"A RANDOMISED CLINICAL TRIAL TO COMPARE THE POST DURAL PUNCTURE HEADACHE FOLLOWING SPINAL ANAESTHESIA USING 27 G QUINCKE'S AND 27 G WHITACRE'S SPINAL NEEDLES"

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ENDORSEMENT

This is to certify that the dissertation entitled "A RANDOMISED CLINICAL TRIAL TO COMPARE THE POST DURAL PUNCTURE HEADACHE FOLLOWING SPINAL ANAESTHESIA USING 27 G QUINCKE'S AND 27 G WHITACRE'S SPINAL NEEDLES" is a bonafide research work done by the CANDIDATE REG NO. BA0108004 in the Department of Anaesthesiology, Jawaharlal Nehru Medical College, Nehru Nagar, Belgaum – 590 010.

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LIST OF ABBREVIATIONS USED

aPTT	-	Activated partial thromboplastin time
ASA	-	American Society of Anaesthesiologists
BMI	-	Body mass index
BP	-	Blood pressure
C	-	Cervical
CNS	-	Central nervous system
CSF	-	Cerebrospinal fluid
CVS	-	Cardiovascular system
DM	-	Diabetes mellitus
EBP	-	Epidural blood patch
ECG	-	Electrocardiogram
FRC	-	Functional residual capacity
G	-	Gauge
GA	-	General anaesthesia
GIT	-	Gastro-intestinal tract
Hb	-	Haemoglobin
HR	-	Heart rate
HTN	-	Hypertension
I.P.	-	Inpatient number
INR	-	International normalized ratio
IV	-	Intravenous
L	-	Lumbar
LA	-	Local anaesthetic
LP	-	Lumbar puncture

MAP	-	Mean arterial pressure
mEq/L	-	Milli equivalent per litre
mg	-	Milli gram
mg/dL	-	Milli gram per deci litre
ml	-	Milli litre
mm Hg	-	Milli meter of mercury
mm	-	Milli meter
NAS	-	Numeric analog scale
NIBP	-	Non-invasive blood pressure
PACU	-	Post anaesthetic care unit
PDPH	-	Post dural puncture headache
PR	-	Pulse rate
PT	-	Prothrombin time
RR	-	Respiratory rate
RS	-	Respiratory system
S	-	Sacral
S.D.	-	Standard deviation
SA	-	Spinal anaesthesia
SAB	-	Sub-acrachnoid block
SPO_2	-	Oxygen saturation
T	-	Thoracic
TPVR	-	Total peripheral vascular resistance
V/Q	-	Ventilation perfusion ratio

ABSTRACT

Background and objectives

Spinal anaesthesia is one of the most commonly used technique in anaesthesia. It is economical, safe, cost effective, easy, needs less sophisticated anaesthetic equipment, drugs, post operative care hence preferred over general anaesthesia and most popular because of its profound analgesia and muscle relaxation. Objectives of the present study were to know the incidence of post dural puncture headache (PDPH), number of attempts for successful sub arachnoid block and incidence of failed spinal anaesthesia by using 27 G Quincke's and Whitacre's spinal needles.

Methodology

This one year randomized clinical trial was conducted in the Department of Anaesthesiology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2009 to December 2009 on 352 patients between 20 to 60 years of age with ASA grade I and II undergoing lower abdominal and lower limb surgeries during the study period. The Institutional Ethical Clearance and written informed consent from patients was obtained the incidence of PDPH, number attempts and failed spinal anaesthesia were assessed.

Results

In this study female preponderance was seen. Significantly high incidence of PDPH was recorded in Quincke group (3.98%) as compared to 0.57% in Whitacre group ($p=0.031$). Significantly less number of attempts were required using Whitacre 27 G needle ($p=0.0001$). Failed rates were higher in patients

using Whitacre 27 G needle as compared Quincke 27 G needle (3.98% versus 2.84%).

Interpretation and conclusion

Overall the Whitacre 27 G needle has better results with respect to PDPH and number of attempts required for successful subarachnoid block whereas the incidence of failed spinal anaesthesia was less with Quincke 27 G needle.

Keywords

Post dural puncture headache; failed spinal anaesthesia; Sub-arachnoid block; Quincke needle; Whitacre needle.

CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

LIST OF PHOTOGRAPHS

INTRODUCTION

Pain is the most dramatic, complex and universal phenomenon, perhaps the only sensation which is well understood by mankind. It is an unpleasant sensation which only the individual can appreciate. To quote Hippocrates, "Divine is the task to relieve pain." The International Association for study of pain has defined "A conscious sensation of distress, suffering or agony with actual or atleast potential tissue damage".[1]

An essential part of the anaesthesiologists work is rendering the patient insensitive to pain. The control centre is regulated by the brain which receives information through the spinal cord and specialised sensory cells. Spinal anaesthesia act by temporary interruption of transmission of nerve impulses produced by injection of a local anaesthetic agent into subarachnoid space. It is one of the most commonly used anaesthesia technique for lower extremity and lower abdominal surgeries.[2] It is economical, safe, easy technique and is preferred over general anaesthesia (GA).[2]

It was discovered by J. Leonard Corning in 1885, a neurologist from New York – he accidentally pierced the dura and injected cocaine to produce spinal analgesia in dog. He concluded that if "cocaine was injected in between the two spinous processes, it would be absorbed by veins causing sensory and motor blockade".[3]

August Bier first used it deliberately in 16th August 1898 with three ml of 0.5% cocaine. On 24th August, he was administered spinal anaesthesia by his

assistant. During the attempt, a lot of cerebrospinal fluid (CSF) was lost and Bier developed post dural puncture headache (PDPH) and this was the first documented case of PDPH.[4] Since then it has passed through phases, characterized by overly enthusiastic acceptance followed by phases of rejection.

Spinal anaesthesia has become popular because it results in good sympathetic blockade, sensory analgesia, profound muscle relaxation and less operative blood loss. However, the fear of precipitating PDPH after spinal anaesthesia currently limits the use since the incidence of this complication is directly related to gauges (G) and types of needle. Various gauges and tips have been devised to reduce the incidence of PDPH. The newly introduced Whitacre needle is associated with lesser incidence of PDPH.[5]

To combat these side effects, various attempts have been made to change the size and design of the needle. Trials comparing non-cutting (Whitacre's, pencil point) with cutting needles (Quincke's) to decrease PDPH have also been tried out.[6,7,8]

There are very few studies reported in the literature comparing the incidence of PDPH using 27 G Quincke's and 27 G Whitacre's needles. Hence the present study was an attempt to compare these two needles with respect to the incidence of PDPH as well as the number of attempts required to administer successful subarachnoid block (SAB).

OBJECTIVES

Objectives of the present study were to evaluate the following parameters on using 27 G Quincke's and Whitacre's spinal needles.

Primary objective

- To know the incidence of PDPH.

Secondary objective

- To know the number of attempts for successful SAB and incidence of failed spinal anaesthesia.

REVIEW OF LITERATURE

SPINAL ANAESTHESIA

History

Spinal anaesthesia, also referred to as SAB, intrathecal analgesia or central neuraxial blockade. Spinal anaesthesia is produced when a local anaesthetic agent is injected into the subarachnoid space and was the first major regional technique attempted.

Spinal anesthesia was initially produced inadvertently by J. Leonard Corning, a neurologist in New York in 1885. He accidentally pierced the duramater while experimenting with cocaine on spinal nerves of a dog. But spinal anesthesia could not become an acceptable means for use of cocaine until a safe predictable means for performing lumbar puncture was described.[3] Quincke did this in 1891. In 1899, August Bier used Quincke`s technique to inject cocaine in order to produce operative anesthesia in six patients, the first real spinal anesthesia.[4] In the same year, Matas in New Orleans and Tuffier in France also reported on the use of cocaine spinal anesthesia. However, the popularity of cocaine SAB was limited owing to the high incidence of central nervous system (CNS) side effects like tremors, hypereflexia, severe headache and muscle spasm and pains.

Procaine was the first synthetic local anesthetic to be used. Einhorn prepared it in 1904. In 1905, Heinrich Brown, German surgeon, reported the use of procaine for operative SAB. The understanding of causes of spinal anesthesia induced hypotension and its management was described by clinicians like

Babcock, Koster, Labat, and Pitker.[9]

Indications

Spinal anaesthesia is choosen among the patients undergoing lower abdominal surgeries or lowerlimb surgeries.[9]

Complications

Can be broadly classified as immediate (on the operating table) or late (in the ward) or in the post-anaesthesia care unit (PACU).

Adverse exaggerated physiological responses

- Urinary retention.
- High block.
- Total spinal anaesthesia.
- Cardiac arrest.
- Anterior spinal artery syndrome.
- Horner's syndrome

Complications related to needle / catheter placement

- Trauma
- Backache
- Post dural puncture headache
- Diplopia
- Tinnitus
- Neural injury
    - Nerve root damage
    - Spinal cord damage
    - Cauda equina syndrome

- Bleeding
    - Intraspinal epidural hematoma
- Misplacement
    - No effect / inadequate anaesthesia
    - Subdural block
    - Inadvertent SAB
    - Inadvertent intravascular injection
- Catheter shearing retention
- Inflammation
    - Arachnoiditis
- Infection
    - Meningitis
    - Epidural abscess

Drug toxicity

- Systemic local anaesthetic toxicity
- Transient neurological symptoms
- Cauda equina syndrome.[9]

POST DURAL PUNCTURE HEADACHE

It is bilateral, frontal or occipital headache and extends to the neck which may be throbbing or constant in nature, aggravated by sitting or standing and relieved by lying down, usually occurs 12 to 72 hours following the spinal anaesthesia or lumbar puncture (LP). It is believed to result from leakage of CSF from a dural defect and decreased intracranial pressure. Loss of CSF at a rate faster than it can be produced causes traction on the structure supporting the brain, particularly the dura and tentorium. Increased traction on the blood vessels

also likely contributes to the pain. Traction on cranial nerves occasionally cause diplopia (sixth nerve) and tinnitus. The incidence of PDPH is strongly related to needle size and type, and patient population. The larger the needle the greater incidence of PDPH. Cutting point needles are associated with higher incidence of PDPH compared to pencil point needle of the same gauge. A cutting needle introduced with bevel parallel to the longitudinal fibers after dura said to separate these fibers rather than transecting them, therefore reducing chance of PDPH.[10]

SPINAL NEEDLES

The history of the development of spinal needles, and in particular of the tip of the spinal needle, began with the understanding of the anatomy and physiology of the CNS that pertained at the time of the introduction of spinal anaesthesia. This was followed by the development of new equipment and techniques.[11]

The first spinal anaesthetic

By 1841, Zophar Jayne of Illinois had designed a syringe attached to a small, sharp, hollow beak with an opening on the side near the tip. Subsequently, in 1853, Daniel Ferguson developed a syringe and hollow platinum trochar with an oblique opening on one side encased in an outer tubing, also with an oblique opening.[11]

Development of the cutting spinal needle tip

In 1891, Quincke published a paper describing a standardised technique of lumbar puncture for the release of cerebrospinal fluid (CSF) for diseases

associated with increased intracranial pressure. He used a needle of which it is difficult to find a description, except that it was a sharp, bevelled, hollow needle. The needle used was described as a Quincke needle. Bier's work caused a sensation in the medical world, with widespread acceptance of the technique for surgery, although Bier himself still had reservations. Over time, he developed his own needle. He felt that the use of introducers and dilators for the insertion of the finer needles previously used was cumbersome, and he designed a larger bore needle that needed no introducer. The Bier spinal needle was 15 G or 17 G, with a long, cutting bevel and a sharp point.[11]

Pencil-point needles

Once the suggestion that, dural fibres were less likely to be damaged by non-cutting tips had been publicized, it was only a matter of time before the advent of the completely non-cutting needle tip. Kirschner and Rovenstine's needles, which had a lateral orifice, contributed to the next phase of needle tip design. As with many medical discoveries, those credited for the introduction of a new aspect of equipment design were not those who first described it. Hart and Whitacre are commonly associated with the design of the first closed-ended, lateral orifice, pencil-point needle, but a Swedish doctor called Haraldson published a paper in 1951 (several months before Hart and Whitacre) that described a needle he had developed to decrease the incidence of PDPH. The needle was of fine gauge, with a solid non-cutting tapering point and an orifice on the conical surface two mm from the actual tip of the needle. He quoted a PDPH rate of nine percent for the non-cutting needle (none severe) as opposed to 32% (18% severe) for a cutting needle.[11]

Table 1. Types of pencil point needles[12]

Type	Gauge	Diameter
Quincke	22	0.70
	25	0.50
	27	0.41
	29	0.32
Sprotte	24	0.55
	25	0.50
Whitacre	25	0.50
	26	0.45
	27	0.40
Atraucan	26	0.45

Many attempts have been made to reduce the incidence of PDPH by using different gauge spinal needles of different size.

In a study of 75 American Society of Anasthesiology (ASA) grade I female patients of a young age group, authors compared the incidence of PDPH after 25 G and 27 G Qunicke needle, and 27 G Whitacre's needle during spinal anaesthesia for Caesarean section. The incidence was found to be minimum with 27 G Whitacre's needle (although statistically insignificant), but had a higher failure rate with regards to the single needle insertion.[13]

In a brief clinical report wherein the author studied 200 ASA grade I and II patients scheduled for knee arthroscopy to know the PDPH incidence, concluded that, both types of needles were comparable with respect to the incidence, severity and duration of PDPH and failed spinal anaesthesia.[14]

Another study was conducted to assess the incidence of successful spinal anaesthesia and post dural puncture headache using 27 G Quincke and Whitacre's spinal needles in 398 ASA grade I to II patients undergoing elective orthopaedic procedures. It was concluded that, both needles were associated with very low incidence of both PDPH and failed anaesthesia.[15]

A study conducted to compare the frequency of PDPH and failure rate of spinal anesthesia using 25 G Quincke and 25 G Whitacre needles in obstetric patients suggested that, use of 25 G Whitacre needle reduces the frequency of PDPH without increasing the failure rate of spinal anesthesia in obstetric patients.[16]

In a study authors compared 22 G Whitacre or 25 G Whitacre or 26 G Quincke needle among 150 women undergoing elective Caesarean section under spinal anesthesia. Each group was compared for ease of insertion, number of attempts of needle insertions, quality of the subsequent analgesia and incidence of post operative complication. Study concluded that the use of 22 G and 25 G Whitacre needles is associated with a low incidence of post dural puncture headache. The belief that repeated unrecognized dural puncture may cause an increased incidence of PDPH has not been supported by this study.[17]

A retrospective study on 160 patients, regarding 25 G and 26 G needle and age factor in relation to postdural puncture headache concluded that PDPH is more frequent using 25 G needle than 26 gauge.[18]

Another study comparing the incidence of PDPH between 25 G Whitacre with 25 G and 26 G Quincke needles showed there is lower incidence PDPH with 25 G Whitacre spinal needle and also less severity when compared to 25 G and 26 G Quincke needle although the difference was not statistically significant.[19]

Another study conducted among 4125 parturient patients undergoing spinal anaesthesia compared between 26 G and 27 G quincke and 25 G whitacre spinal needle showed 5.2% with 26 G Quincke, 2.7% with 27 G Quincke needle and 1.2% in 25 G Whitacre needle. Concluded use of smallest Gauge needle and the one has non cutting Whitacre needle produced lowest incidence of PDPH.[20]

FAILED SPINAL ANAESTHESIA

It is sequelae of spinal anaesthesia. Failure of spinal anaesthesia was defined as either inability to elicit free flow of CSF after three attempts or clearly inadequate analgesia for surgery at 15 minutes after giving local anaesthetic (LA).[15]

The needle insertion technique is relatively straight forwards with CSF providing both the clear indication of successful needle placement and a medium through which local anaesthetic solution usually spreads readily. The possibility of failure has been recognized by Gaston Labat “The father of modern regional

anaesthesia". The incidence of failed spinal anaesthesia accounts as high as 17%.[21]

For successful SAB using Whitacre needle, tip must be precisely guarded using any type of spinal needle. It is particular issue with the pencil point needle which is now used widely to minimize the incidence of PDPH. The opening at the end of these needles is proximal to the tip, so only a minor degree of backward movement during syringe attachment may result in epidural injection as was recognized at an early stage in the wide spread use of such needles. The distances involved are of the order of a millimetre (mm) or two, but (as with leakage) misplacement of only a small amount of solution can have significant effects. An additional issue with pencil-point needles is that the opening, being much longer than the bevel of a Quincke needle, may 'straddle' the dura so that some solution reaches the CSF, and some the epidural space. This may be exaggerated by the dura acting as a 'flap' valve across the needle opening. Initially, CSF successful, but subsequent injection pushes the dura forward and the solution is misplaced pressure pushes the dura outwards so that aspiration is successful, but subsequent injection pushes the dura forward and the solution is misplaced.[21]

These eventualities, being subtle abnormalities of placement, are impossible to identify at the actual time, but rotation of the needle through 360^0 after the initial appearance of CSF, and before check aspiration, has been advocated as a way of minimizing the possibility of them occurring, the theory being that the rotation reduces the risk of the membrane edges catching on the opening.[21]

A study conducted to examine the failed spinal anaesthesia in orthopedic patients reported 8.5% and 5.5% failed spinal anaesthesia using Quincke and Whitacre 27 G respectively. However, this difference was statistically not significant.[15] whereas, another study conducted to assess incidence of failed spinal anaesthesia in C section reported 4% and 12% using 27 G Quincke and 27 G Whitacre respectively.[13]

BASIC SCIENCES

APPLIED ANATOMY

Sound knowledge of anatomy of vertebral column and its contents is essential to all the anaesthesiologists for safe and successful administration of spinal anaesthesia, not only in terms of performance but also in terms of spread of drug in CSF and level of block achieved.

