INTRODUCTION

Shivering is a frequently occurring post-anesthesia complication. It occurs after both general and regional anesthesia. It is estimated to follow more than 40% of all cases receive anesthesia. Shivering is defined as involuntary, spontaneous, oscillatory muscular activity. It is one of main cause of patient discomfort in the immediate postoperative period. Oxygen consumption increases with the intense of shivering. It may resemble a mild exercise but in some severe cases oxygen consumption may rise to 600%.

Post-anesthesia shivering may be caused by different factors. It is considered a physiological response to core hypothermia that accompanies anesthesia. Core hypothermia is attributed mainly to redistribution of warm core blood to cold peripheral compartment after peripheral vasodilatation that starts immediately after induction of anesthesia. Other factors help hypothermia include cold room temperature and intravenous fluids used intraoperatively. Other factors that may lead to shivering include transfusion reactions, bacteremia and sepsis and drug reaction ***(Bansal and Jain, 2011)***.

The incidence of shivering has been reported to be about 36-85% after SAB. Shivering has detrimental effects like interference in monitoring of pulse rate, blood-pressure (BP), and ECG, increase in oxygen consumption, catecholamine secretion, carbon dioxide production, metabolic rate increase by

400%, increase intraocular pressure (IOP), Intra-cranial pressure (ICP), and lactic acid production. Increase in heart rate, cardiac output and BP maycause problem in patient with low cardiac and pulmonary reserve ***(Shukla et al., 2011).***

Shivering also contribute to increased wound pain, delayed healing, and delay discharge from post-anesthetic care unit ***(Abdelrahman, 2012).***

Pethidine is considered the gold standard in management of shivering but unfortunately it cause various side effect as nausea, vomiting, dizziness, sweating and feeling restless. So there is need for investigating anew drug for management of shivering with less side effect. The aim of this study is to investigate the efficacy of diclofenac sodium in management of shivering.

AIM OF THE WORK

The aim of this study is to investigate the ability of intravenous infusion of diclofenac sodium(1mg/kg maximally 75mg) to treat established post-spinal shivering.

Chapter 1

SPINAL ANETHESIA

History

The first spinal analgesia was administered in 1885 by James Leonard Corning, a neurologist in New York. He was experimenting with cocaine on the spinal nerves of a dog when he accidentally pierced the dura mater ***(Corning, 1960).***

The first planned spinal anesthesia for surgery in man was administered by August Bier on 16 August 1898, in Kiel, when he injected 3 ml of 0.5% cocaine solution into a 34-year-old laborer. Intraoperatively the patient felt no pain at all. Some vomiting and headache were present after surgery. After using it on 6 patients, he and his assistant each injected cocaine into the other's spine. They recommended it for surgeries of legs ***(Bier, 1960).***

Anatomy of spinal column

The spine is a flexible column formed by a series of bones known as the vertebrae which provide support to the head and trunk. The vertebral column is made up of 33 vertebrae: 7 cervical, 12 thoracic, 5 lumbar, 5 sacral vertebrae fused into one structure and 4 coccygeal vertebrae also fused into one structure (fig.1). Each vertebra consists of two major parts- an anterior solid segment, or body, and a posterior segment, or arch. The cervical vertebrae are smaller than those in any other region of the spine. The thoracic vertebrae are intermediate in size, becoming

larger as they descend in the vertebral column. These vertebrae may be easily identified by the presence of costal facets on the sides of the body and a transverse process which articulates with corresponding facets of the ribs. The lumbar vertebrae are the largest segments in the spine and are easily recognized by their size and by the absence of costal facets.

