oral fluid intake should be limited in women with preeclampsia to avoid pulmonary edema. The standard intravenous fluid bolus routinely administered before regional anaesthesia should not be given. The type of fluid is not as critical as the volume of fluid. Hypotension and shock may develop with lesser degrees of hemorrhage because of vascular space contraction.
Urine output is best monitored by an indwelling Foley catheter. A urine output < 15 mL/hour is not unusual in preeclampsia, particularly postpartum. In the absence of pre-existing renal disease or a rising creatinine level, oliguria should be tolerated at least for a few hours. The UK Confidential Enquiry into Maternal Deaths found that excess maternal mortality is associated with aggressive fluid use and not with transient renal compromise. In the presence of oliguria, a careful assessment of volume status and renal function is indicated. When a patient is undergoing medical induction of labour and is receiving MgSO\(_4\) with oxytocin, it is prudent to limit IV fluid intake by concentrating the solutions of oxytocin and MgSO\(_4\). Hourly total intake and urine output must be monitored closely in this situation to prevent pulmonary edema.

**Recommendations in the presence of oliguria (< 15 mL/hour):**

- Clinically assess volume status
- Measure renal function (creatinine)
- Beware of magnesium toxicity
- Consider a small fluid bolus (500 mL normal saline)
- Monitor O\(_2\) saturation (keep > 95%)
- Beware of pulmonary edema
- Consider consultation, if oliguria persists and creatinine is rising

Dopamine or furosemide should not be administered in the presence of persistent oliguria occurring before delivery. (See “Postpartum Management” section regarding furosemide use after delivery.)

Thromboprophylaxis should be considered for all patients who are immobilized or bedridden for prolonged periods.

**Symptomatic Support**

Immediate treatment should include managing symptoms such as nausea and vomiting with an antiemetic to minimize maternal discomfort. A component of maternal hypertension is adrenergic and may be modified by stress reduction.

There is no evidence to support strict bed rest in the lexicon of therapeutic management of hypertensive disorders of pregnancy. Such an intervention is harmful.

**Principles of stress reduction:**

- Quiet environment
- Presence of a supportive family member or professional
- Clear explanation of management plan to patient / family
- Minimization of negative stimuli
- Consistent, confident team approach (nursing, obstetrics, midwifery, anesthesiology, hematology, pediatrics)
Seizure Prophylaxis

Prevention of seizures is crucial in stabilizing a woman who has preeclampsia. As seizures are rare, there is a high number needed to treat to prevent a seizure. Neither maternal symptoms nor blood pressure levels reliably predict seizures; however, because of side effects and cost, selective use of seizure prophylaxis is generally recommended for women with:

- Severe hypertension
- Blood pressures below the severe range but with associated
  - significant headache or clonus
  - visual disturbance (blurring, scotomata)
  - right upper quadrant or epigastric pain (severe and persistent), and/or elevated liver enzymes
  - thrombocytopenia (<100 x 10⁹/L)
  - progressive renal insufficiency (doubling of serum creatinine)
- HELLP syndrome
- Secondary prevention after eclamptic seizures

Magnesium sulphate (MgSO₄) is the agent of choice when seizure prophylaxis is indicated. MgSO₄ is superior to phenytoin for the prevention of seizures, and is superior to either diazepam or phenytoin for preventing recurrent seizures. MgSO₄ reduces the incidence of seizures by 50%.

Magnesium Sulphate Administration

- **Intravenous** administration of MgSO₄ is preferred as therapeutic magnesium levels in the circulation are achieved rapidly. This is especially important after a seizure has occurred.
  - Dosage: 4 g as an IV bolus given over 20 to 30 minutes followed by 1 g/hour IV
  - A recurrent seizure may require a second 2 to 4 g IV bolus
- **Intramuscular**: When intravenous access is unavailable, MgSO₄ may be given intramuscularly (IM). The process for IM MgSO₄ administration is:
  - Initial dosage: 10 g of 50% MgSO₄, one half (5 g) injected deeply in the upper, outer quadrant of both buttocks through a 3-inch, 20-gauge needle (spinal needle). Maintenance dosage: 5 g of 50% solution of magnesium sulphate by deep IM injection in the upper, outer quadrant of alternate buttocks every 4 hours after the initial 10 g dose.
  - The Intramuscular regimen may offer a safer alternative than an IV infusion for patients being transported between health care facilities. The above dosages can be used. Another option shown to be safe is: 4g IV bolus and 10g IM loading dose (at the same time) followed by 5g IM every 4 hours (called the Pritchard regimen).
- **Side effects** of MgSO₄: weakness, paralysis, cardiac toxicity, loss of patellar reflexes, respiratory depression. The data, however, show that these risks are very low.
- **Monitor** reflexes, respiration, level of consciousness, hourly urine output. Magnesium is excreted in the urine. Clinically, magnesium serum levels can be estimated as follows:
BLOOD LEVEL MMOL/L

<table>
<thead>
<tr>
<th>Reflexes present</th>
<th>2 to 3.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss of patellar reflexes</td>
<td>4 to 5</td>
</tr>
<tr>
<td>Respiratory depression</td>
<td>≥ 6</td>
</tr>
</tbody>
</table>

- Routine monitoring of serum magnesium levels is not supported by evidence.  
- Although MgSO₄ should be used with caution when combined with calcium channel blockers (i.e., nifedipine) and in women in renal failure, the risk of complications is low (< 1%). Therefore, calcium channel blockers may be used simultaneously with MgSO₄.
- If toxicity is suspected, discontinue the medication, provide respiratory support, notify the primary health care provider, consider giving calcium gluconate, and monitor the blood level of magnesium. The antidote to magnesium is 10 mL of 10% calcium gluconate, IV over 3 minutes.

The incidence of respiratory suppression with the IV regimen above (4 gram IV bolus over 20 minutes then 1 gram per hour) is less than 1%. Therefore, if toxicity has developed, it is essential to confirm that the appropriate concentration is being infused at the correct rate, and that urine output is adequate. Following this, the magnesium sulphate infusion can be resumed at a lower rate.

**Points to remember in management of seizures (eclampsia):**

- Call for help
- Turn the woman on her side
- Protect the airway
- Start an IV MgSO₄ bolus of 4 g over 20 to 30 minutes and then a maintenance dose at 1 g/hour IV (if recurrent seizure while on MgSO₄, re-bolus with 2 g IV over 20 to 30 minutes)
- When seizure stops, administer oxygen by face mask, clear airway as required, assess BP, pulse, respiration, and fetal heart rate frequently until stable
- Assess for evidence of placental abruption
- Be aware that patients with eclampsia are at risk of developing deep vein thrombosis, cerebrovascular accident, or cardiomyopathy after the seizures

**Protocols for the use of magnesium sulphate should be established, be immediately available in every labour and delivery unit, and include the following:**

- Preparation
- Assessments before administration
- Administration protocol
- Assessment for side effects and drug interactions
- Management of toxicity
- Documentation
Management of HELLP Syndrome

HELLP syndrome consists of Hemolysis, Elevated Liver enzymes (AST, ALT and/or LDH), Low Platelet count.

The management of HELLP syndrome consists of all therapeutic steps for hypertension described above. In addition, the thrombocytopenia may require specific intervention based on severity:

- Platelet count >50x10^9/L: If there is no evidence of platelet dysfunction or excessive bleeding, prophylactic platelet transfusion is not indicated, even before Caesarean section.
- Platelet count <50x10^9/L: platelet count is falling, or a coagulopathy exists: consider blood product/platelet transfusion.
- Platelet count <20x10^9/L: Platelet transfusion before both Caesarean section and vaginal delivery.

Delivery should be undertaken within 48 hours of diagnosis. There are no differences in maternal or perinatal outcomes when immediate delivery versus expectant management of 48 hours followed by delivery were compared. However, expectant management of greater than 48 hours was associated with an increased risk for maternal admission to the ICU.

Patients with HELLP syndrome are at risk of developing eclampsia, hence MgSO₄ should be administered.

Transport

When local resources are limited and maternal and fetal conditions permit, the outcome may be improved by transporting the mother to an appropriate referral centre.

A transport protocol should be readily available in every unit. Before transport, the following must be confirmed:

- Maternal blood pressure is stable
- Fetal condition is stable
- Seizure prophylaxis is given if necessary. Consider MgSO₄ IM dose (see above) for safety during transport.
- Ventilation equipment and calcium gluconate are available during transport
- The woman is accompanied during transport by a health care provider with the skills and qualifications to ventilate and to administer any necessary medications

Delivery

Delivery of the placenta is the only cure for preeclampsia. Any of the hypertensive disorders of pregnancy can progress quickly to endanger mother and infant. Timely delivery minimizes maternal and neonatal morbidity and mortality. Delivery is based on the following:

- Maternal status must be optimized before intervening with a delivery process
- Delay of delivery, to allow transfer, should occur only when maternal and fetal conditions permit
- Corticosteroid therapy to enhance fetal pulmonary maturity should be considered for all women with preeclampsia before 34th weeks' gestation
• Expectant management is potentially harmful
• Maternal status can worsen, and eclampsia can still occur after delivery

Timing of Delivery

Women with the hypertensive disorders of pregnancy are a heterogeneous group who vary greatly in the severity and stability of their conditions. The following are broad guidelines regarding timing of delivery. For women who have chronic hypertension or gestational hypertension and whose BP is stable, ongoing expectant management is reasonable, provided close surveillance confirms maternal and fetal well-being.

<table>
<thead>
<tr>
<th>HYPERTENSIVE DISORDER</th>
<th>SUGGESTED TIME OF DELIVERY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic hypertension</td>
<td>≥ 38 weeks</td>
</tr>
<tr>
<td>Gestational hypertension</td>
<td>≥ 37 weeks</td>
</tr>
<tr>
<td>Preeclampsia without severe features (see below)</td>
<td>37 weeks</td>
</tr>
<tr>
<td>Preeclampsia with severe features:</td>
<td>Deliver regardless of gestational age.</td>
</tr>
<tr>
<td>• Inability to control maternal blood pressure</td>
<td></td>
</tr>
<tr>
<td>• Increasing maternal organ dysfunction (hepatic, renal, neurologic symptoms, HELLP)</td>
<td></td>
</tr>
<tr>
<td>• Fetal indication for delivery</td>
<td></td>
</tr>
</tbody>
</table>

If expectant management is contemplated in a woman with preeclampsia and any of the above severe features, the pregnancy should be managed in a facility with sufficient resources for maternal intensive care support and for continuous monitoring. The facility should have the capability to intervene immediately and to manage a premature infant.

For uncomplicated chronic hypertension, gestational hypertension, and preeclampsia without any of the above severe features, delivery compared to expectant management between 34 and 36\( \frac{6}{12} \) weeks, showed no reduction in adverse maternal outcomes but a significant increase in RDS (RR 3, number needed to harm 25) with that risk being higher at 34 weeks than at 36\( \frac{6}{12} \) weeks.↑↑

Postpartum Management

Gestational hypertension and preeclampsia may present initially or worsen following delivery. The peak time for the appearance of hypertension postpartum is on days 3 to 6 when the mobilization of the extracellular fluid accumulated during pregnancy occurs.

The timing of seizure occurrence is distributed as follows: 50% first appear before labour, 25% first occur during labour, 25% begin in the early postpartum period. Rarely, a woman will have a seizure 2 days or more after delivery.
Women who have received magnesium sulphate for seizure prophylaxis during labour should have it continued for the first 24 hours postpartum. All women at risk for hypertensive disorders of pregnancy must be monitored carefully in the postpartum period with ongoing attention to blood pressure, renal function, seizure risk, and end-organ dysfunction. Laboratory investigations should be directed towards the particular end-organ that has been affected. Of note, for women who had severe hypertension in pregnancy, the addition of postpartum furosemide (e.g., 20 mg by mouth every 24 hours for 5 days) to the antihypertensive regimen appears beneficial in lowering BP and in reducing the need for other antihypertensive medications postpartum.\textsuperscript{124,127}

Postpartum thromboprophylaxis should be considered, particularly if there has been antenatal bed rest for more than 4 days, if the patient is obese, or if delivery has been by Caesarean section.

Severe postpartum hypertension should be treated with antihypertensive therapy to keep the systolic BP < 160 mm Hg and the diastolic BP < 110 mm Hg. In addition, antihypertensive therapy should be considered to treat non-severe postpartum hypertension, particularly in women with comorbidities. Antihypertensive agents acceptable for use in breastfeeding include nifedipine XL, labetalol, methyldopa, captopril, and enalapril.

Discharge from hospital should occur only when there is a clear trend towards improvement in clinical and laboratory assessments, when there is an ability to provide adequate outpatient surveillance, and when follow-up can be arranged within a week for clinical and blood pressure assessment. When there has been end-organ dysfunction, there should be evidence that it has resolved before the patient is discharged. It is reasonable to discharge women whose BP remains at < 160/110 mm Hg for at least 24 hours.\textsuperscript{36}

NOTE

Although rare, new onset preeclampsia has been documented up to 3 weeks after delivery in an otherwise normal pregnancy. Advanced maternal age, diabetes mellitus, obesity, and being of Black or Latina heritage seem to be associated risk factors for these occurrences.\textsuperscript{129}

Future Cardiovascular Risk

Women with a history of a hypertensive disorder of pregnancy have an increased risk of cardiovascular disease later in life. The risk is higher yet if their pregnancies were also complicated by preterm birth and growth restriction. If preeclampsia has recurred (as it does for 15% of women in a subsequent pregnancy), the RR for future CVD is about 2.5 compared to women with an unaffected subsequent pregnancy.\textsuperscript{129} Apart from recognition of this risk factor and encouragement of lifestyle modification, more research is needed to determine what cardiovascular screening or preventive treatment conveys long term benefit in these women.\textsuperscript{130,132} With regard to Lifestyle Risk Factors, being overweight or obese is associated with higher risk of chronic hypertension after a hypertensive disorder in pregnancy, while risk is not influenced by DASH diet, dietary sodium/potassium intake, or physical activity.\textsuperscript{131}

The Canadian Cardiovascular Society recommends that all women with a history of a hypertensive disorder in pregnancy “be approached for screening with a lipid profile”, but states that there is insufficient evidence to prescribe statins based solely on the history of a hypertensive disorder in pregnancy.\textsuperscript{134}
International guidelines differ with regard to when, how and whom to screen, hampered by the fact that the pathophysiology linking preeclampsia and cardiovascular disease is not completely understood.\(^\text{135}\)

### Points to Remember in Management

- Do not reduce blood pressure too rapidly or to too low a level: placental perfusion can be compromised. Keep systolic pressure <160 mmHg to prevent maternal stroke.
- Do not fluid overload: pulmonary edema is more dangerous than transient renal compromise.
- Do remember that MgSO\(_4\) decreases seizure risk by 50%
- Do use an interprofessional approach:
  - obstetrical care provider
  - anaesthesiologist
  - pediatrician and/or neonatologist
  - nurses
  - internal medicine specialist and/or hematologist

### Summary

1. Severe hypertension is an obstetrical emergency.
2. This clinical presentation requires prompt recognition, stabilization of the mother and fetus, and an interprofessional approach to management.
3. The primary obstetrical team in rural and remote areas may have to assume the roles of one or several disciplines until help or transfer is available.
4. The cure is delivery, but the decision to deliver is based on severity, maternal status, fetal maturity, and fetal well-being.
5. The rationale for antihypertensive treatment is to prevent maternal cerebrovascular accidents, not seizures.
6. Seizure prophylaxis, when indicated, should be with magnesium sulphate.
7. There is no evidence that antihypertensive therapy for hypertension below 160/110 improves perinatal outcome.
8. There are no investigations, beyond maternal history, that are clinically useful to predict preeclampsia.
9. Women at high risk for preeclampsia should be offered ASA 81-162mg per day, starting after the diagnosis of pregnancy and before 16 weeks gestation. Significant benefit has not been shown for women at low risk of preeclampsia.
10. Women with a history of a hypertensive disorder in pregnancy have an increased risk of cardiovascular disease later in life.
References


Appendix A

Laboratory and imaging abnormalities encountered in women with the hypertensive disorders of pregnancy

<table>
<thead>
<tr>
<th>INVESTIGATIONS FOR DIAGNOSIS</th>
<th>DESCRIPTION IN WOMEN WITH PREECLAMPSIA</th>
<th>DESCRIPTION IN WOMEN WITH OTHER CONDITIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MATERNAL TESTING</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urine testing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urinalysis (routine and microscopy with/without additional tests for proteinuria)</td>
<td>Proteinuria (as discussed under Proteinuria) without RBCs or casts</td>
<td>Hemoglobinuria (dipstick &quot;hematuria&quot; without RBCs): hemolytic anemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RBCs alone: renal stones, renal cortical necrosis (also associated with back pain and oliguria/anuria)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RBCs and/or casts are associated with other glomerular disease and scleroderma renal crisis and (about half of) TTP-HUS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bacteria: UTI or asymptomatic bacteruria</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Proteinuria is usually absent in secondary causes of hypertension such as pheochromocytoma, hyperaldosteronism, thyrotoxicosis, coarctation of the aorta, withdrawal syndromes</td>
</tr>
<tr>
<td>Oxygen saturation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulse oximetry</td>
<td>SpO₂ &lt;97% associated with a heightened risk of severe complications (including non-respiratory)</td>
<td>May be decreased in any cardiorespiratory complication (e.g., pulmonary embolism)</td>
</tr>
<tr>
<td>CBC and blood film</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>↑ due to intravascular volume depletion ↓ if microangiopathic hemolysis (with HELLP)</td>
<td>↑ due to volume depletion from any cause (e.g., vomiting) ↓ if microangiopathic hemolysis from other cause ↓ with any chronic anemia (nutritional or myelodysplasia) ↓ with acute bleeding of any cause</td>
</tr>
<tr>
<td>WBC and differential</td>
<td>↔</td>
<td>↑ due to neutrophilia of normal pregnancy ↑ with inflammation/infection ↑ with corticosteroids</td>
</tr>
</tbody>
</table>
INVESTIGATIONS FOR DIAGNOSIS | DESCRIPTION IN WOMEN WITH PREECLAMPSIA | DESCRIPTION IN WOMEN WITH OTHER CONDITIONS
--- | --- | ---
Platelet count | ↓ · associated with adverse maternal outcome | ↓ with gestational, immune (ITP), or thrombotic thrombocytopenia (TTP), APS, AFLP, myelodysplasia
Blood film | RBC fragmentation | Microangiopathy due to mechanical causes (e.g., cardiac valvopathy, cavernous hemangioma), DIC or other disorders of endothelial function (e.g., APS, TTP-HUS, vasculitis, malignant hypertension)

Tests of coagulation*

INR and aPTT | ↑ with DIC which is usually associated with placental abruption · ↑ is associated with adverse maternal outcome | May be ↑ in APS, DIC from other causes including sepsis, amniotic fluid embolism, stillbirth, massive hemorrhage, hemangiomas, shock · ↑ is prominent in AFLP
Fibrinogen | ↓ ↔ | ↓ with all causes of DIC including massive hemorrhage, genetic disorders · ↓ more profound with AFLP than with HELLP · Usually normal in TTP-HUS (ADAMTS13 vWF cleaving protein may be moderately decreased in HELLP but ADAMSTS 13 antibody should be absent)

Serum chemistry

Serum creatinine | ↑ due to hemoconcentration and/or renal failure · ↑ is associated with adverse maternal outcome | ↑ with other acute or chronic kidney disease · Renal failure prominent in malignant hypertension, TTP-HUS (along with thrombocytopenia), AFLP (along with liver dysfunction)
Serum uric acid | ↑ not meaningfully associated with adverse maternal or perinatal outcome* | ↑ with dehydration, medication (e.g., HCTZ), genetic causes
Glucose | ↔ | ↓ with AFLP, insulin therapy
AST or ALT | ↑ · associated with adverse maternal outcome | ↑ with AFLP and other ‘PET imitators’↑ but to a lesser degree, and usually normal in TTP-HUS · May be increased in other pregnancy-related conditions (e.g., intrahepatic cholestasis of pregnancy) or conditions not associated with pregnancy (e.g., viral hepatitis or cholecystitis)
LDH | ↑ which may be prominent – the ↑ is associated with adverse maternal outcome | ↑ with AFLP, intravascular hemolysis · ↑ LDH/AST ratio (> 22) with TTP-HUS

Hypertensive Disorders of Pregnancy 409
<table>
<thead>
<tr>
<th>INVESTIGATIONS FOR DIAGNOSIS</th>
<th>DESCRIPTION IN WOMEN WITH PREECLAMPSIA</th>
<th>DESCRIPTION IN WOMEN WITH OTHER CONDITIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bilirubin</td>
<td>↑ - unconjugated from hemolysis or conjugated from liver dysfunction</td>
<td>(early) ↑ in AFLP, ↑ with hemolytic anemia, other liver disease with dysfunction, genetic diseases</td>
</tr>
<tr>
<td>Albumin</td>
<td>↓ - associated with adverse maternal and perinatal outcomes</td>
<td>↓ as negative acute phase reactant with acute severe illness, malnutrition, nephritic syndrome, crystalloid infusion</td>
</tr>
</tbody>
</table>

**FETAL TESTING**

Abnormalities are not specific to the cause of poor placentation and/or placental dysfunction

- **Uterine artery Doppler velocimetry**
  - Unilateral/bilateral notching, or elevated pulsatility index or resistance index may support a diagnosis of placental insufficiency including preeclampsia

- **Fetal monitoring**
  - Abnormal or atypical FHR tracing (e.g., decreased variability)

- **Deepest amniotic fluid pocket**
  - Oligohydramnios associated with adverse perinatal outcomes

- **Ultrasoundographic assessment of fetal growth**
  - Usually intrauterine fetal growth restriction (typically asymmetrical but can be symmetrical if early and/or severe)

- **Umbilical artery Doppler**
  - Increased resistance, absent or reversed end-diastolic flow

- **Ductus venosus Doppler**
  - Increased resistance, especially absent or reverse “a” wave

- **Middle cerebral artery Doppler**
  - Cerebral redistribution (decreased resistance, or “brain sparing effect”). May be lost in extreme cases prior to fetal death

*Note: This is a modification from the original table. Adapted with permission.*

**AFLP:** acute fatty liver of pregnancy  
**APS:** antiphospholipid antibody syndrome  
**CBC:** complete blood count  
**DIC:** disseminated intravascular coagulation  
**FHR:** fetal heart rate  
**HELLP:** hemolysis, elevated liver enzyme, low platelet syndrome  
**TTP-HUS:** thrombotic thrombocytopenic purpura - hemolytic uremic syndrome  
**PET:** preeclampsia-eclampsia; **SpO₂:** oxygen saturation  
**UTI:** urinary tract infection  
**vWF:** von Willebrand Factor.

*Tests of coagulation are recommended if there is thrombocytopenia or placental abruption.

† 'PET imitators' include AFLP, catastrophic APS, TTP-HUS, malignant hypertension and scleroderma renal crisis.

‡ Abnormal uterine artery Doppler velocimetry is practically defined at 22-24 weeks as bilateral notching with mean resistance index (RI) > 0.55 (i.e., > 50th centile), unilateral notching with mean RI > 0.65 (> 90th centile), or no notching with mean RI > 0.70 (> 95th centile).
## Table of Contents

Chapter 17 Preterm Labour and Preterm Birth ................................................................. 412  

Introduction ......................................................................................................................... 412  

Definition .............................................................................................................................. 412  

Incidence ............................................................................................................................... 412  

Etiology and Risk Factors ...................................................................................................... 412  

Predictors of Preterm Birth .................................................................................................. 413  

Fetal fibronectin .................................................................................................................... 413  

Cervical length measured by transvaginal ultrasonography ....................................................... 413  

Morbidity and Mortality ......................................................................................................... 414  

Diagnosis .............................................................................................................................. 414  

Risk Reduction and Prevention ............................................................................................ 415  

Risk Reduction ..................................................................................................................... 415  

Prevention ............................................................................................................................ 415  

Contraindications to Tocolysis ............................................................................................. 420  

Antenatal Corticosteroid Therapy .......................................................................................... 421  

Magnesium Sulphate Therapy for Fetal Neuroprotection in Imminent Delivery at \( \leq 31^6 \) weeks' gestation .......................................................................................................... 424  

Antibiotic Prophylaxis ........................................................................................................... 426  

Maternal Transport ................................................................................................................. 426  

Summary ............................................................................................................................... 427
Chapter 17
Preterm Labour and Preterm Birth

Introduction

Definition

Preterm labour is defined as regular uterine contractions with progressive cervical dilation and/or effacement at > 20 weeks and < 37\(^0\) weeks' gestation. Preterm birth is delivery before 37\(^0\) weeks' gestation. Long-term adverse sequelae of preterm birth occur mainly in infants born at < 34 weeks' gestational age.

Incidence

The incidence of preterm birth in Canada has increased from 6.3% (1981 to 1983) to 7.7% (2009). Only 1% to 2% of pregnancies deliver before 34 weeks, and neonates born at > 34 weeks' gestational age in Level III centres have survival rates of 99% to 100%, although they may require longer hospital stays because of feeding and other difficulties.