Vertebral column

Main function of vertebral column is to protect the spinal cord. The vertebral column comprises total of 33 vertebrae and includes;[22]

- Cervical - 7
- Thoracic - 12
- Lumbar - 5
- Sacrum - 5 (fused)
- Coccyx - 4 (fused)

Curves of spine

In adult, the vertebral column has four curves which have significant effect on spread of drugs in sub arachnoid space namely;[22]

- Cervical curve - Convexity anterior
- Thoracic curve - Concave anterior
- Lumbar curve - Convexity anteriorly

In adults the curves of the spine are important when patient is supine or horizontal. The highest point of cervical and lumbar curves in supine position are at cervical (C) five and lumbar (L) five; lowest points of thoracic and sacral are at thoracic (T) five and sacral (S) two respectively.[22]

Vertebral ligaments[23]

Vertebral column is bound together by following ligaments which give stability and elasticity.

Supraspinous ligament: This is a strong fibrous cord which connects apices of spinous processes from sacrum to cervical five where it is continued as the ligamentum nuchae (Figure 2).

Interspinous ligament: This is a thin membranous ligament which connects spinous processes blending anteriorly with ligamentum flavum and posteriorly with supraspinous ligament (Figure 2).

Ligamentum flavum: This ligament comprises yellow elastic fibres and connects adjacent lamina. Laterally this ligament begins at the root of articular processes and extends posteriorly and medially to the point where laminae join to form spinous process (Figure 2).

Longitudinal ligaments: There are two longitudinal ligaments (anterior and posterior) that bind vertebral bodies together (Figure 2).

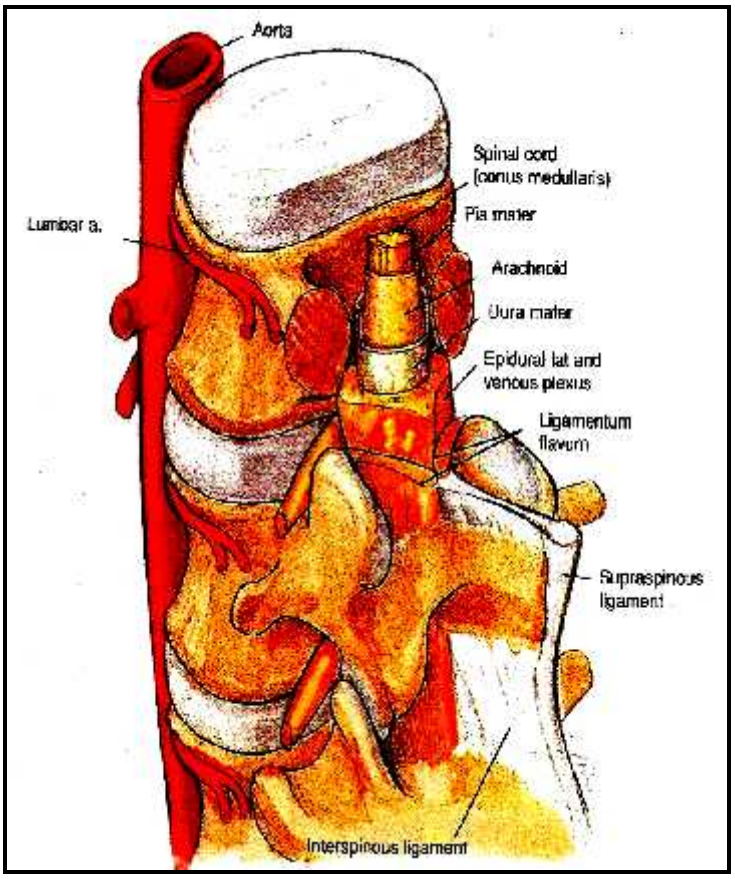


Fig 1: Vertebral Column

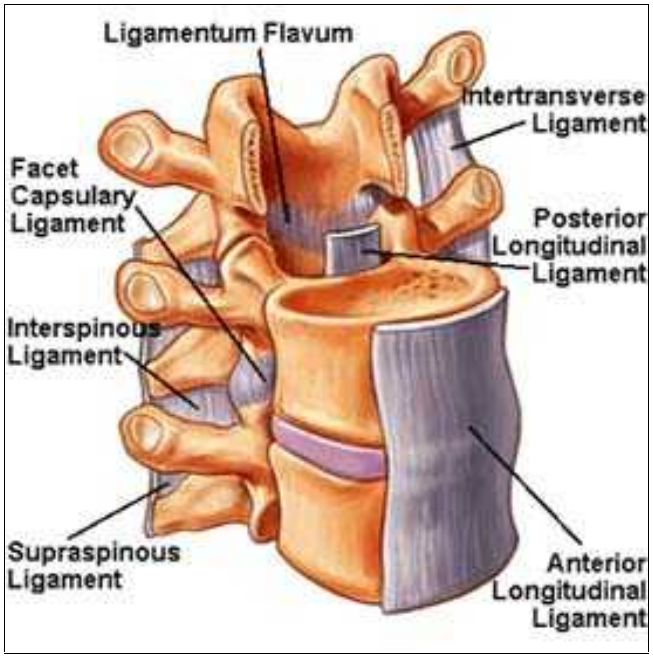


Figure 2: Spinal Ligaments

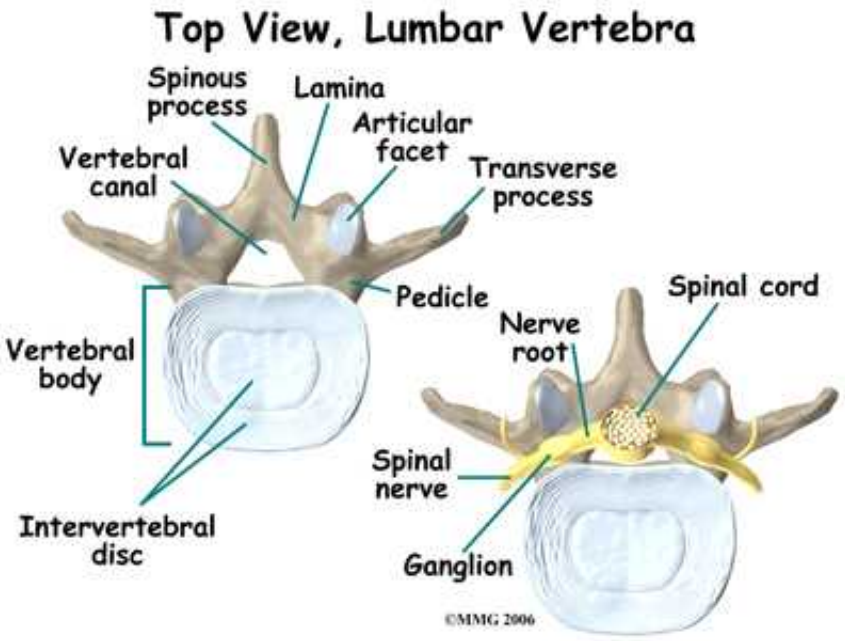


Figure 3: Typical Lumbar Vertebra

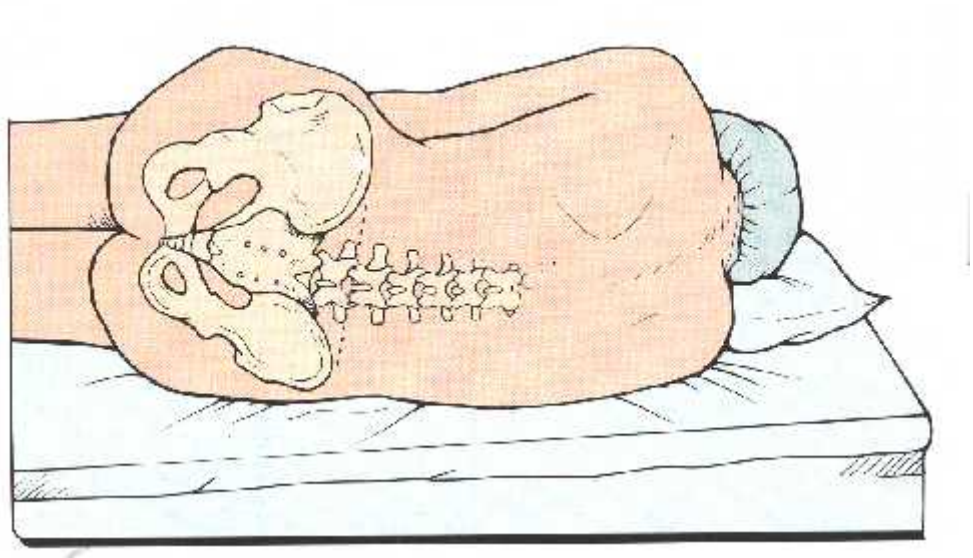


Fig 4: Line of Tuffier

Intervertebral Discs[24]

These are principle connecting link between vertebral bodies. The intervertebral discs account for about 25% of the length of the spine. They have two parts. The outer fibrous part called the annulus fibrosus is made mostly of fibrous tissue, while the softer core of the disc is the nucleus pulposus. Atrophy of the discs along with osteoporosis of the vertebra leads to decreased height and kyphotic deformity of old age (Figure 3).

Topographical Line of Tauffier[25]

This is a horizontal line across the back between the crests of the ilia passing over the spine of the 4th lumbar vertebra in the upright position. In a patient lying in the lateral position it may also pass through L4 and L5 interspaces. The superior iliac crest is used to identify the L4 and L5 interspace during spinal anesthesia (Figure 4).

Lumbar vertebrae[24]

A typical lumbar vertebrae consists of (Figure 3);

- A kidney shaped body.
- Two pedicles directed backwards from the upper part of the body.
- Two transverse processes which are slender
- Two laminae meeting posteriorly and enclosing the triangular vertebral foramen.
- Spinous processes which are thick, broad and quadrilateral in shape.

- Two upper and lower articular processes which prevent rotation but allow limited flexion and extension between contiguous vertebrae.

Vertebral canal

- Spinal cord
- Spinal nerve roots
- Meninges
- Cerebrospinal fluid
- Vessels
- Fat
- Loose areolar tissue

Spinal cord[22]

The average length of the spinal cord in males is 45 centimeter (cm) and females it is 42 cm. The average weight is approximately 30 gm.

The spinal cord is a continuation of the medulla oblongata below the level of foramen magnum and it tapers off into a conical extremity known as conus medullaris. A delicate fibrous filament descends to the back of first segment of coccyx from apex of conus medullaris. This is known as the filum terminale.

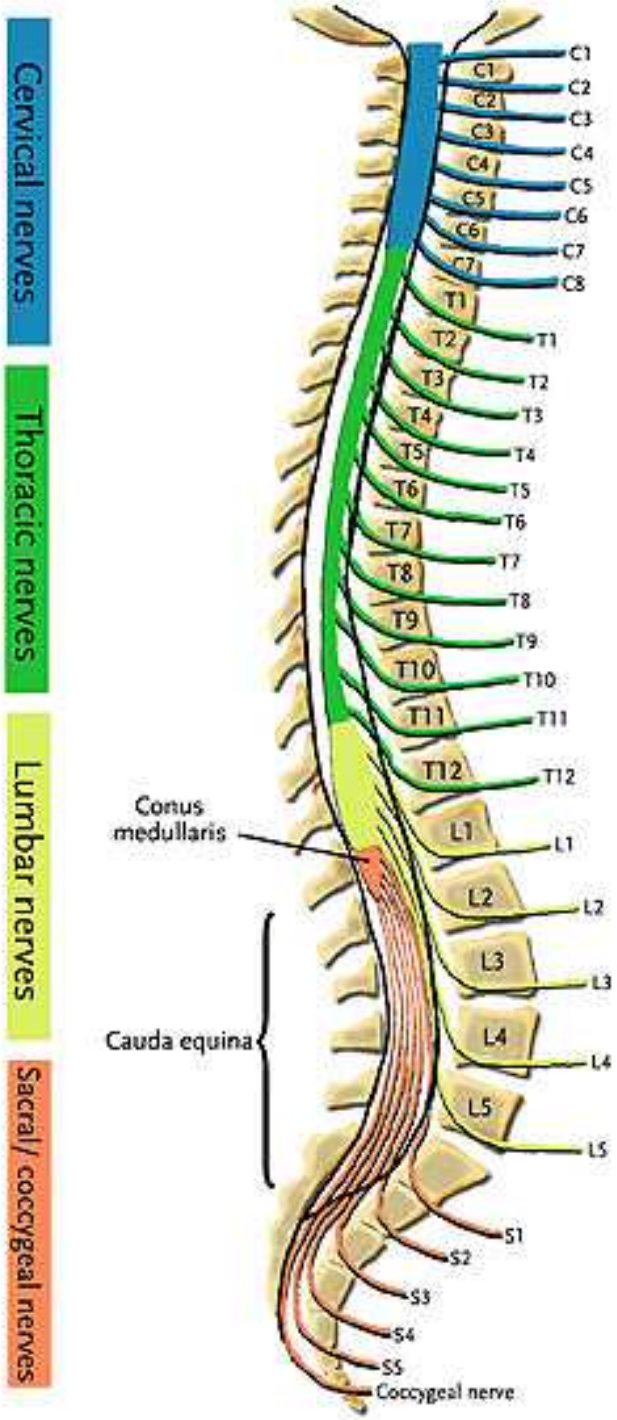


Figure 5: Spinal nerve roots

At birth, spinal cord ends at the level of lower border of lumbar (L) three vertebra. In the adult, the vertebral level of termination of spinal cord may be as follows;

- Lower border of L1 - 50%
- Upper border of L2 - 40%
- Upper border of L3 - 3%

From the spinal cord 31 pairs of spinal nerves arises made of a ventral and a dorsal root. These anterior and posterior roots cross the subarachnoid space, pass through the dura and extradural space independently and unite at the level of intervertebral foramen to form spinal nerve trunks, which soon divide into anterior and posterior primary divisions.

Amount of white matter decline progressively from the cervical down to the lumbar region. The gray matter is greately increased in the both the lumbar and cervical enlargement

Blood Supply of Spinal Cord[22]

The arterial supply is from the anterior and posterior spinal arteries. The anterior spinal artery is a single vessel lying in front of the anterior median fissure. It arises from the meeting of two small arteries, one given off from each vertebral artery at the level of the foramen magnum. It descends along the whole length of the cord receiving small communications from the intercostal and lumbar arteries; to provide the extra blood supply needed in the cervical, thoracic and lumbar enlargements.

There are two posterior spinal arteries-one on each side. They are derived at the base of the brain directly from the vertebral artery or more often from a primary branch of each vertebral artery. They supply the posterior one-third of the spinal cord. This supply is augmented by spinal branches of vertebral, ascending cervical, posterior intercostals, lumbar and lateral sacral arteries, which pass through the intervertebral foramina.

Venous drainage is through a plexus of anterior and posterior veins in the neck, azygous veins in the thorax, lumbar veins in the abdomen, and lateral sacral veins in the pelvis. There is no anastomosis between the anterior and posterior spinal arteries.

The longest of the feeder arteries is the radicularis magna (artery of Adamkiewicz), which supplies the anterior spinal artery in the area of the lumbar enlargement of the cord. It enters by way of a single intervertebral foramen (78% of the time on the left) between the T8 and L3 foramina.

Meninges[26]

The spinal cord has three covering membranes from inward to outward. They are the pia mater, the arachnoid mater and the dura mater. The dural sac of spinal duramater is the continuation of meningeal layer of the cranial duramater. It is a circular sac or sleeve surrounding the spinal cord. Above, it is firmly attached to the circumference of the foramen magnum.

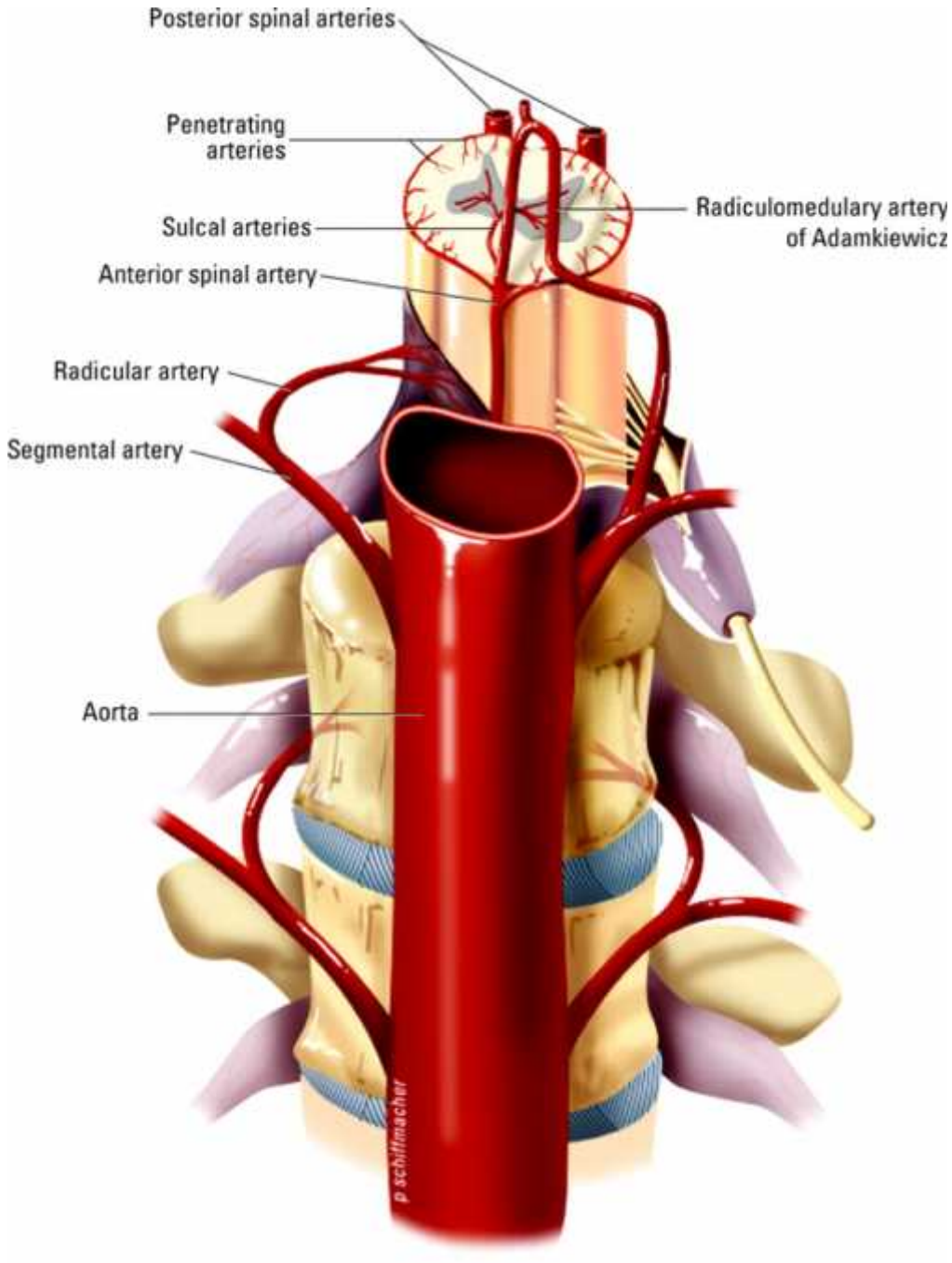


Figure 6: Blood Supply of Spinal Cord

Duramater[26]

It is the outermost membrane, the fibers of which run longitudinally. Although continuous, it can be described in two parts: the cranial and the spinal. The cranial dura consists of an outer layer (endosteal), which lines the skull, and an inner layer (meningeal), which invests the brain and folds inward to form the falx cerebri and tentorium cerebelli.

