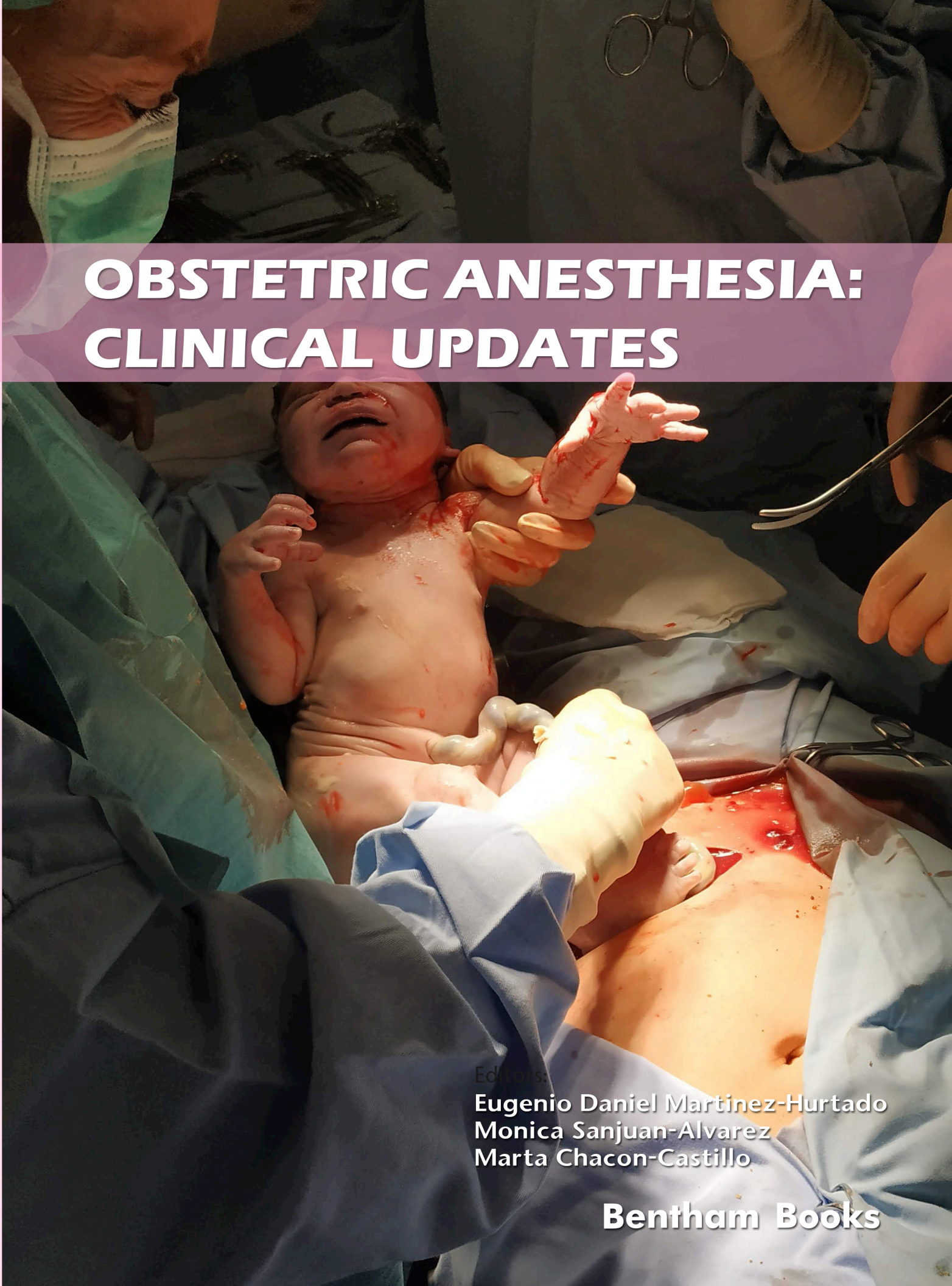

OBSTETRIC ANESTHESIA: CLINICAL UPDATES

Editors:
Eugenio Daniel Martinez-Hurtado
Monica Sanjuan-Alvarez
Marta Chacon-Castillo

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Recent Advances in Anesthesiology

Volume 4

Obstetric Anesthesia: Clinical Updates

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PREFACE

Obstetric anesthesia encompasses the anesthetic and analgesic procedures performed during all the stages of labor for vaginal delivery as well as cesarean section. Anesthesiologists have cared for the security of pregnant women since 1847 when James Young Simpson performed the first analgesia for labor. Since then, and even more in the last decades, obstetric anesthesia has become an important aspect of routine anesthetic practice, as women demand to be able to enjoy painless childbirth.

Obstetric anesthesia is a challenge for all of us, because it involves two patients in the same procedure, with different physiologic characteristics, and with different needs. For this reason, our primary target should be to promote a painless and safe delivery, without disturbing uterine contractions or fetoplacental circulation.

Obstetric anesthesia requires a comprehensive approach, not only limiting us to pain relief during labor, but also including the appropriate management of cesarean section and of any potential complications derived of both labor and surgery, while avoiding any fetal compromise.

The Editors of this handbook consider it essential that anesthesiologists understand the importance of obstetric anesthesia in our daily practice. In this handbook, we intend to compile all updated, didactic, and exhaustive information, and we deem every professional should know to face the act of anesthetizing a pregnant patient.

Finally, we would like to thank the co-authors' collaboration for the achievement of this handbook. As we are all professionals from different institutions, we are able to compare different scenarios and ways to conduct anesthesia, which enriches the text content. Lastly, we would like to acknowledge the readers of this handbook, who are responsible for modern, high-quality and safe anesthesia. These last words are dedicated to the new generation of anesthesiologists; we hope that with this handbook they acquire new knowledge and benefit from our experience.

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CHAPTER 1

Fetomaternal Physiology: Physiological Changes during Pregnancy

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Abstract: Anesthesia during pregnancy is challenging due to the extreme physiological and anatomical changes that occur. Deep knowledge of these changes and how they influence anesthesia is critical in order to offer safe anesthetic care to both, mother and the child. In this chapter, we will review the main features that occur in the respiratory, cardiovascular, central nervous, renal, and gastrointestinal systems, among others, and how it affects pharmacodynamics, pharmacokinetics, airway management and conduct of anesthesia. Fetomaternal circulation and fetal physiology focused on anesthesia will also be discussed.

Keywords: Anesthesia, Fetoplacental circulation, Fetal-maternal exchange, Placenta, Pregnancy.

INTRODUCTION

Changes during pregnancy are meant to serve a double objective: providing fetal well being, guaranteeing oxygen, nutrients supply, carbon dioxide and waste products removal, and preparing the maternal body for labor, delivery and lactation.

These changes can persist weeks after delivery and some may be long lasting [1, 2]. Maternal changes during pregnancy changes are summarized in Table **1**.

Respiratory System

Changes begin in the first trimester. Higher basal metabolism causes oxygen consumption to augment by about 60%; subsequently, a larger amount of carbon

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dioxide is produced. Greater minute ventilation is required to cope with this demand. Progesterone has stimulating effects on respiration, by raising sensitivity to carbon dioxide in the central nervous system (*CNS*). The net result is an increase of 50% in minute ventilation due, mostly, to higher tidal volume, causing physiological hypocapnia of around 30 mmHg. Renal compensation occurs so pH remains near 7.44. Despite this the hemoglobin dissociation curve shifts to the right due to an increase in 2,3-bisphosphoglycerate raising maternal P50 (partial pressure at which hemoglobin is 50% saturated), easing the offload of oxygen across the placenta [1 - 3]. Residual volume (*RV*) and residual functional capacity (*RFC*) are diminished (15% and 20%, respectively), the latter sometimes exceeded by closing capacity (*CC*). Owing to raised oxygen consumption and changes in RV and RFC, pregnant women desaturate more rapidly than their non-pregnant counterparts [3 - 5]. Inhalational anesthetics are up taken and eliminated more rapidly due to the greater minute ventilation and cardiac output [5, 6].

Table 1. Main physiological and anatomical changes during pregnancy.

Respiratory Changes	
Oxygen consumption	+ 60%
Minute ventilation	+ 50%
PCO_2	Decrease to 30 mmHg
Residual volume	- 15%
Functional residual capacity	- 20%
Airway capillaries	Engorgement
Diaphragm	Displaced cephalad
Ribs	Flaring
Cardiovascular	
Left ventricle mass	+ 50%
Cardiac output	+ 40-50%
Heart rate	+ 25%
Stroke volume	+ 25%
Systemic/Pulmonary vascular resistance	- 20%/-34%
Systolic blood pressure	- 6-8%
Diastolic blood pressure	- 20-25%
Hematological and Fluid Changes	
Red blood cells mass	+ 25%
Hemoglobin and hematocrit	-15%

(Table 1) cont.....

White blood cells during labor	Raise 9-11 x 10^9/L Up to 15 x10^9/L
Total plasma protein	- 18%
Plasmatic cholinesterase	- 20-25%
Colloid osmotic pressure	-18%
Plasma volume	+ 50%
Extravascular volume If edema	+ 1.7 L + 5 L
Platelets	=/-10%
Prothrombin time/Activated partial thromboplastin time	- 20%
Antithrombin III	- 10%
Protein S	Decrease
Protein C	No change
Fibrinolysis	Raise
I, VII, IX, VIII factors	+ 100- 150%
X, XII factors	+ 30%
II, V factors	=
XI, XIII factors	- 40-50%
Renal Changes	
Renal blood flow, Glomerular filtration rate	+ 50%
Na^+, H^2O and Cl^- reabsorption	+ 50%
Creatinine clearance	Raise
Serum creatinine	Decrease (~0.5-0.6 mg/dL)
Glucose, amino acids and uric acid reabsorption	Decrease
Urinary tract smooth muscle	Relaxation
HCO_3^- excretion	Raise
Gastrointestinal and Hepatobiliary Changes	
Esophagus	Displaced cephalad
Lower esophageal sphincter pressure	Decrease
Barrier pressure	- 45-50%
Alkaline phosphatase	Raise up to 4 times
Bilirubin, lactic deshydrogenase and transaminases	No change or raise
Cholecystokinin	Decrease
Neurological Changes	
Minimum alveolar concentration	- 40%
Pain threshold	Raise

CHAPTER 2

Safety in the Obstetric Patient: Simulation Training for Anesthesiologists in the Obstetrics Field

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Abstract: The principal goal of health systems is to provide safe and quality healthcare for the patient. Deficiencies in the environment in which obstetric care is provided, inadequate teamwork and communication, and poor individual performance during emergencies have been identified as preventable causes of harm to obstetric patients. There is growing evidence about training in Emergency Obstetric Care (*EmOC*) that reduces the risk of maternal and newborn mortality and morbidity. The Institute of Medicine identifies team-based training and simulation as methods to improve patients' safety, especially in the obstetrics field, these may add value to it. Recent research works review the effectiveness of training in EmOC and the use of simulation in improved health outcomes. It remains unclear whether this translates into improved patient outcomes.

Keywords: Communication, Competency-Based Medical Education, Emergency Obstetric Care, Maternal Mortality, Multidisciplinary Care, Nontechnical and Technical Skills, Obstetric Anaesthesia, Patient Safety, Simulation, Teamwork, Team Training.

INTRODUCTION

Safety and Quality in Obstetrics

The Institute of Medicine (*IOM*) recognizes patient safety as indistinguishable from the provision of quality healthcare. Safety and quality are closely intertwined. Safety methodologies try to avoid preventable adverse events (*pAEs*) while quality projects aspire to achieve the best possible results as health outcomes, patient satisfaction, access, and equity [1, 2].

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Patient safety and quality improvement in obstetrics is an important issue having three factors: obstetric admissions are one of the main reasons for hospitalization; secondly, family's expectations for a healthy and happy outcome, and lastly the increase in medical litigation claims and trials with costs associated [3]. At a global level, the goal is to achieve safe and quality care in obstetrics and labor settings with a reduction of maternal and neonatal morbidity and mortality.

Maternal survival has significantly enhanced since the incorporation of the United Nations Millennium Development Goal (*MDGs*), the maternal mortality ratio (*MMR*) has decreased in 43.9% of the countries from 1990 to 2015 [4]. However, MMR remains high in many parts of the world, especially in low-income and middle-income countries (*LMICs*). Nearly 50% of these direct maternal deaths are caused by hemorrhage and hypertensive disorders of pregnancy. In high-income countries (*HICs*), indirect maternal deaths (underlying cardiac and embolic diseases and associated conditions like obesity, multiple gestations, and assisted reproductive technologies) outnumber direct deaths. The United Nations Sustainable Development Goal (*SDG*) aspires to reduce the global MMR to less than 70 per 100.000 live births by the year 2030. Evidence suggests that the majority of these and other potentially life-threatening complications such as sepsis, complications from delivery, and unsafe abortion could be prevented by timely and effective emergency obstetric care [5, 6]. However, it has been shown that more than half of all women with obstetric complications lack access to this life-saving intervention [7].

Strategies to Improve Quality in Obstetrics

The minimum care package required during pregnancy and childbirth addresses the main causes of maternal death, stillbirth and early neonatal death referred to as emergency obstetric care (*EmOC*) [8]. The basic components include antibiotics, oxytocic drugs, anticonvulsants, manual removal of placenta, removal of retained products of conception, assisted vaginal delivery and resuscitation of the newborn baby using a bag and mask. It has been argued that a more comprehensive set of signal functions includes caesarean section, blood transfusion and care for small and sick newborns [9]. In many cases, the required infrastructure (as equipment and consumables) is available, but staff may lack the competency to provide all EmOC signal functions. EmOC relies on the presence of suitably trained and competent healthcare providers. Short competency-based training in EmOC results in significant improvements in healthcare provider knowledge/skills and change in clinical practice [9]. Regular training is recommended and, in some cases, mandatory, to ensure the continued accreditation of healthcare providers. In the early 1990s, EmOC training courses such as the Advanced Life Support in obstetrics (*ALSO*) and Managing Obstetric Emergencies and Trauma (*MOET*)

were developed to meet this need in high-income settings. However, in the era of the SDGs, competition for limited resources is high, and the cost-effectiveness of training packages is important to aid decision-makers in the most efficient use of resources and assess value-for-money. Very little is published about the costs and cost-effectiveness of training [10]. The wider health, social and economic benefits resulting from relatively small investments in training can be substantial, suggesting that these investments are likely to be of good value for money [11].

Guidelines and protocols endorsed by maternal safety organizations have been developed. They emphasize early and aggressive management of obstetric hemorrhage starting with risk factor identification, rapid diagnosis, timely management and multidisciplinary review. Systems to accelerate the initial response include an obstetric emergency response team, a postpartum hemorrhage cart ("*PPH cart*") and emergency hemorrhage medication packs.