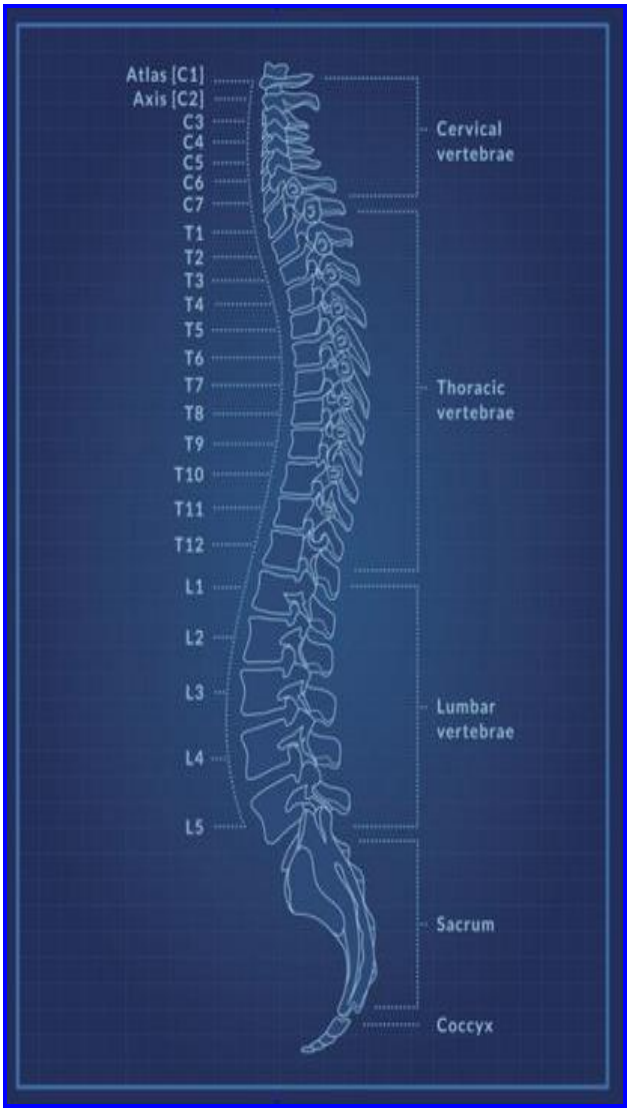

Figure (1): Anatomy of spinal column ***(Gianino, 1996).***

Anatomy of the spinal cord

The spinal cord is roughly cylindrical in shape and occupies the upper two thirds of the vertebral canal. It begins at the foramen magnum at the base of the skull, where it is continuous with the medulla oblongata, and terminates inferiorly at the conus medullaris located at the caudal level of the first lumbar vertebra. Because the spinal cord is approximately 25 cm shorter than the vertebral columns, the lower segments of the spinal cord are not aligned opposite corresponding vertebrae.

Thus, the lumbar and sacral spinal nerves have long roots, extending from their respective segments in the cord to the lumbar and sacral intervertebral foramina. These roots descend from the conus in a bundle known as the cauda equina. The non neural filament referred to as the filum terminale continues caudally until it attaches to the second segment of the coccyx ***(Maton, 1993).***

Meninges

The meninges are three connective tissue sheathings that encircle the spinal cord and brain (fig.2). The spinal meninges surround the spinal cord and are continuous with the cranial meninges, which encircle the brain. The most superficial of the three spinal meninges is the **dura mater** which is composed of dense, irregular connective tissue. It forms a sac from the level of the foramen magnum in the occipital bone, where it is

continuous with the dura mater of the brain, to the second sacral vertebra. The middle meninx is an a vascular sheath called the **arachnoid mater**. It is deep to the dura mater and is continuous with the arachnoid mater of the brain. Between the dura mater and the arachnoid mater is a thin subdural space, which contains interstitial fluid. The innermost meninx is the pia mater (delicate), which is a thin transparent connective tissue layer that adheres to the surface of the spinal cord and brain. It consists of interlacing bundles of collagen fibers and some fine elastic fibers. Within the pia mater are many blood vessels that supply oxygen and nutrients to the spinal cord. Between the arachnoid mater and the pia mater is the subarachnoid space, which contains cerebrospinal fluid.