Arachnoid Mater[26]

The arachnoid mater is a delicate non-vascular membrane applied closely to the dura mater. The lower extent of dural sac is as follows;

- S2 vertebra 35%
- Below S2 40%
- Above S2 25%

Below this the dura continues as the filum terminale. The subarachnoid space is the space between the arachnoid and pia mater. This space is traversed by cobweb trabeculae and by the cranial and spinal nerves, it is bathed in spinal fluid. The space is annular in the cranial and thoracic vertebrae and is about three mm deep. Below the first lumbar vertebrae it is circular.

Subarachnoid Space[26]

The space between the arachnoid and pia is called the subarachnoid space and is filled with cerebrospinal fluid and contains numerous arachnoid trabeculae which form delicate sponge like mass. This space has three divisions which are

free communication to each other: cranial (surrounding the brain), spinal (surrounding the spinal cord) and root (surrounding the dorsal and ventral nerve roots). In the spinal cord these nerve roots are covered only by pia and bathed in CSF. As these spinal nerve roots pass beyond the spinal dura and traverse the epidural space, they carry with them all the three meningeal layers and have a distinct epidural, subdural, subarachnoid and sub pial spaces. The subarachnoid space extends separately along both the dorsal and ventral roots to the level of dorsal root ganglion, where arachnoid and pia continue as perineural epithelium of peripheral nerve.

Pia Mater[26]

The pia mater, the innermost membrane is a vascular sheath which closely invests the brain and spinal cord.

Cerebrospinal Fluid[26]

It is a clear colourless fluid found in the cranial and spinal subarachnoid spaces and in the ventricles. CSF is mainly formed by either secretion or ultrafiltration from the choroidal plexus of lateral ventricles. CSF flows from the lateral ventricles into the third ventricle through the foramina of Monro into the fourth ventricle through the Aqueduct of Sylvius into the cerebromedullary cisterna (cisterna magna) through foramen of Magendie and foramina of Luschka. From the cisterna magna, CSF enters subarachnoid space circulating around brain and spinal cord before being absorbed into the arachnoid granulations over the cerebral hemispheres.

Composition of cerebrospinal fluid

- Specific gravity : 1.003 to 1.009 at 37^0C.
- Volume : 120 ml to 150 ml (25 ml to 35ml in spinal space).
- CSF pressure : 60 to 80 mm Hg in lumbar space.
- pH : 7.27 to 7.37
- PCO_2 : 48 mm Hg
- HCO_3 : 23 mEq/L
- Sodium : 135 to 145 mEq/L
- Calcium : 2 to 3 mEq/L
- Phosphorous : 1.6 mg/dl
- Magnesium : 2 to 2.5 mEq/L
- Chloride : 15 to 20 mEq/L
- Proteins : 23 to 38 mg/dl

It is important to know that certain drugs alter the rate of formation of CSF. Carbonic anhydrase inhibitors like acetazolamide reduce the rate of CSF formation by as much as 50%. Furosemide in large doses may reduce the CSF formation. Inhalational anaesthetics like isoflourane and vasoconstrictors decrease the CSF formation. CSF formation is decreased when the serum osmolality is increases and increased when the serum is made hypotonic. During equilibrium, rate of formation equals the rate of absorption (500 mL/day).

PHYSIOLOGY OF SUB ARACHNOID BLOCK

The well recognized physiological effects of subarachnoid block are often mistakenly termed as complications. It is imperative to make a clear distinction between the physiologic effects of an anaesthetic technique and complications that implies some harm to the patients. The various factors, which control the different effects of a spinal anesthetic technique are;[27]

- Amount and type of drug
- Volume of solution
- Site of injection
- Rate of injection
- Specific gravity of solution – density and baricity
- Barbotage

The various factors which affect the spread of local anaesthetics include;[28,29]

- Patient factors:
- Age
- Height
- Position
- Spinal column configuration
- Cerebrospinal fluid volume
- Technical factors :
- Site of injection

- Spread of injection
- Direction of needle
- Local anesthetic dose
- Local anesthetic baricity
- Local anesthetic volume

The sensory and motor blockade results from direct effects of local anesthetic on the spinal nerve roots. The primary site of action is on both anterior and posterior nerve roots, affecting smaller nerve fibers first and thick large motor fibers last. Generally, the sympathetic paralysis is more diffuse and will extent two to four segments above motor block. The sympathetic fibers are affected first and last to recover, on the other hand motor nerve blockade is usually last to be affected and first to recover.

Sequence of spinal anaesthesia (SA)[30]

- Vasomotor block: Dilatation of skin vessels and increase cutaneous blood flow
- Temperature fibers: Cold first and then warmth
- Loss of temperature discrimination
- Pain – pin prick fibers first
- Loss of tactile sensation
- Motor paralysis
- Pressure sensation
- Proprioception and vibratory sensation.

Sympathetic blockade is the major determinant of physiologic response to spinal anesthesia.

Cardiovascular effects of spinal anesthesia[31]

The most important physiologic response to spinal anesthesia involves the cardiovascular system. They are mediated by the combined effects of autonomic denervation and, with higher levels of neural blockade, added effects of vagal innervation. Because of the importance of sympathetic denervation in the genesis of cardiovascular changes during spinal anesthesia, the effect of spinal anaesthesia on the sympathetic nervous system warrants discussion before consideration of the cardiovascular responses themselves.

Sympathetic blockade

Because the level of sympathetic denervation under SAB determines the magnitude of cardiovascular responses to spinal anesthesia, it might be anticipated that the higher the level of neural blockade, the greater would be the change in the cardio-circulatory parameters. In the presence of partial sympathetic blockade, a reflex increase in sympathetic activity occurs in sympathetically intact areas. The result is vasoconstriction that tends to compensate for the peripheral vasodilatation-taking place in the sympathetically denervated areas. This can be seen in the changes in the arterial pressure waveforms and in the cutaneous blood flow in the upper extremities in the presence of low or midthoracic sensory levels of spinal anaesthesia. Of even greater importance is the fact that the most cephalad preganglionic sympathetic fibers exit the spinal cord at the level of T1. Since sympathetic denervation is

complete at the T1 level, cardiovascular changes are no greater with mid cervical sensory levels of anesthesia than they are with T1 levels.

Functional Anatomy of Sympathetic Nervous System[32]

The sympathetic nervous system originates from spinal cord in the thoracolumbar region, from the first thoracic through the second lumbar segment. The preganglionic neurons have cell bodies within the intermediolateral columns of the spinal gray matter. Nerve fibers from these cell bodies extend to three types of ganglia grouped as paired sympathetic chains, various unpaired distal plexus or terminal or collateral ganglia near the target organs. The 22 paired ganglia lie along either side of vertebral column. Nerve trunks connect these ganglia to each other and gray rami communicants connect the ganglia to the spinal nerves. The preganglionic fibers leave the cord in the anterior nerve roots, join the spinal nerve trunks and enter the ganglion at that level via white ramus. Leaving the ganglion, postsynaptic fibers re enter the spinal nerve via gray ramus, then go on to innervate pilomotor and sudomotor effectors and blood vessels of skeletal muscle and skin. Sympathetic innervation of trunk and limbs is thus carried by the spinal nerves.

The sympathetic distribution to head and neck comes from the three ganglia of cervical sympathetic chain. The unpaired pre vertebral ganglia reside in the abdomen and pelvis anterior to the vertebral column and are primarily the celiac, superior mesenteric, aorticorenal, and inferior mesenteric ganglia. Celiac ganglia is innervated by T5 – T12 and innervates the liver, spleen, kidney, pancreas, small bowel, and proximal colon .The superior mesenteric ganglion

innervates the distal colon, whereas inferior mesenteric ganglion innervates rectum, bladder, and genitals. Adrenal medulla and other chromaffin tissue are homologous to sympathetic ganglia.

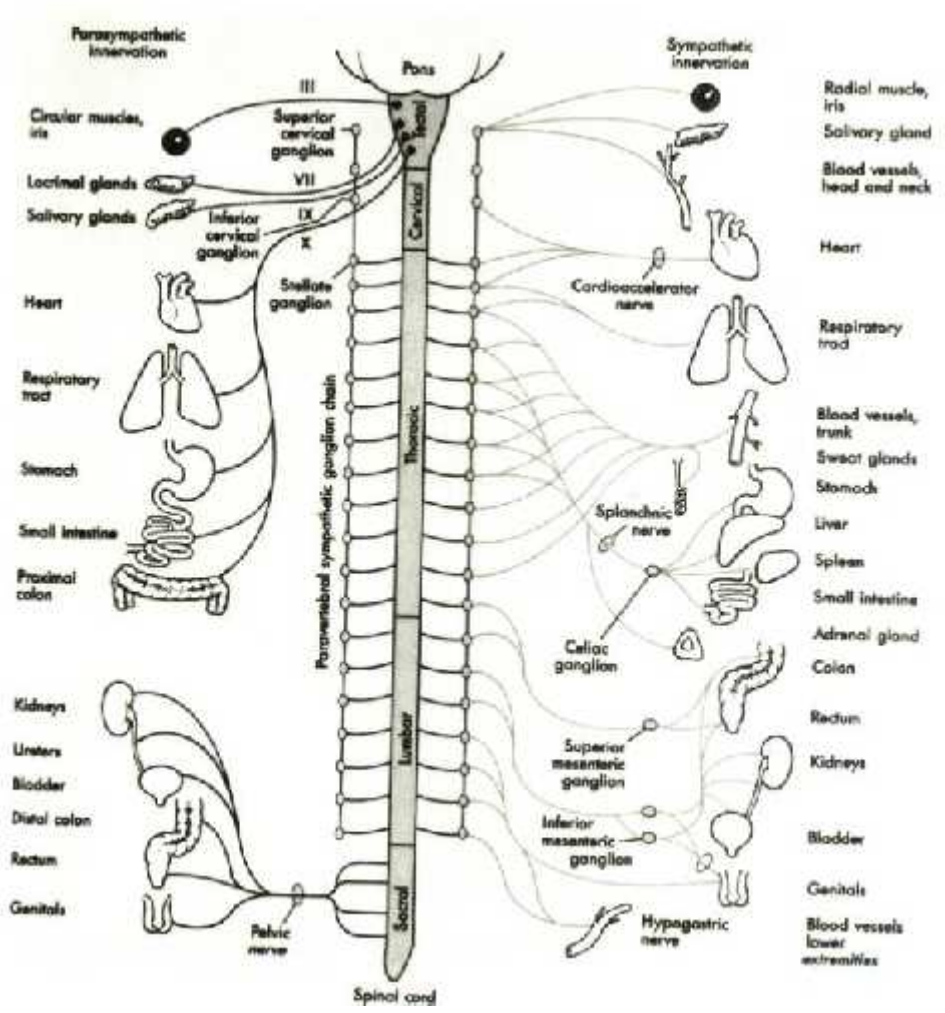


Figure 7: Schematic Representation of Autonomic Nervous System

Sympathetic pre-ganglionic fibers are relatively short because sympathetic ganglia are generally close to the CNS, but they are distant from the effector organs. Therefore, post-ganglionic fibers run a long course before innervating effector organs.

Arterial Circulation

Sympathetic denervation produces arterial and, physiologically more important, arteriolar vasodilatation, although vasodilatation is not maximal. Vascular smooth muscle on the arterial side of the circulation retains a significant degree of autonomous tone following acute, pharmacologically induced sympathetic denervation. As a result, total peripheral vascular resistance (TPVR) decreases only modestly, about 15 to 18% in normal subjects even in the presence of total sympathetic denervation, provided cardiac output, the other determinant of blood pressure, is kept normal. Because TPVR decreases only 15 to 18%, mean arterial pressure decreases only 15 to 18% in the presence of a normal cardiac output.

Venous Circulation

Veins and venules, with only few smooth muscles in their walls, retain no significant residual tone following acute pharmacologic denervation and so they can vasodilate maximally. Whether they do so or not is determined by intraluminal hydrostatic pressure. Intraluminal hydrostatic pressure on the venous side of the circulation depends on the gravity. If the denervated veins lie below the right atrium, gravity causes peripheral pooling of blood in these venous capacitance vessels and if above, gravity causes the blood to flow back to the heart. Preload therefore depends on the position of the patient during spinal anesthesia.

Cardiac Output

Preload is an important determinant of cardiac output. During levels of spinal anesthesia high enough to produce total sympathetic denervation, cardiac output remains unchanged in normovolemic patients as long as they are positioned with the legs elevated above the level of the heart.

Heart Rate

Heart rate characteristically decreases during spinal anesthesia in the absence of autonomically active drugs. The bradycardia is due in part to blockade of preganglionic cardiac accelerator fibers arising from T1 to T4 during high levels of spinal anesthesia. The bradycardia is also mediated by significant decrease in the right atrial pressure and pressure in the great vessels as they enter the right atrium. Placing the patient in a slight head down position increases the venous return, which in turn increases the heart rate. The direct relationship between the right atrial pressure and heart rate during high spinal anesthesia is mediated by the intrinsic chronotropic stretch receptors located in the right atrium and adjacent great vessels. The extent, to which heart rate decreased in response to total sympathetic denervation has been found to be on an average, only moderates (10 to 15%). The mechanism responsible for such cardiovascular responses have been described as the Bezold – Jarisch reflex.[13]

Hypotension

The preceding indicates that slight decreases in the arterial pressure in the range of 15% or so during high spinal anesthesia in normovolemic patients can be

ascribed to decreases in after load that is decreases in TPVR. Severe hypotension, however, can be due only to decreases in cardiac output secondary to decreases in preload associated with peripheral pooling of blood in vasodilated capacitance vessels or to hypovolemia, or to both.

Myocardial Oxygenation

Myocardial oxygen demands decrease during hypotension associated with spinal anesthesia for three reasons:

- After load decreases; the resistance against which the left ventricle ejects blood during systole is decreased.
- Preload decreases; as venous return and cardiac output decrease, so too does the work load of both ventricles because the amount of blood to be ejected per unit time is lessened.
- Heart rate decreases.

Cerebral Blood Flow

Cerebrovascular autoregulatory mechanisms maintain cerebral blood flow in humans at constant levels even in the presence of wide fluctuations of mean arterial pressure. Cerebrovascular autoregulation is independent of the sympathetic nervous system.

Respiratory System

The phrenic nerve supplying the diaphragm arises from the anterior root, root of C3 to C5 and should not be encroached on in spinal analgesia, but phrenic

paralysis can occur. Apnea may be due to medullary ischaemia or due to toxic effects of the drug in extradural blocks. During spinal analgesia breathing becomes quite and tranquil. This is not only due to motor blockade but also due to differentiation with reduction of sensory input to the respiratory center. Lowered arterial and venous tone also lessens the work of the heart and tends to relieve any existing pulmonary congestion. The ventilation perfusion relationship during extradural block is not greatly altered and the effect on respiratory function is relatively small with no evidence of change in functional residual capacity (FRC) or ventilation / perfusion (V/Q) ratio. The pulmonary gas-exchange is preserved. Intercostal paralysis is compensated for by increased descent of the diaphragm, which is made easier by a lax abdomen.

Gastrointestinal System

Pre-ganglionic sympathetic fibers from T5-Ll are inhibitory to the gut. There is no effect on oesophagus, the innervation of which is vagal. The small gut is contracted as sympathetic inhibitory impulses are removed, the vagus being all-powerful. The sphincters are relaxed and peristalsis is active although not more frequent. Pressure within the bowel lumen is increased. Handling of small bowel by the surgeon may cause it to dilate, as may the injection of atropine before the operation. Nausea and vomiting due to the hypotension may occur and usually comes on in waves lasting a minute or so and passes away spontaneously.