Guidelines and protocols for acute management of hypertension focus on early diagnosis, prompt antihypertensive therapy, and seizure prophylaxis with magnesium sulfate [12].

How About Safety in the Obstetric Field?

The combination of gradually more complex systems controlled by "*imperfect*" humans is the basis of the patient safety problem in current medicine.

The World Health Organization (*WHO*) defines patient safety as the "*absence of preventable harm to a patient during the process of health care and reduction of risk of unnecessary harm associated with health care to an acceptable minimum*".

The Institute of Medicine observed that the root cause of 70% of errors in general and up to 80% of obstetric sentinel events can be traced to the process of team skills [13]. As many as 9% of pregnant patients will experience an adverse event during their delivery and up to 87% of adverse events in the obstetric population are deemed preventable [14]. In 2004, Joint Commission Sentinel Event Alert studied 47 perinatal deaths and identified non-medical factors topped the list of identified root causes, particularly communication and organizational culture, which contributed significantly to deficient perinatal outcomes [15]. This sentinel event alert was essential for clarifying obstetric safety threats and for risk reduction strategies that any unit starting a patient safety program should focus on.

Taking into account the five root causes of adverse perinatal events from the Joint Commission Sentinel Event Alert, possible safety interventions would focus on

CHAPTER 3

Airway Management in Pregnancy

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Abstract: Airway management in the obstetric patient is a challenge for anaesthesiologists, not only because of the anatomical and physiological changes during pregnancy, but also because of the surgery´s urgency, the location of the procedure, which sometimes takes place even outside the operation theatre, and also due to conflicts emerging between the needs of the mother and fetus. The arising maternal comorbidities such as obesity, contribute to complications in airway management in this population.

Keywords: Airway Management, Apnoeic Times, Cricothyroid Membrane, Difficult Airway, Difficult Intubation, Laryngoscopy, Maternal Death, Neuraxial Anesthetic Blocks, Predictors, Pregnant Patient, Pregnancy, Preeclampsia, Ultrasonography, Video Laryngoscopy.

INTRODUCTION

The rate of failed tracheal intubation in obstetrics has remained unchanged from 1970 to 2015 as demonstrated by reviewing the scientific literature. The incidence of failed tracheal intubation remains at 2.6 per 1,000 general anesthetics during obstetric procedures, and about 2.3 per 1,000 general anesthetics in case of cesarean section [1]. And, in case of tracheal intubation failure, it is usually

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preferable to maintain general anesthesia using a rescue device rather than awakening the patient.

Pregnancy implies that a potentially life-threatening event will occur within 9 months, in addition to which the management of pregnant women is different from that of other patients in several respects. The most frequent cause of morbidity and mortality related to obstetric anesthesia is failure or difficulties in airway control after the induction of general anesthesia. Furthermore, the causes of this morbi-mortality are different from those of other surgical patients. Therefore, regional anesthesia is being used more and more frequently [2 - 5].

Although induction of a rapid sequence of general anesthesia and tracheal intubation is a standard of management, regional anesthesia is used in most cesarean sections [6 - 8]. Instead, if there is a primary airway pathology, delaying or modifying the treatment should be considered, in view of the fetal well-being and the evolution of the pregnancy [9].

However, although anesthetic procedures may be very different, technical aspects of airway management in pregnant are similar to that of non-pregnant. Therefore, multidisciplinary teamwork is essential, and should begin in the early stages of pregnancy or when an airway problem becomes apparent [10 - 12] (Fig **1**).

SPECIFIC CONSIDERATIONS OF THE OBSTETRIC AIRWAY

Anesthetic management of the obstetric patient has changed in the last years [13 - 15], but it was not until 2015 that the first specific obstetric difficult airway guidelines were published by the Obstetric Anaesthetists Association (*OAA*) and the Difficult Airway Society (*DAS*) [9].

Pregnant airway management is greatly modified by anatomical and physiological changes during pregnancy, predominantly in the third trimester, and remain up to 2-3 weeks after delivery.

Anatomic modifications include weight increase and enlargement of breasts. Upper airway mucosa becomes edematous and Mallampati score increases, not only along pregnancy, but also during labour and delivery [16]. This mucosa bleeds more easily as the vascularization increases, and makes nasal intubation more complicated. Other factors like preeclampsia, oxytocin therapy and fluid administration can increase swelling.

Among the physiological respiratory changes, it should be noted that the pregnant uterus produces a cephalic displacement of the diaphragm that progressively decreases the expiratory reserve volume, which makes pregnant more susceptible

to hypoxemia and hypercapnia, causing shorter apnea times and facilitating earlier desaturation [17 - 22].

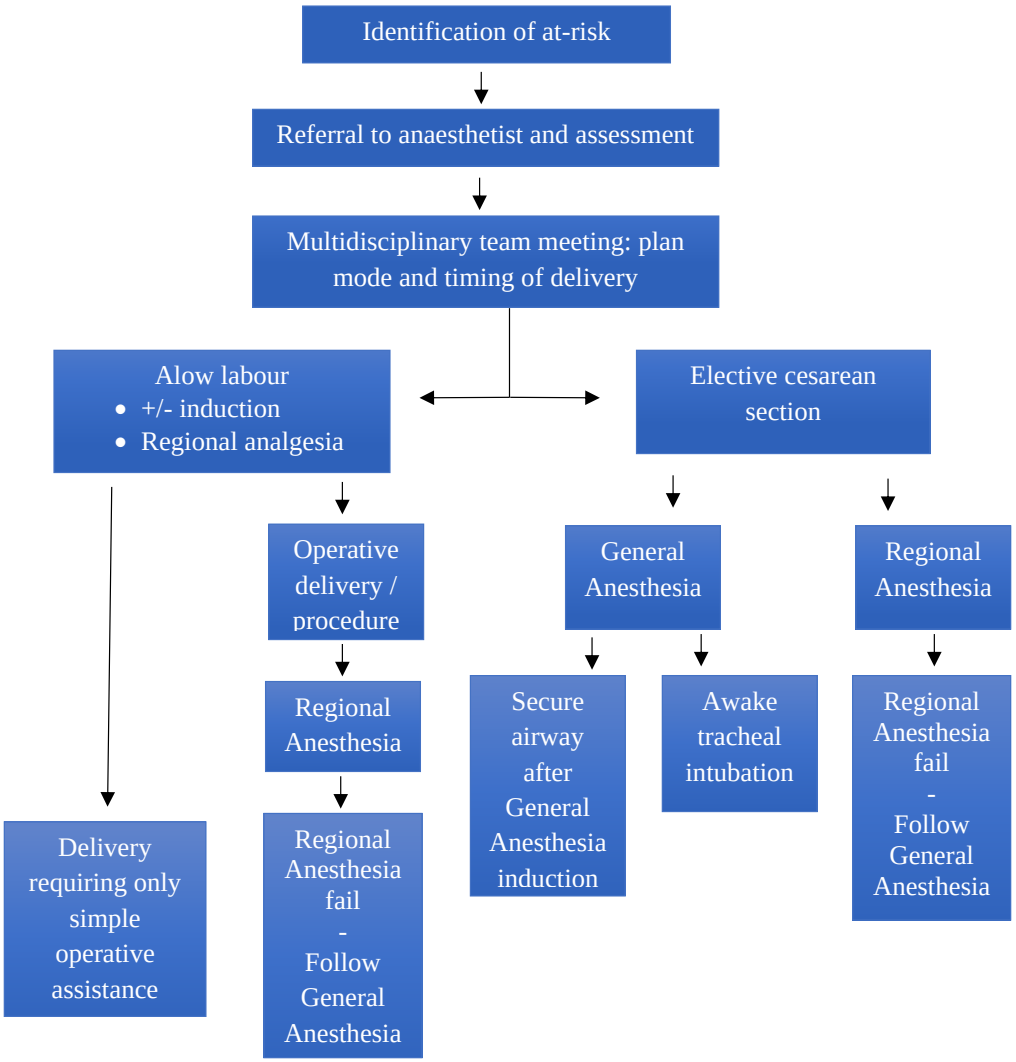

Fig. (1). Management of pregnant women with anticipated difficult airway.

The lower esophageal sphincter is relaxed, and gastric emptying is delayed by the action of progesterone. Increased gastric volume and hydrochloride production are due to placental secretion of gastrin.

CHAPTER 4

Anesthesia for Fetal Surgery

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Abstract: Fetal surgery has evolved in the last decades, mostly because of the technical advances in therapeutic and monitoring devices. The timing and mode of surgery depend on the disease to be treated. Local, neuraxial or general anesthesia can be used on the mother. In some cases, fetal analgesia and paralysis are needed.

The idea of treating the fetus as a patient has evolved in recent years, as a consequence of improvements in diagnostic imaging and surgical devices. In fetuses with congenital airway obstruction, intrapartum surgical correction or airway management can be performed while maintaining perfusion via the umbilical cord.

In 1980, maternal laparotomy and hysterotomy were proposed to treat fetuses with congenital and developmental abnormalities, and the prerequisites for maternal-fetal surgery were first formulated in 1982. They are still in use with some minor modifications. A multidisciplinary approach to fetal intervention is essential. Both obstetric and pediatric anesthesia is involved and it a close collaboration with surgical teams is necessary.

Keywords: Exit Procedure, Fetal Anesthesia, Fetal Surgery, Fetal Surgery Anesthesia, Maternal Anesthesia, Presto Procedure.

INTRODUCTION

The idea of treating the fetus as a patient has evolved in recent years, as a consequence of improvements in diagnostic imaging and surgical devices. The first fetal intervention was in 1963, when Liley performed an intraperitoneal blood transfusion for the treatment of erythroblastosis fetalis. In the 1980s, maternal laparotomy and hysterotomy were proposed to treat fetuses with congenital and developmental abnormalities.

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Most fetal anomalies are not amenable to prenatal correction. However, some entities are better managed before they can cause irreversible organ damage [1]. In fetuses with congenital airway obstruction, intrapartum surgical correction or airway management can be performed while maintaining perfusion via the umbilical cord. The prerequisites for maternal-fetal surgery were first formulated in 1982, and are still in use with some minor modifications. They require the following [2]:

1. The ability to establish an accurate prenatal diagnosis.
2. A well-defined natural history of the disorder.
3. The presence of a correctable lesion which, if untreated, will lead to fetal demise, irreversible organ dysfunction before birth, or severe postnatal morbidity.
4. The absence of severe associated anomalies.
5. An acceptable risk-to-benefit ratio for both the mother and the fetus. Fetal anesthesia is the anesthetic provided to pregnant women, or the anesthesia administered directly to the fetus, or both.

A multidisciplinary approach to fetal intervention is essential. Both obstetric and pediatric anesthesia are involved and it is necessary to have a close collaboration with surgical teams. This chapter focuses on the different procedures that are currently performed and their anesthetic management.

PHYSIOLOGY

Fetomaternal physiology is explained in detail in chapter 1. The most important aspects with regard to the anesthetic procedure will be summarized here.

-*Maternal Physiology*:

- Higher sensitivity to anesthetic agents, including inhalational anesthetics, non-depolarizing muscle relaxants, and epidural anesthetics.
- Hemodynamic changes: decreased systemic vascular resistance with high cardiac output. Risk of supine hypotension with aortocaval compression. The increased sympathetic tone is important for maintenance of blood pressure: marked hypotension can ensue after the use of drugs with vasodilating properties, or neuraxial blocks (sympathectomy). Placental blood flow depends on maternal blood pressure, so mantaining the maternal hemodinamics is essential for the fetus' well-being.
- Higher O_2 consumption and reduction in functional residual capacity lead to rapid oxygen desaturation and hypoxia during apnea. Edema of the upper airway

and mucosal fragility can lead to upper airway bleeding and difficult airway management.

-*Fetal Physiology*:

- Fetal cardiac output is highly dependent on the heart rate. The exposure of the fetus to high doses of volatile anesthetics can cause myocardial depression and bradycardia. Fetal bradycardia can lead rapidly to hypoperfusion, hypoxia, and acidosis.
- The fetus' capacity for thermoregulation is minimal, and depends on maternal temperature. Active warming of the mother and fetus is very important, especially in open fetal surgery.
- The question of pain perception in the fetus remains controversial. However, fetuses exhibit hormonal and circulatory stress changes in response to noxious stimuli. The long-term effects of fetal pain are currently unknown. Besides pain control, fetal analgesia helps to inhibit fetal movement, prevent hormonal stress responses associated with poor fetal outcomes, and possibly prevent adverse effects on long-term neurodevelopment [1, 3, 4].

GENERAL MANAGEMENT IN ALL INSTANCES OF FETAL SURGERY

Regardless of the type and timing of the intervention, there are some common measures that must be taken in almost all women subjected to fetal surgery [3]:

- Aspiration prophylaxis with multiple agents.
- Left lateral tilt to avoid aortocaval compression.
- Restriction of maternal iv fluids to avoid pulmonary edema.
- Aggressive blood pressure management to maintain uteroplacental perfusion: start therapy as soon as blood pressure decrease is detected. Both ephedrine and phenylephrine can be used safely in pregnant patients.
- Postoperative pain control.
- Prevention of preterm labor.
- Available personnel and equipment in case of delivery.

TYPES OF FETAL INTERVENTIONS

Fetal interventions can be divided into minimally invasive, open mid-gestation, EXIT and PRESTO procedures.

CHAPTER 5

Regional and Parenteral Analgesia in Labour

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Abstract: Labour pain is a complex phenomenon involving subjective psychological factors and physiological neurohormonal factors. Many different factors contribute to the perception of pain: cultural factors, bond, and trust in the delivery team, being able to take their own decisions, relaxation ability, previous labour, anatomical and fetal-related factors. Pain relief is one of the elements involved in overall satisfaction but it is not the only one and is important to remember that pharmacological intervention is only a part of it.

Keywords: Combined Spinal-Epidural, Dural Puncture Epidural, Epidural Analgesia, Fentanyl, Labour, Neuraxial Analgesia, Opioids, Paracervical Block, Patient-Controlled Analgesia, Pethidine, Pudendal Nerve Block, Remifentanil, Spinal Analgesia, and Newborn Outcome.

INTRODUCTION

The experience and individual response to labour pain are different in every woman, influenced by her own circumstances, cultural background, support, environment, as well as many labour-related issues (onset of labour, position, instrumental delivery, episiotomy, *etc*.) [1, 2].