The importance of accurate dating in the management of preterm labour cannot be overstated. A difference of 10 days can change the chance of survival from 10% (at 22 weeks) to over 40% (at 24 weeks). Accurate dates must therefore be established and the estimated date of delivery (EDD) must be communicated effectively to the patient. The most accurate time for dating is within the first trimester, between 7 and 14 weeks' gestation. If possible, all women should be offered an ultrasound during this time to confirm dating. Accurate dating will reduce the number of pregnancies prolonged past 41\(^0\) weeks. By 20 weeks' gestation, care providers should be able to inform all pregnant women of their EDD from accurate menstrual data and/or dating from an ultrasound.

Etiology and Risk Factors

The causes of preterm birth can be divided into two main categories: (1) Induced (iatrogenic) preterm birth (accounting for 20% to 30% of preterm births); (2) spontaneous preterm labour (with intact membranes, accounting for 40% to 50% of preterm births, or with preterm pre-labour rupture of membranes, accounting for 30% to 40% of preterm births).

Clinical maternal or fetal conditions associated with induced preterm birth include:

- Preeclampsia
- Complicated insulin-dependent diabetes mellitus
- Abnormal fetal surveillance results
• Intrauterine growth restriction
• Placental abruption
• Intrauterine death
• Chorioamnionitis
• Monochorionic, monoamniotic twins

Risk factors for spontaneous preterm labour and birth include\textsuperscript{1, 14, 15}

• Reproductive history: previous spontaneous preterm birth; advanced reproductive technologies\textsuperscript{16}
• Antepartum bleeding
• PPROM
• Cervical/uterine factors: cervical insufficiency, uterine malformation,\textsuperscript{14} and fibroids\textsuperscript{17}; excisional cervical treatment for cervical intraepithelial neoplasia\textsuperscript{18, 20}
• Fetal/intrauterine factors: multifetal gestation; fetal anomaly; polyhydramnios
• Infection: chorioamnionitis; bacteriuria; periodontal disease\textsuperscript{21, 23}; current bacterial vaginosis with a prior preterm birth\textsuperscript{24}; malaria (particularly in developing countries)
• Demographic factors: low socioeconomic status; single marital status; low level of education; maternal age < 18 or > 35 years
• Other maternal factors: problematic substance use, smoking, physical abuse,\textsuperscript{25} inadequate prenatal care, low pre-pregnancy weight (BMI < 18.5 kg/m\textsuperscript{2}), poor weight gain in pregnancy,\textsuperscript{26} stress,\textsuperscript{27} obesity\textsuperscript{28}

Predictors of Preterm Birth

Fetal fibronectin

Fetal fibronectin (fFN) is a glycoprotein whose presence in cervicovaginal secretions before 34 weeks’ gestation is associated with preterm labour and birth.\textsuperscript{29, 31} The main benefit of fFN assessment is its negative predictive value. A negative fetal fibronectin test indicates a low probability of delivery within 7 days to 14 days, even in the presence of contractions. The chance of delivering within 14 days of a negative fFN test (in women with symptoms) is 1% to 5%. The chance of delivering within 14 days with a positive test in women with symptoms is 17% to 41% (the positive predictive value).

If fFN assessment is used, it is advisable to obtain the swab before a cervical digital examination is performed. If a digital examination is performed first or if the patient has had sexual intercourse in the 24 hours, a false-positive fFN test may result. As lubricants, creams, and disinfectants may also produce a false-positive result, saline, rather than lubricant, should be used in performing the pelvic examination to obtain the fFN swab.

Cervical length measured by transvaginal ultrasonography

The mean cervical length at 24 weeks’ to 28 weeks’ gestation is 34 mm to 35 mm.\textsuperscript{32, 34} The probability of preterm birth increases in singleton pregnancies when the cervical length is less than 25 mm to 30 mm (20 mm in twins), especially
in women at increased risk of preterm birth (e.g., those with a history of previous preterm birth) or those with threatened preterm labour. 33-40

Funnelling of the internal cervical os may be seen on ultrasound imaging but is significant only when the residual cervical length is short. Transvaginal ultrasound imaging is preferred because transabdominal scanning requires some urine in the maternal bladder, which may affect the cervical length. 33, 34, 40, 41

One prospective study of 964 women indicated that routine prenatal cervical length screening of the general obstetrical population by transvaginal ultrasound has poor positive predictive value. 42 However, cervical length measurement can be used for women identified to be at increased risk of preterm birth, 40 as cervical shortening is associated with an increased preterm birth risk. Transvaginal ultrasound measurement of the cervix has a high negative predictive value if length is greater than 30 mm after 24 weeks. This information may be used to prevent unnecessary interventions.

The value of fFN and ultrasound assessment of cervical length is that these tests may make accurate diagnosis of preterm labour more likely, and they may reduce unnecessary transfer, hospitalization, and intervention, thereby improving allocation of resources. 43 Fetal fibronectin testing and ultrasound assessment of cervical length are currently being used in some Canadian centres to assist in the identification and management of women at risk of preterm birth, including those with suspected preterm labour. The effectiveness of these tests needs to be evaluated in large randomized controlled trials before they can be recommended as universal screening tests.

### Morbidity and Mortality

Preterm birth is a major cause of perinatal morbidity and mortality and is estimated to account for 75% of neonatal mortality, excluding lethal malformations. 2-4, 44, 45 The short-term complications of preterm birth include respiratory distress syndrome (RDS), intraventricular hemorrhage (IVH), and necrotizing enterocolitis (NEC).

The long-term sequelae of preterm birth (especially < 28 weeks gestation) include CNS complications (e.g., cerebral palsy), neurodevelopmental delay, 46 respiratory complications (e.g., bronchopulmonary dysplasia), blindness, and deafness.

### Diagnosis

Early in their antepartum care, women should be instructed to be vigilant for signs and symptoms of impending preterm labour—including regular contractions, vaginal fluid loss, vaginal bleeding, change in pelvic pressure, low dull backache, and vaginal discharge—and to report to the hospital or contact their care provider immediately should any of these occur. A timely physical assessment is required to confirm preterm labour; a phone consultation is insufficient.

The presence of uterine contractions and a suggestion of early dilatation or effacement on cervical examination should raise concerns about possible preterm birth. This approach facilitates early institution of therapy but results in an over-diagnosis of preterm labour. In two studies, approximately 50% of women with these signs remained undelivered 48 hours later, despite having no treatment. 47, 48
Risk Reduction and Prevention

Risk Reduction

Screening for risk factors

Screening for and treating bacterial vaginosis in a woman who has had a prior preterm birth has been shown to reduce the risk of low birth weight (odds ratio [OR] 0.31; 95% confidence interval [CI] 0.13 to 0.75) and PPROM (OR 0.14; 95% CI 0.05 to 0.38).  

Screening for and treating asymptomatic bacteriuria has been shown to reduce low birth weight (OR 0.60; 95% CI 0.45 to 0.80).

Encouraging smoking cessation

Cigarette smoking is associated with preterm birth, with a dose–response relationship noted. Smoking cessation should always be encouraged for its general health benefits. It is likely that decreasing or stopping cigarette consumption in pregnancy will reduce the overall incidence of preterm birth but this has not been definitively shown. Smoking cessation counselling is encouraged and is shown to be effective in supporting women to quit smoking during pregnancy. Strategies include simple advice, cognitive behavioural therapy, and support services.

Addressing problematic substance use

Maternal problematic substance use increases the risk of preterm birth. Health care providers should try to identify maternal substance use, provide information on the maternal and fetal risks, and support an approach to care for harm reduction using brief intervention and referral to appropriate community services for counselling and treatment.

Addressing barriers to prenatal care

The absence of prenatal care has been found to be associated with preterm birth but it unclear whether this association is causal or is a marker for other factors that contribute to preterm birth. Retrospective studies cannot adequately control for these confounding factors and prospective randomized trials (no prenatal care versus standard care) would not be ethical.

Prevention

Progestogens

Progestogen treatment has been shown to decrease the risk of preterm birth under 34 weeks. Various routes of administration have been studied, including intramuscular (IM), vaginal suppository, and oral. 17-hydroxyprogesterone caproate has been shown to decrease the rate of preterm birth < 32 to 34 weeks and birth weight < 2500 grams in women at high risk who have a history of spontaneous preterm birth. In a randomized controlled trial comparing intramuscular progesterone with vaginal progesterone, in women with a prior preterm birth,
Intravaginal progesterone was associated with greater compliance, fewer reported side-effects, and greater proportion of deliveries at >34 weeks of gestation than intramuscular progesterone. The OPPTIMUM study in women at high risk showed that vaginal progesterone was not associated with reduced risk of preterm birth or composite neonatal outcomes, and had no effect (beneficial or harmful) on outcomes in children at 2 years of age.

A randomized controlled trial, PREDICT, and a secondary analysis, both published in 2011, found that progesterone treatment does not appear to prevent preterm birth in twin pregnancies or in twin pregnancies at high risk. In 2017, a Cochrane review including 17 RCT studies found there was no reduction in risk of improved neonatal outcome in twins if progesterone was administered.

In 2015, a meta-analysis by Combs et al. found that in women with triplet pregnancies there was no significant difference on perinatal outcome or pregnancy duration between those who received 17-hydroxyprogesterone caproate and those who received placebo. The STOPPIT trial found no evidence that in utero exposure to progesterone had a detrimental or beneficial impact on health and developmental outcomes at 3 to 6 years of age.

Other important maternal and infant outcomes have not been well-studied to date. It is unclear if the prolongation of gestation results in improved maternal and long-term infant health outcomes. Information regarding the potential adverse effects of progesterone therapy to prevent preterm birth is limited.

A 2008 SOGC technical update concluded that women at risk for preterm labour should be encouraged to participate in studies on the role of progestogen treatment in reducing the risks of preterm labour. Women should be informed about the lack of available data from any neonatal outcome variables and about the lack of comparative data on dosing and route of administration. Women with a short cervix should be informed of the single large RCT showing the benefit of progesterone in preventing preterm labour. Women and their care providers should be aware that a previous preterm labour and/or short cervix (< 15 mm at 22 to 26 weeks’ gestation) on transvaginal ultrasound could be used as an indication for progestogen therapy initiated after 20 weeks’ gestation and continuing until the risk of prematurity is low.

A 2012 review by the Society for Maternal-Fetal Medicine recommends the following:

- Singleton pregnancies with prior history of preterm birth: 17-hydroxyprogesterone caproate 250 mg IM weekly from 16–20 weeks until 36 weeks.
- Singleton pregnancies without prior preterm delivery with short cervix ≤ 20 mm at ≤ 24 weeks: vaginal progesterone 90 mg gel or 200 mg suppository daily from diagnosis of short cervix until 36 weeks.

There may be short-term benefits, but there is no evidence of benefit or harm in childhood outcomes. Further studies are needed to determine the efficacy of these therapies, and optimal combination of dosing and routes of administration of the various progestogens. Since 17-hydroxyprogesterone is not readily available in Canada, use of progesterone 200 mg vaginally daily is a reasonable option.
Recommended administration:

1. Women with previous preterm birth < 34° weeks, without a shortened cervix: vaginal micronized progesterone 200 mg daily, from 16 weeks until 36 weeks. (Some experts recommend treatment for previous preterm birth <37° weeks though the evidence for benefit between 34° and 37° is not as strong)69
2. Women with a short cervix ≤ 20 mm ≤ 24 weeks: vaginal micronized progestogen 200 mg daily, from diagnosis until 36 weeks.

Cervical cerclage
A 2013 SOGC technical update recommends that cerclage should be considered in women with a cervix ≤ 25 mm before 24 weeks' gestation only if they have a prior history of preterm birth or a prior history of suspected cervical incompetence. There is no clear benefit to cerclage for an incidental finding of a short cervix, but further surveillance by ultrasound is recommended in this situation. Additionally, the data do not support the use of progestogen together with cerclage.70

Cervical pessary
The incidence of preterm birth in both singleton and twin pregnancies has been reduced by use of a cervical pessary for women who have a short cervix < 25 mm as measured by transvaginal ultrasound.21,22 However, larger multicentre randomized controlled trials in both singleton and twin pregnancies have not substantiated these early findings.23,24

Management of Preterm Labour
Successful management of preterm labour requires early diagnosis, identification of the cause, and treatment of the underlying cause when possible. Labour should be arrested when appropriate, and interventions made to minimize neonatal morbidity and mortality.

Assessment
- Establish dates: history (EDD, menstrual history, ultrasounds); review the prenatal record for EDD, menstrual history, pertinent ultrasounds, and clinical growth
- Measure vital signs (temperature, blood pressure, respirations, pulse) and assess fetal well-being
- Evaluate contractions: history (frequency, intensity, duration, changes with time); abdominal examination for uterine activity; tocodynamometer (frequency of contractions)
- Perform cervical assessment: undertake speculum examination initially to rule out PPROM, obtain swab for fFN testing, if available, and cultures as required (e.g., gonorrhea culture/Chlamydia culture, GBS swab); defer digital examination until after confirmation that membranes are intact and there is no placenta previa
- Undertake laboratory evaluation (complete blood count for leukocytosis)

Fetal monitoring is recommended for all preterm labours. Caution should be exercised with fetal scalp electrode if the fetus is < 34 weeks gestational age and with fetal blood sampling if the fetus is between 34 and 36 weeks gestation (this is probably best avoided in gestations < 34 weeks).
Tocolytics

Some tocolytics have been shown to prolong pregnancy for 48 hours or more when given before 34 weeks’ gestation. This provides a window of opportunity for the administration of glucocorticoids. It also allows for the transportation of the mother to a tertiary centre if necessary. Tocolysis may be contraindicated in a significant percentage of patients in preterm labour. There is insufficient evidence to support tocolysis for women with PPROM.\textsuperscript{75}

There is evidence that the following have some efficacy

<table>
<thead>
<tr>
<th>AGENT/TREATMENT</th>
<th>EVIDENCE</th>
<th>FINDINGS</th>
<th>DOSE</th>
<th>SIDE EFFECTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium channel blockers (nifedipine)\textsuperscript{76}</td>
<td>• No placebo-controlled trials; 2003 Cochrane review\textsuperscript{77}. 12 trials, n = 1029, comparing nifedipine with another tocolytic (mainly betamimetics)</td>
<td>• Lower rate of delivery within 7 days and &lt; 34 weeks, reduced rates of RDS, necrotizing enterocolitis, IVH and jaundice with nifedipine. Findings driven mainly by Papatsonis et al. study\textsuperscript{78} that found significant differences  • Fewer side effects and hence less need to discontinue treatment.</td>
<td>• Ideal dosage regimen not yet determined, but many centres in Canada are using this as their first line tocolytic.  • Peak onset of oral nifedipine is 30 to 60 minutes. Half-life is 90 minutes. Dosing of nifedipine 10mg PO should occur at a minimum of 1 hour intervals.  • Maintenance therapy is nifedipine regular capsules 10 mg orally every 4 to 6 hours (maintenance therapy should start 6 hours following completion of loading dose). Maintenance can be increased up to 20 mg orally every 4 to 6 hours at physician’s discretion.  • Maximum daily dose is 120 mg. It is stopped 48 hours after first dose of betamethasone.\textsuperscript{a}  • Close monitoring of maternal blood pressure\textsuperscript{80}.</td>
<td>• Generally well-tolerated but may cause maternal dizziness, light headedness, headache, flushing, nausea, and transient hypotension with resulting FHR changes.  • A 2010 systematic review and meta-analysis of 5607 women by Khan suggests caution for total doses of &gt; 60 mg. A total nifedipine dose of &gt; 60 mg is associated with a significant increased risk of adverse events, particularly those associated with significant morbidity such as tachycardia and hypotension.\textsuperscript{81}</td>
</tr>
</tbody>
</table>
### AGENT/TREATMENT

<table>
<thead>
<tr>
<th>AGENT/TREATMENT</th>
<th>EVIDENCE</th>
<th>FINDINGS</th>
<th>DOSE</th>
</tr>
</thead>
</table>
| PG synthetase inhibitors (indomethacin) | 2005 Cochrane review<sup>83</sup> | • 3 small trials (n = 106) compared with placebo: more effective than placebo in delaying delivery to ≥ 37 weeks (this finding based on only 1 small study)  
• 5 trials compared with other tocolytics: PG synthetase inhibitors more effective in delaying delivery to ≥ 37 weeks and decrease in maternal drug reaction requiring cessation of treatment  
• consult local tertiary centre as to local standard of care | • 100 mg suppository for transport and repeat 25 mg to 50 mg every 6 hours for a maximum of 48 hours |
| Nitroglycerin                   |                                       | There is limited evidence of the benefit of nitroglycerin for tocolysis.                                                                      |                                                                    |

• Potential fetal complications of PG synthetase inhibitors: should not be used after 32 weeks’ gestation because of increased sensitivity of the ductus arteriosus to closure reduced fetal urine production causing oligohydramnios; neonatal renal insufficiency has been reported. Therefore, PG synthetase inhibitors should not be used for more than 48 hours without assessment of amniotic fluid volume.  
• A systematic review did not identify statistically significant increased risks of adverse outcomes with indomethacin use. However, the limited power of the review did not allow exclusion of the possibility that indomethacin is associated with adverse neonatal outcomes<sup>84</sup>

---

<sup>83</sup> Modified from Dr Joan Crane, Women’s Health Centre, Eastern Health, St. John’s: personal communication, February 14, 2012.
There is no evidence for the efficacy of the following

<table>
<thead>
<tr>
<th>AGENT/TREATMENT</th>
<th>EVIDENCE</th>
<th>CONCLUSION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Magnesium sulphate</td>
<td>2014 Cochrane review (37 trials and 3571 women)</td>
<td>No benefit at any dose as a tocolytic</td>
</tr>
<tr>
<td>Progestational agents</td>
<td>2010 Cochrane review (4 trials, 192 women)</td>
<td>No benefit as a tocolytic</td>
</tr>
<tr>
<td>Bed rest</td>
<td>2010 Cochrane review (multiple pregnancy)</td>
<td>No reduction of the risk of preterm birth or perinatal mortality</td>
</tr>
<tr>
<td>Fluid bolus</td>
<td>Two small studies (228 women) comparing intravenous hydration with bed rest alone</td>
<td>No evidence of benefit</td>
</tr>
<tr>
<td>Sedation, narcotics</td>
<td>UpToDate review</td>
<td>No evidence of benefit</td>
</tr>
</tbody>
</table>
| Home uterine activity monitoring | Randomized trials                                                     | Home uterine activity monitoring has not been shown to reduce preterm birth rates.  
|                             |                                                                         | It increases visits to labour and delivery units, obstetric intervention, and cost of antepartum care. |

**Contraindications to Tocolysis**

Any contraindication to continuing the pregnancy

- preeclampsia or other medical indication for delivery
- chorioamnionitis
- mature fetus
- imminent delivery
- intrauterine fetal death or lethal fetal abnormality
- abnormal fetal surveillance
- significant antepartum hemorrhage

Contraindications to specific tocolytic agents
The following tocolytics are not available in Canada: terbutaline, ritodrine, atosiban.

**Antenatal Corticosteroid Therapy**

RDS is a major concern with preterm delivery. IVH, necrotizing enterocolitis, persistent pulmonary hypertension, and other respiratory conditions are also associated with preterm birth and are more likely to occur in newborns with RDS. In the past, RDS accounted for > 20% of all neonatal deaths. The increased use of antenatal steroids and innovations in neonatal care have reduced its occurrence and consequences.

The benefits of antenatal corticosteroid therapy are now definitively established. Betamethasone and dexamethasone cross the placenta and induce enzymes that accelerate fetal pulmonary maturity. It takes 48 hours after the first dose for the full benefit to be achieved. An incomplete course of corticosteroid therapy may still offer benefits. Figure 1 shows the results of a meta-analysis of corticosteroid administration in women at risk of preterm birth. Treatment with antenatal corticosteroids is associated with an overall reduction in neonatal death, RDS, intraventricular hemorrhage, necrotizing enterocolitis, respiratory support, neonatal intensive care unit admissions, and systemic infections in the first 48 hours of life.

Debate exists as to the preferred corticosteroid. Two Cochrane reviews, Roberts et al. in 2017 and Brownfoot et al. in 2013 reviewed the effects of these corticosteroids on fetal lung maturity and perinatal outcomes. Indirect comparisons in Roberts et al. showed betamethasone has significant reductions in chorioamnionitis, RDS, and chronic lung disease compared to dexamethasone. Direct comparisons in Brownfoot et al. demonstrated greater reduction in IVH and lower length of NICU stay for dexamethasone compared to betamethasone. Both showed similar effects in other perinatal outcomes. Further studies are may clarify any difference. Currently, betamethasone and dexamethasone are acceptable choices.

**Repeat corticosteroids**

Scheduled repeat courses of corticosteroids are not indicated. It is becoming more evident that there may be some reduction in the occurrence and severity of neonatal lung disease and serious infant morbidity, but these benefits are associated with smaller head circumference and low birthweight, and with unknown long-term sequelae. A 5-year follow-up study from the preterm birth trial demonstrated that children born at term who were exposed to multiple courses of antenatal corticosteroid were higher risk for neurosensory disability (adjusted OR 3.70; 95% CI 1.57-8.87) and severe neurologic disability. The 2016 Australasian Collaborative Trial of Repeat Doses of Prenatal Steroids (ACTORDS) had 6-8 years follow up and found higher frequency of attention deficit and behavioural dysfunction with multiple-courses of steroid group.

In light of this evidence, balancing the neonatal benefits and limited evidence of long term effects, planned multiple courses are not recommended.

**Rescue corticosteroids**

Rescue corticosteroids refers to the administration of one additional course or dose of antenatal corticosteroids after already receiving a full course of antenatal corticosteroids during a pregnancy.
A randomized trial of a rescue course of antenatal corticosteroids (women less than 33 weeks of gestation with a recurring threat of preterm delivery, who had received steroids at least 14 days before) found an improvement in neonatal outcomes (reduction in composite neonatal morbidity, RDS, ventilatory support, and surfactant use) and no increase in adverse outcomes. However, there was no significant short term benefits demonstrated with a delay greater than 7 days between rescue dose and delivery.105

The data on rescue corticosteroids is limited and questions remain regarding the long-term effects of multiple courses of corticosteroids. Rescue corticosteroids can be considered at least 14 days after administration of the first full course, taking into account the gestational age of the first administration, and after a discussion of the risks and benefits.

**Late preterm corticosteroids**

The 2016 ALPS study by Gyamfi-Bannerman et al.106 showed that the administration of betamethasone in women at risk for late preterm delivery (34⁰ weeks to 36⁶ weeks) significantly reduced the rate of neonatal respiratory complications. Neonatal hypoglycemia was more frequent after corticosteroid administration. No long-term data are yet available in the cohort. The ASTECS trial indicates a need for caution in relation to long-term outcomes of exposure to antenatal corticosteroids late in pregnancy: in this trial, school performance was significantly poorer in 7-year-olds.107 108

**Figure 1. Antenatal corticosteroids versus no treatment**

<table>
<thead>
<tr>
<th>OUTCOME</th>
<th>RR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RDS</td>
<td>0.66 (0.59, 0.73)</td>
</tr>
<tr>
<td>IVH Dx by US</td>
<td>0.54 (0.43, 0.69)</td>
</tr>
<tr>
<td>NEC</td>
<td>0.46 (0.29, 0.74)</td>
</tr>
<tr>
<td>Neonatal Infection</td>
<td>0.56 (0.38, 0.85)</td>
</tr>
<tr>
<td>Neonatal Death (all)</td>
<td>0.69 (0.58, 0.81)</td>
</tr>
</tbody>
</table>

Adapted from Roberts D, Cochrane Library 2006, Issue 3
Recommendations for antenatal corticosteroids

Women who are at increased risk of preterm delivery (especially within the next 7 days) are candidates for antenatal steroid therapy.

