ANALGESIA DURING LABOUR:
A COMPARISON BETWEEN TWO METHODS:
EPIDURAL MARCaine AND INTRATHECAL
PETHIDINE IN PREGNANT SUDANESE LADIES.

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قال تعالى:

(لا أنزل الله سوءاً إلا سوءاً وَكَلَّا يَكُونُ كِتَابٌ يَكُونُ كُلُّ مَا كَتَبْتُهُ مَثْلُهُ وَكَلَّا يَأْتِيَ اللَّهِ مُجَارِمًا وَكِتَابُ اللَّهِ مَثْلُ هَذَا كُلُّمَا رَفَعَهُ عَلَى مَيْلٍ فَأَنْهَدْتَهُ فَإِنَّكَ عَلِيمٌ بِمَا يَأْتُونَ)
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DEDICATION

To all my patients who grant me their time & cooperation

To my family who gave me all the support I needed.
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ABSTRACT

This study was designed to determine the awareness of Sudanese ladies about epidural & spinal analgesia and to compare the various effects of epidural Marcaine versus intra-thecal Pethidine during labour.

The prospective experimental study which was conducted at Suba university hospital in the period from August 2003 to January 2004 approached 78 healthy ladies. Only 37 ladies agreed to participate in the study (20 were given intra-thecal Pethidine and 17 epidural Marcaine), whereas most of those who refused had no specific reasons.

Contrary to the previous studies where epidural analgesia was considered as a factor for the increasing rate of instrumental delivery, in this study there was no statistically significant difference in the mode of delivery.

The onset of analgesia among the group which received intra-thecal Pethidine was more rapid compared to epidural Marcaine, while the duration and quality of analgesia during the labour was significantly better in the latter group.

Newborn in both groups were assessed by Apgar Score at 5 minutes and none of them had a score less than seven.

Complication following intra-thecal Pethidine were mainly nausea & vomiting, in the epidural group there was no motor complication.
ملخص الأطروحة

هذه الدراسة صممت لتقرير وعي السيدات السودانيات حول فقدان الشعور بالألم خارج الأم الجافية وداخل الشوكية، ومقارنة التأثيرات المختلفة لعقار الماركين خارج الأم الجافية مقابل عقار البثديين Pethidine عند حقته.

الدراسة التجريبية أجريت في مستشفى سوبا الجامعي في الفترة من أغسطس/آب 2003 إلى يناير/كانون الثاني 2004 شملت 87 سيدة صحيحة، فقط 37 سيدة وافقت على المشاركة في الدراسة (تم تقسيمهم إلى مجموعتين، 20 سيدة تم إعطاؤهم بثديين داخل الشوكية و17 ماركين خارج الأم الجافية)، أغلب أولئك الذين رفضوا ما كان عددهُن أسباب عنيفة.

على نقيض الدراسات السابقة حيث فقدان الشعور بالألم خارج الأم الجافية اعتبار عامل للنسبة المتزايدة للولادة بواسطة الجفت أو الولادة القصرية، في هذه الدراسة ما كان هناك اختلاف هامًا بشكل إحصائي في نمط الولادة.

بداية فقدان الشعور بالألم في المجموعة التي أعطت عقار البثديين داخل الشوكية مع المجموعة التي أعطت عقار الماركين خارج الأم الجافية كان أسرع، بينما المدة ونوعية فقدان الشعور بالألم أثناء العمل كان أفضل جدًا في المجموعة الأخيرة.

المولود الجديد في كلتا المجموعتين قُبِّل نتيجة أبجر Apgar في 5 دقائق ولا أحد منهم كان على نتيجة أقل من سبعة.

الأثر الجانبية التي نتجت عن حق عقار البثديين داخل الشوكية كانت بشكل رئيسي غثيانًا وقفيًا، في المجموعة خارج الأم القاسية ما كان هناك أثار جانبية تذكر.
INTRODUCTION

Labour pain has been around as long as mankind has existed. Many methods of analgesia were known four thousand or more years ago, and the prevalence of pain during childbirth, the importance attributed to it were found in the writings of ancient Babylonians, Egyptians, Chinese and Greeks. Yet in the English speaking Western world until the eighteenth century, no analgesia was offered.

It was only when Queen Elizabeth took inhalation anaesthesia, that analgesia became fashionable and permissible in labour.\(^{1}\)

Certainly for the last 150 years many techniques have been used to provide analgesia during labour. These included psychological, pharmacological, inhalational and regional analgesia.\(^{2}\)

Many authors have contributed papers in the last decade on intrathecal narcotics. "Intrathecal narcotics offer excellent labour pain relief with manageable side-effects and without adverse obstetric outcome".\(^{2}\)

The choice of route is largely epidural/intrathecal. In each of these routes we need to consider which agent might be most effective, cost-effective, and what are the side effects to the mother and the fetus.

Nowadays Systemic analgesia has become less common, whereas the use of newer neuraxial techniques, with minimal motor blockade,
have become more popular. Low- and ultra-low-dose epidural analgesia, spinal analgesia, and combination spinal epidural analgesia have replaced the traditional systemic analgesia for labour \(^{(3)}\)

In the United States the number of parturients given intrapartum epidural analgesia is reported to be over 50 percent at many institutions. \(^{(4)}\)

In Sudan neuraxial analgesia during labour is still uncommon if not obscure. Many doctors consider analgesia in childbirth unnecessary, despite the fact that labour and delivery are considered as medical procedures. It is, however, considered "normal" that this medical procedure takes place with a significant amount of "pain." Some say that Sudanese ladies do not mind (or may even enjoy) the pain they experience during labour, claiming that it is part of their bonding to their newborns. But is that the truth… or is it illiteracy and poor resources coupled with narrow usage of such technique that have conspired to deprive Sudanese women from a safe and pain-free labour. This study aims at exploring these areas and concentrate on advances in analgesia during labour by conducting a comparison between the administration of epidural marcaine and spinal opioid (pethidine).
1.2.1 ANATOMY

1.2.1.1 ANATOMY OF THE EPIDURAL SPACE:

The epidural space surrounds the dural sac and is bounded by the posterior longitudinal ligament anteriorly, the ligamenta flava and the periosteum of the laminae posteriorly, and the pedicles of the spinal column and the intervertebral foramina containing their neural elements laterally. The space communicates freely with the paravertebral space through the intervertebral foramina. Superiorly, the space is anatomically closed at the foramen magnum where the spinal dura attaches with the endosteal dura of the cranium. Functionally, however, local anesthetics can diffuse intracranially during excessively high epidural block. Caudally, the epidural space ends at the sacral hiatus, which is closed by the sacrococcygeal ligament. The epidural space contains loose areolar connective tissue, semi liquid fat, lymphatic, arteries, an extensive plexus of veins, and the spinal nerve roots as they exit the dural sac and pass through the intervertebral foramina.

The lumbar epidural space in adults is segmented and discontinuous. This segmentation may impede the passage of an epidural catheter and promote coiling and misplacement. Contact with the pedicles also divides the posterior epidural space from the lateral epidural space.
The anteroposterior dimension of the posterior space is greatest in the lumbar region and averages 5.0 - 6.0 mm in adult. As the size of the dural sac relative to the epidural space decreases at the L4-L5 level, the posterior longitudinal ligament falls away from the anterior dura, and fat fills the anterior epidural space. The increasing amounts of epidural fat anteriorly may contribute to the long latency of epidural anesthesia typically observed in the L5 and S1 nerve roots.

The epidural venous plexus is a valveless system that communicates with the basivertebral vein, the intracranial sigmoid, occipital, and basilar venous sinuses, and the azygous system. Drugs, air, or other material injected into the epidural space can potentially reach the heart or brain directly through this route. Abdominal and thoracic veins connect with the venous plexus through the intervertebral foramina, and transmit intra abdominal and intra thoracic pressure to the epidural space. Inferiorly, the venous plexus connects with the iliac veins through the sacral venous plexus.

Chronically increased intra abdominal pressure or obstruction of the inferior vena cava (as in late trimester pregnancy) can distend the epidural venous plexus, with important implications for epidural anesthesia. This increases the risk of intravascular cannulation with an epidural catheter. It effectively decreases epidural space volume, allowing local anesthetics to distribute more widely with resulting greater degrees
of block. Exposure to greater vascular surface area also potentially increases the risk for local anesthetic toxicity due to absorption from the epidural space.\(^{(5)}\)

1.2.1.2 ANATOMY OF THE SPINAL CORD:

The spinal cord lies in the vertebral canal and is surrounded by the pia mater, a highly vascularized membrane layer that closely invests the spinal cord and brain. The outer layer is the dura mater and the inner is the pia mater. In between these two layers is the arachnoid mater, which is a delicate nonvascular membrane that is closely attached to the dura mater. Above it is continuous with the cerebral arachnoid, which loosely invests the brain.\(^{(6)}\)

The arachnoid mater actually represents the most important and active meningeal barrier, delineating the region of interest in intrathecal anaesthesia - the subarachnoid space. It is formed by two portions: a compact laminar portion that covers the internal surface of the dural sac; and a trabecular portion that extends like a spider's web around the pia mater. The arachnoid mater is not just a passive container of the cerebrospinal fluid (CSF); it also actively participates in the transport of anaesthetic agents and neurotransmitters that are involved in spinal block.\(^{(7)}\)

The cerebrospinal fluid (CSF) is the diluent for drugs delivered by a subarachnoid route. CSF volume is widely variable between individuals
as has been demonstrated with magnetic resonance imaging, with volumes of lumbosacral CSF ranging from 28 to 81 ml. This volume is significantly less in relatively obese subjects than in non-obese subjects. The decreased CSF volume that results from increased abdominal pressure, such as with obesity or pregnancy, may produce more extensive neuraxial blockade through diminished dilution of anaesthetic (8).

