INTRODUCTION

The delivery of the infant into the arms of a conscious and pain-free mother is one of the most exciting and rewarding moments in medicine. Pain relief in labour has always been surrounded with myths and controversies. Hence, providing effective and safe analgesia during labour has remained an ongoing challenge. Historically, the era of obstetric anaesthesia began with James Young Simpson, when he administered ether to a woman with a deformed pelvis during childbirth. His concept of "etherization of labour" was strongly condemned by critics! The religious debate over the appropriateness of anaesthesia for labour continued till 1853, when John Snow administered chloroform to Britain's Queen Victoria during the birth of her eighth child, Prince Leopold ***(Snow, 2002).***

Pain in labor is multifactorial. In the first stage of labor, pain is caused by distension of the cervix and low uterine segments in combination with isometric contraction of the uterus. Pain in the second stage of labor is dominated by tissue damage in the pelvis and perineum. Labor pain is due to an activation of nociceptors partly resulting from ischemia. The impulses thus generated are conducted into the spinal cord by afferent C fibers from the cervix and lower uterine segments, and by afferent Adelta and C fibers from the pelvis, pelvic organs and perineum. Labor pain is referred to as the dermatomes T(11) and T(12) in the early stage of labor. It

spreads to the neighboring dermatomes T(10) and L(1) and eventually involves the dermatomes S(2-4) during the second stage of labor and delivery ***(Beisher, 1997).***

Modern neuraxial labor analgesia reflects a shift in obstetrical anaesthesia, thinking away from a simple focus on pain relief towards a focus on the overall quality of analgesia. The International Association for the Study of Pain (IASP) declared 2007-2008 as the "Global Year against Pain in Women - Real Women, Real Pain." The focus was to study both acute pain and chronic pain in women. Labor pain was found to be a good study model for treating acute pain. Increasing knowledge of the physiology and pharmacotherapy of pain and the development of obstetric anaesthesia as a subspecialty has improved the training in obstetric anaesthesia, leading to an overall improvement in the quality of labor pain relief ***(Lieberman, 2002).***

The experience of labor pain is different for each woman, and the different methods chosen to relieve pain depend upon the techniques available locally and the personal choice of the individual. There are different methods for labor analgesia those methods are Non pharmacological and pharmacological methods. The advantages of non-pharmacological techniques include their relative ease of administration, easy availability and minimal side-effects; however, there is little evidence to support the efficacy of many of these techniques, and some may be costly and time consuming. Pharmacological

techniques of analgesia and anesthesia include both oral and parenteral routes of administration. Parenteral administration includes inhaled nitrous oxide, as well as subcutaneous, intramuscular, and intravenous injection. Sedatives, tranquilizers, and analgesics usually are given via intramuscular injection. In some cases, the intravenous route is preferred ***(Indian J Anesth 2010).***

Neuraxial labor analgesia (epidural, spinal, or combined spinal-epidural [CSE] analgesia) currently is the most effective method of labor and delivery analgesia. The side effect profile of these techniques is acceptable to most women and obstetricians. Current research aims to decrease the incidence and severity of side effects while maintaining excellent analgesia. The ideal neuraxial analgesic technique would provide rapid onset of analgesia, effective analgesia with minimal motor block, minimal risk of maternal toxicity, minimal placental transfer and effects on the fetus, and long duration of action ***(Wong, 2009).***

Aim of the essay:

The aim of this essay is to discuss different methods to relief pain during labor.

Chapter 1:

PHYSIOLOGICAL AND ANATOMICAL CONSIDERATION OF PAIN

Pain is an unpleasant but most useful sensation developed early in evolution as a protective mechanism. In the context of labor, pain is a symptom of labor and alerts the mother to seek timely help for labor and delivery. The pain of labor gets progressively severe and it is often aggravated by anxiety, fear and ignorance.

In a civilized society, freedom from pain is one of the basic rights of a person. This is often ignored, in the developing world, when it comes to a laboring woman. The reason for this cruelty is tradition, ignorance and plain lack of resources. In the developed world, when some form of analgesia is always available, a few women are brainwashed into equating pain relief in labour with personal failure. More often than not, these women not only suffer needlessly, but also reduce their chance of a normal delivery. Unrelieved maternal pain leads to a series of metabolic changes in the mother, which may adversely affect the foetus ***(Jones and Greiss, 1998).***