- Lower gestation limit 24 weeks (< 24 weeks should be assessed on a case-by-case basis)
- Upper gestation limit 34 weeks 6 days
- Prophylactic administration depends on diagnosis and risk
  - not recommended for pre-labour CS at term (37-39 weeks gestation)

Corticosteroid options

- Betamethasone 12 mg IM every 24 hours x 2 doses
- Dexamethasone 6 mg IM every 12 hours x 4 doses

Special considerations with use of corticosteroids

- Contraindications to the use of corticosteroids: active tuberculosis, gastric ulcers, and chorioamnionitis
- If immediate delivery is indicated, it should not be delayed to wait for corticosteroid treatment effect
- Use of corticosteroids will transiently increase maternal blood sugar; it is recommended to delay testing for gestational diabetes for at least 1 week after administering corticosteroids to avoid elevated glucose results
- Use of corticosteroids will also transiently increase the white blood cell count
- Evidence on the long-term academic performance of school-age children calls into question the use of routine steroids for elective Caesarean section from 37 to 39 weeks. Due to the benefits and potential harms, corticosteroid use is not recommended for pre-labour Caesarean section at term.
- Use of corticosteroids may decrease fetal movements in the first 3 days following initiation of therapy
- Corticosteroids should be administered in the same gestational age range and dosage in women with obesity, with twins or higher order multiples, or with a suspected growth restricted fetus.
Magnesium Sulphate Therapy for Fetal Neuroprotection in Imminent Delivery at ≤ 33\textsuperscript{6} weeks’ gestation\textsuperscript{112}

1. For women with imminent preterm birth (≤ 33\textsuperscript{6} weeks), antenatal magnesium sulphate administration should be considered for fetal neuroprotection (Figure 2).
2. Although there is controversy about upper gestational age, antenatal magnesium sulphate for fetal neuroprotection should be considered from viability to ≤ 33\textsuperscript{6} weeks.
3. If antenatal magnesium sulphate has been started for fetal neuroprotection, tocolysis should be discontinued.
4. Magnesium sulphate should be discontinued if delivery is no longer imminent or a maximum of 24 hours after therapy has been administered.
5. For women with imminent preterm birth, antenatal magnesium sulphate for fetal neuroprotection should be administered as a 4g IV loading dose over 30 minutes, followed by a 1g/hr maintenance infusion until birth.
6. For planned preterm birth for fetal or maternal indications, magnesium sulphate should be started, ideally within 4 hours before birth, as a 4g IV loading dose over 30 minutes, followed by a 1g/hr maintenance infusion until birth.
7. There is insufficient evidence that a repeat course of antenatal magnesium sulphate for fetal neuroprotection should be administered.
8. Delivery should not be delayed in order to administer antenatal magnesium sulphate for fetal neuroprotection if there are maternal and/or fetal indications for emergency delivery.
9. When magnesium sulphate is given for fetal neuroprotection, maternity care providers should use existing protocols to monitor women who are receiving magnesium sulphate for preeclampsia and/or eclampsia.
10. Indications for fetal heart rate monitoring in women receiving antenatal magnesium sulphate for neuroprotection should follow the fetal surveillance recommendations of the SOGC 2007 Fetal Health Surveillance: Antepartum and Intrapartum Consensus Guideline.
11. Since magnesium sulphate has the potential to alter the neonate’s neurological evaluation, causing hypotonia or apnea, health care providers caring for the neonate should have an increased awareness of this effect.
Figure 2. Magnesium sulphate for fetal neuroprotection in imminent preterm birth (≤ 33\(^6\) weeks)

Obstetric care providers should consult their regional referral centre for guidance before starting magnesium sulphate. When inappropriately administered, magnesium sulphate can be cardiotoxic and cause respiratory depression. Magnesium sulphate must always be administered via a medication pump to prevent overdose. \cite{113} Clinical hyporeflexia is the first sign of magnesium toxicity and should prompt discontinuation of magnesium drip and consideration of administration of calcium gluconate and assessment of magnesium levels.
Antibiotic Prophylaxis

Although evidence does not support the routine administration of antibiotics in preterm labour with intact membranes, the antibiotic protocol for group B streptococcus prophylaxis should be followed if delivery is anticipated or imminent.

Maternal Transport

The decision to transport should be made in consultation with the receiving physician.

Considerations

- Availability of neonatal and obstetrical care
- How quickly transport and skilled personnel can be available
- Travel time and travel conditions, e.g., weather
- Stability of the mother and fetus
- Risk of delivery en route
  - parity, length of previous labour
  - contractions: response to tocolytics
  - presentation of fetus
  - cervical status

Contraindications

- Unstable maternal condition
- Abnormal fetal surveillance
- Imminent delivery
- No experienced attendants to accompany mother
- Adverse weather or other hazardous conditions for travel

Transport plan

Every institution should have a transport protocol that includes:

- Availability of antenatal forms, ultrasound reports, and laboratory results
- Communication with patient and family and with receiving health care team (physician and nurses) re: indication, stabilization, mode of transport, estimated time of arrival
- Appropriate attendant for transport
- IV access, indicated medication, appropriate equipment
- Assessment of patient immediately before transport
Location of preterm birth

It has been clearly shown that preterm infants born in tertiary care (level III) centres experience less mortality and long-term morbidity than those born in level II or level 1 centres. Those born during transport fare worst of all.

Summary

- Diagnose promptly and accurately
- Identify and treat underlying cause, if possible; attempt to prolong pregnancy, if indicated
- Consider magnesium sulphate therapy at ≤ 33.6 weeks for neuroprotection
- Intervene to minimize neonatal morbidity and mortality
- **STAT**: a mnemonic
  - **S**teroids: Antenatal corticosteroid therapy
  - **T**ocolytics: If indicated
  - **A**ntibiotics: GBS prophylaxis
  - **T**ransport
References


86. Su LSM, Chong Y. Progestational agents for treating threatened or established preterm labour [Cochrane review]. 1 CDoSRI, editor. Chichester (UK) John Wiley & Sons, Ltd; 2010.


89. Simhan H CS. Inhibition of acute preterm labor. Waltham (MA): UpToDate, Inc.; 2007.


# Table of Contents

Chapter 18 Prelabour Rupture of Membranes (PROM) ................................................................. 438  
   Background ................................................................................................................................. 438  
      Definitions ................................................................................................................................. 438  
      Incidence ................................................................................................................................. 438  
   Etiology and Risk Factors ........................................................................................................... 438  
      Risk Factors: ........................................................................................................................... 439  
   Morbidity and Mortality ............................................................................................................. 439  
   Diagnosis .................................................................................................................................. 440  
      History ................................................................................................................................... 440  
      Speculum Examination .......................................................................................................... 440  
      Complications of Term PROM ............................................................................................ 441  
      Complications of Preterm PROM ....................................................................................... 441  
   Management .............................................................................................................................. 441  
      Management of Term PROM (≥ 37 weeks’ gestation) ............................................................. 442  
      Expectant Management ........................................................................................................... 443  
      Management of PPROM (34° to 36° weeks’ gestation) ........................................................ 443  
      Management of PPROM (< 34 weeks’ gestation) ................................................................... 444  
   Summary .................................................................................................................................... 445  

Prelabour Rupture of Membranes (PROM) ....................................................................................... 437
Chapter 18

Prelabour Rupture of Membranes (PROM)

Background

Definitions

Prelabour rupture of membranes (PROM) is rupture of the membranes before the onset of labour when there is at least an hour between membrane rupture and the onset of contractions.¹ PROM may occur at ≥ 37 weeks’ gestation (term PROM) or at < 37 weeks’ gestation (preterm PROM or PPROM). It is further classified by gestational age: mid-trimester PPROM (before 24 weeks’ gestation), early PPROM (24 to 34 weeks’ gestation), and near-term PPROM (34 to 37 weeks’ gestation).

The latency period is the interval between the rupture of the membranes and the onset of labour. Later gestational age, oligohydramnios and multiple gestation are associated with a shortened latency period.²

Almost 90% of women at term will go into spontaneous labour within 24 hours of membrane rupture.³ In women with preterm rupture of membranes, the latency period tends to be longer. Only 50% of these women establish labour within 24 hours, with 70% to 80% delivering within 1 week of membrane rupture.⁴

Incidence

Term PROM occurs in approximately 8% of pregnancies.³ ⁵

Preterm PROM occurs in 2% to 3.5% of pregnancies but accounts for one third of the cases of preterm delivery.⁶ The frequency of PROM increases with increasing plurality of gestation (13.3% for singleton, 16.81% for twins, and 20% for triplets).⁷

Etiology and Risk Factors

Term PROM results from the normal physiological process of progressive membrane weakening combined with the shearing forces created by uterine contractions. PPROM can result from a whole array of pathological mechanisms that act individually or in concert, with evidence pointing to biochemical processes such as disorders in the extracellular matrix of amnion and chorion, such as those caused by an intrauterine infection.⁸
Risk Factors:

- Amniocentesis
- Cervical insufficiency
- Cervical cerclage
- Prior cervical conization, laser conization, loop electrosurgical excision procedure
- PPROM in a previous pregnancy
- Prior preterm labour/delivery
- Chronic placental abruption
- Vaginal bleeding in pregnancy
- Polyhydramnios
- Multiple pregnancy
- Short interpregnancy interval of less than 6 months
- Cigarette smoking
- Sexually transmitted infection
- Low socioeconomic status
- Bacterial vaginosis (BV)
- Periodontal disease

Bacterial vaginosis is a polymicrobial syndrome resulting in a decreased concentration of lactobacilli and an increase in pathogenic bacteria. It may or may not be symptomatic. Current evidence does not support screening all pregnant women for bacterial vaginosis and then treating those with asymptomatic BV to prevent preterm birth and its consequences. For women with a previous preterm birth, there is little suggestion that screening and treatment of BV will prevent a further preterm birth. However, it reduces the risk of low birth weight (odds ratio [OR] 0.31; 95% confidence interval [CI] 0.13 to 0.75) and PPROM (OR 0.14; 95% CI 0.05 to 0.38). In the symptomatic woman at low risk for adverse obstetrical outcome, oral and vaginal antibiotics are equally effective for symptom relief. However, women who have a past history of preterm birth and who are symptomatic for BV should be treated with oral metronidazole 500 mg or clindamycin 300 mg twice daily for 7 days. Topical (vaginal) therapy is not recommended for this indication because although cure rates are similar to those observed with oral treatment, they have not been shown to be effective-for-preterm birth prevention.

Morbidity and Mortality

The risk to both mother and infant is increased after the occurrence of PROM, whether at or before term. Among women with PPROM, clinically evident intra-amniotic infection occurs in 15% to 25% of these women, with histological chorioamnionitis in 51%. Fetal risks include umbilical cord compression and ascending infection.
Diagnosis

Diagnosis of PROM and PPROM is made by a combination of patient history, clinical suspicion, physical examination, and testing. As patient history has an accuracy of 90% for the diagnosis of PPROM, it should not be ignored.16

Digital pelvic examination is not recommended because of the increased risk of ascending infection17, 18 and shortening of the latent period.19 However, sterile speculum examination is appropriate for confirmation of PROM, assessment of cervical status, and exclusion of cord prolapse. Although ultrasound is not diagnostic, the confirmation of the presence of a normal amount of amniotic fluid makes the diagnosis of PROM less likely.

History

A careful history should be taken to determine the presence of fluid leaking, including the amount, timing, odour, persistence, and colour. The vast majority of women with a history of vaginal fluid leakage will have PROM.20

Speculum Examination

A speculum examination should be undertaken to look for

- Fluid pooling in the posterior fornix
- Free flow of fluid from the cervix
- Cord prolapse
- Ferning: assessment for ferning is performed by obtaining a sample of fluid from the posterior fornix, placing it on a glass slide, and letting it air dry for 10 minutes. When visualized under a microscope, the presence of characteristic arborization (ferning), caused by the crystallization of sodium chloride, suggests the presence of amniotic fluid (see Appendix)21

  - false positive in a woman in labour: 11.8%; in a woman not in labour: 21.2%22
  - false negative in a woman in labour: 2%; in a woman not in labour: 40.6%22

Antiseptic solution, semen, fingerprints, and cervical mucus may cause false-positives, while blood, meconium, and vaginal secretions will not.23 Therefore the result of ferning should be viewed as supportive rather than conclusive in the non-labouring woman with non-specific vaginal fluid loss.20

- pH nitrazine testing of fluid. This test is non-specific. Nitazene paper changes to a dark blue from yellow with a pH above 6.5. During pregnancy, normal vaginal pH is 4.5 to 6.0. Amniotic fluid pH is 7.1 to 7.3. False positive results can result from blood, alkaline vaginal infections (e.g., bacterial vaginosis), alkaline urine, and semen. False negative results may occur with prolonged membrane rupture and minimal residual fluid.20

Fluid in the vagina may be collected to test for fetal lung maturity indices in preterm cases.

Commercial tests based on biochemical markers for the diagnosis of ruptured membranes such as placental alpha microglobulin-1 (PAMG-1) (Amnisure™) are available and may be useful in certain clinical settings. Although the
sensitivity of such tests ranges from 94.8 to 98.9% and the specificity from 87.5 to 100%,\cite{24} their high cost limits the test to where the diagnosis remains uncertain.\cite{25}

### Complications of Term PROM

- Fetal/neonatal infection (e.g., RDS, IVH, NEC)
- Maternal infection (e.g., endometritis, chorioamnionitis, bacteremia)
- Umbilical cord compression/prolapse\cite{26}

### Complications of Preterm PROM

- Preterm labour and delivery
- Fetal/neonatal infection
- Maternal infection
- Umbilical cord compression/prolapse
- Increased Caesarean section rate
- Placental abruption
- With early, severe oligohydramnios
  - pulmonary hypoplasia (< 26 weeks’ gestation)\cite{2, 23}
  - fetal deformity

The most significant complication of PPROM is preterm birth and its consequences.\cite{2, 23}

### Management

The management of PROM at any gestational age requires

- Confirmation of the diagnosis
- Assessment of maternal and fetal well-being
- Determination of the presence of any associated condition that requires concurrent management or that may indicate immediate delivery is desirable
- Avoidance of digital examination whenever possible.\cite{15, 16} If expectant management is planned, the cervix can be assessed during the speculum examination. If the woman is in labour, digital cervical assessment is indicated

Determination of fetal presentation using ultrasound if abdominal assessment is inconclusive.

There is scientific evidence that maternal markers (C-reactive protein (CRP) and white blood cell (WBC) counts) are predictive of early-onset neonatal infection.\cite{25, 22} The bedside assessment of amniotic fluid interleukin-6 also seems a promising measure of microbial invasion of the amniotic cavity (MIAC).\cite{28-30}
There is no evidence to support tocolysis for women with PPROM as it is associated with an increase in chorioamnionitis, increase in Apgar scores < 7 at 5 minutes, and an increase in the need for neonatal ventilation.\textsuperscript{31, 32}

Outpatient management of selective patients with PPROM may be suitable, after a period of observation and provided specific prerequisites are met. A retrospective cohort trial studying 133 patients with PPROM and a latency of at least one week showed that the safety of outpatient management of appropriately selected patients is comparable with the safety of in-hospital management.\textsuperscript{33} These include assessment of maternal or fetal complications, fetal position, fetal growth and well-being, signs of labour or chorioamnionitis, and timely access to hospital. In another similar trial of 414 women with PPROM between 24 and 34 weeks, there was no increase in adverse maternal and perinatal outcomes for women receiving outpatient care compared with hospital care.\textsuperscript{34} Specific departmental protocols however need to be followed.\textsuperscript{1, 35}

Magnesium sulphate is used for neuroprotection in preterm labour before 3 and 6 days' gestation. In a secondary analysis of a randomized controlled trial of magnesium sulphate for prevention of cerebral palsy, it was found not to prolong latency.

### Management of Term PROM (≥ 37 weeks’ gestation)

Digital cervical examination should not be undertaken until induction is initiated or labour has begun. The parturient women should be assessed for infection and cultures obtained, if indicated. Antibiotics should be administered for group B streptococcus (GBS) prophylaxis, if indicated.

Current evidence supports induction of labour (IOL) within 24 hours rather than expectant management for all women with term PROM.\textsuperscript{38} Induction of labour reduces the risk of maternal infection (e.g., chorioamnionitis and endometritis) and NICU admission without increasing the rates of Caesarean section or assisted vaginal birth. Expectant management on the other hand has been shown to increase the likelihood of Caesarean section and prolong maternal hospitalization.\textsuperscript{39} Although IOL with vaginal prostaglandin (PGE2) has been shown to be as effective as oxytocin for labour induction, it is associated with higher rates of chorioamnionitis. Prostaglandins may, however, be considered in women with unfavourable cervix.\textsuperscript{40} A Cervical Ripening Balloon should not be used in the presence of PROM due to a possible increase in the rate of chorioamnionitis.\textsuperscript{41}

Oral misoprostol (50 mcg po q4hrly to a maximum of 4 doses) is an induction agent for PROM. Unlike oxytocin, it has both uterotonic and cervical ripening effects, which are of particular benefit to women with an unripe cervix. It is easier to administer than IV oxytocin and after an initial period of monitoring, it does not need continuous EFM until contractions begin. Compared with vaginal prostaglandins, oral misoprostol does not require vaginal examinations, lessening concerns about infection. A randomized trial of 758 women showed fewer Caesarean sections for dystocia and no difference in fetal or maternal complications with a regimen of oral misoprostol than with vaginal PGE2 and oxytocin. Women receiving misoprostol had a small increase in gastrointestinal side effects.\textsuperscript{42, 43}

For women colonized with GBS, the indication for induction is more compelling because of the additional benefit of this management in reducing neonatal infection.\textsuperscript{44}

- CS if there are contraindications to IOL and/or vaginal birth.\textsuperscript{5, 45}
• Surveillance for infection if management is expectant. Surveillance includes monitoring the maternal pulse and temperature, fetal heart rate, presence of uterine tenderness or irritability, and changes in white blood cell counts and CRP, if indicated.
• Appropriate antibiotics and IOL if chorioamnionitis develops.
• Induction with oxytocin or prostaglandin reduces the risk of chorioamnionitis (relative risk [RR] 0.74, 95% CI 0.56 to 0.97) and endometritis (RR 0.30, 95% CI 0.12 to 0.74) without increasing CS and operative vaginal deliveries. Although a Cochrane review found there was no difference in neonatal infection (RR 0.83, 95% CI 0.61 to 1.12), fewer infants in the induction groups went to the neonatal intensive care unit compared with expectant management (RR 0.72, 95% CI 0.57 to 0.92, number needed to treat 20). A recent Observational study showed that outpatient labour induction with dinoprostone for TPROM was both feasible and safe.
• Chorioamnionitis is reduced and maternal satisfaction is increased if labour is induced with intravenous oxytocin, compared with inducing labour with prostaglandins (+/- oxytocin) or with expectant management.
• Neonatal infection is reduced among women who are GBS positive if labour is induced with intravenous oxytocin, compared with inducing labour with prostaglandins (+/- oxytocin) or with expectant management.

There is insufficient evidence to justify the routine use of prophylactic antibiotics with term PROM in the absence of an indication for GBS prophylaxis.

Expectant Management

For women who are managed expectantly beyond 24 hours:

• No digital examination should be done in the absence of contractions.
• Women need to be advised to report any sign of infection or decreased fetal movement.
• Fetal movements and fetal heart rate should be evaluated every 24 hours.
• Asymptomatic healthy term babies born after 24 hours of PROM should be observed for the first 12 hours for signs of infection.

Management of PPROM (34⁰ to 36⁶ weeks’ gestation)

Optimal timing of delivery between 34⁰ and 36⁶ weeks in pregnancies complicated by PPROM is unclear. There is evidence supporting expectant management for PPROM at less than 34 weeks, but there is no consensus on the optimal management of pregnancies with PPROM and no spontaneous labour at 34⁰ to 36⁶ weeks. The 2010 Cochrane review of planned early birth versus expectant management with PPROM prior to 37 weeks’ gestation did not add any clarity.

The PPROMEXIL-2 trial and meta-analysis published in 2012 by Van der Ham et al. does not indicate that IOL substantially improves pregnancy outcomes compared with expectant management. The authors concluded that the risk of neonatal sepsis (suspected or confirmed) after PPROM near term is low and that IOL does not reduce this risk. Induction does seem to reduce the risk of chorioamnionitis. There were no differences in respiratory distress syndrome and Caesarean sections between the induction and expectant management group. A large multicentre trial of nearly 2000 women comparing active with expectant management in membrane rupture between 34 and 37 weeks showed
neonatal sepsis occurring in 2% of those induced and in 3% of those treated conservatively. Other neonatal morbidities did not reach statistical significance. As far as maternal outcomes were concerned, expectant management carried some risk of antepartum hemorrhage (RR 0.6, 95% CI 0.4-0.9), intrapartum fever and the use of antibiotics, but was associated with a lower Caesarean section rate. The message from these authors was “in the absence of overt signs of infection or fetal compromise, a policy of expectant management with appropriate surveillance of maternal and fetal wellbeing should be followed in pregnant women who present with ruptured membranes close to term.”

Antibiotics can be used in PPROM > 32 weeks’ gestation if fetal lung maturity cannot be proven and delivery is not planned.

The primary motivation for considering IOL earlier than 37 weeks’ gestation in the presence of PPROM is that the risks associated with preterm birth are outweighed by those associated with maternal, fetal, and neonatal infectious morbidity.

Care providers should:

- Not perform digital cervical examination.
- Assess for infection and obtain cultures.
- Undertake ultrasound assessment of fetal position, cervical status, and fluid volume.
- Administer antibiotics for GBS prophylaxis, if indicated.
- Consider transfer to a higher-level centre, if relevant.
- Consider IOL with oxytocin to reduce the risk of chorioamnionitis (at the expense of an increase in mild neonatal morbidity, respiratory and metabolic).
- Inform women of the benefits and risks of IOL compared with expectant management.
- If management is expectant, assess for infection (monitoring maternal pulse and temperature, fetal heart rate, presence of uterine tenderness or irritability, and WBC changes if indicated).
- If chorioamnionitis is suspected, administer appropriate antibiotics and deliver the infant.

Management of PPROM (< 34 weeks’ gestation)

For women who have PPROM at less than 34 weeks’ gestation, expectant management is usually preferred, and attempts should be made to prolong the latent period. A Cochrane meta-analysis of antibiotic treatment with PPROM (involving over 6000 women in 22 trials) found that the use of an antibiotic following PPROM reduced the risk of chorioamnionitis, prolonged the latency period, and reduced markers of neonatal morbidity (such as neonatal infection, use of surfactant, oxygen therapy, and abnormal cranial ultrasound). There are several different regimens of antibiotics that can be used, although two regimens were used in the largest PPROM randomized controlled trials showing a decrease in both maternal and neonatal morbidity. One recommended approach is “ampicillin (2 g IV every 6 hours) and erythromycin (250 mg IV every 6 hours) for 48 hours, followed by amoxicillin (250 mg by mouth every 8 hours) and enteric-coated erythromycin base (333 mg by mouth every 8 hours) for 5 days (Mercer protocol).” Another approach described is erythromycin 250mg orally every 6 hours for 10 days. Azithromycin as well as clarithromycin can be substituted for erythromycin in the appropriate doses without affecting latency or any other measured maternal or fetal outcomes.
Amoxicillin with clavulanic acid should not be administered as it appears to increase the risk of necrotizing enterocolitis in the presence of PROM.\textsuperscript{41}

Assessment of fetal lung maturity may be undertaken at the time of presentation by collecting pooled amniotic fluid from the vagina with a syringe and angiocatheter. Fluid should be sent for fetal lung maturity indices.

A single course of antenatal corticosteroids is recommended between 24\textsuperscript{0} and 33\textsuperscript{6} weeks of gestation in women with PPROM, and may be considered for women starting at 23\textsuperscript{0} weeks who are at risk of preterm delivery within 7 days, irrespective of membrane rupture.\textsuperscript{66-68} If chorioamnionitis is suspected, delivery is recommended, and expectant management is contraindicated.

In certain circumstances (e.g., transfer), tocolytics may be considered in consultation with a tertiary care centre.\textsuperscript{66,69,70} The use of progestogen does not prolong pregnancy in singleton gestations with preterm prelabour rupture of membranes.\textsuperscript{71}

Care providers should:

- **Not perform digital cervical examination.**
- Assess for infection; obtaining cultures, if indicated.
- Undertake ultrasound assessment of fetal position, cervical status, and fluid volume.
- Administer glucocorticoids between 24\textsuperscript{0} and 33\textsuperscript{6} weeks gestation.
- Administer antepartum antibiotics.
- Administer appropriate antibiotics and IOL if chorioamnionitis develops.
- Administer antibiotics for GBS prophylaxis, if indicated. Restart GBS prophylaxis at the onset of labour, if indicated.
- Consider transfer to tertiary care centre, if appropriate.

Amniotic fluid may be collected from vagina to assess fetal lung maturity.

In the case of expectant management, maintain surveillance for chorioamnionitis (monitoring maternal pulse and temperature, fetal heart rate, the development of uterine tenderness or irritability, and differential WBC changes if indicated).

**Summary**

1. PROM/PPROM occurs for many different reasons and at any gestational age.
2. Digital cervical examinations should not be performed; sterile speculum examinations are appropriate to confirm PROM/PPROM.
3. The primary motivation for considering IOL earlier than 37 weeks’ gestation in the presence of PPROM is that the risks associated with preterm birth are outweighed by those associated with maternal, fetal, and neonatal infectious morbidity.
References


Appendix A

Amniotic Fluid Ferning
Mag: x10

Table of Contents

Chapter 19 Antepartum and Intrapartum Hemorrhage................................................................. 454
  Definition .................................................................................................................................. 454
  Incidence .................................................................................................................................. 454
  Physiology ................................................................................................................................. 454
  Morbidity and Mortality ........................................................................................................... 454
Placenta Previa ............................................................................................................................ 455
  Definition .................................................................................................................................. 455
  Diagnosis .................................................................................................................................. 458
  Management ............................................................................................................................. 459
  Delivery .................................................................................................................................. 460
  Summary .................................................................................................................................. 461
Abnormal Placentation .................................................................................................................. 461
  Definition .................................................................................................................................. 461
  Diagnosis .................................................................................................................................. 462
  Management ............................................................................................................................. 463
  Summary .................................................................................................................................. 464
Placental abruption ............................................................................................................................. 464
  Definition .................................................................................................................................. 464
  Diagnosis .................................................................................................................................. 466
  Method and Timing of delivery ................................................................................................. 466
Vasa Previa ....................................................................................................................................... 467
  Definition .................................................................................................................................. 467
  Diagnosis .................................................................................................................................. 468
  Management ............................................................................................................................. 469
  Diagnosis and Management of Antepartum Hemorrhage (General) ........................................... 470
    Hemodynamically unstable woman ....................................................................................... 471
    Hemodynamically stable woman ......................................................................................... 471
  Summary .................................................................................................................................. 472
Appendix A ..................................................................................................................................... 479
Chapter 19
Antepartum and Intrapartum Hemorrhage

Definition
Antepartum hemorrhage is vaginal bleeding after 20 weeks' gestation.