1.2.2. PHYSIOLOGY OF LABOUR:

Labour has been divided into three stages. The 1st stage is defined as that lasting from the start of regular uterine contractions until the complete dilatation of the cervix. It is commonly subdivided into a latent and an active phase, the later being characterized by a rapid acceleration of cervical dilatation. The 2nd stage proceeds from the 1st stage until the delivery of the fetus is complete and the 3rd stage continue until the placenta and membranes have been expelled.

Severe pain, when experienced by a woman in labour, may result in a stress response. Under conditions of stress, the body produces catecholamines: adrenaline and noradrenaline. These have effects, which may improve the body's ability to deal with the stress. Catecholamines released during labour as a result of pain or anxiety will be of little benefit to the mother or fetus under normal conditions. Circulating adrenaline can diminish the strength and synchronous of the uterine contractions. 'Discordant' labour may ensue because the cervix dilates
much more slowly. Stress also tends to make people over-breathe. Over-breathing during labour can cause excessive amounts of carbon dioxide to be breathed out resulting in an imbalance of the mother's acid-base (pH) balance. She may become alkalotic. This may reduce the delivery of oxygen to the fetus. Excessive catecholamines may also do this. When insufficient oxygen is being delivered to the fetus, it mounts a similar stress response. In the fetus, the stress response will be caused by asphyxia. We can monitor the fetus for signs of stress in a number of ways. Increases in the fetal heart rate, a characteristic pattern on the cardiotocograph trace or the appearance of fetal stool in the liquor (meconium) are examples of fetal distress \(^{(9)}\)

Any technique used during labour, which decreases the mother's stress, should be of benefit to the child.

1.2.3. PATHWAY OF LABOUR PAIN:

Labour pain has a visceral component and somatic component. Uterine contractions may result in myometric ischemia, causing the release of potassium, bradykinin, histamine and serotonin. In addition, stretching and distention of the lower segment of the uterus and the cervix stimulate mechanoreceptor. During the first stage of labour, pain primarily results from dilation of the cervix and distention of the lower uterine segment, which occurs with uterine contractions. These pain impulses are transmitted by means of afferent A-delta and C fibers, which
are visceral afferent nerves that accompany the sympathetic nerves and enter the spinal cord at T10 to L1. The visceral pain of uterine contractions is described as dull and aching, although severe, it is poorly localized by the patient. Visceral pain is transmitted by slow-conducting fibers that are easier to block than somatic nerve fibers. These pathways could be blocked successfully by administration of blockade at different levels along the pathway (spinal, epidural, paracervical …etc.

During the second stage of labour, pain results from distention of the pelvic floor, vagina, and perineum. Pain impulses are transmitted to the spinal cord by means of somatic nerve fibers that enter the spinal cord at S2 to S4. Somatic pain is transmitted by rapidly conducting fibers that are more difficult to block. The pain is sharp and well localized by the patient. (9)

1.2.4. WHY DO WE NEED ANALGESIA?

The pain of labour and delivery involves local segmental, suprasegmental and cortical stress responses. Properly administered analgesia should reduce or block these responses.

1.2.4.1 RESPIRATION:

Intrathecal /Epidural analgesia prevents transient hyperventilation
during, and hypoventilation between, uterine contractions \(^{(10)}\), so that maternal PaCO\(_2\) remains 28-32mmHg and PaO\(_2\) about 100mmHg. The incidence of hypoxaemia during first or second stage labour is 3 times greater with no analgesia \(^{(11)}\), or 1.4 times greater with intramuscular pethidine, than when epidural analgesia is provided by Marcaine 0.125% without fentanyl \(^{(12)}\).

1.2.4.2 REDUCE NEUROENDOCRINE EFFECTS OF PAIN:

The relief of pain and associated anxiety with epidural analgesia reduces maternal catecholamine \(^{(13)}\), beta-endorphins, ACTH and cortisol. Total works of labour, maternal metabolism and oxygen consumption are reduced \(^{(14)}\). Maternal and fetal acidosis is reduced though active pushing during second stage sustains some metabolic acidosis whether or not epidural analgesia is administered.

1.2.4.3 REDUCE CARDIOVASCULAR EFFECTS OF PAIN:

Epidural analgesia eliminates the increase in cardiac output, heart rate and blood pressure caused by pain. \(^{(15)}\)

1.2.4.4 UTERINE ACTIVITY:

Catecholamine alpha-receptor stimulation causes uterine hypertonus and beta-receptor stimulation decreases uterine tone and contractility. Epidural analgesia without adrenaline may reduce the effect of circulating catecholamines causing uterine uncoordinated activity or
change incoordinate uterine activity to a normal labour pattern. Epidural analgesia should be continued during the second stage of labour to shorten its duration and prevent increase in the level of catecholamines. (16).

1.2.5. OPIOID TECHNIQUES:

During labour, the ideal analgesic technique should be safe for the mother and fetus, provide flexibility in response to changing conditions, and should not interfere with the progress of labour and delivery. Further, the ideal agent would provide consistent pain relief, have a long duration of action, minimize undesirable side effects, and minimize physician involvement. (16)

No single local anesthetic is an ideal analgesic agent during labour. Disadvantages of epidural and spinal local anaesthetic techniques include hypotension, motor block, nonspecific sensory block, shivering, and the risk of cardiovascular collapse (e.g. from high spinal anaesthesia or systemic local anaesthetic toxicity) (17).

On the other hand, intrathecal administration of short-acting lipid-soluble opioids (e.g., fentanyl and sufentanil) or even pethidine has greater efficacy and results in fewer side effects than administration of a water-soluble opioid such as morphine. Limitations to the intrathecal administration of a lipid-soluble opioid for labour analgesia include a propensity for intense (although brief) pruritus, a relatively short duration
of action and an inability to produce adequate pain relief during the second stage of labour.

Two decades ago, investigators identified dense concentrations of opiate receptors in the dorsal horn of the spinal cord. The application of small doses of an opioid to these receptor sites results in a specific and profound opioid response (18). In contrast, systemic opioid administration activates multiple peripheral receptors, which results in analgesia that is tainted by the occurrence of unwanted side effects.

The introduction of intraspinal opioids appeared to fulfill the prediction made by Benjamin Rush in 1818: "A medicine would be discovered which should suspend sensibility altogether and leave irritability or powers of motion unimpaired."(19)

1.2.5.1 MECHANISM OF ACTION:

In 1979, investigators first reported dramatic pain relief after the epidural and intrathecal administration of opioids in humans (20). Intraspinal morphine administration results in long-lasting pain relief with little if any effect on voluntary motor function or sympathetic tone. Intraspinal opioid administration provides excellent analgesia under certain circumstances, but this technique does not provide anesthaesia (i.e. the complete absence of sensation). Likewise, intraspinal opioids provide effective pain relief in some but not all circumstances in obstetric patients (21).
Intraspinal opioid administration exploits the pharmacology of pain-modulating and pain-relieving systems that exist within the spinal cord. Opioids block the transmission of pain-related information by binding at presynaptic and postsynaptic receptor sites in the dorsal horn of the spinal cord (i.e. Rexed laminae I, II, and V) and in the brainstem nuclei, periventricular gray matter, medial thalamus, and perhaps components of the vagal system. The modulation of pain in the spinal cord is the result of opioid binding at several different subtypes of opioid receptors.

The effects of opioids are defined not only by their relative affinity for various receptors and the location of those receptors in the central nervous system (CNS) but also by the opioids' ability to reach those receptors. If given epidurally, opioids reach the receptor sites by penetrating the dura. Passing through the cerebrospinal fluid (CSF), and entering the superficial laminae of the dorsal horn, where the receptors are located. The movement of opioids into the CSF also accounts for the occurrence of side effects. The transmembrane movement of opioids like that of local anesthetics is modulated by the physicochemical properties of these drugs (22).

Lipid solubility is a major determinant of opioid action. Increased lipid solubility allows a drug to diffuse rapidly to its site of action which results in a more rapid onset of analgesia. For example, fentanyl is highly
lipid soluble (600 times more lipid soluble than morphine), and it has a more rapid onset of action than morphine \(^{(23)}\). However, this increased lipid solubility of fentanyl is a double-edged sword. Fentanyl also diffuses away from the site of action more quickly and thus has a shorter duration of action than morphine.

The relationships between lipid solubility and onset, potency, and duration of action are complex. For example, sufentanil which is even more lipid soluble than fentanyl, also has a rapid onset of action (possibly faster than fentanyl) but it has a longer duration of action than fentanyl when given intrathecally \(^{(24)}\).