Causes of Nausea and Vomiting

- Hypotension
- Hypoxia

- Increased peristalsis
- Traction on nerve endings, especially vagus
- Presence of bile in stomach due to relaxation of pyloric sphincters
- Narcotic analgesics used in pre medication
- Psychological effects

Spleen

The spleen enlarges 2-3 times in high blocks when its sympathetic efferent fibers are paralyzed. Colonic blood supply and oxygen availability are increased in animal following spinal analgesia, perhaps an important factor in the prevention of anastamotic breakdown following gut resection.

Liver

There are no effects of major significance. If the liver is diseased, a decrease in the mean arterial pressure (MAP) affects the liver blood flow and also the metabolism of amide anesthetics.

Endocrine System

Spinal block delays adrenal responses to injury and trauma, so there is no change in the levels of 17- hydroxy corticosteroids.

Spinal block suppresses the hyperglycaemic response to surgery and stress and so is useful in diabetic patients. The response to insulin is augmented, one should be aware of possibility of hypoglycaemia. Infused glucose is well utilised.

Genitourinary System

Sympathetic supply to kidney is from T11 to L1 via the lower splanchnic nerve. Any effects on renal function are solely due to hypotension, renal blood flow is decreased but does not cease until blood pressure has fallen to about 80 mm Hg. These changes are transient and disappear when blood pressure (BP) rises again. The penis is often engorged and flaccid due to paralysis of nervi erigenti (S2 to S3) and this is also a positive sign of a successful block.

Post spinal retention of urine may be moderately prolonged as S2 to S3 contain small autonomic fibers and their paralysis lasts longer that of larger sensory and motor fibers.

Uterus

The tone of uterus is not greatly altered after spinal analgesia in pregnancy. Block of nerves from T11 downwards results in painless labour. In late pregnancy, smaller doses of local anesthetics are required because of decreased extradural space.

Body Temperature

Vasodialtation favours heat loss, absence of sweating favours hyperpyrexia in hot environment, catacholamine secretion is depressed hence heat loss is prevented by metabolism.

PATHOPHYSIOLOGY OF POSTDURAL PUNCTURE HEADACHE

Post-dural puncture headache is common sequelae to LP.[34,35] Bier, the German surgeon who first demonstrated spinal anesthesia, was also the first to propose that PDPH was caused by leakage of CSF through the hole created in the dura mater.[36]

Post-dural puncture headache is a constellation of symptoms seen in patients who have received Intentional or unintentional penetration of the dura mater, either during epidural or spinal placement, or during therapeutic or diagnostic LP. Post-dural puncture headache is defined as bilateral headache that related with position especially improvement during recumbent and worsening during upright position of the body. The pain may be in throbbing in nature, and is extremely variable in severity. The International Headache Society classified it as one that occurs or worsens less than 15 minutes after assuming the upright position and disappears or improves less than 30 minutes after resuming the recumbent position.[37]

The headache is typically transient, develops within two to five days, and spontaneously resolves within two weeks. PDPH typically occurs within 48 hours. The drainage of 20 ml of CSF consistently produces headache. In many cases, the headache is mild in intensity and brief in duration without significant sequelae. PDPH is occasionally severe enough to leave patients bedridden, and it often delays hospital discharge, PDPH can be prolonged with reports of symptoms lasting months or even years, untreated PDPH can lead to the development of persistent cranial nerve palsies and even subdural hematoma.

Early phase head ache (less than 36 hours) due to residual anesthetic could cause intracranial vasoconstriction, and second suggestion was that the effect is caused by the large concentration of glucose acting as an osmotic agent or an irritant.

Firstly, this headache is related with ongoing leakage of CSF through the dural rent made by the LP needle that exceeds the rate of CSF production.[38] The loss of the cushioning effect of CSF allows the brain to sag within the skull. The sagging brain in the upright patient places traction on intracranial vessel. These intracranial vessels transmit pain. The stimulus is refereed along the frontal division of the trigeminal nerves, as well as the ninth and tenth cranial nerves to the occiput and via the upper cervical nerves to the neck and shoulders. Secondly reflex vasodilatation caused by traction on these veins results in a throbbing quality to the pain.

Dural puncture or a rent in the dura with prolonged CSF leakage may cause. Factors affecting postdural puncture headache:

- Age and Sex of the patient
- Experience of the physician performing the procedure
- Single or multiple puncture
- Time to mobilization
- Hydration status of the patient in the perioperative period
- Size of the needle and bevel of the needle
- Body mass index (BMI) and previous history of headache

Higher incidence with the larger needle. Smaller needle size is associated with lesser risk of headache according to a smaller tear in the dura and less

potential for leakage.[39] The incidence is greater in patients with a small BMI.[29] Patients with headache before or during the LP or a history of prior PDPH are at greater risk for PDPH including prevalence and severity.[29,34,35]

Incidence of PDPH decreases about 50%[39] if the bevel of the Quincke is inserted parallel to the dural fibers, rather than perpendicular. The PDPH is considered in the differential diagnosis of Cephalgia, Migraine, Tumor, Infection, Eclapsia, Subdural haematoma, SA hemorrhage, Benign intracranial hypertension (HTN), Malignant HTN, Meningitis, Caffeine withdrawal, Pnemocephalus, intracerebral vein thrombosis, Migraine.

The treatment of PDPH should be based on minimizing the leak of CSF increasing CSF production, or translocating CSF from the spinal to the intracranial compartment. And also PDPH will be relieved by restoration of intracranial CSF volume, but also that cerebral vasoconstrictor might provide symptomatic relief. The treatment of PDPH is traditionally divided into conservative and aggressive treatment.

Conservative treatment: Bed rest, hydration, prone position, abdominal binder, caffeine, oral or parenteral, Theophylline, Sumatriptan, ACTH/ corticosteroids.

Aggressive treatment: intrathecal catheter, Epidural saline, epidural blood patch, prophylactic epidural blood patch, epidural dextran.

The mode of treatment depended on severity. Severity of PDPH was defined by duration of PDPH, or severe disability grading, or Disability based on Numerical Analog Scale grading were;

0 No disability (If no headache occurred)

1 Mild disability (if mild headache, minimally involved activity, not required daytime bed rest)

2 Moderate disability (if moderate headache, moderately involved activity, required partial daytime bed rest)

3 Severe disability (if severe headache, mostly involved activity, required all of daytime bed rest and vomiting).

Patients estimated the Numerical Analog Scale that ranged between 0 through 10 (0: if no pain and 10 if the most severe pain in their life).

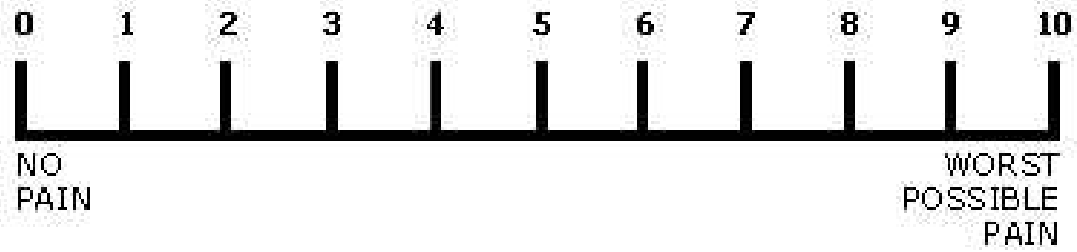


Fig 7. Numerical Analog Scale

Management of post dural puncture headache

Bed rest: Bed rest will provide symptomatic relief of PDPH

Prone position: The prone position can relieve headache in some patients with PDPH. Presumably by, increased intra abdominal pressure translocates CSF from the lumbar spine to the intracranial compartment.

Abdominal binder: It may provide symptomatic relief by the same mechanism as prone positioning. Again, this may not be feasible in patients with an abdominal incision.

Caffeine: Oral a 300mg dose of oral caffeine produces a more significant decrease in headache intensity than placebo the effect is short- lived.[40]

Theophylline: Oral theophylline relieves the symptoms of PDPH. This is presubably due to the cerebral vasoconstrictor effect of the drug.

Sumatriptan: The serotonin agonist sumatriptan is a cerebral vasoconstrictor. 6 mg subcutaneous Sumatriptan can be tried.[41]

Corticosteroids and adrenocorticotropic hormone: There have been the isolated reports of effective treatment of PDPH with corticosteroids of adreno cortico tropic hormone. This therapy has never been evaluated in a prospective randomized study.

Intrathecal catheter: After accidental dural puncture during attempted epidural placement, a catheter can be placed in the subarachnoid space to provide continuous spinal anesthesia. If a spinal catheter is placed, it is critical to

maintain the sterility of the catheter. It is also imperative that all anesthetic providers are aware of the subarachnoid location of the catheter.

Epidural saline: Continuous epidural infusion of normal saline have been reported to relieve the symptoms of PDPH after accidental dural puncture during epidural placement. This technique may be useful in patients, who refuse epidural blood patch, providing symptomatic relief until the dural puncture spontaneously heals.

Epidural blood patch (EBP): It has been proposed as a gold standard for the treatment of PDPH with the early reports suggesting a success rate of as high as 95% and a recent metaanalysis suggest that the success rate of EBP may actually be as low as 65%. Epidural blood patch is least likely to be successful in patients with larger dural punctures, the very patients in whom headache is most likely to be reverse and persistent. In those patients with recurrence of headache after EBP a repeat procedures is usually successful. The most commonly used technique for EBP involves an interlaminar injection of approximately 20 ml of blood, although the suggested volume may be as small as two to three ml.[42,43]

There are three theories explaining the mechanism by which an EBP can provide symptom relief. The first is that the EBP creates a blood clot adherent to the dura mater, there by sealing a hole in the dura and preventing CSF leak. The second theory is that the injected blood leads to an increase in intra spinal pressure, effectively reducing traction on the brain and meninges.

The third hypothesis is that mixing blood with the CSF leads to a rapid coagulation response, sealing the dural tear even if it is far from the blood patch injection site.

Prophylactic Epidural blood patch: Epidural blood patch administered via an epidural catheter placed subsequent to accidental dural puncture has been reported to decrease the incidence of PDPH by as much as half. Epidural dextran in those patients who cannot receive EBP because of fever, or who refuse EBP because of religious reasons epidural dextran has been used with some success. This modality has never been studied in prospective fashion, and concerns about the potential for neurotoxicity and the risk of allergic reaction remain.

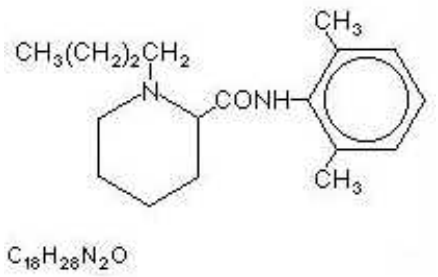
PHARMACOLOGY OF DRUG USED IN SUB ARACHNOID BLOCK[44]

Bupivacaine (Marcain, Marcaine and Sensorcaine)

Bupivacaine, an amino amide local anesthetic was first synthesized in Sweden by A.F Ekenstam and his colleagues in 1957. First report of its use was in 1963 by L.J Teluvio. It is one of the long acting local anesthetic agents available, which is extensively used for intrathecal, extradural and peripheral nerve blocks. It is a white crystalline powder soluble in water.

Chemistry

The molecular weight of chloride salt is 325 and that of base form is 288. pH of plain solutions varied between 4.5 to 6 and pKa 8.16.



Chemical Structure

Bupivacaine has an IUPAC nomenclature of 1-butyl-n-(2, 6-dimethylphenyl) piperidine-2-carboxamide.

Properties

The base is sparingly soluble, but the hydrochloride salt is readily soluble in water. In spinal anesthesia, the onset of action is about three to four minutes

and complete anesthesia occurs in five minutes and lasts for 3.5 to 4 hours. Because bupivacaine is an amide, the liver is the primary site of metabolism. The most of the drug is metabolized by N-dealkylation.

Physiochemical Properties

- Molecular formula C18 H28 N2O
- Molecular weight 288.43 g/mol
- Solubility in water 25 mg/ml
- pH of saturated solution 5.2
- pKa 8.1
- Specific gravity 1.021 at 37°c

Mechanism of Action

Mechanism of action is similar to that of any other local anesthetic. The primary action is on the cell membrane axon, on which it produces electrical stabilization. Bupivacaine prevents the generation and the conduction of the nerve impulse. Bupivacaine blocks conduction by decreasing or preventing the large transient increase in permeability of excitable membranes to sodium that normally is produced by a slight depolarization of the membrane. This action of Bupivacaine is due to its direct interaction with the voltage gated sodium channels. As the anesthetic action progressively develops in a nerve, the threshold for electrical excitability gradually decreases, the rate of rise of the action potential declines, impulse conduction slows, and the safety factor for conduction decreases, these factors decrease the probability of propagation of the

action potential and nerve conduction fails. The mechanism by which local anaesthetics block sodium conductance is as follows:

Local anesthetics in the cationic form act on the receptors within the sodium channels on cell membrane and block it. The local anesthetics can reach the sodium channel either via the lipophilic pathway directly across the lipid membrane, or via the axoplasmic opening. This mechanism accounts for 90% of the nerve blocking effects of amide local anesthetics.

The second mechanism of action is by membrane expansion. This is a non-specific drug receptor interaction. Bupivacaine is available in the following concentrations:

- 0.25%. 0.5% and 1%
- 0.25% and 0.5% solution in isotonic saline
- 0.5% solution in 8% dextrose
- Dosage is two mg/kg limited to 150 mg in four hours. The intrathecal minimum local analgesic dose of Bupivacaine is 2.37 mg.

Anesthetic Potency

Hydrophobicity appears to be a primary determinant of intrinsic anesthetic potency and Bupivacaine is highly hydrophobic, hence is very potent.

Onset of Action

The onset of conduction blockade is dependent on the dose or concentration of the local anesthetic

Differential Sensory Motor Blockade

Bupivacaine in low concentration (0.125%) produces acceptable analgesia with only mild muscular weakness.

Pharmacokinetics

The concentration of Bupivacaine in blood is determined by the amount injected, the rate of absorption from the site of injection, the rate of absorption from the site of injection, the rate of tissue distribution and the rate of biotransformation and excretion of Bupivacaine.

Absorption

The site of injection, dose and addition of a vasoconstrictor determine the systemic absorption of Bupivacaine .The maximum blood level of Bupivacaine is related to the total dose of drug administered from any particular site. Absorption is faster in areas of high vascularity.

Distribution

The two-compartment model can describe this. The rapid distribution phase α is believed to be related to uptake by rapid equilibrating tissue i.e., tissues that have high vascular perfusion. The slow distribution phase β is mainly a function of distribution to slowly equilibrating tissue, biotransformation and excretion of the compound.

More highly perfused organs show higher concentrations of the drug. Bupivacaine is rapidly excreted by lung tissue. Though skeletal muscle does not show any particular affinity for bupivacaine it is the largest reservoir of the drug.

Distribution Characteristics

- T1/2 α 2-7 minutes (uptake by rapid equilibrium tissue)
- T1/2 β 28 minutes (distribution by slowly perfused tissues)
- T1/2 γ 3-5 hours (metabolism and elimination)
- VDSS 72 liters (volume of distribution at steady state)

Actions

Central Nervous System

Bupivacaine readily crosses the blood brain barrier causing CNS depression following higher doses. The initial symptoms involve feeling of light-headedness and dizziness followed by visual and auditory disturbances. Disorientation and occasional feeling of lightheadedness may occur. Objective signs are usually excitatory in nature, which includes shivering, muscular twitches and tremors, initially involving muscles of the face (perioral numbness) and part of extremities. At still higher doses cardiovascular or respiratory arrest may occur. Acidosis increases the risk of CNS toxicity from Bupivacaine, since an elevation of $PaCO_2$ enhances cerebral blood flow, so that more anesthetic is delivered rapidly to the brain

Autonomic nervous system

Bupivacaine does not inhibit the Nor Adrenaline uptake and hence has no sympathetic potentiating effect. Myelinated preganglionic B fibers have a faster conduction time and are more sensitive to action of Bupivacaine. When used for conduction blockade, all local anesthetics, particularly Bupivacaine produces higher incidence of sensory than motor fibers.

Cardiovascular System

The primary cardiac electrophysiological effect of a local anesthetic is a decrease in the maximum rate of depolarization in Purkinje fibers and ventricular muscle. This action by Bupivacaine is far greater compared to Lignocaine. Also, the rate of recovery of block is slower with Bupivacaine. Therefore there is complete restoration of Vmax between action potential particularly at higher rates. Therefore Bupivacaine is highly arrythmogenic. Bupivacaine reduces the cardiac contractility. This is by blocking the calcium transport. Low concentration of Bupivacaine produces vasoconstriction whereas high doses cause vasodilatation.

Respiratory System

Respiratory depression may be caused if excessive plasma level is reached which in turn results in depression of medullary receptor center. Respiratory depression may be also caused by paralysis of respiratory muscles of diaphragm as may occur in high spinal or total spinal anesthesia.

Biotransformation And Excretion

Bupivacaine undergoes enzymatic degradation primarily in the liver. The excretion occurs primarily via the kidney. Renal perfusion and factors affecting urinary pH affect urinary excretion. Less than 5% of Bupivacaine is excreted via the kidney unchanged through urine. The major portion of injected agent appears in urine in the form of 2, 6 pipecolyoxylidine which is a n-dealkylated metabolite of bupivacaine. Renal clearance of the drug is related inversely to its protein binding capacity and pH of urine.

Adverse effects are encountered in clinical practice mostly due to overdose, inadvertent intravascular injection or slow metabolic degradation.

Adverse Effects

CNS: Nervousness, dizziness, blurring of vision or tremors, drowsiness, convulsions and respiratory arrest.