Incidence
Antepartum hemorrhage occurs in 2% to 5% of all pregnancies. The most common identifiable causes of significant antepartum hemorrhage are:

- Placental abruption (incidence: 1 in 100 births)
- Placenta previa (incidence: approximately 1 in 200 to 300 births)
- Lower genital tract lesion

Physiology
In the non-pregnant state, the uterus receives approximately 1% of cardiac output, whereas in the third trimester it receives approximately 20%. Uterine bleeding in the third trimester can be massive and can result in hemodynamic instability.

Morbidity and Mortality
Placental abruption and placenta previa are leading causes of perinatal morbidity and mortality in the third trimester. Together, they account for slightly more than one half of the cases of antepartum hemorrhage.

Antepartum hemorrhage is a leading cause of maternal death in Canada. Placental abruption and placenta previa account for 50% of hemorrhage-related deaths, with postpartum hemorrhage accounting for the other 50%. Intrauterine growth restriction is reported in 16% of women with placenta previa. Antepartum bleeding is associated with an increased risk of preterm birth.
Placenta Previa

Definition

- **Placenta previa**: The placenta is touching or covering the internal os at term
- **Low-lying placenta**: The leading edge of the placenta lies within 2 cm of the internal os at term

On second trimester ultrasound from 18 weeks to 23 weeks, placenta previa is diagnosed when the inferior placental margin reaches or covers the internal cervical os on transvaginal scan. If on endovaginal ultrasound the inferior placental margin is less than 2 cm from but does not cross the internal os, placentation can be considered normal (Figure 1.)

Incidence

The incidence of placenta previa at term remains approximately one in 200.4,5

Risk Factors For Placenta Previa

- Previous placenta previa
- Previous Caesarean section, especially when the interpregnancy interval is < 12 months2
- Previous uterine surgery, including myomectomy and dilatation and curettage
- Advanced maternal age (≥ 35 years)
- Multiparity (≥ 3)
- Smoking or cocaine use in pregnancy
- Multiple gestation
- Pregnancy resulting from in vitro fertilization

Classification

Previously, the classification of placenta previa was based on physical examination and transabdominal sonography that would now be considered imprecise. It described a total (complete) previa as one that entirely covers the internal cervical os, a partial previa as partially covering the cervical os, and a marginal previa as lying next to the os. The use of the terms marginal and partial applied to the digital palpation of the placental edge through the cervix, which is has been replaced in the modern diagnosis of placenta previa by more precise transvaginal ultrasound measurements. The contemporary classification has been reviewed to address areas of controversy in placental location terminology. At term, if the placenta reaches or crosses the internal cervical os on transvaginal ultrasound, the diagnosis is placenta previa and the mode of delivery will be by Caesarean section (CS). If in the third trimester the inferior placental margin is low but does not reach the cervix, “low-lying placenta” is the preferred terminology and vaginal delivery may be considered depending on the distance between the inferior margin and the cervical os.
Historically, based largely on transabdominal ultrasound, the likelihood of bleeding during labour was found to be greater when the placenta is within 2 cm of the cervix near term (see Table 1). High-resolution endovaginal ultrasound has allowed refinement of this threshold. At 35 to 36 weeks’ gestation, the likelihood of safe vaginal delivery can be predicted using a transvaginal scan measurement from the inferior placental margin to the internal cervical os. Although based on a small number (approximately 250 women), the risk of bleeding during labour when the inferior placental margin is less than 11 mm from the internal os is above 70%. A pre-labour CS is recommended. For distances between 11 and 20 mm, the risk of bleeding in labour is substantially lower, and a trial of labour is considered acceptable after appropriate counseling. If a trial of labour is planned, Caesarean section and blood transfusion capability should be readily available.

A distance greater than 2 cm is considered safe for vaginal delivery; however, significant vaginal bleeding has been rarely described with a placenta-os distance greater than 2 cm.\textsuperscript{9,11}

### Table 1. Studies of low-lying placenta in which the outcome was Caesarean section performed for bleeding according to the placenta-os distance at or near term.

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>PLACENTA–OS DISTANCE, MM</th>
<th>CAESAREAN SECTION PERFORMED FOR BLEEDING (% OF THOSE IN LABOUR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dawson et al.\textsuperscript{12}</td>
<td>40</td>
<td>1 to 10</td>
<td>90</td>
</tr>
<tr>
<td></td>
<td></td>
<td>11 to 20</td>
<td>29</td>
</tr>
<tr>
<td>Bronsteen et al.\textsuperscript{13}</td>
<td>86</td>
<td>1 to 9</td>
<td>73</td>
</tr>
<tr>
<td></td>
<td></td>
<td>10 to 20</td>
<td>27</td>
</tr>
<tr>
<td>Vergani et al.\textsuperscript{14}</td>
<td>24</td>
<td>1 to 10</td>
<td>81</td>
</tr>
<tr>
<td>Vergani et al.\textsuperscript{14}</td>
<td>29</td>
<td>11 to 20</td>
<td>0</td>
</tr>
<tr>
<td>Al Wadi et al.\textsuperscript{9}</td>
<td>17</td>
<td>11 to 20</td>
<td>7.1</td>
</tr>
</tbody>
</table>
Figure 1. Diagnosis & Management of Placenta Previa

![Image of Placenta Previa Diagnosis & Management diagram]

Figure 1: Used with permission from Salus Global
Diagnosis

In the setting of undiagnosed vaginal bleeding in the late second and throughout the third trimester, pelvic examination should not be performed until placenta previa has been ruled out. Placenta previa characteristically presents clinically with painless vaginal bleeding in the second or third trimester. A small percentage of women with persistent placenta previa at term do not experience bleeding before labour.

Obstetrical Imaging

The advent of ultrasound has dramatically changed the clinician’s approach to placenta previa. Sonography has become the mainstay in diagnosing placenta previa. The goal is to clearly define the distance of the leading edge of the placenta from the internal cervical os and to determine whether the placenta partially or completely covers the internal cervical os.

Transabdominal Sonography

Transabdominal sonography (TAS) performed in the second trimester will detect over 85% of placenta previa cases. TAS evaluation of a placenta previa can be limited for several reasons, including poor visualization of a posterior placenta, myometrial contractions, obesity, and underfilling or overfilling of the bladder. For these reasons, TAS is associated with a false positive rate of up to 25% and a false negative rate of 7% for the diagnosis of placenta previa. Transabdominal ultrasound is inaccurate in the diagnosis of placenta previa and should be used only as a screening tool.

Transvaginal Sonography

Transvaginal sonography (TVS) is considered to be safe and is the gold standard for diagnosis, with a diagnostic accuracy rate of 99%. Accuracy rates for TVS are high (sensitivity 87.5%, specificity 98.8%, positive predictive value 93.3%, negative predictive value 97.6%). With TVS, the internal cervical os is seen as a discrete point, and the distance from this point to the leading edge of the placenta can be accurately measured. This should be done any time the placenta is situated low in the uterus and the distance from the os cannot be clearly delineated transabdominally. The measurement in millimeters by which the inferior margin is clear of the internal os or across the internal os should be reported. (Figure 1).

In the case of placenta previa with active bleeding, the clinician must proceed with caution, although TVS has been used safely in women with mild to moderate bleeding from a placenta previa.
Management

Clinical management

Most diagnoses of placenta previa are now made by routine second trimester ultrasound. If suspected on TAS, TVS can be used safely to verify the edge of the placenta and to accurately measure the shortest distance from the internal os. The gestational age when placenta previa is diagnosed is critical to management.

“Placental migration” involves the leading edge of the placenta apparently moving away from the cervical os as pregnancy progresses into the late third trimester. At 11 weeks to 14 weeks, approximately 40% of placentas will cover the internal os, yet by 18 weeks to 23 weeks, only 4% to 5% of women will have a low enough placenta on TAS to warrant TVS. On TVS, most of these will be found not to reach or cover the internal os. Only 1% to 2% of women will be found to have a placenta that reaches or covers the internal os on TVS at 18 weeks to 23 weeks.

In observational studies of 16,000 women, if the inferior margin of the placenta was not found to reach or cover the internal os on the 18- to 23-week TVS, the incidence of persistent placenta previa at term was zero. Therefore, unless the placental margin reaches or covers the internal os on the 18- to 23-week TVS, placentation can be considered normal. Follow-up ultrasound is usually not required. It is important to evaluate the placental insertion and the lower uterine segment around the inferior placental margin with Doppler to detect possible vasa previa (see section on vasa previa below). If there is any suspicion of vasa previa, follow-up ultrasound is indicated.

If the placenta reaches or covers the internal cervical os on second trimester TVS, then follow-up ultrasound is recommended to determine placental localization nearer term. In many of these women, placenta previa resolves, leaving only 0.3% to 0.4% with persistent placenta previa at term.

If placenta previa is diagnosed in the second trimester in an asymptomatic woman, there is no evidence for or against restriction on maternal activities. Ultrasound examination should be repeated in the third trimester at 30 to 32 weeks’ gestation to re-establish the position of the leading edge of the placenta. The likelihood of persistence of a placenta previa increases according to the gestational age at which it is first diagnosed. If TVS shows that the placenta overlaps the internal cervical os by ≥ 2.5 cm at 20 weeks to 23 weeks, or by 2 cm after 26 weeks, vaginal delivery is unlikely.

Expectant management

In a hemodynamically stable woman with bleeding from placenta previa remote from term, a policy of expectant management, pioneered by MacAfee, continues to be the standard. Hospitalization and aggressive transfusion and delivery as soon as fetal lung maturity is demonstrated have been shown to decrease perinatal mortality. However, 46% of women with a diagnosis of placenta previa deliver preterm, usually because of antenatal bleeding. Hospital admission with bed rest is an option, but carefully selected women with readily available access to intervention can be managed as outpatients. Administration of corticosteroids in selected patients is advised. Almost 75% of all women with placenta previa experience at least one episode of bleeding at a median gestational age of 29 weeks. The majority remain stable for a prolonged period and will not require delivery until a median of 36 weeks’ gestation.
Clinical outcomes of placenta previa are highly variable and cannot be predicted confidently from antenatal events. A number of retrospective studies and a randomized controlled trial provide evidence for the safety and cost-efficiency of the outpatient management of placenta previa. Women selected for outpatient management are those who are highly compliant, have no ongoing bleeding, are hemodynamically stable, live within a short travel distance from the hospital, and have immediate access to transportation to the hospital.

A Cochrane systematic review did not demonstrate any advantage of home care versus hospital admission in the following outcomes: episodes of bleeding requiring blood transfusion, Caesarean section, Caesarean hysterectomy, episodes of bleeding, gestational age at delivery, respiratory distress syndrome, birth weight, intraventricular hemorrhage, and neonatal sepsis.

**Delivery**

The mode of delivery for placenta previa is CS in an institution where blood transfusions and adult intensive care are available. Consent for possible total abdominal hysterectomy as a life-saving procedure in addition to Caesarean section should be obtained. It should be clearly documented in the chart if the patient does not want blood transfusions (for religious or other reasons), and these women should deliver in a tertiary care centre.

Delivery is recommended when the fetus reaches 37 weeks’ gestation, or in the presence of severe maternal hemorrhage or abnormal fetal surveillance at any gestation.

There is a risk that the placenta may be incised during a low transverse incision at CS. This may cause increased maternal and/or fetal blood loss. Rapid delivery of the baby will minimize blood loss. The patient should be counselled regarding the increased risk for blood transfusion and Caesarean hysterectomy due to persistent placental implantation site bleeding. Placental site uterine bleeding is not usually controlled by uterine muscle contractions and may require direct suturing of the placental bed. Direct injection of dilute vasopressin (5 to 10 units in 20 mL of saline) into the bleeding placental site can temporarily slow bleeding while these sutures are placed. The increased risk of placenta accreta and its associated complications should be considered by clinicians involved in the management of placenta previa and communicated to the woman. Surgeons should consider fertility preservation techniques before considering hysterectomy.

In a woman with a low-lying placenta who is undergoing a trial of vaginal birth, the following precautions should be considered:

- Minimize the number of vaginal examinations
- Do not strip membranes
- Do not use mechanical methods for cervical ripening
- Intravenous access throughout labour and delivery
- Group, screen and possibly cross match
- Be prepared for postpartum hemorrhage
Summary

1. Placenta previa is identified primarily by second trimester ultrasound.
2. Transvaginal ultrasound is recommended to confirm or refute the diagnosis whenever it is suspected on the basis of transabdominal ultrasound. The distance from the inferior placental margin to the internal cervical os or across the os should be reported.
3. Placenta previa is diagnosed when the inferior margin of the placenta reaches or covers the internal cervical os on a second trimester ultrasound scan. If the inferior margin does not reach the internal cervical os, placentation can be considered normal and no follow-up scan is required. Doppler study to preclude vasa previa is indicated.
4. Placenta previa identified in the second trimester must be reassessed by ultrasound in the third trimester.
5. The majority of placentas that reach or cross the internal cervical os in the second trimester resolve by term.
6. At term, if the placenta lies within 10 mm of or crosses the internal cervical os, Caesarean section is indicated. If the placenta is between 11 and 20 mm from the internal os, a trial of labour is reasonable with safety provisions in place for possible hemorrhage.
7. Women with a low-lying placenta are at elevated risk of PPH: pre-delivery preparation and aggressive management are indicated.
8. Premature birth is the primary fetal complication of placenta previa.
9. Selected women with asymptomatic placenta previa remote from term can be monitored in an outpatient setting.

Abnormal Placentation

Definition

Placenta accreta is the abnormal implantation of the placenta with villus attachment to the myometrium resulting in loss of the normal cleavage plane.

Placenta increta refers to trophoblast invasion into the myometrium.

Placenta percreta is placental invasion through the entire wall of the uterus and beyond the serosa of the myometrium, where it could invade the bladder and other pelvic organs.

Incidence

The incidence of placenta accreta has increased dramatically and is linked to the increased rate of Caesarean section. The American College of Obstetricians and Gynecologists estimates that placenta accreta complicates 1 in 2500 deliveries, a 10-fold increase over the past 50 years. A retrospective study of 64,359 deliveries between 1982 and 2002 reports an increase in Caesarean section rates from 12.5% (1982) to 23.5% (2002) and an overall incidence of abnormally invasive placenta (all forms) of between 2 and 90 per 10,000 births. A 2009 study by Flood et al. showed a decrease in peripartum hysterectomy over the last 4 decades. However, placenta accreta as being the indication has increased 10-fold, coinciding with the increase in CS rate.
Placenta accreta is most commonly associated with both placenta previa and previous CS. The incidence of placenta accreta in association with placenta previa (unscarred uterus) is approximately 3%. The incidence related to previous CS is shown in Table 2.

Table 2. Placenta Previa and Placenta Accreta (Includes Increta and Percreta) by Number of Prior Caesarean Sections

<table>
<thead>
<tr>
<th>PRIOR CAESAREAN SECTIONS, N</th>
<th>CAESAREAN SECTIONS, N</th>
<th>PLACENTA PREVIA, (%)</th>
<th>PLACENTA ACCRETA(^a), (%)</th>
<th>PROPORTION OF PLACENTA PREVIA WITH ACCRETA(^a), (%)</th>
<th>Hysterectomy(^a), (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>6201</td>
<td>6.4</td>
<td>0.2</td>
<td>13</td>
<td>0.7</td>
</tr>
<tr>
<td>1</td>
<td>15 808</td>
<td>1.3</td>
<td>0.3</td>
<td>11</td>
<td>0.4</td>
</tr>
<tr>
<td>2</td>
<td>6324</td>
<td>1.1</td>
<td>0.6</td>
<td>40</td>
<td>0.9</td>
</tr>
<tr>
<td>3</td>
<td>1452</td>
<td>2.3</td>
<td>2.1</td>
<td>61</td>
<td>2.4</td>
</tr>
<tr>
<td>4</td>
<td>258</td>
<td>6 (2.3)</td>
<td>2.3</td>
<td>67</td>
<td>3.5</td>
</tr>
<tr>
<td>≥5</td>
<td>89</td>
<td>3 (3.4)</td>
<td>6.7</td>
<td>67</td>
<td>9.0</td>
</tr>
</tbody>
</table>

\(^a\) Includes increta and percreta. \(P < 0.001\) from Cochran-Armitage test for trend

\(^b\) Increased risk with increasing number of Caesarean sections \(P < 0.001\)


Risk Factors\(^{42, 46, 47}\)
- Placenta previa
- Prior CS
- Prior myomectomy
- Asherman’s syndrome
- Endometrial ablation
- Submucous leiomyomata (fibroids)
- Maternal age greater than 35 years

Diagnosis
Placenta accreta should be considered in any woman with a placenta previa. Transabdominal and transvaginal ultrasound\(^{48, 50}\) can detect placenta accreta in up to 85% of cases. Additional investigations using ultrasound Doppler\(^{51, 54}\) and MR\(^{55, 59}\) may be helpful to increase the detection of a placenta accreta in women at risk. Placenta accreta is suspected on second trimester sonographic examination under the following findings.\(^{39}\)
1. The loss of the “clear space” or hypoechogenic space between the placenta and myometrium. It is sensitive but not specific.
2. Bladder line interruption. The interface between the uterus and bladder is represented on grey scale sonography as a continuous white line. Its loss is seen best on transvaginal ultrasound with a partially filled bladder; it is a result of increased vascularity in this space. Its specificity is 96% to 100%.
3. Presence of lacunae, which have high-velocity and low-resistance Doppler flow and are irregular in cross-section on grey-scale ultrasound.
4. Myometrial thickness of less than 1 mm or loss of myometrial thickness higher in the uterus.

The accuracy of diagnosis appears to improve when more than one ultrasound finding is present.

The goal is to diagnose placenta accreta during the antenatal period. Strong clinical suspicion is required in the presence of risk factors. The clinician should aim to rule out an accreta before performing a Caesarean section. Women with suspected placenta accreta should deliver in facilities with adequate resources and personnel to manage the potential complications. In a vaginal delivery, if a lack of a clear cleavage plane is noted during attempted manual removal of the placenta, clinicians should be aware that further forceful attempts to remove the placenta could result in severe hemorrhage.\textsuperscript{60, 61}

Management

The management of placenta accreta requires a multidisciplinary team approach involving representatives of anaesthesiology, blood bank, and other services that may include interventional radiology, urology, and vascular surgery.\textsuperscript{62} When the presence of a placenta accreta is known or suspected antenatally, delivery should take place in a facility with the resources necessary to deal with the potential complications of this condition (preferably a Level III centre).

Caesarean hysterectomy is required in up to 72% of cases.\textsuperscript{63} The treatment of placenta accreta requires clinical judgement and will depend on the clinical situation and whether the diagnosis is made antepartum or intrapartum. Factors that will influence management include the woman’s wish for future fertility, ease of placental removal, success of suture hemostasis, amount of bleeding, patient condition, and whether there is a placenta accreta, increta, or percreta. When bleeding is uncontrollable and the woman is at significant risk of hemodynamic collapse, Caesarean hysterectomy may be necessary. The use of a temporary intra-abdominal aortic balloon is a promising technique to reduce blood loss and the need for hysterectomy.\textsuperscript{64} The use of Cell Saver technology in obstetrics appears to be safe and has been shown to reduce the need for blood transfusion during difficult surgical management of invasive placenta.\textsuperscript{65, 66}

When the decision to perform a Caesarean hysterectomy is made antenatally, the uterine incision is made away from the placental insertion, usually at the uterine fundus. Following delivery of the baby, the cord is clamped, the placenta is left untouched to avoid excessive bleeding, and a hysterectomy is performed. Caesarean hysterectomy can be a complex surgical procedure and requires a skilled pelvic surgeon.

Reports of successful conservative treatment in women who strongly desire future pregnancy have been published. A 2007 study reviewed 60 carefully selected cases of conservative treatment in which either uterine preservation was requested, or risk of damage to surrounding pelvic organs was extremely high because of placenta percreta. Success rate
in this study was 80%. However, in general, the number of reported cases has been small and a significant number of the women in these studies experienced postpartum endometritis and late postpartum hemorrhage. For this reason, conservative management could be attempted in very specific and select cases after multi-disciplinary approach and patient informed consent.

Although there is no consensus with respect to the best time for elective delivery, the period between 34 and 35 weeks appears optimal. Conservative treatments have included:

- The insertion of a balloon catheter through the femoral artery into the internal iliac or the uterine vessels before the surgery. The balloon is inflated during dissection to prevent excessive bleeding and the placenta is left in situ. Postoperative embolization of the uterine arteries is carried out.
- Uterine or internal iliac artery ligation
- Curettage and/or over-sewing of the placental site in the case of a localized area of accreta
- Utero-vaginal packing
- Leaving the placenta in place and performing uterine artery embolization
- Leaving the placenta in place and undertaking close postpartum follow-up

Summary

1. The incidence of placenta accreta has increased significantly, mainly because of increased CS rates.
2. Placenta accreta should be suspected in the presence of a placenta previa, especially when there is a history of prior uterine surgery.
3. The goal is to diagnose placenta accreta during the antenatal period.
4. Diagnosis is generally made by ultrasound; MRI can be used in certain cases.
5. Delivery should take place in a centre with the human resources and facilities to manage the potential complications.
6. Management requires a multidisciplinary team in a tertiary care centre.
7. A conservative approach may be attempted in selected patients.
8. Definitive therapy is Caesarean hysterectomy and should not be delayed in the case of severe hemorrhage.

Placental abruption

Definition

Placental abruption is the premature separation of the placenta from the uterine wall.

Physiology

Bleeding into the decidua basalis leads to placental separation. The most probable cause of the bleeding is a process involving a separation between the decidua and the placenta. Hematoma formation may further separate the placenta.
from the uterine wall and decreases the placental villous surface available for gas and metabolic exchange. If the
underlying condition is not self-limiting, bleeding will continue and may extend through the myometrium to the serosa
(a Couvelaire uterus). Bleeding may spread between the decidua and the fetal membrane and pass through the cervix
or may extravasate through the membranes into the amniotic fluid. The amount of blood seen vaginally often does not
correlate with the severity of the abruption.

Incidence
Placental abruption occurs in 0.5% to 1% of all pregnancies in North America.\textsuperscript{40,49}

Risk Factors

- All hypertensive disorders of pregnancy are important risk factors for placental abruption. A history of a
  previous abruption increases the probability of recurrent abruption in subsequent pregnancies to between
  5.5% and 16.6%.\textsuperscript{70}
- Ischemic placental disease: there is evidence suggesting a link between preeclampsia, small for gestational
  age, and abruption. It is as yet unclear if this triad is causal or simply an association.\textsuperscript{71}

Predisposing Factors and Relative Risk\textsuperscript{40}

- Prior placental abruption 10% to 25%
- Inherited thrombophilia 3% to 7%
- Preterm rupture of membranes 2.4% to 4.9%
- Hypertension 2.1% to 4.0%
- Iron deficiency\textsuperscript{72} 2.4%
- Multiple gestation 2.1%
- Hydramnios 2%
- Chronic hypertension 1.8% to 3.0%
- Maternal age and parity 1.3% to 1.5%
- Smoking 1.4% to 1.9%
- Trauma
- Cocaine abuse
- Previous Caesarean section,\textsuperscript{6} especially when the interpregnancy interval is < 12 months.\textsuperscript{2}

\textbf{Most abruptions are idiopathic.\textsuperscript{40}}
Diagnosis

1. Do not undertake a digital pelvic examination until placenta previa has been ruled out by prior or current ultrasound.
2. History and physical examination. Clinical differences will give the first clues to the diagnosis.
3. Placental abruption cannot be reliably diagnosed by ultrasound examination.
4. Once placenta previa has been ruled out, speculum examination should be performed to assess for lower genital tract bleeding.
5. Electronic fetal monitoring (EFM) and ultrasound will assist in the assessment of fetal well-being.
6. Abdominal pain is usually the presenting symptom of placental abruption. It is generally constant and greatest at the site of the placental attachment.
7. Uterine contractions, hypertonus, and/or irritability are present in most cases.
8. Vaginal bleeding is usually present and the clinician should be aware that the degree of vaginal bleeding may not relate to the severity of the abruption.
9. Not all these signs and symptoms must be present to consider a diagnosis. For example, abruption without vaginal bleeding (concealed abruption) is reported in some cases.