Onset, potency, and duration also are affected by other physiochemical properties, including molecular weight, pKa, and protein binding. For instance, the lower the pKa, the greater the percentage of opioid that exists in the uncharged form (i.e., the anionic base) at a pH of 7.4. In the uncharged form, opioids penetrate the dura mater and dorsal horn more easily, which results in a more rapid onset of analgesia. The physicochemical properties of opioids determine not only their rate of absorption but also their movement within the CSF. The speed and extent of rostral spread of the opioid in the CSF determines the incidence and severity of its side effects. Hydrophilic agents (i.e. those that are not very lipid soluble) are retained in the CSF; these agents may move a great distance within the subarachnoid space before they diffuse into the lipid
tissues of the spinal cord. Hydrophilic agents (e.g. metrizamide) move from the lumbar subarachnoid space to the medulla within 30 minutes of injection (25). This suggests that relatively large quantities of hydrophilic opioids (e.g. morphine) travel freely in the CSF and gain access to the respiratory centers on the ventral surface of the medulla. The rostral spread of opioid within the subarachnoid space may then result in respiratory depression. In contrast, the more lipid-soluble agents (e.g. fentanyl, sufentanil) penetrate tissues rapidly which both limits the amount of opioid that moves cephalad and hastens the clearance of drug from the CSF (26). So, beneficial and worrisome effects of intraspinal opioids depend on:

1. The opioid receptor that is activated (i.e., mu, kappa, delta),
2. The amount of opioid administered,
3. The opioid lipid solubility, and
4. The rate of movement and clearance of the opioid in the CSF.

1.2.5.2 INTRATHECAL OPIOID:

The ideal analgesic agent for labour would provide a rapid onset of pain, have a long duration of action, minimize undesirable side effects (e.g., motor block, hypotension), preserve proprioception, and have no effect on the fetus. Of the analgesic options used clinically, intrathecal opioids perhaps come closest to achieving these goals (19).

However, some disadvantages are associated with the intrathecal
administration of opioids alone. Single doses of lipid-soluble opioids have a limited duration of action (1 to 2 hours). Additional doses require repeat dural puncture or the presence of an intrathecal catheter (both of which increase the risk of postdural puncture headache [PDPH] to unacceptable levels). In addition, intrathecal opioids alone do not provide adequate analgesia during the second stage of labour.

Early studies demonstrated that intrathecal administration of 0.5 to 2 mg of morphine reliably produce analgesia during the first stage of labour, but the analgesia was less reliable during the second stage of labour and during instrumental vaginal delivery. Intrathecal administration of these relatively large doses of morphine resulted in a high incidence of side effects, including somnolence, nausea and vomiting, pruritus, and respiratory depression. Abouleish reported a case of life-threatening respiratory depression 1 hour after delivery and 7 hours after intrathecal administration of 1 mg of hyperbaric intrathecal morphine.

In 1984, Nordberg et al demonstrated that intrathecal administration of as little as 2.5 mg of morphine resulted in high CSF concentration of the drug. The intrathecal administration of small doses of morphine has a considerable appeal with regard to avoiding side effects, especially respiratory depression. However, morphine alone has a prolonged onset and is unacceptable for many labouring women.
Intrathecal injection of a more lipid-soluble opioid results in a more rapid onset of analgesia with fewer side effects. Several studies have demonstrated the efficacy of intrathecal sufentanil and fentanyl in labouring women \(^{(30)}\). When used alone, intrathecal sufentanil or fenatanyl provides analgesia with a rapid onset (usually within 2 or 3 minutes) and duration of 70 to 100 minutes. Intrathecal administration of a lipid-soluble opioid provides analgesia in 90% to 95% of nulliparous women, when administered before 5 cm cervical dilatation.

Unfortunately, the duration of analgesia is brief (i.e., 70 to 100 minutes \(^{(31)}\). Further, the addition of morphine 0.25mg to sufentanil 10µg prolongs the duration of intrathecal sufentanil analgesia by only 20 minutes \(^{(32)}\). This problem (i.e., brief duration) may be overcome by placement of a spinal catheter, which allows repeated intrathecal injection of a lipid-soluble opioid. An epidural catheter if placed in the subarachnoid space, results in a high incidence of PDPH. Thus the unavailability of the small-gauge spinal microcatheter limits the intrathecal administration of a lipid-soluble opioid to a single injection. Typically this injection is given during the administration of combined spinal-epidural (CSE) analgesia. With this technique the patient receives the benefit of the initial intrathecal injection of a lipid-soluble opioid, an epidural catheter to allow administration of additional drug(s) is needed.

An alternative drug is meperidine. Meperidine is unique among the
opioids in that it posses weak local anesthetic properties (33) and has been used in large doses (e.g. 1 mg/kg) as the sole agent to provide spinal anaesthesia for surgical procedures (34). Intrathecal administration of meperidine (10 to 20mg) results in effective labour analgesia within 2 to 12 minutes, with a duration of 1 to 3 hours (35). Honet et al (35) compared the efficacy of intrathecal meperidine 10 mg, fentanyl 10µg, or sufentanil 5µg in 65 labouring women. These three regimens resulted in a similar onset (less than 5 minutes) and duration (80 to 100) of effective analgesia. However, the meperidine group had significantly lower pain scores after cervical dilation had progressed beyond 6 cm. As labour advances, the nature of pain becomes increasingly somatic; only meperidine functions as a spinal anaesthetic. This explains why meperidine may provide more effective analgesia during advanced labour, including the second stage. However, we recently observed that intrathecal administration of both sufentanil and a local anesthetic was superior to intrathecal meperidine alone for the relief of pain during labour (36). In Great Britain, some anaesthetists have advocated the intrathecal administration of diamorphine (heroin). This drug is unavailable for clinical use in the clinical practice. Kestin et al (37) observed that intrathecal administration of diamorphine (0.2 to 0.5 mg) provided good-to-excellent analgesia in 90% of labouring women. The mean duration of analgesia was approximately 100 minutes. However,
75% of patients had pruritus, nausea, and vomiting. In contrast, Sneyd et al (38) observed prolonged analgesia after intrathecal administration of diamorphine 2.5 mg, with a lesser incidence of side effects.

To treat the more intense pain of advanced labour, some investigators have added small doses of bupivacaine to sufentanil (39). In nonrandomized studies, patients with a cervical dilation greater than 6 cm received intrathecal sufentanil 10µg and bupivacaine 2.5mg as part of a CSE technique. All of the patients received effective analgesia, and a majority did not require additional local anaesthetic administered through the epidural catheter before delivery. When given to patients in early labour, the addition of bupivacaine 2.5 mg to sufentanil 10µg results in a higher sensory level and a greater incidence of hypotension than the administration of intrathecal sufentanil alone (40). Sia et al (40) recommended "that the dose of local anaesthetic be reduced or omitted to improve the haemodynamic profile as well as the success of maternal ambulation in labour."

1.2.5.3 PETHIDINE

This synthetic opioid agonist is approximately one tenth as potent as morphine with a slightly more rapid onset and shorter duration of action. Compared with morphine pethidine may be more effective in neuropathic pain. Pethidine has mild vagolytic and antispasmodic effects. It may produce orthostatic hypotension at therapeutic doses and has a
direct myocardial depressant effect at high doses. Norpethidine, the active metabolite, is a cerebral stimulant and excreted primarily in the urine. Spinal and epidural administration of pethidine produces analgesia by specific binding and activation of opioid receptors in the substantia gelatinosa. Once activated, the opioid receptors inhibit the release of substance P from nociceptive afferent C fibers. Unlike other opiates, pethidine has potent local anaesthetic activity and epidural/spinal analgesia is accompanied by sensory, motor and autonomic blockade\(^\text{(41)}\).

1.2.5.4 COMPLICATIONS AND SIDE-EFFECTS :

1.2.5.4.1 NEUROTOXICITY :

Clinicians should exercise caution before injecting any agent into the epidural space. The potential for irritation or outright damage to neural structures always must be considered. The anaesthesiologist should be especially careful before injecting a new agent into the subarachnoid space. The subarachnoid space is far less forgiving than the epidural space. Preservative-free morphine (which is not available in Sudan for epidural and intrathecal administration) has no deleterious effect on neural tissue\(^\text{(42)}\). There are few laboratory studies on the effect of fentanyl on neural tissue. However, this drug has been administered to a large number of patients, with no published reports on neurotoxicity. Thus it is reasonable to conclude that either epidural or intrathecal administration of modest dose of fentanyl does not cause neurotoxicity.
Few studies have evaluated the safety of other agents that have been administered into the epidural and intrathecal space. Rawal et al (43) demonstrated histological changes in sheep consistent with neurotoxicity after intrathecal administration of 7.5µg/kg of sufentanil every 6 hours for 72 hours. Smaller doses (i.e., 0.75µg/kg) resulted in only mild changes. These doses are much larger than those in clinical practice, and the clinical relevance of these observations is unclear. To our knowledge, there are no published reports of neurologic deficits after the epidural or intrathecal administration of sufentanil in humans.

12.5.4.2 EFFECTS ON PROGRESS OF LABOUR:

Some controversy remains as to epidural or spinal analgesia prolongs labour and increases the incidence of operative delivery. The cause-and-effect relationship between the use of these analgesic techniques and prolonged labour is unclear. Severe discomfort during early labour may signal abnormal labour and may predict an increase risk of fetal heart rate (FHR) abnormalities and operative delivery (44). It remains unclear whether intrathecal administration of opioid results in a less motor block than epidural administration of a more concentrated solution of local anaesthetic results in a decreased rate of operative delivery. Reynolds (45) correctly noted that only one prospective, randomized study (46) has confirmed that the decreased motor block associated with the former regimen results in a decreased rate of
instrumental vaginal delivery. However, other studies have demonstrated that intense motor block prolongs the second stage of labour and increases the incidence of instrumental vaginal delivery \(^{(47)}\). Decreased motor block is advantageous, in part because most patients prefer little motor block during labour. Further, it seems intuitive that decreased motor block should result in fewer operative delivery.