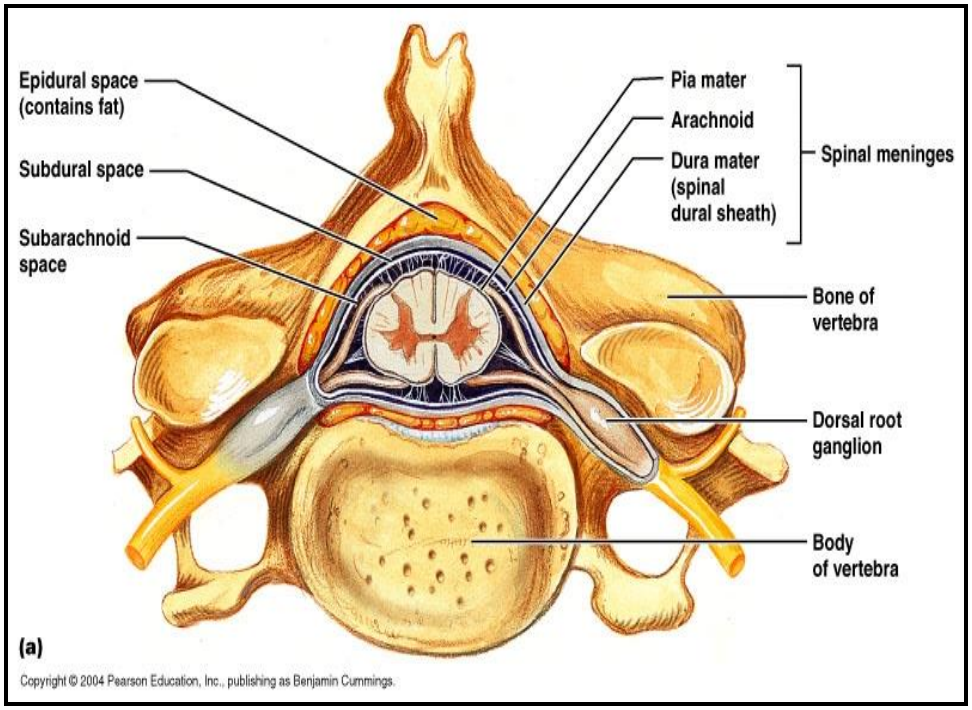

Figure (2): Meanings of the spinal cord ***(Dalley, 2009).***

Segmental and Longitudinal Organization

The spinal cord is divided into four different regions: the cervical, thoracic, lumbar and sacral regions. The different cord regions can be visually distinguished from one another. Two enlargements of the spinal cord can be visualized: The cervical enlargement, which extends between C3 to T1, and the lumbar enlargements which extends between L1 to S2 (Figure 3).