Method and Timing of delivery

Management depends on the hemodynamic condition of the mother, fetal well-being, gestational age, and the degree of cervical dilatation. A classification of placental abruption that may prove useful in guiding management decision-making is shown in Table 3. This classification is based on fetal condition. The degree of bleeding and the maternal hemodynamic status will also influence management.

Table 3. Classification of Placental Abruption

<table>
<thead>
<tr>
<th>ABRUPTION</th>
<th>DEFINITION</th>
<th>MANAGEMENT</th>
</tr>
</thead>
</table>
| Mild      | Evidence of abruption with no fetal compromise | • Conservative management if preterm  
• Initiate delivery if fetal maturity (induction with continuous EFM if cervix favourable) |
| Moderate  | Evidence of abruption with fetal compromise | • Emergency delivery regardless of gestational age  
• Induce if atypical EFM, favourable cervix, and continuous EFM available  
• CS if abnormal EFM or unfavourable cervix |
| Severe    | Evidence of abruption with fetal death | • Initiate delivery process (non-emergent)  
• Be vigilant for disseminated intravascular coagulopathy |
Vasa Previa

Definition
Fetal vessels in the membranes run across the cervical os in front of the presenting part. It can be found with a velamentous insertion of the umbilical cord or with a succenturiate placental lobe where the umbilical cord vessels are unsupported in the membranes. In these circumstances, the blood vessels may tear during spontaneous labour or during spontaneous or artificial rupture of the membranes. Occasionally the vessels may rupture spontaneously in the antenatal period.

Figure 2: Velamentous Insertion & Vasa Previa
Incidence

Vasa previa occurs in 1 in 2000 to 5000 pregnancies.\textsuperscript{73, 74} The incidence is higher in twin pregnancies (due to the increased incidence of velamentous cord insertion) and also in the presence of placenta previa.

Risk Factors

- Velamentous insertion of the cord
- Pregnancy following in vitro fertilization
- Placenta previa
- Presence of succenturiate lobe
- Twin pregnancy

Morbidity and Mortality

When vasa previa is undiagnosed prior to labour, fetal mortality is estimated to be as high as 60%.\textsuperscript{75, 76} Antenatal diagnosis is not always possible; however, when antenatal diagnosis is made, up to 97% neonatal survival rate is possible.\textsuperscript{77}

Diagnosis

In the presence of risk factors, antenatal diagnosis should be considered. Using a standardized ultrasound screening protocol in pregnancies with risk factors, Rebarber et al. identified 31 cases out of 27 573 pregnancies, for an incidence of 1.1 per 1000.\textsuperscript{10}

Routine documentation of the placental cord insertion during the second trimester ultrasound should be done to screen for vasa previa.\textsuperscript{11} If vasa praevia is suspected, a repeat scan at 30 weeks is recommended since spontaneous resolution of the vasa previa can occur. Women with a low-lying placenta on second trimester ultrasound should have the lower uterine segment investigated with Doppler ultrasound to detect any errant fetal vessels near the internal cervical os.

Prenatal diagnosis is now possible using transvaginal ultrasound to observe fetal vessels crossing the internal os or coursing within 2 cm of the os. A prenatal diagnosis mandates closer observation of the woman and delivery before term by an elective CS. Some suggest hospital admission at 30 weeks to 32 weeks with CS at 35 weeks to 36 weeks.\textsuperscript{76} Occasionally, the vessels may be detected on routine ultrasound or felt on digital examination before rupture of the membranes. A clinical diagnosis should be considered when rupture of the membranes is associated with acute painless vaginal bleeding and an abrupt change in the fetal heart rate (tachycardia, bradycardia, or sinusoidal pattern). A bedside Apt test or Wright’s stain on vaginal blood to detect fetal hemoglobin would suggest the diagnosis, although its clinical utility is questionable and availability may be limited and should not delay management.
Management

If vasa previa is diagnosed and persistent at 30 weeks, glucocorticoids and admission at 30 weeks to 32 weeks to a centre with a minimum Level II capability can be considered. The pediatric team should be consulted antenatally. A planned Caesarean section at 35 to 36 weeks’ gestation is recommended. The patient’s chart should be clearly labelled with the diagnosis to facilitate immediate recognition and CS should rupture of membranes or vaginal bleeding occur. Delay may result in fetal or neonatal death and must be avoided. With this approach more than 97% neonatal survival is expected.\(^75\)

When vasa previa is not known to be present, rupture of the membranes associated with acute painless vaginal bleeding and an abrupt change in the fetal heart rate (tachycardia, bradycardia, or sinusoidal) make the clinical diagnosis. Immediate Caesarean section is required to prevent fetal exsanguination.
Diagnosis and Management of Antepartum Hemorrhage (General)

1. Obtain history and perform physical examination. Clinical differences will give the first clues to the diagnosis.

Table 4: Comparative clinical presentation and risk factors of placental abruption and placenta previa (*these are NOT exclusive*)

<table>
<thead>
<tr>
<th>PLACENTAL ABRUPTION</th>
<th>PLACENTA PREVIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age over 35</td>
<td>Multiparity \textsuperscript{28}</td>
</tr>
<tr>
<td>In vitro fertilization</td>
<td>Previous uterine surgery</td>
</tr>
<tr>
<td>Preterm labour</td>
<td>Painless (unless in labour)</td>
</tr>
<tr>
<td>Smoker\textsuperscript{28}</td>
<td>Uterus not tender</td>
</tr>
<tr>
<td>Hypertensive disorders (pre-existing and gestational)</td>
<td>Uterus soft</td>
</tr>
<tr>
<td>Uterine over distension, abdominal trauma</td>
<td>No uterine irritability/contractions</td>
</tr>
<tr>
<td>Abdominal pain or backache (often unremitting)</td>
<td>Malpresentation or high presenting part</td>
</tr>
<tr>
<td>Uterine tenderness</td>
<td>Fetal heart usually normal</td>
</tr>
<tr>
<td>Increased uterine tone</td>
<td>Shock and anemia correspond to apparent blood loss</td>
</tr>
<tr>
<td>Uterine irritability/contractions</td>
<td>Coagulopathy very uncommon initially</td>
</tr>
<tr>
<td>Usually normal presentation</td>
<td>Transvaginal ultrasound is the definitive diagnostic test for placenta previa.</td>
</tr>
<tr>
<td>Fetal heart may be absent, atypical or abnormal</td>
<td></td>
</tr>
<tr>
<td>Shock and anemia disproportionate with apparent blood loss</td>
<td></td>
</tr>
<tr>
<td>May have coagulopathy</td>
<td></td>
</tr>
<tr>
<td>Placental abruption may be seen on transabdominal ultrasound but a negative ultrasound does not rule out abruption.</td>
<td></td>
</tr>
</tbody>
</table>

2. Determine hemodynamic stability and evaluate uterine tone and activity.
3. Evaluate fetal well-being including electronic fetal monitoring and ultrasound. **Do not perform a pelvic examination until placenta previa has been ruled out.**
4. Perform an ultrasound examination to rule out placenta previa, if possible, before performing a speculum examination. Speculum examination is performed to assess the cervix for dilatation or for any lower tract lesion.
5. A team capable of performing an emergency Caesarean section should be available.
6. Given the inaccuracies in estimating blood loss and the maternal ability to withstand hemorrhage, it is essential to maintain careful surveillance of the maternal hemodynamic status and fetal well-being.

7. Laboratory assessment should include
   a. cross match blood
   b. complete blood count (hemoglobin, hematocrit, platelet count)
   c. other investigations dictated by the presence of comorbid conditions (e.g., hypertension)

8. Rh immune globulin should be given to all unsensitized Rh negative women with any bleeding or a suspected concealed abruption.
   a. A Kleihauer-Betke test is appropriate in this setting to assist in determining the required dose of Rh immune globulin.
   b. 300 mcg of Rh immune globulin should be given for every 30 mL of fetal blood detected in the maternal circulation (equivalent to 15 mL of packed red blood cells).

a) Hemodynamically unstable woman

   The two immediate objectives for those women actively bleeding and hemodynamically unstable are fluid replacement and delivery.

   While expediting delivery the following management steps occur concurrently:
   
   • Ongoing assessment of maternal (vital signs, urine output) and fetal well-being.
   • Active fluid resuscitation and/or blood transfusion through two large bore intravenous lines.
   • Maternal oxygen saturation monitoring.
   • Oxygen administration for all women who are hypotensive because oxygen consumption is increased 20% in pregnancy and the fetus is sensitive to hypoxia.

   If bleeding is due to a placenta previa or placental abruption and maternal or fetal health is compromised, a Caesarean section will be necessary (unless vaginal delivery is imminent). Disseminated intravascular coagulation should be considered. A bedside clot test (no visible clotting in 6 minutes at room temperature) may be helpful. If a coagulopathy is present, it must be corrected immediately with fresh frozen plasma or cryoprecipitate. Delivery should be performed as soon as the clotting factors have been corrected and volume replacement is adequate. A protocol for massive transfusion is helpful. The risk of DIC is increased if the woman presents with an intrauterine fetal death.

   If maternal and fetal status is stable and local resources are not available to manage the woman or her baby, consider transfer to a high-risk centre.

b) Hemodynamically stable woman

   • Continue maternal and fetal surveillance for 12 to 24 hours. Appropriate attention should be paid to the maternal hemodynamic status.
   • If the woman has suffered abdominal trauma and is ≥ 20 weeks’ gestation, it is recommended that she be monitored for a minimum of four hours after the trauma. Placental abruption is seen in about 7% of such
cases. If there is more than one contraction in 15 minutes or there are ominous signs such as bleeding or uterine pain, the duration of surveillance should be longer (at least 24 hours).

- If the fetus is preterm, expectant management may be appropriate depending on the maternal hemodynamic status and fetal well-being. Antepartum corticosteroids are indicated for a gestational age of 24 weeks to 34 weeks. Weigh the risk of significant subsequent bleeding against fetal maturity.
- Transfer to a high-risk centre may be indicated based on the maternal or fetal condition and local resources.
- All women with an antepartum hemorrhage are at risk of recurrent bleeding.

**Summary**

1. A standard protocol for the management of antepartum hemorrhage is recommended.
2. A “massive transfusions” protocol is recommended in each delivery unit.
3. A medical directive for nursing and midwifery staff to initiate management is recommended.
4. The life-threatening nature of placental abruption and placenta previa for both woman and fetus should be borne in mind, as should the potential for rapid evolution of these conditions.
5. Vigorous resuscitation should be undertaken when appropriate.
6. Ultrasound determination of placental location should precede pelvic examination when the situation allows.
7. An antenatal diagnosis of vasa previa warrants the management course previously outlined in this document to optimize fetal and neonatal outcome.
8. Ongoing surveillance of the maternal, placental, and fetal status and appropriate, active management are required.
References


Antepartum Hemorrhage

Assess maternal hemodynamic status and fetal well-being

Mother and fetus unstable

- Resuscitation
- Continuing instability
- Deliver and watch for DIC

Mother and fetus stable

- Ultrasound examination
- Fetal surveillance

Placental Abruption

- Live fetus
  - Condition abnormal/atypical
    - Deliver
  - Condition normal
    - Gestational age
      - Mature
        - Vaginal delivery or CS
      - Immature

- Dead fetus ± coagulopathy
  - Delivery (watch for DIC)

Previa

- Gestational age
  - Mature
    - Caesarean section (watch for accreta)
  - Immature

Not Previa

- Speculum:
  - Contact bleeding
  - Inflammation / infection
  - Labour
  - Cervical lesion (e.g., cancer)
- Follow-up appropriately

Condition abnormal/atypical

Condition normal

Gestational age

Mature

Immature

Deliver

Consider:
- Steroids
- Transfusion
- Transfer

Consider:
- Corticosteroids
- Transfusion
- Transfer
Table of Contents

Chapter 20 Prevention of Early-Onset Neonatal Group B Streptococcal Disease ................................................................. 481
  Introduction ........................................................................................................................................................................... 481
  Incidence ............................................................................................................................................................................. 481
  Morbidity and Mortality .................................. 482
  Physiology .............................................................. 482
  Risk Factors ................................................................. 482
  Prevention ................................................................. 483
    Antepartum Screening ............................................... 483
  Culture technique ............................................................. 484
  Management ................................................................. 485
    Antepartum Management ............................................... 485
    Intrapartum Management ............................................... 486
    Antibiotic prophylaxis ................................................... 487
    Current recommendations ............................................... 488
    Recommended intrapartum antibiotic prophylaxis ..................... 489
  Management of Infants ...................................................... 489
    Potential effects of intrapartum antibiotics on neonates and infants .......................................................... 490
    Current recommendations for infants of mothers who had an indication for GBS prophylaxis ...................... 490
  Summary .............................................................................................................................................................................. 494
Chapter 20

Prevention of Early-Onset Neonatal Group B Streptococcal Disease

Introduction

Our understanding of how to prevent early-onset group B streptococcus disease in neonates is evolving, and there is, as yet, no consensus.

Evidence from several population-based studies and the publication of guidelines from the Society of Obstetricians and Gynaecologists of Canada,1 the Canadian Paediatric Society (CPS),2 and the US Centers for Disease Control and Prevention (CDC)3 have led to the current recommendations.

Incidence

GBS is a gram-positive bacterium that causes invasive disease primarily in infants, pregnant or postpartum women, and older adults (the highest incidence is among young infants).1 It remains the most significant cause of early-onset sepsis in the term infant.4 Approximately 10% to 30% of pregnant women are colonized in the vagina or rectum. This colonization may be transient, intermittent, or persistent.3

Without intrapartum antibiotic prophylaxis (IAP), approximately 40% to 70% of neonates born to GBS carriers will be colonized. Of these infants, 1% to 2% will develop early-onset GBS infections, giving an overall incidence of approximately 1.8 per 1000 live births.5

GBS disease can be early-onset (< 7 days) or late-onset (7 days to 3 months).

Coinciding with active prevention efforts in the 1990s, the incidence of early-onset GBS disease declined to approximately 0.5 per 1000 live births in 1999; and to 0.24 cases per 1000 live births by 2014.6 An international 2012 systematic review of 42 studies (published 2000 to 2011; only 81% reported the use of IAP) estimated the global burden of early-onset disease to be 0.43/1000 live births (95% confidence interval [CI] 0.37 to 0.49). The incidence was highest in Africa (0.53), followed by the Americas (0.50), Europe (0.45), and Southeast Asia (0.11). The case fatality rate was 12.1%.7

Widespread use of intrapartum antibiotic prophylaxis for GBS disease has led to concerns about the potential adverse impact on the incidence of E. coli early onset neonatal disease, Reassuringly however, a 2017 US multistate review by Schrag et al, covering the years 2005 to 2014 found no evidence of an increasing burden of early-onset E coli infections.8
Morbidity and Mortality

GBS has been associated with stillbirth and is responsible for asymptomatic bacteriuria, urinary tract infection, and chorioamnionitis during pregnancy.

GBS may cause endometritis and wound infection in the early postpartum period.

From 89% to 95% of neonatal early-onset GBS infection presents in the first 24 hours of life and may be characterized by bacteremia, pneumonia, or meningitis. A 2003 case-control study found that nearly 40% of neonates with GBS meningitis will be left with moderate to severe neurologic disability. The overall mortality rate ranges from 5% to 12%.

Physiology

The maternal lower gastrointestinal tract is the primary reservoir for GBS and is the likely source of vaginal colonization.

Early-onset neonatal infection may occur when the fetus aspirates GBS in amniotic fluid that has ascended from the vagina after the onset of labour or rupture of the membranes, although GBS also can invade through intact membranes. Infection can also occur as the fetus passes through the birth canal. Most infants exposed to GBS will remain well but may have their mucous membranes, gastrointestinal, and respiratory tracts colonized with GBS.

Risk Factors

Significant risk factors for early-onset disease include:

- a previous infant with invasive GBS disease
- GBS bacteriuria (of any level of colony-forming units per mL) during current pregnancy
- maternal colonization with GBS (positive screening culture within 5 weeks of labour or membrane rupture)

When maternal GBS culture status is unknown, they also include:

- preterm labour (< 37 weeks' gestation)
- amniotic membrane rupture ≥18 hours
- intrapartum temperature ≥ 38°C

A 2018 case controlled study from India, where routine antepartum GBS screening is not practiced (a risk-based intrapartum antibiotic prophylaxis strategy is used) and where the incidence of early onset GBS disease is 0.55/1000 live births, found that >3 vaginal pelvic exams after rupture of membranes was a significant risk factor (OR 8.57 95% CI 3.10-23.6) for early onset GBS disease. The authors concluded that where antepartum screening is not available, their findings should guide caregivers to reduce unnecessary vaginal examinations after ROM.

Note: If GBS status is unknown, knowledge of colonization status in a previous pregnancy may be considered along with the above risk factors. Four retrospective studies published since 2008 have reported a GBS colonization recurrence rate
in a subsequent pregnancy of 38% to 53%. The authors of the 2015 review suggested that prior GBS colonization be considered as an indication for intrapartum antibiotic prophylaxis in women with unknown GBS status.

It is important to note, however, that a 2008 retrospective review found that 38% of the 65 neonates diagnosed with early-onset GBS had no recognizable antepartum or intrapartum predisposing risk factors (including known maternal colonization). A more recent (2016) retrospective review from Winnipeg found that in the 13 cases of early-onset GBS disease identified between 2009 and 2013, 9 of the mothers had screened negative for GBS. This emphasizes the importance of the recommendation that any neonate presenting with signs of sepsis should be evaluated appropriately.

**Prevention**

The findings of a small prospective, double-blind randomized trial conducted in Taiwan suggest that probiotics may reduce GBS colonization at the time of birth. The study compared oral probiotic (2 strains of lactobacillus) with placebo given to GBS positive women at 35 to 37 weeks and found that 43% in the probiotic group were negative at delivery compared with 18% in the placebo group. However, because of the trial’s limitations, more study is necessary to corroborate these findings.

**Antepartum Screening**

On the basis of a large multicentre retrospective analysis and other supporting studies, including a 2015 systematic review, it is recommended that all pregnant women be screened at 35 to 37 weeks’ gestation for GBS colonization and that all positive women be treated with intrapartum antibiotic prophylaxis at the time of membrane rupture or labour. The findings of these and other studies suggest that the universal screening approach is over 50% more effective than the risk-based approach in preventing perinatal GBS disease. In addition, a 2017 meta-analysis of 14 studies provided reliable evidence that prophylactic antibiotics for GBS-colonized women significantly reduce the vertical transmission from colonized mothers to their infants and the risk of all cause infections, early onset GBS infection and non-GBS infections.

A 2009 systematic review found that the positive predictive value (PPV) of screening for the presence of GBS colonization at delivery decreased when the interval between screening and delivery increased, especially if it was greater than 5 to 6 weeks. The PPV of cultures done before 35 weeks was 59% to 63% and of those done after 35 weeks, 70% to 93%. Interestingly, the negative predictive value (NPV) of screening done before 35 weeks (90% to 93%) was only slightly lower than the NPV (95% to 98%) found after 35 weeks. Because the accuracy of the PPV correlates with shorter duration between culture and delivery, this review confirmed the recommendation to screen at 35 to 37 weeks. It also noted, however, that antenatal testing will fail to detect GBS in approximately 6% of colonized women.

If a GBS screening culture is done before 35 to 37 weeks (e.g., threatened preterm labour) and any time that the interval between the culture and delivery is > 5 weeks, it should be repeated. Applying a screening-based strategy will not necessarily increase antibiotic use. A 1998 to 1999 multi-state review of labour and delivery records suggested that the perfect implementation of either the screening or the risk-based strategies would result in the same proportion of women receiving intrapartum antibiotics (24%).
There has been research on the applicability of using a rapid test to detect GBS status. A 2017 study of 902 women in Denmark (where the GBS prevalence is 12%) concluded that in a population with a low prevalence of GBS, intrapartum PCR assay performs better than the antepartum culture for identification of GBS vaginal carriers during labour. A 2015 study of 200 vaginal/rectal swabs submitted for GBS culture and 3 commercially available nucleic acid amplification tests (NAATs) following 18- to 24-hour broth enrichment found the culture to be positive in 15%; however, 31.5% were positive by at least 1 NAAT (sensitivity of the NAATs was 90.9% to 100%, whereas the sensitivity of culture was 53.6%). Concerns with intrapartum testing methods include the time the test would take to be reported and the absence of sensitivity data for women who have anaphylactic allergy to penicillin. Although the 2010 CDC guideline states that current data do not support NAAT, it also states that in settings where NAAT is available, a positive result could be an indication for intrapartum GBS prophylaxis in the limited circumstance of a woman at term with unknown colonization status and no other risk factors.

**Culture technique**

- A current standardized method should be used for collection, transport, testing, and reporting.
- It is essential that the result of the swab be available at the intended place of delivery and that the woman be informed of the result and the recommended interventions.

A single swab should be taken from the lower one third of the vagina and then from the anorectum (through the anal sphincter). Failure to swab the anorectum is associated with a significant false negative rate. A 2018 study from Denmark reported an overall combined rectovaginal GBS colonization of 17%, however, 9.0% were colonized only in the vagina and 13% colonized only in the rectum.

- Cervical cultures are not recommended, and a speculum is not used.
- The swab should be placed in non-nutritive transport medium (e.g., Amies or Stuart). Although this medium will maintain GBS viability for up to 4 days at room temperature, recovery does decline over this period, but this can be minimized if the specimen is kept refrigerated. The sensitivity is greatest when the specimen is stored at 4°C before culture and processed within 24 hours of collection.
- There is evidence that self-collection of swabs by pregnant women, with appropriate instruction, in the clinic examination room or washroom, is as effective as collection by care providers. However, a 2018 study of 422 Hong Kong women who performed self-screening (after instructions including a video) and also had screening by a health care worker found the sensitivity of self-screening to be only 61% whereas that by the health care worker to be 98%. The authors recommended that cultural difference needs to be considered if implementing self-screening.
- If the woman is suspected to be allergic to penicillin
  - Skin testing can be done safely during pregnancy.
  - If she does not have confirmatory testing and has a history of a Penicillin allergy including a Type I IgE hypersensitivity reaction and no documented allergy to cefazolin, she can safely receive cefazolin for intrapartum prophylaxis. Type I IgE hypersensitivity reaction includes: anaphylaxis, urticaria, angioedema, hypotension, bronchospasm, stridor, pruritus.
Recent literature has demonstrated that allergy cross reaction between penicillin and the cephalosporins is due to similarities in their chemical side chains.

- Cefazolin does not share a similar side chain to any penicillin or any other cephalosporin available in Canada and can be safely administered in the case of suspected penicillin or cephalosporin allergy other than to cefazolin itself.

- If the reaction to penicillin or cephalosporins is a severe non-IgE mediated hypersensitivity reaction, cefazolin should be avoided and sensitivity testing for positive GBS cultures should be requested.

  - Non-IgE mediated hypersensitivity reactions, usually > 72 hours after exposure include: delayed reactions such as toxic epidermal necrolysis, Stevens-Johnson syndrome, drug reaction with eosinophilia and systemic symptoms syndrome, severe hepatitis, interstitial nephritis, or hemolytic anemia. 38

  - Sensitivity testing for positive GBS cultures: Penicillin allergy should be stated on the culture requisition along with a request to perform sensitivity testing for clindamycin. GBS has become increasingly resistant to clindamycin (13% to 28%). 39,40 A 2012 study of 309 GBS isolates in Alberta found resistance clindamycin to be 22%. 41

The following chart outlines the risk of GBS disease when antibiotics are not used in women with or without risk factors. It indicates that neonatal GBS disease is most likely to occur when women screen positive and have risk factors.

**Table 1: Risk of Early-Onset GBS Disease in the Absence of Antibiotic Prophylaxis**

<table>
<thead>
<tr>
<th>RISK FACTORS* PRESENT</th>
<th>RISK FACTORS* ABSENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Culture positive</td>
<td>1:25</td>
</tr>
<tr>
<td>Culture negative</td>
<td>1:1100</td>
</tr>
<tr>
<td>Culture unknown (or not done)</td>
<td>1:120</td>
</tr>
</tbody>
</table>

*In this study, the risk factors (different from those currently recommended by the CDC) were defined as preterm labour (< 37 weeks), prolonged membrane rupture (> 12 hours), or intrapartum fever (> 37.5°); these are slightly different from those used in the current guidelines. 42

**Management**

**Antepartum Management**

Women with antepartum GBS urinary tract infections (symptomatic or with colony counts > 100 000 CFU/mL [10⁸ CFU/L]) should be treated with appropriate antibiotics for the prevention of adverse pregnancy outcomes such as pyelonephritis. 43 Asymptomatic women with GBS colony counts < 100 000 CFU/mL in pregnancy should not be treated with antibiotics for the prevention of adverse maternal and perinatal outcomes such as pyelonephritis, chorioamnionitis, or preterm birth. 43 If antibiotics are given to treat GBS bacteriuria, they typically do not eliminate GBS colonization. 2 Women found to have GBS bacteriuria in any concentration should be regarded as colonized at delivery. They do not require further GBS screening and should receive antibiotic prophylaxis in labour. 3
There is no benefit in treating women with positive GBS vaginal cultures before labour or membrane rupture.\textsuperscript{3} Research continues on development of a vaccine that could be administered to pregnant women and potentially provide protection for infants during their period of vulnerability to GBS invasive disease (up to 3 months of age).\textsuperscript{44}

**Intrapartum Management**

No method prevents all GBS disease. Women should be advised that current management approaches will not always eliminate GBS disease.