### 1.2.5.4.3 SENSORY CHANGES:

In one of the early intrathecal opioid administration during labour, Cohen et al \(^{(48)}\) observed sensory changes in women who received intrathecal sufentanil. Subsequent studies have demonstrated that these sensory changes do not result from the local anaesthetic effect of sufentanil. Sensory changes do not predict the quality or the duration of analgesia or the degree of haemodynamic change \(^{(48)}\). Further, intrathecal sufentanil does not cause a sympathectomy \(^{(49)}\). Wang et al \(^{(50)}\) have provided the best of explanation for these sensory changes. They showed that intrathecal opioids block the afferent information from A-delta to C fibers to the spinal cord, but that efferent nerve impulses are unaffected.

### 1.2.5.4.4 HYPOTENSION:

Several studies have described a decrease in blood pressure after intrathecal opioid administration in labouring women \(^{(48)}\). Initially some
investigators concluded that intrathecal opioids exerted a local anaesthetic effect, which resulted in a sympathectomy. However, subsequent studies have demonstrated that decreased blood pressure results from pain relief rather than from sympathectomy \(^{(49)}\). However, sympathetic blockade can be expected if either a local or clonidine is administered intrathecally with the opioid.

1.2.5.4.5 NAUSEA AND VOMITING :

It is difficult to determine the incidence of nausea and vomiting as a direct side effect of epidural and intrathecal administration of opioid. Other causes of nausea and vomiting in the parturient include pregnancy itself, the pain of labour, and systemic opioids, which often are administered before intrathecal or epidural opioids. The cause of nausea mediated by neuraxial opioid administration is unclear, but it may be caused by the modulation of afferent input at the area of potrema (i.e., the chemoreceptor trigger zone) or the nucleus of the tractus solitarius, which is a key relay station in the visceral sensory network \(^{(41)}\). Of interest, nausea is less common after epidural or intrathecal opioid administration during labour than after the administration of the same drugs for post cesarean analgesia. Norris et al \(^{(51)}\) noted that women who received epidural or intrathecal opioid analgesia during labour had an incidence of nausea of only 1.0\% and 2.4\%, respectively. Metoclopramide 10mg intravenously is very effective and has few significant side effects.
1.2.5.4.6 RESPIRATORY DEPRESSION:

The risk of delayed respiratory depression after intraspinal opioid administration appears to be greatest early in the course of therapy. There are no reported cases of this complication occurring later than 24 hours after administration of the initial dose of drug. Consistently impressive among predisposing risk factors in reported cases are advanced age, concomitant use of systemic opioids or other CNS depressants (52).

Delayed respiratory depression is the most feared side effect of intrathecal opioids, and its true incidence is not known. In a prospective study of 856 patients given 0.2 mg intrathecal morphine for caesarean section, 8 had respiratory depression.

1.2.5.4.7 PRURITUS:

The incidence is particularly high in obstetric patients. Itching may be generalized or localized, with the face being a common site. This side effect is seen both with opioids containing preservatives and with preservative-free preparations. Although the pruritus is probably not due to histamine release, antihistamines often provide symptomatic relief. Nalbuphine may also be of value (53).

1.2.5.4.8 URINARY RETENTION:

Urinary retention is a troublesome side effect of neuraxial opioids. The etiology may be the rapid onset of detrusor muscle relaxation that results from the sacral spinal action of opioids (54). The onset of urinary
retention appears to parallel the onset of analgesia. It is difficult to determine the magnitude of this problem during labour because parturients often require catheterization for other reasons.

1.2.5.4.9 DELAYED GASTRIC EMPTYING:

Labour may result in delayed gastric emptying. The delay may be exacerbated by opioids administration. Intravenous or intramuscular opioids administration results in delayed gastric emptying in labouring women. In contrast, clinically useful doses of epidural fentanyl have effect on gastric emptying during labour. However, intrathecal fentanyl administration seems to delay gastric emptying more than epidural fentanyl. Delayed gastric emptying may predispose a patient to nausea and vomiting. Further, it may result in a greater volume of gastric contents, which- in theory-might be problematic in patients who require induction of general anaesthesia for emergency cesarean section. (54)

12.5.4.10 POSTDURAL PUNCTURE HEADACHE:

In the past, the risk of post dural puncture headache (PDPH) limited the use of spinal techniques in obstetric patients. The use of non-cutting, pencil-point spinal needles (e.g. Whitcare, Sprotte) has substantially reduced the incidence of PDPH after intrathecal opioids administration. In a prospective but nonrandomized study, Norris et al (51) evaluated outcomes for 924 women who chose either epidural or CSE analgesia, the anaesthesiologist can use the spinal needle, which may help
prevent unintentional dural puncture with the epidural needle.

1.2.5.4.11 FETAL SIDE-EFFECTS:

Epidural and intrathecal opioids may affect the fetus in either (or both) of two ways. First, systemic absorption of the opioids is followed by transplacental transfer of the drug, which may result in a direct effect on the fetus. Second, the effects on the mother by an opioid may affect the fetus indirectly (51).

1.2.6. EPIDURAL ANALGESIA:

1.2.6.1 INTRODUCTION:

In most cases, a single-shot subarachnoid injection of local anaesthetic is not suitable for the first stage of labour. A single-shot injection has a finite duration, and multiple injections result in an increased risk of post dural puncture headache (PDPH). Alternatively, a single subarachnoid injection of an opioid may be appropriate.

Placement of a catheter in the subarachnoid space allows the anaesthesiologist to administer continuous spinal analgesia/anaesthesia by intermittent bolus injection or continuous infusion of a local anaesthetic. This technique has been described for use in patients in whom placement of an epidural catheter is problematic (e.g., in patients with morbid obesity or abnormal vertebral anatomy such as kyphoscoliosis) (47). Early reports of this technique described the use of a standard epidural catheter placed through an 18- or 19-gauge needle (55). Early clinical studies have
suggested that subarachnoid injection of 1.0 mL of 1.0% lidocaine\textsuperscript{15} or 0.5 to 1.5 mL of 0.25% bupivacaine\textsuperscript{(55)} provides satisfactory analgesia for labour. Analgesia can be maintained with intermittent injections of 1.0% lidocaine or 0.25% bupivacaine or with a continuous infusion of 0.125% bupivacaine at a rate of 1.5 mL/hr\textsuperscript{(55)}.

1.2.6.2 BUPIVACAINE (MARCAINE) :

During labour, the ideal epidural local anesthetic would provide a rapid onset of effective analgesia with minimal motor blockade, minimal risk of maternal toxicity, and negligible effect on uterine activity and uteroplacental perfusion.

Local anaesthetics block the generation and the conduction of nerve impulses, by increasing the threshold for electrical excitation in the nerve, by slowing the propagation of the nerve impulse, and by reducing the rate of rise of the action potential. In general, the progression of anaesthesia is related to the diameter, myelination, and conduction velocity of affected nerve fibers. Clinically, the order of loss of nerve function is as follows:

Pain,
Temperature,
Touch,
Proprioception, and
Skeletal muscle tone.
Systemic absorption of local anaesthetics produces effects on the cardiovascular and central nervous systems (CNS). At blood concentrations achieved with normal therapeutic doses, changes in cardiac conduction, excitability, refractoriness, contractility, and peripheral vascular resistance are minimal. However, toxic blood concentrations depress cardiac conduction and excitability, which may lead to atrioventricular block, ventricular arrhythmias, and cardiac arrest, sometimes resulting in fatalities. In addition, myocardial contractility is depressed and peripheral vasodilatation occurs, leading to decreased cardiac output and arterial blood pressure. Recent clinical reports and animal research suggest that these cardiovascular changes are more likely to occur after unintended intravascular injection of bupivacaine. Therefore, incremental dosing is necessary (test dose in epidural analgesia). Following systemic absorption, local anaesthetics can produce central nervous system stimulation, depression, or both. Apparent central stimulation is manifested as restlessness, tremors and shivering progressing to convulsions, followed by depression and coma progressing ultimately to respiratory arrest. However, the local anaesthetics have a primary depressant effect on the medulla and on higher centers. The depressed stage may occur without a prior excited state.

1.2.6.2.1 PHARMACOKINETICS:

The rate of systemic absorption of local anaesthetics is dependent
upon the total dose and concentration of drug administered, the route of administration, the vascularity of the administration site, and the presence or absence of epinephrine in the anaesthetic solution. A dilute concentration of epinephrine (1:200,000 or 5 mcgm/mL) usually reduces the rate of absorption and peak plasma concentration of marcaine, permitting the use of moderately larger total doses and sometimes prolonging the duration of action. The onset of action with marcaine is rapid and anaesthesia is long lasting. The duration of anaesthesia is significantly longer with marcaine than with any other commonly used local anaesthetic. It has also been noted that there is a period of analgesia that persists after the return of sensation, during which time the need for strong analgesics is reduced.

Local anaesthetics are bound to plasma proteins in varying degrees. Generally, the lower the plasma concentration of drug the higher the percentage of drug bound to plasma proteins. Local anaesthetics appear to cross the placenta by passive diffusion. The rate and degree of diffusion is governed by

(1) The degree of plasma protein binding,

(2) The degree of ionization, and

(3) The degree of lipid solubility.

Fetal/maternal ratios of local anaesthetics appear to be inversely related to the degree of plasma protein binding, because only the free, unbound
drug is available for placental transfer. Marcaine with a high protein binding capacity (95%) has a low fetal/maternal ratio (0.2 to 0.4). The extent of placental transfer is also determined by the degree of ionization and lipid solubility of the drug. Lipid soluble, non-ionized drugs readily enter the fetal blood from the maternal circulation.