CVS: Myocardial depression, hypotension, arrhythmia, ventricular type conduction defect, SA node depression and cardiac arrest

Allergic reactions: Urticaria, bronchospasm, hypotension

Other: Constriction of pupil and tinnitus

SPINAL NEEDLES[11]

Whitacre Pencil Point Spinal Needles

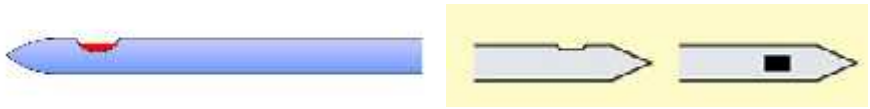


Figure 8: Whitacre's Pencil Point Needle

Development of the pencil-point needle Thirty-seven years after Whitacre's development of the pencil-point needle, Sprotte published his paper on a modification of the Whitacre needle. Sprotte modified the needle by increasing the size of the distal orifice to combat the problems of slow CSF flow, difficulty in aspiration and resistance to injection of the local anaesthetic solution. The wider hole also allowed greater mixing of the local anaesthetic solution with the CSF to allow a more even distribution of local anaesthetic solution in the subarachnoid space. The tip of the Sprotte needle was elongated in an attempt to allow more gradual separation of dural fibres and therefore less CSF leakage and a decreased incidence of PDPH. The size of the lateral hole sometimes caused the orifice to straddle the dural layers, resulting in partial loss of the local anaesthetic solution and incomplete blocks. The width of the hole also left the distal portion of the tip relatively prone to damage, including fracturing. Studies of the Sprotte needle concluded that the needle orifice area could be decreased to the cross-sectional area of the cannula without affecting flow rate. These claims were strongly refuted by Sprotte and by the early 1990s the Sprotte needle had been modified to the needle that is in common use today.

Pencil-point needles

Once the suggestion that dural fibres were less likely to be damaged by non-cutting tips had been published. Kirschner and Rovenstine's needles, which had a lateral orifice, contributed to the next phase of needle tip design. Hart and Whitacre are commonly associated with the design of the first closed-ended, lateral orifice, pencil-point needle to decrease the incidence of PDPH. The needle was of fine gauge, with a solid non-cutting tapering point and an orifice on the conical surface two mm from the actual tip of the needle. He quoted a PDPH rate of nine percent for the non-cutting needle as opposed to 32% for a cutting needle. The Haraldson needle failed to gain the recognition it deserved, possibly because it was manufactured in only small numbers locally, so it was the writers of the next description of this sort of needle who have received the recognition.

The original Whitacre needle had its problems, namely, the orifice was very small, making aspiration and injection difficult, and the stylet did not occlude the orifice, which could therefore become blocked with tissue. However, the basic design was a success, and the Whitacre needle is still in common use today with only a few minor modifications to its original design. Even after the widespread acceptance of the pencil-point needle and the acknowledgement that it was an advance in the design of spinal needles, there were still modifications of cutting bevel needles being developed, mainly the introduction of finer gauges.

Quincke Needles

Development of the cutting spinal needle tip In 1891, Quincke described a standardized technique of lumbar puncture for the release of cerebrospinal fluid (CSF) for diseases associated with increased intracranial pressure. He used a needle of which it is difficult to find a description, except that it was a sharp, bevelled, hollow needle.

Figure 9: Quincke Needle

The next major development in the history of spinal anaesthesia was the work of Augustus Karl Gustav Bier. In 1898, both he and his assistant underwent spinal anaesthesia with cocaine, the assistant first injecting Bier, and then Bier his assistant. The first procedure resulted in Bier experiencing a lancinating pain in his leg while his assistant struggled to connect the syringe to the needle, with a resultant significant leakage of CSF and cocaine solution. Bier then performed a successful block on his assistant. Over time, he developed his own needle. He felt that the use of introducers and dilators for the insertion of the finer needles previously used was cumbersome, and he designed a larger bore needle that needed no introducer. The Bier spinal needle was 15G or 17G, with a long, cutting bevel and a sharp point.

Soon after the introduction of the Bier needle, the importance of the size of the needle and the shape of its bevel was recognised. Bier's needle was

criticised for causing pain on insertion, leaving a large hole in the dura and causing an extradural loss of local anaesthetic solution because the orifice could straddle the dura. Bainbridge described a needle in 1900 that was attached to a metal syringe. It had a small circular hub, a short, sharp cutting bevel and a stylet with a matching bevel. It was made of a flexible metal. Barker was the next pioneer in needle design. He described his technique in 1907. Initially, he used a modified Bier needle but, as he appreciated the problems associated with a long bevel, he designed a hollowed-out point to 'secure sharpness without lengthening the terminal opening'. He stated that 'the object of this is that when the puncture is made, the whole of the open end of the needle shall lie within the dura without the point being pushed so far as to wound structures within the sac. It is obvious that if the open end of the needle were only partially through the dura the spinal fluid might run freely enough through the needle, but that when the analgesic compound was injected part of it might enter the sac while part of it escaped in the extradural space. Barker developed a blunt cannula to fit inside the Bier needle that projected one mm beyond the needle tip. It was placed after dural puncture by the Bier needle and was connected to a syringe of local anaesthetic solution. The blunt tip was designed to limit damage to intradural structures. At a later, unknown date, Barker designed a needle without the inner cannula, referred to as the Barker needle. This 18 G or 19 G needle had a sharp, medium-length bevel and a stylet with a matching bevel. It was a large firm needle that was associated with a significant incidence of PDPH.

As early as 1898, Sicard realised that the cause of PDPH was the loss of CSF through dural tears. In 1914, Ravaut advised the use of finer needles to limit

the size of the dural tear. In 1914, Babcock described a needle that was closer in design to the original Corning needle but with a finer cannula to limit the incidence of PDPH. It had a sharp, medium-length bevel with a matching stylet. It was made of iridised platinum or gold and was 20 G in diameter. Referred to as the Quincke Babcock needle, it was a very successful needle design and became the standard spinal needle for comparative studies.

Gaston Labat was a well-respected French clinician who moved to America. He was one of the pioneers of the widespread acceptance of spinal anaesthesia in the 1920s in both Europe and the United States of America. He designed a spinal needle that was made of unbreakable nickel. It was a medium-gauge cannula with a short, sharp bevel and matching stylet, with the tip ground to match the bevel of the cannula. Labat's theory was that the shorter bevel acted as a wedge, pushing tissues aside rather than cutting them, and therefore minimizing damage to the dura.

FAILED SPINAL ANAESTHESIA[21]

Spinal anaesthesia is generally regarded as one of the most reliable of regional block methods: the needle insertion technique is relatively straightforward, with CSF providing both a clear indication of successful needle placement and a medium through which local anaesthetic solution usually spreads readily. However, the possibility of failure has long been recognized, Most experienced practitioners would consider the incidence of failure with spinal anaesthesia to be extremely low, perhaps less than 1%. However, a figure as high as 17% has been quoted from an American teaching hospital, yet most of

the failures were judged to be ‘avoidable’. The clear implication is that careful attention to detail is vital, and it has been shown that a failure rate of, one percent is attainable in everyday practice. Minimizing the incidence of failure is obviously a pre-requisite for gaining the benefits of spinal anaesthesia, and prevention must start with full recognition of the potential pitfalls so that clinical practice can be tailored to their avoidance.

In general terms, block failure is usually ascribed to one of three aspects: clinical technique, inexperience (of the unsupervised trainee especially), and failure to appreciate the need for a meticulous approach. However, such broad categories reveal little about the many detailed ways in which an intrathecal injection can go astray within each of the five phases of an individual spinal anaesthetic, these being, in sequence, lumbar puncture, solution injection, spreading of drug through CSF, drug action on the spinal nerve roots and cord, and subsequent patient management.

Mechanisms and their prevention

Failed lumbar puncture - Inability to obtain CSF, sometimes referred to as a ‘dry tap’, is the only cause of failure which is immediately obvious. A needle with a lumen blocked at the outset is a theoretical possibility, but is most unlikely with modern equipment. However, both needle and stylet must be checked for correctness of fit before use, and the needle should not be advanced without the stylet in place because tissue or blood clot can easily obstruct the fine bore needles used now.

Positioning - The patient is placed on a firm surface; the lumbar laminae and spines are 'separated' maximally by flexing the whole spine (including the neck), the hips, and knees; rotation and lateral curvature of the spine are avoided; these points apply to lumbar puncture in both sitting and lateral horizontal positions;

Needle insertion - Although its accurate identification can be difficult using clinical land-marks, what is judged to be the third lumbar inter-space is used usually. With the midline approach, insertion should start precisely in the mid-line, mid-way between the posterior spines, with the needle shaft at right angles to the back in both planes. Small, incremental changes in needle angle should be made only if there is resistance to advancement; if resistance is met, cephalad angulation should be tried first, and such angulation may be appropriate from the start if the patient is unable to flex fully (for example, the obstetric patient at term).

Adjuncts - A calm, relaxed patient is more likely to assume and maintain the correct position, so explanation (before and during the procedure) and gentle, unhurried patient handling are vital; light anxiolytic premedication contributes much to relaxing the patient; local anaesthetic infiltration at the puncture site must be effective without obscuring the landmarks, but must include both intradermal and sub cutaneous injection.

Solution injection errors - The appearance of CSF in the needle hub is an essential pre-requisite for spinal anaesthesia, but it does not guarantee success, which also requires that a fully effective dose is both chosen and actually deposited in the CSF.

Loss of injectate - The Luer connection between syringe and needle provides a ready opportunity for leakage of solution. A particular variant of this problem being a leak through a defect at the junction of needle hub and shaft. Given the small volumes involved, the loss of even a few drops may cause a significant decrease in the mass of drug reaching the CSF, and thus in its effectiveness. To avoid this, it has long been conventional teaching that the syringe containing the injectate must be inserted very firmly into the hub of the needle, and that a subsequent check is made that no leakage occurs.

Misplaced injection - Needle and syringe must be connected firmly, but great care should be taken to avoid either anterior or posterior displacement of the needle tip from subarachnoid to epidural space, where deposition of a spinal dose of local anaesthetic will have little or no effect. Fluid aspiration, after attachment of the syringe, should confirm free flow of CSF and, thus, that the needle tip is still in the correct space. To prevent displacement at any stage, it has been advocated that the dorsum of one hand should be anchored firmly against the patient's back and the fingers used to immobilize the needle, while the other hand is used to manipulate the syringe.[29]

Anatomical abnormality - Intrathecal spread is governed by interplay between solution physical characteristics, gravity, and the configuration of the vertebral canal. Anatomical abnormalities that lead to problems with spread can be both overt and covert. The curves of the vertebral column are integral to solution spread and any obvious abnormality, kyphosis, or scoliosis, may interfere with the process.

Ineffective drug action - The last possible explanation for a failed spinal is that the solution actually injected reaches the target nerves, but is inactive or ineffective, with a variety of explanations being possible.

Chemical incompatibility - The mixing of two different pharmaceutical preparations also raises the possibility of ineffectiveness as a result of interaction between local anaesthetic and adjuvant. Chemical reaction might generate an obvious precipitate, but another possibility is that the pH of the local anaesthetic solution becomes even lower than it was to start with. This would decrease the concentration of the un-ionized fraction that is what diffuses into nerve tissue and, unless the solution mixes well with CSF, a decreased effect could result.

Management of failure

Failure of a spinal anaesthetic is an event of significant concern for both patient and anaesthetist even when it is immediately apparent, but it can have serious consequences (clinical and medico-legal) if the problem only becomes evident once surgery has started. If there is any doubt about the nature or duration of the proposed surgery, a method other than a standard spinal anaesthetic should be used. If a spinal anaesthetic does fail in some way, the management options are limited; so, the first rule is to expend every effort in prevention.

Prevention is better than cure

Having made the decision to use a spinal anaesthetic, the block should be performed with meticulous attention to detail as has been indicated above.

The failed block

The precise management of the failed block will depend on the nature of the inadequacy and the time at which it becomes apparent. The slower the onset of either motor or sensory block, the more likely is the block to be inadequate, so the more detailed this assessment should be. While the onset of spinal anaesthesia is rapid in most patients, it can be slow in some; so, 'tincture of time' should always be allowed.[10] However, if most of the expected block has not developed within 15 minute, some additional manoeuvre is almost certainly going to be needed. The possibilities, their explanations, and suggested immediate responses are as follows:

1. No block: the wrong solution has been injected, it has been deposited in the wrong place, or it is ineffective. Repeating the procedure or conversion to general anaesthesia are the only option. If, after operation, the patient has significant pruritus, it is likely that only an opioid was injected.

2. Good block of inadequate cephalad spread: the level of injection was too low, anatomical abnormality has restricted spread, or some injectate has been misplaced. If a hyperbaric solution was used, flex the patient's hips and knees and tilt the table head down. This straightens out the lumbar curve, but maintains a cephalad 'slope' and allows any solution 'trapped' in the sacrum to spread further.

3. Good, but unilateral block: this is most likely because of positioning, but it is possible that longitudinal ligaments supporting the cord have blocked spread.

4. Patchy block: This term is used to describe a block that appears adequate in extent, but the sensory and motor effects are incomplete.

5. Inadequate duration: the most likely explanation is that for one of several reasons an inadequate dose of local anaesthetic was delivered to the CSF.

METHODOLOGY

The present study was conducted in the Department of Anaesthesiology, K.L.E.S. Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2009 to December 2009.

Source of Data

Patients between 20 to 60 years of age with ASA grade I and II undergoing lower abdobinal and lower limb surgeries during the study period at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Nehru Nagar, Belgaum.

Study design

A one year randomized clinical trial.

Study Period

One year from January 2009 to December 2009.

Sample Size and sampling procedure

176 patients are selected in each group based on the following calculation.

$$\text{Sample Size} = \frac{2 \text{ X } (Z\alpha + Z\beta)^2 \text{ X } p \text{ X } q}{d^2}$$

$$= \frac{2 \text{ X } (1.96 + 0.84)^2 \text{ X } 8.5 \text{ X } 91.5}{8 \text{ X } 8}$$

$$= 176$$

Randomization procedure

Based on the above calculation a total of 352 patients are allocated randomly into group A and group B by using a computer generated randomization table.

Selection Criteria

Inclusion criteria

- Patients undergoing lower abdominal surgeries.
- Patients undergoing lowerlimb surgeries.
- Age between 20 to 60 years.
- No clinically significant cardiovascular, respiratory and central nervous system disease (ASA Grade I and II).

Exclusion criteria

- Patient refusal.
- Allergy to Bupivacaine.
- History of bleeding diathesis.
- Severe to moderate hypotension.
- Arrhythmias.
- Infection at the site of spinal needle insertion.
- Severe spinal abnormalities like spina bifida, meningocele.
- Raised intracranial tension, hydrocephalus.

Procedure

The study was approved and ethical clearance was obtained from Human Ethics Committee, Jawaharlal Nehru Medical College, Belgaum. After finding the suitability according to selection criteria patients were selected for the study and briefed about the nature of the study, the interventions used and written informed consent was obtained (Annexure–I). Further, descriptive data of the patients like name, age, sex, detailed history, were obtained and recorded on predesigned and pretested proforma (Annexure-II).

Pre-anaesthetic evaluation

A thorough pre-anaesthetic evaluation was performed by taking history and clinical examination. In all the patients, height, weight, basal heart rate, respiratory rate and blood pressure were measured and recorded. Investigations like complete blood count, urine for albumin, sugar and microscopy were done. Blood sugar, electrocardiogram and chest x-ray were performed.

Anaesthesia procedure

Intravenous (IV) line was secured using 18 G cannula. All patients were preloaded with 500 ml of ringer lactate solution. Electrocardiogram (ECG), non-invasive blood pressure (NIBP), oxygen saturation (SPO_2) were monitored. Patients of ASA I - II aged 20 to 60 yrs undergoing lower abdominal and lower limb surgeries were taken. Patients were allocated into group A and group B by computer generated randomisation table. Group A patients received spinal

anaesthesia with 27 G Quincke's spinal needle and Group B received spinal anaesthesia with 27 G Whitacre's spinal needle.

Under strict aseptic precautions, using midline approach 27 G Quincke's or 27 G Whitacre's spinal needle inserted into L_3–L_4, sub arachnoid space. Three ml of 0.5% heavy bupivacaine injected after confirming for free flow of CSF.

Experienced anaesthesiologists performed the blocks. Failure of spinal anaesthesia was defined as either inability to elicit free flow of CSF after three attempts or clearly inadequate analgesia for surgery at 15 minutes after giving local anaesthetic.

The number of attempts of dural puncture and the presence or absence of tactile identification, usually described as a click phenomenon on dural puncture.

Heart rate (HR), NIBP, SPO_2 were recorded every three minutes for 15 minutes, then every five minutes for 30 minutes and thereafter every 10 minutes. All patients were seen on the day of surgery and every day for three days. They were questioned about postdural puncture headache or were contacted by telephone if discharged early. Criteria for post dural puncture headache was;

a. Onset after spinal anaesthesia within 48 to 72 hours.
b. Mostly located to occipital or frontal region.
c. Aggravated by erect or sitting position, coughing and straining.
d. Relieved by lying flat.

PDPH assessed on the basis of standard numeric analog scale (NAS) 0-100.