Pain relief can be achieved by numerous techniques, some of which require medical intervention (intravenous drugs, regional analgesia, nitrous oxide), while others do not (relaxation, hypnosis, acupuncture, reflexology, *etc*). Whatever method is chosen, it must be safe for both mother and baby and it should make the

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birth experience as positive as possible. Pain is one of the factors with the most influence on labour and birth satisfaction [2].

Labour pain is a combination of visceral and somatic pain from uterine contractions and cervical dilatation, as well as from fetal descent through the pelvis, vagina and perineum. It is initially transmitted through T10-L1 roots, and as labour progresses it involves sacral roots S2-S4 (pudendal nerves) [3].

REGIONAL ANALGESIA

Regional analgesia for labour pain can include two groups of techniques. First, neuraxial techniques [4, 5], which are widely used, effective and safe, but are surrounded by many controversies yet to be clarified. Secondly, there are pelvic regional blocks that can be used to ease the pain, mainly during the second stage of labour [6, 7] (Table **1**).

Table 1. Techniques for regional analgesia for labour pain.

Technique	Advantages	Disadvantages
Continuous epidural analge sia (*CEA*)	- Continuous analgesia - No dural puncture - Adjustable dose	- Slow onset - Larger LA doses → higher risk of LAST, higher fetal exposition
Combined spinal-epidural analgesia (*CSE*)	- Rapid onset of analgesia - Wider sacral spread - Lower rate of epidural catheter misplacement	- Delayed verification of epidural catheter placement - Higher risk of postdural puncture headache (*PDPH*)
Dural Puncture Epidural (*DPE*)	- Faster onset - Better sacral spread - Lower incidence of asymmetrical block	- Increased risk for PDPH
Continuous spinal analgesia (*CSA*)	- Low dose of LA and opioid - Less hemodynamic impact - Rapid onset of analgesia	- Increased risk for PDPH - Risk of total spinal anaesthesia if spinal catheter mistaken for epidural catheter
Single shot spinal analgesia (*SSS*)	- Rapid onset of analgesia - Immediate sacral analgesia - Low LA dose	- Limited duration of action - Greater risk of maternal hypotension
Pudendal nerve block	- Less invasive technique - Low dose of LA	- Requires bilateral puncture - Limited duration of action
Paracervical block		- High risk of uterine artery puncture: fetal death, LA absorption

Pain and stress responses induce the release of corticotropin, cortisol, norepinephrine, beta-endorphins and epinephrine, all of which decrease uterine blood flow.

Pain reduction and sympathectomy caused by neural blockade result in lower levels of catecholamines and improvement in uteroplacental perfusion, especially in states of low uterine blood flow.

The ideal local anesthetic (*LA*) for labour should produce a reliable sensory block with no motor block, cause no tachyphylaxis and have a good safety profile so that inadvertent intravascular administration or overdose are harmless.

Neuraxial Analgesia

There are significant differences in the use of neuraxial analgesia for labour between different countries worldwide, and even between different hospitals in the same country.

Neuraxial analgesia in obstetrics includes spinal puncture, epidural catheter or a combination of both by a combined spinal epidural (*CSE*) technique [3 - 5].

Low dose LA, usually in combination with low dose opioid (fentanyl, sufentanil or morphine) is given by an initial bolus and different continued regimens to maintain analgesia through labour.

Neuraxial Techniques

-***Spinal Puncture*****:** The effect of a dural puncture and LA with or without adjuvant opioids (usually fentanyl, morphine or sufentanil) has a rapid onset but is limited by the duration of the LA/mixture administered. Hence, its use is limited to situations where a fast relief is required and a limited duration of the pain is expected, such as delivery. The low concentration of LA (bupivacaine 0,25%) is generally used in order to minimize hemodynamic effects and motor block.

- ***Standard Epidural Technique*****:** where a loss of resistance technique is used to identify the epidural space and a catheter is placed.

- The combined spinal-epidural (*CSE*) technique has shown several advantages over the epidural technique alone. It has a shorter onset of analgesia, as the initial dose is given in the subarachnoid space [4, 5, 8]. Even in the early stages, pain can be controlled by spinal opioids alone, with no sympathetic or motor block. It also allows better and faster sacral analgesia. It is associated with a lower rate of misplaced epidural catheters, but the verification of its proper placement and function is delayed by the initial spinal dose. It is also associated with a higher incidence of pruritus and possible higher risk of fetal bradycardia [5] (due to the rapid decrease in circulating catecholamines, which have a tocolytic effect, causing uterine tachysystole).

CHAPTER 6

Local Anesthetics and Adjuvants for Labor: Local Anesthetic Systemic Toxicity

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Abstract: The choice of drugs used during labor is almost as important as the analgesic technique selected since effective pain relief contributes directly to satisfaction: the better the pain relief, the higher the satisfaction. Although bupivacaine has traditionally been the most widely used local anesthetic, L-bupivacaine and ropivacaine have similar action profiles with a lower risk of cardiovascular and neurologic toxicity and especially less motor blockade, when used under low-concentration strategies. The use of adjuvants, especially opioids, allows us to improve the analgesic quality while reducing the total dose of local anesthetics, although their use should be individualized, and patients should be monitored and treated for side effects if they appear.

Keywords: Anesthetic Complications, Body Distribution, Bupivacaine, Dosage, Drugs, Fat Emulsions, Intralipid, Levobupivacaine, Lidocaine, Liposomal Bupivacaine, Lipid Emulsion, Lipid Shuttle, Lipid Sink, Local Anesthetic, Local Anesthetic Systemic Toxicity, Metabolism, Pharmacokinetics, Procaine, Regional Anesthesia, Ropivacaine, Tetracaine, Toxicity.

INTRODUCTION

Local anesthetics (*LA*) are a group of pharmacological agents that block the conduction of electrical nerve impulses, temporarily and predictably, causing a loss of sensitivity, that can affect any nervous structure, including the CNS.

The ideal LA for obstetric analgesia should have a good safety profile when acci-

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dentally overdosed or administered intravenously, produce a reliable sensory block, have no motor block, and no tachyphylaxis.

MECHANISM OF ACTION OF LOCAL ANESTHETICS

Local anesthetics inhibit the propagation of the nerve impulse by reducing the permeability of the voltage-dependent sodium channel, blocking the initial phase of the action potential. The lower sodium influx depresses excitability, the rate of depolarization, and therefore the amplitude of the action potential. To do this, local anesthetics must cross the nerve membrane, since their fundamental pharmacological action is carried out on the cytoplasmic side of the sodium channel [1].

The interruption of the afferent transmission of the painful stimuli that is achieved with neuraxial sensory block allows to achieve adequate anesthesia or analgesia, depending on the local anesthetic used and its dose. Since the effect of LAs varies depending on the size of the nerve and the amount of myelin around it, a differential block of fibers occurs following this chronology:

1. Increased skin temperature, and vasodilation (blocking of autonomous B and SC fibers).
2. Loss of thermal sensation, and pain relief (Aδ and C fiber block).
3. Loss of proprioception (AϒFibers).
4. Loss of touch and pressure sensation (Aβ fibers).
5. Loss of motor skills (Aα fibers).

The recovery from these neurological effects follows the reverse order of its onset. The differential blocking of the different types of fibers according to their thickness and amount of myelin can not only be explained by the differences between the layers of myelin, but also by the electrophysiological properties of the ion channel.

Properties and Implications of Local Anesthetics

Physiochemical properties of local anesthetics vary between them, defining their differences. These properties include potency, latency and duration of action.

Potency is determined by the liposolubility of the molecule. To exert their pharmacological action, local anesthetics must permeate the nerve membrane, which is 90% lipidic. There is a positive correlation between the liposolubility coefficient of the local anesthetics and their potency. Another factor that affects both the anesthetic potency and the duration of action is the vasodilator and redistribution power to the tissues, an intrinsic property of each local anesthetic

(Lidocaine is more vasodilator than mepivacaine and ropivacaine is more vasoconstrictor than bupivacaine) [1].

The onset of action or latency is highly conditioned by the pKa of each drug. The percentage of the non-ionized portion is inversely proportional to the pKa of the anesthetic (Table **1**). It is the main indicator that sets the beginning of action for the local anesthetic.

Table 1. Local Anesthetics doses of administration.

DRUG	BUPIVACAINE	L-BUPIVACAINE	ROPIVACAINE	LIDOCAINE	MEPIVACAINE
ANALGESIC	0.0625-0.2%	0.0625-0.25%	0.1-0.2%	0.75%	0.75%
ANESTHETIC	0.5%	0.5%	0.5-0.75%	2-5%	2%
MAX. DOSE (mg/kg)	2'5	2	2	4'5	5
ONSET (min)	10-30	10-12	6-7	5-10	10-15
DURATION (min)	280-480	240-360	180-480	120-240	180-360
pKa	8.1	8.1	8	7.9	7.6

Local anesthetics are highly protein-bound molecules, which correlate with their duration of action, where highly lipid-soluble local anesthetics have a longer duration of action. This is partly explained by the fact that said binding capacity determines the percentage of free ionized and non-ionized form and, therefore, its effect, as the non-bound fraction of the molecules are the ones responsible for the action of the local anesthetic [2].

In low plasmatic protein states, such as pregnancy, neonates or hypoproteinemia, local anesthetic systemic toxicity events can be seen, as the free active fraction is increased. We must have these hypoproteinemia states in mind as we estimate both the administered dose and the administration time.

The pH of the organism also determines the percentage of the anesthetics protein binding. Acidosis situations generate a marked decrease in the said fixation of the local anesthetic to plasma proteins, causing an increase in the drug free fraction, which can also lead to systemic toxicity. This is especially relevant with bupivacaine, whose free drug concentration can increase from 5% to 30% just because of the presence of acidosis [3].

Placental transfer of bupivacaine seems to occur by a passive diffusion mechanism, rather than active transport, and appears to be influenced by maternal and fetal plasma protein binding capacity, fetal pH, and placental performance.

CHAPTER 7

Anesthesia for Cesarean Section

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Abstract: Caesarean section is the most frequently performed surgery in adults, with a total of 20 million procedures per year. More than 70% of cases are due to lack of labor progression, fetal distress, breech presentation or previous cesarean section.

Obstetric anesthesia practice has substantially changed over the last 20 years. The main cause of this is the introduction of regional techniques to the detriment of general anesthesia, which has reduced maternal mortality due to complications such as gastric aspiration or difficulty in orotracheal intubation. In general, we can affirm that regional anesthesia is the most frequently used anesthetic technique for cesarean section, reserving general anesthesia for urgent or life-threatening situations.

Keywords: Cesarean Section, COVID-19, General Anesthesia, Hypotension, Spinal Anesthesia.

INTRODUCTION

An estimated of 20 million cesarean sections are performed worldwide every year, being the most frequent abdominal surgery carried out in adults [1].

In the USA, cesarean section is the most common surgical procedure, being performed approximately one million times per year [2]. More than 70% of cesarean sections are due to lack of labor progression, fetal distress, breech presentation or previous cesarean section.

Obstetric mortality related to anesthesia has dropped to the seventh leading cause of maternal mortality in the USA, and it accounts for 1-3 maternal deaths/million births in both the USA and the UK [3, 4].

The reduction in mortality has been associated with an increase in the use of

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regional techniques as opposed to general anesthesia. The use of the latter for cesarean section has decreased notably, being reserved for situations in which, either because of the urgency of the surgery, the woman's personal history, or failure of a regional technique, general anesthesia is the last alternative.

REGIONAL ANESTHESIA FOR CESAREAN SECTION

Complications of Regional Anesthesia

It is the most commonly used technique for cesarean section. Regarding neuraxial anesthesia, epidural anesthesia requires a longer time from its administration until the skin incision can be made (Table **1**), and it provides poorer intraoperative analgesia than the spinal technique (Table **2**). The latter, due to its rapidity of action, allows an earlier start of surgery and is the most frequently used anesthesia technique for cesarean section.

Table 1. Features of epidural anesthesia.

INDICATIONS	CONTRAINDICATIONS
- Elective or urgent cesarean section with previously established epidural block.	- Those inherent to epidural anesthesia. - Maternal refusal. - Emergent cesarean section (life-threatening situations).
ADVANTAGES	**DISADVANTAGES**
- Reduces the risk of bronchoaspiration and complications derived from orotracheal intubation difficulty. - Less administration of drugs with depressant effect on the fetus compared to general anesthesia. - Less arterial hypotension compared to intradural anesthesia. - Possibility of postoperative analgesia. - Allows the mother to enjoy childbirth.	- Long latency time. - Risk of arterial hypotension. - Risk of technique failure.

A sensory blockade level above T10 impedes cesarean-dependent somatic sensations. Elimination of visceral pain by peritoneal stimulation and uterus manipulation requires more extensive anesthesia. Only when an anesthesia level above T5 is achieved, women will be reliably pain-free during cesarean section.

The most frequently used local anesthetics are bupivacaine, levobupivacaine, and lidocaine. Opioids such as fentanyl or sufentanil can be added to enhance intraoperative analgesia. In the case of epidural anesthesia, 3-4 mg of morphine chloride can be administered through the catheter to ensure adequate postoperative analgesia.

Cardiovascular Effects

Maternal arterial hypotension is defined as a systolic blood pressure < 100 mmHg or a decrease in systolic blood pressure greater than 20% from its baseline level [5]. The importance of arterial hypotension after regional anesthesia is that it can cause adverse events in both mother and fetus. Maternal symptoms such as nausea, vomiting or dyspnea frequently accompany severe arterial hypotension. Adverse effects on the fetus, such as a low Apgar test score or umbilical acidosis, are related to the severity and duration of arterial hypotension.

Table 2. Features of spinal anesthesia.

INDICATIONS	CONTRAINDICATIONS
- Urgent cesarean section without previous epidural block. - Scheduled cesarean section.	- Those inherent to spinal anesthesia. - Maternal refusal.
ADVANTAGES	**DISADVANTAGES**
- Short latency. - Reduces the risk of bronchoaspiration and orotracheal intubation difficulty. - Simple technique with a low failure rate. - Allows the mother to enjoy childbirth.	- Increased risk of arterial hypotension when compared to epidural anesthesia. - Risk of postdural puncture headache.

The duration of the hemodynamic event is more important than its severity. A drop in maternal blood pressure of ≥ 30% does not affect the newborn need for oxygen [6], but arterial hypotension longer than 4 minutes is associated with neurological changes at 4-7 days of life [7].

Hemodynamic changes following neuraxial anesthesia result from the sympathetic nervous system block added to inferior vena cava compression by the gravid uterus, especially if the patient is in the supine position. The speed and extent of the sympathetic blockade, and thus the severity of hypotension, are direct consequences of the onset and spread of the local anesthetic in the neuraxial space. This explains the lower incidence of arterial hypotension in the epidural technique when compared to the spinal technique; in the former, the onset of the block is slower, allowing compensatory mechanisms to be activated.