Depending upon the route of administration, local anaesthetics are distributed to some extent to all body tissues, with high concentrations found in highly perfused organs such as the liver, lungs, heart, and brain. The elimination of drug from tissue distribution depends largely upon the ability of binding sites in the circulation to carry it to the liver where it is metabolized.

After injection of marcaine for caudal, epidural, or peripheral nerve block in man, peak levels of bupivacaine in the blood are reached in 30 to 45 minutes, followed by a decline to insignificant levels during the next three to six hours.

The half-life of marcaine (bupivacaine) in adults is 2.7 hours and in neonates 8.1 hours.

Amide-type local anaesthetics such as marcaine are metabolized primarily in the liver via conjugation with glucuronic acid. Patients with hepatic disease, especially those with severe hepatic disease, may be more susceptible to the potential toxicities of the amide-type local anaesthetics. Pipecoloxylidine is the major metabolite of marcaine.
The kidney is the main excretory organ for most local anaesthetics and their metabolites. Only 6% of bupivacaine is excreted unchanged in the urine.

When administered in recommended doses and concentrations, marcaine does not ordinarily produce irritation or tissue damage and does not cause methemoglobinemia.

**1.2.6.2.2 INDICATIONS AND USAGE:**

Marcaine is indicated for the production of local or regional anaesthesia or analgesia for surgery, dental and oral surgery procedures, diagnostic and therapeutic procedures, and for obstetrical procedures. Only the 0.25% and 0.5% concentrations are indicated for obstetrical anaesthesia.

Experience with non-obstetrical surgical procedures in pregnant patients is not sufficient to recommend use of 0.75% concentration of marcaine in these patients.

Marcaine is not recommended for intravenous regional anaesthesia (Bier Block). (56)

**1.2.6.3 COMPLICATIONS**

**1.2.6.3.1. HYPOTENTION:**

With the onset of sympathetic blockade, peripheral vasodilatation and increased venous capacitance result in decreased venous return to the heart, which may result in decreased maternal blood pressure and cardiac
output. Hypotension often is defined as a 20% to 30% decrease in systolic blood pressure (when compared with baseline) or a systolic blood pressure of less than 100 mm Hg. Without prophylactic hydration, epidural administration of 4 to 6 mL of 0.5% bupivacaine results in hypotension in approximately 17% of labouring women. Modest hypotension rarely results in adverse consequences in young, non-pregnant patients. However, during pregnancy, uteroplacental perfusion depends on the maintenance of normal maternal blood pressure. Uncorrected hypotension results in decreased uteroplacental perfusion. If hypotension is severe and prolonged, hypoxia and acidosis will develop in the fetus.

The prevention of hypotension includes volume expansion before the induction of epidural analgesia and avoidance of aortocaval compression. Treatment includes

- Administration of additional intravenous crystalloid,
- Placement of the mother in the full lateral position, and
- Administration of supplemental oxygen.

If these measures do not result in prompt restoration of blood pressure or if hypotension is severe, the anaesthesiologist should give 5 to 10 mg of ephedrine intravenously. The FHR is monitored continuously. Ephedrine crosses the placenta and may cause an increase in FHR and an increase in FHR variability (e.g., salutatory
1.2.6.3.2. INADEQUATE ANALGESIA:

The failure rate for epidural analgesia ranges from 1.5% to 5.0% depending on the skill of the anaesthesiologist (55). Successful location of the epidural space is not always possible, and satisfactory analgesia does not always occur, even when the epidural space has been identified correctly. Patient factors [e.g., obesity, abnormal lumbar spine anatomy, depth of the epidural space] (55) increase the likelihood of an unsatisfactory result. Unfortunately, failure to provide adequate analgesia not only results in a dissatisfying experience for the patient but also may result in litigation (59). The risk of failed anaesthesia and the potential need to place a second epidural catheter should be discussed with the patient during the preanaesthetic evaluation, before placement of the first epidural catheter.

Typically, small doses of local anaesthetic are used for epidural analgesia during labour. Thus the resulting block may be asymmetric or have missed segments. Crawford (59) observed this complication in 15.4% of patients who received 8 mL of 0.25% bupivacaine. Maternal position has only a small effect on the development of an asymmetric block. Marx gave 10 mL of 1.5% lidocaine epidurally to pregnant women who were in the lateral position: they observed only a slightly greater spread (two to three spinal segments) of anaesthesia on the dependent side (57). Anatomic
barriers e.g., a longitudinal connective tissue band between the dura and ligamentum flavum) or placement of the catheter tip in the anterior epidural space or paravertebral space may explain some cases of single nerve root, unilateral or asymmetric block\(^{(5)}\).

If epidural analgesia during labour is unilateral or asymmetric, administration of a large volume of a dilute solution (e.g. 10 mL of 0.125% bupivacaine) may result in a symmetric block. Moreover, withdrawal of the catheter 0.5 to 1 cm followed by the administration of additional local anaesthetic may result in a more satisfactory spread of local anaesthetic.

Pain often becomes more intense as labour progresses. An epidural block that was adequate at 4 cm cervical dilation may not be adequate at 8 cm cervical dilation. The anaesthesiologist should be aware of the progress of the patient's labour when assessing an inadequate block. The patient may need a larger dose of local anaesthetic. A 5 to 10 mL bolus of 0.125% or 0.25% bupivacaine often is adequate. The anaesthesiologist should give a test dose to exclude intravenous migration of the catheter. The response to the bolus dose should be assessed and the catheter should be replaced (with the patient's consent) if no block is obtained\(^{(4)}\).

1.2.6.3.3. INTRAVASCULAR INJECTION OF LOCAL
ANAESTHETIC:

The incidence of fatal systemic toxic reactions to local anaesthetics apparently has declined since 1984 \(^{60}\). Nonetheless, systemic toxicity remains a serious potential complication during administration of epidural analgesia in obstetric patients. Leighton \(^{60}\) noted that maternal convulsions (as a result of the unintentional intravascular injection of local anaesthetic) were the single most common untoward event in obstetric anaesthesia-related malpractice claims. Convulsions resulted in serious damage to the mother or newborn in 74% of cases.

Intravenous injection of a large dose of local anaesthetic causes CNS symptoms (e.g. restlessness, dizziness, tinnitus, difficulty speaking, seizures, loss of consciousness).

Cardiovascular effects may progress from increased blood pressure (as a result of sympathetic stimulation) to bradycardia, depressed ventricular function, and ventricular tachycardia and fibrillation. Marcaine cardiotoxicity may be fatal in pregnant women \(^{61}\). Factors that increase the risk of local anaesthetic toxicity in pregnant women include

1. The frequent use of bupivacaine in this population;
2. A decreased maternal concentration of plasma proteins (e.g. alpha-1-acid glycoprotein) which results in a higher free concentration of local anaesthetic
(3) More frequent cannulation of an epidural vein in obstetric patients.

(4) The rapid onset of hypoxemia during hypoventilation or apnea; and

(5) The technical difficulty associated with administration of effective cardiopulmonary resuscitation in pregnant women.\(^{(61)}\)

1.2.6.3.4. UNINTENTIONAL DURAL PUNCTURE:

The incidence of unintentional dural puncture during attempted identification of the epidural space ranges from less than 1.0% to 7.6% in obstetric patients\(^{(59)}\). The incidence depends in part on the skill of the anaesthesiologist. Crawford\(^{(59)}\) observed an incidence of 13% if the anaesthesiologist had performed fewer than 10 epidural blocks. 6% if the anaesthesiologist had performed 10 to 49 blocks. 2% if the anaesthesiologist had performed 60 blocks, and 1.2% if the anaesthesiologist had performed more than 100 blocks. Dural puncture may be detected at the time of insertion of the epidural needle or after placement of the catheter. If dural puncture is detected with the epidural needle, the anaesthesiologist can remove the needle and place an epidural catheter at another interspace. Local anaesthetic (injected epidurally) may pass through the dural puncture site into the subarachnoid space, which will result in an unexpected high block\(^{(60)}\). One advantage to the placement of an epidural catheter after unintentional dural puncture is that
saline or blood can be injected through the epidural catheter to prevent a PDPH. However, some anaesthesiologists question the wisdom of performing a prophylactic epidural blood patch through an epidural catheter placed earlier during labour.

If dural puncture is not recognized until CSF is aspirated from the catheter or if administration of the test dose results in spinal anaesthesia, the anaesthesiologist has two options:

1. Placement of an epidural catheter at an alternative interspace and
2. Administration of continuous spinal anaesthesia through the existing catheter (not recommended).

1.2.6.3.5. UNEXPECTED HIGH BLOCK:

An unexpected high level of anaesthesia may result from one of two situations. First, a high (or total) spinal block results after unintentional placement of the catheter in either the subarachnoid or subdural space, followed by injection of an epidural dose of local anaesthetic through that catheter. Second, the epidural catheter may migrate into the subarachnoid or subdural space during the course of labour and delivery. Crawford (59) reported six cases of high or total spinal block in a series of nearly 27,000 cases of lumbar epidural anaesthesia administered during labour (an incidence of approximately 1 in 4500). Paech et al (61) reported eight cases of unexpectedly high block in a series of 10,995 epidural blocks in obstetric patients (an incidence of
approximately 1 in 1400). Two patients required intubation and mechanical ventilation.