1. Mild (0-33) when sitting or ambulating.
2. Moderate (34-66) when sitting.
3. Severe (67-100) when supine.

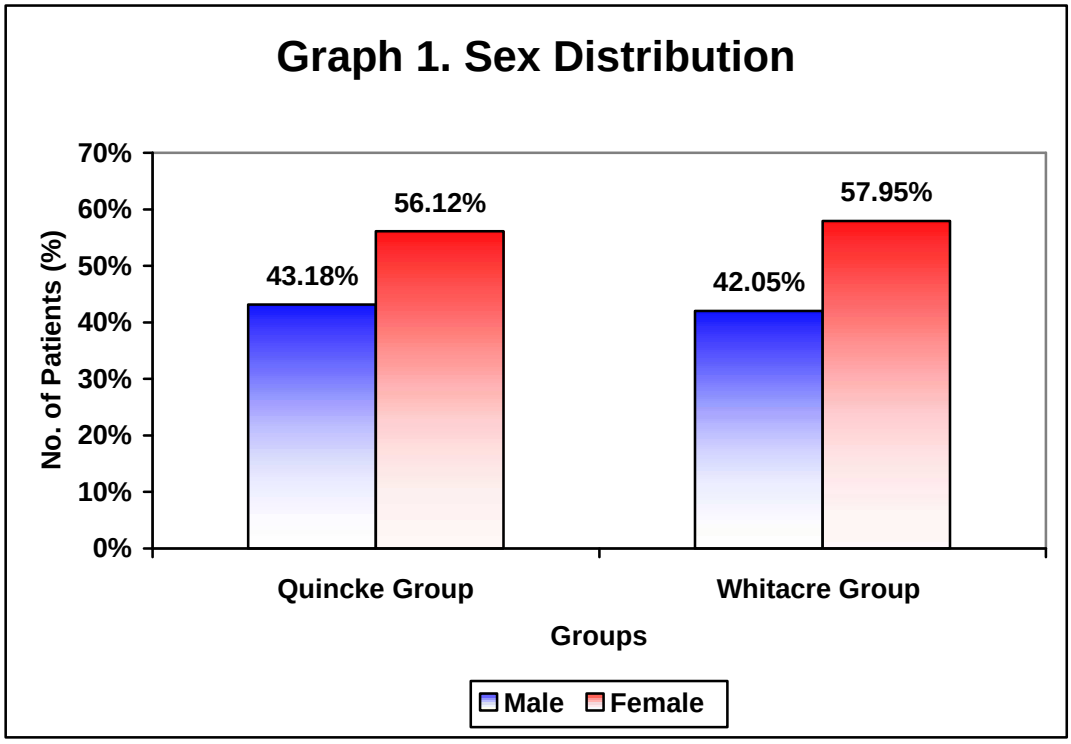
Statistical Analysis

The data obtained was tabulated and analysed for rates, ratios and percentages. The test of proportion was used for incidence of PDPH and Chi square test was applied for number of attempts and failed spinal anaesthesia.

RESULTS

Table No. 2. Sex distribution

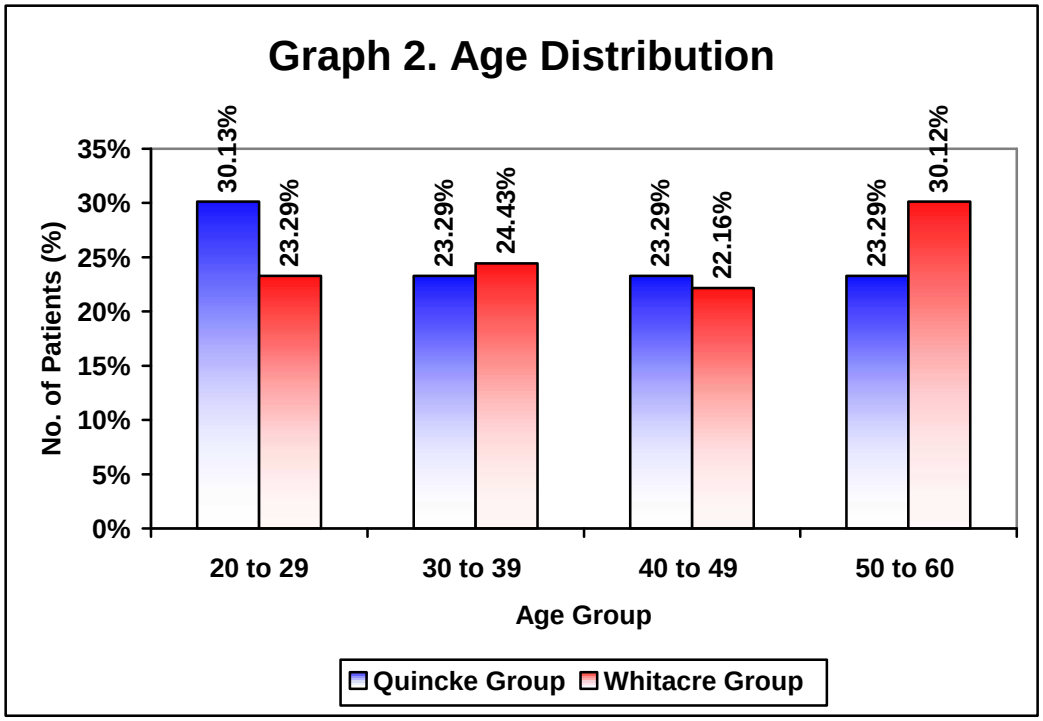
Gender	Quincke group		Whitacre Group	
	Number	Percentage	Number	Percentage
Male	76	43.18%	74	42.05%
Female	100	56.12%	102	57.95%
Total	176	100%	176	100%



In this study females outnumbered males in both the groups (56.12% and 57.95%) with male to female ratio of 1:1.31 in Quincke group and 1:1.37 in Whitacre group.

Table No. 3. Age distribution

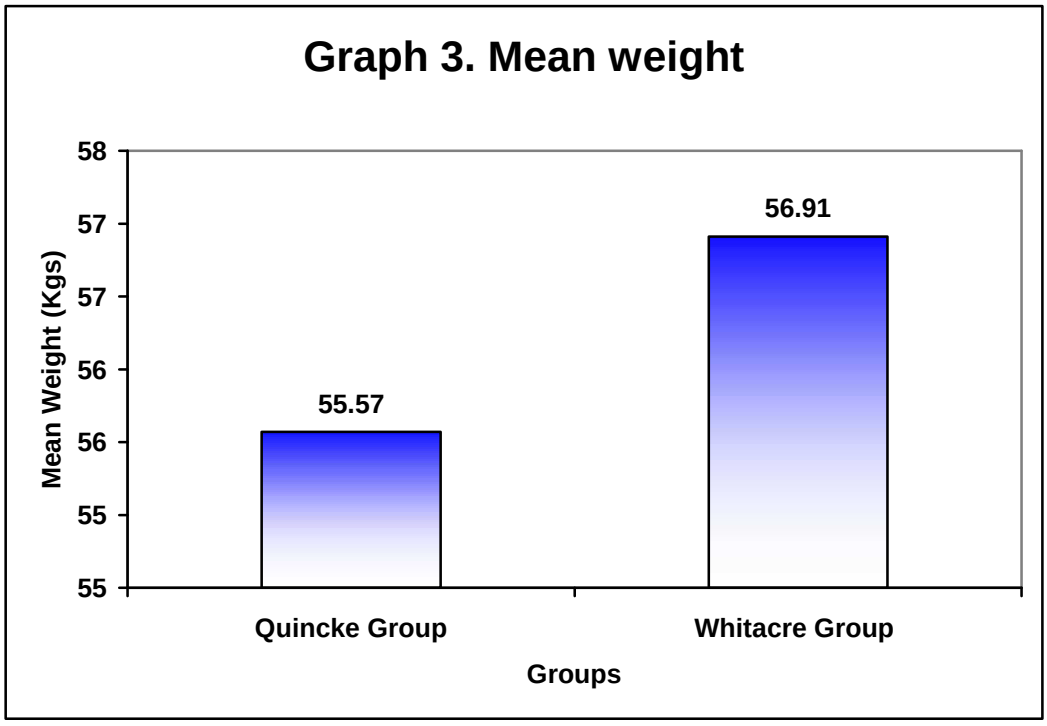
Age (Years)	Quincke group		Whitacre Group	
	Number	Percentage	Number	Percentage
20 to 29	53	30.13%	41	23.29%
30 to 39	41	23.29%	43	24.43%
40 to 49	41	23.29%	39	22.16%
50 to 60	41	23.29%	53	30.12%
Total	176	100%	176	100%



In the present study majority (30.13%) of the patients had age between 20 to 29 years in Quincke group compared to 30.12% of the patients in the age ranging from 50 to 60 years in Whitacre group. The mean age in Whitacre group was 40.74 ± 12.25 years whereas in Quincke group it was 38.58 ± 11.95 years.

Table No. 4. Mean Weight

	Quincke group		Whitacre Group	
	Mean	**S.D.**	**Mean**	**S.D.**
Weight	55.57	11.95	56.91	7.43

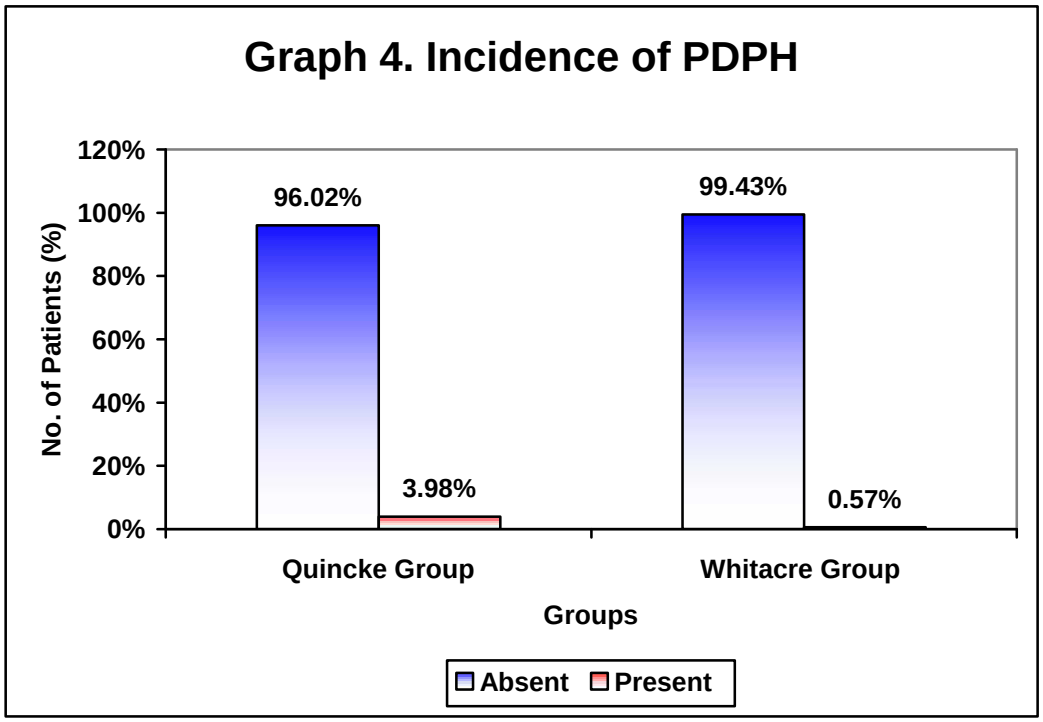


In this study the mean weight of the patients in Quincke group was 55.57 ± 11.95 kg whereas in Whitacre group it was 56.91 ± 7.43 Kg.

Table No. 5. Incidence of PDPH

Grade	Quincke group		Whitacre Group	
	Number	**Percentage**	**Number**	**Percentage**
Present	7	3.98%	1	0.57%
Absent	169	96.02%	175	99.43%
Total	**176**	**100%**	**176**	**100%**

p=0.0319 (Test of Proportion)

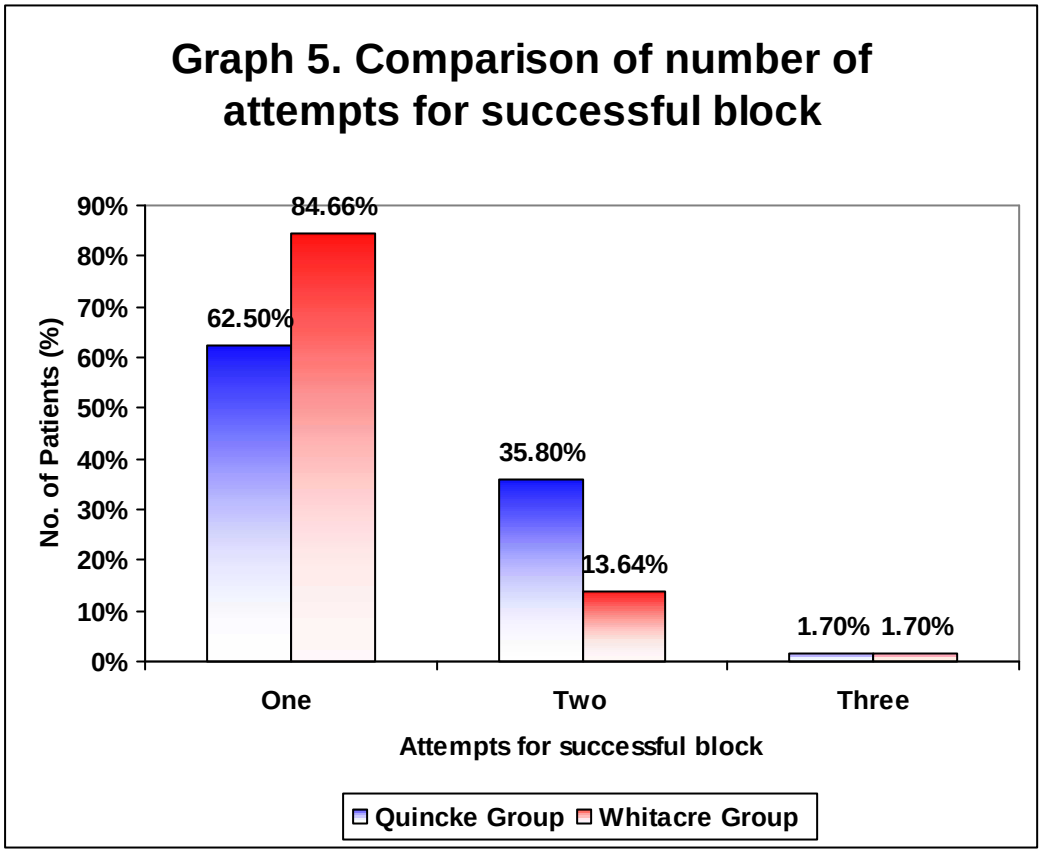


In the present study the 3.98% patients had PDPH in Quincke group and 0.57% patients in Whitracre group and this difference was statistically significant using test of proportion (p=0.031).

Table No. 6. Comparison of number of attempts for successful block

No. of attempts	Quincke group		Whitacre Group	
	Number	**Percentage**	**Number**	**Percentage**
One	110	62.5%	149	84.66%
Two	63	35.8%	24	13.64%
Three	03	1.70%	03	1.70%
Total	**176**	**100%**	**176**	**100%**

p=0.0001 (Chi square test)

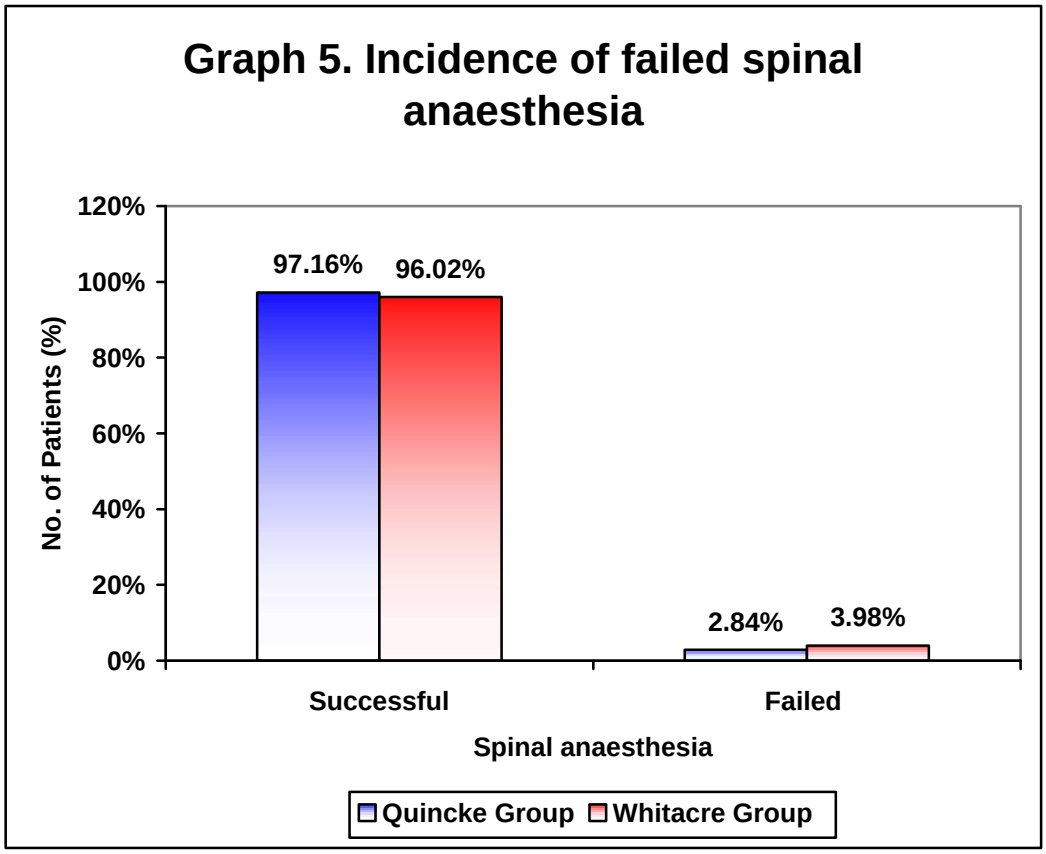


In the present study, 62.5% of the patients in Quicke group required one attempt, 35.80% required two attempts and 1.70% required three attempts for successful block. Whereas in Whitacre group 84.66%, 13.64% and 1.70% of the patients required one, two and three attempts respectively. When these values were compared using chi-square test significant association was recorded between the type of needle and number of attempts (p=0.0001).