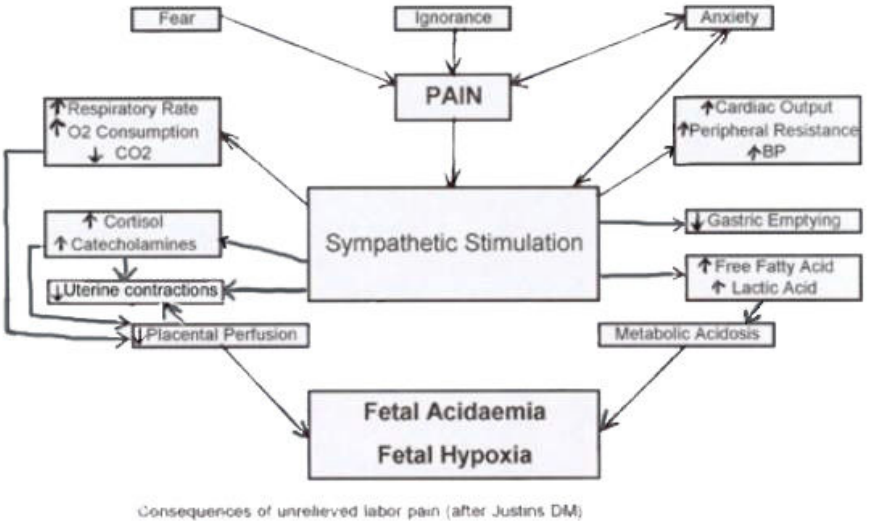

Fig. (1): Effects of pain on mother and foetus ***(Jones and Greiss, 1998).***

Labor is characterized by regular, painful uterine contractions that increase in frequency and intensity and are associated with progressive cervical effacement and dilatation. Labor has been divided into three stages. The first stage occurs from onset of cervical change to 10 cms dilatation. It can be divided into latent and accelerative phases. The latent phase can last up to 8 hrs, without the need of intervention, while the active phase is associated with a faster rate of cervical dilatation and usually begins at 2-4cms dilatation and the duration varies from 2 to 6hrs. The second stage occurs from full cervical dilatation (10 cm) to delivery of the baby. Normally the second stage lasts for 2 hrs (3 hrs with regional anaesthesia) in a primipara and 1 hr (2 hrs with regional anaesthesia) in a multipara. The third stage occurs from

delivery of the baby to separation and expulsion of placenta and the membranes ***(Faure, 1991).***

Pain pathways

***Mechanism of labor pain*:**

Perception of pain during the first stage of labor begins with nociceptive stimuli arising in the mechanical and chemoreceptors in the uterus and cervix. High threshold mechanoreceptors get stimulated due to intense pressure generated during contractions of the uterus. Myocellular injury due to repeated contractions in later stages, release bradykinin, histamine, serotonin, acetylcholine and potassium ions which activate chemical nociceptors ***(Crawford, 1995).***

a. Peripheral pathways:

First stage of labor: Pain of the first stage of labor is due to uterine contractions and stretching of the cervix. It is cramping and visceral in nature, diffuse and poorly localized. Sensations are carried through Ad and C primary afferent fibres which pass sequentially through the inferior, middle and superior hypogastric plexus, the lumbar and lower thoracic sympathetic chain and end in rami communicantes associated with T10-L1 spinal nerves. (fig.2) it is predominantly carried by the C fibres.

Second stage labor: During the late first and second stage of labor, somatic pain predominates, as a result of distension and

traction on the pelvic structures, the pelvic floor and the perineum and is carried via the pudendal nerve (table 1) through the anterior rami of S2 through S4. Unlike visceral pain of first stage, it is sharp and well localised, due mainly to less arborization and the faster conduction velocity in the sacral pathways. It is predominantly carried by the Ad fibres ***(Evron et al., 2005).***

Table (1): Labor pain: Pathways and mechanisms ***(Evron, 2005).***

Site Of Origin	Cause	Neural Pathway	Site Of Pain
Uterus and cervix	Contraction and distension of uterus and dilatation of cervix	Afferent T10 – L1 Post. Rami T10 – L1	Upper abdomen to groin, mid back and inner upper thighs (referred pain)
Peri-uterine tissue (mainly posterior)	Pressure often associated with occipito posterior position and flat sacrum	Lumbo sacral plexus L5- S1	Mid and lower back and back of thighs (referred pain)
Lower birth canal	Distension of vagina and perineum in second stage	Somatic roots S2- S4	Vulva, Vagina and Perineum (not referred)
Bladder	Over distension	Sympathetic T11-L2 Parasympathetic S2-S4	Usually suprapubic (not referred pain)
Myometrium and uterine visceral peritonium	Abruption Scar dehiscence	T10-L1	Referred Pain to site of pathology