Aspiration alone is an inadequate method of excluding sub-arachnoid placement of the catheter \(^{(62)}\). Administration of an appropriate test dose and careful assessment of the patient's response to the test dose should minimize the chance of unintentional injection of a large dose of local anaesthetic into the subarachnoid space \(^{(63)}\).

High or total spinal anaesthesia results in hypotension, dyspnoea, the inability to speak, and loss of consciousness. Evidence of spinal anaesthesia may be apparent shortly after intrathecal injection of a local anaesthetic but the maximal spread may not be evident for several minutes. This underscores the need for the anaesthesiologist to carefully assess the effects of both the test and therapeutic doses on the mother and fetus \(^{(62)}\). If total spinal anaesthesia should occur, the anaesthesiologist must be prepared to maintain oxygenation ventilation, and circulation. Immediate management includes

- Avoidance of aorto caval compression,
- Ventilation with 100% oxygen,
- Endotracheal intubation, and
- Administration of intravenous fluids and ephedrine to support the blood pressure as needed.

The FHR should be monitored continuously.
A high block also may result from subdural injection of a local anaesthetic. The subdural space is the potential space between the dura mater and the arachnoid mater. A retrospective review of 2182 epidural catheters placed in non obstetric patients demonstrated that the clinical signs of subdural catheter placement occurred in approximately 0.82% of patients. Subdural injection of local anaesthetic typically results in an unexpectedly high (but patchy) block with an onset time that is intermediate between spinal and epidural anaesthesia (i.e., 10 to 20 minutes). Cranial spread is more extensive than caudal spread of the local anaesthetic; thus sacral analgesia typically is absent. The block may involve the cranial nerves. (The subdural space, unlike the epidural space, extends intracranially.) Thus apnea and unconsciousness can occur during a subdural block.

A subdural block results in less intense motor block than that which occurs with high or total spinal anaesthesia. This may reflect the limited spread of the local anaesthetic within the subdural space, which helps spare the anterior motor fibers. Subdural block results in less severe hypotension than that which occurs with high or total spinal anaesthesia, most likely because subdural injection results in a slower onset of anaesthesia. The unpredictable spread of local anaesthetic, the delayed onset of maximal spread (compared with spinal anaesthesia), the patchy nature of the block, and the sacral sparing make it difficult to use a
subdural catheter safely during labour and delivery. If it becomes suspected that a catheter is positioned within the subdural space, replace it with an epidural catheter.

An unexpected high block may result from migration of an epidural catheter into the subdural or subarachnoid space (5). It is unclear how a soft epidural catheter can penetrate the dura. Disposable epidural needles are sharp, and insertion of the needle into the epidural space may result in an unrecognized nick in the dura, which may represent a site for delayed migration of the catheter into the subdural or subarachnoid space.

1.2.6.3.6. EXTENSIVE MOTOR BLOCK:

Clinically significant motor block may occur after repeated bolus doses (67) or after many hours of a continuous infusion (68) of epidural bupivacaine. Administration of bupivacaine with epinephrine results in a greater likelihood of dense motor block than administration of bupivacaine alone (67). Extensive motor block often is bothersome for the patient (68) and it may impair maternal expulsive efforts during the second stage of labour and increase the likelihood of instrumental vaginal delivery. Some obstetricians argue that pelvic floor relaxation prevents internal rotation of the fetal head and increases the likelihood of an abnormal position of the vertex at delivery. Further, some anaesthesiologists contend that motor block increases the likelihood that the mother will assume an unnatural position, which may increase the risk
of postpartum back pain (68).

If intense motor blockade develops during the continuous epidural infusion of a local anaesthetic, the infusion can be discontinued for a short period (e.g., 30 minutes). Subsequently, the infusion can be restarted at a reduced rate or with a more dilute solution of local anaesthetic. If this results in inadequate analgesia, an opioid may be added to the solution of local anaesthetic.

1.2.6.3.7. URINARY RETENTION:

Urinary retention may occur during labour, and it may be difficult for a labouring woman to feel the urge to void or to coordinate urination during administration of epidural analgesia. Nurses and physicians should observe for evidence of a distended bladder, especially if the patient complains of suprapubic pain during a contraction. The patient's inability to void and bladder distention should prompt catheterization to empty the bladder (69).

Urinary retention also may occur after vaginal delivery, with or without epidural analgesia (70). Some degree of bladder dysfunction was observed in 14.2% of women who had a normal delivery and in 37.5% of women who underwent instrumental vaginal delivery, (both groups were not offered epidural analgesia). Crawford (59) evaluated the incidence of urinary retention in a population of women who had received epidural analgesia for labour and delivery by saying that:
This complication was observed in 6.8% of women who had undergone spontaneous vaginal delivery and in 17.3% of women who had undergone instrumental vaginal delivery. A more recent study of postpartum urinary retention in a large series of patients (n = 3364) noted a lower incidence of this complication (0.9%) (71). Although the use of epidural analgesia was not randomized, a higher incidence of urinary retention was found among those women who received epidural analgesia (2.7%) compared with those who did not (0.1%).

The role of epidural analgesia in the etiology of postpartum urinary retention is unclear. Obstetric factors (e.g., long labour, edema, instrumental delivery, perineal trauma, hematoma, pain) may predispose women to difficulty in voiding. Some of these factors also are indications for the use of epidural analgesia, which may result in selection bias. Dense or prolonged epidural anaesthesia suppresses the urge to void. Postpartum patients should be observed for urinary retention. A prolonged inability to void mandates catheterization of the bladder to prevent over distention.

1.2.6.3.8. UNEXPECTED PROLONG BLOCK:

Rarely, the duration of epidural anaesthesia exceeds the time expected. Most cases of unexpected prolonged block follow the epidural administration of a high concentration of local anaesthetic with epinephrine (72). Abnormal neurological findings after the administration
of epidural anaesthesia should prompt the anaesthesiologist to look for evidence of peripheral nerve injury or an epidural hematoma or abscess. Factors that argue against the presence of an epidural hematoma or abscess include

(1) The absence of back pain.

(2) A unilateral block, and

(3) Regression (rather than progression) of the symptoms.

Peripheral nerve injuries typically result in a neurological deficit in the distribution of a specific peripheral nerve. Neurological or neurosurgical consultation should be obtained if there is any question about the etiology of a prolonged block. Avoiding the use of a high concentration of local anaesthetic should help minimize the incidence of this side effect during and after labour and vaginal delivery.

1.2.6.3.9. BACK PAIN:

Back pain is a common complaint during pregnancy and the puerperium. Back pain often results from the exaggerated lumbar lordosis of pregnancy. However, other factors may predispose a woman to backache after delivery. (70) Observed back pain in 40% of women who had had a spontaneous vaginal delivery and 25% of women who had undergone instrumental vaginal delivery all without epidural analgesia. Other studies have reported the occurrence of postpartum backache in 3% to 45% of women who received epidural analgesia for labour and
delivery (59). Factors proposed to increase the risk of postpartum backache after administration of epidural analgesia include

(1) The use of a large needle.

(2) Supraspinous ligament hematoma.

(3) Difficult identification of the epidural space.

(4) Prolonged assumption of an unnatural position during labour and delivery, and

(5) Sacroiliac strain as a result of moving the lower extremities before resolution of anaesthesia (72).

MacAnthur et al. (72) has suggested that the administration of epidural analgesia increases the risk of postpartum back pain. Data collected from a retrospective review of 11,701 case records and patient questionnaires (mailed 1 to 9 years after delivery) demonstrated a significant association between the use of intrapartum epidural analgesia and persistent, postpartum back pain (epidural analgesia 19%. no epidural analgesia 11%). The excess back pain associated with epidural analgesia was unaffected by the mode of delivery, but an increased incidence of back pain occurred only among those women who had experienced labour. The authors suggested the following:

The backache is not solely a consequence of epidural anaesthesia but is probably due to a combination of muscular relaxation and postural stress in labour. The problem now is to determine in precise detail the
mechanisms that result in backache and to refine the management of epidural anaesthesia in labour.

This retrospective study suffers not only from patient recall bias (i.e., patients with a problem are much more likely to complete and return the questionnaire) but also from selection bias in the epidural and nonepidural groups. Patients who select epidural analgesia for labour may have obstetric, orthopedic, social, or other unidentified factors that predispose them to postpartum back pain. Further, details of anaesthetic management were not described in either article. If large doses of a concentrated solution of local anaesthetic were used, motor block and analgesia may have allowed women to adopt positions that predisposed them to back strain.

In an attempt to assess anaesthetic factors that might contribute to postpartum backache. Russell et al. \(^{(73)}\) randomly assigned labouring women requesting epidural analgesia to receive bupivacaine alone or bupivacaine plus an opioid. Despite the expected differences in motor block, the incidence of backache did not differ between the two anaesthetic groups (bupivacaine alone 39%, bupivacaine plus opioid 30%). Further, the incidence of backache in both epidural groups was similar to that in a nonrandomized control group of women who laboured without epidural analgesia (31%) \(^{(73)}\). Antepartum backache predicted the occurrence of postpartum backache.
1.2.6.4 WHY DO STUDIES GIVE CONFLICT ABOUT EPIDURAL ANALGESIA DURING LABOUR?

The perception that epidural analgesia prolongs labour began in the 1950's and was based on a few retrospective, biased, uncontrolled studies of outmoded anaesthetic techniques. The studies most often quoted examine the effect of caudal, not lumbar epidural block on labour\(^{(74,75)}\).