Once spinal anesthesia has been performed, all efforts must be directed to prevent maternal arterial hypotension. A 15° left tilt of the patient is used to lessen the hemodynamic effect of aortocaval compression by the pregnant uterus. Leg compression has been shown to be more effective than its absence, but its benefit depends on the degree of compression employed.

CHAPTER 8

Locoregional Anesthesia Comments in the Obstetric Patient and Eventual Complications

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Abstract: Labor pain is associated with increased stress response and when it is excessive, it may lead to hypoxemia and fetal acidosis. The most important factor in obstetric analgesia is the desire for pain relief by the patient and neuraxial analgesia is the mainstay procedure in labor and in anesthesia for cesarean delivery. Continuous lumbar epidural analgesia is the mainstay of neuraxial labor analgesia. There are other methods, such as intrathecal block or combined spinal-epidural, that can be useful in specific cases. Despite being the safest and most effective method, the epidural labor analgesia may have some complications. Other therapies include bilateral paracervical block and pudendal block, which provide rapid onset analgesia (2–5 min). Although useful, they require training and are risky in cases of placental insufficiency or prematurity.

Keywords: Adverse Reactions, Central and Peripheral Blocks, Labor Pain, Local Anesthetics, Neuroaxial Analgesia.

INTRODUCTION

In 2018 in Western Europe, there were 5 million living births (birth rate of 9.7/1000 inhabitants) and nearly 28% of these had cesarean delivery (*CD*); Northern Europe had lower figures (for instance less than 10% CD in Scandina*via*), Southern Europe with higher figures was close to 28% CD [1]. Meanwhile in the same year 2018, there were 372.777 births in Spain, 90.3% from mothers under 40 years of age and 9.7% from mothers aged 40 or over (birth rate of 7.9/1.000 inhabitants) [2].

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Nearly 15.1% of these had instrumental vaginal delivery (*ID*) and 26.23% had cesarean delivery (*CD*), although 70%-80% were low risk pregnancies at the start of labor [3].

This epidemiological context determines to a greater or lesser extent, the assistance of the anesthesiologists during parturition. The incidence of analgesic epidural in childbirth in Spain varies greatly from region to region and from one type of hospital to another; it ranges between 30%-75% [4, 5].

In North America (USA, Canada), births with epidural average to 60-70% [6] and in France, nearly 80% [7].

PAIN AND LABOR ANALGESIA

Pain during labor and delivery is common. Most women describe labor pain as severe or most severe [8] and only 15% refer it as light to moderate pain [9]. It is of utmost important to have in mind that labour pain is not associated with a pathological event but rather with a physiological process that has psychological and cultural aspects as well [10].

MECHANISM OF PAIN

Although a large description of the mechanisms involved in labour pain [10 - 12] is out of the scope of this article, so a short summary is presented. Uterine contractions (first stage of labour) produce pain from mechanical distention of the lower uterine segment and cervix. It is transmitted through the hypogastric plexuses and the dorsal nerve roots of T10-L1 nerves to the spinal cord. It is mainly the visceral pain type (slow conduction and poorly localized) that is referred to the abdomen, lower back and rectum and readily susceptible to central neural blockade.

During the second stage of labour, the stretching of the lower birth canal by the fetal head descent produces pain in the thighs, legs, vagina, perineum and rectum; it is mainly somatic pain type (S2-S4 nerve roots, predominantly pudendal nerve dependent) with sharp, intense and well localized features and a little less susceptible to neural blockade.

CONSEQUENCES OF PAIN

Severe labor pain is associated with increased stress response generally innocuous during the course of an uncomplicated labor, but when excessive (extreme pain), it may lead to hypoxemia and fetal acidosis from increased plasma catecholamines and decreased oxygen transfer from the mother to the fetus (decreased placental perfusion plus leftward shift of oxygen-hemoglobin

dissociation curve from hyperventilation caused by pain), and even to incoordinate uterine activity and dysfunctional labor [11 - 16]. Besides it may also be related in some cases to postpartum depression events [17, 18].

LABOR ANALGESIA

The most important factor in obstetric analgesia is the desire for pain relief by the patient and neuraxial analgesia is the mainstay procedure in labor and in anesthesia for cesarean delivery [19]. The change from epidural analgesia to epidural anesthesia in the event of cesarean delivery allows to avoid general anesthesia and it is associated with a higher risk of difficult tracheal intubation [20 - 23]. In fact, neuraxial blockades in obstetrics are associated with lower morbidity and mortality compared to general anesthesia in CD [24 - 27].

Neuraxial Obstetric Analgesia

Neuraxial analgesia techniques are the most effective and safe means to relieve pain throughout parturition; they are the method of choice for the relief of labor pain and should be available to all women in labor (ACOG level of evidence I a, A grade recommendation) [19, 28 - 31]. Theses techniques lead to lower pain scores and patients are more satisfied with neuraxial analgesia than with any other labor analgesia schedule [12, 32].

Indications [11, 28, 29, 31, 33]

- ***Patient Request*:** Always with anamnesis and eventual physical and analytical examination. In women at high risk of developing anesthetic complications, antepartum anesthetic evaluation is recommended.
- ***Obstetric Specific Clinical Situations*:** Such as dynamic dystocia (increased catecholamines due to pain can interfere with uterine coordination); some cases of preterm parturition; difficult breech delivery; instrumental vaginal birth; some cases of twin and multiple pregnancy; some cases of previous uterine surgery.
- ***Medical Indications*:** Such as preeclampsia (spinal analgesia leads to 15%-25% mean reduction in high blood pressure values) (ACOG B grade of recommendation); many cases of severe heart and/or lung diseases; retinal detachment; some cases of cerebral vascular diseases; cases of contraindicated general anesthesia.

Contraindications [11, 28, 29, 31, 33]

- Refusal by the patient, misunderstanding or non-acceptance of the procedure.
- Local anesthetic and/or opioid alergy.
- Severe hypotension unresponsive to treatment including shock and severe hemorrhage.

CHAPTER 9

Uterotonic Agents

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Abstract: Postpartum haemorrhage due to uterine atony is one of the major causes of maternal morbidity and mortality worldwide. Different control strategies have been postulated, especially during the third stage of labour, but the gold standard treatment is the use of uterotonic drugs. There are currently three well-defined groups of drugs: oxytocics, ergot derivatives and prostaglandins. Although the literature is heterogeneous, it is clear that oxytocin is the uterotonic of choice in both prophylaxis and treatment of postpartum haemorrhage. Detailed knowledge of protocols based on current evidence is mandatory, which vary according to the different medical societies and dictate the doses and order of administration of different drugs.

Keywords: Caesarian Section, Carbetocin, Dinoprostone, Ergot Alkaloids, Methylergometrine, Misoprostol, Obstetric Myometrium, Oxytocin, Oxytocics, Postpartum Haemorrhage, Prostaglandins, Prostaglandins Synthetic, Uterotonics, Uterine Contraction.

INTRODUCTION

Nowadays, postpartum haemorrhage (*PPH*) is still the main cause of maternal death from obstetric haemorrhage, despite great efforts to reduce its incidence, especially in developed countries [1 - 6].

Uterine atony is the leading cause of PPH, both in caesarean section and vaginal delivery [3, 4]. Active control of the third stage of labour, through the administration of uterotonic drugs, early cord clamping and controlled traction of the umbilical cord until placental prevent bleeding during this period [1, 3 - 5].

Historically, the first drugs used as uterotonics were ergot alkaloids, later oxytocin and finally modern prostaglandins [5, 7]. According to available evidence, the order of administration of these drugs has been modified based on their efficacy, tolerance and adverse effect profile [5].

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Oxytocics are currently considered the drugs of choice for the prevention of PPH, since they have a safety and efficacy profile that is superior to that of other drugs of this group [4, 5, 7, 8].

Ergotamine derivatives, despite being effective in the prevention of postpartum haemorrhage, have greater adverse effects, so they are reserved as a second therapeutic step.

Prostaglandins play an important role in the treatment of postpartum haemorrhage, but they are not considered first-line drugs, neither for prophylaxis nor for treatment [4, 5, 7, 8].

Uterine atony risk factors include uterus overdistension, oxytocin infusion during labour, chorioamnionitis, placenta previa, previous uterine atony, multiple gestations and high parity [8].

Prompt communication between the obstetrician and the anesthesiologist is very important in order to establish the patient's individual needs according to the clinical setting, which determines uterotonic requirements in each specific case.

UTEROTONIC AGENTS

Uterotonic drugs promote adequate uterine contraction by increasing uterine basal tone and activating the frequency, intensity and duration of uterine smooth muscle contractions [1, 3, 4].

The characteristics of an ideal uterotonic are shown in Table **1** [4].

The administration of uterotonic drugs, therapeutically or prophylactically, is a fundamental measure to reduce the incidence of PPH due to uterine atony [4].

Oxytocin

Oxytocin is a hormone synthesized in the hypothalamus and secreted by the posterior pituitary gland in a pulsatile fashion. It is also synthesized in the umbilical cord, chorion and decidua, and is responsible, among other actions, for uterine contractions [1, 3, 5 - 7].

Table 1. Characteristics of an ideal uterotonic.

- Highly effective
- Few adverse effects
- Thermoresistant
- Easy to administer
- Predictable and optimal pharmacokinetic profile

Oxytocin was discovered in 1906 by Sir Henry Dale [6, 7]. Since Vincent du Vigneaud first artificially synthesized it in 1954, it has become an essential drug in obstetric practice for both, induction and optimization of labour, and subsequently for the prevention and control of PPH [4 - 8]. In the postpartum period, it acts by slowing placental bed haemorrhage [6].

Oxytocin is also synthesized in peripheral tissues such as the uterus, corpus luteum, amnion, umbilical cord, placenta and testis. It is involved in various physiological and pathological actions like maternal behaviour, milk ejection, erectile dysfunction and ejaculation [1, 3, 5, 6].

Oxytocin is poorly protein bound, can be administered intravenously and it has an onset of action of 1 to 2 minutes and a half-life of 15 minutes. Therefore, it should be given as a continuous infusion. Intramuscular administration is also feasible; by this route, its onset of action is 2 to 4 minutes and its half-life is lengthened, lasting between 30 and 60 minutes [4].

The concentration of oxytocin receptors in the myometrium of non-pregnant women is low, increasing progressively during pregnancy until labour, when it is doubled [1, 6, 8]. Both oxytocin and carbetocin do not show significant uterine contraction in non-pregnant women [8].

Oxytocin and its analogues are agonists of the transmembrane oxytocin receptor (*OTR*), G-protein-coupled receptors family involved in the transmission and signalling of various intracellular pathways [1, 5, 6, 8]. Although some of these routes and mechanisms are not fully elucidated, OTR functions are clear in the physiology of pregnancy and childbirth [1]. OTR is expressed throughout the body, *e.g.* in the circulatory system, the central nervous system, as well as in the myometrium and endometrium [6, 8].

Oxytocin molecule binding to OTR leads to activation of a phospholipase that catalyses the conversion of phosphoinositide-bis-phosphate (*PIP2*) to inositol-tri--phosphate (*IP3*) and diacylglycerol (*DAG*) [1, 6]. IP3 enhances calcium release from the sarcoplasmic reticulum and thus, cytoplasmic calcium increases, which results in the stimulation of Ca^{2+}-dependent calmodulin. The latter activates a myosin light chain kinase (*MLCK*) that triggers the contraction-relaxation cycle of smooth muscle [1, 3, 6]. Additionally, DAG, through a series of kinase-mediated cascades, increases prostaglandin E2 synthesis, which is also involved in myometrial muscle contraction [1, 6].

The action of oxytocin has a ceiling effect. Once all the receptors are blocked, a phenomenon called oxytocin receptor desensitization, uterine contractions intensity will not increase, predisposing to increased bleeding and potentiation of

CHAPTER 10

Anesthesia for Non-Obstetric Surgery in Pregnancy

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Abstract: The need for non-obstetric surgery during pregnancy is relatively frequent and can occur at any time during pregnancy. In this chapter, we will develop the anesthetic implications of changes in maternal physiology, and the repercussions of anesthesia on the fetus, and we will delve into the peculiarities of anesthetic management of these patients. Urgent/emergent procedures should not be postponed in these patients due to their pregnancy conditions. However, elective surgeries should be delayed whenever possible, taking into account the maternal-fetal risk-benefit.

Keywords: Anesthesia, Embryogenesis, General Anesthesia, High-Risk Pregnancy, Laparoscopy, Maternal-Fetal Risk, Monitoring, Non-Obstetric Surgery, Obstetrics Anesthesia, Obstetrics, Outcome, Positioning, Pregnancy, Preoperative Assessment, Safety, Surgical Procedures, Surgery, Spinal Anesthesia, Teratogenicity, Trimester.

INTRODUCTION

It is estimated that 1-2% of pregnant women would undergo non-obstetric surgery during pregnancy [1]. Forty-two percent of cases occur during the first trimester, 35% in the second, and 23% in the last trimester [2]. This percentage does not include patients with undetected or suspected pregnancies who undergo elective or emergency surgeries. The most frequently performed procedures are appendectomy and cholecystectomy, followed by other surgeries such as intestinal obstruction, adnexal torsions, breast pathology, and trauma interventions. Pregnancy predisposes to the development of cholelithiasis due to an increase in biliary lithogenicity and a decrease in gallbladder motility secondary to hormonal effects, and the risk of acute cholecystitis increases with advancing gestational age [3]. Some cancers are sometimes aggravated by the

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hormonal changes in pregnancy, such as meningiomas that express estrogen or progesterone receptors and grow during this period [4].

Diagnosis of surgical processes is more complex than in non-pregnant patients due to the difficulty of the abdominal exploration and the anatomical, physiological and analytical alterations of pregnancy. This delay in diagnosis can result in diseases reaching advanced stages (*e.g.,* peritonitis) and increased maternal morbidity and mortality [5].

ANESTHETIC IMPLICATIONS OF PHYSIOLOGICAL CHANGES IN THE PREGNANT WOMAN

The physiological changes in the pregnant patient are discussed in detail in chapter **1**. In this section, we describe the implications of these changes on anesthesia and the perioperative period (Table **1**).

Table 1. Anesthetic risks of physiological changes in pregnant women.