Table No. 7. Incidence of failed spinal anaesthesia

Spinal anaesthesia	Quincke group		Whitacre Group	
	Number	**Percentage**	**Number**	**Percentage**
Successful	171	97.16%	169	96.02%
Failed	05	2.84%	07	3.98%
Total	**176**	**100%**	**176**	**100%**

p=0.557 (Chi Square Test)



In the present study incidence of 2.84% failed spinal anaesthesia in Quincke Group and 3.98% in Whitacre Group was recorded. However, no statically significant association between the type of the needle and the number of failed spinal anaesthesia could be recorded (p=0.557).

DISCUSSION

Spinal anaesthesia is one of the most commonly used technique in anaesthesia. It is economical, safe, easy and needs less sophisticated anaesthetic equipments, drugs, post operative care hence preferred over general anaesthesia and most popular because of its profound analgesia and muscle relaxation.[2]

Loss of CSF from the punctured site produces low CSF pressure which in turn leads to intracranial venous dilatation resulting in an increase in brain volume in the upright position. There occurs a difference in CSF volume and also pressure difference between the intracranium and intravertebral part of the subarachnoid space. Venous dilation and compensatory increase in brain volume will result in brain sag which in turn will exert traction and stimulate pain sensitive anchoring structures like dural vessels, basal dura and tentorium cerebelli, causing post spinal headache.[13]

Pain arising from the tentorium cerebelli is transmitted by the fifth nerve and from the structures on or below the inferior surface of the tentorium is transmitted by the ninth, tenth cranial nerves and the upper cervical nerves. Post dural puncture headache due to low cerebrospinal fluid pressure is differentiated from other headaches as it aggravates on sitting, standing, moving around, coughing and straining. Inadequate intake of fluid and conditions causing loss of fluids such as diarrhoea, vomiting, haemorrhage, sweating and lactation tend to make the condition worse.[3]

August Bier first reported post dural puncture headache. Post dural puncture headache would be familiar to anyone in practice today.[46] Needle tip configuration and needle size greatly influenced incidence of headache in patients.[39]

One year randomized clinical trial was an attempt to compare role of two needles that is Quincke 27 G and Whitacre 27 G needles with respect to the incidence of PDPH as well as the number of attempts required to administer successful subarachnoid block (SAB).

In the present study, 56.12% and 57.95% were females whereas 43.18% and 42.05% were males in Quincke and Whitacre groups respectively whereas, in a study[15] 108 were males and 91 were females in Quincke group and 116 were males and 83 were females in Whitacre group. Another study[14] reported male predominance that is 74 males and 23 females in Quincke group and 71 males and 26 females in Whitacre group.

In this study the mean age in Quincke group was 38.58 ± 11.95 years whereas in Whitacre group it was 40.74 ± 12.25 years. A study[15] reported similar results that is, mean age of 37 ± 14 years in Quincke group and 36 ± 15 years in Whitacre group whereas, another study[14] reported average age of 32.5 years in Quincke group and 31.7 years in Whitacre group.

In the present study mean weight of the patients in Quincke group was 55.57 ± 11.95 kg whereas in Whitacre group it was 56.91 ± 7.43 Kg. Similar findings were reported by authors in a study[13] that is 52.8 ± 5.20 in Quincke

group and 52.6 ± 6.10 in Whitacre group. Whereas another study[15] reported mean weight of 73 ± 12 Kg in Quincke group and 74 ± 13 Kg in Whitacre group.

There is considerable evidence that the PDPH is due to a low CSF pressure consequent upon seepage of CSF through the dural puncture hole, choroid plexus is unable to secrete sufficient fluid to maintain the CSF pressure. Moreover the negative pressure in the epidural space may draw CSF from subarachnoid space. Cerebro spinal fluid leakage from the punctured dural site produces loss of CSF pressure, which in turn leads to intracranial venous dilatation resulting in an increase in brain volume in the upright position. There occurs a difference in CSF volume and also pressure difference between the intracranial and intravertebral part of the subarachnoid space. Venous dilation and compensatory increase in brain volume will result in brain sag which in turn will exert traction and stimulate pain sensitive anchoring structures like dural vessels, basal dura and tentorium cerebelli, causing post spinal headache. Larger the hole in dura mater, more will be the leakage of CSF and longer the time required for repair. The number of holes in the dura also makes a difference in the loss of CSF. It takes about two weeks or more for the holes to seal.[13]

In this study the incidence of PDPH was 3.98% in Quincke group and 0.57% patients in Whitracre group and this difference was statistically significant using test of proportion (p=0.031). A study[15] conducted to assess failed spinal anaesthesia and PDPH in orthopedic patients using 27 G Whitacre and Quincke needles reported incidence of one percent in Quincke group and 0.5% in Whitacre group. The incidence of PDPH with the Quincke 27 gauge needle

compares well with several other studies which report zero to four percent with highest occurrence in obstetric population.[48,49]

Whereas another study[14] conducted to assess PDPH after spinal anaesthesia in young orthopaedic patients reported higher incidence of PDPH in Quincke 27 G group compared to Whitacre 27 G group (10.3% versus 8.2%).

A similar Indian study[13] conducted to assess PDPH in caesarean section using 27 G Whitacre and Quincke needles reports 12.5% incidence in 27 G Quincke and 4.5% in 27 G Whitacre.

In the present study, 62.5% of the patients in Quincke group required one attempt, 35.80% required two attempts and 1.70% required three attempts for successful block. Whereas in Whitacre group 84.66%, 13.64% and 1.70% of the patients required one, two and three attempts respectively. When these values were compared using chi-square test significant association was recorded between the type of needle and number of attempts ($p=0.0001$). A study[15] conducted to assess failed spinal anaesthesia and PDPH in orthopedic patients using 27 G Whitacre and Quincke needles reported one attempt in 81% patients, two attempts in nine percent and three attempts in four percent among the patients with Quincke group and 82.5%, 9.5% and three percent patients in Whitacre group required one, two and three number of attempts for successful spinal anaesthesia.

In the present study incidence of 2.84% failed spinal anaesthesia in Quincke Group and 3.98% in Whitacre Group was recorded. However, no statically significant association between the type of the needle and the number of

failed spinal anaesthesia could be recorded (p=0.557). A similar Indian study[13] conducted to assess PDPH in caesarean section using 27 G Whitacre and Quincke needles reports four percent failure rate in 27 G Quincke group and 12% in 27 G Whitacre and these failure rates were not statistically significant. Whereas in another study[15] failure to achieve dural puncture was more common with Quincke group than with Whitacre needle (5.5% versus 3.5).

This variation of failure rates may have attributed to the difference in tactile sensation on dural puncture. Another possible explanation may be that the appearance of CSF in Quincke needle hub is no guarantee of the needle bewel being completely within the subarachnoid space. Another possibility may be side port may straddle the dura causing leakage into the subdural or epidural space which is most commonly seen in Whitacre needles and as with all finer gauge needles, pain staking care is required to avoid dislodging the needle tip in subarachnoid space leading to loss of some local anaesthetic.

To summarise the Whitacre 27 G needle has better results with respect to PDPH and number of attempts required for successful subarachnoid block whereas the incidence of failed spinal anaesthesia was less with Quincke 27 G needle. More studies need to be done in this regard to find out the effectiveness of these needles.

CONCLUSION

This one year randomized clinical trial conducted in the Department of Anaesthesiology, K.L.E.S. Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2009 to December 2009 on 176 patients each in Whitacre 27 G and Quincke 27 G needle group. The study may be concluded as below;

- In this study females outnumbered males in both the groups.
- The mean age in Quincke group was 38.58 ± 11.95 years whereas in Whitacre group it was 40.74 ± 12.25 years.
- The mean weight of the patients in Quincke group was 55.57 ± 11.95 kg whereas in Whitacre group it was 56.91 ± 7.43 Kg.
- Significantly high incidence of PDPH was recorded in Quincke group (3.98%) as compared to 0.57% in Whitacre group (p=0.031).
- Significantly less number of attempts were required using Whitacre 27 G needle (p=0.0001).
- Failed rates were higher in patients using Whitacre 27 G needle as compared to Quincke 27 G needle (3.98% versus 2.84%).

SUMMARY

Spinal anaesthesia is one of the most commonly used technique in anaesthesia. It is economical safe, cost effective, easy, needs less sophisticated anaesthetic equipment, drugs, post operative care hence preferred over general anaesthesia and most popular because of its profound analgesia and muscle relaxation.

This one year randomized clinical trial was an attempt to compare role of two needles that is Quincke 27 G and Whitacre 27 G needles with respect to the incidence of PDPH as well as the number of attempts required to administer successful subarachnoid block (SAB).

Objectives of the present study were to know the incidence of PDPH, number of attempts for successful SAB and incidence of failed spinal anaesthesia on using 27 G Quincke's and Whitacre's spinal needles.

This study was conducted in the Department of Anaesthesiology, K.L.E.S. Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the period of January 2009 to December 2009 on 352 patients between 20 to 60 years of age with ASA grade I and II undergoing lower abdominal and lower limb surgeries during the study period.

In this study female preponderance was seen. Significantly high incidence of PDPH was recorded in Quincke group (3.98%) as compared to 0.57% in Whitacre group (p=0.031). Significantly less number of attempts were required using Whitacre 27 G needle (p=0.0001). Failed rates were higher in patients

using Whitacre 27 G needle as compared Quincke 27 G needle (3.98% versus 2.84%).

Overall the Whitacre 27 G needle has better results with respect to PDPH and number of attempts required for successful subarachnoid block whereas the incidence of failed spinal anaesthesia was less with Quincke 27 G needle.

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ANNEXURE I - INFORMED CONSENT

YOUR PARTICIPATION

A study, "**A RANDOMISED CLINICAL TRIAL TO COMPARE THE POST DURAL PUNCTURE HEADACHE FOLLOWING SPINAL ANAESTHESIA USING 27 G QUINCKE'S AND 27 G WHITACRE'S SPINAL NEEDLES"** is being conducted by Dr. ***** *******, post graduate in anaesthesiology at Jawaharlal Nehru Medical College, KLE University, Belgaum, Karnataka. Under guidance of Dr. ******* Professor Department of Anaesthesiology, Jawaharlal Nehru Medical College, KLE University, Belgaum, Karnataka.

Respected _____ we request you to participate in our study as you are eligible to be included. During the study you will be asked questions regarding your present and past medical history and you are suppose to answer to the best of your knowledge.

Your participation in this study is voluntary. Your decision whether or, not, to participate in the study will not affect your relationship with Jawaharlal Nehru Medical College, Belgaum. If you decide to participate you are free to withdraw at any point of time. The purpose of the study is to compare the post dural puncture headache between 27 G Quincke's and 27 G Whitacre's spinal needle following spinal anaesthesia.

Objective of the study

Objective of my study is to compare the the post dural puncture headache between 27 G Quincke's and 27 G Whitacre's spinal needle following spinal anaesthesia.

Procedure involved

If you agree to enroll yourself in my study, you will be interviewed regarding your present, past and family history then you will be clinically examined in detail and investigated accordingly. You will be randomly allocated either into study Group A or Group B, if you are in Group A will receive spinal anaesthesia with 27 G Quincke's spinal needle and if in group B you will receive spinal anaesthesia with 27 G Whitacre's spinal needle.

Benefits and Risks

The benefits of taking part in this research are that we can avoid general anaesthesia with good quality of analgesia. The risks associated are minimal which include hypotension, bradycardia, headache, meningitis, nerve injury and backache.

Voluntary participation / Withdrawal

Taking part in the study is voluntary. You may choose not to enroll yourself in this study. Your decision will not change present or future health care services offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

Alternatives

Even if you decline the participation in the study, you will get the routine line of management.

Confidentiality

All information collected about me during the course of the study will be kept confidential to the extent permitted by law. The code numbers will identify you in this study records and the information from this study may be published but your identity will be confidential in any publication.

Financial Incentives for participation

No financial incentives are being offered to enrolled patients. It is purely being done with the idea of research and all the cost of the study will be borne by the investigator.

Compensation

In the event of injury, related to the study, treatment will be made available at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. No reimbursement, compensation or free medical care will be given, by law. If you are injured, you may contact Dr. ********* at Department of Anaesthesiology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum or by Phone No. 08312473777.

Queries/ Contact details

If you have any queries, in future or in case of study related injury or

illness, you may contact. Dr. ****** ******* at Department of Anaesthesiology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum, Phone No. 0831-2473777.

If you have any queries about your rights as a study subject, you may call Principal and Chairman, Jawarlal Nehru Medical College Institutional Ethical Committee for Human Subjects Research, Phone No. 0831-2473777 at Jawaharal Nehru Medical College, Belgaum.

CONSENT TO PARTICIPATE IN A RESEARCH STUDY:

I, Mr./Mrs. _____

voluntarily agree to take part in this study, by signing this consent form I am not giving up my legal rights. I may withdraw at any time. I am signing after having read, or been read to me in the vernacular language including risks and the benefits and having all queries cleared.

Signature of the participant: _____

Witness name: _____

Signature of the participant: _____

Date: _____

Place: _____

Signature of Investigator: _____

ANNEXURE II – PROFOMA

STUDY: "A RANDOMISED CLINICAL TRIAL TO COMPARE THE POST DURAL PUNCTURE HEADACHE FOLLOWING SPINAL ANAESTHESIA USING 27 G QUINCKE'S AND 27 G WHITACRE'S SPINAL NEEDLES"

Patient Name :	I.P. No:
Age :	Weight:
Height :	Gender:
Date of Operation:	Occupation:
Address :	Anaesthesiologist:

Preanaesthetic evaluation

Chief Complaints

Past History

a. HTN / DM / Asthma / Epilepsy / Drug allergy

b. Drug therapy

c. Previous exposure to anaesthesia

Family history

General Physical Examination

Pallor / Icterus / Clubbing / Lymphadenopathy / Odema

P.R.: B.P.:

R.R.:

Musculoskeletal disorders

Jaw movements Teeth:

Airway assessment: Spine:

Systemic examination

R.S. CNS

CVS GIT

Investigations

Hb%: Total count:

Differential count: Bleeding time:

Clotting time: PT:

aPTT: INR:

Urine routine

Any others

Preoperative physical status: ASA Grade I II III IV V

Diagnosis:

Proposed surgery:

Preoperative baseline:

HR : BP:

Monitors attached

Pulse oximeter Non invasive blood pressure:

ECG

1. Group: Group A / Group B

2. Number of attempts

3. Failed spinal anaesthesia Yes / No

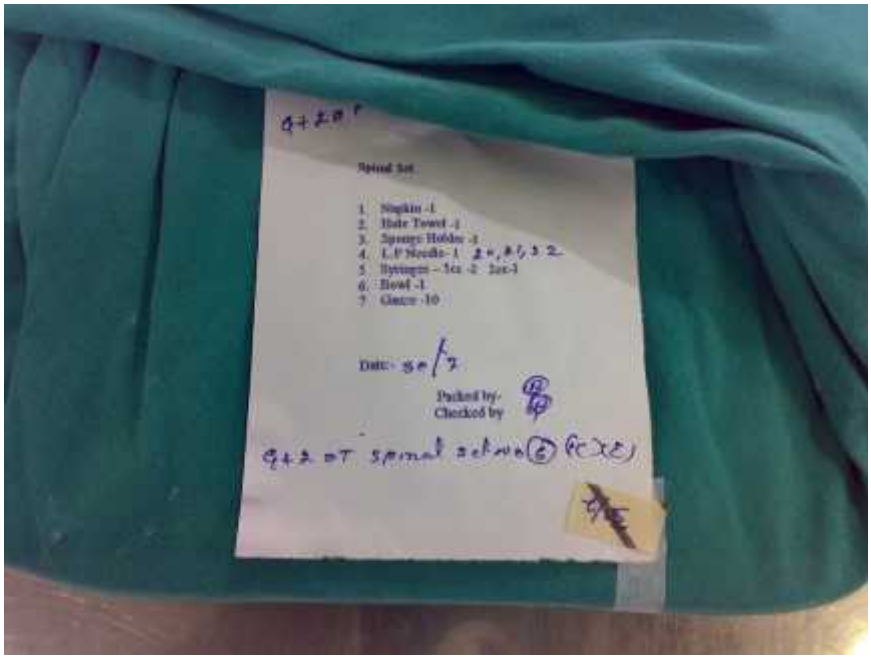
4. Vital parameters

Time	**Heart rate**	**Blood pressure**	**SPO_2**
3 minutes			
6 minutes			
9 minutes			
12 minutes			
15 minutes			
20 minutes			
25 minutes			
30 minutes			
40 minutes			
50 minutes			
60 minutes			

5. Post dural puncture headache

Day	PDPH	Degree of severity
< 24 hours	Yes / No	
24 to 48 hours	Yes / No	
48 to 72 hours	Yes / No	