The results these authors report actually show little or no effect of regional anaesthesia on the progress of labour. In one study, Friedman and Sachtleben found it "rather odd" that their data did not reflect any slowing of cervical dilation with "premature" (< 7 cm) caudal anaesthesia \(^{(74)}\). In a subsequent study, they could attribute only 5.9% of cases of prolonged latent labour to caudal anaesthesia \(^{(75)}\). These conclusions have been exaggerated with time. In their follow-up study, Friedman and Sachtleben claimed that their initial study documented "the sensitivity of early labour to major regional-conduction anaesthesia" \(^{(75)}\).

Other different study designs often yield different results. Two prospective, randomized trials comparing epidural block and parenteral meperidine found that epidural local anaesthetics did not prolong labour \(^{(76,77)}\).

Two other prospective, randomized studies found no deleterious effect of induction of epidural block in early labour (< 5 cm cervical dilation) compared to more advanced labour \(^{(78,79)}\).
Other studies have shown an association between epidural analgesia and cesarean delivery \(^{(80, 81, 82, 83, 84, 85)}\). Three of these studies are nonrandomized \(^{(80, 81, 85)}\). Thus, they cannot control for selection bias.

Thus, when considering the effects of epidural analgesia on the progress of labour, it is important to note the status of labour before induction of epidural block. Women with abnormal, slow, painful labours are the very women who request epidural analgesia.

Study design, investigator bias, and anaesthetic technique all contribute to the "harmful" effects of epidural analgesia on the progress and outcome of labour. However, most of the purported harmful effects of labour epidural analgesia result from patient factors (women having abnormal labours are more likely to request epidural block) and obstetrical decisions, and not from the analgesic technique itself.

### 1.2.7. NEW TECHNIQUES FOR LABOUR ANALGESIA:

#### 1.2.7.1 COMBINED SPINAL–EPIDURAL ANALGESIA:

Combined Spinal-epidural (CSE) analgesia has gained increasing popularity during the last several years. With this technique, the anaesthesiologist first places an epidural needle in the epidural space. A spinal needle is then introduced into the subarachnoid space. An opioid is injected into the subarachnoid space, the spinal needle is withdrawn, and an epidural catheter is placed. The epidural catheter can be used later or an infusion can be started immediately \(^{(39)}\).
**1.2.7.2 PATIENT-CONTROLLED EPIDURAL ANALGESIA:**

In most cases, either an anaesthesiologist or a certified registered nurse anaesthetist manages the continuous epidural infusion of the local anaesthetic, with or without an opioid. Ideally, the anaesthesia care provider will titrate the epidural infusion to the needs of each individual patient. Pain varies greatly among individuals and during the course of labour and delivery in an individual patient. Some women want to participate more actively in all aspects of their intrapartum care, including the provision of pain relief during labour. Patient-controlled epidural analgesia (PCEA) allows the parturient to titrate the delivery of analgesic drug to minimize periods of either inadequate or excessive analgesia\(^{(39)}\).

**1.2.7.3 CONTINUOUS SPINAL ANALGESIA:**

Continuous spinal analgesia is an old technique that has several advantages over single-shot spinal analgesia. The continuous spinal technique allows greater control over the dose of drug and duration of analgesia. Historically, the major disadvantage in obstetric patients was the high incidence of PDPH, which results from the use of epidural needle to place a large-gauge catheter. The renewed interest in intrathecal opioid administration during labour has resulted in a resurgence of interest in the use of continuous spinal analgesia in obstetric patients. In 1989, the introduction of spinal microcatheters (i.e. 28- and 32-gauge) appeared to be a boon for continuous spinal analgesia. Theses
microcatheter allowed the anaesthesiologist to provide continuous spinal analgesia with a very low risk of (postdural puncture headache) PDPH \(^{(39)}\).
OBJECTIVES

GENERAL OBJECTIVE:

To determine the awareness of Sudanese ladies about Epidural/Spinal analgesia during labour.

SPECIFIC OBJECTIVES:

To compare between the epidural Marcaine and spinal Pethidine in the following fields:

1- Duration of analgesia
2- Quality of analgesia they provided in the first and second stage of labour
3- Their effect on the duration of labour and the mode of delivery
4- Their effect on the Apgar score of the neonate
5- The complications (incidence and severity)

METHODOLOGY
2.1-STUDY AREA:

The study was conducted at Soba University Hospital, in the period from August 2003 to January 2004.

2.2-STUDY DESIGN:

This is a prospective experimental study in which the effects of the two methods of analgesia during labour (i.e. spinal Pethidine and epidural Marcaine) were compared.

2.3-POPULATION AND STUDY SAMPLE:

78 healthy parturients were approached; 37 healthy women, with normal pregnancies agreed to participate and were included in the study. Patients were randomized into two groups, 20 parturients received (20 mg) pethidine intrathecally and 17 parturients received epidural marcaine

Criteria of exclusion:

Patient refusal

Pregnancies with:

1- Complications; PIH, DM and other systemic diseases.

2- Previous scar,

3- Based indications for Caesarian section,

4- Malpresentation,

5- Contraindications for regional analgesia,

Multiple pregnancy.
Preterm labour.

Cervical dilatation more than 8cm or less than 4 cm.

Women who received other medication for analgesia during labour.

2.4. DATA COLLECTION:

Data was collected by a guided questionnaire and clinical observation.

2.5. DATA ANALYSIS AND PRESENTATION:

The data collected was analyzed using a master sheet and excel computer programme and SPSS. The data were then presented in figures and table. Hypotheses were tested and 0.05 probability level was predetermined as the level of significance.

STUDY PROCEDURE:

Preparation:

With appropriate staff and appropriate privileges in facilities where resuscitation equipment and drugs are immediately available.

The patient is examined and the maternal and fetal status and progress of labour have been evaluated (as soon as the diagnosis of active labour has been established).

Also the patients, vital signs (blood pressure, pulse, respiratory
rate, FHR) should be documented and the patient should be asked to relate any symptoms of pruritus, nausea, or vomiting. Each patient should also complete a baseline 100-mm visual analogue pain scale (VAS), with 0 representing no pain and 100 being the worst possible pain. (The VAS scale was modified to a verbal scale if the patient was in severe pain or could not understand the VAS score).

Illustration to the pregnant lady, the advantage of analgesia during labour and the possible risks and complications associated with (spinal pethidine or epidural marcaine) and consent to the procedure (verbal or in writing) were taken.

The parturient empty her bladder before the analgesia, and at full dilatation the midwife inserts a catheter before the delivery of the fetus.

An intravenous line is secured (using 18-20 gauges I/V cannula) and 500 ml normal saline is infused pre-anesthesia.

**STEPS OF EPIDURAL ANALGESIA:**

**For induction of epidural analgesia:**

The preparation (as above) was checked before use.

The protocol of failed epidural and the protocol of management of
local anesthetic toxicity should be available.

10ml of Marcaine 0.25 % (by diluting 5 ml of 0.5 mg Marcaine with normal saline) is prepared.

L3- L4 intervertebral space is identified and skin is infiltrated in this area using lidocaine 2 % (3 ml).

A sterile, 18 gauge (epidural) touhy needle with its introducer is slowly pointing in a slightly cephalad direction until identification of epidural space by loss of resistance.

Epidural catheter is introduced.

The test dose of 3ml of 5% lidocain with 1: 200 000 epinephrine is injected and wait for three minutes and if negative test dose.

The 10 ml of 0.25% marcaine is slowly injected with the observation for any numbness in the lower limbs or in the lips of the parturient by asking the parturient.

The patient is placed in the lateral position to prevent aortocaval compression.

**STEPS OF ADMINISTRATION OF SPINAL PETHIDINE:**

**For induction of spinal analgesia:**

The preparation (as above) was checked before use.

20 mg of pethidine in 2 ml saline is prepared. (By diluting 1 ml of 50 mg pethidine with 4 ml normal saline).

L3- L4 interspace is identified and infiltration of skin in this area
using lidocaine 2%(3 ml) is done.

A sterile disposable 22-guaze spinal needle is introduced through L3-L4 until the dura is punctured, stylette is removed and the flow of CSF checked.

A syringe containing pethidine is attached to the hub of the spinal needle to aspirate the CSF, then slow injection of pethidine maintained.

The patient is kept in the lateral position to prevent aortocaval compression.

**Follow up and observation for epidural and spinal analgesia:**

The time needed for fixation and the time of onset, change in blood pressure, pulse, fetal heart rate, and any complications including numbness, vomiting or nausea was meticulously observed by the registrar of anaesthesia (every 5 minutes in the first 30 minutes and then every 30 minutes by the obstetric house officer) and managed.

Pain and analgesia were assessed by Visual Analogue Scale, asking the patient about the pain she experienced before and after the administration of the anaesthetic and by examining the sensation of spirit in the abdomen (An upper level of T10 is adequate). The motor blockade are assessed by Bromage score.

The duration of the 1st, 2nd and 3rd stage and the degree of analgesia was observed. The mode of delivery and the Apgar score
after 5 minutes were also observed.

For epidural marcaine, Top-up the analgesia by 10 ml of 0.125% marcaine hourly or before that if the analgesia not adequate.

The parturient was visited in the postpartum period to check her response to the analgesia, or any complication and record this in the sheet.