A 2014 Cochrane review\textsuperscript{45} identified only 3 RCTs (n = 500) of intrapartum antibiotics for known maternal GBS colonization, and noted serious concerns with all 3 regarding bias. The review concluded that intrapartum antibiotic prophylaxis appears to reduce neonatal early-onset GBS disease but that there was a lack of evidence from well-designed and conducted trials. Intrapartum antibiotics compared with no treatment for GBS-positive women resulted in the following:

- **A significant reduction in the incidence of early-onset GBS infection** (RR 0.17; 95% CI 0.04 to 0.74; number needed to treat to benefit (NNTB) 25, 95% CI 14 to 100)
- No statistically significant effect on
  - neonatal mortality from all causes, from GBS infection, or neonatal mortality from infections by bacteria other than GBS
  - the incidence of late-onset (≥ 7 days) GBS infection
  - the incidence of neonatal sepsis, meningitis, urinary tract infection or pneumonia due to bacterial organisms other than GBS
  - maternal peri- and/or postpartum sepsis, puerperal infection

Similar to the Cochrane review\textsuperscript{45} a 2018 review of the effectiveness of intrapartum antibiotic prophylaxis (IAP) (including one systematic review, three clinical trials and five observational studies) concluded that studies have found IAP to be effective. However the studies did have significant methodological flaws and have not considered the short and long term risks of IAP.\textsuperscript{46}

The aims of IAP are to:

- decrease maternal colony counts
- prevent ascending maternal infection
- achieve effective concentrations of antibiotic in the fetus during labour

The duration of intravenous antibiotic prophylaxis is important and will guide the management of the neonate. Current guidelines recommend that prophylaxis with penicillin, ampicillin, or cefazolin given for ≥ 4 is optimal. A 2013 retrospective analysis of 7691 women estimated that the effectiveness of penicillin to prevent early-onset GBS disease given for ≥ 4 hours was 89%; when given < 4 hours the effectiveness decreased to 38% to 47%.\textsuperscript{47} A 2014 prospective study of 60 women who were vaginal-rectal culture GBS positive at 35 to 37 weeks found only 43 (72%) to be positive at the time of labour. Of the 43 receiving IV penicillin G prophylaxis, 20 (47%) remained positive at 2 hours, and 5 (12%) were still positive at 4 hours. This study supported the recommendation of a ≥ 4-hour optimal duration for IAP.\textsuperscript{48}
However, when labour is progressing rapidly and is not expected to last ≥ 4 hours, it is recommended that prophylaxis be started as soon as possible because there is evidence that even a short duration of antibiotic therapy (e.g., 1 to 2 hours) will reduce the risk of neonatal colonization and early-onset GBS disease of the newborn. Furthermore, a study of fetal penicillin G levels after maternal IV administration demonstrated that the levels peaked at 1 hour and that fetal serum levels far exceeded the minimal inhibitor concentration at durations of well under 1 hour. The effectiveness of antibiotics other than penicillin and cefazolin (e.g., clindamycin and vancomycin) given for a short duration before delivery has not been demonstrated. Studies have reported that placental transfer of these antibiotics occurs but at a slower rate.

**Antibiotic prophylaxis**

- IV penicillin G is the drug of choice unless the woman is allergic to penicillin. In North America GBS remains universally susceptible to the penicillin and cefazolin. For women with for penicillin or cephalosporin allergy (other than specifically to cefazolin) it is now recognized that it is safe to administer cefazolin.
- For women with documented cefazolin allergy or non-IgE mediated hypersensitivity reactions to penicillin or cephalosporin, then penicillin and cefazolin should be avoided and sensitivities should be obtained.
  - Clindamycin should be used if the GBS isolate is sensitive to clindamycin. Maternal administration of clindamycin has been shown to reach therapeutic cord blood levels within 1 to 2 hours. If the GBS isolate is resistant to clindamycin, vancomycin is indicated. GBS remains highly sensitive to vancomycin, which also has a satisfactory transplacental passage. A 2018 prospective study from the USA concluded that by 2 hours after vancomycin infusion vaginal GBS colony counts declined significantly to 6.7% of the initial value and were essentially zero within 6 hours.
- A 2003 Alberta retrospective review of 90 cases of early-onset GBS disease (between 1993 and 1997), before the recommendation for universal GBS maternal screening, identified intrauterine monitoring as an independent risk factor for early-onset GBS disease (odds ratio [OR] 2.24; 95% CI 1.22 to 4.13). However, a case-control study (covering the years 2000 to 2011) of 40 cases of early-onset neonatal sepsis (EONS) made up of 8 GBS, 11 *Escherichia coli*, 12 coagulase negative staphylococci, 4 viridans group streptococci, one *Enterococcus faecalis*, and 4 other non-specified organisms versus 80 controls, did not demonstrate a significant relationship between fetal scalp electrode use and EONS.

Although there is a theoretical concern about performing procedures such as membrane stripping and mechanical and/or pharmacologic cervical ripening on GBS-colonized women, the 2010 CDC guideline suggested that "the available data are not sufficient to determine whether these procedures are associated with an increased risk for early-onset disease." A 2015 prospective study of 542 women (135 were GBS+) treated with routine membrane stripping beginning at 40 weeks, found no difference in maternal or neonatal outcomes.

Topical chlorhexidine as a vaginal disinfectant in labour to prevent early-onset GBS infection was assessed in a 2014 Cochrane review. The authors reported that vaginal chlorhexidine was not associated with reductions in early-onset GBS sepsis, and although it may reduce GBS neonatal colonization it was associated with increased maternal side effects (stinging or local irritation). The conclusion did not support its use for the preventing early-onset GBS disease.
A 2014 review of 309 cases of early-onset disease estimated that the potential reduction in cases with optimal prevention implementation could have been as much as 26% to 59%. The most common errors were in prenatal screening (36%) and intrapartum prophylaxis (54%). In 60% of cases, there was more than one error.\(^{62}\)

**Current recommendations**

- Intrapartum antibiotic prophylaxis at the time of membrane rupture or labour should be administered for all women who have had the following:
  - previous infant with invasive GBS disease
  - GBS bacteriuria during current pregnancy
  - positive screening culture, routinely done at 35 to 37 weeks during current pregnancy
- If GBS status is unknown, intrapartum prophylaxis should be administered for any of the following:
  - preterm labour (< 37\(^{0}\) weeks)
  - rupture of membranes for > 18 hours
  - maternal fever ≥ 38°C (this is best detected by core temperature measurement e.g., rectal).

Note: If GBS status is unknown, knowledge of colonization status in a previous pregnancy may be considered along with the above risk factors. Retrospective studies published since 2008 have reported a GBS colonization recurrence rate in a subsequent pregnancy of 38% to 53%.\(^{14,17}\)

- Preterm labour with intact membranes
  - If GBS status negative within 5 weeks, antibiotic prophylaxis is not indicated.
  - If GBS status unknown, GBS vaginal/rectal screening should be performed on admission, and intrapartum prophylaxis is recommended until the results of culture are known. Antibiotics may be stopped if culture is negative.
- Prelabour rupture of membranes (PROM) and preterm PROM (PPROM) (management is discussed in the chapter on PROM).
  - For women with term PROM who are colonized with GBS, it is recommended that intrapartum prophylaxis be initiated and labour induced with oxytocin\(^1\)\(^,\)\(^{63}\) or misoprostol.\(^{64}\) Analysis of data from the Term PROM study found an overall decreased risk of maternal infection (as indicated by clinical chorioamnionitis, antibiotics during labour, or postpartum fever) in women who were induced with oxytocin compared with those in whom labour was induced with prostaglandin or who were managed expectantly.\(^{65}\) For women colonized with GBS, the Term PROM trial also found that induction of labour with oxytocin resulted in reduced rates of neonatal infection compared with those in whom labour was induced with vaginal prostaglandin or who were managed expectantly.\(^{66}\)
  - For women with PPROM (< 37 weeks) not in labour with unknown GBS status, IV GBS prophylaxis should be given for 48 hours (or less if the GBS culture proves negative) along with additional antibiotics, if indicated (e.g., to prolong latency), while awaiting spontaneous or obstetrically indicated labour.\(^1\)
- Situations in which GBS intrapartum prophylaxis is not indicated:
- Previous pregnancy with a positive GBS screening culture (unless a culture was also positive within 5 weeks in the current pregnancy).
• Planned Caesarean section performed in the absence of labour or membrane rupture (regardless of maternal GBS culture status). However, routine 35- to 37-week GBS culture is indicated in these women in case they experience labour or PROM before the planned Caesarean section.3
• Negative vaginal and rectal GBS screening culture, within 5 weeks, regardless of intrapartum risk factors.
• Chorioamnionitis: regardless of GBS status, this diagnosis should be considered in the presence of is maternal fever > 38°C, fetal tachycardia, and signs of maternal sepsis. Maternal oral temperature may be inaccurate; consider rectal temperature measurement. If chorioamnionitis is present, treat with broad spectrum antibiotics (e.g., ampicillin and gentamicin, cefoxitin etc.) and expedite delivery.

Recommended intrapartum antibiotic prophylaxis

• Preferred (narrow spectrum): IV penicillin G 5 million units, followed by 2.5 or 3 million units q4h
  – alternative: IV ampicillin 2 g followed by 1 g q4h
• In the presence of a penicillin or cephalosporin (other than cefazolin) allergy
  – administer cefazolin 2 g IV, followed by 1 g every 8 hours until delivery
• In the presence of documented cefazolin allergy or non-IgE mediated hypersensitivity reaction to penicillin or cephalosporins
  – if the GBS isolate is sensitive to clindamycin, administer clindamycin 900 mg IV, every 8 hours, until delivery
  – if the GBS isolate is not sensitive (or unknown) to clindamycin administer vancomycin 1 g IV, every 12 hours until delivery.

Management of Infants

Management of asymptomatic infants of women who had an indication for GBS prophylaxis is based on clinical presentation, the adequacy of the maternal intrapartum chemoprophylaxis, gestational age, the duration of membrane rupture, and the degree of maternal fever if present.3 Studies have indicated that 89% to 95% of infants who develop early-onset GBS infection have clinical signs within 24 hours (e.g., temperature instability, tachycardia, poor peripheral perfusion, respiratory distress) or an abnormal CBC (e.g., total WBC count < 5.0x10⁹/L or absolute neutrophil count (ANC) <1.5 x 10⁹/L). Four percent of infants will develop signs between 24 hours and 48 hours, and only 1% after 48 hours.67 A 2012 retrospective study of 140 000 women at a single centre found 94 neonates with early-onset GBS sepsis. In 93, this was diagnosed in the first hour of life, and most of these had evidence of sepsis peripartum with an increase in preterm delivery, Caesarean section, low Apgar scores, and abnormal umbilical cord pH and base deficit.68

• Adequate intrapartum antibiotic prophylaxis is defined as ≥ 4 hours of IV penicillin, ampicillin, or cefazolin before delivery.3,67
• Inadequate intrapartum antibiotic prophylaxis is defined as < 4 hours of IV penicillin, ampicillin, or cefazolin before delivery or when alternative antibiotics (e.g., clindamycin, vancomycin) are used. There is insufficient evidence regarding these alternative antibiotics to consider them adequate for purposes of neonatal management.3,67 This recommendation is supported by a 2013 retrospective analysis, which found that although penicillin given for ≥ 4 hours was 89% effective in preventing early-onset disease, this
Potential effects of intrapartum antibiotics on neonates and infants

- There is increasing interest in the effect of intrapartum maternal antibiotic administration on the neonate’s microbiome. A 2015 study of 198 healthy term infants found that intrapartum antibiotics (including those used for GBS prophylaxis and for prophylaxis at Caesarean section) were associated with changes in the neonate’s gut microbiota. It appeared that breastfeeding modified some of this effect. The authors concluded that further research is warranted to explore the healthy consequences of this association.

- One concern about antenatal and neonatal antibiotic exposure is a potential increase in the child's later diagnosis of allergic disease. Two retrospective studies published in 2015 offer some reassurance, however.
  - A study from Pennsylvania of 492 women (who delivered vaginally) and their children found no increase in atopic dermatitis when intrapartum antibiotics were used for < 24 hours.
  - A study of 80 children with penicillin allergy and 724 without found that intrapartum antibiotics did not alter the risk of penicillin allergy.

Current recommendations for infants of mothers who had an indication for GBS prophylaxis

For full-term infants that appear well

- when the mother received adequate intrapartum antibiotic prophylaxis: there is no need for septic work-up, additional therapy, or investigations. If these infants are well and other discharge criteria are met, they may be discharged from hospital after 24 hours if those who will be caring for the infant at home are knowledgeable about the signs of sepsis and have ready access health care resources. This recommendation is supported by Berger et al. (2012), whose cost-effectiveness analysis suggested that with adequate IAP, discharging asymptomatic term neonates after 24 hours is preferred over 48 hours inpatient observation.

- when the woman received inadequate intrapartum antibiotic prophylaxis:
  - the CPS statement recommends careful in hospital assessment (e.g., close observation with vital signs every 3 to 4 h) for at least 24 hours. A CBC is no longer routinely recommended. If the infant is well and other discharge criteria are met, the infant may be discharged after 24 hours if those who will be caring for the infant after discharge are knowledgeable about the signs of sepsis and have ready access to care. If these conditions not met, infants should be observed in hospital for 48 hours.
  - the CDC guideline recommends that the infant should be observed for ≥ 48 hours:
    › for infants delivered at ≥ 37 weeks’ gestational age and when membranes ruptured < 18 hours, no routine diagnostic testing is recommended.
    › for infants delivered at < 37 weeks’ gestational age or when membranes ruptured ≥ 18 hours, a blood culture and CBC with differential and platelets is recommended.
For GBS-positive mothers who have additional risk factors (ruptured membranes ≥ 18 hrs or fever ≥ 38°C), whether or not they received adequate intrapartum antibiotic prophylaxis, the CPS statement recommends clinicians consider the severity of each factor, the intrapartum antibiotic exposure, and clinical status of the infant to determine an individualized management plan. Recommendations include close observation for 24 to 48 hours, and a CBC at 4 hours of age if needed for decision-making.

A 2017 Irish retrospective study by Nielsen et al reported that of the 53 cases of EOGBS disease occurring in 112,361 births, only 3 had no clinical suspicion for sepsis. The number of blood cultures taken to detect one case of GBS bacteraemia in an infant who is well at the time of testing was 3996. This study highlighted that infected babies generally present early in the postpartum period meaning that an observational approach in certain GBS-exposed infants could be considered safe.

A 2014 study of 38 infants with early-onset sepsis (including 10 cases of GBS) found that maternal intrapartum antibiotic treatment did not prolong the incubation time required for blood cultures to become positive.

Preterm infants require individualized evaluation and management. However, the CPS and the CDC suggest that well-appearing infants delivered at ≥ 35 weeks’ gestation whose mothers receive adequate intrapartum antibiotic prophylaxis do not need routine diagnostic testing or therapy for prevention of early-onset GBS disease, but they should be observed for 48 hours before discharge home.

Infants of mothers with chorioamnionitis require close observation for at least 24 hours and individualized strategies. These will often include a diagnostic and therapeutic evaluation for sepsis.

In the case of a planned home birth, it is important that the same principles of care for the mother and neonate are provided and maintained.

Symptomatic infants are at very high risk for morbidity, so consultation, investigation, and treatment should be undertaken early. There is no laboratory test that has sufficient sensitivity to rule out early-onset sepsis. All infants with signs of sepsis must be treated immediately with IV antibiotics (e.g., amoxicillin and aminoglycoside) following prompt investigation including CBC, blood culture, LP, and chest X-ray if respiratory distress is present. These infants are best managed in an intensive care facility.

Signs of neonatal sepsis:

- Respiratory distress (apnea, tachypnea)
- Seizures
- Temperature instability, poor peripheral perfusion
- Tachycardia
- Lethargy, hypotonia
- Poor feeding
- Acidosis
Figure 1. Algorithm for the secondary prevention of early-onset group B streptococcal disease among newborns

| Signs of neonatal sepsis? | YES | Full diagnostic evaluation\(^a\)  
Antibiotic therapy\(^b\) |
|--------------------------|-----|--------------------------------------|
| NO                       |     | Close observation\(^a\) for 24 hrs  
Individualize management but will often\(^a\) include limited evaluation\(^l\)  
and antibiotic therapy\(^b\) |
| Maternal chorioamnionitis\(^c\) | YES | Routine clinical care\(^g\) |
| NO                       |     | Close observation\(^a\) for  
≥ 37 wks: 24 hrs\(^i\)  
35–36 wks: 48 hrs\(^i\) |
| GBS prophylaxis indicated  
for mother | NO | Routine clinical care\(^g\) |
| YES                      |     | Close observation\(^d\) for  
≥ 37 wks: 24 hrs\(^i\)  
≥ 24K–48 hrs |
| Adequate maternal intravenous prophylaxis\(^h\) | NO | Limited evaluation\(^l\)  
Close observation\(^a\) for ≥ 48 hours |
| ≥37 weeks, no maternal fever and  
membrane rupture < 18 hrs | YES | Close observation\(^d\) for  
≥ 37 wks: 24 hrs\(^i\)  
35–36 wks: 48 hrs\(^i\) |
| NO                       |     | Close observation\(^d\),g for  
24K–48 hrs |
| < 37 wks or maternal fever ≥ 38°C  
or membrane rupture ≥ 18 hrs | YES | Limited evaluation\(^l\)  
Close observation\(^a\) for ≥ 48 hours |

\(a\) CBC, blood culture, LP & CXR (if respiratory symptoms)  
\(b\) Antibiotics: ampicillin + coverage for other potential organisms (e.g., aminoglycoside)  
\(c\) Diagnose on clinical grounds: fever, tender uterus, purulent/foul amniotic fluid  
\(d\) Close observation: in hospital, including vital signs every 3 to 4 hours  
\(e\) With multiple risk factors, no intrapartum maternal antibiotics or mother unwell  
\(f\) CBC± blood culture  
\(g\) If signs of sepsis → full diagnostic evaluation & antibiotic therapy  
\(h\) Penicillin, ampicillin, or cefazolin for ≥ 4 hrs before delivery  
\(i\) May be discharged at 24 hrs if criteria are met and the parents/family have access to care  
\(j\) If the infant is well, and if those who will provide care for the infant at home are knowledgeable about signs of sepsis and have ready access to care  
\(k\) If maternal fever ≥ 38°C or membrane rupture ≥ 18 hrs: observation for 24 to 48 hrs and consider CBC at 4 h

Modified from the 2010 CDC guideline; and the CPS 2017 Statement\(^l\)
Figure 2. Management of Term Infants at Risk for Early Onset Bacterial Sepsis

Flow chart for management of term infants (infants ≥37 weeks’ gestational age) who are at risk for early onset bacterial sepsis.

GA, gestational age; GBS, Group B Streptococcus; CBC, complete blood count; LP, lumbar puncture; CXR, chest X-ray; IAP, intrapartum antibiotic prophylaxis.

Algorithm from the CPS Fetus and Newborn Committee, Position Statement, January 2017.
Summary

1. No protocol prevents all GBS morbidity or mortality.
2. Women with GBS bacteriuria in the current pregnancy or who had a prior infant with invasive GBS disease do not need to be screened and require intrapartum prophylaxis.
3. Screening of all other women at 35 to 37 weeks' gestation (or within 5 wks of delivery) is recommended, with intrapartum prophylaxis, if positive.
4. Antepartum treatment of GBS colonization is not justified with the exception of GBS urinary tract infection (≥ 10^5 CFU/mL).
5. Individual centres must adopt strategies for GBS disease prevention.
6. All infants with signs of sepsis must be treated empirically with IV antibiotic once cultures have been taken.
References


# Table of Contents

Chapter 21 Venous Thromboembolism and Amniotic Fluid Embolus ................................................................. 502

Introduction .......................................................................................................................................................... 502
  Definitions ........................................................................................................................................................ 502
  Incidence .......................................................................................................................................................... 503

Pathophysiology .................................................................................................................................................. 503

Morbidity and Mortality ..................................................................................................................................... 504
  Risk Factors for Venous Thromboembolism ................................................................................................. 505

Prevention of VTE ............................................................................................................................................. 506
  Thromboprophylaxis .................................................................................................................................... 506
  Consent .......................................................................................................................................................... 509

Diagnosis ............................................................................................................................................................ 510
  Diagnosis of DVT ......................................................................................................................................... 510
  Diagnostic Tests ........................................................................................................................................... 510
  Pelvic/Ovarian Vein Thrombosis .................................................................................................................. 512
  Diagnosis of Pulmonary Embolism ................................................................................................................ 512
  Diagnostic Tests ........................................................................................................................................... 512

Management of DVT and Pulmonary Embolism in Pregnancy ......................................................................... 515
  Available Anticoagulants .............................................................................................................................. 515
  Non-Medical Management ............................................................................................................................ 518
  Anticoagulation during Pregnancy .................................................................................................................. 518
  Caring for Women During the Acute Phase of Anticoagulation ................................................................ 519
  Management of Labour and Delivery on prophylactic Heparin ................................................................... 521
  Management of Labour and Delivery on Therapeutic Heparin .................................................................. 521
  Postpartum Management .................................................................................................................................. 522

Summary ............................................................................................................................................................ 522

Amniotic Fluid Embolism .................................................................................................................................. 523
Chapter 21

Venous Thromboembolism and Amniotic Fluid Embolus

Introduction

The incidence of thrombophlebitis and deep vein thrombosis in pregnancy has decreased dramatically over the past six decades. This reduction is very likely the result of reduced prolonged bed rest as an antenatal therapy and early postpartum ambulation. However, pulmonary embolism remains a leading cause of maternal death.

This chapter reviews venous thromboembolism (VTE) in pregnancy and the puerperium:

- Associated risk factors
- Pathophysiology
- Signs and symptoms
- Diagnostic tools to investigate VTE
- Management of acute thromboembolic events
- Thromboprophylaxis to prevent VTE
- Care of women receiving anticoagulant medications

Definitions

**Thrombophlebitis:** Inflammation of the wall of a vein leading to clot (thrombus) formation at the site of the inflammation.

**Deep vein thrombosis (DVT):** Thrombus formation in the deep veins, usually of the lower extremity but occasionally in the pelvic veins.

**Venous thromboembolism (VTE):** A term that includes DVT and embolism of a thrombus that breaks free from venous thrombosis and travels to another organ, usually the lung.

**Post-thrombotic leg syndrome:** leg pain, swelling, dermatitis, and ulcers caused by permanent damage to leg vein valves from prior DVT.

**Pulmonary embolism (PE):** transport of a thrombus, usually from a DVT, through the venous circulation and heart to the pulmonary circulation where it becomes trapped. Small emboli lodge in small lung vessels and can dissolve spontaneously. Larger emboli can lead to cardiopulmonary failure and death.
Low molecular weight heparin (LMWH): a fractionated heparin given subcutaneously for VTE prophylaxis or therapy. It has a better side effect profile than unfractionated heparin and does not require platelet monitoring; however it cannot be reversed with protamine.

Incidence

The incidence of VTE is 1-2/1000 pregnancies; however, pulmonary embolism is responsible for 9.2% of maternal deaths in the US.\(^5\)

In a large prospective health database in the United Kingdom, the incidence of VTE during the first and second trimester was not elevated; however the incidence in the third trimester was 6-fold higher than in non-pregnant women.\(^11\) Additionally, “The first 6 weeks postpartum was associated with a 22-fold increase in risk, with the peak occurring in the first 3 weeks (postpartum). The rate then declined rapidly.” Despite this high relative risk, the absolute risk per birth is low.

DVT occurs most often in the left leg during pregnancy and in the puerperium.\(^13,16\) This is thought to be due to compression of the left iliac vein by the right iliac artery the weight of the pregnant or postpartum uterus. PE occurs more often postpartum than in pregnancy and is more often associated with Caesarean section.\(^9,10,12\)

Pregnant women with mechanical heart valves have a particularly high risk of VTE. Studies published in the 1990s reported thromboembolic events in 7% to 23%, with valve thrombosis in 50%, leading to a mortality rate of up to 40%.\(^24\) More recent studies suggest that modern valves are safer, with associated mortality rates of 1% to 4%.\(^24\) Anticoagulation of patients with mechanical heart valves is problematic because of a preference for warfarin, which is known to have fetal effects.\(^23\) Involvement of a hematologist familiar with pregnancy and cardiology is recommended. Mitral valve stenosis is also a risk factor for VTE and warrants prophylaxis.