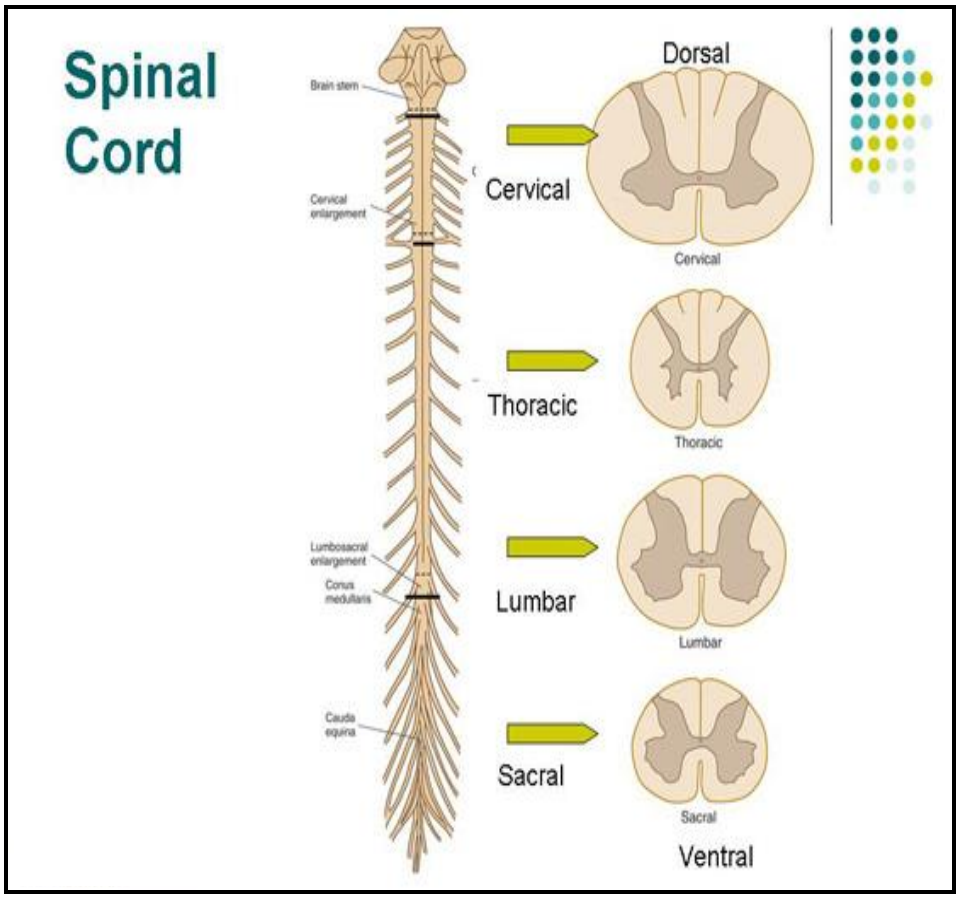

Figure (3): Spinal cord topography.

Cerebrospinal fluid (CSF)

CSF is clear, colorless fluid produced from arterial blood by the choroid plexuses of the lateral and fourth ventricles by a combined process of diffusion, pinocytosis and active transfer. A small amount is also produced by ependymal cells. It bathes and protects the central nervous system (the brain and the spinal cord) ***(Chuder, 2012).***

The CSF is produced at a rate of 0.2 - 0.7 ml per minute or 600-700 ml per day. The circulation of CSF is aided by the pulsations of the choroid plexus and by the motion of the cilia of ependymal cells. CSF is absorbed across the arachnoid villi into the venous circulation and a significant amount probably also drains into lymphatic vessels around the cranial cavity and spinal canal. The arachnoid villi act as one-way valves between the subarachnoid space and the dural sinuses. The rate of absorption correlates with the CSF pressure. CSF acts as a cushion that protects the brain from shocks and supports the venous sinuses (primarily the superior sagittal sinus, opening when CSF pressure exceeds venous pressure). It also plays an important role in the homeostasis and metabolism of the central nervous system ***(Ballabh et al., 2004).***

SPINAL ANESTHESIA

Spinal anesthesia also called spinal block, subarachnoid block, intradural block and intrathecal block, is a form of regional anesthesia involving injection of local anesthetic in

subarachanoid space. Subarachnoid (spinal) block is a safe and effective alternative to general anesthesia when the surgical site is located on the lower extremities, perineum (e.g., surgery on the genitalia or anus), or lower body wall (e.g., inguinal hernia). Because of the technical challenges of readily identifying the epidural space and the toxicity associated with the large doses of local anesthetics needed for epidural anesthesia, spinal anesthesia was the dominant form of neuraxial anesthesia well into the 20th century ***(Bronwen and Kathleen, 2011).***

Advantages of spinal anesthesia

Include avoidance of general anesthesia and the airway management concerns that accompany general anesthesia. However All patients with difficult airways, no matter what anesthetic plan is chosen, should have a well plan for airway management, should it be needed. Additional benefits may include reducing the metabolic stress response to surgery, reduction in blood loss, decrease in the incidence of venous thromboembolism, reduction in pulmonary compromise (particularly in patients with advanced pulmonary disease), and the ability to monitor the patient's mental status.

Contraindications of Spinal anesthesia

Absolute Contraindications:

- Patient refusal
- Inability to guarantee sterility of medications/equipment

- Infection at the site of injection
- Coagulopathy (acquired, induced, genetic)
- Severe hypovolemia
- Increased intra-cranial pressure (i.e. brain tumor or
- Recent head injury
- Severe aortic stenosis
- Severe mitral stenosis
- Ischemic hypertrophic sub aortic stenosis
- An allergy to local anesthetics.

Relative Contraindications:

- Sepsis (may spread infection to subarachnoid space)
- Uncooperative patient(psychosis, emotional instability)
- Preexisting neurological deficits (hard to differentiate natural progression versus neurological trauma related to neuraxial blockade)
- Demyelinating lesions (i.e. multiple sclerosis may be exacerbated by the stress of surgery, temperature changes, or natural progression. However, it may be difficult to differentiate these potential causes from the use of spinal anesthesia).
- Stenotic valvular heart lesions
- Severe spinal deformity

(Kleinman and Mikhail, 2006)