**RESULTS**

78 parturients were enrolled in the study. 38 parturients were allotted to have intrathecal pethidine and forty parturients epidural marcaine. There was a high rate of refusal of analgesia among the parturients, eighteen (47.37%) in the intrathecal pethidine group refused and twenty-three (57.50%) in the epidural marcaine group. 20 in the intrathecal pethidine group and 17 parturients in the epidural marcaine group achieved adequate analgesia, resulting with the initial study dose and satisfactorily completed the study (table 1). There were no differences in demographic variable, age, parity, cervical dilatation at the
time of enrollment, rupture of membrane, or oxytocin use between the two study groups (figure 1, 2, 3, 4). In the intrathecal pethidine group, 4 parturients were primiparous, versus 4 in the epidural marcaine group. Baseline VAS pain scores, nausea, and pruritus were similar in both groups. Parturient's satisfaction and pain were scored on verbal and visual analog scales. The median VAS score in both groups decreased>60% by the 15 minutes evaluation. VAS pain score were similar between groups at all time intervals, except for 5 and 10, which were high in the epidural marcaine group (p<0.01)(figure 9). Duration of analgesia in the intrathecal pethidine was 4:36 hours SD 40 minutes and in the epidural marcaine due to top up it was through all the 1st stage (figure 8). The incidence of pruritus was significantly higher in the intrathecal pethidine group at 5,10,15,20 min (p<0.05) but it was of mild severity (figure 6). 2 parturients in the epidural marcaine group reported nausea at baseline, which resolved with the onset of pain relief. 11 parturients in the intrathecal pethidine group (55.00%) reported nausea after analgesia administration that had not been present at the baseline (figure 6). No patient in either group demonstrated motor block as defined by using the Bromage scale (86). So all the parturients in both groups were allowed to ambulate after 30 minutes of the induction of analgesia although 17 parturients in the intrathecal pethidine complained of numbness at 5 minutes that resolved at 10 minutes (figure 6). Hypotension was recorded
in 4 parturients in the epidural marcaine group which responded to intravenous saline (table 3) while the mean blood pressure remained within the 20% range from the baseline records in the intrathecal pethidine group. Fetal distress was recorded in the fetus of one parturient in the intrathecal pethidine group (figure 6). In the epidural marcaine group fetal heart rate change was within the normal range (table 3). There was no difference in the time from analgesia to delivery, the 1st stage was 5:36 hours with SD 23 minutes in the intrathecal pethidine while in the epidural marcaine group the 1st stage was 5:46 hours with SD 32 minutes (table 2) (p=not significant). 2 parturients (10%) in the intrathecal pethidine group underwent cesarean delivery, the indication for operative delivery of the 1st was obstructed outlet, and for the 2nd was fetal distress (table 3)(p=not significant). 14 of 20 parturients rated satisfaction as excellent, with the remaining 6, five of them rated it as good and one parturient rated unsatisfactory. In the epidural marcaine group 13 of 17 parturients rated satisfaction as excellent, with the remaining 4, three of them rated it as good and the last one refused to re-insert the epidural catheter after failure (table 5). Expulsive efforts were excellent in all the 18 parturients in the intrathecal pethidine while in the epidural marcaine the expulsive efforts were excellent in 14, good in 3, and fair in one.

The 5 minutes Apgar score of the neonate at the study were >7 (figure 5). There were 4 parturients (20.00%) complaining of headache
after the delivery in the intrathecal pethidine (figure 6). No parturient in either group had a respiratory rate < 12 breath/minutes. A significant number of parturients, 17 in the intrathecal pethidine group and 14 in the epidural marcaine group, requested this form of analgesia for relief of the next labour pain (figure 7).

**DISCUSSION**

Diluted local anaesthetic solutions have become popular because they provide good analgesia without a motor block. There are data to suggest that the initiation of early epidural analgesia (3-5 cm cervical dilatation versus > 5 cm) with diluted local anaesthetic may not adversely affect the rate of operative delivery \(^{(78)}\). However, aversion to the possibility of motor block and the belief that ambulation may facilitate the early phase of labour \(^{(87, 88)}\) has contributed to the growing popularity of intrathecal opioids (such as pethidine, morphine, fentanyl or sufentanil). Most studies of this technique have used sufentanil, with the most popular dose being 10ug \(^{(89, 90)}\).

Unfortunately, sufentanil and fentanyl are not registered in Sudan so we used in this study pethidine (more lipid soluble than morphine, preservative free solution available and also has local anaesthetic
properties). The intrathecal dose was 2 ml of 1% pethidine (20 mg single dose). As in literature pethidine has been used in large doses (e.g. 1 mg/kg) (34).

Intrathecal administration of meperidine (10 to 20mg) results in effective labour analgesia within 2 to 12 minutes, with duration of 1 to 3 hours (34).

In the epidural group the initial dose was 10 ml of 0.25% marcaine, followed by 10ml of 0.125 % as a top up dose.

The first thing we faced was the high rate of refusal among women in labour. Lack of information available to them was one of the reasons. Secondly many cultures consider the pain of delivery as a "gift" to the baby. Most women think that pain is a major part of giving birth. Other females have a negative attitude towards any injection in their back considering paralysis as an inevitable result to this injection or at the very best, backache. So to administer this service we should first change the concept of those females about the epidural and spinal analgesia.

The shortage in the anaesthetic department was one of the reasons mentioned by the obstetricians. They also consider analgesia in childbirth unnecessary, despite the fact that labour and delivery are considered to be medical procedures. It is, however, considered "normal" that this medical procedure takes place with a significant amount of "pain." There is no other circumstance where it is considered acceptable for a person to
experience severe pain, amenable to safe intervention, while under a physician's care(91).

In comparing the intrathecal pethidine group with the epidural marcaine group, we found that the time to maximum analgesia for the epidural marcaine group (due to top up dose) was longer than that for intrathecal pethidine group. Although the epidural marcaine group achieved significant reduction in VAS scores at 5 and 10 minutes, it took 15 minutes to reach maximum analgesia, whereas the intrathecal pethidine group had achieved this by the 5 minutes observation. This result is similar to that result which was reached by Steven M.Dunn (91). The average duration of analgesia for intrathecal pethidine group was 4:36 hours and in the literature it was found about 3 hours (34); this may be due to the dose that was used in this study (20 mg). In Swayze CR et al (92) study, 20 term parturients were given 10 mg meperidine and then 7 mg through intrathecal catheter but in this study parturients were given 20 mg single dose.

Visual analog pain scale scores (mean +/- SD) were 8.57 +/- 1.43 before block, 0.62 +/- 0.89 immediately after block, and 0.33 +/- 0.57 at one hour after block (p < 0.0001), this VAS score is in the range of that mentioned in by Swayze CR et al (92).

Pruritus had an earlier onset in the intrathecal pethidine group. Cammann et al, (93) has shown that adding epinephrine to intrathecal
opioid decreases the incidence of pruritus. In contrast to the cammann et al. (93) study, in this study there was nausea in 11 parturients (55.00%) at 5 minutes after the administration of the intrathecal pethidine, which was mild and need no medical intervention. On the other hand there was no nausea in the epidural marcaine group; this may be due to the use of opioid in this group in camann et al study.

No motor block occurred in any of the patients. C.D Elton, and M.C Mushambi used 10-15 ml of marcaine 0.25% in their study and no motor blockade was recorded (91). So the dose of 10 ml of 0.25% is a safe dose with less motor blockade.

The status of the newborn was assessed by the Apgar score at 5 minutes, within the two groups; none of the newborn had 5 minutes Apgar score less than seven.

The effectiveness of (epidural marcaine /intrathecal pethidine) analgesia has altered the expectations of many women regarding labour pain control. A significant number of parturients are requesting this form of analgesia for relief of the next labour pain.

From the preceding discussion and literature review, it's clear that not all of the results obtained could be compared with other studies. The demographical and cultural differences between parturients in this study and those mentioned in the discussion and the literature review are not similar to those found in other studies. Also the small number of
parturients in this study precludes from drawing any conclusion about the incidence of complication.

**CONCLUSION**

- There was a high refusal rate of analgesia among pregnant women mainly due to unawareness and lack of information about analgesia during labour.
- VAS pain scores were similar between the two groups at all time intervals, except for 5 and 10 min., which were high in the epidural marcaine group (p<0.01).
- Duration of analgesia in the intrathecal pethidine was 4:36 hours SD 40 minutes while in the epidural marcaine due to top up it was throughout the 1st stage of labour.
- Apgar score at 5 min. of all the neonates within the two groups was more than 7.
- The incidence of complications was more among the intrathecal pethidine group, particularly numbness, nausea & vomiting but were all of mild severity.
- Instrumental delivery following the two methods of analgesia was of no statistical significance.
- A significant number of parturients, 17 in the intrathecal pethidine
group and 14 in the epidural marcaine group, are requesting this form of analgesia for relief of the next labour pain

**RECOMMENDATION**

- Obstetricians should discuss the use of analgesia for pain control with their parturients parentally. The risks and benefits of these methods, as well as other options for pain control, should be objectively presented to each woman well before the onset of labour.

- To preclude the dispute about the efficacy of analgesia during labour, further studies to pick up the anticipated disadvantages of analgesia (i.e., prolongation of the second stage of labour and effects on the mode of delivery) should be conducted to reach a consensus.

- "The Use of Analgesia in Labour" should be endorsed both as theory and practical sessions in the curricula of anaesthesia and obstetrics in this country.

- Adequate tools for more training should be provided.
REFERENCES


56. WWW\Astra Zeneca .com \data sheet \bupivacaine 0.5%.


Thorpe JA, Hu DH, Albin RM, McNitt J, Meyer BA, Cohen GR, Yeast JD. The effect of intrapartum epidural analgesia on...