-	Physiological changes	Risks
Respiratory	↓ functional residual capacity ↑ metabolic intake of O_2	Hypoxemia, hypercapnia, acidosis. ↓ uteroplacental blood flow Fetal hypoxemia
Airway	Edema and mucosal congestion ↑ breast and body mass	Difficult ventilation and intubation Maternal-fetal hypoxemia
Cardiovascular	"*Supine hypotensive syndrome*" Susceptibility to sympathetic blocks or vasodilator drugs	↓ cardiac output ↓ uteroplacental blood flow Fetal hypoxemia
Neuroaxial	Reduced epidural space ↑ epidural venous plexus	↑ diffusion and blockade by local anesthetics Risk of intravascular injection and local anesthetic toxicity
Gastrointestinal	↓ gastric emptying ↑ intragastric pressure ↑ intragastric acidity ↓ lower esophageal sphincter pressure	Regurgitation and aspiration Maternal-fetal hypoxemia
Hepatorenal	Hepatorenal alteration Alteration of plasma proteins	↑ drug toxicity ↑ pharmacological clearance
Hematological	Dilutional anemia Hypercoagulability	↓ symptoms of moderate bleeding ↑ thromboembolic risk

Impact of Respiratory Changes

Pregnancy is characterized by a decrease in the functional residual capacity that becomes significant around week 20 and an increase in metabolic oxygen

consumption. All this predisposes to hypoxemia, hypercapnia and acidosis, which can be accentuated in the presence of hypoventilation and apnea. Pre-oxygenation with 100% oxygen, administration of supplemental oxygen in the perioperative period and the monitoring of oxygen saturation are essential. A decrease in pCO_2 can produce vasoconstriction of the uterine arteries and a decrease in uteroplacental blood flow, therefore it is mandatory to monitor CO_2 exchange, and target normocapnia. Decreased functional residual capacity and increased minute ventilation lead to an increase in the induction rate with inhalation anesthesia [5].

Impact of Changes on the Airway

The risk associated with ventilation and intubation of the pregnant patient is created by edema secondary to fluid retention, mucosal congestion, increased tongue size, reduction in the diameter of the oropharynx, and increased size of the breasts and fatty tissues. These conditions increase the risk of difficult ventilation and intubation, so it is a priority to expect problems in airway management and anticipate a failed intubation [6].

Impact of Cardiovascular Changes

In the second half of pregnancy, attention must be paid to aortocaval compression produced by the uterus, which reduces venous return, preload, and cardiac output in a condition known as "*supine hypotensive syndrome*" [7]. A drop in cardiac output of up to 25% can occur when the patient is placed supine with respect to the left lateral decubitus, while the displacement of the uterus to the left decreases compression, increasing end-diastolic volume, stroke volume, and left ventricular ejection fraction [8]. Neuroaxial or general anesthesia can accentuate cardiovascular depression in the supine position, due to sympathetic block or vasodilation.

Impact of Neuroaxial Changes

The epidural space of the pregnant woman presents distended venous plexuses and a decreased capacity for distension, increasing the risk of intravascular injection of local anesthetics, more extensive dissemination of local anesthetics, and blockage of unwanted levels.

Impact of Digestive Changes

Mechanical and hormonal factors delay gastric emptying, increase intragastric pressure and acidity, and decrease the pressure of the lower esophageal sphincter (*LEE*), increasing the risk of regurgitation and aspiration from the second trimester. Therefore, in pregnant women, over 18 to 20 weeks, prophylaxis thirty

CHAPTER 11

Anesthetic Management of the Pregnant Patient with Comorbidities

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Abstract: An increase in pregnant patients with comorbidities has been seen in the last decade. Nevertheless, these patients are able to enjoy longer and better quality lives nowadays. During pregnancy, patients can experience decompensations of their chronic disease which can be sometimes challenging for the medical team. Complexity has risen; that is why the anesthesiologist must be updated and capable of facing different scenarios both in the delivery room and before or after birth.

This chapter offers a practical and synthetical approach to the most common situations in which a general anesthesiologist can be involved, aiming to emphasize main points for safe and accurate anesthetic care.

Keywords: Anemia, Aortic Stenosis, Autoimmune, Chorioamnionitis, Cardiovascular, Comorbidity, Coagulation, Diabetes Mellitus, Epilepsy, Epidural, Fever, Hepatic, Hyperthyroidism, Lupus, Mitral Stenosis, Multiple Sclerosis, Myasthenia Gravis, Neurologic, Neuraxial, Neurofibromatosis, Obesity, Platelets, Pregnancy, Pulmonary Hypertension, Renal, Respiratory, Sepsis, Septic Shock, Spinal Cord Injury, Thromboprophylaxis.

INTRODUCTION

Neurological, neuromuscular and musculoskeletal disorders can influence the obstetric outcome during operative deliveries, and ideally all such operative interventions should be referred to tertiary care centers.

During the preanesthetic stage, cardiorespiratory evaluation should be thoroughly done. Regional anesthesia is preferred in the majority of these patients except for a few strong contraindications such as increased intracranial pressures, tethered spinal cord and others. Patients with the high risk of developing intra-operative

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respiratory insufficiency should preferably be administered regional anesthesia in an incremental manner.

Multiple Sclerosis

For labor analgesia with epidural, use lower concentrations of local anesthetic when possible. Neuraxial anesthesia is the preferred anesthetic technique for cesarean delivery. Anyway, the type of anesthesia selected does not influence the relapse rate. If general anesthesia is given, succinylcholine should be administered cautiously and only if strongly indicated.

Spinal Cord Injury

Epidural analgesia should be used as soon as the patient goes into labor to prevent autonomic hyperreflexia (injuries at or above T6) and mass reflex. For cesarean section, epidural anesthesia is preferable to spinal anesthesia because the chances of hypotension are less. If general anesthesia is essential, avoid succinylcholine. Also remember that pregnancy may aggravate medical complications of spinal cord injury (decreased respiratory reserve, atelectasis, pneumonia, anemia, deep venous thrombosis, renal insufficiency, decubitus ulcers, autonomic hyperreflexia).

Myasthenia Gravis

Neuraxial anesthesia is preferred for labor analgesia, and amide local anesthetics are preferable. Because of the need for a higher level of sensory anesthesia for cesarean delivery, there is a danger of impairment of the respiratory and swallowing muscles following regional anesthesia. Unless contraindicated because of respiratory insufficiency, regional anesthesia should be the technique of choice.

We should have caution with the use of opioids, and avoid magnesium sulfate, aminoglycosides, fluoroquinolones, tetracyclines, and macrolides antibiotics. Also, patients are extremely sensitive to non-depolarizing muscle relaxants and resistant to depolarizing muscle relaxants. There is a risk for postoperative ventilation if the duration of myasthenia is greater than 6 years, there is a history of chronic respiratory disease, pyridostigmine dose is higher than 750 mg/day, and female gender.

Epilepsy

There is no contraindication to the administration of neuraxial analgesia or anesthesia. If general anesthesia is necessary, we should avoid ketamine, enflurane, and meperidine, as these may lower the seizure threshold. Some

antiepileptic medications induce liver enzymes, which may lead to the rapid breakdown of anesthetic agents metabolized by the liver.

Muscular and Myotonic Dystrophy

Neuraxial anesthesia is preferred due to a higher risk of apnea with opioids in these patients.

Neurocutaneous Syndromes

Severe kyphoscoliosis may be present, and lesions that involve the neck and larynx are common in neurofibromatosis type 1. Asymptomatic paraspinal and intracranial tumors may be present; clinical and radiologic evaluations may be indicated prior to neuraxial anesthesia.

Brain Tumors

Painful uterine contractions and bearing-down efforts increase intracranial pressure; hence epidural analgesia may be indicated, but bear in mind the consequences of accidental dural puncture. General anesthesia is preferred for cesarean deliveries.

Cerebrovascular Accidents

For labor and delivery, a continuous epidural block is advisable. The use of forceps is indicated to shorten the second stage. In the immediate postpartum period, one should be prepared to treat hypertension aggressively if it occurs. For cesarean delivery, an epidural block is the anesthesia of choice; however, if there is fetal distress or if general anesthesia is indicated for some other reason, be careful about the hypertensive response following endotracheal intubation.

PREGNANT PATIENT WITH CARDIOVASCULAR DISORDERS

Heart disease is the primary medical cause of non-obstetric maternal mortality. The most common cardiac condition encountered in pregnant women in developed countries is congenital heart disease followed by rheumatic heart disease.

During pregnancy, there are physiological changes in hemodynamics such as: an increase in the intravascular volume, a decrease in systemic vascular resistance (*SRV*), an increase in the heart rate (*HR*) and thus in cardiac output (*CO*). There is also a hypercoagulability status and a decrease in the functional residual capacity. Due to all these changes, cardiac decompensation is the main concern in pregnant patients with a heart disease.

CHAPTER 12

Anesthetic Management of Pregnant Patients with Infectious Disease

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Abstract: Fever is often the result of an infection. The most common sites for infection during pregnancy are fetal membranes, urinary and respiratory tracts, and the postpartum uterine cavity. The most frequent etiologies of intrapartum fever are chorioamnionitis and neuraxial anesthesia. Maternal and fetal exposure to hyperthermia and inflammation is associated with adverse consequences for the mother and the neonate. In pregnant women with fever, anesthesiologists are not only involved in providing analgesia, but also in the correct anesthetic management for the surgical treatment of the infectious region. Thus, as pyrexia may change both obstetric and anesthetic management, preventing maternal fever is imperative. Emerging and challenging infectious diseases, as COVID-19, remind us of the susceptible nature of pregnant and early postpartum women to severe respiratory infections, reinforcing the importance of vaccines and therapeutic measures during pregnancy.

Keywords: Anesthetic Management, Chorioamnionitis, COVID-19, Epidural Analgesia, Epidural Abscess, Febrile Pregnant, Fever, Fetal Membranes, Inflammation, Infection, Intrapartum Fever, Intraamniotic Infection, Labor, Maternal Fever, Neuraxial Procedures, Neuraxial Anesthesia, Postpartum Infection, Pregnancy, Pyrexia, Sepsis.

INTRODUCTION

Fever is defined as an elevation of body temperature above normal daily variation, occurring when the hypothalamic thermoregulation centre is reset at a higher temperature by the systemic release of endogenous pyrogens, including cytokines, interleukin (*IL*)-1, IL-6, tumor necrosis factor (*TNF*) and interferon-alpha (*IFN-α*), produced in response to infection, inflammation, injury or antigenic challenge. This cascade is chiefly commanded by IL-6 and IL-1B and is inhibited by the

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anti-pyrogenic cytokine IL-1 receptor antagonist (*IL-1ra*) [1]. High circulating levels of inflammatory biomarkers, such as IL-6 and IL-10, have been demonstrated in patients with active labor [2].

Intrapartum fever refers to maternal oral temperature ≥ 38°C, as axillar measurements (1-2°C lower), are susceptible to user error. The vaginal temperature might be affected by epidural analgesia-induced sympathectomy and vaginal mucosal vasodilation. Fetal/intrauterine temperature is 0.2-0.9°C higher. A progressive increase of temperature with each contraction over time (from 1.5 °C to 0.5°C depending on parity) has been known since the XIX century, attributed to the muscular work of the uterus. Some studies have shown that it is more likely to occur in women with placental inflammation.

A prospective cohort study among 6057 deliveries suggested that the incidence of maternal intrapartum fever was approximately 6.8%, even though its incidence has been reported to be higher in long labors (> 18h) compared to shorter ones (< 6h) (36% *vs*. 7%) [3].

Burgess *et al.* showed that its development is associated with: nulligravidity, length of first stage ≥ 720 minutes (min), length of second stage ≥ 120 min, membrane rupture ≥ 240 min, increasing number of vaginal exams, oxytocin, and meperidine. Other related factors are induced labor, use of epidural analgesia, method of how analgesia is administered, duration of exposure to epidural analgesia, higher birthweight and prolonged gestation.

Maternal and fetal exposure to hyperthermia and inflammation causes both significant maternal effects (increased maternal heart rate, cardiac output, oxygen consumption, and catecholamine production) and adverse neonatal outcomes. Associated morbidity includes caesarean delivery (C-section), Apgar score < 7 at 5 min, and neonatal intensive care unit admission due to hypotonia, assisted ventilation and seizures in the newborn [4, 5].

Different treatments have been tested, with diverse results: high-dose methylprednisolone resulted in lower levels of IL-6 and less fever, but it is associated with increased risk of neonatal bacteremia [6]. Prophylactic acetaminophen and preventive antibiotics are not useful. Intrapartum magnesium-sulfate reduced temperature during labor, either by inhibiting the expression of IL-6 [7], or inducing peripheral vasodilation [8]. And dexmedetomidine can reduce both intrapartum fever and pain during labor, with no increased adverse events, by inhibiting cytokines and alle*via*ting inflammation.

ETIOLOGY OF MATERNAL FEVER

Infectious Causes

Intraamniotic Infection (Chorioamnionitis)

Chorioamnionitis refers to infection of the amniotic fluid, membranes, placenta, and/or decidua, and is one of the most common infections in pregnancy. Independent risk factors include low parity, a history of prior chorioamnionitis, the number of vaginal examinations, both duration of total labor and ruptured membranes, and use of internal monitors [9].

The diagnosis of chorioamnionitis has been based upon the presence of maternal fever (≥ 38°C) and at least two of the following: maternal tachycardia (> 100 beats per minute [bpm]), fetal tachycardia (> 160 bpm), uterine tenderness, foul odor of the amniotic fluid, and maternal leukocytosis (> 15.000 cells/mm^3) [10].

Since intrapartum fever is the key clinical sign of a chorioamnionitis, and as there are no findings either sensitive nor specific, antibiotics are usually administered in the presence of maternal fever when other infection sources have been excluded. Once chorioamnionitis is suspected, prompt treatment with broad-spectrum antibiotics with coverage for group B *Streptococcus* must be initiated (*i.e*. Ampicillin plus gentamicin) as this reduces maternal and neonatal morbidity.

The most common isolated organisms from the amniotic fluid in chorioamnionitis are *Bacteroides* species (sp.), group B *Streptococcus*, *Mycoplasma* and *Ureaplasma sp*., and *E. coli*. Additionally, general supportive measures (acetaminophen, rehydration…) are very important.

Maternal complications of this infection include preterm labor, placental abruption, postpartum infection, uterine atony, postpartum hemorrhage, peripartum hysterectomy, sepsis, and death [9].

The cornerstone of obstetric management of these patients is prompt delivery. Several studies suggest that early, antepartum, treatment results in decreased maternal and neonatal morbidity. The early use of antibiotics may also affect the anesthesiologist's decision regarding the performance of neuraxial technique [9].

Urologic Infections

Urinary tract infections are common in pregnancy due to increased concentrations of progesterone (which causes ureteral dilatation that facilitates bacterial ascent from the bladder) and the partial ureteral obstruction caused by the gravid uterus (provoking urinary stasis) [9].