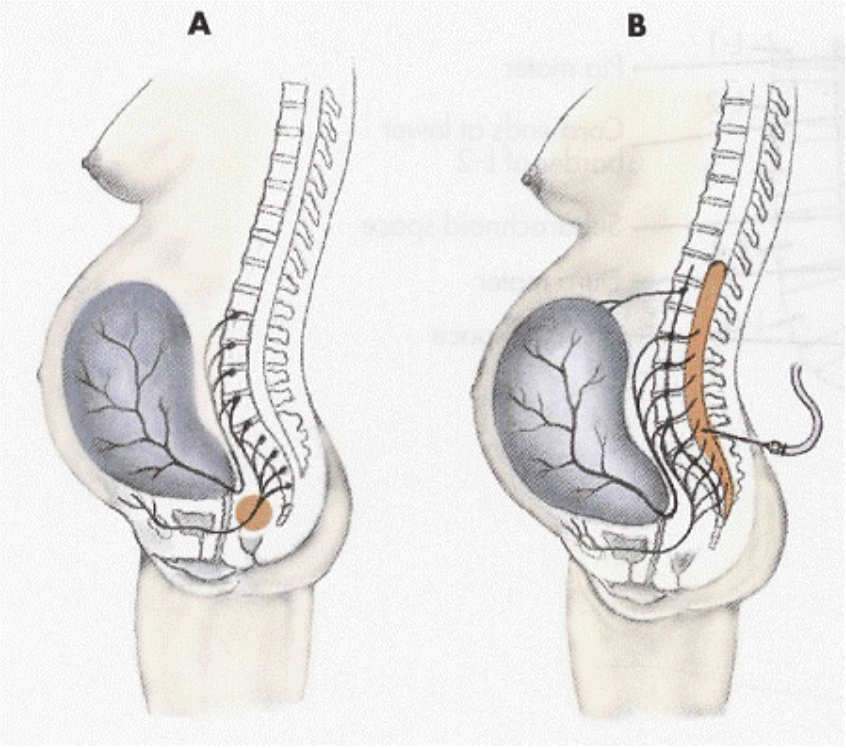

Fig. (2): Peripheral neural pathways associated with labor pain. The uterus, Including the cervix and lower uterine segment is supplied by afferents that pass from the uterus to the spinal cord by accompanying sympathetic nerves through the inferior hypogastric plexus (IHP). The hypogastric nerve, the superior hypogastric plexus (SHP). The lumbar and lower thoracic sympathetic chain. And the nerves at T10. T11. T12. and L1 ***(Bonica and Mcdonald, 1990).***

b. Central pathways:

The pathways of labor sensation travels after entry into the central nervous system includes both the ascending and the descending pathways.

Ascending pathways:

The first synapse in the pathways occurs in the dorsal grey matter of spinal cord (Rexed's Laminae I to V). Most of the primary afferent neurons synapse initially in the more superficial laminae I and II (substantia gelatinosa); locally projecting interneurons in turn synapse on the more deeply located wide dynamic range (WDR: lamina V) neurons. The WDR neurons receive synaptic excitatory input from both the large myelinated A mechanoreceptor afferents and C polymodal nociceptive afferents. The fact that all of the lamina V cells which respond to visceral high threshold afferents also respond to low threshold cutaneous afferent from an area of skin supplied by the same spinal cord segments is important. Thus the lamina V cells provide the neural basis for the phenomenon of referred pain which occurs during each uterine contraction (fig. 3) ***(Terman and Bonica, 2001).***