Pathophysiology

Pregnancy and the puerperium predispose to the development of VTE because of increases in each of the factors in Virchow’s triad:

**Figure 1: Virchow’s triad**
Coagulation proteins are generally elevated in pregnancy which favors blood clot formation. However, there is also an enhancement of physiological fibrinolysis. The balance between these two physiological tendencies is not well understood. Plasminogen activator levels return to normal within 1 hour of birth.\textsuperscript{26}

### Table 1. Physiologic tendencies enhancing clot formation in pregnancy

<table>
<thead>
<tr>
<th>HYPERCOAGULABILITY</th>
<th>STASIS</th>
<th>VESSEL WALL INJURY</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Increased clotting factors &amp; fibrinogen\textsuperscript{27}</td>
<td>• Decreased venous tone</td>
<td>• Vascular damage during birth (Caesarean section or assisted vaginal birth)\textsuperscript{29}</td>
</tr>
<tr>
<td>• Increased platelet aggregation</td>
<td>• Reduced venous flow from the legs (uterine compression of pelvic veins)</td>
<td></td>
</tr>
<tr>
<td>• Decreased protein S, tissue plasminogen activator, factors XI, XIII\textsuperscript{28}</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Increased resistance to activated protein C</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Morbidity and Mortality

When a DVT occurs, prompt treatment must be initiated to reduce:

- Extension of the DVT
- Morbidity and mortality associated with pulmonary embolism (PE)
- Post-thrombotic leg syndrome

Pulmonary embolism is a leading cause of maternal death in North America and Europe. Post-thrombotic syndrome is a permanent and disabling condition. Sixty-five percent of women who experience DVT during pregnancy will develop venous insufficiency within 7 years – a higher proportion than in non-pregnant women.\textsuperscript{31}

### Table 2. Diagnoses associated with maternal deaths in Canada (excluding Quebec), 2002–2003 to 2009–2010

<table>
<thead>
<tr>
<th>DIAGNOSIS</th>
<th>N</th>
<th>MATERNAL MORTALITY RATE PER 100 000 BIRTHS</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ante- &amp; intra-partum hemorrhage</td>
<td>15</td>
<td>0.7</td>
<td>0.4–1.1</td>
</tr>
<tr>
<td>Puerperal infection</td>
<td>18</td>
<td>0.8</td>
<td>0.5–1.4</td>
</tr>
<tr>
<td>Ectopic-abortion/molar pregnancy</td>
<td>19</td>
<td>0.9</td>
<td>0.5–1.4</td>
</tr>
<tr>
<td>Pulmonary &amp; amniotic fluid embolism</td>
<td>30</td>
<td>1.4</td>
<td>0.9–2.0</td>
</tr>
<tr>
<td>Hypertension</td>
<td>34</td>
<td>1.6</td>
<td>1.1–2.2</td>
</tr>
<tr>
<td>Postpartum hemorrhage</td>
<td>34</td>
<td>1.6</td>
<td>1.1–2.2</td>
</tr>
<tr>
<td>Cardiovascular disease</td>
<td>67</td>
<td>3.1</td>
<td>2.4–3.9</td>
</tr>
<tr>
<td>Other indirect causes</td>
<td>44</td>
<td>2.0</td>
<td>1.5–2.8</td>
</tr>
</tbody>
</table>

Source: Canadian Institute for Health Information, Discharge Abstract Database.\textsuperscript{32}

Notes:
- Diagnoses do not represent underlying cause of death. Cases could have more than one associated diagnosis hence sum of individual diagnoses exceeds the overall maternal mortality rate.
- Manitoba data, which were incomplete for earlier years, were included from 2004–05.
Risk Factors for Venous Thromboembolism\textsuperscript{33, 34}

In retrospective case-control studies from large databases, many risk factors have been associated with VTE in pregnant and postpartum women.\textsuperscript{35} However, the magnitude of absolute risk with most risk factors is very small. To what extent risk accumulates in women with more than one risk factor is unknown.\textsuperscript{36} Pregnant women with a potent thrombophilia or personal history of VTE are at particularly high risk.

Table 3: Comparative incidence of VTE by risk factors\textsuperscript{37}

<table>
<thead>
<tr>
<th>RISK FACTOR</th>
<th>RISK OF VTE (PER 1000 WOMAN-YEARS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Use of third generation contraceptive pill</td>
<td>0.3</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>1.23</td>
</tr>
<tr>
<td>Puerperium</td>
<td>3.2</td>
</tr>
<tr>
<td>Pregnancy in thrombophilic woman</td>
<td>40</td>
</tr>
<tr>
<td>Pregnancy and previous VTE</td>
<td>110</td>
</tr>
</tbody>
</table>

Table 4. Risk factors associated with VTE

<table>
<thead>
<tr>
<th>GENETIC</th>
<th>BEFORE PREGNANCY\textsuperscript{38}</th>
<th>PREGNANCY RELATED\textsuperscript{34}</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thrombophilias\textsuperscript{15, 39, 40}</td>
<td>Previous VTE\textsuperscript{44-46}</td>
<td>ICU admission</td>
</tr>
<tr>
<td>- Factor V Leiden\textsuperscript{42-44}</td>
<td>Antiphospholipid antibody syndrome</td>
<td>Caesarean section\textsuperscript{15, 48, 49}</td>
</tr>
<tr>
<td>- Protein C &amp; deficiency Protein S\textsuperscript{14}</td>
<td>Mechanical heart valve</td>
<td>Operative vaginal delivery</td>
</tr>
<tr>
<td>- Antithrombin deficiency</td>
<td>Mitral valve stenosis</td>
<td>Preeclampsia\textsuperscript{31, 45, 52}</td>
</tr>
<tr>
<td>- Prothrombin gene variant\textsuperscript{2, 27}</td>
<td>Family history of VTE\textsuperscript{45}</td>
<td>Pelvic surgery postpartum</td>
</tr>
<tr>
<td></td>
<td>Age $&gt; 35$</td>
<td>Bed rest\textsuperscript{15, 18}</td>
</tr>
<tr>
<td></td>
<td>BMI$&gt; 30$kg/m\textsuperscript{2}</td>
<td>Infection\textsuperscript{48}</td>
</tr>
<tr>
<td></td>
<td>Medical disease (e.g. nephrotic syndrome, sickle cell disease, inflammatory bowel disease, cancer)</td>
<td>Multiple pregnancy</td>
</tr>
<tr>
<td></td>
<td>Smoking</td>
<td>Gestational diabetes</td>
</tr>
<tr>
<td></td>
<td>Atrial fibrillation</td>
<td>Dehydration</td>
</tr>
<tr>
<td></td>
<td>Current IV drug use\textsuperscript{49}</td>
<td>Postpartum hemorrhage</td>
</tr>
<tr>
<td></td>
<td>Parity $\geq 3$</td>
<td>Preterm birth</td>
</tr>
<tr>
<td></td>
<td>Gross varicose veins</td>
<td>Stillbirth\textsuperscript{48}</td>
</tr>
<tr>
<td></td>
<td>Paraplegia\textsuperscript{49}</td>
<td>Prolonged labour ($&gt; 24$ h)</td>
</tr>
</tbody>
</table>
Prevention of VTE

Women should avoid bed rest during pregnancy and should mobilize early after birth, particularly following Caesarean section (CS). Compression and/or intermittent pneumatic compression stockings have been recommended during periods of required bed rest. Intermittent ambulation is advised during prolonged air or automobile travel.

Selective screening for thrombophilia (e.g., factor V Leiden) on the basis of positive personal or family history of VTE is cost-effective, but universal population screening is not.

Thromboprophylaxis

Preventing VTE in hospitalized patients is a priority worldwide. The goal is to identify patients at elevated risk of VTE and provide them with chemoprophylaxis – usually low molecular weight heparin (LMWH). This approach has been shown to be beneficial for patients at particularly high risk of VTE (major orthopedic surgery, active cancer, ICU admission, prolonged bed rest, prior history of VTE, and potent thrombophilia). However, for most hospitalized patients who lack these risk factors, benefit is not well-established.

Research using asymptomatic DVT as an outcome is misleading, since more than 90% of asymptomatic DVT resolve without treatment. The absolute risk of clinical VTE in most hospitalized patients is 1% or less, and randomized trials using clinical VTE as an outcome show little or no benefit in most patients. Since LMWH therapy carries a risk of bleeding, newer guidelines have scaled back recommendations and emphasize the importance of carefully weighing any reduction in VTE against bleeding risk.

In an effort to prevent mortality and morbidity from VTE in birthing women, the obstetrical profession also adopted this approach. Women with a potent thrombophilia, antiphospholipid antibody syndrome, or personal history of VTE have up to 10% risk of VTE during pregnancy. Unless there is active bleeding or a high risk of bleeding, they should receive LMWH during pregnancy and for at least four weeks postpartum. Withholding LMWH or switching to unfractionated heparin at term can be considered to lower the risk of intrapartum and postpartum hemorrhage and to allow regional anaesthesia.

In an effort to extend this benefit to more birthing women, several professional organizations have published guidelines that recommend LMWH for women with more common and less potent risk factors. Using relative risks from case-control studies, dozens of risk factors have been incorporated into complex scoring tools that identify up to 50% of birthing women as high-risk, including up to 85% of those delivering by CS. Amid some controversy, the American College of Obstetricians and Gynecologists has not followed suit.

These risk scoring tools have not been validated in prospective trials. When applied retrospectively to a birthing population that did not receive heparin, they did not detect the few women who developed VTE. There is not observational or experimental evidence that LMWH is beneficial for women with common risk factors, who make up a vast majority of women identified as "high risk." Cochrane reviewers have noted this lack of evidence and called for randomized trials.

The absolute risk of VTE for women with common risk factors is very low and is inadequately reported in guidelines. Several factors inflate the implied benefit of LMWH:
• Case control studies have been used to provide relative risks for VTE without attention to the magnitude of risk.
• Similar ICD 9/10 codes for superficial & deep thrombosis; amniotic fluid and venous pulmonary embolism; and heparin prophylaxis vs. treatment inflate the incidence of VTE in case control studies by up to 100%.
• Screening studies of asymptomatic rather than clinical DVT have been used in a risk analysis model that underpins several guidelines.
• Data used to estimate VTE risk after CS is taken from non-pregnant general surgical patients who have a risk that is ten-fold higher.
• VTE risk for the full postpartum period has not been adjusted for the reality that LMWH is usually only given for one week.

The actual magnitude of postpartum VTE risk is very small and difficult to establish. The best prospective data comes from a large prospective UK database. After CS, the postpartum incidence was 1.5/1000, or 0.35/1000 during the first postpartum week. The highest risk was after stillbirth: 1.35/1000 during the first postpartum week. Assuming 70% protection from LMWH approximately 4000 women would need to be treated for one week after CS and 1000 after stillbirth to prevent one VTE (NNT=4000 and 1000 respectively).

Even women with multiple common risk factors have a low risk of VTE. In a scoring model based on the UK database and validated using a Swedish database, a 20 Y/O woman with a BMI of 32 undergoing CS in labour has a VTE risk of 1.1/1000 over 6 postpartum weeks, or approximately 0.27/1000 during the first postpartum week. This gives a NNT of 4300. In a separate study, the RCOG risk scoring tool applied retrospectively to a birthing population of 6000 women from one center yielded a NNT of 4800 for women who qualified as “high-risk”.

When the NNT is high, the chances are greater that harm outweighs benefit. LMWH is a potent anticoagulant that increases the risk of bleeding and wound complications compared with placebo. In general surgical patients, compared with placebo, randomized trials demonstrated an increase in serious hemorrhage of 1.5% (number needed to harm (NNH) = 67) and need for transfusion of 3.8% (NNH = 26).

Since there are very few trials comparing LMWH with placebo in pregnant and postpartum women, the excess risk of bleeding from prophylactic LMWH is unknown. Birthing women have a high physiological risk of hemorrhage, which might put them at increased risk compared with general surgical patients. However, they are young, healthy, and generally hypercoagulable, which might lower the risk. In women who received therapeutic doses of LMWH, the increase in hemorrhage >1000 ml was approximately 4% (NNH = 25). If the risk from lower prophylactic doses of LMWH after CS is only 0.5%, the NNH would be 200. Since the NNT to prevent one VTE is 4000, this means that 20 women would have serious bleeding for every woman who avoided VTE.

LMWH also increases wound complications. In a large observational study of 1600 women with a BMI >30 and/or age >35, LMWH increased the risk of significant wound separation after CS by approximately 2% (NNH=50). About half of these women required re-admission. Again the NNH is much lower than the NNT, meaning far more women will experience wound disruption than will avoid VTE.

It has been suggested that wound complications and harm from bleeding are less important than avoiding a potentially fatal pulmonary embolus; however, fewer than 1% of all VTE episodes are fatal PE. Assuming 70% protection with LMWH, the NNT to prevent one PE death is about 400,000. If LMWH increases the risk of major hemorrhage by only 0.25%
(1/400), then for every PE death avoided with LMWH, 1000 women will experience a major hemorrhage. Obstetrical bleeding can be serious, and some of these may be fatal.

It has also been suggested that widespread postpartum LMWH prophylaxis has reduced maternal deaths from pulmonary embolism in the UK. However recent data does not support this claim. Before the widespread use of heparin, UK data demonstrated a five-fold decrease in triennial obstetrical pulmonary embolism deaths from 168 (1955–57) to 32 (1985–87), likely caused by the abandonment of postpartum bed rest. Since 1985, the number of triennial deaths from PE has oscillated – up and down – between 18 and 48 (table 5). This three-fold fluctuation is within normal statistical variance and similar that for deaths from amniotic fluid embolism, sepsis, and hemorrhage. Nonetheless, improved LMWH prophylaxis for the small number of women with antiphospholipid antibody syndrome, potent thrombophilias, or a personal history of VTE has very likely improved safety for women with these potent risk factors.

Table 5: Triennial UK Maternal Deaths (MBRACE 2016)

<table>
<thead>
<tr>
<th></th>
<th>1955-1957</th>
<th>85-87</th>
<th>88-90</th>
<th>91-93</th>
<th>94-96</th>
<th>97-99</th>
<th>00-02</th>
<th>03-05</th>
<th>07-08</th>
<th>09-11</th>
<th>12-15</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sepsis</td>
<td>9</td>
<td>17</td>
<td>15</td>
<td>16</td>
<td>18</td>
<td>13</td>
<td>18</td>
<td>26</td>
<td>16</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>Amniotic Fluid</td>
<td>9</td>
<td>11</td>
<td>10</td>
<td>17</td>
<td>8</td>
<td>5</td>
<td>17</td>
<td>13</td>
<td>7</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>Embolism</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bleeding</td>
<td>10</td>
<td>22</td>
<td>15</td>
<td>12</td>
<td>7</td>
<td>17</td>
<td>14</td>
<td>9</td>
<td>14</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>Venous Thrombo-</td>
<td>168</td>
<td>32</td>
<td>33</td>
<td>35</td>
<td>48</td>
<td>35</td>
<td>30</td>
<td>41</td>
<td>18</td>
<td>30</td>
<td>20</td>
</tr>
<tr>
<td>embolism</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Pregnant and postpartum women with rare clinical conditions that have a strong association with VTE should be considered individually for LMWH prophylaxis. This includes:

- Medical comorbidities: (e.g., cancer, heart failure, active systemic lupus, inflammatory bowel disease or polyarthritis, nephrotic syndrome, type I diabetes with nephropathy, sickle cell disease.)
- Intensive care admission
- Orthopedic surgery in pregnancy
- Bedrest for three days or longer

Despite a paucity of evidence, clinical judgement is also warranted for women with uncommon combinations of multiple, marked risk factors such as:

- Class III obesity (BMI > 40) and CS in labour.
- Family history of unprovoked or estrogen-related VTE
- Puerperal sepsis after CS
- Low-risk thrombophilia + family history of VTE
- Stillbirth with pre-eclampsia
Involvement of a hematologist may help guide decisions in complex cases, especially those involving thrombophilias or a family history of provoked or unprovoked VTE. Potent thrombophilias for which LMWH is currently recommended include:

- Homozygous Factor V Leiden
- Homozygous Prothrombin Gene mutation
- Heterozygous for both Factor V Leiden & Prothrombin Gene mutation
- Antithrombin III deficiency
- Protein S deficiency
- Protein C deficiency

LMWH is typically given once daily subcutaneously. Table 6 lists recommended prophylactic doses of LMWH based on bodyweight.

Table 6: Prophylactic daily dose of LMWH

<table>
<thead>
<tr>
<th>WEIGHT</th>
<th>ENOXAPARIN</th>
<th>DALTEPARIN</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 50 kg</td>
<td>20 mg</td>
<td>2500 units</td>
</tr>
<tr>
<td>50 to 90 kg</td>
<td>40 mg</td>
<td>5000 units</td>
</tr>
<tr>
<td>91 to 130 kg</td>
<td>60 mg</td>
<td>7500 units</td>
</tr>
<tr>
<td>131 to 170 kg</td>
<td>80 mg</td>
<td>10,000 units</td>
</tr>
<tr>
<td>&gt;170 kg</td>
<td>0.6 mg/kg</td>
<td>75 u/kg</td>
</tr>
</tbody>
</table>

Both LMWH and unfractionated heparin can increase the risk of neuraxial hematoma during epidural or spinal anaesthesia. Consultation with an anaesthetist is recommended regarding necessary time delays between heparin dosing and placement or removal of an epidural catheter or dural puncture for spinal anaesthesia.

Unfractionated heparin is uncommonly used as it has an increased risk of heparin induced thrombocytopenia (HIT) and osteoporosis compared with LMWH. However, women approaching delivery or at high risk of bleeding may be switched to unfractionated heparin because it is shorter acting and reversible with protamine. Prophylactic unfractionated Heparin is given subcutaneously every 12 hours.

Consent

Close scrutiny of the best available evidence shows that the incidence of VTE in women with common risk factors listed in current obstetrical VTE guidelines is very low. The estimated benefit and harm from LMWH is not quantified in guidelines. Without access to this information, it is not possible for women to consent to treatment. Giving therapy without informed consent is not generally considered acceptable in modern medical practice.

The absolute risk of VTE in women with most common risk factors appears to be too low to warrant LMWH. This includes BMI >30, age >35, smoking, parity > 3, varicose veins, pre-eclampsia, assisted vaginal birth, CS, postpartum...
hemorrhage, diabetes, preterm birth, multiple gestation, and prolonged labour. Before LMWH can be recommended for these women, randomized controlled trials of LMWH versus placebo are needed. Benefit and harm must be measured and stratified by risk factors and this information made available to clinicians and birthing women. Given that the magnitude of benefit in most women with risk factors is very small, the study sample size will need to be very large, and the logistics will be daunting. However, when benefit of therapy is small, the chances are higher that harm outweighs benefit, making it even more important to do the trials before implementing therapy.

Diagnosis

Diagnosis of DVT

The symptoms suggestive of DVT are non-specific and common in pregnancy. Approximately 50% of women thought to have DVT on clinical examination have a negative ultrasound or venography. However, a high index of suspicion must be maintained, and investigations and consultation with colleagues used liberally.

A missed diagnosis could be fatal!

DVT in pregnancy occurs in the iliofemoral or calf veins of the left leg 90% of the time.

Signs and symptoms

- Leg pain and tenderness
- Swelling
- Warmth
- Positive Homan’s sign
- Left leg involvement (83% of DVTs)
- An isolated iliac vein thrombosis may present with vague lower abdominal or pelvic pain with diffuse pain and swelling of the entire lower limb on the affected side.

Diagnostic Tests

Diagnostic tests may be non-invasive or invasive. If therapy will be altered by the performance of an invasive diagnostic test, then the benefit outweighs the risk to the mother and fetus.

1. Duplex Doppler ultrasound (compression ultrasonography) 88-92

This is the initial test for suspected DVT in most centres. It
- is non-invasive
- has 95% correlation with venography in the non-pregnant population for popliteal and femoral veins (90% sensitive and 99% specific for proximal deep vein thrombosis; 73% sensitivity for distal DVT)
Venous Thromboembolism and Amniotic Fluid Embolus

- is less sensitive for calf veins and pelvic vein thrombosis
- should be performed serially if suspicion of DVT persists despite an initial negative result

A positive result warrants treatment with anticoagulation. A negative result does not rule out a pulmonary embolus.

2. Impedance plethysmography

- Is no longer commonly used
- Measures changes in electrical impedance as the result of changes in blood volume within the limb
- Can rule out DVT when performed serially (at least 3 tests over 7 to 14 days)

The sensitivity and specificity of this test are compromised in late pregnancy because of physiologic compression of iliac veins and the resulting decreased venous return.

Both duplex Doppler and impedance plethysmography have poor sensitivity in detecting a DVT in the calf veins. The tests should be repeated if the first scan has a negative result but there is a high clinical index of suspicion. It is safe to withhold treatment if serial studies are negative.

3. Venography

This is the gold standard for diagnosis of DVT.

- It is invasive and exposes the woman and fetus to radiation. The fetus is exposed to 0.05 rads (50 mrad) when lead apron shielding is used
- It has a negative predictive value of 98% (if venography is negative, there will be no DVT 98% of the time)

Consider venography if duplex Doppler is unavailable or if the results are equivocal.

4. D-dimer

D-dimer is a fibrin degradation product that is present in the circulation when a thrombus (clot) is present. Measuring D-dimer levels to exclude the presence of thrombosis is standard practice in non-pregnant patients. However, D-dimer levels are elevated in normal pregnancy. Pregnancy-specific D-Dimer levels have not yet been established reliably enough to be clinically useful.

5. Radioactive labelled fibrinogen scanning

This is contraindicated in pregnancy.

NOTE: Confirmation of thrombosis by either a non-invasive study or venography justifies treatment with anticoagulation.
Pelvic/Ovarian Vein Thrombosis

This rare but serious complication usually occurs within one week after delivery. It can present with fever, chills, nausea, vomiting, leukocytosis, and adnexal or abdominal pain. The right ovarian vein is most commonly affected. When the large pelvic veins (e.g., iliac veins) are involved in a thrombophlebitis process, the thrombus may extend into the inferior vena cava.

Pelvic/ovarian vein thrombosis may be diagnosed with the use of ultrasound or duplex Doppler. Magnetic resonance imaging or CT should be considered if clinical suspicion is high.

Diagnosis of Pulmonary Embolism

Signs and symptoms

The signs and symptoms of a PE are non-specific and have a poor diagnostic reliability. However, they should raise clinical suspicion and prompt appropriate diagnostic testing and consultation.

The most common symptoms of pulmonary embolism and their prevalence in pregnancy-associated PE are:

- Tachypnea 89%
- Dyspnea 81%
- Pleuritic pain 72%
- Apprehension 60%
- Cough 54%
- Tachycardia 43%
- Hemoptysis 34%
- Temperature >37.5°C (99.5°F) 35%

1. Diagnostic Tests

Early diagnostic testing is warranted whenever a diagnosis of PE is suspected. Pulmonary angiography is the gold standard for the diagnosis of PE but is invasive and carries higher morbidity. Other tests accompanied by thorough clinical evaluation are, in most cases, used first. These include the following:

a) Chest X-ray and electrocardiogram

- The chest X-ray is rarely, if ever, diagnostic. It is used in conjunction with a ventilation/perfusion (V/Q) scan to rule out other causes of hypoxemia.
- Results of electrocardiogram are often normal or show non-specific changes.
b) **Arterial blood gases**

- Measurement of arterial blood gases in women with suspected PE is not diagnostic because arterial blood gas measurements lack specificity and are only moderately sensitive for PE. Hypoxemia and hypocarbia occur in conditions that simulate PE, and arterial oxygen tensions can be normal in patients with a minor PE.  
- Significant hypoxemia excludes hyperventilation as the cause of the patient’s symptoms, although this condition is rare.

c) **Ventilation / perfusion scan**

This is the least invasive test, and it is therefore often used as the primary investigation.

- Minimal radiation to the fetus
- An inhaled radioactive agent (ventilation) combined with an intravenous injection of a radioactive agent (perfusion) is administered. The combination of ventilation and perfusion assessments increases the accuracy of the test over either used alone, especially in cases where other conditions such as pneumonia are present.
- V/Q scans are reported as high, intermediate, or low-probability scans
- In 1990 the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) study in non-pregnant patients reported that:
  - Virtually all patients with a pulmonary embolus had an abnormal (high, intermediate or low-probability) V/Q scan, BUT
  - Most patients without a pulmonary embolus also had an abnormal (high, intermediate or low-probability) V/Q scan (sensitivity 98%; specificity 10%)
  - 88% of patients with high-probability V/Q scans had a pulmonary embolism on pulmonary angiography, BUT
  - Only a minority of patients with a pulmonary embolus have a high-probability V/Q scan (sensitivity 41%; specificity 97%)
- A low-probability V/Q scan together with a low clinical suspicion for a pulmonary embolus essentially rules out a diagnosis of pulmonary embolism
- A high-probability V/Q scan combined with a high clinical index of suspicion is diagnostic of a pulmonary embolism
- An intermediate-probability V/Q scan is not helpful in establishing or excluding a diagnosis of pulmonary embolism. In such circumstances further investigation is necessary. The following should be considered:
  - Duplex Doppler ultrasound (compression ultrasonography) to look for a DVT
  - Serial impedance plethysmography
  - Pulmonary angiography: if high clinical suspicion is present and Duplex Doppler ultrasound and impedance plethysmography are normal
  - The combination of a chest X-ray, a V/Q scan and pulmonary angiography exposes the fetus to less than 0.5 rad radiation (not associated with a significant risk of fetal injury in most studies)
In summary, the ventilation-perfusion scan can direct clinical decision-making when the results are normal or indicate a high-probability of a pulmonary embolus. In other circumstances, spiral CT, pulmonary angiography, or CT pulmonary angiography is indicated to rule out a PE.\textsuperscript{22, 98}

**d) Spiral computerized tomography**

This is a non-invasive test that exposes the fetus to minimal radiation. It is accurate to at least the segmental, and possibly the sub-segmental, branches of the pulmonary artery.