CHAPTER 13

Obstetric Hemorrhage

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Abstract: Postpartum hemorrhage (*PPH*) is the most common cause of obstetric hemorrhage (*OH*) and the first cause of maternal death worldwide. However, its mortality has decreased in the United States in recent decades due to the implementation of certain measures. Several factors are significantly associated with the onset of PPH. Identifying them early will help us to establish an appropriate strategy for the management of these patients. One of the most useful prevention measures is the active management of the third stage of labor, especially the routine administration of oxytocin. The existence of acting algorithms will also contribute to a rapid and orderly response to the presence of expected or unplanned bleeding.

Keywords: Artery Embolization, Bleeding, Blood Bank, Cesarean Birth, Clamping Umbilical Cord, Coagulopathy, Disseminated Intravascular Coagulation, Hemorrhage, Hysterectomy, Intrauterine Tamponade Balloon, Labor, Massive Transfusion, Massive Obstetric Hemorrhage, Oxytocin, Postpartum Period, Postpartum Hemorrhage, Placental Disorders, Rotational Thromboelastography, Thromboelastography, Trauma, Uterotonic Drug, Uterine Atony, Uterine Massage, Vaginal Delivery.

INTRODUCTION

Postpartum hemorrhage (*PPH*) is the most common cause of obstetric hemorrhage (*OH*) and the first cause of maternal death worldwide. When the bleeding occurs within the first 24 hours after delivery, PPH is called early PPH, and it will be the subject of this chapter. Hemorrhage between 24 hours and 6-12 weeks after delivery is called late PPH [1].

Definitions vary depending on the author or organization, with the World Health Organization's (*WHO*) definition being one of the most frequently used. According to it, PPH is a blood loss greater than 500 mL within the first 24 hours

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postpartum. When the blood loss is larger than 1000 mL within the same time frame, it is considered a severe PPH, and if the blood loss is greater than 40% of the patient's total blood volume, which during pregnancy is increased by 100 mL/kg, it is named massive obstetric hemorrhage (*MOH*).

The American College of Obstetricians and Gynecologists defines PPH as bleeding associated with the process of childbirth that either has an accumulated blood volume loss greater than 1000 mL or any blood loss accompanied by signs or symptoms of hypovolemia, regardless of the type of delivery (vaginal or cesarean section) [2].

The incidence of PPH depends on the diagnostic criteria used. When the diagnostic criteria are met by quantified blood loss, PPH incidence is higher than when the losses are estimated. Its mortality has decreased steadily in the United States since the late 1980s, due to increased transfusions and hysterectomy rates.

CAUSES OF POSTPARTUM HEMORRHAGE

The mnemonic rule of the "*4Ts*" summarizes the most frequent causes of PPH [3]:

- *Tone*: This includes abnormalities in uterine contractions. Uterine atony is the most common cause of PPH and is responsible for at least 80% of cases, mainly in its diffuse type. Uterine atony occurs when the uterus cannot contract after delivery, and it can happen with or without tissue retention. A soft, poorly contracted uterus suggests this diagnosis.

In diffuse atony, blood loss can be significantly greater than clinically estimated as a substantial amount of blood can remain inside a flaccid and dilated uterus. When the atony is focal, only the lower segment of the uterus tends to be the most affected, and it is difficult to detect during physical examination.

- *Tissue*: This includes causes of PPH related to retained products of conception.
- *Trauma*: When the bleeding is due to any type genital trauma during childbirth. The most frequent are secondary to genital tract lacerations (in this case, diagnosis can be delayed until excessive vaginal bleeding ensues), uterine body lacerations, surgical incisions in cesarean deliveries (generally due to lateral extension of the incisions), or uterine rupture.
- *Thrombin*: Postpartum hemorrhage due to clotting disorders. Both acquired and hereditary coagulopathies are responsible for at least 7% of PPH cases. Also, coagulopathies can result from PPH when significant and persistent bleeding induces a severe reduction of the clotting factors.

Risk Factors

Several factors are significantly associated with the onset of PPH. However, many patients without any risk factor also develop OH.

The following tables list the main risk factors according to the moment of onset (Tables **1** and **2**) [3].

Table 1. Main risk factors according to the moment of onset. Presentation before delivery: significant risk of PPH.

RISK FACTOR	4T	Odds ratio (CI 95%)
Placental abruption	Thrombin	13 (7,61-12,9)
Placenta previa	Tone	12 (7,17-23)
Multiple pregnancy	Tone	5 (3,0-6,6)
Preeclampsia	Thrombin	4
Previous PPH	Tone	3
Asian ethnicity	Tone	2 (1,48-2,12)
BMI>35 kg/m2	Tone	2 (1,24-2,17)
Hemoglobin<9g/dl		2 (1,63-3,15)

Table 2. Main risk factors according to the moment of onset. Presentation during or after delivery.

RISK FACTOR	4T	Odds ratio (CI 95%)
Urgent cesarean section	Trauma	4 (3,28-3,95)
Programmed cesarean section	Trauma	2 (2,18-2,80)
Induction of labor		2 (1,67-2,96)
Retained placenta	Tissue	5 (3,36-7,87)
Mid-lateral episiotomy	Trauma	5
Instrumental delivery	Trauma	
Prolonged delivery (> 12h)	Tone	2
Large for gestational age newborn (> 4 kg)	Tone/Trauma	2 (1,38-2,60)
Fever during labor	Thrombin	2
Mother age> 40	Tone	1,4 (1,16-1,74)

The California Risk Classification subdivides risk factors according to the intensity of the risk [1]:

CHAPTER 14

Hypertensive Disorders in Pregnancy

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Abstract: Due to the high risk of morbidity and mortality in pregnant women with unrecognised and untreated preeclampsia, a high index of suspicion for signs of preeclampsia should be used to evaluate, treat and monitor patients. Early blood pressure control and seizure prophylaxis during labour are essential to ensure maternal safety. However, a limited proportion of pregnancies and deliveries may present a wide range of complications that may require admission to a critical care unit (*CCU*). Hypertensive disorders of pregnancy and massive hemorrhage are among the most common causes of admission to the CCU in pregnant and post-partum women.

Keywords: Blood Pressure, Biomarkers, Cardiovascular Disease, Eclampsia, Fetal Outcome, Foetus, HELLP Syndrome, Hypertension, Hypertensive Disorders, Labetalol, Maternal Morbidity, Maternal Health, Nifedipine, Pathogenesis, Placental Dysfunction, Preeclampsia, Pregnancy.

INTRODUCTION

Hypertensive pregnancy disorders (*HRD*) are a complication in about 5% to 10% of pregnancies, with a 25% increase over the past 20 years [1, 2]. Hypertensive disorders, associated with delayed or inappropriate treatment of severe systolic hypertension, remain the leading cause of maternal death. Thus, in the United States (US), there is a death/day, and worldwide there are up to 50-60,000 deaths/year [3 - 6]. The vast majority of these deaths result from hemorrhagic strokes and complications related to seizures [7 - 10]. Furthermore, for every maternal death associated with HRD, there are between 50 and 100 complications [11 - 15]. Therefore, the goal of treatment of pregnant women with HRD is to prevent morbi-mortality, which is achieved through aggressive blood pressure

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(*BP*) treatment, seizure prophylaxis with magnesium, timely delivery, and post-partum surveillance.

PATHOPHYSIOLOGY OF PREECLAMPSIA

The pathophysiology of preeclampsia remains unclear. It is triggered by a marked vasoconstriction state, secondary to vascular endothelial dysfunction, when adequate vasodilation typical of normal pregnancy is not carried out.

Preeclampsia is a complex disease whose aetiology seems multifactorial. Placental disturbance plays a central role, although other genetic and immunologic factors must also be considered, as well as various predisposing factors of the fetus and the mother.

All these elements, taken together, condition a pathology that, although it begins at the placenta level, leads to multi-systemic damage, with a particular renal, hepatic and neurological impact.

There are a number of pathophysiological mechanisms which may be regarded as responsible for preeclampsia.

Uterine Artery Remodelling

The formation of the placenta is a key process in gestation, as this organ is responsible for adequate blood perfusion from the mother to the foetus, which is essential for normal fetal development. The placenta is formed from fetal cytotrophoblastic tissue, which forms the trophoblastic villi, structures that support the blood vessels of the fetus. Spiral arteries, branches of the maternal uterine artery, direct oxygenated blood and nutrients from the maternal circulation to the fetal circulation.

In some patients, it has been shown that interstitial invasion by cytotrophoblasts cannot penetrate the myometrium [16]. Trophoblast differentiation towards the endothelial phenotype characteristic of normal placentation is not effectively completed [17]. As a result, the spiral arteries remain reduced in calibre, limiting blood flow to the foetus and leading to placental ischemia.

Immunological Factors

Natural killer (*NK*) cells and macrophages are involved in the placentation process. Uterine NK cells migrate to the decidua and secrete cytokines, interferon-γ (*IFN-γ*), vascular endothelial growth factor (*VEGF*), placental growth factor (*PlGF*), among others, all of which are involved in spiral artery remodelling [18].

A predominance of uterine NK cells with an aberrant phenotype has been demonstrated in pregnant women with preeclampsia. It contributes to the disruption of placentation through the production of unusual amounts of cytokines that prevent proper interactions with cytotrophoblasts [19, 20].

On the other hand, during normal gestation, the immune system provides a privileged state for the foetus to avoid maternal reaction to fetal tissues, favouring the proliferation of Th2 lymphocytes and the action of regulatory T lymphocytes.

In contrast, a decrease in the number and activity of these lymphocyte populations has been observed in preeclampsia, at the expense of increased production of Th1 and Th17 lymphocytes, involved in the production of cytokines, such as interleukin-2, IFN-γ or tumour growth factor beta (*TGF-β*), which limit immunotolerance and induce a proinflammatory state [21 - 24].

Immune component may also contribute to the increased risk of preeclampsia in primigravid women. It has been postulated that, during early pregnancy, maternal immune system develops tolerance to paternally derived fetal antigens. And, in subsequent pregnancies, memory T cells induce immunotolerance to these antigens more rapidly, reducing the risk of developing preeclampsia [25, 26].

Genetic Factors

Preeclampsia development has a genetic component, as evidenced by the increased risk of women who have first or second-degree relatives. She present preeclampsia 5 and 2 times more, respectively, than those with no family history of preeclampsia [27 - 30].

Some family segregation studies claim that individual susceptibility to develop the disease is genetically conditioned by more than 50%.

DIAGNOSIS OF HYPERTENSIVE DISORDERS OF PREGNANCY

The most frequent manifestations of preeclampsia, hypertension and proteinuria, remain for many professionals a pre-requisite for diagnosis. However, preeclampsia is a multi-organ disorder with highly variable forms of presentation (Fig. **1**).

Preeclampsia may in some cases manifest as increased capillary permeability (proteinuria, ascites, pulmonary oedema) or as impaired haemostasis with hepatic dysfunction, but without hypertension.

Patients who do not have proteinuria are the most common, as proteinuria may not be relevant even when signs and symptoms of severe preeclampsia have

CHAPTER 15

Air and Amniotic Fluid Embolism

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Abstract: Amniotic fluid embolism (*AFE*) is an uncommon pathology, whose incidence ranges from 2 to 8 per 100,000 births, depending on the country. This syndrome has four cardinal symptoms: circulatory collapse, respiratory distress, cyanosis and coma. If the patient survives cardiorespiratory failure, disseminated intravascular coagulopathy occurs, leading to incoercible bleeding and eventually death. Clinical diagnosis is based on Clark's four criteria: sudden cardiorespiratory arrest, established disseminated intravascular coagulation prior to bleeding, and all of these occurring peripartum in the absence of fever. The two main differential diagnosis syndromes are pulmonary thromboembolism and myocardial infarction. Treatment consists of cardiopulmonary support of the patient. Despite aggressive measures, such as the placement of ventricular assist devices and external oxygenation membranes, the prognosis continues to be poor. The main death cause is incoercible bleeding caused by disseminated intravascular coagulopathy.

Keywords: Amniotic Fluid Embolism, Cardiopulmonary Arrest, Coma, Cyanosis, Death, Disseminated Intravascular Coagulopathy, Embolism, Extracorporeal Membrane Oxygenation, Myocardial Infarction, Postpartum Hemorrhage, Pulmonary Embolism, Respiratory Distress Syndrome, Ventricular Assist Devices.

INTRODUCTION

Amniotic fluid embolism (*AFE*) is a disease that has a fatal prognosis, for both the mother and the fetus, because sadly it is an untreatable disease. Fortunately, its occurrence is very rare [1].

JR Meyer first published amniotic fluid embolism as a case report in the Brazilian Medical Journal in 1926. It is not found in the literature again until 1941, when Steiner and Luschbaugh described it as a sudden peripartum shock syndrome with acute pulmonary edema in eight parturients. After an autopsy, they were found to

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have fetal squamous cells in the pulmonary vascular tree. All eight women had died unexpectedly and suddenly during labor [2, 3].

Nowadays, although the morbimortality of many pregnancy conditions, such as preeclampsia, has been reduced, amniotic fluid embolism syndrome continues to be devastating. This is the result of an unknown pathogenesis. Its diagnosis is only confirmed with an autopsy having no specific treatment; its prognosis is calamitous.

INCIDENCE AND MORTALITY

Amniotic fluid embolism is a usually devastating obstetric syndrome with an incidence that is difficult to estimate due to its high mortality and the impossibility of diagnosis until autopsy.

Its incidence ranges from 2 to 8 cases per 100 000 habitats in different countries. It is one of the main causes of death directly related to labor, accounting for 5 to 15% of death cases worldwide. In Australia, it is the first cause of death and the second in the United Kingdom [4] (Table **1**).

Table 1. Amniotic fluid embolism incidence. Modified according to Rath *et al* [4].

Country	Period	Incidence (n/100.000 Birth)	Case-related Mortality	Perinatal Mortality
Australia	2001 to 2007	3.3	35%	32%
USA	1999 to 2003	7.7	21.6%	No data
UK	1991 to 2002	6	13%	No data
The Netherlands	2005 to 2009	2	20%	13.5%

Recurrence rate is unknown due to the rarity of the syndrome and its high mortality rate. According to the literature, in the few survival cases that have had a new pregnancy, it has been normal. A history of amniotic fluid embolism seems to not predispose to a new episode of this type of embolism, so no relationship with recurrence has been found.

AMNIOTIC FLUID CHARACTERISTICS

The volume of amniotic fluid increases from an average of 50 ml at 12 weeks to 1000 ml at 38 weeks, after which it begins to decrease.