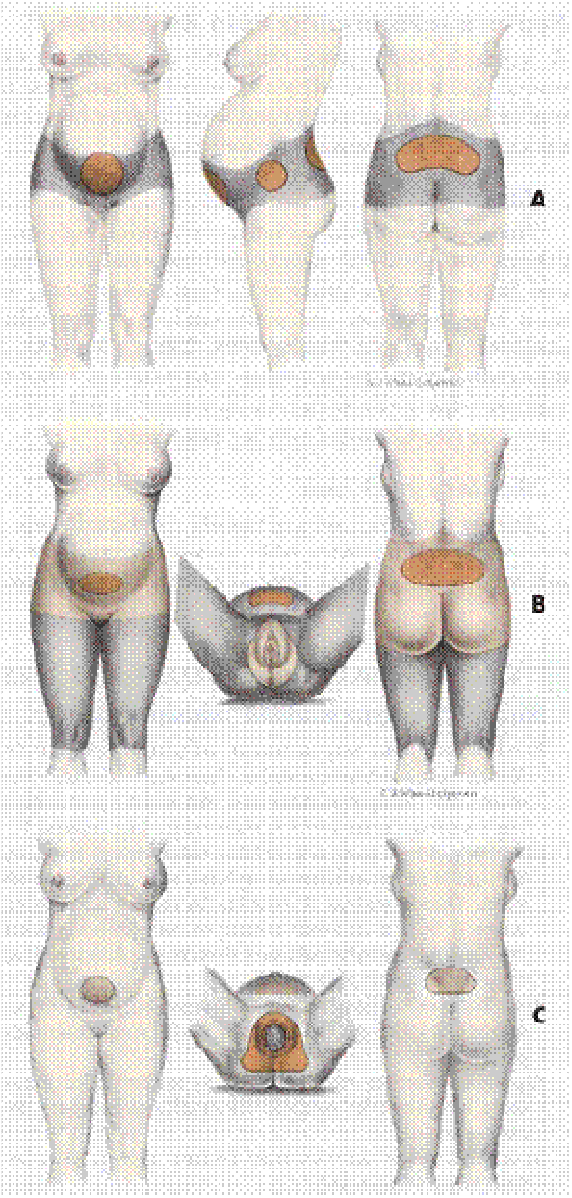

Fig. (3): Discomfort during labor. A, Distribution of labor pain during first stage. B, Distribution of labor pain during transition and early phase of second stage. C, Distribution of pain during late second stage and actual birth. *(Gray areas* indicate mild discomfort; *light-colored areas* indicate moderate discomfort; *dark-colored areas* indicate intense discomfort) ***(Palmer, 2000).***

Projections from the dorsal grey matter cross to the contralateral ventral white matter of the cord and then cephalad via the spinothalamic tract to the thalamus, brain stem, and cerebellum, (fig. 4) where spatial and temporal analysis occurs, and to the hypothalamic and limbic systems, where emotional (affective) and autonomic responses originate ***(Terman and Bonica, 2001).***

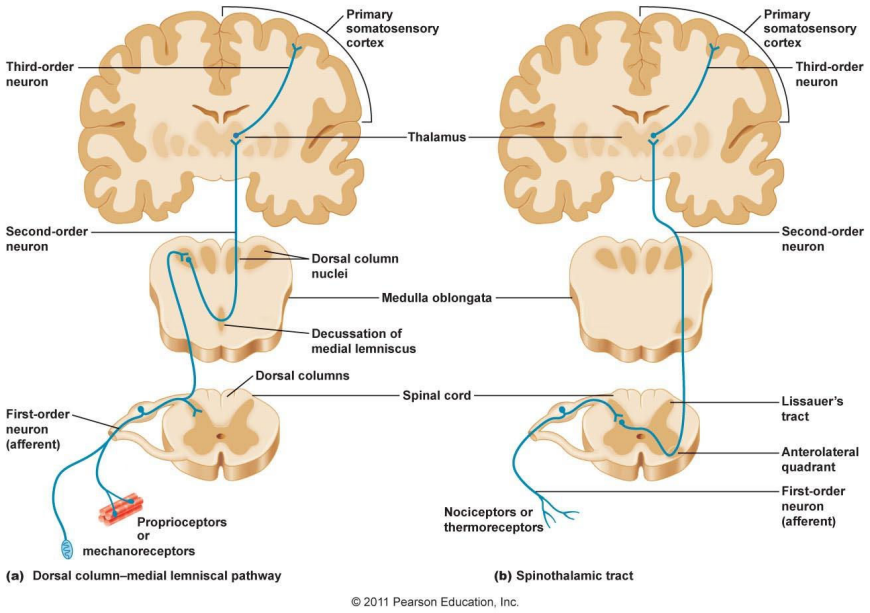

Fig. (4): Cephalad extension of labor sensory pathways.

Secondary neurons whose cell bodies lie in the dorsal gray matter of the spinal cord project via the spinothalamic tract to the thalamus. Another synapse occurs in the pathway before projection to the primary sensory cortex ***(Terman, 2001).***

Descending pathways:

These pathways originate in primary sensory cortex and project to peri aqueductal grey matter in the midbrain which further project to rostral ventral nuclei in thalamus. Projections from thalamus enter the spinal cord through dorsilateral funiculus and end in dorsal grey matter of the spinal cord (fig. 5) ***(Terman, 2001).***