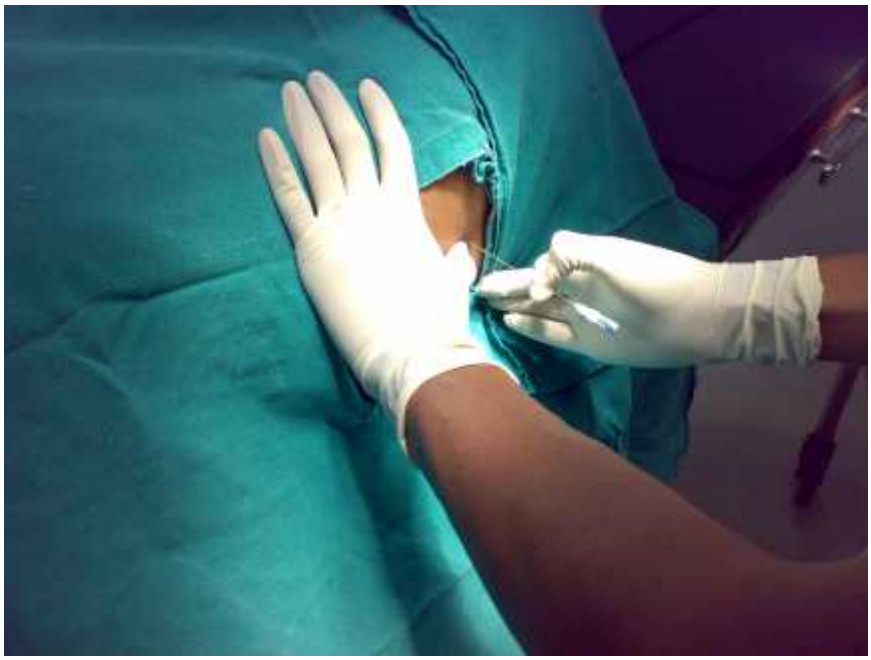
ANNEXURE III – PHOTOGRAPHS



Photograph 1. Spinal set

Photograph 2. Spinal tray

Photograph 3. Positioning



Photograph 4. Procedure of spinal anaesthesia

MASTER CHART - QUINCKE GROUP

Sl. No.	IP No	Sex	Age (Years)	Weight (Kg)	PDPH	No. of Attempts	Failed spinal anaesthesia
1	312275	M	28	45	-	2	-
2	310848	F	26	52	-	2	-
3	315062	M	30	58	-	2	-
4	314280	M	47	67	-	2	-
5	314042	M	41	58	-	2	-
6	314008	F	35	60	-	1	-
7	314483	M	28	52	-	2	-
8	314327	F	50	48	-	2	+
9	313467	M	22	45	-	2	-
10	314573	M	25	60	-	2	+
11	314293	F	28	68	-	1	-
12	318844	M	59	70	-	2	-
13	312525	F	50	67	-	1	-
14	312435	M	30	52	-	1	-
15	312687	M	58	58	-	2	-
16	318478	M	24	70	-	1	-
17	313482	F	42	52	-	2	-
18	311774	F	25	50	-	1	-
19	315501	M	35	70	-	1	-
20	312784	M	58	67	-	1	-
21	316242	M	32	67	-	1	-
22	312572	F	20	52	-	1	-
23	314778	F	59	56	-	1	-
24	318848	F	32	42	-	2	-
25	312831	M	23	50	-	1	-
26	314971	F	46	58	-	2	-
27	315239	F	25	72	-	1	-
28	316070	F	58	50	-	2	-
29	316003	F	58	42	-	1	-
30	315664	M	44	70	-	1	-
31	313140	M	30	56	-	1	-
32	311539	M	28	50	+	2	-
33	312046	F	55	68	+	2	-
34	315990	M	30	60	-	2	-
35	327888	M	39	60	-	2	-
36	323778	M	60	58	-	2	-
37	324251	M	47	62	-	2	-
38	323267	M	30	50	-	1	-
39	326927	F	43	58	-	2	-
40	326928	F	44	58	-	2	-
41	324882	M	40	58	-	2	-
42	325186	F	27	30	-	2	-
43	324522	M	50	52	-	1	-
44	326682	F	47	58	-	2	-
45	309271	M	45	56	-	1	-
46	311579	F	40	40	-	2	-
47	306383	M	55	58	+	2	-

MASTER CHART - QUINCKE GROUP

Sl. No.	IP No	Sex	Age (Years)	Weight (Kg)	PDPH	No. of Attempts	Failed spinal anaesthesia
48	306689	F	52	52	-	1	-
49	323667	M	37	58	+	1	-
50	323456	M	56	58	-	1	-
51	324565	M	58	56	-	1	-
52	308179	M	28	70	-	1	+
53	305082	M	44	64	-	2	-
54	305161	M	28	58	-	1	-
55	325056	F	26	52	+	1	-
56	314343	M	22	58	-	1	-
57	313461	M	23	50	-	2	-
58	314393	M	50	50	-	2	-
59	313402	M	35	58	-	2	-
60	313491	M	23	58	-	1	-
61	316131	M	22	58	-	2	-
62	343234	F	25	50	-	1	-
63	314313	M	38	54	-	2	-
64	313606	M	56	70	-	2	-
65	305281	M	55	54	+	1	-
66	310968	M	35	48	-	1	-
67	311024	F	39	52	-	1	-
68	310781	M	31	60	-	1	-
69	310952	F	31	48	-	1	-
70	321456	M	42	48	-	1	-
71	310847	F	27	48	-	1	-
72	310844	F	25	47	-	1	-
73	310848	F	25	60	-	1	-
74	316951	M	23	58	-	1	-
75	323573	M	53	58	-	1	-
76	323996	M	26	48	-	2	-
77	323267	M	30	60	-	1	-
78	324251	M	42	65	-	1	-
79	323778	M	60	65	-	3	-
80	81156	F	28	48	-	2	-
81	71803	F	24	48	-	1	-
82	318045	F	24	52	-	2	-
83	318145	M	27	50	-	1	-
84	3181107	M	48	68	-	1	-
85	318159	M	45	60	-	1	-
86	317661	F	23	49	-	1	-
87	315247	F	31	48	-	1	-
88	324573	F	50	56	-	1	-
89	324173	M	60	60	-	1	-
90	323667	M	38	67	-	2	-
91	323190	M	60	70	-	1	-
92	326742	M	22	42	-	1	-
93	323578	M	32	58	-	2	-
94	324422	M	45	58	-	1	-

MASTER CHART - QUINCKE GROUP

Sl. No.	IP No	Sex	Age (Years)	Weight (Kg)	PDPH	No. of Attempts	Failed spinal anaesthesia
95	316951	M	55	74	-	1	-
96	306542	F	55	77	+	1	-
97	323573	M	23	52	-	1	-
98	325518	M	55	68	-	1	-
99	326842	F	26	49	-	3	-
100	326680	M	20	48	-	2	-
101	323996	F	25	50	-	2	-
102	326080	M	20	72	-	2	-
103	325375	F	42	59	-	1	-
104	324449	F	35	44	-	1	-
105	326063	M	38	58	-	2	-
106	325067	F	40	58	-	2	-
107	326826	M	32	58	-	2	-
108	326826	M	59	58	-	2	-
109	326848	M	45	52	-	1	-
110	327265	F	21	42	-	2	-
111	326526	M	35	60	-	1	-
112	356406	F	50	54	-	1	-
113	325039	F	45	58	-	2	-
114	326647	M	53	58	-	2	-
115	325856	M	55	58	-	1	-
116	323335	M	22	52	-	1	-
117	324093	F	45	48	-	1	-
118	325240	F	48	50	-	2	-
119	323351	M	28	54	-	1	-
120	322888	M	38	52	-	1	+
121	322088	M	38	52	-	1	-
122	329372	M	48	52	-	1	-
123	324800	M	60	56	-	1	-
124	312828	M	58	58	-	1	-
125	324628	F	28	52	-	1	-
126	316951	M	28	58	-	1	-
127	316618	M	39	52	-	1	-
128	313641	F	58	58	-	1	-
129	313416	M	36	58	-	2	-
130	325375	F	42	60	-	1	-
131	313426	M	35	58	-	2	-
132	331020	M	22	44	-	1	-
133	331080	F	42	52	-	1	-
134	331816	M	28	58	-	2	-
135	328701	M	50	52	-	1	-
136	338166	M	38	58	-	1	-
137	328433	F	42	52	-	1	-
138	331516	F	48	48	-	3	-
139	327487	F	48	52	-	1	-
140	330586	M	48	48	-	2	-
141	363041	M	38	52	-	1	-

MASTER CHART - QUINCKE GROUP

Sl. No.	IP No	Sex	Age (Years)	Weight (Kg)	PDPH	No. of Attempts	Failed spinal anaesthesia
142	326098	M	52	52	-	1	-
143	327656	F	22	48	-	2	-
144	3126928	F	58	52	-	2	-
145	338881	F	40	50	-	1	-
146	313416	M	26	58	-	2	-
147	336650	F	31	58	-	1	+
148	336650	F	40	40	-	1	-
149	336706	F	46	60	-	1	-
150	336706	F	40	50	-	1	-
151	336370	F	40	60	-	1	-
152	336512	F	50	60	-	1	-
153	336667	F	32	50	-	1	-
154	337734	F	28	50	-	1	-
155	332545	M	45	48	-	1	-
156	326235	F	25	38	-	1	-
157	327426	M	40	68	-	2	-
158	326407	F	37	54	-	1	-
159	328793	F	60	62	-	1	-
160	328765	F	40	60	-	1	-
161	327448	F	25	50	-	1	-
162	326842	F	55	54	-	1	-
163	324882	M	40	72	-	2	-
164	325099	F	45	48	-	1	-
165	323416	M	36	52	-	2	-
166	314361	M	52	60	-	2	-
167	316387	M	32	58	-	2	-
168	313614	M	60	79	-	1	-
169	313614	M	36	52	-	1	-
170	3167752	F	24	52	-	1	-
171	317956	F	25	48	-	1	-
172	317980	F	30	52	-	1	-
173	317561	F	27	53	-	1	-
174	317908	F	35	50	-	1	-
175	312451	F	27	50	-	1	-
176	312456	M	34	53	-	1	-

MASTER CHART - WHITACRE GROUP

Sl. No.	IP No	Sex	Age (Years)	Weight (Kg)	PDPH	No. of Attempts	Failed spinal anaesthesia
1	314405	F	25	42	-	1	-
2	313923	F	28	58	-	2	-
3	312932	F	45	58	-	2	-
4	314218	M	46	64	-	2	-
5	314433	F	58	70	-	1	-
6	310849	F	26	50	-	1	-
7	231254	M	58	43	-	1	-
8	313806	M	54	54	-	2	-
9	313855	M	23	45	-	1	-
10	313303	F	30	48	-	1	-
11	313197	M	38	60	-	1	-
12	315732	F	45	67	-	1	-
13	313024	M	58	58	-	1	-
14	312984	M	28	48	-	1	-
15	315442	F	58	60	-	1	-
16	312475	M	35	54	-	1	-
17	314271	F	42	54	-	2	-
18	310275	F	31	70	-	1	-
19	311506	M	21	70	-	1	-
20	318276	M	58	70	-	1	-
21	313486	M	20	68	-	1	-
22	318050	F	25	67	-	1	-
23	319680	M	25	72	-	1	-
24	316570	M	55	68	-	1	-
25	313356	M	45	58	-	1	+
26	313606	M	58	58	-	1	-
27	312776	M	35	54	-	1	-
28	313377	m	32	48	-	2	-
29	312599	M	30	52	-	1	-
30	313182	M	43	48	-	1	-
31	313434	M	45	48	-	1	-
32	315231	M	56	54	-	1	-
33	326037	M	21	54	-	1	-
34	326087	F	53	58	-	1	-
35	325242	F	56	54	-	1	-
36	323762	F	30	50	-	1	-
37	323441	F	23	50	-	1	-
38	323765	M	57	62	-	1	-
39	323765	F	21	50	-	1	-
40	323836	F	37	58	-	1	-
41	324639	F	56	50	-	1	-
42	324713	M	58	58	-	1	-
43	325267	F	42	52	-	1	-
44	325236	F	47	58	-	1	-
45	325291	M	27	52	-	1	-
46	326294	F	58	58	-	1	-
47	326280	M	60	52	-	1	-

MASTER CHART - WHITACRE GROUP

Sl. No.	IP No	Sex	Age (Years)	Weight (Kg)	PDPH	No. of Attempts	Failed spinal anaesthesia
48	326224	M	52	60	-	1	-
49	328014	M	38	70	-	1	-
50	326681	M	57	65	-	1	-
51	327088	F	50	50	-	1	-
52	326315	M	40	52	-	1	-
53	326952	M	23	52	-	1	-
54	326382	M	42	50	-	1	-
55	328182	M	58	65	-	1	-
56	299801	M	48	52	-	1	-
57	303214	F	26	53	-	2	-
58	304137	F	24	48	-	2	-
59	306078	M	41	58	-	1	-
60	303454	M	58	58	-	2	-
61	305073	M	39	60	-	3	+
62	302395	F	31	54	-	1	-
63	304065	M	43	54	-	1	-
64	328148	M	58	58	-	1	-
65	323667	M	38	68	-	1	-
66	316951	M	55	55	-	1	-
67	322886	M	38	56	-	1	-
68	323778	M	58	62	-	1	-
69	323573	M	23	55	-	1	-
70	324173	M	59	58	-	2	-
71	324573	M	50	60	-	3	+
72	323578	M	32	59	-	1	-
73	324832	M	40	70	-	1	-
74	325062	F	50	56	-	1	-
75	322325	F	25	60	-	1	-
76	326147	M	53	72	-	1	-
77	325039	F	45	68	-	1	-
78	325186	F	22	57	-	1	-
79	324053	F	45	62	-	1	-
80	326790	M	39	50	-	1	-
81	326063	M	38	70	-	1	-
82	322866	M	55	68	-	1	-
83	325886	F	28	52	-	1	-
84	323472	M	27	52	-	1	-
85	323331	M	28	70	-	1	-
86	331447	M	53	60	-	1	-
87	331468	F	28	45	-	2	-
88	328370	F	60	55	-	2	+
89	328811	M	55	58	-	1	-
90	330710	M	35	58	-	2	-
91	328899	M	58	62	-	1	-
92	331385	F	42	43	-	2	-
93	331560	M	38	60	-	1	-
94	334128	M	32	58	-	1	-

MASTER CHART - WHITACRE GROUP

Sl. No.	IP No	Sex	Age (Years)	Weight (Kg)	PDPH	No. of Attempts	Failed spinal anaesthesia
95	323267	M	30	52	-	1	-
96	323190	M	60	58	-	1	-
97	324247	M	60	52	-	1	-
98	323996	F	25	50	-	1	-
99	324358	F	20	52	-	1	-
100	336555	F	32	60	-	1	-
101	337059	F	35	62	-	1	-
102	336885	F	33	64	-	1	-
103	336796	F	26	56	-	1	-
104	304065	F	23	48	-	1	-
105	302908	F	23	46	-	1	-
106	304088	F	29	62	+	2	-
107	306071	F	41	78	-	3	-
108	305921	F	42	62	-	1	-
109	305281	F	42	52	-	1	-
110	305882	F	48	48	-	1	-
111	322529	M	21	48	-	1	-
112	311859	M	22	47	-	1	-
113	312013	M	23	54	-	1	-
114	307171	M	25	52	-	1	-
115	305841	M	45	52	-	1	-
116	301117	F	40	43	-	1	-
117	310818	M	47	78	-	1	-
118	304349	F	26	45	-	1	-
119	310943	F	50	56	-	1	-
120	310877	F	45	56	-	1	-
121	346988	M	38	58	-	1	-
122	311594	M	55	58	-	1	-
123	310849	F	25	46	-	1	-
124	321497	M	51	61	-	2	-
125	323446	M	30	62	-	1	-
126	320587	M	58	60	-	1	-
127	321518	F	40	58	-	2	-
128	324207	M	59	50	-	2	-
129	323190	M	60	75	-	1	-
130	322888	M	38	65	-	1	-
131	323299	F	44	52	-	1	-
132	314468	F	20	42	-	1	-
133	321273	M	58	58	-	1	-
134	323258	F	52	52	-	1	-
135	323140	F	35	52	-	1	-
136	323416	M	30	59	-	1	+
137	323244	F	30	52	-	1	-
138	323446	M	28	58	-	1	-
139	322488	F	48	54	-	1	-
140	323446	M	25	58	-	1	-
141	320375	M	42	72	-	2	-

MASTER CHART - WHITACRE GROUP

Sl. No.	IP No	Sex	Age (Years)	Weight (Kg)	PDPH	No. of Attempts	Failed spinal anaesthesia
142	320120	M	28	62	-	1	-
143	322136	M	50	62	-	2	-
144	321456	F	54	54	-	1	-
145	324512	F	34	54	-	1	-
146	322272	F	53	52	-	1	-
147	304321	F	37	54	-	1	-
148	304323	F	39	52	-	1	-
149	324576	M	58	54	-	1	-
150	321138	M	27	58	-	2	-
151	321576	F	30	52	-	1	-
152	321598	F	36	49	-	1	-
153	323066	M	45	52	-	2	-
154	323088	F	42	54	-	1	-
155	322397	F	44	52	-	1	-
156	321828	M	57	58	-	1	-
157	327506	M	38	65	-	1	-
158	325260	M	37	58	-	1	-
159	324543	M	48	50	-	1	-
160	324598	F	49	56	-	1	-
161	323456	F	54	65	-	1	-
162	324578	M	50	66	-	1	-
163	326011	M	58	57	-	2	+
164	309867	M	37	58	-	1	-
165	324531	F	54	49	-	1	-
166	324987	M	49	57	-	1	-
167	327248	M	30	60	-	1	+
168	327654	M	39	65	-	1	-
169	327932	M	40	58	-	1	-
170	324221	F	36	59	-	1	-
171	324576	M	54	67	-	1	-
172	327090	F	25	63	-	1	-
173	326574	M	45	56	-	1	-
174	326757	M	38	38	-	2	-
175	324325	F	48	54	-	1	-
176	327802	M	30	70	-	1	-

ANNEXURE IV

KEY TO MASTER CHART

F	-	Female
IP No.	-	Inpatient number
Kg	-	Kilo gram
M	-	Male
No.	-	Number
PDPH	-	Post dural puncture headache
Sl. No.	-	Serial number