At the beginning of pregnancy, amniotic fluid is a kind of dialysate of maternal serum, so the electrolytes have the same concentration found in maternal blood. As pregnancy progresses, amniotic fluid is diluted with fetal urine and it becomes

more hypotonic. Other components such as urea, creatinine and uric acid are twice as high as in maternal blood, there is no fibrinogen or bilirubin, but prostaglandins can be found [1]. Fetal materials such as skin flakes, lanugo hair and intestinal mucin can be found in amniotic fluid.

The volume of fluid needed to pass into maternal circulation to produce symptom is unknown.

RISK FACTORS

Knowing the risk factors of the disease would be very beneficial in order to be alert and prevent this syndrome with such high mortality (Table **2**).

Table 2. Amniotic fluid embolism risk factors. Modified from Fitzpatrick [5].

- Advanced maternal age
- Multiple pregnancy
- Polyhydramnios
- Placenta previa
- Placental abruption
- Labor induction
- For postnatal syndrome: cesarean section and instrumental delivery

Unfortunately, owing to such a high mortality rate, it is difficult to find associated risk factors; therefore, articles from multicenter studies should be analyzed [5].

PATHOGENESIS

Amniotic fluid enters the bloodstream through a tear in the membranes that opens the decidual vessels in an abnormal manner. This can occur in different situations such as cesarean section, ruptured uterus or placenta accreta. Some cases are devoid of any detectable underlying mechanism.

Three processes occur as a result:

1. *Pulmonary artery obstruction*: this acute obstruction produces dilatation of the right ventricle and atrium, leading to a displacement of the septum and thus hindering blood outflow from the left ventricle. This results in systolic dysfunction and finally hypotension.

2. Ventilation-perfusion mismatch produces severe anoxia, which explains cyanosis, tachypnea, altered mental function and seizures. Blood replacement by the amniotic fluid is also involved in anoxia [1].

Hypoxemia and hypotension lead to a sudden cardiovascular collapse.

CHAPTER 16

Postoperative Management of Postnatal Complications

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Abstract: The postpartum period is the time after delivery when physiological changes by the pregnancy return to the previous state. Primary postpartum haemorrhage takes place during the first 24 hours, and secondary postpartum haemorrhage occurs between 24 hours and 6 weeks after delivery.

Many disorders can occur in the immediate postpartum period, there is a considerable source of morbidity and mortality in women of reproductive age, which can be mild to severe and life-threatening.

Protocols aimed at the multidisciplinary management of postpartum haemorrhage, and together with the use of coadjuvant hemostatic agents, the activation of massive transfusion protocols in a responsible manner, and surgical management have improved the prognosis of these patients.

Keywords: Amniotic Fluid Embolism, Postpartum Complications, Postanesthetic Complications, Postpartum Haemorrhage, Postpartum Thromboembolism, Preeclampsia.

INTRODUCTION

Primary postpartum haemorrhage takes place during the first 24 hours, and secondary postpartum haemorrhage occurs between 24 hours and 6 weeks after delivery [1, 2].

It is one of the main causes of maternal morbidity and mortality worldwide and represents an approximate incidence of 4-6% of pregnancies [3]. It can be classified as mild (500-1000 ml), moderate (1001-2000 ml) and severe (greater than 2000 ml) [4]. Early identification and its etiology are essential for its imme-

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diate management and treatment. Maternal predisposing factors include, obesity (body mass index greater than 35 kg/m^2), advanced maternal age (more than 35 years), hypertensive states or diabetes mellitus [5].

The implementation of protocols aimed at the multidisciplinary management of postpartum haemorrhage has improved the prognosis of these patients. In addition to specific treatment according to the cause, the use of coadjuvant haemostatic agents, the activation of massive transfusion protocols in a responsible manner, and surgical management should be considered if necessary. An overview of obstetric management of postpartum haemorrhage is presented in Fig. (**1**).

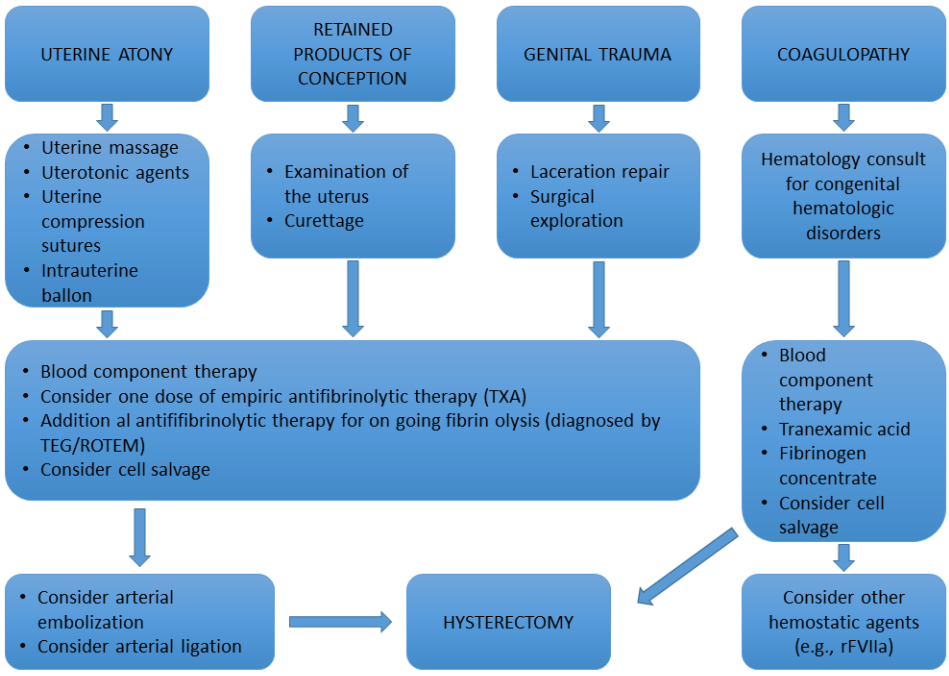

Fig (1). Obstetric management of postpartum hemorrhage.

Uterine Atony

It is the most common cause of postpartum haemorrhage (over 70% of cases). Risk factors are uterine overdistension, precipitous or prolonged labour or administration of oxytocin augmentation dosage, magnesium sulphate or halogenated anesthetic agents.

Uterotonic Treatment

The choice of the therapeutic agent to be used should be based on the comorbidities of the patient and the clinical judgment of the practitioners involved in the case [6].

Oxytocin

Oxytocin is almost universally accepted as the more effective uterotonic agent in the management and prevention of uterine atony after vaginal and operative delivery. Adverse effects include hemodynamic instability (hypotension, tachycardia, myocardial ischemia, and arrhythmias), nausea, vomiting, headache, and flushing. They are generally related to the dose and rate of administration. Routes for administration are intravenous (immediate onset of action) or intramuscular (onset between 3-7 minutes, 10 units after delivery of the placenta).

An optimal regimen may be the application of the "*Rule of Threes*" algorithm [7, 8]:

- Initial dose of 3 units given over 5 seconds after delivery of the fetus, uterine tone is assessed every 3 minutes. 3 units of oxytocin is given if inadequate tone is observed after each 3-minute interval. If a third bolus of oxytocin is ineffective, it is often appropriate to administer a second-line uterotonic agent).
- A continuous intravenous infusion is required to maintain the uterus in a contracted state and it takes approximately 20 to 30 minutes to reach a steady-state in plasma. The usual dose is 20 IU in 500 mL of crystalloid solution, with the dosage rate adjusted according to response.

Carbetocin

Carbetocin is administrated in a single dose either intravenously or intramuscularly (Table **1**) [9, 10]. Side effects include, headaches, tremor, hypotension, flushing, nausea, abdominal pain, pruritus and a feeling of warmth.

Table 1. Carbetocin usage criteria.

MATERNAL	FETAL OR PLACENTAL
- More than 4 deliveries or 2 caesarean sections in the past - Uterine surgery in the past or clinical suspicion of large uterine fibroids - Postpartum haemorrhage or uterine atony in the past - BMI > 35 kg/m^2 - Haematocrit < 35%, fibrinogen < 4 g/L or Platelet count < 100000/m^3	- Polyhydramnios - Suspected fetal macrosomia - Multiple pregnancy - Placenta previa/accreta

CHAPTER 17

Analgesia after Labor and Cesarean Section: Chronic Pain after Pregnancy

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Abstract: Chronic pain (*CP*) conditions after childbirth include persistent pain after caesarean section (*CPCS*), perineal pain after instrumental vaginal delivery, lower back pain and pelvic girdle pain. Any type of CP before or during pregnancy increases the risk of CP after delivery. Scar pain is the most recognized etiology for CPCS with a neuropathic component, although it is less frequent than in other surgeries. Reported CPCS incidence ranges from 1 to 23%. Pain intensity is moderate and decreases with time in all studies. The severity and duration of peripartum pain are the main risk factors for CP and its control is the most recommended strategy for reducing risk. Fear of fetal and neonatal adverse events means that CP is often undertreated, but after delivery, pharmacological restrictions disappear and many pain drugs are compatible with breastfeeding. Education of obstetric teams about early detection and referral to specialized consultation of women with CP is the key. In this chapter, available information in the recent literature, mainly during the last years, is presented. This chapter focuses on CP conditions after childbirth, as analgesia for labor and childbirth and immediate pain after CS and vaginal delivery are covered in other chapters of this book.

Keywords: Chronic pain, Obstetric patient, Incidence, Postpartum, Risk Factors, Treatment.

INTRODUCTION

Women´s health-related to maternity is recognized as of utmost importance due to the number of childbirths worldwide and the characteristics of the affected population: young women at an active and productive stage of their life both in their families and productive fields of all societies.

After delivery, chronic pain (*CP*) may be due to different types and causes, but

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mainly includes persistent pain after cesarean section (*CPCS*), perineal pain after vaginal delivery, lower back pain, and pelvic girdle pain. The risk to present any of these pain conditions is increased in women with any type of CP before or during pregnancy.

Pain present in the postpartum period can be related to the delivery mode, such as operational assisted vaginal delivery or cesarean section (*CS*). It may have originated during pregnancy such as low back pain, pelvic girdle pain, or headaches, or correspond to a previous history of any type of CP. Rarely, CP conditions present before pregnancy improve after childbirth, except for migraine.

Most research in postpartum CP has been dedicated to persistent or chronic pain after CS (*CPCS*). Little information is available regarding the overall prevalence of pre-existing CP disorders in pregnant women and their course during pregnancy and after delivery, although their negative impact on pregnancy and the postpartum period is recognized [1].

In addition to the scarcity of research on postpartum CP in relation to the high prevalence of childbirths, there is another issue in obstetric pain management: it is widely acknowledged that pain management teams and clinics frequently discharge or decline to manage women during pregnancy, and transition of care is frequently referred to the obstetrical team, which has inadequate training and limited time to manage patients with CP. Therefore, pregnancy and early postpartum period can turn into periods of suboptimal medical care of pre-existing pain due to both the lack of evidence-based treatment guidelines, limited formal training and expertise of the obstetric team, and the patient's acceptance of pain due to her fear of harming her child with any treatment [1].

However, obstetric pain management is considered an indicator of the quality of health organizations. Well-informed obstetrical teams and mothers must come in advance for specialized pain consultation since established post-surgical CP is difficult to treat. Awareness that CPCS is associated with other CP conditions and psychological and emotional problems should be part of the obstetric team’s education on CPSC prognosis, as well as that any other CP condition improves with the earliest reference to the pain clinic.

POSTPARTUM CHRONIC PAIN RELATED TO DELIVERY METHOD

The relevance and available pieces of evidence of postpartum CP have been comprehensively exposed in the recent reviews by Lavand´Homme P and Komatsu R et al [2, 3]. The reported incidence of persistent pain after childbirth varies depending on the method of delivery, study population, and, above all, the study design.

Chronic Pain After Cesarean Section

Persistent pain after CS and vaginal delivery is considered a type of postsurgical CP. The terms "*persistent pain*" or "*chronic pain*" are used indistinctly after surgery. It is defined as "*pain that develops or increases in intensity after a surgical procedure and persists beyond the healing process, i.e.*, at least 3 months after the initiating event", and it frequently has a neuropathic component. Some studies have considered two months of pain persistence in the definition. Postsurgical CP develops in one out of ten surgical patients and becomes an intolerable pain condition after one of every 100 operations – an incidence that has not changed over time. It represents more than 22% of pain clinic consultations [4].

The development of any postsurgical CP is due to complex neurophysiologic mechanisms, which are only partly known, and which appear and develop after the surgical wound or other severe tissue trauma. The type of surgery influences the incidence of postsurgical CP. Cesarean section is in the ninth position in frequency in the list of surgeries causing CP [2] behind thoracic, orthopedic, and abdominal surgical procedures.

The detailed description of the neurobiological changes causing the transition from acute to chronic pain after surgery and trauma is beyond the scope of this chapter. We refer the readers to excellent published reviews [4 - 8].

In Fig. (**1**), the locations in the peripheral and central nervous system where the sensitization mechanisms responsible for the transition from acute to chronic pain take place are outlined. Neural sensitization is triggered by nociceptive inputs from the inflammation and nerve lesion at the surgical location. The resulting local molecular changes lead to peripheral neural pain sensitisation in the form of primary hyperalgesia and allodynia.

Peripheral sensitization increases pain transmission, which in turn ensures maladaptive neuroplastic changes in primary sensory neurons of the dorsal root ganglia and at the spinal dorsal horn and/or higher central nervous system structures known as central sensitization.

Central sensitization reflects the interaction of multiple factors, neurotransmitters, activation of neurons and microglia at the spinal dorsal horn, called "*wind up*" that is manifested by neuro-sensitive changes in the wound surroundings. It´s called secondary hyperalgesia and allodynia. Sustained hypersensitivity to pain has developed in the peripheral and central nervous systems [2, 9, 10]. The descending inhibitory modulation of noxious signalling in the spinal pathways is compromised and maladaptive changes in the brain function and structure

CHAPTER 18

Anesthesia for Assisted Reproduction

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Abstract: Infertility is a common aspect globally affecting couples to 15%, and it is frequently increasing the need for anesthesiologists' participation in assisted reproductive techniques.

Currently, the procedures used to assist reproduction are unable to fully cover the detrimental effects of age. During anesthesia-analgesia in oocyte retrieval, the role of the anesthesiologist is to provide the patient with adequate anxiolysis, analgesia, and sedation as the key to success in the procedure. An adequate pre-anesthetic assessment is required to identify derivative diseases and take the appropriate care of each patient. Modern anesthetic techniques for oocyte retrieval include conscious sedation, general anesthesia, regional anesthesia, and other alternative techniques, such as electroacupuncture, or even a combination of these.

In this chapter, the main characteristics of these techniques will be exposed, as well as their complications and the recommendations so that anesthetic procedures are safe not only for the patient, but also for the whole process' success.