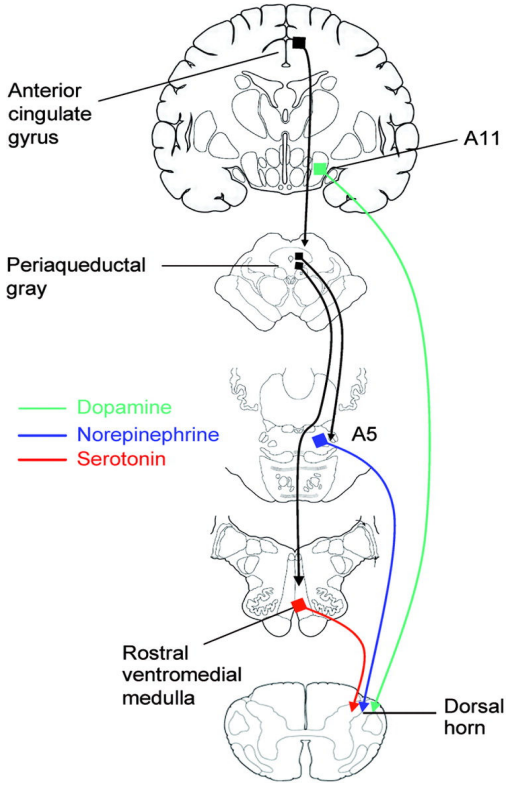

Fig. (5): Descending inhibitory pathway Neurons with cell bodies in the sensory cortex project caudally, synapsing in the periaqueductal gray matter of the midbrain. Another synapse occurs in the medulla before the pathway terminates in the gray matter of the dorsal lumbar spinal cord ***(Terman, 2001).***

Anesthetic implications physiologic and anatomic changes during pregnancy and labor:

Many physiologic and anatomic changes occur during pregnancy, labor and delivery, and the postpartum period that directly impact the administration of analgesia and anesthesia (Table 2).

Table (2): Physiologic and anatomic changes during pregnancy: anesthetic implications.

Change	Anesthetic Implications
Circulation	
Hyperdynamic: increased reliance on sympathetic nervous system	Increase in incidence and severity of hypotension after neuraxial analgesia/anesthesia
Capillary engorgement, increase in airway edema	Increased incidence of difficult airway
Aortocaval compression	More profound hypotension with parturient in the supine position after induction of neuraxial analgesia/anesthesia
Metabolism and respiration	
Increase in O_2 requirement and CO_2 production	Greater risk of desaturation after induction of general anesthesia
Decrease in FRC	Greater risk of desaturation after induction of general anesthesia
Gastrointestinal system	
Increased progestrone levels	Decreased tone of lower esophageal sphincter, increased risk of aspiration pneumonitis
Displacement of the intraabdominal esophagus into thorax	Decreased tone of the lower esophageal sphincter, increased risk of aspiration pneumonitis
Increased intraabdominal (gastric) pressure	Increased risk of aspiration pneumonitis
Endocrine	
Increased progesterone levels	Increased pain threshold
Nervous system	
Increase in ß-endorphin concentration	Increased tolerance to pain
Increased susceptibility to local anesthetics	Decrease in local anesthetic dose requirements
Anatomic changes in the spinal column	Decrease in local anesthetic dose requirements
Increased susceptibility to CNS depressants	Decrease in dose requirements for general anesthetics and adjuvants
Pharmacokinetics	
Altered volume of distribution	Change in drug pharmacokinetics
Altered protein binding of drugs	Change in drug pharmacokinetics
Increased renal blood flow	Change in drug pharmacokinetics
Altered hepatic microsomal enzyme activity	Change in drug pharmacokinetics

FRC, functional residual capacity; CNS, central nervous system.

(Bucklin et al,, 2005)

Circulation

In general, the cardiovascular system is hyperdynamic during pregnancy. However, arterial responsiveness to vasopressors is reduced in pregnancy in both *in vivo* and *in vitro* animal models. In contrast, the venous pressor response increases in pregnancy Indeed, hemodynamic stability becomes more dependent on sympathetic nervous system activity as pregnancy progresses, probably as a result of dependence on venous return. Neuraxial analgesia/anesthesia-induced sympathetic blockade in the term parturient results in a more marked decrease in blood pressure compared with that in nonpregnant control subjects ***(Hohmann et al., 1990).***

Capillary engorgement of the oral, nasal, pharyngeal, and tracheal mucosa may contribute to a markedly increased incidence of difficult endotracheal intubation in obstetric patients. Nasotracheal intubation should be avoided because of the risk of epistaxis. An overall increase in vascularity, particularly in the pelvis, may contribute to the altered rate of absorption of local anesthetics injected for nerve blocks, and increase the risk of inadvertent intravascular injection ***(Guise et al., 2010).***

AORTOCAVAL COMPRESSION

Term parturients in the supine position experience a 10–20% decrease in cardiac output secondary to aortocaval compression. Compression of the vena cava begins as early as