The single detector spiral CT has an 86% sensitivity and 93% specificity for pulmonary embolus in non-pregnant patients.\textsuperscript{99} There is a risk in extrapolating the data from studies in men and non-pregnant women.\textsuperscript{100}

It has a negative predictive value of 98%.\textsuperscript{20}

The newer four-channel multi-detector spiral CT is faster and provides higher resolution, allowing for detection of smaller and more distal emboli.

In some centres, the multi-detector spiral CT has replaced the V/Q scan as the first-line investigation for pregnant women.\textsuperscript{20} However, it is important to understand that the hemodynamic changes in pregnancy offer a challenge to the diagnosis of PE using even the latest CT technology. It has been recommended that in the presence of an abnormal chest X-ray, CT be offered to diagnose a PE. A V/Q scan should be offered in the presence of a normal chest X-ray.\textsuperscript{100}

**e) Magnetic resonance angiography\textsuperscript{20}**

This is a fast, high-resolution test that involves no exposure to radiation.

Its sensitivity for isolated subsegmental, segmental, and lobar pulmonary emboli is 40%, 84%, and 100% respectively. However, it has not been widely used in pregnant women.

**NOTE:**

- Pregnant women with positive non-invasive investigations should be treated for pulmonary embolism
- Symptomatic pregnant women with negative non-invasive studies should undergo pulmonary angiography since non-invasive tests may be negative in 57% of non-pregnant patients with pulmonary embolism demonstrated on angiography and V/Q scans of low or intermediate probability
- Non-invasive tests often miss emboli that originate in the pelvic veins during pregnancy\textsuperscript{101, 102}

**f) Pulmonary angiography**

This highly specific and accurate test is the gold standard for pulmonary embolism. It is, however, invasive and requires right heart cardiac catheterization and contrast injection. It exposes the fetus to 0.25 rads. Side effects include patient discomfort, allergic reaction to the injected contrast material, and renal dysfunction or failure.

Serious morbidity (1%) and minor morbidity in 5%\textsuperscript{103}; 1 in 200 (0.5%) mortality.\textsuperscript{103}
NOTE: There are significant implications to a diagnosis of deep vein thrombosis and/or pulmonary embolism. They include:

- the need for long-term use of anticoagulation with its potential fetal and maternal risks
- the potential need for prophylactic anticoagulation in future pregnancies
- the impact on the woman's future use of oral contraceptives and estrogen replacement therapy

Because of the seriousness of the diagnosis and the significance of its future implications, any woman suspected of having venous thromboembolic disease must be aggressively investigated with definitive studies to confirm or rule out the diagnosis prior to treatment.

Management of DVT and Pulmonary Embolism in Pregnancy

1. Available Anticoagulants

The anticoagulants currently available for use during pregnancy include unfractionated heparin, heparin-like substances, warfarin derivatives, and ASA.

a) Unfractionated heparin (UFH)

- Inhibits thrombin by activating antithrombin
- Prevents conversion of fibrinogen to fibrin
- Advantages:
  - Does not cross the placenta and is not teratogenic\textsuperscript{104}
  - Does not cause bleeding in the fetus
  - Not secreted in breast milk
  - Anticoagulation is rapidly reversible with protamine sulfate (1 mg per 100 units of heparin)
- Disadvantages:
  - Usually administered more than once daily
  - Half-life may be shorter in pregnant than in non-pregnant women, so higher dosing may be required
  - Maternal bleeding is the greatest risk, but the risk is very low on prophylactic heparin therapy
  - Heparin-induced thrombocytopenia is a life-threatening complication that may occur in up to 3% of non-pregnant patients\textsuperscript{105}
    - Heparin-induced thrombocytopenia usually occurs during the first 2 weeks of treatment
    - Close monitoring of the woman's platelet count during this interval must take place
    - Once a heparin-induced thrombocytopenia develops, low molecular weight heparin cannot be substituted for UFH.
It is recommended that if a woman is on UFH for more than four days that platelet counts be done before neuraxial procedures to rule out HIT\textsuperscript{4}.

Heparin-induced osteopenia is relatively common in patients given UFH for longer than 1 month. A reduction in bone density occurs in up to 30\% of non-pregnant women, and 2\% to 3\% will develop symptomatic vertebral fractures. Heparin-induced osteopenia may not be rapidly reversible because heparin is sequestered in bone for an extended time.\textsuperscript{104} Vitamin D and calcium supplements are recommended for pregnant women who are on thromboprophylactic therapy. Yet research is showing that at prophylactic dosing during pregnancy, bone loss may not be significant.\textsuperscript{6}

b) Low molecular weight heparin (LMWH) and heparin-like substances\textsuperscript{67, 106, 107}

- **Advantages:**
  - Usually administered once daily depending on maternal weight
  - Can convert to subcutaneous therapy faster following initial intravenous treatment with unfractionated heparin
  - Longer half-life, better bioavailability, and a more predictable dose response than UFH\textsuperscript{108}
  - Does not cross the placenta, and is not teratogenic\textsuperscript{104}
  - Does not cause bleeding in the fetus
  - Not secreted in breast milk
  - The risk of heparin-induced thrombocytopenia exists but is much lower (<1\%) than for UFH\textsuperscript{4, 105} It is recommended that routine monitoring of platelet count is unnecessary.

- The risk of heparin-induced osteopenia is also lower with LMWH than UFH, and research suggests that at prophylactic dosing during pregnancy, bone loss may not be significant.\textsuperscript{6}

- Maternal weight-based protocols, using weights at 10 to 16 weeks' gestation, are reliable, at least for women at intermediate risk for VTE during Caesarean section.\textsuperscript{109}

- **Disadvantages:**
  - More expensive than UFH
  - Cannot reverse anticoagulation effect with protamine sulfate
  - The longer half-life of LMWH raises issues of timing of an epidural
  - Half-life may be shorter in pregnant than in non-pregnant women, so higher dosing may be required\textsuperscript{6}
  - Maternal bleeding is the greatest risk, but the risk is very low with prophylactic LMWH therapy compared with therapeutic LMWH
  - Not yet demonstrated to be superior to unfractionated heparin for the treatment of DVT or PE in pregnancy.\textsuperscript{110}
  - Weight-based dosing appears inappropriate in the treatment of acute VTE in pregnancy. While too early to generalize across the class, weight-based dosing failed to provide therapeutic anticoagulation with tinzaparin in a small 2013 study by Gibson et al.\textsuperscript{111}

- **Contraindications or cautions in use\textsuperscript{69}**
  - Known bleeding disorders
  - Active antepartum or postpartum bleeding
  - Women considered at risk for major hemorrhage (e.g., placenta previa)
− Thrombocytopenia (platelet count < 75x10^9/L)
− Acute stroke in previous 4 weeks
− Severe renal disease
− Severe liver disease
− Uncontrolled hypertension (systolic blood pressure > 200 mmHg or diastolic blood pressure > 120 mmHg)

**NOTE:** Low molecular weight heparins should **not** be used for anticoagulation in pregnant women with prosthetic heart valves because of the increased risk of valvular thrombosis (even in the face of apparent adequate anticoagulation). 6,112,113

**NOTE:** The multi-dose vials of low molecular weight heparins contain benzyl alcohol as a preservative. Benzyl alcohol has been associated with the uncommon but potentially fatal “gasing syndrome” in neonates. Because benzyl alcohol may cross the placenta, LMWH preparations **preserved with benzyl alcohol** should **not** be used in pregnant women. 114

c) Warfarin

− Inhibits vitamin K
− Blocks synthesis of coagulation factors II, VII, IX, and X
− **Contraindicated during pregnancy** because of potential fetal effects, **BUT may still be considered** in pregnancy for women with mechanical heart valves: advice should be sought from the appropriate specialist.
− May be used for postpartum management
− Advantages:
  − Given orally
  − Safe during breastfeeding
  − Low cost
− Disadvantages:
  − Crosses the placenta (fetal anticoagulation)
  − Risk of fetal and neonatal hemorrhage particularly at the time of birth
  − Risk of teratogenesis (the exact level of risk is not known)
  − Congenital warfarin embryopathy (exposure between 6 and 12 weeks’ gestation) including nasal hypoplasia and/or stippled epiphyses (chondrodysplasia punctata) 30
  − May cause intrauterine growth restriction
  − Increase in CNS anomalies if given in the second or third trimester
  − Requires regular monitoring of international normalized ratio (INR) to achieve appropriate therapeutic levels

**For ongoing postpartum therapeutic anticoagulation with warfarin**

− Postpartum therapy may be started on the day following delivery
− Administer 7.5 mg/day on postpartum day 1 and 2
− Overlap with intravenous heparin and adjust the dose until the INR is 2 to 3 times the control value for 2 consecutive days
− Monitor the level of anticoagulation by measuring the INR regularly once the woman is on a maintenance dose
d) Acetylsalicylic acid

- Inhibits platelet aggregation
- Advantages:
  - Inexpensive
  - Administered orally
  - Low dose ASA (<150 mg/day) administered in the second and third trimester has been found to be safe for mother and fetus, but the safety of higher doses has not been determined
- Disadvantages:
  - Crosses the placenta
  - Safety in the first trimester is unknown
  - Potential for birth defects
  - Maternal and neonatal bleeding
  - Not proven effective in the prevention or management of VTE in pregnancy

2. Non-Medical Management

The woman should

- Have her legs raised as much as possible to reduce edema
- Not wear compression stockings (no controlled trials using compression stockings during pregnancy or postpartum are available). Stockings increase the risk of post-thrombotic leg syndrome.
- Avoid prolonged sitting

3. Anticoagulation during Pregnancy

Management is constantly evolving, and there are wide regional differences. There is limited evidence favouring one treatment over another. It is important to consult with local experts in hematology for guidance. Evidence supports the following:

a) Initial treatment of a DVT with unfractionated heparin (UFH)

- Obtain baseline CBC, including platelets and activated partial thromboplastin time (aPTT).
- Administer an initial bolus of 5000 units IV
- Measure the aPTT 6 hours following the bolus
- Infuse at a rate of 30 000 units/24 hours
- Repeat the serum aPTT every 6 hours until therapeutic heparin levels are reached.
- Adjust the dosage to keep the aPTT at 1.5 to 2 times the control value for the relevant laboratory
- Repeat the aPTT or serum heparin level daily once stabilized in the therapeutic range. If the serum heparin levels are being measured, serum heparin should be maintained at 0.2 to 0.4 IU/mL (an aPTT of 60 to 85 seconds)
- Continue IV anticoagulation for 5 to 7 days
• Switch to subcutaneous unfractionated heparin or low molecular weight heparin
  - **Unfractionated heparin:**
    › Multiply the total 24-hour dose being administered by 1.5 and divide into 2 doses per day
    › This is usually approximately 12 500 units subcutaneously twice daily
    › Maintain the aPTT at 1.5 to 2.5 times the control
  - **Low molecular weight heparin** (e.g., a 50 to 70 kg woman):
    › Tinzaparin 175 IU/kg subcutaneously once daily
    › Dalteparin 200 IU/kg subcutaneously once daily
    › The dose is chosen to achieve an anti-Xa heparin level of 0.3 to 0.75 4 hours post-injection
    › No regular monitoring is necessary, but a single anti-Xa level taken in the third trimester will ensure that the therapeutic range has been achieved, some experts suggest that regular monitoring in high risk women may be beneficial.
    › Twice per day administration of LMWH during the third trimester appears to result in better therapeutic levels

**b) Longer-term treatment**

• Unfractionated heparin or low molecular weight heparin should be used during pregnancy and can also be given postpartum
• Warfarin can be used postpartum. Treatment is usually initiated with a combination of intravenous UFH and warfarin. The unfractionated heparin is discontinued after approximately 5 days. If the woman is already being treated with UFH or LMWH, add the warfarin for four to five days and then discontinue the heparin.

---

**4. Caring for Women During the Acute Phase of Anticoagulation**

The 2017 SOAP guidelines recommend that women on anticoagulants be fully informed and have an advanced plan for antepartum and intrapartum management. They add that a comprehensive interprofessional approach be followed. Women on anticoagulants must be readily identified and the date and time of their last dose be known. Furthermore, the team (which includes the woman and her family) needs to know if dosing can be held during labour and delivery, or if conversion from LMWH to UFH can occur in advance of labour. Care providers should

• Direct women to rest until swelling is reduced and anticoagulation has reached therapeutic levels
• Monitor coagulation laboratory values
• Carefully assess for unusual bleeding. Heavy vaginal bleeding, generalized petechiae, bleeding from the mucous membranes, hematuria, or oozing from venipuncture sites should alert the care provider to investigate further
• Ensure the presence of protamine sulfate in close proximity to the point-of-care
• Assess for muscle pain, tenderness, swelling, positive Homan's sign, and dilated superficial veins

The 2017 SOAP guideline is the only guideline currently available specifically addressing pregnant women.

Their guideline includes excellent decision tools for urgent and emergent neuraxial procedures in the OB patient receiving both UFH and LMWH. Those recommendations include very clear guidance on when to insert a spinal or epidural and when to restart anticoagulants upon removal of epidurals.

---

*Venous Thromboembolism and Amniotic Fluid Embolus*
Summary of recommended doses for prophylactic, therapeutic, and postpartum anticoagulation

<table>
<thead>
<tr>
<th>MEDICATION</th>
<th>PROPHYLACTIC DOSE</th>
<th>THERAPEUTIC DOSE</th>
<th>POSTPARTUM VAGINAL DELIVERY DOSE</th>
<th>POST-CS DOSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unfractionated heparin (UFH)</td>
<td>• 5000 IU twice daily OR • 5000 IU twice daily (first trimester) • 7500 IU twice daily (second trimester) • 10 000 IU twice daily (third trimester)</td>
<td>5000 IU bolus and ~30 000 IU daily (aPTT at 1.5 to 2.5 control)</td>
<td>Restart the UFH 4 to 6 hrs post-delivery or removal of epidural catheter (assuming bleeding is in control and a straightforward epidural placement procedure-defer to anaesthetist for opinion) at therapeutic or prophylactic dose depending on the situation. Safe in breast-feeding.</td>
<td>Restart UFH 4 to 6 hrs after CS or removal of epidural catheter or spinal (assuming bleeding is in control and straightforward regional anesthesia procedure-defer to anaesthetist for opinion) at prophylactic or therapeutic dose, depending on the situation. Safe in breast-feeding.</td>
</tr>
<tr>
<td>Dalteparin</td>
<td>• 2500 IU if &lt; 50 kg • 5000 IU if 50 to 90 kg • 7500 IU if 91 to 130 kg • 10 000 IU if 131 to 170 kg • 75 U/kg/day if &gt;170 kg once daily</td>
<td>200 IU/kg once daily</td>
<td>Restart LMWH 4 to 6 hrs after delivery or removal of epidural catheter (assuming bleeding is in control and epidural procedure was straightforward - defer to anaesthetist opinion) at therapeutic or prophylactic dose depending on the situation. Limited experience in lactating women. Safe in breast-feeding.</td>
<td>Restart LMWH 4 to 6 hrs after delivery or removal of the epidural catheter or spinal anaesthesia (assuming bleeding is in control and regional anaesthesia procedure was straight-forward-defer to anaesthetist opinion) at therapeutic or prophylactic dose depending on the situation. Safe in breast-feeding.</td>
</tr>
<tr>
<td>Tinzaparin</td>
<td>• 3500 units daily if &lt; 50 kg • 4500 IU daily if 51 to 90 kg • 7000 IU daily if 91 to 130 kg • 9000 IU daily if 131 to 170 kg • 75 U/kg/day if &gt;170 kg</td>
<td>Weight-based dosing is not appropriate in achieving therapeutic anticoagulation</td>
<td>As above</td>
<td>As above</td>
</tr>
<tr>
<td>Enoxaparin</td>
<td>• 20 mg daily if &lt; 50kg • 40 mg daily if 51 to 90 kg • 60 mg daily if 91 to 130 kg • 80 mg daily if 131 to 170 kg • 0.6 mg/kg/day if &gt; 170 kg</td>
<td>1.0 mg/kg twice daily or 1.5 mg/kg once daily</td>
<td>As above</td>
<td>As above</td>
</tr>
<tr>
<td>Warfarin</td>
<td>Contraindicated (except with mechanical heart valves)</td>
<td>Contraindicated (except with mechanical heart valves)</td>
<td>7.5 mg daily and adjust to keep INR at 2 to −3</td>
<td>No clear guidelines. Keep on UFH</td>
</tr>
</tbody>
</table>
Management of Labour and Delivery on PROPHYLACTIC Heparin

The woman must be fully informed about the possible complications of anticoagulation. She must be instructed to discontinue unfractionated heparin

- At the onset of regular uterine contractions
- 6 to 8 hours before the start of induction
- 6 to 8 hours before a planned Caesarean section

Low molecular weight heparin

Change to unfractionated heparin at 36 to 37 weeks' gestation

- For preterm delivery
  - Discontinue at the onset of regular uterine contractions
  - 24 hours before a planned induction
  - 24 hours before elective Caesarean section.

Management of Labour and Delivery on THERAPEUTIC Heparin

1. Obtain an aPTT on admission.
2. Switch from low molecular weight heparin to intravenous unfractionated heparin and adjust the dose to achieve a therapeutic level of aPTT, if not already done. It is common make the switch between 36 and 37 weeks.
3. **Protamine sulfate, 1mg per 100 units of unfractionated heparin** may be used to reverse an aPTT that is above the therapeutic level. Do not give > 50 mg protamine sulfate over 15 minutes IV. The 2017 SOAP guideline indicates that its use in pregnancy has not been studied. They are careful to indicate that a single case study after 25mg IV before delivery was associated with a severe respiratory depression in the infant.\(^5\)
4. Minimize trauma at the time of delivery:
  - Midline episiotomy, if an episiotomy is required
  - Avoid tears
  - Ensure good hemostasis at Caesarean section
  - There is a slight increase in hematoma formation in vaginal tears or episiotomies but no increase in postpartum hemorrhage
5. Regional anaesthesia is contraindicated in women on therapeutic heparin.
6. Continue anticoagulation for three to 6 months after the thromboembolic event.

The Use of Regional Anaesthesia in Patients Receiving Anticoagulation

Intraoperative or postoperative anticoagulation after regional anaesthesia is thought to be safe. Ideally, LMWH should not be given until 4 hours after removal of an epidural catheter.
The Society of Obstetric Anesthesia and Perinatology (SOAP) have produced the only guidelines addressing the special needs of the obstetric patient. Their guidelines are required reading in this field. ([https://www.ncbi.nlm.nih.gov/pubmed/29099429](https://www.ncbi.nlm.nih.gov/pubmed/29099429))

The safety of using anticoagulants before regional anaesthesia is unclear. The Food and Drug Administration reported cases of epidural or spinal hematomas with regional anesthesia in non-pregnant patients receiving LMWH. The American Society of Regional Anesthesia has recommended that patients receiving higher doses of LMWH should not receive regional anaesthesia for 24 hours from the last dose. In patients receiving low-dose LMWH, needle placement for regional anesthesia may occur 12 hours after the last dose of prophylactic LMWH or 24 hours after the last therapeutic dose of LMWH.^

### Postpartum Management

- Heparin can be resumed 4 to 12 hours postpartum, if required. The clinical picture, presence of risk factors, and the mode of delivery will influence management.
- If ongoing anticoagulation is required, oral warfarin or subcutaneous heparin can be prescribed. Both are safe for breastfeeding.
- It is important to note that the available evidence reassures us that therapeutic doses of LMWH in pregnancy does NOT increase the likelihood of a PPH or of a severe PPH.  

**NOTE:** Anticoagulation in women experiencing a VTE in pregnancy should continue throughout pregnancy and for at least 6 weeks postpartum or for a total of 3 months of anticoagulation.  

### Summary

1. Careful screening for risk factors for VTE in the antepartum period is critical.
2. The management of venous thromboembolism in pregnancy is ideally done in conjunction with a hematology service.
3. Consultation with a perinatal centre is recommended.
4. There are local variations in management regimens, especially with respect to thromboprophylaxis in a woman with a known thrombophilia or a family history of thrombophilia, with or without a previously documented DVT or PE.
5. Women experiencing a DVT or PE in pregnancy or the puerperium require investigation for an underlying thrombophilia once treatment is complete.
6. Functional tests of coagulation will be affected by the pregnant state and in the immediate postpartum period.
7. Women with a history of VTE, potent thrombophilia or antiphospholipid antibody syndrome should be recommended thromboprophylaxis with LMWH.
8. Randomized trials are needed to evaluate the magnitudes of risk and benefit from LMWH prophylaxis in women with most other risk factors for VTE.
Amniotic Fluid Embolism

Amniotic fluid embolism (AFE) is a rare, but often catastrophic, complication of pregnancy. The estimated incidence is between 1:15,200 and 1:53,800 births. The maternal mortality rate is between 11% and 44%. The perinatal mortality rate is between 9% and 44%. In addition, increased risks of stillbirth, hysterectomy, and prolonged length of hospital stay have also been observed.

- **Risk factors associated with AFE**
  - Induction of labour
  - Multiple pregnancy
  - Advanced maternal age
  - Operative delivery (vaginal and Caesarean section)
  - Eclampsia
  - Polyhydramnios
  - Cervical laceration
  - Tetanic uterine contractions
  - Tumultuous labour
  - Uterine rupture

- **Pathophysiology**

  The basic mechanism of the condition is related to the effects of amniotic fluid on the respiratory, cardiovascular, and coagulation systems. In normal labour, only 1 to 2 mL of amniotic fluid is transferred to the maternal circulation so that enhanced communication between amniotic fluid sac and the maternal venous circulation is necessary for AFE.

  Amniotic fluid with mucin, fetal debris, vernix, lanugo, fetal hair, and fetal squamous cells coated with white cells blood cells and granular debris is present in confirmed diagnoses. If meconium is present, the response is more dramatic.

  Once amniotic debris enters the venous system, it travels rapidly to the right heart and enters the cardiopulmonary circulation.

  The exact nature of the response is unknown but is probably immune mediated: direct myocardial depressant effect, vasosclerotic mediators (histamines, prostaglandins, serotonin etc.) leading to shock and an anaphylactoid state.

  **The hemodynamic response is biphasic**: initial pulmonary hypertension and right ventricular failure (within the first hour), followed by left ventricular failure.

  Effects of hypotension and hypoxemia lead to multiple organ failure: cardiovascular collapse, renal insufficiency, hepatic failure, seizures and coma.

  **Effects on hematological system**: potent thromboplastin and antifibrinolytic activity trigger clotting in the pulmonary vasculature and **result in a consumptive coagulopathy**.
Clinical Features

General—can occur at any point during labour and delivery. Clinical manifestations are variable, making diagnosis difficult:

- Sudden, unexplained peripartum respiratory distress, cardiovascular collapse, and coagulopathy
- Bleeding secondary to coagulopathy or uterine atony
- Possible presentations:
  - Respiratory distress, cyanosis
  - Hypotension
  - Seizures or seizure-like activity
  - Fetal bradycardia

Differential Diagnosis

- PE (patients experience more severe chest pain with PE than with AFE)
- CHF (fluid overload and/or pre-existing heart disease)
- Other: myocardial infarction, hypotension from sepsis, pulmonary aspiration, anaphylaxis, placental abruption, reaction to local anaesthetic

Management

Early diagnosis and treatment are the most critical factors associated with survival. The mortality rate is very high within the first hour. If mother and baby are to survive, rapid delivery of the baby is essential. For the mother, aggressive respiratory and circulatory support and interventions must be provided as required.

- Use a multidisciplinary approach
- Provide adequate oxygenation, avoid fluid overload
- Treat PPH from any or all of atony, tear, consumptive coagulopathy
- Give replacement of depleted hemostatic components as required for the development of consumptive coagulopathy

NOTE: A small 2013 study of case reports suggested that caution be used in the treatment of PPH in the presence of consumptive coagulopathy in patients with AFE. "[T]he use of rVIIa to treat postpartum hemorrhage in patients with severe consumptive coagulopathy and AFE was associated with worse outcome compared with cohorts who did not receive rVIIa". They went on to say "We recommend that the initial therapy of AFE-associated consumptive coagulopathy should consist of blood component replacement, including PRBC, FFP, platelets, cryoprecipitate, and possibly fibrinogen concentrate. We recommend that rVIIa be used in AFE patients only when the hemorrhage cannot be stopped by massive blood component replacement."
References


Venous Thromboembolism and Amniotic Fluid Embolus 525