Keywords: Acupuncture, Anesthesia for Assisted Reproduction, Ambulatory Gynecological Procedures, Assisted Reproductive Technique/Technology, Infertility, Oocyte Retrieval, Sedation.

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INTRODUCTION

Infertility is a common aspect globally affecting 15% couples, as shown by the statistics of the US National Survey of Family Growth (*NSFG*) reporting an infertility prevalence of 12.1% in women between 15 and 44 years during the period between 2010 and 2015. The primary infertility rate by age group was: 9% in 15- to 34-year-old women and 16% in 35- to 44-year-old women [1].

A woman under the age of 30 has a possibility of pregnancy per cycle of between 12 to 15% and over 40 years less than 5%, causing an increase in the demand for fertility clinics' services.

Currently, the procedures used to assist reproduction are unable to fully cover the detrimental effects of age. The average success rate of reproductive techniques is 41% in 35-year-old women and 4% after the age of 42 [1, 2].

BACKGROUND

The fusion of a human egg and sperm out of the woman and the subsequent transfer of the resulting embryo back to a uterus is relatively recent. The first reports of a successful implantation and pregnancy after this procedure's implementation in humans were published during the 1970s. From that time to date, *in vitro* fertilization (*IVF*) techniques have led to new knowledge about gamete interaction and early embryonic development, as well as the advent of thousands of normal pregnancies [3].

Ovarian Hyperstimulation

Ovulation induction involves homogeneous growth of a follicular cohort to produce a higher number of good-quality oocytes, *i.e.*, achieving the growth of more than one follicle per cycle using different treatment schemes [4]. Ovarian hyperstimulation is the first step in the assisted reproductive process. Multiple follicular development can be achieved by the proper selection of specific medications. Each with its advantages and indications, and the dose based on a woman's age, ovarian reserve, previous stimulation response, and body mass index. Some examples of such medications include gonadotropins (urinary or recombinant), gonadotropin releasing hormone analogues (agonists or antagonists), and recombinant or urinary human chorionic gonadotropin (*hCG*).

Follicular Puncture

Follicular puncture for oocytes retrieval is performed 34 to 36 hours after the administration of the above-mentioned medications. Transvaginal ultrasound-guided aspiration is performed under sedation or local anesthesia, which will

depend on the location and access to the ovaries, the surgeon's experience, the patient's pain threshold, the medical site's availability, among other facts [5, 6].

Fertilization Techniques

In Vitro Fertilization

In vitro fertilization (*IVF*) is defined as an assisted reproductive technique involving extracorporeal fertilization. IVF is the most common procedure within assisted reproduction. In general terms, this technique initially consists of ovarian stimulation controlled by medications applied subcutaneously or intramuscularly. Its purpose is to obtain multiple follicles, which contain the oocytes that will then be vaginally aspirated under ultrasound guidance. These oocytes will be fertilized (*in vitro*) in the laboratory and subsequently, those which are fertilized and properly progress to embryos, either in day 3 or 5, will be transferred to the uterine cavity. This procedure generally taking about 2 weeks is called an IVF cycle [4].

IVF was initially developed for the treatment of tubular infertility, although it is currently used for many other indications [7]:

- Tubal factor.
- Endometriosis.
- Artificial insemination failure.
- Male factor (total motile sperm count (*TMSC*) less than 3 million).
- Infertility of unknown origin.
- Premature ovarian insufficiency.
- Decreased ovarian reserve.
- Cryopreservation of oocytes in cancer patients or with medical disease.
- Fertility preservation.

Intracytoplasmic Sperm Injection

It refers to a technique in which a single sperm is injected directly into the cytoplasm of a mature oocyte. This procedure is performed as part of an IVF cycle and provides an effective method to aid fertilization in men with altered semen parameters or who experienced null or low fertilization rates after conventional IVF. The efficiency of this technique has made it the most successful treatment for male infertility since 2016. The use of Intracytoplasmic Sperm Injection (*ICSI*) in male infertility increased from 84% in 2003 to 93% in 2012 [8].

ICSI is mainly indicated for the treatment of male factor infertility [9]:

CHAPTER 19

Anesthetic Management for External Cephalic Version

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Abstract: Approximately 3% to 4% of term fetuses are in breech presentation, and this is a common indication for cesarean delivery. Twenty percent of elective cesarean sections are due to breech position.

External cephalic version (*ECV*) is an obstetric maneuver that applies external pressure to the fetal posture through the maternal abdomen, to convert a breech presentation to a vertex presentation. Since the risk of adverse events after an ECV is small, the possibility of ECV should be offered in all pregnancies with breech presentation, provided that there is no contraindication.

A standardized protocol, an experienced gynecologist and adequate analgesia can facilitate the maneuver and improve the success rate, turning the ECV into a maneuver with an excellent safety profile which is an interesting option to avoid a cesarean section.

Keywords: Analgesia, Anesthesia, Breech Presentation, Breech Delivery, Cesarean Section, External Cephalic Version, Epidural Anesthesia, Inhalational Anesthesia, Intravenous Anesthesia, Neuraxial Anesthesia, Remifentanyl, Spinal Anesthesia, Tocolytics.

BACKGROUND

In recent years, WHO has highlighted in several reports the substantial increase in caesarean birth rates, exceeding the recommended rate to ensure optimal maternal and neonatal outcomes.

This situation, which occurs in both developed and developing countries, is concerning, because caesarean section is associated with an increase in maternal

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morbidity and mortality, and a higher cost of healthcare compared to vaginal delivery. Therefore, there is considerable interest in the obstetric community in identifying and implementing strategies to reduce the need for caesarean delivery.

Approximately 3% to 4% of term fetuses are in breech presentation, and this is a common indication for cesarean delivery. Twenty percent of elective cesarean sections are due to breech position.

EXTERNAL CEPHALIC VERSION

The external cephalic version (*ECV*) is a maneuver that applies external pressure to the fetal posture through the maternal abdomen, to turn a breech or transverse fetal position into a vertex presentation, allowing for a vaginal delivery.

The Term Breech Trial (Hannah, 2000) [1] concluded that elective caesarean section was associated with a lower risk of perinatal morbidity and mortality than scheduled breech delivery, thereby increasing the rate of elective caesarean sections in this type of presentation.

A subsequent study conducted in 2003 [2] concluded that, following Hannah's study, 92.5% of hospitals stopped performing vaginal breech deliveries and adopted elective caesarean section in breech presentations.

Subsequent analyses showed clear methodological deficiencies in this study, but there had already been a definite change in clinical practice in developed countries, with an increase in the rate of scheduled caesarean section in breech presentation, and with a reduction in breech delivery training and experience in obstetricians.

The obstetric experience needed to safely assist a breech delivery has disappeared and this, coupled with an effort to reduce the frequency of caesarean sections, has led to a resurgence of ECV, and anesthesia to increase its success.

Morbidity and mortality associated with elective caesarean section are three times higher compared to vaginal delivery [3]. In addition, there is a greater likelihood of caesarean sections in future pregnancies. The presence of a uterine scar increases the risk of complications such as ectopic pregnancy, placenta previa or accretion, placental abruption and uterine rupture.

The external cephalic version is a safe technique. The frequency of complications is very low. Severe complications, such as placental abruption and emergent cesarean section due to fetal distress, appear in less than 1% of procedures.

ECV is not associated with increased perinatal morbidity or mortality. Interesting in this regard, in a recent observational study [4], the authors compared perinatal outcomes among women with breech presentation at term who underwent an attempt at ECV with those who were treated expectantly. They did not find greater perinatal morbidity or mortality associated with attempted EVS compared to expectant management. In addition, although success can be difficult to predict for sure, an attempt at ECV reduces the chance of caesarean section compared to expectant follow-up.

In addition, it is an effective technique. Although the heterogeneity of studies does not allow for precise statistics, the success rate of ECV is 40-70%, managing to significantly decrease the number of caesarean sections for breech presentation from 9-16%. Spontaneous reversion to the breech position after successful ECV is less than 5%.

Currently, scientific societies recommend ECV due to the low risks to both the pregnant woman and the fetus [5 - 7].

Factors that Predict a Successful ECV

Among the factors associated with successful ECV are: the use of tocolysis, posterior insertion of the placenta, complete breech or transverse presentation, amniotic fluid index higher than 10 cm and maternal weight under 65 kg. Success rate is also higher in multiparous than in primiparous women and if the obstetrician who performs the ECV has experience in the technique.

Contraindications

The evidence on contraindications is limited, but according to the opinion of experts, there are situations that would contraindicate the ECV:

- Placenta previa or placental abruption.
- Severe oligoamnios or premature rupture of membranes.
- Fetal monitoring indicating risk of loss of fetal well-being.
- Severe fetal malformation.
- Uterine anomalies.
- Multiple gestation. In this scenario, ECV is contraindicated before delivery, but it can be performed for the extraction of the second fetus.
- Relative contraindications: maternal hypertension, maternal obesity.

CHAPTER 20

Mindfulness-Based Interventions during Pregnancy and Labour

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Abstract: During pregnancy, events occur that can negatively affect a woman's mental health, such as vaginal bleeding, concern for the health of the fetus, decreased fetal movements, ultrasound results, or fear of childbirth itself. Pregnant women must be able to cope with these stressful events, as perinatal mental health problems can have adverse consequences for both parents and babies.

Psychological disturbances in the mother during pregnancy can adversely affect the development of the fetus, leading to long-term negative effects on the health of the child. It is therefore important to identify prenatal interventions that can reduce this maternal distress, and one possible approach to address these perinatal mental health difficulties is mindfulness-based interventions.

Keywords: Acceptance-Based Coping, Adverse Events, Antidepressant Drugs, Anxiety, Behavioural Intervention, Child Health Outcomes, CBT, Cognitive Behavioural Therapy, CBT-I, Cognitive Behavioural Therapy for Insomnia, Comorbidity, Compassion, Childbirth, Depression, Emotion Regulation, Fetal Programming, Fear, Iatrogenic, Labour, Maternal Mindfulness, Maternal Anxiety, Mindfulness, Mindfulness-Based Interventions, Mindfulness-Based Cognitive Therapy, Mindfulness Yoga, Mindful Motherhood Training, MMT, Mindfulness Mom Training, Noradrenaline, Obesity, Pregnancy, Perceived Stress, Perinatal Depression, Postpartum Depression, Pain, Reuptake Inhibitors, Self-Regulation, Stress, Safety, Serotonin, Side Effects, Temperament, TAU, Treatment As Usual, Tolerability, Tricyclic.

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INTRODUCTION

One in four women will develop a perinatal mental disorder, with depression being the most frequent complication [1]. In fact, anxiety multiplies the risk of depression threefold and its presence is a factor that can maintain and/or aggravate the depressive state [2].

Pregnancy-specific anxiety and stress reflect specific emotional concepts [3 - 6]. Postpartum depression (*PPD*) is diagnosed in up to 20% of women during the first 6 months after childbirth. Depressive symptomatology (maternity or baby blues) has a prevalence of 50-85% [7], and these rates are higher when there has been depression in other periods of life or in a previous pregnancy [8].

It has been observed that this symptomatology worsens in the first postpartum week and begins to improve days later, with the exception of cases that progress to PPD. Generalized anxiety disorder (*GAD*) in the perinatal stage has a prevalence of 8.5-10.5% [9]. Similarly, anxious symptomatology in pregnant women is higher, reaching up to 39% [10]. PPD and GAD, usually diagnosed in the postpartum period, may be a consequence of depressive and anxious symptomatology already initiated during pregnancy [11].

There are few studies analysing outcomes associated with postpartum anxiety. In the mother, this condition may be associated with negative effects on factors that will establish the mother-infant relationship (*e.g.*, maternal parenting behaviours, mother-infant interaction, bonding). In the infant, there may also be negative effects (*e.g.*, crying, distress at novelty, social and physiological responses), which may increase the risk of behavioural problems and be a source of infant psychopathology [10, 12, 13].

Perinatal stress, anxiety and depression do not only affect parents. In the long term, as these disorders can also affect the mental and physical health of the child [14, 15]. ring pregnancy, stress levels or negative effects of the mother have a direct impact on the fetus, being risk factors for disorders as they grow [16, 17].

Once born, the attachment between mother and baby plays an important role in the child's cognitive and emotional development, as well as in his or her later mental health [18]. If parents are depressed or distressed, they may not be able to recognize the infant's cues, and this may result in an inappropriate attachment style. Thus, an insecure attachment style in childhood increases the risk of anxiety and behavioural disorders later in life [19].

Considering the crucial role that parental mental health plays in the future mental health of children, and considering the increasing mental health problems in today's society, we understand the importance of research in this area [13].

The Developmental Origins of Behaviour, Health, and Disease (*DOBHD*) analysed the short- and long-term effects of an individual's experiences during the perinatal period on later phenotypic variations in health and disease [20]. They found that the magnitude of these effects was clinically relevant, estimating that the risk on emotional and behavioural problems in childhood attributable to prenatal anxiety was approximately 10-15%. Thus, given the negative impact of prenatal exposure to maternal anxiety, anxious women and their infants may benefit from processes that support maternal well-being during pregnancy. Unfortunately, emotional care is often poorly monitored [21].

MENTAL DISSORDES EXPERIENCED BY PREGNANT AND POSTPARTUM WOMEN

Pregnancy and childbirth represent a time of great vulnerability during which women experience many physiological and psychosocial changes. These changes put pregnant and postpartum women at increased risk of mental health problems. This risk is higher in low- and lower-middle-income countries (*LMIC*) [22] than in high-income countries (*HIC*) [23].

The most common mental health problems in pregnant and postpartum women are anxiety, perinatal depression, and postpartum depression [24, 25]. These conditions can hinder the mother's ability to care for herself, but also for her newborn, jeopardizing the establishment of a positive bond between the mother and her baby [26].

In addition, mental illness can contribute to adverse outcomes in the child during pregnancy and in the neonatal period [27 - 29], such as a small for gestational age baby [30], lower head circumference, retarded growth, delays in child development [31], poor mother-infant interaction [32], lower neonatal test scores (*e.g*. lower APGAR) [33 - 37], erratic sleep, irritability, excessive crying, and in the medium/long term emotional and behavioural difficulties [38], negative affect in the infant [39], infant cognitive developmental problems [40], delayed motor development [41], and affective disorders, attention-deficit or hyperactivity disorder (*ADHD*) in children [42].

Maternal anxiety during pregnancy can produce, in addition to problems during childbirth, alterations in socioemotional, behavioural and early neurocognitive development, and even mental health problems in adolescence and early adulthood [20, 43, 44].

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OBSTETRIC ANESTHESIA: CLINICAL UPDATES

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