Mechanism of action

Regardless of the anesthetic agent (drug) used, the desired effect is to block the transmission of afferent nerve signals from peripheral nociceptors. Sensory signals from the site are blocked, thereby eliminating pain. The degree of neuronal blockade depends on the amount and concentration of local anesthetic used and the properties of the axon. Thin unmyelinated C-fibers associated with pain are blocked first, while thick, heavily myelinated A-alpha motor neurons are blocked moderately. Heavily myelinated, small preganglionic sympathetic fibers are blocked first. The desired result is total numbness of the area. A pressure sensation is permissible and often occurs due to incomplete blockade of the thicker A-beta mechanoreceptors. This allows surgical procedures to be performed with no painful sensation to the person undergoing the procedure ***(Morgan et al., 2006).***

Physiological effect of spinal anesthesia

Cardiovascular effect

The effects of spinal block anesthesia on the cardiovascular system typically include a reduction of blood pressure and central venous pressure. Those effects are direct and indirectly related to the sympathetic nervous system blockade promoted by spinal anesthesia ***(Lovstad et al., 2000).***

Since the level of sympathetic blockade extends two to six dermatomes above the sensorial blockade, a patient with

sensorial blockade in T4 can have a blockade of all his cardio accelerator fibers (T1-T4), resulting in progressive reduction in heart rate. Sympathetic blockade at the T1 level or above results in increased vagal tone, which causes negative inotropic, chronotropic, and dromotropic changes without opposition from the sympathetic nervous system ***(Pollard, 2001).***

Respiratory effect

Spinal blockade plays a very minor role in altering pulmonary function. Even with high thoracic levels of blockade, tidal volume is unchanged. There is a slight decrease in vital capacity and expiratory reserve volume as a result of paralysis of abdominal muscle necessary for forced expiration rather than affection of phrenic nerve. The phrenic nerve is innervated by C3-C5 and is responsible for the diaphragm. The phrenic nerve is extremely hard to block, even with a high spinal. In fact, apnea associated with a high spinal is thought to be related to brainstem hypoperfusion and not blockade of the phrenic nerve. This is based on the fact that spontaneous respiration resumes after hemodynamic resuscitation has occurred ***(Kleinman and Mikhail, 2006).***

Somatic Blockade

Spinal anesthesia effectively stops the transmission of painful sensation and abolishes the tone of skeletal muscle, enhancing operating conditions for the surgeon. Sensory blockade involves somatic and visceral painful stimulation.

Motor blockade involves skeletal muscles. Spinal anesthesia results in a phenomenon known as differential blockade. This effect is due to the activity of local anesthetics and anatomical factors. Local anesthetic factors include the concentration and duration of contact with the spinal nerve root. As the local anesthetic spreads out from the site of injection the concentration becomes less, which may in turn effect which nerve fibers are susceptible to blockade. Anatomical factors are related to various fiber types found within each nerve root. Small myelinated fibers are easier to block than large unmyelinated fibers. In general, the differential blockade found after spinal blockade is as follows: sympathetic blockade is 2-6 dermatome segments higher than sensory and sensory blockade is generally 2 dermatome levels higher than motor ***(Warren and Liu, 2008).***

Technique of spinal anesthesia

The technique of administering spinal anesthesia can be described as the "4 P: preparation, position, projection, and puncture**.**

Preparation

Preparation of equipment/medications is the first step:

1-**Discuss** with the patient options for anesthesia.

2-**Explain** risk and benefits.

3-**Inform** the patient about the following: despite sedation the patient may remember portions of the surgical procedure but shouldn't feel discomfort, the patient may feel pressure sensations but no pain, the patient will not be able to move their legs, and the approximate length of time that the block will last.

4-**Choose** an appropriate local anesthetic.

5-**Choose** the appropriate spinal needle. Spinal needles are available in a variety of sizes (from 16-30 gauge), lengths, bevel types, and tip designs. Commonly, a 22 gauge needle is used in patients that are 50 years and older. A 25-27 gauge needle is used in patients that are less than 50 years of age. A smaller needle is used in the younger patient to decrease the incidence of post dural puncture headache ***(Brown, 2005).***

Positioning

There are three positions used for the administration of spinal anesthesia: lateral decubitus, sitting, and prone.

Projection and Puncture

There are two approaches to accessing the subarachnoid space: the Para median and midline approach

- Scrub and glove up carefully.
- Check the equipment on the sterile trolley.